Intracranial Pressure Monitoring in the Management of Penetrating Brain Injury

I. RECOMMENDATIONS

A. Standards

The available data are not sufficient to support a treatment Standard for the use of intracranial pressure (ICP) monitoring in the management of acute penetrating brain injury (PBI).

B. Guidelines

The available data are not sufficient to support a treatment Guideline for this topic.

C. Options

Early ICP monitoring is recommended when the clinician is unable to assess the neurologic examination accurately; the need to evacuate a mass lesion is unclear; or imaging studies (e.g., computed tomographic scan) suggest elevated intracranial pressure. In the absence of studies specific to managing intracranial hypertension in PBI patients, we recommend that the clinician follow the recommendations in Guidelines for the Management of Severe Head Injury and the enlarged revision Guidelines for the Management of Severe Traumatic Brain Injury.

II. OVERVIEW

ICP monitoring has been established as an important variable in the prognosis and management of severe nonpenetrating traumatic brain injury (TBI) because intracranial hypertension is clearly associated with worse recovery and effective control of elevated ICP appears to improve outcome. In contrast to nonpenetrating TBI, few studies on ICP in PBI have been performed, and the majority of those studies do not provide ICP-related data. Few studies on intracranial hypertension after PBI in the military have been published, and there is a dearth of civilian literature on use of ICP monitoring for patients with PBI. Because all available studies are observational and without controls, this section of the Guideline can make no recommendations stronger than Options (see definitions above). However, some aspects of ICP management discussed in the literature on nonpenetrating TBI can be generalized and applied logically to PBI, even though all information is derived from Class III data.

III. PROCESS

A MEDLINE search from January 1966 to January 2000 using the search terms wounds, gunshot, and brain injuries or head injuries, limited to human subjects, identified 382 articles from peer-reviewed journals. Eighty-eight articles were rejected on the basis of clearly irrelevant titles; an additional 33 articles were then identified from the bibliographies of reviewed articles. The primary selection process, therefore, identified 327 articles for further review. Two independent reviewers read the abstracts of all 327 articles and selected 65 for inclusion. Articles were rejected on the basis of relevance to the topic (e.g., non-English language, case reports, irrelevance to project, atypical mechanisms of injury, and series of less than 10 subjects with no other unique reasons for inclusion). The 65 articles were then read in detail, and more articles were considered from their bibliographies. Of these 65 articles, 52 were directly relevant to the topics of surgical management, monitoring intracranial pressure, and treating cerebrospinal fluid leaks. A total of seven articles were appropriate for this topic and were analyzed in detail for this section.

IV. SCIENTIFIC FOUNDATION

Studies that include analyses of data on ICP in PBI reveal that intracranial hypertension is common after PBI. Lilbard reported that in a series of 18 patients with PBI who died and had a postmortem examination, all had evidence of increased intracranial pressure with temporal lobe and/or cerebellar tonsillar herniation. He also recorded ICP in 12 patients and found “significant” (degree not specified) intracranial hypertension in 11 (92%). Miner et al. monitored ICP in 18 of 33 children with gunshot wounds to the brain who were deemed salvageable but not so neurologically intact as to preclude monitoring. Of the 12 children with ICP values uncontrollably above 40 mm Hg, 8 died (75%), 2 were left with severe deficits, and 2 were left with moderate deficits. In contrast, all patients having ICPs controllable at < 20 mm Hg had good outcomes or moderate disabilities at 6 months after injury. Nagib et al. recorded ICP in 13 civilian patients with PBI and found that the majority of those with intracranial hypertension that responded to treatment survived, whereas all those in whom intracranial hypertension was refractory died. They also found that outcome was superior in those who never manifested intracranial hypertension. Crockard recorded ICP in 20 patients very early after PBI and found a very high mortality associated with elevated ICP and a much better outcome in those in whom intracranial hypertension was never manifest. He also found a group with very low ICP associated with profound shock, but when ICP rose to normal after resuscitation, it presaged survival, whereas intracranial hypertension after resuscitation was uniformly associated with mortality. His analysis of the relationship between ICP and blood pressure suggested that pressure autoregulation was de-
fective after PBI, and that this commonly resulted in intracranial hypertension associated with respiratory difficulties, such as coughing or respiratory distress. He stressed the importance of full-volume resuscitation and aggressive airway and respiratory management, including endotracheal intubation and mechanical ventilation.

Unfortunately, in all of these reports, the percentage of total patients who received ICP monitoring was small and not randomized, so their results cannot be generalized. Nevertheless, all reports indicate that intracranial hypertension is common after PBI and that response to treatment may be associated with improved outcome in some patients.

Siccardi et al. published the only study in which ICP monitoring was not described favorably. In their report, recording of ICP “was performed in several of the surgical cases and in a few of the conservatively treated cases.” They stated that “there was no significant difference in the outcome between patients who were and who were not submitted to postoperative measurement of intracranial pressure.” Unfortunately, they presented no data to support this statement, did not describe the characteristics of the patients undergoing ICP monitoring, and did not describe either their method for managing intracranial hypertension or its results.

The physiology of intracranial hypertension after PBI has not been well studied. Postmortem studies of PBI victims reveal a preponderance of evidence for intracranial hypertension. Kirkpatrick and DiMaio found sulcal flattening and signs of transtentorial herniation in 39 of 42 PBI patients at necropsy. Some of the marked elevation in ICP deduced from this evidence may have been associated with the cavitation associated with the missile wound. However, it appeared that cerebral edema can appear extremely rapidly and contribute to intracranial hypertension in patients with treatable PBI. As noted above, Crockard’s study found that intracranial hypertension occurred frequently among patients in whom ICP monitoring was instituted in under 20 minutes after arrival at a medical facility. It appears that some patients with intracranial hypertension can be treated and survive with acceptable morbidity. However, the literature does not clearly relate successful treatment of intracranial hypertension after PBI to an improved outcome.

In addition, no solid information is available to guide management strategies for intracranial hypertension (ICH) after PBI. It cannot be assumed that the intracranial pathology or its evolution over time is the same as in nonpenetrating TBI. It has been suggested that failure of cerebral pressure autoregulation is a common source of ICP elevations after PBI. Hyperventilation has been considered a mainstay of treatment and even in prophylaxis of post-PBI intracranial hypertension. The degree of hyperventilation reported in the literature for both of these indications has often been extreme (i.e., Paco2 levels of 20–25 mm Hg). In light of the current concern about induced hypoperfusion caused by hyperventilation, however, the risk/benefit ratio of hyperventilation needs to be compared with the other measures available for controlling ICH. Absent the evidence of a clear relationship between hyperventilation in PBI and subsequent hypoperfusion, our present poor level of understanding of the pathophysiologic alterations after PBI is clearly an area for further study.

One practical use for ICP monitoring is to follow patients with depressed levels of consciousness as an early sign of progressive intracranial pathology that may require repeated imaging and surgical decompression. The utility of ICP monitoring as a surrogate for a more sensitive clinical indicator of neurologic deterioration is probably the most logical parallel between nonpenetrating TBI and PBI.

In cases where ICP is monitored and intracranial hypertension is present, treatment has involved the same measures used in nonpenetrating TBI (e.g., hyperventilation, mannitol, cerebrospinal fluid drainage, and high-dose barbiturates). Prophylaxis against intracranial hypertension, an aspect of care frequently addressed in the limited literature, uses the same agents. In this context, the potential toxicity of such agents compared with the risks of monitoring ICP becomes an important consideration. This same risk/benefit analysis was carried out for nonpenetrating TBI in Guidelines for the Management of Severe Head Injury. That evidence-based document concludes that the risks of ICP monitoring are frequently outweighed by the potential toxicity of anti-ICH agents that may not be necessary. For example, several prospective, randomized, controlled trials showed that high-dose barbiturates were not useful as prophylaxis for preventing ICH in nonpenetrating TBI. Furthermore, although high-dose barbiturates are effective in lowering ICP elevations refractory to less aggressive treatments in these same patients, they should not be used in patients without proven intracranial hypertension and in whom less hazardous treatment measures have not been tried. The unclear parallelism between nonpenetrating TBI and PBI precludes direct application of the analysis contained in Guidelines for the Management of Severe Brain Injury to PBI. Nevertheless, the risk/benefit question for “empiric” treatment versus ICP monitoring may be pertinent to patients with PBI and suggests that empiric treatment of increased ICP is not beneficial.

V. SUMMARY

The role of ICP monitoring and its application in PBI have been incompletely studied. In the available literature, intracranial hypertension appears to be common after PBI and, when present, is predictive of less favorable outcome. It also appears that systemic hypertension can exacerbate, and potentially be solely responsible for, ICP elevation after PBI. Unfortunately, few data reveal whether or how successful management of intracranial hypertension improves outcomes in patients with PBI. Most of the few available Class III reports suggest that patients whose ICP can be maintained below 20 mm Hg have more favorable recovery than patients with refractory intracranial hypertension. There is some Class III evidence that indicates that ICP monitoring can be useful to help detect deterioration in patients with depressed levels of consciousness.
Unfortunately, almost no data are available on the optimal method for treating post-PBI intracranial hypertension. Few data are available on the efficacy of individual methods of management, and there are no data on their relative efficacy. Without such evidence, it seems appropriate to defer to what is known about treating intracranial hypertension in nonpenetrating TBI regarding the absolute and relative efficacies of individual treatment methods, as outlined in *Guidelines for the Management of Severe Traumatic Brain Injury*.2

**VI. KEY ISSUES FOR FUTURE INVESTIGATIONS**

Our knowledge regarding the role of intracranial hypertension in determining outcome in PBI is more primitive than for nonpenetrating TBI. Nevertheless, many of the issues are similar. The nature of the pathophysiologic processes involved, their similarity to those occurring in nonpenetrating TBI, and the optimal methods for detecting, measuring, and treating them are yet to be determined. Formal studies are needed to assess the prognostic significance of ICP elevations after PBI. How to identify those patients at risk for intracranial hypertension and what level of ICH requires treatment also need to be defined. Finally, whether managing intracranial hypertension improves outcome in PBI and how this management interacts with the manipulation of other physiologic parameters, such as cerebral perfusion pressure and cerebral oxygen delivery, are topics for future study.

Because there is so little research on the above issues, there is no clear evidence that any particular diagnostic or therapeutic approach is superior to any others in PBI. As such, the ethical constraints that have prevented definitive research into the utility of ICP monitoring and management in improving outcome in patients with nonpenetrating TBI will not a priori apply to PBI. For this reason, it should remain possible to perform a prospective, randomized, controlled trial on the role of ICP management in improving PBI.

**VII. Evidentiary Table: ICP Monitoring in Penetrating Brain Injury**

<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Description of Study</th>
<th>Data Class</th>
<th>Conclusions</th>
</tr>
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<tbody>
<tr>
<td>Crockard, 19756</td>
<td>Civilian (n = 20). Intracranial pressure was monitored within 20 min in patients with gunshot wounds to the head in Belfast.</td>
<td>III</td>
<td>Intracranial hypertension was frequent in patients in whom ICP monitoring was instituted in less than 20 min. Reported very high mortality associated with elevated ICP and much better outcome among patients who never manifested intracranial hypertension. Concluded that patients with intracranial hypertension can be treated and survive with acceptable morbidity. ICP monitoring useful as an early sign of progressive intracranial pathology and as an indication for repeated imaging and surgical decompression. Thought that failure of cerebral pressure autoregulation is a common source of ICP elevations after PBI.</td>
</tr>
<tr>
<td>Kirkpatrick and DiMaio, 197813</td>
<td>Civilian (n = 42). Postmortem anatomic study of fatal gunshot wounds to the brain.</td>
<td>III</td>
<td>Found sulcal flattening and signs of transtentorial herniation in 39 of 42 PBI patients at necropsy.</td>
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<tr>
<td>Lillard, 197813</td>
<td>Civilian (n = 83). The report reviewed clinical course and conservative surgical approach to victims with civilian PBI seen over a 5-y period.</td>
<td>III</td>
<td>Recorded ICP in 12 patients and found “significant” intracranial hypertension in 11 (92%).</td>
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<td>Miner et al., 199014</td>
<td>Civilian (n = 33). Retrospective study of outcome from gunshot wounds to the brain in a pediatric population (8 mo through 5 y) in an urban setting.</td>
<td>III</td>
<td>Monitored ICP in of 33 children with gunshot wounds to the brain that were deemed neither unsalvageable nor too intact to preclude the need for monitoring. 75% mortality in 12 with ICP values uncontrollably &gt; 40 mm Hg, with two left with severe deficits, and two with moderate deficits. All patients with ICPs controllable at &lt; 20 mm Hg had good outcomes or moderate disabilities at 6 mo after injury.</td>
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<td>Nagib et al., 198615</td>
<td>Civilian (n = 55). Over a 5-y period, victims of cranial gunshot wounds to the head were managed and factors related to outcome including GCS score, fragmentation, and suicide were evaluated.</td>
<td>III</td>
<td>Recorded ICP in 13 patients and found that the majority of those with intracranial hypertension that responded to treatment survived, whereas all in whom intracranial hypertension was refractory died. Also found superior outcome in those who never manifested intracranial hypertension.</td>
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<tr>
<td>Sarnaik et al., 198916</td>
<td>Civilian (n = 14). The effect of aggressive control of intracranial pressure on long-term outcome of children with penetrating gunshot wound of the brain was evaluated.</td>
<td>III</td>
<td>Used ICP monitoring of severely injured children to decide whether to operate (debridement, hematoma removal). Thought that the survivors in this study had sufficient recovery of physical, behavioral, and intellectual functions to support ICP/CPP-based management in comatose pediatric victims of gunshot wounds to the brain.</td>
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ICP Monitoring in the Management of Penetrating Brain Injury

VII. Continued

<table>
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<tr>
<td>Siccardi et al., 1991</td>
<td>Civilian (n = 314). Studied the outcome of patients with gunshot wounds to the brain presenting to hospital dead or alive. Operated on all patients with postresuscitation GCS scores &gt; 3.</td>
<td>III</td>
<td>Stated that “there was no significant difference in the outcome between patients who were and who were not submitted to postoperative measurement of intracranial pressure.” Authors present no data in support of this statement.</td>
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GCS, Glasgow Coma Scale; CPP, cerebral perfusion pressure.

Given the growing importance of PBI in today’s society, such research seems highly indicated.

References