Blood Pressure Management in Traumatic Brain Injury

Lori A. Shutter, MD
Raj K. Narayan, MD

From the NSICU/Neurocritical Care Program, Clinical Neurosurgery and Neurology Departments (Shutter), and the Department of Neurosurgery (Narayan), University of Cincinnati College of Medicine, Cincinnati, OH.


Contrary to the goal in most of the other conditions discussed so far, the focus of care after traumatic brain injury is the maintenance of cerebral perfusion, rather than treatment of hypertension per se. The injured brain is vulnerable to hypotension, particularly in the presence of raised intracranial pressure. The impact of systemic insults on the injured brain has been long recognized,1 but the influence of blood pressure on outcome after traumatic brain injury has never been studied in a randomized fashion; rather, the information comes from retrospectively analyzed data. Hypotension occurs commonly in the out-of-hospital and in-hospital periods and is associated with markedly worse outcomes after brain injury.2,3 When hypertension occurs in the setting of traumatic brain injury, treatment may result in overshoot hypotension. In general, the treatment of acute hypertension in patients with traumatic brain injury is not recommended.

Out-of-Hospital Hypotension

Out-of-hospital hypotension is usually due to trauma-related blood loss and shock. Information from the Traumatic Coma Data Bank2,4 showed that in patients with severe head injury (Glasgow Coma Scale [GCS] score of 8 or less), a single episode of out-of-hospital hypotension (systolic blood pressure <90 mm Hg) was a powerful predictor of outcome, independent of the other major predictors such as age, admission GCS score, admission GCS motor score, intracranial diagnosis, and pupillary status. In addition, a single episode of hypotension was associated with increased morbidity and a doubling of mortality (55% versus 27%) compared with that of a matched group of patients without hypotension.2

In terms of treatment, studies suggest that aggressive correction of out-of-hospital hypotension using normal saline solution, lactated Ringer’s, hypertonic saline solution, or blood products improves outcome.5-7 The optimal resuscitative fluid for this patient population has not yet been established, but there is no controversy about the need for adequate resuscitation.

Impact of Cerebral Perfusion Pressure

Rosner and Daughton8 reported better outcomes in 34 patients receiving a management protocol stressing cerebral perfusion pressure maintenance greater than 70 mm Hg rather than intracranial pressure control. Cerebral perfusion pressure management became widely practiced, and it came to be assumed that if some induced hypertension was good, then more must be better. However, subsequent reports began to question the advisability of pushing the blood pressure too high. Robertson et al9 reported a randomized controlled trial comparing cerebral perfusion pressure- versus intracranial pressure-focused management. The cerebral perfusion pressure group was treated with fluids and vasopressors to maintain a cerebral perfusion pressure greater than 70 mm Hg. The control arm (traditional management) focused on maintaining an intracranial pressure of less than or equal to 20 mm Hg and a cerebral perfusion pressure greater than 50 mm Hg. Although the induced hypertension group demonstrated a significant decrease in the number of episodes of jugular oxygen desaturation (an indicator of global cerebral ischemia), they found no significant difference in outcome between the 2 groups at 6 months. Instead, they found that the risk of adult respiratory distress syndrome was 5 times greater among patients in the induced hypertension group. The risk of adult respiratory distress syndrome appeared to be associated with an increased use of vasopressors to maintain cerebral perfusion pressure. This finding was confirmed in another analysis by Contant et al.10 Thus, although maintaining an adequate cerebral perfusion pressure is clearly important in traumatic brain injury patients, there appears to be an upper ceiling to this beneficial intervention, beyond which there may be a deleterious systemic effect.

Hypertension After Traumatic Brain Injury

Cerebral perfusion pressure in patients with intracranial pressure monitoring should be maintained between 50 and 70 mm Hg, but one must also keep in mind that there are fairly strong data suggesting that more is not necessarily better. Induced hypertension should not be considered as an alternative to lowering intracranial pressure. Hypertension after traumatic brain injury may indicate preserved autoregulation efforts to optimize cerebral blood flow and cerebral perfusion pressure. Thus, aggressive treatment of blood pressure in patients with increased intracranial pressure can be dangerous and may result in worsened morbidity or even
death. Sustained hypertension beyond acceptable physiologic boundaries may represent an underlying medical condition, pain, or anxiety. Although treatment with analgesics and anxiolytics is warranted, the hypotensive potential of these agents must be recognized; therefore, low doses of short-acting agents are recommended. If treatment with appropriate interventions does not result in improvement and marked hypertension is observed, then short-acting agents such as labetalol should be used; ideally, antihypertensives should be used only when the potential effects on cerebral perfusion pressure can be directly measured. Nitroprusside should be avoided because it causes dilation of cerebral vasculature, which can disrupt autoregulation and increase intracranial pressure.

**SUMMARY RECOMMENDATIONS**

The treatment of acute hypertension in patients with traumatic brain injury is not recommended.

Cerebral perfusion pressure in patients with intracranial pressure monitoring should be maintained between 50 and 70 mm Hg. Induced hypertension with intravenous vasopressors to augment cerebral perfusion pressure as an alternative to decreasing intracranial pressure is not recommended.

**Funding and support:** By Annals policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article, that might create any potential conflict of interest. The authors have stated that no such relationships exist. See the Manuscript Submission Agreement in this issue for examples of specific conflicts covered by this statement.

**REFERENCES**