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Critical Care Update

Management of Traumatic Intracranial Hypertension: Old Questions With New Answers

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Traumatic brain injury (TBI) remains a significant source of morbidity and mortality worldwide. With over 10 million TBIs resulting in either mortality or hospitalization annually worldwide and 1.4 million in the United States alone,[1] protocols for the management of TBI and associated intracranial hypertension are vitally important for all medical practitioners who may encounter TBI patients. After the release of the third edition of guidelines for the management of TBI by the Brain Trauma Foundation in 2007,[2] new guidelines for medical and surgical management of elevated intracranial hypertension have been developed. New recommendations have been detailed in the 4th edition,[3] and it covers novel therapies that have been developed as well as revised approaches to existing therapies.

The causes of TBI vary significantly based on patient demographics. For example, among younger patients, motor vehicle collisions and assaults are among the most common causes of injury. In the elderly patient population, falls are the predominant cause of TBI necessitating hospitalization. After the initial injury, a patient remains at risk for further damage to brain tissue from elevated intracranial pressure (ICP) and associated cerebral ischemia. Prompt recognition and management of increased ICP are essential in reducing morbidity and mortality.

Pathophysiology of TBI

A closed head injury is composed of 2 separate but integrally related pathologic processes: the primary injury, with the associated mass effect and neurologic dysfunction, and secondary cerebral edema resulting in increased ICP. The primary injury can result in direct parenchymal injury at the site of impact (coup) or on the contralateral side (countercoup) as the brain shifts within the strict confines of the cranial vault. The regenerative capacity of central nervous system tissue is limited, and the end result of this primary injury is frequently scarring by gliosis. Perhaps more subtle but no less debilitating is the diffuse axonal injury that can occur when significant shear force acts on the delicate axonal projections of brain parenchyma as happens with high-speed collisions.

A secondary injury occurs when edema and dysregulation of cerebral blood flow result in elevated ICP and further cerebral ischemia. Under normal circumstances, ICP is tightly regulated, and perfusion is maintained by vascular autoregulation. In the model described by the Monro-Kellie doctrine, the skull has a fixed volume that is composed of blood, cerebrospinal fluid (CSF), and brain tissue. When the mass effect from intracranial hemorrhage and associated edema is coupled with dysfunctional cerebral blood flow, the compensatory mechanisms of the brain are overwhelmed, and ICP increases exponentially. This results in decreased cerebral perfusion pressure (CPP) equal to mean arterial pressure minus ICP and a further ischemic insult to an already damaged brain. The management of elevated ICP begins with accurate measurement, medical therapies such as hyperosmolar solutions, and, ultimately, decompressive craniectomy if needed.[4,5]

Monitoring of ICP

Monitoring of ICP is required not only to assess cerebral perfusion but also to gauge the need for and response to therapeutic interventions.[2,6] Normal ICP in an adult is < 15 mm Hg, and sustained pressure above 20 mm Hg is detrimental and necessitates treatment. Repeated physical examination, assessment of patient function, and radiographic studies remain the cornerstone of management in a patient with a TBI who is able to participate in neurologic evaluation. However, in a patient with a progressive decline in mental status resulting in loss of consciousness, as often happens in patients with TBI, ICP monitors may guide therapy.

The recommendations put forward by the Brain Trauma Foundation in 2007 suggest that all patients with an abnormal head computed tomographic and clinical signs of severe TBI (Glasgow Coma Scale [GCS] 3-8) should have an ICP monitor placed. Furthermore, ICP monitoring is warranted in patients with severe TBI (GCS $\leq$ 8) in the setting of a normal computed tomographic scan when they have 2 or more of the following criteria: age > 40 years, motor posturing (unilateral or bilateral), or systolic blood pressure (SBP) < 90 mm Hg. In the most recent review released by the Brain Trauma Foundation, the evidence supporting these recommendations has been found to be lacking, and further research is required before definitive guidelines can be recommended. Until that time, however, the preexisting recommendations serve as a reasonable metric on which a practitioner can gauge the need for invasive ICP monitoring.

There are 2 main classes of ICP measuring devices: intraventricular catheters and bolt-type transducers. An intraventricular catheter such as an external ventriculostomy drain can be placed at the bedside. After exposure of an area of the frontal skull at the midpupillary line, a burr hole is created, and the catheter is advanced through the parenchyma until the ventricle is encountered. Various drainage or pressure sensor systems are available, but the systems are designed to allow CSF drainage. The system can be "rezeroed" by setting the outflow at the level of the patient's ear, allowing for a more

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consistent and accurate read of ICP. Risks of placement include malposition, hemorrhage, and infection. The risk of infection increases with the time the drain has been in place, with many studies reporting a significantly increased incidence after 5 to 10 days of external ventriculostomy drain placement.

Bolt-type transducers measure parenchymal pressure, and placement is less invasive when compared with ventriculostomy drains. These fiberoptic monitors are most commonly placed through a burr hole in the right frontal region into the parenchyma. Bolts are calibrated or zeroed at the time of placement, and pressure measurements can become less accurate over time; some studies note a daily drift of 0.5 to 3.2 mm Hg. Parenchymal monitors are equipped to measure local pressures at the site of insertion, and regional differences in compartmental pressures within the brain have been reported, which could lead to inaccurate global pressure assessment. Complication rates from bolts are relatively low, and placement can be easily accomplished at the bedside.

Although invasive ICP monitoring has long been a mainstay of treatment for severe TBI, little in the way of randomized, controlled trials has been done on this topic. The Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure[6] trial showed that TBI patients randomized to receive continuous ICP monitoring and management to keep ICP < 20 mm Hg had no better outcomes when compared with patients managed solely with clinical examination and imaging. This work questions the need for continuous ICP monitoring as a mainstay of treatment. The high mortality and geographic distribution of the study population limit the generalizability of these results, but this work emphasizes highlights that invasive monitoring, with the associated risks of placement and infection, should be used on a limited, goal-oriented basis.

Medical Management of Elevated ICP: General Recommendations

After initial stabilization of a patient with TBI, attention should be turned to optimization of hemodynamic, respiratory, and nutritional parameters during the recovery period. Typically, the cerebral vasculature is able to ensure perfusion over a wide range of mean arterial pressures. This “autoregulation” is disrupted with cerebral injury. Initially, cell death at the site of injury causes release of excitatory amino acids such as aspartate and glutamate. This is coupled with the activation of ionic channels including voltage-gated Ca^{2+} and Na^{2+} channels triggering an injury cascade leading to apoptosis. Taken together, these events cause excitatory oxidative stress, hypoperfusion, and vasospasm, which lead to cerebral ischemia and edema.

It has been well established that hypotension is linked to a higher rate of mortality among patients with TBI.[3] Initial recommendations regarding the target SBP to maintain among patients with TBI have since been revised to age-based goals. For patients 50 to 69 years old, a goal SBP of > 100 mm Hg is recommended, whereas in patients 14 to 49 or > 70 years of age, SBP > 110 mm Hg should be considered. Among patients who have continuous ICP monitoring in place, a CPP goal of 60 to 70 mm Hg has been shown to be associated with improved outcomes. Although there was initial enthusiasm for a higher target CPP, Robertson et al[7] showed that aggressively pursuing a CPP goal > 70 mm Hg resulted in no better neurologic outcomes when compared with a control group with a goal CPP > 50 mm Hg.

With regard to achieving the recommended blood pressure goals, it is equally important to take a judicious approach to the use of isotonic fluids in TBI patients. In a review of recommendations regarding fluid management in patients with neurologic injury, van der Jagt[8] noted that just as lower arterial pressures can decrease cerebral perfusion, elevated central venous pressure can result in venous outflow impedance and lead to an increase in cerebral edema. Consensus guidelines based on existing studies suggest that maintenance of euvolemia with isotonic fluids should be the standard of care among patients with severe TBI. Furthermore, in their study on CPP goals among TBI patients, Robertson et al[7] noted a 5-fold higher rate of acute respiratory distress syndrome in the experimental group in which fluid intake was greater and the net intake versus output balance was more positive when compared with the control ($P < .05$). Prophylactic hypervolemia or resuscitation with large volumes of isotonic fluid confer no significant benefit and have been shown to be detrimental in patients with TBI.

Mechanical ventilation is frequently instituted in patients with TBI because of depressed mental status, inability to protect the airway, or another cause of respiratory failure. Ensuring adequate oxygenation by maintaining O_2 saturation > 95% and PaO_2 > 80 mm Hg is well documented as part of the management of patients with TBI. $PaCO_2$, 1 of the most significant determinants of cerebral blood flow, has received special attention in recent years because of the practice of hyperventilation as a means of managing elevated ICP. This effect results from cerebral vasoconstriction stemming from low $PaCO_2$. More recent studies have shown an increase in cerebral ischemia resulting from hyperventilation, and this practice is no longer advocated outside of transient use for the management of acutely elevated ICP when a patient is > 24 hours out from the time of injury. Current guidelines advocate the maintenance of $PaCO_2$ between 35 and 40 mm Hg.

Therapeutic hypothermia has been endorsed for neuroprotection after cardiac arrest. The initial interest in the use of therapeutic hypothermia in TBI patients has been tempered by risks including coagulopathy, immunosuppression, and induced cardiac dysrhythmia. Although hypothermia has been shown to reduce tissue damage in the central nervous system and decrease ICP, 2 recent trials in pediatric patients have failed to show any outcome benefit. Research is ongoing into whether targeted cerebral cooling (rather than systemic hypothermia) and approaches such as gradual rewarming could offer new therapeutic options for a patient with TBI. At the current time, recommendations emphasize the maintenance of normothermia because the elevated metabolic rate associated with fever can aggravate ischemic injury.

Posttraumatic seizure (PTS) prophylaxis remains a subject of debate and study. PTS is classified as either early, when it occurs within 7 days of injury, or late, when it occurs more than 7 days after injury. Prophylactic use of phenytoin or levetiracetam has been used because there is a relatively high rate of PTS after severe TBI. Sequelae from PTS include the development of chronic epilepsy, acute changes in hemodynamic status, cerebral herniation, and death. At the current time, PTS prophylaxis is not recommended for the prevention of late PTS. Prophylaxis with phenytoin may be justified when the overall benefit is judged to be greater than the complications associated with treatment. Of note, risk factors for early PTS include GCS < 10, immediate seizure after injury, posttraumatic amnesia lasting > 30 minutes, linear or depressed skull fractures, penetrating head injury, any hematoma or contusion, age > 65 years, and chronic alcoholism.

Another support measure in the care of a patient with TBI is early enteral nutrition. A postpyloric feeding tube is recommended to reduce the incidence of regurgitation, aspiration, and ventilator-associated pneumonia. The rate of tube feeding should be adjusted with the goal of attaining patient basal caloric requirements by the 5th day postinjury. Maintaining the head of the bed > 30° helps to avoid aspiration and also aids in cerebral venous drainage, thereby reducing ICP while maintaining CPP.