MANAGING PATIENTS WITH SEVERE

traumatic brain injury

By Devon Lump, MSN, ACNP-BC

IN THE UNITED STATES, an estimated 17 million people sustain a traumatic brain injury (TBI) annually, and about 52,000 die (see A snapshot of TBI).¹ This article discusses primary and secondary brain injuries, nursing assessment and initial treatment, medical and surgical management, postoperative care, and long-term management for patients with severe TBI.

Causes and classifications
The most common causes of TBI are falls, motor vehicle crashes, and violence, including gunshot wounds.¹ TBI can be classified as penetrating or nonpenetrating, as well as focal or diffuse. Focal injuries include contusions and hematomas; diffuse injuries include concussions and diffuse axonal injury (DAI).²

The Department of Defense and the Department of Veterans Affairs define TBI as any traumatically induced structural injury and/or physiologic disruption of brain function as a result of an external force that's indicated by new onset or worsening of at least one of the following clinical signs immediately after the event:
- any period of loss of or decreased level of consciousness
- any loss of memory for events immediately before or after the injury
- any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking)
• any neurologic deficit (including weakness, loss of balance, change in vision or other sensory alterations, paresis or plegia, aphasia) that may or may not be transient, or presence of an intracranial lesion.3

TBI is commonly stratified by severity (mild, moderate, severe) as determined by Glasgow Coma Scale (GCS) score: 13 to 15 is mild, 9 to 12 is moderate, and 3 to 8 is severe TBI.4 (See Reviewing the Glasgow Coma Scale.) The GCS score can be difficult to determine if a patient has received sedatives or paralytics, or is endotracheally intubated.

TBI severity is also measured by degree or duration of loss of consciousness or time spent neurologically impaired. Mild TBI is defined as mental status change or loss of consciousness for less than 30 minutes. In moderate TBI, these symptoms last between 30 minutes and 6 hours, and in severe TBI, they last more than 6 hours.5,6 Under emergency neurologic life-support guidelines, any unconscious patient with an identified mechanism of injury consistent with TBI and a GCS score of less than 9 is considered to have severe TBI.7

Understanding primary injury
Primary brain injury occurs at the time of injury. Penetrating or open head injuries (caused by an object penetrating both skull and brain), direct impact, and rapid acceleration/deceleration are common mechanisms.8 If severe, the primary injury can be immediately fatal.

In a closed head or nonpenetrating injury, the primary injury is the result of direct impact of neuronal tissue against the skull or bone and shearing of neurovascular structures due to rotational injury (see Mechanisms of head injury).9 This type of injury often occurs in motor vehicle crashes when the head hits the dashboard. The high-speed collision and rapid deceleration force the brain to strike the inside of the skull on both sides, causing injury at the site of impact (coup) and 180 degrees from the site of impact (contre-coup).

DAI results from the shearing of axons in the cerebral white matter during acceleration/deceleration head injury.10 (See A closer look at diffuse axonal injury.) Most lesions appear at the interface between the gray and white matter junctions; in its severe form, however, DAI often occurs in the corpus callosum and brain stem. DAI in the brain stem can cause a persistent vegetative state. Often, DAI isn’t accompanied by significant focal findings on a noncontrast head computed tomography (CT) scan; in the absence of focal radiographic lesions, it’s often defined clinically by the rapid progression to coma or severe encephalopathy. DAI often results in
neuropsychological complications and financial burden.11

In an open head or penetrating injury, primary injury is caused by a foreign body, either high velocity (as in a gunshot wound) or lower velocity (as in a stabbing). The penetrating object may tear vascular structures along with brain tissue.10

Neurologic deterioration after a primary brain injury can be explained by the Monro-Kellie hypothesis: the brain, its vascular supply, and the cerebrospinal fluid (CSF) are enclosed in a rigid container (the skull) that can’t expand. The brain accounts for about 80% of cranial contents; blood and CSF account for 10% each.12 Anything that causes an increase in volume of one component (for example, a lesion, intracranial bleeding, or cerebral edema) creates competition for space and can lead to increased intracranial pressure (ICP) and brain herniation.

Understanding secondary injury

In TBI, secondary injury results from various molecular injury mechanisms.8 It may be associated with or exacerbated by neurotransmitter-mediated excitotoxicity, hypoxia, hypotension, ischemia, seizure activity, intracranial hypertension, and fever.8

Secondary brain injury begins immediately following the primary insult and can continue for hours or days. A secondary brain injury can be more difficult to treat and is associated with higher morbidity and mortality than the primary injury itself.

Hypoxia and hypoperfusion are two of the leading contributors to secondary brain injury.8 One study found that patients with severe TBI who developed two or more episodes of hypotension had a two-to-eight times higher risk for death.13 Hypotension within the first 48 hours of ICU admission also has been found predictive of mortality.14 Patients with severe TBI need intensive monitoring in a neurologic ICU so they can receive treatment to reduce the risk of secondary brain injury.

First steps in treatment

Recognizing TBI is the first step to facilitating streamlined, effective, neurologic critical care for the patient, who should be transported to a Level I or Level II trauma center for management, advanced neuroimaging, neuromonitoring, and neurosurgery, if indicated. In the ED, initial management involves determining level of consciousness, protecting the airway, and ensuring adequate oxygenation. Patients with severe TBI typically are comatose on ED arrival and need an advanced airway, such as an endotracheal tube.

Patients with severe TBI should have large-bore peripheral I.V. access and cardiac monitoring, pulse oximetry, and continuous waveform capnography if available. After the airway and hemodynamics are stabilized, the patient should be assessed for other injuries. Often, pan-computed tomography (pan-CT) of the head, cervical spine, chest, abdomen, and pelvis with thoracic and lumbar spine reconstructions is performed quickly.

Immediate surgical intervention for life-threatening hemorrhage may mean that a pan-CT isn’t performed initially; however, injury to the thorax and spine must be ruled out as soon as possible.

A noncontrast head CT scan is the diagnostic tool of choice in severe TBI. An extra-axial hematoma (bleeding within the cranium but outside the brain or cord parenchyma)
greater than 1 cm (0.4 in) in thickness, and an intraparenchymal hematoma greater than 3 cm (1.2 in) in diameter with greater than 5 mm midline shift associated with a hematoma often are considered surgical lesions.\textsuperscript{15}

If the patient doesn’t immediately meet criteria for surgical intervention, an ICP monitor may be considered. Placement guidelines, which vary from institution to institution, are based on clinical assessment findings and GCS score. Brain Trauma Foundation guidelines for the management of severe TBI recommend ICP monitoring in all patients with severe TBI, GCS score below 9, and an abnormal CT scan.\textsuperscript{16}

In adults, ICP is normally less than 10 mm Hg.\textsuperscript{10} For patients with TBI, the standard of care is a target ICP of less than 20 mm Hg; however, an individualized approach may be indicated in some cases.\textsuperscript{10}

Cerebral perfusion pressure (CPP), an estimate of the adequacy of cerebral circulation, is calculated by subtracting ICP from mean arterial pressure (normally between 60 and 100 mm Hg).\textsuperscript{2,10,17} The target CPP range is 60 to 70 mm Hg in brain-injured adults.\textsuperscript{10} CPP of less than 50 mm Hg correlates with poor outcomes, even when that CPP level occurs only periodically.\textsuperscript{18}

The Traumatic Brain Injury Guidelines recommend using ventricular or intraparenchymal catheters for ICP monitoring. Advanced intraparenchymal probes also are available to measure brain temperature, brain tissue oxygen tension (PbtO\textsubscript{2}, normally 25 to 30 mm Hg), and cerebral blood flow.\textsuperscript{19}

Mixed venous oxygen saturation measured via jugular venous bulb oximetry (SjO\textsubscript{2}) is normally 45% to 70%.\textsuperscript{20} The SjO\textsubscript{2} will fall when there is an imbalance between cerebral oxygen consumption and delivery. Studies have shown that maintaining a PbtO\textsubscript{2} of greater than 20 mm Hg (in addition to meeting current ICP and CPP goals) may improve outcomes in patients with severe TBI.\textsuperscript{21}

Medical therapies and the placement of ICP monitoring devices must be performed concurrently. For example, if the neurosurgeon is preparing to insert an intraventricular catheter for ICP monitoring, specimens for lab work must already have been drawn, coagulopathies reversed, and electrolyte abnormalities corrected. If a family member can provide an accurate health history, the patient’s use of antiplatelet agents (including aspirin) and anticoagulation should be investigated.

Noninvasive measures to lower ICP may also be implemented at the bedside. The clinical nurse should ensure that the patient is turned and positioned in proper body alignment, maintaining the neck in neutral position at all times, and elevating the head of the bed to 30 degrees to facilitate cerebral venous drainage once the patient’s spine has been evaluated and stabilized. Avoid lateral flexion of the neck, Trendelenburg or prone position, and extreme hip flexion.\textsuperscript{2}

Hyperventilation may be considered as a bridging or rescue therapy in select cases but shouldn’t be

---

**A closer look at diffuse axonal injury**

In diffuse axonal injury (DAI), acceleration-deceleration and shearing forces damage nerve fibers at the moment of injury. Areas most often affected are the corpus callosum, the dorsolateral area of the midbrain, and the parasagittal white matter.

---

implemented or continued as management of increased ICP. Aggressively lowering PaCO2 decreases cerebral blood flow, leading to ischemia.2

Medical management
Before surgery is considered, medical management is usually attempted in an effort to reduce ICP. Mannitol or hypertonic sodium chloride solution are usually the first-line therapies after pain and agitation have been treated and the patient is in proper body position as described earlier. I.V. administration of hyperosmolar agents, including hypertonic sodium chloride solution and mannitol, creates an osmolar gradient, drawing water across the blood-brain barrier and decreasing interstitial volume.8 This intervention has been shown to decrease ICP and improve CPP in patients with severe TBI.22,23

Patients should be monitored for hypotension and other signs of hypovolemia, especially if they have multiple traumatic injuries, because they may already be volume depleted.21 Hypertonic sodium chloride solution doesn’t cause hypovolemia but it may lead to pulmonary edema.

Both mannitol and hypertonic sodium chloride solution can cause hypernatremia and hypertonicity. Patients receiving hypertonic sodium chloride solution or mannitol require frequent monitoring for serum sodium levels and osmolarity, an indwelling urinary catheter, strict recording of intake and output, and chest X-rays to rule out fluid overload. Continuous arterial BP and central venous pressure monitoring can also help guide management during fluid therapy.

Sedation and analgesia should be considered to help prevent agitation and pain, which increase metabolic demand and ICP.2 Only the minimum amount of medication necessary for the desired effect should be used so an accurate, comprehensive neurologic assessment can be performed.

Sedation and analgesia should be considered to help prevent agitation and pain, which increase metabolic demand and ICP.2 Only the minimum amount of medication necessary for the desired effect should be used so an accurate, comprehensive neurologic assessment can be performed.

Propofol, an I.V. sedative-hypnotic agent, is often used for sedation in adults who are mechanically ventilated in the neurologic ICU. Because it’s rapid acting and has a short half-life, it permits intermittent neurologic assessment. Monitor BP in patients receiving a propofol infusion because the drug can cause significant hypotension.24 Propofol is contraindicated in patients with an allergy to eggs, egg products, soybeans, or soy products.

Similarly, I.V. dexmedetomidine, a relatively selective alpha2-adrenergic agonist, is used for sedation and can help control agitation in mechanically ventilated adults; the drug often permits reliable serial neurologic assessments. Monitor heart rate and BP in patients receiving dexmedetomidine because it can cause significant bradycardia, sinus arrest, and hypotension. Dexmedetomidine has no known contraindications.

Fentanyl, an opioid analgesic used for pain management, may also be administered as an adjunct and as needed for to treat tachycardia, tachypnea, and agitation.23 Monitor respiratory function and heart rate in patients receiving fentanyl because it can cause respiratory depression and bradycardia. Fentanyl is contraindicated in patients with a known intolerance to the drug. Use a pain intensity rating scale designed for nonverbal patients, such as the Richmond Agitation Sedation Scale, to assess the patient’s need for analgesia.23

Surgical management
Patients with a GCS score of 8 or lower who have a large lesion on noncontrast head CT scan are candidates for surgical evacuation of the lesion. Surgery should be expedited if the patient’s neurologic status deteriorates. Surgical repair is indicated for patients with depressed skull fractures that are displaced more than the thickness of the skull table, especially if the fractures are open or complicated.20

In decompressive craniectomy, a portion of the skull is removed to relieve ICP. In essence, decompressive craniectomy provides an exception to the Monro-Kellie hypothesis because it converts the closed compartment of the skull into an open system that lets the brain expand, thereby decreasing ICP and avoiding brain herniation.27,28

Although decompressive craniectomy is the most effective way to reduce ICP, the long-term prognosis for patients is variable.29 One study found a poor prognosis associated with the presence of bilateral or contralateral lesions in patients with TBI who underwent craniectomy.30 Another study concluded that bifrontotemporal decompresive craniectomy decreased ICP and length of ICU stay in patients with severe TBI but was also associated with more unfavorable outcomes.29

Bilateral decompressive craniectomy has been shown to be a favorable treatment in select patients, specifically younger patients with
reductive pupils whose ICP and CPP values are stabilized within 24 hours of undergoing surgery. 

Studies on the long-term prognosis for patients after craniectomy are conflicting, and more research is needed in this area. 

Most would agree that timely decompressive craniectomy before the development of irreversible brain damage is key to a positive patient outcome. ICP, clinical assessment findings, and PbtO2 are the monitoring parameters most helpful in predicting optimal timing for decompressive craniectomy. 

Postoperative care

After surgery, patients are taken to the neurocritical care ICU for monitoring and continued management. Changes in ICP, CSF circulation, cerebral blood flow, and autoregulation continue even after a portion of the skull or bone flap has been removed. Progression of contusions, cerebral edema, and hydrocephalus can still occur and may require intervention. If the patient didn’t have an advanced neuronitoring device placed previously, one can be placed postoperatively to supplement ICP monitoring.

Humidified mechanical ventilation should be optimized to target PaCO2 between 35 and 45 mm Hg, maintain normothermia, optimize CPP, and avoid secondary brain injury. In a recent study, average ICP during the first 48 hours following injury was predictive of mortality and neuropyschological outcomes at 6 months.

Following TBI, the patient’s metabolic demands increase and early nutritional support is critical. Initiation of early enteral feeding (within 72 hours of injury) has been shown to decrease infection rates and overall complications.

Metabolic rate depends on level of consciousness, presence of infection, other injuries, fever, and presence of sympathetic storming (an exaggerated stress response). Signs and symptoms of sympathetic storming include diaphoresis, restlessness, agitation, tachycardia, hyperventilation, and hypertension. Comatose patients with abnormal flexor or extensor posturing have been documented to have higher metabolic demands than comatose patients without abnormal posturing or sympathetic storming events.

Due to the increased metabolic demands of critically ill patients with TBI, resting energy expenditure (REE) may be helpful to accurately determine the patient’s nutritional needs and avoid overfeeding as well as malnutrition. REE is calculated using indirect calorimetry and protein status using urine urea nitrogen.

Patients with severe TBI are at increased risk for deep vein thrombosis (DVT), especially if they’re intubated; up to 25% of patients with TBI develop DVT. Minimize the risk with range-of-motion exercises, intermittent pneumatic compression devices, and pharmacologic prophylaxis as indicated.

Long-term management

After cerebral edema, hydrocephalus, and infection have resolved, cranioplasty can be considered to replace the patient’s bone flap or reconstruct the area with mesh or plastic. Cranioplasty usually isn’t considered until 2 to 6 months after the initial injury. One study found that patients who underwent early cranioplasty (less than 2 months after initial injury) had more postoperative complications than those who waited to have surgery until more than 2 months after the initial injury. The same study also showed that patients with ventriculoperitoneal shunts had a higher rate of device-related complications.

Aside from the obvious need for skull reconstruction, patients with severe TBI who’ve had decompressive craniectomy also need neuro-psychological, physical, speech, and occupational therapy. Inpatient rehabilitation provides the ideal transition once the patient’s critical care needs (ICP and hemodynamic management, infection control, and weaning from mechanical ventilation) have been met. Patients often need weeks to months of TBI rehabilitation followed by outpatient therapy.

By understanding TBI and how to reduce the risk of secondary brain injury, you can help your patient survive TBI with the best possible outcome.

REFERENCES


Devon Lump is a neurosurgery nurse practitioner at departments of neurosurgery and neurocritical care at the University of Pennsylvania in Philadelphia, Pa.