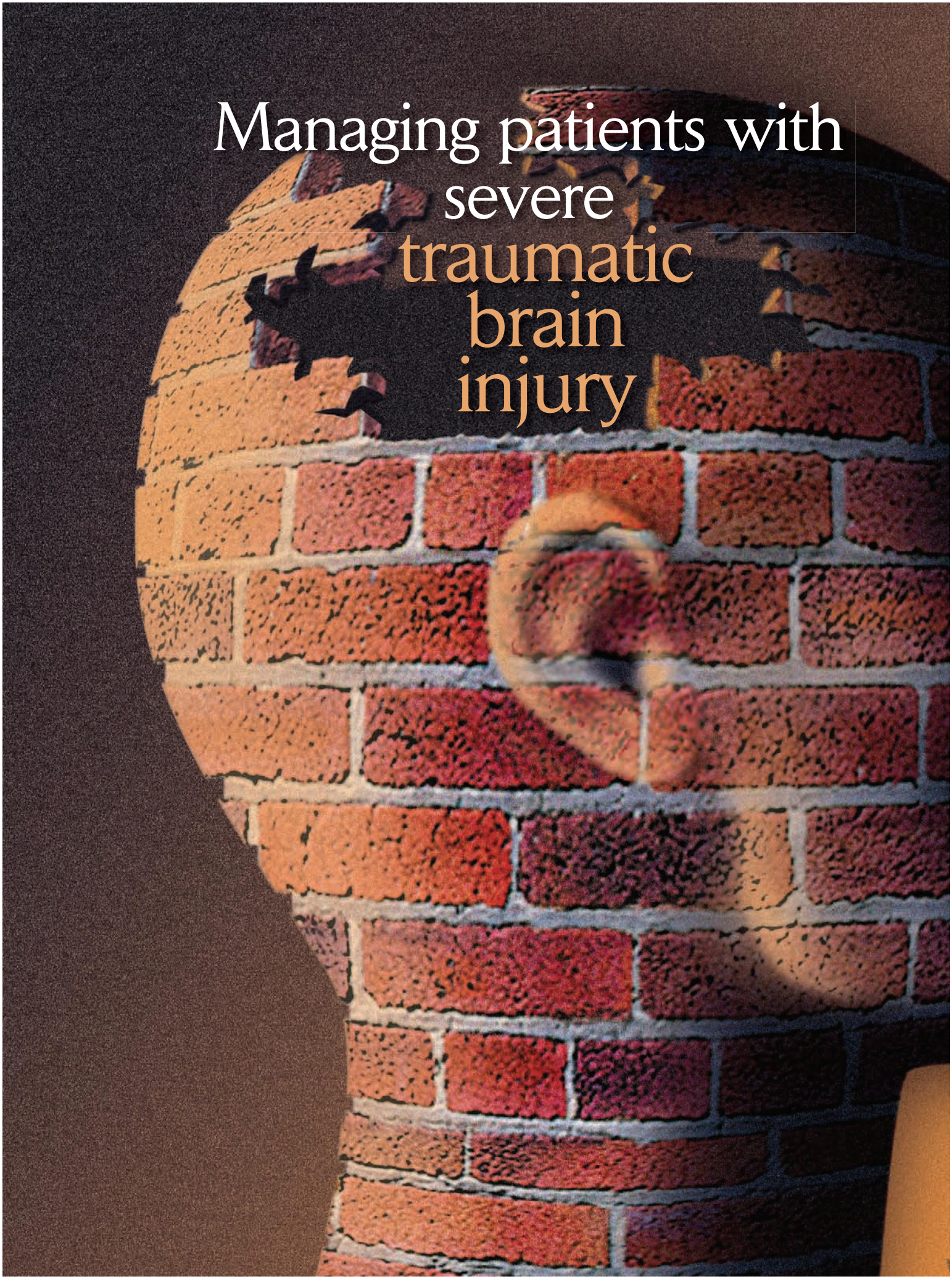
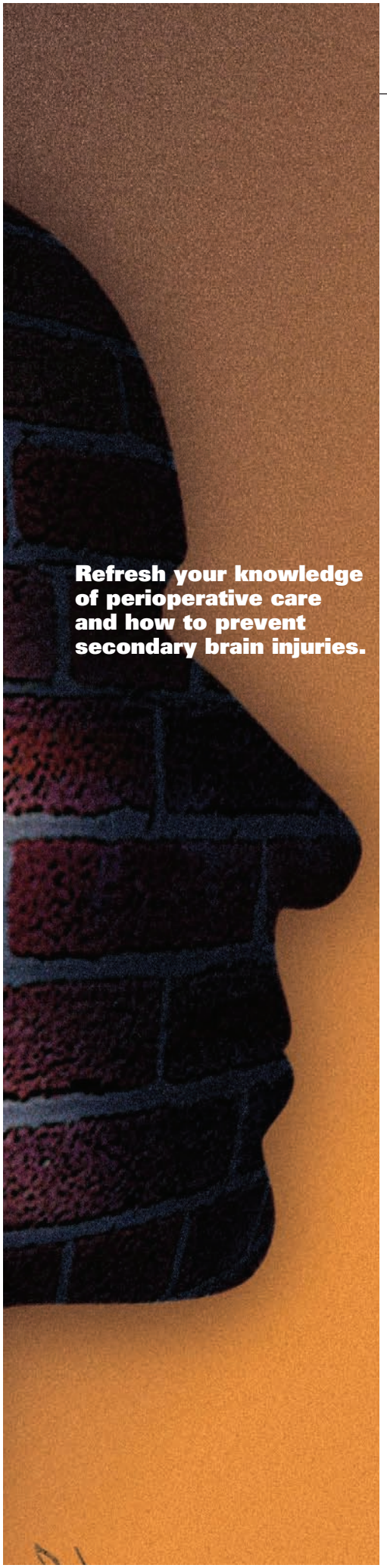
Managing patients with severe traumatic brain injury





Refresh your knowledge of perioperative care and how to prevent secondary brain injuries.

CE
2.3
ANCC CONTACT HOURS

By Devon Lump, MSN, ACNP-BC

Traumatic brain injury (TBI) is the leading cause of death in North America for patients between the ages of 1 and 45 (see *A snapshot of TBI*).[1] The most common causes of TBI are motor vehicle accidents, falls, and violence, including gunshot wounds. TBI can be classified by mechanism (penetrating versus nonpenetrating) or location (in a focal or specific location, or a more diffuse process such as traumatic subarachnoid bleeding). One of the more common ways to stratify TBI is by severity (mild, moderate, severe) as determined by Glasgow Coma Scale (GCS) score.

The departments of Defense and Veterans Affairs define TBI in a more comprehensive way, as a traumatically induced structural injury and/or physiologic disruption of brain function as a result of an external force, 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 a 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 deficits (weakness, loss of balance, change in vision, praxis, paresis or plegia, sensory loss, aphasia) that may or may not be transient, or presence of an intracranial lesion.[2]

Severity of TBI is determined by GCS score: 13 to 15 is mild, 9 to 12 is moderate, and 8 or less is severe TBI.[3] GCS score can be difficult to determine if a patient has received sedatives, paralytics, or is endotracheally intubated (see *The Glasgow Coma Scale*). 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.[4,5] Under emergency

neurologic life-support guidelines, any unconscious patient with an identified mechanism consistent with TBI and a GCS score of less than 9 is considered to have severe TBI.[6]

Understanding primary injury

If severe, the primary injury in TBI can result in immediate death. In a *closed head injury,* 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*).[7] This type of injury often occurs in motor vehicle accidents. The high-speed collision and rapid deceleration force the brain to strike both sides of the inside of the skull, causing injury at the site of impact (coup) and 180 degrees from the site of impact (contracoup).

Diffuse axonal injury (DAI) occurs as a result of the shearing of axons in the cerebral white matter during acceleration/deceleration head injury.[8] Most lesions are seen at the interface between the gray and white matter junctions; in its severe form, DAI often occurs in the corpus callosum and brain stem. DAI in the brain stem can cause a persistent vegetative state.

A snapshot of TBI

Many survivors of TBI live with significant disabilities including cognitive deficits, motor and sensory abnormalities, receptive and expressive language difficulties, visual disturbances, and ventilator dependence necessitating tracheostomy and gastrostomy. The need for support services at home, inpatient rehabilitation or long-term care, and ongoing medical treatment and follow-up translates into a major socioeconomic burden. In 2000, the economic effect of TBI in the United States was estimated to be $9.2 billion in lifetime medical costs and $51.2 billion in productivity losses.[1]

Many patients with mild TBI are treated in the ED and discharged. Moderate-to-severe TBI only accounts for about 20% of all TBI. However, the incidence of severe TBI is over three times higher for men than for women and more frequently affects patients of lower socioeconomic status. Falls are the leading cause of TBI in adults over age 65, followed by motor vehicle accidents and violence. Some 52,000 deaths occur each year as a result of TBI, and an estimated 5.3 million Americans (or 2% of the population) are living with disabilities from TBI.[7]

Often, DAI isn't accompanied by significant focal findings on a noncontrast head computed tomography (CT) scan; because of this, it's often defined clinically by the rapid progression to coma or severe encephalopathy in the absence of focal radiographic lesions.[9] DAI often results in significant morbidity as well as neuropsychological complications and financial burden.

In a *penetrating head 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 in addition to brain tissue.

Understanding secondary injury

Neurologic deterioration after a primary brain injury can be explained by the Monro-Kellie Doctrine: 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 the cranial contents and blood and CSF account for 10% each.[10] 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 (see *The normal brain and the herniated brain*).

Secondary injury may also cause impaired autoregulation of cerebral blood flow, cellular inflammatory responses, and excitotoxicity from overactivation of biochemical receptors. Factors associated with secondary injury include hypoxia, hypotension, ischemia, seizure activity, intracranial hypertension, and fever.

Secondary injury begins immediately following the primary insult and can continue for a prolonged period of time. Often, secondary injury is more difficult to treat and carries a higher rate of morbidity and mortality than the primary injury itself. Hypoxia and hypoperfusion are two of the leading contributing factors to secondary brain injury.[7] One study found that patients with severe TBI who developed two or more episodes of hypotension had an increased odds ratios of two to eight times higher risk for death.[11] Hypotension within the first 48 hours of ICU admission also has been found predictive of mortality.[12]

Patients with severe TBI need intensive monitoring in a neurocritical care ICU, so steps can be taken to reduce the risk and progression of secondary brain injury.

The Glasgow Coma Scale

The range of possible scores is 3 to 15. A score of 15 indicates that the patient is fully alert and oriented, and a score of 3 indicates a complete lack of responsiveness. A score of 3 to 8 suggests severe impairment (associated with coma), 9 to 12 suggests moderate impairment, and 13 to 15 suggests mild impairment.

		Score
Best eye-opening response	• Spontaneously	4
	• To speech	3
	• To pain	2
	• No response	1
Best verbal response	• Oriented	5
	• Confused conversation	4
	• Inappropriate words	3
	• Garbled sounds	2
	• No response	1
Best motor response	• Obeys commands	6
	• Localizes stimuli	5
	• Withdrawal from stimulus	4
	• Abnormal flexion (decorticate)	3
	• Abnormal extension (decerebrate)	2
	• No response	1

Source: Morton PG, Fontaine DK. *Critical Care Nursing: A Holistic Approach.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:725.

First steps in treatment

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

Patients with severe TBI should have large-bore peripheral I.V. access and should be monitored via cardiac telemetry, pulse oximetry, and continuous end-tidal carbon dioxide monitoring if available. After the patient's airway and hemodynamics are stabilized, they should be assessed for other injuries. Often, a pan-CT scan (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, bleeding and injury to the thorax, torso, 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 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.[13] If the patient doesn't immediately meet criteria for surgical intervention, an ICP monitor may be considered. Placement guidelines vary from institution to institution but are based on clinical exam findings and GCS score. Brain Trauma Foundation guidelines for the management of severe TBI recommend that ICP be monitored in all patients with severe TBI, GCS score below 9, and an abnormal CT scan.[14] (Normal ICP should be less than 10 mm Hg for adults.)[8] ICP monitoring may be accomplished with an external ventricular drain or by an intraparencymal probe. Advanced intraparencymal probes also are available to measure brain temperature, brain tissue oxygen tension ($PbtO_2$, normally 25 to 30 mm Hg), and cerebral blood flow.[15]

Once you have an ICP measurement, you can derive cerebral perfusion pressure (CPP) by subtracting ICP from mean arterial pressure (normally between 60 and 100 mm Hg).[8,16] Normal CPP is

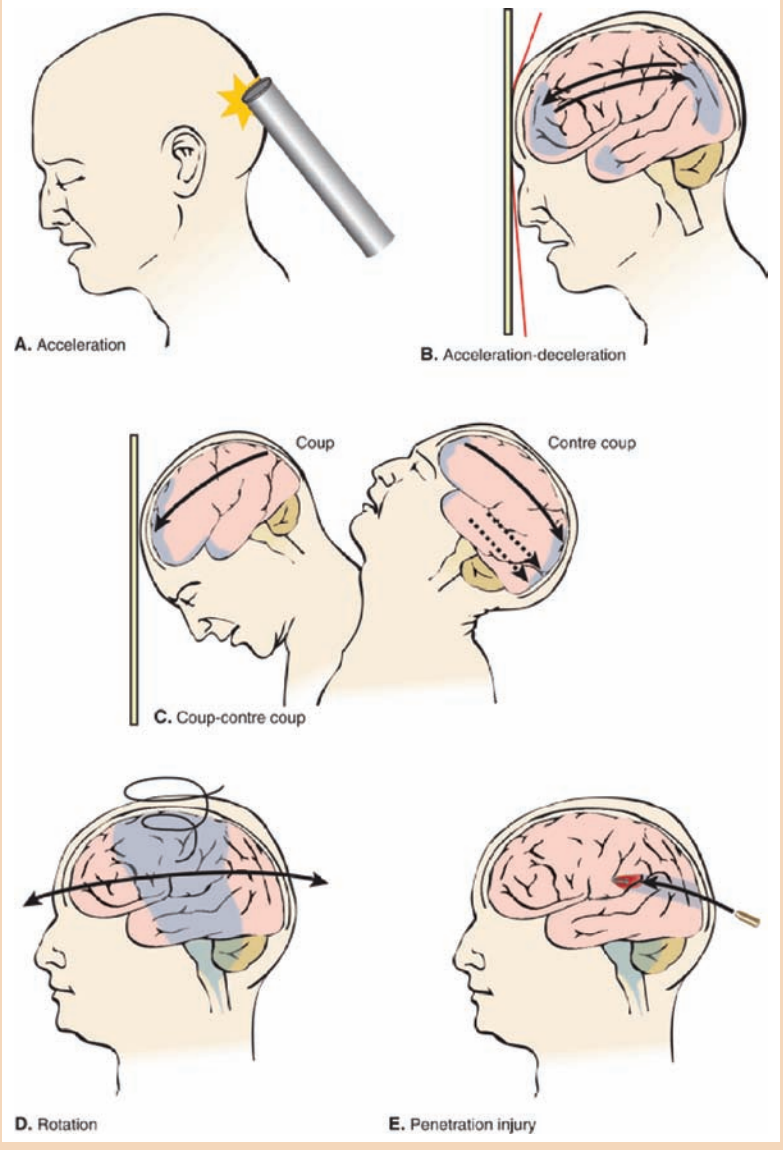
50 mm Hg, but is targeted at 60 to 70 mm Hg in brain-injured patients.[8] Traditionally, the standard of care is a target ICP of less than 20 mm Hg; however, an individualized approach may be indicated in some patients.[8] CPP of less than 50 mm Hg is correlated with poor outcomes even when that CPP level only occurs periodically.[17] A brain tissue oxygen monitor or jugular oximetry monitor (SjO_2) measures brain oxygenation, and is normally 45% to 70%.[18] The jugular bulb catheter measures diffuse, global brain oxygenation. The brain tissue oxygen monitor measures focal brain oxygenation and is used as a marker for cerebral hypoxia. Studies have shown that a $PbtO_2$ of greater than 20 mm Hg (in addition to current ICP and CPP goals) may improve outcomes in patients with severe TBI.[19]

Medical therapies and the placement of ICP monitors must be carried out in a simultaneous fashion. If the neurosurgeon is setting up to place a brain tissue oxygen monitor, labs must already have been drawn, coagulopathies reversed, and electrolyte abnormalities corrected. If a family member can provide an accurate medical history, aspirin, antiplatelet agents, and anticoagulant therapy should be investigated.

Noninvasive measures to lower ICP may also be implemented at the bedside. The direct care nurse should ensure that the patient is in good spinal alignment from head to toe, straightening the neck and raising the head of the bed to 30 degrees once spinal precautions are lifted. Hyperventilation may be considered as a bridging therapy but shouldn't be implemented or continued as management of increased ICP. A target $PaCO_2$ of 28 to 35 mm Hg has been demonstrated to decrease ICP.[16]

Usually before surgery is considered, medical management is 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 the

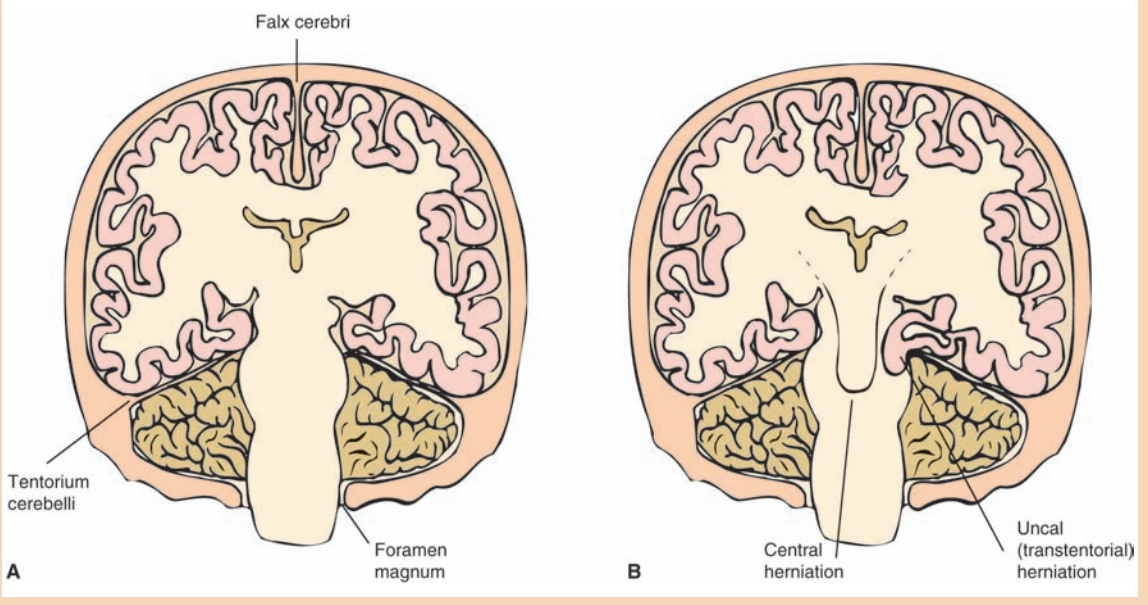
Mechanisms of head injury



Source: Morton PG, Fontaine DK. *Critical Care Nursing: A Holistic Approach.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:807.

The normal brain and the herniated brain

Illustration A shows the normal brain; B shows a herniated brain. Herniation associated with brainstem compression is called central herniation. Herniation associated with the supratentorial structures is called uncal (or transtentorial) herniation.



Source: Morton PG, Fontaine DK. *Critical Care Nursing: A Holistic Approach.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:811.

appropriate position (neck straight, head of bed 30 degrees). Hypertonic sodium chloride solution and mannitol have been shown to decrease ICP and improve CPP in patients with severe TBI.[20,21] Patients should be monitored for hypovolemia and hypotension, especially if they have multiple traumas and may already be volume depleted.[19] Hypertonic sodium chloride solution doesn't cause hypovolemia but may lead to pulmonary edema.

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

Sedation and analgesia should be considered, and may assist in managing increased ICP. The patient requires respiratory support and must be intubated and mechanically ventilated when receiving sedation. Only the minimum medication necessary for desired effect should be used, so that the patient can still respond to a neurologic exam. Propofol, an I.V. sedative-hypnotic anesthetic agent, is often used for sedation in adult patients who are mechanically ventilated in the neurocritical care ICU. It's rapid-acting and has a short-half life that allows for pauses in therapy during which the neurologic assessment can be done. Monitor BP in patients receiving a propofol infusion because the drug can cause significant hypotension.[22]

Propofol is contraindicated in patients with an allergy to eggs, soybean, or soy or egg products. Similarly, dexmedetomidine, an I.V. selective alpha$_2$-adrenergic agonist, is used for sedation and can help control agitation in mechanically ventilated adults; the drug often permits reliable serial neurologic exams. Monitor heart rate and BP in patients receiving dexmedetomidine because the drug can cause significant bradycardia, sinus arrest, and hypotension. Dexmedetomidine has no known contraindications.

Fentanyl is an opioid analgesic used for pain management and may also be administered as an adjunct and as needed for signs of 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.[21]

Up to 25% of patients with TBI develop DVT. Intubation increases the risk.

Surgical management

Patients with a GCS score of 8 and 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.[24]

Decompressive craniectomy, in essence, provides an exception to the Monro-Kellie doctrine because it converts the closed compartment of the skull into an open system that lets the brain expand, thereby avoiding herniation. After bone removal, brain compliance increases.[25] Although decompressive craniectomy is the most effective way to reduce ICP, the long-term prognosis for patients is variable.[26] One study concluded that the presence of bilateral or contralateral lesions in patients with TBI who underwent craniectomy carries a poor prognosis.[27] Another study concluded that bifrontotemporoparietal decompressive craniectomy decreased ICP and length of ICU stay in patients with severe TBI but was also associated with more unfavorable outcomes. Bilateral decompressive craniectomy has been shown to be a favorable treatment in select patients, specifically younger patients with reactive pupils, whose ICP and CPP values are stabilized within 24 hours of undergoing surgery.[27] The usefulness of bifrontal decompressive craniectomy is still controversial; however, in recent studies, it seems that division of the falx at the floor of the anterior cranial fossa in combination with removal of the skull is recommended in an effort to maximize decompressive effect.[28] Studies on the long-term prognosis for patients after craniectomy are conflicting, and more research is needed in this area.[29,30]

Most would agree that timely decompressive craniectomy, before the development of irreversible brain damage, is key to a positive patient outcome. ICP, clinical exam, and $PbtO_2$ are the monitoring parameters most helpful in predicting optimal timing for decompressive craniectomy.[31]

A large craniectomy with large dural opening is required for adequate decompression. The size of the bone flap should be about 12 cm (4.7 inches) (anterior-posterior) by 9 cm (3.5 inches) (superior-inferior), in addition to duraplasty.[32] Often a subgaleal drain is left in place to allow for continued drainage of blood that may accumulate in the subgaleal space postoperatively. Drain placement was statistically significant in one study as a protective factor against postoperative complications.[33] Medical management is continued intraoperatively and postoperatively to minimize elevations in ICP.

Postoperative care

After surgery, patients are taken to the neurocritical care ICU for monitoring. Changes in ICP, CSF circulation, cerebral blood flow, and autoregulation continue even after a portion of the skull or bone flap has been removed. Contusion blossoming, brain edema, and hydrocephalus can still occur and may require intervention.[31] Medical management is continued, and if the patient didn't have a brain tissue monitor, one can be placed postoperatively to monitor ICP, CPP, and $PbtO_2$.

Humidified mechanical ventilation should be optimized to target $PaCO_2$ 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 neuropsychological outcomes at 6 months.[34]

Following TBI, the patient's metabolic demands are increased, and early nutritional support is critical in ensuring optimal patient outcomes. Initiating

early enteral feeding (within 72 hours of injury) has been shown to decrease infection rates and overall complications.[35] Metabolic rate depends on level of consciousness, presence of infection, other injuries, fever, and presence of autonomic storming. (Autonomic or sympathetic storming is the result of a sympathetic and parasympathetic nervous system imbalance. Signs and symptoms include diaphoresis, restlessness, agitation, tachycardia, hyperventilation, and posturing.) Comatose patients with posturing responses (flexor and extensor) have been documented to have higher metabolic demands than comatose patients without posturing or autonomic storming events.[36]

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.[37] An REE is performed by a respiratory therapist and a clinical nutritionist.

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).[38] Take preventive steps, including range-of-motion exercises, sequential compression devices, and prophylaxis with subcutaneous unfractionated heparin or low-molecular-weight heparin.

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 patients who waited to have surgery until more than 2 months after the initial injury.[39] The study also showed that patients with ventriculoperitoneal shunts had a higher rate of device-related complications.[39]

Aside from the obvious need for skull reconstruction, patients with severe TBI who've had decompressive craniectomy also need neuropsychological, 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 ventilator weaning) 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 have a positive outcome. **OR**

REFERENCES

1. Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. *J Head Trauma Rehabil.* 2006;21(6):544-548.

2. Department of Defense and Department of Veterans Affairs. Traumatic Brain Injury Task Force. DOD/VA common definition of TBI. 2008. http://www.cdc.gov/nchs/data/icd9/Sep08TBI.pdf.

3. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet.* 1974;2(7872):81-84.

4. Greenwald RM, Gwin JT, Chu JJ, Crisco JJ. Head impact severity measures for evaluating mild traumatic brain injury risk exposure. *Neurosurgery.* 2008;62(4):789-798.

5. Andriessen TM, Horn J, Franschman G, et al. Epidemiology, severity classification, and outcome of moderate and severe traumatic brain injury: a prospective multicenter study. *J Neurotrauma.* 2011;28(10):2019-2031.

6. Swadron SP, LeRoux P, Smith WS, Weingart SD. Emergency neurological life support: traumatic brain injury. *Neurocrit Care.* 2012;17(suppl 1):S112-S121.

7. Ling GS, Marshall SA. Management of traumatic brain injury in the intensive care unit. *Neurol Clin.* 2008;26(2):409-426.

8. Greenberg MS. *Handbook of Neurosurgery.* 6th ed. Tampa, FL: Thieme; 2006:649-660.

9. Li XY, Feng DF. Diffuse axonal injury: novel insights into detection and treatment. *J Clin Neurosci.* 2009;16(5):614-619.

10. Mangat H. Severe traumatic brain injury. *Crti Care Neurolog.* 2012;18(3):532-546.

11. Manley G, Knudson MM, Morabito D, Damron S, Erickson V, Pitts L. Hypotension, hypoxia, and head injury: frequency, duration, and consequences. *Arch Surg.* 2001;136(10):1118-1123.

12. Brenner M, Stein DM, Hu PF, Aarabi B, Sheth K, Scalea TM. Traditional systolic blood pressure targets underestimate hypotension-induced secondary brain injury. *J Trauma Acute Care Surg.* 2012;72(5):1135-1139.

13. Simard JM, Sahuquillo J, Sheth KN, Kahle KT, Walcott BP. Managing malignant cerebral infarction. *Curr Treat Options Neurol.* 2011;13(2):217-229.

14. Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma.* 2007;24(suppl 1):S1-S106.

15. Martini RP, Deem S, Treggiari MM. Targeting brain tissue oxygenation in traumatic brain injury. *Respir Care.* 2013;58(1):162-169.

16. Nakagawa K, Smith WS. Evaluation and management of increased intracranial pressure. *Continuum (Minneap Minn).* 2011;17(5 Neurologic Consultation in the Hospital):1077-1093.

17. Stein DM, Hu PF, Brenner M, et al. Brief episodes of intracranial hypertension and cerebral hypoperfusion are associated with poor functional outcome after severe traumatic brain injury. *J Trauma.* 2011;71(2):364-373.

18. Chieregato A, Calzolari F, Trasforini G, Targa L, Latronico N. Normal jugular bulb oxygen saturation. *J Neurol Neurosurg Psychiatry.* 2003;74(6):784-786.

19. Spiotta AM, Stiefel MF, Gracias VH, et al. Brain tissue oxygen-directed management and outcome in patients with severe traumatic brain injury. *J Neurosurg.* 2010;113(3):571-580.

20. Rockswold GL, Solid CA, Paredes-Andrade E, Rockswold SB, Jancik JT, Quickel RR. Hypertonic saline and its effect on intracranial pressure, cerebral perfusion pressure, and brain tissue oxygen. *Neurosurgery.* 2009; 65(6):1035-1041.

21. Oddo M, Levine JM, Frangos S, et al. Effect of mannitol and hypertonic saline on cerebral oxygenation in patients with severe traumatic brain injury and refractory intracranial hypertension. *J Neurol Neurosurg Psychiatry.* 2009;80(8):916-920.

22. Bahouth MN, Yarbrough KL. Patient management: nervous system. In: Morton PG, Fontaine DK, eds. *Critical Care Nursing: A Holistic Approach.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013:744-761.

23. Roberts DJ, Hall RI, Kramer AH, Robertson HL, Gallagher CN, Zygun DA. Sedation for critically ill adults with severe traumatic brain injury: a systematic review of randomized controlled trials. *Crit Care Med.* 2011;39(12):2743-2751.

24. Sessler CN, Gosnell MS, Grap MJ, et al. The Richmond Agitation-Sedation Scale: validity and reliability in adult intensive care unit patients. *Am J Respir Crit Care Med.* 2002;166(10):1338-1344.

25. Plesnila N. Decompression craniectomy after traumatic brain injury: recent experimental results. *Prog Brain Res.* 2007;161:393-400.

26. Cooper DJ, Rosenfeld JV, Murray L, et al. Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med.* 2011;364(16):1493-1502.

27. Yatsushige H, Takasato Y, Masaoka H, et al. Prognosis for severe traumatic brain injury patients treated with bilateral decompressive craniectomy. *Acta Neurochir Suppl.* 2010;106:265-270.

28. Huang X, Wen L. Technical considerations in decompressive craniectomy in the treatment of traumatic brain injury. *Int J Med Sciences.* 2010;7(6):385-390.

29. Wen L, Wang H, Wang F, et al. A prospective study of early versus late craniectomy after traumatic brain injury. *Brain Inj.* 2011; 25(13-14):1318-1324.

30. Rubiano AM, Villarreal W, Hakim EJ, et al. Early decompressive craniectomy for neurotrauma: an institutional experience. *Ulus Travma Acil Cerrahi Derg.* 2009;15(1):28-38.

31. Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. *Neurosurg Focus.* 2009;26(6):E7.

32. Yao Y, Mao Y, Zhou L. Decompressive craniectomy for massive cerebral infarction with enlarged cruciate duraplasty. *Acta Neurochir (Wien).* 2007;149(12):1219-1221.

33. Chang V, Hartzfeld P, Langlois M, Mahmood A, Seyfried D. Outcomes of cranial repair after craniectomy. *J Neurosurg.* 2010;112(5):1120-1124.

34. Badri S, Chen J, Barber J, et al. Mortality and long-term functional outcome associated with intracranial pressure after traumatic brain injury. *Intensive Care Med.* 2012;38(11):1800-1809.

35. Agarwal R, Aloupis M, Compher C, et al. Selection and initiation of specialized nutrition support therapy for the University of Pennsylvania Health System: recommendations of the UPHS Nutrition Task Force. Philadelphia, PA: University of Pennsylvania; 2008.

36. Rajpal V, Johnston J. Nutritional management of traumatic brain injury patients. *Support Line.* 2009;31(1):10-19.

37. Vizzini A, Aranda-Michel J. Nutritional support in head injury. *Nutrition.* 2011;27(2):129-132.

38. Reiff DA, Haricharan RN, Bullington NM, Griffin RL, McGwin G Jr, Rue LW 3rd. Traumatic brain injury is associated with the development of deep vein thrombosis independent of pharmacological prophylaxis. *J Trauma.* 2009;66(5):1436-1440.

39. Schuss P, Vatter H, Marquardt G, et al. Cranioplasty after decompressive craniectomy: the effect of timing on postoperative complications. *J Neurotrauma.* 2012;29(6):1090-1095.

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

The author and planners have disclosed that they have no financial relationships related to this article.

DOI-10.1097/01.ORN.0000429401.25469.58

INSTRUCTIONS

Managing patients with severe traumatic brain injury

TEST INSTRUCTIONS

- To take the test online, go to our secure Web site at http://www.nursingcenter.com/ORnurse.
- On the print form, record your answers in the test answer section of the CE enrollment form on page 31. Each question has only one correct answer. You may make copies of these forms.
- Complete the registration information and course evaluation. Mail the completed form and registration fee of $21.95 to: Lippincott Williams & Wilkins, CE Group, 74 Brick Blvd., Bldg. 4 Suite 206, Brick, NJ 08723. We will mail your certificate in 4 to 6 weeks. For faster service, include a fax number and we will fax your certificate within 2 business days of receiving your enrollment form.
- You will receive your CE certificate of earned contact hours and an answer key to review your results.There is no minimum passing grade.
- Registration deadline is June 30, 2015.

DISCOUNTS and CUSTOMER SERVICE

- Send two or more tests in any nursing journal published by Lippincott Williams & Wilkins together and deduct $0.95 from the price of each test.
- We also offer CE accounts for hospitals and other health care facilities on nursingcenter.com. Call 1-800-787-8985 for details.

PROVIDER ACCREDITATION

Lippincott Williams & Wilkins, publisher of *ORNurse2012* journal, will award 2.3 contact hours for this continuing nursing education activity. Lippincott Williams & Wilkins is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

Lippincott Williams & Wilkins is also an approved provider of continuing nursing education by the District of Columbia and Florida #FBN2454. This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.3 contact hours.

Your certificate is valid in all states.

The ANCC's accreditation status of Lippincott Williams & Wilkins Department of Continuing Education refers only to its continuing nursing educational activities and does not imply Commission on Accreditation approval or endorsement of any commercial product.