

# Chapter 10

## HEAD AND NECK TRAUMA

RYAN KENEALLY, MD\*; MICHAEL SHIGEMASA, MD†; AND ARTHUR R. MIELKE, MD, MPH‡

---

### INTRODUCTION

### CERVICAL SPINE INJURIES

- Epidemiology and Injury Patterns
- Airway Management
- Radiologic Assessment
- Steroids

### HEAD TRAUMA

- Assessment and Monitoring
- Management

### SUMMARY

\*Lieutenant Colonel, Medical Corps, US Army; Assistant Professor, Department of Anesthesiology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, Maryland 20814

†Captain, Medical Corps, US Army; Anesthesiologist, Department of Anesthesia, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Building 9, Bethesda, Maryland 20889

‡Captain, Medical Corps, US Army; Physician, Department of Anesthesia, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Building 9, Bethesda, Maryland 20889

## INTRODUCTION

Injury to the head and neck has always been a major cause of morbidity in military casualties. The principles for dealing with these injuries were defined in the work of Major Harvey Cushing during World

War II. Close attention to the principles of resuscitation combined with advances in surgical and intensive care have markedly improved the outcome from neurological combat injury.

## CERVICAL SPINE INJURIES

### Epidemiology and Injury Patterns

Cervical spine injuries (CSIs), which include bony and ligamentous injuries to the spine and spinal cord injuries with or without a detected fracture, are a common complication of acute traumatic events. CSI sometimes occurs with other major injuries, but other major injuries nearly always accompany CSI. The estimates of cervical spine trauma or spinal cord injury in civilian trauma patients has ranged from less than 0.41%<sup>1</sup> to 4.3%<sup>2</sup> to 6%,<sup>3</sup> and the incidence of CSI varies with mechanism of injury. Rhee et al found a three-fold higher risk of a CSI after gunshot wounds compared to blunt trauma, and a 3.5-fold lower risk of a CSI among stab-wound victims compared to blunt-trauma victims.<sup>1</sup>

Victims of direct injuries to the neck carry a higher risk for a CSI. One study found a 12.1% rate of CSI after a cervical gunshot wound, and a 1.5% rate of CSI after a cervical stab wound.<sup>4</sup> An earlier study found a similar rate for CSI among patients with penetrating neck trauma.<sup>5</sup> Data from combat-wounded service members in Iraq and Afghanistan suggests CSI is common with penetrating neck injuries sustained in combat. Just over 22% of 90 patients with penetrating neck injuries seen at a British combat hospital were found to have CSI.<sup>6</sup> A US study found a 30% rate of CSI after patients sustained penetrating neck wounds. This evidence suggests combat-related penetrating neck trauma may be complex and yield a significant incidence of CSI.

Certain injury patterns, such as facial injuries, are associated with CSI. Among all facial injuries, there was a 6.7% incidence of CSI in a retrospective study,<sup>7</sup> and the incidence appears to increase with greater facial injury severity.<sup>8</sup> Among patients with head injuries, there was a 7% rate of CSI.<sup>7</sup> Patients with known CSI are also at high risk for concomitant injuries. CSI patients had a 13.5% incidence of facial injuries and a 40% incidence of head or brain injuries<sup>7</sup>; however, CSI should be considered in all trauma patients. Similarly, patients with known CSI should be considered high risk for coexisting head and facial injuries.

The most common site of a cervical spine fracture is C2 (24% of fractures), and the most common region

for a cervical spine fracture is the lower cervical region (39% at C6 or C7).<sup>9</sup> The vertebral body is the most common location of a fracture. Injury to the spinal cord can result from fractures or ligamentous disruption. Axial-load injuries are seen in diving accidents among the civilian population, but may also occur in the combat environment; a C1 fracture (Jefferson burst fracture) can result from an axial load and may cause neurologic damage directly or as a result of damage to the vertebral arterial system. Flexion injuries can lead to anterior cord syndrome, which may result in quadriplegia and the loss of pain and temperature sensation. Extension injuries can also occur from rapid deceleration injuries. Neurologic assessment should focus on motor and sensory function to determine a deficit and the level of injury.

### Airway Management

For patients with known or possible CSI, provide a patent and secure airway and avoid the secondary insult that can result from hypoxia or hypoperfusion; minimize or prevent further neurological insult that can result from mechanical forces. Airway support may be needed before CSI can be assessed or ruled out. If a patient must be intubated prior to a full cervical spine evaluation, providers should proceed using manual inline stabilization (MILS) and rapid-sequence induction if successful intubation is anticipated. If intubation attempts will be predictably unsuccessful based on standard airway evaluation criteria, other options should be employed. MILS is preferable to traction for minimizing cervical motion during intubation<sup>10</sup>; it is also an effective technique for minimizing cervical spine motion while attempting intubation compared to the use of a soft or rigid collar.<sup>11</sup> The best method to immobilize an adult's cervical spine is with the use of a rigid cervical collar, sand bags, and restraining tape holding the patient's head to a spine board.<sup>12</sup> Unfortunately, the use of maximal immobilization techniques makes intubation more difficult. Full immobilization leads to a worsened grade of view of the vocal cords with direct laryngoscopy. One study found a 64% incidence of a grade III or worse view, compared to a 22% grade III or worse view with MILS.<sup>13</sup> The use of a

rigid cervical collar also decreases mouth opening.<sup>14</sup> The best-practice recommendation is to use MILS and a rigid cervical collar with the front portion removed to allow for greater mouth opening.

No significant difference in cervical spine motion has been found when comparing the various blades used for direct laryngoscopy in cadaveric models or in live patients without cervical pathology.<sup>15,16</sup> Several comparisons of video laryngoscopes to direct laryngoscopy tools have shown some degree of superiority of the video laryngoscopes. Use of the Bullard laryngoscope (Circon ACMI, Stamford CT) decreases cervical spine motion and improves vocal-cord visualization compared to direct laryngoscopy<sup>17,18</sup> and, although the Bullard laryngoscope was associated with a longer time to successful intubation, the mean time was less than 30 seconds.<sup>19</sup> Similar results were found for the Glidescope (Verathon Inc, Bothell, WA) when compared to direct laryngoscopy.<sup>20,21</sup>

Awake fiberoptic intubation for patients with a possible or known CSI is common. In cadavers with induced cervical instability, tracheal intubation using a flexible fiberoptic bronchoscope caused the least cervical spinal canal distortion of all techniques used.<sup>22</sup> The disadvantage of intubating a patient's trachea using a flexible fiberoptic bronchoscope prior to inducing anesthesia is that it can be time- and resource-consuming and it requires patient cooperation and operator experience. There is no living human data supporting the theory that an awake technique with a flexible bronchoscope will lead to lower rates of secondary CSI, but it is standard practice when time and patient condition allow.<sup>23</sup>

In emergent intubations, awake intubation with a flexible fiberoptic bronchoscope may not be feasible. In this situation, providers must determine if the patient can be successfully intubated, then MILS should be employed with an apneic technique for laryngoscopy and intubation. When ventilation or intubation difficulty is anticipated, awake fiberoptic bronchoscopy or awake tracheostomy can be performed. In one study, tracheostomy in a cadaveric model of cervical instability led to a small amount of canal distortion of unknown clinical significance.<sup>24</sup>

The impact of cricoid pressure on cervical spine motion has been evaluated. No significant change in spinal canal size or shape was found after cricoid pressure was applied in cadaveric models of cervical instability.<sup>25</sup> Other airway maneuvers, such as jaw thrust and chin lift, may displace a patient's cervical spine in a similar magnitude as direct laryngoscopy.<sup>26</sup> A laryngeal mask airway decreases the motion in the cervical spine relative to direct laryngoscopy in living patients and cadavers with cervical pathology,<sup>27</sup>

but the insertion and inflation of a laryngeal mask airway was found to increase pressure in the cervical spinal canal in a cadaveric model.<sup>27</sup> The significance of increased pressure in the spinal canal is unknown but should be considered when weighing the benefits of a laryngeal mask airway (ie, decreased neck motion and an improved ability to ventilate or intubate a patient when conventional methods for either are inadequate) in a patient with a possible CSI.

Tracheal intubation with a nasotracheal tube using a flexible fiberoptic bronchoscope reduces cervical spine motion relative to other forms of airway manipulation,<sup>22</sup> but inserting a tube through the nares is concerning because it could pass out of the nasopharynx or potentially worsen a facial fracture in a patient with facial and head injuries. There is some evidence that this phenomenon is not clinically significant and nasal intubation may be safe in patients with head and facial trauma.<sup>28</sup> Despite its safety, nasal intubation is time consuming and can lead to epistaxis, which can make airway management far more challenging and lead to an emergent direct laryngoscopy with resulting cervical motion or an aspiration event.

### Radiologic Assessment

If a patient is stable and there is not an obvious CSI, yet injury mechanism provides reason to suspect one, CSI should be evaluated. There are two major sets of criteria used to determine whether further radiological assessment is necessary or an injury can be ruled out without further studies. The National Emergency X-Radiography Utilization Study (NEXUS) criteria have been proposed as a way to determine the need for radiologic studies to evaluate for a CSI. Patients who meet NEXUS criteria for low risk of CSI should not have further radiological studies.<sup>29</sup> Low-risk criteria are as follows:

- No midline cervical tenderness
- No focal neurological deficit
- Normal alertness
- No intoxication
- No painful distracting injury (in which pain from the injury distracts from pain associated with a coexisting cervical spine injury).

The second set of criteria is the Canadian C-Spine Rule (CCR; Exhibit 10-1). Using the CCR, if the patient does not meet high-risk criteria but does meet one or more low-risk criteria and can turn his or her head 45 degrees left and right, further studies are not needed. If the patient has limited cervical motion, high-risk criteria, or no low-risk criteria, he or she must be

**EXHIBIT 10-1**

**CANADIAN C-SPINE RULE HIGH- AND LOW-RISK CRITERIA**

**High-risk criteria:**

- age greater than 64
- dangerous mechanism of injury
- paresthesias in the extremities

**Low-risk criteria:**

- simple rear-end motor vehicle accident
- sitting position in the emergency department
- patient was ambulatory at any time after the injury
- delayed onset of neck pain
- no midline cervical tenderness

evaluated radiologically.<sup>30</sup> The CCR was more sensitive than the NEXUS criteria (99.4% versus 90.7%,  $P < 0.001$ ) and more specific (45.1% versus 36.8%,  $P < 0.001$ ) in one comparison, and CCR usage would lead to a lower rate of radiological studies indicated (55.9% versus 66.6%,  $P < 0.001$ ).<sup>31</sup> The NEXUS criteria were also found to be insufficiently powerful when the incidence of CSI was higher. The NEXUS criteria was found to have a sensitivity of 82.8%, a specificity of 45.7%, and a negative predictive value of 97.6% in a single-site study with a higher percentage of CSIs sustained than in the NEXUS trial (6.02% versus 2.4% in the NEXUS trial) and using computed tomography (CT) scans rather than radiography to detect cervical fractures.<sup>3</sup> Including high-risk injury (as in the CCR) improved the sensitivity of the NEXUS criteria. The CCR appears to be a superior tool for evaluating the need for a radiological study, but the NEXUS criteria are nearly as good in the low-risk population.

The best radiological study for detecting CSI has also been evaluated. Radiographs of the cervical spine were traditionally performed, including lateral, anterior-posterior, open mouth odontoid, and swimmer's views. In a metaanalysis of several comparisons between plain films and CT scans, plain films had a sensitivity of 52% while CT scans had a sensitivity of

98% for detecting a cervical spine fracture in blunt-trauma victims.<sup>32</sup> Nearly all fractures missed on CT scan were determined to be clinically insignificant. CT scans were also more time efficient because radiographs frequently provided poor visualization and had to be repeated.<sup>33</sup> If it is available and the patient is stable, a cervical CT scan is preferable in both efficacy and efficiency to plain films.

In cases of negative CT scans, ligamentous injury or occult spinal cord injuries are still possible. There is considerable controversy about how to proceed with patients who have normal cervical spine CT scans but unreliable examinations because of depressed level of consciousness. After the initial resuscitation and stabilization, patients with a residually depressed level of consciousness should be examined using magnetic resonance imaging as soon as feasible. Multiple studies have shown low rates of injuries missed on CT scan evaluation, but these studies have disagreed on the clinical significance; some studies have classified the few cases of CSI missed on CT scan as clinically insignificant,<sup>34,35</sup> while others have found significant rates of instability requiring intervention.<sup>36,37</sup> In facilities with magnetic resonance imaging capability, obtunded patients are evaluated after stabilization with magnetic resonance imaging before the cervical collar is removed. In forward settings, case-by-case decisions of when to discontinue CSI precautions must be made by weighing the harm of immobilization against the low likelihood of a significant CSI after a normal CT scan in a patient without a highly suggestive injury pattern.

**Steroids**

Steroids have been used to treat blunt spinal-cord injuries. Preclinical animal data has shown steroids provide a post-induced-injury benefit, but human studies have been ambiguous on neurological outcomes and may suggest greater overall harm.<sup>38</sup> Systemic glucocorticoid treatment may be considered when caring for combat-injured patients, but it is not recommended. Patients with complex polytraumatic combat injuries will likely suffer greater overall morbidity or mortality if given steroids. The decision to administer steroids should be made by the trauma team on a case-by-case basis.

**HEAD TRAUMA**

**Assessment and Monitoring**

Traumatic brain injury (TBI) is common among polytraumatized war casualties. Over 200,000 US service members suffered TBI from 2001 to 2011.<sup>39</sup> The

most commonly used assessments of brain injury are the Glasgow coma scale (Table 10-1) and CT imaging. When available, CT imaging is indicated in obtunded patients or when TBI is suspected. Imaging can yield signs of intracranial pathology, such as bleeding, or

**TABLE 10-1**  
**GLASGOW COMA SCALE**

	Eye Opening	Verbal Response	Motor Response
1	None	None	None
2	To painful stimuli	Incomprehensible sounds	Decerebrate
3	To verbal command	Confabulation	Decorticate
4	Spontaneously	Confused, disoriented	Generalized pain response
5		Oriented and appropriate	Localizes painful stimuli
6			Follows commands

signs of intracranial hypertension, such as midline shift or diminished ventricular volume. In one study, patients with suspected TBI and abnormal CT scan had a greater than 50% chance of developing intracranial hypertension.<sup>40</sup>

Monitoring intracranial hypertension can be difficult in obtunded or anesthetized patients. The Brain Trauma Foundation recommends placing an intracranial pressure (ICP) monitor in salvageable patients with Glasgow coma scale scores less than 8 and CT scans revealing hematoma, contusion, swelling, herniation, or compressed basal cisterns.<sup>41</sup> An intracranial monitor should be considered in TBI patients anesthetized or sedated for long periods of time to monitor ICP for severe elevations. There are many ICP monitors available, but an extraventricular drain is the most commonly used in the forward-deployed setting.

### Management

Cerebral oxygen delivery depends on arterial oxygen content and cerebral blood flow (CBF). The first challenge in optimizing cerebral oxygen delivery is avoiding hypoxia. Arterial hemoglobin oxygen saturation levels below 90% have been linked to worsened outcomes.<sup>42</sup> Hypoxia results from five major causes:

1. low inspired oxygen content (eg, resulting from fire, or carbon monoxide inhalation);
2. hypoventilation (potentially due to drugs or a brainstem injury);
3. perfusion deficits in the lung (eg, hypotension, embolic events);

4. ventilation deficits in the lung (eg, pneumothorax); or
5. poor diffusion into pulmonary capillaries (resulting from pulmonary edema, acute respiratory distress syndrome, transfusion-related acute lung injury, etc).

The second portion of cerebral oxygen delivery is via CBF. The most common clinically used measure of CBF in patients with head injuries is cerebral perfusion pressure (CPP). CPP can become critically decreased even with supranormal cardiac output when ICP is elevated. CPP can be calculated using the following formula:

$$\text{CPP} = \text{mean arterial blood pressure} - \text{central venous pressure or ICP}$$

Patients who experience brain hypoperfusion (measured as a single systolic blood pressure reading of less than 90 mm Hg) have worse outcomes than those who do not.<sup>42</sup> Previously, authors had advocated maintaining CPP greater than 70 mm Hg based on this finding, but the cause-and-effect relationship was unclear. Recent recommendations have changed based on evidence of worsened overall mortality from systemic complications. Currently there is no clear recommended target CPP, and the Brain Trauma Foundation states maintaining a CPP between 50 and 70 mm Hg is an option for care, but it does not meet criteria for a recommendation.<sup>43</sup> Maintaining a goal CPP of greater than 70 mm Hg should be avoided (Brain Trauma Foundation class II recommendation)<sup>43</sup> based on the potential for overall harm to the patient. There is currently no evidence to determine the benefits versus the harm of delayed resuscitation on brain-injured patients.

In addition to concern for mean arterial blood pressure, providers need to consider central venous pressure and ICP; increases in either can decrease cerebral perfusion. Central venous pressure can be increased by using positive end-expiratory pressure, or relatively increased in the brain if the patient is in the Trendelenburg position or if the normal venous outflow pathways are obstructed. The need for positive end-expiratory pressure for better oxygenation must be balanced against the resulting change in central venous pressure and CPP. There are no studies of the impact of jugular venous system cannulation on direct measurements of cerebral venous outflow, though one case series reported no adverse events related to the placement of an internal jugular cannula in patients with intracranial hypertension.<sup>44</sup> There is significant collateral brain drainage in healthy patients via the

vertebral venous system.<sup>45</sup> Patients who are positioned with their heads in a manner that does not compress the contralateral jugular or vertebral venous drainage systems will not likely suffer damage from a unilateral jugular cannula; however, the likelihood of possible concomitant CSI means most patients with severe head injuries and intracranial hypertension will likely require cervical collars, and a subclavian vein cannulation may be more easily accomplished acutely. If subclavian vein cannulation is contraindicated, an internal jugular vein cannulation is likely safe. The addition of positive end-expiratory pressure or the impact of positioning on venous drainage and the resulting change in CPP has been well documented and described, but each patient is different and may tolerate differing levels of each.<sup>46,47</sup>

An elevation in ICP can also decrease CPP and lead to a worsened neurological injury.<sup>48</sup> The degree of ICP elevation that leads to harm is debatable, but the Brain Trauma Foundation recommends that ICP values of 20 to 25 mm Hg should be treated to avoid harm.<sup>49</sup> This recommendation should be taken in context of the patient's clinical examination and CT scan results, if available. If the patient is anesthetized or obtunded and no CT scanner is available, ICPs above 20 mm Hg should be treated. ICP is determined by the volume of brain, blood, and cerebrospinal fluid present in the closed cranial vault.

ICP can be decreased in several ways, targeting all three components. Blood volume in the brain can be altered to decrease ICP, and improved venous drainage from reverse Trendelenberg positioning can decrease the volume of venous blood in the intracranial space. Arterial blood flow can also be reduced through hyperventilation, though hyperventilation is mostly detrimental in cases of head injury and should be reserved for the most refractory cases of acute intracranial hypertension.<sup>50</sup> Hyperventilation will rapidly decrease ICP, but after several hours the brain will equilibrate to the lowered arterial partial pressure of carbon dioxide and the reduction in cerebral arterial blood flow will be decreased. After equilibration, an abrupt normalization of arterial partial pressure of carbon dioxide will lead to increased cerebral arterial blood flow and worsened ICP. Hyperventilation should be used sparingly and reversed slowly.

Brain parenchyma volume can also be decreased to reduce ICP. Mannitol can be used to reduce ICP by removing cytosolic fluid from brain tissue; it is believed the fluid loss occurs mostly from healthier parts of the brain because edema in an injured brain results from chemical pathways that may not be responsive to osmolar gradients. Mannitol may im-

part a survival advantage, but this effect has limited documentation<sup>51</sup> and further analysis is necessary. The Brain Trauma Foundation recommends the use of 0.25 to 1 g/kg of mannitol for intracranial hypertension management (class II evidence). The clinical effects of mannitol can be seen in 15 to 30 minutes and may last up to 6 hours. The potential side effects of mannitol use are renal failure and hemodynamic compromise from fluid shifts and diuresis. Providers should restrict the use of mannitol to patients with signs of intracranial hypertension or elevated measured ICPs.<sup>52</sup>

Other strategies for reducing ICP include removing cerebrospinal fluid or improving compliance with a decompressive craniectomy. Cerebrospinal fluid can be withdrawn using a ventriculostomy, but the benefit can be short-lived due to continuous production.<sup>53</sup> Decompressive craniectomy has been used as an effective treatment for intracranial hypertension.<sup>54</sup> The benefit of early craniectomy, as opposed to craniectomy as a treatment for refractory intracranial hypertension, is still controversial.

### *Decreasing Cerebral Oxygen Consumption*

The cerebral metabolic rate of oxygen (CMRO<sub>2</sub>) can be reduced pharmacologically or through the use of hypothermia. Hypothermia has been shown to reduce oxygen consumption in experimental models, but clinical results are mixed. There is insufficient evidence to recommend induced hypothermia after a traumatic brain injury.<sup>55</sup> Patients who are unintentionally hypothermic are at higher risk of morbidity (odds ratio 2.9; 95% CI, 1.3–6.7), although this may indicate worse injury or greater need for resuscitation rather than a primary effect of hypothermia.<sup>56</sup> Hypothermic patients suffer from myocardial depression and a coagulopathy. Additionally, maintaining mild hypothermia is challenging through the evacuation chain.

A drug-induced "coma" or electroencephalographic burst suppression can decrease ICP and CMRO<sub>2</sub>. There is no evidence of a survival benefit for treating intracranial hypertension with a barbiturate coma,<sup>57</sup> nor is prophylactic barbiturate coma beneficial in patients at risk for intracranial hypertension.<sup>58</sup> A pharmacologic coma should be used as a last-line medical therapy for refractory intracranial hypertension. Propofol has also been used to effectively induce burst suppression, but it carries the risk of propofol infusion syndrome (PIS) with prolonged use. The signs of PIS are lactic acidosis, rhabdomyolysis, and cardiac collapse. PIS appears to be related to dose rate and duration of drug therapy, occurring with doses of 4 mg/kg/h or greater after several hours.<sup>59</sup>

### Other Anesthetics and Intracranial Hypertension

Volatile anesthetic agents cause a dose-dependent decrease in  $CMRO_2$ . Isoflurane produces the greatest decrease (up to 50% reduction in  $CMRO_2$ ), while halothane produces the least effect (less than 25% reduction). Volatile anesthetics also have direct cerebral vasodilatory effects, which can increase CBF and ICP in cases where intracranial compliance is abnormal. These CBF and ICP increases can be inhibited with hyperventilation. Patients with intracranial hypertension undergoing prolonged anesthetics with high doses of volatile agents would likely benefit from ICP monitoring because of the mixed effect the agents have on  $CMRO_2$  and CBF. Lower doses generally reduce ICP through reduced  $CMRO_2$  but higher doses, approaching 1 MAC (minimum alveolar concentration percentage at 1 atmosphere), will lead to an elevation of ICP through increased CBF.

Intravenous anesthetic administration leads to the preservation of cerebral autoregulation and responsiveness of the vasculature to carbon dioxide. Barbiturates and propofol decrease CMR and ICP even at higher doses. Commonly used opioids have minimal intrinsic effect on ICP, but can increase ICP by depressing respiratory drive and the resulting hypercarbia. Regional anesthetics may be appropriate in patients with head injuries who are undergoing surgical procedures or for pain control.

### Resuscitation and Intracranial Hypertension

Many authors have advocated small-volume resuscitation and the use of hypertonic saline in civilian patients with head trauma; however, complex polytraumatic injuries usually require large-volume resuscitations. The goal of resuscitation should be restoration of adequate systemic perfusion and oxygen delivery. Although over resuscitation should be avoided, it should not be used as a justification for under resuscitating because many patients with complex polytraumatic injuries have lost greater than one blood volume in the first 24 hours after injury. The need to limit cerebral edema must be carefully balanced on a case-by-case basis against the need for further volume expansion to combat shock. Hyponatremia should be avoided, which is relatively easy in large-volume resuscitations because of the amount of blood that is given with 0.9% normal saline. Excessive hypernatremia appears to be a more significant problem than avoiding hyponatremia. In many cases, severe-trauma patients with refractory acidosis required treatment with tris(hydroxymethyl)

#### EXHIBIT 10-2

#### TREATING INTRACRANIAL PRESSURE

##### Optimize oxygenation

- Increase oxygen delivery by treating hypoxia and hypotension.
- Decrease oxygen demand by inducing hypothermia or coma.

##### Decrease ICP

- Decrease CSF volume with ventriculostomy; decrease production and increase absorption.
- Decrease intracranial blood volume by hyperventilation or by placing the patient in the reverse Trendelenburg position.
- Decrease brain parenchymal volume with mannitol or by inducing hypernatremia.
- Improve intracranial compliance with decompressive craniectomy.

CSF: cerebrospinal fluid  
ICP: intracranial pressure

aminomethane<sup>60</sup> rather than sodium bicarbonate because of the severe hypernatremia that resulted from resuscitation (Exhibit 10-2).

### Seizures

Seizures occur in a significant number of patients who sustain head trauma. Posttraumatic seizures can be categorized as early (occurring in the first 7 days after injury) or late. Antiepileptic drugs given shortly after injury decrease early seizure activity but not late seizures. In a study by Temkin et al, there was no survival difference between groups treated with antiepileptic drugs and untreated groups.<sup>61</sup> The decision to administer prophylactic antiepileptic drugs should be made on a case-by-case basis, but all patients who develop PTS should be treated. Risk factors for PTS include the following:

- younger age;
- penetrating head wound;
- depressed skull fracture;
- subdural hematoma, intracerebral hematoma, epidural hematoma, or cortical contusion; and
- Glasgow coma scale score less than 10.

## SUMMARY

Severe neurological injury can result in a devastating outcome. Optimum survival requires close medical support from the point of wounding and throughout the continuum of care. The

anesthetic input can be best defined as detailed attention to each aspect of resuscitation and above all else avoiding morbidity from secondary cerebral insult.

## REFERENCES

1. Rhee P, Kuncir EJ, Johnson L, et al. Cervical spine injury is highly dependent on the mechanism of injury following blunt and penetrating assault. *J Trauma*. 2006;61(5):1166–1170.
2. Grossman MD, Reilly PM, Gillett T, Gillett D. National survey of the incidence of cervical spine injury and approach to cervical spine clearance in U.S. trauma centers. *J Trauma*. 1999;47:684–690.
3. Duane TM, Mayglothling J, Wilson SP, et al. National Emergency X-Radiography Utilization Study criteria is inadequate to rule out fracture after significant blunt trauma compared with computed tomography. *J Trauma*. 2011;70(4):829–831.
4. Lustenberger T, Talving P, Lam L, et al. Unstable cervical spine fracture after penetrating neck injury: a rare entity in an analysis of 1,069 patients. *J Trauma*. 2011;70(4):870–872.
5. Ordog GJ, Albin D, Wasserberger J, Schlater TL, Balasubramaniam S. 110 bullet wounds to the neck. *J Trauma*. 1985;25(3):238–246.
6. Ramasamy A, Midwinter M, Mahoney P, Clasper J. Learning the lessons from conflict: pre-hospital cervical spine stabilisation following ballistic neck trauma. *Injury*. 2009;40(12):1342–1345.
7. Mulligan RP, Friedman JA, Mahabir RC. A nationwide review of the associations among cervical spine injuries, head injuries, and facial fractures. *J Trauma*. 2010;68(3):587–592.
8. Merrit RM, Williams MF. Cervical spine injury complicating facial trauma: incidence and management. *Am J Otolaryngol*. 1997;18:235–238.
9. Goldberg W, Mueller C, Panacek E, Tigges S, Hoffman JR, Mower WR. Distribution and patterns of blunt traumatic cervical spine injury. *Ann Emerg Med*. 2001;38(1):17–21.
10. Lennarson PJ, Smith DW, Sawin PD, Todd MM, Sato Y, Traynelis VC. Cervical spine motion during intubation: efficacy of stabilization maneuvers in the setting of complete segmental instability. *J Neurosurg*. 2001;94(2):265–270.
11. Majernick TG, Bienek R, Houston JB, Huges HG. Cervical spine movement during orotracheal intubation. *Ann Emerg Med*. 1986;15:417–420.
12. Podolsky S, Baraff LJ, Simon RR, Hoffman JR, Larmon B, Ablon W. Efficacy of cervical spine immobilization methods. *J Trauma*. 1983;23(6):461–465.
13. Heath KJ. The effect on laryngoscopy of different cervical spine immobilisation techniques. *Anaesthesia*. 1994;49:843–845.
14. Goutcher CM, Lochhead V. Reduction in mouth opening with semirigid cervical collars. *Br J Anaesth*. 2005;95:344–348.
15. Gerling MC, Davis DP, Hamilton RS, et al. Effects of cervical spine immobilization technique and laryngoscope blade selection on an unstable cervical spine in a cadaver model of intubation. *Ann Emerg Med*. 2000;36:293–300.
16. MacIntyre PA, McLeod AD, Hurley R, Peacock C. Cervical spine movements during laryngoscopy. Comparison of the Macintosh and McCoy laryngoscope blades. *Anaesthesia*. 1999;54:413–418.
17. Hastings RH, Vigil AC, Hanna R, Yang BY, Sartoris DJ. Cervical spine movement during laryngoscopy with the Bullard, Macintosh and Miller laryngoscopes. *Anesthesiology*. 1995;82:859–869.

18. Cooper SD, Benumof JL, Ozaki GT. Evaluation of the Bullard laryngoscope using the new intubating stylet: comparison with conventional laryngoscopy. *Anesth Analg.* 1994;79:965–970.
19. Watts AD, Gelb AW, Bach DB, Pelz DM. Comparison of Bullard and Macintosh laryngoscopes for endotracheal intubation of patients with a potential cervical spine injury. *Anesthesiology.* 1997;87:1335–1342.
20. Turkstra TP, Craen RA, Pelz DM, Gelb AW. Cervical spine motion: a fluoroscopic comparison during intubation with lighted stylet, Glidescope, and Macintosh laryngoscope. *Anesth Analg.* 2005;101:910–915.
21. Agro F, Barzoi G, Montecchia F. Tracheal intubation using a Macintosh laryngoscope or a Glidescope in 15 patients with cervical spine immobilization. *Br J Anaesth.* 2003;90:705–706.
22. Brimacombe J, Keller C, Kunzel KH, Gaber O, Boehler M, Puhlinger F. Cervical motion during airway management: a cinefluoroscopic study of the posteriorly destabilised third cervical vertebrae in human cadavers. *Anesth Analg.* 2000;91:1274–1278.
23. Manninen PH, Jose GB, Lukitto K, Venkatraghavan L, El Beheiry H. Management of the airway in patients undergoing cervical spine surgery. *J Neurosurg Anaesthesiol.* 2007;19:190–194.
24. Gerling MC, Davis DP, Hamilton RS, et al. Effect of surgical cricothyrotomy on the unstable cervical spine in a cadaver model of intubation. *J Emerg Med.* 2001;20(1):1–5.
25. Donaldson WF 3rd, Heil BV, Donaldson VP, Silvaggio VJ. The effect of airway maneuvers on the unstable C1-C2 segment. A cadaver study. *Spine.* 1997;22(11):1215–1218.
26. Aprahamian C, Thompson BM, Finger WA, Darin JC. Experimental cervical spine injury model: evaluation of airway management and splinting techniques. *Ann Emerg Med.* 1984;13(8):584–587.
27. Kihara S, Watanabe S, Brimacombe J, Taguchi N, Yaghuci Y, Yamaski Y. Segmental cervical spine movement with the intubating laryngeal mask during manual in-line stabilization in patients with cervical pathology undergoing cervical spine surgery. *Anesth Analg.* 2000;91:195–200.
28. Rosen CL, Wolfe RE, Chew SE, Branney SW, Roe EJ. Blind nasotracheal intubation in the presence of facial trauma. *J Emerg Med.* 1997;15(2):141–145.
29. Hoffman JR, Wolfson AB, Todd K, Mower WR. Selective cervical spine radiography in blunt trauma: methodology of the National Emergency X-Radiography Utilization Study (NEXUS). *Ann Emerg Med.* 1998;32(4):461–469.
30. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA.* 2001;286(15):1841–1848.
31. Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. *N Engl J Med.* 2003;349(26):2510–2518.
32. Holmes JF, Akkinepalli R. Computed tomography versus plain radiography to screen for cervical spine injury: a meta-analysis. *J Trauma.* 2005;58:902–905.
33. Daffner RH. Cervical radiography for trauma patients: a time-effective technique? *AJR Am J Roentgenol.* 2000;175(5):1309–1311.
34. Como JJ, Thompson MA, Anderson JS, et al. Is magnetic resonance imaging essential in clearing the cervical spine in obtunded patients with blunt trauma? *J Trauma.* 2007;63:544–549.
35. Hogan GJ, Mirvis SE, Shanmuganathan K, Scalea TM. Exclusion of unstable cervical spine injury in obtunded patients with blunt trauma: is MR imaging needed when multi detector row CT findings are normal? *Radiology.* 2005;237:160–173.
36. Stassen NA, Williams VA, Gestring ML, Cheng JD, Bankey PE. Magnetic resonance imaging in combination with helical computed tomography provides a safe and efficient method of cervical spine clearance in the obtunded trauma patient. *J Trauma.* 2006;60:171–177.

37. Sarani B, Waring S, Sonnad SS, Schwab CW. Magnetic resonance imaging is a useful adjunct in the evaluation of the cervical spine of injured patients. *J Trauma*. 2007;63:637–640.
38. Hurlbert RJ. The role of steroids in acute spinal cord injury: an evidence based analysis. *Spine*. 2001;26(24 suppl):39–46.
39. Defense and Veterans Brain Injury Center website. Available at: <http://www.dvbic.dcoe.mil/dod-worldwide-numbers-tbi>. Accessed April 21, 2014.
40. Narayan RK, Kishore PR, Becker DP, et al. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury. *J Neurosurg*. 1982;56:650–659.
41. Marshall LF, Smith RW, Shapiro HM. The outcome with aggressive treatment in severe head injuries. Part I: the significance of intracranial pressure monitoring. *J Neurosurg*. 1979;50:20–25.
42. Chesnut RM, Marshall LF, Klauber MR, et al. The role of secondary brain injury in determining outcome from severe head injury. *J Trauma*. 1993;34:216–222.
43. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, et al. Guidelines for the management of severe traumatic brain injury. IX. Cerebral perfusion thresholds. *J Neurotrauma*. 2007;24(1 suppl):59–64.
44. Woda RP, Miner ME, McCandless C, McSweeney TD. The effect of right internal jugular vein cannulation on intracranial pressure. *J Neurosurg Anesthesiol*. 1996;8(4):286–292.
45. Doepp F, Schreiber SJ, von Münster T, Rademacher J, Klingebiel R, Valdueza JM. How does the blood leave the brain? A systematic ultrasound analysis of cerebral venous drainage patterns. *Neuroradiology*. 2004;46(7):565–570.
46. Zhang XY, Wang QX, Fan HR. Impact of positive end-expiratory pressure on cerebral injury patients with hypoxemia. *Am J Emerg Med*. 2011;29:699–703.
47. Caricato A, Conti G, Della Corte F, et al. Effects of PEEP on the intracranial system of patients with head injury and subarachnoid hemorrhage: the role of respiratory system compliance. *J Trauma*. 2005;58(3):571–576.
48. Marmarou A, Anderson RL, Ward JD. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg*. 1991;75:59–66.
49. The Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury, 3rd edition. Available at: <https://www.braintrauma.org/coma-guidelines/btf-guidelines>. Accessed December 17, 2012.
50. Muizelaar JP, Marmarou A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg*. 1991;75:731–739.
51. Schwartz ML, Tator CH, Rowed DW, Reid SR, Meguro K, Andrews DF. The University of Toronto head injury treatment study: a prospective, randomized comparison of pentobarbital and mannitol. *Can J Neurol Sci*. 1984;11(4):434–440.
52. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, et al. Guidelines for the management of severe traumatic brain injury. II. Hyperosmolar therapy. *J Neurotrauma*. 2007;24(1suppl):14–20.
53. Kerr ME, Weber BB, Sereika SM, Wilberger J, Marion DW. Dose response to cerebrospinal fluid drainage on cerebral perfusion in traumatic brain-injured adults. *Neurosurg Focus*. 2001;11(4):E1.
54. Eberle BM, Schnüriger B, Inaba K, Gruen JP, Demetriades D, Belzberg H. Decompressive craniectomy: surgical control of traumatic intracranial hypertension may improve outcome. *Injury*. 2010;41(9):894–898.
55. Peterson K, Carson S, Carney N. Hypothermia treatment for traumatic brain injury: a systematic review and meta-analysis. *J Neurotrauma*. 2008;25(1):62–71.

56. Konstantinidis A, Inaba K, Dubose J, et al. The impact of nontherapeutic hypothermia on outcomes after severe traumatic brain injury. *J Trauma*. 2011;71:1627–1631.
57. Roberts I, Sydenham E. Barbiturates for acute traumatic brain injury. *Cochrane Database Syst Rev*. 2005, CD000033. DOI: 10.1002/14651858. CD000033.pub2.
58. Schwartz ML, Tator CH, Rowed DW, Reid SR, Meguro K, Andrews DF. The University of Toronto head injury treatment study: a prospective, randomized comparison of pentobarbital and mannitol. *Can J Neurol Sci*. 1984;11(4):434–440.
59. Otterspoor LC, Kalkman CJ, Cremer OL. Update on the propofol infusion syndrome in ICU management of patients with head injury. *Curr Opin Anaesthesiol*. 2008;21(5):544–551.
60. Hoste EA, Colpaert K, Vanholder RC, et al. Sodium bicarbonate versus THAM in ICU patients with mild metabolic acidosis. *J Nephrol*. 2005;18(3):303–307.
61. Temkin NR, Dikmen SS, Wilensky AJ, Keihm J, Chabal S, Winn HR. A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med*. 1990;323:497–502.

