Medic 1 respond to I-684 southbound for a single-car MVA. Within eight minutes you arrive on scene to find a late-model, red hatchback wrapped around a tree off the side of the highway.

You tell your partner to bring the airway and trauma bags as you grab the backboard and cervical collars and make your way to the car. You immediately notice that it has heavy front-end damage, with approximately two feet of front-end intrusion into the passenger compartment and a "spidered" windshield. Your patient, a middle-age male whose seatbelt is unfastened, is unconscious, unresponsive and slumped in the driver’s seat.

You rapidly extricate the patient and move him into the back of the ambulance to continue your assessment and begin transport to the local trauma facility. You find the patient’s airway compromised with blood and debris from the wreckage, so you suction the airway and drop in an oral-pharyngeal airway (which the patient accepts), prior to assessing the patient’s breathing. The patient’s breathing is slow and shallow. You instruct your partner to begin ventilation, using a bag-valve mask (BVM)...
HYPOXIA

The JEMS continuing education program is coordinated by the Center for Emergency Medicine, Pittsburgh, and the University of Pittsburgh, School of Health and Rehabilitation Sciences.
attached to an oxygen source at 15 LPM. Your partner says she’s getting good chest rise and fall with limited resistance. A quick radial and carotid pulse check reveals pulses are present, but the rate is slow, in the low 50s.

Examination of the patient’s head reveals a bloody region on the forehead. On palpation, the wound feels like an indentation of the skull. You recall that the car had spidered windshield and suspect the patient struck his head on impact. You continue your assessment and find obvious deformities to the patient’s facial bones, which you suspect are fractured. The patient’s nose is shattered and bleeding heavily. Pupils are equal and reactive to light. The rest of the exam proves unremarkable. Baseline vitals pulse 54 and regular, BP 168/104, respirations 14 (your partner is controlling the rate), ECG sinus bradycardia, pulse oximeter 96% \( \text{SpO}_2 \). You take a deep breath and ponder how best to manage the airway of this head-injured patient.

**Introduction**

Every 21 seconds, someone in the United States receives a brain injury. Every five minutes, one of these individuals will die and another will become permanently disabled. According to the Centers for Disease Control and Prevention (CDC), of all types of injury, those to the brain are among the most likely to result in the death or permanent disability of an individual.

Traumatic brain injury (TBI) can be defined as “an insult to the brain, not of degenerative or congenital nature, caused by an external physical force that may produce a diminished or altered state of consciousness, which results in an impairment of cognitive abilities or physical functioning. It can also result in the disturbance of behavioral or emotional functioning.”

TBI is a common injury that has a devastating physical, emotional, social and economic impact on our society. The CDC estimates that 1 million people each year are treated for TBI and released from hospital emergency departments (EDs). Approximately 230,000 individuals are hospitalized and survive cases of TBI, and 70,000–90,000 suffer permanent neurological disabilities. An estimated 52,000 people die as a result of TBI each year.

CDC data analysis reveals that the risk of having a TBI is especially high among adolescents, young adults and people older than 75. Males are at twice the risk of females for suffering a TBI. The leading cause of brain

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**Objectives**

- Explain the relationship between intracranial pressure, mean arterial pressure and cerebral perfusion pressure.
- Recognize the signs and symptoms of increasing intracranial pressure.
- Describe the airway and ventilatory treatment for a patient with a traumatic brain injury.
injury is vehicle crashes, which account for 50% of all TBIs. Falls are the second leading cause overall, and the leading cause of brain injury in the elderly. Violence, specifically firearm violence, is the third leading cause of TBI in the United States.3

The economic cost associated with brain injury is devastating to patients and society as a whole. The cost of TBI in the United States is estimated as $48.3 billion annually.1 Hospitalization accounts for $31.7 billion, and fatal brain injuries cost $16.6 billion each year.8

TBI threatens both the airway and ventilatory status of an injured patient, raising unique issues for the treating emergency medical provider. A critically injured patient suffering from TBI will generally be unconscious or could rapidly deteriorate into unconsciousness, at which point the patient cannot protect their airway, and aggressive airway management becomes necessary.

Simply stated, all acute emergency medical care provided to the TBI patient is aimed at ensuring that the maximal amount of oxygenated blood reaches the injured brain tissues. Although in many regions throughout the country pulse oximetry technology is commonly being used, end-tidal CO₂ detection devices (specifically capnography) are not routinely used in the prehospital setting. These technologies are the gold standards for assessing the oxygenation level and ventilatory status of the TBI patient.

Medical providers can ensure the greatest possible outcome by maintaining a high SpO₂ (preferably 100%) as measured by a pulse oximeter and by maintaining normocapnia (PaCO₂ 35–37 mmHg), as measured by capnography, unless the patient’s clinical condition necessitates otherwise.9 Only when the TBI patient’s clinical condition deteriorates to a degree at which obvious external signs are manifested will the airway and ventilation treatments change.

The importance of not allowing the TBI patient to suffer from a hypoxic event cannot be overstated. Hypoxia is defined as an oxygen saturation of less than 90% or a PaO₂ less than 60 mmHg.10 Hypoxia in the brain-injured patient often leads to devastating damage in already fragile brain tissues and can drastically increase morbidity and mortality. The damage caused by even the shortest hypoxic event is often not
evident at the time of the event, but appears several hours or even days later. This means that initial medical caregivers, including prehospital providers, ED nurses and ED physicians won’t likely see the morbidity and mortality associated with these hypoxic episodes in the TBI patient.

This article summarizes the unique airway and ventilation challenges posed by the TBI patient and provides information on the proper management of these cases.

Pathophysiology of head trauma

Within the past two decades, we have begun to decipher the pathophysiology of TBI. A central concept that has emerged in the understanding of brain injury is the distinction between primary and secondary brain injury. Research has shown that not all neurological damage occurs at the moment of impact (primary injury), but, rather, evolves over the ensuing hours or days following the trauma (secondary injury).9

Secondary injury results from a combination of several factors and can significantly worsen disability and increase mortality. One such factor is the brain’s physiologic response to the primary injury, which involves swelling, causing decreased perfusion. The second factor results from complications, such as hypoxia or hypotension following the initial injury, which further damage brain tissue.11

A key factor in minimizing damage from primary and secondary brain injury is the maintenance of cerebral perfusion pressure (CPP), the...
The brain has a compensatory reflex to autoregulate for a constant blood flow, regardless of blood pressure, by altering the resistance of cerebral blood vessels. These compensatory mechanisms are often lost after head trauma. It should be noted that although the brain accounts for only 2% of an individual’s total body weight, it consumes approximately 20% of the body’s oxygen supply.12

In an uninjured brain, the ICP is ordinarily low, generally below 10 mmHg.12 Normal ICP does not significantly impede blood flow as long as the MAP remains at least 50 mmHg.12 However, the initial response of a traumatically injured brain is to swell. Because little extra space exists within the cranial vault, this swelling causes ICP to increase. This rise in ICP decreases blood flow to the brain and has disastrous effects on already injured, delicate brain tissue. The swelling brain has a limited ability to compensate as cerebrospinal fluid and blood move into the spinal canal and extracranial vasculature, respectively. ICP is considered dangerous when it rises to 15 mmHg and is generally treated at 20 mmHg. Cerebral herniation may occur at pressures above 25 mmHg.11,15

A force from the primary injury great enough to cause bruising on the underlying brain can further complicate the issue of brain swelling. Bruising causes vasodilation, allowing increased blood flow to injured brain tissue and resulting in an accumulation of blood in the brain. This blood takes up space and further exerts pressure on the already swollen underlying brain tissue.11

Management of the TBI patient, therefore, focuses on stabilizing the injured patient and preventing secondary neuronal injury by maintaining perfusion of oxygenated blood to the brain.16 Adequate CPP management is key to preventing further injury in a brain-injured patient.14
Cerebral blood flow may be lowered to the ischemic threshold after brain injury; thus, in order to prevent secondary injury, the flow of well oxygenated blood must be restored immediately.

Randomized, controlled trials involving human subjects have derived no evidence of the optimal CPP level. Most current research suggests CPP should be kept above 70–80 mmHg. It’s believed that mortality increases approximately 20% for each 10 mmHg loss of CPP. Further, in previously published studies where CPP is maintained above 70 mmHg, the reduction in mortality is as much as 35% for those with severe head injuries.

In order to maintain CPP at the critical threshold of 70–80 mmHg, a medical clinician may raise the MAP or lower the ICP. It’s not within the scope of this article to discuss how to raise the mean arterial blood pressure. However, several treatment modalities focusing on aggressive airway control and ventilation can be utilized by emergency medical clinicians to temporarily reduce ICP, buying time to get a critical patient to a trauma center and into neurosurgery for definitive care.

**Assessment & TBI patient presentation**

In most cases, a complete and detailed scene survey should be the first clue for EMS providers that they’re dealing with a TBI patient. Often, the mechanism of injury will suggest a brain injury. The EMS provider should evaluate airway, breathing and circulation, followed by a rapid trauma survey to identify any life-threatening injuries that must be corrected immediately.

Two diagnostic tools critical in the assessment of the TBI patient are pulse oximetry and end-tidal CO₂ detection devices, preferably in the form of capnography. A low or falling SpO₂ (<93%) indicates that the airway or ventilatory status may be compromised. A pulse oximeter measures oxygenation and alerts caregivers to hypoxic events. Capnography measures ventilation and will alert the caregiver to ventilatory events before a pulse oximeter can do so.

The assessment of the TBI patient is vested in serial neurological evaluations, most importantly the Glasgow Coma Score (GCS) and pupil examination. Getting an accurate GCS in the field as soon as possible is important because it serves as a baseline marker for the TBI patient’s status (see Figure 1, p. 109).

Traditionally, a GCS of less than 8 indicated severe brain injury. Because of faster recognition of TBI patients, improvements in prehospital patient care and a greater understanding of the importance of rapid transport and short scene times in patients with brain injuries, patients now arrive at EDs earlier, with evolving brain injuries.

Ultimately, this means that pa-
tients with a GCS of 12 or lower may have injuries indicative of major TBI. Short scene times, aggressive airway management and treatment and rapid transport to a trauma center will give your patient the optimal chance for recovery.

**Predictable head injuries**

Brain injury often manifests in a predictable manner, yielding a “classic” TBI patient. These patients present with signs and symptoms that necessitate immediate change in airway and ventilatory treatment.

A patient suffering from a TBI that results in increased ICP and subsequent compression and displacement of brain tissue presents with an altered level of orientation and/or altered level of consciousness. Specific signs and symptoms related to the brain structures affected by bruising, compression and/or ischemia may also appear. Thus, as a portion of the brain is disrupted by injury, the specific activity it controls is affected.

Example: If the temporal lobe is affected, speech and auditory disturbances are likely. If the occipital lobe is injured, visual disturbances are likely. If a large region of cortical tissue is affected by brain injury, the patient’s level of awareness could decrease; this could manifest as amnesia (either antegrade or retrograde), disorientation (to person, place or time), confusion or combativeness. Hemiplegia, seizures and weakness are indicative of focal deficits and brain injury. Figure 2 (p. 110) shows where in the brain certain symptoms may appear.

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**FIGURE 1: Glasgow Coma Scale**

<table>
<thead>
<tr>
<th>Eye Opening (E)</th>
<th>Verbal Response (V)</th>
<th>Motor Response (M)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 = Spontaneous</td>
<td>5 = Normal conversation</td>
<td>6 = Normal</td>
</tr>
<tr>
<td>3 = To voice</td>
<td>4 = Disoriented conversation</td>
<td>5 = Localizes to pain</td>
</tr>
<tr>
<td>2 = To pain</td>
<td>3 = Words, but not coherent</td>
<td>4 = Withdraws to pain</td>
</tr>
<tr>
<td>1 = None</td>
<td>2 = No words, only sounds</td>
<td>3 = Decorticate posture</td>
</tr>
<tr>
<td></td>
<td>1 = None</td>
<td>2 = Decerebrate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 = None</td>
</tr>
</tbody>
</table>

**GCS Total = E + V + M**
activities are controlled.18

The somewhat predictable signs and symptoms for TBI are commonly referred to as central syndrome. This syndrome occurs if the patient suffers from compression of the brain along the central region of the cerebrum.12 The clinical manifestations resulting from central syndrome occur in a predictable manner as progressive pressure is first directed to the midbrain, then the pons and, finally, to the medulla oblongata.12

In central syndrome, as ICP rises and the upper brainstem is compressed, there is an increase in the blood pressure to maintain CPP and a reflex decreasing heart rate in response to vagus nerve (CN X) parasympathetic stimulation.12 The TBI patient suffering from central syndrome also commonly exhibits the Cheyne-Stokes respiratory pattern, characterized by alternating periods of apnea and tachypnea.19,12 The combination of increasing
blood pressure, slowing pulse and altered respiratory pattern is known as Cushing’s reflex (or triad).

Cushing’s reflex does not occur in all TBI patients; however, if present, it’s generally considered a late indication of TBI and rising ICP. Other manifestations of upper brainstem compression include body temperature changes and vomiting (often without any nausea) if the hypothalamus is involved. The patient may also manifest decorticate posturing in response to painful stimuli. While compression is limited to the upper brainstem, the pupils should remain equal and reactive.

As the ICP rises and progresses to compression of the middle brainstem, pulse pressure widens, and heart rate slows. The respiratory pattern may change to central neurologic hyperventilation, characterized by deep, rapid breaths.

The patient with middle brainstem compression may also present with decorticate posturing. When the ICP rises to about 25 mmHg, the brain swelling leads to either internal or external herniation. External herniation occurs when the cerebellar peduncles (cerebellar brain structures) are forced down through the foramen magnum. Internal herniation, more common in TBI than external herniation, occurs when the temporal lobe is pushed down onto the midbrain through the tentorium incisura, an opening in the dura mater (the tough outer layer of the meninges). This compresses the oculomotor nerve (CN III), causing ipsilateral pupil dilation and giving the EMS provider a vital external sign that the process of herniation has begun in the patient. Figure 5 (left) shows how the oculomotor nerve can become compressed as a result of brain swelling and increased ICP.

As herniation progresses, the contralateral (opposite) oculomotor nerve may be compressed, producing bilateral pupil dilation. This is why serial assessment is so vital. Patients may arrive with a mildly impaired GCS (due to limited initial swelling) and rapidly deteriorate due to increasing cerebral swelling. Thus, pupils may initially appear normal and then dilate as ICP rises and the brain begins to herniate.

Finally, as the ICP increases and affects the lower brainstem, both pupils will appear fully dilated and unreactive. The breathing pattern changes to apneustic (also called
Biot's) pattern. The apneustic respiratory pattern literally has no pattern. The breaths vary in depth and rate with pauses.

At this point, the patient's ICP has increased to the point that CPP has effectively been reduced to zero, and the brain cells begin to die. The pulse rate drops dramatically, and blood pressure falls. Death is imminent for such patients.

**TBI treatment**

The specific goals of the acute treatment of the TBI patient include the early recognition of brain injury, aggressive airway protection, oxygenation, ventilation to normocapnia (unless clinically indicated otherwise), hemorrhage control, prevention of hypotension (systolic blood pressure less than or equal to 90 mmHg) and fluid resuscitation to a normovolemic status.9,16

A short on-scene time and rapid transport to an appropriate trauma center are critical. These treatment modalities are ultimately aimed at restoring CPP to get oxygen-rich blood to the injured brain tissue as quickly as possible.

Treatment of TBI patients begins with securing the airway while maintaining cervical spine stabilization. Begin by opening the airway using the modified jaw-thrust technique. Be prepared to suction fluids or other foreign material in the airway that could cause an obstruction. Next, use a simple airway adjunct, such as the oropharyngeal airway (OPA) or the nasopharyngeal airway (NOA), to help protect the obstructed airway.

A conscious patient will not tolerate an OPA, so you may have to use an NOA (which may be contraindicated in patients with suspected basilar skull fractures).

Endotracheal intubation is the most definitive airway protection technique for the brain injury patient. Any TBI patient with a GCS ≤8 should have their airway protected through endotracheal intubation (with a cuffed tube), using rapid sequence intubation (RSI) if necessary.9,10

If permitted by protocol, administer lidocaine (1.5 mg/kg IV approximately three minutes prior to intubation) or other appropriate medication to blunt the increases in ICP, systolic blood pressure and pulse rate usually associated with intubation.10,22

If endotracheal intubation is not an option and other advanced airway management techniques, such as nasotracheal intubation, the laryngeal mask airway or Combitube, are not...
viable alternatives, consider performing a needle cricothyrotomy (or other appropriate surgical airway). This procedure may be appropriate for a patient with massive craniofacial trauma that prevents you from locating known anatomic landmarks or for a patient with an airway obstructed due to edema of the glottis, fracture of the larynx or severe oropharyngeal hemorrhage.

Data suggest that hypoxia (apnea, cyanosis or SaO₂ less than 90%) is a significant parameter that's associated with poor patient outcome and must be completely avoided, if possible, or corrected immediately.9

After protecting the airway, the provider should oxygenate the TBI patient with 100% FiO₂ by BVM. The use of a pulse oximeter helps the medical provider assess the patient’s current oxygenation status.

Ideally, SpO₂ is maintained at 100%. The provider should ventilate the TBI patient normally, defined as a respiratory rate of 10–12 breaths per minute (BPM) for adults, 12–16 BPM for children and 16–20 BPM for infants.

More important than relying on the BPM is relying on capnography and, if possible, arterial blood gas (ABG) samples. The normal arterial CO₂ (PaCO₂) range in an ABG is 35–45 mmHg. The corresponding normal ETCO₂ from capnography is 30–43 mmHg.23

In the past, it was recommended that caregivers hyperventilate patients with increased ICP to maintain hypocapnea. Current research indicates that ventilatory rate should be aimed at keeping the PaCO₂ at the lower end of normal.10 To ensure the best outcome for TBI patients, the prehospital provider with airway and ventilation management experience should focus on this task.

Routine prophylactic hyperventilation to lower ICP is no longer recommended and should be avoided.9,10 In the past, the theory behind its use was that during periods of hypoventilation or hypoxia, CO₂ increases. CO₂ causes vasodilation in
the cerebral blood vessels, allowing a greater volume of blood to flow into the intracranial vault, thereby raising ICP. On the other hand, hyperventilation causes a decrease in CO₂, resulting in vasoconstriction and providing the swollen brain additional space.

Recent studies show that after TBI, cerebral blood flow reduces by as much as two-thirds. Routine hyperventilation can further decrease blood flow to the brain, potentially to the point of cerebral ischemia.

Essentially, both hypoventilation (leading to increased brain swelling and ICP) and hyperventilation (causing severe vasoconstriction of the blood vessels in the brain) cause cerebral hypoxia and increase both morbidity and mortality. Therefore, hyperventilation of the TBI patient should be performed only when clinical signs indicate the presence of an impending or progressive herniation.

In these cases, use hyperventilation as a last ditch effort to temporarily reduce ICP and herniation, buying time to get the patient into neurosurgery. The signs of impending herniation include:

1. Unequal, nonreactive or dilated pupils;
2. Extensor posturing; and
3. Neurologic deterioration [decrease in GCS of more than two points in patients with initial GCS less than 8] after correction of any hypotension or hypoxemia.

Hyperventilate adults at a rate of 16–20 BPM, 20–24 BPM for children and 24–28 BPM for infants. Once again, the gold standard is to rely on capnography and/or ABGs to direct ventilatory treatment. Hyperventilation should achieve a PaCO₂ of 30–35 mmHg.

Summary

Brain injury, specifically TBI, is a common occurrence in our society, with devastating physical, emotional, social and economic costs. Injury not only occurs on initial impact, but also develops slowly over ensuing hours or days. TBI poses unique challenges and requires special care, especially with airway management and ventilatory treatment. Serial assessment, particularly of the GCS and pupillary changes, is vital in continuing to monitor patient status. All treatment associated with the TBI patient is aimed at restoring CPP and allowing oxygenated blood to reach injured brain tissues.

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References

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