**Chapter 30  
Traumatic brain injury**

**Kraigher O’Keefe**

**Introduction**

An estimated 1.7 million people sustain traumatic brain injuries (TBI) annually in the United States, with total costs estimated at $60 billion per year [1,2]. Overall, TBI-related deaths account for one-third of all trauma-related deaths, or 53,000 deaths annually in the United States [2]. Traumatic brain injuries result primarily from falls (35%), motor vehicle collisions (17%), and direct blows to the head (16%) [2]. Men are more likely to sustain TBI than women for virtually all age groups. Children aged 0–14 account for approximately one-third of the cases of TBI [3]. Children (up to age 18) and adults over 75 years old are more likely to present to the ED, and are more likely to die from head injuries [2].

The initial brain insult occurs from direct impact, acceleration/deceleration injury, or penetrating wound resulting in bleeding, contusion, and ultimately cell death. Prevention measures include use of helmets, seat belts, car seats for children, and efforts to reduce falls in the elderly [4]. Once the primary brain injury has occurred, reversal of the insult is impossible. Prevention of secondary brain injury is the goal of therapeutic intervention. Treatment must start with initial management on scene, and continue until the eventual resolution of the patient’s injuries. Aggressive treatment of severe head injury patients has been shown to be cost-effective, with an increase in quality-adjusted life-years when all costs are considered [5].

A review of a few physiological concepts is necessary for health care providers to understand how to prevent secondary brain injury. Cerebral perfusion pressure (CPP) is equal to the mean arterial pressure (MAP) minus the intracranial pressure (ICP). Measuring an accurate MAP in the prehospital setting may be difficult, making the systolic blood pressure (SBP) a surrogate that has been used in published guidelines and research. Rapid rises in ICP cause compression of the brain within an enclosed space (skull). As the pressure increases, the brain can be pushed downward, herniating in several possible directions. This herniation can cause compression of cranial nerves, posturing, changes in respiration, paralysis, and sudden death.

Management of severe traumatic brain injury is focused on transport to a trauma center while preventing secondary brain injury. Secondary brain injury occurs through a complex biological cascade, which can continue for hours to days. Both hypotension and hypoxia are independently associated with increased mortality and poorer neurological outcomes [6]. When hypotension and hypoxia occur together, a 75% mortality rate has been reported [7].

**Primary assessment**

The initial management of all injured patients should begin with airway, breathing, and circulation. Adequate oxygenation must be considered a critical priority in brain-injured patients. Hypoxemia occurs more frequently in brain-injured patients than is clinically suspected or recognized. Even a single episode of hypoxemia (SaO2 <90%) can add to the overall morbidity, and has been associated with a 150% increase in mortality [8,9]. Supplemental oxygen should be administered to all potential TBI patients with continuous monitoring of the oxygen saturation using pulse oximetry. Adequate circulation is also important in the head-injured patient. Just a single episode of hypotension, defined as a systolic blood pressure less than 90 mmHg, is associated with increased morbidity, and with a 150% increase in mortality [9]. Intravenous fluids should be administered to maintain a systolic blood pressure of at least 90 mmHg [10]. The optimal fluid choice for volume restoration and maintenance of blood pressure has been intensely debated [11,12]. Isotonic crystalloid is recommended in both adults and children. Alterations in mental status due to hypoglycemia can easily be mistaken for those related to a traumatic brain injury. Patients with altered mental status should have a fingerstick glucose checked in the prehospital setting.

**Secondary assessment**

Performing an efficient neurological assessment is essential in the triage and management of brain-injured patients. Providers will need to repeat and reassess a patient’s neurological status as it frequently changes rapidly ([Table 30.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#c30-tbl-0001)).

[**Table 30.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#R_c30-tbl-0001) Glasgow Coma Scale

| **Eye opening** | **Verbal response** | **Motor response** |
| --- | --- | --- |
|  |  | 6–obeys commands |
|  | 5–oriented | 5–localizes pain |
| 4–spontaneous | 4–confused | 4–withdraws to pain |
| 3–to speech | 3–inappropriate | 3–flexor posturing |
| 2–to pain | 2–incomprehensible | 2–extensor posturing |
| 1–none | 1–none | 1–none |

The Glasgow Coma Scale (GCS) was first introduced in 1974 by Teasdale and Jennett as a way to quickly evaluate the neurological status of brain-injured patients [13]. The GCS has been widely adopted as a way to categorize head injury severity ([Table 30.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#c30-tbl-0002)). The GCS should not be used as a static number and prehospital providers must frequently reevaluate neurological status, assessing for improvement or deterioration. A decrease of two or more points suggests increased ICP related to a potentially enlarging mass lesion (hematoma) [14]. A recent National Trauma Data Bank study of 250,000 head-injured patients found that 9% experienced prehospital neurological deterioration, defined as a decrease in two or more points in GCS from EMS to the emergency department measurement. This patient subgroup had higher in-hospital mortality even after adjusting for type of injury and presence of intracranial hemorrhage. Patients with measurable decline in mental status are high risk, and their initial care and evaluation should reflect the seriousness of this clinical finding [15].

[**Table 30.2**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#R_c30-tbl-0002) Severity of head injury based on GCS

| **Head injury severity** | **Glasgow Coma Scale** |
| --- | --- |
| Mild | 14–15 |
| Moderate | 9–13 |
| Severe | 3–8 |

The GCS has been criticized for insufficient interrater reliability, especially in the outpatient setting [16]. Recent studies have demonstrated that use of a Simplified Motor Scale (SMS) can be as predictive in outcome when compared to the traditional GCS in head-injured patients in both inpatient and out-of-hospital settings [17]. The SMS scale gives only one score: 2 = obeys commands, 1 = localizes to pain, 0 = withdraws to pain or worse. The SMS has not been widely adopted but data are promising that SMS represents an alternative to traditional GCS, especially in the out-of-hospital setting. Current Brain Trauma Foundation guidelines are to continue using the GCS in the prehospital setting for now [10].

The pupils must be evaluated for equality and reactivity to light. Asymmetry is defined as greater than 1 mm difference in diameter, and a fixed pupil is defined as less than 1 mm response to bright light. Unilateral pupillary dilation with decreased reactivity is a sign of increased ICP with uncal herniation causing compression of the ipsilateral third cranial nerve. The eye and orbit should be assessed for signs of direct trauma as unilateral pupillary dilation may be a normal variant. Bilateral pupillary dilation is more likely to be due to a metabolic or toxic cause and, if it is due to trauma, is a poor predictor with mortality reported at 60% [18].

**Other assessment considerations**

Alcohol use results in higher rates of traumatic injury, including head injuries. Intoxicated patients may be agitated or excessively sedated, making initial evaluation difficult. Blood alcohol concentrations above 80 mg/dL have been shown to have a linear effect on GCS [19]. Safety precautions may prompt the use of sedatives such as benzodiazepines, opioids, and antipsychotics. It is preferable to overtriage potentially intoxicated patients to higher levels of care and assume that their changes in consciousness are related to brain injury and not intoxicants alone. Only with time and serial examinations can alterations in mentation be ascribed solely to alcohol or drugs.

Anticoagulant and antiplatelet therapies are commonly used for a variety of medical conditions. Medications that affect platelet function (aspirin), platelet aggregation (clopidogrel), coagulation (warfarin), and thrombin (dabigatran) increase the risk of intracranial hemorrhage after trauma. EMS providers should inquire about the use of “blood thinners” and these patients should be treated with a high degree of concern for intracranial bleeding even in cases of mild head injury.

Penetrating head injuries can be from missiles or impaled objects. Impaled objects should be left in place during transport as these objects will likely need to be removed in a surgical setting. Firearms are the leading cause of TBI death (40%) in the US, with an estimated 68% self-inflicted [20]. The prognosis for penetrating head injuries is quite variable. Approximately two-thirds of patients die prior to hospital arrival. Poor prognostic indicators for the one-third who arrive alive to the hospital include a GCS of 3–5 on arrival, hypotension, bilateral hemisphere involvement, and bilaterally non-reactive pupils [21].

**Prehospital intubation**

Endotracheal intubation helps prevent both hypoxia and aspiration in severely head-injured patients. The controversy regards when the intubation should occur and by whom.

A randomized trial of bag-valve-mask ventilation versus intubation in all children requiring prehospital airway management in Los Angeles County showed no difference in survival or neurological outcomes. When looking specifically at head-injured children, there again was no difference in outcome [22]. This remains the best clinical study to date; however, it was performed in children in an urban EMS system, making generalizability to adult or non-urban settings questionable. Investigators in San Diego have published multiple studies on intubation in head-injured patients, showing consistently poor outcomes attributed at least partially to the adverse effects of inadvertent hyperventilation [23]. They also question whether paramedic inexperience may lead to poor outcomes as they report an average of 0.5 intubations per paramedic annually [24,25]. A randomized trial from Australia restricting intubation with end-tidal carbon dioxide monitoring to highly trained prehospital specialists when transport times were over 10 minutes reported a 97% intubation success rate, with no differences in the primary study outcome of extended Glasgow Outcome Scale at 6 months, but improved neurological outcomes at 6 months and no increase in mortality [26].

Previously, hyperventilation was recommended for severely head-injured patients after intubation to decrease intracranial pressure. While hyperventilation does decrease intracranial pressure, it also decreases cerebral blood flow due to cerebral vasoconstriction, leading to decreased oxygenation of the brain. Currently, mild hyperventilation is indicated for brief periods to treat suspected cerebral herniation. Signs of cerebral herniation include dilated and unreactive pupils, asymmetric pupils, a motor exam that identifies either extensor posturing or no response, or decrease in GCS score by 2 points or more [10]. Hyperventilation goal should be end-tidal CO2 of 30–35 mmHg, monitored with capnography and used only as a *temporizing* measure [10].

An end-tidal carbon dioxide of 35–40 mmHg is recommended for intubated head-injured patients [10]. Unfortunately, inadvertent hyperventilation occurs in as many as 70% of cases, perhaps due to unintentional provider actions or confusion over prior hyperventilation recommendations [27]. Continuous end-tidal capnography is recommended and has been shown to reduce hyperventilation [28].

A Cochrane review article from 2008 suggests that there is no evidence for prehospital intubation in urban, ground transport systems [29]. The Brain Trauma Foundation recommends that EMS systems develop specific protocols that include monitoring of oxygen saturation, blood pressure, and when possible end-tidal carbon dioxide prior to EMS intubations [10]. Prehospital intubation use must be clarified at a local level in the context of transportation distance and time along with local infrastructure and geographical factors.

**Additional treatments**

Mannitol is widely used in the hospital setting to reduce intracranial pressure, which may reduce relative risk of death. There is currently insufficient evidence to recommend the use of mannitol in the prehospital setting [10,30].

In a recent randomized study, prehospital use of hypertonic saline following severe TBI showed no improved outcomes at 6 months, nor change in survival in patients who were not in shock [31]. Normal saline for volume resuscitation to maintain adequate blood pressure, defined as SBP of greater than 90, is currently recommended. The most recent Brain Trauma Foundation guidelines recommend hypertonic fluid resuscitation as an “option” for patients with GCS <8 [10].

The administration of albumin has been shown to worsen outcome in patients with TBI, therefore its use is not recommended [32]. Steroids have been shown to increase the risk of death and are no longer recommended in head-injured patients and are no longer widely used [33]. Seizures resulting from brain injury place excessive metabolic strain on an already injured brain and should be treated quickly to prevent further hypoxic insult. The risk of post-head injury seizures is noted to be higher in children. There is no evidence to treat head-injured patients prophylactically for seizures in the prehospital setting.

The adoption of therapeutic hypothermia in the context of post-cardiac arrest care has led to research into the use of therapeutic hypothermia in severely brain-injured patients [34]. To date, the effectiveness of therapeutic hypothermia for head injury has largely been inconclusive [35–37]. Some have argued that hypothermia treatment has not been initiated early enough in prior trials, and that a difference in outcome may be noticeable if cooling is started closer in time to the injury [38]. Currently there are no randomized controlled studies to support the use of prehospital therapeutic hypothermia in brain-injured patients.

**Sports-related head injuries**

Sports-related head injuries occur frequently, estimated at 3.8 million sports-related concussions annually in the US, with the most common sports being football, hockey, rugby, soccer, and basketball [39]. EMS personnel often provide care at youth athletic events and are the first to evaluate these athletes after injury. All athletes with suspected head injuries should be removed from activities and be evaluated by a medical professional. Athletes who sustain head injuries should not return to play that day. Patients with symptoms such as altered mental status, continued vomiting, retrograde amnesia, and loss of consciousness should be transferred to an emergency department.

**Pediatrics (**[**Table 30.3**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#c30-tbl-0003)**)**

[**Table 30.3**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c30.xhtml#R_c30-tbl-0003) Pediatric Glasgow Coma Scale

| **Eye opening** | **Verbal response** | **Motor response** |
| --- | --- | --- |
|  |  | 6–normal spontaneous movement |
|  | 5–coos, babbles | 5–withdraws from touch |
| 4–spontaneous | 4–irritable crying | 4–withdraws to pain |
| 3–to speech | 3–cries to pain | 3–abnormal flexion |
| 2–to pain | 2–moans to pain | 2–abnormal extension |
| 1–none | 1–none | 1–none |

Prehospital concepts for pediatric traumatic brain injury are similar to those in adults; however, there are some important physiological differences. Children are more susceptible to TBI because of their large heads, thinner bones, and developing brains. Pediatric patients are more prone to brain edema, with more diffuse axonal injuries than primary bleeding or brain contusions. The classic Cushing reflex (hypertension, bradycardia, and respiratory irregularity) is more commonly seen in children. Skull fractures have a significantly higher rate of having intracranial pathology compared to adults, along with a much higher rate of seizures.

Non-accidental trauma must also be considered in pediatric head injuries, especially with injury patterns that do not match the history given by caregivers, or if there are other concerning injuries such as multiple bruises or old, unexplained fractures are found on exam or imaging.

Therapeutic cooling trials have had some positive studies to date in children; however, currently there is not enough evidence to recommend that cooling be initiated in the prehospital setting in children [40].

**Prevention**

The frequency and severity of TBI can be reduced through preventive efforts. Prehospital personnel can identify potential hazards or risk-taking behaviors as they are usually the only health care providers who actually enter a patient’s living environment or witness the scene of a traumatic event. This allows the provider to either directly educate the patient and family or relate appropriate observations to ED staff when the patient is transported to the hospital.

Examples of preventive environmental modifications to reduce the potential for head injury include window guards and safety gates at staircases to prevent children from falling [41]. Falls in the elderly can be reduced by removing loose rugs and encouraging exercise program participation to maintain or improve muscle strength. Firearms in the home should be kept unloaded in a secure, locked container or cabinet with ammunition stored in a different location. Appropriate protective equipment including a helmet should be worn whenever riding any wheeled device or when engaging in contact sports such as hockey, football, boxing, baseball or softball, riding a horse, skiing, or snowboarding. Seat belts should always be used by adults and appropriately sized child safety and booster seats should always be used by children riding in vehicles.

**Transportation and destination decisions**

Destination decision making can significantly alter the outcome of a patient with TBI. When an organized trauma system is in place and patients are taken directly to an appropriate facility, survival from TBI improves [42]. Patients in the moderate or severe TBI group (GCS 13 or less) should be directly transported to a trauma center that is fully equipped and staffed to manage acute neurosurgical emergencies [43]. Patients classified as mild TBI (GCS 14 or 15) can generally be transported to other facilities based on established destination protocols, assuming the patient’s other injuries do not require care at a trauma center [44]. A retrospective evaluation of trauma databases suggested that transporting elderly TBI patients (>70 years) with a GCS of 14 directly to a trauma center may reduce this group’s higher morbidity and mortality [45]. Transport mode (air versus ground) should consider local factors including but not limited to traffic, weather, available transport vehicles, and provider availability to minimize overall prehospital time [10].

**Conclusion**

Traumatic brain injury remains a common cause of disability and death. Injury prevention through public health initiatives such as the use of seat belts and helmets is the mainstay of primary brain injury prevention, with the major goal of treatment being to prevent secondary brain injury.

Secondary brain injury is caused by hypoxia and hypotension. Preventing hypotension and hypoxia are the goals of prehospital care. Prehospital intubations, when performed, should be undertaken by experienced providers with the use of continuous end-tidal monitoring, especially in cases of severe injury with long transfer times. Once intubated, providers should avoid hyperventilation. The mental status of head-injured patients should be continually reassessed as those who have a precipitous deterioration in mental status have increased morbidity and mortality. There is not enough consensus to recommend prehospital cooling, hypertonic saline, or prehospital administration of mannitol in severe head injury patients.