(CSF) can be shunted out of the skull via the ventricles and cisterns. However, after severe TBI, this capacity is quickly overcome, and a rapid rise in ICP with compromised cerebral blood flow and cerebral ischemia ensues. Ultimately, the brain tissue itself may be forced downward across the rigid tentorium or out of the base of the skull itself, thereby resulting in a herniation syndrome and rapid death. Systemic hypoxia and hypotension occur with high frequency in patients with TBI and are associated with increased mortality

**PRESENTING SIGNS AND SYMPTOMS**

**GLASGOW COMA SCALE SCORE**

An altered level of consciousness in the setting of trauma is the primary indicator of TBI requiring an advanced level of care. The Glasgow Coma Scale (GCS) score is the method most commonly used to quantify level of consciousness. In particular, changes in the GCS score over time are highly predictive of outcome, thus mandating repeated assessments both in the field and in the emergency department (ED).

**CLINICAL FINDINGS**

In addition to the GCS score, the initial clinical examination should include assessment for pupil reactivity and symmetry, focal sensorimotor deficits, and cerebellar abnormalities. A thorough cranial examination should be performed to identify external evidence of trauma, potential skull fracture, or evidence of basilar skull fracture. Signs of basilar skull fracture include periorbital (raccoon eyes) or retroauricular (Battle sign) ecchymosis, hemotympanum, and CSF otorrhea and rhinorrhea. In the setting of TBI, any fluid leaking from the nose or ears should be suspected to be CSF. Because of the high coincidence of cervical spine fractures in the setting of severe TBI, the cervical spine should be examined carefully and imaged liberally as clinically indicated.

Historical features that should raise suspicion for serious intracranial injury include a history of loss of consciousness or posttraumatic amnesia, severe headache, vomiting, and confusion. Patients taking anticoagulant medications and those with bleeding disorders are at higher risk for intracranial hemorrhage and should be imaged liberally.

**EPIDEMIOLOGY**

Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality worldwide, with more than 100,000 deaths annually in the United States alone. In addition, an estimated 2 million individuals suffer permanent, life-altering disabilities each year from these injuries. TBI has resulted in a greater number of years of productive life lost than either heart disease or stroke. The most common causes of TBI are falls, motor vehicle collisions, and assault.

**PATHOPHYSIOLOGY**

Hemorrhage or edema following TBI leads to rapid increases in intracranial pressure (ICP). Initially, cerebrospinal fluid (CSF) can be shunted out of the skull via the ventricles and cisterns. However, after severe TBI, this capacity is quickly overcome, and a rapid rise in ICP with compromised cerebral blood flow and cerebral ischemia ensues. Ultimately, the brain tissue itself may be forced downward across the rigid tentorium or out of the base of the skull itself, thereby resulting in a herniation syndrome and rapid death. Systemic hypoxia and hypotension occur with high frequency in patients with TBI and are associated with increased mortality (Fig. 73.1).

**KEY POINTS**

- Emergency treatment of traumatic brain injury is aimed at rapidly identifying surgically correctable lesions and preventing secondary insults such as hypoxemia and hypotension.
- Triage to a neurosurgical trauma center is recommended for patients with intracerebral hemorrhage or persistent altered mental status.
- Early computed tomography is used to identify intracranial hemorrhage in patients with a significant mechanism of injury, history of altered mental status, or risk factors such as anticoagulant therapy.
- Osmotic agents such as mannitol or hypertonic saline are important first-line therapies for patients with elevated intracranial pressure.
- Hyperventilation should be avoided except as a temporizing measure in patients with impending herniation.
- A significant proportion of patients with mild head injury will have persistent postconcussive symptoms. All head-injured patients should be counseled about this possibility and be given appropriate outpatient referral.
- Athletes suffering from concussion should return to play only after completing a supervised stepwise rehabilitation program.
The approach to patients with suspected TBI is directed toward reversal of physiologic derangements and avoidance of secondary insults, early triage to a facility with appropriate resources, and expedited neurosurgical care.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

IMAGING
Non–contrast-enhanced computed tomography (CT) is the initial imaging study of choice in the evaluation of patients with TBI. Plain radiographs are neither sensitive nor specific in identifying intracranial lesions or skull fracture and are therefore not recommended as a diagnostic study. CT has excellent sensitivity in detecting the presence of intracranial hemorrhage, a mass effect such as ventricular compression or midline shift, and the presence of significant cerebral edema. CT also has the advantage of being widely available and rapid. See Box 73.1 for examples of CT findings in patients with TBI.

Other modalities used to diagnose TBI include magnetic resonance imaging (MRI), functional imaging, brain acoustic monitoring, and bispectral electroencephalography. MRI is superior to CT in identifying cerebral edema and diffuse axonal injury. In addition, analysis sequences allow sensitive detection of acute hemorrhage, particularly in the brainstem and posterior fossa, where CT is less sensitive. However, application of MRI in the management of TBI has been limited because of the lack of uniform availability, the increased time needed for administration and patient isolation during the procedure, and the added challenges of resuscitation and ventilator management within the strong magnetic field. For this reason, MRI is used more commonly in the subacute or chronic phases or in patients whose signs and symptoms are not well explained by the findings on CT. Functional imaging with positron emission tomography, single-photon emission CT, xenon-enhanced CT, and MRI-based imaging may also be useful later in a patient’s course to assess cerebral blood flow and oxygenation, which has prognostic value in predicting functional outcomes but is unlikely to be useful in the acute resuscitation and management of patients with TBI in the ED. Finally, newer modalities such as brain acoustic monitoring and bispectral electroencephalography appear to be useful in the detection of TBI because of prognostic ability rivaling that of CT and the ability to provide continuous data. Future investigations should focus on the utility of these modalities in the ED setting.

BOX 73.1 Findings on Computed Tomography in Individuals with Traumatic Brain Injury

Acute hemorrhage appears hyperdense on computed tomography (CT) scans, with the shape and location of hemorrhage suggesting the underlying pathology. Epidural hematomas are classically lentiform (lens shaped) because of their relationship to arterial injury, with the higher pressure compressing the brain parenchyma (Fig. 73.2). Subdural hematomas are more commonly crescent shaped, with blood from torn veins tracking along the surface of the brain beneath the dura mater (Fig. 73.3). Intraparenchymal hemorrhage can exist as a discrete hematoma or as multiple smaller foci throughout a contused area of brain (Fig. 73.4). In addition to focal areas of hemorrhage, cerebral contusions typically involve cerebral edema, which may progress markedly over a period of several days. Skull fractures may be seen on plain radiographs, but more important is the potential for injury to the underlying brain parenchyma or the existence of intracranial hemorrhage (Fig. 73.5). Subarachnoid hemorrhage appears as hyperdensities within the ventricles, along the falx and tentorium, and around the circle of Willis (Fig. 73.6). One of the most elusive diagnoses is diffuse axonal injury, in which the findings on CT are often much less impressive than the degree of obtundation. Small, punctate hemorrhages along the gray-white interface at the cortical periphery suggest this diagnosis, although the initial scan may be completely normal.

TREATMENT

GENERAL
The approach to patients with suspected TBI is directed toward reversal of physiologic derangements and avoidance of secondary insults, early triage to a facility with appropriate resources, and expedited neurosurgical care.
PREHOSPITAL MANAGEMENT
Half of all patients who die of TBI do so within the first few hours after their injury.

Prehospital assessment of patients with TBI should include rapid airway evaluation, continuous pulse oxygen saturation monitoring, frequent measurement of blood pressure, determination of GCS scores, and pupillary evaluation. Prehospital intubation should be avoided in patients who are spontaneously breathing and maintaining greater than 90% oxygen saturation. Prehospital airway management may be necessary in patients with a GCS score lower than 9 or those unable to maintain oxygen saturation greater than 90% with supplemental oxygen. If prehospital endotracheal intubation is performed, confirmation of placement should be done with auscultation and end-tidal capnography. Even mild hyperventilation should be avoided in all cases with the exception of patients who have evidence of herniation or acute neurologic deterioration. Hypotension should be treated with isotonic fluids, although protocols involving prehospital hypertonic saline administration are reasonable for patients with GCS scores lower than 9. Rapid transport is a priority, ideally to a facility with immediately available imaging and neurosurgical care.
AIRWAY AND BREATHING

Trauma data registries have shown increased mortality in TBI patients with hypoxemia. This association has led to an aggressive approach to airway management in patients with severe TBI, including oxygen supplementation and early intubation. All patients with TBI should undergo pulse oximetry monitoring and administration of supplemental oxygen to correct the hypoxemia (Pao₂ < 60 mm Hg or oxygen saturation < 90%).

In patients with severe TBI, endotracheal intubation is critical. Indications for endotracheal intubation include a GCS score lower than 9, airway protection when airway protective reflexes are in question, hypoxia refractory to supplementary oxygen, and agitated or combative patients who cannot comply with a rapid and thorough assessment, including CT.

Rapid-sequence intubation (RSI) is the recommended strategy for securing the airway in patients with TBI because of its ability to produce optimal intubation conditions and minimize the adverse effects of laryngoscopy on the injured brain. The most serious risk associated with RSI is the potential for apnea and hypoxia during paralysis. Aggressive preoxygenation and early use of adjunctive airway measures can minimize hypoxic insults.

Patients with TBI may be sensitive to the increases in ICP associated with RSI, and some experts recommend neuroprotective adjuncts to traditional RSI medications. Preadministration of lidocaine at a dose of 1.5 to 2 mg/kg may blunt the rise in ICP associated with laryngoscopy and intubation. Coadministration of 2.5 to 3 mcg/kg of fentanyl with the induction agent may prevent the tachycardia and hypertension.
associated with tracheal intubation. Finally, a small dose of a nondepolarizing neuromuscular agent, such as pancuronium, before administration of succinylcholine can protect against fasciculations, which may lead to a rise in ICP. The “defasciculation” dose is typically one tenth of the full paralytic dose of the agent. Box 73.2 presents a sample neuroprotective RSI strategy. It should be noted that although all the aforementioned adjuncts to RSI in patients with TBI are reasonable considerations, they should be implemented as part of a streamlined approach to TBI patients. The higher priority is rapid and safe intubation that avoids hypoxia and aspiration. If implementation of these adjuncts to traditional RSI results in delay or complications, all theoretic benefit is lost. Therefore, it is also reasonable to use traditional RSI in TBI patients without medications.

The postintubation ventilation strategy significantly influences outcomes in patients with TBI. This reflects the adverse effects of positive pressure ventilation on cardiac output, hypocapnic cerebral vasoconstriction, and retrograde cerebral transmission of intrathoracic pressure via the jugular venous system, all of which can lead to cerebral hypoperfusion and ischemia. PaCO₂ should be maintained as close to normal as possible, within the range of 30 to 39 mm Hg. Hyperventilation does decrease ICP, but it does so by causing cerebral vasoconstriction and results in decreased cerebral blood flow. Transient hyperventilation, to a PaCO₂ in the range of 30 to 35 mm Hg, is therefore reserved as a temporizing measure in the setting of acute deterioration as a means of avoiding herniation. Serial arterial blood gas analysis or PETCO₂ monitoring should be performed after intubation to ensure proper ventilation because traditional pulse oximetry monitoring reflects only oxygenation status.

CIRCULATION
Systemic hypotension has been associated with increased mortality in studies of TBI. Although the normal brain can maintain cerebral blood flow despite a range of mean arterial pressure levels, this ability appears to break down following TBI. In addition, intracranial hypertension requires higher mean arterial pressure to maintain adequate cerebral perfusion pressure. Every effort should be made to avoid hypotension in TBI patients and maintain systolic blood pressure lower than 90 mm Hg at all times if possible.

The use of pressors is generally discouraged in the initial resuscitation of multiple-trauma victims. Thus, the main focus of therapy is volume replacement with intravenous fluids and blood products. Albumin and nonprotein colloids have no role in the initial resuscitation of patients with TBI. Hypertonic saline solutions appear to have osmotic properties that may lower ICP and decrease cerebral edema. However, outcome data have been disappointing, and at this time the initial resuscitation fluid of choice remains standard crystalloid (lactated Ringer or 0.9% saline solution).

AVOIDING CEREBRAL HERNIATION
The development of unilateral papillary dilation, hemiparesis, or deterioration in the level of consciousness is very concerning for the devastating complication of cerebral herniation. Heroic measures are in order. Aside from definitive neurosurgical management, the emergency physician should perform two interventions in this setting: hyperosmolar therapy and hyperventilation.

Patients with cerebral herniation should receive mannitol, 0.25 to 1 g/kg in bolus form, repeated every 4 to 6 hours. Mannitol is an osmotic diuretic that can decrease brain edema, reduce blood viscosity, and cause a transient increase in circulating volume that improves cerebral blood flow and oxygenation. Hypertonic saline at concentrations of 7.2% to 23.4% has been studied as an alternative or adjunct to mannitol with favorable results. This is a promising therapy that has been beneficial in multiple small studies, but at the time of this writing, evidence has not yet reached the level to change practice guidelines, and we therefore still recommend mannitol as the first-line hyperosmolar therapy.

Hyperventilation temporarily lowers ICP as a result of cerebral vasoconstriction. As stated earlier, the decrease in ICP seen with hyperventilation comes at the price of an even greater compromise in cerebral blood flow, which ultimately results in cerebral ischemia. Impending herniation is the one clinical setting in which transient hyperventilation, to a PaCO₂ value of 30 to 35 mm Hg, should be initiated as a temporizing measure until hyperosmolar therapy takes effect or surgical decompression can be performed.

SEIZURE PROPHYLAXIS
Seizures in patients with TBI have a constellation of effects, including elevated ICP, hypoxia, hypercapnia, and massive release of excitatory neurotransmitters, all of which result in secondary insults to an already injured brain. Seizures should be treated immediately with benzodiazepines. There is no evidence that early seizure prophylaxis decreases the likelihood that a delayed seizure disorder will develop, but prophylaxis does decrease the likelihood of early seizures (within the first 7 days after injury). The decision to empirically administer seizure prophylaxis should weigh the likelihood of seizures against the possible side effects of medication. There is currently no consensus regarding which patients should receive prophylaxis, but it is reasonable to consider administration in high-risk patients. Patients at high risk for seizures include those with a GCS score lower than 10, cerebral contusion or hematoma, depressed skull fracture, and penetrating injury. The first-line agent for seizure prophylaxis is phenytoin.
STEROIDS IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Corticosteroids do not improve outcome in TBI patients, and their use is associated with harmful complications. Steroids should not be administered as treatment of TBI.

MILD TRAUMATIC BRAIN INJURY

OVERVIEW

Current practice in EDs and trauma centers focuses on identifying TBI patients who require emergency neurosurgical intervention or close monitoring. However, about two thirds of patients with TBI in the United States sustain mild injury and are discharged home after a brief observation period. Mild TBI is generally defined as blunt injury to the head with resulting symptoms (which may or may not include loss of consciousness) and a GCS score higher than 13. For the emergency physician there are three important components of the management of every patient with mild TBI: when to image, disposition planning, and discharge instructions.

IMAGING

As discussed earlier, the modality of choice for the initial imaging of patients after TBI is non–contrast-enhanced CT. The prevalence of CT abnormalities is about 5% in patients arriving at the hospital with a GCS score of 15 and increases significantly with a lower initial GCS score. Approximately 1% of all patients with mild TBI ultimately require neurosurgical intervention. Several clinical decision rules designed to help clinicians decide which patients with mild TBI require imaging have been validated. Their recommendations differ because of variations in definitions and study populations. That said, certain features are consistently associated with intracranial pathology, such as older age, vomiting, focal neurologic deficit, and persistent alteration in level of consciousness. Recent consensus guidelines have been developed to guide imaging decisions and are summarized in Box 73.3.

FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION

DISPOSITION

Patients suffering TBI who have abnormalities on head CT or persistent alteration in mental status should be admitted to a hospital with neurosurgical capability and be closely monitored for deterioration (Fig. 73.7). There is good evidence that patients with head injury who have normal findings on head CT and neurologic examination can be safely discharged from the ED without an extended observation period. It must be noted that the studies that came to this conclusion excluded certain populations, in particular, patients with bleeding disorders and those taking anticoagulant medications. Therefore, the data are insufficient to be certain that this recommendation is safe in these patients. We recommend a more careful approach to patients with acquired or inherited bleeding diatheses: either close observation in the hospital or discharge in the care of responsible caregivers who can watch the patient closely for signs of deterioration and have rapid access to return to the hospital for reevaluation. It is reasonable to include patients and their families in this decision and have an open discussion of the uncertainty in our understanding of the risk for deterioration in patients with TBI.

PATIENT TEACHING

The most important consideration when counseling patients sent home from the ED is to communicate the signs and symptoms of an evolving intracranial process, especially in patients who have not undergone CT imaging. Such signs and symptoms include worsening headache, persistent

BOX 73.3 Consensus Guidelines to Direct Imaging Decisions

A non–contrast-enhanced head computed tomography (CT) scan is indicated in head trauma patients with loss of consciousness or posttraumatic amnesia if one or more of the following is present:

• Glasgow Coma Scale score lower than 15
• Vomiting
• Age older than 60 years
• Drug or alcohol intoxication
• Deficits in short-term memory
• Physical evidence of trauma above the clavicle
• Posttraumatic seizure
• Headache
• Focal neurologic deficit
• Coagulopathy

A non–contrast-enhanced head CT scan should be considered in head trauma patients with no loss of consciousness or posttraumatic amnesia if one or more of the following is present:

• Focal neurologic deficit
• Vomiting
• Severe headache
• Age 65 years or older
• Physical signs of a basilar skull fracture
• Glasgow Coma Scale score lower than 15
• Coagulopathy
• Dangerous mechanism of injury
  – Ejection from a motor vehicle
  – Striking of a pedestrian
  – A fall from a height of more than 3 feet or 5 stairs

Fig. 73.7  Algorithm depicting the differential diagnosis of traumatic brain injury. CT, Computed tomography; GCS, Glasgow Coma Scale; RSI, rapid-sequence induction.

Table 73.1  Stepwise Progression of Rehabilitation After Concussion

<table>
<thead>
<tr>
<th>REHABILITATIVE STAGE</th>
<th>FUNCTIONAL EXERCISE AT EACH STAGE OF REHABILITATION</th>
<th>OBJECT OF EACH STAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No activity</td>
<td>Complete physical and cognitive rest</td>
<td>Recovery</td>
</tr>
<tr>
<td>2. Light aerobic exercise</td>
<td>Walking, swimming, or stationary cycling while keeping the intensity at &lt;70% of the maximum predicted heart rate; no resistance training</td>
<td>Increase the heart rate</td>
</tr>
<tr>
<td>3. Sport-specific exercise</td>
<td>Skating drills in ice hockey, running drills in soccer; no head impact activities</td>
<td>Add movement</td>
</tr>
<tr>
<td>4. Noncontact training drills</td>
<td>Progression to more complex training drills, such as passing drills in football and ice hockey; may start progressive resistance training</td>
<td>Exercise, coordination, and cognitive load</td>
</tr>
<tr>
<td>5. Full-contact practice</td>
<td>Following medical clearance, participation in normal training activities</td>
<td>Restore athlete’s confidence; coaching staff assesses functional skills</td>
</tr>
<tr>
<td>6. Return to play</td>
<td>Normal game play</td>
<td></td>
</tr>
</tbody>
</table>

If symptoms recur during any step, the patient should return to step 1. If asymptomatic, the patient may advance to the next step every 24 hours.

vomiting, confusion, and balance problems. Any of these findings should prompt return to the ED for immediate reevaluation and consideration of appropriate imaging studies.

It is also important to communicate to patients the possibility of postconcussive syndrome (PCS). This syndrome of persistent neurologic, behavioral, and cognitive symptoms develops in a substantial proportion of patients with mild TBI. Symptoms include headache, memory impairment, difficulty concentrating, anxiety, and depression. Of all patients with mild TBI, about 50% at 3 months and 15% at 1 year will have persistent PCS. Even though no specific treatments are available for PCS, early psychosocial intervention appears to reduce postconcussion symptoms and limit the emergence of persistent problems. Awareness can help validate symptoms that might otherwise not be attributed to the traumatic incident and lead to referral for neurorehabilitation or psychologic services or support groups.

CONCUSSION AND RETURN TO PLAY

The term concussion refers to short-lived impairment of neurologic function after trauma. Such impairment may or may not involve loss of consciousness and typically resolves over a sequential course, although postconcussive symptoms may be prolonged. A primary concern of emergency physicians and trainers is second-impact syndrome, which has been reported in athletes who return to play while still symptomatic from a concussion and sustain another head injury. These athletes, despite having mild symptoms and sustaining apparently mild second injuries, are at risk for the rapid development of brain swelling, herniation, and death. Additionally, athletes who sustain repeated concussions are at higher risk for long-term cognitive deficits. A stepwise progression of rehabilitation after concussion is recommended before return to play (Table 73.1), ideally supervised by an experienced athletic trainer or health care provider.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES