

Closed Head Injury

- Author: Eric Deutsch MD, PGY2, Emory University SOM
- Author: Joshua Wallenstein MD, Asst Professor, Emory University SOM
- Editor: Rahul Patwari, Rush University, Chicago, Illinois.
- Last Updated: 2016

Introduction

Head injuries are a common presentation to the Emergency Department. While most are minor requiring no further work up beyond a history and physical, others may be severe requiring prompt recognition and treatment to avoid significant morbidity and mortality. The Emergency Physician needs to quickly identify concerning signs and symptoms that require neuroimaging and intervention.

Objectives

- Calculate a Glasgow Coma Score
- Discuss the various types of head injury
- Decide which patients with head injury require imaging
- Recognize the Cushing reflex and herniation syndromes

• Describe bedside temporizing measures for patients with concern for increased intracranial pressure

The Glasgow Coma Scale (GCS) is used to define severity of traumatic brain injury (TBI). The score measures the patients level of consciousness independent of their specific injury mechanism. The patient's best score from each of three categories (Table 1) is added together to determine the final score, which ranges from 3 to 15. Scores of 14 or 15 are considered mild TBI and are low risk for significant morbidity and mortality. Moderate TBI encompasses scores of 9 to 13, with mortality rates of <20% but long-term disability as high as 50%. Scores of 8 and lower define severe TBI with high rates of mortality and long term disability, approaching 40% and 90% respectively. Note that these rates are for isolated head injuries and may differ with concomitant injuries.

Glasgow Coma Scale				
Score	Eye Opening	Verbal Response	Motor Response	
6	N/A	N/A	Obeys commands	
5	N/A	Oriented, converses normally	Localizes painful stimulus	
4	Opens eyes spontaneously	Confused, disoriented	Flexion, withdrawal to painful stimulus	
3	Opens eyes in response to voice	Utters inappropriate words	Abnormal flexion to painful stimulus (decorticate response)	
2	Opens eyes in response to painful stimulus	Incomprehensible sounds	Extension to painful stimulus (decerebrate response)	
1	Does not open eyes	Makes no sounds	Makes no movements	

Initial Active and Primary Survey

As with all patients in the emergency department, head injury patients should be evaluated and treated according to the ABCs with a few additional points. Patients with head injury are at risk of concomitant spinal injury. Spinal precautions should be maintained throughout the workup until spinal injury can be excluded, including application of a cervical collar and supine positioning.

The injured brain is extremely susceptible to further injury, and even brief, transient episodes of hypoxia or hypotension may have profound effects on prognosis. A GCS score should be quickly calculated, as all patients with severe TBI (GCS of 8 or lower) require control of their airway and breathing to ensure that hypoxia does not develop. Rapid sequence intubation should be performed promptly while maintaining manual in-line cervical spine stabilization. Care should be taken not to hyperventilate the patient, as low PaCO2 levels may cause excess cerebral vasoconstriction and resultant hypoxemia.

Once the airway has been controlled attention should be turned to circulation. Episodes of hypotension result in decreased cerebral perfusion pressure (CPP) which may exacerbate injury. Current guidelines recommend maintenance of a mean arterial pressure >80 mm Hg to maintain adequate CPP. This may be accomplished through fluid boluses or vasopressors if necessary. Hypotensive patients should be evaluated for signs of active bleeding, which should be addressed rapidly if identified. If the patient is hypertensive it should prompt assessment for other signs of increased intracranial pressure (ICP) (make this a link to the other section: to be discussed in further detail later in this chapter).





As previously mentioned, a GCS score should be calculated during the primary survey of all head injury patients. Attention should also be paid to the pupillary exam and motor response in all extremities. Abnormalities may be a sign of herniation and require further evaluation (to be discussed in further detail later in this chapter).

Presentation

Once the primary survey is complete and the patient has been stabilized, a full history and physical exam should be performed. The physician should ascertain the mechanism and timing of the injury, any ongoing neurologic symptoms (ie alterations of mental status, headache, dizziness, vision or hearing changes, weakness, numbness), or episodes of vomiting. Past medical history and medications should be noted, with particular concern for bleeding disorders or anticoagulants use. Intoxication with alcohol or other substances may complicate clinical picture. In addition to the GCS and pupil exam performed during the primary survey, physical exam should also include inspection and palpations of any external injuries, looking for step-offs or instability, as well as a full neurologic exam in any cooperative patient.

There are several types of injury that may occur as a result of head trauma:

Epidural Hematoma

An epidural hematoma occurs between the skull and dura mater. On CT scan this appears as a biconvex hyper-dense collection of blood that does not cross suture lines (Image 1). It typically occurs from a blow to the temporal region with disruption of the middle meningeal artery. Because the bleed is arterial it can progress rapidly and requires prompt intervention. The classic presentation consists of a blow to the head with a brief loss of consciousness, followed by a lucid period, then rapid neurologic demise, although this sequence occurs in only a minority of patients.



(https://cdemcurriculum.files.wordpress.com/2016/11/955ae06d-8d61-4fa5-ae66-fbe3e390d857.jpg)

Subdural Hematoma

A subdural hematoma (SDH) collects between the dura and arachnoid mater, appearing as a crescent shape that may cross suture lines on CT scan (Image 2). Depending on timing, they may appear as hyper-dense (acute, 0-3 days post-injury), isodense (subacute, 3-14 days post-injury), or hypodense (chronic, >14 days post-injury). They result from shearing of the bridging veins from a sudden acceleration-deceleration injury. Individuals with high degree of cerebral atrophy, such as the elderly and alcoholics, are at higher risk from this type of injury. As the bleeding is venous, they may progress slowly and not present until days or weeks after the initial injury.



(https://cdemcurriculum.files.wordpress.com/2016/11/73b8f488-2e67-405c-8201-950cab916f17.jpg)

Acute subdural hematomas typically occur after a significant trauma and may occur in patients of any age. Chronic SDH, on the other hand, occur in those with significant atrophy as a result of minor trauma that the patient may not even recall. Patients will typically present with vague symptoms such as weakness or confusion. A high level of suspicion must be maintained in elderly patients presenting with alterations in mental status or other neurologic complaints.

Traumatic Subarachnoid Hemorrhage

Head injury may also cause bleeding into the subarachnoid space from a disruption of the subarachnoid vessels. This appears as hyper-dense blood within the CSF on CT scan (Image 3). Patients may complain of severe headache, meningeal signs, or photophobia. These injuries may be missed on early CT scans, with improved sensitivity 6-8 hours post-injury.



(https://cdemcurriculum.files.wordpress.com/2016/11/cde3a2f1-1468-46db-b9f1-687968c04e2a.jpg)

Cerebral Contusion and Intracerebral Hemorrhage

Contusions typically occur in the frontal and temporal lobes or occasionally the occipital lobes. They may occur at the site of trauma or the opposite side of the brain (contracoup injury). Depending on the areas of the brain involved they may present with focal neurologic deficits. Contusions may convert to intracerebral hemorrhage (ICH) several days after the initial injury, so any changes in neurologic status should be investigated by serial CT scans. ICH appears as hyper-dense blood within the brain parenchyma (Image 4).



Diffuse Axonal Injury

Diffuse axonal injury results from shearing forces on axonal fibers of the white matter and brainstem. The mechanism of injury is typically a sudden deceleration, such as from a motor vehicle collision. Initial CT scans may be read as negative, although classically display punctate hemorrhages along the gray-white junction of the cerebral cortex and in deep structures of the brain. Edema can develop rapidly requiring prompt intervention. Prognosis can be devastating, often with permanent deficits if the initial injury is survived.

Skull Fractures

Patients with head injury should be carefully assessed for signs of skull fracture. Any lacerations should be meticulously explored for underlying fractures. Signs of basilar skull fracture include CSF otorrhea or rhinorrhea, hemotympanum, periorbital or retroauricular ecchymosis ("raccoon eyes" and Battle signs), deafness, or 7th nerve palsy. Fractures may be classified as linear, depressed, or comminuted. Any fractures with overlying lacerations are considered open and IV antibiotics should be given.

Increased Intracranial Pressure and Herniation

The cranium is an enclosed structure with a fixed volume, normally occupied by the brain parenchyma, CSF, and blood within the vessels and sinuses. Because there is no room for expansion of this space, if there is significant bleeding or edema within the cranium it results in an increase in the intracranial pressure (ICP). An increase in the ICP may result in decreased perfusion of the brain, causing hypoxemia and further exacerbating injury. The response to increasing ICP is termed the Cushing reflex, consisting of hypertension, bradycardia, and respiratory irregularity.

Increased ICP may also result in herniation of the brain parenchyma. There are four herniation syndromes.

- 1. Uncal herniation occurs when the uncus of the medial temporal lobe is displaced inferiorly through the tentorium. This results in an ipsilateral fixed and dilated pupil and contralateral motor paralysis.
- 2. Central transtentorial herniation occurs from midline lesions of the frontal or temporal lobes with downward displacement of the parenchyma through the tentorium. Clinical features include bilateral pinpoint pupils, bilateral Babinski's signs, and increased muscle tone.
- 3. Cerebellotonsillar herniation occurs when the cerebellar tonsils are displaced through the foramen magnum, resulting in bilateral pinpoint pupils, flaccid paralysis, and sudden death.
- 4. Upwards transtentorial herniation results from posterior fossa lesions, consisting of conjugate downward gaze with lack of vertical eye movements and pinpoint pupils. Development of the Cushing reflex or a herniation syndrome requires prompt intervention to relieve intracranial pressure and prevent further injury.

Mild TBI and Concussion

A mild TBI, also known as a concussion, is defined as a GCS score of 14 or 15 with associated signs or symptoms after a blunt force or accelerationdeceleration head injury. Signs and symptoms may include headache, vomiting, weakness, numbness, dizziness, decreased concentration, memory problems, sleep disturbance, irritability, fatigue, visual disturbances, depression, or anxiety. While there are no specific treatments, it is important to distinguish patients at risk for more serious injury that may require imaging and further intervention (further discussed in Diagnostic Testing section). Patients with negative imaging or in whom it has been determined that imaging is not necessary may be discharged, but require follow up with either their primary care doctor or mTBI specialist if symptoms persist. Those patients should be advised to avoid sports or other activities that put them at risk for further head injury until medically cleared. A graded return-to-activity program is recommended before full resumption of normal activity. Repeat concussion during the vulnerable healing period following an initial concussion can result in second impact syndrome, with rapid onset of cerebral edema and death.

Diagnostic Testing

The main decision point in the assessment of a patient with head injury is whether or not to obtain a CT scan. All patients with head injury and a GCS less than 15, neurologic deficits, coagulopathy, or on anticoagulants should undergo a non-contrast CT scan of the head to assess for intracranial injury. For patients not meeting those criteria, two evidence-based decision rules, the New Orleans Criteria and the Canadian CT Head Rules, have been developed to further assess the need for CT scan (Table 2). Both sets of criteria have 100% sensitivity for detection of patients requiring neurosurgical intervention. Specificity of the New Orleans Criteria is 5%, while the Canadian CT Head Rules is 38%. The major difference in specificity between the rules is due to the New Orleans Criteria being developed to detect all patients with abnormality on CT, while Canadian CT Head rules account for only patients requiring intervention. As a result, sensitivity for detection of any intracranial abnormality is 100% for the New Orleans Criteria, while only 83% for the Canadian Head CT Rules.

CT Head Decision Rules			
New Orleans Criteria	Candian CT Head Rule		
Obtain head CT if any of the following are present			
Headache	GCS < 15 at 2 hours after injury		
Vomiting	Suspected open or depressed skull fracture		
Age > 60 years	Any sign of basilar skull fracture		
Intoxication	Vomiting > 1 episode		
Persistent antegrade amnesia	Retrograde amnesia > 30 minutes		
Evidence of trauma above the clavicles	Dangerous mechanism (pedestrian struck by vehicle, ejection from motor vehicle, fall from more than 3 feet or 5 stairs)		
Seizure	Age > 65 years		

Treatment

ABCs should be addressed as previously discussed. Depending on injury, clinical course may change rapidly so frequent reassessment and appropriate intervention is crucial. Neurosurgical consultation should be obtained immediately in patients with pathology on CT scan or clinical signs of increased ICP or herniation. Emergent surgical decompression may be necessary to prevent impending herniation. If neurosurgery is not available at your center, arrangements should be made for rapid transfer to a trauma center. Several temporizing bedside measures may be initiated to decrease ICP prior to definitive intervention. The head of the bed should be elevated to 30 degrees. If there is concern for spinal injury this may be accomplished via reverse Trendelenburg positioning. Mannitol may be used as an osmotic diuretic if the patient is not hypotensive, or hypertonic saline if hypotension is present. If not done previously, the patient should be intubated and adequately sedated to prevent agitation and increase in ICP. Seizures should be treated with benzodiazepines and IV antiepileptics. If there is concern for impending herniation a temporary course of hyperventilation may be instituted, with a goal PaCO2 of 30-35 mm Hg.

In patients with coagulopathy found to have intracranial bleeding, reversal should be instituted immediately. This may include administration of vitamin K, fresh frozen plasma (FFP), prothrombin complex concentrate (PCC), or other factors depending on your institutions policies.

Patients with intracranial injury on CT scan, persistently abnormal GCS, or persistent neurologic deficits require admission to the hospital for further observation even if intervention is not immediately indicated. Those who do not meet admission criteria may be discharged home with a reliable companion who can observe them for at least 24 hours with proper care instructions and return precautions.

Pearls and Pitfalls

- Maintain spinal precautions (c-collar, supine positioning) in all head injury patients until spinal injury can be excluded
- When preparing to intubate, note a GCS, pupillary exam, and motor function in all extremities prior to sedating and paralyzing.
- Patients with head injury require higher MAP goals (>80 mm Hg) than typical trauma patients.
- Watch for signs of increased ICP or herniation, including Cushing's reflex, abnormal pupillary exam, or abnormal motor exam. Treat with elevation of the head of the bed, mannitol or hypertonic saline, controlled hyperventilation, and emergent neurosurgical consultation.
- The elderly, chronic alcoholics, and those with coagulopathies or on anticoagulants are at high risk for significant injury from seemingly minor mechanisms have a low threshold for imaging in these patients.
- Reverse coagulopathy emergently if intracranial bleeding is revealed by imaging.
- Negative imaging and a normal exam does not exclude injury. Patients with concussions should be given proper care and follow up instructions prior to discharge.

References

• Emergency Medicine: A Comprehensive Study Guide, 7th ed, Chapter 254, "Head Trauma in Adults and Children", DW Wright and LH Merck.