Chapter 257: Head Trauma

David W. Wright; Lisa H. Merck

INTRODUCTION AND EPIDEMIOLOGY

Traumatic brain injury is brain function impairment that results from external force.[1] The clinical manifestations represent a broad constellation of symptoms from brief confusion to coma, severe disability, and/or death. The underlying pathology ranges from temporary shifts in cellular ionic concentrations to permanent structural damage.

Traumatic brain injury (TBI) is classified as mild, moderate, and severe based on the Glasgow Coma Scale (GCS) score. Over 80% of TBI is defined as **mild** (GCS 14 to 15) (**mTBI**) and is often called "concussion."[2] The label of mild, however, is a misnomer. mTBI may lead to significant, debilitating short- and long-term sequelae. **Moderate** TBI (GCS 9 to 13) accounts for approximately 10% of head injuries. Mortality rates for patients with isolated moderate TBI are <20%, but long-term disability can be higher. Overall, 40% of patients with moderate TBI have an abnormal finding on CT scan, and 8% will require neurosurgical intervention. In **severe** TBI (GCS 3 to 8), mortality rate approaches 40%, with most deaths occurring in the first 48 hours after injury. Fewer than 10% of patients with severe TBI experience good recovery.[2,3]

The prevalence of TBI is twice as high in males as in females. Distribution of age at injury is trimodal, with peaks at 0 to 4 years, 15 to 24 years, and >75 years of age. Mortality rate increases with age at time of injury.[4,5] Motor vehicle collisions are the primary cause of blunt head injury in young adults and children, and falls are more common in the elderly.[2] TBI has been called the "signature injury" of the conflicts in Iraq and Afghanistan.[6]

PATHOPHYSIOLOGY

CEREBRAL BLOOD FLOW

Autoregulation, cerebral perfusion pressure (CPP), mean arterial pressure (MAP), and intracranial pressure (ICP) are interrelated factors that affect cerebral blood flow (Table 257-1). Under normal circumstances, **autoregulation** regulates local cerebral blood flow to maintain equilibrium between oxygen delivery and metabolism.[7] Other systemic factors, such as hypertension, hypocarbia, and alkalosis, can affect cerebral blood flow by causing vasoconstriction.

TABLE 257-1

Factors that Affect Cerebral Blood Flow

MAP = DBP + [(SBP – DBP)/3]
CPP = MAP – ICP

Abbreviations: CPP = cerebral perfusion pressure; DBP = diastolic blood pressure; ICP = intracranial pressure; MAP = mean arterial pressure; SBP = systolic blood pressure.

Under normal situations, autoregulation can adjust to CPPs from 50 to 150 mm Hg to maintain local cellular oxygen demands and regional cerebral blood flow. In brain injury, autoregulation is often impaired, so even modest drops in blood pressure can decrease brain perfusion and result in cellular hypoxia. A CPP <60 mm Hg is considered the lower limit of autoregulation in humans, below which local control of cerebral blood flow cannot be adjusted to maintain flow adequate for function.[8] Traumatic hypotension leads to ischemia within low flow regions of the injured brain, so aggressive fluid resuscitation may be required to prevent hypotension and secondary brain injury. In the absence of an ICP monitor, it is important to maintain a MAP of **≥80 mm Hg**, because **low blood** pressure in the setting of elevated ICP will result in a low CPP and brain injury.

The cranium is an enclosed space with a fixed volume. Any changes to the volume of the intracranial contents (such as bleeding) affect the **ICP**, and an increase in ICP can decrease the CPP. ICP is determined by the volume of the three intracranial compartments: the brain parenchyma (<1300 mL in the adult), cerebrospinal fluid (100 to 150 mL), and intravascular blood (100 to 150 mL). When one compartment expands, there is a compensatory reduction in the volume of another, and/or the baseline ICP will increase (Figure 257-1). Elevations in ICP are life threatening and may lead to a phenomenon known as the **Cushing reflex** (hypertension, bradycardia, and respiratory irregularity). Hypertension is an attempt to maintain cerebral perfusion. Normal values for ICP vary with age (Table 257-2).

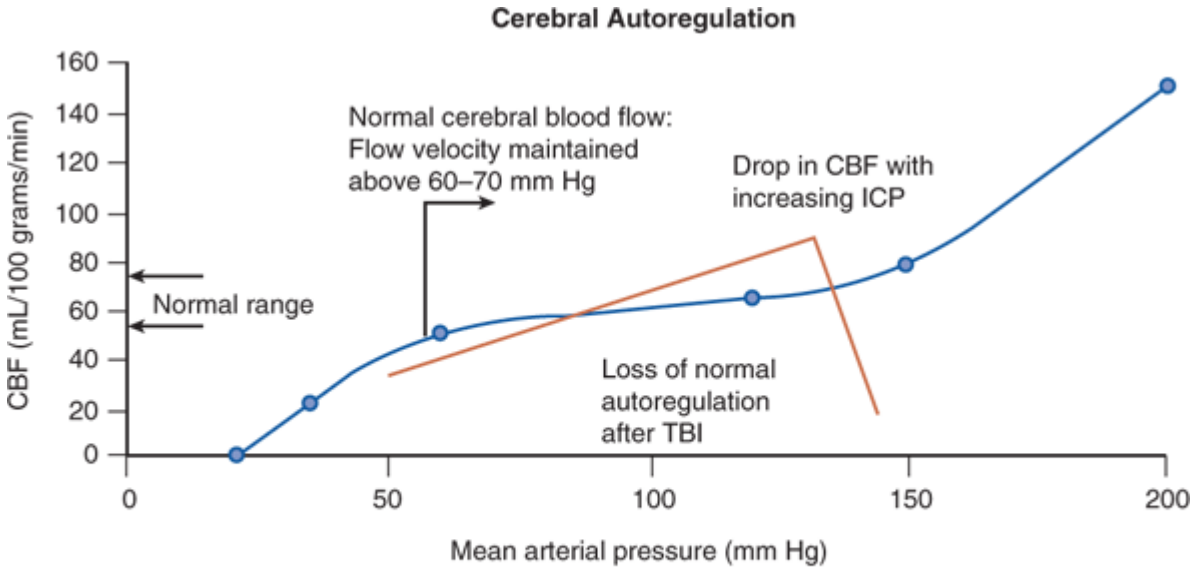
TABLE 257-2

Intracranial Pressure by Age Group

Age Group	Intracranial Pressure (mm Hg)
Adults	<10–15
Young children	3–7
Infants	1.5–6.0

FIGURE 257-1.

Pressure–volume relationship in brain injury. Normal cerebral blood flow autoregulation curve and the abnormal curve with traumatic brain injury (TBI). Normal autoregulatory control (*blue line*) maintains a relatively constant cerebral blood flow over a broad range of mean arterial pressure (MAP).9 Loss of autoregulation results in a more linear relationship between cerebral blood flow (CBF) and MAP. Elevated intracranial pressure (ICP) can dramatically decrease CBF when autoregulation is impaired (*inflection point of red line*). Increases in ICP may result in a net loss in CBF.



PRIMARY BRAIN INJURIES

The initial insult associated with moderate and severe TBI imparts mechanical forces that produce high levels of direct damage and strain to the brain parenchyma. The **primary injuries** include contusions (bruises to brain parenchyma), hematomas (subdural, epidural, intraparenchymal, intraventricular, and subarachnoid), diffuse axonal injury (stress or damage to axons), direct cellular damage (neurons, axons, and other supportive cells), loss of the blood–brain barrier, disruption of the neurochemical homeostasis, and loss of the electrochemical function.

SECONDARY BRAIN INJURIES

A wave of secondary damage is unleashed by the impact that results in a series of deleterious cellular and subcellular events (also known as the **secondary neurotoxic cascade**).[10,11] The secondary neurotoxic cascade causes ongoing damage to the brain and ultimately results in a poorer neurologic outcome than might have occurred based on the original mechanism.

The secondary neurotoxic cascade should not be confused with the term **secondary insults**, a term used in the clinical literature to describe conditions or circumstances (e.g., hypotension, hypoxemia, hyperglycemia) that accelerate neurotoxic damage and worsen long-term outcome.[12,13] Mediation of secondary insults reduces morbidity and mortality and is discussed in the treatment section.

The **secondary neurotoxic cascade** is a massive release of neurotransmitters, such as glutamate, into the presynaptic space, with activation of *N*-methyl-D-aspartate, α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid, and other receptors.[10] Ionic shifts activate cytoplasmic and nuclear enzymes, induce mitochondrial damage, and lead to cell death and necrosis.[10,14,15] Proinflammatory cytokines and other enzymes are released in an attempt to clean and repair the damage. Secondary injury, however, is indiscriminant and produces extensive neuronal loss. Additionally, many survivable cells undergo apoptosis, or programmed cell death, during secondary injury.[16] Apoptosis has been reported to occur longer than a year after injury.[16,17]

BRAIN EDEMA

Brain edema results from two distinct processes and can be fatal in TBI.[18] Cellular swelling, or **cytotoxic edema**, results from large ionic shifts and the loss of cellular membrane integrity from mitochondrial damage (loss of adenosine triphosphate, ion pump productivity, and increased free radical production). **Extracellular edema** results from direct damage to, or the breakdown of, the blood–brain barrier, ionic shifts, and alteration of water exchange mechanisms (e.g., aquaporins).[19,20] As intracellular and extracellular water content rises, the brain swells and the ICP increases, leading to direct compressive tissue damage, vascular compression-induced ischemia, brain parenchyma herniation, and brain death.

BRAIN HERNIATION

There are four major brain herniation syndromes: uncal transtentorial, central transtentorial, cerebellotonsillar, and upward posterior fossa. The most common is uncal herniation, which occurs when the uncus of the temporal lobe is displaced inferiorly through the medial edge of the tentorium. This is usually caused by an expanding lesion in the temporal lobe or lateral middle fossa. **Uncal transtentorial herniation leads to compression of parasympathetic fibers running with the third cranial (oculomotor) nerve, causing an ipsilateral fixed and dilated pupil due to unopposed sympathetic tone. Further herniation compresses the pyramidal tract, which results in contralateral motor paralysis**. In some cases, the pupillary changes can be contralateral, whereas the motor changes are ipsilateral.

Central transtentorial herniation is less common and occurs with midline lesions, such as lesions of the frontal or occipital lobes, or vertex. **The most prominent symptoms are bilateral pinpoint pupils, bilateral Babinski's signs, and increased muscle tone**. Fixed midpoint pupils follow along with prolonged hyperventilation and decorticate posturing.

Cerebellotonsillar herniation occurs when the cerebellar tonsils herniate through the foramen magnum. **This may lead to pinpoint pupils, flaccid paralysis, and sudden death**. Upward transtentorial herniation results from a posterior fossa lesion and leads to a conjugate downward gaze with absence of vertical eye movements and pinpoint pupils.

THE GLASGOW COMA SCALE

TBI severity is classified using the GCS (Table 257-3). The scale is composed of three components: eye opening (1 to 4 points), verbal response (1 to 5 points), and motor response (1 to 6 points) (Table 257-3). The sum of these components defines the TBI severity classification into **severe** (GCS score of 3 to 8), **moderate** (GCS score of 9 to 13), and **mild** (GCS score of 14 or 15). The motor score independently correlates with outcome, almost as well as the full score.[21]

TABLE 257-3

Glasgow Coma Scale for All Age Groups

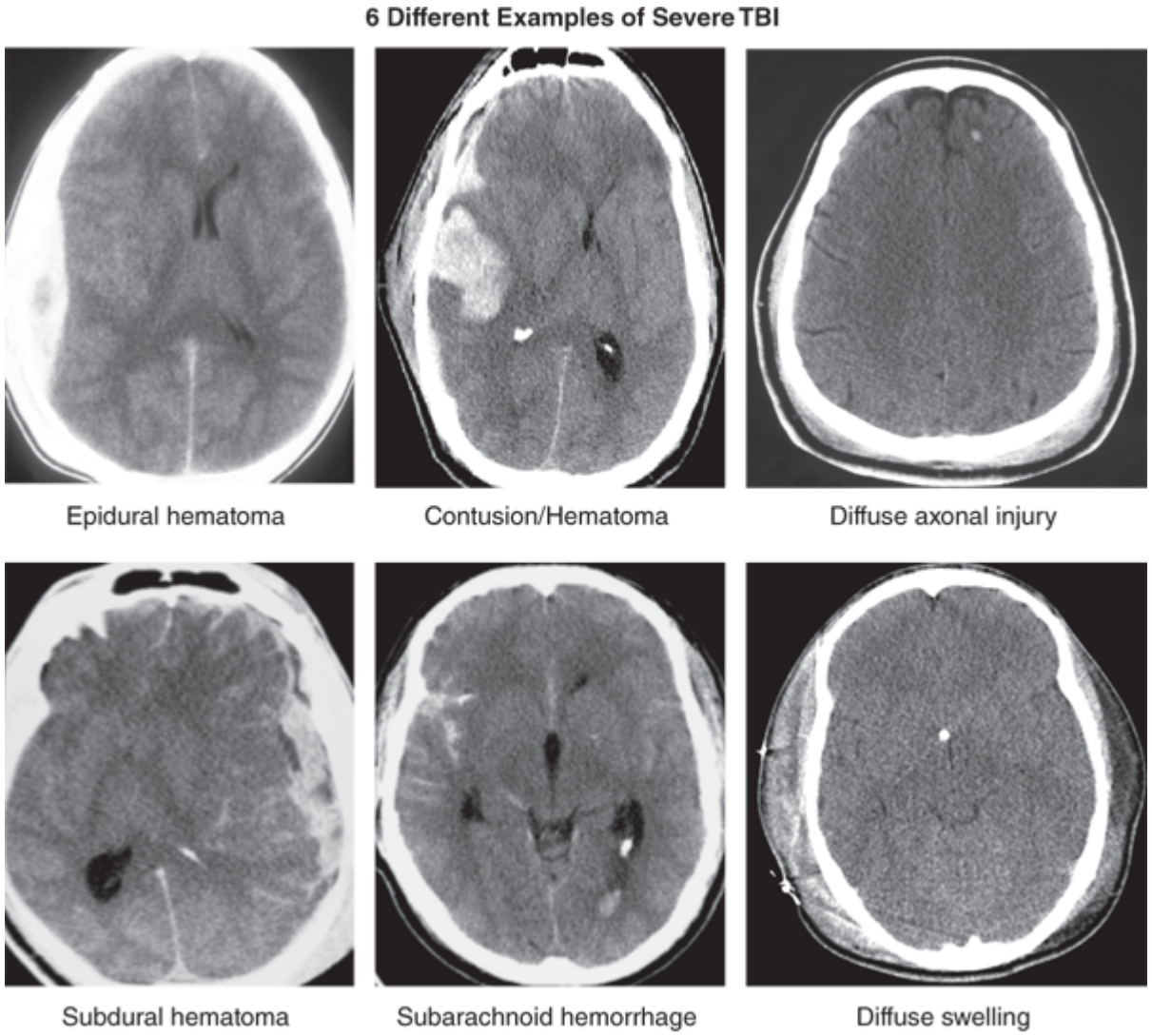
	4 y to Adult	Child < 4 y	Infant
Eye opening			
4	Spontaneous	Spontaneous	Spontaneous
3	To speech	To speech	To speech
2	To pain	To pain	To pain
1	No response	No response	No response
Verbal response			
5	Alert and oriented	Oriented, social, speaks, interacts	Coos, babbles
4	Disoriented conversation	Confused speech, disoriented, consolable, aware	Irritable cry
3	Speaking but nonsensical	Inappropriate words, inconsolable, unaware	Cries to pain
2	Moans or unintelligible sounds	Incomprehensible, agitated, restless, unaware	Moans to pain
1	No response	No response	No response
Motor response			
6	Follows commands	Normal, spontaneous movements	Normal, spontaneous movements

	4 y to Adult	Child < 4 y	Infant
5	Localizes pain	Localizes pain	Withdraws to touch
4	Moves or withdraws to pain	Withdraws to pain	Withdraws to pain
3	Decorticate flexion	Decorticate flexion	Decorticate flexion
2	Decerebrate extension	Decerebrate extension	Decerebrate extension
1	No response	No response	No response
3–15			

Note: In intubated patients, the Glasgow Coma Scale verbal component is scored as a 1, and the total score is marked with a "T" (or tube) denoting intubation (e.g., 8T).

The GCS is an objective measurement of clinical status, correlates with outcome, is a reliable tool for interobserver measurements, and is effective for measuring patient recovery or response to treatment over time. However, the scale has several limitations. It measures behavioral responses, not the underlying pathophysiology. Patients with similar GCS scores may have dramatically different underlying structural injuries and require different clinical interventions (**Figure 257-2**). It is not as useful as a single acute measure of severity as it is as a tool to measure disease progression over time. The GCS may additionally be affected by drugs, alcohol, medications, paralytics, or ocular injuries. Finally, the scale lacks the granularity necessary to assess mTBI.

FIGURE 257-2.
Each of these CT images shows a distinct trauma-induced pathophysiologic abnormality, yet all patients had a Glasgow Coma Scale score of 4. [Image used with permission of Alisa Green, MD, University of California, San Francisco.]



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

CLINICAL FEATURES

The results of history, examination, and diagnostic imaging will allow the distinction into two categories of injury: moderate-severe brain injury and mild brain injury. Treatment and disposition are quite different in the two categories and are detailed below.

HISTORY

Obtain an accurate history from the patient, witnesses, and EMS crews to gain important insight into the mechanism of injury and overall severity of TBI (e.g., height of fall, impact surface condition, damage sustained to vehicle, airbag deployment, seat belt use, history of ejection from the vehicle, or report of fatalities at the scene). Premorbid medical history, medications (especially anticoagulants), drug use, and/or alcohol intoxication are also important in the assessment and treatment of acute TBI. **Initial clinical findings and physical exam as reported by EMS are an essential component of triaging and managing TBI**. The presence of a focal neurologic deficit, seizures, emesis, or depressed level of consciousness increases concern for underlying brain injury.

PHYSICAL EXAMINATION

Follow Advanced Trauma Life Support principles to perform the trauma-focused examination, with simultaneous lifesaving procedures as needed. Protect the cervical spine during evaluation, treatment, and imaging.

Obtain the GCS. Classify the injury as **severe (GCS score of 3 to 8), moderate (GCS score of 9 to 13), or mild (GCS score of 14 or 15)**. **If emergency intubation is necessary, obtain a preintubation GCS and record the patient's best score**.

Determine **pupillary response**.[22] In an unresponsive patient, a single fixed and dilated pupil may indicate an intracranial hematoma with uncal herniation that requires rapid surgical decompression. **Bilateral fixed and dilated pupils** suggest increased ICP with poor brain perfusion, bilateral uncal herniation, drug effect (such as atropine), or severe hypoxia. Bilateral pinpoint pupils suggest either opiate exposure or central pontine lesion.

Altered **motor function** can indicate brain, spinal cord, or peripheral nerve injuries. Assess movement in a coma patient by observing the patient's reaction to noxious stimuli, such as pressure to a nail bed. **Decorticate posturing** (upper extremity flexion and lower extremity extension) indicates severe intracranial injury above the level of the midbrain. **Decerebrate posturing** (arm extension and internal rotation with wrist and finger flexion and internal rotation and extension of the lower extremities) indicates a more caudal injury. For completely unresponsive patients, respiratory pattern and eye movements can provide information regarding brainstem function. Remember, do not assess oculovestibular (cold caloric) and oculocephalic (doll's eyes) responses in a patient under cervical spine precautions.

IMAGING

Individually assess each patient's mechanism of injury, history, comorbidities, and signs and symptoms when determining the need for CT imaging of the head and cervical spine.

Head CT is exquisitely sensitive to the presence of blood and guides ED management. Do not delay head CT, because expanding hemorrhagic lesions need emergency neurosurgical intervention. Therefore, **if the patient is uncooperative or combative, intubation and sedation are often the best options to enable rapid CT imaging**. Other means to control agitated patients with TBI include midazolam (1 to 2 milligrams IV) and propofol (20 milligrams every 10 seconds to desired effect).

Several decision rules have been developed to minimize unnecessary head CT imaging.[23,24,25,26,27] The guidelines strive to identify patients with surgical emergencies. These studies do not specifically address the relationship between minor CT findings (which may place the patient at risk for the development of seizures), the duration of postconcussive symptoms, and progressive changes on CT during the course of a patient's evaluation. Adults with mTBI and a GCS score of 14 or 15 will have an intracranial lesion on CT about 15% of the time, but <1% will require neurosurgical intervention.[28]

The prevalence of **cervical fractures** in comatose TBI patients is approximately 8%, and an estimated 4% of injuries are missed on the initial assessment of the trauma patient.[23] Cervical imaging is a vital component in the care of the brain-injured patient. Perform CT imaging of the cervical spine in patients with altered mental status and who were injured by a mechanism that increases the risk of cervical spine injury. CT is superior to plain radiography in patients with altered mental status and can be performed at the same time as the head CT.

The **NEXUS** and **Canadian Cervical Spine Rules** are discussed in detail in chapter 258, "Spine Trauma."

MRI can detect subtle lesions missed by CT imaging and can better define the extent of contusions. However, MRI may not detect subtle lesions, cannot be performed if the patient is unstable, and is not always available.

DECISION RULES FOR HEAD CT IMAGING IN ADULTS

Decision rules can guide clinical practice, but each patient must be assessed individually, and none of the rules described below address short- or long-term nonoperative sequelae of TBI. See chapter 110, "Pediatric Trauma," for a discussion of the role of head CT imaging in children with minor head injury.

The two most commonly used evidence-based clinical decision rules for head CT in adults are the **New Orleans Criteria**[29] and the **Canadian CT Head Rule**.[23] Both rules have been validated and are 100% sensitive in detecting patients who will need neurosurgical intervention, but they have

limited specificity (5% versus 37%, respectively). The Canadian CT Head Rule is less sensitive (83%) if intracranial lesion is the defined end point. *A negative feature of these two decision rules is that loss of consciousness or amnesia is required as the entry point*. **Most minor brain injury events do not result in loss of consciousness, and loss of consciousness is not the best predictor of intracranial pathology (Table 257-4). Do not apply these rules to patients taking anticoagulants or antiplatelet agents, or to children, because these variables were not included in the validation studies**.

TABLE 257-4

New Orleans Criteria and Canadian CT Head Rule Clinical Decision Rules

New Orleans Criteria—GCS 15*	**Canadian CT Head Rule—GCS 13–15***
Headache	GCS <15 at 2 h
Vomiting	Suspected open or depressed skull fracture
Age >60 y	Age ≥65 y
Intoxication	More than one episode of vomiting
Persistent antegrade amnesia	Retrograde amnesia >30 min
Evidence of trauma above the clavicles	Dangerous mechanism (fall >3 ft or struck as pedestrian)
Seizure	Any sign of basal skull fracture
Identification of patients who have an intracranial lesion on CT	
100% sensitive, 5% specific	83% sensitive, 38% specific
Identification of patients who will need neurosurgical intervention	
100% sensitive, 5% specific	100% sensitive, 37% specific

Abbreviation: GCS = Glasgow Coma Scale.

*Presence of any one finding indicates need for CT scan.

The *National Institute for Clinical Excellence* and the *Neurotraumatology Committee of the World Federation of Neurosurgical Societies* have evaluated clinical signs and symptoms associated with TBI in adults and adolescents and adults, respectively.[27,30] The resultant decision rules for head CT have been applied to large data sets and shown to be relatively sensitive (National Institute for Clinical Excellence: 94% for neurosurgical lesions, 82% for intracranial lesions; Neurotraumatology Committee: 100% for neurosurgical lesions and intracranial injuries). One study evaluated 1101 patients with mTBI who had GCS scores of 14 or 15; approximately 2% of these patients without loss of consciousness had intracranial lesions and 0.6% required surgery (rates similar to patients with loss of consciousness).

One of the most important findings from these studies is the relative significance of certain elements of the history and physical examination. For example, nausea and vomiting after concussion has an odds ratio comparable to that of loss of consciousness for a positive CT finding (**Table 257-5**). Importantly, the predictive value of individual clinical signs and symptoms differs between adults and children (see chapter 138, Head Injury in Infants and Children).

TABLE 257-5

Odds Ratio (OR) for Head CT and Clinical Features

	Smits et al[30]	Ibanez et al[26]	Fabbri et al[27,70]
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Glasgow Coma Scale score of 14	2 (1–3)	7 (4–14)	19 (14–26)
Neurologic deficits	2 (1–3)	7 (2–25)	19 (13–28)
Signs of basilar skull fracture	14 (8–22)	11 (6–23)	10 (6–16)
Loss of consciousness	2 (1–3)	7 (4–11)	2 (2–3)
Posttraumatic amnesia	1.7 (1–2)	3 (2–5)	8 (6–12)
Headache	1.4 (1–2)	3 (2–6)*	—
Vomiting	3 (2–4)	4 (2–7)	5 (3–8)
Posttraumatic seizure	3 (1–10)	2 (0.25–17)	3 (2–5)
Intoxication	1 (0.6–2)	1 (0.3–3)	—
Antithrombotics	2 (1–4)	4 (3–7)	8 (3–9)
Age >65 y	—	2 (1–3)	2 (1–3)
Dangerous mechanism of injury	2 (1–4)	—	3 (2–4)

Abbreviation: CI = confidence interval.

*For severe headache.

A summary of the American College of Emergency Physicians recommendations[28] is given in **Table 257-6**. A combination of rules helps identify patients *at risk* and determine the possible need for a head CT (**Tables 257-4, 257-5 and 257-6**).

TABLE 257-6

CT Scanning for Adults with Brain Injury (American College of Emergency Physicians Guidelines)

Adults with a Glasgow Coma Scale score of < 15 at the time of evaluation should undergo CT imaging
Mild traumatic brain injury with or without loss of consciousness: if one or more of the following is present:
Glasgow Coma Scale score <15
Focal neurologic findings
Vomiting more than two times
Moderate to severe headache
Age >65 y
Physical signs of basilar skull fracture
Coagulopathy
Dangerous mechanism of injury (e.g., fall >4 ft)
Mild traumatic brain injury with loss of consciousness or amnesia: if one or more of the following is present:
Drug or alcohol intoxication
Physical evidence above the clavicles
Persistent amnesia
Posttraumatic seizures

TREATMENT

PREHOSPITAL CARE

Early appropriate management can have a profound impact on the patient's final outcome. For patients with moderate to severe head injury, provide stabilization and rapid transport to a facility with experience in the management of brain injury. The most important prehospital interventions are airway and blood pressure management. If the patient needs prehospital intubation, avoid hyperventilation (which causes cerebral vasoconstriction and can negatively affect outcome), and use **capnometry** to keep PCO_2 at 35 to 45 mm Hg. Treat hypotension aggressively. If transport times are short, do not give mannitol or hypertonic saline for elevated ICP. Guidelines for prehospital care are available at http://www.braintrauma.org.

ED TREATMENT

Principles for ED care of moderate/severe brain injury are provided at http://www.braintrauma.org and are discussed in the following section. The primary goals of treatment are to maintain cerebral perfusion and oxygenation by optimizing intravascular volume and ventilation; prevent secondary injury by correcting hypoxia, hypercapnia, hyperglycemia, hyperthermia, anemia, or hypoperfusion; recognize and treat elevated ICP; arrange for neurosurgical intervention to evacuate intracranial mass lesions; and treat other life-threatening injuries.

Systolic blood pressure of <90 mm Hg and hypoxemia (PaO_2 <60) are associated with a 150% increase risk in mortality.[31]

Observe for the **signs/symptoms of elevated ICP**: change in mental status, pupillary irregularities, focal neurologic deficits, decerebrate or decorticate posturing, or CT pathology. Some CT signs of intracranial hypertension are attenuation of the visibility of sulci and gyri, because the brain is compressed against the skull; compressed lateral ventricles; and poor grey/white matter distinction. Papilledema may not be evident if pressure rises rapidly. **Sedation and analgesia** may decrease baseline ICP and prevent transient rises in ICP from agitation, coughing, or gagging from the endotracheal tube. Prevent and control **seizure activity**.

Treat hypotension, hypoxemia, hypercarbia, and hyperglycemia. A single occurrence of hypotension and hypoxia after brain injury is associated with a 150% increase in mortality.[22] TBI is progressive, so appropriate early management will have a greater impact on outcome than treatments initiated after neuronal cell death and the development of secondary injury, such as cerebral edema. Jointly develop and apply goal-directed protocols with emergency medicine, trauma, neurosurgery, and intensive care teams. An example of early goal-directed therapy is provided in Table 257-7.

TABLE 257-7

Checklist for ED Treatment of Brain Injury

	Treatment	Comments
Cervical spine	Spinal precautions	
Airway	Maintain airway, intubate for GCS <8 or as needed	
Oxygenation and ventilation	Oxygen saturation >90; PCO_2 35–5	No prophylactic hyperventilation
BP	Systolic BP >90 mm Hg, MAP 80 mm Hg; give NS, blood products, or transfuse as needed	No permissive hypotension; pressors may be required if fluids not sufficient
Exam and GCS	GCS before paralytics if possible; treat life-threatening injuries and active bleeding	Serial GCS is helpful in identifying change; keep goal of "brain resuscitation" as top priority
Stat head CT and cervical spine CT	Identify mass lesions and signs of increased ICP	Protect cervical spine until cleared
Repeat exam	Check GCS for changes and for signs of impending herniation/deterioration	Change of more than 2 points should prompt further workup
Check glucose	Treat hypoglycemia *and* hyperglycemia	Hyperglycemia is bad for the brain
Control temperature	Maintain between 36°F and 38.3°F	Aggressive cooling: Tylenol, cooling blanket, etc.
Seizure prophylaxis	Give antiepileptic drug if GCS ≤10, acute seizure with injury, or abnormal head CT scan	Phenytoin (Dilantin)/fosphenytoin/levetiracetam

	Treatment	Comments
Identify and treat elevated ICP, herniation	Keep head of the bed at 30 degrees; ensure good BP, ventilation, and temperature control; give mannitol 1 gram/kg IV bolus; urgent NS consult	Consider adding hypertonic saline (3% NaCl 250 mL/30 min) for refractory elevations in ICP; monitor BP and electrolytes
Neurosurgery referral/transfer for advanced care	ICP monitoring, ventriculostomy for ICP management, aggressive tiered approach to management, emergency surgery	ICP monitoring and CSF diversion in GCS ≤8

Abbreviations: BP = blood pressure; CSF = cerebrospinal fluid; GCS = Glasgow Coma Scale; ICP = intracranial pressure; MAP = mean arterial pressure; NS = normal saline.

Airway and Breathing

Treat any condition that compromises ventilation (e.g., altered mental status, facial/neck trauma, pneumothorax). **Patients with severe injury (GCS score of ≤8) require intubation**. Use short-acting induction agents that have limited effect on blood pressure or ICP (**Table 257-8**). Avoid nasotracheal intubation if facial trauma or basilar skull fracture is evident or suspected. Monitor blood pressure throughout the procedure. Preinduction agents such as low-dose succinylcholine, vecuronium, pancuronium, and lidocaine do not improve outcome, but can be used as adjuncts if they do not delay airway control.[32] Maintain in-line cervical spine stabilization during intubation.

TABLE 257-8

Intubation Agents in Brain Injury

Agent	Comments
Induction agent Etomidate, 0.3 milligram/kg IV Propofol 1–3 milligrams/kg IV	May be neuroprotective; may lower intracranial pressure; adrenal suppression unlikely with single use Rapid onset and offset; antiseizure properties; can cause hypotension if inadequate fluid resuscitation
Paralytics Succinylcholine 1–1.5 milligrams/kg IV Rocuronium, 0.6–1.0 milligram/kg IV	Short acting; avoid in burns, extensive muscle injury, etc. Short acting, safe in hyperkalemia

Maintain oxygenation and use capnometry to control PCO_2 and avoid hyperventilation. Prolonged (>6 hours) hypocapnia causes cerebral vasoconstriction and worsens cerebral ischemia. **Keep oxygen saturation >90, PaO_2 >60, and PCO_2 at 35 to 45**.

Circulation

Traumatic hypotension leads to ischemia within low flow regions of the injured brain. Ischemia amplifies the neurotoxic cascade and increases cerebral edema. Provide aggressive fluid resuscitation to prevent hypotension and secondary brain injury. **Maintain systolic blood pressure at >90 mm Hg and MAP >80 mm Hg**. A blood pressure within "normal" range may be inadequate to maintain adequate flow and CPP if ICP is increased. **Permissive hypotension worsens outcome in patients with brain injury**.

Isolated head injury rarely produces hypotension, except as a preterminal event. Hypovolemic shock may be seen with polytrauma, massive blood loss from scalp lacerations, or in small children from subgaleal hematoma. If fluid and blood resuscitation is not effective, use vasopressors to preserve cerebral perfusion.

Pain and increased ICP can cause hypertension. Treat pain, and assess for impending herniation (*Cushing reflex*). For management, see discussion within this chapter under "Increased Intracranial Pressure Management" section.

Patient

Positioning Raising the head of the bed may improve cerebral blood flow by lowering ICP. However, the interaction between ICP, MAP, and tissue oxygenation is complex and highly variable. Response to position change depends on many factors such as degree of intact autoregulation, brain compliance, and individual patient variability. There is still uncertainty as to whether this procedure is beneficial, but in the setting of suspected elevated ICP, it is currently recommended as a simple maneuver to improve cerebral blood flow. One must ensure that the patient's blood pressure is maintained above the minimum recommended level (MAP 80 mm Hg), because elevation of 30 degrees can drop the mean pressure within the brain by up to 10 to 15 mm Hg and improve CPP (remember CPP = MAP – ICP, so lowering the ICP improves CPP, but lowering MAP in the setting of hypotension could be counterproductive and lower CPP). Elevating the head of the bed to 30 degrees can be safely accomplished even when the spine has not been cleared, as long as neck movement is secured.[33]

Glucose Control

Hyperglycemia in the setting of neurologic injury (both stroke and TBI) is associated with worse outcome. Tight hyperglycemic control is recommended in patients with moderate to severe TBI. Insulin drips may be required to achieve adequate control (glucose 100 to 180 milligrams/dL or 5.55 to 9.99 mmol/L).

Temperature Control

Elevated temperature is associated with an increased metabolic demand and excessive glutamate release. Elevated temperature elevates ICP and worsens outcome in many neurologic critical care conditions including TBI. Treat fever with the goal of normothermia. The evidence for hypothermia in TBI is not sufficient to recommend its use.

Seizure Treatment and Prophylaxis

Seizures after head injury can change the neurologic examination, alter oxygen delivery and cerebral blood flow, and increase ICP. Prolonged seizures can worsen secondary injury. Treat acute seizures with IV lorazepam, and if seizures continue, treat as for status epilepticus. Give prophylactic phenytoin/phospheny-toin if the GCS is ≤10, if the patient has an abnormal head CT scan, or if the patient has had an acute seizure after the injury. The dose is 18 milligrams/kg IV at 25 milligrams/minute. Prophylactic anticonvulsants reduce the occurrence of posttraumatic seizures within the first week. Phenytoin/phosphenytoin is the agent most studied. Levetiracetam can be used, but there are less data supporting its use. Steroids have no role.

Develop a team approach to ICP management between emergency medicine, neurosurgery, intensive care unit, and trauma teams.

Use patient history and physical examination to identify signs and symptoms of impending herniation. Indicators of rising ICP include severe headache, visual changes, numbness, focal weakness, nausea, vomiting, seizure, change in mental status, lethargy, hypertension, coma, bradycardia, and agonal respirations. Signs of impending transtentorial herniation include unilateral or bilateral pupillary dilation, hemiparesis, motor posturing, and/or progressive neurologic deterioration.

Measure neurologic deterioration by comparing sequential GCS scores. In a patient with a rapidly deteriorating GCS, if time permits, obtain a repeat head CT to identify an expanding intracranial hematoma.

Mannitol and/or hypertonic saline can lower ICP. Mannitol is an osmotic agent that can reduce ICP and improve cerebral blood flow, CPP, and brain metabolism. Mannitol is also a free radical scavenger. It generally has an effect within 30 minutes. Mannitol expands plasma volume and can improve oxygen-carrying capacity. Administer mannitol by repetitive bolus (0.25 to 1 gram/kg), and not by constant infusion. Because no dose-dependent effect is seen with mannitol, some clinicians advocate beginning at the lower range of the suggested dose. Mannitol results in a net intravascular volume loss because of its diuretic effect. Monitor the patient's input and output. Osmotic diuresis is *relatively contraindicated* in hemorrhage and hypotension. However, in the setting of acute herniation, mannitol has been demonstrated to effectively reduce life-threatening elevations of ICP.

Hypertonic saline may be used as an alternative to mannitol in the patient who is not adequately fluid resuscitated or hypotensive. The Brain Trauma Foundation indicates that at this time, data support the primary use of mannitol for the acute treatment of ICP. Most EDs have 3% NaCL available; the dose for adults is 250 mL over 30 minutes. Intensive care units may stock 23.4% sodium chloride solution; the dose for adults is 30 mL over 30 minutes. Monitor serum osmolality and serum sodium.

Mannitol and hypertonic saline may be given serially and in conjunction with one another.

ADVANCED TREATMENT OF BRAIN INJURY

Advanced treatment of brain injury requires invasive and close monitoring (**Table 257-9**).

TABLE 257-9

Goal-Directed Therapy of Brain Injury

Goal-Directed Therapy—Suggested Targets		
Pulse oximetry ≥90%	CPP ≥60 mm Hg	Physiologic sodium 135–140 mEq/L
SBP ≥90 mm Hg	ICP <20 mm Hg	INR ≤1.4
MAP ≥80 mm Hg	$PbtO_2$ ≥15 mm Hg	Platelets ≥75×10^3/μL
$PaCO_2$ 35–45 mm Hg	pH 7.35–7.45	Hemoglobin ≥8 grams/dL
Temperature 36.0–38.3°C	Glucose 80–180 milligrams/dL	

Abbreviations: CPP = cerebral perfusion pressure; ICP = intracranial pressure; MAP = mean arterial pressure; $PbtO_2$ = brain tissue oxygen tension monitoring; SBP = systolic blood pressure.

Cerebral Perfusion Pressure Management

If the GCS is ≤8, arrange for placement of an intracranial bolt or extraventricular drain with monitoring capabilities as soon as possible to monitor ICP and to direct treatment. Maintain CPP at 55 to 60 mm Hg to adequately perfuse brain tissue.[8] Increasing CPP >70 mm Hg may result in injury to other organs (e.g., acute respiratory distress syndrome from lung tissue trauma).

Consider ICP monitoring for patients with a normal admission brain CT scan if two or more of the following criteria are met: age over 40 years, unilateral or bilateral motor posturing, and systolic blood pressure <90 mm Hg. In addition, provide ICP monitoring in patients undergoing emergency surgery (e.g., orthopedic repair). Management of CPP is essential intraoperatively, where the patient with elevated ICP may experience large shifts in central volume status due to surgical blood loss.

Increased Intracranial Pressure Management

An ICP of >20 mm Hg increases morbidity and mortality. Early consultation with neurosurgery for direct ICP monitoring, cerebrospinal fluid diversion, or surgical intervention is highly recommended in moderate and severe TBI. In certain circumstances, an ICP monitor will be placed in the ED by neurosurgery to help guide medical management of ICP, as well as for direct cerebrospinal fluid diversion to lower ICP.

SPECIFIC HEAD INJURIES

SCALP LACERATIONS

Scalp lacerations can lead to massive blood loss, so control bleeding as rapidly as possible. If direct pressure is not effective, locally infiltrate lidocaine with epinephrine and clamp or ligate bleeding vessels. Before closure, carefully examine wounds to identify foreign bodies, underlying fractures, and galeal lacerations. Large galeal disruptions should be repaired. For discussion of repair of scalp lacerations, see chapter 42, "Face and Scalp Lacerations."

SKULL FRACTURES

Patients who have or are suspected of having a skull fracture require a head CT scan (see Table 257-4). Skull fractures are usually categorized by location (basilar versus skull convexity), pattern (linear, depressed, or comminuted), and whether they are open or closed (**Figures 257-3 and 257-4**). A linear skull fracture with an overlying laceration is an open fracture. Explore wounds gently to avoid driving bone fragments into the brain.

FIGURE 257-3.
Linear fracture seen on CT. *Arrow* indicates skull fracture; *asterisks* indicate normal cranial suture lines. [Image used with permission of Joseph Piatt, Jr., MD, Division of Neurosurgery, A. I. duPont Hospital for Children, Wilmington, Delaware; Departments of Neurological Surgery and Pediatrics, Thomas Jefferson University, Philadelphia, Pennsylvania.]

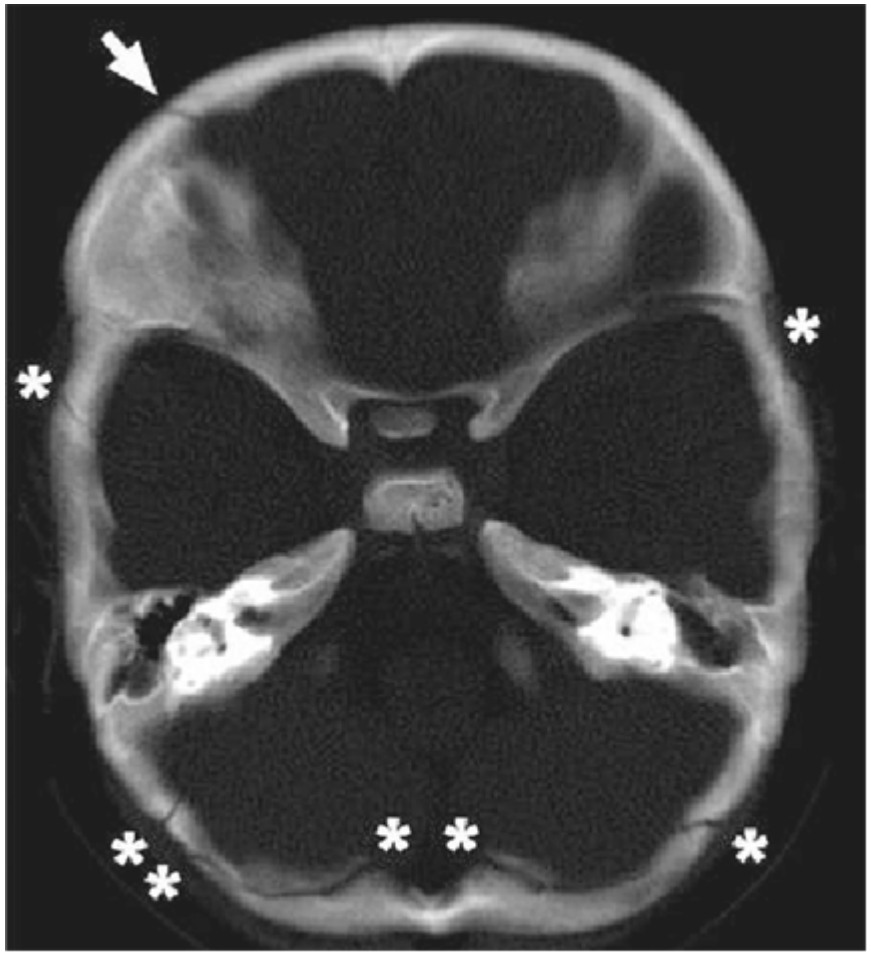
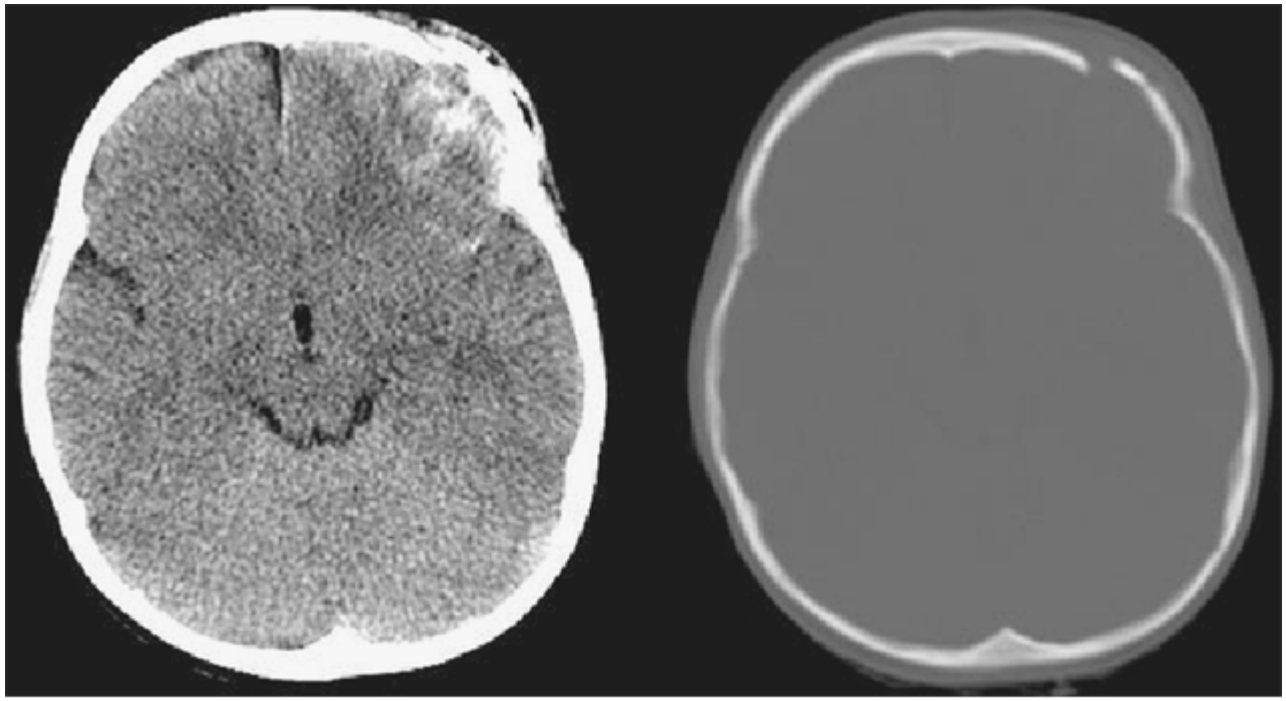


FIGURE 257-4.

Open skull fracture with underlying cerebral contusion. This injury was sustained from a fall of two stories. [Image used with permission of Joseph Piatt, Jr., MD, Division of Neurosurgery, A. I. duPont Hospital for Children, Wilmington, Delaware; Departments of Neurological Surgery and Pediatrics, Thomas Jefferson University, Philadelphia, Pennsylvania.]



Fractures that cross the middle meningeal artery, a major venous sinus, or linear occipital fractures have high intracerebral complication rates. Patients with skull fractures that are open or depressed, involve a sinus, or are associated with pneumocephalus should be given antibiotics (vancomycin, 1 gram IV, and ceftriaxone, 2 grams IV). A skull fracture that is depressed by more than the thickness of the skull usually requires operative repair.

BASILAR SKULL FRACTURE AND CEREBROSPINAL FLUID LEAKS

The presence of a basilar skull fracture is a significant risk factor for intracranial injury. The most common basilar skull fracture involves the petrous portion of the temporal bone, the external auditory canal, and the tympanic membrane. It is associated with dural tearing, which often leads to otorrhea or rhinorrhea. Basilar skull fractures may occur anywhere along the skull base, from the cribriform plate through the occipital condyles. Do not place a nasogastric tube through the nares if cribriform plate fracture is suspected; this can lead to direct intracranial injury. **Signs and symptoms associated with basilar skull fractures include cerebrospinal fluid leak, mastoid ecchymosis (Battle sign), periorbital ecchymoses (raccoon eyes), hemotympanum, vertigo, decreased hearing or deafness, and seventh nerve palsy**. Periorbital and mastoid ecchymoses develop gradually over hours after an injury and are often absent in the ED. Cerebrospinal fluid leaks (otorrhea or rhinorrhea) are

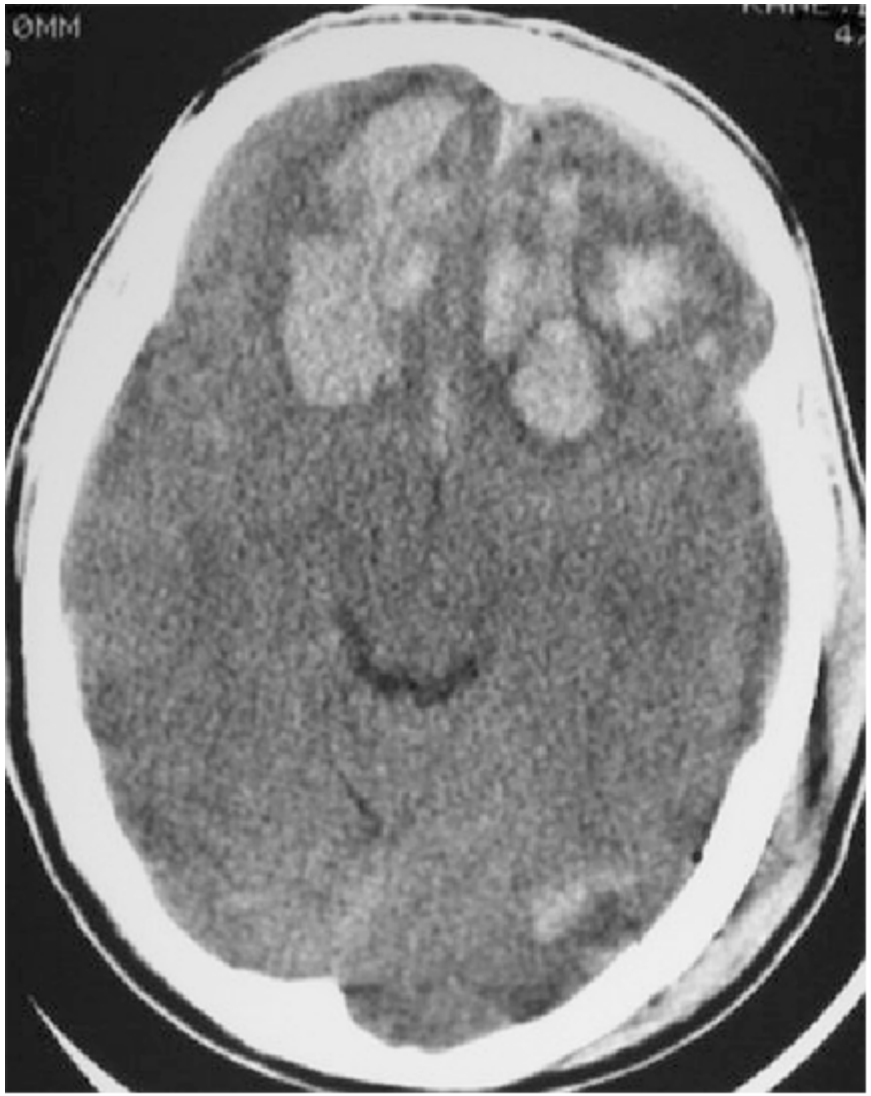
difficult to diagnose; however, the patient often complains of discharge of clear fluid from the nose or ears. Fluid may be collected and sent for analysis (identification of β transferrin). The β2 transferrin isoform of transferrin is found only in cerebrospinal fluid, and not in blood, mucus, or tears.

Patients with acute cerebrospinal fluid leaks are at risk for meningitis. Antibiotic prophylaxis is often recommended to reduce the incidence of infection.[30] Administration of antibiotics should be done in consultation with the neurosurgeon who will be following the patient. If prophylactic antibiotics are instituted, the drugs selected should have broad coverage with good penetration into the meninges, such as ceftriaxone, 2 grams IV, and vancomycin, 1 gram IV. The head of the patient's bed should be elevated to 30 degrees. A lumbar drain is often placed by the neurosurgical team. Cerebrospinal fluid leaks may require repair by a neurosurgeon or otolaryngologist.

CEREBRAL CONTUSION AND INTRACEREBRAL HEMORRHAGE

Contusions most commonly occur in the subfrontal cortex, in the frontal and temporal lobes, and, occasionally, in the occipital lobes (**Figure 257-5**). They are often associated with subarachnoid hemorrhage. Contusions may occur at the site of the blunt trauma or on the opposite side of the brain, known as a *contrecoup* injury.

FIGURE 257-5.
CT scan demonstrating delayed intraparenchymal hemorrhages from a traumatic contusion. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]



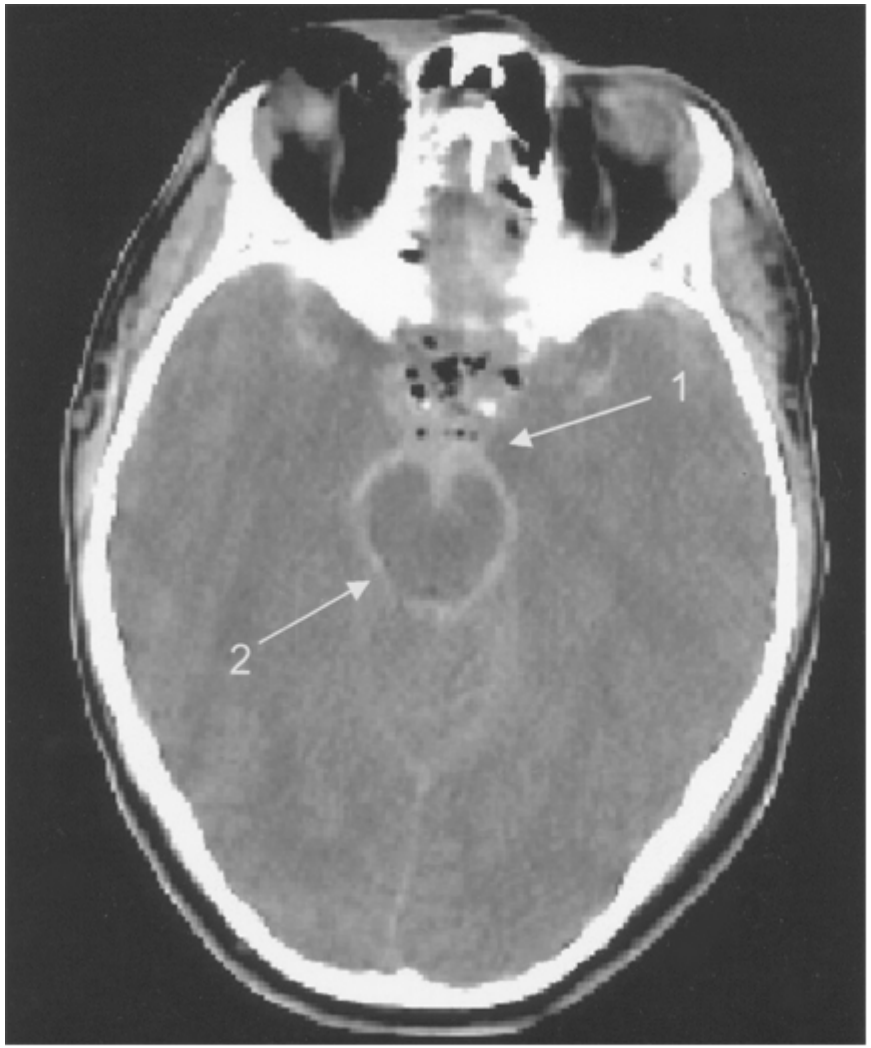
Intracerebral hemorrhage can occur days after significant blunt trauma, often at the site of resolving contusions. This complication is more common in patients with coagulopathy. CT scan findings immediately after injury may be normal. Obtain serial CTs if any change in mental status occurs in a patient with coagulopathy until the clot is stable.

SUBARACHNOID HEMORRHAGE

Traumatic subarachnoid hemorrhage results from the disruption of the parenchyma and subarachnoid vessels and presents with blood in the cerebrospinal fluid (**Figure 257-6**). Patients with isolated traumatic subarachnoid hemorrhage may present with headache, photophobia, and meningeal signs. **Traumatic subarachnoid hemorrhage is the most common CT abnormality in patients with moderate to severe TBI**. Patients with early development of traumatic subarachnoid hemorrhage have a threefold higher mortality risk than those without traumatic subarachnoid hemorrhage (42% versus 14%, respectively).[34] Some traumatic subarachnoid hemorrhages can be missed on early CT scans. Generally, CT scans performed 6 to 8 hours after injury are sensitive for detecting traumatic subarachnoid hemorrhage.

FIGURE 257-6.

CT scan demonstrating subarachnoid hemorrhage. *Arrow 1* indicates prepontine cisternal blood, and *arrow 2* identifies blood in the ambient cistern. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]

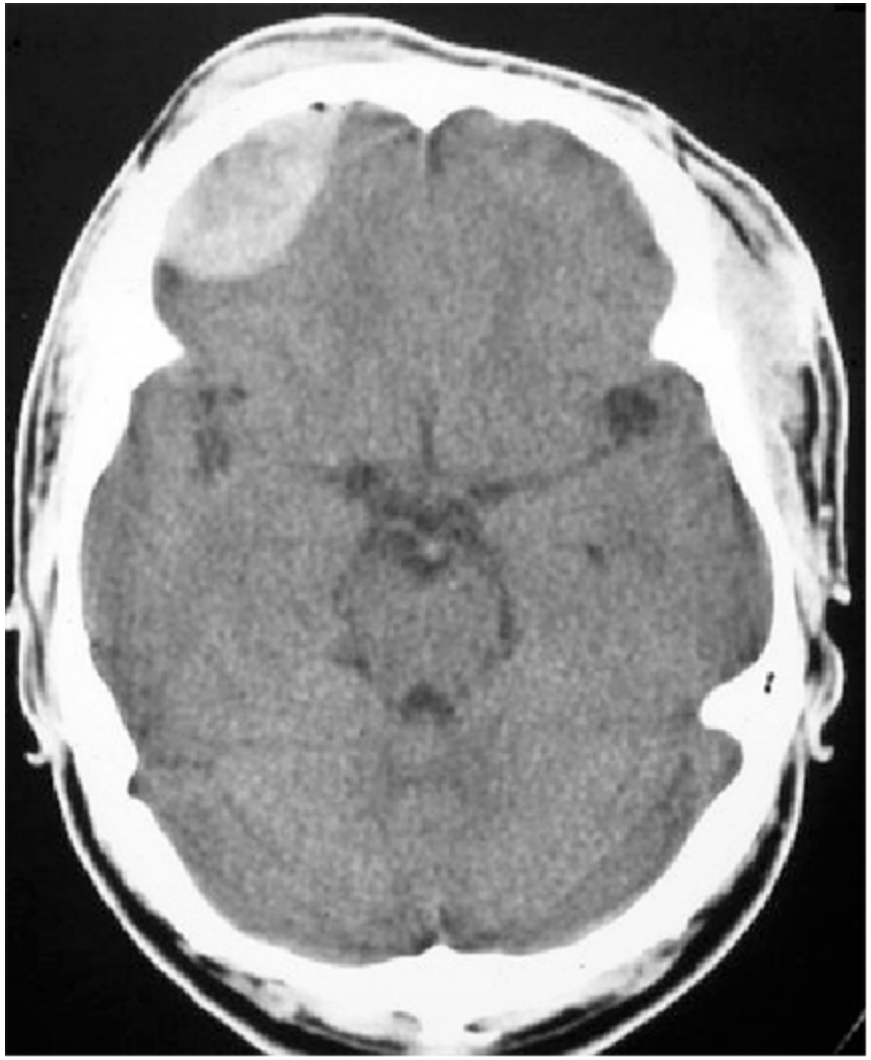


EPIDURAL HEMATOMA

An epidural hematoma results when blood collects in the potential space between the skull and the dura mater (**Figure 257-7**). The anatomic relationships of the branches of the middle meningeal artery and the sequelae of fracture and laceration of the artery are shown in Figure 257-2.

FIGURE 257-7.

Epidural hematoma. Note the convex shape and focal location. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]



Blunt trauma to the temporal or temporoparietal area with an associated skull fracture and middle meningeal arterial disruption is the primary mechanism of injury. Occasionally, trauma to the parieto-occipital region or the posterior fossa causes tears of the venous sinuses with epidural hematomas.

The classic history of an epidural hematoma involves a significant blunt head trauma with loss of consciousness or altered sensorium, followed by a lucid period and subsequent rapid neurologic demise. This clinical presentation occurs in a minority of cases. Traumatic blows to the thin temporal bone over the lateral aspect of the head carry the highest risk (e.g., baseball or pool stick injury). The diagnosis of an epidural hematoma is based on CT scan and physical examination findings. The CT appearance of an epidural hematoma is a biconvex (football-shaped) mass, typically found in the temporal region.

The high-pressure arterial bleeding of an epidural hematoma can lead to herniation within hours after an injury. Early recognition and evacuation reduces morbidity and mortality. Underlying injury of the brain parenchyma is often absent; full recovery may be expected if the hematoma is evacuated prior to herniation or the development of neurologic deficits.

SUBDURAL HEMATOMA

Subdural hematoma is caused by sudden acceleration-deceleration of brain parenchyma with subsequent tearing of the bridging dural veins. This results in hematoma formation between the dura mater and the arachnoid (**Figures 257-8 and 257-9**). Subdural hematoma tends to collect more slowly than epidural hematoma because of its venous origin. However, subdural hematoma is often associated with concurrent brain injury and underlying parenchymal damage. **Brains with extensive atrophy, such as in the elderly or in chronic alcoholics, are more susceptible to the development of acute subdural hematoma**. Even seemingly benign falls from standing position can result in subdural bleeding in the elderly. Children <2 years old are also at increased risk of subdural hematoma.

FIGURE 257-8.
Small subdural hematoma in the right frontotemporal region in an adult. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]

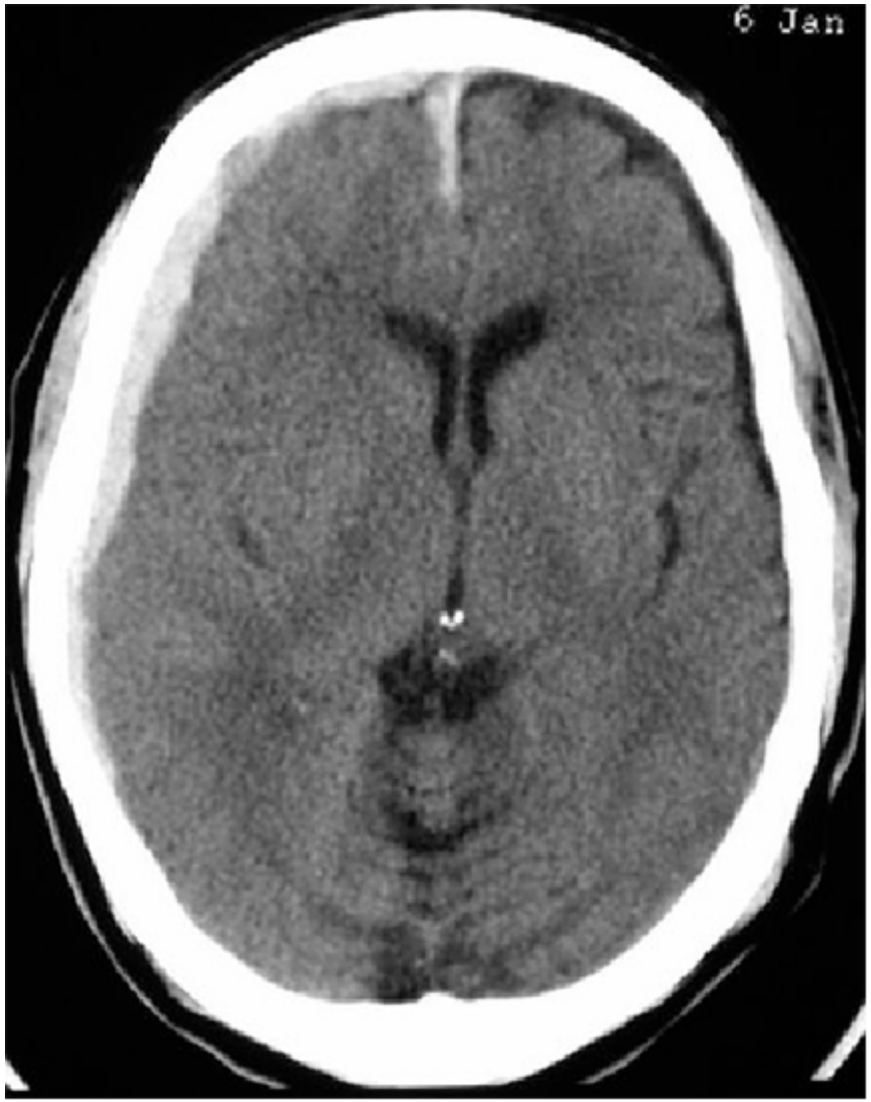
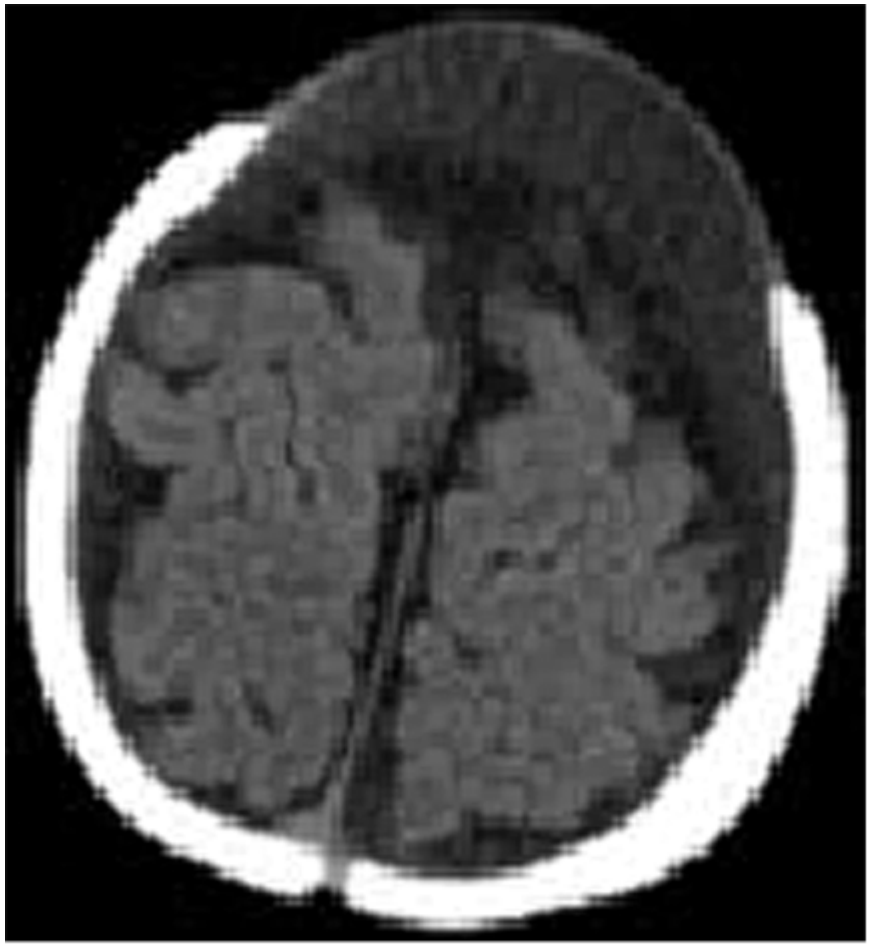
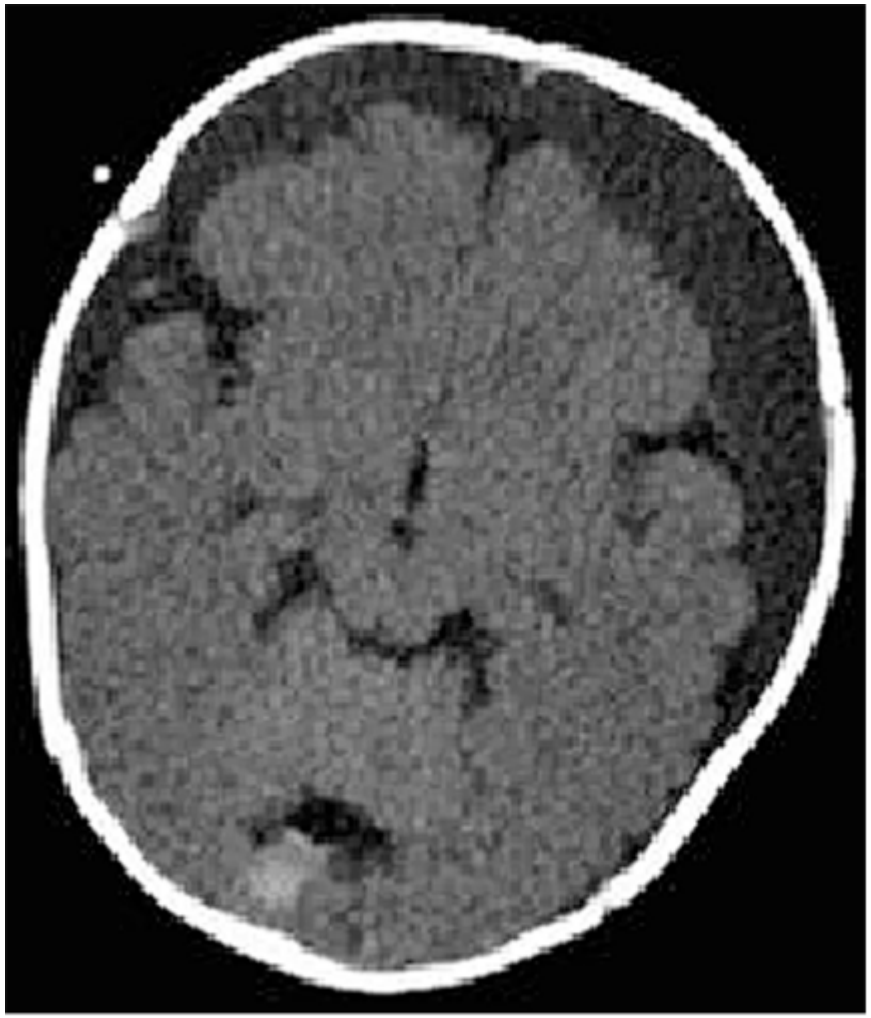


FIGURE 257-9.

A. Bifrontal chronic subdural hematoma extending through the anterior fontanelle in a 1-month-old child. B. Second image in the same child showing bifrontal chronic subdural hematoma, as well as small, acute intraparenchymal hemorrhage in the posterior fossa.



A



B

Traditionally, subdural hematomas have been classified as acute, subacute, or chronic depending on the length of time from onset and occurrence of active hemorrhage. Acute symptoms usually develop within 14 days of the injury. After 2 weeks, the term *chronic subdural hematoma* is used. There is no specific clinical syndrome associated with a subdural hematoma. Acute cases usually present immediately after severe trauma, and often the patient is unconscious. In the elderly or in alcoholics, chronic subdural hematomas may result in vague complaints or mental status changes. Often, there is no recall of injury. On CT scan, acute subdural hematomas are hyperdense (white), crescent-shaped lesions that cross suture lines. Subacute subdural hematomas are isodense and are more difficult to identify. CT scanning with IV contrast or MRI

can assist in identifying a subacute subdural hematoma. A chronic subdural hematoma appears hypodense (dark) because the iron in the blood has been metabolized.

The definitive treatment depends on the type, size, effect on underlying brain parenchyma, and the associated brain injury. Mortality and the need for surgical repair are greater for acute and subacute subdural hematomas. Chronic subdural hematomas can sometimes be managed without surgery depending on the severity of the symptoms. **Table 257-10** compares intracranial injuries.

TABLE 257-10

Comparison of Intracranial Injuries

	Type of Patient	Anatomic Location	CT Findings	Common Cause	Classic Symptoms
Epidural	Young, rare in the elderly and those age <2 y	Potential space between skull and dura mater	Biconvex, football-shaped hematoma	Skull fracture with tear of the middle meningeal artery	Immediate LOC with a "lucid" period prior to deterioration (only occurs in about 20%)
Subdural	More risk in the elderly and alcoholic patients	Space between dura mater and arachnoid	Crescent- or sickle-shaped hematoma	Acceleration-deceleration with tearing of the bridging veins	Acute: rapid LOC, lucid period possible Chronic: altered mental state and behavior with gradual decrease in consciousness
Subarachnoid	Any age group after blunt trauma	Subarachnoid	Blood in the basilar cisterns and hemispheric sulci and fissures	Acceleration-deceleration with tearing of the subarachnoid vessels	Mild, moderate, or severe traumatic brain injury with meningeal signs and symptoms
Contusion/intracerebral hematoma	Any age group after blunt trauma	Usually anterior temporal or posterior frontal lobe	May be normal initially with delayed bleed	Severe or penetrating trauma; shaken baby syndrome	Symptoms range from normal to LOC

Abbreviation: LOC = loss of consciousness.

DIFFUSE AXONAL INJURY

Diffuse axonal injury is the disruption of axonal fibers in the white matter and brainstem. Shearing forces on the neurons generated by sudden deceleration cause diffuse axonal injury. The condition is seen after blunt trauma, such as from a motor vehicle crash. In infants, shaken baby syndrome is a well-described cause.[35]

In severe diffuse axonal injury, edema can develop rapidly. The underlying injury can result in devastating and often irreversible neurologic deficits. A CT scan of a patient with diffuse axonal injury may appear normal, but classic CT findings include punctuate hemorrhagic injury along the grey-white junction of the cerebral cortex and within the deep structures of the brain (**Figures 257-10 and 257-11**). Treatment options are very limited, but an attempt should be made to prevent secondary damage by reducing cerebral edema and limiting pathologic increases in ICP.

FIGURE 257-10.
Diffuse axonal injury with intraventricular blood. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]

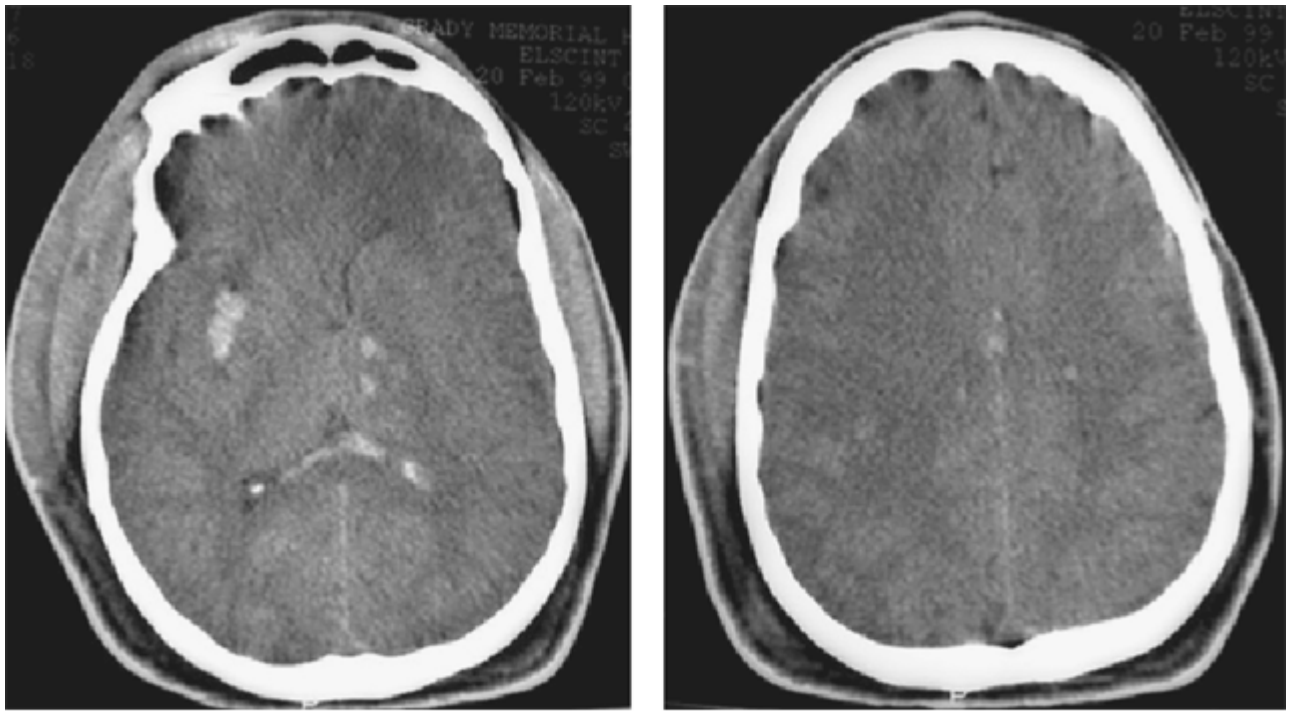
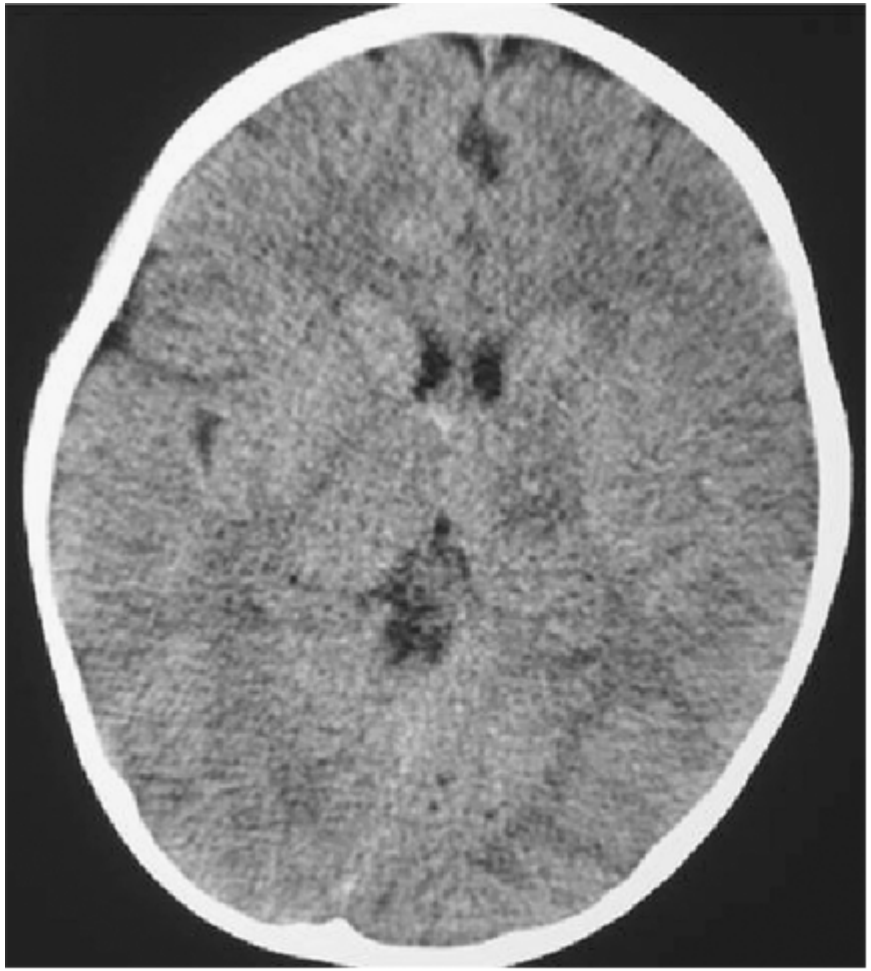


FIGURE 257-11.

Diffuse axonal injury with loss of the grey matter–white matter interface. [Image used with permission of Daniel Curry, MD, PhD, Texas Children's Hospital and Baylor College of Medicine.]



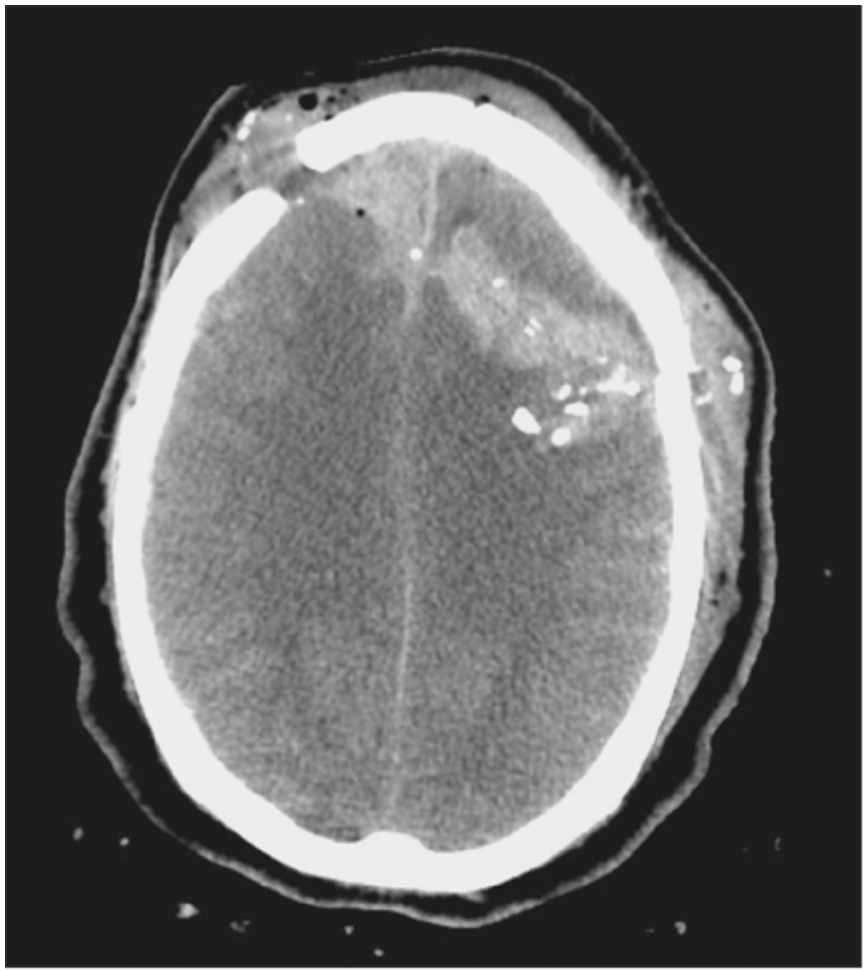
PENETRATING INJURY

As a bullet passes through the brain, it creates a cavity three to four times larger than its diameter. Direct penetration of the bullet through the brain substance and the transfer of kinetic energy cause the majority of the destruction (**Figure 257-12**). The GCS can be used to predict the

prognosis for nonintoxicated patients with a gunshot wound to the brain. Patients with a GCS score of >8 and reactive pupils have a 25% mortality risk, whereas mortality approaches 100% in those with a GCS score of <5. Patients with a penetrating gunshot wound to the brain should be intubated and treated with prophylactic antibiotics, such as vancomycin, 1 gram IV, and ceftriaxone, 2 grams IV.

FIGURE 257-12.

Gunshot wound traversing through frontal lobes bilaterally; note bone fragments. [Image used with permission of Thomas Egglin, MD, Director of Emergency Radiology and an Associate Professor of Diagnostic Imaging at the Warren Alpert Medical School of Brown University, Providence, RI.]



Stab wounds have very low energy and impart only direct damage to the area contacted by the penetrating object. Patients with penetrating injury require admission, broad-spectrum antibiotics, and operative intervention. **Leave impaled objects in place until controlled surgical removal is facilitated**.

MILD TRAUMATIC BRAIN INJURY

mTBI (often called a concussion) is impairment in brain function without overt hemorrhage or other gross lesions, is caused by an external force, and results in a GCS score of 14 or 15 (also see chapter 110 and **Table 257-3**).[1] The diagnosis is made by a history of any alteration in consciousness at the time or shortly after the inciting event (acceleration-deceleration or blunt force). Alteration in consciousness includes the individual's account of "getting his/her bell rung," "seeing stars," or being dazed or confused as a result of the force. The presence of amnesia further supports the diagnosis and is often associated with more significant injury.

Signs and symptoms such as vomiting, headache, loss of consciousness, focal neurologic deficit, age >65 years, coagulopathy, and/or dangerous mechanism of injury are factors that increase risk of serious injury (see Tables 257-4, 257-5, and 257-6).[26,27,30,36] The presence of alcohol, distracting injuries, and other barriers to obtaining a clear history of the event confound the signs and symptoms of mTBI.

PATHOPHYSIOLOGY

In its mildest form, mTBI is an ionic shift that causes a momentary disruption in function. Symptom recovery is rapid, and the concussive injury results in no obvious structural damage. However, mild insults can also cause a temporary upregulation of ion channels, especially along axons.[37,38] After a single injury, the ion channel density returns to normal over time. Repeated exposure to injury, however, greatly increases the resting number of channels. An increase in the density of ion channels leaves the brain vulnerable to overactivation, neuronal toxicity, and cell death.

In addition to ion channel upregulation, large shifts of balance in ion concentrations may lead to mitochondrial dysfunction and depletion of intracellular energy stores in mTBI.[14,39,40,41] This state creates a metabolic "mismatch" during which neuronal dysfunction persists until recovery occurs. This pathway is a recognized pattern of injury in moderate/severe TBI and is thought to also play a role in mTBI.

Metabolic insults, electrochemical imbalances (calcium influx and sodium and potassium shifts), and mitochondrial dysfunction also result in damage to axonal transport systems. Structural abnormalities are not always identified on MRI or CT. However, histopathology shows microscopic injury. Indeed, evidence of damage on diffusion tensor imaging has been demonstrated in high school athletes after a single football

season, even without clinical signs or symptoms of a concussion.[42,43] Chronic traumatic encephalopathy[44] is hypothesized to occur as a result of repeated exposure to TBI in sports.[45,46,47]

Repetitive concussions can result in long-term cognitive deficits and structural damage to the brain.[48,49,50,51] In extreme cases, when a second concussion occurs prior to recovery from the first, rapid onset of cerebral edema and death can occur, particularly in athletes prematurely returning to play (**second impact syndrome**).[52]

DIAGNOSIS

The physical examination findings in isolated mTBI are often normal. Currently, there are no reliable tests that can confirm the diagnosis of concussion. The GCS lacks the detail to assess the full spectrum of signs and symptoms. Head CT scans are usually normal, and a normal scan only eliminates the concern for an underlying lesion requiring surgery.

Perform a thorough neurologic examination and obtain the GCS. Focal findings suggest potential intracranial pathology or a postictal state. Assess for signs of global impairment, such as confusion, perseveration, or amnesia.[53] Observe gait and test balance. **The most consistent abnormalities in mTBI are subtle impairments in cognitive function (see Table 257-7)**. The gold standard written neuropsychological examination is impractical to perform in the ED setting. However, perform some assessment of cognition.

Clinical symptoms (**Table 257-11**) may begin immediately after the insult or may be delayed for days to weeks. Therefore, **the lack of obvious signs and symptoms at the time of evaluation does not exclude mTBI if the historical account is consistent with such injury**. Another complicating factor is that many of the signs and symptoms are nonspecific and overlap with those of other conditions.

TABLE 257-11

Signs and Symptoms of mTBI

Cognitive Symptoms	Physical Signs and Symptoms	Behavioral Changes
Attention difficulties	Headaches	Irritability
Concentration problems	Dizziness	Depression
Amnesia and perseveration	Insomnia	Anxiety
Short-term and long-term memory problems	Fatigue Uneven gait	Sleep disturbances Emotional lability
Orientation problems	Nausea, vomiting	Loss of initiative
Altered processing speed	Blurred vision	Loneliness and helplessness
Altered reaction time Calculation difficulties and problems with executive function	Seizures	Problems related to job, relationship, home, or school management

Note: At 3 months after injury, <30% are symptomatic; at 1 year, 15% are symptomatic.

The practice of grading concussions is widely employed but is not evidence based. There are >20 different classification systems in existence.[54]

Biomarkers

Serum markers specific to neurologic injury may improve future diagnosis and management.[36,55,56]

Of the biomarkers currently under study, S100B (calcium binding protein B antibody) is the only one that is relatively sensitive (94% to 99% sensitive) for detecting the presence of injury, but only under specific conditions. S100B serum levels rise and fall rapidly, so time from injury determines the relevance of a negative finding. S100B is also not currently approved by the U.S. Food and Drug Administration for mTBI. The American College of Emergency Physicians provides the following guidance: "***Level C recommendations****. In mTBI patients without significant extracranial injuries and a serum S-100B < 0.1 μg/L measured within 4 hours of injury, consideration can be given to not performing a CT.*"[57]

Cognitive Screening and Psychometrics

Cognitive testing in the ED is currently limited to the use of brief memory screens such as the **Mini-Cog** (see Figure 288-2) or the **Quick Confusion Scale** (see Table 286-7). Other tools, primarily developed for sports, such as the Sports Concussion Assessment Tool, the Standardized Assessment for Concussion, and other similar instruments, have not been validated for ED use.

Neuropsychological testing[58,59,60,61] consists of a battery of individual tests that evaluate a number of domains required for normal brain function, including memory, attention, concentration, executive function, and reaction time. Several of these tests are used by sports programs to assess recovery from concussion. They are most valuable when baseline scores are available for comparison.

TREATMENT AND DISPOSITION

The primary treatment for mTBI is rest. **Make sure the patient avoids aspirin and nonsteroidal anti-inflammatory drugs after acute injury**. ED treatment objectives are to identify patients who have intracranial lesions requiring neurosurgical intervention; to admit patients whose condition might deteriorate over time; and for those discharged, to provide instructions for cognitive and physical rest and provide follow-up for reassessment before return to normal activities.

When the patient is safe for discharge, one of the most important "interventions" is to provide thorough concussion discharge instructions. A template is available at http://www.cdc.gov/ncipc/tbi/Physicians_Tool_Kit.htm. Physical and neurologic rest is needed until symptoms abate fully. Discharge the patient to the care of a responsible individual and provide instructions to both the patient and that individual. Patients with mTBI may not comprehend or remember detailed discharge instructions. Have the patient return to the ED for increasing symptoms, headaches, altered mental status, nausea, or vomiting. Refer patients for further evaluation and follow-up care. Patients at an increased risk for reinjury, such as athletes, should undergo a formal graduated return-to-activity program (**Table 257-12**).

TABLE 257-12

Return-to-Activity Program

Sports Related	Non–Sports Related
No activity (rest until symptom-free)	No activity (rest until symptom-free)
Light aerobic exercise	Light aerobic exercise
Sport-specific training (noncontact)	Moderate aerobic exercise
Noncontact drills	Return to normal activities
Full-contact drills	
Game play	

Note: Patient must remain asymptomatic for 24 hours between each step. Development of symptoms at any level requires return to the previous symptom-free level.

Return to Activity

Symptoms reflect underlying metabolic dysfunction and are currently the only reliable guide to brain health.[40,54] Return to play or work decisions are based on symptoms and a graduated evaluation program.

Assessments incorporate serial symptom checklists, neuropsychological tests (memory and reaction time assessment), and a balance evaluation (see Table 257-12).[54,62] **Because this type of assessment is not practical in the ED, ED clinicians should not provide definitive return-to-activity directions**.

SPECIAL CONSIDERATIONS

POSTCONCUSSIVE SYNDROME

Patients often report a series of physical, emotional, and cognitive symptoms in the days and weeks after mTBI. The estimated prevalence of postconcussive syndrome varies widely, with about 20% to 40% of patients reporting symptoms at 3 months and about 15% at 1 year.[63,64] The most commonly reported postconcussion symptoms are headache, dizziness, decreased concentration, memory problems, sleep disturbances, irritability, fatigue, visual disturbances, judgment problems, depression, and anxiety.[65] When a cluster of symptoms becomes chronic after mTBI, they are often called **persistent postconcussive symptoms** or **postconcussion syndrome**. Clinical findings at the time of the injury do not reliably predict the development of postconcussive syndrome. Postconcussive syndrome symptoms can overlap those of posttraumatic stress disorder. Neuropsychological testing and use of a symptom checklist are the cornerstones of diagnosis and management. Treatment is symptomatic. Refer patients to a neuropsychologist or mTBI clinic.

RECURRENT CONCUSSIONS

Three or more concussions pose a risk for long-term sequelae, especially in adolescents and young children.[48,49,66] Almost all cases of **second impact syndrome** have occurred in young athletes. **Chronic traumatic encephalopathy**,[44] characterized by early onset of memory loss and depression, is a concern in professional athletes. Pathologically large deposits of tau protein are seen in the brains of deceased chronic traumatic encephalopathy patients.[47] Tau protein deposits have recently been discovered even in youth football players who died from other causes.

SECOND IMPACT SYNDROME

Second impact syndrome is a rare disorder that results in rapid cerebral edema and high mortality (60% to 80%).[52] The pathophysiology and the predictors are not well understood. It is hypothesized that occurrence of a second impact before the brain has reset or recovered from a first mTBI causes a loss of autoregulation and ion imbalance, and leads to rapid cerebral edema. This explanation fits well with the concept of enhanced vulnerability due to metabolic disturbances, energy-demand mismatch, and ion channel upregulation after a concussion.[67,68]

ANTICOAGULATION

Anticoagulants and antiplatelet agents increase the risk of intracranial hemorrhage after injury, especially in the elderly.

Intracranial hemorrhage in patients taking warfarin and who have an elevated INR is associated with a high mortality rate (89%).[69] In general, patients with head trauma, who are taking anticoagulants or antiplatelet agents, should undergo emergent head CT. The OR for the risk of intracranial lesions after mild head injury in patients taking any antiplatelet therapy is 2.6.[70] Clopidogrel seems to be a potent risk factor.[71] The effect of low-dose aspirin (162 mg or less taken daily) on post-head injury bleeding has not been determined.

Patients with intracranial hemorrhage need immediate anticoagulant reversal. Patients taking Warfarin who have an elevated INR are optimally treated with plasma or 4-factor concentrate. See chapters 239, "Thrombotics and Antithrombotics" and 166, "Spontaneous Subarachnoid and Intracerebral Hemorrhage" for further discussion.

A negative initial CT finding in an asymptomatic TBI patient receiving anticoagulation or antiplatelet therapy is reassuring, but delayed hemorrhage may occur and is not easily predicted. [71]

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USEFUL WEB RESOURCES

Acute Concussion Evaluation (ACE), Emergency Department (ED) Version v1.4—http://www.cdc.gov/ncipc/tbi/ACE_ED.pdf

Brain Trauma Foundation—http://www.braintrauma.org

Heads Up: Brain Injury in Your Practice—A Tool Kit for Physicians, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control—http://www.cdc.gov/ncipc/tbi/Physicians_Tool_Kit.htm