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Head Injury

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EPIDEMIOLOGY

Injuries are the leading cause of death in persons younger than 45 years old with approximately one-third of these deaths a result of head trauma. Traumatic brain injury (TBI) results from either direct or indirect forces to the brain matter. Direct injury is immediate and caused by the force of an object striking or penetrating the head. Indirect injuries from acceleration/deceleration forces are generated by the variable movements of different areas of the brain against one another and by the impact of the brain against the skull.

Annually, in the United States, there are approximately 1.5 million nonfatal TBIs, 370,000 persons hospitalized as a result of a TBI, and 52,000 persons who die from TBI.1 TBI also leads to 80,000 annual cases of residual neurologic disability. The costs for treatment of both acute and chronic TBI are estimated to be $4 billion dollars annually.2

The peak incidence of TBI occurs in males between the ages of 15 and 24 years, but ethanol-intoxicated individuals, the elderly, and young children are at increased risk of TBI because of underlying anatomic and physiologic factors. The cause of TBI varies greatly by age and demographic factors. For example, for 15-to 24-year-olds the leading cause is gunshot wounds, while for those older than age 65 years it is falls.

ANATOMY

The outermost layer, the scalp, is composed of five layers: skin, subcutaneous tissue, galea, areolar tissue, and the pericranium. Because of the rich blood supply, the scalp has a major role in temperature regulation, being capable of liberating 50 percent of our total body heat. This same generous blood supply, combined with the loose areolar connection to the pericranium, can lead to severe blood loss after injury.

The calvaria or skull is a rigid container made up of eight major bones. These bones are composed of two solid layers separated by cancellous bone, which adds rigidity and strength. The cranial sutures between bones initially serve as expansion joints, but eventually fuse, halting expandability in adults.

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Because the basilar skull is the exit and entry point for the cranial nerves and blood vessels basilar fractures places these structures at risk. The *inion* is the anatomic meeting point of the frontal, sphenoid, temporal, and parietal bones. Fractures of the skull at this point can disrupt the underlying middle meningeal artery, leading to

epidural hematoma.

The adult brain weighs between 1300 and 1500 g and occupies 80 percent of the total volume of the skull. The brain's three basic structures—the cerebral hemispheres, the cerebellum, and the brainstem—are divided by two major fixed dura attachments. The *falx cerebri* vertically separates the cerebral hemispheres down to the brainstem. The *tentorium cerebelli* separates the cerebellum and brainstem from the cerebrum at the base of the skull. The inner edge of the tentorium cerebelli is the site of the most common brain herniation syndrome, uncal herniation. The cerebrum is further anatomically divided into major lobes named after the bones overlying them: frontal, temporal, parietal, occipital. The cortices encase the deeper brain structures such as the basal ganglia, the major systems integration site. The cerebellum occupies the posterior cranial fossa, and is responsible to pattern motor memory and balance. The brainstem contains the cranial nerve nuclei and the inflow and outflow functional somatic tracts.

The brain is covered with multiple anatomic layers and potential spaces. The outermost layer, the *dura mater*, is firmly adhered to the inner skull and has fixed attachments at the cranial sutures. At some edges of dural reflections, the dura separates into two layers and forms channels called the dural venous sinuses, which serve to drain blood and cerebrospinal fluid from the brain. Underneath the dura mater is a thinner connective tissue layer called the *arachnoid mater*. The arachnoid mater perforates the dura mater at the venous sinuses where it forms arachnoid granulations. The arachnoid granulations serve as filtration and drainage points for the cerebrospinal fluid. The arachnoid mater is loosely adhered to the pia mater making possible the potential subarachnoid space. The *pia mater* is closely associated with the gray matter of the brain and is the innermost layer. Between the arachnoid and the pia is the *subarachnoid space*, where cerebrospinal fluid (CSF) circulates. In the average adult, there is 150 mL of CSF surrounding the brain and spinal cord. Approximately 500 mL of CFS is produced in the choroid plexus of the lateral ventricles each day.

Several subarachnoid spaces known as *cisternae* surround the brain and correspond to large cortical surface irregularities. These include the ambient, prepontine, supracerebellar, cerebellomedullary, interpeduncular, superior, and magna. There are four spaces contained within the brain known as *ventricles:* two lateral ventricles (separated by the septum pellucidum), a third ventricle, and a fourth ventricle. These CSF-containing spaces communicate by foramina: Monroe (between the lateral and third ventricle), aqueduct of Sylvius (between the third and fourth ventricle), and foramen of Luschka and Magendie (outlets from the fourth ventricle into the cerebellomedullary cistern and cisterna magna).

PATHOPHYSIOLOGY

Acute brain injury is usually divided into primary and secondary phases. In the case of traumatic brain injury, the acute or primary phase describes the cellular injury and death that are a direct result of force of the injury. Primary cell death is irreversible and only preventing the injury event and mitigation of the injury forces on the brain reduce morbidity and mortality.

Secondary injury cascades can extend the damage to cells that are not initially irreversibly damaged. In the hours to weeks after the injury, local tissue ischemia from compressive forces or vascular injury lead to secondary cellular death. Prevention of

the ischemia and hypoxia are the main therapeutic goals for treating patients with TBI.

Normal Physiology

The cerebrovascular system delivers energy substrates and oxygen while simultaneously removing the byproducts of metabolism. The brain accounts for only 2 percent of total body weight, but consumes 20 percent of the body's total oxygen requirement and 15 percent of total cardiac output. Maintaining adequate brain tissue perfusion is critical to avoid secondary brain injury. The cerebral perfusion pressure (CPP) is the difference between inflow and outflow (ΔP) and is essentially the driving pressure for cerebral blood flow (CBF). Estimates of CPP assume that the relevant inflow pressure is equivalent to the mean arterial pressure (MAP) and the outflow is related to intracranial pressure (ICP). The ICP is used to estimate outflow pressure because the venous system is collapsible at the entry point into the sinuses and changes in this outflow pressure occurs in parallel with increases in the ICP. The CPP is calculated in lieu of CBF:

$$CPP = MAP - ICP$$

The local adjustment of CBF within the brain microcirculation that result from changes in arterial pressure is termed “autoregulation.” Autoregulation of CBF is functional with CPP between 50 and 150 mm Hg, but is often lost in patients with TBI where normal relationship between flow and perfusion may be affected. A pressure-volume relationship occurs in the brain microvasculature that illustrates cerebral autoregulation (Figure 255-1). Under normal circumstances the major regulatory mechanisms of CBF are the partial pressure of carbon dioxide (PCO_2), blood pressure, and blood pH. Hypotension or hypoventilation leads to increased PCO_2 and decreased pH. Under these circumstances, the cerebral vasculature dilates to increase CBF and deliver more oxygen. Hypertension, hypocarbia, and alkalosis cause vasoconstriction. Decreased partial pressure of oxygen (PO_2) also leads to increased CBF by causing cerebral vasodilation.

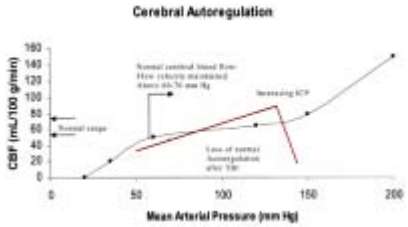

FIG. 255-1. Normal cerebral blood flow (CBF) autoregulation curve and the abnormal curve with TBI. Normal autoregulatory control *(black line)* maintains a relatively constant CBF over a broad range of mean arterial pressure (MAP). Loss of autoregulation results in a more linear relationship between CBF and MAP, potentially resulting in increased edema and intracranial pressure (ICP) *(red line)*. Increases in ICP may result in a net loss in CBF.

Pressure-Volume Relationship in Brain Injury

Circulation to the brain is vulnerable to conditions that increase the intracranial volume. Normal ICP is less than 15 mm Hg and 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 must be a compensatory reduction in the volume of another or the ICP will increase.

When the ICP is outside its normal range autoregulation is lost, and the CBF follows a more linear passive-pressure relationship to CPP. As a result, normal cerebral blood flow may not be restored until the CPP is increased to greater than 90 mm Hg.3 Cerebral blood flow is generally maintained when the CPP is above 60 to 70 mm Hg in the setting of hypotension and elevated ICP. This CPP level is considered the lower limit of autoregulation, below which local control of CBF cannot be adjusted to maintain flow adequate for function.

Rapid rises in the ICP that cause compression of the brain can lead to a phenomenon known as the *Cushing reflex* (hypertension, bradycardia, and respiratory irregularity). This triad is classic for an acute rise in ICP, but is seen in only one-third of cases, and is more common in children than adults.

Mechanisms of Secondary Cellular Death

Neurons that survive the primary insult face several secondary injury mechanisms that are characterized as *excitotoxicity, oxidative stress, inflammation*, and *apoptosis* (programmed cell death).

Immediately following the acute injury event there is a decrease in the production of ATP, affecting ATP-dependent processes. This results in influx of sodium and water leading to cellular edema, cellular acidosis from anaerobic metabolism, and calcium influx with the release of the excitatory amino acid glutamate. Glutamate is the central initiator of excitotoxicity, leading to multiple lethal reactions that alter the structural and functional integrity of the cell. If the tissue is reperfused, the damage is compounded as free radicals are produced (oxidative stress). Iron-dependent lipid peroxidation products lead to further membrane damage.

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The local production of cytokines causes the inflammatory component of secondary injury. Migration of neutrophils, monocytes, and macrophages into the site of injury amplifies the injury to surviving cells. These cells contribute to the oxidative stress and inflammatory cascades by releasing proteases, free radicals, and vasoconstrictive mediators.

Finally, cells that escape early death may die from an endogenous mechanisms of cell death, broadly called *apoptosis*. The cellular triggers and mediators of apoptosis are beginning to be defined. Treatments that disrupt these secondary cellular injury pathways are still being developed, so the primary clinical treatment remains the prevention of increased ICP, ischemia, and hypoxia.

INITIAL EVALUATION AND MANAGEMENT

Because preventing secondary brain injury is the major goal of TBI management, correcting or preventing hypoxemia, hypotension, anemia, hyperglycemia, and hyperthermia, and evacuation of intracranial masses is critically important. Hypotension [systolic blood pressure (SBP) <90 mm Hg] and hypoxemia (PaO_2 <60) are associated with a doubling of TBI mortality.3 Anemia (hematocrit <30 percent) also leads to

increased mortality because of decreased oxygen-carrying capacity.

Airway control with cervical spine stabilization, breathing, and circulation are the first priorities for all trauma patients. Only then can a neurologic and mental status examination be conducted to evaluate disability. Early recognition and treatment of hypoxemia, hypotension, and anemia are essential.

Prehospital Care

The care of the head-injured patient begins with emergency medical services (EMS) personnel in the prehospital setting. In addition to the ABCs (airway, breathing, circulation), assessing the history of the event and the patient's condition and mental status immediately after the injury is important. Hypoxia and hypotension need to be identified and corrected rapidly on the scene. There is no indication for prehospital hyperventilation or use of mannitol for patients with TBI.

ED Resuscitation

Important historical points include the mechanism of injury, the patient's condition before and after the trauma, the past medical history, and the recent use of drugs or alcohol. Important information regarding the condition after the injury includes the length of the loss of consciousness, vomiting, and if seizure activity occurred. A history of anticoagulant use or a coagulopathy should be determined. As with all patients with altered mental status, the potential for other injuries such as hypothermia, inhalation injuries, and toxic exposures should be investigated.

AIRWAY AND BREATHING

Hypoxia increases mortality from TBI, therefore aggressive airway and breathing management is needed. All patients with severe TBI require intubation and ventilation initially with 100 percent O_2. In-line cervical spine stabilization is essential until cervical spine injury or fracture is definitively excluded. Orotracheal rapid-sequence intubation (RSI) using appropriate agents optimizes the patient's physiology, prevents increased ICP, and has the lowest complication rate (see Chap. 19). The ideal induction agent should both blunt the increase in ICP and yet not decrease the MAP. Because some of the drugs commonly used for RSI (Table 255-1) cause hypotension, strict attention needs to be paid to assure hemodynamic stability during the procedure.

TABLE 255-1 Recommended Agents for Rapid Sequence Intubation of Patients with Severe TBI

Preinduction agents* (prevent ICP elevation)
Succinylcholine 0.1 mg/kg IV *or*
Vecuronium 0.01 mg/kg IV or pancuronium 0.01 mg/kg IV
Induction agents (sedative/hypnotics and analgesia)
Thiopental 3–5 mg/kg IV (normotensive) or 0.5–1.0 mg/kg IV (hypotensive)
Fentanyl 3–5 µg/kg IV
Propofol 1–4 mg/kg IV

Neuromuscular blocking agents (long-acting agents are not recommended for TBI patients)
- Succinylcholine 1.0–1.5 mg/kg IV

*Not clinically validated to improve outcome in TBI patients.

Lidocaine has been used as a pretreatment agent because of its potential to prevent hemodynamic changes and elevation of intracranial pressure, but recent reports question its utility.4 A defasciculating dose of succinylcholine (0.1 mg/kg IV), vecuronium (0.01 mg/kg IV), or pancuronium (0.01 mg/kg IV) given 2 to 3 min prior to succinylcholine has been used to prevent RSI-induced increases in ICP, but again, to date there are no clinical trials that demonstrate improved outcome with preinduction agents.

Barbiturates decrease ICP and decrease cerebral metabolic oxygen demand. Therefore, a short-acting barbiturate can be used as an induction agent. Thiopental (3 to 5 mg/kg IV) has a rapid onset and is short-acting.

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Because thiopental is a cardiovascular depressant, patients who are hypotensive should receive only 0.5 to 1 mg/kg or another inducting agent, such as etomidate (0.3 mg/kg). Ketamine has traditionally been avoided as it was thought to cause ICP elevation; however, some studies have shown a decrease in ICP when ventilation is controlled. There is renewed interest in ketamine because it blocks potentially deleterious amino acid [*N*-methyl-D-aspartate (NMDA)] receptors in the brain.5 Further study in the ED, however, is warranted before a recommendation can be made.

The use of etomidate (0.3 mg/kg) for induction in the ED has not been studied extensively. Those studies that have looked at the effects of etomidate administration on ICP and CPP (in either the intensive care unit or operating room) in head-injured patients have been inconclusive, with mixed reports.6

Propofol as an induction agent has several theoretical benefits (rapid onset/recovery and minimal hemodynamic effects). Propofol is commonly used for RSI in the operating setting; however, few reports exist for use in the ED for severely head-injured patients.

Neuromuscular blocking drugs (NMBDs) are used to facilitate airway control and to prevent complications without increasing ICP. The use of NMBDs prevents any meaningful neurologic exam and limits the ability to detect changes in the condition of the patient (e.g., seizure). Therefore the shortest-acting agent, succinylcholine is usually chosen.

RSI techniques should also be used for comatose patients, who are susceptible to increases in ICP with laryngoscope and intubation. Nasotracheal intubation is not specifically contraindicated in TBI, but it should rarely be used because of its higher complication rate, lower success rate, and potential for marked increase in ICP. In the presence of a basilar skull fracture, nasotracheal intubation also leads to an increased

risk of meningitis.

CIRCULATION

Improved blood pressure resuscitation decreases mortality for patients with severe TBI.7 Therefore, aggressive fluid resuscitation may be required to prevent hypotension and secondary brain injury. Adequate fluid resuscitation has not been shown to increase ICP and guidelines recommend that the MAP be maintained at 90 mm Hg (systolic blood pressure of 120 to 140 mm Hg) to achieve adequate cerebral perfusion.8 Isolated head injury rarely produces hypotension, except as a preterminal event. Hypovolemic shock may be seen in infants because of epidural bleeding or subgaleal hematoma or with massive blood loss from scalp lacerations in any age group. If fluid resuscitation is not effective, vasopressors should be used to maintain MAP at 90 mm Hg to preserve CPP. External and internal bleeding need to be controlled quickly and the hematocrit should be maintained above 30 percent.

Hypertension is a critical finding and must be assumed to be an indicator of increased ICP in a patient with a head injury. This *Cushing reflex* requires immediate measures to decrease the intracranial pressure. If hypertension exists independent of increased ICP, then the systemic pressure should be lowered by no more than 30 percent of the MAP by using labetalol.

DISABILITY AND THE NEUROLOGIC EXAMINATION

The neurologic examination that is part of the primary survey describes the patient's level of consciousness with the AVPU system: *alert*, responds to *verbal* stimuli, responds to *painful* stimuli, or *unresponsive*. This same assessment provides most of the information needed to calculate the Glasgow Coma Scale (Table 255-2). The GCS is only one component of a complete neurologic examination. The best possible neurologic examination should be conducted as soon as the initial resuscitation is complete, to serve as a baseline for subsequent serial examinations and to dictate treatment.

TABLE 255-2 The Glasgow Coma Scale for All Age Groups

	4 years to Adult	Child <4 years	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 moves
5	Localizes pain	Localizes pain	Withdraws to touch
4	Movement or withdrawal to pain	Withdraws to pain	Withdraws to pain
3	Decorticate	Decorticate flexion	Decorticate

	flexion		flexion
2	Decerebrate extension	Decerebrate extension	Decerebrate extension
1	No response	No response	No response
3–15			

Note: GCS reporting should be modified for intubated and paralyzed patients.

The GCS was developed as a standardized scoring system to allow reliable interobserver neurologic assessment of patients with TBI. An accurate GCS score is used to direct treatment and predict outcome. Accurate GCS scores can only be obtained after resuscitation, and prior to sedation or intubation. The postresuscitation GCS is based upon three factors: eye opening, verbal function, and motor function. When assessing the GCS in the severely obtunded, noxious stimuli need to be delivered in the form of nailbed pressure on both sides of the body or severe sternal pressure. This is important for grading the depth of disability at the level of more rudimentary reflexes. The best response from each category is added for a score of 3 to 15. The GCS for intubated patients cannot be assessed for verbal response, and paralyzed patients cannot be assessed at all. For nonintubated, nonsedated postresuscitation patients **a GCS of less than 9 is considered a severe TBI, moderate is 9 to 13, and mild is 14 to 15**.

The other important aspect of the neurologic examination is pupil assessment (size, reactivity, and anisocoria). In an unresponsive patient a single fixed and dilated pupil may indicate an ipsilateral intracranial hematoma with uncal herniation that requires rapid opera-tive decompression. Direct ocular trauma should also be considered. Bilateral fixed and dilated pupils suggest increased ICP with poor brain perfusion,

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bilateral uncal herniation, drug effect or severe hypoxia, whereas bilateral pinpoint pupils suggest either opiate use or a pontine lesion.

Altered motor function can indicate brain, spinal cord, or peripheral nerve injuries. Movement in unresponsive patients is assessed by noxious stimuli to a nail bed in all extremities. Decorticate posturing (upper extremity flexion and lower extremity extension) indicates an injury above the midbrain. Decerebrate posturing (arm extension and internal rotation with wrist and finger flexion and internal rotation and extension of the lower extremities) is from a more caudal injury and predicts a worse outcome. For completely unresponsive patients, the respiratory pattern and eye movements will provide information regarding brainstem function. Oculovestibular (cold calorics) and oculocephalic (doll's eyes) responses are not checked until the cervical

spine has been fully cleared.

After the primary survey a secondary survey is needed to identify other significant injuries.

THE CLINICAL SPECTRUM OF TBI

Mild Traumatic Brain Injury

Severity grading of TBI uses the GCS score to categorize patients based on their potential outcomes. Mild TBI has traditionally included head-injured patients with a history of loss of consciousness, amnesia or loss of memory to the event, any change in mental status at the time of event, and/or persistent or transient focal neurological deficit. The convention has been to include patients with GCS scores of 13 to 15 in to the mild group, which accounts for most TBI patients. However, because of the wide variation in outcomes those previously described as “mild head injury,” are further subdivided into “low risk,” “medium risk” or “high risk” mild head injury.8 Low-risk mild-injury patients include those with a GCS of 15 without a history of loss of consciousness, amnesia, vomiting, or diffuse headache. The risk for intracranial hematoma requiring surgical evacuation is less than 0.1 percent. Medium-risk patients have a GCS of 15 along with one or more of the following: loss of consciousness, amnesia, vomiting, or diffuse headache. These patients have a 1 to 3 percent risk of intracranial hematoma requiring surgical evacuation and CT scans should be obtained for such patients. If computed tomography (CT) is not available, skull x-rays are recommended, and those patients with fractures should be placed in the *high-risk* category and be emergently transferred to a facility with neurosurgical availability (Figure 255-2). The medium-risk mild TBI group includes those patients meeting diagnostic criteria for postconcussion syndrome (PCS).

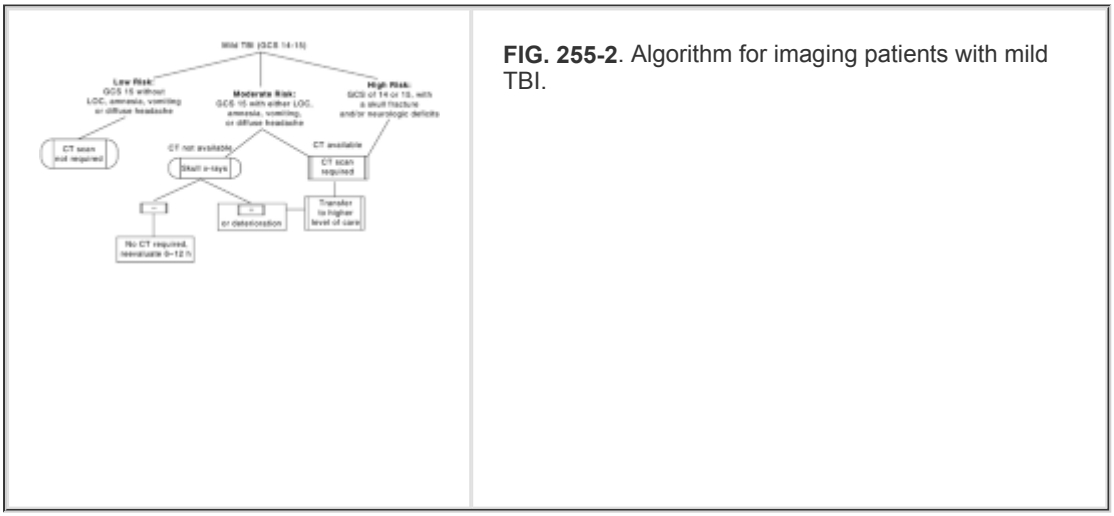

FIG. 255-2. Algorithm for imaging patients with mild TBI.

High-risk mild head-injury patients are those with an admission GCS of 14 or 15, with a skull fracture and/or neurologic deficits. As many as 10 percent of the patients in this *high-risk* mild TBI group will require surgical evacuation of intracranial hematomas. Patients with coagulopathy, drug or alcohol consumption, previous neuro-surgical procedures, epilepsy, or age greater than 60 years should be included in the high-risk group independent of their clinical presentation.

The rate of injury findings on brain CT scans increases within this mild injury group as a

function of the GCS.9 Regardless of the need for neurosurgical intervention, nearly 10 percent of high-risk, mild TBI patients with a GCS of 15 will have a positive brain CT, as compared to 40 percent in those with a GCS of 13.9

Mild TBI patients with a GCS of 15, no indication for head CT or a negative CT, and adequate supervision may be safely discharged home from the emergency department. Those patients with a GCS of 14 and a negative head CT should be observed for 6 to 12 h for deterioration (accumulation of edema, seizure, etc.). Those patients with a positive CT scan or persistent neurologic findings require neurosurgical consultation and hospital admission (Figure 255-3).

FIG. 255-3. Algorithm for the evaluation and treatment of mild brain injury.

Moderate Traumatic Brain Injury

Moderate TBI (GCS 9 to 13) accounts for approximately 10 percent of patients with head injuries. Mortality rates for patients with isolated moderate TBI is less than 20 percent, but long-term disability is as high as 50 percent. Overall, 40 percent of moderate TBI patients have a finding on CT scan and 8 percent require neurosurgical intervention.10 Patients with moderate TBI should be admitted to try to prevent deterioration from secondary brain injury and progression to severe TBI (Figure 255-4). Most patients in this category should be intubated, receive neurosurgical consultation, and admission to a monitored unit.

FIG. 255-4. Algorithm for the evaluation and treatment of moderate brain injury.

Severe Traumatic Brain Injury

In severe TBI (GCS <9), mortality approaches 40 percent, with most deaths occurring within 48 h. Less than 10 percent of these patients make even a moderate recovery. The management of patients with severe TBI has three primary goals: to identify other life-threatening

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injuries, to prevent further secondary brain injury, and to identify treatable mass lesions. All patients with severe TBI require a CT scan and should be admitted to an intensive care unit in a hospital with neurosurgical capabilities (Figure 255-5).

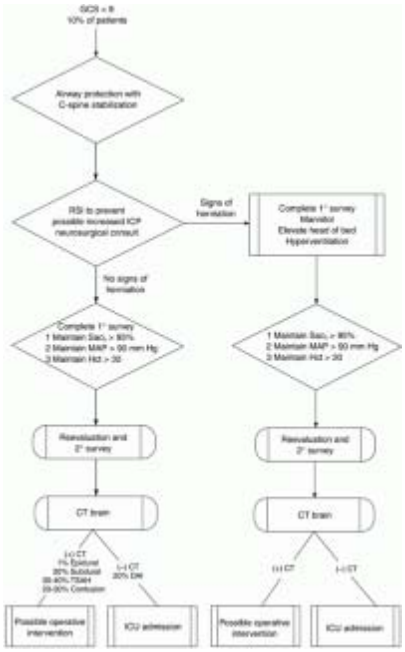

FIG. 255-5. Algorithm for the evaluation and treatment of severe brain injury.

DIAGNOSTIC RADIOGRAPHY FOR TBI

All patients with moderate to severe TBI require urgent head CT scans after stabilization. Delays in obtaining a CT scan can lead to a catastrophic delay in emergent neurosurgical interventions. Therefore, **if a patient with TBI is uncooperative or combative, RSI is often the best option**. If this is undertaken, a GCS score and the best neurologic exam possible should be performed prior to intubation. Sedation alone should be used infrequently as it is often ineffective and may put patients at risk for further injury or aspiration. Other means to control agitated patients with TBI include haloperidol (5 to 10 mg IM or IV, may repeat), midazolam (1 to 2 mg IV), and propofol (20 mg every 10 s to desired effect). A CT scan is generally diagnostic but there have been

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reports of delayed intracranial hematomas, especially subdural hematomas, for patients with nonfocal neurologic examinations with negative initial CT scans.11

CT of the head is indicated for all patients with a GCS of 14 or less. There are

numerous factors that dictate the need for a CT in patients with a GCS of 15 (Table 255-3). In addition, liberal use of head CT in patients of older than 60 years of age is recommended.12

TABLE 255-3 Indications for CT Scanning for Patients with Mild TBI

Signs
A Glasgow Coma Scale 13–14
A skull fracture or large subgaleal swelling
Focal neurologic findings
Unexplained asymmetric pupils
Distracting injuries or intoxication
Symptoms
A reported loss of consciousness or posttraumatic seizure
A history of coagulopathy
Continued diffuse headache
Amnesia
Vomiting

INCREASED ICP AFTER TBI

An intracranial pressure of greater than 20 to 25 mm Hg in the TBI patient increases subsequent morbidity and mortality. In the ED, ICP monitoring is rarely available, therefore physical findings must be used to identify increased ICP and to direct therapy. Indicators of increased ICP include signs of transtentorial herniation (unilateral or bilateral dilated pupils, hemiparesis, motor posturing) and progressive neurologic deterioration (as determined by repetitive AVPU and GCS examinations). Several strategies may be used to lower ICP. All patients with severe TBI and evidence of increased ICP should have the head of the bed elevated to 30 degrees, volume resuscitation to a MAP of 90 mm Hg or a 30 percent reduction in MAP if hypertensive, and maintenance of arterial oxygenation. After these steps mannitol should be used.

Mannitol

Mannitol is the best osmotic agent to reduce ICP. It has beneficial effects on the ICP, CBF, CPP, and brain metabolism. Additionally, mannitol is known to scavenge free radicals. It reduces ICP within 30 min and lasts variably up to 6 to 8 h. Mannitol has the additional benefit of expanding volume, initially reducing hypotension, and improving the blood's oxygen-carrying capacity. Mannitol is administered by repetitive bolus (0.25 g/kg to 1 g/kg), not by constant infusion. Because there is no dose-dependent effect seen with mannitol, some authors advocate the lower range of the suggested dose. Mannitol will lead to a net intravascular volume loss because of its diuretic effect, therefore adequate glomerular filtration rate (GFR) hemodynamic and input-output monitoring is required to maintain euvolemia.

Hyperventilation

Hyperventilation is no longer recommended as a prophylactic intervention during the first 24 h after a severe TBI.14 Nor is hyperventilation below a PaO_2 of 25 mm Hg ever indicated. Hyperventilation does reduce the ICP, but the vasoconstriction caused by reducing carbon dioxide (CO_2) levels also leads to cerebral ischemia.

Hyperventilation is still used as a last resort for hospitalized patients with signs of increasing ICP despite other therapeutic measures. In this case it is a temporary measure and the PaO_2 is monitored closely to maintain the range of 30 to 35 mm Hg.

Other Modalities

Initiation of barbiturate coma is not indicated in the ED, but there is evidence that it may be considered for hemodynamically stable patients with severe TBI who have continued increased ICP despite other therapies. The recommended dose of pentobarbital is 10 mg/kg over 30 min.

There is some indication that prophylactic anticonvulsants reduce the occurrence of early post-traumatic seizures, but no evidence that these drugs improve long-term outcome. They should be used in consultation with a neurosurgeon. There is no indication to use steroids for the treatment of TBI or increased ICP.13

Intracranial Pressure Monitoring

Internal monitoring of ICP should be initiated on all patients with evidence of increased ICP, herniation, and a GCS score of less than 9. This treatment has the most favorable risk:benefit ratio. A ventricular catheter offers the best method to directly monitor ICP and thus calculate CPP. Increases in the ICP above 20 to 25 mm Hg may be reduced by CSF drainage. If CSF drainage is not effective in reducing ICP, then mannitol should be administered, (assuming adequate MAP).

TREATMENT OF SPECIFIC HEAD INJURIES

Scalp Lacerations

Scalp lacerations can lead to massive blood loss. Scalp hemorrhages should be controlled as rapidly as possible. If direct pressure is not effective, lidocaine with epinephrine can be infiltrated locally, and vessels can be clamped or ligated. Wounds should be carefully examined prior to closure for underlying fractures and galeal lacerations. Large galeal disruptions should be repaired, but small ones can be left alone.

Skull Fractures

All patients suspected or found to have a skull fracture require a head CT scan (see Table 255-3). Skull fractures are usually categorized by location (basilar vs. the skull convexity), pattern (linear, depressed, or comminuted) and whether they are open or closed. A careful examination and wound exploration of scalp lacerations is needed to identify a skull fracture in the patient without a loss of consciousness or neurologic findings. Complicated skull fractures include those that are open or depressed, those that involve a sinus, and those that cause intracranial air (pneumocephalus).

LINEAR AND COMMINUTED FRACTURES

The management of linear and simple comminuted skull fractures is the same. Isolated fractures with an overlying laceration require careful evaluation, cleaning, and repair. Wounds should be explored gently so as not to drive bone fragments into the brain. The use of prophylactic antibiotics following an open skull fracture is controversial and should be discussed with the neurosurgeon.

Particular care should be taken in evaluating fractures that cross the middle meningeal artery or a major venous sinus. Linear occipital fractures also have higher intracerebral complication rates. Fractures that are depressed beyond the outer table of the skull require operative repair.

BASILAR SKULL FRACTURE

The most common basilar skull fracture involves the petrous portion of the temporal bone, the external auditory canal, and the tympanic membrane. It is commonly associated with a torn dura leading to CSF leakage from the ear. Signs and symptoms associated with basilar skull fractures include CSF otorrhea or rhinorrhea, mastoid ecchymosis (Battle sign), periorbital ecchymoses (raccoon eyes), hemotympanum, vertigo, decreased hearing or deafness,

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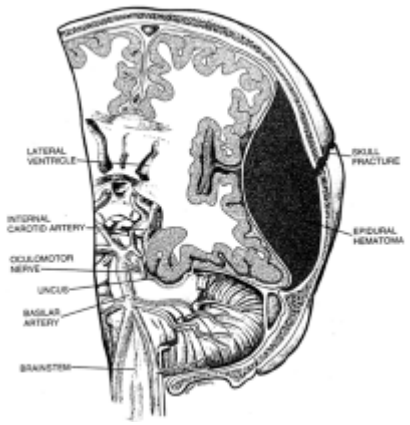
and seventh nerve palsy. Periorbital and mastoid ecchymoses develop gradually over hours after an injury and are often absent in the ED. CSF leaks are difficult to diagnose. A simple but nonsensitive test for a basilar skull fracture with a CSF leak is to see if the fluid leaves a single or double ring when dripped onto a paper towel. Leaking fluid can also be tested for a specific CSF protein, transferrin. Patients with a basilar skull fracture should be hospitalized.

Patients with CSF fistulas can develop meningitis. Prophylactic antibiotics reduce the incidence of meningitis for patients with basilar skull fractures, thus prophylactic antibiotics in those patients with a basilar skull fracture is recommended.14 Administration of antibiotics should be done in consultation with a neurosurgeon who will be following the patient. If prophylactic antibiotics are instituted, they should have broad coverage with good penetration into the meninges. A third-generation cephalosporin such as ceftriaxone at 1 to 2 g per day is a reasonable choice.

Brain Herniation

There are four major brain herniation syndromes: uncal and central transtentorial, cerebellotonsillar, and upward posterior fossa. The most common is uncal and it occurs when the uncus of the temporal lobe is displaced inferiorly through the medial edge of the tentorium (Figure 255-6). This is usually caused by an expanding lesion in the temporal lobe or lateral middle fossa. Uncal transtentorial herniation leads to compression of the third cranial (occulomotor) nerve, causing an ipsilateral fixed and dilated pupil. Further herniation compresses the pyramidal tract leading to contralateral motor paralysis. In some cases, the pupillary changes are contralateral, or motor changes are ipsilateral.

	FIG. 255-6. Anterior view of a transtentorial uncal



herniation caused by a large hematoma under a skull fracture.

Central transtentorial herniation is less common and occurs with midline lesions in the frontal or occipital lobes, or in the vertex. The most prominent symptoms are initial bilateral pinpoint pupils, bilateral Babinski 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 causes 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, pinpoint pupils, and rapid death.

When all other methods to control the ICP have failed, patients with signs of herniation may need emergency decompression by trephination (“burr holes”). CT scanning before attempting trephination is recommended to localize the lesion and direct the decompression site. If CT scan is unavailable, or the patient is unstable for CT because of signs of rapidly progressive neurologic deterioration and herniation, then emergency department trephination should be considered.

Cerebral Contusion/Intracerebral Hemorrhage

Contusions are one of the most frequent types of TBI. Contusions most commonly occur in the subfrontal cortex, in the frontal and temporal lobes, and, occasionally, in the occipital lobes. They are often associated with a subarachnoid hemorrhage. Contusions may occur at the site of the blunt trauma or the opposite site of the brain, known as a “contre-coup” injury.

Intracerebral hemorrhage can occur days after significant blunt trauma, often at the site of resolving contusions. This complication is more common with patients with a coagulopathy. CT scans in the immediate postinjury phase can be normal.

Subarachnoid Hemorrhage

Traumatic subarachnoid hemorrhage (tSAH) results from the disruption of subarachnoid vessels and presents with blood in the CSF (Figure 255-7). Patients can present with mild to severe TBI. Those with isolated tSAH often present with a headache and photophobia and mild meningeal signs. tSAH is probably the most common CT abnormality in patients with moderate to severe TBI. Patients with a tSAH are twice as likely to suffer from death, persistent vegetative state, or severe disability. Early

development of tSAH (42 percent) represents a trifold mortality risk (42 vs. 14 percent), as compared to those without tSAH.15 The amount of blood seen on the CT scan is inversely related to the presenting GCS and percent of patients with a positive outcome. Some tSAH can be missed on early CT scans. Generally, scans performed 6 to 8 h after injury are more sensitive for detecting tSAH. Unfortunately, findings may be delayed in a few patients. Careful follow-up instructions, referral for reexamination by a physician, and discharge in the care of a competent adult are necessary, even with a normal CT scan.

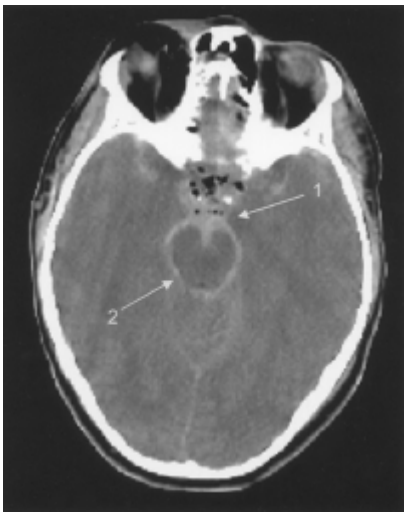


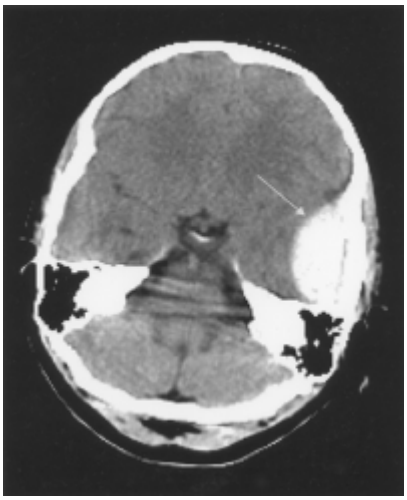
FIG. 255-7. Computed tomography scan illustrating a subarachnoid hemorrhage. *Arrow 1* illustrates prepontine cisternal blood and *arrow 2* represents blood in the ambient cistern.

The use of nimodipine in patients with tSAH reduces the likelihood of death or severe disability by 55 percent as compared to placebo.16 Nimodipine should be started as soon as possible after stabilization at 2 mg per h for 7 to 10 days followed by 360 mg daily until day 21. Patient with tSAH requires immediate neurosurgical consultation and admission to an intensive care unit.

Epidural Hematoma

Epidural hematomas are the result of blood collecting in the potential space between the skull and the dura mater (Figure 255-8). Most epidural hematomas result from blunt trauma to the temporal or temporoparietal area with an associated skull fracture and middle meningeal arterial disruption. Occasionally, trauma to the parieto-occipital region or the posterior fossa causes tears of the venous sinuses with epidural hematomas. Almost all epidural hematomas are associated with skull fractures, and some will have additional cerebral lesions.

FIG. 255-8. Computed tomography scan illustrating a lenticular epidural hematoma. Note that the hematoma does not cross the suture lines.



The classic history of an epidural hematoma is a lucent period following immediate loss of consciousness after significant blunt head trauma.

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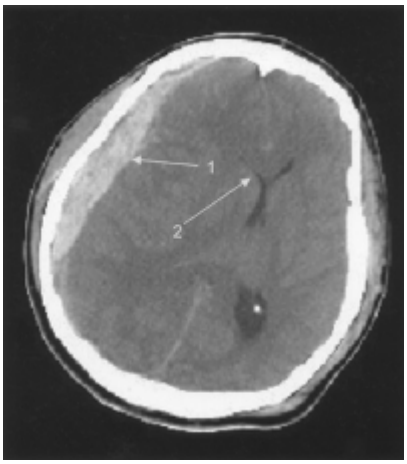
The patient then awakens prior to again falling unconscious. This clinical pattern occurs in a minority of cases. Most patients either never lose consciousness or never regain consciousness after the injury. The diagnosis of an epidural hematoma is based on CT scan and physical findings. On CT scans, epidural hematomas appear biconvex (football shaped), typically in the temporal region.

The high-pressure arterial bleeding of an epidural hematoma can lead to herniation within hours after an injury. Therefore, early recognition and evacuation is key to reduce morbidity and mortality. Bilateral emergency department trephination (burr holes) is only indicated if definitive neurosurgical care is not available. Full recovery can be expected if the hematoma is evacuated prior to herniation or the development of neurologic deficits.

Subdural Hematoma

Subdural hematomas (SDHs) are caused by sudden acceleration-deceleration of brain parenchyma with subsequent tearing of the bridging veins. This results in blood clots forming between the dura mater and the arachnoid (Figure 255-9). Brains with extensive atrophy, as in the elderly and in alcoholics, are more susceptible to subdural hematomas. Children younger than age 2 years are also at increased risk of subdural hematomas. This is related to transfer of the force that limits the risk of sustaining epidural hematomas.

FIG. 255-9. Computed tomography scan of a subdural hematoma (*arrow 1*). Note that the hematoma crosses the suture lines. *Arrow 2* demonstrates midline shift.



Blood tends to collect more slowly than in epidural hematomas because of its venous origin. However, SDHs are often associated with other brain injuries.

Traditionally, subdural hematomas have been classified as acute, subacute, or chronic, depending on time they present. Acute SDH symptoms usually develop within 14 days of the injury. After 2 weeks, patients are defined as having a chronic SDH. There is no specific clinical syndrome associated with a subdural hematoma. Acute cases usually present immediately after severe trauma and often the victim is unconscious. Chronic subdural hematomas may present in the elderly or in alcoholics with vague complaints or mental status changes. These patients often do not recall an injury. On CT scan, acute SDHs are hyperdense (white), crescent-shaped lesions that cross suture lines. Subacute SDHs are isodense and more difficult to identify. A CT scan with intravenous contrast can assist in identifying a subacute SDH. A chronic SDH appears hypodense (dark) as the iron in the blood is phagocytized.

The definitive treatment of subdural hematomas depends on the type and on associated brain injuries. Mortality and the need for surgical repair are greater for acute and subacute SDH. Chronic subdurals hematomas can sometimes be managed without surgery depending of the severity of the symptoms. Table 255-4 compares intra-cranial hematomas.

TABLE 255-4 Intracranial Hematomas

	Type of Patient	Anatomic Location	CT Findings	Common Cause	Class Symp
Epidural	Young, rare in	Potential space	Biconvex, football-	Skull fracture with tear of	Imme LOC

	the elderly and age <2 years	between skull and dura mater	shaped hematoma	the middle meningeal artery	"lucid prior deter (only abou
Subdural	More risk for the elderly and alcoholics	Space between dura mater and arachnoid	Crescent-or sickle-shaped hematoma	Acceleration–deceleration with tearing of the bridging veins	Acute LOC-perio possi Chro altere and b with g decre cons
Subarachnoid	Any age group following blunt trauma	Subarachnoid	Blood in the basilar cisterns and hemispheric sulci and fissures	Acceleration–deceleration with tearing of the subarachnoid vessels	Mild t mode with meni signs symp
Contusion/intracerebral hematoma	Any age group following blunt trauma	Usually anterior temporal or posterior frontal lobe	May be normal initially with delayed bleed	Severe or penetrating trauma; shaken-baby syndrome	Symp range norm unco

Abbreviations: LOC = loss of consciousness; MS = mental state; TBI = traumatic brain injury.

Diffuse Axonal Injury

Diffuse axonal injury (DAI) is the disruption of axonal fibers in the white matter and brainstem. Shearing forces on the neurons generated by sudden deceleration cause DAI. Classically, DAI is seen after blunt trauma, such as from a motor vehicle accident. In infants, the "shaken-baby syndrome" is a well-described tragic cause.

Injury occurs immediately and is essentially irreversible. There is a rapid or immediate increase in ICP. Patients present unresponsive. The state may be prolonged or permanent until death. A CT scan of a patient with DAI may be normal, but the classic CT demonstrates hemorrhagic injury to the mostly the deep structures of the brain. The

treatment options for DAI are very limited, but attempt to prevent secondary damage by reducing cerebral edema.

Penetrating Injury

As a bullet passes through the brain, it creates a cavity three to four times greater than its diameter (see Chap. 264). Direct penetration of the bullet through the brain substance and the transfer of kinetic energy cause the majority of the destruction. The GCS can be used to predict the prognosis of unintoxicated patients with a gunshot wound to the brain. Patients with a GCS >8 and reactive pupils have a 25 percent mortality risk, while those with a GCS <5 approach 100 percent mortality. All patients with a penetrating gunshot wound to the brain should be intubated and treated with prophylactic antibiotics.

Stab wounds have very low energy and impart only direct damage to the area contacted by the penetrating object. This results in a much lower morbidity and mortality than from gunshot wounds. Essentially all patients sustain penetrating injury require admission, broad-spectrum antibiotics and operative intervention. Impaled objects should be left in place until surgical removal.

COMPLICATIONS/LONG-TERM PROBLEMS

Seizures

The reported incidence of early posttraumatic seizures ranges from 2 to 5 percent, but this incidence rises to near 30 percent in children, alcoholics, and those with intracranial hematomas. A significant association exists between early posttraumatic seizures and unfavorable outcome, but this association is small by comparison to subdural hematoma and cerebral contusion.17 Prophylactic antiepileptic drugs given after TBI decreases the incidence of early posttraumatic seizures, but there has been no observed reduction in the occurrence of late-seizures, death, or neurologic disability.18 Thus, at the present time, **use of prophylactic antiepileptic drugs is not supported by the literature**. Acute management of early posttraumatic seizures is the same as for managing any seizure in the ED, but requires close hemodynamic monitoring to prevent antiepileptic drug-induced hypotension or hypoventilation.

Concussion and Postconcussive Syndrome

Concussion is defined as any alteration of cerebral function caused by a force to the head resulting in one or more of the following: a brief loss of consciousness; light-headedness; vertigo; headache; nausea; vomiting; photophobia; cognitive and memory dysfunction; tinnitus; blurred vision; difficulty concentrating; amnesia; fatigue; personality change; or a balance disturbance.19 In 30 to 80 percent of patients with TBI, symptoms will remain 3 months postinjury; in 15 percent of patients, symptoms will remain at 1 year.20 Persistence of these signs or symptoms has been termed “postconcussion syndrome” (PCS) (Table 255-5). Prior to discharge from the ED patients should be warned that their symptoms usually clear, but the time needed is generally weeks to months. In addition, they should be referred to a neurologist for long-term care.

TABLE 255-5 Postconcussion Syndrome: Complex Involving Somatic, Cognitive, and

Affective Symptoms

Somatic
Headache
Sleep disturbance
Nausea
Sensitivity to photostimuli
Dizziness/vertigo
Fatigue
Cognitive
Memory dysfunction
Concentration difficulty
Attention deficit
Affective
Depression
Anxiety
Irritability
Emotional lability

Note: At 3 months 30–80% are symptomatic; at 1 year 15% are symptomatic.

Postconcussive syndrome patients continue to have complaints such as headaches, dizziness, inability to concentrate, and memory changes. After 1 year, 85 to 90 percent of these patients recover. The remaining

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patients have “persistent postconcussion syndrome” (PPCS). Risk factors for patients PPCS are female sex, ongoing litigation, and low socioeconomic status. While all PCS and PPCS patients should be referred to their primary physicians or neurologist, amitriptyline at 25 to 50 mg PO per d helps with headaches, depression, and insomnia.

Sports-related concussions are common injuries, but their true incidence is greatly underreported. Sports most commonly associated with concussion are boxing, football, soccer, baseball, and basketball. The management of any patient with TBI or concussion is the same as described above. However, for the athlete with a concussion, special consideration must be made for their safe return to a sporting activity. Neurophysiologic assessment instruments are typically used in the hours following TBI and then repeated 5 days after the injury. Some of the tools used for this assessment include the Trail-making test, Stroop test, and the Digit span from the Wechsler Memory Scale, Revised.19 **When athletes present with symptoms of concussion, they should be referred for testing prior to being cleared to return to sports**.

Infections

Several factors increase the risk for infections following a head injury. Skull fractures and CSF leaks are risk factors for meningitis. In addition to central nervous system (CNS) infections, intubated patients on neuromuscular blockade have longer intensive

care unit stays, an increased risk for aspiration pneumonias, and tend toward sepsis. Because of controversies regarding use of prophylactic antibiotics, consultation with the neurosurgeon or intensivist should be obtained prior to their administration.

Patients who present with a history of a skull fracture and fever or other symptoms of meningitis should be treated with antibiotics immediately. The source of infection depends on the time since the injury. In the first 72 h after a head injury, pneumococcus is generally the cause. After that, gram-negative organisms and *Staphylococcus aureus* become more common. Patients should be given vancomycin (1 g IV q12h) and a third-generation cephalosporin, such as ceftazidime 1 g IV q8h, until cultures confirm the cause.

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