Traumatic Brain Injury

Samir Parikh, MD
Marcella Koch, MD
Raj K. Narayan, MD
University of Cincinnati College of Medicine
Cincinnati, Ohio

Traumatic brain injury (TBI) is physical injury to brain tissue that temporarily or permanently impairs brain function. Diagnosis is suspected clinically and confirmed by imaging [primarily computed tomography (CT), although magnetic resonance imaging (MRI) can be helpful later when it is logistically possible to obtain]. Initial treatment consists of ensuring a reliable airway and maintenance of adequate ventilation and blood pressure. Surgery is often needed in more severe cases to remove intracranial hematomas, provide room for the brain to swell, or place monitors to track intracranial pressure (ICP) and brain oxygenation. In the first few days after the injury, significant efforts are made to maintain adequate brain perfusion and oxygenation, and prevent complications that can result from an altered sensorium. Various periods of rehabilitation are often needed.

In the United States, as in much of the world, TBI is a common cause of death and disability.[1–3] Causes include motor vehicle crashes and other transportation-related causes (eg, bicycle crashes, collisions with pedestrians), falls (especially in older adults and young children), assaults, and sports activities.

Pathology

Head injuries can cause various types of structural damage. Structural changes may be gross or microscopic, depending on the mechanism and forces involved. Patients with less severe injuries may have no gross structural damage. Clinical manifestations vary markedly

Reprints: Samir Parikh, MD, Department of Neurosurgery, University of Cincinnati College of Medicine, 231 Albert Sabin Way, PO Box 670515, Cincinnati, OH 45267-0515, E-mail: Samir.Parikh2@uc.edu

in severity and consequences. Injuries are commonly categorized as open or closed.

Open injuries involve penetration of the scalp and skull (and usually the meninges and underlying brain tissue). They typically involve bullets or sharp objects, but a skull fracture with overlying laceration due to severe blunt force is also considered an open injury.

Closed injuries typically occur when the head is struck, strikes an object, or is shaken violently, causing rapid brain acceleration and deceleration. Acceleration or deceleration can injure tissue at the point of impact (coup), at its opposite pole (contrecoup), or diffusely; the frontal and temporal lobes are particularly vulnerable. Axons, blood vessels, or both can be sheared or torn. Blood vessels that are disrupted leak, producing contusions, intracerebral or subarachnoid hemorrhages, and hematomas (epidural and subdural).

■ Concussion

Concussion is defined as transient and reversible posttraumatic alteration in mental status (eg, loss of consciousness or memory) lasting from seconds to minutes and, by arbitrary definition, <6 hours.[4] Gross structural brain lesions and serious neurologic residua are not part of concussion, although temporary disability can occur and postconcussion symptoms such as nausea, headaches, dizziness, and memory disturbances can be considerably disabling.[5,6]

■ Diffuse Axonal Injury

Diffuse axonal injury (DAI) occurs when deceleration causes shear-type forces that result in generalized, widespread disruption of axonal fibers and myelin sheaths (although DAI may also result from minor head injury).[7,8] Gross structural lesions are not part of DAI, but small petechial hemorrhages in the white matter are often observed on CT scan (and histopathologic examination).[9] DAI is sometimes defined clinically as a loss of consciousness lasting >6 hours in the absence of a specific focal lesion. Edema from the injury often increases ICP, leading to various manifestations. DAI is typically the underlying injury in shaken baby syndrome.

■ Brain Contusions

Contusions (bruises of the brain) can occur with open or closed injuries and can impair a wide range of brain functions, depending on contusion size and location.[10] Larger contusions may cause brain edema and increased ICP. Contusions may grow and evolve

into larger lesions that are referred to as intracerebral hematomas. The distinction between contusions and intracerebral hematomas is not well defined.

■ Hematomas

Hematomas (collections of blood in or around the brain) can occur with open or closed injuries and may be epidural, subdural, or intracerebral. Subarachnoid hemorrhage (bleeding into the subarachnoid space) is common in TBI.

A subdural hematoma is blood between the dura mater and the pia-arachnoid mater. Acute subdural hematomas, which are often caused by laceration of brain or cortical veins or avulsion of bridging veins between the cortex and dural sinuses, often occur after falls or motor vehicle crashes. Compression of the brain by the hematoma, along with swelling of the brain due to edema or hyperemia (engorged blood vessels) can result in increased ICP. When these processes coexist, mortality and morbidity can be high.[11–13]

A chronic subdural hematoma may appear and produce symptoms gradually over several weeks after trauma. These hematomas occur more often in elderly patients (especially in those taking antiplatelet drugs or anticoagulants, or in those with brain atrophy).[14,15] These patients may consider the head injury relatively trivial or may have even forgotten it. In contrast to acute subdural hematomas, edema and increased ICP are unusual in patients with chronic subdural hematomas.

Epidural hematomas (blood between the skull and dura mater) are less common than subdural hematomas.[16,17] Expanding epidural hematomas are usually caused by arterial bleeding, classically owing to damage to the middle meningeal artery by a temporal bone fracture. Without intervention, patients with arterial epidural hematomas may rapidly deteriorate and die. Small, venous epidural hematomas are rarely lethal. Surgery is indicated for arterial hematomas, while venous epidurals can usually be managed without surgery.[18]

■ Skull Fractures

Penetrating injuries by definition involve fractures. Closed injuries may also cause skull fractures, which may be linear, depressed, or comminuted. Although serious and even fatal TBI may occur without skull fracture, the presence of a fracture suggests that significant force was involved. Fractures in patients with diffuse head trauma indicate increased risk of intracranial hematomas.[19,20] Also, a simple linear skull fracture is not usually high risk unless there is neurologic impairment or the fracture occurs in an infant.

Depressed fractures have the highest risk of tearing the dura, damaging the underlying brain, or both.

If temporal bone fractures cross the area of the middle meningeal artery, an epidural hematoma is more likely. Fractures crossing one of the major dural sinuses may cause significant hemorrhage and venous epidural or venous subdural hematoma. Fractures that involve the carotid canal can result in carotid artery dissection.[21]

Because the occipital bone and base of the skull (basilar bones) are thick and strong, fractures in these areas indicate a high-intensity impact. Basilar skull fractures that extend into the petrous part of the temporal bone often damage middle and inner ear structures and can impair facial, acoustic, and vestibular nerve function.

In infants, the meninges may become trapped in a linear skull fracture with subsequent development of a leptomeningeal cyst and "growth" of the original fracture (thus, called a growing fracture).

■ Pathophysiology

Brain function may be immediately impaired by direct damage (eg, crush, laceration) of brain tissue. Further damage may occur shortly thereafter from the cascade of events triggered by the initial injury.[22,23]

TBI of any sort can produce edema in the damaged tissues. The cranial vault is fixed in size (constrained by the skull) and almost completely filled by noncompressible fluid [cerebrospinal fluid (CSF)] and minimally compressible brain tissue; consequently, any swelling from edema, or an intracranial hematoma has nowhere to expand and thus increases ICP. Cerebral blood flow is proportional to the cerebral perfusion pressure (CPP), which is the difference between mean arterial pressure (MAP) and ICP. Thus, as ICP increases (or MAP decreases), CPP decreases, and when it is below about 50 mm Hg, the brain may become ischemic.[24] This mechanism may produce ischemia at a local level when compression from focal edema or hematoma compromises blood flow in the region of the lesion.[25] Ischemia and edema then trigger mechanisms (eg, release of excitatory neurotransmitters free radicals, and cytokines, cell membrane damage) causing further edema, which further increases ICP.[22,26,27] Systemic complications from trauma (eg, hypotension, hypoxia) can also contribute to cerebral ischemia and are often called secondary brain insults.

Excessive ICP initially causes global cerebral dysfunction. If excessive ICP is unrelieved, it can push brain tissue across the tentorium or through the foramen magnum, causing herniation, which increases morbidity and mortality risk. Also, if ICP increases to equal MAP, CPP becomes zero, resulting in complete brain ischemia, which rapidly leads to brain death; absent cranial blood flow is objective evidence of brain death.

■ Symptoms and Signs

Initially, most patients with significant TBI lose consciousness (usually for seconds or minutes), although with minor injuries, some have only confusion or amnesia (amnesia is usually retrograde and lasts for seconds to a few hours). Young children may simply become irritable. Some patients have seizures, often within the first hour or day. After these initial symptoms, patients may be fully awake and alert, or consciousness and function may be altered to some degree, from mild confusion to stupor to coma. Duration of unconsciousness and severity of obtundation are roughly proportional to injury severity but are not specific. The Glasgow Coma Scale (GCS; Table 1) is a quick, reproducible scoring system to be used during the initial examination to estimate severity of TBI.[28] It is based on eye opening, verbal response, and the best motor response. The lowest total score of 3 indicates likely fatal damage, especially if both pupils fail to respond to light and oculovestibular responses are absent. Higher initial scores tend to predict better recovery.[29–31] By convention, the severity of head injury is initially defined by the GCS (GCS score of 13 to 15 is mild TBI; GCS score of 9 to 12 is moderate TBI; GCS score of 3 to 8 is severe TBI); however, the severity and prognosis are predicted more accurately by also considering CT scan findings and other factors. Some patients with initially moderate TBI and a few patients with initially

Table 1. *Glasgow Coma Scale**

Area Assessed	Response	Points
Eye opening	Open spontaneously	4
	Open verbal command, speech, or shouting	3
	Open in response to pain applied to the limbs or sternum	2
	None	1
Verbal	Oriented	5
	Disoriented, but able to answer questions	4
	Inappropriate answers to questions; words discernible	3
	Incomprehensible speech	2
	None	1
Motor	Obeys commands	6
	Responds to pain with purposeful movement	5
	Withdraws from painful stimuli	4
	Reponds to pain with abnormal flexion (decorticate posture)	3
	Responds to pain with abnormal extension (decerebrate posture)	2
	None	1

*Combined scores <8 are typically regarded as coma.
Adapted from *Lancet*. 1974;2:81–84.

mild TBI deteriorate. It is important to assess the GCS after the patient has undergone cardiopulmonary resuscitation because the neurologic examination is very sensitive to hypotension, hypoxia, and pharmacologic sedation.[32] Symptoms of various types of TBI may overlap considerably.

Symptoms of epidural hematoma usually develop within minutes to several hours after the injury ("lucid interval") and consist of increasing headache, decreased level of consciousness, focal neurologic deficits (eg, hemiparesis), and pupillary dilation with loss of light reactivity.[33,34] Some patients lose consciousness, followed by a transient lucid interval, then gradual neurologic deterioration. Subdural hematomas are usually associated with an immediate loss of consciousness. However, any type of expanding intracranial pathology (hematoma, edema, hyperemia) can result in a progressive loss of consciousness.

Vomiting may indicate increased ICP but is nonspecific. Markedly increased ICP classically manifests as a combination of hypertension (usually with increased pulse pressure), bradycardia, and respiratory depression (Cushing's triad); respirations are usually slow and irregular.[35,36] Severe diffuse brain injury or markedly increased ICP may produce decorticate or decerebrate posturing. Regardless, both are poor prognostic signs.

Transtentorial herniation may result in coma, unilaterally or bilaterally dilated and unreactive pupils, hemiplegia (usually on the side opposite a unilaterally dilated pupil), and Cushing's triad.

Basilar skull fracture may result in leakage of CSF from the nose (CSF rhinorrhea) or ear (CSF otorrhea), blood behind the tympanic membrane (hemotympanum) or in the external ear canal if the tympanic membrane has been ruptured, and ecchymosis behind the ear (Battle's sign) or in the periorbital area (raccoon eyes). Loss of smell and hearing are usually immediate although they may not be noticed till the patient regains cognition. Facial nerve function can be impaired immediately or develop in a delayed fashion. Other fractures of the cranial vault are sometimes palpable, particularly through a scalp laceration, as a depression or step-off deformity. However, blood under the galea aponeurotica may mimic such a step-off deformity.

Patients with chronic subdural hematomas may present with increasing daily headache, fluctuating drowsiness or confusion (which may mimic early dementia), and mild-to-moderate hemiparesis, or other focal neurologic deficits.

■ Long-term Symptoms

Amnesia may persist and be both retrograde and anterograde. Postconcussion syndrome, which commonly follows a moderate or severe concussion, includes headache, dizziness, fatigue, difficulty concentrating, variable amnesia, depression, apathy, and anxiety.[5,37,38]

Commonly smell (and thus taste), sometimes hearing, or rarely vision is altered or lost.[39] Symptoms usually resolve spontaneously over weeks to months.

A range of cognitive and neuropsychiatric deficits can persist after severe and even moderate TBI, particularly if structural damage was significant. Common problems include amnesia, behavioral changes (eg, agitation, impulsivity, disinhibition, lack of motivation), emotional lability, sleep disturbances, and decreased intellectual function.

Late seizures (>7 d after the injury) develop in a small percentage of patients, often weeks, months, or even years later. Spastic motor impairment, gait and balance disturbances, ataxia, and sensory losses may occur.

A persistent vegetative state is a rare complication of TBI that destroys forebrain cognitive functions but spares the brain stem. The capacity for self-aware mental activity is absent; however, autonomic and motor reflexes and normal sleep-wake cycles are preserved. Few patients recover normal neurologic function when a persistent vegetative state lasts for 3 months after injury, and almost none recover after 6 months.

Neurologic function tends to improve for at least 2 years after TBI, although it occurs most rapidly during the initial 6 months.

■ Diagnosis

An initial overall assessment of injuries should be done; diagnosis and treatment occur simultaneously in seriously injured patients.

A rapid, focused neurologic evaluation is a part of the initial assessment, including assessment of the components of the GCS, adequacy of the airway and breathing, and pupillary light response. Patients are ideally assessed before paralytics and sedatives are given. Patients are reassessed at frequent intervals (eg, q 15 to 30 min initially, then q 1 h after stabilization). Subsequent improvement or deterioration helps estimate injury severity and prognosis. Complete neurologic examination is done as soon as the patient is sufficiently stable. Infants and children should be examined carefully for retinal hemorrhages, which may indicate shaken baby syndrome.[40,41] Fundoscopic examination in adults is insensitive for elevated ICP in the TBI patient and cannot be relied upon to rule out intracranial pathology.

Imaging is generally performed in all patients with a history of significant TBI, imaging should always be performed in patients with more than transiently impaired consciousness, GCS score <15, focal neurologic findings, persistent vomiting, seizures, or clinically suspected fractures. However, a case can be made for obtaining a CT scan of the head in all patients with more than a trivial head injury, because the clinical and medicolegal consequences of missing a hematoma are severe.

Although plain x-rays can detect some skull fractures, they cannot help assess the brain and are not used routinely. CT is the best choice for initial imaging; it can detect hematomas, contusions, skull fractures (thin cuts are obtained to reveal clinically suspected basilar skull fractures, which may otherwise not be visible), and sometimes DAI. On CT scan, contusions and acute bleeding appear as more radiopaque (dense) than brain tissue. Subdural hematomas classically appear as crescent-shaped opacities overlying brain tissue.[42] Arterial epidural hematomas classically appear as lenticular-shaped opacities over brain tissue, often in the territory of the middle meningeal artery.[42] A chronic subdural hematoma may have a similar radiopacity as brain tissue (isodense). Isodense subdural hematoma, particularly if bilateral and symmetric, may appear only subtly abnormal. Among individual patients, lesions often differ from these classic appearances. Signs of increased ICP include sulcal effacement, ventricular and cisternal compression, and midline shift. A septal shift of more than 5 mm from the midline is considered to be significant and is generally considered to be an indication for surgical evacuation of the hematoma.[43]

MRI may be useful later in the clinical course to detect more subtle contusions and DAI; it is usually more sensitive than CT for the diagnosis of very small acute or isodense subacute and isodense chronic subdural hematomas. There is early evidence that certain MRI data may be of prognostic value.[44–46] Angiography, CT angiography, or MR angiography are all useful modalities for the evaluation of vascular injury.

■ Prognosis

In the United States, adults with severe TBI who are treated have a mortality rate of about 25% to 33%; mortality is lower with higher GCS scores.[47] Mortality rates are lower in children $\geq$5 years ($\leq$10% with a GCS score of 5 to 7).[48] Children overall do better than adults with a comparable injury.[49]

Most of the patients with mild TBI retain good neurologic function. With moderate or severe TBI, the prognosis is not as good but is much better than is generally believed. The most commonly used scale to assess outcome in TBI patients is the Glasgow Outcome Scale.[50] On this scale, the possible outcomes are good recovery (return to previous level of function), moderate disability (patient capable of self-care), severe disability (incapable of self-care), vegetative (no cognitive function), and death. Over 50% of adults with severe TBI have a good recovery or moderate disability. Occurrence and duration of coma after a TBI are strong predictors of disability; of patients whose coma exceeds 24 hours, 50% have major persistent neurologic sequelae, and 2% to 6% remain in a persistent vegetative state at 6 months. In adults with severe TBI, recovery occurs most rapidly within the initial 6 months; smaller

improvements continue for perhaps as long as several years. Children have a better immediate recovery from TBI regardless of severity and continue to improve for a longer period of time.[48]

Cognitive deficits, with impaired concentration, attention, and memory, and various personality changes are a more common cause of disability in social relations and employment than are focal motor or sensory impairments[51–53] Posttraumatic anosmia and acute traumatic blindness seldom resolve after 3 to 4 months. Hemiparesis and aphasia usually abate, except in the elderly.

■ Treatment

Multiple noncranial injuries, which are likely with motor vehicle crashes and falls, often require simultaneous treatment.

At the injury scene, a clear airway is secured and external bleeding is controlled before the patient is moved. Particular care is taken to avoid displacement of the spine or other bones to avoid damaging the spinal cord and blood vessels. Proper immobilization should be maintained with a cervical collar and long spine board until stability of the entire spine has been established by appropriate examination and imaging. After the initial rapid neurologic assessment, pain should be relieved with a short-acting opioid (eg, fentanyl).

In the hospital, after quick initial evaluation, neurologic findings (GCS and pupillary reaction), blood pressure, pulse, and temperature should be recorded frequently for several hours because any deterioration demands prompt attention. CT and serial GCS results stratify injury severity within the categories defined by GCS, which helps guide treatment.

The cornerstone of management for all patients is maintenance of adequate pulmonary gas exchange and brain perfusion to avoid secondary brain insult.[54–58] Aggressive early management of hypoxia, hypercapnia, hypotension, and increased ICP helps avoid secondary complications. Bleeding from injuries (external and internal) is rapidly controlled as required, and intravascular volume is promptly replaced with crystalloid (eg, 0.9% saline) or sometimes blood transfusion to maintain cerebral perfusion. Hypotonic fluids (especially D5W) are contraindicated because they contain excess free water, which can increase brain edema and ICP.[59,60]

Other complications to check for and prevent include hyperthermia, hyponatremia, hyperglycemia, and fluid imbalance.[61,62]

■ Mild Injury

Injury is mild (by GCS score) in 80% of patients who have TBI and present to an emergency department.[63] If there is brief or no loss of consciousness and if patients have stable vital signs, a normal head

CT scan, and normal mental and neurologic function, they may be discharged home provided family members or friends can observe them closely for an additional 24 hours. These observers are instructed to return patients to the hospital if any of the following develop: decreased level of consciousness, focal neurologic deficits, worsening headache, vomiting, or deterioration of mental function.

Patients who have minimal or no neurologic changes but have minor abnormalities on head CT generally have a follow-up CT scan of the head about 4 to 8 hours after the initial study. Patients who have loss of consciousness of a duration longer than a few minutes, any abnormalities in mental or neurologic function, or cannot be observed closely after discharge are generally admitted for observation and follow-up CT.

■ Moderate and Severe Injury

Injury is moderate in 10% of patients who have TBI and present to an emergency department.[64] They often do not require intubation and mechanical ventilation (unless other injuries are present) or ICP monitoring. However, because deterioration is possible, these patients should be admitted and observed even if head CT is normal.

The remaining patients who present to an emergency department have severe TBI.[64] They are admitted to a critical care unit. Because usually airway protective reflexes are impaired and ICP may be elevated, patients are intubated endotracheally while measures are taken to avoid increasing ICP. Close monitoring using the GCS and pupillary response should continue, and CT scan is repeated, especially if there is an unexplained ICP rise.

■ Increased ICP

Patients with TBI who require airway support or mechanical ventilation undergo rapid sequence oral intubation (using paralysis) rather than nasotracheal intubation, which is done while the patient breathes spontaneously and cause more coughing and gagging, thus increasing ICP to a greater extent. Drugs should be used to minimize the increase in ICP that may occur when manipulating the airway—eg, some clinicians recommend giving lidocaine 1.5 mg/kg intravenously (IV) 1 to 2 minutes before giving the paralytic. Etomidate is a good choice for an induction agent because it has minimal effects on blood pressure; IV dose in adults is 0.3 mg/kg (or 20 mg for an average-sized adult) and in children is 0.2 to 0.3 mg/kg. Alternatively, if hypotension is absent and unlikely and if propofol is more readily available, intubation can be done using 0.2 to 1.5 mg/kg. Succinylcholine 1.5 mg/kg IV is typically used as a paralytic.

Pulse oximetry and arterial blood gases (if possible, end-tidal CO_2) should be used to assess adequacy of oxygenation and ventilation. The goal is a normal $Paco_2$ level (38 to 42 mm Hg). Previously, prophylactic hyperventilation ($Paco_2$ 25 to 35 mm Hg) was recommended. However, although this lower $Paco_2$ reduces ICP by causing cerebral vasoconstriction, it also decreases cerebral perfusion and thus may produce ischemia.[65,66] Therefore, hyperventilation is used only within the first several hours to treat increased ICP unresponsive to other measures, only to achieve $Paco_2$ of 30 to 35 mm Hg, and only for short periods.

In patients with severe TBI who by definition cannot follow simple commands, especially those with an abnormal head CT scan, ICP and CPP monitoring and control are recommended. The goal is to maintain ICP at <20 mm Hg and CPP at 60 mm Hg. Cerebral venous drainage can be enhanced (thus lowering ICP) by elevating the head of the bed to 30 degrees and by keeping the patient's head in a midline position. If a ventricular catheter is in place, CSF drainage can lower ICP.

Preventing agitation, excessive muscular activity (eg, from delirium), and pain can also help prevent increases in ICP. For sedation, propofol is often used in adults (contraindicated in children) because it has quick onset and duration of action; dose is 0.3 mg/kg/h continuous IV infusion, titrated gradually upward as needed (up to 3 mg/kg/h). An initial bolus is not used. The most common adverse effect is hypotension and, albeit uncommon, has been associated with the development of pancreatitis.[67] Benzodiazepines (eg, midazolam, lorazepam) can also be used for sedation. Antipsychotics can delay recovery and should be avoided if possible. For delirium, haloperidol may be used for a few days; delirium that lasts longer can be treated with trazodone, gabapentin, valproate preparations, or quetiapine, although it is not clear that these drugs are better than haloperidol. Rarely, paralytics may be needed, if so, adequate sedation must be ensured. Opioids are often needed for adequate pain control.

Patients should be kept euvolemic and normosmolar or slightly hyperosmolar (target serum osmolality 295 to 320 mOsm/kg). Osmotic diuretics (eg, mannitol) may be given IV to lower ICP and maintain serum osmolality. However, they should be reserved for patients whose condition is deteriorating or used preoperatively for patients with hematomas. Mannitol 20% solution is given 0.5 to 1 g/kg IV (2.5 to 5 mL/kg) over 15 to 30 minutes and repeated in a dose ranging from 0.25 to 0.5 g/kg (1.25 to 2.5 mL/kg) given as often as needed (usually q 6 to 8 h); it lowers ICP for a few hours. Mannitol must be used cautiously in patients with severe coronary artery disease, heart failure, renal insufficiency, or pulmonary vascular congestion because mannitol rapidly expands intravascular volume. Because osmotic diuretics increase renal excretion of water relative to sodium, prolonged use of mannitol may also result in water depletion and hypernatremia.

Furosemide 1 mg/kg IV is also helpful to decrease total body water, particularly when the transient hypervolemia associated with mannitol is to be avoided. Fluid and electrolyte balance should be monitored closely while osmotic diuretics are used. A hypertonic saline solution (usually 2% to 3%) is being studied as another potential osmotic agent to control ICP.[68,69]

Hyperventilation (eg, to a $PaCO_2$ of 30 to 35 mm Hg) may be necessary briefly when increased ICP is refractory to other modulating strategies. An alternative treatment for intractable increased ICP is decompressive craniotomy. For this procedure, a bone flap is removed (to be replaced later), and duraplasty is performed to allow outward brain swelling.

A more involved and currently less popular option for intractable increased ICP is pentobarbital coma.[70] Coma is induced by giving pentobarbital 10 mg/kg over 30 minutes, 5 mg/kg/h for 3 doses, then 1 mg/kg/h. The dose may be adjusted to suppress bursts of electroencephalography activity, which is continuously monitored. Hypotension is common and managed by giving fluids and, if necessary, vasopressors.[70]

Therapeutic systemic hypothermia has not proved helpful.[71,72] Corticosteroids are not useful to control ICP and are not recommended.[69]

■ Seizures

Prolonged seizures, which can worsen brain damage and increase ICP, should be prevented if possible and treated promptly when they occur.[25,73] In patients with significant structural injury (eg, larger contusions or hematomas, brain laceration, depressed skull fracture) or a GCS score <10, a prophylactic anticonvulsant should be considered. If phenytoin is used, a loading dose of 20 mg/kg IV is given (at a maximum rate of 50 mg/min to prevent cardiovascular adverse effects such as hypotension and bradycardia). The starting maintenance IV dose for adults is 2 to 2.7 mg/kg t.i.d.; children require higher doses (up to 5 mg/kg b.i.d. for children <4 y). Serum levels should be measured to adjust the dose. Duration of treatment varies and depends on the type of injury and electroencephalography results. If no seizures develop within 1 week, anticonvulsants should be stopped because their value in preventing future seizures is not established.[74] Newer anticonvulsants, such as fosphenytoin and leviteracitam (Keppra), can also be used.

■ Skull Fractures

Aligned closed fractures require no specific treatment. Depressed fractures sometimes require surgery to elevate fragments, manage

lacerated cortical vessels, repair dura mater, and debride injured brain. Open fractures require debridement. Use of antibiotic prophylaxis is controversial because of limited data on its efficacy and the concern that it encourages drug-resistant strains.

■ Surgery

Intracranial hematomas may require urgent surgical evacuation to prevent or treat brain shift, compression, and herniation; hence, early neurosurgical consultation is mandatory. However, all hematomas do not necessarily require surgical removal. Small intracerebral hematomas rarely require surgery. Patients with small subdural hematomas can often be treated without surgery. Factors that suggest a need for surgery include a midline brain shift of >5 mm, compression of the basal cisterns, and worsening neurologic examination findings.[43] Chronic subdural hematomas may require surgical drainage but much less urgently than acute subdural hematomas. Large or arterial epidural hematomas are treated surgically, but small venous epidural hematomas can be followed with serial CT scans.

■ Rehabilitation

When neurologic deficits persist, rehabilitation is needed. Rehabilitation is best provided through a team approach that combines physical, occupational, and speech therapy; skill-building activities; and counseling to meet the patient's social and emotional needs. Brain injury support groups may provide assistance to the families of brain-injured patients.

For patients whose coma exceeds 24 hours, 50% of whom have major persistent neurologic sequelae, a prolonged period of rehabilitation, particularly in cognitive and emotional areas, is often required, and rehabilitation services should be planned early.

■ References

1. Rutland-Brown W, Langlois JA, Thomas KE, et al. Incidence of traumatic brain injury in the United States, 2003. *J Head Trauma Rehabil.* 2006;21:544–548.
2. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil.* 2006;21:375–378.
3. Adekoya N, Thurman DJ, White DD, et al. Surveillance for traumatic brain injury deaths—United States, 1989-1998. *MMWR Surveill Summ.* 2002;51:1–14.
4. Ropper AH, Gorson KC. Clinical practice concussion. *N Engl J Med.* 2007;356:166–172.
5. Ryan LM, Warden DL. Post concussion syndrome. *Int Rev Psychiatry.* 2003;15: 310–316.
6. de Kruijk JR, Leffers P, Meerhoff S, et al. Effectiveness of bed rest after mild traumatic brain injury: a randomised trial of no versus six days of bed rest. *J Neurol Neurosurg Psychiatry.* 2002;73:167–172.

7. Adams JH, Doyle D, Graham DI, et al. Microscopic diffuse axonal injury in cases of head injury. *Med Sci Law*. 1985;25:265–269.
8. Gentry LR, Godersky JC, Thompson B. MR imaging of head trauma: review of the distribution and radiopathologic features of traumatic lesions. *AJR Am J Roentgenol*. 1988;150:663–672.
9. Gentry LR, Godersky JC, Thompson B, et al. Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. *AJR Am J Roentgenol*. 1988;150:673–682.
10. Adams JH, Doyle D, Graham DI, et al. The contusion index: a reappraisal in human and experimental non-missile head injury. *Neuropathol Appl Neurobiol*. 1985;11: 299–308.
11. Reilly PL, Graham DI, Adams JH, et al. Patients with head injury who talk and die. *Lancet*. 1975;2:375–377.
12. Rockswold GL, Leonard PR, Nagib MG. Analysis of management in thirty-three closed head injury patients who "talked and deteriorated". *Neurosurgery*. 1987;21: 51–55.
13. Klauber MR, Marshall LF, Luerssen TG, et al. Determinants of head injury mortality: importance of the low risk patient. *Neurosurgery*. 1989;24:31–36.
14. Rust T, Kiemer N, Erasmus A. Chronic subdural haematomas and anticoagulation or anti-thrombotic therapy. *J Clin Neurosci*. 2006;13:823–827.
15. Itshayek E, Rosenthal G, Fraifeld S, et al. Delayed posttraumatic acute subdural hematoma in elderly patients on anticoagulation. *Neurosurgery*. 2006;58:E851–E856. Discussion E851–E856.
16. Maloney AF, Whatmore WJ. Clinical and pathological observations in fatal head injuries. A 5-year survey of 173 cases. *Br J Surg*. 1969;56:23–31.
17. Lindenberg R. Trauma of meninges and brain. In: Minckler J, ed. *Pathology of the Nervous System*. New York: McGraw-Hill; 1971;1705–1765.
18. Bullock MR, Chesnut R, Ghajar J, et al, and Surgical Management of Traumatic Brain Injury Author Group. Surgical management of acute epidural hematomas. *Neurosurgery*. 2006;58:S7–S15. Discussion Si–Siv.
19. Teasdale GM, Murray G, Anderson E, et al. Risks of acute traumatic intracranial haematoma in children and adults: implications for managing head injuries. *BMJ*. 1990;300:363–367.
20. Miller JD. Minor, moderate and severe head injury. *Neurosurg Rev*. 1986;9:135–139.
21. Wysocki J. Cadaveric dissections based on observations of injuries to the temporal bone structures following head trauma. *Skull Base*. 2005;15:99–106. Discussion 106–107.
22. Nortje J, Menon DK. Traumatic brain injury: physiology, mechanisms, and outcome. *Curr Opin Neurol*. 2004;17:711–718.
23. DeWitt DS, Prough DS. Traumatic cerebral vascular injury: the effects of concussive brain injury on the cerebral vasculature. *J Neurotrauma*. 2003;20:795–825.
24. Joshi J, Ornstein E, Young WL. Cerebral and spinal cord blood flow. In: Cottrell JE, Smith DC, eds. *Anesthesia and Neurosurgery*. 4th ed. St Louis: Mosby Inc; 2001:19.
25. Graham DI, Lawrence AE, Adams JH, et al. Brain damage in non-missile head injury secondary to high intracranial pressure. *Neuropathol Appl Neurobiol*. 1987;13: 209–217.
26. Armstrong BD, Hu Z, Abad C, et al. Lymphocyte regulation of neuropeptide gene expression after neuronal injury. *J Neurosci Res*. 2003;74:240–247.
27. Nimmo AJ, Cernak I, Heath DL, et al. Neurogenic inflammation is associated with development of edema and functional deficits following traumatic brain injury in rats. *Neuropeptides*. 2004;38:40–47.
28. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974;2:81–84.

29. Jane JA, Rimel RW. Prognosis in head injury. *Clin Neurosurg.* 1982;29:346–352.
30. Rimel RW, Giordani B, Barth JT, et al. Disability caused by minor head injury. *Neurosurgery.* 1981;9:221–228.
31. Rimel RW, Giordani B, Barth JT, et al. Moderate head injury: completing the clinical spectrum of brain trauma. *Neurosurgery.* 1982;11:344–351.
32. Kelly DF, Doberstein C, Becker DP. General principles of head injury management. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. *Neurotrauma*. New York: McGraw-Hill; 1996:81.
33. Lobato RD, Rivas JJ, Gomez PA, et al. Head-injured patients who talk and deteriorate into coma. Analysis of 211 cases studied with computerized tomography. *J Neurosurg.* 1991;75:256–261.
34. Miller JD, Murray LS, Teasdale GM. Development of a traumatic intracranial hematoma after a "minor" head injury. *Neurosurgery.* 1990;27:669–673. Discussion 673.
35. Cushing H. Concerning a definite regulatory mechanism of the vaso-motor centre which controls blood pressure during cerebral compression. *Johns Hopkins Hosp Bull.* 1901;12:290–292.
36. Fodstad H, Kelly PJ, Buchfelder M. History of the Cushing reflex. *Neurosurgery.* 2006;59:1132–1137. Discussion 1137.
37. Willer B, Leddy JJ. Management of concussion and post-concussion syndrome. *Curr Treat Options Neurol.* 2006;8:415–426.
38. De Kruijk JR, Leffers P, Menheere PP, et al. Prediction of post-traumatic complaints after mild traumatic brain injury: early symptoms and biochemical markers. *J Neurol Neurosurg Psychiatry.* 2002;73:727–732.
39. de Kruijk JR, Leffers P, Menheere PP, et al. Olfactory function after mild traumatic brain injury. *Brain Injury.* 2003;17:73–78.
40. Levin AV. Ophthalmology of shaken baby syndrome. *Neurosurg Clin N Am.* 2002;13:201–211, vi.
41. Morad Y, Kim YM, Armstrong DC, et al. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. *Am J Ophthalmol.* 2002;134:354–359.
42. Diaz-Marchan PJ, Hayman LA, Carrier DA, et al. Computed tomography of closed head injury. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. *Neurotrauma*. New York: McGraw-Hill; 1996:140–141.
43. Narayan RK, Kishore PR, Becker DP, et al. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury. *J Neurosurg.* 1982;56:650–659.
44. Gerber DJ, Weintraub AH, Cusick CP, et al. Magnetic resonance imaging of traumatic brain injury: relationship of T2*SE and T2GE to clinical severity and outcome. *Brain Injury.* 2004;18:1083–1097.
45. Mannion RJ, Cross J, Bradley P, et al. Mechanism-based MRI classification of traumatic brainstem injury and its relationship to outcome. *J Neurotrauma*. 2007; 24:128–135.
46. Wedekind C, Hesselmann V, Klug N. Comparison of MRI and electrophysiological studies for detecting brainstem lesions in traumatic brain injury. *Muscle Nerve.* 2002;26:270–273.
47. Hyam JA, Welch CA, Harrison DA, et al. Case mix, outcomes and comparison of risk prediction models for admissions to adult, general and specialist critical care units for head injury: a secondary analysis of the ICNARC case mix programme database. *Crit Care.* 2006;10(suppl 2):S2.
48. Alberico AM, Ward JD, Choi SC, et al. Outcome after severe head injury. Relationship to mass lesions, diffuse injury, and ICP course in pediatric and adult patients. *J Neurosurg.* 1987;67:648–656.

49. Teasdale G, Jennett B. Assessment and prognosis of coma after head injury. *Acta Neurochir (Wien)*. 1976;34:45–55.
50. Jennett B, Snoek J, Bond MR, et al. Disability after severe head injury: observations on the use of the Glasgow outcome scale. *J Neurol Neurosurg Psychiatry*. 1981;44: 285–293.
51. Blyth B. The outcome of severe head injuries. *N Z Med J*. 1981;93:267–269.
52. Gensemer IB, McMurry FG, Walker JC, et al. Behavioral consequences of trauma. *J Trauma*. 1988;28:44–49.
53. Gensemer IB, Smith JL, Walker JC, et al. Psychological consequences of blunt head trauma and relation to other indices of severity of injury. *Ann Emerg Med*. 1989; 18:9–12.
54. Miller JD, Becker DP. Secondary insults to the injured brain. *J R Coll Surg Edinb*. 1982;27:292–298.
55. Marshall LF, Becker DP, Bowers SA, et al. The national traumatic coma data bank. part 1: design, purpose, goals, and results. *J Neurosurg*. 1983;59:276–284.
56. Maciver IN, Frew IJ, Matheson JG. The role of respiratory insufficiency in the mortality of severe head injuries. *Lancet*. 1958;1:390–393.
57. Pigula FA, Wald SL, Shackford SR, et al. The effect of hypotension and hypoxia on children with severe head injuries. *J Pediatr Surg*. 1993;28:310–314. Discussion 315–316.
58. Wald SL, Shackford SR, Fenwick J. The effect of secondary insults on mortality and long-term disability after severe head injury in a rural region without a trauma system. *J Trauma*. 1993;34:377–381. Discussion 381–382.
59. Simma B, Burger R, Falk M, et al. A prospective, randomized, and controlled study of fluid management in children with severe head injury: lactated ringer's solution versus hypertonic saline. *Crit Care Med*. 1998;26:1265–1270.
60. Zornow MH, Todd MM, Moore SS. The acute cerebral effects of changes in plasma osmolality and oncotic pressure. *Anesthesiology*. 1987;67:936–941.
61. Mcilvoy LH. The effect of hypothermia and hyperthermia on acute brain injury. *AACN Clin Issues*. 2005;16:488–500.
62. Lam AM, Winn HR, Cullen BF, et al. Hyperglycemia and neurological outcome in patients with head injury. *J Neurosurg*. 1991;75:545–551.
63. Valadka AB, Narayan RK. Emergency room management of the head-injured patient. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. *Neurotrauma*. New York: McGraw-Hill; 1996:122.
64. Valadka AB, Narayan RK. Emergency room management of the head-injured patient. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. *Neurotrauma*. New York: McGraw-Hill; 1996:122–124.
65. Barbaccia JJ, Williams JM. The acute management of head injury. *Curr Opin Anaesthesiol*. 2001;14:227–231.
66. Muizelaar JP, Marmarou A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg*. 1991;75:731–739.
67. Gottschling S, Larsen R, Meyer S, et al. Acute pancreatitis induced by short-term propofol administration. *Paediatr Anaesth*. 2005;15:1006–1008.
68. Mauritz W, Janciak I, Wilbacher I, et al. Severe traumatic brain injury in Austria IV: intensive care management. *Wien Klin Wochenschr*. 2007;119:46–55.
69. Rangel-Castillo L, Robertson CS. Management of intracranial hypertension. *Crit Care Clin*. 2006;22:713–732. Abstract ix.
70. Roberts I. Barbiturates for acute traumatic brain injury. *Cochrane Database Syst Rev*. 1999;3:CD000033.
71. Clifton GL. Is keeping cool still hot? an update on hypothermia in brain injury. *Curr Opin Crit Care*. 2004;10:116–119.

72. Robertson CL, Clark RS, Dixon CE, et al. No long-term benefit from hypothermia after severe traumatic brain injury with secondary insult in rats. *Crit Care Med*. 2000;28:3218–3223.
73. Lassen NA. Control of cerebral circulation in health and disease. *Circ Res*. 1974;34:749–760.
74. Temkin NR, Haglund M, Winn HR. Post-traumatic seizures. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. *Neurotrauma*. New York: McGraw-Hill; 1996: 611–619.