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Evaluation and Management of Traumatic Brain Injury

Scott R. Shepard

Objectives

1. **To describe the physiology of intracranial pressure (ICP) and cerebral perfusion pressure (CPP).**
2. **To understand the effects of blood pressure, ventilatory status, and fluid balance on ICP and CPP.**
3. **To describe the evaluation and management of different types of head injury.**

Case

A 22-year-old man is brought to the emergency room following a high-speed motorcycle accident. The paramedics report that the patient struck a tree and that there was a 5-minute loss of consciousness. On arrival, the patient has the following vital signs: respiratory rate, 12; blood pressure, 150/75; heart rate, 92. He opens his eyes to painful stimuli, follows simple commands, and answers questions with inappropriate words.

Introduction

Traumatic brain injury (TBI) continues to be a major public health problem despite the technologic revolution in medicine. The annual incidence of TBI has been estimated to be approximately 200 cases per 100,000 persons in the United States (550,000 annually in the U.S.). The majority of head injuries (80%) are mild head injuries, with the remainder divided equally between moderate and severe head injuries. The majority of those with severe TBI and many of those with moderate TBI are disabled permanently, resulting in an annual expenditure in the U.S. of $25 billion to cover the care of these individuals.

Table 32.1. Glasgow Coma Scale (GCS).

Eye opening (E)	Motor response (M)	Verbal response (V)
Spontaneous (4)	Follows commands (6)	Oriented (5)
To voice (3)	Localizes (5)	Confused (4)
To painful stimuli (2)	Withdraws (4)	Inappropriate words (3)
None (1)	Abnormal flexion (3)	Moaning (2)
	Extension (2)	None (1)
	None (1)	
GCS = E + M + V		

Source: Reprinted from Gustilo RB, Anderson JT. Prevention of infection in the treatment of 1025 open fractures of long bones. J Bone Joint Surg 1976; 58(A), 453, with permission.

Initial Evaluation

Initial Systemic Trauma Evaluation, Focused Head Injury Evaluation, and Neurologic Assessment

The primary evaluation of the TBI patient involves a thorough **systemic trauma evaluation** according to the Advanced Trauma Life Support (ATLS) guidelines. After completion of the initial trauma evaluation and if the patient is hemodynamically stable, a **focused head injury evaluation** should be initiated. It is important to attempt to obtain a **thorough history** of the mechanism of the trauma as well as of the events immediately preceding the trauma, because specific information, such as the occurrence of syncope prior to the accident, necessitates an evaluation for the etiology of such an event.

After a sufficient history has been obtained, the **neurologic assessment** begins. The first part of the neurologic evaluation is the **Glasgow Coma Scale (GCS) (Table 32.1). Although the GCS is part of the initial trauma evaluation, it should be repeated periodically to assess for neurologic deterioration.** The GCS was developed by Teasdale and Jennett[1] and is used to describe the general level of consciousness of TBI patients as well as to define the severity of head injuries. It is important to remember that the GCS is a screening exam and does not substitute for a thorough neurologic exam. The GCS is divided into three categories: **eye opening (E)**, **motor response (M)**, and **verbal response (V)**. The score is determined by the sum of the score in each of the three categories, with a maximum score of 15 and a minimum score of 3. Since patients who are intubated cannot be assessed for a verbal response, they are evaluated only with eye opening and motor scores, and the suffix "T" is added to their GCS to indicate that they are intubated. Mild head injuries are defined as TBI patients with a GCS of 13 to 15, and moderate head injuries are defined as those with a GCS of 9 to 12. A GCS of 8 or less defines a severe head injury. These definitions are not rigid and should be considered as a general guide to the level of injury. In the case presented above, the patient's eye opens to stimuli (E = 2), and the patient follows simple commands (M = 6) and uses inappropriate words (V = 3), resulting in a GCS of 11 (2 + 6 + 3).

[1] Teasdale G, Jennett B. Assessment of coma and impaired consciousnes. A practical scale. Lancet 1974;2:81–84.

The neurologic assessment of the TBI patient should include a complete brainstem exam (pupillary exam, ocular movement exam, corneal reflex, gag reflex), a motor exam, and a sensory exam. Many TBI patients have significant alterations of consciousness and/or pharmaceuticals present that will limit the neurologic exam.

Brainstem Exam

Pupillary Exam: A careful **pupillary exam** is an essential part of the evaluation of the TBI patient, especially in patients with severe injuries. When muscle relaxants have been administered to a patient, only the pupillary exam is available for evaluation. Several factors can alter the pupillary exam. Narcotics cause pupillary constriction, and medications or drugs that have sympathomimetic properties cause pupillary dilation. These effects often are strong enough to blunt or nearly eliminate pupillary responses. Prior eye surgery, such as cataract surgery, also can alter or eliminate pupillary reactivity.

A normal pupillary exam consists of bilaterally reactive pupils that react to both direct and consensual stimuli. Bilateral, small pupils may be caused by narcotics or pontine injury (disruption of sympathetic centers in the pons). Bilateral fixed and dilated pupils are secondary to diffuse cerebral hypoxia, which may result from either severe elevations of ICP, preventing adequate blood flow to the brain, or diffuse systemic hypoxia. A unilateral fixed (unresponsive) and dilated pupil has two potential causes. If the pupil does not constrict when light is directed at the pupil but constricts when light is directed into the contralateral pupil (intact consensual response), this usually is the result of a traumatic optic nerve injury. If a unilateral dilated pupil does not respond to either direct or consensual stimulation, this usually is a sign of transtentorial herniation. Unilateral pupillary constriction usually is secondary to Horner's syndrome, in which the sympathetic input to the eye is disrupted. Horner's syndrome may be caused by a disruption of the sympathetic system, either at the apex of the lung or adjacent to the carotid artery.

Ocular Movement Exam: When there is a significant alteration in the level of consciousness, there often is a loss of voluntary eye movement, and abnormalities in ocular movements may occur. Ocular movements involve the coordination of multiple centers within the brain, including the frontal eye fields, the parapontine reticular formation (PPRF), the medial longitudinal fasciculus (MLF), and the III and VI cranial nerve nuclei. When voluntary eye movements cannot be assessed, **oculocephalic and oculovestibular testing** may be performed.

Oculocephalic testing (doll's eyes) assesses the integrity of the horizontal gaze centers and involves observation of eye movements when the head is rotated rapidly from side to side. This maneuver is contraindicated in any patient with a known or suspected cervical spine injury. Oculocephalic testing is performed by elevating the head 30 degrees and briskly rotating it from side to side. A normal response is for the eyes to rotate away from the direction of the movement as if

they are fixating on a target that is straight ahead, similar to the way a doll's eyes move when its head is turned. If the eyes remain fixed in position and do not rotate, this is indicative of dysfunction in the lateral gaze centers and is referred to as negative doll's eyes.

Oculovestibular testing (cold water calorics) is another method for the assessment of the integrity of the gaze centers. Oculovestibular testing is performed with the head elevated to 30 degrees and requires the presence of an intact tympanic membrane. In oculovestibular testing, ice-cold water slowly is instilled into the external auditory canal. This causes an imbalance in the vestibular signals and initiates a compensatory response. Cold water irrigation in the ear of an alert patient results in a fast nystagmus away from the irrigated ear and a slow, compensatory nystagmus toward the irrigated side. If warm water is used, the opposite will occur. This is the basis for the acronym **COWS** (cold—opposite, warm—same) and refers to the direction of the fast component of nystagmus. As the level of consciousness declines, the fast component of nystagmus gradually fades, and, in the unconscious patient, only the slow phase of nystagmus is present. If there is a normal oculocephalic response to cold water calorics (eye deviation toward the side of irrigation), this indicates that the injury is rostral to the upper brainstem and that the PPRF, the MLF, and third and sixth cranial nerve nuclei are intact.

Corneal Reflex and Gag Reflex: The **corneal reflex** is assessed by gently stroking the cornea with a soft wisp of cotton. The normal response is a single blink on the side of stimulation. The corneal reflex is mediated by the fifth and seventh cranial nerves, and intact corneal reflexes indicate integrity of the pons. The **gag reflex**, in which gentle stimulation of the posterior oropharynx results in elevation of the soft palate, assesses the integrity of the lower brainstem (medulla).

Motor Exam and Sensory Exam

After the brainstem exam has been completed, a **motor exam and a sensory exam** should be performed. A thorough motor or sensory exam is difficult to perform in any patient with an altered level of consciousness. When a patient is not alert enough to cooperate with strength testing, the motor exam is limited to an assessment for asymmetry. This may be demonstrated by an asymmetric response to central pain stimulation or a difference in muscle tone between the left and right side. If there is asymmetry in the motor exam, this may be indicative of a hemispheric injury and may raise the suspicion for a mass lesion. It often is difficult to perform a useful sensory exam in the TBI patient. Patients with altered levels of consciousness are unable to cooperate with sensory testing, and a sensory exam may not be reliable in intoxicated or comatose patients.

Diagnostic Evaluation

After the patient has been stabilized and an appropriate neurologic exam has been performed, the diagnostic evaluation may begin.

Radiographic Evaluation

X-Rays

Skull x-rays rarely are used today in the evaluation of closed head injury. They occasionally are used in the evaluation of penetrating head trauma and can provide a rapid assessment of the degree of foreign-body penetration in nonmissile penetrating head injuries (e.g., stab wounds).

Computed Tomography

Computed tomography (CT) scan is the diagnostic study of choice in the evaluation of TBI because it has a rapid acquisition time, it is universally available, and it accurately demonstrates acute hemorrhage.

The subject in the case presentation would undergo a head CT scanning during his evaluation. The standard CT scan for the evaluation of acute head injury is a noncontrast scan with three data sets: bone windows, tissue windows, and subdural windows. The bone windows provide a survey of bony anatomy, and the tissue windows allow for a detailed survey of the brain and its contents. The subdural windows provide better visualization of intracranial hemorrhage. **Table 32.2**

Table 32.2. Checklist for interpreting a trauma head computed tomography (CT) scan: features to examine.

Soft tissue windows: start inferiorly and work up to the vertex

1. **Fourth ventricle: is it shifted? compressed? blood in it?**
2. **Cerebellum: bleed or infarct?**
3. **Brainstem cisterns obliterated? check the quadrigeminal and ambient cisterns**
4. **Check lateral ventricles for blood (especially in occipital horns), size (especially temporal horns), and mass effect**
5. **Extraaxial blood: EDH is lens-shaped, does not cross sutures; SDH crosses sutures; SAH channels into sulci and fissures; measure maximum thickness of clot in millimeters; check circle of Willis, sylvian fissure for SAH**
6. **Look for intraparenchymal hematomas and contusions, especially frontal and temporal tips, inferior frontal lobes, and under any fractures (measure clot thickness in mm)**
7. **Measure midline shift in millimeters at level of septum pellucidum**
8. **Check top cuts for effacement of sulci, often a subtle sign of mass effect**

Bone windows

1. **Check five sets of sinuses (ethmoid, sphenoid, frontal, mastoid, maxillary) for fracture or opacification; the maxillary sinuses may only be partially seen on standard head cuts**
2. **Look for fracture of orbital apex (? CN II compression), petrous temporal bone (? basilar skull fracture), or convexities (if depressed, is it more than a table's width? measure the depression in mm)**
3. **Check for intracranial or intraorbital air; this is much easier to see on bone windows than soft tissue windows**

CN, cranial nerve; EDH, extradural hematoma; SAH, subarachnoid hemorrhage; SDH, subdural hematoma.

Source: Reprinted from Starr P. Neurosurgery. In: Norton JA, Bollinger RR, Chang AE, et al, eds. Surgery: Basic Science and Clinical Evidence. New York: Springer-Verlag, 2001, with permission.

Table 32.3. CT classification of head injury.

Injury level	Midline shift	Basal CSF cisterns	Hematoma or contusions
I	None	Cisterns widely patent	Minimal, if any
II	Not present or shift	Cisterns widely patent	No lesion with volume >25 mL
III	Shift < 5 mm	Partial compression or absent	No lesion with volume >25 mL
IV	Shift > 5 mm	Partial compression or absent	No lesion with volume >25 mL

CSF, cerebrospinal fluid.

provides a suggested checklist for the evaluation of the head CT in a TBI patient.

It is important to use a systemic approach when reviewing a CT scan and to follow the same protocol each time. Consistency is much more important than the specific order used. First, the bone windows should be examined for fractures, beginning with the cranial vault itself, and then the skull base and the facial bones should be examined. Next, the tissue windows should be examined for the presence of any of the following: extraaxial hematomas (e.g., epidural or subdural hematomas), intraparenchymal hematomas, or contusions. Next, the brain should be surveyed for any evidence of pneumocephalus, hydrocephalus, cerebral edema, midline shift, or compression of the subarachnoid cisterns at the base of the brain. Finally, the subdural windows should be examined for any hemorrhage that may not be visualized easily on the tissue windows.

Computed tomography scans may be used for classification as well as for diagnostic purposes. There is a classification scheme published by Marshall et al[2] that classifies head injuries according to the changes demonstrated by CT scan. This system defines four categories of injury, from diffuse injury I to diffuse injury IV (**Table 32.3**). These levels of injury are based on the presence of three different abnormalities—midline shift, patency of cerebrospinal fluid (CSF) cisterns at the base of the brain, and presence of a contusion or a hematoma—seen on the CT scan.

Skull Fractures: Skull fractures are classified as either nondisplaced (linear) fractures or comminuted fractures. Linear skull fractures sometimes are difficult to visualize on the individual axial images of a CT scan. The scout film of the CT scan, which is the equivalent of a lateral skull x-ray, often demonstates linear fractures, which may be difficult to appreciate on the axial views of a CT scan. Comminuted fractures are complex fractures with multiple components. A comminuted fracture may be displaced inward, which is defined as a depressed skull fracture.

Intracranial Hemorrhages: Intracranial hemorrhages are divided into two broad categories: **extraaxial hematomas** and **intraaxial hematomas (Table 32.4).** On CT, acute hemorrhage is hyperintense when compared with the brain and usually appears as a bright white signal.

[2] Marshall LF, Marshall SB, Klauber MR. A new classification of head injury based on computerized tomography. J Neurosurg 1991;75:S14–S20.

Table 32.4. Intracranial hemorrhages.

Intraaxial hematomas	Extraaxial hematomas
Intracerebral hematoma	Epidural hematoma
Subarachnoid hemorrhage	Subdural hematoma
Cerebral contusion	

Extraaxial hematomas include **epidural and subdural hematomas**. **Epidural hematomas** are located between the inner table of the skull and the dura. They typically are biconvex in shape because their outer border follows the inner table of the skull, and their inner border is limited by locations where the dura is firmly adherent to the skull (**Fig. 32.1**). Epidural hematomas usually are caused by injury to a dural-based artery, although 10% of epidurals may be venous in origin. Epidural hematomas, especially those of arterial origin, may enlarge rapidly. **Subdural hematomas** are located between the dura and the brain. Their outer edge is convex, while their inner border usually is irregularly concave (**Fig. 32.2**). Subdural hematomas are not limited by the intracranial suture lines, and this is an important feature that aids in their differentiation from epidural hematomas. Subdural hematomas usually are venous in origin, although some are due to arterial bleeding.

Intraaxial hematomas are defined as hemorrhages within the brain parenchyma. These hematomas include **intraparenchymal**

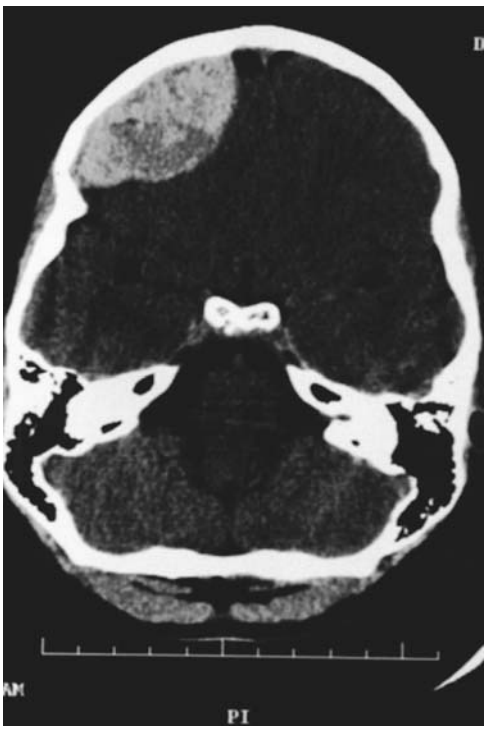


Figure 32.1. Computed tomography (CT) of epidural hematoma.

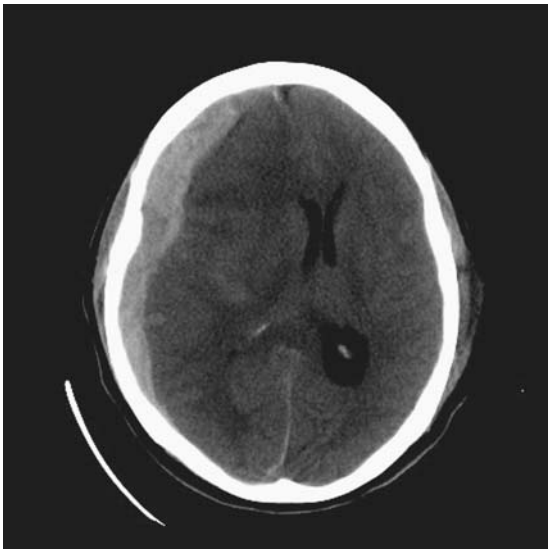


Figure 32.2. CT of subdural hematoma.

hematomas, cerebral contusions intraventricular hemorrhages, and subarachnoid hemorrhages. **Intraparenchymal hemorrhages** are homogeneous regions of hyperintense signal on CT (**Fig. 32.3**). **Cerebral contusions** are posttraumatic lesions in the brain that appear as irregular, heterogeneous regions in which hyperintense changes (blood) and low-density changes (edema) are intermixed (**Fig. 32.4**).

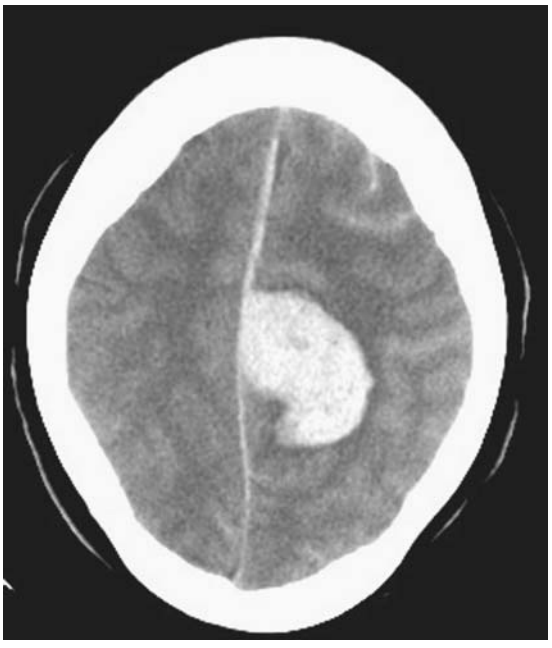


Figure 32.3. CT of intraparenchymal hematoma.

Intraventricular hemorrhages are regions of high intensity within the ventricular system. **Subarachnoid hemorrhages** that occur as a result of trauma typically are located over gyri on the convexity of the brain. These are thin layers of high-intensity signal located on the surface of the cortex. They are distinct from the subarachnoid hemorrhages that occur as the result of a ruptured cerebral aneurysm, which usually are located in the arachnoid cisterns at the base of the brain.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) has a limited role in the evaluation of acute head injury. Although MRI provides extraordinary anatomic detail, it commonly is not used to evaluate acute TBI. This is due to its long acquisition time and the difficulty of using it in the critically ill. **It may be used to evaluate patients with unexplained neurologic deficits.** It is superior to CT for identifying diffuse axonal injury (DAI) and small strokes. Diffuse axonal injury is defined as neuronal injury in the subcortical gray matter or the brainstem as a result of severe rotation or deceleration. It often is the explanation for a depressed level of consciousness in a patient with normal intracranial pressure and without evidence of significant injury on CT scan. Magnetic resonance angiography may be used in some TBI patients to assess for vascular injury.

Angiography

Prior to the development of CT, **cerebral angiography** was used to demonstrate the presence of an intracranial mass lesion. **Currently, angiography is used in acute head injury only when there is the suspicion of a vascular injury.** This includes patients with evidence of a

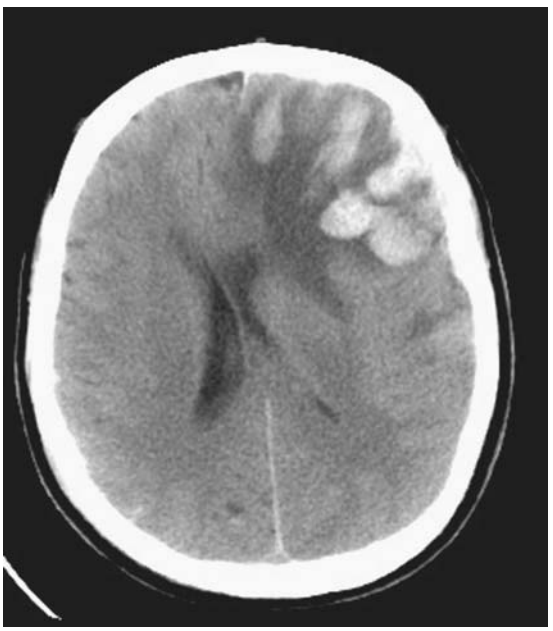


Figure 32.4. CT of cerebral contusion.

Table 32.5. Relative volume of intracranial components.

Brain	**85% to 90%**
Intravascular blood	**8% to 10%**
CSF	**2% to 3%**

potential carotid injury (hemiparesis without a significant hematoma or the presence of Horner's syndrome) and patients with temporal bone fractures that traverse the carotid canal.

Pathophysiology

Intracranial Compliance

Appropriate management of TBI requires an appreciation of some of the anatomic features of the brain. The brain floats in CSF within the skull, a rigid and inelastic container. The skull cannot expand to accommodate any increases in volume of the brain, thus, only small increases in cerebral volume can be tolerated before ICP begins to rise dramatically. This concept is defined by the **Monro-Kellie doctrine**, which states that the total intracranial volume is fixed.[3] The intracranial volume (V i/c) is equal to the sum of its components: **V i/c = V (brain) + V (CSF) + V (blood)**. The brain comprises 85% to 90% of the intracranial volume, while intravascular cerebral blood volume accounts for 8% to 10% and CSF accounts for the remainder, 2% to 3% (**Table 32.5**). When cerebral edema is present, it increases the relative volume of the brain. Since the intracranial volume is fixed, unless there is some compensatory action, such as a decrease in the volume of one of the other intracranial components, the intracranial pressure will rise. This is related intimately to intracranial compliance, which is defined as the change in pressure due to changes in volume. The brain has very limited compliance and cannot tolerate significant increases in volume that can result from diffuse cerebral edema or significant mass lesions, such as a hematoma. Individual treatments for elevated ICP are designed to decrease the volume of one of the intracranial components, thereby improving compliance and decreasing ICP.

Cerebral Perfusion Pressure

A second crucial concept in TBI pathophysiology is the concept of **cerebral perfusion pressure (CPP)**, which is defined as the difference between the mean arterial pressure (MAP) and the ICP: **CPP = MAP – ICP**. In the noninjured brain, cerebral blood flow (CBF) is constant in the range of CPP between 50 and 150 mm Hg due to autoregulation by the arterioles. When the CPP is less than 50 mm Hg or greater than 150 mm Hg, the autoregulation is overcome, and blood flow becomes

[3] Chestnut RM, Marshall LF. Treatment of abnormal intracranial pressure. Neurosurg Clin North Am 1991;2(2):267–284.

entirely dependent on the CPP, a situation defined as pressure passive perfusion. In pressure passive perfusion, the CBF is no longer constant but proportional to the CPP. Thus, when the CPP falls below 50 mm HG, the brain is at risk of ischemia due to insufficient blood flow. Autoregulation also is impaired in the injured brain, and, as a result, there is pressure passive perfusion within and around injured regions of the brain.

Herniation

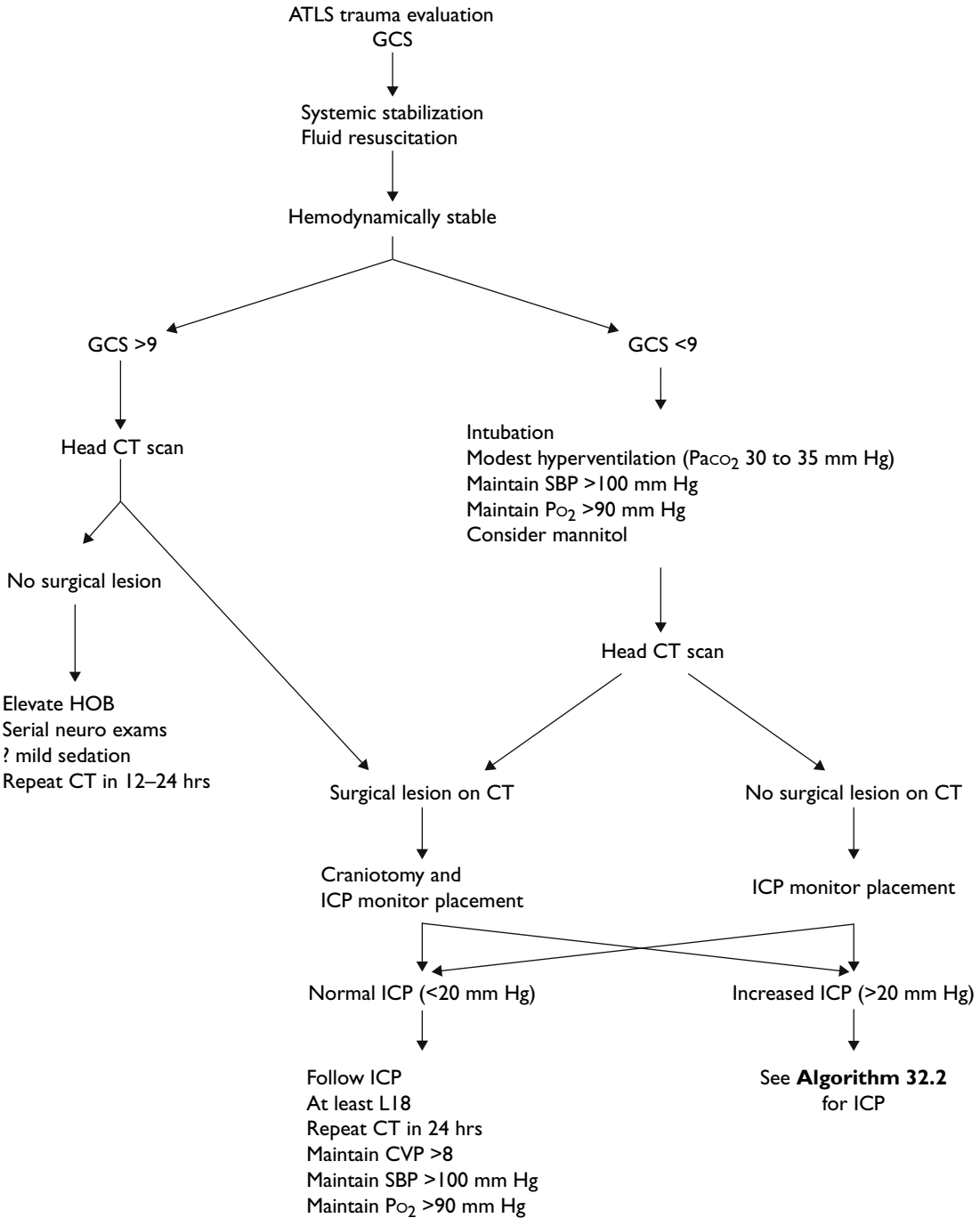
Elevated ICP is deleterious because it can decrease CPP and CBF, which may result in cerebral ischemia. Also, uncontrolled ICP may result in **herniation**, a process that involves the movement of a region of the brain across fixed dural structures, resulting in irreversible and often fatal cerebral injury. The intracranial compartment is divided into three compartments by two major dural structures, the falx cerebri and the tentorium cerebelli. When there is a significant increase in ICP or a large mass lesion is present, the brain may be displaced across the edge of the falx or the tentorium, a phenomenon known as herniation. As the brain slides over these dural edges, it compresses other regions of the brain (e.g., the brainstem) and cause neurologic injury. There are five types of herniation: **transtentorial herniation, subfalcine herniation, central herniation, cerebellar herniation**, and **tonsillar herniation**. **Transtentorial herniation** occurs when the medial aspect of the temporal lobe (uncus) migrates across the free edge of the tentorium. This compresses the third cranial nerve, interrupting parasympathetic input to the eye and resulting in a dilated pupil. This unilateral dilated pupil is the classic sign of transtentorial herniation and usually (80%) occurs ipsilateral to the side of the transtentorial herniation. The changes that occur in the other types of herniation are listed in **Table 32.6**.

Treatment

The treatment of TBI may be divided into **the treatment of closed head injury and the treatment of penetrating head injury**. While there is significant overlap in the treatment of these two types of injury, there are some important differences that are discussed later in this chapter. Closed head injury treatment is divided further into the treatment of mild and moderate/severe head injuries. See **Algorithm 32.1** for initial management of the traumatic brain injury patient.

Table 32.6. Herniation syndromes.

Herniation syndrome	Mechanism
Transtentorial herniation	Medial temporal lobe is displaced across the tentorial edge
Subfalcine herniation	Medial frontal lobe is displaced under the falx
Central (downward) herniation	Cerebral hemisphere(s) is displaced down through the tentorial incisura
Cerebellar (upward) herniation	Cerebellum is displaced up through the tentorial incisura
Tonsillar herniation	Cerebellar tonsils are displaced through the foramen magnum



Algorithm 32.1. Initial management of the traumatic brain injury patient. ATLS, Advanced Trauma Life Support; CT, computed tomography; GCS, Glasgow Coma Scale; HOB, head of bed; ICP, intracranial pressure; SBP, systolic blood pressure. Reprinted from Bullock R, Chestnut R, Clifton G, et al. Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma 1996 Nov; 13(11):641–734. Copyright © 1995, Brain Trauma Foundation. With permission of Mary Ann Leibert, Inc., Publishers.

Closed Head Injury

Mild Head Injury Treatment

The majority of head injuries are **mild head injuries**. Most people presenting with mild head injuries do not have any progression of their head injury; however, up to 3% of mild head injuries progress to more serious injuries.

Patients with mild to moderate headaches, dizziness, and nausea are considered to have a low-risk injury. Most of these patients require only observation after they have been assessed carefully, and many do not require radiographic evaluation. These patients may be discharged if there is a reliable individual to monitor them at home. After a mild head injury, those displaying persistent emesis, severe headache, anterograde amnesia, loss of consciousness, or signs of intoxication by drugs or alcohol should be evaluated with a head CT scan.

Patients with mild head injuries typically have concussions. A **concussion** is defined as physiologic injury to the brain without any evidence of structural alteration, as in the case presented. Loss of consciousness frequently occurs in concussions, but it is not part of the definition of concussion. Concussions may be graded on a scale of I to V based on criteria such as length of confusion, type of amnesia following the event, and length of loss of consciousness (**Table 32.7**).

As many as 30% of patients who experience a concussion develop a **postconcussive syndrome (PCS)**, which occurs when there is a persistence of any combination of the following after a head injury: headache, nausea, emesis, memory loss, dizziness, diplopia, blurred vision, emotional lability, and sleep disturbances. The PCS may last between 2 weeks and 6 months. Typically, the symptoms peak from 4 to 6 weeks following the injury. On occasion, the symptoms of PCS last for a year or longer, and some patients are disabled permanently by PCS.

Moderate and Severe Head Injury Treatment

The treatment of **moderate and severe head injuries** begins with initial cardiopulmonary stabilization by ATLS guidelines. **The initial resuscitation of a head-injured patient is of critical importance to prevent hypoxia and hypotension.** Analysis of the Traumatic Coma Data Bank, a database of 753 severe head injury patients, revealed that TBI patients who presented to the hospital with hypotension had twice the

Table 32.7. Classification of concussion.

Concussion Grade	Confusion or disorientation	Type of amnesia	Loss of consciousness
I	Transient	None	None
II	Brief	Anterograde	None
III	Prolonged	Retrograde	<5 minutes
IV	Prolonged	Retrograde	5 to 10 minutes
V	Prolonged	Retrograde	>10 minutes

Table 32.8. Surgical indications in nonpenetrating head trauma.

Subdural/epidural hematoma resulting in midline shift >5 mm
Intracerebral hematoma >30 cc
Temporal or cerebellar hematoma with diameter >3 cm
Open skull fracture
Skull fracture with displacement >1 cm

mortality rate of those patients who were normotensive on presentation[4]. The combination of hypoxia and hypotension resulted in a mortality rate two-and-one-half times greater than if both of these factors were absent. After **initial stabilization** and assessment of the GCS, a **neurologic exam** as described earlier should be performed.

After a thorough neurologic assessment has been performed, a **CT scan of the head** is obtained. If there is a surgical lesion present, then arrangements are made for immediate transport to the operating room. Although there are no strict guidelines for defining surgical lesions in head injury, most neurosurgeons consider any of the following to represent **indications for surgery** in the head-injured patient: extraaxial hematoma with midline shift greater than 5 mm, intraaxial hematoma with volume >30 cc, an open skull fracture, or a depressed skull fracture with more than 1 cm of inward displacement (**Table 32.8**). Also, any temporal or cerebellar hematoma that is greater than 3 cm in diameter usually is evacuated prophylactically because these regions of the brain do not tolerate additional mass as well as other regions of the brain.

If there is no surgical lesion present on the CT scan, or following surgery if one is present, **medical treatment** of the head injury begins. The first phase of treatment is to institute general supportive measures. After appropriate fluid resuscitation has been completed, intravenous fluids are administered to maintain the patient in a state of euvolemia or mild hypervolemia. A previous tenet of head injury treatment was fluid restriction, which was thought to limit the development of cerebral edema and increased ICP. Fluid restriction decreases intravascular volume and decreases cardiac output. A decrease in cardiac output often results in decreased cerebral flow, which results in decreased brain perfusion and may cause an increase in cerebral edema and ICP. Thus, **fluid restriction is contraindicated in the TBI patient**.

Another supportive measure used to treat TBI patients is **elevation of the head**. When the head of the bed is elevated to 30 degrees, the venous outflow from the brain is improved, and this helps to reduce ICP. If a patient is hypovolemic, elevation of the head may cause a drop in cardiac output and cerebral blood flow. Therefore, the head of the bed is not elevated in hypovolemic patients. Also, the head should not be elevated in patients in whom a spine injury is suspected or until an unstable spine has been stabilized.

[4] Marshall LF, Gautille T, Klauber M, et al. The outcome of severe closed head injury. Neurosurgery 75:S28–S36, 1991.

Table 32.9. Indications for ICP Monitoring in TBI Patients.

GCS < 9
Patient requiring prolonged deep sedation or muscle relaxants
TBI patient undergoing prolonged general anesthesia

GCS, Glascow Coma Scale; ICP, intracranial pressure; TBI, traumatic brain injury.

Sedation often is necessary in TBI patients. Some patients with head injuries are significantly agitated and require sedation. Also, patients with multisystem trauma often have painful systemic injuries that require analgesics, and most intubated patients require sedation. Short-acting sedatives and analgesics should be used to accomplish proper sedation without eliminating the ability to perform periodic neurologic assessments. This requires careful titration of medication doses and periodic weaning or withholding of sedation to allow for neurologic assessment.

The use of **anticonvulsants** in TBI is a controversial issue. There is no evidence that the use of anticonvulsants decreases the incidence of late-onset seizures in patients with either closed head injury or traumatic brain injury. Temkin et al[5] demonstrated that the routine use of Dilantin in the first week following TBI decreases the incidence of early-onset (within 7 days of injury) seizures, but it does not change the incidence of late-onset seizures. Also, the prevention of early post-traumatic seizures does not improve the outcome following TBI. Therefore, the prophylactic use of anticonvulsants is not recommended for more than 7 days following TBI and is considered optional in the first week following TBI.

Intracranial Pressure Monitoring: After general supportive measures have been instituted, the issue of ICP is addressed. Intracranial pressure monitoring consistently has been shown to improve outcome in head-injured patients. It is indicated for any patient with a GCS <9 or for any patient in whom serial neurologic examinations cannot be performed (e.g., any patient with a head injury who requires prolonged deep sedation/pharmacologic relaxants or any head injury patient undergoing extended general anesthesia) (**Table 32.9**).

Intracranial pressure monitoring involves the placement of an invasive probe. Intracranial pressure may be monitored by means of an **intraparenchymal monitor or an intraventricular monitor (ventriculostomy)**. **Intraparenchymal ICP monitors** are devices that are placed into the brain parenchyma and measure ICP by means of fiberoptics, strain gauge, or other technologies. These monitors are very accurate; however, they do not allow for drainage of CSF. A **ventriculosotomy** is a catheter placed into the lateral ventricle through a small twist drill hole in the skull. The ICP is then measured by transducing the pressure in a fluid column. Ventriculostomies allow for the drainage of CSF, which can be effective in decreasing the ICP.

[5] Temkin NR, Dikmen SS, Wilensky AJ, et al. A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. N Engl J Med 1990;323(8): 497–502.

Once an ICP monitor has been placed, ICP is monitored continuously. The normal range of ICP in the adult is 0 to 20 mm Hg. The normal ICP waveform is a triphasic wave in which the first peak is the largest peak and the second and third peaks progressively are smaller. When intracranial compliance is abnormal, the second peak becomes larger than the first peak. Also, when intracranial compliance is abnormal, pathologic waves may appear. Lundberg et al[6] described three types of abnormal ICP waves: A, B, and C. Lundberg A waves (plateau waves) have a duration of between 5 and 20 minutes and an amplitude up to 50 mm Hg over the baseline ICP. After an A wave dissipates, the ICP is reset to a baseline level that is higher than when the wave began. Lundberg A waves are a sign of severely compromised intracranial compliance. The rapid increase in ICP caused by these waves can result in a significant decrease in CPP and may lead to herniation. Lundberg B and C waves have a shorter duration and a lower amplitude than A waves, and, as a result, these waves are not as deleterious as A waves.

Treatment of Increased ICP: The goal of treatment of increased ICP is to optimize conditions within the brain to prevent secondary injury. Maintaining ICP within the normal range is part of an approach designed to optimize both CBF and the metabolic state of the brain. There are many potential interventions used to lower ICP, and each of these is designed to improve **intracranial compliance**, which results in improved CBF, increased CPP, and decreased ICP. The **Monro-Kellie doctrine** provides the framework for understanding and organizing the various treatments for elevated ICP.[7] In the TBI patient with increased ICP, the volume of one of the three components of intracranial volume must be reduced in order to improve intracranial compliance and decrease ICP. If there is an intracranial mass lesion greater than 30 cc in volume, it should be evacuated prior to initiating treatment of increased ICP. The discussion of the different treatments for elevated ICP will be organized according to which component of intracranial volume they affect. **Algorithm 32.2** provides an overview of the treatment of increased intracranial pressure.

Management of elevated ICP involves using a combination of some of the treatments. Although there are no rigid protocols for the treatment of head injury, there are many algorithms published that provide treatment schema. The American Association of Neurologic Surgeons published a comprehensive evidence-based review of the treatment of traumatic brain injury called the **Guidelines for the Management of Severe Head Injury**.[8] In these guidelines, there are three different categories of treatments: standards, guidelines, and options (**Table 32.10**).

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[6] Lundberg N, Troup H, Lorin H. Continuous recording of ventricular pressure in patients with severe acute traumatic brain injury. J Neurosurg 1965;22:581–590.

[7] Chestnut RM, Marshall LF. Treatment of abnormal intracranial pressure. Neurosurg Clin North Am 1991;2(2):267–284.

[8] Bullock R, Chestnut R, Clifton G, et al. Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma 1996;13(11):641–734.

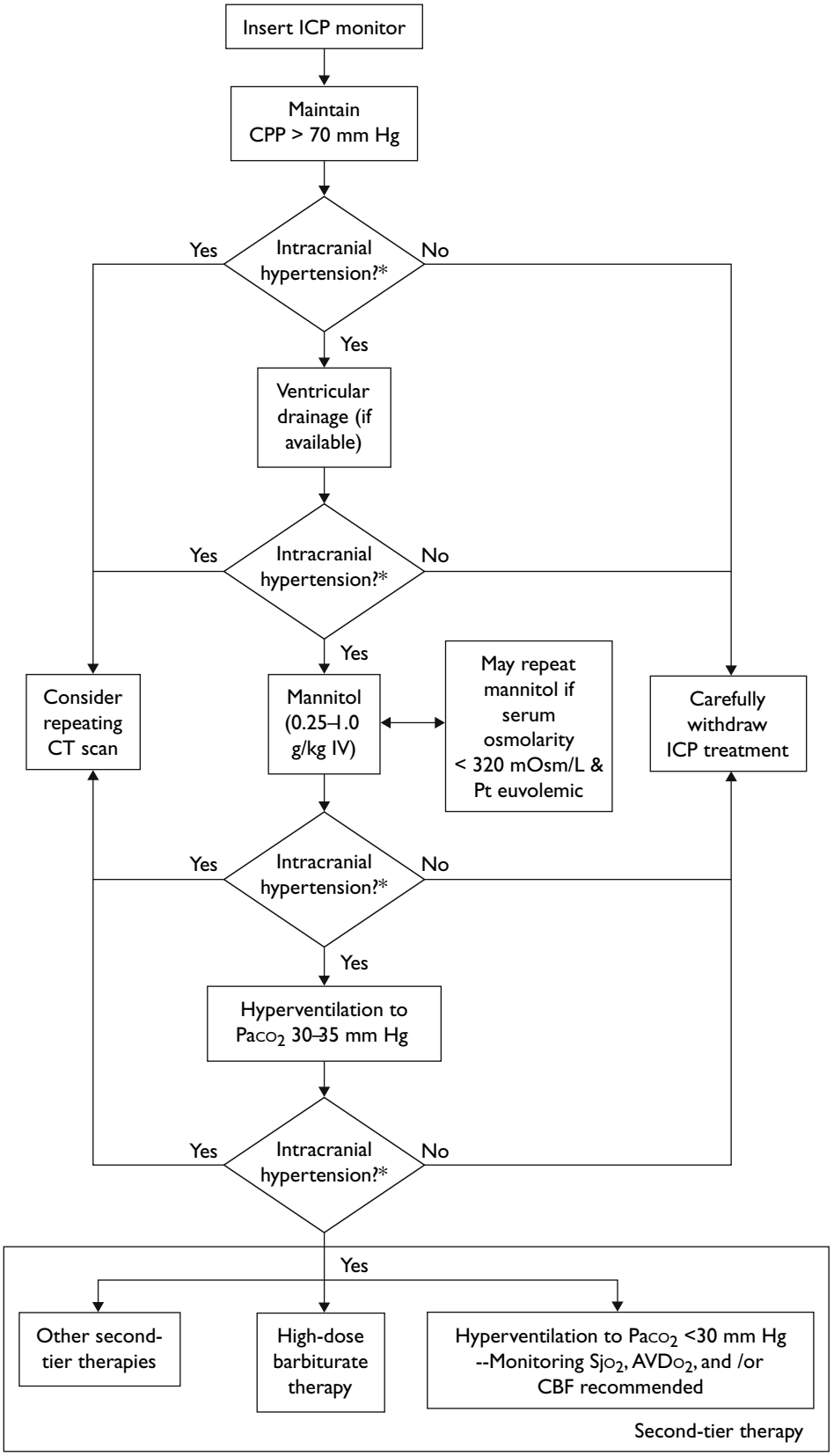
Insert ICP monitor
Maintain CPP > 70 mm Hg
Intracranial hypertension?*
Yes
No
Ventricular drainage (if available)
Intracranial hypertension?*
Yes
No
Consider repeating CT scan
Mannitol (0.25–1.0 g/kg IV)
May repeat mannitol if serum osmolarity < 320 mOsm/L & Pt euvolemic
Carefully withdraw ICP treatment
Intracranial hypertension?*
Yes
No
Hyperventilation to $Paco_2$ 30–35 mm Hg
Intracranial hypertension?*
Yes
No
Other second-tier therapies
High-dose barbiturate therapy
Hyperventilation to $Paco_2$ <30 mm Hg --Monitoring Sjo_2, $AVDo_2$, and /or CBF recommended
Second-tier therapy

Table 32.10. Techniques for controlling elevated intracranial pressure (ICP) (level II and level III evidence).

Technique	Description	Level of evidence
Surgical evacuation of mass lesions	When a mass lesion is >30 mL and is surgically accessible, craniotomy for removal will contribute to ICP control	III
Head elevation	The head should be elevated to 30 degrees or greater, slightly extended, and not rotated	III
Optimize venous drainage	Loosen devices that constrict venous return in the neck, such as a cervical collar or an excessively tight tie, for the endotracheal tube	III
Intravenous mannitol	Osmotic diuretics such as mannitol may be given to keep serum osmolarity at 290–310 mOsm/dL; diuresis beyond 320 mOsm/dL is counterproductive as it diminishes cerebral perfusion and may cause renal failure	II[a]
CSF drainage	Drainage performed by ventriculostomy, *not* by spinal tap or spinal drain; may be impractical when ventricles are very small; increasingly performed, but not yet convincingly supported by class I evidence	II[b]
Sedation	Indicated for patients with elevated ICP who are agitated and straining against mechanical ventilation; may use 2% propofol infusion; shown to lower ICP (day 3) in comparison with controls; increasingly performed, but not yet convincingly supported by class I evidence	II[a]
Mild temporary hyperventilation	For temporary control of elevated ICP, mild reduction of P_{CO_2} to 30–35 may be useful; however, randomized studies have shown that long-term or aggressive hyperventilation is harmful because it lowers cerebral perfusion, and efficacy of mild temporary hyperventilation for improved outcomes has not been demonstrated	III[b]
Pentobarbital coma	Load 10 mg/kg over 30 min, then 5 mg/kg q 1 h for 3 doses, then 1 mg/kg/h; titrate to keep serum pentobarbital level 3–4 mg/dL, or to maintain a "burst suppression pattern" on a portable EEG (only some randomized studies have supported the effectiveness of pentobarbital coma for lowering ICP)	II[a,c]
Mild hypothermia	Now under study for severely elevated ICP in head trauma; preliminary class II evidence suggests efficacy	II[a]

[a] Allen CH, Ward JD. An evidence-based approach to management of increased intracranial pressure. Crit Care Clin 1998;14:485–495.

[b] Poungvarin N, Boopat W, Viriyavejakul A, et al. Effects of dexamethasone in primary supratentorial intracerebral hemorrhage. N Engl J Med 1987;316:1229–1233.

[c] Ward JD, Becker DP, Miller JD, et al. Failure of prophylactic barbiturate coma in the treatment of severe head injury. J Neurosurg 1985;62:383–388.

Source: Reprinted from Starr P. Neurosurgery. In: Norton JA, Bollinger RR, Chang AE, et al, eds. Surgery: Basic Science and Clinical Evidence. New York: Springer-Verlag, 2001, with permission.

Algorithm 32.2. Critical pathway for treatment of intracranial hypertension in the severe head injury patient. CPP, cerebral perfusion pressure. (Reprinted from Bullock R, Chestnut R, Clifton G, et al. Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma 1996;13(11):641–734. Copyright © 1995, Brain Trauma Foundation.)

Blood, CSF, and Brain Components: The first component of total intracranial volume to be considered is the **vascular component**. This includes both the venous and arterial compartments. Elevation of the head, as discussed earlier, increases venous outflow and decreases the volume of venous blood within the brain. This results in a small improvement in intracranial compliance, and therefore, it has only a modest effect on ICP. Another component of intracranial vascular volume is the arterial blood volume. This may be reduced by mild to moderate **hyperventilation**, in which the P_{CO_2} is reduced to between 30 and 36 mm Hg. This decrease in P_{CO_2} causes vasoconstriction at the level of the arteriole, which decreases cerebral blood volume enough to reduce ICP. The effect of hyperventilation has a duration of action of approximately 2 to 3 days, after which the cerebral vasculature resets to the reduced level of P_{CO_2}. This is an important point because, **once hyperventilation is implemented, the P_{CO_2} should not be returned rapidly to normal**, as this may cause rebound vasodilatation, which can result in increased ICP. Severe hyperventilation was, at one time, an important component of the treatment of TBI and increased ICP. It has been shown that reducing P_{CO_2} to 25 mm Hg or lower, causes enough vasoconstriction that CBF is reduced to the point where there is a high probability of developing cerebral ischemia. Thus, **prolonged severe hyperventilation is not used routinely to treat elevated ICP**. Brief periods of severe hyperventilation may be used to treat patients with transient ICP elevations due to pressure waves or in the initial treatment of the patient in neurologic distress until other measures can be instituted. There are rare instances in which ICP elevations are due to excessive CBF, a condition known as hyperemia. When hyperemia is the cause of increased ICP, severe hyperventilation may be utilized. This requires the use of jugular venous saturation monitoring to evaluate for excessive cerebral oxygen extraction, which indicates that the degree of hyperventilation is too severe and may cause cerebral ischemia.

Cerebrospinal fluid represents the second component of total intracranial volume and accounts for 2% to 3% of total intracranial volume. Total CSF production in the adult is approximately 20 cc per hour. In many TBI patients with elevated ICP, a ventriculostomy may be placed, and CSF may be drained. Cerebrospinal fluid drainage frequently results in significant improvement in ICP.

The third and largest component of total intracranial volume is the **brain or tissue component**, which comprises 85% to 90% of the total intracranial volume. When there is significant brain edema, it causes an increase in the tissue component of the total intracranial volume and results in decreased compliance and an increase in ICP. Treatments for elevated ICP that reduce total brain volume are diuretics, cerebral perfusion pressure augmentation (CPP strategies), metabolic suppression, and decompressive procedures.

Diuretics: Diuretics are very powerful in their ability to decrease brain volume and therefore decrease ICP. **Mannitol**, an osmotic diuretic, is the most common diuretic used. Mannitol draws water out of the brain into the intravascular compartment. It has a rapid onset of action and

an average duration of 4 hours. Mannitol is more effective when given in intermittent boluses than if it is used as a continuous infusion. The standard dose is between 0.25 g/kg and 1.00 g/kg given every 4 to 6 hours. Electrolytes and serum osmolality must be monitored carefully during its use. Also, sufficient hydration must be administered to maintain euvolemia. Mannitol should not be given if the serum Na is >147 or if the serum osmolarity is >315. There is a maximum dose of 4 g/kg of mannitol/day. At doses higher than this, mannitol can cause renal toxicity.

Cerebral Perfusion Pressure (CPP) Management: Cerebral perfusion pressure (CPP) management involves artificially elevating the blood pressure to increase the MAP and the CPP. Because there is impaired autoregulation in the injured brain, there is pressure passive cerebral blood flow within these injured areas. As a result, these injured areas of the brain often have insufficient blood flow, and there will be tissue acidosis and lactate accumulation. This causes vasodilation, which increases cerebral edema and ICP. When the CPP is raised to 65 or 70 mm Hg, the ICP often is lowered because increased blood flow to injured areas of the brain results in better perfusion and decreases the tissue acidosis. This often results in a significant decrease in ICP.

Metabolic Therapies: Metabolic therapies are designed to decrease the cerebral metabolic rate, which, in turn, decreases the ICP. Metabolic therapies are a powerful means of reducing ICP, but they are reserved for situations in which other therapies have failed to control ICP because of their many potential adverse effects. Some of these adverse effects are hypotension, immunosuppression, coagulopathies, arrhythmias, and myocardial suppression. Metabolic suppression may be achieved through drug-induced metabolic inhibition or induced hypothermia.

Barbiturates are the most common class of drugs used to suppress cerebral metabolism. Barbiturate coma typically is induced with pentobarbital. A loading dose of 10 mg/kg is given over 30 minutes, and then 5 mg/kg/hr is given for 3 hours. A maintenance infusion of between 1 and 2 mg/kg/hr is begun after loading is completed. The infusion is titrated to provide burst suppression on continuous electroencephalogram (EEG) monitoring and serum level of 3 to 4 mg/dL. Typically, the barbiturate infusion is continued for 48 hours, and the patient is weaned off the barbiturates. If the ICP again escapes control, the patient may be reloaded with pentobarbital and weaned again in several days.

Hypothermia also may be used to suppress cerebral metabolism. The use of mild hypothermia involves decreasing the core temperature to 34° to 35°C for 24 to 48 hours and then slowly rewarming the patient over 2 to 3 days. Hypothermia patients also are at risk for hypotension and systemic infections.

Decompressive Procedures: Another treatment that may be used in the TBI patient with refractory ICP elevation is **decompressive craniectomy**. This is a surgical procedure in which a large section of the skull is removed and the dura is expanded.

Penetrating Trauma

There are two main aspects of the **treatment of penetrating brain injuries**. The first is the **treatment of the traumatic brain injury caused by the penetrating object**. Penetrating brain injuries, especially from high-velocity missiles, frequently result in severe ICP elevations. This aspect of penetrating brain injury treatment is identical to the treatment of closed head injuries. The second aspect of penetrating head injury treatment involves **debridement and removal of the penetrating objects**. Bullet wounds are treated by debridement of as much of the bullet tract as possible, dural closure, and reconstruction of the skull as needed. If the bullet can be removed without significant risk of neurologic injury, it should be removed to decrease the risk of subsequent infection. Penetrating objects, such as knives, require removal to prevent further injury and infection. If the penetrating object is either near or traverses a major vascular structure, an angiogram is necessary to assess for potential vascular injury. When there is the possibility of vascular injury, penetrating objects should be removed only after appropriate vascular control is obtained.

Penetrating brain injuries are associated with a high rate of infection, both early infections as well as delayed abcesses. Appropriate debridement and irrigation of wounds help to decrease the infection rate. Late-onset epilepsy is a common consequence of penetrating brain injuries and can occur in up to 50% of patients with penetrating brain injuries. There is no evidence that prophylactic anticonvulsants decrease the development of late-onset epilepsy.

Head Injury in Children

There are several ways in which **head injury in children** differs from head injuries in adults. Children tend to have more diffuse injuries than adults, and traumatic intracerebral hematomas are less common in children than in adults. Also, early posttraumatic seizures are more common in children than in adults.

When a child with a head injury is being evaluated, **nonaccidental trauma must be ruled out**. Traumatic brain injury is the most common cause of morbidity and mortality in nonaccidental trauma in children. Radiographic signs of nonaccidental trauma include unexplained multiple or bilateral skull fractures, subdural hematomas of different ages, cortical contusions and shearing injures, cerebral ischemia, and retinal hemorrhages. If any of these are present, the case should be referred to the proper child welfare agency.

Complications

Neurologic complications resulting from TBI are quite common and include neurologic deficits, hydrocephalus, seizures, cerebrospinal fluid fistulas, vascular injuries, infections, and brain death.

The **neurologic deficits** that result from TBI include focal motor deficits, cranial nerve injuries, and cognitive dysfunction. Common

cranial nerve deficits following TBI include olfactory nerve dysfunction, hearing loss, and facial nerve injury. Facial nerve injuries are common when there is a temporal bone fracture and occur in 10% to 30% of longitudinal temporal bone fractures and 30% to 50% of transverse fractures.

Hydrocephalus is a common, late complication of TBI. Posttraumatic hydrocephalus may present as either ventriculomegaly with increased ICP or as normal pressure hydrocephalus. Those patients with increased ICP secondary to posttraumatic hydrocephalus demonstrate the typical signs of hydrocephalus including headaches, visual disturbances, nausea/vomiting, and alterations in level of consciousness. Normal pressure hydrocephalus usually presents with memory problems, gait ataxia, and urinary incontinence. Any patient who develops neurologic deterioration following TBI should be investigated for the possibility of hydrocephalus.

Posttraumatic seizures are a complication of TBI and are divided into three categories: early, intermediate, and late. Early seizures occur within 24 hours of the initial injury; intermediate seizures occur between 1 and 7 days following injury; and late seizures occur more than 7 days after the initial injury. Posttraumatic seizures are very common in those with a penetrating cerebral injury, and late seizures occur in as many as one half of these patients.

Cerebrospinal fluid fistulas, either rhinorrhea or otorrhea, may occur in as many as 5% to 10% of patients with basilar skull fractures. Approximately 80% of acute cases of CSF rhinorrhea and 95% of CSF otorrhea resolve spontaneously within 1 week. There is a 17% incidence of meningitis with CSF rhinorrhea and a 4% incidence of meningitis with CSF otorrhea. Prophylactic antibiotics have not been demonstrated to decrease this meningitis risk. When acute CSF fistulas do not resolve spontaneously, potential treatments include CSF drainage by means of a lumbar subarachnoid drain or craniotomy for dural repair.

Vascular injuries are uncommon sequelae of traumatic brain injury. Arterial injuries that may occur following head trauma include arterial transections, posttraumatic aneurysms, dissections, and carotid-cavernous fistulas (CCFs).

Intracranial infections in uncomplicated closed head injury also are uncommon. When basilar skull fractures or CSF fistulas are present, there is an increased risk of infection as discussed above. As expected, there is a higher incidence of infection in penetrating cerebral injuries and open depressed skull fractures.

Brain death can result from either massive initial injury or prolonged severe elevations of ICP. Brain death is defined as the absence of any voluntary or reflex cerebral function. **A strict protocol is necessary to prove that brain death has occurred.** It must be established that there are no sedating medication or neuromuscular blocking agents present. The patient's electrolytes, blood count, body temperature, and arterial blood gas all must be within the normal range. The neurologic exam should demonstrate the absence of all brainstem reflexes and no response to central painful stimuli. The lack of any neu-

Table 32.11. Glasgow Outcome Scale.

Assessment	Definition
Good recovery (G)	Patient returns to preinjury level of function
Moderately disabled (MD)	Neurologic deficits but able to look after self
Severely disabled (SD)	Unable to look after self
Vegetative (V)	No evidence of higher mental function
Dead (D)	

rologic response is not sufficient to establish brain death, and confirmatory testing must be performed. Confirmatory tests include the apnea test, cerebral nuclear blood flow study, angiogram, and EEG. Two neurologic exams and two confirmatory tests are required to establish brain death.

Outcome

The outcome of traumatic brain injury, as one would expect, is related to the initial level of injury. While the initial GCS provides a description of the initial neurologic condition, it does not correlate tightly with outcome. The **Glasgow Outcome Scale (GOS)** is the most widely used tool to follow TBI patient outcomes.[9] This scale divides outcome into five categories: good, moderately disabled, severely disabled, vegetative, and dead (**Table 32.11**). Many methods have been devised in an attempt to predict the outcome of TBI. These algorithms often are complex, and few are able to accurately predict the outcome of TBI. There is one simplified model that uses three factors: age, motor score of the GCS, and pupillary response (normal, unilateral unresponsive pupil, or bilateral unresponsive pupil); it provides a reasonable assessment of outcome. While it is extremely difficult to predict the outcome of TBI, several factors have been identified that correlate with poor outcomes. The Traumatic Coma Data Bank analyzed 753 patients with TBI and identified five factors that correlated with a poor outcome[10]: age >60; initial GCS <5; presence of a fixed, dilated pupil; prolonged hypotension or hypoxia early after injury; and the presence of a surgical intracranial mass lesion.

Summary

Traumatic brain injury is a common problem in the United States, affecting approximately 550,000 people annually. Patients with TBI should be evaluated by the ATLS protocol and serial GCS assessments, followed by a thorough neurologic exam when they are systemically stable. Head CT is the diagnostic imaging modality of choice in the

[9] Jennet B, Bond M. Assessment of outcome after severe brain damage: a practical scale. Lancet 1975;1:480–484.

[10] Marshall LF, Gautille T, Klauber M, et al. The outcome of severe closed head injury. Neurosurgery 75:S28–S36, 1991.

evaluation of TBI, and MRI scanning is used only in special situations. Some patients with mild head injuries may not require CT scanning; however, patients with moderate or severe injuries must be evaluated with a CT scan. If no surgical lesion is present or following surgery if one is present, specific treatment of the head injury begins. Patients with a GCS of 8 or less require placement of an ICP monitor. If the ICP is elevated, these patients are treated with some or all of the following: CSF drainage, modest hyperventilation, and diuretics. These strategies are designed to decrease the volume of one of the three intracranial components (brain, blood, and CSF) in order to improve intracranial compliance and decrease ICP. If these strategies are not effective in controlling ICP, then other treatment modalities, such as barbiturate coma, hypothermia, or decompressive craniectomy, may be utilized. There are many potential neurologic complications of head injury including cranial nerve deficits, seizures, infections, hydrocephalus, and brain death. While patients with mild head injury usually do well, some of those who develop a postconcussive disorder are disabled permanently. It is very difficult to predict the outcome of moderate and severe head injuries, and most algorithms devised for this purpose do not reliably predict outcome. As expected, many of these patients have poor outcomes.

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