CHAPTER 34

Head Trauma

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PRINCIPLES OF DISEASE

Background and Importance

Head trauma is a broad term describing an external trauma to the craniofacial area of the body from blunt, penetrating, blast, rotational, or acceleration-deceleration forces, the term *head injury* refers to a clinically evident injury on physical examination and is recognized by the presence of ecchymosis, lacerations, or deformities, and the term *traumatic brain injury* (TBI) indicates an injury to the brain itself.

Head trauma accounts for approximately 1.5 million emergency department (ED) visits annually in the United States; one-third of these are children younger than 14 years. Falls and motor vehicle collisions (MVCs) account for almost 75% of head trauma in the civilian population.[1] TBI caused by blasts has resulted in disproportionate morbidity to combatants in the recent wars in Iraq and Afghanistan.[2,3] As veterans return to the United States, the number of patients experiencing the consequences of TBI continues to increase.

Gunshot wounds (GSWs) to the head are particularly lethal; the overall mortality rate is estimated to be 90%, with 70% of deaths occurring at the scene.[4] However, in a subset of these patients with good initial neurologic function, survival approaches 75%.

The ultimate survival and neurologic outcome of the brain-injured patient depends on the extent of TBI occurring at the time of injury (primary injury) and the effects of systemic insults (secondary injury), such as those caused by hypotension and hypoxia. Thus, clinical care of patients with TBI emphasizes early management to minimize the occurrence of secondary brain injury. Emergency clinicians influence the incidence and severity of primary brain injury only through injury prevention programs (see Chapter e2).

A number of terms describing mild traumatic brain injury (MTBI) have been used in the past, including minor, minimal, grade I, class I, and low risk. The American Congress of Rehabilitation Medicine defines a patient with MTBI as one who has a Glasgow Coma Scale (GCS) score of 13 to 15, with traumatically induced physiologic disruption of brain function, as manifested by at least one of the following: (1) any period of loss of consciousness less than 30 minutes; (2) any loss of memory for events immediately before or after the accident (posttraumatic amnesia should last <24 hours); (3) any alteration in mental state at the time of the accident (eg, feeling, dazed, disoriented, or confused); and (4) focal neurologic deficits that may or may not be transient (Box 34.1).

Individuals with MTBI are acutely at risk for serious intracranial injuries. Up to 17% of patients with suspected MTBI in the ED have abnormal computed tomography (CT) scans. Although the incidence of life-threatening lesions that require neurosurgical intervention in suspected MTBI is only about 1%, these patients have an important risk of subsequent deterioration from intracranial bleeding. If these cases are recognized and treated early, a full recovery is likely; if not, severe disability or death may ensue.

The GCS score was not originally intended for use in MTBI patients,[5] and some authors have suggested that patients with a GCS score of 13 or 14 be excluded from the mild category and placed into the moderate-risk group due to the higher risk of neurosurgical intervention.[6] However, a patient who is intoxicated from drugs and alcohol may present with a GCS score of 13 to 14. Furthermore, over 10% of patients who become comatose start with a GCS score of 15. Patients can deteriorate from an expanding intracranial hematoma after what appears clinically to be a MTBI. Among MTBI patients, those with GCS scores trending downward (worsening neurologic status) are at higher risk of neurosurgical intervention and have a less favorable outcome than those with GCS scores trending upward (improving neurologic status).[7,8]

Anatomy and Pathophysiology

Scalp and Cranium

The scalp consists of five tissue layers (Fig. 34.1). The skull is comprised of the frontal, ethmoid, sphenoid, and occipital bones and two parietal and two temporal bones. Each bone consists of solid inner and outer layers separated by a layer of cancellous bone tissue (the diploë). In adults, the bones of the skull average 2 to 6 mm in thickness; the bones in the temporal region are usually the thinnest of the skull. The cranial bones form a smooth outer surface of the skull, but within the cranial vault are many bone protrusions and ridges. Contrecoup injuries and contusions far from the site of head impact occur as the brain strikes against uneven bone surfaces. After the first few months of life, the cranial bones begin to fuse, ultimately forming the rigid, nonexpendable cranial vault. The inner aspect of the skull is lined with the periosteal dura, which is a thick connective tissue layer that adheres closely to the bone surface. The inner meningeal layer of the dura is the outermost covering of the brain. This dural membrane reflects back on itself to make folds within the cranial space. These folds serve to protect and compartmentalize different components of the brain. The midline falx cerebri separates the two cerebral hemispheres from each other. The tentorium cerebelli partitions the cerebellum and brainstem from the cerebral hemispheres. The U-shaped free margin of this dural fold is important in the pathology of the transtentorial herniation syndromes. Within the margins of the dural reflections, the two dural layers separate to form large dural venous sinuses. Injury to the dural sinuses is associated with significant morbidity and mortality because of the potential for uncontrolled hemorrhage.

Brain and Cerebrospinal Fluid

The brain is a semisolid structure that weighs approximately 1400 g (3 lb) and occupies approximately 80% of the cranial vault, with the remaining space occupied primarily by vasculature

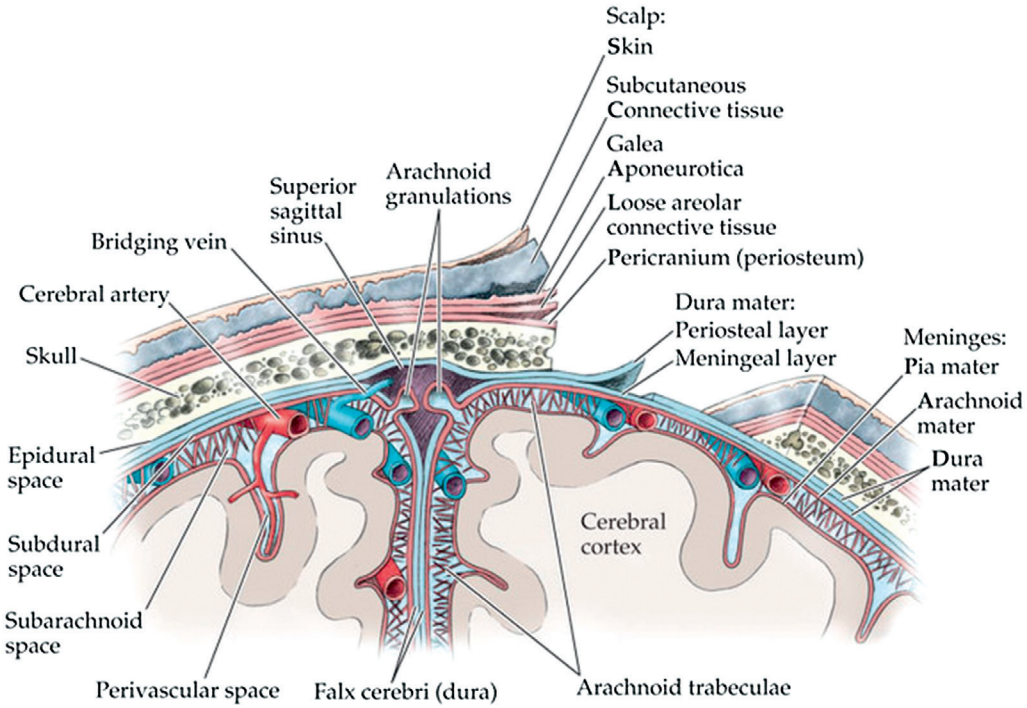

Fig. 34.1. Layers of the soft tissues, skull, and meninges. The dermis is the outermost layer and is among the thickest layers of skin on the body. The underlying subcutaneous tissue contains the hair follicles and rich blood supply of the scalp. The galea, made of tough fascial tissue, contains the occipitofrontalis and temporoparietalis muscles, which move the scalp backward and forward, elevate the eyebrows, and wrinkle the forehead. Under the galea is a loose areolar tissue layer. The deepest layer of the scalp, the pericranium, is firmly adhered to the skull. (From Blumenfeld H: Neuroanatomy through clinical cases, Sunderland, 2002, Sinauer Associates, Incorporated.)

BOX 34.1

Definition of Mild Traumatic Brain Injury

According to the American Congress of Rehabilitation Medicine, a person with mild traumatic brain injury (MTBI) is a patient with a GCS of 13–15 who has had a traumatically induced physiologic disruption of brain function, as manifested by at least one of the following:

1. Any period of loss of consciousness less than 30 min
2. Any loss of memory for events immediately before or after the accident (posttraumatic amnesia should last <24 hr)
3. Any alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused)
4. Focal neurologic deficit(s) that may or may not be transient

and cerebrospinal fluid. The brain is covered by three distinct membranes—the meningeal dura, arachnoid layer, and pia (Fig. 34.2). The location of traumatic hematomas relative to these membranes defines the pathologic condition and determines the consequences of the injury.

The brain is suspended in the cerebrospinal fluid (CSF), which provides some physical buffering for the brain during trauma. CSF is produced by the choroid plexus, located primarily in the lateral ventricles of the brain. CSF passes from the ventricular system into the subarachnoid space that surrounds the brain and spinal cord. The normal pressure exerted by the CSF is 65 to 195 mm H_2O or 5 to 15 mm Hg.

The blood-brain barrier (BBB) maintains the microenvironment of the brain tissue and CSF. Extracellular ion and neurotransmitter concentrations are regulated by movement across this barrier. When the BBB is intact, the ability of neuroactive drugs

Fig. 34.2. Diffuse axonal injury (DAI), otherwise known as traumatic axonal injury (TAI), is characterized by axonal stretching leading to axolemmal disruption, ionic flux, neurofilament compaction, and microtubule disassembly, resulting in axonal swelling and disconnection. Axonal swelling and disconnection can lead to axon death. **a,** Normal neuron, **b, c,** Axon reaction to increasing stretch. **d,** Retraction balls have formed, and aggregates of axonal material lie along the course of the axon. (From Peerless SJ, Rewcastle NB: Shear injuries of the brain. CMAJ 96:577–582, 1967.)

to penetrate into the brain tissue usually depends on their lipid solubility. However, the biomechanics of a brain injury or posttraumatic cerebral edema can cause a disruption of the BBB for up to several hours after the insult. Prolonged disruption of the BBB further contributes to the development of posttraumatic vasogenic cerebral edema and higher maximum intracranial pressure.[9]

Cerebral Hemodynamics and Increased Intracranial Pressure

The brain has an extremely high metabolic rate, using approximately 20% of the entire oxygen consumed by the body and requiring approximately 15% of the total cardiac output. In the normal brain, cerebral blood flow (CBF) is maintained at constant levels. Optimal regional CBF is maintained by the ability of the cerebral vessels to alter their diameter in response to changing physiologic conditions. The responses of the cerebral vasculature to changing physiologic conditions protect the brain by increasing the delivery of oxygen to tissue, enhancing the removal of metabolic end products, and allowing nearly instantaneous adjustments to meet changing metabolic demands. Hypertension, alkalosis, and hypocarbia promote cerebral vasoconstriction, whereas hypotension, acidosis, and hypercarbia cause cerebral vasodilation.

Cerebral vasoactivity is also very sensitive to changes in the partial pressures of carbon dioxide and oxygen (P_{CO_2} and P_{O_2}, respectively). The response to changes in P_{CO_2} is nearly linear between P_{CO_2} values of 20 and 60 mm Hg. In this range, lowering P_{CO_2} by as little as 1 mm Hg decreases the diameter of cerebral vessels by 2% to 3%, corresponding to an overall change in CBF of 1.1 mL/100 g of tissue/min. This is the physiologic rationale for intentional hyperventilation in the setting of rapid and marked increases in intracranial pressure (ICP). Hyperventilation causes P_{CO_2} to fall, resulting in cerebral vasoconstriction, but this is no longer recommended as a mechanism for reducing ICP. The cerebral vessels also respond to changes in P_{O_2}. As P_{O_2} declines, cerebral vessels dilate to ensure adequate oxygen delivery to brain tissue. When brain injury occurs, increased CBF, vascular dilation, and a disrupted BBB promote vasogenic edema and can further increase ICP.[9] Thus, avoiding or reversing hypoxia is essential in managing the brain-injured patient.

CBF also depends on the cerebral perfusion pressure (CPP), which is the pressure gradient across the brain. CBF remains fairly constant when CPP is 50 to 160 mm Hg. This is referred to as autoregulation and occurs with a mean arterial pressure (MAP) of 60 to 150 mm Hg. The determinants of CPP are MAP and the resistance to CBF produced by the mean systemic venous pressure and ICP. Because ICP is higher than mean systemic venous pressure, ICP effects predominate, and CPP can be approximated as follows:

$$CPP = MAP - ICP$$

If CPP falls below 40 mm Hg, autoregulation is lost and CBF declines, resulting in tissue ischemia and altered cerebral metabolism. Avoidance of hypotension or elevation in ICP in the head-injured patient helps ensure that CPP can be maintained.

The recommended target CPP value for improved outcomes is between 60 and 70 mm Hg. Whether 60 or 70 mm Hg is the minimum optimal CPP threshold is unclear and may depend on the patient's autoregulatory status.[10]

Increased Intracranial Pressure

Increased ICP is defined as CSF pressure greater than 15 mm Hg (or 195 mm H_2O) and is a frequent consequence of a severe TBI. Initially, as ICP increases as a result of a traumatic mass lesion or edema, CSF is displaced from the cranial vault to the spinal canal, offsetting the increased blood or brain volume. When this compensatory mechanism is overwhelmed, the elastic properties of the brain substance allow tissue compression to provide buffering for the increasing pressure. Depending on the location and rate of mass expansion and edema formation, the intracranial compensatory mechanisms can accommodate an increased volume of 50 to 100 mL. Beyond that, even small changes in intracranial relationships, such as from vasodilation, CSF obstruction, or areas of focal edema, may increase ICP. If ICP increases to the point at which CPP is compromised, vasoparalysis occurs and autoregulation is lost. The CBF then depends directly on the systemic MAP. With the loss of autoregulation, massive cerebral vasodilation occurs. Systemic pressure is transmitted to the capillaries, contribute to vasogenic edema, and further increase ICP.

ICP above 22 mm Hg is associated with increased mortality and warrants treatment.[10] Methods to reduce elevated ICP include use of osmotic and diuretic agents and CSF drainage. Simple techniques to reduce ICP include head of bed elevation to 30 degrees and keeping the neck in a neutral position.[11] Therapeutic hyperventilation, once almost universally used, is potentially harmful and is now used only in as a temporizing measure for a select group of patients for whom other measures are not available or have failed. If ICP is not controlled, herniation will occur, resulting in brainstem compression and cardiorespiratory arrest.

Cushing's Reflex

Progressive hypertension associated with bradycardia and diminished respiratory effort is a specific response to acute, potentially lethal increases in ICP. This response is called Cushing's reflex or Cushing's phenomenon, and its occurrence indicates that the ICP has reached life-threatening levels. However, only one-third of cases of life-threatening increased ICP manifest the full triad of hypertension, bradycardia, and respiratory irregularity.

Definitions and Patterns of Injury

Traumatic Brain Injuries: Severe, Moderate, and Mild

Traditionally, TBI has been separated into the three broad categories of mild, moderate, and severe, primarily based on the GCS score following resuscitation and stabilization. Severe brain injury is defined as a TBI with a postresuscitation GCS score of 8 or lower, moderate as a GCS score of 9 to 12, and mild as a GCS score of 13 to 15. Overall, 80% of patients sustain MTBIs, 10% moderate brain injuries, and 10% severe brain injuries (Table 34.1).[1] The term *concussion* is commonly used to describe MTBIs in sports.

The degree of brain injury following a MTBI or concussion also depends on the primary mechanism and magnitude of injury, secondary insults, and the patient's genetic and molecular response.[12] Primary damage is caused by the initial impact or force that although not usually as evident as severe TBI, may lead to smaller contusions, hematomas, axonal damage, and microvascular injury. Following a MTBI without evidence of lesions on computed tomography (CT) scans, there is a decrease in cerebral blood flow over the ensuing hours and days after injury,[13] as well as cortical neurometabolic abnormalities.[14,15] Traumatic axonal injury (TAI) is also an important determinant of outcome.[16-18]

Increasing evidence has suggested that a single MTBI can produce long-term gray and white matter atrophy, precipitate or accelerate age-related neurodegeneration, and increase the risk of developing Alzheimer's, Parkinson's, and motor neuron disease.[19,20] In addition, repeated episodes of MTBI can provoke the development of chronic traumatic encephalopathy (CTE), a term used to describe clinical changes in cognition, mood, personality,

TABLE 34.1

Traumatic Brain Injury (TBI) as a Portion of All Injuries and Emergency Department (ED) Visits

		ALL INJURIES		TRAUMATIC BRAIN INJURIES		
PARAMETER	ALL VISITS	NO.	PERCENT OF ALL VISITS	NO.	PERCENT OF ALL INJURIES	PERCENT OF ALL VISITS
ED visits[a]	96,839,411	28,697,028	29.6	1,364,797	4.8	1.4
Hospitalizations[b]	36,693,646	1,826,548	5.0	275,146	15.1	0.7
Deaths	2,432,714	169,055	6.9	51,538[c]	30.5	2.1
Total	135,965,771	30,692,631	22.6	1,691,481	5.5	1.2

[a]Persons who were hospitalized, died, or were transferred to another facility were excluded.
[b]In-hospital deaths and patients who transferred from another hospital were excluded.
[c]28 mortality records (from 2002-2006) were omitted because of missing age information.
Adapted from Faul M XL, Wald MM, Coronado VG: Traumatic brain injury in the United States: emergency department visits, hospitalizations and deaths 2002-2006, Atlanta, 2010, Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, pp 1–74.

behavior, and/or movement occurring years following concussion.[21,22] CTE has recently been found to occur after other causes of repeated head trauma, suggesting that any repeated blows to the head, such as those that occur in American football, hockey, soccer, and professional wrestling, in military personnel, and in victims of physical abuse, can also lead to neurodegenerative changes.[20,22,23]

Direct and Indirect Injuries

Direct Injury. Direct head trauma occurs when the head is struck, or its motion suddenly arrest by, an object. The resulting damage to the skull and brain depends on the consistency, mass, surface area, and velocity of the object striking the head. Direct injury can also be caused by compression of the head. External signs of trauma are frequently noted at the site of application of the impact or compression force. The skull initially bends inward at the point of contact. If the force is sufficient, a skull fracture can occur. The cranium absorbs some of the applied energy, whereas some energy is transmitted to the brain by shock waves that travel distant to the site of impact or compression. With sufficient and prolonged application of compression force, the ability of the skull to absorb the force is overcome, and multiple linear skull fractures occur. These resulting fractures can be depressed if a high-energy rapid compression force is applied to a small area of the skull. The extent of direct injury depends on the vasoelastic properties of the underlying region of brain tissue, duration of the force applied, magnitude of the force reaching the brain tissue, and surface area of the brain that is affected.

Indirect Injury. In indirect brain injury, the cranial contents are set into motion by forces other than the direct contact of the skull with an object. A common example is an acceleration-deceleration injury. No direct mechanical impact is sustained, but the cranial contents are set into vigorous motion. As the bridging subdural vessels are strained, subdural hematomas (SDHs) may result.

Differential acceleration of the cranial contents occurs, depending on the physical characteristics of the brain region. As one brain region slides past another, shear and strain occur. These movements result in diffuse injuries, such as a concussion or TAI. Additional injury occurs as the movement of the intracranial contents is abruptly arrested, and the brain strikes the skull or a dural structure. Contrecoup contusions are an example of this injury. In penetrating injury, the object produces pressure waves that can strike structures distal to the path of the missile.

Neurochemical Cascade

There can be secondary insults mediated through physiologic events, which can decrease the supply of oxygen and energy to the brain tissue, or a cascade of cytotoxic events, mediated by many molecular and cellular processes. These events include activation of inflammatory responses, imbalances of ion concentrations (eg, potassium, calcium), an increase in the presence of excitatory amino acids (eg, glutamate), dysregulation of neurotransmitter synthesis and release, imbalance in mitochondrial functions and energy metabolism, and production of free radicals.[24]

Penetrating Head Trauma

The morbidity and mortality from missile injuries to the head depend on the intracranial path, speed of entry, and size and type of the penetrating object. Projectiles that cross the midline or geographic center of the brain, pass through the ventricles, or come to rest in the posterior fossa are associated with extremely high mortality. High-velocity wounds are associated with greater mortality than low-velocity injuries. Large missiles or missiles that fragment within the cranial vault are usually fatal. The design of the bullet and its fragmentation potential (capacity to deform or fragment) also contribute to final tissue destruction and patients' morbidity and mortality.

Tangential wounds are caused by an impact that occurs at an oblique angle to the skull. If the missile has high velocity but low energy, it can travel around the skull, under the scalp, without passing through the skull. Intracranial damage, primarily cortical contusions, can occur at the initial site of impact secondary to pressure waves generated by the impact. Although many patients with tangential GSWs have a GCS score of 15 on presentation, up to 24% also have intracranial hemorrhage, and 16% sustain skull fractures.[25]

Most civilian penetrating brain injuries are penetrating missile wounds, which are produced by moderate- to high-velocity projectiles discharged at close range. The penetrating object may travel through the entire skull, bounce off the opposite inner table of the skull and ricochet within the brain, or stop somewhere within the cranial cavity.

As the bullet passes through the brain, a tissue cavity as much as 10 times the diameter of the missile is created. A percussion shock wave is also created, lasting 2 milliseconds but causing little tissue destruction. The wounding capacity of a firearm is related to the kinetic energy of its missile on impact and how much energy is dissipated. Low-velocity missiles tend to be deflected by

intracranial structures. The final track is therefore erratic and occasionally bears no relation to the exit or entrance site of the missile.

Scalp Wounds

The large blood vessels of the scalp do not fully constrict if they are lacerated and can be the source of substantial blood loss. Because the areolar attachments to the rest of the scalp are loose, scalp avulsions frequently occur through this layer. Subgaleal hematomas can become large because blood easily dissects through the loose areolar tissue. Hemostasis may be difficult to achieve, and blood loss may be significant to the point of causing hemodynamic compromise.

Skull Fractures

Skull fractures are local injuries caused by direct impact to the skull. Although the presence of a skull fracture does not always indicate underlying brain injury, the force required to fracture the skull is substantial, and all patients with skull fractures must be carefully evaluated to ensure that no additional injury is present. The pattern, extent, and type of skull fracture depend on the force of the impact applied and ratio of the impact force to the impact area. Clinically significant features of skull fractures include intracranial air, association with an overlying scalp laceration (open skull fracture), depression below the level of the skull's inner table, and location over a major dural venous sinus or middle meningeal artery.

Linear Fractures

A linear skull fracture is a single fracture that goes through the entire thickness of the skull. Linear skull fractures are clinically important if they cross the middle meningeal groove or major venous dural sinuses; they can disrupt these vascular structures and cause the formation of epidural hematomas (EDHs). Most other linear skull fractures are not clinically significant.

Sutural diastasis is the traumatic disruption of a cranial suture. In adults, sutural diastasis often involves the coronal or lambdoid sutures. Sutural diastasis usually occurs when a linear fracture extends into the suture line, and it is rare after sutures have undergone bone fusion. Comminuted skull fractures, which are multiple linear fractures that radiate from the impact site, usually suggest a more severe blow to the head than that producing a single linear fracture. A linear vault fracture substantially increases the risk of intracranial injury.

Depressed Fractures

Depressed skull fractures are usually caused by direct-impact injury with small blunt objects, such as a hammer or baseball bat. Most depressed skull fractures occur over the parietal or temporal regions. These fractures are clinically important because they predispose to significant underlying brain injury and to complications of head trauma, such as infection and seizures.

Basilar Fractures

Basilar fractures are linear fractures at the base of the skull, usually occurring through the temporal bone. Patients with basilar fractures are at risk for extra-axial hematomas because of the proximity of the fracture to the middle cerebral artery. Dural tears, resulting from a basilar skull fracture, may produce a communication among the subarachnoid space, paranasal sinuses, and middle ear. This offers a route for the introduction of infection into the cranial cavity and is suggested by a CSF leak. These fractures are the result of considerable impact force and are highly associated with an underlying brain injury.

Extra-Axial and Intra-Axial Intracranial Injuries

Extra-axial refers to injury or bleeding that occurs within the skull but outside of the brain tissue. Intra-axial injury or bleeding occurs within the brain tissue itself. Extra-axial intracranial lesions include epidural hematoma, subdural hematoma, traumatic subarachnoid hemorrhage, and subdural hygroma. Intra-axial intracranial lesions include traumatic axonal injury, cerebral and cerebellar contusions, and cerebral and cerebellar hematomas.

Extra-Axial Injury

Epidural Hematoma. An EDH is bleeding that occurs between the inner table of the skull and dura. Most EDHs result from a direct-impact injury that causes a forceful deformity of the skull. Often, a fracture occurs across the middle meningeal artery, or vein, or a dural sinus. The temporoparietal region is the most likely site for an EDH. The high arterial pressure of the bleeding vessel dissects the dura away from the skull, permitting hematoma formation.

EDH is primarily a disease of the young and accounts for up to 5% of all patients who have experienced TBI.[26] EDHs are rare in older adults and children younger than 2 years because of the close attachment of the dura to the skull in both patient populations.

Subdural Hematoma. A SDH is a hemorrhage that occurs between the dura and brain and is usually caused by acceleration-deceleration injuries. SDH occurs most commonly in patients with brain atrophy, such as alcoholic or older patients, because bridging vessels traverse greater distances than in patients with no atrophy. As a result, the vessels are more likely to rupture with rapid movement of the head. Once they are ruptured, blood can fill the potential space between the dura and arachnoid. SDH is much more common than EDH, occurring in up to 30% of patients with severe head trauma. The slow bleeding of venous structures delays the development of clinical signs and symptoms. As a result, the hematoma compresses the underlying brain tissue for prolonged periods and can cause significant tissue ischemia and damage. Approximately 20% of patients will present with a bilateral SDH. The prognosis of SDH does not entirely depend on the size of the hematoma but rather on the degree of brain injury caused by the pressure of the expanding hematoma on underlying tissue or by other intracranial injuries. Mortality is highest in older adults, patients who have a GCS score of 8 or less, and those with signs of acute herniation syndrome on initial ED presentation. Posterior fossa SDHs make up less than 1% of all reported SDHs. They are caused by occipital trauma that tears bridging vessels or venous sinuses and have a very poor prognosis.

Traumatic Subarachnoid Hemorrhage. A traumatic subarachnoid hemorrhage (SAH) is blood within the CSF and meningeal intima and probably results from tears of small subarachnoid vessels. Traumatic SAH is detected on the first CT scan in up to one-third of patients with severe TBI and ultimately is identified in almost 50% of patients with severe head trauma. It is therefore the most common CT scan abnormality seen after head trauma. Data from the National Traumatic Coma Data Bank have demonstrated a 60% unfavorable outcome in severely brain-injured patients when traumatic SAH is present compared with a 30% unfavorable outcome when it is not present. Traumatic SAH also is considered a risk factor for early mortality.[27]

Subdural Hygroma. A subdural hygroma (SDHG) is a collection of clear, xanthochromic blood-tinged fluid in the dural space. The pathogenesis of an SDHG is not certain. It may result from a tear in the arachnoid that permits CSF to escape into the dural space or effusions from injured vessels through areas of abnormal permeability in the meninges or in the underlying parenchyma. They may accumulate immediately after trauma or in a delayed manner.

Intra-Axial Injury

Diffuse Axonal Injury and Traumatic Axonal Injury. Prolonged traumatic coma not caused by mass lesions or ischemic insult is thought to result from diffuse axonal injury (DAI).[28] Although the term *diffuse axonal injury* has been widely adopted, the distribution of axonal injury is usually not diffuse but multifocal. Axonal injury occurs on a spectrum, with milder cases primarily localized. Furthermore, DAI has been used to describe axonal injury from nontraumatic causes in other neurologic conditions. Accordingly, the term *traumatic axonal injury* is preferred, particularly in milder cases. In more severe cases, when the axonal injury is more diffuse, the term *diffuse traumatic axonal injury* can be used.

In TAI, axons sustain a primary insult in which they are torn (axotomy) or stretched, and secondary insults lead to axonal swelling and disconnection and can lead to axon death (see Fig. 34.2).[29] Moreover, acute uncoupling of cerebral blood flow, metabolism, and apoptosis are thought to be the important factors linked to axonal cell death after TAI.[13,30]

Most patients with TAI present with persistent traumatic coma that begins immediately at the time of trauma; however, some patients may recover consciousness briefly before lapsing into prolonged coma. Because diagnostic studies cannot predict the extent of the axonal damage, the severity of the injury is determined by the clinical course. Clinical grades of diffuse TAI have been based on length of coma: (1) grade 1 (mild)—ccoma for 6 to 24 hours; (2) grade II (moderate)—coma for longer than 24 hours but not decerebrate; (3) grade III (severe)—coma for longer than 24 hours and decerebrate or flaccid. Currently, no early clinical or biomarker predictor exists that differentiates patients with mild, moderate, or severe diffuse TAI. Experimental laboratory data have indicated that neurons can partially repair and regenerate damaged axons.[29,31]

Cerebral Contusions. Contusions are bruises on the surface of the brain, usually caused by impact injury. Most often, contusions occur at the poles and inferior surfaces of the frontal and temporal lobes, where the brain comes into contact with bone protuberances in the base of the skull. If the contusion occurs on the same side as the impact injury, it is a coup injury; if it occurs on the opposite side, the contusion is a contrecoup injury. Contusions also often develop in the brain tissue that underlies a depressed skull fracture. Multiple areas of contused tissue may be produced with a single impact, often in association with other intracranial injuries. Contusions are produced when parenchymal blood vessels are damaged, resulting in scattered areas of petechial hemorrhage and subsequent edema. Contusions develop in the gray matter on the surface of the brain and taper into the white matter. Often, subarachnoid blood is found overlying the involved gyrus. With time, the associated hemorrhages and edema of a contusion can become widespread and serve as a nidus for hemorrhage or swelling, thus producing a local mass effect. Compression of the underlying tissue can cause local areas of ischemia, and tissue infarction is possible if the compression is significant and unrelieved. Eventually, these ischemic areas become necrotic, and cystic cavities form within them.

Intracerebral Hematoma. Intracerebral hematomas (ICHs) are formed deep within the brain tissue and are usually caused by shearing or tensile forces that mechanically stretch and tear deep small-caliber arterioles as the brain is propelled against irregular surfaces in the cranial vault. Resulting small petechial hemorrhages coalesce to form ICHs, with 85% in the frontal and temporal lobes. An ICH is often found in the presence of extra-axial hematomas and, in many patients multiple ICHs are present. Isolated ICHs may be detected in as many as 12% of all patients with severe head trauma.

Intracerebellar Hematoma. Primary traumatic intracerebellar hematomas are rare but can occur after a direct blow to the occipital area. Often, these patients have an associated skull fracture, posterior fossa EDH or SDH, or supratentorial contrecoup hematomas and contusions.

Primary and Secondary Brain Injuries

The acute clinical picture of the patient with TBI is dynamic and represents the sum of primary and secondary injury.

Primary Brain Injury

A primary brain injury is mechanical damage that occurs at the time of head trauma and includes brain lacerations, hemorrhages, contusions, and tissue avulsions. On the microscopic level, primary injury causes permanent mechanical cellular disruption and microvascular injury. Other than the evacuation of traumatic hematomas, no specific intervention exists to repair or reverse primary brain injury.

Following the primary injury, there is a cascade of events at the cellular and molecular level that continues for hours to days that contribute further to the brain injury. This secondary brain injury results from intracellular and extracellular derangements that lead to alterations in cell function and propagation of injury through processes such as depolarization, excitotoxicity, disruption of calcium homeostasis, free radical generation, BBB disruption, ischemic injury, edema formation, and intracranial hypertension.[32] Animal and human studies have revealed a complicated series of neurochemical, neuroanatomic, and neurophysiologic reactions after brain injury (Fig. 34.3). The cell has some compensatory mechanisms to protect itself from widespread damage, such as endogenous free radical scavengers and antioxidants. However, these systems are quickly overwhelmed, and the functional and structural integrity of the cell is threatened. Investigational agents aimed at specific steps in the destructive processes have suggested that some aspects of secondary brain injury may be reversed or modified. Multiple ongoing brain injury trials have been performed with numerous investigational therapeutic interventions; to date, none have proved useful in the clinical setting.[32,33]

Secondary Systemic Insults

The final neurologic outcome after head trauma is influenced by the extent and degree of secondary brain injury. In turn, the amount of secondary brain injury depends on certain premorbid and comorbid conditions, such as the age of the patient and trauma-related systemic events.[34] A primary goal in the emergency care of a head trauma patient is prevention or reduction of systemic conditions that are known to worsen outcome after TBI, such as hypotension, hypoxia, anemia, and hyperpyrexia.[34,35]

Hypotension. Defined as SBP less than 90 mm Hg, this has been found to have negative impact on severe brain injury outcome. Systemic hypotension reduces cerebral perfusion, thereby potentiating ischemia and infarction. Hypotension is

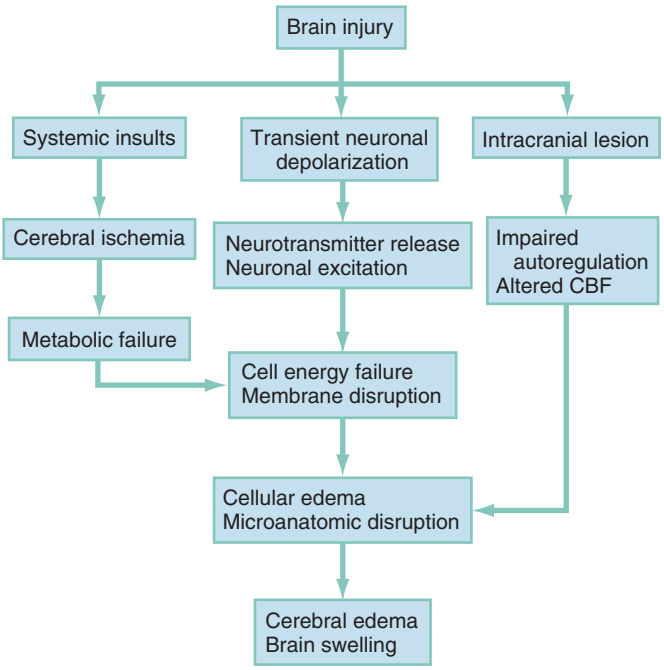

Fig. 34.3. Contributing events in the pathophysiology of secondary brain injury. *CBF,* Cerebral blood flow.

associated with a near-doubling of the mortality from TBI and worse outcomes for patients who survive.[10]

Hypoxia. Defined as a Po_2 less than 60 mm Hg, this occurs often in the brain-injured patient. Causes include the following: (1) transient or prolonged apnea caused by brainstem compression or injury after the traumatic event; (2) partial airway obstruction caused by blood, vomitus, or other debris in the airway of the traumatized patient; (3) injury to the chest wall that interferes with normal respiratory excursion; (4) pulmonary injury that reduces effective oxygenation; and (5) ineffective airway management, such as the inability to bag-valve-mask or intubate the patient in an effective or timely manner. When hypoxia is documented, the overall mortality from severe TBI may double.[10] Hyperoxia also is associated with worse outcome after traumatic brain injury.[36,37]

Hypocarbia and Hypercarbia. Hypocarbia ($Paco_2 \leq$ 35 mm Hg) and hypercarbia ($Paco_2 \geq 46$ mm Hg) are each associated with increased mortality following TBI. Hypercarbia causes cerebral vasodilation, with a resultant increase in cerebral edema and ICP, and thus is associated with a worsened neurologic outcome.[38,39] Hyperventilation to induce hypocarbia has been discredited for patients with elevated ICP; current patient management emphasizes maintenance of normal to slightly reduced $Paco_2$ levels.

Anemia. Anemia caused by blood loss can be detrimental to the head-injured patient by reducing the oxygen-carrying capacity of the blood, thus reducing the amount of necessary substrate delivered to the injured brain tissue. When anemia (hematocrit, 30%) occurs in patients with severe brain injury, the mortality rate increases.[34] Other potential reversible causes of systemic insult in brain injury include hypercarbia, coagulopathy, and seizures.[34]

Hyperpyrexia. Hyperpyrexia (core body temperature > 38.5° C [101.3° F]) is also correlated with worse outcomes after TBI, its magnitude and its duration seem to contribute. The exact mechanism whereby it causes damage likely involves increased metabolism in injured brain areas, thus recruiting blood flow, with a resultant increase in ICP.[34]

Altered Levels of Consciousness

Consciousness is the state of awareness of the self and environment, and it requires intact functioning of the cerebral cortices and reticular activating system (RAS) of the brainstem. A patient who has sustained TBI typically has an altered level of consciousness (LOC), but reversible conditions that can alter mental status, such as hypoxia, hypotension, or hypoglycemia, should be corrected as they are identified. Head trauma patients may be hypoxic from injury to respiratory centers or from concomitant pulmonary injury. Hypotension from other associated injuries can compromise CBF and affect consciousness. Global suppression may result from an intoxicant consumed before the injury, hypoglycemia, posttraumatic seizure (PTS), or postictal period after a seizure from any cause. With increasing ICP from brain swelling or an expanding mass lesion, brainstem compression and subsequent RAS compression can occur.

Cerebral Herniation Syndromes

Cerebral herniation occurs when increasing cranial volume and ICP overwhelm the natural compensatory capacities of the central nervous system (CNS; Fig. 34.4). When the signs of herniation syndrome are present, however, mortality approaches 100% without rapid implementation of temporizing emergency measures and definitive neurosurgical therapy.

Uncal Herniation

The most common clinically significant traumatic herniation syndrome is uncal herniation, a form of transtentorial herniation. Uncal herniation is often associated with traumatic extra-axial hematomas in the lateral middle fossa or the temporal lobe. As compression of the uncus begins, the third cranial nerve (CN) is compressed; anisocoria, ptosis, impaired extraocular movements, and a sluggish pupillary light reflex develop on the side ipsilateral to the expanding mass lesion. As the herniation progresses, compression of the ipsilateral oculomotor nerve eventually causes ipsilateral pupillary dilation and nonreactivity.

Initially in the uncal herniation process, motor examination findings can be normal, but contralateral Babinski responses develop early. Contralateral hemiparesis develops as the ipsilateral peduncle is compressed against the tentorium. With continued progression of the herniation, bilateral decerebrate posturing eventually occurs; decorticate posturing is not always seen with the uncal herniation syndrome.

In a certain percentage of TBI patients, the contralateral cerebral peduncle is forced against the opposite edge of the tentorial hiatus. Hemiparesis is then detected ipsilateral to the dilated pupil and mass lesion. This is termed *Kernohan's notch syndrome* and causes false-localizing motor findings. As uncal herniation progresses, direct brainstem compression causes additional alterations in the LOC, respiratory pattern, and cardiovascular system. Mental status changes may initially be subtle, such as agitation, restlessness, or confusion, but soon lethargy occurs, with progression to frank coma. The patient's respiratory pattern may initially be normal, followed by sustained hyperventilation. With continued brainstem compression, an ataxic respiratory pattern develops. The patient's hemodynamic status may change, with rapid fluctuations in blood pressure and cardiac conduction. Herniation that is uncontrolled progresses rapidly to brainstem failure, cardiovascular collapse, and death.

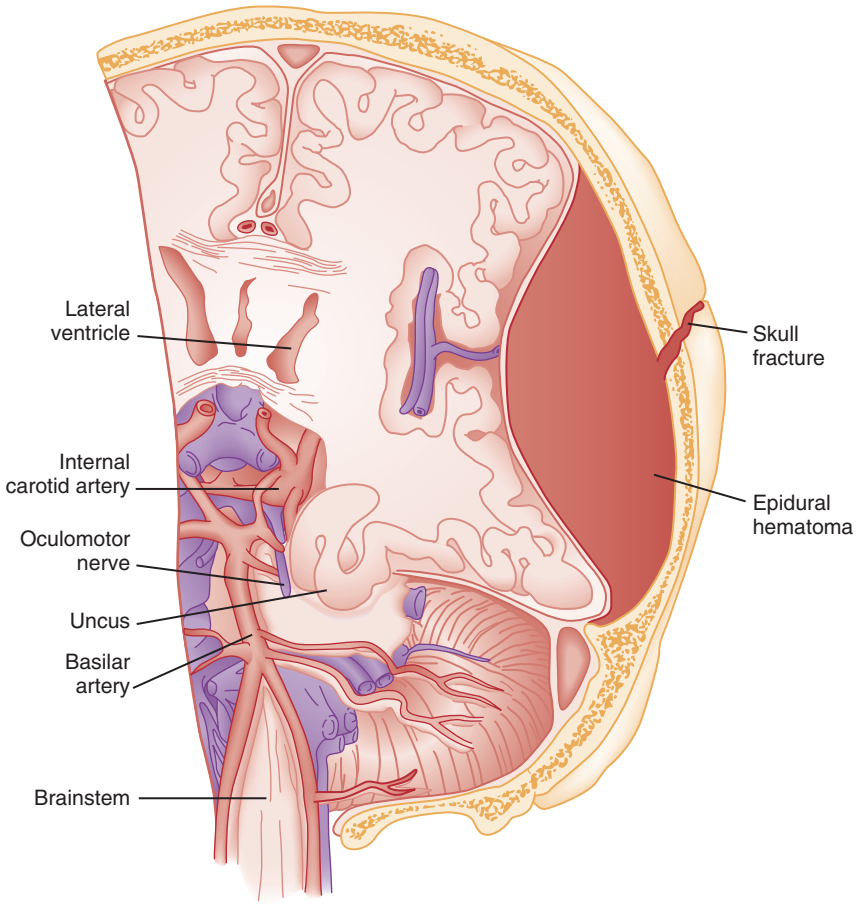

Fig. 34.4. Anterior view of transtentorial herniation caused by large epidural hematoma. A skull fracture overlies the hematoma. (From Rockswold GL: Head injury. In Tintinalli JE, et al editors: Emergency medicine, New York, 1992, McGraw-Hill, p 915.)

Central Transtentorial Herniation

Less common than uncal transtentorial herniation, the central transtentorial herniation is demonstrated by rostrocaudal neurologic deterioration caused by an expanding lesion at the vertex or frontal or occipital pole of the brain. Clinical deterioration occurs as bilateral central pressure is exerted on the brain from above. The initial clinical manifestation may be a subtle change in mental status or decreased LOC, bilateral motor weakness, and pinpoint pupils (2 mm). Light reflexes are still present but are often difficult to detect. Muscle tone is increased bilaterally, and bilateral Babinski's signs may be present. As central herniation progresses, both pupils become midpoint and lose light responsiveness. Respiratory patterns are affected, and sustained hyperventilation may occur. Motor tone increases. Decorticate posturing is elicited by noxious stimuli. This progresses to bilateral decorticate and then spontaneous decerebrate posturing. Respiratory patterns initially include yawns and sighs and progress to sustained tachypnea, followed by shallow slow and irregular breaths immediately before respiratory arrest.

Cerebellotonsillar Herniation

Cerebellotonsillar herniation occurs when the cerebellar tonsils herniate downward through the foramen magnum. This is usually the result of a cerebellar mass or large central vertex mass causing the rapid displacement of the entire brainstem. Clinically, patients demonstrate sudden respiratory and cardiovascular collapse as the medulla is compressed. Pinpoint pupils are noted. Flaccid quadriplegia is the most common motor presentation because of bilateral compression of the corticospinal tracts. Although mortality is high, timely neurointensive care and neurosurgical intervention results in recovery to a minimal or moderate level of disability in over 50% of patients.

Upward Transtentorial Herniation

Upward transtentorial herniation occasionally occurs as a result of an expanding posterior fossa lesion. The LOC declines rapidly. These patients may have pinpoint pupils from compression of the pons. Downward conjugate gaze is accompanied by the absence of vertical eye movements.

MODERATE AND SEVERE TRAUMATIC BRAIN INJURY

Clinical Features and History

Although the history may be delayed by the need for emergent resuscitation and stabilization, details regarding the mechanism of injury, circumstances surrounding the injury, and any concomitant drug or alcohol use should be solicited. The patient, prehospital providers, or any witnesses should be queried as to loss of consciousness or seizure activity. The patient should be asked about recall of the incident and the time periods before and after and about any symptoms, including severe headache, nausea, vomiting, or amnesia. The past medical history should be obtained, with particular attention to coagulopathies such as hemophilia. In

addition, the patient's medications, particularly anticoagulant or antiplatelet agents, should be determined. If there has been a change in the patient's GCS score, this should be noted.

The patient's current LOC, as well as that immediately before and after the injury and at the arrival of first responders, should be determined. Worsening mental status or deteriorating GCS scores since the injury indicate the presence of moderate to severe injury. Witnessed seizures or apnea should be reported. If the patient is now awake but was unconscious at some point, it should be determined if the patient has returned to baseline mental status.

Common Presentations of Specific Lesions

Epidural Hematoma

The classic presentation of an EDH is described as head trauma producing a decreased LOC followed by a so-called lucid interval. Although the patient's consciousness is less decreased during the lucid interval, a completely normal mental status may not return before a second episode of decreased consciousness occurs. The lucid interval is not pathognomonic for an EDH and occurs in patients who sustain other expanding mass lesions. Approximately 47% of patients with EDHs present classically. The development of symptoms and signs of EDH is entirely dependent on how quickly the EDH is developing within the cranial vault. Patients with an EDH often complain of a severe headache, sleepiness, dizziness, nausea, and vomiting. A small EDH may remain asymptomatic, but this is rare.

If the EDH is rapidly detected and evacuated, the functional outcome is excellent. Because of their rapid formation, EDHs from arterial bleeding are usually detected within hours after injury and often earlier in children. EDHs that develop from a dural sinus tear develop more slowly, and clinical manifestations may be delayed, with resultant delays in detection.

A posterior fossa EDH is the result of direct occipital trauma resulting in a skull fracture that disrupts a venous sinus is the usual cause, and most patients have external evidence of occipital injury. Most patients become symptomatic within 24 hours after injury, with complaints of headache, nausea, vomiting, and nuchal rigidity. Most patients eventually have a decreased LOC.

Subdural Hematoma

The patient's clinical presentation depends on the amount of brain injury sustained at the time of trauma and the rate of SDH expansion. If the patient with an SDH was rendered unconscious at the time of trauma, the prognosis is poor; these patients often have concurrent TAI. The signs and symptoms after injury that produces an SDH are initially related to the other intracranial injuries that may have been sustained and then to the slow expansion of the SDH. SDHs are classified by the time to clinical presentation. Acute SDHs are symptomatic within 24 hours after trauma. Patients with acute SDHs often have a decreased LOC. Most patients with an SDH have a GCS score less than 8. Approximately 12% to 38% of patients will have a lucid period at some point in their presentation. The overall mortality of patients who have an SDH and require surgical intervention is 40% to 60%.

A chronic SDH becomes symptomatic 2 weeks or more after trauma. The signs and symptoms may be very subtle or nonspecific, but many patients demonstrate unilateral weakness or hemiparesis. Patients with unilateral chronic SDH have more frequent occurrence of hemiparesis than those with bilateral chronic SDH.[40] Most report an altered LOC, but some patients are unable to recall the trauma or describe only a minor injury. A chronic SDH may have initially been a small asymptomatic SDH that eventually expanded owing to a combination of recurrent hemorrhage and escape of plasma into the hematoma. At some point, a critical mass is reached, and the chronic SDH becomes symptomatic.

Clinical manifestations of posterior SDH vary but usually include nausea, vomiting, headache, and decreased LOC. Occasionally, CN palsies may be found, as well as nuchal rigidity, cerebellar signs and symptoms, and papilledema.

Traumatic Subarachnoid Hemorrhage

An increased incidence of skull fractures and contusions is found in patients with a traumatic SAH (tSAH) compared with patients with no tSAH. The amount of blood within the tSAH correlates directly with the outcome and inversely with the presenting GCS score. Patients may complain of headache and photophobia.

Subdural Hygroma

Clinically, an SDHG cannot be distinguished from other mass lesions. Often, patients have a decreased LOC or focal motor deficits. They may complain of headaches, nausea, and vomiting. The ICP can increase because of the mass effect, and signs of increased ICP may be present.

Traumatic Axonal Injury

The duration of loss of consciousness or coma following injury is directly related to the extent of axonal pathology in the brainstem.[28] Even with extensive axonal pathology in the white matter, there may be little or no loss of consciousness if the brainstem is relatively spared. Therefore, it appears that the distribution, rather than the overall extent, of axonal pathology is important in determining consciousness immediately following TBI.[41]

Cerebral Contusion

The clinical presentation of patients with contusions is frequently delayed. They may have sustained only a brief loss of consciousness, but the duration of posttraumatic confusion and obtundation may be prolonged. If contusions occur near the sensorimotor cortex, focal neurologic deficits may be present. Many patients with significant contusions make uneventful recoveries, but contusions may cause significant neurologic problems, including increased ICP, PTSs, and focal deficits.

Intracerebral Hematoma

The clinical effects of intracerebral hematomas depend on size and location and whether the bleeding is continuing. ICHs have been reported with all degrees of severity of head trauma. More than 50% of patients with ICH sustain loss of consciousness at the time of impact. The patient's subsequent LOC depends on the severity of the impact and coexisting lesions. Combined with contusions, other concurrent lesions, and subsequent perilesion edema, an ICH can produce substantial mass effects and precipitate a herniation syndrome (Fig. 34.5).

Traumatic Intracerebellar Hematoma

The clinical presentation of an isolated traumatic cerebellar hematoma is similar to that of other posterior lesions. When other traumatic lesions are present, the picture may be confusing.

Physical Examination

In the setting of head trauma and suspected brain injury, management should be guided by the principles of trauma resuscitation.

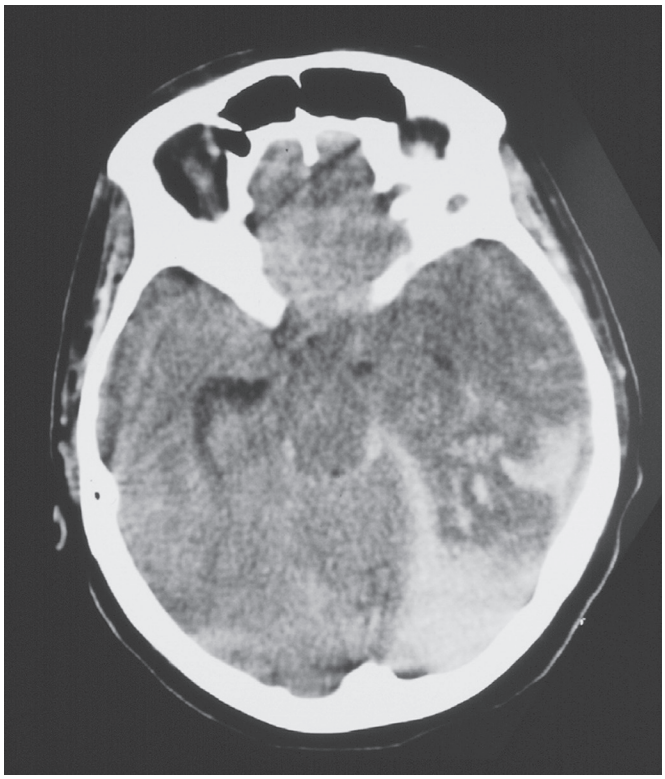

Fig. 34.5. Non–contrast-enhanced computed tomography (CT) scan of intracerebral hematoma and contusion in the left occipital region. The scan also shows layering of a tentorial subdural hematoma. Mass effect and early uncal herniation are visible as well.

BOX 34.2

Clinical Characteristics of Basilar Skull Fractures

Blood in ear canal
Hemotympanum
Rhinorrhea
Otorrhea
Battle's sign (retroauricular hematoma)
Raccoon sign (periorbital ecchymosis)
Cranial nerve deficits
Facial paralysis
Decreased auditory acuity
Dizziness
Tinnitus
Nystagmus

A primary survey focusing on airway, breathing, and hemorrhage control should be performed expeditiously. After immediate life threats are adequately addressed, a secondary survey should evaluate for underlying head injury, brain injury, and neurologic compromise.

The head and neck should be carefully examined for external signs of trauma that may have also produced an underlying TBI. A scalp laceration, contusion, abrasion, or avulsion may overlie a depressed skull fracture. The clinical examination for a depressed skull fracture may be misleading. The mobility of the scalp can result in nonalignment of the fracture with an overlying scalp laceration. As a result, the skull underlying the laceration may be normal, with the depressed area several centimeters away. Scalp swelling may interfere with physical examination findings and hide any palpable bone defects. The signs and symptoms of a depressed skull fracture depend on the depth of depression of the free bone piece. About 25% of patients sustaining a depressed skull fracture report loss of consciousness. Neurologic deficits may be present, depending on the extent of underlying brain tissue injury.

Basilar skull fractures are often diagnosed by the clinical examination (Box 34.2), and the physical examination should evaluate for such signs as hemotympanum, periauricular or periorbital ecchymoses, and clear otorrhea or rhinorrhea. In the case of an impalement injury, the penetrating object should be left in place to be removed at surgery. Patients with basilar fractures are at risk for extra-axial hematomas because of the proximity of the fracture to the middle cerebral artery. Basilar fractures can compress and entrap the CNs that pass through the basal foramina, dislocate the bones of the auricular chain, and disrupt the otic canal or cavernous sinuses with subsequent injury to CNs III, IV, and V. Fractures of the sphenoid bone can disrupt the intracavernous internal carotid artery, creating the potential for the formation of pseudoaneurysms or carotid venous fistulae. The diagnosis of a basilar skull fracture is based on associated clinical signs and symptoms (see Box 34.2).

The percentage of concurrent cervical spine injury in patients with severe head trauma ranges up to nearly 20%.[42] Often, other spinal regions are also injured. The neck should be evaluated for evidence of a cervical spine fracture. Carotid artery dissections caused by a hyperflexion-extension neck injury can occasionally be detected by auscultation of a carotid bruit. In these patients, a careful neurologic examination should assess for subtle asymmetry between the carotid arteries. Finally, all patients should undergo a thorough secondary evaluation after initial stabilization, evaluating for additional injuries, including an evaluation for spinal cord pathology.

Acute Neurologic Examination

General. The goals of the acute neurologic assessment of head trauma patients include detection of life-threatening injuries and identification of neurologic changes in the immediate post-trauma period. An accurate neurologic assessment in this period serves as a basis for comparison in subsequent examinations. An efficient neurologic examination in the emergency setting includes evaluation of mental status, GCS score, pupillary size and responsiveness, and motor strength and symmetry. If a formal GCS measure is not possible or is difficult because of comorbid confounders, the patient's mental status should be described in as much detail as possible. Declining mental status after head trauma suggests increasing ICP from an expanding mass lesion or worsening cerebral edema, which may rapidly become life-threatening. The strongest predictors of outcome following moderate and severe TBI are age, pupillary reactivity, and GCS motor score. Additional predictors include CT characteristics, hypotension, hypoxia, laboratory parameters (eg, glucose, hemoglobin levels), and extracranial injuries.[43]

Glasgow Coma Scale. The GCS is a 15-point scale used in an attempt to quantify the patient's LOC and is an objective method of following the patient's neurologic status (Table 34.2). It was originally developed during a time when CT scanning was not available to communicate changes in neurologic status in comatose patients with TBI (Fig. 34.6). The score assigns points based on the patients best eye opening (spontaneous opening = 4 to no response = 1), motor response (obeys commands = 6 to no response = 1), and verbal response (oriented = 5 to no response = 1). Due to its ease of use, it has been adopted in the routine

TABLE 34.2

Glasgow Coma Scale

RESPONSE	SCORE	SIGNIFICANCE
EYE OPENING		
Spontaneously	4	Reticular activating system intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
No eye opening	1	Does not open eyes to any stimuli
VERBAL STIMULI		
Oriented, converses	5	Relatively intact CNS, aware of self and environment
Disoriented, converses	4	Well-articulated, organized, but disoriented
Inappropriate words	3	Random exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No verbal response	1	No response or intubated
MOTOR RESPONSE		
Obeys verbal commands	6	Readily moves limbs when told to
Localizes to painful stimuli	5	Moves limb in an effort to remove painful stimuli
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decerebrate rigidity
No motor response	1	Hypotonia, flaccid—suggests loss of medullary function or concomitant spinal cord injury

CNS, Central nervous system.

assessment of all trauma patients, including those with MTBI who are not comatose.[5] However, the GCS score can reflect impairment from conditions other than brain injury, such as distracting injuries, intoxication from drugs and alcohol, hypoxemia, and sedative medications. Furthermore, patients can deteriorate from an expanding intracranial hematoma after what appears clinically to be a mild brain injury. Although TBI is often categorized into mild, moderate, and severe based on the GCS score, it really represents a spectrum of injury.

Pupillary Examination. An evaluation of the patient's pupil size and responsiveness is performed early in the initial assessment of the head-injured patient. Pupillary asymmetry, the loss of the light reflex, or a dilated pupil suggests herniation syndrome as increasing pressure on the CN III results in compromise of the parasympathetic fibers and pupillary dilation on the affected side. However, use of the pupillary examination for localization of an intracranial lesion is neither sensitive nor specific. Further, traumatic mydriasis, resulting from direct injury to the eye and periorbital structures, may confuse the assessment of the pupillary responsiveness. As with the GCS score, a change in pupillary response is more indicative of intracranial pathology than the initial findings.

Motor Examination. The patient's acute motor examination assesses for strength and symmetry. Paralysis obscures involuntary reflexes; attempts should be made to perform the motor examination before paralytic agents are given. If the patient is not cooperative or is comatose, motor movement should be elicited by the application of noxious stimuli. Any movement should be recorded. Voluntary purposeful movement must be distinguished from abnormal motor posturing.

Decorticate posturing is abnormal flexion of the upper extremity and extension of the lower extremity. The arm, wrist, and elbow slowly flex, and the arm is adducted. The leg extends and rotates internally, with plantar flexion of the foot. Decorticate posturing implies injury above the midbrain. Decerebrate posturing is the result of a more caudal injury, and therefore is associated with a worse prognosis. The rate of decerebrate posturing increases significantly in the presence of midbrain lesions.[44] The arms extend abnormally and become adducted. The wrist and fingers are flexed, and the entire arm is internally rotated at the shoulder. The neck undergoes abnormal extension, and the teeth may become clenched. The leg is internally rotated and extended, and the feet and toes are plantar-flexed.

Brainstem Function. In the acute setting, brainstem activity is assessed by the patient's respiratory pattern, pupillary size, and eye movements. The oculocephalic response (doll's eyes maneuver) tests the integrity of the pontine gaze centers. This response should not be elicited until cervical spine fractures have been ruled out. The oculovestibular response (cold water calorics) also permits assessment of the brainstem. Comatose patients no longer demonstrate nystagmus when cold water is placed in the ear canal; the only response is tonic deviation of the eyes toward the instilled cold water. This response is dampened by cerumen or blood in the patient's ear canal, and the tympanic membrane needs to be intact for this test to be performed.

In the severely head-injured patient, the CN examination is often limited to the pupillary responses (CN III), gag reflex (CNs IX and X), and corneal reflex (CNs V and VII). Facial symmetry (CN VII) can sometimes be assessed if the patient grimaces with noxious stimuli. In patients who are awake and cooperative, a formal CN examination should be performed.

Differential Diagnosis

In the context of trauma, conditions presenting with an altered LOC include seizures and associated postictal state, intoxication with alcohol or drugs, and systemic trauma resulting in hypoxemia or hypoperfusion. A patient's mental status may also be impaired by sedatives or motor function may be altered by neuromuscular blocking agents administered prior to ED arrival. However, intra-axial and extra-axial lesions may cause alterations in mental state and may coexist with other traumatic injuries. Head trauma may result in confounding injuries to other parts of the head or neck, including skull or facial bone fractures, cervical spine or spinal cord injuries, eye injuries, otolaryngeal injuries, and damage to blood vessels within the neck. Although a GCS score of 15 does not exclude the possibility of brain injury, a decreasing GCS score suggests an expanding intracranial lesion, which may be intracerebral or subdural or epidural. Signs include worsening headache, focal neurologic signs, confusion, and lethargy, which may progress to coma. Presentation of a subdural hemorrhage may be acute, subacute, or chronic. Epidural or intracerebral hemorrhages have an acute abrupt presentation, which may be delayed by minutes to hours from the initial injury. Patients with an epidural hemorrhage may have a lucid interval following a brief loss of consciousness or period of confusion. A skull fracture may be accompanied by underlying traumatic pathology, including brain contusions, dural tears, and vascular trauma. Given the proximity of the middle meningeal artery to the temporal bones, consider extra-axial hematomas (especially EDH) if there are signs of a basilar skull fracture. Decorticate posturing implies injury above

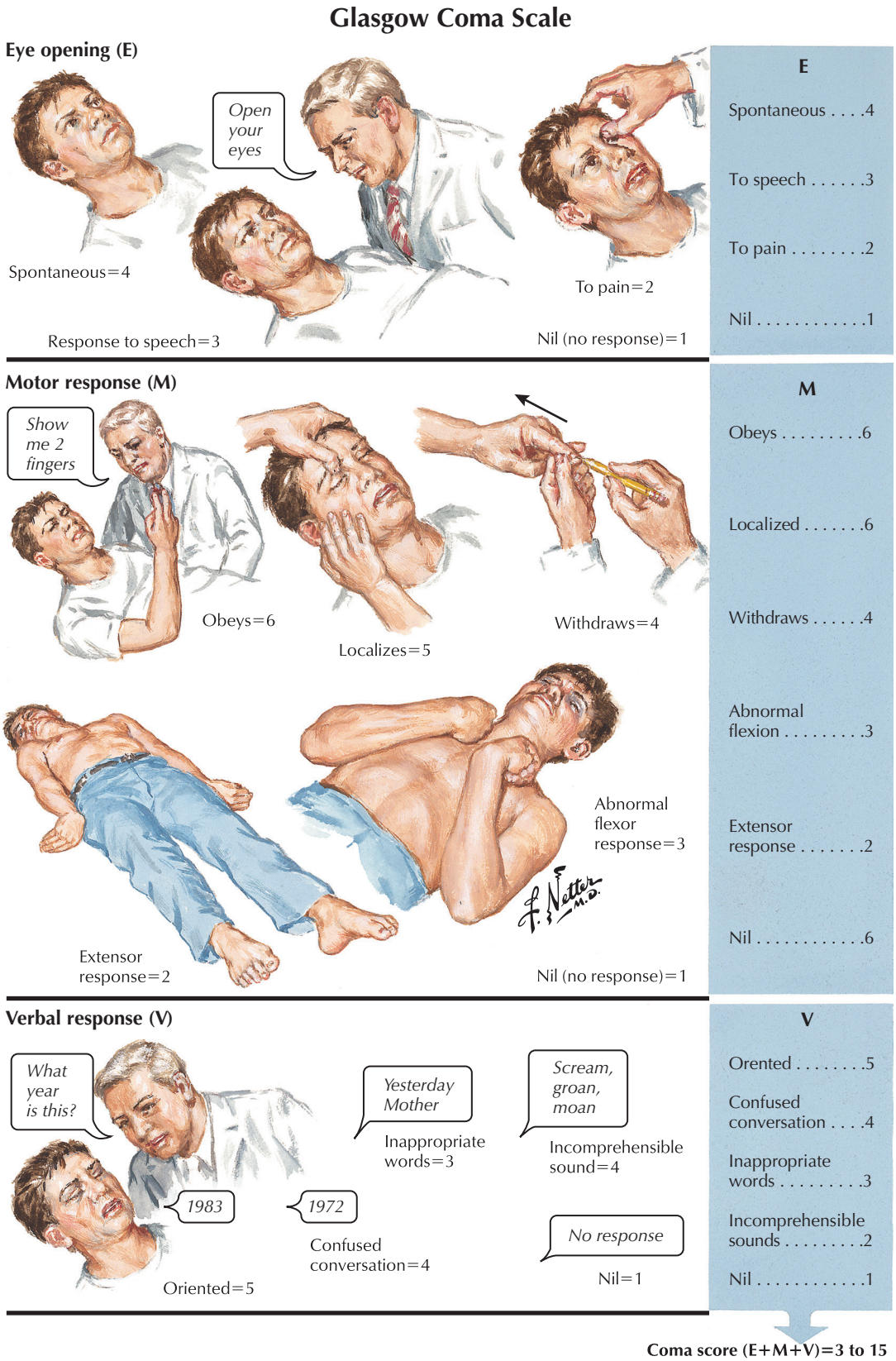

Fig. 34.6. How to calculate a Glasgow Coma Scale (GCS) score. (Copyright 2016 Elsevier Inc. All rights reserved. www.netterimages.com.)

the midbrain, whereas decerebrate posturing is suggestive of a midbrain lesion. Pupil inequality and unilateral motor deficits may help localize a lesion. In vulnerable populations, consider nonaccidental trauma in all patients with brain injury. Furthermore, consider traumatic brain injury in all patients with head trauma and advanced age or those on anticoagulant or antiplatelet agents, regardless of symptoms. With age, the brain atrophies and creates more space within the cranial vault for blood to accumulate, so older adults can have significant hemorrhage and not show signs of deterioration.

Diagnostic Testing

Laboratory Tests

Routine laboratory tests are generally not needed for patients with isolated mild TBI in the acute setting, except for a bedside glucose test in patients with altered mental status and determination of the blood alcohol level in patients suspected of alcohol intoxication and head trauma. Suspicion of a systemic insult as the cause of the head trauma, such as when a diabetic patient sustains a MVC after losing consciousness from hypoglycemia, warrants directed testing for culprit conditions. Coagulation studies are indicated in patients with coagulopathies (eg, hemophilia, Von Willebrand disease), suspected liver disease, and those on anticoagulants. Ancillary laboratory tests that may provide useful information in the subsequent management of the patient include a urine toxicology screen, blood alcohol level, complete blood count, and electrolyte levels. In severe TBI patients, coagulation parameters (eg, platelet count) can indicate a worse prognosis.[45]

Neuroimaging

Skull Radiography. Skull radiography after head trauma rarely is indicated and has long been replaced by cranial computed tomography (CT), which is the cornerstone of imaging for acute head trauma. Although patients with clinical signs of skull fracture have a substantially increased incidence of intracranial lesions, numerous studies have shown that skull radiographs are neither sensitive nor specific for intracranial injury. When the clinical examination shows evidence of skull fractures, CT should be performed. Although plain skull radiographs were used in the past to localize missile fragments or ascertain penetration of the skull, CT also is the radiologic test of choice for penetrating head trauma.[46] The bone windows of the CT scan can detect skull fractures (including basilar fractures). CT defines the precise location of the missile, its intracranial path, the presence of bone or missile fragments, extra-axial or intracerebral blood collections or other traumatic lesions, and pneumocephalus. Skull radiographs for adults with MTBI are not recommended.

Computed Tomography. Noncontrasted CT of the head is the diagnostic standard for identifying intracranial injury in the ED. This scan delineates acute intra-axial and extra-axial bleeding, cerebral swelling, ischemic infarction caused by hypoxia after trauma, evidence of increased ICP, and pneumocephalus. It is sensitive for demonstrating mass effect, ventricular size and configuration, bone injuries, and acute hemorrhage, regardless of location (ie, parenchymal, subarachnoid, subdural, or epidural spaces).[47] Follow-up CT should be performed if there is any clinical deterioration.

Pneumocephalus. This is often associated with missile wounds that penetrate the sinuses but can be caused by free air sucked into the penetration cavity behind the projectile. All tangential GSWs should be evaluated with a head CT scan secondary to the high incidence of associated intracranial injury.[25] Angiography may be indicated to better discern location referable to key vascular structures. Pneumocephalus is also associated with open skull fractures.

Epidural Hematoma. On CT scan, an EDH appears hyperdense, biconvex, ovoid, and lenticular. The EDH does not usually extend beyond the dural attachments at the suture lines. The margins are sharply defined, and the hematoma usually bulges inward toward the brain (Fig. 34.7). EDHs of mixed density on CT may be actively bleeding. The temporoparietal region is the most likely site for an EDH. An EDH is usually unilateral, and 20% of patients have other intracranial lesions, usually SDHs or contusions. The deterioration of a patient who has an EDH from arterial bleeding can be rapid and dramatic.

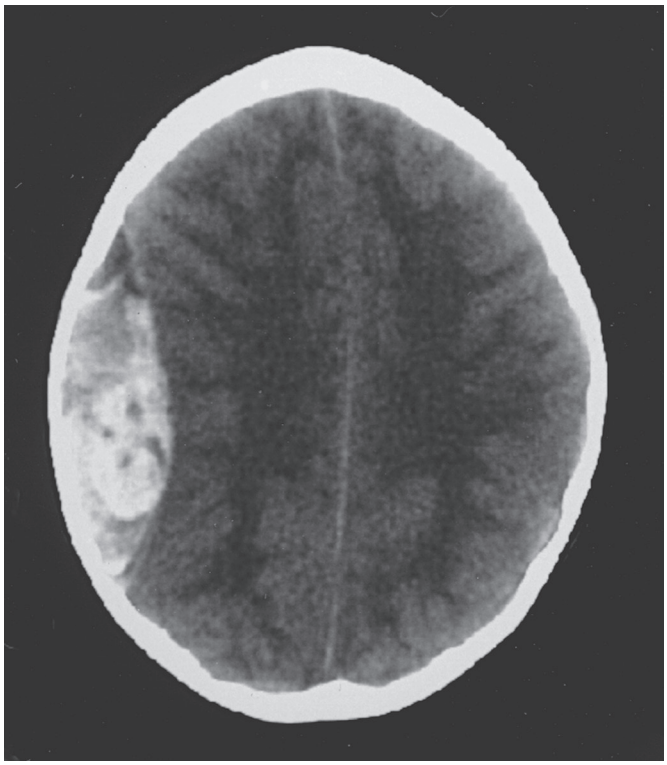

Fig. 34.7. Non–contrast-enhanced CT scan of acute epidural hematoma at the level of right midconvexity. There is an associated mass effect and moderate midline shift.

A posterior fossa EDH is the most common traumatic mass lesion of the posterior fossa and accounts for 5% of EDHs. On CT scan, a posterior fossa EDH looks similar to other EDHs, but may cross the midline and extend above the tentorium to the supratentorial compartment (Fig. 34.8).

Subdural Hematoma. Unlike EDHs, SDHs often extend beyond the suture lines (Fig. 34.9). An SDH may follow the contour of the tentorium and be detected within the interhemispheric fissure (Fig. 34.10). Many patients with an acute SDH also show CT evidence of intracerebral lesions contralateral to the SDH. A subacute SDH is symptomatic between 24 hours and 2 weeks after injury. It may appear hypodense or isodense on CT scans. Contrast increases the detection of isodense lesions. Patients complain of a headache, altered mental status, or focal deficits.

On a CT scan, a chronic SDH may appear isodense or hypodense to brain parenchyma. Indirect evidence of the lesion includes a midline shift, effacement of the ipsilateral cortical sulci, and ventricular compression. Contrast may increase the likelihood of identifying a chronic SDH that has become isodense. On a CT scan, blood of various ages is seen as a mixed-density lesion. On a magnetic resonance imaging (MRI) scan, a chronic SDH appears hyperdense.

Posterior fossa SDHs are caused by occipital trauma and, on a CT scan, they do not cross the midline or extend above the tentorium. The outcome of a posterior SDH is very poor.

Traumatic Subarachnoid Hemorrhage. A noncontrast CT scan allows the diagnosis to be made, with increased density noted within the basilar cisterns. Blood can also be seen within the interhemispheric fissures and sulci. The amount of blood within the tSAH correlates directly with the outcome and inversely with the presenting GCS score.

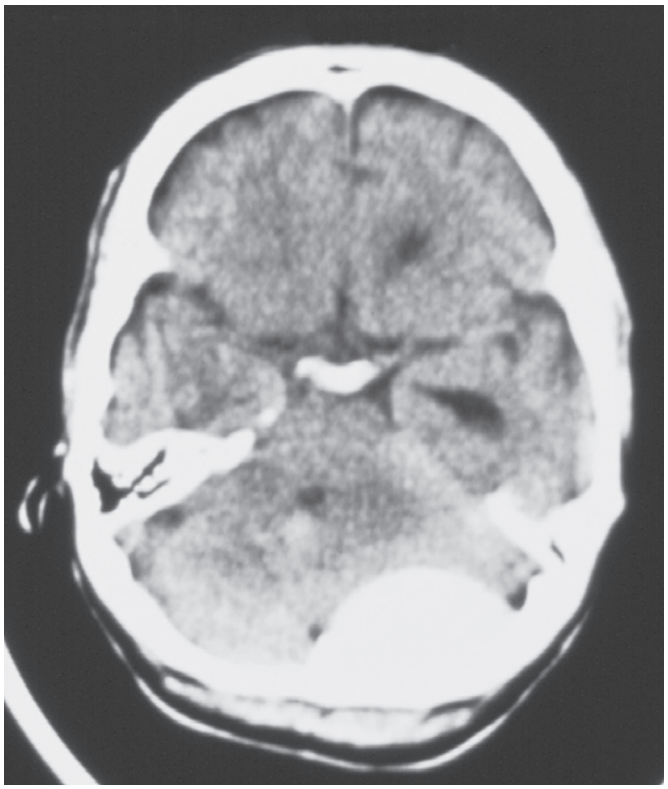

Fig. 34.8. Non–contrast-enhanced CT scan of large left posterior fossa, epidural hematoma. The size of the lesion at this high level suggests that it crosses into the supratentorial compartment. This lesion is often associated with occipital bone fracture that disrupts transverse sinus.

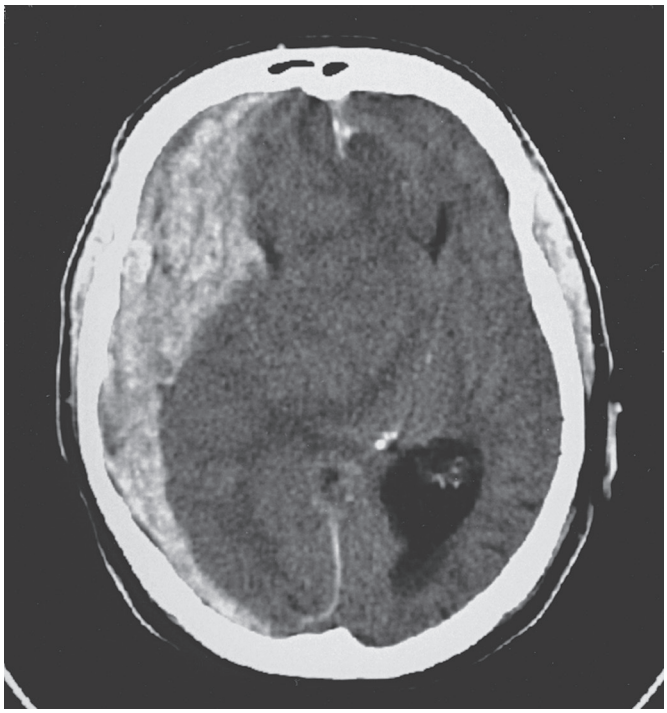

Fig. 34.9. Non–contrast-enhanced CT scan of acute right temporal subdural hematoma. There is acute bleeding and delayed bleeding, which explains the mixed density. Mass effect is large, with a midline shift measuring approximately 2.7 cm right to left. The right lateral ventricle has been obliterated.

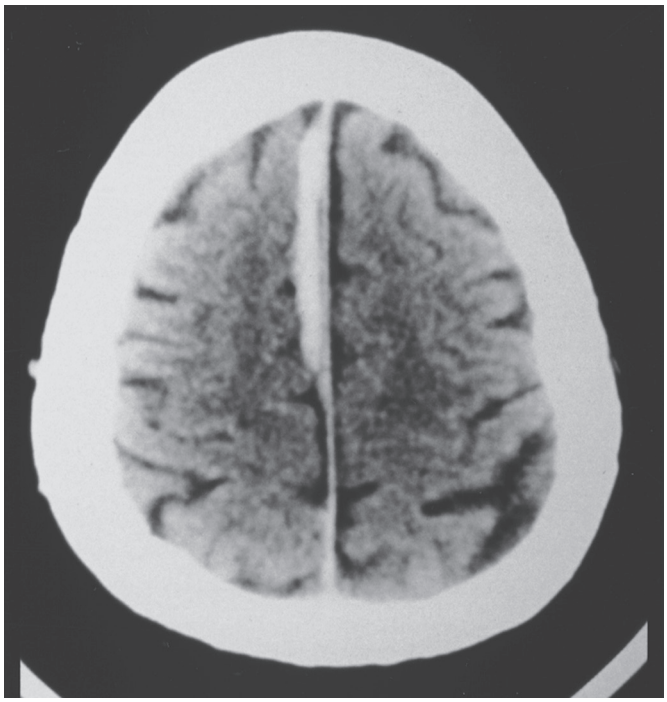

Fig. 34.10. Non–contrast-enhanced CT scan of interhemispheric acute subdural hematoma.

Subdural Hygroma. On CT, SDHGs appear crescent shaped in the extra-axial space; the density is the same as that of CSF. Bilateral SDHGs are common.

Diffuse Axonal Injury/ Traumatic Axonal Injury. Diffuse TAI is the most common CT finding after severe head trauma, estimated to occur in over 50% of all comatose head trauma patients. However, in milder cases, there is no specific acute focal traumatic lesion noted on a head CT scan or on structural MRI scan. Occasionally, small petechial hemorrhages in proximity to the third ventricle and within the white matter of the corpus callosum or internal capsule of the brainstem are detected.[28] An intraventricular hemorrhage on the initial CT scan has been reported to be an early predictor of DAI lesions in the corpus callosum on MRI.[48] Although histopathologic examination of postmortem brain tissue[28] is the gold standard for diagnosing TAI, advanced MRI neuroimaging techniques, such as diffusion tensor imaging (DTI), may help assess white matter integrity (Fig. 34.11).[49]

Cerebral Contusions. Non–contrast-enhanced CT is the best diagnostic test to discover contusions in the early posttraumatic period. These appear heterogeneous and irregular because of mixed regions of hemorrhage, necrosis, and infarction. Often, the surrounding edematous tissue appears hypodense. By posttrauma days 3 and 4, the blood located within the contusions has begun to degrade, and structural MRI becomes more useful.

Intracerebral Hematoma. An intracerebral hematoma may be detected on the first CT scan immediately after injury but often is not seen for several hours or days. Unlike contusions, ICHs are usually deep in the brain tissue and often become well demarcated over time. On CT scan, an ICH appears as a well-defined hyperdense homogeneous area of hemorrhage (Fig. 34.12).

Traumatic Intracerebellar Hematoma. Often, these patients have an associated skull fracture, posterior fossa EDH or SDH, or supratentorial contrecoup hematomas and contusions.

Cerebral Edema. On CT scans, diffuse edema manifests as bilateral compression of the ventricles, loss of definition of the cortical sulci, or effacement of the basal cisterns (Fig. 34.13). Focal

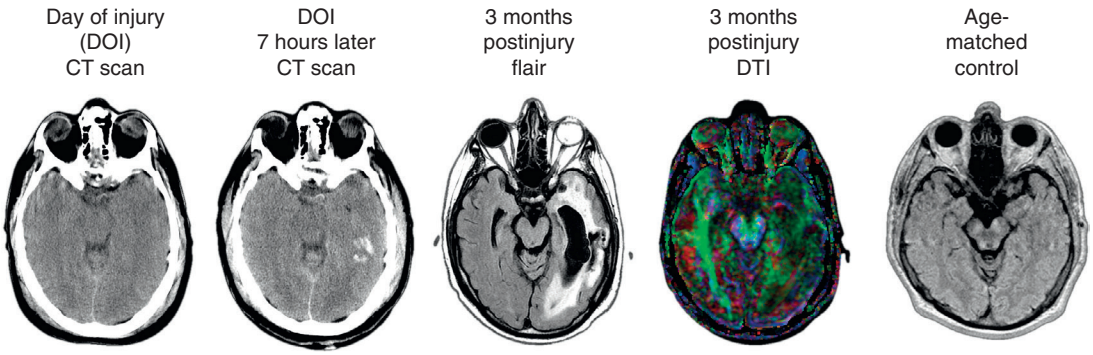

Fig. 34.11. Sequential scan findings in severe traumatic brain injury (TBI) with traumatic axonal injury (TAI). In the ED, on initial day of injury (DOI) CT imaging, the scan on the *far left* shows primarily generalized edema but, by 7 hours, distinct intraparenchymal hemorrhages appear, particularly within the left temporal lobe. By 3 months postinjury, TAI and intraparenchymal hemorrhages result in massive degenerative effects, reflected in temporal horn dilation and markedly abnormal white matter signal differences throughout the temporal and occipital lobes on the fluid-attenuated inversion recovery (FLAIR) sequence. Temporal horn dilation is also evident in the right temporal lobe as well, reflective of the generalized atrophy. The loss of white matter integrity is more distinctly observed using diffusion tensor imaging (DTI), where there is no coherent direction noted in the left temporal region; even though the right temporal lobe exhibits atrophic changes, the inferior occipitotemporal fasciculus (*arrow*) is distinctly visible. The control MRI scan is a T1 image showing symmetric temporal lobe morphology with the normal slitlike appearance of the temporal horns. (Adapted from Bigler ED, Maxwell WL: Neuropathology of mild traumatic brain injury: relationship to neuroimaging findings. Brain Imaging Behav 6:108–136, 2012.)

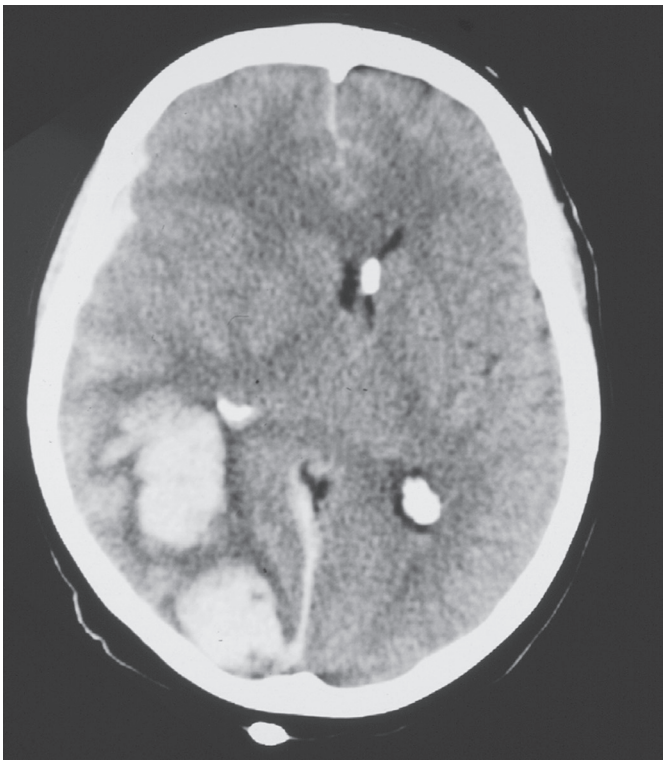

Fig. 34.12. Non–contrast-enhanced CT scan of right occipital and temporal intracerebral hematomas, surrounded by mild edema and hemorrhagic contusion. A small, interhemispheric, subdural hematoma is visible in the posterior interhemispheric fissure. Midline shift is obvious. A ventriculostomy has been placed and is visible as high-density image within the ventricles.

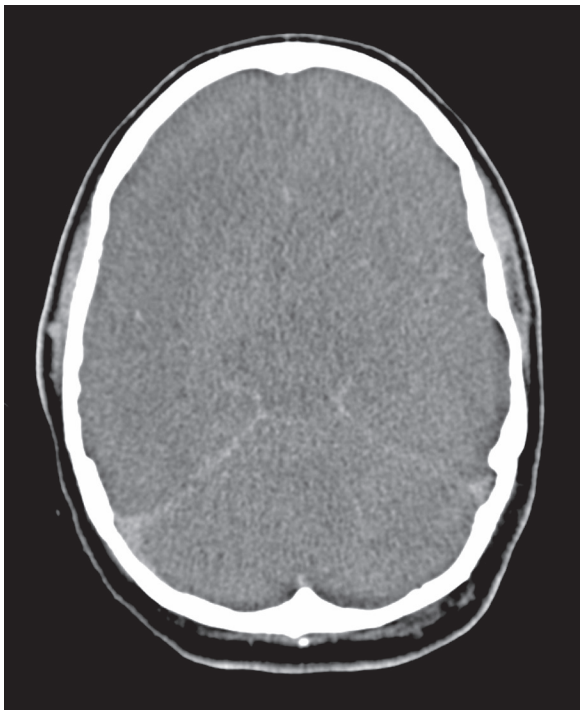

Fig. 34.13. Non–contrast-enhanced CT scan showing diffuse cerebral edema. Loss of gray-white differentiation in the brain parenchyma is present. Bilateral compression of the ventricles has occurred, with loss of cortical sulci.

edema adjacent to traumatic mass lesions demonstrates decreased density on CT scans compared with normal tissue.

Rotterdam Computed Tomography Score. The Rotterdam score was developed to determine the risk for mortality in traumatic brain injury. It is based on initial noncontrast CT findings of basal cistern compression, midline shift, presence of an epidural hematoma, and the presence of intraventricular blood and/or traumatic subarachnoid hemorrhage (Box 34.3).

Management

Out-of-Hospital Care

The out-of-hospital management of the head-injured patient should focus on preventing or minimizing secondary brain injury.

BOX 34.3

Rotterdam Score of Initial Noncontrast Computed Tomography for Predicting 6-Month Mortality Following Traumatic Brain Injury[a]

1. Basal cistern effacement
   0 = none
   1 = partially effaced (compressed)
   2 = completely effaced (compressed)
2. Midline shift
   0 = no shift or ≤5 mm
   1 = >5 mm
3. EDH (epidural hematoma)
   0 = EDH present
   1 = no EDH
4. IVH (intraventricular hemorrhage) or SAH (subarachnoid hemorrhage)
   0 = neither present
   1 = either present
   2. Add 1 to score

Total score = 1–6 points

[a]Probability of mortality at 6 mo postinjury based on score:
1 ≥ 0%
2 ≥ 7%
3 ≥ 16%
4 ≥ 26%
5 ≥ 53%
6 ≥ 61%

The two major systemic insults are hypotension and hypoxia. Interventions should be aimed at maintenance of oxygenation and prevention of hypotension through directed fluid resuscitation and control of hemorrhage.

Airway. Controversy exists regarding the benefits of out-of-hospital intubations in patients with brain injuries. In systems with short transport times, and in patients in whom an oxygen saturation more than 90% can be maintained with supplemental oxygen, field intubation is of questionable benefit and may potentially lead to worse outcomes.[50-52] Although unsuccessful attempts at field intubations may add to out-of-hospital time and increase the risk of aspiration or hypoxia, patients who are hypoxic or are unable to maintain their airway have improved outcomes in terms of mortality and neurologic outcome with field intubation.[50,52]

The ultimate goal in the field is to prevent or minimize hypoxia. Out-of-hospital airway protocols balance the risks of emergency intubation in an uncontrolled setting with the need to secure an at-risk airway and prevent hypoxia. If oxygenation can be maintained and transport time is short (most urban settings), definitive airway management should be delayed until arrival in the ED.[53] If endotracheal intubation is undertaken in the field, it should be performed by skilled practitioners with a rigorous quality assurance program and continuous provider training. All advanced airway placement should be confirmed with quantitative end-tidal capnography ($ETCO_2$), which dramatically improves the detection of improperly placed airway devices and inadvertent hyperventilation by field providers. Prehospital endotracheal intubation is associated with poorer prognosis in children and should be avoided in this population (see Chapter 165).

Hypotension. Avoiding and managing hypotension are critical elements of the prehospital treatment of the head-injured patient. The secondary survey of the head-injured patient should include a search for external signs of head trauma. Scalp lacerations may bleed a large volume into a bulky dressing, and a less bulky dressing should be used with firm constant manual pressure applied to avoid excessive blood loss. Any other ongoing external hemorrhage should be expeditiously addressed and controlled. Although permissive hypotension may be beneficial in some trauma patients, it is detrimental in the setting of brain injury.[54]

Agitation. Many severely head-injured patients are initially combative or agitated. Transporting an agitated patient who is fighting against physical restraints may exacerbate physical injury, cause an increase in ICP, and interfere with appropriate stabilization and management. Management of agitation in the out-of-hospital setting mirrors that used in the ED, as described below.

Emergency Department Management

General. In the ED phase of patients with severe head trauma, management is in accordance with ATLS (Advanced Trauma Life Support) protocols. Monitoring of vital signs should be continuous, such as respiratory status (pulse oximetry, capnography) heart rate, blood pressure, and temperature. Tetanus status should be determined and prophylaxis given, as appropriate. Pregnancy status in women of childbearing age should be verified.

Airway. Primary airway compromise in the setting of head trauma may result from craniofacial or neck trauma, bleeding, or vomiting. Secondary airway compromise may also result from brain injury, as in the case of loss of brainstem reflexes, patient agitation, severe systemic hypotension, or alterations in mental status. In either case, the airway should be secured early to protect against aspiration and prevent secondary brain injury as a result of hypoxia or hypercarbia (Fig. 34.14).

If possible, a rapid but detailed neurologic examination should be performed before the patient is given any sedative or neuromuscular blocking agent. This focused examination includes careful recording of the elements of the GCS, characterization of movement of all four extremities (to command, purposeful response to pain, localizing pain, withdrawal, posturing), tone, and pupillary reflexes. These elements are essential in correlating CT findings with clinical injury and are also useful in following the patient's progression. The drug selection and technique of intubation for the head-injured patient is discussed in Chapter 1.

Hypotension. If hypotension is detected at any time in the emergent management of a potentially brain-injured patient, a cause other than the brain injury should be sought (see Chapter 33). Systemic hypotension has profound implications for neurologic outcomes. In fact, a single episode of hypotension doubles mortality risk. As such, fluids or blood transfusion should be delivered to maintain a SBP of at least 90 mm Hg. Maintaining the SBP above 100 mm Hg may be considered to decrease mortality and improve outcome.[10]

Brain-Directed Hyperosmolar Therapy. If there are signs of impending herniation syndrome, such as deepening coma, a newly asymmetric pupil, or other substantially diminishing neurologic parameters, we recommend the use of osmotic diuretics, such as mannitol or hypertonic saline (HTS). Mannitol is the time-honored mainstay for the control of elevated ICP in acute severe TBI.[10] Mannitol (0.25–1 g/kg) can effectively reduce cerebral edema by producing an osmotic gradient that reduces brain volume and provides increased space for an expanding hematoma or brain swelling. The osmotic effects of mannitol occur within minutes and peak approximately 60 minutes after bolus administration. The ICP-lowering effects of a single bolus may last for 6 to 8 hours. Mannitol is also an effective volume expander and, in the presence of hypovolemic hypotension, may aid in maintaining

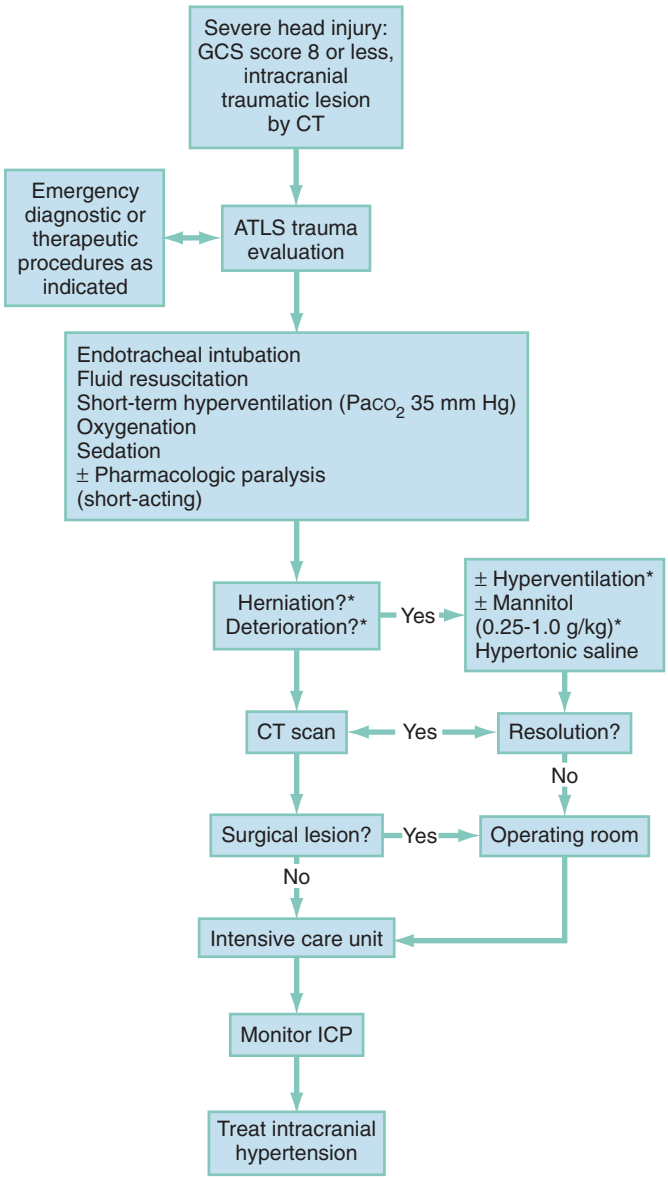

Fig. 34.14. Treatment options for initial resuscitation of a patient with a severe head injury. *ATLS,* Advanced Trauma Life Support; *CT,* computed tomography; *GCS,* Glasgow Coma Scale; *ICP,* intracranial pressure; *$Paco_2$,* partial pressure of arterial carbon dioxide. (From Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons; Joint Section on Neurotrauma and Critical Care; AANS/CNS; J Neurotrauma 24[Suppl 1]:S1–106, 2007.)

the systemic blood pressure required for adequate cerebral perfusion. It also promotes CBF by reducing blood viscosity and microcirculatory resistance. It is an effective free radical scavenger, reducing the concentration of oxygen free radicals that may promote cell membrane lipid peroxidation.

HTS also has been used effectively to reduce ICP in TBI. Few comparative data exist on brain-directed osmotic therapy, however. Selection of mannitol versus HTS should be made on an institutional basis, so that providers across various specialties (eg, emergency medicine, trauma surgery, neurosurgery, anesthesiology) provide consistent care.[55,56]

HTS given as bolus therapy is more effective than mannitol in lowering cumulative and daily ICP elevations after severe TBI, but it does not improve mortality.[55,57] Proposed benefits of HTS include reducing secondary injury through effects on cellular modulation, decreasing cerebral edema, improving peripheral perfusion, and decreasing ICP through vasoregulatory mechanisms[58] and upregulation of proinflammatory and prothrombotic mediators. Numerous clinical studies have demonstrated that HTS can significantly reduce ICP; however, the total number of enrolled patients in these trials is small, and interpretation is complicated by variation in protocols, HTS concentration, and rate of administration.[10,55,58-60]

Among patients with severe TBI not in hypovolemic shock, initial resuscitation with hypertonic saline or hypertonic saline-dextran, compared with normal saline, was found not to result in superior 6-month neurologic outcome or survival.[61,62] Mannitol can produce renal failure or hypotension if given in large doses. It may also induce a paradoxical effect of increased bleeding into a traumatic lesion by decompressing the tamponade effect of a hematoma. Potential adverse events associated with HTS include renal failure, central pontine myelinolysis, and rebound ICP elevation.[60] Osmotic therapy should be guided by findings on ICP monitoring. Prior to initiation of such monitoring, brain-directed osmotic therapies should be reserved for patients with signs of transtentorial herniation or progressive neurologic deterioration not attributable to extracranial causes.[10]

Osmolar therapy with mannitol or hypertonic saline can draw water across an intact BBB and thereby lower ICP. Mannitol, 0.25 to 1 g/kg, is given every 6 hours, up to a serum osmolality of 320 mOsm/kg. Treatment with 30 mL of 23.4% HTS appears to be at least as effective as mannitol at lowering ICP rapidly and reversing herniation, although a central line is necessary for safe administration. Hypertonic saline (23.4%), 30 to 60 mL, can be given every 6 hours, up to a maximum serum sodium level of 160 mEq/L. Because it is a potent diuretic, mannitol is preferred in cases of fluid overload, whereas HTS can be used as a resuscitative fluid; a 3% or 23.4% solution can be used.[63]

Hyperventilation. Under normal conditions, $Paco_2$ is the most powerful determinant of CBF and, between a range of 20 and 80 mm Hg, CBF is linearly responsive to $PaCO_2$. Formerly, so-called therapeutic hyperventilation was recommended as a method to reduce ICP. Unfortunately, however, this reduction in ICP is accomplished by reducing CBF, which is important in meeting the brain's metabolic demands. A low $Paco_2$, therefore, and the resulting low CBF, may result in cerebral ischemia, whereas high $Paco_2$ levels can result in cerebral hyperemia and high ICP. Normal ventilation is currently the goal for severe TBI patients in the absence of cerebral herniation, and $Paco_2$ is maintained in the normal range, from 35 to 45 mm Hg.[10] In the case of life-threatening cerebral herniation or significant ICP elevation, therapeutic hyperventilation is appropriate only as a short-term intervention, bridging to more definitive therapy (eg, craniectomy). Therefore, hyperventilation is recommended only as a temporizing measure for the reduction of elevated ICP and should not be used for routine management or prophylaxis. Hyperventilation should be avoided during the first 24 hours after injury when CBF is often critically reduced. If hyperventilation is used, jugular venous oxygen saturation (Sjo_2) or brain tissue O_2 partial pressure ($Btpo_2$) measurements are recommended to monitor oxygen delivery.[10,64] The neurologic effects of hypocapnia are illustrated in Fig. 34.15.

Cranial Decompression. In patients with impending herniation who do not respond to osmotic therapy and hyperventilation, particularly those with a history of so-called talk and deteriorate after head trauma, emergency cranial decompression may temporarily reverse or arrest the herniation syndrome.[65,66] Emergency trephination may allow enough time for a patient to

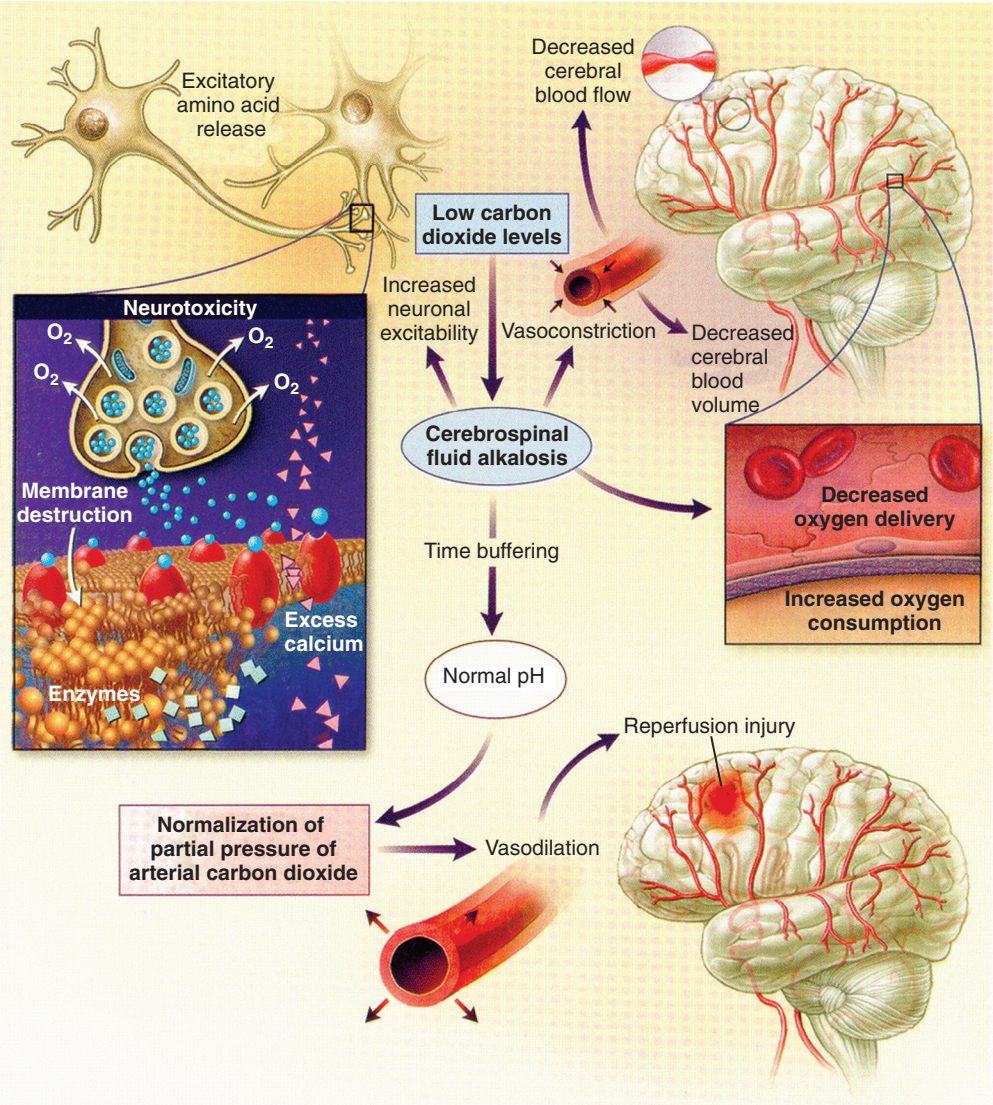

Fig. 34.15. Neurologic effects of hypocapnia. Systemic hypocapnia results in cerebrospinal fluid alkalosis, which decreases cerebral blood flow, cerebral oxygen delivery and, to a lesser extent, cerebral blood volume. The reduction in intracranial pressure may be lifesaving in patients in whom the pressure is severely elevated. However, hypocapnia-induced brain ischemia may occur because of vasoconstriction (impairing cerebral perfusion), reduced oxygen release from hemoglobin, and increased neuronal excitability, with the possible release of excitotoxins, such as glutamate. Over time, cerebrospinal fluid pH and hence cerebral blood flow gradually return to normal. Subsequent normalization of the partial pressure of arterial carbon dioxide can then result in cerebral hyperemia, causing reperfusion injury to previously ischemic brain regions. (From Laffey JG, Kavanagh BP: Hypocapnia. N Engl J Med 347:43–53, 2002.)

undergo a formal craniotomy in the operating room. However, most patients presenting unconscious have sustained diffuse massive brain injury, with no focal lesion amenable to emergency decompression. Patients with erratic or absent respiratory effort, bilateral fixed and dilated pupils, no spontaneous eye movements, and decerebrate posturing do not benefit from emergent burr holes. Furthermore, placement of a burr hole is a blind invasive procedure, and the chances of localizing the expanding lesions are uncertain. Trephination should be undertaken only after confirmation of an extradural collection by neuroimaging[67] and only by, or under the guidance of, an emergency clinician with specific training.

Decompressive craniectomy (DC) is the surgical removal of the skull bone and has been performed for the purpose of relieving elevated ICP, with outcome improvement in some TBI patients.[68,69] Bifrontal DC has been used in severe TBI patients with diffuse injury (without mass lesions) who have ICP elevation to more than 20 mm Hg for more than 15 minutes within a 1-hour period, refractory to first-tier therapies. There is no evidence for improved outcome, as measured by the Glasgow Outcome Scale-Extended (GOS-E) score, at 6 months postinjury.[10] However, this procedure has been demonstrated to reduce ICP and to minimize days in the intensive care unit (ICU). A large frontotemporoparietal DC (not <12 × 15 cm or 15 cm diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurologic outcomes in patients with severe TBI.[10]

Hemostatic Agents. Patients taking warfarin may be managed with prothrombin complex concentrate, fresh-frozen plasma), or vitamin K. Reversal of warfarin-associated anticoagulation is discussed in Chapter 114. There is inadequate evidence to support the routine use of platelet transfusion for intracranial hemorrhage for patients taking preinjury antiplatelet medications

(eg, aspirin, clopidogrel).[70] Idarucizumab, a reversal agent against dabigatran, is approved for emergency surgery and urgent procedures or in life-threatening or uncontrolled bleeding. Although studies are lacking in the setting of TBI, when significant intracranial bleeding is identified in patients with acute TBI who are taking dabigatran, we recommend reversal with idarucizumab. The recommended dose for idarucizumab is 5 g (2.5 g/vial) administered intravenously as two consecutive 2.5-g infusions or as a bolus injection by injecting both vials consecutively, one after another, via syringe.

Recombinant factor VIIa (rFVIIa) is a hemostatic agent that was originally developed to treat bleeding in hemophiliacs. Limited military experience led to interest in the use of rFVIIa for traumatic intracerebral hemorrhage as well.[71,72] However, results of clinical trials are mixed, and there has been no convincing evidence of benefit for this expensive agent in traumatic intracranial hemorrhage in the absence of preexisting coagulopathy. We do not recommend routine use of rFVIIa for patients with traumatic intracranial hemorrhage.[72,73]

When given early after injury (within 3 hours), tranexamic acid (TXA) has demonstrated benefit in trauma with hemorrhage without increasing the risk of adverse events.[74-76] The use of TXA is discussed in Chapter 33. Tranexamic acid is of no benefit for patients with isolated head trauma or head trauma without significant systemic hemorrhage and may even be harmful. TXA should not be used in this population.

Induced Hypothermia. Hyperpyrexia worsens outcome after severe TBI, and guidelines emphasize maintaining normothermia with antipyretic medications and cooling devices.[34] Induced therapeutic hypothermia has been proposed to decrease ICP, including reducing proinflammatory cytokines and stabilizing the BBB. However, the routine use of hypothermia for the treatment of TBI has met with mixed results in adult and pediatric populations.[77-79] Although hypothermia remains a significant area of research and promise for patients with severe and moderate TBI, the available scientific evidence is inconclusive with regard to improved mortality or morbidity.[80] Some studies have shown the potential for benefit,[77] but a recent randomized trial has shown that therapeutic hypothermia for severe TBI does not improve the neurologic outcomes or risk of mortality compared with strict temperature control.[78] Similarly, in another randomized trial in patients with an elevated ICP of more than 20 mm Hg, therapeutic hypothermia plus standard care to reduce ICP did not result in outcomes better than those with standard care alone.[81] We do not recommend the routine use of therapeutic hypothermia for the treatment of TBI.

Seizure Prophylaxis. Acute symptomatic seizures may occur as a result of severe traumatic brain injury (TBI). Such PTSs are classified as early when they occur within 7 days of injury or late when they occur after 7 days following injury. Posttraumatic epilepsy (PTE) is defined as recurrent seizures more than 7 days following injury. Up to 12% of all patients who sustain blunt head trauma and 50% of those with penetrating head trauma develop early PTSs.[46,82] Although the occurrence of seizures in the immediate posttrauma period is not predictive of future epilepsy, early seizures can cause hypoxia, hypercarbia, release of excitatory neurotransmitters, and increased ICP, potentially worsening secondary brain injury.

Prophylactic use of phenytoin or valproate is not recommended for preventing late PTS, but phenytoin is recommended to decrease the incidence of early PTS (within 7 days of injury), when the overall benefit is thought to outweigh the risk of complications associated with such treatment.[10,83] Early PTSs have not been associated with worse outcomes,[10] but the use of prophylactic anticonvulsants in penetrating brain injuries is recommended for 7 days postinjury.[46] There is insufficient evidence to recommend levetiracetam over phenytoin; we recommend phenytoin for early PTSs and toxicity.[10]

If the patient is actively seizing, benzodiazepines are administered as effective, rapid-acting, first-line anticonvulsants. Lorazepam (0.05–0.1 mg/kg IV) is the preferred agent for aborting seizures because of its high effectiveness and prolonged duration of action. Diazepam (0.1–0.2 mg/kg) or midazolam (0.05–0.1 mg/kg) is an effective alternative. For long-term anticonvulsant activity, phenytoin (18–20 mg/kg IV) or fosphenytoin (phenytoin equivalents, 15–18 mg/kg) can be given. Fosphenytoin has the advantages of rapid administration, smaller volume of fluid for the dose delivered, and less potential for hypotension than phenytoin.

Antibiotic Prophylaxis. Although the practice was once widespread, there is no evidence to support the use of antibiotic prophylaxis for the prevention of meningitis or other infection in patients with blunt basilar skull fractures, with or without evidence of CSF leakage.[84,85] Penetrating brain injury, however, is a different matter. Contamination with skin, bone, hair, and tissue occurs and may be widespread when there is cavitation caused by the missile as it passes through the brain.[46] Evidence supports the use of intravenous (IV) prophylactic, broad-spectrum antibiotics to cover for staphylococci, gram-negative bacilli, and anaerobes for penetrating craniocerebral trauma. Although there are several potential antibiotic regimens, a combination of vancomycin, 1 g bid, gentamycin, 80 mg tid, and metronidazole, 500 mg qid, will provide adequate coverage.[86,87]

Patients undergoing ICP monitoring are reported to have related infection rates as high as 27%.[88] For external ventricular drains (EVDs), routine catheter exchanges has been replaced by attention to proper care during insertion, CSF sampling techniques and, in some cases, prophylactic IV antibiotics. In a single-institution study, bundle implementation (including hand hygiene, prophylactic antibiotics, sterile technique, hair removal for dressing adherence, skin preparation using iodine and isopropyl alcohol, full surgical attire for the surgeon and other bedside providers), together with an antimicrobial-impregnated catheter, dramatically decreased EVD-related infections. We recommend training and situational awareness of best practices for infection control, assisted by checklists. However, there is insufficient evidence at this time to recommend antibiotic-impregnated EVDs for minimizing infection.

Other Therapies

Corticosteroids. Corticosteroids have no benefit for patients with head trauma, and in fact demonstrate an increase in adverse events, including infection, gastrointestinal bleeding and mortality. In patients with severe TBI, high-dose methylprednisolone is associated with increased mortality and is contraindicated.[10]

Barbiturates. Barbiturate therapy has historically been used in severely brain-injured patients to reduce cerebral metabolic demands of the injured brain tissue and reduce elevated ICP. However, barbiturates also can cause a decrease in SBP. Compared with placebo, barbiturates offer no mortality benefit; furthermore, any benefit of decrease in ICP is offset by the risk of hypotension.[89] The only remaining value of barbiturates in TBI is the use of high-dose barbiturate therapy to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.[10] Prophylactic administration of barbiturates to induce burst suppression measured by electroencephalography is not recommended.

Monitoring of Intracranial Pressure and Cerebral Spinal Fluid Drainage. Management of severe TBI patients using information from ICP monitoring is recommended to reduce

in-hospital and 2-week postinjury mortality.[10] However, management of EVD systems in patients with severe TBI remains a controversial topic. An EVD in a closed position allows for monitoring of ICP, whereas in an open position drainage of CSF can occur. Practice patterns regarding whether the EVD should be maintained in a closed or open position vary widely based on a number of variables, including patient age, institutional resources, and physician preferences. Continuous CSF drainage is a relatively common practice in the pediatric population. In adults, there is variability in practice with three options: (1) continuously monitoring ICP and only intermittently draining for ICP elevations; (2) intermittently monitoring ICP with continuous drainage of CSF; or (3) continuously monitoring ICP with continuous drainage of CSF.[90,91] An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use. The use of CSF drainage to lower ICP in patients with an initial GCS less than 6 during the first 12 hours after injury may be considered.[10]

Glucose Control and Nutrition. Hyperglycemia and hypoglycemia are associated with worsened outcomes following a severe TBI, but the optimal glucose target and best treatment regimen is yet to be determined.[10] The complex interaction of the body with nutritional support is magnified during illness, particularly after severe TBI. Severe TBI is associated with increased energy expenditure early after injury. Guidelines recommend feeding patients to attain basal caloric replacement, at least by the fifth day and, at most, by the seventh day postinjury is recommended to decrease mortality.[10] Furthermore, transgastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.[10]

Erythropoietin. In a recent multicenter randomized controlled trial of patients with moderate to severe TBI, erythropoietin did not reduce the number of patients with severe neurologic dysfunction or increase the incidence of deep venous thrombosis of the lower limbs. There was no effect on mortality at 6 months. It is not recommended for use at this time.[92]

Progesterone. Progesterone has been shown to improve neurologic outcome in early-phase trials involving patients with TBI. In a double-blind, multicenter clinical trial, progesterone was administered to TBI patients with moderate to severe TBI within 4 hours of injury. Progesterone did not improve outcomes in patients with TBI over placebo.[93] These findings are consistent with a recent meta-analysis.[94] Progesterone is not recommended for treatment of TBI.

Hyperbaric Oxygen Therapy. Hyperbaric oxygen therapy following severe, acute TBI provides the injured brain with an increased partial pressure of oxygen and theoretically reduces cerebral edema.[95] However, although the pooled results of several small studies have suggested some benefit in terms of survival, the clinical significance is questionable. Thus, the use of hyperbaric oxygen therapy for the treatment of TBI injury cannot be recommended.[95]

Management of Specific Injuries

Scalp Wounds. If blood loss is brisk, rapid hemostasis is a priority. Initially, hemostasis may be achieved by the application of a temporary, tight pressure dressing, allowing prompt attention to other priorities. Alternatively, a stapler may be used to staple the scalp laceration rapidly to control bleeding. The wound later can be reopened for proper irrigation and reclosure. Other methods to achieve hemostasis include direct digital compression of the bleeding vessel against the skull, infiltration of the wound edges with lidocaine plus epinephrine, and clamping or ligation of identified bleeding vessels. The best approach is repair with sutures or staples because the closure will assist in tamponade. In stable patients, closure of the wound, after proper débridement and irrigation, is the most effective way to stop a bleeding scalp laceration and prevent the tissue crush injury that may occur if other compressive methods are used for too long.

Once hemostasis is obtained, the wound should be irrigated to rinse away any debris. The emissary vessels of the subgaleal layer of the scalp drain directly into the diploë veins of the skull. These in turn drain into the venous sinuses. Contaminated or infected scalp wounds, therefore, have the potential to cause serious intracranial infections. Blood clots and other debris should be removed and the galea and underlying cranium palpated to detect any remaining debris, disruptions, or bone step-offs. Shear injuries to the scalp may deposit contaminants at sites distant from the apparent injury. The complexity of stellate lacerations often interferes with thorough inspection and débridement, making them particularly susceptible to infection. Digital exploration of a scalp wound should be performed gently; if done too vigorously, comminuted or depressed bone pieces may be depressed further.

It is easy to confuse a disruption in the galea or tear in the periosteum with a skull fracture. The base of the laceration should therefore be directly visualized. Clipping away a small area of hair parallel to the edges of the wound may facilitate this. Alternatively, an antibiotic ointment can be applied to the hair immediately surrounding the wound and used to plaster the hair away from the injury site. If hair is accidentally embedded within the repaired laceration, it can delay healing by producing an inflammatory reaction or by serving as a nidus of infection. If the laceration begins on the forehead and extends upward beyond the hairline, surrounding hair should not be removed. Removal obliterates a useful landmark for cosmetic closure and may result in malalignment of the two laceration edges.

Several studies have evaluated the use of staples versus sutures to close scalp lacerations that do not involve the galea. For adult and pediatric scalp lacerations that begin beyond the hairline, staples have been shown to be cheaper, take less time, and have the same outcome than sutures if used in the appropriate manner. However, staples cannot be used to close the galea and may not be effective alone when hemostasis is a problem. Large lacerations of the galea are closed to prevent the edges of the wound from pulling apart as the muscles within the galea contract. The skin, dermis, and galea can usually be repaired in a single layer with interrupted or vertical mattress sutures of 3-0 nylon or polypropylene.[96] Recently, an alternative method of scalp laceration closure has been described, in which bundles of the patient's hair on each side of the wound are twisted together and then secured with tissue glue.[96] This may provide another method of effective repair and prevent the need to remove the closure material after the wound has healed, particularly in the pediatric population. Because of the rich blood supply of the scalp, even very large scalp avulsions may remain viable. If the avulsion remains attached to the rest of the scalp by a tissue bridge, it should be reattached to the surrounding tissue. If the avulsion is completely detached from the scalp, it should be treated as any other amputated part and reimplanted as soon as possible.

Scalp abrasions are often contaminated with pieces of dirt or other debris. The wound should be cleaned as thoroughly as possible and inspected for puncture wounds or other areas that penetrate beyond the superficial layers of the skin to ensure the removal of unsuspected foreign bodies. A careful inspection often reveals a small scalp laceration within the abraded area. Systemic antibiotics are usually not needed for carefully managed scalp wounds because rapid healing is facilitated by the rich blood supply of the scalp. However, special consideration may be given to large or highly contaminated wounds, bite wounds, and immunocompromised patients.

Skull Fractures. A noncontrast head CT scan with bone windows has become the imaging modality of choice for patients

with suspected skull fractures or to identify intracranial foreign bodies. Plain radiographs can be useful when CT is not available.

Linear Fractures. Linear skull fractures are clinically important if they cross the middle meningeal groove or major venous dural sinuses; they can disrupt these vascular structures and cause the formation of EDHs. No specific intervention is necessary for linear skull fractures if a noncontrast CT scan reveals no underlying brain injury. Patients with no evidence of intracranial injury on CT and no other significant extracranial injuries should be observed in the ED for 4 to 6 hours prior to discharge. If there is any suspicion or clinical evidence of brain injury, patients should be admitted for observation. Those with intracranial injuries should have an emergent neurosurgical consultation. Patients with simple linear skull fractures may demonstrate concussive symptoms or other evidence of mild TBI and should be provided appropriate discharge instructions (see later, "Mild Traumatic Brain Injury: Disposition").

Depressed Fractures. When a depressed fracture occurs, traumatic impact drives the bone piece below the plane of the skull. The edges of the depressed portion of skull may become locked underneath the adjacent intact bone and fail to reduce into their anatomic position. As a result, the depressed piece of bone can penetrate tissue and lacerate the dura. Fractures in which the free piece of bone is depressed deeper than the adjacent inner table of the skull require surgical elevation. Depressed skull fractures are usually open fractures with disruption of the galea, which can often be felt with palpation of the skull. However, this examination should be done cautiously to avoid driving a depressed bone fragment deeper into the cranium. The clinical examination for a depressed skull fracture may be misleading. The mobility of the scalp can result in nonalignment of the fracture with an overlying scalp laceration. As a result, the skull underlying the laceration may be normal, with the depressed area several centimeters away. Scalp swelling may interfere with the physical examination findings and hide any palpable bone defects.

Depressed fractures may be difficult to visualize on plain skull radiographs. The free piece of bone demonstrates increased or double density because it often overlaps the nonfractured bone, or it is rotated from the rest of the adjacent cranium. Tangential views of the skull may increase the ability to visualize the fracture. However, CT scanning with bone windows, if available, remains the imaging modality of choice.

An open depressed skull fracture, as well as any type of penetrating skull injury, increases the risk for developing intracranial and meningeal infection and seizures and should receive prophylaxis.[97] Patients with depressed skull fractures should be admitted for continued observation. Patients with open (compound) cranial fractures depressed greater than the thickness of the cranium should undergo operative intervention to prevent infection. They may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination. Nonoperative management of closed (simple) depressed cranial fractures is a treatment option. For all open depressed skull fractures, we suggest that prophylactic antibiotics be given for 5 to 7 days to prevent the risk of subsequent CNS infection. Suggested antibiotics are identical to those for penetrating head trauma.

Basilar Skull Fractures. Basilar fractures are the result of considerable impact force and are highly associated with underlying brain injury. Emergency clinicians should be suspicious of an epidural hematoma in patients with a temporal bone basilar skull fracture. All patients with basilar skull fractures should be admitted for observation, regardless of the need for surgical intervention. A systematic review and meta-analysis of antibiotic prophylaxis following basilar skull fracture has concluded that routine prophylaxis is not supported by the available evidence, whether or not there is evidence of CSF leakage.[84,85] We do not recommend routine antibiotics for basilar skull fractures unless the patient is immunocompromised.[84,85] Most CSF leaks resolve spontaneously within 1 week, with no complications.[98] If the leak persists beyond 7 days, the incidence of bacterial meningitis increases significantly; prophylactic antibiotics should be given in such cases. Antibiotic selection is identical to that for penetrating head trauma. If a patient with a previously diagnosed CSF leak returns to the ED later with fever, the diagnosis of meningitis should be strongly suspected and appropriate evaluation (ie, lumbar puncture) and antibiotic treatment initiated immediately. Treatment of posttraumatic meningitis is discussed in Chapter 99.

Extra-Axial Lesions

Epidural Hematoma. Expert consensus guidelines support rapid surgical evacuation for any patient who has mass effect on a CT scan or progressive neurologic deterioration. Indications for urgent surgical evacuation include epidural hematomas larger than 30 cm^3, regardless of the patient's GCS score, as well as comatose patients with an acute EDH and anisocoria on pupillary examination. For patients with acute EDH who are awake and have no focal neurologic deficits, nonsurgical management is based on the size of the hematoma (<30 cm^3), thickness of the clot (<15 mm), and degree of midline shift (<5 mm). In those managed nonoperatively, close neurologic observation in a neurosurgical center is required, and the first repeat CT scan should be obtained within 6 to 8 hours postinjury.

Subdural Hematoma. Because of associated brain injury caused by the SDH, the delay in clinical signs and symptoms, and the more advanced mean age of the at-risk population, the mortality associated with SDH is much higher than that associated with EDH. Pupil inequality, motor deficit, and other signs consistent with increased brain swelling may be present on the initial examination. If the patient is deeply comatose at presentation, with flaccidity and without signs of brainstem activity, he or she may best be served by simply providing supportive care. Subsequent management decisions should be discussed with the patient's family and attending neurosurgeon. A small SDH (only a few millimeters thick at its widest point on CT scan) often is amenable to serial observations of the patient's status and appearance of the SDH on CT scan. Even a small SDH may be accompanied by extensive brain tissue damage that can cause an increase in ICP sufficient to precipitate a herniation syndrome.

Indications for surgical evacuation include acute SDHs with a thickness more than 10 mm or a midline shift of more than 5 mm on a CT scan, regardless of the patient's GCS score. Other parameters for surgical evacuation include a worsening GCS score (≥2 points from the time of injury to hospital admission) in comatose patients, asymmetric or fixed and dilated pupils, and persistent elevation in ICP. Most patients with subacute SDH require surgical evacuation of the lesion.

The treatment of chronic SDHs is controversial. Symptomatic chronic SDHs require surgical evacuation. Most patients have a good outcome after surgery. Overall, the mortality from surgically drained chronic SDH approaches 5%, with decreased survival in older adults.[99]

Traumatic Subarachnoid Hemorrhage. If there is no other brain injury, tSAH does not generally carry a poor prognosis. The most serious complication of tSAH is worsening of cerebral vasospasm, which may be severe enough to induce cerebral ischemia. Posttraumatic vasospasm is common, occurring approximately 48 hours after injury and persisting for up to 2 weeks. Vasospasm following TBI is characterized by a different time course, duration, and associated profile of risk factors than those of aneurysmal SAH (aSAH).[100] Although, no treatments have been shown to affect outcomes conclusively in tSAH, calcium channel

blockers, such as nimodipine and nicardipine, have been used in the acute ICU setting to prevent or reduce vasospasm after tSAH.[100] However, we do not recommend the routine use of calcium channel blockers in tSAH. Patients with severe TBI and large amounts of SAH may benefit from serial noninvasive monitoring and institution of therapy in the setting of radiographic or clinical deterioration.

Subdural Hygroma. If SDHGs are asymptomatic, observation is reasonable. Otherwise, they are surgically evacuated. Mortality approaches 20% and appears to depend on the severity of other intracranial injury.[101]

Intra-Axial Lesions

Cerebral Contusion. In one series of patients with brain contusions who initially received conservative treatment, 45% had significant progression on CT, and 19% required surgical intervention.[102] Patients with lower GCS scores and larger cerebral contusions are at higher risk for hemorrhagic progression and the need for delayed surgical decompression.[102] Hemorrhagic progression of a contusion generally occurs within the first 12 hours, but may occur as late as 3 to 4 days after head trauma. Small contusions that progress are usually clinically silent and are unlikely to require surgical decompression.

Intracerebral Hematoma. Many patients with an ICH require emergent intervention or surgery to lower elevated ICP. Mortality is low in patients who are conscious before surgery, whereas in unconscious patients, mortality approaches 45%. ICHs that bleed into the ventricles or cerebellum also carry a high mortality rate.

Intracerebellar Hematoma. Acute management should first address the most clinically significant lesion. Mortality from isolated traumatic intracerebellar hematoma is very high. Emergent neurosurgical consultation is indicated.

Complications and Outcome

Seizures

PTSs are classified as early when they occur within 7 days of injury or late when they occur after 7 days following injury. PTE is defined as recurrent seizures more than 7 days following injury. In patients with severe TBI, the rate of a clinical PTS may be as high as 12%, whereas that of subclinical seizures detected on electroencephalography may be as high as 20% to 25%.[10] The risk factors for early PTS include GCS score of 10 or lower, immediate seizures, posttraumatic amnesia lasting longer than 30 minutes, linear or depressed skull fracture, penetrating head injury, subdural, epidural, or intracerebral hematoma, cortical contusion, age 65 years or younger, or chronic alcoholism.[82] Rates of PTE are substantially higher than the risk of developing epilepsy in the general population. The risk factors for PTE include severe TBI, early PTSs prior to discharge, acute intracerebral hematoma or cortical contusion, posttraumatic amnesia lasting longer than 24 hours, age older than 65 years, and premorbid history of depression.[82]

Early PTSs, within 24 hours of injury, are usually brief and are probably caused by transient mechanical and neurochemical changes within the brain. In the 24 to 48 hours after trauma, seizures are caused by worsening cerebral edema, small hemorrhages, or penetrating injuries. PTSs are common in children and can be precipitated by MTBI but are more common in moderate and severe TBI.[103,104] Acute PTS prophylaxis in the ED is recommended for penetrating brain injury.[46,105] Prophylactic use of phenytoin or valproate is not recommended for preventing late PTS from a nonpenetrating injury. Phenytoin is recommended to decrease the incidence of early PTS (within 7 days of injury), when the overall benefit is thought to outweigh the complications associated with such treatment. However, early PTSs have not been associated with worse outcomes.[10] Newer agents, such as levetiracetam, have been assessed for this purpose as well.[106] However, at present, there is insufficient evidence to recommend levetiracetam over phenytoin regarding efficacy for preventing early PTSs and toxicity.[10] The decision to maintain the head trauma patient on long-term anticonvulsant therapy during the recovery period depends on the patient's subsequent course. Long-term seizure prophylaxis is not indicated for all patients who have had PTSs in the acute or subacute period.[10] Prophylactic anticonvulsants are not recommended to prevent late PTSs.[10,107,108]

Central Nervous System Infections

Meningitis After Basilar Fractures. Posttraumatic meningitis is caused by a variety of microbes, depending on the portal of bacterial entry. Patients have typical signs and symptoms of meningitis, including fever, altered mental status, and occasional focal neurologic signs. In patients with a CSF leak after basilar fracture, early meningitis (ie, within 3 days of injury) is usually caused by pneumococci. Treatment of posttraumatic meningitis is discussed in Chapter 99.

Brain Abscess. Brain abscesses develop infrequently after penetrating missile injuries to the head. Abscesses can also develop after open depressed skull fractures if bone fragments are not removed or as a postoperative complication. Posttraumatic CSF fistulae and fractures that disrupt air-filled sinuses predispose to the formation of brain abscesses. Clinical manifestations include headaches, nausea, vomiting, declining mental status, signs of increased ICP, and new focal neurologic findings in patients who had been improving after trauma. Evaluation and treatment of brain abscess are discussed in Chapter 99.

Cranial Osteomyelitis. Cranial osteomyelitis can occur after penetrating injury to the skull. The clinical manifestations include pain, tenderness, swelling, and warmth at the infected site. More than 50% of cases are obvious on plain skull radiographs. Technetium bone scans can help in the diagnosis when the skull radiographs are negative, but false-positive bone scans occur in patients with previous trauma or craniotomy. Adding a gallium scan helps differentiate infection from other causes of a positive technetium scan. Patients with posttraumatic cranial osteomyelitis require surgical débridement and removal of the infected bone. Antibiotic choice is determined by culture results. If systemic symptoms are present, an underlying subdural or epidural empyema is often present.

Medical Complications

There are several systemic manifestations of TBI that can occur in the absence of any specific organ injury or systemic infection. The nature and severity of these manifestations depend mainly on the severity of the brain injury.

Disseminated Intravascular Coagulation. The injured brain is a source of tissue thromboplastin that activates the extrinsic clotting system. Disseminated intravascular coagulation (DIC) can develop within hours after any injury disrupting brain tissue. Cerebral intravascular coagulation is a universal response to TBI and is found in tissues from surgical specimens of human cerebral contusions. Coagulation abnormalities, including systemic DIC, are detected in over 50% of severe TBI patients.[109] Isolated severe TBI patients who develop coagulopathy have higher mortality rates than isolated severe TBI patients without coagulopathy.[110] DIC not only increases morbidity and mortality after severe TBI, it increases the risk of delayed intracranial hemorrhage. If a stable

patient with DIC suddenly deteriorates, a repeat CT scan should be obtained to rule out hemorrhage.

The extent of tissue destruction determines the degree of DIC that develops. The diagnosis is based on abnormalities in the international normalized ratio (INR), prothrombin time (PT), partial thromboplastin time (PTT), platelets, plasma fibrinogen levels, and fibrin degradation products. Patients with coagulopathy or abnormal platelet function require interventions to correct these.[111] Furthermore, patients with moderate or severe head trauma are at extremely high risk to experience venous thromboembolic events (VTEs) after admission to the hospital. Recent evidence has suggested that patients with moderate or severe head trauma can be safely treated with low-molecular-weight heparin to reduce the risk of VTEs without significantly increasing the risk of intracerebral hematoma expansion.[112]

Neurogenic Pulmonary Edema. Neurogenic pulmonary edema can develop minutes to days after head trauma.[113] It leads to an increase in extravascular fluids in the lungs, which causes hypoxia and decreased lung compliance. Theories on its pathophysiology include the following: (1) catecholamine surge or blast from the TBI, resulting in increased intravascular pressure, increased capillary permeability, and hydrostatic edema[114]; and (2) a systematic inflammatory reaction leading to endothelial damage and vasogenic edema. Treatment of acute lung injury in TBI is challenging because measures such as hypercapnia, fluid restriction, and prone ventilation (which raises ICP) that are routinely used to treat lung injury are contraindicated in TBI.[113] Positive end-expiratory pressure (PEEP) is commonly used to reduce lung fluids. Although PEEP is thought to increase ICP by reducing venous return, studies have shown that with adequate intravascular volume and MAP, PEEP does not adversely affect ICP and may reduce ICP by improving cerebral oxygenation. Furthermore, controlling ICP also appears to reduce the neurogenic stimulation that may contributes to this edema. Close ICP and ventilator management are essential to improved outcomes in patients with acute lung injury secondary to TBI.

Cardiac Dysfunction. A variety of cardiac rhythm, rate, and conduction abnormalities are detected after TBI. Harvey Cushing noted a connection between cardiac dysrhythmias and intracranial bleeding in the early 20th century. Many brain-injured patients with cardiac dysfunction have concurrent myocardial injury from underlying disease or chest injury. However, brain injury can cause primary cardiac dysfunction. High levels of circulating catecholamines have been measured in head-injured patients, with increased sympathetic nervous system activation.[115,116] Cardiac rhythm abnormalities have been reported in up to 70% of all patients with tSAH and more than 50% of all patients with traumatic intracranial hemorrhage.[115] In SAH, the cardiac dysrhythmias may result from autonomic nervous system dysfunction that subsequently affects ventricular polarization.

The most common cardiac dysrhythmia after TBI is supraventricular tachycardia, but many other rhythms have been observed. Findings on the electrocardiogram include diffuse large upright or inverted T waves, prolonged QT intervals, ST segment depression or elevation, and U waves. The primary goal in the emergency management of cardiac dysfunction after head trauma is ensuring adequate tissue perfusion and avoiding hypoxia. Dysrhythmias in head-injured patients often resolve as ICP is reduced.

Disposition

All patients with moderate TBI should be admitted for a period of observation, even with an initial, apparently normal CT scan. Frequent neurologic checks should be initiated, and a repeat CT scan is indicated if the patient's condition deteriorates or fails to improve over the first 48 hours after trauma. In patients with persistent symptoms of headache, confusion, or memory difficulties, delayed MRI may define lesions in the regions related to cognition that cannot be seen on CT. Although not useful in the acute setting, MRI has prognostic value during subsequent care and assists in directing the future rehabilitation of these patients.

All patients with moderate to severe head trauma require imaging to determine the extent and nature of the brain injury and necessity of neurosurgical intervention. Neurosurgical consultation should be obtained as soon as possible to help direct the patient's subsequent management. Moderate and severely head-injured patients require admission to an institution capable of intensive neurosurgical care and acute neurosurgical intervention. If this is not available at the receiving hospital, the patient should be transferred to an appropriate institution.

MILD TRAUMATIC BRAIN INJURY

Clinical Features and History

As with moderate and severe TB, a comprehensive history includes information from the patient, prehospital personnel, family members and witnesses about the mechanism of injury, events before and after the injury, age, comorbidities, coagulopathies (eg, hemophilia, Von Willebrand disease, hepatic insufficiency, use of anticoagulants), consumption of alcohol or drugs, changes in mental status or deteriorating GCS scores, previous TBI or concussion, symptoms of other potential injuries, and TBI symptoms (including postconcussive symptoms). By definition, the diagnosis of MTBI is largely clinical. It is not uncommon for MTBI symptoms to dissipate by the time patients reach the ED. It is important to ask patients specifically about symptoms of disorientation, confusion, amnesia, or disordered awareness, with or without loss of consciousness. A number of MTBI patients do not experience a loss of consciousness and, if they do, it is difficult to quantify unless there are witnesses. Patients may report headache, dizziness (vertigo or imbalance), lack of awareness of surroundings, and nausea and vomiting. Patients may also complain of mood and cognitive disturbances, sensitivity to light and noise, impaired verbal memory, delayed language comprehension, and slowed speech and exhibit balance problems.

Alert patients are questioned regarding cervical spine pain. Immobilization is indicated until cervical spine injury is excluded.

Physical Examination

The general physical examination is as for the patient with moderate or severe head injury, as described earlier. A more detailed neurologic examination is often possible in patients with mild MTBI who are able to interact with the examiner and cooperate with the examination. The examination includes evaluation of the cranial nerves, because CN injuries can occur in MTBI, particularly in conjunction with skull base fractures.[117] Assess for anosmia and hyposmia, because the olfactory nerve (CN I) is one of the most common CNs affected after MTBI.[117] This may be done by having the patient smell ground coffee or a citrus-scented beverage or cleaning agent. The facial nerve (CN VII) and oculomotor nerves (CNs III, IV, and VI) are also frequently injured, so assess for facial paralysis, change in taste, and/or diplopia. In MTBI, CNs IV and VI are more commonly injured than CN III.[117] CN VII palsy may indicate a fracture of the temporal bone, particularly if it occurs in association with decreased hearing (CN VIII). Hearing impairment can be one of the more subtle deficits seen after TBI.[118] Facial pain (CN V) and occipital neuralgia may also occur in association with MTBI. Assess coordination, balance, and gait. MTBI patients often can have difficulty with the finger-nose-finger test and will use slow purposeful movements to complete the task.

Romberg testing may demonstrate significant sway. Vestibular problems can affect balance and gait.[119] Assess sensation, motor strength, symmetry, and reflexes. Note if the patient displays a peculiar flat affect, appears devoid of emotion, and speaks in a slow monotone voice without inflection, which may indicate damage to the prefrontal cortex or frontal lobes.[120]

Differential Diagnosis

MTBI (GCS score of 13–15) is characterized by symptoms of confusion and amnesia, with or without preceding loss of consciousness. The differential diagnosis, in the context of trauma, therefore includes intoxication (drugs, alcohol, medications), posttraumatic seizures and postictal state, hypoglycemia, and other injuries that may impair a patient's ability to communicate, such as hypoxemia or hypoperfusion (from extracranial injuries), facial bone fractures, cervical spine or spinal cord injuries, injuries to the eyes or tympanic membranes, laryngeal or vocal cord injuries, and vascular injuries of the neck. Patients can deteriorate from an expanding intracranial hematoma after what appears clinically to be a MTBI, even if the patient presents with a GCS score of 15. In such a case, the injury would be reclassified as moderate or severe TBI.

Prior to the trauma, patients may have experienced a syncopal episode, seizure, or cardiac event that produced a loss of consciousness that led to the subsequent trauma. For example, an MVC may have resulted from the driver becoming distracted or incapacitated by important symptoms suggestive of an unrelated disorder. Therefore, potential precipitants to the trauma should also be considered in the differential diagnosis.

An MTBI or concussion may go unrecognized in the ED, especially if symptoms are transient or if more visible injuries dominate the assessment. An underlying dementia or psychiatric illness may make it very difficult to distinguish an MTBI from baseline cognitive or mental dysfunction. Other conditions that could cofound the diagnosis include stroke, encephalopathies (eg, hepatic, uremic), delirium from alcohol or drug withdrawal, neurologic conditions (eg, Parkinson's or Alzheimer's disease, autism), and infection or sepsis. Even low-mechanism trauma should alert the emergency clinician to the possibility of brain injury in susceptible populations. A thorough medical evaluation is critical.

Diagnostic Testing

Neuroimaging in the Emergency Department With Computed Tomography

CT imaging of the head is the diagnostic standard for identifying intracranial injury in the ED. However, CT is associated with exposure to ionizing radiation and higher health care costs.[121] Therefore, a number of clinical decision rules have been prospectively derived and validated to identify patients at risk for neurosurgical intervention and intracranial lesions on CT scan in adult patients with suspected MTBI in the ED. These include the New Orleans Criteria (NOC), Canadian Computed Tomography Head Rule (CCHR), and National Emergency X-Radiography Utilization Study II (NEXUS-II; Box 34.4). Others have developed guidelines based on available evidence, including the American College of Emergency Physicians clinical policy on neuroimaging of adult ED patients with MTBI (ACEP), National Institute for Health and Clinical Excellence (NICE), Neurotraumatology Committee of the World Federation of Neurosurgical Societies (WFNS), Scandinavian Neurotrauma Committee, and Scottish Intercollegiate Guidelines Network (SIGN). Although most of the rules and guidelines produce high sensitivities for detecting neurosurgical intervention and intracranial lesions, the specificities are variable. Additional clinical decision rules have been developed for use in the pediatric population (see Chapter 165).[122]

The most widely researched clinical decision rules for MTBI are the CCHR and NOC, with external validation studies in the United States and internationally.[123-126] The CCHR was developed for use in patients with a GCS score of 13 to 15; it divides clinical variables into high- and medium-risk categories. The NOC was developed for use in patients with a GCS score of 15 only and is composed of seven clinical variables. For injuries requiring neurosurgical intervention, both the CCHR and NOC have a high sensitivity (99%–100%) but the CCHR has a much higher specificity (CCHR, 48%–77%; NOC, 3%–31%).[127] For identification of traumatic intracranial lesions on CT, the CCHR and NOC have a high sensitivity (CCHR, 80%–100%; NOC, 95%–100%) but specificity is higher with the CCHR.[127] In terms of potential for CT reduction, adherence to the NOC results in an increase in head CT use; adherence to the CCHR results in a decrease in head CT use compared to current practice.[124] Imaging of patients in this population should follow a validated guideline. Clinicians in emergency medicine, trauma surgery, neurosurgery and, as indicated, neurology, should review the relevant guidelines (CCHR, NOC) and select the system thought to be most applicable for their setting and patient population. Oversight should ensure that cases not following the adopted guidelines are reviewed and feedback is provided to emergency clinicians.

BOX 34.4

Clinical Decision Rules for Neuroimaging in Adults With Mild Traumatic Brain Injury

CANADIAN COMPUTED TOMOGRAPHY HEAD RULE (CCHR)

High-Risk Injury (May Require Neurologic Intervention)

1. GCS score < 15 at 2 hr after injury
2. Suspected open or depressed skull fracture
3. Any sign of basal skull fracture (hemotympanum, raccoon eyes, CSF otorrhea or rhinorrhea, Battle's sign)
4. Vomiting ≥ two episodes
5. Age ≥ 65 years

Medium-Risk Injury (May Have Important Brain Injury on CT)

6. Amnesia before impact ≥ 30 min
7. Dangerous mechanism (pedestrian struck by vehicle, occupant ejected from vehicle, fall from elevation >3 feet [five stairs])

NEW ORLEANS CRITERIA (NOC)

1. Headache
2. Vomiting
3. Age > 60 yr
4. Drug or alcohol intoxication
5. Persistent anterograde amnesia
6. Trauma above the clavicle
7. Seizure

NEXUS II CRITERIA

1. Evidence of significant skull fracture
2. Scalp hematoma
3. Neurologic deficit
4. Altered level of alertness
5. Abnormal behavior
6. Coagulopathy
7. Persistent vomiting
8. Age ≥ 65 yr

CSF, Cerebrospinal fluid; *GCS,* Glasgow Coma Scale.

Other Neuroimaging Modalities

Structural MRI. CT is the imaging modality of choice for initial screening to exclude serious traumatic intracranial lesions in MTBI. However, many patients who develop persistent symptoms and cognitive deficits have no detectable abnormalities on CT. MRI is better than CT in detecting posttraumatic ischemic infarctions, subacute nonhemorrhagic lesions and contusions, axonal shear injury, and lesions in the brainstem or posterior fossa. Structural MRI, particularly at a 3-T strength, improves structural sensitivity, can be performed when neurologic findings cannot be explained by CT,[128] and is particularly valuable in assessing the brainstem, posterior fossa, and brain parenchyma adjacent to the calvaria.[129] Structural MRI (without contrast) can also be used for the evaluation of TBI-related symptoms in the subacute and chronic phases of injury.[128]

Susceptibility-Weighted Imaging. A significant advancement in the imaging of MTBI has been the development of susceptibility-weighted imaging (SWI). This technique is an imaging method that grew out of and is part of MRI. It uses differences in magnetic susceptibility between tissues and is particularly helpful for the evaluation of TAI and punctate hemorrhages in the deep subcortical white matter not visible on CT or structural MRI scans. It takes about 4 minutes to image the entire brain and, in the ED, SWI detects additional lesions 30% of the time compared to CT and structural MRI.[130] The number and volume of SWI hemorrhagic lesions correlate with clinical outcome.[131]

Diffusion Tensor Imaging. DTI uses MRI technology to analyze the movement of water molecules in the white matter of the brain and also provides the opportunity to perform tractography—visualization of major white matter pathways—to assess damaged nerve fiber tracts.[16] DTI detects white matter abnormalities and underlying cognitive deficits when conventional imaging is normal[132] and may be a sensitive marker of TAI in MTBI at acute and chronic stages of its clinical course.[133]

Computed Tomography Angiography and Magnetic Resonance Angiography. Vascular imaging such as CT angiography and MR angiography are not recommended routinely for patients with MTBI unless there is suspicion of a traumatic vascular injury, such as pseudoaneurysm, dissection, or uncontrolled hemorrhage. Typically vascular injuries occur with penetrating trauma, skull base fractures, blunt neck trauma, and/or skull base or cervical spine fractures. Independent predictors of arterial injury in blunt trauma include cervical facet subluxation or dislocation, fracture lines approaching an artery, and high-impact injury mechanisms.[134]

Ancillary Studies

Laboratory Testing. Laboratory tests are not needed for patients with isolated MTBI except for a bedside glucose level in those with a GCS score less than 15. Coagulation parameters such as INR, PT, and PTT are indicated for those with inherent coagulopathies or suspected liver disease and those on anticoagulants.

Although not in clinical use at this time, glial fibrillary acidic protein (GFAP) is a promising brain-specific biomarker for MTBI in adults and children (Fig. 34.16).[135-140] GFAP is released into serum following a MTBI within 1 hour of injury,[135,138,140] and its level is elevated in MTBI patients with axonal injury, as evidenced by MRI at 3 months postinjury.[136] It can remain elevated for seven days post-injury.[140a] In adults and children, studies have shown that serum GFAP levels distinguish MTBI patients from trauma patients without TBI and detect intracranial lesions on CT with a sensitivity of 94% to 100%.[135,138-140]

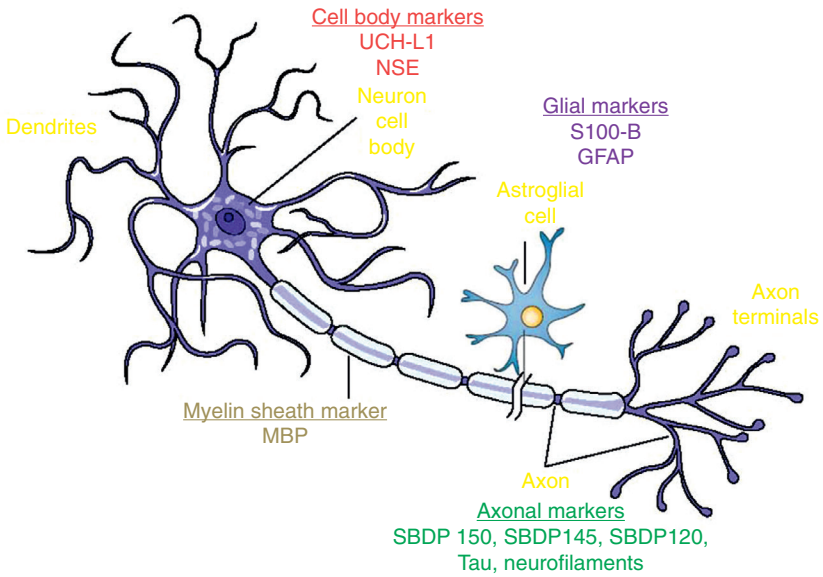

Fig. 34.16. Neuron and neuroanatomic locations of potential TBI biomarkers. S100β is the major low-affinity calcium binding protein in astrocytes that helps regulate intracellular levels of calcium. Glial fibrillary acidic protein (GFAP) is a monomeric intermediate protein found in the astroglial skeleton and in white and gray brain matter and is strongly upregulated during astrogliosis. Neuron-specific enolase (NSE) is one of the five isozymes of the gycolytic enzyme enolase found in central and peripheral neuronal cell bodies. UCH-L1 is highly abundant in neurons and was previously used as a histologic marker for neurons. Alpha II-spectrin is the major structural component of the cortical membrane cytoskeleton and is particularly abundant in axons and presynaptic terminals. Tau is an intracellular, microtubule-associated protein that is highly enriched in axons. Neurofilaments are heteropolymeric components of the neuron cytoskeleton. (From Papa L: Exploring serum biomarkers for mild traumatic brain injury. In Kobeissy F, editor: Brain neurotrauma: molecular, neuropsychological, and rehabilitation aspects in brain injury models. London, 2015, CRC Press, p 303.)

Neuropsychological Testing. Neuropsychological testing is used to assess cognitive function after MTBI and is commonly used following sports concussion. Although not routinely done in the ED, referral to a neuropsychologist is warranted for patients having persistent symptoms following a concussion.

Disposition

Most patients with MTBI can be discharged from the ED with a normal examination and after a reasonable period of ED observation (4–6 hours) or following a negative head CT scan, except in the presence of therapeutic anticoagulation, when more prolonged observation, up to 12 hours, sometimes with repeat head CT, is warranted.[141] If the emergency clinician decides that the patient with MTBI can be sent home, an appropriate early follow-up should be arranged. Providing patients and families with educational information about postconcussive syndrome and what to expect after injury helps improve outcome. Patients should also be given contact information for the brain injury association in their state. State brain injury associations can connect patients and families with support groups, programs, and professionals who understand the injury (www.biausa.org/mild-brain-injury.htm).

Patients should be discharged with instructions describing the signs and symptoms of acute and delayed complications of MTBI, have access to a telephone, and be monitored in the acute posttrauma period by a responsible sober adult. All discharge instructions should be written and told to a responsible third party. Warning signs for acute deterioration, such as inability to waken the patient, severe or worsening headaches, somnolence or confusion, restlessness, unsteadiness, or seizures, difficulties with vision, vomiting, fever, or stiff neck, urinary or bowel incontinence, and weakness or numbness involving any part of the body should prompt the caregiver to seek immediate medical help. If any doubt exists regarding the safety of the discharged patient with MTBI, a brief inpatient observation period (12–24 hours) is advisable.

If resources allow, prolonged ED observation may be practical in some circumstances. For example, intoxicated patients with MTBI who otherwise fulfill low-risk criteria should undergo serial evaluations in the ED until clinical sobriety is achieved. In these patients, a CT scan may be unnecessary, and ED observation is beneficial.

If a patient with MTBI returns to the ED because of persistent symptoms, delayed complications of injury should be sought. If a CT scan was not initially obtained, the intensity of symptoms may guide the decision to obtain a CT scan at the second visit. If a negative scan was initially obtained, the likelihood of the subsequent development of an intracranial lesion is exceedingly low.[7,8] The decision to rescan is more complex in patients from certain subgroups who may be considered more likely to develop delayed complications. These include patients on anticoagulation, those with preexisting neurologic injuries that may obscure an examination, and those with previous neurosurgical procedures (eg, ventriculoperitoneal shunts). The literature about repeat CT scanning in MTBI suggests that patients who are unchanged or improving neurologically do not benefit from a repeat CT, but repeat imaging is indicated to assess a deteriorating patient.[7,8]

Complications

In addition to being at risk for serious intracranial injuries, patients with a suspected MTBI can have elusive axonal injuries[15,142] and, over the long term, can suffer impairment of physical, cognitive, and psychosocial functioning.[143] It has been reported that more than one-third of MTBI patients do not resume work until 1 to 3 months after their injury, and lingering cognitive complaints are reported by as many as 15% of patients 1 year postinjury.[144] Recovery can also be complicated by psychiatric or substance abuse problems, health problems, concurrent orthopedic and/or traumatic injuries, chronic pain, lack of family and social support, unemployment, and litigation.[145]

Postconcussive Syndrome

Postconcussive syndrome (PCS) refers to a constellation of symptoms that include somatic (headache, dizziness, vertigo, nausea, fatigue, sensitivity to noise and light), cognitive (difficulties with attention, concentration, and memory) and affective complaints (irritability, anxiety, depression, emotional lability) that occur following a MTBI or concussion and persist beyond the expected recovery period. Affected patients commonly report headache, dizziness, memory or concentration difficulties, irritability, sleep disturbances, dizziness, and depression. There appears to be psychological and structural components to postconcussive syndrome, because patients with a history of migraines, depression, or anxiety are more likely to experience postconcussive syndrome.[146] The severity and duration of postconcussive symptoms may correlate with the abnormalities found with early functional imaging.[147] Studies of ED patients have indicated that as many as 30% of patients with a discharge diagnosis of MTBI will have symptoms at 3 months postinjury, and up to 15% will continue to be symptomatic at 1 year postinjury. In the ED, patients with more severe symptoms, such as prolonged amnesia, dizziness, headache, anxiety, noise sensitivity, or trouble with verbal recall have been shown to be at a higher risk of developing postconcussive syndrome.[148] Other factors that have been identified as conferring an increased risk for the development of PCS symptoms include prior MTBI, history of depression and/or anxiety, multiple injuries, forgetfulness or poor memory, noise and/or light sensitivity, and history of migraine.[149,150] There are a wide range of treatments being studied, including cognitive and behavioral therapies, medications, devices, dietary supplements, return to activity and rest and others.[151]

Seizures

Posttraumatic seizures occur in fewer than 1% of MTBI patients, and acute antiseizure prophylaxis is not indicated.[152,153] The cumulative incidence of posttraumatic epilepsy in the first 3 to 5 years after discharge is about 4% for patients with MTBI.[153] PTE usually develops within the first 2 years after injury. Prophylactic treatment with anticonvulsants does not prevent delayed-onset PTE and is not recommended.[154]

Posttraumatic Transient Cortical Blindness

Posttraumatic transient cortical blindness syndrome is characterized by transient visual loss, normal pupillary response, and normal funduscopic examination within hours following MTBI. This syndrome has been reported mainly in children. In most cases, vision returns to normal within minutes to hours (usually within 24 hours) following injury and leaves no neurologic sequelae. Headache, confusion, irritability, anxiety, nausea, and vomiting are common related symptoms. Although the mechanism for the transient blindness is unknown, it has been suggested that it is an abnormal vascular response to trauma, with resultant transient hypoxia and cerebral dysfunction.

Special Populations With Mild Traumatic Brain Injury

Mild Traumatic Brain Injury and Concussion in Sports

It has been estimated that 3.8 million concussions occur in the United States annually from organized and recreational sports.[155] Football, ice hockey, soccer, and lacrosse tend to have the highest

concussion incidence rates when calculated by athlete exposure.[155] Although concussion is under the umbrella of MTBI, the term *concussion* is typically used to describe MTBI in athletes. It has been suggested that sports-related concussions are associated with less disability and more rapid recovery than concussions in non-athletes. However, neuroimaging has suggested similar patterns of neuronal disruption for sports- and nonsports-related MTBI.[156] Athletes are more vulnerable to the deleterious long-term effects of MTBI because they are often subjected to repetitive trauma and greater levels of physical exertion during recovery.[156] Only recently has chronic traumatic encephalopathy (CTE) come to public attention due to autopsy findings in high-profile athletes.[157] Originally identified in boxers,[21] CTE has recently been found to occur after other organized sports, including US football, hockey, soccer, and professional wrestling.[20,22,23] Meta-analyses of neuropsychological outcomes following MTBI have suggested that recovery from impairments in the general population takes longer (weeks to months) than in athletes who tend to show recovery within 2 to 14 days following concussion. Among athletes there is a tremendous motivation to return to play. As a result, athletes often underreport symptoms, return to their regular activities prematurely, and may create the impression that they recover more quickly than they actually do.[156]

Second-impact syndrome is thought to be an exceedingly rare yet catastrophic, and sometimes fatal, consequence of repeated MTBI in sports occurring within a short period of time (days). It is defined as occurring when "an athlete who has sustained an initial head trauma, most often a concussion, sustains a second head trauma before symptoms associated with the first have fully cleared,"[158] leading to diffuse cerebral swelling. The controversy surrounding second-impact syndrome is whether a repeated head trauma is required to cause it or whether the brain swelling is the result of a single blow to the head.[159] Most reported cases of this entity, including the index case, actually sustained a single blow and did not involve a "second" impact. Furthermore, many reported cases had evidence of other structural brain injuries, such as acute subdural hematomas, in addition to the cerebral swelling. Based on the published case studies, the two groups of athletes at a higher risk of this entity are boxers and children and adolescents. Accordingly, the Centers for Disease Control and Prevention (CDC) has developed the HEADS UP Concussion in Youth Sports initiative to offer information about preventing, recognizing, and responding to a concussion to coaches, parents, and athletes involved in youth sports.

In 2013, several new or updated clinical practice guidelines and position statements were published on the diagnosis, treatment, and management of MTBI and concussion in sports. Three of these guidelines were produced by the American Medical Society for Sports Medicine,[160] American Academy of Neurology,[161] and Zurich Consensus working group.[162] It was agreed that concussion is a clinical diagnosis that is ideally made by a licensed health care provider with experience in the evaluation and management of patients with a concussion. Any athlete suspected of having a concussion should be immediately removed from play. Graded symptom and clinical sign checklists can be useful, particularly if they can be compared to preseason data.[163] The Sport Concussion Assessment Tool, third edition (SCAT3), is a standardized tool for evaluating injured athletes for concussion and can be used in athletes 13 years of age and older. It replaces the original SCAT and SCAT2, published in 2005 and 2009, respectively[162] (http://bjsm.bmj.com/content/ 47/5/259.full.pdf). The SCAT3 takes 15 to 20 minutes to complete and computes a composite score comprised of the GCS, Standardized Assessment of Concussion (SAC) score (cognitive and physical evaluation, delayed recall), and balance assessment score (modified Balanced Error Scoring System [BESS]). For children from 5 to 12 years of age, the Child-SCAT3 is used (http://bjsm.bmj.com/content/47/5/ 263.full.pdf). The SCAT3 also includes a page of information to be given to the athlete and parents after discharge.

When evaluating an athlete with suspected MTBI or concussion in the ED, obtain a comprehensive history, taking into account additional information from parents, coaches, teammates, and eyewitnesses, such as mechanism of injury, events after the injury, changes in mental status or deteriorating GCS scores, prior MTBI or concussion, current symptoms (including postconcussive symptoms), and symptoms of other potential injuries (eg, cervical spine injury). As with any trauma patient, perform a thorough examination looking for other signs of trauma, and remember to evaluate for cervical spine injury. The neurologic examination should include an assessment of pupillary reactivity, cognitive functioning, and gait and balance. As with all MTBI patients, monitoring of the injured athlete with serial assessments is critical, because signs and symptoms may evolve over hours after injury. A head CT scans is not routinely recommended but should be considered if there is clinical suspicion of a traumatic intracranial lesion. Before return to play, a gradual, a stepwise increase in general physical activity, followed by sports-specific activities, is recommended. Progression to more strenuous steps is only recommended if the athlete is asymptomatic at the current level of activity (Table 34.3). Ideally, a multidisciplinary approach to assessment and management is used, with the inclusion of sports medicine specialists from various subspecialties, as

TABLE 34.3

Graduated Return to Play Protocol

REHABILITATION STAGE	FUNCTIONAL EXERCISE AT EACH STAGE OF REHABILITATION	OBJECTIVE OF EACH STAGE
1. No activity	Symptom–limited physical and cognitive rest	Recovery
2. Light aerobic exercise	Walking, swimming, or stationary cycling, keep intensity to <70% of maximum predicted heart rate; no resistance training	Increase heart rate
3. Sport-specific exercise	Skating drills in ice hockey, running drills in soccer; no head impact activities	Add movement
4. Noncontact training drills	Progression to more complex training drills, for example, passing drills in football and ice hockey; may start progressive resistance training	Exercise, coordination, and cognitive load
5. Full-contact practice	After medical clearance, participate in normal training activities	Restore athlete's confidence; coaching staff assesses functional skills
6. Return to play	Normal game play	

Adapted from McCrory P, Meeuwisse WH, Aubry M, et al: Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. J Athl Train 48:554–575, 2013.

appropriate for the athlete's symptoms and signs.[164] Because many states have passed laws regarding concussion management in organized youth sports, it is good practice for emergency clinicians to familiarize themselves with the laws of the state in which they are practicing.[155]

Key recommendations of all three recent position statements have stated that any athlete suspected of having a concussion should not be allowed to return to play on the day of the injury.[160-162] Athletes with concussion should not return to play until they have been evaluated by a licensed health care provider with expertise in concussion management. Head CT should only be considered if intracranial lesions are suspected. Furthermore, there should be a gradual stepwise increase in physical activity.[164]

Military Personnel and Blast Injury

Mild TBI is also a common injury among soldiers who have participated in combat. Explosive blast brain injury is becoming recognized as a distinct entity from the penetrating form of blast injury and closed brain injury.[165] In recent US conflicts in Iraq and Afghanistan, over 60% of combat casualties were from explosive blast, mostly from improvised explosive devices (IEDs).[166] Other mechanisms included falls, motor vehicle accidents, fragment shrapnel, and bullet wounds. In an explosive blast, the primary injury to the brain occurs when the physical forces emanating from a detonation impart loading on the head and consequently the brain. The definition of MTBI from the American Congress of Rehabilitation Medicine is currently being applied to explosive blast TBI (including loss of consciousness, amnesia, altered mental status, and focal neurologic deficit).[165] A soldier who has been exposed to a blast may lack overt evidence of a head injury, such as lacerations, bruising, or hematomas. Recognizing an MTBI acutely is important so that the soldier can receive appropriate medical attention and be removed from combat-related duty to avoid another TBI.[166] After the first blast exposure, many soldiers do not recognize that they may have been injured, and thus will not seek medical care. The first indication of injury may be persistent postconcussive symptoms, such as headaches, vertigo, short-term memory loss, and difficulty concentrating and multitasking.[166] The clinical presentation of explosive blast MTBI can be confounded by the considerable overlap between the symptoms of MTBI and posttraumatic stress disorder (PTSD), such as mood fluctuations, sleep disturbances, and difficulty concentrating. Both may occur in the same individual. PTSD symptoms usually include bursts of anger, irritability, hypervigilance, and increased startle response.[165]

Anticoagulated Patients

Patients on Anticoagulant Medications. Most clinical decision making rules exclude patients who are taking anticoagulants such as warfarin (vitamin K antagonists), antiplatelet medications (aspirin, clopidogrel), and non–vitamin K antagonists (factor IIa or Xa inhibitors). Overall, there is a higher incidence of intracranial bleeding in TBI patients on anticoagulants following head trauma, with an incidence of up to 22% in patients with MTBI.[167-169] As a result, most practice guidelines propose that patients who sustain head trauma and are on anticoagulation treatment should undergo a CT scan without contrast and should have the INR determined, because an initial INR greater than 3 confers a much higher risk of intracranial bleeding.[168-170] Some guidelines advocate observation for the first 24 hours following MTBI, along with a second CT scan.[168] However, intracranial bleeding can be delayed beyond the first 24 hours in 1% to 3% of anticoagulated patients and can present as late as 4 weeks following injury.[168-172] Antiplatelet medications are more likely to lead to immediate traumatic intracranial hemorrhage (12%) compared with patients receiving warfarin (5%), who are more likely to have delayed bleeding.[169] In anticoagulated patients with a negative CT scan, routine admission is not required.[173] Admission should be considered for anticoagulated patients with a high risk for delayed bleeding (eg, supratherapeutic INR), those with significant comorbidities, those who live alone, and those who cannot return to the hospital in a timely manner should symptoms of delayed bleeding appear.[173,174] This underscores the importance of detailed patient instructions on discharge from the hospital.[171]

Patients with therapeutic anticoagulation and a negative initial head CT scan do not need to have their anticoagulation reversed.[175] Patients on warfarin with traumatic intracranial lesions should undergo reversal with fresh-frozen plasma or prothrombin complex concentrates, and vitamin K should also be initiated in the ED.[176] However, the transfusion of platelets in patients on antiplatelet medications does not reduce mortality. Dabigatran can be reversed with hemodialysis, but rivaroxaban and apixaban cannot.[177] Idarucizumab, a Fab fragment of a monoclonal antibody directed specifically against dabigatran, was approved by the US Food And Drug Administration in 2015 for emergency surgery and urgent procedures and for life-threatening or uncontrolled bleeding. The recommended dose for idarucizumab is 5 g (2.5 g/vial), administered as two consecutive 2.5-g IV infusions or bolus injection by injecting both vials consecutively, one after another, via syringe. Andexanet alfa is a class-specific antidote targeted to reverse the oral direct factor Xa inhibitors as well as the indirect inhibitor, enoxaparin; ciraparantag is a universal antidote targeted to reverse the direct thrombin and factor Xa inhibitors, as well as the indirect inhibitor, enoxaparin.[178] Studies evaluating these antidotes in the setting of TBI are currently lacking.

Patients With Inherent Bleeding Disorders. The most serious site of bleeding for children and adults with inherent bleeding disorders, such as hemophilia, is the CNS. Intracranial hemorrhage in patients with hemophilia can occur spontaneously or following mild head trauma. Over 50% of hemophiliacs with MTBI who have intracranial bleeding are initially asymptomatic, with a normal neurologic examination. Therefore, patients with inherent bleeding disorders should undergo head CT following a MTBI.[179] There should be a low threshold for factor replacement (eg, factor VIII or IX, cryoprecipitate, fresh-frozen plasma) in patients with severe hemophilia or in those with MTBI symptoms, even prior to performing head CT.[180]

Head Trauma in Older Adults

Older patients have increased morbidity and mortality from TBI and have higher rates of intracranial injuries following head trauma.[1,181,182] They also experience MTBI more frequently than severe TBI. Furthermore, frequent falls put them at risk for repetitive brain injury. Within 7 months following a mild or moderate TBI, older patients can show a decline in language, memory, executive function, activities of daily living, and mood compared with their preinjury functioning and compared to controls.[183] Accordingly, older adults with preinjury warfarin or clopidogrel use are at an increased risk for unfavorable long-term neurologic outcomes compared with similar patients without preinjury use of these medications.[184]

With age, the brain atrophies and creates more space within the cranial vault for blood to accumulate before symptoms appear. Moreover, with atrophy, comes stretching of bridging veins that may tear and lead to subdural hematomas more easily. Therefore, older adults can have significant hemorrhage into their brain and not show signs of deterioration, especially if their baseline cognitive functioning is impaired. Occult intracranial hemorrhages occur in over 2% of older patients with head trauma.

Alcohol abuse is one of the most prevalent comorbid conditions found in older patients admitted to the hospital with TBI,[185] so screening for alcohol abuse in older patients with head trauma is recommended.[186] Elder abuse is an important consideration in this population as well and should be assessed during the ED evaluation.[187]

The presence of comorbid medical conditions, use of anticoagulants, preexisting cognitive deficits, polypharmacy, alcohol consumption, and unique physiology of the aging brain make it challenging for the emergency clinician to detect brain injury.[186] Even low-mechanism falls should prompt health care providers to consider the possibility of brain injury in older patients. Many clinical decision rules recommend CT for patients older than 60 to 65 years following any suspected MTBI. Reducing the risk of falls in older adults can reduce the risk of TBI.[188] Particular attention needs to be given to polypharmacy, drug interactions, safety issues in the living environment, risk of elder abuse, and covert alcohol consumption.[186,188]

KEY CONCEPTS

- *Head trauma* is a broad term describing an external trauma to the craniofacial area of the body from blunt, penetrating, blast, rotational, or acceleration-deceleration forces, the term *head injury* refers to a clinically evident injury on physical examination, and the term *brain injury* indicates an injury to the brain itself.
- TBI is often categorized into mild (GCS score, 13–15), moderate (GCS score, 9–12), and severe (GCS score, 3–8), but this actually represents a spectrum of injury. Patients with a presentation GCS score of 13 to 15 who are stable or improving are exceedingly unlikely to have CT findings that warrant intervention.

Severe and Moderate Traumatic Brain Injuries

- Secondary systemic insults such as hypoxia and hypotension worsen neurologic outcome and should be corrected as soon as detected.
- Noncontrast head CT is the imaging modality of first choice when TBI is suspected.
- The motor component of the GCS is the strongest predictor of outcome following TBI.

Penetrating Head Trauma

- Anticonvulsant prophylaxis with phenytoin and broad-spectrum antibiotics should be given to patients with penetrating brain injuries for 7 days postinjury.

Mild Traumatic Brain Injury

- Patients can deteriorate from an expanding intracranial hematoma after what appears clinically to be MTBI, and should undergo serial evaluations, including serial GCS scoring.
- An MTBI can be easily overlooked when an alert patient presents with other more obvious traumatic injuries. Specifically, ask patients about symptoms of disorientation, confusion, amnesia, or disordered awareness (with or without loss of consciousness).
- Imaging of patients in this population should follow a validated guideline, such as the Canadian CT Head Rule and New Orleans Criteria. Emergency clinicians should work collaboratively to select the system thought to be most applicable for their setting and patient population.
- Alcohol and drug use affects the GCS score and significantly obscures the neurologic examination. Intoxicated individuals are high-risk patients.
- Most patients with MTBI can be discharged from the ED with a normal examination and after a reasonable period of ED observation (4–6 hours) or following a negative head CT.
- Patients should be discharged with instructions describing the signs and symptoms of acute and delayed complications of MTBI. All discharge instructions should be written and relayed to a responsible third party.

Special Populations

- Any athlete suspected of having a concussion should be immediately removed from play.
- Athletes with concussion should not return to play until they have been evaluated by a licensed health care provider with expertise in concussion management. There should be a gradual stepwise increase in physical activity.
- Older adults can have significant hemorrhage into their brain and not show signs of deterioration, especially if their baseline cognitive functioning is impaired. Patients older than 60 years should have a CT scan obtained.
- Falls in older adults, including low-mechanism falls, should prompt health care providers to consider the possibility of brain injury.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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CHAPTER 34: QUESTIONS & ANSWERS

34.1. Injured brain ischemia may be seen with partial pressure of carbon dioxide (Pco_2) levels below what value?
A. 35 mm Hg
B. 30 mm Hg
C. 25 mm Hg
D. 20 mm Hg

Answer: D. Cerebral vasculature is exquisitely sensitive to Pco_2 levels. The degree of vasoconstriction below a Pco_2 of 20 mm Hg may be so severe as to induce ischemia. Modest hyperventilation to a target of 30 to 35 mm Hg is recommended once acute resuscitation is completed.

34.2. Which of the following statements is true regarding cerebral blood flow (CBF), cerebral perfusion pressure (CPP), and intracranial pressure (ICP)?
A. CBF autoregulation is lost below a CPP of 60 mm Hg.
B. CPP closely parallels diastolic blood pressure.
C. Normal ICP is 65 to 195 mm Hg.
D. CPP = mean arterial pressure (MAP) − ICP.
E. The only resistance to CBF is ICP.

Answer: D. CBF depends on CPP, which is the blood flow pressure gradient. CBF resistance is provided by mean systemic venous pressures and ICP, predominantly by the latter. CPP closely parallels MAP offset by ICP; thus, the formula CPP = MAP − ICP. ICP is estimated clinically by the central venous pressure unless a ventricular catheter is in place and ICP can be directly determined. CBF autoregulation is lost below a CPP of 40 mm Hg. Normal ICP is 5 to 15 mm Hg or 65 to 195 mm H_2O.

34.3. Which of the following parameters are associated with a worsened outcome after traumatic brain injury (TBI)?
A. Both D and E
B. Core body temperature > 37.5° C (99.5° F)
C. None of the above
D. Partial pressure of oxygen (Po_2) < 60 mm Hg
E. Systolic blood pressure < 90 mm Hg

Answer: A. The following are associated with worsened outcomes after TBI:
Hematocrit (Hct) < 30%
Temperature > 38.5° C (101.3° F)
Systemic blood pressure (SBP) < 90 mm Hg
Po_2 < 60 mm Hg

34.4. A 27-year-old man presents after a motor vehicle collision (MVC) with a severe closed head trauma. On examination, you calculate a Glasgow Coma Scale (GCS) score of 5 and a left dilated pupil, with a sluggish pupillary reflex compared with the right. What other finding will your examination likely reveal?
A. Left carotid bruit
D. Left foot weakness
E. Loss of controlled pain/temperature sensation
B. Right carotid bruit
C. Right-sided hemiparesis

Answer: C. Uncal herniation is the most common posttraumatic herniation syndrome. The initial pressure compresses the third cranial nerve (CN III), causing ipsilateral pupillary sluggishness, ptosis, anisocoria, and impaired extraocular movements. Contralateral hemiparesis can develop early after an initial normal motor examination. In some cases, the contralateral uncus is compressed, resulting in ipsilateral weakness (Kernohan notch syndrome).

34.5. A 24-year-old man presents with a closed head injury after a MVC. The physical examination is remarkable for a sluggish left pupil, right-sided hemiparesis, and a GCS score of 12. What should be the next step in this patient's management?
A. 3% hypertonic saline IV
B. Intubation and hyperventilation
C. Mannitol, 1 g/kg IV
D. Methylprednisolone IV
E. Pentobarbital IV

Answer: B. The most rapid effect on ICP reduction is achieved via intubation and moderate hyperventilation to a Pco_2 of 30 to 35 mm Hg. The effect peaks within minutes and should be considered a short-term intervention, with an expected ICP reduction of 25%. Prolonged hyperventilation may be dangerous. Steroids may worsen outcome after TBI. Mannitol is generally efficacious and exerts an effect within minutes and lasts hours. Other neuroprotective effects include volume expansion, viscosity reduction, CBF improvement, and free radical scavenging. Hypertonic saline data are encouraging but inconclusive; stronger data exist in the pediatric literature. Barbiturates exert a modest ICP-lowering effect because of their lowering of the cerebral metabolic rate and oxygen demand.

34.6. What is the minimum time after becoming asymptomatic that an individual should refrain from playing sports after a concussion if no loss of consciousness (LOC) or prolonged posttraumatic amnesia occurred at the time of injury?
A. 24 hours
B. 48 hours
C. 1 week
D. 2 weeks
E. 1 month

Answer: C. All current recommendations for return to play after a sports-related concussion state that players with concussion should not return to play for at least 1 week after they have become asymptomatic. This is usually increased to at least a symptom-free month for an extended LOC or prolonged posttraumatic amnesia occurring at the time of the concussion.

34.7. A 15-year-old boy presents after being hit in the head with a baseball. He has a GCS score of 7 and a large hematoma of his scalp, anterior and superior to his right ear. In addition, he is noted to have unequal pupils and a sluggish papillary light reflex of the right eye. Which of the following is most likely in this patient?
A. Central transtentorial herniation
B. Cerebellotonsillar herniation
C. Downward transtentorial herniation
D. Uncal herniation
E. Upward transtentorial herniation

Answer: D. Uncal herniation is often associated with traumatic extra-axial hematomas in the lateral middle fossa of the temporal lobe. The classic signs and symptoms are caused by compression of the ipsilateral uncus of the temporal lobe on the U-shaped edge of the tentorium cerebelli as the brain is forced through the tentorial hiatus. As compression of the uncus begins, CN III is compressed; anisocoria, ptosis, impaired extraocular movements, and a sluggish pupillary light reflex develop on the side ipsilateral to the expanding mass lesion. This phase may last for minutes to

hours, depending on how rapidly the expanding lesion is changing. As the herniation progresses, compression of the ipsilateral oculomotor nerve eventually causes ipsilateral pupillary dilation and nonreactivity.

Initially, in the uncal herniation process, the motor examination can be normal, but contralateral Babinski responses develop early. Contralateral hemiparesis develops as the ipsilateral peduncle is compressed against the tentorium. With continued progression of the herniation, bilateral decerebrate posturing eventually occurs; decorticate posturing is not always seen with the uncal herniation syndrome. In some patients, the contralateral cerebral peduncle is forced against the opposite edge of the tentorial hiatus. Hemiparesis is then detected ipsilateral to the dilated pupil and mass lesion, termed *Kernohan notch syndrome*, and causes false-localizing motor findings. As uncal herniation progresses, direct brainstem compression causes additional alterations in the LOC, respiratory pattern, and cardiovascular system. Mental status changes may initially be subtle, such as agitation, restlessness, or confusion, but soon lethargy occurs, with progression to frank coma. The patient's respiratory pattern may initially be normal, followed by sustained hyperventilation. With continued brainstem compression, an ataxic respiratory pattern develops. The patient's hemodynamic status may change, with rapid fluctuations in blood pressure and cardiac conduction. Herniation that is uncontrolled progresses rapidly to brainstem failure, cardiovascular collapse, and death.

34.8. Central pontine myelinolysis is a potential adverse event associated with the administration of which of the following medications?

A. Etomidate
B. Hypertonic saline
C. Mannitol
D. Methylprednisolone
E. Pentobarbital

Answer: B. Central pontine myelinolysis is a potentially adverse event associated with hypertonic saline administration.