

CHAPTER 184

Altered Mental Status

DISORDERS OF CONSCIOUSNESS

Consciousness is a cortical function that allows for awareness of self and environment (place, time). **Arousal** represents the system that initiates and maintains consciousness and is mediated by the reticular activating system (RAS), which extends from the pons through the midbrain to the hypothalamus and thalamus. Acute changes in consciousness vary in degree from mild lethargy and confusion to deep coma. **Lethargic** patients have difficulty maintaining an aroused state. Patients who are **obtunded** have decreased arousal but are responsive to stimuli. **Stupor** is a state of responsiveness to pain but not to other stimuli. **Coma** is a state of unresponsive unconsciousness and is caused by dysfunction of the cerebral hemispheres bilaterally, the bilateral thalami, and/or the brainstem.

Acute Disorders of Consciousness

In childhood, the most common causes of altered mental status are toxins, infections, head trauma, hypoxia-ischemia (e.g., cardiac arrest, near-drowning), and seizures (e.g., postictal state, subclinical status epilepticus) (Table 184.1).

Assessment

The most common cause of long-term morbidity in a patient with depressed consciousness is hypoxia; therefore airway, breathing, and circulation are addressed first. Vital signs, including pulse oximetry, must be assessed. Breathing patterns may provide important clues to the neurologic localization and etiology of the depressed consciousness. A low respiratory rate may be associated with central nervous system (CNS) depressants or increased intracranial pressure (ICP). Tachypnea may be due to hypoxia, metabolic acidosis, or fever, but in more ominous situations may be due to brainstem herniation. Systemic diseases such as sepsis, ingestions, or cardiac failure may lead to shock. Hypotension may also result from severe traumatic brain injury (TBI) or spinal cord injury. Hypertension may be due to increased ICP. The glucose level should also be checked immediately because hypoglycemia is a rapidly treatable cause of altered mental status. Physical examination searches for clues as to the cause of altered consciousness, such as unusual odors, needle tracts, trauma, dehydration, or organ system failure.

The **Glasgow Coma Scale** (see Table 42.1) can be used to assess unresponsive patients regarding their best verbal and motor responses and eye opening to stimulation with scores ranging from 3–15 points.

Testing the structures immediately adjacent to the reticular system is essential. Changes in pupillary response generally suggest midbrain lesions, though pontine lesions can cause pinpoint pupils. In comatose patients, eye movements are observed or elicited with the **oculocephalic vestibular reflexes** (doll's-eye maneuver) (see Chapter 179). If the oculocephalic responses are not elicited or are unclear, the **oculovestibular response** (cold caloric stimulation) is elicited by flushing cold water into the external ear canal. In a comatose patient, cold

water irrigation into the ear canal elicits a tonic eye deviation toward the irrigated ear if the brainstem is functioning.

Body posture at rest and after noxious stimulation can indicate the anatomic level responsible for the alteration of consciousness. Posturing in response to noxious stimulation indicates more serious neurologic conditions. **Decorticate posturing** consists of rigid extension of the lower extremities, flexion and supination of the arms, and fisting of the hands. It indicates bilateral cortical or subcortical abnormalities or herniation, with preserved brainstem function. **Decerebrate posturing** (rigid extension and internal rotation of the upper and lower extremities) is associated with herniation with midbrain compression or toxic-metabolic abnormalities. These postures may be symmetric or asymmetric. A structural cause is most likely when posturing is asymmetric, whereas symmetric posturing may suggest an underlying toxic-metabolic etiology.

Etiology

The evolution of change in consciousness is an important clue to the etiology. A detailed history and physical examination usually provide sufficient clues to differentiate among the three major diagnostic categories producing coma: **metabolic/toxic**, **infectious**, and **structural**. In most clinical situations, the cause of coma is readily identified.

Care must be taken to investigate background medical conditions that may produce a decline in consciousness (e.g., diabetes mellitus, leukemia, kidney failure, liver disease). Metabolic derangements are common causes of altered mental status and are suggested by spontaneous fluctuations in the level of consciousness, tremors, myoclonus, asterixis, visual and tactile hallucinations, and deep coma with preservation of pupillary light reflexes. Disturbances of blood chemistries (e.g., glucose, sodium, calcium, bicarbonate, blood urea nitrogen, ammonia) may produce depressed mental status, as can hypoxemia and cerebral ischemia (inadequate cerebral perfusion). Acute metabolic or toxic disorders usually produce a hypotonic, limp state, but hypertonia, rigidity, or posturing can be seen in some causes of coma (e.g., hypoglycemia, hepatic encephalopathy, short-acting barbiturates).

Intoxication and ingestion are common causes of acute alteration of consciousness. A thorough history must be taken to search for the offending agent (see Chapter 45). A history of social stressors, depression, substance disorder, or other mental health issues raises concern for self-inflicted injury or toxic ingestion. A subacute course of somnolence progressing to difficulty arousing from a deep sleep (stupor) over hours suggests drug intoxication or organ system failure (e.g., kidney, liver) producing a metabolic encephalopathy. CNS infection usually causes abrupt alteration of mental status, although viral meningoencephalitis (particularly herpes simplex virus) can present with subacute alterations in mental status. The presence of fever, petechiae, chills, and sweats suggests infection. Prodromal photophobia and pain on movement of the head or eyes are symptoms of meningeal irritation. Preceding symptoms, such as abdominal pain, diarrhea, sore throat, conjunctivitis, cough, or rash, point toward viral encephalitis or postinfectious syndrome as the cause of the altered consciousness.

Structural processes, such as hemorrhage, acute hydrocephalus, or stroke, can cause sudden depressed consciousness

TABLE 184.1 Causes of Altered Mental Status and Diagnostic Approach

CAUSES	
Infection	
Meningitis	
Encephalitis (e.g., herpes simplex virus, arboviruses)	
Brain abscess or subdural empyema	
HIV	
Tuberculosis meningitis	
Toxic shock syndrome	
Postinfectious (e.g., acute disseminated encephalomyelitis)	
Trauma	
Abusive head trauma	
Hemorrhage (e.g., epidural, subdural, subarachnoid)	
Brain contusion	
Concussion	
Diffuse axonal injury	
Toxins (Intoxication or Withdrawal)	
Ethanol	
Narcotics	
Barbiturates	
Antihistamines	
Anticonvulsants	
Iron	
Acetaminophen	
Aspirin (Reye syndrome)	
Illicit drugs	
Lead poisoning	
Thiamine deficiency (Wernicke encephalopathy)	
Hypoxia-Ischemia	
Near-drowning	
Post–cardiopulmonary arrest (e.g., cardiac arrhythmia, obstructive cardiomyopathy)	
Carbon monoxide intoxication	
Perinatal asphyxia	
Strangulation	
Epilepsy	
Subclinical (or nonconvulsive) status epilepticus	
Postictal states	
Stroke	
Arterial ischemic stroke	
Cerebral sinovenous thrombosis	
Hemorrhage	
Increased Intracranial Pressure	
Brain tumor	
Cerebral edema	
Hydrocephalus	
Migraine	
Systemic Disorders	
Gastrointestinal (e.g., intussusception)	
Vasculitis (e.g., systemic lupus erythematosus)	
Hepatic failure	
Hypertensive encephalopathy	
Reye syndrome	
Endocrine disorders (e.g., adrenal insufficiency, thyroid disorders)	
Renal disorders (e.g., uremia)	
Demyelinating disorders (e.g., multiple sclerosis, ADEM)	
Metabolic Derangements	
Hypoglycemia	
Hyponatremia or rapid correction	
Hypernatremia or rapid correction	
Hyperosmolality or rapid correction	
Hypercapnia	
Hyperammonemia	
Inborn errors of metabolism	
Diabetes mellitus—ketoacidosis or hypoglycemia	
DIAGNOSTIC APPROACH	
Routine laboratory testing	
Glucose	
Sodium, potassium, calcium, magnesium, chloride, bicarbonate, BUN, creatinine, AST, ALT, blood gases, ammonia, TSH	
Blood, urine analyses for toxic substances	
Blood, urine cultures if infection suspected	
Skeletal survey, ophthalmologic examination if child abuse suspected	
Neuroimaging	
Head CT	
Brain MRI, MRA, MRV	
CSF analysis (if no evidence for increased intracranial pressure on neuroimaging)	
Opening pressure	
White and red blood cell counts, protein, glucose	
Culture (±viral PCR testing)	
Electrodiagnostic studies	
EEG	
Secondary laboratory testing (if cause remains unknown)	
Lead level, pyruvate, lactate, serum amino acids, urine organic acids, acylcarnitine profile	

ADEM, Acute disseminated encephalomyelitis; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CSF, cerebrospinal fluid; CT, computed tomography; EEG, electroencephalogram; MRI/MRA/MRV, magnetic resonance imaging/angiography/venography; PCR, polymerase chain reaction; TSH, thyroid-stimulating hormone.

in children. An evolution of headache and morning vomiting suggests increased ICP. A gradual fading of alertness or declining school performance over preceding weeks suggests an expanding intracranial mass, subdural hematoma, or chronic infection.

Clinical Manifestations

Papilledema or paralysis of cranial nerves III (dilated “blown” pupil, eye deviated down and laterally) or VI (unable to move eye laterally) in a patient with depressed consciousness is strong evidence of elevated ICP, a medical and neurosurgical

TABLE 184.2 Herniation Syndromes

LOCATION	ETIOLOGY	DESCRIPTION	CLINICAL FINDING(S)
Transtentorial Unilateral (uncal) or bilateral	Supratentorial mass lesions, diffuse brain swelling, focal edema, or acute hydrocephalus	Downward displacement of supratentorial brain tissue into infratentorial compartment (below tentorium), compressing the cerebral peduncles, midbrain, third cranial nerve, and posterior circulation	Headache Altered consciousness Dilated ipsilateral pupil Cranial nerve III palsy Hemiparesis Decerebrate posturing Cushing triad Respiratory arrest
Subfalcian	Increased pressure in one hemisphere (mass, focal edema)	Brain tissue displaced under the falx cerebri, compressing the anterior cerebral artery	Weakness, bladder incontinence, coma
Foramen magnum	Cerebellar mass or edema	Downward displacement of cerebellar tonsils, compressing the medulla and upper spinal cord	Bradycardia, bradypnea, hypertension, death

emergency. **Herniations** can occur under the falx, through the tentorial notch, or into the foramen magnum (Table 184.2). Recognition of the signs associated with the major herniation syndromes can be lifesaving if emergent neuroimaging and treatment are initiated. **Uncal herniation** implies displacement of one mesial temporal lobe over the tentorial edge, producing a unilateral third nerve palsy and hemiparesis (which can be ipsilateral due to compression of the contralateral cerebral peduncle or contralateral if the uncus compresses the ipsilateral peduncle). Another type of transtentorial herniation, **central herniation**, occurs when both temporal lobes and the diencephalon (thalamus and hypothalamus) herniate through the tentorium. A progressive loss of consciousness accompanied by miosis, decorticate posturing, and Cheyne-Stokes respirations (rhythmic waxing and waning of respiratory amplitude) suggests incipient transtentorial herniation. Late findings include decerebrate posturing and Cushing triad (bradycardia, irregular respirations, hypertension).

Laboratory and Diagnostic Imaging

Evaluation is guided by the history and physical examination. Clinicians should rapidly consider causes of unresponsiveness that could easily and quickly be reversed (see Table 184.1). If trauma or immersion injuries are suspected, neck manipulation should be avoided until cervical spine imaging excludes vertebral fracture or subluxation.

Computed tomography (CT) remains the preferred imaging technique in emergency situations because it can be performed rapidly, and it accurately identifies intracranial acute hemorrhages, large space-occupying lesions, edema, and shifts of the midline. The initial head CT scan is done without contrast to identify blood and calcifications. Contrast can be administered to identify inflammatory and neoplastic lesions but is not usually required for identification of major lesions.

Examination of the cerebrospinal fluid (CSF) may establish the cause of the alteration of consciousness. Lumbar puncture (LP) in a patient with elevated ICP can result in transtentorial herniation, so neuroimaging must be performed before LP if elevated ICP is suspected. The presence of red blood cells in the CSF may suggest primary subarachnoid hemorrhage,

parenchymal hemorrhage, or hemorrhagic infection (e.g., herpes simplex virus), or trauma from the procedure causing bleeding into the subarachnoid space. White blood cells in the CSF usually denote infectious meningitis or meningoencephalitis but may also be associated with autoimmune disorders or parainfectious syndromes. Elevated CSF protein is seen in patients with meningeal inflammation, tumors, or white matter diseases, whereas abnormal CSF glucose levels are also markers of infection and inflammation (e.g., neurosarcoidosis).

Treatment

Beyond supportive measures, the etiology of altered mental status determines the treatment. Ingestions may necessitate charcoal administration, forced diuresis, dialysis, or specific antidotes (see Chapter 45). Infections are treated with appropriate antibiotics or antiviral agents (see Chapters 100 and 101). If mental status changes are due to increased ICP, acute medical interventions (e.g., osmotic agents, a trial of hyperventilation, and elevating the head of the bed to 30 degrees) must be instituted, emergent neuroimaging performed, and urgent neurosurgical consultation obtained. Structural brain lesions may necessitate medical treatment of increased ICP or surgical excision.

Prognosis

The outcome of altered mental status relates to many variables, including etiology. Intoxication typically carries a good prognosis, whereas herniation carries a poor prognosis. Other prognostic factors include the duration of coma and the age of the patient; children have better outcomes than adults. Complete recovery from traumatic coma of several days' duration is possible in children; however, many survivors of prolonged coma are left in a persistent vegetative state or with severe neuropsychiatric disability.

Transient, Recurrent Depression of Consciousness

Episodic alteration or depression of consciousness with full recovery is usually due to seizure, migraine, syncope, or

metabolic abnormality (e.g., hypoglycemia). Consciousness can be impaired during seizures or in the postictal state. Nonconvulsive (subclinical) status epilepticus (generalized or focal) can directly impair consciousness and is a common complicating factor for infants and children with acute neurologic disorders (e.g., head trauma, meningitis, stroke, following convulsive status epilepticus). **Confusional migraines** can last hours and be accompanied by agitation, ataxia, cortical blindness, vertigo, or cranial nerve palsies; headache may precede or follow the neurologic signs.

Syncope is one of the most common causes of abrupt, episodic loss of consciousness. Neurocardiogenic syncope, cardiac arrhythmia, or obstructive cardiomyopathy can cause recurrent episodes of loss of consciousness. Twothirds of children with syncope have irregular, myoclonic movements as they lose consciousness (anoxic seizures), which must be distinguished from epilepsy (unprovoked seizures). Children with unexplained syncope require a complete cardiac evaluation (see [Chapter 140](#)).

Metabolic derangements, particularly **hypoglycemia**, can give rise to episodes of lethargy, confusion, seizures, or coma. Several other metabolic disorders cause recurrent bouts of **hyperammonemia** (see [Chapter 53](#)). Symptoms include nausea, vomiting, lethargy, confusion, ataxia, hyperventilation, and coma.

INCREASED INTRACRANIAL PRESSURE

Etiology

When the cranial sutures are fused, the skull becomes a rigid container enclosing a fixed volume, including the brain (80–85%), CSF (10–15%), and blood (5–10%). The exponential relationship between the volume within a closed container and pressure results in a massive increase in ICP as intracranial volume increases. The brain accommodates increased ICP initially by expelling CSF and blood from the intracranial compartment into the spinal subarachnoid space. When the limits of this accommodation are reached, the brain itself begins to shift in response to the continuing elevation of ICP. Brain shifts may cause herniations across the dural extensions or skull barriers (see [Table 184.2](#)).

The most common causes of increased ICP include mass lesions, hydrocephalus, cerebral edema due to trauma or infection, and idiopathic intracranial hypertension (pseudotumor cerebri) ([Tables 184.3 and 184.4](#)).

Mass lesions (e.g., brain tumor, abscess, hemorrhage) may produce increased ICP not only by virtue of large size, but by blockage of CSF pathways, blockage of venous outflow, or creation of vasogenic cerebral edema. Brain abscesses usually present as mass lesions producing focal neurologic signs and increased ICP. Symptoms of infection, such as fever, malaise, anorexia, and stiff neck, may be subtle or absent. Children with chronic cardiac or pulmonary disease may embolize infected material to the brain, predisposing them to brain abscesses. Brain tumors in children are most often located in the posterior fossa and can result in elevated ICP due to obstruction of CSF flow through the cerebral aqueduct and fourth ventricle. Brain tumors and abscesses can both result in vasogenic edema, which further increases ICP.

Hydrocephalus from congenital abnormalities or due to an intracranial mass is typically characterized by a slowly evolving syndrome of increased ICP extending over weeks or months.

TABLE 184.3 Causes of Increased Intracranial Pressure

MASS EFFECT

Hydrocephalus
Hemorrhage
Tumor
Abscess
Cyst
Inflammatory mass
Arterial ischemic stroke with edema
Cerebral sinovenous thrombosis

DIFFUSE EDEMA

Hypoxic-ischemic injury
Trauma
Infection
 Meningitis
 Encephalitis
Hypertension
Metabolic derangement or toxin
 Hyponatremia
 Diabetic ketoacidosis
 Dialysis disequilibrium syndrome
 Reye syndrome
 Fulminant hepatic encephalopathy
 Pulmonary insufficiency with hypercarbia
 Lead intoxication
Idiopathic intracranial hypertension (pseudotumor cerebri)
Drugs
 Withdrawal of long-term steroid administration
 Endocrinologic disturbance
Obesity

CSF is continuously produced by the choroid plexus within the lateral, third, and fourth ventricles. CSF circulates through the ventricular systems and is then absorbed, predominantly by the arachnoid villi into the large dural sinuses. Hydrocephalus is due to the obstruction of CSF flow anywhere along its course (see [Table 184.4](#)). **Obstructive hydrocephalus** is caused by a block before the CSF flows to the subarachnoid space, usually within the fourth ventricle or at the level of the aqueduct. Congenital hydrocephalus can result from CNS malformations, intrauterine infection, intraventricular hemorrhage, genetic defects, trauma, and teratogens. Acquired hydrocephalus may be caused by CNS infections, brain tumors, or hemorrhage. Impairment of CSF flow within the subarachnoid space or impairment of absorption is known by the misnomer “communicating hydrocephalus,” where there is actually extraventricular obstruction of CSF flow. Hydrocephalus caused by overproduction of CSF without true obstruction is seen in choroid plexus papillomas, which account for 2–4% of childhood intracranial tumors.

Bacterial meningitis may produce increased ICP by blockage of CSF pathways, cytotoxic cerebral edema, increase in cerebral blood flow, or multifocal cerebral infarctions (see [Chapter 100](#)). Most children with bacterial meningitis can undergo LP safely because the brain swelling is diffuse and distributed evenly throughout the brain and spinal CSF compartments. A few

TABLE 184.4 Causes of Hydrocephalus

PATHOGENIC MECHANISM	ETIOLOGIC DISORDER/CONDITION
Obstruction of CSF pathways	Intraventricular foramina (Monro) obstruction Parasellar mass (craniopharyngioma, germinoma, pituitary tumor) Intraventricular tumor (ependymoma) Tuberous sclerosis with subependymal giant cell astrocytoma Aqueduct of Sylvius (cerebral aqueduct) obstruction/stenosis Midbrain or pineal region tumor Impaired flow from the fourth ventricle foramina of Luschka and Magendie Dandy-Walker malformation Arnold-Chiari malformation Bone lesions of the cranial base (achondroplasia, rickets) Postinfectious or postinflammatory Posthemorrhagic
Overproduction of CSF	Choroid plexus papilloma
Defective reabsorption of CSF (extraventricular obstruction or communicating hydrocephalus)	Hypoplasia of the arachnoid villi Postinfectious or posthemorrhagic destruction of arachnoid villi or subarachnoid fibrosis Extensive cerebral sinovenous thrombosis

CSF, Cerebrospinal fluid.

patients with meningitis, however, develop transtentorial herniation within a few hours of LP. Focal neurologic signs, poorly reactive pupils, and a tense fontanel are contraindications to LP in a patient with suspected bacterial meningitis.

Idiopathic intracranial hypertension (pseudotumor cerebri) is a cause of increased ICP with normal brain imaging. Patients exhibit a daily debilitating headache associated with diplopia, abducens palsy, transient visual obscurations, and papilledema. If untreated, permanent visual field loss may develop. The syndrome has been associated with ingestion of medications (e.g., tetracycline, vitamin A, oral contraceptive agents) and endocrine disturbances (e.g., thyroid disease, Addison disease). Most commonly, this condition is idiopathic and affects children who are otherwise well except for being overweight, with rapid weight gain being a predisposing factor. Weight loss and cessation of triggering medications are mainstays of treatment. Additional treatment with acetazolamide, another diuretic, or topiramate may be necessary. Infrequently, chronic papilledema from persistent idiopathic intracranial hypertension produces visual impairment, and more aggressive management is required (optic nerve fenestration) to preserve visual function.

Clinical Manifestations

The symptoms and signs of increased ICP include headache, vomiting, lethargy, irritability, altered consciousness, sixth nerve palsy, strabismus, diplopia, and papilledema. Classically,

headaches related to increased ICP worsen with time and with maneuvers that increase ICP such as lying down (nocturnal headaches or upon awakening), bending over, coughing, or Valsalva maneuvers. Specific signs of increased ICP in the infratentorial compartment (posterior fossa) include stiff neck and head tilt. Focal neurologic deficits reflect the site of the lesion that is producing the increased ICP and may include hemiparesis from supratentorial lesions, or ataxia and cranial nerve palsies from infratentorial lesions. Critically increased ICP may result in herniation syndromes, as discussed earlier. In addition, the **Cushing triad** of elevated blood pressure, bradycardia, and irregular respirations is a late sign of critically elevated ICP. Infants who have an open fontanel may present with less specific symptoms such as irritability, lethargy, and poor feeding. Specific signs of increased ICP in infants consist of a bulging fontanel, suture diastasis, distended scalp veins, a persistent downward deviation of the eyes (“sunsetting”), and rapid growth of head circumference. Manifestations of increased ICP may evolve slowly when there is slow growth of a mass or incomplete obstruction allowing for transependymal absorption of CSF into the veins. On the other hand, symptoms may develop rapidly in the setting of acute intracranial hemorrhage or abrupt CSF obstruction.

Laboratory and Diagnostic Studies

The cause of increased ICP is determined by brain imaging with either CT or magnetic resonance imaging (MRI). CT is rapid, readily available, and provides clear visualization of an acute hemorrhage, dilated ventricles, or mass lesions. Diffuse brain swelling produced by hypoxic-ischemic injury, meningitis, encephalitis, metabolic abnormalities, or toxins may be better delineated by MRI. MRI is also superior to CT for visualizing the posterior fossa and cortical contusions. To avoid radiation from head CT, many centers now use limited sequences to allow for a rapid MRI without sedation as an initial screen for acute causes of elevated ICP. LP is the diagnostic test for infection and for documentation of elevated ICP (essential for patients with idiopathic intracranial hypertension). However, LP is contraindicated in the setting of an intracranial mass lesion or clear hydrocephalus because withdrawal of CSF can change pressures between the intracranial compartments and promote brainstem shifts and herniation. Idiopathic intracranial hypertension is diagnosed when direct measurement of ICP with a manometer during LP reveals elevated ICP in the absence of other CSF or imaging abnormalities.

Treatment of Elevated Intracranial Pressure

The treatment of acute increased ICP should be carried out in an intensive care setting with continuous monitoring and vigilant attention to vital signs. The first goal is stabilization of the cardiopulmonary status. Once the child is stabilized, the head is placed at midline, and the head of the bed is elevated to 30 degrees. Indications for endotracheal intubation include refractory hypoxia, hypoventilation, Glasgow Coma Score of 8 or less, loss of airway protective reflexes, and acute herniation requiring controlled hyperventilation. Slight hyperventilation to a P_{aCO_2} not lower than 35 mm Hg produces rapid, but transient, reduction in ICP by cerebral vasoconstriction, leading to decreased cerebral blood volume. More aggressive hyperventilation may cause cerebral ischemia and is only indicated

if there are clinical signs of acute herniation. Mannitol or 3% saline may be used acutely to produce an osmotic shift of fluid from the brain to the plasma. A ventricular catheter may be used to monitor the ICP continuously and to remove CSF. In refractory cases, a pentobarbital-induced coma reduces pressure by severely suppressing cerebral metabolism. The anti-inflammatory effects of corticosteroids may be helpful in the management of vasogenic edema associated with tumors and abscesses. Because acute interventions such as hyperventilation, osmotic therapies, and barbiturates can have negative impacts on systemic and cerebral perfusion and have only transient effects on ICP, they must be used judiciously.

Vasopressor drugs may be necessary to maintain adequate arterial blood pressure and cerebral perfusion pressure. Serum electrolytes and osmolality should be monitored because of risk of the syndrome of inappropriate antidiuretic hormone or cerebral salt wasting (see [Chapter 35](#)). Maintaining normoglycemia may have a positive impact on outcomes. Other supportive measures include controlling agitation, fever, and seizures.

All treatments for increased ICP are temporary measures intended to prevent herniation until the underlying disease process either is treated or resolves spontaneously. Timely intervention can prevent or reverse cerebral herniation. Complete neurologic recovery is possible, but rare, after signs of transtentorial or foramen magnum herniation have begun. When these signs are complete, however, with bilaterally dilated, unreactive pupils, absent eye movements, and flaccid quadriplegia, recovery is no longer possible.

The **treatment of hydrocephalus** may be medical or surgical, depending on the etiology. After subarachnoid hemorrhage or meningitis, the flow or absorption of CSF may be transiently impaired. In this circumstance, the use of medication that decreases CSF production, such as acetazolamide, may be beneficial. Surgical management consists of removing the obstructive lesion, placing a shunt, or both. A shunt consists of polyethylene tubing extending usually from a lateral ventricle to the peritoneal cavity (**ventriculoperitoneal shunt**). Shunts carry the hazards of infection or sudden occlusion with signs and symptoms of acute hydrocephalus.

TRAUMATIC BRAIN INJURY

Following head injury, children may have immediate depression of consciousness and neurologic deficits or may be completely alert without any immediate signs of neurologic injury. Most serious trauma results from motor vehicle crashes, sports, recreation-related injuries, and violence. Head trauma may result in concussion, posttraumatic intracranial hemorrhage, skull fractures, or cranial nerve or cervical spine injuries. Head injury, or other injuries, with associated retinal hemorrhages in infants and young children should raise the suspicion of abusive head trauma (see [Chapter 22](#)).

Concussion

Concussion is the process in which traumatic forces on the brain result in the rapid onset of short-lived neurologic impairment that typically resolves spontaneously. In some cases, however, symptoms and signs may evolve over minutes to hours, so patients should not be left alone following the injury. Patients may have signs or symptoms in one or more

TABLE 184.5 Protocol for Return to Sport After Concussion

Return to play protocol follows a stepwise progress with each step taking ≥ 24 hr. The athlete should continue to the next level if asymptomatic at the current level.

1. Back to regular activities such as school.
2. Light aerobic exercise such as walking or stationary cycling; no weight lifting.
3. Moderate activity such as jogging, brief running, moderate-intensity stationary biking, moderate-intensity weightlifting (less than their prior weightlifting routine).
4. Heavy, noncontact physical activity such as sprinting/running, regular weightlifting routine, noncontact sport specific drills.
5. Practice and full contact.
6. Competition.

Centers for Disease Control and Prevention. Managing return to activities; 2018. https://www.cdc.gov/headsup/providers/return_to_activities.html. Accessed August 21, 2020.

domain: somatic (headache), cognitive (slowed reaction times, poor attention), emotional (lability, irritability), sleep disturbance (drowsiness), or physical signs (loss of consciousness, amnesia). A player with any features of a concussion should be evaluated by a licensed health care provider onsite or be referred to a physician. Acute symptoms usually reflect a functional disturbance rather than structural injury, and no abnormalities are seen on standard imaging. If the child appears well after several hours and is discharged home, parents should be instructed to call their physician for any change in alertness, orientation, or neurologic functioning. An increase in somnolence, headache, or vomiting is cause for concern and emergent neuroimaging. The majority of concussions resolve within 10 days, although the recovery may be slower in children, and in a small percentage of cases the symptoms are prolonged. Physical and cognitive rest are the cornerstones of initial concussion management, with subsequent gradual return to school and physical activities ([Table 184.5](#)). Repeated concussions, especially within a short time frame (days or weeks), carry a significant risk of permanent brain injury (**second impact syndrome**).

Traumatic Intracranial Hemorrhage

Head trauma can result in epidural, subdural, parenchymal, and subarachnoid intracranial hemorrhages, as well as brain contusions ([Table 184.6](#)). Symptoms include headache, vomiting, lethargy, decreased consciousness, and seizures. The classic presentation of **epidural hemorrhages** involves a lucid interval: Following the primary brain injury, a child has a decreased level of consciousness, then returns to normal for several hours before developing rapidly progressive neurologic symptoms. When associated with neurologic decompensation, prompt evacuation of the epidural hematoma is the primary therapy. **Subdural hemorrhages** may result from direct trauma or rotational forces from vigorous shaking (abusive head trauma). In neonates and infants with open fontanels, signs and symptoms may be absent or nonspecific. **Subarachnoid hemorrhages** classically present as a sudden,

TABLE 184.6 Syndromes of Posttraumatic Intracranial Hemorrhage

SYNDROME	CLINICAL AND RADIOLOGIC CHARACTERISTICS	TREATMENT
Epidural	Onset over minutes to hours Lucid interval followed by progressive neurologic deficits Lens-shaped extracerebral hemorrhage compressing brain	Surgical evacuation or observation Prognosis good with prompt treatment, otherwise poor
Acute subdural	Onset over hours Focal neurologic deficits Crescentic extracranial hemorrhage compressing brain	Surgical evacuation Prognosis guarded
Chronic subdural	Onset over weeks to months Anemia, macrocephaly Seizures, vomiting Crescentic, low-density mass on CT	Subdural taps or subdural shunt as necessary Prognosis can be good
Intraparenchymal	Depressed consciousness Focal neurologic deficits ± Additional multiple contusions	Supportive care Prognosis guarded
Subarachnoid	Stiff neck Worst headache of life Late hydrocephalus	Supportive care Prognosis variable
Contusion	Focal neurologic deficits Brain swelling with transtentorial herniation CT findings: multifocal low-density areas with punctate hemorrhages	Medical treatment of elevated intracranial pressure Prognosis guarded

CT, Computed tomography.

severe headache due to rupture of an intracranial aneurysm. Brain **contusions** are areas of bruising with associated localized ischemia, edema, and mass effect.

Skull Fractures

Skull fractures may present with localized swelling and pain, subcutaneous bleeding over the mastoid process (**Battle sign**) or around the orbit (**raccoon eyes**), blood behind the tympanic membrane (**hemotympanum**), or CSF leak from the nose (rhinorrhea) or ear (otorrhea). Skull fractures may be linear, diastatic (spreading the suture), depressed (an edge displaced inferiorly), or compound (bone fragments breaking the skin surface). Linear, diastatic, and minimally depressed

fractures necessitate no specific treatment, but imaging is indicated to evaluate for associated intracranial hemorrhages. If the depression is more than 0.5–1 cm, surgical elevation of bone fragments and repair of associated dural tears are generally recommended. Compound fractures or penetrating injuries necessitate emergent surgical debridement and tetanus prophylaxis. The risk of associated brain contusion and early seizures is high in compound fractures.

CSF leak occurs when a skull fracture tears adjacent dura, creating communications between the subarachnoid space and the nose, paranasal sinuses, mastoid air cells, or ear. Clear fluid that leaks from the nose or ear after head trauma is presumed to be CSF until proved otherwise. The presence of air within the subdural, subarachnoid, or ventricular space also indicates a dural tear and open communication between the nose or paranasal sinuses and brain. In most cases, the dura heals spontaneously when the patient's head is kept elevated. Patients with CSF leaks are at risk for the development of meningitis or extradural abscesses. If the leak persists or recurs or if meningitis develops, the dura is surgically repaired. **Cranial nerve palsies**, secondary to a laceration or contusion of the cranial nerves, may result from a skull fracture and may be transitory or permanent.

Other Complications of Head Trauma

Cervical spine injuries must be suspected in any unconscious child, especially if bruises are present on the head, neck, or back. In conscious children, findings of neck or back pain, burning or stabbing pains radiating to the arms, paraplegia, quadriplegia, or asymmetric motor or sensory responses of arms or legs suggest spinal cord injury. Cervical spine injury (displaced or fractured vertebra) may result in complete transection of the cord with spinal shock, loss of sensation, and flaccid paralysis. A contused cord (without vertebral abnormality) may present in a similar manner. Any patient with a clinical or radiologic abnormality of the spine requires immediate spine immobilization, cardiopulmonary stabilization, and neurosurgical consultation.

Posttraumatic seizures are divided into one of three patterns: impact seizures, early posttraumatic seizures, and late posttraumatic seizures. Impact seizures occur within seconds of the injury and are presumed to reflect a direct mechanical stimulation to the cortex. The prognosis is excellent, and likelihood of later epilepsy is negligible. Early posttraumatic seizures occurring within the first week of the head injury are likely the result of a localized area of cerebral contusion or edema. The long-term prognosis for these seizures is quite favorable. Late posttraumatic seizures arise more than a week after the trauma and most likely indicate an area of cortical gliosis or scarring that will be a source for long-term epilepsy. These patients often require long-term antiepileptic drug therapy.

Transient neurologic disturbances may develop a few minutes after head trauma and last for minutes to hours before clearing. The most common symptoms are cortical blindness and confusional states, but hemiparesis, ataxia, or any other neurologic deficit may appear. These symptoms may represent a trauma-triggered migraine in susceptible children. Care must be exercised to exclude intracranial pathology.

Evaluation and Treatment

Children who have loss of consciousness or amnesia following a head injury should be evaluated in an emergency

department. High-risk patients include those with persistent depressed or decreasing level of consciousness, focal neurologic signs, penetrating skull injury, depressed skull fractures, or worsening symptoms. Because patients with intracranial hemorrhage may progressively deteriorate and require immediate neurosurgical care, these patients warrant care in a skilled trauma center. The **Glasgow Coma Scale** is a valuable tool for monitoring the course of patients after trauma for signs of deterioration (see [Chapter 42](#)). The observation period varies with the severity of the injury and may include hospitalization.

Immediate management of severe injuries includes ensuring an appropriate airway, breathing, and circulation. Neuroimaging with CT or MRI and cervical spine radiographs are obtained emergently. Patients with intracranial hemorrhage may require emergent surgical intervention for decompression and drainage of the blood collection. Increased ICP may require monitoring, placement of a ventricular drain, and aggressive medical management, including intubation and ventilation, osmotic therapy, and sedation.

Neuroimaging is not necessary for uncomplicated concussions but should be employed whenever suspicion of structural intracranial lesion exists. Drowsiness, headache, and vomiting are common after head trauma. They are not, by themselves, of concern if consciousness is preserved, the clinical trend is one of improvement, and results of the neurologic examination are normal. Children are especially susceptible to somnolence after head trauma but should be easily arousable. If symptoms worsen or persist for more than 1–2 days, neuroimaging may be indicated to evaluate for intracranial hemorrhage or cerebral edema.

Prognosis

Children with mild TBI (without subsequent neurologic deficits) have a favorable long-term prognosis, and late sequelae are rare. Children with moderate TBI usually make good recoveries even when neurologic signs persist for weeks. Long-term sequelae may include headaches, poor memory, slowing of motor skills, decline in cognitive skills, behavioral alterations, or attention deficits. Language function, especially in a young child, frequently makes a good recovery. Rehabilitation programs with physical therapy, behavioral management, and appropriate education may be necessary.

Coma lasting for weeks after head trauma may be compatible with a good outcome, although the risk of late sequelae is significant. Mortality rates for children with severe TBI range from 10–30%. Poor prognostic signs include a Glasgow Coma Scale score below 4 on admission without improvement in 24 hours, absent pupillary light reflexes, and persistent extensor plantar reflexes. Extracranial trauma also contributes to the morbidity of these patients (e.g., aspiration pneumonia, acute respiratory distress syndrome, sepsis, emboli). Children with TBI who experience acute cerebral swelling may improve gradually or may remain in a vegetative state. Maximal recovery may take weeks or months. Patients who remain in a vegetative state for months after head injury are unlikely to improve.

PEARLS FOR PRACTITIONERS

See *Neurology: Pearls for Practitioners* at the end of this section.