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Altered States of Consciousness

Jeffrey R. Avner, MD*

Author Disclosure Dr Avner did not disclose any financial relationships relevant to this article. **Objectives** After completing this article, readers should be able to:

- 1. List the common causes of altered level of consciousness.
- 2. Discuss how to differentiate medical and structural causes of altered level of consciousness.
- 3. Develop a plan for the initial phase of evaluation for an altered level of consciousness.
- 4. Recognize the importance of radiologic imaging in a child who has an altered level of consciousness.
- 5. Know which ingestions are likely to cause neurologic adverse effects.

Definition

During the course of normal interaction with one another, we observe each other's mannerisms, responses, movements, and communications. In a sense, a person's behavior is determined by how he or she acts or reacts to internal and external stimuli. What is considered normal behavior is often age-specific and person-specific. For example, the response of a teenager to the early morning "buzz" of an alarm clock is usually a purposeful attempt to shut the alarm off; an infant may cry as a response to the same stimulus. Similarly, children who have certain chronic illnesses, such as static encephalopathy, may have blunted responses as a baseline behavior. Although major changes in behavior are readily apparent to any clinician, subtle changes often are appreciated best by parents and caretakers.

Essential to the evaluation of abnormalities in a child's behavior is an understanding of levels of consciousness. Various terms that define specific conditions or alterations of normal levels of consciousness often are used interchangeably and incorrectly by clinicians and parents alike. Consciousness is a state of awareness of both one's self and the environment. A child who has a normal level of consciousness can be awakened and is aware of what is happening to and around him- or herself. Alteration of the level of consciousness usually begins with reduced awareness of one's self, followed by reduced awareness of the environment, and finally by an inability to be aroused. The opposite of consciousness is coma, a state in which a person is unresponsive to all stimuli, including pain.

Although consciousness and coma represent the extremes of mental status, there are many abnormal states of consciousness along that spectrum that may, at times, blend imperceptibly into one another. Appropriate use of terminology begins with a proper understanding of the definition of such states of consciousness. Confusion occurs when there is a loss of clear thinking, usually manifested by impairment of cognitive abilities and decision-making. Disorientation often accompanies confusion. In general, disorientation to time occurs first, followed by disorientation to place, and then by deficiency in short-term memory. Loss of recognition of one's self is a later finding. In delirium, there is a succession of confused and unconnected ideas. Delirious children often have extreme mental and motor excitement, so they become disoriented, fearful, irritable, offensive, or agitated. Lethargy is a state resembling profound slumber, in which the child's movement or speech is limited. A lethargic child can be aroused with moderate external stimulation but immediately relapses into a state of limited responsiveness. Stupor is a condition of deep sleep or unresponsiveness from which the child can be aroused only with repeated

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Table 1. Mnemonic for Altered Level of Consciousness

- A Alcohol, Abuse of Substances
- E Epilepsy, Encephalopathy, Electrolyte Abnormalities, Endocrine Disorders
- I Insulin, Intussusception
- O Overdose, Oxygen Deficiency
- U Uremia
- T Trauma, Temperature Abnormality, Tumor
- I Infection
- P Poisoning, Psychiatric Conditions
- S Shock, Stroke, Space-occupying Lesion (intracranial)

vigorous stimuli. Finally, in coma, the child is unresponsive even to painful stimuli. A persistent vegetative state and coma often are confusing. In both the vegetative state and coma, there is no evidence of self-awareness (no response to communication or purposeful movements) or communication (either verbal or by gestures). However, in the vegetative state, the child's eyes may open spontaneously, giving the appearance of a state of arousal (albeit poorly sustained and sluggish) as opposed to that of coma, in which the eyes are always closed.

Epidemiology

An altered level of consciousness in children has many causes, with a fundamental differentiation being between structural and nonstructural (referred to as "medical" in this article) conditions. The mnemonic AEIOU TIPS (tips on vowels) is helpful in listing the major categories of illness or injury to be considered (Table 1). Although these disorders can occur at any age, certain conditions are more prevalent at specific ages. Nontraumatic coma has a bimodal distribution, being most common in infants and toddlers and having another smaller peak in adolescence. Infection either of the brain (encephalitis), meninges (meningitis), or both is the most common cause of altered level of consciousness, accounting for more than one third of nontraumatic cases. (Wong, 2001) Congenital malformations, especially those of the central nervous system (CNS), typically present in the first few postnatal months, but complications from surgical correction of such problems (eg, ventriculoperitoneal shunt obstruction) may occur at any age.

Diabetic ketoacidosis, the most common metabolic disorder presenting with alteration of consciousness, can occur at any age but is more common in adolescence. Inborn errors of metabolism, including those that present with electrolyte and glucose abnormalities, typi-

Table 2. Commonly Ingested Agents that Cause an Altered Level of Consciousness

- Amphetamines
- Anticholinergics
- Anticonvulsants
- Barbiturates
- Benzodiazepines
- Clonidine
- Cocaine
- Ethanol
- Haloperidol
- Narcotics
- Phenothiazines
- Salicylates
- Selective serotonin uptake inhibitors (SSRIs)
- Tricyclic antidepressants

cally present in infancy. Prolonged seizures, anticonvulsive therapy, and the postictal state also can alter the level of consciousness.

In childhood and adolescence, toxic exposure or ingestion is most common. A toddler has the ability to explore the environment but does not yet have the cognitive ability to know that ingesting pills may be harmful. Many medications (especially those targeted for use in pediatrics) are brightly colored and taste like candy, creating an inviting stimulus for accidental ingestion. Commonly ingested agents that cause an altered level of consciousness are listed in Table 2. Ingestion by adolescents usually is intentional and typically involves over-the-counter medication (eg, acetaminophen, ibuprofen) or psychotropic drugs such as antidepressants.

Although the overall incidence of traumatic and nontraumatic coma is similar, the rate of traumatic injury tends to increase throughout childhood. Trauma, especially head trauma, can cause intracerebral, epidural, or subdural bleeding, leading to cerebral dysfunction either by primary neuronal damage or the effects of cerebral herniation or brainstem compression. Intentional trauma (child abuse) always should be considered in any infant presenting with an altered level of consciousness.

Pathogenesis

The clinician can determine the child's state of awareness by the child's behavior. The content of a child's behavior can be inferred by his or her actions and appearance. Normal behavior requires appropriate cognition and affect, enabling children to perceive the relationship between themselves and their environment. This component of behavior is controlled by the cerebral hemispheres.

In a typical day, the body goes through a normal cycling of alertness. From a state of wakefulness, it is normal to become drowsy and, eventually, to fall asleep. At some point during sleep (or even during drowsiness), external stimuli are processed through sensory inputs to increase awareness and cause one to be more awake. This cycling of behavior is modulated predominately by the ascending reticular activating system (ARAS), a core brainstem structure that often is considered the "sleep center." Thus, normal behavior can be thought of as a combination of appropriate "content" and arousal.

A useful method of approaching altered level of consciousness is the bulb-switch analogy. Consider the content of behavior (controlled by the cerebral hemispheres) to be a light bulb and the arousal component (controlled by the ARAS) to be a light switch. For the bulb to be lit (at a normal level of consciousness), the bulb has to be functional and the switch on. There are three possibilities if the bulb is not lit (altered level of consciousness): a defect in the bulb itself (diffuse dysfunction of the cerebral hemispheres), a defect in the switch (a localized abnormality of the ARAS), or defects in both the bulb and the switch (global CNS dysfunction).

This model also helps differentiate the etiology of the alteration in consciousness. The ARAS is located in the vicinity of several brainstem reflexes, including those for pupillary light reflexes (cranial nerves II and III) and those for reflex eye movements (cranial nerves III, VI, VIII, and the medial longitudinal fasciculus). Thus, preservation of these reflexes suggests that the ARAS is functional. Under this condition, the altered level of consciousness likely is due to a dysfunctional bulb (involvement of both cerebral hemispheres).

On the other hand, impingement on the area of the ARAS causes loss of the brainstem reflexes and an altered level of consciousness, even though the cerebral hemispheres are functioning normally. Diffuse cerebral dysfunction usually has a medical basis, such as toxic, metabolic, or infectious causes; compression of the ARAS usually is the result of structural causes (Table 3). About 90% of cases of nontraumatic coma in children are due to medical causes. (Wong 2001)

It is important to note, however, that although focal neurologic signs suggest a structural lesion and lack of focality suggests a medical cause, there are many exceptions. For example, structural disorders that may present without focality include acute hydrocephalus, bilateral subdural hematomas, and acute bilateral cerebrovascular disease. Medical encephalopathies that often present

Table 3. Differential Diagnosis of Altered Level of Consciousness

Structural Causes

- Cerebral vascular accident
- Cerebral vein thrombosis
- Hydrocephalus
- Intracerebral tumor
- Subdural empyema
- Trauma (intracranial hemorrhage, diffuse cerebral swelling, shaken baby syndrome)

Medical Causes (Toxic-Infectious-Metabolic)

- Anoxia
- Diabetic ketoacidosis
- Electrolyte abnormality
- Encephalopathy
- Hypoglycemia
- Hypothermia or hyperthermia
- Infection (sepsis)
- Inborn errors of metabolism
- Intussusception
- Meningitis and encephalitis
- Psychogenic
- Postictal state
- Toxins
- Uremia (hemolytic-uremic syndrome)

with apparent focal neurologic signs include hypoglycemia, hyperglycemia, hypercalcemia, hepatic encephalopathy, uremia, and the postictal state that includes Todd paralysis.

Pathophysiology

The differentiation of structural and medical causes of altered level of consciousness is assessed best by imaging modalities such as computed tomography (CT) scan or magnetic resonance imaging (MRI), which are readily available in most acute-care settings. Although imaging can pinpoint specific structural defects, the presence of cerebral swelling, and focal neurologic abnormalities, it also is important to have a clear understanding of the underlying pathophysiology that accompanies both structural and medical causes of altered level of consciousness. Three major responses (ie, pupillary reflexes, extraocular movements, and motor response to pain) are helpful in evaluating both the level and progression of a child's state of consciousness. The pupillary reflex is a balance between parasympathetic (pupillary constrictors) and sympathetic (pupillary dilators) innervation. Because the pathways that control this reflex lie adjacent to the brainstem area that controls consciousness, lesions that

impinge or affect the brainstem alter pupillary size or the ability of the pupil to react to light. For example, a midbrain lesion interrupts the parasympathetic and sympathetic fibers equally, resulting in pupils that are in midposition and fixed; a pontine lesion primarily interrupts the descending sympathetic fibers, causing pinpoint pupils. Expanding lesions in the temporal area of the brain may cause uncal herniation and compress cranial nerve III, leading to a unilateral dilated and fixed pupil on the side of the lesion.

On the other hand, the pupillary reflex is relatively resistant to metabolic insult; although the pupils may be small, they maintain the ability to react to light. Therefore, a child who has unequal, sluggishly reactive, or unreactive pupils should be presumed to have brainstem dysfunction in the area of the ARAS and likely a structural cause for the abnormal level of consciousness, as opposed to a medical cause, which would spare the pupillary reflex. For that reason, the presence or absence of the pupillary reflex is one of the most important



findings for differentiating structural and medical causes of altered consciousness.

Dysfunction of certain extraocular movements also may accompany structural causes of altered consciousness. In particular, the oculocephalic reflexes are helpful in assessing low brainstem function. For example, when the head is turned to one side in a child who has a functioning brainstem, the eyes move in conjugate fashion, regardless of the level of consciousness. Stimuli from the cervical muscles in the neck, the semicircular canals in the ear, and the cerebellum synapse with the vestibular nucleus in the brainstem. The impulse, then, is transmitted caudally through the medial longitudinal fasciculus (MLF) to the ipsilateral abducens nucleus (cranial nerve VI) that contracts the lateral rectus muscle and abducts the ipsilateral eye. The impulse also continues caudally but crosses the brainstem to synapse with the contralateral oculomotor nucleus (cranial nerve III), which contracts the medial rectus muscle and adducts the contralateral eye. Thus, the eyes move in conjugate fashion (one eye adducts and the other abducts). If there is a brainstem lesion at the level of the MLF, the eyes move dysconjugately when the head is turned. If there is a low brainstem lesion, the eyes do not move at all relative to the head; in this "doll eyes" phenomenon, the eyes appear as if they were painted on the head.

Finally, motor response to a painful stimulus can help localize the level of brainstem dysfunction. Lesions at or above the diencephalic level are associated with decorticate posturing, so the legs stiffen and the arms are rigidly flexed at the elbow and wrist. As the lesion moves rostrally to the level of the midbrain or upper pons, the arms and legs extend and pronate in response to pain, in what is called decerbrate posturing. If the lesion extends to the medulla, the child's muscles are flaccid, and there is no response to painful stimuli.

Clinical Aspects

The history and physical examination should focus on identifying both the cause and progression of the altered

level of consciousness. Information about the onset of the neurologic symptoms is particularly important. Time of day, location, and duration of initial symptoms may offer clues to the underlying cause. Clearly, a history of trauma directs the differential diagnosis toward traumatic causes. However, especially in the young child, a history of minor trauma may be overlooked. Early morning headaches, dizziness, and somno-

lence often are seen with increased intracranial pressure. Proximity to a poorly ventilated combustible gas source (home heating system, car exhaust) can cause carbon monoxide poisoning. Dizziness and lethargy following a party or school event should raise suspicion for a toxic ingestion. An abrupt change in the child's mental status often results from an acute event such as a CNS hemorrhage or obstructive hydrocephalus.

A gradual onset of symptoms over hours or days suggests a metabolic, infectious, or toxic cause. Continued clinical deterioration may signal increasing intracranial pressure, systemic infection, or progressive metabolic derangement (acidosis, electrolyte abnormality). A past history of medical conditions associated with altered level of consciousness may be seen in diabetes mellitus, hypoglycemia, hypertension, or uremia.

Evidence of drug or alcohol use or availability of prescription or nonprescription drugs should be ascertained. In both intentional and accidental ingestions, there may be no specific history of ingestion. Therefore,

Table 4. Important Questions in the History of Ingestion

- To what potential poisons was the child exposed?
- What medications (prescription and nonprescription) are in the home?
- Where did the poisoning take place?
- When (or over what potential time period) did the poisoning occur?
- How much poison was involved?
- By what route did the poisoning occur?
- Was the poisoning accidental or intentional?

the clinician must determine the availability and type of medication in the home. Because ingestions are a common cause of altered mental status in children, a list of important historical questions is shown in Table 4.

Key findings on the physical examination can help differentiate structural from medical causes of altered consciousness. Accurate assessment of the child's vital signs is of paramount importance. If the child is febrile, an infectious cause is likely, although some toxic ingestions (eg, anticholingerics) also can cause fever. The respiratory rate and pattern may be helpful in localizing the level of a brainstem lesion. Because the respiratory center is located in the pons and medulla, compression on this area of the brainstem changes the pattern of respiration. If there is primary cerebral dysfunction, there may be posthyperventilation apnea. As the level of the brain dysfunction progresses from caudal to rostral through the brainstem, the respiratory pattern progresses from Cheyne-Stokes respiration (crescendo-decrescendo pattern followed by intervals of apnea) to central neurogenic hyperventilation (sustained, rapid, deep respiration). In low brainstem lesions, respiratory effort is gasping, irregular, or sporadic.

The pulse or blood pressure often is abnormal in cases of impending cerebral herniation. The cranial vault can accommodate a modest increase in intracranial contents caused by brain swelling, cerebral mass, or hemorrhage. The anterior fontanelle and the flexible skull of the infant allow the intracranial cavity to expand. In the older child and adult, an intracranial buffering system (displacement of cerebrospinal fluid and venous blood) maintains the intracranial pressure despite increasing intracranial contents. However, once the capabilities of these protective mechanisms are exceeded, intracranial pressure rises precipitously. The resulting intracranial hypertension causes brain injury by a variety of mechanisms.

Cerebral perfusion pressure is estimated by subtracting the intracranial pressure from the mean arterial pressure. As the intracranial pressure rises, the cerebral perfusion pressure falls, thus compromising the delivery of oxygen and nutrients to the brain and causing cerebral ischemia. Systemic hypertension is the body's attempt to maintain mean arterial pressure and preserve cerebral perfusion. In particular, the Cushing triad (systemic hypertension, bradycardia, abnormal respiration) is a late sign of increased intracranial pressure.

Differences in pressure between cerebral compartments may lead to herniation of cerebral contents and compression of the brainstem. The tentorium cerebelli, which divides the anterior and posterior fossa, is the usual opening through which the midbrain passes. If the temporal fossa has an expanding lesion (such as an epidural hematoma), the medial temporal lobe (uncus) on the

Stage	Mental Status	Pupils	Extraocular Movements	Motor Response
Central Herniation				
Diencephalic (early)	Normal or decreased	Small, reactive	Normal	Appropriate
Diencephalic (late)	Decreased	Small, reactive	Normal	Decorticate
Midbrain, upper pons	Decreased	Midpoint, fixed	Asymmetric	Decerebrate
Lower pons, medulla	Decreased	Pinpoint, fixed	Absent	Flaccid
Uncal Herniation				
Third nerve (early)	Usually normal	Unilateral, dilated, sluggish	Normal or asymmetric	Appropriate or asymmetric
Third nerve (late)	Decreased	Unilateral, dilated, fixed	Asymmetric or absent	Decorticate or decerebrate
Midbrain, upper pons	Decreased	Midpoint, fixed	Asymmetric or absent	Decerebrate
*Adapted from Plum F, Posner J, 1980.				

Table 5. Progression of Herniation Syndromes*

side of the lesion can herniate through the tentorium, in what is known as uncal herniation (Table 5). Because the oculomotor nerve (cranial nerve III) passes alongside the midbrain, it usually becomes compressed along with the brainstem. Thus, uncal herniation is associated with a unilateral, dilated pupil on the side of the lesion. In the early stages of herniation, the pupil is sluggishly reactive; as the symptoms progress, however, the pupil becomes fixed and dilated. In addition, there is dysconjugate gaze due to asymmetric extraocular movements.

Motor findings progress from asymmetric to decorticate posturing and, in the late stages, to decerebrate posturing. In central herniation, parenchymal lesions of frontal, parietal, or occipital lobes cause cerebral swelling and downward displacement of both temporal lobes through the tentorium (Table 5). The herniation syndrome progresses rostral to caudal, affecting first the diencephalic brain (small reactive pupils, normal extraocular movements, appropriate or decorticate posturing), then the midbrain/lower pons (midpoint and fixed pu-



pils, dysconjugate gaze, decerebrate posturing), and finally the lower pons/medulla (pinpoint and fixed pupils, absent extraocular movements, flaccid paralysis). Therefore, it is important to note the level of consciousness, pupillary size and reactivity, extraocular movements, and motor response when examining a child whose level of consciousness is altered.

It should be emphasized that early imaging of the brain with a CT scan or MRI remains the cornerstone of rapid and accurate diagnosis. Because the MRI does not use ionizing radiation and displays greater anatomic detail, it often is the preferred imaging modality. However, CT scanning typically is used in the acute setting because of availability and logistic considerations.

In addition to signs noted on a complete neurologic examination, other findings on physical examination may suggest certain conditions. Papilledema on funduscopic examination is a late finding of increased intracranial pressure because it usually requires more than 12 hours to develop. Therefore, normal fundi do not rule out the presence of increased intracranial pressure. In an infant or young child, the presence of retinal hemorrhages should raise the concern for child abuse. Meningismus on the neck examination results from meningeal irritation, usually from meningitis or a focal neurologic lesion. A child in heart failure may present with a tachydysrhythmia, hepatomegaly, or a gallop on cardiac examination. The presence of hepatomegaly may represent an acute infectious hepatitis or a metabolic disorder causing hepatic encephalopathy. In a febrile child, petechiae, especially if present below the nipple line, are a hallmark of meningococcemia. In an afebrile child, petechiae may be a sign of thrombocytopenia that places the child at risk for cerebral hemorrhage.

Management

A child who has any acute alteration in level of consciousness should be transported immediately to an acute care facility for additional evaluation and management. However, if hypoglycemia is suspected, intramuscular gluca-

> gon should be administered. Once in the emergency department, management begins with assessment of airway, breathing, and cardiovascular status (the ABCs of resuscitation). Often, a child whose level of consciousness is altered has recognizable stridor caused by downward displacement of the mandibular block of tissue and tongue into the upper airway. This obstruction usually is relieved by a chin-lift or jaw thrust, but if

such maneuvers fail, an oral airway or endotracheal intubation may be needed. All children initially should be given 100% oxygen by nonrebreather facemask until adequate oxygenation is assured.

Because many of the causes of an altered level of consciousness require immediate intravenous (IV) fluid or medication, most children should have an IV line placed. Rapid infusion of IV normal saline solution often is needed to restore and maintain adequate perfusion of the vital organs. Blood tests to help determine the cause should be obtained and a bedside glucose concentration determined immediately. Hypoglycemia is a common direct cause as well as associated symptom of an altered level of consciousness and is immediately correctable with the administration of IV glucose. Therefore, the blood glucose concentration never should be overlooked. If the child has a history of possible ingestion or shows signs of opiate toxicity (pinpoint pupils, coma, respiratory depression), the opiate antagonist nalaxone can be given IV.

Once life-saving management is performed and the child has stabilized, results of the history and physical examination should point to either a structural lesion or a medical disorder. If a structural lesion is suspected, immediate intervention to control an increase in intracranial pressure is warranted. The head should be elevated to 30 degrees and kept in a midline position to facilitate venous drainage from the intracranial vault. An emergent CT scan should be obtained, although the child may require endotracheal intubation to be sedated properly for the procedure. Because many structural causes involve surgical management, a neurosurgeon should be consulted.

If a medical cause is suspected, additional laboratory and radiologic testing may help determine whether the cause is infectious, metabolic, or toxicologic. Serum electrolyte concentrations may be abnormal, and any acidbase alteration should be corrected. A febrile child is likely to have an infectious etiology and should be given empiric antibiotics IV after a blood culture is obtained. Because meningitis is a common infectious cause of altered consciousness, a lumbar puncture can help confirm that diagnosis. However, if the child is clinically unstable or has focal neurologic signs, the lumbar puncture should be deferred and antibiotics administered immediately. Empiric acyclovir is recommended if herpes encephalitis is a concern. Serum ammonia and liver function tests may diagnose an inborn error of metabolism, other metabolic disorder, or hepatic encephalopathy. A positive stool guaiac test raises the concern of intussusception, which can be diagnosed with ultrasonography or air-contrast enema. Cardiac dysrhythmias or conduction abnormalities often can be identified with electrocardiography. After immediate treatment and stabilization, additional testing may include electroencephalography and an MRI.

In the absence of a clear cause, especially in an afebrile toddler or adolescent, a toxic ingestion should be suspected. Family members should be questioned about the availability of any medication, whether prescription or over-the-counter. Cough medication, antipyretics, and iron pills often are ingested by toddlers. If possible, the bottle of the medication should be checked for remaining pills to estimate the maximum amount ingested. If the ingested agent is determined, the appropriate antidote can be given. Specialists at the local poison control center can assist in additional management of a suspected overdose. Many children require a dose of activated charcoal, which binds the toxin, thereby limiting intestinal absorption.

After stabilization and initial management, the child should be observed in a monitored setting until his or her mental status improves. The specific disorder and the severity of the child's clinical state determine whether management can be accomplished on the inpatient unit or in an intensive care unit.

Summary

An altered level of consciousness is an important clinical entity in pediatrics and carries the potential for significant morbidity and mortality. The clinician must assess the level of consciousness rapidly as well as determine likely causes. Structural or medical causes usually can be differentiated through a focused history and physical examination. Asymmetric neurologic findings such as a dilated and fixed pupil, dyscongugate extraocular movements, and asymmetric motor findings suggest brainstem dysfunction as a result of a structural lesion; slowly progressive but symmetric neurologic findings usually result from a medical disorder. Specific neurologic findings associated with elevated intracranial pressure are seen in both central and uncal herniation. Disorders of metabolism, liver, kidneys, lungs, or heart as well as toxic exposure are common medical causes for alteration of consciousness. After initial assessment and stabilization, management centers on determining the specific diagnosis and continuing supportive care.

Suggested Reading

- Kanich W, Brady WJ, Huff S, et al. Altered mental status: evaluation and etiology in the ED. Am J Emerg Med. 2002;20:613–617
- King D, Avner JR. Altered mental status. Clin Pediatr Emerg Med. 2003;4:171–178
- Kirkham FJ. Non-traumatic coma in children. Arch Dis Child. 2001;85:303-312
- Meyer PG, Ducrocq S, Carli P. Pediatric neurologic emergencies. Curr Opin Crit Care. 2001;7:81–87
- Pattisapu JV. Etiology and clinical course of hydrocephalus. Neurosurg Clin North Am. 2001;36:651–659
- Plum F, Posner J. The Diagnosis of Stupor and Coma. Philadelphia, Pa: FA Davis; 1980
- Wong CP, Forsyth RJ, Kelly TP, Eyre JA. Incidence, aetiology, and outcome of non-traumtaic coma: a population-based study. *Arch Dis Child.* 2001;84:193–199

PIR Quiz

Quiz also available online at www.pedsinreview.org.

- 5. A 2-year-old boy is found unresponsive in his bedroom by his mother. He had eaten his breakfast 2 hours earlier, and he seemed normal at the time. On physical examination, the child is obtunded and responds to only deep, persistent stimuli by moaning and brief opening of his eyes. He is afebrile and has a heart rate of 60 beats/min, respiratory rate of 16 breaths/min, and blood pressure of 88/56 mm Hg. Pupils are 2 mm, and they react briskly to light. Extraocular movements and the oculocephalic reflex are intact. No dystonic movement is noted. Dysfunction of which of the following best explains these clinical findings?
 - A. Ascending reticular activating system.
 - B. Cerebral hemispheres.
 - C. Limbic system.
 - D. Medulla oblongata.
 - E. Pons.
- 6. A 12-year-old boy is brought to the emergency department after being struck by a baseball while batting during his Little League game. He lost consciousness immediately after being hit and has remained unresponsive to his environment. On arrival at the hospital, his heart rate is 60 beats/min, respiratory rate is 16 breaths/min, and blood pressure is 180/120 mm Hg. A large hematoma is noted on his right temporal region. His right pupil is 8 mm and reacts very sluggishly to light. The left pupil is 3 mm and reacts briskly to light. Dysconjugate gaze is noted because of limitation of the movement of the right eye. Both the upper and lower extremities are extended and pronated with increased tone. Which of the following is the most likely explanation for these findings?
 - A. Cerebral infarction with right middle cerebral artery pattern.
 - B. Cervical spine injury.
 - C. Diffuse axonal injury.
 - D. Subarachnoid hemorrhage.
 - E. Uncal herniation.
- 7. A 6-year-old girl has been complaining of fever and headaches for the last 2 weeks. She has been treated with amoxicillin for sinusitis for the last week without improvement. She is now brought to the emergency department for decreasing responsiveness. Examination reveals an axillary temperature of 102.9°F (39.4°C), respiratory rate of 24 breaths/min, heart rate of 90 beats/min, and blood pressure of 138/95 mm Hg. She is somnolent and can be aroused only with persistent external stimulation. She opens her eyes to painful stimuli and appears disoriented and confused, using inappropriate words. She does not obey verbal commands but localizes the site of a painful stimulus. Her pupils are 3 mm and react briskly to light. Lateral deviation of the left eye and drooping of the right angle of the mouth are noted. Her neck is stiff, with positive Kernig and Brudzinski signs. After ensuring adequacy of airway, drawing blood for culture, and administering antibiotics, the next *most* appropriate step in management is:
 - A. Antihypertensive medication.
 - B. Computed tomography scan of the head with contrast.
 - C. Lumbar puncture.
 - D. Neurology consultation.
 - E. Toxicology screen.
- 8. A 3-year-old boy is brought to the emergency department after falling from monkey bars 6 hours ago. He has been vomiting for the last hour and now has become increasingly unresponsive. A diffuse swelling with bluish discoloration is noted over his left temporal-parietal region. Which of the following pupillary abnormalities is *most* consistent with expanding left temporal-parietal epidural hematoma?
 - A. Bilateral constricted, unreactive pupils.
 - B. Unilateral constricted, unreactive left pupil.
 - C. Unilateral constricted, unreactive right pupil.
 - D. Unilateral dilated, unreactive left pupil.
 - E. Unilateral dilated, unreactive right pupil.