

# 12 COMA, COGNITIVE IMPAIRMENT, AND SEIZURES

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Coma is the neurologist's favorite consultation: the history is usually straightforward, the neurologic examination is focused, and the differential diagnosis is limited. A good neurologic examination, in combination with a thoughtful battery of tests, will invariably achieve the correct diagnosis. It is vital for the intensivist to become equally comfortable with the rapid assessment of coma. Once initial stabilization of the patient has been achieved, management of coma is determined by the specific causative condition or conditions present. Rapid recognition of potentially reversible causes of coma (e.g., basilar artery occlusion [BAO], nonconvulsive status epilepticus, and herniation), followed by the appropriate emergency consultations, can often prevent or reduce morbidity and mortality.

This chapter is intended as a practical, rational, and efficient approach to the diagnosis and management of coma. To that end, an algorithmic approach to diagnosis is formulated, general treatment measures for comatose patients are outlined, specific causes of coma are reviewed, and prognostic issues are considered. Detailed discussions of neuroanatomy and cranial nerve pathways, which are readily available elsewhere,<sup>1</sup> are intentionally omitted, as are detailed descriptions of specific treatment of individual causes of coma (other than BAO, status epilepticus, cardiac arrest, increased intracranial pressure [IICP], and certain metabolic derangements).

## Classification of Levels of Consciousness

### AWAKENESS, ALERTNESS, AND AWARENESS

A patient's level of consciousness can usefully be described in terms of the three As of consciousness: awake, alert, and aware. To be awake means to be fully roused and thus not asleep. To be alert means to be able to pay attention to one's environment or to an examiner. Finally, to be aware means to have an understanding of oneself and one's environment. Being oriented is a manifestation of being aware of one's environment [see Figure 1].

A person who is awake, alert, and aware may be said to be fully conscious. One who is awake and alert but has lost awareness is severely demented. Someone who is merely awake and not alert or aware is delirious. A delirious patient's level of wakefulness may wax and wane. A person who is not awake, alert, or aware either is asleep (in which case he or she can be rendered awake—i.e., is arousable) or is somewhere along the so-called spectrum of coma (see below).

Admittedly, describing patients simply in terms of awakeness, alertness, and awareness omits many important nuances; nevertheless, it is an excellent and easily reproducible way of assessing level of consciousness.

### UNRESPONSIVE PATIENT

The term unresponsive is used frequently, but often in a vague manner that does not yield a clear meaning. Fortunately, in the

setting of coma management, there are only three possible meanings that need be considered [see Figure 2].

First, the term unresponsive may be applied to a patient who is actually fully conscious but is unable or unwilling to respond verbally. For example, a patient with aphasia (a common manifestation of stroke) may be awake, alert, and aware, yet unable to speak. As another example, a patient who is in a locked-in state (either from a brain stem injury or from generalized muscle paralysis) is fully conscious but cannot speak and cannot communicate except through subtle eye movements. Finally, patients with a psychiatric disorder may present with a so-called functional coma while remaining fully conscious; simple examination techniques quickly reveal that they are completely awake.

Second, the term unresponsive may be applied to a patient who has a waxing-and-waning level of wakefulness. This fluctuation between wakefulness and coma is what defines delirium, and it is invariably caused by infection, metabolic disturbances, or alcohol withdrawal.

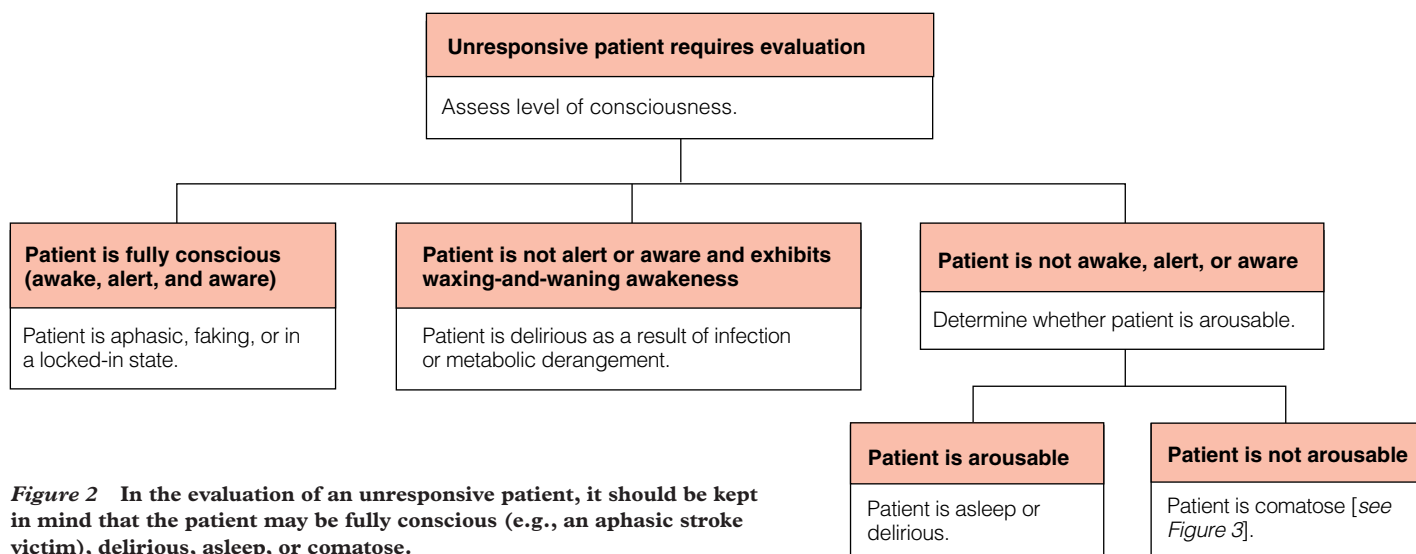
Third, the term unresponsive may be applied to a patient who truly is not even awake. In this sense, the term could of course be applied to a person who is simply asleep, but for the purposes of this chapter, it should be understood as referring to a patient who is comatose.

### SPECTRUM OF COMA

Patients who are not aware, alert, or even awake (except for those who are simply asleep) fall somewhere along the spectrum of coma. This spectrum reflects varying levels of impairment of

			Aware
		Alert	
	Awake		
Sleep, Coma	Delirium	Dementia, Infant Consciousness	Normal Adult Consciousness

**Figure 1** Awareness depends on alertness, which in turn depends on arousal (i.e., how awake one is). Normally cognizant adults are aware of themselves and their environment. Persons who have lost awareness but retain alertness and wakefulness may be characterized as severely demented. Persons who have lost alertness are delirious by definition, though their level of wakefulness may wax and wane. Finally, persons who are not aware, alert, or awake may be either asleep or in a coma.



**Figure 2** In the evaluation of an unresponsive patient, it should be kept in mind that the patient may be fully conscious (e.g., an aphasic stroke victim), delirious, asleep, or comatose.

the response to stimuli. On the high end of the spectrum, for example, is the patient who can be aroused (albeit perhaps only temporarily) by the sound of a voice. Somewhat farther down is the patient who can be temporarily aroused by a painful stimulus. Farther down still is the patient who cannot be aroused by a painful stimulus but at least exhibits some motor response to it. On the low end of the spectrum is the patient who has no motor response to a painful stimulus; this is the generally accepted definition of coma.

The terms lethargy, stupor, and obtundation, though widely used, have come to signify different things to different people. Accordingly, to ensure precise communication, it is perhaps advisable to employ clear clinical patient descriptions rather than rely on these particular terms.

### Initial Stabilization

The first step in dealing with coma is to stabilize the patient by addressing the ABCs of resuscitation (*A*irway, *B*reathing, and *C*irculation). The patient's airway must be cleared of all foreign material, and the patency of the airway must be verified. The spontaneous rate and rhythm of respiration should be noted. Endotracheal intubation is indicated in patients who are dyspneic, hypoventilating, or vomiting uncontrollably. If a patient is to be intubated, however, it is extremely helpful first to obtain a focused neurologic examination (which can be done in 60 seconds) because the information that can be gained from this examination will be lost when the patient is paralyzed for the intubation. Hyperventilation with a bag and mask and 100% oxygen should be performed before intubation to ensure adequate oxygenation during the procedure. If there is any possibility of cervical spine injury, intubation should be delayed, if possible, until fracture can be ruled out radiographically.

Circulation must be vigorously supported, especially in the setting of brain injury or hypoxia. This is accomplished by inserting a large-bore I.V. or central venous catheter and infusing isotonic fluids or volume expanders. The use of solutions containing free water (e.g., 5% dextrose in water) should be avoided, especially in the setting of brain injury or stroke, because such solutions have the potential to increase cerebral edema.

### Clinical Evaluation

As the patient is being stabilized, evaluation should be initiated. A witness or someone else capable of providing a history should be sought. The differential diagnosis of coma is wide but limited [see Table 1]. Clues from the history, the focused neurologic examination, and the general physical examination are often helpful and sometimes diagnostic [see Figure 3].

#### History

The ideal historian is a person who knows the patient well. Optimally, if the coma was of sudden onset, a witness was present who can describe what occurred. A history of trauma, drug use, medications, recent febrile illness, heart disease, organ failure, or seizures often rapidly leads to the correct diagnosis [see Table 2]. Typically, the most challenging cases are those patients who are "found down"; such cases necessitate a clear diagnostic approach that begins with the physical and neurologic examinations.

#### Focused Neurologic Examination

There are two good reasons for performing a neurologic examination on a coma patient. First, such an examination allows the physician to assign the patient a Glasgow Coma Scale (GCS) score [see Table 3], which may be used in making decisions about the necessity for intubation and which provides an objective means of following (at least superficially) the patient's neurologic status. Second, the neurologic examination may quickly yield important diagnostic information.

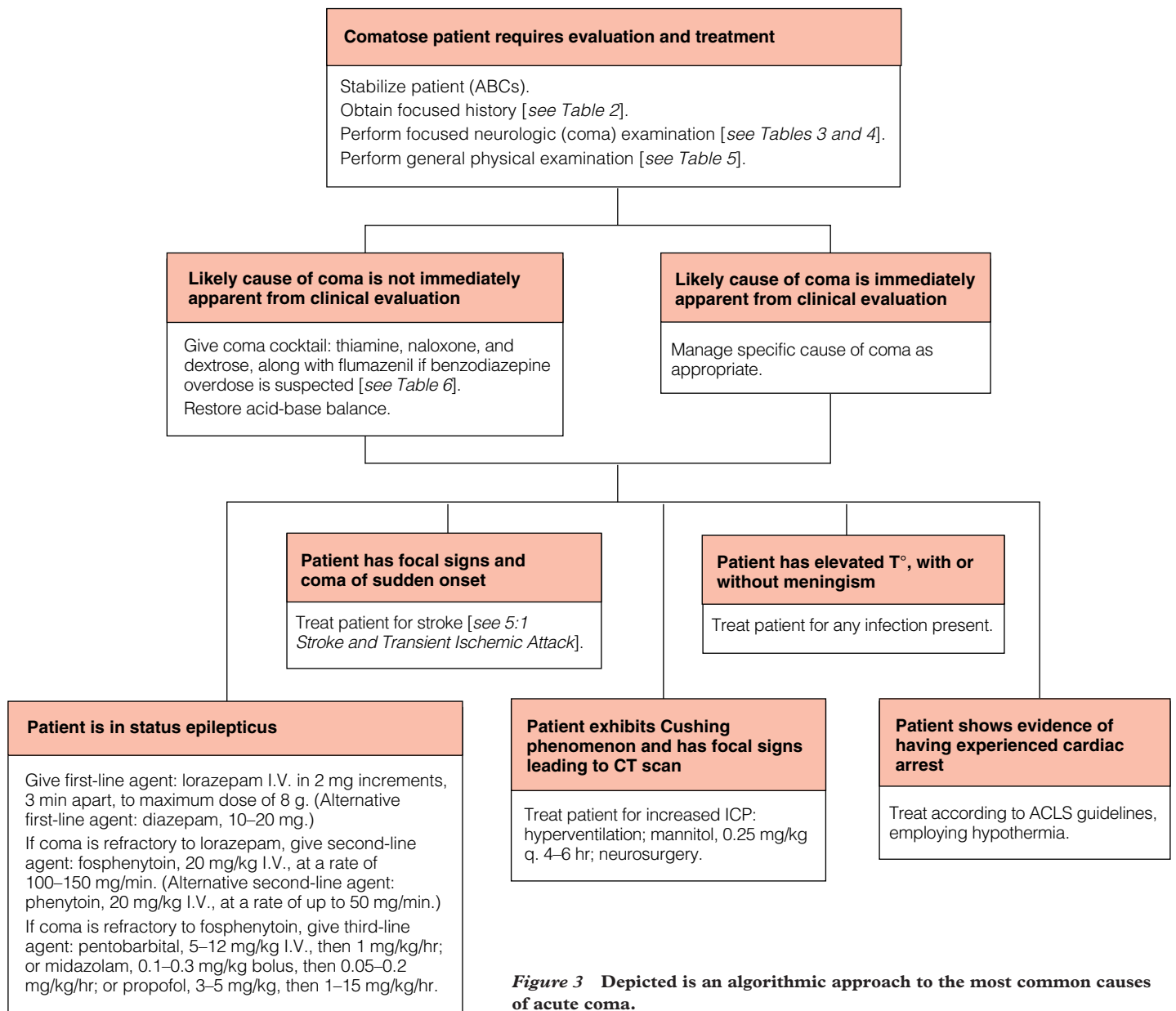
A complete and exhaustive neurologic examination is totally unnecessary. For the purposes of evaluating coma, a focused neurologic examination is preferable, being both valuable and rapid (~ 60 seconds). If intubation is indicated, the coma examination should be performed quickly before sedative and paralytic agents are given. This examination addresses a number of key findings [see Table 4], but in general, it may be thought of as assessing four neurologic variables: (1) spontaneous movements (~ 15 seconds), (2) papillary response (~ 15 seconds), (3) ocular motility (~ 15 seconds), and (4) motor response (~ 15 seconds). A reflex hammer is not needed.

Spontaneous movements should be observed over a period of 10 to 20 seconds. Generalized seizures may present as tonic or tonic-clonic movements of one or both sides. Tonic seizures are character-

ized by sustained contractions with upper-extremity flexion and lower-extremity extension. Tonic-clonic seizures are characterized by tonic contractures alternating with periods of muscle atonia, resulting in rhythmic contractions. Myoclonus consists of motor jerks that are sudden, brief, shocklike, and randomly distributed; it may be seen in patients with hypoxic-ischemic encephalopathy (e.g., after cardiac arrest) or other metabolic disturbances. Other findings (e.g., a flaccid arm that hangs down the side of the stretcher or a leg that is extorted) may be indicative of hemiparesis. The motor response to pain can be tested and assigned a GCS score. Asymmetry should be noted.

Pupillary responses are important in that their presence or absence distinguishes structural from metabolic coma (because the pupils are generally resistant to metabolic insult); they also indicate the integrity of the brain stem [see Table 5]. The origin of the reticular activating system (RAS), the so-called on switch of consciousness, may be physically located adjacent to the brain stem nuclei that control pupillary response.

The pathways that control ocular motility also lie adjacent to the RAS. Roving eye movements usually indicate that the brain stem is intact and that a metabolic problem is affecting the brain. Minimal or absent eye movement in conjunction with reactive pupils also typically signifies a metabolic process. Gaze deviation may indicate a stroke: a cortical stroke will cause the eyes to look toward the damaged side of the brain, whereas a pontine stroke will cause the eyes to look away from the damaged side of the pons. Gaze deviation may also signify an ongoing seizure: the eyes look away from the hemisphere in which the seizure is occurring or, after the seizure is over, toward the postictal hemisphere. Vertical disconjugation of the eyes (skew deviation) is indicative of brain stem disease and frequently occurs during basilar artery thrombosis. Ocular bobbing (rapid downward movements with a slow drift back to the original position) is occasionally seen in the setting of extensive pontine damage.



**Figure 3** Depicted is an algorithmic approach to the most common causes of acute coma.

*General Physical Examination*

Blood pressure, heart rate, and cardiac rhythm are key to the diagnosis of the various cardiovascular and hemodynamic causes of coma [see Table 1]. Hypothermia is noted in cases of exposure,

**Table 1 Differential Diagnosis of Coma**

Potential Cause of Coma	Differential Diagnosis
Cerebrovascular	Large brain stem stroke (with or without basilar artery occlusion) Massive stroke or hemorrhage with mass effect Subarachnoid hemorrhage Subdural or epidural hematoma Hypoxic-ischemic encephalopathy after cardiac arrest Hypertensive encephalopathy
Cardiovascular	Cardiac arrest Cardiac arrhythmia Congestive heart failure Hypotension Myocardial infarction
Infectious	Bacteremia or sepsis Meningitis Encephalitis (e.g., herpesvirus, arbovirus)
Traumatic	Multiple severe concussions (diffuse axonal injury, second-hit syndrome) Subdural or epidural hematoma Multiple contusions Penetrating head injury
Toxic	Alcohol intoxication/withdrawal Opiate intoxication Anesthesia Overdose Antianxiety agents or antidepressants Anticholinergics Anticonvulsants Antihistamines Insulin Lithium Neuroleptics Opiates Salicylates Sedatives
Metabolic	Hyperglycemia or hypoglycemia Diabetic ketoacidosis Hyperosmolar nonketotic coma Hyponatremia or hyponatremia Osmotic demyelination syndrome: central pontine myelinolysis Hepatic failure Renal failure Hypoxia or hypercarbia Hypothermia
Epileptic	Grand mal status epilepticus Nonconvulsive status epilepticus Postictal state
Mass	Massive stroke or hemorrhage with mass effect Tumor or abscess with mass effect
Hematologic	Thrombotic thrombocytopenic purpura
Psychiatric	Conversion Malingering

**Table 2 Coma History**

Questions to Be Asked	Possible Causes to Be Considered
Is there a witness?	[If no, see Table 4.]
Was it sudden?	Stroke If stepwise deterioration, consider BAO Seizure Cardiac arrest
Was there trauma?	Traumatic brain injury Subarachnoid hemorrhage Subdural or epidural hematoma with mass effect and herniation
Has there been drug use? If so, which drugs?	Recreational drug overdose Opioids, alcohol Prescription drug overdose Opioids, benzodiazepines, barbiturates, anticholinergics
Was there a recent febrile illness?	Meningitis or encephalitis Bacteremia or sepsis
Is there a history of heart disease?	Cardiac arrest Myocardial infarction Congestive heart failure Cardiac arrhythmia Hypotension
Is there a history of organ failure?	Renal failure Hepatic failure
Is there a history of seizures?	Postictal from single seizure Convulsive status epilepticus Nonconvulsive status epilepticus

BAO—basilar artery occlusion

drowning, methanol poisoning, and septic shock. Hyperthermia is obviously suggestive of an ongoing infectious process. A stiff neck is often caused by meningeal irritability resulting from infection or inflammation; it may be indicative of meningitis, but it also may be the only physical sign of subarachnoid hemorrhage. Various other physical findings may be observed in comatose patients as well [see Table 5].

A rise in intracranial pressure (ICP) occurs with any space-occupying lesion in the calvaria. Signs of rising ICP include increasing blood pressure, decreasing heart rate, and slowing or periodic respiration (Cushing phenomenon). Papilledema caused by increased ICP usually takes weeks to develop and is seldom a presenting sign.

**Management**

GENERAL MEASURES, TRIAGE, AND TEST BATTERY

As emergency management is being provided, laboratory studies should be obtained, including serum electrolyte, calcium, magnesium, phosphorus, blood urea nitrogen (BUN), and creatinine levels, as well as liver function tests. A complete blood count (CBC), a urinalysis, and a urine toxicity screen should also be obtained. A lumbar puncture should be performed only if meningitis is suspected on the basis of clinical examination.

The so-called coma cocktail—consisting of dextrose, naloxone, and thiamine—is administered if the cause of coma is not immediately apparent from the brief history and physical examination; the addition of flumazenil is considered if benzodiazepine over-

**Table 3 Glasgow Coma Scale**

Test	Response	Score
Eye opening (E)	Spontaneous	4
	To verbal command	3
	To pain	2
	None	1
Best motor response (arm) (M)	Obedience to verbal command	6
	Localization of painful stimulus	5
	Flexion withdrawal response to pain	4
	Abnormal flexion response to pain (decorticate rigidity)	3
	Extension response to pain (decerebrate rigidity)	2
	None	1
Best verbal response (V)	Oriented conversation	5
	Disoriented conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Total (E + M + V)		3–15

dose is suspected [see Table 6].<sup>2,3</sup> It should be kept in mind that administering dextrose to a thiamine-depleted patient (especially one who is malnourished or alcoholic) may precipitate the Wernicke-Korsakoff syndrome. As a rule, therefore, thiamine should always be given before dextrose.

Any comatose patient with focal signs on the coma examination should undergo head CT scanning. Any patient with signs of meningitis on the physical examination should receive emergency antibiotic therapy. When drug overdose or toxin ingestion is suspected, activated charcoal, 50 to 100 mg (with or without gastric lavage), should be given to prevent systemic absorption. As noted, flumazenil may be given for suspected benzodiazepine overdose and may produce dramatic arousal; however, this agent is contraindicated in patients taking tricyclic antidepressants because of an increased risk of seizures.

Once the recommended general treatment measures have been carried out, the next step is to look for and address specific causes of coma.

MANAGEMENT OF SPECIFIC CAUSES OF COMA

*Basilar Artery Occlusion*

BAO is an uncommon cause of stroke but also an underrecognized one. Morbidity and mortality are high, with only about 15% to 45% of victims surviving.<sup>4</sup> BAO may be caused by embolism (most often from the heart but sometimes from the vertebral arteries), by intrinsic atherothrombosis, or by vertebral dissection. It frequently presents with a stepwise accumulation of neurologic deficits that culminates in a coma.

A typical case scenario for BAO, occasionally heard by stroke neurologists, is as follows. A patient presents with double vision, followed hours later by ataxia. This may prompt an emergency department (ED) visit. The patient is admitted to the hospital but, on the nursing unit, becomes suddenly unresponsive and needs intubation for airway protection. In another typical scenario, a patient who has just undergone cardiac catheterization becomes suddenly unresponsive on the angiography table but is hemodynamically stable.

**Table 4 Focused Neurologic Examination (Coma Examination)**

Neurologic Variable Assessed	Findings	Likely Cause of Coma
Spontaneous movement (15 sec)	Rhythmic movements	Seizure
	Random jerks (myoclonus)	Hypoxic-ischemic encephalopathy
Pupillary response (15 sec)	Unilateral fixed and dilated pupil	Brain herniation (mass) Posterior communicating artery aneurysm
	Midposition unreactive pupils	Midbrain lesion
	Pinpoint unreactive pupils	Pontine lesion
	Small and sluggishly reactive pupils	Drug intoxication
	Large and unreactive pupils	Atropine
	Unreactive pupils	Barbiturates, paralytics, lidocaine, phenothiazines, methanol, aminoglycoside antibiotics, hypothermia
Ocular motility (15 sec)	Roving eye movements	Metabolic insult to brain; brain stem is intact
	Gaze deviation	Stroke (eyes look toward damaged side of brain) Seizure (eyes look away from side of brain producing seizure)
	Skew deviation	BAO
	Ocular bobbing	Extensive damage to pons
	Unilateral weakness	Structural injury (e.g., stroke)
Motor response (15 sec)	Posturing (flexor or extensor)	Herniation (mass) Extensive cortical injury

**Table 5** General Physical Examination

Physical Variable Assessed	Finding and Possible Causative Condition
Skin	Signs of trauma around head/neck Jaundice Hepatic failure Exanthema Viral infection Petechial rash Meningococcal infection
Head and neck	Brudzinski or Kernig sign Meningitis, meningoencephalitis Subarachnoid hemorrhage
Temperature	Fever Infection (meningitis, encephalitis, bacteremia, sepsis) Hypothermia Exposure Intoxication (alcohol, benzodiazepine, barbiturate) Myxedema coma Sepsis
Breath	Alcohol intoxication Fetor hepaticus Hepatic failure Uriferous smell Uremia Ketoacidosis
Blood pressure	Hypotension Shock Sepsis Intoxication Myocardial infarction Addison disease Hypertension Hypertensive encephalopathy
Cardiac conduction and rhythm (ECG)	Arrhythmia Acute myocardial infarction
Abdomen	Hepatomegaly Splenomegaly

A comatose patient with BAO usually manifests obvious clinical signs of brain stem injury, such as quadriplegia, skew deviation, and diminished gag reflex (many physicians report that intubation is unexpectedly easy). On occasion, CT scanning may reveal an apparently hyperdense basilar artery [see Figure 4]; imaging of the posterior circulation invariably reveals the problem. Management of BAO should be aimed at immediate recanalization of the occluded artery. Thrombolysis, either intravenous (with tissue plasminogen activator) or intra-arterial (with urokinase or mechanical clot disruption), may be lifesaving.

As noted, patients with extensive brain stem injury resulting from BAO often die. Some enter what is known as the locked-in state, in which they remain conscious but are unable to move, breathe, or swallow. Occasionally, patients retain the capacity for subtle eye movements or eye blinking and thus are able to communicate through yes/no responses to questions. Most stroke neurologists consider the locked-in state a fate worse than death.

### Seizures

A seizure disorder can cause coma in two different ways. First, the coma may be the initial manifestation of the immediate postictal state after a generalized seizure. In such cases, the coma resolves rapidly, and further management is unnecessary. The duration of the postictal coma may be directly correlated with patient age and inversely correlated with baseline functional status. Second, coma may develop when multiple generalized seizures occur in succession and there is not enough time between seizures to allow patients to recover. The resulting state is status epilepticus.

Generalized convulsive status epilepticus (GCSE) may be defined either as (1) continuous seizure activity lasting longer than 5 minutes or as (2) two or more discrete generalized seizures between which there is incomplete recovery of consciousness. GCSE is most frequently the result of noncompliance with an antiepileptic drug regimen, alcohol use or withdrawal, or drug toxicity. Less common causes include CNS infection, a cerebral tumor, trauma, refractory epilepsy, stroke, metabolic disorders, and cardiac arrest. If untreated, GCSE leads to cerebral edema, herniation, and death.

A tonic-clonic seizure begins with a tonic contraction that lasts as long as 30 seconds, followed by several minutes of repeated muscle contractions, loss of pupillary response to light, sweating, tachycardia, excessive bronchial secretion, and marked hypertension. Repeated seizures cause lactic and respiratory acidosis, rhabdomyolysis and myoglobinuria, aspiration and pulmonary edema, shoulder dislocations, rib fractures, and cardiac arrhythmias. The neuronal damage induces an inflammatory response, and if seizures continue, cerebral edema ensues.

In the treatment of GCSE, the aim is not to stop the body from convulsing but to stop the abnormal cerebral electrical activity immediately. The longer GCSE continues, the more refractory it will be to treatment. Paralytics mask (but do not prevent) the seizures; therefore, intubation and paralysis should be avoided if possible.

Like all other coma victims, patients with GCSE should first be stabilized [see Initial Stabilization, above]. I.V. access should then be obtained. Glucose should be given empirically, along with thiamine, and blood should be drawn and sent for laboratory tests (including anticonvulsant levels).

Anticonvulsant management of status epilepticus is discussed in detail elsewhere<sup>5,6</sup> and so need only be summarized here. The first-line treatment for GCSE consists of administration of lorazepam [see Figure 3]. Although there is no significant difference between the benzodiazepines with respect to rapidity of seizure control, lorazepam is thought to bind more tightly to brain receptors and thus is believed to have a longer duration of action. Lorazepam is given in 2 mg increments 3 minutes apart to a maximum dose of 8 mg before another agent is tried [see Table 7]. Diazepam may be employed as an alternative. The second-line agent is fosphenytoin, 20 mg/kg at up to 150 mg/min I.V.; phenytoin may be given as an alternative. If the patient does not respond to either lorazepam or fosphenytoin, a third-line agent—pentobarbital, midazolam, or propofol—should be given in a continuous infusion. Of the three third-line choices, midazolam may be the most effective, and it certainly has the lowest side effect profile.

The goal of therapy is to achieve a burst-suppression pattern on electroencephalography for 12 to 24 hours before any attempt is made to taper medications.

### Increased Intracranial Pressure

If IICP is suspected on the basis of clinical examination and herniation is identified, treatment must be initiated immediate-

**Table 6** Coma Cocktail

Drug	Indications	Dosing (Adults)	Potential Adverse Effects
Thiamine	Wernicke-Korsakoff syndrome Ethylene glycol ingestion	100 mg I.V. over 5 min	Anaphylactoid reaction Hypotension Angioedema
Dextrose	Symptomatic hypoglycemia Altered mental status without ability to rapidly obtain serum glucose level	50–100 ml of 50% dextrose I.V. Alternatively, 250 ml of 10% dextrose (to prevent the phlebitis that frequently occurs with administration of 50% dextrose)	Phlebitis Cellulitis
Naloxone	Reversal of CNS depression and respiratory depression caused by overdose of opioid medication	0.1 mg I.V. initially (I.V. route is more effective than subcutaneous or endotracheal), aimed at producing subtle improvement in ventilation Repeat doses given in 2–3 min intervals, increased very slowly each time If no response after total dose of 10 mg, opioid toxicity ruled out	Hypersensitivity reaction Precipitation of sudden narcotic withdrawal syndrome (If heroin has been tainted with scopolamine, withdrawal of opioid can precipitate anticholinergic crisis) Lung injury, hypertension, and cardiac arrhythmias (rarely reported)
Flumazenil	Benzodiazepine overdose	0.2 mg I.V. over 30 sec If no response, 0.3 mg I.V. over 30 sec If still no response, 0.5 mg I.V. every 30 sec to maximum dose of 3 mg	Contraindicated in patient experiencing seizure Benzodiazepine withdrawal, which may cause seizures and autonomic dysfunction

ly.<sup>7</sup> Medical management of IICP is a temporizing measure, aimed at slowing the process of herniation until the problem can be corrected neurosurgically.

The head of the patient’s bed should be elevated to an angle of 30° to 45° to facilitate venous return from the head. If the patient is intubated, hyperventilation to bring the patient’s carbon dioxide tension below 25 mm Hg will rapidly, but only transiently, lower

the ICP. Mannitol, 0.25 mg/kg every 4 to 6 hours, will also control IICP temporarily. Steroids may be used in the setting of tumors or abscesses of the brain. In most cases of IICP, the eventual treatment is neurosurgical decompression.

*Cardiac Arrest*

Cardiac arrest, if ongoing, is treated according to advanced cardiac life support (ACLS) guidelines. Several preliminary studies on the use of hypothermia for brain resuscitation after ventricular fibrillation (VF) cardiac arrest led to the publication of two randomized, controlled clinical trials in back-to-back issues of the *New England Journal of Medicine* in 2002.<sup>8,9</sup> Both trials clearly demonstrated that hypothermia significantly improved survival and neurologic outcomes. As a result, the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation issued a guideline statement containing the following recommendation: “Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C for 12 to 24 hours when the initial rhythm was VF. Such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.”<sup>10</sup> This recommendation has spurred the implementation of hypothermia for resuscitation of cardiac arrest victims in many hospital centers.

*Metabolic Derangements*

**Intoxication** Many comatose patients who are seen in the ED are suffering from an overdose of alcohol, narcotics, sedatives, or some combination thereof. Most of these drugs induce depression of respiration and cardiovascular function, which is a major cause of mortality in comatose patients. Anticipation and early treatment of these complications may alleviate their effects. Early intubation, respiratory support, and maintenance of normal blood pressure are essential. Gastric lavage is effective when ingestion of a drug is discovered no more than 4 hours after the event (except in the case of salicylates, which may be removed in substantial amounts as long as 10 hours after ingestion). Accordingly, in most cases of drug overdose, little is gained by performing gastric lavage more than 4 hours after ingestion, unless the patient has been in shock and consequently manifests delayed gastric emptying and slowed absorption. When



**Figure 4** In this noncontrast head CT scan, the basilar artery, which runs ventral to the pons and the lower midbrain, appears hyperdense. In the setting of acute coma, this finding suggests BAO.

**Table 7** Drugs Used to Treat Status Epilepticus

Drug	Priority of Use	Dosing	Potential Adverse Effect
Lorazepam	First-line agent	2 mg increments I.V. 3 min apart to maximum of 8 mg	Respiratory depression Hypotension
Diazepam	Alternative first-line agent	10 mg I.V., 1 or 2 doses	Respiratory depression Hypotension
Fosphenytoin	Second-line agent	20 mg/kg, 150 mg/min	Arrhythmia Hypotension
Phenytoin	Alternative second-line agent	20 mg/kg, 50 mg/min	Arrhythmia Hypotension (Risk is lower than with fosphenytoin)
Pentobarbital	Third-line agent	5–12 mg/kg, 1–10 mg/kg/hr	Poor WBC chemotaxis Paralysis of respiratory cilia Poikilothermia
Midazolam	Third-line agent	0.1–0.3 mg/kg, 0.05–2.0 mg/kg/hr	Tachyphylaxis Death (mortality lower than with propofol)
Propofol	Third-line agent	3–5 mg/kg, 1–15 mg/kg/hr	Recurrent seizures after abrupt discontinuance Hypotension Hypertriglyceridemia Anemia Death (mortality higher than with midazolam)

WBC—white blood cell

a patient is comatose or uncooperative, however, the examiner is rarely able to determine the exact time of ingestion; thus, gastric lavage is almost always indicated in this situation.

Patients are intubated with a cuffed endotracheal tube (to protect the airway from aspirated gastric contents) and placed in the left lateral decubitus position (to allow pooling of the gastric contents) with the head down. Lavage is then performed with copious amounts of fluid until the returned fluid is clear. After evacuation, a slurry containing 10 g of activated charcoal in 30 to 50 ml is instilled into the stomach via a nasogastric tube. After lavage, cathartic agents may be used to shorten the transit time through the GI tract and thus to decrease absorption of the ingested material. Diuresis (saline, ionized, or osmotic), dialysis, and charcoal hemoperfusion all facilitate excretion of toxins. Specific antidotes exist for several common intoxicants; however, discussion of these antidotes is beyond the scope of this chapter.

**Hypoglycemic coma** Symptomatic hypoglycemia may occur when plasma glucose levels fall below 45 mg/dl. Symptoms may range from jitteriness with palpitations and diaphoresis to focal neurologic symptoms mimicking ischemic stroke to frank coma. The most common predisposing cause is diabetes; other common causes include alcoholism, hepatic failure, and renal failure.

If hypoglycemic coma is prolonged, it may result in permanent brain damage. Therefore, initial management involves urgent restoration of the patient to a euglycemic state. This may be accomplished through I.V. injection of glucose. Traditionally, injection of an ampule (50 ml) of a 50% solution is prescribed; however, this measure frequently results in loss of the peripheral I.V. site used. Alternatively, 250 ml of a 10% dextrose solution is rapidly injected; this measure provides the same amount of dextrose without causing any loss of venous access. If necessary, infusion of a 5% dextrose solution may be continued subsequently. As noted [see General Measures, Triage, and Test Battery, *above*], if the patient is thiamine depleted, 100 mg of thiamine should be given before dextrose so as not to precipitate Wernicke-Korsakoff syndrome.

Further treatment consists of prevention, in the form of educating patients in the use of insulin and encouraging them to carry glucose tablets or sweets with them at all times. A supply of glucagon (1 g I.M.) that can be administered by relatives may also be stored at home.

**Diabetic ketotic coma** Because patients who are comatose as a result of diabetic ketoacidosis often present in a profoundly dehydrated state, treatment must be initiated as soon as possible. The following are the key components of the management of diabetic ketoacidosis.

1. Replacement of fluid losses (the average fluid loss is approximately 7 L).
2. Normalization of electrolyte levels and careful monitoring of serum potassium.
3. Restoration of acid-base balance (bicarbonate infusion is reserved for severe cases).
4. Correction of insulin deficiency (4 to 6 U/hr).
5. Replenishment of energy stores (e.g., through dextrose infusion with insulin coverage until the patient can resume oral feeding).
6. Investigation to identify an underlying cause (infection is common).

**Hyperglycemic hyperosmolar nonketotic coma** Management of hyperosmolar nonketotic coma is essentially the same as that of diabetic ketotic coma. It is particularly important, however, that meticulous rehydration be performed, given that many patients with this condition are elderly.

#### Vegetative State

Approximately 10% to 12% of comatose patients eventually lapse into a vegetative state (VS). Whereas comatose patients have closed eyes and do not respond to pain, VS patients have spontaneous eye opening according to sleep-wake cycles and may have

minimal, purposeless movement. Specifically, the diagnosis of VS can be made if there is intermittent wakefulness (sleep-wake cycles); no evidence of awareness of self or environment or ability to interact with others; no evidence of sustained, reproducible, purposeful, or voluntary behavioral responses to external stimuli; no evidence of language comprehension or expression; and sufficiently preserved hypothalamic and brain stem autonomic function to permit survival with medical and nursing care. VS that persists for more than 1 month is referred to as a persistent vegetative state (PVS); PVS that lasts for 1 year or longer is considered permanent VS.<sup>11,12</sup>

### Prognosis

At least 50% of patients who are in traumatic VS will recover consciousness within 1 year, compared with only 15% of patients in nontraumatic VS. In one series, 69% of nontraumatic VS patients died, 20% survived with severe disability, and 8% survived without severe disability. Indicators of poor prognosis in nontraumatic VS patients include the absence of papillary reflexes for 24 hours after the event; no withdrawal from painful stimuli after 72 hours; the absence of roving eye movements after 7 days; and the absence of motor response to noxious stimuli after 72 hours.<sup>13-15</sup>

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