Emergency Neurological Life Support® Approach to the Patient With Coma Protocol Version 6.0

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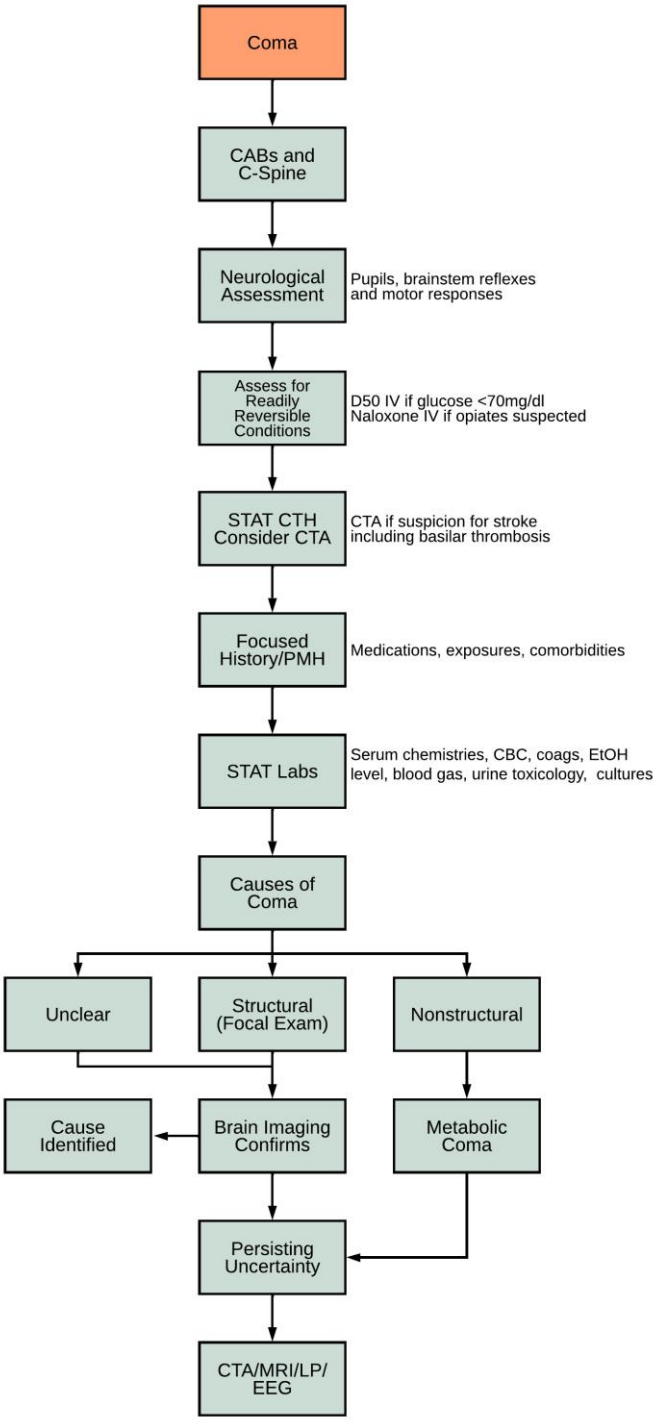
Last updated: August 2024

APPROACH TO THE PATIENT WITH COMA ALGORITHM



CHECKLIST

- ☐ Evaluate/ treat circulation, airway, breathing, and ventilation issues
- ☐ Ensure adequate immobilization of cervical spine, if warranted
- ☐ Exclude/ treat hypoglycemia or opioid/ benzodiazepine overdose
- ☐ Serum chemistries, arterial blood gas, urine toxicology screen
- ☐ Emergent head CT (CT angio head and neck, if appropriate) to determine if coma etiology is structural or vascular

COMMUNICATION

- ☐ Clinical presentation and time last seen well, if known
- ☐ Relevant past medical history/surgical/substance use history
- ☐ Findings on neurological exam including details with pertinent abnormalities.
    - Standardized communication when able: GCS, FOUR score, and pupillometery/NPi values
- ☐ Relevant resultant labs including glucose, blood gas, renal and hepatic function
    - Relevant pending labs, such as drug levels or infectious studies
- ☐ Brain imaging, LP, or EEG results, if available
- ☐ Treatments administered
- ☐ Next of kin/surrogate decision-maker

UNCONSCIOUS PATIENT

Coma is characterized by the absence of arousal, wakefulness, vigilance, and awareness of self and environment, lasting for more than 1 hour.

Determine unresponsiveness:

- Observation: eyes closed, immobility, lack of facial expression, obliviousness to environmental stimuli

Examiner evaluates response to graded stimulus:

- Verbal stimulus — Ask "Are you OK?" or "What is your name?" Other auditory stimulus may be a loud handclap.
- Tactile stimulus to body parts with large cortical representation — face and hands.
- Noxious stimulus — should be intense but not cause tissue injury. Recommended maneuvers include nail-bed pressure, trapezius squeeze, sternal rub, pressure on supraorbital ridge or on posterior aspect of mandibular ramus.

ASSESS CABS AND C-SPINE

The unconscious patient's Circulation, Airway, and Breathing (CABs) should be quickly assessed and concurrently treated. Verifying adequate circulation is the initial priority. This is followed closely by ensuring patency of the airway and adequate oxygenation and ventilation. The patient's cervical spine should be immobilized if the possibility of injury cannot be ruled out.

- Circulation, airway, and breathing are assessed and concurrently treated as detailed in ENLS protocol *Airway, Breathing and Mechanical Ventilation*.
- Rapid survey of head and neck, chest, abdomen, and extremities. Immobilize cervical spine if there is any likelihood of cervical spine instability.
- Bedside glucose testing is performed on all unconscious patients. If blood glucose is < 70 mg/dl, administer dextrose. Intravenous thiamine should be given prior to dextrose in patients at risk for nutritional deficiency (e.g., chronic alcohol users, bariatric surgery patients, patients with malabsorptive states) (refer to ENLS *Pharmacology* module for details).
- If there is suspicion of opioid toxidrome (e.g., history of drug use, coma, apnea or bradypnea, small pupils), administer naloxone IV/IM and repeat as needed up to 4 mg. Intranasal naloxone could be given initially, but switch to IV/IM when possible (Refer to ENLS *Pharmacology* module for details).

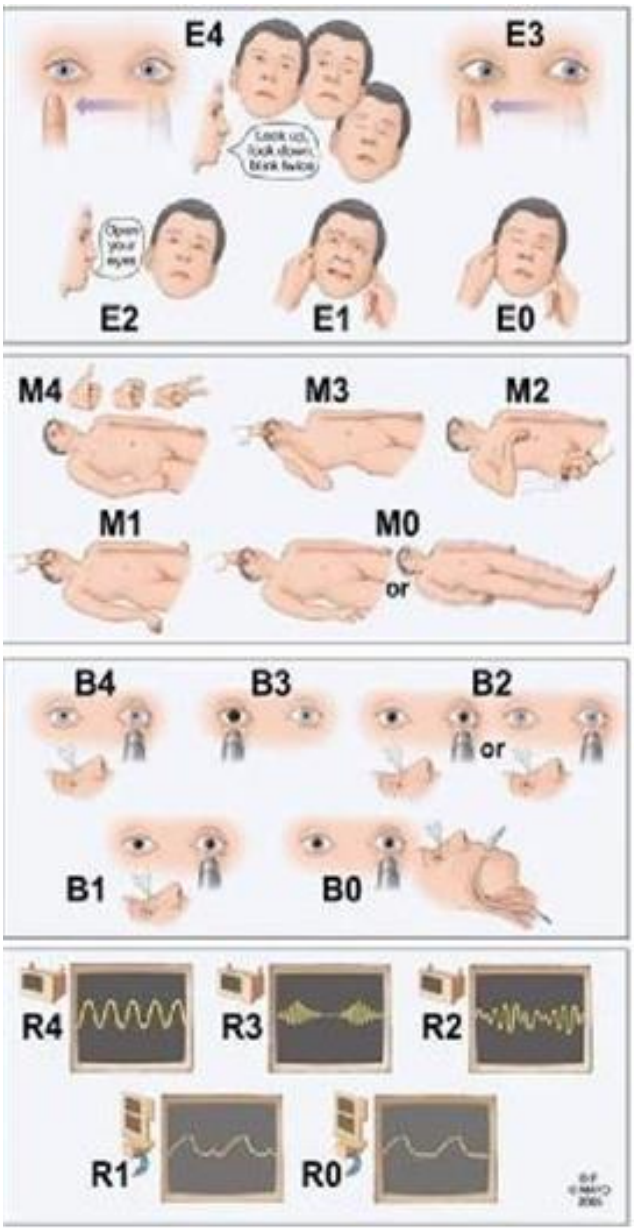
NEUROLOGICAL ASSESSMENT

Focused Neuro Exam

The emergency neurological assessment of the unconscious patient has four parts: level of consciousness, brainstem assessment, evaluation of motor responses, and appraisal of breathing patterns. Many scales are available to aide in emergent neurological assessment of a comatose patient

- Glasgow Coma Scale (GCS) for use in adult and pediatric patients

Examination	Response	Modified Pediatric Response (≤ 2 yrs)	Score (3-15)
Eye Opening Response (E)	Eyes open spontaneously	Eyes open spontaneously	4
	Eyes open to speech	Eyers open to speech	3
	Eyes open to pain	Eyes open to pain	2
	Eyes do not open	Eyes do not open	1
Motor Response (M)	Follows commands	Spontaneous movement	6
	Localizes (purposeful) movement toward a painful stimulus	Withdraws to touch	5
	Withdraws (normal flexion from pain	Withdraws to pain	4
	Abnormal flexion to pain (decorticate)	Abnormal flexion to pain (decorticate)	3
	Extension response to pain (decerebrate)	Extension response to pain (decerebrate)	2
	None	None	1
Verbal Response	Oriented	Coos/babbles	5
	Confused	Irritable/cries	4
	Inappropriate words	Cries to pain	3
	Incomprehensible sounds	Moans	2
	None	None	1



EYE RESPONSE

4 = Eyelids open or opened, tracking or blinking to command
3 = Eyelids open but not to tracking
2 = Eyelids closed but opens to loud voice
1 = Eyelids closed but opens to pain
0 = Eyelids remain closed with pain stimuli

MOTOR RESPONSE

4 = Thumbs up, fist, or peace sign
3 = Localizing to pain
2 = Flexion response to pain
1 = Extension response
0 = No response to pain or generalized Myoclonus status

BRAINSTEM REFLEXES

4 = Pupil and corneal reflexes present
3 = One pupil wide and fixed
2 = Pupil or corneal reflexes absent
1 = Pupil and corneal reflexes absent
0 = Absent pupil, corneal, or cough reflex

RESPIRATION

4 = Regular breathing pattern
3 = Cheyne-Stokes breathing pattern
2 = Irregular breathing
1 = Triggers ventilator or breathes above ventilator rate
0 = Apnea or breathes at ventilator rate

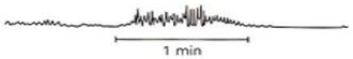
FOUR Score

- Level of consciousness: Refer to Glasgow Coma Scale or FOUR Score. Assess additional potential signs of arousal including visual fixation, visual pursuit (tracking), and forced eye closure/resisting the examiner.
- Brainstem (cranial nerve) examination:
- Pupillary size, reactivity, and symmetry (see Table for pupillary changes reflecting underlying etiology)
  - Corneal reflex
  - Visual threat response
  - Oculocephalic reflex (avoid if there is any change of cervical spine instability)
  - Gag reflex
  - Cough reflex
- Motor function: Spontaneous movements or posturing, response to verbal command, response to noxious stimulus. Examiner should distinguish purposeful from reflexive activity. Examples of purposeful activity include following commands, pushing examiner away, reaching for endotracheal tube, localizing to noxious stimulus. Examples of reflexive activity include withdrawal, flexion, or extension to noxious stimulus or spontaneously.

Pupillary changes reflecting underlying etiology

Pupillary change	Possible etiologies/localization
Pinpoint pupil	Opioids Cholinergic intoxication Pontine damage (interrupts descending sympathetic pathways)
Dilated, reactive pupils	Pretectal lesions Stimulants (cocaine, methamphetamine), hallucinogens including PCP/LSD
Anisocoria (pupillary asymmetry)	3rd nerve compression from uncal herniation Localized drug effect (e.g., ipratropium, tropicamide)
Dilated, non-reactive pupils	Bilateral 3rd nerve compression or ischemia (e.g., global cerebral anoxia, brain death) Barbiturates Atropine Hypothermia
Mid-position, fixed or irregular	Midbrain lesion

- Breathing pattern: The breathing pattern may have localizing value in comatose patients with brainstem lesions
  - Cheynes stokes — global metabolic encephalopathy, impaired forebrain or diencephalon
  - Central neurogenic hyperventilation: metabolic encephalopathy, high brainstem tumors (rare)
  - Apneusis bilateral pons lesion
  - Cluster breathing or ataxic breathing — pontomedullary junction lesion
- Apnea — lesions affecting ventrolateral medulla bilaterally

Respiratory Patterns	Patterns	Localization
Cheyne-Stokes		Global/metabolic encephalopathy Impaired forebrain or diencephalon
Central neurogenic hyperventilation		Metabolic encephalopathy High brainstem tumors (rare)
Apneusis		Bilateral pontine lesions
Cluster breathing/ataxic breathing		Pontomedullary junction lesions
Apnea		Lesions affecting ventrolateral medulla bilaterally (ventral respiratory group)

STAT HEAD CT

Consider CT angiography of the head and neck in appropriate circumstances

Head CT will help assess for possibility of acute intracranial process. Primary neurological etiologies of coma are described in the table below, many of which may be apparent on noncontrast head CT.

Use caution in the following situations: ruling out acute ischemic stroke or brainstem pathology as head CT may be negative early on. Metabolic, toxic and pharmacologic intoxications leading to coma, which may be identified by relevant laboratory testing may additionally have negative head CT imaging.

Noncontrast head CT should be obtained emergently in any unconscious patient with a presumed structural cause of coma or with an unclear cause of coma after initial assessment and stabilization of CAB and the cervical spine.

If an acute ischemic stroke is suspected, CT angiography of the head and neck, as well as consideration of head CT perfusion imaging, could be considered (see ENLS module *Acute Ischemic Stroke*). When basilar artery thrombosis is a consideration in sudden onset coma and CT angiography of the head and neck will be diagnostic. If head CT without angiography is done, the basilar artery may be abnormally hyperdense — this may suggest basilar artery thrombosis. A rapid sequence MRI brain may be obtained if there is suspicion of a hyperacute ischemic stroke or when the cause of coma is not otherwise explained by other tests.

When a CNS infection is being considered, head CT with and without contrast should be obtained to evaluate for abscess, extra-axial fluid collections, hydrocephalus, hemorrhagic transformation, and vasculitic infarcts.

Primary neurological etiologies of coma

Primary Neurological Etiologies of Coma	
Cause	**Exam/History Findings**
Trauma	
Subdural hematoma	Focal weakness, seizure, altered level of consciousness, aphasia
Epidural hematoma	Lucid period with rapid decline
Parenchymal hemorrhage	Focal neurological findings
Diffuse axonal injury	Nonfocal exam, seizure, dense coma
Neurovascular	
Intracerebral hemorrhage	Focal neurological findings; Coma if brainstem hemorrhage or overlapping hydrocephalus, seizure
Subarachnoid hemorrhage	Coma with/without focal findings
Ischemic stroke	Focal findings consistent with vascular distribution Coma if basilar artery occlusion, bilateral thalamic infarct (Artery of Percheron), diffuse multifocal infarcts or late presentation with cerebral/cerebellar edema, seizure
CNS infections	
Meningitis	Fever, meningismus, seizures. Stupor and coma when associated with meningoencephalitis, cerebral edema and/or hydrocephalus
Encephalitis	Fever, coma or stupor, seizures
Abscess	Focal neurological deficits, exposure history, seizures
Neuroinflammatory disorders	
Acute disseminated encephalomyelitis	History of preceding illness, headache, fever, N/V, acute coma, focal motor deficits, brainstem findings
Autoimmune encephalitis	Subacute progression, seizures, psychiatric symptoms
Neoplasms	
Metastatic	History of primary cancer, focal findings, slow progressive symptoms
Primary CNS	Focal neurological deficits, neuropsychiatric symptoms, seizures, cranial neuropathies
Carcinomatous meningitis	Headache, meningismus , encephalitis/stupor, seizures, cranial neuropathies, cerebellar symptoms

Primary neurological etiologies of coma (cont'd)

Primary Neurological Etiologies of Coma	
Cause	**Exam/History findings**
Seizures	
Non-convulsive seizures or status epilepticus	Epilepsy/recent seizure, acute neurologic injury or recent neurosurgical procedure, sepsis, unexplained coma
Postictal state	Recent seizure, focal neurological deficits (Todd's paralysis), EEG without ongoing seizure
Other	
Posterior Reversible Encephalopathy Syndrome (PRES)	Severe hypertension, sympathomimetic use, history of immunosuppressant or chemotherapeutic use Pregnancy
Osmotic demyelination syndrome	History of polydipsia, vomiting, excessive alcohol use or prior low sodium levels
Anoxic-ischemic encephalopathy	History of cardiac arrest or asphyxiation
Hydrocephalus	History of ventriculoperitoneal shunt, cerebral hemorrhage, stroke, SAH, meningoencephalitis, congenital infections, intracranial mass

Metabolic / Toxic & Environmental Etiologies of Coma

Metabolic Encephalopathies	Exam / History Findings	Tests to Consider
Hypoglycemia	Seizures, evidence of adrenal insufficiency	Glucose, proinsulin
Hypoxia, hypercapnia	Respiratory distress, tachypnea	Arterial or venous blood gas, pulse oximetry
Diabetic ketoacidosis; hyperosmolar hyperglycemic state	Deep and rapid breathing, seizures, focal neurological findings, acetone smell, shock	Glucose, osmolar gap, ketones, basic metabolic panel, ABG Remember to correct sodium level for hyperglycemia
Hepatic encephalopathy	Jaundice, hyperreflexia, rigidity myoclonus, bradykinesia, asterixis	Ammonia, hepatic function panel, coagulation panel
Uremia	Prior lethargy, disorientation, hallucinations; diffuse weakness, tremor, myoclonus, asterixis	Basic metabolic panel; renal ultrasound
Hyponatremia	Moderate symptoms: nausea, headache, confusion Severe symptoms: vomiting, seizures, coma, cerebral edema on imaging	Basic metabolic panel; serum & urine osmolarity, serum & urine sodium level
Hypernatremia	Clinical signs of dehydration, subdural hygromas or pituitary on CT, history of diabetes insipidus, history of lithium use	Basic metabolic panel; urine osmolarity (if diabetes insipidus is suspected)
Myxedema coma	Hypothermia, hypotension	Thyroid function panel, EKG
Thyrotoxicosis	Tachycardia, hypertension, cardiac arrhythmias	Thyroid function panel, EKG
Adrenal failure	Hypotension, hypothermia	Cortisol

Metabolic Encephalopathies	Exam / History Findings	Tests to Consider
Hypercalcemia	May include constipation, nausea, anorexia, cognitive dysfunction	Total serum calcium or ionized serum calcium
Wernicke's disease	Confusion, ataxia, ophthalmoparesis, history of vomiting and malnutrition, hyperemesis, gravidarum, malabsorption or short gut syndrome	Thiamine level (history and clinical exam are more important than laboratory testing)
Sepsis	Fever, tachycardia, hypotension. • In early shock: warm hands, brisk capillary refill, bounding pulse • In late shock: thready pulse, worsening hypotension, cold hands, poor capillary refill	Complete blood count, blood urine and/or respiratory cultures as appropriate, serum lactic acid, procalcitonin

Drug / medication overdose	Exam / History Findings	Tests to Consider
Abuse (opioids, alcohol, amphetamine, cocaine)	Pupillary changes, HR changes, stigmata of cirrhosis suggesting ETOH, injection markings on skin, respiratory failure	Toxicology screen: Consider quantitative pupillometry if visual inspection is concerning for nonreactive pupils
Methanol, Ethylene Glycol	Obtaining a history of quantity and time of ingestion is critical Comas, seizures, hyperpnea, hypotension, a relative afferent pupillary defect in methanol toxicity, tetany and cranial nerve palsies Examine the clothes, mouth, vomitus and/or urine for fluorescence due to fluorescein in commercial antifreeze preparations	Toxicology screen: measure anion gap, osmolar gap, serum methanol and ethylene glycol levels, basic metabolic panel, urine microscopy for oxalic acid crystals and EKG Consider neuroimaging if focal deficits or brainstem dysfunction and normal head CT
Sedatives: hypnotics benzodiazepines, non-benzodiazepine hypnotics, muscle relaxants and barbiturates	Bradycardia, respiratory depression Carisoprodol can produce anticholinergic side effects Baclofen overdose can sauce seizures	Toxicology screen: EKG
Narcotics: opium, heroin, codeine, oxycodone, hydrocodone, tramadol, morphine, hydromorphone, fentanyl, carfentanil	Constricted or pinpoint pupils, decreased respiratory rate, Tramadol overdose can present with seizures and serotonin syndrome	Toxicology screen: Fentanyl analogues such are carfentanil may not be detected on standard urine drug screens
Aspirin	Tinnitus, vertigo, nausea/vomiting/diarrhea, hyperpyrexia, noncardiac pulmonary edema, cerebral edema is possible in salicylate toxicity	Arterial blood gas, toxicology screen, serum acetaminophen level, CMP, consider neuroimaging

Drug / medication overdose	Exam / History Findings	Tests to Consider
Acetaminophen	Acute liver failure, nausea/vomiting, diaphoresis, pallor oliguria, hepatomegaly, jaundice, encephalopathy, asterixis, coma	Acetaminophen level, hepatic function panel, coagulation panel, basic metabolic panel, plasma ammonia level
Serotonergic medications i.e. SSRI, SNRI, MAO inhibitors, fentanyl	Agitated delirium, seizure, diaphoresis, mydriasis, hyperthermia, ocular clonus, hypertonia, hyperreflexia, tremor, ankle clonus, seizures, rigidity Hunter criteria for diagnosis	Toxicology screen, creatine, kinase, basic metabolic panel, calcium, magnesium and phosphate, coagulation panel and serum lactate
Tricyclic antidepressants	Refractory hypotension, arrhythmias, dilated pupils, dry warm skin and mouth, constipation, hyperthermia, seizures, prolonged QRS	Electrocardiogram, urine/serum tricyclic antidepressants if diagnosis is uncertain, arterial blood gas
Antipsychotics	Variable: mild hypotension, pupil variability, tachycardia, hyperthermia, extrapyramidal side effects, acute dystonia, akathisia, anticholinergic toxicity, ventricular arrhythmias	Electrocardiogram, toxicology screen, basic metabolic panel, magnesium, calcium
Antiseizure medications	Variable depending on the medication used	Drug levels, toxicology screen, electrocardiogram

Environmental Causes	Exam / History Findings	Tests to Consider
Heat stroke	Hyperthermia, tachycardia, tachypnea, widened pulse pressure, hypotension. May develop rhabdomyolysis and acute liver failure due to shock liver	Complete blood count, basic metabolic panel, electrocardiogram, creatinine kinase, liver function panel
Hypothermia	Hypoventilation, pulmonary edema, hypotension, bradycardia, arrhythmia, oliguria if severe	Complete blood count, basic metabolic panel, electrocardiogram
Carbon monoxide	Variable: seizures, syncope, coma, arrhythmia, pulmonary edema, lactic acidosis	Complete blood count, co-oximetry, electrocardiogram, basic metabolic panel, chest x-ray

FOCUSED HISTORY OF PRESENT ILLNESS

Patient history is obtained concurrently with resuscitative measures. Historical information elicited from witnesses, friends, family, co-workers, or EMS personnel may suggest the cause of coma. EMS personnel may have invaluable details about the circumstances in which the patient was found. Medical and surgical history, prescription medications, alcohol or substance use, environmental exposures or evidence of trauma should be systematically queried.

The timeline leading to loss of consciousness may be helpful in suggesting an etiology. An abrupt onset may suggest a stroke, seizure, or cardiac event with impaired cerebral perfusion. A more gradual onset of coma may suggest a metabolic, toxic, or infectious process.

STAT LABS

Laboratory studies should be obtained emergently, and point-of-care (POC) testing should be utilized when possible. Studies include serum chemistries, complete blood counts, coagulation studies, EtOH level, blood gas, serum and urine toxicology, and blood cultures when indicated.

- Serum chemistries should include Na, K, creatinine, BUN, and transaminases
- Hematological panel including hemoglobin/hematocrit, platelets, and white blood cell count; coagulation studies
- Arterial blood gas
- Toxicology: Serum studies such as alcohol level, acetaminophen, salicylates or dedicated drug levels when appropriate; urine toxicology screen for opioids, benzodiazepines, illicit drugs. (Note: Some toxins that cause unconsciousness are not detectable in common toxicology screens)
- Microbiology: Urinalysis, urine culture, blood and sputum cultures

CAUSES OF COMA

Information accrued so far is used to establish a preliminary impression of the etiology of coma: either structural, a nonstructural cause, or an unclear etiology. Structural and nonstructural causes of coma may coexist. Caution must be exercised in patients with non-focal exam and unrevealing head CT, as brainstem strokes or nonconvulsive seizures can present without focal or structural abnormalities apparent initially. Orofacial dyskinesia and posturing may be seen in brainstem stroke and may be mistaken for seizures.

UNCLEAR ETIOLOGY

In many patients, the etiology of coma cannot be easily identified after initial assessment or emergent noncontrast head CT. Advanced imaging like previously described CT angiography, perfusion imaging rapid sequence MRI or contrasted head CT studies should be considered if there remains ongoing suspicion of ischemic stroke or occult pathology. Additionally, diagnostics should be considered when uncertainty persists, including a lumbar puncture (LP) and EEG. A LP may be indicated when CNS infection, neuroinflammatory, autoimmune disorders, or CNS involvement of hematological or solid organ cancers is suspected. Additionally, when there is clinical suspicion of an aneurysmal subarachnoid hemorrhage presenting more than 24 hours after onset of headache, an LP should be obtained even if the noncontrast CT is negative. Caution must be exercised to ensure there are no clinical or radiographic signs of elevated intracranial pressure prior to lumbar puncture to avoid risk of herniation.

STRUCTURAL

Structural causes of coma include traumatic brain injury, acute ischemic stroke, intracerebral hemorrhage, meningitis, encephalitis, brain tumors, or other mass lesions and typically present with a focal neurological exam.

Management should be initiated in consultation with appropriate subspecialists: Neurology and/or neurosurgery.

Focal neurologic findings on physical examination suggest a localized brain lesion. Consider a structural etiology when the following are encountered:

- A history of trauma, acute onset of symptoms, immunodeficiency, or malignancy
- A physical examination that has asymmetric findings on cranial nerve, or motor responses (e.g., hemiparesis)
- Absence of an obvious toxic-metabolic etiology

Unless proven otherwise, coma is presumed to be structural in origin and should be immediately assessed with a non-contrast head CT since emergent neurosurgical management may be needed. Emergent CT angiography should be considered in any patient with an exam concerning for focal brainstem findings and suspected basilar artery ischemia.

Patients with new-onset seizures, a change in seizure pattern, or status epilepticus should be evaluated for structural abnormalities. See ENLS protocol *Status Epilepticus*.

NONSTRUCTURAL

Caution must be exercised in patients with nonfocal neurologic exams and unrevealing head CT imaging. Brainstem ischemia or nonconvulsive seizures can present nonfocally.

A nonstructural cause of coma may be suggested by:

- A progressive, gradual onset of symptoms
- History of medication, alcohol, or illicit substance use; metabolic derangement; or toxic exposure
- Nonfocal neurological exam with symmetric cranial nerve and motor findings (Table 9 highlights some important, non-neurological causes of coma).

METABOLIC COMA

Common nonstructural causes of coma include anoxic-ischemic encephalopathy, seizures, metabolic derangements, endocrinopathies, systemic infections, CNS infections, medication overdose, alcohol and illicit substance use and intoxication, or toxic exposure (Tables 8 and 9).

Treatment is guided by the underlying etiology. Where appropriate, specific antagonists/antidotes should be administered. For example:

- Opioid overdose: naloxone See the ENLS *Pharmacology* protocol
- Acetaminophen overdose: N-acetylcysteine See the ENLS *Pharmacology* protocol
- In some cases, a primary metabolic encephalopathy may evolve toward a structural process, such as acute liver failure leading to cerebral edema and herniation.
- Severe hyponatremia can contribute to coma and should be managed with caution.
- Wernicke's encephalopathy may not present with the full classic triad of encephalopathy, ataxic gait, and ophthalmoplegia. High-dose thiamine should be initiated in patients with coma and risk factors for Wernicke's encephalopathy.
- Seizures and status epilepticus commonly are not associated with any detectable lesion on cranial CT. However, in patients with new-onset seizures or a change in seizure pattern, a structural cause must be excluded with cranial CT or MRI. CNS infections may have no structural correlate on noncontrast CT or MRI; however, this study should be obtained with and without contrast to exclude brain abscess.
- Remember to initiate antimicrobials and dexamethasone (if indicated) prior to the head CT if you suspect infectious meningoencephalitis and specifically do not delay therapy for a diagnostic lumbar puncture. Do not forget to treat empirically for *Listeria* in at-risk populations (immunocompromised persons, neonates, the elderly, etc.) or for HSV meningitis with acyclovir if the clinical situation is suggestive, such as a patient with fever and seizures.

PERSISTING UNCERTAINTY

Next Steps

Depending on the history and presentation, advanced imaging like CT angiogram, perfusion imaging, and rapid sequence MRI must be considered if initial CT head is non-contributory. Stat EEG may be considered to assess for non-convulsive seizures.

When diagnostic uncertainty persists despite initial assessment, additional test measures include:

- Noncontrast head CT is obtained in all comatose patients with an undiagnosed etiology, if not done already.
- Consider basilar artery thrombosis (look for a hyperdense basilar artery sign on noncontrast head CT); CTangiography (CTA) or MR-angiography (MRA) is definitive.
- EEG to evaluate for non-convulsive seizures or status epilepticus, burst suppression, or patterns consistent with metabolic encephalopathy. Be aware of dyskinesia seen in brainstem stroke that may mimic seizures.
- Lumbar puncture (LP) is obtained if there is suspicion of CNS infection, inflammation, infiltration with lymphoma or malignant cells, or to substantiate a suspicion of aneurysmal subarachnoid hemorrhage in patients with negative CT findings. A space occupying lesion should be ruled out with noncontrast head CT prior to performing the LP.
- MRI is obtained when the cause of coma is not explained by other tests or if there is a presumption of hyperacute ischemic stroke.