Emergency Neurological Life Support Approach to the Patient with Coma Protocol Version 5.0

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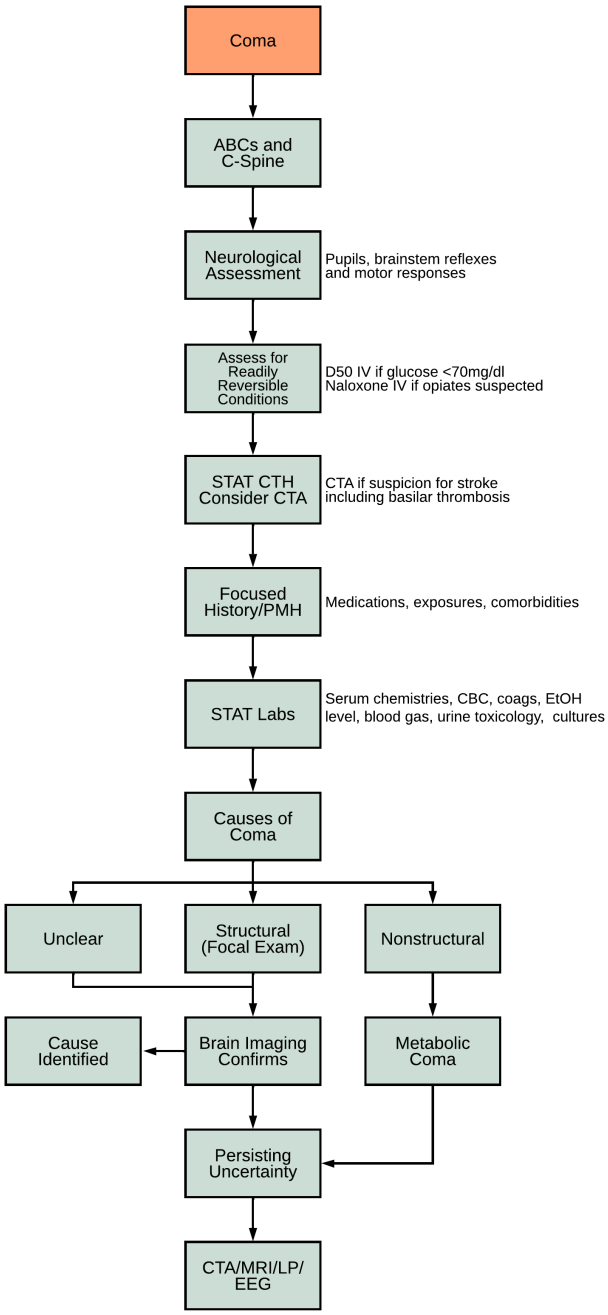
Approach to the Patient with Coma Protocol

↑ Flowchart ↑

Approach to the Patient with Coma Algorithm



Approach to the Patient with Coma Protocol

↑ Flowchart ↑

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 cranial CT (CT angio brain if appropriate) to determine if coma etiology is structural or vascular

Communication

☐ Physician and advanced provider communication
☐ Clinical presentation and time last seen well if known including history from bystanders, witnesses, contextual or environmental observations (e.g., pill bottles, seizures, trauma, etc.)
☐ Findings on neurological examination including details on GCS components and any abnormality with brainstem reflexes, if found
☐ Relevant past medical history/surgical history
☐ Relevant laboratory tests including glucose, blood gas, renal and hepatic function
☐ Brain imaging, LP, or EEG results (if available)
☐ Treatments administered so far
☐ Nursing, physician/advance practice provider communication

Sample Sign-off Narrative:
"54-year-old male patient was found unresponsive at home with agonal respirations. Unknown last well time. Bottle of narcotics found at bedside. He was intubated on scene without sedatives, paralytics. Given naloxone at scene without significant improvement in exam. Hypertensive with SBP in 190s; given labetalol, now in the 140s. No spontaneous respirations on ventilator on controlled mechanical ventilation at 100% FiO_2 with tidal volume of 350 ml and PEEP 8 saturating 95%, $PaCO_2$ on blood gas was 38 mmHg; 5.06 kPa (goal PCO_2 35–40 mmHg; 4.6–5.3 kPa). On exam, GCS is 4T, X time after intubation. He is not responsive, does not attend. He has absent brainstem reflexes with extension to motor response to pain but pinpoint pupils. Toxicology screen positive for opioids and cocaine. Neurological consults or neuroimaging CT angio are not available here; hence being transferred."

Unconscious Patient

Eyes closed, unresponsive

A patient who has eyes closed and is unresponsive is considered comatose.

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 sternal rub, nail-bed pressure, pressure on supraorbital ridge or on posterior aspect of mandibular ramus.

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↑ Flowchart ↑

Assess ABCs and C-Spine

The unconscious patient's ABCs should be quickly assessed and concurrently treated (see ENLS protocol Airway, Ventilation, and Sedation). Verifying patency of the airway is an overriding initial priority to ensure adequate oxygenation and ventilation. The patient's cervical spine should be immobilized if the possibility of injury cannot be ruled out.

- Airway, breathing and circulation are assessed and concurrently treated as detailed in ENLS protocol Airway, Ventilation and Sedation.
- Rapid survey of head and neck, chest, abdomen, and extremities. Cervical spine is immobilized if there is any likelihood of traumatic instability.
- Bedside glucose testing is performed on all unconscious patients. If blood glucose is < 70 mg/dl administer 20-50 ml of 50% dextrose. Thiamine 100 mg IV 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) (see Table 3, Prehospital pharmacological therapy for coma).
- If there is suspicion of opioid toxidrome (e.g., history of drug use, coma, apnea or bradypnea, small pupils), administer naloxone 0.04-0.4 mg IV/IM and repeat as needed in total dosing up to 4 mg. 1–2 mg per nare into both nares can be given initially but switch to IV/IM when possible (see Table 3, Prehospital pharmacological therapy for coma).

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.

See Table 4 for Adult Glasgow Coma Scale
See Table 5 for Pediatric Glasgow Coma Scale
See Tables 6 for pupillary changes
See Chart 1 for respiratory changes reflecting underlying etiology
See Figure 2 for FOUR Score

- Level of consciousness: Refer to Glasgow Coma Scale (see Tables 4 and 5) or FOUR Score (see Figure 2). 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 6 for pupillary changes reflecting underlying etiology)
  - Corneal reflex
  - Visual threat response
  - Oculocephalic reflex (doll's eyes - only if no suspicion of cervical instability)
  - Gag reflex
  - Cough reflex
- Motor function: Spontaneous muscle position/posture, spontaneous movements, 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.
- Breathing pattern: The breathing pattern may have localizing value in comatose patients with brainstem lesions (see Chart 1 for respiratory pattern reflecting underlying etiology)
  - 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

Assess for Readily Reversible Conditions

Prehospital and initial hospital evaluation should focus on assessing and treating readily reversible conditions like airway compromise, hypotension, hypoglycemia, opioid overdose (see table 3, Prehospital pharmacological therapy for Coma).

STAT CTH

Consider CTA

Head CT will help assess for possibility of acute intracranial process. Primary neurological etiologies of coma are described in Table 7, many of which may be apparent on noncontrast CT head. Use caution in ruling out ischemic stroke and brainstem pathology as head CT may be negative early on. See Table 9 for metabolic, toxic and environmental etiologies of coma, for which relevant laboratory testing should be performed routinely in the initial management of coma.

Non-contrast cranial CT should be obtained emergently in unconscious patients with a presumed structural cause of coma and in patients with an unclear cause of coma after initial assessment of ABCs and cervical spine stabilization.

If an acute ischemic stroke is suspected, cranial CT angiography and CT perfusion can provide valuable information on vascular patency and regional perfusion (see ENLS protocol Acute Ischemic Stroke). Basilar artery thrombosis is a consideration in sudden onset coma and CT angiography will be diagnostic. If CT alone is done, look at the basilar artery and see if it is abnormally hyperdense - this may suggest basilar artery thrombosis. A rapid sequence MRI may be obtained if there is a presumption of hyperacute ischemic stroke or when the cause of coma is not explained by other tests.

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

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↑ Flowchart ↑

Focused History/PMH

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 valuable details about the circumstances in which the patient was found. Medical and surgical history, medications, alcohol and illicit drug use, and environmental exposures or evidence of trauma should be systematically queried.

The time course of the alteration in consciousness may be helpful in suggesting etiology. An abrupt onset suggests a stroke, seizure, or a cardiac event with impaired cerebral perfusion. A more gradual onset of coma suggests a metabolic or possibly infectious process.

STAT Labs

Unless a readily reversible cause of unresponsiveness has been discovered and corrected, additional laboratory work (serum chemistries, CBC, coags, EtOH level, blood gas, urine toxicology, cultures) is obtained emergently. Point of care (POC) testing should be utilized where available.

- Serum chemistries including Na, K, creatinine, BUN, and transaminases
- Hematological panel including hemoglobin/hematocrit, platelets, and white blood cell count; coagulation studies
- Arterial blood gas
- Toxicology: Blood alcohol level; urine toxicology screen for opioids, benzodiazepines, illicit drugs. (Note: Some toxins that cause unconsciousness are not detectable in common toxicology screens); acetaminophen & salicylate levels if warranted
- Microbiology: Urinalysis; urine culture; blood cultures

Causes of Coma

Three possibilities

Information accrued so far is used to establish a preliminary impression of either a structural cause, a nonstructural cause, or an unclear cause. Structural and nonstructural causes of coma may coexist. Caution must be exercised in patients with non-focal exam and noncontributory CT head as brainstem stroke or nonconvulsive seizures can present without focal or structural abnormalities apparent initially. Orofacial dyskinesias and posturing may be seen in brainstem stroke and may be mistaken for seizures. Please see Tables 7 and 8 for neurological and non-neurological causes of coma.

Unclear Etiology

In many patients, the etiology of coma cannot be easily identified after initial assessment or emergent non-contrast cranial CT. Advanced imaging like CT angiogram, perfusion imaging or rapid sequence MRI or contrast imaging should be considered if suspicion of ischemic stroke or occult pathology exists based on risk factors. If diagnostic uncertainty persists, a lumbar puncture (LP) and EEG. A LP may be indicated for suspicion of CNS infection, neuroinflammatory and autoimmune disorders, and suspected central nervous system involvement of hematological or solid organ cancers. 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 non-contrast CT is negative.

Structural

(Focal Exam)

Structural causes of coma include Traumatic Brain Injury, Acute Ischemic Stroke, Intracerebral Hemorrhage, Meningitis and Encephalitis, and brain tumor and other mass lesions.

Management should be initiated in consultation with Neurology and/or Neurosurgery.

Asymmetric or focal findings on physical examination suggest a localized brain lesion or disturbance. A structural etiology is suggested by:

- History: trauma, acute onset of symptoms, immunodeficiency, malignancy
- Physical examination: asymmetric cranial nerve findings, asymmetric 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 cranial 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 or suspected vertebrobasilar ischemia.

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

Nonstructural

Caution must be exercised in patients with non-focal exam and noncontributory CT head as brainstem stroke or nonconvulsive seizures can present this way.

A nonstructural cause of coma is suggested by:

- Progressive, gradual onset of symptoms
- History of medication, alcohol, or illicit drug use, or environmental toxic exposure
- Non-focal neurological exam with symmetric cranial nerve and motor findings (Table 8 highlights some important, non-neurological causes of coma).

Metabolic Coma

Global or metabolic causes

Common nonstructural causes of coma include anoxic-ischemic encephalopathy, seizures, metabolic alterations, endocrinopathies, systemic infections, CNS infections, medication overdose, alcohol and illicit drug use, and exposure to nonpharmacologic neurotoxic compounds (Tables 7 & 8).

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

- Opioid overdose: naloxone (Table 3)
- Acetaminophen overdose: N-acetylcysteine (Table 3)
- In some cases, a primary metabolic encephalopathy may evolve toward a structural process, such as acute liver failure leading to cerebral edema and herniation. Table 9 lists causes of hyperammonemia
- Severe hyponatremia can contribute to coma and should be managed according to the algorithm outlined in Table 10
- 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 (see Table 11)
- 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 non-contrast 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, neonates, 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, 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:

- Non-contrast 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 non-contrast head CT); CT-Angiography (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 dyskinesias 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 lesions should be ruled out with non-contrast 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.
- Consultation with a specialist.