PEDIATRIC ALTERED LEVEL OF CONCIOUSNESS

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- **Coma** – Coma, a state of "unarousable unresponsiveness," is the most profound degree to which arousal and consciousness are impaired
- **Lethargy** – Lethargy, obtundation, and stupor refer to states in which arousal is somewhat less impaired. These patients have some difficulty maintaining attention during an examination, tend to fall asleep when not stimulated, and respond poorly (if at all) to questions and commands. These terms are imprecise. In clinical situations, it is more useful to describe the patient's responses to specific stimuli
- **Delirium** – Delirium is a disturbance of consciousness with reduced ability to focus, sustain, or shift attention. Patients with hyperactive delirium show both hyperactivity and diminished sleep. Confusion, excitement, hallucinations, and irritability are common. Patients with hypoactive delirium have little interaction with their environment and often appear somnolent

- **Persistent vegetative state** – Persistent vegetative state (PVS) describes patients who are completely unconscious but have spontaneous eye opening during cyclical periods of arousal . Such patients often have reflexive vocalizations (sounds but not words), facial expressions, and movements that can be misinterpreted by hopeful observers as reflecting awareness of their internal or external environment. Required features of PVS:
- **Minimally conscious state** – The minimally conscious state (MCS) describes patients with severe alteration in consciousness who nonetheless can be shown to have some degree of preserved awareness of the external world. They can at least occasionally demonstrate purposeful movements or responses. These can include following simple commands, making gestural or verbal responses to questions, making intelligible verbalizations, smiling or crying in response to evocative sounds or images, reaching accurately toward the location of an object, or fixating on and pursuing visual stimuli. Functional neuroimaging studies suggest that these patients have a different, less severe neuroanatomic substrate than do patients in PVS.
- **Brain death** – Brain death criteria include coma, apnea, and absent brainstem reflexes. A diagnosis of brain death specifically implies no chance of recovery and is synonymous with death in most countries. Age-specific diagnostic criteria exist.

HISTORY

- The etiology may be apparent from the history, such as when coma results from the expected progression or complication of a known illness or injury(e.g. child who presents after a drowning injury or a child with diabetes presenting with hypoglycemic coma or with cerebral edema from diabetic ketoacidosis)
- The history of symptoms leading up to coma may also provide clues. Abrupt and unexplained (intracranial hemorrhage, seizure, trauma, or intoxication)

A gradual deterioration of mental status(infectious process, metabolic abnormality, slowly expanding intracranial mass lesion)

A history of preceding headache, double vision, or nausea suggests increased intracranial pressure (ICP)

Inborn errors of metabolism may also present with slowly evolving coma or recurrent episodic coma .

A history from the caregiver that is vague or inconsistent with the examination may engender suspicion for nonaccidental trauma

Toxic ingestions in young children are often unwitnessed, and parents should be questioned regarding the possibility of available substances

PHYSICAL EXAM

- Temperature – Hyperthermia suggests infection, but is also seen with inflammatory disorders, environmental or exertional heat stroke, neuroleptic malignant syndrome, status epilepticus, hyperthyroidism, and anticholinergic poisoning. When infection is suspected, possible complications such as intracranial seeding, septic shock, seizures, Reye syndrome, and exacerbation of an underlying metabolic disorder should also be considered
- Hypothermia can occur with infection in infants but is more often due to drug intoxication, environmental exposure, or hypothyroidism. Hypothermia itself blunts cognitive function and arousal, presumably by decreasing cerebral blood flow
- Heart rate – Tachycardia can occur with fever, pain, hypovolemia, cardiomyopathy, tachyarrhythmia, and also in status epilepticus. Bradycardia occurs with hypoxemia, hypothermia, and increased ICP as part of Cushing triad (bradycardia, hypertension, irregular respirations

PHYSICAL EXAM

- Respirations – Tachypnea can be seen with pain, hypoxia, metabolic acidosis, and pontine injury. Slow, irregular, or periodic respirations occur with metabolic alkalosis, diabetic ketoacidosis, sedative intoxication, and injury to extrapontine portions of the brainstem
- Blood pressure – Hypotension suggests hypovolemic, septic, or cardiogenic shock, intoxication, or adrenal insufficiency. Impaired consciousness may be an early indicator of poor end-organ perfusion in a patient with shock. Hypertension may be due to: Pain or agitation, Certain toxidromes (eg, sympathomimetics, stimulants), Increased ICP: Hypertension associated with bradycardia and irregular respirations is referred to as "Cushing triad" in this setting

PHYSICAL EXAM

The skin appearance provides useful information

- Mottling and delayed capillary refill suggest a shock state
- Bruising suggests traumatic injury (including abusive head trauma)
- Petechial and purpuric rashes may be suggestive of meningococcal infection
- Jaundice may suggest hepatic encephalopathy
- A cherry-red appearance is suggestive of carbon monoxide poisoning

PHYSICAL EXAM

- **Funduscopy** – Papilledema suggests increased ICP of more than several hours duration. Retinal hemorrhages are most commonly associated with shaken baby syndrome
- **Meningismus** – Meningeal irritation or inflammation suggesting meningitis is demonstrated by passive resistance to neck flexion (nuchal rigidity), involuntary knee flexion with forced hip flexion (Kernig sign), or involuntary hip and knee flexion with forced neck flexion (Brudzinski sign). These signs are often absent in infants and young children

NEUROLOGIC EXAMINATION

Directed at determining whether the pathology is structural or due to a systemic metabolic derangement (including drug toxicity or infection)

- Level of consciousness
- Pupil responsivity
- Brainstem reflexes: pupillary responses to light, extraocular movements, and corneal reflexes
- Motor responses
- Important findings are abnormal reflexes that indicate dysfunction in specific regions of the brainstem and/or impending transtentorial herniation, or a consistent asymmetry between right- and left-sided responses

Sign	Glasgow Coma Scale[1]	Pediatric Glasgow Coma Scale[2]	Score
Eye opening	Spontaneous	Spontaneous	4
	To command	To sound	3
	To pain	To pain	2
	None	None	1
Verbal response	Oriented	Age-appropriate vocalization, smile, or orientation to sound, interacts (coos, babbles), follows objects	5
	Confused, disoriented	Cries, irritable	4
	Inappropriate words	Cries to pain	3
	Incomprehensible sounds	Moans to pain	2
	None	None	1
Motor response	Obeys commands	Spontaneous movements (obeys verbal command)	6
	Localizes pain	Withdraws to touch (localizes pain)	5
	Withdraws	Withdraws to pain	4
	Abnormal flexion to pain	Abnormal flexion to pain (decorticate posture)	3
	Abnormal extension to pain	Abnormal extension to pain (decerebrate posture)	2
	None	None	1
Best total score			**15**

DIAGNOSTIC STUDIES

- Serum electrolytes, calcium, magnesium, glucose
- Arterial or venous blood gas
- Liver function tests, ammonia
- Complete blood count with differential
- Blood urea nitrogen, creatinine
- Urine and serum toxicology screening

DIAGNOSTIC STUDIES

- Neuroimaging — Computed tomography (CT) is the best initial neuroimaging test . CT quickly detects pathology in need of immediate surgical intervention, including hydrocephalus, herniation, and mass lesions due to infection, neoplasia, hemorrhage, and edema.
- Magnetic resonance imaging (MRI) provides greater structural detail and is more sensitive for early evidence of encephalitis, infarction, diffuse axonal injury from head injury, petechial hemorrhages, cerebral venous thrombosis, and demyelination [MRI may also offer information regarding prognosis in patients with anoxic or traumatic coma

DIAGNOSTIC STUDIES

- Lumbar puncture — Urgent evaluation of cerebrospinal fluid (CSF) is required when there is suspected infection of the central nervous system. In a patient with altered level of consciousness, neuroimaging to exclude an intracranial mass lesion is required prior to LP in order to avoid precipitating transtentorial herniation. Coagulation test results should also be obtained beforehand if coagulopathy is suspected. Opening pressure should be measured and recorded
- CSF testing should include cell count and differential; glucose and protein; Gram stain and bacterial culture; viral testing, including an encephalitis panel herpes simplex virus (HSV) polymerase chain reaction (PCR); and other tests as clinically indicated. A serum glucose should be drawn as near in time as possible to the CSF, to ensure that the CSF:serum glucose ratio can be calculated accurately

DIAGNOSTIC STUDIES

- Electroencephalogram — Electroencephalography (EEG) should be performed in children with coma of unknown etiology. It is often the only means of recognizing nonconvulsive status epilepticus (NCSE)
- Periodic epileptiform discharges may occur in NCSE but also in underlying brain injury without seizures. Periodic lateralized epileptiform discharges suggest herpes encephalitis or infarction. Multifocal or generalized periodic discharges can also be seen with metabolic and infectious etiologies and are characteristic of subacute sclerosing panencephalitis.
- Nonepileptiform features of the EEG, such as slowing or asymmetry, are largely nonspecific findings, but can sometimes provide diagnostic or prognostic information. Continuous EEG can be used to assess and titrate the depth of sedation in patients placed under anesthesia for control of status epilepticus or increased ICP

CAUSES OF ALTERED CONSCIOUSNESS

Infections
Sepsis
Systemic infections; fever-related delirium
Metabolic derangements
Electrolyte disturbance (elevated or depressed): sodium, calcium, magnesium, phosphate
Endocrine disturbance (depressed or increased): thyroid, parathyroid, pancreas, pituitary, adrenal
Hypercarbia
Hyperglycemia and hypoglycemia
Hyperosmolar and hypoosmolar states
Hypoxemia
Inborn errors of metabolism: porphyria, Wilson disease, etc
Nutritional: Wernicke encephalopathy, vitamin B12 deficiency, possibly folate and niacin deficiencies
Brain disorders
CNS infections: encephalitis, meningitis, brain or epidural abscess
Epileptic seizures, especially nonconvulsive status epilepticus*
Head injury*
Hypertensive encephalopathy
Psychiatric disorders*

CAUSES OF ALTERED CONSCIOUSNESS

Systemic organ failure
Cardiac failure
Hematologic: thrombocytosis, hypereosinophilia, leukemic blast cell crisis, polycythemia
Liver failure: acute, chronic
Pulmonary disease, including hypercarbia and hypoxemia
Renal failure: acute, chronic
Physical disorders
Burns
Electrocution
Hyperthermia
Hypothermia
Trauma: with systemic inflammatory response syndrome, head injury*, fat embolism

DRUGS THAT MAY CAUSE ALTERED CONSCIOUSNESS

Drugs and toxins
Prescription medications (eg, opioids, sedative-hypnotics, antipsychotics, lithium, skeletal muscle relaxers, polypharmacy)
Nonprescription medications (eg, antihistamines)
Drugs of abuse (eg, ethanol, heroin, hallucinogens, nonmedicinal use of prescription medications)
Withdrawal states (eg, ethanol, benzodiazepines)
Medication side effects (eg, hyperammonemia from valproic acid, confusion from quinolones, serotonin syndrome)
Poisons:
Atypical alcohols (ethylene glycol, methanol)
Inhaled toxins (carbon monoxide, cyanide, hydrogen sulfide)
Plant-derived (eg, Jimson weed, Salvia)

SUMMARY

Evaluation
Vital signs and general and trauma examination
Neurologic examination and GCS
Fingerstick blood glucose Blood gas (arterial or venous) Screening laboratories (CBC, glucose, electrolytes, BUN, creatinine, blood and urine cultures, LFTs, urinalysis, urine drug screen)
Head CT scan: do urgently if focal neurologic signs, papilledema, or fever; consider rapid MRI instead if available
Lumbar puncture: do urgently after CT scan if fever, elevated WBC, meningismus; otherwise do according to level of suspicion for diagnosis or if cause remains obscure
Other laboratory tests: for metabolic conditions*, coagulation tests, carboxyhemoglobin, specific drug concentrations; do according to level of suspicion for diagnosis or if cause remains obscure
EEG: for possible nonconvulsive seizure, or if diagnosis remains obscure
Brain MRI with DWI, if cause remains obscure

SUMMARY

ABCs:
Intubate if GCS ≤8 or respiratory failure
Stabilize cervical spine
Supplement O_2
IV access
Blood pressure support as needed
Treat hypoglycemia identified on fingerstick. Dextrose 0.25 g/kg (2.5 mL/kg of 10% dextrose solution) after blood glucose drawn, before results back; do NOT delay pending results.
Treat definite seizures. Initial treatment with lorazepam (0.1 mg/kg, maximum single dose 4 mg). If seizures continue treat as for status epilepticus.
Empiric treatments:
For suspected infection:
Ceftriaxone 100 mg/kg (maximum single dose 2 grams) and vancomycin (age-specific dose)
Acyclovir (age-specific dose)
For suspected ingestion:
Naloxone 0.1 mg/kg IV in patients up to 20 kg or ≤5 years; maximum 2 mg IV (use if opioid toxidrome: miosis, respiratory depression, hypotonia)
For suspected increased ICP:
Mannitol 0.5 to 1 g/kg IV; or Hypertonic saline 3% 5 mL/kg *Also, elevate head and keep midline*
For suspected nonconvulsive status epilepticus:
Lorazepam (0.1 mg/kg, maximum single dose 4 mg). If suspicion of seizures continues, treat as for status epilepticus.
Fosphenytoin (10 to 20 PE equivalents/kg). If suspicion of seizures continues, treat as for status epilepticus.