

"He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all." – William Osler

430

MEDICINE
TEAMWORK

ALTERED LEVEL OF CONSCIOUSNESS

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Altered Level of Consciousness

Definitions

- **Consciousness** defined as being awake and aware of both one's self and one's surroundings, OR it is the human awareness of both internal and external stimuli
- **Level of consciousness:** is a measurement of a person's arousability and responsiveness to stimuli from the environment.
- Altered Consciousness covers a spectrum of states:
 - Consciousness
 - Lethargy → mild depression in level of consciousness and can be aroused with little difficulty.
 - Obtunded → More depressed level of consciousness and cannot be fully aroused. (slow response and sleepiness)
 - Stupor → Can not be aroused from a sleep like state.(only respond by grimacing or drawing away from painful stimuli)
 - Coma → More depressed level of consciousness and unable to make any purposeful response.

Pathophysiology

- **Reticular formation is known to play a role in alertness, wakefulness and arousal.**
- Ascending reticular activating system is a group of neural connections that receive sensory input and projects to cerebral cortex through the midbrain and thalamus from the reticular formation.

ALOC Clinical Features

- 4 pathophysiologic variables are helpful
 - Respiratory pattern
 - Pupillary light reflexes
 - Spontaneous eye movements
 - Motor responses

Respiratory Pattern

- Ventilation is governed by lower pons and medulla
- Modulated by forebrain cortical centers

Patterns from rostrocaudal involvement

- Cheyne Stokes respirations
 - Hyperpnea(deep and fast) alternating with apnea
- Central neurogenic hyperventilation
 - Regular and rapid respirations
 - Normal PaO₂ and low PaCO₂
 - Midbrain
 - Brain's attempt to reduce ICP
- Apneustic breathing
 - Deep, gasping inspiration with a pause at full inspiration followed by a brief, insufficient release
 - Signifies damage to Pons/medulla

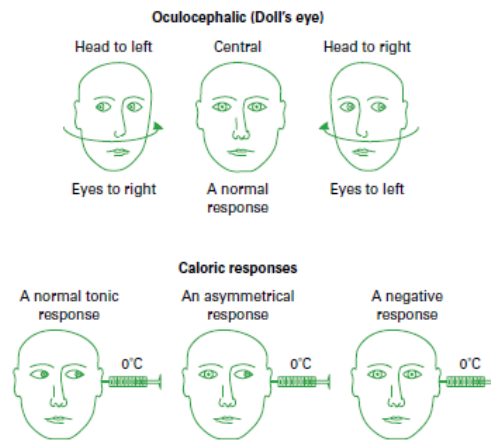
Pupillary Reflex

- Pupillary pathways near ARAS
- Pupillary pathways resistant to metabolic insult
- **Single most important physical finding to distinguish structural vs metabolic disease**
- **Bilateral enlarged and unreactive pupils** indicate massive CNS dysfunction (anoxia, barbiturate severe hypothermia and anticholinergics poisoning) .
- **Pinpoint pupils** indicate pontine hemorrhage, Opiates, organophosphate poisoning.
- Unilateral fixed dilated pupil indicate ipsilateral expanding mass and possible herniation.

- **Drugs also affect pupils**
 - Opiates – pinpoint pupils
 - Anticholinergics – large pupils

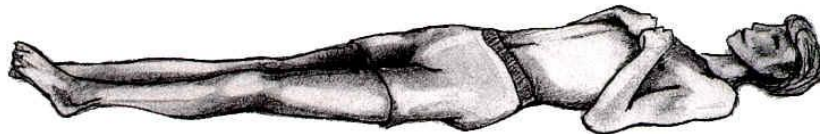
Eye Movements

- In light stage of coma, roving side-to-side movements occur
- Persistent deviation to one side may indicate focal seizure activity
- Structural brainstem lesions abolish conjugate eye movements
- **Oculocephalic reflex (“doll’s eyes”)**
 - Hold eyelids open and rotate head from side to side
 - Normal or positive – conjugate deviation of eyes away from direction of head movement
 - Contraindicated in c-spine injury
- **Oculovestibular reflex**
 - Elevate head of bed 30 degrees and inject 10-50ml of ice water into ear canal
 - Normal response is nystagmus with slow phase towards irrigated ear and fast beats away
 - Unconscious patient with intact brainstem eyes move towards stimulus and **remain tonically** deviated for a minute and slowly return to midline
 - Contraindicated if tympanic membrane not intact

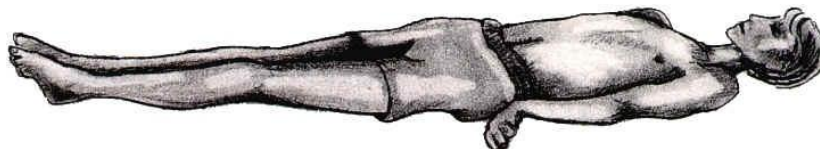


Motor Responses

- Assess muscle strength, tone and deep tendon reflexes for normality and symmetry
- Assess if patient can localize motor responses to determine level of brain lesion
- Decorticate posturing, with elbows, wrists and fingers flexed, and legs extended and rotated inward
 - Lesion in cortex or subcortical white matter



- Decerebrate posturing – rigid extension of arms and legs
 - Lesion at brainstem, usually pons



The Glasgow Coma Scale

- The Glasgow Coma Scale or GCS is a neurological scale that aims to give a reliable, objective way of recording the conscious state of a person for initial as well as subsequent assessment.

Eye opening (E)			
Nil	1		
Pain	2		
Verbal	3		
Spontaneous	4		
Motor response (M)		Verbal response (V)	
Nil	1	Nil	1
Abnormal extension	2	Incomprehensible	2
Abnormal flexion	3	Inappropriate	3
Weak flexion	4	Confused	4
Localising	5	Oriented fully	5
Obeys commands	6		

- MCQ Point:
 - Severe, with GCS ≤ 8
 - Moderate, GCS 9 - 12
 - Minor, GCS ≥ 13 .

Causes

- Etiologies
 - Altered Level of Consciousness (ALOC):
 - One of the most difficult diagnostic and management problems.
 - Requires quick action to avoid irreversible damage
 - Wide array of possible diagnoses
 - ALOC is a symptom of another problem, not a diagnosis itself
- A → Alcohol, Abuse (physical or substance)
- E → Encephalopathy, Electrolytes
- I → Insulin (hypoglycemia)
- O → Overdose, Oxygen deficiency
- U → Uremia
- T → Trauma, Temperature abnormality, Tumor
- I → Infection
- P → Poisoning, Psychiatric, Psychogenic
- S → Shock, Stroke, Seizures, Shunt
- Helpful mnemonic is **AEIOU TIPS**

Electrolytes

ALOC may be caused by:

- Abnormality in any cation (Na, Ca, Mg, Phosphorus)
- Metabolic acidosis or alkalosis
- You must restore electrolyte balance!
- Hypoglycemia – most common – You must check Glucose levels in all comatose patients**
- Hyperglycemia – especially new onset diabetes have ALOC due to hyperosmolarity
- DKA can lead to cerebral edema

Seizure

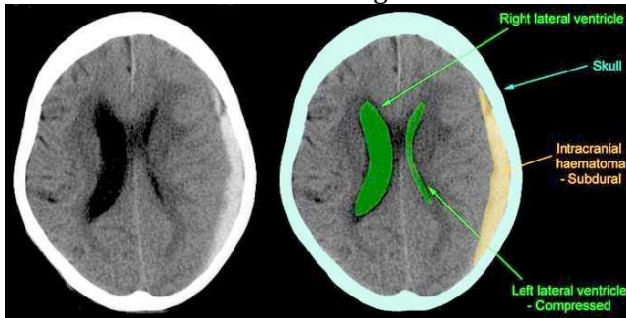
- All seizures except petit mal are followed by a post-ictal state
 - Altered level of consciousness could be due to the patient being in a post-ictal state
- Measure drug levels for patients on anticonvulsants.
- If patient is having non-convulsive seizure, check for seizure signs (Incontinence, tongue biting, injury)
- Comatose patients may have non-convulsive seizures needing an EEG to diagnose.

Trauma

- **Epidural Hematoma**
 - Lens shaped
 - Caused by arterial rupture
 - Skull fracture present in 85% of cases



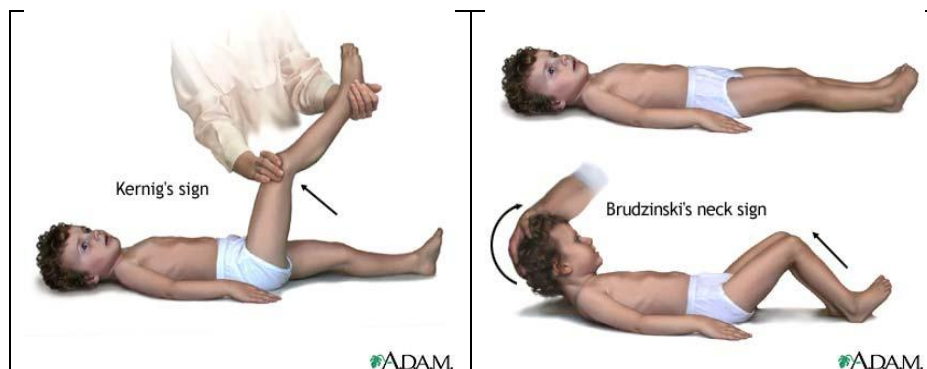
- **Subdural hematoma**
 - Crescent shaped
 - Caused by tearing of bridging veins through dura and arachnoid
 - Skull fracture present in 30% of cases
 - Retinal Hemorrhage in 75% of cases



- Cerebral Contusion
 - Can lead to increased ICP
- **You must do a CT for all unconscious patients**

Meningitis

- Patient comes in with altered level on consciousness and Fever → Think about CNS infections → Most importantly Meningitis
- Bacterial
 - Most common infection severe enough to cause profound ALOC
- Non-bacterial
 - Slower onset of symptoms



Infection

- Brain Abscess

- Chronic sinusitis, chronic otitis, dental infection, endocarditis or uncorrected cyanotic congenital heart disease can increase risk



- Encephalitis
 - Inflammation of the brain parenchyma usually due to **viral infection**
 - **HSV (Herpes Simplex Virus) – most common devastating cause**
 - Death or permanent neurologic damage in 70% of cases
 - Affects temporal lobes causing seizures, parenchymal swelling and uncal herniation
 - Diagnosis via → Lumber puncture and PCR.

Stroke

- Hemorrhagic is usually due to aneurysm
 - Severe headache
- AVM (Arteriovenous Malformation) or cavernous hemangioma
 - Low flow and less acute symptoms



- Thrombosis or Embolic Stroke
 - Occlusion of anterior, middle or posterior cerebral artery will **NOT** cause coma
 - Infarcts eventually lead to increased ICP
 - Cerebellar infarcts rarely have coma
 - Basilar Artery infarcts cause rapid coma due to brainstem damage
- Stroke has to be large or complicated in order for it to cause altered mental status

Hypoxia

- Neurons extremely sensitive to hypoxia and cease function within seconds of hypoxia
- Permanent CNS dysfunction can occur within 4-5 minutes of total anoxia at body temperature
- Hypercarbia can also cause neurologic depression and coma

Temperature

- Hypothermia
 - Each drop by 1 degree celcius causes a 6% drop in cerebral blood flow
- Hyperthermia
 - Headache, vomiting, seizure, obtundation, or coma result especially above 41 degrees C

Investigations

- CT brain
- MRI brain
- Serum
 - Glucose → Patient may be hypoglycemic
 - Electrolytes
 - LFTs → Patient might have hepatic encephalopathy
 - BUN
 - PT, PTT → Patient might have a hypercoagulable state
 - Calcium → High levels may cause Cardiac arrest
 - Ammonia
 - Osmolality
 - Ketones
 - Alcohol
 - Drug concentrations
 - ABGs

Treatment

- Basic
 - Establish an airway, maintain as indicated, suction as needed; assist ventilations as indicated.
 - Administer high concentration oxygen.
 - Transport the patient in the coma/recovery position (if trauma is suspected, transport supine with cervical collar and backboard).
 - Intermediate
 - If the patient is in respiratory arrest, perform advanced airway management.
 - Secure IV access.
 - Obtain blood specimen for glucose determination at the hospital if the receiving hospital desires it.
 - Perform capillary blood glucose determination.
 - If patient's blood glucose level is <80 mg/dl, administer dextrose 50% 25 gm
 - Unless patient responded to dextrose administration, contact medical direction for an order to administer 2 mg of naloxone intravenously. (Opiate antidote)
 - Administer thiamine 100 mg IV if dextrose is to be administered. (If alcohol is poisoning present)
 - If IV access cannot be secured and the patient's blood glucose level is <80 mg/dl, administer 1 mg glucagon IM.
 - Then treat underlying cause.
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