Syncope and Coma

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Syncope

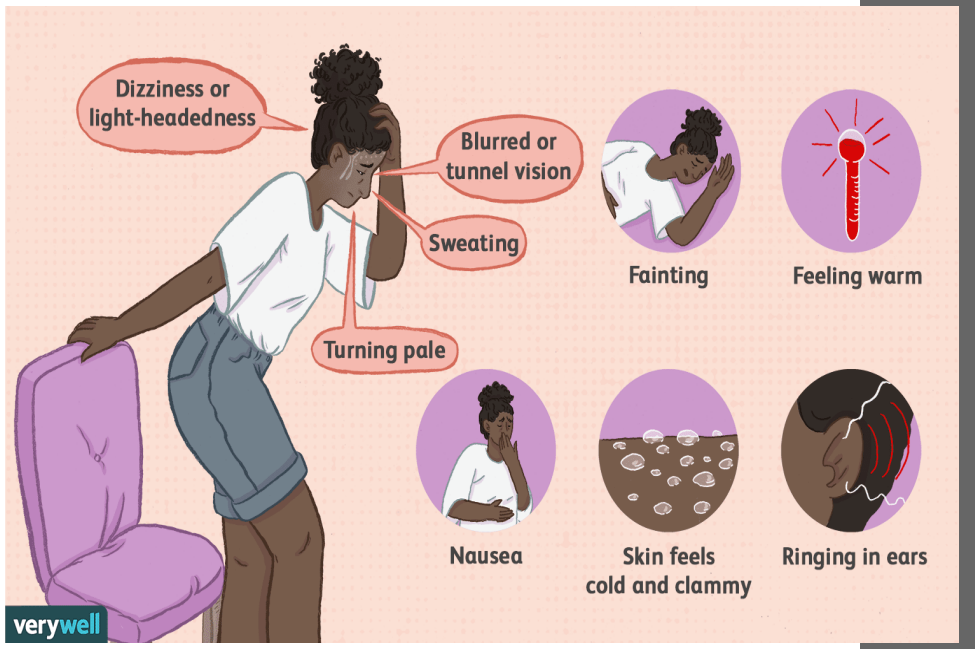
- Definition.
- Prodrome.
- Causes.
- Approach.
- Differentials.

Syncope

- Syncope refers to a non-traumatic transient loss of consciousness secondary to acute decrease in cerebral blood flow.
- Syncopal episodes have a rapid onset and rarely last more than a minute or two.
- Syncope is characterized by having a spontaneous recovery (self-limiting).
- Prognosis is usually good, unless the cause is cardiac.

Prodrome

- It is a period of symptoms that lasts for a few seconds, right before losing consciousness.
- It is also called presyncope.
- These symptoms include:
  - Pallor.
  - Sweating.
  - Lightheadedness.
  - Nausea.
  - Palpitations.
  - Blurred vision.
- Associated with reflex and orthostatic hypotension syncope only.
  <u>Never with cardiac syncope.</u>



Causes

- Neurally-mediated (reflex) syncope:
  - Vasovagal syncope.
  - Situational syncope.
- Orthostatic hypotension (OH) syncope.
- Cardiac:
  - Arrhythmias.
  - Structural disease.
  - Great vessel disease.
  - Systemic.

- Both orthostatic hypotension and neurally-mediated syncope can only occur in the sitting or standing position, but never in the recumbent position.
- These upright positions pose some form of physiological stress on the body that leads to the pooling of blood in the lower limbs. This causes a decrease in the venous return to the blood and subsequently a decrease in the cardiac output.
- In normal situations, the hemodynamic changes are sensed by baroreceptors in the carotid sinus and aortic arch that results in a reflex response causing an increase in the sympathetic outflow and a decrease in the vagal nerve activity. The reflex increases peripheral resistance and venous return to the heart and thus limits the fall in cardiac output.

- If this reflex response fails, cerebral hypoperfusion and syncope occur.
- Neurally-mediated syncope is characterized by a transient change in the reflexes responsible for maintaining cardiovascular homeostasis. Episodic vasodilation (or loss of vasoconstrictor tone), decreased cardiac output, and bradycardia occur in varying combinations, resulting in temporary failure of blood pressure control.
- Whereas in orthostatic hypotension syncope, patients have chronically impaired reflexes due to some sort of autonomic dysfunction.

Neurally-mediated syncope

- The most common cause of syncope.
- Vasovagal syncope is triggered by emotional distress, pain and orthostatic stress (ex: Sight of blood).
- Situational syncope is triggered by certain situations and are classified according to the involved organ system:
  - Urogenital: Micturition, prostate massage…
  - Gastrointestinal: Defecation, swallowing…
  - Pulmonary: Coughing, sneezing…
  - Carotid: Carotid sinus hypersensitivity, carotid sinus massage…
- Occurs when the patient is sitting or standing up, never in the recumbent position.
- Treatment involves avoiding triggers mainly.

Orthostatic hypotension syncope

- A drop in systolic blood pressure of more than 20mmHg or a drop in diastolic blood pressure of more than 10mmHg that occurs on standing for more than 3 minutes.
- Orthostatic hypotension has 4 causes:
  - Volume depletion (the most common cause of orthostatic hypotension is dehydration).
  - Autonomic insufficiency, primary (ex: Parkinson's disease)or secondary (ex: Diabetes).
  - Drug-induced (ex: antihypertensives and beta-blockers).
- Occurs when the patient is sitting or standing up, never in the recumbent position.
- Treated according to the cause:
  - Drug-induced: Must be stopped and replaced.
  - Volume depletion: Fluids.
  - Avoid standing up for long periods of time, regardless of the cause.

Cardiac

- The most serious cause of syncope.
- Unlike the latter, cardiac syncope is never associated with a prodrome. The patient suddenly falls, with no triggers.
- If syncope occurs with exertion, assess for potentially life-threatening causes such as hypertrophic cardiomyopathy or aortic stenosis.
- Only type of syncope that can happen when the patient is in a recumbent position.
- Treatment of the underlying heart condition.

Cardiac

- Arrhythmia:
  - They are usually either due to a bradyarrhythmia or a tachyarrhythmia.
  - The most common cause of cardiac syncope.
- Structural disease:
  - Includes: aortic stenosis, cardiac tamponade, pacemaker malfunction, hypertrophic cardiomyopathy and ischemia (MI).
  - They all lead to some sort of obstruction in blood flow.
- Great vessel disease, most notably a pulmonary embolism.
- Systemic:
  - Ruptured aortic aneurysm.
  - Severe hemorrhage.

Approach to syncope

- Our first concern is to rule out any life-threatening causes of syncope, such as: severe hemorrhage, an MI, severe dehydration, etc.
- However, our main concern is to distinguish if the syncope is cardiac or non-cardiac; since cardiac syncope has the poorest prognosis.
- The approach involves:
  1. ABCs (are almost always normal in neurally-mediated and orthostatic hypotension syncope, may be abnormal in cardiac syncope).
  2. History (patient and witness).
  3. Physical exam.
  4. Labs and investigations.

History

- Six key elements need to be determined:

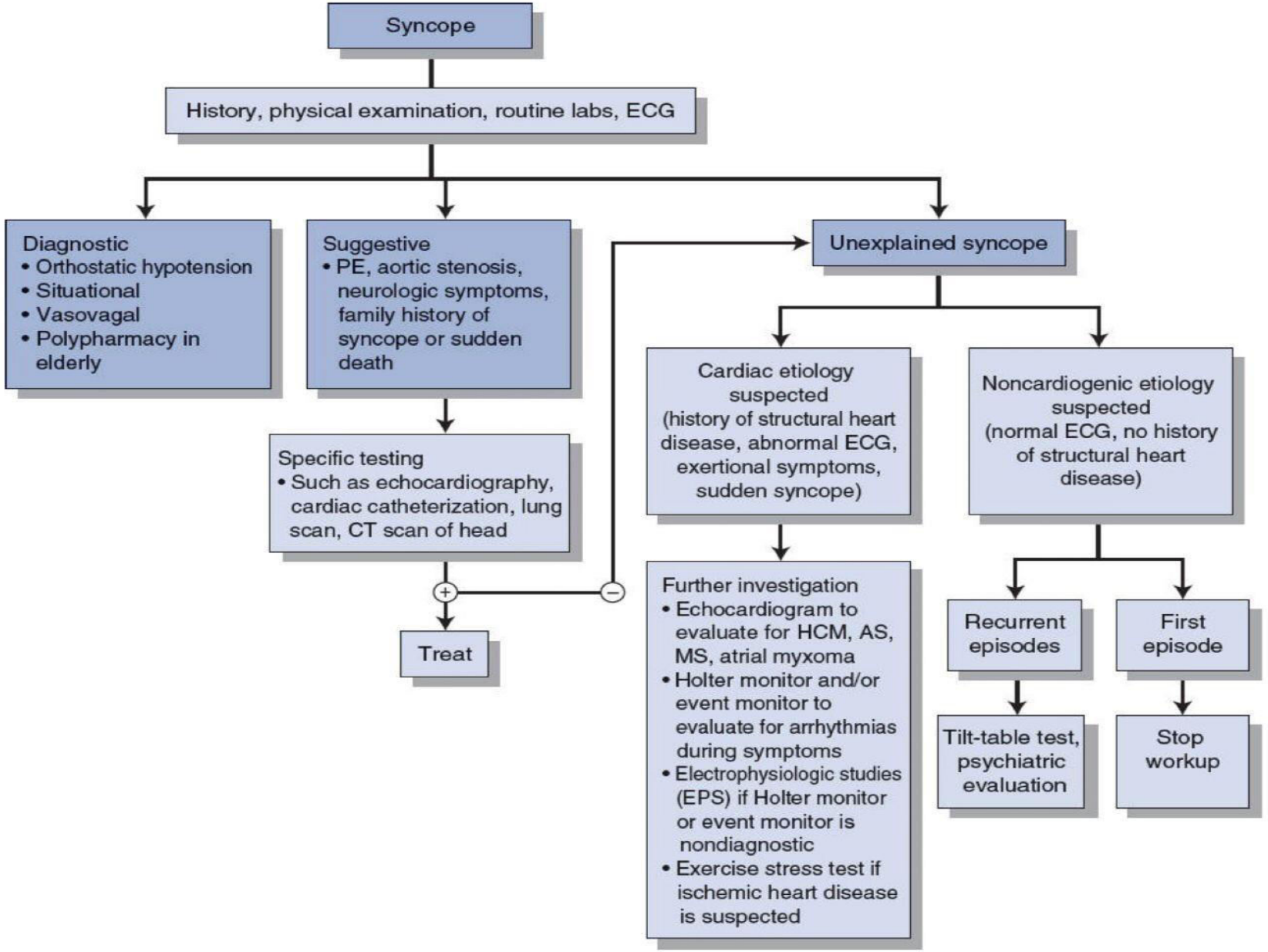
1. Events before the syncopal episode: the presence or absence of a prodrome, if there were any triggers, the position the patient was in before the episode or if there was a change in position.
2. Events during the syncopal episode: how long it lasted and if there were any convulsions (indicative of seizures and not syncope).
3. Events after the syncopal episodes: Postictal confusion, drowsiness lasting more than a few minutes is indicative of seizures.
4. If this is a first time or recurrent issue; if it's recurrent, find out the frequency and usual duration of the episodes.
5. Drug history.
6. Witness history if available.

Physical exam

- Full neurological exam.
- Red flags after the syncope include:
  - Abnormal vitals, such as prolonged hypotension or a drop in blood pressure from sitting down to standing up (OH).
  - Abnormal cardiac, respiratory or neurological exam, for example; a cardiac murmur heard on auscultation may indicate an underlying structural heart disease.
  - Check for any injuries that may have happened if the patient fell during their syncopal episode.

Investigations

- **An ECG should be in all patients to look for evidence of an arrhythmia (or possibly ischemia).**
  - Sometimes, an ECG doesn't capture an intermittent rhythm disturbance and thus a Holter monitor (24h) can be used.
- An echocardiogram is very helpful if a structural heart disease is suspected.
- Other tests that are helpful include: exercise testing, carotid sinus massage, an EEG and tilt-table tests (controversial nowadays).


Syncope
History, physical examination, routine labs, ECG
Diagnostic
• Orthostatic hypotension
• Situational
• Vasovagal
• Polypharmacy in elderly
Suggestive
• PE, aortic stenosis, neurologic symptoms, family history of syncope or sudden death
Specific testing
• Such as echocardiography, cardiac catheterization, lung scan, CT scan of head
+
−
Treat
Unexplained syncope
Cardiac etiology suspected (history of structural heart disease, abnormal ECG, exertional symptoms, sudden syncope)
Noncardiogenic etiology suspected (normal ECG, no history of structural heart disease)
Further investigation
• Echocardiogram to evaluate for HCM, AS, MS, atrial myxoma
• Holter monitor and/or event monitor to evaluate for arrhythmias during symptoms
• Electrophysiologic studies (EPS) if Holter monitor or event monitor is nondiagnostic
• Exercise stress test if ischemic heart disease is suspected
Recurrent episodes
First episode
Tilt-table test, psychiatric evaluation
Stop workup

Syncope differentials

- Psychiatric conditions, such as anxiety disorders.
- Metabolic causes, most notably hypoglycemia.
- Drug or alcohol abuse.
- Cerebrovascular accidents (strokes). On rare occasions, a TIA in the vertebrobasilar circulation may lead to syncope.
- Seizures.
- Head trauma.

Seizure	Syncope
Aura, focal symptoms, olfactory hallucinations, automatism (ex. lip smacking) before	Prodromal presyncope, palpitations, diaphoresis before
Myoclonic jerks *before* LOC	Myoclonic jerks *after* LOC
Usually between 1-2 minutes	Brief, generally <1 minute
EMS vitals: usually BP & HR generally elevated (exception: rare types of temporal lobe seizure can cause bradycardia)	EMS vitals: could have low BP & HR
Post-ictal confusion	Rapid recovery
More often horizontal deviation or flickering of eyelids, blank stare	Vertical deviation (rolling back), can also see flickering of eyelids
Eyes open	Usually eyes closed or rolled back
Lateral tongue biting	

Coma

Coma

Definition:

- Coma is a state in which a patient is unresponsive to environmental stimuli and unable to communicate in any manner.
- Coma is associated with extensive structural or physiologic damage to both cerebral hemispheres or to the ascending RAS (reticular activating system) in the diencephalons, mesencephalon, or pons.

The state of consciousness or alertness depends on:

- 1- An intact reticular activating system (R.A.S.): This is a collection of nuclei present in the brain stem, hypothalamus & thalamus. It receives impulses from the pathways carrying sensations from the outside world & transmits them through ascending fibres to the cerebral cortex. Its function is the activation of the cerebral cortex
- 2- An intact cerebral cortex

Etiology

1- Intracranial causes:

"Local causes in the brain"

***Coma with lateralising signs

- Trauma: head injury with cerebral concussion
- Tumor: brain tumor e.g. meningioma
- Vascular: Hemorrhage (Cerebral & subarachnoid), thrombosis, embolism, Hypertensive encephalopathy.
- Inflammatory: meningitis, encephalitis, brain abscess
- Epilepsy

The signs of lateralization are:

- unequal pupils
- deviation of the eyes to one side
- facial asymmetry
- tilting of the head to one side:
- unilateral hypo- or hypertonia.
- asymmetric deep reflexes.
- unilateral +ve Babinski
- unilateral focal or Jacksonian fits.

2- Extracranial causes:

"General causes with 2ry effects on the brain"

***Coma without lateralizing signs

- Toxic:

✓Belladona (atropine) poisoning,

✓Aspirin (salicylate) poisoning,

✓Alcohol intoxication,

✓Barbiturate poisoning,

✓Carbon monoxide poisoning,

✓Morphine (opiate) poisoning,

- Hypoxic: Pulmonary disease.
- Cardiac causes: cardiac arrest, MI, arrhythmia, Hypotensive drugs.

- Metabolic: Hypo & hyperglycemia (D.M.), Hypo & hyperthermia (heat stroke), Respiratory failure, Renal failure, Hepatic failure
- Endocrine:

✓Pituitary: Hypopituitarism

✓Thyroid: Hypothyroidism & thyrotoxic crisis.

✓Adrenal: Addisonian crisis

- Fever (febrile coma) // Infections:

✓Meningitis

✓Malaria, especially the Cerebral type

✓Status typhosus.

✓Septicemia

CAUSES OF FEBRILE COMA:

- 1. Infective: encephalitis, meningitis & other hyperpyrexias.
- 2. Vascular: pontine hemorrhage, subarachnoid hemorrhage.
- 3. Metabolic: diabetic ketoacidosis, hepatic cirrhosis.
- 4. Endocrinal: thyrotoxic & Addissonian crisis.
- 5. Toxic: Belladonna & salicylate poisoning.
- 6. Sun stroke & heat stroke.
- 7. Coma with 2ry infection due to hypostatis pneumonia, U.T, infection or bed sores.

GLASGOW COMA SCALE (GCS)

What is Glasgow coma scale?

- It is a neurological scale used to reliably measure a person's level of consciousness after a brain injury.
- Glasgow coma scale assessment:
- Based on the persons ability to perform eye movements, speak, move their body.
- The highest score one can get on GCS is 15 which means that there is no brain damage.
- The lowest score one can get is 3.

3 brain levels that are assessed by GCS:

- 1. Cortex: where many of the higher-level functions take place (e.g. decision-making and language).
- 2. Subcortical: where we process more primitive functions.
- 3. Brain stem: where basic (almost reflex like) responses in our body.
- ** if the damage reached the brain stem and altered its function severe brain damage.

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

VEGETATIVE STATE

- A wakeful unconscious state (individual is unaware of self and environment), yet able to breath spontaneously (preserved brainstem function), with a stable circulation and cycles of eye closure and opening resembling sleep and waking.
- Also called unresponsiveness wakefulness syndrome it is not coma.
- This state may be permanent (after 4 weeks) the patient then is classified as in a persistent vegetative state.
- Someone in a vegetative state still has a functioning brain stem which means:
  - some form of consciousness may exist.
  - breathing unaided is usually possible.
  - there is slim chance of recovery because the brain stem cores function may be unaffected.

CAUSES

1. Disruption of blood flow to brain.
2. Severe head injury.
3. Drug overdose.
4. Stroke.

DIAGNOSIS

- No evidence of awareness of self or environment.
- no purposeful and sustained response to stimuli.
- Presence of sleep-awake cycles.
- Presence of brain stem function and some reflexes.
- General incontinence.

- If a person in a vegetative state for a long time, it might be considered as:

1. A continuing (persistent) vegetative state: when its been for more than 4 weeks. Persistent vegetative state: it is a disorder of consciousness in which patients with severe brain damage are in a state of partial arousal rather than true awareness.

2. A permanent vegetative state: when its been for more than 6 months if caused by a non-traumatic brain injury, or more than 12 months of caused by a traumatic brain injury.
   If a person is diagnosed with it, recovery is extremely unlikely but not impossible.

Brain death

- Irreversible cessation of cerebral and brainstem function
- It is a complete loss of brain function (including involuntary activity necessary to sustain life).
- Irreversible brain damage may have occurred with permanent destruction of brainstem function (total lack of vital signs “heartbeat and respiration”).
- Patients classified as brain-dead can have their organs surgically removed for organ donation.

Diagnosis

Clinical Presentation

+

Neurologic Examination

+

Apnea Test

Brain Death

*Brain death should be confirmed by two different attending physicians

Clinical presentation

- Clinical or imaging evidence of acne CNS catastrophe.
- Exclusion of other conditions:
  - Electrolyte imbalance.
  - Acid-base.
  - Endocrine.
- No drug intoxication or poisoning
- Circulatory (shock).
- Core temperature normal (no hypothermia).
- Systolic blood pressure >100 mmHg.

Neurological examination and apnea test

- Coma.
- Absent high level motor response (no withdrawal to pain).
- Absent pupillary light and corneal reflex.
- Absent oculovestibular reflex.
- Absent cranial nerves and primitive reflexes (gag, jaw jerk, sucking, rooting).
- Apnea test:
  - Preformed after other criteria have met.
  - Preoxygenate and disconnect from the ventilator.
  - Positive test:

No respiratory movement for 8-10 mins.

ABG: $PaCO_2$ >60 mmHg or >20mmHg from baseline.

Locked in syndrome

- Mimics coma.
- Patient is conscious but paralyzed.
- Patients are fully aware of their surroundings and capable of feeling pain.
- Sparing of respiratory muscles and eye movement.
- Caused by ventral pons infarct/hemorrhage.
- Usually due to basilar artery stroke.
- Diagnosis : voluntary vertical eye movements.

Causes of coma

- Structural intracranial disorder:
  - Supratentorial bilateral.
  - Unilateral large lesion with trans-tentorial herniation.
  - Infratentorial.
- Toxic and metabolic disorders.
- Ictal or post ictal state.
- Psychogenic unresponsiveness.

TABLE 5.1 Causes of Stupor and Coma

1. Structural intracranial disorders
   a. Trauma
      (1) Epidural, subdural, intracerebral, or subarachnoid hemorrhage
      (2) Diffuse axonal injury
      (3) Concussion
   b. Cerebrovascular events
      (1) Intracerebral or subarachnoid hemorrhage
      (2) Hemispheric or brain stem infarction
      (3) Dural sinus thrombosis
      (4) Posterior reversible encephalopathy syndrome
   c. Infection
      (1) Meningitis
      (2) Encephalitis
      (3) Abscess
   d. Inflammatory disorders
      (1) Autoimmune vasculitis or cerebritis
      (2) Demyelinating disease (e.g., multiple sclerosis)
   e. Neoplasm
   f. Hydrocephalus
2. Toxic or metabolic disorders
   a. Global hypoxia-ischemia
   b. Electrolyte or acid-base disorders
      (1) pH disturbances
      (2) Hypernatremia or hyponatremia
      (3) Hyperglycemia or hypoglycemia
      (4) Hypercalcemia or hypocalcemia
   c. Drug intoxication or withdrawal
   d. Temperature disorder (hyperthermia or hypothermia)
   e. Organ system dysfunction
      (1) Liver (hepatic encephalopathy)
      (2) Kidney (uremia)
      (3) Thyroid (myxedema, thyrotoxicosis)
      (4) Adrenal (hyperadrenalism or hypoadrenalism)
      (5) Multisystem organ failure
   f. Seizure and postictal states
   g. Thiamine or vitamin B_{12} deficiency
3. Psychogenic unresponsiveness

Coma due to structural lesions

- **Supratentorial:**
- May either cause widespread bilateral disease, increased intracranial pressure, or herniation.
- Starts with focal cerebral dysfunction.
- Rostral to caudal progression.
- Signs usually localize a single area (ex diencephalon).
- Asymmetrical motor signs.

Infratentorial:

- Lesions involve RAS , usually with associated brainstem signs.
- Sudden onset of coma.
- Cranial nerve palsies are usual.
- Involvement of brainstem nuclei/ tract with focal finding.
- Bizarre respiratory patterns at onset.

Coma due to hypoxic and metabolic causes

- ***Metabolic:***
- Patient generally have signs of patchy, diffuse and symmetric neurologic involvement that can't be explained by loss of function at any single level or in a sequential manner, although focal or lateralized deficits may occur in hypoglycemia. Moreover, pupillary reactivity is usually preserved, while other brainstem functions are often grossly impaired.

Cerebral ischemia:

In patient with coma due to cerebral ischemia and hypoxia, the absence of pupillary light reflex at the time of initial examination indicate that there is little chance of regaining independence; by contrast preserved pupillary light response, the development of spontaneous eye movement and extensor, flexor, or withdrawal response to pain at this early stage imply a relatively good prognosis.

Coma due to meningeal irritation

- Comatose patients with meningitis, encephalitis or subarachnoid hemorrhage may also exhibit little in the way of focal neurologic signs, however, and clinical evidence of meningeal irritation is sometimes very subtle in comatose patients.
- Examination of CSF in such patient is essential to establish correct diagnosis.

Approach to an unconscious patient

- History.
- Physical examination.

History

- History is taken whenever possible from relatives, friends and reliable attendees:
- You must ask about:
  - Patient profile (name, age, occupation).
  - Onset (sudden or gradual).

Recent complaints

- Headache.
- Weight change.
- Fever.
- Depression or suicidal ideations.
- Focal weakness.
- Recent trauma.

Previous medical illnesses or family history

- DM.
- Hypertension.
- Chronic lung disease.
- Renal failure.
- Thyroid disease.
- Heart disease.
- Genetic diseases.

Drug history

- Insulin.
- Thyroxine.
- Recreational drugs.

Social history

- Recent travel: We must look for any recent trips to any endemic regions that may cause:
  - Malaria (Africa).
  - Japanese Encephalitis (Japan).
- Sexual history: Any activity that could lead to STDs.

Physical examination

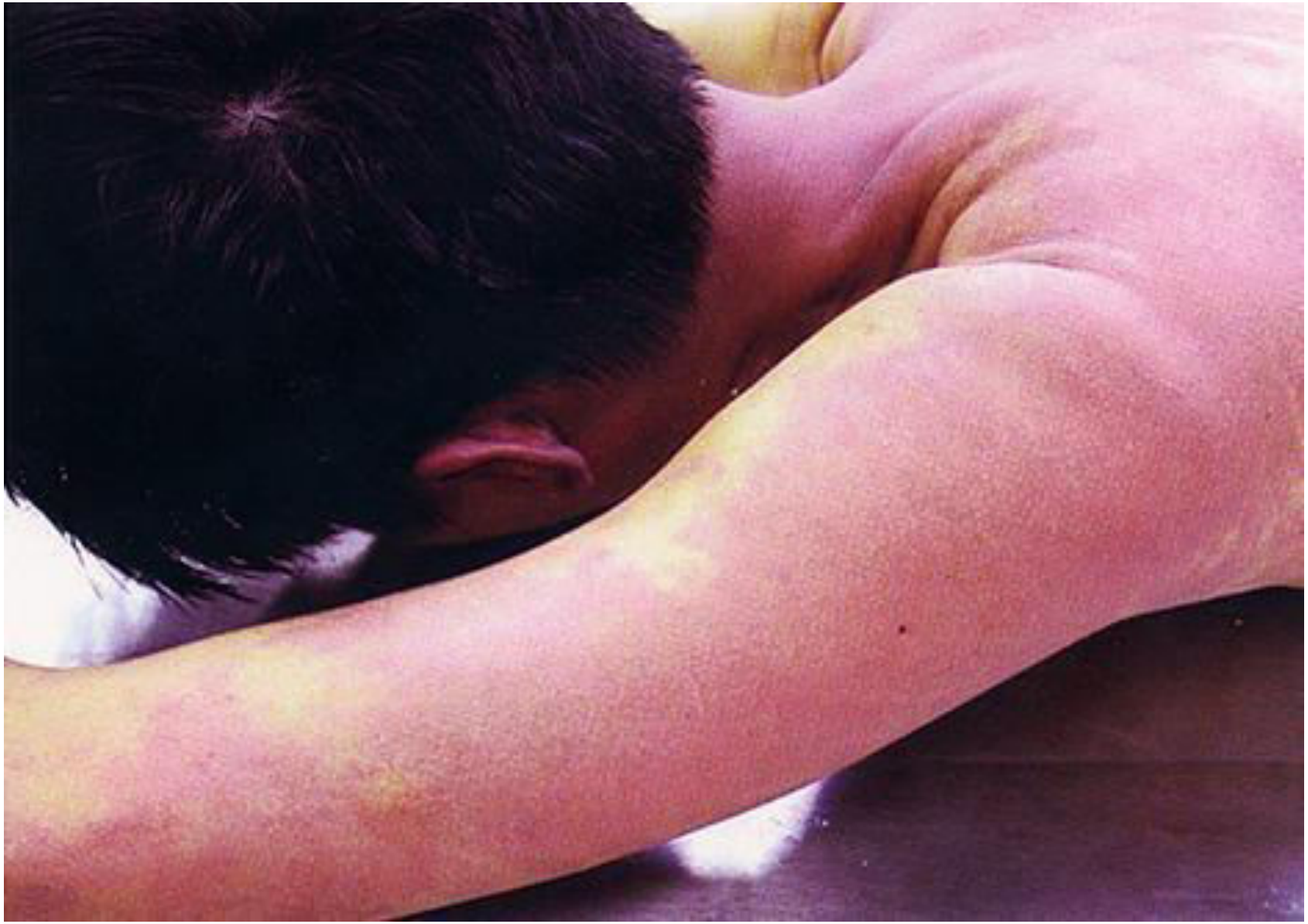
- General examination.
- Neurological examination.

General examination

1. Temperature:
   - Hyperthermia: may lead to a febrile coma.
   - Hypothermia: may be caused by:
     - Hypothyroidism.
     - Barbiturate use.
     - Alcohol use.
2. Pulse:
   - Bradycardia: may be caused by brain tumors, opiates and myxedema.
   - Tachycardia: may be caused by hyperthyroidism or uremia.

3. Blood pressure:
   - High: may cause hypertensive encephalopathy.
   - Low: may be caused by an Addisonian crisis or alcohol use.
4. Respiration:
   - Slow: seen in morphine toxicity.
   - Rapid and deep (kussmaul): seen in diabetic or uremic acidosis.
   - Hyperpnea regular alternating with apnea: seen in lesions affecting both cerebral hemispheres.
   - Central neurogenic hyperventilation: Like kussmaul but there's a lesion at the junction between the midbrain and pons.
   - Apneustic: prolonged pause after full inspiration.
   - Ataxic: phases of deep and shallow breathing alternate irregular: seen in medullary lesions.

5. Breath odor:
   - Fruity/ acetone odor: seen in diabetic ketoacidosis (DKA).
   - Fetor hepaticus: seen in hepatic comas.
   - Uriniferous odor: seen in uremic comas.
   - Alcohol: seen in alcohol intoxication.
6. Skin:
   - Injuries: seen in trauma.
   - Dry skin: seen in DKA or atropine poisoning.
   - Moist skin: seen in hypoglycemic comas.
   - Cherry-red skin: seen in CO poisoning.
   - Needle/ track marks: seen in drug abuse.
   - Rashes: seen in endocarditis or meningitis.



Neurological examination:

- The goal of a neurological examination in a comatose patient is to determine if the coma is induced by a structural lesion or from a metabolic derangement, or possibly from both.

- Two findings on exam strongly point to a structural lesion:
  1. consistent asymmetry between right and left sided responses.
  2. abnormal reflexes that point to specific areas within the brain stem.

Neurological examination:

- **Mental status** is evaluated by observing the patient's response to visual, auditory and noxious (i.e., painful) stimuli.

The three main maneuvers to produce a noxious stimulus in a comatose

patient are:

1. press very hard with your thumb under the bony superior roof of the orbital cavity
2. squeeze the patient's nipple very hard
3. press a pen hard on one of the patient's fingernails.

Neurological examination:

Abnormal posturing is a common outcome of severe brain injury. It refers to involuntary and abnormal positioning of the body due to preserved motor reflexes.

* **Decorticate posturing:** consists of adduction of the upper

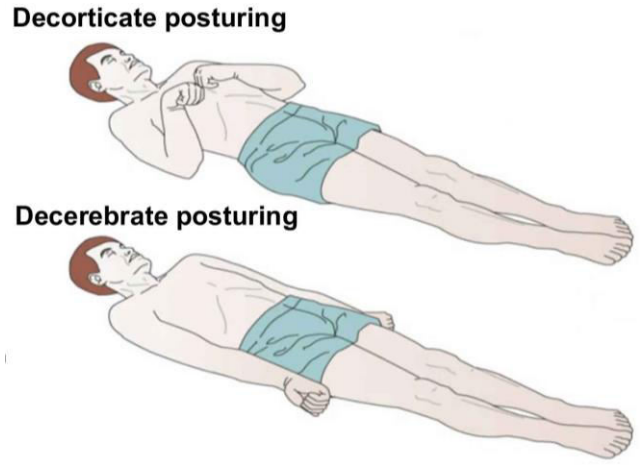
arms, flexion of the lower arms, wrists and fingers.

- The lower extremities extend in decorticate posturing.

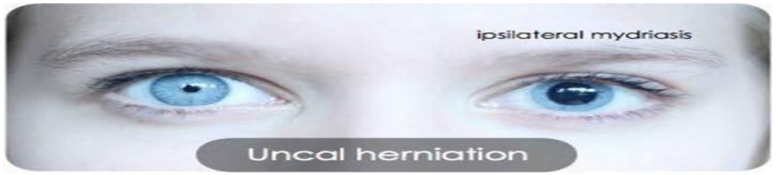
* **Decerebrate posturing:** consists of adduction of the upper

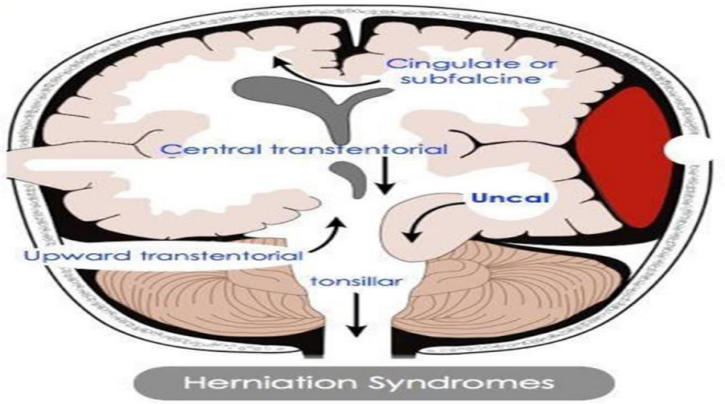
arms, extension and pronation of the lower arms, along with extension of the lower extremities.

**** These postures are response typically to noxious stimuli**



Neurological examination:





- Pupillary signs:
  - Dilated, unreactive to light:
    - Unilateral: 3rd nerve compression (uncal herniation)
    - Bilateral: e.g. Atropine poisoning
  - Constricted:
    - Horner's syndrome however , alone, this syndrome does not cause coma
    - Bilateral reactive to light : metabolic coma
    - Unreactive to light: pontine hemorrhage , morphine poisoning (pin-point pupil)

Neurological examination:

- **Extra ocular muscle examination :**

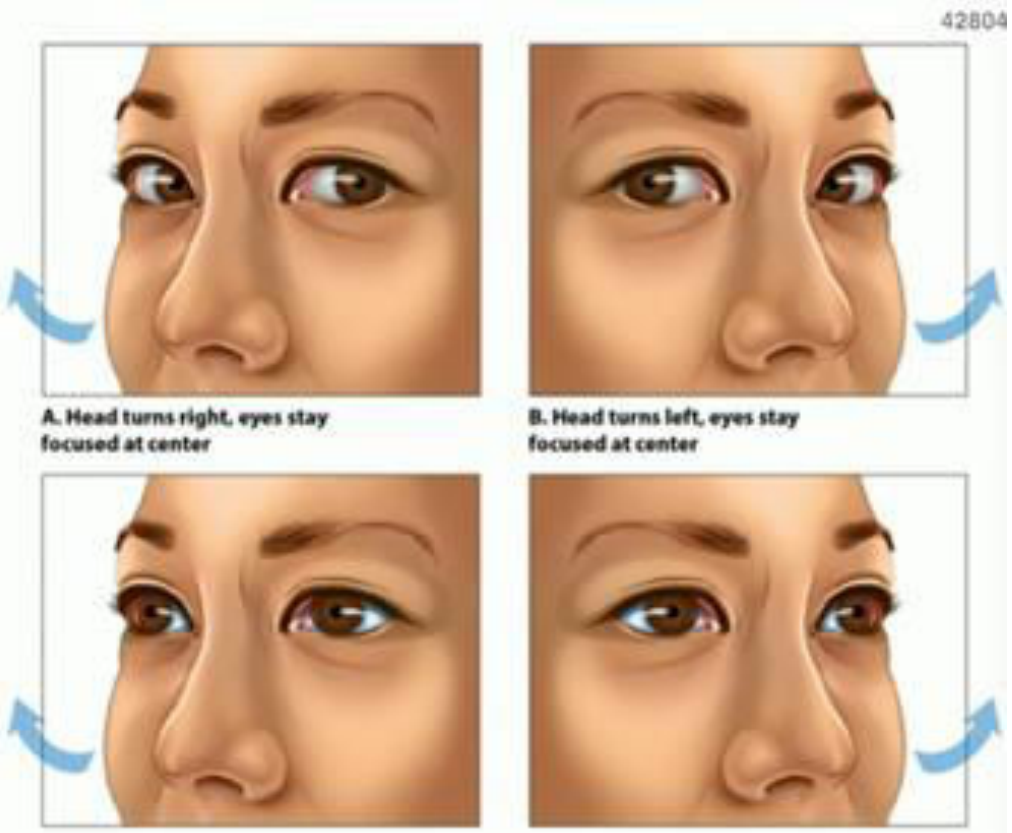
1-**The doll's eyes reflex (oculocephalic test):** it is elicited by turning the head of the unconscious patient while observing the eyes. The eyes will normally move as if the patient is fixating on a stationary object. If there is a negative doll's eyes reflex then the eyes remain stationary with respect to the head.

2- **Caloric test:** Is a test of the vestibule-ocular reflex that involves irrigating cold or warm water or ear into the external auditory canal

- If the water is warm …causing an increased rate of firing in the vestibular afferent nerve. This situation mimics a head turn to the ipsilateral side. Both eyes will turn toward the contralateral ear, with horizontal nystagmus to the ipsilateral ear
- If the water is cold … decreasing the rate of vestibular afferent firing. This situation mimics a head turn to the contralateral side. The eyes then turn toward the ipsilateral ear, with horizontal nystagmus to the contralateral ear

"Doll's eye test"

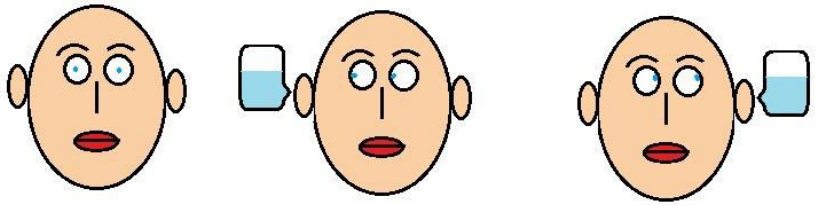
Oculocephalic reflex present



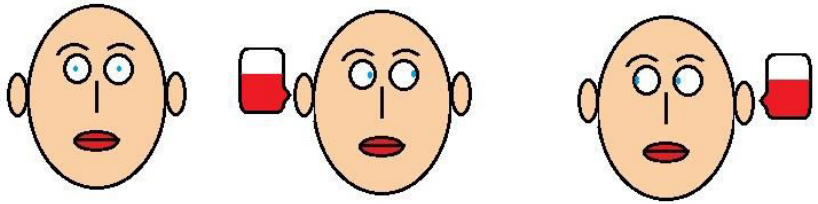
Oculocephalic reflex absent

Vestibulo-ocular Reflex

Cold water:



Warm water:



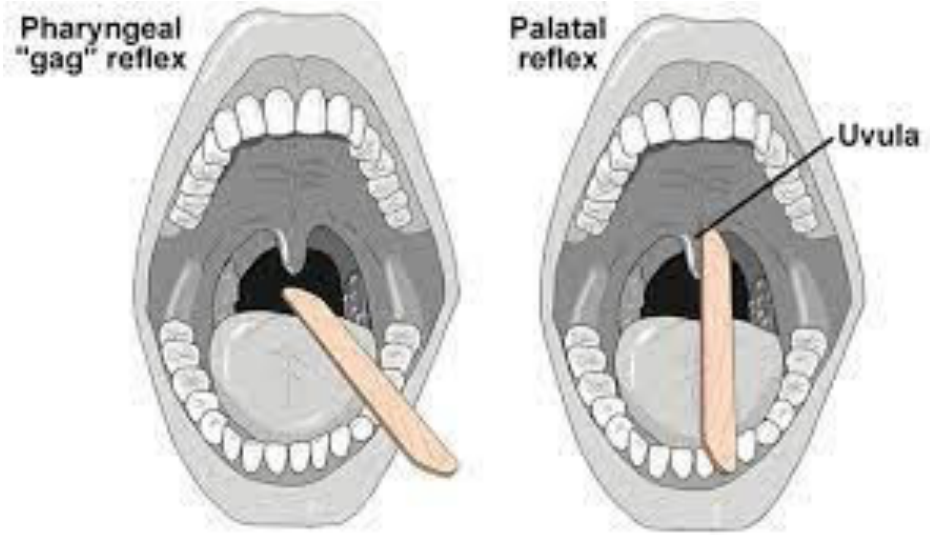
- In comatose patients with cerebral damage, the fast phase of nystagmus will be absent as this is controlled by the cerebrum.

- If both phases are absent, this suggests that the patient's brainstem reflexes are also damaged and this carries a very poor prognosis

Neurological examination:

- Cranial nerve V (Trigeminal) may be tested in the comatose patient with the corneal reflex test.

- Cranial nerves IX(Glossopharyngeal) and X(vagus) may be evaluated with the gag reflex.



- The motor system is assessed by testing deep tendon reflexes, feeling the resistance of the patient's limbs to passive movements, and testing the strength of posturing and local withdrawal movements.

- The sensory system can only be evaluated by observing the patient's response, or lack of response, to noxious stimuli in different parts of the body.

Upper Motor Neuron (UMN) vs. Lower Motor Neuron (LMN) Syndrome

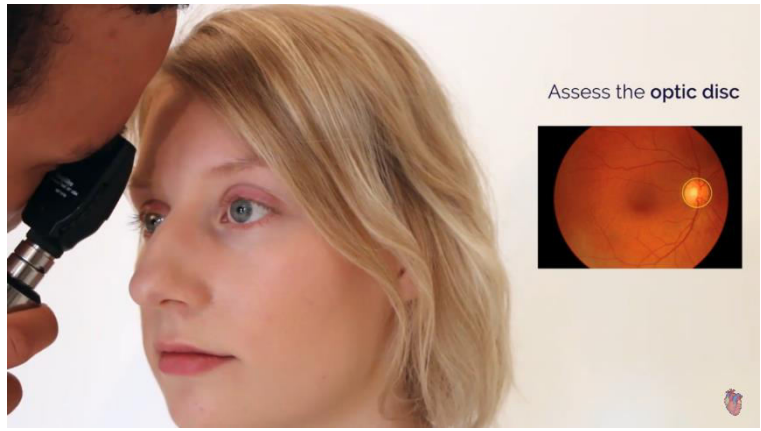
	UMN syndrome	***LMN Syndrome***
Type of Paralysis	***Spastic Paresis***	***Flaccid Paralysis***
Atrophy	**No (Disuse) Atrophy**	**Severe *Atrophy***
Deep Tendon Reflex	***Increase***	**Absent DTR**
Pathological Reflex	**Positive *Babinski* Sign**	**Absent**
Superficial Reflex	**Absent**	**Present**
Fasciculation and Fibrillation	**Absent**	**Could be *Present***

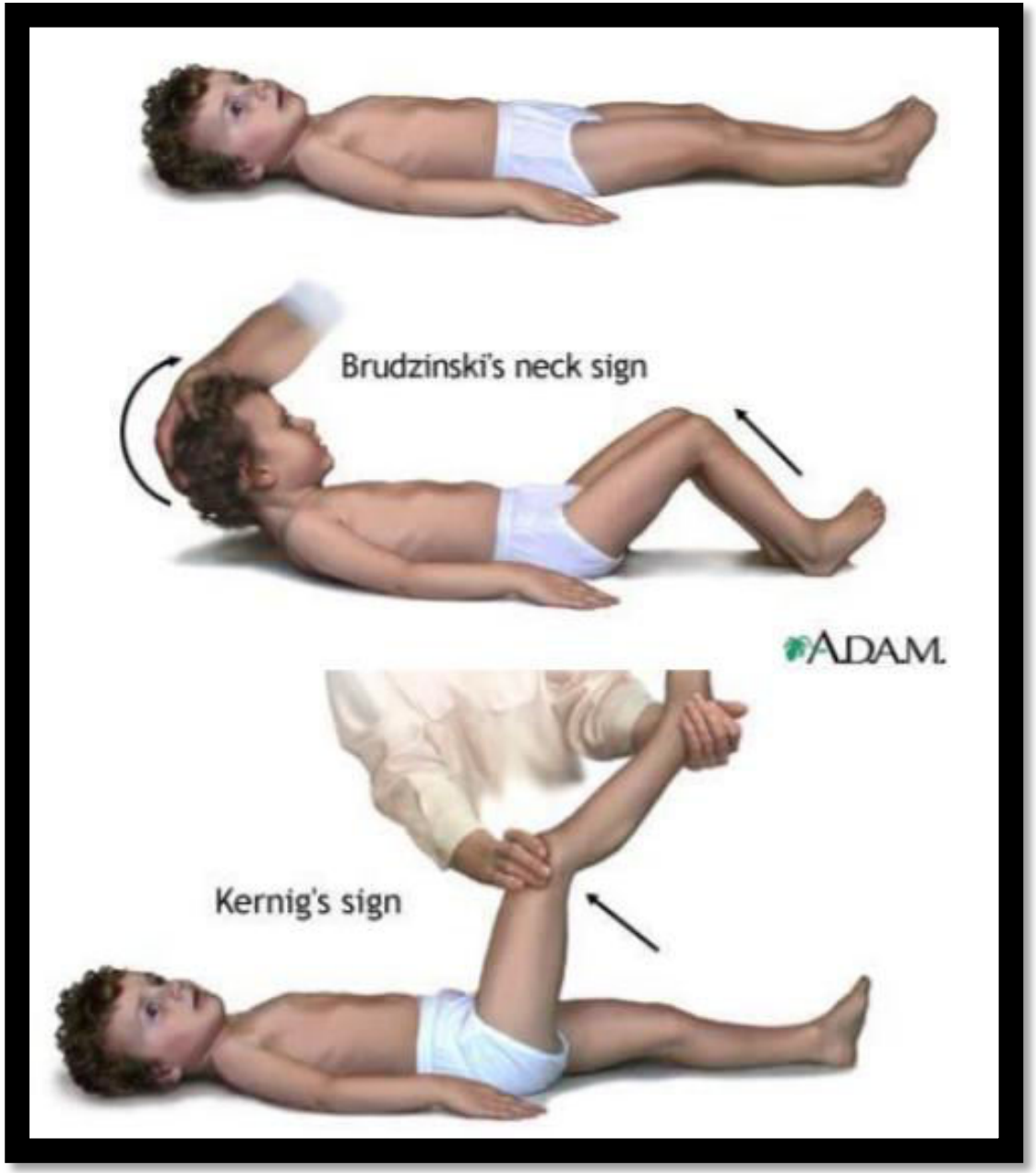
- **Fundus examination:**

  For papilloedema in cases of ICP

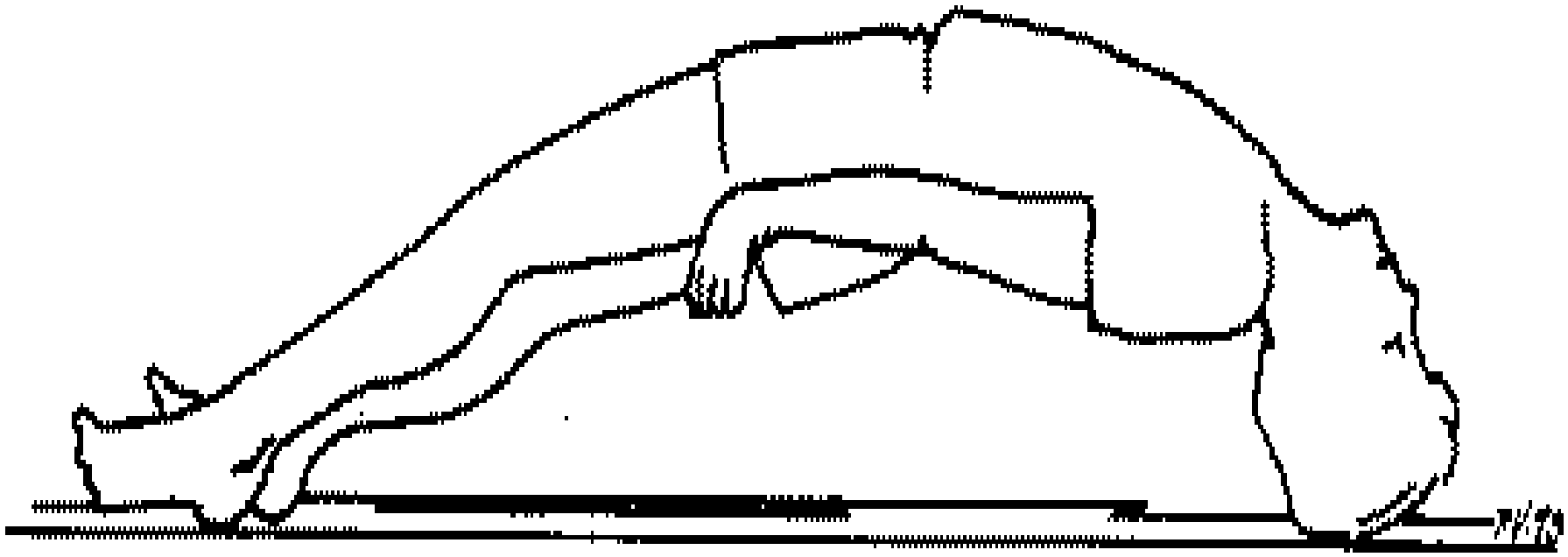
- **Signs of meningeal irritation :**

  Neck stiffness, positive Kernig's sign and Brudzinski's neck sign in cases of meningitis and subarachnoid hemorrhage




Brudzinski's neck sign
ADAM.
Kernig's sign

- **Opisthotonus:** is a symptom seen in some cases of sever cerebral palsy and traumatic brain injury or as a result of severe muscular spasm associated with tetanus



Management

- The emergency management of the unconscious patient consists first of protecting respiratory and circulatory function with standard life support techniques:

1. Assess vital signs, ABC's take priority.
2. Stabilize the cervical spine unless trauma is known not to be the cause.
3. Assess the level of consciousness using the Glasgow coma scale.
4. Check pupils every few minutes during the early stages and observe any changes in pupils' behaviour.

5. Check blood glucose if hypoglycemia is possible.
6. Control any seizures.
7. Smell the breath (alcohol, hepatic fetor, ketosis).
8. Check and correct any abnormalities in BP, body temperature, and electrolytes.
9. Identify the cause of coma, any immediately reversible cause should be treated:

- Hypoglycemia: Give 50ml 20% IV glucose or 50ml 50% IV dextrose.
- Wernicke's encephalopathy (Vitamin B1 deficiency): Give IV Thiamine.
- Drug overdose: Administer appropriate antidote

- Once the patient is stable, full history, detailed neurological examination, lab tests, blood cultures, toxicologic analysis of blood and urine, CT or MRI of the brain, LP may help identify the cause therefore treat the underlying problem.

Thank You