APPROACH TO THE COMATOSE PATIENT

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COMA

- Coma= State of deep sleep
- Coma is a profound state of unconsciousness.
- A person in a coma cannot be awakened
- Fails to respond normally to pain, light or sound
- Does not have sleep-wake cycles, and
- Does not take voluntary actions.
- A person in a state of coma can be described as **comatose.**

- Coma is a medical emergency and may constitute a diagnostic and therapeutic challenge for the intensivist.

- Coma and other states of impaired consciousness represent a severe derangement in cerebral function that may be structural or non structural in origin.

- Many of the underlying processes leading to coma can be both life threatening and potentially reversible with the timely institution of medical or surgical therapy.

Disorders Of Consciousness

- From a clinical perspective, **consciousness** may be schematized as the product of two closely related cerebral functions: *wakefulness* (i.e. arousal, vigilance, alertness) and *awareness* of self or of the environment, often referred to as the “content” of consciousness.

- The relationship between wakefulness and awareness is hierarchical: Awareness cannot occur without wakefulness, but wakefulness may be observed in the absence of awareness (e.g. the vegetative state).

- Wakefulness is linked to the ascending reticular activating system, a network of neurons originating in the tegmentum of the pons and midbrain and projecting to diencephalic and cortical structures.

- Awareness is dependant on the integrity of the cerebral cortex and its subcortical connections.

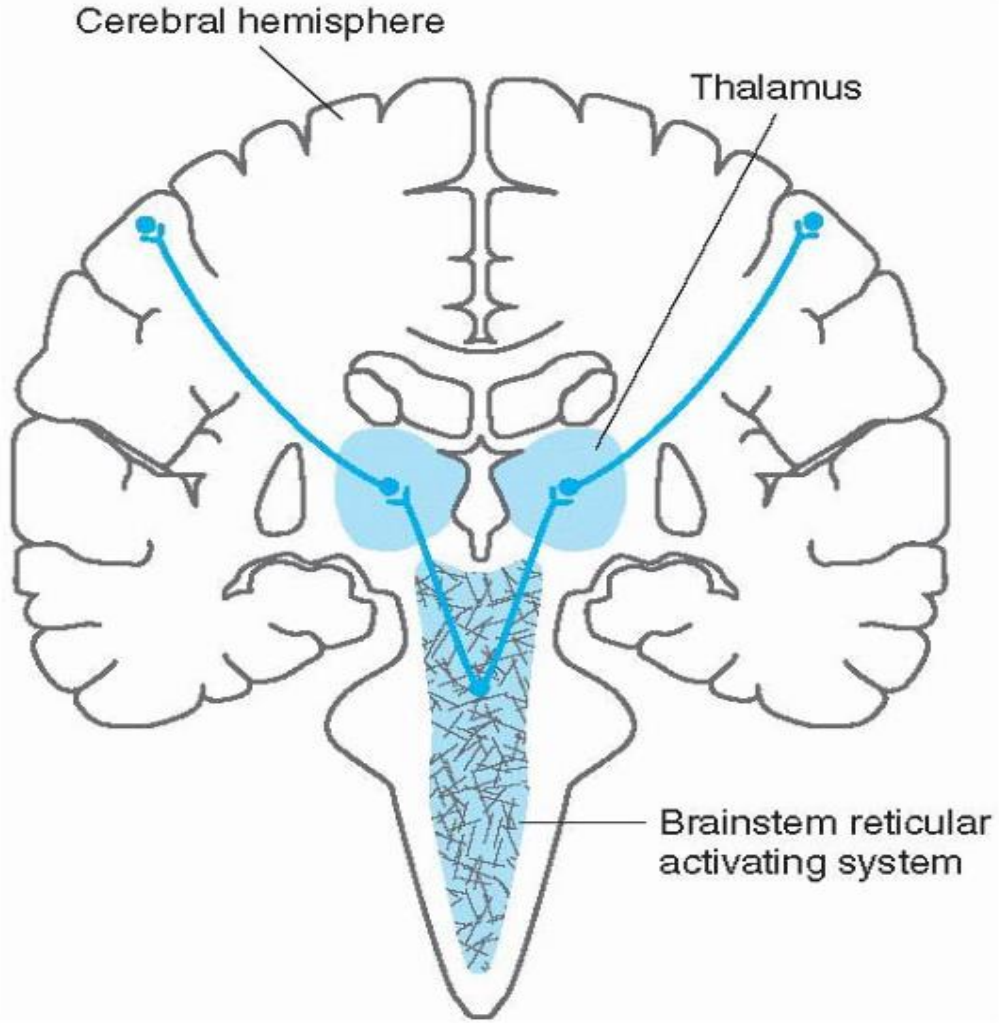


Figure 10-1. Anatomic basis of coma. Consciousness is maintained by the normal functioning of the brainstem reticular activating system above the midpons and its bilateral projections to the thalamus and cerebral hemispheres. Coma results from lesions that affect either the reticular activating system or both hemispheres.

- **Confusion** is clouding of consciousness characterized by impaired capacity to think, understand, respond & remember stimuli.
- **Delirium** is a state of confusion that is accompanied by agitation, hallucination, tremors or illusion.
- **Stupor** is unresponsiveness from which the patient can be aroused only briefly by vigorous repeated stimulation.
- **Coma** is a state in which patient is unarousable and unresponsive and any response to repeated stimuli is only primitive avoidance reflex.

- **Vegetative state** is a subacute or chronic condition after severe brain injury and comprises return of wakefulness accompanied by total lack of cognitive function.

- **Akinetic mutism** refers to partially or fully awake patient who is able to form impression & think but remains immobile, mute particularly when stimulated.

- **Locked in syndrome** is a awake state in which there is total paralysis of limbs, lower cranial nerves with intact consciousness and vertical eye movements.

Etiology & Pathogenesis

- From a pathophysiologic standpoint, coma may be viewed as the expression of

a) Primary insults to the cerebral cortex, diencephalic structures, midbrain or rostral pons; and
b) Secondary cerebral manifestations of systemic toxic, metabolic or endocrine derangements.

Table 3. Etiology of coma and altered consciousness

I. Primary cerebral disorders
- Bilateral or diffuse hemispheric disorders
  - Traumatic brain injury (contusions, diffuse axonal injury)
  - Ischemic (watershed, cardioembolism, vasculitis, hypercoagulable disorder)
  - Hemorrhagic (subarachnoid hemorrhage, intraventricular hemorrhage)
  - Hypoxic-ischemic encephalopathy
  - Cerebral venous thrombosis
  - Malignancy
  - Meningitis; encephalitis
  - Generalized or complex partial seizures; status epilepticus (convulsive, nonconvuls
  - Hypertensive encephalopathy
  - Posterior reversible encephalopathy syndrome
  - Acute disseminated encephalomyelitis
  - Hydrocephalus
- Unilateral hemispheric disorders (with displacement of midline structures)
  - Traumatic (contusions, subdural hematoma, epidural hematoma)
  - Large hemispheric ischemic stroke
  - Primary intracerebral hemorrhage
  - Cerebral abscess
  - Brain tumor
- Brain stem disorders (pons, midbrain)
  - Hemorrhage, infarction, tumor, trauma
  - Central pontine myelinolysis
  - Compression from cerebellar infarct, hematoma, abscess, tumor

II. Systemic derangements causing coma

- Toxic
  - Medication overdose/adverse effects (opioids, benzodiazepines, barbiturates, tricyclics, neuroleptics, aspirin, selective serotonin reuptake inhibitors, acetaminophen, anticonvulsants)
  - Drugs of abuse (opioids, alcohol, methanol, ethylene glycol, amphetamines, cocaine)
  - Exposures (carbon monoxide, heavy metals)
- Metabolic
  - Systemic inflammatory response syndrome-sepsis
  - Hypoxia; hypercapnia
  - Hypothermia
  - Hypoglycemia; hyperglycemic crises (diabetic ketoacidosis, nonketotic hyperosmolar hyperglycemic state)
  - Hyponatremia, hypernatremia
  - Hypercalcemia
  - Hepatic failure
  - Renal failure
  - Wernicke's encephalopathy
- Endocrine
  - Panhypopituitarism
  - Adrenal insufficiency
  - Hypothyroidism; hyperthyroidism

- To affect consciousness, lesions of the cerebral cortex must involve both hemispheres or must be unilateral lesions large enough to cause displacement of midline structures

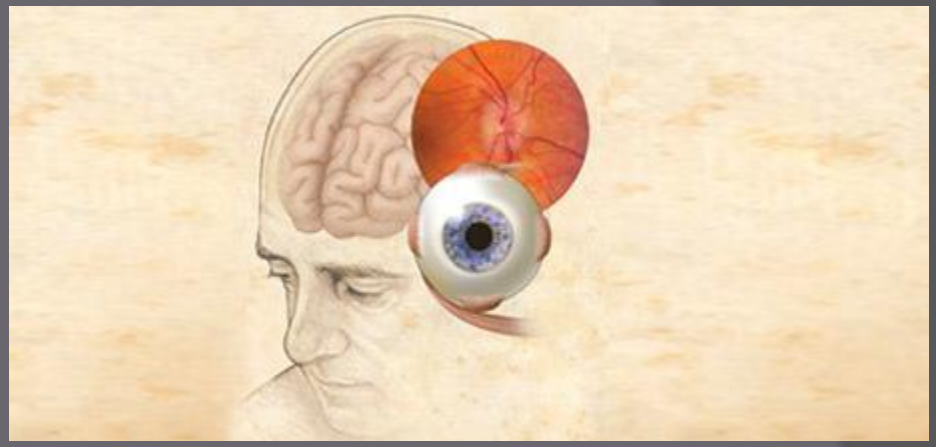
- Brainstem and diencephalic lesions resulting in coma may be comparatively small; however they may also involve bilateral structures.

- The proximity of the RAS to midbrain structures that control the pupillary function and eye movements permits clinical localization of the cause of coma in many cases.

- Pupillary enlargement with the loss of light reaction and loss of vertical and adduction movements of the eyes suggests that the lesion is in the upper brainstem.
- Conversely, preservation of pupillary light reactivity and of eye movement absolves the upper brainstem and indicates widespread structural lesions or metabolic suppression of the cerebral hemispheres is responsible for coma.

- The pathophysiology of toxic and metabolic coma is specific to the underlying cause and in many instances incompletely understood.

- In a simplified view, these conditions have been linked to an interruption in the delivery or utilization of oxygen or substrate (hypoxia, ischemia, hypoglycemia), alterations in neuronal excitability and signaling (seizures, acidosis, drug toxicity), or changes in brain volume (hyponatremia, hypernatremia).



Clinical Approach To The Patient

- Many times the primary disorder underlying coma is perfectly obvious, as with severe cranial trauma. All too often, however, the patient is brought to the hospital in a state of coma and little pertinent medical information is available.

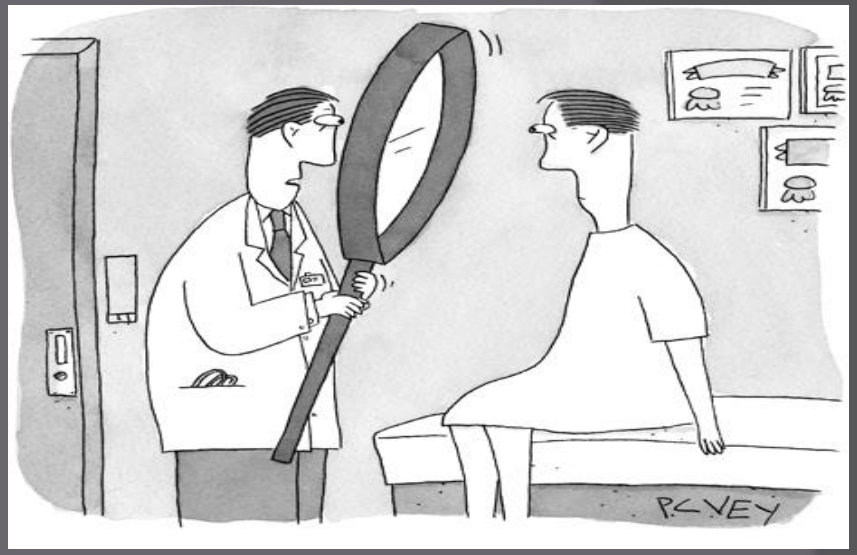
- The need for efficiency in reaching a diagnosis and providing appropriate acute care demands that the physician have a methodical approach that addresses the common and treatable causes of coma.

- An inquiry is then made as to the previous health of the patient, whether there was a history of diabetes, a head injury, a convulsion, alcohol or drug use, or a prior episode of coma or attempted suicide, and the circumstances in which the person was found.

- Persons who accompany the comatose patient to the hospital should be encouraged to remain until they have been questioned.

- In assessing stupor or coma in an already hospitalized patient, it is most instructive to review the patient's medications carefully.

- *From an initial survey, many of the common causes of coma, such as severe head injury, alcoholic or other forms of drug intoxication, and hypertensive brain hemorrhage, are readily recognized.*



General examination

- Alterations in *vital signs* (temperature, heart rate, respiratory rate, and blood pressure) are important aids in diagnosis.

- *Fever* is most often the result of a systemic infection such as pneumonia or bacterial meningitis or viral encephalitis. An excessively high body temperature (42°C [107.6°F] or 43°C [109.4°F]) associated with dry skin should arouse suspicion of heat stroke or intoxication by a drug with anticholinergic activity.

- *Hypothermia* is observed in patients with alcohol or barbiturate intoxication, drowning, exposure to cold, peripheral circulatory failure, advanced tuberculous meningitis, and myxedema.

- Shallow, slow, but regular breathing suggests metabolic or drug depression.
- *Rapid, deep (Kussmaul) breathing* usually implies metabolic acidosis but may also occur with pontomesencephalic lesions.
- *Cheyne-Stokes respiration* in its classic cyclic form, ending with a brief apneic period, signifies bihemispheral damage/bilateral thalamic lesions or metabolic suppression and commonly accompanies light coma.
- Patients with lesions of the midbrain and pons often have prolonged and rapid hyperpnea (neurogenic hyperventilation)

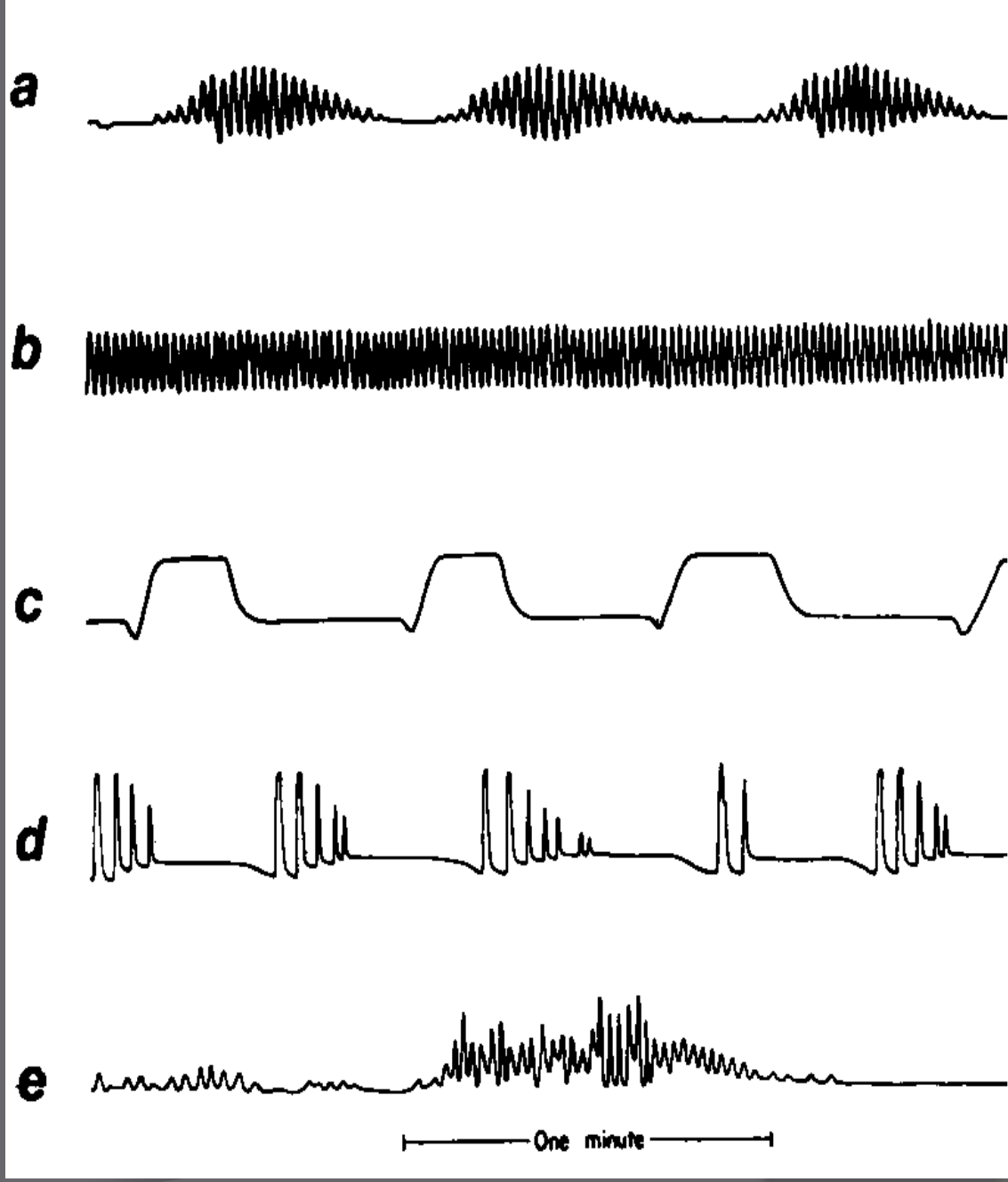
- Apneustic breathing is characterized by a long inspiratory pause, after which the air is retained for several seconds and then released. This abnormality appears with lesions of the lateral tegmentum of the lower half of the pons.

- Cluster Breathing-Breathing with a cluster of breaths following each other in an irregular sequence may result from low pontine or high medullary lesions.

- In ataxic breathing the breathing pattern is irregular with erratic shallow and deep respiratory movements; seen with dysfunction of medullary respiratory centers and may signify impending agonal respirations and apnea

- *Agonal gasps* are the result of lower brainstem (medullary) damage and are well known as the terminal respiratory pattern of severe brain damage.

a) Cheynes stokes respiration
b) Central neurogenic hyperventilation
c) Apneustic breathing
d) Cluster breathing
e) Ataxic / Biot's breathing



- *Vomiting* at the outset of sudden coma, particularly if combined with pronounced hypertension, is highly characteristic of cerebral hemorrhage within the hemispheres, brainstem, cerebellum, or subarachnoid spaces.

- The *heart rate* , if exceptionally slow, suggests heart block from medications such as tricyclic antidepressants or anticonvulsants, or if combined with periodic breathing and hypertension, an increase in intracranial pressure.

- Marked *hypertension* is observed in patients with cerebral hemorrhage and in hypertensive encephalopathy

- *Hypotension* is the usual finding in states of depressed consciousness because of diabetes, alcohol or barbiturate intoxication, internal hemorrhage, myocardial infarction, dissecting aortic aneurysm, septicemia, Addison disease, or massive brain trauma.

- ***Inspection of the skin*** may yield valuable information.
- Cyanosis of the lips and nail beds signifies inadequate oxygenation.
- Cherry-red coloration is typical of carbon monoxide poisoning.
- Multiple bruises (particularly a bruise or boggy area in the scalp), bleeding, CSF leakage from an ear or the nose, or periorbital hemorrhage greatly raises the likelihood of cranial fracture and intracranial trauma.
- Telangiectases and hyperemia of the face and conjunctivae are the common stigmata of alcoholism
- Myxedema imparts a characteristic puffiness of the face.
- Marked pallor suggests internal hemorrhage.
- A macular-hemorrhagic rash indicates the possibility of meningococcal infection, staphylococcal endocarditis, typhus, or Rocky Mountain spotted fever.

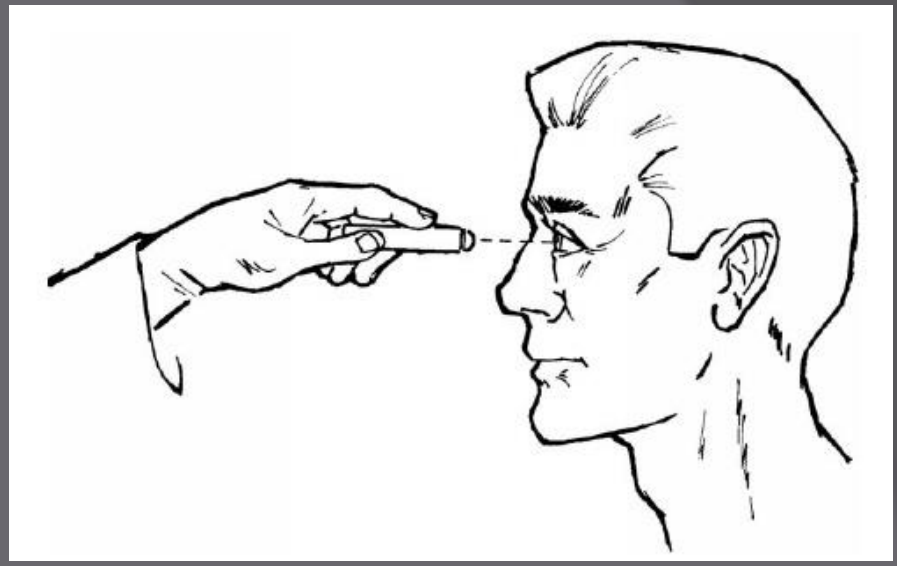
- Excessive sweating suggests hypoglycemia or shock, and excessively dry skin, diabetic acidosis or uremia.

- Thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation, and fat embolism may cause diffuse petechiae or purpura; the last of these are often aggregated in the anterior axillary folds.

- The ***odor of the breath*** may provide a clue to the etiology of coma.

- Alcohol is easily recognized

- The spoiled-fruit odor of diabetic ketoacidotic coma, the uriniferous odor of uremia, the musky and slightly fecal fetor of hepatic coma, and the burnt almond odor of cyanide poisoning are distinctive enough to be *identified by physicians who possess a keen sense of smell.*



Neurological examination

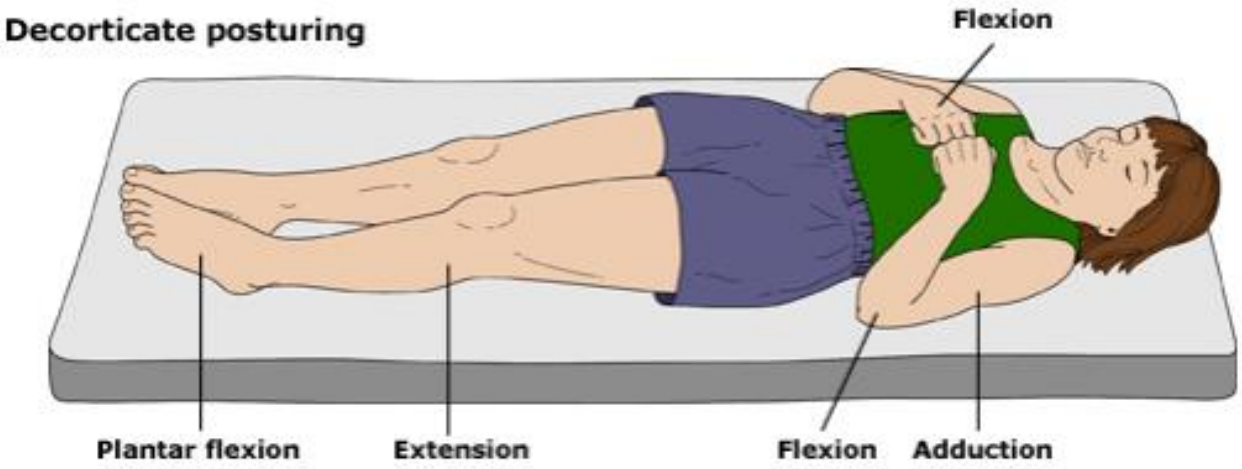
- Although limited in some ways in comparison to the examination of the alert patient, the neurologic examination of the comatose patient is relatively simple and of crucial importance.

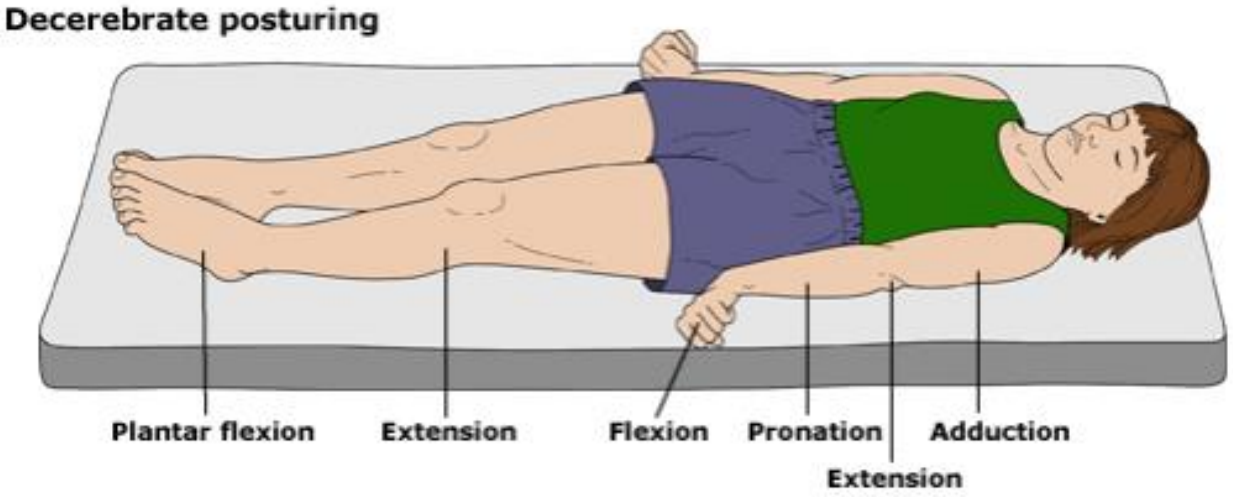
- *Simply watching the patient for a few moments often yields considerable information*. The predominant postures of the limbs and body; the presence or absence of spontaneous movements on one side; the position of the head and eyes; and the rate, depth, and rhythm of respiration give substantial information.

- *Decorticate rigidity* and *decerebrate rigidity*, or "posturing," describe stereotyped arm and leg movements occurring spontaneously or elicited by sensory stimulation.

- Flexion of the elbows and wrists and supination of the arm (decortication) suggests bilateral damage rostral to the midbrain, whereas extension of the elbows and wrists with pronation (decerebration) indicates damage to motor tracts in the midbrain or caudal diencephalon.

Decorticate/decerebrate postures





- The less frequent combination of arm extension with leg flexion or flaccid legs is associated with lesions in the pons.

- *These concepts have been adapted from animal work and cannot be applied with precision to coma in humans.*
- In fact, acute and widespread disorders of any type, regardless of location, frequently cause limb extension, and almost all extensor posturing becomes predominantly flexor as time passes.

- The state of responsiveness is then estimated by noting the patient's reaction to calling his name, to simple commands, or to noxious stimuli such as tickling the nares, supraorbital or sternal pressure, pinching the side of the neck or inner parts of the arms or thighs, or applying pressure to the knuckles.

  By gradually increasing the strength of these stimuli, one can roughly estimate both the degree of unresponsiveness and changes from hour to hour.

- Posturing in response to noxious stimuli indicates severe damage to the corticospinal system, whereas abduction-avoidance movement of a limb is usually purposeful and denotes an intact corticospinal system.

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		

E+M+V = 15: Normal

E+M+V ≥ 11: 5-10% chance of vegetative state

E+M+V = 3-4: 85% chance of vegetative state

NECK SIGNS

- It is usually possible to determine whether coma is associated with meningeal irritation.

- In all but the deepest stages of coma, meningeal irritation from either bacterial meningitis or subarachnoid hemorrhage will cause resistance to the initial excursion of passive flexion of the neck but not to extension, turning, or tilting of the head. It is a fairly specific but somewhat insensitive sign of meningeal irritation.

- Resistance to movement of the neck in all directions may be part of generalized muscular rigidity (as in phenothiazine intoxication) or indicate disease of the cervical spine.

- **Assessment of brainstem function**
- The brainstem reflexes that are conveniently examined are pupillary size and reaction to light, spontaneous and elicited eye movements, corneal responses, and the respiratory pattern.
- As a rule, coma is due to bilateral hemispheral disease when these brainstem activities are preserved, particularly the pupillary reactions and eye movements.
- *However, the presence of abnormal brainstem signs does not always indicate that the primary lesion is in the brainstem because hemispheral masses can cause secondary brainstem pathology by transtentorial herniation.*

Pupillary Reactions -

- A unilaterally enlarged pupil (5.5 mm in diameter) <HUTCHINSON PUPIL> is an early indicator of stretching or compression of the third nerve and reflects the presence of an overlying ipsilateral hemispheral mass.
- A loss of light reaction usually precedes enlargement of the pupil. As a transitional phenomenon, the pupil may become oval or pear-shaped or appear to be off center (corectopia) because of a differential loss of innervation of a portion of the pupillary sphincter.
- The light-unreactive pupil continues to enlarge to a size of 6 to 9 mm diameter and is soon joined by a slight outward deviation of the eye.

- In unusual instances, the pupil contralateral to the mass may enlarge first; this has reportedly been the case in 10 percent of subdural hematomas but has been far less frequent.
- As midbrain displacement continues, both pupils dilate and become unreactive to light, probably as a result of compression of the oculomotor nuclei in the rostral midbrain.
- The last step in the evolution of brainstem compression tends to be a slight reduction in pupillary size on both sides, to 5 mm or smaller.
- *Normal pupillary size, shape, and light reflexes indicate integrity of midbrain structures and direct attention to a cause of coma other than a mass*

- The Horner syndrome (miosis, ptosis, and reduced facial sweating) may be observed ipsilateral to a lesion of the brainstem *or* hypothalamus *or* as a sign of dissection of the internal carotid artery.

  Lateral pontine, lateral medullary, and ventrolateral cervical cord lesions produce an ipsilateral Horner syndrome.

- 2.5 – 5.0 mm reactive pupils rules out midbrain damage
- 1.0 – 2.5 mm (not pinpoint) reactive pupils suggest: Metabolic encephalopathy, Thalamic hemorrhage, Hydrocephalus
- B/L pinpoint pupils:
  - Do not react to light: Pontine hemorrhage
  - Reactive to light: Barbiturate overdose
  - React to naloxone: Opiate overdose
- Unilateral dilated nonreactive 6.0 mm pupil: 3rd CN damage
- Bilateral dilated nonreactive pupil: Severe brainstem damage

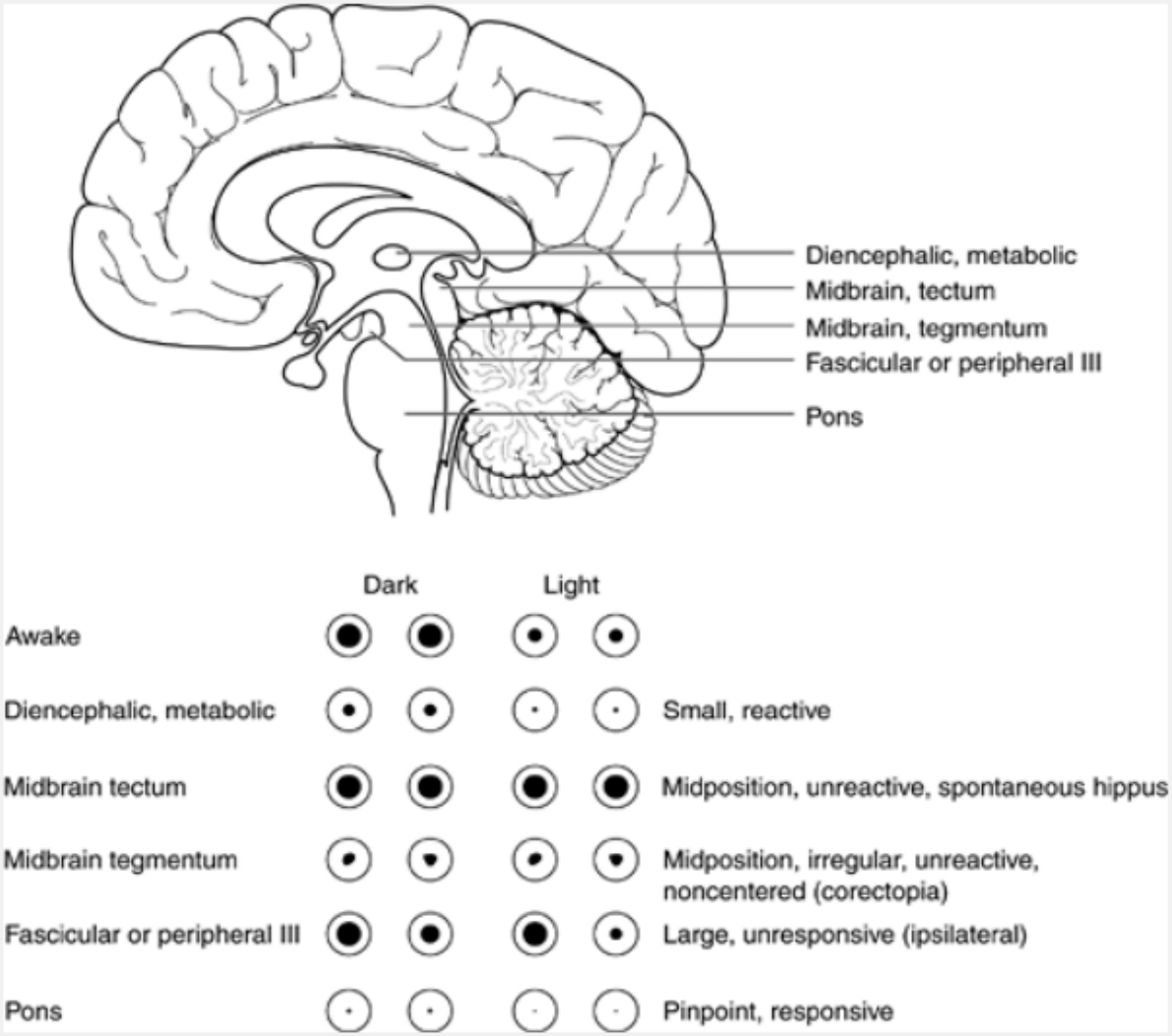


FIGURE 22-3 Pupillary responses characteristic of lesions at different levels of the brain.

Movements of Eyes and Eyelids and Corneal Responses

- In light coma of metabolic origin, the eyes rove conjugately from side to side in seemingly random fashion, sometimes resting briefly in a eccentric position. These movements disappear as coma deepens, and the eyes then remain motionless and slightly exotropic.

- The eyelids may remain tonically retracted because of failure of levator inhibition in some cases of pontine infarction (eyes-open coma)

- There is persistent conjugate deviation of the eyes to one side—away from the side of the paralysis with a large cerebral lesion (looking toward the lesion) and toward the side of the paralysis with a unilateral pontine lesion (looking away from the lesion).

- ***Oculocephalic reflexes (doll's-eye movements)*** are elicited by briskly turning or tilting the head.
- The response in coma of metabolic origin or that caused by bihemispheric structural lesions consists of conjugate movement of the eyes in the opposite direction.
- Elicitation of these ocular reflexes in a comatose patient provides two pieces of information:

  (1) evidence of unimpeded function of the midbrain and pontine tegmental structures that integrate ocular movements and of the oculomotor nerves, and

  (2) loss of the cortical inhibition that normally holds these movements in check.

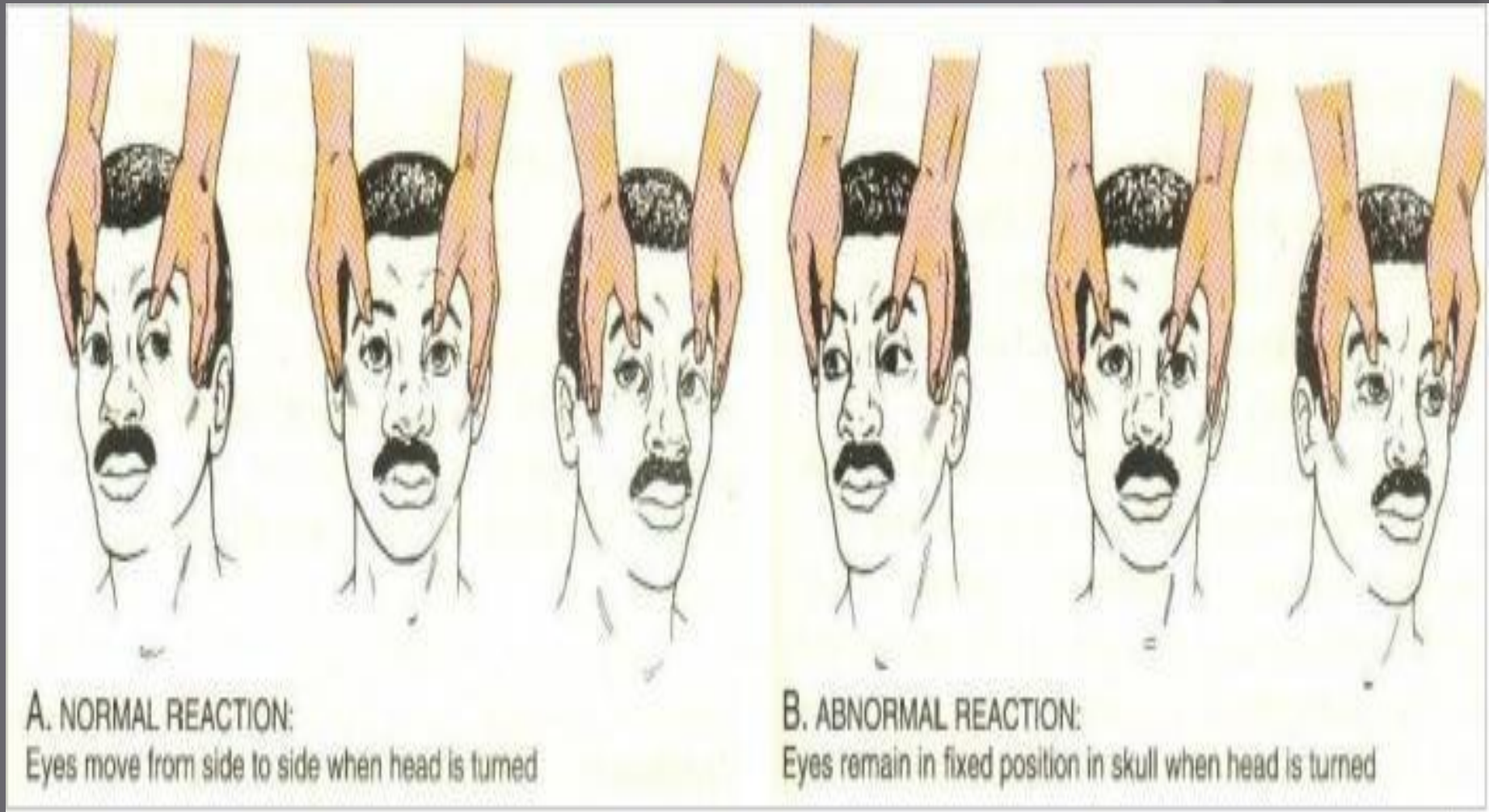
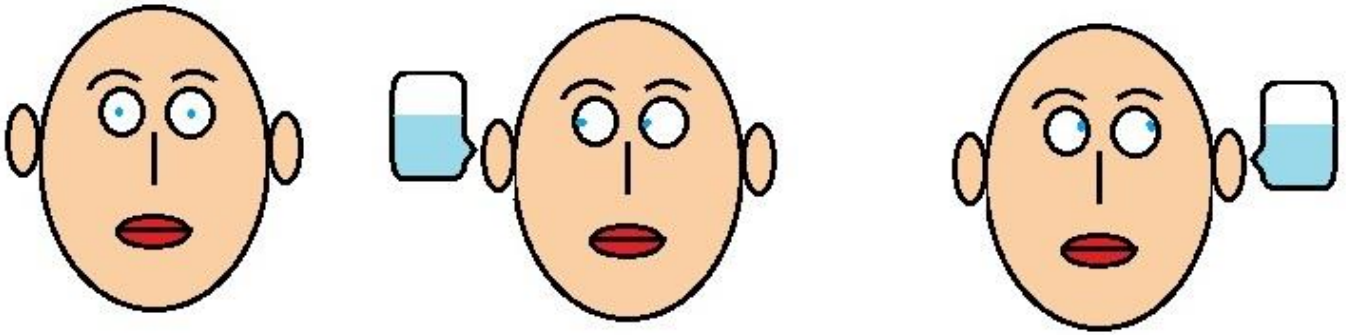


Figure 1. Testing the vestibulo-ocular reflex[8]

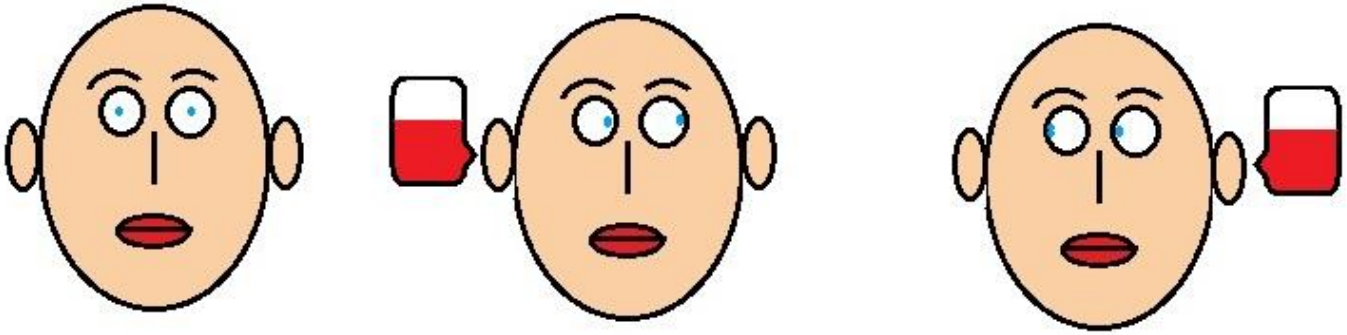
- In other words, *the presence of unimpaired reflex eye movements implies that coma is not caused by compression or destruction of the upper midbrain.* There must instead be widespread cerebral dysfunction, such as occurs after anoxia or with metabolic–toxic suppression of cortical neuronal activity.

Vestibulo-ocular Reflex

Cold water:



Warm water:



- Thermal, or "caloric," stimulation of the vestibular apparatus (oculovestibular response) provides a more intense stimulus for the oculocephalic reflex but provides essentially the same information.

- The test is performed by irrigating the external auditory canal with cool water in order to induce convection currents in the labyrinths.

- After a brief latency, the result is tonic deviation of both eyes to the side of cool-water irrigation and nystagmus in the opposite direction.

- Absence of nystagmus with preserved conjugate deviation: Cerebral hemisphere damage
- Absence of conjugate movement: Brainstem lesion

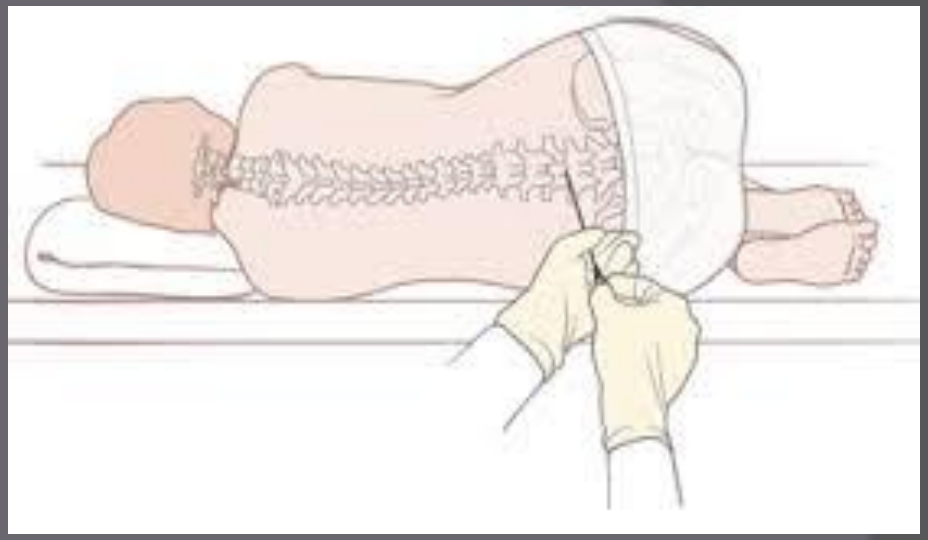
- *As coma deepens, roving eye movements disappear first, followed by the oculocephalic reflex; finally, even cold water instilled in the ear fails to induce eye movements.*

- ***Clinical Signs of Increased Intracranial Pressure***

- A history of headache before the onset of coma, vomiting, severe hypertension beyond the patient's static level, unexplained bradycardia, and subhyaloid retinal hemorrhages (Terson syndrome) are immediate clues to the presence of increased intracranial pressure, usually from one of the types of cerebral hemorrhage.

- *Papilledema develops within 12 to 24 h in cases of brain trauma and hemorrhage, and if it is apparent when coma supervenes, it usually signifies brain tumor or abscess, i.e., a lesion of longer duration.*

- The syndrome of acute hydrocephalus, most often from subarachnoid hemorrhage or from obstruction of the ventricular system by a tumor in the posterior fossa, induces a state of abulia (slowed responsivity), followed by stupor, and then coma with bilateral Babinski signs. The pupils are small and tone in the legs is increased. The signs of hydrocephalus may be accompanied by headache and systemic hypertension, mediated through raised intracranial pressure.



Laboratory Procedures for Coma Diagnosis

- The studies that are most useful in the diagnosis of coma are:
  - Chemical-toxicologic analysis of blood and urine
  - cranial CT or MRI
  - EEG
  - CSF examination
- Arterial blood gas analysis is helpful in patients with lung disease and acid-base disorders.
- The metabolic aberrations commonly encountered in clinical practice require measurement of electrolytes, glucose, calcium, osmolarity, and renal (blood urea nitrogen) and hepatic (NH_3) function.

- The notion that a normal CT scan excludes anatomic lesion as the cause of coma is erroneous.

- Bilateral hemisphere infarction, acute brainstem infarction, encephalitis, meningitis, mechanical shearing of axons as a result of closed head trauma, sagittal sinus thrombosis, and subdural hematoma isodense to adjacent brain are some of the disorders that may not be detected.

- Nevertheless, if the source of coma remains unknown, a scan should be obtained.

- The EEG is useful in metabolic or drug-induced states but is rarely diagnostic, except when coma is due to clinically unrecognized seizure, to herpes virus encephalitis, or to prion (Creutzfeldt-Jakob) disease.

- Examination of the CSF remains indispensable in the diagnosis of meningitis and encephalitis. For patients with an altered level of consciousness, it is generally recommended that an imaging study be performed prior to lumbar puncture to exclude a large intracranial mass lesion.

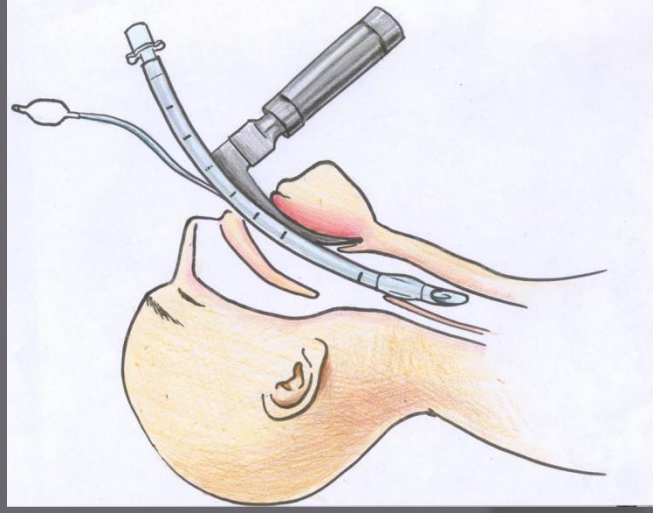
DIFFERENTIAL DIAGNOSIS

PSYCHIATRIC UNRESPONSIVENESS (Hysterical coma)

- CAUSES: Catatonia, hysteria, malingering & depression

MANIFESTATIONS:

Lids close actively
Pupils are reactive or dilated
Oculocephalic are unpredictable
Oculovestibular: Physiologic nystagmus present
Motor tone is normal or inconsistent
Euphoria or hyperventilation is usual
No pathological reflex is present
EEG is normal



Management of the comatose patient

- Shallow and irregular respirations, stertorous breathing (indicating obstruction to inspiration), and cyanosis require the establishment of a clear airway and delivery of oxygen.
- A patient's inability to protect against aspiration and the presence of either hypoxia or hypoventilation dictate the use of endotracheal intubation and a positive-pressure respirator. Endotracheal tube upto 1 wk /Tracheostomy after 1 wk.
- Patients with a GCS of 8 or less usually require endotracheal intubation to protect the airway. This can sometimes be avoided, eg, in patients with large hemispheric strokes or alcohol withdrawal seizures. Intubation is also advised in the presence of hypoxemia (oxygen saturation of <90 percent), recent vomiting, or poor cough or gag reflex. Oxygen supplementation is often needed, whether or not assisted ventilation is required.

- The management of shock, if present, takes precedence over all other diagnostic and therapeutic measures.

- Concurrently, an intravenous line is established and blood samples are drawn for determination of glucose, intoxicating drugs, and electrolytes and for tests of liver and kidney function. Naloxone, 0.5 mg, should be given intravenously if a narcotic overdose is a possibility.

- Hypoglycemia that has produced stupor or coma demands the infusion of glucose, usually 25 to 50 mL of a 50 percent solution followed by a 5 percent infusion; this must be supplemented with thiamine.

- With the development of elevated intracranial pressure from a mass lesion, mannitol should be given and hyperventilation instituted if deterioration occurs, as judged by pupillary enlargement or deepening coma.

- Repeated CT scanning allows the physician to follow the size of the lesion and degree of localized edema and to detect displacements of cerebral tissue.

- A lumbar puncture should be performed if meningitis is suspected on the basis of headache and meningismus (and fever in the case of infectious meningitis), keeping in mind the risks of this procedure and the means of dealing with them. In the case of meningitis, broad-spectrum antibiotics that penetrate the meninges may be instituted, especially if the lumbar puncture is delayed.

- Convulsions should be controlled by antiepileptic drugs.
- The temperature-regulating mechanisms may be disturbed, and extreme hypothermia(<32 C) or hyperthermia >38.5 C) should be corrected. In severe hyperthermia, evaporative- cooling measures are indicated in addition to antipyretics.
- Leg vein thrombosis, a common occurrence in comatose and hemiplegic patients, often does not manifest itself by obvious clinical signs. An attempt may be made to prevent it by the subcutaneous administration of heparin, 5,000 U q12h, or of low-molecular weight heparin, and by the use of intermittent pneumatic compression boots.
- Regular conjunctival lubrication and oral cleansing should be instituted.

- Care of bladder & prevention of bedsores:
  - Condom drainage/catheterization
  - Prone/semiprone position with hourly change of posture
  - Suction as and when required, chest physiotherapy
  - alpha bed
  - Avoid bed wetting

- Physical therapy to prevent skeletal & muscular deformities and pressure palsies

- Adequate fluid and nutrition
  - I/V fluids are given only up to 1 week
  - Ryles tube feeding up to 8 weeks
  - Feeding gastrostomy or gastrojejunostomy after 8 weeks

Prognosis of Coma

- As a general rule, *recovery from coma of metabolic and toxic causes is far better than from anoxic coma*, with head injury occupying an intermediate prognostic position.

- *Most patients who are comatose as a result of a stroke will die*; subarachnoid hemorrhage in which coma is a result of hydrocephalus is an exception and those cases in which brain shift is relieved by craniectomy are also exceptions.

- In regard to all forms of coma, but particularly after cardiac arrest, if there are *no pupillary, corneal, or oculovestibular responses within several hours of the onset of coma, the chances of regaining independent function are practically nil*

- Only approximately 15 % of patients in non traumatic coma make a satisfactory recovery.
- Functional recovery is related to the cause of coma. Diseases causing structural damage, such as cerebrovascular disease including SAH, have the worst prognosis; coma from hypoxia-ischemia due to causes such as cardiac arrest has an intermediate prognosis; coma due to hepatic encephalopathy and other metabolic causes has the best ultimate outcome.
- Age does not appear to be predictive of recovery.
- The longer a coma lasts, the less likely the patient is to regain independent functioning.

- For anoxic and metabolic coma, clinical signs such as the pupillary and motor responses after 1 day, 3 days, and 1 week have been shown to have predictive value. Other studies suggest that the absence of corneal responses may have the most discriminative value.

- The absence of the cortical waves of the somatosensory evoked potentials has also proved a strong indicator of poor outcome in coma from any cause.

Brain Death

- This is a state of cessation of cerebral function with preservation of cardiac activity and maintenance of somatic function by artificial means.

- Ideal criteria are simple, can be assessed at the bedside, and allow no chance of diagnostic error. They contain three essential elements:

(1) widespread cortical destruction that is reflected by deep coma and unresponsiveness to all forms of stimulation;

(2) global brainstem damage demonstrated by absent pupillary light reaction and by the loss of oculovestibular and corneal reflexes; and

(3) destruction of the medulla, manifested by complete apnea.

- The heart rate is invariant and unresponsive to atropine.
- The pupils are usually midsized but may be enlarged; they should not, however, be small.
- Loss of deep tendon reflexes is not required because the spinal cord remains functional.
- Babinski signs are generally absent and the toe response is often flexor.
- Demonstration that apnea is due to irreversible medullary damage requires that the Pco_2 be high enough to stimulate respiration during a test of spontaneous breathing. *Apnea testing* can be done safely by the use of diffusion oxygenation prior to removing the ventilator.
- An isoelectric EEG may be used as a confirmatory test for total cerebral damage.
- The possibility of profound drug-induced or hypothermic depression of the nervous system should be excluded, and some period of observation, usually 6–24 hours, is desirable, during which the clinical signs of brain death are sustained.

THANK YOU