DIFFERENTIAL DIAGNOSTIC OF COMA

Department of internal diseases

*What defines Coma ?

"A state of unresponsiveness in which the patient lies with his eyes closed and cannot be aroused to respond appropriately to stimuli even with vigorous stimulation. The patient may grimace in response to painful stimuli and limbs may demonstrate stereotyped withdrawal responses, but the patient does not make localized responses or discrete defensive movements."

Posner et al, 2007

*In simple terms…

Defined as comatose if they have no ability to take in information from their environment and cannot respond to external stimuli in a meaningful way

*Objectives

*Primary Objective: The physician should be able to stabilize, evaluate, and treat the comatose patient in the emergent setting.

*The physician should understand this involves an organized, sequential, prioritized approach.

*Primary Objectives

* Airway
* Breathing
* Circulation
* Treatment of rapidly progressive, dangerous metabolic causes of coma (hypoglycemia)
* Evaluation as to whether there is significant increased intracranial pressure (ICP) or mass lesions.
* Treatment of ICP to temporize until surgical intervention is possible.

*Secondary Objectives

- The physician should understand and recognize:
  - Coma
  - Herniation syndromes
  - Signs of supratentorial mass lesions
  - Signs of subtentorial mass lesions
- The physician should be able to develop the differential diagnosis of metabolic coma.

* Causes of Coma

Structural causes

1. Trauma (ICP, diffuse axonal injury,concussion)
2. Infection (Meningitis, encephalitis, abscess)
3. ADEM, demyelination
4. Hydrocephalus
5. Nutritional (Wernicke's)
6. Seizure(Non-convulsive status)
7. Vasculitis

Toxic/ Metabolic

1. Hypo or hyperglycemia
2. Renal failure
3. Electrolyte abnormalities
4. Hepatic failure
5. Hypoxia
6. Hypercarbia
7. Porphyria
8. Acid -base disorders
9. Drugs & Toxins

A Alcohol
Acidosis (metabolic disorders)
Ammonia (hepatic encephalopathy)
Arrhythmias (any cardiac cause)

E Endocrine
Electrolytes
Encephalopathy

I Infection

O Oxygen
Overdose
Opiates

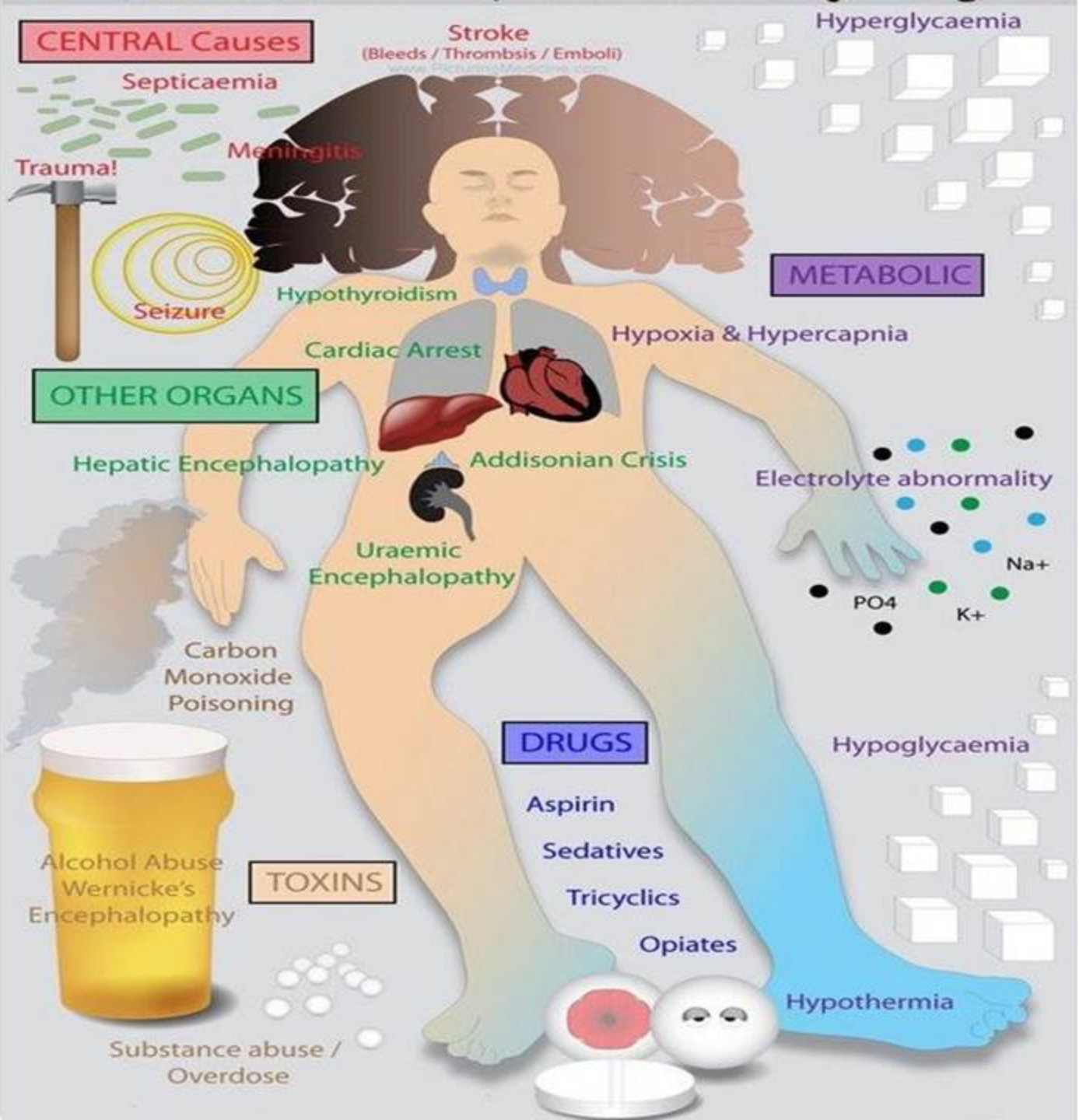
U Uremia

T Trauma
Temperature (hyper/hypothermia)
Thiamine (Wernicke-Korsakoff)

I Insulin (hypo/hyperglycemia)

P Poisoning (all medications)
Pyschiatric

S Stroke
Seizure (or postictal state)
Syncope
Space occupying lesions
Shunt (VP) malfunction



*Neurophysiology

- Consciousness requires:
  - An intact pontine reticular activating system
  - An intact cerebral hemisphere, or at least part of a hemisphere
- Coma requires dysfunction of either the:
  - Pontine reticular activating system, *or*
  - Bihemispheric cerebral dysfunction

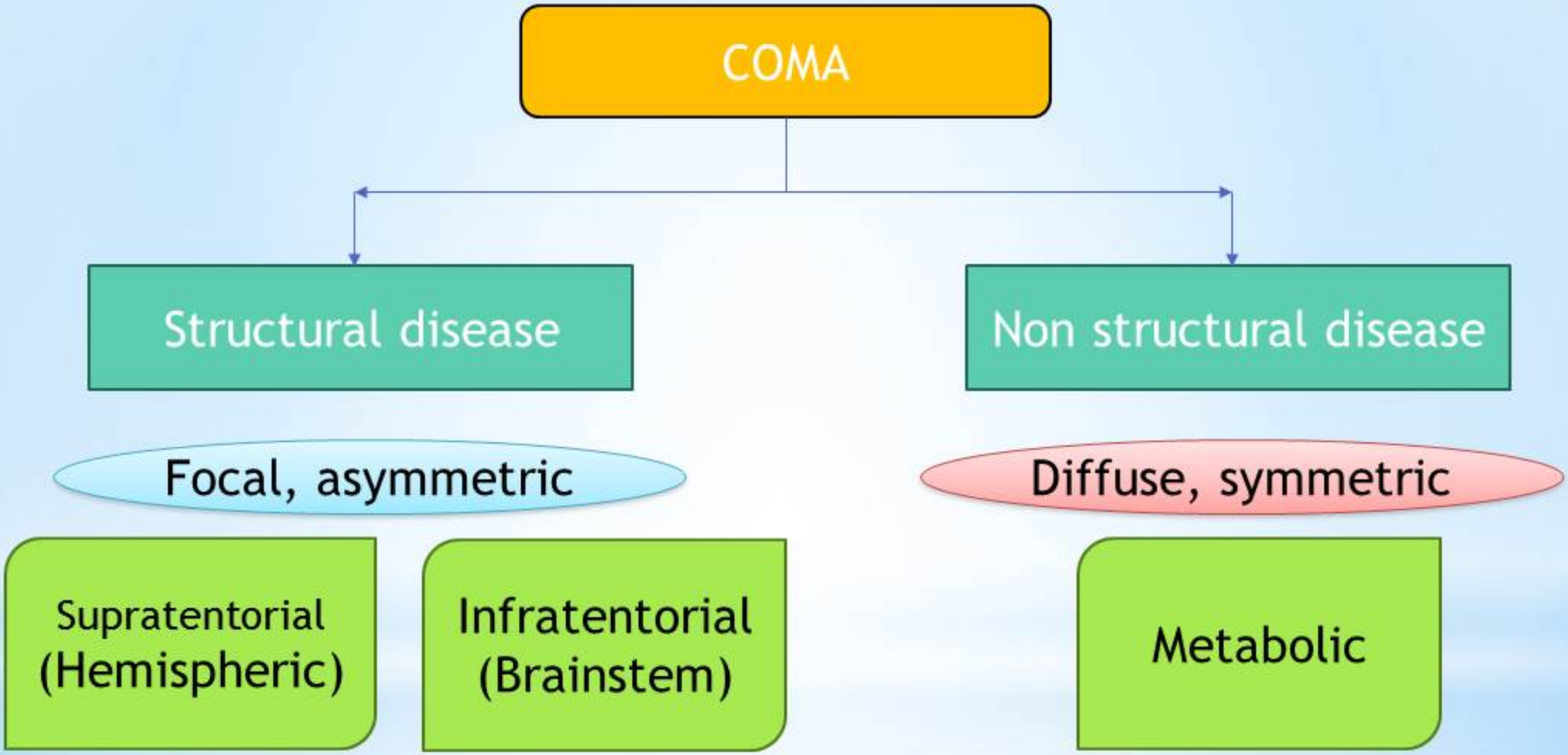
* Differential diagnosis

- Locked in state
- Akinetic mutism
- Persistent vegetative state
- Catatonic stupor
- Pseudocoma
- Abulia

State	Stimulus needed for arousal
Drowsiness	Verbal and light touch
Obtundation	Deep touch
Stupor	Vigorous, painful, or noxious stimulation

* Disorders of consciousness

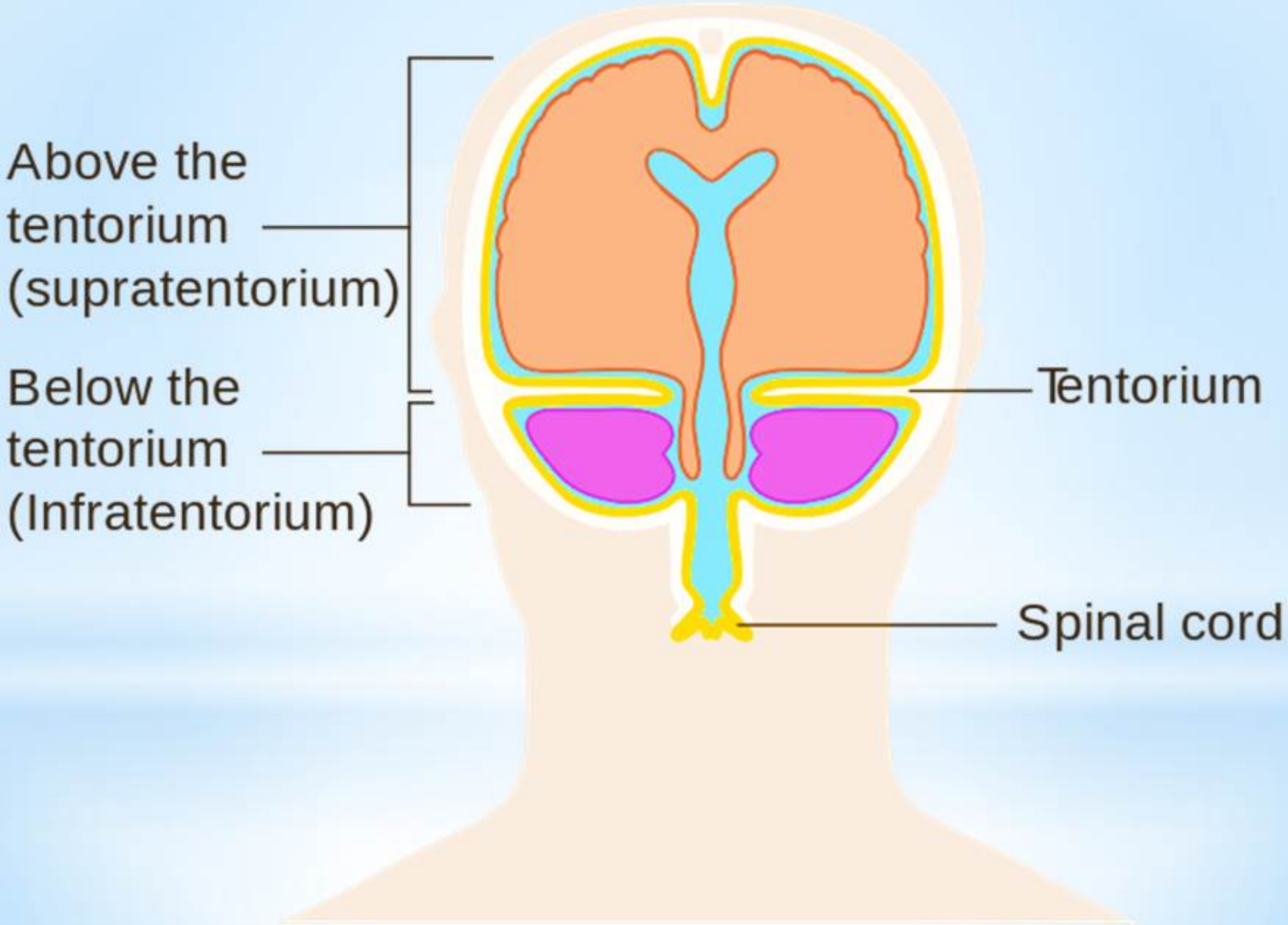
	Arousal	Awareness	Sleep-wake	Motor	Resp
Brain death	-	-	-	-	-
Coma	-	-	-	Non purposeful	Abn, variable
Vegetative state	+	-	+	Non purposeful	+
Min consciousness state	+	Partial	+	Intermit purposeful	+
Akinetic mutism	+	Partial	+	Paucity	+
Delirium	+	Partial	+	Normal	+
Locked in	+	+	+	Quadriplegia, anarthria	Normal


COMA
Structural disease
Non structural disease
Focal, asymmetric
Diffuse, symmetric
Supratentorial (Hemispheric)
Infratentorial (Brainstem)
Metabolic

* Classification of Coma

* Supratentorial lesions cause coma by either widespread bilateral disease, increased intracranial pressure, or herniation.
* Infratentorial lesions involve the RAS, usually with associated brainstem signs
* Metabolic coma causes diffuse hemispheric involvement and depression of RAS, *usually* without focal findings
* Psychogenic

Plum and Posner, 1982

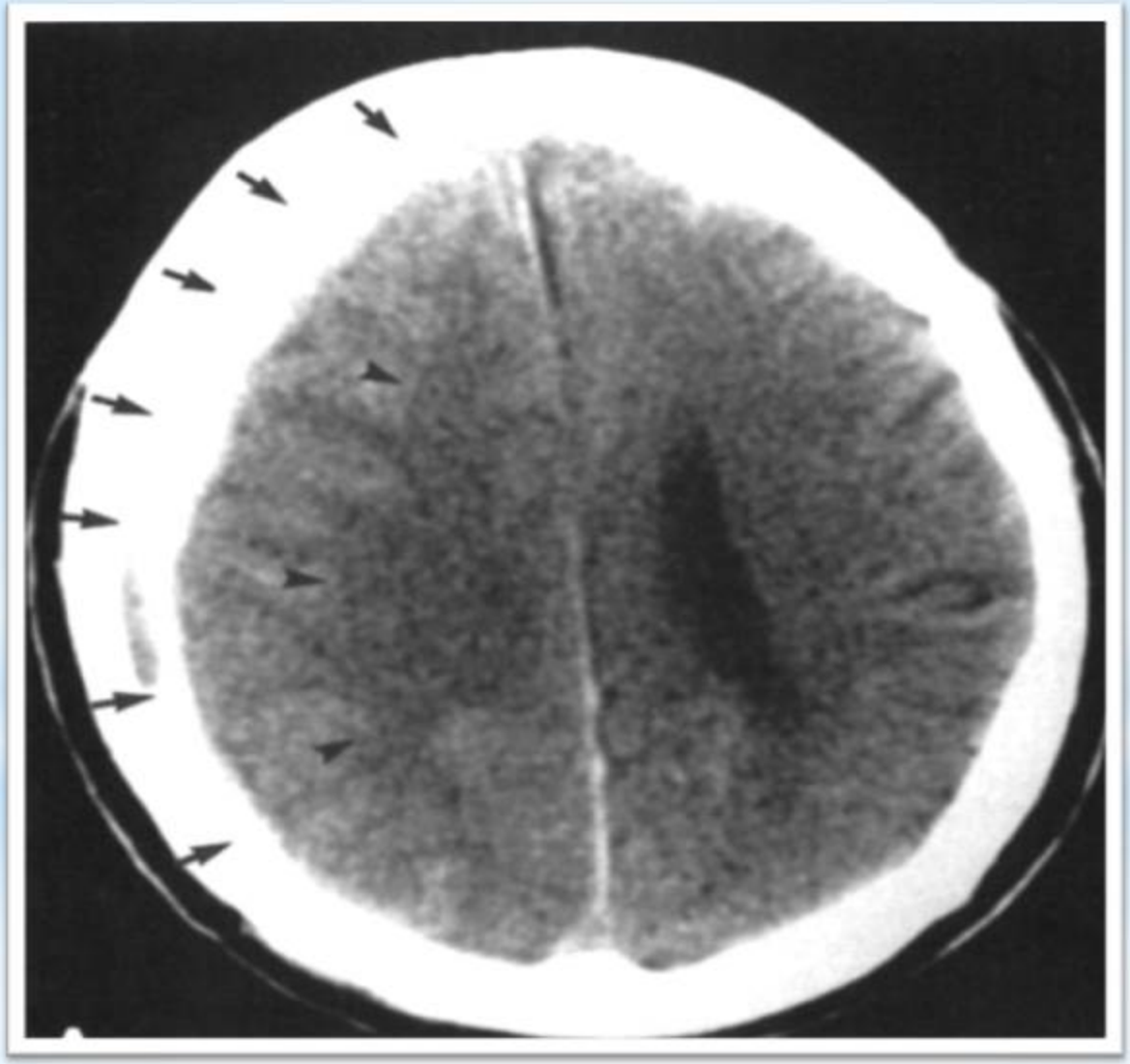

Above the tentorium (supratentorium)
Below the tentorium (Infratentorium)
Tentorium
Spinal cord

Supratentorial Mass Lesions

- Hematoma
- Neoplasm
- Abscess
- Contusion
- Vascular Accidents
- Diffuse Axonal Damage

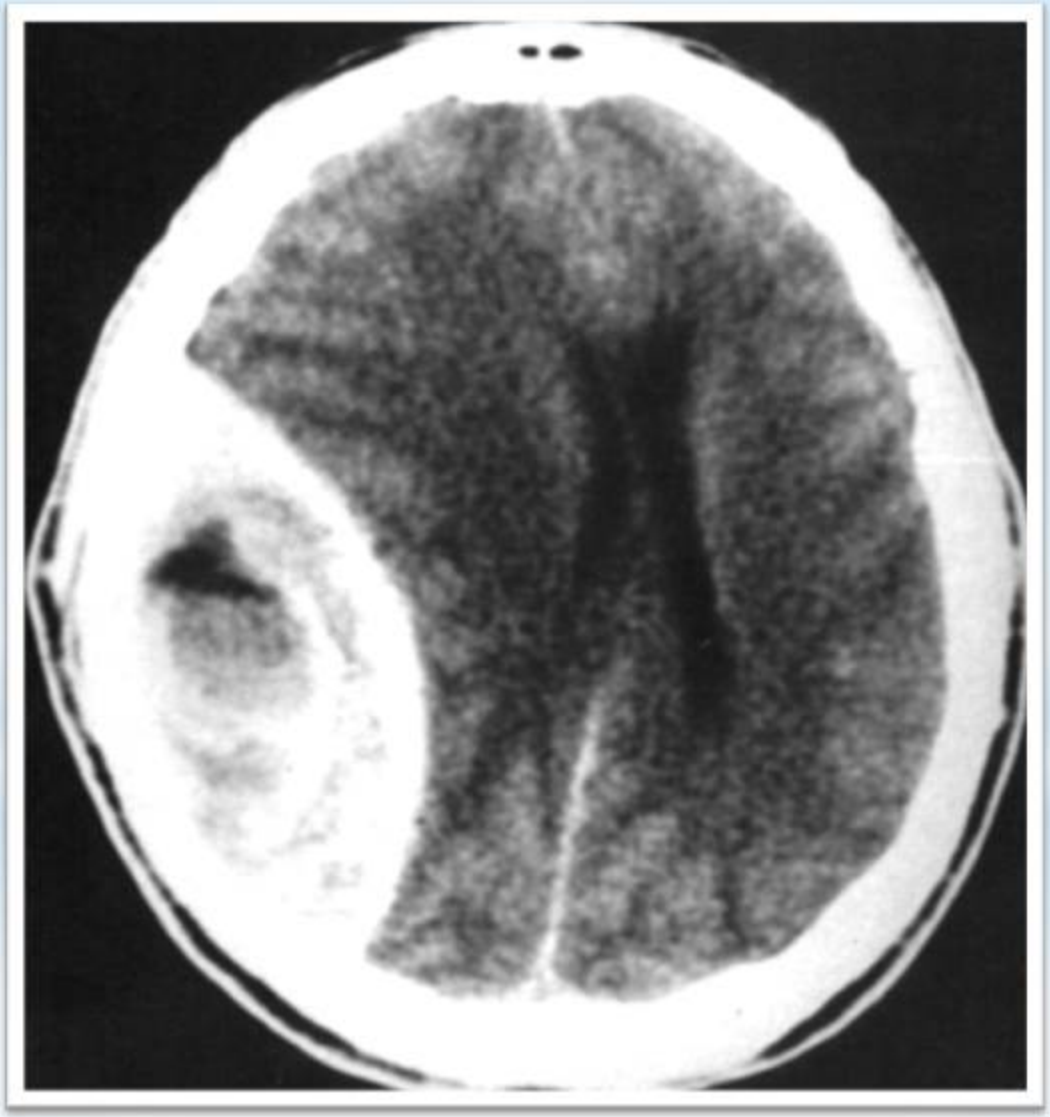
Supratentorial Mass Lesions

Subdural Hematoma

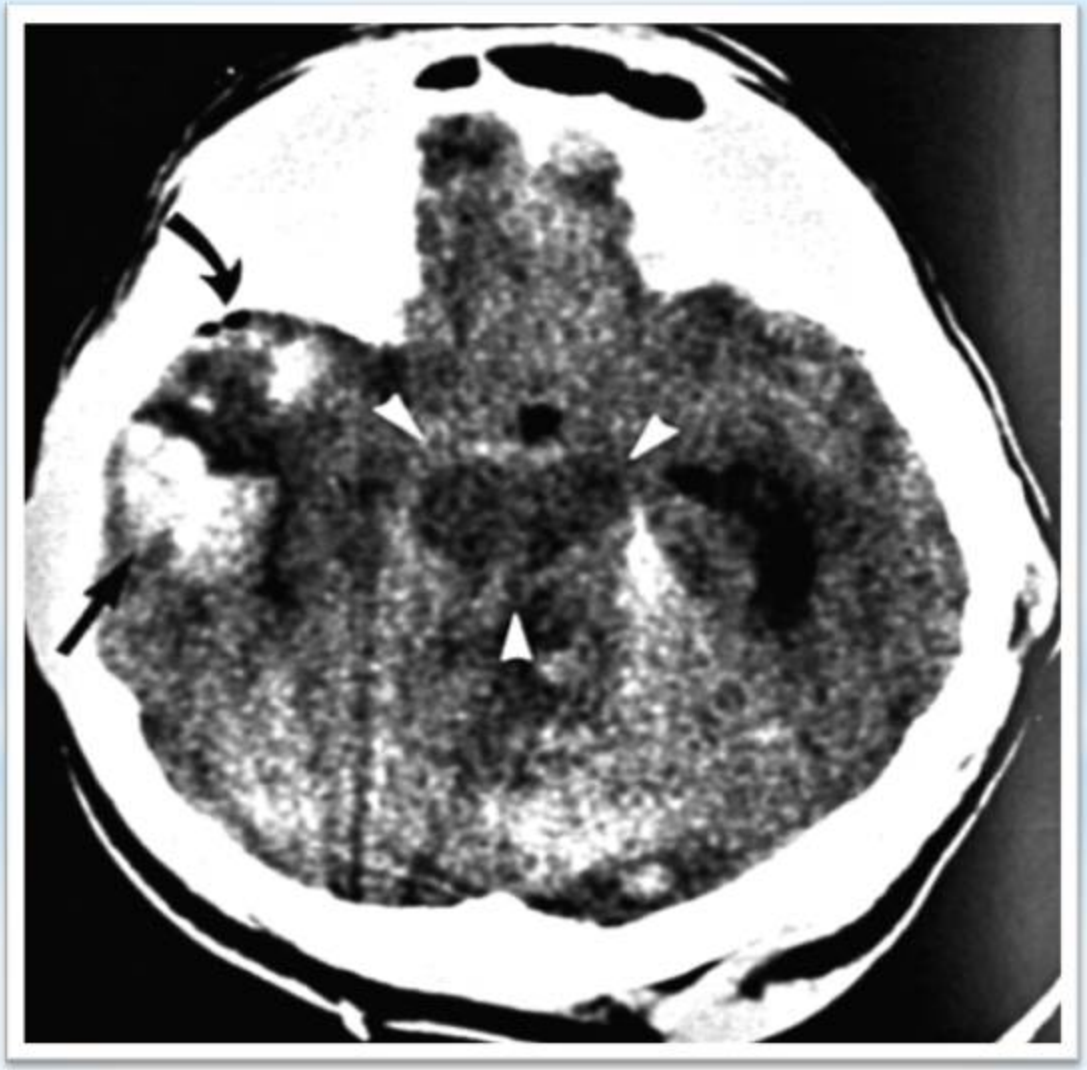


Supratentorial Mass Lesions

Acute epidural hematoma and midline shift

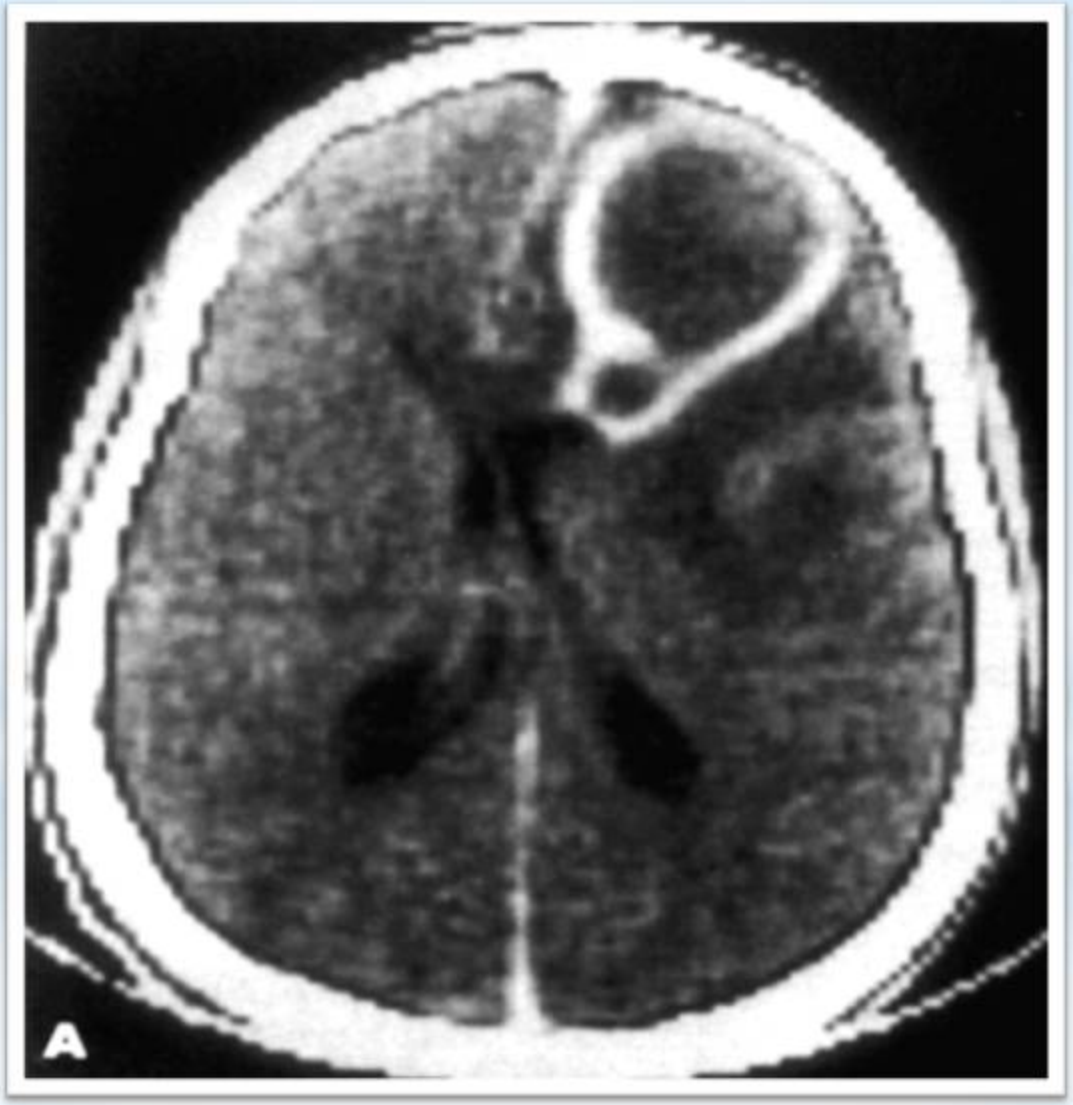


Severe head trauma with basilar skull fracture, right temporal hematoma, cerebral edema, hydrocephalus, and pneumocephalus



Supratentorial Mass Lesions

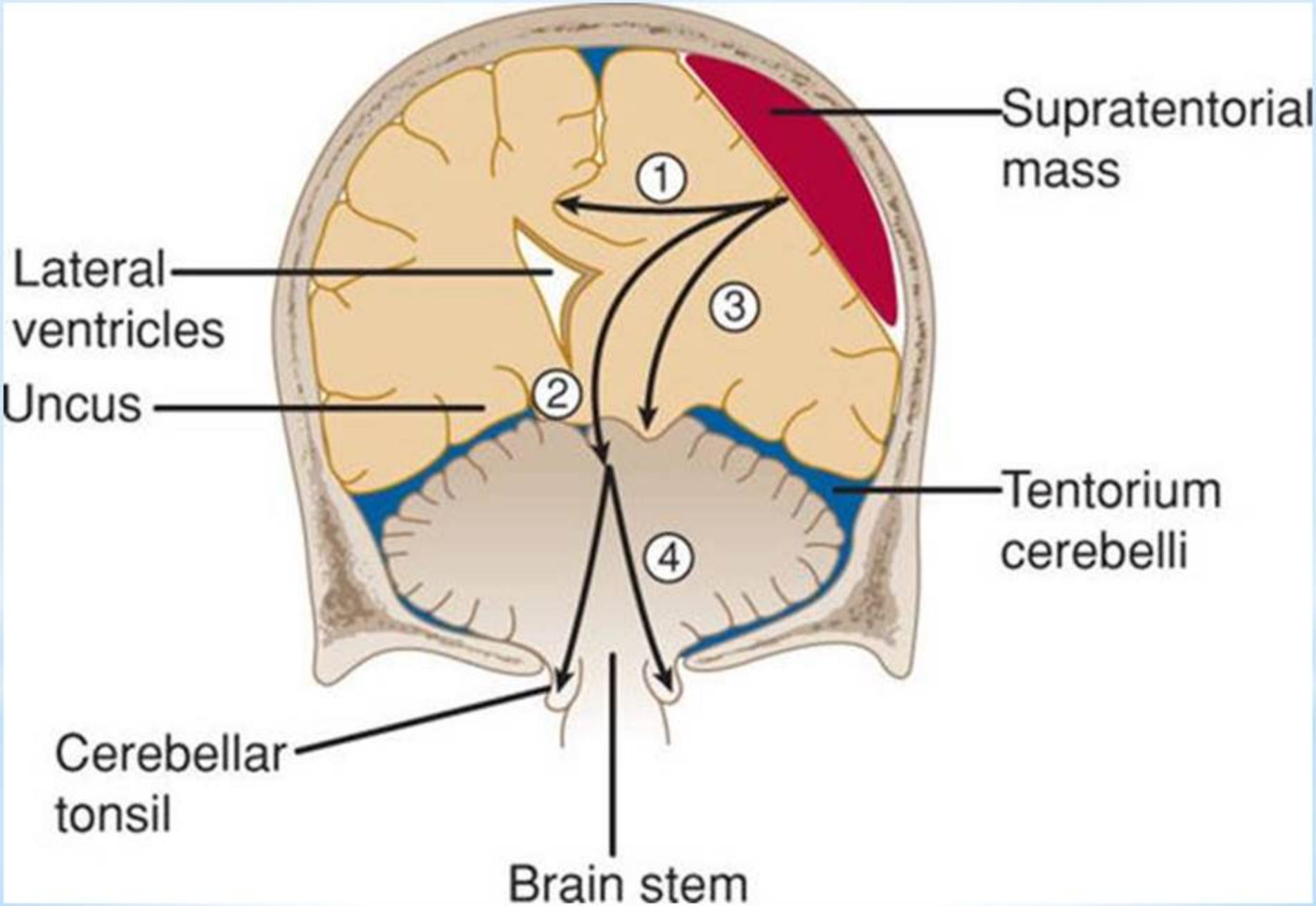
Cerebral Abscess



Supratentorial Mass Lesions

*Pathophysiology

* Altered consciousness is based on
  * Increased intracranial pressure
  * Herniation
  * Diffuse bilateral lesions


Supratentorial mass
Lateral ventricles
Uncus
Tentorium cerebelli
Cerebellar tonsil
Brain stem
1
2
3
4

Herniation Syndromes

Central herniation

- Rostral caudal progression of respiratory, motor, and pupillary findings
- May not have other focal findings

Uncal herniation

- Rostral caudal progression
- CN III dysfunction and contralateral motor findings

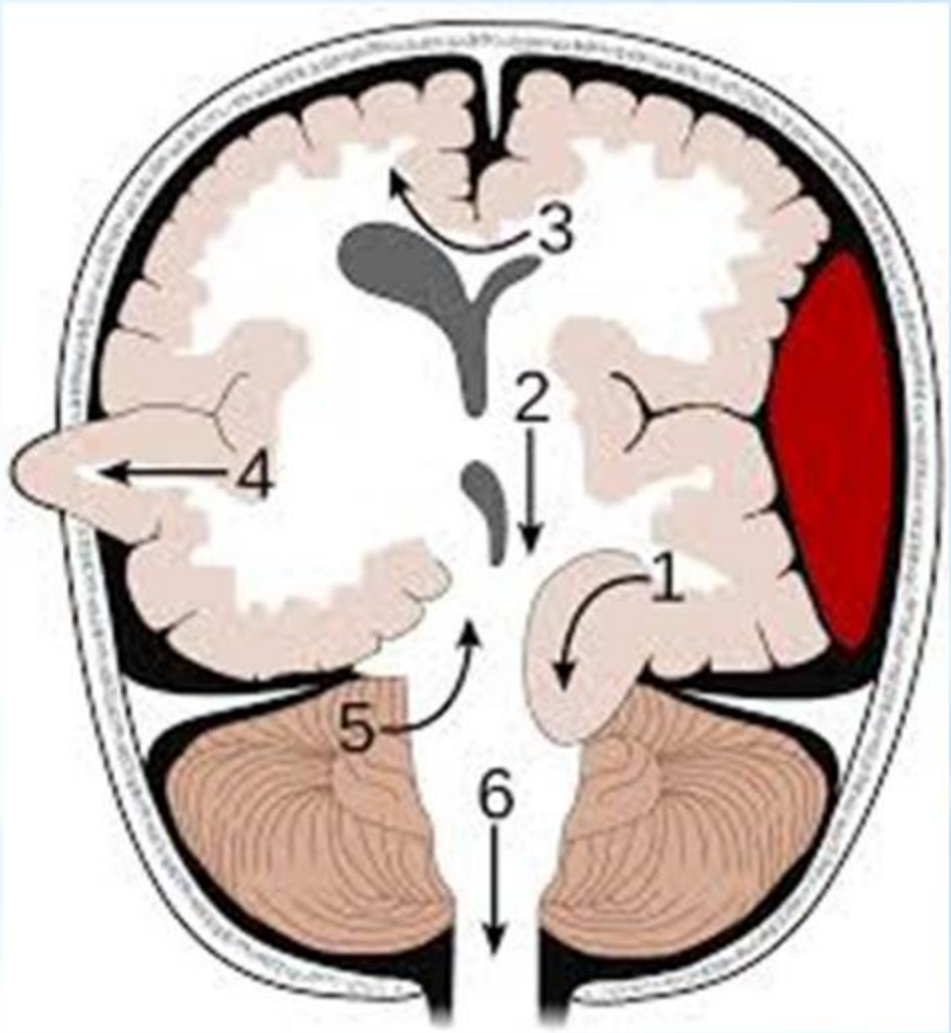
*Brain Herniation

Supratentorial herniation

1. Uncal (transtentorial)
2. Central
3. Cingulate (subfalcine)
4. Transcalvarial

Infratentorial herniation

1. Upward (upward cerebellar or upward transtentorial)
2. Tonsillar (downward cerebellar)



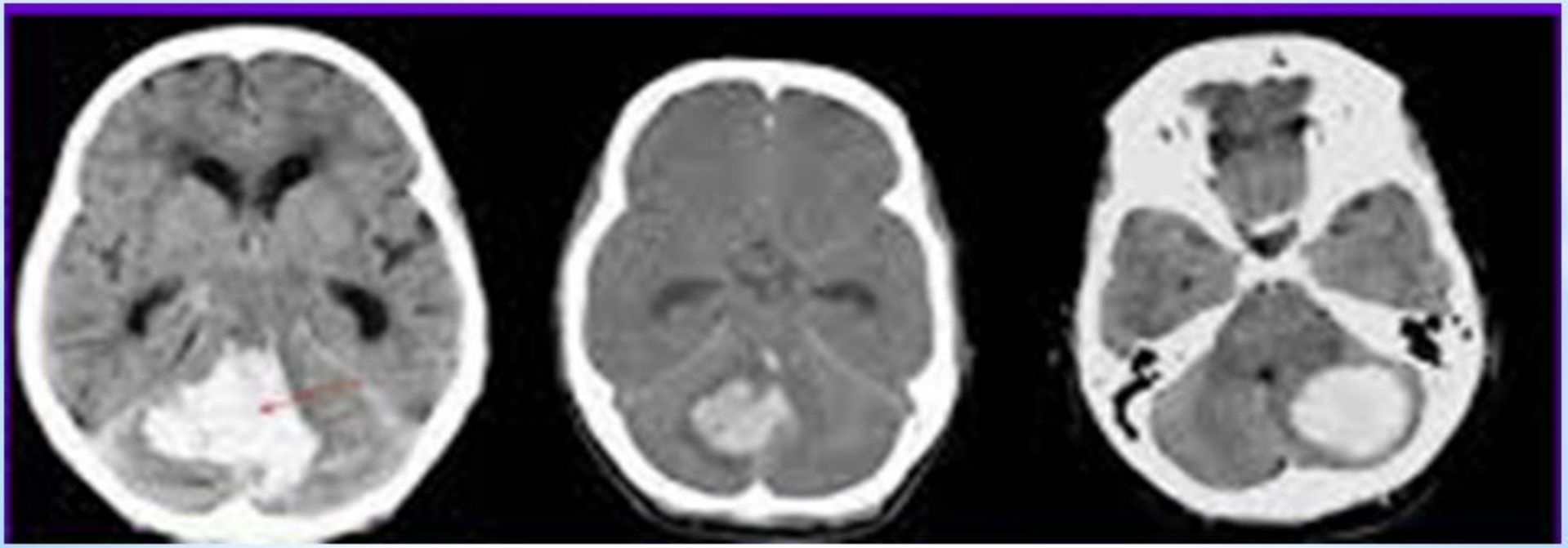
* Herniation syndromes

Uncal herniation	i/l dilated pupil Contralateral hemiparesis Variable impaired consciousness Decerebrate posturing
Central descending transtentorial	Decorticate posturing Midsize fixed pupils Early coma Cheyne Stokes
Central ascending transtentorial	Nausea, stupor
Sub falcine/ Cingulate	Small reactive pupil Headache C/l leg paralysis
Tonsillar	HTN-bradycardia-bradypnoae b/l arm dysesthesia Coma Response arrest

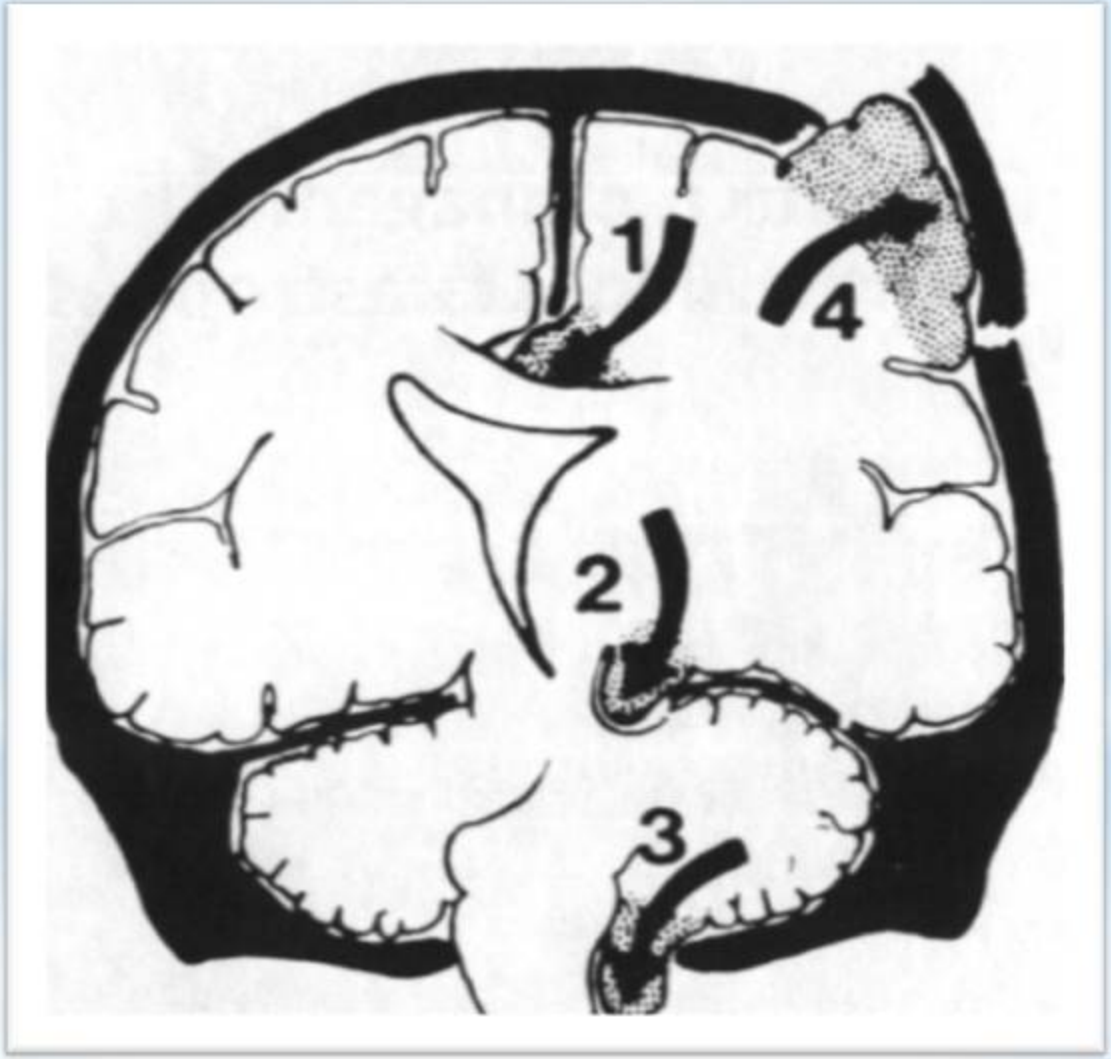
* No features of coning?

*Suspect primary brain stem lesion:

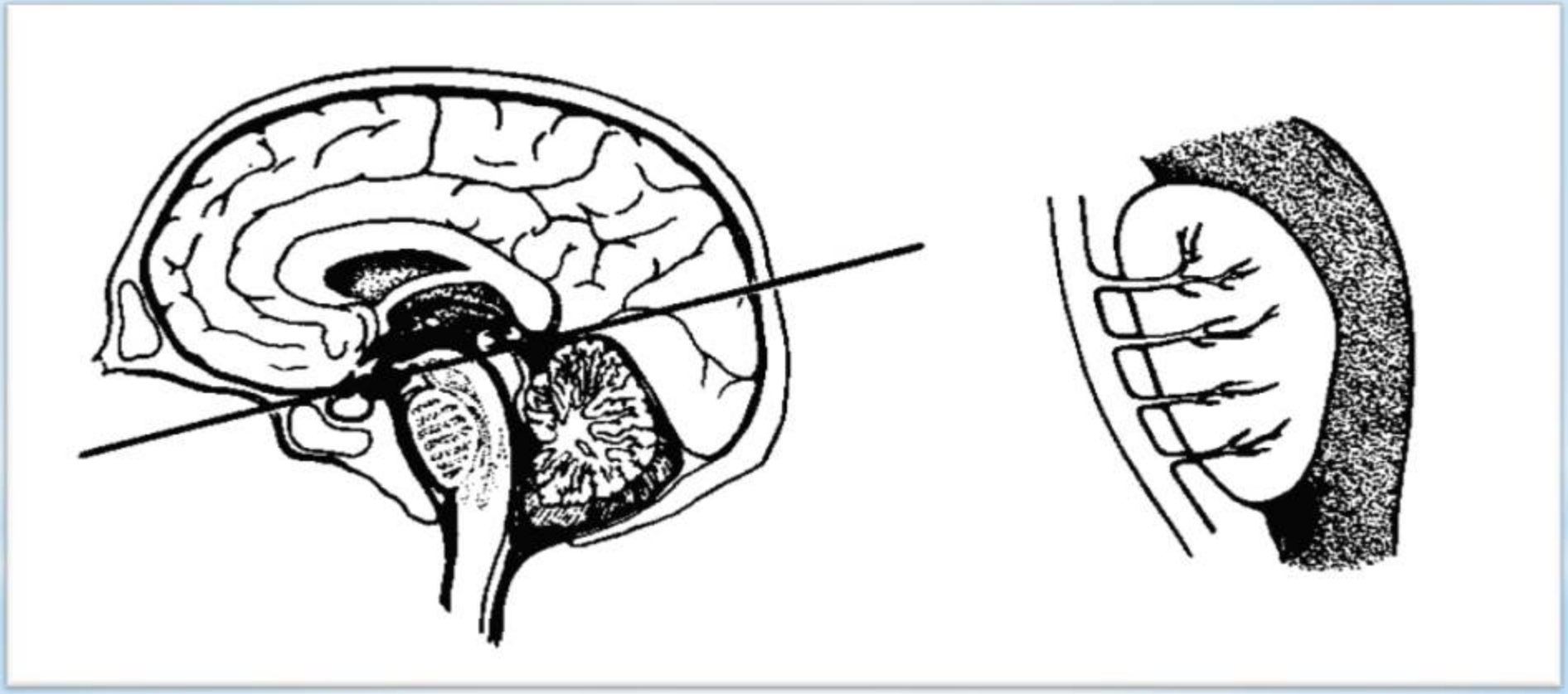
1. **Brain stem infarction or hemorrage**
2. **Cerebellar hemorrage**
3. **Cerebellar infarct with compression**



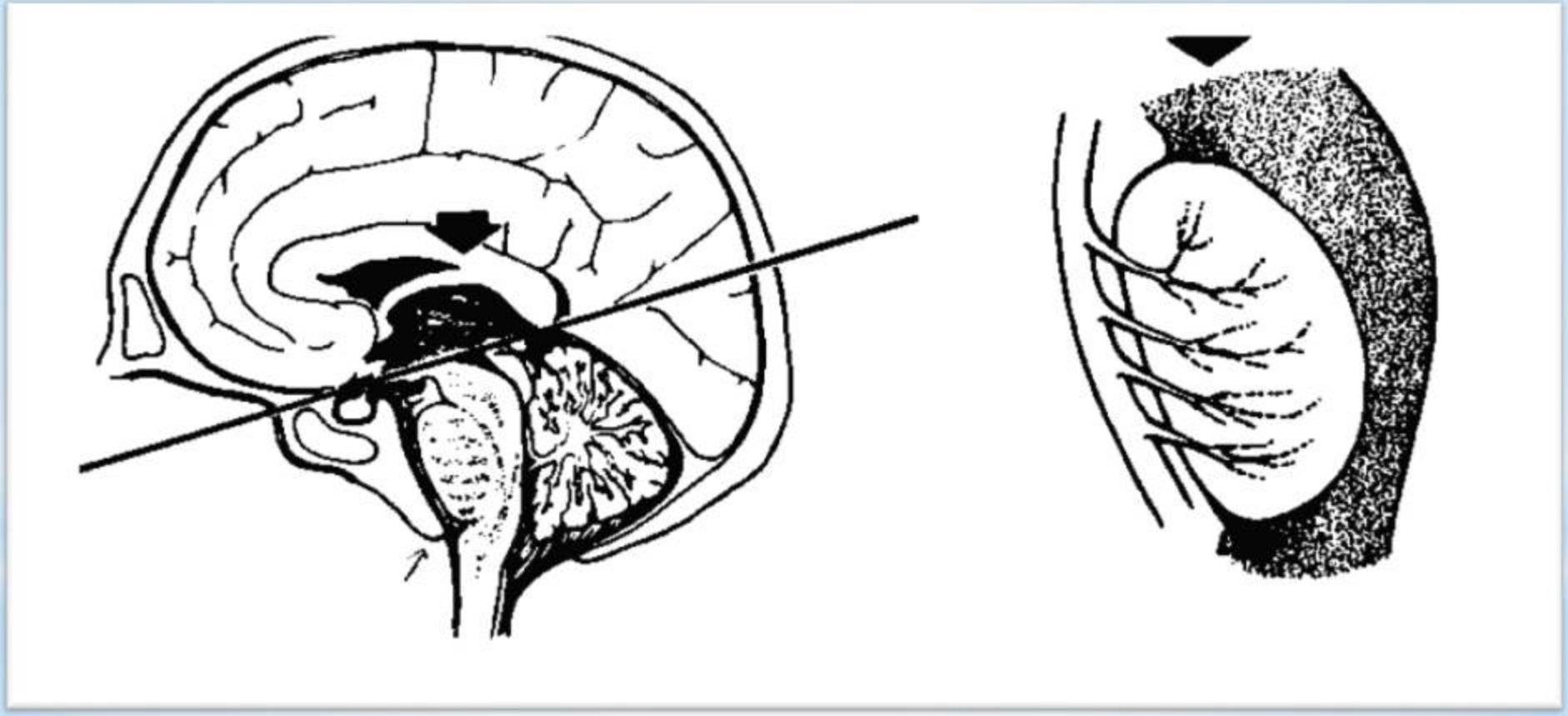
Herniation syndromes



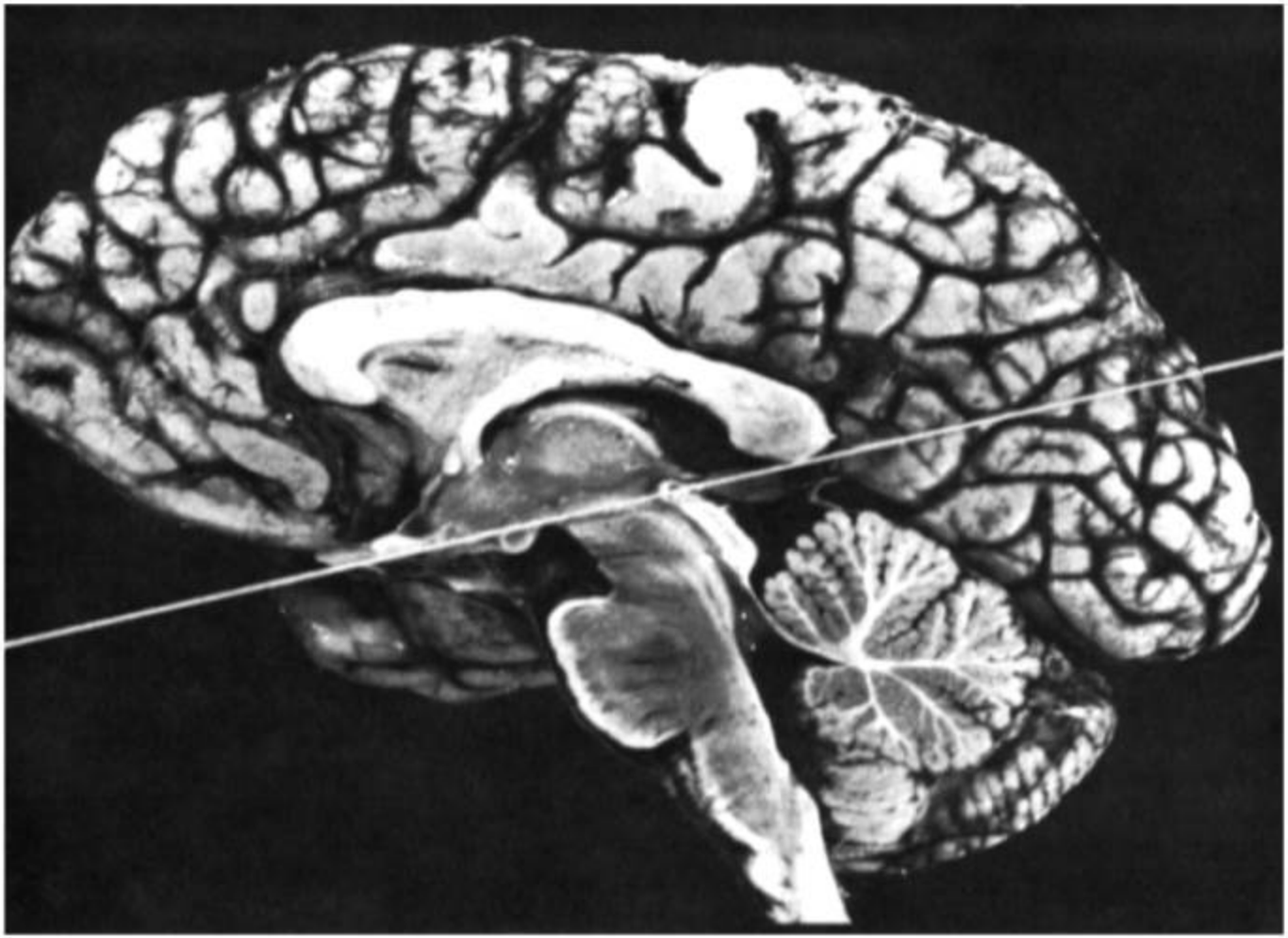
Normal Anatomy



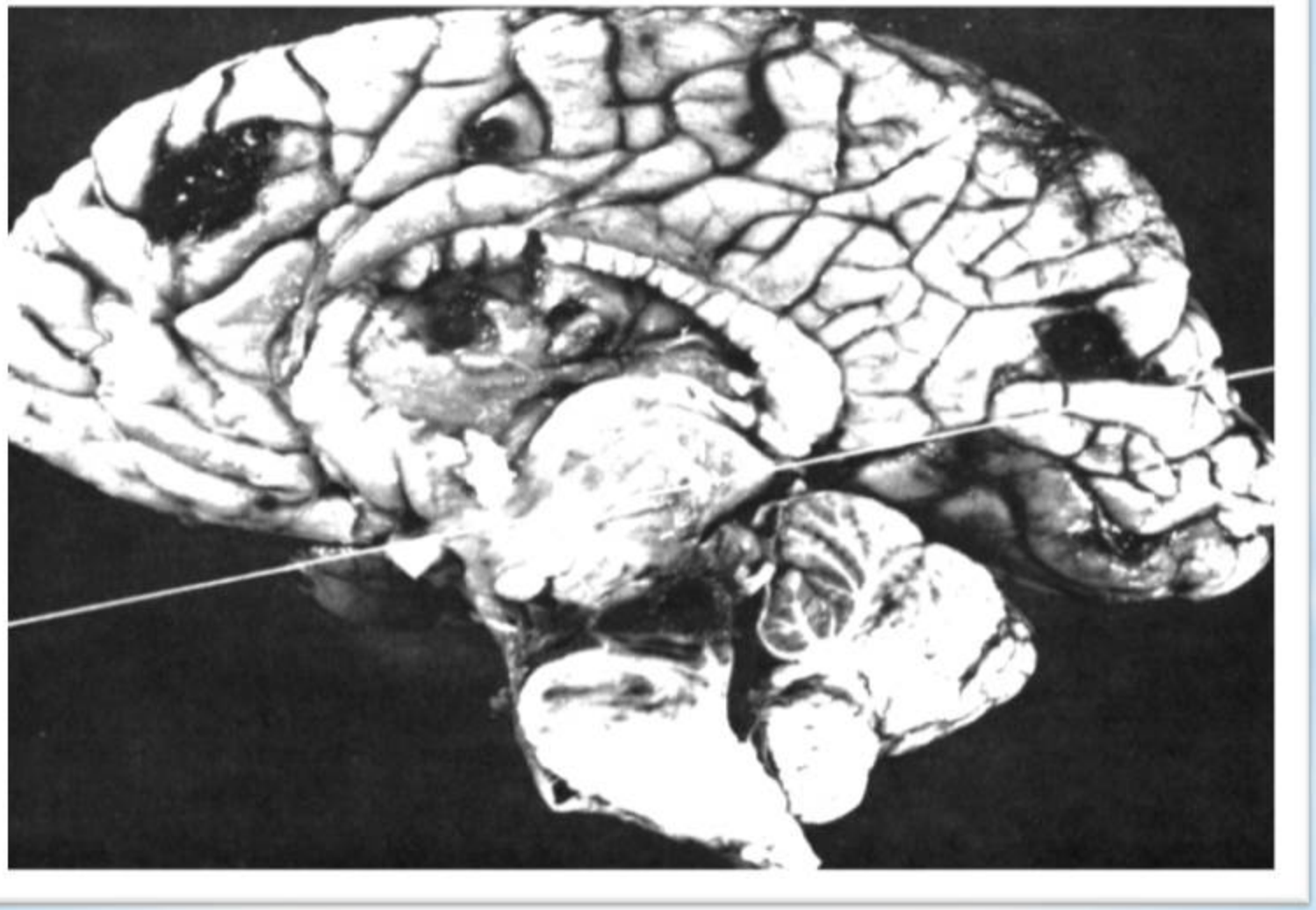
Transtentorial Herniation



Normal Brain



Transtentorial herniation and brain infarction



Supratentorial Mass Lesions

Differential Characteristics

Initiating signs usually of focal cerebral dysfunction

Signs of dysfunction progress rostral to caudal

Neurologic signs at any given time point to one anatomic area - diencephalon, midbrain, brainstem

Motor signs are often asymmetrical

Plum and Posner, 1982

Infratentorial Lesions

- Cause coma by affecting reticular activating system in pons
- Brainstem nuclei and tracts usually involved with resultant focal brainstem findings

* Infratentorial Lesions

Causes of Coma

* Neoplasm
* Vascular accidents
* Trauma
* Cerebellar hemorrhage
* Demyelinating disease
* Central pontine myelinolysis (rapid correction of hyponatremia)

* Infratentorial Lesions

Differential Characteristics

* History of preceding brainstem dysfunction or *sudden onset* of coma
* *Localizing brainstem signs* precede or accompany onset of coma and always include oculovestibular abnormality
* *Cranial nerve palsies* usually present
* *"Bizarre" respiratory patterns* common, usually present at onset of coma

Plum and Posner, 1982

Metabolic Coma
Etiology

- Respiratory
  - Hypoxia
  - Hypercarbia
- Electrolyte
  - Hypoglycemia
  - Hyponatremia
  - Hypercalcemia
- Hepatic encephalopathy
- Severe renal failure
- Infectious
  - Meningitis
  - Encephalitis
- Toxins, drugs

* Metabolic Coma
Differentiating Features

* Confusion and stupor commonly precede motor signs
* Motor sings are usually symmetrical
* Pupillary reactions are usually preserved
* Asterixis, myoclonus, tremor, and seizures are common
* Acid-base imbalance with hyper- or hypoventilation is frequent

Plum and Posner, 1982

*PART II: Work with patient

* How to logically approach ?

STEP I: Rule out coma mimics

STEP II: Whether bispheric or hemispheric problem

STEP III: If its bispheric, is it primary or secondary (coning?)

STEP IV: If its hemispheric, is it structural or metabolic

STEP V: Investigation, Management & Prognosis

*History of patient condition

*Extremely important!!!

*Often difficult and sometimes impossible to obtain!

•Rapidity	Acute vs subacute
•Symptoms prior to coma	Trauma, head ache, fever, seizure, chest pain, breathlessness etc
•History of neurological deficits	
•Past history	DM, HTN, alcoholism, seizure disorder, drug abuse, depression or suicidal attempts etc.
Drug history	

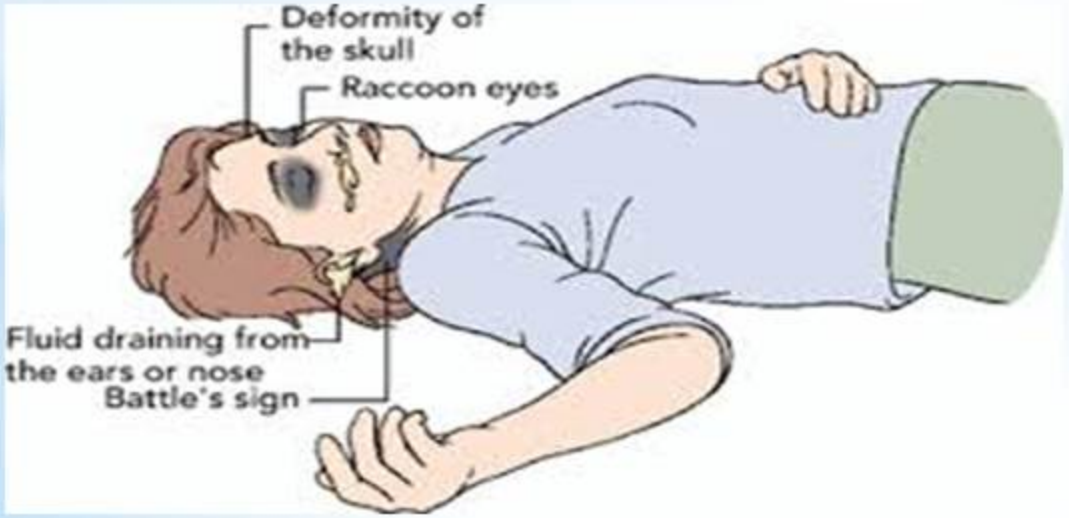
*General Physical Examination

*Appearance

*Evidence of trauma

*Evidence of hepatic or renal dysfunction.

*Incontinence & tongue injury → Seizure



* General physical examination

Needle tracks	Drug overdose
Cyanosis	Hypoxia, cardiac disease, cyanide
Cherry red	Carbon monoxide intoxication
Icterus	Hepatic encephalopathy, hemolysis
Pallor	Anemia, hemorrhage, shock, vasomotor syncope
Petechiae	Disseminated intravascular coagulation, meningococcemia, drugs, fat embolism
Purpuric rash	Meningococcemia, Rocky Mountain spotted fever (RMSF) and others

General physical examination

Maculopapular rash	Toxic shock syndrome, SBE, SLE, and others
Bullous lesions	Drug overdose, especially barbiturates
Flushing, erythema	Polycythemia, fever, alcohol intoxication
Bruises	Trauma, coagulopathy
Sweating	Fever, hypoglycemia

* Blood pressure

Hypertension	Hypotension
• Stroke • Intracranial hemorrhage • Increased ICP • Hypertensive encephalopathy • Renal disease	• Hypovolemia • Myocardial lnfarction • Intoxication (especially ethanol and barbiturates) • Wernicke's encephalopathy • Sepsis

* Respiration examination

Breath odour??

1. Acetone (DKA)
2. Ethanol (intoxication)
3. Fetor hepaticus
4. Uriniferous (uremia)
5. Garlic odor (arsenic poisoning)
6. Household gas (carbon monoxide)

* Respiration examination

Hyperventilation	Hypoventilation
* Hypoxia * Hypercapnia * Acidosis * Fever * Liver disease * Sepsis * Pulmonary emboli * Toxins * Drugs producing metabolic acidosis * Central neurogenic hyperventilation * Salicylism	* Overdose * Myxedema

* Temperature examination

Fever

* Infection
* Inflammation
* Neoplasms (rare)
* Anticholinergics
* Subarahnoid hemorrage (SAH)
* Hypothalamic lesion
* Heatstroke
* Thyroid storm
* Malignant hyperthermia

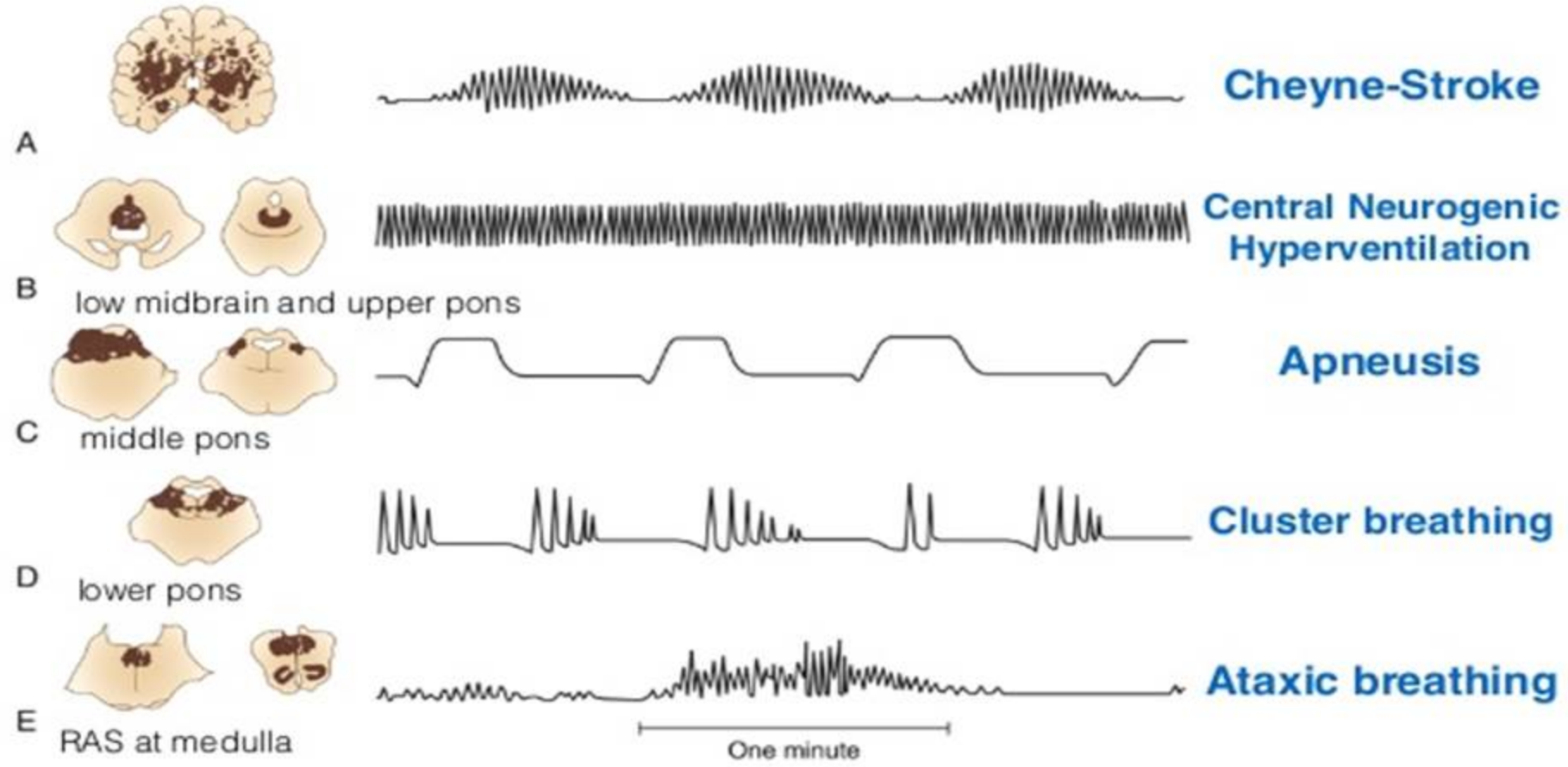
Hypothermia

* Exposure
* Sepsis
* Shock
* Myxedema coma
* Wernicke's encephalopathy
* Drug intoxication (especially barbiturates)
* Hypothalamic lesion
* Hypoglycemia

* Signs of localizing value!

- Respiratory pattern
- Pupillary responses
- Limb movements
- Eye movements

Abnormal Respiratory Pattern



* Systemic examination

Cardiovascular system	•Arrhythmia-Cerebral embolism •Murmur-SBE, embolism
Respiratory system	Pulmonary edema- 1. Neurogenic pulmonary edema 2. CHF 3. Anoxic encephalopathy
Gastrointestinal	Fecal incont- Seizures Stool blood- HE, GI hge
GUT	Hematuria Incontinence
Extremities	Focal seizures

* Eyes movements

Resting position

Spontaneous movts

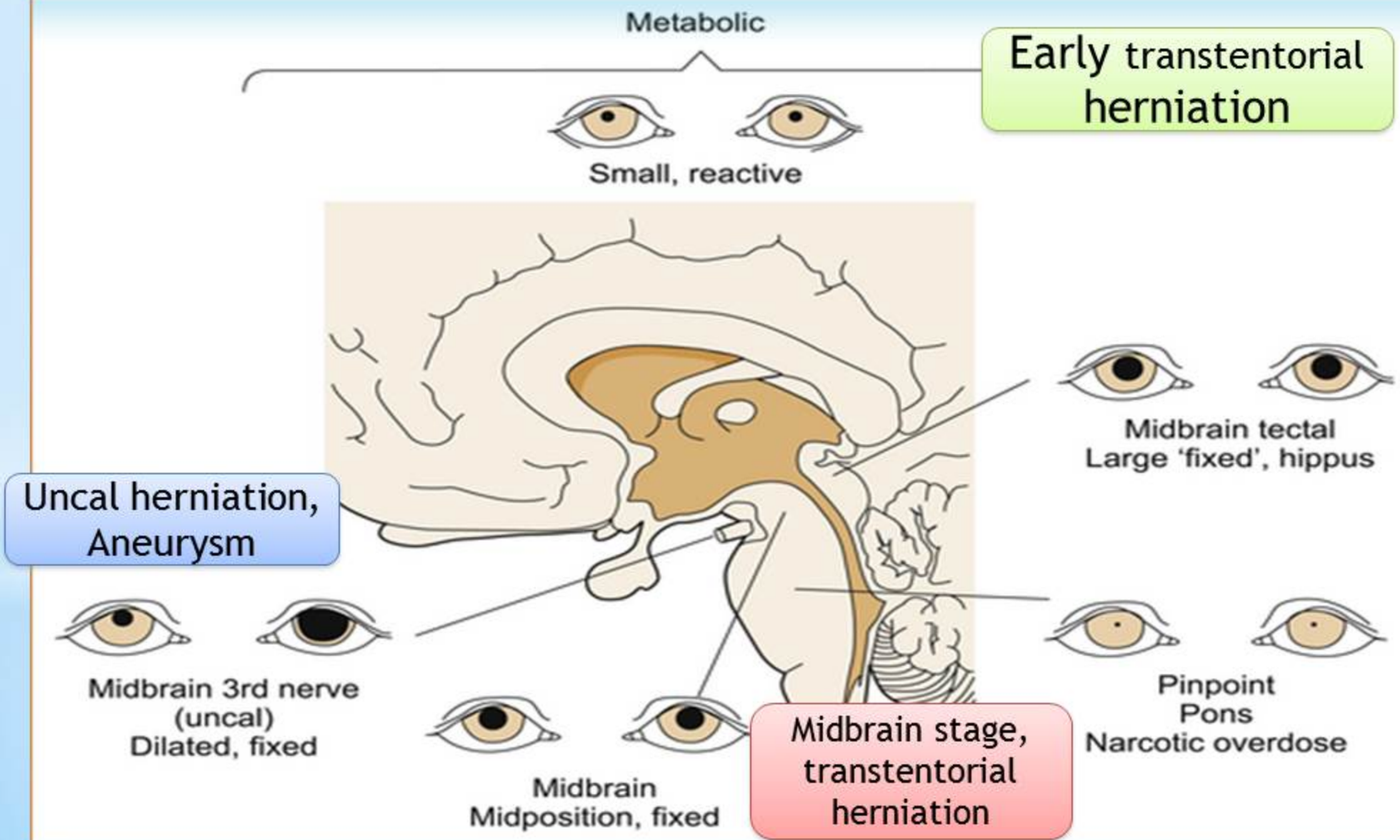
Reflex movts

Lateral gaze	Acute hemispherical, Pontine lesion
Downward gaze	Thalamic lesion, Lesions of pretectum, Hepatic coma, SAH, Hypoxia
Upward gaze	Sleep, Seizure, Syncope, Vermian H'age, Brain stem ischaemia, Brain stem encephalitis, Hypoxic encephalopathy
Horizontal dysconjugate	3,6 CN palsy, INO
Vertical dysconjugate	3 or 4 CN palsy, Skew deviation-posterior fossa lesion

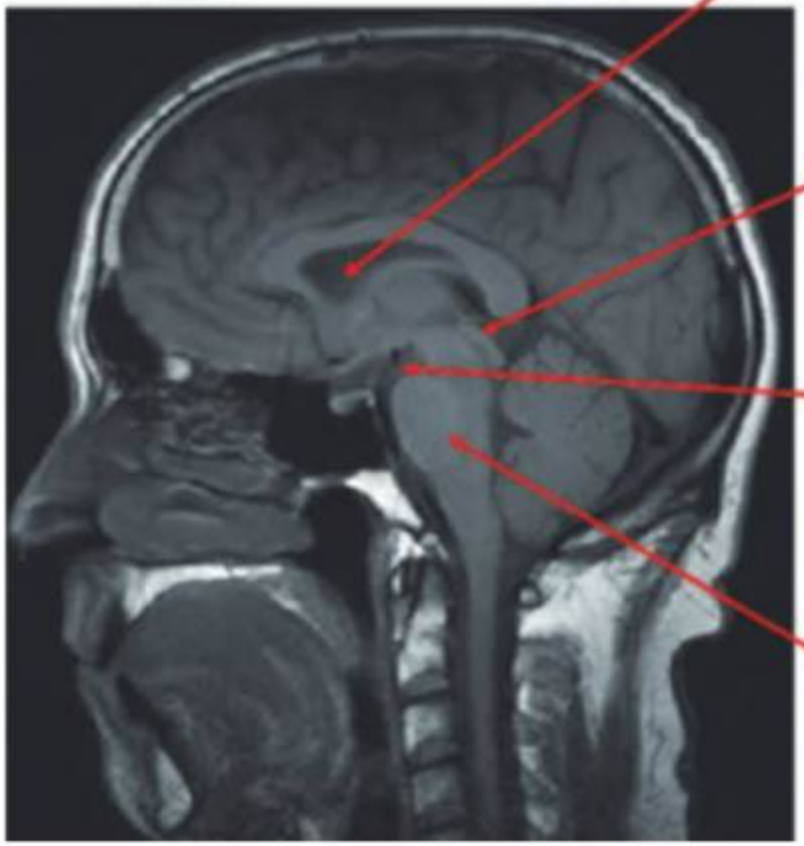
Spontaneous movements

Horizontal roving eye movement	
Nystagmus	Seizures
Ocular bobbing	Pontine lesions, Anoxia
Ocular dipping	Diffuse cerebral damage
Ping Pong gaze	B/l cerebral, post fossa rarely
Nystagmoid jerk of single eye	Middle or lower pons
Vertical myoclonus	Pons

* Pupillary abnormalities



* Pupillary abnormalities



Metabolic: Small pupils, reactive to light

Diencephalon: Small pupils, reactive to light (bilateral Horner syndrome)

Midbrain/Tectum: Midsize pupils, not reactive to light

Third nerve (uncal herniation): Large pupil, not reactive to light

Pons: Pinpoint pupils, reactive to light

* Lateral gaze abnormalities

Hemispheric lesions	Look toward the lesion (away from paretic side) In seizures, briefly away from the lesion Preserved OC, OV reflexes
Brainstem (Lower Pons)	Look toward the paretic side Absent OC,OV reflex
Toxic/ Metabolic	Impair vertical & horizontal movts

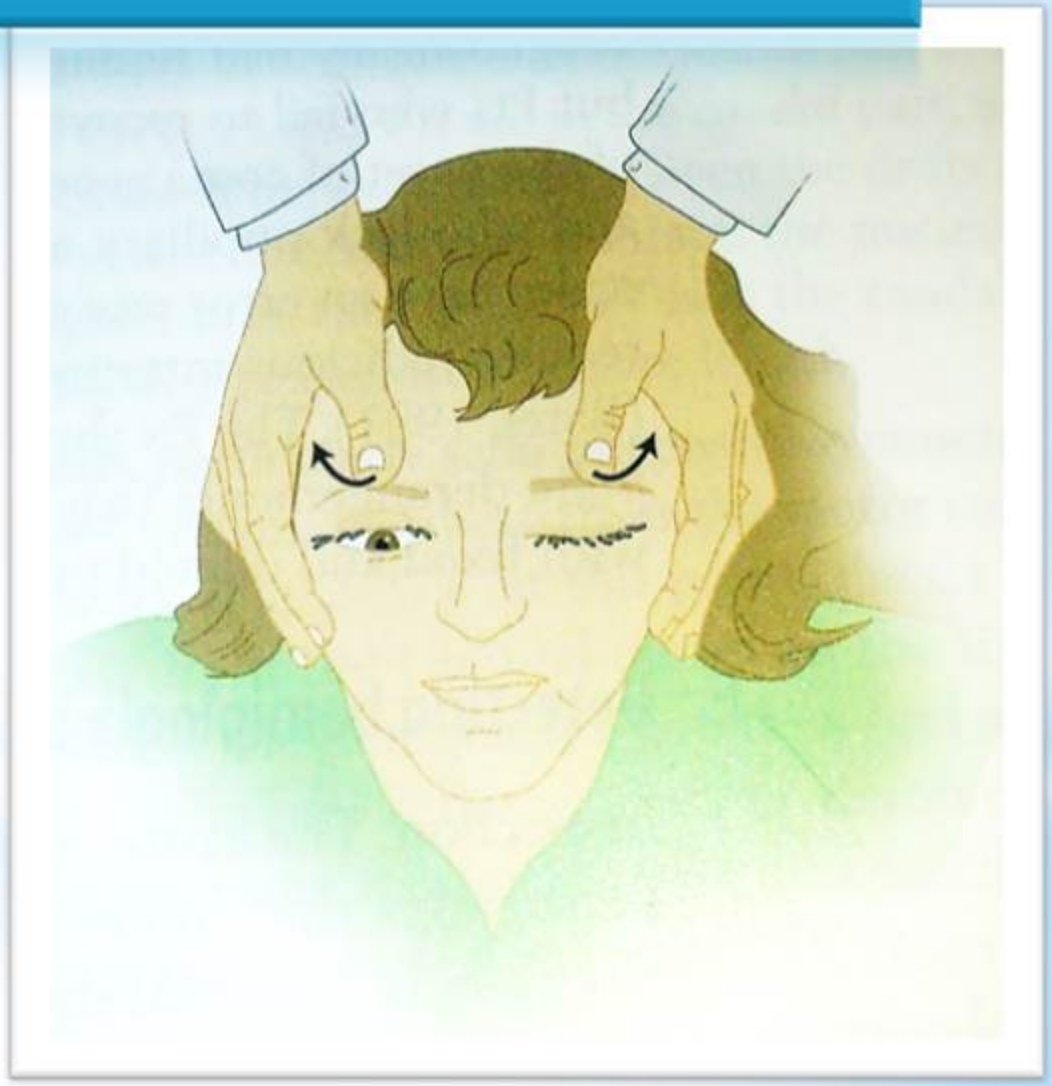
* Rest of cranials

-Fundoscopy

-V: blink

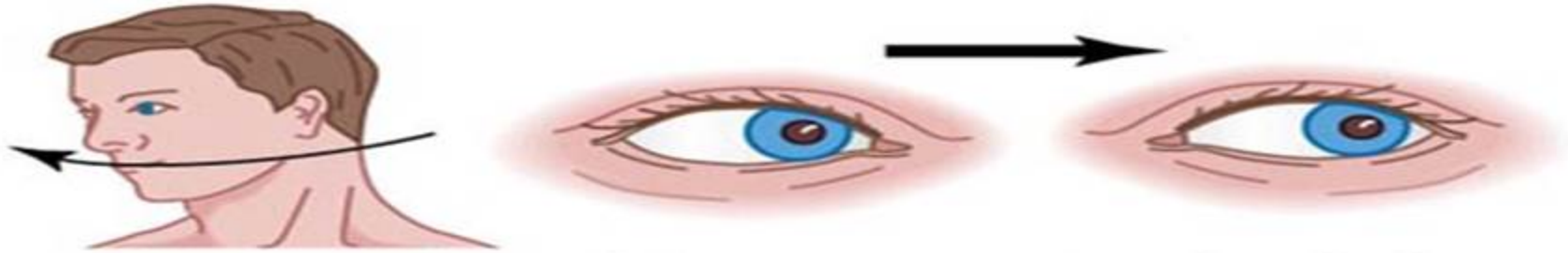
-VII: cheeks and Eyes: Eyelid release test

-Gag



* Oculocephalic reflex(Doll's eye)

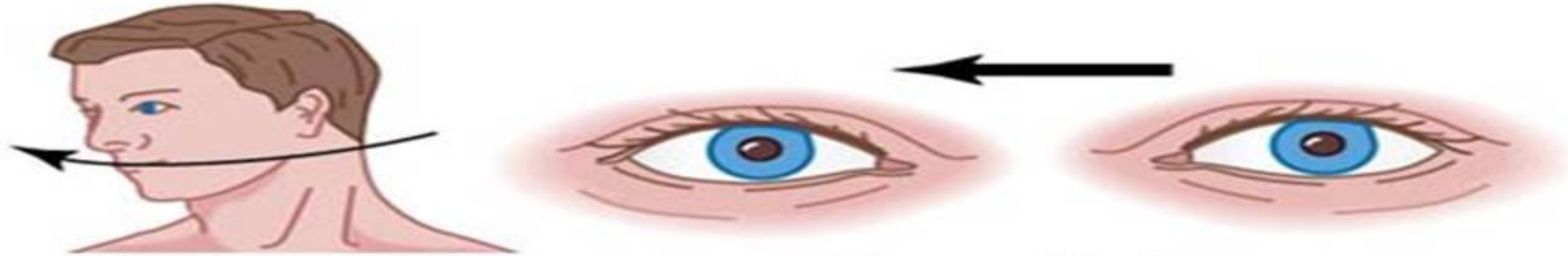
Normal (reflex present)



Head rotated to the right

Eyes move to the left

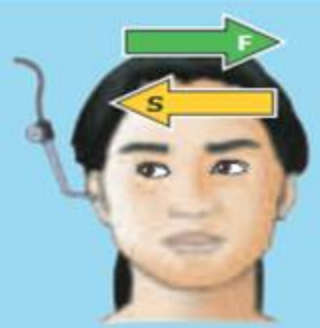
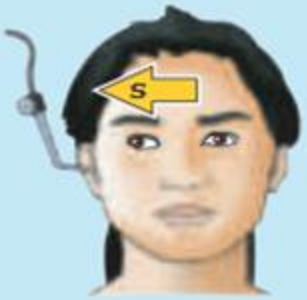
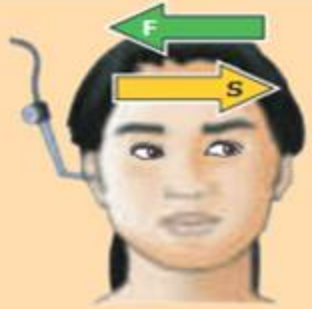
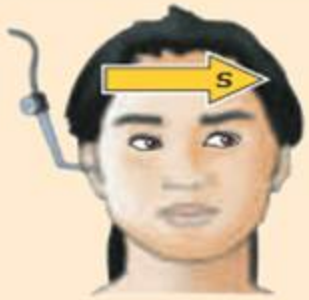
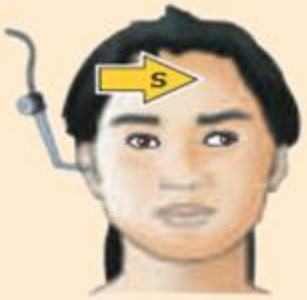
Abnormal (reflex absent)



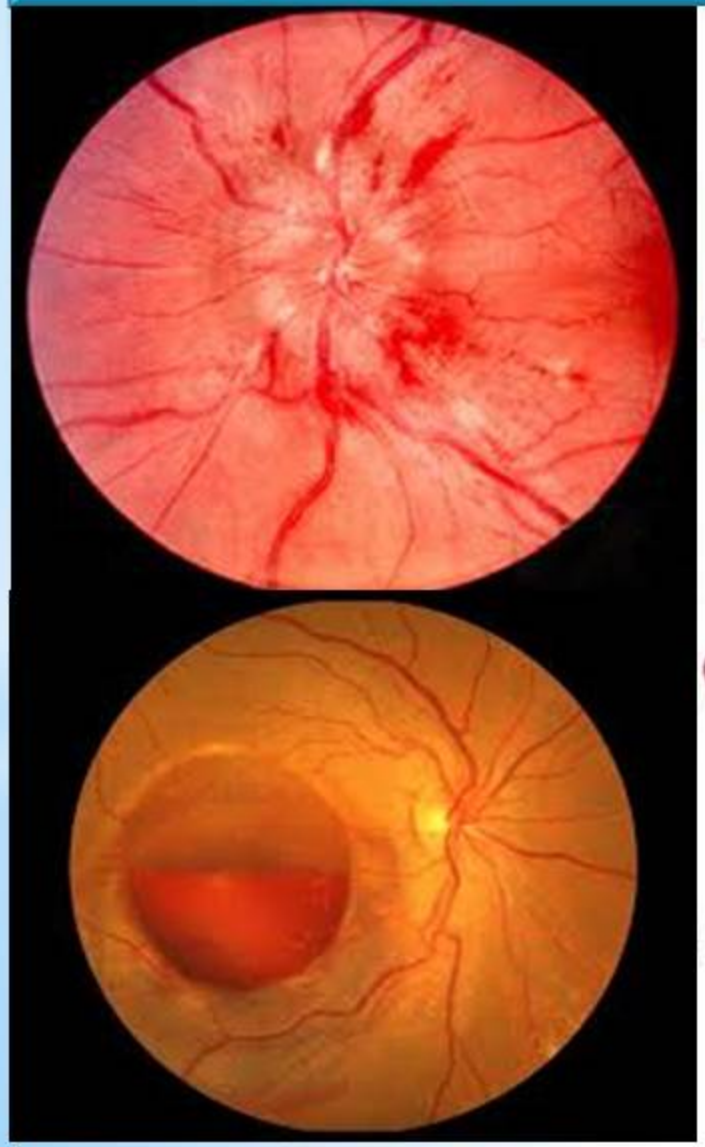
Head rotated to the right

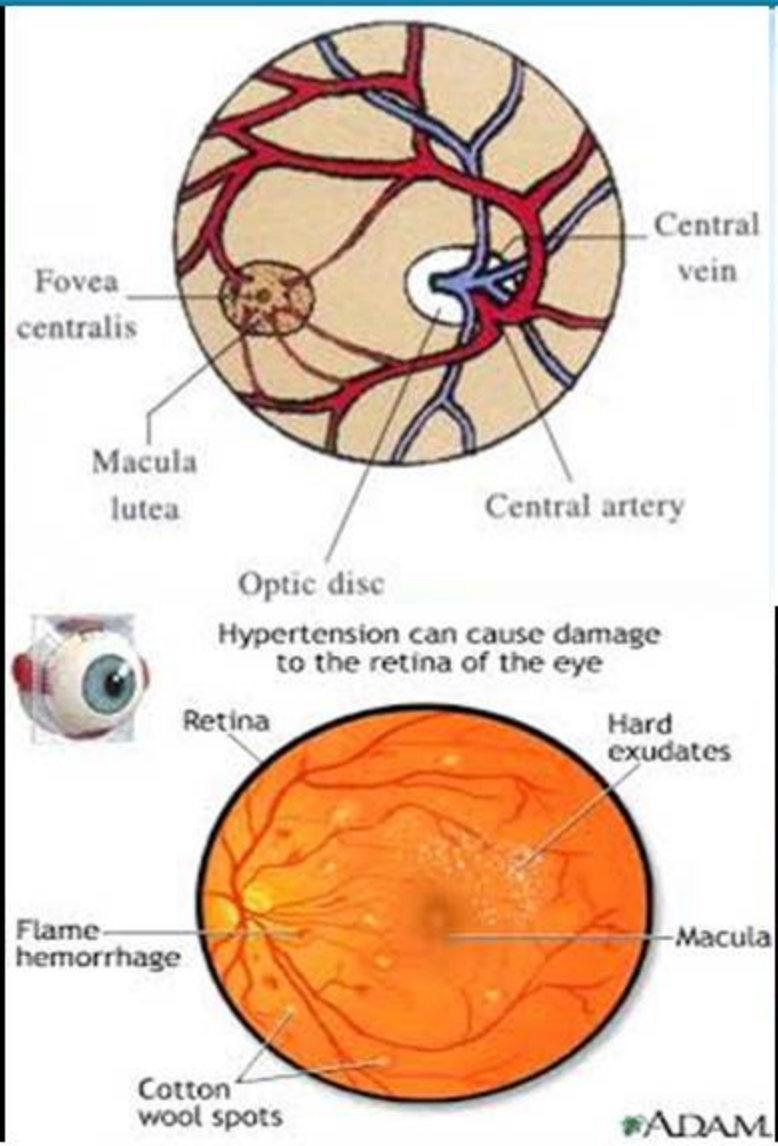
Eyes follow

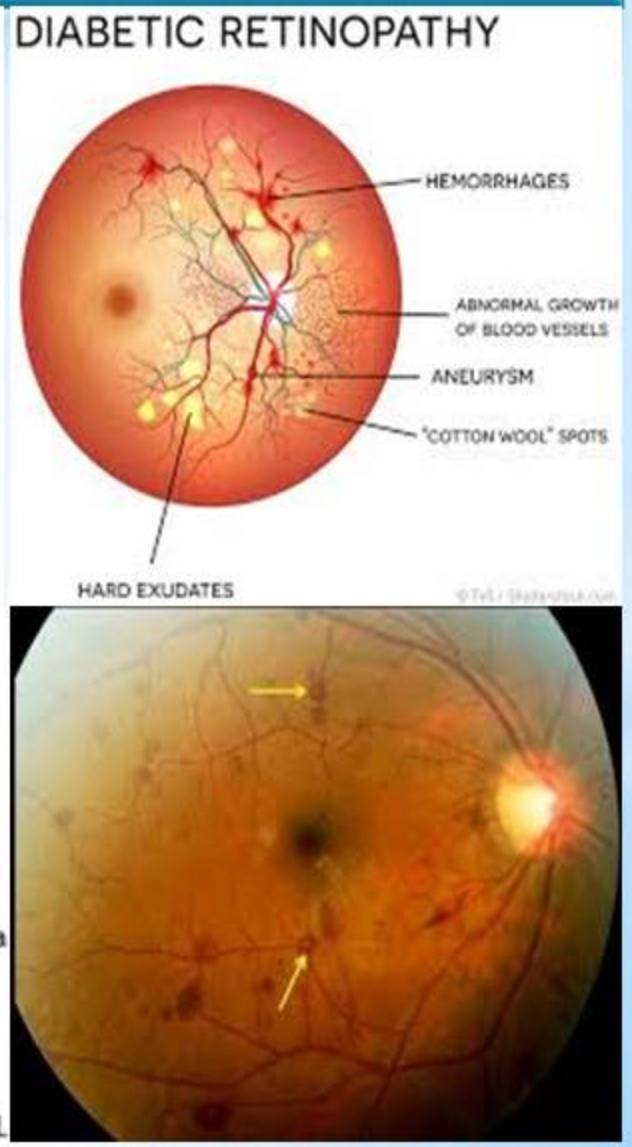
* Oculo-vestibular reflex

Ocular reflexes in conscious patients		Ocular reflexes in unconscious patients	
(1) Normal	(2) Brainstem intact	(3) MLF lesion (bilateral)	(4) Low brainstem lesion
 Cold H_2O	 Cold H_2O	 Cold H_2O	 Cold H_2O
 Warm H_2O	 Warm H_2O	 Warm H_2O	 Warm H_2O

Fundus exam




Fovea centralis
Central vein
Macula lutea
Central artery
Optic disc
Hypertension can cause damage to the retina of the eye
Retina
Hard exudates
Flame hemorrhage
Macula
Cotton wool spots
ADAM.


DIABETIC RETINOPATHY
HEMORRHAGES
ABNORMAL GROWTH OF BLOOD VESSELS
ANEURYSM
"COTTON WOOL" SPOTS
HARD EXUDATES

Approach to the Comatose Patient
Initial Treatment

- **A**irway
- **B**reathing
- **C**irculation
- **ABC** - identify and address life threatening inadequacies
- Treat rapidly progressive metabolic disorders -- hypoglycemia
- Evaluate for intracranial hypertension and imminent herniation and treat

Approach to the Comatose Patient

Priorities

- **ABC**'s are paramount!
- Must prioritize
- Must ensure oxygen and substrate reach CNS and vital organs
- Must address immediately life threatening conditions before addressing CNS

Management of the Comatose Patient

Airway

- Evaluate - is airway patent. Can patient move air without obstruction. Is there trauma or foreign body obstructing airway
- Try chin lift to help open airway -- *protect cervical spine*
- Place airway if indicated - nasal or oral airway, intubation, or surgical airway

Management of the Comatose Patient

Airway

- Intubate (protecting neck) "*anyone who will let you*"
  - Any of the following are adequate criteria
    - GCS < 9
    - Airway not secure or open
    - Respiration not adequate
    - Any significant respiratory failure
    - Uncertainty regarding direction or rate of mental status changes, particularly if constant observation not available (during CT scans, etc..)

Management of the Comatose Patient

Breathing

- Evaluate - is patient moving adequate air, is respiratory rate appropriate, is gas exchange adequate, are breath sounds adequate and symmetrical
- Must assure oxygenation and ventilation
- If intubated don't forget to ventilate
- Identify and immediately treat problems - pneumothorax, airway obstruction, etc..

Management of the Comatose Patient
Circulation

- Is patient in shock?
  - Check pulses, heart rate, blood pressure, perfusion
  - Remember hypotension is *late* sign of shock
- Start treatment for shock
  - Do not restrict fluids in comatose patient with inadequate intravascular volume.
  - Cardiac output and cerebral perfusion are much more important than fluid restriction

Management of the Comatose Patient

Circulation

* Use isotonic solutions and blood, as indicated.
* Do not use hypotonic solutions to treat shock, particularly patients with coma or possible cerebral edema
* Identify life threatening hemorrhage and control it.

* Management of the Comatose Patient
Glasgow Coma Scale

* Glasgow coma scale
  * Provides easily reproducible and somewhat predictive basic neurologic exam
  * This allows rapid assessment and record of baseline neurologic status
  * Allows physician to track neurologic changes over time and multiple examiners

*Glasgow Coma Scale

*Three components. Score derived by adding the score for each component.

- *Eye opening (4 points)
- *Verbal response (5points)
- *Best motor response (6 points)

*Glasgow Coma Scale

- *Eye opening
  - *4 - spontaneous
  - *3 - to speech
  - *2 - to pain
  - *1 - none
- *Verbal Response
  - *5 - oriented
  - *4 - confused conversation
  - *3 - inappropriate words
  - *2 - incomprehensible sounds
  - *1 - none
- *Best Motor Response
  - *6 - obeys
  - *5 - localizes
  - *4 - withdraws
  - *3 - abnormal flexion
  - *2 - abnormal extension
  - *1 - none

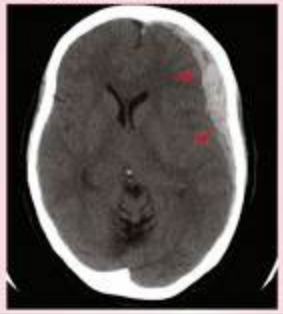
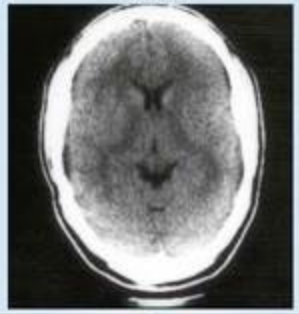
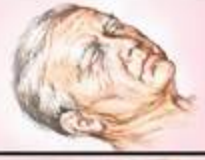
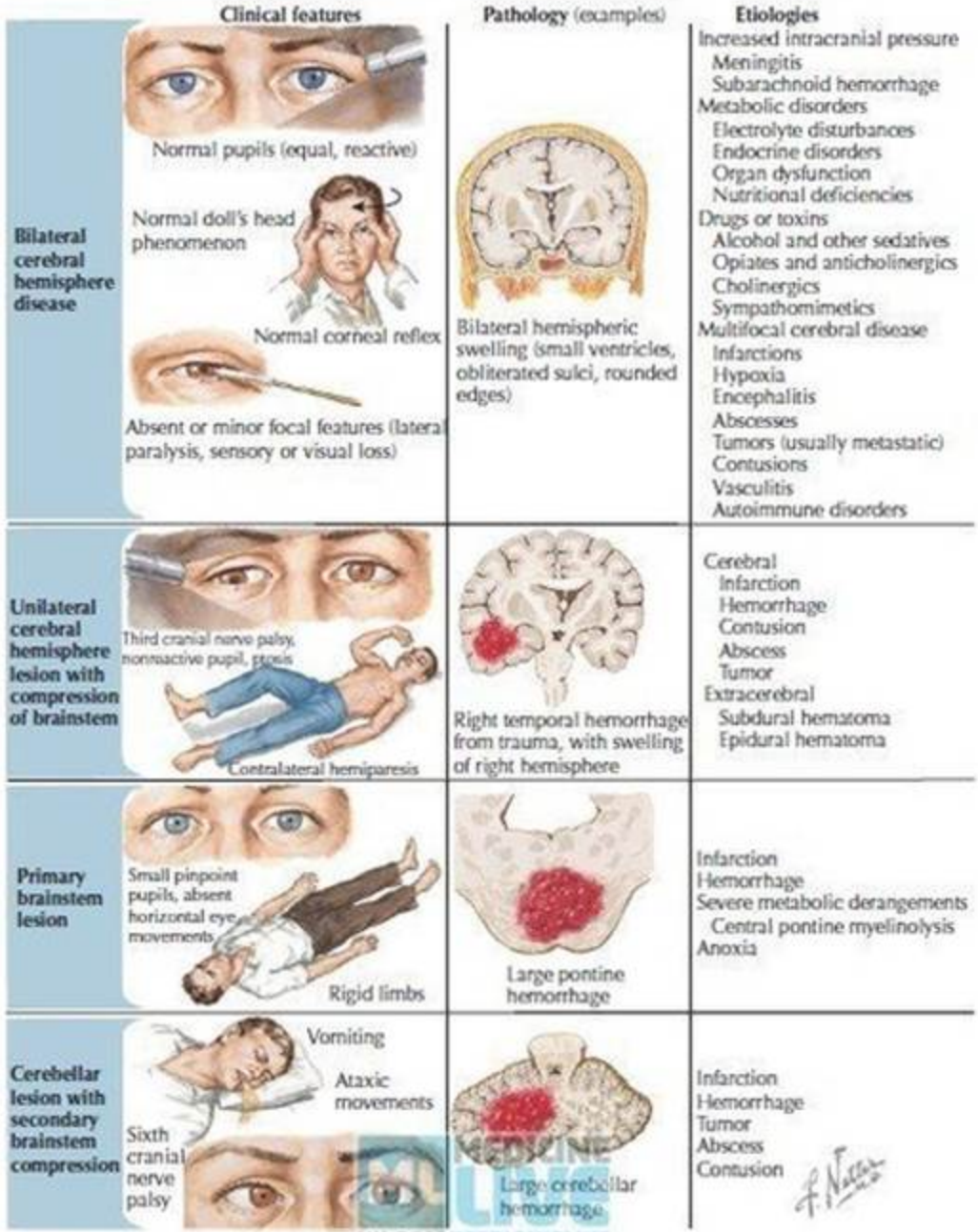
	Poorer	Better
Glasgow Coma Scale	**3–8**	**9–15**
CT scan Axial noncontrast brain CT: Acute subdural hematoma (arrowheads) with moderate associated mass effect.	Subdural hematoma 	Normal 
Age	Older adult 	Youth 
Pupillary light reflex	Pupil remains dilated 	Pupil constricts 
Caloric testing with ice water	Eyes do not deviate 	Eyes deviate to irrigated side 
Motor response to noxious stimuli	Decerebrate rigidity 	Localizes (defensive gesture) 

Figure 121-1 Differential Diagnosis of Altered Consciousness.



Management of the Comatose Patient
Secondary Survey

- Do a quick general exam of the entire body to identify acute life threatening conditions
- In general, major thoracic or abdominal trauma takes precedence after ABC's
- Only very rarely is acute neurosurgical intervention appropriate before other acute life threatening injuries are stabilized (except protection of spine (mostly cervical part) by immobilization)

Management of the Comatose Patient
Secondary Survey

- General motor exam
  - look for focal deficits, posturing (decerebrate or decorticate)
- Reflexes, tone
- Cranial nerve and brainstem function
  - Pupillary response - diencephalon, midbrain, brainstem, CN's II and III
  - Corneal Reflex - CN's V, VII, brainstem
  - Oculocephalic Reflex - not if neck injury possible. Tests CN's III, IV, VI, VIII, and brainstem.
  - Oculovestibular (calorics) can be done if neck questionable.

* Neurological Exam

Oculovestibular Testing

* Check for tympanic perforation
* Instill 120 cc cold water over 2 minutes
* Conscious patient - COWS
* Coma with intact pathways - tonic eye deviation to side of cold

* Management and evaluation of the Comatose Patient

* Does the patient have a rapidly progressive intracranial lesion?
* Assume yes, if:
  * 1. Any evidence of brainstem abnormality
  * 2. Any evidence of rostral caudal progression
  * 3. Any focal deficits
  * 4. Progression of motor exam from withdrawal to posturing

* If the patient have a rapidly progressive intracranial lesion:

* If any factor of such is present, assume increased intracranial pressure is present and herniation and irreversible damage imminent
  * Intubate
  * Hyperventilate
  * Mannitol
  * CT scan, neurosurgical consultation

* If the patient have a rapidly progressive intracranial lesion:

* If none of the findings are present, surgical lesion less likely than metabolic cause
* Mass lesion still possible, though - CT scan
* Urgency of intubation less but should consider
  * Will patient deteriorate, particularly while out of constant observation (CT scanner)?
  * Can patient protect airway?

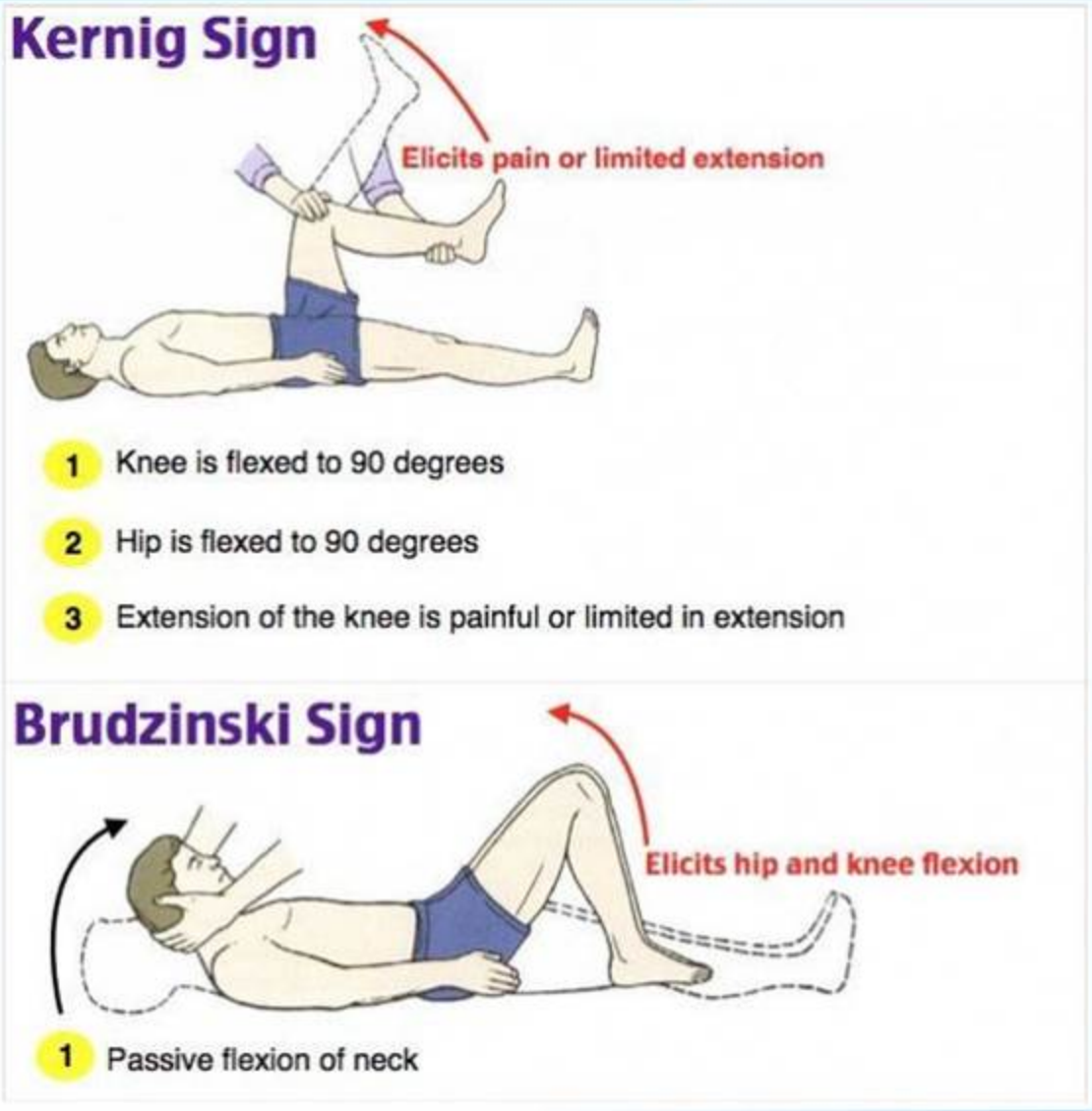
* Management and evaluation of the Comatose Patient

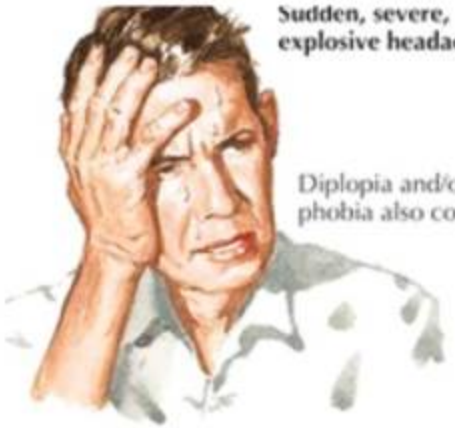
Additional Points

- If scans normal, probably metabolic
- Emergent causes of metabolic coma (even after ABC's)
  - Hypoglycemia - give glucose
  - Infection - LP, consider antibiotics, acyclovir. If diagnostic studies delayed, treat first
  - Certain toxins - antidepressants, salicylates, theophylline, alcohol (methanol and ethylene glycol)
  - Subclinical status epilepticus

*Meningeal signs

Meningeal Irritation Signs
- -Neck stiffness
- -Kernig's sign
- -Brudzniski'

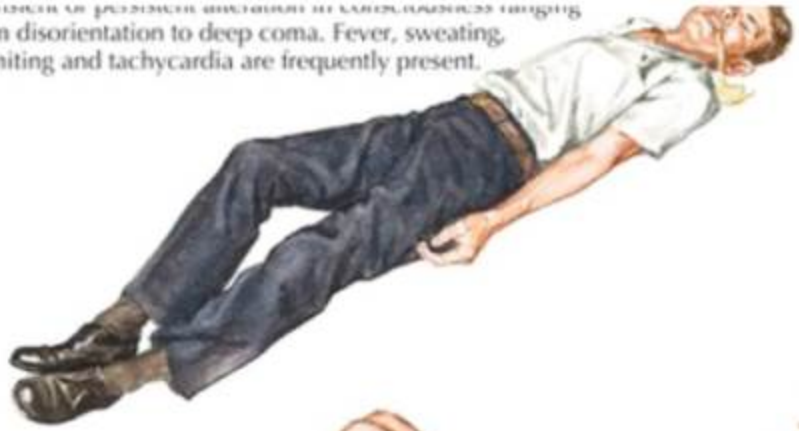




Sudden, severe, explosive headache

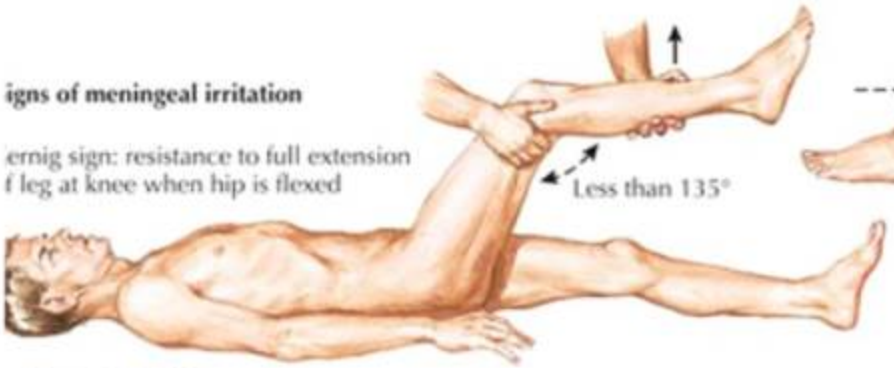
Diplopia and/or photophobia also common

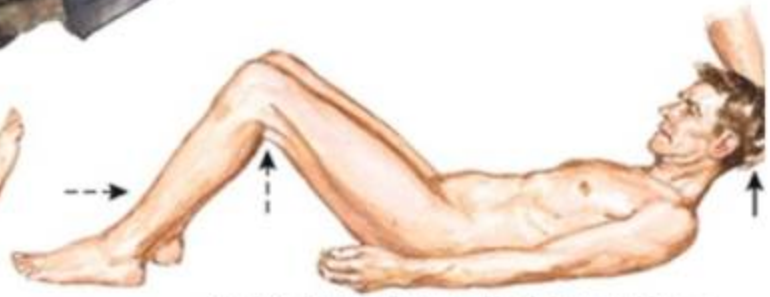
from disorientation to deep coma. Fever, sweating, vomiting and tachycardia are frequently present.



igns of meningeal irritation

ernig sign: resistance to full extension f leg at knee when hip is flexed





Brudzinski sign: flexion of both hips and knees when neck is passively flexed

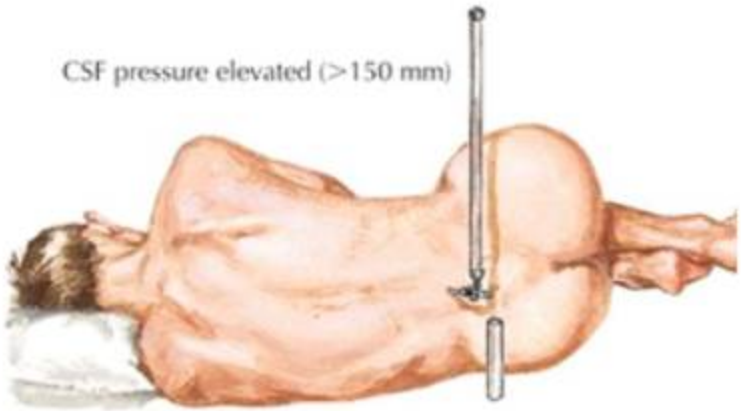
rebrospinal fluid

Three successive fluid samples collected shortly after subarachnoid hemorrhage show frank blood or are orange tinged in

Later, on repeat tap, all 3 samples are xanthochromic (yellow) as a result of hemoglobin release or bilirubin

If blood is due to traumatic tap, fluid clears progressively in successive samples.

CSF pressure elevated (>150 mm)



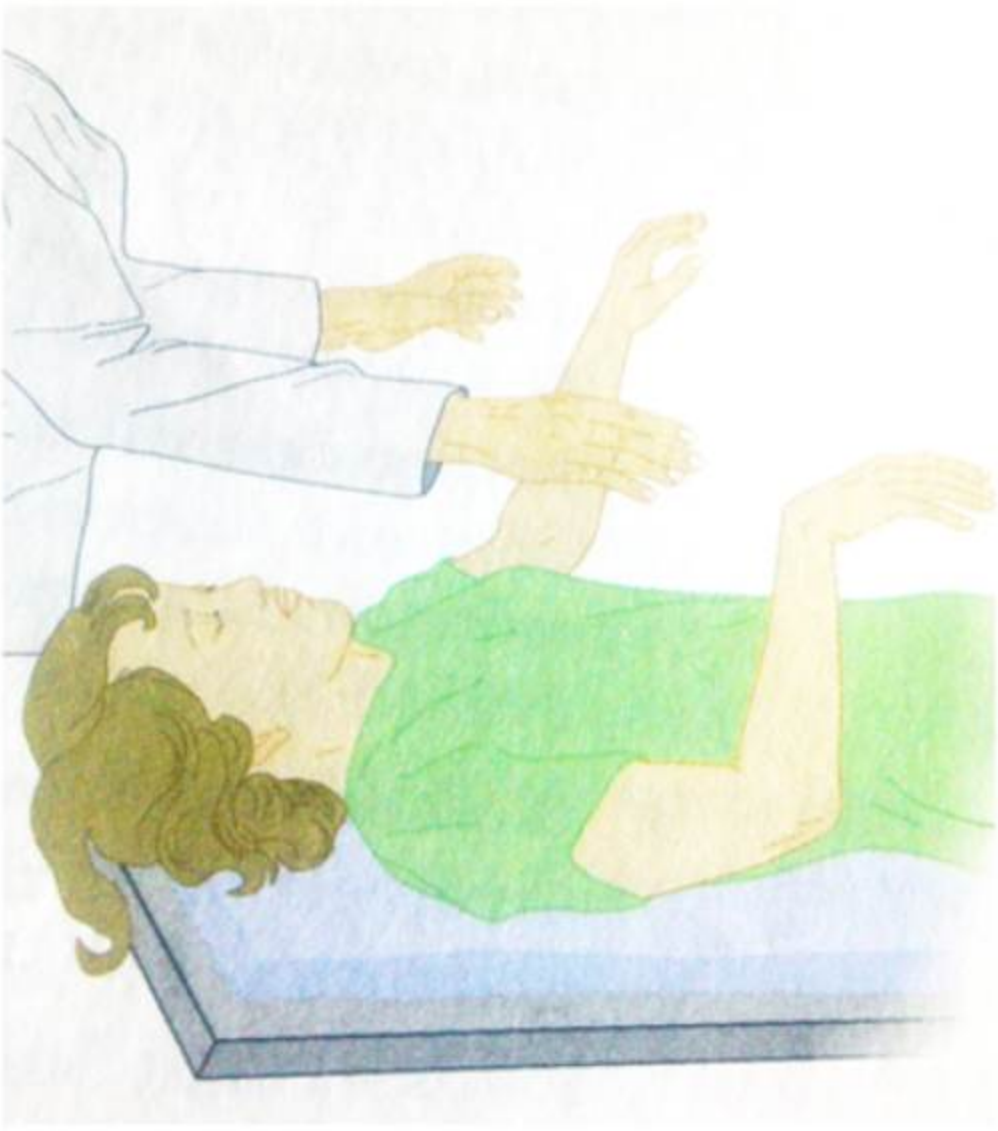
Motor examination

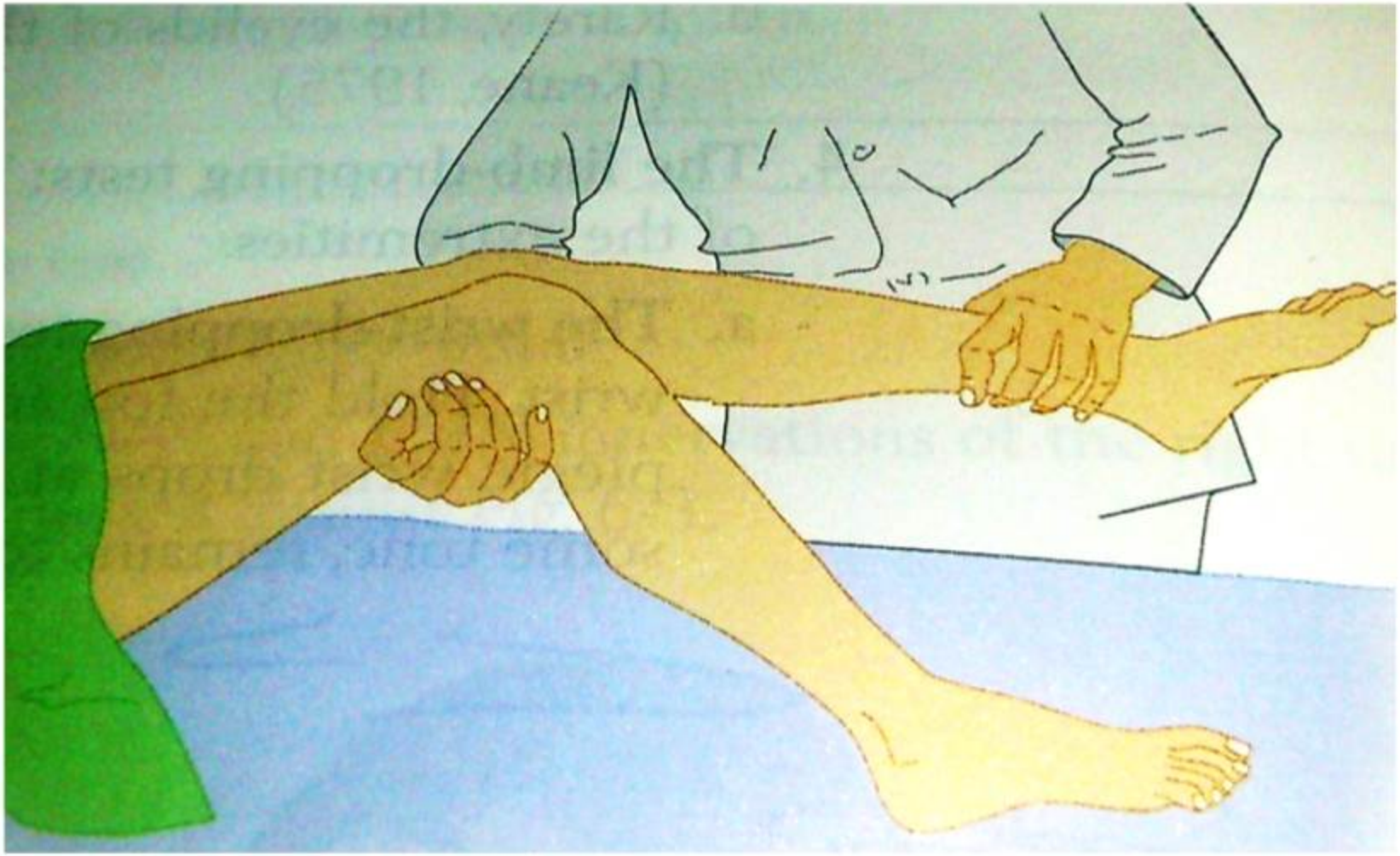
- Difficult to interpret
- Light coma→ lies quietly or thrashes about in bed
- Decorticate rigidity contralateral to hemispheric lesion
- Decerebrate rigidity
- Most common responses are reflexive in reaction to noxious stimuli
- Localizing responses, such as moving the examiner's hand away from the body, are *not consistent* with coma
- Flexion and extension responses to painful stimuli are consistent with coma

* Motor examination

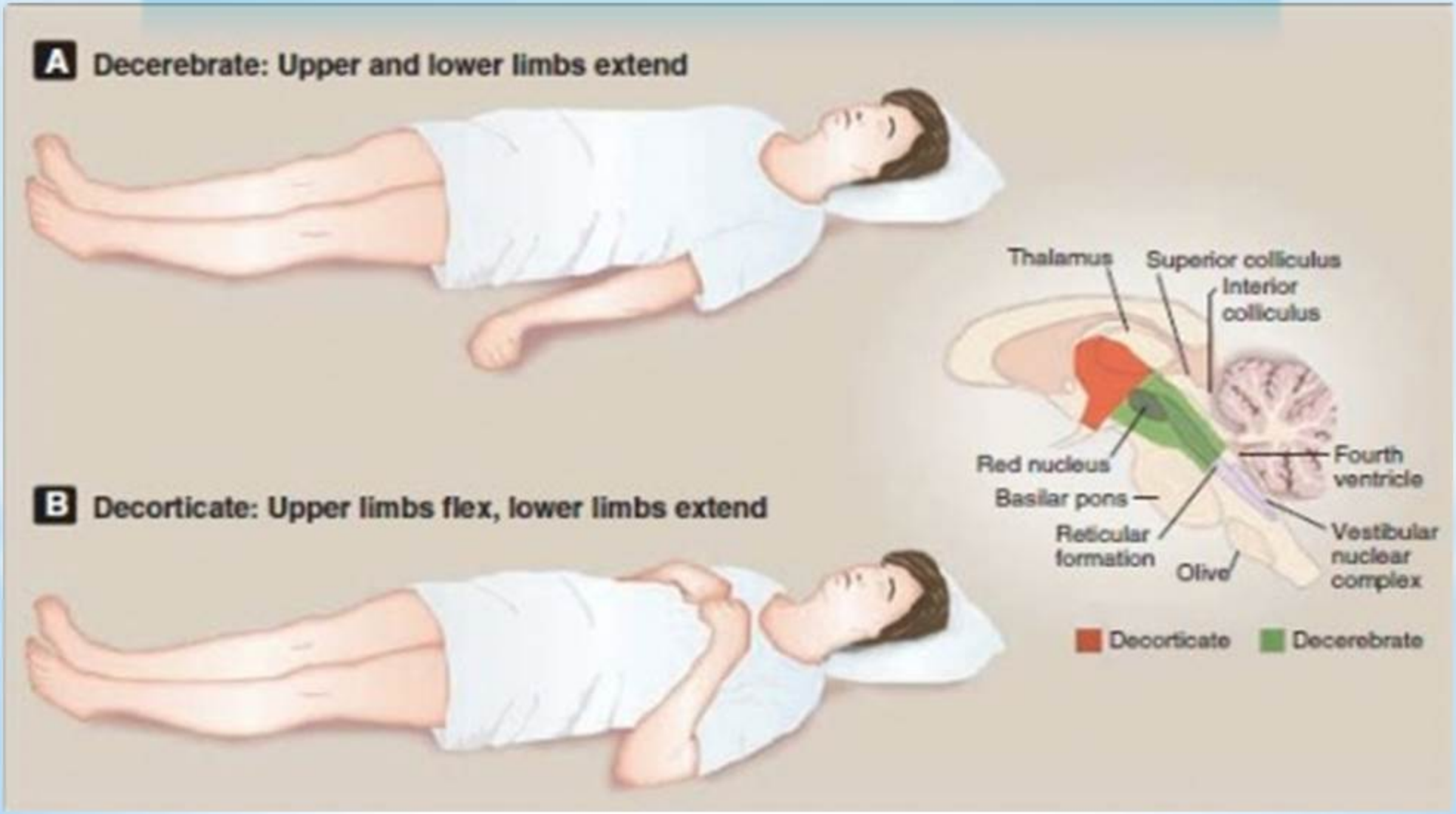
* Motor system
* Signs of hemiplegia

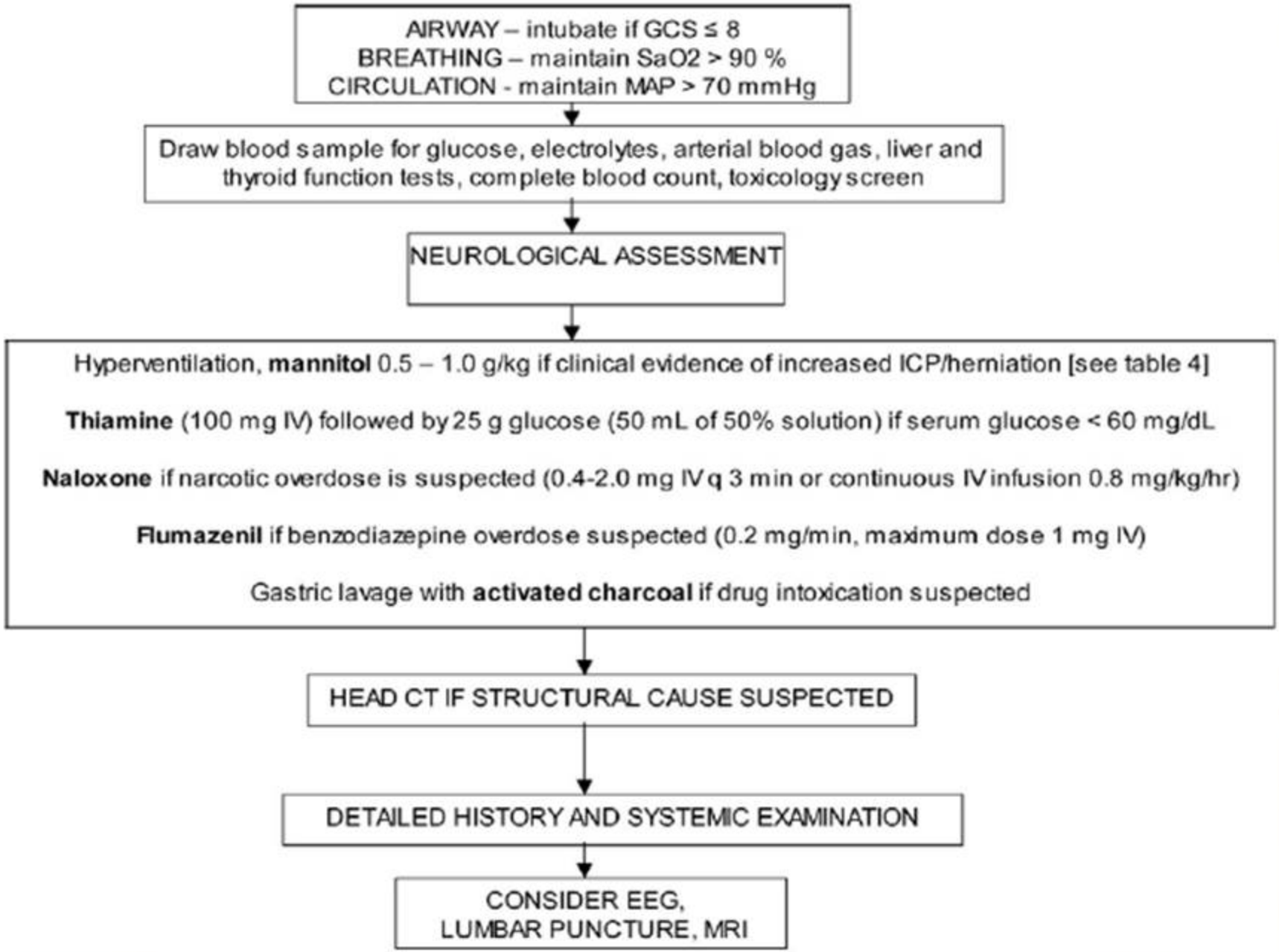
- wrist-dropping test
- Arm-dropping test
- Legs-dropping test
- Driven postures: Decorticate, Decerebrate.





* Posturing


A Decerebrate: Upper and lower limbs extend
B Decorticate: Upper limbs flex, lower limbs extend
Thalamus
Superior colliculus
Interior colliculus
Red nucleus
Basilar pons
Reticular formation
Olive
Fourth ventricle
Vestibular nuclear complex
Decorticate
Decerebrate


AIRWAY – intubate if GCS ≤ 8
BREATHING – maintain SaO2 > 90 %
CIRCULATION - maintain MAP > 70 mmHg
Draw blood sample for glucose, electrolytes, arterial blood gas, liver and thyroid function tests, complete blood count, toxicology screen
NEUROLOGICAL ASSESSMENT
Hyperventilation, mannitol 0.5 – 1.0 g/kg if clinical evidence of increased ICP/herniation [see table 4]
Thiamine (100 mg IV) followed by 25 g glucose (50 mL of 50% solution) if serum glucose < 60 mg/dL
Naloxone if narcotic overdose is suspected (0.4-2.0 mg IV q 3 min or continuous IV infusion 0.8 mg/kg/hr)
Flumazenil if benzodiazepine overdose suspected (0.2 mg/min, maximum dose 1 mg IV)
Gastric lavage with activated charcoal if drug intoxication suspected
HEAD CT IF STRUCTURAL CAUSE SUSPECTED
DETAILED HISTORY AND SYSTEMIC EXAMINATION
CONSIDER EEG,
LUMBAR PUNCTURE, MRI

*Reversible causes of coma

Structural	Diffuse
• Brain mass • Anoxic-hypoxic brain disease with RoSC after cardiac arrest • Raised intracranial pressure • SDH, EDH • ICH • Acute ischaemic stroke • Hydrocephalus • Cerebral oedema resulting from stroke • CVT • Sepsis , CNS infections • Non-convulsive or minimally convulsive status epilepticus	• Hypoglycaemia • Hyperglycaemia, diabetic or alcoholic ketoacidosis • Hyponatraemia • Hypercalcaemia • Hyperammonaemia • Renal failure • Hepatic encephalopathy • Thyroid storm • Myxoedema coma • Adrenal crisis • Pituitary apoplexy • Wernicke’s encephalopathy

* Prognosis in Nontraumatic Coma

* Using the Levy algorithm, a physician can predict the probability of a meaningful recovery 3 days after cardiac arrest

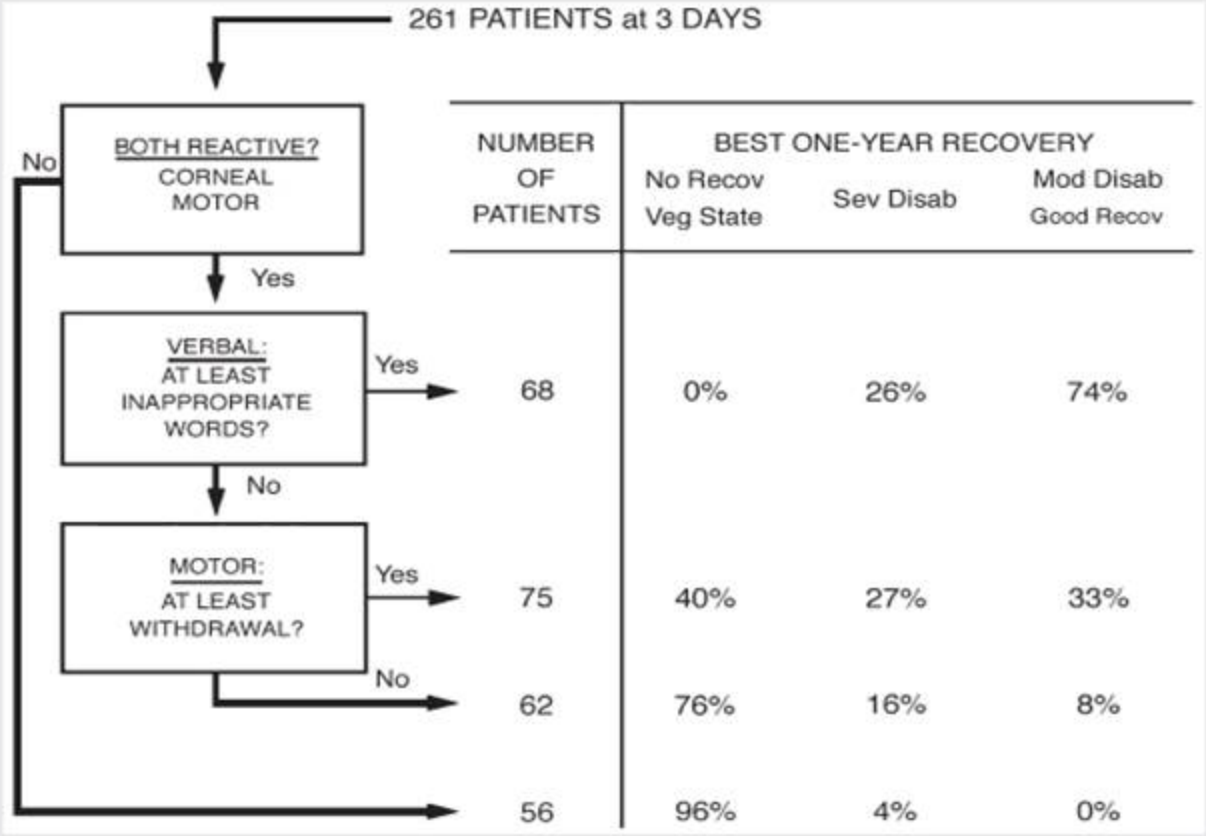


FIGURE 2-1 Decision tree of bedside tests to predict outcome in patients with nontraumatic coma.

Disab = disability; Mod = moderate; Recov = recovery; Sev = severe; Veg = vegetative.

Reprinted with permission from Levy DE, Bates D, Caronna JJ, et al. Prognosis in nontraumatic coma. Ann Intern Med 1981;94(3):293–301.

* Prognostic indicators

Phy Ex parameter

1. Lack of pupillary responses in the first 3 days, lack of corneal response in the first 3 days,
2. Evidence of widespread myoclonic epilepsy in the first 24 hours
3. Lack of extensor motor responses.

Lab parameter

1. A blood test for neuronspecific enolase (NSE)
2. Median nerve somatosensory evoked potentials

* THANK YOU