APPROACH TO ALTERED MENTAL STATUS IN THE EMERGENCY

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WHAT IS ALTERED MENTAL STATUS? LETHARGY/OBTUNDATION/STUPOR/COMA

• Level of arousal (Global/diffuse) vs Content of thought (Focal ie involvement of one class of stimuli)

• Objective scores have replaced archaic terms

• Sleep is the only physiologic form of reduced consciousness

THE THREE MAIN MECHANISMS ARE:

- 1.STRUCTURAL BRAIN LESIONS
- 2.DIFFUSE NEURONAL DYSFUNCTION SECONDARY TO SYSTEMIC PATHOLOGY

3.RARELY PSYCHIATRIC CAUSES

2)	Neurological	Metabolic	Diffuse physiological brain dysfunction	Psychiatric
	Ischaemic stroke	Hypoglycaemia	Seizures – induding nonconvulsive status epilepticus	Psychiatric coma
	Intracere bral haemorrhage	Hyperglycaemia	Alcohol intoxication	Malingering
	Subarach noid haemorrhage	Hyponatraemia	Opioid toxicity	
	Subdural haematoma	Hypernatraemia	Drug overdose	
	Brain tumour	Hypercalcaemia	Poisoning	
	Cerebral lymphoma	Addisonian crisis	Hypothermia	
	Multiple brain metastases	Hypothyroidism	Neuroleptic malignant syndrome	
	Central nervous system infection	Uraemia	Serotonin syndrome	
	Cerebral abscess	Hypercapnia		
	Cerebral oedema	Septic encephalopathy		
	Hydrocephalus	Hepatic encephalopathy		
	Anoxic brain injury (eg post cardiac arrest)			
	Posterior reversible encephalopathy syndrome (PRES)			
	Trauma			

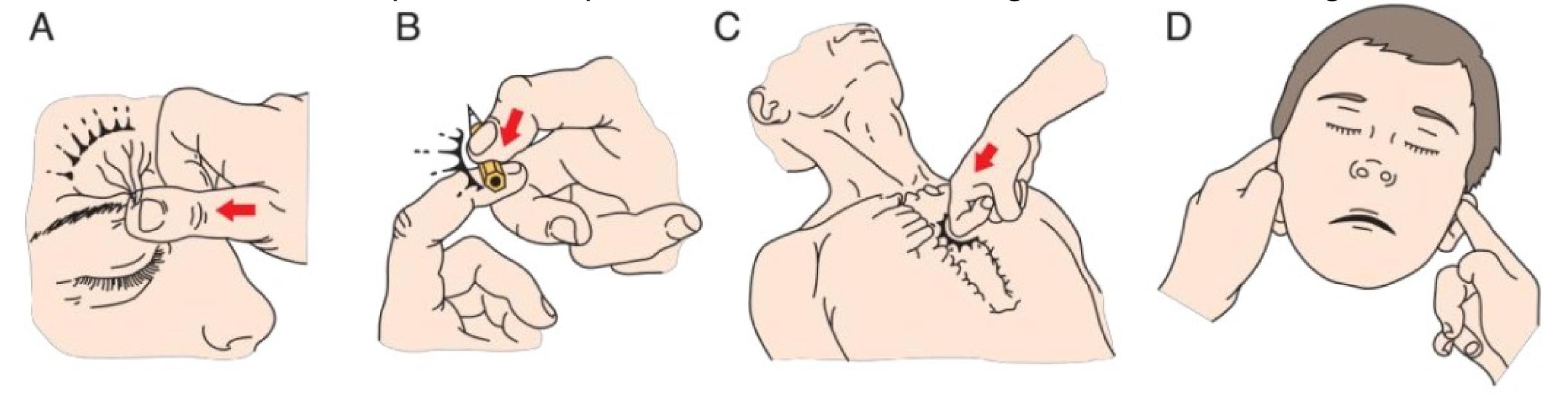
Epidemiology

- The most prevalent etiology of non traumatic coma is ischemic or hemorrhagic stroke (6 to 54%)
- Anoxic injury (3 to 42%)
- Poisoning (1 to 39%)
- Metabolic (1 to 29%)

Non-structural causes tended to slightly out number the structural causes (37-75% vs 28-64%)

SCALES AND MEASURES: LEVEL OF CONSCIOUSNESS

The maneuvers should provide adequate stimuli without inducing actual tissue damage



- Glasgow Coma Scale Scale or Score? -Use the best score
- FOUR Score Full Outline of UnResponsiveness
- AVPU Awake Verbal Pain Unresponsive
- ACDU Alert Confused Drowsy Unresponsive

GCS

GLASGOW COMA SCALE: Do it this way



Institute of Neurological Sciences NHS Greater Glasgow and Clyde



CHECK

For factors Interfering with communication, ability to respond and other injuries



OBSERVE

Eye opening, content of speech and movements of right and left sides



STIMULATE

Sound: spoken or shouted request

Physical: Pressure on finger tip, trapezius or supraorbital notch



RATE

Assign according to highest response observed

THE GLASGOW COMA SCALE

П	Eye opening	Movement	Verbal
	4-Spontaneous	6 — Obeys commands	5 — Oriented
	3 — To speech	5 — Localises to pain	4 — Confused
	2 — To pain	4 — Withdraws from pain	3 — Imap propriate words
	1 None	3 — Abnormal flexion to pain	2 — Incomprehensible sounds
		2 — Extensor response to pain	1 — None
		1 — No response	

FOUR SCORE

Eye Response

- •4 = eyelids open or opened, tracking, or blinking to command
- •3 = eyelids open but not tracking
- •2 = eyelids closed but open to a loud voice
- •1 = eyelids closed but open to pain
- •0 = eyelids remain closed with pain

Motor Response

- •4 = thumbs-up, fist, or peace sign
- •3 = localizing to pain
- •2 = flexion response to pain
- •1 = extension response to pain
- •0 = no response to pain or generalized myoclonic status

Brainstem Reflexes

- •4 = pupil and corneal reflexes present
- •3 = one pupil wide and fixed
- •2 = pupil or corneal reflexes absent
- •1 = pupil and corneal reflexes absent
- •0 = absent pupil, corneal, and cough reflex

Respiration

- •4 = not intubated, regular breathing pattern
- •3 = not intubated, Cheyne-Stokes breathing pattern
- •2 = not intubated, irregular breathing
- •1 = breaths above the ventilator rate
- •0 = breaths at ventilator rate or below

FOUR SCORE

- Incorporates Brainstem reflexes
- No verbal component
- Good Interobserver agreement
- Is it time to shift? Evidence practice mismatch

WHEN IN DOUBT: JUST DESCRIBE IT.

HISTORY

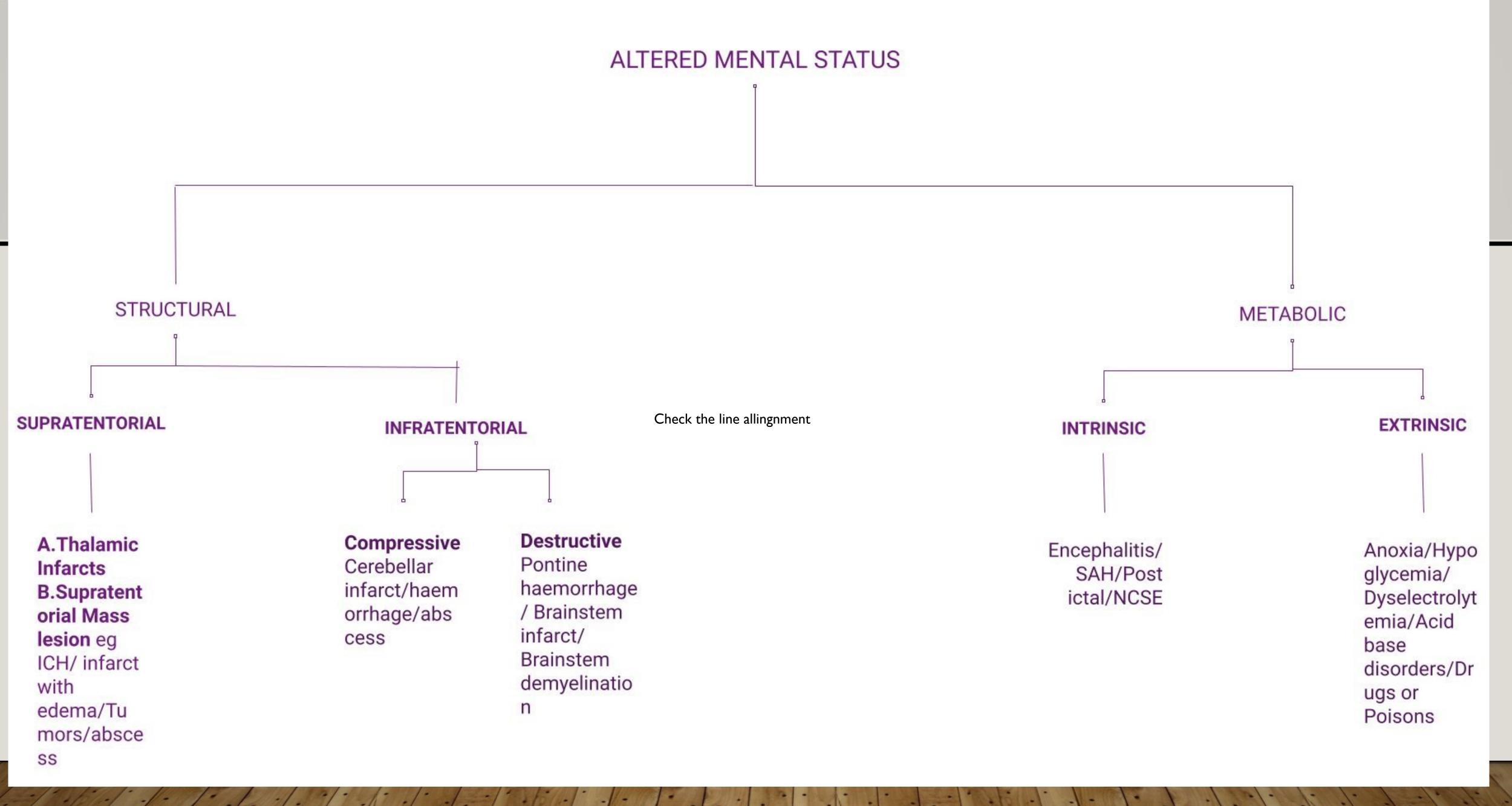
Attempt to Solicit and record history that includes:

- Time and acuity of onset, fluctuations, evolution
- Associated symptoms a. Headache/fever/seizures/nausea vomiting b. Motor activity changes c. Speech changes in content and pattern
- Past history: similar events, Chronic neurologic deficits, HIV status,
- Family history: including neurologic, endocrinological
- Current medication Dose, recent alterations in medicine or in its dosage
- Social history alcohol and drug abuse

NEUROLOGICAL EXAMINATION CHECKLIST

It's brief. Fortunately.

- What we don't want to miss
- Level of consciousness
- Pupillary responses
- Oculomotor responses
- Motor: Tone/Reflexes/Responses
- Pattern of breathing
- Fundus
- Meningeal signs ?LP

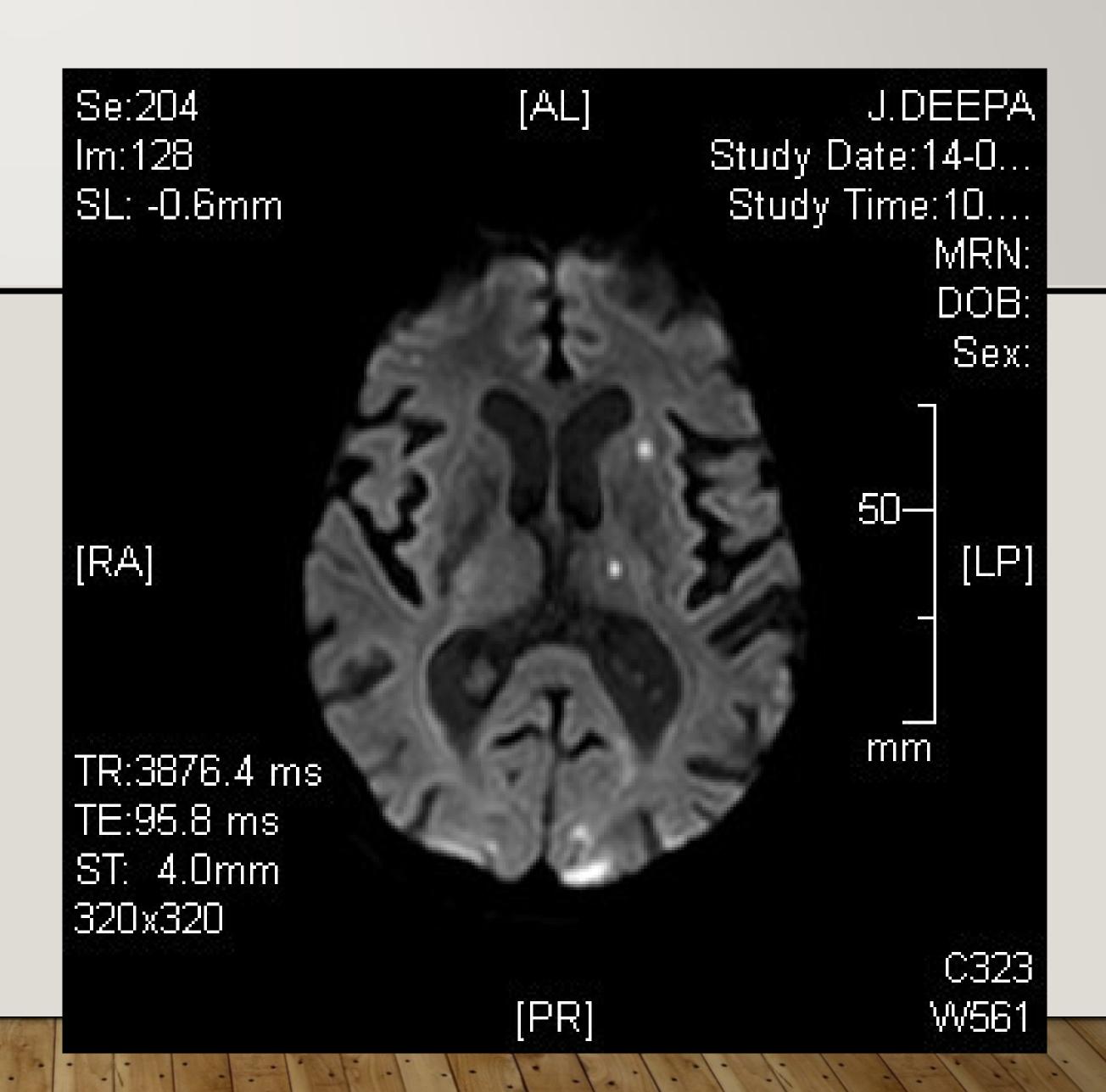


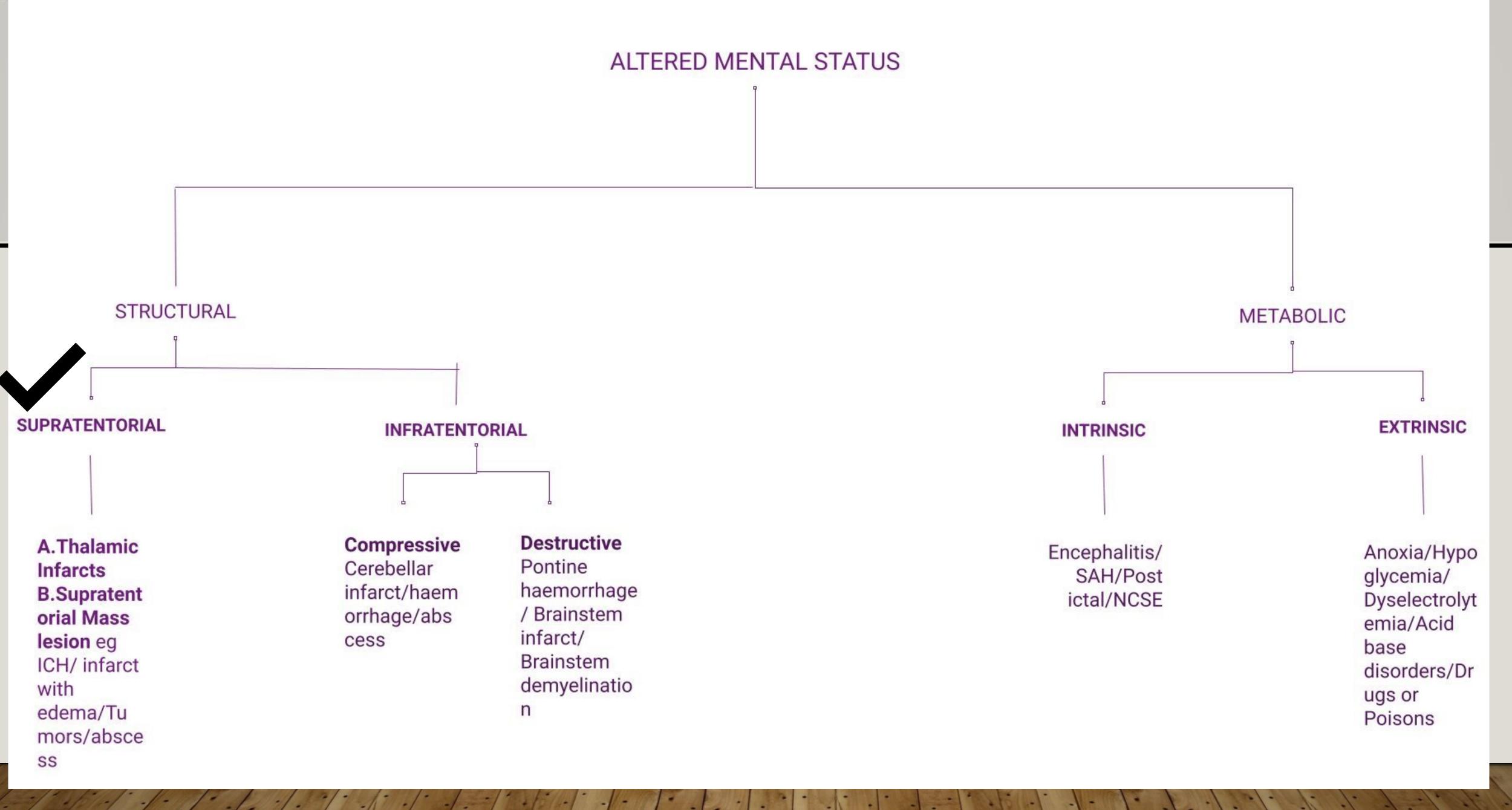
Clinical Vignette 1:

- 84 y, F Acute onset drowsiness for 18h
- O/E : E2V I M 5
- Gaze preference to the left
- Moving all 4 limbs to pain
- NCCT Head : Acute infarct in left posterior temporal and occipital
- Does this explain M5 status?



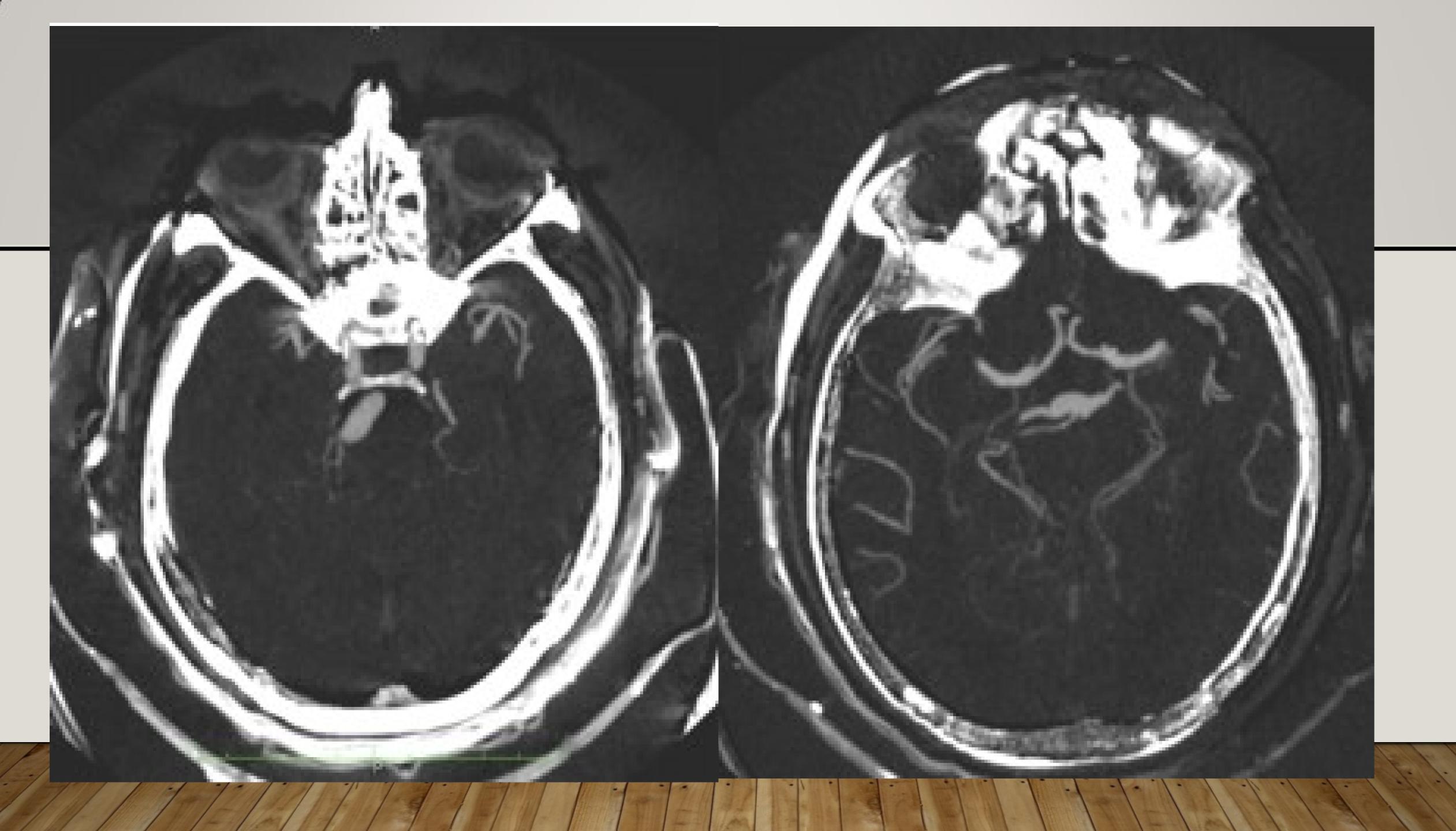
- Unilateral hemispheric lesion will not cause a reduced level of consciousness.
- MRI revealed left thalamic infarct
- Stupor or coma at onset in thalamic stroke occurred in 62%
 IVH vs 6% infarcts vs ICH 13% *





Clinical Vignette 2:

- A 58 year, M
- Hypertension for 20 years
- presented with sudden loss of consciousness and fall in the bathroom, followed by confusion and quadriparesis
- O/E E3V4M6(drowsy)
- Pupils mid-dilated, sluggishly reactive to light on the right
- Tone raised in all 4 limbs.
- B/L Plantar extensor, All DTR exaggerated.
- Fundoscopy Normal



 NCCT head: Hypodense lesion on the right side of Midbrain extending into the Thalamus with mass effect.

CTAngiography :Vertebrobasilar
 Dolichoectasia

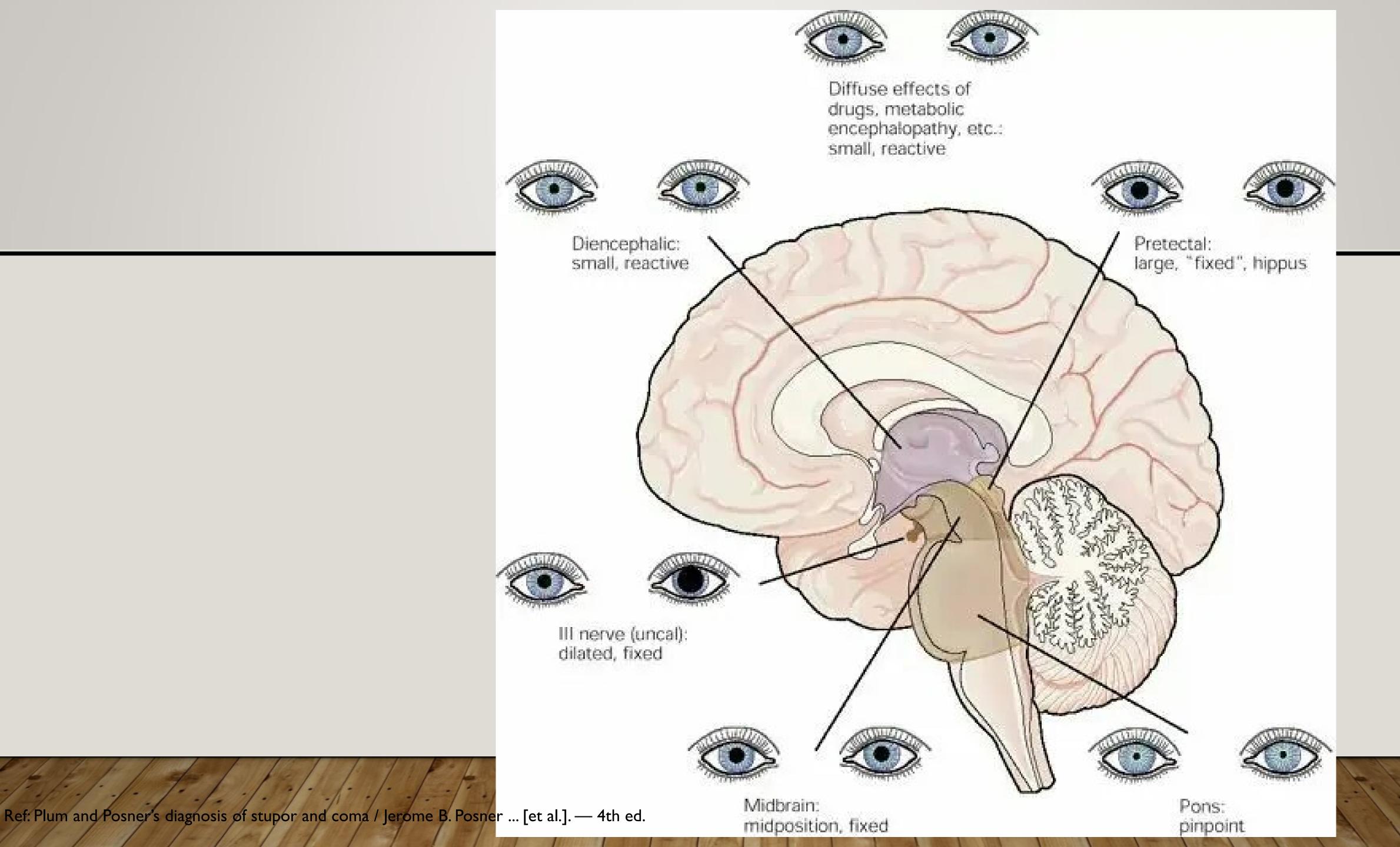
PUPILS IN AMS

Single most important physical finding to differentiate structural vs metabolic

- Illumination penlight vs camera flash
- At least for 10 secs Tonic pupils react slowly
- If pupils are very small ideally a plus 20 lens maybe used
- Remember r/o pharmacologic dilation

LOCALISING VALUE OF PUPILLARY RESPONSES

- Unilateral enlarged poorly reactive pupils first sign of herniation (or an aneurysm in PCOM)
- Look for Horner's
- Metabolic vs Diencephalic both cause small, reactive pupils (difficult to appreciate response) . Pupillary light reflex is most one of the most resistant brain responses to metabolic insults.
- During or following seizures pupils may be large or poorly reactive to light. Look for tongue bite/ incontinence /



Clinical Vignette 3:

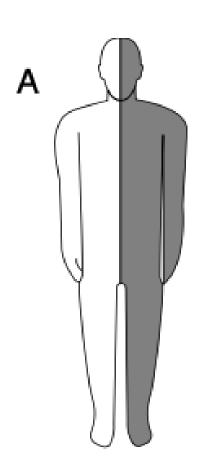
- A 76y lady with HTN, DM
- sudden onset altered mental status for last
 18h
- E3V3M5
- Eyes deviated downwards and nasally
- Tone increased in all 4 limbs
- Generalised hyperreflexia
- B/L extensor Plantar



Ref: Chowdhury, S., Singh, R.K., Vibha, D. et al. Transient obstruction of aqueduct of sylvius: rare case of spontaneously resolving acute hydrocephalus. Acta Neurol Belg. (2022). https://doi.org/10.1007/s13760-022-02074-0

RESTING AND SPONTANEOUS EYE MOVEMENTS

- Gaze Preferences:
- Conjugate Deviations of the eye
 - a. To the side of lesion
 - b. Away "Wrong way"
 - c. Downward and nasally
- Skew Deviation

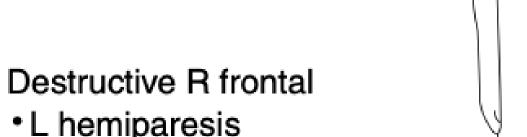




L hemiparesis

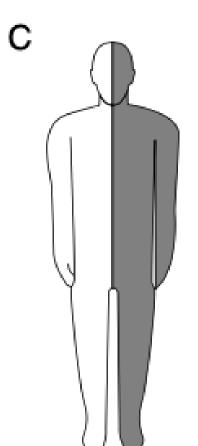
R eye deviation

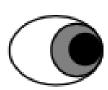






- L hemiparesis(+/-)
- L eye deviation



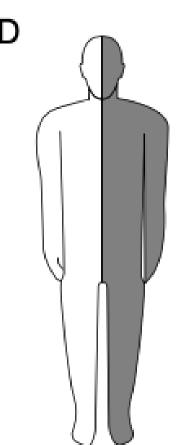


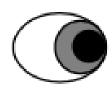






- L hemiparesis
- L eye deviation



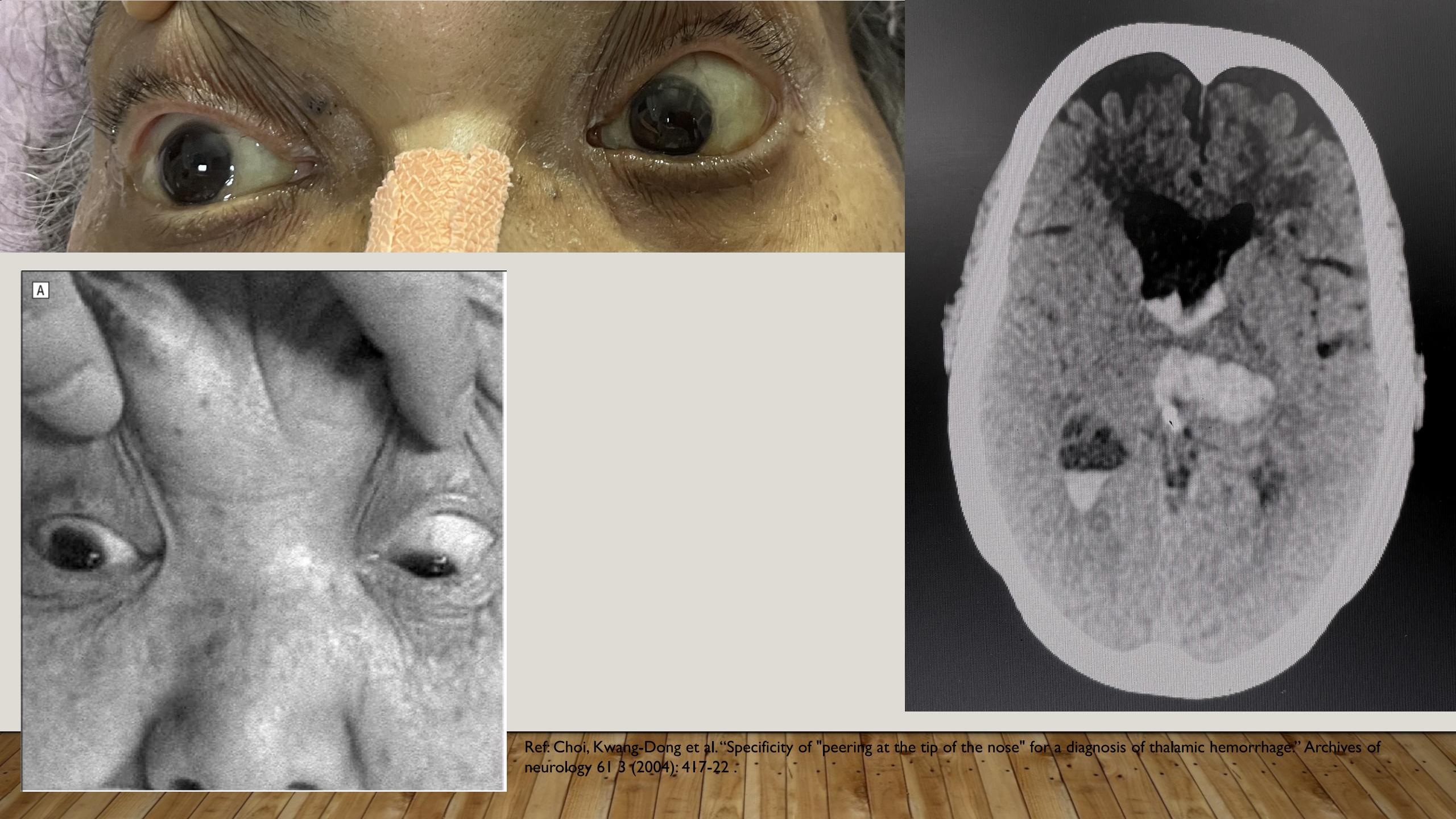




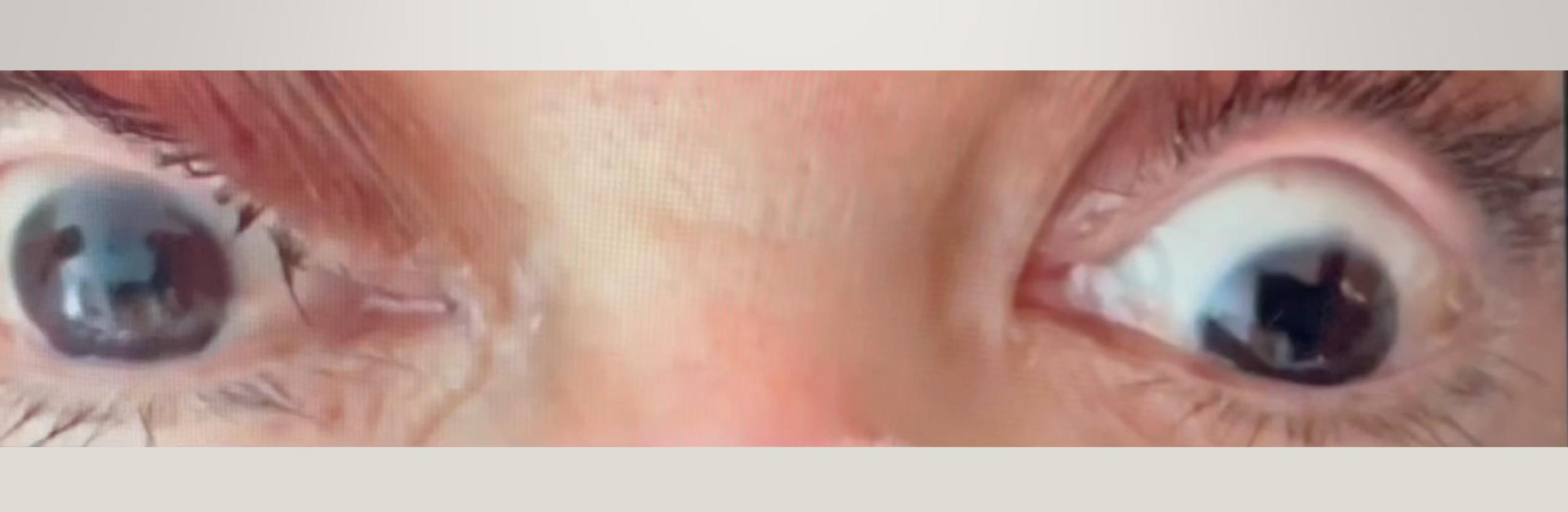
Destructive R pontine

- L hemiparesis(+/-)
- L eye deviation

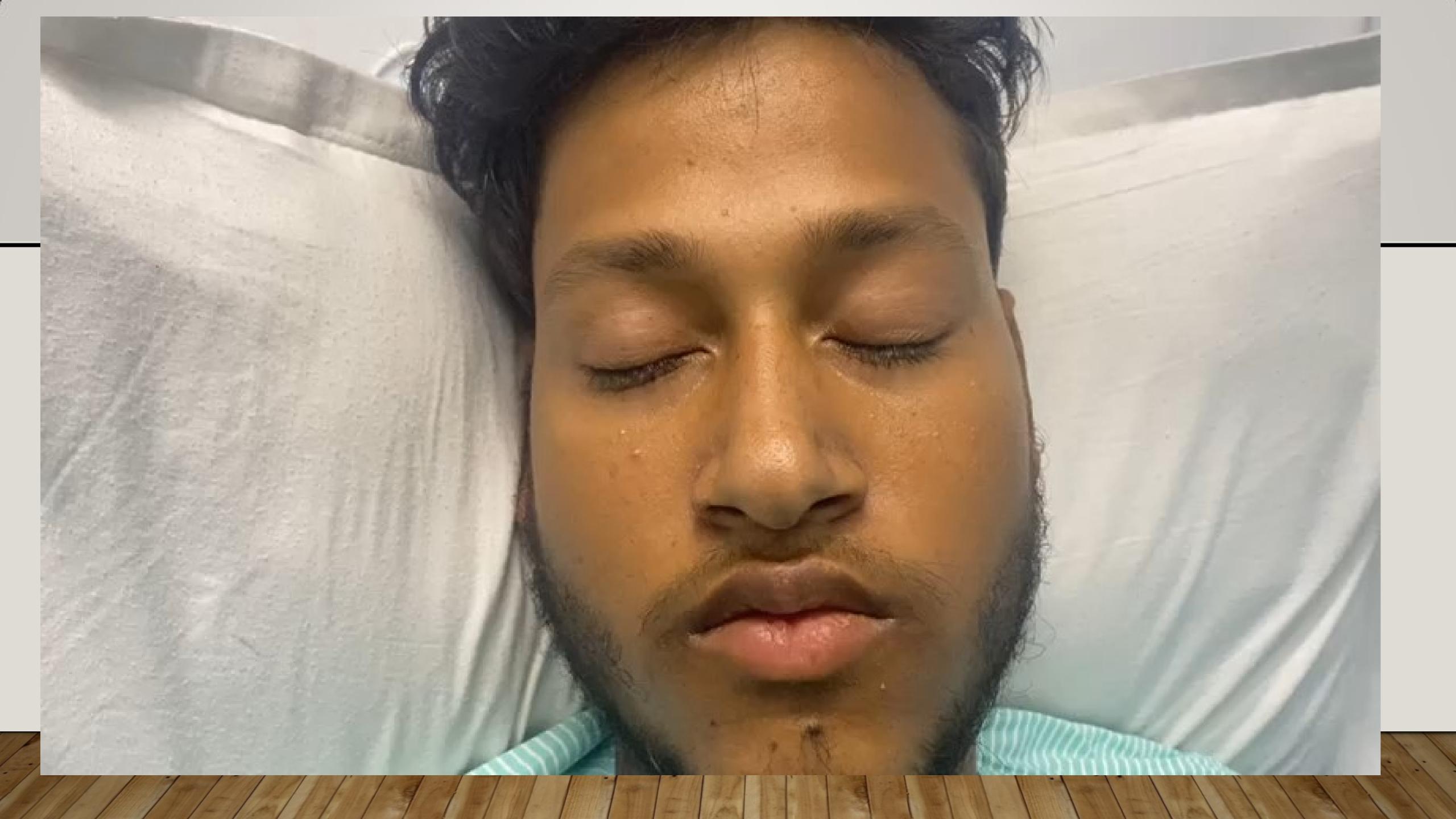
Ref: Neurology: a clinician's approach / Andrew Tarulli. 2011







• Ref:Jeanneret V, Beach PA, Kase CS. Ocular Dipping in Anoxic Brain Injury. JAMA Neurol. 2019;76(10):1252. doi:10.1001/jamaneurol.2019.2393



OCULOCEPHALIC RESPONSES

Pathway of eye movements overlap extensively with arousal system

Structural disease = unusual to have a normal oculocephalic response

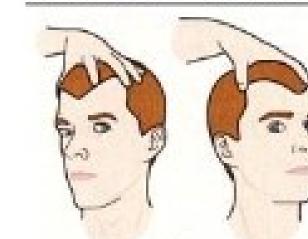
Vs Metabolic = usually it is normal Even exaggerated in Hepatic encephalopathy

Deeply comatose = eye movements may be sluggish or not at all

Oculocephalic responses

Turn left

Brainstem intact (metabolic encephalopathy)



Turn right

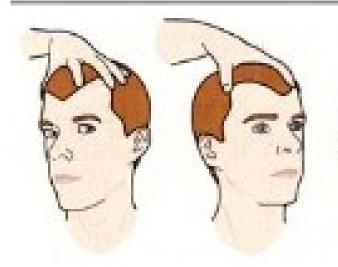


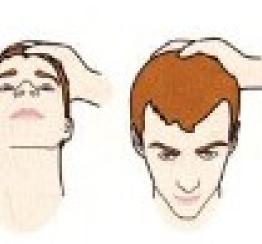
Tilt back Tilt forward



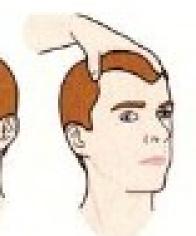
Right lateral pontine lesion (gaze paralysis)

MLF lesion





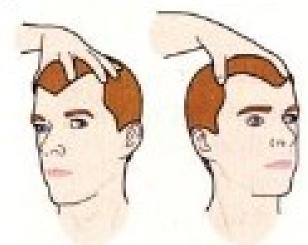
(bilateral internuclear ophthalmoplegia)







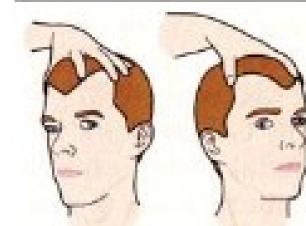
Right paramedian pontine lesion (1 1/2 syndrome)







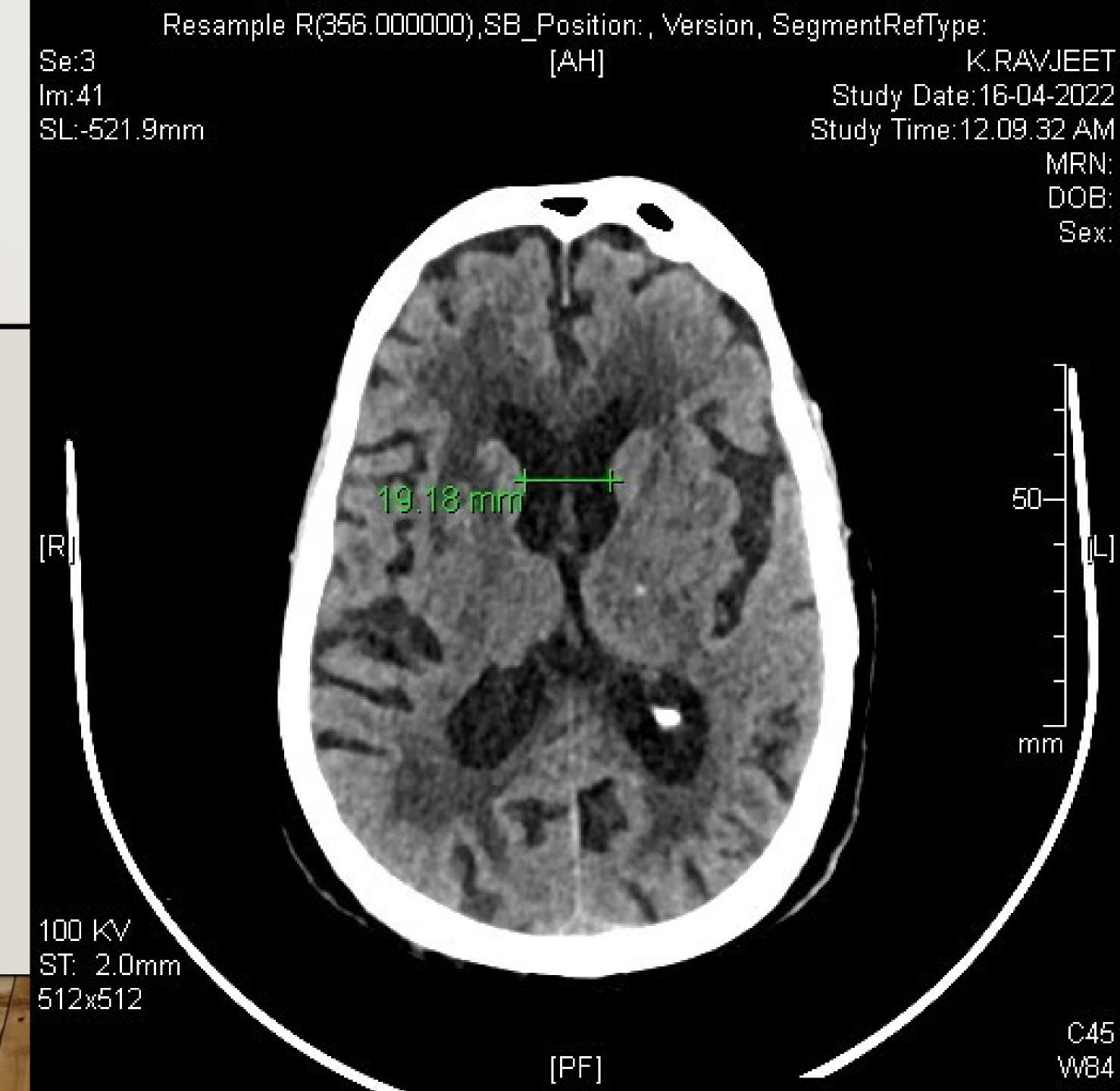
Midbrain lesion (bilateral)

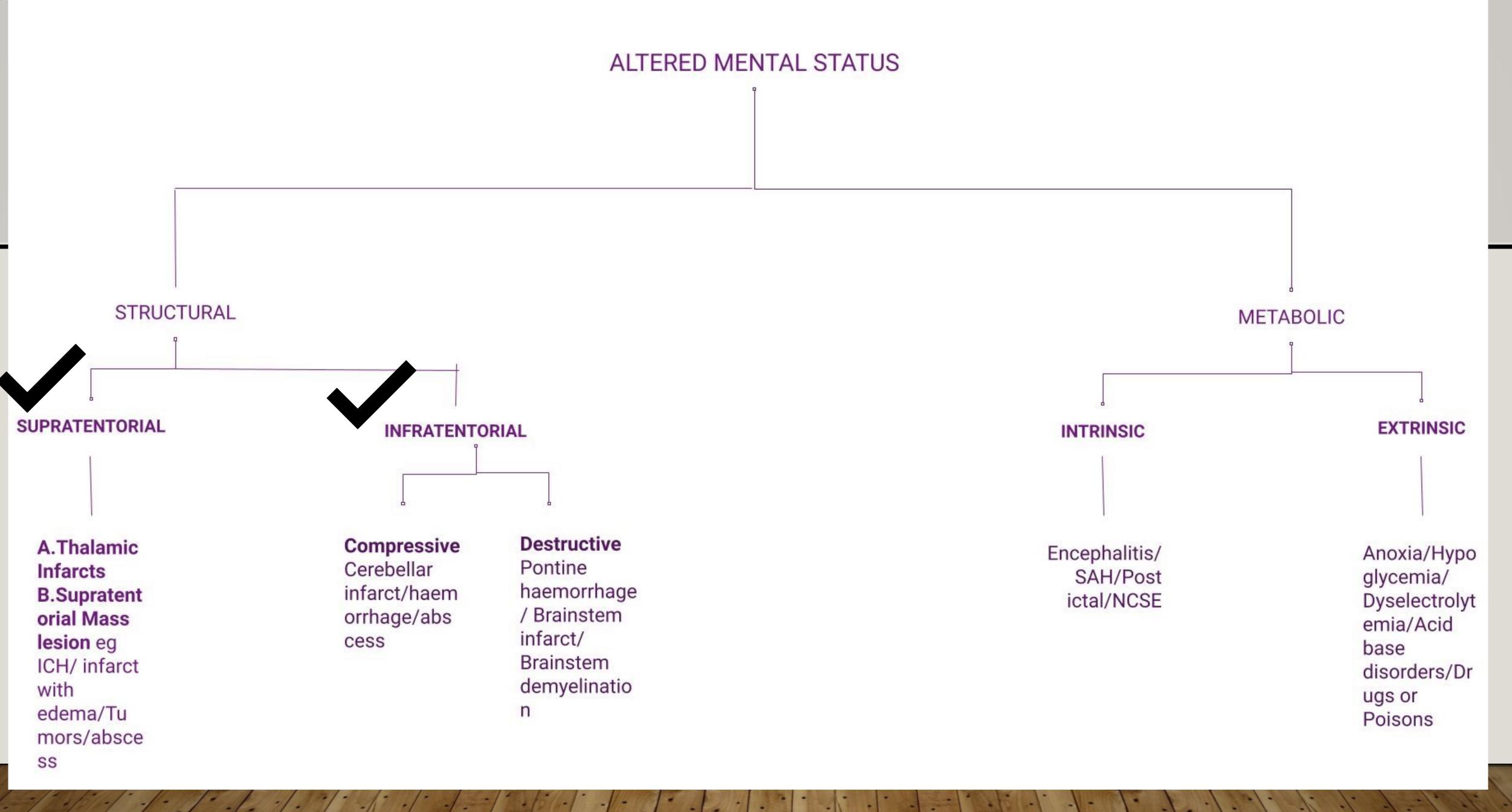






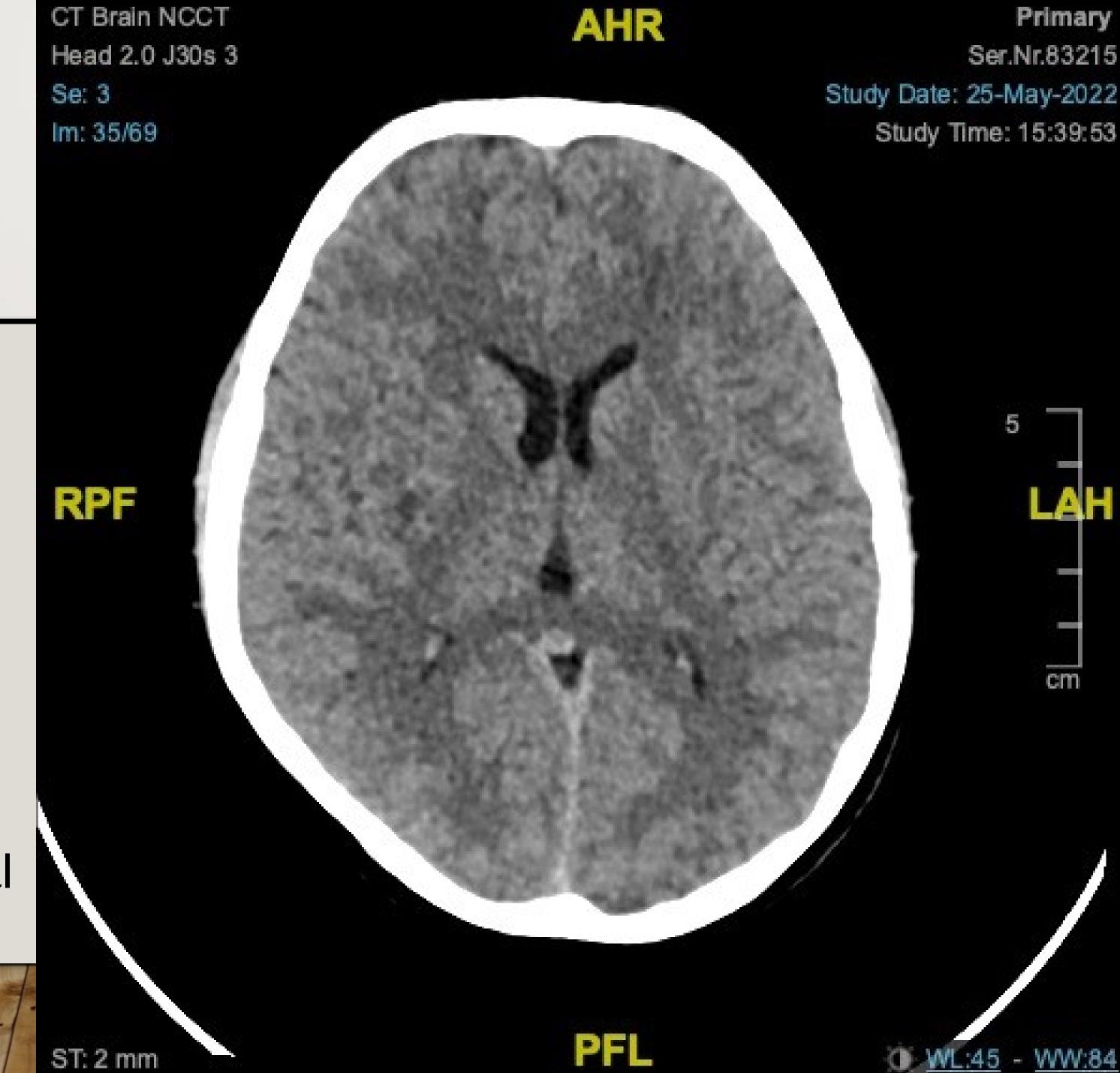
 Patient improved spontaneously over the next 12 h





Clinical Vignette 4

- 19 y male, no comorbidities
- Headache for 2 days
- I episode of vomiting at home
- In the ER I episode of tonic posturing became M4
- HR 42/min
- NO MENINGEAL SIGNS
- NCCT head apparently normal



DIAGNOSTIC ACCURACY OF KERNIG'S SIGN, BRUDZINSKI'S SIGN, AND NUCHAL RIGIDITY FOR PATIENTS WITH SUSPECTED MENINGITIS

	No. of patients		
Sign	With meningitis ^a	Without meningitis	AII
Kernig's ^b			
Present	3	8	11
Absent	63	163	226
Brudzinski's ^c			
Present	3	8	11
Absent	63	162	225
Nuchal rigidity ^d			
Present	24	69	93
Absent	56	148	204

NOTE. LR-, likelihood ratio for a negative test result, LR+, likelihood ratio for a positive test result.

a Defined as ≥6 WBCs/mL of CSF.

^b Sensitivity, 5%; specificity, 95%; positive predictive value, 27%; negative predictive value, 72%, LR⁺, 0.97; LR⁻, 1.0; ratio of LR⁺ to LR⁻, 0.97.

^c Sensitivity, 5%; specificity, 95%; positive predictive value, 27%; negative predictive value, 72%; LR⁺, 0.97; LR⁻, 1.0; ratio of LR⁺ to LR⁻, 0.97.

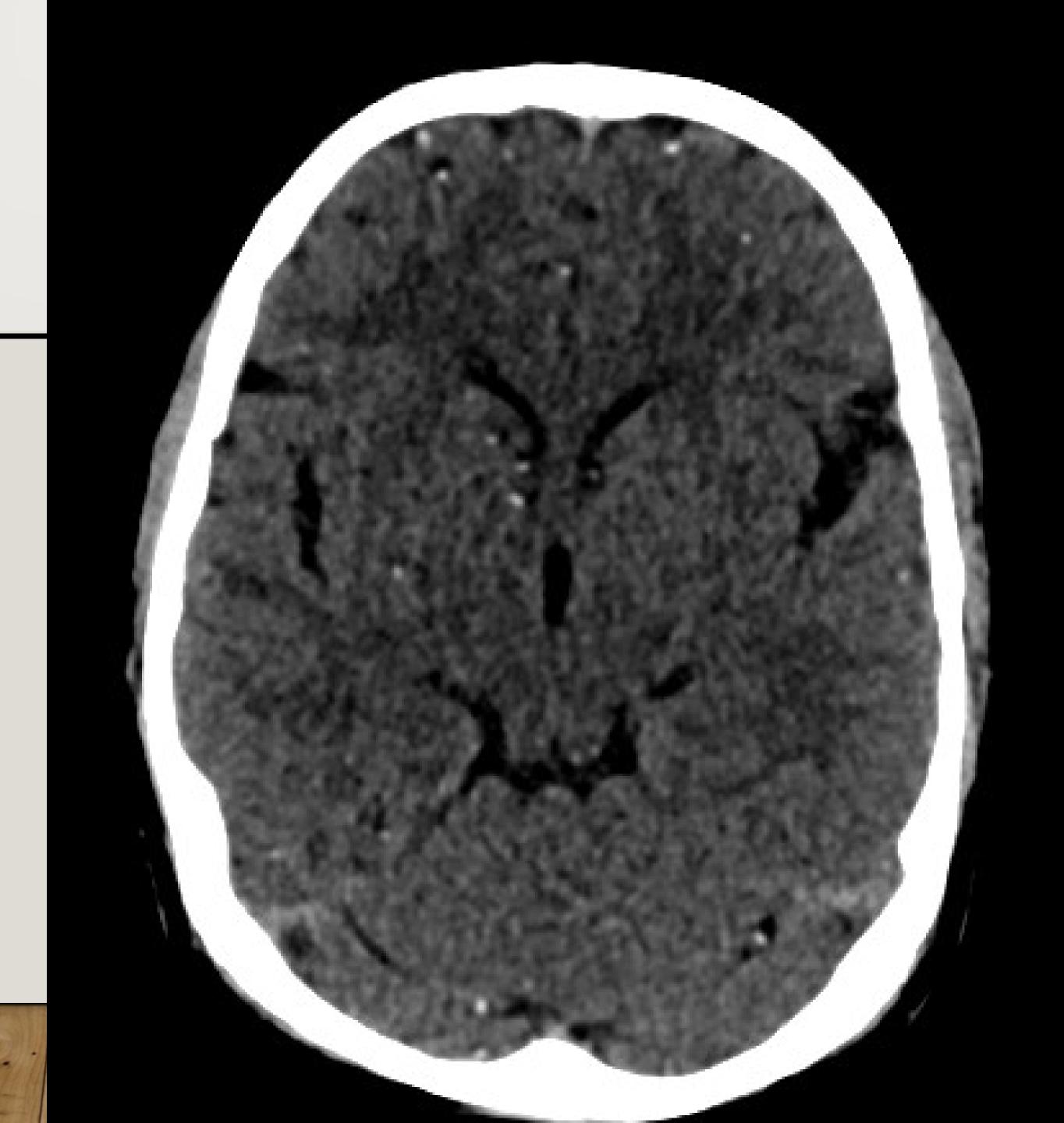
d Sensitivity, 30%; specificity, 68%; positive predictive value, 26%; negative predictive value, 73%; LR+, 0.94; LR-, 1.02; ratio of LR+ to LR-, 0.92.

B/L Papilloedema

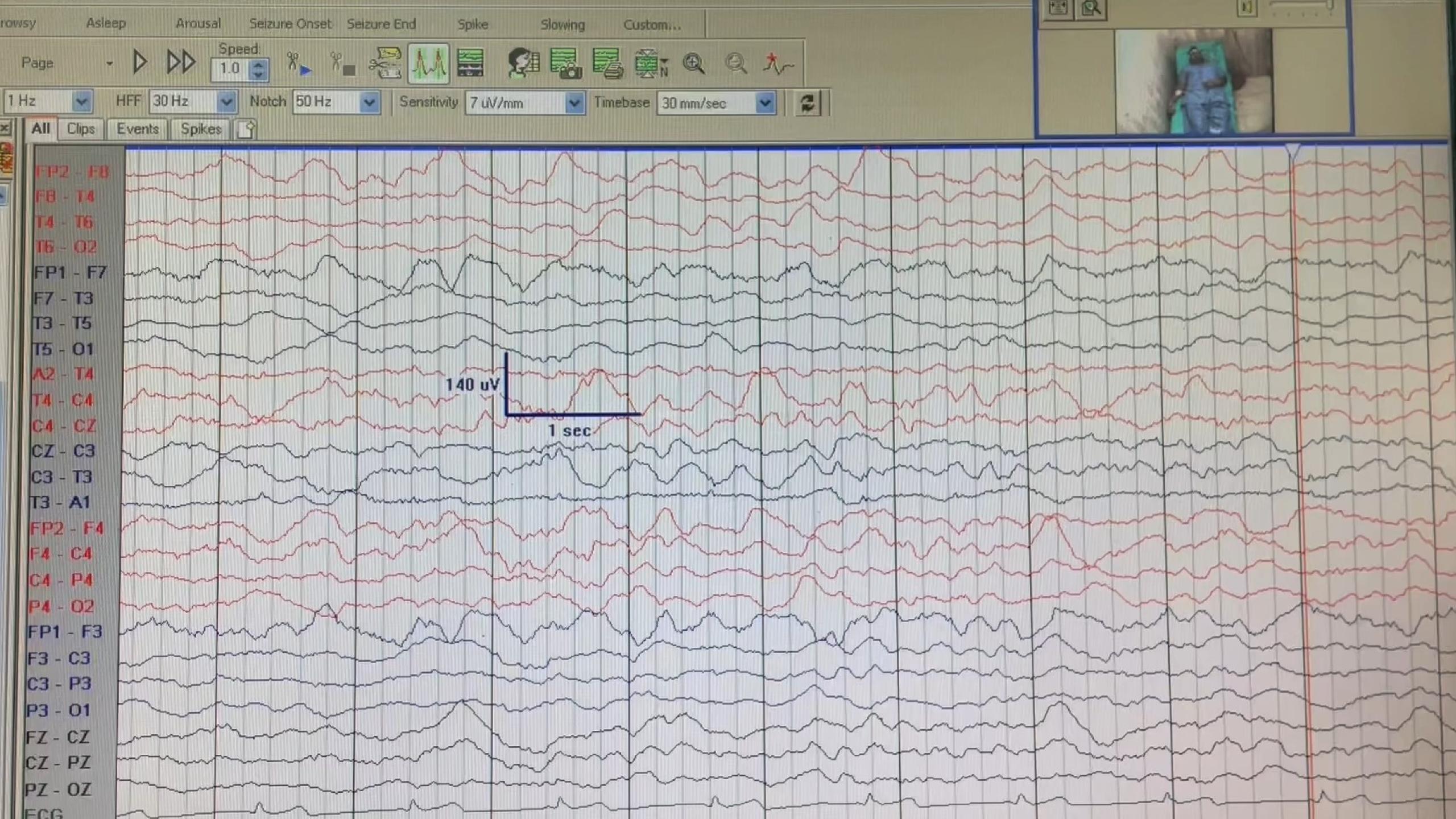
- Guarded LP was done CryAg Positive
 >1:256 titre, India ink +
- HIV Negative
- Prevalence of Cryptococcal meningitis in India 1.09% *

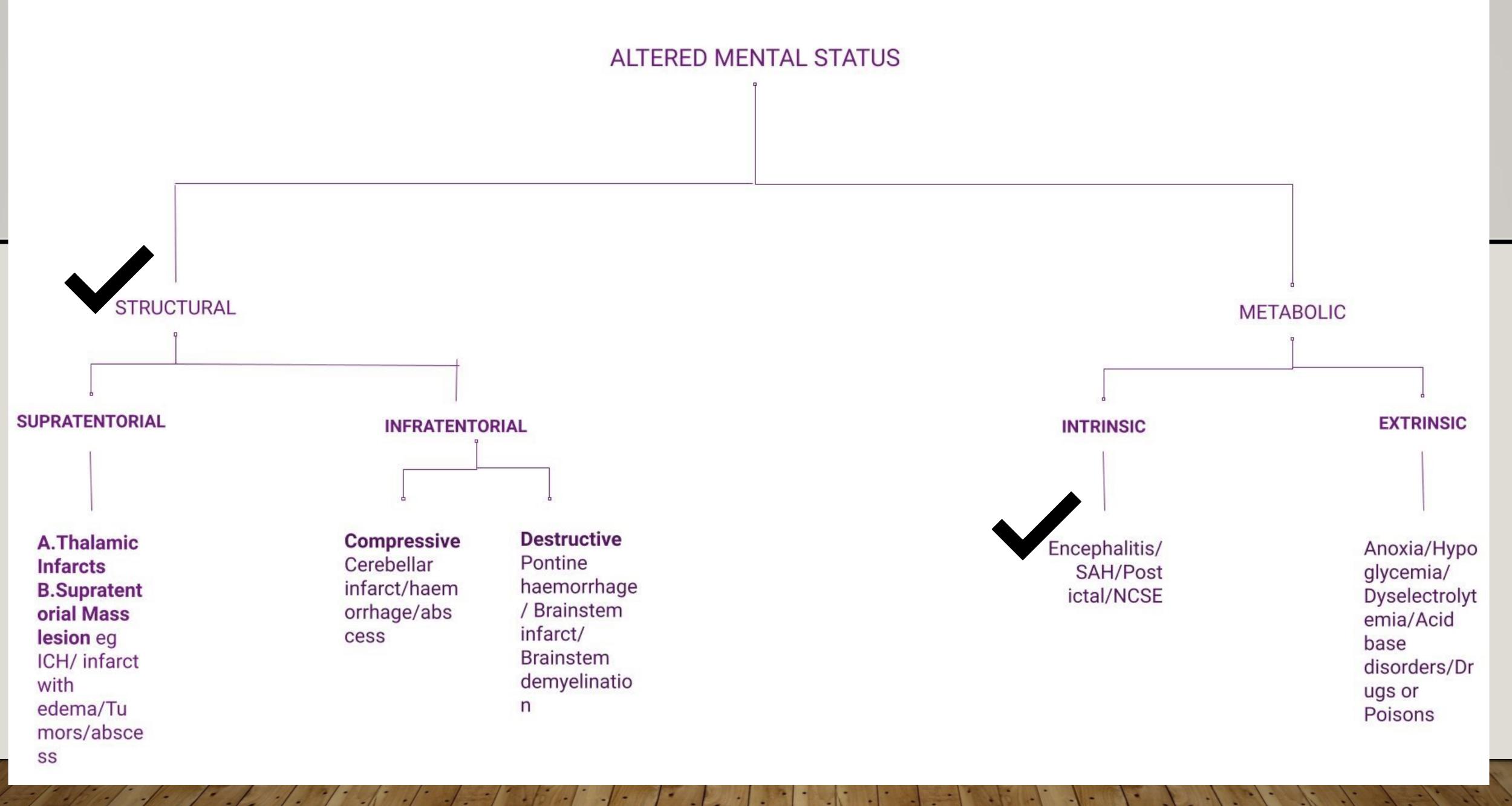
Clinical Vignette 5

- 26 y Male
- Headache with metamorphospsia
- Operated for retinal neurocysticercosis
- I day of irrelevant talk, agitation
- GCS E4V3M5
- Pupils normal size, reactive to light
- Fundus : B/L papilloedema
- NCCT head : Multiple NCC with focal perilesional edema in frontal lobe



 Focal perilesional edema in frontal —-> altered behaviour • However does it explain a reduced state of awareness?



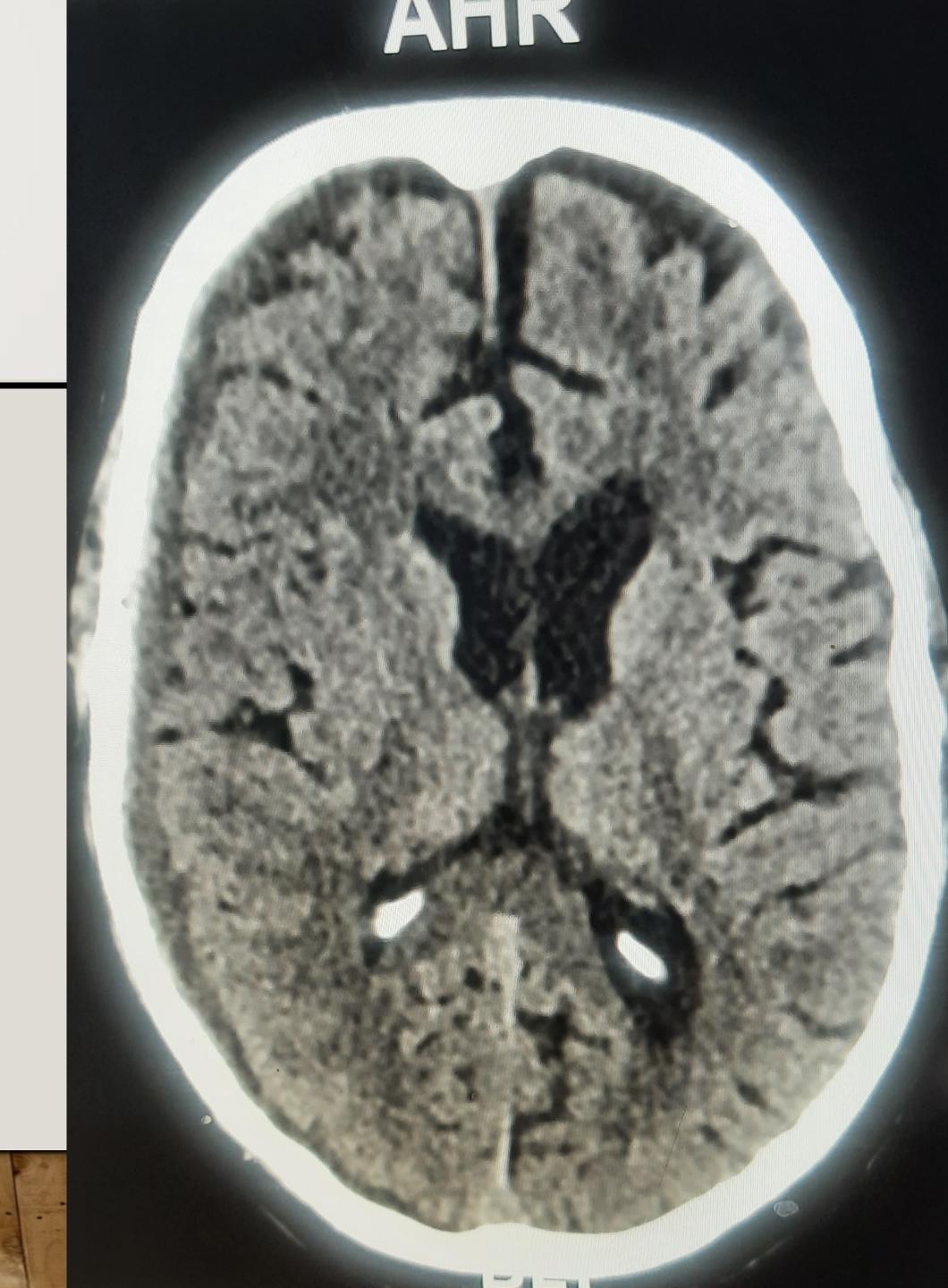


Clinical Vignette 6

- 83 y gentleman
- non-valvular atrial fibrillation on Apixaban
- right parietal subdural hematoma following a traumatic fall.
- CEEG-diffuse slowing with C4-T4 spike-wave activity. Focal status epilepticus
- treatment at discharge: 100 mg BD
 brivaracetam, 200 mg BD lacosamide, and
 10 mg BD clobazam).

Readmitted with reduced level of consciousness

- (GCS) score was E3V3M5
- (CT) of the head revealed no significant changes
- EEG diffuse theta slowing (not responsive to midazolam)
- serum ammonia 197 micromole/L



DRUGS CAUSING HYPERAMMONEMIA

- Valproate
- Topiramate
- Carbamazepine
- 5-FU
- Rifabutin
- Acetazolamide

HOW TO SEND AMMONIA LEVELS:

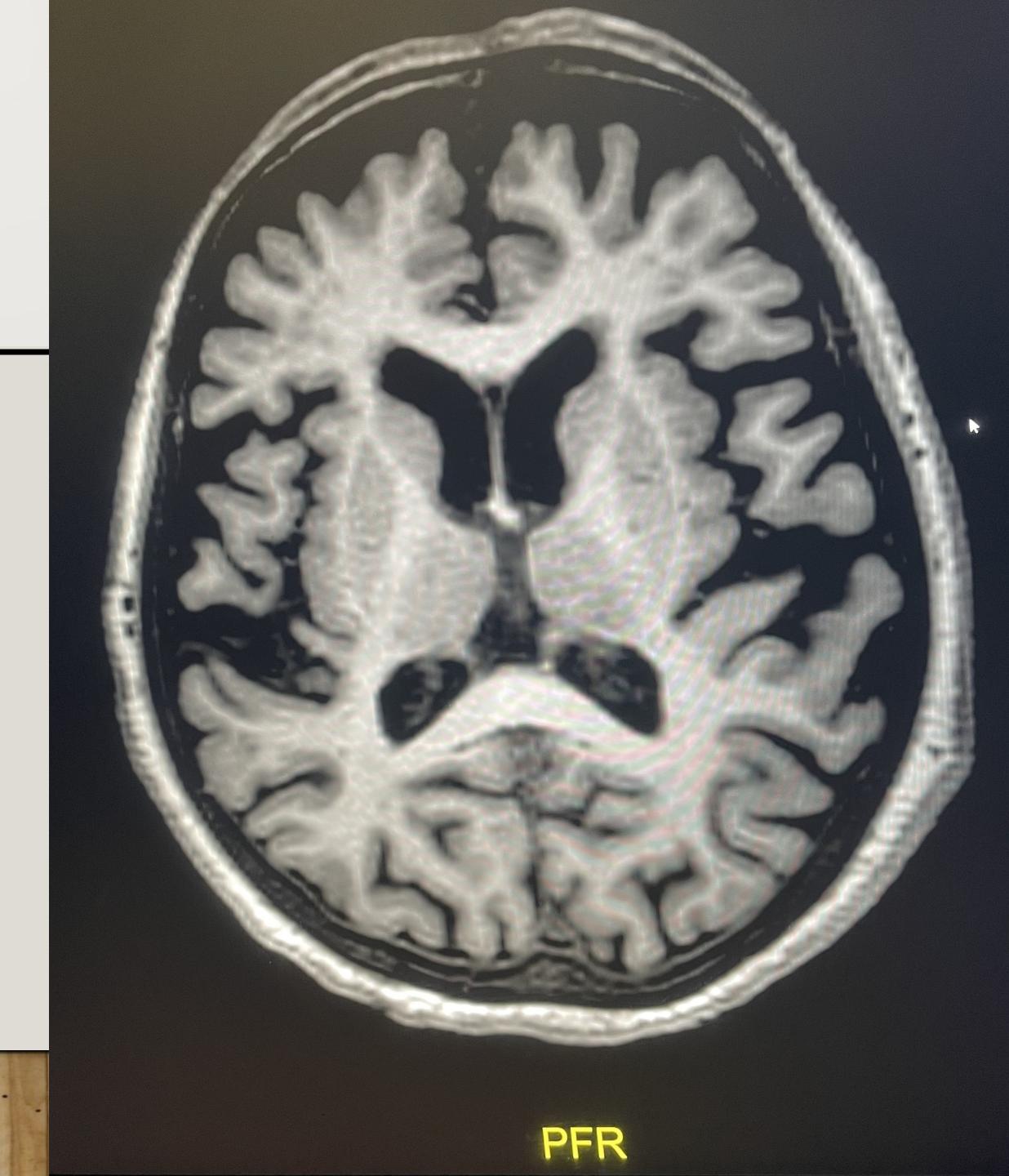
- 1. Pre prandial (3-4 hr) sample
- 2. Without any tourniquet
- 3. Preferably pre chilled but not frozen

Ref: Meijer R, Vivekananda U, Balestrini S, Walker M, Lachmann R, Haeberle J, Murphy E. Ammonia: what adult neurologists need to know. Pract Neurol. 2020 Dec

- Laboratory Investigations to Rule out Metabolic encephalopathy:
- RBS
- ABG
- ECG
- CBC, LFT, RFT
- Serum Electrolytes: Na+, Ca2+, Mg2+
- Tox screen
- CXR

Clinical Vignette 7
88 y gentleman, HTN
Admitted to medicine with low grade fever
6h after admission patient became incoherent,
agitated, confused
Sr Electrolytes - WNL

Urine R/E, C/S awaited



DELIRIUM

Acute change in attention, awareness, cognition

- Failure of attention
- Distractibility
- Perseveration
- Failure to focus
- Patients in early metabolic encephalopathy
 - disoriented to time—->place——>person
- Hallucinations
- Altered sleep wake cycle
- Reversible cognitive decline

PRECIPITATING FACTORS FOR DELIRIUM

- 208 patients
- Infections (49.5%) M/C Lung > UTI
- Fluid and electrolyte disturbances
- Drugs (30.8%) M/C Bzd>anti-depressants> anti-psychotics

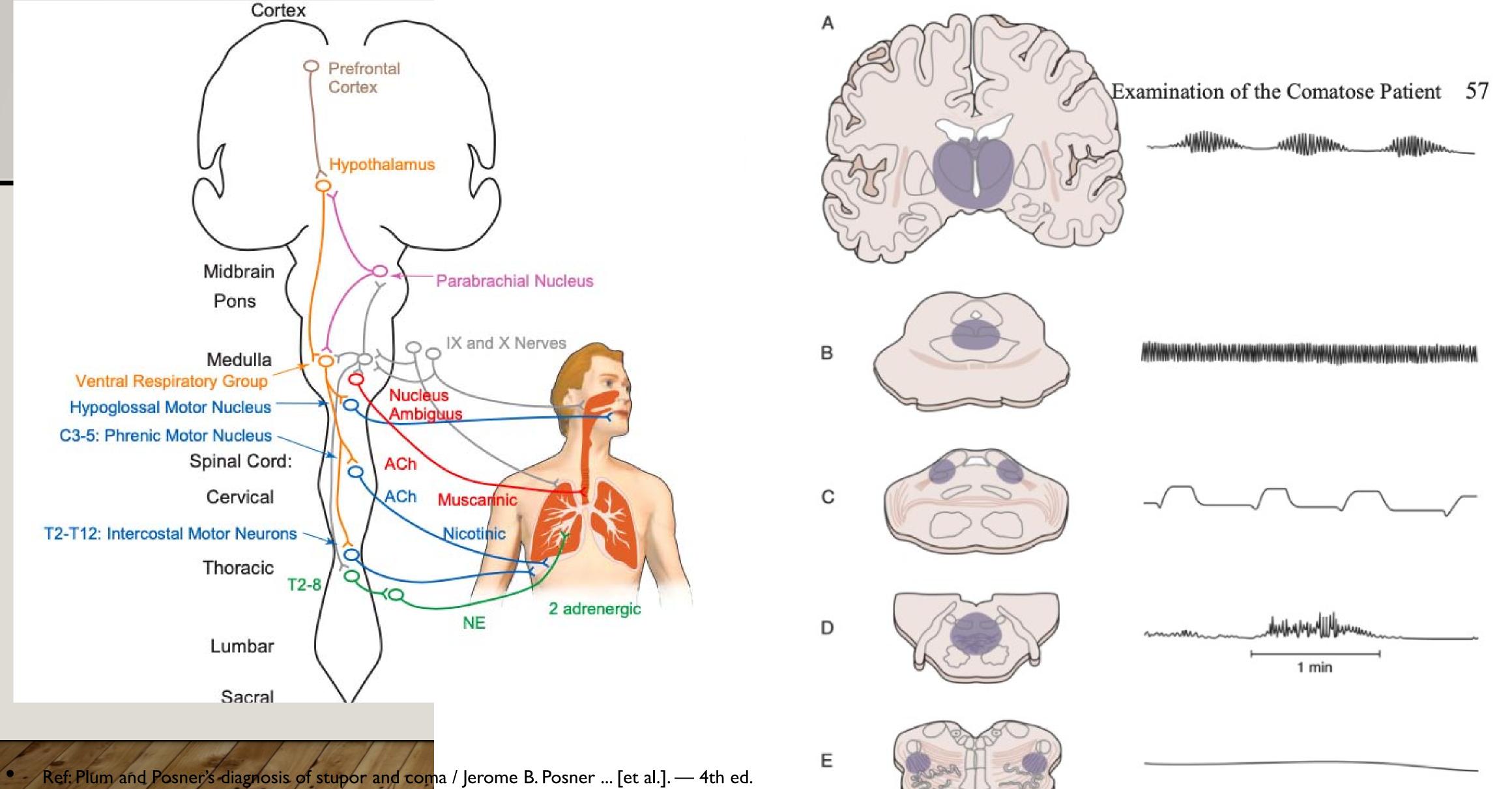
Clinical Vignette 8

- 15 y male Acute onset progressive drowsiness over last 2-3 days
- O/E E2V2M5
- Pupils small, reactive to light
- Kussmaul breathing
- Moves all 4 limbs to pain
- Reflexes brisk
- Terminal Neck Rigidity +
- Dehydration ++

- RBS = 490
- Ketone bodies in urine positive

BREATHING

In intubated patients, observe in PSV



CHEYENES STOKES RESPIRATION

Requires intact brainstem functions

- Medullary chemoreceptors sense pAO2 and reduce respiratory drive
- pCO2 rises
- But there is delay in transit time
- By then PCO2 is at higher levels
- Ramped up respiration
- By the time brain sense fall in PCO2, ACo2 falls further
- Respiration slows or ceases
- So the periodic cycling is due to delay in the feedback loop

KUSSMAUL BREATHING

Points toward Metabolic cause

- Deep slow rhythmic breathing
- Low blood pH——-> deep respiratory efforts
- Compensatory Respiratory alkalosis

• Ref: Plum and Posner's diagnosis of stupor and coma / Jerome B. Posner ... [et al.]. — 4th ed.

Clinical Vignette 9

- 21 y F, k/c/o pallidopyramidal syndrome
- Fever + drowsiness for 8 days
- Intubated in ER for poor GCS
- E1VtM5
- Pupils small, reactive
- Tone Rigidity + +
- Reflexes 3 +
- B/L Plantars Flexor

Review history: Stopped Syndopa for last 2 weeks

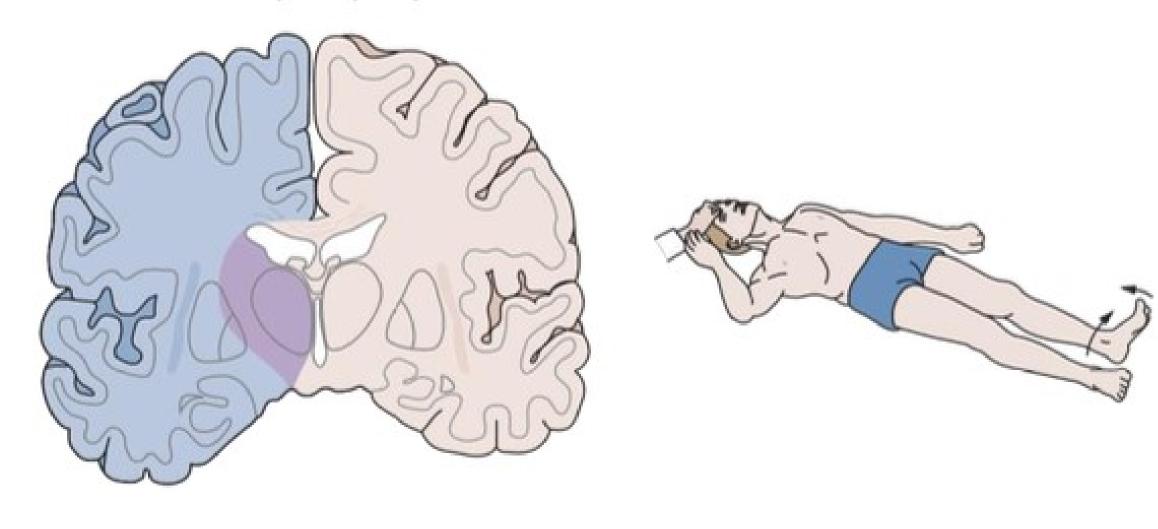
CK 10,510

• LDH 2800

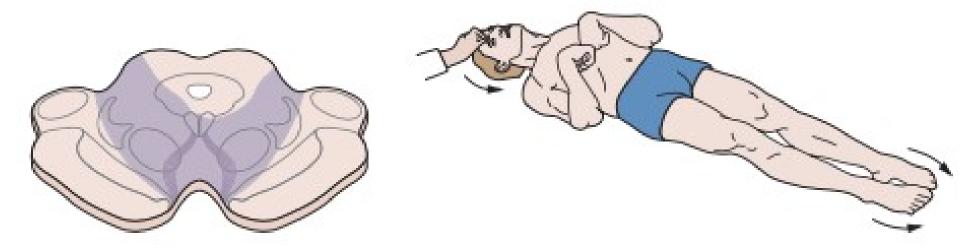
Motor Responses in AMS

- Diffuse metabolic encephalopathy Gegenhalten
- As patients become more stuporous muscle tone tends to decrease and pathologic forms of rigidity becomes less apparent
- Frontal release signs may appear

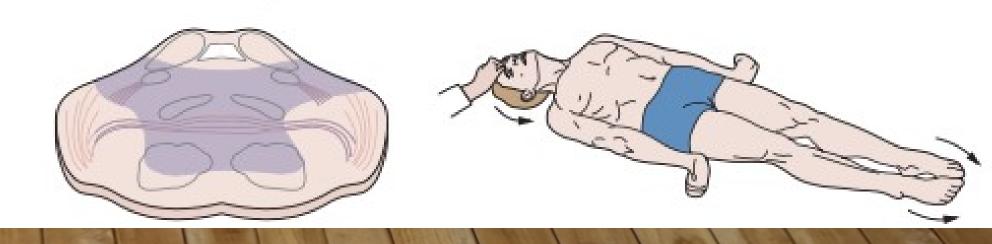
A Metabolic encephalopathy



B Upper midbrain damage



C Upper pontine damage

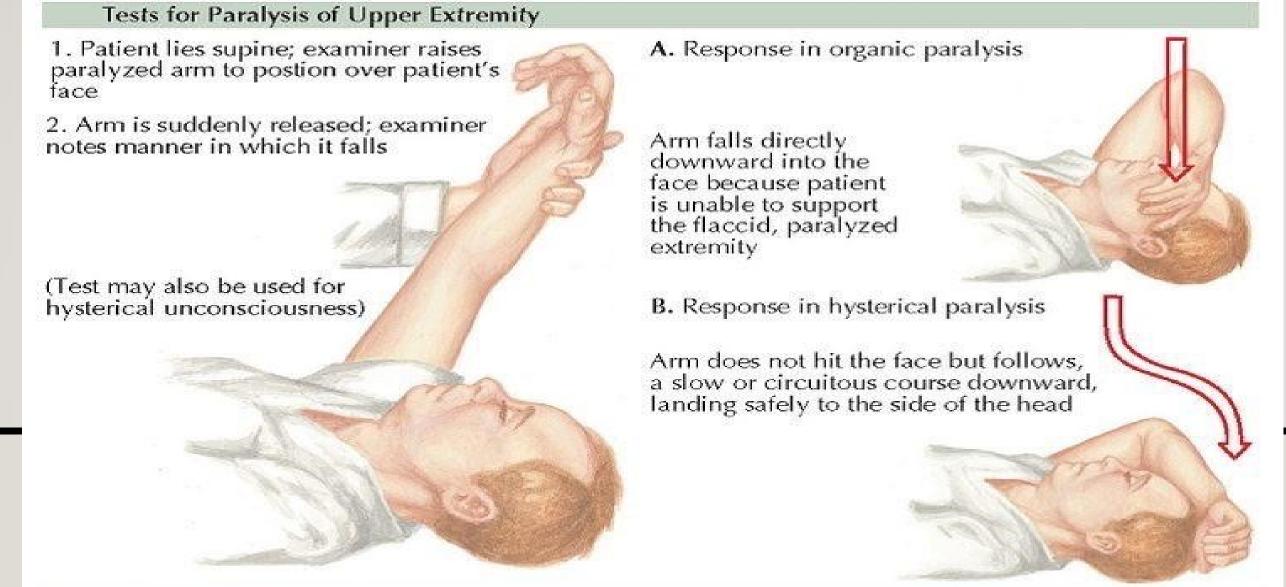


Clinical Vignette 10

- 36 y, M, alcoholic
- Sudden B/L loss of vision right after
 landing
- 1 episode of B/L tonic clonic seizures
- Rapidly progressed to E1V1M1
- NCCT Head Normal

	pH	6.845
	HCO3-	6.1
	pCO2	35
		20.9

• On further review of history: Consumption of illicit liquor a few hours prior to boarding the flight

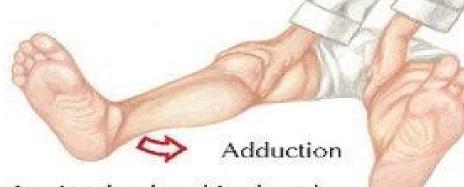


Only 1% of all emergency cases

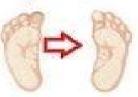
Tests for Weakness in Lower Extremity

Thigh adduction test

1. Patient is instructed to adduct "good" leg against resistance by examiner

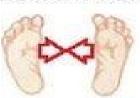


2. Examiner's other hand is placed against "paralyzed" thigh to detect contraction



A. Response in organic paralysis

Patient can accomplish adduction with no contralateral adduction palpable in paralyzed leg "paralyzed" leg



B. Response in hysterical paralysis

In adduction of "good" leg, patient involuntarily adducts

Hoover test

1. Patient is instructed to elevate "good" leg against resistance by examiner

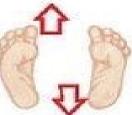


Examiner's other hand is placed beneath heel of "paralyzed" leg to detect reciprocal downward thrust used by patient for leverage



A. Response in organic paralysis

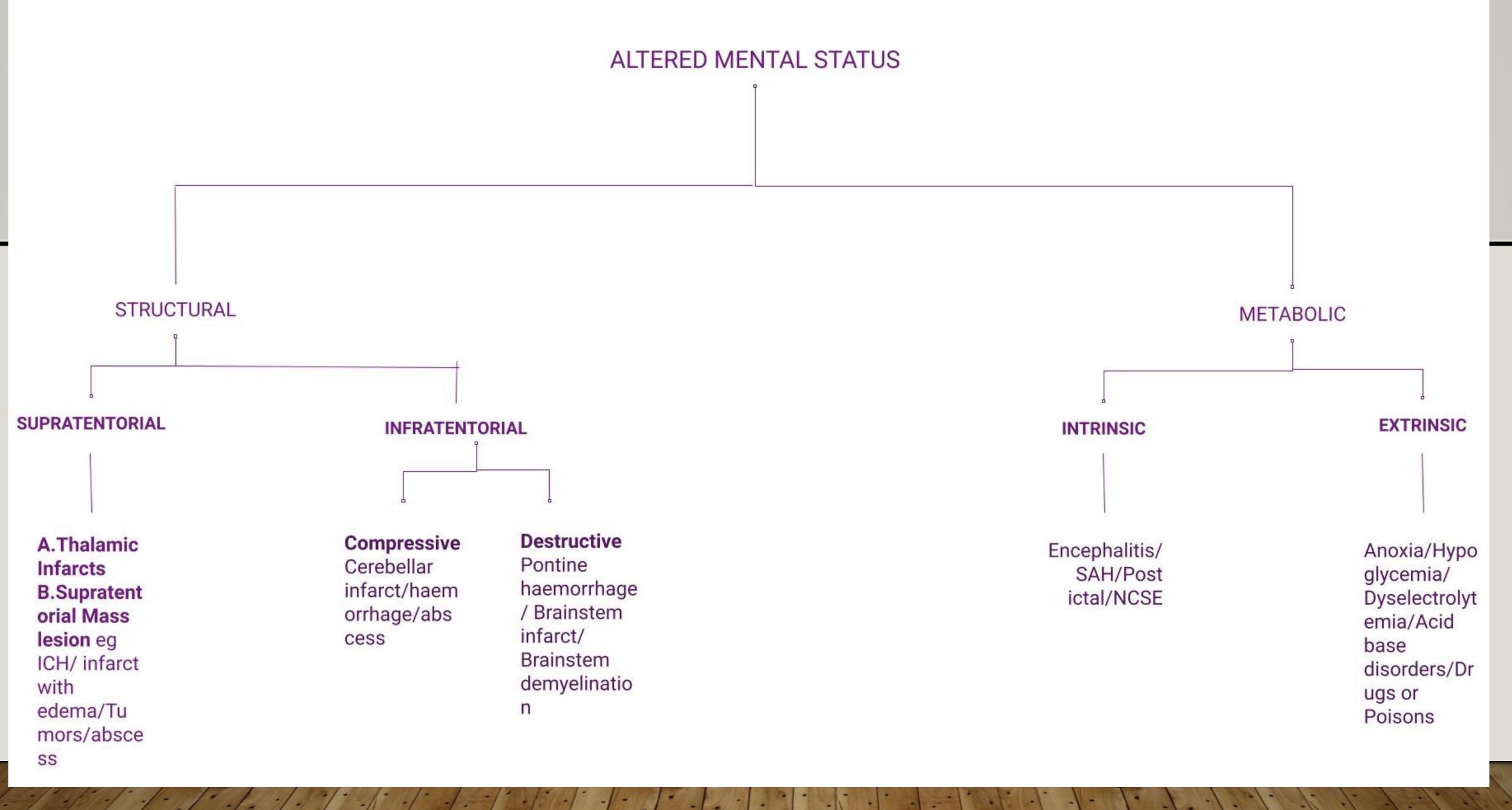
Patient is able to elevate good leg without concomitant downward thrust of paralyzed leg



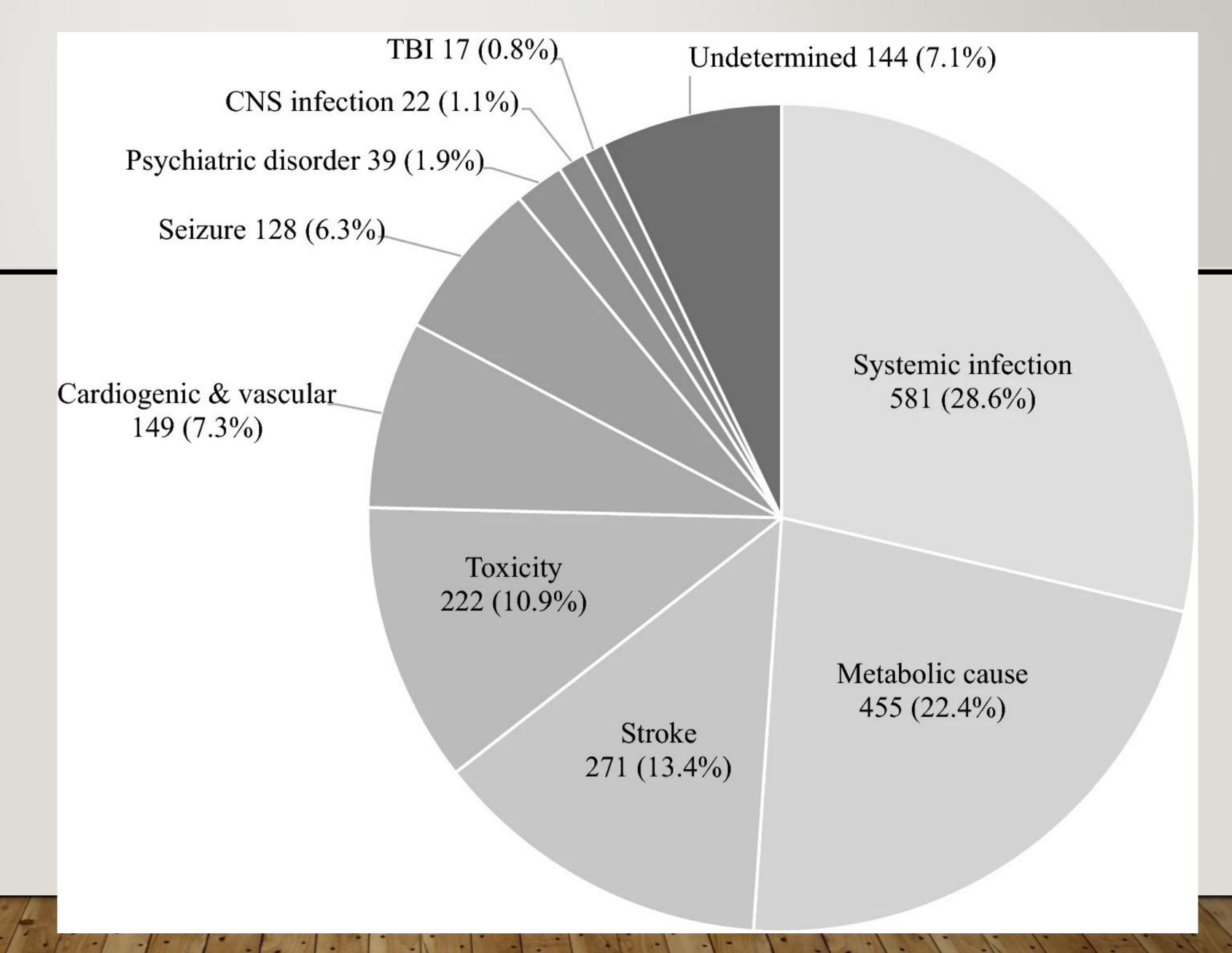
B. Response in hysterical paralysis

Elevation of "good" leg is accompanied by downward thrust of "paralyzed" leg

Ref: Kim, K.T., Jeon, J.C., Jung, CG. et al. Etiologies of altered level of consciousness in the emergency room. Sci Rep 12, 4972 (2022).



- Etiologies of altered mental status in ER
- Analysed 2028
 discharges from ER
 admitted with
 Altered level of
 consciousness
- UniversityHospital, 2018-2020



- small pupils (<2 mm) opioid toxicity or a pontine lesion
- midsize pupils (4–6 mm) unresponsive to light midbrain lesion
- maximally dilated pupils (>8 mm) drug toxicity, eg anticholinergie overdose
- mixed and dilated pupil(s) 3rd (oculomotor) nerve lesion from uncal herniation.

THE PATTERN OF BREATHING SHOULD BE ASSESSED AS WELL AS THE RESPIRATORY RATE

- •KUSSMAUL RESPIRATION DEEP, LABOURED BREATHING, INDICATIVE OF SEVERE METABOLIC ACIDOSIS AND COMMONLY ASSOCIATED WITH DIABETIC KETOACIDOSIS.
- •SHALLOW WITH AN EXTREMELY DEPRESSED RESPIRATORY RATE SEEN IN OPIATE OVERDOSE.
- •ATAXIC BREATHING (BIOT'S RESPIRATION) GROUPS OF QUICK, SHALLOW INSPIRATIONS FOLLOWED BY REGULAR OR IRREGULAR PERIODS OF APNOEA, SUGGESTING A LESION IN THE LOWER PONS.11
- •CENTRAL NEUROGENIC HYPERVENTILATION BREATHING CHARACTERISED BY DEEP AND RAPID BREATHS AT A RATE OF AT LEAST 25 BREATHS PER MINUTE INDICATING A LESION IN THE PONS OR MIDBRAIN.12

•CHEYNE–STOKES BREATHING IS SEEN WITH MANY UNDERLYING PATHOLOGIES AND IS NOT HELPFUL IN MAKING A fiRM DIAGNOSIS.

WHAT COULD BE AT ONCE FATAL!

Cardiac arrest

Airway obstruction

• Breathing (oxygenation)

WHAT COULD BE FATAL IN NEXT FEW MINUTES?

Hypoglycemia

Overdose

• Intracranial hypertension and herniation

- Neuro: Pupils, eye movements; moving all 4 extremeties?; reflexes; any asymmetry?
- Signs of impending herniation: Hypertension, bradycardia, irregular respirations, posturing, dilated pupil?
- Breathing pattern: Regular, Cheyne-Stokes, irregular, apnea?
- Toxidrome: Vital signs, pupils, skin
- Signs of shock: skin warm or cold? BP?
- Trauma: Any clear signs of trauma?
- Abdomen: Any obvious pain? Masses?

WHAT COULD BE FATAL IN NEXT 10 MINUTES?

- Still the ABCs!
- Hypotension
- Anaphylaxis
- Hyperkalemia
- Acute MI
- Aortic disasters

WHAT COULD BE FATAL IN NEXT FEW HOURS?

Stroke

ICH

Sepsis

Alcohol withdrawal

Status Epilepticus

Metabolic encephalopathies

Others

Summing up:

- STEP 1: A,B,C, Vitals, RBS
- STEP 2: History (Attention to baseline cognitive status, change in medication)
- Examination (Look for signs of Structural causes, FND)
- ABG, CBC, LFT, RFT(including Ca, Mg), ECG,CXR,Tox Screen
- STEP 3: CT f/b MRI if indicated
- Lumbar Puncture
- STEP4: Guided by Findings on initial

evaluation

• Serum ammonia, TFT, Morning Cortisol,

ANA, anti TPO, EEG

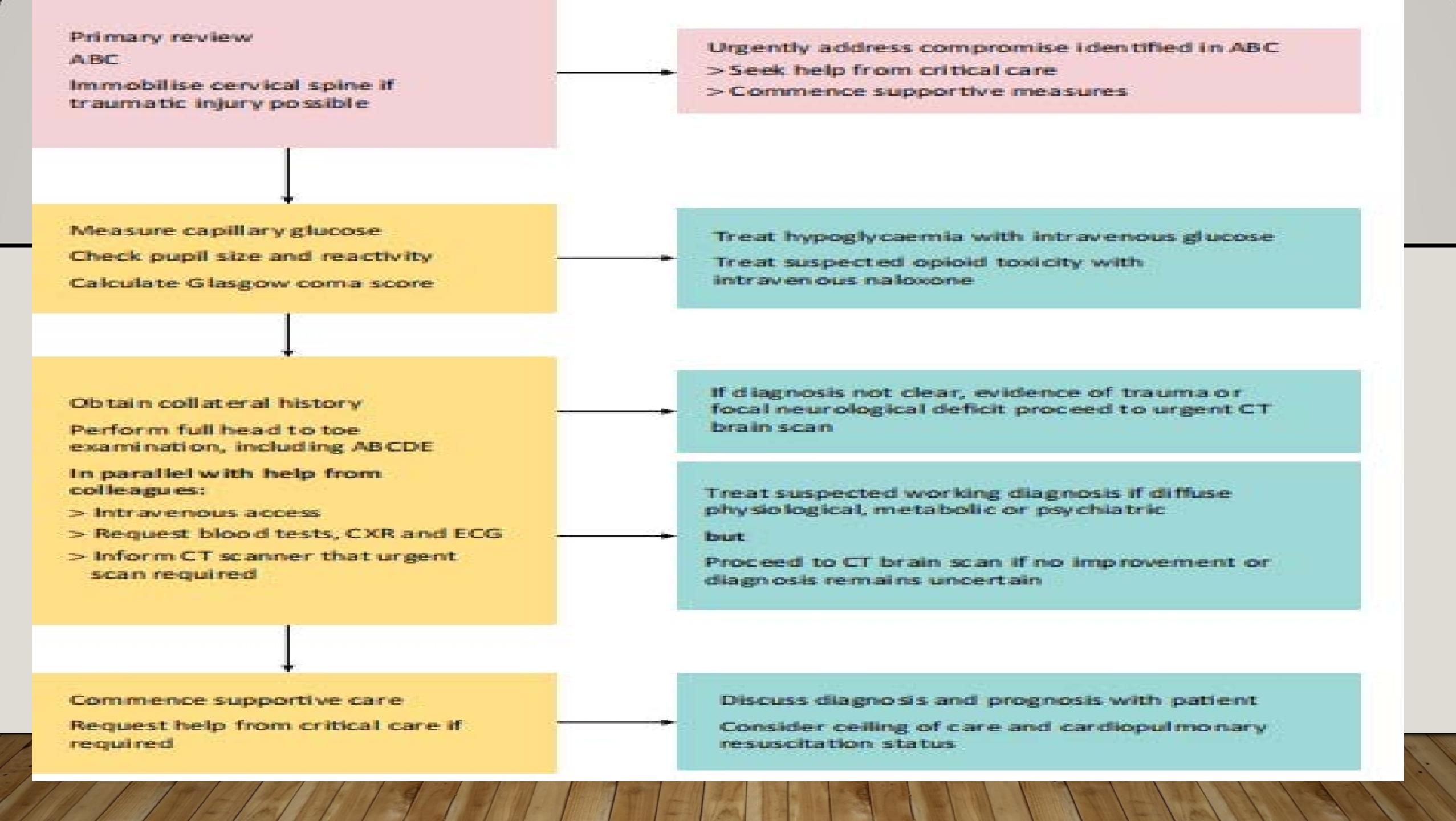
INITIAL INVESTIGATIONS IN AN UNCONSCIOUS PATIENT

- Full blood count
- Blood glucose even if the capillary blood glucose is normal
- Urea and electrolytes
- Calcium and bone profile
- Liver function tests
- Clotting screen
- Toxicology screen including paracetamol, salicylate and blood alcohol level electrocardiogram (ECG)
- Chest X-ray
- Arterial blood gas including carbon monoxide concentration
- Blood cultures should be taken from patients with fever or suspected sepsis, preferably before the administration of empirical antibiotics

• Other microbiology samples should be taken based on the clinical assessment

TREATMENT/ MANAGEMENT

- Ensure oxygenation
- Maintain circulation
- Control glucose
- Lower intracranial pressure
- Stop seizures
- Treat infection
- Restore acid-base balance and electrolyte balance
- Adjust body temperature
- Administer thiamine
- Consider specific antidotes (naloxone, flumazenil)
- Control agitation



UNCONSCIOUSNESS IS A TIME-SENSITIVE MEDICAL EMERGENCY WHERE EARLY PHYSIOLOGICAL STABILITY AND DIAGNOSIS ARE VITAL IN OPTIMISING PATIENT OUTCOMES

AN INITIAL ASSESSMENT OF AIRWAY, BREATHING, AND CIRCULATION MUST BE PERFORMED TO IDENTIFY AND MANAGE THE MOST IMMEDIATE THREATS TO LIFE

ALL FACETS OF CARE, HISTORY, EXAMINATION, INVESTIGATION AND TREATMENT/MANAGEMENT SHOULD BE DELIVERED IN PARALLEL BY A TEAM WORKING IN A SYSTEMATIC WAY

EVEN IN THE APPARENT ABSENCE OF TRAUMA, ESPECIALLY IN OLDER PATIENTS OR PATIENTS TAKING ANTICOAGULANTS, BRAIN INJURY OR TRAUMA SHOULD STILL BE CONSIDERED

SENIOR PHYSICIANS MUST BE INVOLVED EARLY IN THE CARE OF AN UNCONSCIOUS PATIENT, TO LIAISE WITH CRITICAL CARE AND SPEAK WITH THE PATIENT'S RELATIVES OR ADVOCATES, ESPECIALLY WHEN DECISIONS REGARDING CARDIOPULMONARY RESUSCITATION OR CEILING OF CARE ARE REQUIRED

Thank You