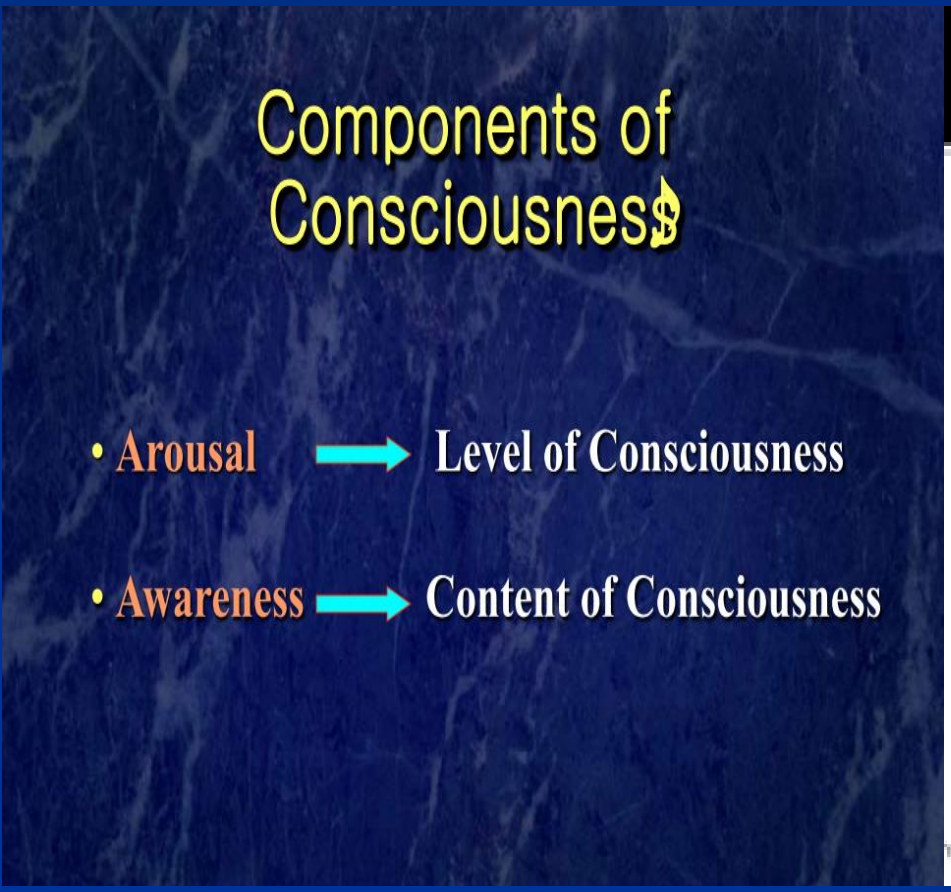
# Neurological Emergencies/ CNS Infections

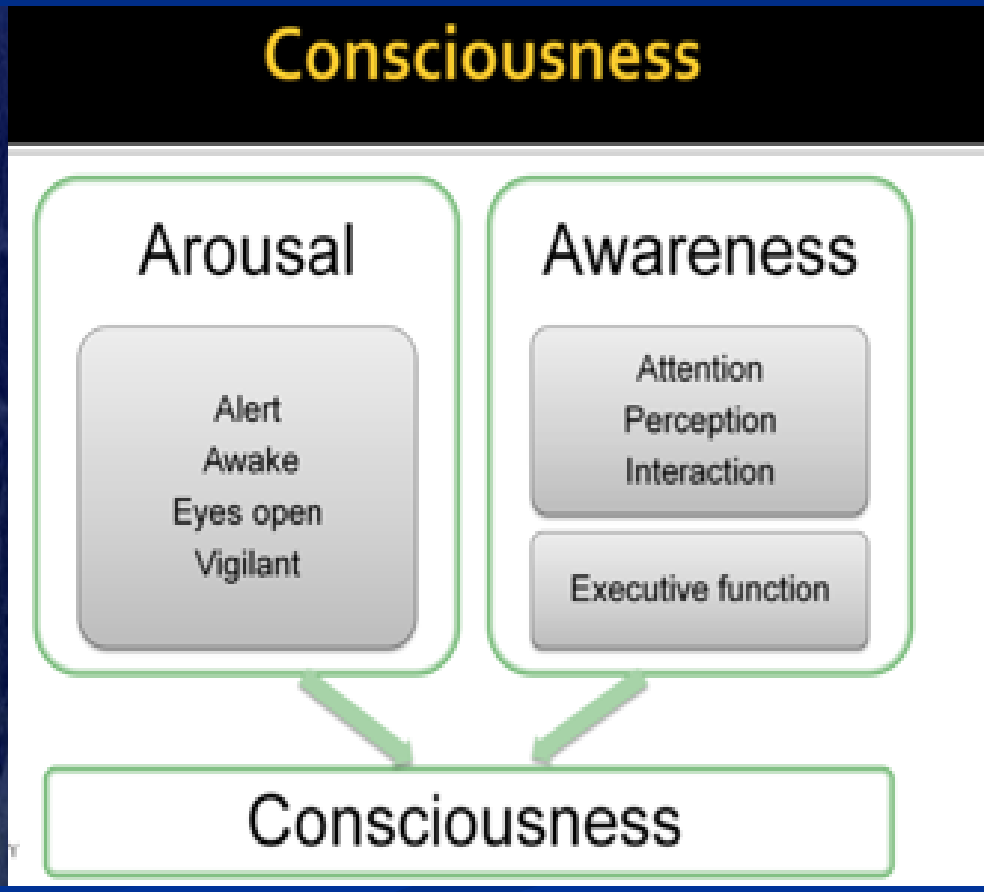
Majed Habahbeh MBBS FRCP

# Neurological Emergencies-At least 20% of Medical ER visits

- Coma.
- Meningitis/encephalitis
- Acute Stroke.
- Seizures/ Status epilepticus.
- Acute headaches/Subarachnoid hemorrhage.
- Acute flaccid paralysis - limbs, bulbar, respiratory (Guillain-Barre' Syndrome, Myasthenia Gravis…)
- Acute myelopathy/spinal cord compression
- Vision loss ( Optic Neuritis, Papilledema )
- Vertigo ( posterior circulation stroke)

# Components of Consciousness

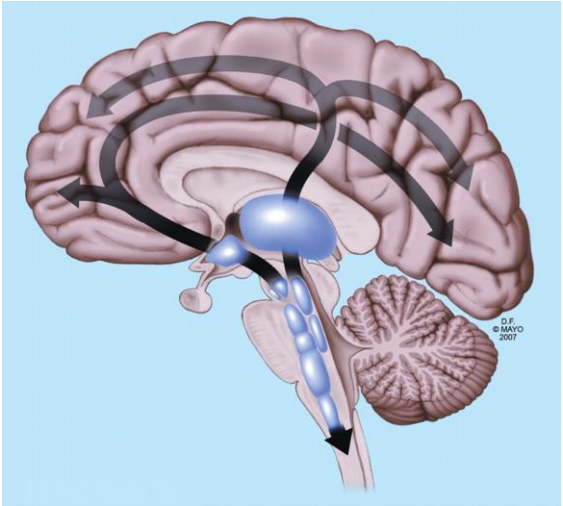


# Anatomy of consciousness

- Level of consciousness is regulated by the ascending reticular activating system in the midbrain and pons with projections to the thalamus and cortex.
- Content of consciousness

The main pathways connecting the ascending reticular formation with the thalamus and cortex.

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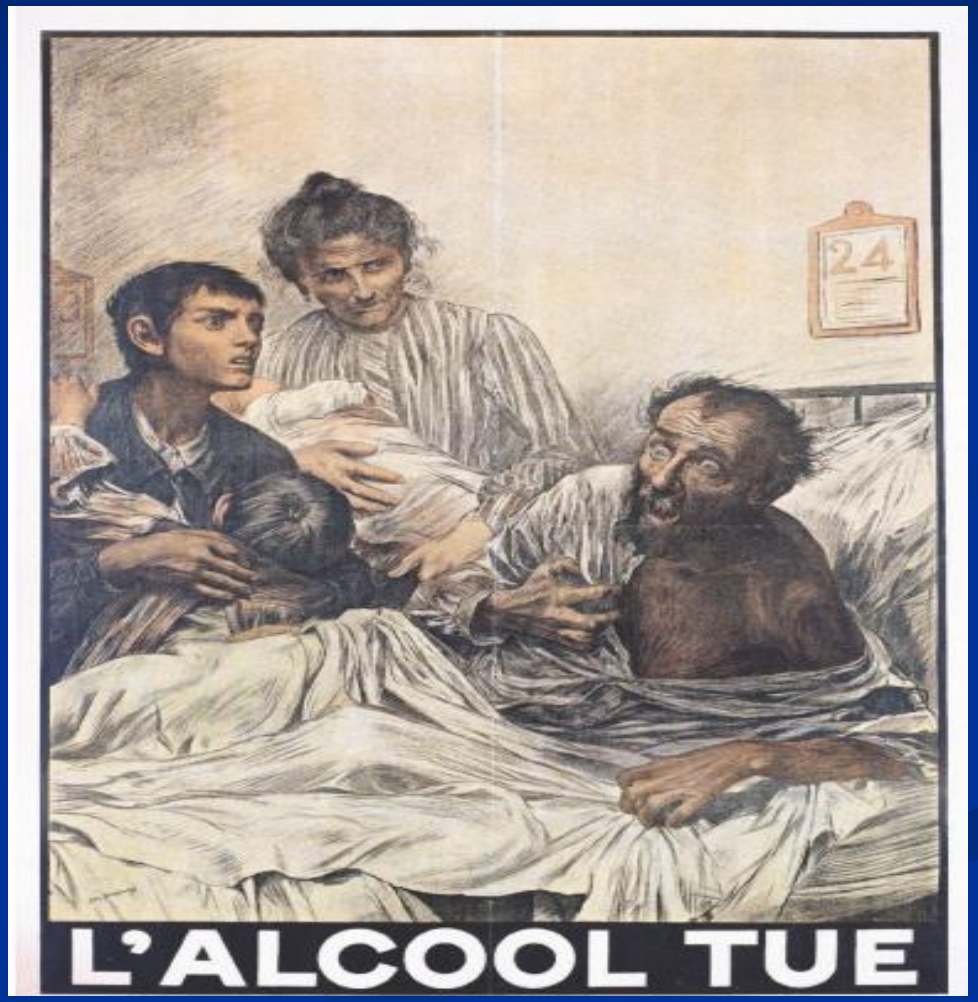
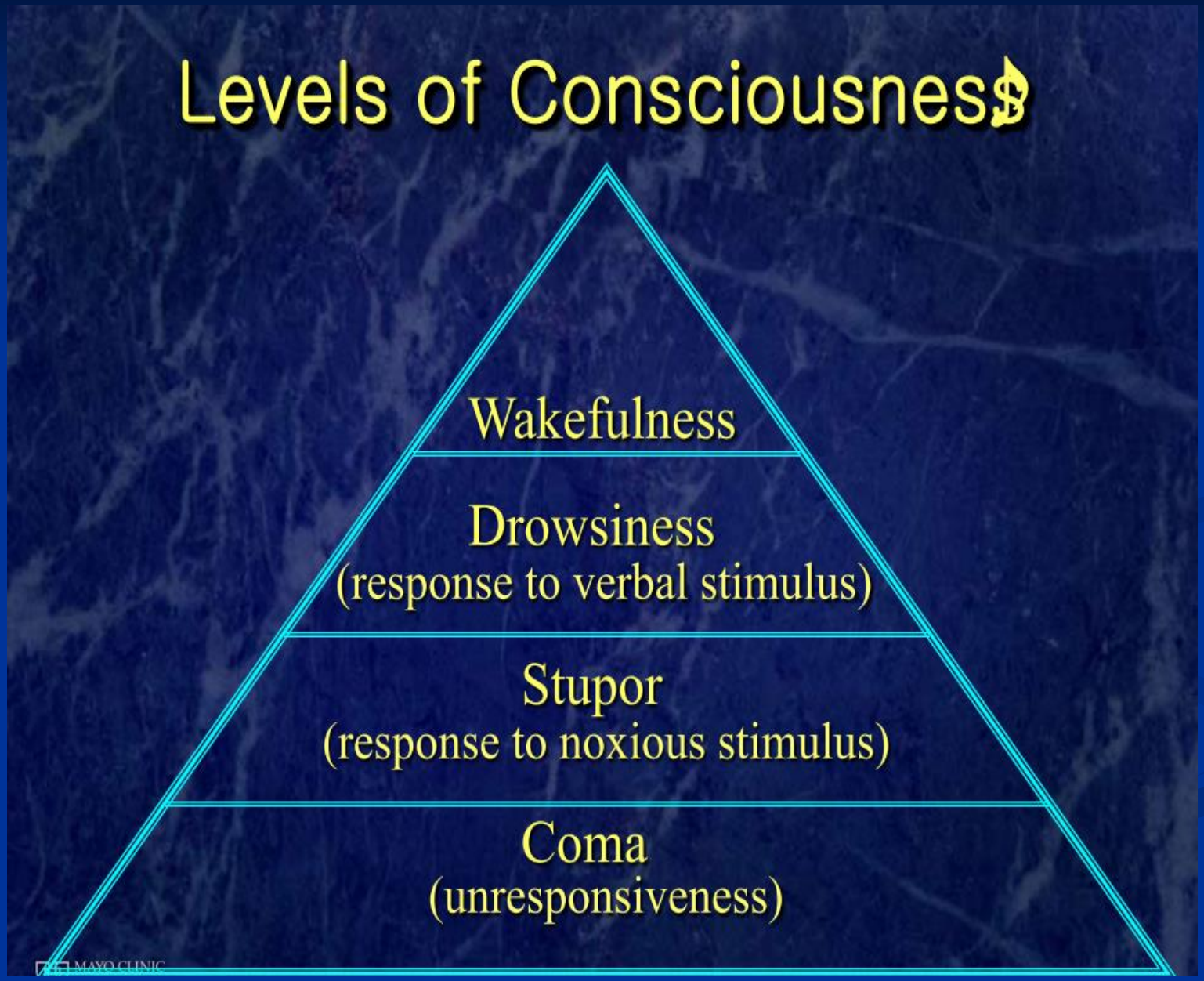
# Content of consciousness Terminology

- Acute confusional state
- Delirium

- Delirium is derived from the Latin verb delira—to deviate/ go crazy.



# Levels of Consciousness



| Glasgow Coma Scale | | |
|---|---|---|
| **Eye Response** | Open Spontaneously | 4 |
| | Open to Verbal command | 3 |
| | Open in response to pain | 2 |
| | No response | 1 |
| **Verbal Response** | Talking / Orientated | 5 |
| | Confused speech / Disorientated | 4 |
| | Inappropriate Words | 3 |
| | Incomprehensible sounds | 2 |
| | No response | 1 |
| **Motor Response** | Obeys commands | 6 |
| | Localizes pain | 5 |
| | Withdraws from pain | 4 |
| | Abnormal flexion | 3 |
| | Extension | 2 |
| | No response | 1 |

## Spectrum of Acute Brain Dysfunction


| RASS | -5 | -4 | -3 | -2 | -1 | 0 | +1 | +2 | +3 | +4 |
|---|---|---|---|---|---|---|---|---|---|---|
| | **Unarousable:** No response to voice or physical stimulation | **Deep sedation:** No response to voice, but responds to physical stimulation | **Moderate Sedation:** Responds to voice, but does not make eye contact | **Light Sedation**: Responds to voice, but can only make eye contact for < 10 seconds | **Drowsy:** Responds to voice and can make eye contact for > 10 seconds | **Alert and calm** | **Restless:** Anxious, but movements not agressive | **Agitated:** Frequent, non-purposeful movement | **Very Agitated:** Pulls or removes tubes or catheters, agressive | **Combative:** Overtly combative, violent, danger to staff |

Coma is defined as a completely unawake patient unresponsive to external stimuli

Brainstem reflexes can be intact or absent

# Evaluation of the Comatose Patient

- All causes of coma fall into one of the following major categories :

1. Structural injury of both cerebral hemisphere or one hemisphere causing mass effect and midline shift.

2. Intrinsic brainstem injury, or compression from surrounding damaged tissue (Cerebellum)

3. Acute metabolic or endocrine derangement

4. Diffuse physiological brain dysfunction-seizures, anoxia, drug toxicity

# Structural brain injury of Cerebral hemisphere(s)

## Unilateral with displacement

- Intraparenchymal hematoma
- Middle cerebral artery ischemic stroke
- Intracranial venous thrombosis
- Cerebral abscess
- Brain tumor
- Subdural or extradural hematoma

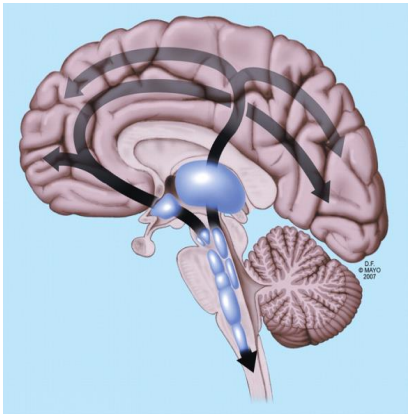
## Bilateral

- Subarachnoid hemorrhage
- Traumatic brain injury
- Multiple cerebral infarcts
- Bilateral thalamic infarcts
- Tumors
- Encephalitis
- Cerebral edema
- Acute hydrocephalus
- Posterior reversible encephalopathy syndrome (PRES)
- Air or fat embolism.

# Intrinsic brainstem injury, or compression from surrounding damaged tissue

- Pontine hemorrhage
- Basilar artery occlusion and brainstem infarct
- Central pontine myelinolysis
- Brainstem hemorrhagic contusion
- Cerebellar infarct
- Cerebellar hematoma
- Cerebellar abscess
- Cerebellar glioma

The main pathways connecting the ascending reticular formation with the thalamus and cortex.



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## CAUSES OF COMA WITHOUT FOCAL SIGNS

| | |
|---|---|
| Toxic | Alcohol, carbon monoxide, lead, cyanide, thallium, sedative drugs |
| Metabolic | Uraemia, hyperammonaemia, neuroleptic malignant syndrome, anoxic-ischaemic encephalopathy, hypercarbnia, hypo/hyper-natraemia, hypo/hyper-calcaemia, hypermagnesaemia, hypoglycaemia, hypothermia, hyperpyrexia, Wernicke's |
| Epileptic | Convulsive/non-convulsive status epilepticus |
| Endocrine | Hypopituitarism, hypothyroidism, hyperthyroidism, hypoadrenalism, Hashimoto's encephalopathy |

# Acute metabolic/endocrine derangement

- Hypoglycemia (<40-50 mg/dl )
- Hyperglycemia ( Ketotic and Non-ketotic)
- Hyponatremia (<110 mmol/l )
- Hypernatremia ( >160 mmol/l )
- Hypercalcemia ( >13.5 mg/dl )
- Hypercapnia ( >65 mmHg )

# What can mimic coma

## 1- locked-in syndrome

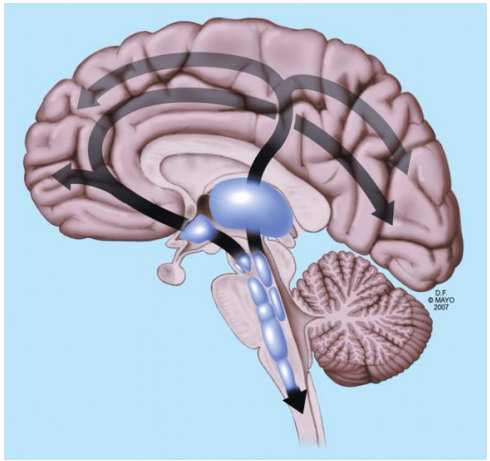
- Eyes open.
- Blink to commands or move their eyes vertically.
- lesion (stroke) in the ventral pons damaging the corticospinal and corticobulbar tracts and sparing the ascending reticular activating system. So they can hear, see and feel pain.

**Patients can be intubated by mistake.**

## 2- Psychogenic unresponsiveness

- Hysterical coma
- Malingering
- **Acute catatonia**

The main pathways connecting the ascending reticular formation with the thalamus and cortex.



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# Psychogenic unresponsiveness

Considered after exclusion of other causes !!

- The hand drop test is a useful test (one arm is lifted and held in front of the face and when let loose slides next to the patient's face rather than on to it).

- Closed eyes which open with tickling the nose hairs

- Some patients may have forced upward or downward gaze that may suddenly change in direction.

- Others have pseudoseizures- 'fish out of water' flopping.

# Examination of the comatose patient

1. Assess the depth of coma.

2. The location of the lesion, and

3. Possibly the underlying cause.

# Glasgow Coma Scale

| Glasgow Coma Scale | | |
|---|---|---|
| **Eye Response** | Open Spontaneously | 4 |
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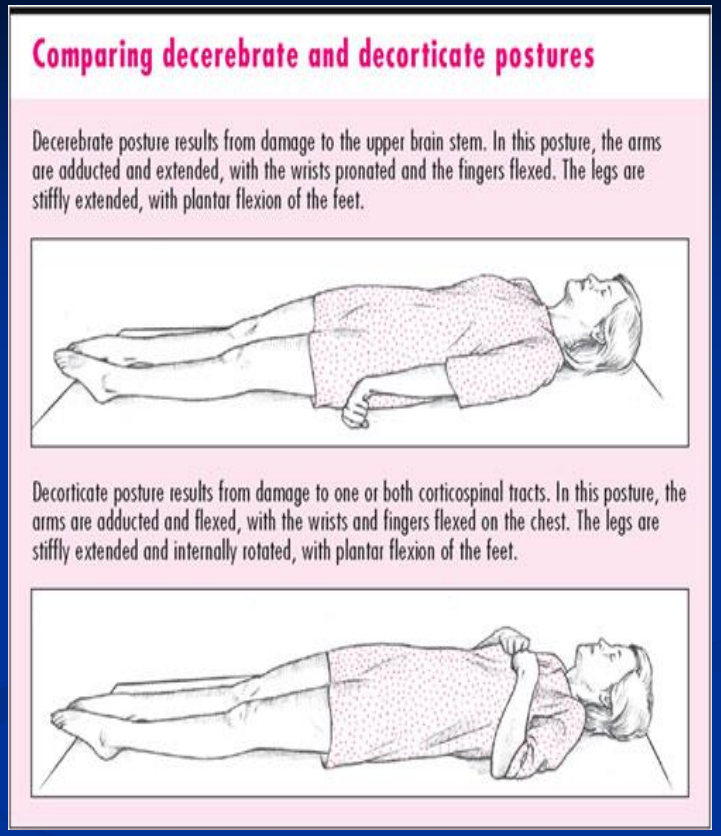



**Record subsets:**

**E( )+ M( ) + V( ) = ?/15**

**A score of < 8 usually indicates coma**

**Verbal response can be compromised by endotracheal intubation …. V(T) should be recorded.**

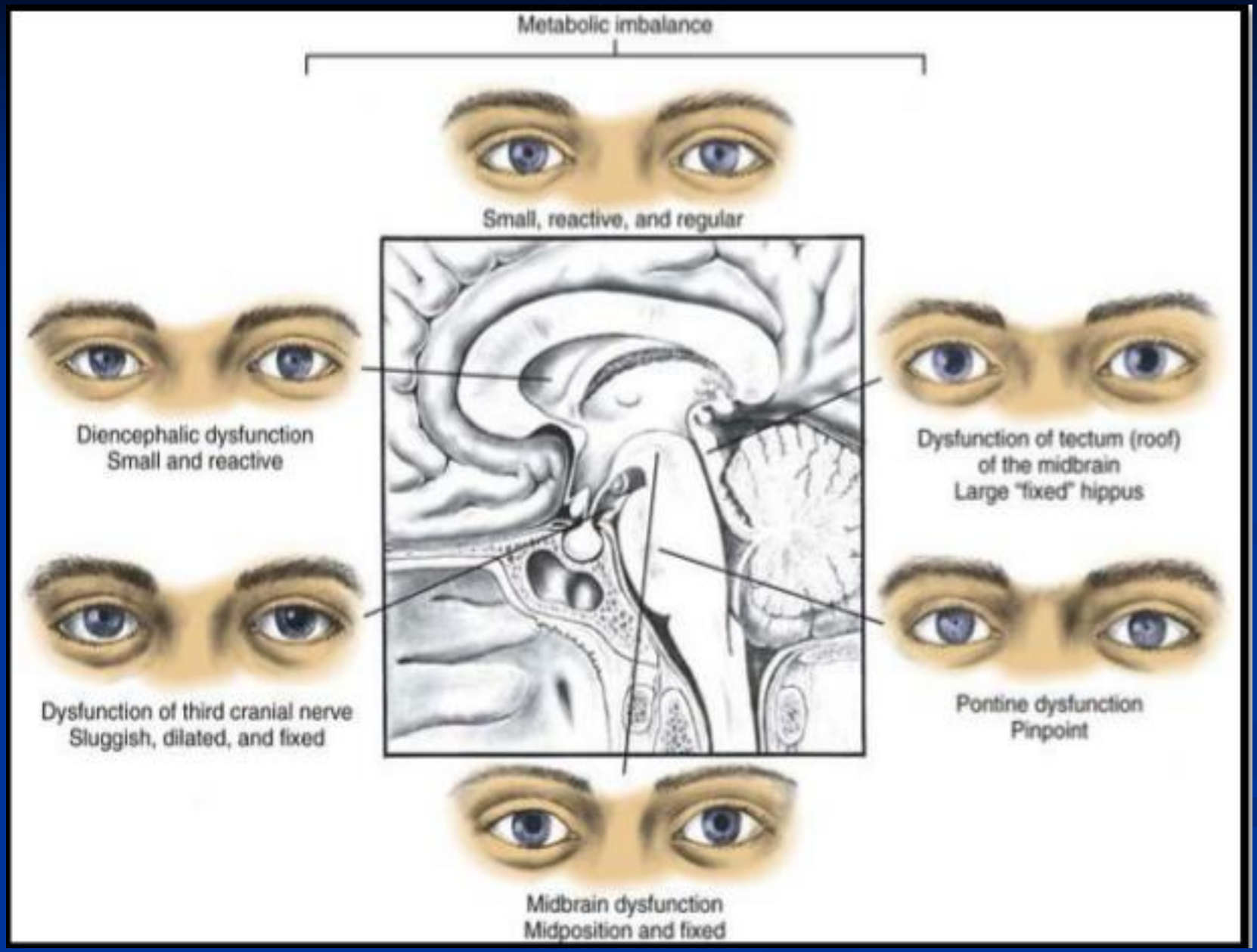
# Examination of the comatose patient

1. Assess the depth of coma.

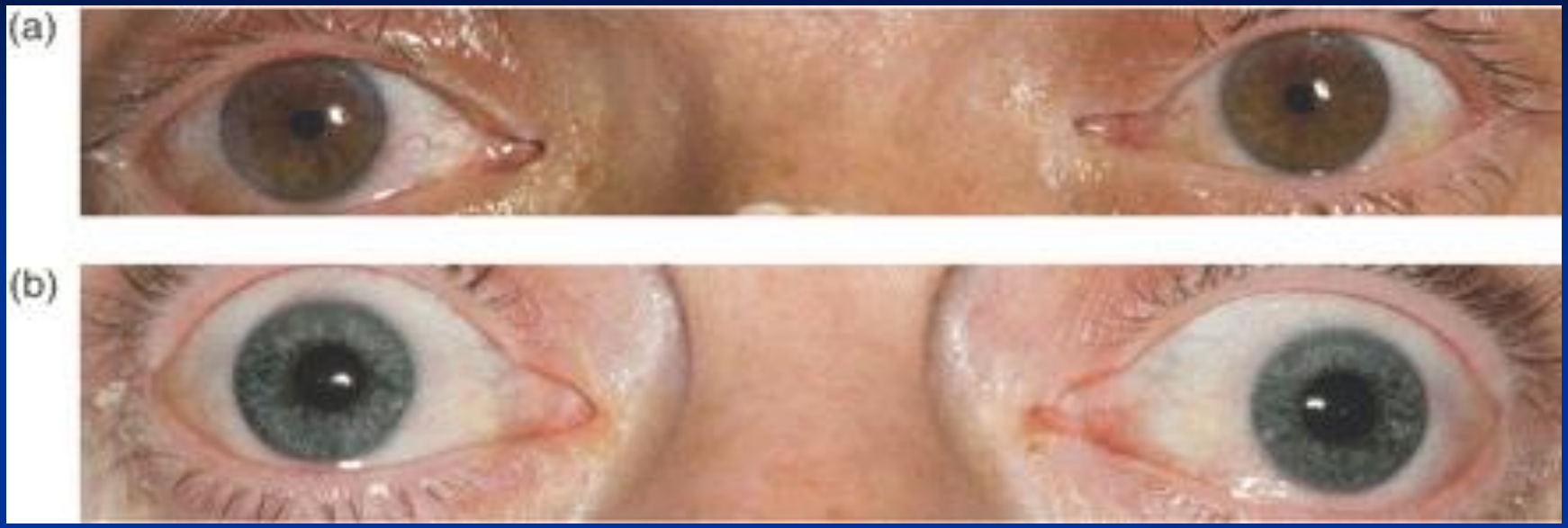
2. Determine if there is structural brain pathology and aim to localize it-

- meningism
- focal weakness
- pupils / eye position and movements
- DTR’s and plantar response

3. Determine the underlying cause if possible.


Metabolic imbalance
Small, reactive, and regular
Diencephalic dysfunction
Small and reactive
Dysfunction of tectum (roof)
of the midbrain
Large "fixed" hippus
Dysfunction of third cranial nerve
Sluggish, dilated, and fixed
Pontine dysfunction
Pinpoint
Midbrain dysfunction
Midposition and fixed

# Lesion Localisation




(a) Pinpoint pupils: opioid intoxication or pontine haemorrhage.

(b) Mid position light fixed pupils (mesencephalic lesion) in downward compression of the upper brainstem from a hemispheric mass but also often the first sign of loss of all brainstem reflexes (brain death).

# Lesion Localisation

- Roving eye movements indicate that the brainstem is intact.
- Skew deviation of the eyes suggests an acute brainstem injury.
- Horizontal deviation of the eyes to one side might be a sign of non-convulsive status epilepticus but also of an ipsilateral hemispheric or contralateral pontine stroke.

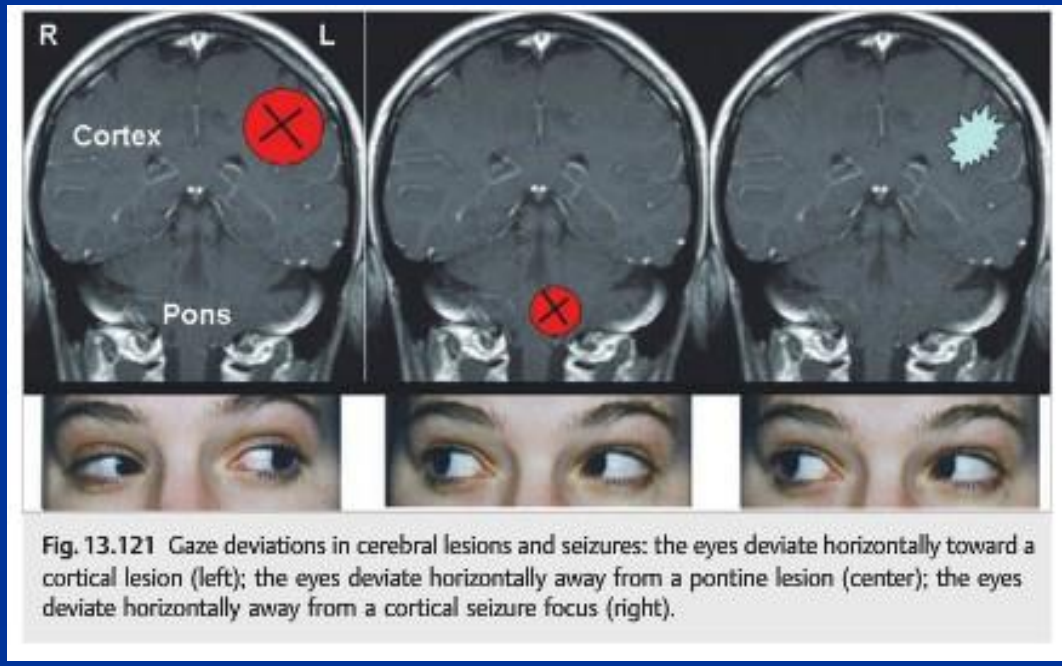
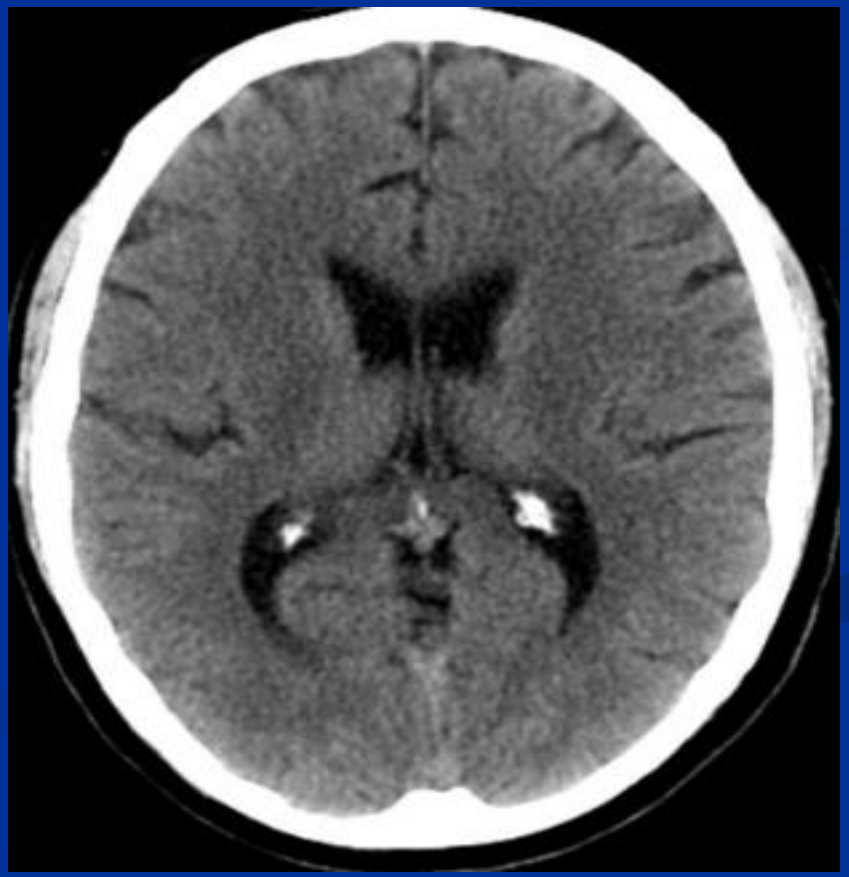


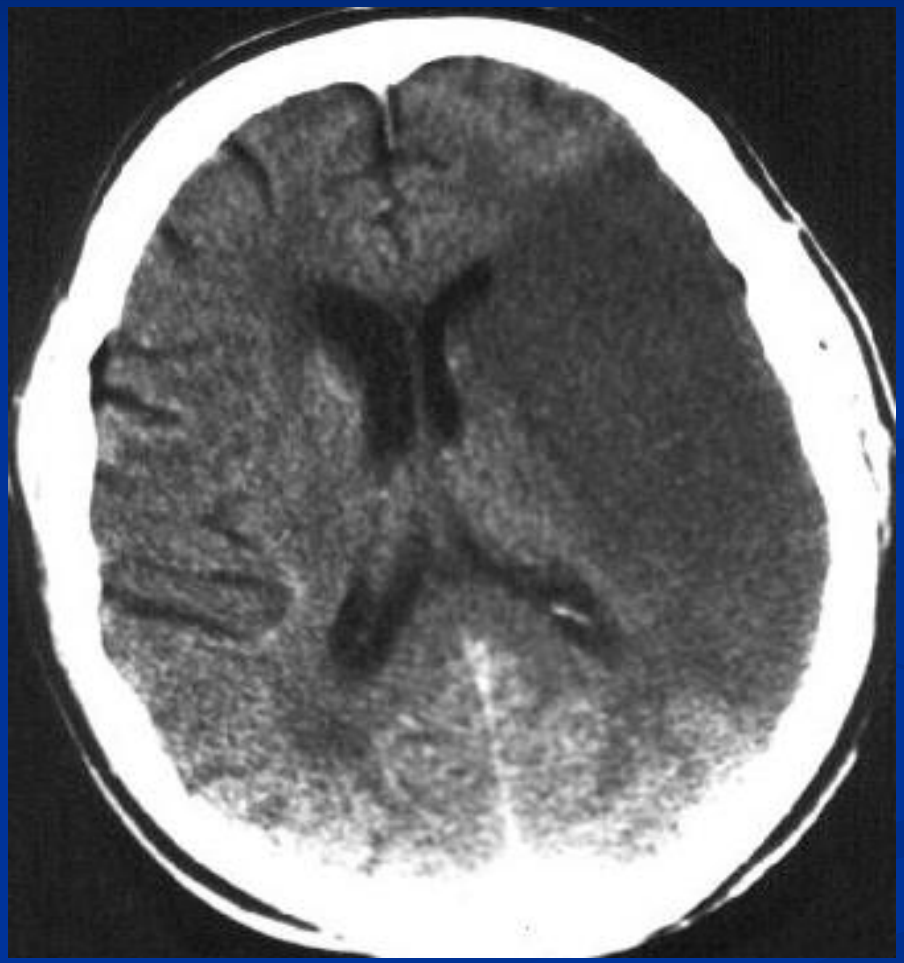

Fig. 13.121 Gaze deviations in cerebral lesions and seizures: the eyes deviate horizontally toward a cortical lesion (left); the eyes deviate horizontally away from a pontine lesion (center); the eyes deviate horizontally away from a cortical seizure focus (right).

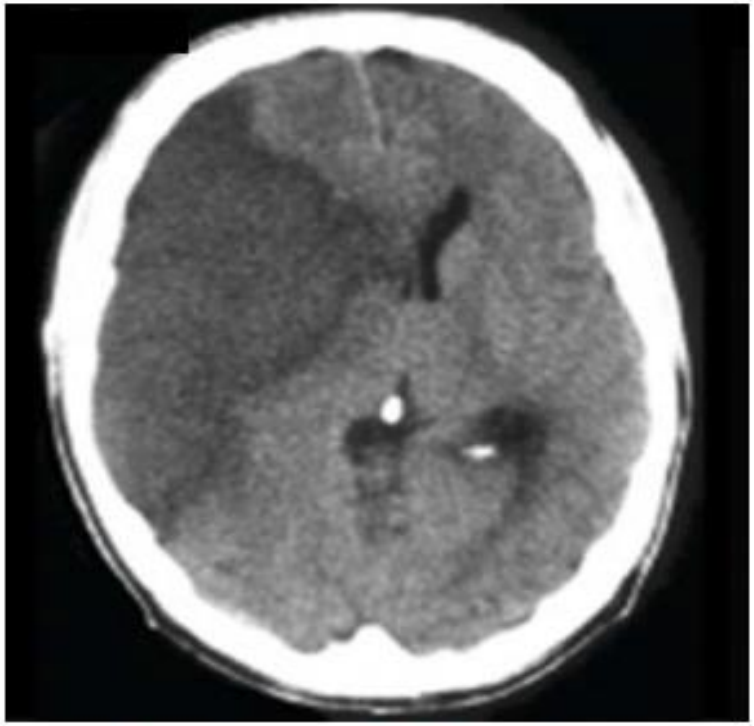
# What is the cause of coma ?

- CT and MRI of the brain are very important in the work-up of a comatose patient. However, in many cases of coma, the brain CT may be normal or only show minor subtle findings.

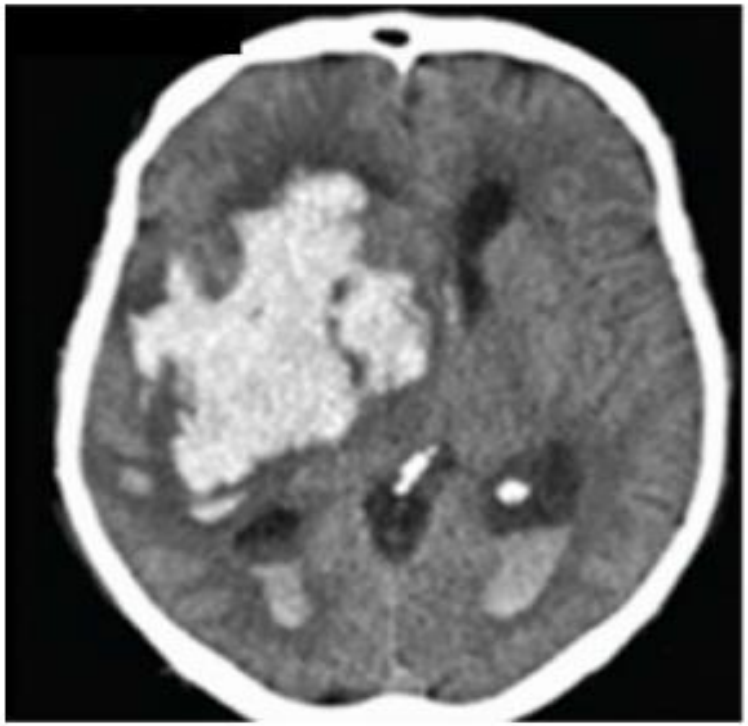


Left Middle Cerebral Artery  Infarction with minimal mass effect – level of consciousness probably normal.



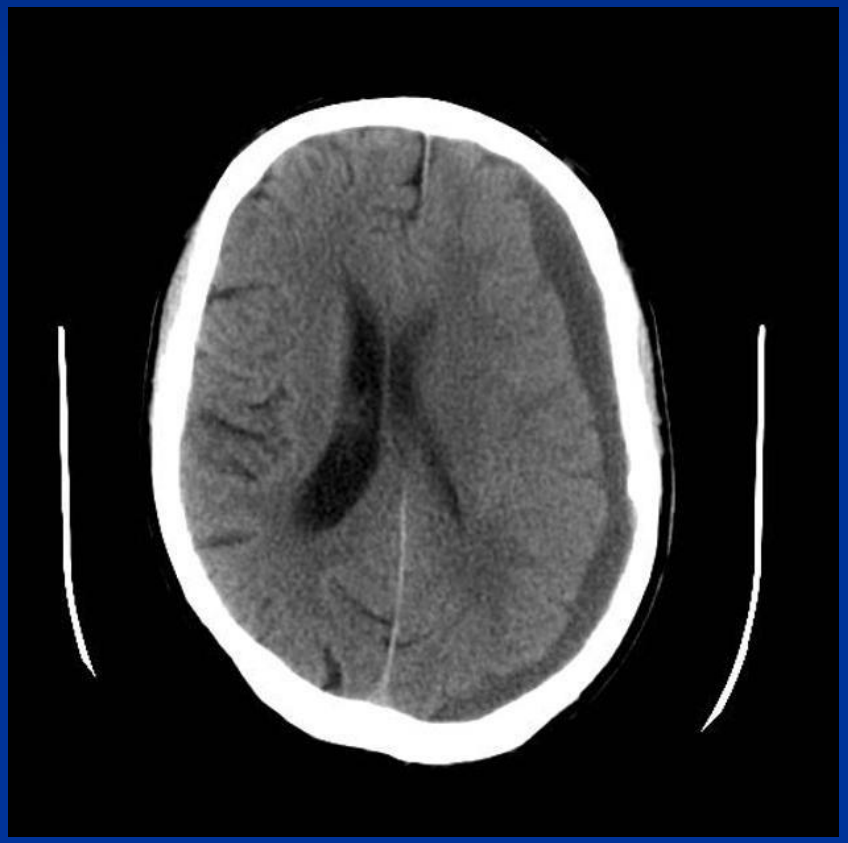


Ischemic Stroke
(dark/hypodense)

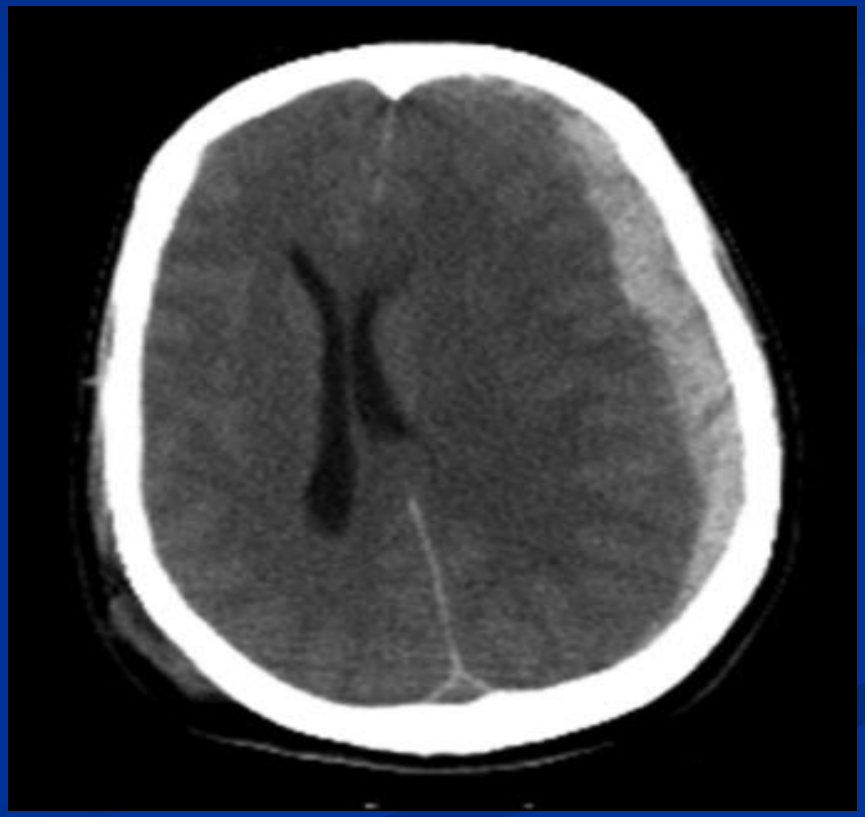


Hemorrhagic Stroke
(bright/hyperdense)

Chronic Subdural Hematoma



Acute Subdural Hematoma



# Management of Coma in the First Hour

- Improve oxygenation (face mask with 10 l/min oxygen flow aiming at a pulse oximeter saturation of >95%).
- Intubate if patient cannot protect the airway (ie, pooling secretions, gurgling sounds) or with increased work of breathing.
- Intubate any comatose patient with irregular ineffective respiratory drive and poor oxygenation.
- Intubate any comatose patient with major facial injury or consider emergency tracheostomy.

# Management of Coma in the First Hour

- No harm is done if a patient with a high likelihood of hypoglycaemia is immediately given 50 ml of 50% glucose, even before the blood sugar is known (with co-administration of 100 mg thiamine intravenously).
- No harm is done administering naloxone if opioid intoxication is suspected.
- Flumazenil reverses any benzodiazepine toxicity.

# Possible CNS Infection ?

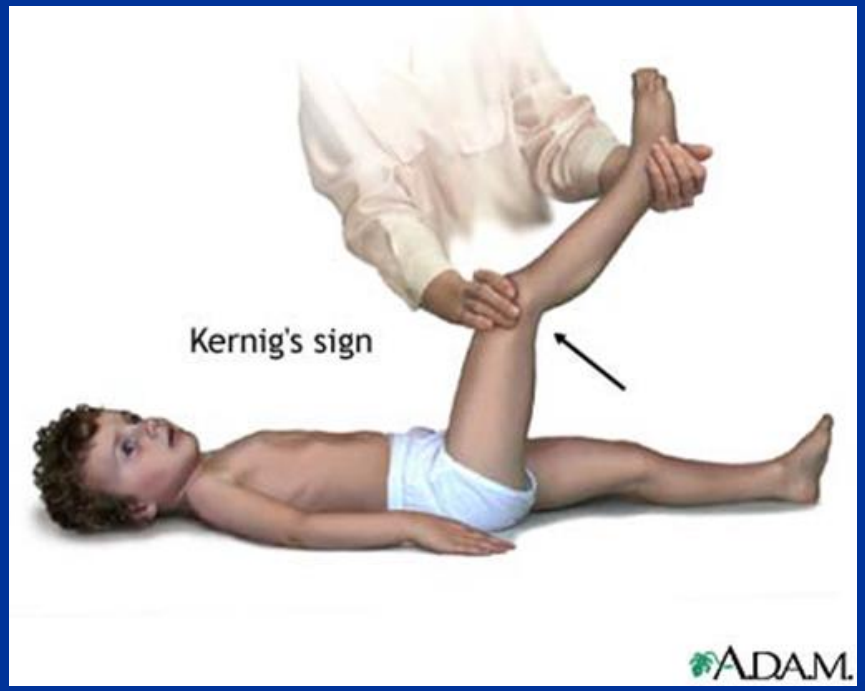
- High index of suspicion in immunosuppressed ( DM, Cancer, Steroids, Biologics, HIV)
- Acute Bacterial Meningitis
- Viral Encephalitis
- Brain abscess
- Subdural Empyema
- Cerebral Malaria –causes rapidly progressive coma
- TB Meningitis

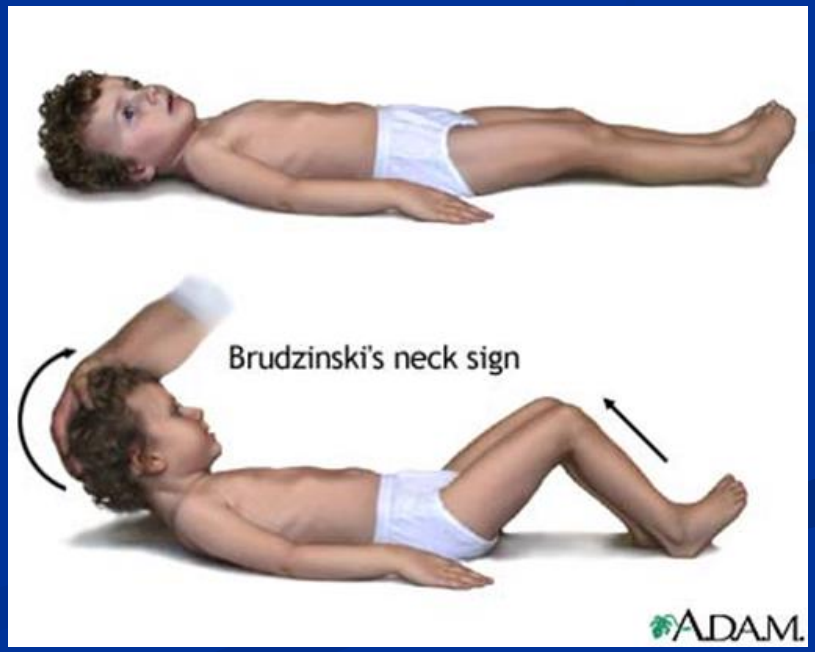
# Meningitis: Symptoms and Signs

- Early flu-like symptoms
- Worsening headache (+/- Nausea or vomiting)
- Chills/High fever
- Confusion/irritability/difficulty concentrating/ drowsiness/coma
- Seizures
- Nuchal rigidity and other meningeal irritation signs/Photophobia
- Purpuric skin rash ( in meningococcal meningitis)

- May develop rapidly over 1-2 days or slowly over many days , esp. in the elderly or immunosuppressed

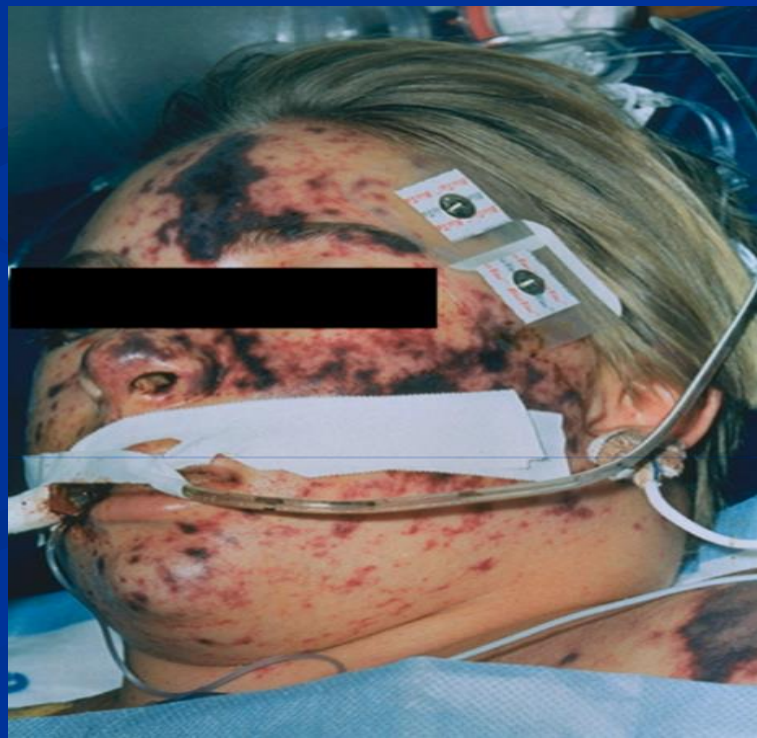
# Meningeal irritation signs



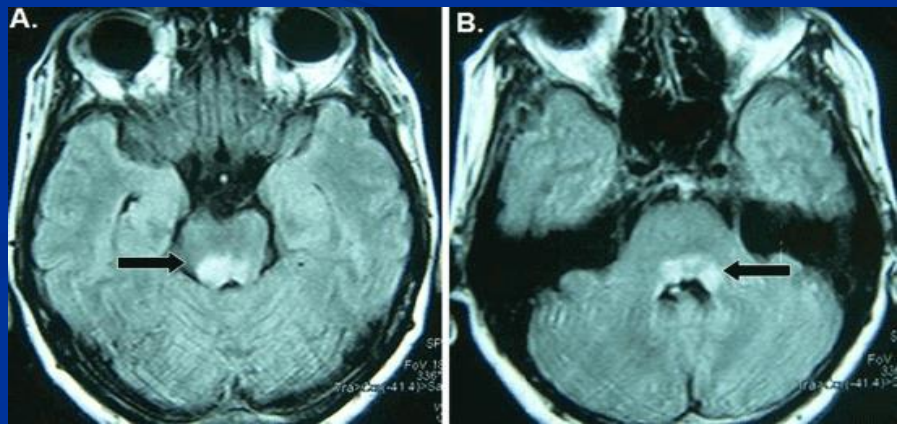
# Acute bacterial Meningitis (ABM)

- Common & serious
- Medical emergency
- 100% curable if treated adequately or 100% fatal
- High index of suspicion important
- Dx by CSF examination



# ABM : Etiology

- **Streptococcus pneumoniae (pneumococcus)- Most common cause**.
- **Neisseria meningitidis (meningococcus)**. This is a highly contagious infection that affects mainly teenagers and young adults**.** It may cause local epidemics in college dormitories, boarding schools and military bases.
- **Listeria monocytogenes -** These bacteria can be found in unpasteurized cheeses, hot dogs and lunchmeats. Pregnant women, newborns, older adults and immunocompromized people. Brainstem involvement is common



# ABM

- Diagnosis
  - High index of suspicion very important
  - Confirm by CSF examination
  - If LP is contraindicated ( Brain CT) , start empirical antibiotics on suspicion

- CSF: ↑Pressure, turbid, ↑cells (mostly polymorphs), ↑protein, ↓sugar to < 40% of blood sugar, ↑lactate > 2.4 mmol/l
- Gram stain, culture
- PCR

# Treatment for bacterial meningitis

- Ceftriaxone (2g every 12 hrs) or cefotaxime (8–12 g daily, divided doses every 6 h intravenously) +/- Vancomyocin (2 g daily, divided dose every 12 h intravenously)
- Add ampicillin (12 g daily, divided dose every 4 h intravenously +/- Gentamicin if Listeria suspected (age >55 yrs, immunosuppressed)

- Consider intravenous dexamethasone 10 mg x 4 with or just before first dose of antibiotics, and continue for 4 days- benefit in pneumococcal meningitis

# Viral meningitis

- Viral meningitis is usually mild and often clears on its own.
- Most cases are caused by a group of viruses known as enteroviruses, which are most common in late summer and early fall.
- Viruses such as herpes simplex virus, HIV, mumps, West Nile virus and others also can cause viral meningitis.

# CSF

**Table 1 | Typical cerebrospinal fluid (CSF) findings in infectious meningitis[1 3 14]**

| Cause of meningitis | White blood cell count (cells/mm3/10⁶ cells/l) | Predominant cell type | CSF: serum glucose (normal ≥0.5) | Protein (g/l) (normal 0.2-0.4) |
|---|---|---|---|---|
| Viral | 50-1000 | Mononuclear (may be neutrophilic early in course) | >0.5 | 0.4-0.8 |
| Bacterial | 100-5000 | Neutrophilic (mononuclear after antibiotics) | <0.5 | 0.5-2.0 |
| Tuberculous | 50-300 | Mononuclear | <0.3 | 0.5-3.0 |
| Cryptococcal | 20-500 | Mononuclear | <0.5 | 0.5-3.0 |

# Encephalitis

Encephalopathy = (altered consciousness persisting for longer than 24 h, including lethargy, irritability or a change in personality or behaviour)

Encephalitis = encephalopathy AND evidence of CNS inflammation, demonstrated by at least two of:

- fever
- seizures or focal neurological findings attributable to the brain parenchyma
- CSF pleocytosis (more than 4 white cells per μL)
- EEG findings suggestive of encephalitis
- neuroimaging findings suggestive of encephalitis.

# Encephalitis

- Encephalitis may be infectious or autoimmune
- Most common cause of sporadic infectious encephalitis is HSV1. Also HSV2 and VZV.
- Many other viruses and bacteria

- In practice, little distinction may initially be seen between meningitis and encephalitis and the term **meningoencephalitis** is often used – both covered initially.

# Herpes simplex virus encephalitis

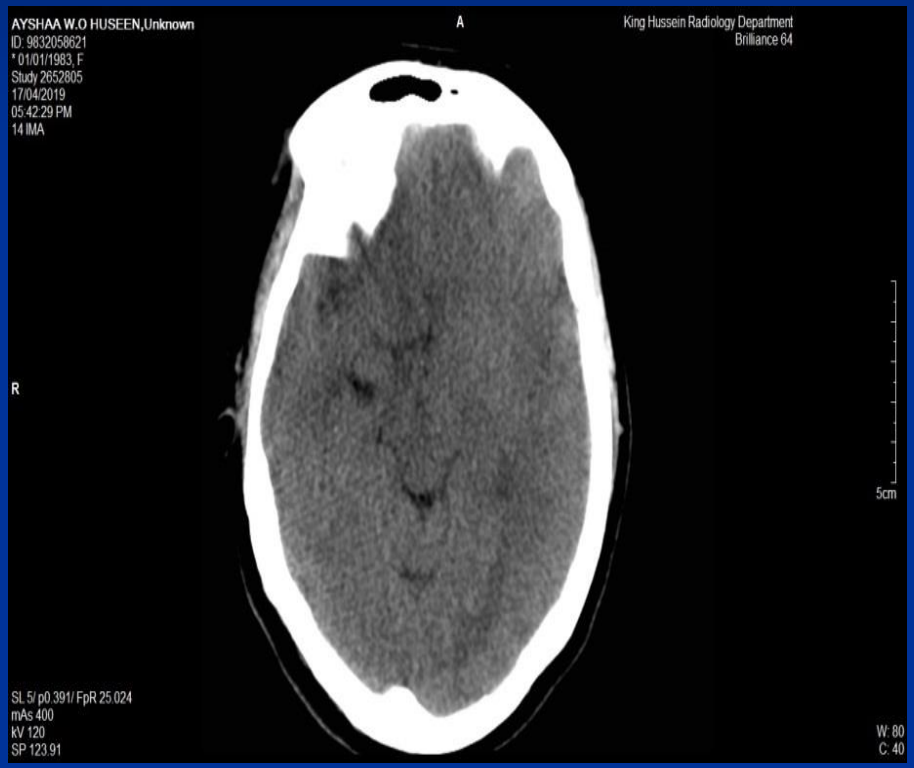
- Most cases are caused by HSV1, but around 10% are caused by type 2.
- The most distinctive presenting features are fever, disorientation, aphasia and behavioural disturbances, and up to a third of patients have convulsive seizures.

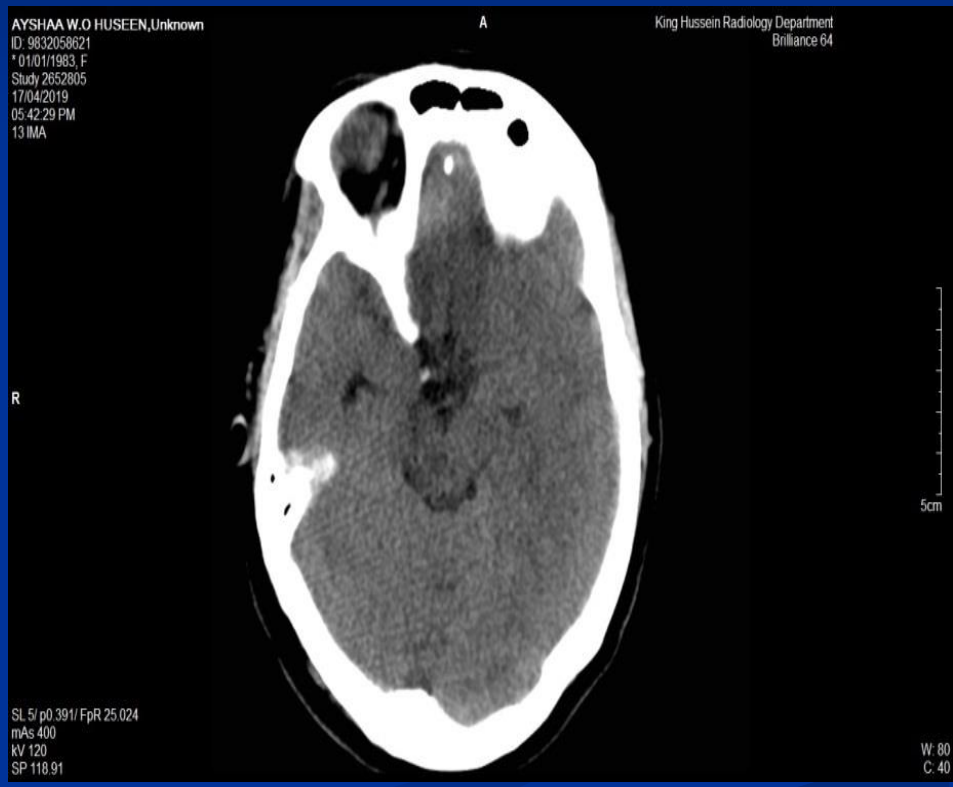
# HSV Encephalitis

- Neuroimaging can be negative acutely, but by 48 hours, over 90% of patients have MR brain imaging abnormalities and sensitivity approaches 100% at 3–10 days.
- MRI shows markedly asymmetric but usually bilateral abnormalities in the limbic system, medial temporal lobes, insular cortices and inferolateral frontal lobes
- CSF clear, pleocytosis +/-. Normal or ↑protein, normal sugar , PCR (may be negative early- repeat after 24-72 hrs)

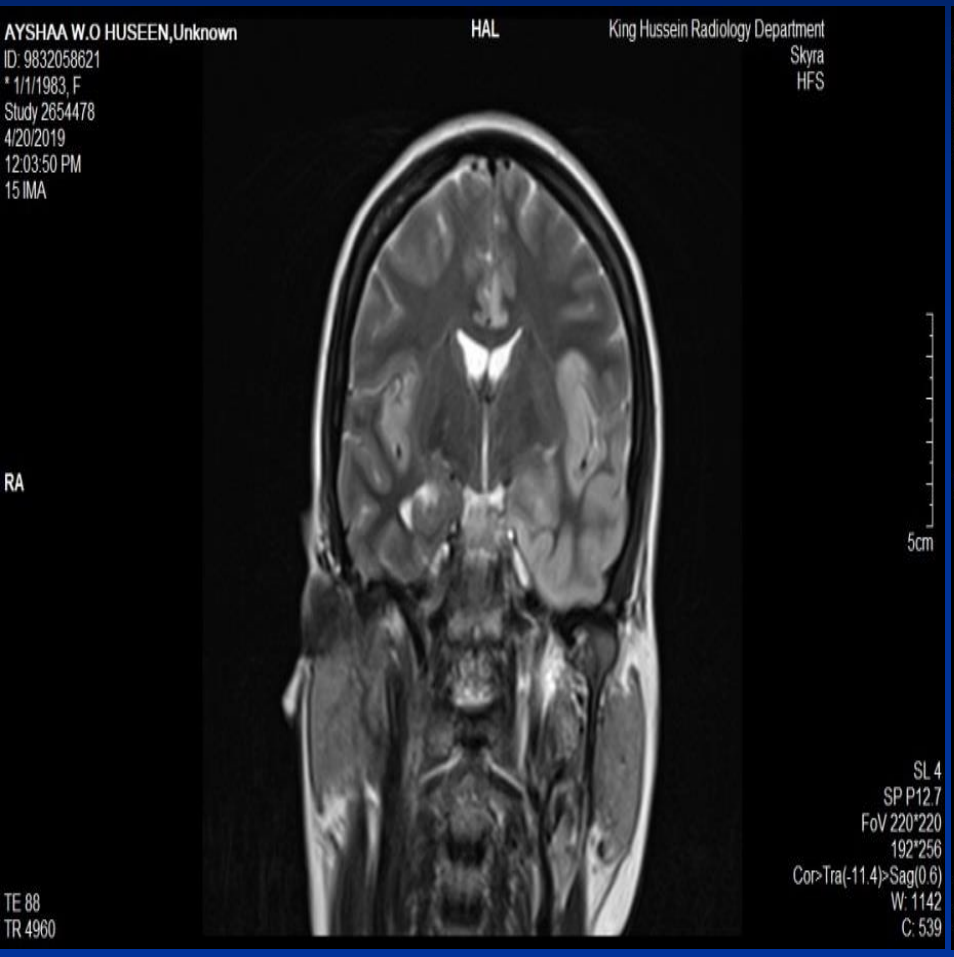
- Prompt Rx with IV aciclovir 10 mg/kg x3 if any suspicion

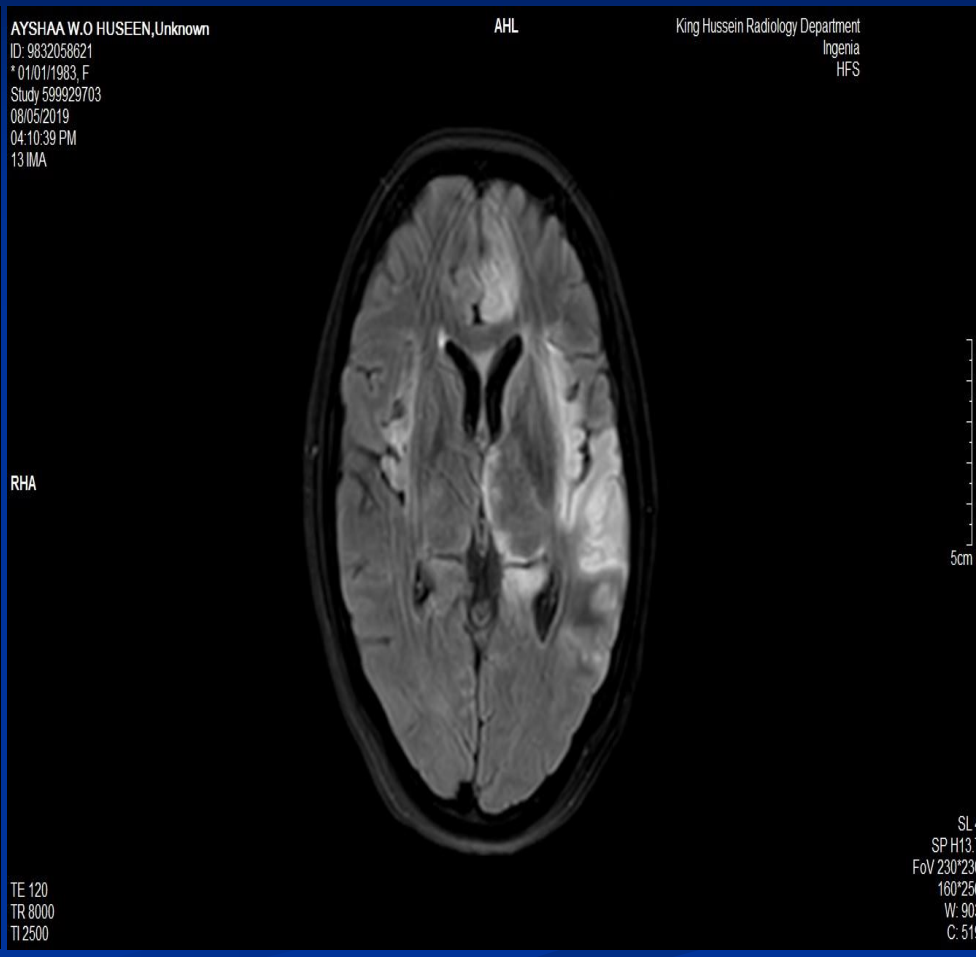
# Brain CT in a case of HSV encephalitis

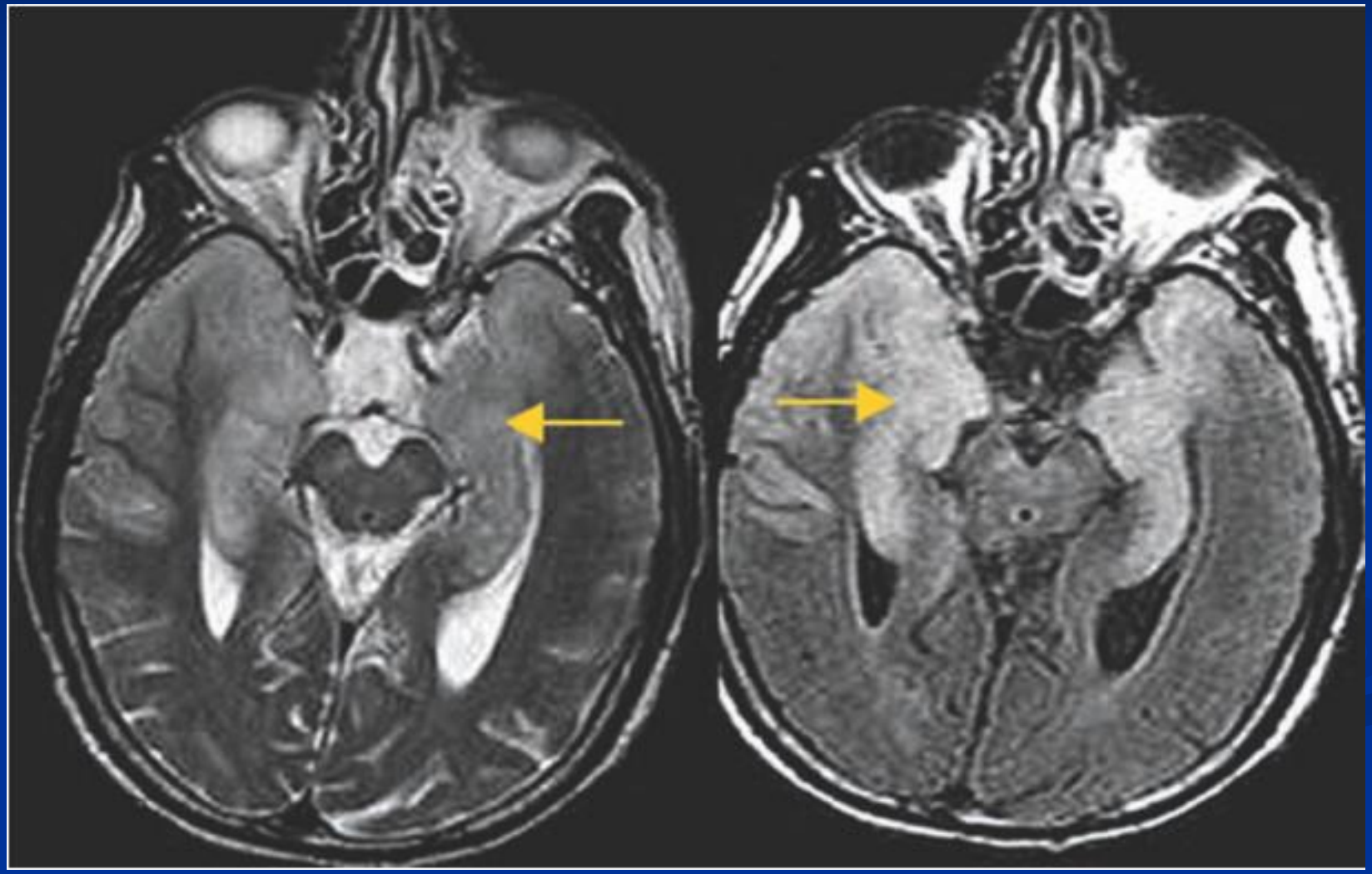


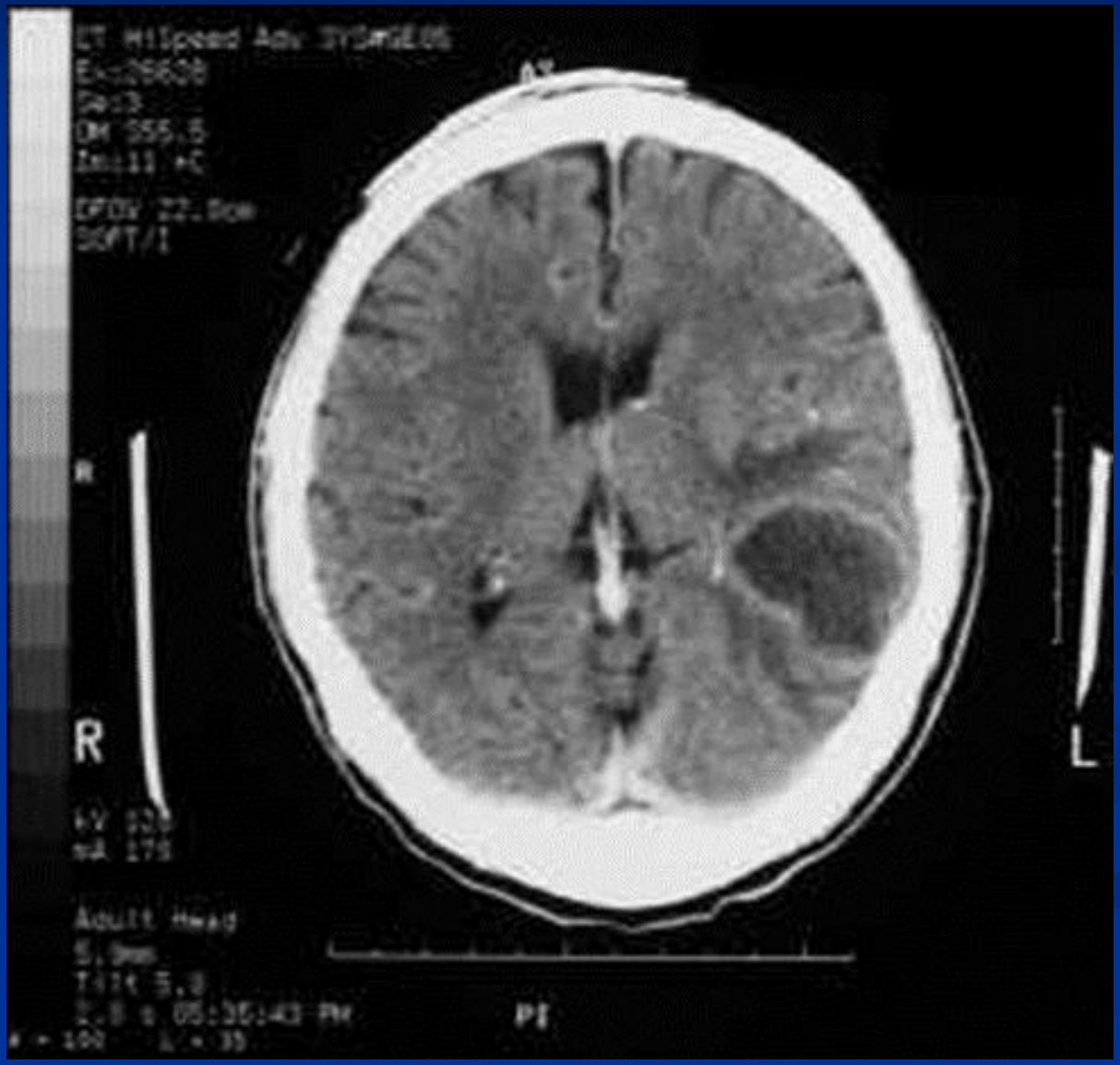
# MRI in previous patient.



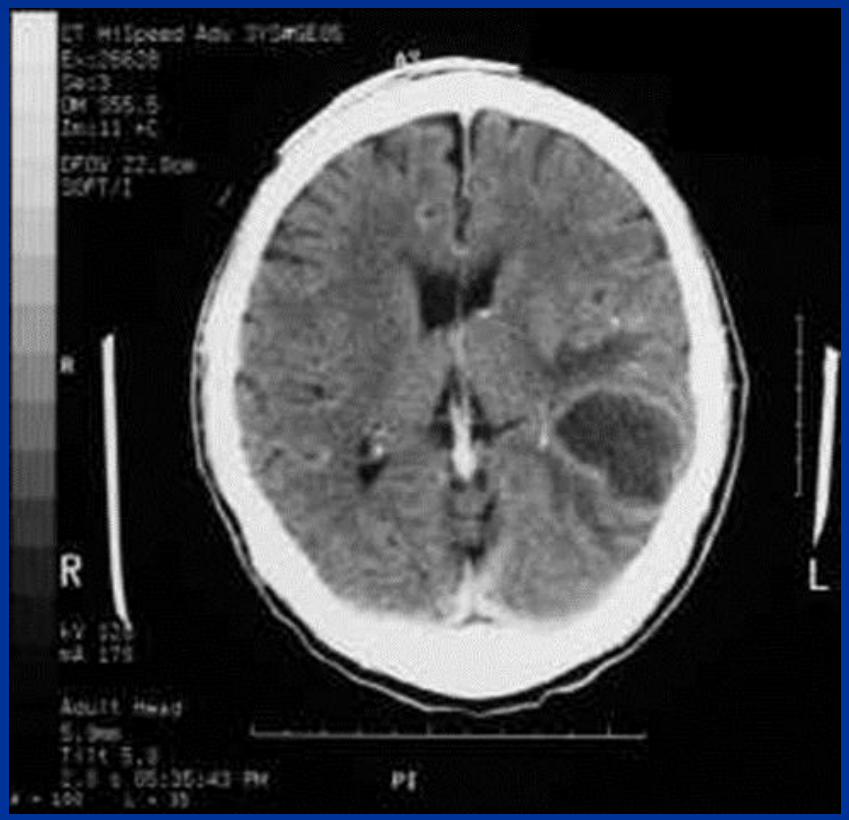





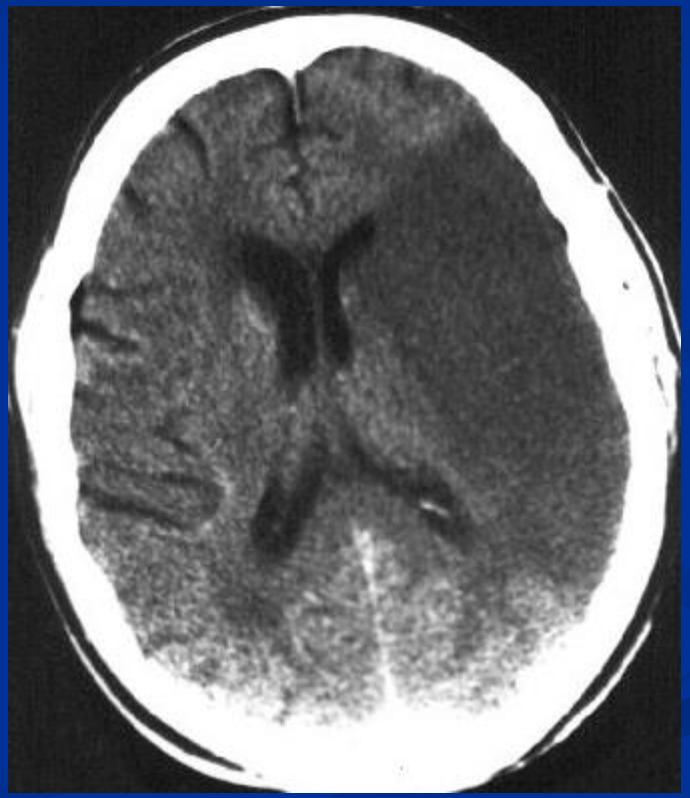
# What is shown on this CT ?



## Brain Abscess




## Ischemic Infarct



# Huge Brain abscess with mass effect and hydrocephalus

# Short Break

# 1916- Guillain-Barre Syndrome (GBS)

SUR UN SYNDROME DE RADICULO-NÉVRITE AVEC HYPERALBUMINOSE DU LIQUIDE CÉPHALO-RACHIDIEN SANS RÉACTION CELLULAIRE. REMARQUES SUR LES CARACTÈRES CLINIQUES ET GRAPHIQUES DES RÉFLEXES TENDINEUX.

par MM. GEORGES GUILLAIN, J.-A. BARRÉ et A. STROHL.

# Landry's Ascending Paralysis 1859
## ( Jean-Baptiste Landry )
## **Landry-Guillain-Barre-Strohl Syndrome**

# Guillain-Barre Syndrome (GBS)

- GBS is an acute immune-mediated radiculo-neuropathy. It is the most frequent cause of acute flaccid paralysis worldwide and constitutes a neurologic emergency
- It shows a pattern of symmetrical "ascending paralysis" in about two thirds of patients, beginning in the lower limbs and then spreading to the upper limbs/trunk/neck/ head. Proximal and distal muscles.
- Hypo/Areflexia
- Sensory (mild) and autonomic symptoms.
- Maximal weakness at 1-2 weeks.
- It can affect the facial muscles (50% of cases) , bulbar and respiratory muscles - 25% of patients need artificial ventilation.
- Extraocular muscle weakness /ptosis - uncommon

# Differential diagnosis of acute flaccid paralysis

- **Brainstem stroke/ encephalitis**

- **Acute myelopathy**
  - **Spinal cord infarction/haemorrhage**
  - **Acute transverse myelitis**

- **Acute poliomyelitis**

- **Peripheral neuropathy**
  - **Guillain-Barré syndrome**
  - Diphtheritic neuropathy
  - Heavy metals (thallium)
  - Acute intermittent porphyria
  - Vasculitic neuropathy
  - Lymphomatous neuropathy

# Differential diagnosis of acute flaccid paralysis

- **Disorders of neuromuscular transmission**
  - **Myasthenia gravis**
  - Botulism
- **Disorders of muscle**
  - **Hypokalaemia** / Periodic paralyses
  - Hypophosphatemia
  - Inflammatory myopathy
  - Acute rhabdomyolysis

# Investigations

- CSF – Albumino-cytological dissociation
- NCS – Demyelinating neuropathy (AIDP) >> Axonal (AMAN, AMSAN)
- Spine MRI and blood work-up ( K/P/other electrolytes, CPK ...) to exclude other diagnoses

# GBS vs. Acute Transverse Myelitis

| *Clinical feature* | GBS | TM |
|---|---|---|
| Onset of paralysis | Hours to 4 weeks | Within 4 days |
| Motor findings | Ascending weakness | Para paresis or quadriparesis |
| Sensory findings | Ascending sensory | Spinal cord levels |
| Autonomous findings | CVS | Bladder and bowel |
| Cranial nerves | EOM palsies or facial weakness | None |
| CSF | Pleocytosis uncommon, raised protein | Pleocytosis Common, protein normal |
| MRI | Normal | Focal abnormalities |

# GBS triggers

- **Infections**
  Two thirds of cases are associated with an infection a few weeks before the onset of neurological symptoms. The spectrum varies depending on geographical location, and includes:
  - Campylobacter jejuni
  - Cytomegalovirus
  - Epstein-Barr virus
  - Mycoplasma pneumoniae / ? Haemophilus influenzae
  - Influenza/ ? COVID-19
  - Hepatitis E
  - HIV seroconversion ( High CSF cell coumt)
- **Postpartum**
- **Surgery**
- **Vaccinations ??**

# GBS treatment- Immunotherapy

- IVIg ( 0.4 g/kg daily for 5 days ) or
- Plasma exchange ( 4-5 sessions)

# Supportive Management for possible complications

- Careful monitoring of vital capacity (VC) with intubation for those with a VC of <15 ml/kg or which is rapidly dropping
- Twenty five per cent of GBS patients require ventilatory support during their illness, which may be predicted if there is rapid progression of limb weakness, facial or bulbar weakness or dysautonomia .
- Cardiac monitoring for possible arrhythmias throughout the acute stages .
- Venous thromboembolism prophylaxis with compression stockings and low molecular weight heparin is recommended for non-ambulant patients

# GBS


56 yr old male

Previously fit and well

4 week history of

- Diplopia- horizontal
- Variable through the day
- Struggling with drinks- leaking from mouth
- Slurred and low volume speech

Admitted to hospital

# Diagnosis ?

## Whilst in hospital

- Weakness of neck- head drop
- Weakness of arms: SAD/ WE, FE)
- Difficulty breathing, but CXR normal- taken to the ICU
  - Low FVC, VC and borderline PEFR
  - Needed ICU as tiring, confused
- Noted to using accessory muscles and accessory muscle use

## Exam

- Ptosis
- Complex ophthalmoplegia
- Facial weakness
- Fatiguable dysarthria
- Neck flex/ext weakness
- Fatiguable UL weakness
- Intact reflexes
- Normal sensory exam

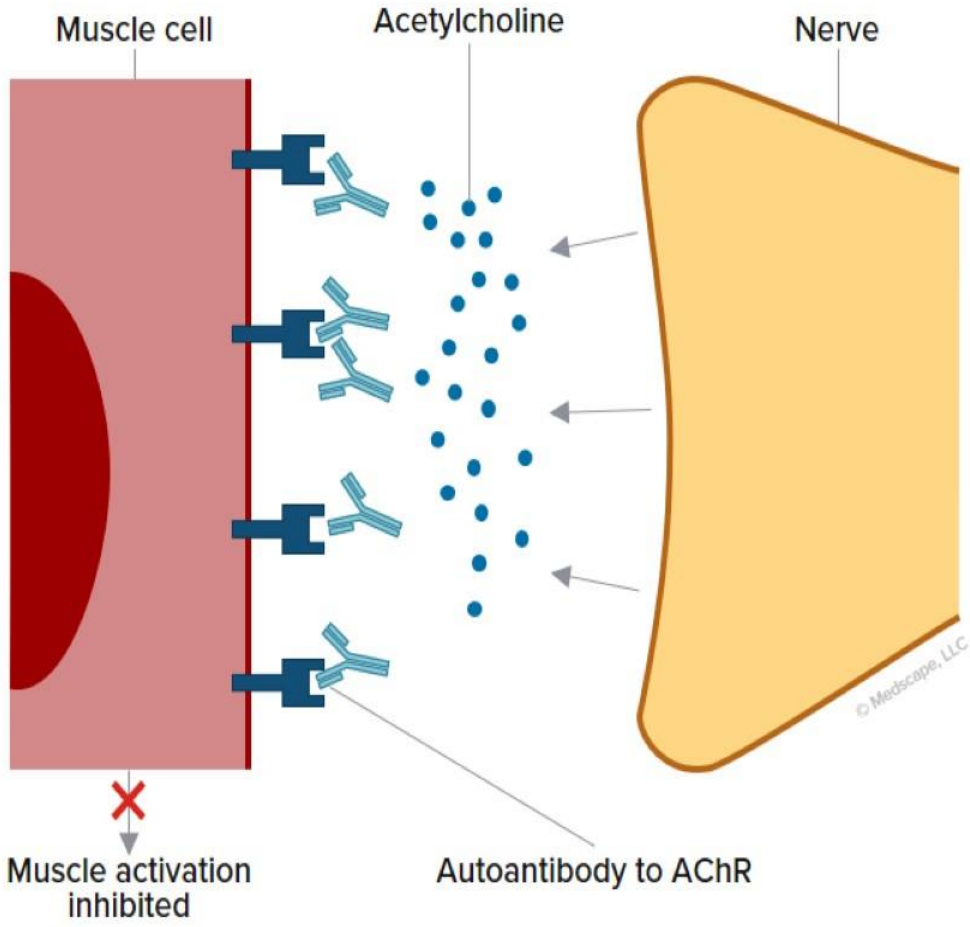
MRI Brain normal

# Myasthenia gravis

## gMG Pathophysiology

**Autoimmune NMJ Disorder**

Characteristic muscle weakness is caused by pathogenic autoantibodies that bind to components of the NMJ[a]




a. Verschuuren JJ, et al. *Autoimmun Rev.* 2013;12:918-923.

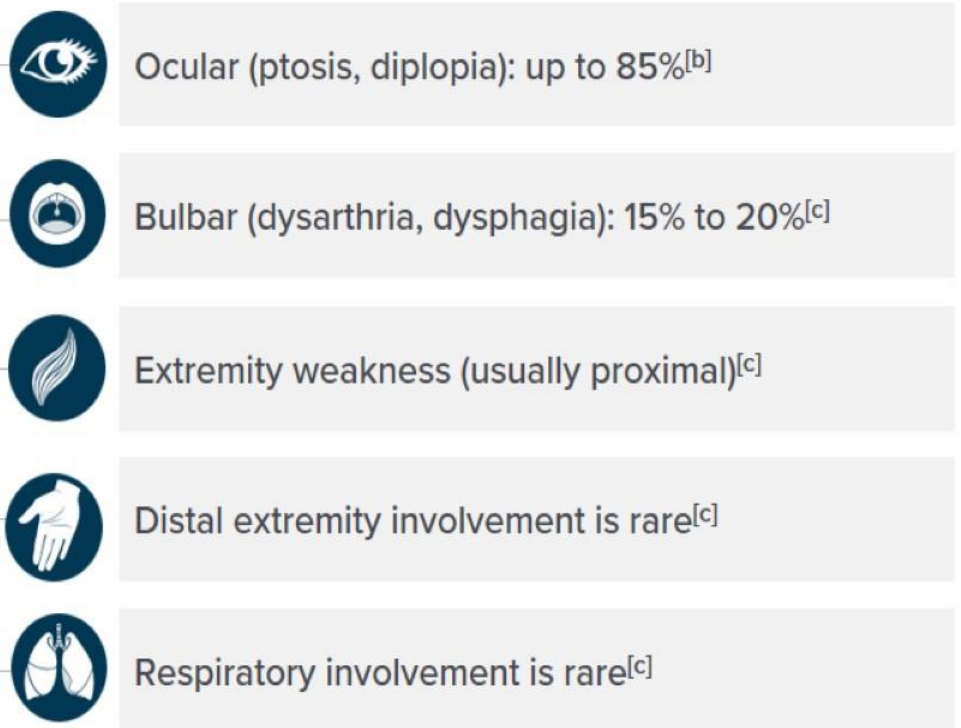
# Myasthenia Gravis

- **Epidemiology**
  - Prevalence: 2-14/100,000
  - Two peaks: 2nd/3rd & 6th/7th decade
  - Rarely familial
- **Presentation**
  - Ocular: diplopia, ptosis (50-60%)
  - Bulbar or limb weakness (30-35%)
  - Respiratory failure: rare as a presenting sx

# Myasthenia gravis

## Clinical Presentation (cont)

**Clinical hallmark: fluctuating, pronounced, fatigable weakness limited to the voluntary muscles[a]**






a. Juel VC, et al. *Orphanet J Rare Dis*. 2007;2:44; b. Grob D, et al. *Muscle Nerve*. 2008;37:141-149; c. Gwathmey KG, et al. *Semin Neurol*. 2015;35:327-39.

# MG: Weakness-1

- Variable: worsens with physical activity and improves with rest; worse at the end of the day
- Ocular
  - Ptosis and/or diplopia
  - Usually asymmetric
  - Normal Pupils
- Facial: very common
- Bulbar
  - Dysarthria; weak mastication
  - Abnormal gag reflex; weak palate, tongue
- Respiratory
  - Weak diaphragm and intercostals: SOB, weak cough, decreased counting/one breath
  - May result from vocal cord paralysis

# MG: Weakness-2

- Neck: Extensors weakness (head droop)
- Limb weakness
  - Proximal > distal
  - Arms > legs
  - Usually symmetric
  - Predilection for certain muscles: triceps, quadriceps
  - Limb-girdle myasthenia (limited to limbs, 2%)

# MG: investigations


- The orbital Ice Test
- IV Edrophonium Test ??

- Serum Autoantibodies
  - Anti-Acetylcholine Receptor antibodies
  - Anti-Muscle-Specific Kinase (MuSK) antibodies
  - Anti-striational antibodies
  - New antibodies
- Electrophysiological tests
  - Repetitive Nerve Stimulation
  - Single Fiber EMG
- CT scan of the chest for thymoma or thymmic hyperplasia
- Search for other autoimmune diseases: thyroid disease, B12 deficiency, collagen vascular disease

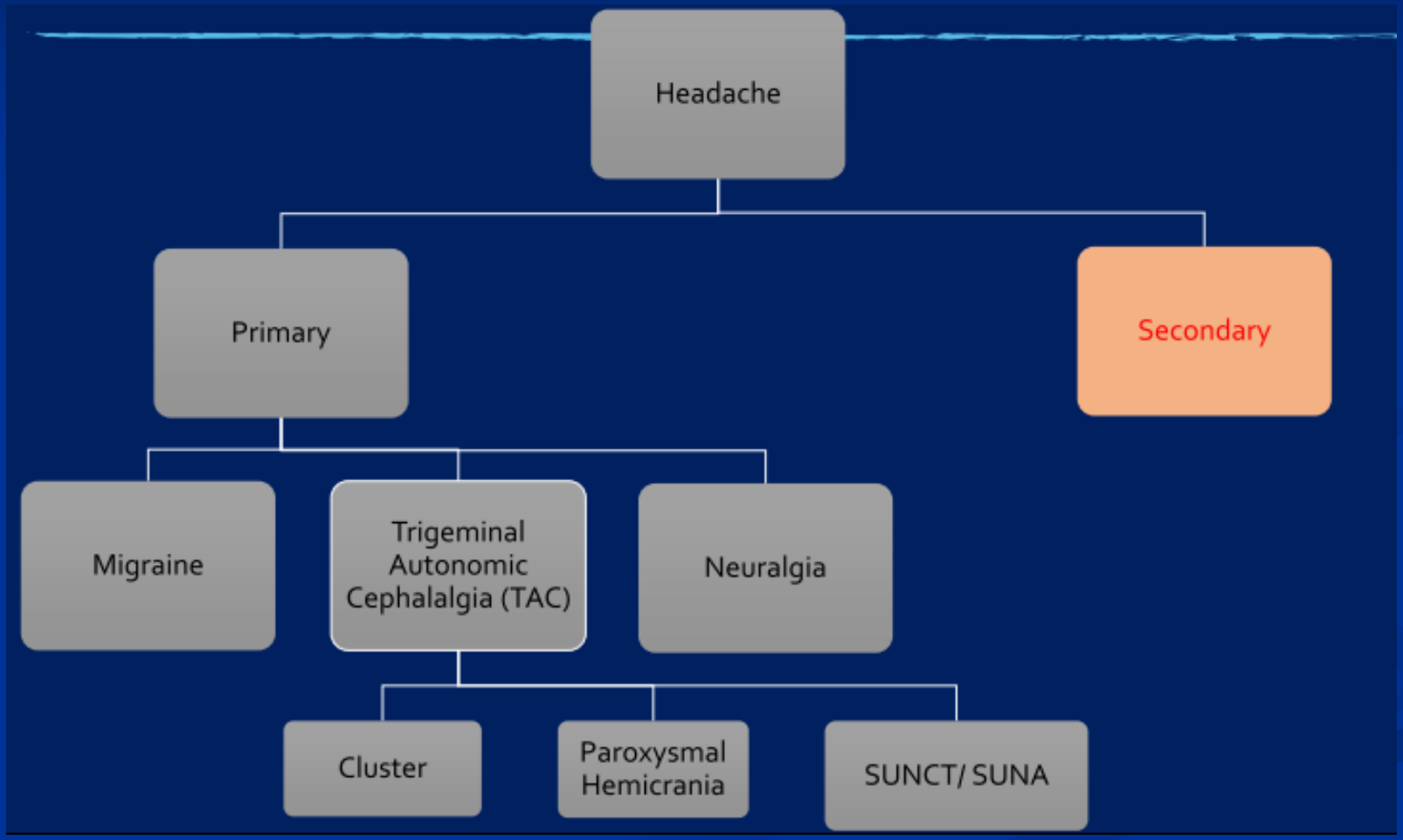
# Anti-Acetylcholine Receptor (AChR) antibodies

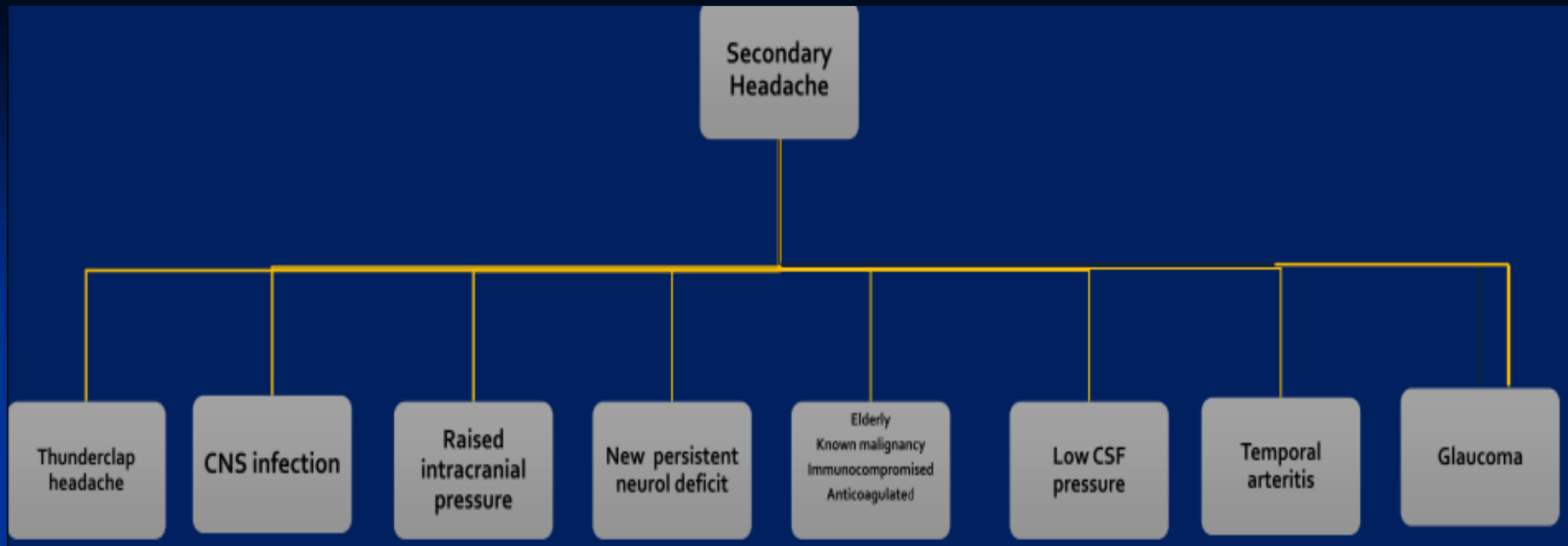
- Detectable in 85% - 90% of generalized MG and 50% of ocular MG
- Rare in normal population: < 1%
- Level correlates with disease status in the individual patient

# MG Treatment

- Myasthenic crisis Rx (IVIg or PE)
- Long-term Rx
  - Oral steroids/immunosuppressives
  - Acetylcholinesterase inhibitors
  - Thymectomy
  - Rituximab and new monoclonals

# Headache


Headache
Primary
Secondary
Migraine
Trigeminal Autonomic Cephalalgia (TAC)
Neuralgia
Cluster
Paroxysmal Hemicrania
SUNCT/ SUNA




**Urgent Assessment in hospital**

**CT BRAIN** with contrast
----Discuss----
Lumbar Puncture
--- Discuss----
Bloods – ESR, CRP
Ophthalmology assessment
IF INVESTIGATIONS ARE NORMAL- REASSESS THE PATIENT
IF RED FLAGS STILL PERSIST- FURTHER OPINION/ INVESTIGATIONS
If no further red flags- diagnose a primary headache disorder

# SNNOOP10

| | Sign or symptom | Related secondary headaches (most relevant ICHD-3b categories) |
|---|---|---|
| 1 | Systemic symptoms including fever | Headache attributed to infection or nonvascular intracranial disorders, carcinoid or pheochromocytoma |
| 2 | Neoplasm in history | Neoplasms of the brain; metastasis |
| 3 | Neurologic deficit or dysfunction (including decreased consciousness) | Headaches attributed to vascular, nonvascular intracranial disorders; brain abscess and other infections |
| 4 | Onset of headache is sudden or abrupt | Subarachnoid hemorrhage and other headaches attributed to cranial or cervical vascular disorders |
| 5 | Older age (after 50 years) | Giant cell arteritis and other headache attributed to cranial or cervical vascular disorders; neoplasms and other nonvascular intracranial disorders |
| 6 | Pattern change or recent onset of headache | Neoplasms, headaches attributed to vascular, nonvascular intracranial disorders |
| 7 | Positional headache | Intracranial hypertension or hypotension |
| 8 | Precipitated by sneezing, coughing, or exercise | Posterior fossa malformations; Chiari malformation |

# SNNOOP10

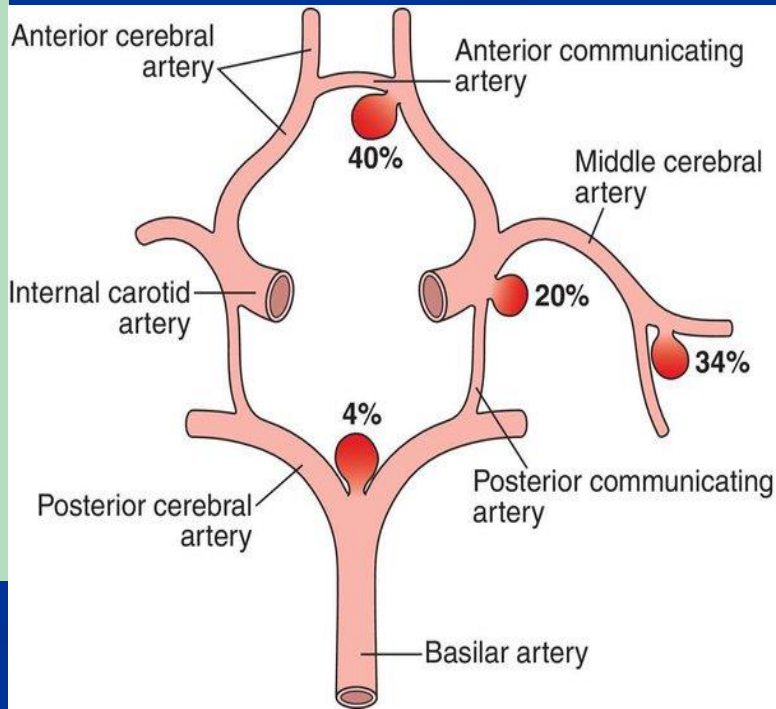
| | | |
|---|---|---|
| 9 | Papilledema | Neoplasms and other nonvascular intracranial disorders; intracranial hypertension |
| 10 | Progressive headache and atypical presentations | Neoplasms and other nonvascular intracranial disorders |
| 11 | Pregnancy or puerperium | Headaches attributed to cranial or cervical vascular disorders; postdural puncture headache; hypertension-related disorders (e.g., preeclampsia); cerebral sinus thrombosis; hypothyroidism; anemia; diabetes |
| 12 | Painful eye with autonomic features | Pathology in posterior fossa, pituitary region, or cavernous sinus; Tolosa-Hunt syndrome; ophthalmic causes |
| 13 | Posttraumatic onset of headache | Acute and chronic posttraumatic headache; subdural hematoma and other headache attributed to vascular disorders |
| 14 | Pathology of the immune system such as HIV | Opportunistic infections |
| 15 | Painkiller overuse or new drug at onset of headache | Medication overuse headache; drug incompatibility |

# Thunderclap Headache

- Very severe, maximal severity at onset
- Feels like being “hit on the head with a bat”
- Commonly occipital
- 25% associated with Subarachnoid hemorrhage

# Subarachnoid hemorrhage
# Non-traumatic causes

| | |
|---|---|
| Intracranial aneurysms: degenerative | 60–70% |
| Peri-mesencephalic haemorrhages | 15–20% |
| Arteriovenous malformations and associated aneurysms | 5–10% |
| Other causes: | ~ 5% |
| Dural fistula | |
| Venous vascular abnormalities | |
| Spinal arteriovenous malformations | |
| Cerebral artery dissections | |
| Moyamoya syndrome | |
| Vasculopathies | |
| Mycotic aneurysms | |
| Coagulopathies | |
| Neoplasia | |
| Pituitary apoplexy | |
| Drug abuse: amphetamine and cocaine | |

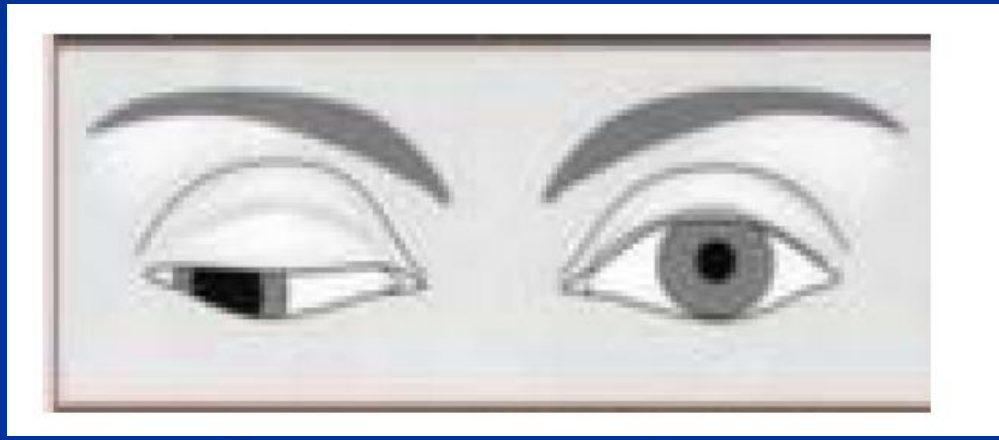


# Classic Symptoms of Subarachnoid Hemorrhage

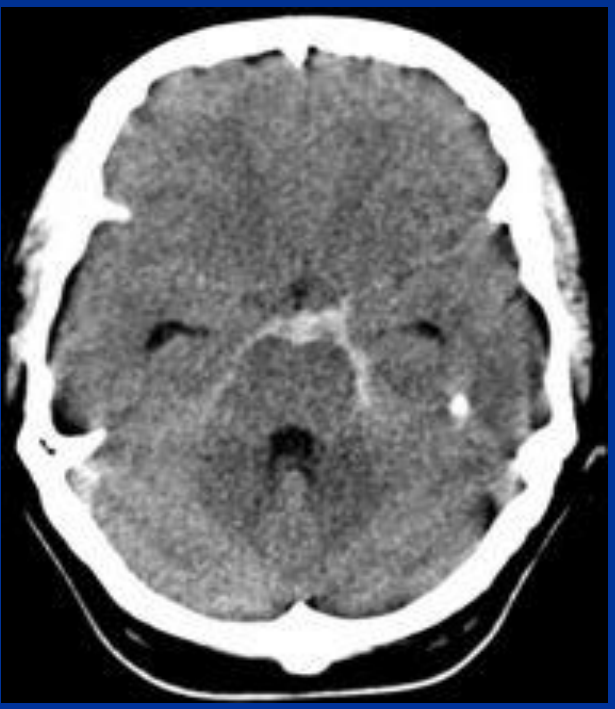
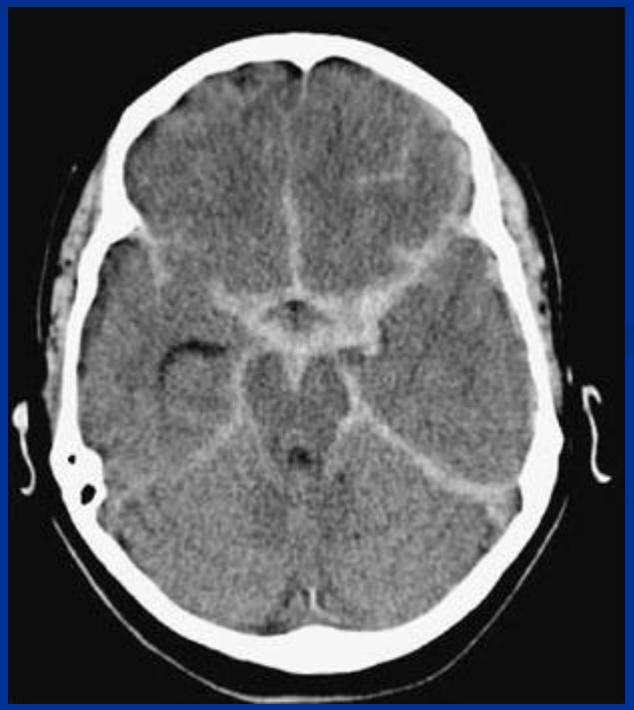
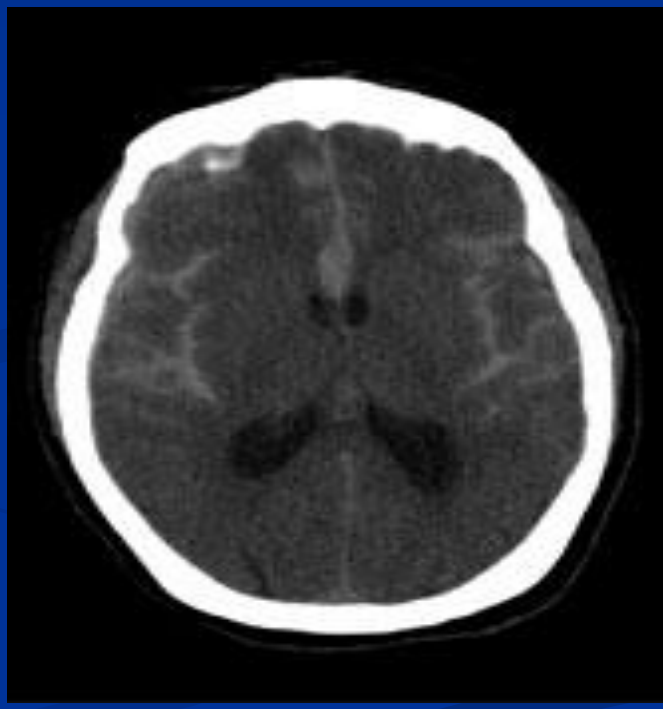
- Sudden, unusually severe or "thunderclap" headache
- Loss of consciousness (transient or persistent if severe )
- Pain in neck, back, eye
- Nausea, vomiting, photophobia

# Classic Signs of Subarachnoid Hemorrhage

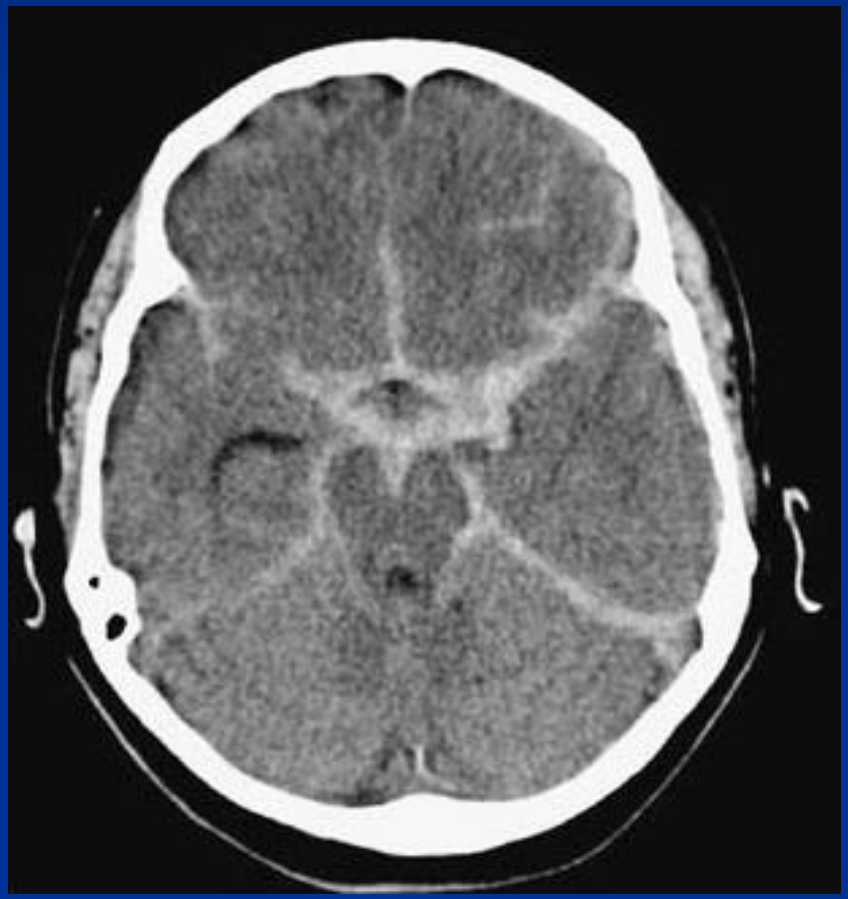
- Abnormal vital signs
  - Respiratory changes, hypertension, cardiac arrhythmias
- Meningism
- Focal neurologic signs may be present
  - III nerve palsy – IC/PCA aneurysm
  - Paraparesis – ACA aneurysm
  - Hemiparesis, aphasia – MCA aneurysm
- Ocular hemorrhages



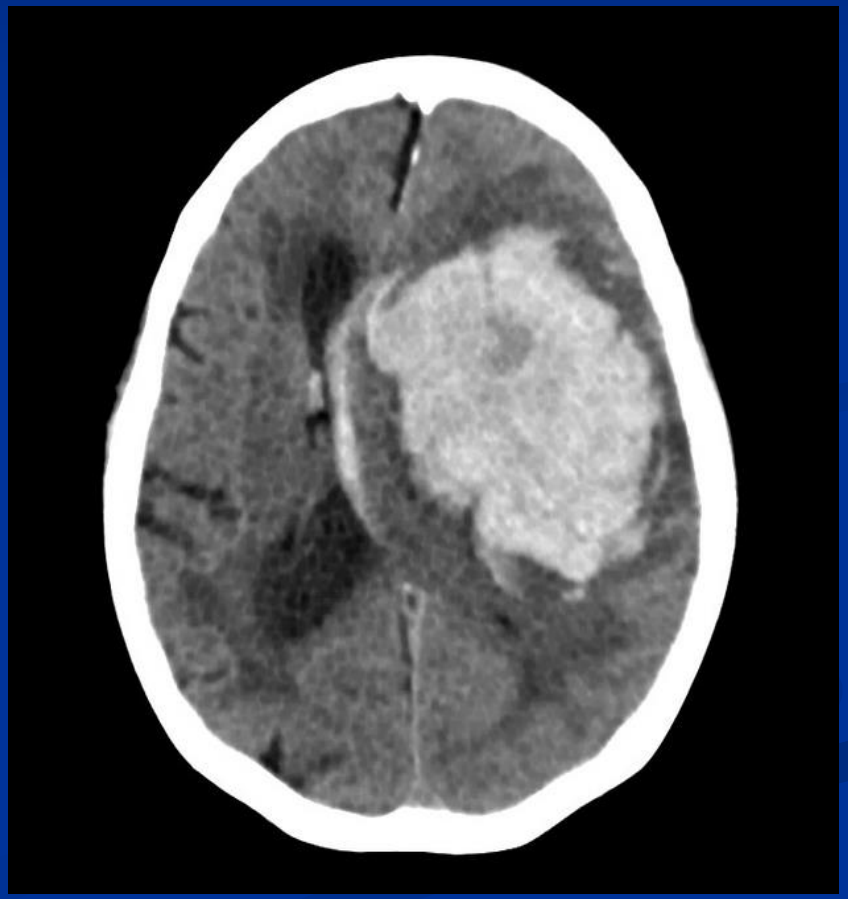
# Subarachnoid hemorrhage

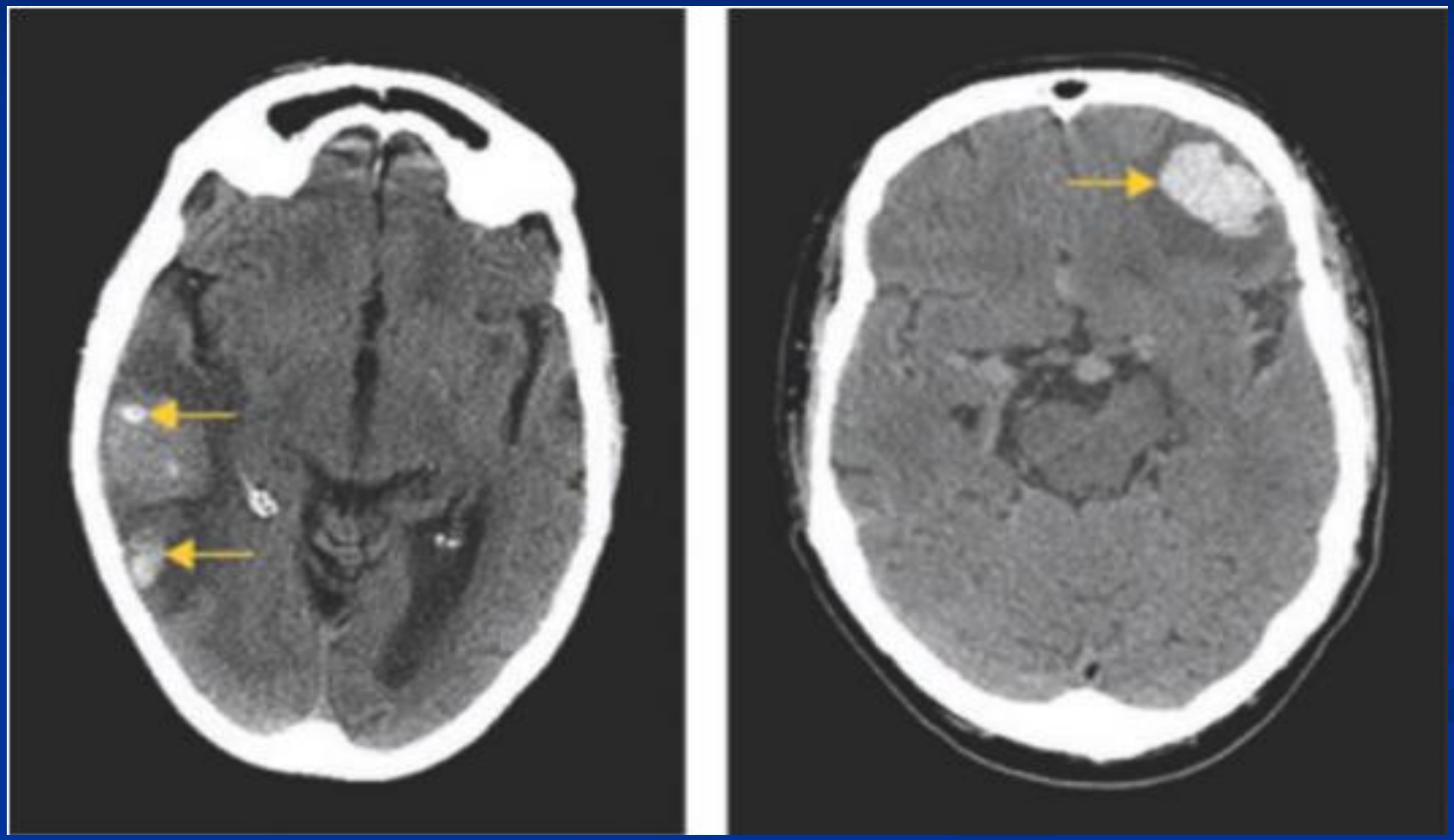
Subarachnoid hemorrhage



Intracerebral parenchymal hemorrhage



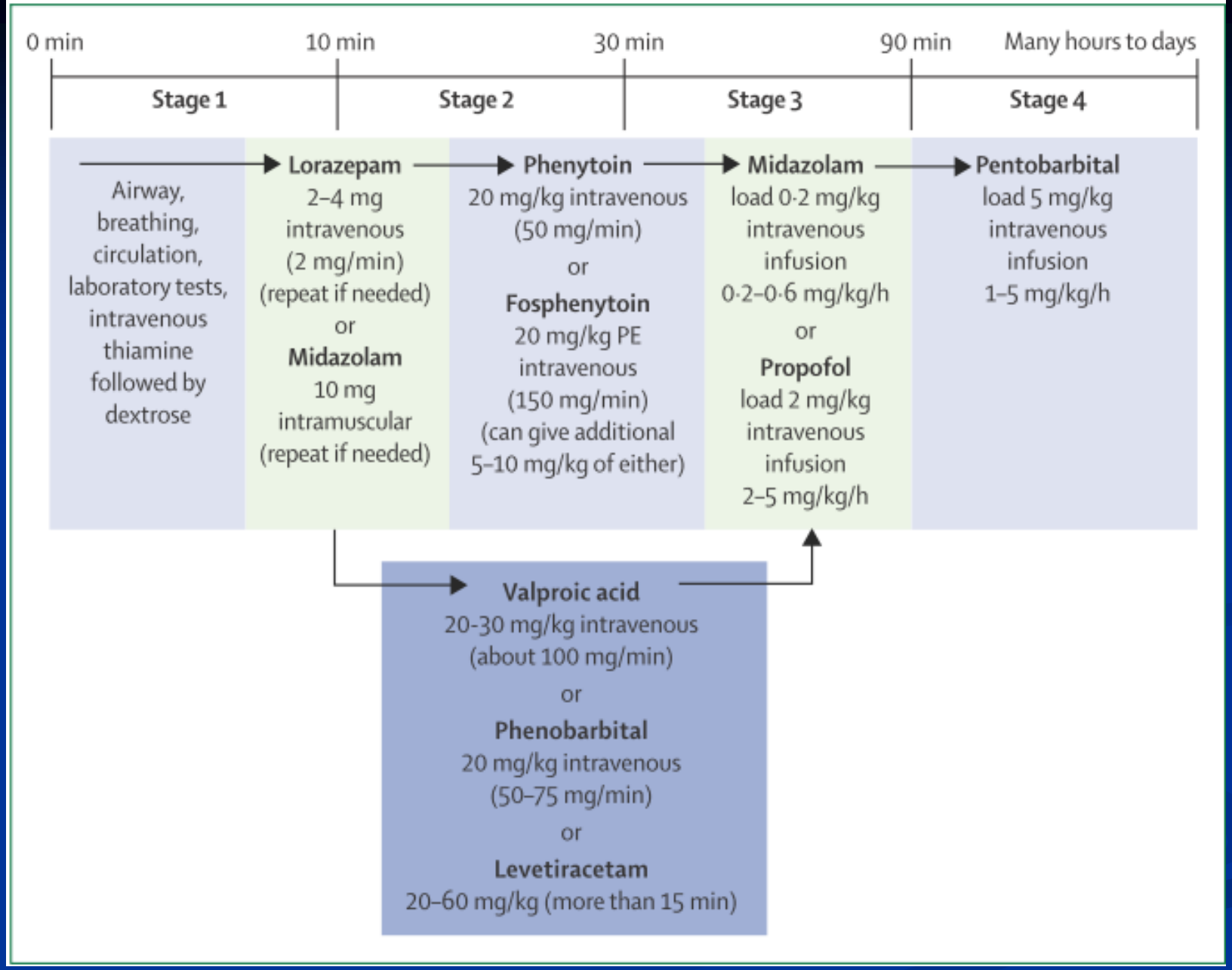
# Cerebral Venous Thrombosis (CVT) with hemorrhagic infarctions – Rx ?



# Seizures, Pseudo-seizures and Status Epilepticus

# Definition of Status Epilepticus

- If the patient has a prolonged (>5 min.) seizure or repetitive (3 or more/hr) seizures without recovery between episodes, they are considered to be in SE and the Rx protocol initiated.




# Status epilepticus in adults

*Lancet Neurol* 2015

*John P Betjemann, Daniel H Lowenstein*

## Box 1: Mimics of generalised convulsive status epilepticus

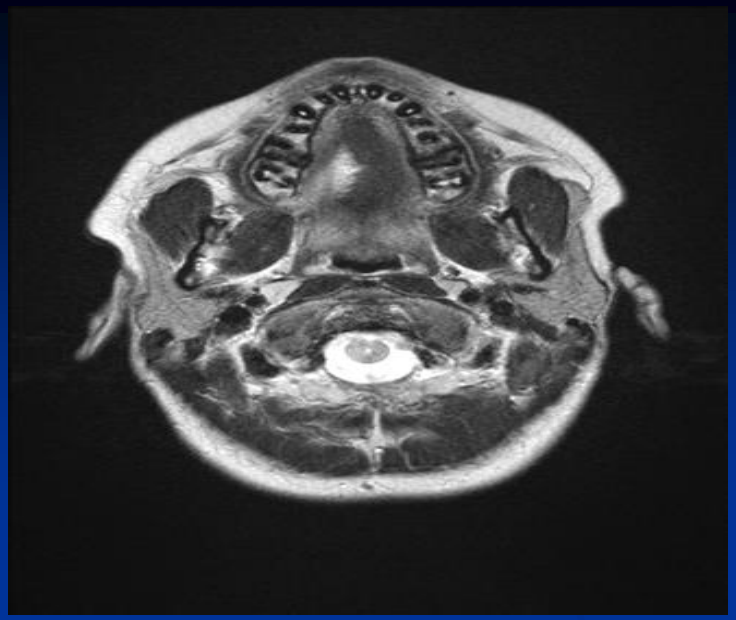
- Psychogenic status epilepticus
- Decerebrate spasms
- Tetanus
- Malignant hyperthermia
- Malignant neuroleptic syndrome
- Paroxysmal dyskinesia
- Acute chorea, ballismus, dystonia

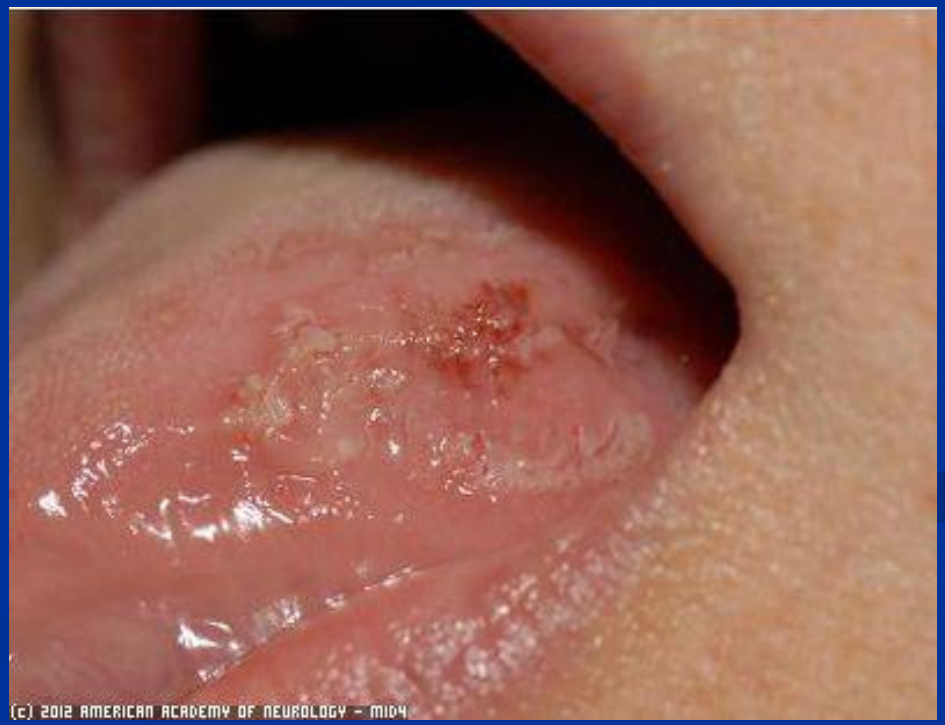
# Clinical distinction of dissociative non-epileptic attacks ("pseudoseizures") from epileptic seizures

| | Dissociative non-epileptic seizures ("pseudoseizures") | Epileptic seizures |
|---|---|---|
| Induced by anger, panic, suggestion | Common | Rare |
| Onset | Often gradual | Usually sudden |
| Duration | Often prolonged, occasionally hours | 1–3 minutes |
| Breathing and colour | Breathing continues, stays pink | Usually apnoeic and cyanosed |
| Retained consciousness | Common | Uncommon |
| Pelvic thrusting, back arching, erratic movements | Common | Rare |
| Fighting, held down, may injure others | Common | Rare |
| Eyes closed | Common | Less common |
| Resisting eye opening and eye contact | Common | Rare |
| Occur only in company | Common | Rare |
| Lateral tongue bite | Rare (minor) | Common |
| Self injury | Rare | Common (occasionally serious) |
| Incontinence | Rare (occasionally with experience) | Common |
| Post-ictal confusion | Rare | Common |

# Back arching in Pseudoseizures

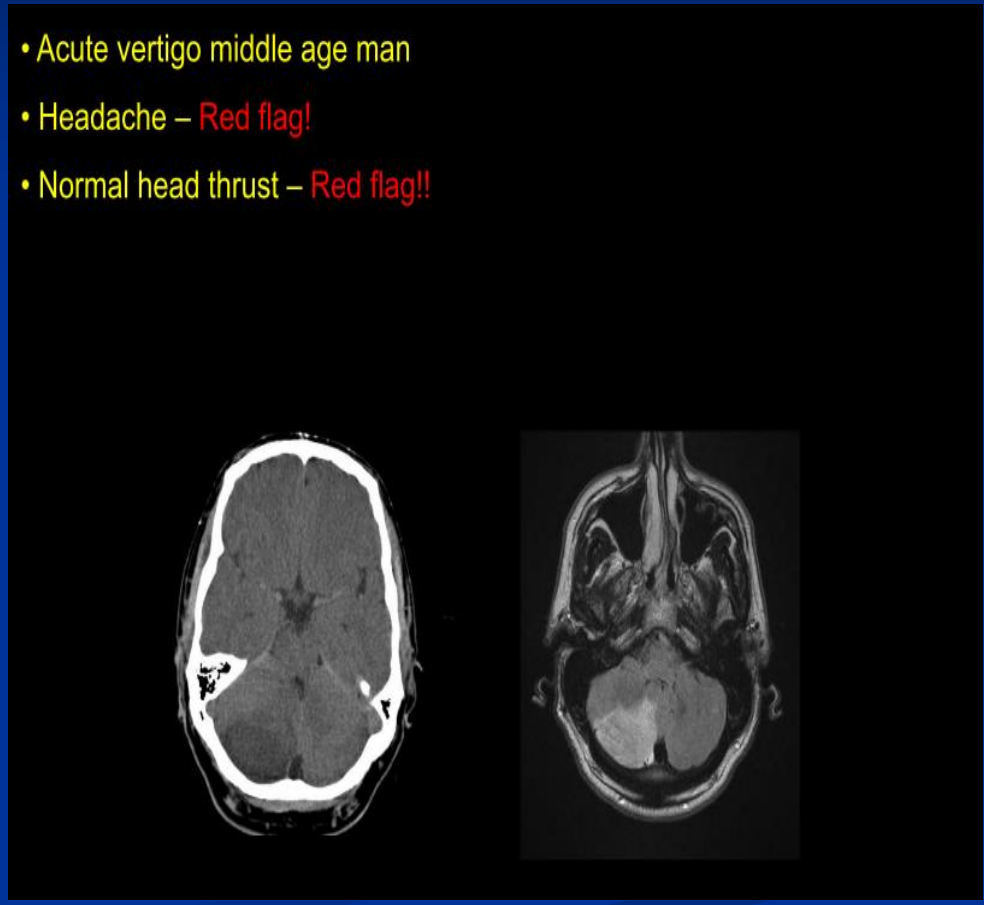
- Lateral tongue biting is poorly sensitive but highly specific (99%) for a generalized seizure.



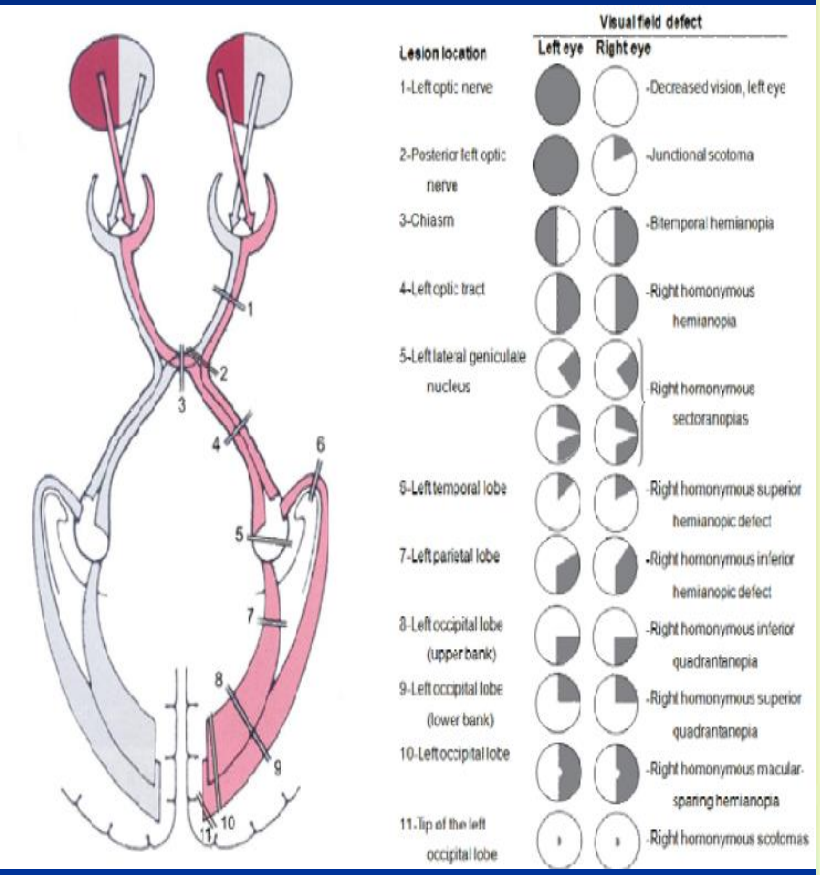


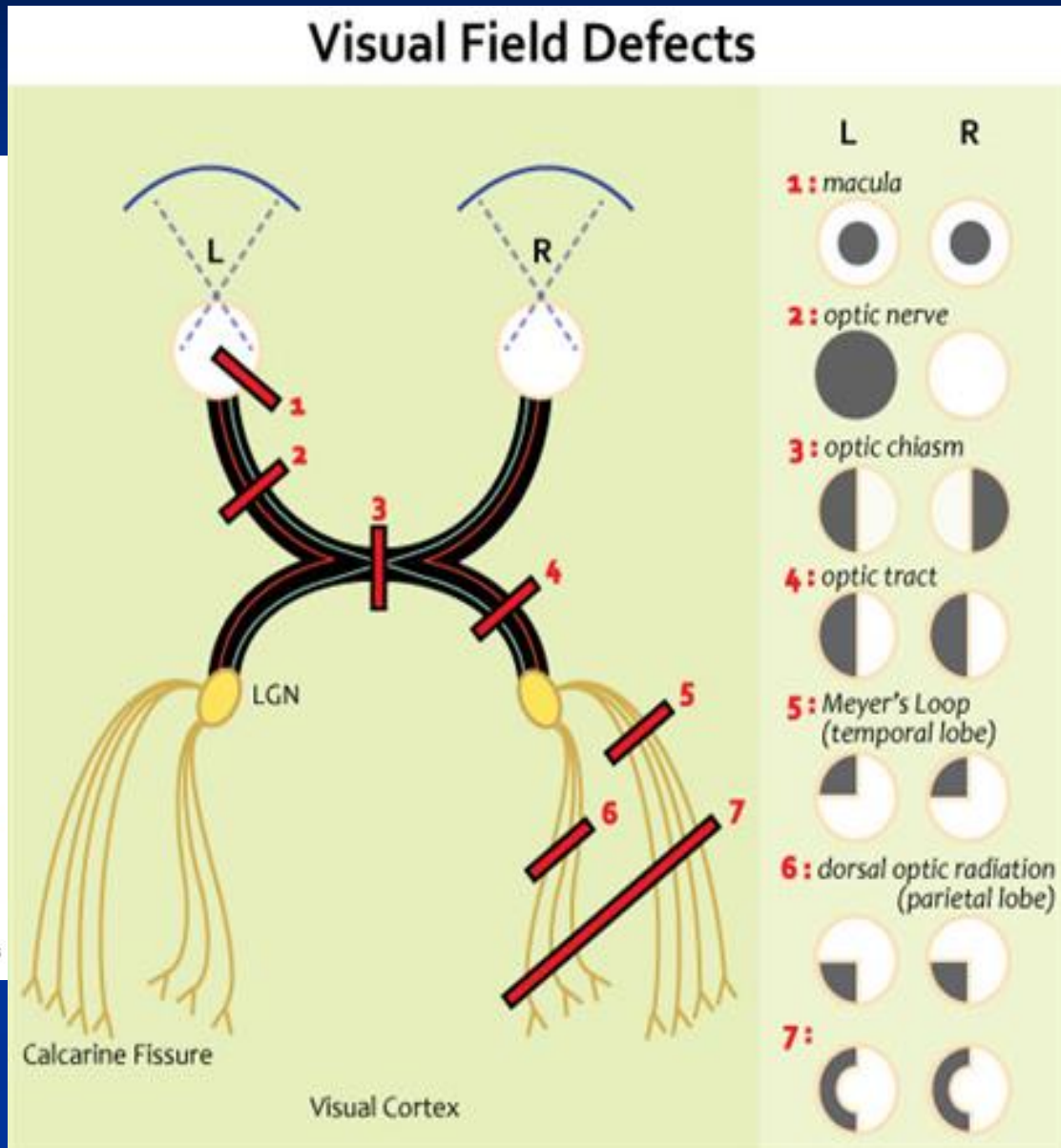
# Vertigo

- No Loss of consciousness
  (not syncope )
- Acute-onset persistent vertigo lasting hours-few days , think of:
  - Cerebellar stroke ( Red flags)
  - Vestibular neuritis
- If recurrent prolonged attacks- Vestibular migraine



# Anatomy of Visual Pathways



# WERNICKE ENCEPHALOPATHY

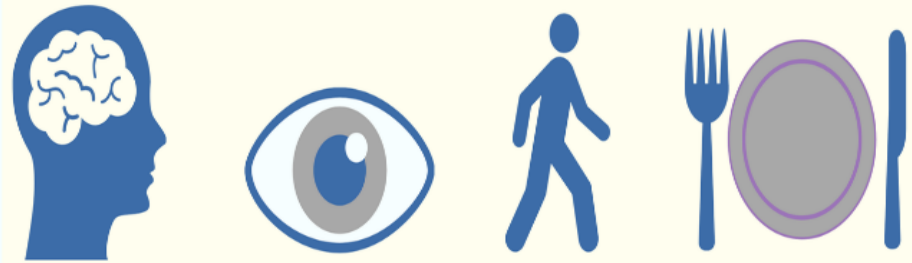
@Neudrawlogy

**Acute** B1 (thiamine) deficiency

www.neudrawlogy.com
Most updated version and website

## Diagnosis

**CLINICAL DIAGNOSIS: CAINE CRITERIA**
2 out of 4



- Changes in mental status or memory impairment
- Oculomotor dysfunctions ( e.g. ophthalmoplegia and nystagmus)
- Cerebellar dysfunction (e.g. gait ataxia - usually not present in upper limb)
- Nutritional deficiency

**This classic triad is present in only 10% -17% of the cases (more commonly in chronic alcoholism)**

## Why?

**Any risk factor for nutritional deficiency!**

- Chronic alcoholism
- Malignancy
- Hyperemesis of pregnancy
- Bariatric surgery
- Anorexia nervosa

## Treatment

***Intravenous thiamin infusion***

**Immediate treatment, as soon as you consider the diagnosis!**

- Administration of glucose without thiamine can worsen Wernicke Encephalopathy
- A prompt treatment can improve symptoms and prevent the development of Korsakoff syndrome (chronic B1 deficiency)

# Delay in the diagnosis and treatment of Wernicke's Encephalopathy may lead to death or dementia in survivors (Korsakoff's psychosis)