Neurological Emergencies/ CNS Infections

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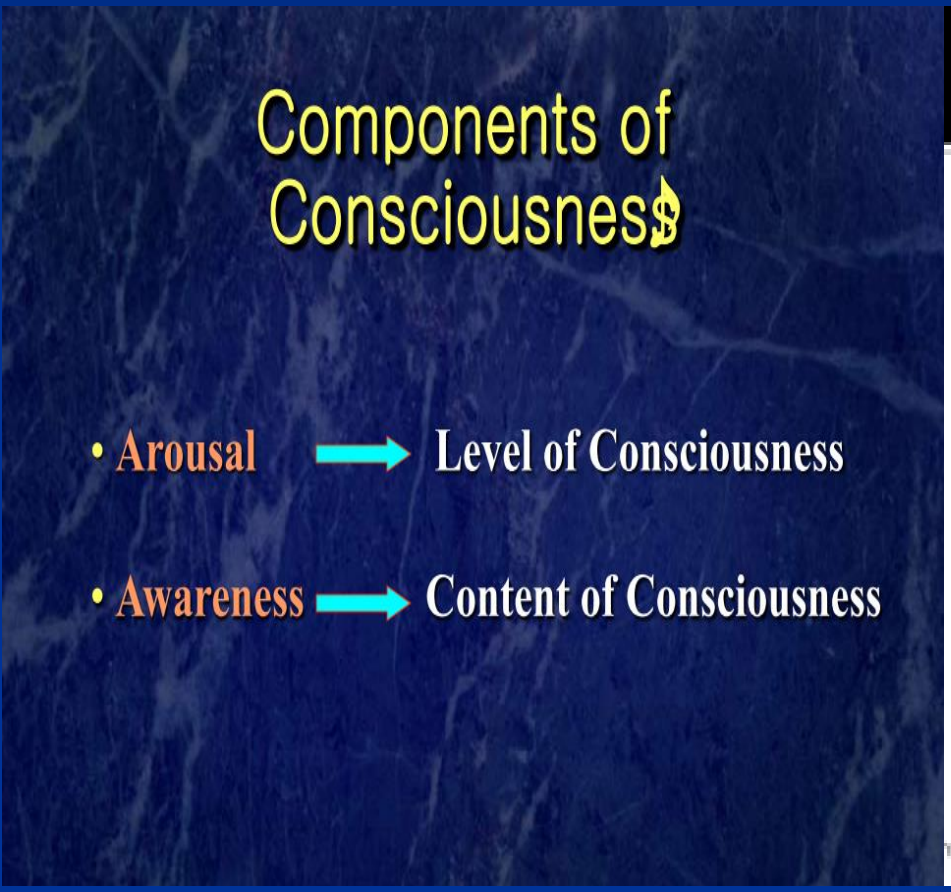
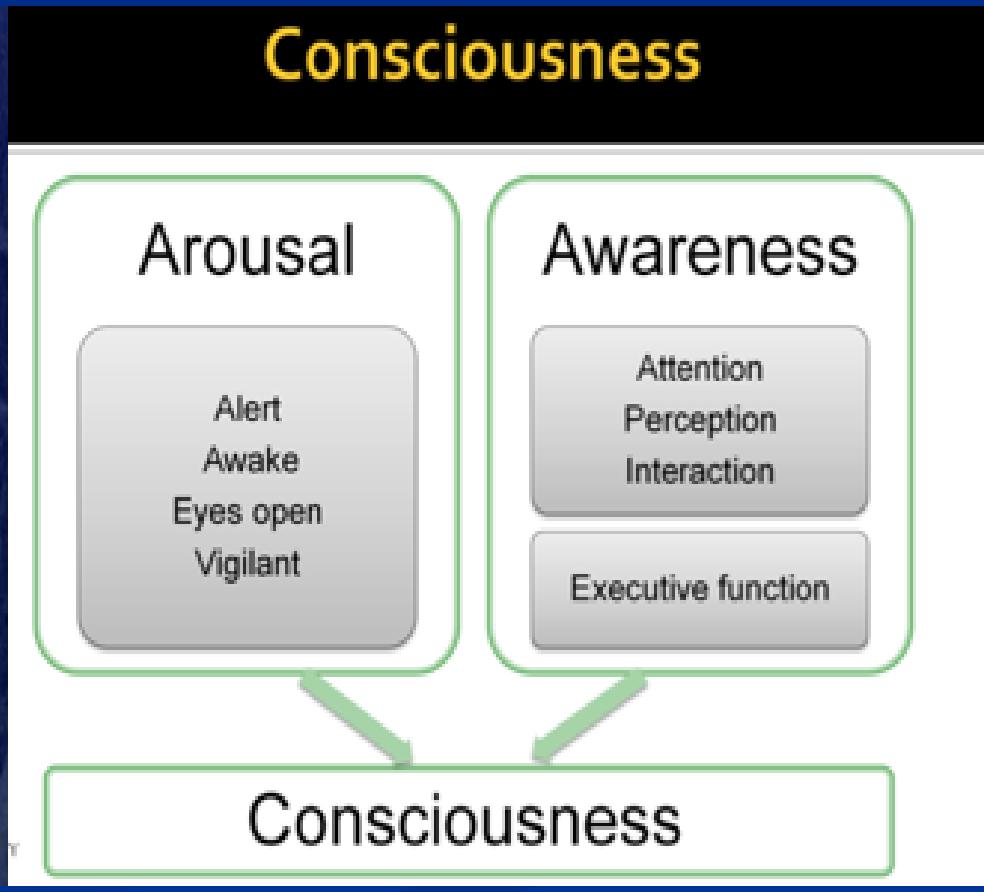
Neurological Emergencies-At least 20% of Medical ER visits

- 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





Content of consciousness Terminology

Table 1

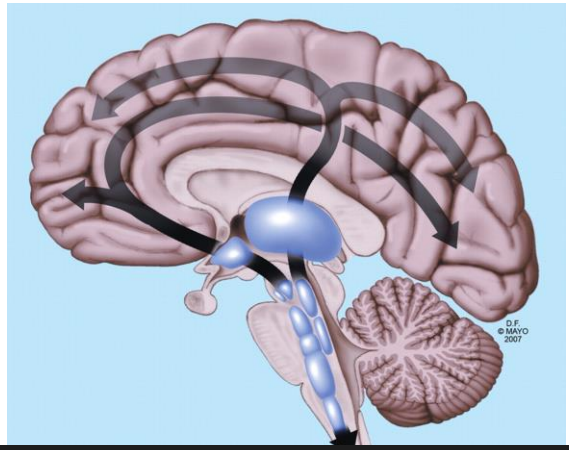
Terms for delirium

Acute confusional state
Acute brain failure
Agitation
Altered mental status
Confusion
Encephalopathy
ICU psychosis
Mental status changes
Sundowning
Terminal restlessness (eg, in palliative care)

Level 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.

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

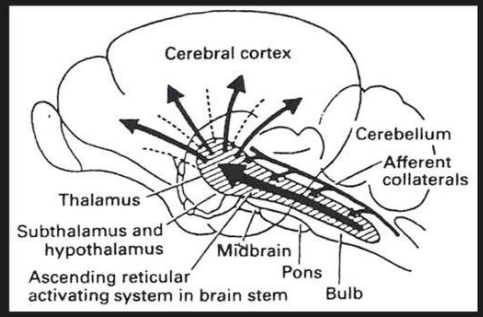


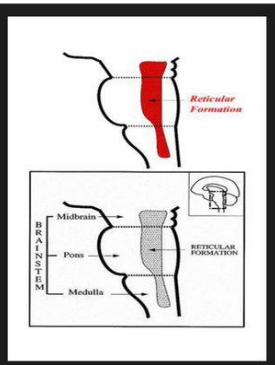
RAS

-Lesion in the mid-pons >>>>>>>unconsciousness

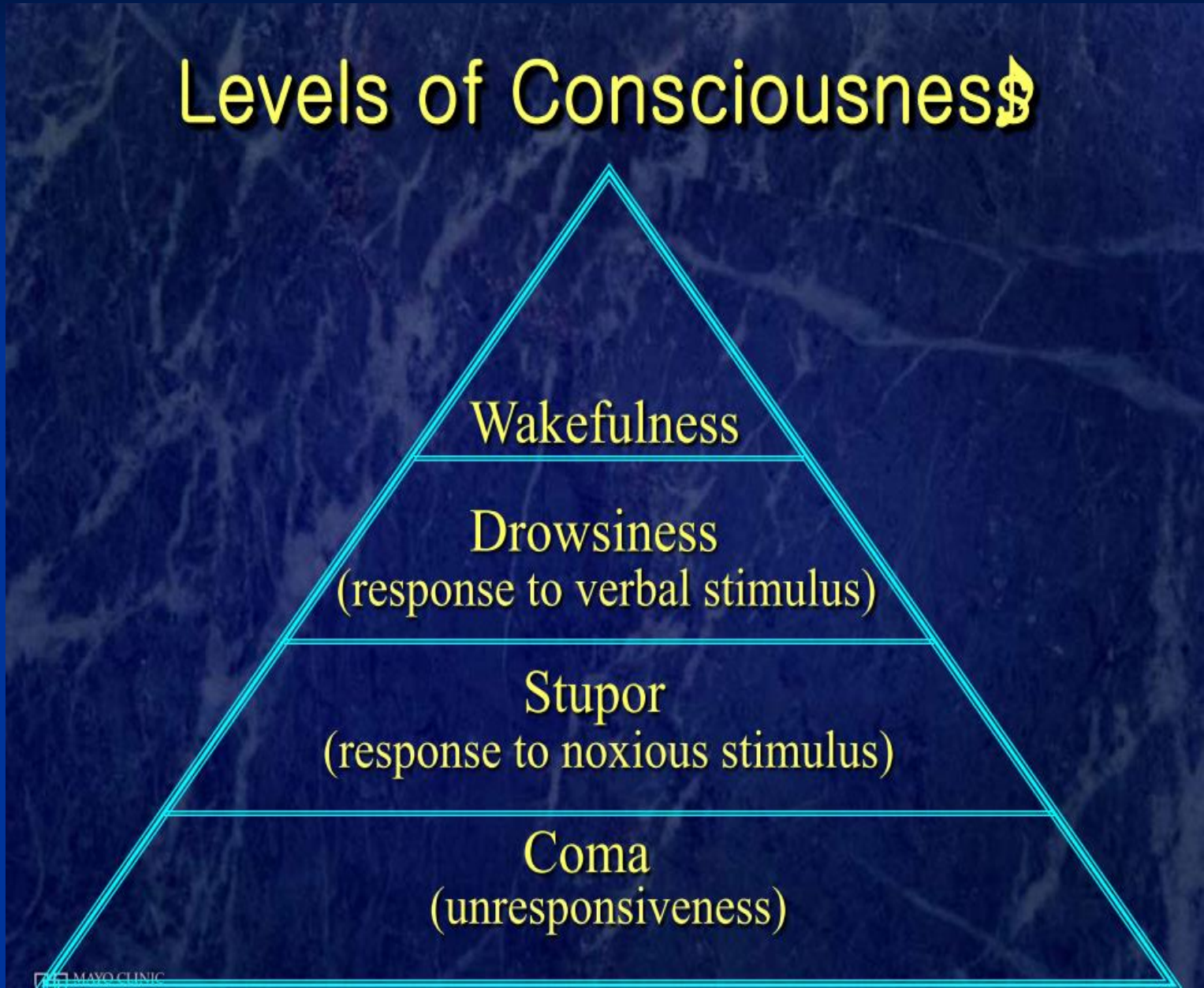
-Pons (uppers & middle) and midbrain are essential for wakefulness .

Bulboreticular Facilitory (Excitatory) = the reticular formation .





Levels of Consciousness



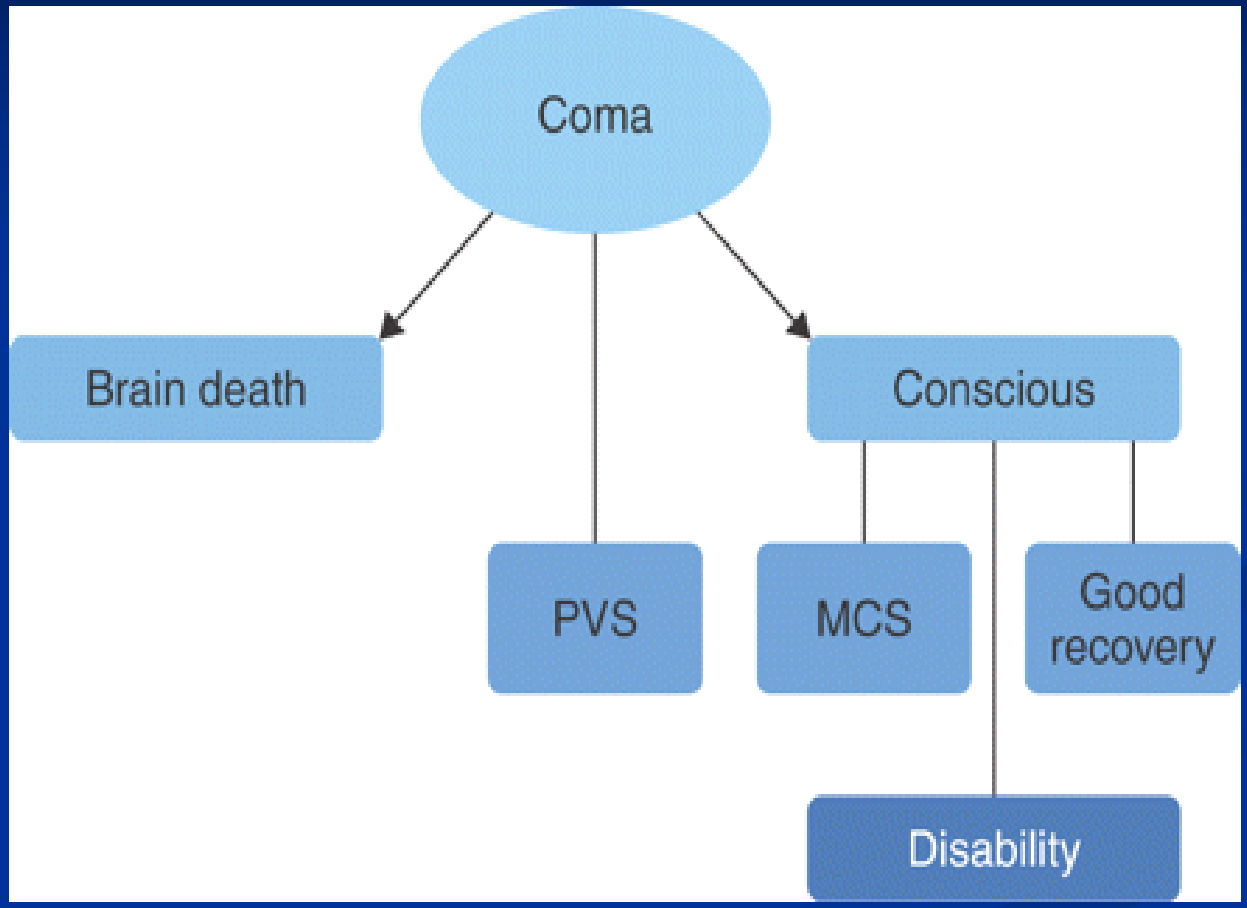
Glasgow Coma Scale to assess level of consciousness

- **Eye opening**
  - 4 = Spontaneous
  - 3 = To speech
  - 2 = To pain
  - 1 = None
- **Best motor response**
  - 6 = Obeying
  - 5 = Localising pain
  - 4 = Withdrawal
  - 3 = Abnormal flexing
  - 2 = Extensor response
  - 1 = None
- **Best verbal response**
  - 5 = Oriented
  - 4 = Confused conversation
  - 3 = Inappropriate words
  - 2 = Incomprehensible sounds
  - 1 = None

Coma is defined as a completely unaware patient unresponsive to external stimuli with:

Brainstem reflexes can be intact or absent

Outcome of Coma



PVS – Permanent vegetative state
MCS – Minimally conscious state

Evaluation of the Comatose Patient

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

1. Structural injury of the cerebral hemisphere(s).

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

3. Acute metabolic or endocrine derangement

4. Diffuse physiological brain dysfunction

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

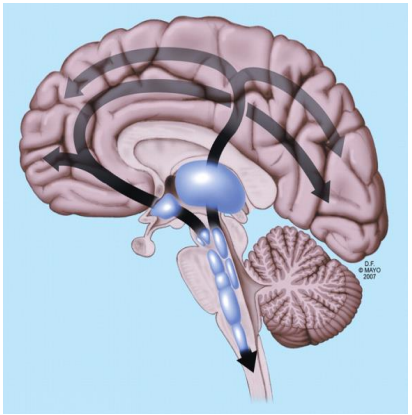
Bilateral

- 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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Acute metabolic/endocrine derangement

- Hypoglycemia (< 40-45 mg/dl)
- Hyperglycemia (non-ketotic hyperosmolar) > 600 mg/dl
- Hyponatremia <110 mmol/l
- Hypernatremia >160 mmol/l
- Addison's disease
- Hypercalcemia> 13.5 mg/dl
- Acute hypothyroidism
- Acute panhypopituitarism
- Acute uremia
- Hyperbilirubinemia
- Hypercapnia > 65 mmHg

Diffuse physiological brain dysfunction

- Generalised tonic–clonic seizures
- Hypoxic-Ischemic Encephalopathy
- Poisoning, illicit drug use
- Hypothermia
- Gas inhalation
- Acute (lethal) catatonia
- Malignant neuroleptic syndrome

Two Pitfalls
Can mimic coma

1- locked-in syndrome

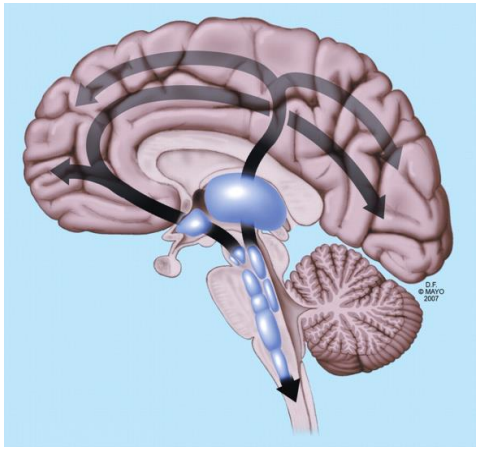
- Eyes open.
- Blink to commands or move their eyes vertically.
- lesion (stroke) in the ventral pons damaging the corticospinal and corticobulbar tracts and sparing the ascending reticular activating system. So they can hear, see and feel pain.

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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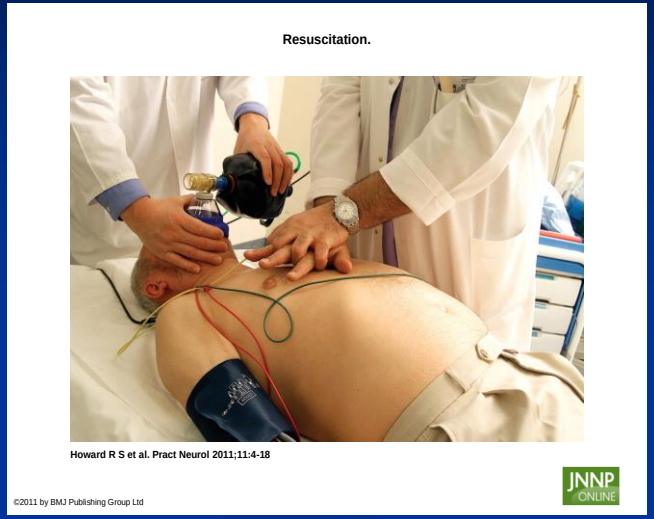
Clinical Assessment- History

- Cardiac arrest ?
- Overdose/ psychiatric problems?
- Could this be a CNS infection?
  - Did the patient use antibiotics for infection?
  - Was there a rapid onset of fever and headache?



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

- **Eye opening**
  - 4 = Spontaneous
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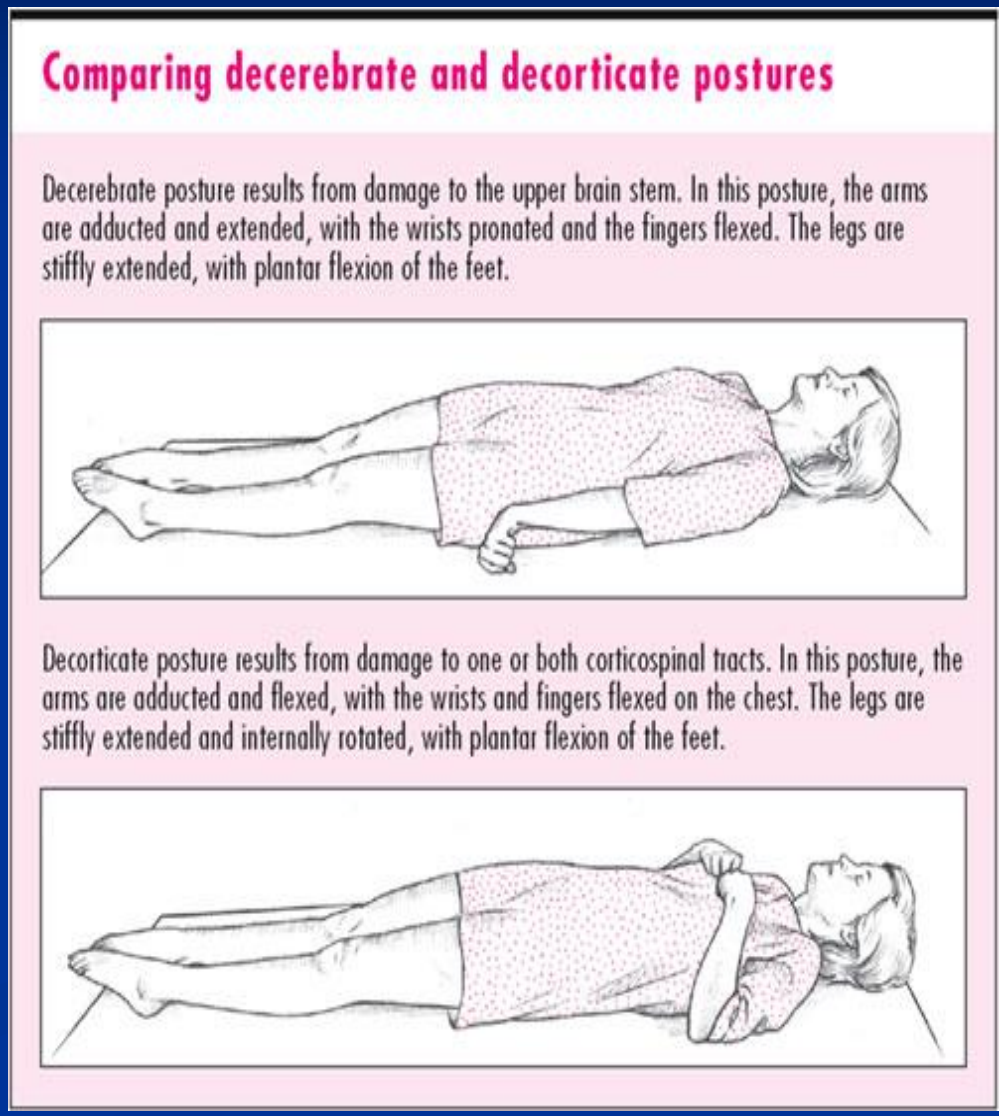
  - 1 = None

- Max. 15
- Min. 3
- Record subsets:

  E( )+ M( ) + V( ) = ?/15
- A score of </= 8 …. coma
- Verbal response can be compromised by endotracheal intubation …. V(T) should be recorded.

Motor responses

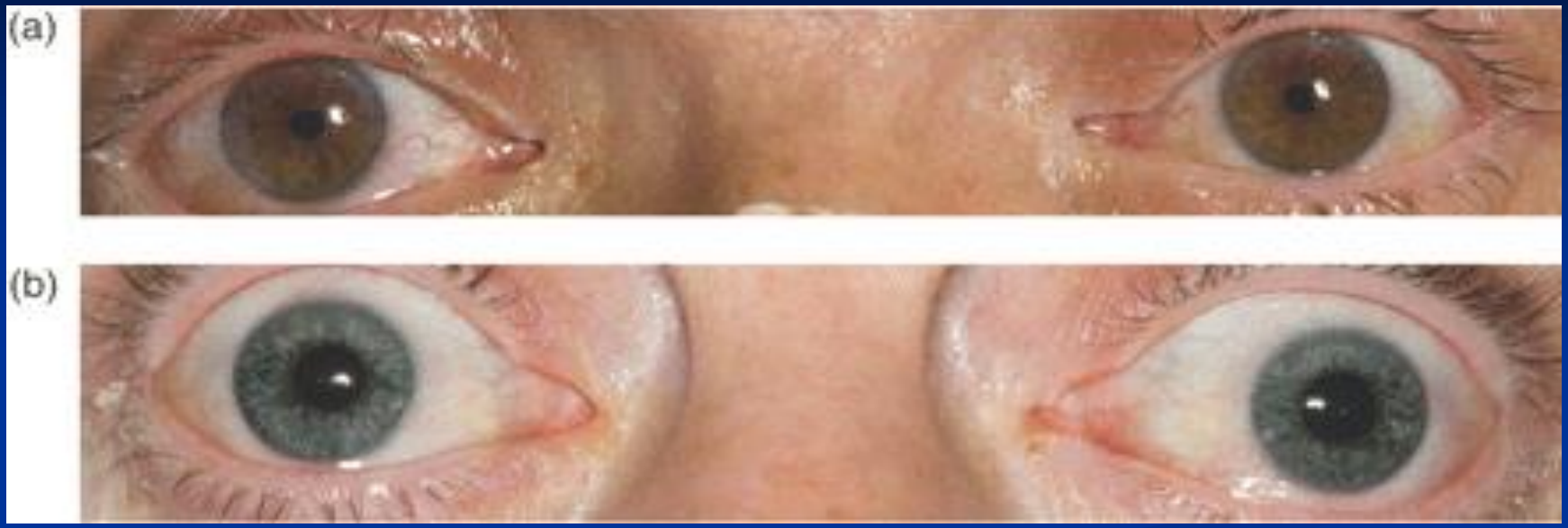
- Compression over the supraorbital nerve, sternum or nailbed.
- Flexion, extension or no response at all.
- The distinction between decerebrate and decorticate responses may not have significance for prognostication (both responses can be present in the same patient).



Lesion Localisation-Brainstem lesions

- Intrinsic lesions are recognised by skew deviation of eyes , internuclear ophthalmoplegia, small or unequal pupils and absent oculocephalic responses
- Brainstem displacement caused by lesions above the tentorium is recognised by a wide, fixed pupil, abnormal motor responses but otherwise intact brainstem reflexes;
- Brainstem displacement from below the tentorium (e.g, cerebellar lesions) is recognised by small pupils, absent corneal reflexes and oculocephalic responses (in some patients.)

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.

Skew deviation of the eyes

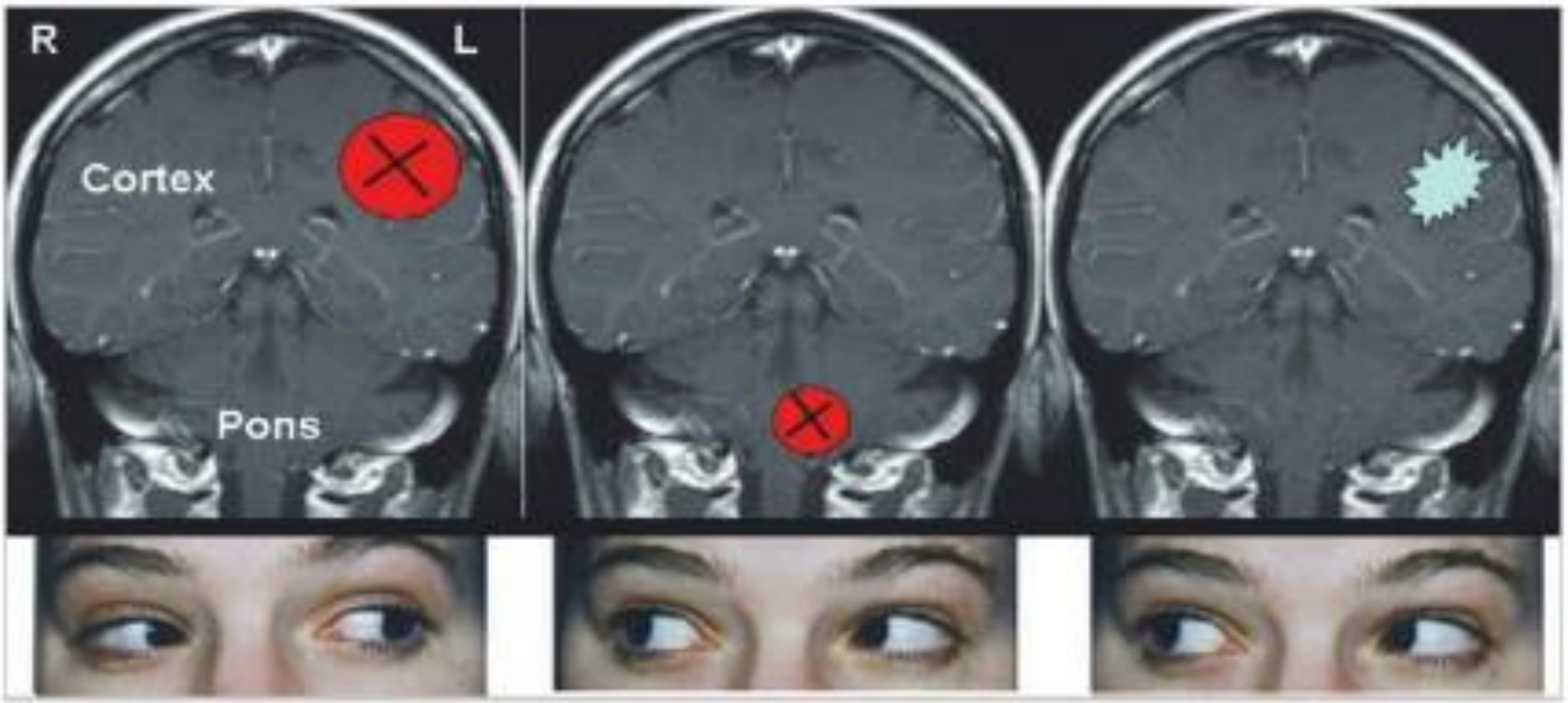
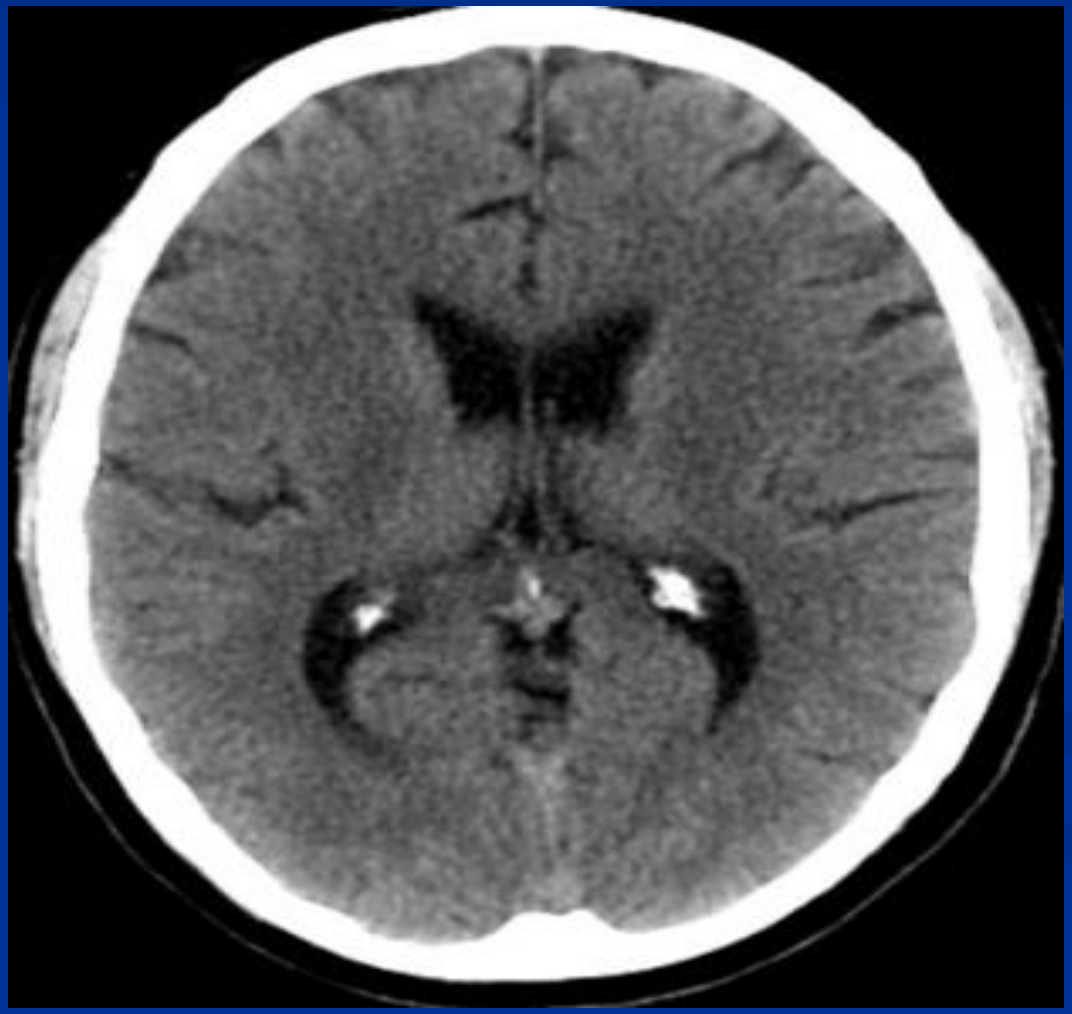


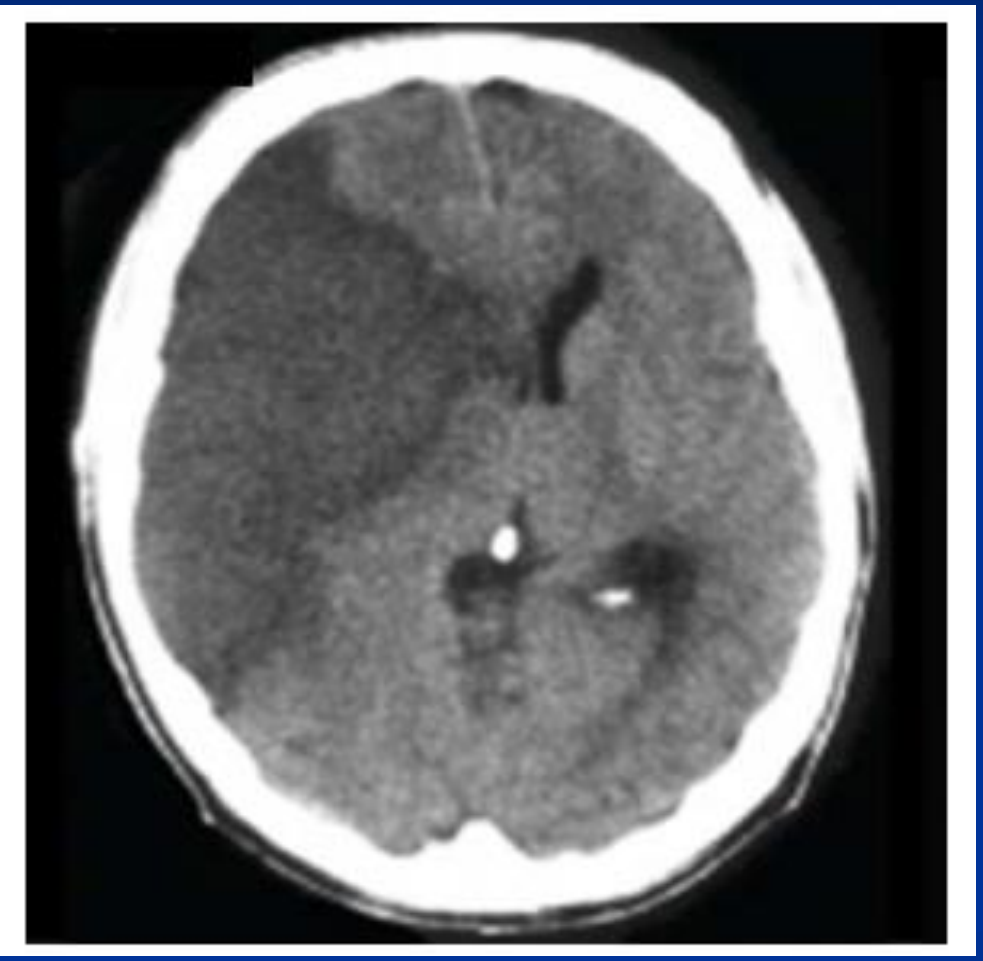
Fig. 13.121 Gaze deviations in cerebral lesions and seizures: the eyes deviate horizontally toward a cortical lesion (left); the eyes deviate horizontally away from a pontine lesion (center); the eyes deviate horizontally away from a cortical seizure focus (right).

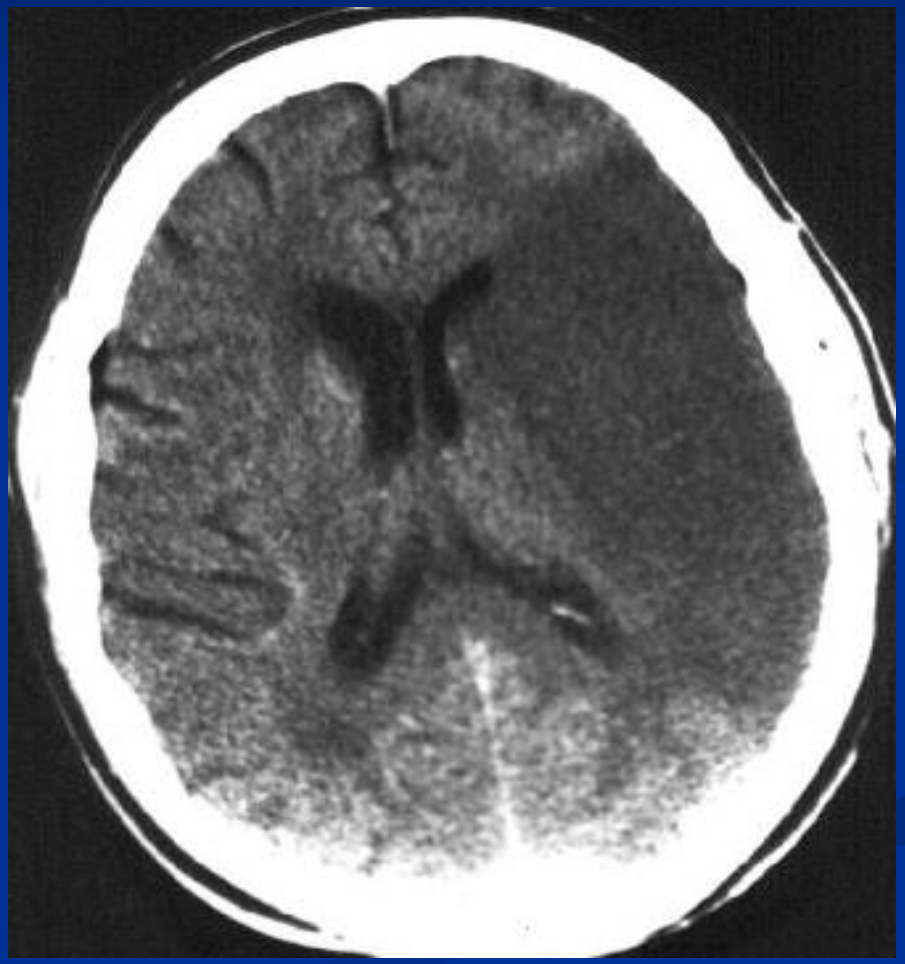
What is the cause of coma ?

- CT and MRI of the brain are very important in the workup of a comatose patient

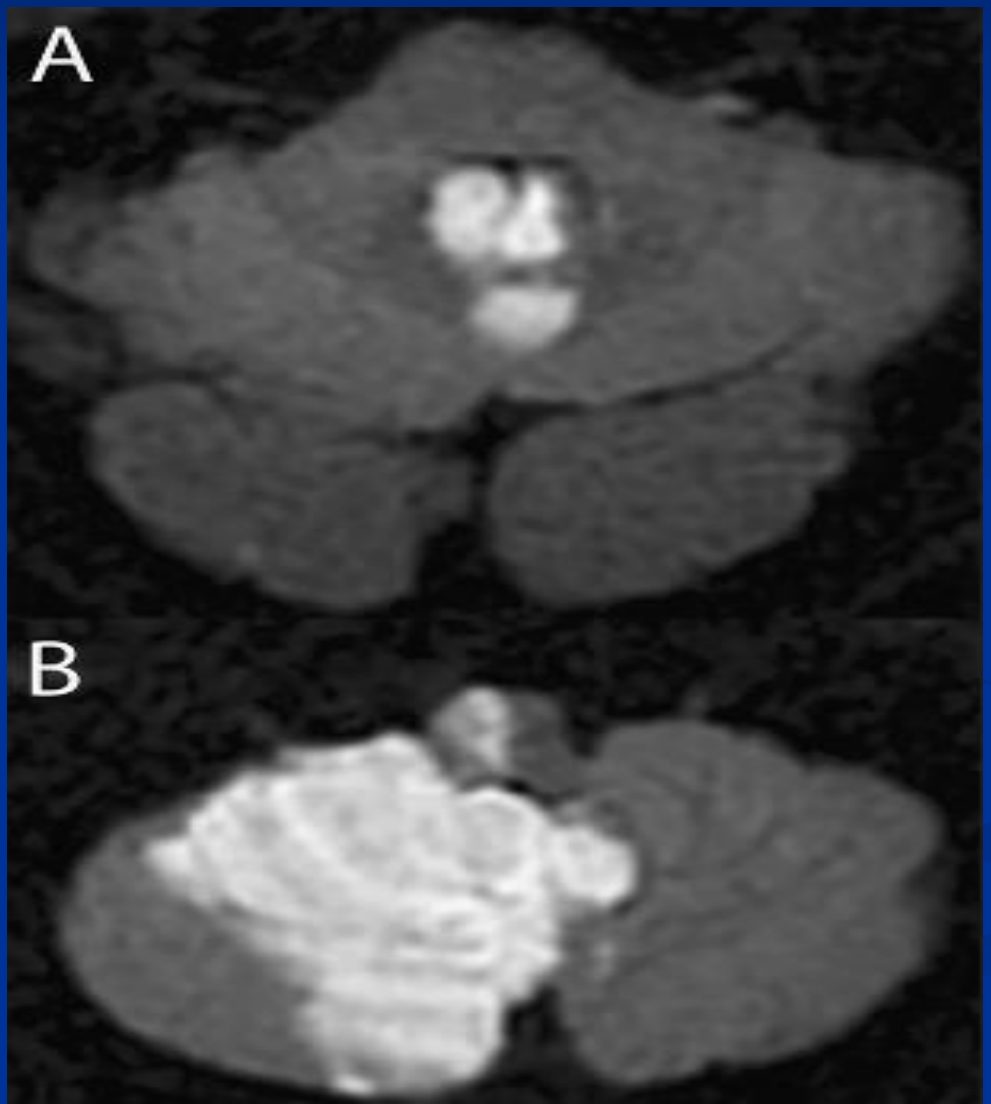


Middle Cerebral Artery Infarction

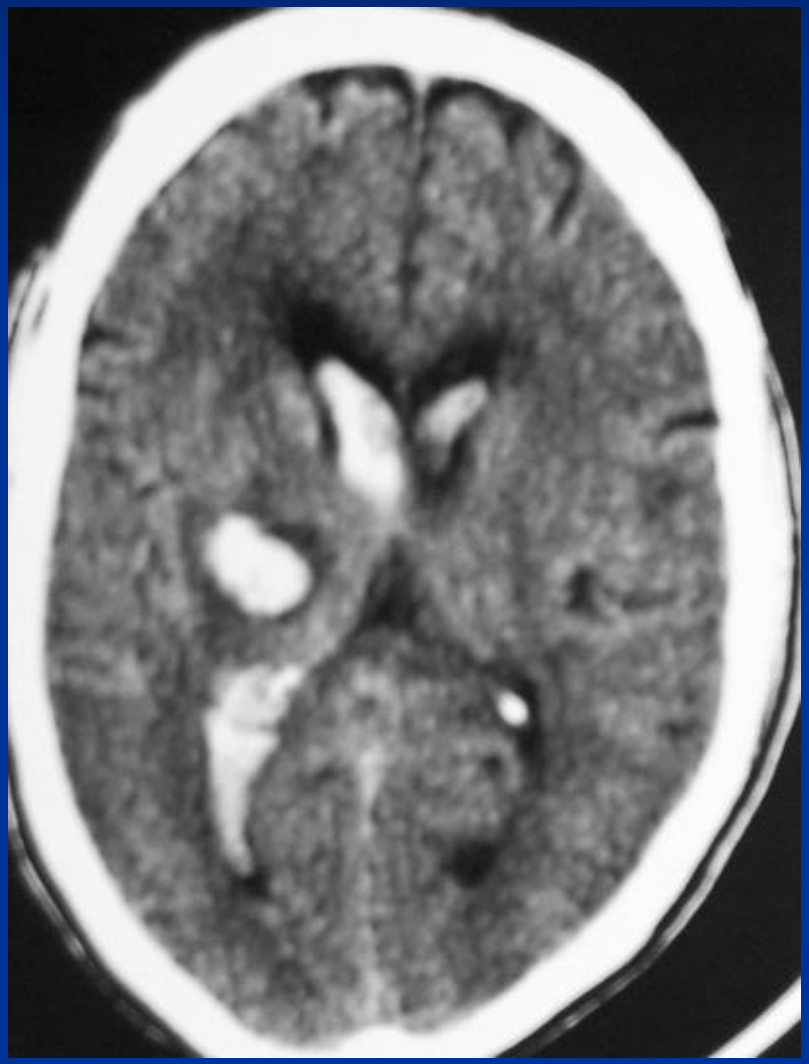


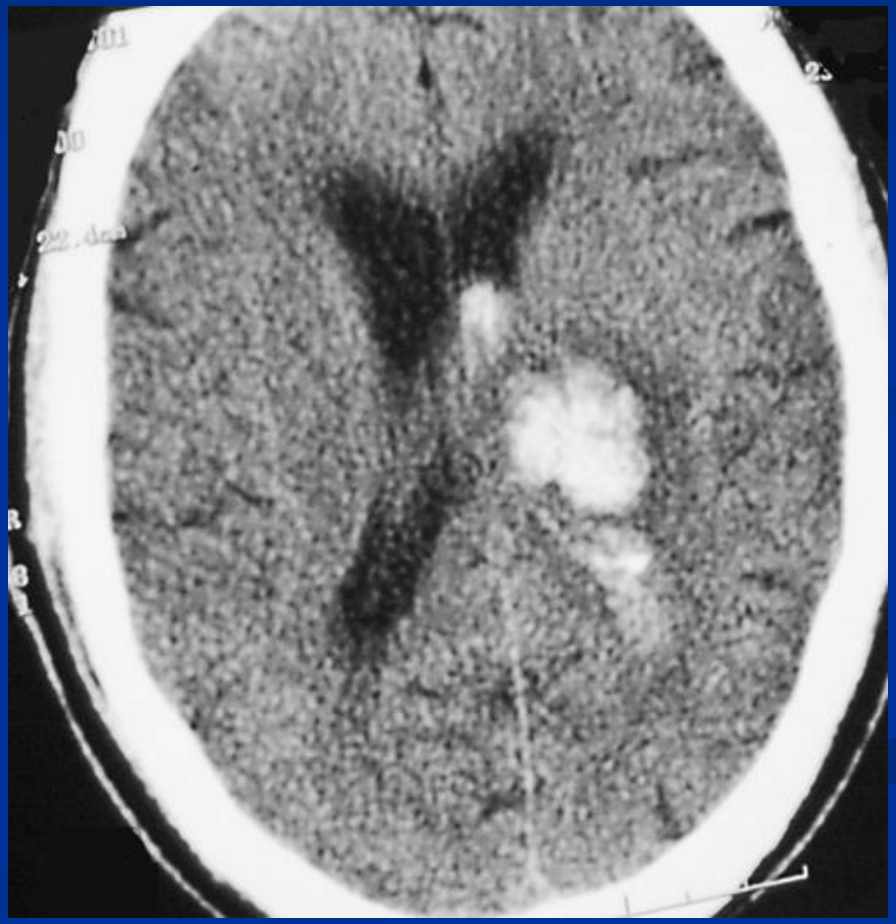


Acute infarction on MRI-DWI sequence

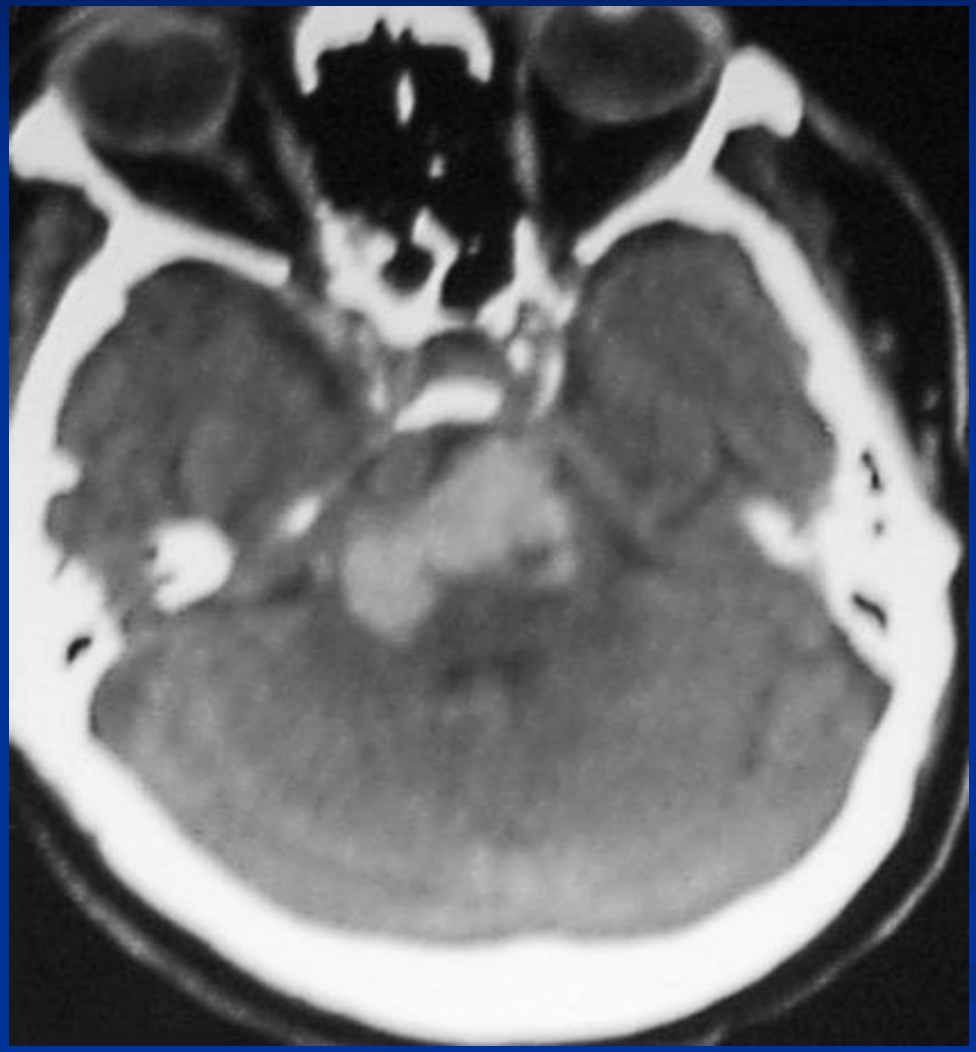


Thalamic Intraparenchymal Hemorrhage with intraventricular extension

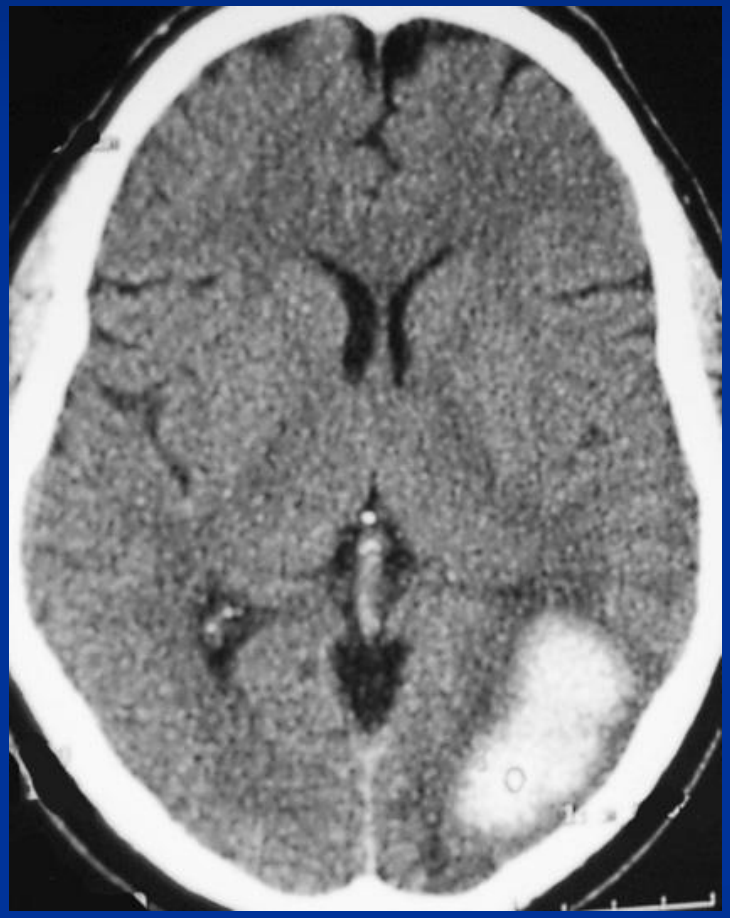


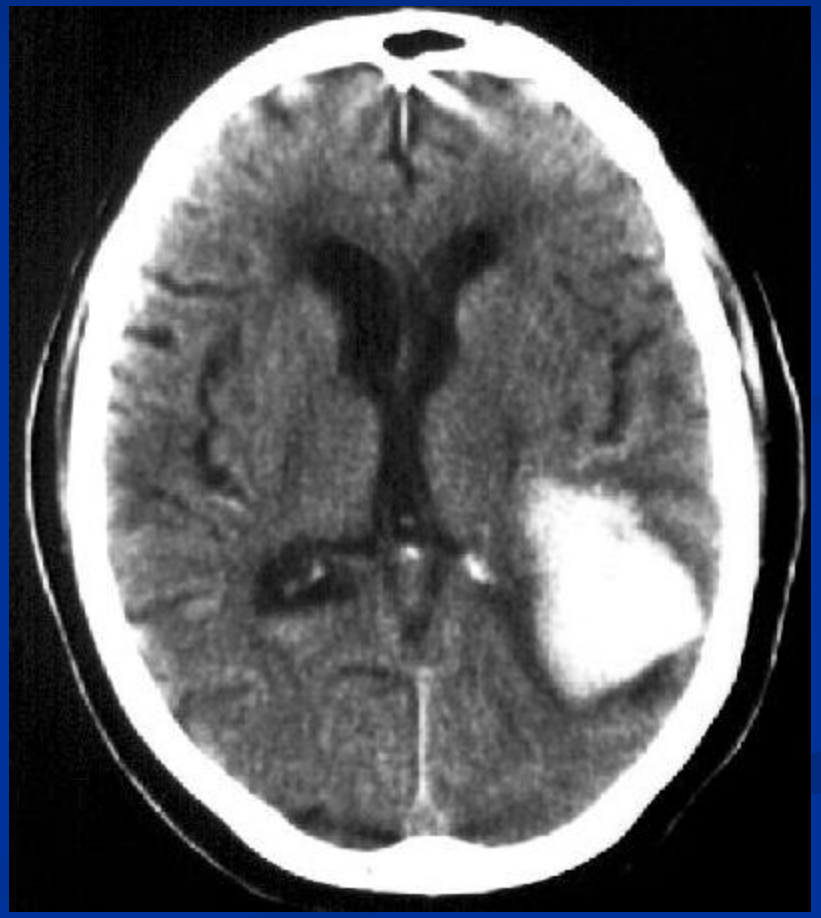


Pontine Intraparenchymal Hemorrhage

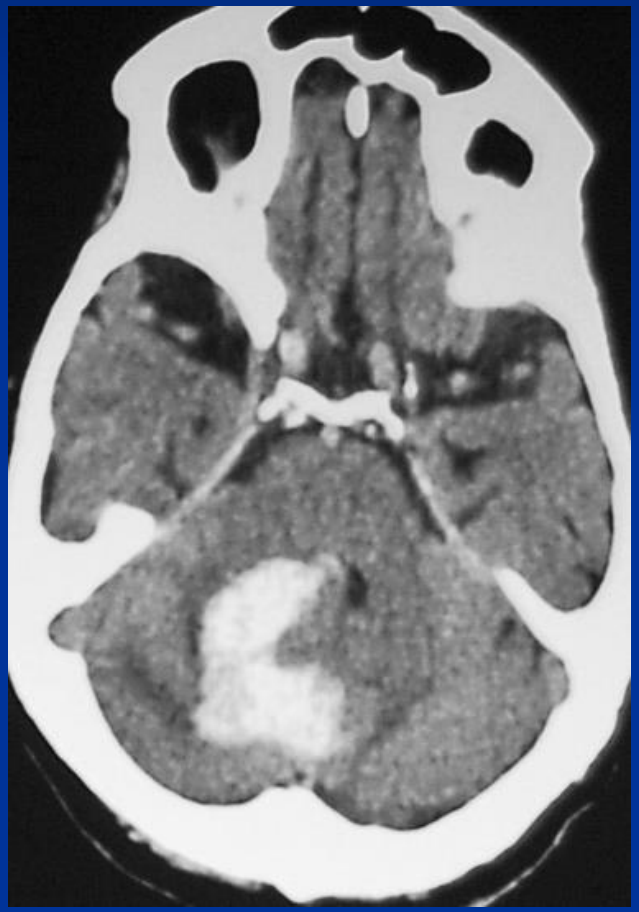


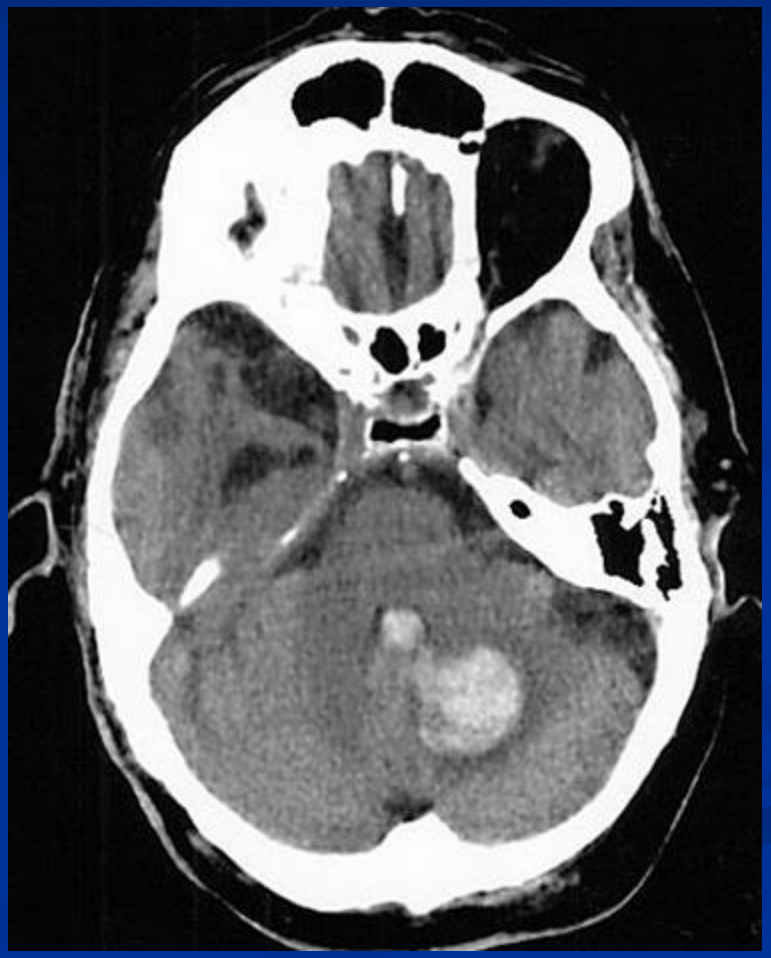
Lobar Intraparenchymal Hemorrhage



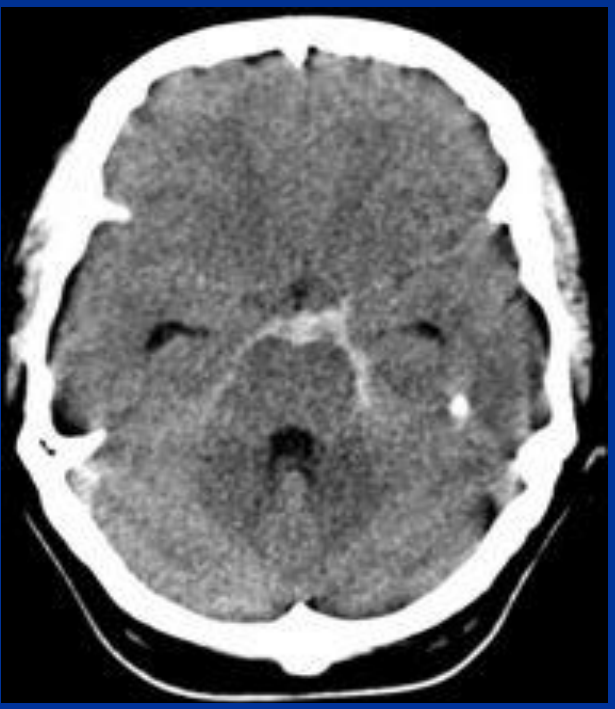
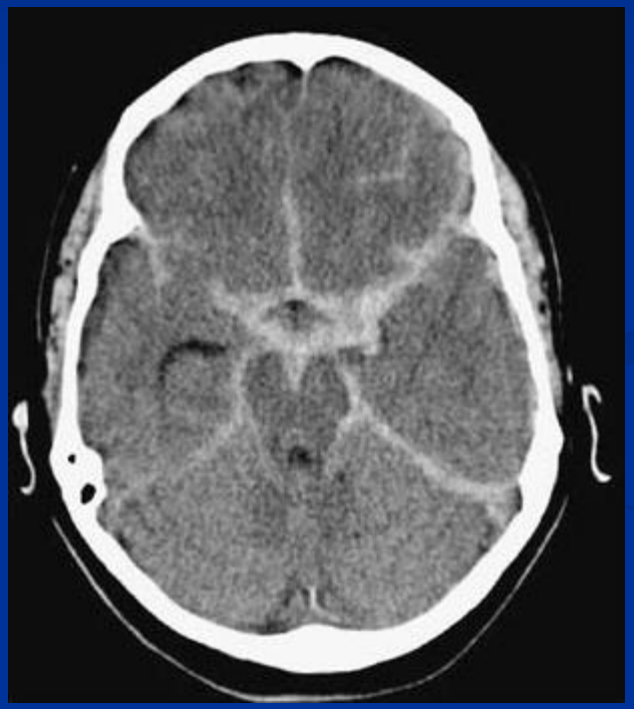
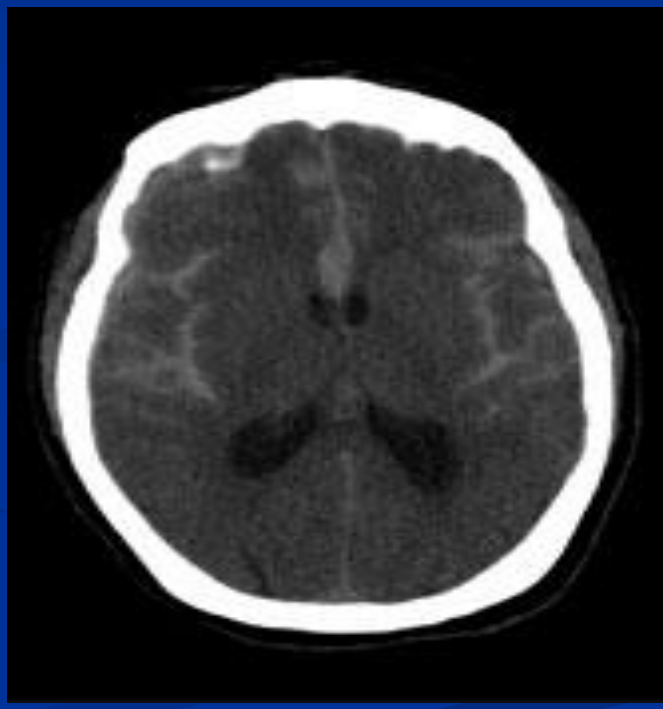


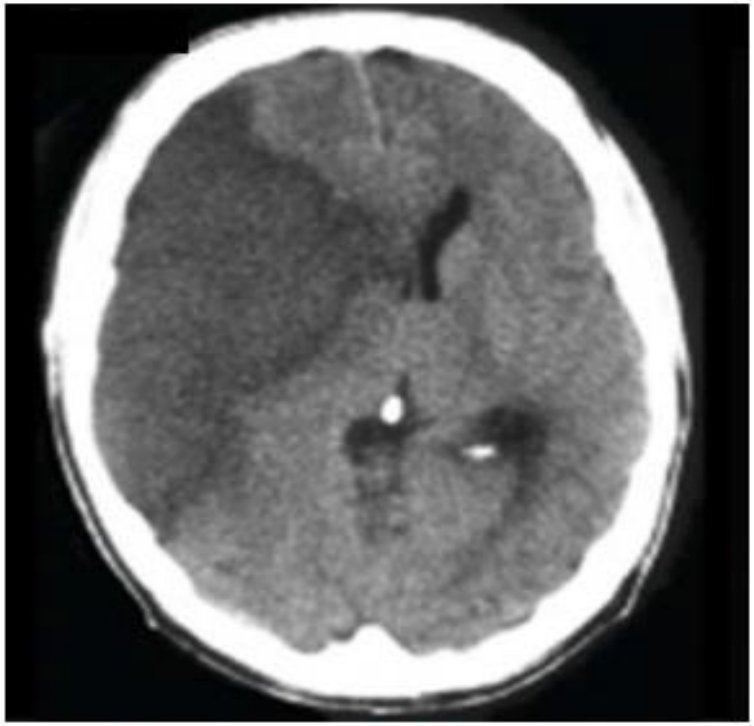
Intraparenchymal Cerebellar Hemorrhage



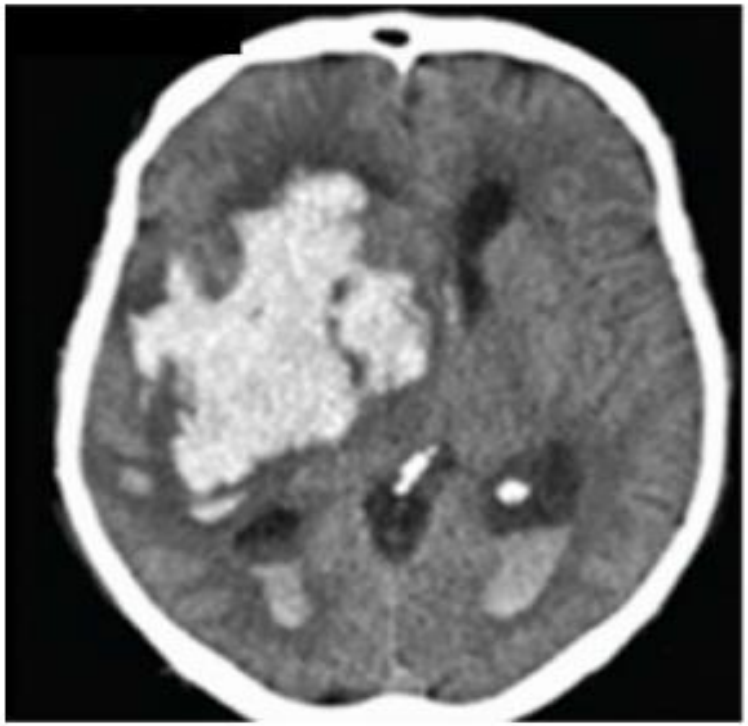


Subarachnoid hemorrhage



Ischemic Stroke
(dark/hypodense)



Hemorrhagic Stroke
(bright/hyperdense)

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, increased work of breathing, pooling secretions, gurgling sounds).
- Intubate any comatose patient with irregular ineffective respiratory drive and poor oxygenation.
- Intubate any comatose patient with major facial injury or consider emergency tracheostomy.

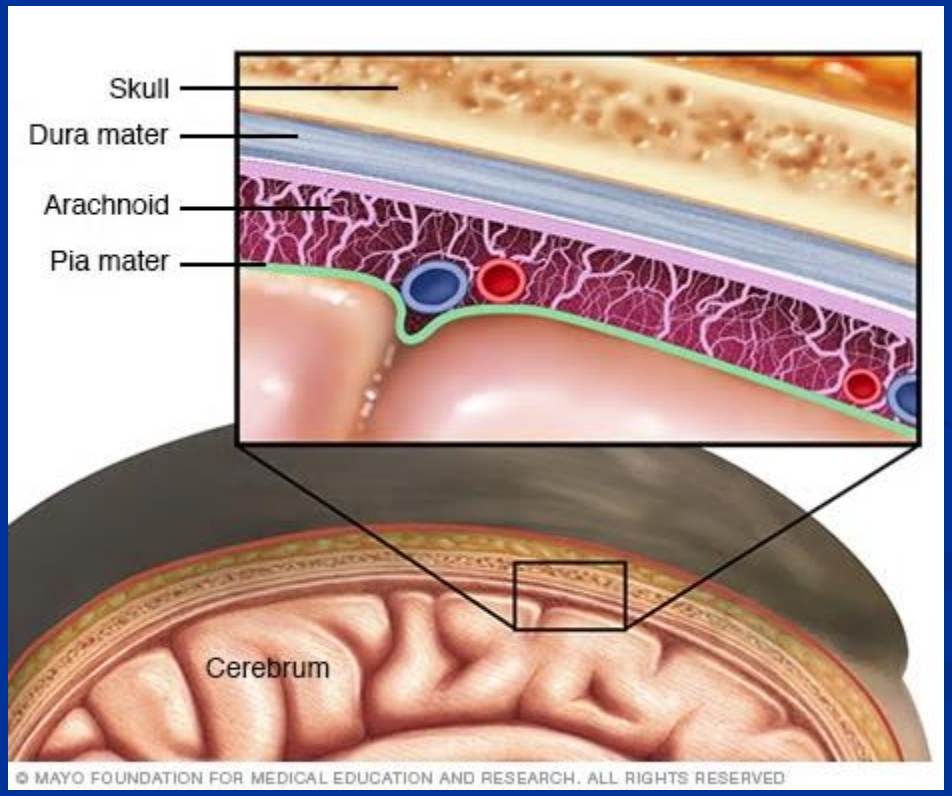
Management of Coma in the First Hour

- 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?

Meningitis

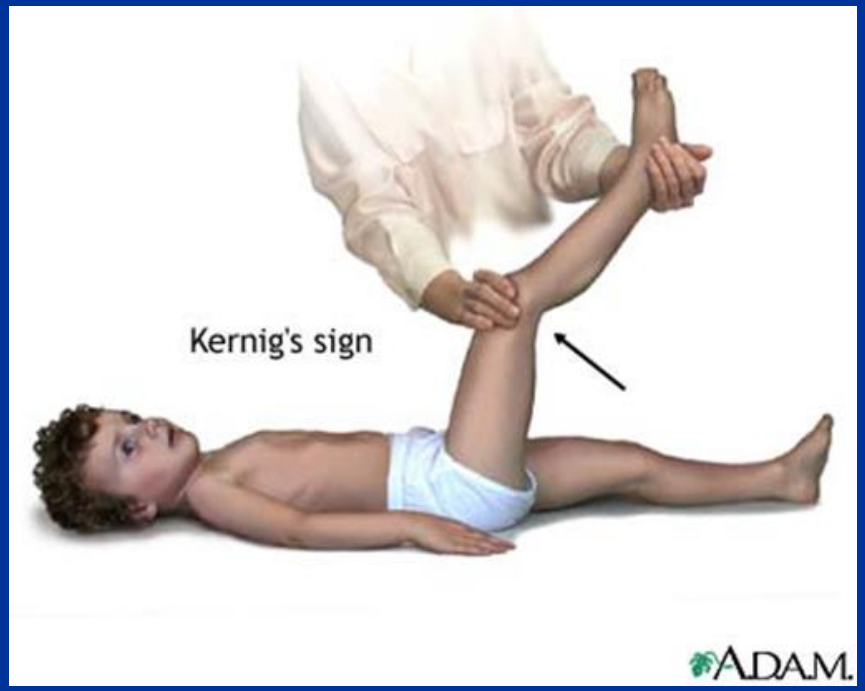
- Meningitis: is an inflammation of the membranes (meninges) surrounding your brain and spinal cord.

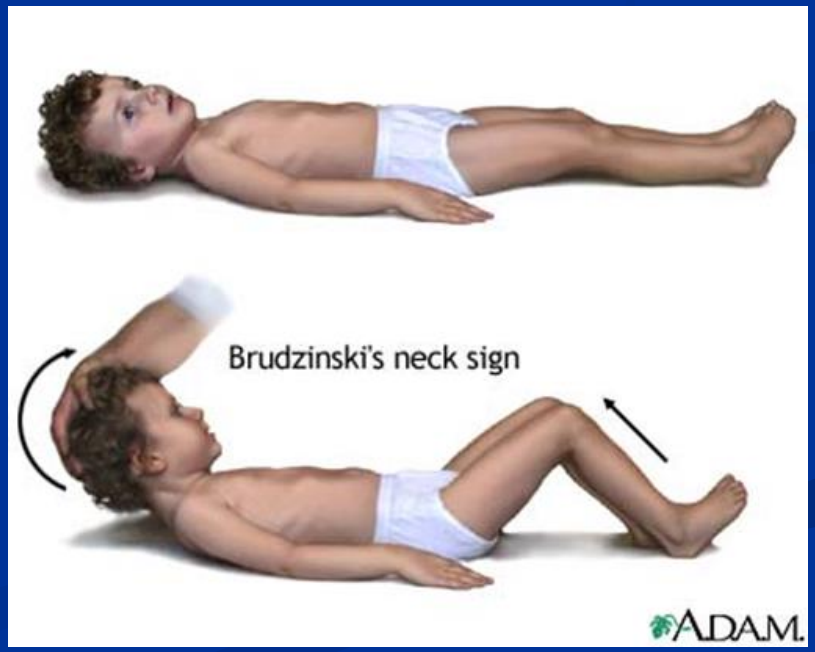


Symptoms

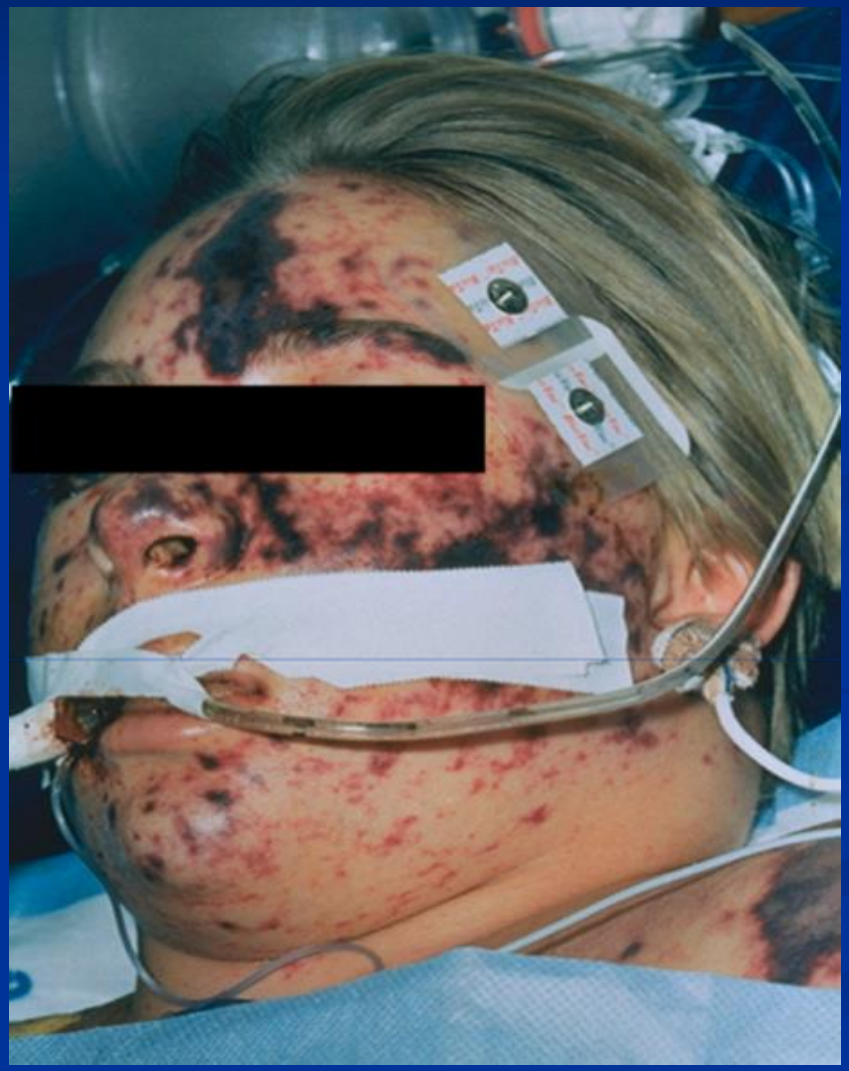
- Early meningitis symptoms may mimic the flu (influenza). Symptoms may develop over several hours or over a few days.
- Possible signs and symptoms:
  - High fever
  - Nuchal rigidity and other meningeal irritation signs/Photophobia
  - Worsening headache (+/- Nausea or vomiting)
  - Confusion/irritability/difficulty concentrating/drowsiness/coma
  - Seizures
  - Skin rash (in meningococcal meningitis)

Important meningeal signs





Purpuric rash of meningococcal meningitis



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

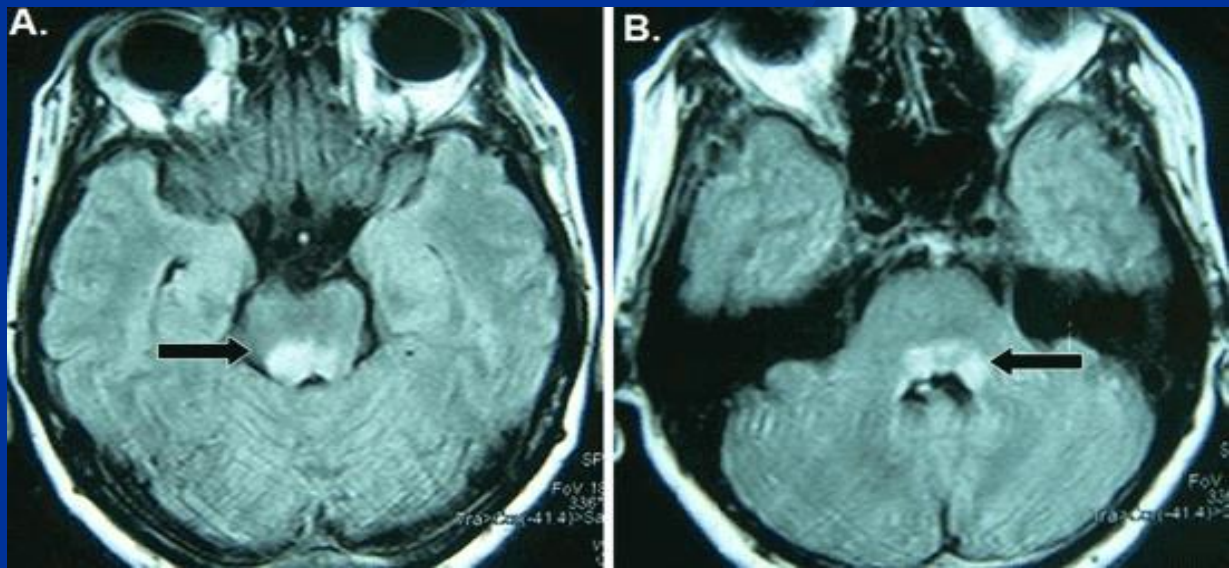
ABM : Etiology

- **Streptococcus pneumoniae (pneumococcus).** This bacterium is the most common cause of bacterial meningitis in infants, young children and adults. A vaccine can help prevent this infection.

- **Neisseria meningitidis (meningococcus)**. This bacterium is another leading cause of bacterial meningitis. These bacteria commonly cause an upper respiratory infection but can cause meningococcal meningitis when they enter the bloodstream. **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. A vaccine can help prevent infection.

ABM : Etiology

- **Haemophilus influenzae type b** was once the leading cause of bacterial meningitis in children. But new Hib vaccines have greatly reduced the number of cases of this type of meningitis.
- **Listeria monocytogenes -** These bacteria can be found in unpasteurized cheeses, hot dogs and lunchmeats. Pregnant women, newborns, older adults and people with weakened immune systems are most susceptible. Listeria can cross the placental barrier, and infections in late pregnancy may be fatal to the baby.



ABM: Epidemiology

- Max in 1st 5 yrs
- Risk Factors:
  - Colonization
  - Crowding: person to person droplet infection
  - Poverty
  - Male
  - Absence of breast feeding
  - Immunodeficiency
  - Skull base/dural defects

ABM: Pathology

- Bacterial colonization of nasopharynx → bacteremia → choroid plexus → meninges

- Meningeal exudates, ventricultis, perivascular inflammatory exudates, venous occlusion, infarction, necrosis, ↑ICP

ABM: Clinical Features

- Sudden onset
- **high fever, headache**, anorexia, myalgia, photophobia, **meningeal signs, altered mental status/coma**
- **↑ICP**: hypertension, bradycardia, bulging fontanelles, 3rd/6th cranial nerve palsy, posturing, breathing abnormalities, papilledema
- Purpuric rash s/o meningococcus
- Septic foci

ABM

- Diagnosis
  - High index of suspicion very important
  - Confirm by CSF examination
  - LP deferred if there is contraindication
  - Start empirical antibiotics on suspicion

- CSF: ↑Pressure, turbid, ↑cells (mostly polys), ↑protein, ↓sugar to < 40% of blood sugar
- Gram stain, culture
- PCR

- Imaging

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

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 0.6 mg/ kg daily with or just before first dose of antibiotics, and continue for 4 days- benefit in pneumococcal meningitis

Treatment

- Subsequent therapy according to sensitivity
- Repeat LP/ imaging indicated if poor response
- Supportive Rx
  - IV Fluids ? Restrict
  - Management of ↑ICP : mannitol, acetazolamide
  - Rx of Seizures, pyrexia
  - Treat shock, DIC if present
  - Nutrition
  - Nursing

Tuberculous meningitis (TBM)

- Most dreaded and dangerous form of TB
- Risk Factors:
  - Young age
  - Household contact
  - Recent infection
  - Measles
- Pathophysiology
  - Primary infection → bacillemia → hematogenous seeding of meninges (Rich's foci) → rupture
  - Thick exudates in basal cisterns
  - Arteritis

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.

Encephalitis

- Encephalitis is a clinical syndrome characterized by alteration of consciousness/confusion and variable combinations of headache, fever, seizures and focal neurological signs, in response to brain inflammatory damage.
- In practice, little distinction may initially be seen between meningitis and encephalitis and the term **meningoencephalitis** is often used – both covered initially.

Encephalitis vs. Encephalopathy

- We also have to distinguish encephalitis from other causes of encephalopathy, including systemic infection, metabolic derangements, toxins, inherited metabolic disorders, hypoxia, trauma, epilepsy, thromboembolic stroke and other vasculopathies.

Encephalopathy

- Clinical syndrome of altered mental status (manifesting as reduced consciousness or altered cognition, personality or behavior)
- Has many causes including systemic infection, metabolic derangement, inherited metabolic encephalopathies, toxins, hypoxia, trauma, vasculitis, or central nervous system infection

Encephalitis

- Inflammation of the brain
- Strictly a pathological diagnosis; but surrogate clinical markers often used, including inflammatory change in the cerebrospinal fluid or parenchyma inflammation on imaging
- Causes include viruses, small intracellular bacteria that directly infect the brain parenchyma and some parasites
- Can also occur without direct brain infection, for example in acute disseminated encephalitis myelitis (ADEM), or antibody-associated encephalitis

Causes of infectious encephalitis

- Historically, encephalitis has been almost synonymous with direct infection, but we now recognise parainfectious or postinfectious causes, as well as non-infectious causes.

- Clinically, infectious encephalitis is characterised by **acute** onset of fever, altered mental status, focal neurological deficits and generalised or focal seizures.

Causes of infectious encephalitis

- It can be difficult to identify a specific cause, which remains undetermined in up to half of cases.

- Of identified **sporadic** causes**,** herpes simplex virus (HSV) is the most frequently found agent (25 – 40% of infectious etiologies), followed by enterovirus, varicella zoster virus and tuberculosis.

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.
- 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.

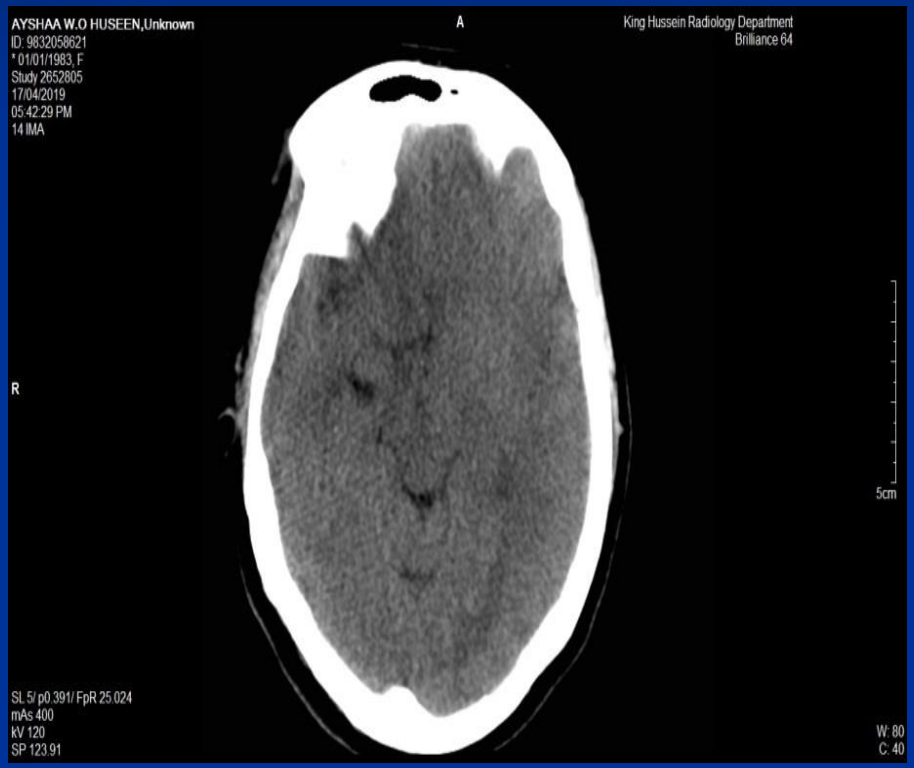
HSV Encephalitis

- Neutrophilia +/-
- CSF clear, pleocytosis +/-. Normal or ↑protein, normal sugar
- Specific Dx by PCR
- Imaging: normal/ edema/ patchy hypodensity/ specific changes
- EEG: nonspecific diffuse slowing, periodic discharges

HSV Encephalitis

- CSF herpes simplex virus PCR is both highly sensitive and specific and usually establishes the diagnosis but can be negative if obtained acutely. Repeated CSF examination 24–72 hours later is usually diagnostic.
- Prompt Rx with IV aciclovir 10 mg/kg tds
- Mortality in HSE is reduced from > 70% to <25 % with aciclovir, and delay in starting treatment is associated with a worse outcome (epilepsy, dementia).

Brain CT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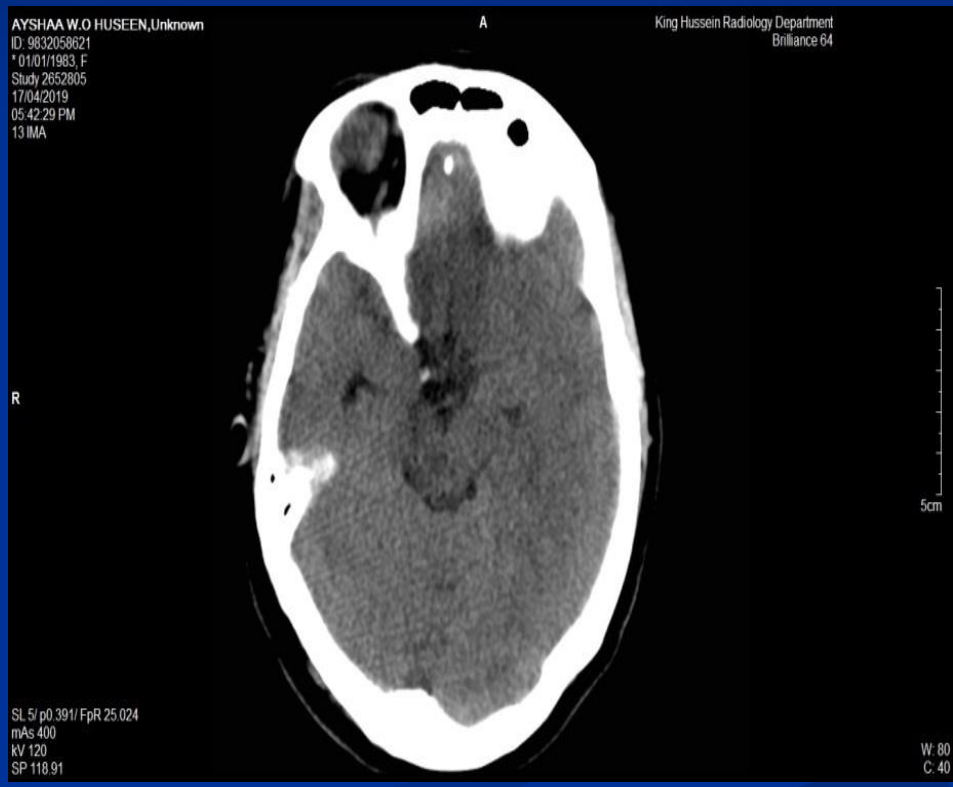
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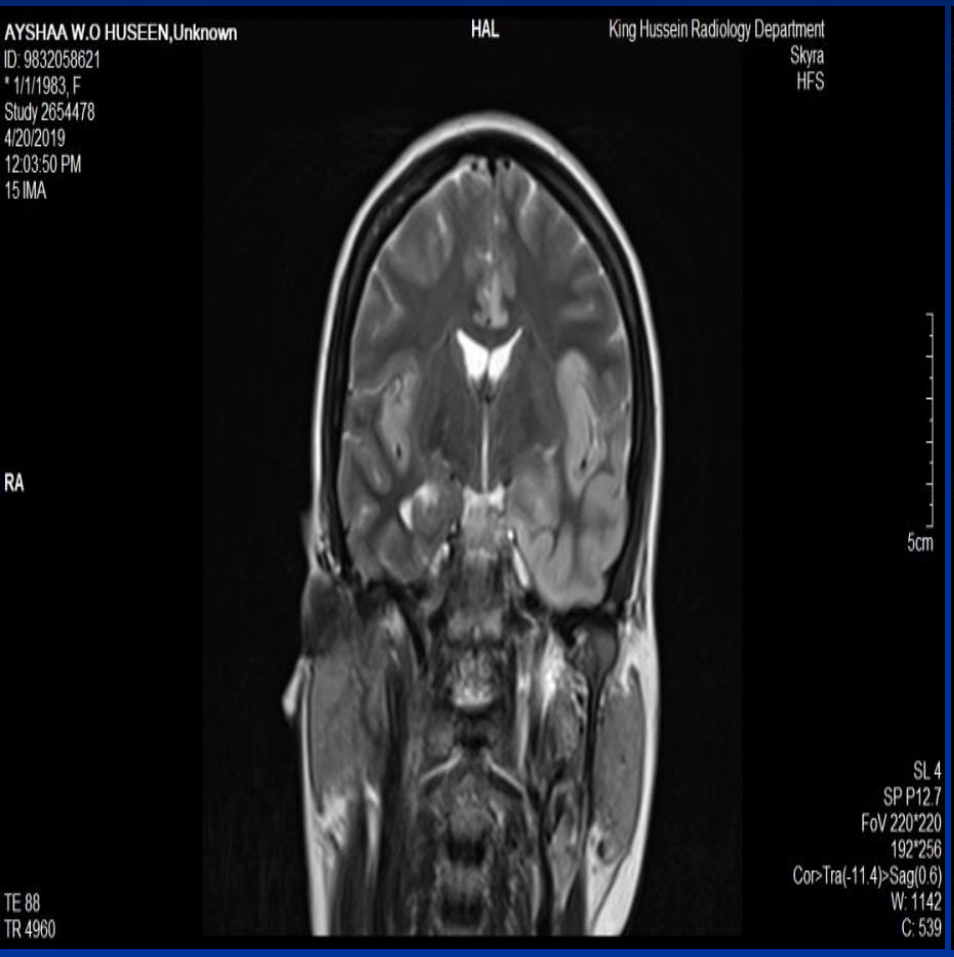
MRI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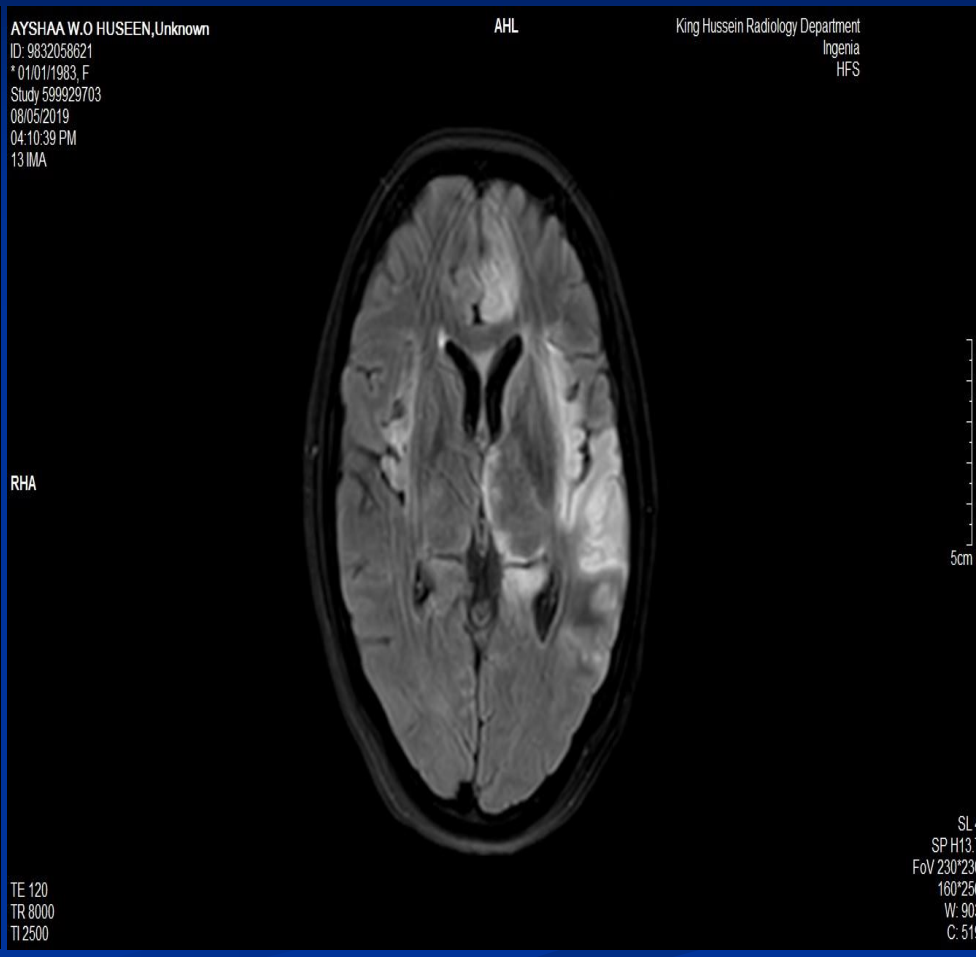
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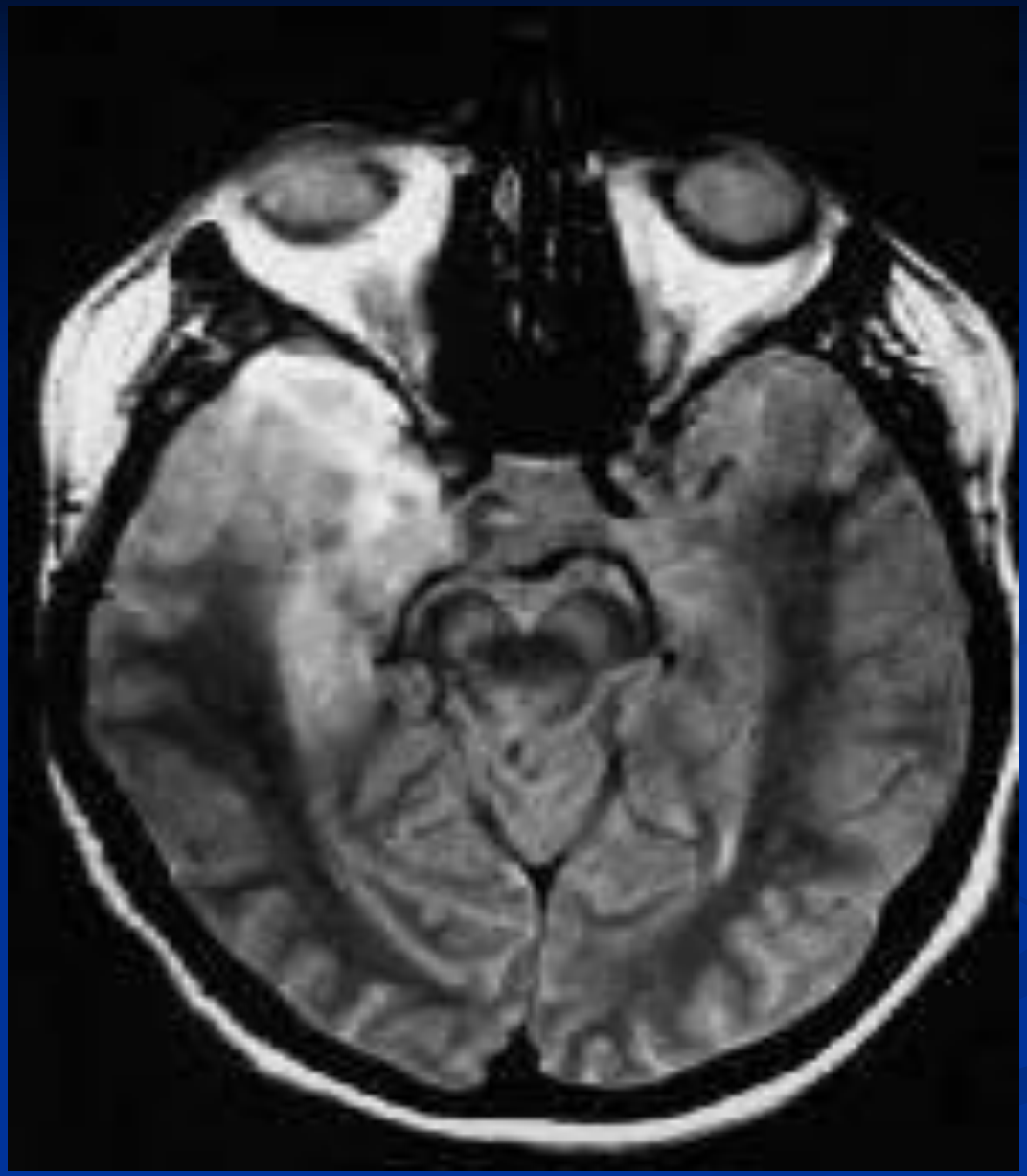
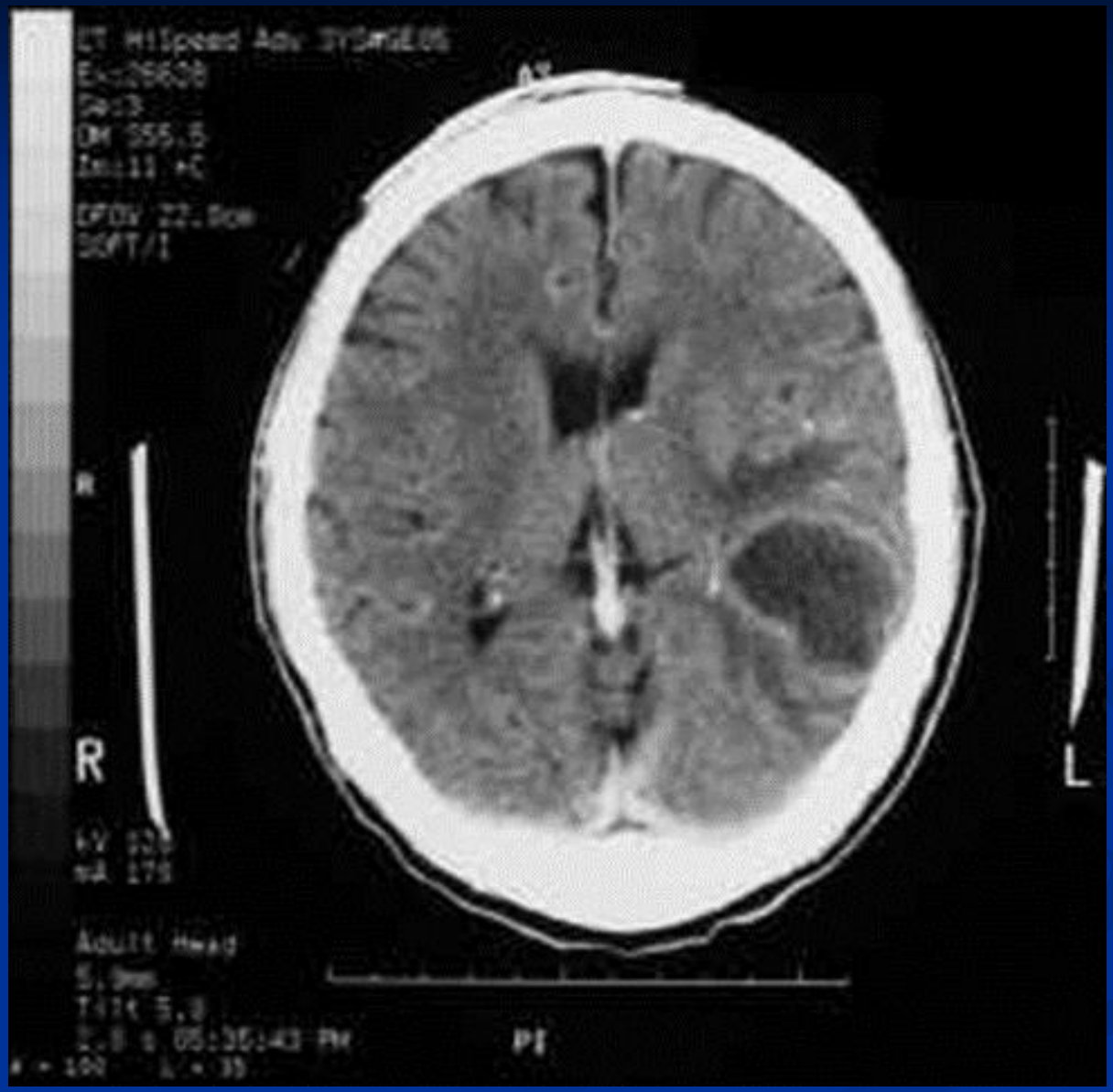



R
L
Adult Head
PI

Brain Abscess

- Predisposing features:
  - Congenital cyanotic heart disease
  - Meningitis
  - Penetrating head injury
  - Local extension from mastod, otitis, sinusitis, soft tissues of face and scalp
- Etiology:
  - S aureus
  - Micro aerophilic strep
  - Other aerobic & anaerobics
  - Mixed infections in 35%
- Clinical Features:
  - Fever
  - Headache
  - Vomiting
  - Focal deficits
  - ↑ICT
- Lab
  - Blood counts non specific
  - EEG: focal slowing
  - CT scan diagnostic
- Treatment:
  - IV antibiotics – cover anaerobes (CP + Chloro)
  - Surgical drainage

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.

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 (1-2/100,000 per year) M:F = 2.5:1.
- The classical features are rapidly progressive, relatively symmetrical tetraparesis of variable severity with mild sensory loss and widespread hypo- or areflexia.
- In some variants, reflexes are retained or brisk.

Clinical Features

- It shows a pattern of "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
- Maximal weakness at 1-2 weeks.
- It can affect the facial muscles (50% of cases) and respiratory muscles, with 25% of patients needing artificial ventilation.

Investigations

LP

Nerve Conduction Studies

- Demyelinating or Axonal
- In first week--- up to 40% normal.

Anti-ganglioside antibodies

- GM1, GD1a, GT1a, GQ1b
- 50 % positive

Ix to exclude mimics– MRI, CPK…etc.

Immunotherapy
IVIg = PE = IVIg+PE

- Treatment of GBS:

  Plasma exchange vs IVIg

  - Overall: No difference in efficacy
  - Indications for treatment: first 2 weeks of disease
    - Bulbar disorders
    - Respiratory dysfunction
    - Inability to walk without assistance
  - Probably indicated: Milder weakness; Early in disease course

Supportive Management

- 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 throughout the acute stages.
- Venous thromboembolism prophylaxis with compression stockings and low molecular weight heparin is recommended for non-ambulant patients

Acute Flaccid Paralysis

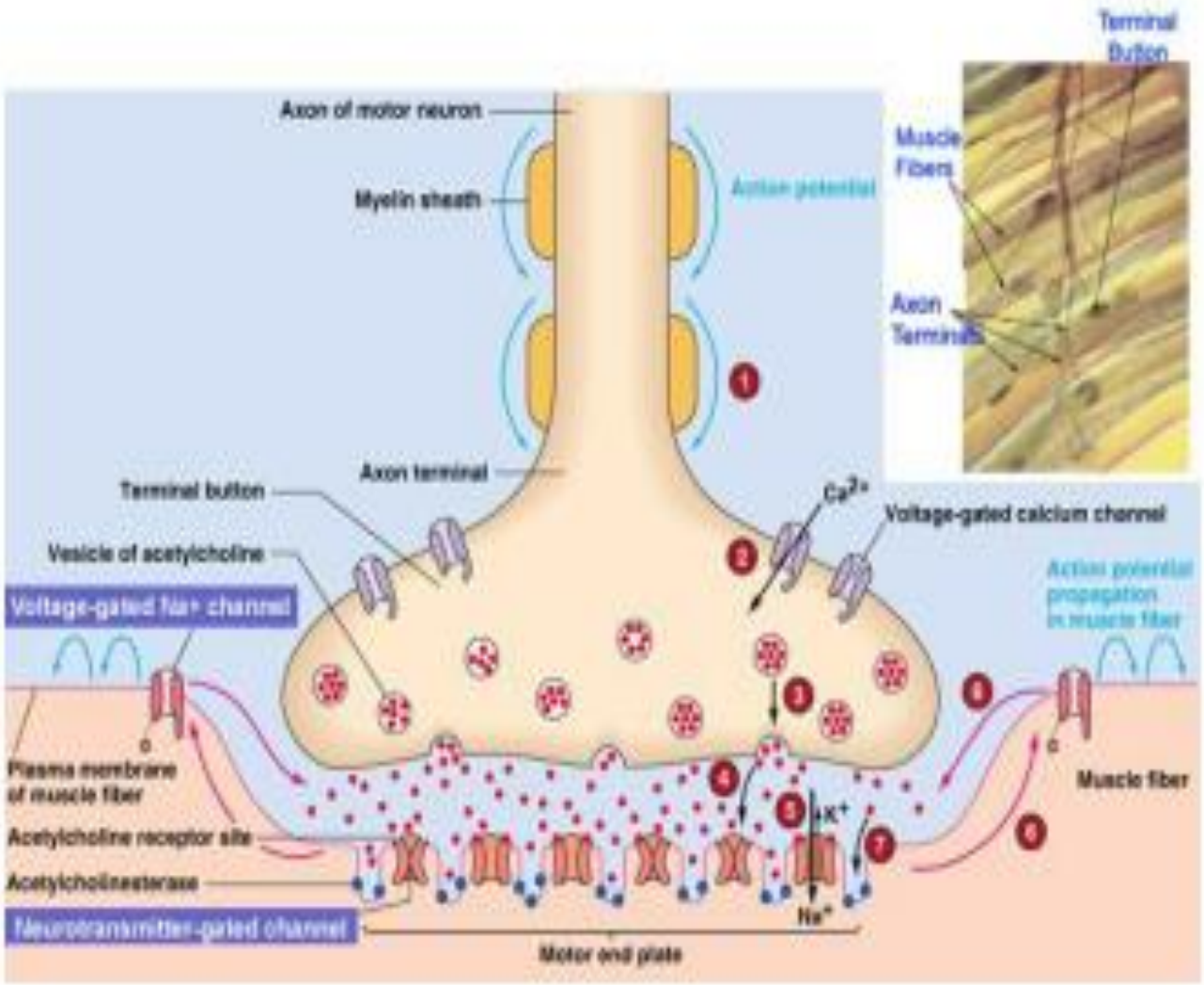
56 yr old male

Previously fit and well

4 week history of

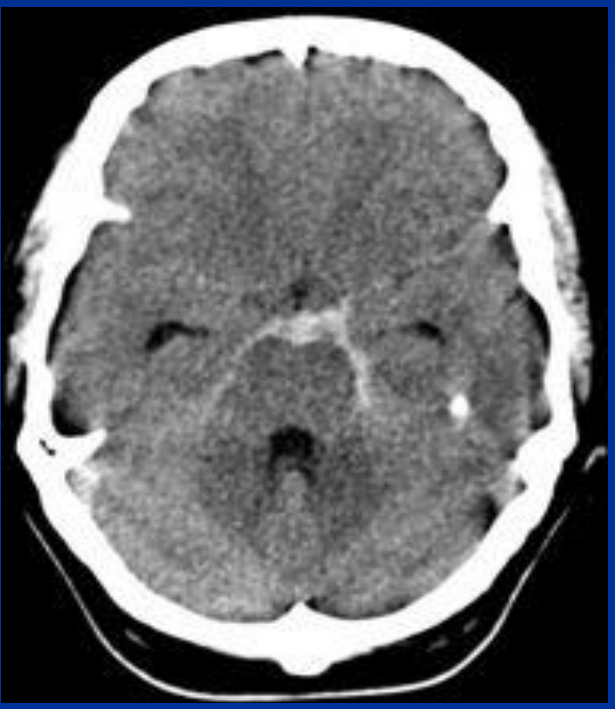
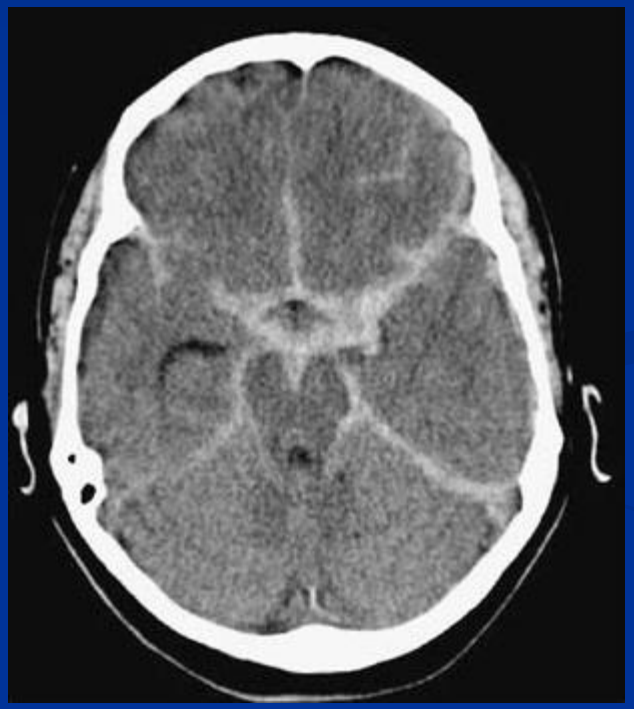
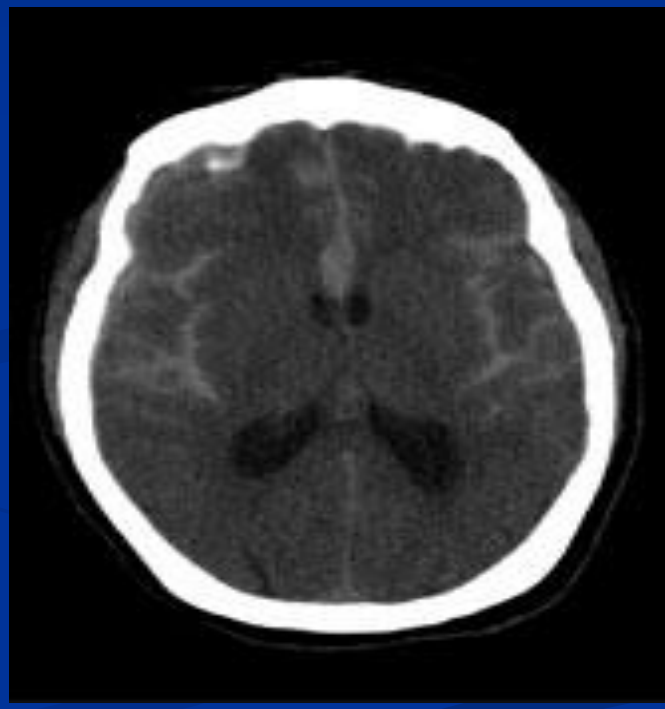
- Diplopia- horizontal
- Variable through the day
- Struggling with drinks- leaking from mouth
- Slurred and low volume speech

Admitted to hospital



	NMJ	Muscle
ro		
	Ocular, bulbar, limb girdle	Symmetric a
	Fatiguable	Limb girdle
ric	Waddle / trendelenberg	trendelenberg
n	No	No

Subarachnoid hemorrhage
Most common cause- rupture of arterial berry aneurysm

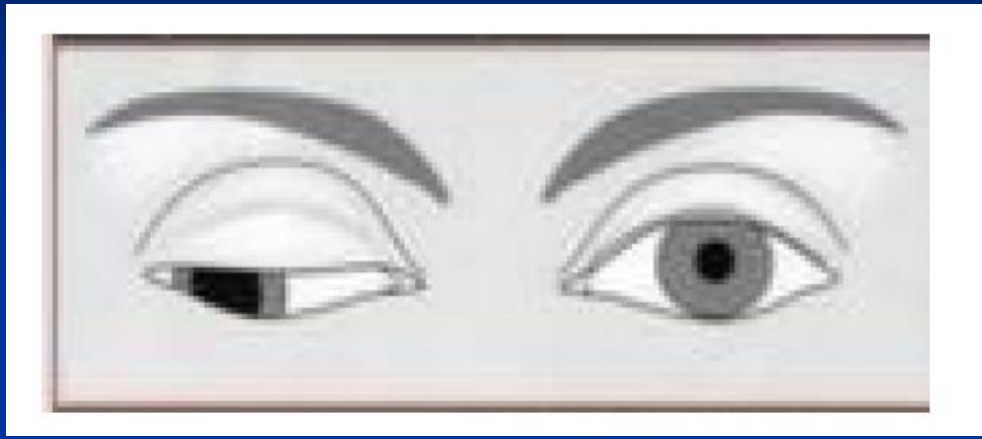




Classic Symptoms of Subarachnoid Hemorrhage

- Sudden, unusually severe or "thunderclap" headache
- Loss of consciousness
- Pain in neck, back, eye or face
- 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 – Posterior communicating artery aneurysm
  - Paraparesis – ACA aneurysm
  - Hemiparesis, aphasia – MCA aneurysm
- Ocular hemorrhages



Subarachnoid Hemorrhage

- Warning leaks in 50%
- CT misses up to 10% small leaks
- Suspect if:
  - > 35 years
  - no previous HA
  - no fading of HA
  - came on with exertion
  - altered LOC or neuro deficits
  - stiff neck

"Thunderclap" Headache

- 25% associated with SAH
- "Warning" headache
  - followed by SAH in 5% to 60%
- Expansion or dissection of unruptured aneurysm
- Cerebral venous thrombosis
- Exertional / coital headache

Status Epilepticus

Definition of SE

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

Status Epilepticus

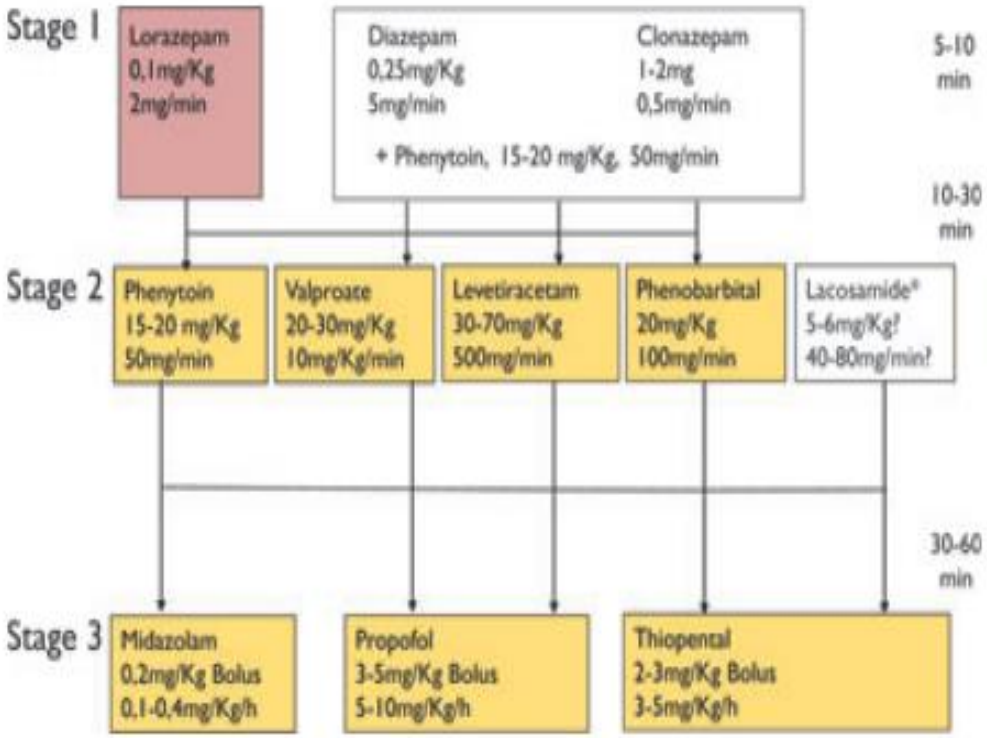


Figure 5.

Staged approach to the treatment of convulsive status epilepticus. *There is currently limited evidence for the use of lacosamide in SE (see Höfler et al., 2011) Modified after Trinka, 2007; Shorvon et al., 2008.

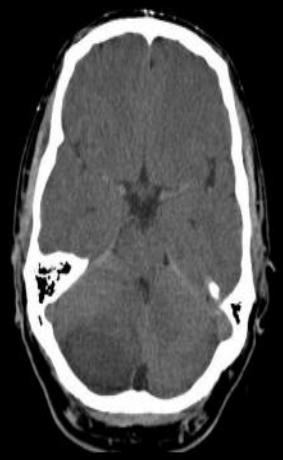
Epilepsia © ILAE

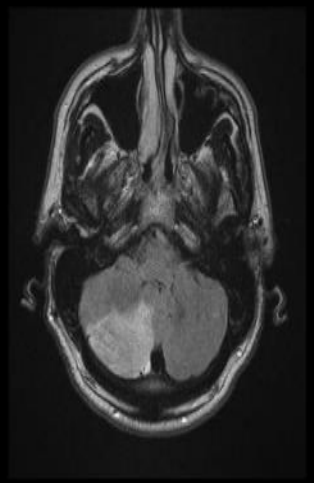
Dizziness/vertigo

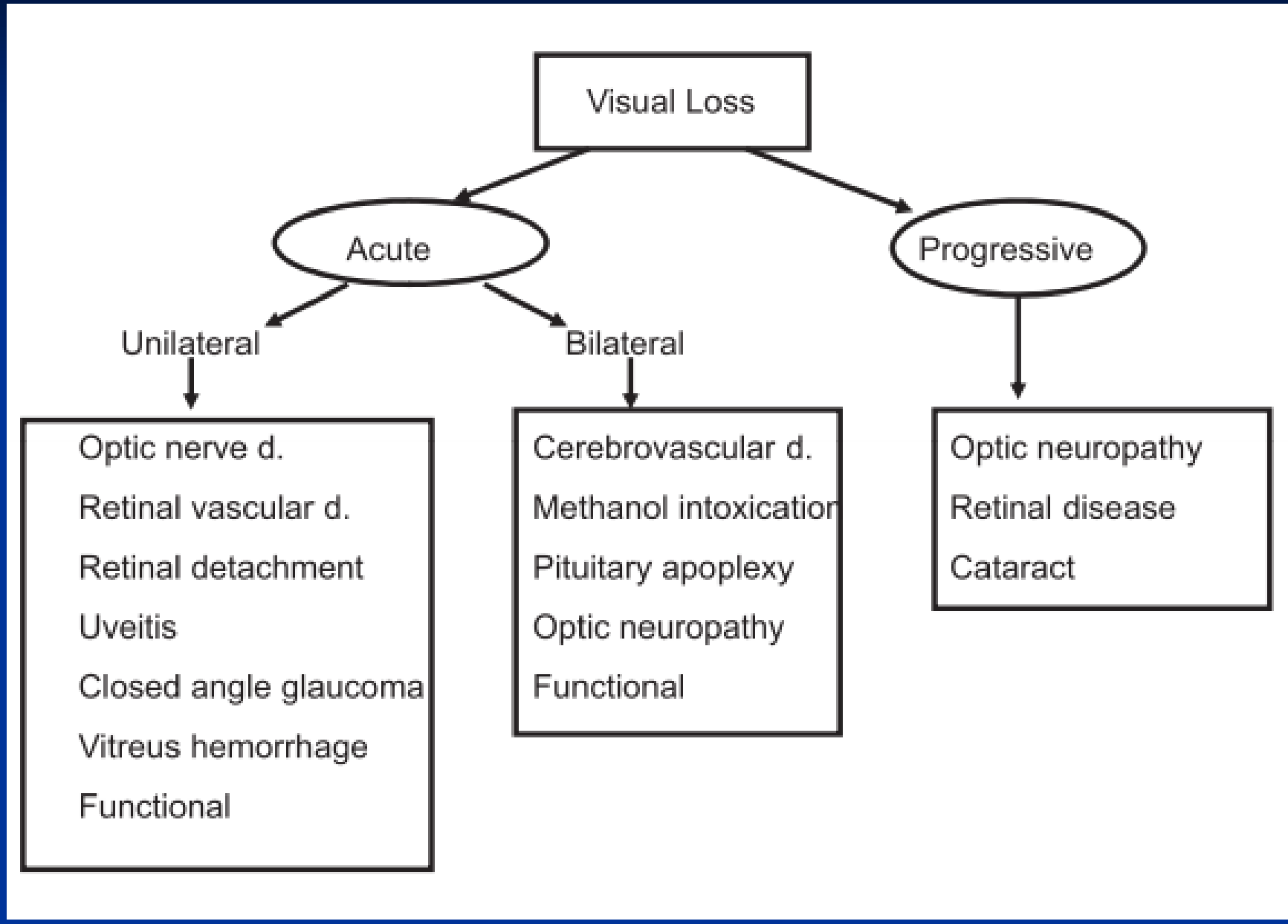
- Acute vertigo middle age man
- Headache – Red flag!
- Normal head thrust – Red flag!!

Brain scan needed in acute vertigo:

- Intact head impulse test
- New onset (occipital) headache
- Any central symptoms or signs
- Acute deafness

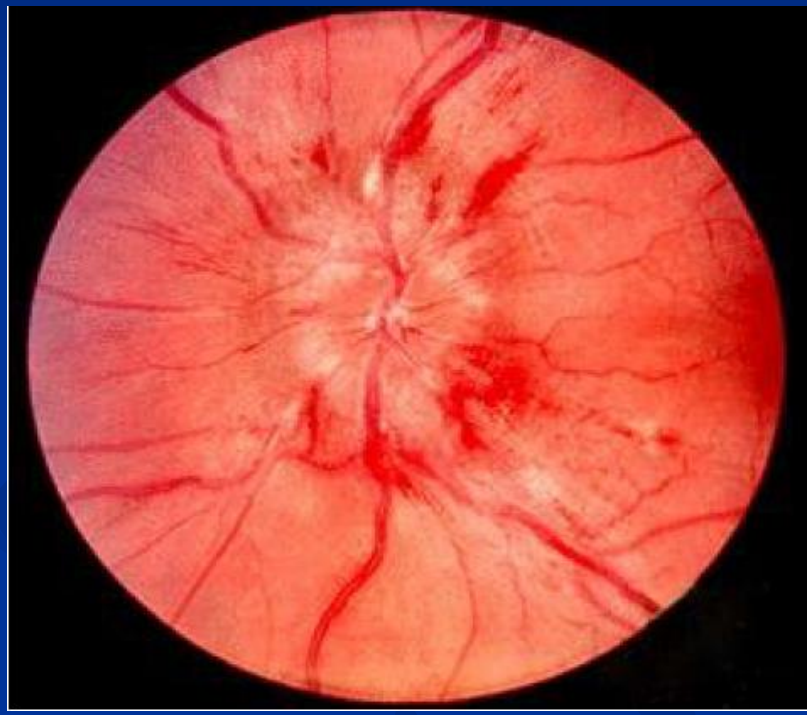




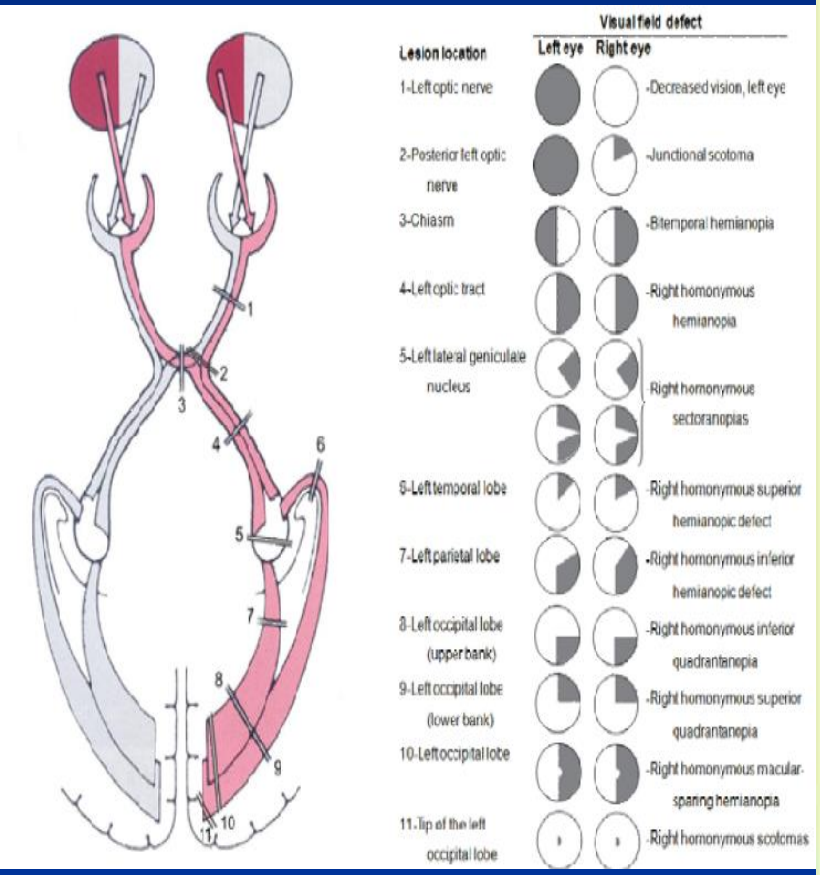

Visual Loss
Acute
Progressive
Unilateral
Bilateral
Optic nerve d.
Retinal vascular d.
Retinal detachment
Uveitis
Closed angle glaucoma
Vitreus hemorrhage
Functional
Cerebrovascular d.
Methanol intoxication
Pituitary apoplexy
Optic neuropathy
Functional
Optic neuropathy
Retinal disease
Cataract

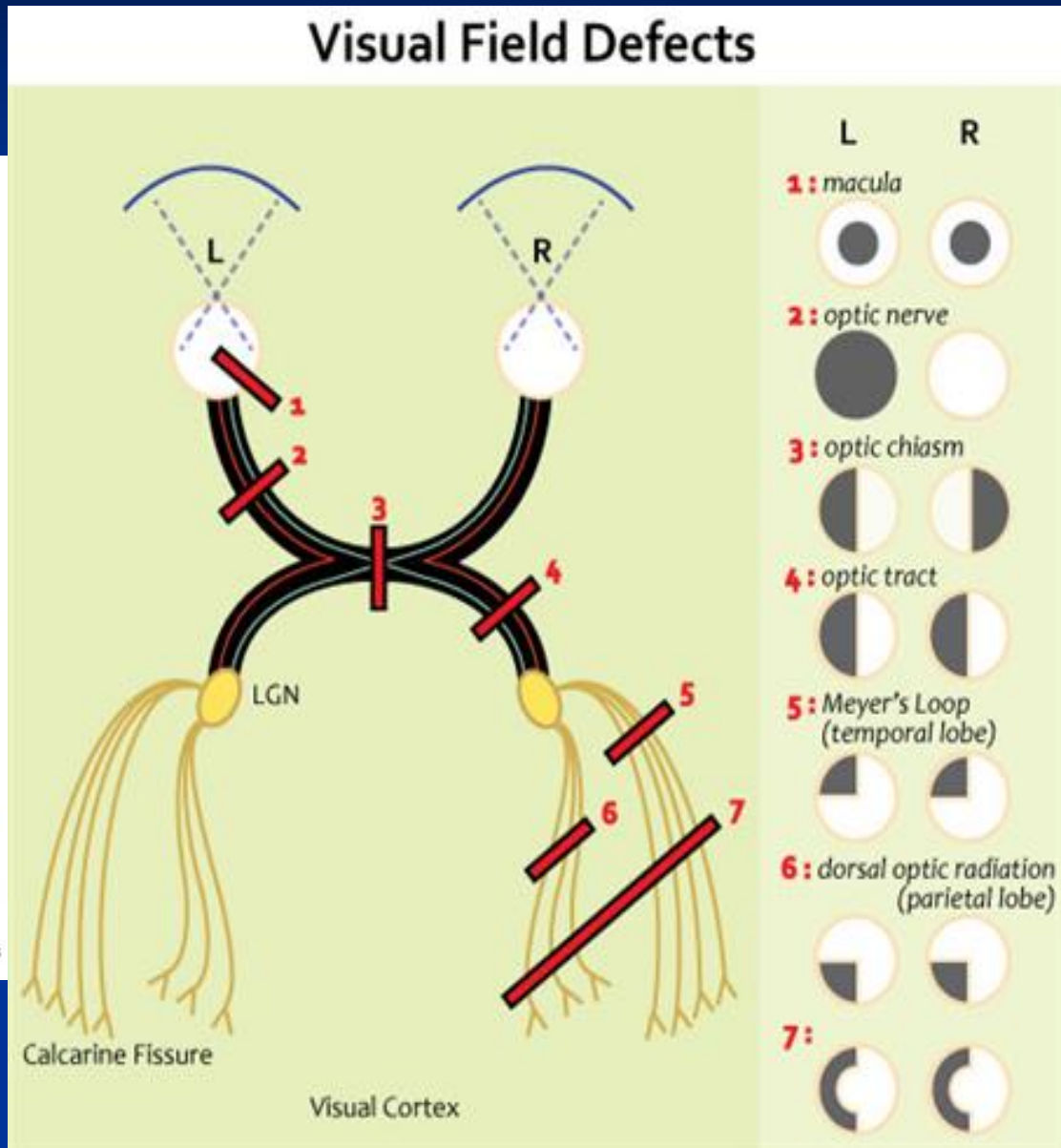
Neurological causes of sudden loss of vision

- Optic neuropathy (Demyelination , Ischemia/vasculitis)
- Papilledema/high ICP (Tumor, Idiopathic Intracranial Hypertension)
- Bilateral occipital lobe pathology (infarcts, Posterior Reversible Encephalopathy Syndrome)
- Functional/psychogenic



Anatomy of Visual Pathways





- Register your attendance with your university number
- Make sure that the settings of your phone allow tracking location

Go to settings > privacy> location> services> make sure that location services is ON