Neurological Emergencies/ CNS Infections

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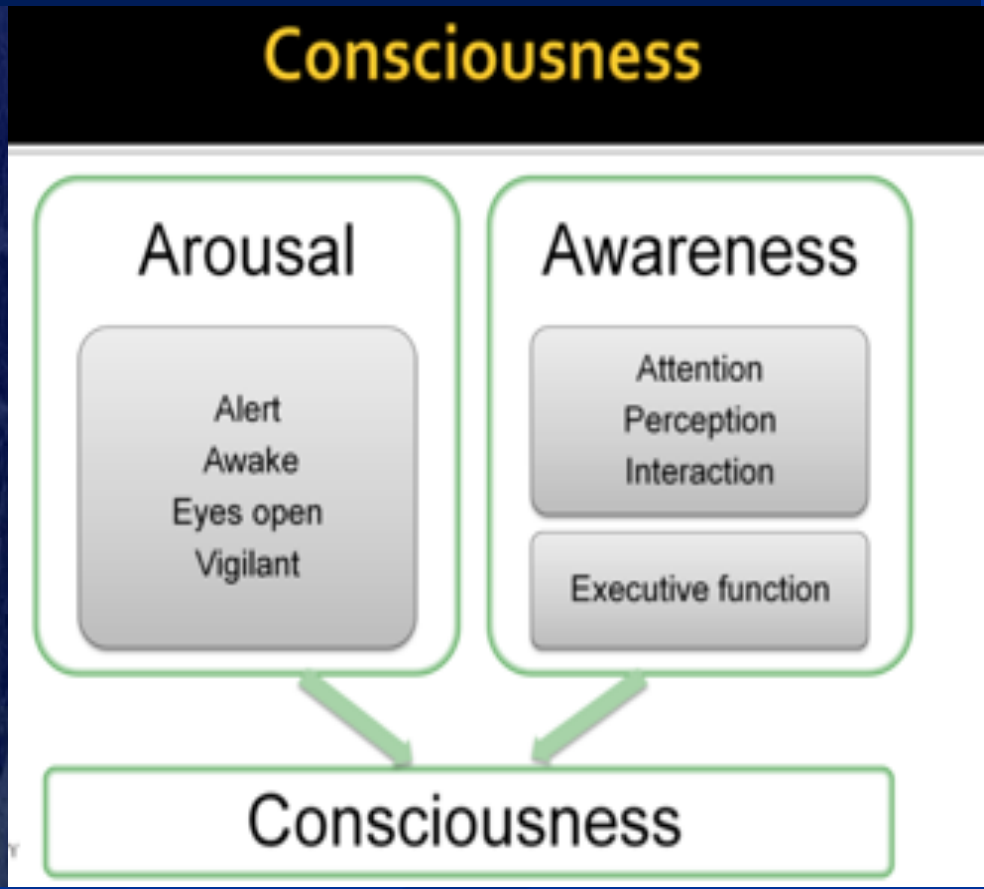
Neurological Emergencies-At least 20% of Medical ER visits

- 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
- Visual problems
- Vertigo

Components of Consciousness

Components of Consciousness

- Arousal → Level of Consciousness
- Awareness → Content of Consciousness

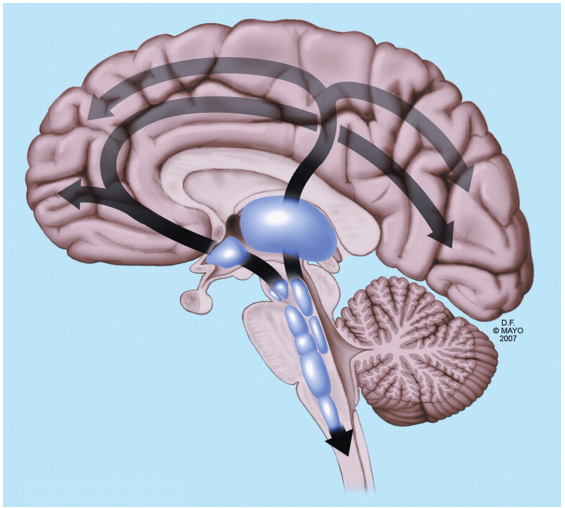


Anatomy of consciousness

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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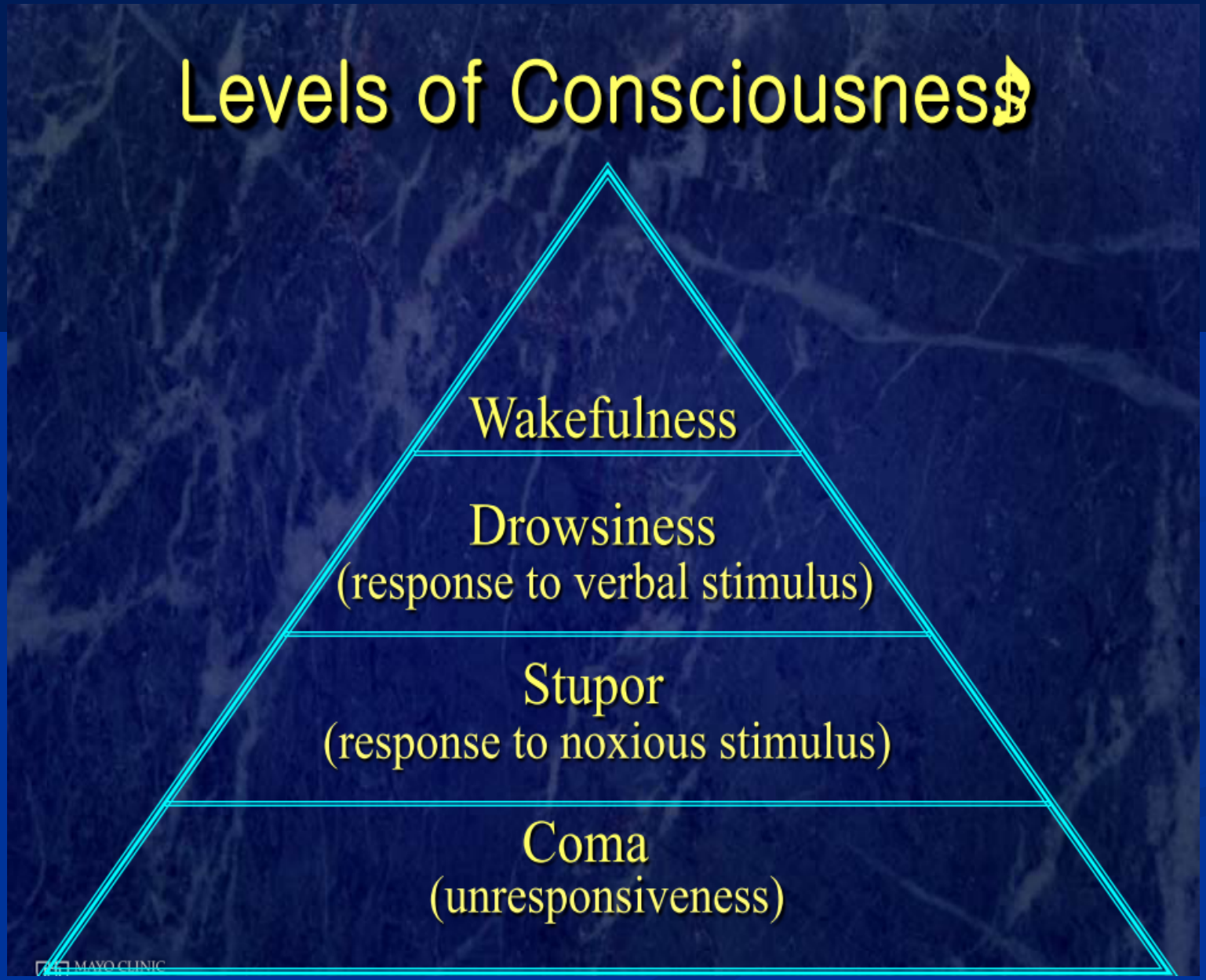
Content of consciousness Terminology

- 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

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

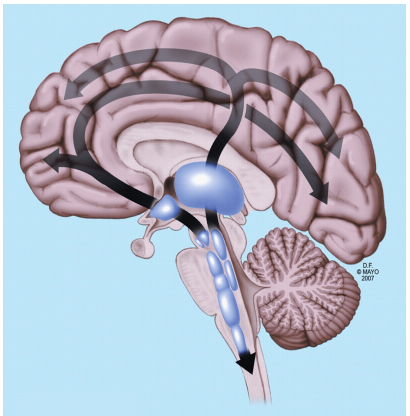
- Subarachnoid hemorrhage
- Traumatic brain injury
- Multiple cerebral infarcts
- Bilateral thalamic infarcts
- Tumors
- Encephalitis
- Cerebral edema
- Acute hydrocephalus
- Posterior reversible encephalopathy syndrome (PRES)
- Air or fat embolism.

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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Acute metabolic/endocrine derangement

- Hypoglycemia (<40 mg/dl)
- Hyperglycemia (non-ketotic hyperosmolar) >900 mg/dl
- Hyponatremia <110
- Hypernatremia >160
- Addison's disease
- Hypercalcemia> 3.4 mmol/L
- Acute hypothyroidism
- Acute panhypopituitarism
- Acute uremia
- Hyperbilirubinemia
- Hypercapnia >9 kPa

Diffuse physiological brain dysfunction

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

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

Psychogenic swoon/**unresponsiveness**

A neurology grand round on Tuesday

Une lecon du mardi.

The famous painting of Jean-Martin Charcot demonstrating a case of hysteria at the Salpetriere Hospital in Paris in 1887.

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

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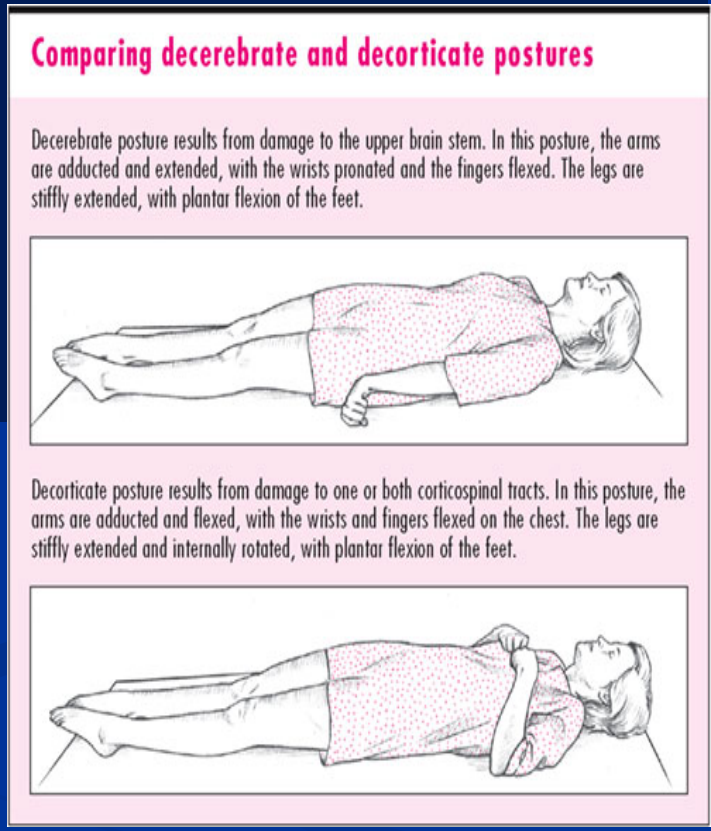


Record subsets:

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

A score of < 8 …. coma

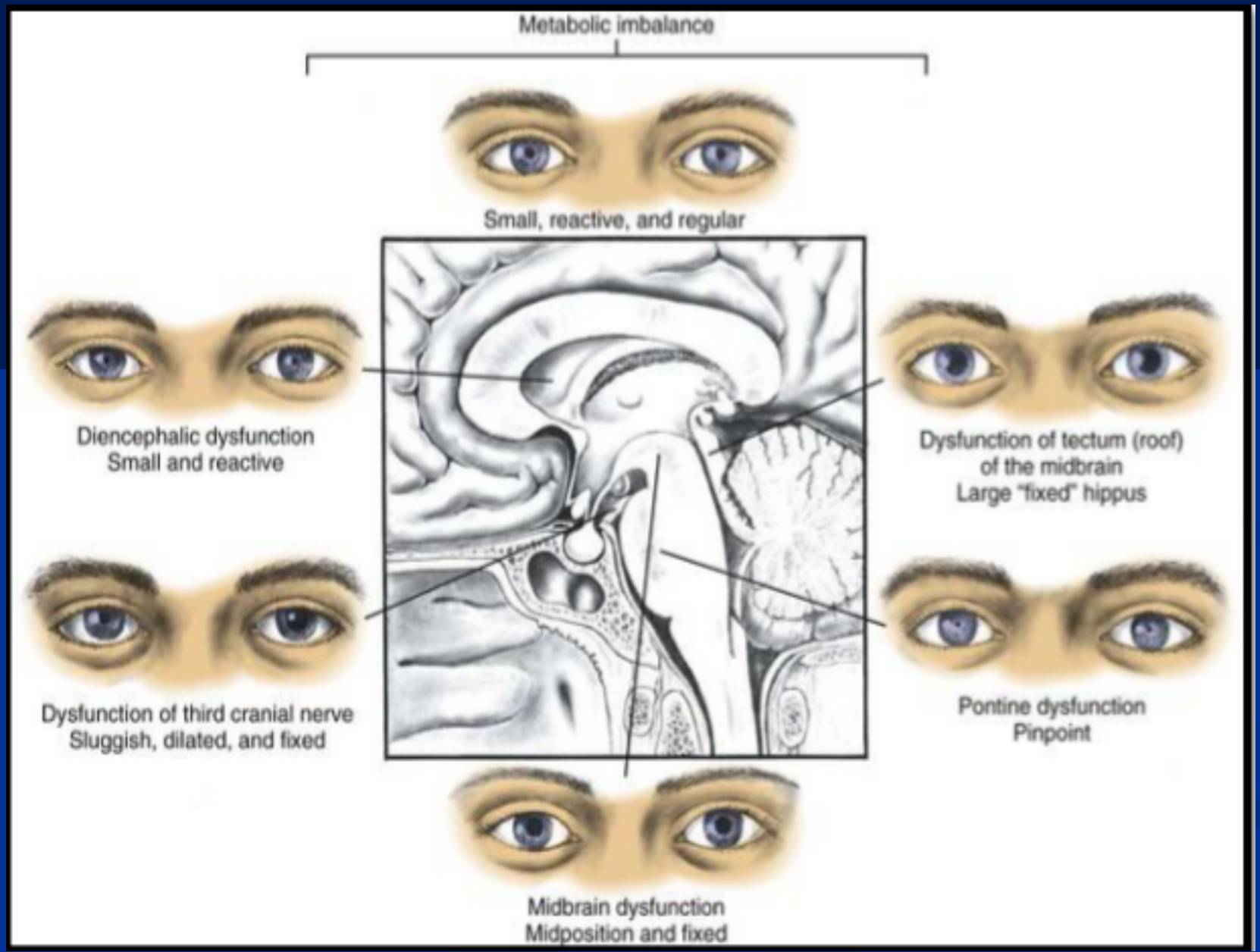
Verbal response can be compromised by endotracheal intubation …. V(T) should recorded.

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.

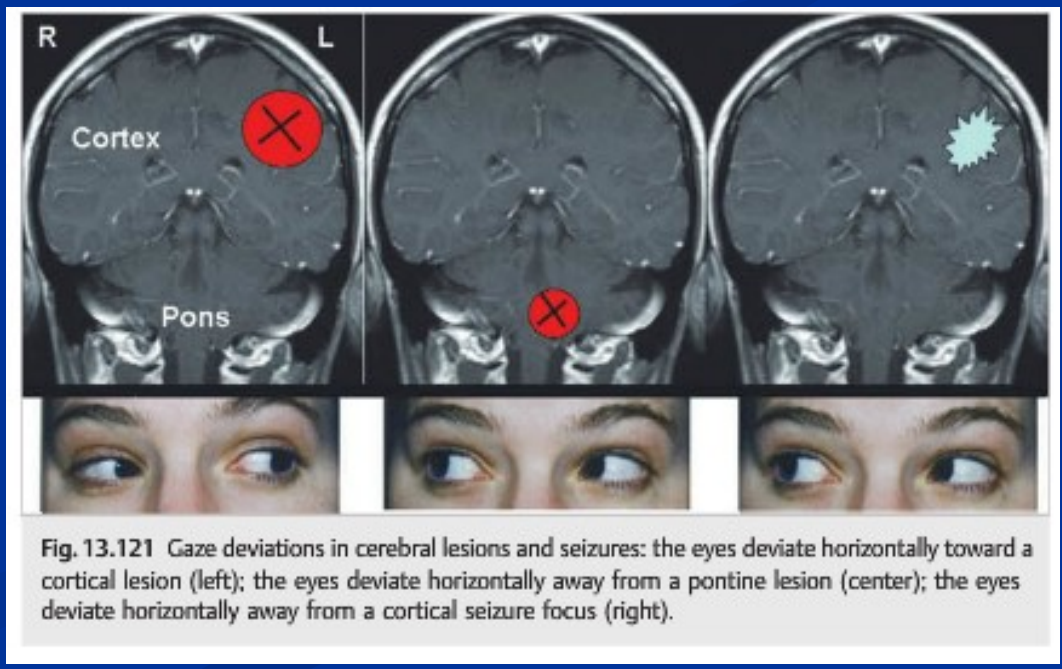


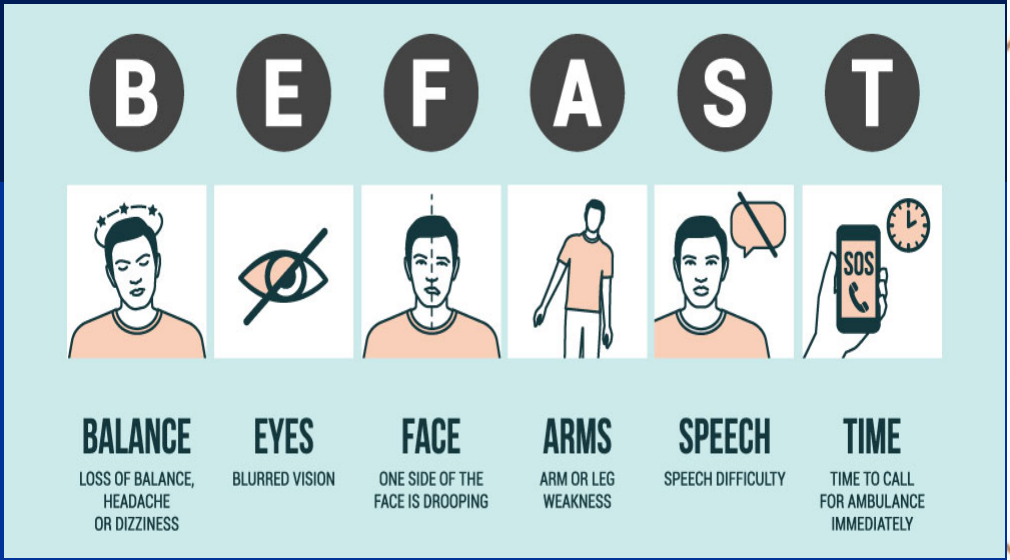
Fig. 13.121 Gaze deviations in cerebral lesions and seizures: the eyes deviate horizontally toward a cortical lesion (left); the eyes deviate horizontally away from a pontine lesion (center); the eyes deviate horizontally away from a cortical seizure focus (right).

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.

Stroke/TIA -20 % of patients with ischemic stroke have a preceding TIA or minor stroke.



ABCD2 SCORE FOR TIA

THE RISK OF STROKE IN TRANSIENT ISCHEMIC ATTACK

Age	1 point: Age > 60 years
BP	1 point: BP ≥ 140/90
Clinical Feature	1 point: Speech disturb. without weakness 2 points: Unilateral weakness
Duration	1 point: 10-59 minutes 2 points: ≥ 60 minutes
Diabetes	1 point

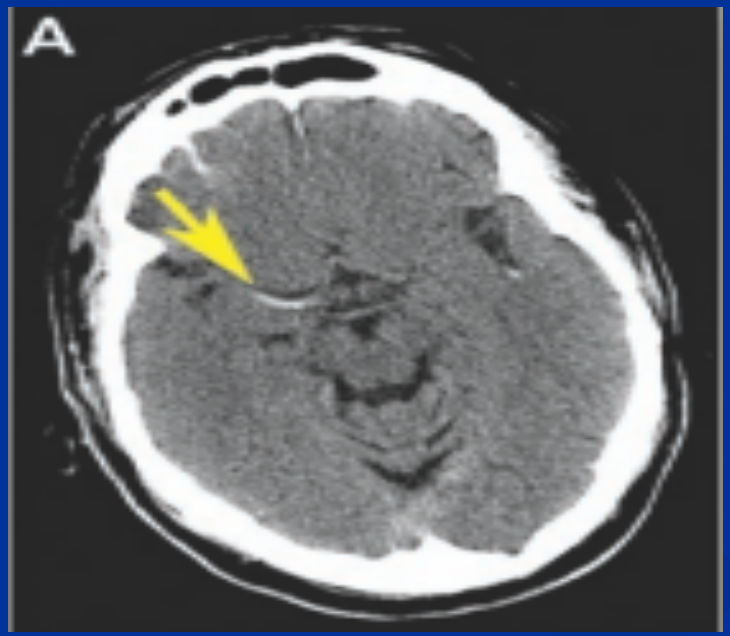
Score	Stroke Risk		
	Day 2	Day 7	Day 90
0-3: Low	1%	1.2%	3.1%
4-5: Mod	4.1%	5.9%	9.8%
6-7: High	8.1%	11.7%	17.8%

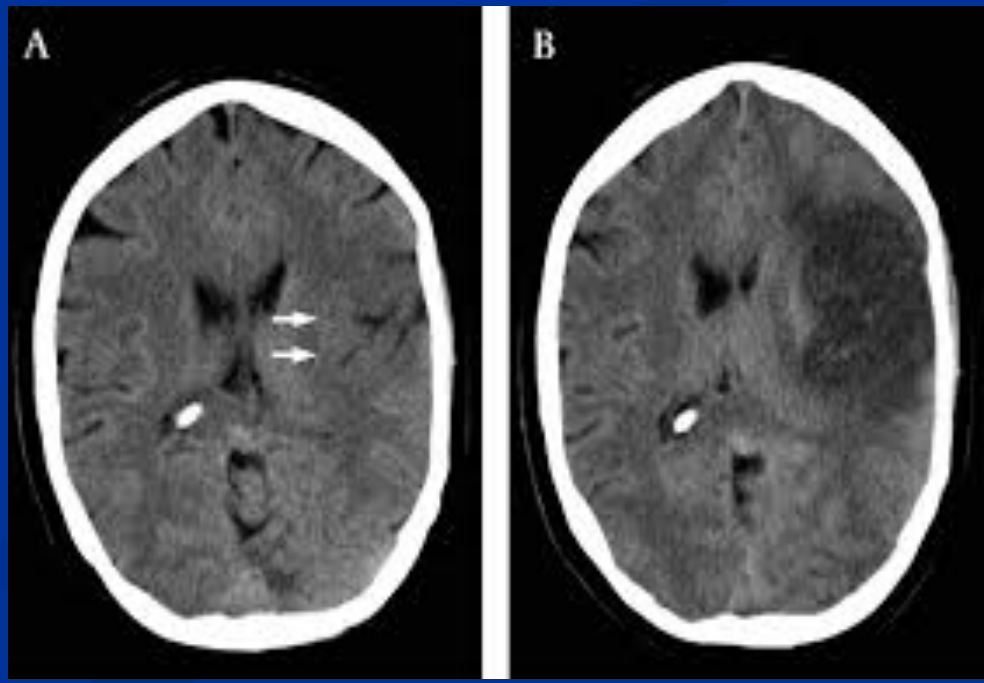
The following are symptoms are unlikely to be due to a TIA- consider other causes

- Altered consciousness or syncope
- Dizziness, wooziness, or giddiness
- Impaired vision ("grey out") with alteration of consciousness
- Amnesia or confusion alone
- Tonic and/or clonic motor activity
- Purely sensory symptoms, especially if they are positive symptoms (paraesthesia = tingling, 'pins and needles', electric shock')
- Sensory march (progressive spread of symptoms, e.g. from hand to arm to face over seconds or minutes)
- Focal positive neurological symptoms associated with migraine; (e.g. scintillating scotomata)
- Bowel or bladder incontinence
- Vertigo, diplopia, dysphagia, or dysarthria that occurs in isolation

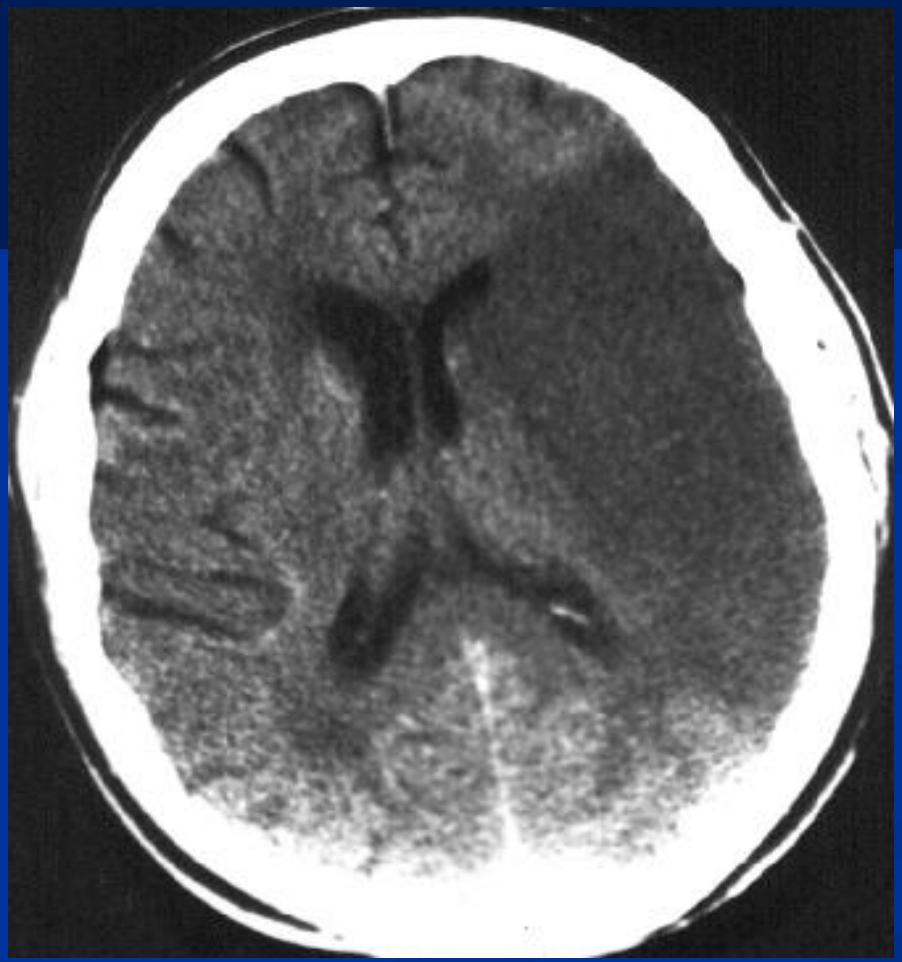
Brain CT in acute ischemic stroke

- Only around 30% of stroke patients will have signs of early ischemic damage in the first 3 hours after symptom onset.
- Early ischemic stroke or TIA does not affect level of consciousness

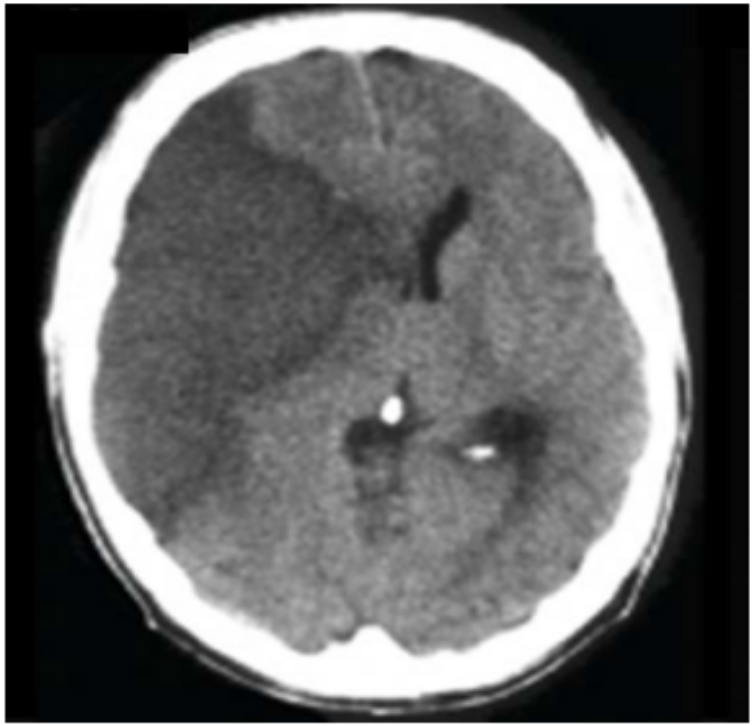




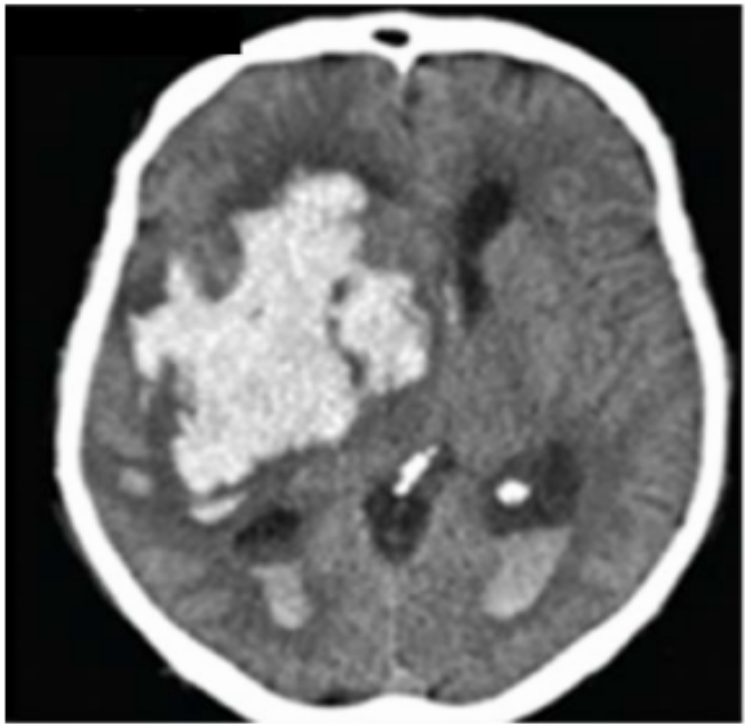
Left Middle Cerebral Artery Infarction with minimal mass effect – level of consciousness probably normal.



These patients will probably have decreased level of consciousness

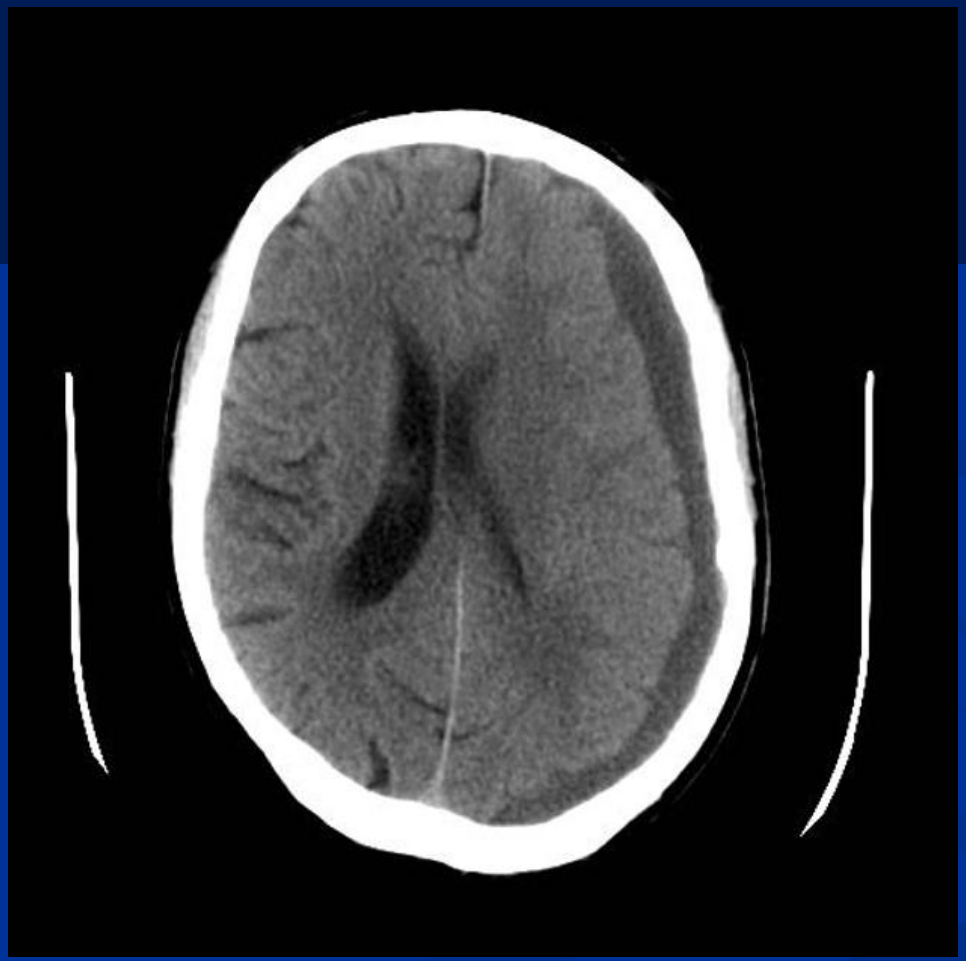


Ischemic Stroke
(dark/hypodense)

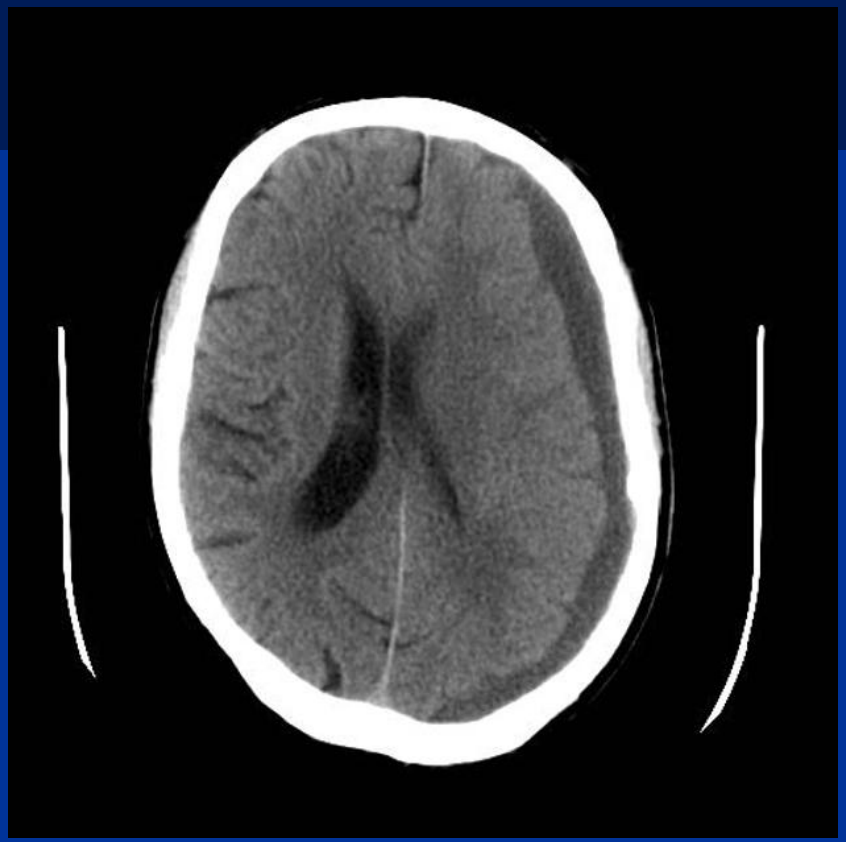


Hemorrhagic Stroke
(bright/hyperdense)

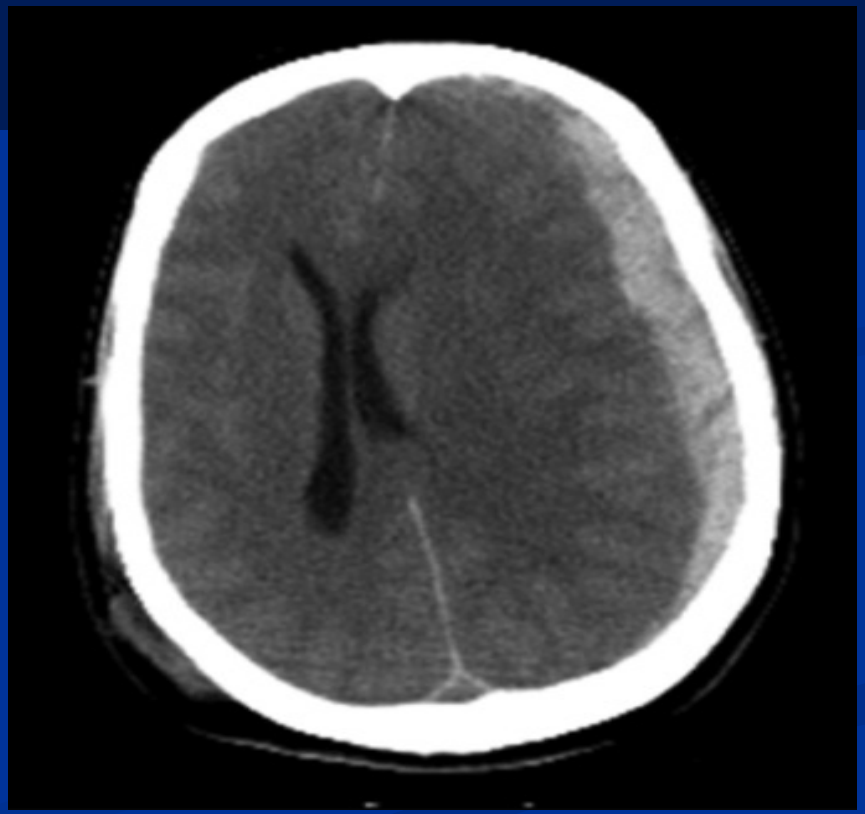
What is shown on this CT ?



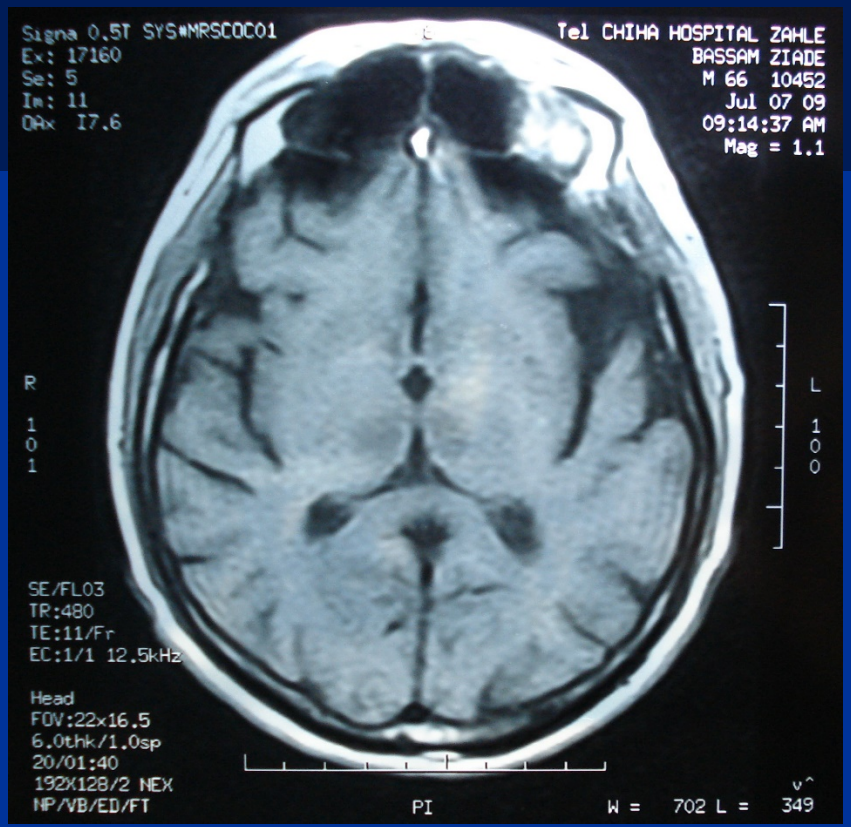
Chronic Subdural Hematoma

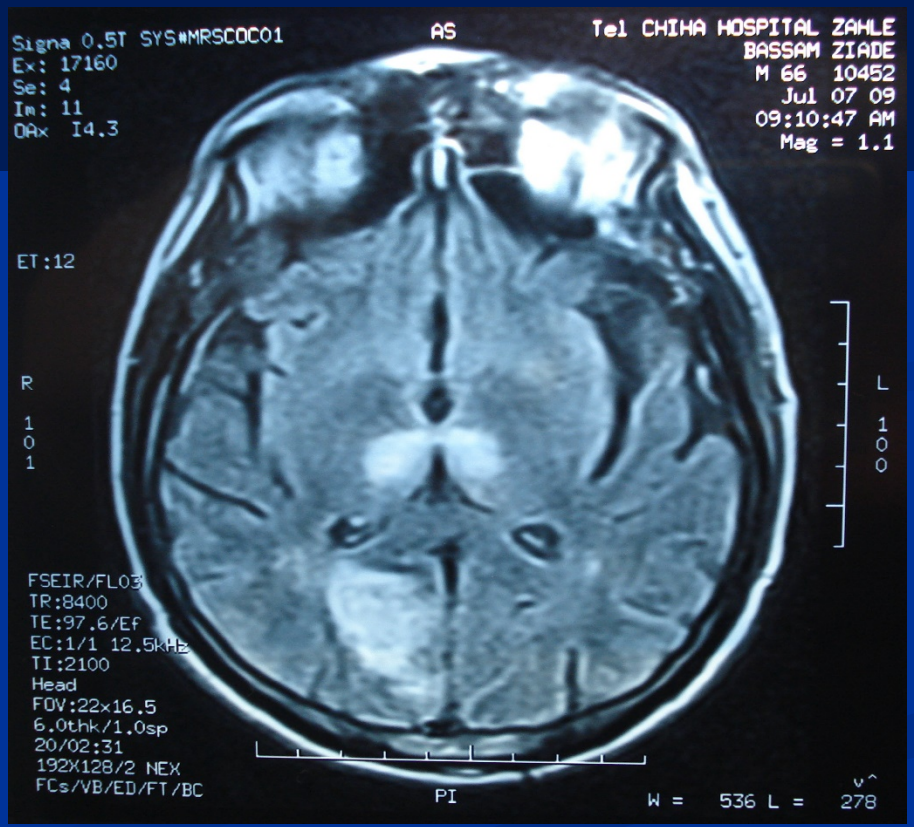


Acute Subdural Hematoma



MRI showing bilateral thalamic infarctions causing coma





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 ?

- 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

Acute bacterial Meningitis (ABM)

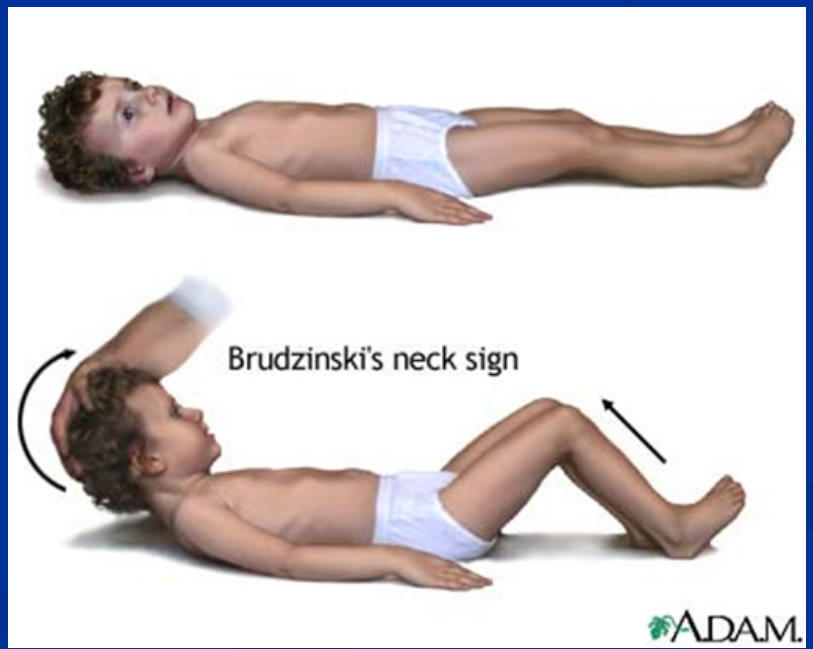
- Common & serious
- Medical emergency
- 100% curable if treated adequately or 100% fatal
- High index of suspicion important
- Dx by CSF examination

ABM: 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

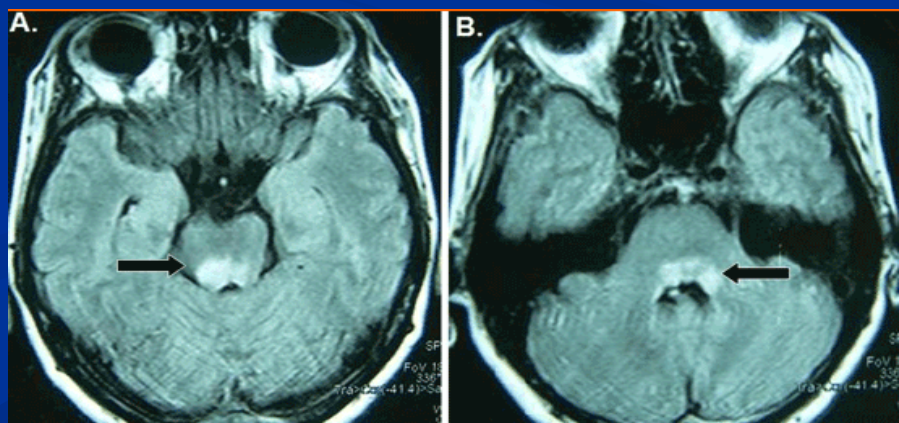


ABM : Etiology

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
  - LP deferred if there is contraindication (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 ABM

- 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.

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.

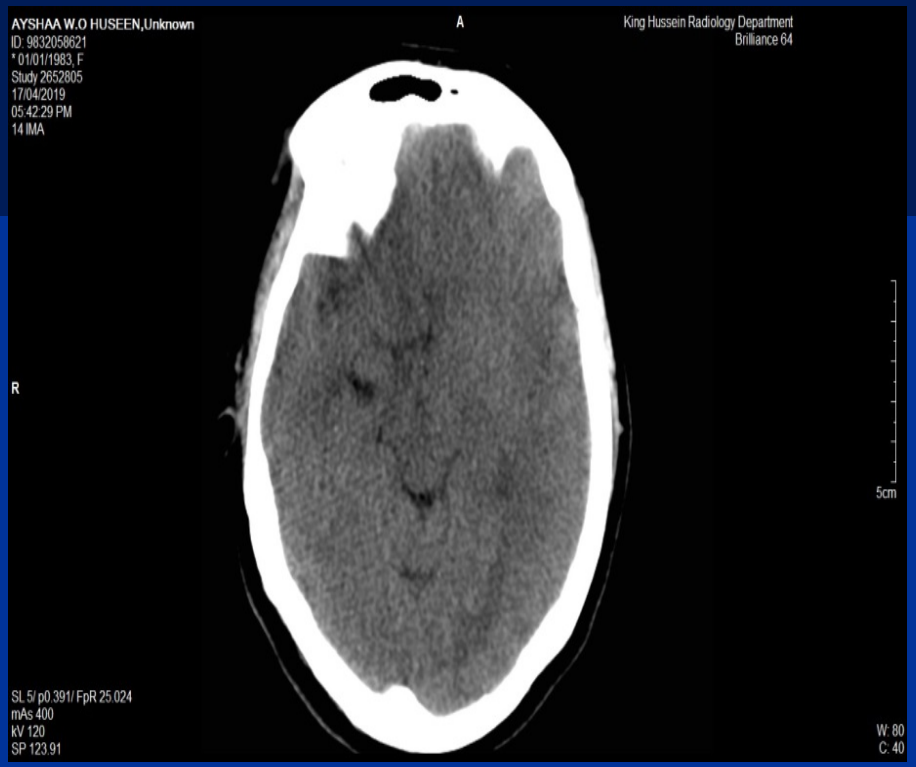
Herpes simplex virus (HSV) encephalitis

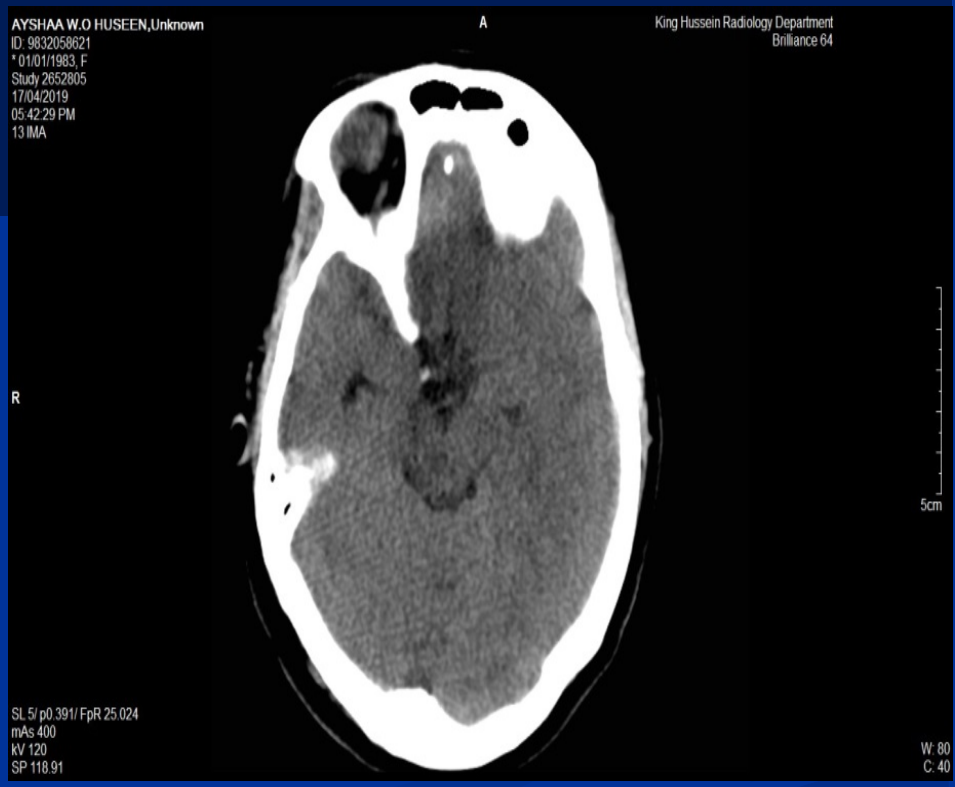
- Encephalitis may be infectious or autoimmune
- Most common cause of sporadic infectious encephalitis is HSV1. Also HSV2 and VZV.
- Many other viruses and bacteria

HSV Encephalitis

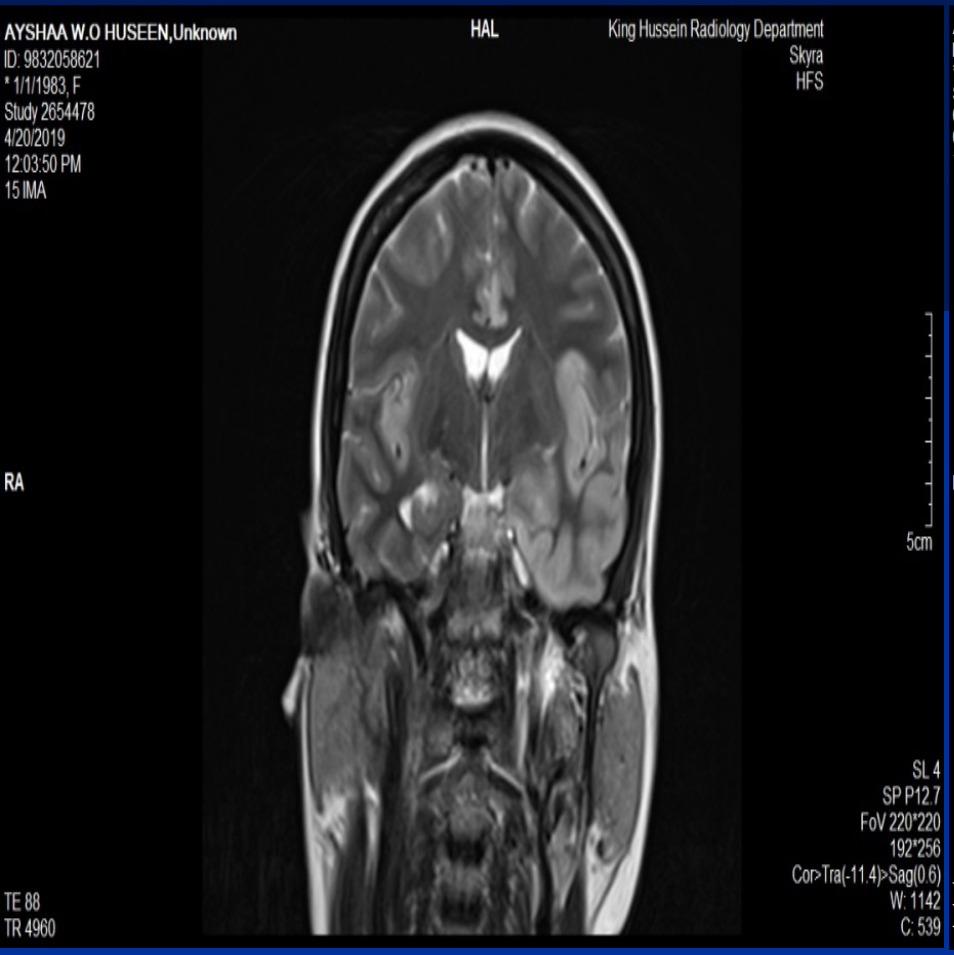
- The most distinctive presenting features are fever, disorientation, aphasia and behavioural disturbances, and up to a third of patients have convulsive seizures.
- MRI shows markedly asymmetric but usually bilateral abnormalities in the limbic system, medial temporal lobes, insular cortices and inferolateral frontal lobes
- CSF clear, pleocytosis, normal or ↑protein, normal sugar , PCR
- Prompt Rx with IV aciclovir 10 mg/kg x3 if any suspicion

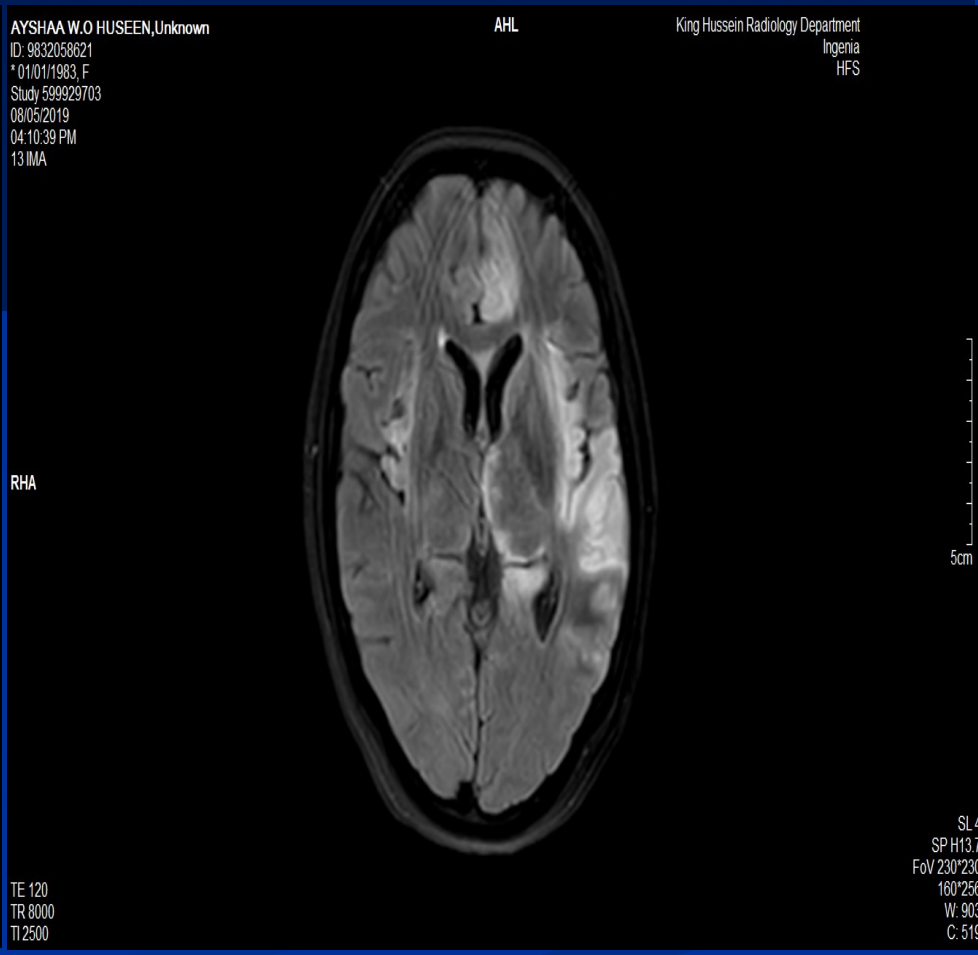
Brain CT in a case of HSV encephalitis

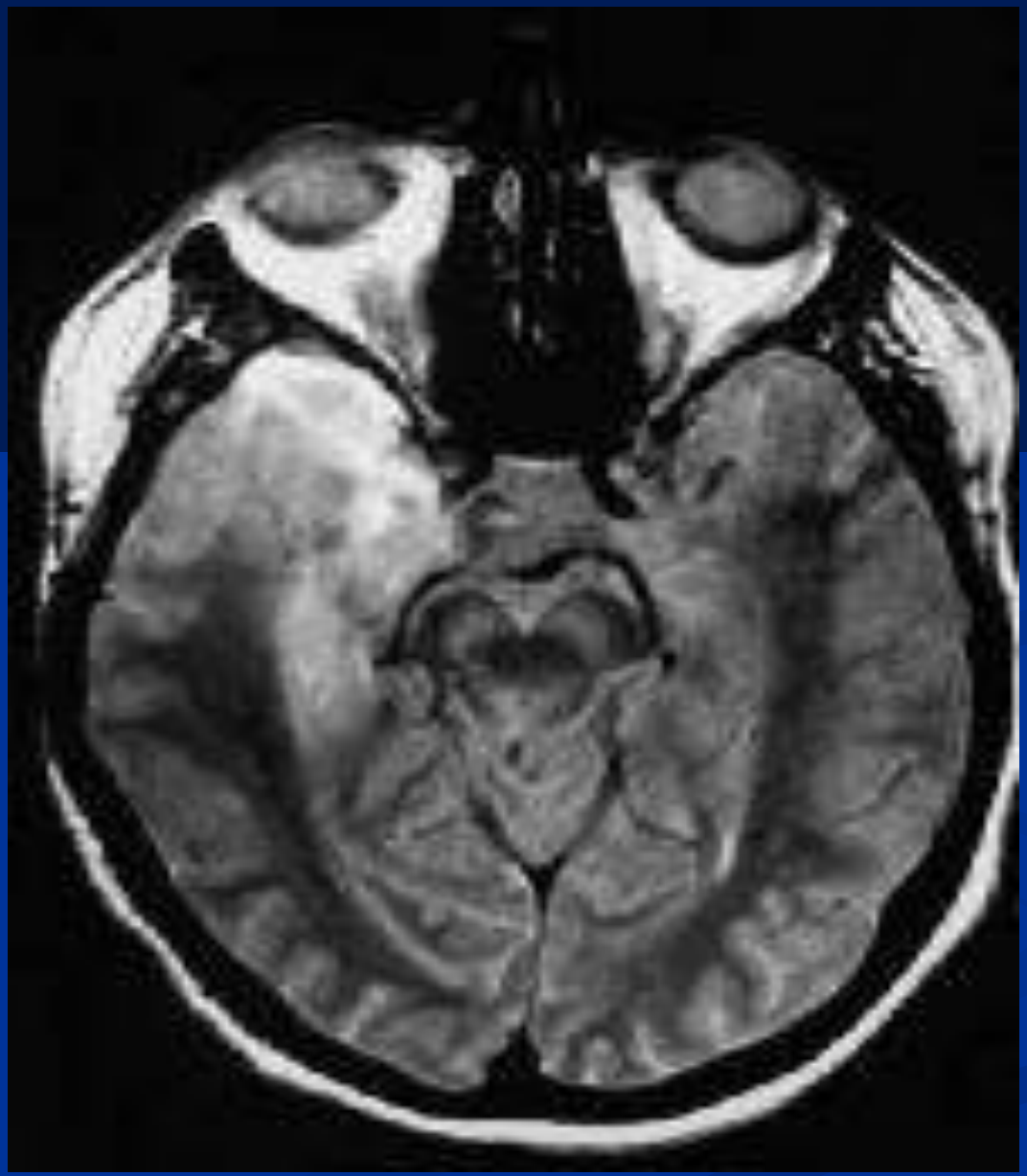




MRI in previous patient.








R
L
Adult Head
PI

Brain abscess with displacement and hydrocephalus

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

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.

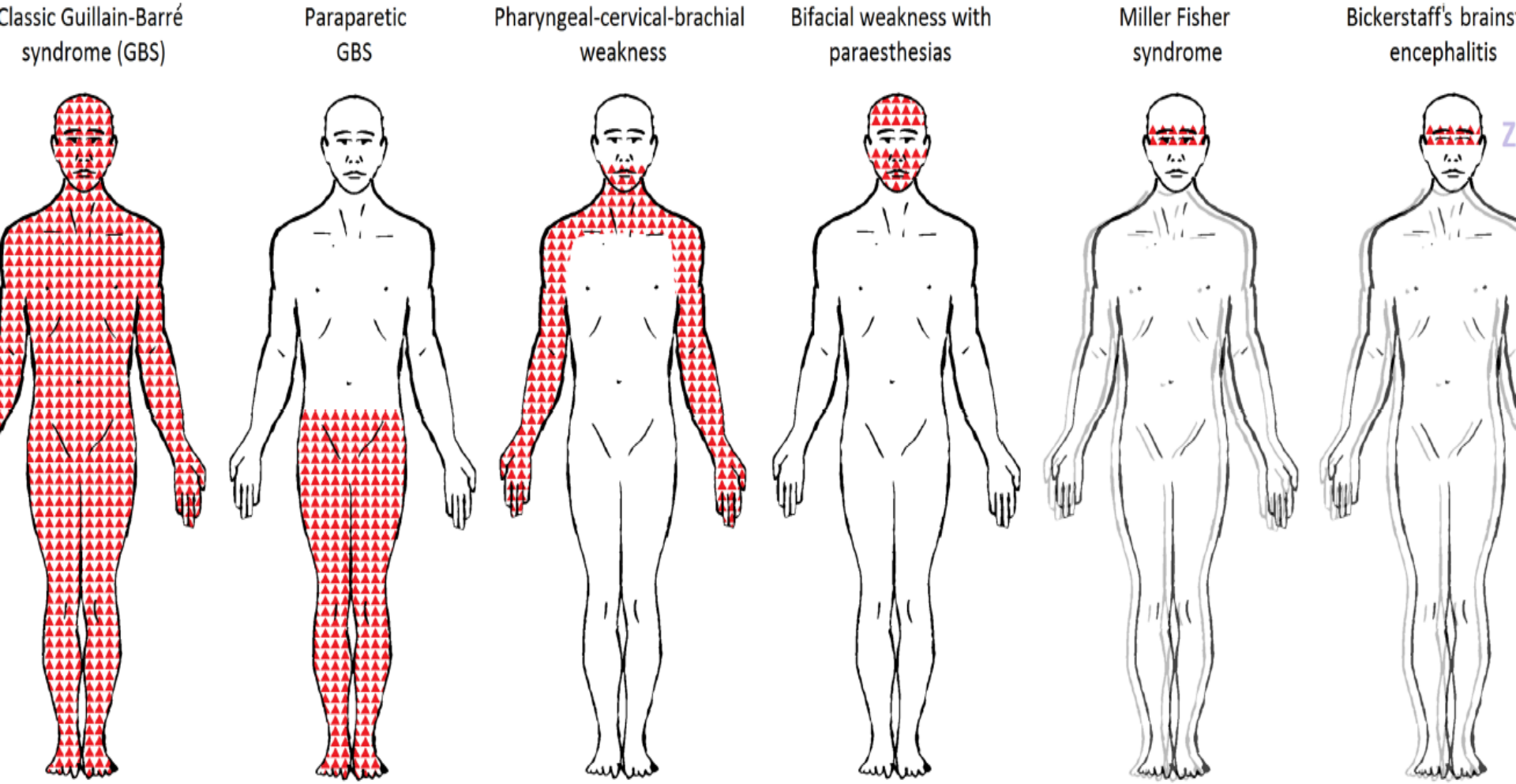
par MM. GEORGES GUILLAIN, J.-A. BARRÉ et A. STROHL.

Landry's Ascending Paralysis
1959

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 "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.
- CSF – Albuminocytological dissociation
- NCS – Demyelinating neuropathy >> Axonal

GBS and Regional Variants



GBS

- **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 (CMV)
  - Epstein-Barr virus (EBV)
  - Mycoplasma pneumoniae
  - COVID-19
  - possibly Haemophilus influenzae , Hep A.

- **Postpartum , Surgery**

GBS treatment- Immunotherapy

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

Supportive Management

- Careful monitoring of vital capacity (VC) with intubation for those with a VC of <15 ml/kg or which is rapidly dropping
- Cardiac monitoring throughout the acute stage.
- 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

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

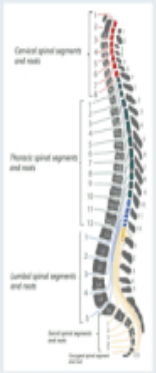
MRI Brain normal

- Diagnosis – Myasthenic crisis
- Rx- IVIg or PE

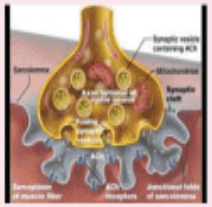
Causes of Neuromuscular Weakness

Spinal Cord

- Demyelinating Disease (MS)
- Epidural abscess
- Infarction
- Syringomyelia
- Tetanus
- Transverse Myelitis
- Trauma
- Tumor



Neuromuscular junction



- Botulism
- Lambert Eaton Syndrome
- Myasthenia Gravis
- Organophosphate poisoning
- Scorpion sting
- Shellfish poisoning
- Meds (anticholinesterase inhibitors, aminoglycosides)

Motor Nerves

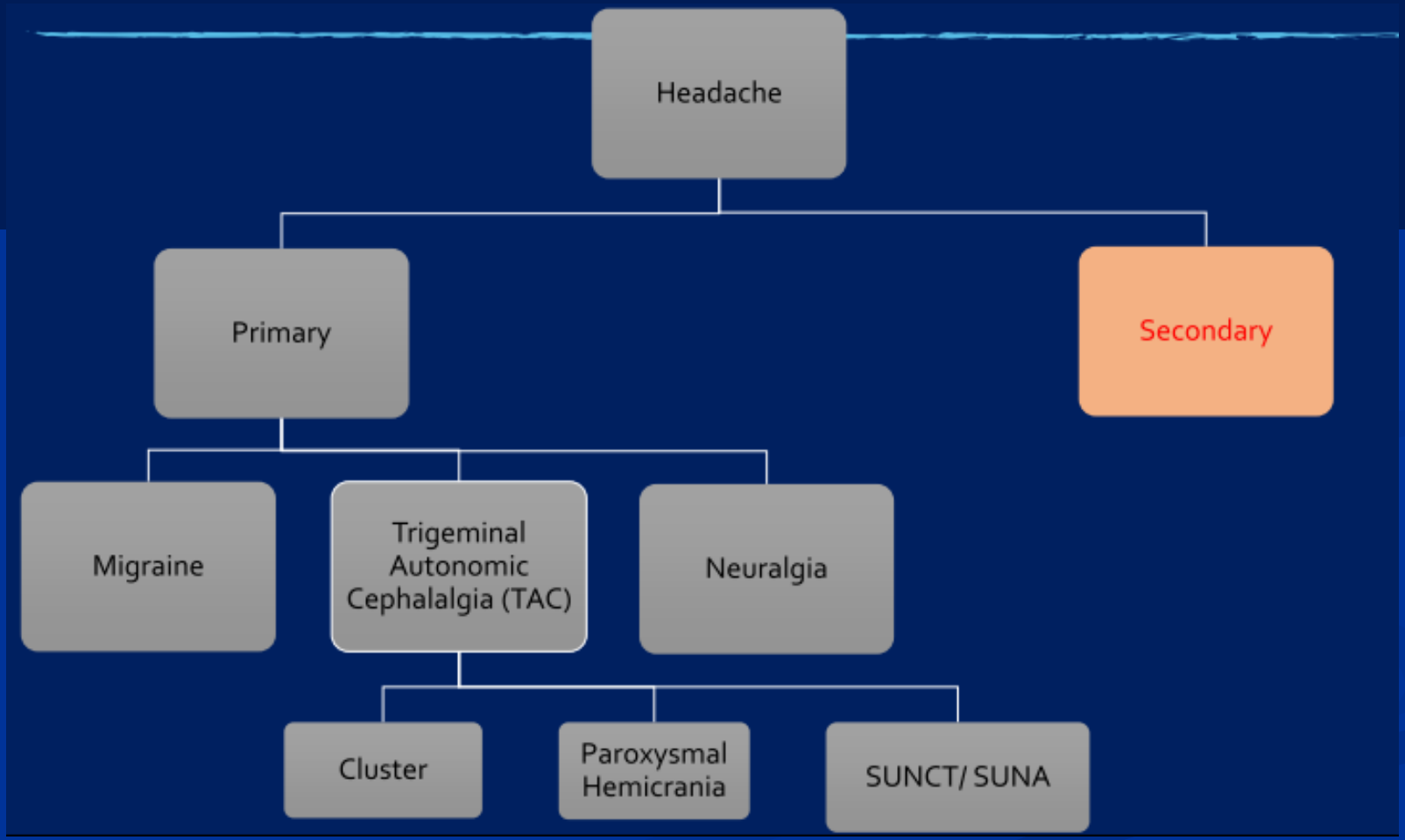
- ALS
- Cervical spondylosis
- Poliomyelitis
- Guillain-Barre syndrome
- Mononeuritis multiplex
- Phrenic nerve injury
- Sarcoid
- Toxins (heavy metals)
- Critical illness neuromyopathy

Muscles

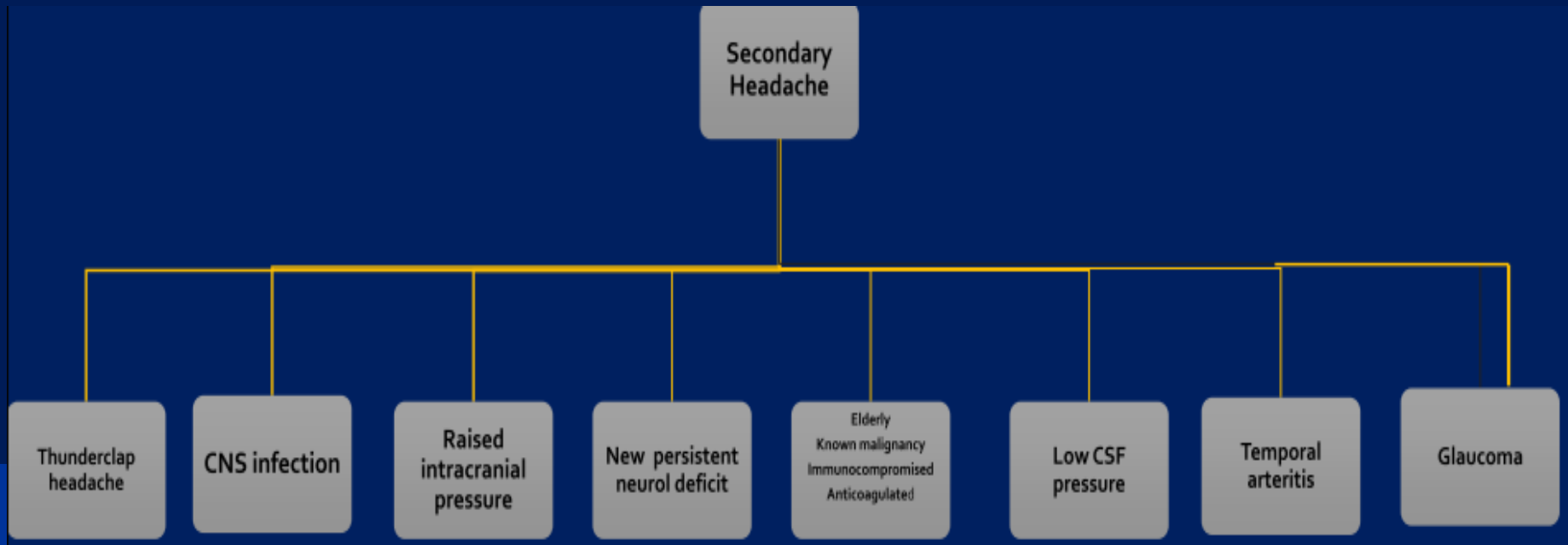
- Acid maltase deficiency
- Malnutrition
- Metabolic abnormalities (hypoK)
- Mitochondrial myopathy
- Muscular dystrophy
- Myotonic dystrophy
- Polymyositis/dermatomyositis
- Thyroid disease

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

Headache in the ER



ACUTE TREATMENT FOR SEVERE PRIMARY HEADACHES	
Headache phenotype	*Drug*
Migraine	Combination therapy IV paracetamol 1g High dose IV aspirin 900mg Triptan (e.g. s/c sumatriptan 6mg) Anti-emetic (e.g. metoclopramide or prochlorperazine) Greater occipital nerve block
Cluster headache	High flow oxygen (12L/minute) Subcutaneous triptan e.g. sumatriptan 3-6mg Intranasal triptan e.g. sumatriptan 10-20mg



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
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

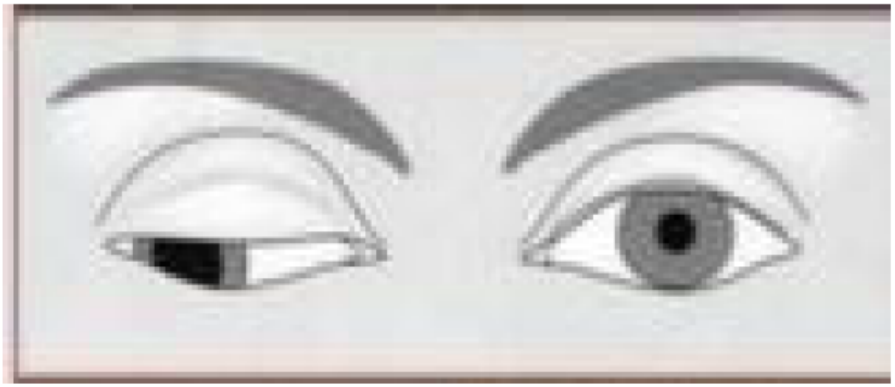
- Very severe, maximal severity at onset
- Feels like being “hit on the head with a bat”
- Commonly occipital
- 25% associated with Subarachnoid hemorrhage

Classic Symptoms of Subarachnoid Hemorrhage

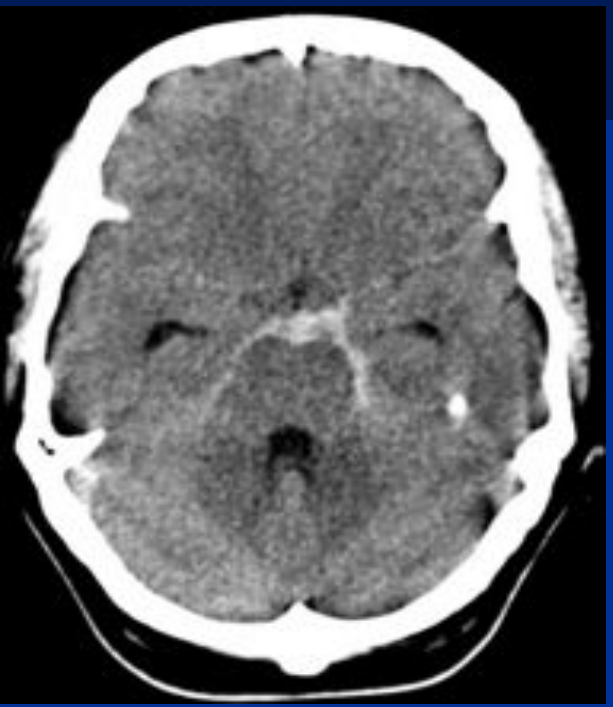
- Sudden, unusually severe or "thunderclap" headache
- Loss of consciousness (transient or persistent if severe)
- Pain in neck, back, eye
- Nausea, vomiting, photophobia

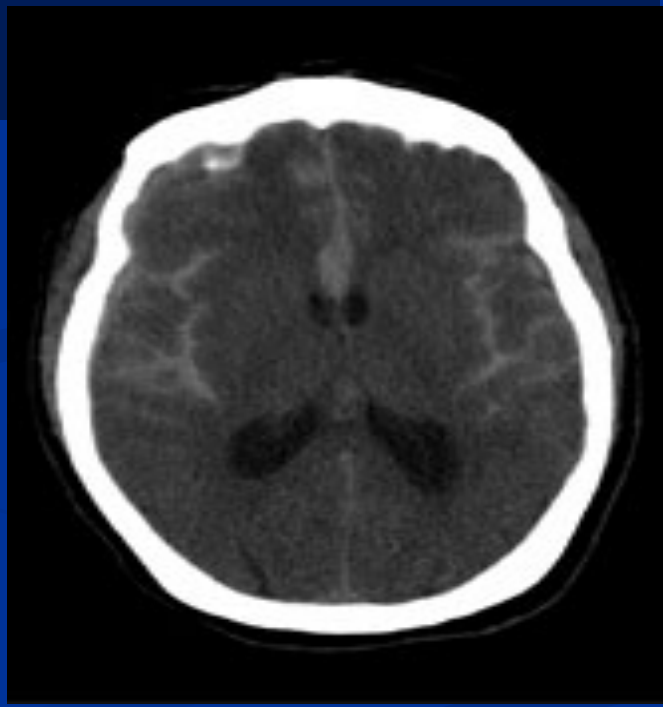
Classic Signs of Subarachnoid Hemorrhage

- 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



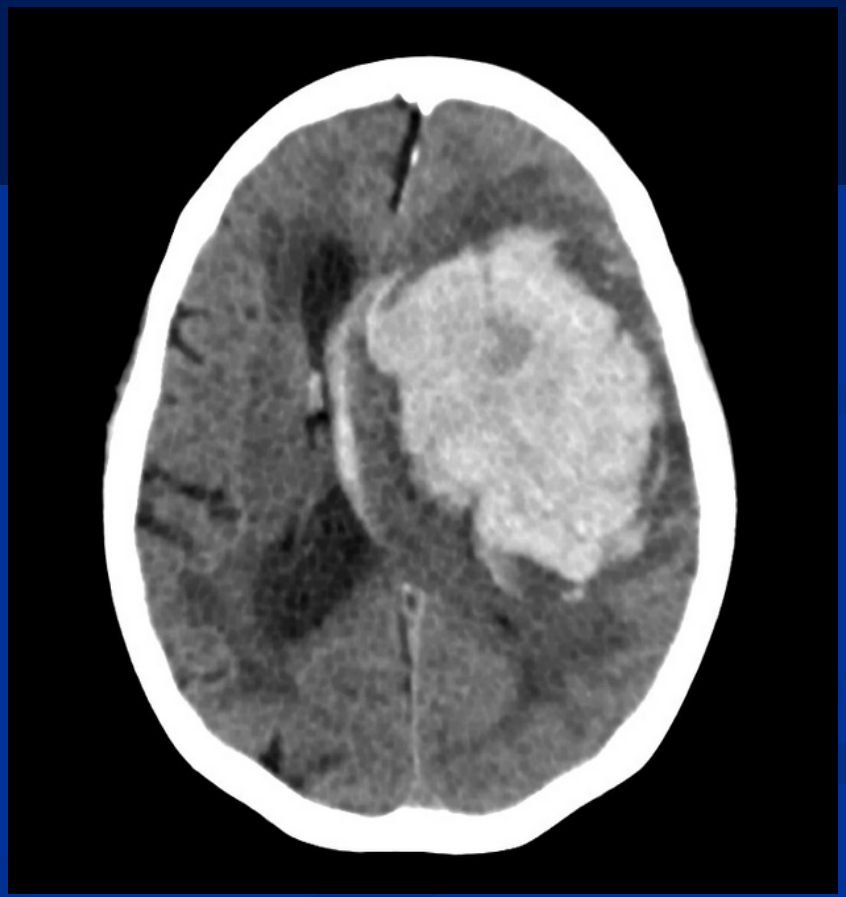
Subarachnoid hemorrhage





Subarachnoid hemorrhage

Intracerebral parenchymal hemorrhage



Seizures, Pseudo-seizures and Status (Pseudo)Epilepticus

First Aid
Tonic-Clonic Seizure

- After seizure ends, turn person on side with face turned toward ground to keep airway clear, protect from nearby hazards
- Transfer to hospital needed for:
  - Multiple seizures or status epilepticus
  - Person is pregnant, injured, diabetic
  - New onset seizures
- DO NOT put any object in mouth or restrain

Evaluation of a First Seizure
Exclude provoking factors

- History, physical
- Blood tests: CBC, electrolytes, glucose, calcium, magnesium, phosphate, hepatic and renal function
- Lumbar puncture
  (only if meningitis or encephalitis suspected and potential for brain herniation is excluded)
- Blood or urine screen for drugs
- ECG ; Electroencephalogram (EEG)
- CT or MR brain scan

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

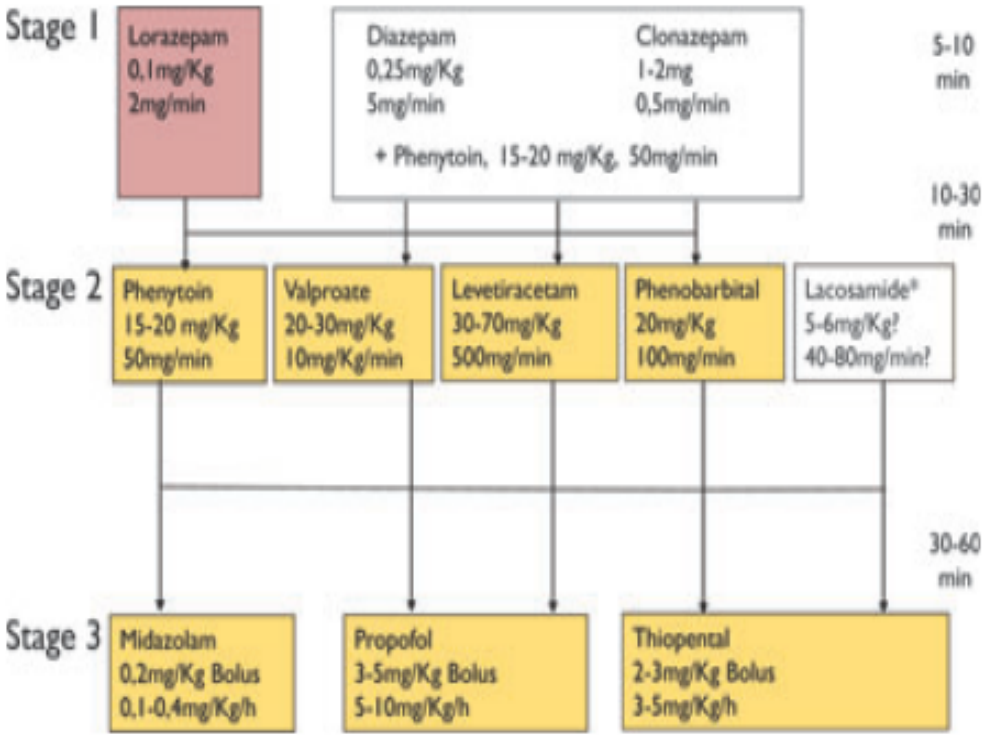


Figure 5.

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

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 PNES

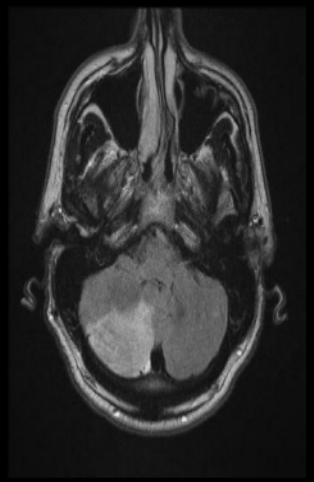
- Lateral tongue biting is poorly sensitive but highly specific (99%) for a generalized seizure.

Vertigo

- No Loss of consciousness (not syncope)
- Acute-onset persistent vertigo lasting hours-few days , think of :
  - Cerebellar stroke
  - Inner ear problems (vestibular neuritis)
- If recurrent , other etiologies (Migraine …)

Vertigo

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

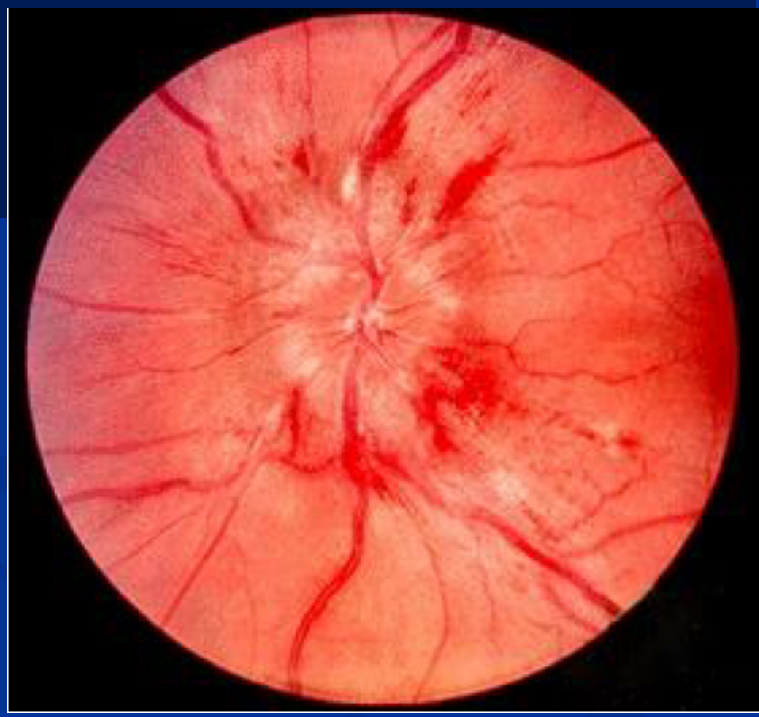


Brain scan needed in acute vertigo:

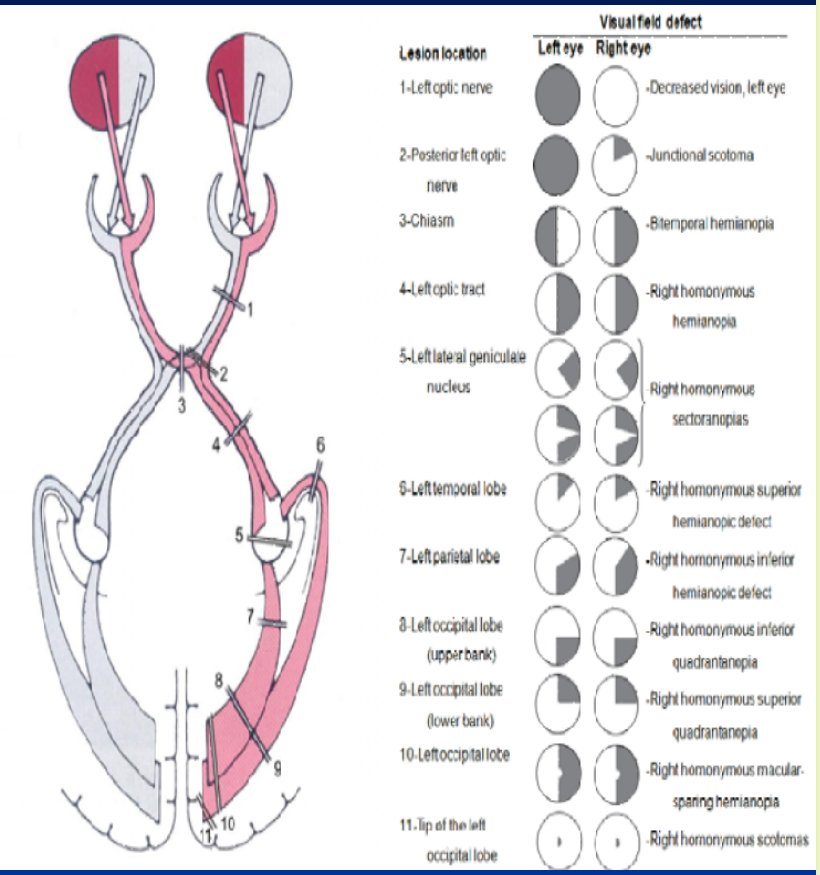
- Intact head impulse test
- New onset (occipital) headache
- Any central symptoms or signs
- Acute deafness

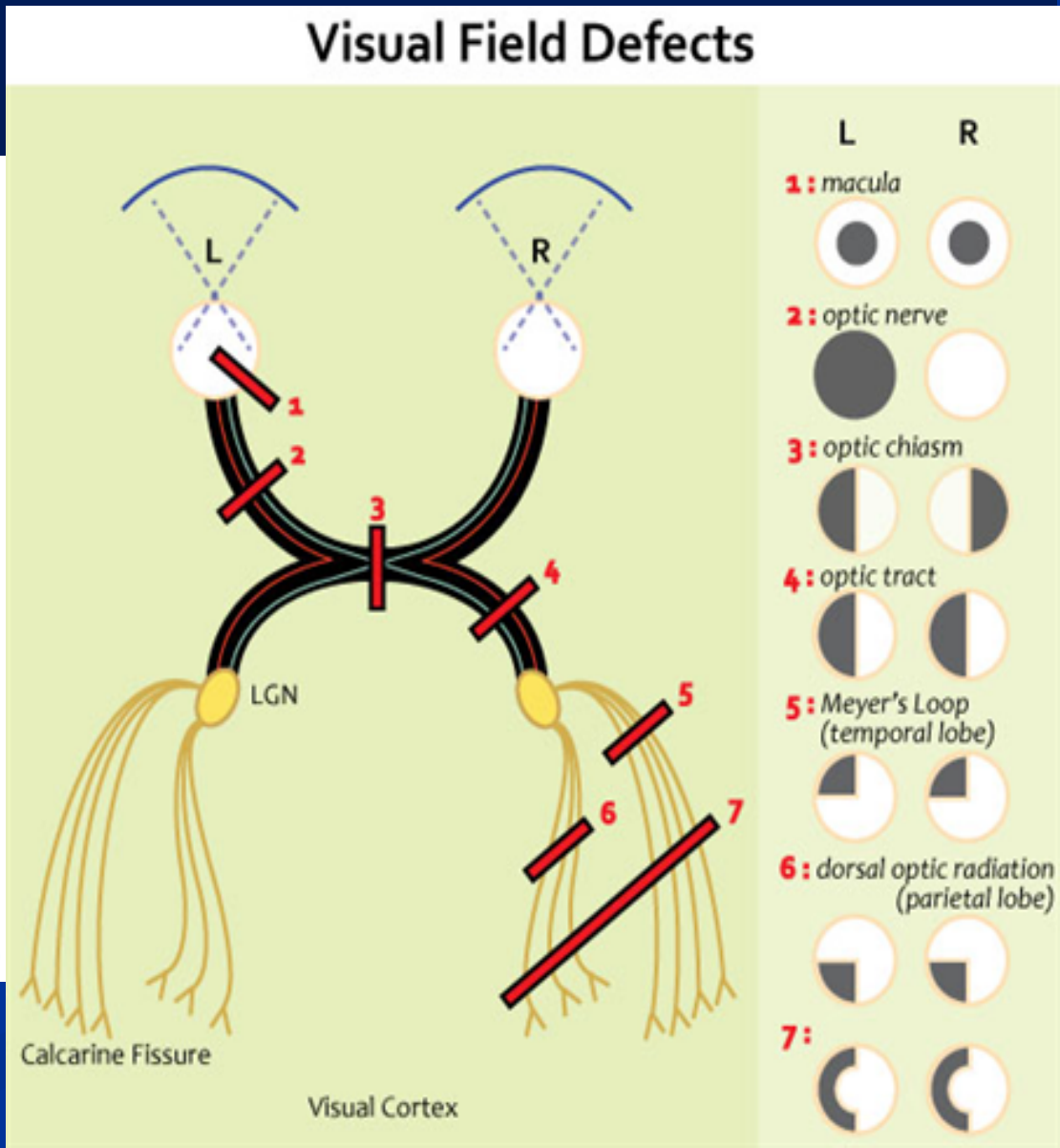
Neurological causes of sudden loss of vision

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



Anatomy of Visual Pathways





Case 3

- 30 year-old-lady-- 2/12 hx of vomiting and poor oral intake after GI surgery

- Last few days: dizzy, unsteady, leg pains, parasthesiae, oscillopsia/diplopia, slow speech and concentration. Sleepy and easy irritability. Then decreased vision and unable to walk (wheel-chair).

- O/E:

  Stable V/S, GCS 15/15, looks anxious/irritable.

  Restriction of eye movements

  Nystagmus. Gait ataxia. Otherwise normal.

- Labs: Na 130, K 2.9, AST 75
- Brain CT- Normal.
- Given Rx and MRI Brain requested.
- Next day--- improvement.
- What was the treatment?

  A. steroids

  B. antibiotics + antivirals

  C. IVIG

  D. something else

What was the treatment?

A. steroids

B. antibiotics + antivirals

C. IVIG

D. something else … High-dose intravenous thiamine (vit. B1)

A case of acute Wernicke's Encephalopathy

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

Register your attendance with your university number

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