

Neurological Emergencies

Dr Majed Habahbeh

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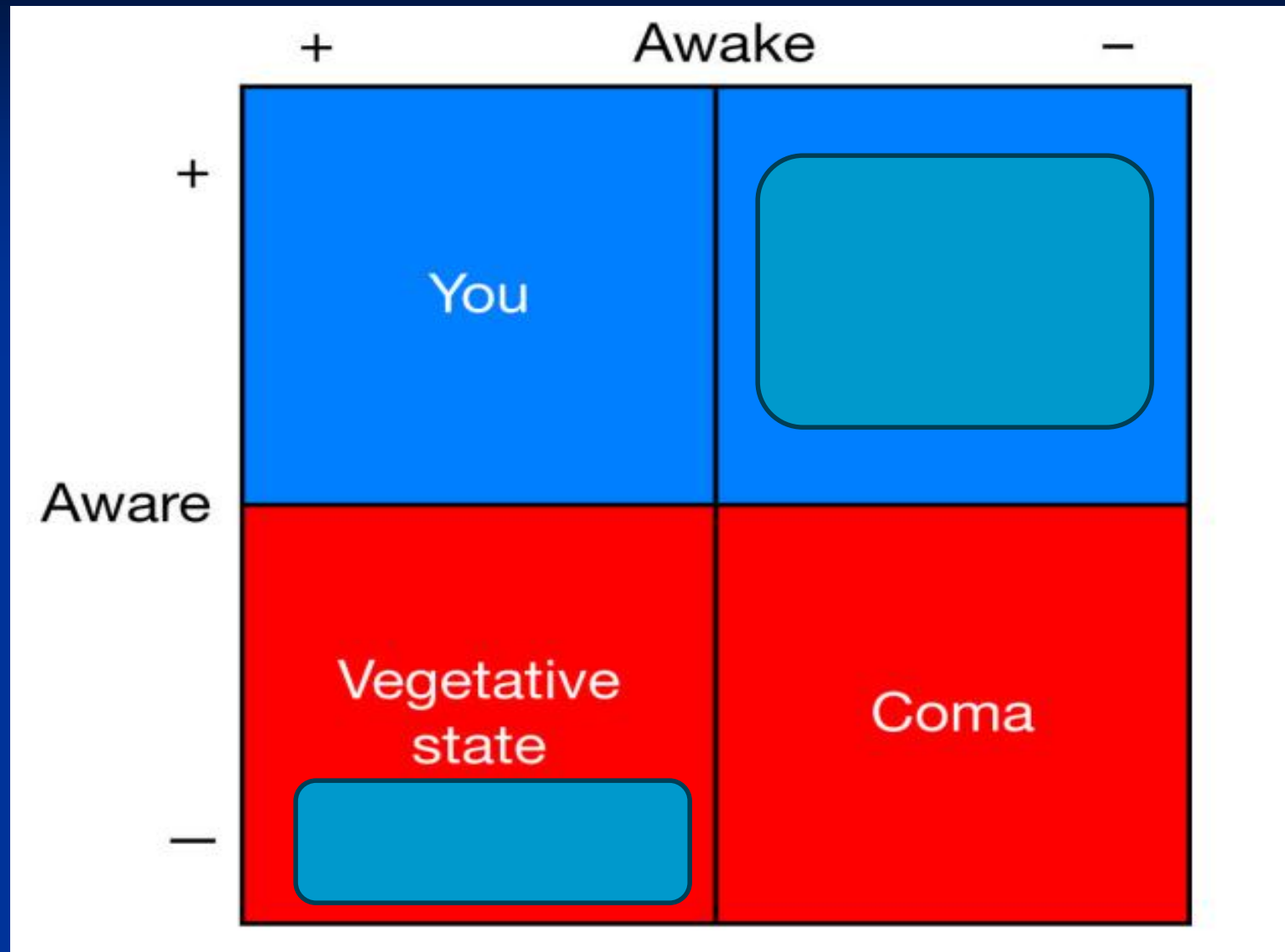
At least 20% of Medical ER visits

- Coma/altered mental status
- CNS infections (Meningitis/encephalitis)
- Acute flaccid paralysis - limbs, bulbar, respiratory (Guillain-Barre' Syndrome, Myasthenia Gravis...)
- Acute myelopathy/spinal cord compression
- Acute Stroke- ischemic and hemorrhagic
- Acute headaches/Subarachnoid hemorrhage
- Seizures/ Status epilepticus
- Vision loss
- Vertigo

Components of Consciousness

Components of Consciousness

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

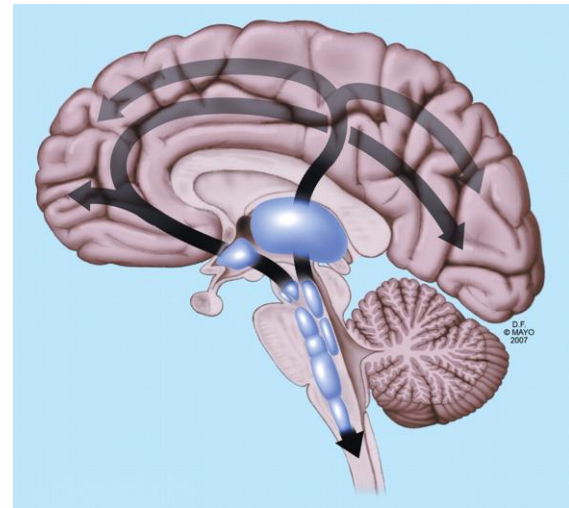


		Awake	
		+	-
Aware	+	You	Dreaming
	-	Vegetative state Acute Confusional state	Coma

Anatomy of consciousness

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

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



Wijdicks E F M Pract Neurol 2010;10:51-60

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Terminology

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



L'ALCOOL TUE

Table 1

Terms for delirium

Acute confusional state

Acute brain failure

Agitation

Altered mental status

Confusion

Encephalopathy

ICU psychosis

Mental status changes

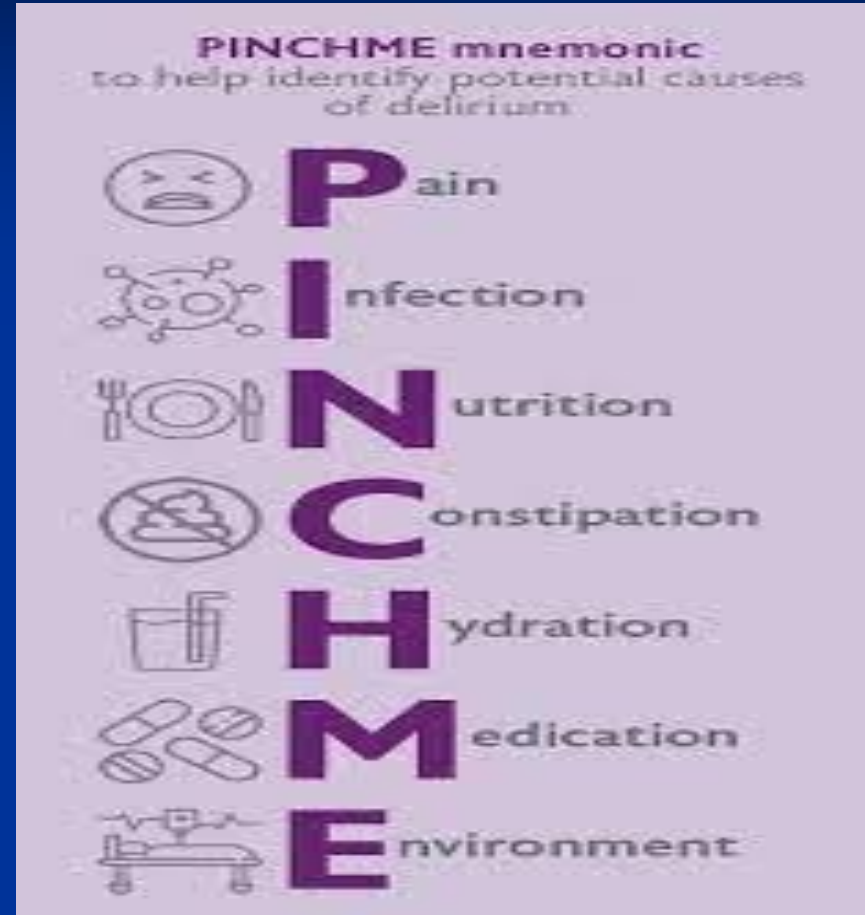
Sundowning

Terminal restlessness (eg, in palliative care)

Delirium

- Delirium is an acute change in mental state, that is characterized by a disturbance in **attention** +/- disorganized thinking and perception (hallucinations) or alertness.
- **Commonly** fluctuating course.
- Types- Hypoactive, **hyperactive** or mixed.
- Common in elderly patients in ICU or post-op.

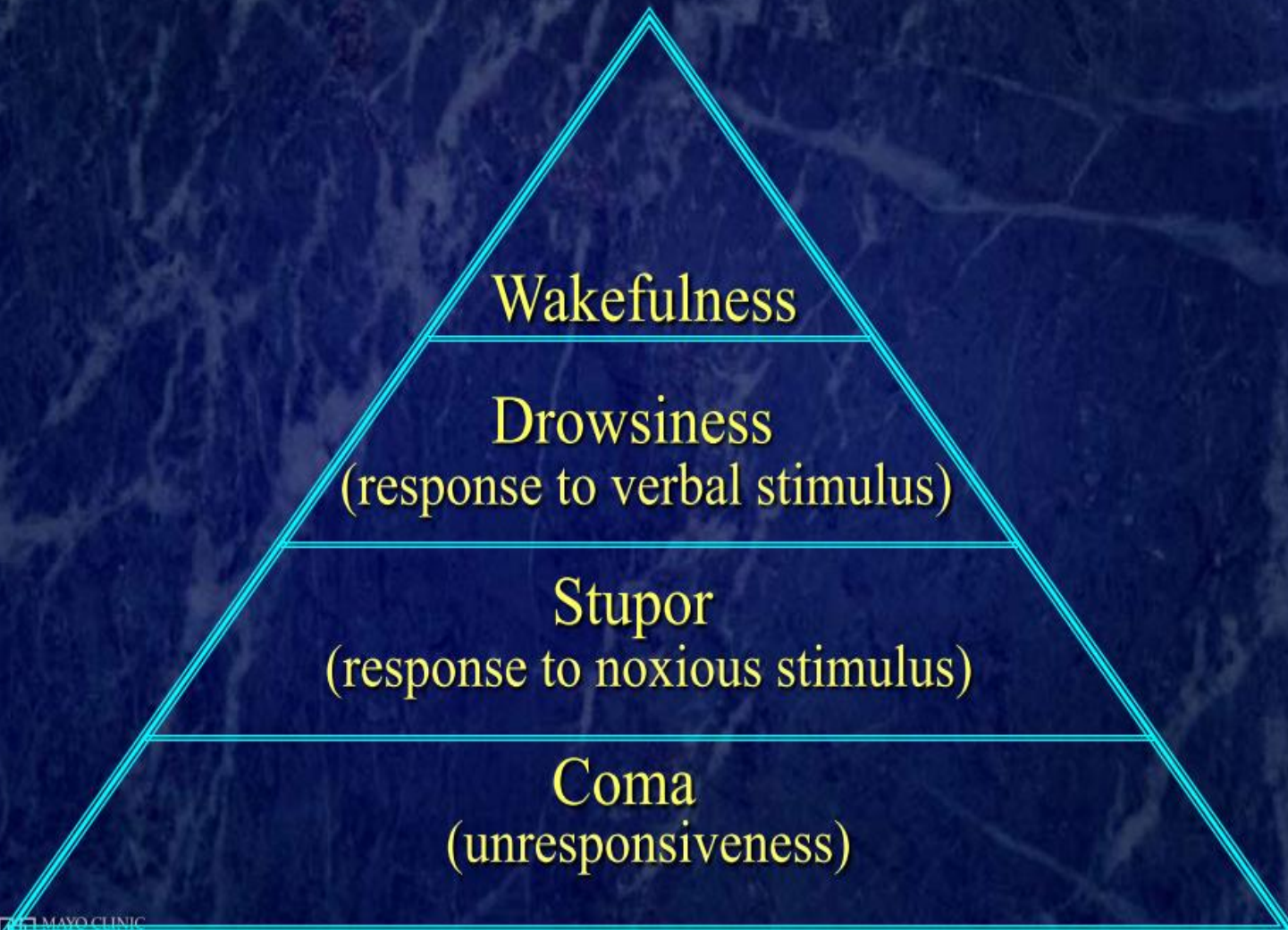
Triggers



The 5 P's :

Pee/Poo/Pain/Pills/Pus

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

Etiology of Coma

- 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

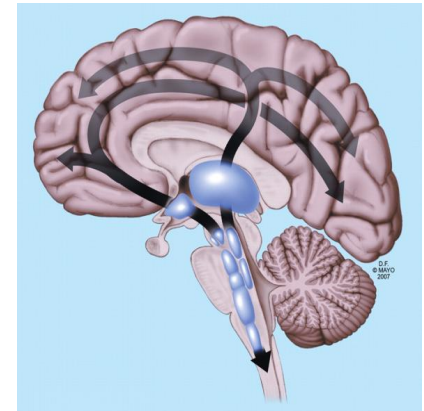
Bilateral

- Intraparenchymal hematoma
- Middle cerebral artery ischemic stroke
- Cerebral venous thrombosis
- Cerebral abscess
- Brain tumor
- Subdural or epidural hematoma
- Subarachnoid hemorrhage
- Traumatic brain injury
- Multiple cerebral infarcts
- Bilateral thalamic infarcts
- Tumors
- Meningitis/encephalitis
- Cerebral edema
- Acute hydrocephalus

Intrinsic brainstem injury, or compression from surrounding damaged tissue

- Pontine hemorrhage
- Basilar artery occlusion and brainstem infarct
- Central pontine myelinolysis
- Brainstem contusions
- 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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Metabolic/Endocrine/Physiological Causes of Coma

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

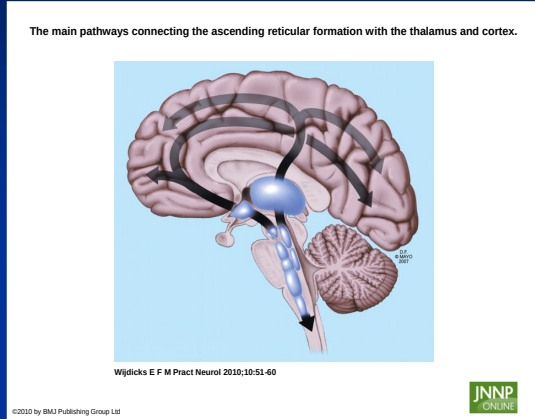
Glucose <40-50 mg/dl

Na <110 or >160 mmol/l

Calcium > 13.5 mg/dl

PaCO₂ > 65 mmHg

Coma mimics : Locked-in Syndrome



Lesion (commonly stroke)
in the ventral pons
damaging the
corticospinal and
corticobulbar tracts and
sparing ARAS

They can hear, see and
may feel pain

Locked-In Syndrome

People with locked-in syndrome are often initially in a coma before gradually regaining consciousness.

Most people with locked-in syndrome **CANNOT** consciously or voluntarily:

- Speak or make facial expressions
- Produce any body movements below their eyes
- Chew or swallow

Most people with locked-in syndrome **CAN**:

- Move their eyes up and down (vertically) but not side to side (horizontally, or laterally)
- Blink

All people with locked-in syndrome **CAN**:

- Hear
- Have sleep-wake cycles
- Think and reason

Comprehend people talking or reading to them

Cleveland Clinic

Another coma mimic !

A neurology grand round in the late 19th century: Une leçon du mardi.



Coma mimics:

Psychogenic swoon/unresponsiveness

- **Hysterical coma/conversion reaction**
- **Factitious disorder/Malingering**
- **Acute catatonia**

Patients can be intubated by mistake

Une leçon du mardi.
(A lesson on Tuesday)

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



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 have pseudoseizures- 'fish out of water' flopping.

Examination of the comatose patient

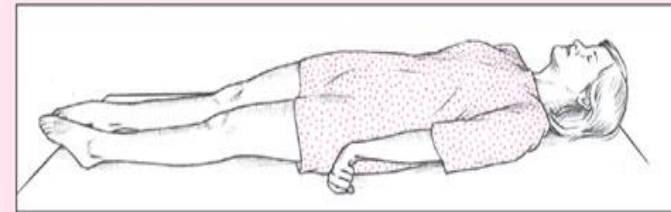
1. Assess the depth of coma.
2. Determine if there is structural brain pathology and aim to localize it.
3. Determine the underlying cause if possible.

Glasgow Coma Scale

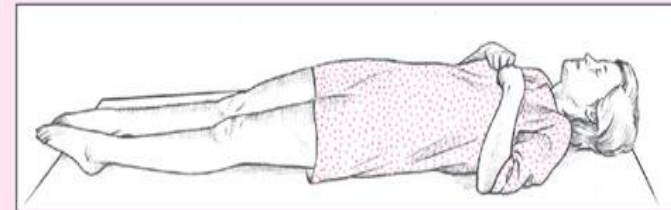
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Comparing decerebrate and decorticate postures

Decerebrate posture results from damage to the upper brain stem. In this posture, the arms are adducted and extended, with the wrists pronated and the fingers flexed. The legs are stiffly extended, with plantar flexion of the feet.



Decorticate posture results from damage to one or both corticospinal tracts. In this posture, the arms are adducted and flexed, with the wrists and fingers flexed on the chest. The legs are stiffly extended and internally rotated, with plantar flexion of the feet.



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

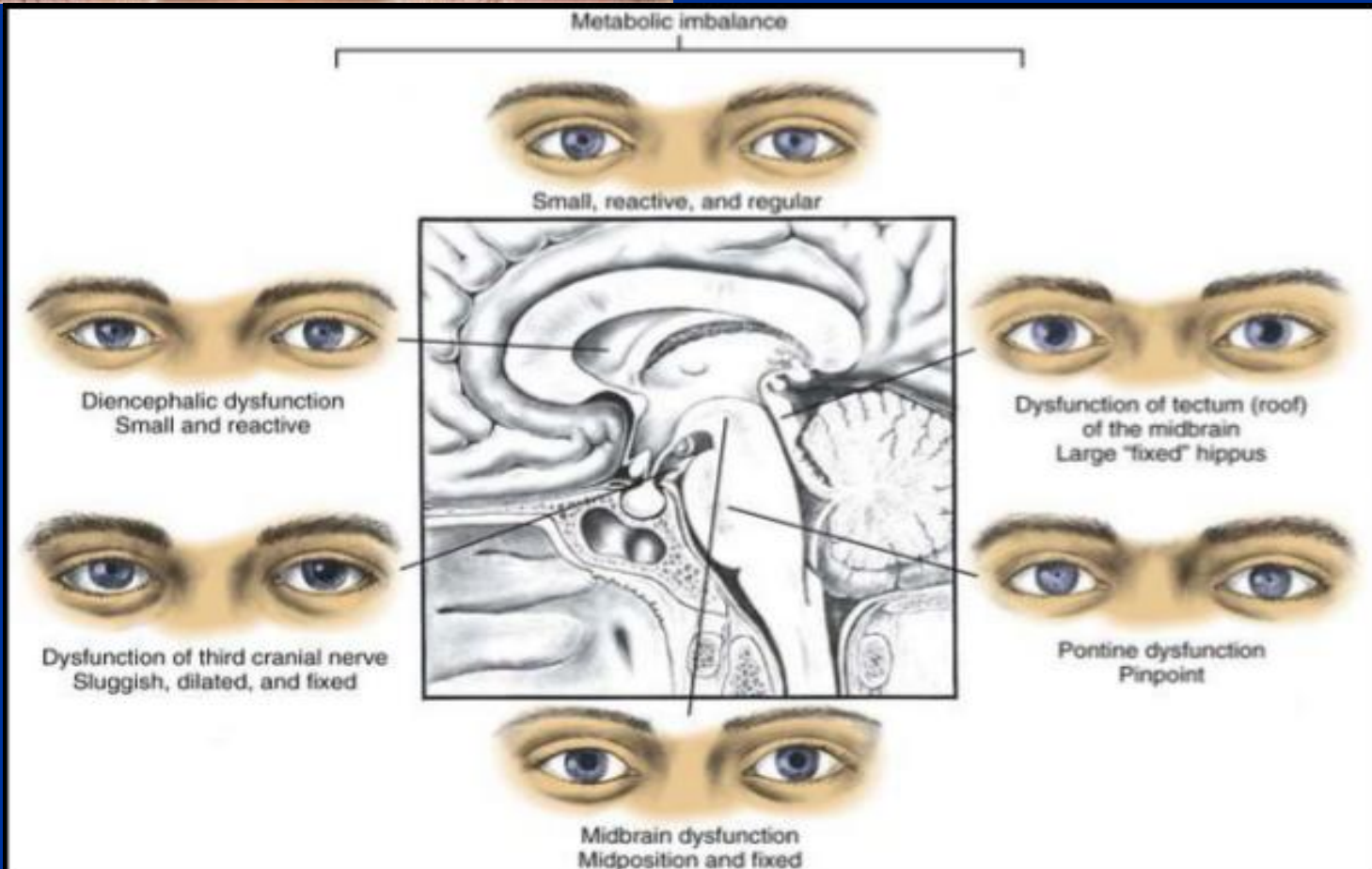
1. Assess the depth of coma.
2. Determine if there is structural brain pathology and aim to localize it.
3. Determine the underlying cause if possible.

Examination of the comatose patient

- Vital signs
- General : Breath smell/skin color/sweating ...etc.
- Determine if there is structural brain pathology and aim to localize it :
 - meningism
 - focal weakness/ tone /abnormal movements
 - pupils/eye position and movement/breathing pattern
 - DTR's and plantar response

Lesion Localization

Pinpoint pupils: opioid intoxication or pontine haemorrhage.



Lesion Localization

- **Roving eye movements** indicate that the brainstem is intact.
- **Skew deviation** of the eyes suggests an acute brainstem injury.



Conjugate gaze deviation

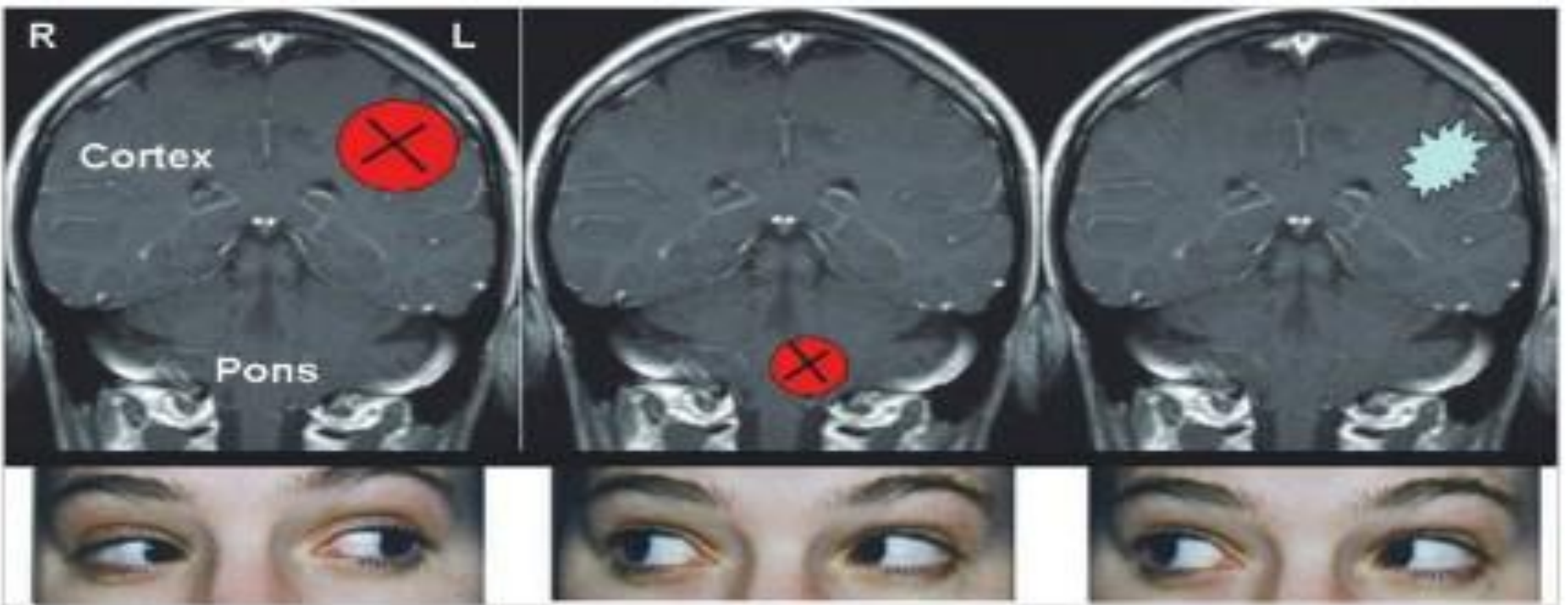


Fig. 13.121 Gaze deviations in cerebral lesions and seizures: the eyes deviate horizontally toward a cortical lesion (left); the eyes deviate horizontally away from a pontine lesion (center); the eyes deviate horizontally away from a cortical seizure focus (right).

Examination of the comatose patient

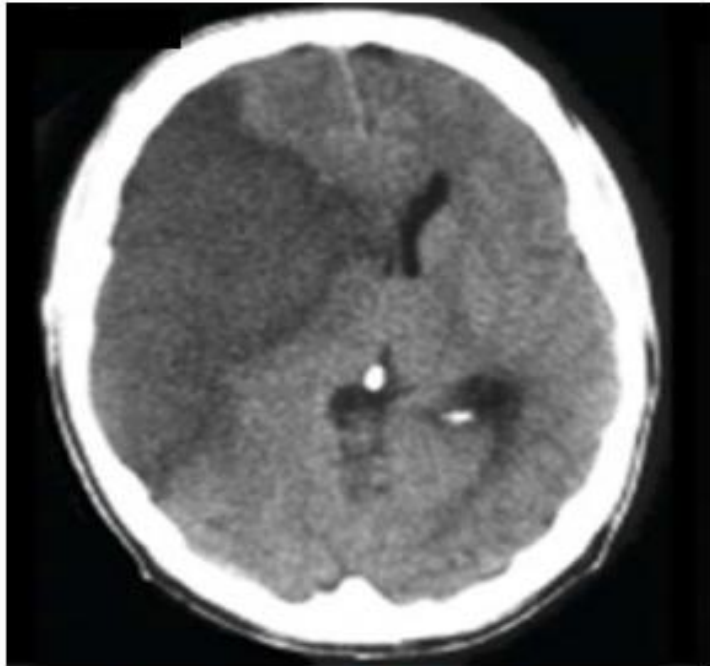
1. Assess the depth of coma.
2. Determine if there is structural brain pathology and aim to localize it.
3. Determine the underlying cause if possible.

What is the cause of coma ?

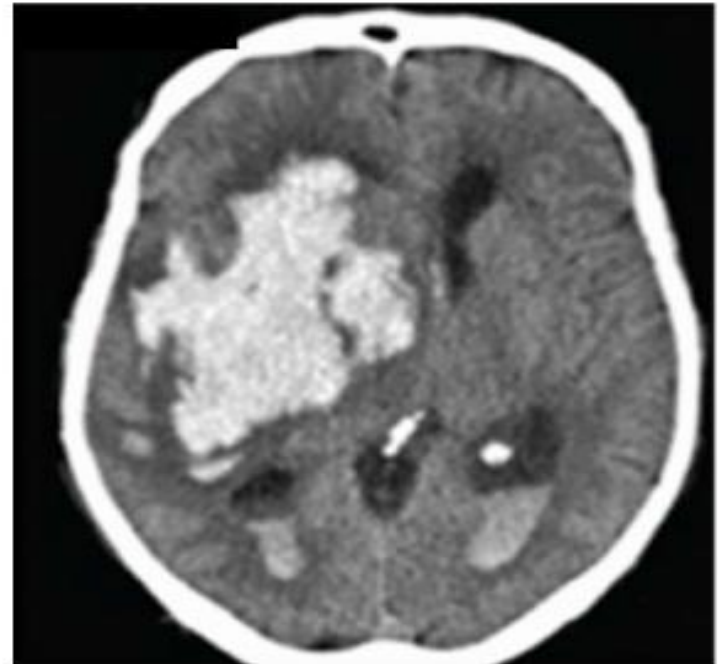
- CT and MRI of the brain are very important in the work-up of a comatose patient. However, **in many cases of coma, the brain CT may be normal or only show minor subtle findings.**



Abnormal Brain CT scan



**Ischemic Stroke
(dark/hypodense)**



**Hemorrhagic Stroke
(bright/hyperdense)**

Management of Coma in the First Hour

- Improve oxygenation
- Intubate if patient cannot protect the airway (ie, pooling secretions, gurgling sounds) or 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).
- Naloxone if opioid intoxication is suspected.
- Flumazenil reverses any benzodiazepine toxicity.

Coma due to drugs/toxins



- Careful history (? mixed overdose)
- Coma of uncertain etiology (normal imaging/Ix) should be assumed drug-induced until proven otherwise and patients supported aggressively (ABC).
- Even with deep coma , absent brainstem reflexes and electrocerebral silence on EEG there is potential for **complete recovery**

Possible CNS Infection ?

- Acute Bacterial Meningitis
- Viral Encephalitis
- Brain abscess
- Subdural Empyema
- Cerebral Malaria –causes rapidly progressive coma
- TB Meningitis



High index of suspicion is necessary esp. in immunosuppressed (DM, Cancer, Steroids, Biologics, HIV)

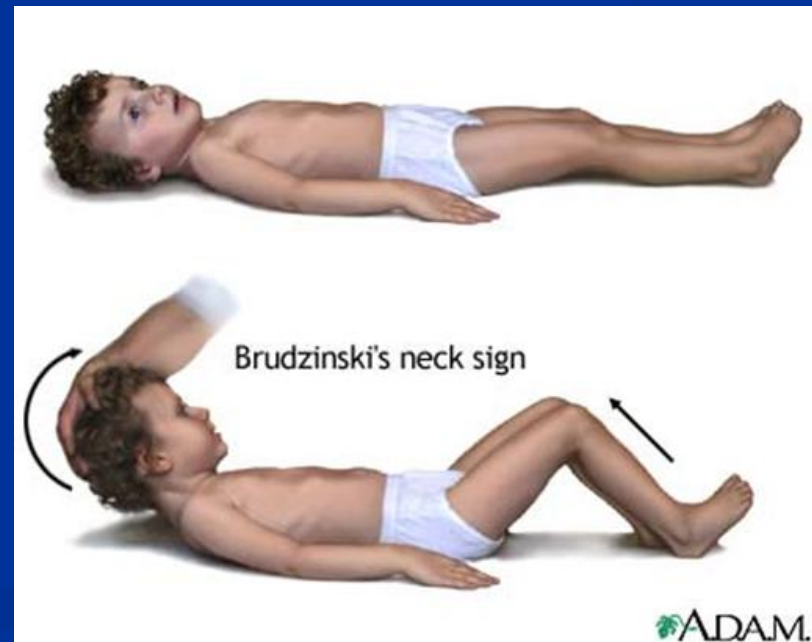
Acute bacterial Meningitis (ABM)

- A common and serious medical emergency
- 100% curable if treated adequately or 100% fatal

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



Meningococcal septicemia rash



ABM : Etiology

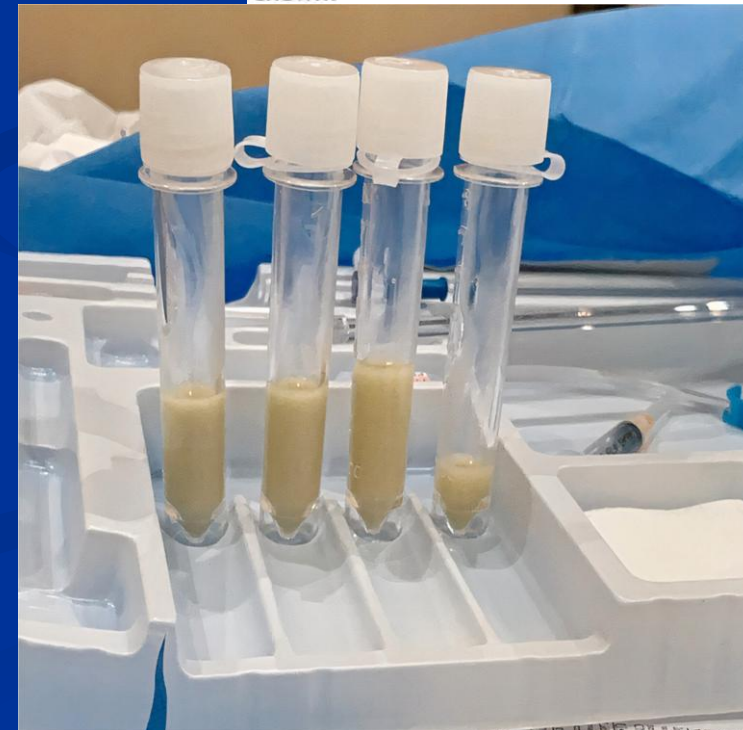
- **Streptococcus pneumoniae (pneumococcus)- Most common cause.**
- **Neisseria meningitidis (meningococcus).** This is a highly contagious infection that affects mainly teenagers and young adults. It may cause local epidemics in college dormitories, boarding schools and military bases.
- **Listeria 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
 - Clinical presentation and blood work-up
 - Confirm by CSF examination
 - If LP is contraindicated (clinically or by Brain CT) , send blood cultures and start empirical antibiotics.
- CSF: ↑Pressure, turbid, ↑cells (mostly polymorphs), ↑protein, ↓sugar to < 40% of blood sugar, ↑lactate > 2.4 mmol/l
- CSF Gram stain, culture
- CSF PCR



Figure 2 – Grossly cloudy cerebrospinal fluid obtained from lumbar puncture is shown.



Treatment of ABM

- Ceftriaxone (2g every 12 hrs) **or** cefotaxime (8–12 g daily, divided doses every 6 h intravenously) +/- Vancomycin (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- **possible benefit in pneumococcal meningitis**

Viral meningitis

- Viral meningitis is usually mild and self-limited. More common in children and infants.
- CSF findings/PCR
- Most cases are caused by a group of viruses known as non-polio enteroviruses (echoviruses and coxsackie viruses), mumps and measles. No treatment needed.
- HSV and VZV are more symptomatic and usually treated with IV Acyclovir.
- Arboviruses (West Nile virus)

CSF

Table 1 | Typical cerebrospinal fluid (CSF) findings in infectious meningitis^{1 3 14}

Cause of meningitis	White blood cell count (cells/mm ³ /10 ⁶ 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

- 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 HSV2 .
- The most distinctive presenting features are fever, disorientation, aphasia and behavioral disturbances, and up to a third of patients have seizures.

HSV Encephalitis

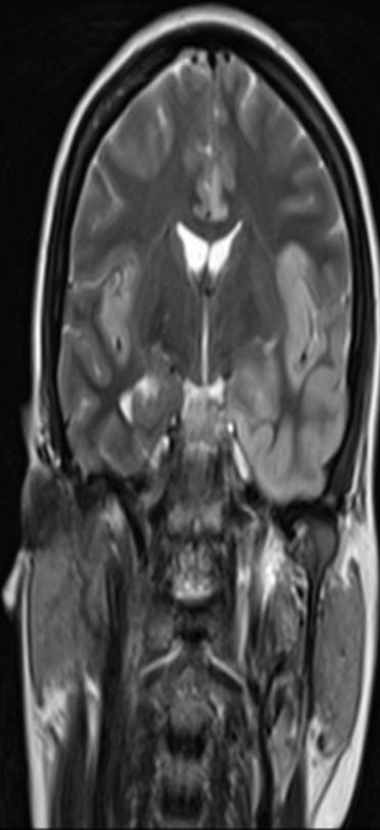
- Brain MRI can be normal in some patients in the first 48 hours , but imaging abnormalities are seen in almost all patients after 3 days.
- **MRI** shows bilateral asymmetric abnormalities in the limbic system, medial temporal lobes, insular cortices and inferolateral frontal lobes
- **CSF** : clear, mild lymphocytic pleocytosis
Normal or mild ↑protein , normal sugar
PCR (may be negative early- repeat after 48 hrs)
- Prompt Rx with IV aciclovir 10 mg/kg x3 if any suspicion

MRI in a case of HSV encephalitis

AYSHAA W.O HUSEEN, Unknown
ID: 9832058621
* 1/1/1983, F
Study 2654478
4/20/2019
12:03:50 PM
15 IMA

HAL

King Hussein Radiology Department
Skyra
HFS



5cm

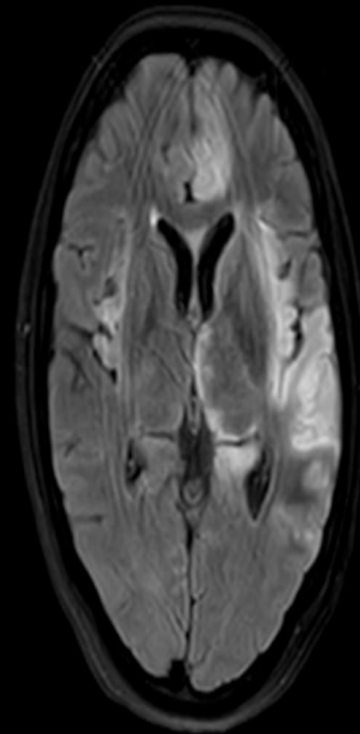
SL 4
SP P12.7
FoV 220*220
192*256
Cor>Tra(-11.4)>Sag(0.6)
W: 1142
C: 539

TE 88
TR 4960

AYSHAA W.O HUSEEN, Unknown
ID: 9832058621
* 01/01/1983, F
Study 599929703
08/05/2019
04:10:39 PM
13 IMA

AHL

King Hussein Radiology Department
Ingenia
HFS

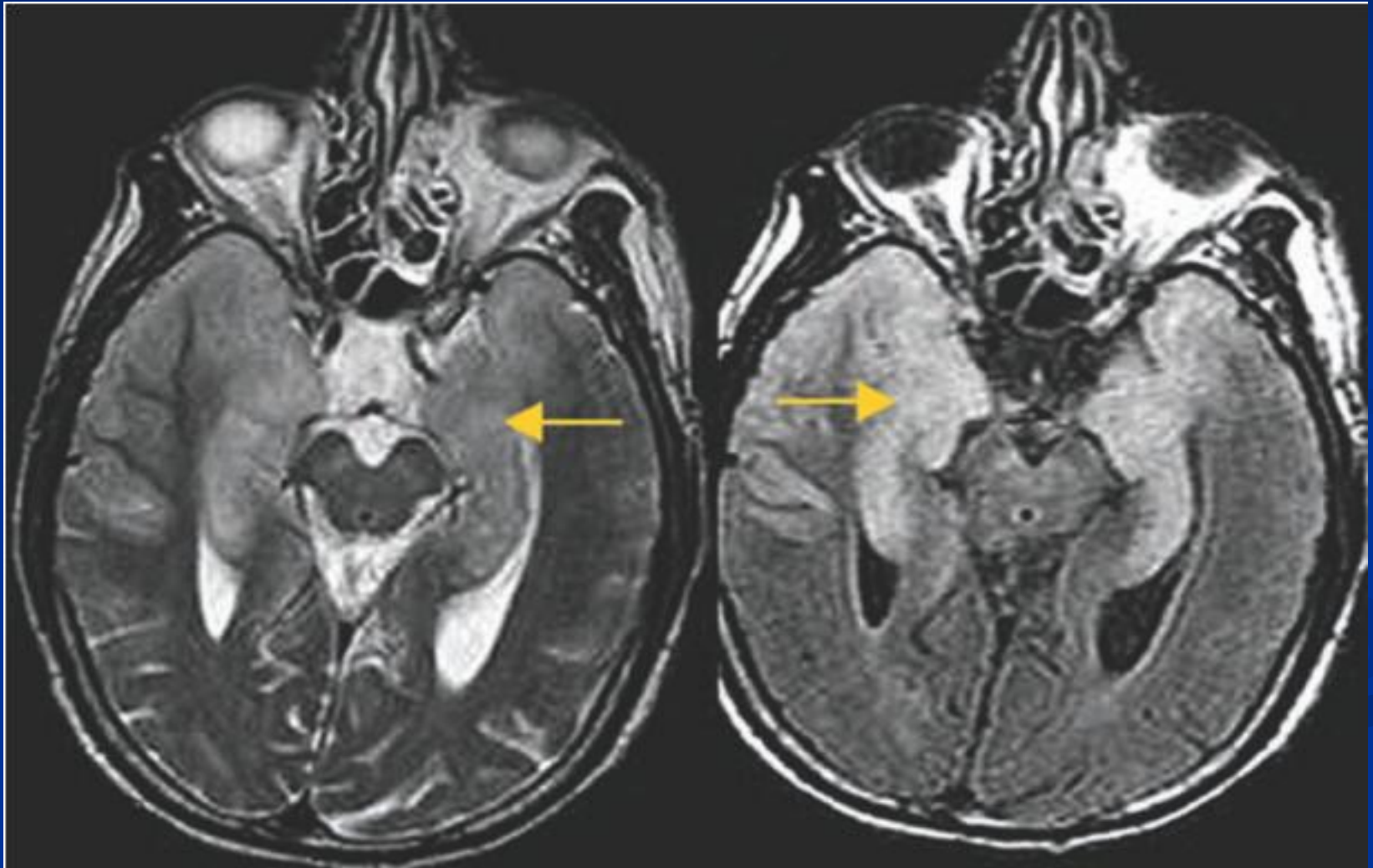


5cm

SL
SP H13
FoV 230*230
160*256
W: 900
C: 512

TE 120
TR 8000
TI 2500

HSV encephalitis



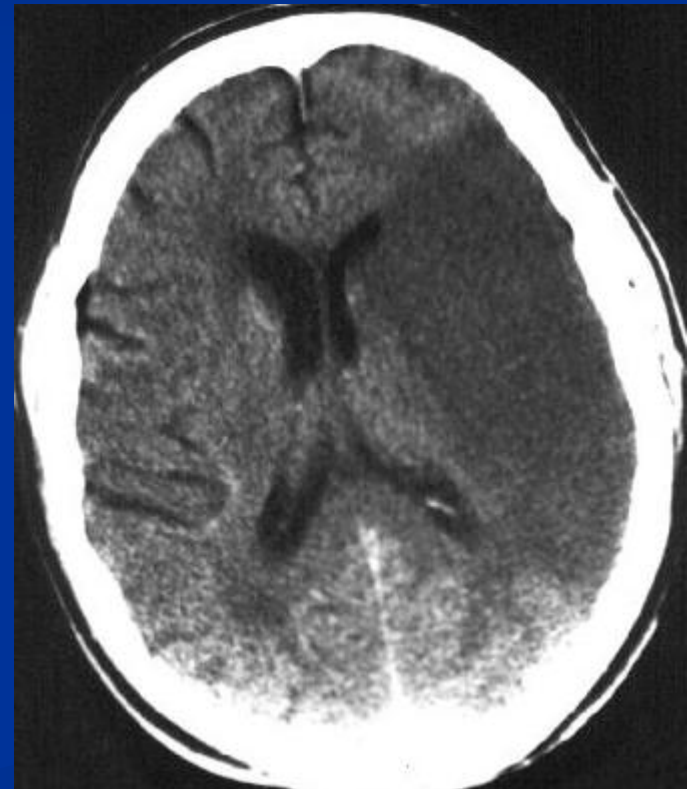
Brain abscess with displacement and hydrocephalus



Brain Abscess



Ischemic Infarct



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



GBS - Clinical Presentation

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.

Weakness is relatively symmetrical and involves proximal and distal muscles.

Maximal weakness at 1-2 weeks.

GBS - Clinical Presentation

Commonly affects the **facial muscles** (> 50% of cases) .

Also bulbar and respiratory muscles - 25% of patients need artificial ventilation.

Extraocular muscle weakness /ptosis - uncommon

Mild/absent sensory and autonomic symptoms.

Back and limb pain common

DTR - Hypo/Areflexia almost always

Differential diagnosis of acute flaccid paralysis

- Brainstem stroke/ encephalitis
- Acute myelopathy
 - Spinal cord infarction/haemorrhage
 - Acute transverse myelitis
- Anterior horn cell - Acute poliomyelitis/WNV. Very rare
- Peripheral neuropathy
 - **Guillain-Barré syndrome**
 - Heavy metals (e.g. thallium)
 - Acute intermittent porphyria
 - Vasculitic neuropathy
 - Lymphomatous neuropathy

Differential diagnosis of acute flaccid paralysis

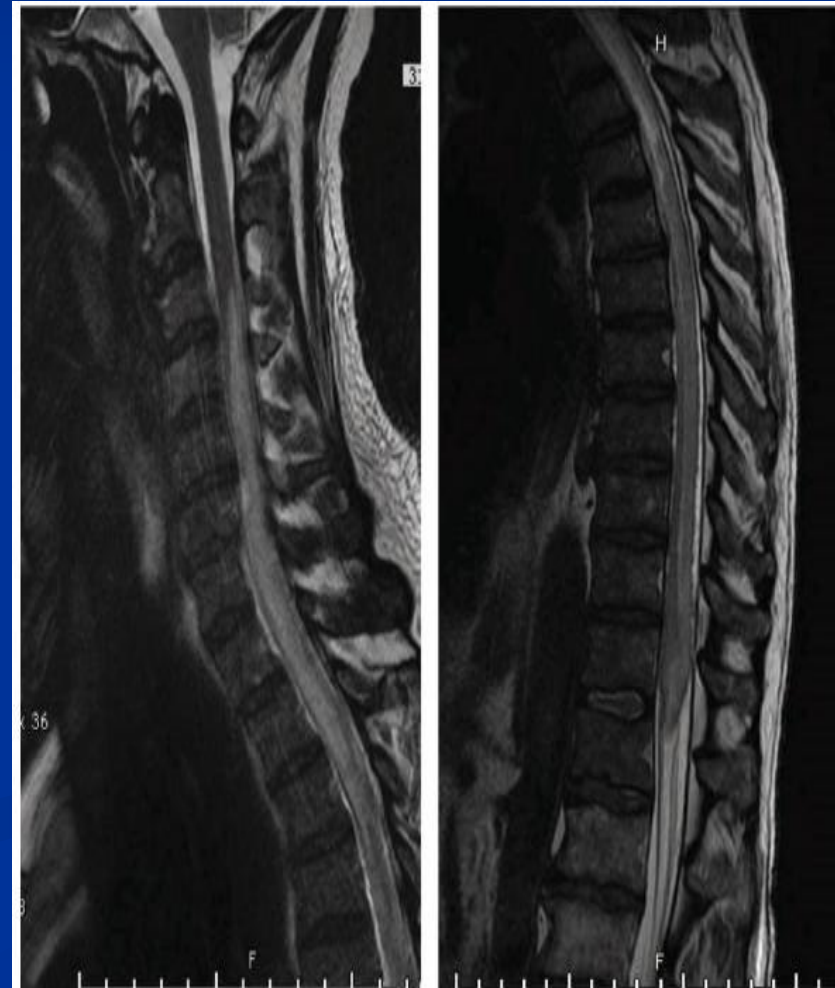
- Disorders of neuromuscular transmission
 - Myasthenia gravis / Lambert-Eston Myasthenic Syndrome
 - Botulism

- Disorders of muscle
 - Hypo- or hyperkalaemia / Periodic paralyses
 - Hypophosphatemia/ Hypermagnesemia
 - Inflammatory myopathy
 - Acute rhabdomyolysis

Investigations

- GBS is a clinical diagnosis - all investigations can be normal in the first few days.
- CSF – Albumino-cytological dissociation
- Nerve Conduction Studies – Demyelinating neuropathy >> Axonal
- Spine MRI
- Blood tests (K/P/Mg/CPK/ESR) to exclude other diagnoses

Spine MRI- Myelitis.



GBS triggers

- **Infections** -two thirds of cases follow an infection (RTI, GI) 1-3 weeks before the onset of neurological symptoms.
 - Campylobacter jejuni
 - Cytomegalovirus
 - Epstein-Barr virus
 - Mycoplasma pneumoniae
 - Influenza/ ? COVID-19
 - Hepatitis E/A
 - HIV seroconversion (High CSF cell count)
- **Postpartum**
- **Surgery**
- **Vaccinations rarely (influenza, VZV)**

GBS treatment- Immunotherapy

- Intravenous Immunoglobulins – 0.4 g/kg daily for 5 days
- OR
- Plasma exchange (PE) : 4-5 exchanges

IVIg = PE = PE + IVIg

- Switching therapies or second course of IVIg- Not recommended by guidelines

Supportive Management for possible complications - Breathing

- SOB , sweating, rapid shallow breaths, tachycardia, use of accessory muscles.
- Neck flexion weakness – mirrors diaphragm weakness
- Careful monitoring of vital capacity (VC) with intubation for those with a VC of $< 10-15$ ml/kg or which is rapidly dropping.
- Mouth PIP and PEP may help – NOT PEFR
- Facial weakness can lead to inaccurate measurement of all these.
- Single breath count (>20 is reassuring and correlates with $VC > 1.5$ L)
- Paradoxical abdominal breathing – $VC < 10\%$ normal
- O2 Sat. not useful for monitoring-occurs late

Supportive Management for other possible complications

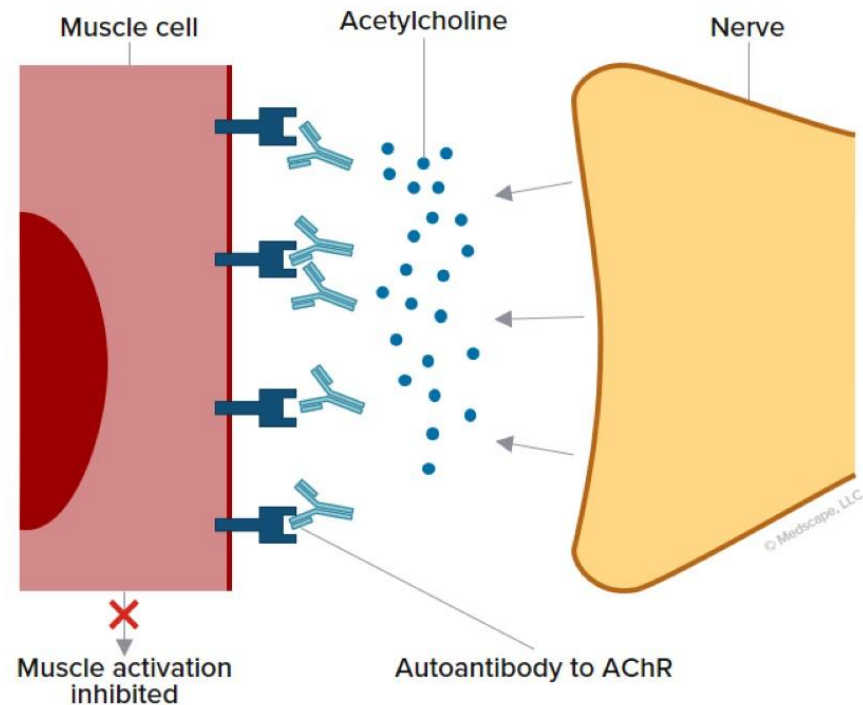
- Bulbar weakness/aspiration – nasal quiet speech , weak cough, choking, gurgling
- Cardiac monitoring for possible arrhythmias throughout the acute stages .
- Venous thromboembolism prophylaxis with compression stockings and low molecular weight heparin is recommended for non-ambulant patients

Myasthenia gravis

gMG Pathophysiology

Autoimmune NMJ Disorder

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



MG: Weakness-1

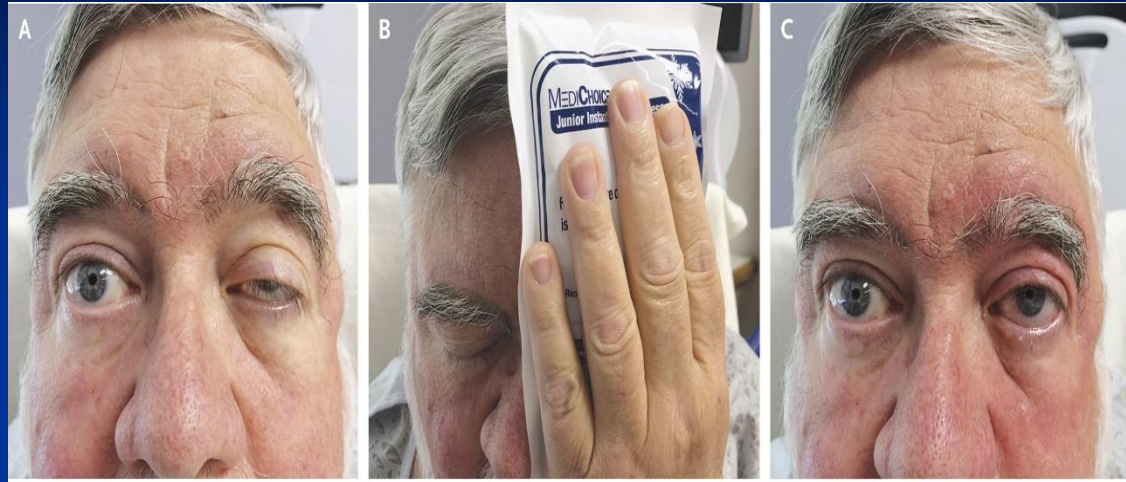
- **Variable**: worsens with physical activity and improves with rest; worse at the end of the day
- **Ocular**-usually asymmetric
 - Ptosis
 - Diplopia/EOM paresis
 - Normal Pupils/No nystagmus
- **Facial**: very common
- **Weak jaw and mastication (Jaw drop)**
- **Bulbar**
 - Dysphagia
 - Dysarthria - weak palate/tongue
 - Dysphonia - vocal cord paralysis
- **Respiratory**
 - Weak diaphragm and intercostals

MG: Weakness-2

- Neck: Extensors weakness (head droop)
- Limb weakness
 - Proximal > distal
 - Arms > legs
 - Usually symmetric
 - Predilection for certain muscles: triceps, finger extensors

MG: investigations

- The orbital Ice Test
- Serum Autoantibodies
 - Anti-Acetylcholine Receptor antibodies
 - Anti-Muscle-Specific Kinase (MuSK) antibodies
- Electrophysiological tests
 - Repetitive Nerve Stimulation
 - Single Fiber EMG
- CT scan of the chest for thymoma or thymic hyperplasia



MG Treatment

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

Thunderclap Headache (TCH)

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



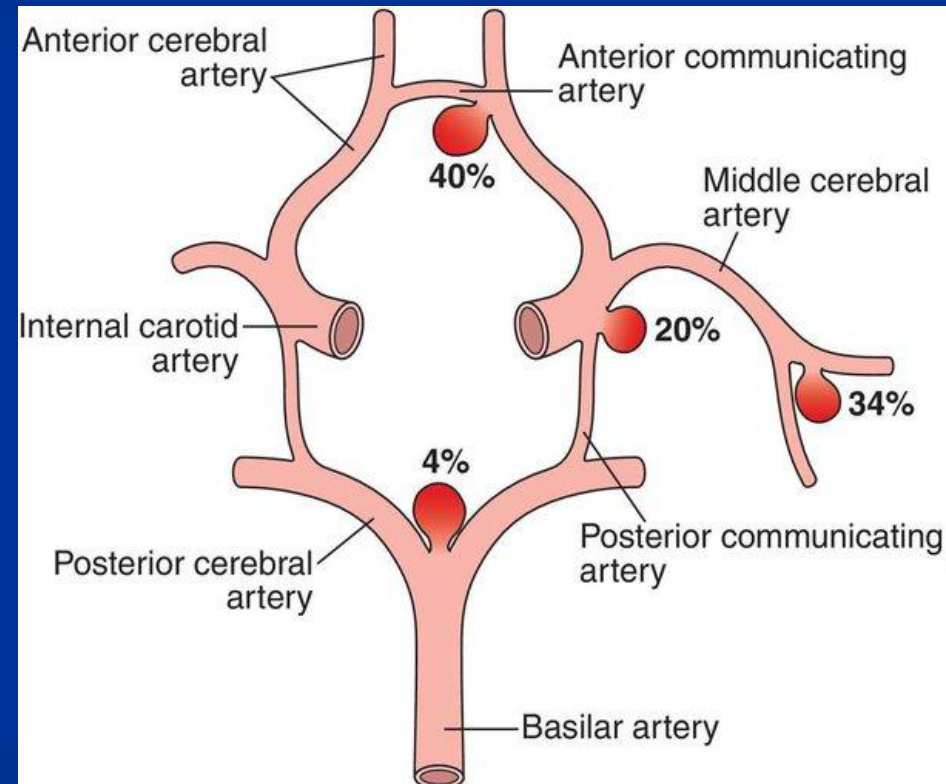
Differential Diagnosis of TCH

- SAH
- Intracerebral intraparenchymal hemorrhage
- Cerebral venous thrombosis
- Arterial dissection
- Reversible cerebral vasoconstriction syndrome (RCVS)
- Pituitary apoplexy
- Spontaneous intracranial hypotension
- Acute obstructive hydrocephalus
- Meningitis /ischemic stroke – rarely
- primary/orgasmic/exertional

SAH: non-traumatic causes

Intracranial berry aneurysms most common

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	

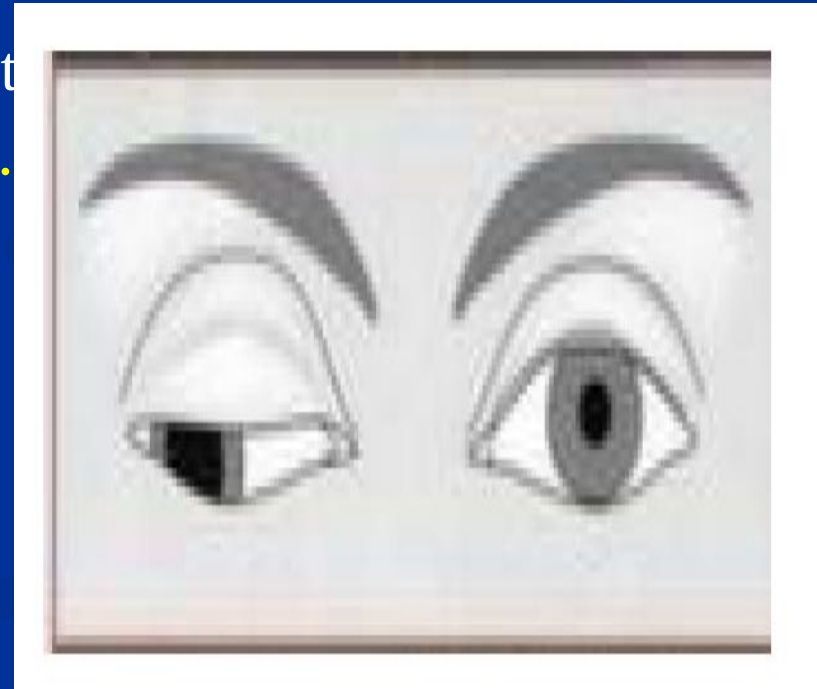


Classic Symptoms of SAH

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

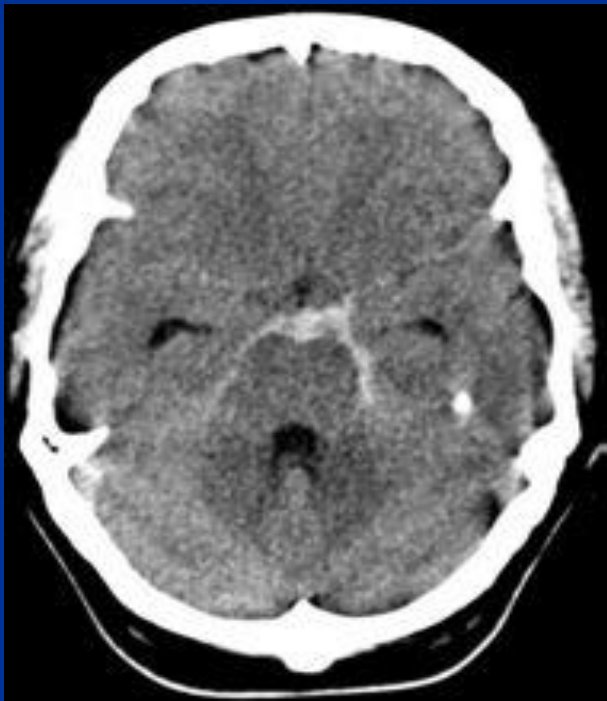
Signs to look for in suspected SAH

- Altered mental status/coma
- Abnormal vital signs
 - Respiratory changes, hypertension, cardiac arrhythmias
- Meningism/photophobia
- Focal neurologic signs may be present
 - 3rd nerve palsy – Int. carotid /post. communicating artery aneurysm
 - Paraparesis – Ant. Cerebral artery aneurysm
 - Hemiparesis, aphasia – Middle cerebral artery aneurysm
- Ocular hemorrhages



Subarachnoid hemorrhage

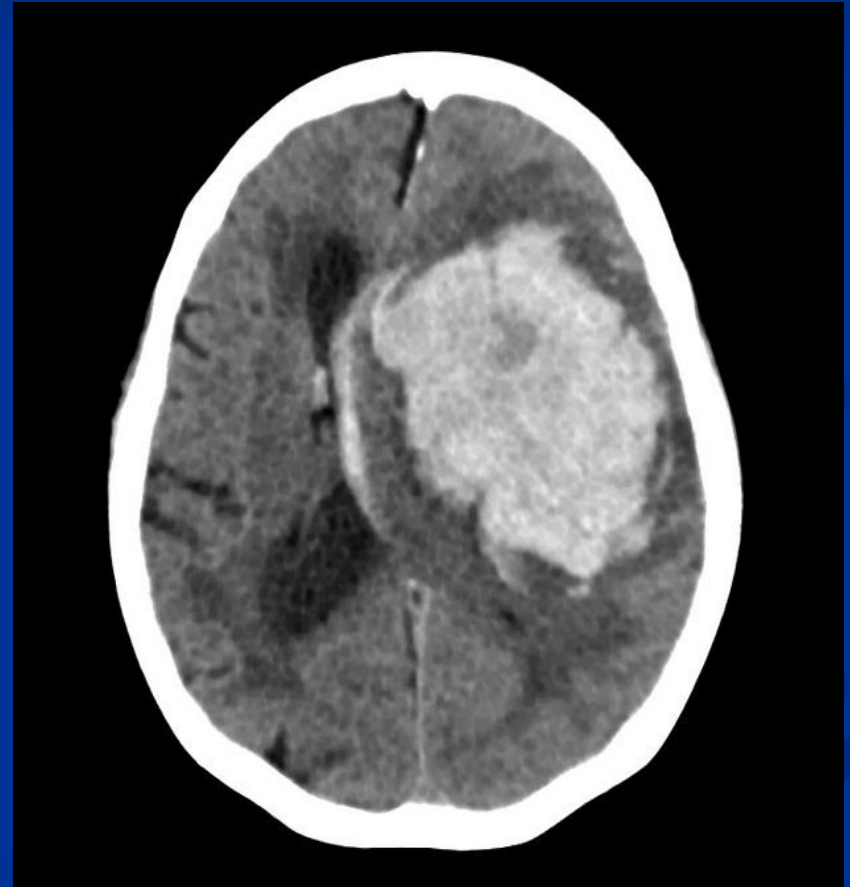
CT not MRI preferred



Subarachnoid hemorrhage



Intracerebral parenchymal hemorrhage



Seizures, Pseudo-seizures and Status Epilepticus

Evaluation of a First Seizure

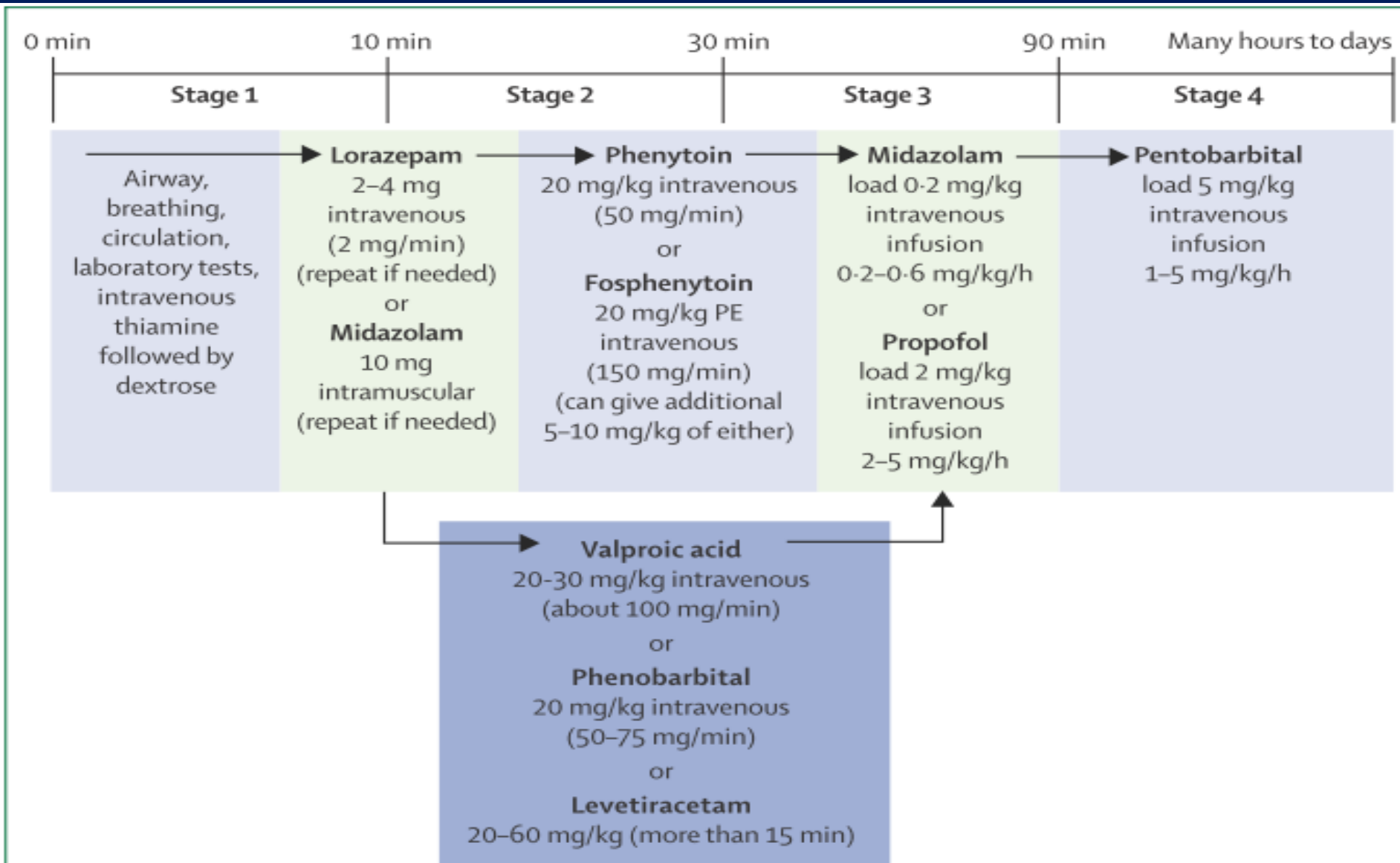
Exclude provoking factors

- ♦ History, physical exam.
- ♦ Blood tests: CBC, electrolytes, glucose, calcium, magnesium, phosphate, hepatic and renal function
- ♦ Lumbar puncture **if meningoencephalitis suspected**
- ♦ Blood or urine screen for drugs
- ♦ **ECG** ; Electroencephalogram (EEG)
- ♦ Brain CT or MR/MRV

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.

Management of Status Epilepticus



Psychogenic Non-Epileptic Status (PNES)

- It is as common as “true” refractory convulsive SE .
- The vast majority of patients with PNES are misdiagnosed and mistreated as refractory SE including the administration of intravenous anaesthetics
- This aggressive treatment may be associated with serious side effects; morbidity and mortality in PNES are exclusively iatrogenic.

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

- The gold standard in discerning the two conditions is ictal EEG and experience in the clinical manifestations of seizures .

Both of which not commonly available off-hours !!



Seizure 2003; **12**: 444–447
doi:10.1016/S1059-1311(03)00052-9

How accurate are witness descriptions of epileptic seizures?

JANNETH B. MANNAN & UDO C. WIESHMANN

The Walton Centre for Neurology and Neurosurgery, University of Liverpool, Liverpool, UK

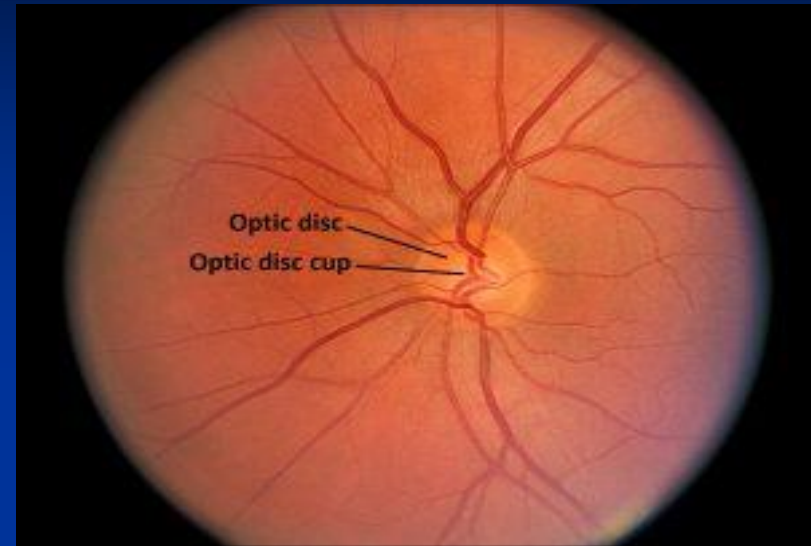
- Significant lateral tongue biting is poorly sensitive but **highly specific (99%)** for a generalized seizure.



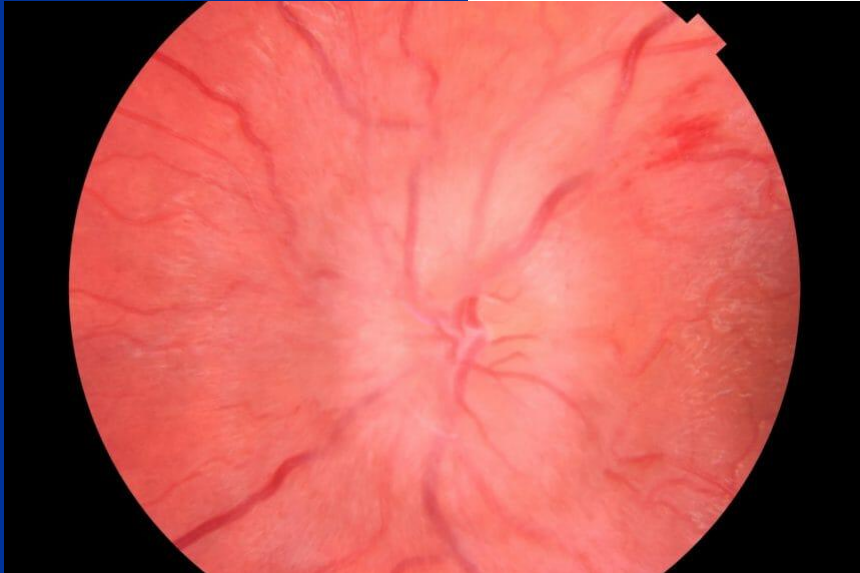
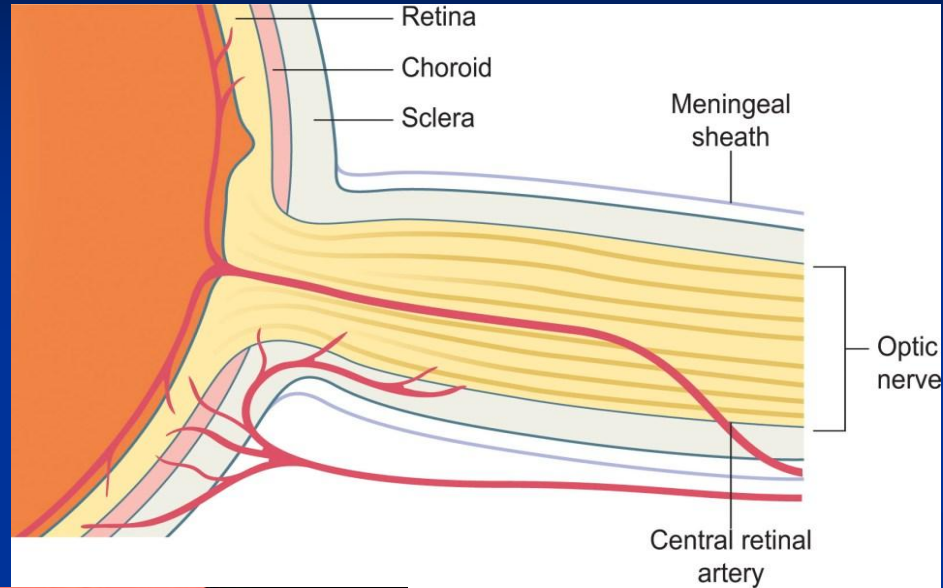
(c) 2012 AMERICAN ACADEMY OF NEUROLOGY - MID4

Neurological causes of sudden loss of vision

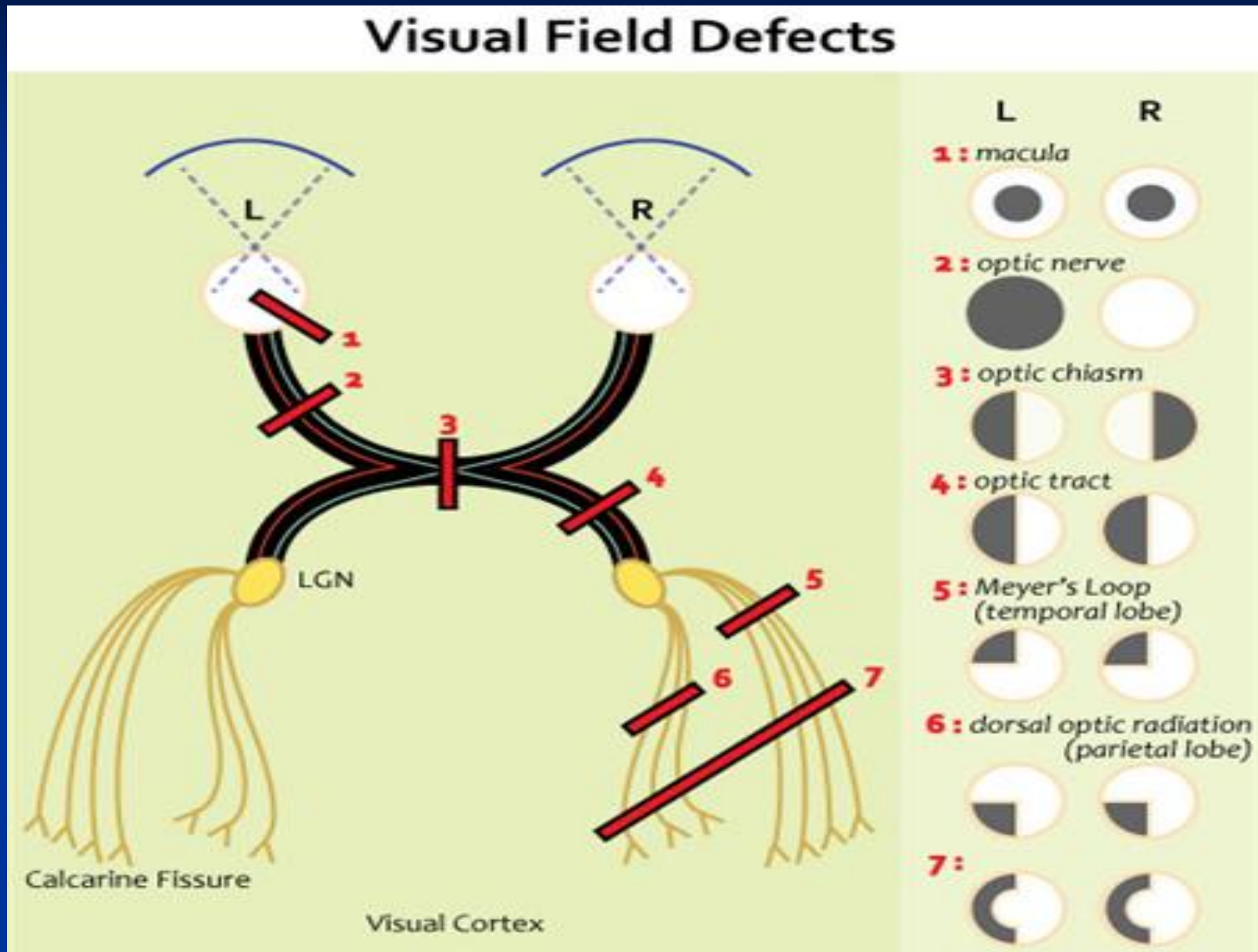
- Optic neuropathy (Demyelination , Ischemia)
- Papilledema/high ICP (Tumor, Idiopathic Intracranial Hypertension)
- Bilateral occipital lobe pathology (infarcts, Posterior Reversible Encephalopathy Syndrome)
- Functional/psychogenic



Papilledema- Optic disc swelling (usually bilateral) due to High ICP



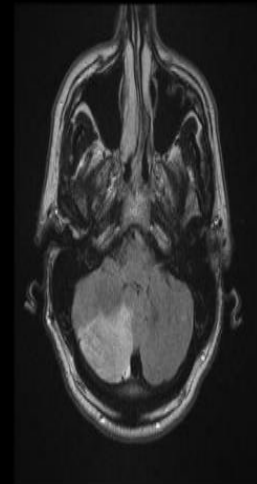
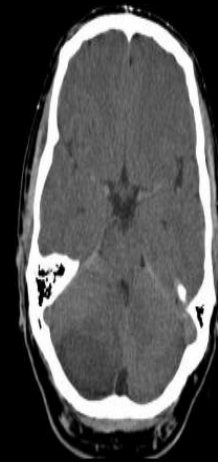
Anatomy of Visual field defects



Vertigo

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

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



WERNICKE ENCEPHALOPATHY

@Neudrawlogy



Acute B1 (thiamine) deficiency

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!

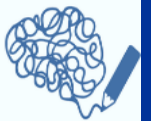
- Chronic alcoholism
- Malignancy
- Hyperemesis of pregnancy
- Bariatric surgery
- Anorexia nervosa

Treatment

**Intravenous thiamin
infusion**

- 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)

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



Questions ?

