Neurological Emergencies

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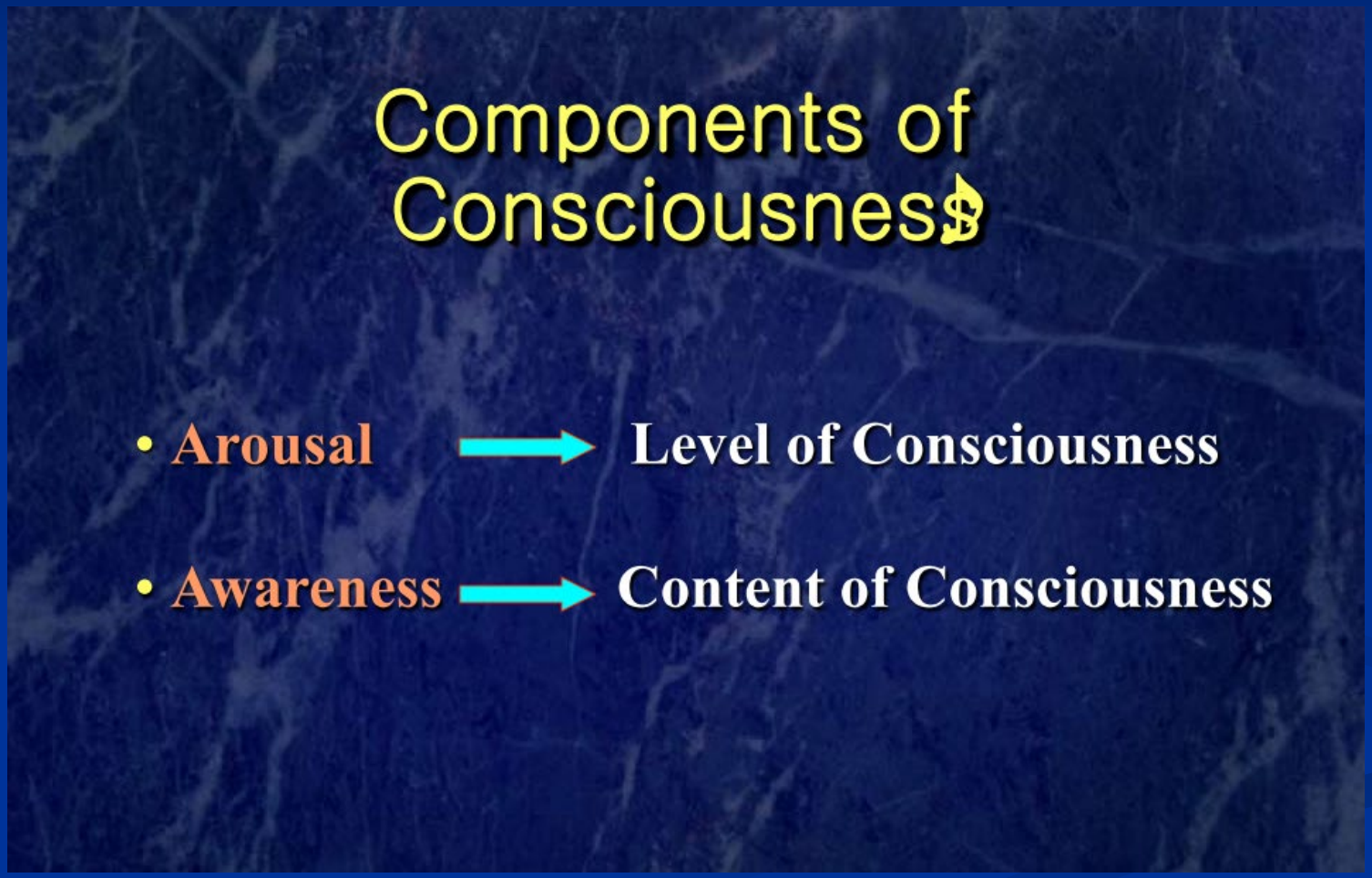
Neurological Emergencies-At least 20% of Medical ER visits

- 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)

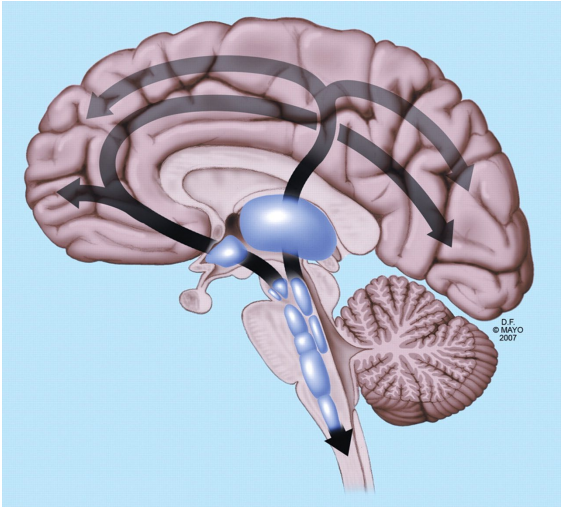
Components of Consciousness



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.



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

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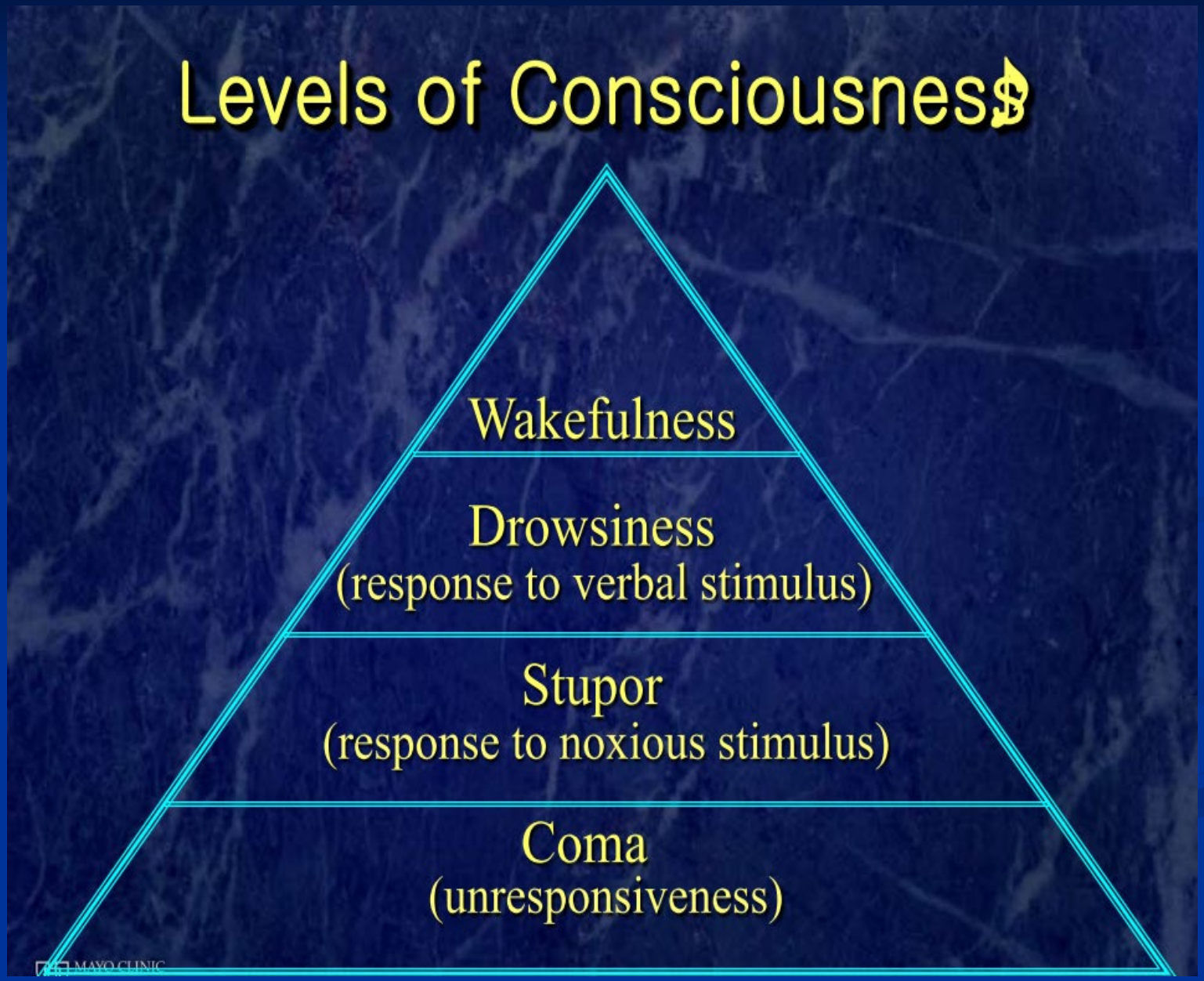
Content of consciousness Terminology

- 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

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

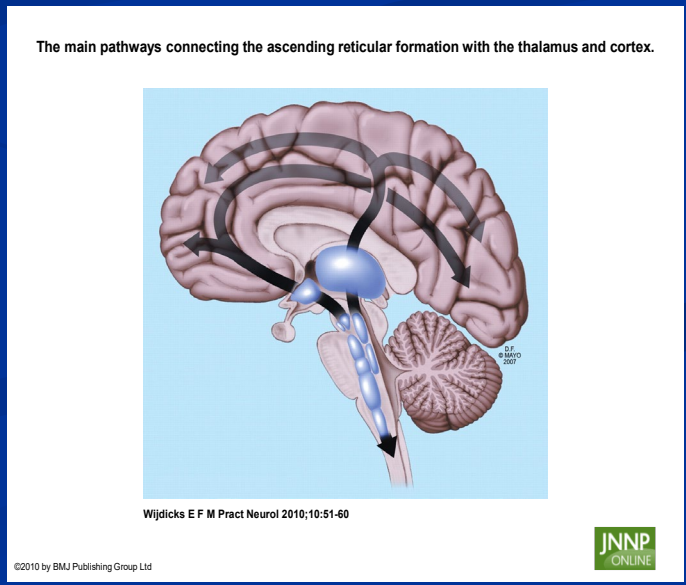
- Intraparenchymal hematoma
- Middle cerebral artery ischemic stroke
- Cerebral venous thrombosis
- Cerebral abscess
- Brain tumor
- Subdural or epidural hematoma

Bilateral

- Subarachnoid hemorrhage
- Traumatic brain injury
- Multiple cerebral infarcts
- Bilateral thalamic infarcts
- Tumors
- Meningitis/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



CAUSES OF COMA WITHOUT FOCAL SIGNS

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

Acute metabolic/endocrine derangement

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

What can mimic coma

1- locked-in syndrome

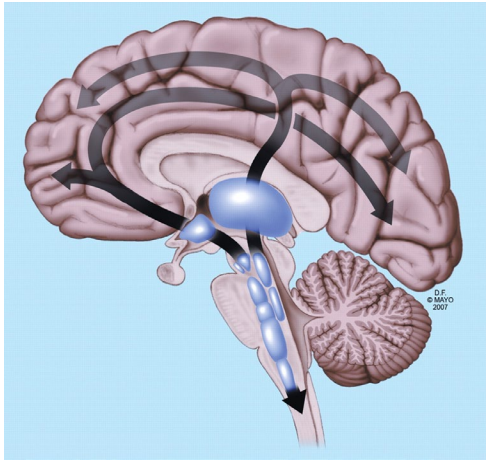
- Eyes open.
- Blink to commands or move their eyes vertically.
- lesion (stroke) in the ventral pons damaging the corticospinal and corticobulbar tracts and sparing the ascending reticular activating system. So they can hear, see and feel pain.

Patients can be intubated by mistake.

2- Psychogenic unresponsiveness

- Hysterical coma
- Malingering
- Acute catatonia

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



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Psychogenic unresponsiveness

Considered after exclusion of other causes !!

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

- Closed eyes which open with tickling the nose hairs

- Others 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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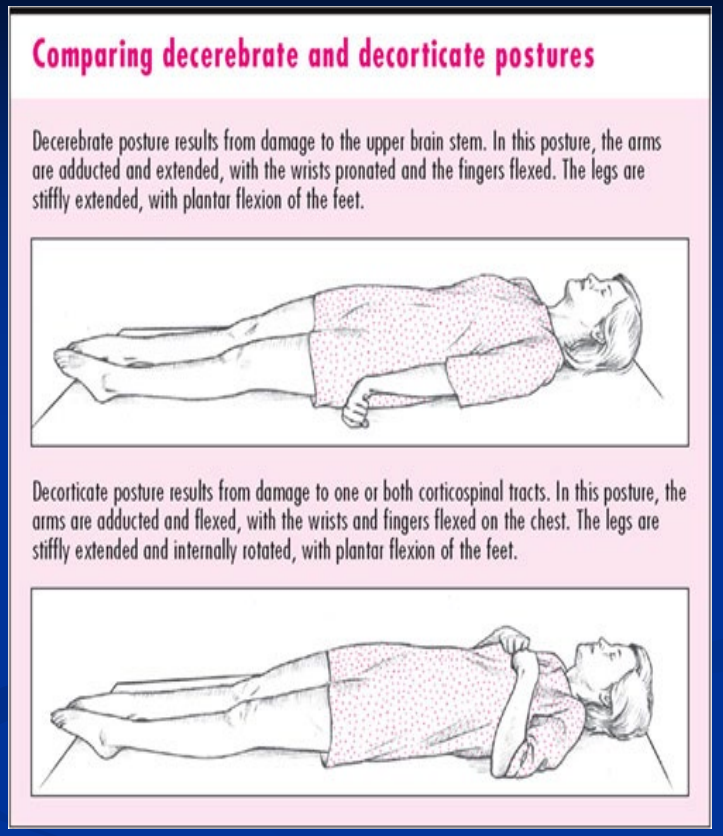


Record subsets:

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.

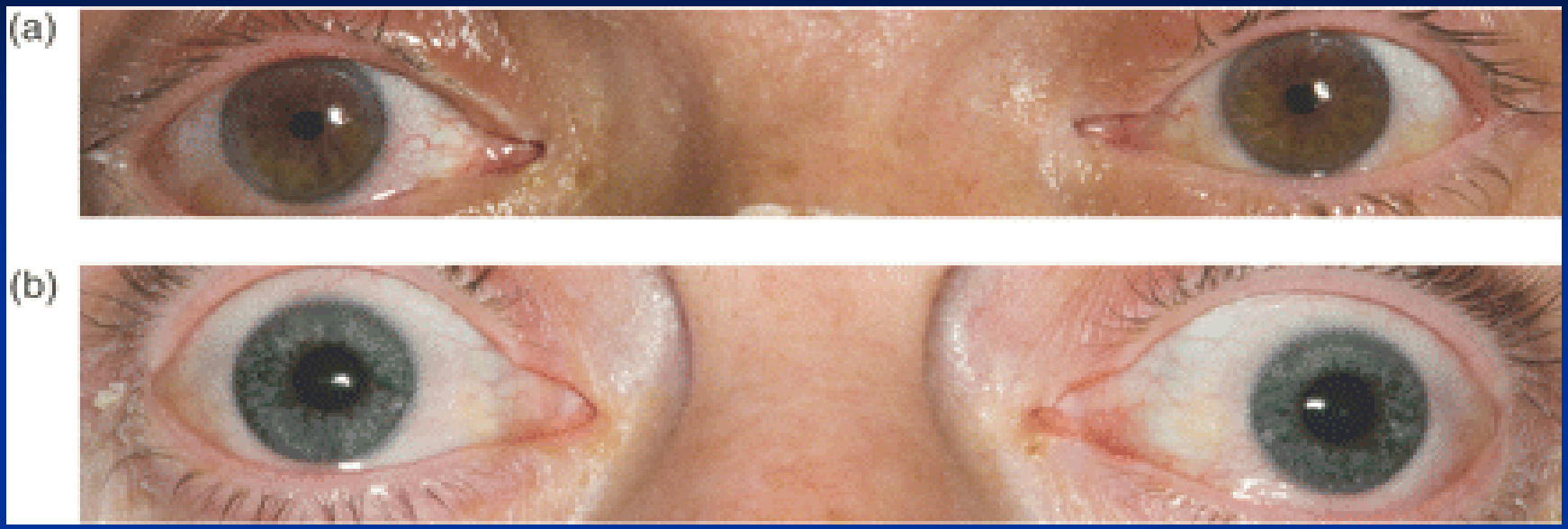
3. Determine the underlying cause if possible.

Examination of the comatose patient

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

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

Lesion Localisation

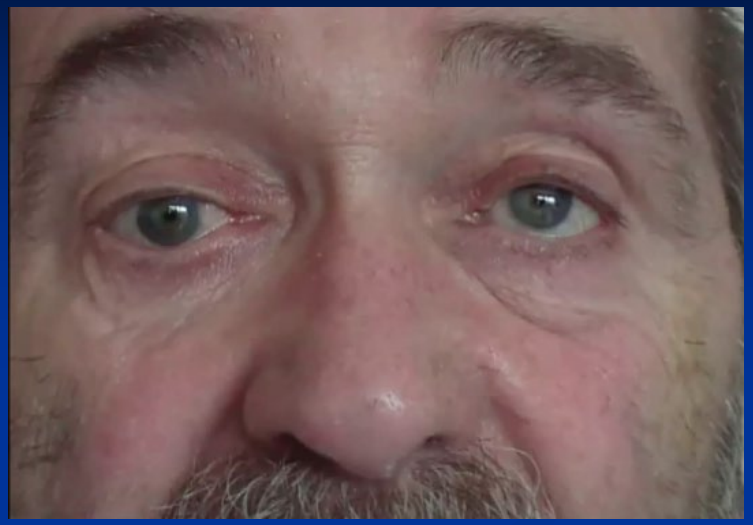


(a) Pinpoint pupils: opioid intoxication or pontine haemorrhage.

(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.



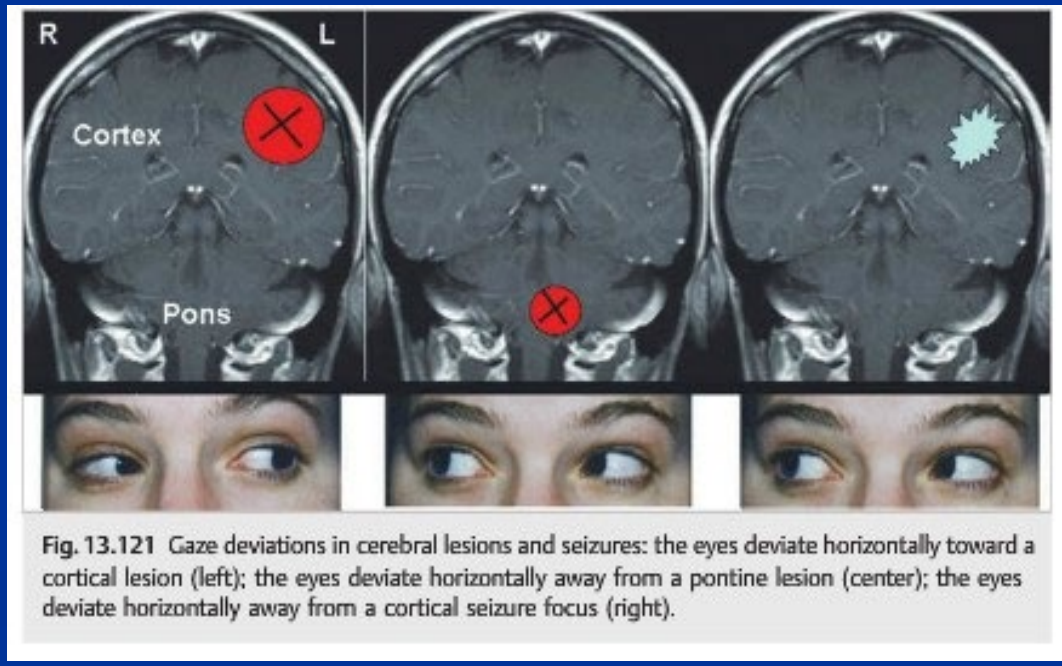


Fig. 13.121 Gaze deviations in cerebral lesions and seizures: the eyes deviate horizontally toward a cortical lesion (left); the eyes deviate horizontally away from a pontine lesion (center); the eyes deviate horizontally away from a cortical seizure focus (right).

Examination of the comatose patient

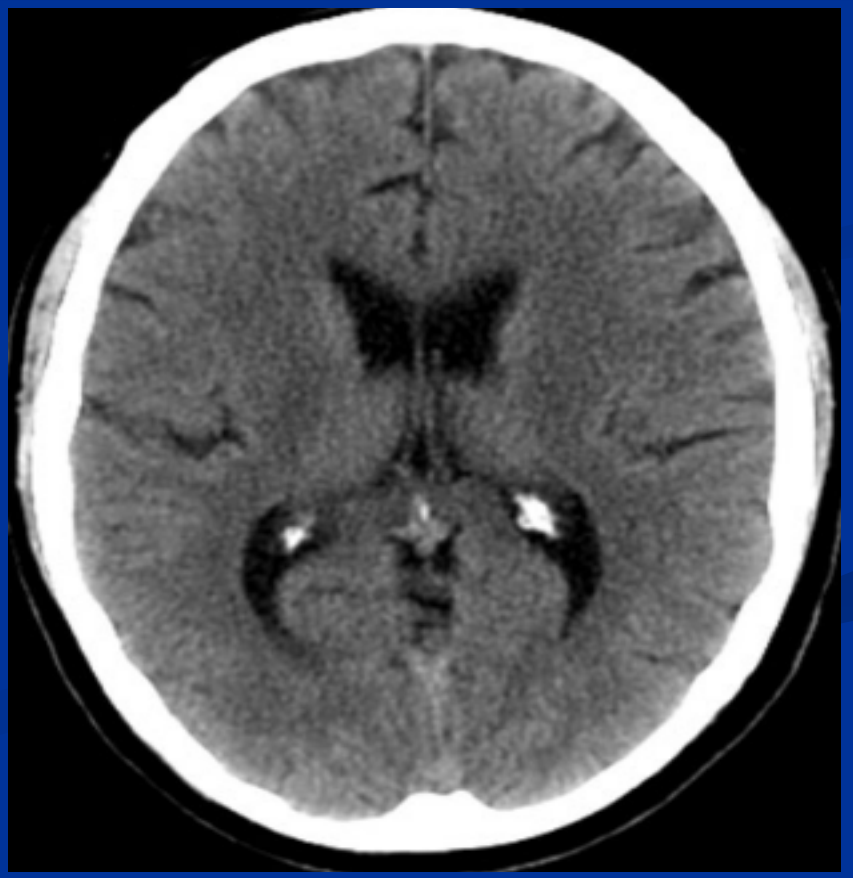
1. Assess the depth of coma.

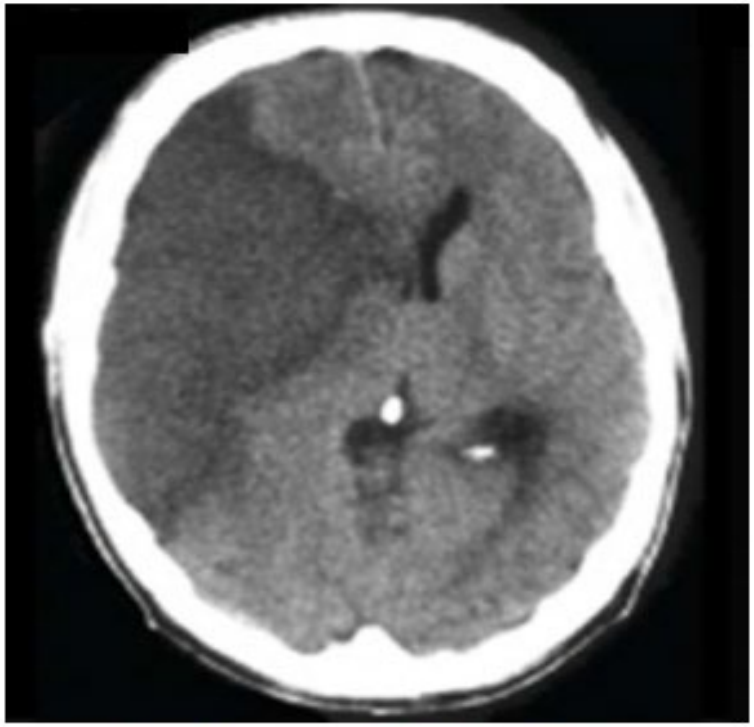
2. Determine if there is structural brain pathology and aim to localize it.

3. Determine the underlying cause if possible.

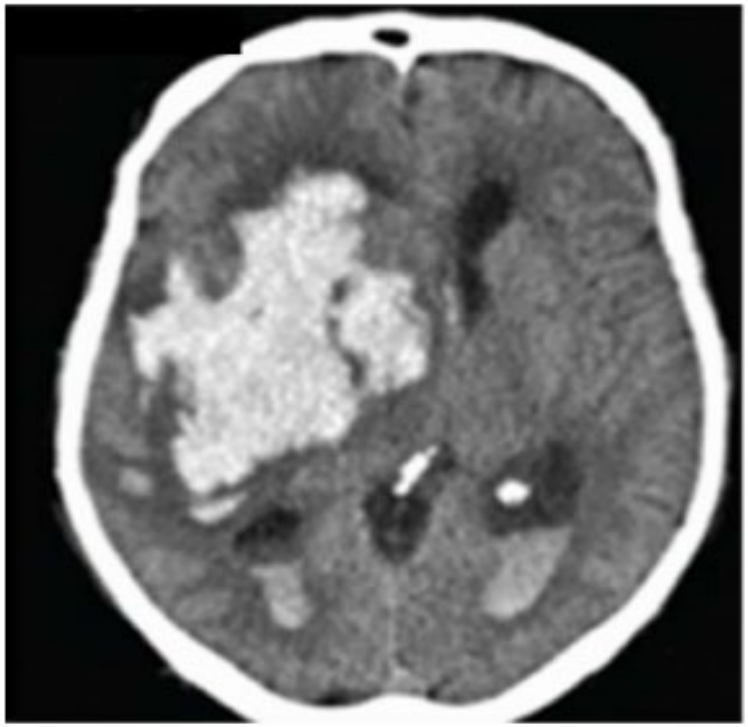
What is the cause of coma ?

- 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.



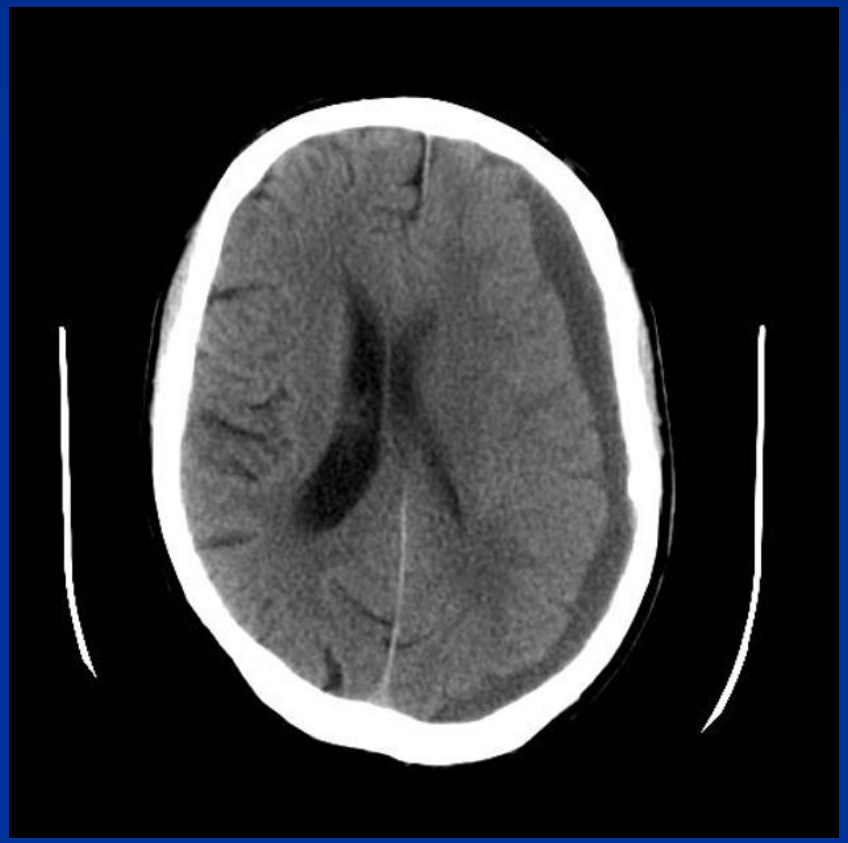


Ischemic Stroke
(dark/hypodense)

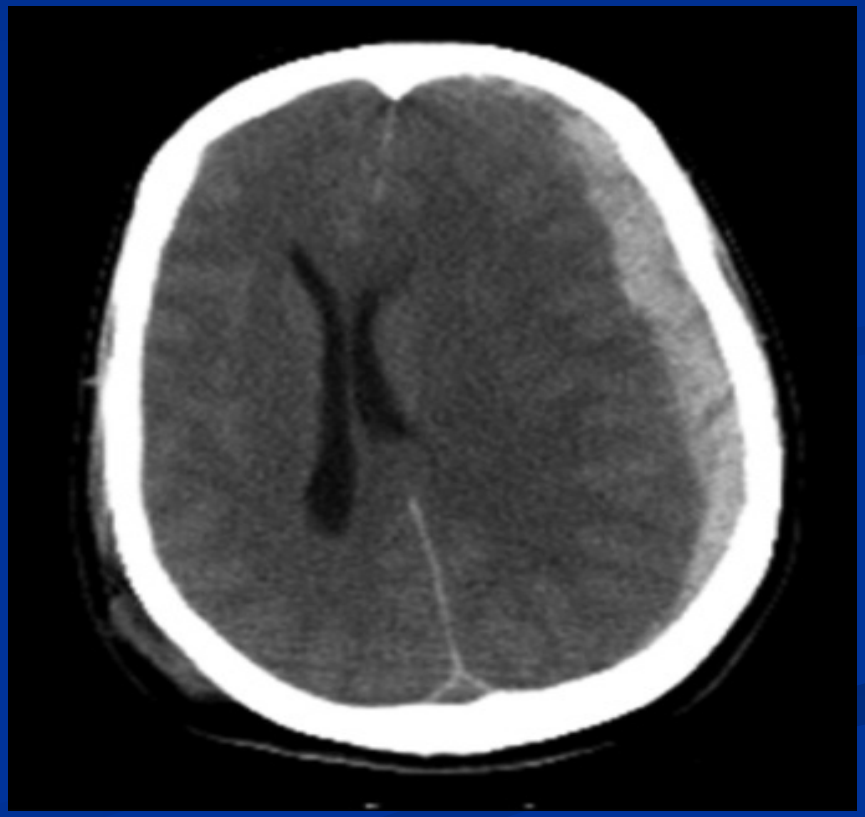


Hemorrhagic Stroke
(bright/hyperdense)

Chronic Subdural Hematoma



Acute Subdural Hematoma



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).
- Nnaloxone 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)

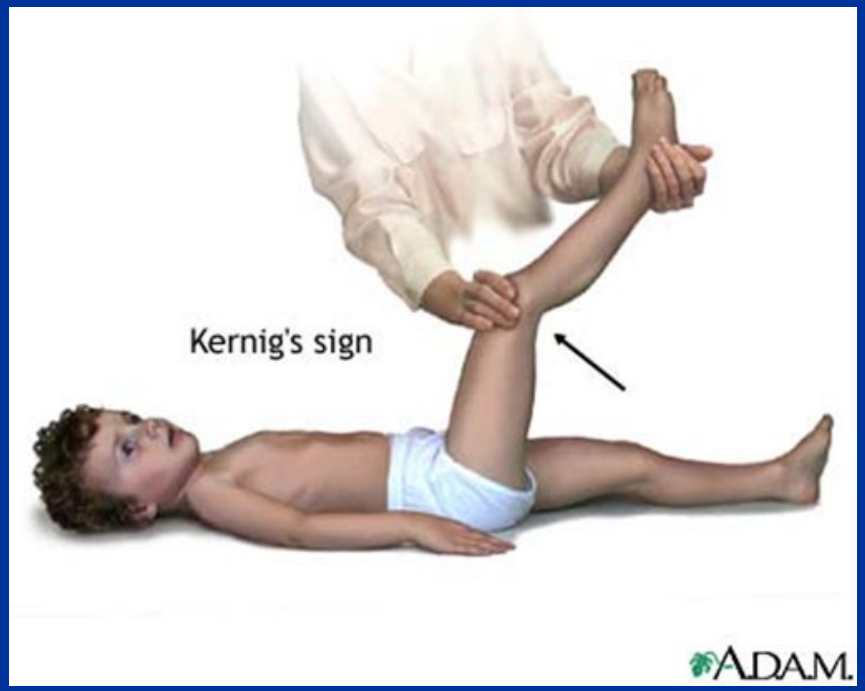
Acute bacterial Meningitis (ABM)

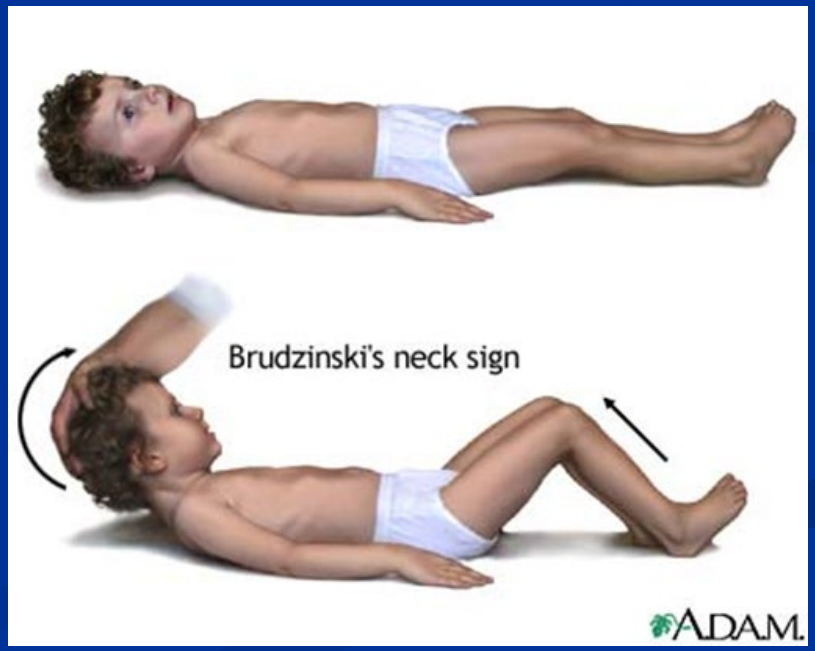
- Common & serious
- Medical emergency
- 100% curable if treated adequately or 100% fatal
- High index of suspicion important
- Dx by CSF examination

ABM : Symptoms and Signs

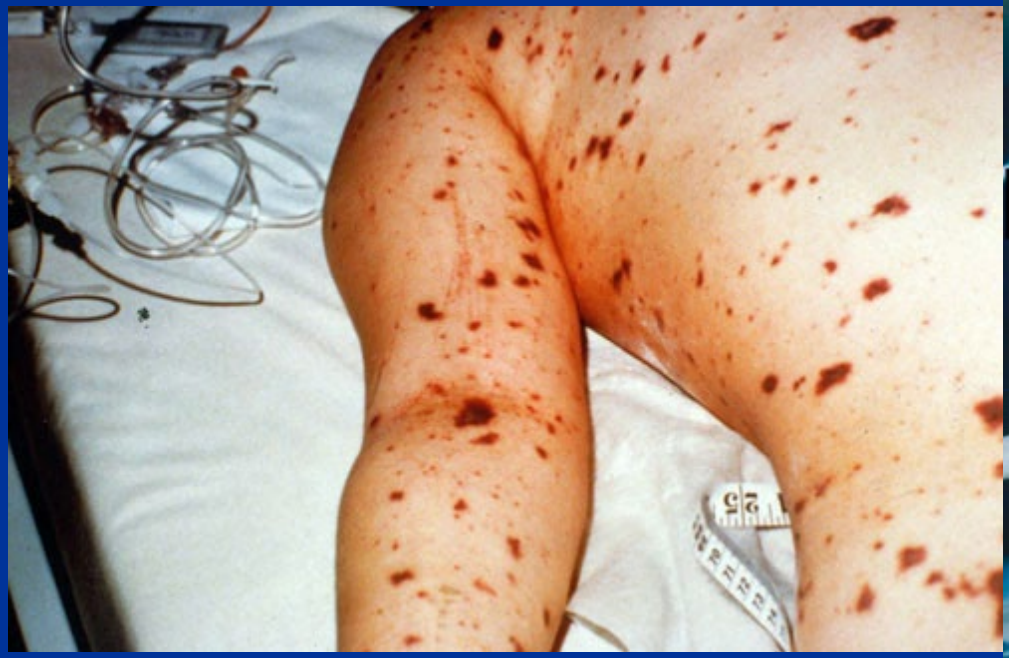
- Early flu-like symptoms
- Worsening headache (+/- Nausea or vomiting)
- Chills/High fever
- Confusion/irritability/difficulty concentrating/ drowsiness/coma
- Seizures
- Nuchal rigidity and other meningeal irritation signs/Photophobia
- Purpuric skin rash (in meningococcal meningitis)
- May develop rapidly over 1-2 days or slowly over many days , esp. in the elderly or immunosuppressed

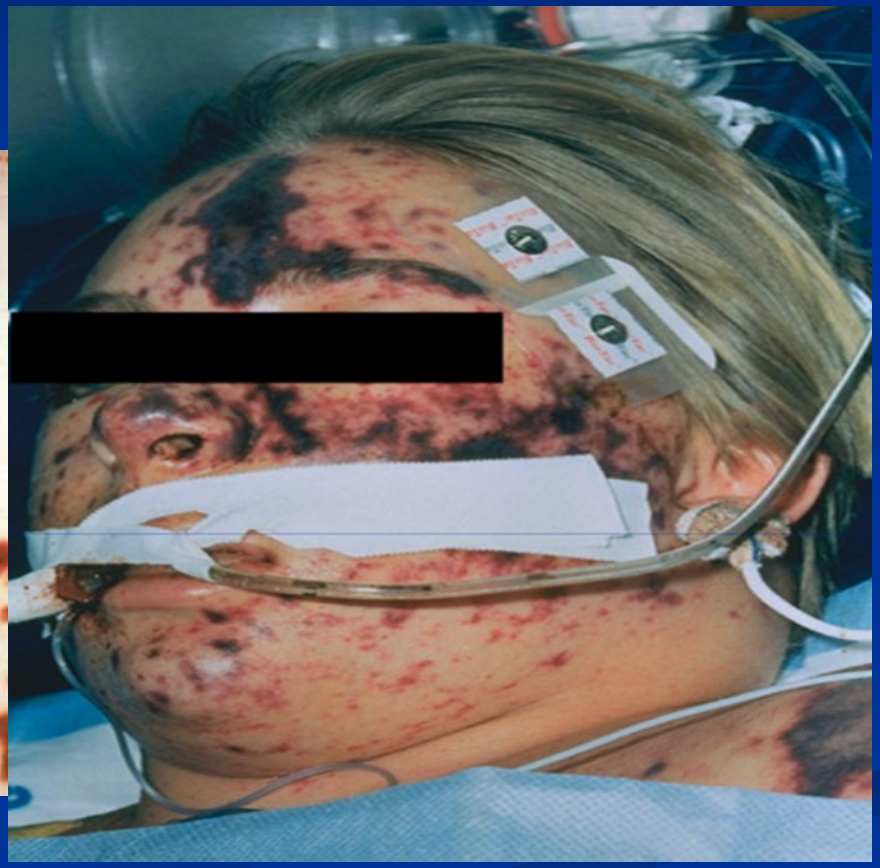
Meningeal irritation signs





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
  - High index of suspicion very important
  - Confirm by CSF examination
  - If LP is contraindicated (clinically or by 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 of 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.

CSF

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

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

Encephalitis

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

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

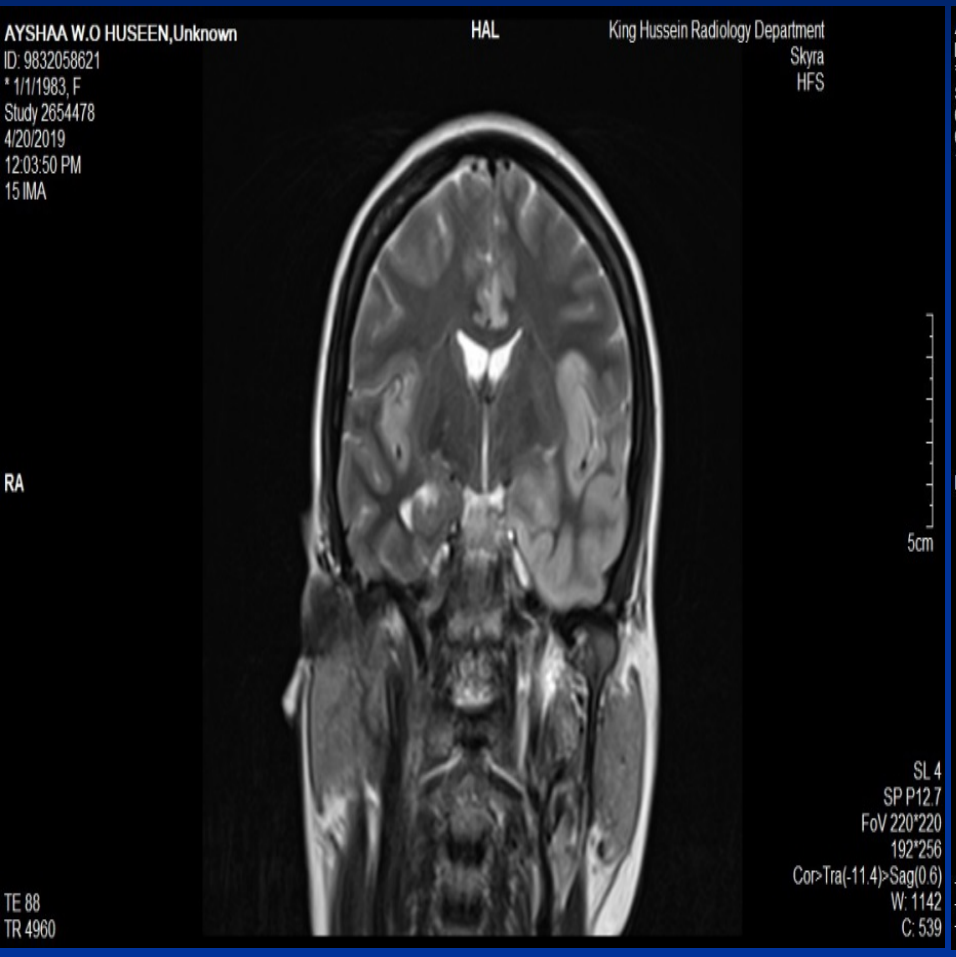
Herpes simplex virus encephalitis

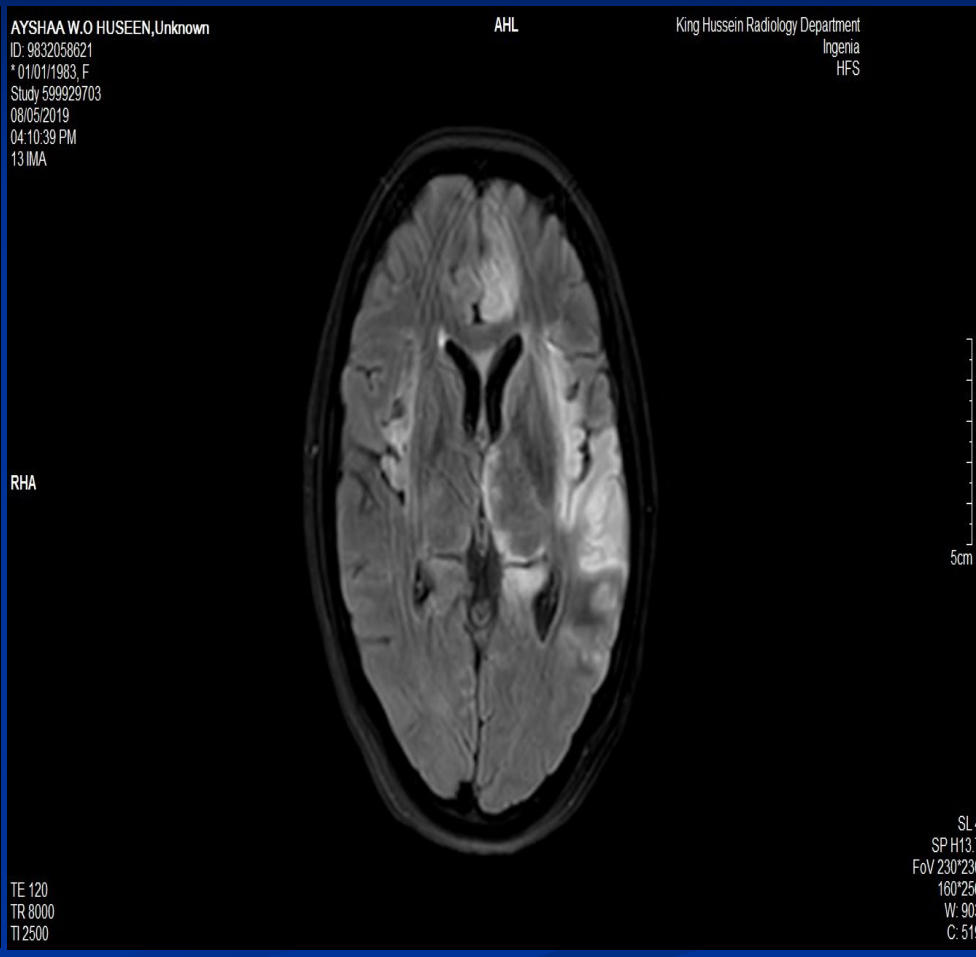
- Most cases are caused by HSV1, but around 10% are caused by type 2.
- The most distinctive presenting features are fever, disorientation, aphasia and behavioural disturbances, and up to a third of patients have convulsive seizures.

HSV Encephalitis

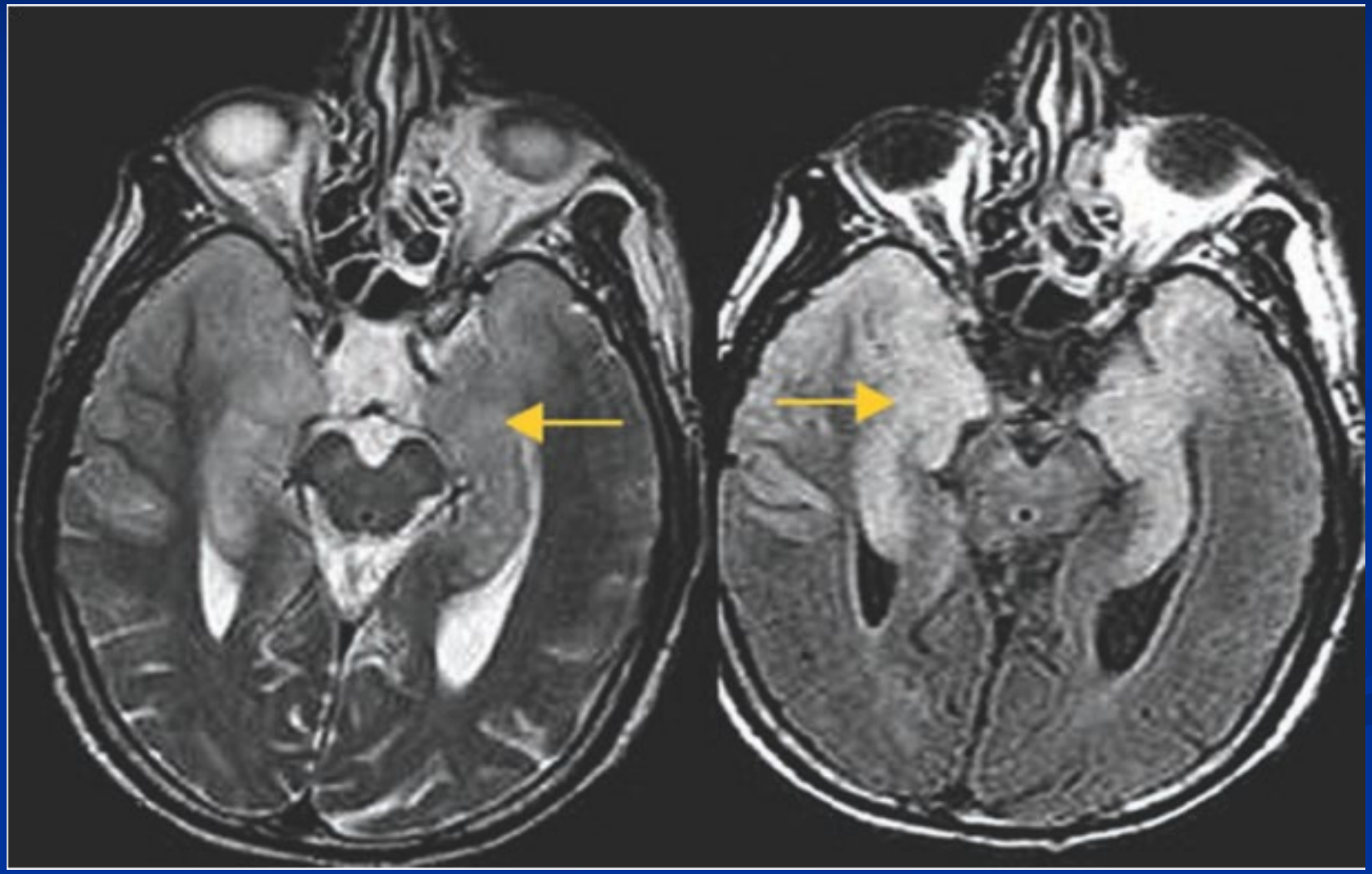
- Neuroimaging can be negative acutely, but by 48 hours, over 90% of patients have MR brain imaging abnormalities and sensitivity approaches 100% at 3–10 days.
- MRI shows markedly asymmetric but usually bilateral abnormalities in the limbic system, medial temporal lobes, insular cortices and inferolateral frontal lobes
- CSF clear, pleocytosis +/-. Normal or ↑protein, normal sugar , PCR (may be negative early- repeat after 24-72 hrs)
- Prompt Rx with IV aciclovir 10 mg/kg x3 if any suspicion

MRI in a case of HSV encephalitis



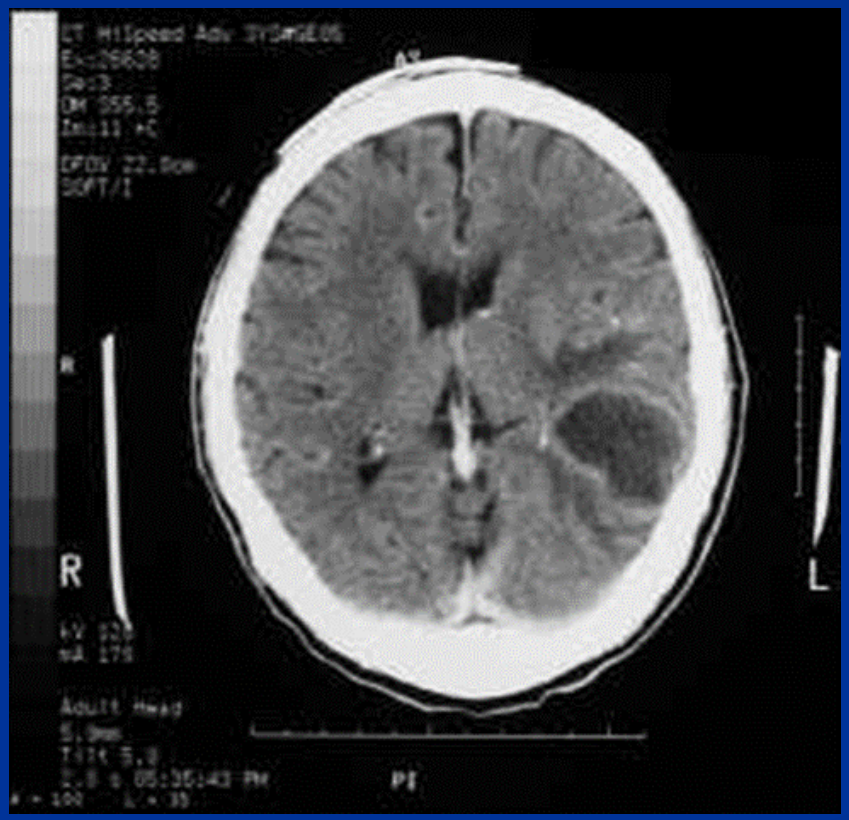


HSV encephalitis

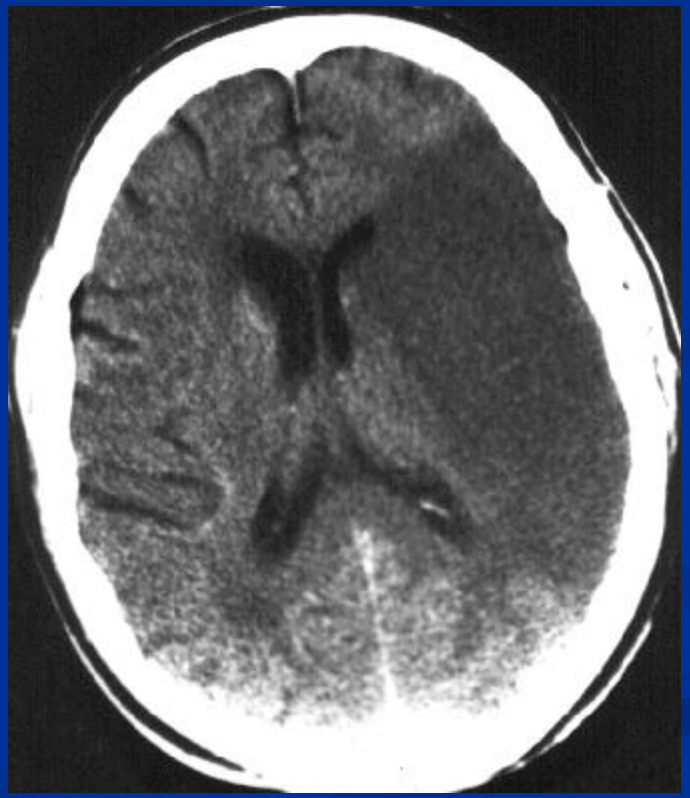


Brain abscess with displacement and hydrocephalus

Brain Abscess



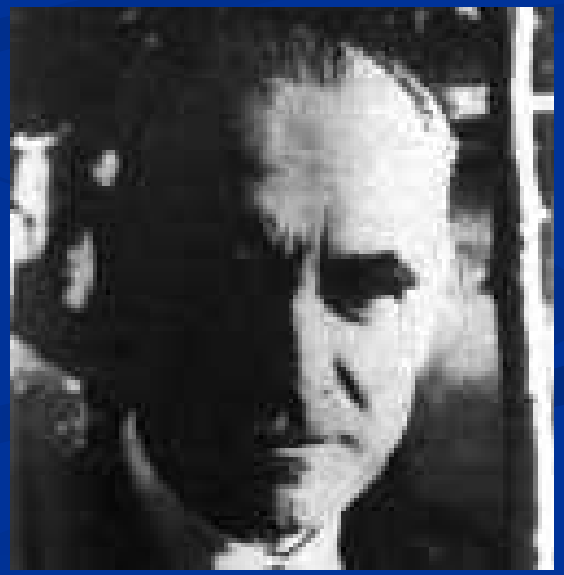
Ischemic Infarct



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.



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 symmetrical "ascending paralysis" in about two thirds of patients, beginning in the lower limbs and then spreading to the upper limbs/trunk/neck/ head. Proximal and distal muscles.

GBS - Clinical Presentation

- Maximal weakness at 1-2 weeks.
- It can affect the facial muscles (50% of cases) , bulbar and respiratory muscles - 25% of patients need artificial ventilation.
- Extraocular muscle weakness /ptosis - uncommon

- DTR - Hypo/Areflexia
- Sensory (mild) and autonomic symptoms.

Differential diagnosis of acute flaccid paralysis

- **Brainstem stroke/ encephalitis**
- **Acute myelopathy**
  - **Spinal cord infarction/haemorrhage**
  - **Acute transverse myelitis**
- **Acute poliomyelitis**
- **Peripheral neuropathy**
  - **Guillain-Barré syndrome**
  - Heavy metals (thallium)
  - **Acute intermittent porphyria**
  - Vasculitic neuropathy
  - Lymphomatous neuropathy

Differential diagnosis of acute flaccid paralysis

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

Investigations

- CSF – Albumino-cytological dissociation
- Nerve Conduction Studies – Demyelinating neuropathy >> Axonal
- Spine MRI and blood work-up (K/P/other electrolytes, Creatine Kinase) to exclude other diagnoses

GBS triggers

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

GBS treatment- Immunotherapy

- Intravenous Immunoglobulins (IVIg) –
  0.4 g/kg daily for 5 days)

  OR
- Plasma exchange (PE)

Supportive Management for possible complications

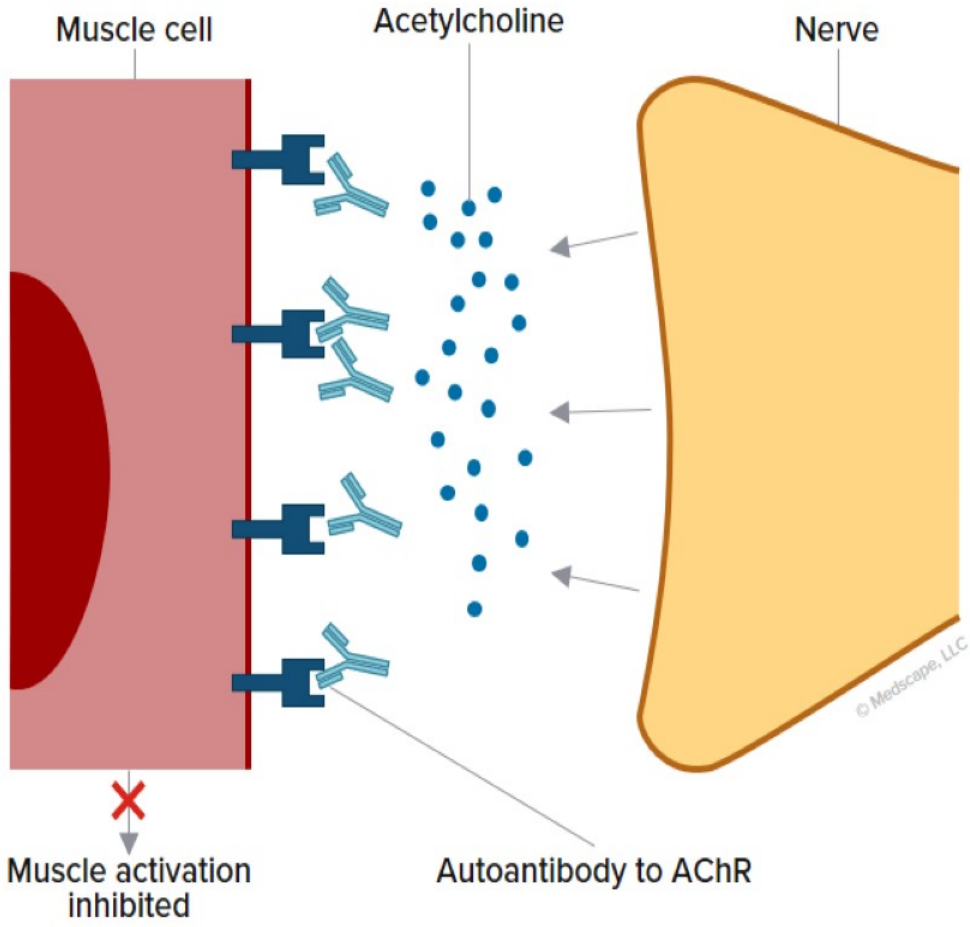
- Careful monitoring of vital capacity (VC) with intubation for those with a VC of <15 ml/kg or which is rapidly dropping
- Twenty five per cent of GBS patients require ventilatory support during their illness.
- Cardiac monitoring for possible arrhythmias throughout the acute stages .
- Venous thromboembolism 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]



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

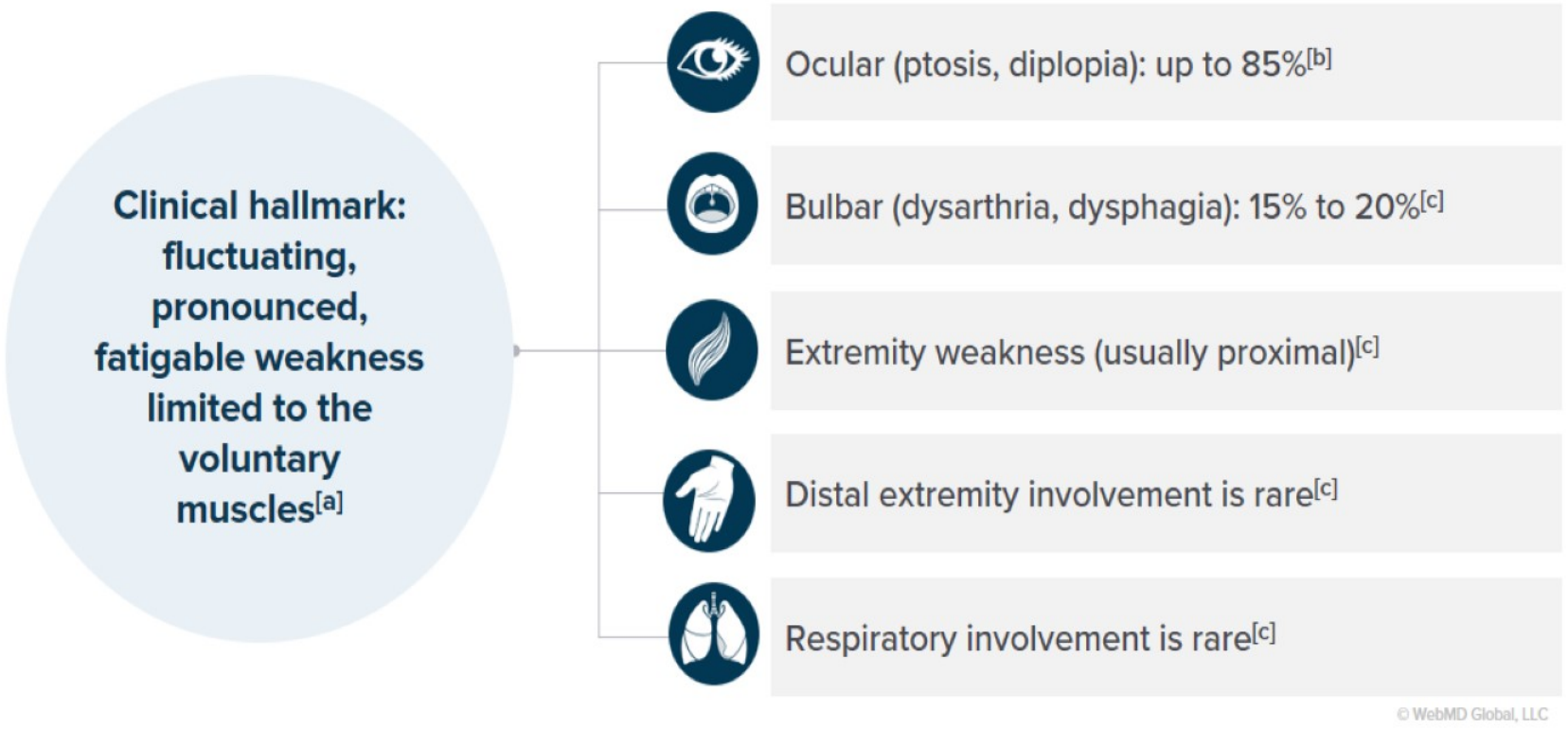
Myasthenia Gravis

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

There are 2 peaks noted in the incidence of MG, one at younger age (2nd and 3rd of life) and another at 6th/7th decade. The later is called late MG

Myasthenia gravis

Clinical Presentation (cont)



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

MG: Weakness-1

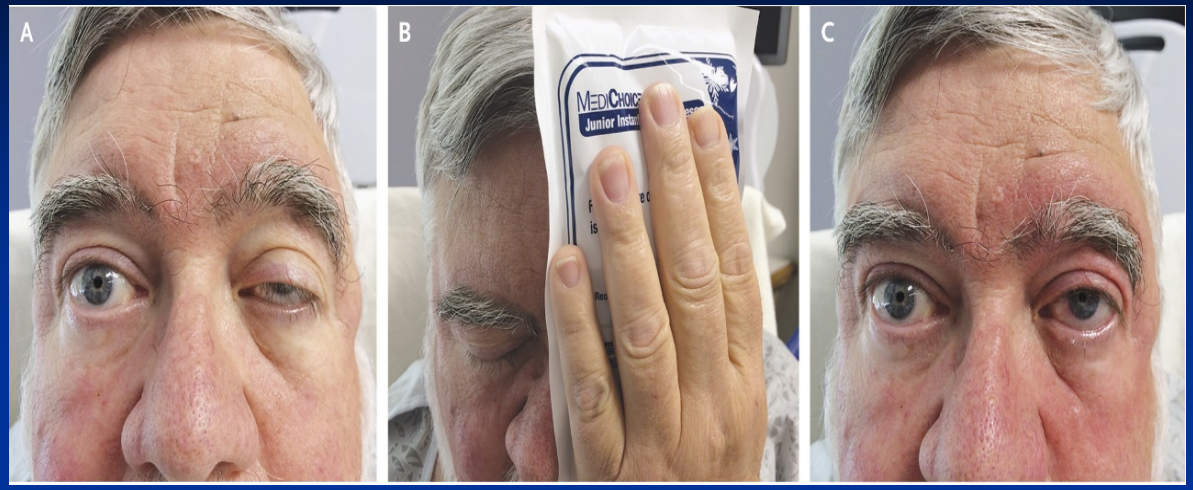
- Variable: worsens with physical activity and improves with rest; worse at the end of the day
- Ocular
  - Ptosis and/or diplopia
  - Usually asymmetric
  - Normal Pupils
- Facial: very common
- Bulbar
  - Dysarthria; weak mastication
  - Abnormal gag reflex; weak palate, tongue
- Respiratory
  - Weak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

Vocal cord paralysis in adductor position leading to stridor. This is an emergency

MG: Weakness-2

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

MG: investigations



- The orbital Ice Test

- 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

More details in the next slide!!!

- Place ice pack over the eye for 2 minutes. An increase in the orbital fissure opening at 2 mm or more is considered positive. This test is 100% specific with a sensitivity of 90% compared to 50% for the **rest test** (Kubis et al. Ophthalmology 2000;107:1995) and (Palace et al Curr Opin Neuro 2001;14:583-589)

- **Muscle specific kinase (MuSK)** is a receptor tyrosine kinase that is expressed during early muscle development and is crucial for the normal formation of the neuromuscular junction. During early development, agarin is released from the motor nerve and interacts indirectly with MuSK. This leads to a sequence of events that are not fully understood that leads to AChR phosphorylation and clustering. Mice lacking MuSK or agarin do not survive after birth. Antibodies bind to the extracellular domain of MuSK at the neuromuscular junction. The role that MuSK plays in the adult neuromuscular junction is not clear. MuSK antibodies occur in 50% of seronegative MG. They are not associated with thymoma and thymectomy is not indicated in these patients.

- **Antibodies** to muscle antigens (**anti-striational antibodies**): Through the work of Mygland and Romi and colleagues, we know that the anti-striational antibodies are in most of cases directed against Ryonidine receptor and Titin. If antibodies against both antigens are present, then it has a 70% sensitivity and 70% specificity for thymoma.

- RNS: should be done in a weak, proximal and warm muscle. Sensitivity is around 70%. The deltoid may be slightly more sensitive than the trapezius. In our laboratory we do RNS at 2 Hz at the orbicularis oculi and the upper trapezius.

- SFEMG: reported to be 100% in a weak muscle; SFEMG of the frontalis is reported to be as sensitive as the orbicularis oculi; however, EMG of the frontalis is more convenient to do.

- There are patients in whom all the above tests are negative and the diagnosis may remain in doubt. One does not want to miss a diagnosis that may mimic myasthenia (chronic fatigue syndrome, CNS tumors, MS, Strokes, latent strabismus, LEMS). Another decision is whether to treat the patient with immunosuppressive drugs that are potentially harmful to the patient.

- Also consider the following:

  - Patients may be treated with steroids and a PPD test may be necessary before starting steroids

  - IgA level should be done as deficiency of IgA occurs in 1:1000. In patients treated with IVIG, one needs to rule out this possibility before patients are treated with IVIG

MG Treatment

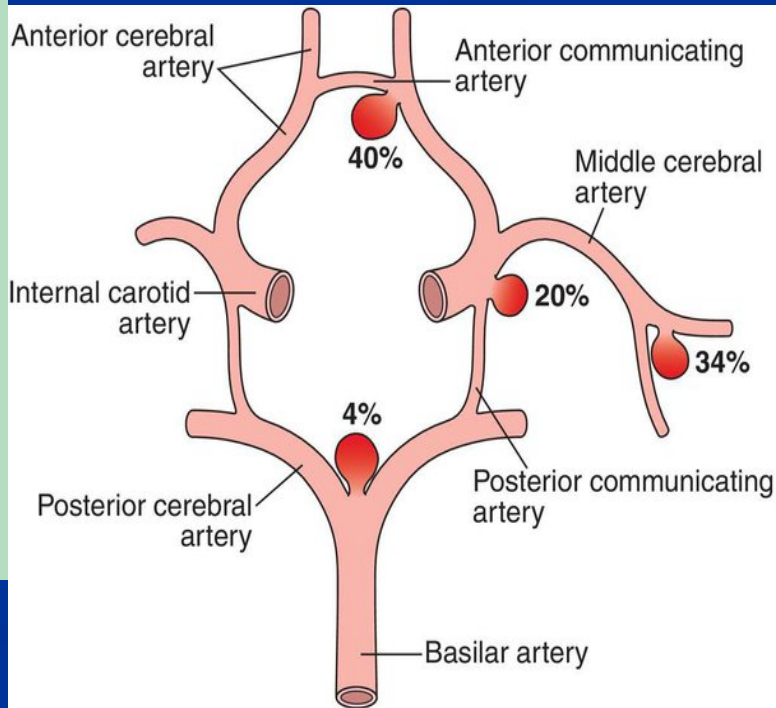
- Myasthenic crisis Rx (IVIg or PE)
- Long-term Rx
  - Oral steroids/immunosuppressives
  - Acetylcholinesterase inhibitors
  - Thymectomy
  - Rituximab and new monoclonals

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 (SAH)

SAH: non-traumatic causes

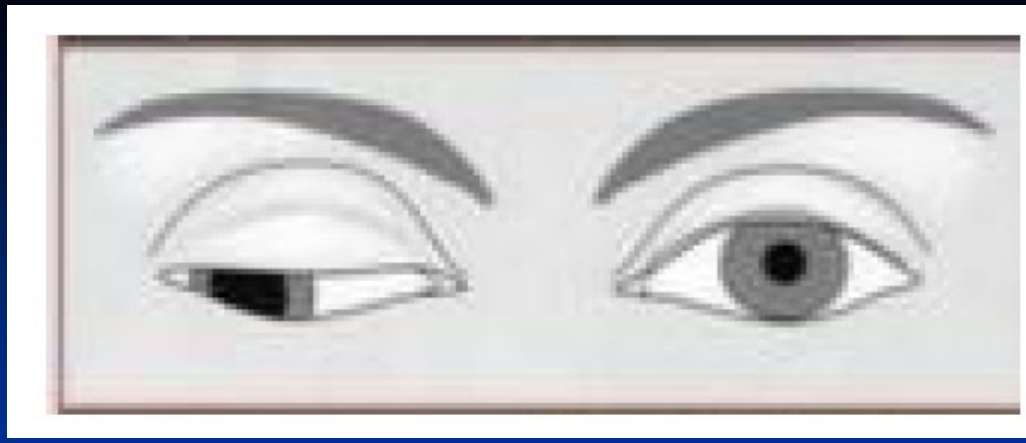
Intracranial aneurysms: degenerative	60–70%
Peri-mesencephalic haemorrhages	15–20%
Arteriovenous malformations and associated aneurysms	5–10%
Other causes:	~ 5%
Dural fistula	
Venous vascular abnormalities	
Spinal arteriovenous malformations	
Cerebral artery dissections	
Moyamoya syndrome	
Vasculopathies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



Classic Symptoms of SAH

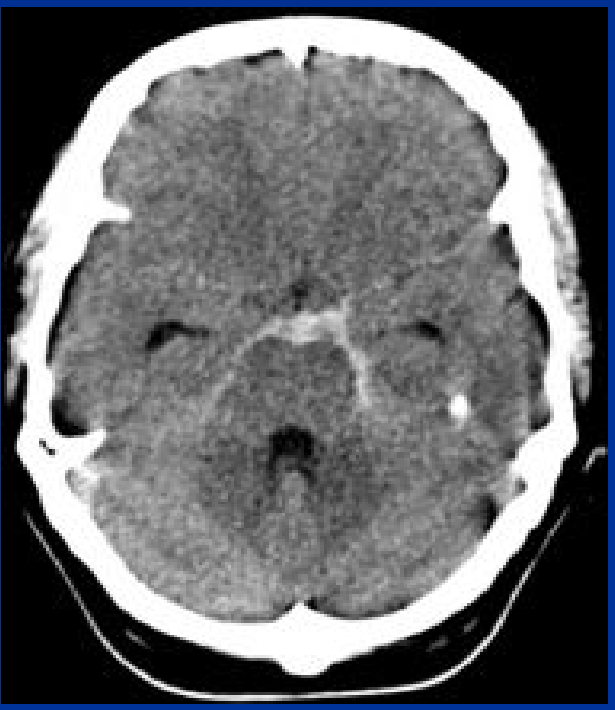
- Sudden, unusually severe or "thunderclap" headache
- Loss of consciousness (transient or persistent if severe)
- Pain in neck, back, eye
- Nausea, vomiting, photophobia

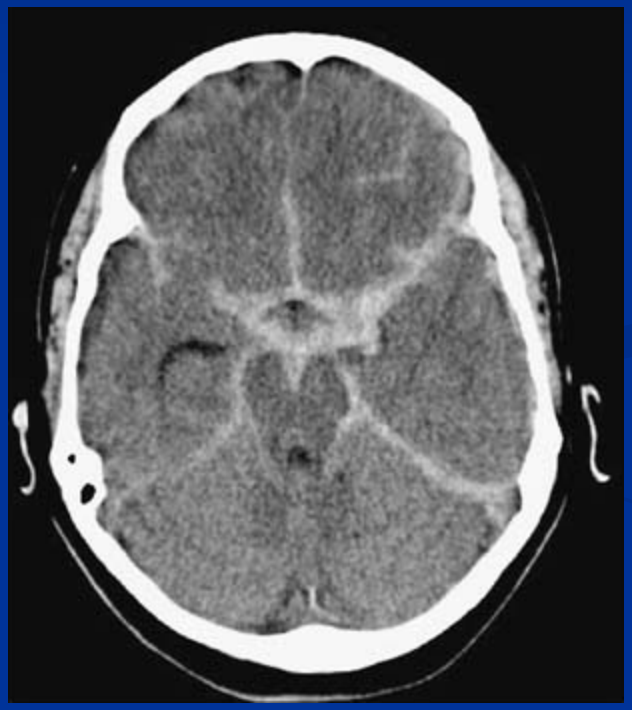
Classic Signs of SAH

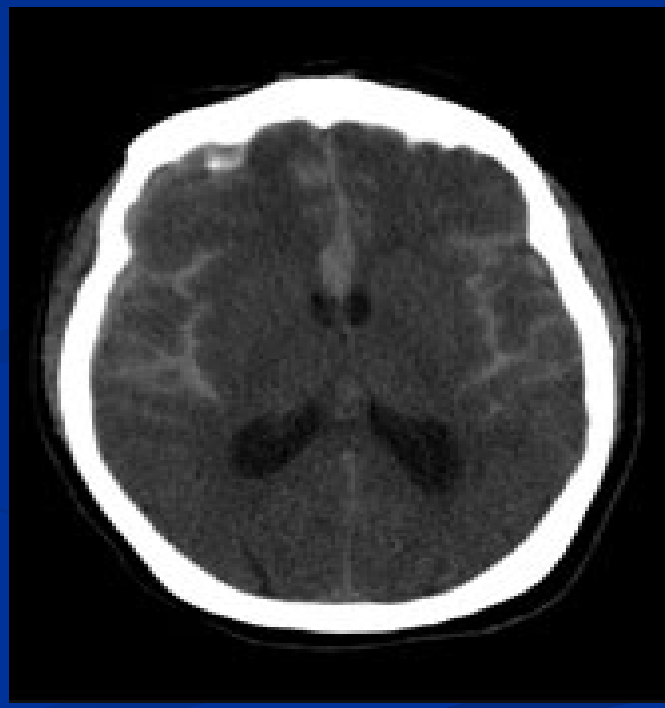


- Abnormal vital signs
  - Respiratory changes, hypertension, cardiac arrhythmias
- Meningism/photophobia
- Focal neurologic signs may be present
  - III nerve palsy – Int. carotid /post. communicating artery aneurysm
  - Paraparesis – Ant. Cerebral artery aneurysm
  - Hemiparesis, aphasia – Middle cerebral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

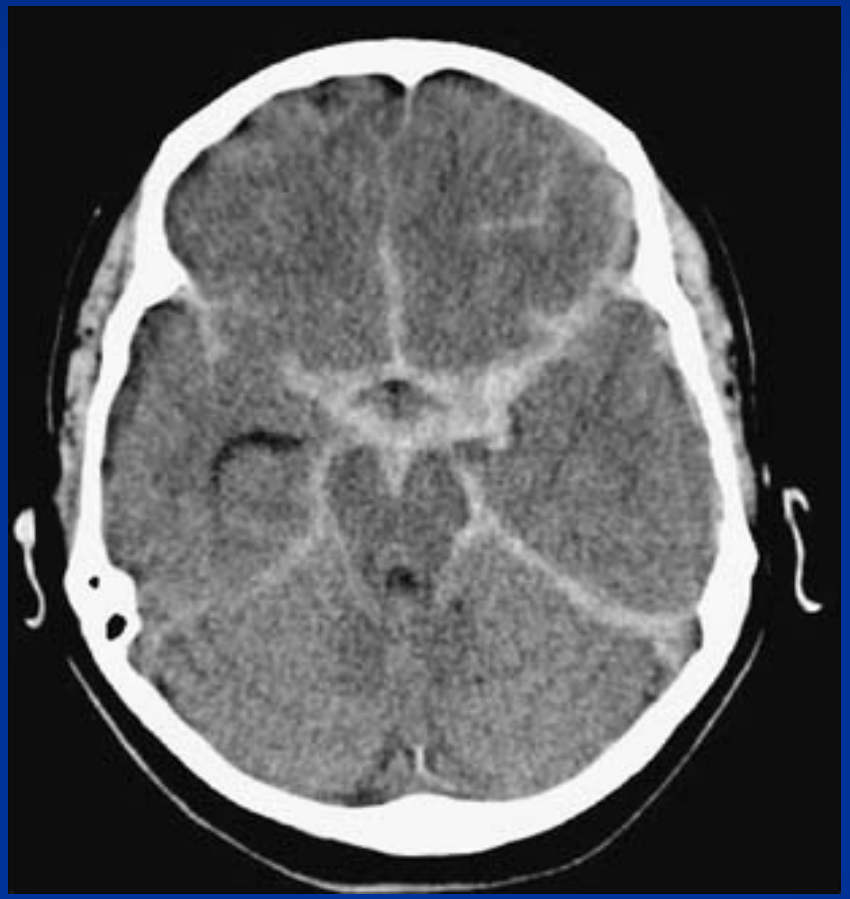
SAH



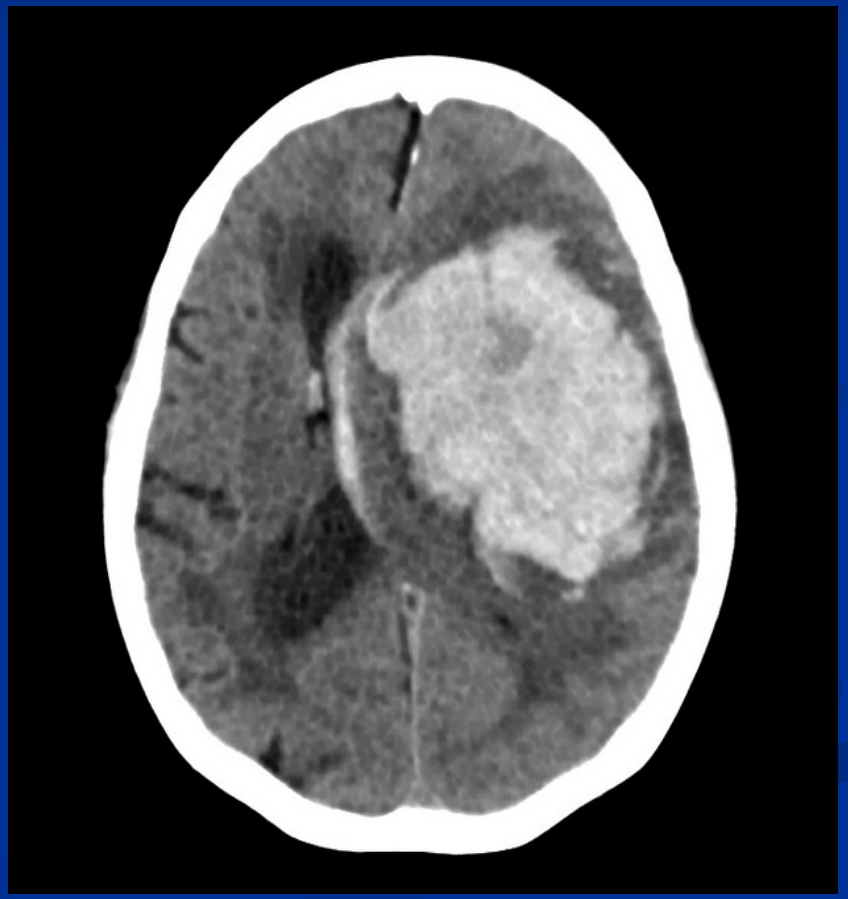




Subarachnoid hemorrhage



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

  (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

Often, the patient is amnestic for the events surrounding the seizure, and the description must be obtained from relatives, friends, or bystanders. Observers may report behavior consistent with a complex partial seizure immediately preceding a convulsion. In other cases, the patient may recall localized motor activity, suggesting a simple partial motor seizure before losing consciousness. At times, the only evidence of partial onset may be a brief subjective event consistent with an aura; in this case it is important to determine whether the identical aura ever occurred before.

The examination of the patient who has experienced a seizure is often most revealing when conducted as soon after the seizure as possible, and should be frequently repeated to determine whether or not any observed deficits are transient. Post-ictal weakness, aphasia, or sensory dysfunction provide powerful lateralizing and sometimes localizing information. Upper motor neuron signs which are briefly present post-ictally (e.g., a transient unilateral Babinski sign) also provide important data. Signs which are not transient may indicate a pre-existing structural lesion (e.g., tumor) or a new condition (e.g., stroke), and may lead to the diagnosis of an acute symptomatic seizure, that is, a seizure resulting from a new brain insult, which does not necessarily imply the existence of epilepsy (although epilepsy may later develop).

Continued on next page

Definition of Status Epilepticus

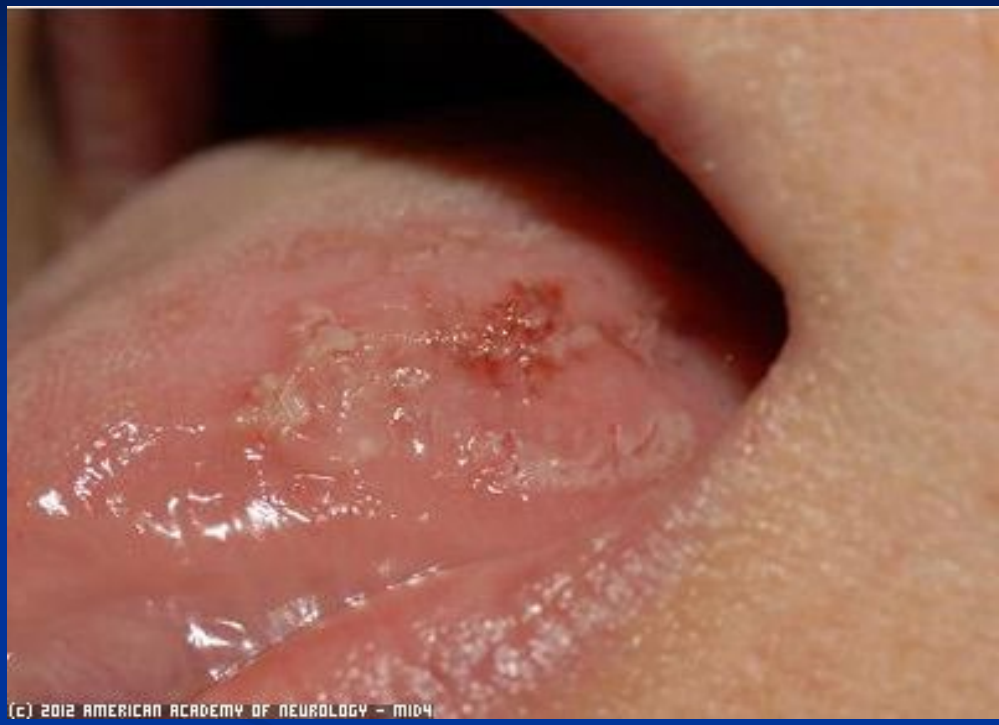
- If the patient has a prolonged (>5 min.) seizure or repetitive (3 or more/hr) seizures without recovery between episodes, they are considered to be in SE and the Rx protocol initiated.

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

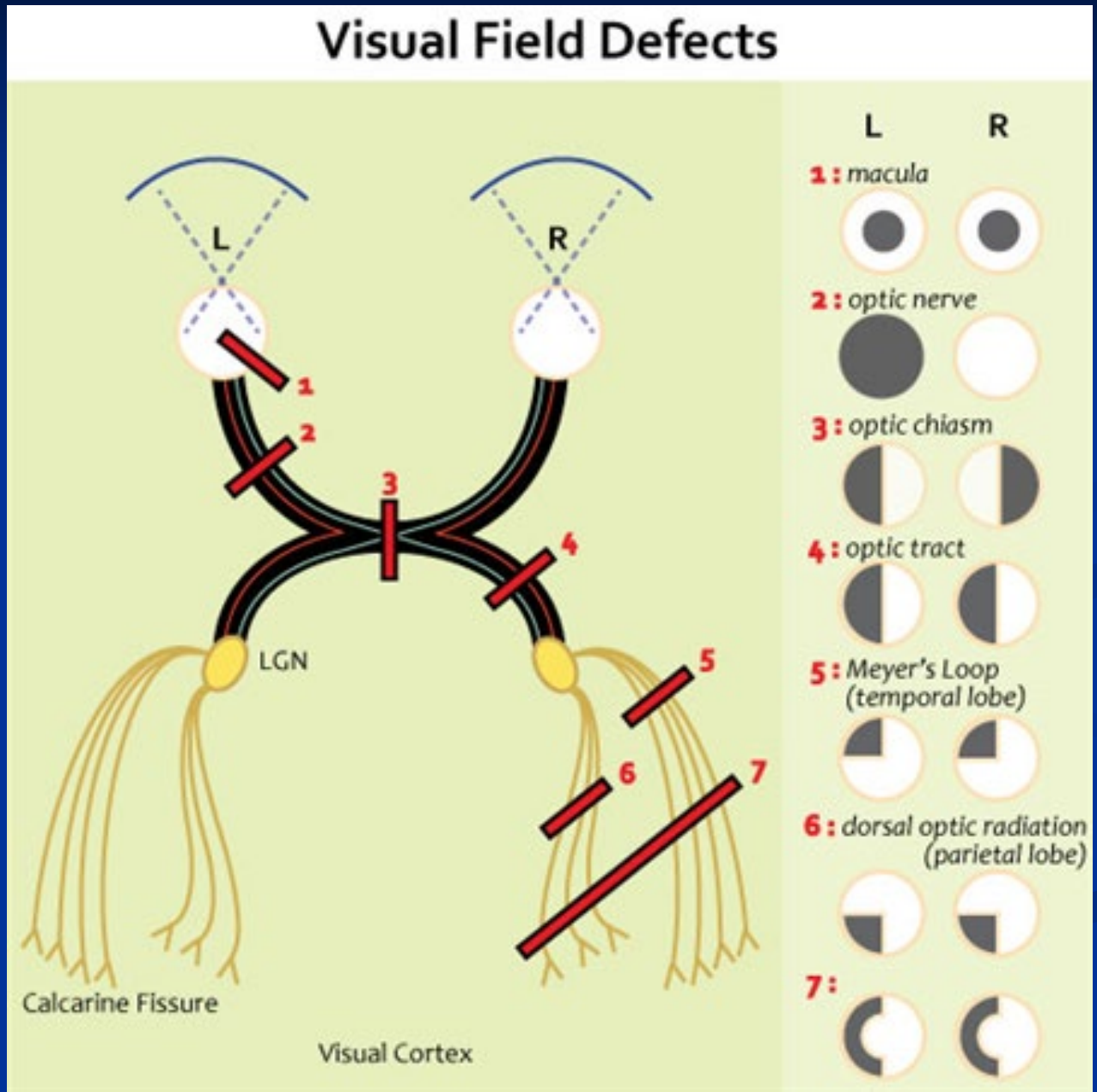
	Dissociative non-epileptic seizures ("pseudoseizures")	Epileptic seizures
Induced by anger, panic, suggestion	Common	Rare
Onset	Often gradual	Usually sudden
Duration	Often prolonged, occasionally hours	1–3 minutes
Breathing and colour	Breathing continues, stays pink	Usually apnoeic and cyanosed
Retained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

Back arching in Pseudoseizures

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



Anatomy of Visual Pathways



Case

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

- Last few days: dizzy, unsteady, leg pains, parasthesiae, diplopia, slow speech and poor concentration. Sleepy and easy irritability.

- O/E:

  Stable V/S, GCS 15/15. Irritable.

  Restriction of eye movements + Nystagmus

  Gait ataxia

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

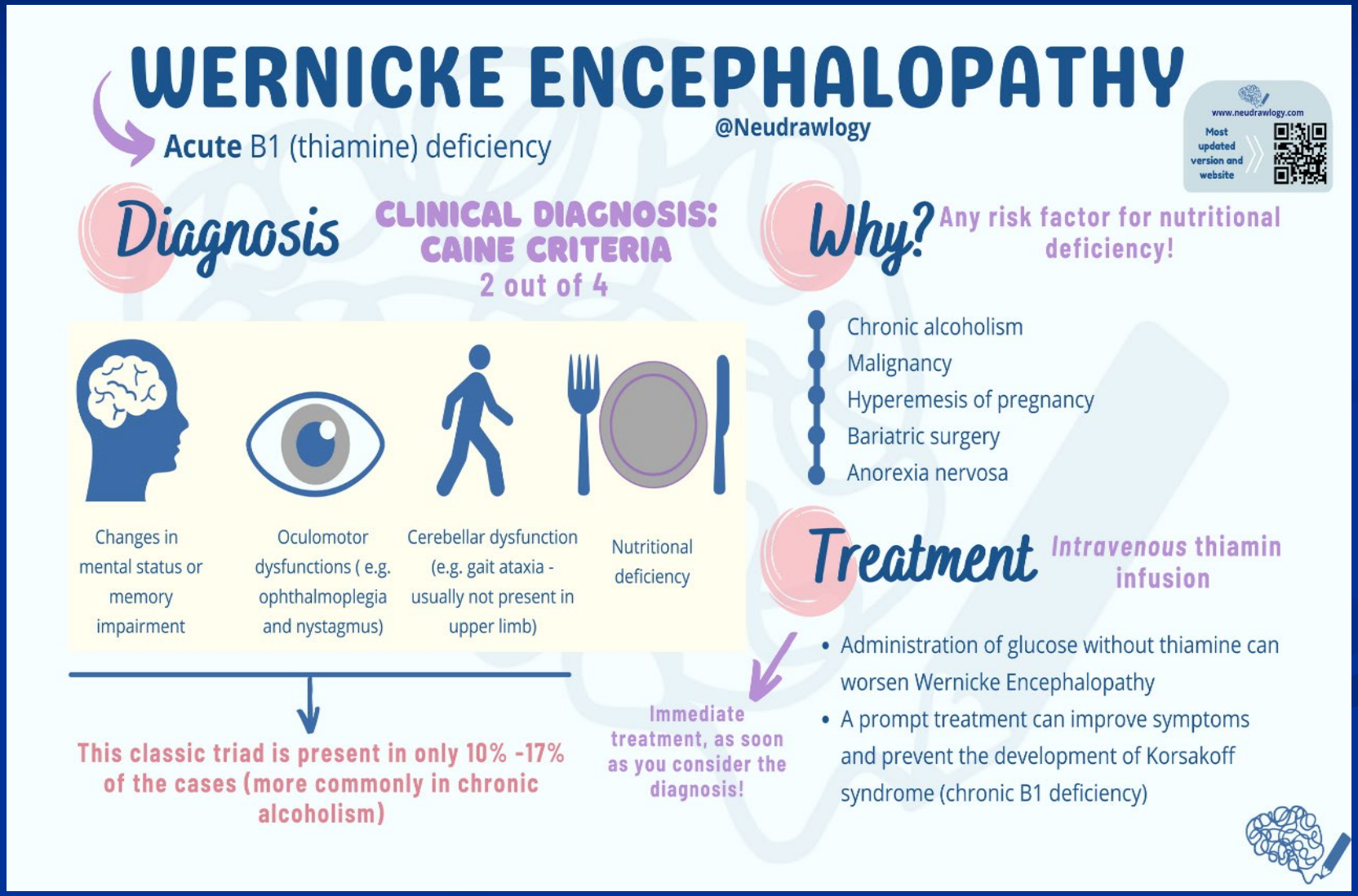
  A. steroids

  B. antibiotics + antivirals

  C. IVIG

  D. something else

What was the treatment?
IV Thiamine (Vit. B1)



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