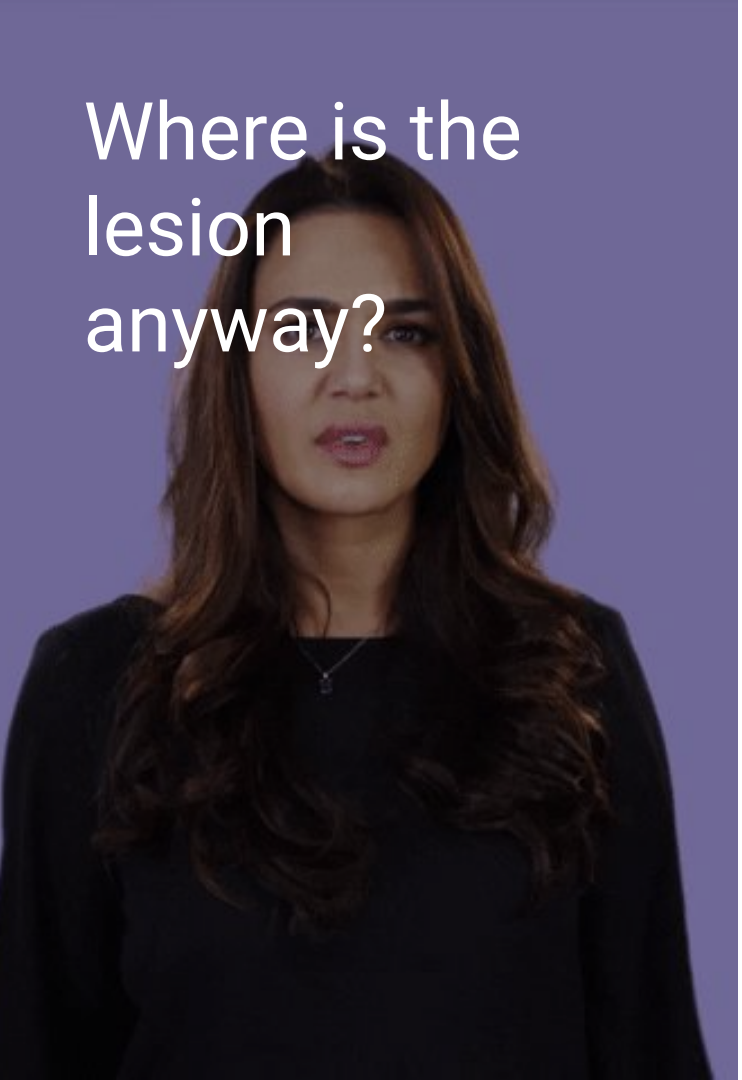
Review

Kaesha Thomas

Where is the lesion anyway?



- I don’t know

- Lesions can be:
  - Brain
  - SC
  - Peripheral Nerves
  - Anterior Horn Cells
  - NM Junctions

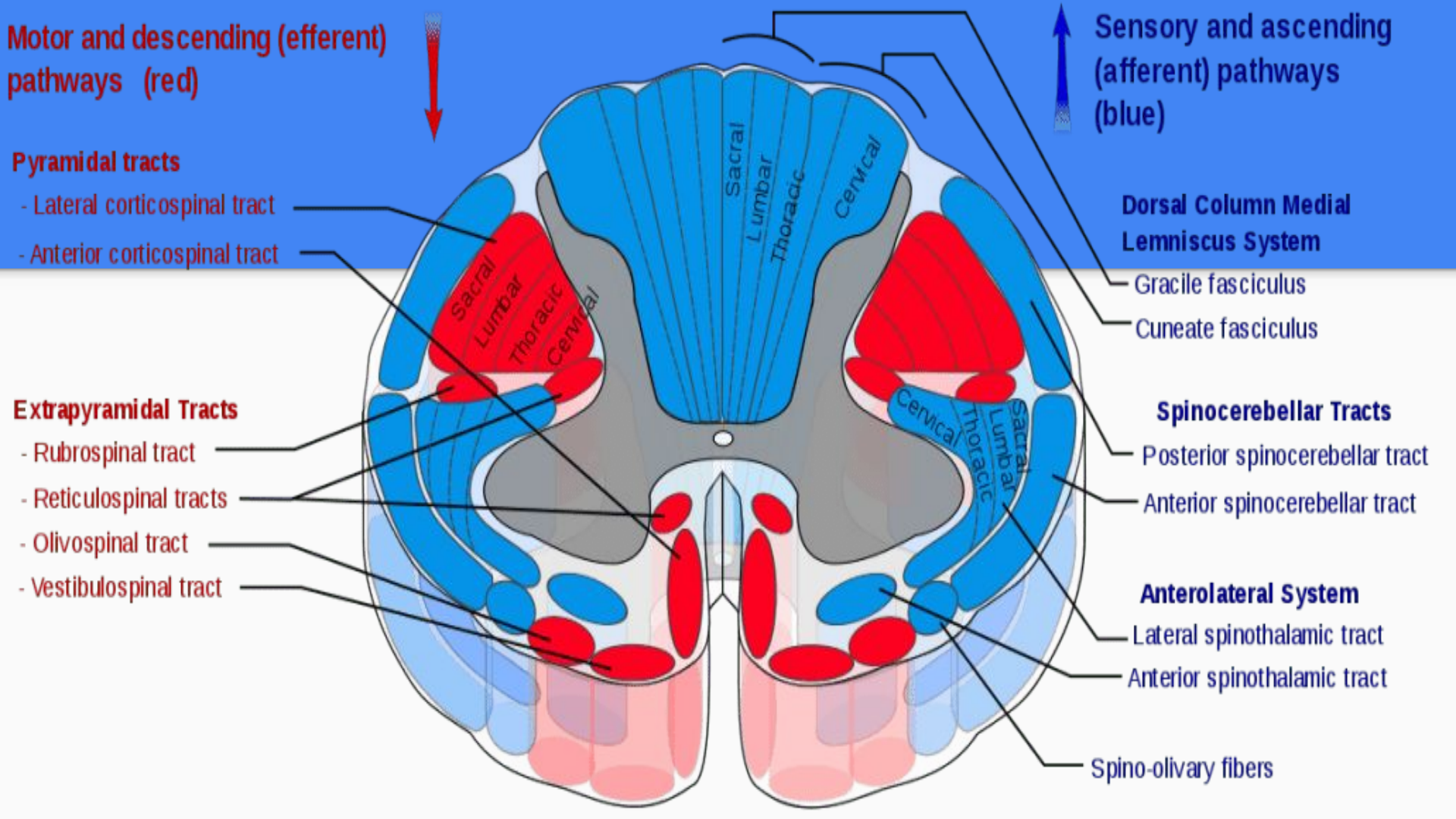
Lesions

- There’s no hard and fast area for the lesion to be
- Presentation can have more than one lesion
    - Lt sided lesion in the brain vs lesion in right upper SC will have similar signs

UMN vs LMN Lesion

Upper Motor Neuron (UMN) vs. Lower Motor Neuron (LMN) Syndrome

	UMN syndrome	*LMN Syndrome*
Type of Paralysis	*Spastic Paresis*	*Flaccid Paralysis*
Atrophy	No (Disuse) Atrophy	Severe *Atrophy*
Deep Tendon Reflex	*Increase*	Absent DTR
Pathological Reflex	Positive *Babinski* Sign	Absent
Superficial Reflex	Absent	Present
Fasciculation and Fibrillation	Absent	Could be *Present*

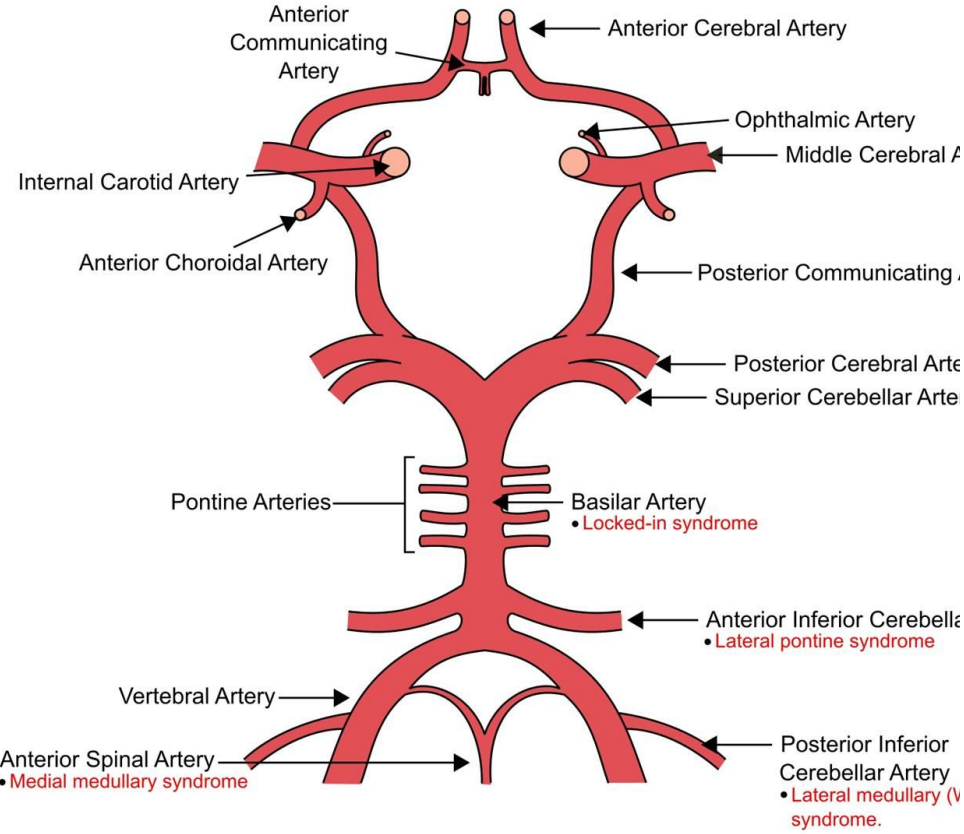

Motor and descending (efferent) pathways (red)
Sensory and ascending (afferent) pathways (blue)
Pyramidal tracts
- Lateral corticospinal tract
- Anterior corticospinal tract
Extrapyramidal Tracts
- Rubrospinal tract
- Reticulospinal tracts
- Olivospinal tract
- Vestibulospinal tract
Sacral
Lumbar
Thoracic
Cervical
Dorsal Column Medial Lemniscus System
Gracile fasciculus
Cuneate fasciculus
Spinocerebellar Tracts
Posterior spinocerebellar tract
Anterior spinocerebellar tract
Anterolateral System
Lateral spinothalamic tract
Anterior spinothalamic tract
Spino-olivary fibers

Extended Matching

A 35-year-old lady comes home from her stressful job and suddenly notices an almighty headache. It is located at the back of her head and by the time you see her she describes it as the worst pain she has ever experienced. She notices associated nausea but no vomiting and starts to complain of neck stiffness.

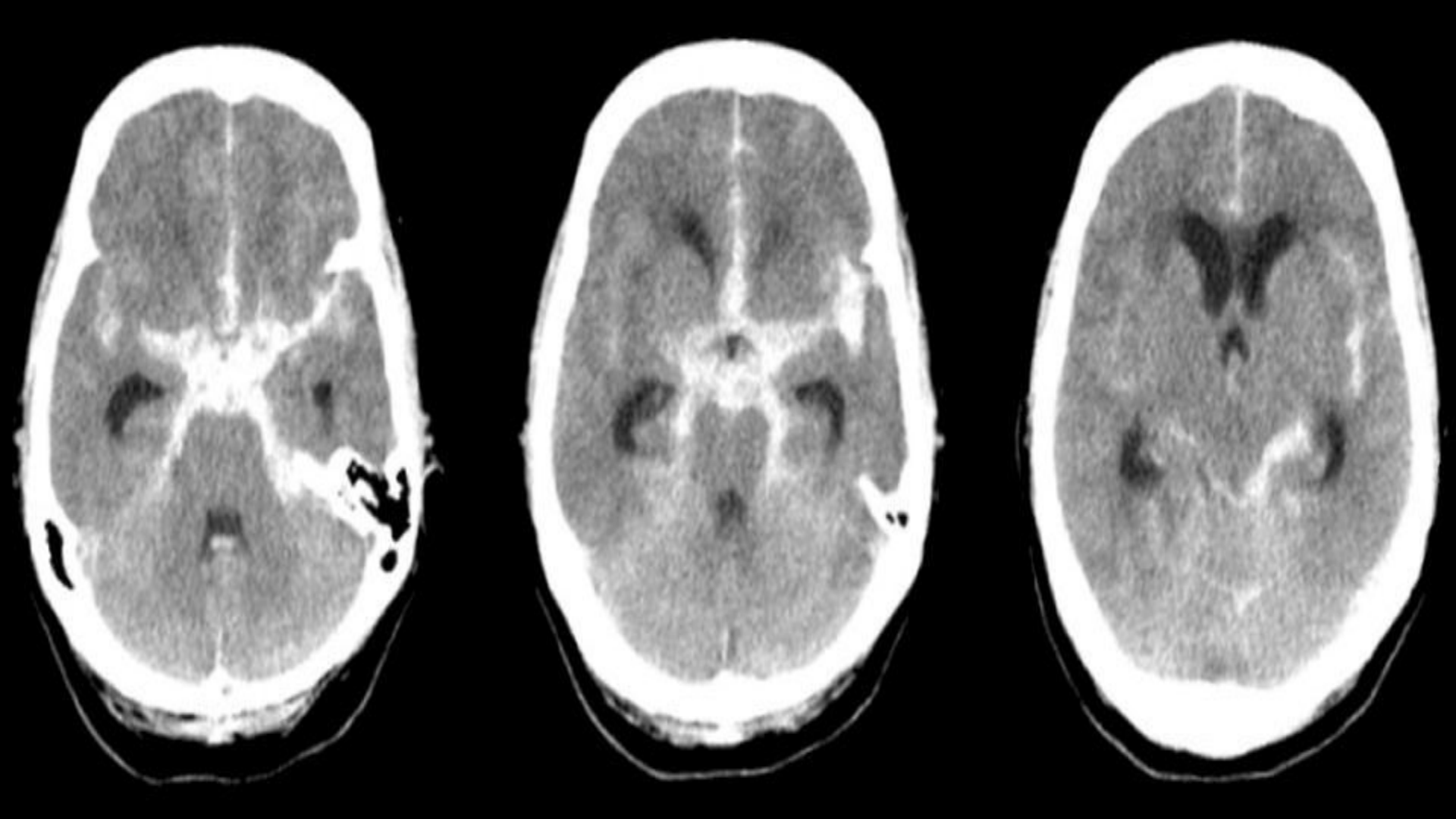
- Tension headache.
- Subarachnoid haemorrhage
- Subdural haemorrhage
- Extradural haemorrhage
- Cavernous sinus thrombosis
- Meningitis
- Sinusitis
- Cluster headache

Circle of Willis



SAH

- Can cause meningism (neck stiffness, photophobia and headache)
- sudden, ‘worst headache ever’ 10/10
- Thunderclap
- Associated with PKD
    - Berry aneurysms in the Circle of Willis



Polycystic Kidney Disease

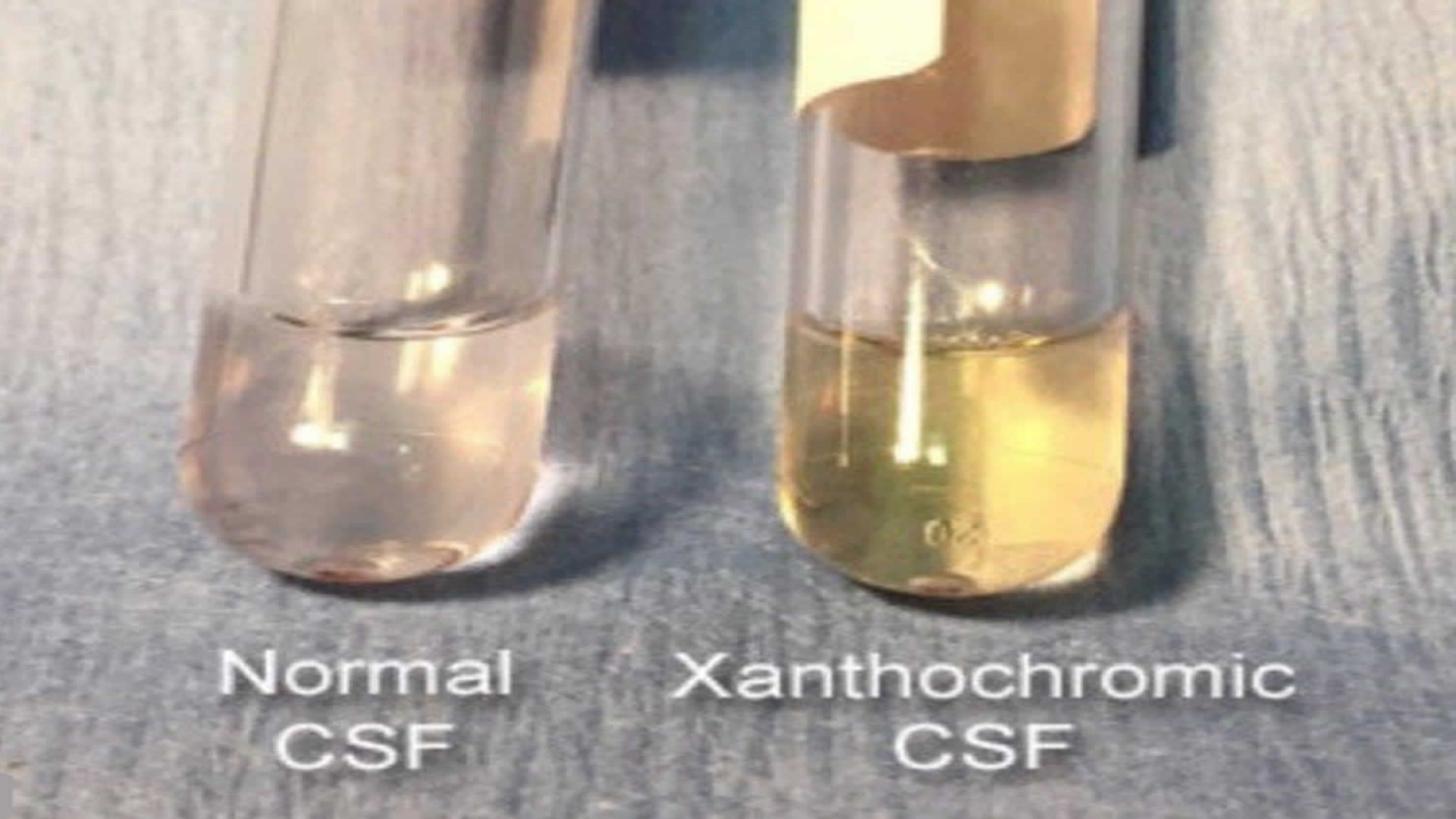
What are the two/three types?

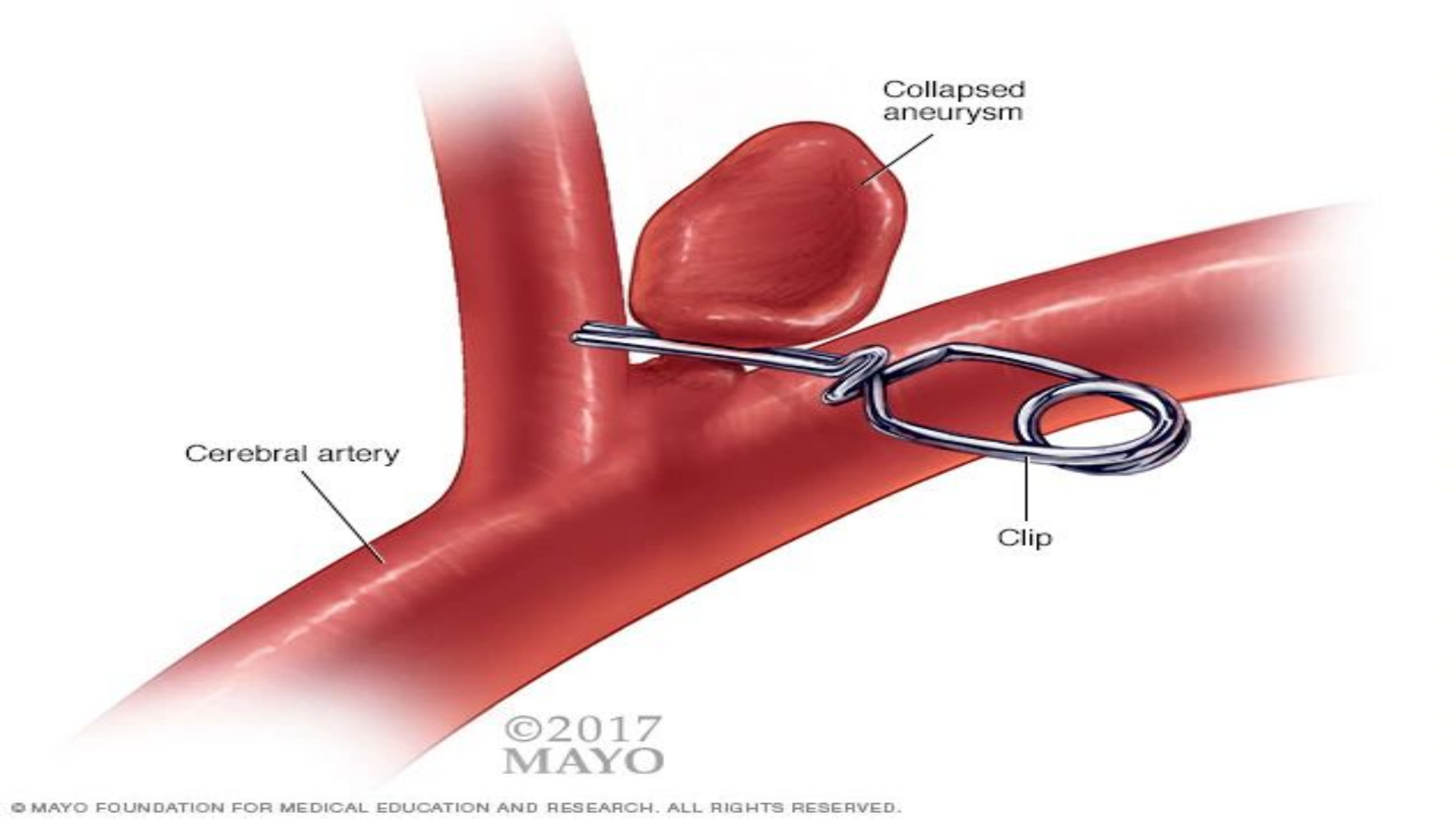
What chromosomes are involved?

	Autosomal dominant polycystic kidney disease (ADPKD)	Autosomal recessive polycystic kidney disease (ARPKD)
Incidence / Prevalence	~1:400 – 1:1000 live births ~600,000 patients in USA	~1:20,000 live births ~115,000 patients in USA
Mutated genes	PKD1 (~85%) PKD2 (~15%)	PKHD1
Major clinical features	Kidney cysts (100%) Bile duct cysts (70%) Hypertension (80%) Vascular aneurysms (10%)	Kidney cysts Bile duct cysts Hypertension
Outcomes and complications	~ 50% of patients develop kidney failure. Aneurysms may rupture causing sudden death.	>50% of patients develop kidney failure before 20 years of age. Portal hypertension
Major advances	Generation of orthologous animal models. On-going human clinical trials.	Generation of orthologous animal models.

SAH

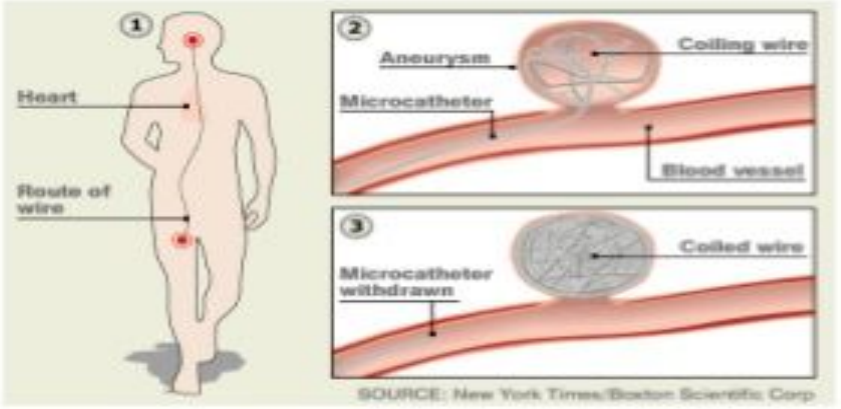
- What would be seen on LP?
- SAH Protocol
  - 6L/min O2
  - Dark room
  - IVF at 125 cc/hr
  - Lack of noise
- Eventual clipping of aneurysm
  - UWI now has a radiological suite for coiling


Normal
CSF
Xanthochromic
CSF

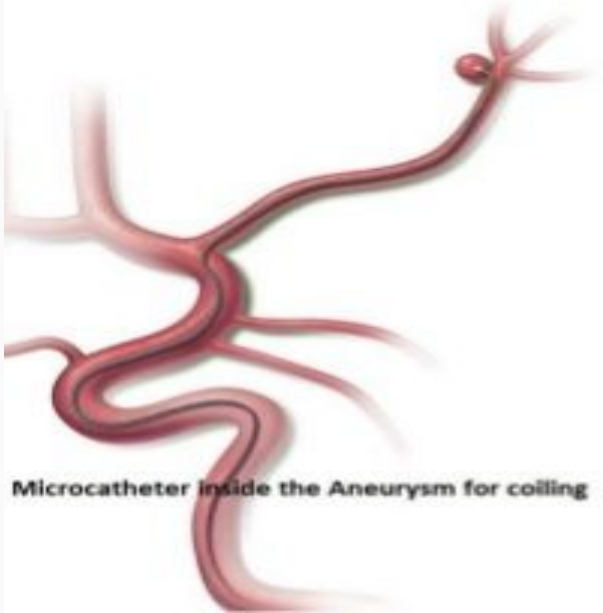


Coiling of aneurysm:

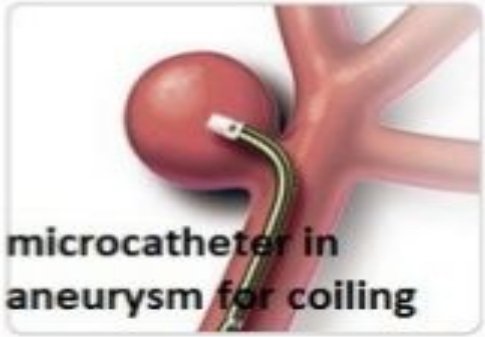
Endovascular (meaning within the blood vessel) embolization, or coiling, uses the natural access to the brain through the bloodstream via arteries to diagnosis and treat brain aneurysms. The goal of the treatment is to safely seal off the aneurysm and stop further blood from entering into the aneurysm and increasing the risk of rupture or possibly rebleeding.

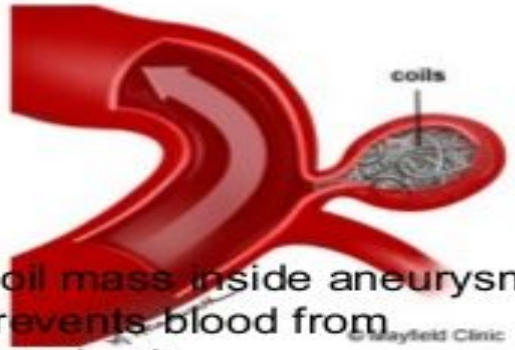


Following diagrams show how aneurysm coiling is done



Microcatheter inside the Aneurysm for coiling





Coil mass inside aneurysm prevents blood from entering it

Visible CSF Xanthochromia

- Oxyhemoglobin resulting from artifactual red cell lysis
- Bilirubin in jaundiced patients
- CSF protein levels > 150 mg/dL
- Carotenoids
- Melanin
- Rifampin thereapy

	Normal	Bacterial	Viral	Fungal/TB
Pressure (cmH20)	5-20	> 30	Normal or mildly increased	
Appearance	Normal	Turbid	Clear	Fibrin web
Protein (g/L)	0.18-0.45	> 1	< 1	0.1-0.5
Glucose (mmol/L)	2.5-3.5	<2.2	Normal	1.6-2.5
Gram stain	Normal	60-90% Positive	Normal	
Glucose - CSF:Serum Ratio	0.6	< 0.4	> 0.6	< 0.4
WCC	< 3	> 500	< 1000	100-500
Other		90% PMN	Monocytes 10% have >90% PMN 30% have >50% PMN	Monocytes

Extended Matching

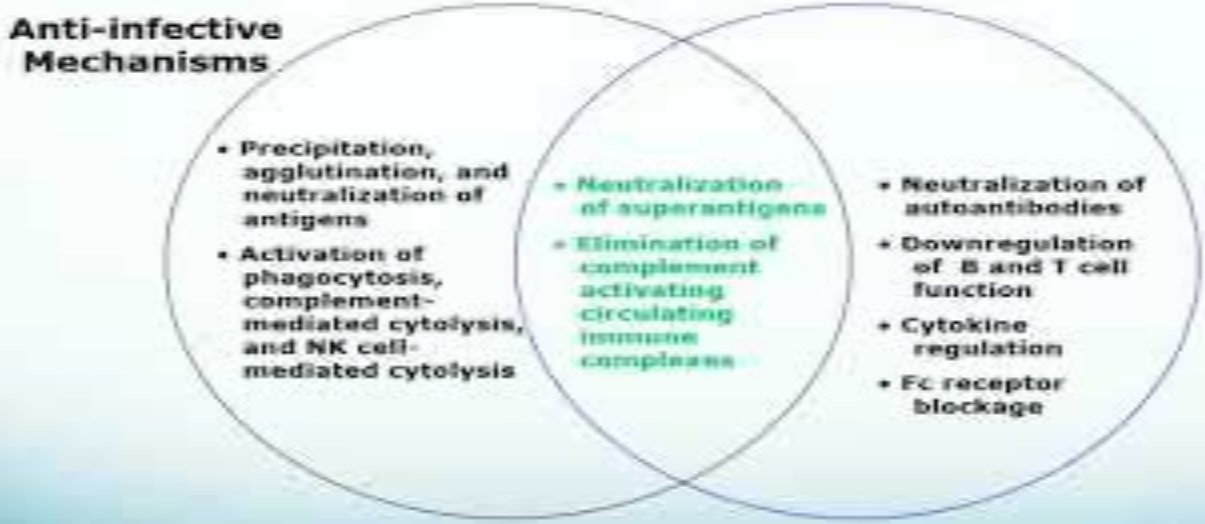
A 7 year old girl comes into your office complaining of difficulty walking. She was otherwise well. Her only PMH is gastroenteritis 2 weeks ago that resolved with supportive care.

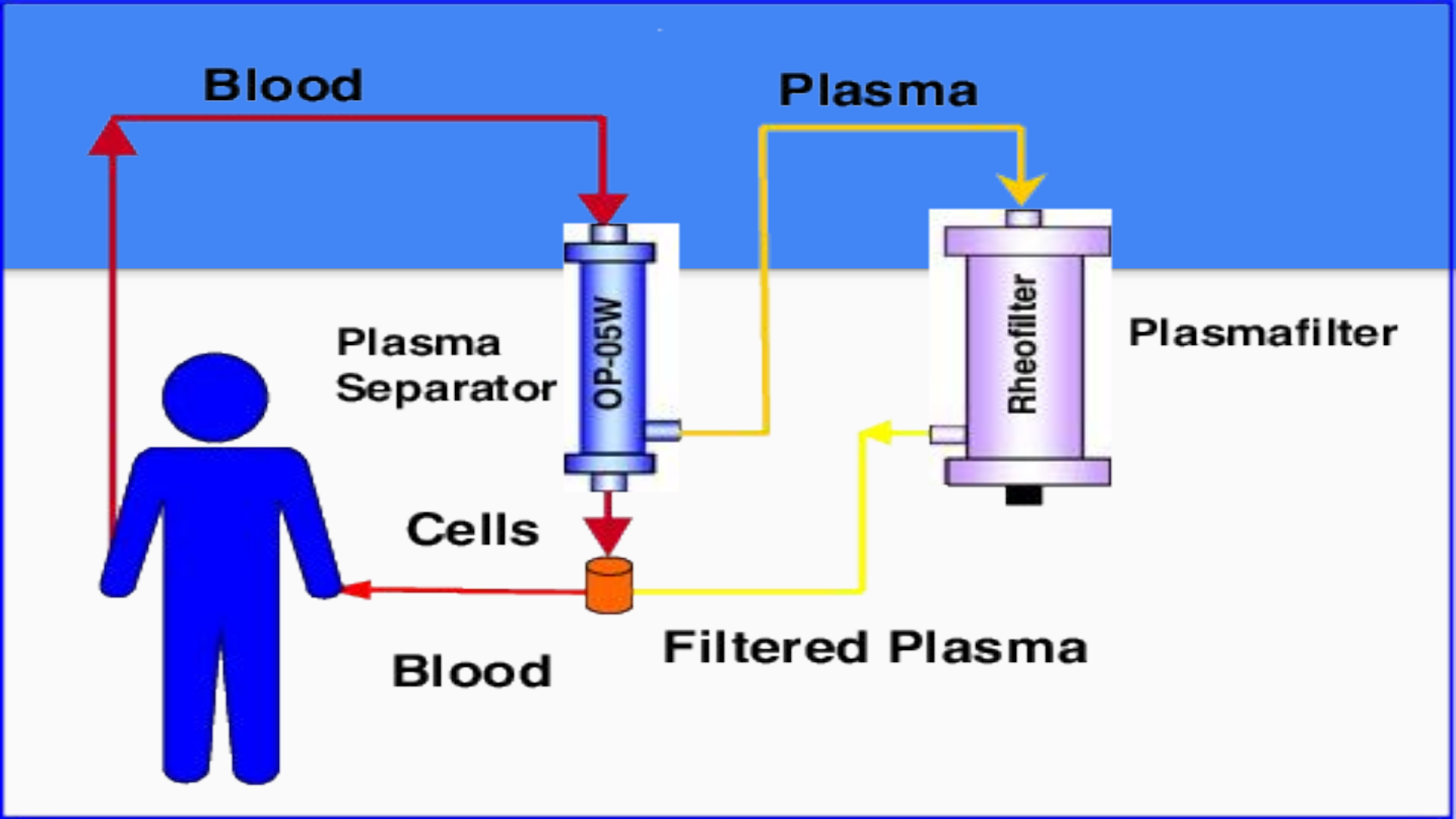
- Diabetes
- Amyloid
- B12 Deficiency
- Guillain–Barre syndrome
- Lead poisoning

Guillain Barre Syndrome

- Ascending paralysis
- Autoimmune disorder
- Can involve respiratory muscles
- Antiganglioside antibodies
- Risk of DVT, Resp Failure, Aspiration Pneumonia
- Treatment with?

The Role of IGIVs




Blood
Plasma
Plasma
Separator
OP-05W
Rheofilter
Plasmafilter
Cells
Blood
Filtered Plasma

GBS is typically associated with what GI infection?

Campylobacter Jejuni

Distal limb weakness phenotype of GBS

Frequency

7.5% of all GBS

Causative factors

Campylobacter jejuni

Clinical features

- Antecedent diarrhea
- Mildly disabled w/o treatment
- Mainly hand weakness
- Partially preserved DTRs
- Normal protein level in CSF
- Positive anti-ganglioside Abs
- Axonal or RCF* pattern

* reversible conduction failure

What is classically seen on LP?

What is classically seen on LP?

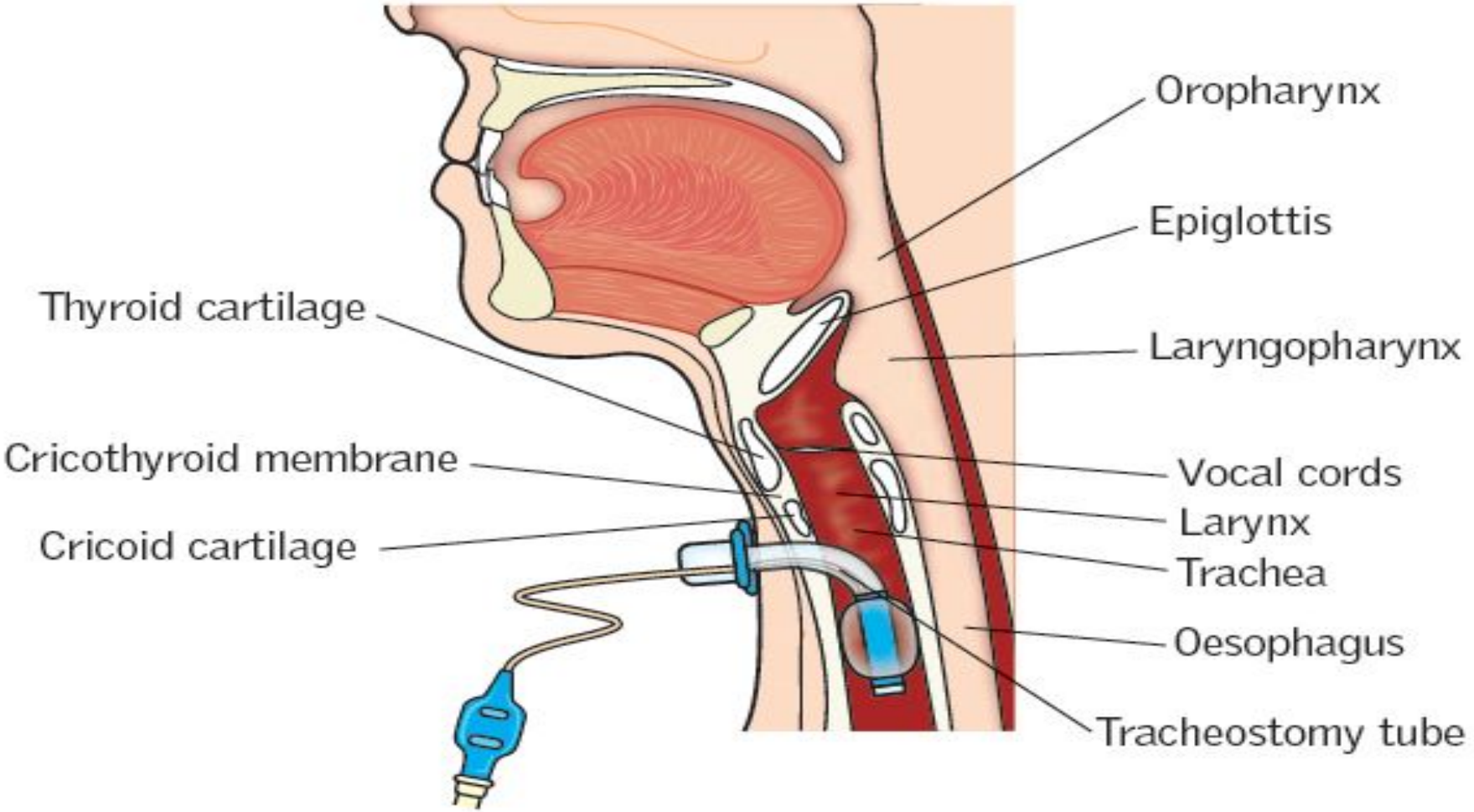
Albuminocytologic Dissociation

What does that even mean?

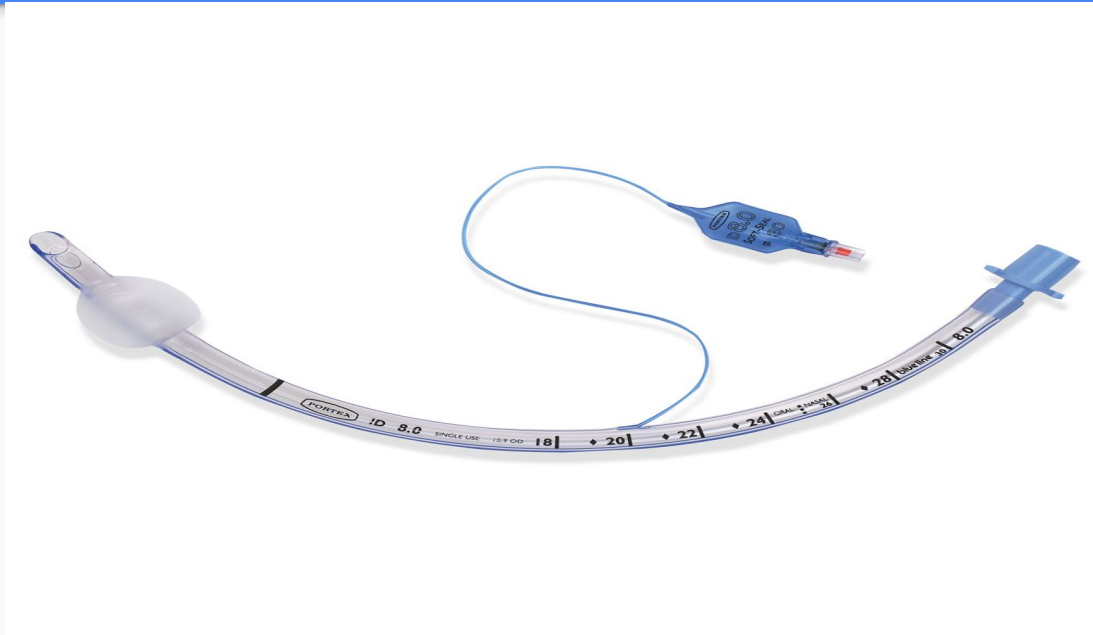
- Elevated protein levels in CSF
- Normal cell counts in CSF

Extended Matching Cont’d

During the course of her treatment she begins to have gasping. O2 sat is 70% and she is centrally cyanotic. ABG done shows type 2 RF. She is intubated and ventilated, and has been for 7 days. Unable to wean, what is your next step in management?


Oropharynx
Epiglottis
Thyroid cartilage
Laryngopharynx
Cricothyroid membrane
Vocal cords
Larynx
Cricoid cartilage
Trachea
Oesophagus
Tracheostomy tube

What drug is typically given prior to intubation?



Succinylcholine

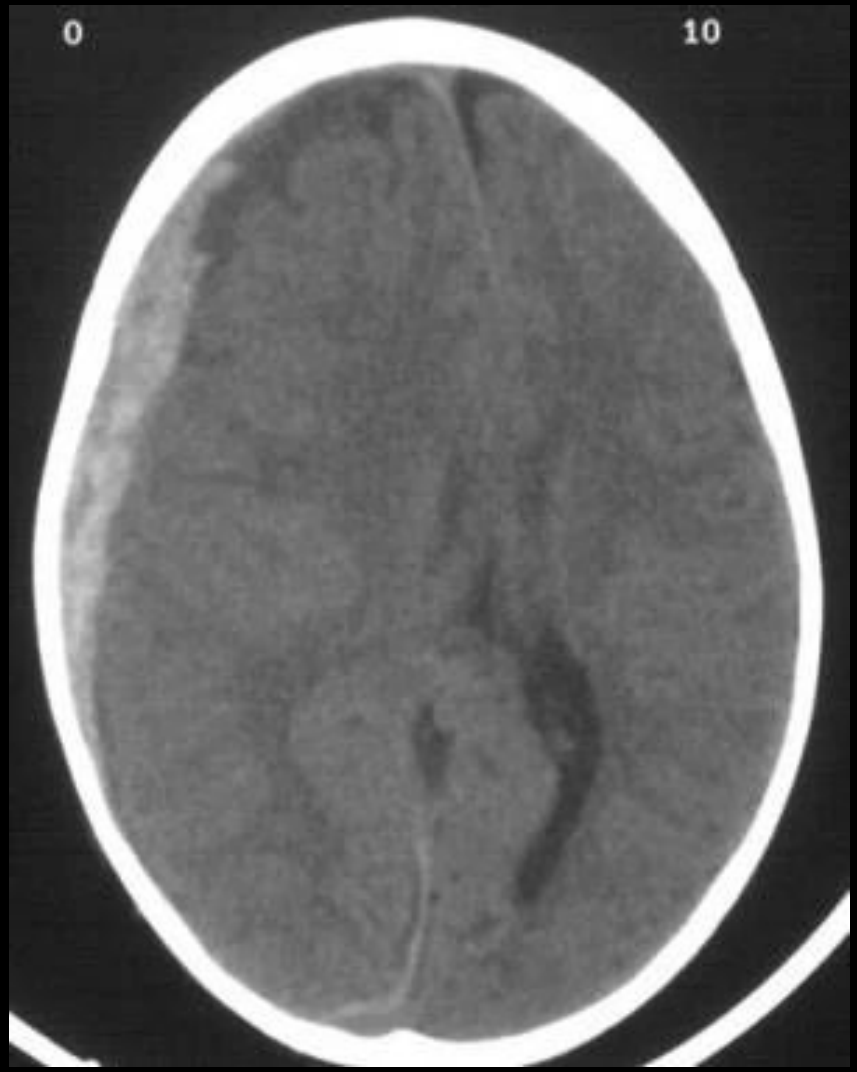
Extended Matching

A 47-year-old lady with a longstanding history of epilepsy has been brought in by her family as she is suffering with a fluctuation in her conscious level and seems to have some weakness on the right hand side of her body. She has been suffering with an increasing number of fits recently. This has come on quite suddenly over the past 6 hours.

- Tension headache
- Subarachnoid haemorrhage
- Subdural haemorrhage
- Extradural haemorrhage
- Cavernous sinus thrombosis
- Meningitis
- Sinusitis
- Cluster headache

Subdural Hematoma

- Elderly, alcoholic, on anticoagulation
- Think brain atrophy
- Stretching of the bridging veins
- FLUCTUATING LOC – Confusion, ataxia, gradual physical and mental deterioration
- What does the CT show?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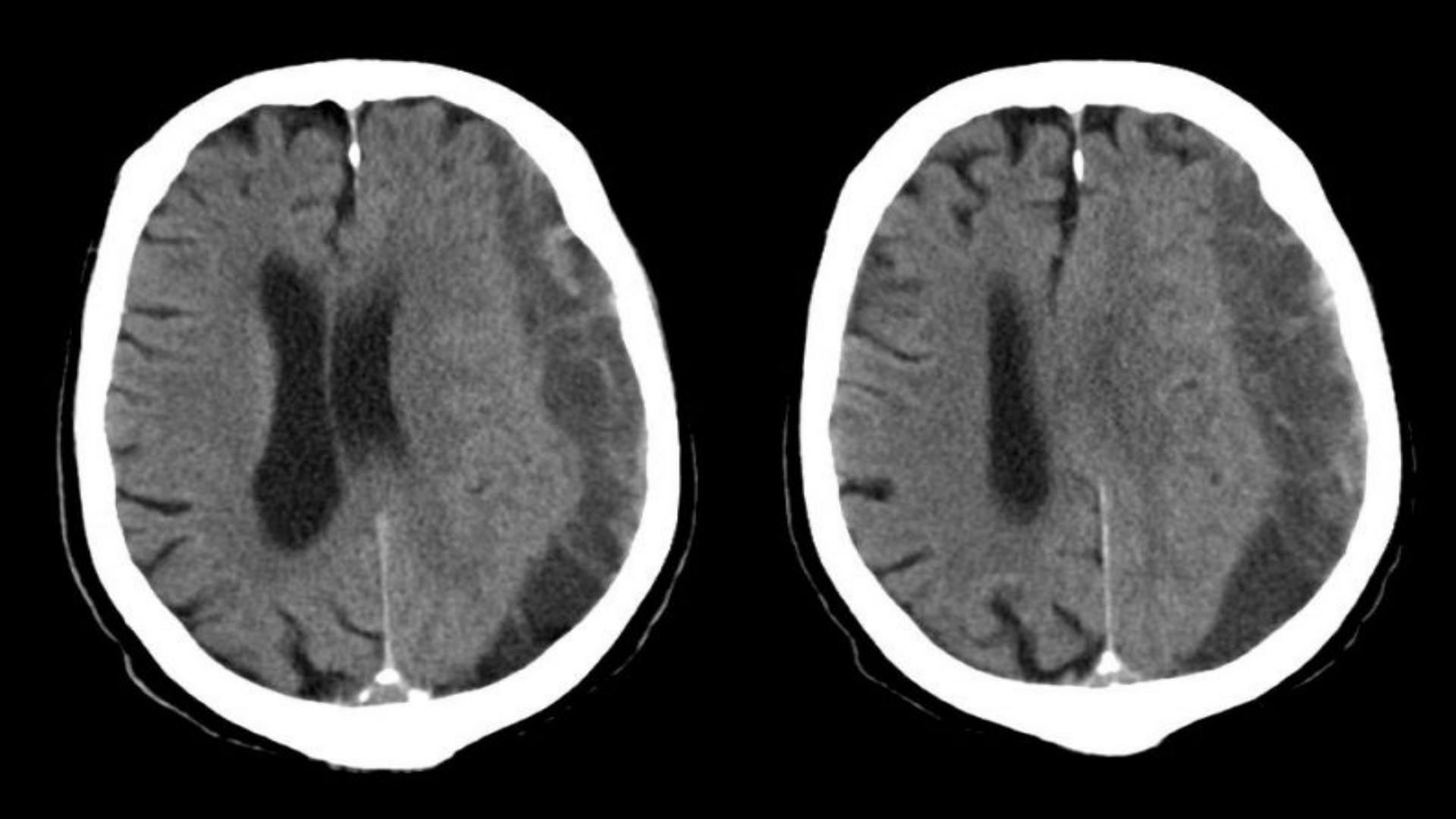
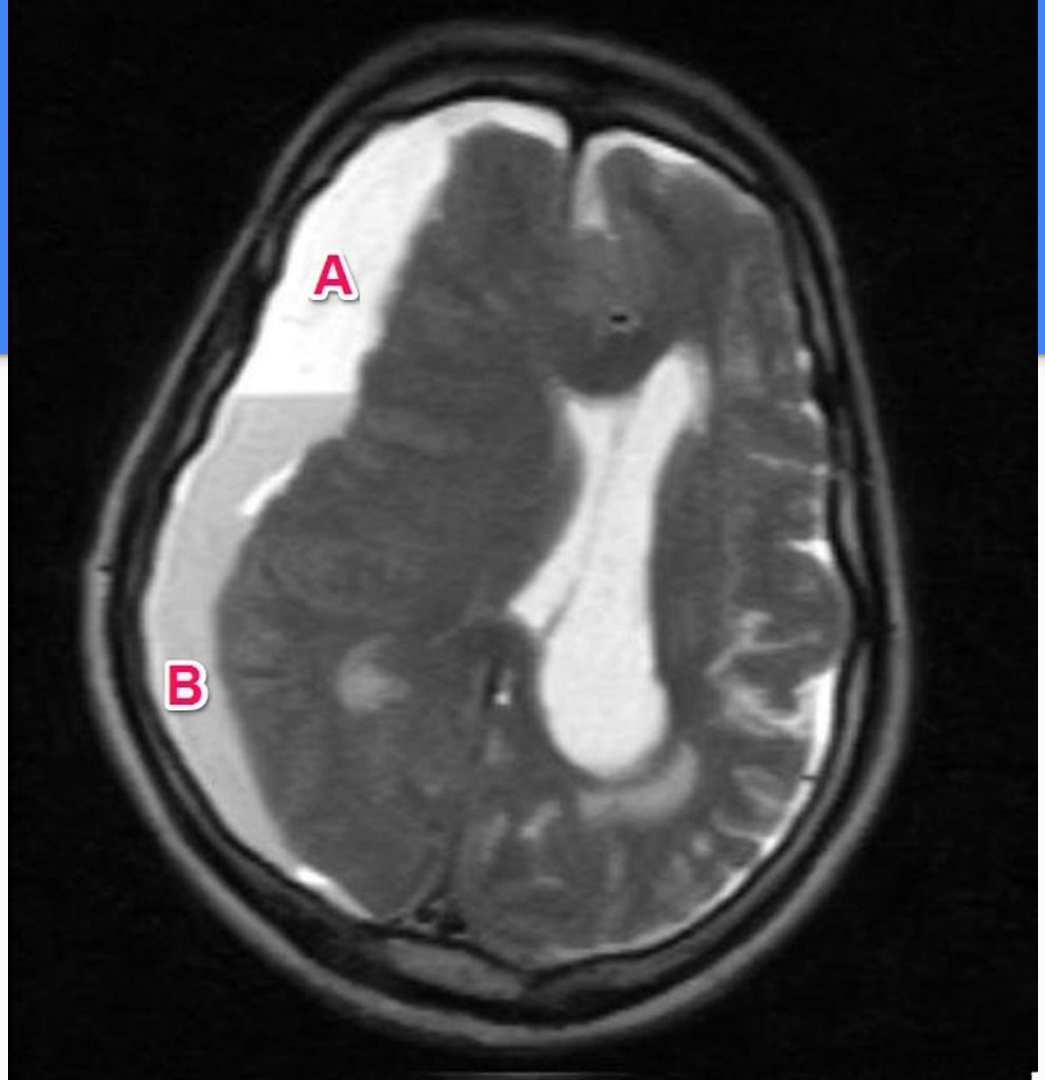
10



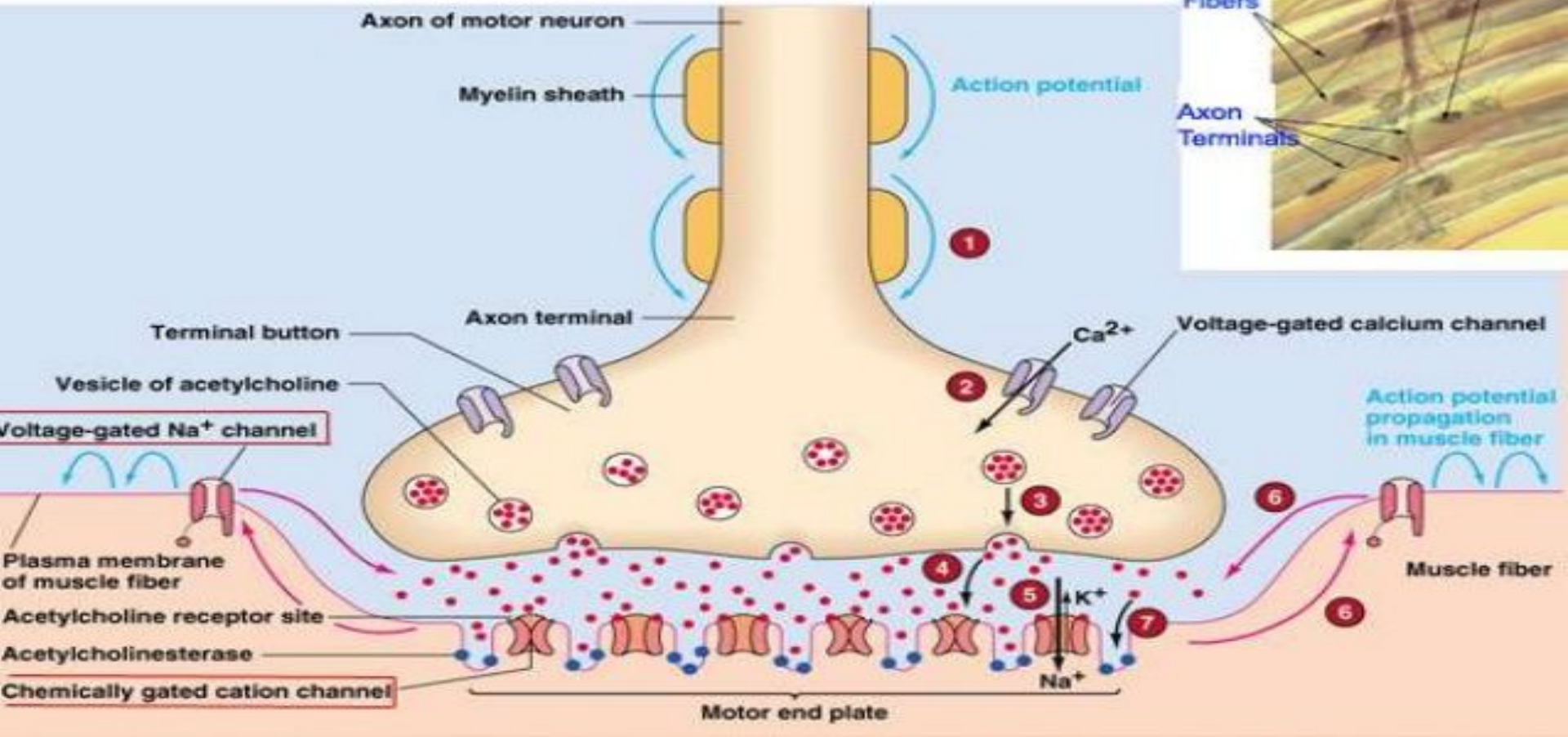

A
B

Extended Matching Questions

A 30 year old female presents with difficulty combing her hair and standing from a seated position.

- Eaton-Lambert Myasthenic Syndrome
- Myasthenia Gravis
- Botulism
- Lyme Disease
- Guillain-Barré Syndrome

The Neuromuscular Junction



Myasthenia Gravis

- **Young women (20-35)**
  - Tend to present with a generalised, and often acute condition
- **Older men (60-75)**
  - Who tend to present with prominent oculobulbar involvement

Fatigable weakness

- Ocular and bulbar involvement is also possible, leading to ptosis, swallowing difficulties and speech disturbance

Antibodies in MG

- Anti-AChR antibodies (90%)
- Anti-MUSK antibodies
  - Antibodies to the Muscle Specific Kinase (MuSK)
- Approximately 10% of patients are ‘seronegative’ (negative AChR and MUSK antibodies)

Treatment

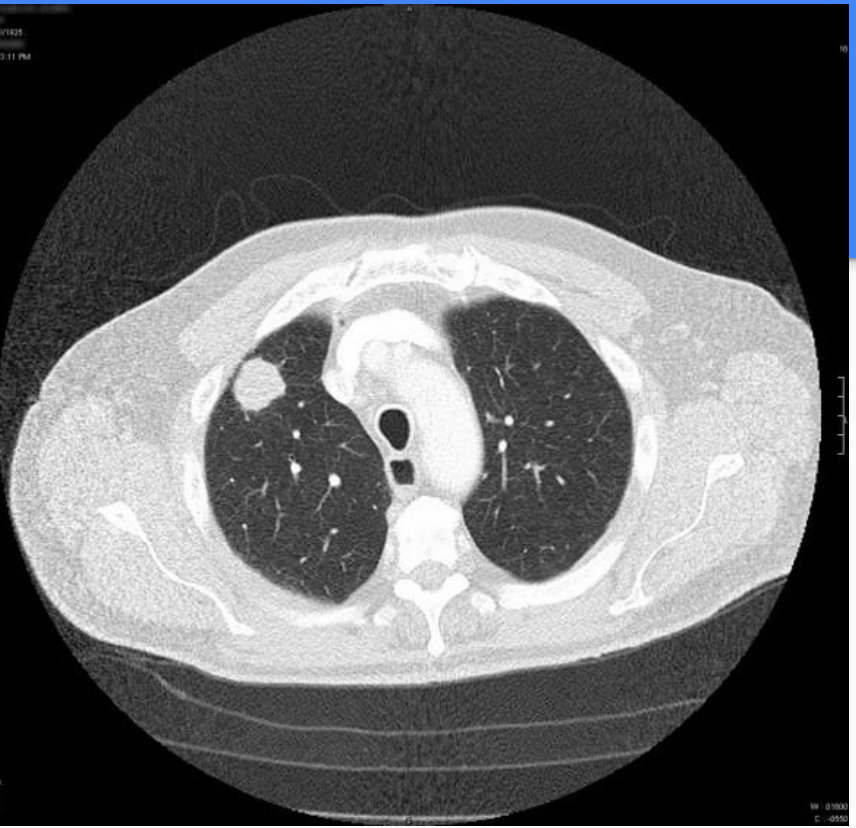
- Immunosuppression is the mainstay of treatment
- Acutely (and in flares)
- Longer term
- Thymectomy
    - Thymectomy can cause improvement and even remission in up to 80% of MG, especially the young population, and is usually recommended

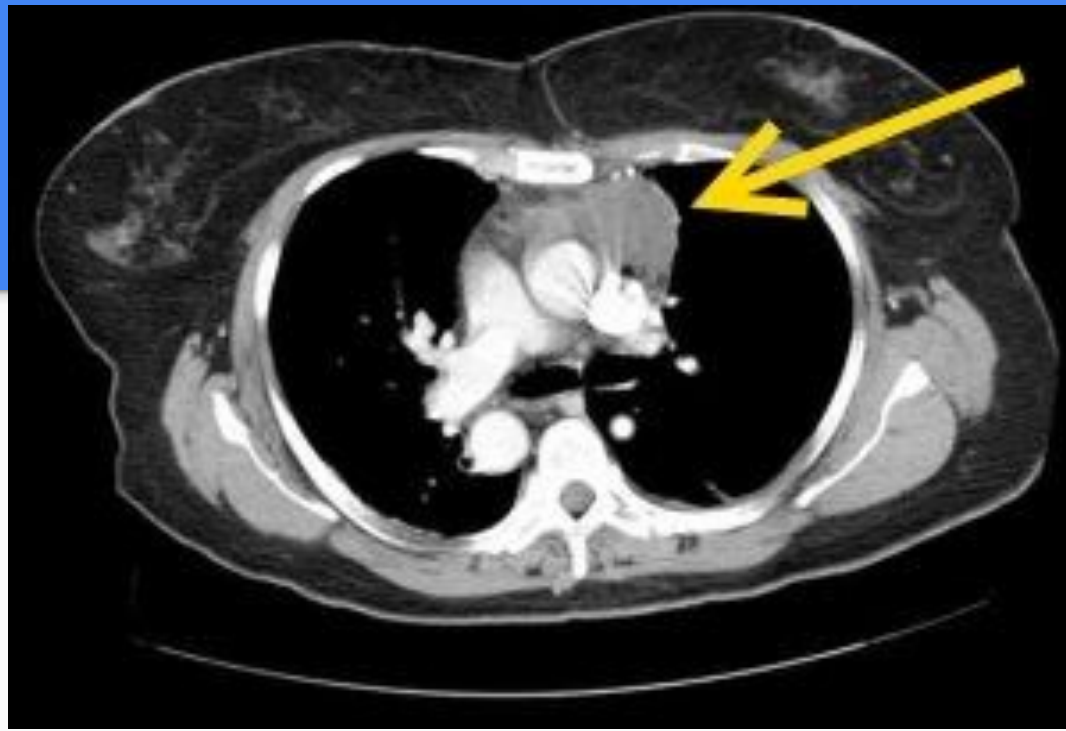
What are common differentials for MG?

Difference between Lambert Eaton syndrome and Myasthenia gravis

Myasthenia gravis	Lambert Eaton syndrome
Antibody against AchR antibody	Antibody against voltage gated calcium channel
Associated with Thymic tumor	Associated with Small cell lung cancer
Weakness worsen on prolonged exercise	Weakness improves on prolonged exercise
Normal Deep tendon reflex	Decreased or absent deep tendon reflex
Autonomic dysfunction is absent	Autonomic dysfunction is present
On repeated nerve stimulation, there is decremental response	On repeated nerve stimulation, there is incremental response

CT Chest in both?
Why?



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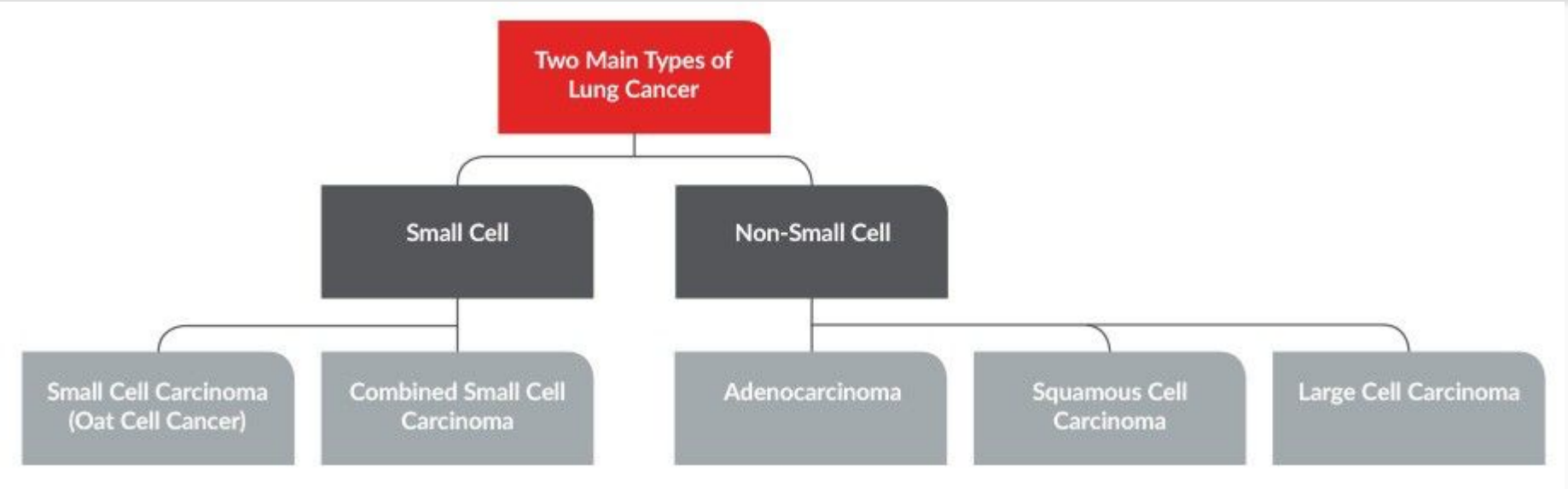
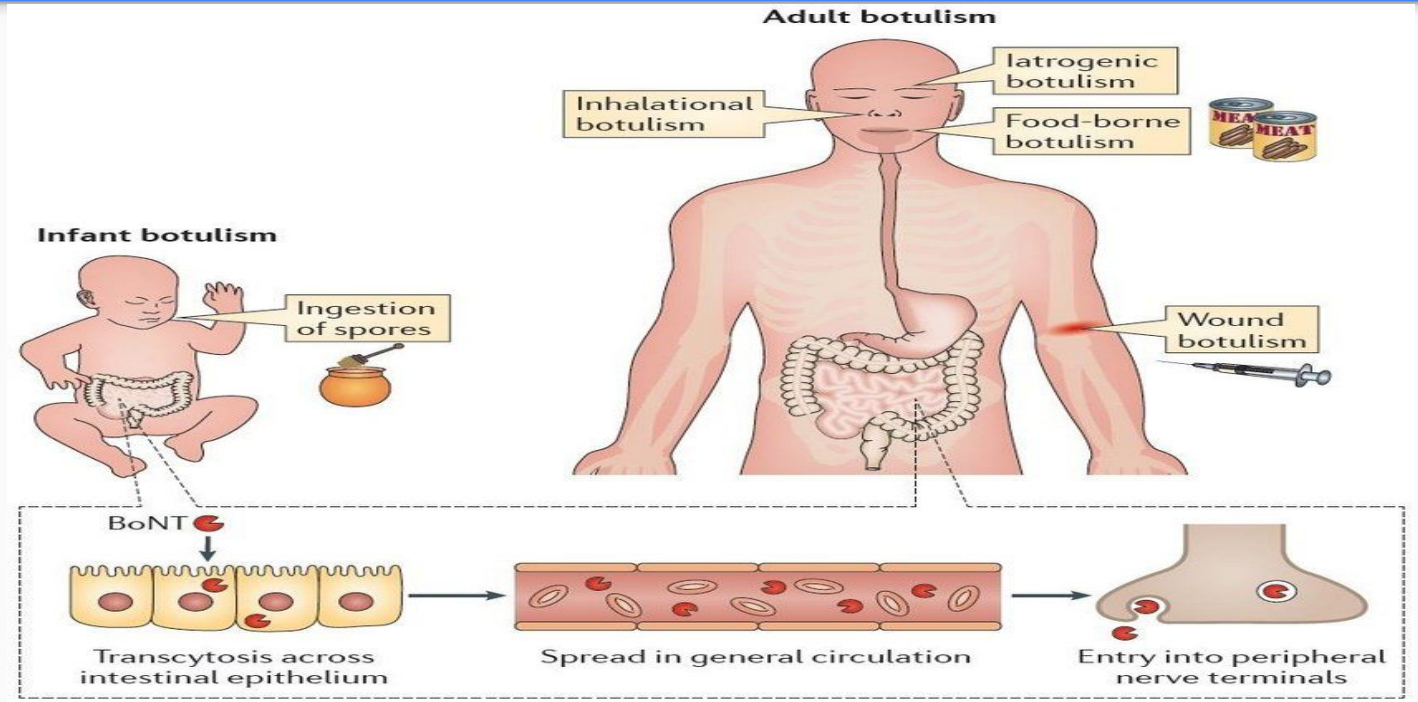
Lung Cancer


Two Main Types of Lung Cancer
Small Cell
Non-Small Cell
Small Cell Carcinoma (Oat Cell Cancer)
Combined Small Cell Carcinoma
Adenocarcinoma
Squamous Cell Carcinoma
Large Cell Carcinoma

Paraneoplastic Syndromes

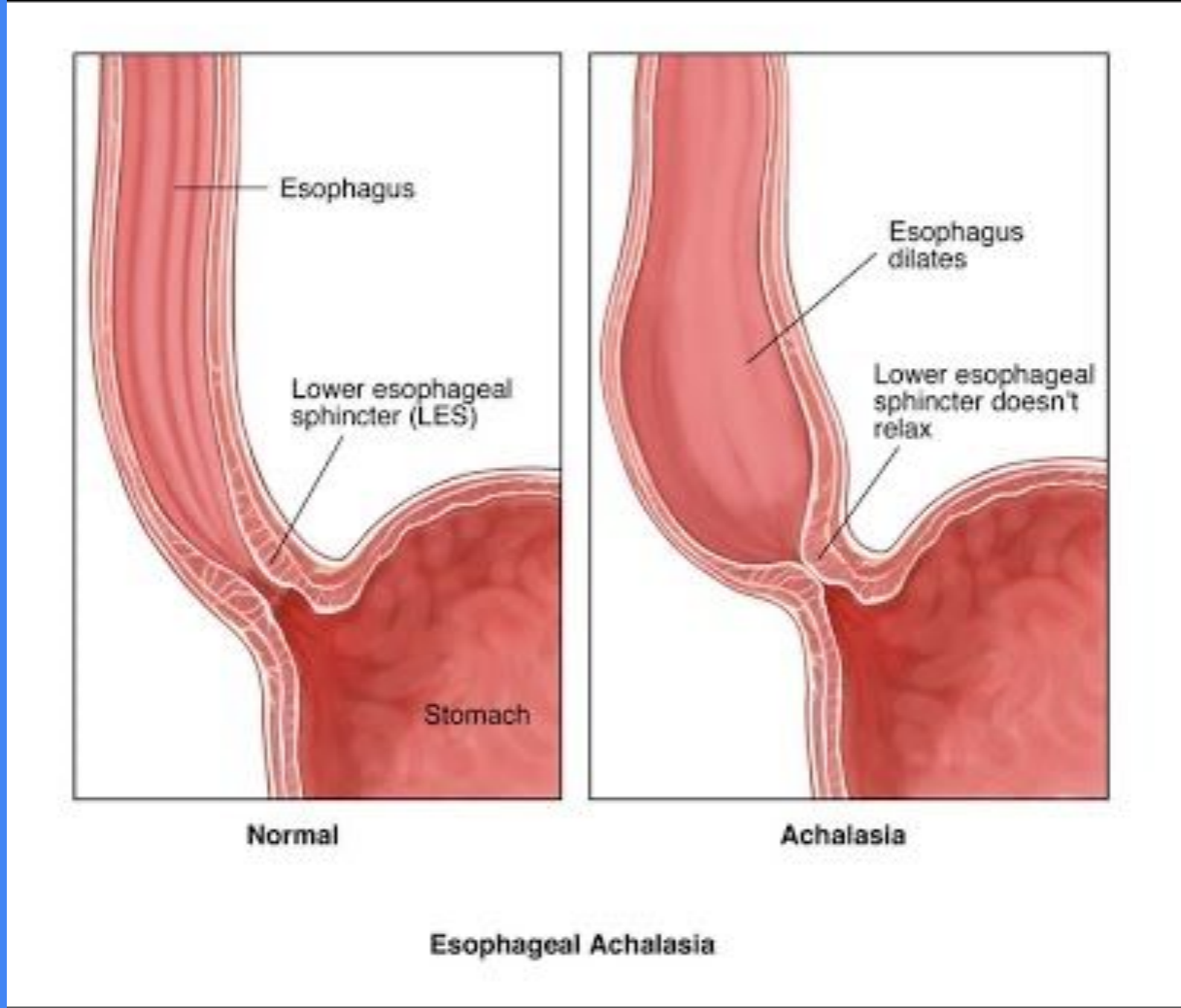
Clinical Syndromes	Major Forms of Underlying Cancer	Causal Mechanism
ENDOCRINOPATHIES		
Cushing syndrome	Small-cell carcinoma of lung	ACTH or ACTH-like substance
	Pancreatic carcinoma	
	Neural tumors	
Syndrome of inappropriate antidiuretic hormone secretion	Small-cell carcinoma of lung; intracranial neoplasms	Antidiuretic hormone or atrial natriuretic hormones
Hypercalcemia	Squamous cell carcinoma of lung	Parathyroid hormone–related protein (PTHRP), TGF-α, TNF, IL-1
	Breast carcinoma	
	Renal carcinoma	
	Adult T-cell leukemia/lymphoma	
Hypoglycemia	Ovarian carcinoma	
	Fibrosarcoma	Insulin or insulin-like substance
	Other mesenchymal sarcomas	
Carcinoid syndrome	Hepatocellular carcinoma	
	Bronchial adenoma (carcinoid)	Serotonin, bradykinin
	Pancreatic carcinoma	
Polycythemia	Gastric carcinoma	
	Renal carcinoma	Erythropoietin
	Cerebellar hemangioma	
	Hepatocellular carcinoma	

Clostridium Botulinum

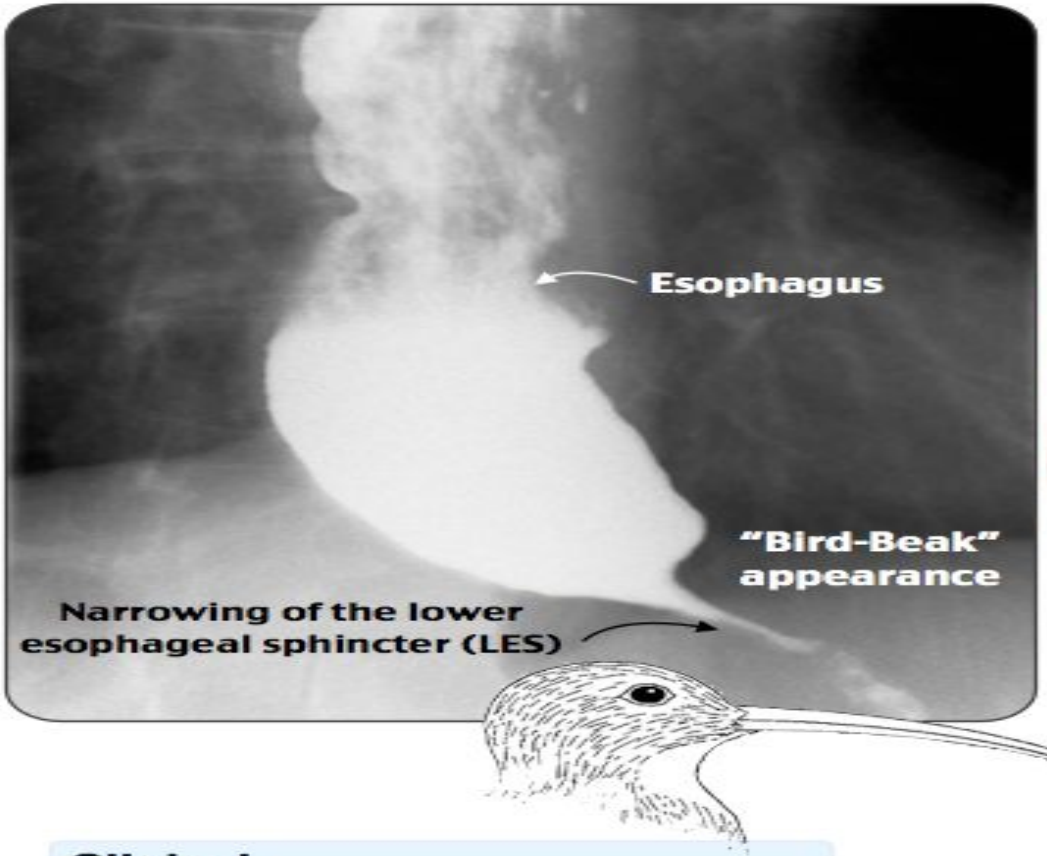


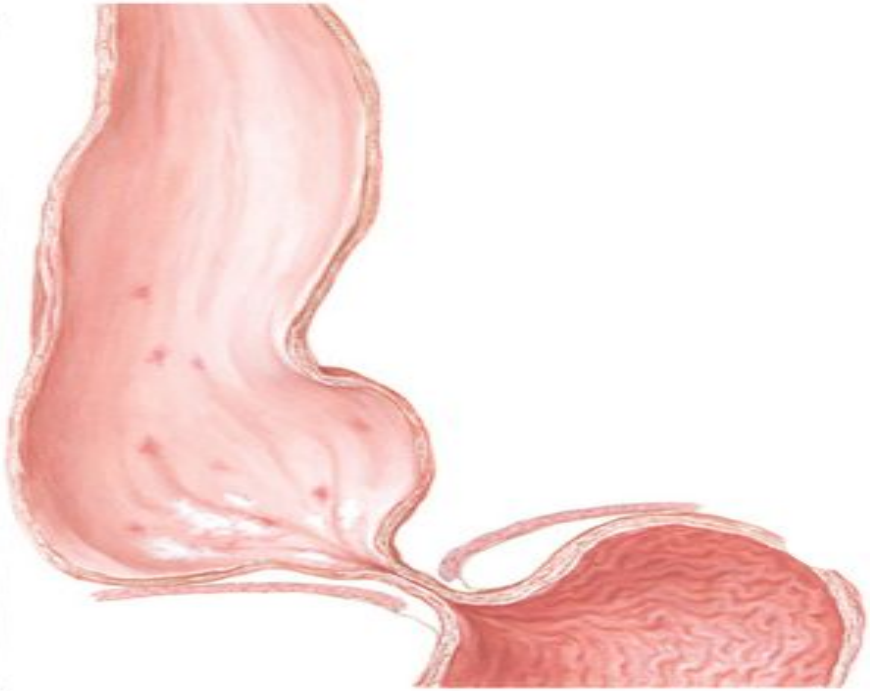
What can botox be used to treat?

Achalasia


Esophagus
Lower esophageal sphincter (LES)
Stomach
Normal
Esophagus dilates
Lower esophageal sphincter doesn't relax
Achalasia
Esophageal Achalasia

Achalasia





Clinical

- Dysphagia
- Odynophagia
- Regurgitation of undigested food

C. Diff

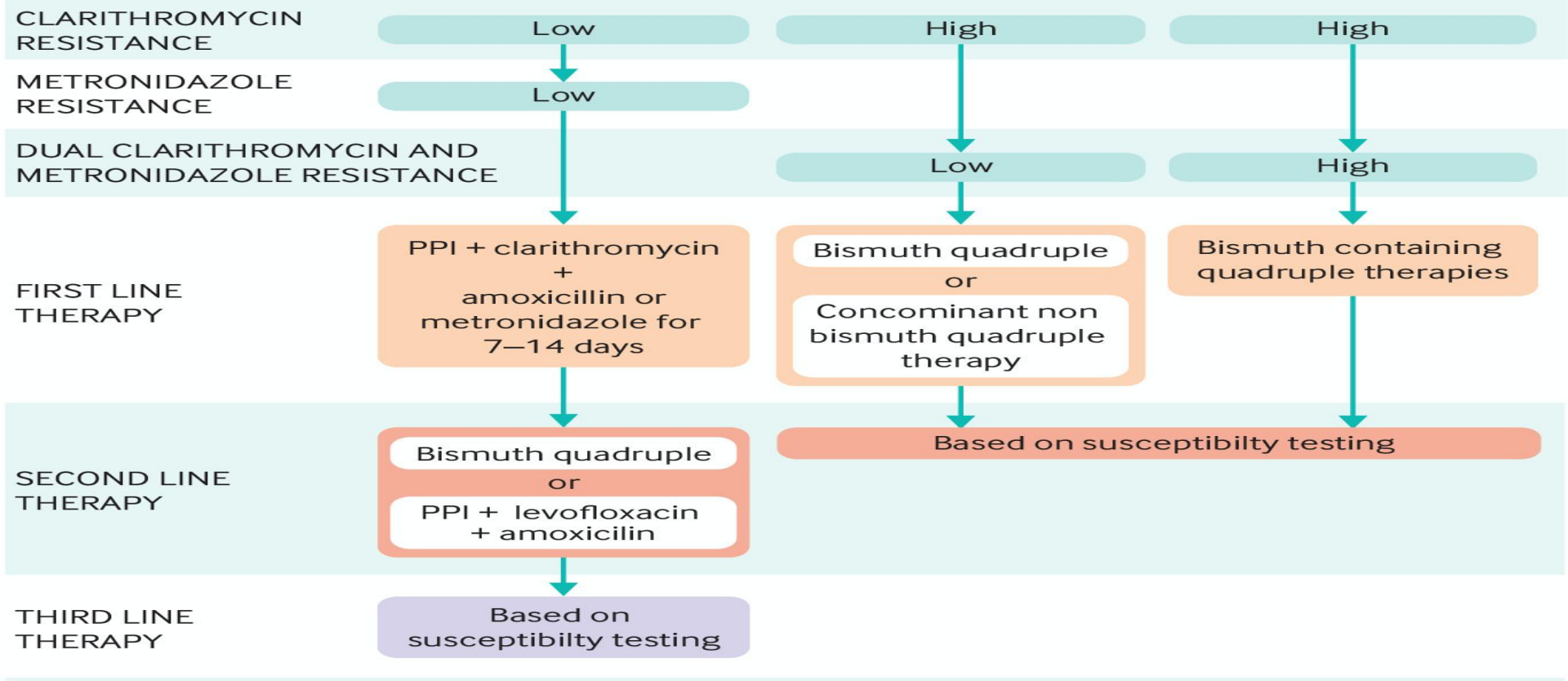
- Pseudomembranous colitis
- Dx on toxins being found in the stool
- Diarrhea, diarrhea, diarrhea
- Classically after treatment with which antibiotic?
- How do you treat?
- What is the route of treatment?


Vancomycin or
Metronidazole

What else can Metronidazole be used to treat?

Metronidazole

- Trichomonas vaginalis
  - Discharge
  - Cervix
- Bacterial vaginosis
  - Discharge
  - Microscopy
  - KOH 10% prep
- PUD



Bismuth is a salt with bactericidal effect

Bismuth quadruple is the triple therapy regimens with bismuth added in

Triple therapy: proton pump inhibitor plus two different antibiotics

Quadruple therapy: proton pump inhibitor plus three different antibiotics

Sequential therapy: for example first 7 days proton pump inhibitor plus amoxicillin, next 7 days proton pump inhibitor plus clarithromycin plus metronidazole

Concomitant therapy: for example simultaneously proton pump plus amoxicilin, clarithromycin and metronidazole for 10 days

Esophagus

Stomach

Small intestine

Duodenal ulcer

Gastric ulcer

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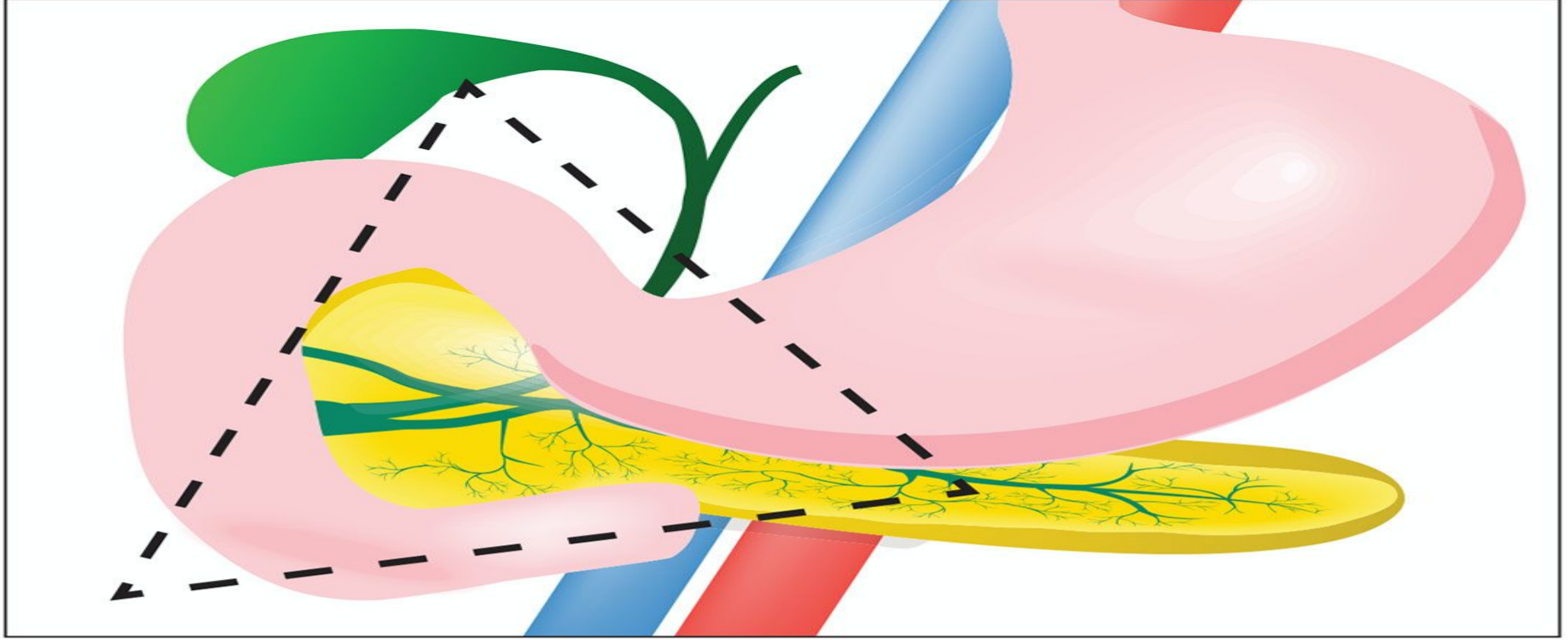


FIGURE

Unlike typical peptic ulcers, gastrinomas most commonly occur within this triangle (outlined by the hepatic portal vein, neck and body of the pancreas, and latter two-thirds of the duodenum) and hypersecrete gastrin—causing debilitating, recalcitrant acid reflux.

Credit: Designua / Shutterstock

Side Effect of Vancomycin?

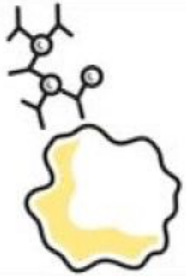
Vancomycin

What is Red Man Syndrome?

- An ADR to vancomycin IV that may occur with the **FIRST** dose of **vancomycin** ,OR when the drug is administered **TOO FAST**
- **Sx**: flushing of face and neck, pruritus, **hypotension**
- **Prevention**: slow the rate of infusion
  - Vancomycin should be given over AT LEAST IH
  - higher doses often ordered over 90 min

*If Red Man Syndrome develops, the nurse should **stop the infusion** and notify the HCP.*

***Benadryl** is often ordered to decrease the effect of the reaction.*

	Type I	Type II	Type III	Type IV
Immune reactant	IgE	IgG	IgG	T cells
Antigen	Soluble molecule	Cell associated molecule	Soluble molecule	Soluble or cell associated molecule
Graphic Icon				
Mechanism	IgE induced mast cell activation	Complement mediated phagocytosis	Tissue damage induced by immune complexes	T cell mediated inflammation or cytotoxicity
Examples	Allergic rhinitis, allergic asthma	Chronic urticaria (auto antibodies)	Serum sickness, arthus reaction	Multiple sclerosis, Contact dermatitis, Crohn's disease, Rheumatoid arthritis

但部份并非由 IgE 抗体引起的

ManIfestations of endocrine disease

A 42-year-old man presents to his GP for a yearly check-up offered in his area. His past medical history is unremarkable and he states that aside from some recent changes in his vision and headaches, both of which he attributes from some new glasses he bought a few weeks ago he feels well. On examination he is a tall and heavy set man who is very tanned from a recent holiday. Cardiovascular, respiratory and abdominal examination is unremarkable but you notice an area of untanned skin around his left ring finger and he tells you regretfully that recently his wedding band has become too tight and is being resized.

A. Cushing's syndrome

B. Acromegaly

C. Diabetes mellitus

D. Diabetes insipidus

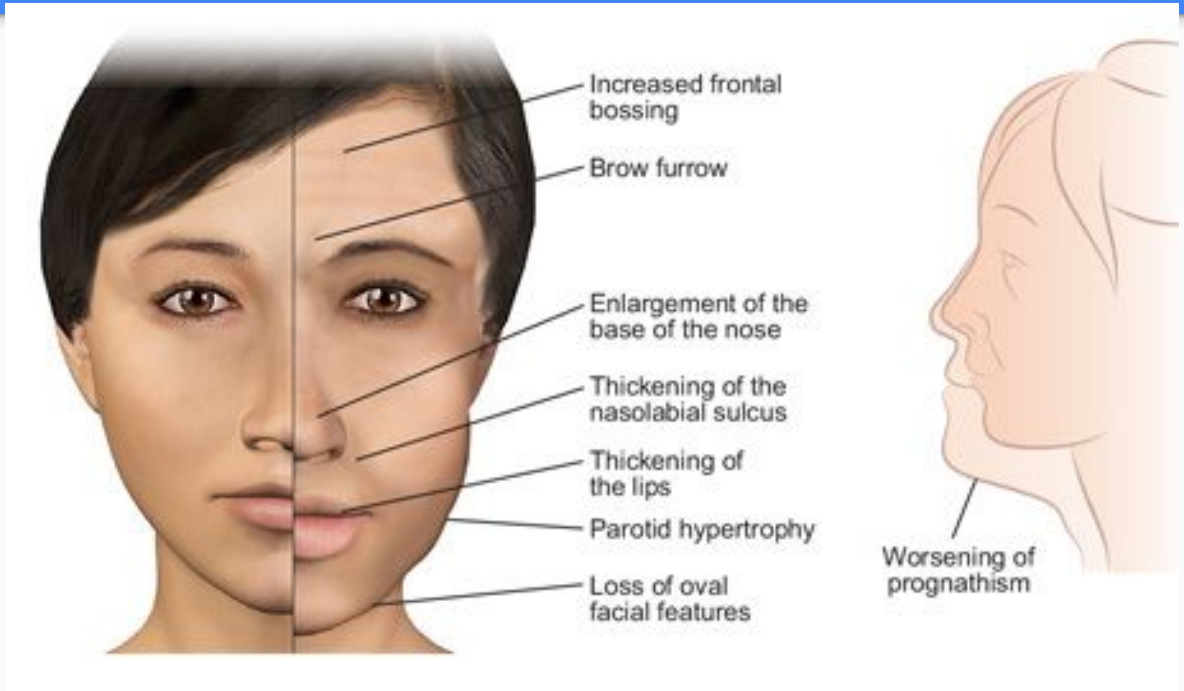
E. Phaeochromocytoma

F. Syndrome of inappropriate ADH secretion (SIADH)

G. Hypothyroidism

H. Hyperthyroidism

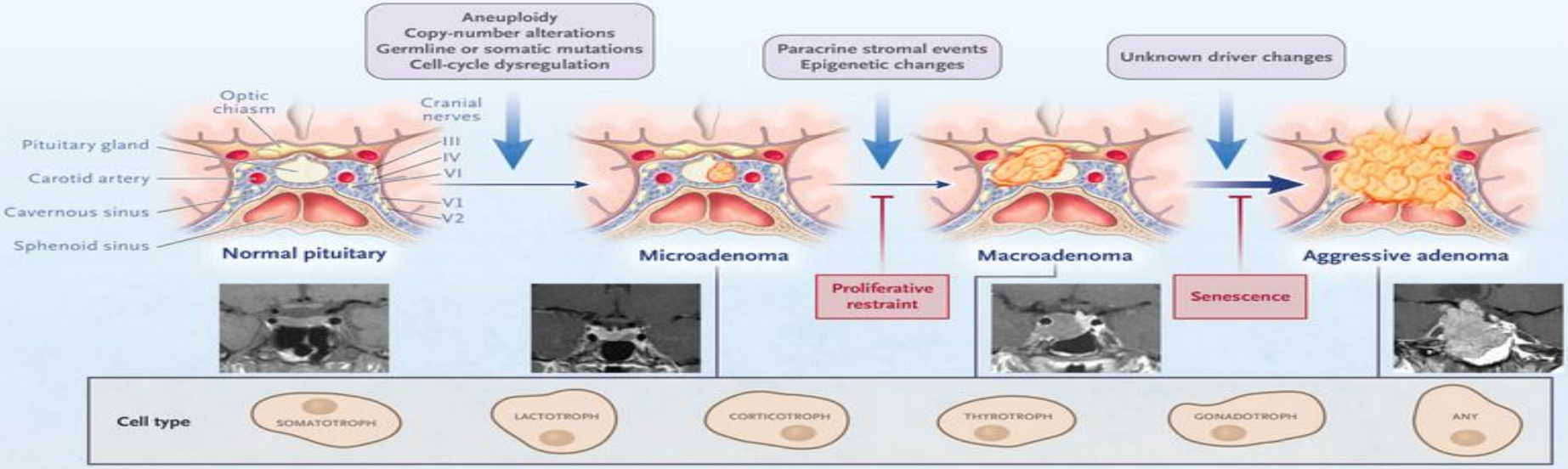
Acromegaly


Increased frontal bossing
Brow furrow
Enlargement of the base of the nose
Thickening of the nasolabial sulcus
Thickening of the lips
Parotid hypertrophy
Loss of oval facial features
Worsening of prognathism

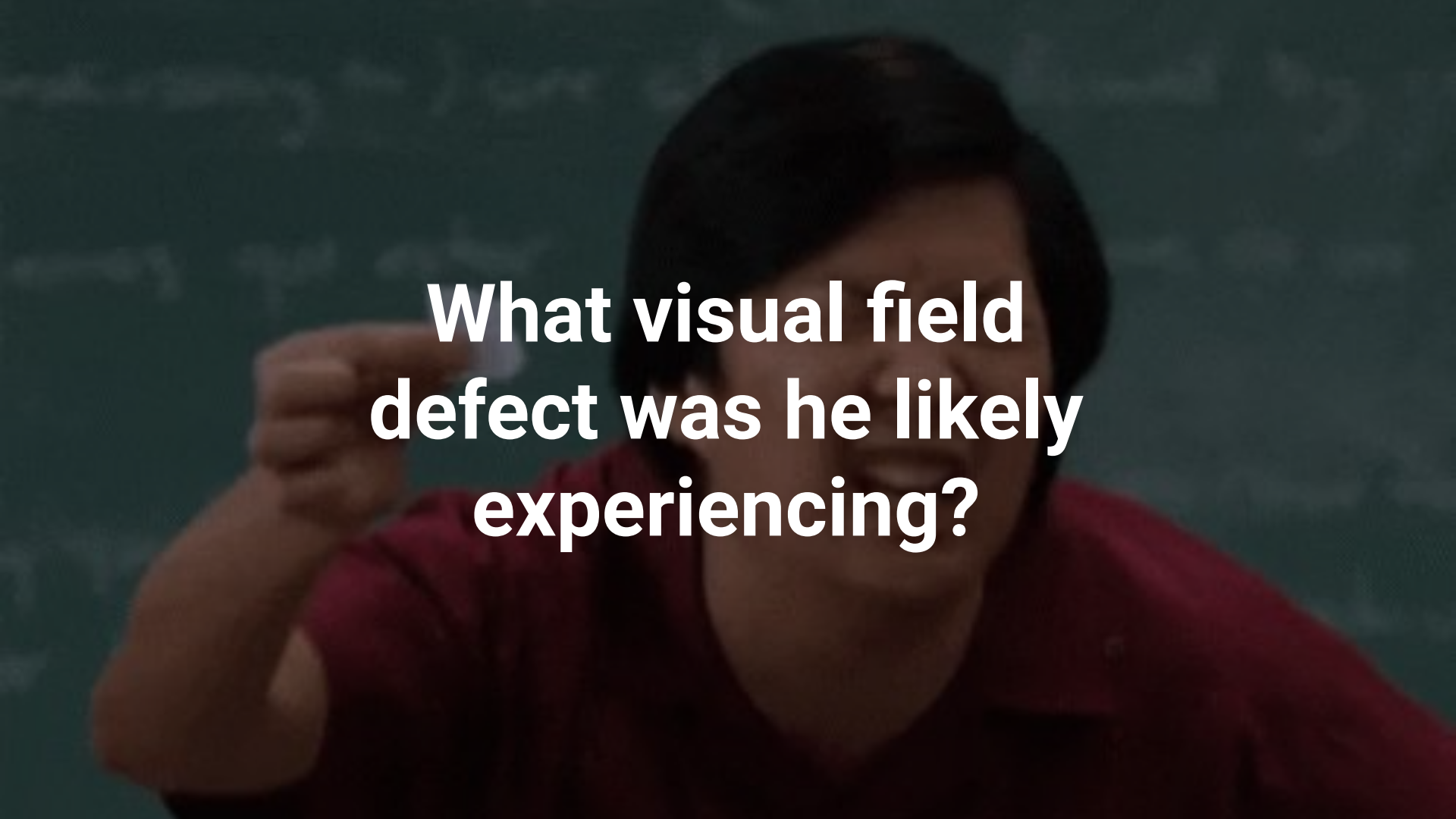
What blood test parameter would be elevated in this patient?

Insulin-Like Growth Factor 1

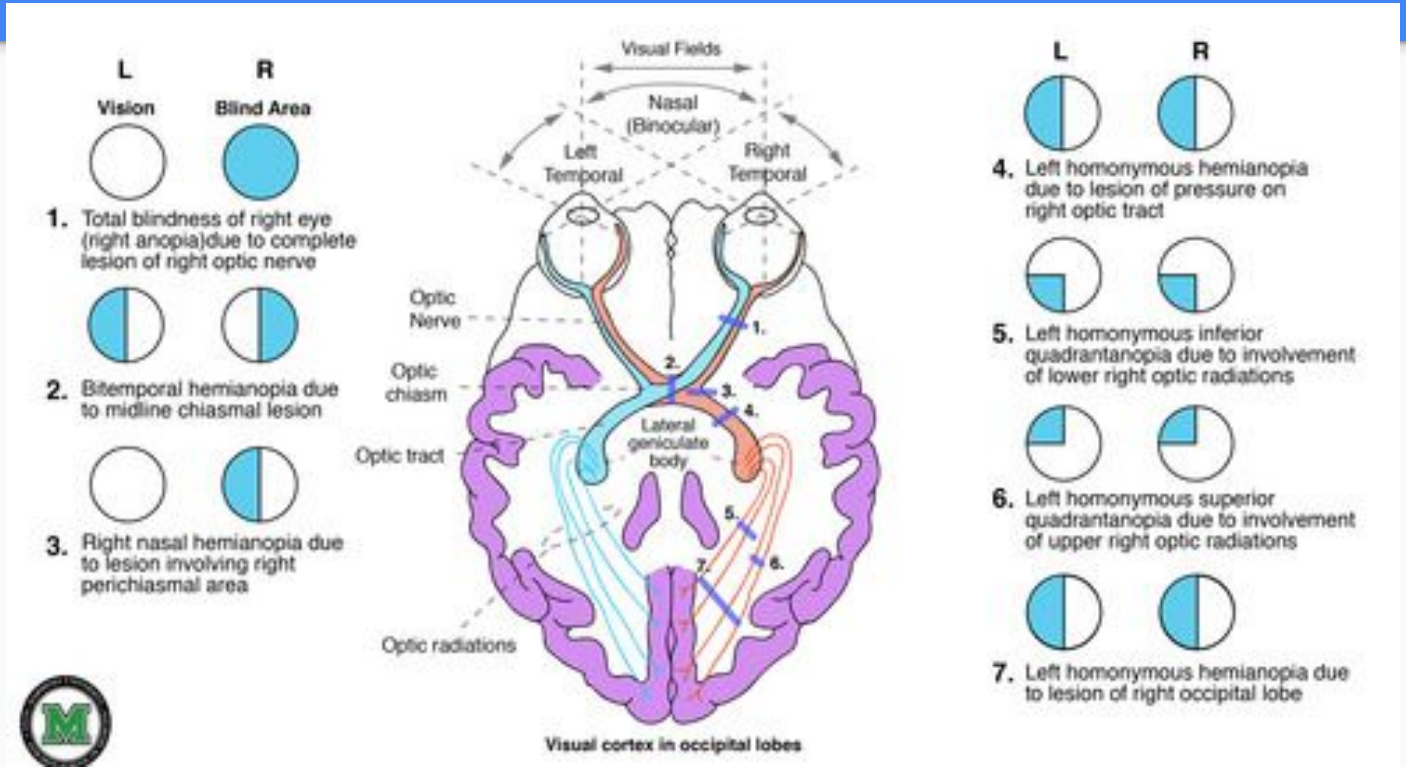
IGF-1



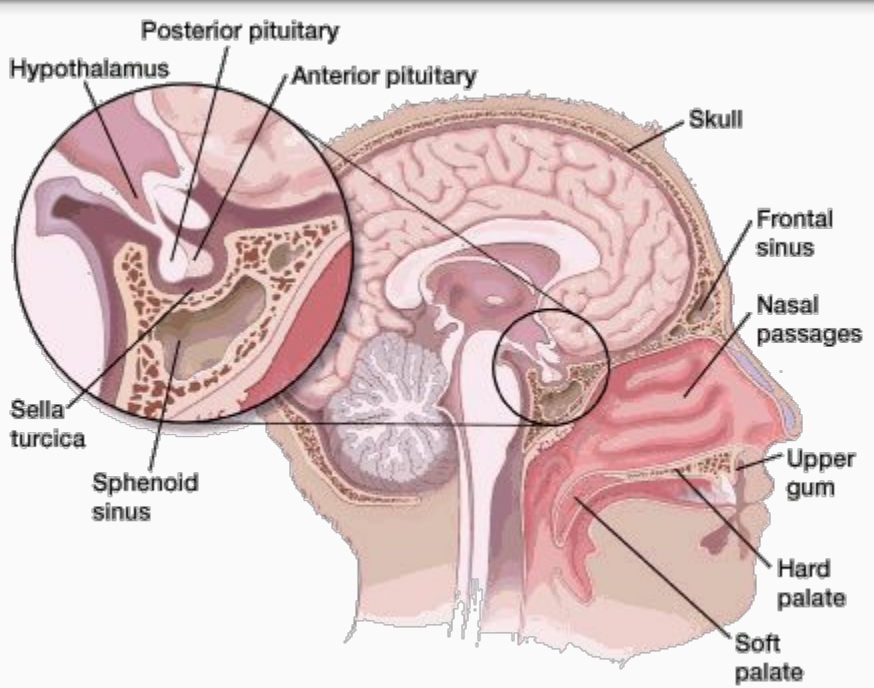
Secreted hormone	Growth hormone	Prolactin	Corticotropin	Thyrotropin	Luteinizing hormone or follicle-stimulating hormone or subunits	None
Relative prevalence (%)	8–15	30–60	2–6	<1	<1	14–55
Adenoma subtypes	Densely granulated Sparsely granulated Mammosomatotroph Mixed somatolactotroph Acidophil stem cell	Densely granulated Sparsely granulated Acidophil stem cell	Densely granulated Sparsely granulated Crooke's cell			Null cell Silent gonadotroph Silent corticotroph Silent somatotroph
Transcription factors	POU1F1	POU1F1 Estrogen receptor alpha	Tpit	POU1F1 GATA2	SF1 GATA2	Variable
Target hormone	IGF-1	Prolactin	Cortisol	T_4 or T_3	Estrogen or testosterone	None
Clinical phenotype	Acromegaly Gigantism	Hypogonadism Infertility Galactorrhea	Cushing's disease Hypercortisolemia	Hyperthyroidism	Hypergonadism	Pituitary failure

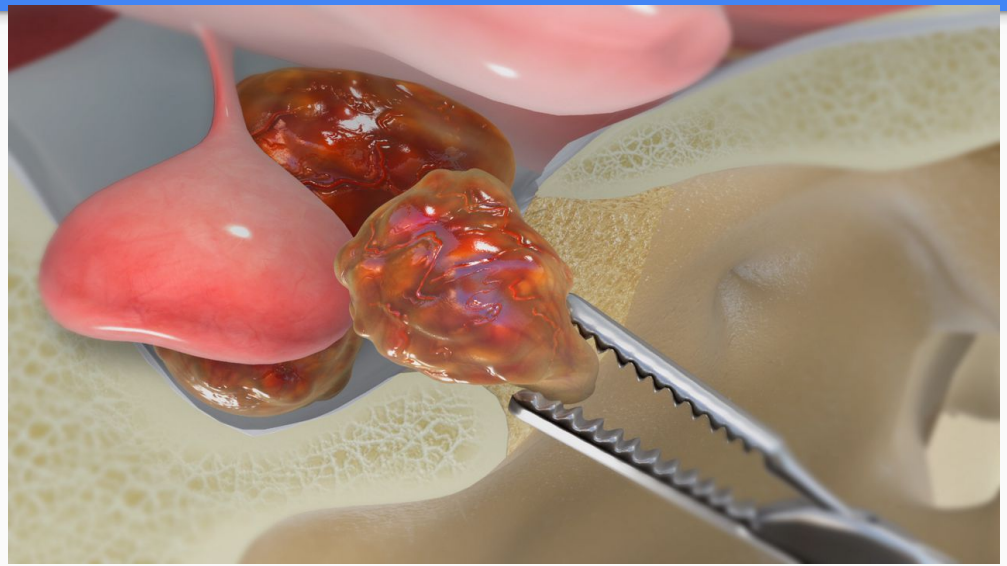

What visual field defect was he likely experiencing?

Bitemporal Hemianopia



Transsphenoidal Approach





Cushing’s Disease

Signs & Symptoms

Most people with Cushing syndrome will have:

- Upper body obesity (above the waist) and thin arms and legs
- Round, red, full face (moon face)
- Slow growth rate in children

Skin changes that are often seen:

- Acne or skin infections
- Purple marks (1/2 inch or more wide) called striae on the skin of the abdomen, thighs, and breasts
- Thin skin with easy bruising

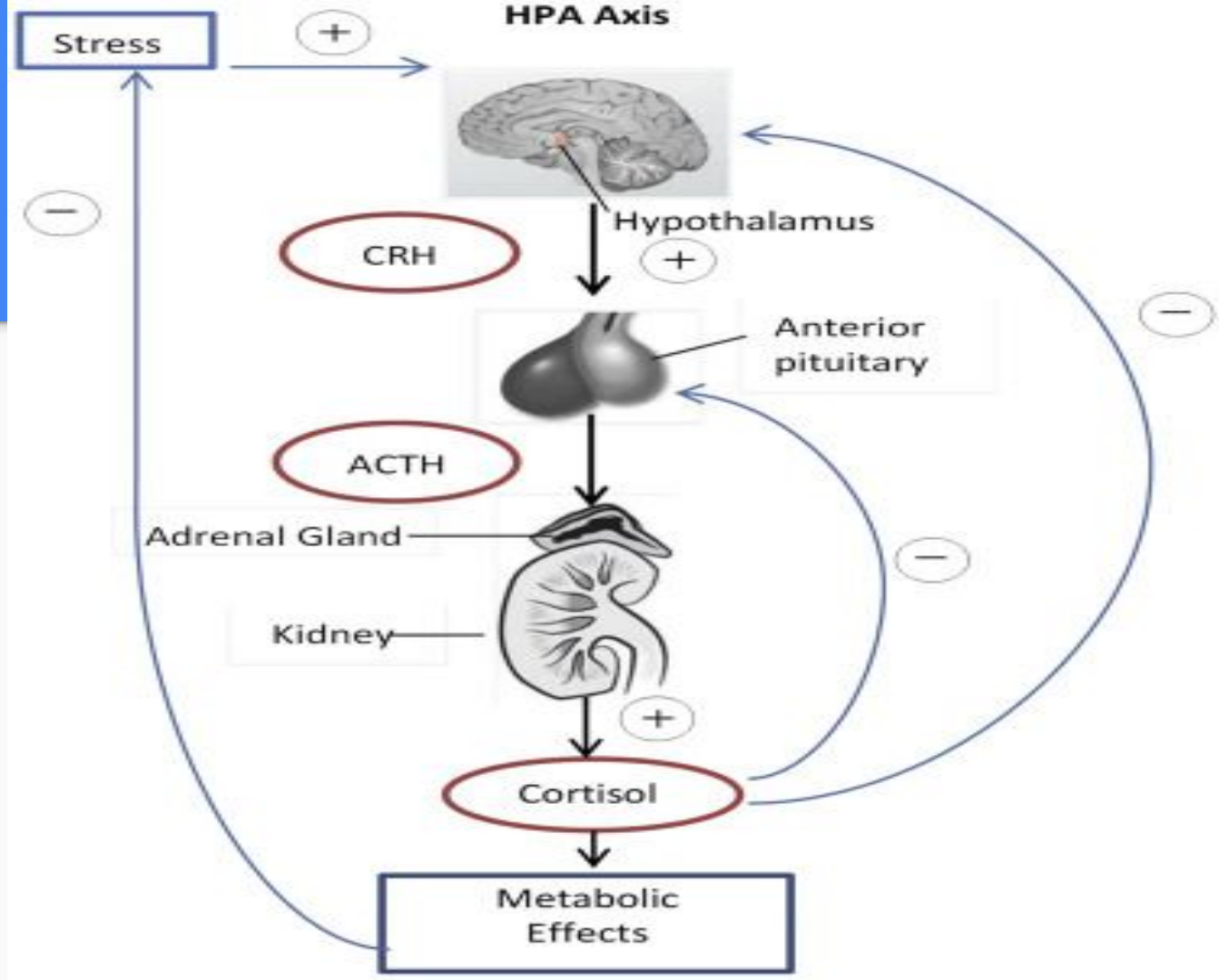
Muscle and bone changes include:

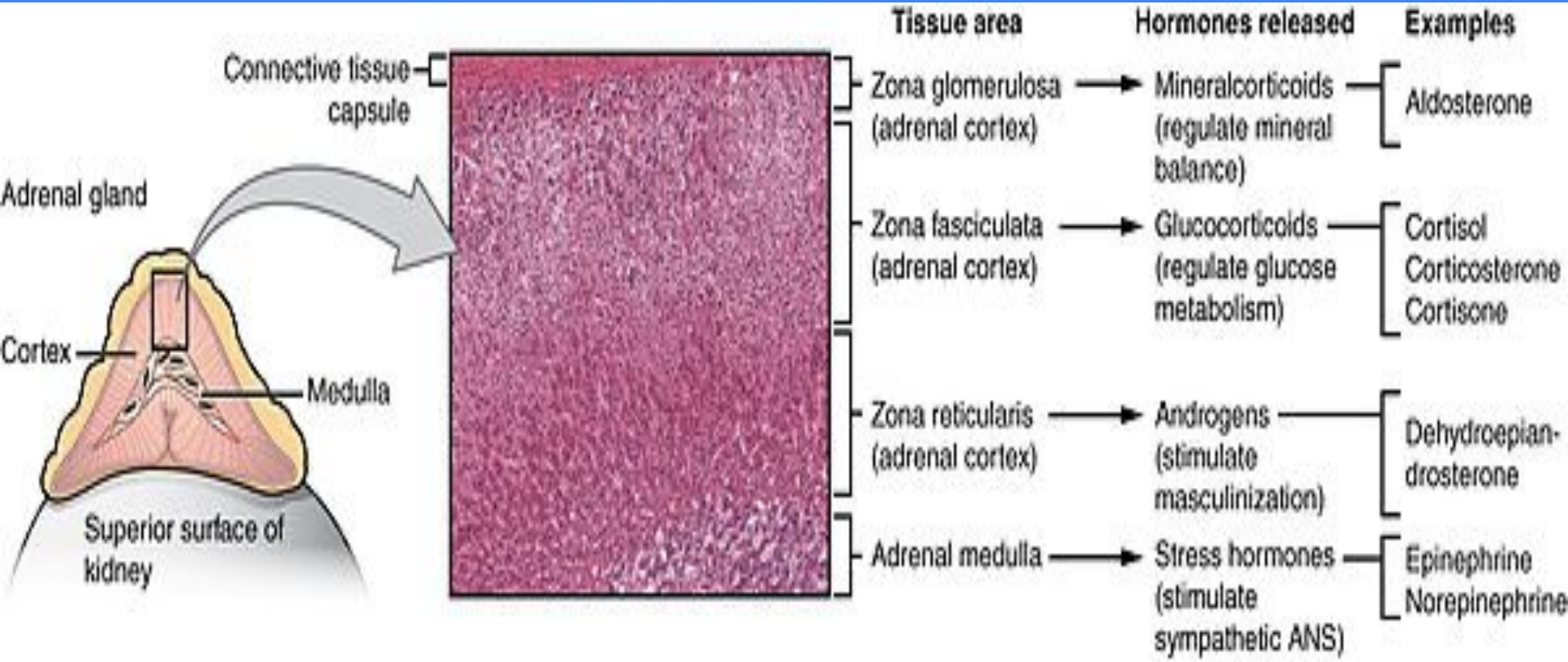
- Backache, which occurs with routine activities
- Bone pain or tenderness
- Collection of fat between the shoulders (buffalo hump)
- Thinning of the bones, which leads to rib and spine fractures
- Weak muscles

What if you’re in a small island?

Bilateral Adrenalectomy

HPA Axis


HPA Axis
Stress
+
Hypothalamus
CRH
+
Anterior pituitary
ACTH
Adrenal Gland
Kidney
+
Cortisol
Metabolic Effects
−
−
−


Connective tissue capsule
Adrenal gland
Cortex
Medulla
Superior surface of kidney
Tissue area
Hormones released
Examples
Zona glomerulosa (adrenal cortex)
Mineralcorticoids (regulate mineral balance)
Aldosterone
Zona fasciculata (adrenal cortex)
Glucocorticoids (regulate glucose metabolism)
Cortisol
Corticosterone
Cortisone
Zona reticularis (adrenal cortex)
Androgens (stimulate masculinization)
Dehydroepian-drosterone
Adrenal medulla
Stress hormones (stimulate sympathetic ANS)
Epinephrine
Norepinephrine

Extended Matching Cont’d

3 years after her bilateral adrenalectomy, she start to get dark and gains weight. What is the most likely diagnosis?

Nelson’s Syndrome

NELSON ‘s SYNDROME

- Aggressive pituitary macroadenoma and very high ACTH levels causing pigmentation
- Nelson ‘s syndrome can be prevented by pituitary irradiation


BLACK LIVES
MATTER