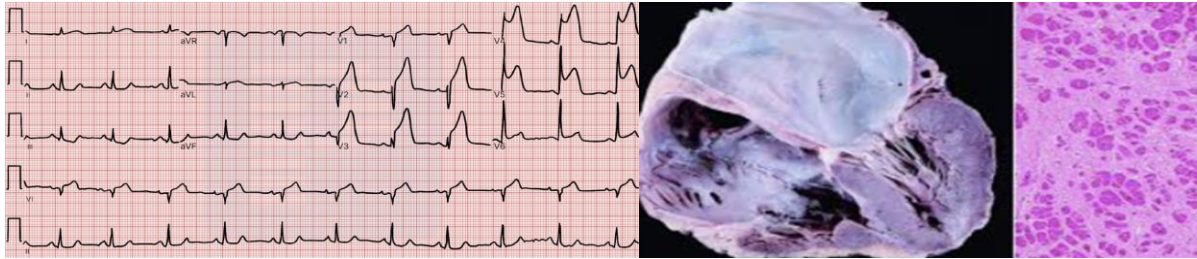


## Cardiology



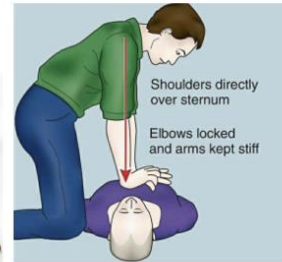
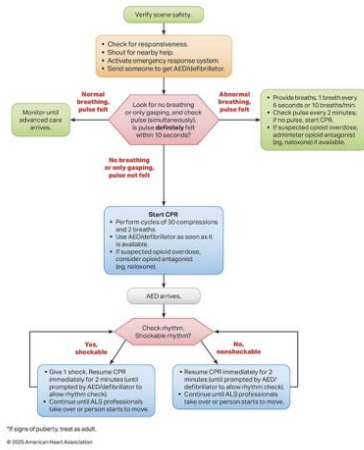
Topics color coded red are optional for FMG students

### Crashing patient



Q. You are posted in CCU and handling a 50 year old man admitted with diagnosis of Anterior wall STEMI. He becomes suddenly unconscious and resident identifies him as suffering from pVT. Which of the following is the first step to be done in management of this case?

- Check Carotid pulse for 6 seconds and asses breathing efforts
- Verify scene safety
- Immediately Place defibrillator leads and give DC shock
- Give chest compressions



1. Check scene safety
2. Check responsiveness
3. Shout for help
4. .
5. .
6. Look for
7. Start @

Rate	
Depth of sternal depression	
Site of chest compression	
Rationale	Maintain Cerebral blood flow
Fulcrum of movement	Hip joint Shoulder joint Elbow joint Wrist joint



8. AED arrives

Left pad placement	
Right pad placement	

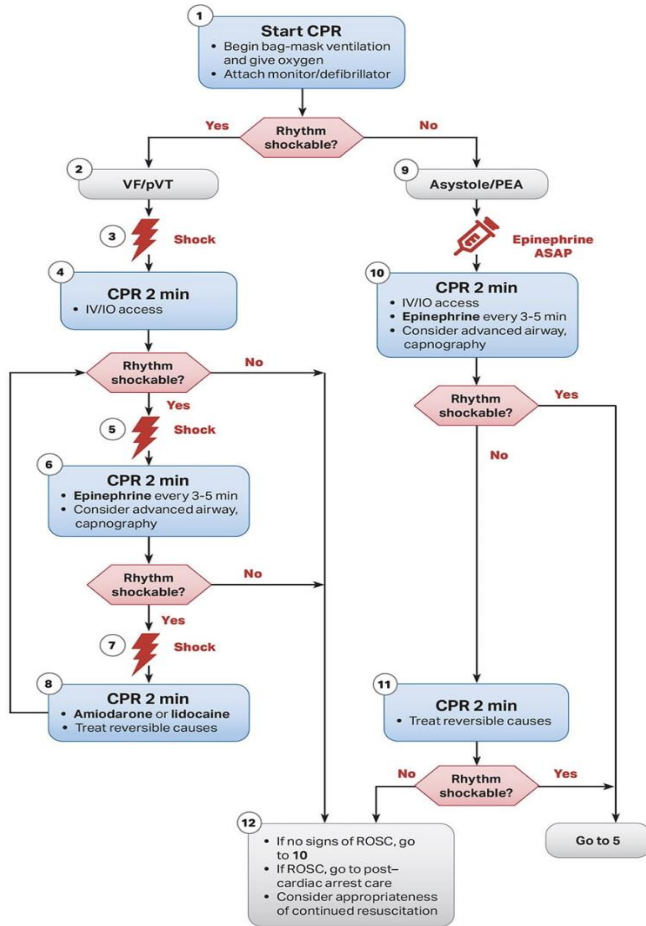
9. Check rhythm and if shockable give one shock and resume CPR, till AED gives next prompt for rhythm check . Continue till ALS team arrives

***Extra mile***

MC ribs fracture during CPR	
MC solid organ traumatised during CPR	



### Adult Cardiac Arrest Algorithm (VF/pVT/Asystole/PEA)

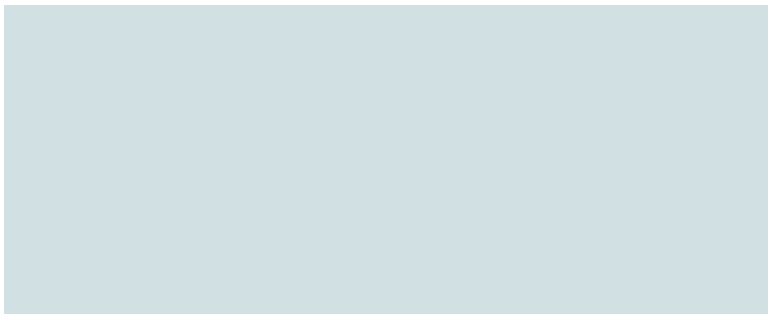
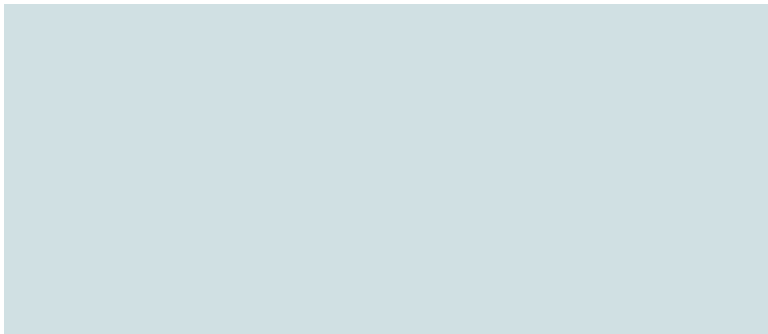
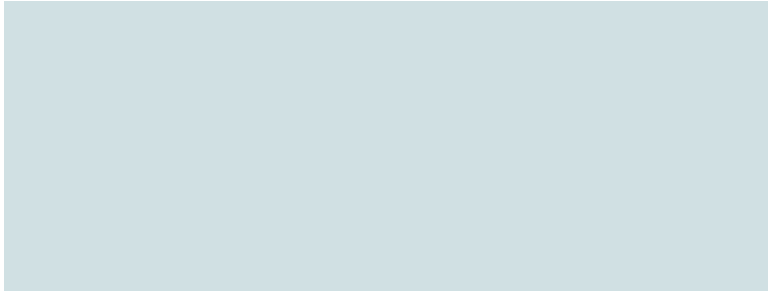


- High-Quality CPR**
- Push hard (at least 2 inches [5 cm]).
  - Push fast (100-120/min) and allow complete chest recoil.
  - Minimize interruptions in compressions.
  - Avoid excessive ventilation.
  - Change compressor every 2 minutes, or sooner if fatigued.
  - If no advanced airway, use 30:2 compression-ventilation ratio.
  - If advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.
  - Continuous waveform capnography
    - If ET CO<sub>2</sub> is low or decreasing, reassess CPR quality.
- Shock Energy for Defibrillation**
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J; if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered).
  - **Monophasic:** 360 J
- Drug Therapy**
- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
  - **Amiodarone IV/IO dose:** First dose: 300 mg bolus; Second dose: 150 mg or **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg; Second dose: 0.5-0.75 mg/kg
- Advanced Airway**
- ET intubation or supraglottic advanced airway
  - Continuous waveform capnography or capnometry to confirm and monitor ET tube placement
- Reversible Causes**
- Hypovolemia
  - Hypoxia
  - Hydrogen ion (acidosis)
  - Hypo-/hyperkalemia
  - Hypothermia
  - Tension pneumothorax
  - Tamponade, cardiac
  - Toxins
  - Thrombosis, pulmonary
  - Thrombosis, coronary

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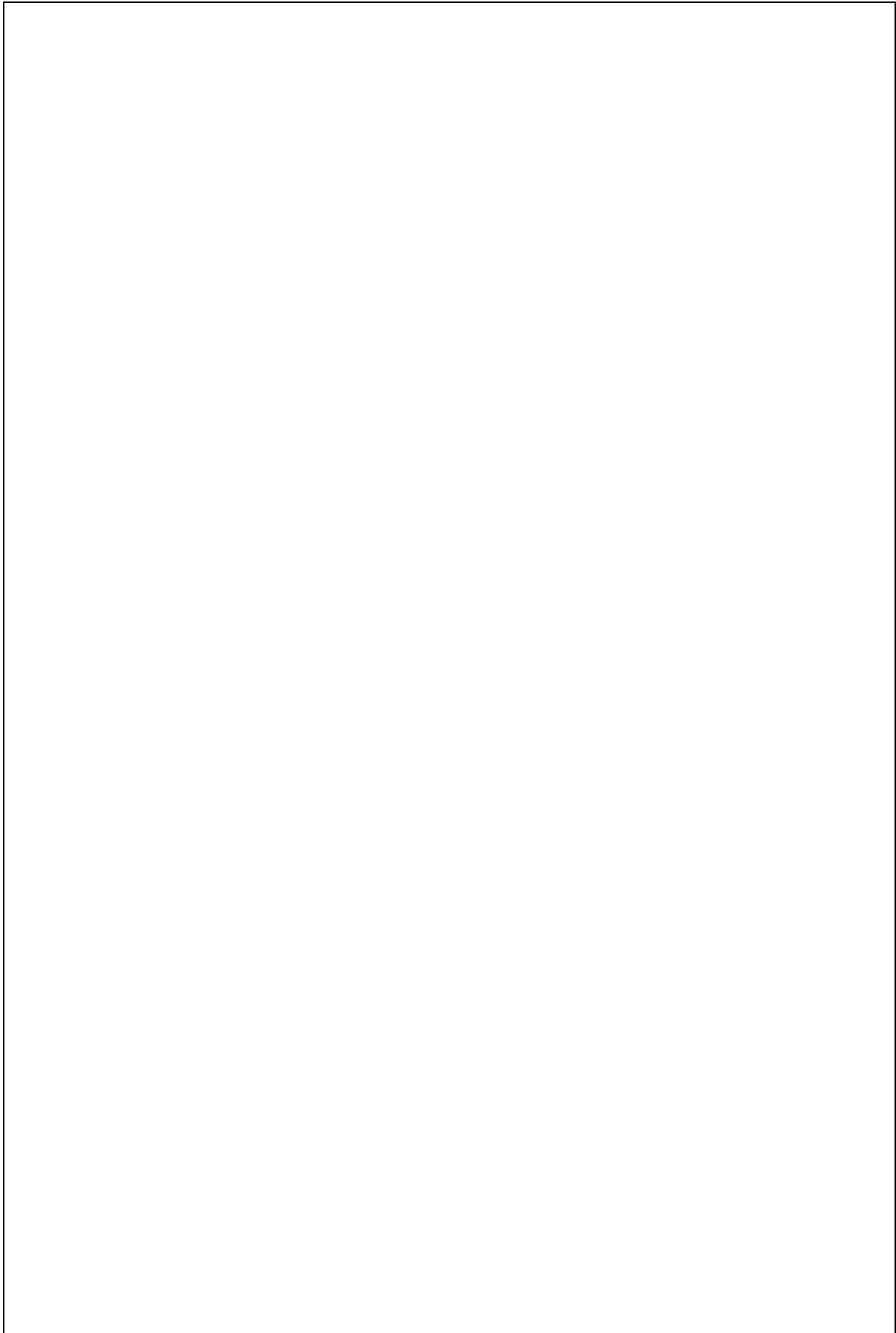


## Rx for pVT and Ventricular fibrillation





## Asystole management

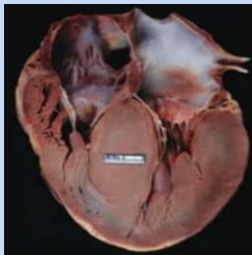


## ABCD of Pulse

**Alternans**

**Anacrotic**

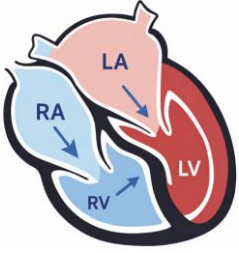
**Bisferiens**



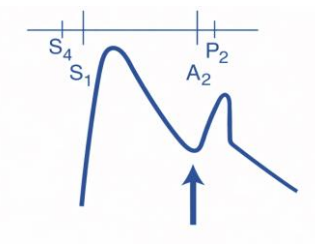
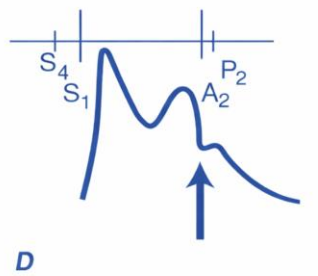
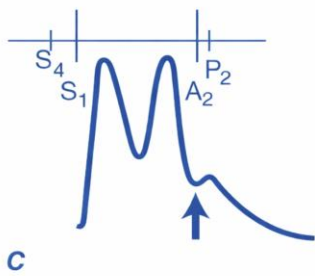
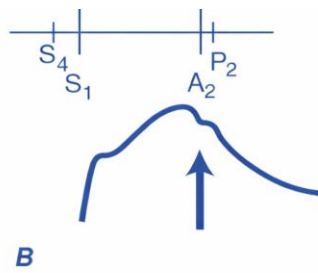
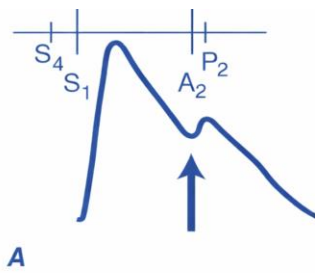
**Collapsing pulse with pistol shot sounds**



**Dicrotic pulse**

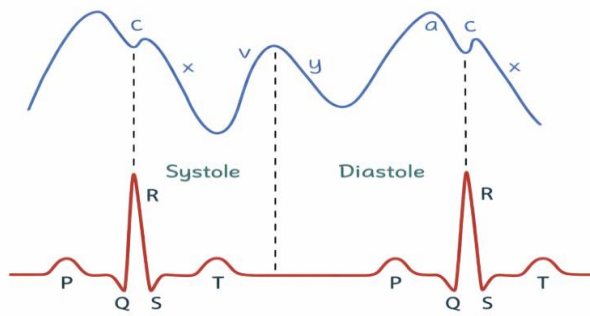
<p><b>Pulsus paradoxus</b></p> <p><b>SBP falls &gt; 12 mm Hg on deep inspiration</b></p> 	<p><b>Mnemonic: MR-CAP</b></p> <p><b>Massive PE</b></p> <p><b>RV MI</b></p> <p><b>Cardiac tamponade</b></p> <p><b>Asthma (status asthmaticus)</b></p> <p><b>Pericarditis Constrictive</b></p>
<p><b>Irregularly irregular pulse</b></p>	

**Line diagrams for types of pulses**



## Abnormalities of JVP

Normal JVP value: \_\_\_\_\_ is evaluated in \_\_\_\_\_



**Kussmaul sign:** Paradoxical rise of JVP on deep inspiration

**Mnemonic:** Cops restrict right heart

Large a waves	
Absent a waves	
Cannon a waves Frog sign	
Absent x descent	
Absent y descent	
Steep x and absent y descent	
Steep x and steep y descent	



## Pericardial diseases

	Acute pericarditis	Pericardial effusion
ECG		
CXR		
Rx	Dressler syndrome	TB Oat cell Cancer



## Cardiac tamponade vs Constrictive pericarditis

	Tamponade	Constrictive pericarditis
<b>Pulse</b>		
<b>JVP character</b>		
<b>JVP waves</b>		
<b>BP</b>		
<b>ECG</b>	<b>Low voltage ECG</b>	<b>Low voltage ECG</b>
<b>CXR</b>		
<b>Rx</b>	<b>Pericardiocentesis</b>	



## Murmurs

Ejection systolic murmurs	Pansystolic murmurs
Mnemonic: <b>PASS</b>	Mnemonic: <b>MTR-Vada</b>



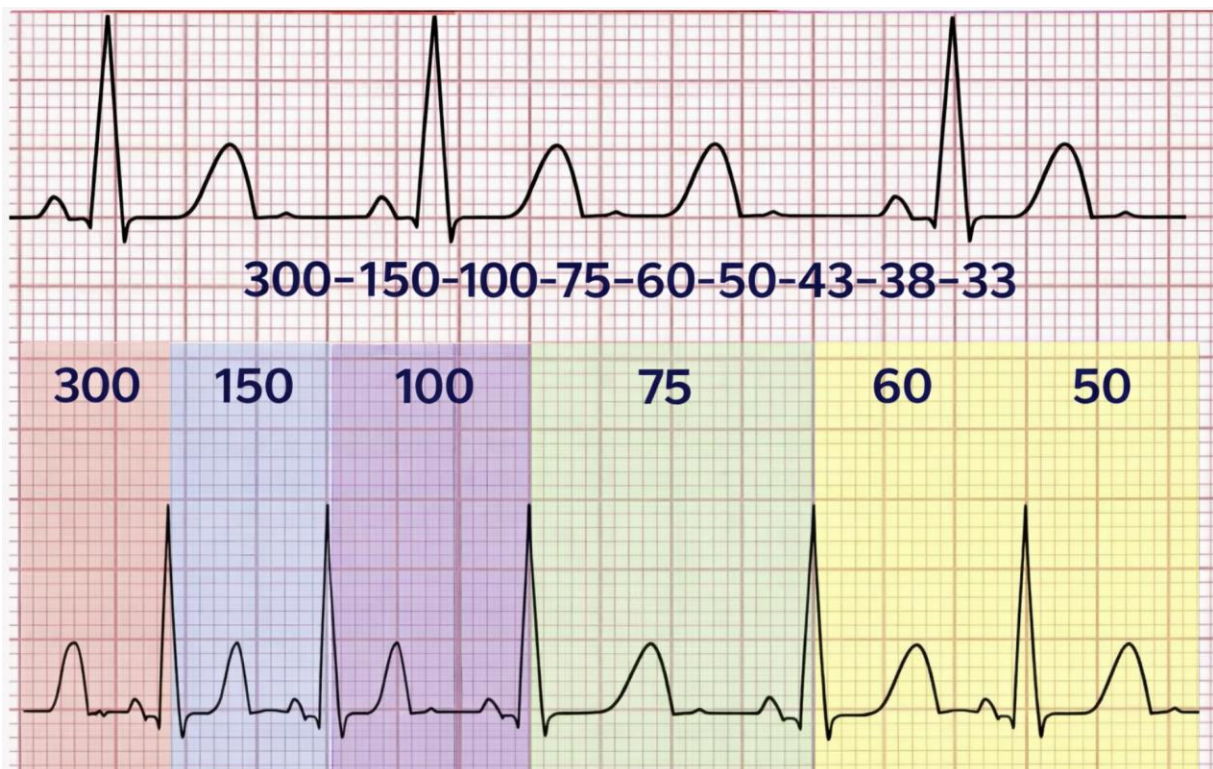
Early diastolic murmur	Mid diastolic murmur
<p><b>GAP: Mnemonic</b></p>	<p><b>CAM: Mnemonic</b></p>

## Continuous murmur

Connaught place main Hum-Tum!

1. Coarctation of Aorta
2. PDA
3. Mammary souffle
4. Venous hum
5. Peripheral pulmonic stenosis

## How to calculate heart rate under 5 seconds



## Tachy and Brady Arrhythmias

### Atrial fibrillation



ECG criteria

Rate control

Rate control in patients with coexistent poorly controlled asthma

Rate control in patients with coexistent COPD

Rate control in patients with acute pulmonary edema

Recent onset atrial fibrillation with crashing BP

### How to manage long standing atrial fibrillation

How to decide on Anticoagulation?

Drugs used for anticoagulation in Atrial fibrillation

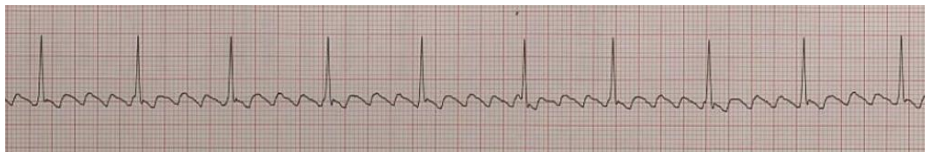
Rhythm control

## Multifocal atrial tachycardia vs Atrial flutter

ECG findings of MAT



ECG findings of Atrial flutter



Rx for Atrial flutter	RX for MAT

## Paroxysmal supraventricular tachycardia



*Management of crashing patient or SBP < 90/60 mm Hg*

*Management of patient with SBP > 90/60 mm Hg*

**Home management:** Etipamil is a short-acting intranasal calcium channel blocker used for paroxysmal supraventricular tachycardia



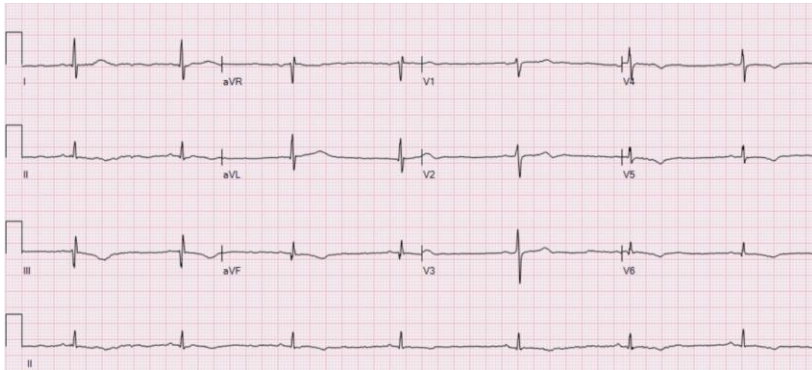
*Best management for prevention of PSVT episodes*

## Ventricular tachycardia



- AV dissociation
- Cannon a waves in JVP
- Premature ventricular complexes
- Why PVC is bad?
- Capture beats
- Fusion beat

## Management of symptomatic bradycardia



1. IV Atropine 1 mg IV, max dose = 3 mg
2. Other drugs
3. Temporary pacing

## First degree heart block

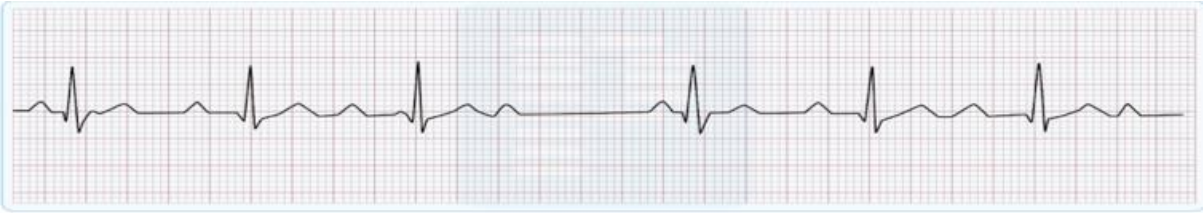


### Causes

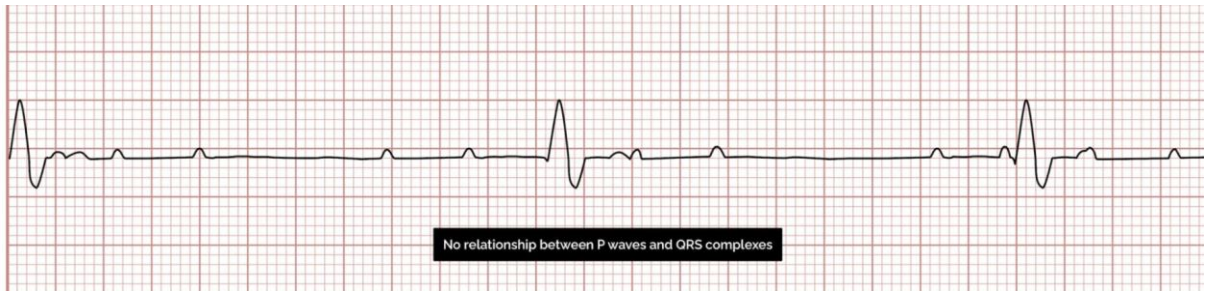
1. Physiological
2. Rheumatic fever
3. Sarcoidosis
4. Hemochromatosis
5. Endo-myocardial fibroelastosis

### ECG findings

## Mobitz I vs Mobitz II heart block



### 3rd degree heart block





**Summary of ECG Special findings**

<b>P Wave</b>	<b>P- Pulmonale: RAE</b> <b>P- Mitrale: LAE</b> <b>Pseudo-P- Pulmonale</b> <b>Absent P Wave</b>
<b>Q waves</b>	<b>Deep Q waves</b> <b>S1Q3T3 pattern</b>

<b>Short PR interval</b>	<b>Prolonged PR interval</b>
<b>Wolf Parkinson white syndrome</b>	<b>PR interval remains normal in mobitz II heart block</b>



**qRS Complex abnormalities**

**Narrow qRS tachycardia**

**Broad qRS tachycardia**

**Delta waves**

**J waves/ Osborn waves**

**Epsilon waves**

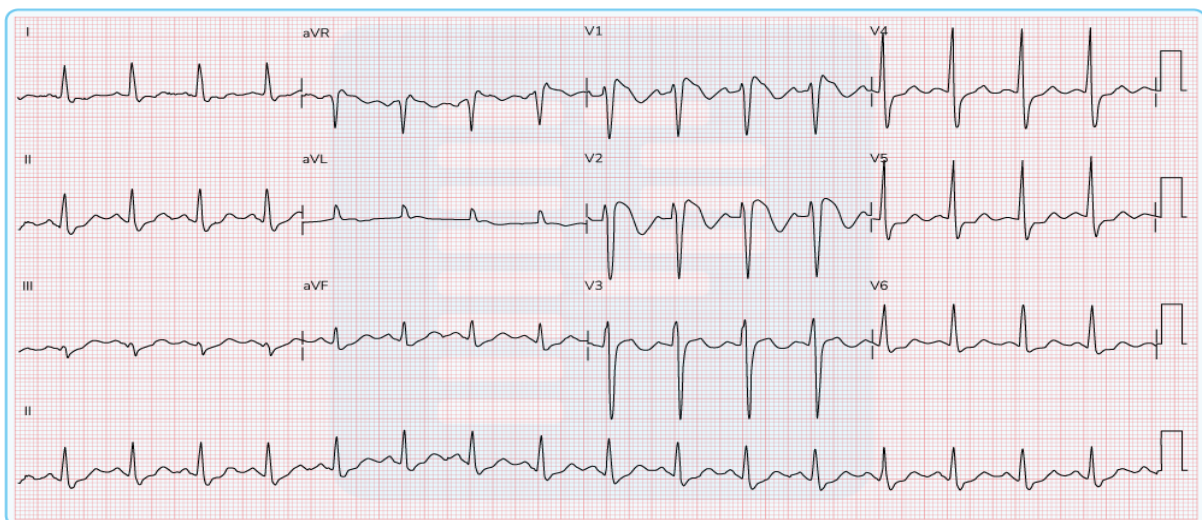


ST elevation	ST depression



<b>T wave inversion</b>	<b>Tall Tented T waves</b>	<b>Hyperacute T waves</b>

<b>Brugada Syndrome</b>	<b>ARVD</b>
<b>Pathophysiology</b>	<b>Pathophysiology</b>
<b>Agonal breathing and syncope</b>	<b>Palpitations and recurrent syncope</b>
<b>ECG</b>	<b>ECG</b>
<b>Rx</b>	<b>Rx</b>



## Infective endocarditis

### Native valve-IE

- Hospital acquired:
- Community acquired:

### Prosthetic valve endocarditis

< 2 months: CONS

2-12 months: CONS

> 12 months:

TAVR\_ IE:

CIED-IE: CONS

### Definite IE

1. *Pathologic criteria* (microorganisms or active endocarditis identified in a vegetation/ intracardiac abscess from cardiac tissue prosthetic material, arterial embolus)
2. *Clinical criteria* (2 major or 1 major + 3 minor or 5 minor)



## Minor Criteria

- A. Predisposition:** Previous history of IE, injection drug use, prosthetic valve, previous valve repair, congenital heart disease (e.g. bicuspid AV), CIED, more than mild regurgitation or stenosis hypertrophic cardiomyopathy
- B. Fever.** T > 38.0°C (100.4°F)
- C. Vascular phenomenon:** arterial emboli, septic pulmonary infarcts, mycotic aneurysm , intracranial hemorrhage, conjunctival hemorrhage, Janeway lesions, cerebral / splenic abscess purulent purpura
- D. Immunologic phenomena:** Roth spots, positive Rheumatoid factor, Osler nodes, glomerulonephritis
- E. Microbiologic evidence:** positive blood cultures (not meeting above criteria)

Positive culture PCR, or other nucleic acid -based test for an organism consistent with IE from a non - endovascular site or single finding of a skin bacteria by PCR on a valve or wire without additional clinical or microbiological supporting evidence

- A. Imaging Criteria:** Abnormal metabolic activity on FDG-PET/T within 3 months of implantation of a prosthetic valve, ascending aortic graft, intracardiac device lead
- B. Physical exam criteria:** New valvular regurgitation on auscultation

### **Microbiological Criteria**

1. **Positive blood culture:** Microorganisms that occasionally or rarely causes IE isolated from 3 or more separate blood culture sets (Non-Typical)

### **2. Positive laboratory tests**

-Positive polymerase chain reaction (PCR for *Coxiella burnetti*, *Bartonella* spp., or *Tropheryma whippelii* from blood

-Single blood culture growing *C. burnetti* or phase I IgG Ab titer  $\geq 1: 800$

-Indirect immunofluorescence assays ( IFA) for IgM Ab and IgG Ab to *B. Henselae* or *B. Quintana* with IgG Ab titer  $\geq 1:800$

### **Imaging Criteria**

1. Echocardiography or cardiac CT showing vegetation, valvular/ leaflet perforation/ aneurysm, abscess, pseudoaneurysm or intracardiac fistula

2. FDG -PET /CT with abnormal metabolic activity involving a native or prosthetic valve, ascending aortic graft, intracardiac device leads or other prosthetic material

**Surgical Criteria:** Evidence of IE documented by direct inspection during heart surgery



## Must know Cardiac emergencies

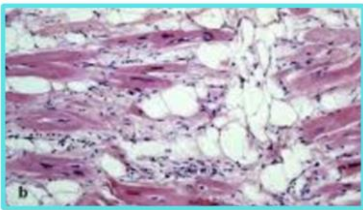
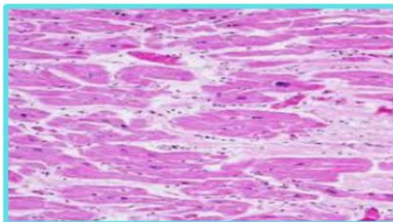
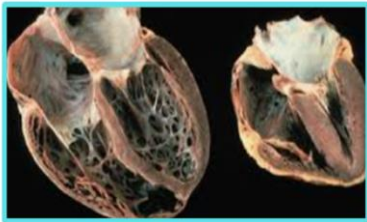
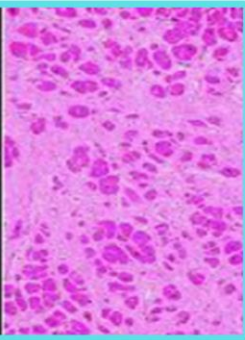
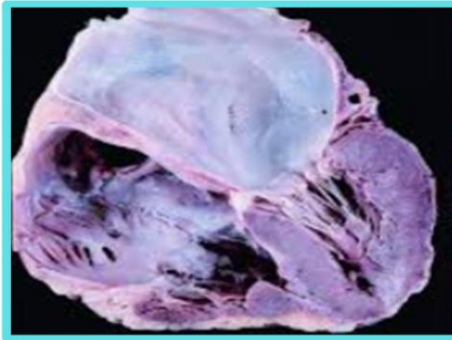
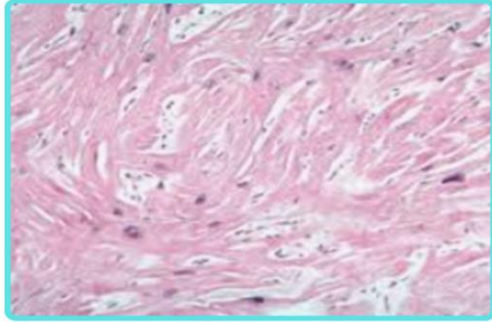
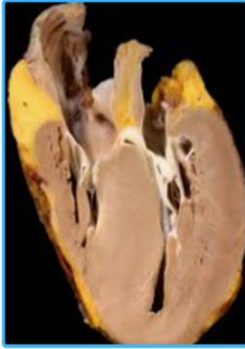
Acute pulmonary edema due to Hypertensive crisis	Cardiogenic shock	HFrEF

**HFrEF**



<b>Hypertensive emergency leading to acute pulmonary edema</b>	Note: beta blockers are contraindicated in Acute HF
<b>Hypertensive emergency leading to hemorrhagic stroke</b> <b>Target BP</b>	
<b>Hypertensive urgency</b>	
<b>Posterior reversible encephalopathy syndrome</b>	
<b>Malignant hypertension</b>	
<b>Scleroderma crisis</b>	
<p>Nitroprusside acts within seconds and has onset within 2 minutes. It breaks down to produce NO (acts via cGMP) and Cyanide. Must be given in light protected infusion set.</p> <p>Antidote for toxicity: Sodium thiosulfate (preferred) and Hydroxocobalamin (binds cyanide)</p>	

## Cardiomyopathy (Histopathology)



## Cardiomyopathy clinical details

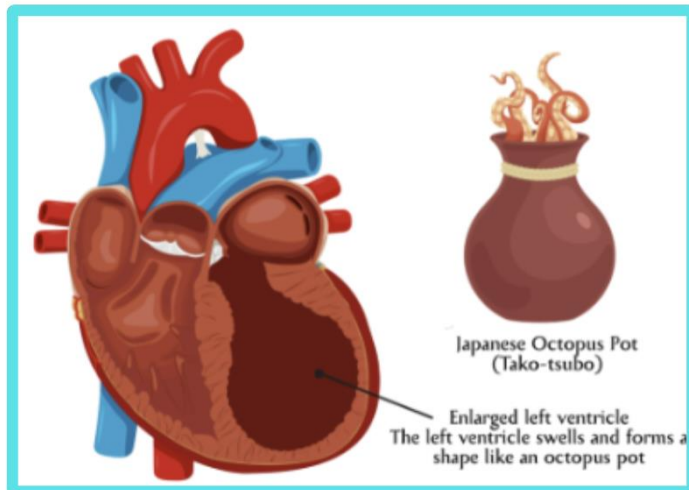
	Hypertrophic	Dilated
<b>Histology</b>	Banana shaped cavity Sub-valvular aortic stenosis Systolic anterior movement of anterior valve Predominant diastolic malfunction	
<b>Pulse</b>		
<b>Murmur</b>		
<b>Treatment of choice</b>		

## Restrictive cardiomyopathy

<b>Histology</b>	
<b>Pulse</b>	
<b>JVP finding</b>	
<b>IOC</b>	Cardiac MRI vs endomyocardial Biopsy
<b>Cardiac catheterization</b>	

## Takotsubo cardiomyopathy/ Broken heart syndrome

- Mimics MI presentation
- Elevated cardiac biomarkers with ST segment elevation
- Diagnosis made in cath lab
- Management :





## Myocardial infarction

Earliest ECG finding

ECG finding that persists for whole life/ Old MI

First cardiac biomarker to rise in MI

Last to rise in MI

Cardiac biomarker of reinfarction > 72 hours

Treatment of Choice for STEMI

First medical contact to balloon time

Window period for thrombolysis

Door to needle time ( cath lab facility is not available)

Rescue PCI (< 50% ST-segment resolution at 60–90 min after fibrinolysis)



## Complications of myocardial infarction

Most common complication in few hours:

MC cause of death > 24 hours

Death 3-5 days

MC valvular lesion seen in MI

Dressler syndrome

### Mnemonic

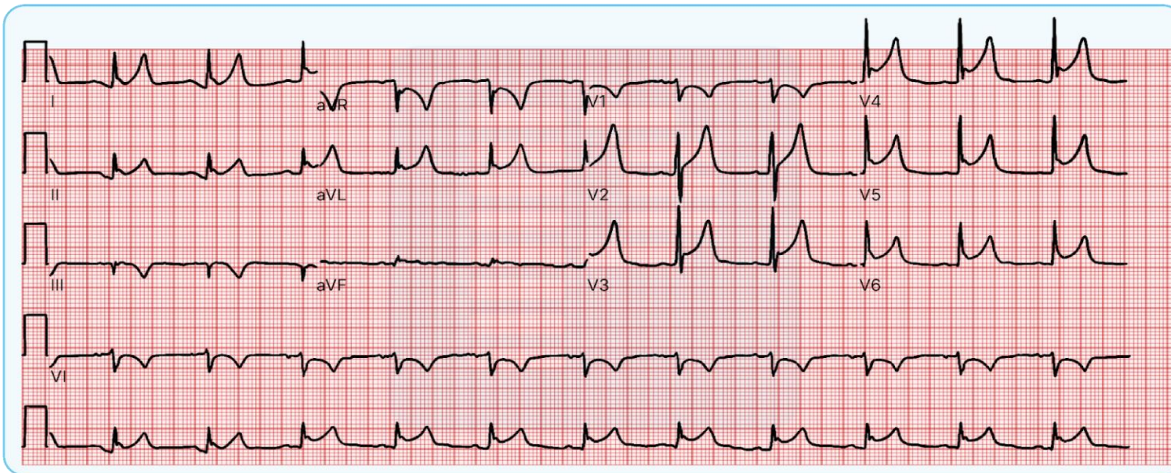
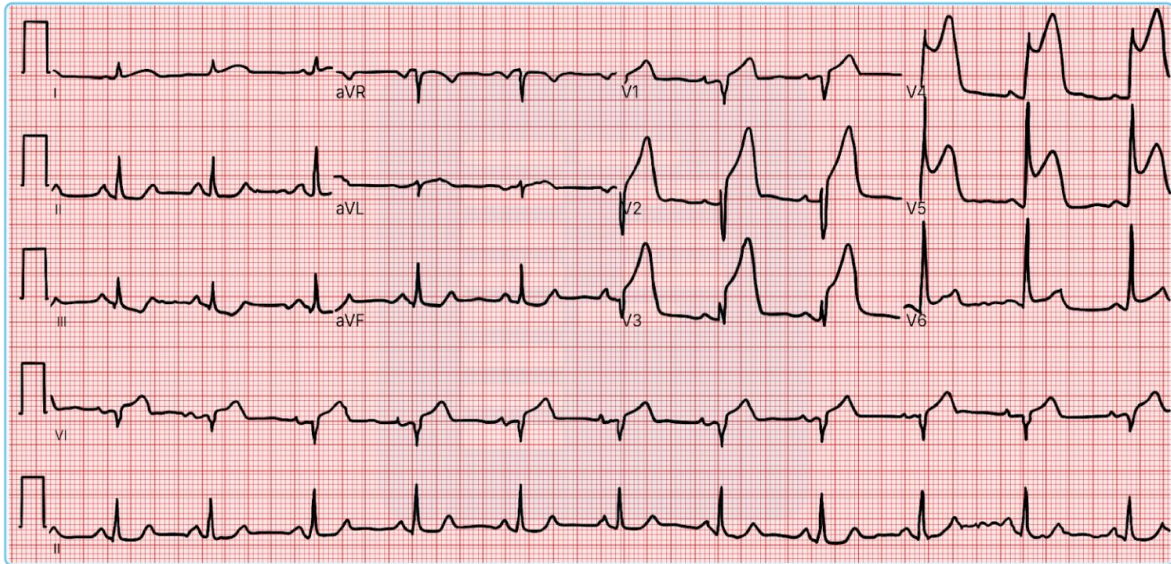
**C**: Cardiogenic shock

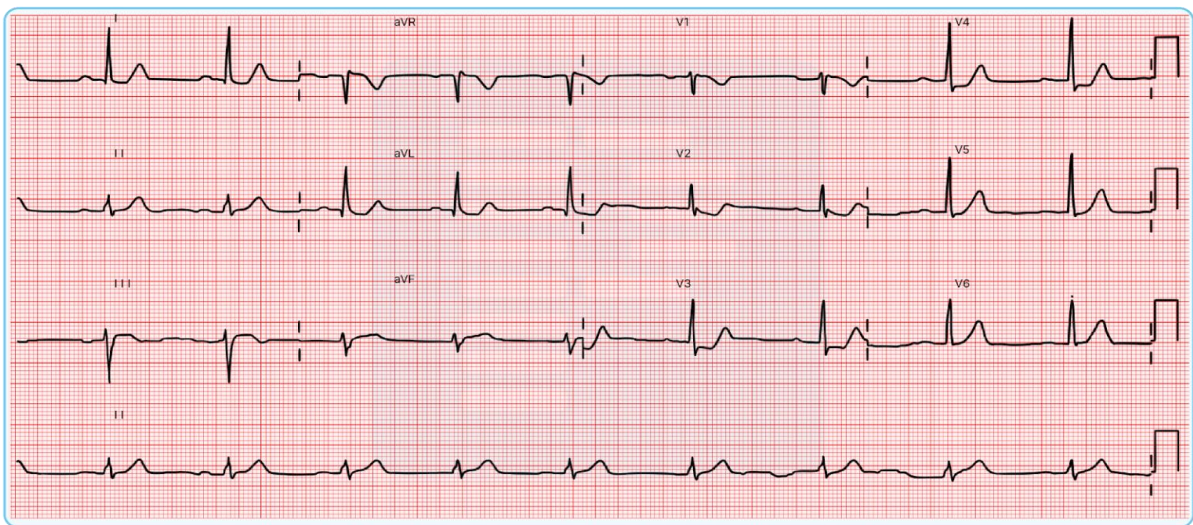
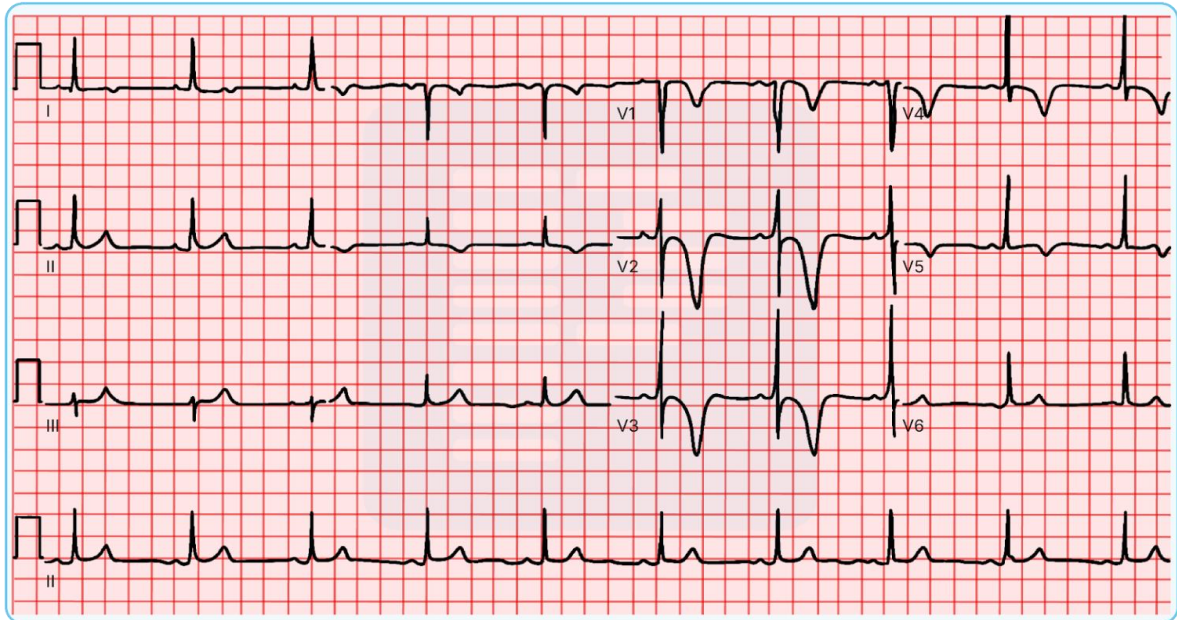
**A**: Arrhythmias

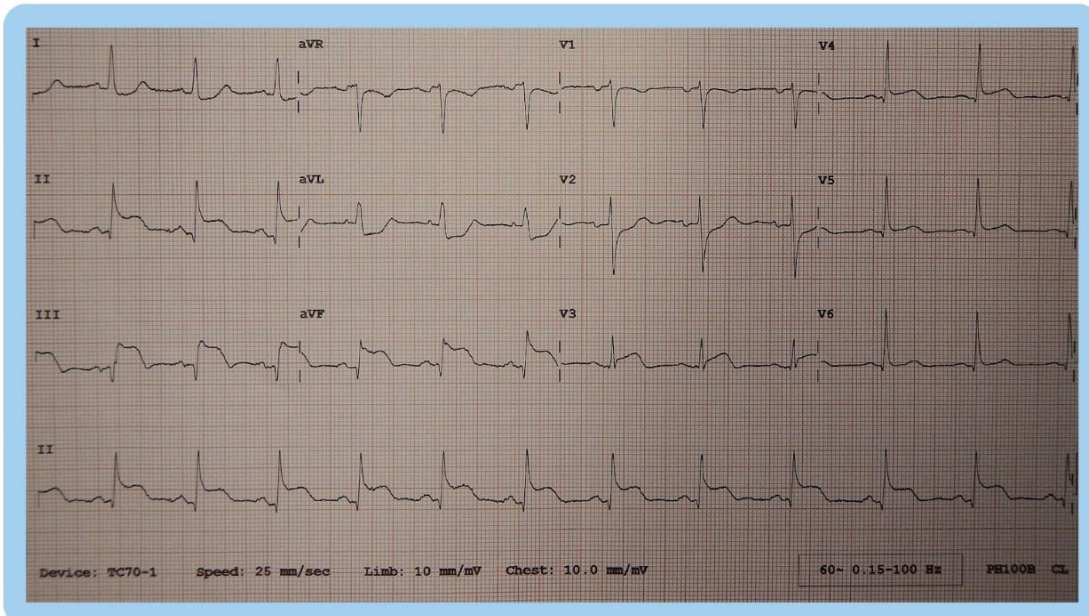
**L**: LV aneurysm

**M**: Mitral regurgitation (papillary muscle dysfunction /rupture)

**D**: Dressler









## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes

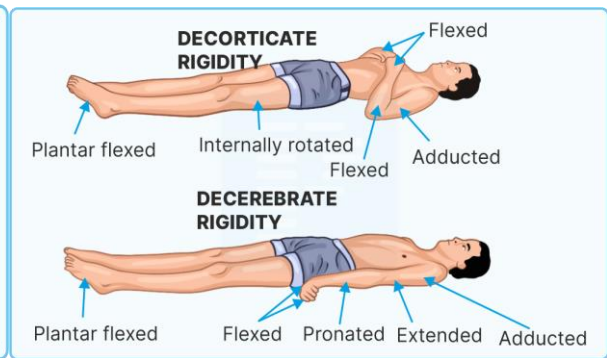
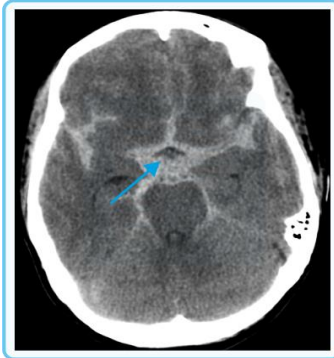


## Additional Notes



## Additional Notes

# Neurology



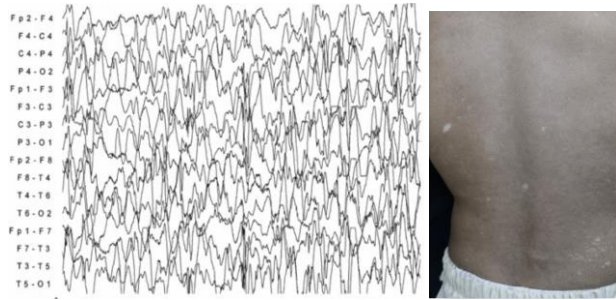
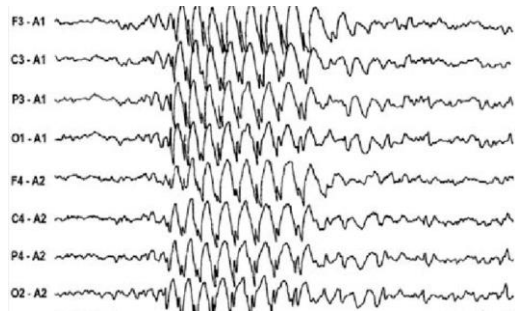
## Seizures and epilepsy

MCC of acquired epilepsy in India is

Normal EEG finding with eyes open:

Normal EEG findings with eyes closed:

EEG finding of < 3 Hz spike and slow wave pattern and multiple seizures types is seen in



Ethosuximide/ Valproate/ Lamotrigine

ACTH / Vigabatrin if associated with TS

<p><b>Drug of choice for Seizures in patient &gt;5 minutes in a patient with GTCS</b></p>	<ol style="list-style-type: none"><li>1. iv Lorazepam and repeat _____ if necessary</li><li>2. Start Phenytoin or Valproate or Levetiracetam</li></ol> <p><b>If failure to respond to BZD 2 doses and 1<sup>st</sup> line AED then diagnose as Refractory SE</b></p> <p><i>T1 = time to treat and T2= risk of neuronal injury</i></p>
<p><b>Focal seizures</b></p>	<p>Starts with Aura</p> <p>Jacksonian march or _____epilepsy with Todd paralysis</p> <p>Complex hallucinations and automatisms.</p> <p>MRI shows hippocampal sclerosis. Diagnosis:</p>

<b>GTCS</b>	<p>Ictal cry</p> <p>Usually treatment is given for 2 years and then tapering is done</p> <p>Lamotrigine can be used in both GTCS and focal seizures</p>
<b>Absence seizures</b>	<p>Sudden brief loss of consciousness without loss of postural control</p> <p>Vacant staring spells</p> <p>EEG finding of 3/sec spike and slow wave</p> <p>Carbamazepine and phenytoin worsen absence seizures</p>

- MC type of epilepsy in children:
- MC type of seizures in children:
- MC type of seizures in neonates:
- Leading cause of subtle seizures: H.I.E

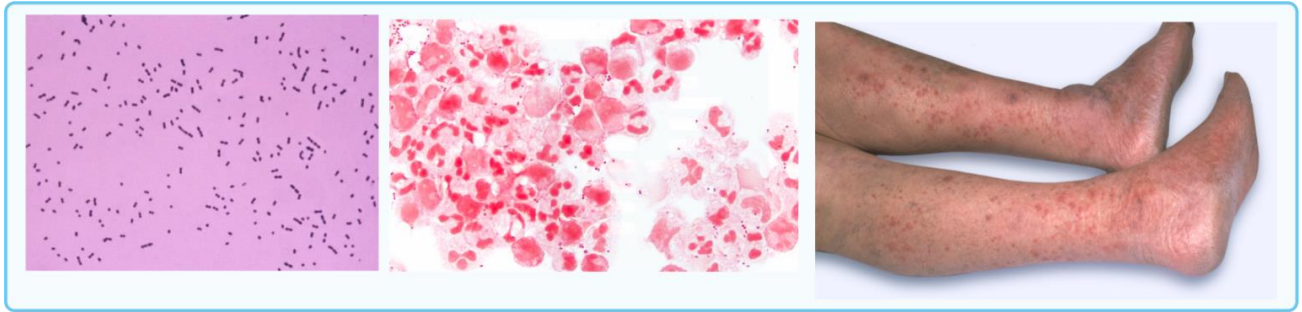
**Febrile seizures**

Best drug for intermittent prophylaxis:

Best drug for continuous prophylaxis: oral valproate. Don't use phenytoin

Best drug for management of acute episode at home:

## Pyogenic Meningitis



MCC in neonate with bulging AF and vacant stare

Leading cause of pyogenic meningitis in adults/ > 3 months to < 55 years

Leading cause of pyogenic meningitis in adults with purpuric rash and distributive shock

Perform guarded LP and then start Empirical antibiotic treatment within \_\_\_\_\_ minutes with \_\_\_\_\_

Add coverage for \_\_\_\_\_ in alcoholics/ heart transplant

Mass Chemoprophylaxis for N.meningitidis with

Recurrent meningitis in adults with meningococcus is due to C5-C9 deficiency

### Whether neuroimaging or LP first in case of meningitis

**Always do neuroimaging first if any one present**

- Papilledema
- Immunocompromised state
- New-onset seizures
- Focal neurological deficit
- Altered sensorium / low GCS
- Neoplasm / known CNS lesion

## Viral encephalitis

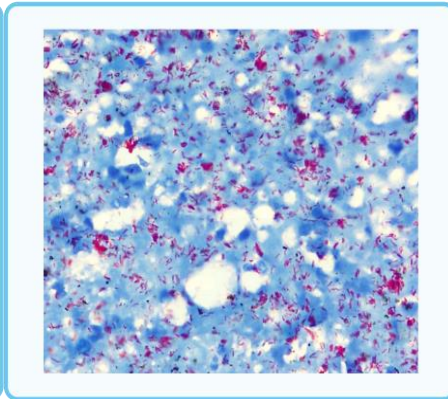
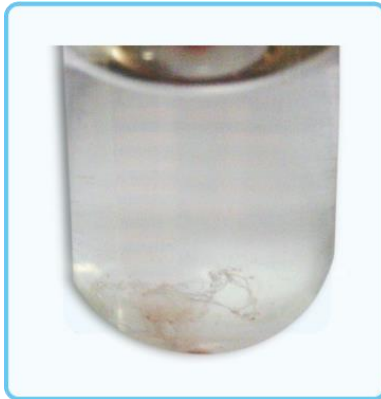
<p><b>Overall MCC</b></p>	<ul style="list-style-type: none"> <li>- Shows predilection for _____ lobe</li> <li>- CSF shows RBC in CSF</li> <li>- EEG Shows PLED</li> <li>- Empirical Rx started with</li> </ul>
<p><b>Leading cause in India</b></p>	
<p><b>Kerala outbreak</b></p>	<p>The diagram illustrates the Kerala outbreak cycle. It features a central bat reservoir at the top, with arrows pointing to 'Blood' and 'Urine' droplets. Below this, a pig is shown on the left and a human on the right. A circular arrow labeled 'Outbreak Pig &gt; Pig' surrounds the pig. A circular arrow labeled 'Outbreak Human &gt; Human' surrounds the human. A double-headed arrow labeled 'Contaminated fruit' connects the pig and the human. A red apple is shown near the pig, and a yellow container is near the human. The text 'Human: 40-75% Mortality' is positioned below the human figure.</p>



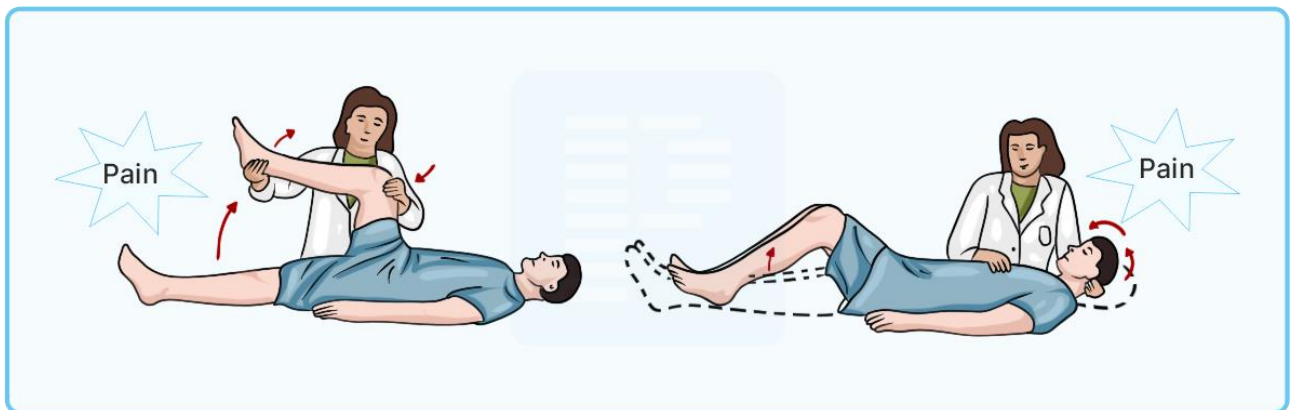
## Findings of CSF

Remarks	Cells	Sugar	Protein	Opening pressure	Color
Normal					
ABM					Turbid / Cloudy
TBM					Clear/ Straw
Viral meningitis					Clear

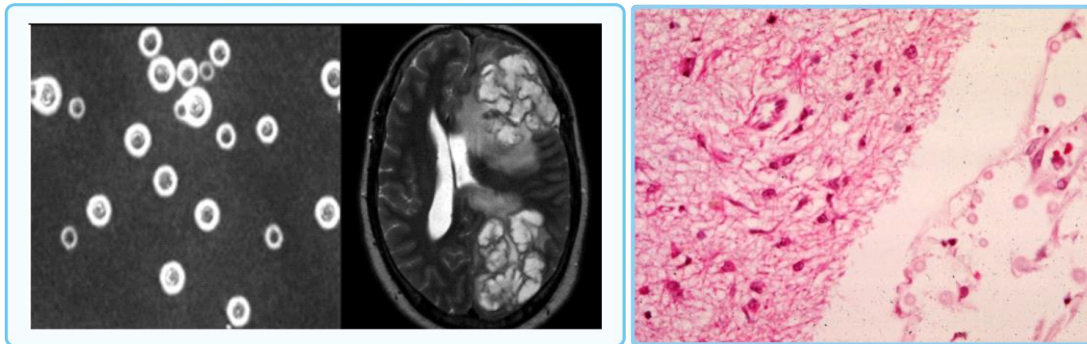
Q. 7-year-old child has been sick for 4 weeks with persistent fever, irritability, vomiting, and recent onset of headache and neck stiffness. For past one day mother says child is in altered sensorium and not recognising the mother. Lumbar puncture reveals: opening pressure raised, protein 180 mg/dL, glucose 35 mg/dL (blood glucose 100 mg/dL), and lymphocytic predominance (120 cells/mm<sup>3</sup>). What is the most likely diagnosis?



- a. Viral meningitis
- b. Pyogenic meningitis
- c. Tuberculous meningitis
- d. Cryptococcal meningitis



Q. HIV positive truck driver comes with fever and headache for the past 3 days. He also has photophobia and cannot drive. On examination nuchal rigidity is noted. Fundus shows papilledema. CSF preparation and MRI head is shown. Diagnosis is?



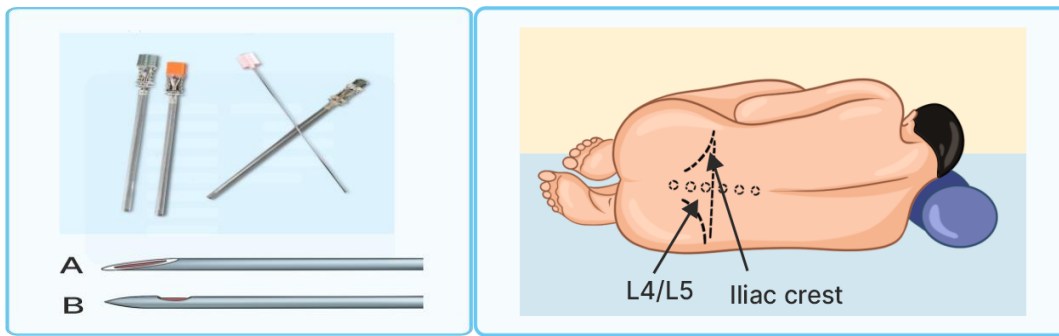
### **Mode of entry of fungus into patient**

#### **Work up**

1. MRI head
  2. Guarded Lumbar puncture and CSF cytology
  3. Urine or blood lateral flow assay for antigenic detection for cryptococcus
- Treatment= Liposomal Amphotericin B +

## Lumbar puncture

Preferred site

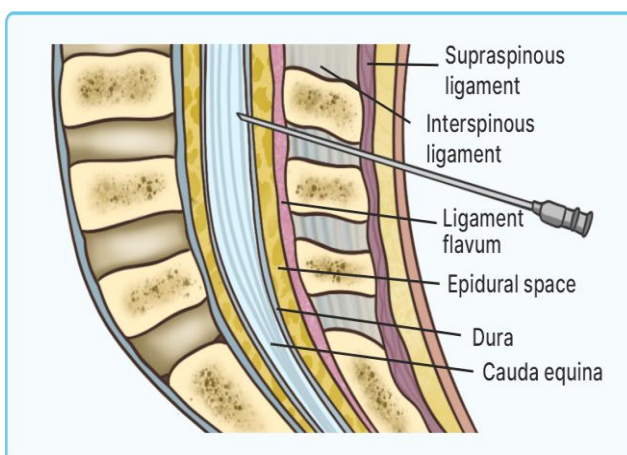


## Contraindications to LP

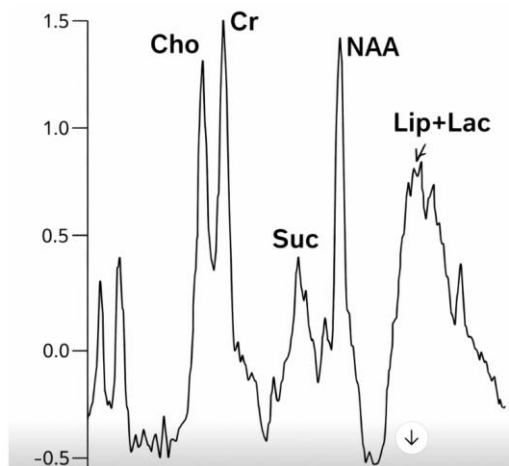
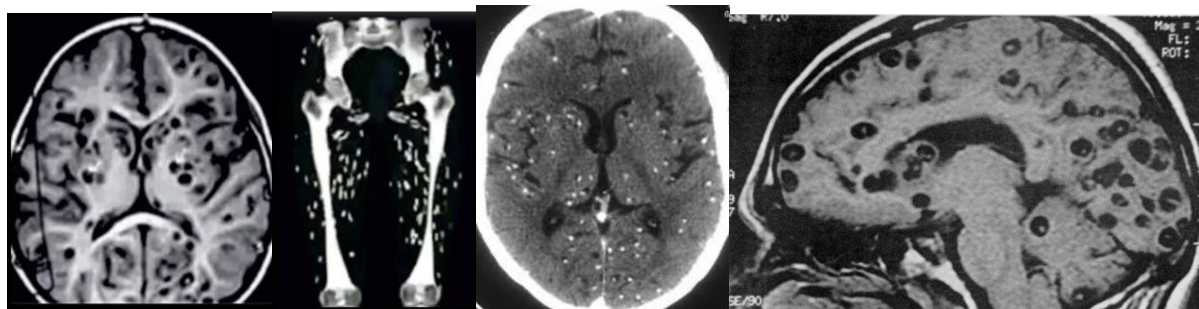
1. Raised ICP
2. Local site infection
3. Kyphoscoliosis

- Highest point of iliac crest joined to form \_\_\_\_\_ that cuts through L4 spinous process

- Most common complication of LP is headache. Best drug for management of PDPH \_\_\_\_\_



**Neurocysticercosis (caused by ingestion of proglottids containing eggs of tenia solium)**



Rx: **Calcified lesions:** Only Valproate X \_\_\_\_\_ years

RX: **Hypointense lesions with/without scolex**

- 1.
2. Albendazole
3. Valproate

**DIFFERENCE BETWEEN NEUROCYSTICERCOSIS AND CNS TUBERCULOMAS LESIONS**

NEUROCYSTICERCOSIS	TUBERCULOMA
<ol style="list-style-type: none"> <li>1. Lesions are &lt;20 mm and may be single or multiple.</li> <li>2. Meningitis feature is not there.</li> <li>3. Present at grey-white matter junction.</li> <li>4. Other involvements like eyes, muscles or subcutaneous tissues.</li> <li>5. T2W shows hyper intensity with hypointense scolex in it. No midline shift and ring enhancement is there depending upon the staging.</li> <li>6. MR spectroscopy shows multiple amino acid peaks.</li> </ol>	<ol style="list-style-type: none"> <li>1. Often multiple and &gt;20 mm because of conglomeration.</li> <li>2. Meningitis is usually associated.</li> <li>3. More common in posterior fossa.</li> <li>4. Spread is mostly secondary to infection somewhere else.</li> <li>5. Hypointensity seen in T2W and midline shift may be present.</li> <li>6. MR Spectroscopy shows lipid peak.</li> </ol>





- DOC of choice
- 1st step in management
- MC location of Berry aneurysm
- MC location of Berry aneurysm prone to rupture
- MC cranial nerve involved in ruptured/ un-ruptured berry aneurysm

**Leading cause of death in SAH**

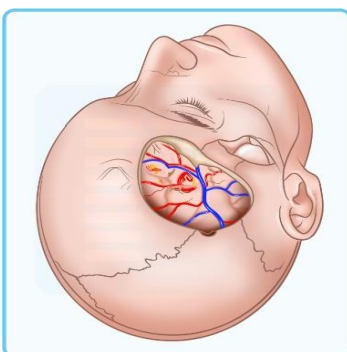
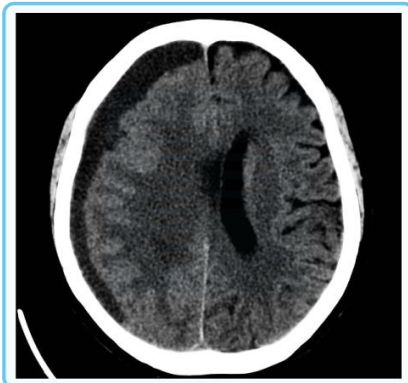
1. Delayed cerebral ischemia
2. Hydrocephalus
3. Bleeding
4. Rebleeding
5. Electrolyte imbalance

Q. Diabetic patient falls in washroom and head slams against the wall. Lucid interval was noted. From the next day onwards, her son notices she is becoming irritable and sleeps for most part of day. He also tells that she is having severe headache and has trouble speaking and recollecting events. NCCT was done on day 3 and is shown below?



cal evacuation if they do not enlarge. Stupor or coma, hemiparesis, and unilateral pupillary enlargement are signs of larger hematomas. The bleeding that causes larger subdural hematomas is primarily venous in origin, although arterial bleeding sites are sometimes found at operation, and a few large hematomas have a purely arterial origin. In an acutely deteriorating patient, an emergency craniotomy is required. In contrast to epidural hematomas, there is significant morbidity and mortality associated with acute subdural hematomas that require surgery.

Q. Diabetic patient falls in washroom and head slams against the wall. Over the next 3 weeks she becomes forgetful and delirious. NCCT was done.

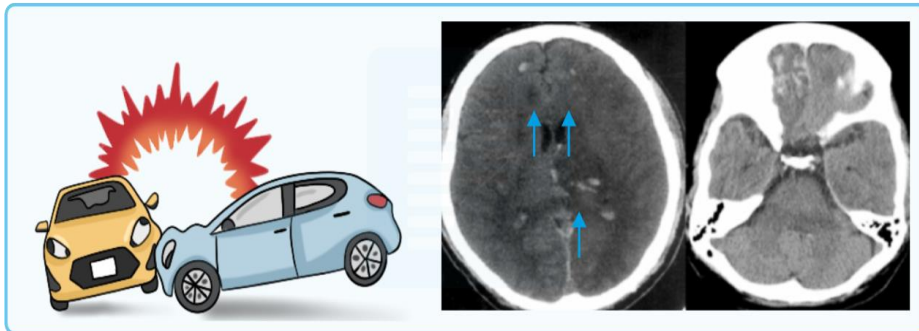


acute and chronic hematomas.

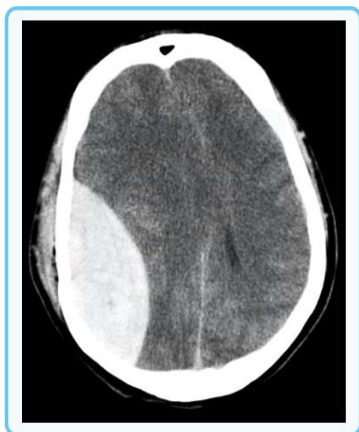
Clinical observation coupled with serial imaging is a reasonable approach to patients with few symptoms and small chronic subdural collections that do not cause mass effect. Treatment with surgical evacuation through burr holes is usually successful, if a cranial drain is used postoperatively. The fibrous membranes that grow from the dura and encapsulate the collection may require removal with a craniotomy to prevent recurrent fluid accumulation.



Q. Patient had a head on collision of car while driving under influence of alcohol. Day 3 of admission GCS score is 7/15 and relatives are arguing about lack of medical care. NCCT head is show below. Diagnosis is?



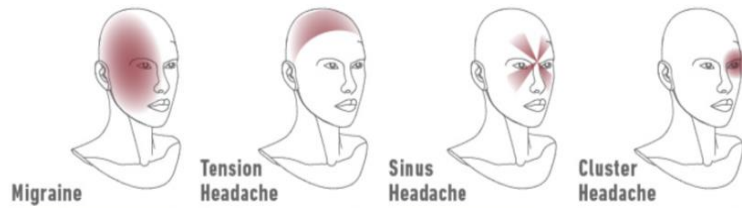
Q. Guy is struck on head with cricket ball. He becomes unconscious for few minutes. He wakes up and resumes playing and score a half century. After the match he tells the coach that he is having headache and wants to sleep in dressing room. After few hours he is non-responsive and is brought to ER.



## Headache

MC type of headache: Tension type headache

Most common cause of secondary headache : Systemic infection



## Migraine

-mediator:

-Aura: zig zag lines

**-POUND**

-Most efficacious triptan: Rizatriptan

-Triptans not given in patients with pregnancy and ASCVD. Use Lasmiditan

-Prevention of attacks with once-a-month injections

-Ona-botulinum A

-MIDAS score for disability with migraine

## Cluster headache

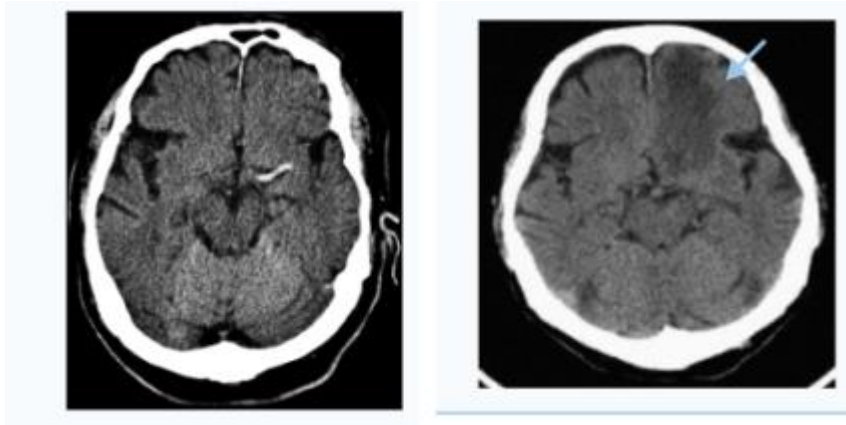
Male with retro-orbital pain and epiphora and nasal congestion (1-8 attacks per day with duration of 15-180 minutes)

RX: First line \_\_\_\_\_ and DOC

Prevention:

## Acute ischemic stroke

(ischemia occurs when brain blood flow decreases to less than 20 ml per min)  
 60-year-old man with DM and HTN develops sudden onset face deviation and arm weakness for last 3 hours. NCCT shows dense MCA sign.



Door to imaging time for stroke

Next investigation if NCCT head is normal

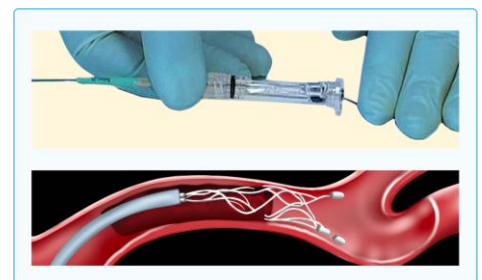
IOC for early detection of Acute ischemic stroke

Window period for management of AIS

### Priorities of acute stroke consultation

Assess Airway and BP

2. Clearly establish the time patient was last seen normal
3. Calculate NIHSS Value (Value > \_\_\_\_ is eligible for EVT or thrombolysis with IVPA or Tenecteplase 0.25 mg per kg)
4. If CT scan is normal, next best investigation is CTA



<b>Thrombolysis</b>	<b>Mechanical thrombectomy</b>
<p>Window period &lt; 4.5 hours</p> <p>2026 update</p> <p>Extended window 4.5–9 hours from last known well in imaging-selected patients (CTP / MRI mismatch)</p> <ol style="list-style-type: none"> <li>1. Wake up stroke</li> <li>2. Salvageable tissue in 4.5 to 9 hours from last know well</li> </ol>	<p>Done for LVO involving</p> <ol style="list-style-type: none"> <li>1. Proximal MCA and ICA</li> <li>2. Basilar artery occlusion</li> </ol> <p>Can be done for which candidates</p> <ol style="list-style-type: none"> <li>1. 0-6 hours: irrespective of thrombolysis eligibility</li> </ol> <p>Give IV Tenecteplase if eligible (do not delay MT)</p> <ol style="list-style-type: none"> <li>2. 6-24 hours: based on CTP report</li> </ol>

### Contraindications for thrombolysis

<b>INDICATION</b>	<b>CONTRAINDICATION</b>
<p><b>Clinical diagnosis of stroke</b></p> <p><b>Onset of symptoms to time of drug administration ≤4.5H</b></p> <p><b>CT scan showing no hemorrhage or edema of &gt;⅓ of the MCA territory</b></p> <p><b>Age ≥18 years</b></p>	<p><b>Sustained BP &gt; 185/110 mmHg despite treatment</b></p> <p><b>Bleeding diathesis</b></p> <p><b>Recent head injury or intracerebral hemorrhage</b></p> <p><b>Major surgery in preceding 14 days</b></p> <p><b>Gastrointestinal bleeding in preceding 21 days</b></p> <p><b>Recent myocardial infarction</b></p>



<b>Thrombolysis is indicated in</b>	<b>Thrombolysis is contraindicated in</b>

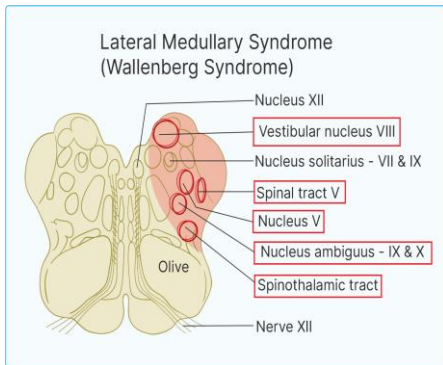
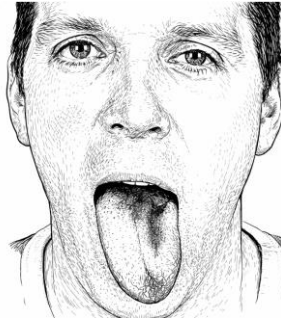
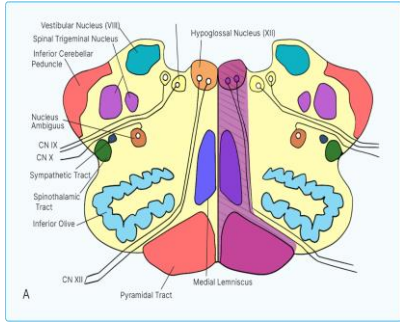
<b>Reperfusion window for STEMI</b>	
<b>Reperfusion window for AIS ( ideal)</b>	
<b>Golden period for MI</b>	
<b>Drugs approved for thrombolysis in AIS</b>	<b>Tenecteplase and alteplase</b>

## Hemorrhagic stroke

60-year-old man with Hypertension develops sudden onset face, arm and leg weakness while eating breakfast at the dining table. He is rushed to hospital where NCCT head is done. What is the site of lesion?



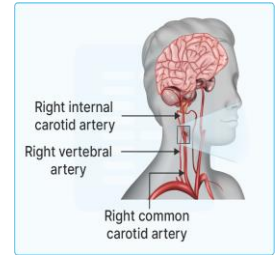
# Stroke Syndromes



- 5
- Lateral spinothalamic pathway
- 8
- 9
- 10
- Sympathetic tract

## Transient ischemic attack

Parameter			Score
A	Age	≥60 years	1
B	Blood pressure	Systolic ≥140 mm Hg or diastolic ≥90 mm Hg	1
C	Clinical	Unilateral weakness	2
		Speech problem, no weakness	1
		Any other	0
D	Duration	≥60 min	2
		10-59 min	1
		<10 min	0
D	Diabetes	Yes	1



## M. Gravis



- Myoid cells of thymus act as source of autoantigen
- 65% cases associated with
- 10% cases associated with
- Decreased synaptic transmission at myoneural junction
- Shows waxing and waning course and does not show complete remission
- Anti Ach R antibody causes
  1. Accelerated turnover of Ach R
  2. Blockage of active site of AchR
  3. Damage to post synaptic muscle membrane

1. Diplopia
2. Ptosis (asymmetrical) is most common ocular finding. Pupils are not affected
3. Diurnal variation of symptoms with weakness more in evening
4. Dysphagia and Dysarthria
6. Diaphragmatic weakness leading to dyspnea in M. Crisis
7. Muscle weakness with DTR normal

### Work up

1. Screening
2. Most specific test (confirmation)
3. IOC
4. Imaging

5. Coexistent autoimmune disease

*Common Errors*

Q. Bilateral ptosis in a patient, which investigation must be done first: MRI head

Q. Antibiotic safe in patients with MG = Linezolid

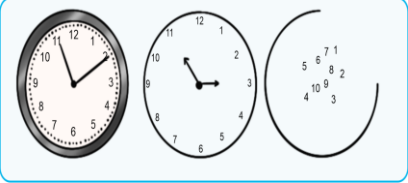
Q: MG are resistant to depolarizing NM blocking agents like Sch

Generalized M. Gravis	Ocular M. Gravis
	<p><b>Ocular muscles are more prone to depolarization block</b></p>

48-year-old woman presents with progressive proximal lower limb weakness for 4 months. Weakness is worse in the morning and improves after walking for a few minutes. Examination reveals absent deep tendon reflexes that transiently improve after sustained muscle contraction. She also reports dry mouth. What is the most likely diagnosis?

- a. Myasthenia gravis
- b. Lambert-Eaton myasthenic syndrome
- c. Polymyositis
- d. Guillain-Barré syndrome

Low frequency stimulation: Decrement  
 High frequency stimulation or post-exercise: Increment

Alzheimer disease	Parkinson disease	Huntington disease
<p><b>Ach deficiency</b></p> <p><b>Nucleus of Meynert basalis</b></p> <p><b>Hirona bodies</b></p> <p><b>Tau protein</b></p> 	<p><b>Dopamine deficiency</b></p> <p><b>Substania nigra</b></p> <p><b>Lewy bodies</b></p> <p><b>Alpha synuclein defect</b></p>	<p><b>Dopamine excess and GABA is less</b></p> <p><b>Caudate nucleus</b></p> <p><b>mutant huntingtin protein</b></p> <p><b>Rich in polyglutamine (poly-Q) repeats</b></p> <p><b>Ubiquitin-positive</b></p> <p><b>Autosomal dominant, chromosome 4 defect, trinucleotide (CAG segment) repeats and anticipation</b></p>



## Guillain Barre syndrome

Bright FAME: Brighton criteria

Flaccid paralysis bilateral

Areflexia , ascending paralysis, Albuminocytological dissociation

Monophasic course

Electrophysiological studies for diagnosis: NCV

Pathophysiology

Demyelination of peripheral nerves and nerve roots from spinal cord due to type II hypersensitivity seen after infection with C.Jejuni, Zika virus, HEV

Work up

1.Electrodiagnostic testing: NCV

2.Lumbar puncture

### Treatment

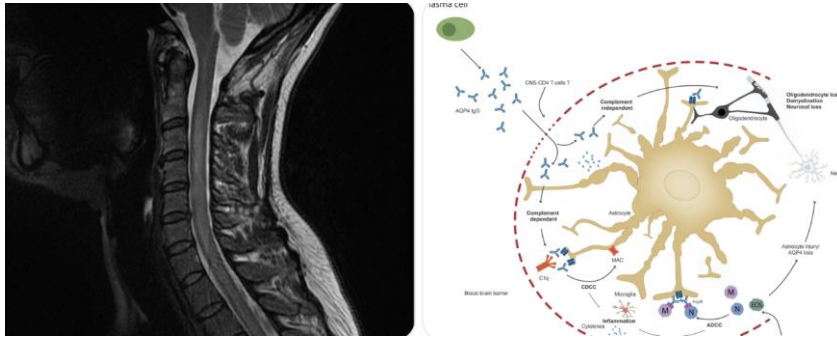
1.IVIG

2.Plasmapheresis

3.Role of steroids

<b>AIDP</b>	
<b>AMAN</b>	
<b>AMSAN</b>	
<b>MFS</b>	

## Neuromyelitis Optica / Devic disease



### Pathophysiology

- IgG against \_\_\_\_\_ on astrocytes
- Severe necrotizing demyelination (optic nerve + long spinal cord segments)

### Clinical features

- A:** Area postrema syndrome: intractable vomiting and hiccups
- B:** Brain stem syndrome
- C:** Cord involvement. Longitudinally extensive transverse myelitis
  - Lesion > 3 vertebral segments
  - Early bladder involvement
- D:** Diencephalic syndrome: unexplained weight loss and hypersomnolence
- E:** Eye involvement: optic neuritis bilateral

### Work up

1. AQP4-IgG positivity
2. MRI spinal cord showing LETMS

### Rx:

- Acute: IV steroids + Plasma exchange
- Maintenance: Rituximab



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



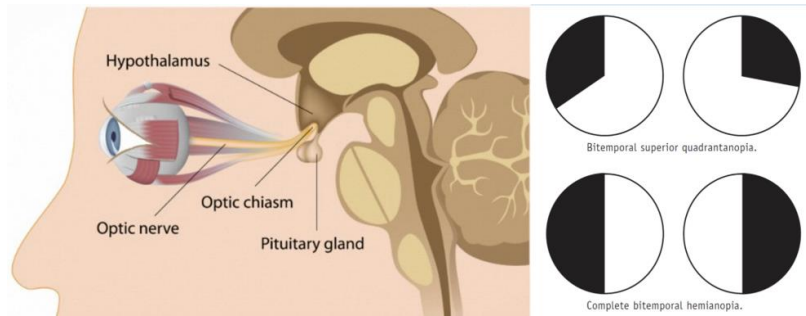
## Additional Notes

# Endocrinology

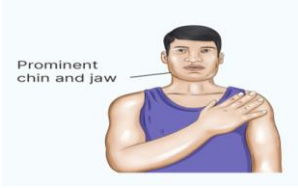
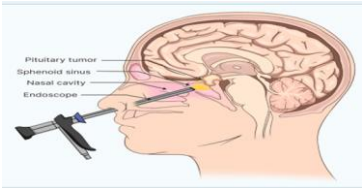
## Pituitary gland

MC tumour of pituitary is

MC *functioning* tumour of pituitary



- Pituitary adenoma grows upward (suprasellar extension)
- Compresses the optic chiasm from below
- *Inferior fibers of optic chiasm that carry superior temporal field vision are compressed first*
- Leads to *superior* temporal quadrantanopia

	<b>Prolactinoma</b>	<b>Acromegaly</b>
<b>Buzz word in clinical bytes</b>		 <p>- _____ like hands</p> <ul style="list-style-type: none"> <li>- Prognathism</li> <li>- Galactorrhoea</li> <li>- Weight gain with IGT</li> <li>- Hypertension</li> </ul>
<b>Screening</b>	Serum Prolactin levels	
<b>IOC</b>	MRI with contrast to evaluate Sellar mass	After 75 g oral glucose: GH remains >1 ng/mL
<b>1<sup>st</sup> line intervention</b>	<p><b>Patient desirous of pregnancy: Bromocriptine</b></p> <p><b>Patient completed family</b></p>	<p><b>Macroadenoma</b></p> <p><b>If high surgical risk or macroadenoma invading cavernous sinus</b></p> <p><b>Residual disease after surgery</b></p> 



## Endocrine emergencies

Emergency	First line intervention	Drug of choice
DKA D K A		
HHS Adult with T2DM is excessively sleepy and drowsy, RBS is > 600 mg/dl, ketones 2+	Aggressive fluid resuscitation with normal saline	
Severe hypoglycaemia (unconscious)	Airway + IV access	
Thyroid storm	Beta blocker	
Myxoedema coma	Secure airway External rewarming IV hydrocortisone to prevent concomitant Addison disease being unmasked and worsen the patient	



## Endocrine emergencies

Addisonian crisis	Rapid IV fluids (NS)	
Pheochromocytoma intra op HTN crisis		<b>Drug that be used both intra- op and post-operatively</b>
Hypercalcaemic crisis		IV Bisphosphonates Calcitonin nasal spray for rapid effect
Tetany		



## Endocrine emergencies

Euglycemic DKA (SGLT2)	IV fluids	IV insulin (stop SGLT2 drug)
------------------------	-----------	------------------------------

*Gradual correction of hyponatremia is done to avoid risk of ODS. Max correction of 6-8 meq rise per day*

-**SGLT2** inhibitor provide Cardio renal benefit and are used in

1. DM plus CHF
2. DM plus nephropathy

**Side effects:**

### Common error

-For management of DM plus ASCVD start

Side effects=

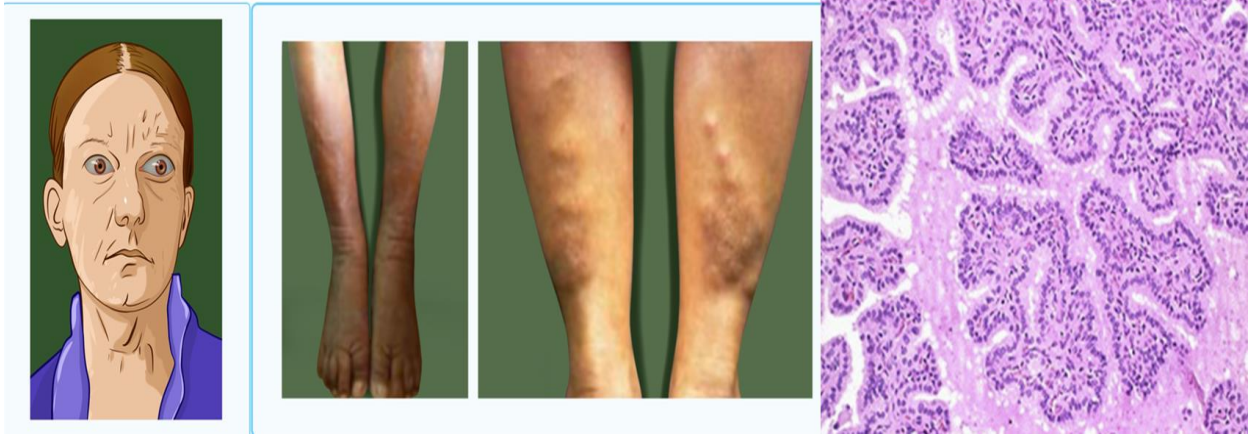
- 1.
2. GI upset
- 3.
4. Vision loss due to sudden fall of HbA1c

*Tirzepatide has the maximum glucose lowering property. Dual agonist: GIP + GLP-1 receptors*

**Diagnostics / Investigation of choice for various endocrine conditions**

<b>Primary hypothyroidism</b>			<b>Secondary hypothyroidism</b>			
TSH	T4	T3	TSH	T4	T3	

<b>Hypothyroidism and Hyperthyroidism</b>	
Suspected thyroid dysfunction (any)	
Primary hypothyroidism	
Central hypothyroidism	
Grave's disease	



**Muscle involved in Grave disease**

**Most specific feature of Grave disease**

**Antibody responsible for grave disease**

**Drug of choice for management of grave disease**

**Drug of choice for management of grave disease in pregnancy T1**

Disease of Adrenal glands	
Condition	Investigation of choice
ACTH independent Cushing syndrome	
Primary aldosteronism	
Pheochromocytoma	24-hour urinary
Adrenal insufficiency	ACTH stimulation test

## Common errors

### 1. Best for monitoring of diabetes mellitus

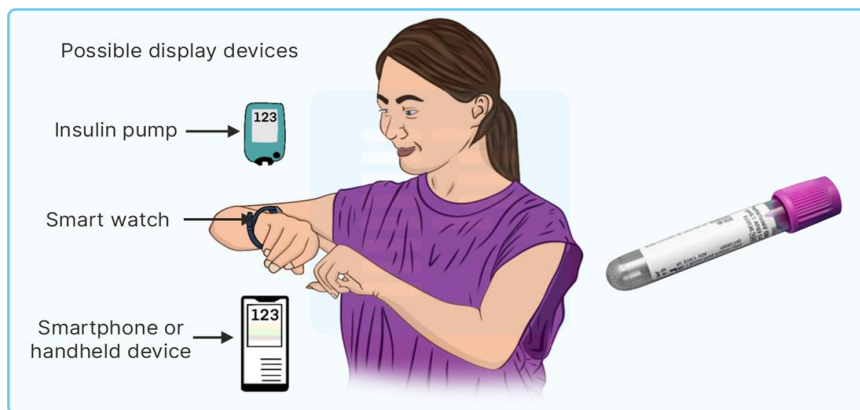
- a. CGM
- b. HbA1c
- c. Glycated albumin
- d. RBS

### 2. Not a classic symptom of Diabetes mellitus

- a. Weight loss
- b. Polyuria
- c. Polydipsia
- d. Polyphagia

### 3. Chromosome involved in pathogenesis of T1DM

- a. 2
- b. 4
- c. 6
- d. 8





### Diagnosis of Diabetes mellitus

<i>mg/dl</i>	Normal		Diabetes mellitus
<b>Fasting</b>			
<b>75 gm Glucose</b>			

Not used for Diagnosis of DM is

### Glycosylated haemoglobin

Normal	
Pre-Diabetes	
Diabetes mellitus	

**Best test for Long term control of DM:**

**Best test for short term control of DM:**

**Falsely high HbA1c:**

**Diagnosis of DM**




## Common errors in management of DKA

**D**

**K**

**A**

**1<sup>st</sup> line management in case of shock / Thready pulse/ Crashing Pulse**

**1<sup>st</sup> line management in case of evidence of dehydration (Tachycardia, low BP)**

1. Normal saline or Ringer lactate infusion
2. Check potassium values and hold insulin if potassium is less than 3.3 meq/L
3. Start regular insulin drip after 1 hour @ 0.05-0.1 unit / Kg/ hr





**Cause of death in DKA**



### Unconscious unresponsive diabetic patient

<b>pH</b>				
<b>pCO2</b>				
<b>HCO3</b>				
<b>Urine ketones</b>				
<b>RBS</b>				
<b>Any special investigation ordered</b>				

## Methods of Insulin administration

### Long-acting weekly insulin:

#### Start insulin if

1. Asymptomatic t2DM with HbA1c > 10%
2. Symptomatic t2DM with weight loss and HbA1c > 9%
- 3.

### Somogyi phenomenon (extra amount of bed time insulin)

Nocturnal

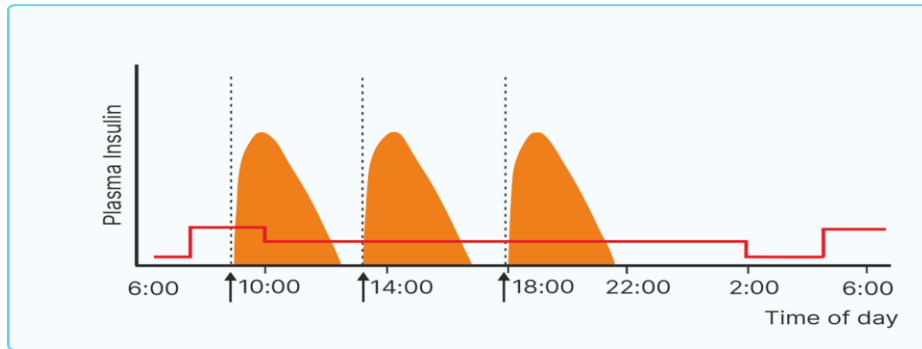
Pre breakfast

### Dawn phenomenon (GH peak between 4 am to 7 am)

Nocturnal

Pre-breakfast

## Basal Bolus regimen



## Skin involvement in DM

MC skin lesion in type 2 DM

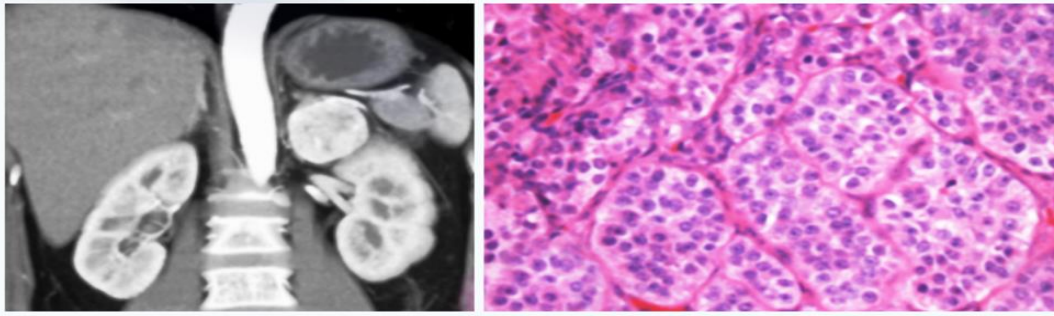
MC skin lesion in type 1 DM

MC skin lesion in glucagonoma

Causes of Acanthosis nigricans



## Pheochromocytoma



**Leading catecholamine produced by normal adrenal medulla**

**Leading catecholamine produced by pheochromocytoma**

**Leading catecholamine produced by pheochromocytoma in MEN-2 is**

**Extra adrenal pheochromocytoma is called**

**NF-1 is associated with Pheochromocytoma**

**Clinical Buzz words**

It can mimic Anxiety neurosis/ Panic attacks

**Screening test**

**IOC**




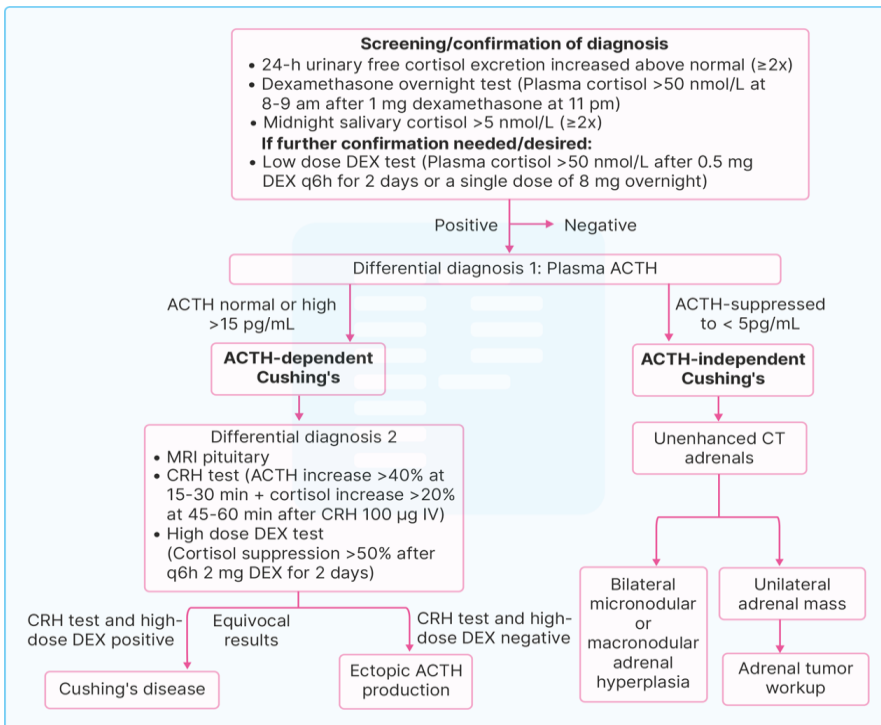
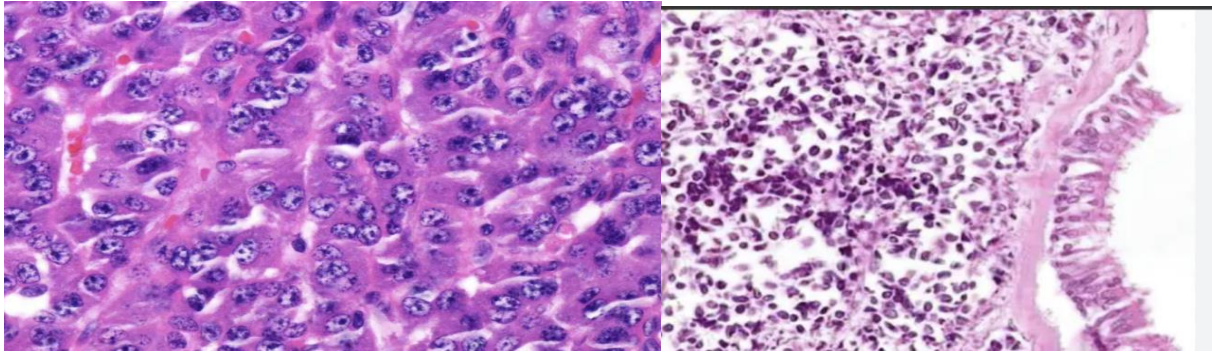
**Rx of benign pheochromocytoma**

**RX of malignant pheochromocytoma**

Use MIBG containing iodine-125 and Chemotherapy

## Cushing Syndrome

	<p><b>Clinical features</b></p> <ul style="list-style-type: none"> <li>Lemon on sticks appearance</li> <li>Dorsocervical fat pad</li> <li>Purple striae</li> <li>Thin skin with increasing bruising</li> <li>Hypokalaemic alkalosis</li> <li>Hirsutism and oligomenorrhea</li> <li>Hyperpigmentation of knuckles and IP joints</li> </ul>
<p><b>Causes</b></p> <p><b>S:</b></p> <p><b>O:</b></p> <p><b>A:</b></p> <p><b>P:</b></p>	<p><b>Screening test</b></p> <p><b>IOC</b></p> <p><b>Endogenous vs ectopic source</b></p>



## Disorders of Calcium Metabolism



DOC for tetany

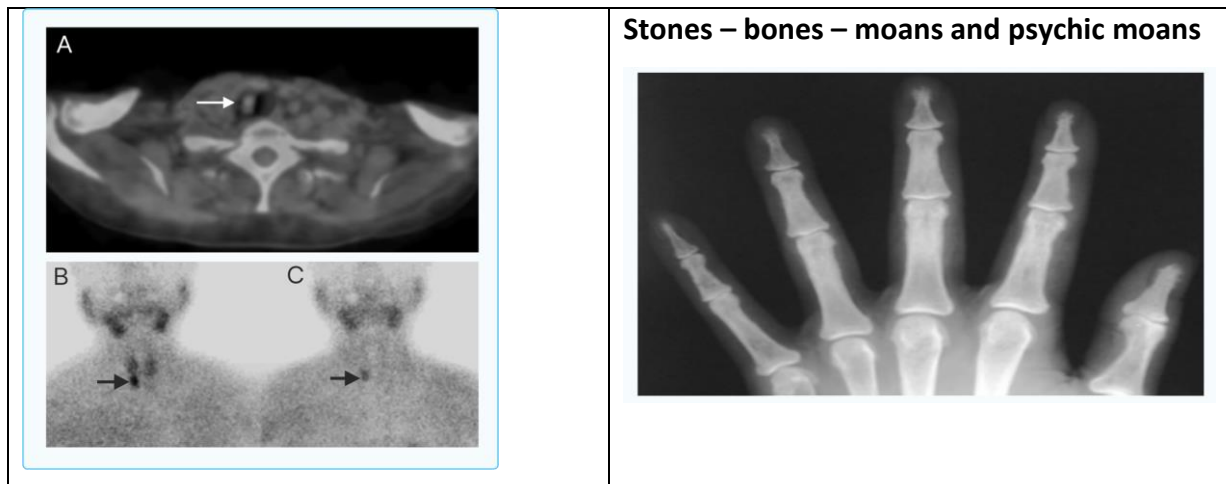
Leading cause of Hypocalcaemia

1. Inadvertent removal of parathyroid gland during total thyroidectomy
- 2.

DOC for management of surgically induced hypoparathyroidism

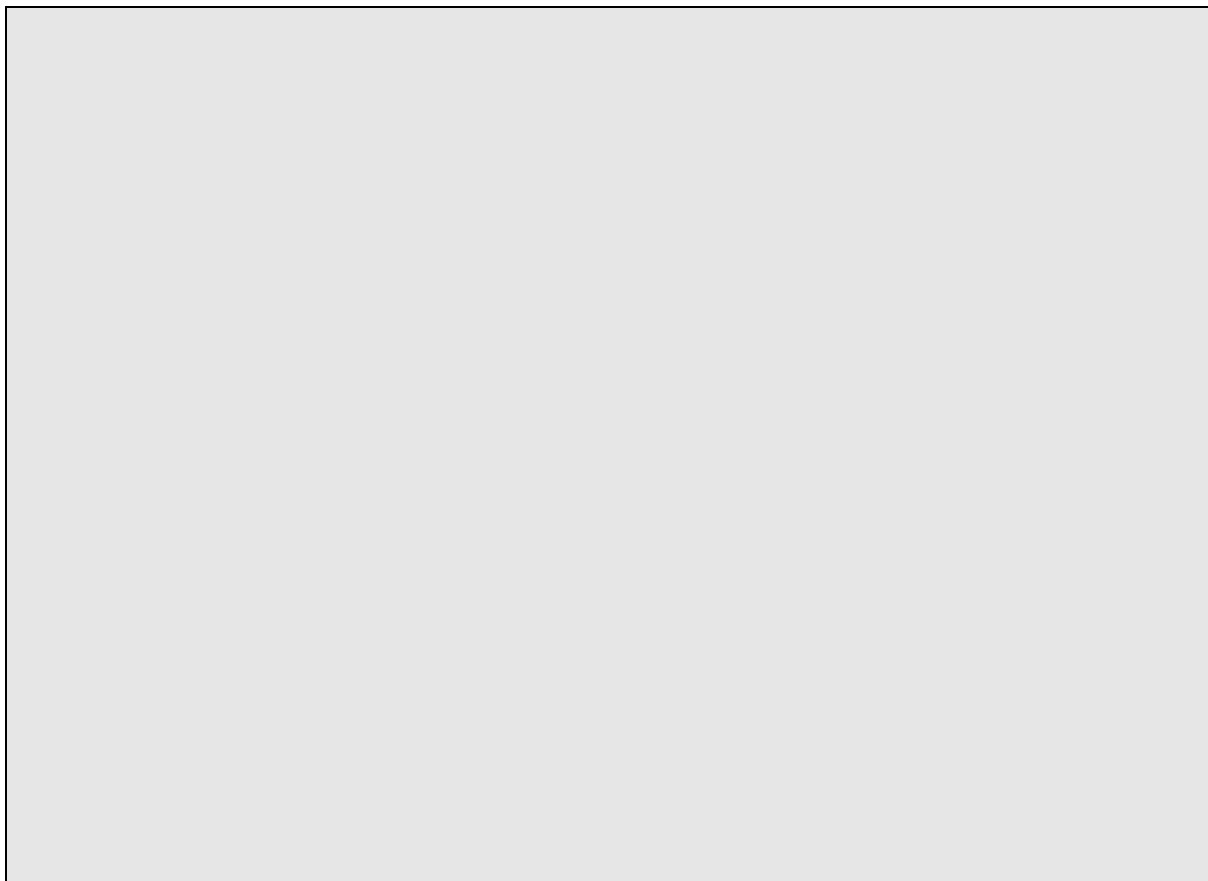
	<b>Primary Hyper PTH</b>	<b>Secondary Hyper PTH</b>	<b>Hypoparathyroidism</b>	<b>PHP</b>
Calcium				
Phosphate				
SAP				

## Primary hyperparathyroidism



Leading cause of hypercalcemia in hospitalized patients

Management of hypercalcaemic crisis





## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes

# Pulmonology



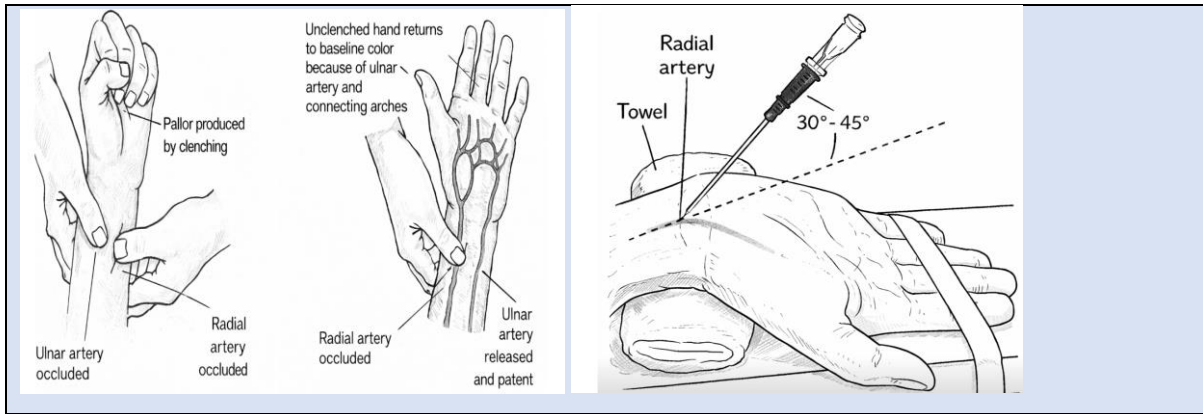
## ABG analysis Hacks

pH	pCO2	HCO3	Interpretation

### Extra mile

Aldosterone has inverse relation with potassium  
 pH has inverse relation with Potassium

**Modified Allen test is done for integrity of palmar arch before doing ABG**



**P02, pCO2 values are by default in mm Hg**

**How to interpret in 30 seconds**

<b>pH</b>	<b>7.2</b>	<b>7.22</b>	<b>7.36</b>
<b>pCO2</b>	<b>60</b>	<b>58</b>	<b>50</b>
<b>HCO3</b>	<b>26</b>	<b>28</b>	<b>32</b>



**Scenario 1:**

Parameters	Values	Interpretation
pH	7.25	
PCO2	25	
HCO3	10	

Expected PCO2= HCO3 plus 15 = 10 + 15 = 25 mm Hg

**Optional for FMGE**

**Scenario 1.1**

Parameters	Values	Interpretation
pH	7.25	
PCO2	30	
HCO3	10	

*Hypoventilation causing decreased CO2 washout*

Expected PCO2= HCO3 plus 15 = 10 + 15 = 25 mm Hg and actual PCO2=



## Scenario 1.2

Parameters	Values	Interpretation
pH	7.1	
PCO2	16	
HCO3	6	
pO2	90	

## Scenario 2: Acute exacerbation of chronic bronchitis

Parameters	Values	Interpretation
pH	7.2	
PCO2	60	
HCO3	28	
pO2	90	

Management =





### Scenario 3: COVID Moderate/ Severe case

Parameters	Values	Interpretation
pH	7.5	
PCO2	30	
HCO3	20	

### Scenario 4: Chronic Vomiting in case of CHPS neonate

Parameters	Values	Interpretation
pH	7.5	
PCO2	45	
HCO3	30	



## Optional for FMG

### Scenario 5

#### CKD patient with multiple episodes of vomiting

Parameters	Values	Interpretation
pH	7.4	
PCO2	40	
HCO3	24	
Na Chloride	130 80	

#### Must know facts

Fluid of choice for metabolic alkalosis

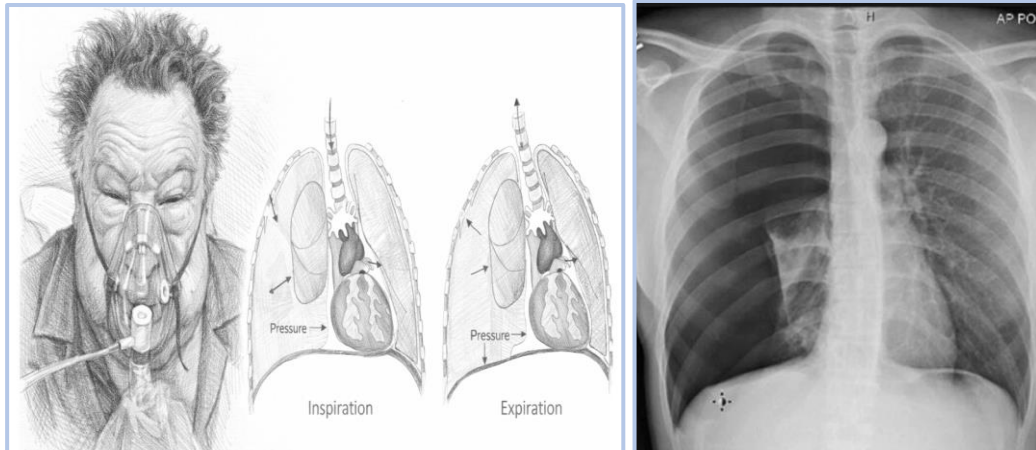
Side of Excess normal saline is

Fluid of choice for Metabolic acidosis

Best for management of Acute exacerbation of chronic bronchitis

Best for management of Respiratory alkalosis

# Pneumothorax

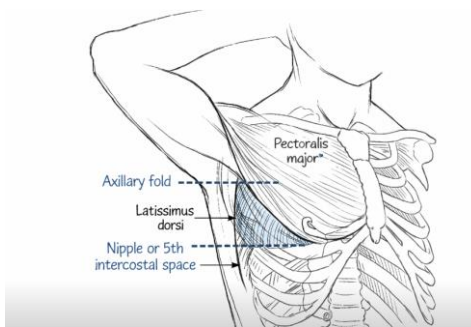
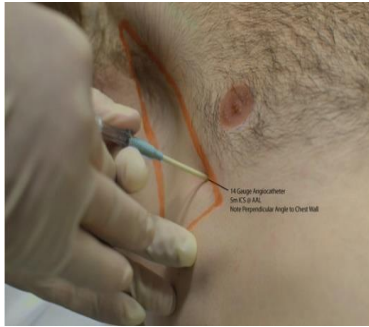


## When to say **Tension Pneumothorax**

### Key words

- Hypotension
- Mediastinal shift/ Apex beat displaced away
- Elevated JVP
  
- Absent air entry
- Absent Breath sounds
- Hyper-resonant percussion note

## First line intervention



Air collection chamber should exhibit intermittent bubbling

-Continuous bubbling in air collection chamber indicates

-Intermittent bubbling stops

## Hemothorax

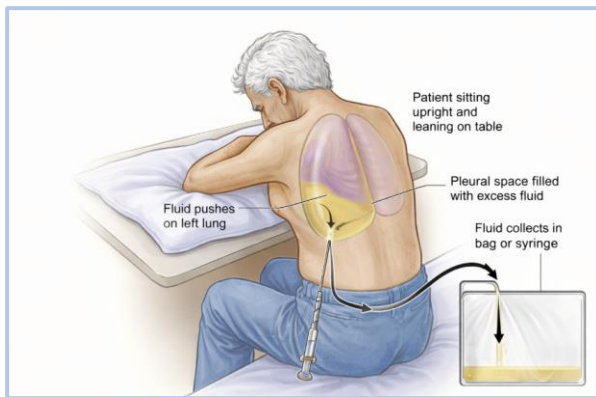
When to inform surgeon

1. Drain amount > \_\_\_\_\_ per hour for \_\_\_\_\_ hours
2. Drain amount > \_\_\_\_\_ single reading

# Pleural effusion



## Site for thoracocentesis



*Maximum amount removed per sitting*

*Site from perspective of avoiding injury to Neurovascular bundle*

Above the upper border of lower rib

**Lights criteria**

***Transudative: CHAP: CHF, Hypoalbuminemia, Ascites and peritoneal dialysis***

**PF protein**

**PF LDH**

**PF Adenosine deaminase levels elevated are seen in**

**Right sided pleural effusion is seen in**

**Bloody pleural effusion is seen in**

**Low sugar with cholesterol crystals are seen in**

**Low sugar in Pleural fluid**

# Pneumonia



Most reliable auscultatory finding of lobar pneumonia

Organism that can cause both typical and atypical pneumonia

**CAP**

**VAP**

**Leading cause in children with Cystic Fibrosis/ Mucoviscidosis ( F508 mutation, del phenylalanine, Chromosome \_\_\_)**

**< 10 years**

**>10 years**

**CURB 65 Score or CRB-65 score**

The CURB-65 criteria include five variables: confusion (C); urea  $>7$  mmol/L (U); respiratory rate  $\geq 30$ /min (R); blood pressure—systolic  $\leq 90$  mmHg or diastolic  $\leq 60$  mmHg (B); and an age of  $\geq 65$  years. Patients with a score of 0 (a 30-day mortality rate of 1.5%) can be treated as outpatients. With a score of 1 or 2, the patient should be hospitalized unless the score is entirely or in part attributable to an age of  $\geq 65$  years; in such cases, hospitalization may not be necessary. Among patients with scores of  $\geq 3$ , mortality rates are 22% overall; these patients may require ICU admission. The PSI has greater efficacy than CURB-65 but is more difficult to calculate.



**RX:**

***Outpatients***

***Inpatient / ICU***

**IV**

Add coverage for S. Aureus

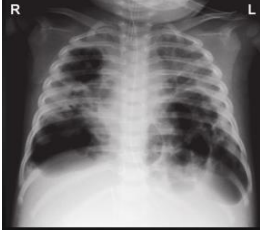
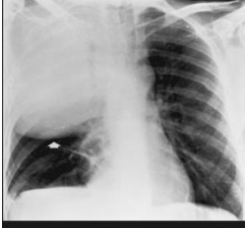

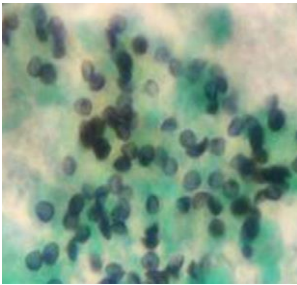

Add coverage for Pseudomonas Aeruginosa



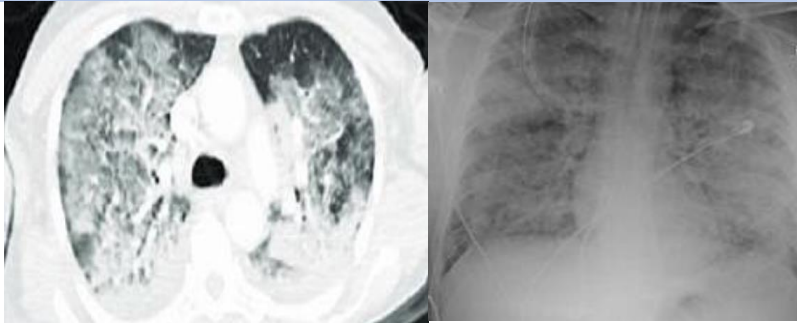
	Pneumonia	Pleural effusion	Pneumothorax
<b>Trachea position</b>			
<b>Percussion - note</b>			<i>Hyper-resonant</i>
<b>Air entry</b> <b>Breath sounds</b>			

- MCC of pneumonia in AIDS patient is Pneumococcus
- MCC of Tension Pneumothorax is Baro trauma > penetrating trauma
- Best site for insertion of wide bore needle in
  - Tension pneumothorax in adults=
  - Tension pneumothorax in children=
- Legionella pneumophila is associated with SIADH
- Legionella can present as both typical and atypical pneumonia
- Atypical pneumonia with evidence of hemolysis, Low Hb and black urine is mycoplasma pneumoniae

**Organism specific pointers in pneumonia**

<p>Legionella pneumonia</p>	<p>Best test: urine legionella antigen</p> <p>Selective culture media</p> <p>DOC</p>
<p>S. Aureus Nosocomial pneumonia</p>	
<p>Klebsiella Pneumoniae Alcoholic with Red currant jelly sputum</p>	
<p>Anaerobic lung abscess</p>	
<p>P. Jiroveci</p>	 

## ARDS



- MCC
- MC direct cause
- MC indirect cause

### Diagnostic criteria

1.  $paO_2 / Fio_2$  ratio =
2. LA pressure is normal / Absence of LA hypertension
3. CXR shows Bilateral infiltrates
4. < 7 days of lung injury

### RX:

Low volume ventilation = Low tidal volume and high PEEP

## COPD

### Modified medical research council grading of dyspnea

#### Grade

I	
II	
III	
IV	

#### GOLD grades of COPD

I	Mild	FEV1/ FVC < 0.7
II	Moderate	FEV1/ FVC < 0.7
III	Severe	FEV1/ FVC < 0.7
IV	Very severe	FEV1/ FVC < 0.7

*FEV1/ FVC < 0.7 post bronchodilator*

## GOLD Groups

A	Low symptoms	0-1 hospitalization	Any BD
B	High symptoms	0-1 hospitalization	LAMA + LABA
E	Any symptoms	>2 exacerbations or >1 hospitalization	LABA + LAMA+ ICS Blood eosinophils > 300

-Clubbing is not seen in COPD

-Barrel chest is seen and patient sits in tripod position with Hoover sign (paradoxical inward movement of chest during inspiration)

-Best to measure residual volume is body plethysmography

-Emphysema has reduced DLCO

- IV hydrocortisone is used in both COPD exacerbation and severe acute asthma. Don't use oral steroids as side effects exceed the benefit.

- NIV is treatment of choice of acute exacerbation of COPD causing Type 2 respiratory failure

- best mortality reducing intervention in COPD is LTOT using low flow oxygen. Target SpO<sub>2</sub> in COPD is 88-92%.

- NIV is contraindicated in unconscious patients with CV instability

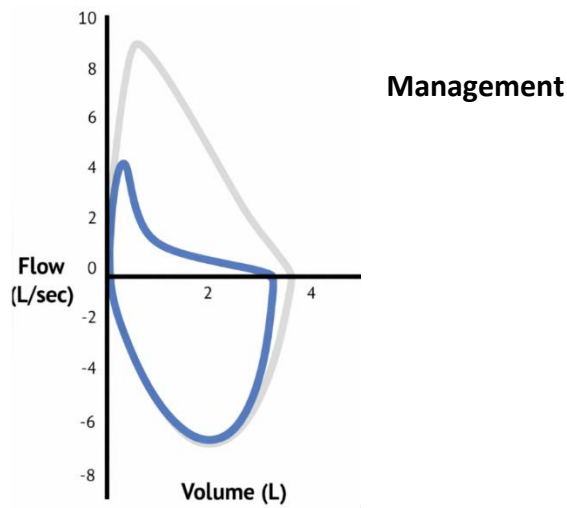
## Bronchial Asthma

- Post bronchodilator FEV1 increases by > 12% or > 200 ml air: Reversible bronchoconstriction
- DLCO is increased in asthma
- idiosyncratic asthma/ extrinsic asthma presents in adult age group and has normal IgE levels and coexists with nasal polyyps

## Extra mile

### -Fe NO measures the amount of nitric oxide in exhaled breath

1. Non-invasive measurement of nitric oxide in exhaled breath
2. **It is a non-invasive biomarker of Type-2 (eosinophilic) airway inflammation, especially in asthma.**
3. Inducible nitric oxide synthase (iNOS) upregulated by IL-4/IL-13
4. Values > 50 ppb indicate Eosinophilic inflammation likely and steroid responsiveness



<b>Step 1-2</b>	Low dose ICS-LABA as needed
<b>Step 3</b>	MART with low dose maintenance ICS-LABA
<b>Step 4</b>	MART with
<b>Step 5</b>	<p>Add on</p> <ul style="list-style-type: none"> <li>-Refer for assessment of phenotype</li> <li>-Consider trial of high dose maintenance ICS-formoterol( risk of adrenal suppression)</li> <li>-Consider Biologicals</li> </ul> <p>Mepolizumab: Block IL-5 itself → ↓ eosinophil survival</p> <p>Benralizumab: Blocks IL-5 receptor → antibody-dependent cell-mediated cytotoxicity → near-complete eosinophil depletion</p>

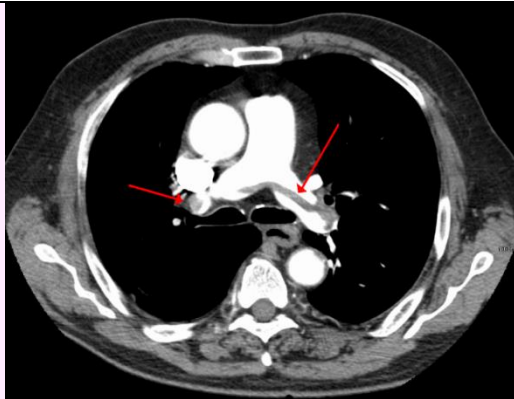
*MART = single inhaler having ICS–Formoterol inhaler for BOTH maintenance + relief*

AIR= anti-inflammatory reliever

### Case Scenario

Q. 25-year-old guy has bike accident and is hospitalized for polytrauma. On day 3 he develops sudden onset respiratory distress and spO<sub>2</sub> is 80%. O/E Pulse is 100/min, BP = 90/60 mm hg and prominent neck veins. Diagnosis is

- a. Pulmonary embolism
- b. Fat embolism syndrome



**First investigation/ Screening test**

**Non-invasive test to identify the cause**

**IOC**

**TOC of massive PE**

**TOC of PE/ Sub-massive PE**

**Parameters to be checked to identify in the question**

1. Homan sign
2. BP Value
3. Echo findings



### Common errors

Q.A 60-year-old man with colorectal cancer underwent a hemicolectomy. During surgery ETCO<sub>2</sub> suddenly dropped from 35 mm Hg to 5 mm Hg with Hypotension. Which of the following best describes this presentation?

- a. Myocardial muscle relaxation due to anaesthetic drugs
- b. Overload of saline
- c. Pulmonary embolism
- d. Mesenteric artery bleeding

Q. 25-year-old guy has bike accident and is hospitalized for polytrauma. On day 3 he develops sudden onset respiratory distress and becomes unconscious and unresponsive. O/E Pulse is 100/min, BP= 110/70 and petechiae are noted in chest wall. Diagnosis is

- a. Pulmonary embolism
- b. Fat embolism syndrome

#### Major features

Axillary or subconjunctival petechiae

Hypoxemia PaO<sub>2</sub> < 60 mmHg;  
FIO<sub>2</sub> = 0.4

Pulmonary edema

Sudden drop in Hb level > 20%

Central nervous system depression disproportionate to hypoxemia

#### Minor features

Tachycardia > 110/minute

Pyrexia > 38.5

Retinal fat or petechiae

Urinary fat globules or oligoanuria

Sudden thrombocytopenia > 50%  
High ESR > 71 mm/hour



## Additional Notes



## Additional Notes



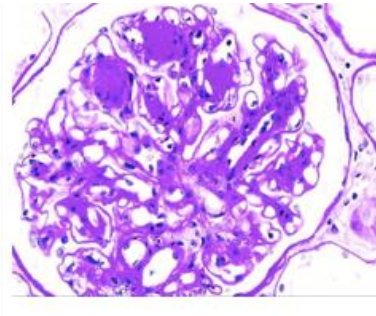
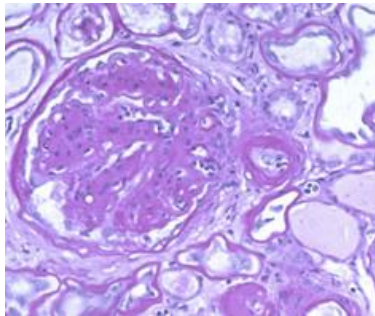
## Additional Notes



## Additional Notes

# Nephrology and electrolytes

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)			Albuminuria categories			
			Description and range			
			A1 Normal to mildly increased <30 mg/g <3 mg/mmol	A2 Moderately increased 30-299 mg/g 3-29 mg/mmol	A3 Severely increased ≥300 mg/g ≥30 mg/mmol	
GFR categories (ml/min/1.73 m <sup>2</sup> )	G1	Normal or high	≥90	Screen 1	Screen 1	Screen 1
	G2	Mildly decreased	60-89	Screen 1	Treat t 2	Treat t 2
	G3a	Mildly to moderately decreased	45-59	Treat 1	Treat t 2	Treat t 3
	G3b	Moderately to severely decreased	30-44	Treat 2	Treat t 3	Treat t 3
	G4	Severely decreased	15-29	Treat 3	Treat t 3	Treat 4+
	G5	Kidney failure	<15	Treat 4+	Treat 4+	Treat 4+



## Chronic Kidney disease

-Leading cause is \_\_\_\_\_

-CKD with normal size kidney/ slight enlargement is seen in \_\_\_\_\_

-Leading cause of death in CKD is \_\_\_\_\_

-Target BP in CKD patients should be < \_\_\_\_\_ mm Hg

-Most life-threatening complication of CKD is \_\_\_\_\_

-Staging of CKD is based on \_\_\_\_\_ and \_\_\_\_\_

-Stage at which complications appears is grade \_\_\_\_\_

-Uraemia features develop in grade \_\_\_\_\_

-Troponin I is elevated in CKD so never rely on single value in CKD patient with chest pain. Instead perform Serial troponin I

### Albuminuria categories

Description and range

CKD is classified based on:			A1	A2	A3	
• Cause (C)			Normal to mildly increased	Moderately increased	Severely increased	
• GFR (G)			<30 mg/g <3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥300 mg/g ≥30 mg/mmol	
• Albuminuria (A)						
GFR categories (mL/min/1.73 m <sup>2</sup> )	G1	Normal or high	≥90	Screen 1	Screen 1	Screen 1
	G2	Mildly decreased	60–89	Screen 1	Treat t 2	Treat 2
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat t 2	Treat 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat t 3	Treat 3
	G4	Severely decreased	15–29	Treat 3	Treat t 3	Treat 4+
	G5	Kidney failure	<15	Treat 4+	Treat 4+	Treat 4+



■ Low risk (if no other markers of kidney disease, no CKD)   
 ■ Moderately increased risk   
 ■ High risk  
■ Moderately increased risk   
 ■ High risk   
 ■ Very high risk

A1	A2	A3
Normal to Mildly Increased	Moderately Increased (Microalbuminuria)	Severely Increased (Macroalbuminuria)
< 30 mg/g (< 3 mg/mmol)	30 - 300 mg/g (3-30 mg/mmol)	> 300 mg/g (> 30 mg/mmol)
Low Renal Risk	Moderate Risk	High Renal & CV Risk
Low Renal	Moderate Risk	High Renal & CV Risk



**Mild CKD:** 60 -89

**Moderate CKD:** 30-59

**Severe CKD:** 15-29

**Renal failure / ESRD** < 15

**Q. Best to differentiate between AKI and CKD?**

- a. Creatinine clearance and Urine albumin
- b. Serum creatinine and BUN
- c. Size of kidney and BUN
- d. Urine output and Serum creatinine

<b>G1</b>	
<b>G2</b>	
<b>G3A</b>	
<b>G3B</b>	
<b>G4</b>	
<b>G5</b>	



## Medical Management of CKD: **ABCDE-PK**

**A**

**B**

**C**

**D**

**Start first with sevelamer to decrease phosphate and then start cinacalcet**

On regularly taking vitamin D and calcium, low PTH levels develop **causing Adynamic bone disease**

**E: First replete iron levels and then start Darbepoetin**

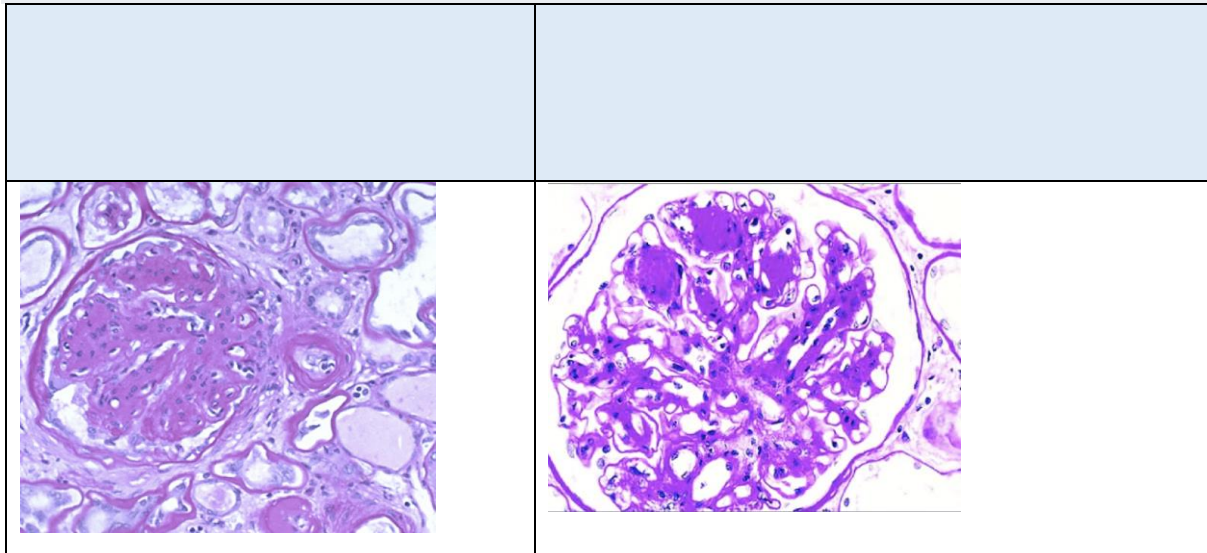
### **Causes of Anaemia in CKD**

- 1. Erythropoietin deficiency**
- 2. Iron deficiency**
- 3. Hyperparathyroidism**

**P**

**K**

## Diabetic Nephropathy

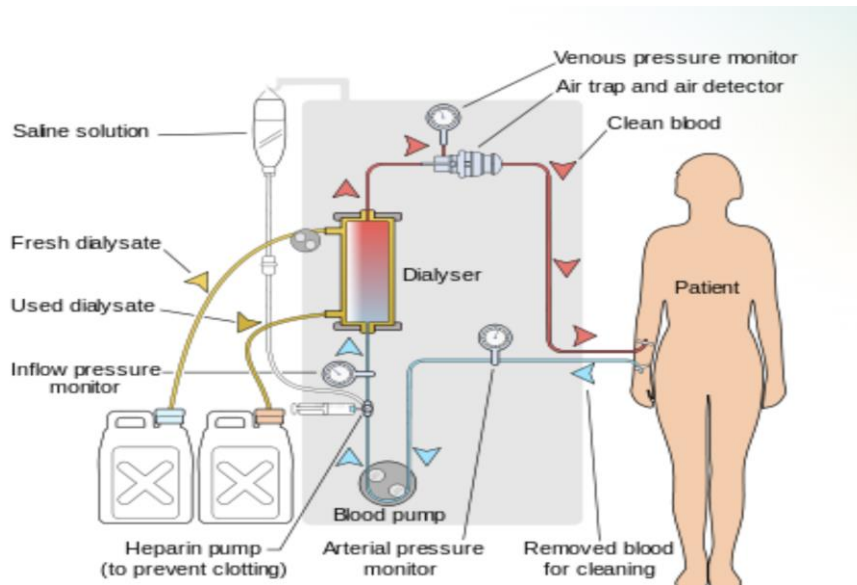


<b>Earliest feature of diabetic nephropathy</b>	
<b>Screening test</b>	
<b>First line drug</b>	
<b>Additional drugs and interventions</b>	

# Renal replacement therapy

Preferred modality: Kidney transplantation

MC used modality: Hemo-dialysis



-MC complication of HD is \_\_\_\_\_ (caused due to acetate used as buffer in dialysate due to its vasodilatory effects). Cooling the dialysate helps too in reducing this complication

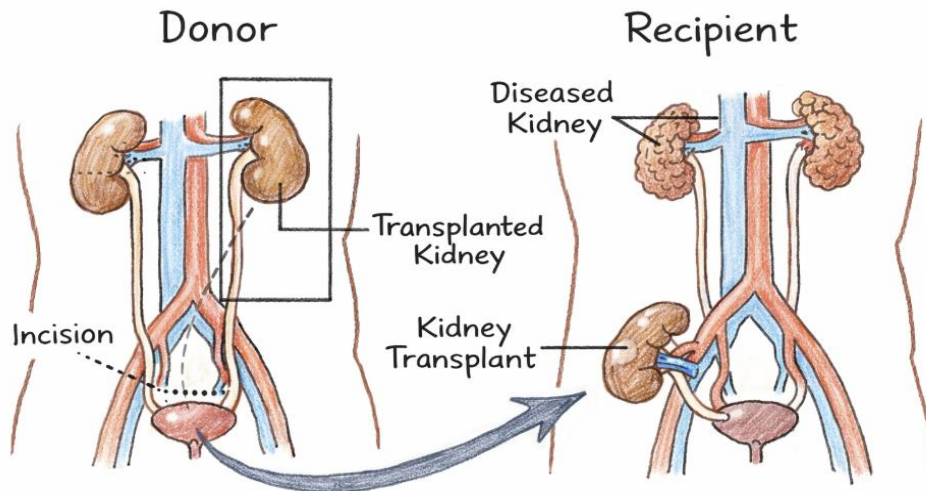
-MC complication of recurrent HD is increased \_\_\_\_\_

-AV fistula site in non-dominant hand between \_\_\_\_\_ artery and \_\_\_\_\_ vein and is called Cimino Brescia fistula

**Q. All of the following improve with dialysis except?**

- Renal osteodystrophy
- Metabolic acidosis
- Hyperkalaemia
- Pulmonary oedema

## Kidney transplant



Life expectancy enhanced by approximately 15 years

Lesion with high recurrence tendency after kidney-transplantation: \_\_\_\_\_

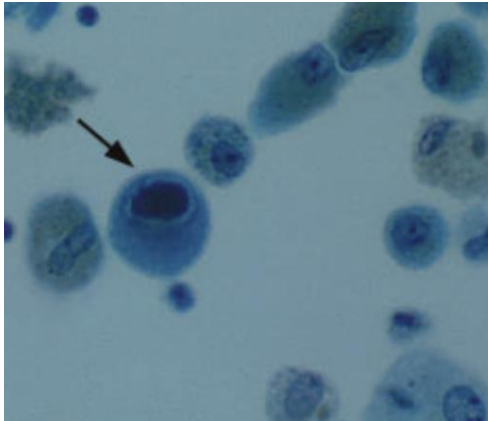
Lesion that does not recur after kidney transplantation: \_\_\_\_\_

**Q. Which of the following is absolute indication for urgent haemodialysis**

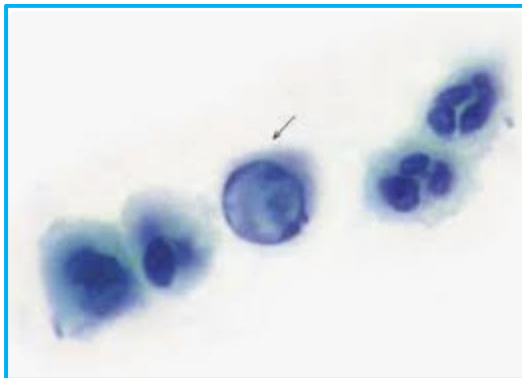
- a. Uremic pericarditis
- b. Uremic melena
- c. Uremic encephalopathy
- d. Uremic asterixis

## Opportunistic infections after kidney transplantation

MC opportunistic infection leading to graft failure after 3 months of kidney transplantation with urine m/e finding shown is



MC opportunistic infection leading to graft failure after 8 months of kidney transplantation with urine m/e finding shown is

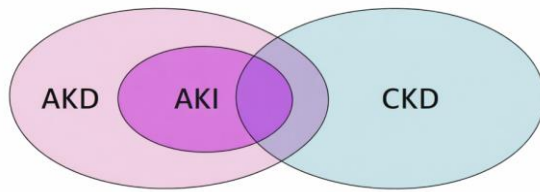


### Graft rejection

Type	Hypersensitivity
Hyperacute	
Acute	
Chronic	

## Acute kidney injury

### KDIGO/ RIFLE / AKIN criteria



#### Functional criteria for AKI

Time frame Within 7 days / 48 hours

Increase in Scr by  $\geq 50\%$  within 7 days.

OR

Increase in Scr by  $\geq 0.3$  mg/dL within 2 days.

Or Oliguria for  $\geq 4$  hours



	Serum creatinine	Urine output	Duration
Stage 1			
Stage 2			
Stage 3			

**-Leading cause of ICU admission:**

**-Anuria is defined as urine output less than 100 ml per day**

**-MC type of acute kidney injury: Pre-renal AKI**

**- Preferred fluid for choice for patients of AKI – Normal saline**



## Indications for haemodialysis

**A:** Acidosis (pH <7.1)

**E:** Electrolytes: Refractory Hyperkalaemia (K > 6.5 mEq/L)

**I:** Intoxications

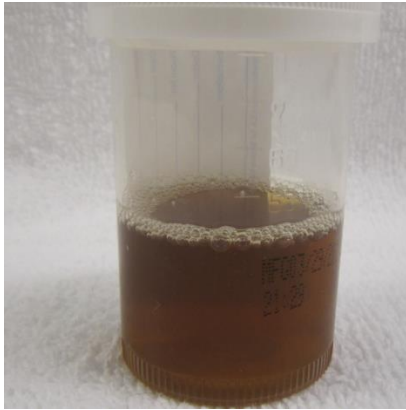
**O:** Overload with fluid refractory to diuresis

**U:** Uremic pericarditis, uremic encephalopathy

**Q. 15-year-old child develops diarrhoea with severe dehydration and oliguria. On admission Serum creatinine is 4 mg/dl with urine osmolality of 700 mosm/kg and urine sodium of 12 meq/L. what is the most likely diagnosis?**

- a. Pre renal AKI
- b. Post renal AKI
- c. Renal AKI
- d. ATN

**Q. Electrician is admitted to ER with burn and passage of red color urine. Labs shows increased CK MM levels with serum creatinine of 4 mg/dl. Which of the following will not be seen in this case**



- a. Hypocalcaemia
- b. Hypokalaemia
- c. Hypophosphatemia
- d. Hypomagnesemia

**Q. Leading cause of renal failure in multiple myeloma**

- a. Hypocalcaemia
- b. Hypercalcemia
- c. Bence jones proteinuria
- d. Urine beta 2 microglobulin



### MCC of post renal AKI

### Non oliguric Acute kidney injury is caused by

1. AIN
2. Contrast induced nephropathy
3. Aminoglycosides

### Features of tumor lysis syndrome: PUKE Calcium

### Renal failure after coronary angiography

Contrast induced nephropathy (iodinated contrast)	Athero-embolic kidney disease
Good prognosis KFT returns to normal within 7 days IV hydration N-acetylcysteine (	Livedo reticularis Rash Blue toes <b>Urine</b> _____

### Renal failure after dysentery/ Diarrhoea

Normal sized kidney PS: Schistocytes	Unilateral or Bilateral enlarged kidneys



## ECG changes in electrolyte abnormalities seen in kidney disease

Hyperkalaemia	Hypokalaemia



## Renal tubular acidosis

### Type 1

**Causes:** \_\_\_\_\_ and amphotericin B

**Mnemonic:** Alpha can't acidify, Stones Fly and potassium says bye

<b>Defect in alpha intercalated cells</b>	Inability to acidify urine and urine pH>
<b>Impaired function of H-K ATP ase</b>	
<b>Low urinary citrate and alkaline urine</b>	
<b>Can't excrete H+</b>	

### Type 2

**Causes:** Fanconi syndrome, Wilson and multiple myeloma

-Impaired bicarbonate resorption from PCT

-Serum bicarbonate: <15 meq/L

- Salt loss occurs – increased distal sodium delivery- hyperaldosteronism and hypokalemia

### Type 4

**Cause:**

**High sugar damages the JG apparatus plus tubulointerstitial fibrosis damages principal cells in collecting duct which is the site of aldosterone resistance**

ENac: Resistance to aldosterone

Hyperkalaemia with inability to acidify urine

## Electrolytes

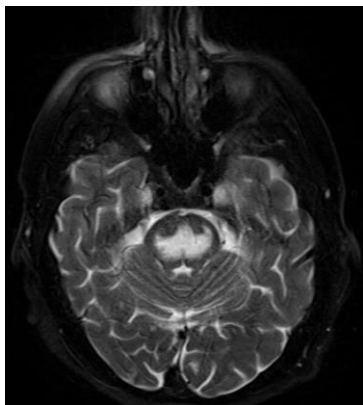
### Hyponatremia

Fast correction of chronic hyponatremia leads to stroke like feature called ODS

Symptoms usually appear **2–6 days after correction**

Mild	Moderate	Severe
Confusion	Quadriparesis	Locked-in syndrome
Dysarthria	Spastic paralysis	Conscious but cannot move limbs
Dysphagia	Pseudobulbar palsy	

MRI findings of ODS shows trident sign



**Q. 28-year-old male collapses at the finish line of a full marathon conducted in hot weather. He is confused and has a generalized tonic-clonic seizure in the emergency department. On examination: BP: 110/70 mmHg, HR: 96/min. History reveals he consumed excessive plain water during the race.**

**Labs show**

- **Serum sodium: 118 mEq/L**
- **Serum osmolality: low**
- **Urine osmolality: elevated**

**What is the most appropriate immediate management?**

- Fluid restriction and slow correction with normal saline
- 3% hypertonic saline bolus (100 mL over 10 minutes)
- 3% hypertonic saline infusion with slow correction @6 meq per 24 hours
- Demeclocycline therapy



**Q. A 62-year-old chronic smoker presents with fatigue, headache, and progressively increasing confusion for 10 days. He has lost 6 kg over the last 3 months. Examination reveals decreased breath sounds over the right upper lung field and disoriented patient.**

**Investigations:**

- **Serum sodium: 118 mEq/L**
- **Serum osmolality: Low**
- **Urine osmolality: High**
- **Urine sodium: Elevated**
- **Chest X-ray shows a right hilar mass**

**What is the most appropriate management?**

- a. 3% hypertonic saline bolus (100 mL over 10 minutes)
- b. 3% hypertonic saline slow correction not exceeding 6–8 mEq/L per 24 hours
- c. Rapid correction with normal saline infusion
- d. Demeclocycline therapy as immediate management

**Q. 55-year-old patient of lung cancer admitted for work up developed seizures. Labs shows Na= 120 meq/L, serum potassium = 4.0 meq/L, Calcium 10 mg/dl. Weight = 60 kg. Calculate the total correction to be given to this case over the next 24 hours?**

- a. 180
- b. 360
- c. 600
- d. 720



### Lab status in SIADH

<b>Serum sodium</b>	<b>Low</b>
<b>Serum osmolality</b>	<b>Low</b>
<b>Urine osmolality</b>	<b>High</b>
<b>Urine sodium</b>	<b>&gt;40 meq/L due to ANF liberation)</b>
<b>Volume status</b>	<b>Euvolemic</b>

<b>Electrolyte imbalance</b>	<b>Possible leading Cause of death</b>	<b>Intervention</b>
<b>Hyponatremia</b>		
<b>Hypernatremia</b>		
<b>Hypokalaemia</b>		
<b>Hyperkalaemia</b>		
<b>Hypocalcaemia</b>		
<b>Hypercalcemia</b>		
<b>Hypermagnesemia</b>	Asystole, bradycardia and Nm paralysis	
<b>Hypomagnesemia</b>	Ventricular arrhythmias and tetany	



## Additional Notes



## Additional Notes



## Additional Notes

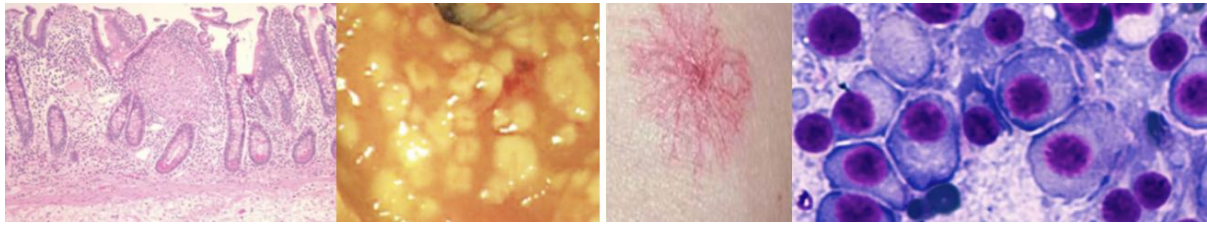


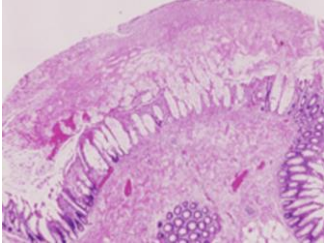
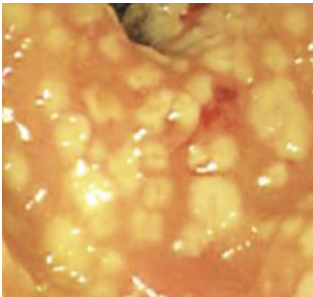
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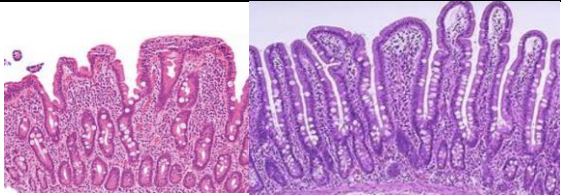


## Additional Notes

## RR LIVER GIT system

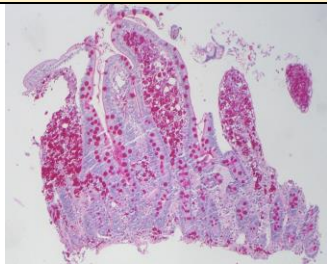


<p><b>Cause:</b> Overgrowth of _____ that produce toxins A (enterotoxin) &amp; B (cytotoxin)</p> <p><b>Triggers</b></p> <ul style="list-style-type: none"> <li>• Cephalosporins</li> <li>• Clindamycin</li> <li>• Fluoroquinolones</li> <li>• Ampicillin / Amoxicillin</li>   <li>• Prolonged _____ use</li> </ul> <p><b>Clinical features</b></p> <ol style="list-style-type: none"> <li>1. Profuse watery diarrhea</li> <li>2. Abdominal pain, fever</li> <li>3. Leukocytosis</li> <li>4. Recent antibiotic exposure (oral or IV), often in a hospitalized or elderly patient.</li> </ol> <p><b>Screening</b></p> <ol style="list-style-type: none"> <li>1. Stool _____ antigen</li> <li>2. Stool EIA for toxin A and B</li> </ol> <p><b>IOC</b> Stool</p> <p><b>Colonoscopy:</b> Yellow–white raised plaques (pseudo-membranes) with patchy distribution over erythematous mucosa in colon</p>	  <p><b>Rx:</b> First episode: Fidaxomicin</p> <p>Severe or relapsed PMC: Vancomycin</p> <p>Multiple recurrences: Faecal microbiota transplant</p>

	<p>BROW is CI</p>
<p><b>IOC- IgA anti-tTG antibody</b></p>	<p>HLA DQ2 DQ8</p>
<p>Anti endomysial antibody- <b>Highest specificity</b></p>	<p>Quinoa, Bajra, maize and rice with iron and Folic acid is given</p>
<p><i>If IgA deficient</i></p>	<p>Follow up required for</p> <ol style="list-style-type: none"><li>1. T1 DM</li><li>2. DH</li><li>3. EATL</li></ol>
<ol style="list-style-type: none"><li>1. IgG anti-tTG</li><li>2. Anti- Deamidated Gliadin Peptide</li></ol>	

IOC for malabsorption: 72-hour fecal fat estimation: > 7g fat (Sudan III stain)

### Whipple disease



#### Cause

Chronic diarrhea, steatorrhea, weight loss

Seronegative arthritis that precedes GI symptoms

Fever

Hyperpigmentation

#### IOC: Duodenal biopsy

-PAS-positive macrophages in lamina propria showing rod shaped bacilli

-PCR on blood sample

#### Rx:

Ceftriaxone


TMP SMX

#### Complications

-CNS cognitive decline

-CVS: culture negative endocarditis (Aortic > mitral)

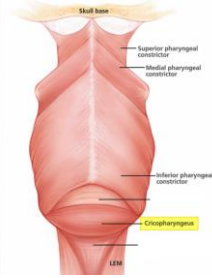
## DISEASES OF ESOPHAGUS

Achalasia cardia											
 <p><b>Ba swallow</b> Shows smooth tapering defect</p> <p>Bird beak</p> <p>Sigmoid esophagus</p>	<p><i>Pathogenesis</i></p> <p>Degeneration of inhibitory neurons (NO &amp; VIP-producing) in the myenteric (Auerbach) plexus of the esophagus</p> <p><b>Most common cause?</b></p> <p><b>UGIE shows</b></p> <p><b>IOC</b></p> <p>High resolution manometry</p> <p><i>Chicago classification</i></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="background-color: #d9e1f2;">Type 1</th> <th style="background-color: #d9e1f2;">Type 2</th> <th style="background-color: #d9e1f2;">Type 3</th> </tr> </thead> <tbody> <tr> <td>Absent peristalsis</td> <td>-Absent peristalsis</td> <td rowspan="3">Premature / spastic distal contractions</td> </tr> <tr> <td>Incomplete LES relaxation</td> <td>-Incomplete LES relaxation</td> </tr> <tr> <td></td> <td>-Uniform pressurization of entire esophagus</td> </tr> </tbody> </table> <p>Type 2 Best response to pneumatic dilatation and Type 3 worst prognosis</p> <p><i>Surgery of choice</i></p> <ol style="list-style-type: none"> <li>1. Heller's myotomy: Longitudinal division of LES muscle fibers to relieve obstruction</li> <li>2. Partial fundoplication (Dor or Toupet): Added to prevent postoperative GERD</li> </ol> <p>Effective for all Chicago types, especially Type I &amp; II</p>	Type 1	Type 2	Type 3	Absent peristalsis	-Absent peristalsis	Premature / spastic distal contractions	Incomplete LES relaxation	-Incomplete LES relaxation		-Uniform pressurization of entire esophagus
Type 1	Type 2	Type 3									
Absent peristalsis	-Absent peristalsis	Premature / spastic distal contractions									
Incomplete LES relaxation	-Incomplete LES relaxation										
	-Uniform pressurization of entire esophagus										



	<p>Other procedures</p> <ol style="list-style-type: none"><li>3. PD</li><li>4. Botulinum toxin</li><li>5. CCB</li></ol>
--	---

## Zenker diverticulum



### *Surgery*

- 1. Dohlman procedure (Endoscopic diverticulotomy)**
- 2. Z POEM**

**Carcinoma esophagus**



**Simultaneous, non-peristaltic, premature contractions** of the esophagus with **normal LES relaxation**.

**Jack hammer esophagus**

Hypercontractile esophagus characterized by very high-amplitude, prolonged peristaltic contractions on HRM

Alcoholic develops multiple episodes of vomiting and retching followed by development of chest pain. CXR is shown First differential diagnosis



1. Mallory Weiss
2. Boerhaave syndrome
3. Bleeding esophageal varices





## HEPATOLOGY

<b>MC of acute viral hepatitis (adults)</b>	
<b>Virus with highest chances of progression to chronicity</b>	
<b>MC of chronic viral hepatitis</b>	
<b>MC viral of Fulminant viral hepatitis</b>	
<b>MCC of Fulminant hepatitis in pregnancy</b>	
<b>MCC of hepatocellular carcinoma</b>	
<b>DOC for Hepatitis B</b>	
<b>DOC for hepatitis C</b>	
<b>DOC for hepatitis D</b>	
<b>MCC of OLT</b>	



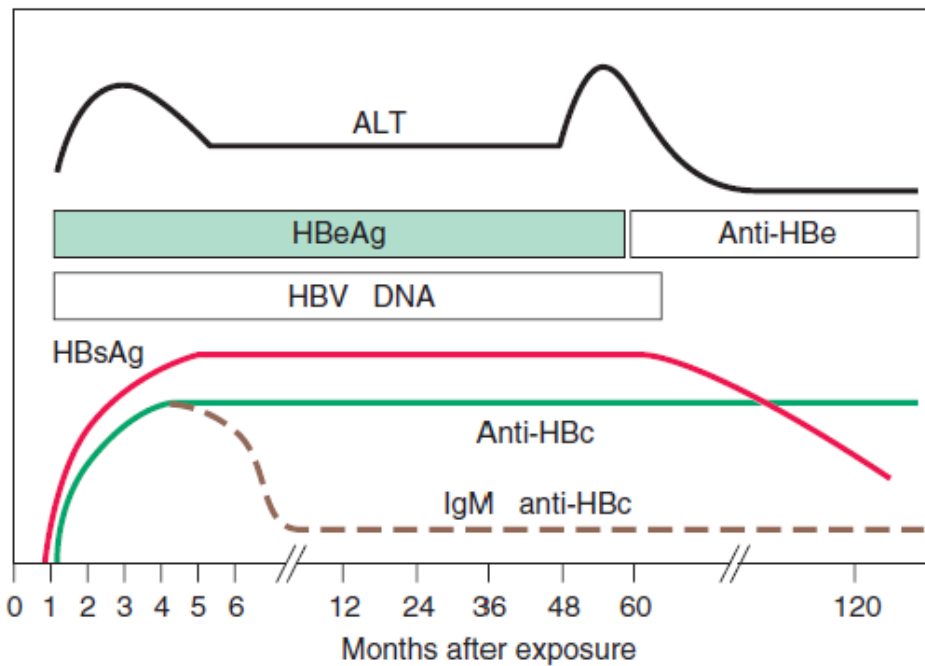
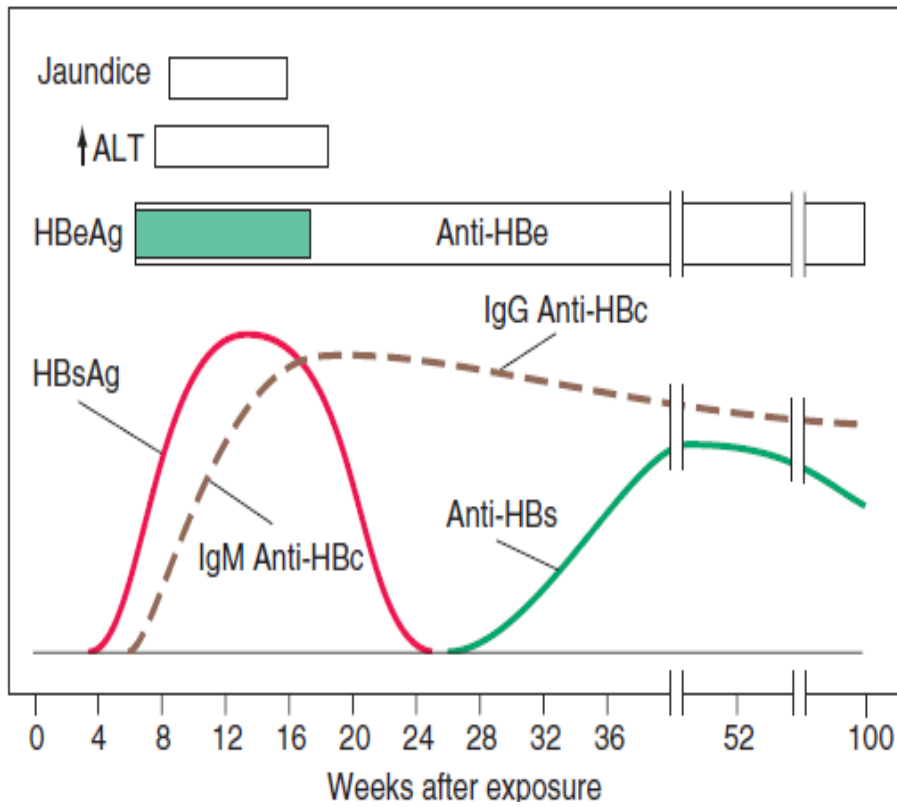
## Hepatitis B

ss/ds DNA virus

### Antigens


### Antibodies


### Serological interpretation



HBsAg	Anti HBs	Anti HBc	HBe Ag	

## CIRRHOSIS

-MCC is NAFLD ( >5% of hepatocytes showing steatosis)

-MCC of need for OLT IS NAFLD

-Non-invasive imaging for NAFLD is Fibro-scan

-Drugs approved for NAFLD

*Resmetirom*

MOA: Thyroid hormone receptor- $\beta$  (THR- $\beta$ ) agonist :  $\uparrow$  Hepatic fat metabolism and reduces liver fat

*Semaglutide (GLP-1 receptor agonist)*

*Lanifibranor: Pan-PPAR agonist in phase III trials*

**Scores for severity of cirrhosis**

<b>*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)</b>
<b>Class A = 5 to 6 points</b>
<b>Class B = 7 to 9 points</b>
<b>Class C = 10 to 15 points</b>



MELD Score	MELD 3.0

### Ascites

#### Serum albumin ascites gradient

SAAG > 1.1 g/dl		SAAG < 1.1 g/dl
Ascitic Fluid Protein < 2.5 g/dL	Ascitic Fluid Protein > 2.5 g/dL	
Alcoholic liver disease	CHF	
Portal vein thrombosis	Early Budd–Chiari syndrome	
Late Budd–Chiari syndrome	Sinusoidal obstruction syndrome (veno-occlusive disease)	



**Site of ascitic tap in refractory ascites**

1. 2cm below umbilicus
2. 2-3 cm above medial to ASIS

**Spontaneous bacterial peritonitis**

Q. A 54-year-old male with CLD and ascites presents with massive ascites. While in hospital he develops fever, abdominal pain and worsening ascites associated with shortness of breath. Which of the following is correct about clinical diagnosis of SBP?

- a. PMN cells > 50 cells/cu.mm in ascitic fluid
- b. PMN cells > 100 cells/cu.mm in ascitic fluid
- c. PMN cells > 150 cells/cu.mm in ascitic fluid
- d. PMN cells > 250 cells/cu.mm in ascitic fluid

**DOC for management of SBP = cefotaxime**

**Prevention of SBP = norfloxacin**

<b>Hepatorenal syndrome</b>	<b>Hepatopulmonary syndrome</b>
<p>Occurs due to Severe splanchnic vasodilation causing effective arterial volume causing reflex renal vasoconstriction</p> <p>Presents as</p> <ol style="list-style-type: none"> <li>1. Oliguria</li> <li>2. AKI</li> </ol>	<p>Nitric oxide–mediated intrapulmonary vascular dilatation</p> <p>Presents as:</p> <ol style="list-style-type: none"> <li>1. Platypnea</li> <li>2. Orthodeoxia</li> </ol>
<ol style="list-style-type: none"> <li>1. Terlipressin + albumin</li> <li>2. Midodrine + octreotide + Albumin</li> <li>3. Definitive is OLT</li> </ol>	

## PORTAL HYPERTENSION



**MC complication of CLD**

**Earliest and most reliable examination finding of portal hypertension**

**Portal hypertension definition is HVPG > \_\_\_\_\_ mm Hg**

**HVPG > \_\_\_\_\_ mm Hg increases risk of hematemesis due to esophageal varices**

**Source of bleeding in esophageal varices**

Portal side = left gastric vein

Systemic vein = azygos vein

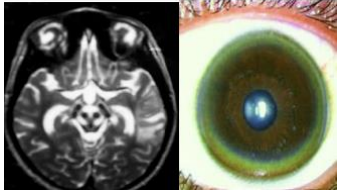
**Best management for prevention of bleeding from esophageal varices**

**DOC for prevention of bleeding from esophageal varices**

### **Management of acute variceal bleeding with BP < 90/60 mm Hg**

3. Wide bore cannula
4. Massive transfusion
5. Permissive hypotension
6. Octreotide / Somatostatin
7. Endoscopic variceal ligation

## Wilson disease



Gene

Chromosome

Inheritance

**Defect**

**C/F: Mnemonic- COPPER**

**Chronic hepatitis**

**Ocular features**

**Psychiatric features**

**PD like features**

**EPS**

**Renal tubular disorder**

**Screening**

**IOC**

**1<sup>st</sup> line drug**



## Additional Notes



## Additional Notes



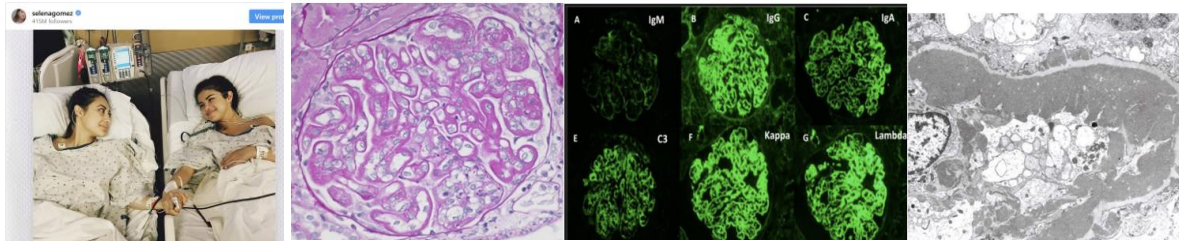
## Additional Notes



## Additional Notes

# RHEUMATOLOGY

## Systemic lupus erythematosus



- Type \_\_\_\_\_ HSR
- Must have criteria for diagnosis is \_\_\_\_\_
- MC body system involved is \_\_\_\_\_
- Type of arthritis seen is non erosive polyarticular involvement
- MC Type of anemia is \_\_\_\_\_
- MC type of anemia taken as diagnostic criteria is \_\_\_\_\_
- Leading cause of death after decade \_\_\_\_\_
- Leading cause of death in first decade of diagnosis is \_\_\_\_\_

## Drug induced lupus erythematosus

- ✓ \_\_\_\_\_ male ratio
- ✓ Renal involvement and CNS involvement is rare
- ✓ Causative drugs are

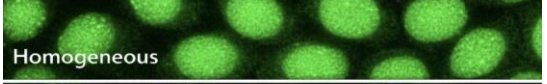
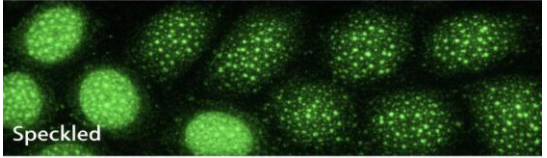
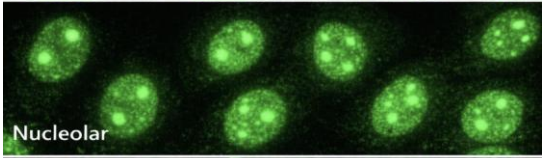
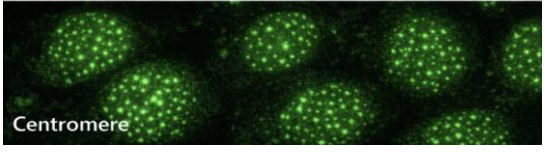
H

I

P

Methyldopa, minocycline and Quinidine

## ANA Patterns

<b>Homogenous Uniform bright staining</b>	Anti ds DNA, Anti histone	
<b>Speckled Granular dots in nucleus</b>	Anti-sm and anti RNP, SSA SSB	
<b>Nucleolar</b>	Anti Scl-70 for scleroderma	
<b>Centromere Polka dot nucleus</b>	Anti centromere CREST	

**Q. Male female ratio of 1:1 is seen in which of the following?**

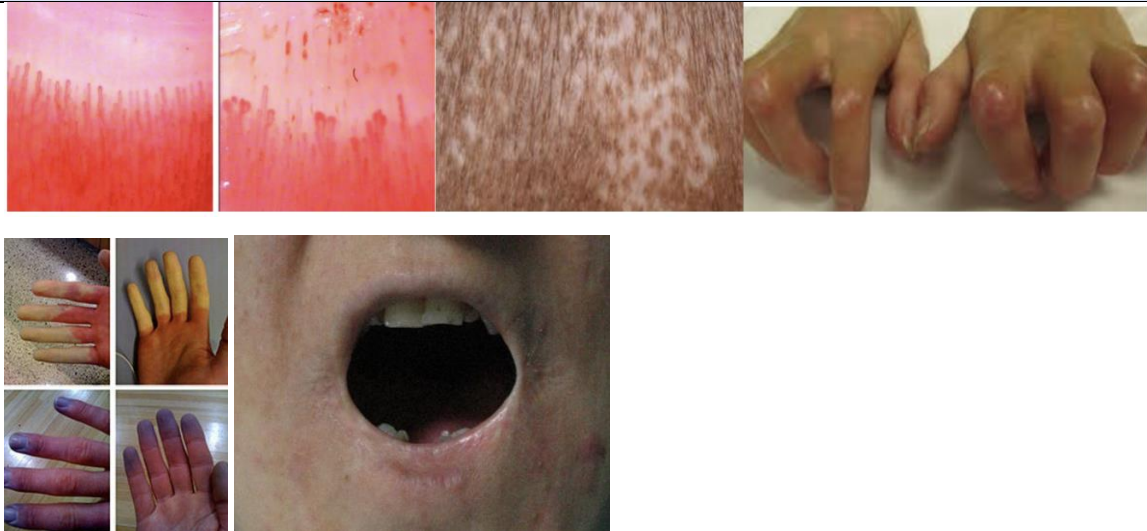
- Systemic lupus erythematosus
- Discoid lupus erythematosus
- Drug induced lupus nephritis
- Lupus nephritis

**Q. Young female presents with following lesion that worsen after hot drinks and going in sunlight**



- SLE
- DLE
- Scleroderma
- Acne rosacea

## Scleroderma



- Screening test for SSc is \_\_\_\_\_

\_\_\_\_\_ is the earliest and most common extra cutaneous manifestation of scleroderma

- High risk of B-cell NHL

- Leading cause of death in Scleroderma is \_\_\_\_\_

- DOC for scleroderma crisis is \_\_\_\_\_

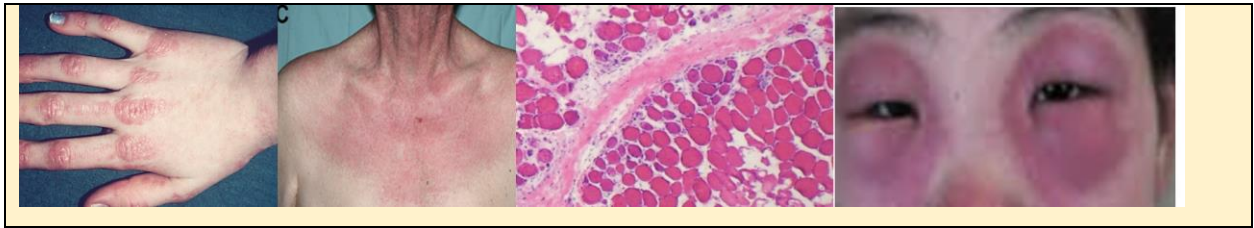
- Scleroderma renal crisis is associated with anti \_\_\_\_\_  
\_\_\_\_\_ antibody

## CREST syndrome

**Antibody of choice is Anti centromere antibody**

**Esophageal dysmotility more common than in SSc**

## Dermatomyositis



Most specific antibody seen is anti \_\_\_\_\_

Anti PM-Scl antibody is associated with PM with scleroderma overlap

Most specific antibody associated with malignancy seen in dermatomyositis

is \_\_\_\_\_ also called anti 155/140

Anti MDA-5 associated with rapidly progressive ILD

Initial investigation to be done is CK levels

Increased risk of \_\_\_\_\_ cancer in dermatomyositis

### *Clinical features*

**D** – Difficulty climbing stairs (proximal muscle weakness)

**E** – Elevated CPK

**R** – Rash (heliotrope rash)

**M** – Malignancy association

**O** - Gottron papules

**S** - Shawl sign

## Mixed connective tissue disorder

**Required**

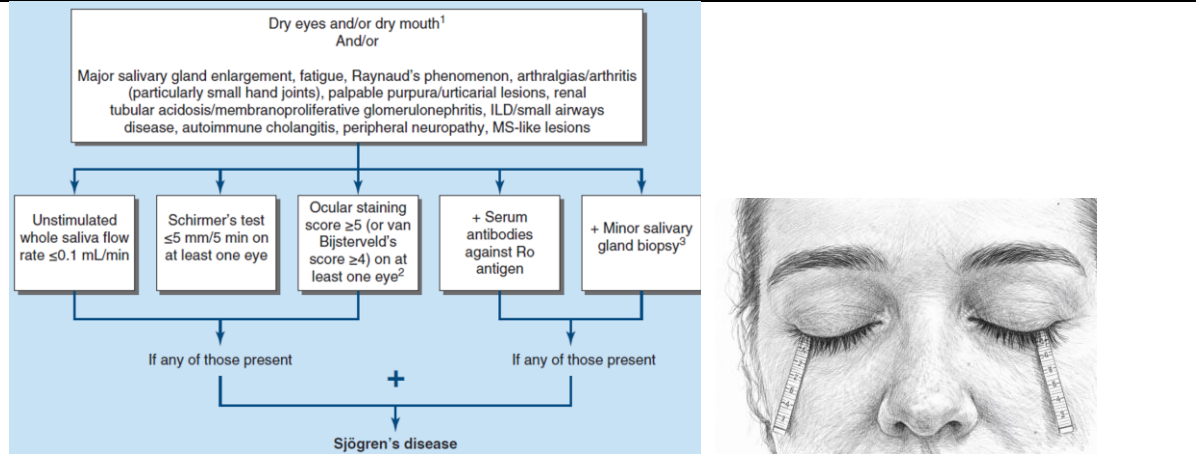
- Anti-U1 \_\_\_\_\_ antibody

**Clinical overlap features from  $\geq 2$  diseases:**

- **SLE-like:** arthritis, malar rash, serositis
- **Scleroderma-like:** Raynaud, sclerodactyly
- **Polymyositis-like:** proximal muscle weakness,  $\uparrow$  CK

## Sjogren syndrome

**Mnemonic: USE\_ROME**



- HLA DR3 association
- Secondary sjogren is associated with Rheumatoid arthritis
- Extraglandular involvement seen is arthritis

*Risk of lymphoma is ~5–15× higher than general population*

*Most common type: Extranodal marginal zone lymphoma (MALT) of salivary glands*

*Typically arises in parotid gland*

## Anti synthetase syndrome



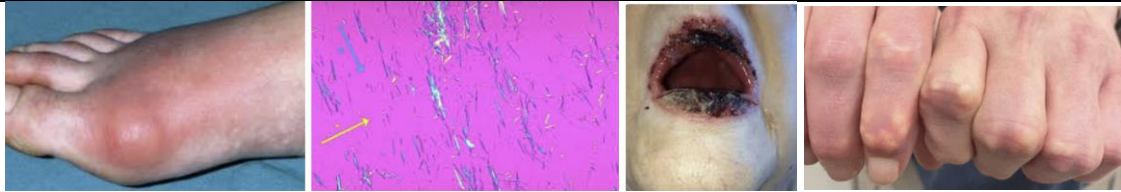
antibodies against **aminoacyl-tRNA synthetases** (most commonly **anti-Jo-1**).

### *Classic core features*

- Inflammatory myopathy (PM or DM phenotype)
- Interstitial lung disease (most important cause of morbidity)
- Non-erosive arthritis
- Raynaud phenomenon
- Mechanic's hands (hyperkeratotic palms)

# Arthritis

## Gouty arthritis



### Crystals of

### Triggers

- HTN with Thiazides
- ATT
- Alcohol binge with non veg

### C/F

- Fever with difficulty in ambulation
- Severe pain at base of big toe/ ankle / knee

### IOC

### DOC

### Chronic gout

- Tophi



-Nephrolithiasis

**Work up**

**Rx**

Allopurinol is CI

**Pseudo gout**

**MC joint involved**

**Polarized microscopy features**

**Rx:**

Polarized microscopy cannot identify crystals of calcium oxalate

High dose salicylates don't cause uric acid retention

Martel sign is seen in chronic gout and is called rat bite erosion

USG findings of MTP involvement is called double contour sign



## Extra mile

### Factors precipitating gout

Mnemonic: **CAN LEAP**

Cyclosporine

Alcohol binge and Aspirin low dose

Niacin

Lasix and Thiazides

Ethambutol

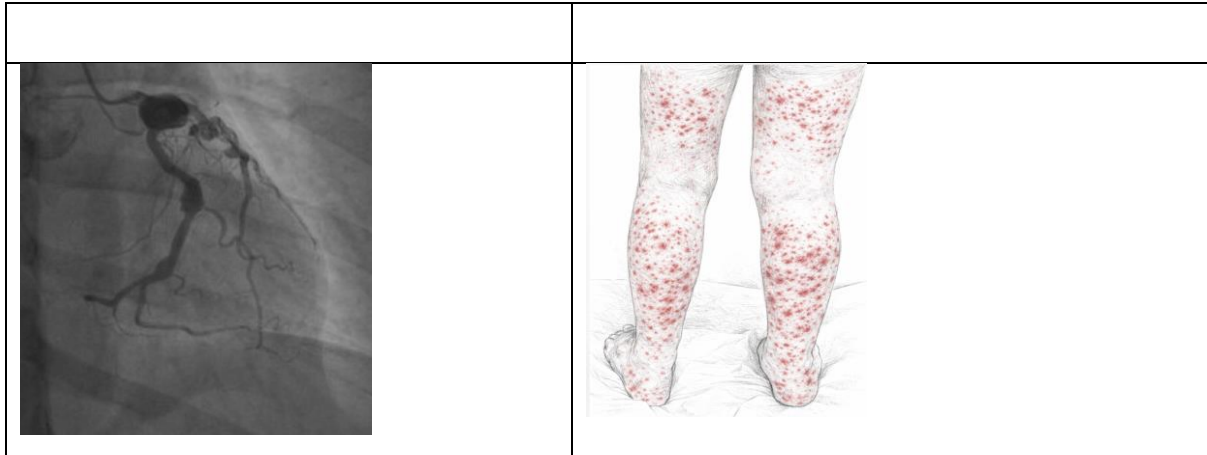
Alcohol

Pyrazinamide, purine rich diet

## VASCULITIS

**MCC of vasculitis in adults:**

**MCC of vasculitis in children**



### Giant cell arteritis



<p><b>HPE features</b></p> <p>Skip lesions seen</p> <p>Granulomatous inflammation that involves the media and intima</p> <p>Multinucleated giant cells seen adjacent to the internal elastic lamina</p> <p>Fragmentation of internal elastic lamina</p>	<p>S – Scalp tenderness</p> <p>C – Claudication of jaw (jaw claudication)</p> <p>O – Old age (&gt;50 years)</p> <p>P – Polymyalgia rheumatica association</p> <p>E – ESR elevated</p>




- ***Start steroids before biopsy***
  
- ***High-dose glucocorticoids are started as soon as GCA is suspected to prevent irreversible vision loss***
  
- **Major complication:**  
**Irreversible blindness due to ischemic optic neuropathy**

## Kawasaki disease

Life-long aspirin if *large* coronary artery aneurysms are detected

 A grayscale medical image showing a coronary artery with a significant dilation or aneurysm.	<p><b>Mnemonic:</b></p> <p><b>Fever &gt; 5 days plus</b></p>  A composite image showing three clinical features: a strawberry tongue (red tongue with white spots), a swollen face, and a hand with skin desquamation.
<p>Conjunctivitis</p> <p>Rash</p> <p>Adenopathy</p> <p>Strawberry tongue</p> <p>Hand and feet skin desquamation</p>	

## Henoch Scholein purpura

 An illustration of a person's legs from the knees down, covered in numerous small, red, raised spots representing palpable purpura.	<p>Palpable purpura</p> <p>Abdominal pain and hematochezia (ileo-ileal intussusception)</p> <p>Arthralgia</p> <p>Renal involvement (may occur)</p> <p><b>Work up:</b></p> <p>C3 is normal and Ig A is elevated</p> <p><b>Rx</b></p> <p>Steroid given for renal and GI involvement with bleeding</p>
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## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes