Structured Notes According to

MEDICINE

Revision friendly **Fully Colored Book/Structured Notes**

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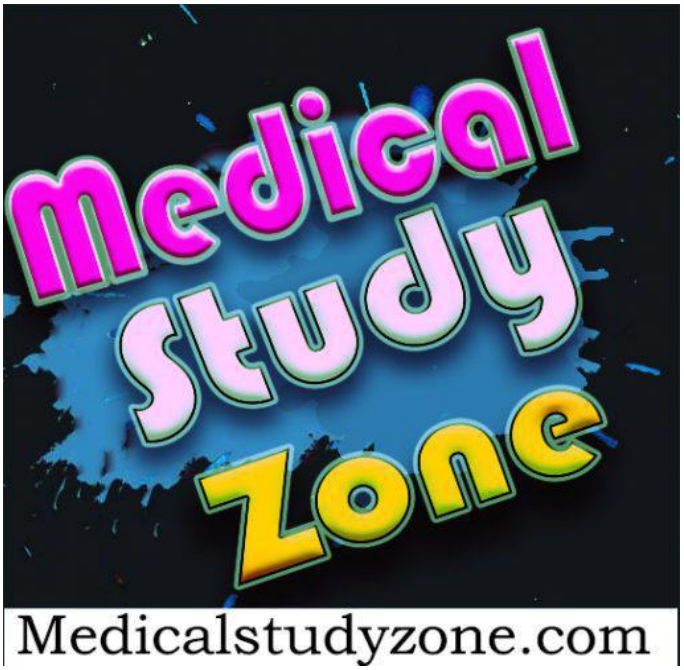

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LIST OF IMPORTANT TOPICS

CVS

- ECG most important- At least one question expected; Particulary important STEMIvs NSTEMI, Changes in Electrolyte Imbalances, Arrythmias- PSVT, A-Fib, Atrial flutter,
- V-Fib,
- RHD- MS, MR, AS, AR With Murmurs, Mx
- Pericardia! Diseases
- MI: Particularly Management
- Infective Endocarditis: Duke's criteria
- Classification of shock : Approach based Q
- Multi focal artrial tachycardia vs atrial fibrillation vs AVNRT vs AVRTsyndrome X vs syndrome Zcardiac tamponade (pulsus paradoxsus)

NEPHROLOGY

- AKI: Criteria, biomarkers
- CKD: Manifestations, Stages
- Glomerulonephritis most important
- Renal Tubular Acidosis Difference
- Polycystic Kidney vs Medullary sponge disease
- Barter syndrome
- Gittelman
- Gordon syndrome

GASTRO

- Approach to Malabsorption
- Inflammatory bowel disease: CD vs UC vs GI TB; extraintestinal manifestations
- Viral/ Alcoholic/ Autoimmune Hepatitis: Criteria, difference
- Acute Pancreatitis: Revised Atlanta classification, criteria, Mx

ENDOCRINE

- Pituitary Adenoma, Sheehan syndrome, Lymphocytic hypophysitis
- Galactorrhea amenorrhea Syndrome
- Disorders of Calcium Metabolism
- MEN syndromes
- Diabetes: Mx, MODY (to be done with pharmacology)
- Parathyroid abnormalities

LEARNING OBJECTIVES

CARDIOLOGY

Mitral Valve Prolapse with animation

- Ejection Click and Mid Systolic Click
- Causes of MVP
- Clinical Presentation
- Examination Findings
- Investigation of Choice
- Treatment

Percutaneous Coronary Intervention

- Golden Period for MI
- Treatment
- ST Elevation MI
- PCI
- Procedure
- Drug Eluting Stents
- Biodegradable Stents
- Rotabletor Atherectomy

Heart Sound 1

- Atrial Myxoma
- Tubercular pericarditis
- Constrictive pericarditis
- summary

Heart Sound 2

- Aortic Stenosis
- Patent Ductus Arteriosus
- Atrial Septal Defect
- Right Bundle Branch Block
- Left Bundle Branch Block

Heart Sounds 3 and JVP

- Heart Sounds
- Mitral Stenosis
- Soft S1
- S3 and S4
- JVP
- Waves of JVP
- Theory
- Abnormalities Related to JVP
- Absent a Waves

- Absent x Wave
- Absent y Descent
- Line Diagram (Inspiration and Expiration)
- Tamponade
- JVP

Mitral Valve Prolapse

- What is Mitral Valve Prolapse?
  - Causes of MVP
  - Pathophysiology of MVP
  - Clinical Features of MVP
  - Examination Findings
  - Treatment

Mitral Regurgitation

- Causes of Acute Mitral Regurgitation
- Chronic Mitral Regurgitation
- Murmurs
- Work Up
- Treatment

Aortic Stenosis

- Causes
- Clinical Features
- Examination Findings
- Work-Up
- Treatment

Aortic Regurgitation

- Causes
- Severe & Progressive AR
- Clinical Features
- Examination Findings
- Work - Up
- Management

Tricuspid Stenosis

- Etiology
- Examination Findings
- Work-Up
- Treatment

Tricuspid Regurgitation and Pulmonic Stenosis and Regurgitation

- JVP finding (CV wave)
- Pulmonic stenosis

Murmurs

- Mitral Stenosis
- Examination Finding
- Work Up

- Management of Mitral Stenosis
- comparison between Mitral stenosis and Aortic regurgitation
- Diastolic Murmur
- Coarctation of Aorta
- Systolic Murmur

Cardiomyopathies

- Hypertrophic obstructive cardiomyopathy
- Case Discussion
- Examination findings
- Work Up
- Treatment
- Sudden Cardiac Death
- Treatment (HOCM)
- Restrictive Cardiomyopathy
- Clinical features
- Work up
- Dilated Cardiomyopathy
- Examination
- Work up
- Treatment
- Peripartum Cardiomyopathy

Takotsubo Cardiomyopathies and Brugada Syndrome

- Takotsubo Cardiomyopathy
- Brugada Syndrome

Rheumatic Heart Disease

- Etiology & Pathophysiology
- Modified Jones Criteria 2015 Update
- Major Manifestations
- Minor Manifestations
- Diagnosis
- Treatment

Infective Endocarditis

- Incidence of IE
- Manifestations
- Modified Duke's Criteria
- Treatment
- Examples of Sterile Endocarditis
- High risk cardiac lesion before dental procedure

ECG and Arrhythmias Part 1

- ECG Analysis
- Calculation of Heart Rate
- Calculation of Axis
- P Wave Abnormalities
- PR Internal Abnormalities
- Wolff Parkinson Syndrome

- Prolonged PR Interval
- Lown Ganong Levine Syndrome
- Pacemaker
- ICD
- Mobitz Heart Block I and II
- 2:1 Heart Block
- QRS Wave Abnormalities
- Atrial Fibrillation
- Atrial Flutter
- Atrial Fibrillation Vs Atrial Flutter
- Saw Tooth Waves

ECG and Arrhythmias Part 2

- AV Nodal Reentrant Tachycardia / P.S.V.T
- Ventricular Tachycardia
- Torsades De Pointes

Multi-Focal Atrial Tachycardia

- Features
- Treatment
- Atrial Flutter v/s M.A.T ECG

ECG Changes in Hypokalemia & Hyperkalemia

- Changes in hyperkalemia
- Treatment for hyperkalemia
- Changes in Hypokalemia

Acute Coronary Syndrome Part-1

- ATHEROSCLEROSIS
- Markers of Atherosclerosis
- FRAMINGHAM CRITERIA
- Case Studies
- Chronic Stable Angina
- DUKE SCORE
- Myocardial Perfusion Imaging
- Hibernating Myocardium VS SCAR
- Drugs reducing mortality in CSA
- Failure of Medical Therapy
- Triple Vessel Disease
- Single Vessel Disease
- Double Vessel Disease
- Important Aspects Of CSA
- Unstable Angina
- Case Study
- Prinzmetal Angina
- Myocardial Infarction
- Chest Leads
- Augmented Leads
- Cardiac Biomarkers
- Infarct Localization

- LEVINE SIGN
- Types of MI

Acute Coronary Syndrome Part-2

- Case-based Scenario ST elevation MI
- Management options
- Anaphylaxis risk
- Thrombolysis: success and failure
- Contraindications of thrombolysis
- Case-based scenario: Non-ST elevation MI
- Management options
- STEMI, NSTEMI and Unstable angina - Comparison
- Complications of MI

Symptomatic Bradycardia with Pulse

- Case Discussion
- ECG Evaluation
- Chemical Pacing
- Clinical Features
- Treatment
- Trans Cutaneous Pacer
- ECG Discussion

Congenital Heart Disease

- NADAS Criteria
- Acyanotic CHD
- ASD / VSD
- Tetralogy of Fallot
- Control Cyanosis
- Tet Spells
- Work-Up
- Tricuspid Atresia
- Transposition of Great Artery
- Truncus Arteriosus
- T.A.P.V.C
- Aortic Atresia-Systemic Circulation
- Ductal Dependancy
- Ebstein Anomaly

Metabolic-Syndrome X and Syndrome Z

- Syndrome X
- Syndrome Z
- Coronary Syndrome X
- Clinical Presentation of pericarditis & Myocardial Infarction
- Investigations
- Treatment
- Pericardial Effusion
- Investigation Findings in pericardial effusion
- Cardiac Temponade
- Pulsus Paradoxus

- Theory of all diseases of pericardium
- Massive Pericardial Effusion
- Clincal case discussion
- Vitals in Cardiac Tamponade
- Clinical scenarios in case of car crash
- Constrictive Pericarditis
- Work-up for constructive Pericarditis
- Treatment
- Differences among acute pericarditis, Pericardial effusion, cardiac temponade, cardiac pericardit

Hypertension

- AOBP & ABPM
- Causes
- Investigations
- Treatment
- Resistant HTN
- HTN Urgency & Emergency
- Target BP

Diseases of Pericardium

- Acute Pericarditis

Congestive Heart Failure

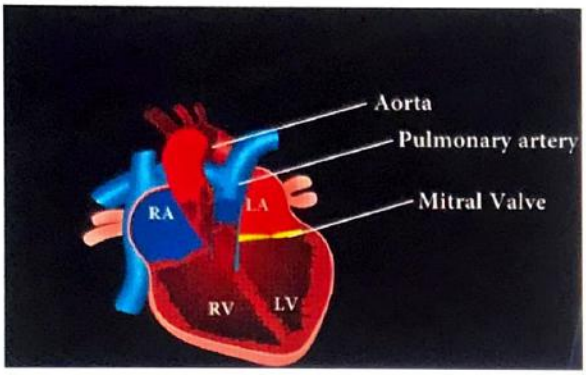
- Framingham Criteria
- Stages of CHF
- Investigation & Diagnosis
- Treatment of Heart Failure

Bundle Branch Block

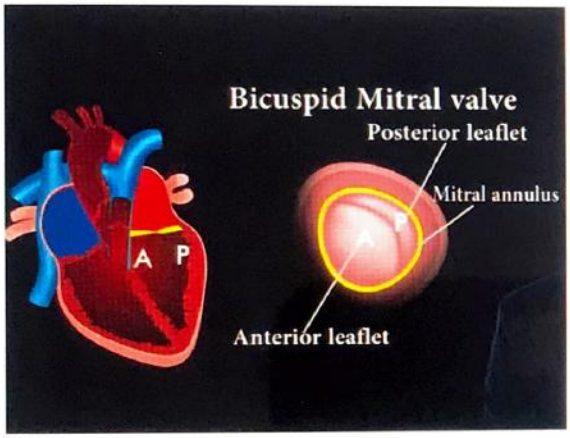
- ECG- Left bundle branch block
- ECG- Findings of LBBB
- Sgarbossa criteria - LBBB & AMI
- Right Bundle branch block (RBBB)
- LBBB V/s RBBB

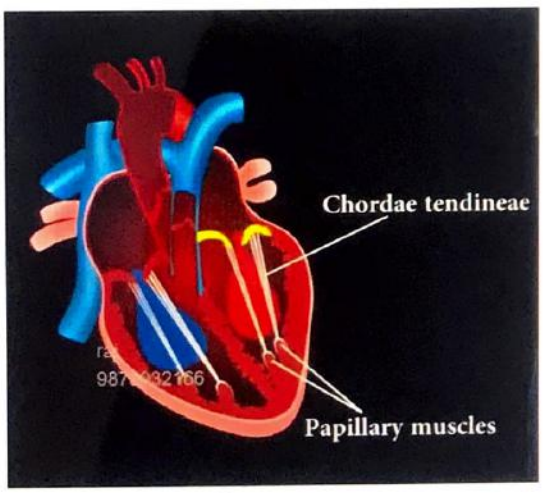
1 MITRAL VALVE PROLAPSE WITH ANIMATION

- Also known as floppy valve syndrome /Barlow syndrome
- During phase of systole LV generate tremendous amount of pressure that push blood into aorta.

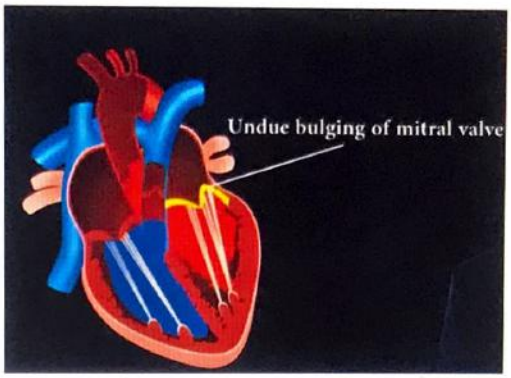


- Creates lot of pressure on mitral valve leaflets.
- Bicuspid mitral valve able to retain its position d/t
  - Chordae tendineae
  - Papillary muscles





- IMVP; under bulging into lumen into left Atria.



- Creates: Mid systolic click
- Due to under stress

Important Information

- Normal finding during ventricular systolic
  - Represent aortic & pulmonic valve opening

MID SYSTOLIC CLICK

00:01:43

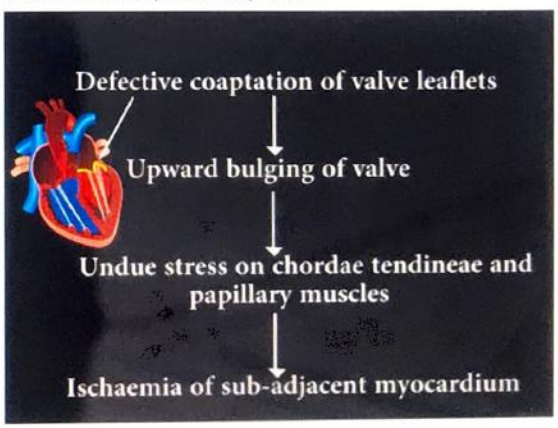
- Due to extra tension generated in the chordae tendineae during exercise bulging up of mitral valve

MVP

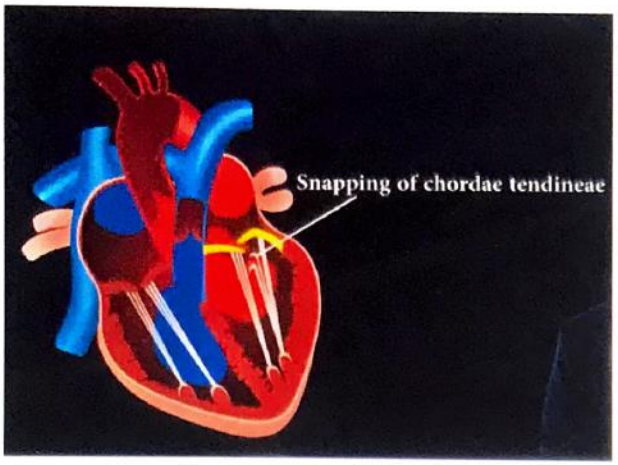
00:02:02

2 problems

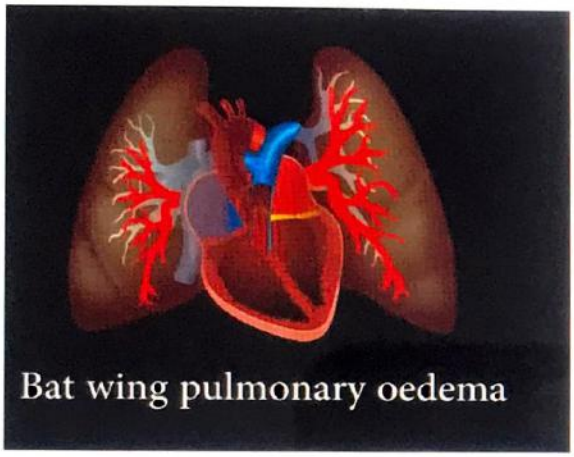
1. Defective coaptation of valve leaflets
   - Upward bulging of valve
   - Under stress on chordae tendineae and papillary muscles
   - Ischemia of sub adjacent myocardium
   - Cause: chest pain, arrythmia



2. Snapping of chordae tendineae
   - Incompetent valve
   - Mitral regurgitation



During ventricular systolic
- 10-30 ml of blood levels into lumen of 1 atrium
- Structural damage to left atrium
- Left ventricular failure (pulmonary oedema)
- Bat wing pulmonary oedema



CAUSE

00:03:18

- Idiopathic
- Defect of type 3 collection
- Connective tissue disorders like martan syndrome, osteogenesis imperfecta, Ehler Danlos syndrome
- ADPKD
- Straight back syndrome
- Ostium secundum ASD

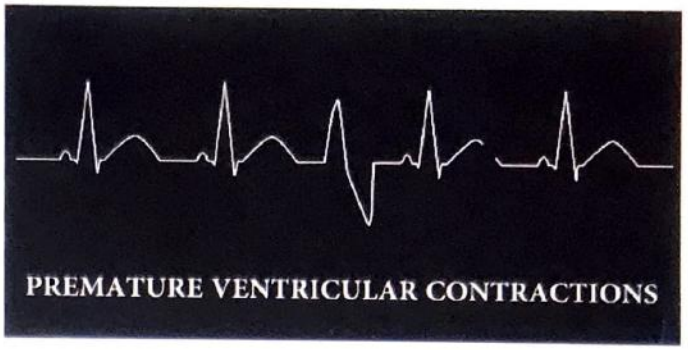
CLINICAL PRESENTATION

00:04:00

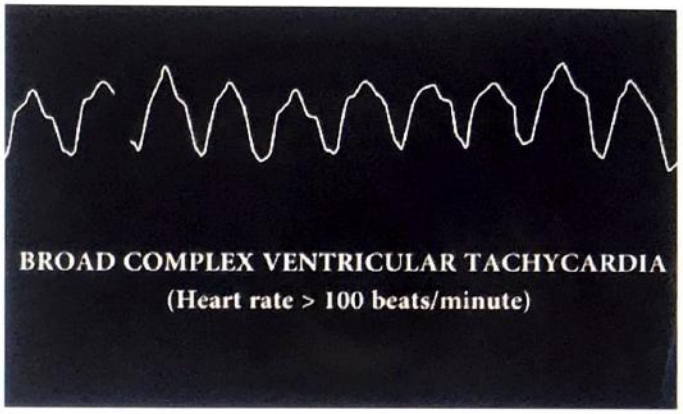
- Asymptomatic (M/C)
- Palpitations
- Syncopal attaches
- Orthopnea
- Paroxysmal nocturnal dyspnoea (PND)

RHYTHM DISORDER

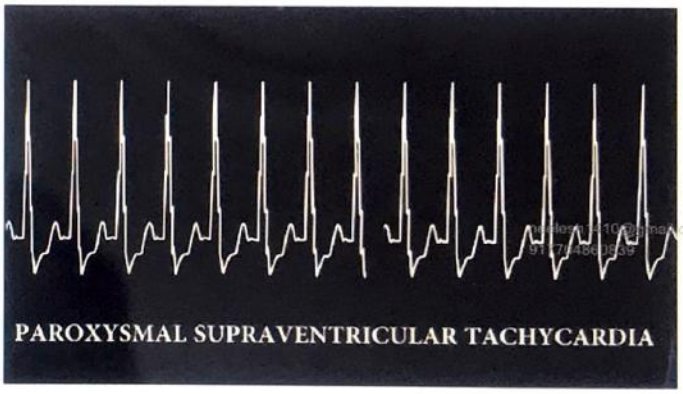
1. Premature ventricular contraction



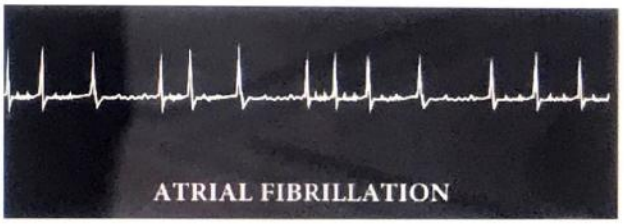
2. VT
   - HR> 100 bpm



3. PSUT



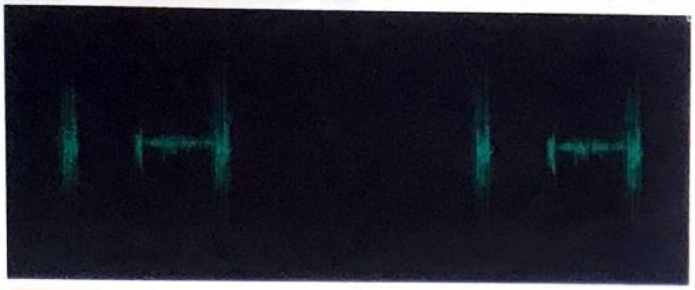
4. Leakage of blood from LV to LA
   - Structural damage to LA
   - Atrial fibrillation



EXAMINATION FINDINGS

00:06:07

1. Mid systolic clicks
2. Late systolic murmur (d/t leakage of blood into L.atrium)

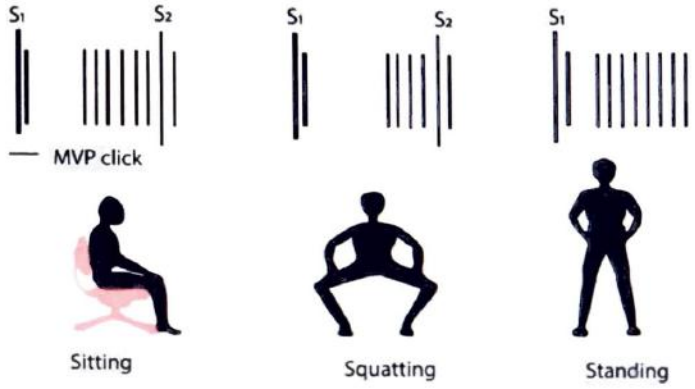


Important Information

- Ejection systolic murmur heard at the phase of aortic stenosis (crescendo-decrescendo murmur)
- Late systolic murmur : mitral valve prolapse
- Ejection systolic murmur : aortic / pulmonary stenosis

MURMUR ON SQUATTING & STANDING

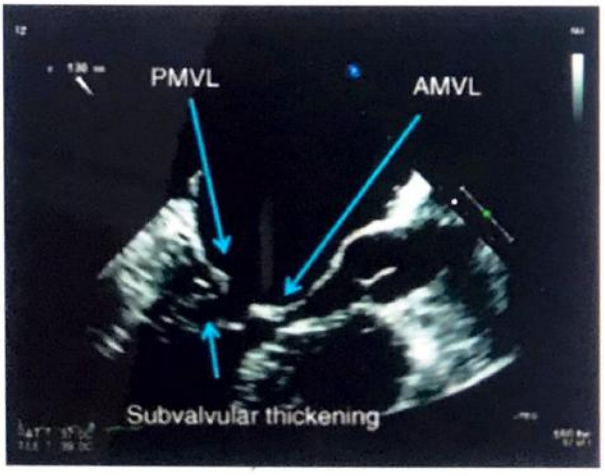
- Squatting: Shorter
- Standing: Longer

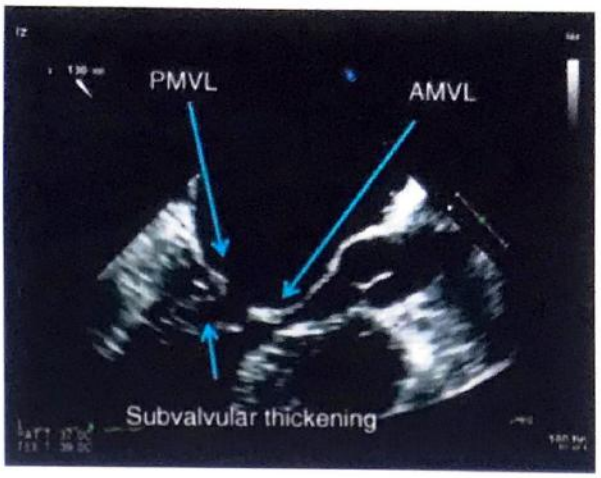


IOC

00:08:00

1. Echocardiogram





- Post leaflet defect
  - Jet of blood moves anteriorly
  - Murmur radiates to base of heart
- Ant. Leaflet defect
  - Jet of blood moves posteriorly
  - Murmur radiates to axilla /back

TREATMENT

00:09:04

1. ß blockers
   - Control heart rate
2. Prophylaxis for infective endocarditis
3. Mitral valve repair
   - To prevent mitral regurgitation

2 PERCUTANEOUS CORONARY INTERVENTION

- Golden Period of MI: 1 " hour during this, person can die suddenly due to sudden cardiac death
  - Causes of sudden cardiac death in Post MI patients
- Tachyarrhythmias – VF
  - TOC: Defibrillation
- Bradyarrhythmia: Mobitz II heart block
  - Rx: Atropine, TCP (Transcutaneous Pacer)

↓

To accelerate heart rate

- Patient with ST elevation MI

Thrombus in right coronary artery

Significant ↑ST elevation in Leads 2, 3, aVF (Inferior leads)

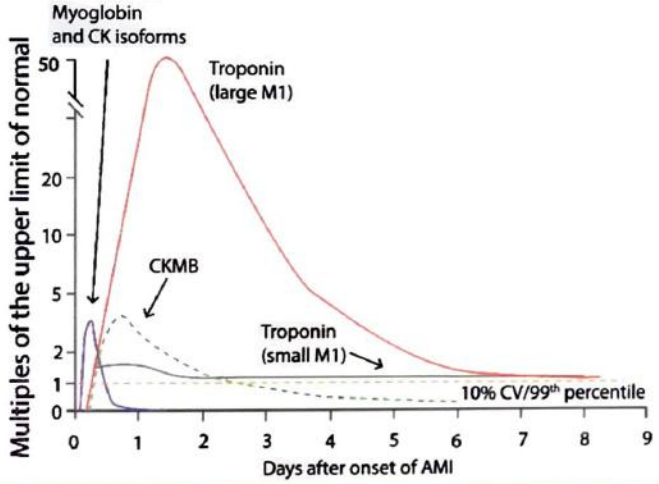
↓

Helps in identifying inferior wall MI

Minimum ST elevation that should be present in

- At least
  - 2mm: Males
  - 1.5mm: Females

TroponinI values show increasing trend



Important Information

- Tropinin I rises by approx. 3 hours so if a person reaches hospital early Troponin I can be normal. That's why we are taking serial evaluation of Troponin I

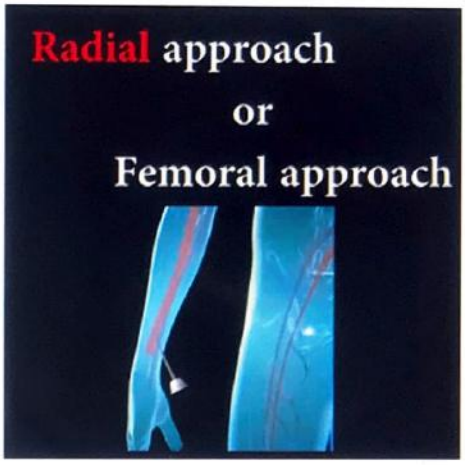
- Troponin I values usually start doubling by 3-4 hours.
- Depending on the severity of illness: it can be triples

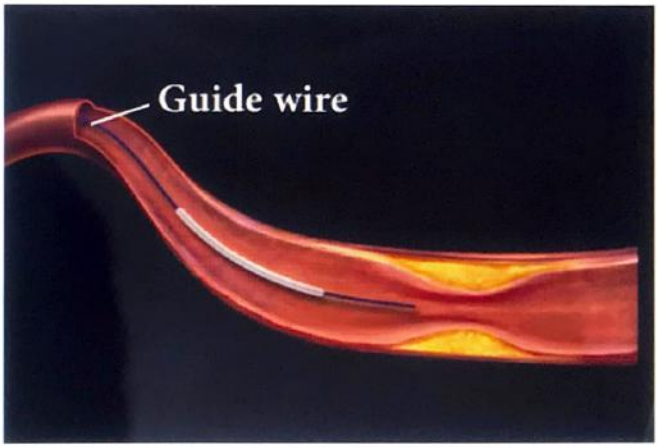
PCI (BALLOON ANGIOPLASTY)

- Ideally done within 90 minutes of patient arriving in hospital
- Door to balloon time: within 90 minutes

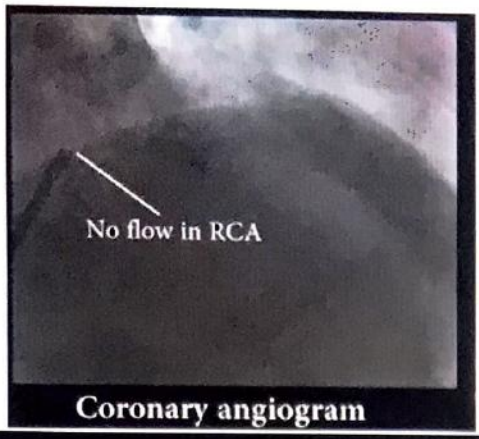
Procedure

- Preferred approach: Transradial approach

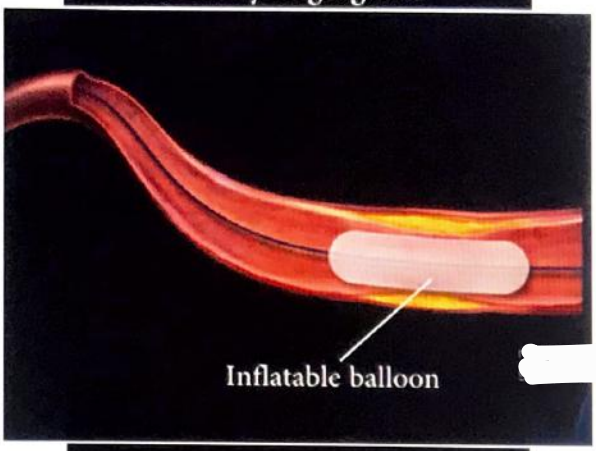


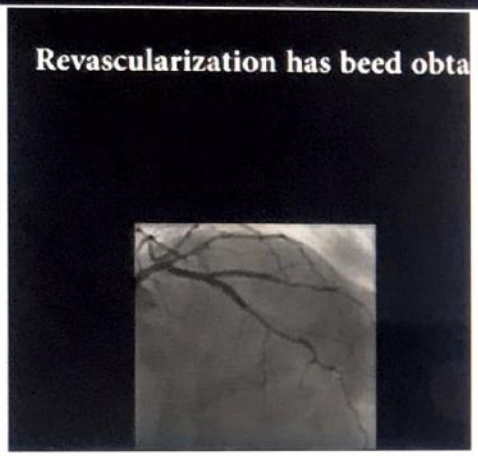


- Guidewire will navigated up from Radial artery→ to the subclavian artery root of Aorta → right coronary artery→ guidewire is steered through thrombus → Inflate the balloon → The force of the balloon will destroy the clot→ Revascularization has been obtained.



Coronary angiogram





The bad news is the atherosclerotic process is still in the blood vessel, there is high probability that atherosclerotic plaque might result in the development of MI at the same site or a site proximal/ distal to the original site.

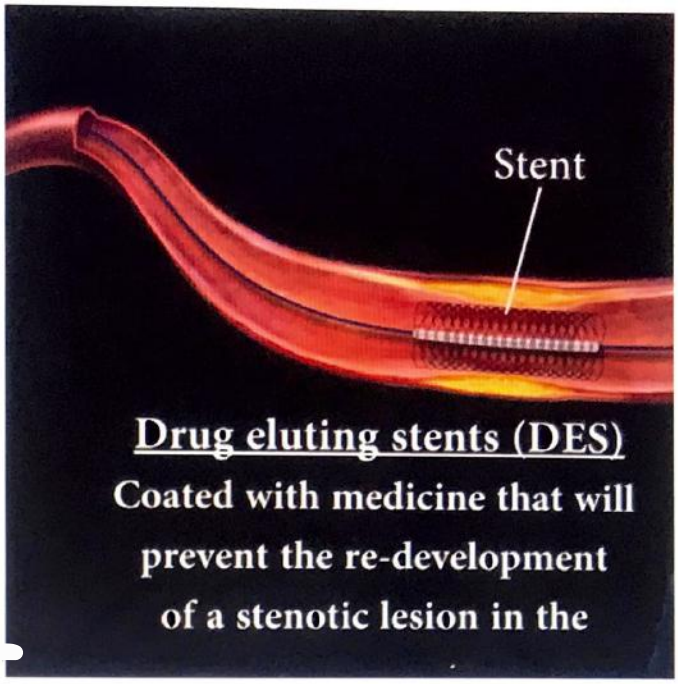
To prevent the development of atherosclerosis from progressing further. We have to give

- Statins
- Deployment of stent
- Most of the stents that are available now days are called as Drug eluting stents (DES)

DRUG ELUTING STENTS (DES)

- Coated with medicine that will prevent the re-development of a stenotic lesion in the coronary artery.
- It is coated with two medicines

1. EVEROUMUS
2. ZOTEROLIMUS



Advantages of DES

- Modify the atherosclerotic process
- Prevent re-development of acute coronary syndrome.

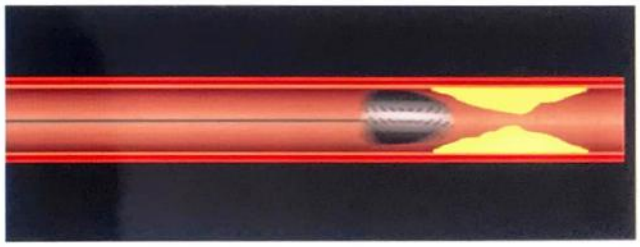
BIODEGRADABLE STENTS

- Get incorporated into the wall of blood vessels
- Modify atherosclerotic process

ROTABLATOR ATHERECTORY

- Device used for patients having chronic stable angina
- To facilitate opening of the narrowed artery, so that stent can be deployed

Balloon Angioplasty: If occlusion of artery by thrombus
Rotablator Antherectomy: If atherosclerotic plaque cause fixed obstruction

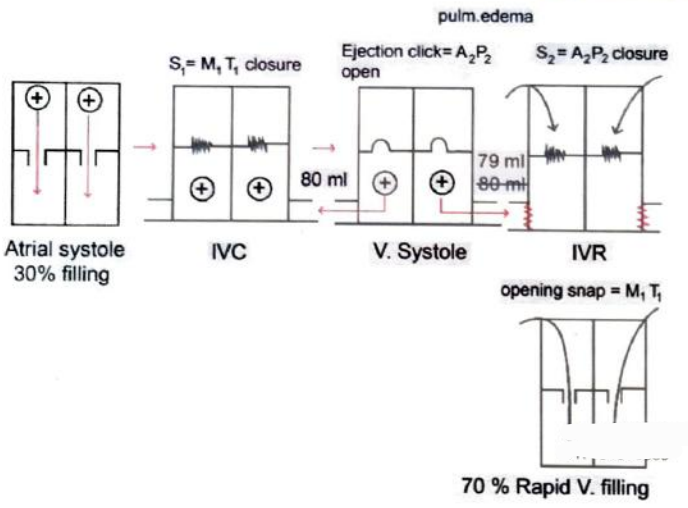


SUMMARY

1. PCI stenting is a procedure that is done in ST elevation MI and should be done in within 90 minutes
2. Rotablator atherectomy: Helps in overcoming obstruction in chronic stable angina, where patient having fibrous coalification plaque resulting in narrowing of the blood vessels.

3 HEART SOUNDS PART-1

00:00:51



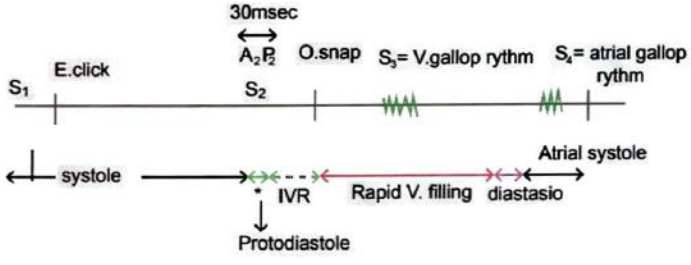
Atrial systole 30% filling

- $S_1 = M_1 T_1$ closure
- IVC (Isovdumetric contraction)
  - S_1= ends of atrial systole

- Ejection click = A_2P_2 open
- Equal amount of blood go through both chambers
  - Ejection click – during ventricular systole

- S_2= $A_2 P_2$ valve close
- Venous filling of atria starting
- IVR (isovolumetric relaxation)

- Opening snap = $M_1 T_1$ (end of isovolumetric relaxation
- 70% rapid venti filing



- $S_1 S_2$ – lub & dub heart sounds
- Duration of systole is lesser then diastole
- Systole – period between S_1 & S_2
- Right after S_1 there will be ejection click
- Moment systole is ending that period of time aortic & pulmonic valve closing
- Aortic & pulmonic valve do not close together at same time
  - First aortic valve close than pulmonic and there is a gap of 30 milli sec
- Moment systole will end, means blood has gone out of the heart
- There will be period where systole has ended and aortic valve will closed

PROTO DIASTOLE

00:10:55

- Period of opening snap (between A_2 & opening snap) isovolumetric relaxation.
- Phase of rapid ventricular filling
- 70% of filling of blood in heart of patient
- Rapid ventricular filling ended – there will be momentary period, when there is nothing happening → diastasis

Important Information

- **Least cardiac motion is present during the phase of diastasis**

Divided diastole in 5 components

1. Protodiastole: Starts from end of systole (aortic valve closure)
2. IVR
3. Rapid ventricular filling
4. Diastasis
5. Atrial systole

ABNORMAL HEART SOUNDS

00:13:54

1. S_3 also known as ventricular gallop rhythm
2. S_4 also known as atrial of algorithm

Important Information

- Heart sound present right before S_1 is S_4

- S_4
  - In long stranding hypertension
  - Which is due to LV hyper whch cause left atrial hypertrophy
  - Atrial will generate more power, creates turbulence which produce S_4 sound
- S_3
- Is heard in the phase of rapid ventricular filling

Important Information

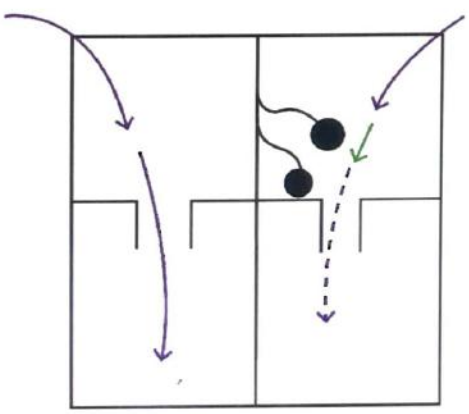
- High pitch heart sound: heard in diaphragm
- Low pitch heart sound: Bell
  - S_3
  - S_4
  - Tumor plop sound
  - Murmur mitral stenosis

TUMOR PLOP SOUND

00:17:27

i. Low pitched heart sound
ii. Diastolic finding
iii. Tumor impacts an already open mitral valve

- Atrial myxoma
- Pedunculated tumor
- Has stalk, suspended in left atrial
- The movement blood enters into chamber, it will not tumor, and tumor will swing like a pendulum tumor will strike the mitral leaflet and the sound produced is called tumor plop sound
- Amount of blood enters into LV will be less



iv. Sound will be heard after the opening snap
v. Tumor originating from inter atrial septum

Clinical Scenario

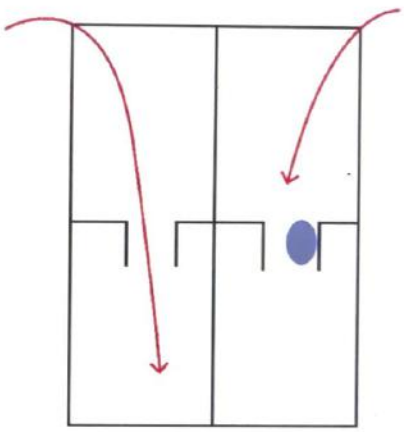
- Young female e/o effort intolerance dyspnea on exertion. Feel more breathlessness while sitting and resolve when lying down, (platypnea). O/E no pallor, tumor plop sound, mid or late diastolic murmur developed to transient ischemic attacks (TIA). IOC transthoriacic echocardiography.

Rx: refer to cardio thoracic vascular surgery (CTUS) & surgical resection of tumor will be done.

TUMOR ORIGINATING FROM HEART VALVES

00:27:42

- Papillary Elastoma
- Can also have embolic manifestation
- Can have TIA events
- No tumor plop sound present



- Treatment: Relapse mitral valve (putting prosthetic valve)

Important Information

- MC tumor heart
  - Oat cell lung Ca
  - Ca breast
  - Malignant melanoma
- MCI malignant tumor of the heart is Angiosarcoma
- Benign tumor of the heart is A. Myxoma

TB PERICARDITIS

00:31:41

- Patient having tubercular pericarditis, form of extra pulmonary tuberculosis

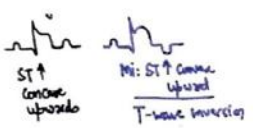
- Serofibrous exdutate outside the heart, cause inflammation, irritation to phrenic nerve
- C/O
  - Chest pain at rest
  - Radiation
  - Pain radiating to left shoulder

Important Information

- **M/C form of extrapulmonary Tb is cervical lymphadenitis**
- **Gohn focus develop on the tensil**

- Low grade fever
- Drenchng night sweats
- Weight loss involuntary (5% weight loss over 6 months or 10 pands)

IOC
- ECG
- ST elevation

	Pericarditis	MI
	• ST ↑ concave In upward direction In all Leads Except (aVR)	• ST ↑convex upward direction • T- wave inversion

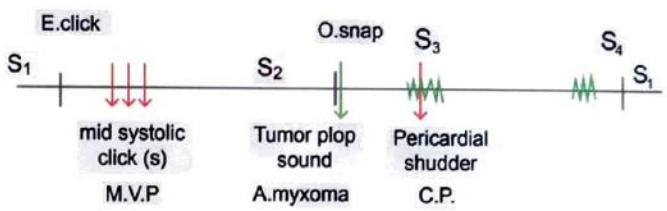
Auscultation
- Pericardical friction rub

Pericardial friction rub	Pleural rub
• Will be present even when patient holds breath	• Heard in the phase of deep inspiration

Echo
- Presence of pericardial fluid
- CBNAA T with pericardial fluid

Treatment
- ATT
- After treatment there might be development of calcification
  - Constrictive pericarditis is the sequelae after treatment
- Amount of blood enter will be lesser
- And when it enter the chamber, it cause turbulence which make a sound called pericardial shutter
  - Pericardial shutter is the diastolic phenomena
  - Heart sound will be heard after the opening snap
- Also known as pericardial knock or pericardial shock

00:45:57



Important Information

- **Presystolic heart sound = S_4**
- **Midsystolic click = Mitral valve prolapse**
- **Tumor plop sound = Atrial Myxoma**
- **Pericardial shulter = S_3(restrictive Pericarditis)**

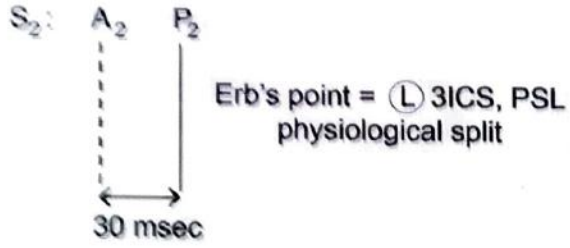
4 HEART SOUNDS PART-2

00:00:15

S

- Heard by the closure of the aortic & pulmonic valves

S_2 : A_2 P_2



- Time lag between closing of aortic & pulmonic valve is 30msec which is called as splitting
  Erb's point- is the point where splitting is best heard
- It is on left side 3rd intercostal space on the parasternal line
- Splitting is best heard in the phase of inspiration
- It is referred as physiological splitting and it varies with reparation
  - If duration is <30msec: narrow split heart sound
  - If duration is >30msec: wide split heart sound

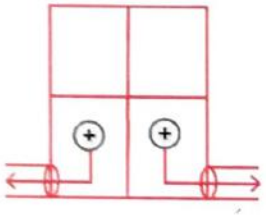
Important Information

- **Normal size of mitral valve : 4-6 cm^2**
- **Normal size of aortic valve : 2.5-4cm^2**

- Aortic stenosis < 2.5 cm^2
- Severe < 1 cm^2 or 0.6 cm^2 / m^2 of body surface area
- Mitral stenosis severe <1.5 cm^2

Aortic stenosis

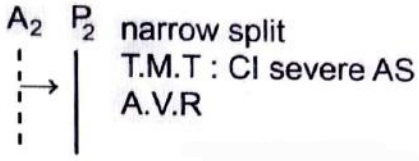
- Blood exit will take extra time



Aortic stenosis < 2.5 cm^2

SEVERE < 1 cm^2

Exit : Extra Time



- Narrow split second heart sound
- Exercise is not recommended in these patients
- Treadmill test is contraindicated

Treatment: Aortic valve replacement

- If Aortic valve replacement is not done the orifice will become narrower and narrower, and at one point if treatment not done the aortic and pulmonic valve will close at the same time
- If both valves will start closing at the same time is called single S_2
- If aortic valve closure occur after the pulmonic valve closure than it is called reverse splitting of second heart sound or paradoxical split
- $P_2 \rightarrow A_2$

Important Information

- **Reverse splitting of S2 represents max. severity of valvular aortic stenosis**
- **Normal size of aortic valve : 2.5-4cm^2**

CAUSES

00:08:15

- Infants : Bicuspid aortic valve
- Child : Rheu fever
- >65 yrs : Senile calcification

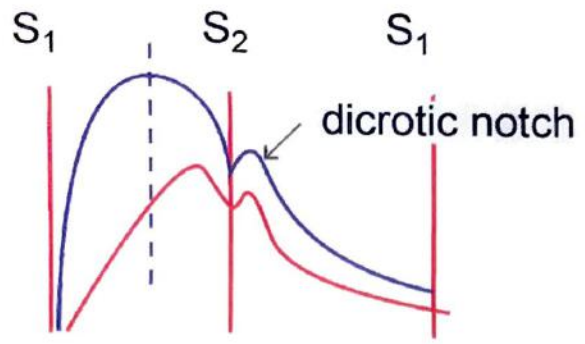
Clinical findings

1. **S - S**yncope on exertion
2. **A - A**nging : Subendocardial
   : Ischemia D
3. **D - D**yspnea : LVEDP

How to remember

- SAD

Examination findings



- Red line
  - Slow rise
  - Pulse because of physical obstruction
  - Peak is also delayed
  - Amplitude leuko
- This is called pulses parvus et Tartus
- Also called Anacrotic pulse

Important Information

- **Dicrotic pulse : Dilated cardiomyopathy**
- **Anacrotic pulse : Valvular aortic stenosis (Pulses Parvus et Tardus)**

- Pulse pressure will be less because systolic blood pressure will be reduced
- Heaving apex beat (LVH)
- Double apical impulse
- Presence of (Carotid thrill)

Important Information

- **Plancing carotids : AR**
- **Carotid thrill : AS**

Ejection systolic murmur intensity ↑ / ↓

- S4 present
- S2 : Narrow / single Parodoxical

IOC

- TTE (Transthoracic echocardiolography)
  - Shows the size of the orifice reduced
  - Ejection velocity >4m/sec
  - Transvalvular gradient >40mmHg

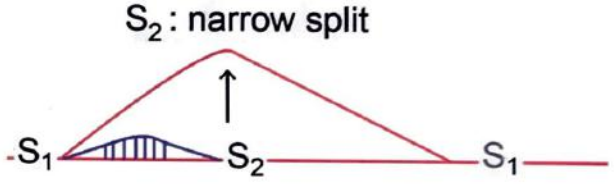
PDA (PATENT DUCTUS AORTOSIS) 00:21:02

Causes

- Preterm
  - Hypoxia produces PGE_2 which causes patent ductus aortosis
- Term
  - Cong. Rubella syndrome
  - L →R Shunt
  - LVF
  - Ex it of blood take extra time
  - Aortic valve will close later
  - Narrow split second heart sound
  - Preterm : Sympathetic at birth
  - Term : 6-8 wks
  - Efforts intolerance (breathlessness on feeding)
  - Sweating on forehead
  - Irritable

O/E

- S_2 : narrow split



Continous murmur
machinery murmur

- Necrotizing enterocolitis ↑ risk

IOC

- TTE

Treatment

- Indomethacin / Ibuprofen
- Term : Surgical ligation

CONDITIONS WITH NARROW SPLIT S2 HEART SOUND 00:30:16

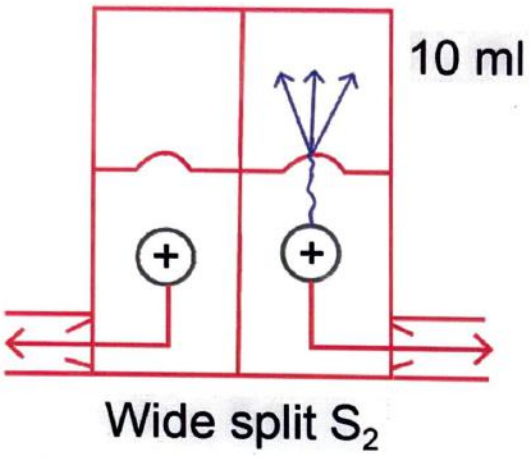
1. AS

2. HOCM / Sub valvular AS
3. LVF :
- Ant wall MI
- Coxsachie B myocarditis
- Wet Beri Beri
- Severe anemia
- PDA

MR

00:32:30

- Blood shunting from LV → LA
- Less blood will go from heart
- Exit of blood : lesser time
- A_2 will more to left

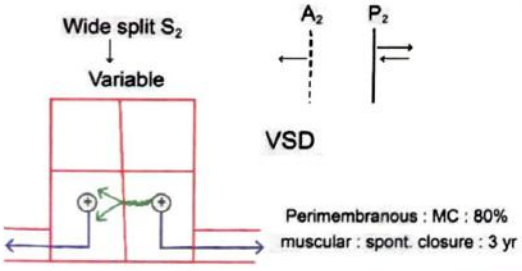


- Gap will be increased
- Wide split sound S_2
- Duration between A_2 & P_2 > 30msec

VSD (VENTRICULAR SEPTAL DEFECT)

00:34:46

- Shunt from LV → RV
- Less blood will go from heart
- Exit of blood : lesser time
- Wide split sound S_2

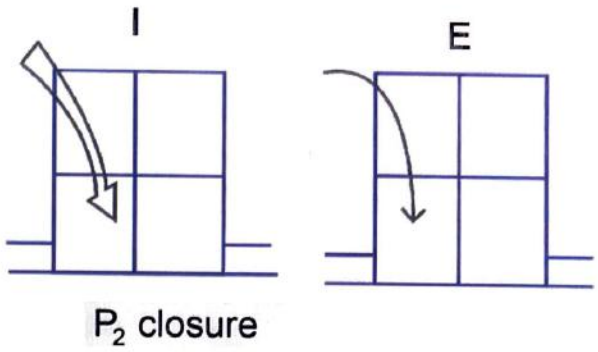


VSD TYPES

00:36:08

1. Peri membraneous – M/C 80%
2. Muscular : spontaneous closure by 3yr
3. Supra cristal – worst associated with AR

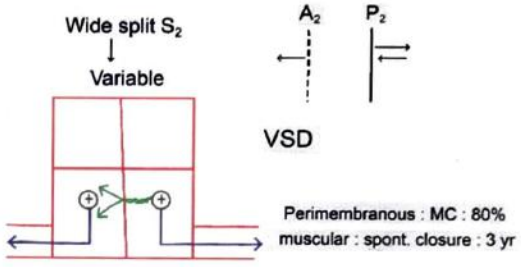
- P_2 closure varies with inspiration & expiration
- Whether its VSR or MR
  - A_2 will be early
  - P_2 will oscillating with respiration (wide variable split S_2 sound)



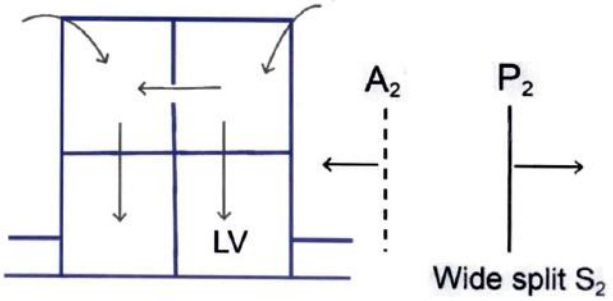
ASD

00:40:05

- Low pressure shunt
- RV will have volume overloading
- More blood will take more time to exit
- P_2 will be delayed
- A_2 will close early



- Wide fixed split S_2 sound

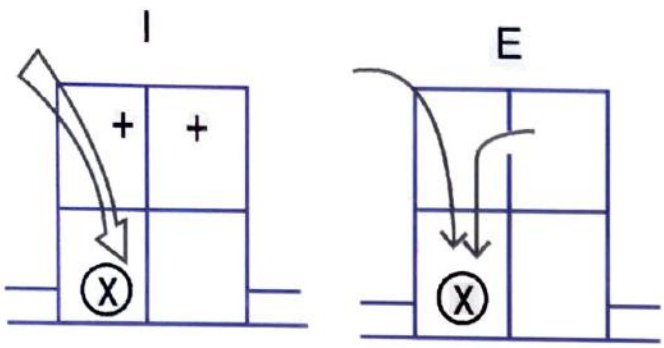


More Time for exit — less Time for exit

P_2 : delayed closure — A_2 : close early

During Inspiration

- More blood will be coming because during inspiration negative pressure will be created and such more blood into the chest cavity.
- No pressure difference
- No shunting



- Amount of blood coming will be lesser
- Shunting

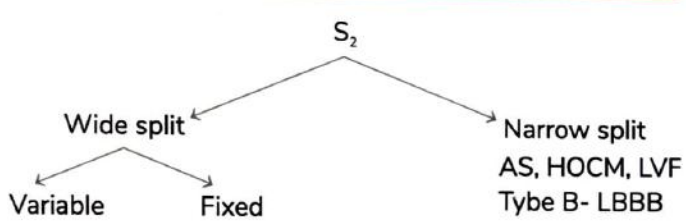
Important Information

- Normal person – Amount of blood coming in expiration & inspiration is differential
- ASD- amount of blood coming in exp & inspiration is same

In normal person

- P_2- delayed in phase of inspiration
- P_2- earlier during expiration

ASD patient - fixed



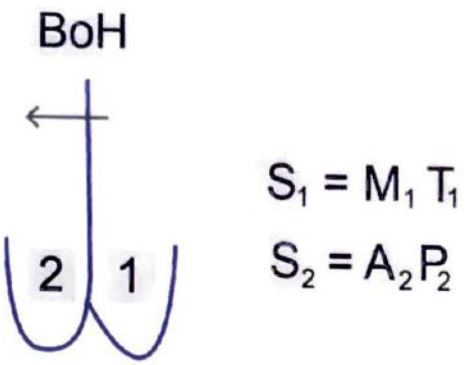
Variable	Fixed
VSD	ASD
MR	1. Lutenbacher syndrome : ASD + MS
WPW type A	2. Holt oral syndrome : ASD+Radius bone defect
RBBB	3. Down syndrome : endocardial cushion defect

TYPES OF ASD

1. Ostium secundum MC
2. Ostium primum / AV canal defect / endocardial cushion defect
3. Sinus venosus

BUNDLE OF HIS

00:49:24



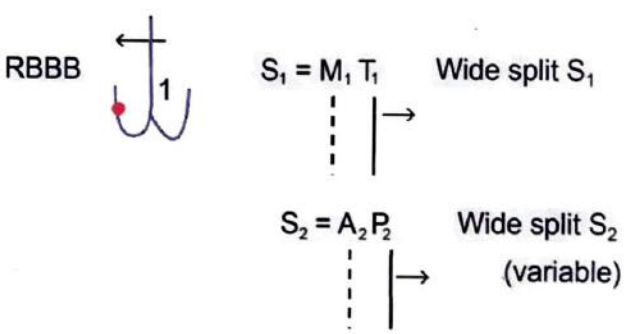
- Refractive period of 2 vesicles are totally different
- Current first goes to left vesicle and then right vesicle
- So mitral valve will close first in S,

$S_1 = M_1 T_1$

- In the same way Aortic valve will close first

$S_2 = A_1 P_2$

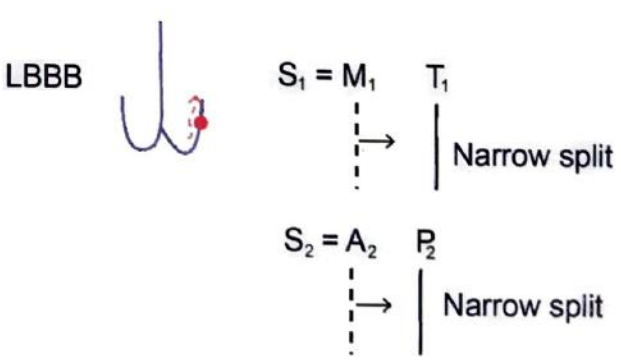
- The closure of heart valve is not only dependent on pressure but on electrical issues also



- Delayed information to the right side valves
- Wide split S_1
- Wide split S_2 (variable)

LBBB

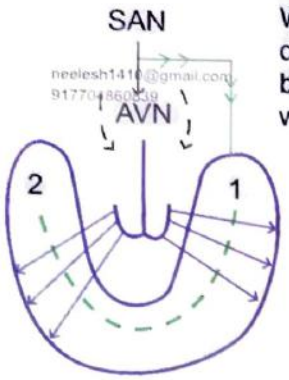
00:53:05



- Mitral valve closing late
- Gap less
- Narrow split

WOLF PARKINSON WHITE SYNDROME WPW TYPE A

00:55:10



WPW : Type A
delayed inf RV
behave like RBBB
wide variable split S_2

- Bundle of Kent – by pass tract
- Current bypass AV node
- LV will activate first and RV will activate later

WPW : Type A

- Delayed inf. RV
- Condition will behave like RBBB
- Wide variable split S_2

WPW: TYPE B

00:57:28

- RV will activate first
- Current will not enter into Purkinje
- Delayed inf into LV
- Behave like LBBB
- Narrow split S_2

5 HEART SOUND PART-3 & JVP

FIRST HEART SOUND S_1 = MITRAL ANT. TRICUSPID VALVE (M_1 T_1) CLOSURE

00:00:20

- Intensity ∝ speed of closure

Loud S_1

- Loud S1 denotes Tachycardia
- PR ∝ 1/HR

1. **Tachycardia**
- Short P-R interval
- CHF
- Pheochromocytoma
- Thyrotoxicosis
- S. anemia

2. **Physiological**: children / pregnancy

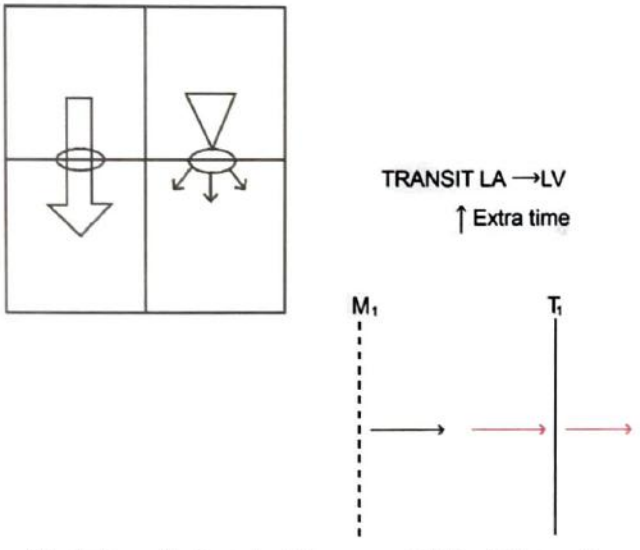
MITRAL STENOSIS

00:03:20

- There is a physical obstruction that leads to increase in left atrial pressure. This leads to
- Transvalvular gradient ↑
- Faster opening of mitral valve
- Faster elastic recoil mitral valve
- MS & TS → Loud S_1

Soft S_1

Calcified MS → Elastic recoil decreases → Soft S_1



Variations in S_1 asked in case of Mitral Stenosis

1. Narrow split S_1
2. Single S_1
3. Paradoxical Split S_1
4. Loud S_1
5. Soft S1 (due to calcification of valve)

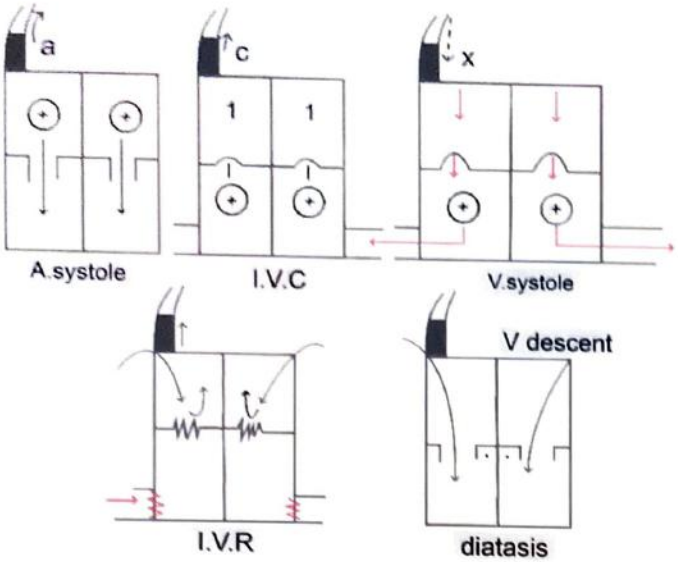
- Soft S_1 denotes bradycardia
  - Prolonged P-R interval
  - Hypothyroidism
  - SAN #: Sick sinus syndrome
  - AVN #: 3rd degree H. block
  - BOH #: Mobitz II H. block
  - Inf. Wall M
  - Morbid obesity (Fat in chest wall) / emphysema (Air trapping)
  - Calcified MS / TS (Due to reduced elastic recoil)
  - MR / TR

00:13:50

S_3 Ventricular Gallop Rhythm	S_4 Atrial Gallop Rhythm
Early diastolic sound	Late diastolic sound
Rapid V. filling in dilated ventricles	Outflow tract obstruction[LVH / LAH]
1. CHF	1. AORTIC STENOSIS: Left sided S_4
2. DCM	2. HOCM: Left sided S_4
3. COPD: Cor pulmonale	3.HTN (Long standing): Left sided S_4
4. PE - Acute cor pulmonale	4. Pulmonic stenosis: Right
Cor pulmonale means RVF due to Non- Cardiogenic causes	5. PAH: Right sided S_4

Important Information

- P wave absent (ECG)
- a wave absent (JVP)
- S_4 Absent in A. fibrillation



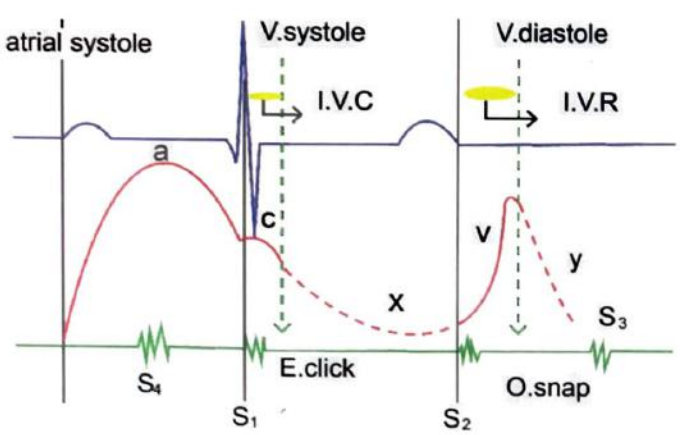
JVP

00:25:42

A = A. systole
C = IVC, Bulge
X = V. systole
V = IVR, rebound
Y = Diastasis
JVP = Internal jugular vein 5-8 cm H_2O @ angle of Louis
Pulsations seen, never felt → 2x carotid frequency
Deep Inspiration → JVP falls → normal
C. pericarditis + deep inspiration → JVP rises (Compliance ↓)
Causes of Kussmaul sign (CRR)
1. Constrictive Pericarditis (Calcification)
2. Restrictive Cardiomyopathy (Fibrosis)
3. Right CHF (Inf. Wall MI / PE / COPD)

How to remember

- **CRR**



- Non pulsatile elevated JVP seen in cardiac tamponade and SVC thrombosis
(Only 2 medical conditions "non pulsatile elevated JVP")

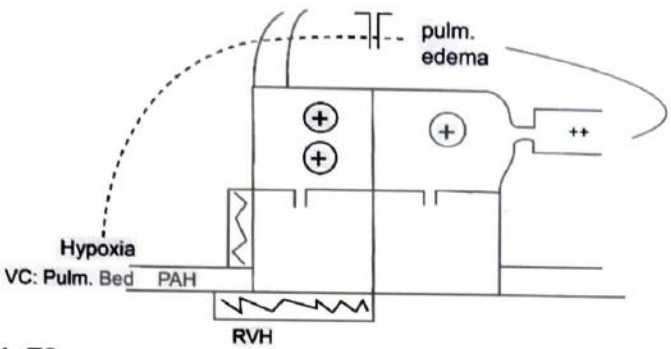
JVP WAVES

00:33:05

A: Atrial systole
C: IVC, bulge
V: atrial filling, peak @ S_2
X: atrial relaxation
Y: IVR

LARGE 'A' WAVE IN JVP

00:47:55



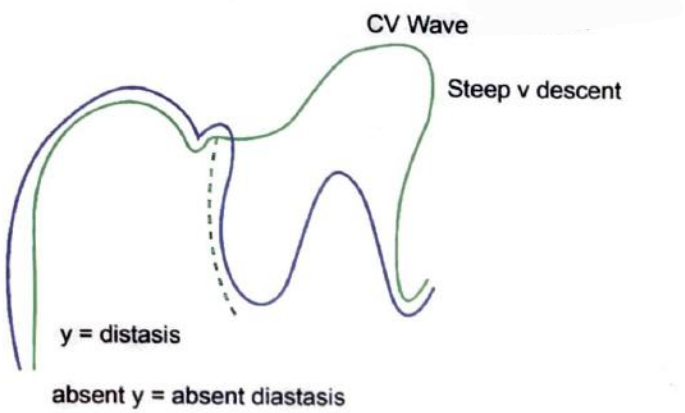
1. TS
2. PS
3. MS (Long standing)
4. PAH
   - Scleroderma
   - Fenfluramines
   - Eisenmenger syndrome/ complex
5. T.O.F (Subpulmonic stenosis)
6. Ebstein Anomaly

Absent 'a' wave → Atrial fibrillation (Twitching)

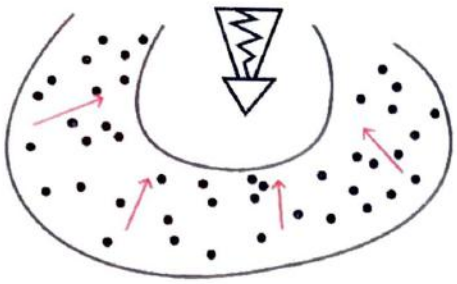
GIANT 'A' / CANON 'A' WAVE

00:53:30

A:V Dissociation
1. V. tachycardia Complete
2. C. Heart block (3rd degree H. block)
3. Junctional Tachycardia



Absent X Descent → Tricuspid regurgitation
Absent Y descent → Absent diastole
cardiac tamponade

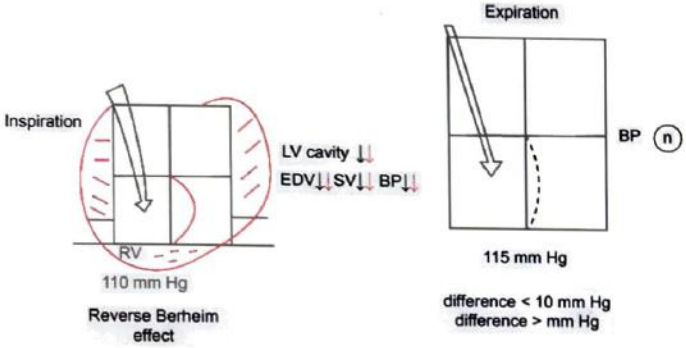


Cardiac tamponade

CARDIAC TAMPONADE

01:00:00

- Filling ↓↓
- SV ↓↓
- CO ↓↓
- BP ↓↓ : Obstructive shock



- P = Pulsus paradoxus (pulse disappearing during inspiration)
- BP = ↓↓
- S_1S_2 = muffled
- JVP = absent y descent

Important Information

- Pulses Paradoxus absent in low pressure cardiac tamponade

REVERSE BERHEIM EFFECT

01:08:00

- Normal difference in SBP is <10mm Hg between inspiration and expiration
- Difference > 12 mm Hg in SBP during Inspiration called as Pulsus Paradoxsus

JVP: QUICK SUMMARY

01:15:45

A wave

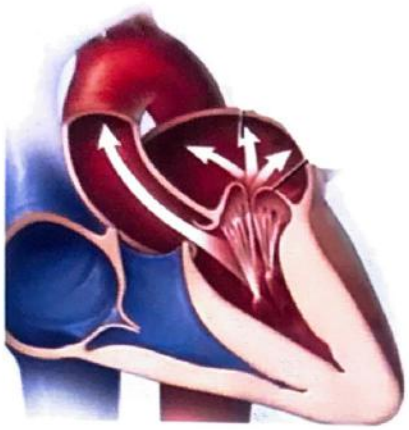
- Absent x = TR
- CV, steep y = TR
- Absent y : C. tamponade
- Steep x, absent y : C. Tamponade
- Kussmaul sign : CP, RCM, RVF
- Steep x, Steep y : C. Pericarditis

6 MITRAL VALVE PROLAPSE

MITRAL VALVE PROLAPSE (MVP) 00:00:16

Also called Floppy valve syndrome or Barlow syndrome

- Valve will be bulging in upward direction



- Problem of this condition will be many folds
- Once the valve will be bulge much higher than normal
- There is possibility that between these defective valve leaflets, might be leakage of blood from LV to LA
- That will contribute to development of a murmur called late systolic murmur

Highlight of disorder

- Lacks or loose Chordae tendineae
  - These lacks or loose are going to be supported by papillary muscles
  - The papillary muscles have to do extra work to keep infrastructure intact to prevent leakage of all the LV cavity blood going into LA
- This patient
  - There is bulge
  - Valve leaflets gets separated, variable amount of blood leaking
- In the later stages of the same disease dilation of mitral valve annulus
- This can set converted into frank mitral regurgitation

Important Information

- MCC of MR is Mitral valve prolapse

CAUSES 00:02:55

1. Myxomatous degeneration of MV apparatus
   - Defect in type 3 collagen
2. Marfan syndrome
   - Ch15
   - Fibrilin -1 protein defect
3. Ehter Danlos Syndrome
   - Hyper extended joints
4. Osteogenesis imperfecta
5. Antero posterior diameter decrease
   - Thoracic kyphosis ↓se: pancaking of the heart (heart is compressed between the sternum and the vertebra)
   - Called straight back syndrome

Important Information

- Straight back syndrome – space available for the heart between the sternum and vertebra is substantially reduced. AP diameter becomes lesser

Important Information

- BARREL CHEST in Emphysena where the AP diameter is symmetrically or disproportionally increased as compare to normal individual

6. Rheumatic fever
   - Aschoff nodules (junction at the caudate and dentate), can set damaged
   - Subacute bacterial endocarditis (SABE), infected damage to caudate and denate
   - Dilated cardiomyopathy- mitral annulus will be dilated
7. Ostium Secondum ASD
   - If patient having loose caudate & dentate, papillary muscle will be working more than normal
   - Long term
   - Presents component of subendocardial ischemia
   - Which causes substermal chest pain and palpitations

DIAGNOSIS 00:11:10

Echocardiography

Pathophysiology

1. Defective Coaptation of mitral valve leaflets
- Valves are bulging up, cause chordae kindneal to stretch, due to sudden tension, develops in the loose chordae tendineal
- Creates mid systolic clicks
2. Lacks chordae tendineae
- Stress on papillary structure
- Sudden cardia arrest can increase

CLINICAL FEATURES

00:13:33

1. Female 15-30 years
2. Males 50 years
3. Severely is more in men as compared to women
4. Asymptomatic initially – MC
- No leakage of blood from one LV to CL
5. Palpitations (PVC)

> **Important Information**
>
> - **Common rhythm disorder m ECG of MVP patients is PVC (premature ventilator contraction)**

6. Syncopal attacks
7. Frank ventricular tachycardia (premature ventricular contractions)

8. Structural damage to LA (atrial fibrillation)

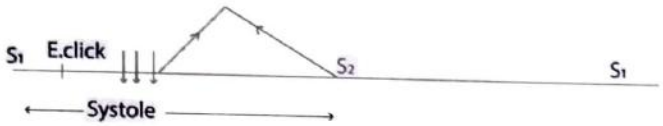
A. FIB

9. Chest pain without exertion
10. TIA events
- Valvular endothelium damage
  - Miniature platelet plugs on the valve
11. Infective endocarditis increase risk
12. Sudden cardiac death risk increase
13. Mitral regurgitation acute
- Pulmonary edema

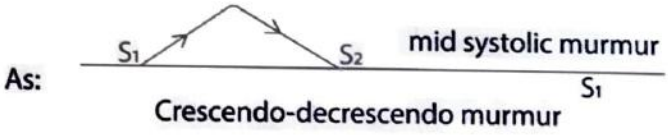
EXAMINATION FINDING

00:20:30

1. Mid systolic clicks
- Tension in slack chordae tendinae

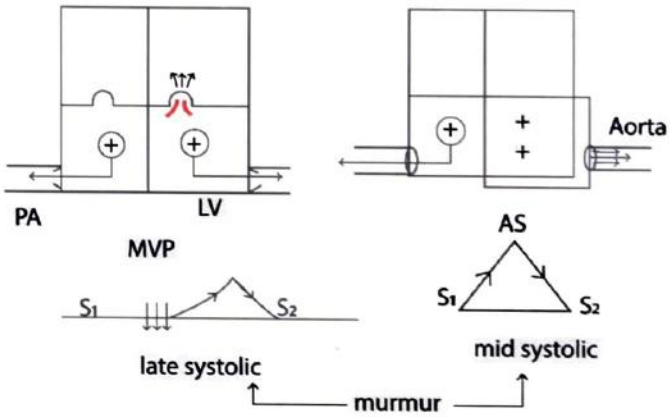


2. Late systolic murmur
or
Late systolic crescendo-decresendo murmur



1. Murmur stants after mid systolic clicks
2. Late systolic

1. Turbulence will be felt during the phase of systole
2. Mid systolic

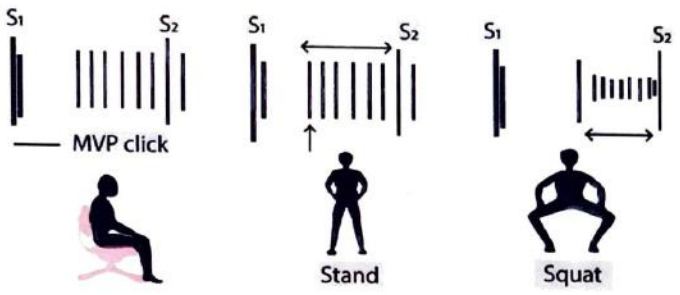


3. Post leaflet → Ant leaflet
↓ ↓
Jet of blood move ant. Jet of blood more post
↓ ↓
Murmur radiation to base of heart Murmur radiation to axilla

3. Position
- Stand
  - Duration of murmur increases
- Sqnat

- Duration of murmur decrease

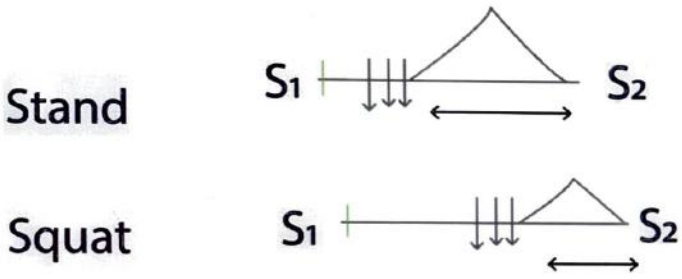
- Prolapse α $\frac{1}{\text{volume of blood in ventricle cavity}}$
  - Stand VR ↓→ cavity size ↓ prolapse ↑→ mid systolic click early



- Squat VR ↑ : cavity size ↑ : prolapse ↓ : mid systolic click late

Important Information

All murmurs ↓ se with valsana & standing except
- HOCM: louder
- MVP: longer



INVESTIGATION OF CHOICE

00:37:06

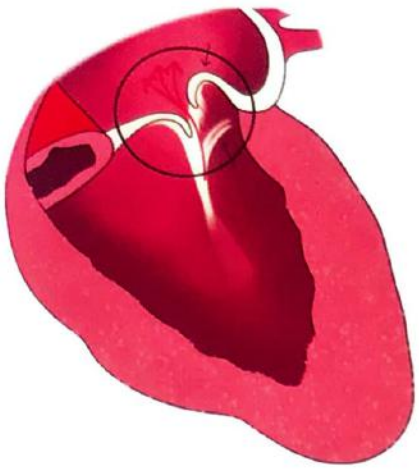
1. TTE (Transthoracic echocardiography)
   - Bulge >2mm above mitral annulus
2. ECG
   - T wave inversion (head II,III,aVF)
   - PVC: MC rhythm abnormally
3. Cardiac MRI
4. Invasive left ventriculography

TREATMENT

00:40:14

1. Palpitation / chest pain
   - -Blockers
2. A.Fib: $CHADS_2$ VASC
   - Oral anticoagulation
3. TIA
   - Low dose aspirin
4. MV repair > replacement
5. Trans catheter repair
6. Prophalaxis on infected endocarditis
   - If previous episode documented

7 MITRAL REGURGITATION



- MR is due to damage of chordae tendinae
- Mitral valve prolapse is mostly associated with MR
- Defective coaptation of valves causes regurgitation of blood to L atria and cause LA enlargement
  - Murmurs of acute and chronic MR are diff.
  - Acute MR causes sudden exposure of large amount of blood
- LA leading to left sided heart failure and requires Sx repair whereas in chronic MR it gets adjusted to it, so presentation of both may vary
  - Whereas the MS causes right ventricular hypertrophy as a sequalae
- Characteristic - systolic murmur

CAUSES OF ACUTE MITRAL REGURGITATION

00:01:00

1. Infective endocarditis
2. Papillary muscle damage due to MI
3. M/C involved posteromedial leaflet (>>anterolateral leaflet) because of single blood supply
4. MVP / Flail leaflet (Myxomatous degeneration)
5. Blunt trauma to chest : Commotio cordis (Due to V. fibrillation) causing "sudden cardiac death"

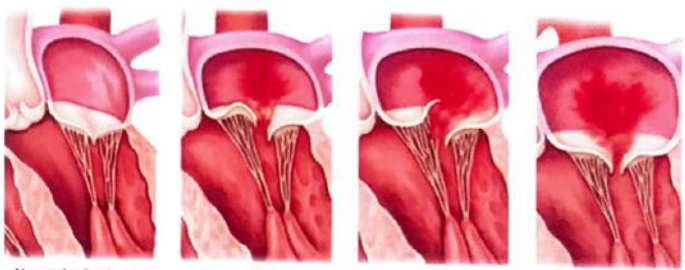
CAUSES OF CHRONIC MITRAL REGURGITATION

00:05:20

1. MVP
2. Rheumatic: Scarring of leaflets valves
3. SABE
4. Congenital cause: Cleft mitral valve due to ostium primum ASD

Chronic MR

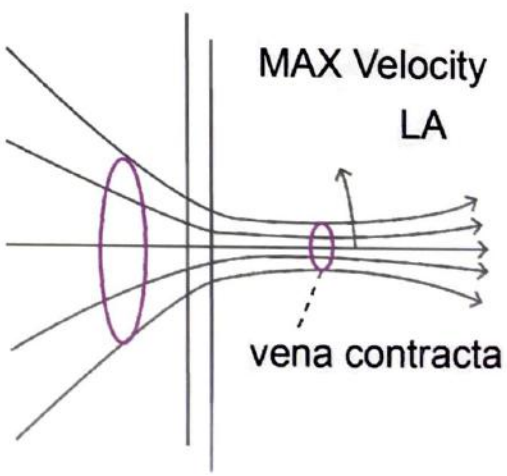
00:08:09



- **Primary**: Intrinsic damage to valve- IE, MVP, radiation, o. primum
- **Secondary**: DCM, ischemic cardiomyopathy, HOCM (Systolic anterior displacement of mitral valve)
  - In chronic cases, atrial fibrillation and left atrial enlargement are seen
- **M/C characteristic finding occurring in secondary varieties**: Mitral annular dilatation

Important Information

- Mitral annulus calcification can result in both (MS & MR)
- SLE & RA associated with mitral stenosis
- Ankylosing spondylitis (HLA B27), syphilis – Aortic regurgitation



- MR severity is directly proportional to vena contracta
- Vena contracta – narrowest part with highest velocity of regurgitant jet of blood from LV to LA

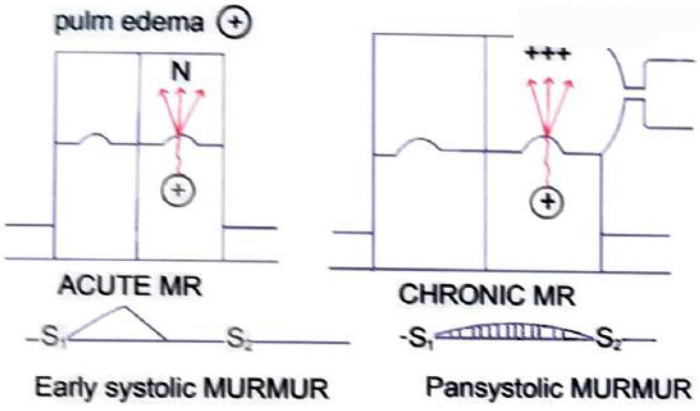
CRITERIA FOR SEVERE MR

00:12:25

1. Vena contracta: >0.7 cm2
2. Regurgitation volume: >60 ml / beat
3. Regurgitation fraction: >50%
4. Regurgitation orifice: ->0.4 cm2

- **Severe MR is treated by** - Mitral valve repair (MVRp)– mostly preferred & superior over mitral valve replacement (MVR)

Acute MR	Chronic MR
Heart size Normal	Large
LA-Normal	Enlarged
Pulmonary Edema +	Pulmonary Edema ±
Early systolic murmur	Pansystolic murmur



Important Information

- Late Systolic Murmur: MVP
- Ejection systolic murmur: Aortic stenosis (Crescendo-decrescendo Murmur)

C/F

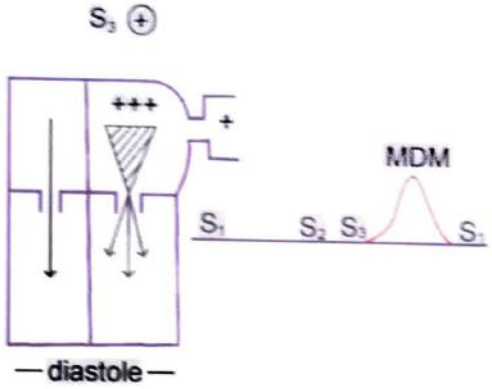
00:15:55

- Mild – moderate MR is well tolerated
- Fatigue, PND (Paroxysmal nocturnal dyspnoea), orthopnoea, hepatomegaly, ankle edema, ↑↑ JVP, TR
- Palpitation – is caused due to AF, and longstanding AF results in formation of clots/Emboli (If clots/emboli goes to brain → can lead to stroke and may prove to be fatal)

O/E

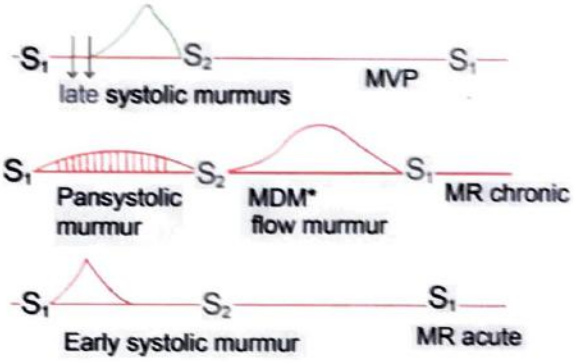
00:17:00

- BP- Normal
- JVP ↑
- Hepatomegaly
- Thrill + at apex on palpation
- Hyperdynamic character at apex
- Displacement of apex



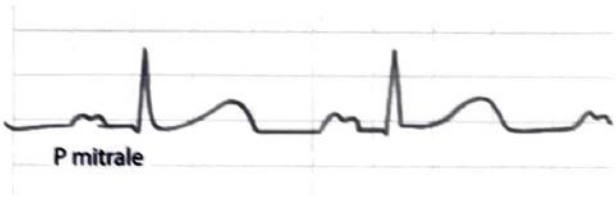
Auscultation

- S_1 – Soft
- S_2 – $A_2 P_2$ (A_2 appears early)
  - Wide variable split S_2
- S_3 – present, diastolic murmur heard – MDM



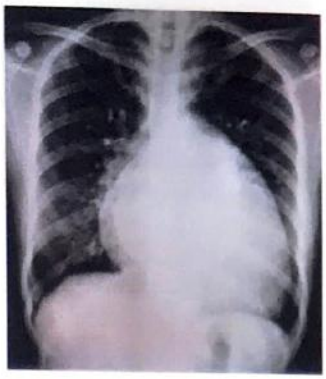
WORK UP

00:29:20



1. **ECG**

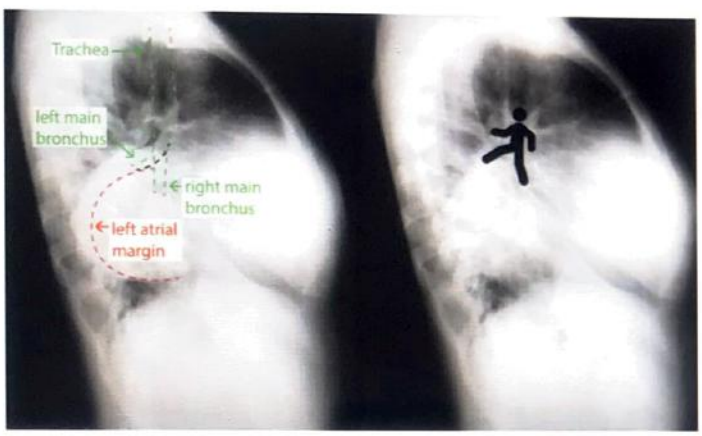
- P- Mitrale:>120 msec
  - Notch in P-wave
- Left axis deviation
- P- Pulmonale : vertical height is > 2.5 mm – RA enlargement
- Variable amplitude of p wave – multifocal atrial tachyarrhythmias



2. **CXR**

- Cardiothoracic ratio increased – cardiomegaly (>0.5- adults, >0.6- children)
- LA enlargement leads to pushing of rt. atria leading to double atrial shadow
- Mild pulmonary congestion with perihilar shadow
- Pulmonary edema – Kerley B line
- Walking man sign on lateral view (Enlarged lt. atria causing enlargement of carinal angle)

3. **IOC:** ECHO



Rx of Acute MR 00:32:19

1. Furosemide
2. Sodium Nitroprusside (Used in acute onset sudden decompensation that leads to LVF to prevent sudden stress on heart)

Rx of Chronic MR 00:33:41

Mitral valve repair (MVRp)

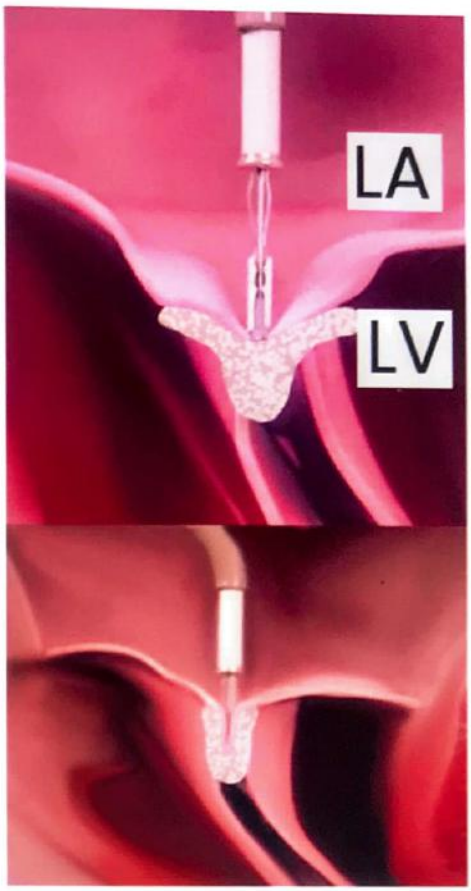
1. Annuloplasty ring
2. Neochords (New chordae tendinae)
3. Chord transfer

(Mitral valve repair >> mitral valve replacement, as it requires lifelong antiplatelet therapy, which can precipitate warfarin toxicity)

INDICATION OF MITRAL VALVE REPAIR: (MVRP)

1. Symptomatic severe chronic MR
2. In asymptomatic chronic MR, done only if
   a. LVEF < 60%
   b. LVESD > 40 mm

NEW APPROACH:

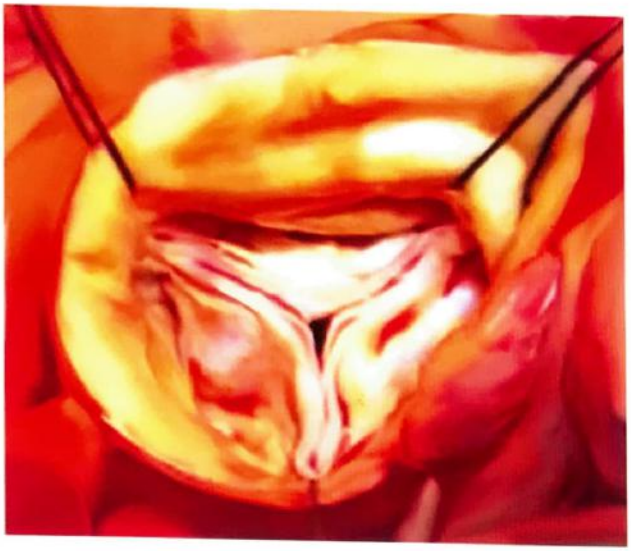


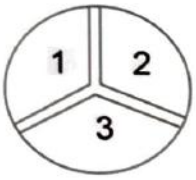
Trans catheter mitral valve

8 AORTIC STENOSIS

OVERALL M/C CONGENITAL HEART VALVULAR LESION

- Most common congenital heart disease → Peri membranous variety of VSD





N

DAMAGED CUSPS OF AORTIC VALVE

Normal aortic valve (tricuspid) (Cross-sectional view)

- Damage of valves can be due to various causes such as
  - Senile Calcification
  - Scarring due to RHD
- Damaged valve can't open & close properly thus leading to aortic stenosis and aortic regurgitation respectively (i.e. defect in valve opening leads to AS & defect in valve closure leads to AR)

ETIOLOGY OF AS

00:02:10

1. **Pediatric presentation:** Bicuspid Aortic valve (M/C CVS lesion in Turner syndrome)/ Unicuspid aortic valve
   - Unicuspid Aortic valve causes more severe disease as compared to bicuspid variety as the ability of valve to allow left ventricular blood to aorta is severely hampered
2. **Old age/ senile presentation**
   - Degenerative calcification (Associated with atherosclerosis– it leads to involvement of abdominal aorta/root of aorta and the area around the aortic valve - Fibro calcification of aortic valve
   - As the disease progresses, there is differentiation of Fibroblasts / Myofibroblast into Osteoblasts, leading to deposition of bone matrix in valve → Calcification of valve
   - Balloon dilatation & balloon valvoplasty is C/I in these patients with calcified valves as balloon dilatation would lead the calcified valve cusps to break into small fragments that may get lodged into brain resulting in development of stroke in the patients
3. **Rheumatic Fever**→ causes commissural fusion & scarring of valve cusps
   - [Normal aortic valve surface area → 2.5 – 3 cm^2 & when the surface area of aortic valve is <1 cm^2 leads to severe AS]
4. **Radiation**
   - **Mediastinal Radiation** (Radiotherapy) given to a patient of Hodgkin's lymphoma for hilar lymphadenopathy (or) chest radiotherapy given to a patient of breast cancer
5. **Shone complex** → Associated with Pediatric AS

Components of Shone complex

a. Parachute mitral valve
b. Supra valvular mitral membrane
c. Subvalvular aortic stenosis
d. Coarctation of aorta

Important Information

- Common heart lesion seen in patient of Turner syndrome → Bicuspid aortic valve (MC)> Coarctation of aorta
- HOCM is associated with SAM (Systolic Anterior Displacement of Mitral Valve)

CLINICAL FEATURES OF AS

00:08:50

- Male : Female = 2-4 : 1
- **Bicuspid aortic valve** has AD pattern of inheritance
- Gene involved → NOTCH 1 gene
- In old age/ geriatric population → Hypertension may lead to AS

Symptoms

- Triad of AS
  - **S - S**yncope (On exertion)
  - **A - A**ngina (Secondary to LVH)
  - **D - D**yspnea (Secondary to ↑ LV end diastolic pressure)

How to remember

- SAD

- LV End Diastolic Pressure (LVEDP) increases as a result of hypertrophy of LV
- Leads to increased left atrial pressure
- Leads to congestion in lungs & pulmonary edema
- Leads to dyspnoea

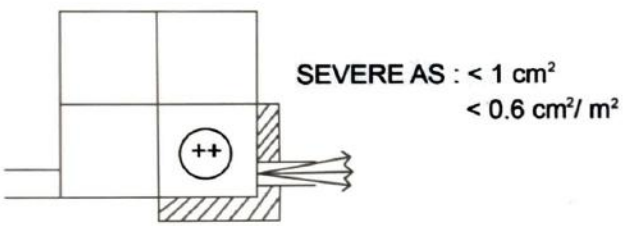
ON EXAMINATION

00:19:45

1. Pulsus parvus et tardus - Slow rise pulse with delayed peak
2. Anacrotic shudder/carotid thrill → due to ↑ in ejection velocity

In severe AS

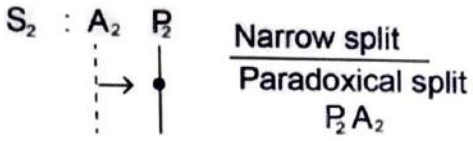
3. **Aortic valve area** < 1 cm2
- < 0.6 cm2/ m2 BSA (Body Surface Area)



- Left ventricle has very high pressure. When the blood from LV passes via stenosed aortic valve, there is very much increased turbulence in blood flow.
- Volume of blood going from LV to aorta is decreased but velocity of blood flow is increased

4. Apex Beat
- Is always displaced
- Double apical impulse (Secondary to hypertrophy of LV)
  Character → Heaving (i.e. impulse hits your finger & lifts it up)
- Displacement → To left side
- Felt in both 5th& 6th left intercostal space (ICS) as it is double apical impulse

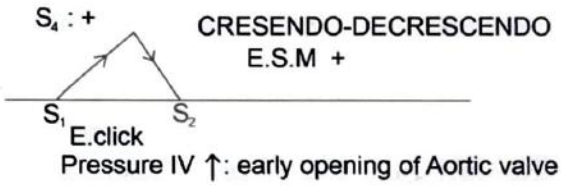
5. S2: Narrow split S_2 (Narrow as gap b/w A_2 & P_2 is < 30 millisecond)
- Paradoxical split i.e. P_2 comes before A_2 [Means pulmonary valve has closed on time but aortic valve is closing late i.e. after the pulmonary valve]



6. S_4: Present
- As patient of AS also have compensatory left atrial hypertrophy
- Left atria generate more pressure to propel blood into left ventricle
- S_4 is produced

[If this patient of AS goes to heart failure then S_3 would also be anterior. However, initial presentation is S_4]

7. Murmur : Ejection systolic murmur
- AKA crescendo – decrescendo murmur



8. The murmur of AS radiates to Carotid artery known as Carotid Thrill
9. Vibrations of valvular cusps when blood passes from aortic annulus leads to murmur to radiate into Axilla (K/A Gallaverdin Phenomenon)
- This radiation into axilla can lead to confuse aortic stenosis with mitral regurgitation as murmur of mitral regurgitation also radiates to axilla

WORK-UP

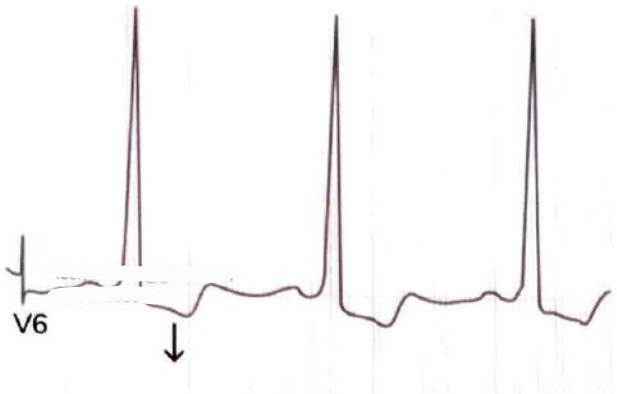
00:29:33

1. ECG

- Very deep S waves in lead V_1
- Very Tall R waves in lead V_5
- [(SV1 + RV5/6)] > 35 mm is the criteria to call it Left Ventricular Hypertrophy

Refer Image 8.1

LV strain pattern: T wave inversion seen in V5 & V6



1. **CXR**
- Shows cardiomegaly
- Cardio – Thoracic ratio in increased (↑ CT ratio)

2. **ECHO**
- Used to see valvular morphology

Valve size	Grade Of AS
• < 1.5 – 2 cm^2	• Mild AS
• < 1.0-1.5 cm^2	• Moderate AS
• < 1.0 cm^2	• Severe AS

3. **IOC** to see any valvular calcification → CT chest
- Coronary Angiography should also be done in patients of aortic stenosis as most of patients are of old age/ geriatric population & atherosclerosis is very common in these old age patients
- Treadmill test and dobutamine stress ECHO are not done in patients with severe aortic stenosis

DEATH IN PATIENTS OF AORTIC STENOSIS

00:35:30

- If syncope/ angina is present as a symptom → death within 3 years
- If dyspnea is present as a symptom → death within 2 years
- If Congestive Heart Failure (CHF) Present → death in 1.5-2 years

Important Information

- Highest chances of death seen in patients with Congestive Heart Failure (CHF)

TREATMENT OF VALVULAR AORTIC STENOSIS

00:37:45

- **Treatment is stage based**

Stages of AS

Stage A
- Risk factors present
- No narrowing of valve

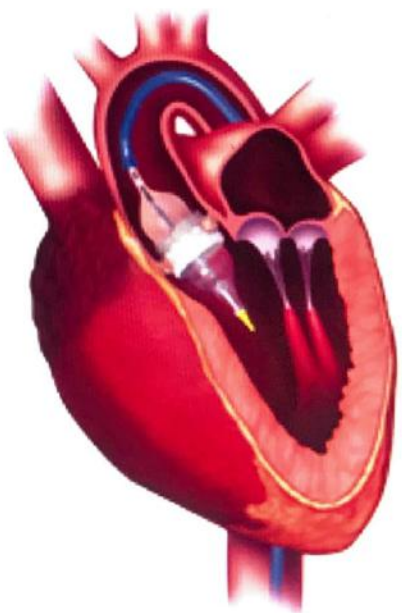
Stage B
- Progressive narrowing of valve → (i.e. mild to moderate aortic stenosis)

Stage C
- Severe aortic stenosis but patient is asymptomatic

Stage D
- Severe aortic stenosis & patient is symptomatic

Severe aortic stenosis

1. **Transvalvular pressure gradient of** > 40 mmHg
- Transvalvular pressure gradient (ΩP)
- LV – Aorta
- Pressure difference b/w left ventricle & aorta
2. **Ejection velocity:** >4m/sec [i.e. 4 times of Normal]
- [Normal Ejection velocity → 1m/sec]
3. **Size of orifice** : < 1 cm^2 / < 0.6 cm^2/m^2 BSA
- (BSA: Body surface area)



Treatment For stage D: Trans catheter aortic valve replacement (TAVR)

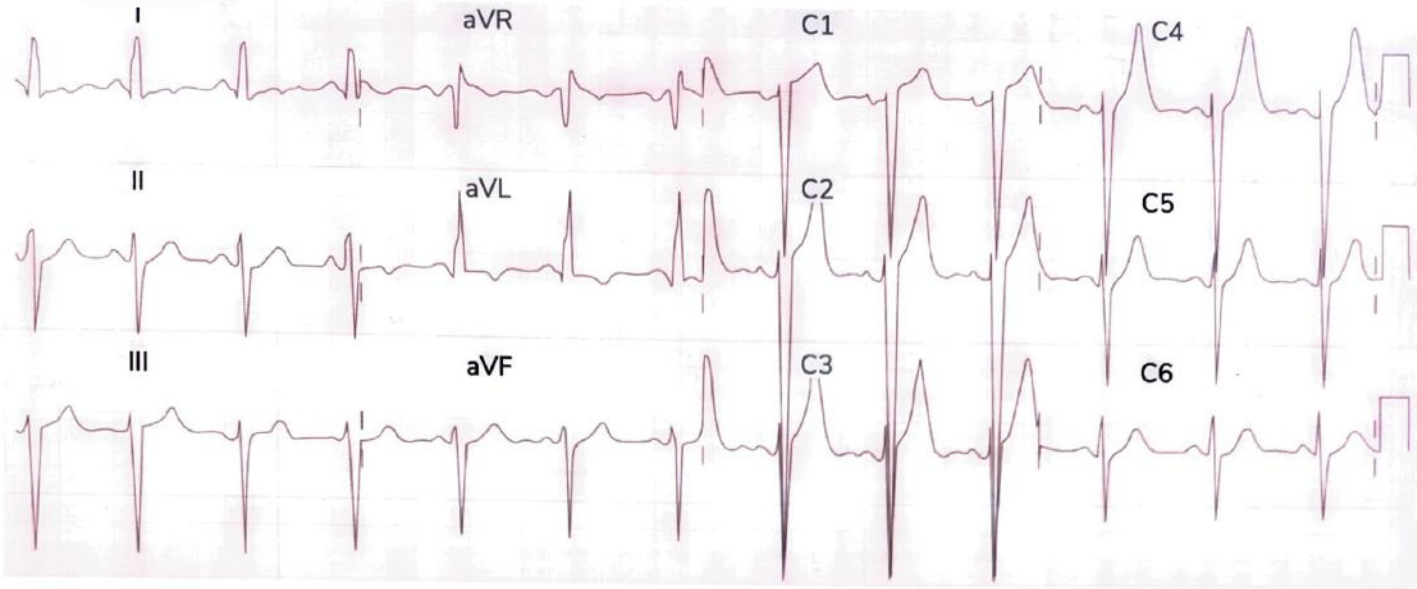
For stage C

- If patient has concomitant risk factors like
  - Concomitant Triple Vessel Disease (TVD)
  - Left ventricular ejection fraction < 50%
- In these conditions, treatment is again Trans catheter aortic valve replacement (TAVR)

Important Information

- Statins are given to prevent further progression of Atherosclerosis & thus Triple Vessel Disease
- Statins doesn't prevent further progression of calcification

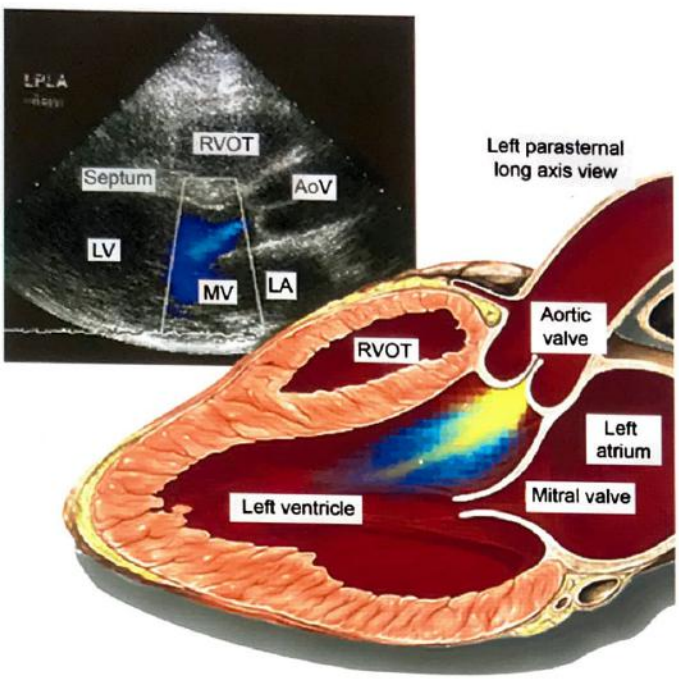
Image 8.1



9 AORTIC REGURGITATION

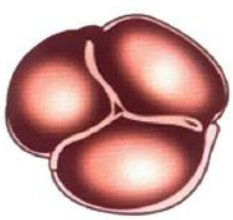
00:00:18

- Valvular lesion of aortic valve
- Incompetent valve
- Turbulence created
- Diastolic murmur (Decrescendo)



AR : Left ventricle pressure ↑↑

Normal aortic valve



Bicuspid aortic valve

- Blood hit the anterior leaflet of mitral valve
- Vibrations produced
- Murmur
- Austin Flint murmur

Murmurs of AR

a. Diastolic murmur
b. Austin Flint murmur

CAUSES OF AR

00:01:38

1. Bicuspid aortic valve: Defective coaptation
2. Infective endocarditis / SABE : Perforation
3. RHD (Rheumatic Heart Disease)
4. Myxomatous degeneration of valve
5. Syphilis
6. Ankylosing spondylitis
7. Aortic root dilation

- Aortitis Takayasu
  - Aortic dissection
  - HTN
  - Cystic medial necrosis
  - Marfan syndrome
- **Severe AR** : When regurgitant volume > 60 mL/beat
- **Progressive AR** : LVEF < 50%
- (Left Ventricle End systolic dimension) LVESD > 50 mm
- (Left Ventricle End diastolic dimension) LVEDD > 65 mm
- In AR, both Pre-load & After-load are ↑sed
  - ↑sed Oxygen consumption

Refer Table 9.1

Angina

- Mismatch b/w demand & supply : Angina
- Eccentric hypertrophy
  - ↓ Demand ↑↑
  - O_2 consumption ↑↑

CLINICAL FEATURES

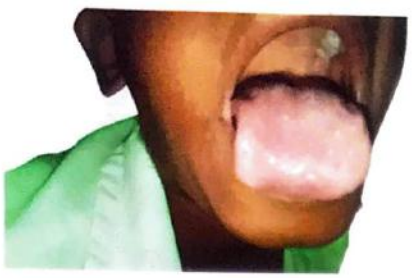
00:18:42

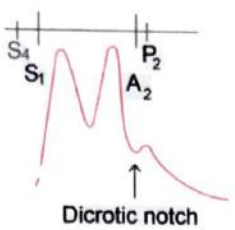
1. Uncomfortable sensation in supine position
2. Head pounding sensation
3. Development of exertional dyspnea
4. Paroxysmal nocturnal dyspnea
5. Orthopnea / diaphoresis
6. Angina – May be present at rest or on exertion

Sx: should be done within 1 year of onset of symptom for faster recovery

EXAMINATION FINDINGS

00:22:15





1. **De-Musset Sign:** Head bobbing to and fro
2. **Muller sign:** Pulsation of uvula
3. **Marfan's** – Arachnodactyly (Thumb Sign)
- Ankylosing spondylitis → Schober's test
4. **Corrigan's pulse** / Water hammer pulse/Collapsing pulse
- According to Frank Starling law, as end diastolic volume of LV increases, the force of contraction of LV increases.

Seen on arm elevation above level of head

5. **Quincke's sign**: Flushing and blanching at distal part of nail
6. **Traube Sign**: Pistol shot sounds heard at femoral artery
7. **Duroziez Sign**: Gently press the femoral artery using stethoscope
- Murmur in heart d/t turbulence
- To and fro murmur

Important Information

- Mill wheel murmur - heard in case of Air embolism

8. **Stroke Volume ↑↑**

a. Frank Starling Law

b. Eccentric Hypertrophy

- SBP ↑↑, DBP ↓↓, Pulse Pressure ↑↑

Low DBP values are seen in

a. AR

b. A-V malformation

c. Aortic Dissection

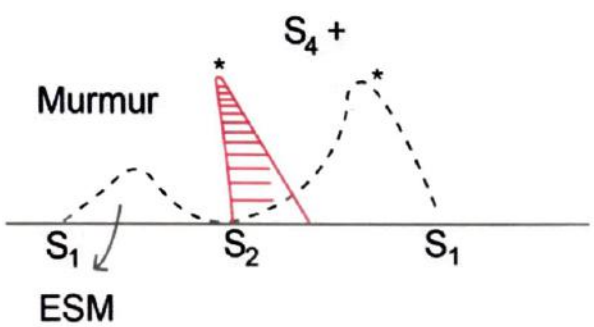
9. BP difference between UL (Upper limb) and lower limb < 20 mm of Hg (N)
- BP difference > 20 mm of Hg is abnormal and is called as Hill sign
- In coarctation of aorta, BP of UL is ↑ & BP of LL is ↓

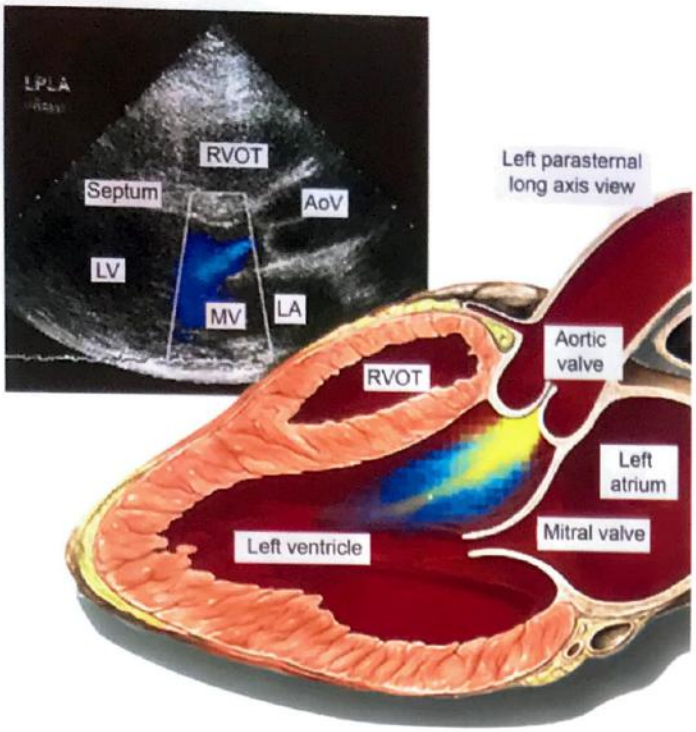
10. Apex beat → Heaving (Palpation)
11. Thrill (Auscultation) → diastolic → left lower sternal border Systolic → Suprasternal notch.
- (AR + AS)

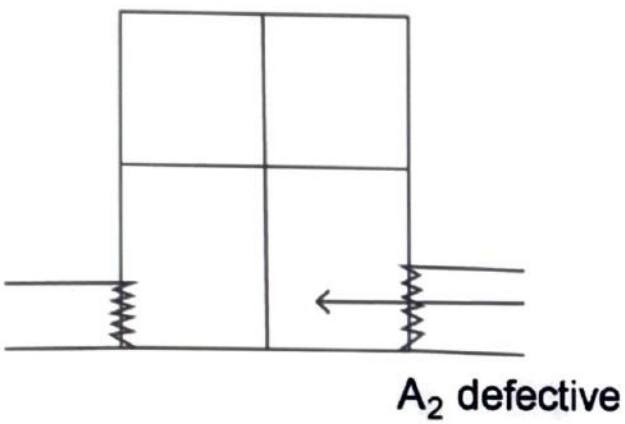
12. S_2: soft/ single $S_2 = A_2$ not heard
- Ejection click is present if bicuspid aortic valve is present

13. S_4: present: Left atrial enlargement

14. Murmur





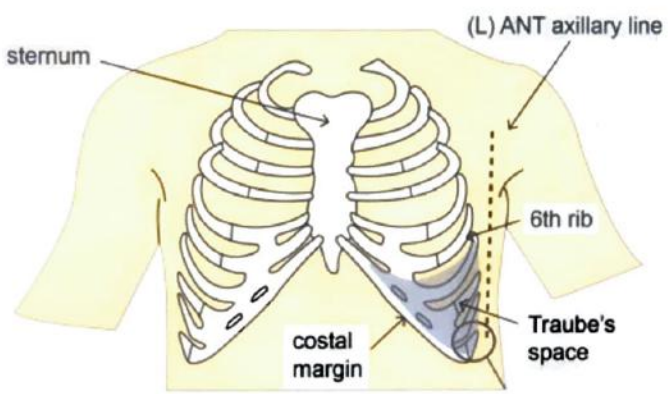


High pitched Decrescendo murmur (Seagull murmur)

- Austin Flint murmur → Mid Diastolic Murmur (MDM)

MURMURS OF AR

00:36:55



1. Early Diastolic murmur: Main murmur
2. Mid Diastolic murmur: Austin Flint murmur (RHD)
3. Ejection systolic murmur: If AS is co-existent
4. **Gerhardt's sign**: Pulsation in spleen
5. **Rosenbach sign**: Pulsation in liver

I. TS (Tricuspid stenosis)
II. TR (Tricuspid regurgitation)
III. AR (Aortic regurgitation)

Important Information

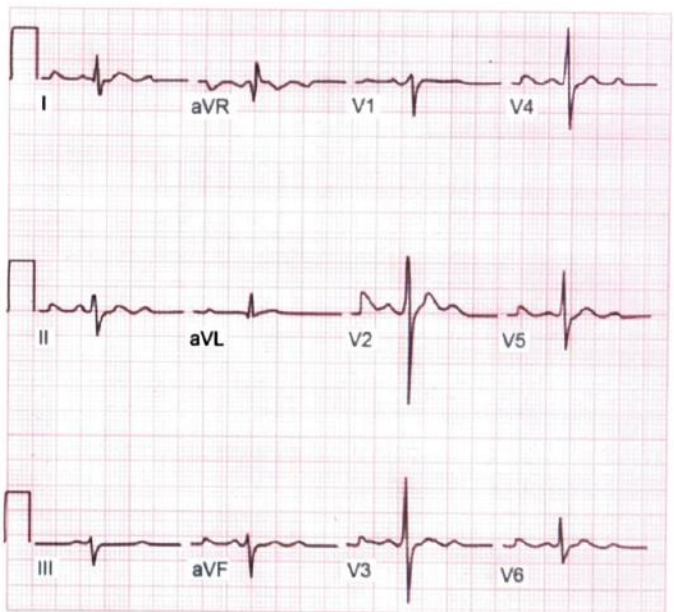
- **Rosenbach sign of eye**
  - **Seen in Grave's disease [Tremor of eyelid]**

6. **Landolphi sign**: Pulsations in pupil

WORK UP

00:43:50

1. **ECG**– 1st line of investigation



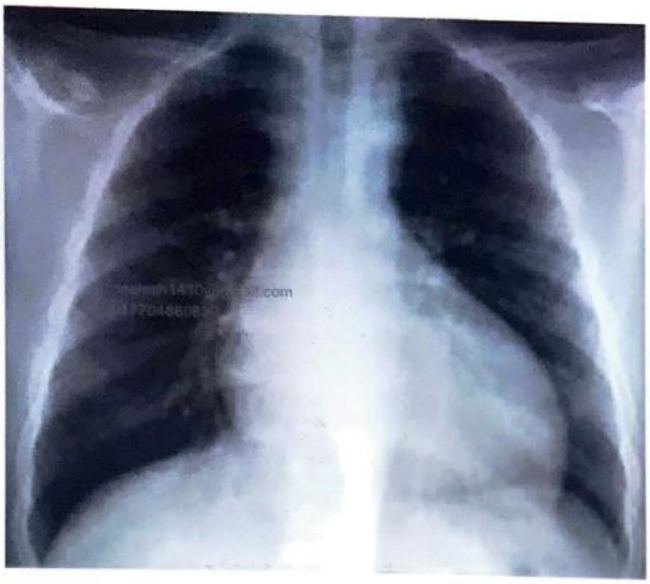
2. **Echocardiography**: IOC

- **Severe AR**
  - **Criteria**

a. Width regurgitant jet > 65% LVOT
b. Regurgitant volume > 60/beat
c. Regurgitant Fraction > 50%

3. **CXR**: LV enlargement + +

- Apex: Inferolateral



4. **Coronary Angiography**: visualization of coronary arteries

TREATMENT

00:45:20

Acute AR

i. Loop diuretics (IV furosemide)
ii. Vasodilator – Na Nitroprusside
iii. Surgical management within < 24 hours

CHRONIC AR

00:47:35

i. Diuretics
ii. ACE inhibitors
iii. CCB

- Keep SBP < 140 mm of Hg
- ↑↑ Stroke volume

a. Frank Starling law
b. Eccentric
   - BP = CO x PR
   - CO = HR x SV

iv. Nitrates: To treat angina
v. Penicillin Benzathine → 2.4 MU IM x 3 shots @ 1 week gap

- For syphilis

Rx: Penicillin G → IV for neuro syphilis x 2 weeks

Important Information

- β-blockers → absolutely contraindicated in acute AR
  - β-blockers can be given in chronic AR
- IABP – Intra-Aortic Balloon pump
  - Contraindicated worsens AR

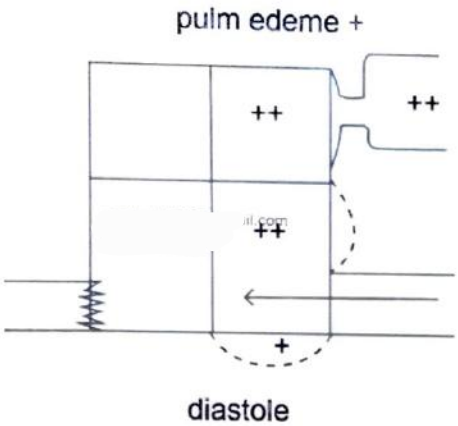
TOC: Aortic valve replacement → Symptomatic

INDICATIONS OF AORTIC VALVE REPLACEMENT

00:50:32

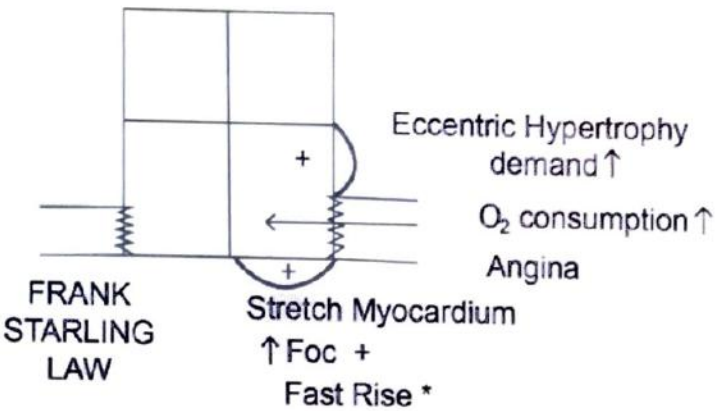
- Symptomatic severe AR
- Asymptomatic severe AR
- Symptomatic moderate AR
  - Surgery should be done with in 1 year

Table 9.1



Acute presentation

(Infective Endocarditis)

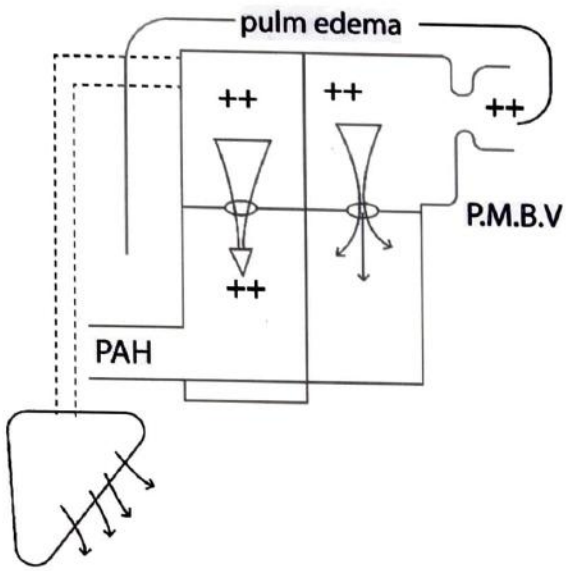


Chronic presentation

Pulse → Corrigan's pulse / water hammer pulse

10 TRICUSPID STENOSIS

- Tricuspid stenosis doesn't occur alone but occurs with Mitral Stenosis 00:00:20
  - In given diagram, both MS and TS diagram are narrowed. So, the amount of blood from RA to RV and from LA to LV is reduced. Mitral stenosis contribute to left Atrial congestion.
  - Most patients would have structural damage to LA & may cause Atrial Fibrillation
  - Dilated Left atria transmits pressure to pulmonary veins
  - Pressure to lungs



- C/O Dyspnea on exertion & orthopnea (d/t pulmonary edema)
  - Pulmonary Artery HTN

- Development of RVH
  - In MS defect on left but hypertrophy seen with Rt. Ventricle
  - Tricuspid stenosis prevent the transmission of pressure changes in backward direction because the orifice is relatively narrow.
  - Thus the gradient between RA & RV will also change therefore, back pressure changes. The transmission of Mitral stenosis disease process to the right side
  - Mainly transmission of pressure changes to SVC & IVC will be lesser, as tricuspid stenosis is acting like barrier

Rx of severe type of MS

- By percutaneous mitral valve balloon valvotomy (PMBV)
- C/F of Pulmonary Congestion will disappear & now symptoms of Tricuspid will manifest & amount of blood from RA → RV will be lesser & there would be lot of congestion in RA
  - Will translate into congestion in SVC & IVC
- As pressure transmitted downward to liver these pt will start developing symptoms
- **In a patient of both MS and TS,** mostly the symptoms of MS will predominate

Initial symptoms

1. Dyspnoea On Exertion
2. Pulmonary oedema
3. Orthopnoea

- MS on ECHO and TS may not be picked up as it may not be severe enough. After treating if patient will still get features of right sided heart failure than its is due to TS

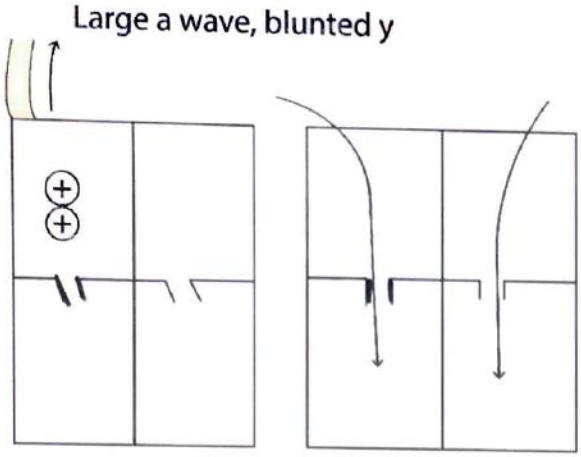
Clinical feature

- Hepatomegaly (Initially)
- Pedal edema and Ascites
- Cardiac cirrhosis (later), Caput Medusae Here problem lies in the right side of heart

O/E

00:04:50

1. **On ECG:** large 'a' wave (d/t tricuspid stenosis, the RA of pt has to generate more force to push blood in forward direction)
   Blunted y descent (as stenosis filling in RV is relatively slower)
2. **Presystolic pulsation of liver** (C/F of TS)
   - Systolic pulsation of liver is seen in TR
3. **Murmur**
   - D/T narrow opening → when blood passes through RA → RV, creates turbulence
   - Mid diastolic murmur (like mitral stenosis)
   - Becomes louder on inspiration
   - Heard on left lower sternal border & radiates to tricuspid area
   - Best appreciated, when heard close to xiphoid process



Important Information

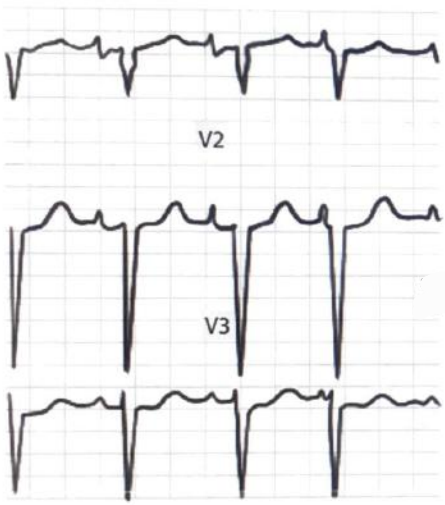
- All events on Rt side of heart become louder on inspiration

WORK UP

00:11:23

1. ECG with simultaneous Echocardiography: Normal

- Large P wave: P- pulmonale (Peaked p-wave)
  - (Limb leads >2.5cm)
  - (Chest leads >1.5cm)
- Rt axis deviation



2. CXR – RA

- More prominent, enlarged
- SVC
- Shadow of Azygous vein enlarged

3. To confirm: **ECHO** – Thickened valve. Severe if <1 cm^2 (Size of orifice)
- MS, severity if < 1.5 cm^2
- Time taken by blood to travel from RA → RV will increase (i.e. T > 190 msec.)

Rx

00:14:00

- Salt restricted diet
- Diuretics - Aldosterone antagonist
- Valve repair (Main treatment)
- Prosthetic valve – metallic or biological prosthetic valve (Disadvantage of Metallic valve – lifelong Anticoagulant therapy needed)

11 TRICUSPID REGURGITATION AND PULMONIC STENOSIS

TR (TRICUSPID REGURGITATION)

- **MCC of tricuspid regurgitation**: Functional due to tricuspid annulus dilation
- **Secondary causes**

1. Severe Pulmonary Arterial Hypertension (Severe PAH > 55mmHg)
   - Normal Pulmonary Arterial Pressure = 15mmHg
2. Right ventricular pacing
3. Carcinoid Syndrome
   - Tricuspid Insufficiency
   - Pulmonic Stenosis

How to remember

- TIPS

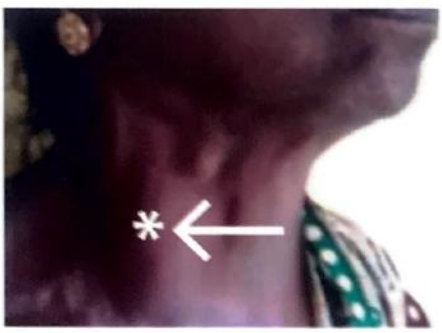
Important Information

- **Two vascular lesions are seen in Carcinoid Heart Disease**

4. Rheumatic Heart Disease (RHD)
5. Radiation exposure
6. Endomyocardial fibroelastosis
7. Infective Endocarditis
8. Ebstein anomaly (Wolf Parkinson White syndrome) - Due to lithium

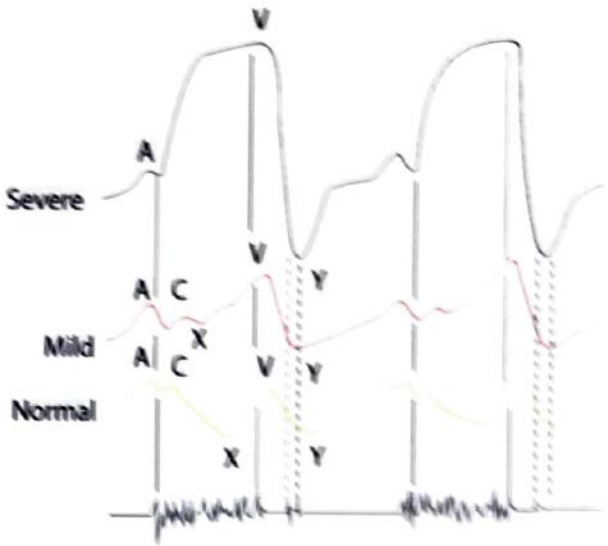
SYMPTOMS

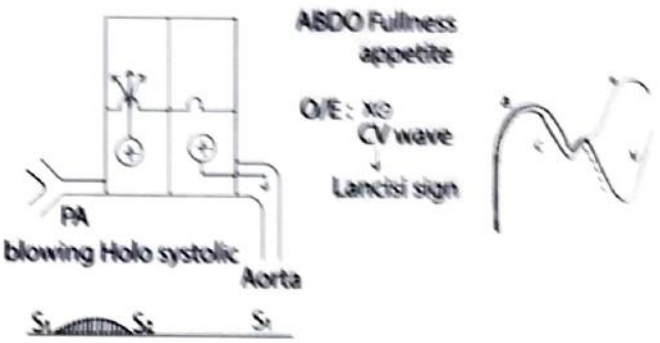
- Low cardiac output (Effort intolerance)
- Cervical pulsations
- Abdominal fullness
- Asthenia



ON EXAMINATION

- **JVP findings**
  - Absent x wave
  - CV wave (LANCISI SIGN)
  - Steep y descent
- Hepatomegaly
- Pulsatile liver
- Pansystolic murmur
- Right sided event increases during inspiration
  - Pan systolic murmur increases with inspiration (Caravallo Sign)



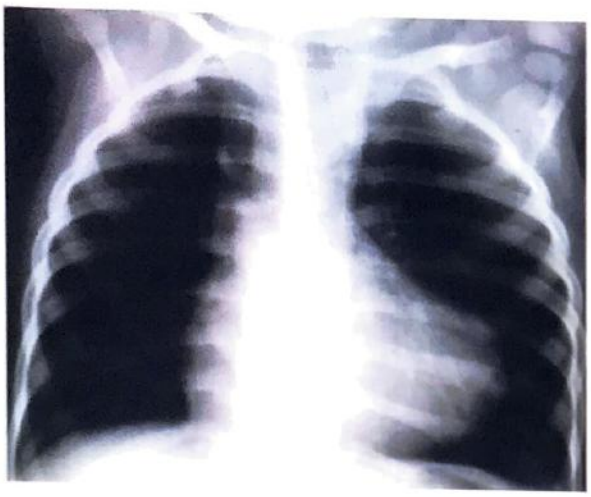


P.S.M: louder on inspiration : Carvallo Sign

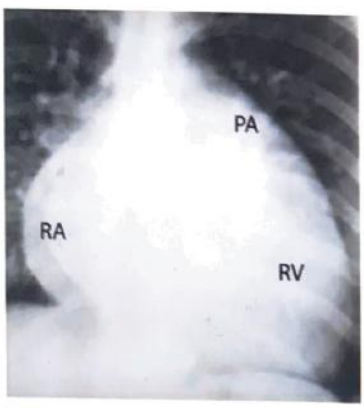
WORK UP OF THE PATIENT WITH TRICUSPID REGURGITATION

1. ECG
   - Right axis deviation
   - P – pulmonale

2. **Chest X-ray**: Cardiomegaly (CT ratio ↑)
- Pulmonary artery segment prominent
- Right atrial enlargement



3. **Echocardiograph**
- Blood flows from right ventricle to right atria.
- Reversed systolic flow in the hepatic vein



TREATMENT

⏱ 00:18:10

- Diuretics
- Surgery
  - Tricuspid valve repair
  - Tricuspid valve replacement

PULMONIC STENOSIS

⏱ 00:22:35

- Common in pediatric age group

Causes

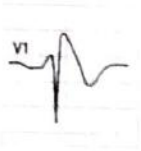
1. Congenital
2. Dysplastic pulmonary valve (Noonan Syndrome)

- Noonan Syndrome looks like Turner syndrome except Noonan is chromosome 12 defect and Turner is aneuploidy (45XO)
- Turner Syndrome (M/C heart lesion) = Bicuspid aortic valve
  - (2nd M/C) = Coarctation of Aorta
- Carcinoid Syndrome (T.I.P.S)
- Rheumatic Heart Disease (Rare)

Brugada Syndrome

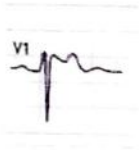
EKG Characteristics

Patients with Brugada have a pseudo-RBBB and persistent ST elevation in V1-V2.



Type 1

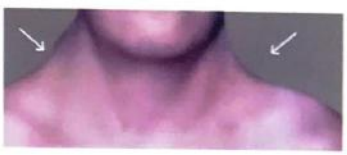
ST elevations> 2mm
Downsloping ST segment
inverted T wave



Type 2

ST elevations > 2mm
'Saddle back' ST-T wave configuration
Upright or biphastic T wave

Turner = 45XO
Webbing of neck

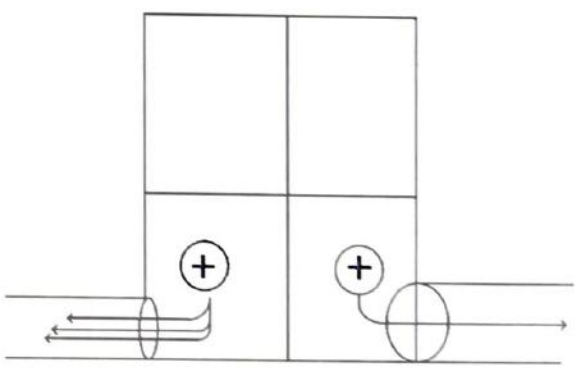


BAV
CoA

Severe Pulmonary Stenosis (> 50 mm Hg)
- Based on pressure gradient
  - (Between right ventricle & pulmonary artery)

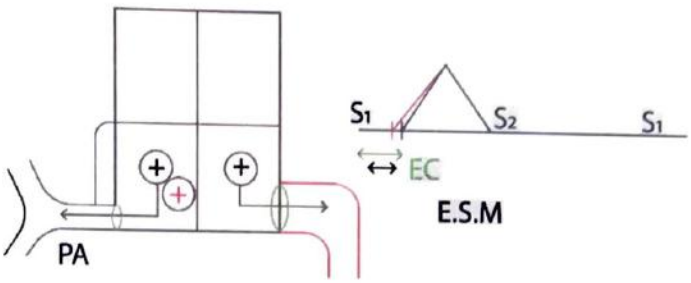
Mild Pulmonary Stenosis / Moderate Pulmonary stenosis
- Mostly asymptomatic

On Examination: Ejection systolic murmur
- Severity of Pulmonary Stenosis is $\alpha \frac{1}{\text{S1 - Ejection click gap}}$
- ESM +
- Ejection click absent
- Ejection click is less prominent with inspiration in pulmonic stenosis



All right sided events tend to increase with inspiration except pulmonary ejection click.

- S2 heart sound
  - A2 heard normally
  - P2 soft
  - Loud P2 is hear in Pulmonary Artery Hypertension
- S4 heart sound +.



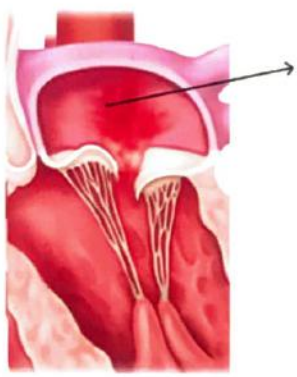
WORK UP OF PATIENT

00:35:41

1. Chest X-ray: Upturned apex of heart
2. ECG: Right axis deviation
   - P-pulmonale
3. Echocardiography (IOC)
   - Tells gradient b/w right ventricle and pulmonary artery > 50 mm Hg.
   - Right ventricle hypertrophy (Eccentric)

Treatment

- Percutaneous pulmonic valve valvotomy
- Pulmonary Regurgitation



Valve leaflets insufficiently closed, causing backflow blood into right ventricle

Causes

- Annular dilatation of pulmonic valve
- Severe Pulmonary Artery Hypertension
- Post Tetralogy of Fallot (TOF) repair
- Post balloon valvotomy

Pathophysiology

- Right ventricular dilatation

Clinical Feature

- Exercise intolerance
- Abdominal fullness
- Pedal edema

Important Information

- IOC
  - CARDIAC MRI > ECHOCARDIOGRAPHY

Treatment

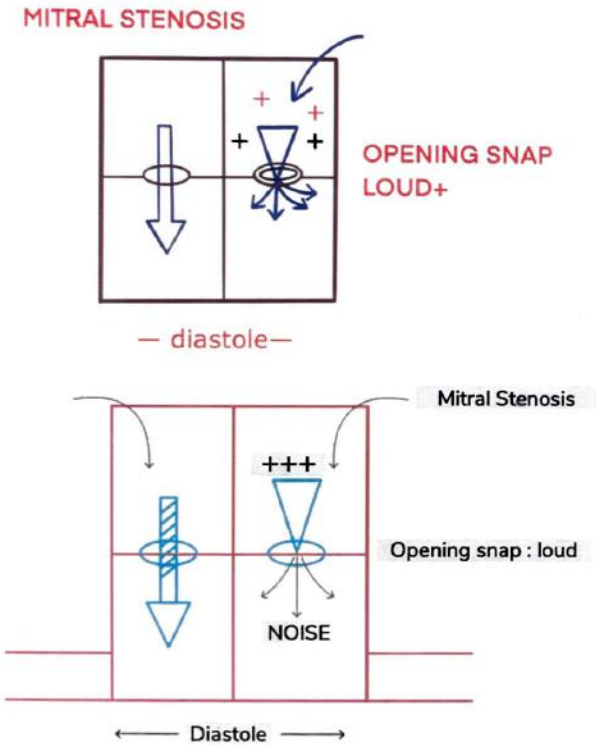
00:42:00

- Diuretics
- Transcatheter pulmonic valve replacement

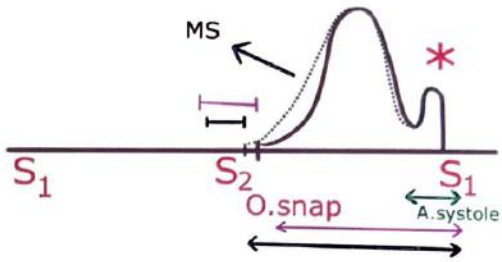
12 MURMURS

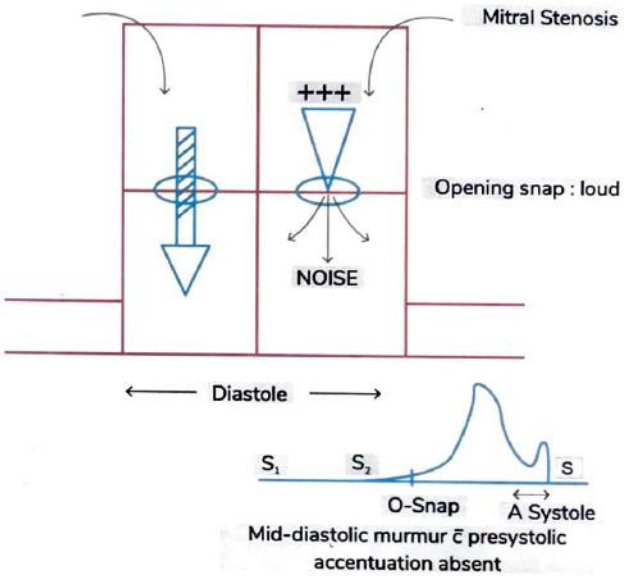
1. MITRAL STENOSIS (MS)

- Leading cause of MS is Rheumatic Heart Disease(RHD)
  - M/C RHD valvular lesion in **children** → MR
  - M/C RHD valvular lesion in **adults** → MS
- Normal size of valve (4-6 cm^2)
- Orifice of mitral valve becomes relatively smaller
  - Severe MS < 1.5 cm2 or Critical < 1 cm^2
- As a result there will be turbulence created in phase during blood enter from LA to LV
- During diastasis phase of cardiac cycle, normally there is free flow blood in ventricular cavity. This free flow will be hampered in case of MS
- Lesser blood will come to LV
- Turbulence will be generated due to narrow valve which results in sound called diastolic murmur
- This murmur always be heard after the mitral valve open
- The opening snap will occue , which will be louder than normal (because the pressure generated in left atria will be higher than normal), which explains the loud opening snap.
- To appreciate the noise , turn patient into left position, so that the heart come close to chest wall, to hear the murmur relatively better



- Murmur in MS: Mid-Diastolic Murmur (Beginning from opening snap)
  - Peak at middle of diastole, has small secondary spike
  - Called as Pre-Systolic Accentuation (PSA)

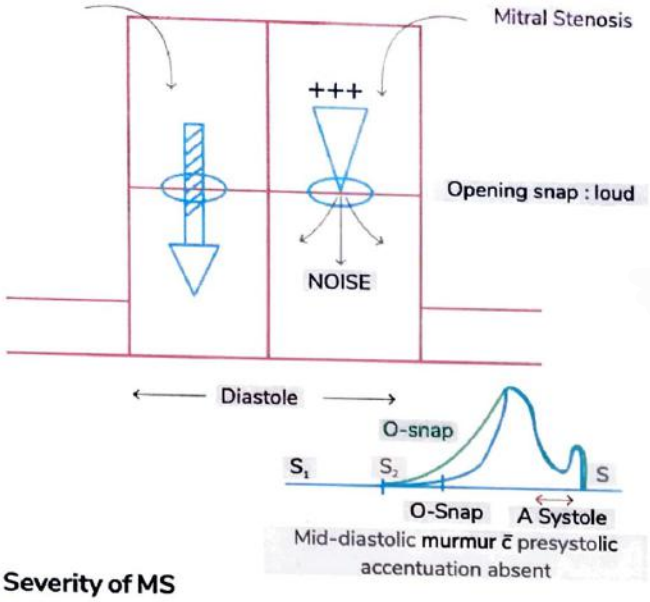




- Atrial systole Contributes to pre-systolic accentuation
- Pre-systolic Accentuation will disappear
- When atrial systole disappear
  - In Atrial Fibrillation (M/C)

Main treatment in severe MS (<1.5 cm^2) is balloon dilation

- Even opening become more smaller , more pressure generated which will cause early opening of the mitral valve which will shift the position of opening snap more towards S_2 (shown in green)
  - Sound will be earlier and louder
  - Dramatic increase in the length of murmur



Severity of MS

- Not decided by
  - Loud S_1
  - Loud P_2
  - Intensity of murmur

> **Important Information**
>
> - Decided by Length of Murmur (duration of murmur)

- Severity MS $\frac{1}{S2-O.S\ gap}$ os (opening snap)
  - ∝ length of murmur
- MS not only contributes to pulmonary edema, can actually cause right ventricular hypertrophy
- Rx: Percutaneous Mitral Balloon Valvotomy (PMBV) (Access taken via basilic vein to right atrium, puncture inter – atrial septum, reach left atria and then the stenosed valve)

Symptoms

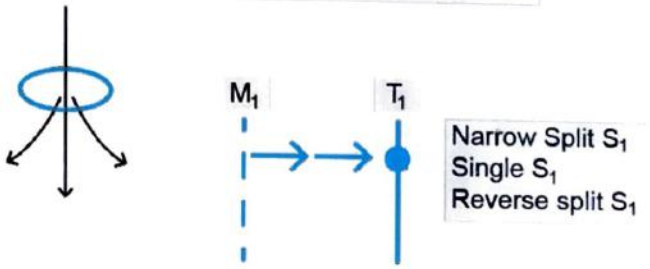
- Breathlessness initially with physical exercise and than on rest, as disease worsen
- Long standing pulmonary edema will cause hypoxia, vasoconstriction of pulmonary bed and PAH will occur
- RVH will occur
  - Dyspnea on exertion
  - Orthpnea
  - Paroxysmal nocturnal dyspnea (supine position of body)
  - LA dilation, press recurrent laryngeal nerve cause hoarseness of voice (ORTNER syndrome)
  - LA ++++: A. fibrillation, form clot cause embolic stroke
  - PAH: exercise intolerance (sudden deterioration), ankle edema

O/E

- Tapping apex beat
- S_1 loud: Transvalvular gradient increase
- Soft S_1: Calcified MS
- Narrow split S_1
- Single S_1
- Recerse split S_1
  - $S_2 = A_2 P2$:normal
  - If PAH :loud P_2
- S_3: Absent

S_1 LOUD :

SOFT S_1 : Calcified MS

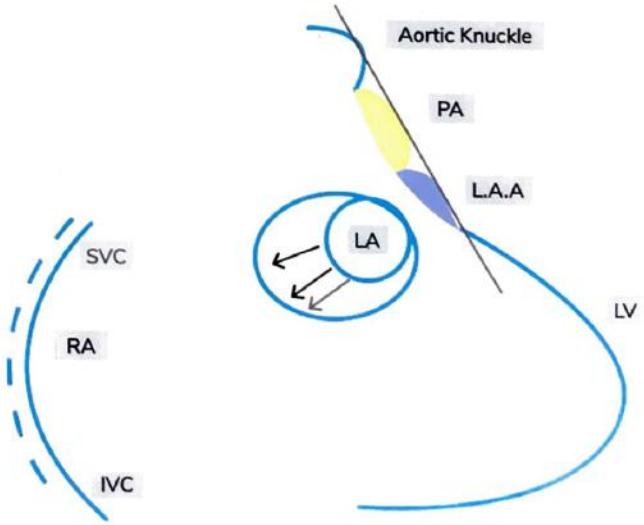


> **Important Information**
>
> - Murmur of MS heard better in Left Lateral Decubitus.
> - Since heart comes closer to chest wall
> - Murmur: MDM with PSA

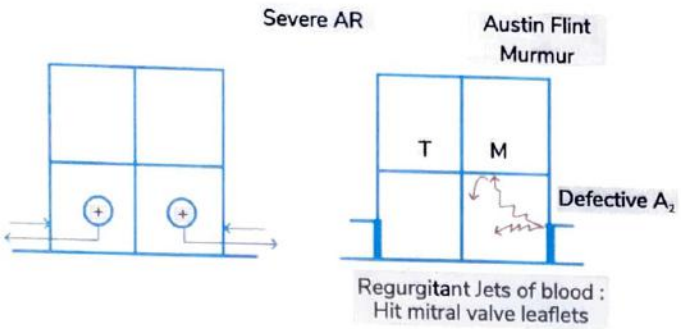
WORK- UP

00:22:38

- ECG



- P-mitrale
  - P- pulmonale> 2.5mm
- CxR
  - Straightening of left heart border



  - Double atrial contour (shadow)
  - Widening of cranial angle
- TTE (Trans thoracic echocardiography)
  - Investigation of Choice
  - Candle flame jet
  - Hockey stick sign

HOCKEY	STICK	SIGN
$\frac{MRI\ HEAD}{*VCJD\ (PRION)}$	$\frac{ECG}{Digoxin}$	$\frac{Echo}{M.S}$

MANAGEMENT

00:31:00

- **Severe MS**

Percutaneous Mitral Balloon Valvotomy (PMBV)

- Access taken via basilic vein to right atrium, puncture inter – atrial septum, reach left atria and then the stenosed valve

Contraindications to PMBV

1. Calcified Severe MS
- PMBV is C/I
  - forced balloon valve broke
  - fragments go to brain
  - Risk of Embolic Stroke

2. Severe MS + MR

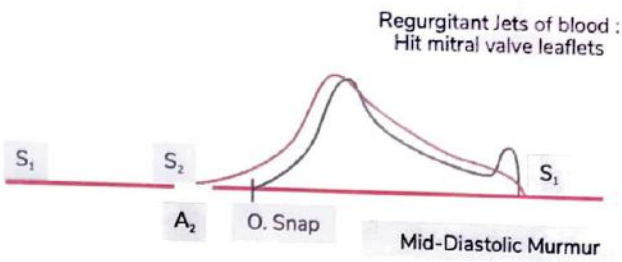
3. Severe MS + Left Atrial Appendage Thrombi (LAA clots)

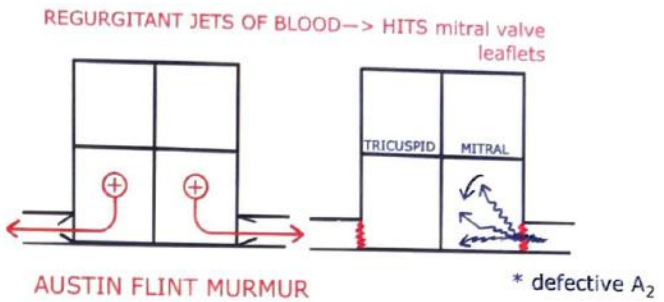
- Mild - Moderate MS
  - Digoxin + Spinolactone
- Pul. Artery HTN
  - AMBRISENTAN

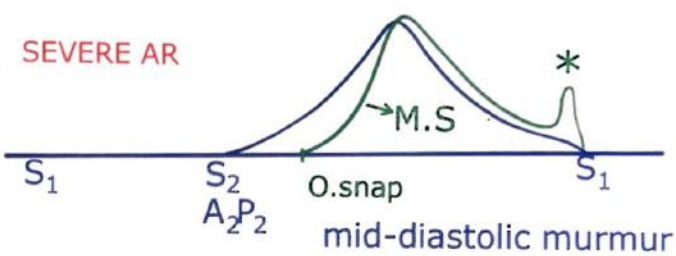
SEVERE AR

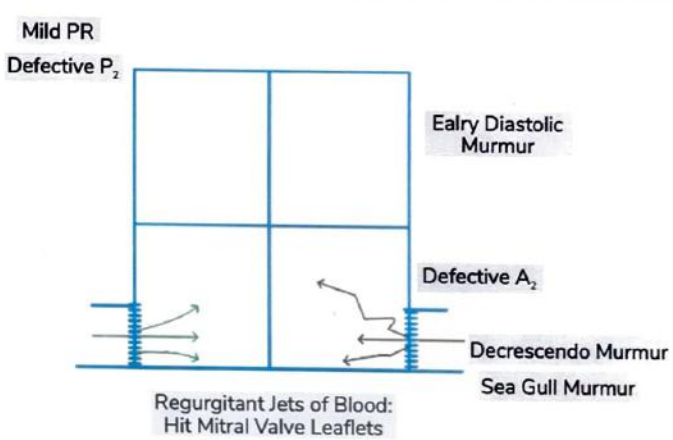
00:38:11

- Blood leaking back to left ventricle hits mitral valve leaflets
- Regurgitation jets of blood hit under surface of mitral valve leaflets
- Mid - diastolic murmur
  - AUSTIN FLINT MURMUR
- Start from S_2 (Not opening snap)

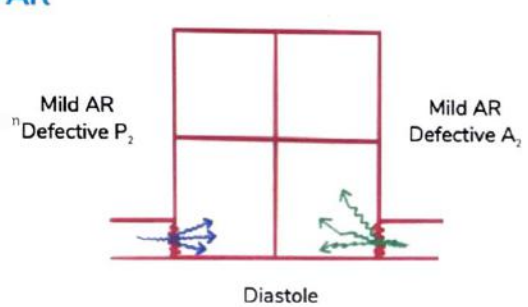


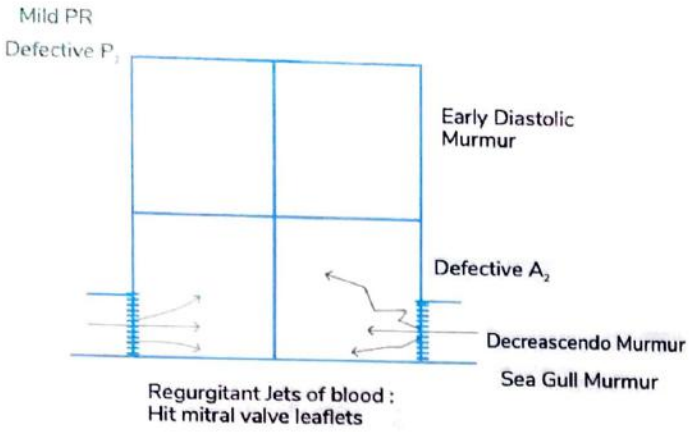






Mild AR

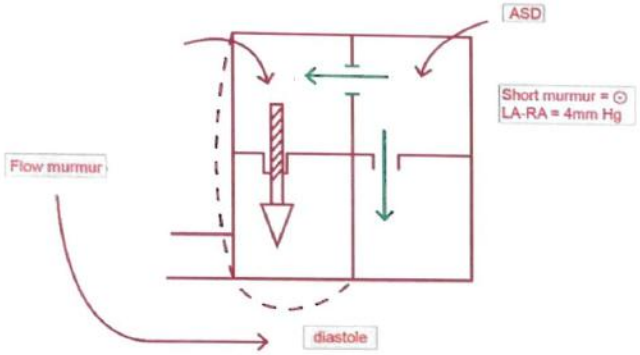




- Short duration murmur
  - Turbulence begins at A_2P_2 and dies down soon
- Decrescendo murmur or sea gull murmur or early diastolic murmur
  - Intensity of murmur decrease with time
- Triangular configuration

Patient with mild variety of pulmonary regurgitation

- Defect P_2
- Early Diastolic Murmur
  - A_2 & P_2 don't close together
  - Time lag - 30 millisec



MURMUR: DIASTOLIC

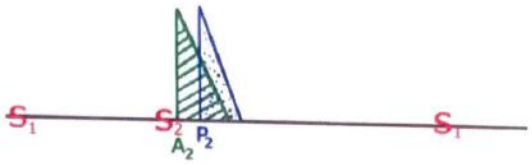
00:46:39

MURMUR: DIASTOLIC

Early	Mid	Late
GAP	**CAMF**	• Rheumatic carditis
• Graham steel	• Carey Coomb	• Carey coomb murmur
• AR mild	• Austin flint	
• PR mild	• MS	
	• Flow Murmur	

- GRAHAM STEEL MURMUR: Severe variety of PAH
- For Carey coombs murmurs if late diastolic is not in choices, then answer as Mid – diastolic murmur
- Carey coombs murmur is flow murmur

FLOW

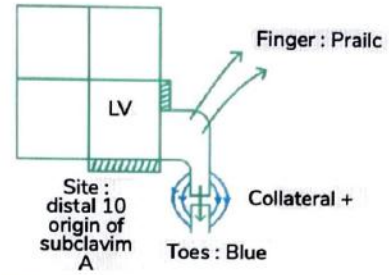


Early Distolic Murmur
Decrescendo/Sea Gull Murmur

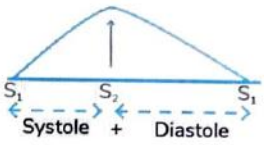
MURMUR

00:52:24

Coarctation of Aorta
45 X 0 : BAV > CoA
ESM



CONTINOUS MURMUR



Peak at S_2

ASD

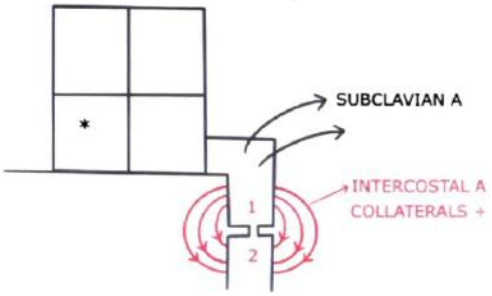
- LA-RA pressure gradient = 4 mm Hg
- Blood movement ⊕
- No shunt murmur θ due to extremely low pressure gradient
- More blood flow through tricuspid valve

FLOW MURMUR

- Mid-diastolic murmur

Important Information

- M/C murmur in Children : Innocent murmur (Systolic murmur)



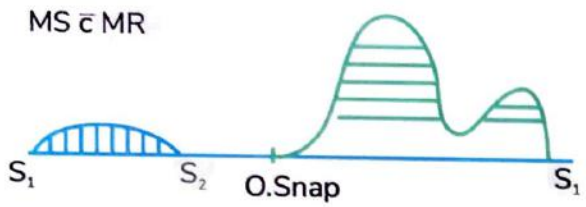
COARCTATION OF AORTA

00:58:13

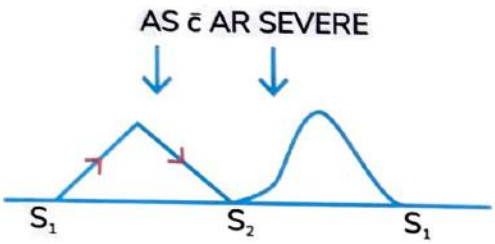
- M/C site: Distal to origin of left Subclavian A
- Murmur heard both systole and diastole
- Peak at S2

Continuous Murmur

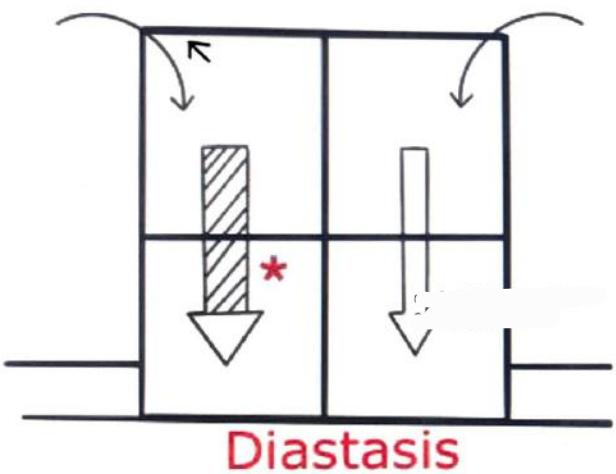
- Collateral formation
  - Toes blue , fingers pink



Pansystolic Murmur



Ejection Systolic Murmur



- Coarctation of Aorta is associated with Turner Syndrome

Important Information

- Commonest cardiac lesion in Turner Syndrome is
- BICUSPID Aortic Valve > Coarctation of Aorta

Causes of Continuous Murmur

1. CoA
2. PDA associated with Congenital Rubella Syndrome
3. Mammary SOUFFLE in pregnant lady
   - ↑ Blood flow (Internal mammary A)
4. Venous Hump : dilated veins
5. Rupture of Sinus of Valsava
   - Communication develop b/w RA & aorta
6. Peripheral pulmonic stenosis

Previous Year's Questions

Q. Which of the following leads to continuous murmur? (FMGE June 2019)

A. Peripheral pulmonic stenosis

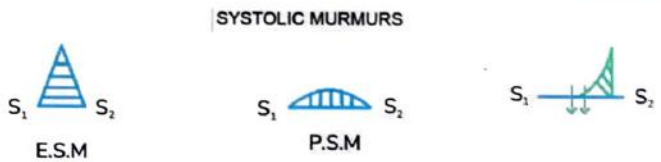
B. Severe pulmonary artery hypertension

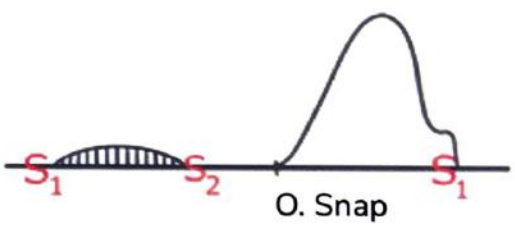
C. Type A aortic dissection

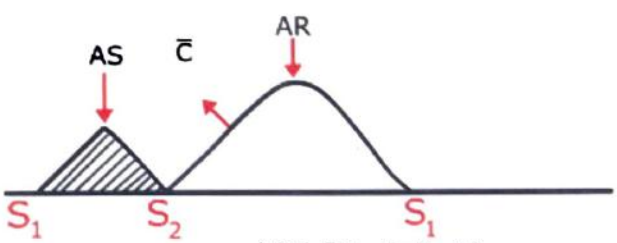
D. Rupture of cardiac chamber

MS WITH MR

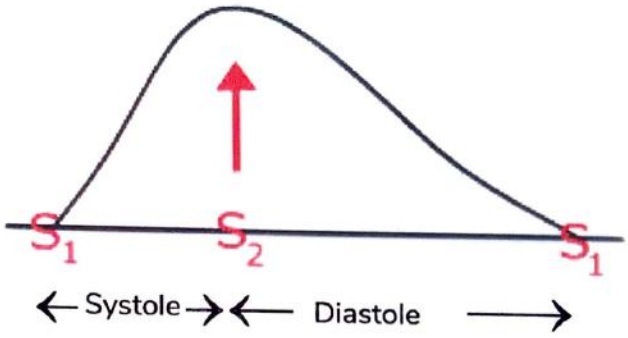
01:06:20

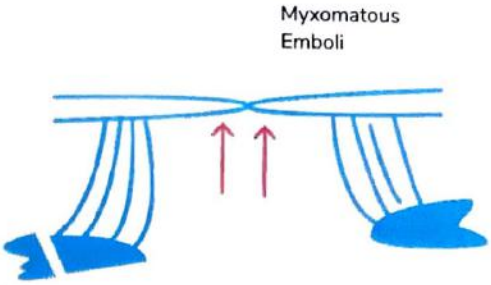






Mid-Diastolic Murmur





MS WITH MR

01:09:41

- Pan systolic murmur

Intensity - same

- Begins at op. Snap
- Not a continuous murmur AS with AR
- Ejection systolic murmur
- Non of combination lesions satisfy the definition of continuous murmur
- Combination lesion never satisfy criteria of continuous murmur

SYSTOLIC MURMURS

Ejection Systolic Murmur PASS

S1 Δ S2

1) PS
   - carcinoid syndrome (Tricuspid insufficiency and pulmonic stenosis TIPS)
   - Noonan syndrome
2) AS
3) HOCM
   - Sub-valvular Aortic stenosis
4) TOF
   - Sub-valvular PS

Pan Systolic

S1 ▨ S2

1) MR:VSD
2) TR

Mid systolic click/late systolic murmur

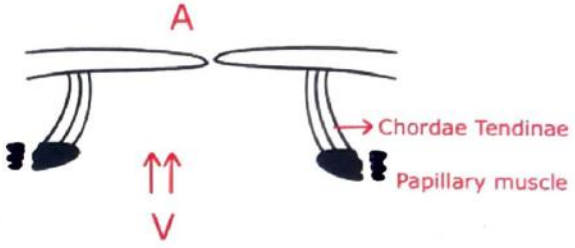
1) Mitral valve prolapse
   - Myxomatosis degeneration of valve
   - Barlow syndrome

MVP

- Damage to papillary muscle (eg: MI)
- No support to chordae tendinae
- If Pressure increases, valve leaflets go higher than normal
- In MVP, there is extra tension in chordae tendinae
  - Resulting in mid systolic click
- There is also leakage of blood after mid systolic click (when leaflet separate)
  - This will result in late systolic murmur

Cause of MVP

- Myxomatous degeneration of papillary muscle - (MC)



- Aschoff Nodules - Rheumatic fever
- MI (Ischemia)
- Infection SABE
- Defect in fibrillin I protein – Marfan Syndrome
- All murmurs ↓ in Intensity with Valsalva / Standing / Amyl nitrate inhalation dueto ↓ venous return

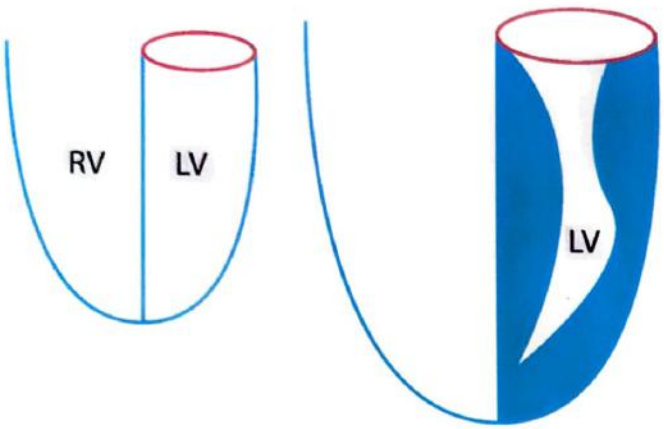
Important Information

- All murmurs ↓ in Intensity Except
  - HOCM – Loudé.
  - MVP - Longer
- All murmurs ↑ in intensity with squat / hand drip
- Except
  - HOCM – Softer
  - MVP- Shorter

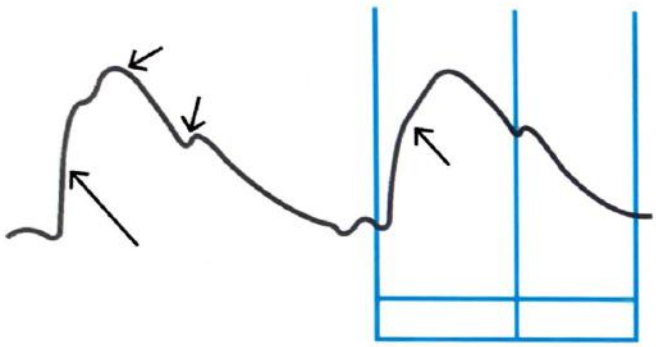
13 CARDIOMYOPATHIES

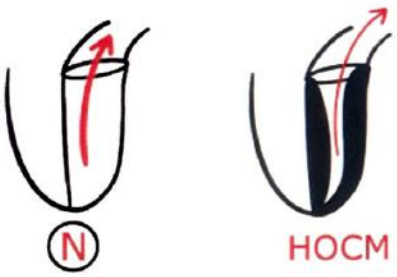
HOCM /HCM

00:00:13



- AD
- β- myosin gene defect (MyH7 gene), ch-14 abnormality





Pathogenesis

1) Diastolic Dysfunction → due to small LV cavity size
2) Asymmetrical septal hypertrophy
3) Left ventricular outflow tract obstruction (LVOTO). Since obstruction is not at the level of aortic valve rather below is so called as subvalvular aortic stenosis
4) Free wall thickness of LV >15mm/1.5cm
5) Banana shaped LV

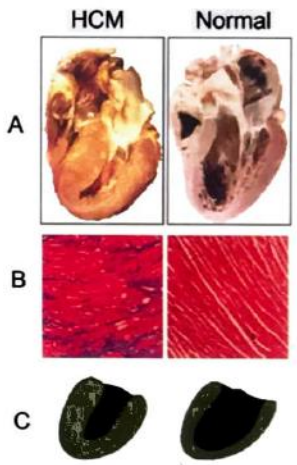
C/F

00:08:15

- 10-20 yrs Male

1. Dyspnea (earliest symptoms)
   - Diastolic malfunction (due to small LV cavity)
   - Pooling of blood in lungs
   - Pulmonary Oedema present
2. Chest pain on exertion– Sub. Endocardial Ischemia
3. Effort intolerance
4. Sudden collapse / death on exercise/ running

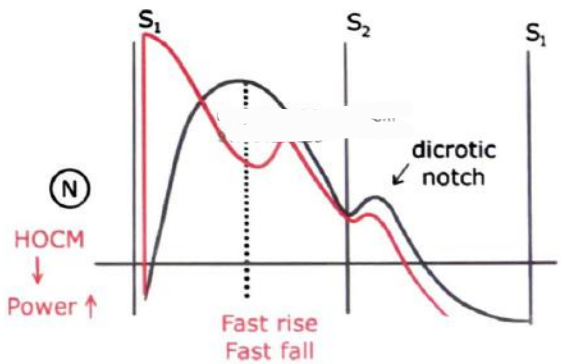
- Due to Ischemic ventricular fibrillation
- Twitching
- Loss of Pulse / BP
- Sustained arrhythmias: A. Fib



O/E

00:16:28

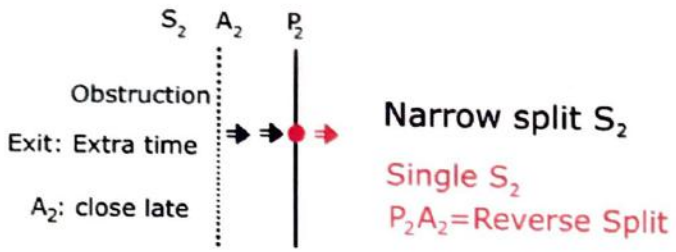
1. **Pulsus Biseriens (aka) "Jerky Pulse"**



- Fast rise initially
- Due to hypertrophy of muscles
- Able to generate more pressure/ power
- Pulse will rise rapidly (initial fast rise)
- Due to LVOTO, sudden dip in pressure (ejection velocity) (Fast fall)
- Leading to Jerky Pulse (Pulsus Bisfiriens)

2. **Double apical impulse** (5^{th} + 6^{th} I/C space)
3. **Heaving apex beat**
- Displaced impulse

4. **S_2: Narrow split/ single S_2 / Reverse Split S_2**
- S_2 (A_2/ P_2)
  - Ⓝ A_2 comes first, then P_2
  - Ⓝ time lag = 30 msec (splitting) A_2P_2, best heard during inspiration at Erb's point

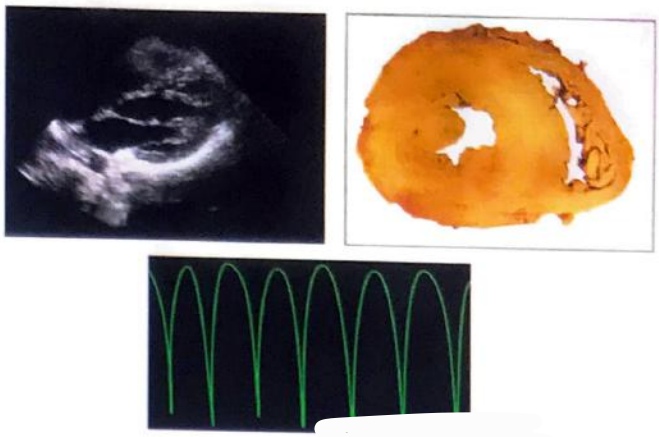


- HOCM → LVOTO (left ventriculat outflow tract obstruction)
  - Aortic valve will relatively take more time to close → Narrow split S_2
  - Can close at same time as pulmonary valve → single S_2
  - May take more time than pulmonary valve → Reverse split (P_2A_2)

5. S_4 is heard due to out flow obstruction, left atrial hypertrophy occurs. The extra force generated by left atrium explains occurrence of S_4.
6. Ejection systolic murmur 00:27:45

E S M ⊕

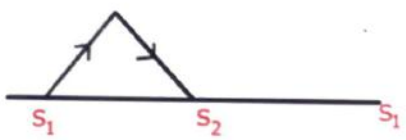
- CRESENDO- DECRESENDO MURMUR
- Intensity is not same (sudden rise then sudden fall in intensity)
- All murmur ↓ in intensity with Valsava, Standing, Amyl Nitrate, except:
  - HOCM = Louder (volume of LV directly proportional LVOTO)
  - MVP = Longer



INVESTIGATION 00:35:28

1. T.T.E- (MC)Banana shape cavity/Asymmetrical systolic hypertrophy/ Systolic antero movement of mitral valve/LV free wall thickness (most imp.)
- >15mm: HOCM
- >30mm: Incidence of sudden cardiac death will increase, patient require implantable defibrillator

2. ECG : determine LVH
- Depth of S wave in V_1 & R wave in V_5 : >35 mm – indicates Hypertrophy



3. ECHO –(IOC) LV Free Wall hypertrophy
- SAM- Systolic anterior movement of mitral valve
- LVEDV ↓ SV↓

4. Cardiac Catherization
- Brockenbrough-Braunwald-Morrow Sign
- Seen in post PVC

Treatment 00:39:20

- DOC:

1. Propranolol
- ↓ HR
- ↓O_2 consumption
  1. Verapamil
  2. Disopyramide
- LVOTO decrease

CLINICAL SCENERIO

- 20 year male chest pain at rest. After sub lingual NTG chest pain started worsening

Diagnosis

HOCM: V. Fib

- LV cavity reduce
- Coronary blood flow decrease

- After NTG RV blood reduce
- That reduce blood flow in LV
- Decrease in SV
- Decrease in coronary blood flow
- Chase pain worsens

Important Information

- NTG. Furosemide. ACEI. ARB. Digoxin. CCB (amlodipine)

Sudden Cardiac Death

1. Athlethe heart: LVH, Kinking of coronary artery
2. Commotio Cordis (martial athleths): Collapse death (H/o blunt trauma): V. Fib
3. Holiday heart syndrome: A. Fib

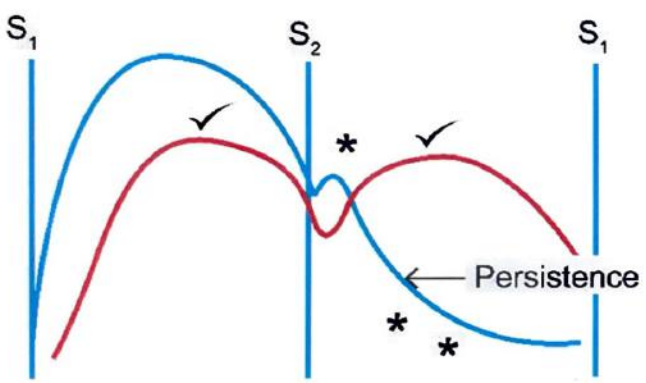
INTERVENTIONS AVAILABLE

00:50:27

1. **ICD** (Implantable Cardioverter Defibrillator): answer it as treatment of choice if there is
   - Family H/O sudden cardiac death
     - LV free wall thickness > 30 mm
     - R. syncopal attacks
2. Alcohol based septal ablation
3. Myomectomy

RESTRICTIVE CARDIOMYOPATHY

00:54:07



- Fibrosis of myocardium
- Rarest cardiomyopathy
- Size of ventricles (N)

Causes

1. Amyloidosis
2. Sarcoidosis
3. Radiation
4. Hemochromatosis
5. Scleroderma, Polymyositis, Dermatomyositis
6. Endomyocardial fibroelastosis
7. Hypereosinophilia

C/F

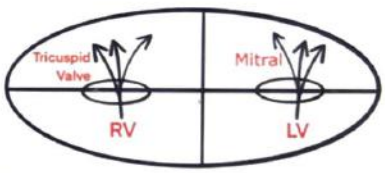
00:59:19

- **RV compliance decrease**
- Ventricle cannot receive much blood
- There will be pooling a blood leading to
  - Pedal edema
  - Ascites
  - Hepatomegaly (RUQ discomfort)
  - Kussmaul sign ⊕
- **LV compliance decrease**
  - Pulmonary edema due to diastolic malfunction, Dyspnea in exertion, Orthopnea, paroxysmal nocturnal dyspnea
  - Effort intolerance
  - Clot (+): Mural thrombus (embolic stroke)

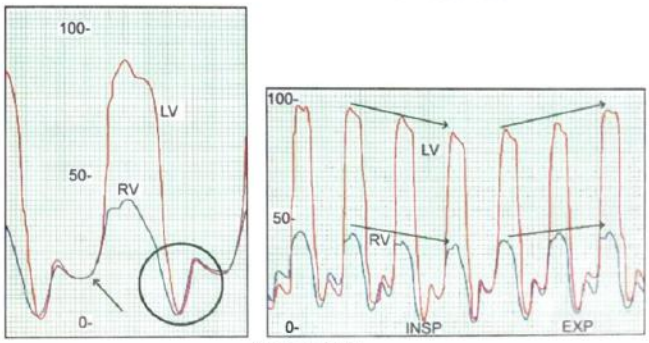
INVESTIGATION

01:03:14

1. **CXR**
   - Pulm. Edema ⊕
   - Peri Hilar infiltration (Bat wing appearance)
2. **ECG**
   - Low Voltage ECG
   - Myxoedema Heart
   - Constrictive pericarditis – Calcification ⊕
   - RCM- Fibrosis ⊕



Restrictive Cardiomyopathy

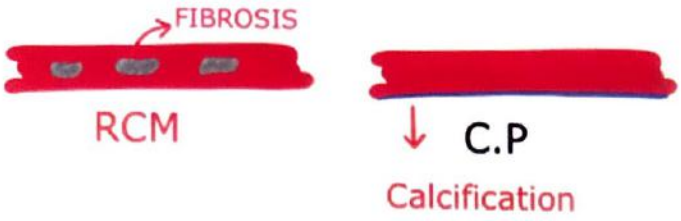


Dip and plateau- "square root"

- R+S = < 5 mm limb lead
  < 10 mm chest lead

3. **Doppler Echo**
- Square root wave sign (also seen in constrictive pericarditis)
- LVEDP increase

4. **Endomyocardial Biopsy (gold standard)**



5. **Cardiac MRI:** Preferred Imaging modality

TREATMENT

01:08:46

1. Oral Anti coagulants (Fibrosis can lead to coagulation)
- ↓ Embolic stroke incidence
2. Oral diuretics
3. Cardiac Transplantation - TOC

DILATED CARDIOMYOPATHY

01:10:10

Causes

1. Sequalae to coxsackie B myocarditis
- Parvo virus
- HHV6
- Coxsackie B
2. Sarcoidosis
3. Duchenne's muscular Dystrophy
4. Toxin – alcohol
5. Functional MR, TR
- Pansystolic murmur

CLINICAL CASE

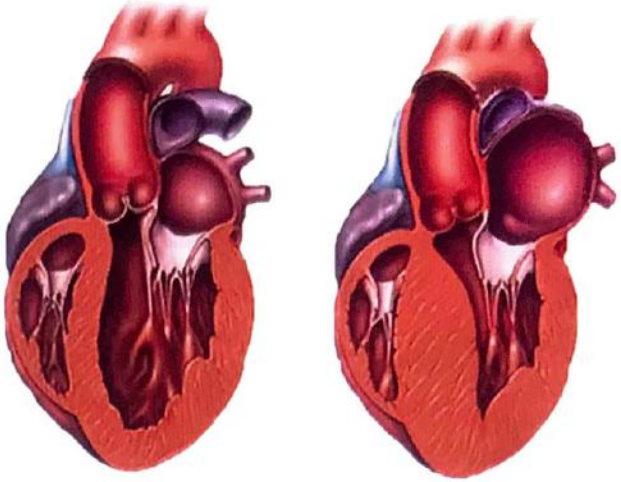
01:16:15

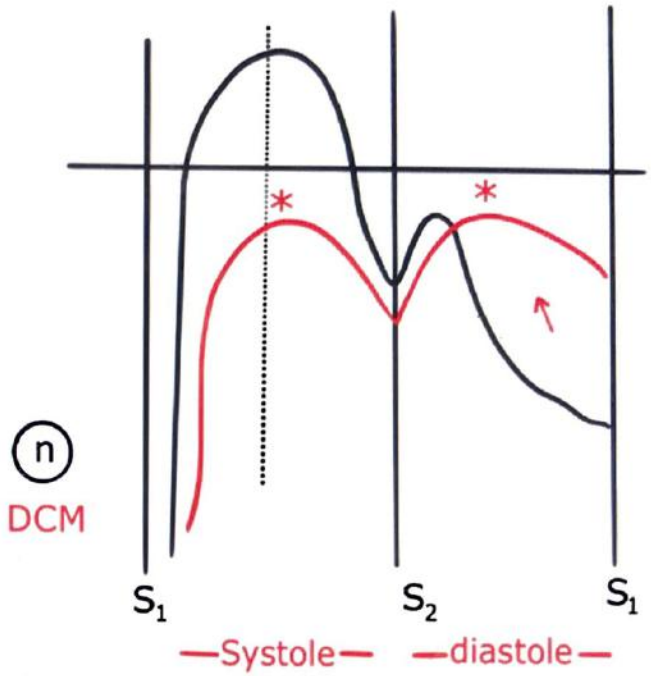
- Alcoholic, RVF (pedal edema, RUQ discomfort), LVF

O/E

01:17:04

1. **Dicrotic Pulse**
- Twice beating pulse palpable both in systole as well as diastole
- Loud S1/ soft S1





2. Pansystolic murmur along with features of biventricular failure
3. S_3 +
4. B/L fine crepitation +
5. Hypokinetic, diffuse, laterally displaced impulse

WORK UP

01:20:58

1. T.T.E: IOC
2. CxR: CT ratio increase
3. Cardiac MRI (overall best)
4. Endomyocardial biopsy

TREATMENT

01:24:16

1. Cardiac transplantation
   ICD can be deployed

2. Cardiac Resynchronization Therapy
3. Oral Thiazides

Important Information

- M/C Cardiomyopathy leading to cardiac transplantation: HOCM

PERIPARTUM CARDIOMYOPATHY

01:27:54

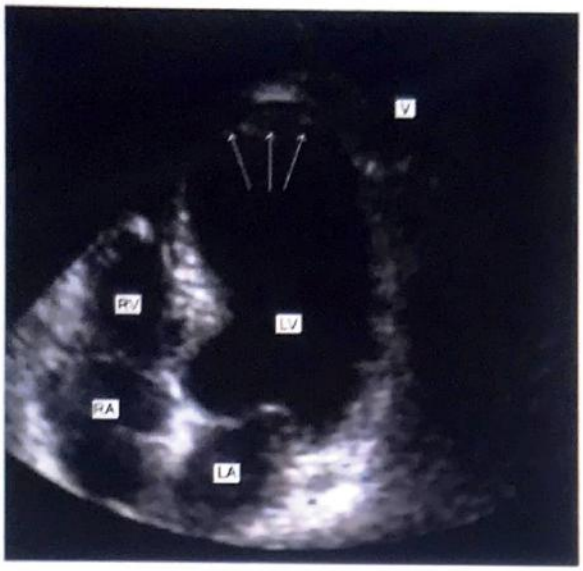
- Best prognosis
- Similar to Dilated cardiomyopathy
- Last month of pregnancy
- 5mth post partum

Ischemic Cardiomyopathy

- Worst prognosis

14 TAKOTSUBO CARDIOMYOPATHY & BRUGADA SYNDROME

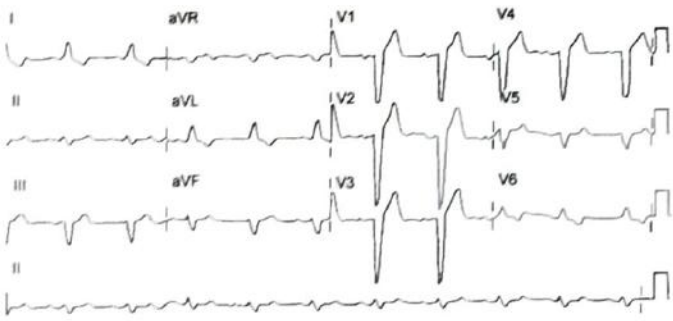
TAKOTSUBO CARDIOMYOPATHY ⏱ 00:00:12



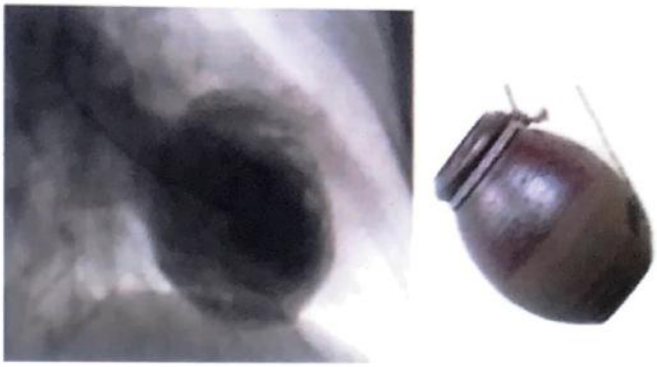
- TTCM is also called Broken Heart Syndrome
- occurs due to catecholamine toxicity
- Intense emotional trauma
- Occur in patient who had life threatening stress. Eg. Earthquake

1. **Case based discussion:** 60 yrs ♀ trapped in a elevator during an earthquake, emergency crew rescued her, she was rushed to hospital. She complained of chest pain at rest / diffuse in character & diaphoresis HR=120/min, BP=90/60mmHg, ECG: V_1-V_6: ST elevation, Trop I : double, Coronary angiography normal, Primary PCI: Abandoned, Echo: bulge of left ventricle

- **ECG**



- "TOMB STONE PATTERN" / Pardee sign: Seen in V2-V6 leads (suggestive of extensive anterior wall MI, due to thrombus in left main coronary artery)



- **Trop I**: 0.08 mg/dl (N)
- **Cath. lab** - Coronary angiography normal
  Primary PCI: Abandoned
- **ECHO**: Alteration of LV shape

Treatment

- Treat as case of cardiogenic shock → IABP (Intra – Aortic Balloon Pump)
- ACEIs, beta blockers
- Causative Agent → Catecholamine therefore dopamine, dobutamine should not be given

Important Information

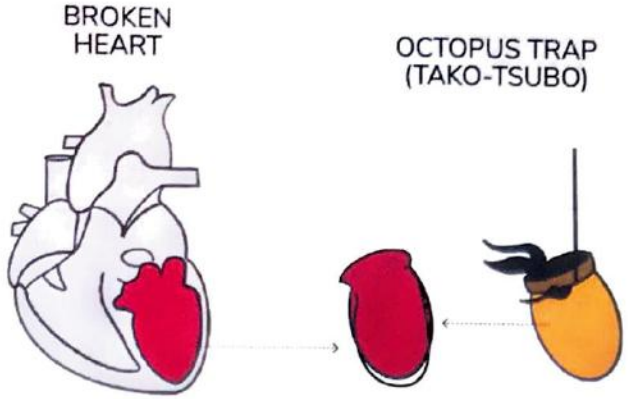
- **Acute onset cardiomyopathy**
- **Improvement can occurs gradually**
- **Mimics STEMI**
- **Cath Lab: Normal angiography**
- **(It is differential diagnosis of STEMI and diagnosis is made in cath lab due to normal coronary angiogram.)**

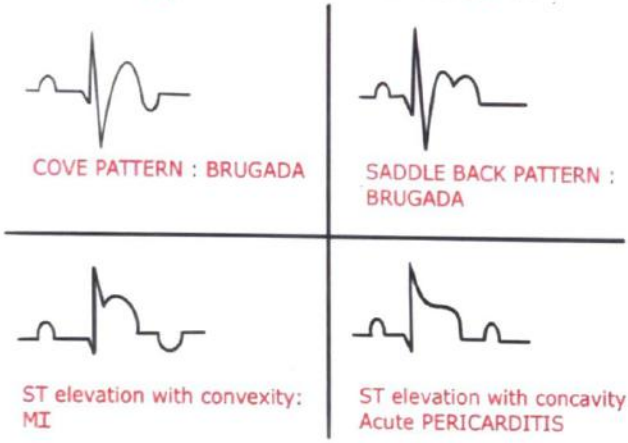
BRUGADA SYNDROME ⏱ 00:14:03

- **SCN5A #** → defective Na+ influx
- Due to sodium channel defect, voltage gradient is created between RV epicardium & normal heart → will trigger
- Fib/VT/TDP (Torsades de pointes)
- Patient will become pulseless
- Family H/O, H/O syncopal attacks
  - Sudden death in sibling

- It is the leading cause of sudden nocturnal death in SE Asian males

- **ECG** → ST elevation in $V_1 - V_2$ (Right chest leads)





Brugada Syndrome
EKG Characteristics

Patients with Brugada have a pseudo-RBBB and persistent ST elevation in V1-V2.

Type 1
ST elevations> 2mm
Downsloping ST segment
inverted T wave

Type 2
ST elevations > 2mm
'Saddle back' ST-T wave configuration
Upright or biphastic T wave

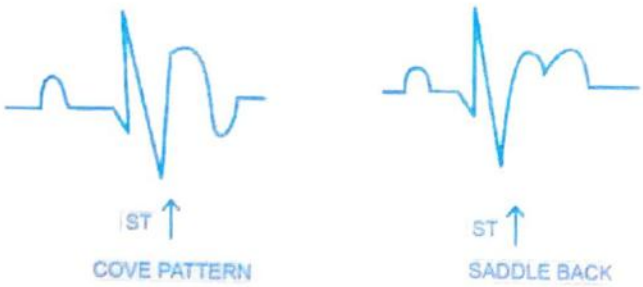
TREATMENT

00:20:33

- I.C.D (Implantable cardio defibrillator)

Indications I.C.D

A
- LV aneurysm,
- Arrythrogenic RV dysplasia (fibro fatty deposition in RV)
  - Family H/o sudden death
  - Epsilon wave



  - T wave inversion V_1-V_4

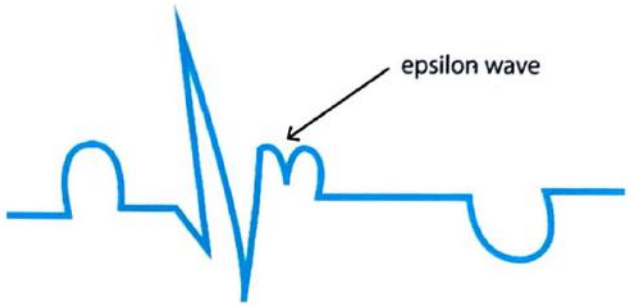
B
- Brugada syndrome

C
- Cardiomyopathy

SUDDEN CARDIAC DEATH

00:27:44

- HOCM: V. Fib/ VT
- Holiday Heart Syndrome: A. Fib
- DM: Silent MI
- Brugada: TDP
- Long QT syndrome: TDP
- WPW: A. Fib - V. Fib



Important Information

Brugada
- **Cove pattern: ST elevation with concavity**
- **Saddle back pattern: ST elevation with concavity**

15 RHEUMATIC HEART DISEASE

RHEUMATIC FEVER

00:00:15

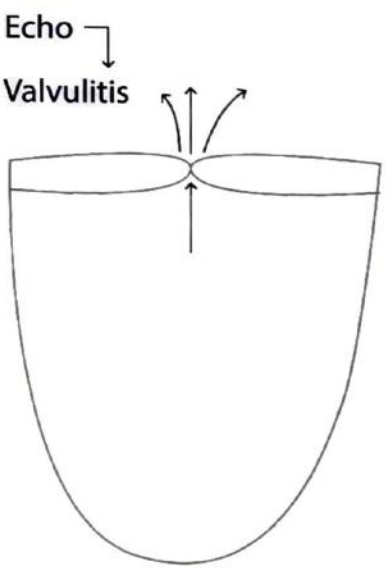
- Organism involved – Group A Beta hemolytic streptococcus
- Type II Hypersensitivity /Molecular mimicry
  - Antibody formed against M-Protein of GAHS
  - Cross react with the N-acetyl glucosamine present in human connective tissue
- Formation of Aschoff nodules
  - Has antischkow cells also known as caterpillar cells

MODIFIED JONES CRITERIA (2015 UPDATE)

00:04:13

Major criteria

1. **Carditis**
   - **Subclinical**
   - Echo with Doppler must be done in all cases of confirmed and suspected acute Rheumatic fever as auscultatory findings take time to develop
   - Subclinical carditis seen on echo is now considered a Major diagnostic criteria
- **Clinical**
  - Pericarditis (M/C)



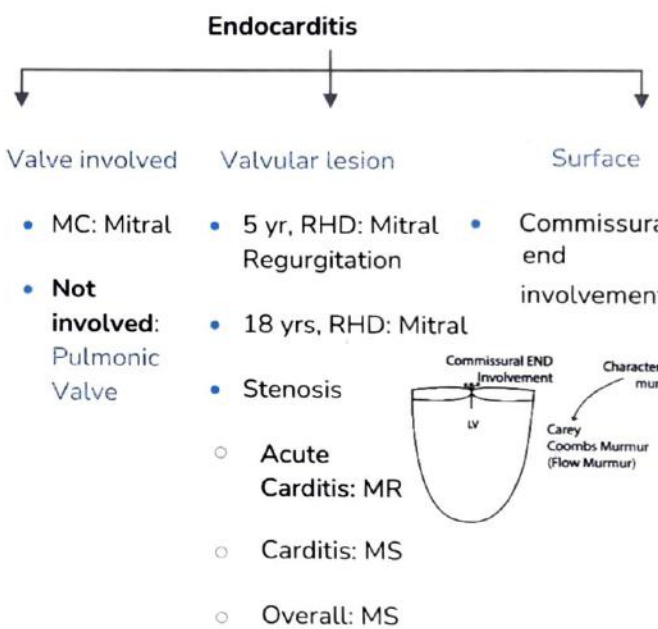
  - 5-15years, Recurrent Sore throat
  - Chest pain at rest, pericardial friction rub
  - ECG- ST ↑, concave upwards present in all leads except aVR.
- **Management**: Steroids
  - Myocarditis: acute CHF
  - Dilation of valve annulus leading to
    - MR functional
    - TR functional
    - Pansystolic murmur
  - Manifestation- Acute Pulmonary edema
  - Management
    - **L - LASIX**
    - **M - Morphine**
    - **NO - NTG, O_2**
    - **P - Head high Position**

How to remember

- LMNOP

ENDOCARDITIS

00:12:00



Aschoff nodules involve commissural ends (tips of valve where cusps touch each other)

- Due to Valvulitis
- Blood leaking across the valve (LV → LA)
- As this extra blood accumulate in left atria and when

come back to LV results in murmur called as Carey Coomb Murmur (flow murmur):Mid diastolic murmur > late diastolic murmur

- Low endemic
  - <2/100,000 school age population
  - <1/1000 all age population
  - MC Callum patch: Characterised by damage to free wall of left atria

Management

- Severe MS (< 1.5 cm^2): PMBV
- Calcified MS: prosthetic vave
- Severe MR: Valvuloplasty

2. Arthritis 00:22:19

- M/C & Earliest manifestation
  - Earliest manifestation within 2 weeks with H/o sore throat
- Ankle swelling
- Knee swelling
- Elbow swelling

} Migratory polyarthritis

- All joints do not affect simultaneously, they affect one after the other
  - When swelling subsides, no residual deformity: Non-erosive arthritis
  - Sometime can cause erosive arthritis called as Jacoud's Arthritis

Low endemic (USA)	High endemic (India)
• Migratory polyarthritis (multiple joint involvement one after the other)	• If single joint involved i.e. Monoarthritis: Sufficient for diagnosis of Arthritis • Polyarthalgia

Management: Aspirin/ Naproxen

- Aspirin + Varicella Zoster : Reye Syndrome
- Aspirin + Influenza B

00:27:54

Rheumatoid arthritis	Rheumatoid
Young female	Child/18yr
Morning stiffness (PIP, MCP, wrist)	Large joint
B/L symmetrical	Asymmetrical
Anti CCP	ASO titer
MTx,Lefluonamide	Aspirin
Erosive arthritis	Non erosive arthritis

3. Syndenham Chorea (Late neurological feature)

- Can develop as late as 90 days or later 00:30:54
- Damage to caudate nucleus
- Fast, purposeless, involuntary distal movements
- Poor handwriting
- School grades ↓se

Difference from Chorea

Athetosis: Slow, writhing distal involuntary movement

- D/t lesion: Globus pallidus
  - Hemibalismus: wide flinging movements proximal & distal
- D/t: Subthalamic nucleus

O/E

- Darting Tongue (Inability to keep tongue in stable position)
- Milk-maid grip (Inability to generate persistent hand grip)
- Hung up reflexes (Hung up ankle jerk – Myxedema)
- Dysarthria (unclear speech)

MANAGEMENT

- Self limiting condition: resolve in < 6 months – 12 months
- Valproate
- Haloperidol
- Phenobarbitone
  - ↓ If Fails
- Steroids
  - ↓ If Fails
- IV Immunoglobulin
- **Medically refractory chorea**: IV Ig

4. Subcutaneous nodules 00:42:40

- Extensor in distribution
- **Sites:** Olecranon, occiput, spinous processes of Vertebrae
- Rheumatoid nodules: - M/C
- Extra- articular manifestation of rheumatoid arthritis
  - Extensor, non-tender
  - Involve lungs, heart, mononeuritis multiplex

5. Erythema Marginatum (Not seen in Indian population)

- **Minor Criteria**

i) Fever (>38.5°C), Low endemicity (>38°C), High endemicity

ii) Arthralagia, Poly, Mono

*

Lab features

- ESR ↑
  - Low endemic : > 60 mm fall/ 1st hour
  - High endemic: > 30 mm fall / 1st hour
- CRP: ⊕
- Prolonged P-R interval / 1stdegree heart block

SAN ⟶ AVN

Aschoff Nodules

↓

Slow down the conduction

↓

Prolonged PR interval

Essential Criteria

Evidence of preceding Grp A Streptococcus infection

1. ASO titer > 200 IU/ml suggestion of recent streptococcal infection in previous. < 45 days (Evidence of infection) or Rapid streptococcal antigen test is positive or positive throat swab

Important Information

- **Chorea**
  - **late feature (can develop as late as 90 days or later)**
  - **ASO values can normalize by the time chorea will develop**

DIAGNOSTIC ALGORITHM

- Major criteria + essential criteria (evidence of preceding Grp A Streptococcal infection)
- 1 major+ 2 minor criteria + essential criteria (evidence of preceding Grp A Streptococcal infection)
- Recurrence ARF: 3 minor criteria

ESSENTIAL CRITERIA, UNIVERSAL CRITERIA, RECOMMENDED TESTS (EVIDENCE OF RECENT STREPT. INFECTION <45 DAYS)

1. Elevated ASO titres 00:53:20
2. Anti DNASE B ab
3. Rapid gp A Strept carbohydrate test
4. Throat swab

Management 00:55:55

1. Aspirin: Arthritis
2. Steroids: Pericarditis/ severe chorea
3. Valproate: chorea
4. IV Ig: medically refractive chorea
5. Injection Benzathine penicillin I.M. every 3 weeks

- **Why?** => To prevent future sore throats
- **How Long?** => RHD with MR → lifelong basis is ideal but not practical
- Upto 40 yrs of age if valvular lesion is present or 10 yr from date of diagnosis. (whichever longer)
- Duration of Prophylaxis with Benzathine Penicillin

RHD

	Age till injection given	Time from date of diagnosis
RHD with valvular lesion	40 yr	10 yr
RHD without valvular lesion	21 yr	10 yr
RF	21 yr	5 yr

- whichever is longer

Best for prophylaxis of RF/RHD in penicillin allergic child: Macrolides

- Azithromycin

Penicillin allergic Anaphylaxis

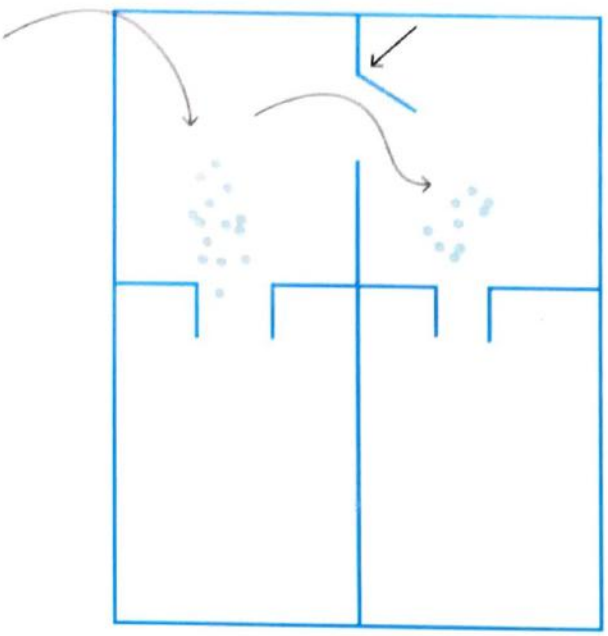
- Basophilis
- IL-4
- Death: histamine induced laryngeal edema
- I/M Adrenaline undiluted (1:1000) Only Scenerio
- Increase S. Tryptase levels confirms anaphylaxis shock

Important Information

Cause of death in anaphylaxis is laryngeal edema

16 INFECTIVE ENDOCARDITIS

00:00:13

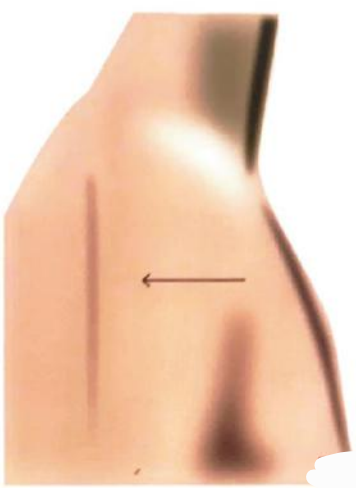


ABE { Hospital acquired: S. Aureus
Community acquired: Streptococcus

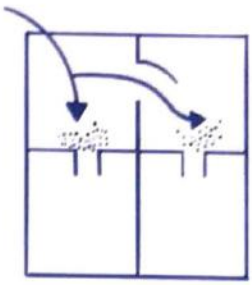
SABE- S. Viridans

Prosthetic Valve Endocarditis

- Early (≤ 60 days) = C. O. N. S (Coagulase –ve staphylococcus)
- Late (> 60 days) = Strep. Viridans
- IV drug abuser/ Right sided Endocarditis – Staph. Aureus



- **Left sided Endocarditis** → Patent foramen ovale
  R → L Shunt)



- **Organisms responsible**
  - S.Aureus
  - Enterococci

NATIVE VALVE ENDOCARDITIS

00:05:17

- Mitral valve is most commonly involved in IE
  - Native value endocarditis → Mitral
  - IE incidence is directly proportional to pressure gradient

1. MS: Diastole
2. MR: Systole: LV-LA (Highest pressure gradient so highest incidence of infective endocarditis)
3. AS:LV – Aorta (Systole)
4. AR: Aorta – LV (Diastole)

INCIDENCE OF IE

00:05:11

- **Highest** = MR
- **Least** = ASD (LA- RA) = 4 mm (pressure difference is less)
- Highest (Children) = VSD> PDA
- Vegetations = Fibrin + Bacteria

- Pt. will be having high grade fever/ chills/ rigors
  - (Septic abscess) → Perforation
- These vegetation can embolize from LV to Aorta lead to systemic manifestations
  - New onset murmur in pre- existing heart disease

complications

- Splenic abscess → Splenomegaly
- Brain abscess → Hemiplegia
- Clubbing (in SABE)

EXTRA MILE

- Duke criteria = IE
- Duke score = Severity of chronic stable angina.
- Duke staging = Colorectal CA

CLINICAL SCENERIO

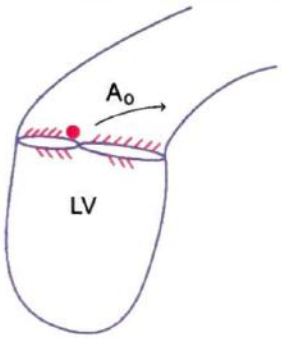
00:15:02

- A child diagnosed case of RHD, feverX3 weeks, all work up for fever done and are negative. O/E splenic tip palpable

Diagnosis : IE

Important Information

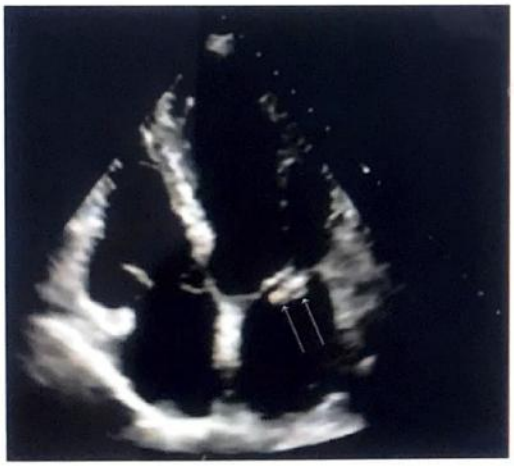
- **Preexisting valvular lesion. unexplained fever with palpable spleen**



MODIFIED DUKES CRITERIA

00:18:14

Major Criteria



1. **Echocardiography:**
   - **vegetations +**
   - Valve abscess / ring abscess
   - Perforation
   - Oscillating intracardiac mass on valve
   - Partial dehiscence of prosthetic valve
2. **Blood culture**

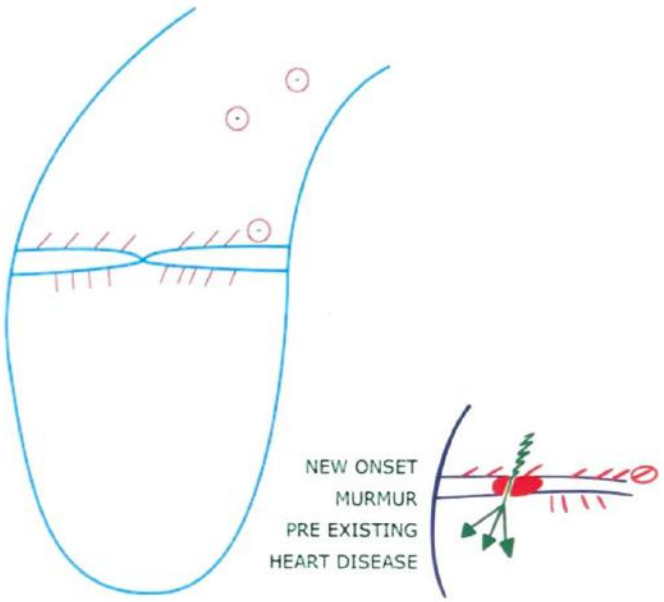
- 3 sets of 2 bottles collect at the interval of 2 hour
- Atleast 2 samples should be positive should be from same organism (sample should be taken from different sites)
- If 1+/3: Coxiella burnetii = causes Q Fever (sheep/goat/cattle)

3. **New onset murmur in pre-existing heart disease**

MINOR CRITERIA

00:27:27

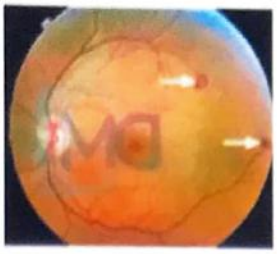
1) **Fever** > 38degree Celsius (101°F) on 2 occasion
2) **Predisposing condition**
   - IV drug abuser
3) **Immunological criteria**



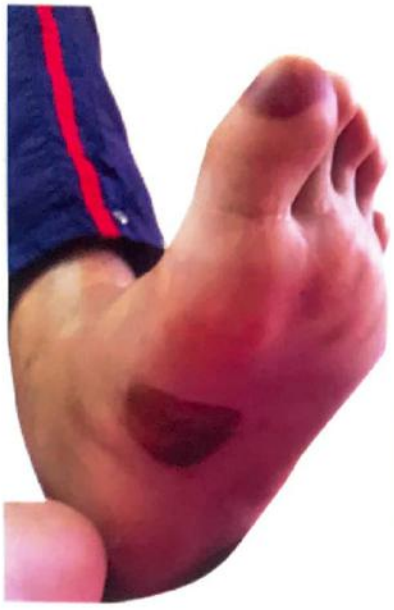
- **R**: **R**oth Spots (white spots in fundus)
- **O**: **O**sler nodes (Pea sized nodules in tip of fingers) toes(tip of fingers, toes)
- **G**: PSGN : Hematuria(Rare manifestations)

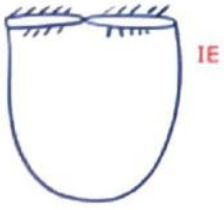
How to remember

- ROG

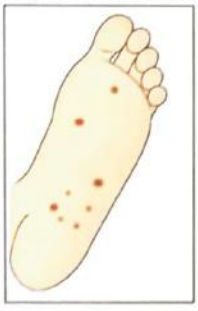


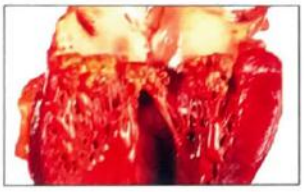
4) **Vascular phenomenon**
- Arterial emboli
- Mycotic aneurysm: S. Aureus
- SAH
- Conjunctival hemorrhage, septic pulmonary infarction
- Janeway lesion





- Erythematous macules in palms / soles





DIAGNOSTIC ALGORITHM

00:34:20

EXTRA MILE
- Roth spots can also seen in
  - Lymphoma
  - Leukaemia
  - Anaemia
  - IE

For diagnosis of IE, require
- 2 major criteria
  (or)
- 1 major criteria + 3 minor criteria
  (or)
- 5 minor criteria

Important Information

- **Duke criteria: IE**
- **Duke Score: ch.stable angina severity**
- **Duke Staging: Ca rectum**

- Non Bacterial thrombotic endocarditis is example of sterile endocarditis also referred as Marantic Endocarditis. Lesions are present on lateral surface of valve

Causes: Malignancy { GIT, Ovary ; M3 AML

TREATMENT

00:36:21

Pen G Ampicillin ⊕ Gentamycin ± Methicillin 00:36:24
- MRSA → Vancomycin
- VRSA → daptomycin
- After 72 hours, culture sensitivity report available, depending upon report
- After treatment, patient will be afebrile within 7 days
- If still febrile after 7 days
- There might be abscess formation
- Surgical intervention may be required
  - **Culture negative endocarditis**
    1. Technical considerations
    2. Fastidious organisms: HACEK
- (Bacteria present in HACEK group)
  - **H-Hemophilus Aprophilus**
  - **A-AGGREGATIBACTER species**
  - **C-Cardiobacterium Hominis**
  - **E-Eikinella Corrodens**
  - **K-Kingella Kingellae**

How to remember

- HACEK

Rx: Ceftriaxone (3rd generation cephalosporin)

LIBMAN SACKS ENDOCARDITIS (STERILE ENDOCARDITIS)

00:42:14

- Predilection under surface of valve
- Damages chordae tendinae
- Valvular lesion (M/C-Mitral Regurgitation)

Rx: Valvuloplasty

Sterile vegetations

1. **RHD** – Tip of cusp which is called as commissural end involvement
2. **NBTE (Non bacterial thrombotic endocarditis)** (Marantic endocarditis) – Line of closure due to Malignancy
   - AML-M3 (Acute Promyelocytic Leukemia)
   - Ca ovary
3. **LSE** – Undersurface of valve due to SLE

ANTIBIOTIC PROPHYLAXIS

00:50:03

High risk cardiac lesions , prophlaxis is required before dental procedure

1. Prosthetic valve
2. Prior endocarditis
3. Unrepaired congenital cyanotic heart disease (CCHD)
4. Completely repaired CCHD <6 mths
5. Incomplete repair CCHD
6. Valvulopathy after cadiac transplantation

Management

- Amoxicillin 2 grams, Azithromycin 500mg , I hour prior to procedure

Previous Year's Questions

Q. A female patient with malar rashes and fever was found to have vegetations on either side of the heart valves. What can be the diagnosis of the patient? (FMGE June 2021)

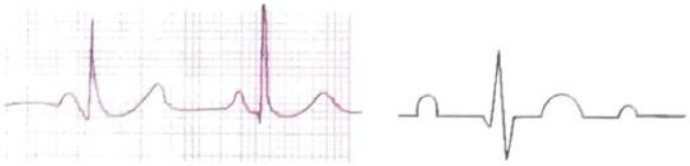
A. Infective endocarditis
B. Libman sacks endocarditis
C. RH
D. NBTE

17 ECG AND ARRHYTHMIAS PART-1

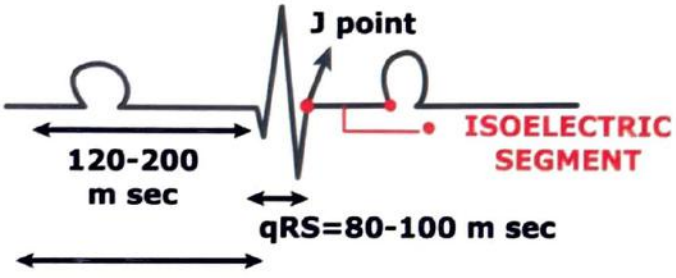
NORMAL ECG

00:00:13

- **Duration of 1 small square 40 msec**
- **P wave** = Atrial depolarization (Duration < 120 m sec) (3 small squares
- Vertical height of P wave



  - < 2.5 mm limb leads
  - < 1.5 mm chest leads
- **P- pulmonale**
  - >2.5mm vertical height
- **PR** = AV Nodal conduction
  - Always inversely related to heart rate
  - (Normal duration 120 - 200 m sec: 3-5 small squares)
- **q wave** = septal indication in V5 & V6
  - < 40 m sec (1 small square)
  - < 1mm depth
  - Depth of q = < 25% R wave
- **QRS (ventricular depolrization):** + 80-100 m sec (2-2.5 small squares)



- **QT**
  - Normal duration: 360-440msec (9-11 small squares)
  - Inverse relation to electrolyte imbalance
- **QT interval (Vent. Depol. + Vent. Repol.)**
- **ST Segment (end of S and start of T)**
  - isoelectric segment
- **T waves** indicates ventricular repolarization
  - Height of T wave

  → < 5 mm: limb leads

  → < 10 mm: Chest leads
- **U wave**: Delayed repolarization of papillary muscle

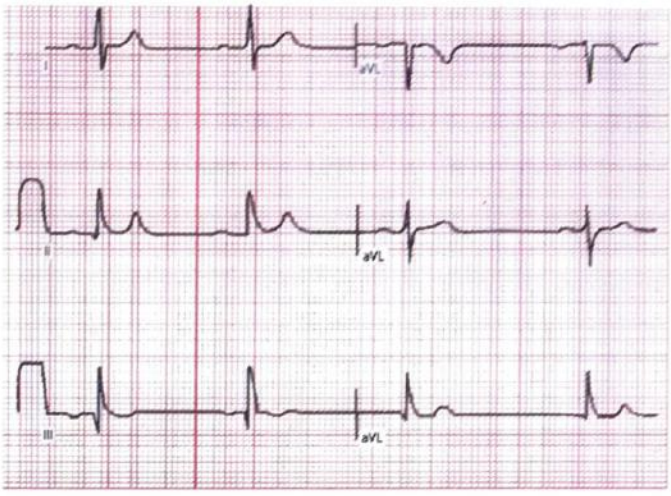
SUMMARY

- P = 3 small squares (ss)
- PR: 3-5 small squares
- Q: 1 ss
- QRS: 2-2.5 s
- QT interval: 9-11 ss

CALCULATION OF HEART RATE

00:14:19

- HR always calculated Lead II
- Count Large squares between R-R waves
- Apply formula: 300/R-R interval



- 4 Large Squares: 75 / min
- 5 Large Squares: 60/min
- 6 Large Squares: 50/min
- 7 Large Squares: 40 / min
- **< 3 Large Squares: > 100 / min:** Tachycardia
- **5 Large Squares: <60/min:** Bradycardia

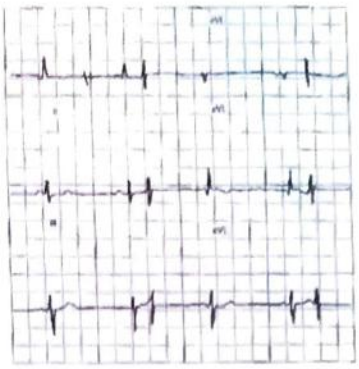
Important Information

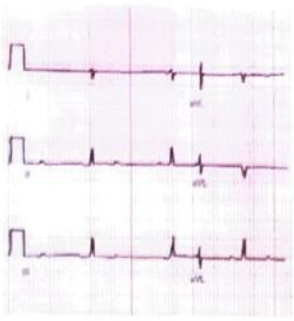
- Irreregulary Irregular heart rate
  - If P wave is absent: A. Fib
  - If P wave is present: Multifocal tachycardia

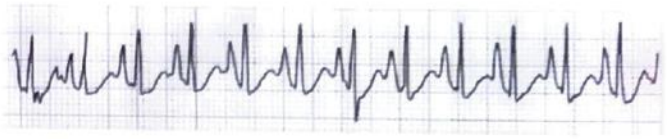
AXIS CALCULATION

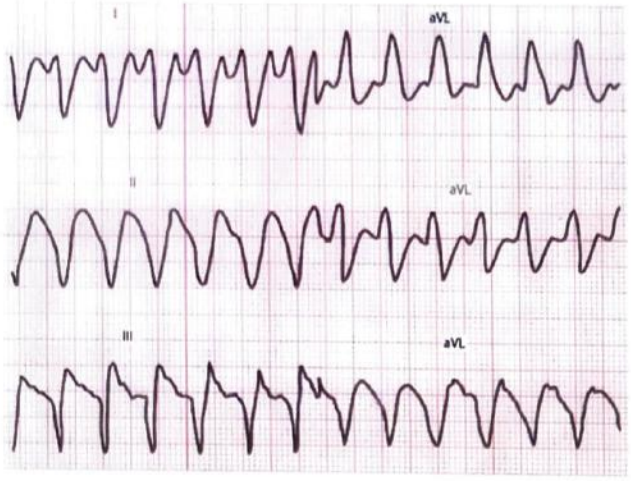
00:19:29

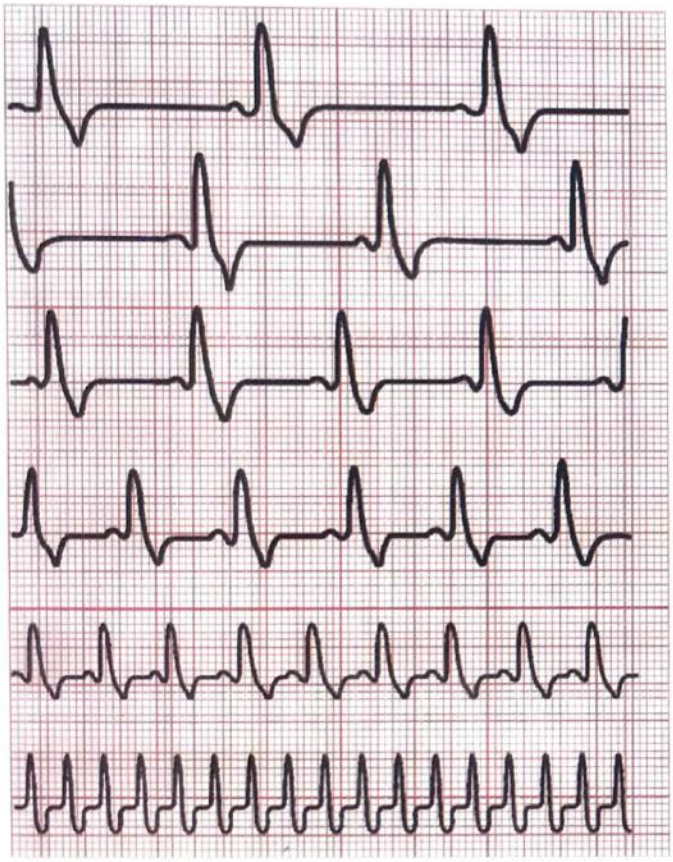
- Normal axis of heart = -30° to + 110°
  - Left axis deviation (LAD): Lt side +
  - RAD: Rt side +

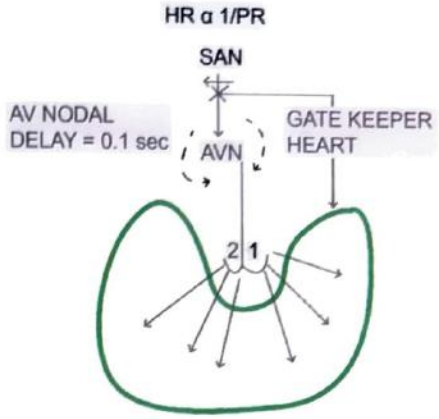










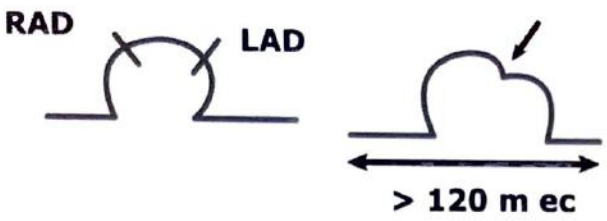


Lead I	R- S: +ve (Height of R - depth of S)	R-S: -ve	R-S: +ve	R-S: -ve
aVF	R-S: +ve (Height of R - depth of S)	R-S: -ve	R-S: -ve	R-S: +ve
	Normal	Extreme axis deviation	Left axis deviation	Right axis deviatio

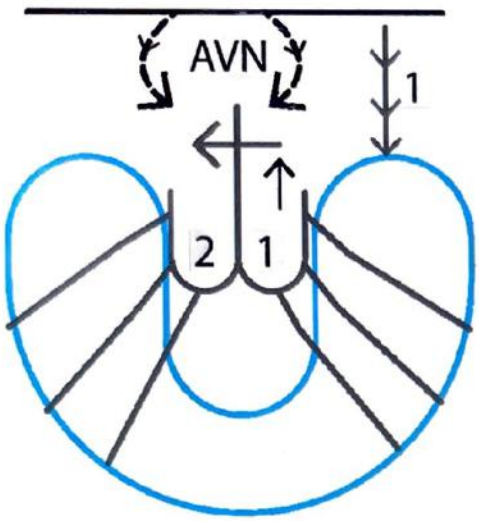
P WAVE & PR INTERVAL ABNORMALITIES

00:28:45

- Normally P wave is upright in all leads
- <120 msec
- <2.5mm vertical height
- P wave is +ve everywhere except aVR
- But is always inverted / negative in leads aVR and always biphasic in lead V1.
- P-mitrale→ LA enlargement > 120 msec

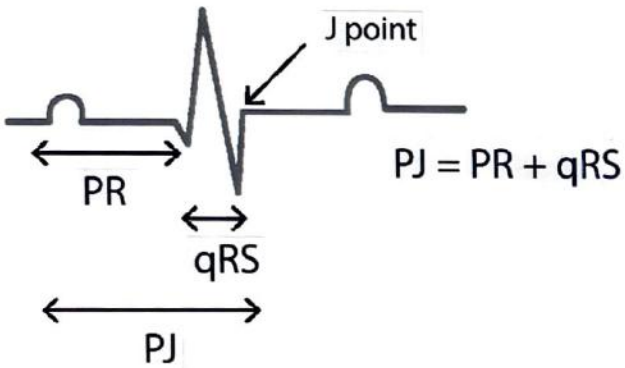


- Once pulmonary artery hypertension occurs then height of P-wave also increases and is called as p – pulmonale.



SHORT PR INTERVAL

00:33:16

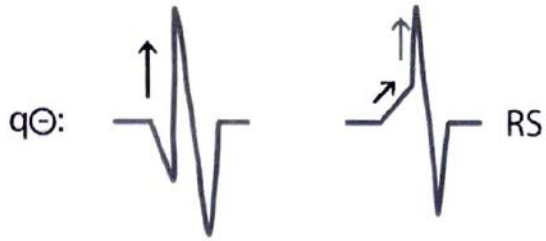


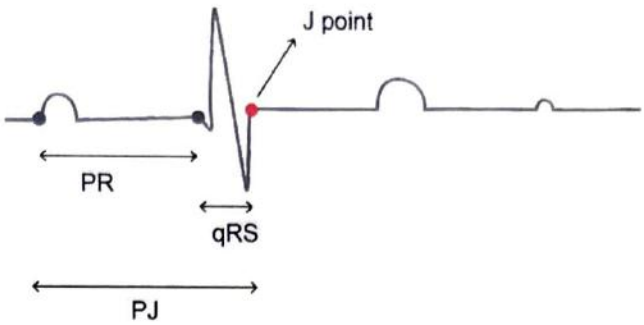
- Wolf Parkinson White Syndrome
- Bundle of Kent (by pass AV node)
- Pre-excitation syndrome
- Low cardiac output
- Contraction of partially filled ventricles

Clinical Scenerio

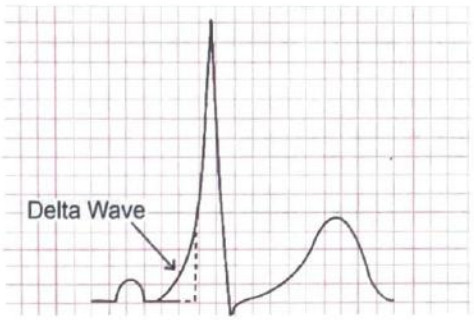
- 25 yr old male complaining of palpitations, dizziness, vertigo, syncopal attacks (Recurrent), Family H/o sudden cardiac death in siblings

Investigation: ECG

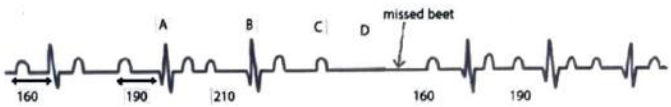


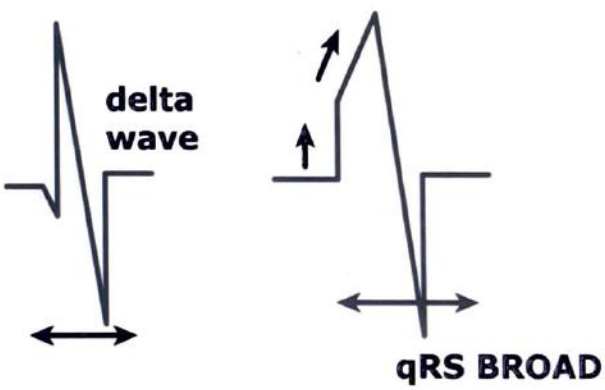


1. **(Fast conduction: Kent)**: Short P-R interval



2. **Intermyocyte conduction (Q absent RS broader)**





3. **PJ** = Normal/ unaffected (PR Reduced + Q absent and RS broad)

- These pts. are medically unfit for army, pilot, police or jobs that require good fitness levels

Treatment

- **DOC:** Oral flecainide to prevent future episodes
- **TOC:** Radio frequency ablation
- **Emergent Management:** IV Procainamide (accessory pathway mediated tachycardia)

LOWN - GANONG LEVINE SYNDROME (LGL)

00:48:04

- Pre excitation syndrome
- Low cardiac output
- C/o Palpitation, dizziness, syncopal attacks: Recurrent
- Family H/o: S.C.D in sibling

On ECG

- Short P-R interval
- QRS complex n
- PJ = PR (short) + QRS(N) = PJ less
  - Pt. Unfit for locomotive driver, commercial pilot, army, police

Treatment

- **AP mediated Tachycardia:** IV Procainamide
- **DOC:** Oral flecainide to prevent future episodes
- **TOC:** Radiofrequency ablation

WPW	L.G.L
BUNDLE OF KENT	JAMES BUNDLE/ MAHAIM FIBERS
Palpitations, SCD in sibling	Syncopal attacks (R)
Short P-R interval	
Delta wave PJ Normal	PJ short

- Oral Flecainide + Radiofrequency Ablation (TOC)

Important Information

- Delta waves in EEG: NREM stage 3
- Delta waves in ECG: WPW syndrome
- Pseudo P-pulmonale: hypokalemia. P>2.5mm in absence of PAH

Previous Year's Questions

Q. Pseudo p pulmonale is seen in? (NEET Jan 2018)

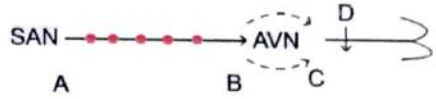
A. Hypokalemia
B. Hyperkalemia
C. Hypomagnesemia
D. Hypercalcemia

PROLONGED PR (BRADYCARDIA)

00:57:40

1st Degree Heart Block

1. Athletes (High vagal tone)
2. Rheumatic fever: Aschoff
3. Hemochromatosis
4. Sarcoidosis
5. Endomyocardial fibroelastosis



- C/o exercise intolerance

ECG

- No change in PR interval with exercise
- PR = 160 millisec
- 5 sit ups: HR ↑
- PR = 140 millisec
- PR interval is always inversely related to heart rate
- PR prolonged > 200 millisec not changed with exercise

Treatment

- Atropine: oral, if can tolerate adverse effects

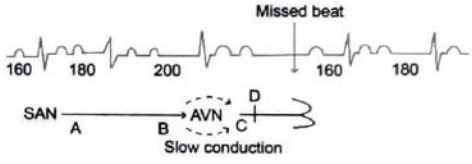
2ND DEGREE HEART BLOCK

01:04:45

- **MOBITZ I/ Wenckebach phenomenon:** Serial prolongation of PR interval

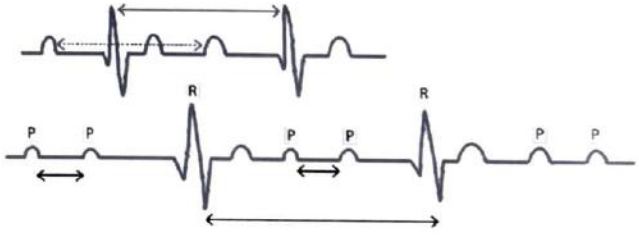
[PR ↑↑↑, P wave QRS Θ, PR ↑↑↑,]

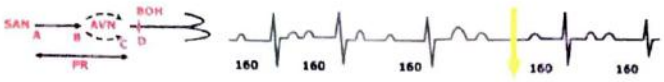
Problem in AV node because of PR ↑



MOBITZ II

- Problem in BoH (bundle of His)
- No PR interval problem
- Infra nodal





- Life threatening
- M/C bradycardia seen post MI
- Can contribute to sudden cardiac death overall
- M/C arrhythmia seen post-MI = V. Fibrillation

Previous Year's Questions

Q. Mobitz II heart block is seen with all except? (NEET Jan 2018)
A. Hypothyroidism
B. Coronary artery disease
C. Sarcoidosis
D. Cushing syndrome

3rd Degree Heart Block/ Stokes Adams Syndrome

01:14:46

- No conduction by AV Node
- SA Node → A. rate = 100/min
- BoH → V. Rate = < 40/min
  - This is called AV dissociation (lack of coordination)

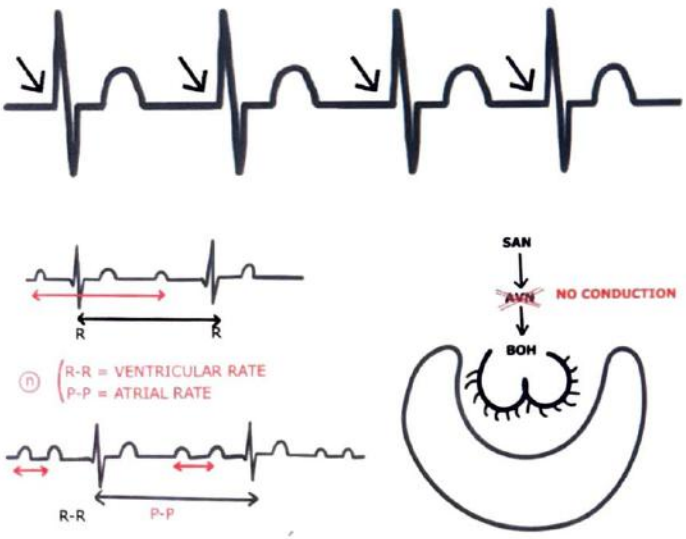
Cardiac output is drastically reduced due to

1. Bradycardia
2. Lack of cooperation between atria and ventricles

- C/o recurrent syncopal attacks
- When pt. stands from supine position (postural hypotension)

ECG: Stokes adam syndrome

- P interval and R-R interval never match
- QRS complex will be broad
- No of P waves will not match with no. of R waves



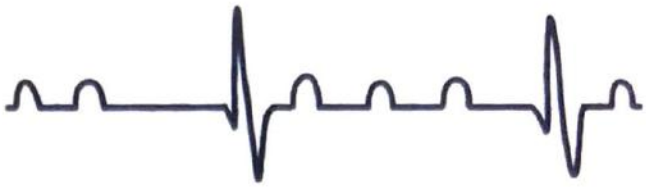
Rx: Permanent pacemaker (Dual pacing)

PACEMAKER USED IN

01:24:18

Heart Rate

- **SAN**: sick sinus syndrome (45-60/min)



  - Absent p wave
- AVN: complete H. block (< 40/min)
  - AV dissociation
- BoH: Mobitz II H. block (15-20/min)

  - Most Dangerous
  - No ventricular activity
  - Sudden cardiac death
  - Temporary pacing

Treatment for suddenly dropping bradycardia

- Atropine: 1mg

SITES OF PACEMAKER'S PLACE MENT

01:32:43

- Left side: Infraclavicular below skin of chest
- Pacing leads: Radio – opaque and can be seen in CXR

LEAD CONNECTS

- 1950 Single lead pacemaker: RV
- Dual lead pacemaker: RA, RV
- Triple lead pacemaker: RA, RV, LV

IMPLANTABLE CARDIOVERTER DEFIBRILLATOR (FUNCTIONS)

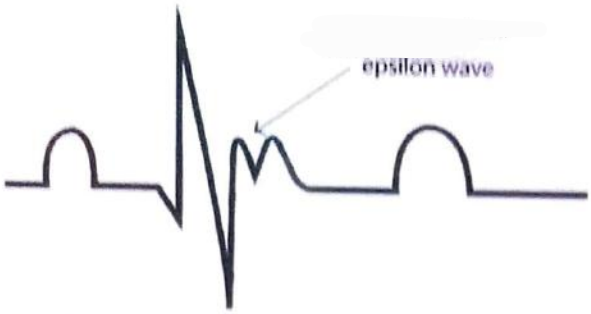
01:37:34

- Read ECG
- Analyse ECG
- Abnormal rhythm detection
- DC shock delivery

INDICATIONS OF USING I.C.D

01:38:30

- Tachy arrhythmias → Tendency
  - **A** - LV Aneurysm, **A**rrhythmogenic RV dysplasia (ARVD), (epsilon wave)



- B - Brugada syndrome: Na^+ channel defect (SCN5A) # in
  - → RV epicardium
  - → Asymptomatic: ST ↑ (cove pattern), (V1 V2) w/o chest pain
  - → Leading cause of sudden nocturnal death in S. E. Asian males
- C - Cardiomyopathy of any etiology (R.C.M / D.C.M/ HOCM)
  - → IV aneurysm

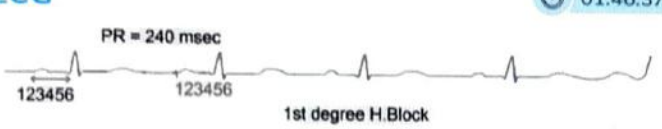
How to remember

- ABC

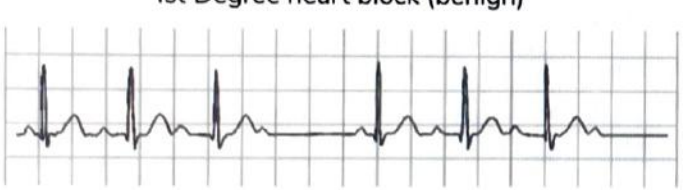
- Pt. on ICD, C/o electrical shocks in chest first investigation is
- Chest x ray.
  - Lead malposition/Lead fracture
  - SVC thrombosis : Facial plethora
- Pt. on ICD, facial swelling, ↑ supine position: probable diagnosis
  - SVC thrombosis due to leads of ICD

ECG

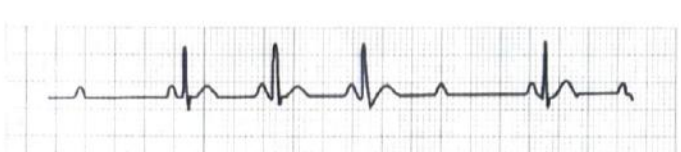
01:46:37



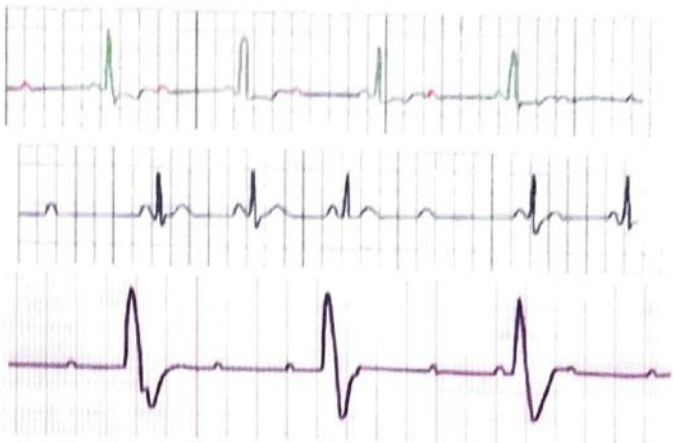
Ist Degree heart block (benign)



Mobitz I H. block



Mobitz II heart block



Complete H. block

2:1 heart block

PR Interval	
Short PR	Prolonged PR
• WPW	• 1st Degree H. block
• LGL	• Mobitz I H. block
• Tachycardia	• Complete H. block

- **Q wave**
  - Best seen in V_5, V_6 leads

- **Deep Q wave**
  - MI
  - HOCM
  - $S_1 Q_3 T_3$(Acute cor pulmonale)
- Deep S: Lead I
- Deep Q: Lead III
- Inverted T: Lead III

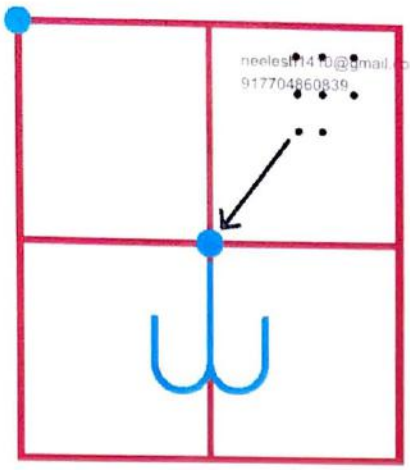
Abnormalities of QRS complex

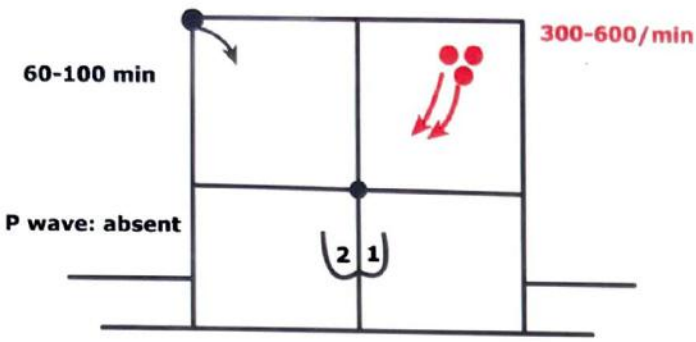
- Narrow QRS: SVT
- Wide Q RS: VT

ATRIAL FIBRILLATION

01:59:00

- M/C arrhythmia in clinical practice
- M/C arrhythmia in HTN
- M/C arrhythmia in geriatric population
- M/C arrhythmia in DCM (alcohol)
- P wave absent in atrial fibrillation





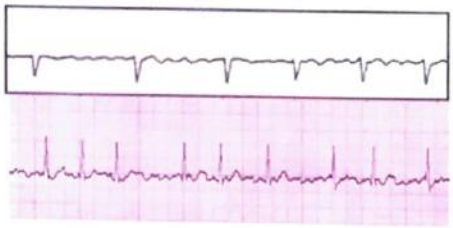
A:V nodal block	V. Rate
2:1	200
3:1	133
4:1	100

- Irregularly irregular heart rate
- M/C cause of embolic stroke : Non Rheumatic A. fibrillation
- M/C cause of sustained arrhythmia in HTN, age > 65y: HOCM

CLINICAL SCENARIO

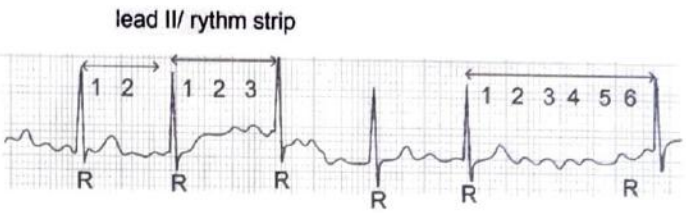
02:08:14

- 40 years old alcoholic (DCM) binge drinking: Palpitation / dizziness / vertigo syncopal attack
- O/E: Pulse: Fast
  - Rhythm: Irregularly irregular
  - Auscultate
    - → Count H. rate
    - → Count pulse rate
- When H. rate & pulse rate do not match it is known as Pulse deficit
  - Pulse deficit: > 10 beats / min
  - BP: 70 / 50 mm Hg



Investigation: ECG

- P wave absent
- R - R:↓
- R - R: Irregular
- QRS: ↓/Ⓝ



Rx:

1. **R-R**ate Control
   - IV Esomolol/ IV verapamil
2. **A-A**nticoagulation
   - Oral dabigatron (thrombin inhibitor) , Rivoroxaban (factor X inhibitor)
3. **C**-Rhythm Control
   - IV Ibutilide/Amiodrone
   - Chemical cardioversion
4. **E**-Electrical cardioversion: DC Shock 200 J biphasic
   - To prevent (1°) atrial fibrillation episodes
     - Oral amiodarone
     - Ablation catheter
   - If pt. have CHF + A. Fibrillation
     - Digoxin (DOC)
     - blocker: **Esmolol:** Contraindicated

How to remember

- RACE

Decision for anticoagulation

- T.E.E
- CHA

Risk Factor	Score
Congestive HF	1
Hypertension	1
Age 75	2

Disbetes mellitus	1
Stroke, TIA, or TE	2
Vascular disease (prior MI, PAD, or CABG)	1
Age 65-75	1
Sex Category (Female)	1

- Clots 2 mm cannot be visualized on TEE

CHA_2DS_2 VASc SCORE

- DM
- CHF
- HTN
- Age > 75 yr
- Stroke history
- Vascular disease: PAD / CAD / UA
- Age: 65 - 75 years
- Sex category: female

Treatment

- Rivaroxaban
- Warfarin (ICH risk); Warfarin not given nowadays

> **Important Information**
>
> - Only Scenerio when A. fib degerates into V. Fib because of Accessory pathway (bundle of kent). which has no decremental response

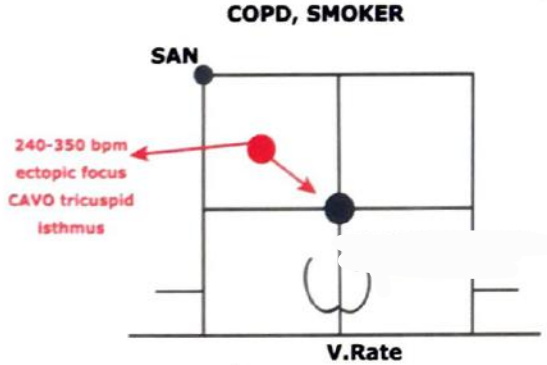
ATRIAL FLUTTER

02:27:50

- Macro reentrant circuit in RA
- COPD smoker pt. will have sudden onset of palpitations, dizziness, loss of consciousness

A.FLUTTER

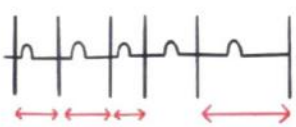
COPD, SMOKER



A. fibrillation	A. flutter
Multiple ectopic • Foci on L. atria • 300 - 600 /min	Macro-reentrant circuit • R.A: Cavo tricuspid isthmus • 240 - 350 / min
AV nodal block 4: 1	AV nodal block 2: 1
H. Rate 150 /min	H. Rate 175/min
Alcoholic	COPD

Palpitation, loss of consciousness
SBP ↓↓↓ pulse fast

ECG
- All leads: Irregular R-R interval



ECG
- Rt. sided leads : II, III, aVF

O/E

- Pulse: 150 / min
- Rhythm = regular
- BP = 80 / 60
- **ECG**: Lead II, III, aVF: Ectopic focus: Right sided leads
- Saw tooth pattern

- R - R ↓, regular

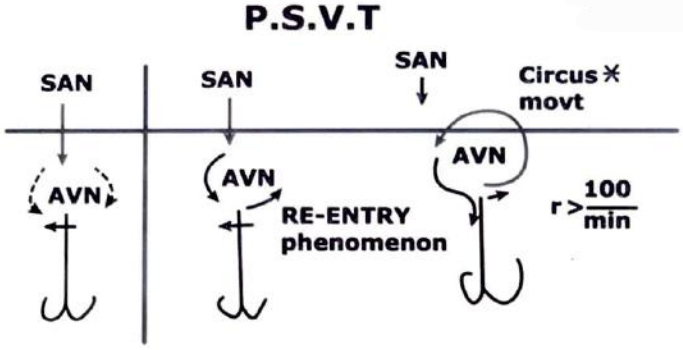
Rx

- IV esmolol (Rate control)/ IV verapamil
- Anticoagulation: 1° pulmonary Embolism
- Rhythm control
  - IV Ibutilide
  - ↓ if fails
- Electrical cardioversion: 25 - 50 J (Biphasic)

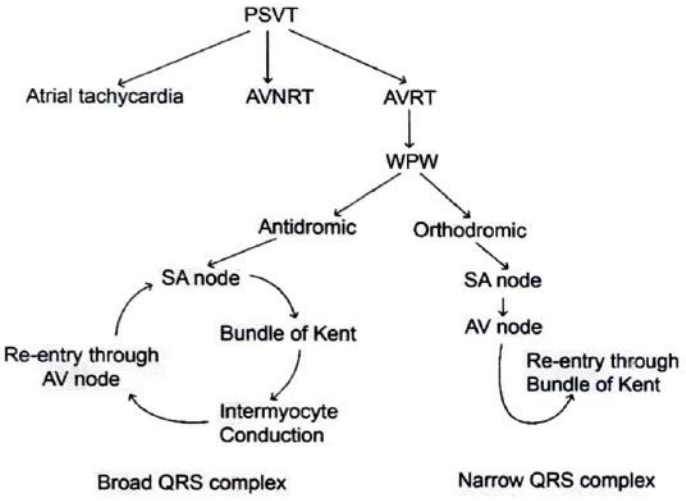
18 ECG AND ARRHYTHMIAS PART-2

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

00:00:13



AV nodal Re-entrant tachycardia (A.V.N.R.T)



CLINICAL SCENERIO

00:06:06

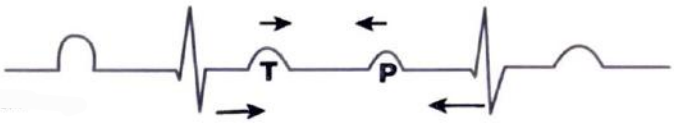
- 25 years old lady with recurrent episodes of palpitations, dizziness and recurrent syncopal attacks

Diagnosis

- ECG : Normal when patient is asymptomatic
- HOLTER advised: device records ECG for 24 hours

Findings

- Narrow QRS complex (HR > 150-200)



- ST segment ↓ (ST segment depression)
- R-R interval decreased
- Narrow QRS
- Hidden P wave

Management

- Rx

Acute	Primary episodes (PSVT)

• SBP <90: DC shock
• SBP>90
  ○ **Carotid sinus massage**
  ○ (Carotid sinus nerve-ix) Efferent close down re entry circuit current
  ○ Given with thumb
  ○ **Site:** perpendicular to angle of jaw, burst of 10 seconds ,given at one side intermittently
  ○ If given continuously, will cause desensitization of receptors
	• Oral verapamil (Prevention)
	• Catheter ablation

Important Information

- Carotid sinus massage is C/I in atherosclerosis patient because of risk of cerebral embolism
- Carotid Artery Bruit is ausculatory finding of atherosclerosis of internal carotid artery

CHILDREN

00:20:10

PSVT Treatment

1. Face ice pack
2. Valsalva maneuver
3. Oculocardiac massage (not recommended)

IV adenosine (chemical cardioversion)

- 6 mg / 12 mg
- A-V nodal delay ↑
- Circus movement ↓
- S/E: Bronchospasm (Self-limiting)

Synchronized DC Shock (Electrical Cardioversion)

- 120-200 joules biphasic

AVNRT

1. Acute episode SBP>90
- Carotid sinus message
- Adenosine
- Syn. DC shock

2. Crashing pt.
- Syn. DC shock (120-200 JB)

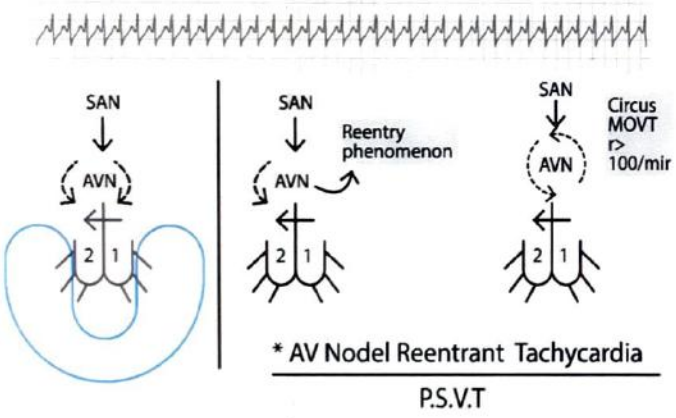
3. Prevention of episode of AVNRT
- EPS (electro physioogical studies)
- Ablation of circuit
- Verapamil

Important Information

- **Remember that in any MCQ of PSVT where SBP is less than 90 mmHg cardioversion is always to be marked**

AVRT (ATRIO VENTRICULAR REENTERANT TACHYCARDIA)

00:28:20



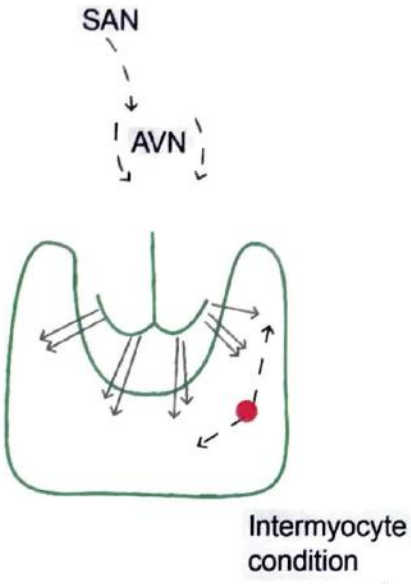
* AV Nodel Reentrant Tachycardia

P.S.V.T

VENTRICULAR TACHYCARDIA (VT)

00:34:49

- Ectopic foci in the ventricle starts firing resulting in intermyocyte conduction
  - Broad QRS complex
- Atrial rate will be regulated by SA node
  - So, Atrial rate is 100/min
  - Ventricular rate > 250/ min
  - A-V dissociation

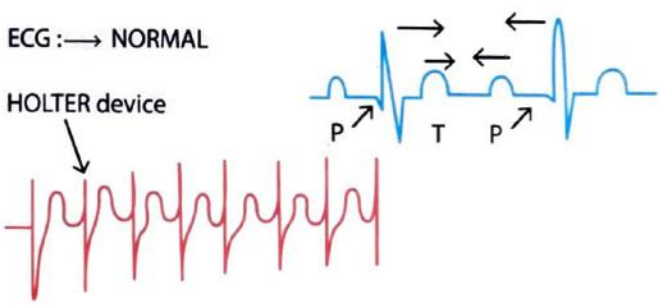


Causes of Low BP in VT

- Lack of Atrioventricular Coordination
  - ↓ Cardiac output: ↓ BP
- Pathological tachycardia
  - Diastolic dysfunction (VR > 250/min)
  - ↓ EDV ↓ SV → ↓ BP

ECG features of VT

- PVC > 3 consecutive contractions
- Broad QRS complex tachycardia
- HR > 100 / min
- Josephson sign: notch in downsloping S-wave
- Capture Beat: Ⓝ sinus rhythm in VT
- Asymmetrical Rabbit ear appearance
- Sinusoidal appearance
- Extreme axis deviation



Rx

- Pulseless VT
  - Non-synchronized DC shock

DC shock 200J biphasic

Synchronized	Non synchronized
(Shock impulse delivery at peak of Rwave) • Given in all Tachyarrhythmias	Given in • Ventricular fibrillation • Pulseless VT

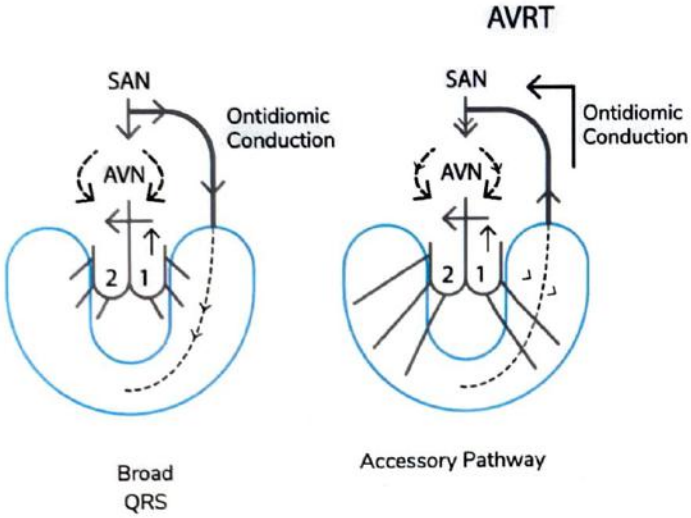
- Synchronized DC Shock implies impulse delivery at peak of R wave

b) Stable Monomorphic VT

- IV Amiodarone / Procainamide

POLYMORPHIC VT (KNOWN BY LATIN NAME TORSADES DE POINTES)

00:50:55



- Prolongation of QT interval Change accessory in figure

Causes

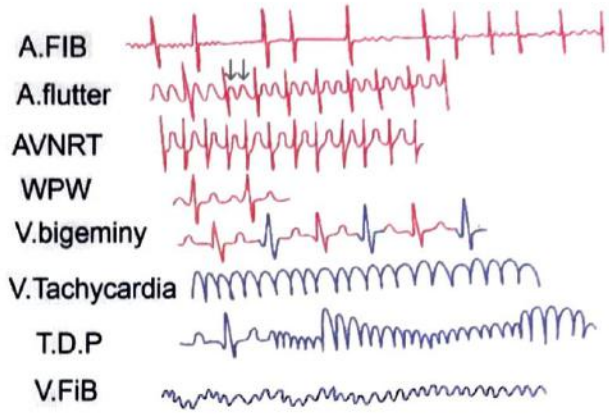
- Hypokalemia
- Mg ↓Hypocalcemia
- Erythromycin +HCQ
- Cisapride
- Astemizole
- Ketoconazole
- Class IA, IC, III, anti-arrhythmics (If Used too much)

Diagnosis

- ECG criteria - QTC ↑ Prolongation
  - Where, QTC = QT / $\sqrt{R-R}$
- DOC for TDP - Magnesium SO_4 IV
- TDP + BP ↓↓ - DC shock

TACHYARRHYTHMIAS

01:00:00



Refer Table 18.1

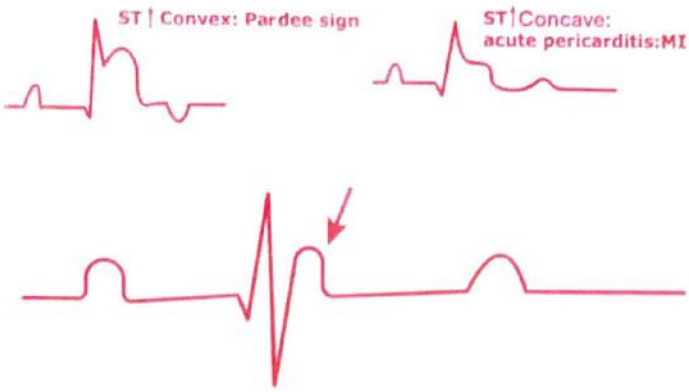
ABNORMALITIES OF ST SEGMENT

01:14:56

- ST Segment Elevation
  - **E** - **E**lectrolyte imbalance: Hyperkalemia
  - **L** - **L**BBB
  - **E** - **E**arly repolarization
  - **VA** - **V**entricular **A**neurysm
  - **T** – **T**rauma: Pericardiocentesis
  - **I** - **MI**: ST ↑ convex - pardee sign (MI)/ tomb stone
  - **O** - **O**sbourne wave/ J wave
  - **N** - **N**on occlusive vasospasm: Prinzmetal angina
  - **A** - **A**cute Pericarditis (concave upwards)
  - **B** - **B**rugada syndrome: SCN5A

How to remember

- ELEVATION-AB



ST SEGMENT DEPRESSION

01:20:24

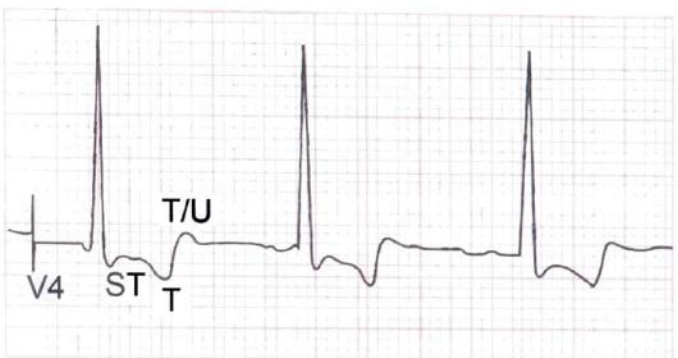
Causes

1. Chronic stable angina / Hibernating Myocardium
- >70% # Blockage of coronary artery
- Chest Pain on Exercise / Emotion
- Cocaine overdose
- Post - prandial angina
- TMT → ST↓ 1 mm 80 m sec: 2 contiguous leads

2. LVH: ↑ demand leading to subendocardial ischemia
- Coronary artery supply – normal
  - HTN, AS, HOCM

3. Normal size Heart, Oxy-Hb availability ↓
- Severe anaemia / cocaine overdosage

4. Digoxin: ST↓ → Hockey stick sign



5. K↓ - Hypokalemia

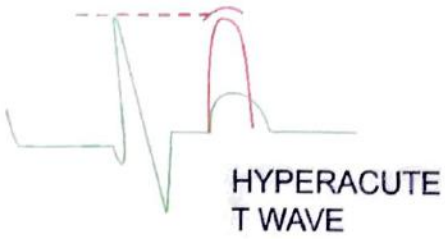
Important Information

Digoxin Toxicity
- NPAT with AV block: most characteristic
  - Rx: Digiband
- V. Bigeminy: MC
  - Rx: Lignocaine

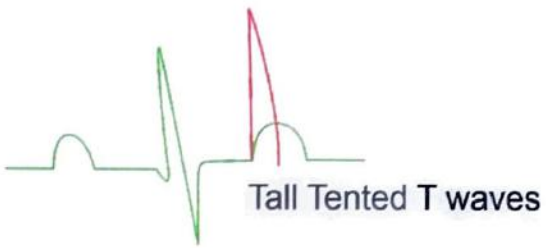
ABNORMALITIES OF QT INTERVAL

01:26:44

- Normal T wave
  - < 5mm limb lead
  - 10mm chest lead



1. Hyperacute T wave: MI (earliest ECG finding)
2. Tall T wave : Hyperkalemia



3. T wave inversion hypokalemia
- MI
- SAH/CVA
- $S_1Q_3T_3$ (acute cor pulmonale)

QT INTERVAL

01:31:00

$$QT\alpha \frac{1}{\text{Calcium, Mg++}}$$

- Normal: 360 - 440 m sec
- 9 - 11 small squares (N)
- Start with Q till end of T wave

Hypercalcemic crisis (Possible systolic arrest) Malignancy
1. Ca breast
2. Squamous Cell Ca lung - PTH – rp (Related Peptide)
3. Sarcoidosis
4. Vit. D3 intoxication
5. parathyroid adenoma

Diagnosis
- ECG → QT shortening

- Ionised Ca++ - ↑
- S. Ca++ - ↑
- S. albumin- Normal

Important Information

- ECG changes with digoxin
  - PR Prolongation
  - ST depression/ Hockey stick sign
  - QT shortening
- T wale always follows potassium levels

Treatment

- IV fluids - Normal saline → Hydration (First line management)
- Furosemide drip (Cause Calcium loss in urine)
  (Do not give furosemide bolus → Diuresis (Dehydration) → can lead to ↑ Ca^{2+} level in blood)
- IV Ibandronate (DOC)
- Calcitonin Nasal Spray (Send Ca^{2+} into Bone)

Tetany

- Thyroid surgery after 72 Hours
- S. Calcium < 7 mg %
- QT prolongation

Clinical features

- Perioral paresthesias
- Inflate BP Cuff - SBP + 20 mm Hg ↓ 3 min
  - Carpopedal spasm
  - Obstetrician Hand

  } Trousseau's sign
- Chvostek sign: Tap on Facial nerve
  - Irritability
  - Facial Grimace
  - Risus sardonicus = Tetanus
- Death: d/t Laryngospasm

Investigations

- ECG - QT ↑
- S. calcium : ↓
- Ionized Ca^{++} : ↓
- S. albumin = n

Treatment of Tetany

- IV calcium gluconate
- But if he will ask: Surgery induced Hypoparathyroidism. Then answer as [Injection Teriparatide (PTH)]

ECG ANALYSIS

01:38:03

- HR: lead II
  - <3LS:>100 Tachycardia
  - >5 LS: <60 Bradycardia
- Axis: LeadI/aVF
- P wave: p pulmonale, p mitrale
- PR
  - short:LGL , WPW
  - Wide: H. block (except Mobitz II)
- QRS
  - Narrow complex tachycardia
  - Wide complex tachycardia
- ST segment elevation/ depression

Table 18.1

Atrial Fibrillation

- Absence of p waves
  Irregular R-R interval

Atrial Flutter

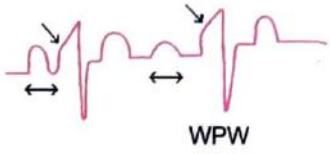
- 2 abnormal waves before every QRS complex
- Two saw tooth waves 3:1 conduction
- Seen in Lead II, III , aVF

AVNRT

- One abnormal wave before QRS complex

WPW

- No Q wave, delta wave present



VT

- Broad QRS complex tachycardia
- Sinusodial rythym

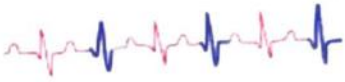
TDP

- Sinusodial pattern will change in amplitude

V. Fibrillation

pulseless

Bigeminy



19 MULTIFOCAL ATRIAL TACHYCARDIA

Introduction 00:00:13

- Transient rhythm between atrial fibrillation and atrial flutter

IRREGULAR R-R INTERVAL CAUSES

- Atrial Fibrillation: Absent P waves
- Multifocal Atrial Tachycardia: Three Different Morphology P waves

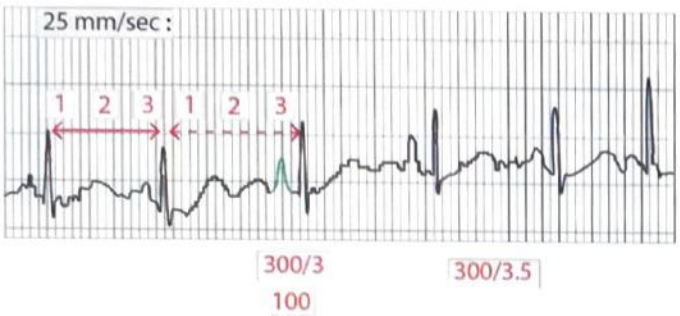
Etiology 00:01:52

- COPD (MC cause)
- Theophylline Toxicity
- Sepsis

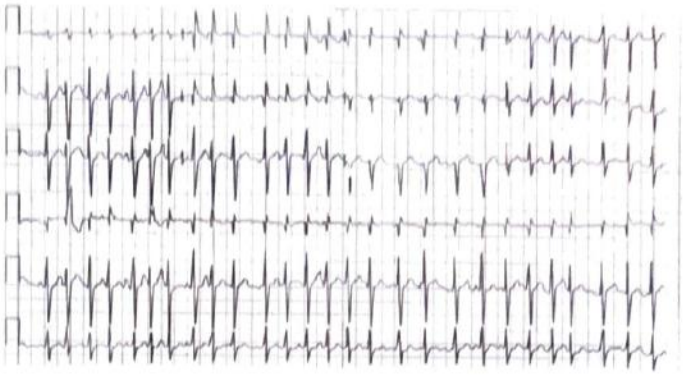
Case 00:02:38

- 65 yr male , smoker , C/o palpitations, dizziness, syncope. O/E : BP unrecordable BP

ECG Features

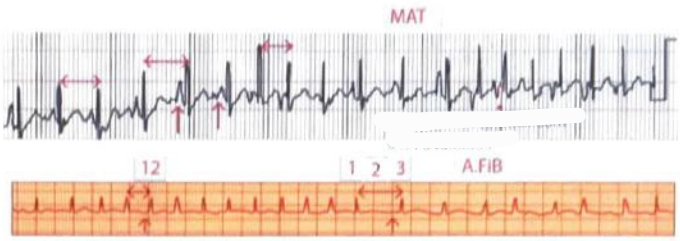


1. R-R interval irregular
2. Variable P wave morphology >3
3. P-R interval → variable
4. Three different amplitudes of P wave
5. RAD
6. Deep S in V_6
7. HR = 100-125/min (Variable)



Treatment 00:10:11

- Manage respiratory failure
- Cardioversion is NOT helpful
- For acute episode- Metoprolol / Verapamil
- For Recurrent episodes – Oral Verapamil



20 ECG CHANGES IN HYPERKALEMIA AND HYPOKALEMIA

T WAVE ABNORMALITY (HYPERKALEMIA)

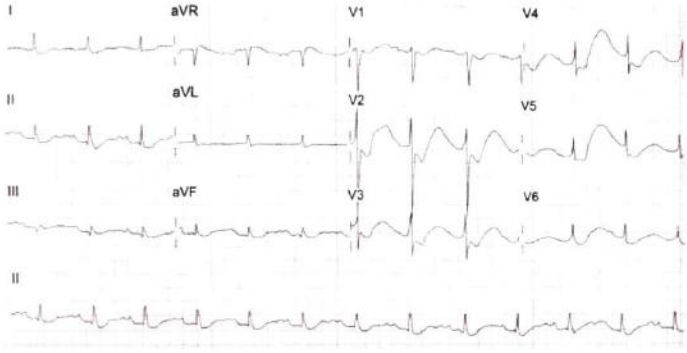
00:00:20

- $K^+ \uparrow \rightarrow$ T wave: repolarization of K^+

1) Tall tented T wave
2) ST elevation
3) P wave: Amplitude/ duration decreases and absent, PR prolonged
4) QRS complex broad
5) Sine wave pattern
6) > 8.0 meq

Important Information

- **Reason for death of the patient is due to Diastolic arrest**



Rx:

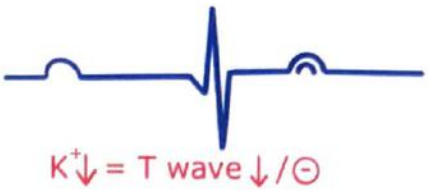
00:05:10

1) IV calcium gluconate /$CaCl_2$: DOC
   - never Calcium Carbonate\
2) (0.5 – 1 meq ↓/ HR): Insulin drip (Regular) } Redistribution of K^+
3) Salbutamol/ nebulization
4) IV furosemide: Kaliuria
5) Hemodialysis: Most effective method
6) K+ binding resin: Enema
   - Ch. Elevated K^+: Patiromer
   - Not used in Emergency management because it takes sometime to show response
   - Not used routinely in $K^+ \uparrow$ management → Soda bicarbonate

HYPOKALAEMIA

00:10:05

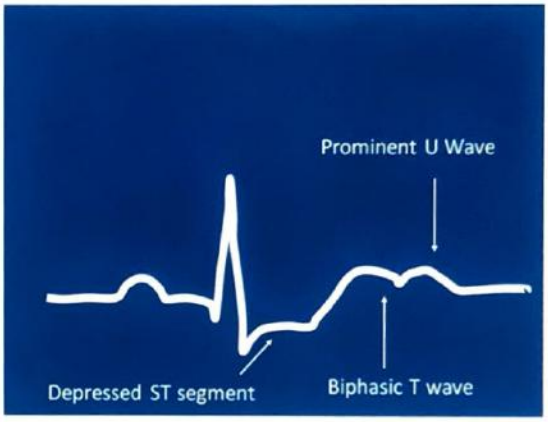
1) T wave decrease, absent or inverted



2)

T wave inversion

3) P wave amplitude increase (Pseudo P- pulmonale)
4) ST ↓
5) Prominent U wave



6) Prolonged QU & PR interval

- **Death** → Respiratory paralysis

Rx

- KCl + IV Fluids
- Peripheral line 20-40 meq/hr

SUMMARY

00:17:49

$K^+ \uparrow$	$K^+ \downarrow$
All tented T	T wave ↓/absent
ST↑	ST↓
P wave absent	Pseudo pulmonale: Prominent U wave
Cal.glucoate	KCl+IV fluids

21 ACUTE CORONARY SYNDROME PART-1

ATHEROSCLEROSIS 00:01:27

- Accelerated atherosclerosis
  - DM
  - Hypothroidism
  - SLE
  - RA

Order of involvement of blood vessels

- Abdominal aorta (MC) > Coronary A > popliteal A (M/C peripheral) > Circle of willis (STROKE)

How to remember

- ACP

Refer Table 21.1

- Least common: Internal mammary artery (Preferred bypass conduit: CABG)

Important Information

- **Acute Coronary Syndrome: Sickle cell anemia**
- **Acute Chest Syndrome: Sickling crisis**

MARKERS FOR ATHEROSCLEROSIS 00:12:04

1. **High sensitivity CRP**: If positive: Predictor for future coronary events
2. $\frac{\text{Total Cholestal}}{\text{HDL}}$ > 3.5 → ↑ risk of atherosclerosis
3. $\frac{\text{Lipoprotein a}}{\text{Apolipoprotein B}}$ = if ratio ↑, risk of coronary artery disease
4. **Homocysteinemia**: Autosomal recessive, risk factor for Premature Atherosclerosis
   - **Management of Homocysteinemia**: Vit. B_6 / B_9 / B_{12}
5. **↓HDL, ↑LDL**
   - ↓HDL is more important to cause heart disease than ↑LDL
   - Exercise
   - Statins
     - Always take at bed time (coz. maximum cholesterol synthesis occur at night)
     - Inform pt. about side effect (myalagia)
   - Niacin (most efficient way to ↑HDL)

FRAMINGHAM CRITERIA FOR CHF 00:22:01

- Calculate risk of heart diseases in an individual
- LDL value should be < 100mg/dL in normal population, but in high risk population (DM / CRR/ patients on hemodylsis) value of LDL should be <70mg/dL

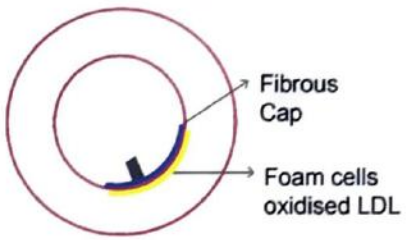
Important Information

Framingham score : 10 yr mortality online calculator

- **LDL >115mg: Low/moderate risk**
- **LDL > 100mg : High risk**
- **LDL > 70mg: Very high risk (DM, CKD, hemo dialysis)**

Imaging for Atherosclerosis

- Oxidised LDL
- Smoking, sedentary lifestyle, lack of exercise
- Fibrosis
- Known as Vulnerable Plaque

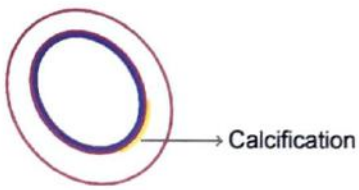


Plaque Fissure

- Foam cells tear → platelets stick → Thrombus formation = macrophages + → OCCLUSION OF LUMEN

Oxidised LDL

- More atherosclerosis
- More fibrosis
- Known as Non-Vulnerable Plaque



Non-vulnerable plaque

IMAGING 00:32:20

- **Multidetector CT scan (MDCT), Intravascular USG**
  - Imaging modality
  - Non-invasive method
  - Differentiates b/w vulnerable & non vulnerable plaque
  - **Calcification**
- Seen in later stages
- **AGATSON Score**: Determine the severity of calcification in Coronary Arteries

CHRONIC STABLE ANGINA / REVERSIBLE ISCHEMIA OR HIBERNATING MYOCARDIUM 00:35:48

Case: 60 yr, smoker, HTN (Non-compliant), was walking fast towards his car. Suddenly develops chest pain which got relieved in few minutes of rest

- Chronic stable Angina

Pain in stable Angina

- Recurrent episodes
- Pain an exercise / emotions
- Diffuse excruciating chest pain
- Relief on rest
- Post prandial angina (chest pain)

> **Important Information**
>
> Post prandial abdominal pain
>
> - Abdominal angina d/t chronic mesenteric insufficiency, narrowing of the mesenteric artery

ECG

- **Reversible Ischemia**
  - **Asymptomatic** → ST normal
  - **Symptomatic**, ST depression
- Sublingual nitroglycerin
  - After relief
  - Repeat ECG (15 min) = ST normal

WORK-UP

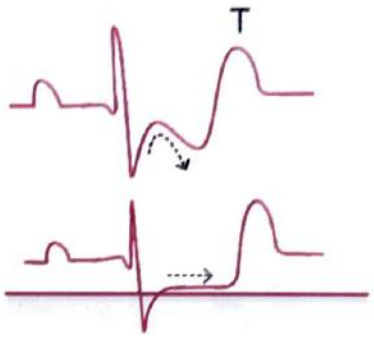
STRESS TEST 00:46:19

1. **Treadmill test** [Bruce Modified Protocol]

- Non invasive test
- Technician will gradually ↑ speed & ↑ inclination (every 3mins interval)
- Continuous ECG and BP monitoring
- Target Hr = 85% of maximum possible physiologically
  = 85% [220 - Age]
- no ECG finding → no blockage
- ECG finding ST ↓ > 1mm > 80 m sec : 2 contiguous leads (Down sloping)

Indicates blockage in coronary arteries

- Resting, ECG after 3 mins of termination of test post exercise ST segment depression



- Indicate BLOCKAGE in coronary Arteries
  - Sensitivity 75%

CONTRAINDICATIONS FOR PERFORMING TREAD MILL TEST (TMT)

1. Morbid obesity (BMi > 40)
2. Severe aortic stenosis <1cm2
   - Fixed obstruction Cardiac Output ↓
3. Non healing ulcer - SOLE
4. DVT (Risk of embolization)
5. Charcot Joints
6. B/L severe osteoarthritis knee
7. Buerger / TAO (Thromboangitis Obliterans)
8. MI < 48 hrs
9. Unstable angina (acute)

Duke score 00:59:31

- No. of minutes walked on the treadmill
  - -(5xST↓)-(4xangina grade)
- ≥5: low risk, medical treatment
- +4to-10: intermediate risk: PCI+ drug eluting stent
- <-11:high risk : CABG

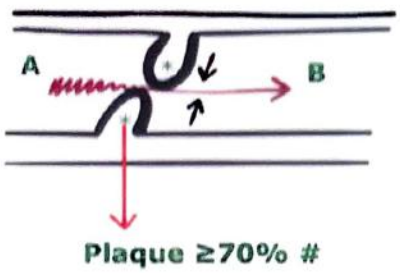
2. Stress echocardiography 01:04:20

- IV Dobutamine: β1 receptor activation, HR ↑↑ O2 consumption
  - **ECG**: ST ↓ > 1mm > 80 m sec: 2 contiguous leads
  - **Echocardiography**: Regional Hypokinesia (Contractility on lower side)
- Sensitivity = 80%

3. Myocardial perfusion imaging (MPI) 01:10:23

i. **Thallium scan**

- Thallium - 201 / contrast given to patient, cardiac scanning done with Gamma camera
- Dobutamine
- Plaque cause luminal obstruction > 70% blockage



ii. Technetium 99 / SESTAMIBI SCAN

- Sensitivity = 90%

Hibernating myocardium V/s scar

- Pet Scan

Sensitivity

- **PET scan** > 95% - Best test to identify viability of Myocardium. It helps to differentiate scar vs ischemic Hibernating myocardium
- Thallium scan = 90%
- Stress Echocardiography = 80%
- TMT = 75% - 1st to be done

GOLD STANDARD INVESTIGATION TO IDENTIFY CORONARY A BLOCKAGES

- Coronary Angiography
- Real time evidence
- % blockage
- Site of blockage
- Number of blockages

Treatment 01:16:37

- DOC : **CSA**
  - β blocker Metoprolol (↓ HR)
  - Mortality ↓ drug = Metoprolol

1. **Tab Aspirin** 75 mg OD (↓ mortality)

(or)

- **Clopidogrel** 75 mg OD
  - New guidelines says asprin should be given on the day of surgery in chronic stable angina as it reduce chances of intraoperative MI (asprin+ β # + statin should be given on the day of surgery)

2.

Nitrates

- Sublingual NTG
- Buccal spray

Emergency

- Transdermal patch
- Tab Isorbide mononitrate (post prandial angina)
- Disadvantage
  - Tolerance develops when used for prolonged period
  - Need drug holiday (8hrs everyday)

3. **Cardiogenic β blockers**

- DOC for chronic stable angina→ Tab metoprolol
  - ↓HR, ↓O_2 consumption
  - ↓ mortality,
  - CI in HR < 60 / min
- Contraindications of βblockers
  - Sick sinus syndrome: SAN #
  - Complete heart block: AVN #
  - Mobitz II heart block: BOH #
  - Acute CHF

4. **Tab Amlodipine**
5. **Tab Rosuvastatin (duration of action 19hr)**

- Atorvastatin (Duration of action 14 hr)
- HMG CoA reductase inhibitors
  - Maximum cholesterol synthesis occurs at night therefore stanin are preferred at night time dose
  - (Bed time dose)

→ Mortality ↓

6. **Smoking Cessation**
7. **Vitamin B6 / 9 / 12 (Homocystinemia)** Best: B6
8. **New drugs**

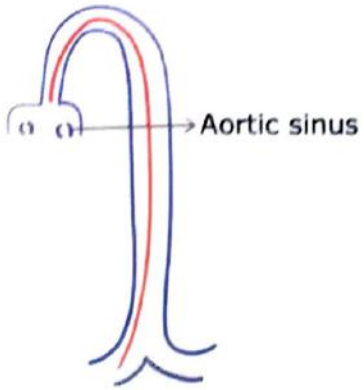
- IVABRADINE: Inhibits funny current
- Ranolazine
- Trimetazidine
- Nicorandil

Important Information

- **Never combined Sildinafil with NTG causedecrease in SBP**
- Mortality reduction in CSA
  - β# >asprin >statin
  - No mortality reduction seen with Nitrates

If Failure of medical therapy

- Coronary angiography (Invasive)
  - Real time evidence
    - → Can tell % of blockage
    - → Site of blockage
    - → Number of blockage



Approach

- Transradial (preferred)
- Trans femoral (Seldinger Technique)
- Under fluoroscopic guidance guidewire is inserted up retrogradilly to route of aorta and then coronary artery
  - First branch of Aorta: Coronary artery
  - First branch of arch of Aorta: Brachiocephalic

Refer Table 21.2 01:33:41

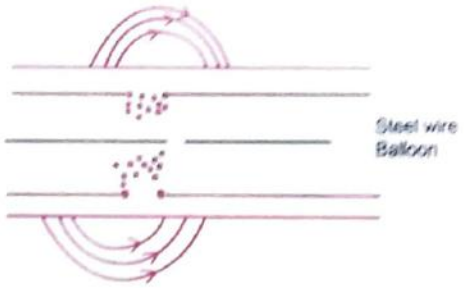
Grafts	Patency
• Internal mammary artery	• 15-20yrs
• Radial artery	• 5yrs
• Great Saphenous vein	• 10-12yrs
• Gastroepiploic vein	• 5yrs

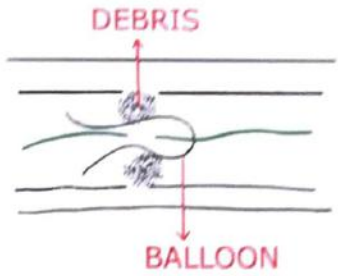
Important Information

- **Arterial graft is always superior to venous graft.**

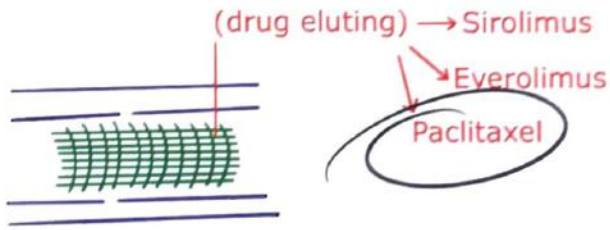
SINGLE VESSEL DISEASE

- **Preoperative management of patient undergoing PCI**
- When guidewire is introduced in blocked vessels
- Ballon inflate and deflate at very high pressure





- Leads to destruction of atherosclerosis plaque
- Debris released and it act as a magnet for platelets
- Platelet plug formation
- **Type 4A MI**
  - ↑ troponin I X 5 times
- To prevent such an event of patient having MI while undergoing P.C.I
- **Pre-op**: Anti-platelets drugs: Ticagrelor/ Prasugel/ Ticlopidine
  - Prevent formation of thrombus
  - **Anti-clotting**: Enoxaparin
- **Intra-op**: Abciximab / Eptifibatide / Tirofiban
- Stenting is done to avoid redevelopment of atherosclerotic lesion
- Drug eluting stent, biodegradable
  - Sirolimus
  - Everolimus
  - Paclitaxel



Important Information

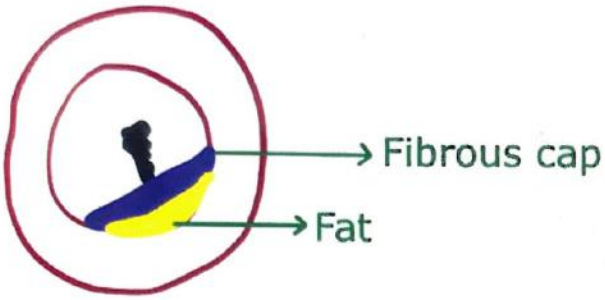
LAD Blockage with ejection fraction < 40%, treat CABG

CSA/ Hibernating myocardium

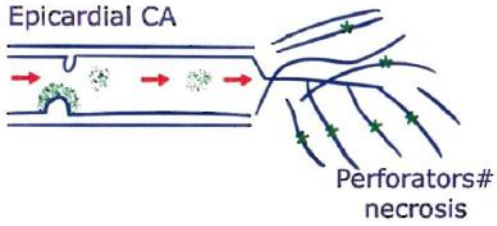
- Trop I : normal
- Ist Invasion: TMT
- Cant walk : stress echo
- Scar V/s hibernating myocardium
  - PET
- Nuclear scan: Sestamibi scan
- DOC: metoprolol
- Revascularization procedure
  - CABG
  - PCI+ stenting
- Gold std.: coronary angiography

UNSTABLE ANGINA

01:58:42



- Plaque fissure
- Causing collagen exposure
- Platelet plug : Thrombus
- Protein C / S / AT III
- Perforators
- Micro occlusions
- Necrosis
- Atheroembolism



Important Information

- **Thrombolysis is absolutely containdicated in unstable angina**

02:04:02

Case: 60 yr male smoker, HTN, diffuse chest pain since morning at rest X 3 hrs

Clinical features

- Chest pain at rest / Levine sign
- Diaphoresis / palpitations
- Dyspnea

O/E

ECG

- ST normal in 50%
- ST ↓ = 25%
- T wave inversion = 25%

Cardiac Marker

- C Troponin I = Normal/ elevation (Mild to moderate) (Normal < 0.04 mg / dl)
- In MI, troponin ↑ x2, x3, x5, x10

TREATMENT

02:17:18

- **A - IV ABCIXIMAB**
- **B - β- blockers: Metoprolol I/**
- **E - Enoxaparin SC**

How to remember

- ABE

- **M - Morphine (2-2.5mg/u)**
- **O - O_2**
- **A - Aspirin (325 mg chewable)**
- **N - NTG drip**

How to remember

- MOAN

- DOC: IV NTG drip > Enoxaparin

- If patient does not improve
- Monitor: SpO_2: ↓↓
- BP : ↓
- Crepitations: ++

↓

Treatment: Delayed percutaneous coronary intervention

PRINZMETAL ANGINA

02:12:02

Normally

- Arginine $\xrightarrow[\text{NOS 3}]{\text{nitric oxide synthase 3}}$ NO (In coronaries)
  - D/t atherosclerosis, there is damage to endothelium leading to ↓function NOS-3 and hence ↓formation of NO
  - leading to vasospastic episodes

Important Information

- **In normal person Ach which helps generation of NO causes vasodilation, but in Prinzmetal angina causes spasm**

- **Case:** Female, 60 yrs, chest pain at rest, 3-4 hours persisting in especially morning hours, diurnal variation of chest pain, multiple attacks and more in winter months
- Past medical history: H/o Raynaud's Phenomenon

Cold H_2O exposure $\xrightarrow{\text{VASO}}$ Constriction of blood vessels of Tip of fingers, Toes → White - blue - Red

- Both Prinzmental Angina and Raynaud Phenomenon are vasospastic disorders and can co-exist in same patient
- **ECG:** ST ↑ (Variant angina) → Only angina with ST. elevation
- **Cardiac Troponin I** = Normal (< 0.04 ng/dl)

Treatment
- IV NTG drip (DOC)
- Tab isosorbide mononitrate + amlodipine (long term treatment)
- Work up for CSA (hibernating myocardium)

MYOCARDIAL INFARCTION

02:25:00

- **ECG findings:** Normal vertical height of T wave
  - < 5 mm: Limb leads
  - < 10 mm: Chest leads
- In MI, T wave will become bigger rounded appearance Height > Breadth

< 5 mm: limb leads
< 10mm: Chest leads

Important Information

- **In MI earliest ECG findings hyperacute tall T wave**

ECG changes in MI

1. Seconds
- Hyperacute T wave (Indicates severe myocardial ischemia)
- T wave inversion

2. Minutes
- 3. ST ↑ elevation - convex in upper direction (Indicates myocardial injury)

- **Pardee sign/ Tomb stone pattern**
- Chest leads
  - ≥ 2mm for males
  - ≥ 1.5 mm for females

3. Hours (> 1 hr)
- Deep Q waves
- INDICATES cell death
- Persists for whole life
- Old MI / previous MI

Important Information

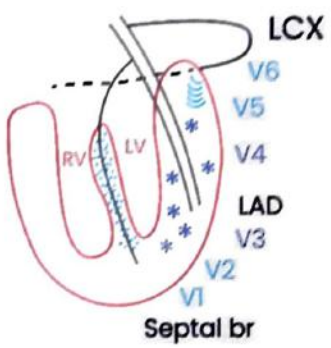
- **If Injury: ST elevation**
- **If Ischemia: hyperacute T wave or T wave inversion**

4. Poor progression V1 of R wave
- R not increasing with normal magnitude
- Non progression of R

LOCALIZATION OF SURFACE I NVOLVED IN MI

02:35:04

- ECG poor at localising right ventricle ischemia and LCX artery ischemic
- V_1 V_2: Septal Ischemia

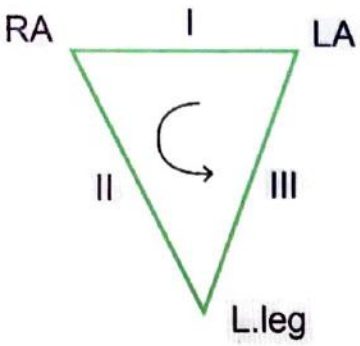


- V_3 V_4: Anterior Wall
- V_5 V_6: Lateral Wall
- RV forms inferior surface of heart
- For RV ischemia use the following leads
  - V4
  - aVF
  - Lead II, III

Chest leads
- V1 - 4th Intercostal space: Rt. parasternal line
- V2 - 4th Intercostal space: Lt. parasternal line
- V3 - V2-V4
- V4 - 5th Intercostal space: Lt. midclavicular
- V5 - 5th Intercostal space: Lt. ant.axillary line
- V6 - 5th Intercostal space: Lt. mid axillary line

- V7 - 5th Intercostal space: Lt. pos. axillary line
- V8 - 5th Intercostal space: Lt. scapular line
- V9 - 5th Intercostal space: Lt. paravertebral line

Limb leads



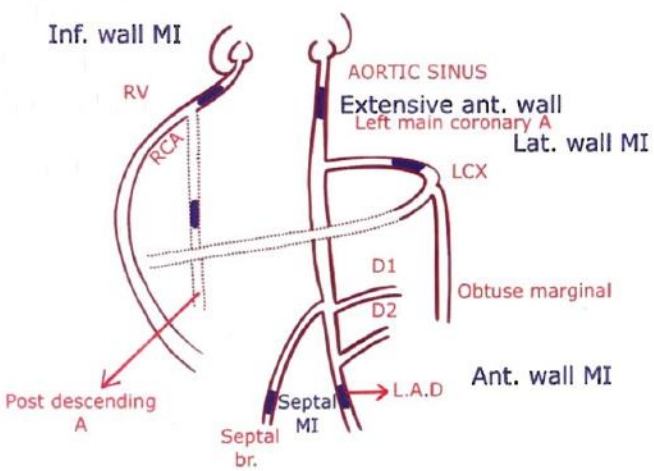
Eithoven law

- II=I+III

> **Important Information**
>
> - Augmented lead voltage is 1.5 times more than limb lead
> - Eithoven invented ECG
> - HANS BERGER invented EEG
> - LAENNEC invented Stethoscope

First branch

- **AORTA**: Coronary Artery
- **Aortic arch**: Brachiocephalic trunk



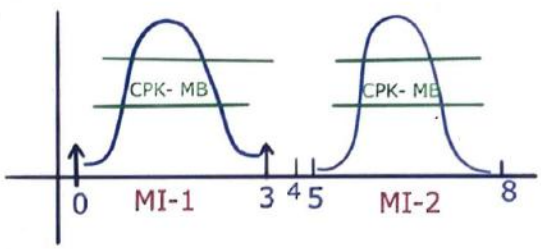
CARDIAC BIOMARKERS

02:53:27

1. **CPK-MB (Older)**: Creatine phosphokinase myocardial band

Marker	Rise	Peak	Fall
• CPK-MB • Trop T • Trop I	• 4-6 hrs • 3 hr • 3 hr	• 12-24 hrs • 24 hr • 24 hr	• 48-72 hrs • 10-14 days • 7-10 days

- 1st to rise in MI = Heart fatty acid binding protein (HFABP)
- Old data = Myoglobin
- Best / IOC / Sensitive / Specific } Cardiac Troponin-I
- IOC MI in Athletes
- Reinfarction > 72 hr: CPK-MB
- Reinfarction > 1 hr: Troponin I: ↑↑ 20% admission base line value
- Reinfarction: Troponin I
- Last to rise in MI: LDH. (Lactate dehydrogenase)



- MI: Normal Troponin I < 0.04 ng/dl (Minimum) x2, x3, x5, x10
- Troponin I Does not rise immediately
- SERIAL TROPONIN I: Admission value: Normal
  - 3hr value: ↑↑

DIAGNOSIS OF MYOCARDIAL INFARCTION

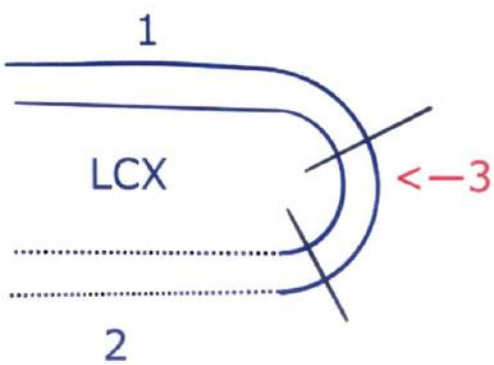
Levine Sign positive x 3hr

1. Elevated Cardio Biomarkers

- Cardiac Troponins are more reliable and / or

2. ECG changes

- 1st done in any case of chest pain



Limitations

1. Poor at localizing right ventricular ischemia
2. Ischemia of left circumflex artery and/or
3. Intra-op MI

- SpO_2 ↓, BP ↓, crepitations ++
- Trans-esophageal echocardiography
- T.E.E

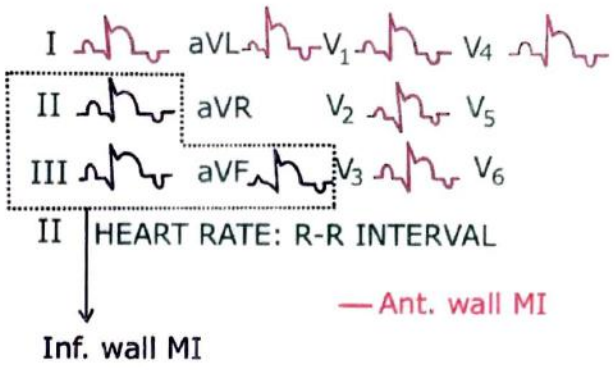
Stunned myocardium

- If any of these are positive, it indicates myocardial injury due to coronary artery thrombosis.
- If asked to select one, elevated cardio biomarkers are most reliable for diagnosis

INFARCT LOCALIZATION

03:06:57

MI	BV #	ECG finding
LAT WALL	LCX	V_5 V_6 I AVL
Septal MI	Septal Branch	V_1 V_2 T AVL
Anterior wall MI	LAD	V_1-V_4 I AVL
Extensive wall MI	Left main CA	V_1-V_6 I AVL
Posterior wall MI	PDA (Intraventricular)	V_1-V_4 → ST ↓ V_7, V_8, V_9 → ST ↑ (Reciprocal changes)
NSTEMI ant wall	LAD	V_1-V_4 → ST ↓
MI Inferior wall MI	Inferior wall MI	lead I, II, aVF



TYPES OF MI

03:21:26

- Type 1 : Coronary artery thrombosis, coronary artery dissection
- Type 2: Cocaine overdose, S. anemia, CO poisoning
- Type 3: sudden cardiac death, V. Fib, VT, Mobitz II heart block
- Type 4A: PCI: x5 times Trop I elevation
  - 4B: Latent stent thrombosis
- Type 5: During CABG: x10 times Trop I elevation

Table 21.1

Abdominal aorta	Coronary Artery	Popliteal artery	Circle of wills

• Abdominal Aorta aneurysm
• If >5.5cm
  ○ Spontaneous rupture
	• chronic stable angina
	  ○ Chest pain on exercise: 70% blockage
	• Acute Coronary Syndrome
	  ○ MI
	  → STEMi
	  → NSTEMi
	  ○ Unstable angina
	• Prinzmetal angina/variant angina
		• Claudication	• TIA
			• Stroke

Table 21.2

Findings of Coronary Angiography				
Triple vessel disease (TVD)			**Double vessel disease (DVD)**	**Single vessel disease (SVD)**
LAD Anterior wall	LCX lateral wall	RCA Inferior wall	**Single site disease** Rx: PCI+stenting	**Multiple site disease** Rx: CABG	• Best prognosis because of collaterals Rx • PCI+stenting (to prevent redevelopment)
Treatment of choice • CABG					

22 ACUTE CORONARY SYNDROME PART-2

STEMI V/S NSTEMI

00:00:17

Case : 60 yr old male smoker, HTN (Non-compliant), Levine sign +, Diaphoresis (+), Dyspnea on minimal activity (Grade III) orthopnea, Pink frothy sputum

O/E: HR – 160/min, BP – 180/100 mm of Hg, JPV ↑, neck veins full, Kussmual sign+, S1- Loud (Tachycardia), S2 – ERB's point – splitting

- Narrow split S2: LVF Wide split S2: RVF
- S3: low pitch / ventricular gallop rhythm
- Bi - Basilar Crepitations → Interstitial lung disease / Idiopathic pulmonary fibrosis
- Chest- B/L fine crepitations
- ECG → V1 - V4: ST ↑: 3mm
- Troponin I → x 2

Diagnosis

- STEMI/ Ant wall / LVF

Rx 00:08:04

- **M - Morphine**
- **O - Oxygen**
- **A - Aspirin**
- **N - Nitrates**

How to remember

- MOAN

1. O_2: SpO_2 < 95%
2. Tab Aspirin 300 mg (Chewable)

- (Absorbed from buccal cavity mucosa when crushed between teeth)
- Inhibit TxA2 production
- Clopidogrel (300mg): P2Y12 receptor → inhibit platelet aggregation

3. Sublingual nitroglycerine (0.4 mg) / buccal spray→ Repeat at every 5 min, max 3 tabs over 15 minutes

- Causes pooling of blood in vein S/E: SBP ↓
- Contraindicated:
  - SBP < 80 mm Hg
  - Inf. wall MI

4. Morphine → SC / IM / IV

- Vagotonic action
  - ↓ Pulmonary edema
  - ↓ Air hunger
  - ↓ Infarct size
  - ↓ Pain
- C/I HR<60

5. IV metoprolol

- ↓ HR ↓ O_2 consumption
- ↓ Infarct size
- Contraindicated in inferior wall MI

6. Inj Enoxaparin (SC) : Prevent clot which has already developed in coronary artery of patient

7. Pulmonary edema

- IV furosemide
- ACE inhibitor (IV enalaprilat)
- Nitroglycerine drip

8. Cath lab : femoral/ radial artery access (Percutaneous coronary intervention)

- Angiography
- Balloon angioplasty
  - Door to balloon time
  - < 90 minutes

Cath Lab: Not available → Thrombolysis Door to needle time < 30 mins

Thrombolysis Drugs

- Alteplase: Preferred in STEMi
- Reteplase: Acute ischemic attack
- Tenecteplase
- STK (Streptokinase)

Mechanism of action

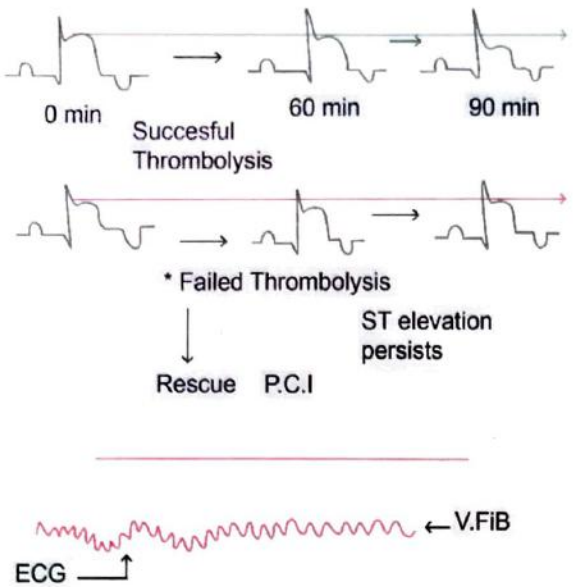
- Generate Plasmin
- Fibrin insoluble → Fibrin soluble
- **MC complication of thrombolysis** → Bleeding
- **Antidote of STK Toxicity** → Epsilon amino caproic acid

STK: Group C Streptococcus

- Streptococcus Equisimilis
- Allergic reactions / anaphylactic shock (Type I)
  - Reuse of STK - after 6 months
  - Mortality due to → Laryngeal edema

Rx of Anaphylaxis 00:30:36

- IM adrenaline 0.5 ml
- Undiluted (1:1000)
- Cytokines in anaphylactic shock → IL-4
- Cell responsible for histamine release → Basophil
- IOC: Serum Tryptase level
- Mortality d/t Laryngeal edema



ST elevation persist after 90 min from thrombolysis infusion: Rescue P.C.I

Important Information

- In case of failure of thrombolysis, rescue PCI is done within 90 minutes of starting thrombolysis
- Note that this 90 minutes is not from entry into hospital but initiation of thrombolysis

STEMI

- Cath lab - 1° PCI
- D2B time < 90 min of admission

Or

- Cath lab - not available
- Thrombolysis
- D2N time < 30 min of admission

↓ Fails

- Rescue PCI

NSTEMI (Thrombolysis contraindicated)

↓

- ABE-MOAN

↓ Fails

- delayed PCI

THROMBOLYSIS CONTRAINDICATED

00:41:30

1. Recent stroke (< 3 months)
2. Bleeding diathesis
   - Platelet count < 1 lac
   - Prothrombin / International normalized ratio → ↑↑ (11-16 sec)
   - aPTT → ↑↑ (30-40 sec)
   - Single donor platelets are given Fresh frozen plasma
3. Unstable angina / Prinzmetal angina → spasm NSTEMI
4. Aorta dissection
5. SBP > 185 DBP > 110 mm Hg - IV Labetalol
6. CPR > 10 minutes (Prolonged)
7. Pregnancy
8. Known case of Brain Tumor → ↑ hemorrhage Risk
9. Known case of A-V aneurysm → ↑ hemorrhage Risk
10. Recent major vascular surgery

00:48:56

Case : Case : 60 yr old male smoker, HTN (Non-compliant), Levine sign +, Diaphoresis (+), Dyspnea on minimal activity (Grade III) orthopnea, Pink frothy sputum

O/E: HR – 160/min, BP – 180/100 mm of Hg, JPV ↑, neck veins full, Kussmual sign+, S1- Loud (Tachycardia), S2 – ERB's point – splitting

- Narrow split S2: LVF Wide split S2: RVF
- S3: low pitch / ventricular gallop rhythm
- Bi - Basilar Crepitations → Interstitial lung disease / Idiopathic pulmonary fibrosis
- Chest- B/L fine crepitations

ECG

- T wave inversion V_1 - V_4
- cTroponin → ↑

Diagnosis

- NSTEMI / ant wall of heart
- **Rx**
  - **A** - IV **A**bciximab
  - **B** - IV blocker (↓ HR)
  - **E** - Inj **E**noxaparin (SC)
  - **M** - **M**orphine
  - **O** - O_2
  - **A** - **A**spirin clopidogrel
  - **N** - IV **N**TG drip

How to remember

- ABE-MOAN

- STEMI + unstable angina → ABE - MOAN
- (DOC = IV NTG drip > Inj Enoxaparin)
- Fails to respond = SpO2 ↓

- BP↓ CREPTS ++
- Delayed PCI
- Unstable angina / NSTEMI

00:52:51

	STEMI	NSTEMI	Unstable angina	Prinzmetal Angina
Trop I	x 2	x 2	Normal / ↑	Normal
ECG	ST ↑	ST normal/ ↓	ST normal/ ↓	↑
Rx	1° PCI or STK	ABE-MOAN ↓ fails Delayed PCI		IV NTG Drip

- IV NTG drip : UA/NSTEMi/PA

COMPLICATIONS OF MI

01:00:31

1. **Arrhythmias (M/C)**
- Ventricular fibrillation: Twitching, pulseless / BP ↓↓ (< 1 hr) : Golden period
- Sudden cardiac death
- M/C bradyarrythmia
- Post MI → Mobitz 11 H Block
- V. Fib - ECG = Twitching activity
- < 5 min (Onset of V. fib) = DC shock 200 J biphasic
- > 5 min (Onset of V. Fib) = 1st step is chest compressions as brain stem is having severe ischemia and by the time DC shock is delivered it may be too late
- **Fib** = > 5 minutes (Onset)

1. CPR = 30: 2 x 2 minutes
2. AED =DC → 200 j Biphasic (Non-synchronized)
3. CPR – DC shock x 3 times
4. IV – adrenaline → ↑ Coronary perfusion

Chest compression
- 30:2 → rescue breaths at 100-120 / min

Sternal depression
- 5 cm lower 1/3rd of sternum
- M/C solid organ traumatised during CPR → Liver
- M/C rib# during CPR → 4-6th ribs (angulated ribs)

Important Information

Adult : CPR Ratio
- 30:2
- Single or two rescuer
- Sternal depression: 5cm

Paediatrics: CPR Ratio
- Single Rescuer – 30:2
- Two rescuer – 15:2
- Sternal depression: 2cm

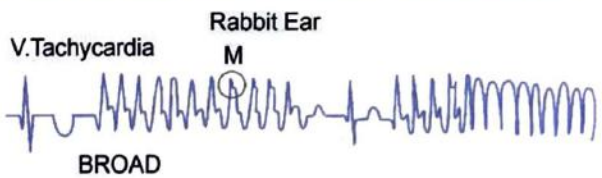
Neonatal resuscitation :CPR Ratio
- 3:1
- Mandatory → 2 rescuers
- Sternal depression: 1cm

Tachycardia
- MI after 12 hours
- Ectopic focus = 200-300 / min: V rate
- Exhibits Inter Myocyte conduction
- (Slower than conduction in Purkinje)
- Hence QRS is broad
- A rate = 100 / min, While V. Rate is 200-300/min
- Leading to AV dissociation

Pulseless VT
- AV dissociation (Lack of co-ordination)
- V rate ↑: Reduced duration of end diastolic volume
- SV ↓
- Cardiac output ↓
- SBP ↓

EGG CRITERIA OF V. TACHYCARDIA

- M shaped appearance/Rabbit ear: At peak of R wave



1. Broad QRS – 80-100 m sec = 2-2.5 ss small square
2. Premature V. contraction > 3 PVC, HR > 100/ min
3. Plateau shaped appearance at start of S wave called as Josephan sign

- Capture beats
- Spindle appearance

Extreme Axis deviation

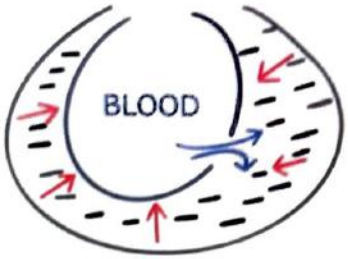
Rx: Pulseless VT

- Cardioversion: DC shock 200 J
- Stable VT → IV Amiodarone
- IV Lignocaine

2. **Cardiogenic Shock (Hospital Mortality)** 01:17:08

- Norepinephrine - DBP ↑ coronary perfusion ↑
- Dopamine: D1 Receptor and ↑ GFR
- Dobutamine B1, Cardiac output ↑ HR
- ↑↑ O_2 consumption
- Refractory Cardiogenic Shock = Impella > Intra Aortic Balloon pump

3. **Cardiac Rupture: Transmural MI**



CARDIAC Tamponade

- Normal pericardial fluid = 20-50 ml
- Cardiac tamponade: Intrapericardial pressure >> LVED
  - Eectro mechanical dissociation
  - EDV ↓↓ SV ↓↓ → CO ↓↓ → BP ↓↓
  - DC Shock: CI

Tamponade

- HR ↑ = R-R ↓ P-R ↓, low voltage ECG
- = qRS amplitude variable

ELECTRICAL ALTERANS

4. **LV Aneurysm**

- Damaged part ↓ Contractility
- CO↓
- Stasis: clots
- Embolic stroke

ECG: Persistent ST ↑

Dx: Echocardiography (IOC)

Rx

- Warfarin + inj Heparin
- Discontinue Heparin after 5 days
- Continue warfarin lifetime
- Same brand (because different brands have different bioavailability)
- Warfarin: PT/INR monitoring

5. **DRESSLER SYNDROME**

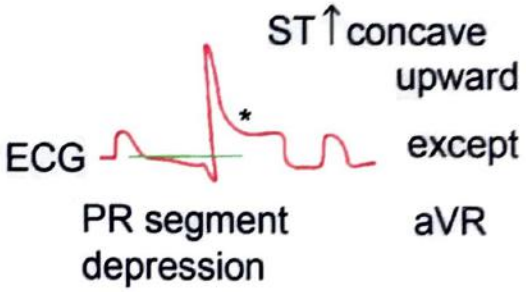
- Autoimmune Pericarditis which present as diffuse chest pain at rest after 3-6 weeks of discharge of patient of MI. 3-6 weeks later
- Radiation of pain → Left shoulder
- Relief = Sitting position

O/E: Pericardial Friction Rub → Inspiration + expiration

- Pleural rub → inspiration
- $S_1 S_2$ split
- Troponin I: Normal / ↑

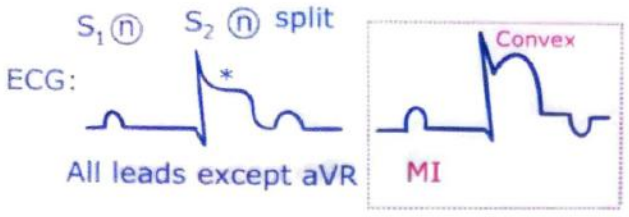
ECG: ST ↑ concave upward exept aVR , PR segment depression

Rx: Aspirin 650 mg TDS x 3 days

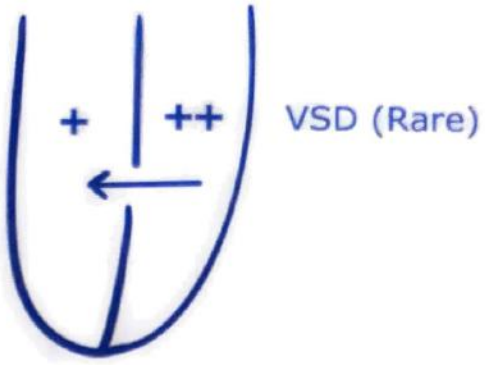


6. **Valvular dysfunction**

- M/C: Mitral regurgitation
- Never seen post MI - AR
- Aorta: Aortic valve

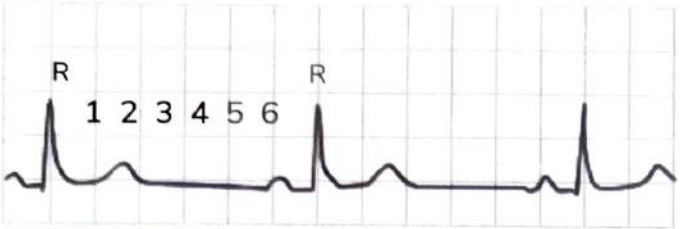


7. **Ventricular Septal Defect (Rare)**



23 SYMPTOMATIC BRADYCARDIA WITH PULSE

- R – R interval (5 to 6 large square) - HR: 50-60 bpm ⏱ 00:00:13



Clinical features: Cut off < 60 bpm (Bradycardia)
- If no symptom in pt. than no treatment required

SYMPTOMATIC PATIENT ⏱ 00:03:50

1. Dizziness
2. Altered mental status
3. Chest pain
4. Pulmonary congestion
5. CHF features

TREATMENT ⏱ 00:05:30

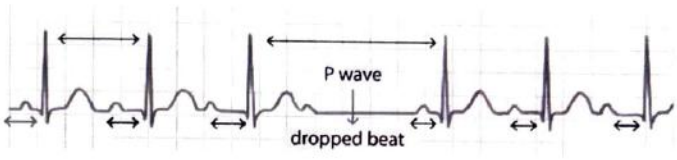
1. Patent airway
2. Monitor SpO_2
3. Assisted ventilation → NIV/ET tube
4. IV Access: Atropine 1mg, repeat every 3-5 mins, max. = 3mg
5. Inspite of Atropine, no change in HR or HR still dropping
- Immediately move to TCP and not next dose of atropine

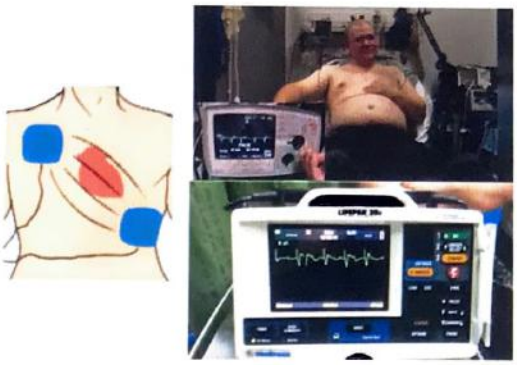
Important Information

Drugs useful for chemical pacing
- **Atropine**
- **Epinephrine: 2-10ug/kg/min**
- **Dopamine: 5-20/ mcg/kg/min**

TRANSCUTANEOUS PACING ⏱ 00:10:20

- Delivery of current in an incremental way (10mA↑ : 70 mA)
- Best method for symptomatic bradycardia patients
  - 1st electrode: below right Clavicle
  - 2nd electrode: Near Axilla of Patient / below the left nipple
- Stickon pads on chest of Patients
- Oxygen with reservoir





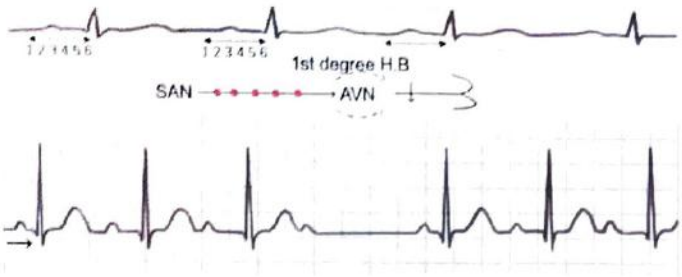
Steps
1. Apply PADS
2. Sedation ±
3. Set HR = 60 – 80 bpm
4. Mode – Synchronous
5. 10 m A ↑ until capture occurs
6. Broad complex QRS → check femoral pulsation
- Don't use carotid pulse, as TCP may cause fluttering of neck muscles

Management algorithm of symptomatic bradycardia
1. **IV atropine 1mg every 3-5 min**
- Maximum dose = 3 mg
- If Atropine fails then→ TCP
2. **IV epinephrine drip**
- Activates β1 in the heart
- Dose → 2-10µg/kg/minute
3. **IV Dobutamine (2-20µg/Kg/ min)**
- Activate β1, D1 receptors
4. **Transcutaneous pacing**
5. **Transvenous pacing**

1ST DEGREE HEART BLOCK ⏱ 00:17:17

- ↑↑ PR Interval (> 200 m sec) → Presence of 1st degree heart block

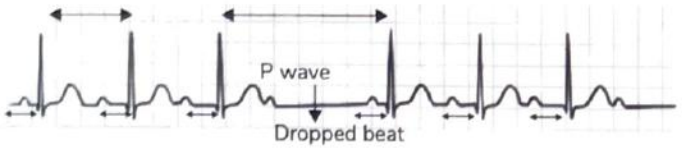
  SAN $\xrightarrow[\text{seen in Rheumatic fever}]{\text{Slow conduction}}$ AVN → Bundle of His
- Most common cause is physiological → Marathon runners



2nd Degree Heart Block

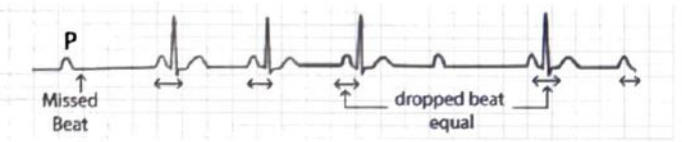
Mobitz I/Wenckebach

- Serial ↑in PR Interval
- Dropped QRS (Missed Beat)
- Compare PR interval before & after the missed beat
- PR interval before missed beat is longer
- Intra nodal defect



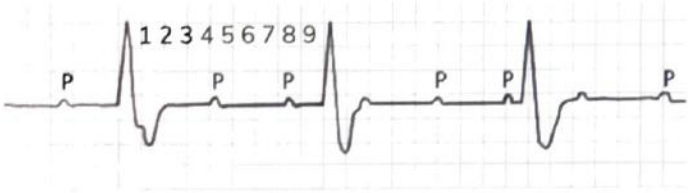
MOBITZ II

- Infra nodal defect
- Bundle of His (Refuses to conduct)
- PR Interval is constant
- Missed beat present
- PR before and after missed beat is equal



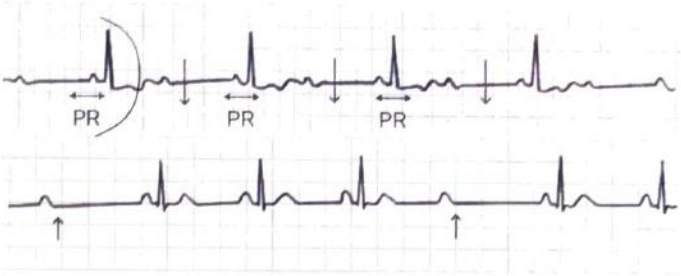
3rd Degree Heart Block/ complete heart block

- Broad QRS complex
- PR interval ↑↑
- PP interval is not matching RR interval
- RR interval (Ventricular Rate)
- PP Interval (Atrial Rate)



2:1 Block

- Alternatively conducted impulse & non conducted impulse
- Dropped beat is present alternatively unlike in mobitz II where multiple impulses are conducted followed by a drop



- **M/C bradyarrhythmia seen post – MI**: MOBITZ II heart block

24 CONGENITAL HEART DISEASE

NADAS CRITERIA

00:00:30

Major

- CHF (L→R shunt)
- Cyanosis (R→L shunt)
- Diastolic murmur
- Systolic murmur grade III

Minor

- Systolic murmur grade I/grade II
- Abnormal S_2
- Abnormal ECG
- Abnormal CXR
- Abnormal BP values

Important Information

- 1 Major or 2 Minor Criteria for the diagnosis of congenital heart disease

ACYANOTIC CHD

00:07:30

VSD	PDA	ASD
6 weeks	Birth/6 weeks	5 y./ 25 y.
CHF + Recurrent Pneumonia Episodes		
S_2	Wide split (variable)	Narrow split	Wide split (fixed)
Shunt murmur	PSM	Continuous murmur	Flow murmur (MDM/ESM)
Rx	Dacron patch	Indomethacin Surgery in term child	Occlusion devices

ASD/VSD

00:10:42

ASD	VSD

• O. secundum (M/C) → RAD
• O. primum, AV canal defect, endocardial cushion defect
  ○ LA++: LAD
• Sinus venosus: PAPVC (Partial)
  ○ Scimitar Sign
	• Peri membranous (M/C)
	• Muscular variety
	  ○ Spontaneous closure
	• Supracristal with AR

CYANOTIC HEART DISEASE

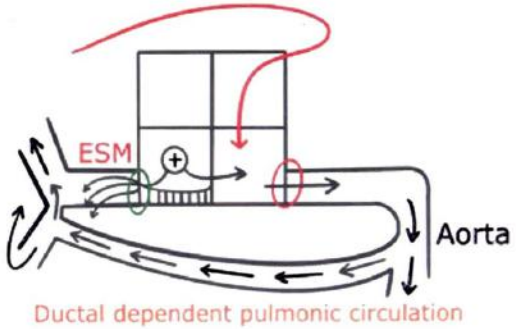
00:18:00

Tetralogy of Fallot

Pathophysiology

I. Subpulmonic stenosis (hallmark)
II. Concentric RVH
III. VSD (R→L Shunt)
IV. Overriding aorta

Tetralogy of Fallot	Triology of Fallot	Pentalogy of Fallot
• Subpulmonic stenosis • Concentric R.V. H • VSD (R→ L shunt) • Overriding aorta	• Same • Same • ASD	• T.O.F + ASD

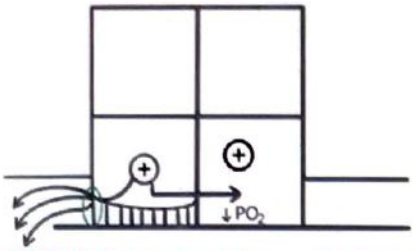


Ductal dependent pulmonic circulation

- Major determinant of central cyanosis →Subpulmonic stenosis
- Central cyanosis in a body on day O – TGA
- Central cyanosis in Tetralogy of Fallot develops on/after day 7

Rx

- **DOC** – Alprostadil (Maintain Ductal Patency): PGE1
- **Palliative surgery** – Blalock Taussig shunt (Connecting subclavian Artery → Pulmonary Artery)



ESM intensity = lesser

TET SPELLS / HYPER CYANOTIC SPELLS

00:27:15

- R → L shunt: ↑
- Lips: blue → black, murmur: softer
- In pts. suffering from Tet spells intensity of ESM murmur become less, murmur will become softer
- **Complication** → Brain infarction: C/L hemiplegia
- ↓ Po_2 leads to ↑EPO+++ → RBC count ↑
- 2° polycythemia: Sluggish circulation

Management of tet spells

1. Knee- chest position
   - ↑ P. resistance (d/t compression of femoral artery)
   - R→L shunting ↓

Knee- chest position

2. IV Morphine
3. IV Soda bicarbonate
4. IV Methoxamine
   - SBP ↑ : ↑ Pul. Resistance
5. IV propranolol

Important Information

- IV calcium gluconate is not useful in the management of tet spells

T.O.F. Clinical Features

- Most common CHD (Cyanotic)
- Central cyanosis day 7
- Tet spells / F.T.T

O/E

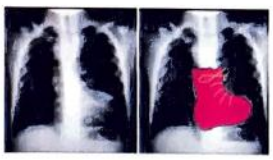
1. Clubbing, cyanosis
2. Shunt murmur of VSD is not heard
3. S_2: A_2 P_2 (single S_2)
4. Flow murmur → E.S.M, intensity ↓, tet spells

WORK UP

00:39:30

Chest X-ray

1. Pulm. Oligaemia
   - RV enlargement (Boot shaped heart)
   - Apex displaced superolateral: RV
   - Apex displaced inferolateral : LV



2. Rt sided aortic arch (30%)
   - "COER – EN- SABOT"

IOC: Transthoracic Echo (TTE)

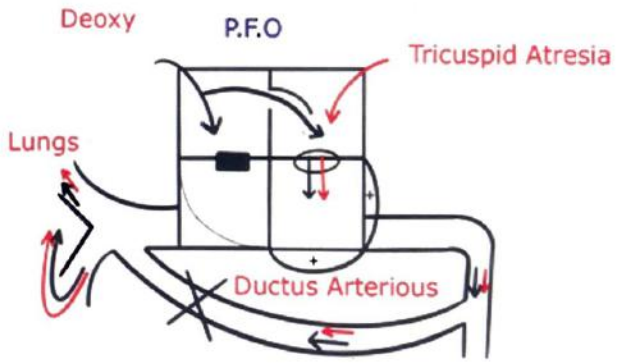
Rx

- Alprostadil IV/ Blalock- taussing shunt (palliative Sx)
- Pink Tet- Milder subpulmonic stenosis
- Old children with T.O.F.- Squatting episode
- Central cyanosis + N. size heart = CT ratio: N

TRICUSPID ATRESIA

00:44:20

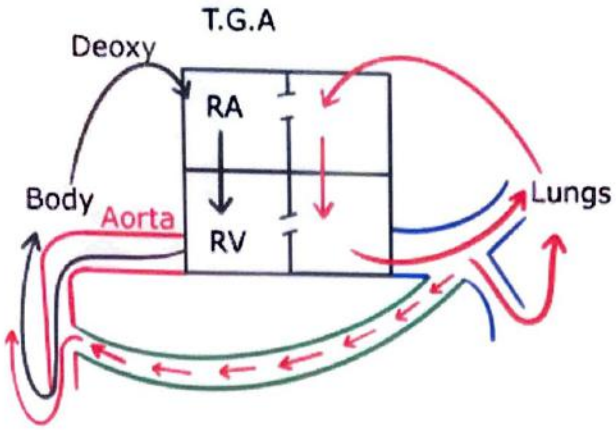
- Central cyanosis – day 0
- Death – day 7
- Chest X-ray: CT ratio ↑ (Left sided enlargement)
- $S_1 = M_1$ louder (Single loud S_1, T1 absent)
- $S_2 = A_2$ normal (Single S_2)



TGA (TRANSPOSITION OF GREAT ARTERIES)

00:53:40

- You can keep patient alive by giving drug alprostadil IV
- Ductus arteriosus = Patency alprostadil
- Immediate = atrial septostomy → switch operation
- Central cyanosis – day 0



CXR → Egg on side appearance

Important Information

- Egg shell calcification: Sarcoidosis, Silicosis
- Egg in a cup: Constrictive pericarditis

Important Information

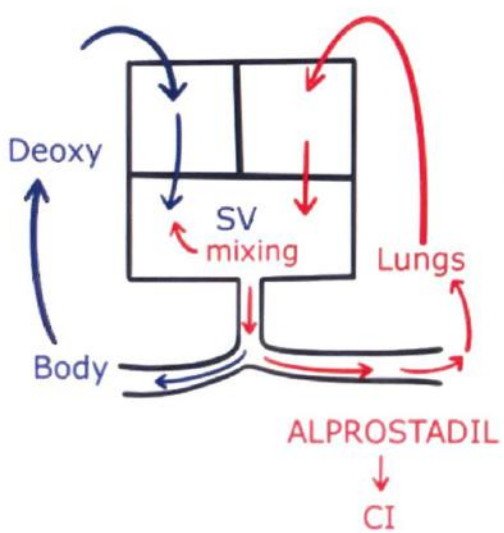
Ductal dependent pulmonary circulation

- T.O.F.
- T.A
- TGA with intact VSD

TRUNCUS ARTERIOSUS

⏱ 00:59:50

- Co - Truncal abnormality
- Alprostadil C/I
- Ductal dependent circulation

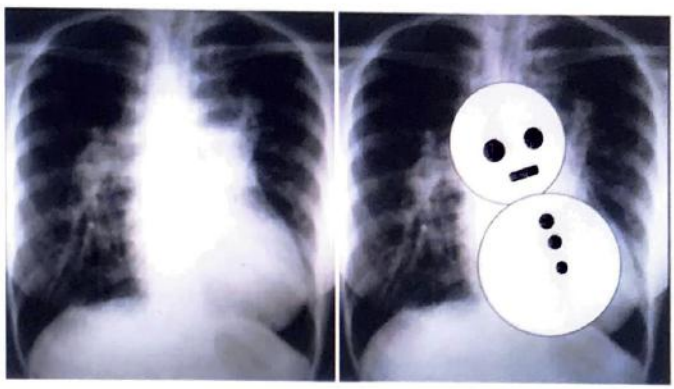


TAPVC

⏱ 01:02:12

- Abnormal Vein → SVC : Supracardiac type
  - Most common : Fig of 8 appearance on CXR
- Abnormal vein → RA : Cardiac
- Abnormal vein → IVC : Infracardiac

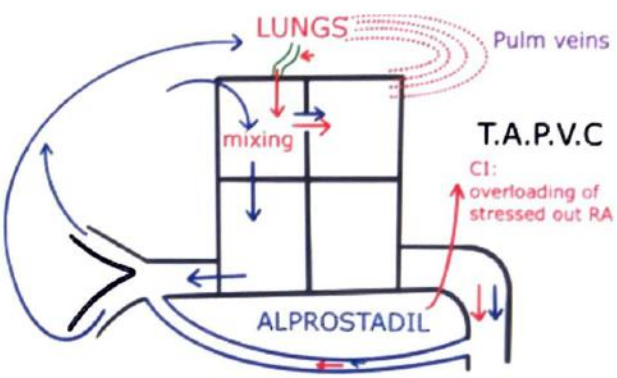
- "Ground glass appearance" on CXR



Supracardiac Type

Important Information

- TAPVC with ASD → Wide fixed split S_2



AORTIC ATRESIA

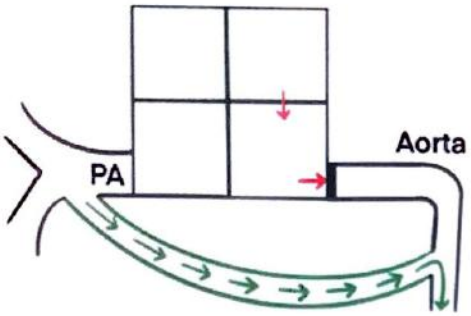
⏱ 01:09:00

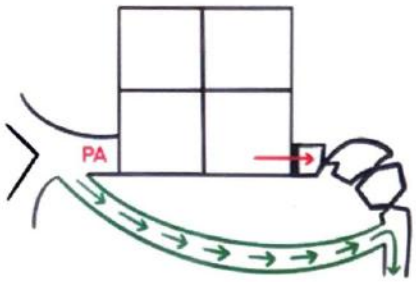
- Symptomatic on day 0 (central cyanosis)
- Neonate: Shock cyanosis
- Ductal patency maintained by alprostadil IV

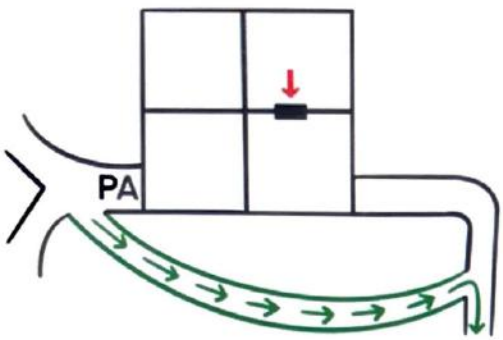
DUCTAL DEPENDENT SYSTEMIC CIRCULATION

⏱ 01:13:35

1. Interrupted aortic arch Neonate: shock cyanosis
   - Alprostadil IV
2. Mitral Atresia
3. Aortic Atresia







Pulmonary	Systemic
1. TOF 2. Tricuspid atresia 3. T.G.A with intact VSD	1. Mitral atresia 2. Hypoplastic left heart syndrome 3. Aortic atresia 4. Interrupted aortic arch.

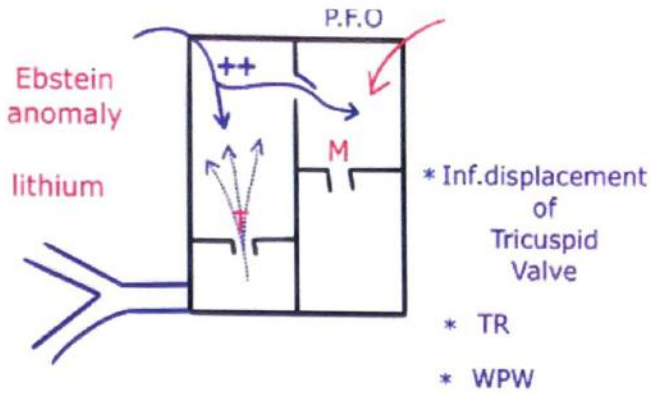
Ductal independent circulation

1. TAPVC
2. Truncus arteriosus (Co - Truncal abnormality)
3. Kawasaki disease

EBSTEIN ANOMALY

01:17:09

- H/o lithium intake by the mother
- Due to inferior displacement of tricuspid valve
- Tricuspid regurgitation
- Association of WPW syndrome



Summary

01:21:07

L → R shunt (CHF + Recurrent Pneumonia)	R → L shunt (Cyanosis, FTT)	Mixed combo (Cyanosis + CHF)
• VSD (wide variable S_2) • ASD (wide fixed S_2) • PDA (Narrow split)	• TOF • TA • Ebstein anomaly	• TGA (Egg on side appearance) • TAPVC (Figure of 8appearance)

25 METABOLIC SYNDROME X AND SYNDROME Z

METABOLIC SYNDROME (SYNDROME X)

00:02:13

1. **Centripetal obesity** →Visceral/ abdominal fat → Heart disease
   - Measured by → Abdominal circumference
     - Female > 88 cm: In Indian Female > 80 cm
     - Male > 102 cm: Indian Male > 90cm

2. **↑ Insulin Resistance**
   - FBS = 100-125 mg%
     - IGT (Impaired glucose Tolerance Range)
   - 2 hr PPBS = 140-199 mg%
   - Pt. on hypoglycemic drug

> **Important Information**
> - if patient on medication (Eg. Metformin) for hyperglycemia
>   - component of metabolic syndrome

3. **↑ Peripheral Resistance** → ↑ BP
   - HTN: > 130/85 mm Hg (On medication for HTN)

4. **TG** > 150 mg% [Or on medication] for hyperlipidemia

5. **HDL** < 40 mg (Males)/ <50 mg/dl (Females)/ [On Medication]

> **Important Information**
> - For diagnosis of metabolic syndrome 3 out of 5 components should be present

SYNDROME Z : SYNDROME X + O.S.A (Obstructive sleep apnea)

- Apnea cutoff- 10 sec
- 5 episodes/hour
- ↑ C V mortality of Patient

> **Important Information**
> - LDL ↑ is not a component of both Syndrome X and Syndrome Z

CORONARY SYNDROME X/ MICRO VASCULAR ANGINA

00:07:23

- Perforator narrowing leading to coronary ischemia
- Chest pain on exertion/ emotion
- Recurrent episodes
- **Treadmill test**: ST ↓ 1mm persisting 80 msec: 2 leads
- Coronary angiography Ⓝ

Rx:
- Isosorbide mononitrate + Aspirin

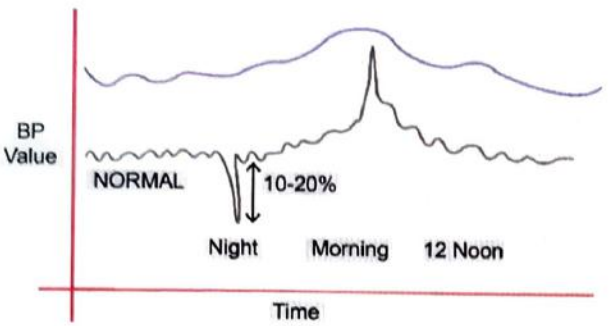
> **Important Information**
> - Stenting is not useful in management of coronary syndrome X

26 HYPERTENSION

INTRODUCTION

00:00:15

- Normally there is fall in BP at night when the muscles are relaxed – Nocturnal Dip is there with approx 10-20% of fall in BP
- The BP values are higher at 12:00 noon w.r.t diurnal variation



- Ambulatory BP recording for normal person (Black tracing), and HTN patient (Blue tracing)
- Loss of nocturnal dip at night time in HTN patient
- Attenuated nocturnal dip:↑risk of cardiovascular mortality in HTN patient

Important Information

- **Antihypertensives should be taken either in early morning or at night, so that morning ↑↑ in BP due to vasoconstriction can be prevented**

- Attenuated nocturnal dip: There is ↑risk of cardiovascular mortality in HTN patient
- In patient with good control HTN
  - Acute coronary syndrome to be ↓↓ by 25%
  - Stroke to be ↓↓ by 30%
  - Heart failure to be ↓↓ by 50%

BLOOD PRESSURE

00:04:50

- Normal value of BP generally varies for all, but if
- BP < $\frac{115}{75}$ mm of Hg
- if SBP > 20 mm of Hg or DBP > 10 mm of Hg is associated with ↑↑ cardiovascular mortality

CLASSIFICATION

00:06:40

According to Harrison

- Pre HTN = 120-139/80-89 mmHg
- Stage 1 = 140-159/90-99 mmHg
- Stage 2 = > 160/100 mmHg

According to AHA guidelines

- Elevated = 120-139/ < 80 mm of Hg
- Stage-1 = 130-139/ 80-89 mm of Hg
- Stage-2 = > 140/90 mm of Hg

Ambulatory BP monitoring (ABPM)

- Average awake BP > 135/85 mm of Hg
- Average sleep BP > 120/75 mm of Hg
- Clinic BP = > 140/90 mm of Hg

Automated office BP (AOBP)

- 3 readings
- Discard 1
- 2 reading = average
- > 135/85 mm Hg

Ambulatory BP monitoring (ABPM)

- Average awake> 135/85mmHg
- Average sleep > 120/75

Home based BP moitoring (HBPM)

- >135/85mm Hg
- 7 days:M/E
- Discard 1
- 6days : average
- Cuff size: 80% of arm circumference
- Width: >40%
- Rest :5 min
- After smoking / coffee consumed : wait 30 min
- BP measure both arms
- BP measure legs

White coat HTN

- BP normal at home but increase in clinic
- Can develop HTN in future

Office BP: ≥130/80 mm Hg but <160/100 mm Hg after 3 mo trail of lifestyle modification and suspected white coat hypertension

↓

Daytime ABPM or HBPM BP<130/80 mm Hg

Yes / No

White Coat Hypertension
- Lifestyle modification
- Annual ABPM or HBPM to detect progression (Class IIa)

Hypertension
Continue lifestyle modification and start antihypertensive drug therapy (Class IIa)

White Coat Hypertension
- Lifestyle modification
- Annual ABPM or HBPM to detect progression (Class IIa)

Masked HTN

- BP normal in clinic but elevated at home (stage1)
- Need anti hyperntensive medicine

Office BP: 120-129/80 mm Hg after 3 mo trail of lifestyle modification and suspected masked hypertension

↓

Daytime ABPM or HBPM BP>130/80 mm Hg

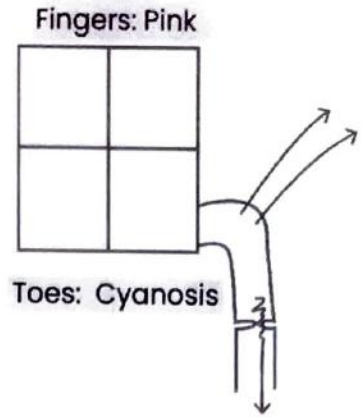
Yes / No

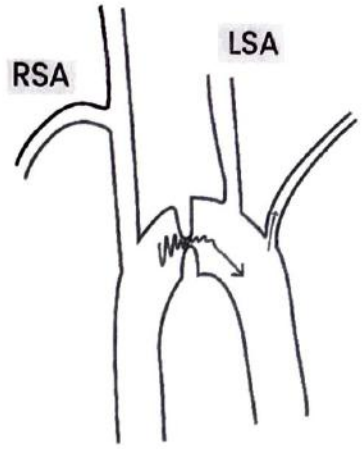
Masked Hypertension
Continue lifestyle modification and start antihypertensive drug therapy (Class IIa)

Elevated BP
- Lifestyle modification
- Annual ABPM or HBPM to detect masked hypertension or progression (Class IIa)

CAUSES OF HYPERTENSION

- Most common cause of HTN: Essential HTN/ idiopathic
- Most common cause of 2° HTN: Renal Parenchymal disease
- 2° HTN (America) → Obstructive sleep apnea
- Congenital cardiovascular causes of HTN → Coarctation of Aorta
- In Coarctation of Aorta (Post ductal) = BP in upper limb ↑↑ and BP in lower limbs ↓↓
- Muscle wasting present in lower limbs
  - BP = CO x P. resistance





Important Information

- Normally BP in upper limb < 120/80, & BP in lower limb < 140/80
- Reverse scenario is seen in coarctation of aorta, where BP in legs ↓ ↓ and upper limb ↑ ↑

Unequal BP left/right arm is seen in 00:27:25

- Takayasu's arteritis (Imp causes of renal artery stenosis in India)
- Supravalvular AS (Aortic Stenosis): William syndrome (Conda Effect)
- Coarctation of Aorta (Pre ductal)
- Aortic Dissection: (acute) antegrade

SECONDARY CAUSES OF HTN 00:30:25

- Identified HTN < 30 year of age
- Newer HTN > 55 year

1. Renal parenchymal disorder
- Chronic Kidney disease
- Chronic glomerulonephritis

2. Renovascular causes

- Fibromuscular dysplasia,
- Atherosclerosis

3. Metabolic syndrome/ syndrome X
- Centripetal obesity
- Insulin resistance
- Hypertriglycedemia
- HTN
- HDL ↓

- 3 out of 5 should be present for diagnosis

4. Obstructive Sleep Apnea (OSA)
- Fluctuation of heart rate during sleep ↑↑ the stress on the heart and that manifests into HTN, LVH
  - During apnea episode there is bradycardia (O_2 cut off from body) and due to hypoxia the person tries to breathe in and then there is cortical arousal – O_2 goes inside and there is sudden increase in the HR

5. Endocrinological
- Hypothyroidism (Isolated diastolic HTN)
- Thyrotoxicosis
- Pheochromocytoma (↑aldosterone)
- CONN syndrome
- OCP: due to secondary aldosteronism
- Neurogenic → Spinal cord transection (At T6 or above → unopposed sympathomimetic outflow to heart)
- Cushing reflex: ↑ BP
- Raised ICP
- Porphyria
- Lead poisoning (↑↑ activity of ACE)
- Mendelian causes 00:41:54

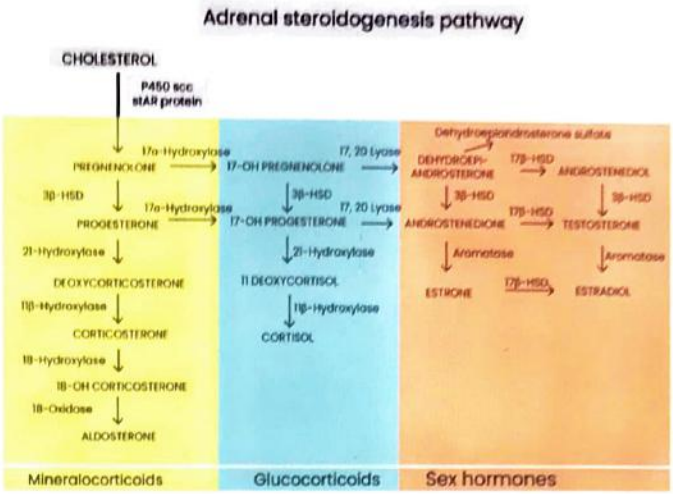
a. **Liddle syndrome (Autosomal Dominant)**
  - Overactivity of ENac – causes more salt/H2O to retain in the body
  - HTN with loss of K+/H+ causing hypokalaemia alkalosis
  - Treatment → Amiloride (ENac blocker)

b. **Gordan syndrome**
  - AD
  - WNK-1/ WNK-4: gain of function - Thiazide dependent NaCl cotransport in DCT
  - HTN due to more NaCl retaining in the body

c. **Polycystic kidneys**
  - Should have 2 or > 2 cyst in kidney
  - **Adults:** hepatic cyst
  - **Pediatric:** hepatic fibrosis
  - AD, AR (AD- adults, AR- paediatrics) and then the destruction of parenchyma in kidney causes ↓↓ GFR and Renin levels ↑↑ resulting in HTN
    → Abdominal pain
    → GI manifestations

d. **Pheochromocytoma (AD)**
  - Associated with MEN-2A (Sipple Syndrome), MEN-2B, VHL, NF-1

e. **Congenital adrenal hyperplasia (CAH)**
  - 17-α hydroxylase deficiency (AR)→ ↑Aldosterone → HTN
  - 11–β hydroxylase deficiency (AR)→ ↑Deoxycortisol→ stimulates ENaC → HTN



WORK UP FOR NEWLY 00:50:25

1. Echocardiography → Left ventricular hypertrophy
2. Fasting blood sugar,
3. lipids
4. Serum electrolytes
5. TSH
6. Urine microscopy, Albumin Excretion Rate (AER)
   $\frac{\text{Urine albumin}}{\text{Urine creatinine}}$ = (30-300 mg/gm)
7. BUN, Serum creatinine

TARGET ORGAN DAMAGE 00:55:25

- Eyes
- Brain
- Kidneys
- Aorta
- Heart

Heart damage
- 1st to damage
  - LVH
  - ↑O_2 demand
  - Subendocardial ischemia

ECG: ST ↓

Eyes
- Hypertensive retinopathy
- Fundus findings

I. Focal attenuation of arterioles (earliest)
II. I+A-V nipping (Salu sign)
III. I+II+flame shaped hemorrhage cotton wool spots
IV. I+II+III+Papilledema

Brain
- Hemmorahagic stroke
- M/C site: putamen

Kidneys
- GFR reduces progressively
- CKD

Aorta
- Aortic dissection
  - Tearing chest pain in interscapular area

Diagnosis
- TEE: in unstable patients
- MRI: in stable patients
- CT chest: Tennis ball appearance

Rx
- Type A (Tear in front) → Sx
- Type B (Tear present behind)
- unrecordalble BP→ Sx
- BP normal to low→ Esmolol (to retard the progression of tear)

When to treat as per ACC/AHA 01:06:30
- ASCVD risk ↑ : <130/80
- ASCVD risk not increased : > 140/90
- Age > 65yr : > 130/80

Treatment
- Life style modification

a. D.A.S.H (dietary approaches to stop hypertension):- Intake of Na^{++}↓↓, K^{+}↑↑

b. Physical activity
- If the patient is HTN despite the lifestyle modifications then switch to drugs

Step 1	ACE inhibitor/ ARB or[3] Calcium channel blocker or Thiazide diuretic[4]
Step 2	ACE inhibitor/ ARB plus Calcium channel blocker or thiazide diuretic[5]
Step 3	ACE inhibitor/ ARB plus calcium channel blocker plus thiazide diuretic
Step 4	ACE inhibitor/ ARB plus calcium channel blocker plus thiazide diuretic plus spironolactone

- **< 55 years**
  - "ACE inhibitor", in case of dry cough, angioedema then switch to "ARB"
  - ACEI/CCB
  - ACEI+ CCB+Thiazides
- **55 years**
  - CCB
  - CCB + ACEI + Thiazides
- **30yrs**
  - **A** - **A**CEI
  - **C** - Add **C**CB
  - **D** - Add thiazides

How to remember
- ACD

- If CCB's are not tolerated by the patient consider thiazides
- With long term use of thiazides
- HBA1c ↑
- Uric acid ↑
  - Can trigger gout in post-menopausal women
- Therefore, aldosterone antagonists can be used instead of thiazides
- **60 yrs**
  - Start with CCB
  - Add ACEI
  - Thiazides / AA

Resistant HTN
- Atleast 3 classes of anti HTN concurrently used in patient
- Must include Thiazide (atleast 2 weeks)
- Improper BP measurement should be ruled out
- Cox-1(-), cocaine, steroids
- Excess salt intake,obesity
- Alcohol intake
- Non compliance of patient

USES OF ANTIHYPERTENSIVE

1. ACE inhibitors
- Acute coronary syndrome -post MI
- Diabetic nephropathy
- Ischemic nephropathy (Unilateral RAS)
- HF with ↓↓ EF
- Chronic K^{+}
  i. Patiromer
  ii. K^{+} biding Resin (SPS)
  iii. Na zirconate
- **B/L RAS**
  - P.T.R.A.• Stenting
- **U/L RAS**

- ACEI

2. β- Blockers (Cardio selective)
- CHF due to systolic malfunction
- Chronic stable angina
  - Carvedilol
  - Metoprolol

3. Aldosterone antagonist → Used in patients of heart failure with preserved ejection fraction

4. α- Blockers:
- HTN in patients having BPH → Prazosin
- Pheochromocytoma → Phenoxybenzamine
- ACEI → Angioedema → Non pitting edema
  - **Rx**: Icatibant

HYPERTENSIVE URGENCY 01:29:00
- If BP > 220/125 mm Hg but life threatening end-organ damage is absent

HYPERTENSIVE EMERGENCY/ CRISIS
- If BP > 220/125 mm of Hg + target organ damage is present
- **Goal/ Objective**: Reduce MAP by 25% within 2 hrs (Mean Arterial Pressure) or maintain BP 160 / 100 mmHg

MALIGNANT HTN 01:31:40
- Fibrinoid necrosis occurs in the vessels supplying various parts in the body and the mortality rate ↑↑ by 50% in 6-12 months
- Retina: Papilledema
- Brain: Encephalopathy
- Kidney: KFT deranged
- Blood vessel: Microangiopathic hemolytic anemia
- If a known patient of HTN on medications with an episode of abrupt rise of BP as the patient is becoming older. There can be 2 scenarios
  - Atherosclerotic narrowing of the ostia of renal artery can be present, and rule out the possibility of B/L Renal artery stenosis by Doppler – Always omit the use of ACE-inhibitors if B/L RAS is present because the BP will fall all of sudden and there can be AKI
  - Malignant HTN
  - in these patients we have to initiate the aggressive treatment, and always rule out the cocaine abuse and recreational drug abuse before starting the treatment

Stroke + HTN
- Thrombolysis (Reteplase < 4.5 hr)
- **Ischemic stroke**
  - Thrombolytic candidate
    → BP <185/110
    → Rx: Nicardipine
  - Not a thrombolytic candidate
    → BP 220/130 mmHg (first lower BP)
- **Intracerebral Hemorrhage**
  - Target BP 130-140mmHg

Rule out recreational drug abuse
- Cocaine → Severe vasoconstriction → MI → Death
  - Rave party death

Treatment of Malignant Hypertension
- Labetalol
- Nicardipine
- Sodium Nitroprusside
- Enalaprilat (ACE inhibitor given IV)
- patient with HTN encephalopathy
- Na Nitroprusside is given as first choice
- In case of the Malignant HTN (Especially in old people)
  - first choice should be Labetalol
    → Because Na Nitroprusside can cause fall in BP all of sudden
    → There is possibility that coronary ischemia might be triggered in the patient.
- In patients of Acute Ischemic Stroke with HTN > 185 / 110
- Na Nitroprusside is not preferred because, the moment we administer the drug, BP will fall fast and results in fall of CPP (Cerebral perfusion pressure)
- C.P.P = MAP – ICP, normal CPP = 60-80 mmHg)
- In these patients, thrombolysis cannot be done immediately, because first we need to lower down the BP using drugs

DRUGS USED
- Nicardipine > Labetalol > Sodium Nitroprusside
- **Candidate for thrombolysis in AIS**
  - If the patient comes to you within 4.5 hours
- **Cut off value intervention > 185 / 110**
  - first lower the BP then initiate thrombolysis because thrombolysis itself can cause brain hemorrhage and the high BP can be fatal
- **Not a candidate for thrombolysis**
  - If the patient presents late after 4.5 hours
  - lower the BP only when it is > 220 / 130 mm of Hg by using antihypertensive
- In patients with postoperative HTN / MI/ Unstable Angina/ Acute decompensated CHF
  - DOC : IV Nitro-glycerine
- In patients of "Adrenergic Crisis" in pheochromocytoma surgery
  - Nitroprusside is used along with Phentolamine

PHEOCHROMOCYTOMA
- Pre op: Oral phenoxybenzamine

- Intra op: Phentolamine
- Intra op + HTN crisis: Nitroprusside

TARGET BP

01:42:05

- Target BP/ Goal to be maintained in patients with HTN: < 135-140/85-90 mm of Hg
- Target HTN along with Diabetic nephropathy = < 130/80 mm of Hg
- BP Malignant HTN : 160/110 mm of Hg to be maintained by using antihypertensives or MAP should be ↓ by 25% over 2 hours
- CKD grade 1-III: ACEI/AERB +Thiazide+CCB
- eGFR< 30ml/min :Diruetic, Metazolone
- ESRD
  - CCB/amlodipine
  - Alpha blocker

27 DISEASES OF PERICARDIUM

ACUTE PERICARDITIS

00:00:18

Etiology

- Idiopathic (Most common)
- Viral
- Rheumatic
- Dressler Syndrome: Autoimmune pericarditis seen post MI [3-6 weeks later]
- Uremia [CKD, Diabetics, Nephropathy]
- Malignancies / 2o: Oat cell ca lung, Ca Breast

CLINICAL FEATURES

00:04:

- Patient presents with diffuse chest pain at rest for hours – days
- Radiation to left shoulder (Phrenic nerve involvement)
- Relief: - on sitting
- Pericardial friction rub heard on auscultation

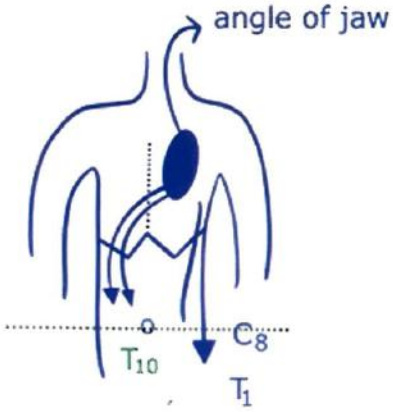
Important Information

LEVINE SIGN (MI): Chest pain (L>R) radiating

- Medial side of arm and forearm (C8, T1 dermatome)
- Epigastrium up to umbilicus- pain due to coronary etiology can extend up to T10 dermatome.
- Angle of jaw

Nerves involved in chest pain of MI / Angina

1. Superior cervical cardiac nerve
2. Inferior cervical cardiac nerve
3. Thoracic splanchnic nerve

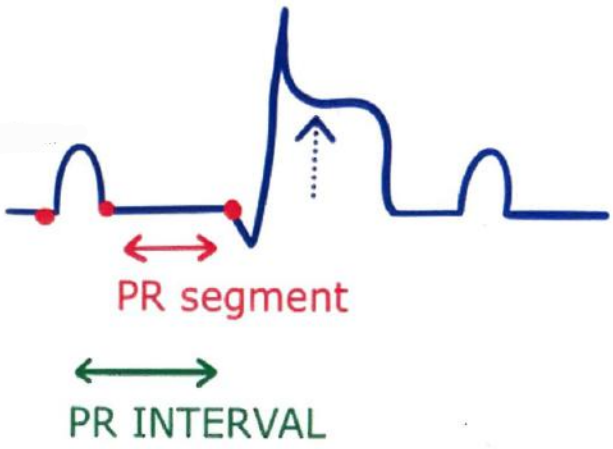


INVESTIGATION

00:09:50

ECG:

- ST ↑ concave upwards, all leads except aVR: ST ↓
- PR segment depression, all leads except aVR
- PR segment elevation



Rx

00:14:55

- Viral Pericarditis = NSAIDS
- Rheumatic Pericarditis = STEROIDS
- TB Pericarditis = ATT + short course of steroids
- 2°Pericarditis = CHEMOTHERAPY
- Uremic Pericarditis = Hemodialysis
- Dressler Syndrome = Aspirin

Complications:

- Pericardial effusion
  - Normal fluid in pericardial space= 20-50 ml
  - Pericardial effusion- fluid accumulation in pericardial space >50ml.
- ECG reveals low voltage ECG (R + S < 5 mm - Limb leads, < 10 mm = Chest leads)

Important Information

Causes of law Voltage ECG

- Restrictive cardiomyopathy (Fibrosis)
- Myxoedema heart
- Constrictive pericarditis (Calcification)
- Pericardial effusion

ELECTRICAL ALTERANS

- Duration will not change but amplitude of QRS will change
- N axis = -30 to +110

CXR: - Narrow vascular pedicle, Money bag appearance, water bottle appearance with increased CT ratio.

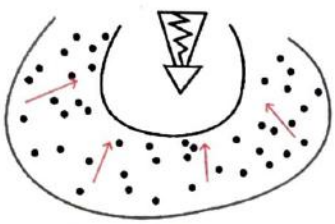
IOC: - Minimal pericardial effusion: - Echocardiography

Rx: - T/t for specific cause

CARDIAC TAMPONADE

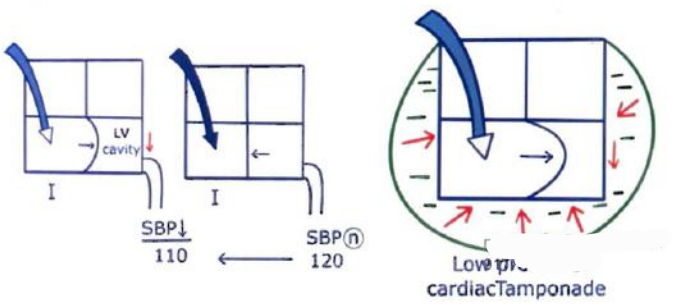
00:24:47

- Intrapericardial pressure >> LVEDP (Left ventricular end diastolic pressure)
- Diastolic collapse of ventricles
  - EDV↓ → SV↓ → CO↓ → BP↓
- Pulse becomes so weak ↔ disappear! (Pulsus paradoxus)

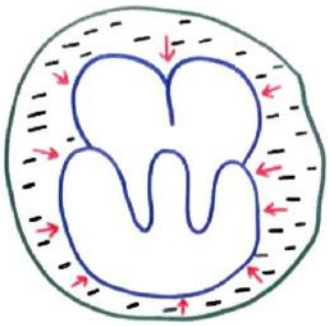


Cardiac tamponade

- Normal SBP ↓ < 10 mm Hg on deep inspiration, if > 10 mmHg (in case of cardiac tamponade) this is k/a Pulsus paradoxus



- In cardiac tamponade, there is an exaggerated septal deviation leading to reduction in LV cavity size.
- This reduces EDV, stroke volume and leads to crashing blood pressure.
  - Pulse pressure = SBP – DBP [normal 40 mmHg]
  - Cardiac tamponade = ↓SBP – DBP = ↓Pulse pressur,e Pulse disappear ,Pulsus paradoxus



Low pressure
Cardiac Temponade

- In cardiac tamponade, in inspiration SBP ↓ > 10 mm Hg – Pulsus paradoxus
- Low pressure cardiac tamponade- atrial collapse 30% ↓ filling of ventricles → HYPOTENSION
- Cardiac tamponade: Atrial + ventricular collapse
- BP decreased (unrecordable)
- Pulsus paradoxus is not seen in low pressure cardiac tamponade and Massive pericardial effusion

Important Information

- RVH → Apex superiolateral displacement • ↑CT ratio
- LVF → Apex inferiolateral displacement • ↑CT ratio
- Pericardial Effusion → ↑CT ratio • Vascular pedicle narrowing

MASSIVE PERICARDIAL EFFUSION

00:36:25

- Seen in: Hypothyroidism = Myxedema
- Lymphedema +++ (20 ml / yr) x 20 → 400ml fluid accumulation in Pericardial space
- Gradual accumulation of fluid in pericardial space. Hence ventricles adjust to this gradual rise of intrapericardial space pressure.
- So pulsus paradoxus is absent

CARDIAC TAMPONADE SUMMARY

1. Exaggerated septal deviation ++
2. Pulsus Paradoxus: SBP ↓↓, Hence an example of obstructive shock
3. BECK'S TRIAD

- MUFFLED S_1S_2
- HYPOTENSION
- Congested neck veins: Non pulsatile ↑ JVP
- Also in SVC syndrome

4. Pulsus Paradoxus + Electrical Alternans = Cardiac Tamponade

Important Information

- If electrical alternans is given alone in a MCQ, answer as Pericardial Effusion

5. Kussmaul sign absent
6. Electrical alternans present
7. Money bag appearance+
8. **TOC**: - Echo guided Pericardiocentesis (Xiphisternal approach)

Important Information

Cause of Pulsus paradoxus

- Status asthmaticus
- Massive pulmonary embolism → RV failure ↓ SBP
- Pregnancy (Gravid uterus) → Press on IVC → ↓ VR → ↓ SBP

CONSTRICTIVE PERICARDITIS

00:53:40

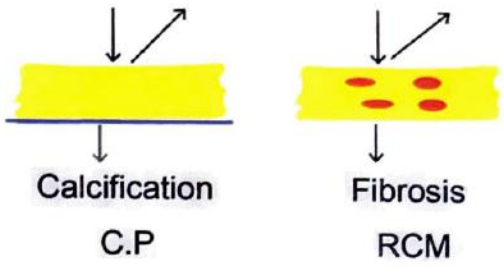
- Sequelae:
  - Pyopericardium
  - TB Pericarditis Compliance is decreased

Pericardial shudder knock/shock

Right Ventricle	Left Ventricle
1. Pedal Oedema	1. Pooling of blood in the lungs
2. Hepatomegaly (RUQ discomfort)	2. Pulmonary edema
3. Ascites	3. Dyspnea
	4. Low CO
	5. Effort intolerance

O/E

1. Pulsus Paradoxus (Rare)
2. JVP: - Kussmaul sign (+)
   - (Paradoxical rise of JVP)
   - Prominent x, prominent y descent
3. Pericardial shudder / knock/ shock
4. Liver span ↑
5. B/L fine crepts +
6. Puddle sign + Ascites



Diagnosis

- Pericarditis V/s RCM
  - Compliance ↓ - calcification in C. pericarditis
  - Fibrosis in RCM
- **CXR:** Egg in a cup appearance
- Egg on side appearance = TGA
- **ECG:** Low voltage ECG
- Electrical alterans : absent
- **CT chest;** calcification outside the heart of the patient
- **Cardiac MRI**
- **Doppler:** Square root wave sign
- **Endomyocardial Biopsy:** Gold Standard investigation to differentiate RCM from constrictive pericarditis

TREATMENT

01:03:00

Pericardial stripping

Refer Table 27.1

Table 27.1

	Acute pericarditis	Pericardial effusion	Cardiac tamponade	Constrictive pericarditis
ECG	ST ↑ Concave	Electrical alternans	Electrical alternans + Pulsus paradoxus	Low voltage ECG + Pulsus paradoxus
	Kussmaul sign ⊖	Kussmaul sign ⊖	Kussmaul sign ⊖	Kussmaul sign ⊕
JVP			Absent y	Prominent y
			Prominent x	
			BECK's Triad	
Treatment	Cause specific	Pericardiocentesis	Pericardial stripping	

20 CONGESTIVE HEART FAILURE

Cardiac output = Heart rate X stroke volume
- ↑ HR → ↑ Sympathetic drive

Clinical features:
1. Palpitations
2. Diaphoresis
3. Dyspnea on exertion / rest
4. Nocturnal cough
5. Paroxysmal nocturnal dyspnea (Orthopnea)
6. Pink frothy sputum
7. Effort intolerance

On examination
- Tachycardia
  - Maximum HR = 220 - Age

Left ventricular failure	Right ventricular failure
• ↓ SBP leads to oliguria • S3 present: Ventricular gallop rhythm • B/L fine crepitations • ↓air entry B/L • ↓Vital capacity	[m/c/c of RVF is LVF] • JVP ↑ • Kussmaul signs → JVP ↑ on Inspiration • Abdomino- jugular reflux • Tender hepatomegaly • B/L pitting pedal edema [Chronic CCF]

FRAMINGHAM CRITERIA: FOR DIAGNOSIS OF CHF

00:14:00

Minor Criteria
1. Dyspnea On exertion
2. Nocturnal cough
3. Tachycardia
4. Pleural effusion
5. ↓ Vital capacity > 1/3rd
6. Tender hepatomegaly
7. Pitting pedal edema
8. Oliguria

Major Criteria = Rest all are major criteria.

Important Information

For Diagnosis
- 2 major criteria should be present or
- 1 major + 2 minor

Framingham heart risk calculator
- To determine chances of coronary events in next 10 yrs (risks)

STAGES OF CHF (ACCORDING TO ACC)

00:17:22

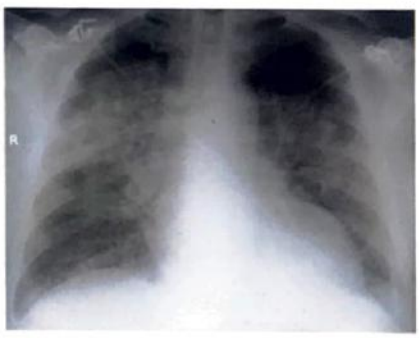
Stage A → No structural damage, high risk of developing symptoms (risk factors present)
Stage B → Structural heart disease + No symptoms of CHF
Stage C → Structural heart disease + Symptoms of CHF
Stage D → Refractory heart failure

WORK UP

00:20:28

1. **BNP levels / N-terminal Pro BN**
- Released from ventricles of heart
- If > 100 pg/ ml – It differentiate from non-cardiogenic pulmonary edema

2. **Chest X-ray**



a. Earliest radiological finding: Prominent upper lobe veins / Antler sign or Reverse moustache sign
b. CT ratio increased > 0.5
c. Bat wing pulmonary edema
d. Costo – phrenic angle blunting
e. B/L pleural effusion
f. Kerley B lines → Perpendicular to pleural surface. Due to thickened interlobular septa

- Also seen in lymphangitis carcinomatosis (Kerley A lines: Present in the periphery of hila
- Due to distension of anastomotic channels between central and peripheral lymphatics Kerley C lines: Reticular opacities at lung base.)

Important Information

- Reverse Bat Wing Edema- Chronic Eosinophilic Pneumonia
- Moustache sign – Pneumoperitoneum (gas under diaphragm)

3. **Echocardiography**

- Ejection fraction = $\frac{\textit{Stroke volume}}{\textit{End diatolic volume}}$
- In systolic malfunction = EF ↓
- In diastolic malfunction = EF can be normal
- In HTN patient → Left ventricular end diastolic pressure increase
- Pooling of blood in lungs

4. **Cardiac MRI**
- Preferred imaging
- Type of malfunction: Systolic Vs Diastolic
- Etiology: Fibrosis / Ischemic / Hypokinesia / DCM
- Prognosis

5. **BORG scale**- Tells functional assessment of the patients

Refer Table 28.1

TREATMENT

00:33:57

Heart failure with preserved EF (HFpEF)
- Preserved EF ~ 50%

Seen in:
- HTN, RCM, Radiation
- CTD, aging
- Endomyocardial fibro elastosis
- Hemochromatosis

Rx

I. Aldosterone antagonist

II. ARNI → Valsartan (ARB) + sacubitril (nephrilysin → inhibit degradation of BNP)

Digoxin
- Ineffective
- ↑O_2 demand of heart
- Can be deterimental

Heart failure with reduced EF (HFrEF)

Seen in:
- CAD: Ischemic cardio myopathy
- HTN: recent onset
- Valvular Lesion
- L→R shunt
- Cor Pulmonale
- Chagas diseases / Ch. Arrhythmias

Management

Treatment of acute pulmonary edema

L → **L**asix (Furosemide)
M → **M**orphine (↓Pulmonary edema)
N → **N**itroglycerine
O → **O**xygen (NIV)
P → **P**ositioning

How to remember

- **LMNOP**

Vasodilators
- Nitroglycerine
- Nesiritide
- Nitroprusside

Diuretics
- Furosemide

Nesiritide
- BNP Analogue

Treatment of Acute decompensation with cardiogenic shock

1. Vasopressers
   - Nor-epinephrine → DBP↑ → CBF↑
   - Dopamine : improves GFR also
   - Dobutamine : β_1 → ↑HR → ↑ O_2 consumption
2. Inodilators : Milrinone & Amrinone
3. Levosimendam
   Omecamtiv→ ↑production of stronger actin-myosin complex

Acute CHF / HFrEF: <40% of EF

Cardiogenic Shock

- Vasopressors

Volume overload

L → Lasix (Furosemide)
M→ Morphine (↓Pulmonary edema)
N → Nitroglycerine
O → Oxygen (NIV)
P → Positioning

Medically Refractory Cardiogenic Shock

1. LVAD: LV assist device: heart – mate
2. CRT: Cardiac resynchronization therapy
3. Cardiac transplantation

Digoxin toxicity

- V. Bigeminy (MC arrythmia)
- Non paroxysmal atrial tachycardia with AV block

Rx

- Lignocaine

CHRONIC CHF

- ↓Post discharge
  - Intiate with ARNI: Valsartan, Sacubitril
  - ACEI + βblockers
  - Spironolactone

Heart failure

HF with preserved ejection fraction EF ≥50%

Causes

- Long standing HTN
- ARNI
- Aldosterone antagonist
- ACE inhibitors

HF with reduced ejection fraction EF <40%

Causes

- Acute pulmonary edema
  - S. anemia with CHF
  - T3 pregnancy
    → LMNOP
    → ARNI
    → ACEI+ βblockers
- IVABRADINE
  - Cardiogenic shock
    → Vasopressors

Table 28.1

RPE Scale	Rate of perceived Exertion
10	**Max effort Activity** Feels almost impossible to keep going. Completely out of breath, unable to talk. Cannot maintain for more than a very short time.
9	**Very Hard Activity** Very difficult to maintain exercise intensity. Can barely breath and speak only a few words
7-8	**Vigorous Activity** Borderline uncomfortable. Short of breath, can speak a sentence.
4-6	**Moderate Activity** Breathing heavily, can hold short conversation, still somewhat comfortable, but becoming noticeably more challenging.
2-3	**Light Activity** Feels like you can maintain for hours. Easy to breathe and carry a conversation
1	**Very Light Activity** Hardly any exertion, but more than sleeping, watching TC, etc.

29 BUNDLE BRANCH BLOCK

NORMAL ECG

00:00:13

1. Check HR - Calculate R-R interval by looking at long lead II by counting no. of large squares then,

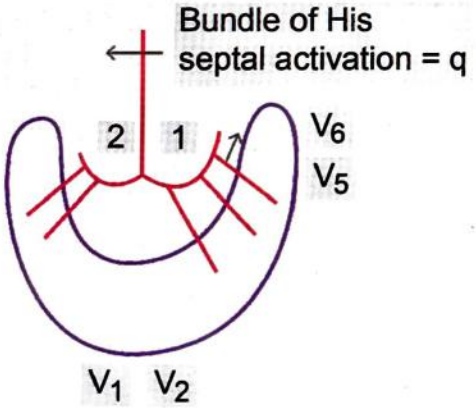
$$\frac{300}{\textit{No. of large squares}} = \text{No. of beats/min}$$

2. Calculate Axis
- In given ECG Lead I – R wave is much bigger than S wave R-S = +ve and R-S = +ve in aVF → ECG shows normal axis

3. Check if every P wave followed by QRS (Normal P wave = < 120 msec)
- In the given lead II - P wave is three small squares and vertical height is < 2.5 mm → Normal and also check R-R interval is constant or not

4. Check for ST segment changes like ST – elevation, depression, coving
5. Height of T wave in precordial lead should be < 10 mm
- Height of T wave In limb leads = < 5 mm and In chest leads = < 10 mm

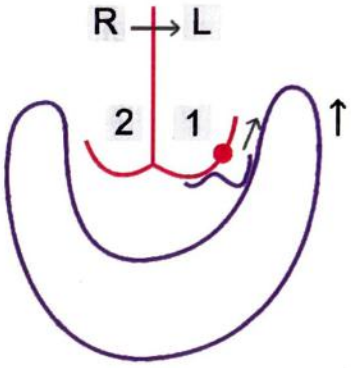
6. Check for QT interval, and Look for 'q' wave in V_5 & V_6
7. Look for progression of R & S wave in V_1 to V_3 leads from V1 to V_3 → R & S become equal in amplitude in lead V_3 and from V_3 → V_6, R becomes bigger than S (progression of R wave)



- Normally – In Bundle of His- current goes to left fascicle and later on to right fascicle - Red arrow (Vector) signifies septal activation which is responsible for generation of 'q' wave, which is away from lead V_5 & V_6.

So it is becomes -ve wave

LBBB



Rabbit ear = M

- Intermyocyte conduction → Broad QRS
- Damage in left fascicle so refractory period of left side is more than right
- Current will go to right side first
- Vector changes w.r.t. change in septal activation (towards right side)

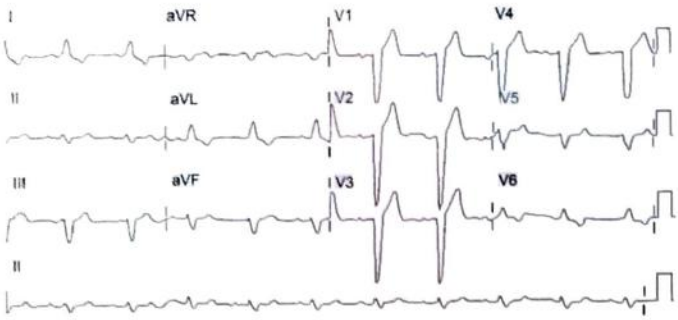
C/F
- Absent 'q' wave in lead V_5 and V_6
- Tall & Broad R wave in V_5 and V_6 (lateral leads)
- Deep & Broad S
- In the peak of 'R' wave -Rabbit ear appearance seen
  - Current travelling via Intermyocyte pathway.
  - Sequential activation of ventricles (instead of simultaneous activation which occurs normally),
  - Ventricles contract independent of each other, results in pooling of blood in lungs cause pulmonary edema
  - LBBB is lethal

Important Information

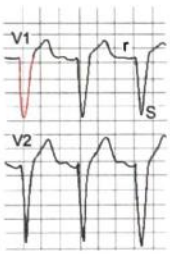
- A new onset LBBB is suggestive of MI as anterior wall MI damage conduction system of heart

ECG

00:13:14



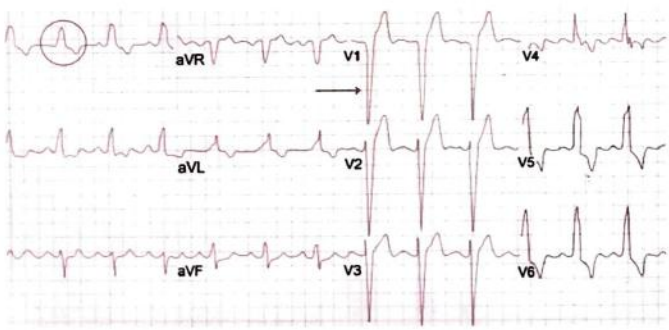
- R-S: +ve on Lead 1
- R-S: -ve on lead aVF
- Axis : Left axis deviation
- Deep 'S' in V_1 & V_2 as the abnormal current is going away from V_1 & V_2
- Discordant ST segment changes (due to abnormal repolarization & depolarization)
- ST/T wave move opposite to the predominant QRS vector
  - e.g. in V_5/V_6 → Tall R wave is seen and also ST depression is seen and in V_1/V_2 deep 'S' and ST segment elevation is also seen.
- QRS (Broad QRS) duration is increased d/t wastage of time (Normal QRS = 80-100 ms)
  - In ECG having Broad QRS –due to conduction delay, HR= Normal
  - Presence of William pattern seen "Rabbit ear" in dominant S & peak of R, W is seen at V_1, M is seen at V_6



CLINICAL SCENARIO

00:21:24

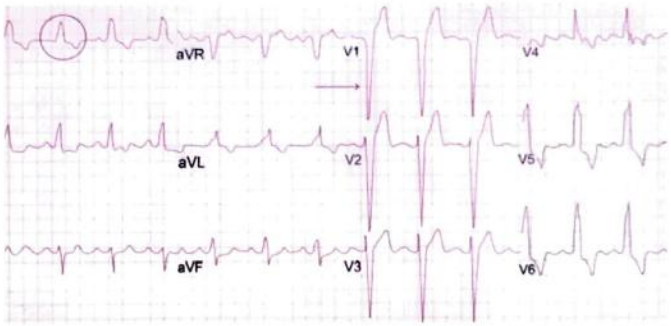
ECG



- ST segment depression
- Predominant broad S waves present in lead V_1 & V_2
- Discordant ST segment changes present
- Absent Q wave
- Broad QRS
- Rabbit ear appearance in V_5

Diagnosis: LBBB

- ECG (LBBB)



  - HR = 300/ min
  - Lead I = +ve, aVF = -ve so there is LAD (always plot the axis to rule out the axis)
  - Broad QRS complex
  - In precordial leads- Dominant 'S' lead V_1 & V_2
  - Failure of progression of R wave
  - Broad dominant R wave in V_5 & V_6
  - Absent q wave

Important Information

- William's is not always applicable

SGARBOSSA CRITERIA

00:24:41

- To diagnose an MI when LBBB is present

Important Information

- New onset LBBB could be present in MI & MI diagnosis can be missed in the patient having LBBB
- The patient can have MI as a cause of LBBB

Causes of LBBB

A - Aortic Stenosis
A - Ant wall MI
D - Digoxin Toxicity (most common – Ventricular bigeminy)
D - Dilated cardiomyopathy
H - HTN
H - Hyperkalemia

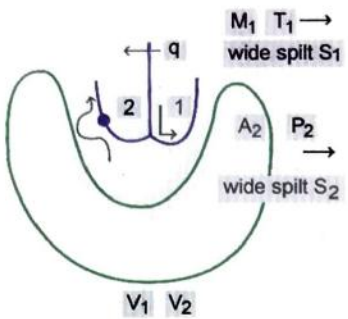
How to remember

- A2 D2 H2

RIGHT BUNDLE BRANCH BLOCK (RBBB)

00:26:23

- Defect in Right bundle branch
- Left ventricle activated normally

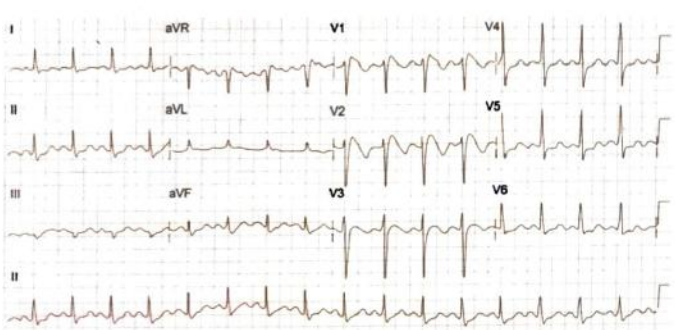


C/F

1. rSR pattern in V_1& V_2. It is also looking like a rabbit ear appearance

2. V5/V6 W pattern and M pattern in V_1/V_2
3. Dominant & Broad S in V5/V6

ECG



- Broad QRS
- M_1 T_1 shifted laterally → Wide split S_1
- P_2 shifted → Wide split S_2
- rSR pattern in V_1&V_2-d/t intermyocyte conduction wrt Right fascicle to the patient

Cause of RBBB

- RVH
- Pulmonary embolism
- Cor pulmonale
- IHD, RHD, Cardiomyopathy.

LBBB VS RBBB

00:31:53

	LBBB	RBBB
V_1 V_2	Dominant S	rSR pattern or M pattern Dominant S
V_5 V_6	Tall & Broad R wave absent q wave	
	WILLIAM	MARROW

CLINICAL QUESTIONS

Q. A 45-year-old female tourist to India presents with diffuse chest pain. There is no past history of heart disease, hypertension, and diabetes. She is admitted for observation and next morning she has a recurrence of chest pain and ECG shows ST elevation in V1 to V4. Troponin is normal and pain is promptly relieved by sublingual NTG. Which of the following is the best management plan for this patient?

A. Echocardiography and anti-inflammatory therapy
B. Exercise stress testing and long-acting nitrates
C. Coronary angiography with long-acting nitrates and CCB
D. Thallium scan and long-acting nitrates

Answer: C

Solution

- Precordial chest pain, often occurring at rest during stress or without known precipitant, **relieved rapidly by nitrates.**
- **ECG evidence of ischemia during pain**, sometimes with ST-segment elevation.
- **Angiographic demonstration of: No significant obstruction** of major coronary vessels.
- Coronary spasm that responds to intra-coronary nitroglycerin or calcium channel blockers.

Prinzmetal Angina

- **Prinzmetal (variant) angina** is a clinical syndrome in which chest pain occurs without the usual precipitating factors and is associated with ST-segment elevation rather than depression.
- It often affects women under 50 years of age.

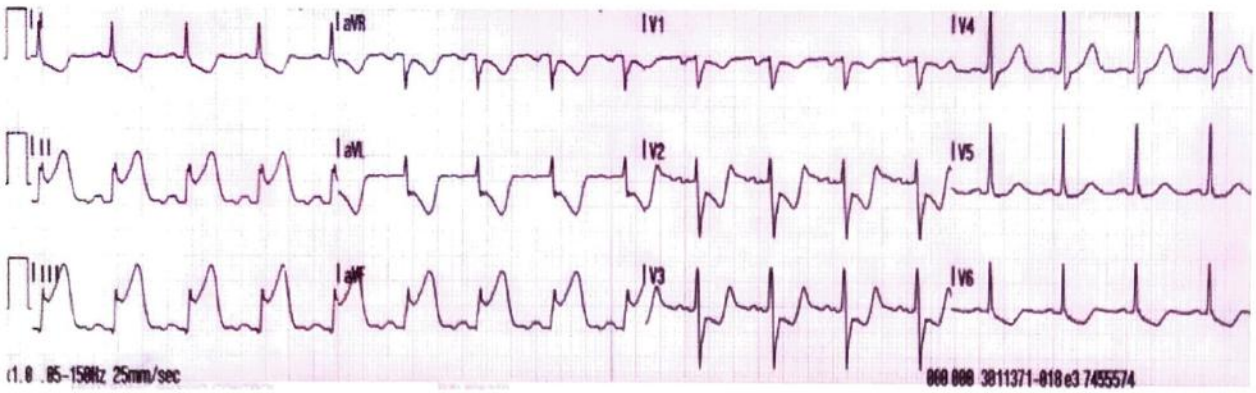
Treatment

- **Nitrate and calcium channel blockers are the main therapeutic agents.**
- Asprin
- Statin therapy
- Coronary revascularisation
- ICD

Choice 2 and 4 are done for chronic stable angina
Choice 1 will offers no diagnostic help.

Reference: Harrison'S Principles Of Internal Medicine 20th Edition page no :**1872**

Q. A 65-year old man presents with crushing chest pain for 2 hours. On examination, BP = 80/60 mm Hg and JVP are elevated 4 cm above the sternal angle. All are true about the condition shown except:



A. ST elevation in V4R

B. Anterior wall MI

C. Right ventricular infarction

D. Kussmaul sign +

Answer: B

Solution

The ECG shows

- Sinus rhythm is present with a normal QRS axis.
- **ST elevation >1 mm in V4R and leads II, III, aVF**
- ST depression (reciprocal) is present in anterior leads.
- Hence the diagnosis is **right ventricular MI.**
- Right ventricular infarction leads to hypotension with clear lung fields and presence of elevated JVP (Kussmaul sign +).

Anterior wall MI shows changes in V1-V4, I & aVL (ST elevation)

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no Page no **1680**

CLINICAL QUESTIONS

Q. A 23yrs old, male patient came to your OPD with history of syncopal attacks. He was anxious and revealed about similar history& sudden death in his sibling 2yrs ago. On further work up, ECG showed characteristic 'Saddle back pattern' in V1-V2 leads. Which of the following statement is incorrect about this condition?

A. SCN5A defect
B. Asymptomatic ST-segment elevation
C. Sudden death
D. Pacemaker is treatment of choice

Answer: D

Solution
Brugada syndrome

- Due to a **diminished inward sodium current** in the region of the RV outflow tract epicardium.
- A loss of the action potential dome in the RV epicardium due to **unopposed ITo potassium outward current** results in dramatic shortening of the action potential.
- The **large potential difference between the normal endocardium and rapidly depolarized RV outflow epicardium** gives rise to ST-segment elevation in V1-V3 in sinus rhythm and predisposes to local ventricular reentry.
- Autosomal dominant inheritance pattern, the arrhythmia syndrome is most common in young male patients (-75%) and is thought to be responsible for the **Sudden Unexpected nocturnal Death Syndrome (SUDS)**
- For patients who have had syncopal attacks, **I.C.D. implantable cardioverter defibrillator is used** as it can deliver a DC shock to terminate the arrhythmia developing in these patients and not pacemaker.

Reference: Harrison 19th ed. 1497-98/ Harrison 20th p 1756; Harrison 19th p 1497-98

Q. A 56-year-old female presents with severe chest pain and breathlessness after coming back from cremation grounds due to death of her husband. ECG is performed which shows ST elevation in the precordial leads, most noticeably in V2-V3. Troponin T and BNP are mildly elevated. Invasive coronary angiography shows completely normal coronary arteries. A transthoracic echocardiogram demonstrates apical akinesis and a left ventricular ejection fraction of 40% . What is the most likely diagnosis?

A. Anxiety
B. Septal myocardial infarction
C. Coronary artery spasm
D. Takotsubo cardiomyopathy

Answer: D

Solution

- **Angiography is Normal in spite of all finding of MI**
- Apical Akinesia & low ejection fraction
- Both pointing towards Takotsubo cardiomyopathy
- Tx= Intraortic balloon pump (IABP)

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no :1790

CLINICAL QUESTIONS

Q. A 911 call brought in 20yr old unconscious patient from a party. Patient attender informed the physician that before fainting, the patient complained of chest pain, racing of heart feeling & shortness of breath. On examination the pulse was very fast and BP was 76/50 and ECG recording showed absent P waves and irregular QRS complexes. TTE revealed clots in left atrium. On managing which of the following should not be done?

A. Diltiazem to reduce the heart rate
B. Warfarin therapy
C. Open mitral commissurotomy and removal of clot
D. Cardioversion with percutaneous balloon valvotomy

Answer: D

Solution

- Atrial fibrillation with retained clots is due to the stasis of blood in the poorly contracting Left atrium.
- If **cardioversion** is done in this setting, the normal sinus rhythm ensues and the **proper contraction of atria may result in dislodgement of clots from the atria.**
- The **PMV procedure** dilates the mitral annulus, relieves the annulus and the chances of clots embolizing via a bigger orifice increase.
- If **cardioversion** is indicated more **urgently**, then intravenous **heparin** should be provided and TEE performed to exclude the presence of left atrial thrombus before the procedure.

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no :**1816; Harrison 19th p 1542**

Q. A 50yr gentleman came into OPD with complaints of weakness, abdominal pain and constipation for past 3-4 days. On examination abdominal distention was noted. Both the lung fields are clear. CVS examination was normal. ECG was taken and it showed height of P wave to be more than 2.5mm in limb leads. Which of the following electrolyte abnormality could produce this Pseudo P-Pulmonale finding?

A. Hypokalemia
B. Hyperkalemia
C. Hypomagnesemia
D. Hypercalcemia

Answer: A

Solution

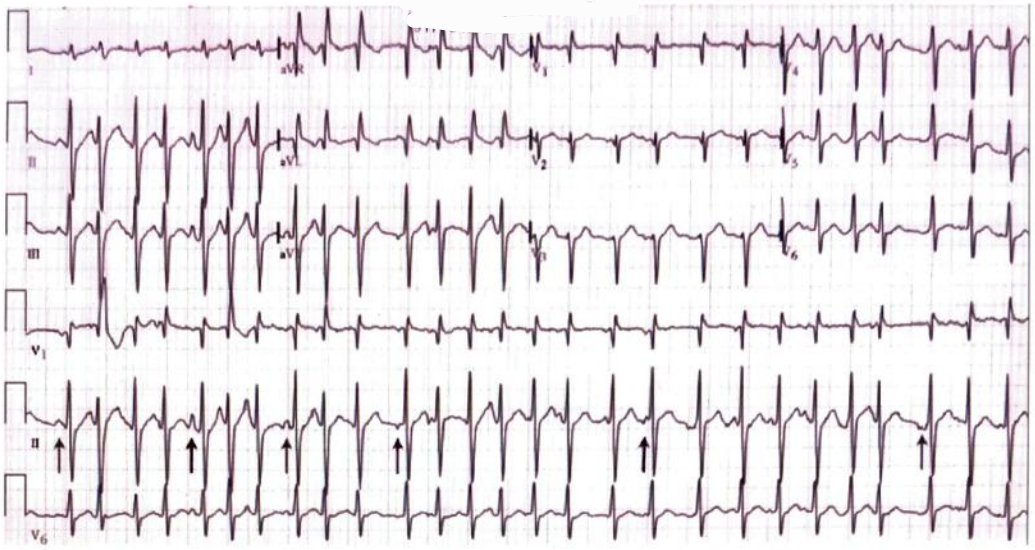
- P-Pulmonale = Height of P wave > 2.5 mm in limb leads of patient seen in patient of PAH.
- **Pseudo P-Pulmonale=** Height of P wave > 2.5 mm in limb leads in **absence** of PAH

Option	Electrolyte imbalance	ECG findings
Option A	Hypokalemia	T becomes smaller, inverted P becomes vertically higher, seems like P pulmonale
Option B	Hyperkalemia	Tall tented T wave
Option C	Hypomagnesemia	QT Prolongation
Option D	Hypercalcemia	QT shortening

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no : 1682

CLINICAL QUESTIONS

Q. Comment on the diagnosis of the patient based on ECG



A. Multifocal atrial tachycardia
B. Atrial fibrillation
C. Mobitz II heart block
D. Wolf Parkinson white syndrome

Answer: A

Solution

- **HR** of patient is **variable**. Notice the **change in RR interval** in lead II. The HR changes from 200 bpm to 150 bpm.
- Notice the **changes in amplitude of P waves** which leads to diagnosis as **Multifocal atrial tachycardia(MAT)**.
- **Choice 2** is ruled out as **A. fibrillation** has an **absent P waves**
- **Choice 3** is ruled out as **Mobitz II heart block** has **bradycardia**
- **Choice 4** is ruled out as **Wolf Parkinson white syndrome** has **Delta waves** with broad qRS complex is not seen

Reference: Harrison 20th ed. p. 1743

Q. Jayaraj, a 55 years old male presented in civil hospital with breathlessness and chest pain. ECG was done and diagnosis of Myocardial infarction was made. After initial management, he was referred to District hospital where Alteplase fibrinolytic therapy was started. after 5 mins of starting Alteplase, ECG monitor of patient started to show >3 consecutive premature ventricular contractions (PVC) with a heart rate of less than 100 bpm. The diagnosis is?

A. Sustained VT
B. Non-sustained VT
C. Accelerated idioventricular rhythm
D. Ventricular flutter

Answer: C

Solution

> 3 consecutive premature ventricular contraction & HR is <100 bpm → **AIVR** (Accelerated Idioventricular Rhythm)

Accelerated Idioventricular Rhythm/Slow ventricular tachycardia

- It is mc Reperfusion Arrhythmia
- Occurs during fibrinolytic therapy at the time of reperfusion.

1. If H.R > 100/min - V. Tachycardia
2. If H.R <100/min- AIVR

Reference: Harrison's 20th edi/pg1757

CLINICAL QUESTIONS

Q. Leela devi, a 46 year old female presented to OPD with complains of diffuse chest pain at rest , breathlessness and fatigue for 2 days. She gives recent history of cough, fever and rhinorrhea lasting for 3 days. O/E - BP= 118/ 70 mm Hg, Pulse = 80 bpm, Temp. = 99° F. Frictional rub is heard on auscultation. ECG was done whose findings are shown below. Diagnosis is ?

A. Acute pericarditis
B. Constrictive pericarditis
C. Takotsubo cardiomyopathy
D. Cor-pulmonale

Answer: A

Solution
The above case scenario is diagnostic of Acute Pericarditis. The points in favour of diagnosis are
1. Sinus rhythm at approx. **75 beats/min**.
2. The QRS axis and intervals are normal.
3. **ST elevations with concave upward** morphology are seen in I and aVL, II and aVF, and V2 through V6.
4. The upward concavity of the ST segment, the PR-segment depression, the lack of Q waves, and the diffuse nature of the ST-segment elevation in more than one coronary artery distribution make **Pericarditis** the likely etiology.
5. Patients with pericarditis will complain of chest pain, typically described as sharp and pleuritic. Radiation of the pain is towards trapezius ridge. The **pain is improved with sitting up and leaning forward and worsened by leaning backward.**

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no : 1842

Q. A 36 years old male presented to OPD with complains of mild chest pain, difficulty in breathing, low grade fever ,fatigue and swelling over both ankles . O/E - Pulsus paradoxus + , JVP shows +ve Kussmaul sign, increased liver span , positive Puddle sign and absent apex beat. On auscultation - Soft S1, S2, Pericardial knock + , B/L fine crepts +, .CXR shows Egg in a cup appearance and doppler shows Square root wave sign. What is your likely diagnosis ?

A. Cardiac tamponade
B. Restrictive cardiomyopathy
C. Constrictive pericarditis
D. Acute pericarditis

Answer: C

Solution
- Pericardial knock- early diastolic sound (earlier than S3) indicates decreased ventricular compliance due to an external force heard in patients with constrictive pericarditis
- **S3 is an early diastolic sound due to rapid ventricular filling with a large volume of blood** so occur in patients with hyperdynamic circulation example: pregnancy, anemia, A-V shunts and congestive heart failure
- **S4 filling of ventricle against a stiff ventricle so the atria** have to give a final kick at end of diastole to completely empty itself

Constrictive pericarditis

- Pulsus paradoxus
- Deep x descent
- Rapid y descent
- Prominent 'v'
- Absent Apex beat
- S1 S2=soft
- S3 S4= Never seen
- Third heart sound=Pericardial Knock
- Doppler = Square root wave sign
- CXR = Egg in a cup appearance

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no : 1846

CLINICAL QUESTIONS

Q. A 75-year-old man is brought to the casualty with sudden syncopal episode while playing with his grandchildren. He is currently alert and describes occasional substernal heaviness and shortness of breath. His lungs have bibasilar rales and BP is 120/80mmHg. Which is the classical finding expected in this patient?

A. Ejection systolic murmur with Soft S2
B. Ejection systolic murmur with wide split S2
C. Harsh Holosystolic murmur with soft S2
D. Harsh pan-systolic murmur with loud S2

Answer: A

Solution
The clinical **presentation of exertional syncope**, angina equivalent and dyspnea in geriatric age group points to diagnosis of valvular Aortic stenosis.

In aortic stenosis
- Soft S2(due to poor mobility of a **stenosed valve)**
- REVERSE S2 SPLIT
- S3, S4 in advanced cases
- Ejection Click
- Ejection systolic murmur
- Pulsus et tardus

So option A is correct
- Choice B is wrong because in aortic stenosis, narrow split S2 or paradoxical split S2 is heard.
- Choice C and D are wrong due to presence of Ejection systolic murmur in aortic stenosis.

Points to remember
- **Ejection systolic murmur** = aortic stenosis / pulmonary stenosis
- **Pan systolic murmur** = VSD, chronic MR, Chronic TR
- **Wide split S2** = VSD, MR, Pulmonary stenosis, RVF
- **Loud S2** = Pulmonary hypertension, systemic hypertension

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no :**1531**

Q. 55 years old patient Lakha Singh presented to OPD with complains of weakness and fatigue, dyspnea on lying down and palpitations. Symptoms aggravate at night time when he tries to sleep at bed. He gives h/o of Rheumatic heart disease in past . O/E - BP = 116/ 80 mm Hg, Pulse = 86bpm , elevated JVP, Hyperdynamic Apex beat with apical thrill on palpation. ECHO was done which revealed Mitral regurgitation. A diagnosis of Chronic Mitral regurgitation was made. Which of following asucultatory finding is false regarding Chronic Mitral Regurgitation?

A. Mid-diastolic murmur
B. Wide split S2

C. Pansystolic murmur radiating to axilla

D. Narrow split S1

Answer: D

Solution

- **Narrow split S1** is a feature of mitral Stenosis. Choice D is incorrect.
- **In case of Mitral Regurgitation,** the extra blood that flows into left atrium has to come back to left ventricle during diastolic phase. This leads to **mid diastolic murmur** .Choice A is Correct.
- Due to **early A2**, wide Split S2 is heard. Choice B is correct
- The most characteristic auscultatory finding is grade 3/6 pansystolic murmur. Choice C is correct.

Chronic MR features

- **Primary murmur: Pansystolic in chronic MR, early systolic in acute MR**
- S1 = soft
- S2 = Wide split = Early A2
- S3++
- S4++ = extreme LV dilatation
- Opening snap = absent
- Secondary murmur = severe MR = Mid-diastolic murmur

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no : 247

CLINICAL QUESTIONS

Q. A 65-year-old woman with type 2 diabetes mellitus and hypertension comes to the office for routine follow-up. She has occasional numbness in her feet. The patient takes ibuprofen for chronic back pain along with hydrochlorothiazide and metformin. Urinalysis shows albuminuria. Lisinopril therapy is initiated . The next day, the patient returns due to lightheadedness and near-syncope. Her blood pressure is 80/45 mm Hg . Which of the following is most likely the major factor contributing to this patient's current symptoms?

A. Autonomic neuropathy
B. NSAID use
C. Diabetic nephropathy
D. Diuretic therapy
E. Unilateral renal artery stenosis

Answer: D

Solution

- This patient with albuminuria was started on an **ACE inhibitor for treatment of early diabetic nephropathy**.
- Although most patients remain asymptomatic with only a mild reduction in blood pressure, first-dose hypotension can be a potential limiting factor when initiating ACE inhibitors.
- **Significant hypotension is most likely to occur in patients with high plasma renin activity, such as those with volume depletion** (eg, from diuretic use [hydrochlorothiazide in this patient]) or heart failure.
- **To prevent the development of first-dose hypotension, therapy should be started at low doses and slowly titrated upward as needed.**

Reference: Harrison's Principles Of Internal Medicine 20th Edition page no : 846;

Q. 72 year man came to emergency department complaining of headache & dizziness. His BP is 190/126. Further examination revealed retinal hemorrhages, papilledema and creatinine levels of 3.5 mg/dl. How would you manage this patient?

A. Bring down mean arterial BP (MAP) by no more than 25% within 1 hour
B. Bring down MAP by no more than 30% within 1 hour
C. Bring down MAP by no more than 25% within first hour
D. Bring down MAP by no more than 30% within 2 hour

Answer: A

Solution

Hypertensive emergency:

- Severe elevations in **BP (>180/120 mmHg)** complicated by evidence of **impending or progressive target organ dysfunction** (hypertensive encephalopathy, retinal hemorrhages, papilledema, or acute and subacute kidney injury)

Treatment

- Bring down **mean arterial BP (DBP + 1/3 PP)** by no more than **25% within 1 hour**.
- If stable→Reduce further to 160/100 mm Hg within the next 2 to 6 hours.

- Reduce further to normal during the next 24 to 48 hours.
- It should always be managed with **parenteral drugs.**

Hypertensive urgency is severe hypertension with **no signs or symptoms of acute end-organ damage.**

Note: If the patient has a condition such as severe pre eclampsia or eclampsia or pheochromocytoma crisis, SBP should be reduced to <140 mmHg during the first hour and to <120 mmHg in aortic dissection.

Reference: Harrison's 20th edi/1893

CLINICAL QUESTIONS

Q. A 25yr old woman comes to OPD with complaint of chest pain for past 10hrs. She also mentions that she gets fatigued and feels breathless while doing routine activities. On auscultation early diastolic murmur is noted and ECG showed findings of LVH. Physician suspects a particular valvular condition. So he gently presses the femoral artery using stethoscope and a to & fro murmur is heard. This Finding is seen in which valvular heart condition?

A. Aortic Regurgitation
B. Aortic Stenosis
C. Mitral Stenosis
D. Mitral Regurgitation

Answer: A

Solution
Duroziez's sign is seen in severe **aortic regurgitation**
1. Duroziez Sign → Gently press the femoral artery using stethoscope
↓
Murmur in heart d/t turbulence

To and fro murmur
Arterial pulse seen with aortic regurgitation
2. Corrigan's pulse: Water hammer pulse
3. Quincke's sign: Flushing and blanching at distal part of nail
4. Traube Sign: Pistol shot sounds heard at femoral artery

Reference: Harrison'S Principles Of Internal Medicine 20th Edition page no :1810

Q. Lalit, a 19 years old male presented to cardiac opd with complains of dyspnea and chest pain following excercise. His elder brother had same symptoms and he died 2 years ago by sudden collapse while participating in a marathon event. O/E - Jerky pulse and double apical impulse on palpation, Narrow split S2 and Crescendo decrescendo murmur is heard on auscultation. Genetic analysis showed the disease to be a/w multiple genes . Which of following is the likely diagnosis ?

A. Mitral Valve Prolapse
B. Hypertrophic Obstructive Cardiomyopathy
C. Dilated Cardiomyopathy
D. Arrhythmogenic Right Ventricular Cardiomyopathy

Answer: B

Solution
- Familial HOCM: AD Mendelian inherited disease in approximately 50% of cases.
- MC gene involved: **β-myosin heavy chain > myosin-binding protein C > troponin T.**
- In Arrhythmogenic right ventricular dysplasia the **ventricular wall is severely thinned as a result of myocyte replacement by massive fatty infiltration and lesser amounts of fibrosis.**

Reference: Harrison 20th p 1794

LEARNING OBJECTIVES

EMERGENCY MEDICINE

BASIC CARDIAC LIFE SUPPORT

- Introduction
- Cardiac Arrest
- Steps of Basic Life Support
- Adult Basic Life Support Algorithm
- Requirement of Basic Life Support

PULSELESS ELECTRICAL ACTIVITY

- Pulseless Electrical Activity
- Outcomes by Diagnosis
- Causes of Pulseless Electrical Activity
- Interventions
- Management

ADVANCED CARDIAC LIFE SUPPORT

- Introduction
- How is ACLS different from BLS
- Difference between Defibrillation and Cardioversion
- Shockable and Non-Shockable Rhythm
- Treatment of Reversible Causes
- Algorithm in Advanced Cardiac Life Support

MECHANICAL VENTILATION STRATEGY

- Invasive Mechanical Ventilation
- Assist Control Mechanical Ventilation
- Synchronized Intermittent Mandatory ventilation
- Waveform Analysis
- Pressure Support Ventilation
- Basic Rules of Ventilation
- N.IV/CPAP

MASSIVE TRANSFUSION PROTOCOL

- Introduction
- Bloody Vicious Cycle
- Triggers Initiating
- Set-up
- How to Initiate MTP
- Challenges in MTP
- Complications of MTP

ARDS

- Introduction
- Cardiogenic Pulmonary Edema and Non Cardiogenic Pulmonary Edema
- Triggers (Direct and Indirect)
- Phases of ARDS (Exudative Phase)
- Features (Exudative phase, Proliferative phase and fibrosis)
- Work up
- Diagnostic Criteria for ARDS
- BERLIN CRITERIA
- Treatment
- Leading causes of Death in ARDS
- Difference between CHF and ARDS

SNAKE BITE

- Introduction
- Snakes present in india
- First aid-do it right
- Nitrogesic ointment/nitrate spray
- Torniquet
- Asv(anti-snake venom)
- Work-up
- Treatment
- Indication of asv
- Clinical features
- Recovery phase

AHA 2020 UPDATE ON CPR GUIDELINES

- Double Sequential Defibrillator
- Epinephrine
- Changes in Pediatric Resuscitation
- 6th Chain (recovery) in IHCA and OHCA
- Cardiac Arrest in Pregnancy
- Presumed case of Cardiac Arrest
- Use of Audio-Visual feedback during training
- Return of Spontaneous circulation (ROSC)

High Altitude Pulmonary Edema

- Introduction
- Acute Mountain Sickness
- Action of Acetazolamide
- Clinical features of HAPE
- Management of HAPE

30 BASIC LIFE SUPPORT

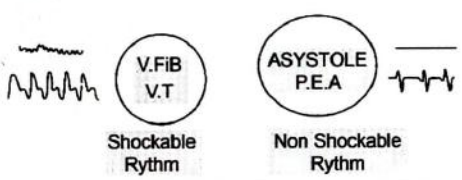
- Out of hospital care to a person who has suddenly become unresponsive & pulseless.
- Any cardiac event occurring out of Hospital in western countries: Survival Rate ≡ 10%
- Any cardiac event occurring out of Hospital & Patient was provided Bystander CPR: Survival Rate ≡ 20%

CARDIAC ARREST

1. Unresponsive person [As there is no cerebral Blood flow]
2. Pulseless [Carotid pulse is checked as it is closest to heart & last one to go in case of cardiac arrest]
3. Apnea/ Gasping Respiration/ Agonal Rhythm

Pulseless conditions

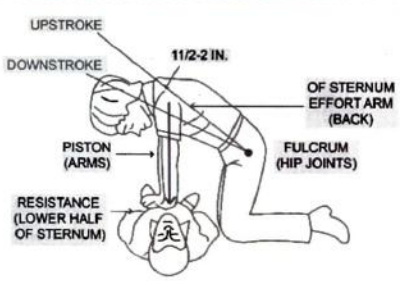
Shockable Rhythms	Non – shockable Rhythms
• Ventricular Fibrillation • Ventricular Tachycardia • Treatment is Cardioversion • Survival chances are higher	• Asystole • Pulseless Electrical Activity [PEA] • Survival chances are low



Important Information

- **Both Shockable & non-shockable Rhythms are inter convertible i.e. Ventricular Fibrillation may convert into Asystole & vice versa.**

CARDIO PULMONARY RESUSCITATION (CPR)



- An elderly male collapsed suddenly in an airport at the boarding gate. What will be the correct management of the patient?

Steps of giving Cardio Pulmonary Resuscitation (CPR)

1st step → Check scene safety (Take the patient to a flat surface)
2nd step → Check unresponsiveness of patient. (Tapping the shoulder)
3rd Step → Call for Help
4th step → Activate emergency response
5th step → Check carotid pulse

Also check breathing effort → Look for

- Chest rise and fall
- Feel the breath sounds of the person

Case scenarios of a collapsed person

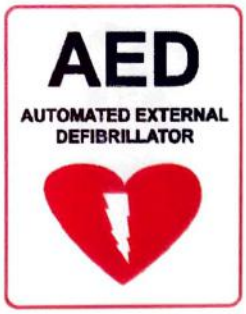
1. **Respiratory Arrest**
   - Pulse → Present
   - Respirataory effort → Absent
   - Treatment → Rescue breaths at the rate of 10-12/min (1 breath/second)

2. **Cardio-Pulmonary Arrest**
   - Pulse → Absent
   - Respiratory effort → Absent
   - Treatment → Cardiopulmonary Resuscitation at a rate of 30 : 2
   - Chest compressions ← 30 : 2 → Rescue Breaths
   - One cycle of CPR is of 2 mins

Automated External Defibrillator Device (AED)

Once CPR is initiated, attach AED electrodes to the chest of patient.

- AED will tell us verbally that whether it is a shockable ryhtm or nonshockable rhytm
- If it's a shockable rhytm: DC shock of 200 J is to be given.
- After shock is delivered, check for return of Pulse & Respiration
- If it's a nobshockable rhytm , continue CPR till medical health team arrives
- Chest compressions are given at a rate of 100 – 120 / min.



Ratio of CPR administration

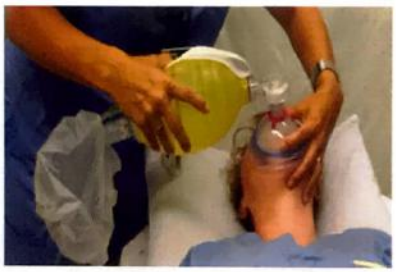
Adult	– 30 : 2	1 or 2 Rescuer
Paediatrics (Child)	– **30 : 2**	**1 Rescuer**
	– **15 : 2**	**2 Rescuer**
Neonate	– 3 : 1	2 Rescuer **mandatory**

- **Sternal Depression**
  - Adults → **5cm**
  - Child → **2 cm**
  - Neonate → **1 cm**
- **Allow for proper recoil of chest so that**
  - Heart can get filled with blood
  - Coronary blood flow can be ensured
- Place hands on lower 1/3rd of sternum while giving chest compressions
- Very forceful & aggressive chest compressions can lead to pressure over Xiphisternum
- As Xiphisternum is pointed → It causes damage to Liver.

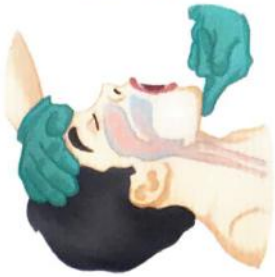
Important Information

- Most common solid organ traumatized in CPR → Liver (Not lungs)
- Lung is not a solid organ
- Hip joint acts as fulcrum while giving CPR
- Elbows should be straight and not flexed.
- Most common ribs damaged while giving CPR → 4th to 6th Ribs
- Do not discontinue CPR even if the Ribs are fractured.

Airway management by Bag & Mask Ventilation (BMV)

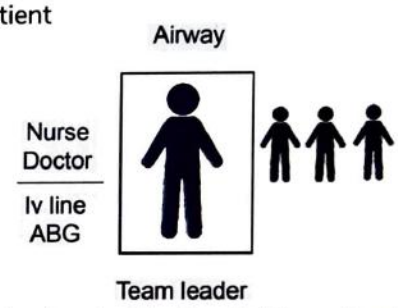


- By AMBU Bag
- Head tilt & chin lift ensure patency of airways so that air can enter into lungs during BMV
- Position of patient → Sniffing position



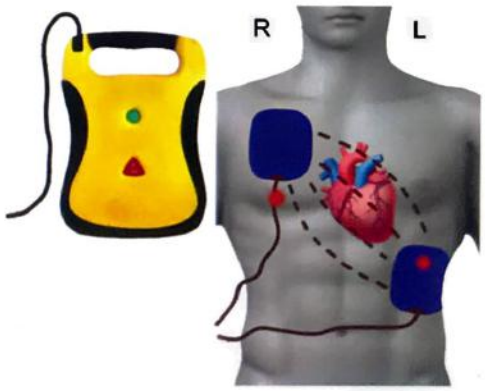
Correct application of mask

- The Mask should be held b/w thumb & an index finger of a left hand in a 'C ' shaped manner so that air does not leak.
- Remaining 3 fingers are kept in an 'E' shaped manner, so as to stabilize the lower part of mandible
- With the right hand we press AMBU bag & give rescue breaths.
- The person who provides airway support stands at head end of patient



- Team leader stands at foot end of the patient
- On the right-hand side is a team of 2-3 doctors and paramedical staff to provide simultaneous chest compressions after every 2 minutes.
- On the left-hand side is the nurse/doctor to secure an I.V. line and collect blood samples for ABG
- Coordination of team members is an important component in CPR

Refer Flow Chart 30.1

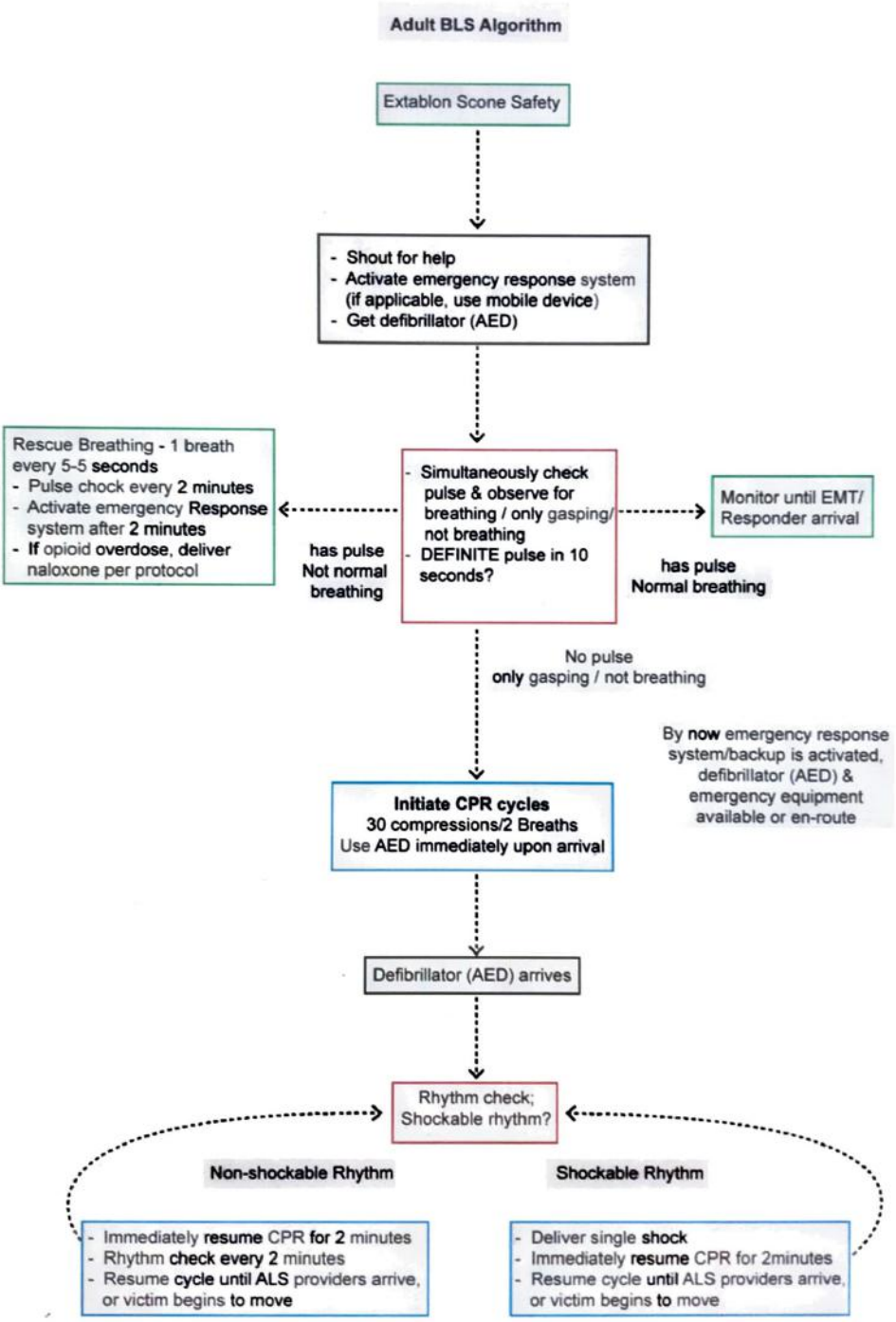


- All AEDs are programmed to fire at 200 J Biphasic

Causes of cardiac arrest

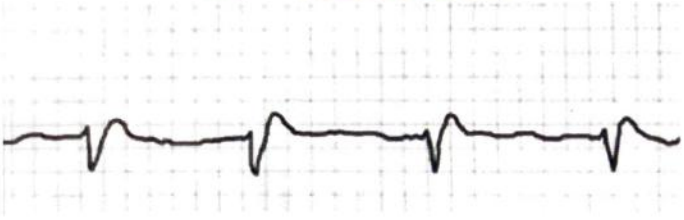
- Coronary Artery Disease
- Cardiomyopathy (HOCM / TTCM) → arrythmia
- Valvular lesions (MS : MR) → LA dilatation → Atrial fib → Stroke
- BBB / Brugada Syndrome (SCN 5A, TDP) → Tachycardia
- Electrolyte imbalance (K↑)
- Toxins: TCA overdosage

Flow Chart 30.1



31 PULSELESS ELECTRICAL ACTIVITY (PEA)

- Electrical activity of heart i.e. SAN, AVN, Bundle of His are normal but pulse is absent



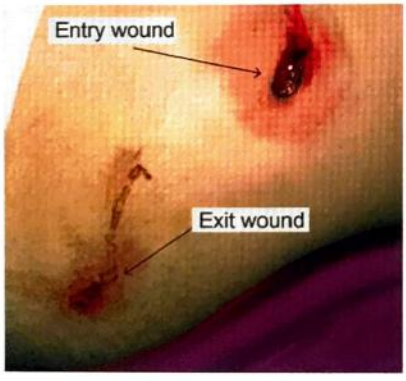
- PEA needs cause specific Rx.
- Giving DC shock in PEA will not help revive the patient.

Outcomes by diagnosis (code blue)

Diagnosis	Survival
• Unstable VT	• 65-70%
• V-Fib	• 25-40%
• PEA	• 11%
• Asystole	• 0.2%

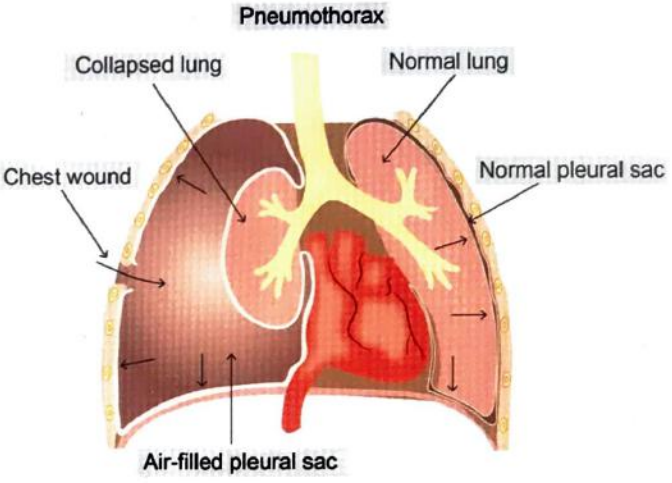
Causes of PEA :

1) Empty Heart

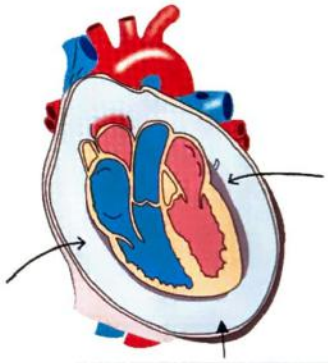


A. Hypovolemia
- Bullet injury to the abdomen causing ripping of aorta / superior mesenteric artery/ inferior mesenteric artery
- Patient can have hypovolemia → resulting in decompensatory shock → pulselessness
- Here, even though muscle of heart is working fine, there is no blood to be pumped

B. Tension pneumothorax
- Bullet injury to the chest causing air rush into the chest cavity- increased +ve pressure- lung collapse - kinking of SVC & IVC.→ venous return is compromised → cardiac output is compromised → pulselessness



C. Cardiac tamponade
- Fluid in pericardial space which exerts pressure on heart from outside → venous return to heart is compromised → Cardiac output is compromised → pulselessness



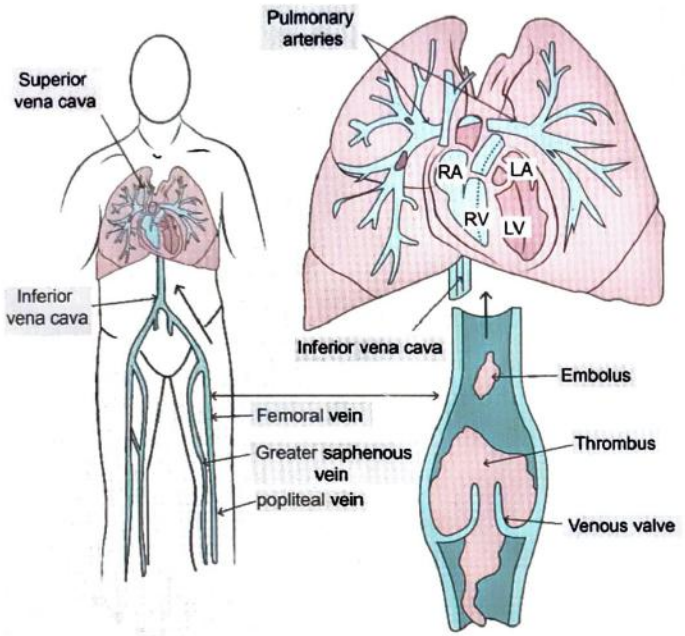
The pericardial sac files with fluid causing severe compression of the heart

2) Electromechanical dissociation
- Conduction normal

A. Acute MI → Stunned myocardium (hypoxia) (extensive ant. wall MI)
   - No effective contractions- ↓ CO

B. Pulmonary embolism: Postpartum patient, Orthopedic surgery pt

- Develop deep vein thrombosis d/t immobilization→ resulting in pulmonary embolism → which causes severe hypoxia & right sided heart failure → pulselessness Echocardiogram → shows dilated right ventricle.



Treatable causes of cardiac arrest

5H's	5T's
• Hypoxia (AMI)	• Toxins: TCA Toxicity
• Hypovolemia (Aortic rupture)	• Tamponade (cardiac)
• Hydrogen ion excess (acidosis)	• Tension pneumothorax
• Hypo / Hyperkalemia	• Thrombosis, Pulmonary
• Hypothermia (Causes BCT < 35°C)	• Thrombosis, Coronary

- Hyperkalemia → K+: >8.0m Eq/L → causes diastolic arrest → pulselessness
- Hypokalemia → causes Diaphragmatic paralysis → resulting in hypoxia → which decreases function of heart

Revise causes

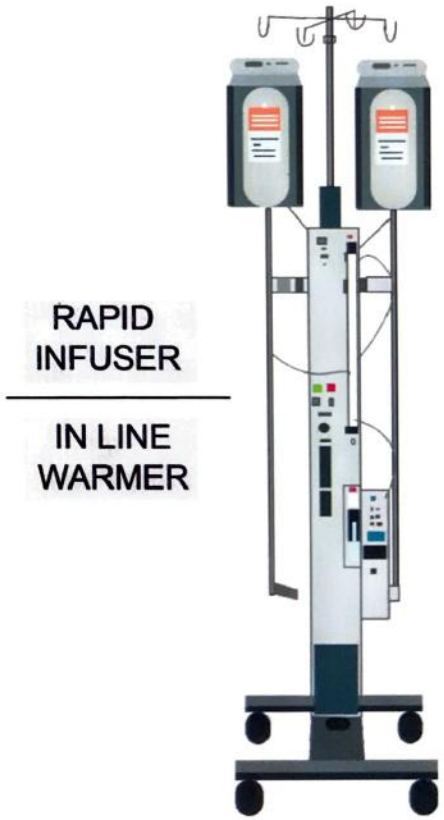
HEART	CONDITION	LUNG
• Hypovolemia	**Not enough blood**	• Hypoxia
• Cardiac Tamponade	**Squeezed**	• Tension pneumothorax
• Acute MI	**Killed**	• Massive pulmonary embolism

The remaining causes of Reversible Cardiac arrest

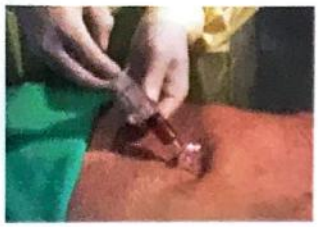
- H Hypothermia
- E Electrolyte Imbalance (↑ K+ / ↓ K+)
- A Acidosis
- P Poisoning

Intervention

- **Rapid infuser**→ Have a in line warmer so that it can prevent the development of hypothermia.



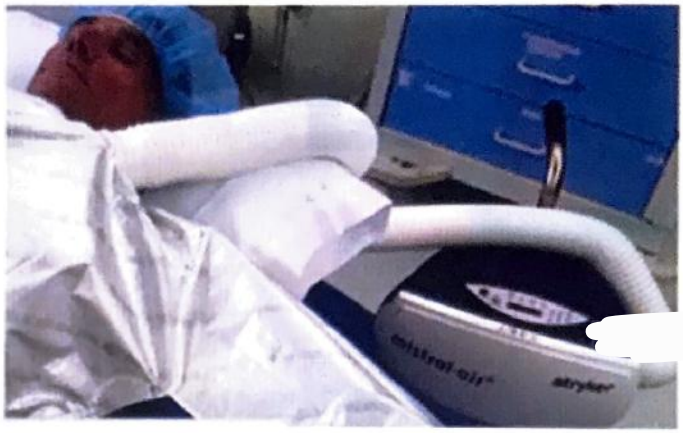
- **Echocardiography guided pericardiocentesis**
- Managing cardiac tamponade



- **Wide bore needle in 2nd intercostal space**
- Managing tension pneumothorax

- If the soldier with tension pneumothorax has bullet proof vest in place/males with big pectoral muscles →then, needle is placed in **5th intercostal space, in mid-axillary line**

AIR BLANKET → TREAT HYPOTHERMIA



32 ADVANCED CARDIAC LIFE SUPPORT

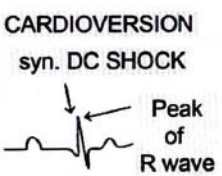
How ACLS is different from BLS?.

- In ACLS, the differential diagnosis(d/d) of the causes of pulselessness are found and the patient is managed accordingly
- In BLS the main aim is to maintain brain perfusion by giving chest compressions

- D/D of Pulselessness
  - Ventricular Tachycardia
  - Asystole
  - Pulseless Electrical Activity
    - Hypovolemia
    - Tension Pneumothorax

- AED (Automated) used in BLS → Decision is made by the machine
- Defibrillator used in ACLS-measures the rhythm, but the physician decides whether shock should be given
- Advanced Airway in ACLS→1 breath is given every 6 seconds (10 breaths /min)

defibrillation - V.FiB
- pVT

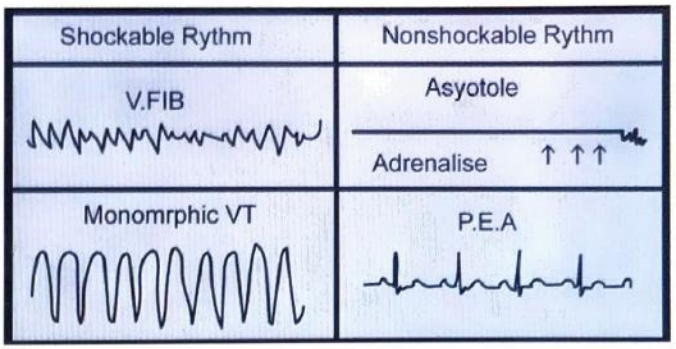


PSVT
A.FiB
A.FLUTTER

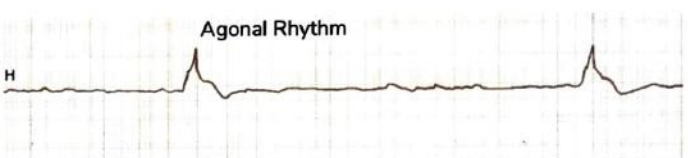
Difference b/w Defibrillation & Cardioversion

- **Cardioversion**
  - Synchronized DC shock at the peak of R Wave
  - Impulse is fired at the peak of R Wave
  - As R wave is the time at which heart is contracting abnormally
  - So, it makes the heart relax, so that, at the next beat SA node takes over
  - Done for → PSVT, A. fibrillation, A. flutter

- **Defibrillation**
  - Impulse will be fixed irrespective of peak of R wave
  - Done for V. Fibrillation, Pulseless Ventricular TachyCardia (PVT) Eg monomorphic VT



- Asystole: Management is to give adrenaline(1:10,000), this adrenaline will convert asystole into V. fib and then DC shock can be given
- PEA : seen in Hypovolemia, Hypothermia, Tension pneumothorax
- Agonal Rhythm → Few last breaths of a person (efforts of brain stem)
  → Irregular Broad QRS due to firing of bundle of HIS: After this Asystole



Treatment of Reversible Causes → 5 H & → 5 T

Refer Picture 32.1

- **Hypokalemia** → Results in TORSADES DE POINTES, Diaphragmatic paralysis
- **Hyperkalemia** →Bradycardia →Diastolic arrest
- **Tamponade** → identified by electrical alternans of ECG
- **Tension pneumothorax** → identified by absent breath sounds on ipsilateral side
- **Coronary Artery Disease** → ST elevation
- **Massive pulmonary embolism** → have right ventricular failure [S1Q3T3] & Kussmaul's sign

- PVT(premature ventricular tachycardia)/V.fib (ventricular fibrillation)
- Non synchronized DC Shock given
- Continue CPR x 2 min, I.V / I.O line established. (as close to the heart)
- Fails: Repeat Non synchronized DC Shock
- CPR x 2 min, IV / IO epinephrine 1 mg in 1:10000 dilution
- Epinephrine increases coronary circulation as it is a vasoconstrictor.
- Advanced Airway, Capnography

(Breathing: 1 breath / 6 Sec after Endotracheal Intubation)

- Fails: 3rd Shock repeated
- CPR x 2 min, IV amiodarone given for shock refractory VT
- Treat reversible cause [hypoxia, toxins acidosis etc]

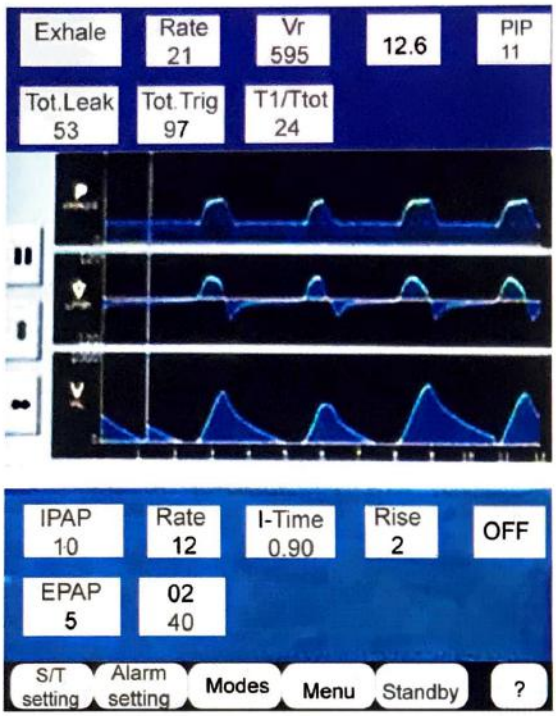
- Amiodarone 3 doses can be given
  - 1st dose = 300 mg
  - 2nd dose = 150 mg after 3-5 mins
  - Amiodarone is useful for VT as well as shock refractory VT

- PEA(pulseless electrical activity) / Asystole
- CPR x 2 min, IV/IO [tibia /sternum] epinephrine
- Advanced Airway & Capnography
- Fails: CPR x 2 min, Repeat epinephrine
- Treat reversible causes mainly Hypovolemia
- Epinephrine repeated every 3-5 minutes

Picture 32.1

H	Hypovolemia	Hypoxia	Hydrogen Ion (acidosis)	Hypo/Hyper-kalemia	Hypothermia
	Loss of fluid volume in the circulatory system. Look for obvious blood loss. Most important intervention is to obtain IV access and administer IV fluids. Use a fluid challenge to determine if the arrest is related to hypovolemia	Deprivation of an adequate oxygensupply can be a significant contributing cause of cardiac arrest. Ensure that the airway is open. Ensure adequate ventilation, and bilateral breath sounds. Ensure oxygen supply is connected properly.	Obtain an arterial blood gas to determine respiratory acidosis. Provide adequate ventilations. Use sodium bicarbonate to prevent metabolic acidosis if necessary.	Both a high and low k + can cause cardiac arrest. Signs of high K + include taller, peaked T-waves, and widening of the QRS complex. Signs of low K+ include flattened T–waves,prominent U-waves and possibly widened WRS complex.	If a patient has been exposed to the cold, warming measures should be taken. Core temp. should be raised above 86 F and 30 C as soon as possible. The patient may not respond to drug or electrical therapy while hypothermic.
T	Toxins	Tamponade	Tension Pneumothorax	Thrombosis (heart: acute,massive MI)	Thrombosis (lungs: massive PE)
	Accidental overdose: Some of the most common include: tricyclics,digoxin, betablockers, and calcium channel blockers). Cocaine: is the most common street drug that increases incidence of pulseless arrest. Physical signs: Include bradycardia, pupil symptoms, and other neurological changes. Poison control can be utilized to obtain information about toxins and rversing agents.	Fluid build-up in the pericardium results in ineffective pumping of the blood which can lead to pulseless arrest. ECG symptoms:Narrow QRS complex and rapid heart rate. Physical signs: jugular vein distention (JVD), no pulse or difficulty palpating a pulse, and muffled heart sounds. Perform: pericardiocentesis to reverse.	Tension pneumothorax shifts in the intrathroacic structure and can rapidly lead to cardiovascular collapse and death. ECG sings: Narrow QRS complexes and slow heart rate Physical signs: JVD, tracheal deviation,unequal breath sounds,difficulty with ventilation,and no pulse felt with CPR. Treatment: Needle decompression.	Cause acute myocardial infarction ECG sings: 12 lead ECG with ST-segment changes, T-wave inversions,and/or Q waves. Physical sings: elevated cardiac markers on lab tests, and chest pain/pressure. Treatments: use of fibrinolytic therapy, PCI (percutaneous coronary intervention). The most common PCI procedure is coronary angioplasty with or without stent placement.	Can rapidly lead to respiratory collapse and sudden death. ECG signs of PE: narrow QRS Complex and rapid heart rate. Physical sings:No pulse felt with CPR.Distended neck veins,positive d-dimer test,prior positive test for DVT or PE. Treatment: surgical intervention (pulmonary thrombectomy) and fibrinolytic therapy.

33 MECHANICAL VENTILATION STRATEGY



- **TIDAL VOLUME [V_T]:** 12-12 Rule
  - V_T = 12ml / Kg (lean body weight) → i.e. Fat component is subtracted.

Important Information

- In ARDS patient, we don't follow 12-12 Rule.
- In ARDS : V_T = 6 ml /kg (lean body weight) → to minimize volutrauma in the pt
- In ARDS, there is inflammation in alveoli → if we inflate these alveoli in standard fashion, it leads to barotrauma to the alveoli → which worsens the situation
- A person having any neurological cause [GBS / Transverse myelitis etc] with diaphragmatic paralysis → there is no inflammation in the lungs → so, this person can be ventilated at tidal volume of 12mg/kg

- **RR**= 12/min : Conditions in which we increase the respiratory rate & make the person hyperventilate are:
  - Raised intracranial pressure
    - ➢ Hyperventilation causes vasoconstriction of cerebral blood vessels resulting in decreased production of CSF → decrease ICP
  - DKA (Diabetic ketoacidosis)
    - ➢ Hyperventilation is a Compensatory mechanism to clear acidosis in DKA patient

- **FiO_2** : 1.0 = 100% of O_2, 0.6 = 60% of O_2; 0.4 =40% O_2
  - Objective of giving O_2 [starting from FiO_2 0.4] to the pt is to maintain oxygen saturation
  - paO_2 > 60 mm Hg & spO_2 > 90%

- **PEEP (Peak End Expiratory Pressure)**
  - There would always be a baseline pressure in the lungs, so whatever the inflation/ deflation that would be done should be always above this base-line pressure.
  - Hence after applying PEEP of 3-5 cm H2O, Recruitment of alveoli will be maintained (more surface area of alveoli is available for gas exchange)
  - Better gas exchange of respiratory gases both during inspiration and expiration and hence, better O2 saturation will be maintained.
- Disadvantage Of Peep
  - If PEEP ↑↑ : it increases pressure on great veins of the chest leading to reduction in venous return to heart : decreases B.P
  - Higher PEEP is a disaster for the hypotensive patients
    - ➢ Eg. Pulmonary embolism (RVF & hypotension)
    - ➢ Inferior wall MI (RVF)
- Therefore, PEEP must be kept at 3-5 cm of water

Undertsand with example

ABG parameters

(A) Patient

- pH = 7.40 (normal)
- pO_2 = 40 (deficient)
- pCO_2 = 40 (normal)

Management:

- ➢ ↑FiO_2
- ➢ ↑PEEP → Recruitment is Better

In hypoxia, parameters to be taken care of are FiO_2 & PEEP.

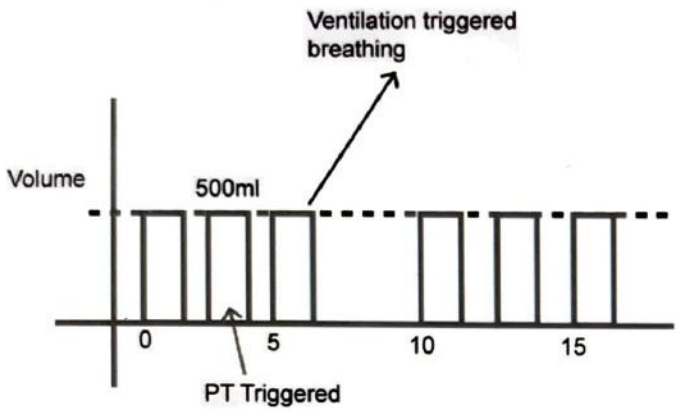
(B) Patient

- pH = 7.20 (acidosis)
- pO_2 = 100(normal)
- pCO2 = 60 (↑↑ ed) (respiratory acidosis)

Management:

- Change the RR ↑↑↑
- Minute Volume ↑↑
- Result in CO_2 washout & correct acidosis
  In Respiratory acidosis, take care of RR & Minute Volume of the ventilator.

ASSISTED CONTROL MECHANICAL VENTILATION (ACMV)



- RR = 12 /min [1 breath every 5 sec]
- V_T = 500 ml
- FiO_2 = 0.4(40%)

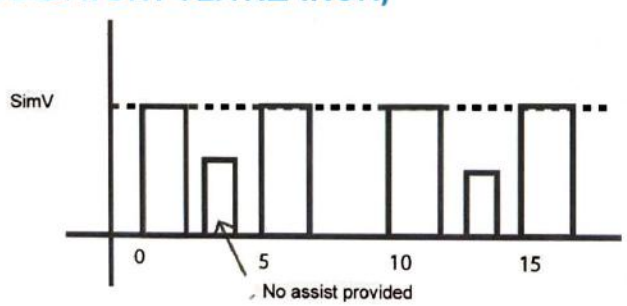
ADVANTAGE-

- Used in patients with no spontaneous breathing
- Patient triggered breaths are also assisted by ventilator.
- ACMV is Most common mode used in invasive mechanical ventilation.

S/E of ACMV

- HYPERVENTILATION (only with ACMV) causes Respiratory Alkalosis resulting in HYPOCALCEMIA (TETANY) and LARYNGOSPASM.
- HYPOXIA leads to Seizures & Myoclonus

SIMV (SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION)



- The breathing is synchronized but not assisted by the ventilator

Advantage:

- Support or wean off the patient from ventilator.
- Ensured minute volume is provided

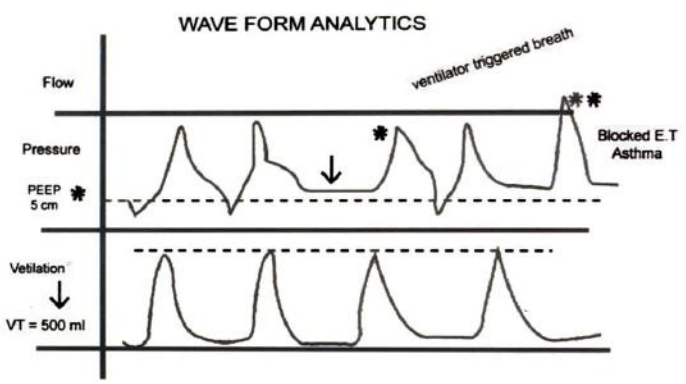
Disadvantage

- HYPOVENTILATION: If patient's breathing effort is occurring at the same time as that of mechanical ventilator breath, it results in inhibition of ventilatory drive & total Minute Ventilation will be reduced.

Important Information

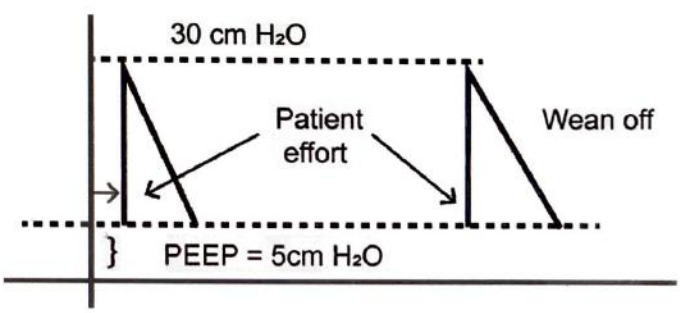
- DO NOT USE SIMV. if patient has tachypnea
- Asynchrony [lack of coordination b/w ventilator & person] can also result in hypoventilation

WAVEFORM ANALYTIC PRESSURE SUPPORT VENTILATION



- Negative spike (red)- Inspiratory effort of the patient (pt. triggered breath)
- Flat line – Ventilator triggered breath
- ** Spike of waveform due to
  - Blocked Endotracheal tube
  - Asthma

PRESSURE SUPPORT VENTILATION (PSV)



- Only pressure support is given
- No guaranteed /backup ventilation
- Use- to wean off patient from ventilator

Important Information

- ACMV- To put patient ON ventilator
- SIMV and PSV - To wean OFF patient from ventilator

PRESSURE CONTROL VENTILATION (PCV) INDICATIONS:

- Pre-existing Barotrauma (pt with pneumothorax on ventilator)
- Patient undergone cardiothoracic Vascular surgery (to reduce shear stress over the surgical scar)
- 'CONTROL' means to limit further inflation of lungs to a level where it may cause worsening of pre-existing barotrauma in a person

PROTECTIVE VENTILATOR STRATEGY:

- Any person who has gone into respiratory failure, 4 basic rules are to be followed while treating that person to increase chance of survival

BASIC RULES OF VENTILATION:

1. Tidal Volume in ARDS = 6 ml/kg (6-8 ml/kg)
2. Peak pressure = 30 cm of H_2O (to limit barotrauma component)
3. FiO2 = 0.4 (initiated and then gradually upgraded)
   a. $SaO_2 > 90\%$
   b. $pO_2 > 60$ ($pO_2 < 60$ is Hypoxia)
4. PEEP = 5 cm of H_2O (to decreases the venous return and recruitment of alveoli simultaneously)

NIV (Non-Invasive Ventilation) VS CPAP (Continuous Positive Airway Pressure)

- Both gives good O_2 delivery → by tight fitt

Refer Table 33.1

SUMMARY:

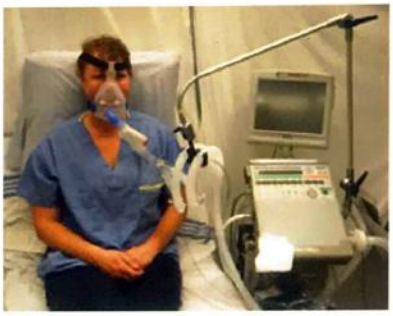
- CPAP:
  - ➢ It is the splint that keeps the airways open [method of non-invasive ventilation]
  - ➢ INDICATIONS: Hyaline Membrane Disease, Obstructive Sleep Apnea
- BiPAP / NIV: variation in iPAP, ePAP
  - ➢ Indication: COPD Exacerbation

INVASIVE MODE (IMV)

- ACMV: Most common modality of ventilation used.
  - ➢ Initiation of ventilation
  - ➢ S/E → Hyperventilation, respiratory alkalosis, seizures, myoclonus
- SIMV, PSV
  - ➢ used for weaning off the pt from ventilation
  - ➢ S/E (Respiratory Acidosis- $\uparrow CO_2$) → hypoventilation
- PCV
  - ➢ CTVS (Cardiothoracic Vascular Sx), Barotrauma (Pre Existing)
  - ➢ S/E (Respiratory Acidosis- ↑CO2) → hypoventilationing face mask

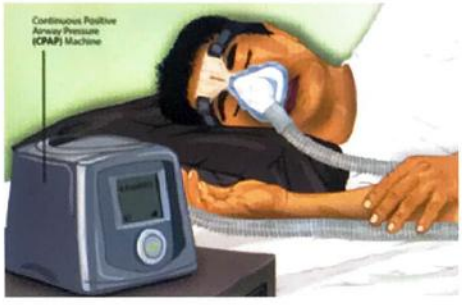
Table 33.1

NIV



- Positive pressure generated which varies in phase of inspiration [i PAP] and expiration [e PAP] [to decrease the load on accessory muscles of respiration]

CPAP



- Constant positive pressure, maintained both in inspiration and expiration
- Acc to Harrison- CPAP not exact form of ventilation
- * Splinting of airways [force to keep the airway open]

INDICATIONS

- COPD exacerbation (Resp acidosis) (PH- 7.25- 7.35)
  - ➢ Intubation is avoided bcoz there is a chance of developing ventilator associated pneumonia & local trauma to the airways of pt
  - ➢ Hence NIV is recommended for patients with Type-2 resp failure [COPD exacerbation with resp acidosis]

Advantage

- ↓ incidence of VAP [pseudomonas > staph aureus]
- ↓ Tracheo-laryngeal trauma

- It is a type of Pressure Support Ventilation (PSV) without intubation

Monitor of Progress

↓ Respiratory rate , ↓ use of accessory muscles of respiration

Contraindications

- Encephalopathy/ People who Can't protect their airways
- Cardio-Respiratory Arrest
- GIT bleeding
- Unstable Angina /MI [any acute pulmonary syndrome]
- Facial surgery
- Upper airway obstruction

INDICATIONS

- Hyaline Membrane disease
- Obstructive sleep apnea [always feel sleepy]
  - In REM, tongue falls back → narrowing of airways → nocturnal awakening
  - CPAP is initiated if there are >15 apneic episodes/hr (minimum)
- Cardiogenic pulmonary edema (CPAP is a supportive measure)

34 MASSIVE TRANSFUSION PROTOCOL

- Replacement of the entire blood volume of patient by more than 10 Units of whole blood within 24 hrs or more than 2.5 L [5 units] of whole blood given within 4 hrs period
- 6 units of Packed RBC / 6 units of FFP / 6 units of PRP can be given to the patient

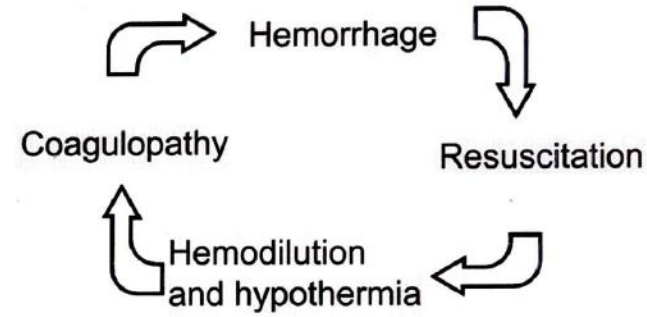
- 6 units of platelet rich plasma is condensed into single bag and is available as SDP (Single donor Platelet) which is prepared by Apheresis
- Efficacy of SDP= 6 Units Of Platelet Rich Plasma (PRP)
- Ratio of administration = PRBC: FFP : PRP = 1:1:1
- In a bleeding patient, there would be hypoxia & acidosis d/t blood loss.
- First thing to be administered in these patients is Packed RBC [f/b FFP & PRP] which carry oxygen & neutralize the acidosis component.
- Acidosis impair the ability of blood to clot, which may worsen the coagulopathy of the patient. Hence, platelets shouldn't be given first

- RED COOLER BAG: Have 6 units of packed RBC
- Plasma is stored at -30 degree C
- Plasma when required for transfusion is thawed and kept at room temperature
- Platelets are not kept in cooler bags → these are stored at room temp → 20-24 degree C

TRAUMA INDUCED COAGULOPATHY

- Trauma in patient causes blood loss, which can lead to loss of clotting factors and hypothermia [d/t loss of heat] leading to TRAUMA INDUCED COAGULOPATHY
- THE HUMAN COAGULATION SYSTEM IS SLOW AND WEAK
  - Clotting takes time (2-10 minutes in the best circumstances)
  - Clots are physically weak
  - There is limited clotting material to work with (even in the whole body) :
    → 10 g of fibrinogen total
    → 15 ml of platelets total in normal individuals
- If we give packed RBC which are stored at 4 degree C to these patients → hypothermia worsens → which further result in coagulopathy

BLOODY VICIOUS CYCLE



- Poor circulation results in metabolic acidosis [because cells are in anaerobic respiration]
- Giving cold blood components triggers → Hypothermia interferes with clotting process leading to coagulopathy
- Triad of acidosis + hypothermia + coagulation → difficult to manage
- To minimize hypothermia → ensure in-line warmers in rapid infusion pump**Understand with example**

In obstetric hemorrhage, resuscitation of patient with massive transfusion protocol :

- **When so much of crystalloid fluids followed by blood transfusion is given → hemodilution occurs→ hypothermia → Interferes with coagulation process & worsens coagulopathy**
- ensure in-line warmers in rapid infusion pump→ components are warmed to sufficient temperature to minimize the hypothermia component

- If there is 150 ml blood loss/min in a patient → within 20 min → depletion of circulating volume → decompensated shock (↓↓ BP) → Death
- So, in a bleeding patient, infused blood should be equal to output i.e blood loss so as to prevent the patient from going into decompensated shock.

TRIGGERS OF MASSIVE TRANSFUSION PROTOCOL

ASSESSMENT OF BLOOD COMPONENT SCORE (ABC SCORE)ComponentsPoints

1. Penetrating Injury
2. FAST positive
3. HR > 100/min
4. SBP < 90 mm Hg1

1

1

1

- If ABC score ≥ 2 then there is 75% accuracy in prediction of Massive Transfusion Protocol [MTP]

SETUP REQUIRED FOR INITIATING MTP IN A PATIENT:

- Pressurized Rapid Transfusion (PRT)
  - Ensures that the blood components reach into the body of patient on time
  - Ensures that infusion matches output & chances of survival increases
  - Has in-Line Warmer → chances of Hypothermia ↓ hence coagulation problems are taken care of and hemostasis achieved.
- Tranexamic Acid
  - Antifibrinolytic agent
  - stabilize the clot → hemodynamic stability

ADULT MASSIVE TRANSFUSION PROTOCOL

TRIGGERS

- ABC Score (ABC > 2)
- Surgery [trauma to major blood vessel]
- Hematemesis d/t PUD (Peptic Ulcer Disease)
- PPH (Postpartum Hemorrhage)
- Penetrating traumatic Injury
- + Low BP

Round 1

- Call Blood Bank & inform MTP protocol to be initiated
- Assign Team Members to do specific tasks and Divide Teams into A, B, C.
- Team A = Administer blood Components to patients (not be responsible for drawing, labeling blood sample or writing notes in a file about units of blood sample given)
- Team B = Record Keeping, Sampling, labeling of samples, entry in the file of units of blood etc, so that proper documentation is maintained.
  - Blood Sample - CBC/CMP
  - PT/aPTT
  - ABG [from radial artery]
- Team C = Runner → go to blood bank & get cooler bag/blood units to hospital

Team A:

- Give 1g of Tranexamic acid to patient I.V Stat, then given 8 hourly
- Connect the pressurized rapid transfuser to the patient
- Give 4 Units of PRBC (O-VE) & 2 units FFP (AB +)
- After Round 1, Reassess the patient : If NO Improvement: ROUND 2

Round 2

- Infuse
  - 4 Units PRBC
  - 4 Units FFP
  - 1 Units SDP [Single Donor Platelet]
- Team B → Resend blood to Lab → so that we can evaluate coagulopathy & metabolic acidosis component.
- Calcium gluconate given to prevent tetany [caused by citrate in PRBC].
- Cryoprecipitate given → If the fibrinogen < 100 mg/L
- If No Improvement: ROUND 3

Round 3:

- Repeat Round 2 + Factor VII a

Important Information

Indications to administer Factor VIIa :

- **Surgical hemostasis**
- **temperature-37 degree celsius (stabilized)**
- **pH-7.35 (stabilized) has been achieved but patient is still bleeding**

CHALLENGES DURING MASSIVE TRANSFUSION PROTOCOL:

1. Hypovolemia
- All efforts should be made to stop the bleeding rather than replace blood loss.
- The loss of 150 ml of blood per minute results in loss of half the blood volume in 20 Minutes

2. Hypothermia
- Because Erythrocytes are stored at 1°C-6°C, rapid blood product administration can lead to hypothermia which leads to coagulopathy
- 6 Units of RBC will decrease body temp. by 1°C.

3. Hypo-coagulopathy

- There is dilution effect from infusion of crystalloid volume expanders.
- Hypothermia reduces the activity of coagulation proteins and prevents activation of platelet.

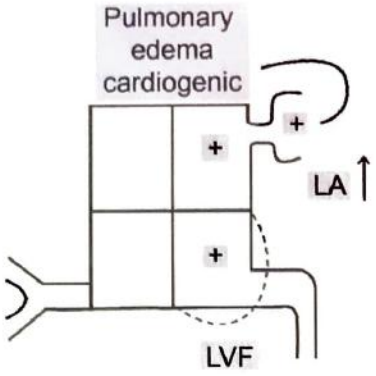
COMPLICATIONS OF MASSIVE TRANSFUSION PROTOCOL:

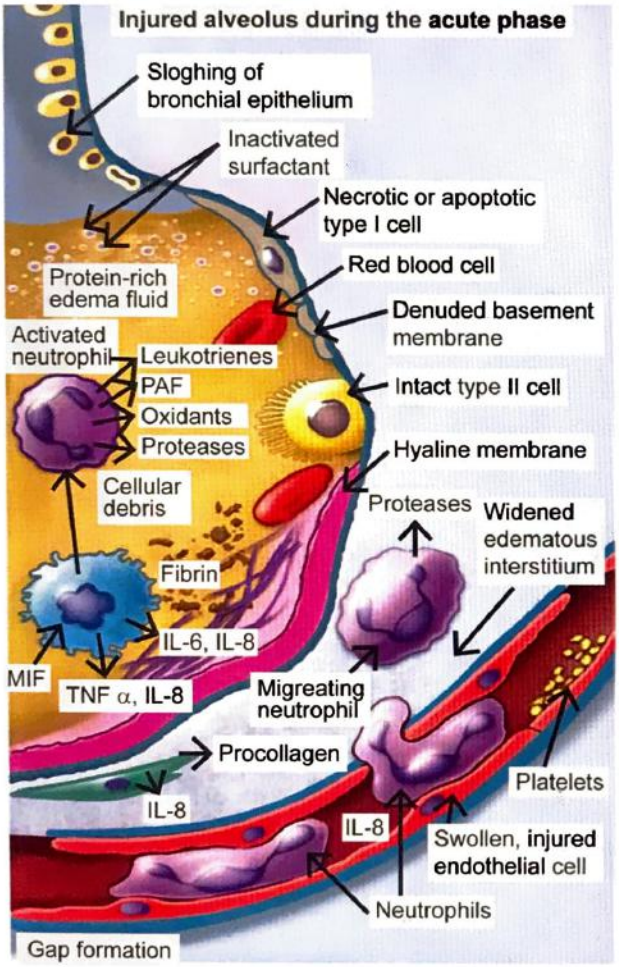
1. Coagulopathy: minimized by Pressurized Rapid Transfusion within line warmer
2. TRALI (Transfusion Related Acute lung injury): Respiratory Distress [non-cardiogenic pulmonary edema] + BP normal + BNP normal
3. TACO (Transfusion Associated Circulatory Overload): Respiratory Distress (cardiogenic pulmonary edema) + BP ↑ + BNP ↑
4. Hyperkalemia: packed R.B.C are stored at lower temp, near expiry date → Na+/ k+ pump is shut down which leads to hyperkalemia → causes Diastolic arrest (Bradycardia)
5. Hypocalcemia: Laryngospasm (Difficulty in breathing, stridor in clear chest)

RESPIRATORY DISTRESS CAN BE DUE TO

- TRALI: Pulmonary edema [non-cardiogenic pulmonary edema]
- TACO: Pulmonary edema (cardiogenic pulmonary edema)
- Laryngospasm: chest is B/L clear but adventitious sounds are heard - (adventitious sounds are the sounds from upper airway)

35 ACUTE RESPIRATORY DISTRESS SYNDROME





- **CARDIOGENIC PULMONARY EDEMA** → ↑ left atrial pressure secondary to LVF → edematous fluid is TRANSUDATE
- Acute Respiratory Distress syndrome is due to NON-CARDIOGENIC PULMONARY EDEMA

NON-CARDIOGENIC PULMONARY EDEMA

- Example , A patient has swine flu/bird flu. As swine flu virus damages pneumocytes without damaging heart, this will end up in ARDS.
- Collapse of alveoli leads to hypoxia and which in turn leads to damage of Endothelium (damage to gap junctions)
- Leakage of fluids from pulmonary capillary into alveoli
- ARDS occurs in 10% of ICU patients
- Sudden onset Respiratory distress
  - + CXR B/L infiltrates (no cardiac cause found)
  - + ↓ pO_2 (demonstrable hypoxia)
  - + Normal left atrial pressure (LAP)

Triggers for ARDS:

Direct (M/C)	Indirect

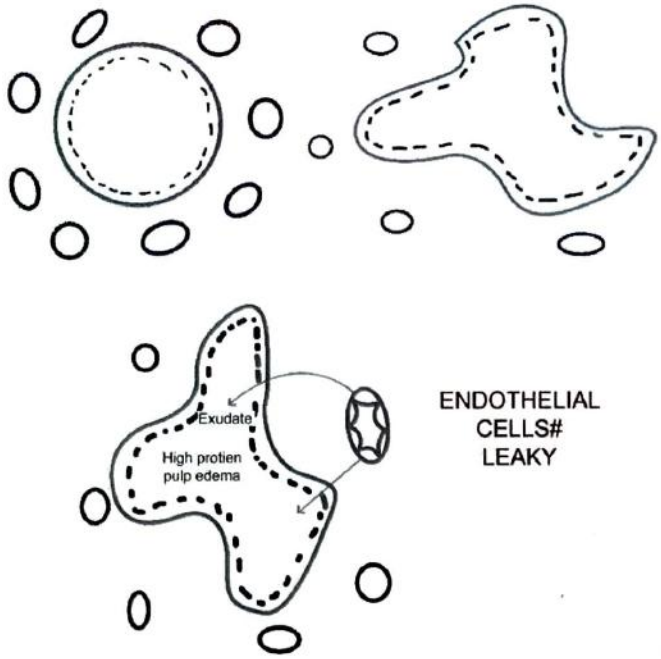
• Pneumonia (H1N1)
• Mendelson syndrome
• Toxic gas inhalation
• Pulmonary contusion
• Near drowning
	• Sepsis
	• Trauma
	  ○ Multiple bone fracture
	  ○ Flail chest
	  ○ Head injury
	  ○ Burns
	• Multiple blood transfusion /TRALI
	• Acute pancreatitis
	• Post cardio pulmonary bypass

- Mendelson syndrome - Aspiration of stomach acid (Chemical Pneumonitis)

Important Information

- Both pneumonia > sepsis are the leading causes of ARDS
- Leading cause of death after blood transfusion – TRALI
- Status asthmaticus (disease of airway) is not ARDS(disease of alveoli)

Clinical Presentation



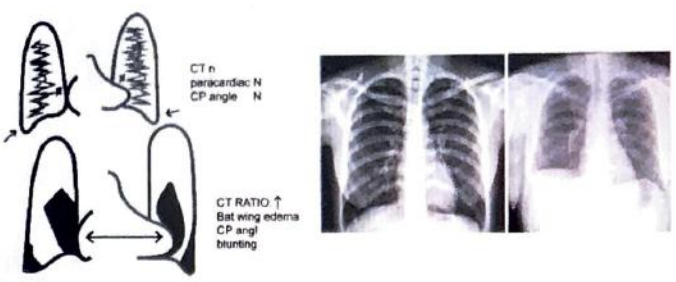
- Type 1 pneumocytes –covers 90% of surface area - (injured during adult ARDS)
- Type 2 pneumocytes – produce surfactant (most abundant cell of alveoli)
- IN ARDS, Type 1 pneumocytes are affected, Type 2 pneumocytes are not affected.
- Normal surfactant with reduced surface area of alveoli for gas exchange
- Ventilation and perfusion imbalance (perfusion is secondary to hypoxia component)
- Hypoxia causes dilatation of all blood vessels in body except vessels of pulmonary circuit
- Most vulnerable cells/most damaged cells in ARDS – endothelial cells of alveoli - d/t hypoxia
- Endothelial cells become leaky resulting in Exudative high protein pulmonary edema.
- In heart failure → low protein pulmonary edema

FEATURES: (Sudden onset Respiratory Distress)

Refer Table 35.1

- In initial phase of ARDS , because of tachypnoea , there will be Respiratory alkalosis (CO_2 washout)
- In acute asthma → type-1 respiratory failure & respiratory alkalosis → d/t CO_2 washout
- In status asthamaticus -type-2 respiratory failure & Respiratory acidosis d/t CO_2 overproduction in lungs
- When PCO_2 = 60mm of Hg, then the compensatory mechanisms begin to fail.

WORK–UP IN ARDS



1. CXR

In ARDS patient– B/L extensive infiltrates

- Cardiothoracic ratio – normal
- Para cardiac area sparing
- CP angle – normal

In cardiogenic pulmonary edema

- Cardio thoracic ratio – increased
- Bat wing edema
- CP angle blunted

2. Echo-

- In ARDS: Ejection fraction is normal, LA pressure – normal
- In cardiogenic pulmonary edema : Decrease ejection fraction and LA pressure increased

3. ABG in ARDS- ↓ pO2, ↑ pCo2 [ventilation, perfusion imbalance]

KEY WORDS: - ESSENTIALS OF DIAGNOSIS

Sudden onset resp. distress

- Central line insertion: Pneumothorax
- S. Aureus pneumonia: receiving i.v Vancomycin for 2 days: Pneumatocele rupture resulting in Pneumothorax
- After multiple bone fractures/ Massive blood transfusion: ARDS : **BERLIN Criteria**
  - Sudden onset resp. distress
  - CXR: B/L pulmonary infiltrates
  - Pao_2 / Fio_2 < 300 → most imp diagnostic criteria for ARDS
  - Absence of LA Hypertension

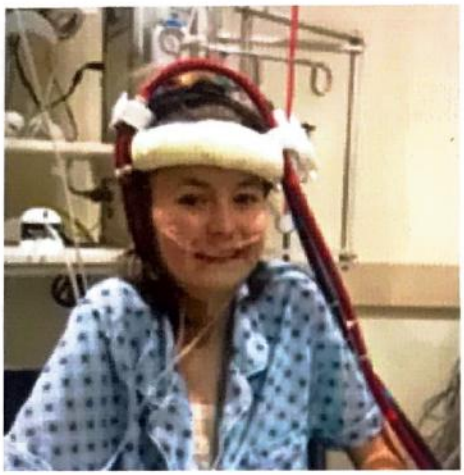
GRADING

ARDS	Pao_2 / Fio_2
• Mild	<300
• Moderate	<200
• Severe	<100

- Volutrauma can occur in ARDS : seen in high/normal volume ventilation resulting in Pneumothorax.

Rx of ARDS

1. Low volume ventilation = 6 ml/kg → to minimize BAROTRAUMA to chest. (Normal tidal volume = 12 ml/kg)
- Plateau pressure of ventilator = <30 cm H2O
- Respiratory rate of ventilator = <35/min
2. Prone position ventilation
- Risk of extubation, CVP line loss, Orthopedic injuries
3. Extracorporeal membrane oxygenation (ECMO)- Patient in which heart and lung are too weak for ventilation



5. Limited fluids & diuretics – To maintain normal LA pressure
6. Ensure neuromuscular paralysis: Cisatracurium (for effective ventilation)
- Steroids, surfactant therapy & high frequency jet ventilation (HFJV) have no role in treatment
- M/c cause of death in ARDS: Sepsis (non-pulmonary causes)

Congestive heart failure	ARDS
1. Transudative pulmonary edema	Exudative pulmonary edema
2. Hydrostatic pressure ↑	Hydrostatic pressure normal
3. LA pressure ↑	LA pressure normal
4. Low protein pulmonary edema	High protein pulmonary edema
5. BNP ↑	BNP normal

Table 35.1

EXUDATIVE PHASE

- 0-7 days
- Respiratory distress starts with in 12-36 hrs of triggers onset
- INTRAPULMONARY SHUNTING[Blood is getting wasted in the lungs→ d/t alveolar collapse/endothelial injury]
- ↑ work of breathing
- ↓ pO_2, ↑ pCO_2 (Refractory hypoxia)
- Dead space ↑
- Type 2 Respiratory failure / Respiratory acidosis

PROLIFERATIVE PHASE

- 7-21 days
- Able to wean off the ventilator
- Proliferation of type 2 pneumocytes
- Some differentiate into type-1 pneumocytes
- Recovery is possible

FIBROTIC PHASE

- >21 days
- Require supplemental oxygen for rest of their life [requirement varies from case to case]
- It results in pulmonary artery hypertension
- Bulla/blebs can also be seen in ARDS

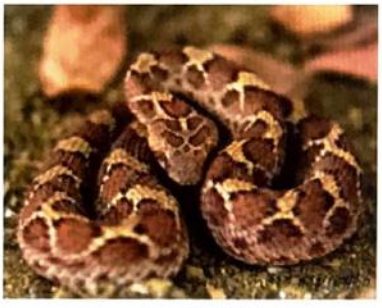
36 SNAKE BITE

STANDARD SNAKES FOUND IN INDIA

Cobra

Krait

Russel Viper



Saw Scaled Viper

Humped Nose Viper

- Cobra and Krait contribute to Neurotoxicity
- Russel viper and Saw scaled viper Contribute to hemotoxicity
- Humped nose viper
  - can also cause hemotoxicity
  - Anti-snake venom available in India is ineffective
  - bleeding manifestation can go as long as upto 3 weeks
- Bite of poisonous snake and non-poisonous snake becomes difficult to identify due to local edema of soft tissue.
- About 50K deaths /yr in India are d/t snake bite.
- About 50 million people are exposed to risk of getting a snake bite especially increased during monsoon/ harvesting season.
- However, 70% of bites that occur are non-venomous
- Bite from venomous snake might be a dry bite (i.e. The snake might bite at an angle that it is able to inject its teeth/ fangs into body of person but may not be able the inject the poison, as person might perceive pain & withdraw the arm away)
- 50% bites: Venomous snake bite is Dry bite.

MANAGEMENT IN CASE OF SNAKE BITE WE FOLLOW FIRST AID COMPONENT

- **R- Reassure the patient.** 70% snake bites – nonvenomous species. Only 50% of bites by venomous species envenomate the pt.
- **I – Immobilize** → which prevent faster spread of venom.
  - DO NOT WALK
  - DO NOT APPLY TORNIQUET → to avoid pressure necrosis
- If tourniquet is already applied then, don't cut tourniquet, as it may cause extensive spread of poison resulting in
  - Diaphragm paralysis
  - Sudden onset hypo-tension d/t histamine release
- So, apply B.P cuff proximal to tourniquet & Inflate it to the pressure almost equivalent to the tourniquet & then cut the tourniquet → Then gradually deflate the B.P cuff
- No CUTTING / ELECTROCAUTERY of that area & no walking as well
- NITROGESIC ointment , nitrate spray can be applied locally → but didn't show any efficacy on clinical trials
- **GH – Get to the hospital immediately**
- **T – Tell the doctor of any systemic symptoms that manifest on way to hosp.**

How to remember

- RIGHT

Important Information

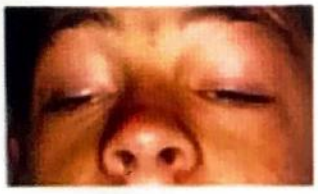
- Cobra bite (neurotoxic) → affects post synaptic transmission. It hampers action of Ach at neuro-muscular junction
- Krait → mainly affects presynaptic transmission i.e. release of Ach at neuro-muscular junction is affected
- Russel viper and Hump nosed viper cause acute kidney injury/ renal failure/ Acute tubular necrosis/ uremia
- Hump nosed viper → can cause hemorrhagic manifestations which can persist for weeks
- ASV should be given within 4 hrs of snake bite
- ASV: Bite to needle Time <4 hrs

VIPER ENVENOMATION MANIFESTATIONS:

Gum Bleeding in Viper Bite

- Local pain, tender lymphadenopathy : Noticed with viper bite and not noticed with cobra bite, krait bite.
- One of the earliest bleeding manifestations → Subconjunctival Hemorrhage
  - Epistaxis
  - Gum bleeding
- Severe abdominal pain (d/t bleeding in mucosa of stomach)
- BP↓ (d/t combination of histamine release, bleeding & vomiting)
- Purpura (I.e. Palpable bleeding on skin)
- Retro peritoneal bleeding
- Kidney: Acute tubular necrosis, flank pain/ tenderness in costovertebral junction, Black/dark urine (d/t hemoglobinuria)

ELAPID ENVENOMATION MANIFESTATIONS (BY KRAIT/ COBRA)



4P's

- Ptosis: Paralysis of Levator Palpebrae , Diplopia / ophthalmoplegia
- Paralysis of jaw/ tongue →inability to swallow saliva.
- Pooling of secretions → cause aspiration & can die d/t aspirational pneumonia.
- Paradoxical Respiration

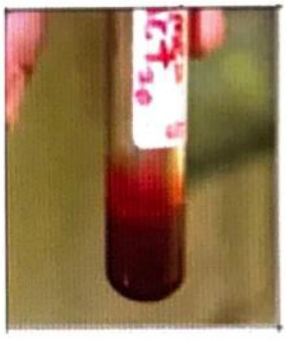
4D's

- Diplopia
- Dysphagia
- Dysarthria/ dysphonia
- Dyspnea

- There is development of DESCENDING PARALYSIS in patient

20 WHOLE BLOOD CLOTTING TEST (WBCT)

- Draw 2 mL of venous blood and transfer directly into a lean and dry glass tube. Leave it upright, open, undisturbed for 20 and/or 30 minutes at room temp
- After exactly 20 minutes, pick up the tube and invert it. If a solid clot is retained, the test indicates normal coagulation.
- If clot breaks down quickly upon inversion of the tube or fails to coagulate, the test indicates a coagulopathy



Collection: a blood sample for 20 WBCT testing immediately after collection.

Normal: a solid clot is retained on inversion of the tube at 20 or 30 minutes (Grade 0, no coagulopathy)

Abnormal: clot degrades rapidly (Grade 1, friable clot) or fails to coagulate whatsoever **(Grade-2)**

Work up:

1. 20min Whole Blood Clotting Test
   Repeat for every 30 min for first 3 hrs of the admission [then after giving ASV it can be done on 1hr basis]
2. CBC
3. LFT
4. KFT (baseline serum creatinine)
5. Coagulogram : PT/ aPTT

Management

1. Pain: Paracetamol
2. BP cuff (above the level of tourniquet)
3. Polyvalent ASV → No effect against humped nose viper

- Dose = > 10 – 30 vials – same for adult/ pregnant/ child.
- T½ of ASV ≈ 90hrs.
- Re-administration of Indian ASV → not required
  - 1 vial can neutralize 6 mg of Russel viper venom
  - Bite to needle time < 4 hrs
- To prevent reaction to ASV = Administer Hydrocortisone, H1 Blockers
- As ASV can result in anaphylactic shock → keep the adrenaline ready in syringe
- Anaphylactic shock can be recognized by presence of Stridor, Cyanosis , Crashing of blood pressure etc
- Adrenaline intramuscular is the 1st line Management of anaphylactic shock but i.m is avoided in case of ASV because there can be muscle hematoma in-case of viper bite and BP can be too less, so that absorption may not occur
- Therefore , I.V adrenaline is given in case of anaphylaxis due to ASV

INDICATIONS OF ASV

1. Coagulopathy
2. Neurotoxicity: 4P's

- Ask patient
  - To raise neck
  - Count numbers in one single breath
- In neurotoxicity, the patient can't do these

3. **CVS** abnormalities: BP ↓, tachy arrhythmia
4. **GIT** -Severe vomiting + abdominal pain → may be d/t retroperitoneal bleeding → diagnosed by CT/MRI abdomen
5. **Local** swelling: ≥ ½ circumference of arm /leg
6. Swelling has extended rapidly above the level of waist (after cutting tourniquet)

Refer Table 36.1

(Neostigmine is avoided as a solo drug to avoid cholinergic crisis)

Neostigmine + Atropine → more useful in neurotoxic cases [mainly cobra poisoning]

Recovery Phase

If an adequate dose of appropriate anti-venom has been administered, the following responses may be seen:

1. Spontaneous systemic bleeding such as gum bleeding usually stops within 15-30 minutes.
2. Blood coagulability is usually restored in 6 hours. Principal test is 20 min WBCT.
3. Post synaptic neurotoxic envenoming such as Cobra may begin to improve as early as 30 minutes after antivenom but can take several hours.
4. Presynaptic neurotoxic envenoming such as the Krait usually takes a considerable time to improve reflecting the need for the body to generate new acetylcholine emitters.
5. Active hemolysis and rhabdomyolysis may cease within a few hours and the urine returns to its normal color.
6. In patients who were in shock, blood pressure may increase after 30 minutes.

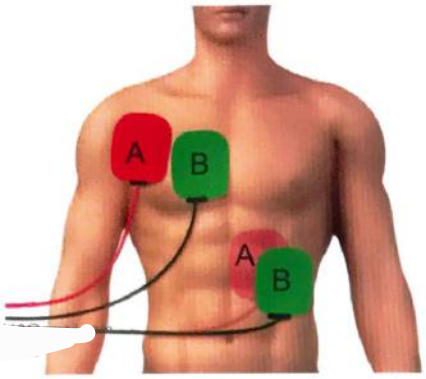
Table 36.1

Clinical features of snakebite

Feature	Cobra (post synaptic)	Krait (pre synaptic)	Russell viper	Saw scaled viper	Humped nose viper
Local Pain/ Tissue damage	Yes	No	Yes	Yes	Yes
Ptosis, Neurological sign	Yes	Yes	No*	No	No
Hemostatic abnormality	No	May Occur	Yes	Yes	Yes
Renal complication	No	No	Yes	No	Yes
Response to neostigmine	Yes	+/-	No	No	No
Response to ASV	Yes	Yes	Yes	YES	No

37 AHA 2020 UPDATE ON CPR GUIDELINES

1. DOUBLE SEQUENTIAL DEFIBRILLATION



Anteroposterior

- A patient has been given 3 DC shocks : Pt still in Ventricular fibrillation : Hook patient to 2 defibrillators

1st
- Right upper sternal border
- Left Cardiac apex

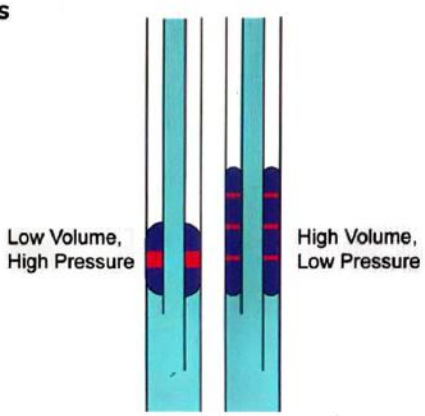
2nd
- Medial to paddles of 1
- Should not be in contact – to avoid damaging circuits
- 1 paddle can be placed posteriorly

- DOUBLE SEQUENTIAL DEFIBRILLATION is not supported.

2. EPINEPHRINE USE

- In pediatrics, within 5 minutes of CPR initiation
- Epinephrine in VF/V. Tachycardia → Enhances coronary perfusion (↓ Ishcemia)
- In Non-Shockable rhythm – Given ASAP
- In VF, CPR given with 2nd shock, Epinephrine also advised.

Pediatrics



- Even in Infants & children → Choose cuffed ETT > Uncuffed (↓chances of aspiration)
- Cuffed ETT: High Volume, Low pressure is chosen to reduce subglottic stenosis, cuff pressure < 20 cm H_2O
- Cricoid pressure is not recommended as it does not ↓ chance of aspiration
- Advanced airway (After intubation)
  - Old: 1 breath / 5-6 Sec i.e. 10-12 breaths /min
  - New: 1 breath / 2-3 Sec i.e. 20-30 breaths/min

STEROIDS

- Used successfully in COVID-19 Pneumonia
- Used for pediatric septic shock which is non-responsive to fluid / vasopressure (Epi/NE/Dopamine)

Refer Image 37.1

3. 6TH CHAIN ADDED TO In Hospital Cardiac Arrest (IHCA)

Refer Image 37.2

IHCA	OHCA
1. Early recognition & Prevention	1. Activation of Emergency response
2. Activation of Emergency response	2. High Quality CPR
3. High Quality CPR	3. Defibrillation
4. Defibrillation	4. Ambulance resuscitation
5. Post cardiac arrest care	5. Post cardiac arrest care
6. Recovery	6. Recovery

NEUROPROGNOSTICATION

Refer Image 37.3

In case of return of spontaneous circulation (ROSC)

- Ist 24 hours: Targettted temperature management: Therapeutic Hypothermia
  - Lower temp of brain
  - Metabolism of energy needs of neuron ↓: they can survive better
- Next 24 hours: Rewarming is done.
- And then controlled normothermia is ensured after rewarming.
- Biomarker: ↑ Neuron Sp. Enolase: marker of dying neurons
- EEG : Somato Sensory Evoked Potential returns (When Heading towards Brain death, BURST SUPPRESSION Pattern is seen, followed by Silence)
- First 24 hours: CT is recommended
- After 24 hours: MRI is recommended.

4. CARDIAC ARREST in Pregnancy

- **Etiology**
  - **A** - Anaesthesia
  - **B** - Bleeding
  - **C** - CVS (Pre-existing)
  - **D** - Drugs
  - **E** - Embolism
  - **F** - Fever
  - **G** - General: 5H and 5T
  - **H** - HTN

Maternal Intervention

1. Airway management
2. 100% O_2
3. IV Line ABOVE diaphragm
4. Mg So_4 stopped (Antidote – $CaCl_2$)

Fetal Intervention

- Perimortem C Section (If no return of circulation within 5 mins)

5. Early CPR For Presumed Cardiac Arrest

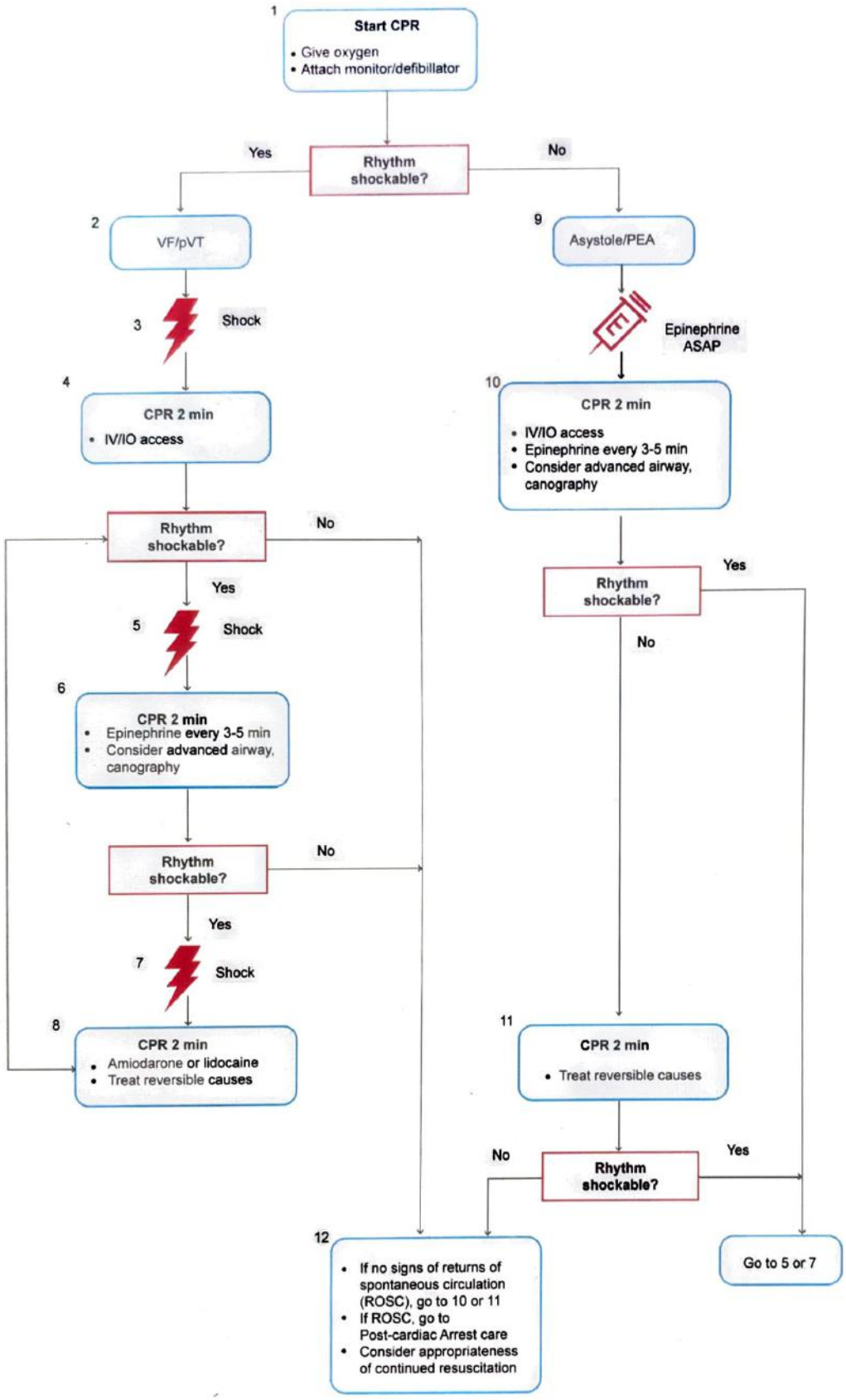
A	B
• Elderly man, DM, Hypoglycemia, Collapsed	• Elderly man: Known case of Chronic Stable Angina, V. Fib collapsed
↓ CPR ⊕	↓ CPR ⊕
• Minimal damage	• Recovery ++

- IF CPR is withheld in Outside Hospital Cardiac Arrest, the risk for damage is high.

6. **USE OF AUDIO – VISUAL FEEDBACK** during training for CPR to ↑ performance in Real time scenario
7. **ROSC**: ET $CO_2 \geq 20$ mm Hg: Higher Chances of ROSC.
8. **IV access is better than IO.**

Image 37.1

Adult Cardiac Arrest Algorithm



CPR Quality

- Push hard (at least 2 inches [5cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruption in compressions,
- Avoid excessive ventilation.
- Rotation compressor every 2 minutes, or sooner if fatigued.
- If no advanced airways, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  If $Petco_2$ <10 mm Hg, attempt to improve 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:** 1mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300mg bolus. Second dose: 150mg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airways in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of spontaneous circulation (ROSC)

- Pulse and blood pressure
- Abrupt susained increase in $PETCO_2$ (typically >40 mm Hg)
- Spontaneous arteries pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Image 37.2

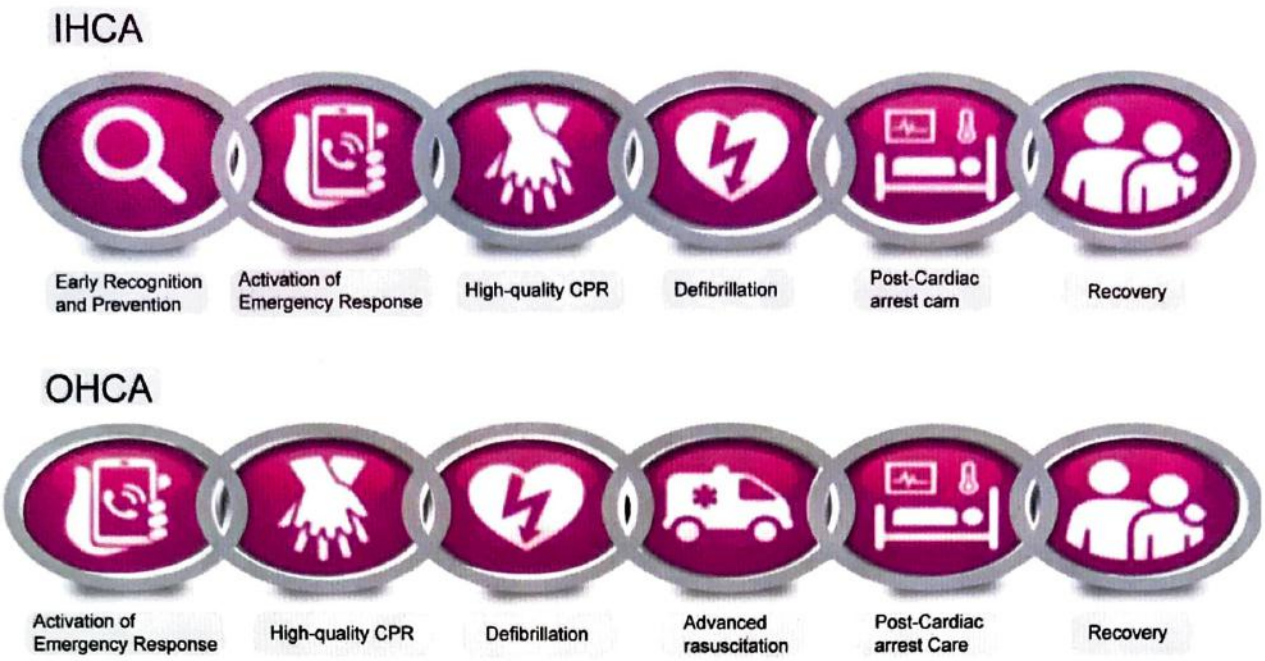
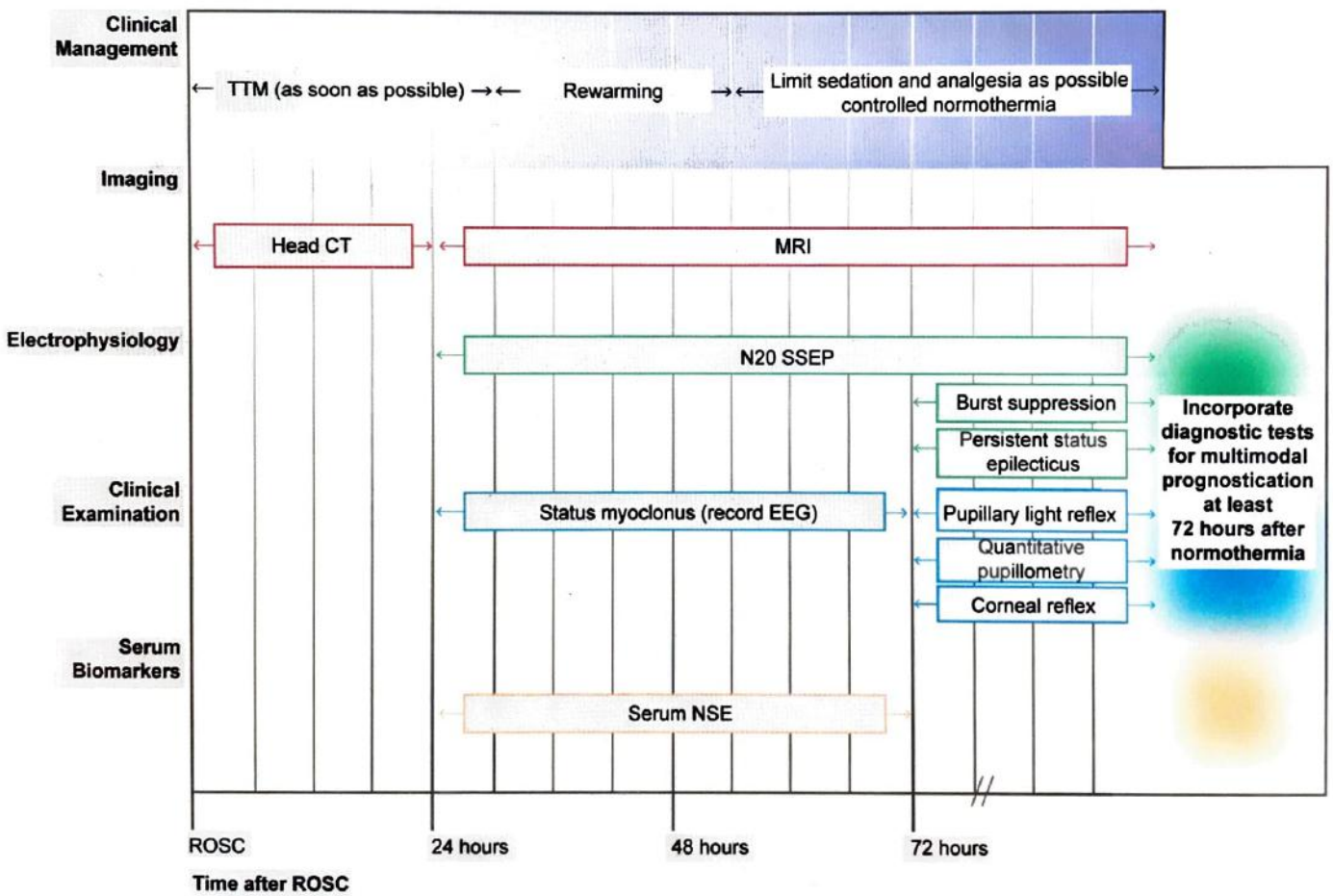


Image 37.3



38 HIGH ALTITUDE & PULMONARY EDEMA

High altitude pulmonary edema

SOME FACTS

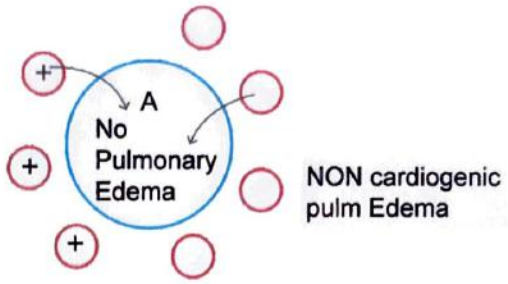
- High Altitude: > 2500m
- High altitude pulmonary edema usually develops on 2nd day and high altitude always causes hypoxia.
- If a person's is staying in high altitude for a longer time, he may feel headache due to low partial pressure of oxygen that results in cerebral vasodilation.
- At Mount everest , inspiratory partial pressure of oxygen is very less: 43mmhg.

ACUTE MOUNTAIN SICKNESS:

- Nausea, vomiting, Flatulence, Abdominal pain/ distension
- Milder form of disease
- Vomiting leads to Dehydration of the person and also person is having tachypnea on altitude that may worsen the dehydration by heat vaporization from the body.
- Acute mountain sickness is Prevented by Acetazolamide

HIGH ALTITUDE PULMONARY EDEMA / CEREBRAL EDEMA

- 1 atm = 760 mm of Hg
- Ascent ↑ : ipo_2 ↓ → Hypobaric hypoxia
- Cerebral Blood vessel dilatation will stretch the Dura mater which is pain sensitive → Headache
- Pulmonary blood vessel vasoconstriction : ↑ hydraulic stress on basement membrane of pulmonary blood vessel. This high pressure results in leakage of fluids from endothelium leading to Non cardiogenic Pulmonary Edema.



- Treatment of choice for HAPE → Immediate descent

Periodic Rhythm

- Most person who go to high altitude, cannot sleep properly due to periodic rhythm of both hyper and hypoventilation simultaneously , so oxygen saturation keeps on fluctuating so persons never feel fresh.

Sleep deficit/ fatigue

- **Acetazolamide** – Carbonic Anhydrase inhibitor
  - Urinary loss of HCO3-
  - Metabolic Acidosis
  - Hyperventilation
- Used to prevent Acute mountain sickness and helps to maintain bodies oxygen saturation by maintaining hyperventilation.
- Acetazolamide – No role in prevention of HAPE/HACE
- BAL (Bronchoalveolar Lavage) → ↑ RBC/↑ Proteins → represents integrity of blood vessels is lost.
- BAL in ARDS (Adult Respiratory Distress syndrome): Neutrophils ↑↑
  - Unacclimatization is a risk factor for HAPE

Clinical Features

- Young adult begins to deteriorate between day 2 to day 4 when he arrived at height 2000- 2500 m
- ↓↓ Exercise intolerance – Person feels breathlessness
- Non productive cough
- Frank Hemoptysis
- Cyanosis
- ↑↑ Heart Rate, ↑↑ Respiratory rate

- Person should drink water continuously to prevent dehydration.
- Crackles in the chest – Mostly involved middle zone of lungs.

Work up

1. CXR: Pulmonary edema on left and right middle zone, but there is no batwing edema, no cardiomegaly
2. ABG: Respiratory Alkalosis: CO2 ↓↓
- If PH is more than 7.7, then person won't survive.
3. Echocardiography: RV strain (Working against constricted PA)
4. PCWP: Normal, because this is an example of non cardio genic edema.

TREATMENT OF HAPE

1. DOC: Nifedipine 60 mg sustained release Tablet: Dilates pulmonary vessels which will decrease pulmonary artery hypertension and pulmonary edema component.
2. Immediate descent should be done.
3. If immediate descent not possible, then Gammow bag can help.
- Gammow bag → provide Stimulated descent above 2000 m. There is a hyperbaric oxygen available in this bag.
4. Supplemental o*xygen (4-6 L/min): sPo2 > 90%
5. Salmeterol / salbutamol with 40% alcohol nebulization /Tadalafil (PDF-5 Inhibitor)

SUMMARY

Acetazolamide	Nifedipine	Dexamethasone
↓	↓	↓
Acute mountain sickness	HAPE	HACE

LEARNING OBJECTIVES

ENDOCRINOLOGY

Pancreatic Neuroendocrine tumor

- Hypoglycemia
  - Levels of Hypoglycemia Causes and Treatment of Hypoglycemia
  - Treatment
- Insulinoma
  - Whipple Triad
- Insulinoma vs Glucagonoma
- Glucagonoma
- Dermatological manifestations seen in various diseases
- Somatostatinoma
- Vipoma
- Secretory Diarrhea

Diabetes Mellitus

- Classical symptoms of DM
- Diagnosis of DM
  - Hba1c test
- Complications of DM
  - Microvascular complications
  - Macrovascular complications
- Types of DM
  - Type 1 DM
  - Type 1.5 DM
  - Type 2 DM
  - MODY (maturity onset diabetes in young)
- Insulin supplementation
  - Uses and insulin delivery
  - Complications of insulin
- Hypoglycaemic drugs
- Long term complications of DM
  - Diabetic retinopathy
  - Diabetic neuropathy
  - Infections in DM
  - Diabetic dermopathy
  - Diabetic nephropathy
- Acute complications in DM
  - Diabetic ketoacidosis
  - Lactic acidosis
  - Non-ketotic hyperosmolar coma

Diabetic Ketoacidosis and Hyperosmolar coma

- Diabetic Ketoacidosis
  - TRIGGER
  - Pathogenesis
  - Examination
  - Investigations
  - Treatment
- Hyperglycemic Hyperosmolar Coma
  - Clinical Features
  - Work up
  - Treatment
- Comparison between Lactic Acidosis, DKA and Hyperosmolar Coma

Disorders of Parathyroid Hormone

- Functions of PARATHYROID HORMONE (PTH)
- Serum alkaline phosphatase
- Primary Hyperparathyroidism
- Raise Of Calcium & Its Manifestations
- Secondary Hyperparathyroidism
- Tertiary Hyperparathyroidism
- Hypoparathyroidism
  - Clinical features, investigations, and Treatment
- Acute Hypocalcemia/ Tetany
  - Manifestations
  - Work Up
  - Management
- Chronic Hypocalcemia
  - Clinical features, investigations, and Treatment
- Pseudo Hypoparathyroidism
  - Clinical features, investigations, and Treatment
- Pseudo Pseudo Hypoparathyroidism
- Comparison between HP, PHP & PPHP
- Nutritional Rickets

Disorders of Adrenal Gland

- Layers Of Adrenal Gland
- 1° Hyperaldosteronism
- Liddle Syndrome
- Addison's Disease
- Waterhouse-Friderichsen Syndrome
- Sheehan Syndrome
- Hypothalamus Damage
- CONN Syndrome Vs Addison disease
- Pheocromocytoma
  - Imaging modality
  - Clinical features
  - Tumor localization
  - Treatment

Cushing Syndrome

- Cushing Syndrome
  - Causes
  - Clinical features
  - Work Up
  - Investigations
  - Treatment
- Nelson Syndrome
- Hyperpigmentation

Disorders of Thyroid

- Thyroid Storm
  - Causes
  - Mechanism
  - Clinical features
  - Treatment
- Wolf Chaikoff effect
- Jod Basedow Effect
- Thyrotoxicosis Factitia
- Hyperthyroidism
  - Clinical features, investigations, and Treatment
- Grave's Ophthalmopathy
- Hypothyroidism
  - Clinical features, investigations, and Treatment
- Primary Hypothyroidism
- Secondary Hypothyroidism
- Complications Of Hypothyroidism
- Sick Euthyroid Syndrome

Disorders of Anterior Pituitary

- Acromegaly
- Gigantism
- Sheehan Syndrome
  - Imaging and Treatment
- Hypopituitarism
  - Clinical features, investigations, and Treatment
- Summary of various Endocrine disorders and diagnostic tests
- PROLACTINOMA
  - Clinical features, investigations, and Treatment

Multiple Endocrine Neoplasia

- MEON (Multiple Endocrine & Other Organ Neoplasia's)
- MEN I / WEMER Syndrome
- MEN-2 / Sipple Syndrome / MEN 2a
- MEN – 3 / MEN 2B
- MEN – 4

39 PANCREATIC NEUROENDOCRINE TUMORS

- Two types 00:00:11
  - Functioning NET
    - → M/c is Insulinoma
  - Non-Functioning NET
    - → M/c is PP-OMA (Pancreatic polypeptide)
    - → Doesn't cause any symptoms from hormones perspective
    - → Produces Chromogranin A/B
    - → Produce mass effect (mimic features of pancreatic adenocarcinoma)
      - Abdominal pain
      - Obstructive jaundice
      - Weight loss
    - → Diagnosed at later stages

HYPOGLYCEMIA 00:10:48

- If Sugar lever fall < 54 mg%– patient will have symptoms of Hypoglycemia
- Normally, When Blood sugar < 70 mg%
  - Alert value for DM patient
  - Insulin production stops
  - Counter regulatory hormone Increase
    - → Glucagon
    - → Catecholamines
    - → Growth hormone
- Levels of Hypoglycemia
  - Level 1
    - → 55-70%
  - Level 2
    - → <54%
    - → Ability to perform substantially decreased
    - → Lethargic and sleepy
    - → Neuroglycopenia starts
  - Level 3
    - → Requirement of assistance
    - → Hypoglycemic seizures
    - → Coma
- Causes of Hypoglycemia
  - Chronic kidney disease
  - Severe liver disease
  - Alcoholism
  - Poor nutrition
  - Extra pancreatic tumor
  - Insulin/Sulfonylureas abuse
  - Insulinoma
- Treatment
  - IV Dextrose
  - In case of failed IV access IM Glucagon

INSULINOMA 00:10:52

- It is Pancreatic Neuroendocrine Tumor
- Overall number 1 functioning Pancreatic Neuroendocrine Tumor
- High Insulin
- Usual age of presentation: 40-50 years of age
- Associated with MEN 1

Whipple Triad

1. Symptoms of hypoglycemia
   - Eccentric psychiatric behavior
   - Emotional outburst
2. Blood sugar <54 mg%
3. Immediate relief of symptoms with iv glucose

Insulinoma vs Glucagonoma

	Insulinoma	Glucagonoma
Malignancy	• Majority are benign • 5-15 % Malignant	• Majority cases are Malignant
Size	• Size at dx < 2 cm • difficult to diagnose at early stages with CT/MRI	• 5-10 cm
Location	• Equal distribution in all areas: Head, neck, tail of Pancreas	• Mc location at Tail of Pancreas

Insulinomatosis

- Patient has multiple micro or macro adenomas/ island of tumor in Pancreas behaving like Insulinoma.

Nesidioblastosis

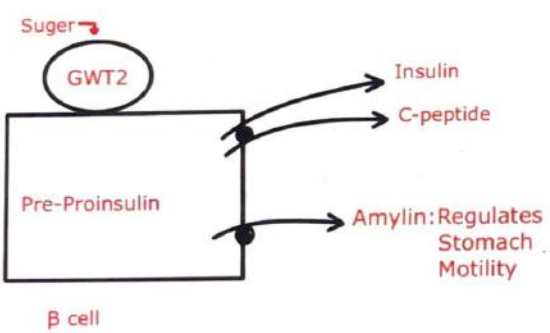
- it is a pediatric precursor of Insulinoma characterized by excessive insulin (hyperinsulinism) concomitant with Hypoglycemia

Clinical features of Insulinoma

- Frequent eating and Weight gain
- Concentration Span ↓
- Irritability
- Rage attacks and emotional liability
- Visual disturbance
- Irrational behavior
- Symptoms of sympathomimetics
  - Fine tremors
  - Heart racing
  - Diaphoresis
  - Palpitations

Work Up

- 72-hour prolonged fasting test
  - IOC
  - check Blood sugar Every 4 hours
- Low blood sugar levels < 40 mg% and RIA insulin elevated > 6 µu/ml
  - In normal persons when Blood sugar > 40 mg%
    - → Insulin production stops
    - → Insulin by RIA < 6 µu/ml
- C peptide elevated
  - Insulin produced by Pancreas comes into circulation along with amylin & C-peptide
  - Active Insulin has 51 amino acids
  - A chain = 21 AA
  - B chain = 30 AA
  - C-peptide = 33 AA, is a interlinking chain
  - Insulin coming in blood is pro insulin (inactive) form



- Proinsulin elevated
- Insulin/ glucose ratio = > 0.3
  - Normal value < 0.3
- Low plasma β OH butyrate values
- Check for Urine/ Plasma Sulfonylureas (to r/o factitious hypoglycemia as Sulfonylureas causes hypoglycemia like features)

Imaging

- Endoscopic Ultrasound
- Somatostatin receptor Scintigraphy/ Imaging (SRI)
  - useful in localizing pancreatic Neuroendocrine tumor including GI endocrine tumors Except Insulinoma
  - as they are small & have low densities of Somatostatin receptors
- PET CT -gallium 68
- MRI/CT abdomen → for tumors > 2cms

Treatment

- Frequent small meals
- Diazoxide
  - DOC
  - Insulin production ↓ from β cells hormone
- Octreotide/ Lanreotide
  - Somatostatin analog
  - Decreases GH and Blood sugar
- In case of malignant Insulinoma
  - mTOR inhibitors
    - → Everolimus
    - → Rapamycin
  - Chemoembolization
  - Radiolabeled Somatostatin

GLUCAGONOMA

00:31:13

- Mostly located in Tail of Pancreas
- Tumor is malignant; 5-10 cm in size
- Characteristic finding in skin: necrolytic migratory erythema

Clinical features

- **D** - **D**iabetes Mellitus / Impaired glucose Tolerance
- **D** - **D**iarrhea
- **D** - **D**ermatological features
  - Migratory necrolytic erythema
  - Rash→ Bullae→ Erosions
  - Present at Intertriginous/ Peri-orofacial sites
- Weight loss

How to remember

- 3D's

Work Up

- Plasma glucagon > 1000 pg/ml
- CT/MRI abdomen
  - Metastasis to liver 80%
- SRI – to identify location of tumor (Tail)
- Prognosis is bad as it is mostly malignant

Treatment

- Debulking surgery
- Radiolabeled somatostatin analogues

Dermatological manifestations seen in various diseases

- Necrobiosis lipoidica diabeticorum
  - characterized by erythematous lesion with central clearing
  - Grossly red and inflamed
  - usually on shin
- Acanthosis Nigricans
  - Diabetic patient with hyperpigmentation
  - Present on groin, axilla or back of the neck
  - Seen in PCOD, DM, Ca pancreas
- Migratory necrolytic erythema
  - Seen in glucagonoma, Myeloproliferative disorders

SOMATOSTATINOMA

00:37:32

- Excess of somatostatin
  - Inhibitor of other hormones
  - It is a tetra-decapeptide
- Found in CNS (in pituitary)/GIT

Clinical Features

- **D - D**iabetes Mellitus
  - Due to inhibition of insulin
- **D** - Secretory **D**iarrhea
  - Due to inhibition of pancreatic amylase
- **G - G**all bladder disease
  - Incidence of gallstones increases
  - Due to inhibition of CCK
- Steatorrhea
  - Due to inhibition of pancreatic amylase
- Hypochlorhydria

How to remember

- DDG

Imaging

- Somatostatin receptor Scintigraphy/ Imaging (SRI)

VIPOMA

00:40:42

- Excess production of vasoactive intestinal polypeptide
- It is series of 28 AA peptide
- Released by F cells of pancreas
- Present in both GIT/CNS
  - Causes vasodilation
- Mechanism
  - Causes opening of water channel in gut
    - → Causing secretory diarrhea
    - → Consistency of stool is just like stools in cholera. Therefore, also known as Pancreatic cholera
  - Large volume depletion cause
    - → Hypokalemia
    - → Dehydration
    - → Achlorhydria
    - → If severe → M. Acidosis

Clinical features

- Rice water stools
  - Aka Pancreatic cholera
  - Secretory Diarrhea
- Hypokalemia
- Flushing episodes
  - D/D Carcinoid syndrome
- Achlorhydria
- Hyperglycemia
- Elevated S. Calcium

Important Information

- **VIPoma is also called as WDHA**
  - **Watery Diarrhea**
  - **Hypokalemia**
  - **Achlorhydria**

SECRETORY DIARRHEA

00:44:42

- Nonresponsive to fasting
- Causes
  - **D - D**iabetic diarrhea
  - **M - M**edullary carcinoma thyroid
  - **M - M**astocytosis
  - **C - C**arcinoid syndrome
  - **C - C**holera and Pancreatic Cholera
  - **L - L**axative abuse
  - **G - G**astrinoma

How to remember

- DMCLG

- Stool osmolar gap
  - Differentiate b/w secretory and osmotic diarrhea
  - stool osmolar gap = 290 – 2 [(stool Na+) + (Stool K+)]
  - If stool osmolar gap > 100 = osmotic diarrhea
  - If stool osmolar gap < 50 = secretory diarrhea
- Treatment
  - Octreotide

Important Terminologies

- P. NET: Primitive Neuroectodermal Tumors
  - Neuroblastoma
- PpNET: Peripheral primitive Neuroectodermal tumor
  - Medulloblastoma
  - Ewing Sarcoma

CLINICAL QUESTIONS

Q. A 27-year-old male patient, brought in with history of sudden loss of consciousness which lasted for almost 45 minutes. He had similar history of episodes of light headedness associated with diaphoresis, palpitation, tremulousness, feeling of impending doom in past, off and on for last 3 years, which were relieved with eating something or taking glucose water orally. He had no other significant past medical history. No family history of diabetes. Initial investigations were done. The physician suspected this as a case of insulinoma. Which is the most sensitive Imaging modality of choice for this diagnosis?

A. USG
B. CT abdomen
C. Somatostatin Receptor Scan (SRS)
D. E.U.S

Answer: D

Solution

- For PETs in the pancreas, EUS is highly sensitive, localizing 77-100% of insulinomas, which occur almost exclusively within the pancreas.
- Tests for location of pNET:
  - Somatostatin Receptor Scintigraphy is the initial imaging modality but is less available.
  - Helical CT scan has a sensitivity of 82-94%.
  - Gadolinium based MRI has sensitivity of 85%.
  - If above scans are negative, then Endoscopic ultrasound will be able to pick up the insulinoma which is usually <1.5 cm in size.
  - If all the above tests turn negative then calcium stimulated angiography can be used to localize the tumor.
- The intra-arterial calcium test also allows differentiation of the cause of the hypoglycaemia and indicates whether it is due to an insulinoma or a nesidioblastosis.

Reference: Harrison's Principles of Internal Medicine - 20th Edition, Page No: 2887

40 DIABETES MELLITUS PART-1

CLASSICAL SYMPTOMS

00:01:00

- Polyuria
  - >3L/day or >40 ml/kg
- Polydipsia
  - >6L H_2O intake/ day
- Polyphagia
- Weight loss

Important Information

Polyphagia is not a classic symptom of diabetes mellitus as it is not measurable.

DIAGNOSIS

00:06:00

- Classic Symptoms + RBS ≥ 200mg%
- Asymptomatic Patient with FBS > 126mg%
- After Giving 75gm glucose → 2hr value > 200mg%
- Asymptomatic patient → HbA_1C (glycosylated Hb) ≥ 6.5% (best answer)
  - Normal HbA1C = ≤ 5.6%
  - IGT = 5.7-6.4%
  - DM = > 6.5 %
- Cut off values for DM acc to American Diabetes association

	Normal	Impaired Glucose tolerance	Diabetes Mellitus
Fasting	100 mg%	100-125 mg%	≥ 126 mg%
2-hour value after 75 gm glucose	<140 mg%	140-199 mg%	≥ 200 mg%

Important Information

Postprandial values are not useful for diagnosing DM. It is useful for follow up & monitoring

Previous Year's Questions

Q. 20-year-old military recruit is found to have a HbAIC of 6.1% and FBS of 120 mg%. which is correct about this patient? (FMGE June 2019)

A. Normal
B. Impaired glucose tolerance
C. Diabetes mellitus
D. Maturity onset diabetes in young

HbA_1C test

- It is retrospective test
- It gives average value of blood sugar level of last 3 months (8-12 weeks)
  - This is not one-time Value
- Not effected by exercise, fasting and recent food intake
- If HbA1C value >8%, multiply by 25, it gives average blood sugar value
  - E.g.: HbA1C=10%; Average blood sugar= 250mg%
- If HbA1C value <8%, multiply by 21, it gives average blood sugar value
  - E.g.: HbA1C=7%; Average blood sugar= 147mg%

Important Information

- IOC/ Best/ Most specific test/ Best test for Long term control/ Best test for severity in DM is HbAIC
  - TARGET HbAIC < 7% → Good sugar control
- Best test for short term control is Serum fructosamines (Glycated albumin)
  - Retrospective test
  - Formed by Non enzymatic glycosylation of serum proteins
  - Average value of blood sugar level of last 2-3 weeks
- Best test for control in bronze diabetes is Serum fructosamines (Glycated albumin)
  - As HbAIC values are less in these patients

COMPLICATIONS

00:26:55

MICROVASCULAR COMPLICATIONS

- It is directly proportional to HbA1C levels
- Best way to reduce the incidence of MICROVASCULAR complications is HbA1C CONTROL
- Retinopathy
  - 25 times Increase incidence of blindness
- Neuropathy
  - Increase Incidence of Silent MI
  - Overall m/c complication of DM
- Nephropathy
  - MC cause of CKD in India / USA

MACROVASCULAR COMPLICATIONS

- Strict BP control
- Coronary Artery Disease
  - M.I, Unstable angina, prinzmetal angina etc.
  - 3-5 times increase in risk
- Peripheral Artery Disease
  - 100 times increase in risk
  - increase incidence of gangrene
- Stroke
  - Due to accelerated Atherosclerosis

Important Information

Best way to reduce the incidence of MICROVASCULAR complications is HbAIC CONTROL and Best way to reduce the incidence of MACROVASCULAR COMPLICATIONS is Strict BP control

Previous Year's Questions

Q. MC joint involved in diabetes is seen is?
(FMGE June 2019)

A. Ankle
B. Knee
C. Shoulder
D. Foot

Management

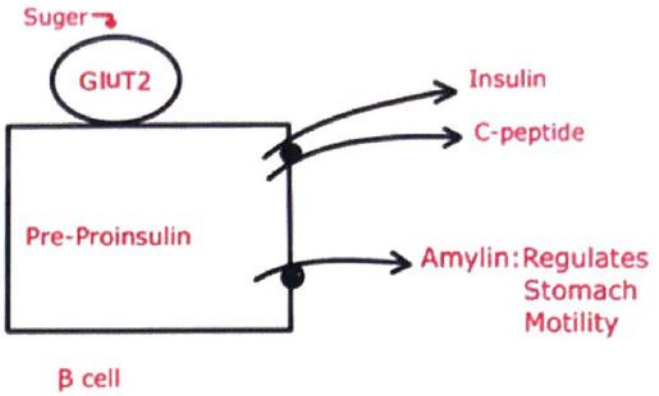
- Depends upon type of Diabetes

TYPES OF DIABETES

00:34:05

TYPE I DM

- Beta cells are damaged
- If >80% β-cell mass is damaged, clinical features appear
- Beta cells are damaged → decrease production of insulin → resulting in INSULINOPENIA
- Insulin and C - peptide are released at ratio of 1:1
  - Insulinoma → both insulin & c-peptide values increases
  - DM → both values decreases
- Apart from insulin, hormone released by β-Cell is AMYLIN
  - It regulates stomach motility
  - Regulates entry of osmotic contents into small intestine



Important Information

- GLUT-2 is sensor that continuously monitors blood sugar

- If Glucose level is < 70 mg%,
  - Insulin production stops
  - Glucagon production starts
  - Increase in Glucagon, Growth hormone, Cortisol, and catecholamines
- Cut off value for Diagnosis of hypoglycemia
  - Adults <55 mg%
  - Pediatrics <45mg%
- K+ channel helps in release of insulin in circulation

Case scenario T1DM

- 8yr child admitted with pneumonia, IV Azithromycin is given, after 48 hours of admission Drowsy and decreased ORAL intake & pulse oximeter: spo2: 98% on Room Air. On ABG/VBG PH=7.2, PCO2 = 40 mm Hg, HCO3 =15 meg (22-26 meg), urine ketones: ++ and RBS = 300 mg %
  - Diagnosis: Diabetic ketoacidosis
  - Mechanism of injury: Insulinopenia and increased energy requirement in pneumonia causes SC fat oxidation and formation of ketones. As ketones are acidic in nature causes damage to blood brain barrier and cerebral oedema
  - IOC: Plasma Beta hydroxy butyrate

- Treatment: 1st line NS drip (RL is C/I as causes hyperkaliemia and diastolic arrest)
- TOC: Regular Insulin drip

Causes

- Autoimmunity
  - Most Common cause
  - HLA DQ2, DR 3, DR 4 genes are involved
- Viral causes
  - Causes Fulminant Diabetes
    → DM developing with in 8-12 weeks period
  - Coxsackie B
    → MC viral cause
    → Can also cause viral myocarditis
  - Mumps, Rubella
- Bronze diabetes
  - Caused d/t Iron toxicity
  - Triad of bronze dm
    → Bronzing / Hyperpigmentation of skin (increase in melanin)
    → Cirrhosis
    → Beta cell mass decrease: Insulinopenia OR Type-I DM
    → Fasting insulin level ↓ by radio immune assay (RIA)

00:59:15

Treatment

- Carbohydrate counting
  - Regulate the amount of carbohydrate intake & give insulin proportionate to it
  - 1 unit of lispro is sufficient to neutralize 15g of Carbohydrates
- Insulin supplementation

Previous Year's Questions

Q. MCC of fulminant diabetes is? (FMGE June 2019)
A. Viruses
B. Diabetic ketoacidosis
C. Non ketotic hyperosmolar coma
D. Autoimmunity

INSULIN SUPPLEMENTATION

01:01:55

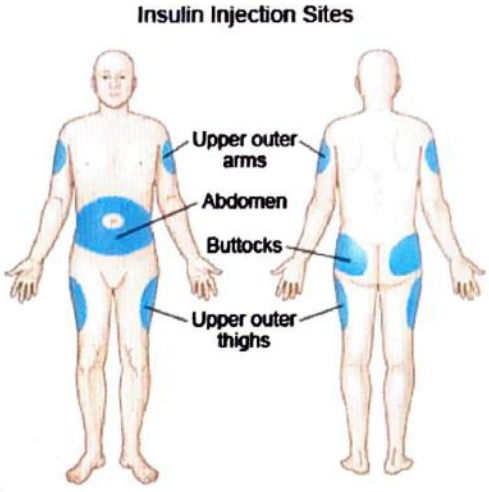
- Longest acting insulin: Degludec
  - Act for 42 hours
- Mc route of administration: Subcutaneous
- Sites of insulin injection
  - Anterior abdominal wall

→2cms away from umbilicus in the form of round circular band)

  - Arm
  - Anterolateral aspect of thigh
  - Buttock

Important Information

- **Never give insulin in Dorsum of Hand**

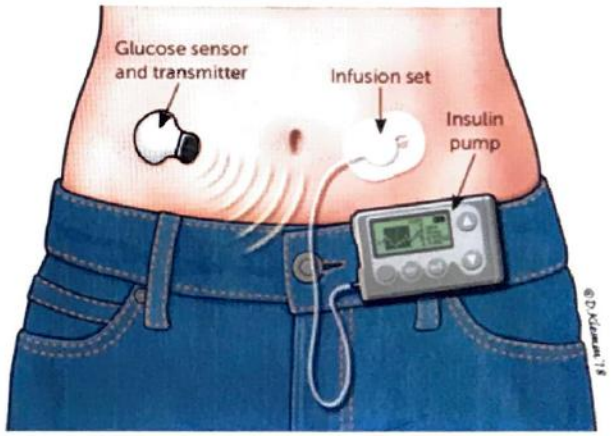


Uses

- T1DM
- T2DM
  - Newly diagnosed
  - Beta cell exhaustion
- Diabetic with Pregnancy
- Diabetic with End organ damage

Insulin Delivery

- Insulin Pump
  - Best method
  - Mimics artificial pancreas
  - Microprocessor device
  - Continuous SC insulin infusion
  - Continuously Releases Basal Insulin into body
  - Bolus insulin delivery at Mealtimes is also programmed proportionate to carbohydrate intake
  - It is very expensive



- Insulin Pen: (31 G)
  - Painless and Bloodless delivery of insulin
- Inhaled Insulin
  - Afreeza / Exubera
- Multidose Vial + 1ml syringe

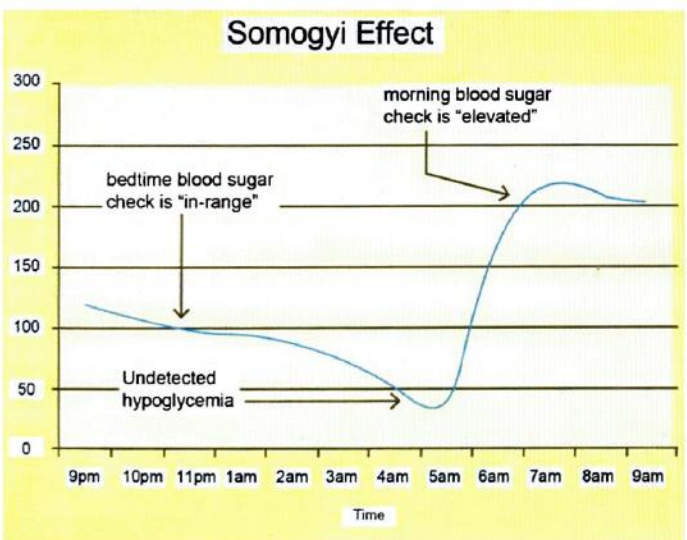
COMPLICATIONS OF INSULIN

01:09:28

- Hypoglycemia
  - Blood sugar <54mg%
  - Clinical features
    - → Sympathetic system stimulation
    - → Rage attacks
    - → Emotional lability
    - → Diaphoresis
    - → Drowsy
    - → Stupor
    - → Seizures
  - Beta blockers are contraindicated in DM as they mask the symptoms of hypoglycemia
  - Treatment
    - → IV 25-50% Dextrose
    - → In case of failed IV access INJ glucagon Sc
  - Avoid long-acting insulin
  - Avoid intramuscular injection
- Weight gain
- Somogyi phenomenon

01:17:28

  - Early morning hypoglycemia d/t intake of overdose of insulin at bedtime
  - At 4A.M → Person wakes up with sympathetic symptoms like Palpitations, Tremors, Diaphoresis etc.
  - At 7A.M → Glucagon in circulation → glycogenolysis → Blood sugar value → pre-breakfast hyperglycemia
  - Thus, main manifestation of Somogyi phenomenon is EARLY MORNING HYPOGLYCEMIA.



- Dawn phenomenon

01:22:28

  - 4 AM: Early morning hyperglycemia → D/t downregulation of GLUT-4 receptors in muscle in T2DM
  - 7 AM: Pre-breakfast hyperglycemia

Previous Year's Questions

Q. 50-year-old man on admission to ER has blood sugar of 350mg% and shows evidence of dehydration with ketones detected in urine. Lab work up shows serum electrolytes report revealing Na+130 mEq/L, potassium+ 3.8 mEq/L. The patient is on insulin, but stopped using due to personal issues. What should be added to saline? (FMGE June 2021)

A. Glucose
B. Insulin
C. Soda bicarbonate
D. Potassium chloride

Previous Year's Questions

Q. In a newly diagnosed case of sick child with type I diabetes mellitus (DM), insulin was given. Which of the following will increase? (FMGE Dec 2017)

A. pH
B. Breathing rate
C. Glucosuria
D. Urine osmolality

Previous Year's Questions

Q. Which type of insulin is used to manage a case of diabetic ketoacidosis? (FMGE June 2019)

A. Regular
B. Lispro
C. Glargine
D. Aspart

41 DIABETES MELLITUS PART-2

TYPE 1.5 DIABETES MELLITUS

00:00:12

- Aka LADA → Latent autoimmune Diabetes in Adults
- Variant of Type- II D.M presenting in adults (Shares etiology with Type-1 DM)
- Anti-GAD:Anti-Glutamic acid decarboxylase antibody (Main Ab)
- Anti-islet cell Antibody (ICA) → 2nd Ab
- Anti-GAD- also present in paraneoplastic manifestation of oat cell Ca of lung K/A stiff Person Syndrome
- Insulinopenia is seen

Treatment

- Start initially with Glipizide / Sulfonylureas
- Insulin: synthesized by DNA Recombinant Tech (Human)
  - Beef/ Pork insulin (S/E: lipodystrophy, Resistance)
  - Route: Subcutaneous route, in abdominal wall & insulin pump is preferred
  - Rapid acting insulin
    - → Lispro/ Aspart/ Glulisine
    - → Given by S.C route, 15-25 minute before meal
    - → Mc recommended
  - Long-acting insulin
    - → Glargine (~ 24 hours)
  - Ultra- long acting
    - → Degludec (~ 42 hour)
  - Regular insulin→ short acting
- Pramlintide (best answer) – Used for both Type 1 & Type 2
- Acarbose can also be used (off label) for both Type 1 & 2, as it inhibits absorption of sugar from G.I.T

Important Information

- Uses of SC Insulin
  - Type I DM → Insulinopenia
  - Type 2 DM → refractory to oral hypoglycemic drugs
  - Type I.5 DM

Important Information

- I.V uses of Regular insulin
  - Hyperkalemia
  - DKA (Type I)
  - Non-Ketotic Hyperosmolar Coma (Type II)
  - Regular insulin can be given in both I.V & S.C routes

TYPE 2 DM

00:14:27

- Polygenetic (Doesn't follow mendelian law of inheritance)
- Insulin resistance is d/t
  - Resistin
  - High levels of Adipokines → promote weight gain
  - Decreased levels of Adiponectin: Guardian angel against obesity

Treatment

- Diet control (1st line intervention in T2DM)
  - Carbohydrate intake is strictly regulated.
  - Carbohydrate with low glycemic index → Oat, multigrain bread, egg white, brown rice, white meat, and multigrain Atta
  - Fats → Butter / Ghee
  - PUFA → Safflower Oil
  - Transfat → Olive Oil, Sunflower Oil
- Exercise
  - Increases GLUT4
    - → Promotes Sugar entry into muscle resulting in decreased blood sugar level
  - Jogging: 30 min X 5 DAYS / week is recommended (150 min/week)
- Oral Hypoglycemic Drugs
  - causes HbA1C control

HYPOGLYCEMIC DRUGS

00:26:49

BIGUANIDES

- Inhibit Hepatic gluconeogenesis
  - HbA1C is decreased by 1.5 - 2% over a period of 3-6months (slow acting)
  - Metformin
  - Excreted via kidney

- Causes Lactic Acidosis if given in patient with nephropathy
- Avoided in kidney damage
- Metformin comes as 500 mg / 850 mg / 1 gm. Max dose → 2.5 g/day.

Important Information

- **DOC for obese T2DM: Metformin**
- **DOC obese T2DM + nephropathy: Linagliptin / Glipizide / Tolbutamide**
  - **These drugs are metabolized by liver**
  - **Metformin is contraindicated in nephropathy.**

- Side effects of metformin
  - Nausea / vomiting
  - Lactic acidosis: if there is Concomitant renal failure
  - B12 deficiency
- Phenformin
  - Not given Nowadays as it causes lactic acidosis (Drug induced lactic acidosis → Type B LA)
  - If Biguanides are not successful, give sulfonylureas.

SULFONYLUREAS 00:36:45

- ATP sensitive K+ channel is blocked → Burst of Insulin → ↑ Receptor sensitivity
- Decrease HbA1C by 1.5%
- S/E- Hypoglycemia attack (Needs Time Regulation)
- 1st Generation → Tolbutamide
- 2nd Generation → Glibenclamide, Glipizide, Gliclazide
- 3rd Generation → Glimepride

THIAZOLIDINEDIONES

- Major side effects are reported in phase-4 clinical trial (post marketing surveillance)
- Pioglitazone (bladder cancer), Rosiglitazone (CHF)
- Decrease HbA1C by 1%
- MOA → ↑ Peripheral Utilization of glucose by ↑↑ GLUT4 Receptors on muscle / adipose tissue

ALPHA-GLUCOSIDASE INHIBITORS

- DRUGS: Acarbose, Voglibose
- These are taken with meals. They act by inhibiting sugar absorption from GIT.
- Decreases HbA1C by 0.25%
- Control P.P spikes
- S/E: Osmotic diarrhea
- MOA: ↓ Serum triglyceride

MEGLINITIDE

- Drug: Repaglinide.
- Behave like sulfonylureas
- Insulinogenic action → causes burst of insulin into circulation
- Also improves sensitivity of receptor
- S/E – Hypoglycemia
- MOA → Insulinogenic (Burst of Insulin ↑ → Receptor Sensitivity ↑)
- Short acting drug (t ½) = 1-2 hours, So for control of post prandial spike of Sugar

DPP-4 INHIBITORS 00:49:00

- Inhibits degradation of GLP-1 (Glucose limited insulotropic Peptide → Sends sugar into muscles). Thus, increases duration of action of GLP-I

- Drugs: Linagliptin (metabolized by Liver); Sitagliptin (metabolized by kidney)

INCRETINS

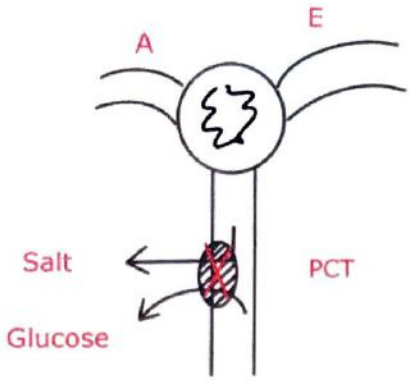
- Terminal ileum has 'L' cells
  ↓
  Produce a hormone which Behaves like insulin
  ↓
  ↑ GLP - 1

- Drugs
  - Exenatide
    → Injection once a week
    → Expensive
    → brand name: ByeTTA
  - Liraglutide
    → Given by Parenteral Route (S/E: Hemorrhagic Pancreatitis)
    → Expensive

SGLT – 2 INHIBITORS

- Promotes urinary loss of sugar resulting in decrease of blood sugar level
- S/E → Incidence of UTI increases
- Drugs: Canagliflozin
- These ↓ Cardiovascular mortality
- SGLT-2 Transporters are present in PCT and help in reabsorption of both salt and sugar
- Pramlinitide → Used in both Type 1 & Type 2 DM
- In newly diagnosed patient of TYPE-2 DM, we can start with combination of Metformin (decrease HbA1C) & Acarbose (prevent P.P spikes)
  - Decreases overall comorbidity

- Initially in Type-2 DM → Insulin levels INCREASES
- But later, d/t Beta cell exhaustion → insulin levels fall → hence insulin injections are useful.



Drugs decreasing HbAIC	Drugs for P.P spikes
• Metformin • Glipizide • Pioglitazone	• Acarbose • Repaglinide • Sitagliptin • Exenatide • Canagliflozin

MODY (MATURITY ONSET DIABETES IN YOUNG)

01:03:06

- AD (Autosomal Dominant- 75% chance of expression of disease in next generation)
- TYPES – 6types → MODY 1-6
- MC type in India/ USA – MODY 3
- Gene involved in MODY 3: HNF 1 Alpha
- Chromosome involved in MODY-3 : Chromosome-12
- Insulin secretion defective d/t defective genes
- Case Scenario: MODY
  - 25 yr. Asymptomatic, FBS > 250 mg%, Father DM +, Grandfather DM +
  - Expressed in every generation
  - Radio Immune Assay of insulin → normal/low
- Case Scenario: Type II DM
  - Grandson -40yrs DM+, Father – Normal, Grandfather – +
  - RIA insulin levels increases

Important Information

- GENE INVOLVED IN INSULIN PRODUCTION → CTL-4A → its defect leads to insulinopenia

Treatment of MODY

- Initiation of treatment by Sulfonylureas → stimulate β-cells to produce more insulin.
- Diet control
- Exercise

		Why?	Treatment
T_1DM	Insuline	Autoimmune, Pediatric onset	Insulin S.C
T_2DM	Insulin ↑	Resistin, adipokines ↑, adiponectin ↓	Metformin
$T_{1.5}$DM	Insulin ↓	Anti-GAD, Adult onset	Sulfonylureas / insulin
MODY	Insulin ↓/ N	AD, Ch12 defect – in MODY-3	Sulfonylureas

Important Information

- MC type of DM → Type-2 DM
- Rarest type → MODY
- Most Acidic ketone body – Beta hydroxyl Butyrate → IOC for DKA
  - High levels of beta hydroxyl butyrate with elevated blood sugar is the best way to diagnose DKA
  - Urine dipstick → low sensitivity & specificity

LONG TERM COMPLICATIONS IN DIABETES

01:13:13

1. DIABETIC RETINOPATHY

- Severity α Duration of DM
- DM with HbA_1C > 7% (poor sugar control) progresses to D. Retinopathy / Nephropathy in
  - T_1DM = 5 years
  - T_2DM= 20 years
  - Both Progress simultaneously

Important Information

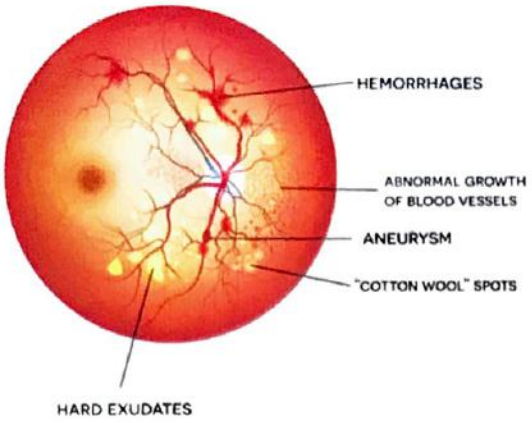
- Progression of retinopathy is monitored by raise in serum homocysteine levels
  - Best method

Treatment

- Vit. B6 (best answer) /Vit B9 / Vit B12
- Raise in serum creatinine
  - Raises after 60% of kidney is damaged
- Fundus Examination:
  - Microaneurysm
- Earliest seen in Inner nuclear layer

NON-PROLIFERATIVE RETINOPATHY

- Macular edema → leads to visual loss



PROLIFERATIVE RETINOPATHY

- Clinical Features
  - Neovascularization
  - Vitreous hemorrhage
  - Tractional Retinal Detachment
- Treatment
  - Pan Retinal photocoagulation by ND: YAG → destroys pre-existing Blood vessels
  - Bevacizumab (ANTI-VEGF) → Intravitreal injection → prevents formation of new B.V

Important Information

- Cause of visual loss → macular edema
- Cause of blindness → tractional retinal detachment

PRIMARY PREVENTION OF BLINDNESS IN DIABETIC

- By controlling levels of HbA1C < 7% → Best method (even if HbA1C <7%, leakage of dye on fundus fluorescent angiography can't be prevented); Normal value of HbA1C= < 5.6%
- Pan Retinal Photocoagulation
- Bevacizumab Injection.

2. DIABETIC NEUROPATHY

01:23:21

- 50% of DM Patient will develop neuropathy
- Irreversible process
- Sequence of involvement → Sensory > motor> Autonomic

Sensory Involvement

- Vibration sense → First affected → tested by tuning fork of 128 Hz
- Small unmyelinated C-fibers are involved → Hence DM is called → Small fiber neuropathy
- Clinical feature: pain in soles of feet (nocturnal pain)
- Another cause of small fiber neuropathy → HIV

Treatment

- Duloxetine
- Pregabalin
- Amitriptyline

Important Information

- In DM → the combination of following factors explains development of NON-HEALING ULCER (Neuropathic ulcer) in heel & metatarsal heads
  - Decreased pain sensation
  - Angiopathy→ decreased healing
  - Increased sugar in local tissues → ↑ Bacterial Infection
- Non-healing ulcer in sole of feet → think of DM
- Non- Healing ulcer in hands → think of Syringomyelia (Malformation of spinal cord)

Motor Involvement

- Cranial Nerve involvement
  - MC cranial nerve involved is 3rd Cranial Nerve
- 3rd nerve palsy with pupillary sparing is seen.

Important Information

Conditions in which 3rd cranial nerve palsy is seen

- Ruptured & unruptured Berry aneurysm
- Weber syndrome (d/t midbrain stroke)
- Miller Fisher syndrome (variant of GBS having descending paralysis)
- Diabetes mellitus

3rd Nerve Palsy with Pupillary Sparing	3rd nerve Palsy with Pupillary Involvement
• Unruptured Berry aneurysm • AV Malformation • Diabetic Neuropathy	• Weber Syndrome • Claude Surgery • Benedicts Surgery • Miller Fisher Surgery

Autonomic Involvement

- Silent MI → Leading cause of sudden death
- Hypoglycemic Unawareness
- Postural hypotension
  - SBP ↓ > 20 mm of Hg on changing position from supine to standing
  - DBP ↓ > 10 mm of Hg

- Calculate B.P change from supine to standing position in a time lag of 3 mins
- Midodrine – DOC for postural hypotension
- Constipation (sympathetic action dominating)/ Diarrhea (vagal action dominating)

Important Information

6th Nerve Palsy is Seen in:

- Raised ICP as 6th nerve has longest intradural course. It is false localizing sign because disease is
- not present in 6th nerve
- Millard gubler syndrome → d/t pontine stroke (7th nerve is also involved)
- Wernicke's Encephalopathy → feature of Dry Beri Beri (d/t involvement of thalamus)

7TH Nerve Palsy

- Unilateral 7th nerve palsy: Bell's palsy
- B/L LMN palsy of 7th nerve: feature of Facial diplegia. Seen in
  - G.B.S
  - Sarcoidosis
  - Melkersson-rosenthal syndrome

3. INFECTIONS IN DM

01:37:54

- Case scenario: 60-year maleT2DM, H/O aggressiveness, antisocial behavior, black nasal discharge O/E: Nasal speculum; Black fungal mass seen in both nostrils, MRI head → shows fungal mass at cribriform plate (roof of nose)

- Diagnosis
  - RHINOCEREBRAL MUCORMYCOSIS
  - Anti-social behavior is d/t extension of fungal mass from roof of nose to frontal lobe of brain
- Treatment
  - IV. Amphotericin B

Other Infections

- Aspergillus Niger
  - Angio-invasive i.e., invasive aspergilloma → causes Hemoptysis
  - CT chest: shows Cavity with fungal Ball
  - Treatment: I.V voriconazole

4. DIABETIC DERMOPATHY

01:41:14

- Non-healing ulcer → MC dermopathy in DM

- Acanthosis nigricans
  - Type 2 DM + hyperpigmentation (At extensor aspects like back of neck etc.)
  - Also seen in
    - → PCOD
    - → Ca Pancreas/ colon

- Necrobiosis Lipoidica Diabeticorum
  - Ulcer seen in shin of patient
  - Differential Diagnosis: Martorell ulcer
  - Seen in shin d/t atherosclerosis

5. DIABETIC NEPHROPATHY

- Discussed In Kidney Section

ACUTE COMPLICATIONS IN DM (CASE BASED DISCUSSION)

01:44:27

Case scenario

- Unconscious, unresponsive known case of (T2 DM) patient is brought to hospital. Vitals are secured. First investigation to be done is:
  - Consider hypoglycemia & perform capillary blood sugar values by → DEXTROSTIX
  - If value is 20-40mg%, treat the patient by giving 25-50% Dextrose I.V
  - If the patient presents with Hypoglycemic seizures & failed IV access, give Injection of Glucagon/ adrenaline by Subcutaneous route
  - However, if blood sugar is elevated, in Dextrostix (400-600mg%), following differentials are to be considered

- In Non-Ketotic Hyperosmolar Coma
  ↓
  Sugar crosses Blood Brain Barrier
  ↓
  Water follows sugar
  ↓
  Cerebral Edema

	LACTIC ACIDOSIS (Type – A)	D.KETO ACIDOSIS	NON-KETOTIC HYPEROSMOLAR COMA
URINE KETONES	Negative	Positive	Negative
pH (arterial blood gas analysis)	< 7.35	< 7.35	7.40
HCO3- (22-26 meq)	< 22	< 22	24meq
BLOOD SUGAR	300mg%	300mg%	600mg%

- It presents with Hemiballismus → sudden, violent jerky movements in proximal & distal parts of the body d/t involvement of subthalamic nucleus
- Treatment
  - Fluid of choice: Normal saline along with regular insulin infusion (10L in 48hrs to treat intracellular dehydration)
  - For every 100mg% increase in value of sugar, Na+2 decreases by 3meq
  - Reason: sugar draws water resulting in DILUTIONAL HYPONATREMIA

Important Information

- Glucose level (< 30-45mg%) → NEUROGLUCOPENIA → causes hypoglycemic seizures/ stupor & coma
- Blood glucose level <70mg% → insulin production stops
- Hypoglycemia cutoff → 55mg%

- In DM, usually Lactic Acidosis is triggered by intercurrent infections (E.g., Pneumonia, UTI etc.)/ stroke/ MI → It is TYPE A Lactic Acidosis

Important Information

Indications of normal saline

- Decompensated Shock: very low SBP (unrecordable B.P)
- Metabolic alkalosis
- DKA
- Non-ketotic Hyperosmolar coma Indications of ringer lactate solution
- Burns
- Cholera
- Shock (but in crushing Patient with unrecordable B.P → use Normal saline)

CLINICAL QUESTIONS

Q. A 75yrs old, male patient diagnosed of having Type 2 DM for the past 20yrs with poor metabolic control came with complaints of passing foamy urine. Blood analysis revealed glucose 200mg/dl, urea 52mg/dl, creatinine 1.2mg/dl, uric acid 5.3mg/dl. The physician suspected this condition as a complication of long-standing diabetes mellitus. Which of the following statement is correct regarding this condition?

A. Urine dipsticks can easily detect microalbuminuria
B. 24 hr urine albumin is more sensitive than spot sample urine albumin: urine creatinine
C. Microalbuminuria correlates with nocturnal spike in Systolic blood pressure
D. Most common cause of death in type 2 diabetes mellitus

Answer: C

Solution

The above history & findings lead to the diagnosis of Diabetic Nephropathy

- Sensitive radioimmunoassay methods measure small amounts of urinary albumin in contrast to less sensitive dipstick strips. Hence choice A is wrong.
- **24 hour urine albumin values are affected by exercise, dietary protein and sustained erect posture**. This problem is circumvented with spot sample urine albumin to urine creatinine values. Hence choice B is wrong.
- **Most common cause of death in type 2 diabetes mellitus is myocardial infarction**. Hence choice D is wrong.

Reference: Page 1239: CMDT 2017/ CMDT 2019 page 1248

Q. A 66yrs old, female with a known case of Type 2 DM came with complaints of painful, ulcerated lesion on her right leg as shown in the image below. What is the diagnosis?

A. Necrobiosis lipidoica diabeticorum
B. Acanthosis nigricans
C. Lupus pernio
D. Pretibial myxedema

Answer: A

Solution

- Option A- lesions present on the shins with erythema, some areas having yellowish appearance. These could be tender to touch d/t secondary infections and mostly seen with patients having DM-II
- Option B- characterized by hyperpigmentation mostly seen at the back of the neck of the patients.

- Option C- Manifestation seen with sarcoidosis, involves only the face and nose of the patient. Erythema nodusum seen on extremities in sarcoidosis patients.
- Option D- Lymphedema only presents with swelling i.e non pitting edema but no ulceration or erythema is seen.

Reference: Harrison's principle of internal medicine 18th edition

42 DIABETIC KETOACIDOSIS & HYPERGLYCEMIC HYPEROSMOLAR COMA

DIABETIC KETOACIDOSIS

- Complication more Common in type I DM > type II DM
- Basic Trigger is Insulinopenia (due to which sugar cannot enter muscle)

↓

Intracellular starvation

↓

Since cell Cannot utilize glucose due to insulin deficiency

↓

Counter regulatory hormones (GH, Cortisol, Glucagon, Catecholamines)

↓

They Increase blood sugar level

Oxidation of fat → FFA will generate ketones (Acidic)

↓

Acidic pH will damage BBB

↓

Metabolic Encephalopathy

- ↑↑ Sugar levels in blood → Osmotic dieresis → dehydration
- Mortality Rate depend upon the age group of the patient
  - 40 years = 20% Mortality
  - < 40 years = 5% Mortality

TRIGGER

- I - Insulin Pump Malfunction
  - Poor Compliance
- I - Infection - (energy requirement of body increase)
  - UTI
  - Pneumonia
- I - Infarction
  - MI
  - Stroke
- Surgery
- Drug abuse

How to remember

- 3'I'

- Triggers leads to
  - Counter hormone increase
  - Blood sugar increase
  - Ketolysis decrease

Pathogenesis

- Infection, Surgery, Myocardial infarction, Cerebro Vascular accident

↓

Energy requirement is more (sugar and insulin required)

↓

In DKA- blood sugar increases- counter hormone more- ketolysis - more ketone)

↓

Deterioration of patient

- As Blood sugar increases, Osmolality of blood increases

↓

leading to fluid shift across brain causing hyperosmolality

↓

leading to CNS MANIFESTATION - stupor and coma.

Clinical features

00:07:57

- Nausea and severe vomiting
  - ketonemia which trigger vomiting center- CTZ
- Abdominal pain
- Osmolality increase= 310-320mosm
  - Stupor
  - Coma
- Polyuria
  - Due to osmotic dieresis
  - ↑ Thirst
- Encephalopathy

Examination

- Fruity odor breath
- Tachycardia
- Orthostatic hypotension
- Dry Oral Mucosa
- Urine Output initially increases

↓

As dehydration occur Urine Output ↓

↓

kidney function deranged)

- RR increases (Kussmual breathing-- acidotic hyperventilation-- washout of Co2)

$$2H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow H_2O + CO_2$$

(↑) (↑) (↑)

- H_2Co_3 reduced less than 15meq (as they are consumed more)

↓

More Co_2 formed - trigger Respiratory center

↓

Leading to washout of Co_2 - Leading to more proton consumption

↓

Proton is responsible for damaging BBB, Encephalopathy etc.

↓

Kussmual Breathing is a compensatory Mechanism (Fruity breath)

↓

Fruity breath is due to acetone (Ketone bodies are -acetone, acetoacetate, and beta hydroxybutyrate)

Investigations 00:14:31

- Urine Ketostix
  - Measures Acetoacetate

Important Information

- Level of acetoacetate is falsely normal in DKA
  - Acetoacetate breakdown into acetone
- urine ketostix is not best the way to diagnose DKA

- Plasma beta hydroxybutyrate
  - Increases
  - Ideal way to diagnose DKA
- Blood Glucose → 350 mg% (substantially high)
- Electrolyte Disturbances
  - Sodium
    - → Decrease
    - → High Glucose draws water
    - → H2O will dilute Na value
    - → Hypertonic Hyponatremia

Important Information

- $\text{Glucose} \propto \frac{1}{\text{Sodium}}$
- $\text{pH} \propto \frac{1}{\text{Potassium}}$

  - Potassium
    - → Elevated
    - → Acidosis causes k+ efflux (out from cell) -leading to compartment shift)
    - → Total body potassium maybe less but elevated levels in blood
  - Phosphate
    - → Increase/ Normal in blood

Important Information

Causes of Hypokalemia in DKA:

- Vomiting
- Osmotic diuresis
- On treatment of DKA with insulin
  - Insulin causes influx of K^+ in cells
  - This can lead to dangerous hypokalemia
  - While treating patience with DKA gradual correction of K^+ is done as hypokalemia can lead to Torsade's de pointes

    - → Total body phosphate decreases
- ABG Analysis / VBG analysis
  - pH ↓
  - Pco2 ↓
  - HCO_3^- ↓
  - Thus, above condition is METABOLIC ACIDOSIS
  - If CO_2 is low
    - → Hyperventilation
    - → PARTIALLY COMPENSATED METABOLIC ACIDOSIS
  - If CO_2 is normal
    - → UNCOMPENSATED METABOLIC ACIDOSIS

    Anion GAP = Na+ - (Cl- + HCO3-) -- Anion gap is high
- S. Amylase / lipase
  - N / slightly Increases
  - Rule Out Acute Pancreatitis
- Lipid and Triglycerides: Increases
- TLC: Increases
  - if Trigger is infection
- KFT
  - Urea, creatinine is highly increased
  - Due to dehydration
- High anion Gap Metabolic acidosis (HAGMA)-- as positive charges are high

Causes of Death in DKA

- Children
  - Cerebral oedema
- Adults
  - ARDS

- ESRD
- Bowel infarction

Treatment 00:31:40

- Grading of severity of disease

Ph	β Hydroxy	Grade
7.25 - 7.30	3-4	Mild (alert)
7.00 – 7.25	4 – 8	Moderate (drowsy)
< 7	8	Severe (Stupor)

- First line of management
  - Fluid Resuscitation
    - → NS 0.9% (fluid of choice)

Important Information

- **RL is not given because it is rich in potassium and in DKA potassium is already raised--IF raised further will lead to diastole arrest.**

    - → 5L of NS is given (in next 8 hours) 1L/hour in first 2 hours
    - → Decrease after 2 hours
    - → 400 ml NS/hour
    - → On treatment in DKA → fluids given → sugar will be diluted →sodium level rises → when $Na^+ > 150$ meq → 0.45% N/2 is Given
    - → $\text{Glucose} \propto \frac{1}{\text{Sodium}}$
  - Insulin administration
    - → Regular Insulin (can be administer by any route I/M, I/V, S/C) is given
    - → Bolus is given initially 0.1 U/Kg to Prime Insulin receptor
    - → Infusion later on (0.1 U/kg/hr.)
      ↓
      Blood Sugar level falls 10% initially in first Hour
      ↓
      Subsequently 50 mg% decreases/hour
    - → Fast correction can lead to Death-- most common cause of fast correction (in Pediatrics) in treating DKA is Cerebral Edema.
    - → When Blood sugar level falls to 250 mg%, change the fluid to 5% Dextrose.
  - Maintenance of Potassium Levels
    - → On treatment as acidosis resolves hypokalemia occurs
      → Lead to Torsade's de pointes, muscle paralysis → Respiratory Paralysis
      → Monitor K+ levels
      → Give KCL to the patient, add to IV fluids (10-30 mEq/hr. infusion)
  - Acidosis
    - → Give Soda Bicarbonate Only if pH < 7.0 (in severe DKA only)
      ↓
      If excess of HCO3 given to person - HCO3 will trigger Metabolic alkalosis (Tetany, Laryngospasm)
      ↓
      K+ influx into cell- hypokalemia - can lead to arrythmia.
  - Infection control
  - Switch over to subcutaneous insulin

HYPERGLYCEMIC HYPEROSMOLAR COMA 00:47:20

- More Common in type II DM/Occult DM
- Mortality Rate = 10 times higher than DKA
- Middle-aged person is more affected.

Clinical Features

- Occult DM
- Insidious onset
- Pre-Renal AKI: Azotemia
- Patient Usually Presents in Unconscious, Unresponsive State
- Excessive drowsiness
- Triggers for hyperglycemic hyperosmolar coma
  - Myocardial Infarction
  - CVA (Cerebrovascular accident)
  - Pneumonia
  - Surgery

Work up

- Blood Sugar level ≥ 600 mg% → Cerebral Edema
- pH = Normal
- HCO_3 = Normal
- pCO_2 = Normal
- Urine Ketostix = Negative
  - Plasma Beta Hydroxy butyrate: Normal/Increased
- AG → Normal/↑ Lactic acidosis
- Plasma osmolality >330 mosm

Treatment 00:55:00

- Normal Saline = 10 L/24hr
  - Blood sugar<250 mg% change to 5%Dextrose
- No need of Insulin Bolus
- Insulin Drip Infusion (0.05 U/Kg/hour)
- If potassium deficient add KCL
- PO_4 Replacement

	Lactic Acidosis	DKA	Hyperosmolar Coma
Blood Sugar	300 mg%	350 mg%	800 mg%
pH	7.25	7.25	Normal
HCO_3^-	15	15	Normal
Urine dipstick	- Ve	++	- Ve
β hydroxy butyrate	Normal	↑	Normal/ ↑
Characteristic features	↑S.Lactate values	↑βOH Butyrate levels	Plasma osmolality= 330-380 PCO2=Noramal HCO3 >15meq

CLINICAL QUESTIONS

Q. A 20-year-old male has been admitted to ICU for diabetic ketoacidosis (DKA). His mother gives history that his thirst has increased lately & he goes to loo frequently. He has not been diagnosed with diabetes previously. There is no family history of diabetes. His BMI is 42 kg/m2. Anti-GAD antibodies and anti–islet cell antibodies (ICA) are not detected in this patient. Which of the following statements is true regarding this patient?

A. Due to the young age of onset, type 1 diabetes is suspected.
B. Due to your presentation with diabetic ketoacidosis, type 1 diabetes is suspected
C. Patient has maturity-onset diabetes of the young.
D. Patient has type 2 diabetes mellitus.

Answer: D

Solution

- Features of type 2 diabetes:
  - Usually develops after the age of 30 years
  - Patients are usually obese
  - May not require insulin therapy initially
  - Associated with insulin resistance, hypertension, cardiovascular disease, dyslipidemia, or PCOS.
  - Age should not be the sole basis for determining the type of diabetes present.
  - Age of diagnosis of T2DM is declining as obesity is increasing among children and adolescents.
  - Some individuals with phenotypic T2DM present with diabetic ketoacidosis but lack autoimmune markers.
- Maturity-onset diabetes of the young- diabetes onset at <30 years of age, an autosomal pattern of diabetes inheritance (which this patient lacks), and the lack of nearly complete insulin deficiency.

Reference: Harrison Principle of Internal Medicine 20th edition page 2856

Q. An 11-year-old type 1 diabetes mellitus patient was on CSII. While on holiday with her family she has become disoriented. On admission Na=126mEq/dl, potassium= 4.3mEq/dl, BUN= 100mg/dl, bicarbonate is 10mEq/dl and blood sugar is 600mg%. All are required for management except?

A. ABG
B. Potassium hydrogen phosphate
C. Intravenous potassium
D. 3% saline

Answer: D

Solution

- A patient of type 1 Diabetes mellitus is on Continuous subcutaneous insulin infusion. Due to device malfunction/tubing malfunction the delivery of insulin was halted.
- Since patients of type 1 diabetes are ketosis prone, she has gone into Diabetic ketoacidosis. The low bicarbonate points to acidosis.
- The patient's elevated blood sugar is drawing water into the intravascular compartment and hence volume expansion explains the sodium deficit. However there is no need of hypertonic saline as correction of hyperglycemia by insulin shall suffice in managing sodium values.
- Hypertonic saline is only given in acute onset hyponatremia with neurological features.
- If severe hypophosphatemia can develop (<1mg/dl), phosphate should be replaced at no more than 3-4mmol/h via infusion.
- Potassium replacement should be started in 2nd to 3rd hour as acidosis begins to resolve.

Reference: Harrison's 20th ed 572

43 DISORDERS OF PARATHYROID GLAND

PARATHYROID HORMONE (PTH) 00:00:15

Functions

- Causes Bone resorption: ↑ Ca++
- Acts on PCT kidney → PO4 WASTING → Po4 ↓
- It increases Bone turnover
  - ↑Serum Alkaline Phosphatase
  - Best marker for bone turn over
- Osteoclasts→ Bone destroying action
- Osteoblasts → Bone-forming action → forms serum alkaline phosphatase [sap]
- In bone resorption → osteoclastic activity increases → resulting in secondary increase in osteoblastic activity → which increases SAP

Important Information

- Increased SAP
  - Rickets (in children)
  - Osteomalacia (in adults)
  - Hyperparathyroidism
- Decrease SAP
  - Hypophosphatasia
- Disproportionate ↑↑ in SAP: Paget's Disease
- SAP normal
  - Hypoparathyroidism
  - Multiple Myeloma
  - Osteoporosis

PRIMARY HYPERPARATHYROIDISM 00:06:34

- Leading cause → Parathyroid Adenoma
- Tumor in any of the parathyroid glands → ↑ PTH → BONE RESORPTION ↑ → Ca2+ ↑
- Normal calcium level: 9-11 mg%

Raise of calcium & its manifestations

- 11-12 mg%
  - Nephrocalcinosis
  - Renal Colic
    - → Presents as flank pain going towards umbilicus
    - → A/W multiple hospitalizations d/t recurrent vomiting episodes caused by pylorospasm (because of Reno gastric Reflex)
  - Constipation → d/t spasm of sphincters of gut → may present with abdominal pain
  - Calcinosis cutis (Ca+2 deposited in skin of the patient)
  - Pseudo-hypertension [HTN d/t hardening of blood vessels]
  - Psychosis (Moaning & groaning)
- 12-13 mg%
  - 2° Parkinsonism [d/t calcification of basal ganglia] Bradykinesia / Rigidity
- 13-14 mg%
  - Systolic arrest

Hypercalcemic Crisis:

- Malignancy – Squamous cell Ca of lung d/t PTH, CA Breast
- Parathyroid Adenoma
- Sarcoidosis → Non caseating Granuloma → Synthesis of Vit D3
- Vitamin D3 Intoxication
- Milk Alkali Syndrome → excessive intake of antacids

Steroid responsive Hyperkalemia

- Sarcoidosis
- Vit D_3 Intoxication

WORK-UP 00:16:46

- S. CALCIUM - ↑
- S. PO4 - ↓
- SAP - ↑
- S. PTH ASSAY: ↑↑
- Tumor Localization is done by Sestamibi Scan (Tc 99)
  - Also Used in Chronic Stable Angina

Treatment 00:18:30

- Medical Stabilization of hypercalcemia component of the patient
- Normal saline (dilution effect) : Hydration → 5-6 liters in 24 hours (Furosemide Cause Ca2+ loss in Urine)
- Furosemide Drip (Causes Ca++ loss in urine)
- IV Ibandronate → DOC
- Calcitonin Nasal spray → Antagonist of PTH → sends Ca+2 into bones
- After stabilization get preanesthetic checkup & fitness certificate for surgery
- Treatment of Choice: Surgery → Resection of adenoma
  - 1° PTH

→ Parathyroid adenoma.
→ Treatment of choice is surgery

SECONDARY HYPERPARATHYROIDISM

- M/c cause is Chronic Renal Failure
- Mechanism
  - Chronic Renal Failure → ↓ Vit D_3 (PCT Kidney)
    ↓1α Hydroxylase
    Hypocalcemia
    ↓
    [illegible]
    ↓
    Bone Resorption
    (Bone Pain, Rib, Vertebrae, Clavicle, Fracture)
    ↓
    Azotemic Osteodystrophy / Osteitis Cystica Fibrosis (Brown Tumor)
  - Cinacalcet → Regulate PTH secretions

Work Up

- Ca++ ↓
  - D/t deficiency of Vit D3
- PO_4^{2-} Raised
  - D/t non excretion of PO_4^{2-}
- SAP ↑
  - D/t ↑ PTH
- Vit D3 ↑↓
- RFT – Serum Creatinine ↑, BUN ↑

TERTIARY HYPERPARATHYROIDISM

- D/t untreated 2° PTH / CKD
- Autonomous gland resection

HYPOPARATHYROIDISM

00:36:10

- Incidence of Hypoparathyroidism d/t resection during thyroid surgery has ↓Since nowadays parathyroid implantation is done
- Site: Brachioradialis

Causes

- Autoimmunity → leading cause
- Thyroid Surgery (Inadvertent parathyroid resection)
- DiGeorge Syndrome
  - Defect in chromosome 22
  - Leads to Thymus and Parathyroid hypoplasia
  - Thymus Hypoplasia
    ↓
    CMI
    ↓
    Risk of infections ↑
  - Parathyroid hypoplasia
    ↓
    ↓PTH
    ↓
    $\downarrow Ca^{+2}$
    ↓
    Rickets like illness

ACUTE HYPOCALCEMIA/ TETANY

00:40:52

- Post Thyroid Surgery
- > 72 hours → S. CALCIUM < 7 mg % → Tetany

Manifestations

- Perioral & Periungual Paresthesia
- Trousseau Sign
  - Apply BP Cuff & inflate it
  - Results in Carpopedal Spasm (Obstetrician hands)
- Chvostek Sign
  - Tap on anterior border of Parotid gland
  - Results in contraction of muscles of one side of face → Facial Grimace
- Stridor: Laryngospasm

Work Up

- Tetany is a medical emergency
- ECG
  - QT Prolongation
  - Calcium $\propto \frac{1}{\text{QT Interval}}$
  - Normal QT Interval = 360-440
- S. Calcium ↓
- Ionized calcium ↓
- S. Albumin is normal
- Values of ↓ Ca2+ are observed in 2-3 days
- Early way to Dx tetany = ECG

Management

- IV Calcium gluconate

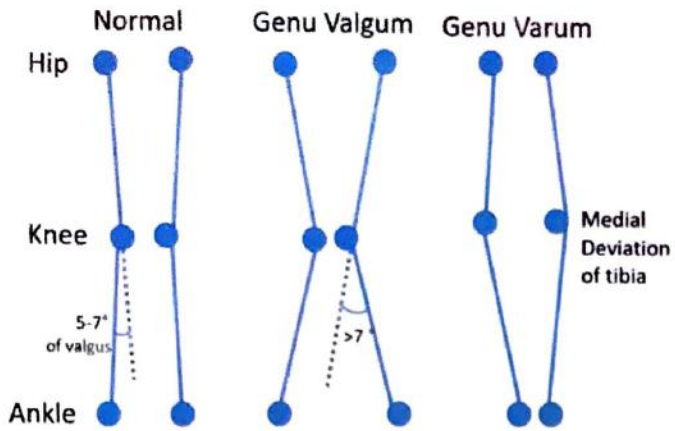
CHRONIC HYPOCALCEMIA

00:51:55

- Rickets like illness

Features

- Craniotabes
- Skull is soft & easily deformable
- Widening of wrist joint
- Disproportionate Short Stature
- Wide open Anterior Fontanelle
- Delayed dentition [no teeth beyond age of 13 months]
- Skeleton abnormalities
  - Knock knees
  - Bow legs



Investigations

- Calcium - ↓
- PO4 - ↑
- SAP – Normal
- S. PTH Assay - ↓↓
- Tc99 Scan / SESTAMIBI Scan - ↓/ absent uptake by gland

Important Information

$$\text{Calcium} \propto \frac{1}{PO_4}$$

Due to Deficiency of PTH

- ↓ Ca^{2+} in blood
- ↑ PO_4^- in blood d/t defective secretion of phosphate

Management

- Calcium & Vit D supplements
- Drug of choice for Surgically induced hypoparathyroidism: Teriparatide Injections

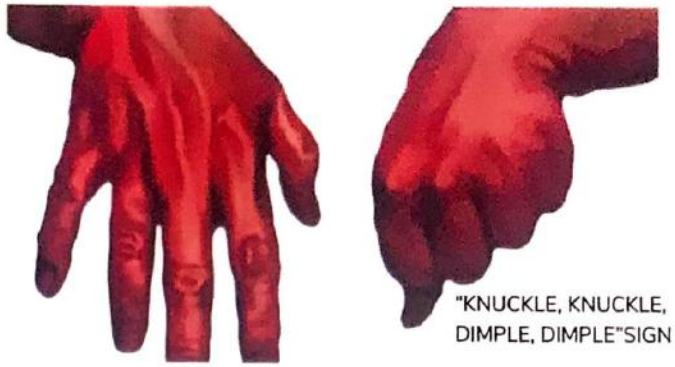
PSEUDO HYPOPARATHYROIDISM

00:56:07

- PTH values: ↑↑
- Receptors: Resistance to PTH, because of defect in GNAS gene

Clinical features

- Similar to Hypoparathyroidism/ Rickets like illness + Archibald sign
- Patient will be having short 4th and 5th metacarpal bones
  - Leading to Knuckle-Knuckle- dimple–dimple sign/ Archibald sign.
- This is also described as Albright Hereditary Osteodystrophy



Investigations

- Serum Calcium: Decreased
- PO_4 Increased
- SAP: Normal
- Pseudo-hypoparathyroidism is differentiated from Hypoparathyroidism based on the levels of PTH
- PTH ↑ in pseudo-hypoparathyroidism and reduced in hypoparathyroidism:

Management

- No definite treatment

PSEUDO PSEUDO HYPOPARATHYROIDISM

01:02:22

Etiology

- D/t Genomic Imprinting from Paternal Side
- Tissue specific difference in action
  - Action of PTH on bones is defective → Have AHO (Clinical features)
  - Action on Kidney → Normal → PO4 normal
- PTH level → Normal

Comparison between HP, PHP & PPHP 01:06:20

Refer Table 43.1

Important Information

Pseudohypoparathyroidism & pseudo pseudohypoparathyroidism are differentiated by phosphate levels

Refer Table 43.2

NUTRITIONAL RICKETS

01:12:21

- Vitamin D3 ↓
  ↓
  Calcium ↓
  ↓
  PTH ↑
  ↓
  PO4 Wasting [urinary loss]
  ↓
  PO_4 ↓
- Therefore, in Rickets there will be ↓ Ca2+ & ↓ PO4-, ↑ SAP

Investigations

- X-Ray distal end of Radius
  - Cupping
  - Splaying
  - Fraying
- Follow Up
  - White line of Calcification is seen on Xray (Sign of Healing Ulcers)

Treatment

- 6 Lakh international units of Vit D
  - Given in single dose I.M / in sachets
- 60,000 IU orally X 10 day

Important Information

- Phosphate is increased in CKD due to low GFR. Though PTH is high, it is not able to kick out phosphate, since filtration of PO4 itself is defective.

Table 43.1

DISEASE	CAUSE	PO4-	PTH	C/F
Hypoparathyroidism	• Autoimmunity	• Increased	• Decreased	• Tetany, Rickets like illness
Pseudohypoparathyroidism	• Receptor resistance	• Increased	• Increased	• Albright hereditary osteodystrophy (AHO) • PO4↑
Pseudo pseudo hypoparathyroidism	• Action on bones - Defective	• NORMAL	• NORMAL	• AHO • PO4 Normal

Table 43.2

	1° PTH ↑	2° PTH ↑	HP	PHP
	Adenoma	Chronic kidney Disease Vit. D3↓	Autoimmune PTH ↓	Defective GNAS Gene PTH ↑
Ca+2	• Increased	• Decreased	• Decreased	• Decreased
PO4	• Decreased	• Increased	• Increased	• Increased
SAP	• Increased	• Increased	• N	• N
Treatment	• Surgical Resection	• Vit. D3 + Ca2+ • Cinacalcet	• DOC in surgically Induced Hypo PTH TERIPARATIDE INJECTIONS	• No Treatment.

CLINICAL QUESTIONS

Q. A 65-year-old lady hospitalized for cervical spondylosis was found to have serum calcium of 12.5mg%. Her haematocrit and KFT is normal with phosphate of 2.3mg/dl. Which is the first investigation to be done in the patient?

A. Serum PTH
B. PTH-rP levels
C. Serum electrophoresis for M spike
D. Vitamin D3 levels

Answer: A

Solution

- **In an Asymptomatic patient with hypercalcemia, the leading cause happens to be hyperparathyroidism.** PTH causes **urinary loss of Phosphate**, which explains low phosphate in the patient. (Normal phosphate=2.5-4.5mEq/dl) PTH assay is the first investigation to be done in this case.
  - **In symptomatic hypercalcemia, cancer is the leading cause.** Hence work up for squamous cell cancer of lung and breast cancer should be done.
- Choice C is done for multiple myeloma. It is ruled out since **phosphate is normal in Multiple myeloma**.
- Choice D is ruled out as hypervitaminosis is a rare cause and there is no history of taking any health supplements. Also both **calcium and phosphate are elevated in hypervitaminosis D**.

Reference: Harrison principle of Internal Medicine 20th Edition page 2928

Q. A 25-year male presented to opd with complains of back pain, shin pain, weakness and fatigue. He also complains of muscle pains. Serology reveals elevated alkaline phosphate, low calcium and low phosphate. Likely diagnosis is -

A. Paget's disease
B. Osteoporosis
C. Primary hyperparathyroidism
D. Vitamin D deficiency

Answer: D

Solution

- Option A - Paget's disease: Normal Ca^{+}, Normal PO_4, SAP ↑↑↑↑
- Option B - Osteoporosis: Normal Ca+, Normal PO4, SAP normal
- Option C - Primary Hyperparathyroidism ↑ Ca^{+}, ↓ PO_4, SAP ↑, Bone turnover↑
- Option D - **Vitamin D deficiency: Calcium ↓, PTH↑, PO_4↓, SAP ↑**
- Note: Ca+ is inversely proportional to PO4

Reference: Harrison's internal medicine ,20th edition, page-2913

44 DISORDERS OF ADRENAL GLAND

ADRENAL GLAND

00:00:13

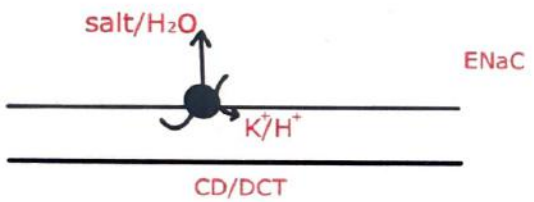
- Functioning depends on CRH (from hypothalamus)
  ↓
  ACTH (from anterior pituitary)
  ↓ acts on
  Adrenal glands

Pyramidal shape

Semilunar shape

LAYERS OF ADRENAL GLAND

1. Zona Glomerulosa
- Produce Aldosterone (under control of RAAS)
- Aldosterone
  - Receptors are in kidney (K/A Epithelial Na+2 Channels)
  - Acts on Collecting duct >>DCT
  - Functions of Aldosterone
    → Absorption OF SALT & WATER
    → Excretion OF POTASSIUM & HYDROGEN
    → Main function Postural adjustment of Blood pressure



2. Zona Fasciculata
- Produce cortisol
- Cortisol is a Stress Hormone which regulates Blood Sugar Levels
- Cortisol ↑ → ↑ Blood Sugar → Impaired Glucose tolerance

3. Zona Reticularis
- Releases sex steroids like DHEAS (Di Hydro Epi Androsterone Sulfate)
- Responsible for 20 sexual characteristics

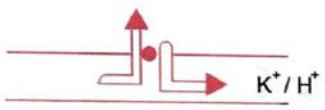
4. Medulla
- Synthesize & release catecholamines (Epinephrine, Norepinephrine, Dopamine)
- Major catecholamine– Epinephrine (60%)
- Most of pheochromocytomas → produce Norepinephrine → have Shortest ½ life
- A.A involved in synthesis of catecholamines → Tyrosine
- Epinephrine & Norepinephrine → after metabolism, forms→ Vanillylmandelic Acid
- Dopamine → after metabolism forms → Homovanillic Acid
- Function of Epinephrine/Norepinephrine → Fight or Flight Phenomenon

1° HYPERALDOSTERONISM

00:10:32

- Excess production of aldosterone
- Causes
  - B/L Adrenal Hyperplasia → Most common cause (90%)
  - Adrenal Adenoma (tumor in the 1st layer of cortex) → CONN'S Syndrome
  - Cirrhosis, Nephrotic, CHF, CP, RCM

↓
Massive Ascites
↓
Decrease circulating Fluid volume
↓
Decrease in GFR
↓
RAAS ⊕ → ↑ Aldosterone (2° hyperaldosteronism)



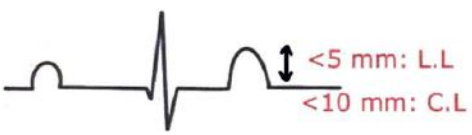
- ↑Aldosterone → ⊕ E NaC
  - Increase sodium & water absorption resulting in expansion of plasma volume
  - Contralateral urinary loss of K+ & Na+

Clinical Features 00:15:34

- Headaches – due to Hypertensions
- Urinary loss of potassium → Hypokalemia → resulting in Muscle Cramps (weakness)
- Metabolic Alkalosis → d/t loss of hydrogen ions
- Gain of H2O
  ↓
  Expansion of plasma volume
  ↓
  ↑Venous Return
  ↓
  ANF release (From Right Atrium)
  ↓
  Natriuresis (Loss of H_2O)
  - Gain of H2O = Loss of H_2O → So, no Pedal edema
  - Polyuria (d/t ANF)
  - Polydipsia
- Cognitive dysfunction, ↑ Chances of type 1 DM

Investigations

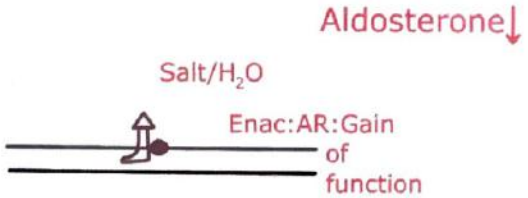
- Na+ levels increase
  - Serum sodium values may be normal d/t simultaneous excess of water. But total body salt is more
- K+ levels decreases
  - In the presence of Aldosterone → Na α 1/K
- ECG
  - Normal T-wave → < 5mm in limb leads
  - < 10mm in chest leads
  - In these pts → T-Wave smaller / absent / Inverted T-Wave
  - ST-depression can be seen



- CT Abdomen
  - Shows B/L Adrenal Hyperplasia or Tumor
- Plasma Renin Plasma Aldosterone Ratio
  - Best screening test
  - Autonomous tumor
  ↓
  ↑Aldosterone
  ↓
  (-) RAAS
  ↓
  Decrease Renin levels
  ↓
  Renin-Aldosterone ratio decreases
  ↓
  Results in Low renin HTN
- Saline infusion test (best diagnostic yield) / Salt loading test (IOC)

Treatment 00:32:02

- CONN'S Syndrome
  - Initially give Spironolactone for 4-6 weeks
    → K+ sparing diuretic so manages both hypertension & hypokalemia
  - Then get PAC fitness → do Surgery U/L Laparoscopic Adrenalectomy → definitive treatment.
  - D/D of CONN'S → LIDDLE Syndrome
- B/L Adrenal hyperplasia
  - DOC: life-long treatment with spironolactone
  - S/E: Gynecomastia in males
  - So, Alternate drug: Eplerenone for males



LIDDLE SYNDROME

00:37:14

- Autosomal dominant
- Epithelial Na-Channel defect → exhibits gain of function mutation.
- Causes HTN
  ↓
  (-) RAAS system
  ↓
  Renin decreases
  ↓
  Aldosterone decreases

Important Information

- In LIDDLE Syndrome Aldosterone levels are decreased due to feedback mechanism but hypertension occurs d/t excess salt & water in the body secondary to excessive activity of epithelial sodium channel.

- Imaging: shows normal Adrenal gland
- DOC → Amiloride → Inhibit function of Epithelial Na+2 channel

Important Information

- Conditions with (HTN + Hypokalemic alkalosis + Low Renin)

1. CONN syndrome
   - → Tumor producing excess Aldosterone
   - → Treatment: Spironolactone/Eplerenone -f/b surgery
2. Liddle syndrome
   - → Gain of function of epithelial Na channel → Aldosterone ↓
   - → Treatment: Amiloride

Previous Year's Questions

Q. Hypertension with hypokalemic alkalosis is?
(NEET Jan 2018)

A. Liddle syndrome
B. Gitelman syndrome
C. Barter syndrome
D. Fanconi syndrome

ADDISON'S DISEASE

00:46:00

- 1°: APS, CAH, Adenoma, Autoimmune
- 2°: TB, HIV, Infiltration, Drug Induced

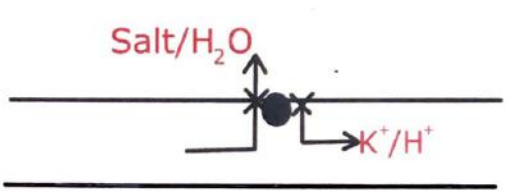
Causes

- Autoimmune destruction (Leading cause)
- Tuberculosis of Adrenals (MCC in India)
  - Acc to Harrison 20th edition HIV/ AIDS is the Leading infectious cause of Addison's disease globally
- Causes ↓ Aldosterone, ↓ Cortisol, ↓ DHEAS

Clinical Features

00:51:46

- Aldosterone ↓ resulting in
  - Salt wasting, craving for salty foods
  - Polyuria (urine output >3L/day)
  - Postural Hypotension
  - K↑
  - Metabolic Acidosis



- Cortisol ↓ → Sugar↓ → Palpitations, Tremors, Diaphoresis, RAGE attack, emotional fragility
- ACTH↑↑ → have Partial Melanocyte Stimulating Hormone like action resulting in
- Hyperpigmentation
  - Palmar/ sole creases
  - Scars / Areola / axilla / cubital fossa
  - Groin area
  - Oral Mucosa
  - Nails

  } Very characteristic for Addison's disease
- ↓DHEAS resulting in → ↓Libido, erectile dysfunction

Investigations

- Na+↓, K+↑
  - Rule out Sampling errors as usage of thin bore needle may result in Factitious Hyperkalemia
- CT Abdomen
  - Shows damaged adrenals
- IOC → ACTH Stimulation test / Cosyntropin test
  - Normally ACTH → Cortisol↑ → Blood Sugar↑
  - In this disease, there is No raise of blood sugar levels

Treatment

- DOC
  - HYDROCORTISONE
- For management of Low BP
  - Fludrocortisone given
  - Give initially, observe response & then go for D.O.C
- For management of Decreased Sugar levels
  - Dexamethasone given

WATERHOUSE-FRIDERICHSEN SYNDROME

- Complication of infections like N. Meningitidis / Pseudomonas
- Leads to
  - Sepsis
  - DIC → Adrenal Hemorrhage → leading to ADDISONIAN CRISIS
- Treatment
  - I.V Hydrocortisone + Antibiotics + fluid replacement

SHEEHAN SYNDROME

- Postpartum hemorrhage → causes Pituitary damage → results in secondary Addison's disease
- Results in ↓ ACTH → ↓ CORTISOL → Hypoglycemia
- B.P is normal as aldosterone is not affected
- No hyperpigmentation as ACTH levels are low
- Treatment: Dexamethasone

HYPOTHALAMUS DAMAGE

- Caused d/t → Trauma / rupture of AV aneurysm / metastasis etc.
- Results in → CRH↓ → ACTH↓ → Cortisol↓

- Aka Tertiary Addison's Disease
- No hyperpigmentation & B.P is normal
- Treatment Dexamethasone (DOC)

Previous Year's Questions

Q. A 56-year-old underwent transsphenoidal hypophysectomy for pituitary tumor. Now she has low ACTH, TSH, FSH, LH. Which hormone will not be given? (AIIMS Nov 2019)
A. Glucocorticoids
B. Mineralocorticoids
C. Levothyroxine
D. Estradiol

Summary of Addison's disease

- 1°: Autoimmune
- 2°: Pituitary damage (SHEEHAN / SIMMONDS ds (non-obstetric cause))
- 3°: Hypothalamus damage
- Crisis: Waterhouse-Friedrichsen Syndrome/ Sudden stoppage of Steroids

	ACTH	Hyperpigmentation	DOC
1°	Increased	Present	Hydrocortisone
2°	Decreased	Absent	Dexamethasone
3°	Decreased	Absent	Dexamethasone
Crisis	(N)	Absent	I.V. Hydrocortisone

CONN Syndrome Vs Addison disease 01:20:51

	CONN Syndrome (↑ Aldosterone)	Addison disease (↓ Aldosterone)
Cause	Tumor	Autoimmune destruction of adrenals
BP	Increased	Decreased
K+	Decreased	Increased
H+	Decreased	Increased
Special	Pedal edema is absent	Hyper Pigmentation
IOC	Saline infusion Test/ salt loading test	ACTH stimulation test or COSYNTROPIN Test
Treatment	SPIRONOLACTONE	HYDROCORTISONE

PHEOCROMOCYTOMA 01:24:00

- Tumor of adrenal medulla
- Aka chromaffinoma
- Chromaffin cells are found in
  - Adrenal medulla
  - Sympathetic ganglia (paravertebrally located)
- Produce NE 80% >> Epi 20% >>> Dopamine
- 10% pheochromocytoma are Malignant
- Benign vs Malignant differentiated by
  - MRI Abdomen is indicated
  - FNAC → Contraindicated → as it may result in damage to normal layers of gland
- 15% tumor → Extra-adrenal
  - Tumors could be deep seated in medulla (85%) → MIBG scan can be done
  - Tumor could be inside sympathetic chain (15%) → ORGAN OF ZUCKER KANDL

Imaging Modality

- MIBG scan
  - Done for Adrenal Pheochromocytoma if MRI abdomen is normal

Important Information

- **Imaging modality of choice in pheochromocytoma is MRI abdomen. in case tumor size is < 1.5 cm, we have to perform the scans mentioned above**

- Pheochromocytoma is associated with other endocrine malignancies
  - MEN-2A- Multiple endocrine Neoplasia (SIPPLE SYNDROME)
    - → **P - Pheochromocytoma - 20 years**
    - → **P - Parathyroid Adenoma - 40 years**
    - → **M - Medullary Ca thyroid 60 years**

How to remember

- PPM

- D/t Ret oncogene, defect in chromosome 10
- 10-15% tumors are B/L

Clinical Features 01:46:15

- Triad
  - PALPITATIONS
  - Headache/Hypertension
  - Diaphoresis

- If we see this triad of symptoms in young patient → Suspect Pheochromocytoma
- Hypertension is episodic / Paroxysmal
  - Catecholamines have a short duration of action and hence BP can be increased for only few hours / minutes initially
  - Later excess of catecholamines will lead to exaggerated vasoconstriction
  - This reduces intravascular volume → leading to postural hypotension in the same patient in later stages of disease.
- ß1: increased HR. palpitations Misdiagnosed
- ß2: (+) Fine tremors in fingers as Anxiety
- Diaphoresis (+) → Sweaty palms / soles Neurosis
- Fasting Blood sugar → 100- 125: 2 hr BS 140-199 mg → Impaired Glucose Tolerance
- Weight loss
- Falsely increased calcium and Increased Hematocrit
- d/t volume contracted state

Investigations 01:58:18

- 24 hours Urinary metanephrine levels → for screening (Best answer)
- 24 hours urinary catecholamines
- IOC - Plasma fractionated Metanephrine levels

Tumor Localization

- MRI Abdomen → Overall best modality
- MIBG scan → if Adrenal pheochromocytoma size < 1.5cm.
- PET dopa scan → Useful for extra -adrenal pheochromocytoma with size < 1.5cm

Treatment 02:01:23

- Benign Pheochromocytoma → medically stabilize the patient & then do surgery (TOC)
- Oral Phenoxybenzamine → Best drug for Pre-operative Hypertension management
- β1 - Palpitations β2 - fine tremors → use propranolol
- Vasoconstriction is max in early morning hours so, for symptomatic management give
  - Alpha- blocker → Oral phenoxybenzamine (controls B.P) → in early morning
  - β- blocker → Oral propranolol (control palpitations & tremors) → post breakfast
  - Alpha- blocker should be always given before – ß blocker (i.e., α+ ß)
  - If we give ß - blockers prior to -blocker, vasodilation is blocked → unopposed alpha receptors stimulation → vasoconstriction → resulting in severe hypertension (may result in CNS Bleed / hypertensive encephalopathy)
  - Thus, (ß + α) is Contraindicated
- Intra-Operative HTN crisis
  - Tumor manipulation causes spikes in levels of catecholamines
  - Treatment: IV NTG / Sodium nitroprusside
  - Surgery is contraindicated in Malignant pheochromocytoma.
  - Chemotherapy and 1-131 loaded MIBG scan can be used in case of metastatic Tumor. Debulking surgery is not a curative surgery.

Previous Year's Questions

Q. Increased levels of 5HIAA are seen in ? (NEET Jan 2018)

A. Carcinoid syndrome
B. Pheochromocytoma
C. Gastrinoma
D. Phenylketonuria

Previous Year's Questions

Q. All of the following syndromes are seen? with obesity except? (FMGE Dec 2017)

A. Prader Willi syndrome
B. Cohen syndrome
C. Laurence moon-Biedl syndrome
D. Carcinoid syndrome

CLINICAL QUESTIONS

A 30yrs old, female presented with complaints of chronic fatigue & weight loss for the past 3 months. On physical examination, her face, palmar creases & gingivae appear to be hyperpigmented. Initial lab tests demonstrated an elevated serum k^+ levels and decreased serum Na^+ levels of 5.5mmol/L & 130mmol/L respectively. Her past medical record is uneventful. The physician suspected this as a case of adrenal insufficiency. Which is the best test for the diagnosis of adrenal insufficiency?

A. CT abdomen
B. Steroid autoantibodies
C. Fludrocortisone challenge test
D. Cosyntropin test

Answer: D

Solution:

- The diagnosis of **Primary Adrenal Insufficiency** is established by **Cosyntropin test**
- Cortisol values of <18µg/dl sampled 30-60 minutes after ACTH stimulation will diagnose the disease.
- This is followed by plasma ACTH values to identify primary versus secondary Addison disease.
- Steroid auto-antibodies are marker for **Autoimmune Adrenalitis**.
- **Fludrocortisone challenge test** is done for **Conn** syndrome.

Reference: Harrison's Principles of Internal Medicine - 20th Edition, Page No:2737

Q. A 27-year-old male has had a hard to control hypertension for 2 years. He is taking clonidine, hydrochlorothiazide, verapamil, and lisinopril. His blood pressure is 170/105 mmHg, pulse 90 beats/min, and respirations 16/min. The cardiopulmonary exam is normal. Pedal pulses are intact and there is no edema or mis-distribution of fat. Laboratories show potassium of 2.7 mEq/L, BUN 20 mg/dL, creatinine 1.2 mg/dL, bicarbonate 33 mg/dL, and fasting glucose 98 mg/dL. What is the most likely diagnosis?

A. Conn syndrome
B. Renal vascular hypertension
C. Cushing syndrome
D. Carcinoid syndrome

Answer: A

Solution

- Presentation: $K^+ \downarrow\downarrow$
- RFT= N
- HCO_3 (22-26) here $\uparrow\uparrow$ so alkalosis present
- Therefore, **Hypokalemic alkalosis with HTN**
- **Option A-** characterized by $\uparrow\uparrow$BP as aldosterone is elevated which also l/t urinary loss of K^+ and hydrogen causing Hypokalemic alkalosis.
- **Option B-** In renal vasculature hypertension there is $\downarrow$RFT
- **Option C-** presents with weight gain in patients, HTN as cortisol can stimulate aldosterone receptors l/t hypokalemic alkalosis.
- **Option D-** carcinoid syndrome doesn't lead to hypertension because metabolites produced may cause bronchospasm and Histamine/Serotonin release which will cause vasodilation not HTN.
- **1ST likely diagnosis= CONN syndrome**

Reference: Harrison's Principles of Internal Medicine - 20thEdition, Page No: 2729

45 CUSHING SYNDROME

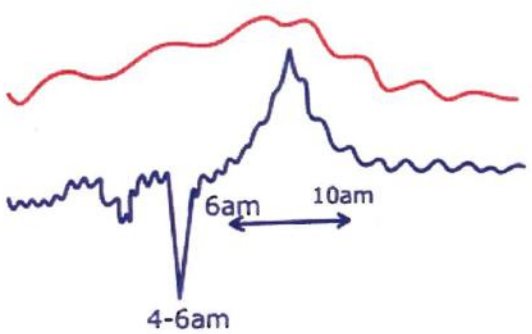
CAUSES

00:00:38

- Iatrogenic steroids
- Exogenous
  - M/c cause
  - Increased Cortisol decrease ACTH by feedback
- Carcinoid tumor
  - ↑ Ectopic ACTH → ↑ Cortisol
- Oat cell CA lung
- Pituitary Adenoma
  - Aka Cushing's disease
  - Endogenous cause
  - ACTH ↑ → Cortisol ↑
- Adrenal adenoma involving zona Fasciculata
  - Cortisol ↑ → ACTH ↓

Important Information

- **Earliest manifestation of Cushing's syndrome is loss of diurnal variation of cortisol production**



Clinical features

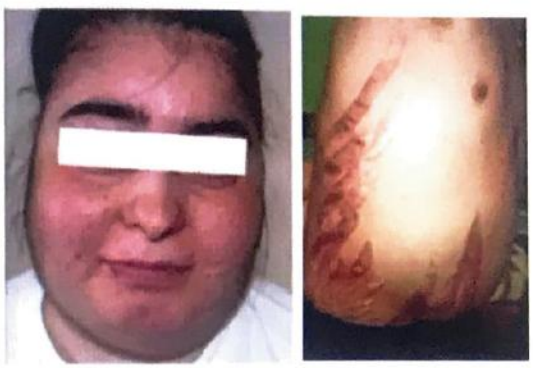
00:16:14

- Moon facies
- Centripetal Obesity
- Proximal myopathy
- Violet/ purple Striae, Thin skin, Purpura
- Buffalo hump

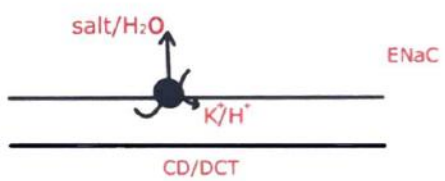
Important Information

- **In Pregnancy pink striae are seen**

- Weight gain / Lemon on sticks description Term for Point 1-5.
- ↑ Peripheral resistance → HTN



- Secondary DM
  - FBS > 126 mg%
  - 2hr > 200 mg%
  - Caused by
    - → Insulin resistance → sugar ↑
    - → Cortisol ↑ → sugar ↑
  - Treatment
    - → Metformin
- Cortisol ↑ →Activates ENaC
  - causes salt & water retention
  - Hypokalemic alkalosis



Important Information

- **Cortisol ↑ → Activates Sex steroid receptors**
  - **Causes Hirsutism, weight gain, oligomenorrhea, infertility**
  - **PCOD >> Cushing Syndrome**

- Hyperpigmentation: (±)
- Ectopic ACTH → (+)
- Exogenous cause → (-)

Work Up 00:29:24

- Screening test
  - 24-hour urinary cortisol ↑↑
  - Spot Salivary cortisol ↑
- Overnight dexamethasone suppression test
- Low dose dexamethasone suppression test (IOC)

Important Information

- **High dose dexamethasone suppression test is used for etiological diagnosis. It Helps to Pinpoint the cause of disease and not disease per se.**

Investigations

- MRI Head [pituitary adenoma]
- HRCT Chest [lung cancer]
- CT Abdomen [Adrenal causes]
- ACTH levels ↑/↓
  - Can be Suppressed in pituitary adenoma
    → Also referred as ACTH dependent cause
  - Not suppressed in ectopic causes

Treatment 00:44:59

- Iatrogenic steroids
  - Taper steroids
  - Start alternate immunosuppressive drugs like Azathioprine
- Oat cell lung cancer
  - Chemotherapy with Cisplatin + Irinotecan
- Pituitary adenoma/Cushing disease
  - Trans-sphenoidal Surgery
- Adrenal adenoma
  - Medical Adrenalectomy done using
    → Oral Ketoconazole (preferred)
    → Aminoglutethimide (IV)
    → Mitotane IV

NELSON SYNDROME

- Caused due to B/L Adrenalectomy performed to treat bilateral adrenal adenoma producing a mass effect.
- During Surgery to prevent Addisonian crisis → IV Hydrocortisone drip can be given
- Post op → Life-long Hydrocortisone supplementation
- Follow up → D/t static levels of steroids, no feed-back inhibition on ACTH → ACTH ↑↑↑ → resulting in Hyperpigmentation ++
  - Only Pulsatile release of cortisol can inhibit ACTH production

Important Information

- **ACTH Dependent Cushing Syndrome: Pituitary Tumor and ectopic Tumors producing ACTH**
- **ACTH Independent Cushing Syndrome: Adrenal adenoma**
- **High dose dexamethasone suppression Test is used to differentiate between these causes.**

HYPERPIGMENTATION 00:55:00

- Seen in
  - **C** – **C**ushing Disease: ACTH↑
  - **A** – 1° **A**ddison Disease: ACTH↑
  - **N** – **N**elson syndrome: ACTH↑

How to remember

- CAN

- Not found in
  - Iatrogenic steroids
  - Sheehan / Simmonds's disease
  - Adrenal adenoma

CLINICAL QUESTIONS

Q. A 28-year-old lady has put on weight (10 kg over a period of 3 years) and has oligomenorrhoea followed by amenorrhoea for 8 months. The blood pressure is 160/100 mm of Hg. Which of the following is the most appropriate investigation?

A. Serum electrolytes
B. Plasma cortisol
C. Plasma testosterone and ultrasound
D. T3, T4 and TSH

Answer: B

Solution

- **Weight gain + Oligomenorrhea → Cushing syndrome.**
- Cortisol inhibits gonadotropin release that explains the amenorrhea.
- **Hypertension in these patients is secondary to increased cortisol that has some mineralocorticoid activity also.**
- Excess glucocorticoids also interfere with central regulatory systems, leading to suppression of gonadotropins with subsequent hypogonadism and amenorrhea, and **suppression of the hypothalamic pituitary-thyroid axis, resulting in decreased TSH (thyroid-stimulating hormone) secretion.**
- Weight gain + menorrhagia + **isolated diastolic hypertension** in the question would have prompted **thyroid dysfunction** as the first answer.

Reference: Harrison's Principles of Internal Medicine - 20th Edition, Page No:2725

4c DISEASES OF THYROID

THYROID STORM

00:00:21

Causes

- Preoperative illness
- Intraoperative complication of thyroid surgery
- Radio ablation iodine

Mechanism

- Patient operated without adequate pre-operative preparation
  - Massive release of thyroid hormones into the circulation occurs
  - Resulting in Thyroid storm
- Prevention
  - Give saturated solution of Potassium Iodide/ Lugol Iodine pre-operatively
  - SSKI x 4 weeks → Causes shrinking of gland → I2 TRAPPING ↓ → T4 ↓ → T3 ↓
  - Occurs by Wolf Chaikoff effect → Downgrade process of iodine trapping which decreases T3 & T4

Clinical features

- This massive release of thyroid hormones into circulation
- T4 ↑, T3 ↑ → persistent Sympathetic stimulation
- Fever
  - Body Temp ↑↑ > 41° C [d/t calorigenesis] → results in further increase in H.R → leading to heart failure [for every 1° raise in temp, H.R increases by 10 beats/min]

1. CNS
   → Delirium
   → Seizure
   → Coma
2. CVS : Arrythmias
   → HR ↑↑
   - High out CCF
   - Pulmonary edema

   → B.P starts falling + SpO2 ↓
3. GI
   → Nausea
   → Vomiting
   → Diarrhoea
4. Liver
   → Jaundice

- Atleast 3 should be present of above 4 with thyrotoxicosis and fever for diagnosis of thyroid storm

Treatment

- Switch off Intra Vascular Fluids / Blood Transfusions in case of pulmonary edema
- DOC: Propylthiouracil
  - By NG Tube
  - Can be given by rectal route
  - Inhibits conversion of T4 to T3
  - If not available methimazole can be given
- ICE packs / Soda Bicarbonate
  - Treat metabolic acidosis
- IV propranolol
  - Prevents further increase of Heart Rate
- Hydrocortisone
- Cholestyramine
  - Sequestration of thyroid hormones
- Saturated solution of potassium iodide
  - Prevent further formation of thyroid hormone

Important Information

- **Summary Of Thyroid Storm**
  - **Reason → Inadequate preparation of patient. [i.e., Lugol's iodine was not given]**
  - **Cause of death → CCF (congestive Cardiac Failure)**
  - **Prevent Death → by giving S.S.K.I which acts by → by Wolf chaikoff effect and decrease production of Thyroid hormones.**
  - **Treatment - PTU → inhibit T4 → T3 conversion**

Wolf Chaikoff effect

- Downgrade process of iodine trapping which decreases T3 & T4

JOD BASEDOW EFFECT

00:21:17

- Opposite of wolf chaikoff effect
- Seen with intake of iodized salt for long duration in iodine Replete population
- Has upgrade of I_2 Trapping leading to increased production of T4/T3

THYROTOXICOSIS FACTITIA

- It is Thyrotoxicosis occurring d/t any other cause other than high salt intake.

- Examples
  - Seen in patient consuming poor quality beef containing thyroid gland of animal.
  - Consumption of Avurvedic / Homeopathic medicine containing Levothyroxine. Lot of these are marketed as weight loss medication

HYPERTHYROIDISM

00:24:37

- Hyperthyroidism: Function of gland
- Thyrotoxicosis: Excess T4 levels

Important Information

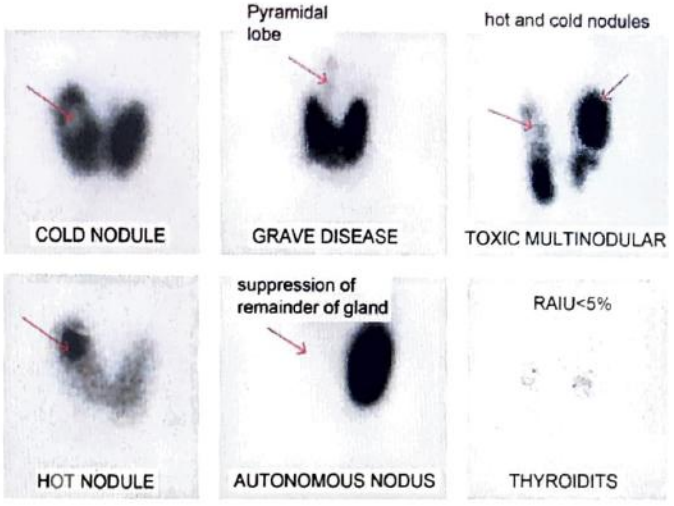
- Thyrotoxicosis without Hyperthyroidism
  - Subacute thyroiditis
  - Silent thyroiditis
  - Thyrotoxicosis factitial
  - Radiation / Amiodarone / Infarction of Large tumor

Causes

- Primary Hyperthyroidism
  - MCC of PRIMARY HYPERTHYROIDISM → GRAVE'S DISEASE
  - Grave's disease → have L.A.T.S- Ab (long acting Thyroid stimulating Antibody)/TSI/TBII (Thyrotropin binding inhibitory immunoglobulin assay) → identical to TSH → T4 ↑ T3 ↑; TSH ↓↓
- Secondary Hyperthyroidism
  - D/t Pituitary adenoma → TSH ↑ → T4 ↑ T3 ↑
- Toxic Nodular Goitre
  - Frequent in Areas of Iodine deficiency
  - Diagnosed by Thyroid scan using I-123 / I-132 [T1/2= 2.3hrs].
  - I-131 is for ablation
  - Warm nodule: Normal Uptake
  - Cold nodule: Suggestive of Malignancy
  - Hot nodule: over active tissue
- Jod Basedow Effect
- Thyrotoxicosis Factitia
- Struma Ovari
  - Ovarian tumor synthesizing hormones
- Gestational Trophoblastic Neoplasia (HCG α TSH. Thus, HCG can bind to TSH receptors & stimulate them)

Important Information

- On Thyroid scan
  - Warm nodule
    - → Normal uptake
  - Hot nodule
    - → Increased uptake
  - Cold nodule
    - → Decreased uptake
    - → Suggestive of malignancy



CLINICAL FEATURES

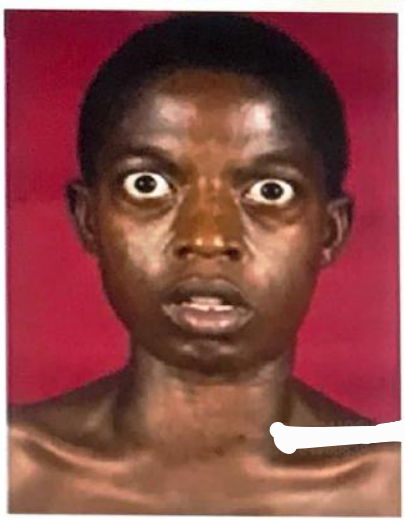
00:46:00

- Sympathomimetic +
  - Palpitations → β1 (+)
  - Fine Tremors → β2 (+)
  - Sweaty Palms/soles
  - HTN → α1 (+)
- BMR ↑ → resulting in weight loss
- Calorigenesis ↑ → causes heat intolerance
- In female
  - Oligomenorrhea
  - Infertility
- Proptosis [d/t retrobulbar fat deposition & swelling of EOM] → Blinking action is inadequate → results in drying of cornea → Exposure Keratitis → causes grittiness / sand like sensation in eyes

Examination

- Sleeping Pulse Rate ↑ → Most reliable sign to diagnose thyrotoxicosis
- Resting Tachycardia
- Fine Tremors
- CVS → loud S1, MEANS Lerman Scratch [d/t hyperdynamic circulation]
- Proptosis, Stare sign, Stell wag sign, Von Graef sign (described Later)

- Pretibial Myxedema
  - Present on shins
  - Myxedema → seen in both hyper & hypothyroidism
  - Non pitting variety as it is lymphoedema
- Pemberton Sign: On Raising arms, there will be Facial congestion d/t compression of SVC by retrosternal goiter.



GRAVE'S OPHTHALMOPATHY

- 0 = **N**o signs or symptoms
- 1 = **O**nly signs (lid retraction or lag), no symptoms
- 2 = **S**oft tissue involvement (periorbital edema)
- 3 = **P**roptosis (>22 mm)
- 4 = **E**xtraocular muscle involvement (diplopia)
- 5 = **C**orneal involvement
- 6 = **S**ight loss

Lid retraction
  - D/t contraction of Muller's muscle [sympathetic overactivity
- Lid-lag sign/ stare sign
  - Earliest feature
  - Inability of upper eyelid to follow the object from top to bottom
  - Inadequate blinking
  - Exposure keratitis
- Periorbital oedema
- Unilateral/ Bilateral / Asymmetrical proptosis is seen
- "Inferior Rectus" is most common extra ocular muscle involved
  - Diplopia
- Retrobulbar neuritis
  - D/t pressure on optic nerve
  - Cause of blindness

Work-UP
- Thyroid Function Tests → Total T4 & T3 ↑, Free T4 & T3 ↑
- TSH
  - If low
    - → Primary hyperthyroidism [MC Grave's]
    - → Next investigation will be TBII/ TSI
  - If increased/ normal
    - → Pituitary adenoma
    - → Next investigation will be MRI Head
- Thyroid scan
- USG / CT of Neck (To Rule out Retrosternal extension)
- USG Abdomen [especially female patient to rule out ovarian/ uterine tumor]
  - If high → Secondary hyperthyroidism → pituitary adenoma → Dx MRI head.

Treatment of Grave's Disease
- Spontaneous relapse/ remission
- Anti-Thyroid Drugs
  - PTU (Propylthiouracil)
    - → Safe in pregnancy [T1] / breastfeeding → as it has high protein binding
    - → Black box warning: Hepatotoxic on prolonged use
  - Carbimazole/ Methimazole
    - → Safe in 2nd Trimester
    - → DOC
    - → Causes Aplasia cutis, Choanal atresia
  - Total Thyroidectomy in case of Poor response to medical Therapy
  - Propranolol → for symptomatic management

Important Information

- **Dose limiting S/E of Anti-Thyroid drugs is agranulocytosis**

- Ablation of thyroid gland using Radioiodine 1-131
  - Beta rays with t½ = 8 days
  - TOC
  - Contraindications
    - → Pregnancy
    - → Breast feeding

Treatment of 2° HYPERTHYROIDISM

- D/t pituitary adenoma
- Treatment: Trans-sphenoidal Sx

- SUMMARY OF MANAGEMENT OF GRAVE'S
  - Treatment of choice: Radio-iodine
  - Surgery of Choice: Total Thyroidectomy
  - Drug of choice: Carbimazole
  - Drug of Choice in T1: Propylthiouracil

HYPOTHYROIDISM

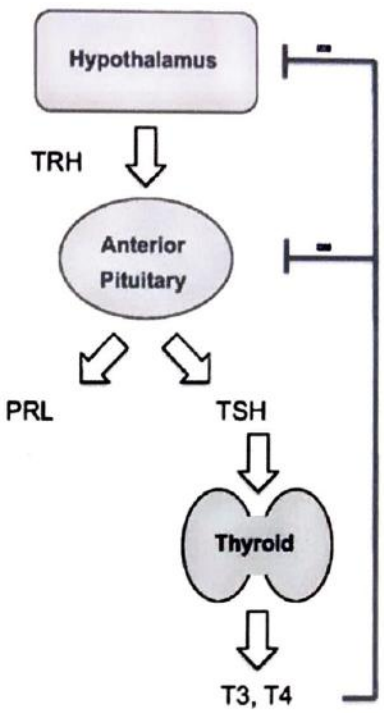
01:21:00

Primary	Secondary
• Thyroid Gland is involved	• Pituitary Gland is involved
• T4 ↓ T3 ↓, TSH ↑	• T4 ↓, T3 ↓, TSH ↓
• Causes ○ INDIA MCC →Hashimoto thyroiditis ○ GLOBALLY→ Endemic goiter ○ ENDEMIC CRETINISM → Baby with hypothyroidism since birth ○ Food Goitrogen→ Cabbage, Turnip ○ Drug induced hypothyroidism (Amiodarone)	• Causes ○ Sheehan syndrome → PPH L/t pituitary infarction ○ Non obstetric cause of pituitary damage called as Simmond's disease→ Occurs due toAVM Rupture, HTN, Trauma and [illegible] anemia ○ Cranial Radiation / Sarcoidosis ○ Empty sella syndrome / CSF leak syndrome

CLINICAL FEATURES

01:28:50

- BMR ↓
  - Progressive weight gain
  - ↓ Calorigenesis → Cold Intolerance
- Myxoedema
  - Involve scalp - Cause alopecia
  - Involve face - Puffy face
  - Vocal cords - Hoarseness voice
  - Body –Thick Coarse skin → Leather like skin → scleroderma
  - Gut - Constipation
- Menorrhagia & infertility
- Headache
- Myxedema Coma
- Hashimoto encephalopathy
  - Myoclonus
  - EEG: Slow waves
  - Steroids responsive
- Galactorrhoea
  - TRH stimulate Ant pituitary to produce TSH & Prolactin
  - In Endemic goiter patient → T3 & T4 are less → by feedback TRH Increases resulting in increase of both TSH & PROLACTIN → causes Galactorrhea
  - Never seen in secondary hypothyroidism [as pituitary is damaged]. Eg: Sheehan syndrome which leads to lactation failure



Examination

- Bradycardia
- Pale yellow skin → occurs due to β Carotenemia. sclera is normal [jaundice sclera becomes yellow]
  - Conversion of β-carotene to Vit A in liver require T3 & T4
  - Thus, if thyroid levels decrease, β-carotene accumulates resulting in yellow skin.
- Isolated diastolic Hypertension
  - BP=120/90mmHg
  - Causes of Isolated diastolic Hypertension
    - → Essential hypertension
    - → Hypothyroidism
    - → CONN syndrome
    - → Cushing syndrome
- Coarse / Dry Skin
- Hung up ankle jerk
  - Most reliable sign

- It occurs due to myxedema to achilles Tendon leading to delayed Relaxation.

Important Information

- **Hung up refex → Chorea**
- **Pendular knee jerk → Cerebellum damage**

WORK-UP 01:43:34

- TFT
  - Total T4, T3- ↓
  - Free T4, T3- ↓
  - TSH
- Anti T.P.O and Anti TG antibody titre
- MRI Head if TSH is low
- USG/ MRI neck
- ECG: Low voltage ECG/ Electrical Alternans
  - Serous cavity effusions
  - Lymphedema/ myxoedema: Massive pericardial effusion
  - Gradually over long duration
  - Water bottle heart/ Money bag appearance

Important Information

- **Massive pericardial effusion developed suddenly causes cardiac tamponade and Pulsus Paradoxus is seen**

- Lipids and cholesterol
  - Increases
  - Accelerated atherosclerosis
- CBC
  - MCV ↑

Primary hypothyroidism	Secondary hypothyroidism
• TSH is high	• TSH is low
• Defect is in thyroid gland	• Pituitary defect [obstetric/ non-obstetric]
• Next best investigation	• MRI head
• Anti-TPO [thyroid peroxidase] Ab	

Treatment

- Levothyroxine: 25 µg / 50 µg / 88 µg / 100 µg
- If dose is >100µg, it increases O2 consumption → resulting in Angina. so, a very careful Titration of dose increments should be done.

Complications of hypothyroidism

- Myxedema Coma 01:54:11
  - Example of case history of myxoedema coma
    - → Elderly hypothyroidism patient not taking thyroid medications, non-compliant, suffers from UTI/Pneumonia/MI/STROKE. She is found Unconscious, unresponsive, hypothermia [Core temp. ↓]- (Measured at lower esophagus) ECG shows Osbourne wave
    - → The Trigger is an intercurrent illness such as UTI, MI, Pneumonia, CHF, Sedatives etc.
  - Treatment
    - → IV Levo-thyronine [Active T3 derivative]
    - → Levothyroxine by NG Tube (DOC)
    - → IV Hydrocortisone
    - → Space blanket [Active rewarming]
    - → Myxedema madness → Development of Psychosis
    - → Myxedema heart / Serous cavity effusion: Patient. have massive pericardial effusion / Pleural effusion→ it is ultra-slow process → So, Pulsus paradoxus is Absent.
- Macrocytic anemia

Important Information

- **Causes of macrocytic anemia**
  - **MYXEDEMA COMA**
  - **CLD (chronic liver disease)**
  - **Aplastic anemia**
  - **Hypothyroidism**
  - **B12 and Folic acid deficiency**

- Accelerated Atherosclerosis: give statins like Atorvastatin (based on LDL levels)

Important Information

- **Conditions that lead to Accelerated Atherosclerosis**
  - **SLE**
  - **Syndrome X**
  - **CRF (Chronic Renal Failure)**
  - **DM**
  - **Hypothyroidism**
  - **Nephrotic syndrome**

- Subclinical hypothyroidism
  - T4 normal
  - T3 Normal
  - TSH ↑
  - Asymptomatic
  - Treatment
    → Levothyroxine only if TSH > 10

SICK EUTHYROID SYNDROME

⏱ 02:00:37

- Impaired conversion of T4 to T3
- Triggers
  - Illness
  - Sepsis
  - IL-6
- Work up
  - TSH↓
  - TSH Ⓝ / ↑: Recovery
  - T_4: Normal and T_3: Decreased

CLINICAL QUESTIONS

Q. A 40 year old lady presents to OPD with weight loss and palpitations is having heart rate of 110/min, BP=130/70mmHg, bilateral proptosis and warm moist skin. Investigations show undetectable TSH and normal free T4. What is the next best step in diagnosis?

A. RAIU scan
B. TPO antibody screen
C. Thyroid stimulating antibody screen
D. Free T3 levels

Answer: D

Solution

- The clinical diagnosis is **Grave's disease.**
- Since TSH is undetectable it further highlights this diagnosis.
- Since the question says next best step, we first have **to demonstrate diagnosis of thyrotoxicosis with values of Free T3.** The algorithm shown below highlights the importance of measurement of free T3 levels.
- Measurement of thyroid stimulating antibodies is not routinely performed to diagnose Grave's disease.

Reference: Harrison 19th edition,Page No. 2295

A 38yrs old, female diagnosed with Grave's disease is found to have transient inhibition of thyroid iodide following excessive iodide. Which of the following auto-regulatory phenomenon best describes this condition?

A. Jod-Basedow phenomenon
B. Wolff-chaikoff effect
C. Thyrotoxicosis factitia
D. De Quervain's thyroiditis

Answer: B

Solution

- **Excess iodide transiently inhibits thyroid iodide organification which decreases T_3 and T_4 synthesis, a phenomenon known as the Wolff-Chaikoff effect.**
- **Jod-Basedow phenomenon :** Thyroid hormone synthesis becomes excessive as a result of increased iodine exposure (opposite to Wolff Chaikoff effect).
- Iodine deficiency increases thyroid blood flow and upregulates the iodine trapping, stimulating more efficient iodine uptake.

- **Ingestion of excess thyroid hormone or thyroid tissue is known as Thyrotoxicosis factitia.** In the first and second statements of this explanation iodine intake was affected but in Thyrotoxicosis factitia is due to the ingestion of hormone.

Reference: Harrison 20th p 2710

47 DISEASES OF ANTERIOR PITUITARY

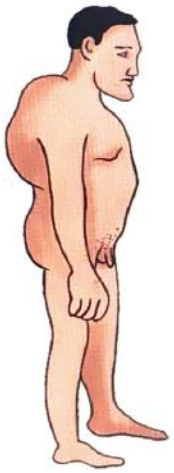
ACROMEGALY

00:00:12

- Occurs d/t excess GH after puberty
- Somatotrophs are abundant cells in pituitary
- Somatotroph adenoma is MCC of developing Acromegaly
- Mammosomatotroph adenoma
  - GH & Prolactin increases
  - C/F also includes galactorrhea
- Leading cause of extra pituitary source of excess GH++ → Pancreatic adenoma (islet cells tumor)
- Leading cause of excessive GHRH production → Carcinoid Tumor

Important Information

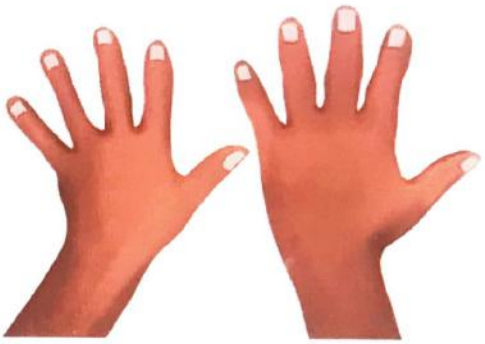
- Hypothalamic Hamartoma presents with
  - Gelastic seizures → patient have uncontrolled laughing
  - Precocious puberty
  - Short stature
  - SIADH



CLINICAL FEATURES

00:08:02

- Acral Enlargement
  - Increase in size of Hand/Feet
  - Increase in Finger thickness leading to spade like hands
  - Lower Jaw Prominence called as Prognathism

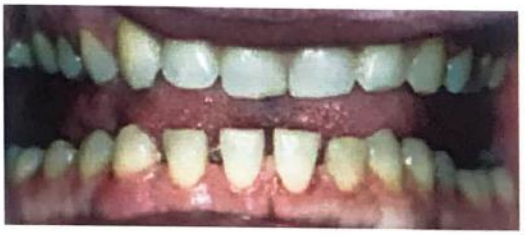


- Frontal Bossing
- Heel pad thickness > 25 mm
  - Used for monitoring growth in these patients
  - Also seen in
    - → **M** - Myxedema
    - → **A** - Acromegaly
    - → **P** - Phenytoin
    - → **C** - Callus
    - → **O** - Obesity
    - → **P** - Peripheral oedema

How to remember

- MADCOP

- Coarse Facies/ deep Hollow voice
- Hyperhidrosis (excessive sweating)



- sebum ↑ (increase incidence of acne)
- Acanthosis nigricans

Important Information

- ACANTHOSIS NIGRICANS can be seen in
  - DM
  - PCOD
  - Ca Pancreas
  - Ca Stomach
  - Acromegaly

- skin tags
- Premature Osteoarthritis → in bilateral knees → d/t weight gain
- Weakness & fatigue
- ↑ Peripheral resistance → resulting in HTN
  - Explains daily headaches
- Increased GH results in Impaired Glucose Tolerance
  - FBS = 100-125 mg%
  - 2hrs = 140-199 mg%
- Obstructive sleep Apnea d/t growth of tongue
- Colonic polyps
- Galactorrhea
- Visual deficit [Bitemporal Hemianopia]

Important Information

- Causes of IGT
  - Glucagonoma
  - Pheochromocytoma
  - Cushing Syndrome
  - Acromegaly
- Causes of Galactorrhea
  - Prolactinoma
  - CKD
  - Drug – Antipsychotics
  - I° Hypothyroidism
  - Agromegaly

Previous Year's Questions

Q MC characteristics physical finding of prolactinoma after galactorrhea is?
(FMGE June 2019)

A. Bitemporal hemianopia
B. Anovulatory cysts
C. Amenorrhea
D. Infertility

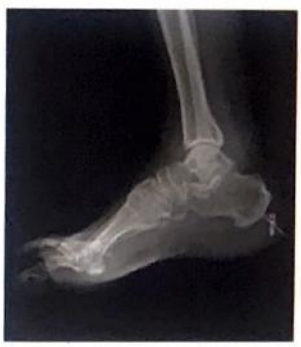
\GIGANTISM

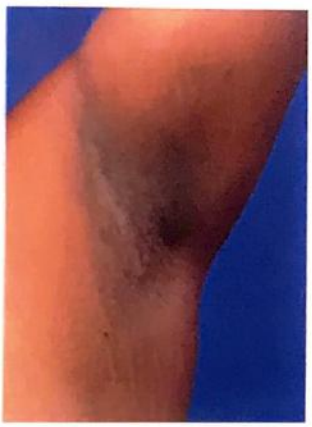
- Occurs d/t increase in GH before puberty [rare condition]
- Height of the patient increases → used to monitor growth
- Visceromegaly is seen
- Cause of death: High output cardiac failure

WORKUP FOR ACROMEGALY

00:20:33

- Screening: IGF-1 level ↑ [Insulin like Growth Factor-1]
- IOC: Failure to suppress GH levels with oral glucose load
- Serum Prolactin levels increases
- MRI Head- Size of Tumor> 10mm
- X-ray of lateral as pect of Foot is done to see Heel Pad ThicknessHeel pad thickness





Treatment

00:23:44

- Trans-sphenoidal surgery
  - 1st to improve After surgery is → soft tissue swelling
  - After (1-2hrs) → GH levels decreases
  - After (3-4hrs) → IGF-1 levels decreases
- Prior to the surgery, shrink the size of the tumor using drugs→ Somatostatin analogues
  - Octreotide
  - Lanreotide
  - Pasireotide

  } Blocks SSTR 2 & SSTR 5 Receptors → GH decreases
- PEGVISOMANT
  - GH Receptor blocker
  - GH levels ↑ due to feedback
  - Used to prevent recurrence of tumor
- Radiation

SHEEHAN SYNDROME

00:28:16

- Anterior Pituitary defect
  - Pan- hypopituitarism
- Growth Hormone → 1st to fall
  - Results in hypoglycemic attack
    - → Earliest manifestation
    - → Rage attacks
    - → Tremors
    - → Palpitations

→ Diaphoresis

- Prolactin levels fall → resulting in Lactation Failure
- LH, FSH ↓ → 2° Amenorrhea → 2° Infertility
- ACTH ↓ → Cortisol ↓ → Blood sugar levels ↓
- TSH ↓: T4 ↓, T3 ↓→ resulting in secondary hypothyroidism → wt. gain, cold intolerance etc

Important Information

- SIMMOND DISEASE
  - Non-obstetric cause to pituitary damage
  - Presents with HTN, AVM Rupture, Sickle cell anemia, Trauma etc.
  - Treatment is same as Sheehan syndrome

Imaging

- MRI head

Treatment

- Dexamethasone
  - 1st to be supplemented
  - Stabilize blood sugar levels
- Life-Long Levothyroxine
- O.C.P/ C.O.C → Oestrogen / Progesterone

HYPOPITUITARISM

00:35:34

- HYPOPITUITARISM
  - Implies only GH deficiency
  - Seen in pediatric population
  - May be d/t pituitary dysplasia
- Pan Hypopituitarism
  - Decrease of all the 6 Hormones produced by anterior pituitary
  - Seen in SHEEHAN SYNDROME and SIMMOND DISEASE

Features

- Present since birth (developmental defects)
- Short stature
- Squeaky high-Pitched Voice
- Doll like Facies
- Bone age delayed

INVESTIGATIONS 00:39:12

- Screening test: Decreases IGF-1 levels
- IOC: Insulin challenge test / Arginine challenge test

Treatment

- GH injection (DNA Recombinant tech) till Puberty

Summary

ENDOCRINE DISORDER	TEST
Acromegaly	Glucose challenge Test
CONN	Saline infusion test / Salt Loading test
ADDISON	ACTH ⊕ Test / cosyntropin test
DI	Water deprivation test
SIADH	Water loading test
Hypopituitarism	Arginine Challenge Test
Cushing	Low dose dexamethasone suppression test
Pheochromocytoma	Serum Fractionated metanephrine levels

- Please note posterior pituitary diseases like SIADH and DI have been covered in kidney section with Barter Syndrome and gitelman syndrome

PROLACTINOMA

00:42:35

- M/c Functioning pituitary tumor
  - M/c Pituitary Tumor is non-functioning Tumor
- Dopamine inhibits prolactin production
  - So Dopamine agonists are used for t/t of prolactinoma
- Prolactin has inhibitory effect on LH/FSH

Clinical features

- Females
  - Anovulation
  - Amenorrhoea
  - Galactorrhea
- Males
  - Galactorrhea
- Bitemporal hemianopia
  - Tumor size > 10mm
  - Late presentation

Investigations

- Raised Serum Prolactin levels
- MRI head

Treatment

- Bromocriptine/cabergoline (Long acting)
- Surgery needed rarely

CLINICAL QUESTIONS

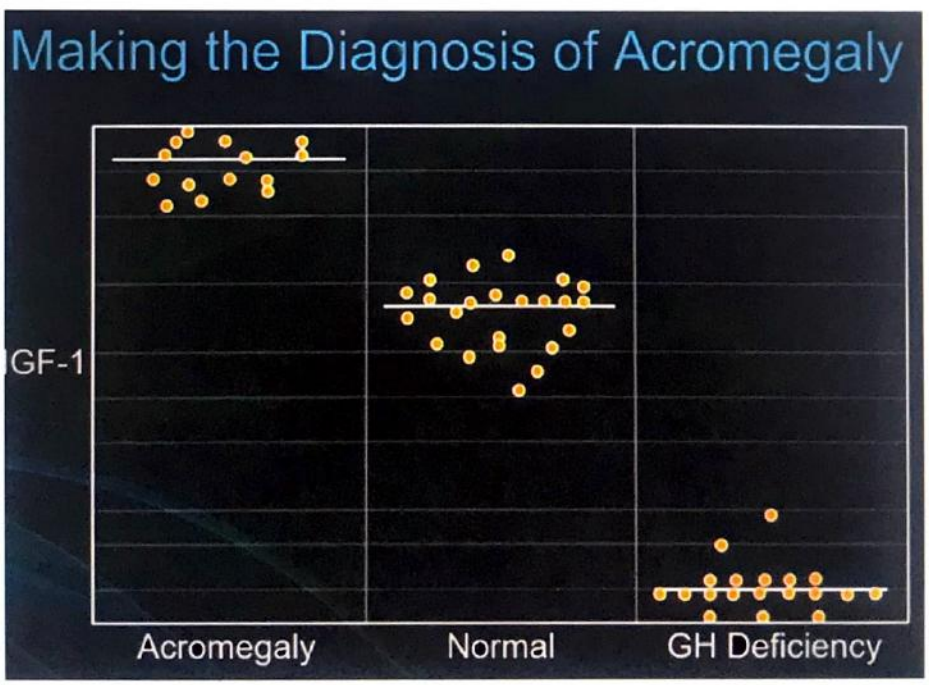
A 36yr old man comes to OPD with complaints of headache with some visual disturbance for past 6 months. He also complaints of decreased libido, excessive sweating and weight gain to the extent that his rings and shoes doesn't fit him anymore. What will be most appropriate screening test in this patient?

A. Serum IGF-2
B. Oral glucose tolerance test
C. GH levels
D. Serum IGF-1

Answer: D

Solution

- The case described above is Acromegaly described by weight gain, sweating and especially **acral enlargement increase in size of hand and fee**t (shoes and ring doesn't fit him)
- **Age-matched serum IGF-I levels are elevated in acromegaly**. Consequently, an IGF-I level provides a useful laboratory screening measure when clinical features raise the possibility of acromegaly.
- The diagnosis of acromegaly is confirmed by demonstrating the failure of **GH suppression to <0.4 µg/L within 1–2 h of an oral glucose load (75 g)**. When newer ultrasensitive GH assays are used, normal nadir GH levels are even lower (<0.05 µg/L.**(Oral glucose tolerance test)**
- Owing to the pulsatility of GH secretion, measurement of a single random GH level is not useful for the diagnosis or exclusion of acromegaly and does not correlate with disease severity.
- **IGF-2** is responsible for **prenatal growth.**
- Oral glucose tolerance test with GH obtained at 0, 30 and 60 minutes. **Normal people can suppress growth hormone to <1g/L.**



Reference: Harrison's Principles of Internal Medicine - 20th Edition, Page No: 2679

Q. A 17-year old girl who was evaluated for short height was found to have an enlarged pituitary gland. Her T4 was low and TSH was increased. Which of the following is the most likely diagnosis?

A. Pituitary adenoma
B. TSH-secreting pituitary tumor
C. Thyroid target receptor insensitivity
D. Primary hypothyroidism

Answer: D

Solution

- **The clinical picture of short stature with low T4 and elevated TSH suggests primary hypothyroidism.** The enlarged pituitary can be explained by the feedback provided by low T4 in the blood.
- To rule out other choices

Pituitary adenoma	Can produce any hormone and if it produces TSH , then **T4 should be elevated**
TSH Secreting pituitary tumor	Both TSH and T4 should be elevated
Thyroid target receptor insensitivity	1. **Autosomal dominant** disorder insensitivity. 2. Characterized by elevated thyroid hormone levels and inappropriately **normal or elevated TSH**. 3. The clinical features of Resistance to Thyroid Hormone can include goiter, attention deficit disorder, mild reduction in IQ, delayed skeletal maturation, tachycardia, and impaired metabolic responses to thyroid hormone.

Reference: Harrison 19th p 2287, 2292/ Harrison 20th p 2697, 2699

48 MULTIPLE ENDOCRINE NEOPLASIA

00:00:19

1. ≥ 2 endocrine tumors in a person, developed over different decades of life
2. Familial basis – Expressed in every generation
3. Genetic analysis
   - MEN – II / Type II a / Sipple Syndrome – RET mutations
   - MEN – I – (MEN – I) gene mutations
- Dx Criteria – Atleast 1 out of 3 should be Present

MEON (MULTIPLE ENDOCRINE & OTHER ORGAN NEOPLASIA'S)

- Von Hipple-Lindau Syndrome (VHL)
  - (Renal Cell Ca+ Cerebral & retinal hemangioblastomas)
- COWDEN Syndrome
  - (A PTEN hamartoma tumor syndrome)
- McCune Albright Syndrome
  - (Polyostotic fibrous dysplasia, caffe-au-lait spots and autonomous endocrine hyperfunction)

Important Information

- **MEN syndromes have Autosomal dominant inheritance**

MEN I / WEMER SYNDROME

00:04:08

(11q 13 / MEN-1 gene / Menin)

- Menin
  - Regulation of cell division
  - Dysfunction leads to Dysregulation of cell cycle
    → Responsible for MEN 1 syndrome
- Parathyroid adenoma (MC)
- Pancreatic tumor (Gastrinoma / Non-functioning tumor)
- Pituitary adenoma (Prolactinoma)
- Multiple Ulcers even upto 2nd part of duodenum
  ↓
  Perforation
  ↓
  MC cause of death
- Associated conditions
  - Angiofibroma
  - Adrenal cortical tumor
  - Carcinoid tumor
    → Thymus
    → Bronchus
- Presents with
  - Hypercalcinaemia
  - Epigastric pain
  - Infertility etc.
- Presents in 5th- 6th decade of life

MEN-2 / SIPPLE SYNDROME / MEN 2A

00:10:02

- Mutation in RET gene
  - Present on Chromosome 10
- Pheochromocytoma (Epi > NE) – MC Cause of death
- Medullary Ca thyroid – MC presentation
  - Amyloid deposition
  - ↑ Calcitonin levels
  - Neck USG, FNAC Thyroid Scan (Thyroid Ca-Cold Nodule)
  - Treatment: Total Thyroidectomy + Central neck dissection
- Pheochromocytoma → Catecholamines ↑ → Short duration
  - Episodic HTN (paroxysmal)

Important Information

- **In Both MEN 2 & 3 – MC presentation – Medullary Ca thyroid (Marker - ↑ Calcitonin levels)**

MEN – 3 / MEN 2B

00:16:32

- Medullary Ca thyroid (MC Presentation)
- Pheochromocytoma
- 4M
  - Mucosal neuromas (Bumpy Lips & Tongue)
  - Marfanoid Habitus (when arms are spread – longer than height of the person)
  - Medullated Corneal nerve fibres
  - Mega colon

MEN – 4

00:20:34

- CDKN 1B gene (Chr. 12 p 13) involved
- MEN - 1 + Reproductive tract tumor
  - Testicular Ca
  - Neuro endocrine cervical Ca
  - Adrenal, Renal Tumor

CLINICAL QUESTIONS

Q. A 38yrs old, female presented with complaints of headache & palpitation. She is noted to have profuse sweating. On examination, her blood pressure was 155/95mmHg.ECG showed sinus tachycardia with Heart rate of 108/min. Investigations were done & she is diagnosed as a case of pheochromocytoma. Which of the following hormones would be secreted in a higher concentration in such a patient?

A. Norepinephrine
B. Epinephrine
C. Dopamine
D. VMA

Answer: A

Solution:

- Normally adrenal medulla secrete epinephrine>norepinephrine.
- The major catecholamine secreted in a **Pheochromocytoma** is norepinephrine in 80% cases.
- The term paraganglioma is used to describe catecholamine producing tumors in the head and neck. These tumors may secrete little or no catecholamines.
- If the size of **Pheochromocytoma** is < 5cm, then the **major catecholamine** produced is **epinephrine.**
- If **Pheochromocytoma** is seen with **MEN 2A**, then the **major catecholamine** produced is **epinephrine.**

Reference: Harrison's Principles of Internal Medicine - 20th Edition, Page No: 2740

LEARNING OBJECTIVES

NEPHROLOGY

Electrolyte Imbalance

- Hyponatremia, Hypernatremia, Hypokalemia, Hyperkalemia
- Hypo and hypercalcemia, Hypomagnesemia, Hypermagnesemia
- Metabolic acidosis and Metabolic alkalosis

Bartter Syndrome, Gitelman Syndrome, Liddle Syndrome & Comparison with SIADH

- Gitelman syndrome: Features, Work Up, Treatment
- Bartter syndrome: Manifestations, Work Up, Treatment
- Liddle syndrome: Work Up, Treatment
- Diabetes insipidus: Features, Types, Work Up, Treatment
- SIADH

Ciliopathies / Chronic Tubulointerstitial Disorders

- CILIA, Ciliopathy
- Medullary sponge kidney, Investigation
- ARPKD, Extra Renal Manifestation of Autosomal Dominant PKD
- ADTKD (Autosomal Dominant Tubulointerstitial Kidney Disease)

Polycystic Kidney Disease

- Clinical features, Extra renal manifestation (ADPKD), Work up of patient of ADPKD
- Treatment

Thrombotic Thrombocytopenic Purpura

- Causes of TTP, Clinical features, Work up, Treatment
- H.U.S (Hemolytic Uremic syndrome), Atypical H.U.S, Treatment of HUS
- HUS vs TTP
- HUS vs RVT

Urine Analysis

- Proteinuria, Hematuria, Eosinophiluria

Acute Kidney Injury

- Etiology of A.K.I, Vascular Cause Responsible for Aki, Clinical Features Of A.K.I
- Work Up, Management of A.K.I, Indication for Hemodialysis

Chronic Kidney Disease

- Cockcroft Gault Formula, Grading, Causes of CKD, Treatment
- Allogenic kidney transplant, HLA inheritance, Maintenance therapy
- Opportunistic infection post kidney transplantation, Hemodialysis

Diabetic Nephropathy

- Histopathological aspect of Diabetic nephropathy
- Treatment

Kidney Stones

- Calcium Oxalate Stone, Calcium Phosphate Stones, Triple Phosphate Stone, Urate Stone, Cystine Stone, Xanthine Stone
- IOC For Renal Stones, Treatment of Renal Stones

Renal Tubular Acidosis

- RTA – 1, RTA -2, RTA-4
- Summary of Renal Tubular Acidosis

Nephrotic and Nephritic Syndrome

- Nephrotic Syndrome, 1° Nephrotic Syndrome
- Membranous Glomerulopathy (MGN)
- Congenital Nephrotic Syndrome
- MCD (Minimal Change Disease)
- Nephritic Syndrome
- Berger's Disease / IGA Nephropathy

Renal Artery Stenosis

- Etiology, Clinical Features
- Work up, Treatment
- Atheroembolic Kidney Disease

49 ELECTROLYTE IMBALANCE

HYPONATREMIA

00:01:00

- M/c electrolyte imbalance abnormality in hospitalized pts.
- Affects plasma osmolality
- Changes in plasma Na^+ levels will affect plasma osmolality that will cause fluid shift across brain cells and can cause life threatening seizures.
- Plasma Osmolality: $2(Na^+ + K^+) + \frac{BUN}{2.8} + \frac{Glucose}{18}$
- Normal plasma osmolarity: 285-295 mOsm/L
- Urine osmolality: 100-900 mOsm/L, it usually fluctuates a lot and helps to maintain plasma osmolality in a narrow range of 10 mOsm/L.
- Normal Na^+: 135-145 meq
- Mild Hypo Na^+: 130 – 135 meq
- Moderate Hypo Na^+: 125-130 meq
- Severe Hypo Na^+: < 125 meq – High risk of causing the fluid shift across the brain predisposing - Life threatening seizures

a. Hypotonic Hyponatremia

- There will be abnormal plasma osmolality i.e < 285 mOsm/L.
- Subsequent types of Hyponatremia depend on volume status of patient.
- In Diarrhoea/ Vomiting the TBW ↓ and TBS ↓ therefore this type of hypotonic hyponatremia is K/a Hypovolemic Hyponatremia. Rx - fluids
- In SIADH → ADH ↑↑ → Acts on V_2 receptor and causes more reabsorption of H_2O → TBW ↑ and TBS is normal = Dilutional Hyponatremia (Euvolemic hyponatremia)

Important Information

SIADH can be caused by

- Infection (cerebral toxoplasmosis/Meningitis/ Encephalitis / Brain abscess) in which there is relative ↑ADH
- Ectopic production (Oat cell Ca lung and Carcinoid syndrome) in which there is absolute ↑ ADH

Clinical scenario

- In a Patient with Massive ascites there will be a reduction in Circulating fluid volume → therefore Blood supply to kidney is also less → GFR ↓ → This activates the RAAS causing 2° Hyperaldosteronism (values of aldosterone is elevated) which leads to → Activation Epithelial Na channels → salt and water enter the system → TBW ↑↑ and TBS ↑ causing Hypervolemic Hyponatremia

Clinical scenario

- In Patients of SAH (Worst headache of life. thunderclap headache). because of excruciating pain there is → Release of B.N.P causing Natriuresis → because there is loss of salt from the body → TBW ↓ and TBS ↓ causing Hypovolemic Hyponatremia (Cerebral salt wasting syndrome)

Summary of Hypotonic Hyponatremia

Refer Table 49.1

b. Hypertonic Hyponatremia

- ↑↑ in Osmolality of plasma > 295 mOsm/L
- Examples
  - Hyperosmolar comma: In patients with Hyperosmolar coma (high blood sugar values) → High blood sugar will increase the Plasma Osmolality and the high sugar content will → Draw in H_2O into the vascular compartments and ↓ Na+ levels.

Important Information

- If blood sugar ↑↑ by 100 mg% → Na^+ ↓↓ by 1.6 meq. Therefore, there is inverse relation b/w sugar & Na^+

  - Mannitol
  - Radiocontrast

c. Isotonic Hyponatremia

- Pseudo hyponatremia, because Na^+ is normal but values are low
- This can be due to 2 factors
  1. Hyperlipidemia
  2. Hyperproteinemia

Management of hyponatremia

- Whenever the Na^+ < 125 mEq in blood, there could be development of Seizures
- For Acute hyponatremia
  - Administering 3% saline (hypertonic saline)
  - Correction of Hyponatremia: 4-8 meq/ day (Gradual rise is better)

Important Information

- If fast correction of Hyponatremia is done, it result in → Stroke like manifestation in the patient K/a Central pontine myelinosis or Osmotic demyelination syndrome
- Clinical features include: stroke like Quadriplegia + Babinski sign +ve

Clinical Scenario

- 60 kg female athlete, marathon runner, collapsed before finishing the race. On examination Na^+ value 120 mEq. Calculate Na^+ correction over the next 24 hours.

Solution

- Formula for correction
  - Na^+ deficit = TBW x (desired value − actual value)
  - TBW = wt x 0.6 (males) / 0.5 (females)
- Na deficit = 60 x 0.5 x (128 - 120) → 240mEq over 24 hrs via infusion pump
- 1000ml of 3% saline contains 514 mEq of Na^+ and 514 mEq of Cl^-
- So, for 250 mEq we need to give 500ml of 3% saline (Approx) given over 24 hrs
- So, 500/24 = 20.83 ml/hr 3% saline need to be given
- In case of 0.9% normal saline = 154 mEq of Na^+ and Cl^- in 1000 ml
- 1ml N.S. =154/1000 = 0.15 mEq Na^+
- 1ml 3% saline = 514/1000 = 0.5 mEq Na

Important Information

Correction formula for hyponatremia

- Na deficit = wt. x 0.5/0.6 x (Desired Na value − Actual Na value)
- Desired value is always 8 more than actual value
- So Na deficit = TBW x 8 in 24 hours

HYPERNATREMIA

00:42:45

- Dangerous Hyper Na+ > 158 mEq/L: leads to development of seizures in a patient.
- Rx: Diluted fluids (5% dextrose or N/2 in 5% dextrose)
- Hypernatremia is mostly seen in Geriatric age group
- Cause: Loss of water from body like in extremely debilitated old patients having extreme thirst
- Investigation: Urine osmolality
  - If osmolality is < 250 mOsm/L in case of DI with polydipsia and no access to water
  - If osmolality is > 400 mOsm/L Can be due to excessive loss of water by
    - → Extreme sweating
    - → Lactulose will cause Osmotic diarrhea
    - → Mannitol in excess
- In asymptomatic hypernatremia: Liberal H_2O intake
- Volume of fluid correction in case of Na↑ ~160

$$mEq = TBW \times \frac{Na^+ actual - 140}{140}$$

- Example
  - A 60 kg female having a post operative day 3 status of sodium 160 meq the amount of water that has to be

$$\text{given to this patient} = 60 \times 0.5 \times \frac{160-140}{140}$$

$$= 30 \times \frac{20}{140} = 4 \text{ L/day}$$

Important Information

- In case of DKA there will be increase in Sugar value
- Sugar drag water into the blood circulation k/a Solvent drag
- Because of solvent drag the water will come into blood circulation along with K^+ causing K^+ Efflux
- Therefore, $PH \propto \frac{1}{K^+}$
- Eg. Any condition where K^+ is high in the blood → affects the function of the kidney tubules → it inhibits tubular exertion of NH_3^+ ions →Therefore H^+ ions accumulate in blood causing metabolic acidosis
- K ↑ by 0.7 meq/l for every 0.1 ↓ in pH

HYPOKALEMIA

00:56:33

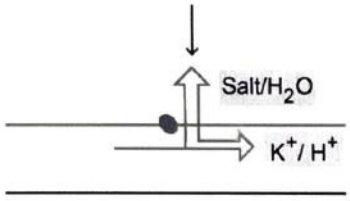
- Death can occur in Hypokalemia due to respiratory muscle paralysis leading to buildup of Co_2 in the body, Co_2 narcosis will also occur in the patient

Causes of Hypokalemia

a. Decreased intake
b. Alkalosis: Causes potassium shift into cells
c. Excessive Sympathomimetic stimulation
- Trauma
- Thyrotoxic periodic paralysis
- Beta 2 agonist toxicity (Salbutamol)

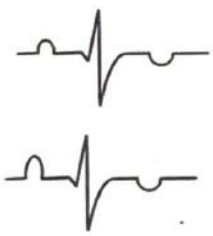
d. Renal loss
- ↑ Aldosterone = Conn's syndrome, Bilateral adrenal Hyperplasia, Ascites, CHF
- Barter's, Gitelman syndrome
- Salt wasting nephropathy
- RTA type 1 and 2

e. Vomiting, diarrhea



Clinical features
- Cramp/ weakness (Earliest)
- Ileus
- Flaccid paralysis
- Hypercapnia: CO_2 narcosis

Work up
- Serum K^+ (Best test)
- TTKG: Trans tubular K^+ gradient
  - If > 4 = renal wasting of K^+
- ECG
  - T wave: height of T wave will be ↓ / absent/ inverted
    → D/D: Unstable angina, NSTEMI, Hypokalemia



  - ST ↓
  - Pseudo P-pulmonale: p wave > 2.5mm in absence of PAH
  - Prolongation of PR interval
  - Prominent U waves
  - Prolonged QU interval

Important Information

- Hypokalemia can trigger ventricular Arrhythmia
  ↓
  Torsades De Pointe (Hypomagnesemia also causes this)

Management
- If K^+ b/w 3.0 – 3.5 mEq/l: Oral KCL (POTCHLOR)
- If < 3 mEq (Can't take orally): I.V KCl (slow infusion)

Important Information

- For 1 meq/l ↑ rise in blood: give 200 meq of KCl (slow I.V)
- E.g. A Patient is on Amphotericin B. K^+ = 2.3 mEq. Calculate correction?
  - Solution
    → 2.3 mEq → 3.0 mEq: ↑ 0.7 mEq
    → 0.7 mEq x 200 → 140 mEq of KCl/24 hrs fluid

- Conc of K^+ delivered on central line = 20 mEq/l
- Conc of K^+ delivered on peripheral line = 40 mEq/l

Previous Year's Questions

Q. Pseudo p pulmonale is seen in? (NEET Jan 2018)
A. Hypokalemia
B. Hyperkalemia
C. Hypomagnesemia
D. Hypercalcemia

HYPERKALEMIA

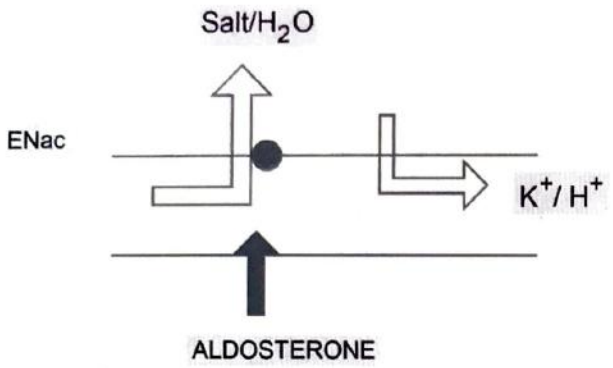
01:13:42

- $PH \propto \frac{1}{K^+}$ (K^+ ↑ 0.7 mEq for 0.7 ↓ pH)
- $K^+ > 8.0$ mEq
- Causes of death: diastolic arrest (Systolic arrest seen when Ca > 13 mg %)

Causes
- Pseudo hyperkalemia
  - Fist clenching
  - Narrow bore needle

- Cooling of sample
- Elevated TLC and Platelet, RBC↑

- Acidosis: Trans-cellular shift
- CKD / AKI
- Aldosterone deficiency: aldosterone causes loss of K^+ via distal tubule, if aldosterone level goes down then K^+ in blood will be elevated
  - Addison disease
  - Histoplasmosis
  - HIV
  - Waterhouse Frederickson syndrome



- Gordon syndrome: There is gain of function of NaCl cotransport in the distal convoluted tubule → ↑ Salt in body →⊖ RASS → ↓ Aldosterone

Work-up

- Values of serum K^+ will be increased
- ECG shows
  - Tall tented T wave
  - ST ↑
  - Height of P wave ↓ (usually when the values of K^+ is 7 mEq)
  - QRS broad
  - If > 8.0 mEq, ECG shows Sine Wave Pattern: In these patients Diastolic arrest will occur

Management

1. Antagonism: we use
- Ca gluconate (Usually used)
- Ca chloride (Best)

2. Redistribution
- Insulin drip with 50% dextrose (Most Effective)
- Salbutamol nebulization: Dose used here is 4x than asthma (cause extensive sympathomimetic stimulation

3. Removal of K+: we use
- Furosemide
- Sodium polystyrene sulphonate (resin)
- PATIROMER (K^+ binding agent)
- ZS – 9 (Na^+ zirconium cyclosilicate)

Important Information

- Soda bicarbonate is not routinely used for Mx of metabolic acidosis

Previous Year's Questions

Q. Which of these has no role in treatment of dangerous hyperkalemia? (FMGE Dec 2017)

A. Inj calcium chloride
B. Salbutamol
C. Hemodialysis
D. IV soda- bicarbonate

HYPO AND HYPERCALCEMIA

01:24:24

Hypocalcemia	Hypercalcemia

• Tetany: Death due to Laryngospasm
• Management
  ○ I.V 10% Ca gluconate (DOC)
	• Acute hypercalcemia crisis: Death due to systolic arrest
	• Management
	  ○ Normal Saline: cause Hydration
	  ○ Furosemide drip: cause Calciuria
	  ○ I.V. bisphonate (Ibandronate)
	  ○ Calcitonin nasal spray: Antagonist of PTH
	  ○ I.V. hydrocortisone: in sarcoidosis, vit D_3 intoxication

HYPOMAGNESEMIA

01:28:38

- Normal Mg range = 1.3 – 2.1 mEq/l
- Causes
  - Diarrhea, Alcoholics
  - Drugs
    - → Thiazides: Thiazides act on the DCT which contains a transporter TRPM6, responsible for magnesium reabsorption. Thiazides block this receptor causing magnesium loss
    - → Amphotericin B
    - → Aminoglycosides
  - Renal wasting: Gitelman syndrome

Pathophysiology

- Wherever Mg ↓, Ca^{2+} antagonism ↓ → Blood Vessel constriction → BP ↑
- Wherever Mg ↑, It antagonizes intracellular Ca^{2+} → Blood vessel relaxation
- If Mg ↑/↓ in the blood it inhibits PTH release → less PTH decreases the calcium values and also contribute to increased neuromuscular irritability

Clinical feature

- Muscle cramps
- HTN, HR ↑, arrhythmia (Torsades de pointes)
- Tremors / nystagmus / athetosis

Work up

- S. Mg^{2+}
- Urine Mg^{2+}
- ECG shows a prolonged QT which predisposes to Torsades de Pointes

Management

- I.M / I.V $MgSO_4$
- Oral Mg oxide
- When giving infusions of Mg always watch DTRs, Urine output

Important Information

- I.V $MgSO_4$ is also given in
  - Eclampsia
  - Severe acute asthma
  - PEM grade III
  - Gitelman Syndrome

HYPERMAGNESEMIA

01:35:15

- Cause of Death: Asystole

Cause

- CKD
- Eclampsia: $MgSO_4$ toxicity
- Antacids / Laxative abuse

Pathophysiology

- High magnesium inhibits PTH release → ↓ Ca^{2+} → BV relaxation
- Neuromuscular excitability ↓ → decreased reflexes

Clinical features

- Shock Non responsive to vasopressors and IV fluid
- DTR ↓
- Respiratory rate ↓: cause CO_2 ↑
- Urinary output ↓

Management

- Vigorous IV hydration
- Ca gluconate
- Hemodialysis

Summary

01:38:56

	Cause of death	TOC
• NA^+ <125 mEq	Seizures	• 3% saline
• NA^+ >158 mEq		• 5% dextrose
• K^+ > 8.0 mEq	Diastolic arrest	• Ca gluconate
• K^+ < 2.5 mEq	Diaphragmatic Paralysis	• KCL drip
• Ca^{2+} > 13 mg%	Systolic arrest	• Ibandronate
• Ca^{2+} < 7 mg%	Laryngospasm	• Ca gluconate
• Mg^{2+} > 10 mEq	Asystole	• Ca gluconate
• Mg^{2+} < 1.0 mEq	V. Arrhythmia	• $MgSO_4$

METABOLIC ACIDOSIS

01:41:51

Causes

- **K** - Ketoacidosis: DM/ Starvation/ Alcoholic
- **U** - Uremia: AKI/ CKD/ RAS/ ATN
- **L** - Lactic acidosis: 3 types
  - Type A: Mainly due to shock, CO poisoning
  - Type B: D.M/ Drugs (Phenformin, Vancomycin)
  - Type D: Short bowel syndrome, Jejunoileal bypass Sx
    - → Carbohydrate fermentation by bacterial flora in patients having short bowel syndrome produce D-lactate.
- **T** - Toxins
  - Methyl alcohol
    - → Metabolized by Alcohol dehydrogenase to form → Formaldehyde
    - → This formaldehyde gets oxidized to form Formic acid
    - → Formic acid has a very low PH and H^+ are

produced which causes damage to BBB causing the Encephalopathy

→ Management

- To inhibit conversion of Methyl alcohol into formaldehyde: Fomepizole

- Ethylene glycol (Antifreeze agent)

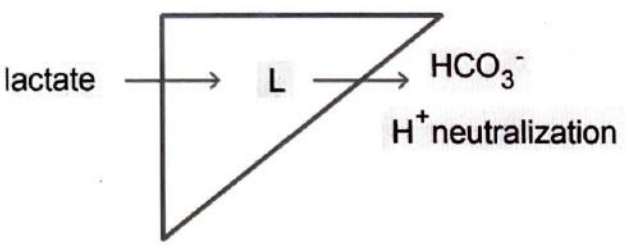
→ Consumption can cause precipitation of Ca oxalate crystals and cause ATN

How to remember

- KULT

Management

- Fluid of choice: Ringer lactate

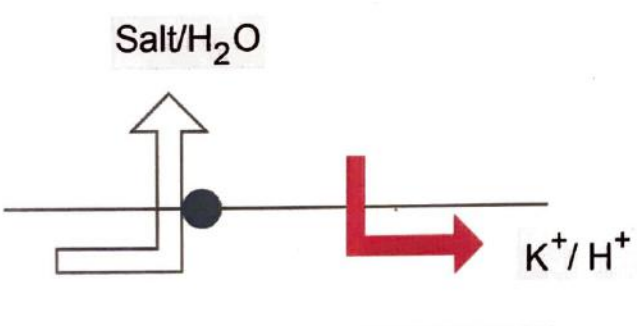


- If pH <7.2, inspite of adequate fluid resuscitation: give soda bicarbonate.
  - Total correction: 0.5 x wt. x (24-actual value)
    → ½ is given as bolus
    → ½ with IV fluid infusion
  - Initial correction: 0.5 x weight x (15 - Actual value)

METABOLIC ALKALOSIS

01:53:21

- In chronic vomiting there will be loss of H_2O + HCl (Hypochloremia) → Dehydration → this will lead to GFR ↓↓, activation of RAAS and ↑ Aldosterone → Hypokalemic hypochloremic metabolic alkalosis



- Cause of chronic vomiting can be: Congenital hypertrophic pyloric stenosis, Ca stomach, healed peptic ulcer disease.

Management

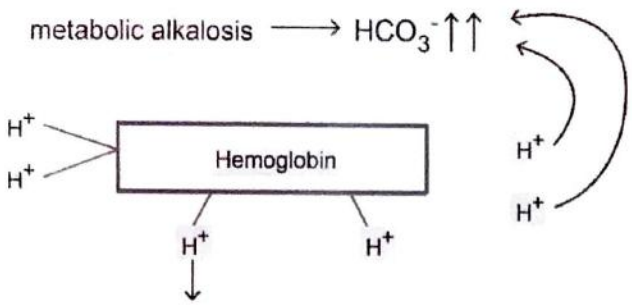
- Fluid of choice: Normal Saline (0.9%)
- When normal saline is used to treat the patient is referred as saline / chloride responsive metabolic alkalosis
- But if still pH: > 7.55 in spite of fluids + give NH_4Cl
- DKA (Insulinopenia)
  - Efflux of K^+ out of cell causing Hyperkalemia
  - RL (K^+ rich fluid): so Contraindicated as it will further increases potassium values.
  - Fluid of choice in DKA: Normal Saline
  - TOC: Insulin drip/ I.V regular insulin
  - So in all acidosis we give RL but in DKA we give NS
- In patient with Ascites
  - Circulating fluid volume ↓ and GFR ↓↓ and causes the activation of RAAS
  - Activation of RAAS causes release of Aldosterone which leads to the activation of ENaC channels.
  - There occurs the loss of K^+/H^+ and retaining of salt/H_2O causing Hypokalemic metabolic alkalosis
  - TOC: Spironolactone (Aldosterone antagonist)
  - Causes Hypokalemic metabolic alkalosis - chloride non responsive metabolic alkalosis
    → Nephrotic syndrome
    → Protein losing enteropathy E.g. celiac sprue, Menetrier's disease
    → CHF
    → Cirrhosis

Summary

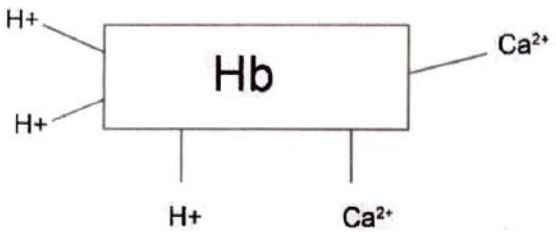
Metabolic alkalosis		
Chloride / Saline Responsive	**Chloride / Saline non-responsive**	
1. PUD healed 2. Ca stomach 3. CHPS	• Ascites o CHF o Cirrhosis o Nephrotic	• ↑ Aldosterone o CONN o Cushing o B/L adrenal hyperplasia
•**Rx:** Normal saline	• **Rx:** Spironolactone and restrict salt intake	

Tetany in metabolic alkalosis

1. Mobilization of H^+ ions to neutralize excess HCO3



2. Creation of vacant site on Hb
3. Vacant sites get occupied by lonized Ca^{2+}



4. Ionized Ca^{2+} ↓↓ → Tetany (d/t Ca^{2+} re – distribution) → metabolic alkalosis can lead to Tetany by causing redistribution of calcium.

Previous Year's Questions

Q. A 3 week old boy baby with diagnosis of congenital hypertrophic pyloric stenosis. The baby is scheduled for Ramstedt operation tomorrow morning. Pre-operative electrolyte report shows Ca^{2+} 6.0 mg%. What is next best step?

A. Cancel the surgery
B. Administer ringer lactate
C. Calcium Gluconate
D. Normal saline

Ans D.

- Ringer lactate if administered will produce HCO_3^-. When HCO_3^- is produced in excess will worsen the metabolic alkalosis
- As it is a redistribution and not a loss of Calcium. calcium gluconate is not given

Previous Year's Questions

Q. A 20 year female suffering from anxiety neurosis, fear of heights, travel by air and hyperventilates causing respiratory alkalosis develops perioral paresthesias and carpopedal spasm

Rx: Paper bag Rebreathing

Respiratory Acidosis Vs Respiratory Alkalosis

02:15:29

R. Acidosis	R. alkalosis
$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$	
Causes	**Causes**

• Severe acute asthma
• Flail chest
• COPD exacerbation
• Diaphragmatic paralysis
  ○ ↓ K^+, GBS, Transverse myelitis, Botulin ism
  ○ Cervical fracture C3, C4, C5
	• Hyperventilation
	• High altitude pulm. Edema
	• Pneumonia
	• Pleural effusion
	• ILD
	• Acute asthma

Summary

	Causes	Treatment
• M. acidosis	• **K – K**etoacidosis • **U – U**remia • **L – L**actic acidosis • **T - Toxins**	• RL / Soda Bicarbonate
• M. Alkalosis	• Gastric outlet obstruction • Conditions contributing to ascites or Endocrine Aldosterone	• G.O.O: Normal saline • Aldosterone↑: Spironolactone
• R. Acidosis	• Increase in CO_2	• IPPV
• R. Alkalosis	• CO_2 washout	• Paper bag rebreathing

Normal values

- pH: 7.35 – 7.45 mmHg
- po2: 60-100 mmHg
- pco2: 35-45 mmHg
- HCO3: 22-26 meq (Average = 24 mEq)

Important Information

- CO2 narcosis → Asterixis → Flapping tremors
- Cause
  - Ammonia ↑
  - BUN ↑
  - CO_2 narcosis

Table 49.1

Hypovolemic	Euvolemic	Hypervolemic
• TBW and TBS are decreased	• TBW↑ and TBS normal	• TBW ↑↑ and TBS↑
Causes 1. GI causes: urinary Na ↓ • Diarrhea • Vomiting 2. Renal causes: Urinary Na ↑ • Cerebral salt wasting • Diuretics • Addison disease	**Causes** 1. SIADH 2. Post-operative Nausea/Vomiting d/t anaesthetic drugs which stimulates release of ADH ↑ 3. Endurance sports 4. Psychogenic Polydipsia 5. Beer Potomania 6. Hypothyroidism	**Causes** 1. CHF 2. Cirrhosis 3. Nephrotic 4. CKD
Management • ORS / RL	**Management** • Initially: fluid restriction • Vaptan: V_2 #	**Management** • Diuretics

CLINICAL QUESTIONS

Q. A patient of chronic kidney disease is having protracted vomiting. ABG report is pH= 7.4, pCO_2 =40 mm Hg, HCO_3= 25 meq, Na= 145 meq and Chloride s 100 meq. Diagnosis is?

A. Normal anion gap metabolic acidosis
B. High anion gap metabolic acidosis
C. No acid base abnormality
D. High anion gap metabolic acidosis and metabolic alkalosis

Answer: D

Solution

- CKD leads to metabolic acidosis while **vomiting leads to metabolic alkalosis**
- The key to the answer is anion gap which is elevated to 25 **implying unmeasured anions. (Normal average of 10 meq)**
- It points to mixed etiology of metabolic acidosis and alkalosis.
- Normal values of pH, pCO_2 and HCO_3 does not ensure absence of acid base imbalance.

50 BARTTER SYNDROME, GITELMAN SYNDROME, LIDDLE SYNDROME & COMPARISON WITH SIADH

GITELMAN SYNDROME

00:00:12

- Autosomal recessive
- Defect in "Na^+- Cl^- co-transporter and TRPM6 transporter" in DCT
- Hallmark feature: Salt wasting and Polyuria.

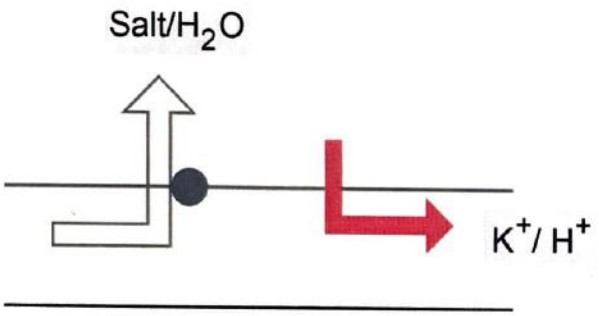
Features

- Failure to thrive
- Dehydration: Sunken AF/ delayed skin pinch
- Diaper change frequency ↑
- GFR ↓: Renin ↑: Aldosterone ↑ (2° Aldosteronism)
- Sluggish MORO/ Poor cry: manifestation of Hypokalemia
- Mg^{2+} wasting: Release of PTH ↓ → hypocalcemia (tetany)

Work Up

00:07:37

1. Serum Electrolyte: Na^+ ↓ K^+ ↓
2. Urine Osmolality: Low, ↓
3. 24-hour Urinary Chloride↑: IOC
4. Serum Mg^{2+}: ↓↓

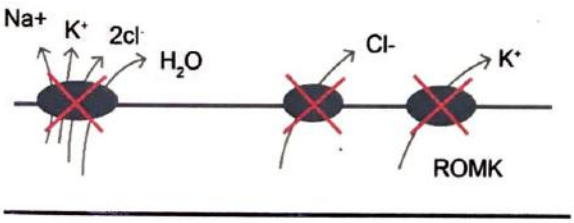


Treatment

- No definitive treatment

BARTTER SYNDROME

00:11:02



- Hyper prostaglandin E syndrome: Term used to describe one of the severe verity of Barter syndrome i.e antenatal form
- Autosomal recessive
- Defect in "Na-K-2Cl co-transporter, Cl channel & ROMK" in loop of Henle
- These channels are responsible for voltage gradient which causes Ca++ reabsorption.
- Hence defect in these channels leads to Ca^{2+} wasting
- Cl^- Channels present in inner ear are also defective: SN deafness.
- Poly hydramnios
- Salt wasting / polyuria

Manifestations

- Failure of thrive
- Dehydration: Sunken AF/ delayed skin pinch
- ↑ Aldosterone (2° Aldosteronism) → Hypokalemic alkalosis
- Chloride non-responsive/ saline non responsive metabolic alkalosis
- K^+ channel defect: Hypokalemia severity ↑ than Gitelman syndrome
- Voltage gradient ↓→ calcium resorption↓ → Hypercalciuria
  - Hypercalciuria can manifest in 2 ways
    - → Kidney stones
    - → Renal rickets: Delayed dentition, wide open AF, short stature

Important Information

- **Calcium loss in the patients is sufficient to cause stone formation and renal rickets but not severe enough to cause tetany**

Work Up

00:19:22

1. Serum electrolyte: Na ↓, K↓
2. 24 hr. U. chloride: ↑
3. Serum magnesium: Normal (TRPM-6 is still functional in DCT)
4. 24 hr U. calcium: ↑

Treatment

- Indomethacin: inhibit prostaglandin synthesis and there will be improvement

GITELMAN (DCT #) BARTTER (TAL#)

- Common features: Failure to thrive, Polyuria, Dehydration, Na↓, K↓
- Common test: 24 hr urine Cl↑
- BP can be Normal / decreased but never increased in both

- TRPM6 # → Mg ↓
- Tetany
- Treatment: no definitive treatment

- SN deafness
- Calcium wasting
  - Nephro-calcinosis
  - Renal rickets
- Treatment: indomethacin

Conditions in which Indomethacin is the DOC

- PDA (PT)
- Acute gout
- Acute migraine (Mild – moderate)
- BARTTER Syndrome

LIDDLE SYNDROME

00:26:33

- Gain of function of epithelial Na channel (ENaC)

Features

- HTN
- Hypokalemic alkalosis
- Low renin HTN (HTN → feedback RAAS → ↓ Renin → ↓Aldosterone)
- Dehydration is absent

Treatment

- Amiloride (ENaC inhibitor)

Important Information

- Hypokalemic Metabolic alkalosis seen in
  - BAH/CONN
  - Cushing syndrome/disease
  - Chronic vomiting (CHPS/ Healed PUD/ Ca stomach)
  - Ascites: CHF / Cirrhosis/Nephrotic syndrome
  - BARTTER syndrome
  - Gitelman syndrome
  - Liddle syndrome
- For Chronic vomiting (CHPS/ Healed PUD/ Ca stomach) Normal saline is used, as all the conditions mentioned are chloride responsive alkalosis
- For other conditions except BARTTER, Gitelman and Liddle syndrome Spironolactone is used
- For BARTTER: Indomethacin
- For Liddle: Amiloride

Previous Year's Questions

Q. Hypertension with hypokalemic alkalosis is? (NEET Jan 2018)

A. Liddle syndrome
B. Gitelman syndrome
C. Barter syndrome
D. Fanconi syndrome

DIABETES INSIPIDUS

00:33:25

Features

- Polyuria: Urine output > 3l /day
- Polydipsia
- Nocturia

Types

1. Central Diabetes Insipidus
2. Nephrogenic Diabetes insipidus

1. Central Diabetes Insipidus
- Damage to the Posterior pituitary or the Hypothalamus which can be due to
  - Idiopathic cause
  - Tumor
  - Post cranial surgery
  - Head injury
  - Granulomatous damage to the posterior pituitary
    - → Sarcoidosis
    - → Histiocytosis-x
  - AV Malformation
  - Sheehan syndrome

2. Nephrogenic Diabetes insipidus
- Due to V_2 Receptor resistance
- Cause of NDI
  - Drugs: Lithium, Amphotericin B
  - Hypercalcemia
  - Sickle cell anemia
  - Amyloidosis

Work up

00:37:31

1. Timed 24hr Urine collection
2. Urine Osmolarity (decreased), Plasma osmolarity (increased), Na concentration (Increased)
3. Plasma ADH level
4. Water deprivation test (Miller-Moses)

Treatment

- Central Diabetes Insipidus
  - Desmopressin (first line drug)

- Nephrogenic diabetes insipidus
  - Thiazides
  - Indomethacin
  - Amiloride

SIADH

00:47:46

Causes

- CNS infection leading to damage of H-P axis causing a Resetting of Osmostat in the Hypothalamo Pituitary Axis
- Ectopic sources
  - Kulchitsky cell Tumor/ carcinoid syndrome
  - Oat cell cancer
  - Drugs: Vincristine, Chlorpromazine, Haloperidol, Chlorpropamide
  - Multiple sclerosis

Criteria

1. Plasma osmolarity <275 mosm/Kg H_2O
2. Clinical euvolemia (TBW↑, TBS normal): no edema, no ascites, no orthostatic hypotension
3. Urine Osmolarity ↑
4. Urine sodium: >40 mmol/L
5. Normal Thyroid function test, N adrenal function test
6. Normal KFT
7. No use of diuretics
8. No hypokalemia

Supportive evidence in diagnosis of SIADH 01:01:52

- BUN: ↓
- Uric acid: ↓
- IOC: Water loading test

Treatment

- Primary treatment: Water restriction
- VAPTANS: V_2#
  - Tolvaptan: Also used for Hyponatremia in CHF

Diabetes Insipidus VS SIADH 01:05:50

Refer Table 50.1

Normal values

- Plasma osmolarity: 275- 295 mosm
- Urine Osmolarity: 100 – 900 mosm

Table 50.1

	SIADH (gain of H_2O)	DI (loss of H_2O)	Psychogenic polydipsia	Adipsic Hypernatremia
U. osmolarity	↑	↓	↓	↑
P. osmolarity	↓	↑	↓	↑
Na^+ concentration	↓	↑	↓	↑

51 CILIOPATHIES / CHRONIC TUBULOINTERSTITIAL DISORDERS

CILIA

- Located in
  - PCT
  - DCT
  - Collecting Duct
- Not present in Loop of Henle as its primary function is reabsorption of water
- Motile cilia: Lungs, Embryonic Development Affected in Kartagener syndrome
- Non-motile cilia: Collecting Duct of kidney, Retinal pigmentary Epithelium (RPE)

Ciliopathy

1. ADPKD
2. ARPKD
3. Medullary sponge kidney
4. ADTKD (T: Tubulointerstitial kidney) akaMCKD (Medullary cystic kidney disease)
5. Nephronophthisis

Gross specimen of kidney

Refer Table 51.1

Medullary sponge kidney

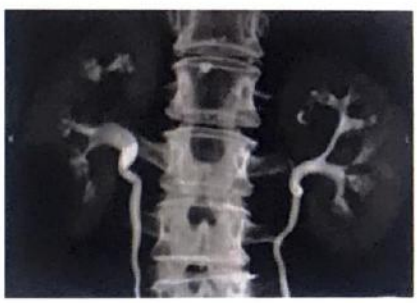
- Sporadic
- Developmental Malformation of cilia
- Cystic Dilatation of collecting Duct (CD)
- Incidental diagnosis: 50 - 60 years
- Clinical Presentation
  - Renal stones: Calcium oxalate (Recurrent stones)
  - Renal UTI/ Pyelonephritis/ Tenderness at costovertebral junction
  - Hematuria:jagged stones
  - Polyuria: Tubules and collecting Duct are defective
  - Erythropoietin ↓:Since peritubular cell affected, therefore Anemia can also be present
  - RTA Type 1:DCT affected

Simple cyst

Important Information

- Anemia also seen in CKD viz Both the glomerulus & Tubules are affected

Investigation



1. IVP
   - Paint Brush appearance aka/ bouquet flowers / aka papillary blush
   - IVP has been discontinued due to risk of Anaphylaxis
2. On CT scan: Papillary calcification.

Important Information

- Papillary calcification should not be confused with papillary necrosis Papillary necrosis seen with
  - DM
  - Analgesic Nephropathy
  - Sickle cell anemia

ARPKD

- PKHD 1 gene affected: Chr. 6
- Protein affected : Fibrocystin / Polyductin. Leads to Distal and collecting duct cyst
- Clinical features
  - Can be diagnosed as early as 24week of Gestation, Antenatal USG shows Echogenic kidney
  - Oligohydramnios: Kidney is malfunctioning
  - Pulmonary Hypoplasia: Leading cause of death in neonate.
  - At 1 years: Renomegaly / HTN and by 20 years of age ESRD
- Cause of death
  - In neonates: pulmonary hypoplasia
  - Leading cause of death in infant: HTN
- Extra Renal manifestation of Autosomal Recessive PKD
  - Congenital hepatic periportal fibrosis (MC)
    → Portal HTN
    → Esophageal varices
    → Hematemesis
  - Biliary Dysgenesis
  - Caroli Cyst: Aka Type 5 choledochal cyst
- Work up
  - USG/CT: Echogenic kidney with loss of cortico medullary differentiation
- Treatment
  - ACE Inhibitors: For HTN
  - For complications
    → Hyperkalemia: Patiromer / SPS (Resin)
    → If disease progress to ESRD, perform allogenic kidney transplantation

Extra Renal Manifestation of Autosomal Dominant PKD

- Includes
  - Hepatic cyst
  - Pancreatic/ Splenic/ Arachnoid cyst
  - MVP (Mitral valve prolapse)
  - Berry Aneurysm
  - Colonic Diverticulosis
- But pulmonary cysts are not generally encountered

ADTKD (Autosomal Dominant Tubulointerstitial Kidney Disease)

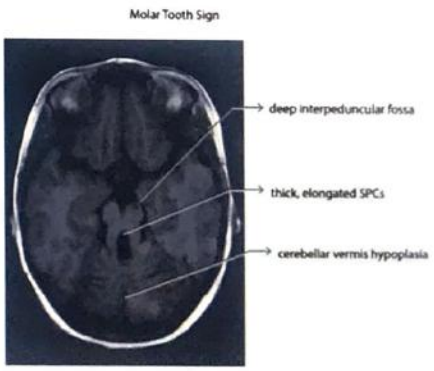
- Aka Medullary Cystic Kidney Disease
- Gene affected
  - MCKD1 Genes: Protein affected MUCIN 1
  - MCKD 2 Genes: Protein affected UMOD (Uromodulin) aka Tamm Horsfall proteins
    → These proteins are synthesis by cells of Ascending limb of loop of Henle
    → Then excreted in form of cast (Hyaline cast)
    → In these condition Tamm Horsfall proteins are not excreted and gets accumulate in Tubulo-Interstitial cells
- Always have adult presentation

ADTKD	Nephronophthisis
• Medullary cystic kidney disease • MCKD 1 → Mucin 1 • MCKD 2 → UMOD (Uromodulin aka Tamm Horsfall proteins) • Adult	• NPHP 1 gene: Ch 2 • NPHP 2 gene: Ch 9 • Juvenile / child

- Salt wasting, polyuria, Anemia, Failure to thrive, Stunting Features not seen
- Hematuria – Hallmark feature of glomerular disease

- ↑ Uric Acid

- Associated with Joubert syndrome
- C/F: Agenesis of vermis of cerebellum
- Radiological Finding
  - Molar tooth appearance
  - Cerebellar vermis Hypoplasia.
    Bat wing on CT Head

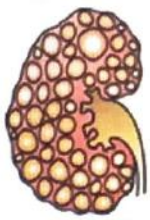


Important Information

- Bat wing CXR: Acute Decompensated CHF
- Bat wing CT head: Joubert Syndrome
- NPHS 1 Gene: Finnish variety of nephrotic syndrome (Nephrin protein)
- NPHS 2 gene: Podocin protein

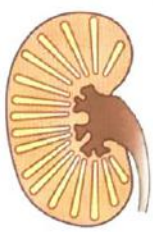
Table 51.1

1. ADPKD
   - Big cyst where whole kidney parenchyma is replaced by cyst & complete lack of cortico medullary differentiation
   - ADPKD (Usually diagnosed in b/w 30-50 years



Autosomal dominant polycystic disease

2. ARPKD
   - Renomegaly with radial striationspresents across the parenchyma of kidney.

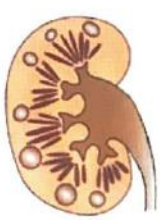


Autosomal recessive polycystic disease

3. Medullary sponge kidney
   - Cysts present in medulla with characteristic paint brush appearance

Medullary sponge kidney

4. Medullary cystic kidney disease
   - Cyst presentin cortico medullary junction aka (ADTKD)



Medullary cystic disease complex

5. Nephronophthisis: Pediatric version of ADTKD

52 POLYCYSTIC KIDNEY DISEASE

- Genes affected
  - PKD1 gene (Ch 16): Polycystin 1 protein affected
  - PKD2 gene (Ch 4): Polycystin 2 protein affected
- Multiple cysts present both in medulla and cortex.
- Renomegaly and progressive development of cyst occurs
- Development of bleeding in cyst can occur and which can cause flank pain.
- High penetrance, variable expression (expressed in every generation but presentation will be variable)
- Radial striations seen with ARPKD
- Usual age of presentation = 30 – 45 yrs
- Hematuria is not a feature of ADPKD

Clinical features
- Flank pain (M/C)
- UTI: Higher chance of pyelonephritis (Tenderness in Costovertebral junction)
- Stones incidence ↑
  - M/C stone in polycystic kidney: Urate stones
  - M/C kidney stone otherwise: Oxalate stones
- Can develop RCC: B/L, multicentric (Severity is more)
- GFR ↓ as a result Renin concentration ↑ causing retention of Na → HTN, resulting in daily Headache

Extra renal manifestation (ADPKD)
- Liver cyst: M/C extra renal manifestation
- Pancreas cyst
- Spleen cyst
- CNS
  - Arachnoid cyst
  - Berry aneurysm / A-V malformation
  - Arterial dolichoectasia: Malformation in vascular supply of brain, predisposes patient to arterial dissection and can cause Stroke manifestation

Important Information
- Leading cause of death in ADPKD: Cardiovascular mortality (HTN related complications)

- CVS
  - Mitral valve prolapse: Mid systolic clicks, late systolic murmur
  - Mitral regurgitation
  - Tricuspid regurgitation: Pansystolic murmur
- GIT: Colonic diverticulosis (Can present as constipation)

Work up of patient of ADPKD
1. KFT
2. Serum electrolyte
3. CT abdomen / T_2 Weighted imaging in MRI

Important Information
- Contrast study done only after kidney function test as contrast induced nephropathy can occur.
- D.O.C for Contrast induced nephropathy: – Acetyl cysteine and supportive therapy with IV fluids

Important Information
- To diagnose ADPKD
  - 2 cysts/kidney Age group 30-59 years
  - > 4 cysts/kidney after age 60yr

4. Genetic linkage study: best way for diagnosis, to identify gene involved

Treatment
- Control hypertension: ACE inhibitors/ARB's
  - Target BP to be maintained in ADPKD: < 140/90

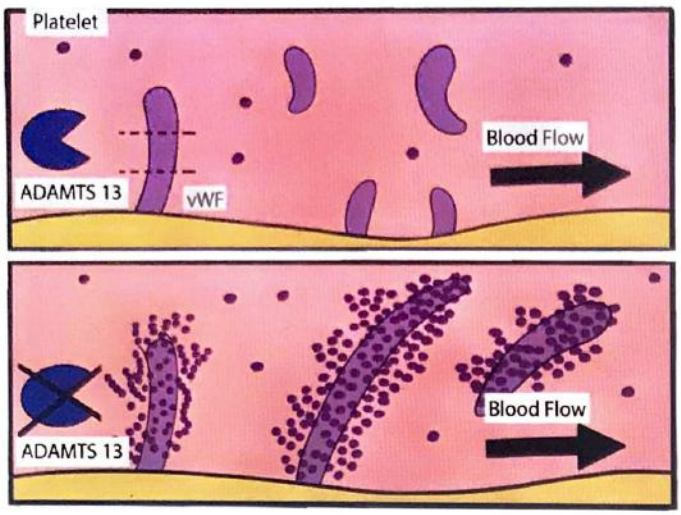
- Target BP in CKD: <130/80
- Hyperkalemia in Grade 4 and Grade 5 CKD: PATIROMER/SPS
- Lipid soluble antibiotics: Cotrimoxazole, Quinolones
- Analgesics: Tramadol
  - There is no role of Gabapentin and Pregabalin.
- Transcutaneous electrical nerve pacing in done to reduce pain
- Allogenic Kidney transplant with pretransplant nephrectomy
- Sirolimus (mTOR inhibitor): Inhibits cell proliferation and can reduce progression of cyst.
- Tolvaptan (V2 ® antagonist) / Octreotide (Long acting): Slows the decline of GFR

Important Information

- Diagnosis of diverticulosis: Ba enema
- Diagnosis of diverticulitis: CT abdomen
- Diagnosis of Berry Aneurysm: MRA
- Diagnosis of MVP/MR/TR: Echo

53 THROMBOTIC THROMBOCYTOPENIC PURPURA

Thrombotic Thrombocytopenic Purpura



ADAM TS – 13
(A disintegrin and metalloproteinase with thrombospondin motif 13) 00:00:15

- Metalloproteinase enzyme
- Function: Disintegration of the ultra large multimers of VWF (Von Willebrand Factor)
- Absence of ADAM TS-13 or Decreased ADAM TS-13 leads to
  - Microthrombi Formation → lead to consumption of platelet → Low platelet count leads to ↑ bleeding time → Palpable purpura → Stroke
  - Micro Angiopathic Hemolytic Anemia (M.A.H.A)
  - Consumption of Platelets

Causes of TTP 00:06:52

a. Primary/ Idiopathic: Antibodies which increases clearance of ADAM TS-13
b. Secondary
- Anticancer drug: Mitomycin – C, Gemcitabine
- Immunomodulator
  - Cyclosporine
  - Interferons
  - Sirolimus, Everolimus
- Antiplatelet drug: Clopidogrel, Quinine
- Anti VEGF drug: Bevacizumab (used in diabetic retinopathy)

c. Congenital Absence of ADAM TS-13
- AKA Upshaw Shulman disease
- Treatment: Multiple Plasma infusion

d. Pancreatitis, Malignancy: Neutrophils produce peptides which inhibits activity of [ADAM TS-13]

Clinical features 00:11:26

- **F** – FEVER (never presents in H.U.S)
- **A** – Anemia (M.A.H.A)
  - Bilirubin ↑↑
  - LDH ↑↑
  - Reticulocyte ↑↑
  - Coombs' Negative
- **T** - Thrombocytopenia
- **N** - Neurological features
  - Acute Ischemic stroke
  - Aphasia
  - Hemiplegia
- **R** - Renal failure
- **I** - x

How to remember

- FAT – NRI

Work up 00:14:43

- PT: N/↑ (mild); aPTT: N/↑ (mild) because clotting factors are consumed
- PBF shows Schistocytes
- Fibrinogen: Normal (reduced in DIC)
- FDP - ↑↑ (Fibrin Degradation products), D – Dimer Assay ↑↑
- Reticulocyte count ↑↑
- Comb's Negative Hemolytic Anemia
- ↑↑ LDH
- NCCT head in Acute Ischemic stroke shows
  - Hyperdense MCA
  - Loss of grey and white matter differentiation at Basal ganglia
  - Hypodensity

Treatment 00:19:00

- Plasmapheresis (TOC)
  - Helps in clearing Antibodies

- x 5 times ~ 10 days
- Upshaw Shulman disease: Cooled Plasma Transfusions
- Rituximab: Anti CD-20 (immunomodulator)
- Caplacizumab
- Hemodialysis: in complications of Uremia

H.U.S (Hemolytic Uremic syndrome) (D'HUS) 00:21:23

- Etiology
  - Shiga Toxin producing E. Coli (STEC)
  - E. Coli 0157:H7: aka EHEC
  - Verocytotoxin / Shiga Toxin
  - It causes complement activation resulting in RBC damage
- Clinical features
  - Child with Gastroenteritis like symptoms
  - Nausea
  - Vomiting
  - Abdominal pain
  - Cramping
  - Fever absent
  - After 7 days
    - → Pallor ++,
    - → Scantly Urine output
    - → M.A.H.A
    - → Acute Kidney injury
  - CNS manifestation: Seizures, Encephalopathy, Agraphia
  - Platelets ↓↓ (Thrombocytopenia)

Important Information

- HSP (Henoch Schoenlein purpura) - **Nonthrombocytopenic Purpura (Extensor purpura)**

- Pneumococcus can produce the enzyme Neuraminidase which can trigger complement activation leading to
  - M.A.H.A (Micro angiopathic hemolytic anemia)
  - A.K.I (Acute Kidney Injury)
- Antibiotics should not be given in E. coli but in Pneumococcus, antibiotics should be given.

Atypical H.U.S 00:28:08

- Due to Complement system dysregulation
- Factor - H deficiency

Treatment of HUS

- Plasma exchange: Not effective in STEC (Shiga toxin producing E. Coli)
- Stop Antibiotics
- Plasma Infusion/ Ecluzimab
- Packed RBC
- Hemodialysis ±

HUS vs TTP 00:30:07

H.U.S	T.T.P
• Child	• Adult
• H/O G.I illness and after 7 days o MAHA (Pallor) o A.K.I o Thrombocytopenia	• Headaches for days/ weeks / months, after which development of o MAHA o A.K.I o Thrombocytopenia o STROKE
• Rx o Supportive care o PRBC o Hemodialysis	• Rx: Plasmapheresis

Refer Table 53.1

HUS vs RVT 00:35:40

HUS (Hemolytic uremic syndrome)	R.V.T (Renal vein thrombosis)
• Begins with G.I illness → A.K.I	• Begins with G.I. Illness → A.K.I (Acute kidney injury)
• USG: B/L Kidney Size (n)	• USG: U/L enlarged Kidney
• SCHISTOCYTES present	• Painful enlarged Kidney
• Decreased platelets	

Table 53.1

ITP (Epistaxis, Petechiae)	HSP (Extensor purpura)	TTP (Headache, AIS)	DIC (Septic patient)
• Autoimmune • BT ↑ • PT (n) • aPTT (n) • Rx: Steroids and splenectomy	• IgA + • BT (n) • PT (n) • aPTT (n)	• ADAM TS 13 ↓ • BT ↑ • PT ↑ • aPTT ↑ • Schistocytes • Fibrinogen (n)	• Sepsis • BT ↑ • PT ↑ • aPTT ↑ • Schistocytes • Fibrinogen Reduced

54 URINE ANALYSIS

PROTEINURIA

00:00:20

Refer Image 54.1

- Dipstick test
  - Very good for negatively charge proteins (Albumin) but not good in picking up positively charged proteins aka Paraprotiens
  - As a result, in condition like multiple myeloma the dipstick can be false negative
- Proteins in urine
  - Protein present in urine of a normal individual: <150mg/day
  - Proteins can come out through Glomerulus or the tubule
    - → Through Glomerulus: Albumin (<30mg/day)
    - → Through Tubules: Tamm Horsfall proteins/ Uromodulin (120mg/day)
- Estimation of Albumin in spot sample
  - Albumin excretion rate = $\frac{\text{Urine albumin}}{\text{urine creatinin}}$
  - Albumin excretion rate can be expressed as
    - → Milligram per day (mg/ day)
    - → Milligram per gram of urinary creatinine (mg/gm of urinary creatinine)

Moderately increased Albuminuria

00:04:02

- Albumin excretion rate (AER) /Urinary albumin creatinine (UAC) ratio is 30-300 mg/ gm = Moderately ↑ albuminuria (earlier known as Microalbuminuria)
- Has an ↑sed risk for cardiovascular mortality
- Causes for moderate ↑ albuminuria
  - Diabetic nephropathy
  - Hypertension
  - Glomerulonephritis

Severely increased Albuminuria

- Albumin excretion rate (AER) /Urinary albumin creatinine (UAC) ratio is 300 mg–3500 mg/gm = Severely ↑ albuminuria (earlier known as Macroalbuminuria)
- Increased cardiovascular mortality
- Causes for severely ↑ albuminuria
  - Diabetes Mellitus
  - HTN
  - Glomerulonephritis
  - Multiple myeloma/ Para- proteinuria
  - Congestive Heart Failure
  - Fever
  - Exercise
  - Orthostatic proteinuria

Overload proteinuria

00:11:52

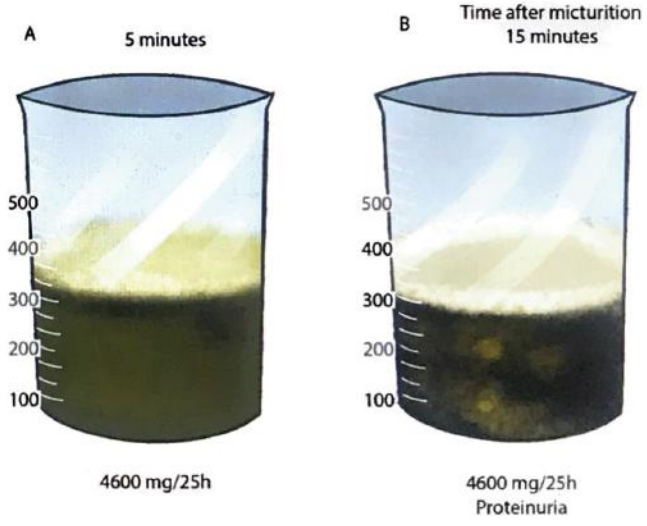
- Causes
  - Multiple myeloma / MGUS
  - Rhabdomyolysis
  - Hemoglobinuria

Functional proteinuria

- Causes
  - Jogging
  - Exercise

Nephrotic range proteinuria

- Albumin excretion rate (AER) /Urinary albumin creatinine (UAC) ratio is >3500 mg /gm = Nephrotic Range proteinuria
- Causes for nephrotic range proteinuria
  - Diabetic Nephropathy
  - Amyloidosis
  - Minimal change disease
  - Focal segmental glomerulosclerosis (FSGS)
  - Membrano-glomerulonephritis (MGN)



- Urine sample
  - First morning sample is foamy, and the foam does not settle
  - Urine turns yellow on air exposure (component: urochromes)
- Screening Test: Urine Electrophoresis

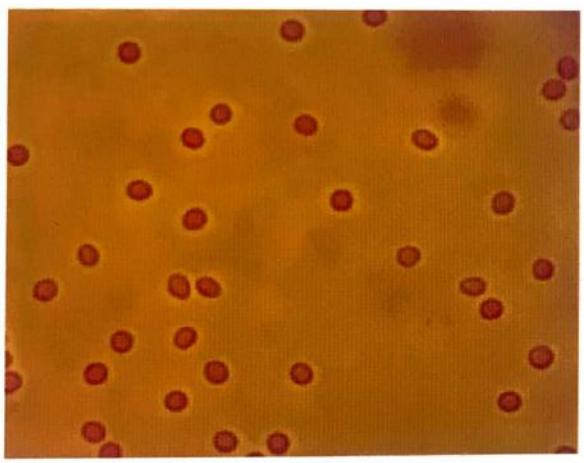
Important Information

- **CSF electrophoresis is done in Multiple Sclerosis**
- **Oligoclonal Ig G bands are present in CSF of patients of multiple sclerosis**

Dipstick test	
Trace	15-30 mg/dl
1+	30-100 mg/dl
2+	100-300 mg/dl
3+	300-1000 mg/dl
4+	>1000 mg/dl

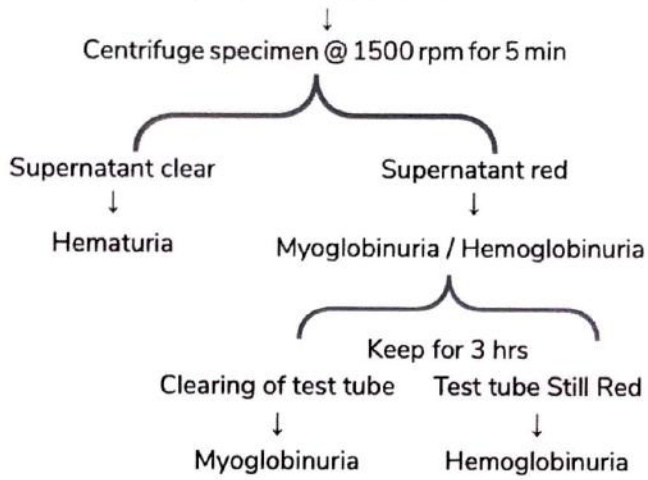
HEMATURIA

00:20:47



- RBCs are also present in urine of normal person
- Significant / Persistent hematuria
  - > 5 RBC/ HPF centrifuged specimen x 3 times; at an interval of 1 week
  - > 100 RBC/HPF in single sample
- Dysmorphic RBCs: Glomerular bleed
- Causes of gross hematuria: Urological (90%) > Renal parenchymal (9%) > Hemolysis (1%)
- Causes of Microscopic hematuria: IgA nephropathy
- Causes of red urine
  - Clofazimine
  - Porphyrins

- Red urine tested positive for dipstick test

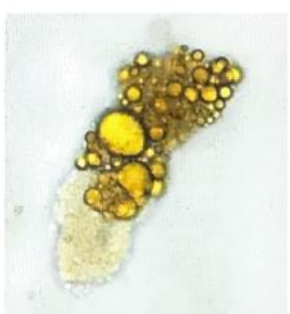


- Hematuria + Pyuria/ Bacteriuria: UTI
- Hematuria + RBC cast / dysmorphic RBC + Proteinuria: Glomerulonephritis
- Schistosomiasis haematobium
  - Causes hematuria & fever (known as Katayama fever)
  - Eosinophilia in blood
  - Its egg has terminal spine

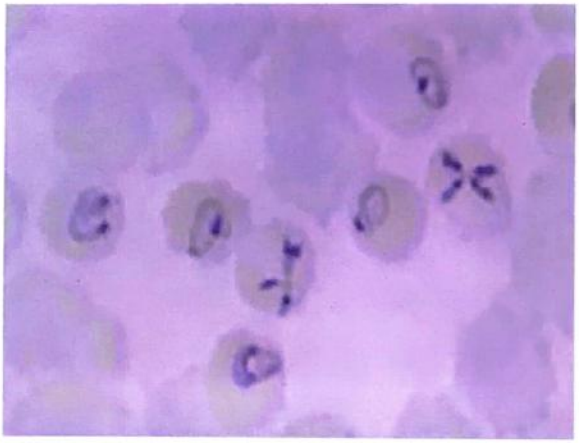
EOSINOPHILURIA

00:34:14

- Causes
  - Atheroembolic kidney disease: (Coronary Angiography → Damage to atherosclerotic plaque → Fragments trapped in Renal Artery → AKI)
  - Allergic interstitial nephritis (AKI 2° to antibiotic Intake)
- Stain used to evaluate eosinophiluria = Wright/ Hansel's stain



- Fats in urine / Oval fat bodies found in
  - Nephrotic syndrome
  - Fat embolism syndrome
  - Chyluria
- Maltese cross appearance
  - In Urine
    - → Nephrotic syndrome
    - → Fabry's disease
      - GLA gene involved
      - Lysosomal storage disorder
      - Enzyme defective is alpha Galactosidase A
  - In Peripheral smear: Babesia microti (Vector is Ixodes)
  - In CSF: Cryptococcal neoformans



Peripheral smear

Polarized microscopy

Specific gravity of urine 00:44:51

- Normal: 1.020 – 1.030
- Isosthenuria: Inability to concentrate the urine
  - Cause
    - → Chronic tubulointerstitial disease
    - → Sickle cell anemia
- Sp. Gravity helps in differentiating Pre-renal vs. Renal cause of AKI
- Pre-renal AKI the Specific gravity ↑ (normal), while in Renal AKI the specific gravity ↓ (Urine cannot be concentrated due to tubular damage).

PH of urine

- Normal pH of urine: 4-4.5

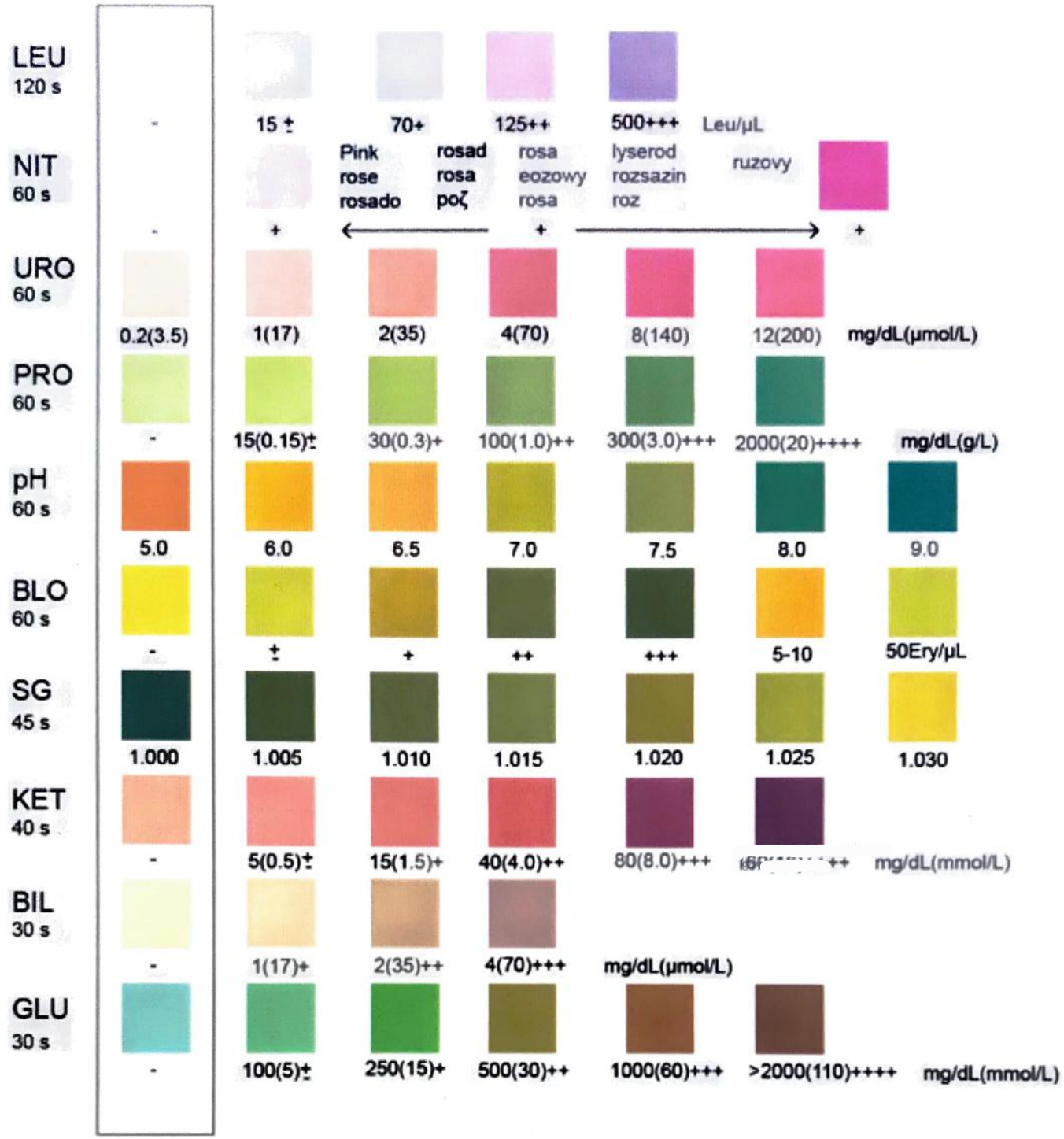
If pH > 5.5	
PCT	**DCT**
• Damage to PCT causes HCO_3^- loss k/a Bicarbonaturia as a result the PH will be increased k/a RTA type 2	• Damage to DCT causes Inability to acidify the urine k/a RTA type1

Color of urine

- Pink urine= Deferoxamine
- Pink diaper sign = Caused by Serratia Mascarenes
- Red urine = Clofazimine / Porphyria / Myoglobinuria
- Cola colour = Hematuria
- Black = Hemoglobinuria (PNH/PCH)
- Black on air exposure = Alkaptonuria
  - Prevention by vitamin C

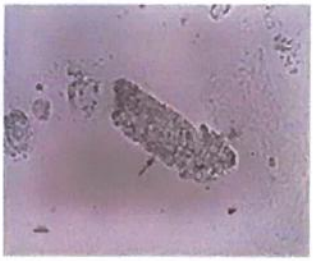
Refer Table 54.1

Image 54.1

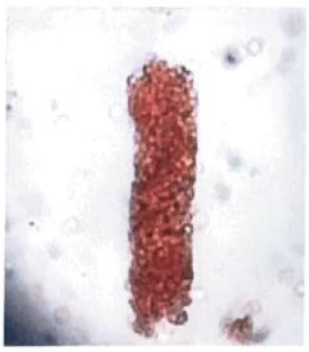

LEU
120 s
-
15 ±
70+
125++
500+++
Leu/µL
NIT
60 s
-
+
Pink
rose
rosado
rosad
rosa
poζ
rosa
eozowy
rosa
lyserod
rozsazin
roz
ruzovy
+
+
URO
60 s
0.2(3.5)
1(17)
2(35)
4(70)
8(140)
12(200)
mg/dL(µmol/L)
PRO
60 s
-
15(0.15)±
30(0.3)+
100(1.0)++
300(3.0)+++
2000(20)++++
mg/dL(g/L)
pH
60 s
5.0
6.0
6.5
7.0
7.5
8.0
9.0
BLO
60 s
-
±
+
++
+++
5-10
50Ery/µL
SG
45 s
1.000
1.005
1.010
1.015
1.020
1.025
1.030
KET
40 s
-
5(0.5)±
15(1.5)+
40(4.0)++
80(8.0)+++
++
mg/dL(mmol/L)
BIL
30 s
-
1(17)+
2(35)++
4(70)+++
mg/dL(µmol/L)
GLU
30 s
-
100(5)±
250(15)+
500(30)++
1000(60)+++
>2000(110)++++
mg/dL(mmol/L)

CASTS

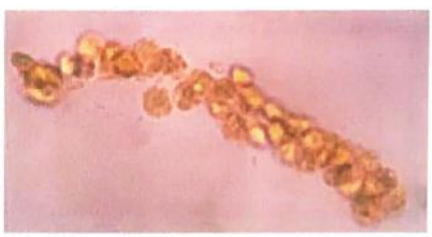
- Hyaline cast are seen normally
  - Composed of Tamm Horsfall Protein



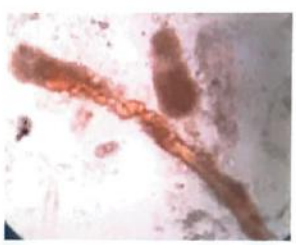
- RBC cast: Acute glomerulonephritis



- WBC cast
  - Acute pyelonephritis
  - Acute interstitial nephritis



- Muddy brown cast: Acute tubular necrosis



- Broad cast: (D. nephropathy) CKD

- Granular waxy cast: CGN

55 ACUTE KIDNEY INJURY

Stages	S. Creatinine	Urine Output	Time
I	• 1.5 – 1.9 times of normal	• <0.5 ml/kg/hr	• Over 6 hr
II	• 2-2.9 times of normal	• <0.5 ml/kg/hr	• Over 12 hrs
III	• ≥3 times of normal or S. creatinine > 4mg %	• <0.3 ml/kg/hr	• Over 24 hrs

Biomarkers of acute kidney injury

1. NGAL = Neutrophil gelatinase associated lipocalcin
2. K.I.M-1 = Kidney injury molecule – 1 (Best)
3. IL-18
   - All these parameters are checked in urine

ETIOLOGY OF A.K.I

1. Pre- renal cause: Most common cause

a. Hypovolemia

b. Congestive heart failure

c. Ascites (massive) → circulating fluid volume decreases
   - In a cirrhotic pt. Hepatorenal Syndrome can develop.
   - It is of two types
     - HRS-1
       - → Pt. deteriorates in as early as 2 weeks
       - → ↑↑ s. creatinine >2.5 mg%
       - → Normal kidney perfusion ensured by giving IV fluids and stop diuretics, but creatinine continues to rise in HRS 1
     - HRS-2
       - → In case of refractory ascites

d. Drugs
   - NSAIDS: as PGs are not produced and Afferent arteriolar dilatation does not occur
   - ACE inhibitors in B/L renal artery stenosis pt.
   - Cyclosporine: acts through altering intrarenal hemodynamics.

Important Information

- Filtration gradient at glomerulus is maintained by.
  - Vasodilation at afferent Arteriole → NSAIDs.
  - Vaso Constriction at efferent arteriole → ACE ⊖

2. Renal cause

a. Glomerulus

b. Tubules

c. Vascular

a. **Glomerular cause**
   - Acute glomerulonephritis
   - Post streptococcal GN
   - S.L.E
   - Good pasture syndrome/ Anti Glomerular Basement Membrane Ds.
   - Vasculitis related to ANCA

b. **Vascular causes**
   - Hemolytic Uremic Syndrome
   - Thrombotic Thrombocytopenic Purpura
   - Malignant HTN
   - Renal vein thrombosis
   - Athero-embolic kidney disease: Eosinophiluria is present.

c. **Tubular cause**

i. Sepsis: Toxins can cause tubular necrosis

ii. Ischemia
   - 20% of cardiac output goes to kidney
   - 10% of O_2 consumption is by kidney but weight of kidney is only 0.5%
   - Outer medulla is most vulnerable (S_3 Segment of PCT)
   - Causes of Renal Ischemia
     - Post-operative renal ischemia due to prolonged anaesthesia.
     - Post cardiac surgery → Cardiopulmonary bypass → Non pulsatile blood flow to kidney → Kidney ischemia (Normally Kidneys have pulsatile blood flow that ensures adequate blood supply to cortex

and medulla)
- Post-operative Atheroembolic disease
- Burns & acute pancreatitis → Massive fluid resuscitation → Due to leaky capillaries → Massive ascites → Abdominal compartment syndrome (> 20 cm H_2O) → Renal vein compression & ischemic damage to kidney

iii. Nephrotoxins: Renal variety of AKI

Exogenous Endogenous

- Exogenous Causes
  - Aristolochic acid (Chinese herbal medicine): responsible for Balkan nephropathy
  - Aminoglycoside toxicity
    - → Non oliguric A.K.I (Urine output > 400 ml/day)
    - → Aminoglycoside damages V_2 receptor in collecting duct
  - Amphotericin B
  - Cisplatin
  - Contrast induced nephropathy:
    - → Recovery occurs within 7 days
    - → Antidote: N acetyl cysteine
- Endogenous cause
  - Hemolysis (Mismatched BT → Hemolysis → Hemoglobinuria → Block Kidney)
  - Rhabdomyolysis (Crush Injury → Myoglobinuria → ATN)
  - Multiple myeloma (Bence jones Proteins → Tubules → ATN)
  - Crystals
    - → E.g., If a person consumes antifreeze agent/ ethylene glycol poisoning → Production of Ca-oxalate crystals → Blocks tubules of kidney
      - Antidote: Fomepizole
      - Fomepizole is also used in Methyl Alcohol poisoning
    - → Uric acid crystals: Chemotherapy in Cancer →Anaplasia (↑↑ Nucleus: Cytoplasm ratio) → More amount of DNA → DNA breaks to produce uric acid → Uric acid crystal causes blockage of renal tubules
    - → Tumor lysis syndrome: When chemotherapy setting is started in case of malignancy which leads to
      - K^+↑
      - PO_4↑
      - Calcium↓
      - DNA damage = ↑ uric acid crystal → Renal shutdown
    - → Management
      - Chemotherapy should always be started in morning hours so that monitoring of pt. can be done
      - For prophylaxis: Adequate hydration (N.S Drip)
    - → Treatment of Tumor Lysis Syndrome
      - I.V Fluid + Furosemide Drip + IV Allopurinol
      - IV Rasburicase
      - Urine alkalinization is removed from treatment

Vascular cause responsible for AKI

i. Renal vein thrombosis
- AKI + U/L enlarged painful kidney (Hydronephrosis have enlarged but not painful kidney)
- I.O.C = Doppler USG
- Complication: Pulmonary embolism
- Etiology
  - In child: Diarrhea or Dehydration → increased viscosity & sluggish circulation
  - In adult: Membranous GN

ii. Crush injury: Red urine (Myoglobin blocks tubules and causes renal shutdown)

Important Information

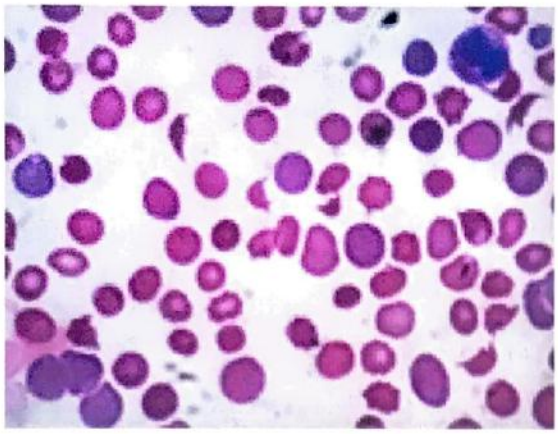
- **Other causes of red urine**
  - **Porphyria**
  - **Beet root**
  - **Crush injury**
  - **Urological cause**

iii. P. falciparum malaria → Hemolysis: Hemoglobinuria → blocks the renal tubules → Black water fever

iv. Athero-embolic kidney disease
- During angiography, guide wire can cause damage to atherosclerotic plaque and multiple small embolic fragments develop which can cause blockage of small renal vessel and cause ischemia of kidney
- Post angiography Acute Kidney Injury
  - Eosinophilia
  - S. creatinine levels
    - → Pre procedure = normal
    - → Post procedure = ↑↑↑
  - Prognosis: bad
- Livedo reticularis (Fish net appearance of skin) and retinal plaques are also seen in Athero-embolic kidney disease

d. Hemolytic Uremic Syndrome (H.U.S)
- Caused by E. coli 0157: H7
- Shiga toxin
- Features
  - Complement mediated vasculitis

- M.A.H.A (microangiopathic hemolytic anemia) / Coombs negative hemolytic anemia
- Uremic manifestations like nausea, vomiting, encephalopathy or uremic pericarditis
- Peripheral smear: Helmet Cells / Schistocytes



Important Information

Case scenario
Pt. had diarrhea/ slight dysentery for which he was managed conservatively, after a week pt. develops severe hemolysis

- Dx
  - Typical HUS or D⁺ HUS
  - Atypical HUS/D⁻ H.U.S • due to drugs

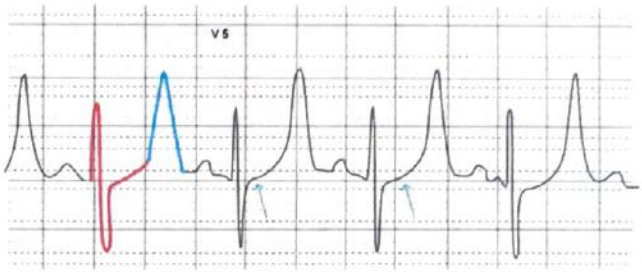
Important Information

- Do not give antibiotics in HUS because antibiotics can worsen the ds. and increase mortality by as much as 17 times.

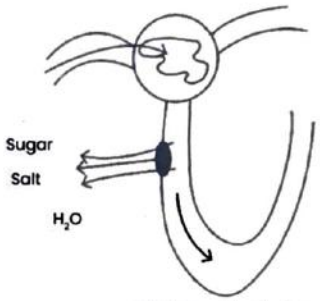
3. Post Renal causes
- B/L stones at pelvi-ureteric junction
- BPH
- Bladder outlet obstruction d/t stones or tumor
- Blocked foley catheter
- B/L ureteric fibrosis could be seen in scleroderma or with the use of bleomycin

Clinical Features of A.K.I
- Urea ↑↑
  - Irritation of stomach mucosa causes severe vomiting
  - Irritate diaphragm causes Hiccups
  - ↑↑Urea → Pericarditis - Chest pain
  - Urea → Cross Blood Brain Barrier → Draws water →Cerebral edema →Encephalopathy and develops asterixis (flapping tremors)
- GFR ↓: Renin ↑ = Hypertensive encephalopathy
- Volume overload
  - Puffy eyes
  - Pulmonary edema (dyspnea)
- Renal shut down = Anuria (U. output < 100ml/day)

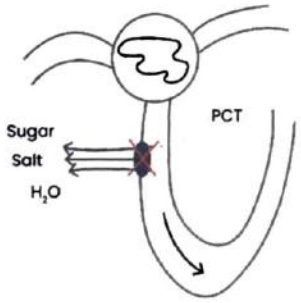
Work up
- Kidney function test: S. creatinine ↑↑
- Electrolytes = Na^+ ↓ and K^+ ↑ (hypervolemic hyponatremia)
- ECG
  - Tall, tented T waves
  - ST elevation



- USG of kidney = To rule-out post renal causes
- A.B.G = Metabolic acidosis pH < 7.35, HCO_3^- <22
- Fe Na (Fractional excretion of Na^+) differentiate b/w Pre-renal & Renal AKI.



Cholera : dehydration
Urinary Na⁺:Low



Tubular # :Salt wasting
Urinary Na+ : Low

Cholera	Tubular damage
• Dehydration	• Salt wasting
• Urinary Na^+ = low (↓ Flow in PCT → ↓ Urinary Na^+)	• Urinary Na^+ = High (Tubular Damage → ↑ Concentration of Urine)
• Fe Na <1% (fractional excretion of Na^+)	• Fe Na >1%
• Pre-Renal	• Renal
• Urine osmolality = High (>500 mosm)	• Urine osmolality =low (300 mosm)

Management of A.K.I

1. Input fluids = urine output last 24 hr + Insensible losses 400 – 800 ml / day
   - Insensible losses are in the form of
     - Water vapor during breathing
     - Stool
   - Input fluid: 10% dextrose is the fluid used
2. Furosemide drip
3. If Na+ < 125 meq → causes seizures then give 3% saline
4. If K+ > 6.5 meq → give Ca^{2+} Gluconate (antagonize K^+ effects)
   - Insulin drip regular (only this causes K^+ influx into cells): Most Effective drug to lower ↑ K^+
   - Salbutamol nebulization
   - Hemodialysis → most effective method for acute ↑ K^+
5. If pH <7.2: IV soda Bicarbonate is given
6. HTN crisis / encephalopathy: Give IV labetalol or IV fenoldopam or IV Nicardipine
7. Uremia features: Hemodialysis is done through Int. jugular vein or subclavian vein

Recovery phase of A.K.I characterized by

- Polyuria
- ↑↑ Na^+
- ↓↓ K^+

Indication for Hemodialysis

- Uremic Pericarditis
- Uremic Encephalopathy
- Uremic Malena
- HTN refractory to Medication
- Hyperkalemia

CLINICAL QUESTIONS

Q. A 38 years female presented to emergency with complains of Headache, confusion, decreased urinary output, swelling on both ankles, nausea, fatigue and shortness of breath. Urine investigations revealed: Urine osmolality: 800 mOsm/kg. Urinary sodium 10 mmol/L.BUN: creatinine=20:1. What is the most likely diagnosis?

A. Pre-renal acute renal failure
B. Acute tubular necrosis
C. Acute cortical necrosis
D. Urinary tract obstruction

Answer: A

Solution

	Pre renal	Renal (ATN)	Post renal
Urine osmolality	> 500 mOsm	~ 300 mOsm	<400 mOsm
Urine sodium	<20 mmol/dl	>20 mmol/dl	Variable
FeNa	<1	>1	Variable
Urine microscopic findings	Hyaline casts	Muddy brown casts	Normal or RBC/WBC
BUN: creatinine ratio	>20:1 (normal 10:1)	<20:1	>20:1

56 CHRONIC KIDNEY DISEASE

- CKD is characterized by a progressive nephron loss in a patient.00:02:14

CKD-EPI Equation

- GFR = 141 x [min (Scr/ κ), 1)α x max (Scr/ κ), 1) -1 209] x Age -0.993 x 1.018 [If female] x 1.157 [If Black]
- α is 0.329 for females and 0.411 for males; min indicates minimum of Scr/κ or 1, and max indicated maximum of Scr/κ or

Female	≤ 0.7 →	$GFR = 144 \times (Scr/0.7)^{-0.329}$	x Age -0.993 x 1.157 (If Black)
Male	> 0.7 →	$GFR = 144 \times (Scr/0.7)^{-1.209}$	
	≤ 0.9 →	$GFR = 141 \times (Scr/0.9)^{-0.411}$	
	> 0.9 →	$GFR = 141 \times (Scr/0.9)^{-1.209}$	

Test to determine GFR

- "CKD – EPI - Cystatin C" method (BEST) (Produced by Nucleated Cell)
- Modification of diet in renal disease (MDRD) formula.
- Cockcroft Gault formula
- Creatinine clearance
- Inulin clearance (PAH/Urea/glucose)

Cockcroft Gault Formula

- Cockcroft gault formula: $GFR = \frac{(140\text{-Age}) \times Wt}{72 \times S.Creatinine}$
- In Females, Multiply by 0.85

Important Information

- **Definition of CKD: CKD =GFR ≤ 60ml/min/1.73m² + proteinuria x 3 months**

Grading

- G1 = > 90 ml/min/1.73 m2 (Evidence of Proteinuria)
- G_2 = 60 – 89 ml/min/1.73 m^2 (HTN develops in this stage)
- G_3 = 30 – 59 ml/min/1.73 m^2 (Anemia develops in this stage)
  - From G_2 TO G_3 transition, value of PTH increases thus increasing the bone turnover but despite being PTH INCREASE the phosphate levels increases from G_3 to G_4 transition as kidneys are not working properly.
- G4 = 15- 29 ml/min/1.73 m2 (Acidosis, hyperkalemia develops in this stage)
- G_5 = < 15 ml/min/1.73 m^2 (URAEMIA develops)

Spot urine sample

$$\text{AER (Albumin Excretion Rate)} = \frac{\text{Urine Albumin (mg)}}{\text{Urine Creatinine (g)}}$$

- A_1: < 30 mg/g
- A_2: 30-300 mg/g
- A_3: ≥300 mg/g

				Albuminuria categories		
				A1	A2	A3
				normal to Mildly increased	Moderately increased	Severely increased
				<30mg/g <3 mg/mmol	30-299 mg/g 3-29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR stages	G_1	Normal or High	>90			
	G_2	Mildly to decreased	60-90			
	G_{3a}	Mildly to Moderately decreased	45-59			
	G_{3b}	Moderately to severely decreased	30-44			
	G_4	Severely decreased	15-29			
	G_5	Kidney failure	<15			

Causes of CKD

00:20:24

- Diabetic nephropathy (m/c)
- Chronic glomerulonephritis
- Ischemic nephropathy / Cardiorenal syndrome
- ADPKD (M/C cause of Death - HTN)
- Chronic tubulointerstitial disorder / Ciliopathies

Treatment

a. G_1 to G_3 CKD treatment

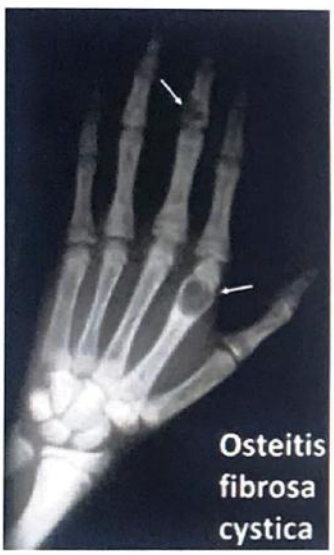
i. Keep HbA1c <7%

- Stop metformin as it is excreted by kidney.
- Metformin is contraindicated if S. creatinine >1.5 mg% or GFR < 45.
- Give insulin injection (excreted by kidney)
- Dose reduction is required to avoid increased risk of hypoglycemia as insulin is not effectively excreted (80% calculated dose)

ii. BP control
- CKD pt. have high Renin Hypertension
- Target BP < 130 / 80 mm Hg if tolerated (If Orthostatic Hypotension due to autonomic dysregulation target should be < 140/90 mmHg).
- Drug = ACE inhibitor/ ARB + CCB (calcium channel blockers)

iii. Cholesterol control
- CKD pt. have ↑ cholesterol
- Target = LDL<70 mg%
- Drugs = Statins → Atorvastatin (Always monitor CPK-mb)

iv. Low Vit. D_3
- Vit D_3 synthesis occur in PCT (by 1α Hydroxylase enzyme)
- Hypocalcemia will activate Ca sensitive receptor
- ↑ PTH (2° Hyperparathyroidism): patients will be having high bone turnover rates
- Bone pain / pathological fracture of ribs
- On X ray: Subperiosteal resorption of phalanges
- PTH causes bone marrow fibrosis → Anemia → Osteitis cystica fibrosa (Brown tumor)
- This is known as Azotemic osteodystrophy/ high bone turnover state



- Rx of high bone turnover rate
  - Increase PO_4
    - → Sevelamer (phosphate binder)
    - → Lanthanum Carbonate (Phosphate binder)
    - → Calcium Acetate
  - PTH release inhibitor = Calcimimetic drug (Cinacalcet)
- Low bone turnover rate can be due to
  - Due to more/excess calcium supplementation + vit D_3 → ↑ Ca^{2+} leads to ↓ PTH (low bone turnover)

Important Information
- 1° PTH ↑ • Parathyroid Adenoma
- 2° PTH ↑ • CKD
- 3° PTH ↑ • Untreated 2° hyperparathyroidism leading to autonomous function of parathyroid gland. (Calcium phosphate ↑ due to ↓ Excretion 2° CKD → Calcification of blood vessels & Worsening of Atherosclerosis)

v. Low Erythropoietin: Anemia (Normocytic normochromic)
- Causes of Anemia in CKD
  - ↑ PTH → Cause BM fibrosis
  - Anemia of chronic disease
  - Bleeding manifestations

- Rx of Anemia
  - Iron parenteral
  - Erythropoietin s/c or i/v, Darbepoetin, Epoetin B → S/E = Cardiovascular accident ↑, MI risk ↑

vi. K^+ ↑ - Hyperkalemia
- Sodium polystyrene sulphonate (SPS resin → K+ binders)
  - Side effect: Necrosis of GUT
- Patiromer
- Sodium zirconate cyclosilicate (Zs-9)

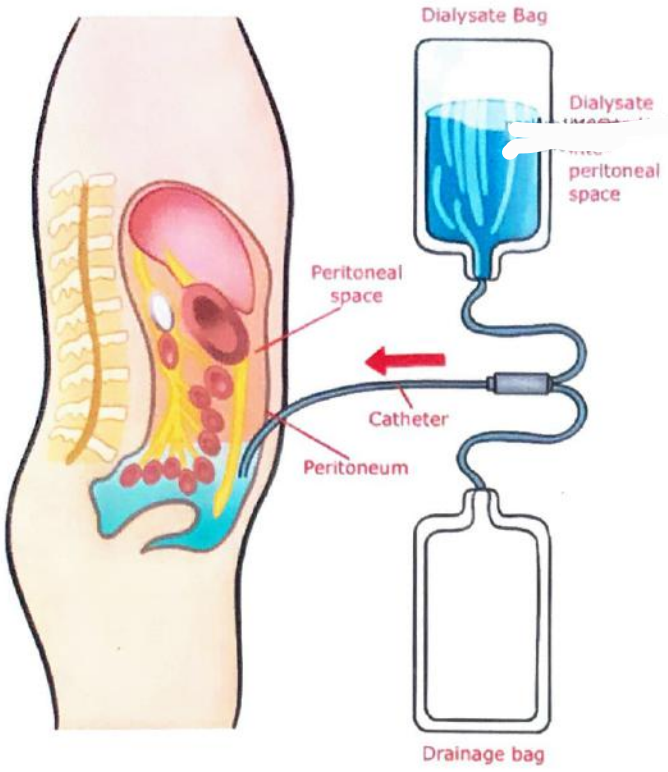
Important Information
- Treatment of G_1-G_3 CKD patients
  - A - Insulin (low dose)
  - B - ACE - / ARB + CCB (Target BP < 130/80)
  - C - Control cholesterol using Statins (LDL < 70 mg%)
  - D - PO_4 ↑ (Phosphate binders-sevelamer/ lanthanum). cinacalcet
  - E - Erythropoietin s/c or i/v
  - K - Patiromer / SPS 125-9

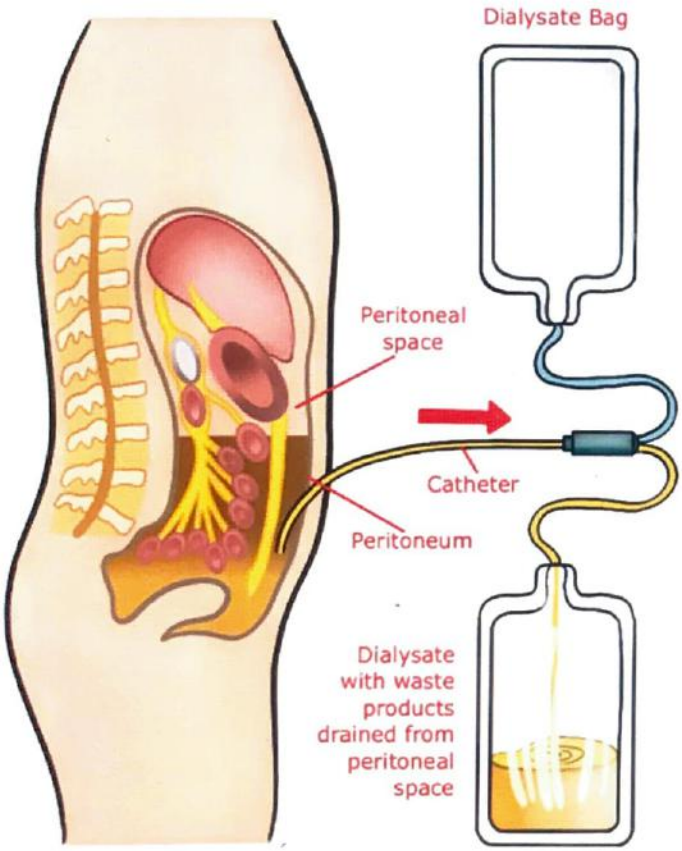
How to remember
- ABCDEK

b. $G_4 \rightarrow$ Renal replacement therapy

i. Dialysis

ii. Allogenic kidney transplantation





I. Hemodialysis

- 3 times / week
- 1 session = 4 hour
- 12 hr per week
- Peritoneal dialysis: Risk of infections (C/I ADPKD, ARPKD)

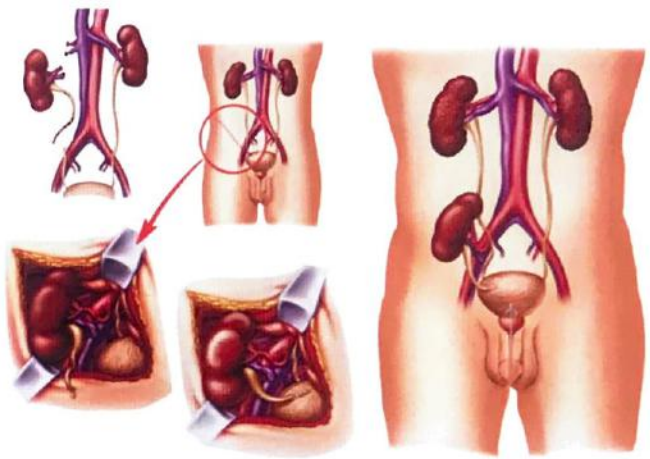
Allogenic kidney transplant

- HLA matching mandatory
- Blood group matching is also required

Important Information

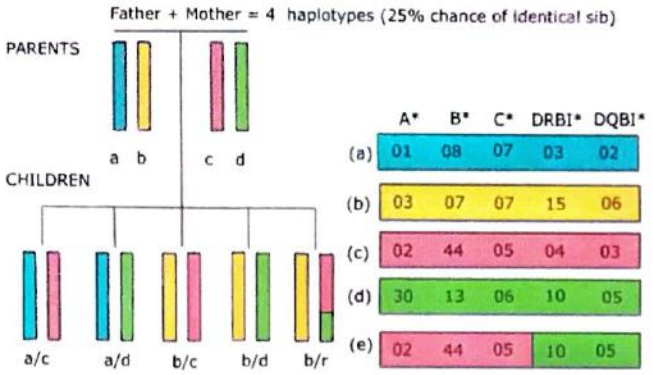
- **HLA matching not mandatory in**
  - **Liver**
  - **Cornea**
  - **Heart**

- Test for donor = HIV, CMV, Hep. B, Hep. C, Syphilis, & Doppler (to rule out U/L Renal artery stenosis)
- In Donor, left kidney is removed (because of longer left iliac vein).
- New kidney is placed in right iliac fossa because of ease of removal (In Case of rejection).



HLA inheritance

- HLA A, B, C, DP, DQ, DR
- HLA matching is done for A, B, DR for kidney transplantation
- In monozygotic twins' rejection chances are 0% as HLA coding are the same
- Syngeneic transplantations/ isograft: chances of rejection are negligible



1/6 – 2/6 Match	3/6 Match	4/6 – 5/6 Match
• Induction with anti-thymocyte globulin (A.T.G)	• Induction with Basiliximab (Anti CD 25)	• No need for induction, only maintenance therapy

Rejection

	Hypersensitivity reaction	Time
1. Hyperacute	II	Minutes
2. Acute	IV	< 6 months
3. Chronic	IV	> 6 months

Maintenance therapy

- Steroid
- Tacrolimus/ cyclosporine
- Mycophenolate
- Azathioprine
- Sirolimus (non-nephrotoxic)-mTOR inhibitor

Opportunistic infection post kidney transplantation

- <1 month: Surgical site infection (SSI) with MRSA
- 1-6 months
  - Pneumocystis jiroveci
  - Cytomegalovirus (CMV)
  - Hep B and C
- >6 month
  - Aspergillus
  - Nocardia
  - Bk virus (polyoma virus)
- CMV: m/c infection that is responsible for graft failure after kidney transplantation. / Solid organ transplantation.
- CMV: m/c infection after hematopoietic stem cell transplantation
- EBV: m/c cause of post transplantation lymphoma
- M/C malignancy post kidney transplantation: skin cancer

Hemodialysis

- Cimino – Brescia Fistula: Radial Artery – Cephalic vein is connected (Pressure created results in continuous blood flow)

↓

Bruit/ Doppler used to identify patency of fistula

↓

Arterilization of Vein

(Internal Lining of vein will change when blood flows from Artery to Vein)

↓

Thrombophlebitis incidence is reduced

(Site can be used for longer time)

- Complications of hemodialysis
  - Hypotension: M/c complication
  - Accelerated atherosclerosis: m/c cause in Recurrent hemodialysis
  - Dialysis dementia (Due to $A\beta_2$ amyloid deposition)
  - Carpal tunnel syndrome
  - Dialysis disequilibrium (Cerebral oedema): seizures
  - Peripheral neuropathy / myopathy (d/t loss of vitamins & nutrients during dialysis)
- Principles of hemodialysis
  - Diffusion (Main - Facilitated Diffusion)
  - Solvent drag (Ultra filtration): Counter Current mechanism → Blood Flows in opposite direction to the dialysate

Previous Year's Questions

Q. Which of these is the renal feed for CKD patients?
(AIIMS Nov 2019)

A. Low calorie low volume
B. Low calorie high volume
C. High calorie low volume
D. High calorie high volume

Previous Year's Questions

Q. A hypertensive patient presents with grade 4 CKD with e GFR <30mL/min. doctor wants to prescribe a thiazide diuretic. Which of the following will be the best for the patient?
(INICET July 2021)

A. Hydrochlorothiazide
B. Chlorthalidone
C. Metolazone
D. Indapamide

CLINICAL QUESTIONS

Q. A 52 yrs old plumber came with complaints of severe pain & swelling over the big toe. On examination, his BP was 165/95mmHg & also having signs of progressive renal failure. Which of the following would be the probable diagnosis?

A. Diabetic nephropathy with hyporeninemic hypoaldosteronism
B. Lead nephropathy
C. Sickle cell nephropathy
D. Aristolochic acid nephropathy

Answer: B

Solution

The triad of 'saturnine gout' + hypertension + renal failure is seen in Lead nephropathy.

Saturnine gout

Cause: Lead toxicity

Symptoms

1. Symptoms of lead toxicity **precede the development of gout.**
2. Hence **patient will have anemia**, basophilic stippling of RBC, abdominal pain, and nerve palsy
3. The **damage to the kidney will lead to hypertension** and **progressive renal malfunction.**
4. **Knee joint involvement is more common than MTP involvement** in saturnine gout

57 DIABETIC NEPHROPATHY

00:00:19

- Most common causes of C.K.D (chronic kidney disease)
- Most common indication of Kidney Transplantation
- Occurrence depends on Duration of disease and Severity of the disease
- DM (HbA_1C >7%) for > 5years in Type 1 DM and > 20 years in Type 2 DM leads to nephropathy
- B/L enlarged kidneys → Differential Diagnosis
- Other conditions where B/l enlarged kidneys are seen include

a. HIV associated Nephropathy
b. Amyloidosis
c. Diabetes Mellitus
d. Polycystic kidney disease
e. Hydronephrosis

- Painful unilateral enlarged kidney with Acute kidney Injury: Renal vein Thrombosis
- Screening: Albumin Excretion Rate or Urine Albumin creatinine Ratio
  - In Spot urine sample we check for Urinary Albumin Creatinine Ratio

$$\text{Spot sample} = \frac{\text{Albumin (mg)}}{\text{Urinary Creatinine (gm)}}$$

  - 30-300 mg/gm (moderately ↑ albuminuria): ↑ cardiovascular mortality
  - Serum creatinine may be Normal
    → Rises late (After 60% of kidney damaged)
    → Most specific: Kidney function test

- RX: Low dose ACE inhibitors or ARB can reduce the progression of disease

Histopathological aspect of Diabetic nephropathy

00:13:01

1. Diffused glomerular sclerosis: M/c histopathological finding seen in a patient of Diabetic nephropathy
2. Nodular glomerulosclerosis: Kimmelstiel Wilson Change
3. Armani Ebstein change: PCT
4. Damage to DCT leads to development of Type – 4 RTA (Renal tubular acidosis)

- In RTA 4, Aldosterone Resistance > Aldosterone deficiency
- Thus ENaC (Epithelial sodium channel) become defective resulting in aldosterone resistance, Impaired excretion of K^+/H^+

GFR in Diabetic Nephropathy

- Initial 0-5 years tere will be GFR ↑↑: Glomerular hyperfiltration
- 5-10 years → Albuminuria → Irreversible damage.
- False (+)ve albuminuria:
  - HTN
  - CHF
  - Pyelonephritis

- Co-existing: Complications in DM at onset of Albuminuria

1. HTN
2. Non healing ulcer
3. Peripheral vaso-occlusive disease
4. Retinopathy: Blindness

Important Information

- **Progression of Both D. Retinopathy & D. Nephrology occurs simultaneously. Therefore, to monitor D. Retinopathy we do**
  - **Fundus Exam**
  - **S. Homocysteine Level (Also ↑ in – CAD, Stroke, Alzheimer's disease)**

Treatment

00:19:22

1. Stop Metformin / Sulfonylurea: GFR < 30 ml/min
   - Glipizide
   - Linagliptin

   } Can be given in kidney disease as they are metabolized by liver

2. Initiate Insulin: 80% of calculated dose
3. Target BP < 130/80 mm of Hg if Tolerated
   - ACE Inhibitor / ARB
     - S/E → Hyperkalemia (due to Type IV RTA)

- If K^+ is increasing the stop ACE inhibitors and start CCB
- If Potassium is not rising and BP is not under control the combination of A+C can be given, if edema present then add thiazide
  - If eGFR <30ml then the diuretic used: metazolone
- In ESRD patient, K+ is increasing then Alpha blockers are used

4. Treatment of Hyperkalemia

a) K^+ Binding Resins - Sodium polystyrene sulfate
b) Patiromer
c) Sodium Zirconate

5. Transplant indication

- eGFR < 20 ml/min/ 1.73 m^2

Practice question

Q. A chronic renal insufficiency patient present with peripheral edema and reduced urine output. Which of the following drugs will be suited in this patient for management of high renin Hypertension?

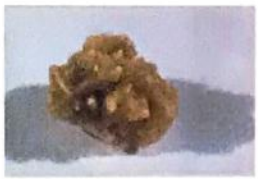
A. Aliskiren
B. Chlorthalidone
C. Prazosin
D. Beta blocker

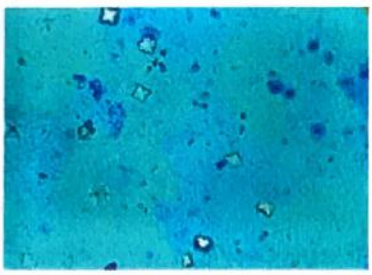
Ans: Prazosin

58 KIDNEY STONES

CALCIUM OXALATE STONE

00:00:33





- Most common
- Leading cause: Idiopathic hypercalciuria (Defective handling of calcium by kidney tubule)
- Test: 24-hour urinary calcium ↑↑
- Formation independent of urinary PH
- Work up
  - Urine microscopic examination shows "Envelope shaped" crystals
  - 24 hr urinary calcium is elevated
- Treatment
  - Thiazides MOA: (↓ Urinary calcium excretion)
  - Normal calcium & vit. D_3 and low sodium diet.

Important Information

- Furosemide: ↑ urinary calcium excretion (Contraindicated)
- Calcium monohydrate oxalate crystals are found in individuals with ethylene glycol toxicity and have "dumbbell / picket fence" form.

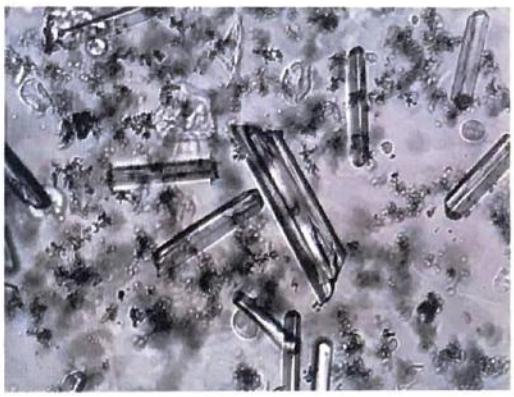
CALCIUM PHOSPHATE STONES

00:04:21

- Microscopy Urine: Rosette crystals/blunt end needles
- Whenever multiplication product of Calcium x PO_4 > 55, the two chelate and get deposited in tissues
- Seen in hyperparathyroidism
- Hyperparathyroidism → ↑ Ca^{2+}
  - Rx - Resection parathyroid adenoma
- Hypoparathyroidism (phosphate ↑)
  - Rx - Teriparatide injection

TRIPLE PHOSPHATE STONE

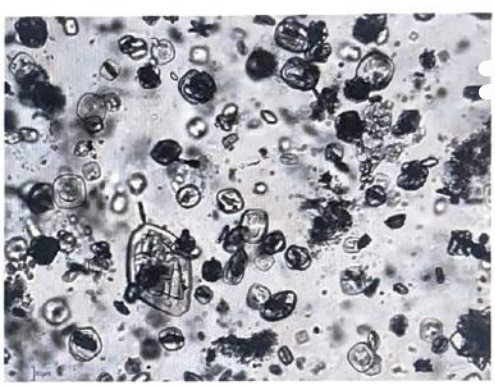
00:05:58



- Magnesium ammonium phosphate ($MgNH_4PO_4$)
- Due to alkaline nature of urine
- Proteus mirabilis urease positive
- Splits urea → ammonia (alkaline media) → precipitates $MgNH_4PO_4$ stones
- Microscopic appearance: coffin lid appearance

URATE STONE

00:07:54



- Softest
- No particular shape of crystals
- Can be - rhomboid/ rosette/ barrel shaped
- Formed in acidic urine
- Cause: Chronic gout
- TEST: 24 hr urinary uric acid to differentiate between Over producer (Rx: Allopurinol) and under excreter (Rx: Probenecid)

CYSTINE STONE

00:08:29

- Hardest stones
- M/e: "Hexagonal crystals"
- Etiology - Cystinuria d/t AR defect, there is defective proximal renal tubular reabsorption of cystine
- Sodium cyanide nitroprusside test is the initial screening test.
- It gives an intense purple colour.
- Urine may have rotten egg smell
- Rx: Cystine stone = d - penicillamine / tiopronin

XANTHINE STONE

00:10: 02

- Irregular shape
- Xanthinuria: Deficiency of xanthine oxidase.
- Rx: Xanthine stone = d - penicillamine / tiopronin
- Clinical Features:

1. Flank pain radiating to umbilicus or to Tip of genitalia
2. Reno gastric reflex → Vomiting
3. Renal colic
4. Gross Hematuria

IOC FOR RENAL STONES

00:12:26

- For renal stones CT abdomen (Non contrast)
- For ureteric stones: CT abdomen (Non contrast)
- KFT
- Urine microscopic examination

TREATMENT OF RENAL STONES

00:13:08

- Surgical Management
  - Renal Stone < 2 cm: Extracorporeal shock wave lithotripsy (ESWL) (More than once in cystine stone)
  - Renal stone > 2 cm: Percutaneous nephrolithotomy (PCNL)
- Ca oxalate stone: thiazide
- Ca phosphate: treat the cause
- Triple Po4: antibiotics, acetohydroxamic acid (for irrigation)
- Urate stone: Allopurinol, probenecid
- For prevention of cystine stone: Tiopronin > d-penicillamine
- For prevention of xanthine stone: Tiopronin > d-penicillamine
- ESWL contraindicated in (Absolute CI): Pregnancy, bleeding diathesis

Important Information

- Stone development independent of pH urine: Ca Oxalate
- Secondary xanthine stone is seen in case of treatment with allopurinol of severe hyperuricemia associated with myeloproliferative disorders and Lesch nyhan syndrome.
- Radiolucent stones
  - L - Lucent
  - U - Urate
  - X - Xanthine

59 RENAL TUBULAR ACIDOSIS

RTA – 1

00:00:37

- Distal convoluted Tubule (DCT) involved.
- α Intercalated cells responsible for H^+ excretion
- β Intercalated cells responsible for HCO_3^- excretion
- In RTA-1, α Interrelated cells are not working

a. Impaired H^+ excretion (Inability to acidify Urine)
b. Impaired NH_4Cl excretion
c. Salt wasting → ↑↑Aldosterone
d. Hypokalemia
e. Metabolic Acidosis which impairs tubular reabsorption of Ca^{2+}: Nephrocalcinosis

- Causes

a. Multiple Myeloma
b. Scleroderma
c. Amphotericin – B

- Treatment: Oral soda Bicarbonate

RTA -2

00:05:30

- PCT (Proximal convoluted Tubule) involved
- Associated with Fanconi syndrome
- Characterized by:
  - Bicarbatonuria
  - Salt wasting
  - Polyuria
  - Aminoaciduria
  - Glucosuria
  - Phosphaturia

Treatment

1. Thiazides: Cause Volume contraction
   - GFR ↓↓: RAAS activation
   - Aldosterone ↑↑ (Leads to increase H^+ Excretion thus causing alkalosis and neutralizing the acidosis component).
2. Soda Bicarbonate

Etiology

a. Multiple Myeloma
b. Wilsons disease
c. Expiry date Tetracycline

RTA-4

00:08:34

- M/c type of RTA

- Etiology: Aldosterone is not able to function (d/t Resistance/Deficiency)

a) Diabetes Mellitus – Most common association
b) AIDS
c) Chronic Tubular Interstitial disorders
d) Hypertensive nephrosclerosis

- C/F:

1. Salt wasting/ polyuria
2. Hyperkalemia
3. Inability to excrete H+

- Treatment:

1. Fludrocortisone
2. Diet – K^+ Restriction
3. Furosemide
4. Soda Bicarbonate

Summary of Renal Tubular Acidosis

00:11:20

RTA-1	RTA-2	RTA-4
• Defective α-Intercalated	• Damage to PCT	• Aldosterone Deficiency/ Resistance
• Nephrocalcinosis • K ↓	• Bicarbatonuria • Aminoaciduria • Glucosuria,	• Inability to acidify urine • K ↑

- All types of RTA → Normal Anion Gap Metabolic Acidosis (NAGMA)
- High Anion Gap Metabolic Acidosis (HAGMA) causes
  - **K** - Ketoacidosis
  - **U** - Uremia
  - **L** - Lactic Acidosis
  - **T** - Toxins – Methyl alcohol poisoning
- Urinary Anion Gap → Positive in RTA {$[Na^+] + [K^+] - [Cl^-]$}
  - Normal UAG = 0

Important Information

a) NAGMA (Normal anion gap **metabolic** acidosis)
b) RTA
- RTA – 1 → Kidney Stones
- RTA – 2 → Bicarbatonuria
- RTA – 4 → Hyperkalemia

c) Urinary Anion Gap → Positive (Blood anion gap is normal)
d) **Oral** Soda Bicarbonate is used to **neutralize** the **acidosis**

NAGMA

- **D** – Diarrhea
- **R** – **RTA**
- **F** – Fistula
- **U** – Uretero-sigmoidoscopy

How to remember

- DR FUSE

HAGMA

- **K** - Ketoacidosis
- **U** - Uremia
- **L** -Lactic Acidosis
- **T** - Toxins – Methyl alcohol poisoning

How to remember

- KULT

60 NEPHROTIC AND NEPHRITIC SYNDROME

NEPHROTIC SYNDROME

00:00:17

1. Massive proteinuria > 3.5 g /day
- Spot sample: urine protein = >2g of protein/ gram of urinary creatine
2. Hypo albuminemia: S. Albumin <2.5 gm%
3. Oncotic pressure decreased: Oedema
4. Increased lipids–accelerated atherosclerosis
- Also Seen in
  - SLE
  - APLAS
  - Syndrome X
  - DM
  - Hypothyroidism
5. Lipiduria
- Also seen in
  - Filariasis syndrome
  - Fat embolism syndrome
- Under microscope: oval fat bodies / maltase cross appearance
6. Hypercoagulable State
- Urinary loss of protein C/S / Anti thrombin III Loss of
- Deficiency of ferritin
- Ceruloplasmin loss in urine
- Fibrinogen values are either normal or elevated
- An important cause of hypercoagulable state is Factor 5 leiden mutation

1° NEPHROTIC SYNDROME

00:10: 40

- FSGS: M/C in adults
  - Primary FSGS: Idiopathic
  - Secondary FSGS: cause include
    - → HIVAN
    - → HBV
    - → Human parvovirus B19 (Slapped cheek appearance, Erythema infectiosum, Aplastic crisis)
    - → Heroine
    - → Pamidronate
    - → Lithium
    - → Reflux Nephropathy
    - → Hypertensive Nephrosclerosis and Alport syndrome
    - → SCA
- Glomerulo Nephropathy (M/C in > 65 years)
- Minimal Change Disease (M/C in children)

2° Nephrotic Syndrome

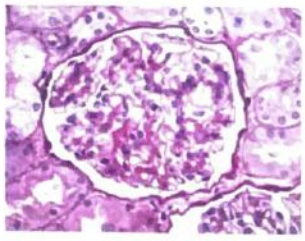
- DM (M/C)
- Amyloidosis
- Lupus nephritis
- Pregnancy Induced HTN

Clinical features

1. Hematuria / cola color urine
2. HTN – Headache
3. Nephrotic range proteinuria: Foamy urine, periorbital edema, Pedal edema, Ascites, Pleural effusion
4. ANASRCA
5. Decrease GFR: Uremia 50% next 6-8 years (Uremic Frost, U. pericarditis)

Work-up

1. KFT deranged
2. Urine microscopy: > 3 RBC / HPF
- (N) Centrifuged specimen < 3 RBC / HPF
3. Spot sample> 2g/gram of urinary creatine
4. USG guided Kidney Biopsy:
- On LM
  - Segmental obliteration of glomerulus/ solidification of glomerular tuft
- On Electron microscopy
  - Podocytes foot process – effacement of podocytes
  - Podocyte Vacuolization
  - Podocyte Detachment
5. Infection Panel
6. Urine toxicology screen
7. USG → B/L Enlarged Kidney (HIV Associated Nephropathy)
8. SLE → ANA, ds DNA



Management

1. Primary - HTN - ACE inhibitors
2. Edema → Thiazides if refractory / Furosemide + metolazone
3. Proteinuria: Steroids for 4-16 weeks or until complete

remission / Cyclosporine

MEMBRANOUS GLOMERULOPATHY (MGN)

00:32:10

- MC in Geriatric population
- 1°: Antibody against PLA_2 receptor
- 2°: Causes include

a. Infection – HBV / HCV
b. Autoimmune disorders like Hashimoto thyroiditis/Graves
c. Connective tissue disorder: RA / SLE / Scleroderma / MCTD
d. Solid organ tumors: Lymphoma, Leukemia
e. Drugs – Penicillamine, Captopril, Mercury based compounds

Clinical Features

- Puffy eyes
- Pedal edema
- Anasarca
- HTN

Work up

1. KFT: creatinine increased
2. Urine microscopy: oval fat bodies, maltase cross appearance
   - Maltase cross appearance in Peripheral smear: Babesia Microti
   - Maltase cross appearance in CSF: Cryptococcus neoformans
   - Maltase cross appearance in urine under microscopy also seen with Fabry's disease
3. Urine protein > 2g/gm of UC
4. Urinary protein electrophoresis reveals albumin
   - If it was multiple myeloma then it shows globulin
5. Anti-PLA_2 Ab – (+) highly diagnostic
6. Kidney Bx on Electron Microscopy shows Sub epithelial deposits + spikes
7. S. electrolytes
8. Infection panel, autoimmune panel
9. FDG-PET scan
10. Ceruloplasmin & Ferritin ↓: Nonselective proteinuria

Treatment–

- HTN – ACEI / ARB
- Edema: thiazides
- For management of proteinuria: Steroids / Cyclophosphamide (alternate) In geriatric population

Complication of MGN

- Most important complication: renal vein thrombosis
  - U/L enlarged painful kidney with AKI features
- Renal vein thrombosis in pediatric age group: Severe Diarrhea contribute to AKI +U/L enlarged painful kidney
- Child with dysentery (E. coli O157:H7) followed by AKI + Schistocytes: HUS

CONGENITAL NEPHROTIC SYNDROME

00:53:32

- NPHS1-gene – Nephrin decreased zipper mechanism # defect
- NPHS2 gene – Podocin decreased (Bad Prognosis)
- α_1Actinin Gene

Clinical features

a. Non-Immune Hydrops fetalis
b. Anasarca
c. Foamy urine

Work up

a. 24 hr urinary protein Increased ↑ = > 40mg / m^2 BSA
b. Serum Albumin – decreased

Treatment

- Daily Albumin Infusion
- Cause of Death – Infection

MCD (MINIMAL CHANGE DISEASE)

01:00:16

- Most common cause of nephrotic syndrome in children.
- M/c cause of MCD is Idiopathic
- Other causes include: NSAIDS usage and Hodgkins lymphoma

Clinical features

1. Puffy eyes
   - Vulval edema / Scrotal edema
   - Pedal edema, with diurnal variation of edema
2. Wt gain (+)
3. Oncotic pressure decreased – Albumin decreased
   - Selective proteinuria
   - Pleural effusion B/L (Transudative) – dyspnea
   - Ascites – Abdominal girth increased, everted umbilicus

Work up M.C.D:

1. KFT: normal
2. BUN / creatinine – (N)
3. Urine m/e – Oval fat Bodies
4. 24 hr urine protein increased
5. Urine protein electrophoresis
6. C_3 levels (N) / decreased
7. Kidney Biopsy – LM (N)
   - EM – Podocyte effacement

Treatment

1. **Steroids**
   - Prednisolone X 8 weeks (Taper dose), good response

and hence called as steroid responsive nephrotic syndrome.

- Sudden withdrawal cause – Addisonian crisis
- Toxicity: Cataract, Hirsutism, BP increase, nephropathy, Wt gain
  - Discontinue Steroids and Start cyclophosphamide / levamisole

Remission	Urine albumin nil or trace (or proteinuria <4 mg/m h) For 3 consecutive early morning specimens.
Relapse	Urine albumin 3+ or 4+ (or proteinuria > 40 mg/ mh) For 3 consecutive early morning specimens, after having been in remission previously.
Frequent relapses	Two or more relapses in initial 6-month period or more than 3 relapses in any 12 months.
Steroid dependence	Two consecutive relapses when on alternate day steroid therapy or within 14 days of its discontinuation.
Steroid resistance	Prednisolone at a dose of 2 mg/kg/d for 4 weeks.

- For Steroid Resistant Nephrotic Syndrome: Rx - Cyclosporine / Tacrolimus

Previous Year's Questions

Q. Which of the following statement is correct about definition of steroid resistant nephrotic syndrome? (FMGE June 2021)

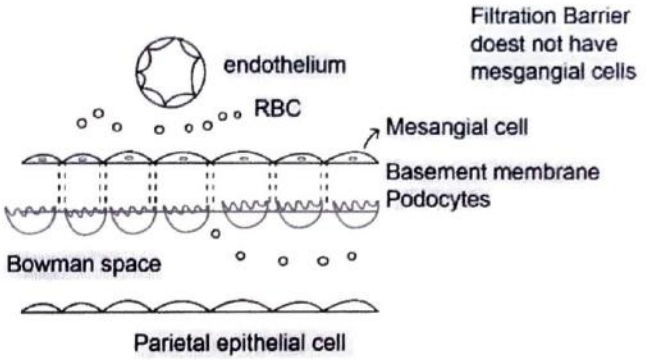
A. Absence of remission x 4 weeks
B. Absence of remission x 6 weeks
C. Absence of remission x 8 weeks
D. Absence of remission x 12 weeks

Quick Summary of Causes of Nephrotic Syndrome

Refer Table 60.1

NEPHRITIC SYNDROME

01:20:53



- Vasculitis – GFR Decreased, and Renin increased leading to

1. HTN
2. Hematuria
3. Sub Nephrotic proteinuria

Post Streptococcal Glomerulonephritis

- Type III Hypersensitivity reaction
- Immunological manifestation seen with Nephritogenic stain – Streptococcus
- Antigen – Streptococcal Pyrogenic exotoxin B (SPEP)
- Immune complex (+)
- Complement mediated vasculitis
- PSGN- begin as Impetigo or Pharyngitis

Impetigo
Strains-47,49,59,55,60
2-6 week

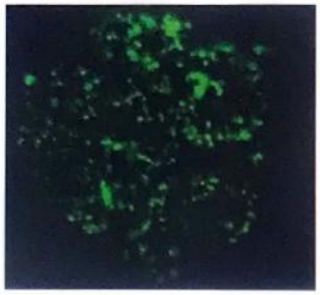
Pharyngitis
Strains-1,2,3,4,25

Nephritic Syndrome

1. Hematuria
   - Cola colored urine
   - Smoky urine (Freshly voided sample)
2. HTN – sudden onset – LVF
   - Dyspnea – pulmonary edema (+)
   - Encephalopathy → Loss of consciousness
3. Renal Insufficiency leading to features of uremia

Work Up

1. Urine m/e - >3 RBC / HPF or Gross Hematuria
   - RBC casts
   - Dysmorphic RBC: Always indicate Glomerular bleeding.
2. $C_3 \downarrow CH_{50}$ decreased C_4 (n)
3. KFT → ↑ S. Electrolytes
4. Potassium increased
5. Anti DNAase increased, Anti Hyaluronidase Increased ↑
6. Kidney Bx
   - LM – Hyper cellular glomerulus, PMN infiltration
   - EM → IgG / IgM / C3 deposits (sub epithelial cell deposits)



7. Immuno florescence study – Starry Sky appearance

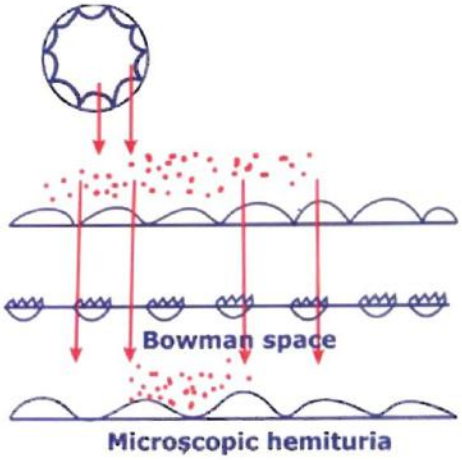
Treatment

1. Conservative management
2. Acute Pulmonary Edema – Furosemide
3. HTN crisis – IV Labetalol, sodium nitroprusside,
4. Benzathine penicillin

BERGER'S DISEASE / IgA NEPHROPATHY

01:40:04

- 1st Line of defense – Ig A (Mucosal Immunity)
- MC form of glomerulonephritis worldwide
- In Berger disease IgA ↑ with diffuse mesangial deposits
  - Capillaries (N)
  - GFR (N), RFT (N), Urine Output (N)
  - BP (N)
  - Microscopic Hematuria



Microscopic hemituria

- If Pt develops URTI – IgA levels rise and over next 48 hrs,
- Microscopic Hematuria is replaced by macroscopic/Gross hematuria.
- Some patients have asymptomatic microscopic Hematuria and may be picked up on annual clinical medical checkup.
- O/E – BP (N), no edema is noted

Work up

- >3RBC / HPF
- C_3 NORMAL in most cases

Treatment

- No definitive treatment
- Steroids: only when patient is recovered from URTI
- ACE inhibitors: May be used (mostly Bp comes back to normal in few days)
- Fish oil, Tonsillectomy

Synpharyngitic Hematuria

PSGN	Burgers
• C3 decreased	• H/O : URTI (for 48 hr)
• KFT ↑	• KFT – N
• BP ↑↑	• BP- ↑/N
• Anti-DNA ase antibody	• C3 normal

- MC cause of Hematuria [Microscopic] → BERGER DISEASE

Table 60.1

FSGS (Adult)	• Non-selective proteinuria	• EM Foot process detachment, Hyaline effacement, vacuolization deposits
MGN (Geriatric age)	• > 65 yrs – R.V.T	• Subepithelial deposits +spike
MCD (Child)	• 2-8 yrs selective proteinuria	• IgM, Foot process effacement
Finnish (neonate)	• Neonate-hydrops	• Like FSGS

CLINICAL QUESTIONS

Q. A 36 years female presented to OPD with complains of blood in urine, nausea, weakness and swelling of both legs and puffiness of face . O/E - BP = 150/ 110 mm Hg, pulse = 80 bpm. Urine analysis revealed non selective proteinuria and red cell casts. Renal biopsy showed glomerular crescents. Likely diagnosis is -

A. MGN
B. MCD
C. RPGN
D. PSGN

Answer: C

Solution

- Rapidly progressive glomerulonephritis (RPGN) - characterized by a **rapid loss of renal function with glomerular crescent formation**
- If left untreated, it rapidly progresses into acute renal failure and death within months.
- C/F: **hematuria, red blood cell casts in the urine and non selective proteinuria**
- Hypertension and edema are also seen.

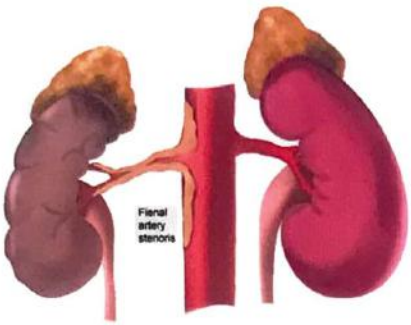
61 RENAL ARTERY STENOSIS

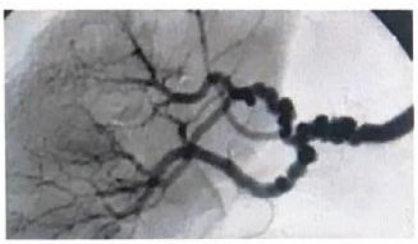
- On USG: Asymmetric kidney (In U/I Renal artery stenosis)

Etiology

00:00:47

1. Most common cause – Atherosclerosis/ ARAS (Ostial narrowing)
2. Fibromuscular dysplasia (FMD) (Young females)
- Distal Blood vessels affected
3. Takayasu arteritis – Asian population (Pulseless disease)





- PAN is not a cause of Renal Artery stenosis

Clinical Features

00:04:57

1. HTN Persisting with medication
2. ACEI Increase undetected B/I RAS → AKI
3. Flash pulmonary edema (d/t acute LVF)
4. Presence of Abdominal Bruit (Systole + diastole) (Intensity ∝ Severity of disease)
5. Peripheral pulses absent

Work up

00:13:21

1) USG: Asymmetric kidneys size
- Not responding to anti-hypertensive medication
2) Doppler – Best Screening test
3) CT Angiography ... k of Contrast nephropathy
4) Magnetic Resonant Angiography (M-R-A)-expensive
- False positive results – due to turbulent flow
- Gadolinium → cause Nephrogenic systemic fibrosis
5) IOC: Renal Angiography (Invasive test) (Gold standard)
6) DTPA scan before and after giving ACE inhibitor (Captopril Renogram)
7) KFT
8) Urine albumin excretion rate increase

Treatment

00:19:18

1. U/I RAS: ACEI + CCB + α-Blocker
2. For B/I RAS
- ACE Inhibitors: Contraindicated in Bilateral RAS because it causes efferent arteriole dilatation leading to loss of filtration gradient and increasing the risk of acute kidney injury.
3. FMD: PTRA [Percutaneous Transluminal Renal Angioplasty] + Stenting
4. ARAS: medical therapy

Malignant HTN

- BP rise disproportionately
- On fundus examination: retinal damage can be seen
- Encephalopathy
- KFT deranged
- Microangiopathic hemolytic anemia: Schistocytes present on Peripheral smear
- Postmortem Kidney biopsy shows

a. Fibrinoid necrosis
b. Onion skinning appearance

- More common in Afro-Americans

Atheroembolic Kidney Disease

00:28:37

- Post angiography: Sudden derangement of KFT (immediately or 1 to 14 days after the procedure)
- AKI
- Toe gangrene
- Fever, abdominal pain
- Eosinophil uria
- Postmortem Kidney Biopsy finding: Microvessel occlusion by cholesterol crystals

LEARNING OBJECTIVES

GIT

Bleeding from the Gut

- Bleeding from the Gut
- Upper GI Bleed
- Hematochezia
- Melena
- Hemobilia
- Diseases of Esophagus
- Zenker's Diverticulum
- Diffuse Esophageal Spasm
- Achalasia Cardia
- Boerrhaave syndrome
- Stomach Volvulus
- GERD

Peptic Ulcer Disease

- Introduction
- Causative agent of type B gastritis
- Mechanism of Action of H.pylori
- Work up
- Treatment
- Type A Gastritis
- Difference between Gastric ulcer and Duodenal ulcer
- Epigastric pain
- Investigation of choice
- Treatment for Type A gastriis
- Complications of GU and DU
- Image based questions
- Case discussions
- Premalignant conditions of gut
- Peutz Jeghers syndrome

Zollinger Ellison Syndrome

- Clinical features
- Work up
- Tumor localization
- Treatment

Malabsorption Syndrome

- Introduction
- Carbohydrate malabsortion
- Fat malabsorption
- Celiac spruce
- Tropical spruce
- Whipple's disease
- Bacterial overgrowth syndrome
- Schilling test

Inflammatory Bowel Syndrome

- Introduction
- Inflammatory bowel disease
  - Most common site
  - Serpiginous ulcers
  - Clinical features
  - Fistula formation
  - Work-up
  - Treatment
- Pseudomembranous colitis
  - Clinical features
  - Work-up
  - Treatment
- Endoscopic image of esophagus
  - Ulcerative colitis
  - Clinical features
  - Investigation
  - Treatment
- Entra intestinal ibd

Irritable Bowel Syndrome

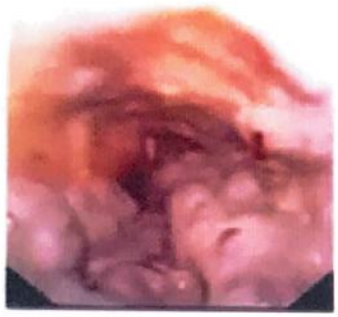
- Irritable Bowel Syndrome
- Manifestations
- Dysbiosis
- Clinical features
- Investigation
- Treatment

62 BLEEDING FROM THE GUT

- Anatomical landmark to differentiate between Upper GI & Lower GI Bleeding: Ligament of TREITZ [DJ Flexure]

CAUSES OF UPPER GI BLEEDING / HEMATEMESIS: 00:01:05

1. Peptic ulcer disease (leading cause for hematemesis)



- **Duodenal ulcer** = Gastroduodenal artery (Source of bleeding)
- Location
  - Anterior - perforation
  - Posterior – bleeding
- **Gastric ulcer** - Left gastric artery Source of bleeding
- M/C site for Gastric Ulcer is: lesser curvature close to incisura angularis
- Presentation –
  - Unrecordable BP / SBP < 90 mm/hg incase of torrential hemorrhage

Management

- Grey IV cannula insertion
- Fluid bolus
- Massive transfusion protocol

↓

Hemodynamic stability achieved

↓

UGIE + cautery

2. Drug induced gastritis:

- COX -1inhibitors (like Indomethacin, ketorolac)
- Erosive gastritis

Note: H. Pylori causes Non- Erosive gastritis

3. Portal hypertension

- Presents with Hematemesis + Splenomegaly
- Defined as pressure > 5mmHg
- Once the pressure > 12 mmHg rupture of esophageal varices
- Source of bleeding – Coronary veins

Note:

- Leading cause of PORTAL HYPERTENSION – Alcoholic Cirrhosis
- Liver damage with Negative viral markers, non – alcoholic, non-metabolic cause – CRYPTOGENIC CIRRHOSIS / NON CIRRHOTIC PORTAL FIBROSIS
- Portal Hypertension in pediatric patient with splenomegaly – Extrahepatic portal vein obstruction

Management

- For non bleeding oesophageal varices – propranolol
- Bleeding oesophageal varices – Octreotide, Terlipressin

Rx

- UGIE + Sclerotherapy

Note:

- Propranolol
  - Management of non bleeding oesophageal varices
  - Best for prophylaxis of migraine
  - Best for hypertrophic cardiomyopathy

4. Mallory Weiss syndrome: (sub mucosal tear)

- KEY POINTS:- RETCHING episodes: Alcoholic binge drinking/hyperemesis gravidarum
  - **Primary site of tear** = Lower esophageal sphincter and starts from cardia.
  - **Source of bleeding** = Left gastric artery
  - **Presentation** = Retching + Hematemesis ± Vasovagal Syncope
  - **Treatment** = Mostly self limiting condition
  - If Bleeding reoccurs – UGIE + Inj. Adrenaline / Cautery / Endoscopic Clipping

5. Dieulafoy's lesion: Aberrant sub mucosal artery bleed [Pin point bleeding]

6. GAVE: gastric antral vascular ectasia (Water melon stomach) - Least common cause / rare

Rx

- Injection of Intra-Arterial [hepatic artery] – 5-FU for heptic metastasis

5. Angiodysplasia of colon (Vascular malformation of colon):

- CASE SCENARIO:
- In a patient of 70 years of age 2 colonoscopies are done in last 1 year, both reports are normal but hematochezia is persisting off & on (+), diagnosis will be angiodysplasia colon.
- KEY POINTS - Pin point bleeds and may not bleed on the day colonoscopy was done.
- Hence high probability of missing the lesion
- IOC : CT - Angiography

MELENA

00:47:00

- Presentation: 60-80 ml blood, 12-16 hours in the gut to results in Black tarry stool.
- **Cause-**

1. P. U.D (Peptic ulcer disease)
2. Erosive gastritis - Drug induced
3. Portal HTN
4. Mallory Weiss
5. Dieulafoy's Lesion

Investigation of choice

- Upper GI Endoscopy

HEMOBILIA-(UPPER GI BLEEDING)

00:49:43

1. Trauma – Lap cholecystectomy [M/C]
2. Instrumentation: (like ERCP)
3. Cholangiocarcinoma
4. Klatskin tumor (also a type of cholangiocarcinoma)
5. Parasites

Investigation of choice

- Angiography

Rx

- Gel embolization of bleeder

Summary

- Hematemesis + BP ↓ → 1st step → Fluid resuscitiation
- Hematemesis, Melena → Peptic ulcer Ds.
- Hematemesis + Splenomegaly → Portal hypertension
- Alcholic binge / Hyperemesis gravidarum + vomiting → Mallory weis syndrome
- Hematochezia → Diverticulitis

Causes of Hematochezia in Pediatric:

- Rectal polyp> Meckel's diverticulum
- Remnant of vitello intestinal duct and occurs on anti – Mesenteric Border.
- Asymptomatic: > 98%, Maroon color stool
- **IOC: -** TC99 pertechnate scan

Rule Of 2

- 2% population
- 2 inches size
- 2 feet from ileocaecal junction
- 2 mucosa :stomach |pancreatic

Rx

- Resection and end to end anastomosis

Intussuception

- Enlargement of peyers patches
- Red currant jelly stools

Neonate With Hematochezia

- Case Scenario
  - Home delivery of baby, mother expired
  - feeding with cow's milk → blood in the stool
  - DX – NECROTIZING ENTEROCOLITIS
  - Staging used – BELL staging

Pneumatosis Intestinalis

- Air inside the intestinal walls

ESOPHAGEAL DISORDERS

01:05:11

1. **Zenker diverticulum: (Location: Killian Δ > Laimers Δ)**

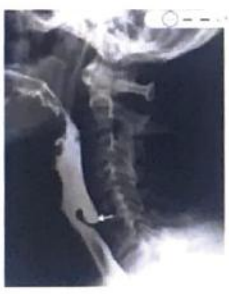
- Geriatric, Non- progressive dysphagia
- Halitosis
- Regurgitation of yesterday food items

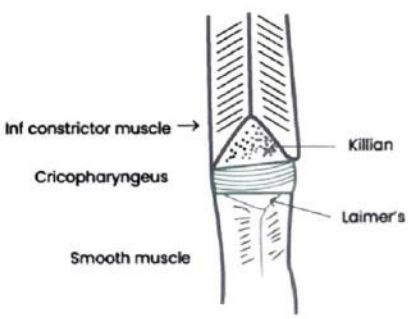
Investigation of choice

- Ba swallow

Rx

- Surgery





Note:

- Dysphagia symptom → > 2/3rd Luminal obstruction

DIFFUSE ESOPHAGEAL SPASM

01.11.25

- M/c cause: Idiopathic
- **Clinical Features:-**

i. Intermittent Chest pain at rest (Symptoms of CHF are absent) – R/O CAD – TMT / stress echo / ECG [Esophageal angina]
ii. Dysphagia : non- progressive

Ba swallow : Cork screw appearance / Rosary bead appearance

- **Investigation of choice**
  - Esophageal Manometry
  - Luminal pressure ≥ 120mm Hg for > 3sec

Ba swallow → irregular shaggy appearance → [esophageal candida infections]

- **Nut cracker esophagus**
  - If pressure > 180mm Hg > 6.5 sec

Note:

- Tone of LES – 15-25 mm Hg

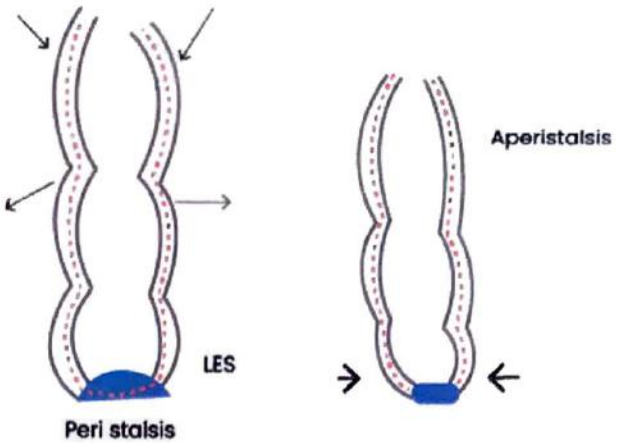
- **Rx**

1. Anxiolytics
2. Nitrates → Isosorbide mononitrate (long acting)
3. Calcium channel blocker → Amlodipine

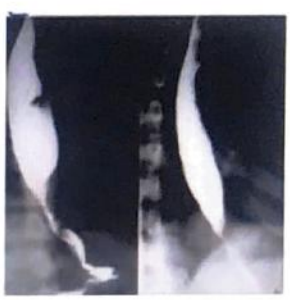
ACHALASIA CARDIA

01:22:00

- Causes
  - Autoimmunity (M/C)
  - Oat cell Ca lung
  - Chagas disease (Reduviid bug)



- Pathophysiology of Achalasia: Aperistalsis and increased Tone of LES [> 25mm Hg] due to loss of inhibitory control.
- C/F-
  - Female: 25-year progressive dysphagia
  - Liquids > solid (Dysphagia)
  - Halitosis
  - Regurgitation of previous day food items
  - Recurrent pneumonia episodes

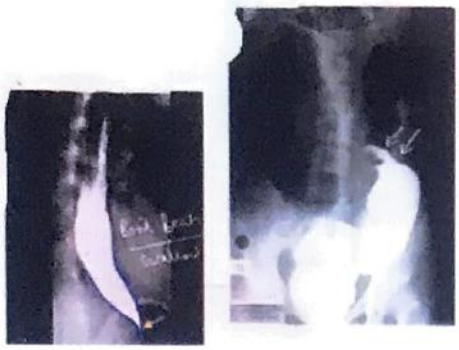


- IOC
  - Esophageal manometry: (LES tone increase)
  - Ba swallow: Bird beak appearance
  - Ba enema: Bird beak → Sigmoid volvulus Q***

- Rx-
  - Laparoscopic Heller's myotomy + Nissen fundoplication [partial]
  - Botulinum toxin + CCB

Note:

- Oat cell carcinoma
  - Anti-Hu antibodies – Achalasia cardia
  - SIADH
  - Cushing syndrome
  - Lambert eaten syndrome



Note:

- Carcinoma Esophagus
- Shouldering sign – irregular filling defect – Rat tail appearance

Case scenario

- 25yr old female presents with progressive dysphagia(oropharyngeal dysphagia) but LES tone: decreased, leather like skin, Raynauds phenomenan, HTN crisis
- Then Dx is Scleroderma and not Achalasia cardia where LES tone is increased

IOC

- Anti-Topoisomerase Ab

DYSPHAGIA

OROPHARYNGEAL	ESOPHAGEAL
• Strctural defect ○ Zenker's diverticulum ○ Neoplasm ○ Plummer Vinson syndrome → Post cricoid dysphagia-web → IDA → Koilonychia • Neurogenic defects ○ CVA • Myopathic defects ○ Myasthenia gravis	• SCHATZKI RING ○ Lower 1/3rd ○ Not premalignant ○ Meat impaction ○ Aphagia

01:39:00

Case Scenario: 30 years alcoholic, binge drinking, retching chest pain, syncope

- O/E:
  - SC Emphysema – Crepitus
  - HR= 120/mm
  - BR = 90/60 mm Hg

Probable diagnosis?

Boerhaave syndrome

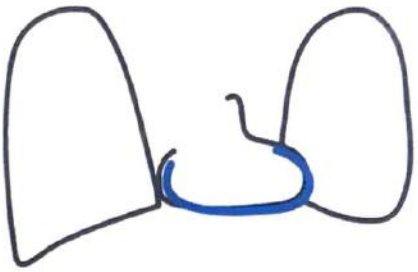
- Oesophageal tear: Lower 1/3 of esophagus is involved (post lateral)
- Chemical mediastinitis explosive chest pain
- Pneumomediastinum: Kinking SVC/IVC venous return decrease BP decrease Vasovagal component
- Mackler's triad = Chest pain + Vomiting + SC emphysema (l/t crepitus +
- Instrumentation of Esophagus : Cervical Esophagus

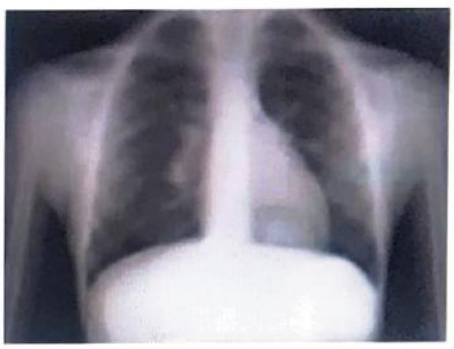
Investigation-

1. Chest X-Ray
2. **IOC** - CT chest + contrast (Water soluble)

Rx

- Surgical repair

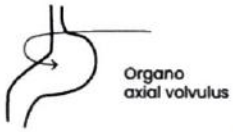


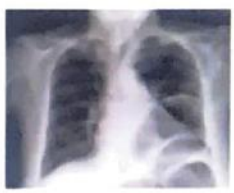


01:45:17

Case Scenario: 30 years alcoholic presents with epigastric pain, trying to vomit but no vomiting yet, failure to pass NG tube probable diagnosis?

Dx: Stomach Volvulus





Borchardt Triad

- Epigastric pain, ncausea mimics vomiting, failure to pass

NG tube

Investigation

1. Chest X-Ray
2. IOC: CT abdomen with oral contrast, Barium contrast

Rx

- Surgical exploration

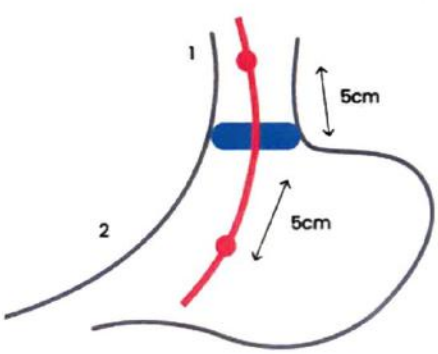
NG Tube should be put in conscious patient in sitting position with neck partially flexed to protect the airway

GERD | NON ULCER DYSPEPSIA (NUD): 01:52:58

- **Clinical features:**

1. Chest pain/heart burn/LES tone decrease/retrosternal pai
2. Sour brash
3. Dental enamel damage
4. Nocturnal cough (chemical tracheitis) - seen in asthma/ post nasal drip/GERD
5. Sore throat (multiple episodes) – chemical laryngitis

- **Investigation of choice**
  - 24 hr pH monitoring Esophageal pH: < 4.0 for > 4hrs/day



Rx

- DOC is PPI + Prokinetic agents (like Mosapride / Itopride) improve symptoms faster.

Note:

- Curling ulcer of burns: MC in the Duodenum
- Cushing ulcers of raised ICT: MC in the Stomach
- Cameron ulcer: Hiatus hernia

Barrett Esophagus:

- Histopathology: GOBLET cells in Esophagus
- Metaplasia: Squamous to columnar
- Dysphagia
- Carcinoma in situ
- Cancer

Investigation

- Upper GI Endoscopy and Punch biopsy (Goblet cells+)

Rx

- Esophageal resection + Stomach mobilization

Extra Mile:

- M/C type of metaplasia
  - Columnar to squamous due to effects of smoking.

63 PEPTIC ULCER DISEASE

TYPE B GASTRITIS

00:01:00

Helicobacter pylori (MC)

- Leading cause of PUD: Type B gastritis
- Gram negative coccobacilli
- Route of spread: Feco-oral route
- Non-Sporing: eradication possible
- Extra Cellular Bacteria / Microaerophilic
- MOA:

00:02:37

Urea —urease→ Ammonia(Alkalin)
↓
Irritation of G cells& parietal cell ⊕
↓
Excessive acid production (Hyperchlorhydria)
↓
Ulcer
Duodenal ulcer>Gastric ulcer

- Work Up

00:05:09

1. Screening test → Breath urea test (Non-invasive)
2. Urease test (Invasive) (CLO TEST- commercial name) → scraping wall of stomach endoscopy
3. Biopsy: Warthin starry stain

- Treatment: - Antibiotics

00:07:02

  - Triple Therapy
    - → Pantoprazole (PPI)
    - → Clarithromycin
    - → Amoxicillin/clavulanic acid (Resistant cases) – 2 weeks
  - Quadruple therapy
    - → Bismuth
    - → Tetracycline
    - → Metronidazole
    - → Pantoprazole
  - Erythromycin Not used for Rx
  - Bismuth acts by reducing acid-mucosa contact time

TYPE A GASTRITIS

00:09:44

- Autoimmune disorder, premalignant condition
- Anti-parietal cell antibodies
- Achlorhydria → gastric ulcer
- Intrinsic factor ↓, B12 ↓: Pernicious anemia
- Hypergastrinemia, Achlorhydria

00:11:49

Refer Table 63.1

Important Information

H. Pylori is an Extracellular bacterium while Tropheryma whippelii leading to Whipple's disease is intracellular bacteria found inside macrophages of intestine.

Workup

- IOC for PUD: – Upper GI endoscopy (UGIE) + Biopsy
- Urease test

Treatment

- PPI x 6 weeks

Important Information

- Refractory PUD may be due to Zollinger-Ellison Syndrome

Complications of Gastric and Duodenal ulcer 00:17:21

Gastric ulcer

1. Bleeding: Left gastric Artery
   - Management: when hemodynamically stable

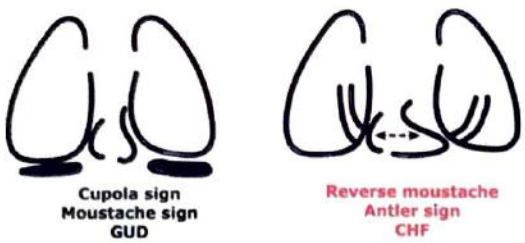
- Upper GI Endoscopy +Cautery at base

2. Perforation: Lesser sac peritonitis
3. Penetration: Slow, posterior by,
- Pseudo pancreatic cyst
- Necrotizing pancreatitis

4. Gastric outlet obstruction (GOO): Tea pot stomach

In duodenal ulcer:

1. Perforation (Most common): Anterior > posterior
- M/c cause of peritonitis
- Cupola sign
- Moustache sign
- Gas under diaphragm



2. Bleeding: Posterior > Anterior Source - Gastroduodenal artery
- MX: - Adrenaline at base of ulcer
- Cautery, ligation, exploratory laparotomy

Refer Table 63.2

SIGMOID VOLVULUS

- Important cause of Large Bowel Obstruction
- Sudden onset Left iliac fossa crampy pain
- Obstipation (Can't even pass flatus)
- X- Ray abdomen: Coffee bean app.
- Barium enema: Bird BEAK app

Treatment

1. Intravenous Fluids
2. Flatus tube
3. Colonoscopic detorsion

Important Information

Q. A 60 yrs old, non-vegetarian with chronic constipation having LIF pain + Hematochezia
- Diagnosis: Diverticulitis
- Investigation: CT abdomen

PRE- MALIGNANT CONDITIONS OF GUT 00:30:00

1. **Oral cavity**
- Oral submucosal fibrosis: Gutka/ Betel nut
- Leukoplakia: More common
- Erythroplakia: More malignant
- Syphilitic glossitis

2. **Esophagus:**
- Tylosis palmaris: Hyperkeratosis in palms and soles (SCC risk↑)
- Achalasia cardia
- Barrett's esophagus
- Plummer Vinson syndrome: Esophageal web +IDA (Iron deficiency anemia) + Koilonychia

3. **Stomach**
- Type A Gastritis
- Gastric ulcer
- Menetrier's disease
  - Proliferation of foveolar cells ⊕ & ↓parietal cells → Achlorhydria → Gastric ulcer
  - Cerebriform appearance of stomach mucosa
  - Rx: Cetuximab

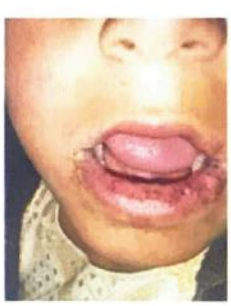
4. **Small Intestine**
- Crohn's disease
- Cronkhite Canada Syndrome: Polyps in duodenum
  - Sporadic hamartomatous polyp ⊕

5. **Large Intestine**
- Inflammatory bowel disease
  - Malignancy Incidence is equal in both UC = CD
- Familial adenomatous polyposis (FAP)
  - APC gene
  - Chromosome 5 #
  - >100 polyps / colon
  - Primary colectomy (Prophylactic)

PEUTZ JEHGER SYNDROME 00:38:52

- Hamartomatous polyps in jejunum that lead to bleeding, obstruction & intussusception
- STK 11 / LMB 1 gene, AD
- Chromosome 19

Arborizing pattern



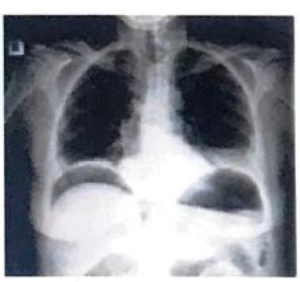
Mucosal melanosis

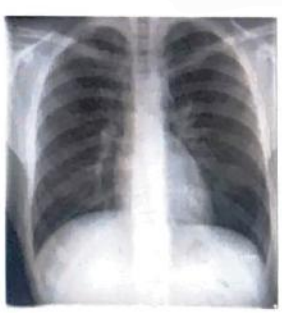
- Mc site: Jejunum
- Mucosal melanosis of lips/anus
- Increased incidence of Ca stomach / ovary / cervix/ Hepatobiliary cancer
- Polyps themselves are not premalignant but a definitive association with both intestinal, hepatobiliary, and extra intestinal malignancy.

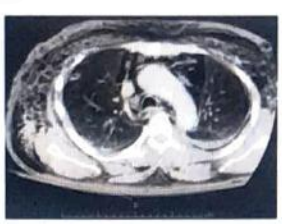
Table 63.1

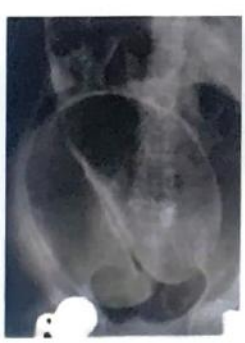
	Gastric ulcer	Duodenal ulcer
Blood group:	A	O
Site:	Type 1: lesser curvature (MC site) near incisura Angularis	D_1: Duodenal Cap
Pain:	Epigastric pain (Immediately after food Intake) weight loss	Epigastric pain "hunger pain" Snacking by patient leads to weight gain

Table 63.2









Gas under diaphragm: Pneumoperitoneum	Pneumomediastinum; continuous diaphragm sign	Coffee bean appearance, Bird beak appearance: Sigmoid volvulus

64 ZOLLINGER ELLISON SYNDROME/ GASTRINOMA

Clinical Features 00:00:13

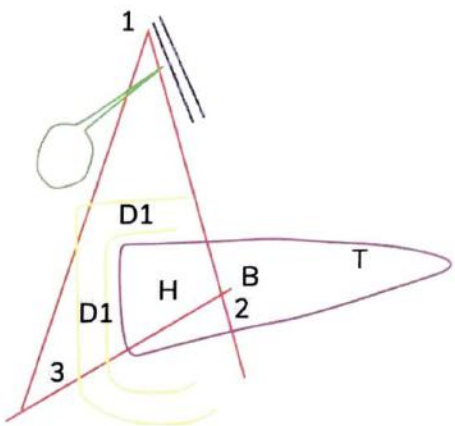
- MC Site of presence of G Cells : Stomach But
- MC Site of Gastrinoma: Duodenum > Pancreas > Stomach
- Clinical Scenario – 30-50 yr old female,
  - Epigastric pain (Usage of PPI) for undescribed long periods of time
  - Diarrhea: Gastric hypersecretion leads to Acidic pH in duodenum, This leads to:
    - → inactivation of pancreatic enzymes → Unabsorbed Sugar
    - → Mucosal damage 2° to ↑ HCL from stomach in response to ↑Gastrin
  - GERD

MEN I
Pituitary adenoma →↑ **prolactin**
Parathyroid adenoma →↑ **Ca^{2+}**→↑ **acid secretion**
Pancreatic adenoma → **Zollinger Ellison syndrome**

  - Family h/o Kidney stones
  - Past medical h/o kidney stones
  - Liver metastasis – Hard consistency of liver edge

Work – up 00:07:30

- UGIE – Multiple duodenal ulcer / Atypical (2nd part of duodenum / Giant ulcers)
  - PUD usually seen in – 1st part of duodenum
- Breath urea Test: (-) ve :R/o H. Pylori
  - UREASE: (-) VE: R/o H. Pylori
- PTH assay, S. Calcium: ↑ PTH if adenoma (+)
  - Pancreatic polypeptide : ↑ ed if adenoma (+) ve
  - Prolactin : ↑ if prolactinoma (+)
- IOC : SECRETIN STUDY
  - Check fasting Gastrin (x 10 times → 1000 pg/ mL)
    Falsely high – PPI, H. pylori, G.O.O
  - Basal Acid Output > 15 meq/ hr
  - Basal Acid output/ Max. Acid output > 0.6
- IOC for tumor localization – Endoscopic US
  - Duodenum > Pancreas > Stomach
  - It could also be in the mesentery, wall of duodenum, ovary or heart.
- Imaging of ZES : Endo USG
- Imaging for malignant ZES – Somatostatin Scintigraphy



Gastrinom Triangle

Treatment 00:16:15

- DOC – PPI (To heal ulcers)
- Lanreotide inj. (Long acting derivative of octreotide)
- Surgery – Resection > 1.5 – 2 cm
  - ↑ Ca2+2° ↑ PTH (Imaging OC to locate PTH adenoma

SESTAMIBI SCAN Technetium 99 scan)
(Technicium 99 pertechnetate – to identify Merkel's diverticulum)

- Hepatic Metastasis → Hepatic Wedge Resection.

65 MALABSORPTION SYNDROME

Introduction 00:00:13

- In Pancreatic tumors like Insulinoma, there is → secretory diarrhea, which is Non – responsive to fasting
- In Malabsorption syndrome: due mucosal damage.
- Unabsorbed Carbohydrate draws out H2O → leading to osmotic diarrhea.
- Therefore During Fasting →↓ed osmotic load →↓ diarrhea severity
- Damage to mucosa of:
  - Duodenum → Absorption of Fe ↓
    → S. Ferritin ↓
  - Jejunum →↓ Absorption of FA
    → RBC folate levels
    → Urinanry FIGLU
  - Ileum→↓ Absorption of Vit B12
    → Schillings test
    → S. Vit B12↓
    → S. Homocystiene↑
    → Methylmalonic Acid ↑
- There is also ↓ fat soluble Vitamins (A, D, E, K) due to steatorrhea

CARBOHYDRATE MALABSORPTION 00:05:56

- D. Xylose absorption test - Screening test
- 14-C - D – Xylose Breath test
- Stool for reducing substances
- Breath H2 test
- Smell intestinal mucosal biopsy – IOC

FAT MALABSORPTION 00:09:56

- Steatorrhea (Greasy, bulky, foul smelling stools)
  - Breath triolein test
  - 24 hr fecal fat estimation

CELIAC SPRUE/ GLUTEN SENSITIVE ENTEROPATHY 00:12:47

- Exclusive breast feeding – 6 months
- Prelacteal feeds :contraindicated
- So symptoms arise after 6 months when complimentary feed is initiated.

Pathophysiology:

- Cereals (gluten / gliadin in BROW leads to production of antibodies(ATTA)

↓

Anti-tissue transglutaminase antibody

↓

Microvilli damage leads to reduction of absorbtive surface area

↓

Osmotic diarrhea

BROW
B - Barley
R - Rye
O - Oats
W - Wheat

C/F:

- > 6 months
- Failure to thrive (Signs of malnourishment)
- Persistent diarrhea
- Pallor (+)
- Delayed milestone
  - Microcytic Hypochromic anemia (due to Fe ↓)
  - Macrocytic anemia (due to folate deficiency)
- Disease affects Proximal parts of the intestine more commonly, hence, Microcytic anemia is more common

Screening

- Anti TTG antibody (screening test)
- Anti endomysial antibody
- Anti gliadin antibody.

IOC

- Small Intestine mucosal biopsy which is repeated again after 4-6 weeks.
  - Shows Blunting of villi/villous atrophy
  - Gluten Elimination
    ↓ 4-6 wks
  - Repeat Biopsy → Regeneration of Villi

Rx

- QUINOA (Cereal)
- Maize

- (Rice-gluten in minimal amount-more practical in India)
- Strict diet restriction
- Important to continue with follow up
- Severity ↓ after 10 years of age

Follow up of celiac sprue to detect development of following

1. Dermatitis Herpetiformis
   - Pruritic vesicles on Elbows & Knees (Vs. Gottron Papule – Seen on Knuckles – Dermat. Myositis)
   - Anti-Epidermal Transglutaminase Ab
   - Biopsy shows break in basement membrane with complement deposition.
2. T1DM (HbA1C > 6.5)
3. May develop lymphoma of gut and happens to be MCC death in celiac sprue.

TROPICAL SPRUE

00:33:24

Causes

- Coliforms – E. Coli/ Giardiasis
- Recurrent infection leads to → Mucosal injury)
  - Osmotic contents remains in gut for relatively longer duration
  - Osmotic Diarrhea

C/F : Adult

- Osmotic Diarrhea : Bloating, borborygmi
- Wt. loss
- Steatorrhea
- Def of Vit.
  - A → Nyctalopia
  - D → Bone Pain
  - E → Ataxia Acanthocytes
  - K → Purpura
- Def. of B. complex – stomatis, gingivitis
- Muscle weakness 2 electrolyte imbalance.
- Edema 2↓ Albumin
- IOC is Small intestinal mucosal Biopsy
- Diagnostic criteria for tropical sprue
  - 2 product malabsorption (carbohydrate & Fat) present with mucosal biopsy showing villous atrophy
  - H/o travel to developing countries
- Rx – Tetracycline + folic acid supplementation.

WHIPPLE'S DISEASE

00:41:36

- Cause: Tropheryma Whippelii (Intra cellular bacteria In macrophages of gut)
- C/F-Adult : Same as tropical sprue+ protein losing enteropathy leading to hypoalbuminemia
  - CNS – Dementia, nystagmus, Seizures
  - CVS – Aortic Valve disease

IOC

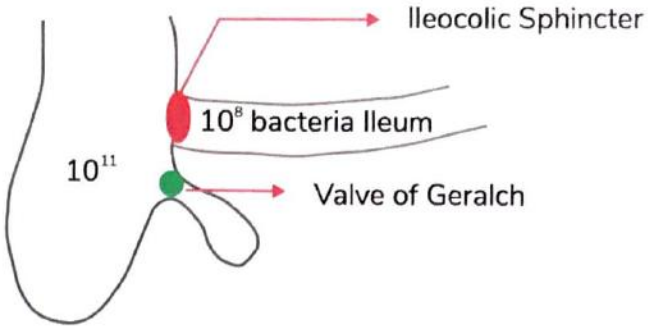
- Small intestinal mucosal biopsy
  - PAS (+) intracellular bacteria
- PCR [α1 antitrypsin def.-PAS (+) inclusion in hepatocytes]

Rx

- IV Ceftriaxone (at least for 2 weeks)
- Oral Cotrimoxazole: 6 months

BACTERIAL OVERGROWTH SYNDROME

00:48:06



- If Ileocolic sphincter malfunctions due to TB, typhoid, Crohn's→ large intestinal flora move into Ueum causing mucosal damage and bile acid malabsorbtion
  - Vit B12↓
  - Folic acid↑(Bacterial metabolism produces folate compounds)
- Clinical features: Adult presenting with abdominal Bloating, weight loss, bile acid diarrhea

IOC :

- Jejunal Aspiration Culture
  - >1011 organisms/ ml
  - Breath H2 test
  - Breath Triolein test

SCHILLING TEST

00:56:41

- If Abnormal schilling test shows, normalization with a short course of metronidazole →SIBO
- If Ab (n) Schilling test shows normalization with pancreatic enzymes supplementation→Chronic Pancreatitis
- If Ab (n) Schilling test shows normalization with supplementation with intrinsic factor → Type A gastritis

Celiac sprue	SI mucosal Biopsy
While disease	SI mucosal Biopsy shows PAS EB inclusions ingut macrophages
SIBO	Jejunal aspirate and culture showing greater 10^{11} organism / ml

- Intestinal Lymphangiectasia
  - Malformation of lymphatic channels in the gut

- Steatorrhea
- Schilling's test (n)
- D-xylose test (n)

- Diagnosis: Biopsy - abnormal dilated lymphatics

Results of diagnostic Studies in different causes of Steatorrhea

	D-Xylose test	Schilling Test	Duodenal Mucosal Biopsy
Chronic pancreatitis	Normal	50% abnormal; if abnormal, normal with pancreatic enzymes	Normal
Bacterial overgrowth syndrome	Normal or only modestly abnormal	Often abnormal; if abnormal, normal after antibiotics	Usually normal
Ileal disease	Normal	Abnormal	Normal
Celiac sprue	Decreased	Normal	Abnormal: probably "flat"
Intestinal lymphangiectasia	Normal	Normal	Abnormal: "dilated lymphatics"

66 INFLAMMATORY BOWEL SYNDROME

Introduction 00:00:14

- Not an Auto immune disorder.
- It is an inflammatory disorder with misfiring of the immune system that leads to gut inflammation

CROHN'S DISEASE 00:00:20

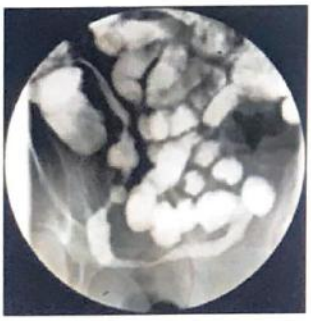
- MC Site: Terminal ileum
- Sparing: Rectum
- Earliest Presentation: Apthous ulcers. They progress to cause deep serpiginous ulcers in entire gut
- Serpiginous ulcers in esophagus: CMV
- Transmural involvement and Submucosal fibrosis leads to Irregular appearance of mucosa called as cobble stone pattern. Occasional sparing is called skip lesions.

Clinical Features 00:11:19

- Colicky pain due to formation of multiple structures
- Bile acid diarrhea due to damage to ileum
- Hematochezia (more pronounced in UC) 2°damage to BV
- Toxic megacolon (Bowel Loop > 6 cm size – M/C involving transverse colon).
- Intraabdominal abscess
- Adhesions (between bowel wall and fallopian tube)
  - Infertility
- Fistula (Hallmark feature of Crohn's)
  - Perianal Sepsis
  - Vesico colic fistula → cystitis
  - Entero cutaneous fistula → Opens into ant. Abdominal wall

Work-up 00:19:33

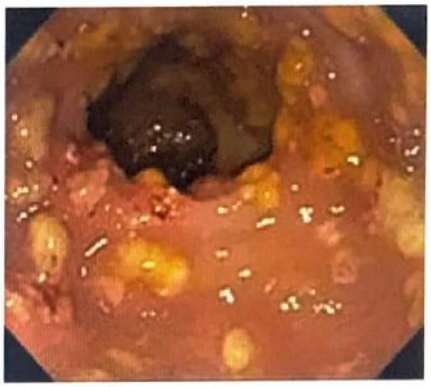
- ASCA – Anti Saccharomyces Cerevisiae Antibody
- Imaging – Capsule endoscopy / Upper GI endoscopy/ Colonoscopy + Biopsy (Granulomas are seen)
- CT Enterography
- Ba meal follow through (Enteroclysis)
  - "String Sign of Kantor" due to stricture formation



Rx 00:23:14

- DOC for CD: Steroids, Mesalamine
- Bile acid diarrhea: Cholestyramine/ Colestipol
- Sulphalazine Bacteria → 5 ASA (Amino Salicylic Acid)
  - (Local anti-inflammatory action to heal apthous ulcers)
  - Works in large bowel but not in the small bowel due to lack of bacteria
- Azathioprine
- Infliximab
  - (TNF – α antagonist): Used for healing of enterocutaneous fistula.
  - 1st step in management of perianal fistula maintain perineal hygiene and use antibiotics

PSEUDO MEMBRANOUS COLITIS 00:27:44



- Cause – Clostridium difficile toxin (When antibiotics have been taken in then last 3 weeks) leading to Alteration in gut flora
- Antibiotic M/C causing PMC – Cephalosporins > Clindamycin
- C/F: Explosive watery diarrhea, cramps, abdominal pain

Work-up 00:30:01

- Stool culture
- Stool ELISA – Glutamate dehydrogenase (most sensitive)
- IOC – PCR for clostridium difficile: Toxic A,B
- Stool ELISA for Toxin AColonoscopy Colonic mucosa → exudates of 2-5 mm coalesce →Pseudo membrane.
- HPE: Mushroom cloud extruding above damaged colonic mucosa

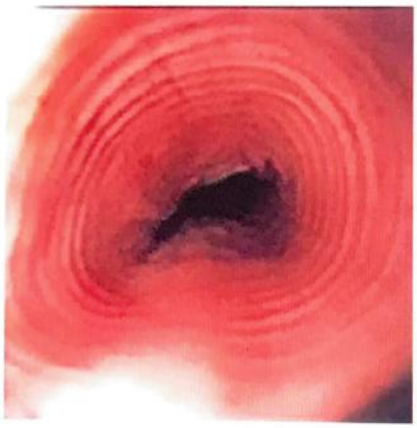
Rx 00:34:50

- Vancomycin oral (Best)

- Metronidazole
- Fidaxomicin
- Teichoplanin
- Recurrent PMC – Fecal Transplantation (fecal microbiota transplant)

Important Information

- UGIE Esophagus
  - "Feline Esophagus" seen in
- Eosinophilic esophagitis Biopsy > 15 eosinophils/ HPF



ULCERATIVE COLITIS

00:38:59

- "Pseudopolyps" Partial Thickness involvement
- + Pan Colitis
- M/c site – Rectum

Clinical features

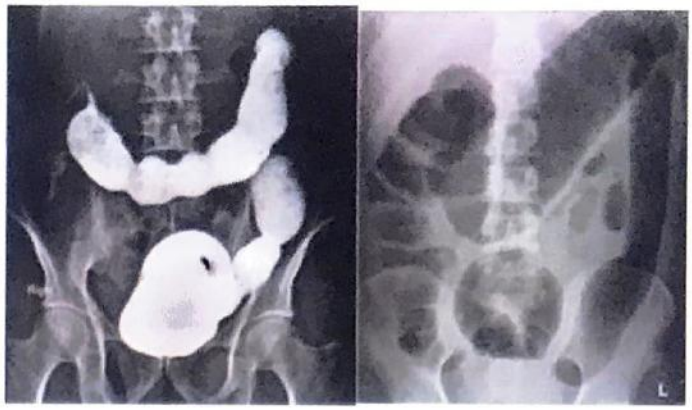
00:41:50

- Painless bloody diarrhoea
- Anemia
- Protein Losing enteropathy (Albumin ↓ : Puffy eyes)
- Toxic megacolon (colon loop dilation> 6cm) – [UC > CD]
- Malignancy incidence in UC is equal to that in Crohn's disease

Work-up

00:44:30

- p- ANCA
  - Also in
    - → Primary Sclerosing Cholangitis
    - → Microscopy Poly Angitis
- IOC : Colonoscopy – Proctitis + Biopsy (Granulomas) are not seen.
- CT Enterography
- Ba. enema – (Earliest features is Granular appearance due to pseudopolyps)
  - Later – loss of Haustrations leading to PIPE STEM COLON appearance.



- TOXIC MEGACOLON PIPE STEM COLON on Barium enema
- Fecal Calprotectin levels help to differentiate IBD vs IBS (No Blood in stool)

Rx:

- DOC of UC – Sulphasalazine
- DOC of UC exacerbation – Budosonide enema
- Infliximab infusions in case of disease progression.

Indication for Sx in UC

1. Toxic megacolon
2. Rectal mucosa friable – Bleeds on touch

- Surgery of choice: Proctocolectomy + ileal pouch Anal Anastomosis (IPAA)

EXTRA INTESTINAL IBD

00:54:24

- MC – Arthritis (Migratory Polyarthritis)
  - HLA B27 (+) – Sacroiliitis (Also in AS, PsA, JRA, Reiter's syndrome, IBD)
- Hepatobiliary – PSC (UC > CD)
  - Extrahepatic fibrosis → Intrahepatic fibrosis
  - Biliary Atresia
  - Obstructive Jaundice
- Dermatologic – Erythema Nodosum over Shin (also seen in Sarcoidosis)
  - Sweet syndrome – febrile neutropenic dermatitis (Upper back)
- Clubbing
- Osteoporosis
- Eye manifestations – Scleritis

Summarize

UC > CD	UC = CD	CD > UC
• Toxic megacolon • PSC	• Malignancy • Death	• Extra-intestinal manifestation

67 IRRITABLE BOWEL SYNDROME

Introduction

- Rome IV criteria:
- C/f: Recurrent Abdominal Pain > 1 day/ week for min 3 months with any 2 or more of the following
- Others

1. Associated with defecation
   - Passage of flatus with fecal matter → relief of pain
2. Associated with change in frequency of stool
   - 33% - diarrhea/ constipation (varies with time/ season)
3. Associated with change in appearance of stool
   - If a patient of < 45 years age arrives with 2/3 of the above for a duration > 3 months, IBS should be considered.
   - It's a functional disorder.

IBD Vs IBS

00:03:40

IBD	IBS
• increased Fecal calprotectin levels	• Normal range
• Bleeding always (+)	• Not seen
• Sleep deprived due to colicky pain	• Rare
• Malnutrition seen	• Not seen
(-)	• Pain ↑ anxiety
(-)	• Exaggerated with menses in female
(-)	• Nocturnal diarrhea (+)

- If pain occurs 2 hrs post prandial at epigastrium → suspect duodenal cause.
- If pt C/o diffuse abdominal Pain (Post Prandial) → Suspect Abdominal angina

Abdominal angina – characterized by Narrowing of both Sup. & Inf. mesenteric artery
↓
↓ed blood supply to bowel
↓
No relief with defection & no changes in stool

Why?

- Dysbiosis 00:09:24
  - Flora of the gut is altered.
  - Firmicutes:(F:B Ratio) in IBS ↑es
  - Bacteriodetes: (F:B Ratio) in IBS ↑es
- In Pseudomembranous colitis, Clostridium defficile produces enterotoxin A&B which leads to manifestation
- Genetic
- Environment

Altered gut permeability
↓
↑ antigen presentation
↓
Mast cell activation
↓ Cytokines
Altered enteric Neuronal function (Diarrhea/ constipation)
Smooth muscles activation (colicky/ crampy pain)

Clinical features

00:13:53

- Age group: < 45 yrs.
- Constipation with poor response to laxatives Stool
  - Hard & Narrowed caliber.
  - Incomplete evacuation (anxious about need to use toilets)
- Diarrhea < 200 ml/ day
  - Nocturnal Diarrhea not seen
  - Bleeding from gut is not seen. Belching/ Bloating/ Borborgymi
- Nausea/ Vomiting/ Dyspepsia
- Post prandial pain

Investigations

00:17:56

- CBC (Anemia (+) in IBD, (-) in IBS)
- Stool for parasites
- Sigmoidoscopy with biopsy (r/o microscopic colitis)
- Colonoscopy
- Breath H2 test (can be false (+)ve)
- Anti – TTG Antibody titer

Treatment

00:19:34

- Diet low in FODMAPs
  - Fermentable
  - Oligosaccharides
  - Disaccharides
  - Monosaccharides

- Polyols
- Constipation
  - Tegaserod(5 HT4 Receptor Agonist)
    → (+) Peristalsis
  - Lubiprostone
    → Cl- Channel activator
    → Passive Na+ loss & H O loss to maintain soft stools.
  - Linaclotide: Guanylate cyclase C agonist acting on luminal surface of enterocytes
    → ↑ GI motility &↓ nociception
- Antispasmodics – Dicyclomine
- SSRI – Paroxetine, Alosetron (↓ perception of gut motility as pain)
- Ritaximicin, Neomycin – Bacteriostatic Antibiotics Prebiotics – ↑ activity of good bacterial
- Probiotics – Live microbes
  - Bifidobacterium
  - Lactobacillus
- Diarrhea – Loperamide, Cholestyramine
- High fiber diet – Bran, Psyllium

PREP NUGGETS

Prep Nuggets

Auscultatory findings	Cardiac condition
_____	ASD
Continuous machinery murmur (Gibson murmur)	_____
	Pericardial effusion
Narrow variable split S2	_____
	VSD
No shunt murmur with mid-diastolic shunt murmur	
	Mitral stenosis
Early diastolic murmur	

Prep Nuggets

High risk factors for Sudden cardiac death in HOCM

1.

2.

3.

4.

5.

Major criteria

Low-risk populations	Moderate-and high-risk population
Carditis Clinical and/or subclinical carditis	Carditis Clinical and/or subclinical carditis
Arthritis?	**Arthritis?**
Chorea	Chorea
Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules

Minor criteria

Low-risk populations	Moderate-and high-risk population
Arthralgia?	**Arthralgia?**
Fever (>38.5°)	Fever(>38°C)
ESR ?	ESR ?

Prep Nuggets

Metabolic syndrome

1. Fasting plasma glucose level _____
2. Blood pressure _____
3. Fasting triglyceride level _____
4. HDL cholesterol level _____
5. Waist circumference _____

Prep Nuggets

	Serum Ca	Serum phosphorus	Alkaline phosphatase	PTH
• Osteomalacia (Rickets) (↓Vit D)		↓		↑
• Primary hyperparathyroidism bone disease	↑		↑	
• Bone disease in renal failure with secondary hyperparathyroidism		↑		↑
• Lytic bone neoplasms	N/↑		N/↑	
• Osteoporosis		N		N
• Paget's disease		N	↑	