



CARDIOLOGY

WORKBOOK

By

DR. PRIYANSH JAIN





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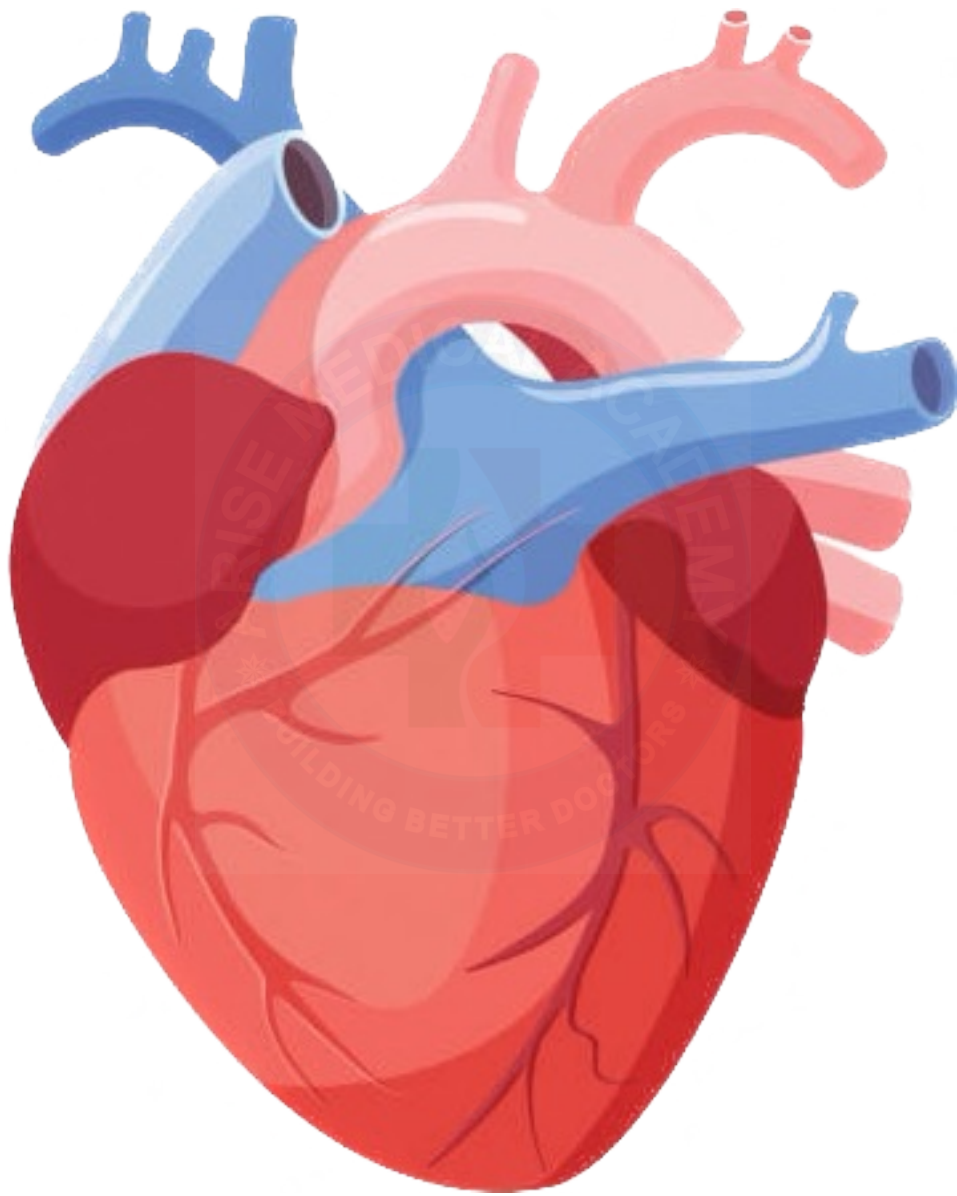
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Cardiology



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DR. PRIYANSH JAIN



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- MBBS (Gold Medalist)
- MD (General Medicine) – JLN Medical college
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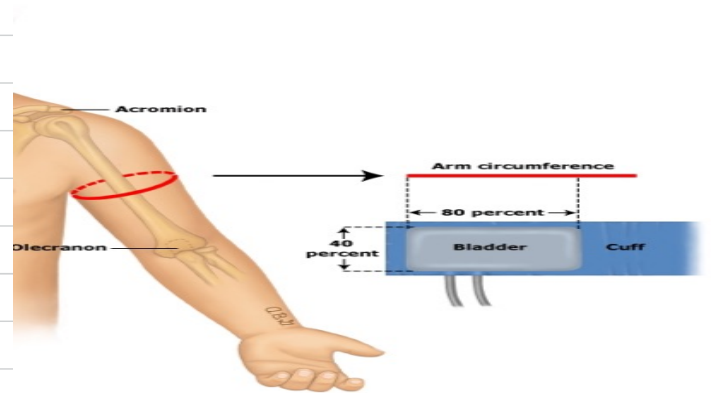
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CHAPTER-1 HYPERTENSION

DEFINITION:

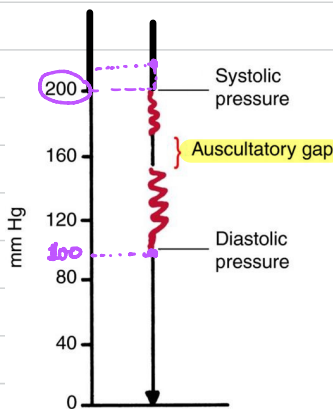
Bladder Size

- width - **40%** of arm circumference
- length - **80%** of arm circumference

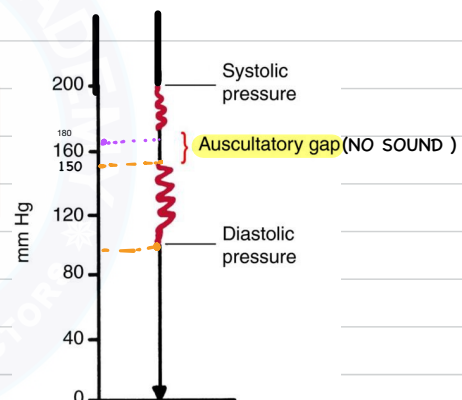


If cuff size is smaller than required - **false high**

- Deflation rate ≤ 3 mm of Hg/sec.



BP- **200/100**



BP- **150/100**

So, auscultatory gap can lead to $\rightarrow$ **false low SBP.**

TABLE 277-1 Blood Pressure Classification in Adults

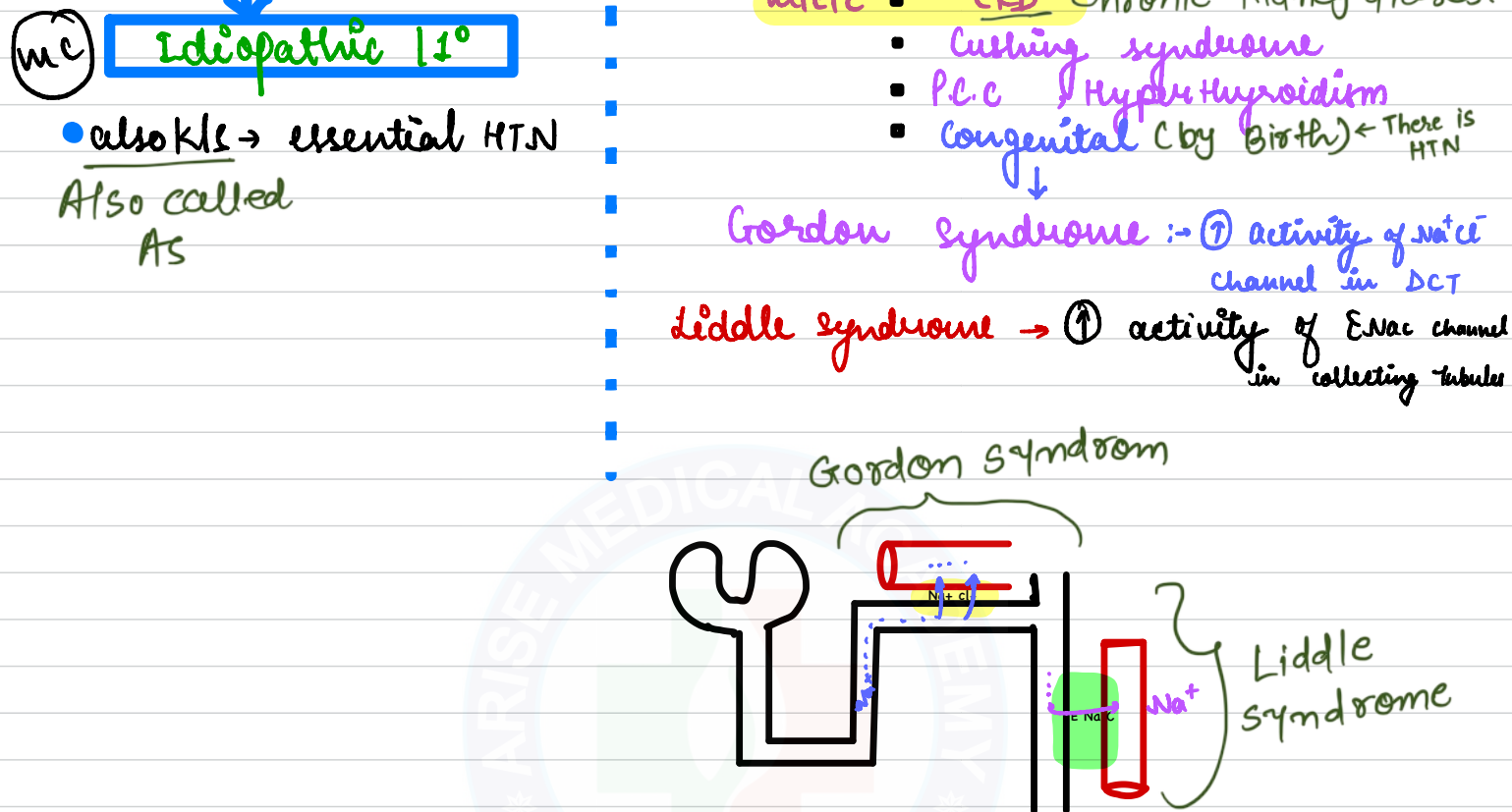
BLOOD PRESSURE CATEGORY	SYSTOLIC (mmHg)		DIASTOLIC (mmHg)
Normal	<120	and	<80
Pre-HTN	120-129	and	<80
Hypertension			
Stage I	130-139	or	80-89
Stage II	≥ 140	or	>90

- If only increase in systolic or diastolic BP- **Isolated HTN.**

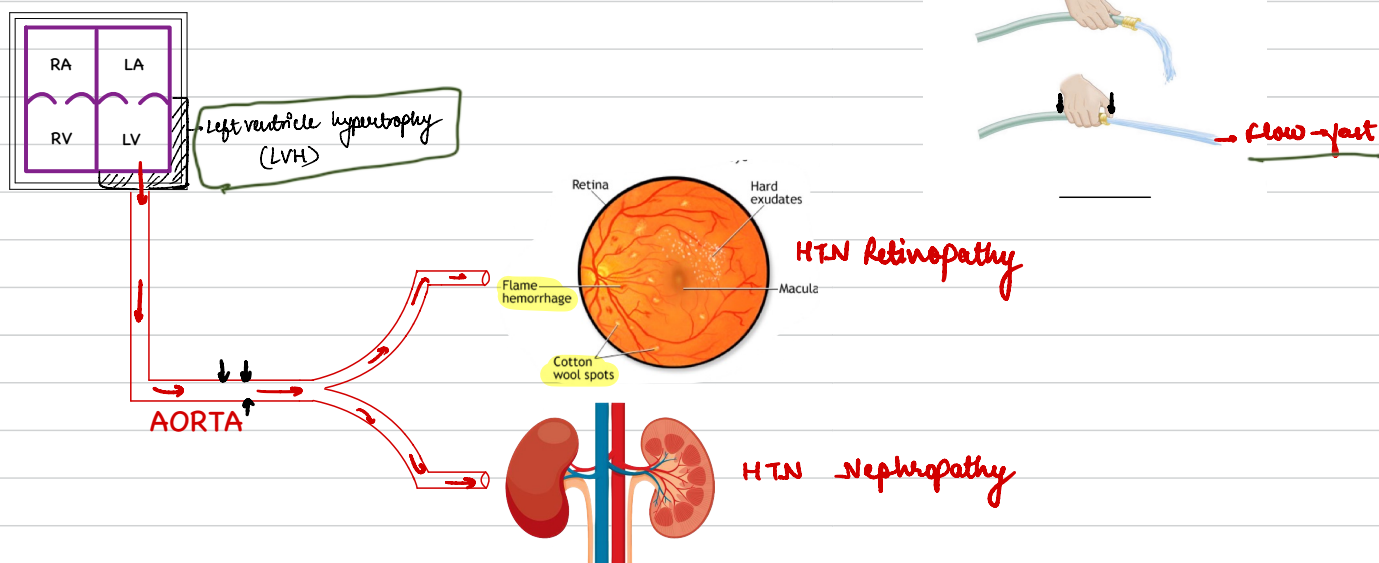
- Cause of **Isolated**

- **Systolic HTN**
 - ↳ Pheochromocytoma (P.C.C)
 - ↳ Hyperthyroidism
- **Diastolic HTN**
 - ↳ Conn. syndrome **CONN**
 - ↳ Liddle syndrome
 - ↳ Hypothyroidism

CAUSES OF HYPERTENSION

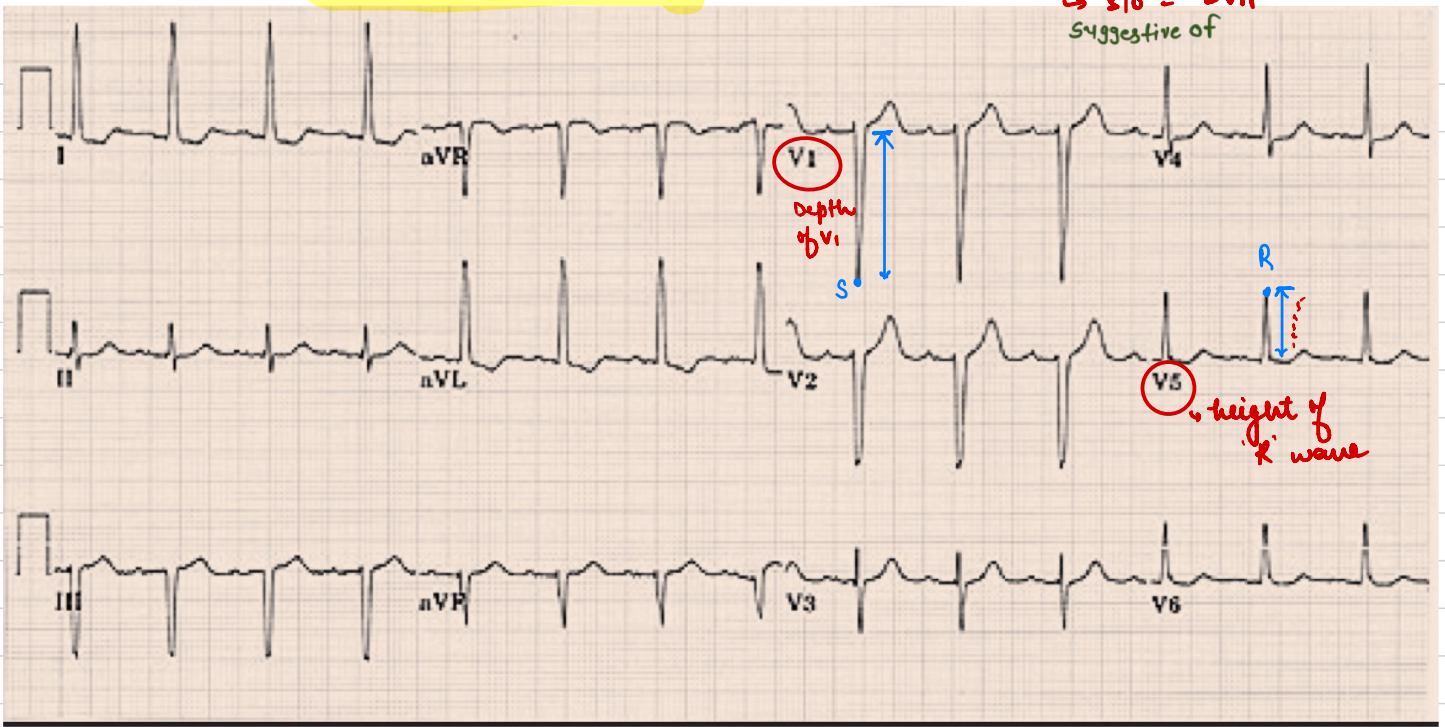


TARGET ORGAN DAMAGE

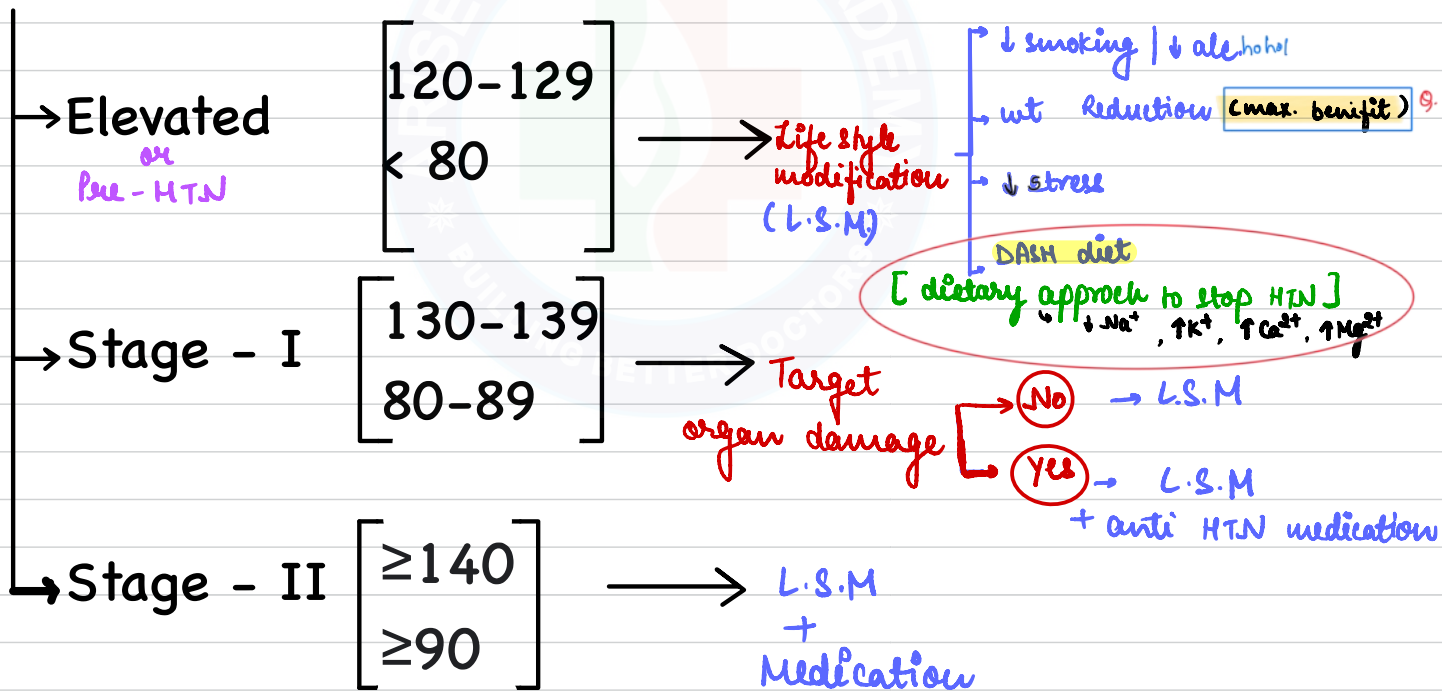


ECG → Sokolow law = $V_1S + V_5R = >35$

↳ $S/R = LVH$
Suggestive of



MANAGEMENT OF HYPERTENSION



Extra point

- HTN After 20 week of pregnancy + proteinuria - Pre-eclampsia
- HTN After 20 week of pregnancy + proteinuria + seizure - eclampsia
- Tumor of adrenal medulla - P.c.c. pheo chromo cytomet.

ANTI-HTN MEDICATION - ABCD³ Mnemonic

A-Angiotensin converting enzyme inhibitors [ACE#] (in young age grp. we prefer these grp.)

- Eg: Ramipril
- Additional effect: → ↑ insulin sensitivity ↓ Proteinuria
- So, preferred in: → DM, Nephrotic syn., CKD
(= HTN) (nephrotic)
- S/E → dry cough

-Angiotensin Receptor Blocker [ARB] (in young age grp. we prefer these grp.)

- Eg: Losartan
- Additional effect: ↑ insulin sen. ↓ Proteinuria
- So, preferred in: → same as ACE#

B- β-blocker

- Eg: Metoprolol
- Preferred in: coronary art. disease

C- CCB (Calcium channel Blockers)

- Eg: Amlodipine → old age
- S/E: ankle edema

D- Diuretic

- Eg: → Chlorthalidone (Preferred)
- S/E: Hyperglycemia / ↑ uric acid / ↑ lipid
[Hyper - GLU]
↑
Mnemonic

65yr.  depressed

Ist visit	IIInd visit	IIIrd visit	IVth visit
BP-164/94 →	BP-158/90 →	BP-150/90 →	BP- still not controlled
Rx- CCB	Rx- CCB + β-#	Rx- CCB + β-# Diuretics	

Δ - If BP not controlled despite 3 classes of anti-HTN drug including diuretic.

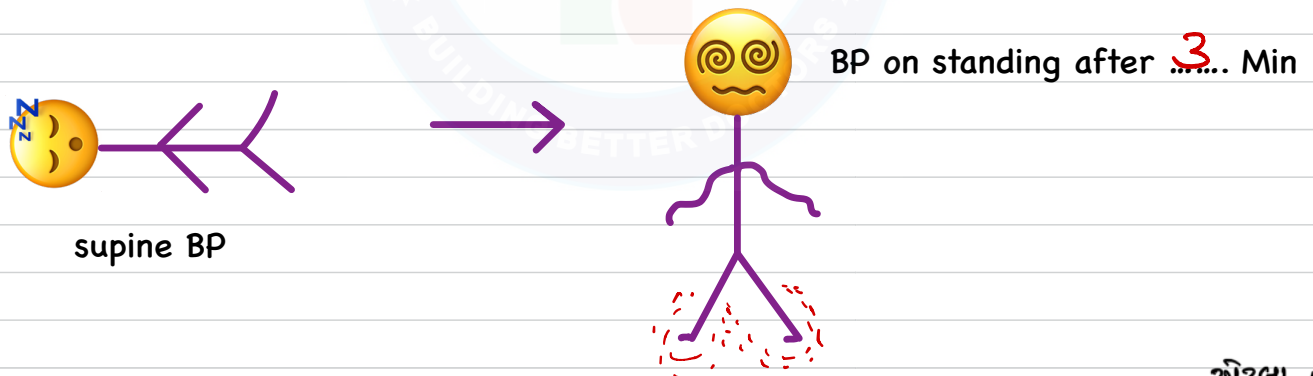
called AS C/a- Δ - Resistant HTN → Wk/c → Non-compliance Pt દવા સરખી નો ખાતો દીધ.

Rx- spironolactone ↓

→ clonidine
→ prazosine
→ α-methyl dopa] ① sympathetic activity.

↓ S18

ORTHOSTATIC HYPOTENSION OR POSTURAL HYPOTENSION



If SBP falls : >20mmHg
or
OR DBP falls : >10mmHg

એટલા mm Hg
ફાલ વાળ ને
supine
position માં
ડોઇ ત્યાં પછી
જોખો પણ પછી એટલું ઓછું વાળ ત્યારે
એ ૧૫ mm.

Rx of Orthostatic Hypotension: → Midodrine
MIDODRINE

HYPERTENSIVE URGENCY vs HYPERTENSIVE EMERGENCY

If SBP > 180...or /& DBP > 120...

Target organ damage

Absent

HYPERTENSIVE...urgency

Control the BP in next 48 Hrs

Present

HYPERTENSIVE...Emergency

Associated disease

- 1 p.c.c
- 2 pre-eclampsia
- 3 eclampsia
- 4 Aortic dissection

Control the BP in next 1 Hr

Rx - inf → Nicardipine | Labetalol
(निकार्दिपिन) (लेबेतालोल)

But next 1 hr → ↓ MAP by 25%

Not also
in the
4 disease

Control the BP
in next 24 Hrs

MALIGNANT HYPERTENSION

If BP more than 220/120

+
Papilloedema

Associated with → Fibrinoid necrosis

Onion Peel Appearance



Normal

Papilloedema

↑ ICP
Intra
cranial
Pressure



SUMMARY FOR HYPERTENSION

Deflation rate = <3 mm Hg/sec. (<2 / <3 / <4 / <5)

If small cuff is used = false high BP. (false high / false low)

HTN pt. taking Allopurinol which anti-HTN should be avoided diuretic. (ACE# / ARB / Diuretic / B-blocker)

Allopurinol use → Gout (Uric Acid ↑)
(Uric Acid ↑ & Diuretic on S/E @)

DM+HTN — ACE #. (ACE# / ARB / Diuretic / B-blocker)

(more potent)

HTN+Proteinuria — ACE #. (ACE# / ARB / Diuretic / B-blocker)

(more potent)

Post MI — HTN — Rx 1st line- β-Blocker

2nd line- ACE #

BP+Papilloedema — c/a — Malignant HTN

Mean Arterial Pressure = $\frac{2}{3} \text{ DBP} + \frac{1}{3} \text{ SBP}$ [$\text{DBP} + \frac{1}{3} \text{ PP}$]

Pulse Pressure — SBP - DBP

High pulse pressure — Aortic regurgitation

Low pulse pressure — Aortic stenosis

permissive HTN — Ischemic stroke

DOC for scleroderma crisis — (ACE# / ARB / Diuretic / B-blocker)

DOC for u/l renal artery stenosis — (ACE# / ARB / Diuretic / B-blocker)

↳ B/L renal artery stenosis → CCB [ACE# → C/I in B/L art. stenosis]

If HTN is due to excessive sympathomimetic activity such as the use of cocaine → Rx will be — CCB / Prazosin / ACE#

Rough → $\text{SBP} - \text{DBP} = \text{PP}$

$\text{MAP} = \frac{2}{3} \text{DBP} + \frac{1}{3} \text{SBP}$

$\frac{1}{3} \text{SBP} - \frac{2}{3} \text{DBP} = \text{PP}$

$\text{SBP} - \text{DBP} = \frac{2}{3} - \frac{1}{3} = \frac{1}{3}$

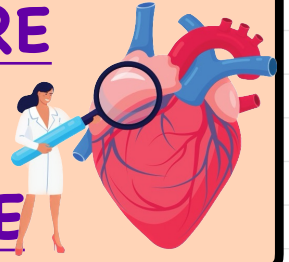
$\text{MAP} = \text{SBP} - \text{DBP} + \frac{1}{3} \text{PP}$

CHAPTER 2

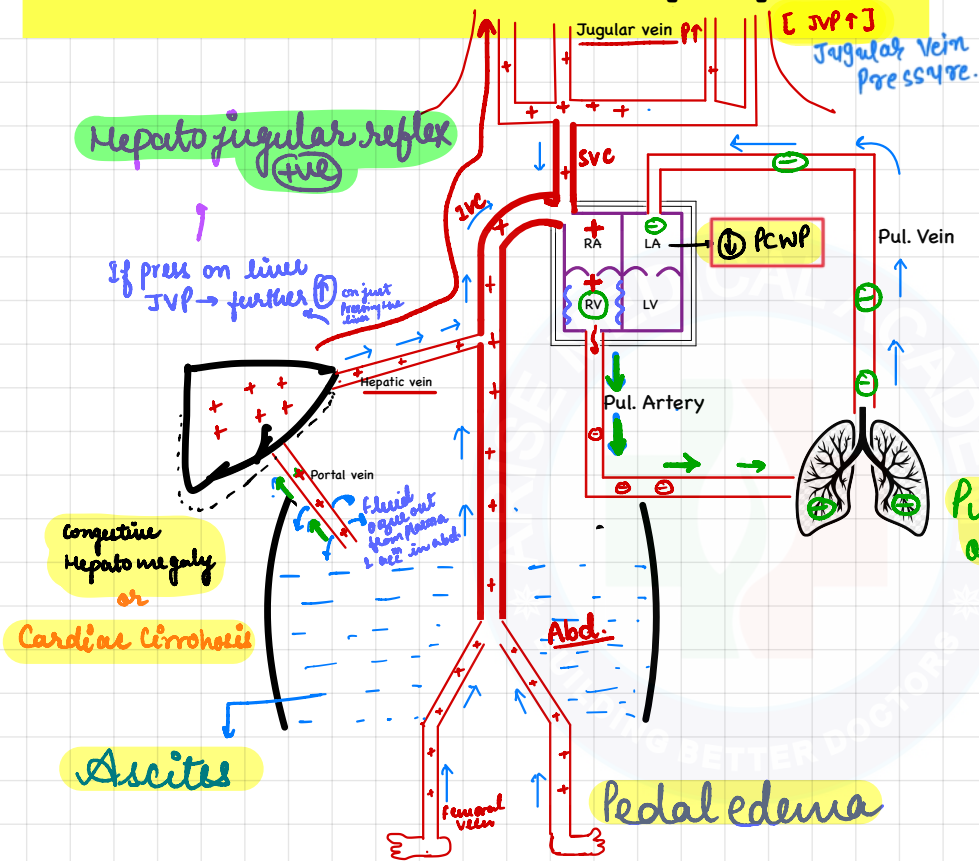
RIGHT VENTRICULAR FAILURE

VS

LEFT VENTRICULAR FAILURE



RIGHT VENTRICULAR FAILURE [RVF]



PCWP ⇒ Pressure of Left Atria
(Pulmonary capillary wedge pressure)

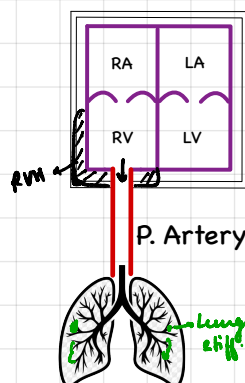


Cardiac output in RVF → ↓↓

PCWP — pulmonary capillary wedge pressure

COR PULMONALE

* So, Cor pulmonale is **low** cardiac output failure.



Chronic pulmonary pathology

Lung becomes stiff

Strain on ...RV...

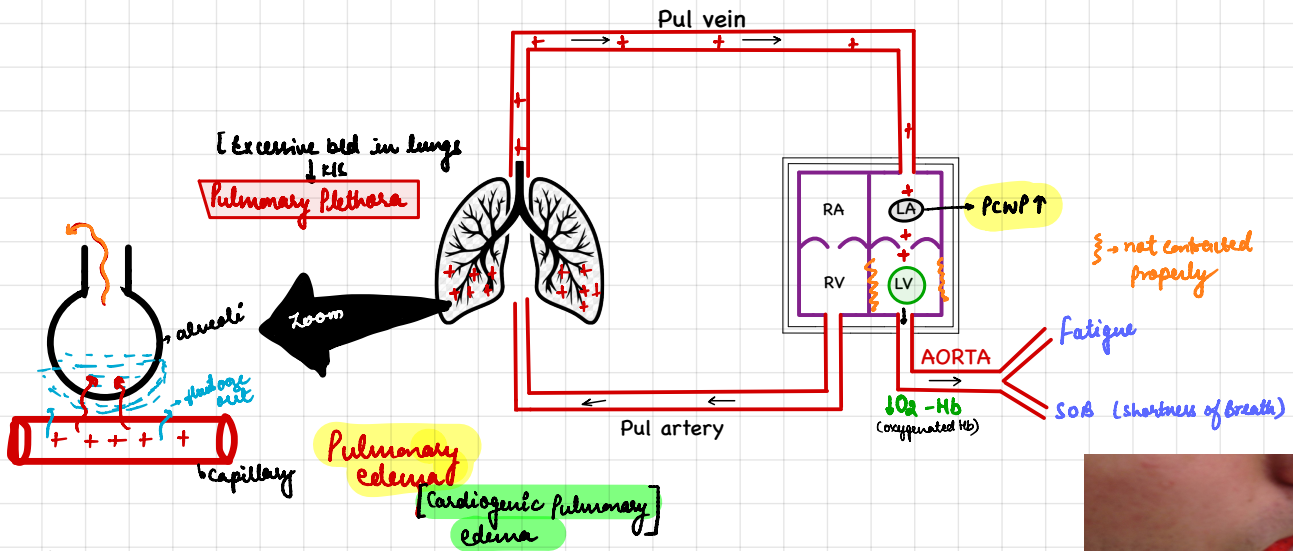
Leads to ...RVH...

ULTIMATELY ...RVF...

CALL AS ...Cor pulmonale...

(Right Ventricular failure)

LEFT VENTRICULAR FAILURE [LVF]



C/F → Cough & pink frothy sputum
Sob [dyspnea] → PND → orthopnea

On auscultation- lungs - B/L basal crepitation

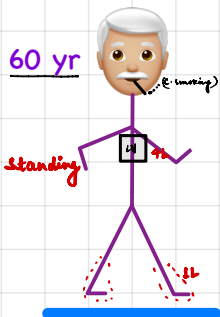
Heart - S₃ ⊕ [S₁, S₂ & S₃]
gallop rhythm

Pulse - pulses alternance

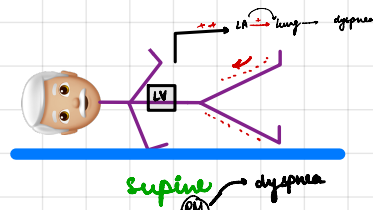
(dyspnea = shortness of breath)



PAROXYSMAL NOCTURNAL DYSPNEA [PND]



after 1 1/2 hr.

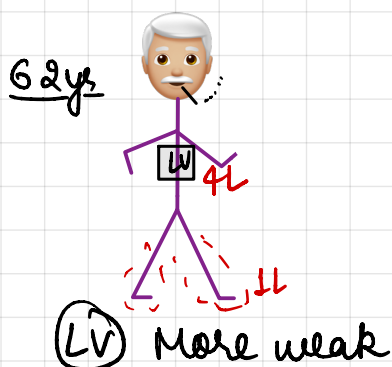


Uncontrolled HTN

↓
LV weak

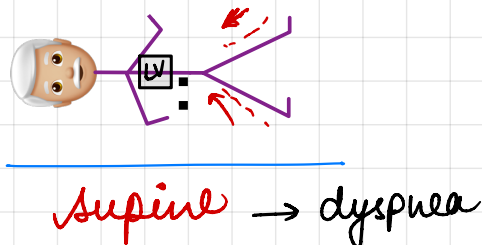
Paroxysmal → reverse
① → dyspnea → not at rest
In this pt's dyspnea happens on Resting position
↓
Paroxysmal dyspnea

ORTHOPNEA



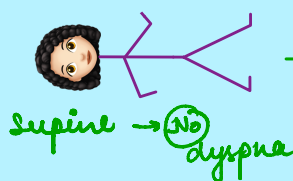
As soon as pt's go on supine position → dyspnea → orthopnea

as soon as



EXTRA POINT

PLATYPNEA



Platypnea is seen in → LA Myxoma
→ Hepato-pulmonary syndrome (HPS)



SUMMARY

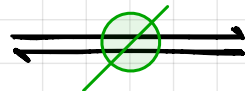
- ♥ Hepatojugular Reflex — RVF. (RVF / LVF)
- ♥ ↑ JVP — RVF. (RVF / LVF)
- ♥ ↑ PCWP — LVF. (RVF / LVF)
- ♥ P. alternance — LVF. (RVF / LVF)
- ♥ Hepatomegaly & Ascites — RVF. (RVF / LVF)
- ♥ PND & ORTHOPNEA — LVF. (RVF / LVF)
- ♥ Platypnea - seen in - Atrial myxoma, HPS.

CHAPTER 3 HEART FAILURE



DEFINITION

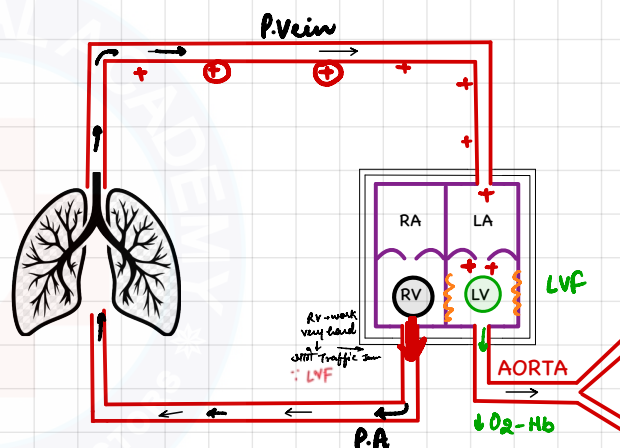
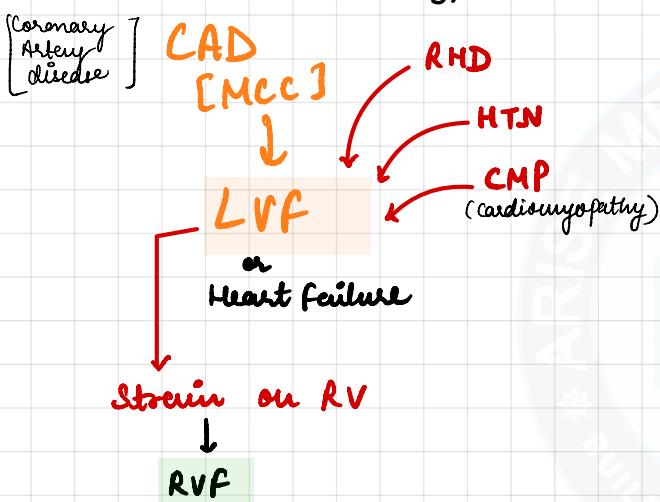
Pumping of oxygenated blood



Demand of body

ETIOPATHOGENESIS

Risk factor / ethology -



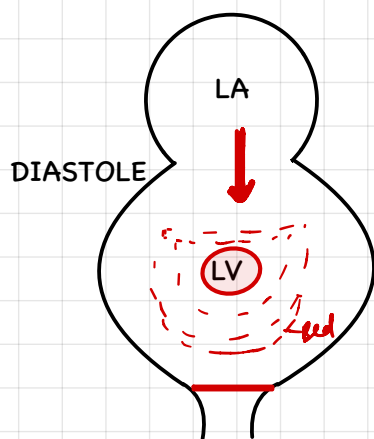
Q-) M/c of RVF → LVF
M/c of LVF → CAD

CONCEPT OF EJECTION FRACTION

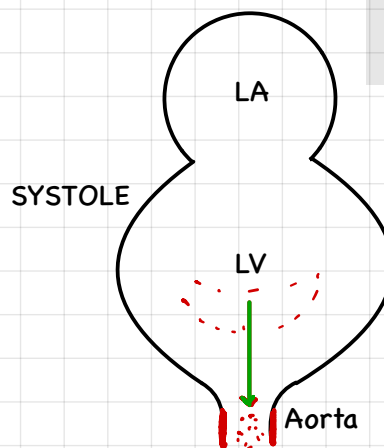
$$EF = \frac{SV}{EDV} \times 100\%$$

(Ejection fraction)

eg:- $\frac{60}{100} \times 100 = 60\%$

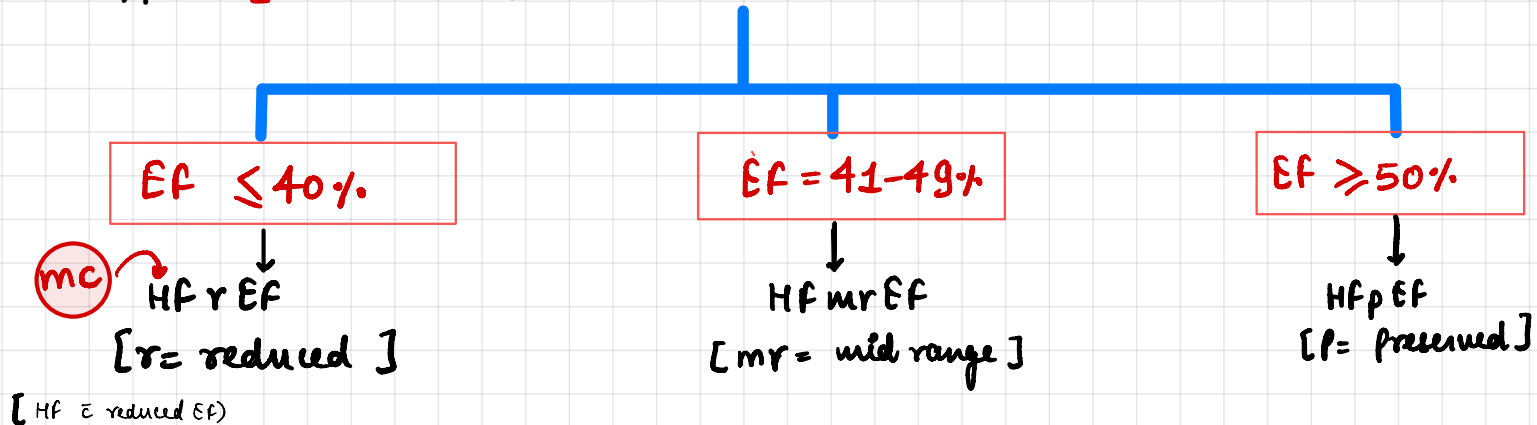


Amount of blood in LV at the end of diastole - **EDV**
[End Diastolic Volume]



Blood pumped in aorta during systole - **SV**
(Stroke Volume)

Type of ^{LVF} Heart Failure based on EF



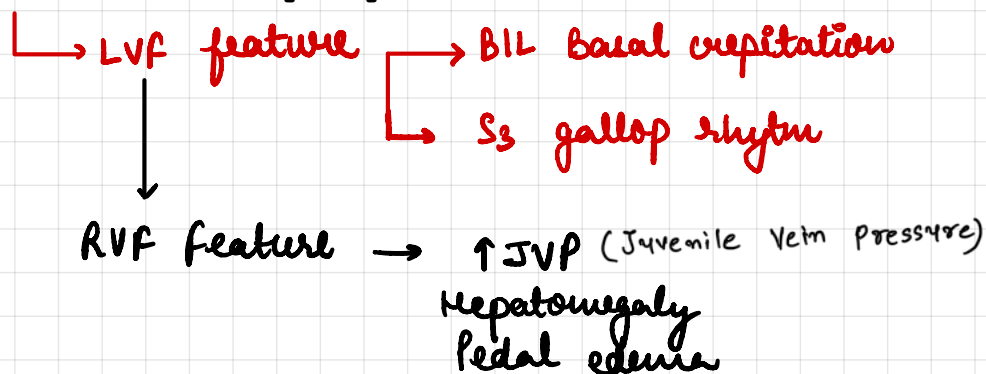
C/F → Same as LVF → SOB / fatigue

↓
RVF → distended neck vein / Abd discomfort → (eg. Hepatomegaly, Ascites)

★ NYHA [NEW YORK HEART ASSOCIATION] STAGE FOR DYSPNEA

NYHA CLASS	Level of Impairment
I ^{No dyspnea}	No symptom limitation with <u>ordinary</u> physical activity
II ^{and short or only on exertion}	<u>Ordinary physical activity</u> somewhat limited by dyspnea (eg: long-distance walking, climbing flights of stairs) ^{and 4 floor stairs}
III ^{same short or only on exertion}	Exercise limited by Dyspnea with <u>moderate workload</u> (eg: short-distance walking.)
IV	Dyspnea at <u>rest</u> with very little exertion

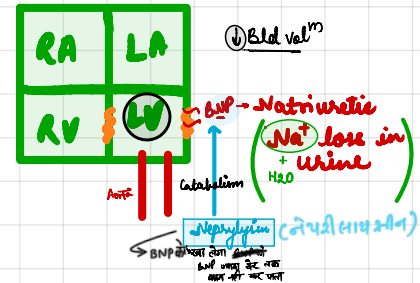
Objective Examination [O/E]



Investigation

Marker of HF - **BNP** (Brain Natriuretic Peptide)

ECHO -
↓
sonography of heart
→ LV dysfunction
→ EF (Ejection Fraction)



± STAGES OF HEART FAILURE

Risk factor for heart failure - **Smoking** | DM | HTN | family Hx
↓
is never reversible.

	STAGE A	STAGE B	STAGE C	STAGE D ^{Refractory HF}
RISK FACTOR	⊕	⊕	+	+
ECHO CHANGE	Normal	+ LVH/↓EF	+	+
C/F	asymptomatic	asymptomatic	dyspnea	dyspnea at rest
NYHA	I	I	II III	IV

MANAGEMENT OF HEART FAILURE

- L.S.M →
 - ⊙ H₂O intake
 - ⊙ Na⁺ [salt] intake
 - ⊙ exercise

■ Medication → **AB**

ⓐ **↓ Mortality of HF**
means **↑ survival**

ⓑ **↓ symptom of HF**

A → ACE #
→ ARB
→ Aldosterone-rec. antagonist [spironolactone]
→ ARNI → ARB + NI [neprilysin #]
↓ angiotensin II receptor
Valsartan + Sacubitril

C → ↑ Contractility → Digoxin
D → Diuretic
dilator [Nitrate]

B → β# → Metoprolol

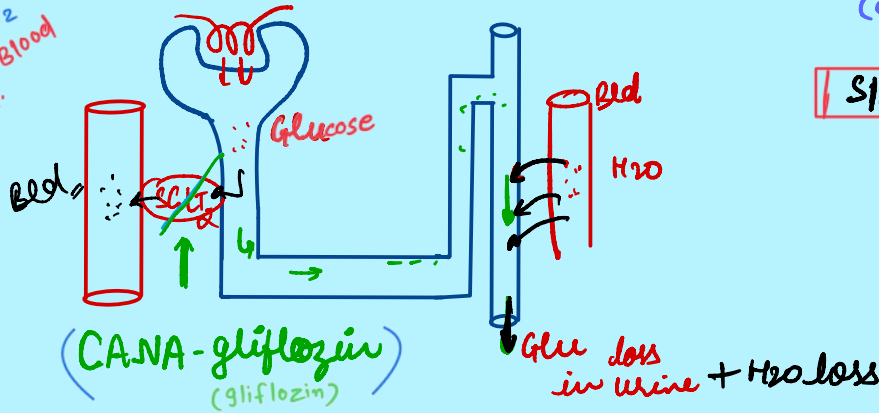


EXTRA POINT

If - DM+HF → Preferred anti-diabetic drug → SGLT-2 #

(eg. canagliflozin, empagliflozin)

S/E → Ⓢ risk of UTI



HF+Atrial fibrillation [AF] → Digoxin (↑ contractility)

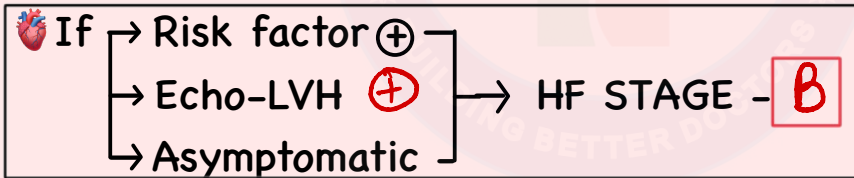
(Atrial many contract
तो सब 216-11 5121)



SUMMARY OF HEART FAILURE

MCC of LVF- ... CAD ... (HTN / RHD / CAD / CMP / RVF)

MCC OF RVF- ... LVF ... (HTN / RHD / CAD / CMP / LVF)



Drugs which ↑ survival in HF

A → ACE # / ARB / ARNI
→ Ald. Recp. antagonist → Spirinolactone

B → β # → Metoprolol

Cause of death in HFpEF → Cardio or Non-Cardio

HFrEF → Cardio or Non-Cardio

* ACUTE DECOMPENSATION OF HEART FAILURE

Sudden Worsening

Definition: Sudden worsening of HF

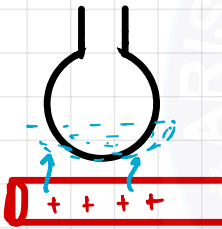
500ml → 200kg
1000ml → 300kg
Gogobhgh.

100% fluid = Power
500ml → 200kg
1000ml → 300kg
Gogobhgh.

Goma Singh
- 55yr/M

- Ch. smoker / LVH
- Uncontrolled HTN
- No dyspnea

HF Stage B



C/F →

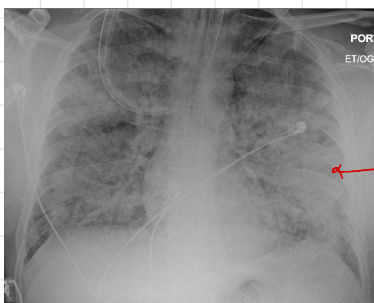
Cardiogenic Pulmonary edema

- Cough → Pink frothy sputum
- dyspnea
- sweating, ↑HR, ↑RR (Respiratory Rate)

O/E → Lungs auscultation - Crepitation

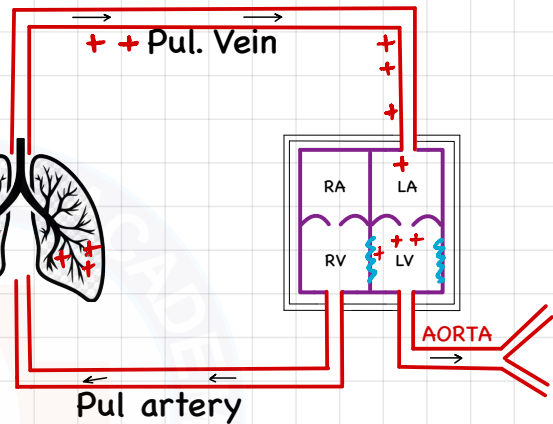
SpO2 - ↓↓

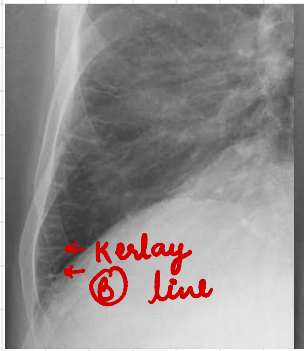
CxR -



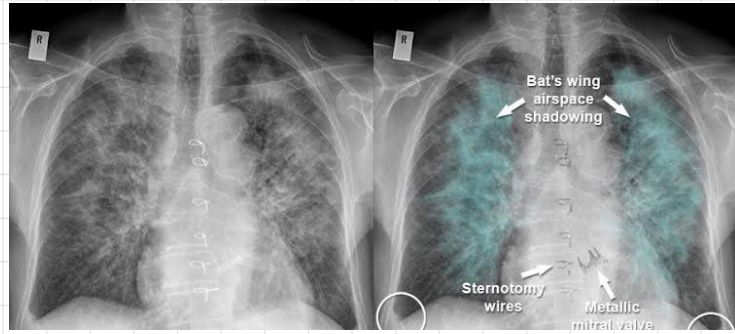
BIL Haziness

Fluid → white
air → black





Kerley
B line



Bat wing appearance

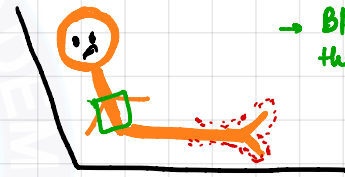
Rx - DOC → eg:- loop diuretics (inj) → Furosemide (FRUSEMIDE)

But if the BP is low (Hypotension) → then 1st → inj. Nor-adrenaline & once BP is stable then ...inj. Furosemide 498P

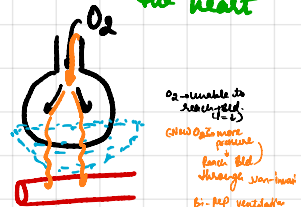
Other ways to ↓ effective blood volume reaching to Heart

→ Nitrate

→ FOWLER'S Position



→ B/c of gravity the bld will remain in dependent part of body (not at heart) & less bld will be there near the heart



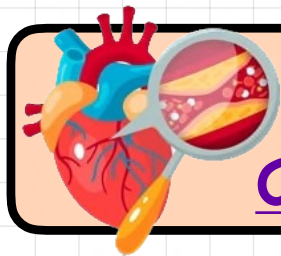
To improve oxygenation [SpO2]



Non-invasive bi-pep Ventilation

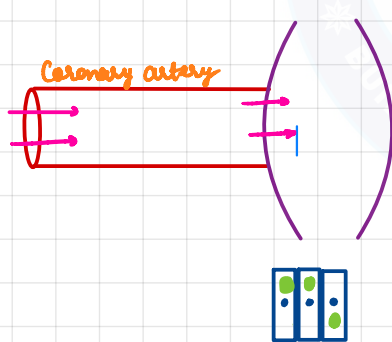
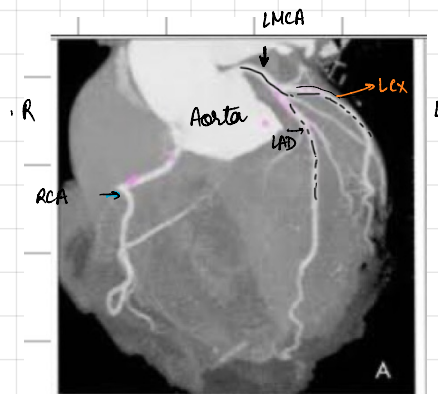
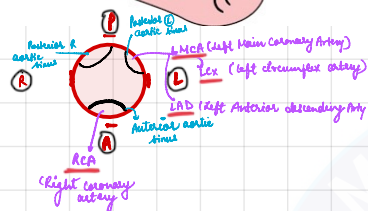
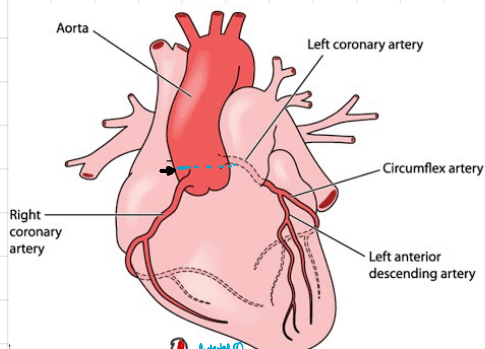


invasive ventilation [ET-tube]
(endotracheal intubation)

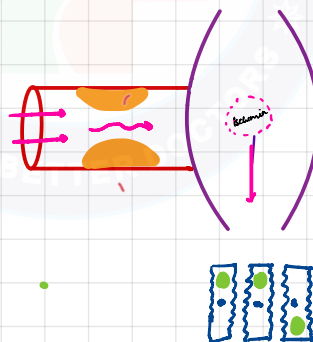


CHAPTER 4

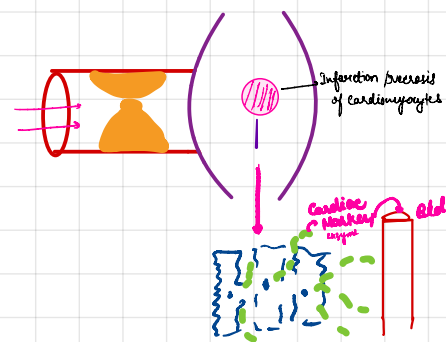
CORONARY ARTERY DISEASE / Ischemic Heart Disease



Cardiomyocyte



Cardiomyocyte



Cardiomyocyte

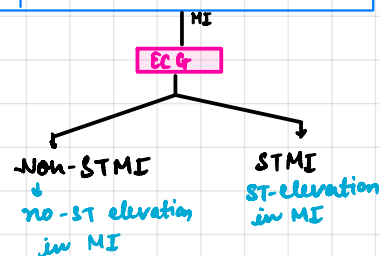
chest pain



	Normal	Ischemia	Infraction
chest pain	No	+	+
Cardiac enzyme in blood (TROPONIN)		Not increased	increased



CPK MB > Myosin > Troponin
4 hr



RISK FACTORS OF CORONARY ARTERY DISEASE

or Risk factor for atherosclerosis

Non-Modifiable

- Age
- M > F
- Family History

Modifiable

- DM
- Smoking
- HTN
- Hyper homocystenemia
- Lipoprotein A
- Chlamydia
- Dyslipidemia [LDH/HDL ratio]
- ① cholesterol
- Familial Hypercholesterolemia or [Type II Lipid disorder]

defect in LDL Receptor

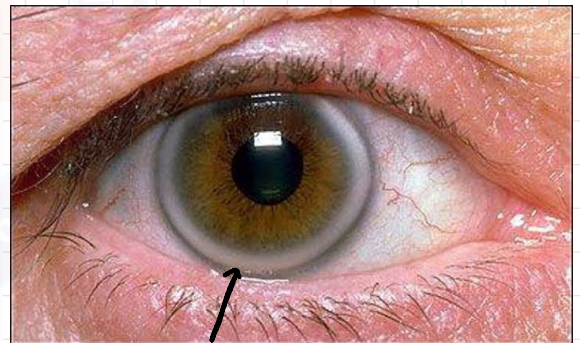
Extra point



Tendon Xanthoma



Xanthelasma



Arcus Senilis

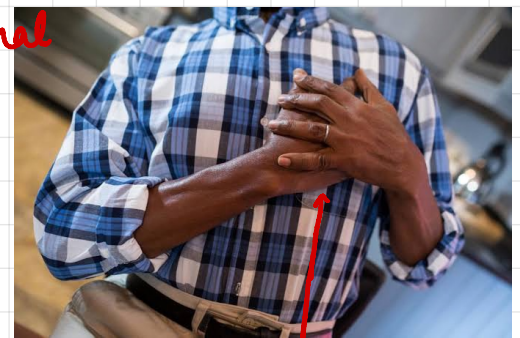
Alcohol → Not a Risk factor for atherosclerosis

[CHEST PAIN]

WHO Rose Questionnaire :-

- 1 - character - ... squeezing type ... Location - Retrosternal
 - radiation to - ... ① shoulder ② VL ③ neck
 - associated with - ... sweating SOB
- 2 - triggered by - ... Exertion
↳ chest pain here start after 1000 ft
- 3 - relieved by - ... Rest

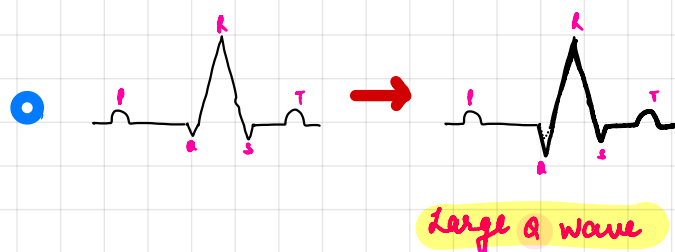
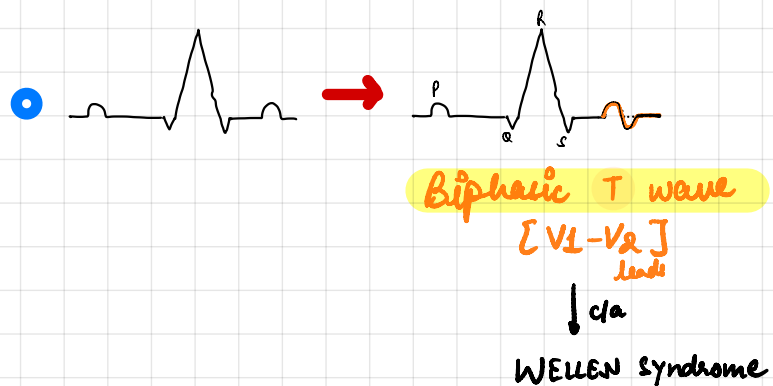
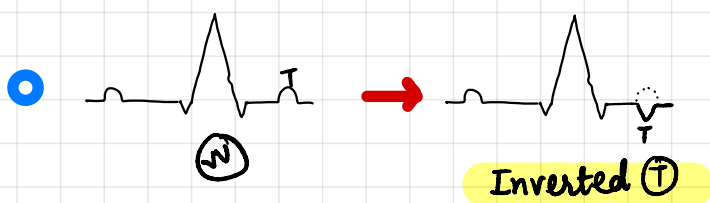
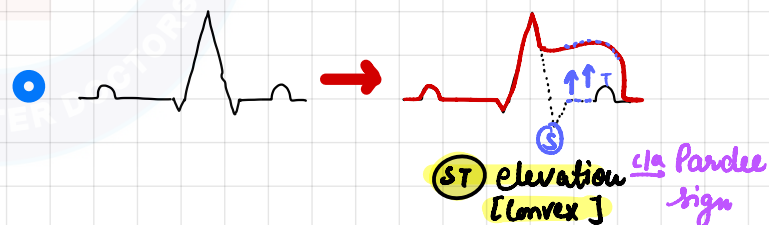
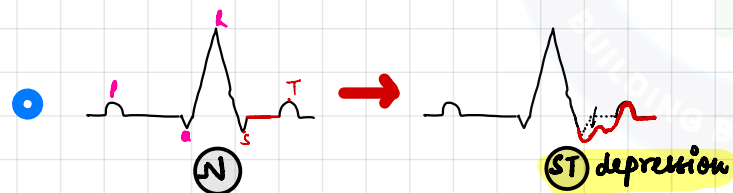
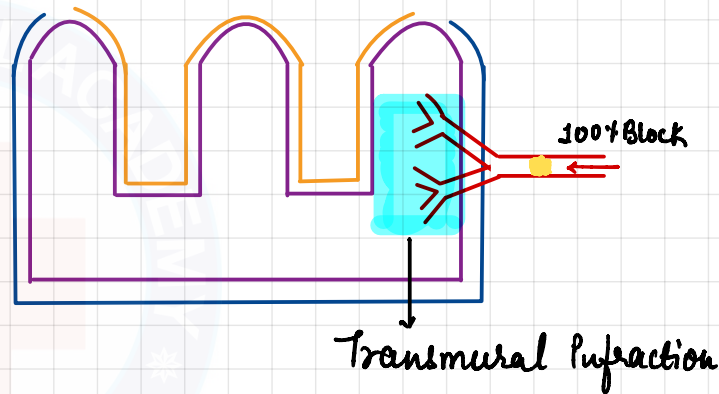
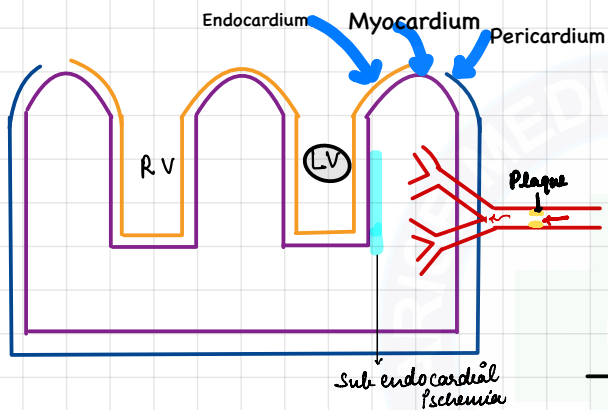
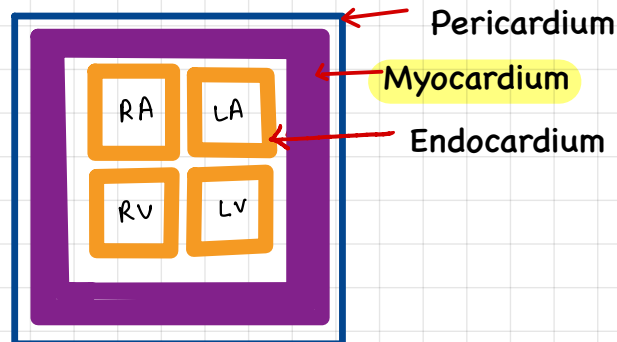
- 3/3 ⊕ → typical anginal pain
- 2/3 ⊕ → atypical anginal pain
- 1/3 ⊕ → Non-anginal pain

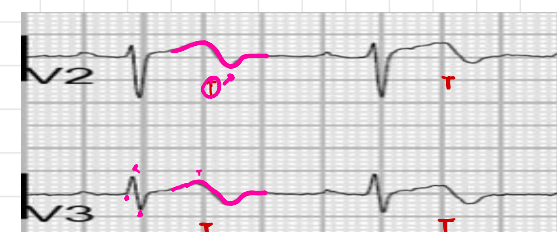
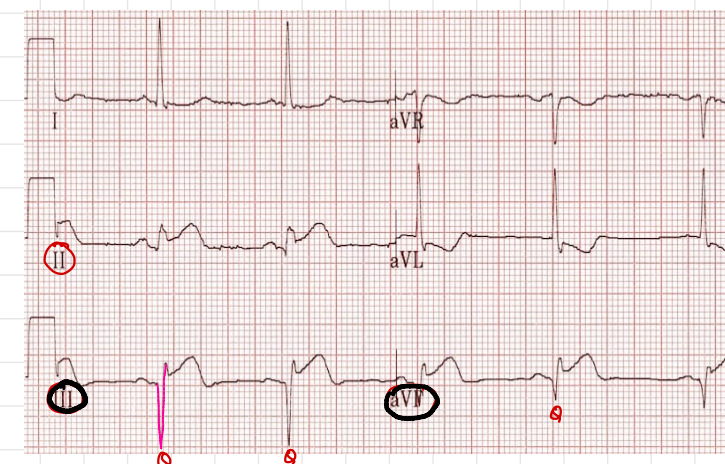
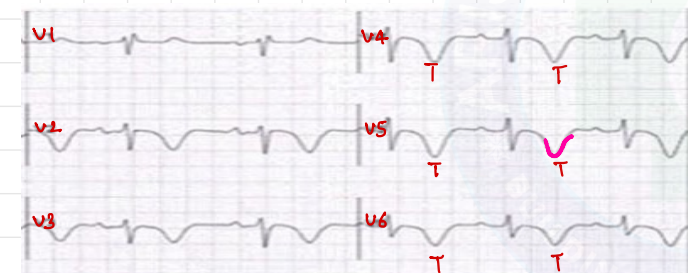
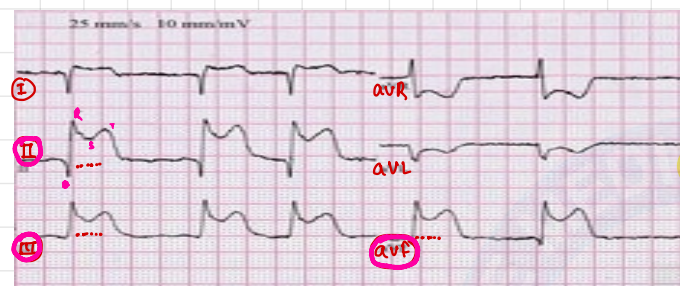
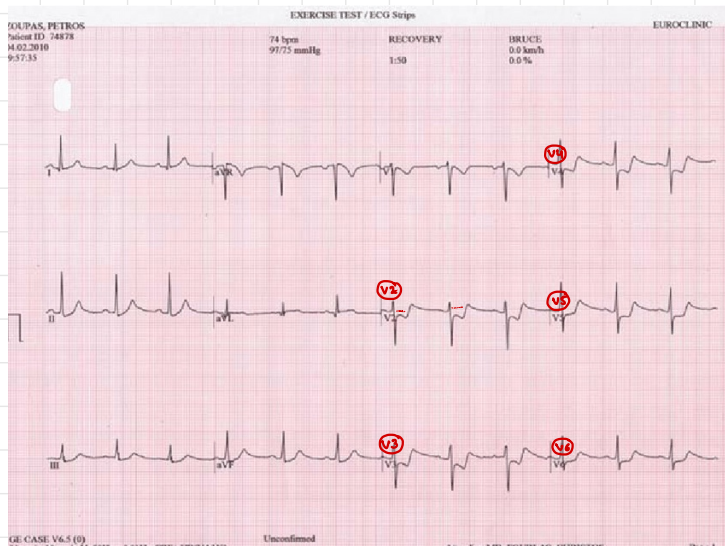


Patient puts fist on chest c/a Levine sign

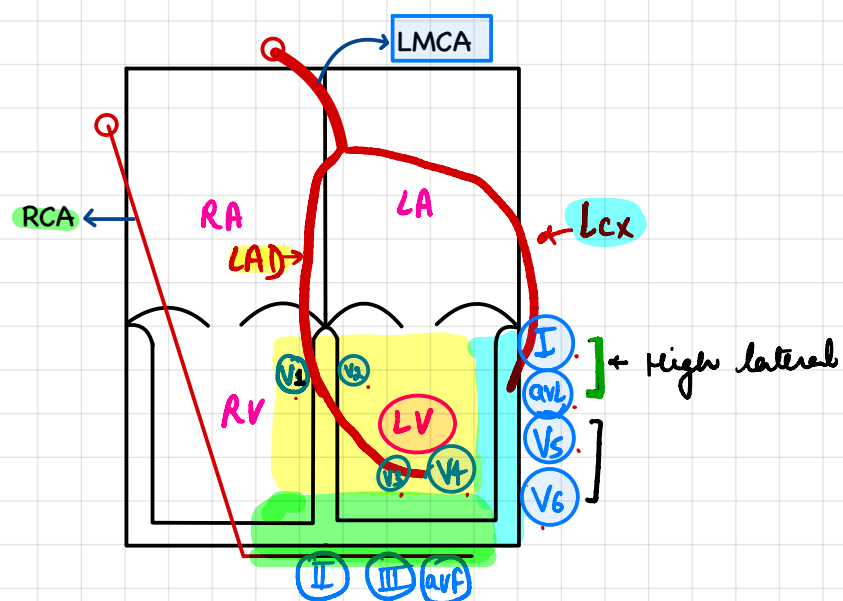
Investigation in CAD

ECG: → <10 min of Arrival





LOCALISATION OF LESION VIA ECG



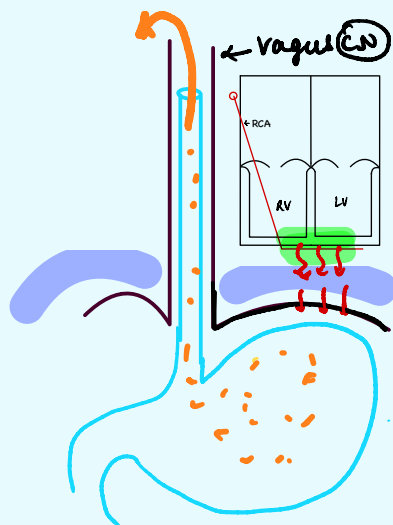
ECG Leads	Localisation of MI	Artery involved
V1-V4	Anterior wall MI	LAD
I/aVL/V5/V6	Lateral wall MI	LCx
I/aVL/V1-V6	Extensive wall MI	LCMA
II/III/aVF	Inferior wall MI	RCA
V1-V2	Septal MI	LAD
I/aVL	High lateral wall MI	LCx

Options - anterior wall MI / lateral wall / high lateral wall / septal / inferior / extensive

Blood vessel - LAD / LCX / LMCA / RCA



EXTRA POINT



Irritation to vagus (X) →

[II/III/aVF]

Inf. Wall MI → only MI in which there is bradycardia happen.

↑ Parasympathetic activity

↓ HR + Nausea Vomiting
(bradycardia)

If V4R lead → showing changes → RV-MI

CARDIAC MARKERS

Earliest to increase → Heart Fatty acid Binding Protein > Myoglobin

Best - Troponin [I > T]

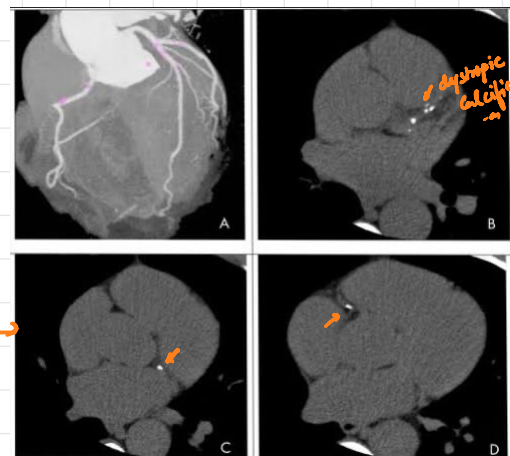
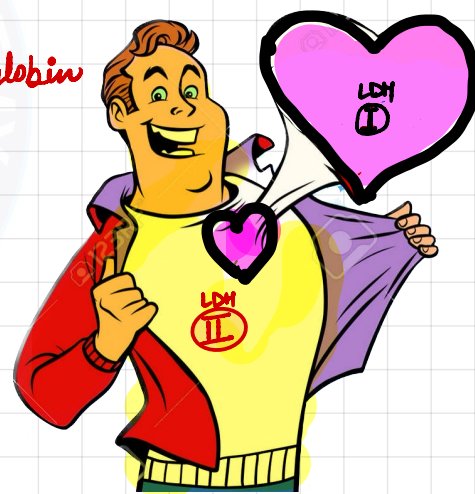
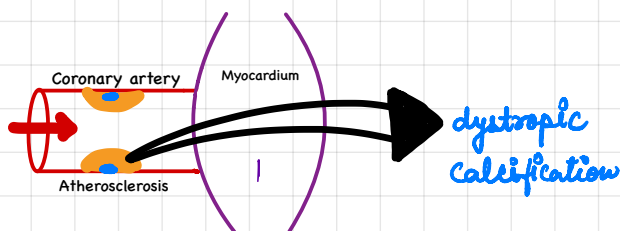
Marker of reinfarction → Quantitative Troponin > CKMB

Future predictor → highly sensitive CRP (hs-CRP)

• SGOT ↑↑ (B/C not in cardiac us also)

• LDH [N] = II > I (flipping of LDH)
[NL] = I < II

* CT-SCAN — NCCT → Non-contrast CT



MC artery to undergo atherosclerosis → Abdominal aorta
MC coronary artery undergo atherosclerosis → LAD (widow's artery)

CONCEPT OF STRESS TEST

[demand of myocardium & HR]



HR —	Less	More
Myocardial ischemia —	No	+
Chest Pain —	No	+



(TMT) Treadmill test

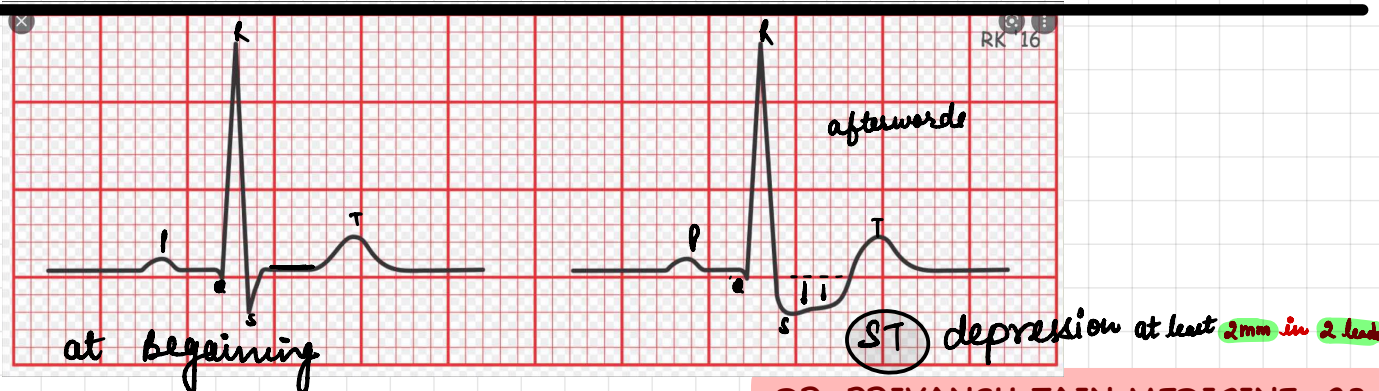
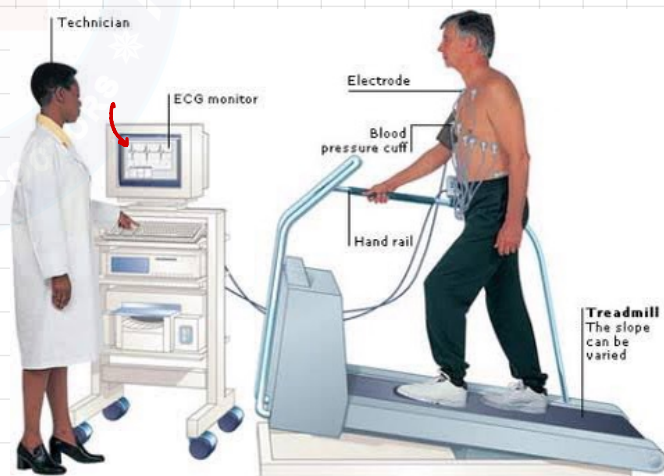
Protocol - Bruce Protocol

Target HR - $220 - \text{Age}$

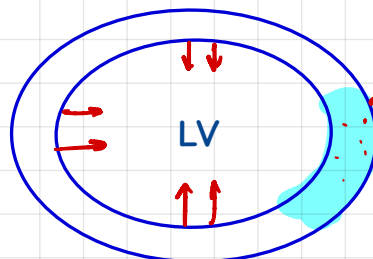
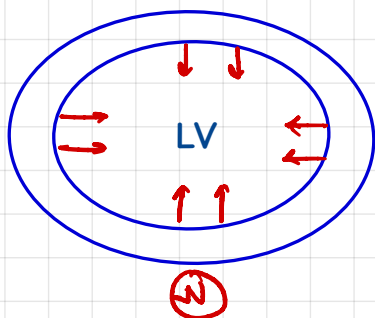
85 % of target HR

Achieved
TMT (-ve)

Not Achieved
TMT (+ve) → Duke Score



2-D ECHO



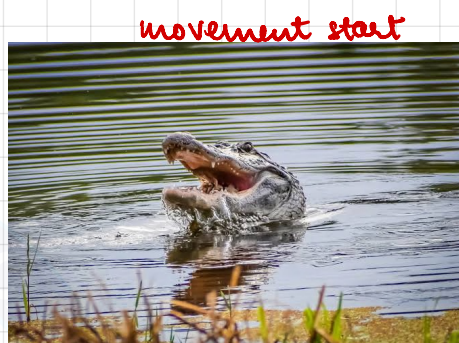
RWMA - Regional wall motion abnormality

PET scan - Best test
Thallium scan
↑
check for viability

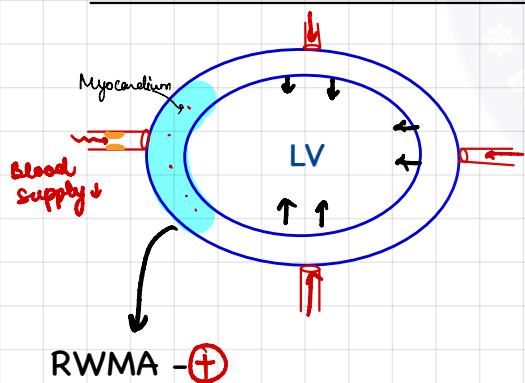
RWMA - area not contracting



Unfavourable condition

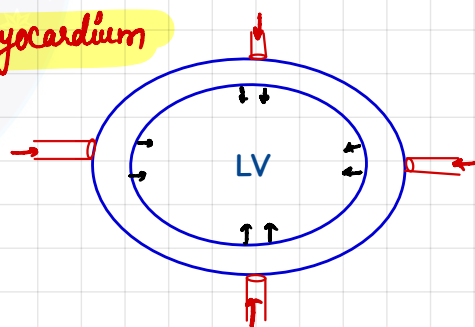


Favourable condition

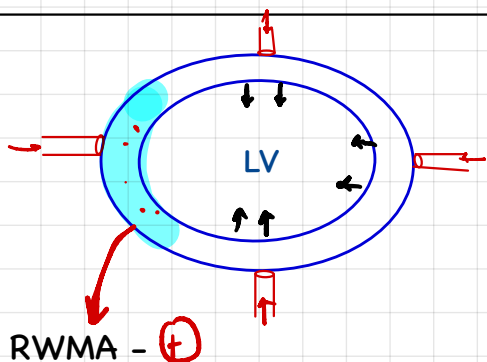


MI → Hibernating myocardium

Revascularization

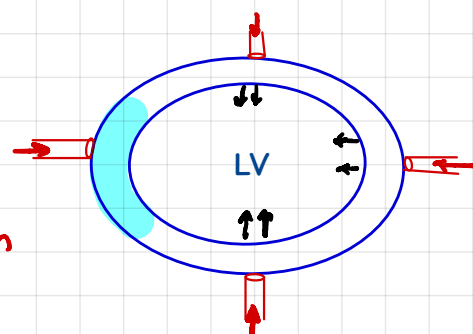


Myocardium viability → PET scan → alive



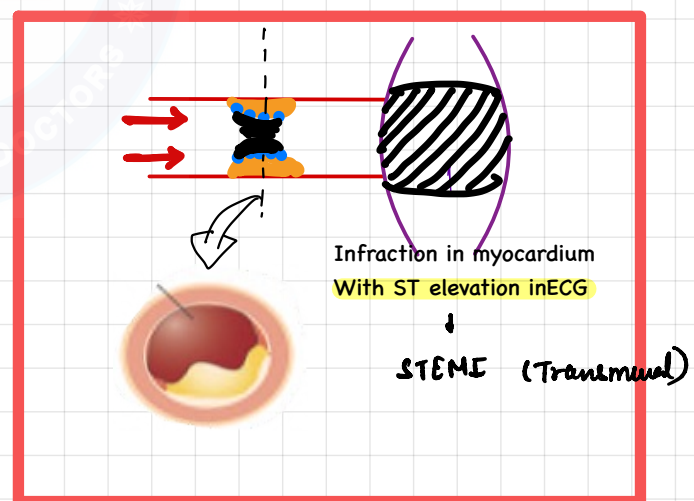
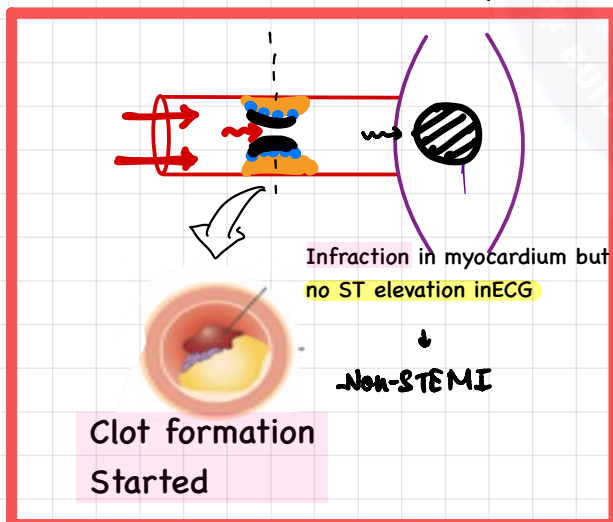
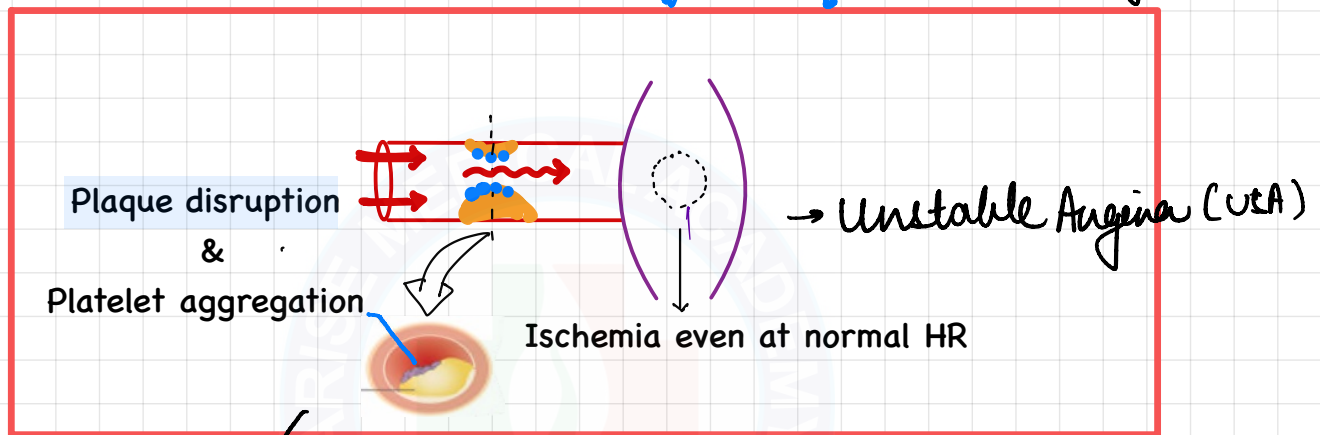
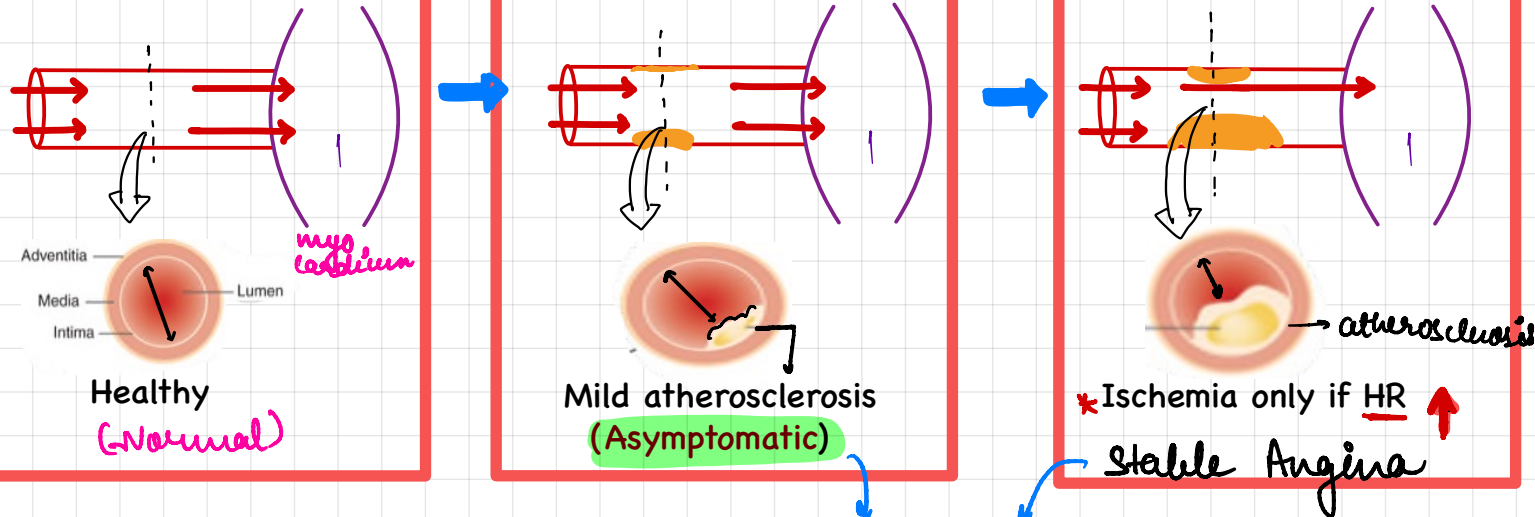
MI → Stunned myocardium

no role of revascularization



Myocardium viability - PET-scan alive

CORONARY ARTERY DISEASE/ISCHEMIC HEART DISEASE



Chest Pain

At exertion
↓
Stable Angina

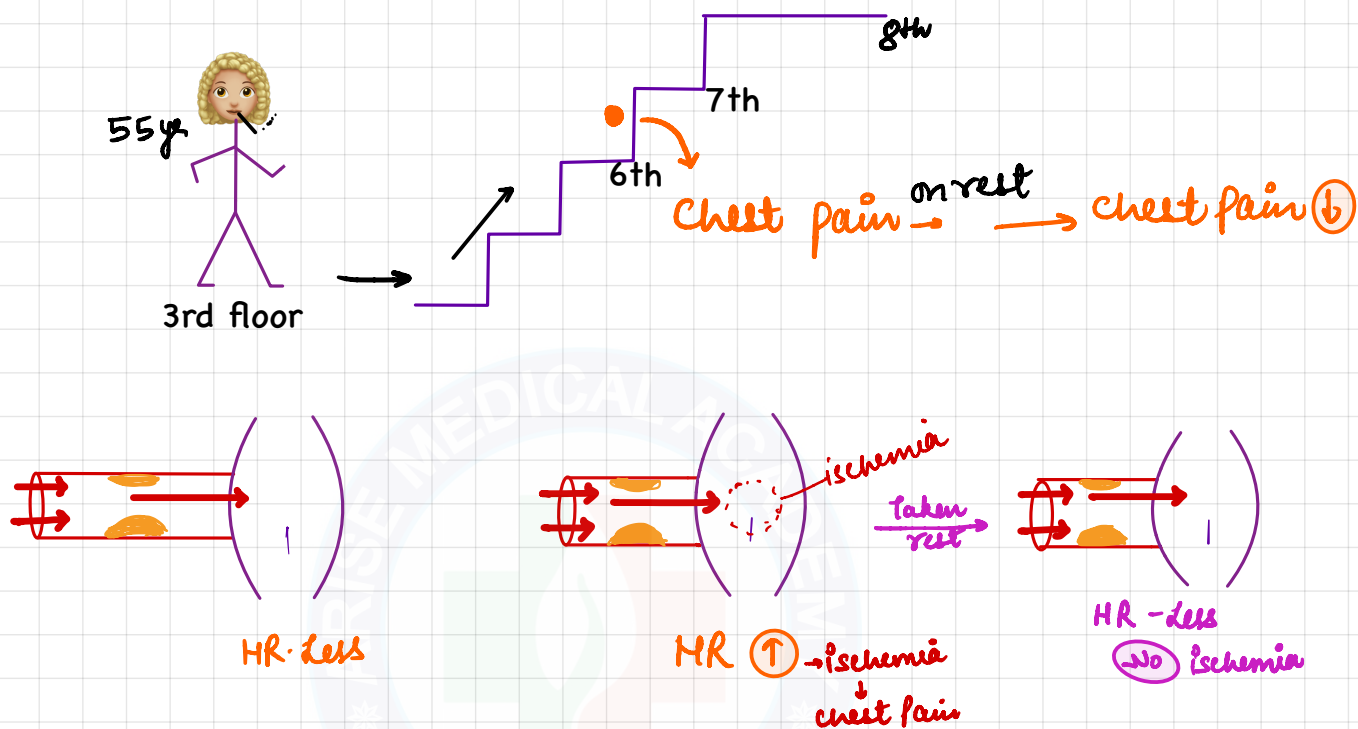
At Rest
↓

ACS
↓
Acute Coronary Syndrome

Unstable Angina	No ST elev.	Normal
Non-STEMI	No ST elev.	↑
STEMI	ST elevation	↑
	ECG	Troponin

STABLE ANGINA [Angina Pectoris]

C/F: → chest pain at exertion [$< 10 \text{ min}$]



INV. → ECG → No ST elevation
CT angiography [better]
TMT → ⊕
↳ Treadmill Test]

Rx → ① β # → ⑥ Cardiac remodelling
↓ HR

② Nitrate

③ Plaque stabilizer → Tab. Atorvastatin

④ Antiplatelet drug → Tab. Aspirin

+/- → To keep HR around 60/min → Tab. Ivabradine [If channel # in SA-node]

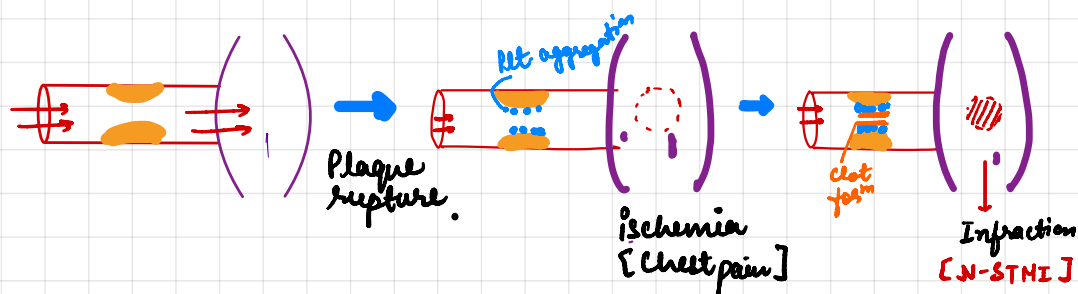
Maximum mortality reduction by - β # > Tab. Aspirin

MC side effect of Nitrate - Headache

Other side effect of nitrate → Methemoglobinemia lead to chocolate cyanosis

USA [Unstable Angina] - NSTEMI COMPLEX

Pathogenesis:



C/F: → chest pain at Rest x 10-30 min

Investigation:

ECG - No ST-elevation → Ruled out → STEMI

Cardiac enzyme - Troponin

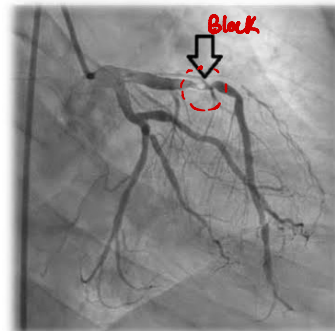
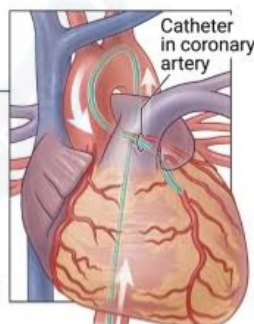
- ⊖ → USA
- ↑ → N-STEMI

Rx: → ① Anticoagulant → Injection of LMWH (Low molecular wt. Heparin)

② dual antiplatelet → Aspirin

P2Y₁₂ #
 • Tab. clopidogrel
 • T. Prasugrel
 • T. Ticagrelor

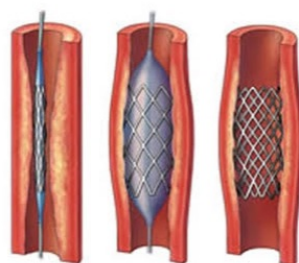
Catheter insertion sites



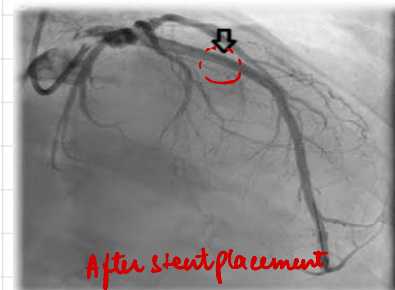
- ③ β #
- ④ Nitrate
- ⑤ Statin

If SpO₂ < 91% → O₂ support

- PCI
- Angiography → inv.
 - Angioplasty → stent placement



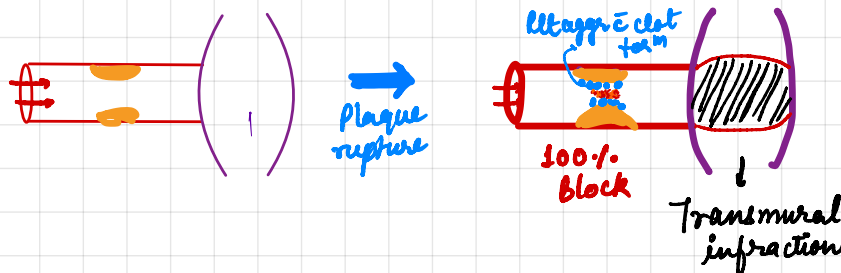
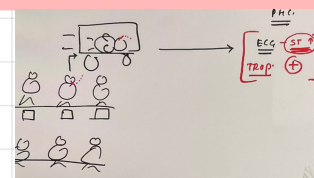
stent placement



So in USA-NSTEMI — plan for PCI if Timi score is ... ≥ 3

* PCI in USA-NSTEMI is a Delayed PCI

ST-ELEVATED MI [STEMI]



Pathogenesis:

C/F: chest pain at Rest $\times > 30 \text{ min}$

Investigation: ECG - **ST** elevation

TROPONIN - $\uparrow$

Rx: $\rightarrow$ **Thrombolysis** (just give aspirin) or **1° PCI** [better]

$\rightarrow$ **★ if PCI facility is more than ... 2 ... Hours away**

Then go for **Thrombolysis**

Door to needle time = $< 30 \text{ min}$

Agents $\rightarrow$ injection of streptokinase
1.5 million IU $\downarrow$

chest pain & ST elevation in next 90 min

Yes
Plan for **Successful Thrombolysis**
CHECK PCI

No
Plan for **Unsuccessful Thrombolysis**
RESCUE PCI

If PCI facility is less than 2 Hours away

then go for **1° PCI**

Door to needle time $\leq 90 \text{ min}$

Other treatment - same as ... VSA ... NSTEMI

- inj. LMWH
- dual antiplatelet
- β #
- Nitrate
- statin
- if $\text{SpO}_2 < 91\%$ → O₂ support

+/- Morphine → if chest pain not subsided (काफ़ी हो जाता है)



EXTRA POINTS

After PCI → dual antiplatelet is given for → 12 month.

* Inf. Wall MI → RVMI → HR ↓ } → β # are avoided
 BP ↓ } instead inj of atropine is given

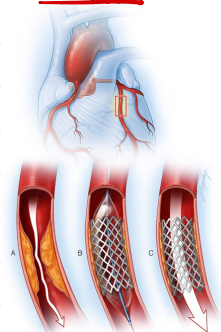
So in Inf. wall MI if patient in shock before doing thrombolysis give → IV fluid

ANGIOGRAPHY

If single vessel disease [SVD] or double vessel disease [DVD]

Plan for PTCA [stent placement]

drug coated stent → sirolimus } Anti Cancerous
paclitaxel } drug

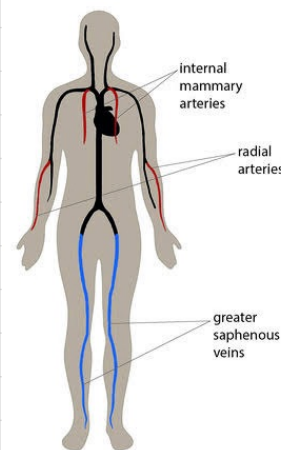
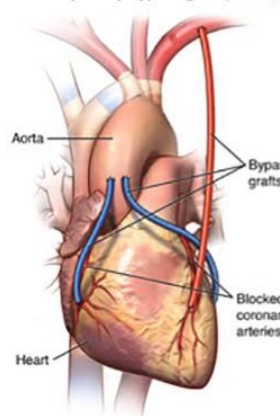


PTCA-Percutaneous transluminal coronary angioplasty*

If TVD (triple vessel disease)

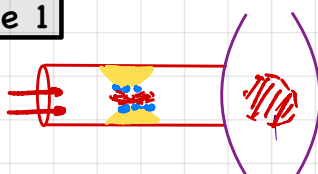
Rx → Plan for CABG [Coronary Artery Bypass Grafting]
 for graft →
 ↳ Lft. Internal Mammary artery (best)
 ↳ GSV (Great saphenous vein)
 ↳ Radial artery

Coronary artery bypass graft (CABG)



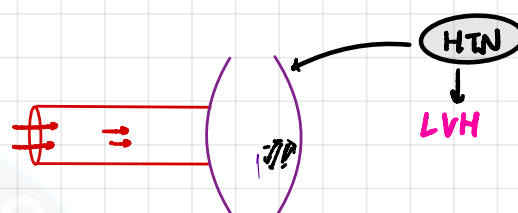
TYPES OF MI

Type 1



↓ Bld supply → dlt plaque rupture

Type 2



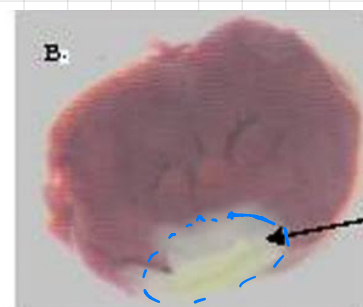
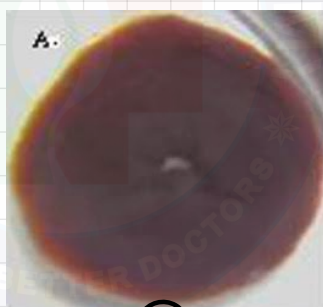
if there ↑ in demand → MI (Type II)

Type 3

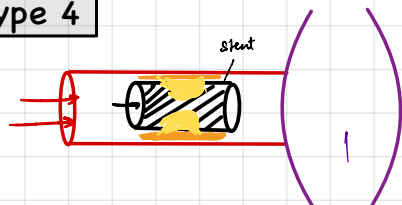
Sudden cardiac death

Hx is suggestive of MI

To confirm → Autopsy

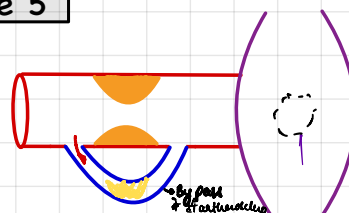


Type 4



angioplasty → stent failure → MI (Type IV)

Type 5

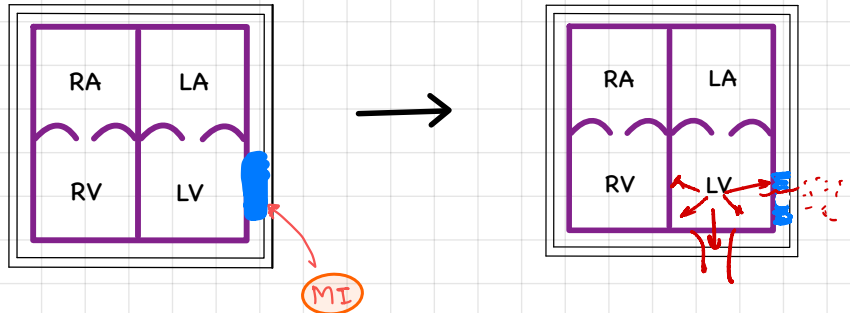


CABG → MI (Type V)

COMPLICATIONS OF MI

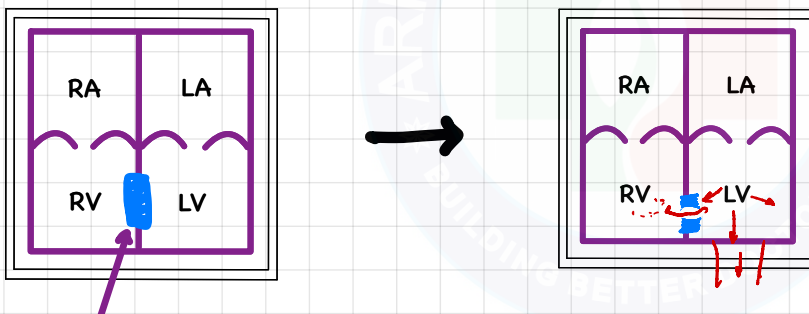
a) with 1st few hours $\rightarrow$ of MI $\rightarrow$ sudden ^{due to} cardiac death Mostly due to Ventricular Arrhythmia

b) after 4 - 7 days of MI $\rightarrow$ ① BP $\rightarrow$ Δ ss $\rightarrow$ Free wall Rupture



c) after 4 - 7 days of MI $\rightarrow$ New murmur $\rightarrow$ systolic $\rightarrow$ at ② sternal border

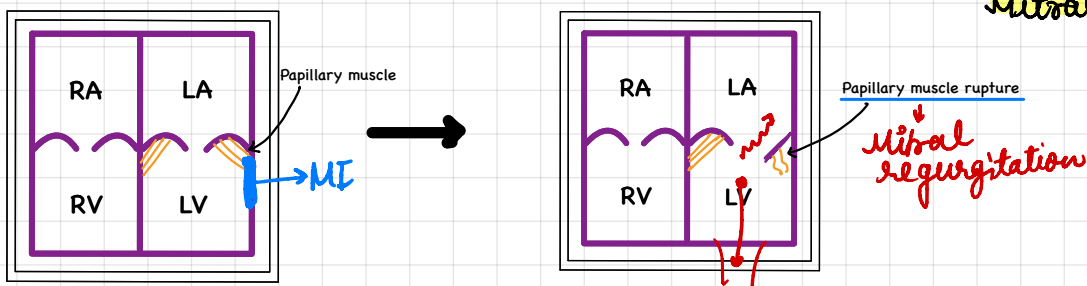
Δ ss $\rightarrow$ VSD
(ventricular septal defect)



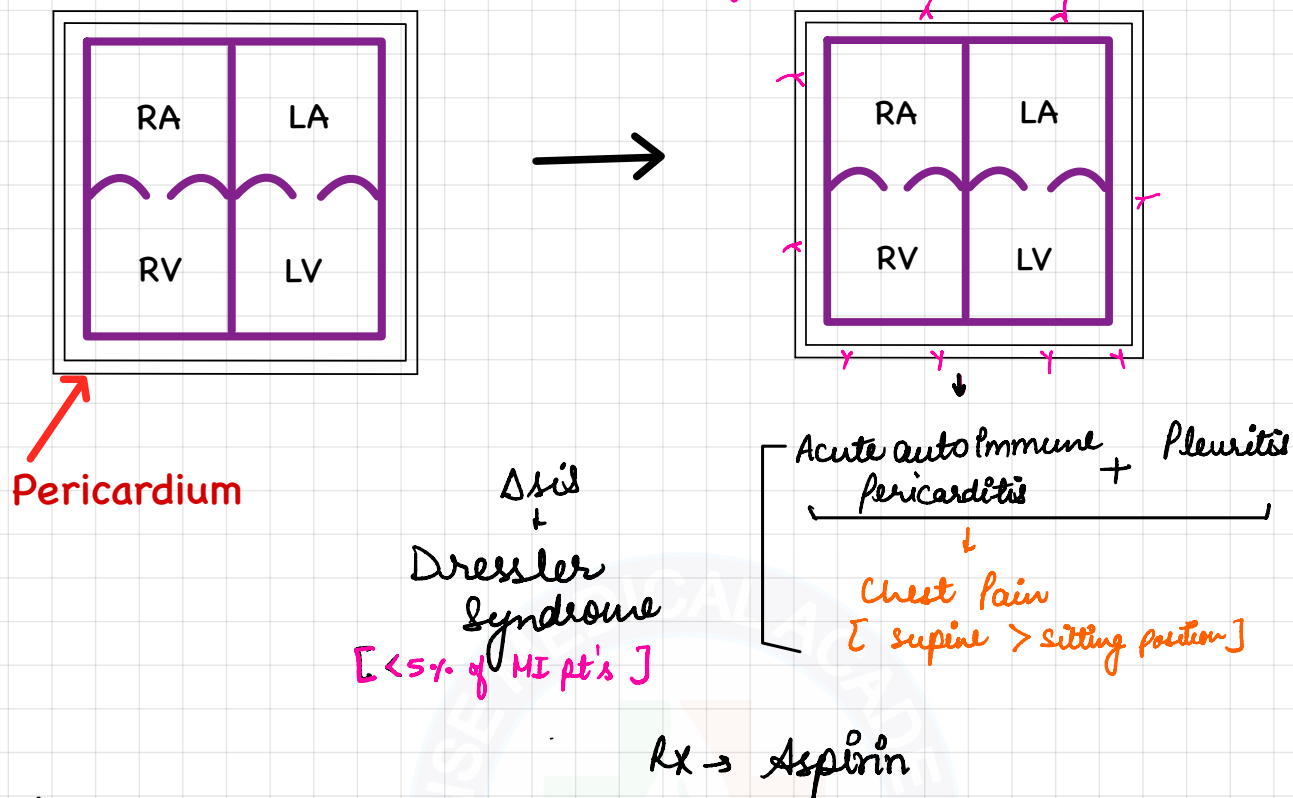
Septal MI

d) after 4 - 7 days of MI $\rightarrow$ New murmur at mitral area during systole

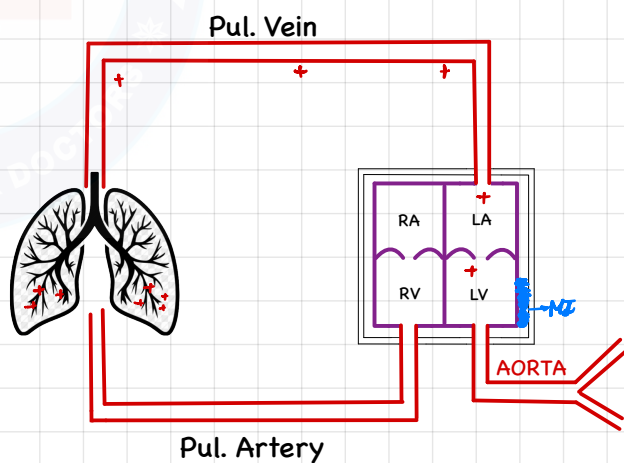
$\downarrow$
Mitral Regurgitation



d) MI → after 4-6 week. → *wrong antibody form against* Pericardium & pleura



e) MI → LVF → Pulmonary edema or HF



KILIP CLASSIFICATION

MI

No sign of HF

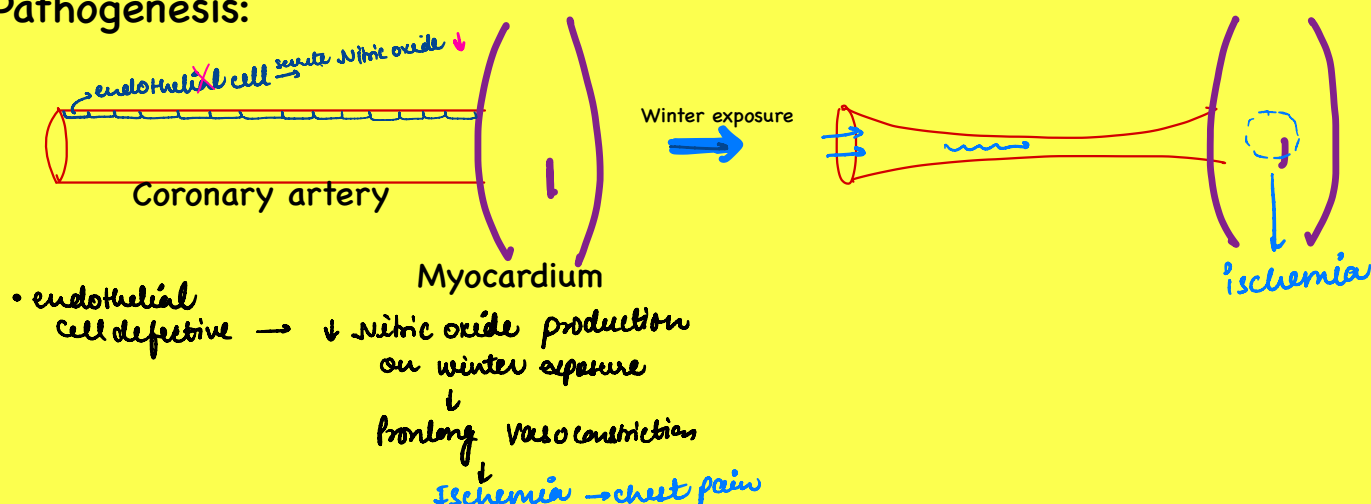
B/L crep. in 50% lungs

Frank Pul. edema

Pul. edema + Shock

PRINZMETAL ANGINA / Vasospastic angina / Variant angina

Pathogenesis:



C/F: Recurrent chest pain on winter exposure

Associated with- Raynaud's phenomenon / cocaine intake

ECG → ST elevation + Troponin (N)

Rx → nitroglycerin / CCB

Match the following

- 1) chest pain at rest + no ST elevation + troponin ↑ → NSTEMI
- 2) chest pain at exertion only + no ST elevation + troponin N → SA
- 3) chest pain at rest + ST elevation + troponin ↑ → STEMI
- 4) chest pain at rest + no ST elevation + troponin (N) → USA
- 5) recurrent chest pain on winter exposure + ST elevation P.A
- 6) chest pain after 4 weeks of MI → Dressler syn.
- 7) shock on 5th day POST MI → free wall rupture

- A) stable angina (SA)
- B) unstable angina (USA)
- C) N-STE-MI
- D) STEMI
- E) free wall rupture
- F) Prinzmetal angina (PA)
- G) Dressler syn

1 2 3 4 5 6 7



SUMMARY OF CORONARY ARTERY DISEASE

ECG-Change — Transmural infarction — ST Elevation / ST depression

— Subendocardial ischemia — ST Elevation / ST depression

Localisation of MI — II/III/aVF — _____ wall. (ant / inferior / lateral)

— I/aVL/V5/V6 — _____ wall (ant / inferior / lateral)

— V1-V4 — _____ wall. (ant / inferior / lateral)

MI — associated with bradycardia — _____ wall MI (ant / inferior / lateral)

Marker of reinfarction — quantitative Troponin > CPKMB

Best viability test — _____ (PET scan / thallium scan)

TMT — is + if — ST depression $\geq$ 2 mm for 80 ms

Stable angina — max. Mobility by — nitrate / B-blocker / aspirin ' statin

Door to needle time — thrombolysis — < ____ min (< 30 / < 60 / < 90 / < 120)

— 1 PCI — < ____ min (< 30 / < 60 / < 90 / < 120)

PCI — after successful thrombolysis — ____ PCI (check / rescue / delayed)

— after unsuccessful thrombolysis — ____ PCI (check / rescue / delayed)

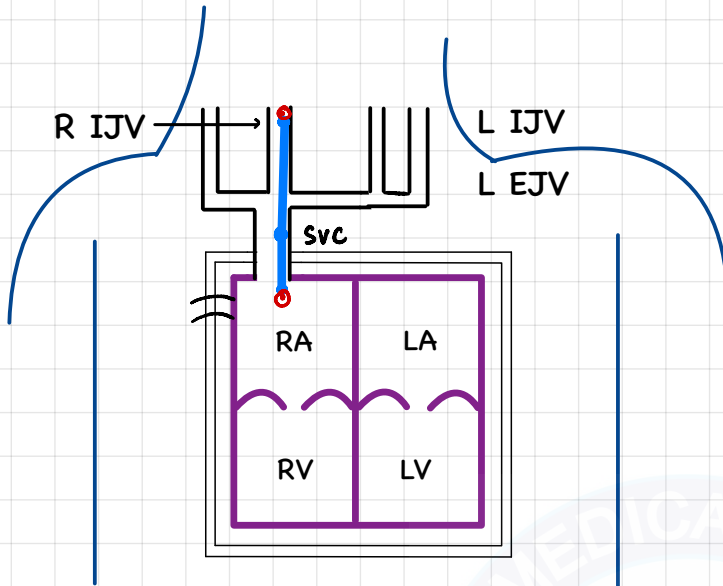
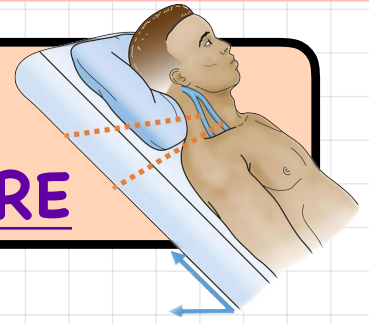
— after USA-NSTEMI — ____ PCI (check / rescue / delayed)

Variant angina — Rx — 1) nitrates 2) CCB.

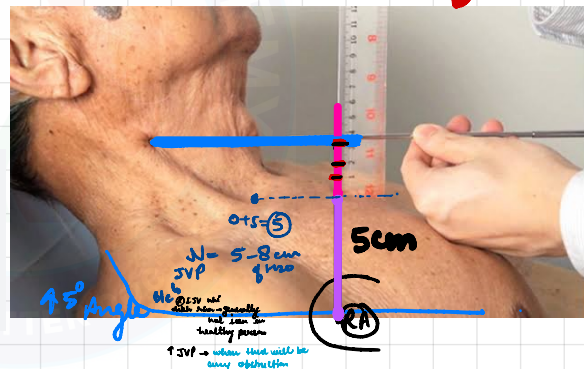
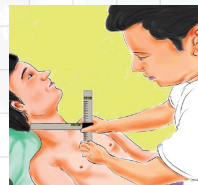
or
prinzmetal
angina

CHAPTER 5

JUGULAR VENOUS PRESSURE



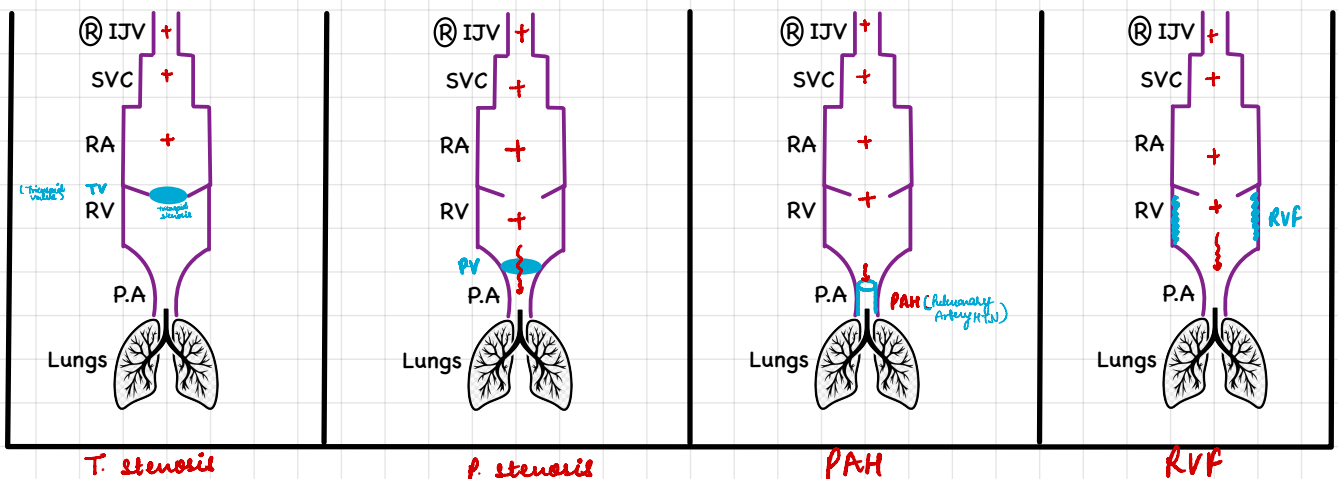
- Why → b/c it is a mirror of @ Heart
- Where → @ IJV to measure
- Normal JVP $\approx$ @ RA pressure
 - = 2-5 mm of Hg
 - = 5-8cm of H₂O
- How → 2 scale technique



Carotid artery pulsation are outward & JV inward.

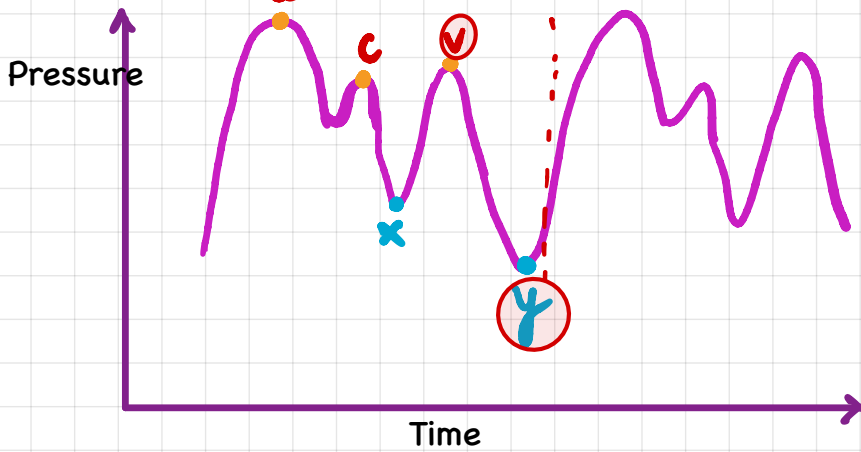
[JVP = pressure of RA]

CAUSES OF RAISED JVP → @ Heart outflow obstruction → TS / PS / PAH / RVF

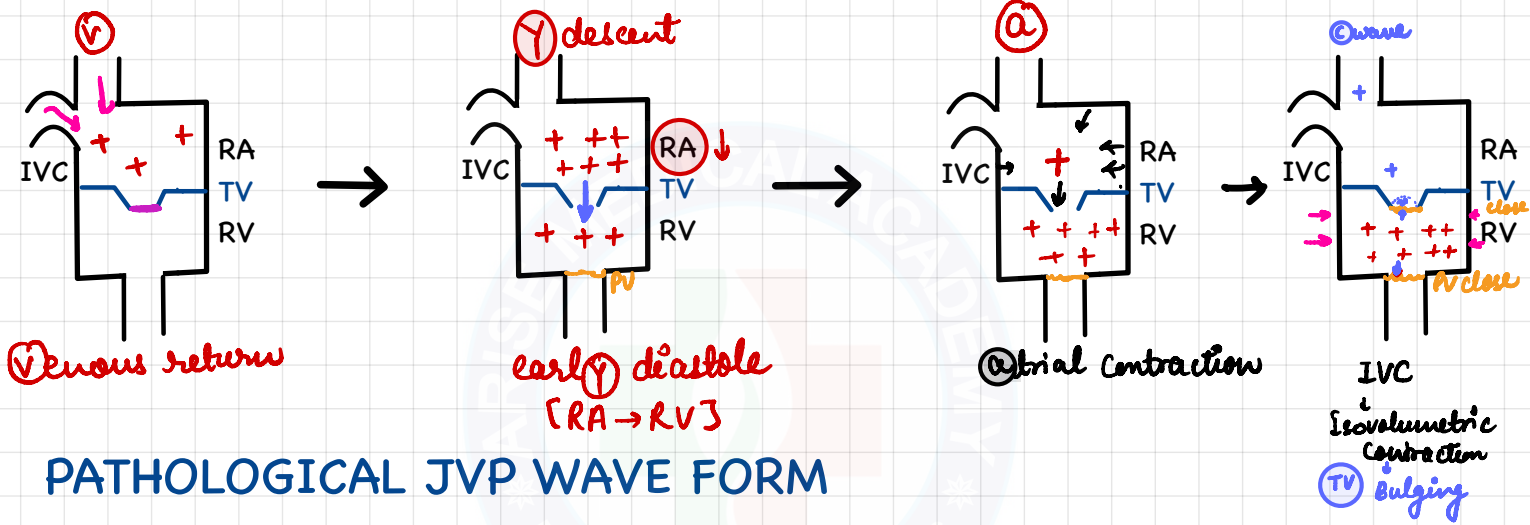




JVP WAVE-FORM



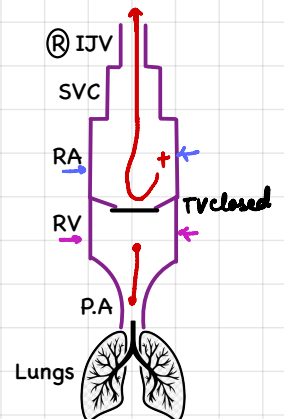
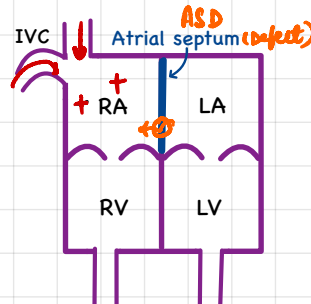
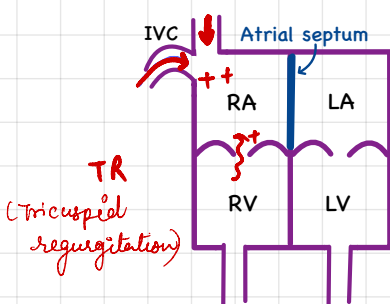
- **a** wave → **RA** contraction
- **c** (TV) Bulging during IVC
- **v** wave → Venous return to RA
- **x** descent → atrial relaxation + ventricular contraction
- **y** descent → early diastole [RA → RV]



PATHOLOGICAL JVP WAVE FORM

- a-wave** [blc of atrial contraction]
 - Absent → Atrial Fibrillation
 - Large → **R** Heart → TS / PS / PAH obstruction
 - Cannon → **C** HB / Junctional Rhythm
 - complete heart block
 - Atria & ventricle contract simultaneously

v wave → large **v** wave → TR / ASD
[Venous return]

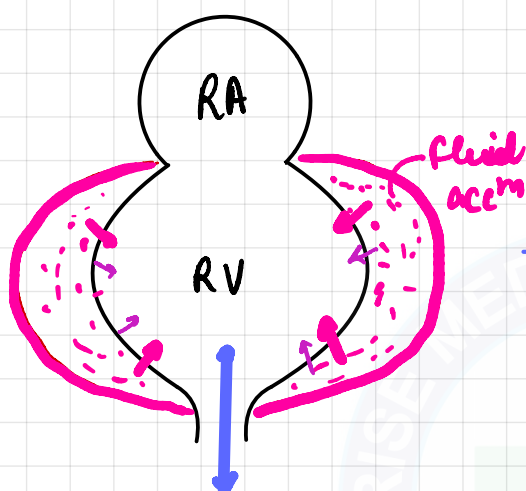


X-Descent $\rightarrow$ atrial relaxation + ventricular contraction [systole]

In view of

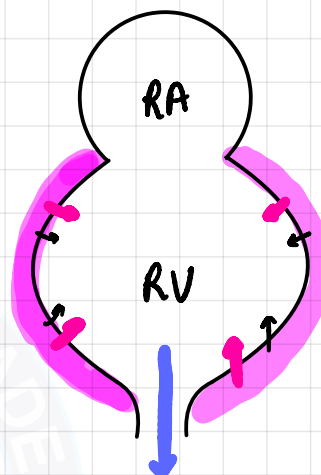
Cardiac Tamponade

Prominent X descent



Constrictive Pericarditis

Prominent X descent

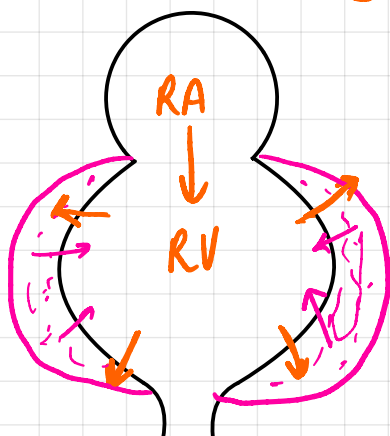


Y-Descent $\rightarrow$ early diastole [RA $\rightarrow$ RV]

In view of

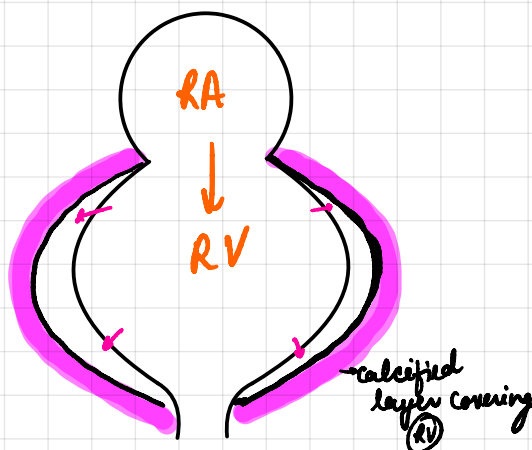
Cardiac Tamponade

diminished Y descent



Constrictive Pericarditis

Prominent Y descent



	Cardiac Tamponade	Constrictive Pericarditis
X	Prominent	Prominent
Y	diminished	Prominent

Kussmaul Breathing
↓
seen in metabolic
acidosis.

* Prominent Y descent → is called friedrich's sign

KUSSMAUL SIGN

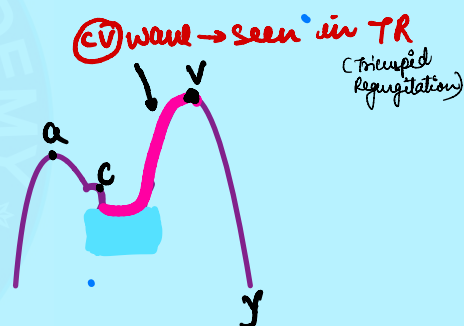
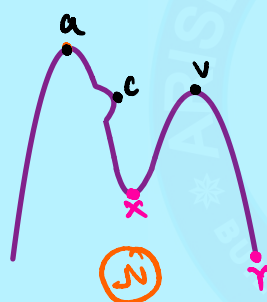
→ (N) during inspiration → JVP falls
But if during inspiration → JVP ↑ → c/a Kussmaul sign

→ seen in → Constrictive Pericarditis / RVMI

→ (Not) seen in → Cardiac Tamponade (CT)



EXTRA POINT



SUMMARY OF JVP

Measured in → R IJV. (R IJV / R EJV / L IJV / LEJV)

Method → 2 Scale technique.

C wave → d/t TV Bulging during IVC phase. (IVE → Isometric contraction)

Large (a) wave → TS / PS / PAH.

Cannon (a) wave → CHB / TR.

Large (v) wave → TR / ASD.

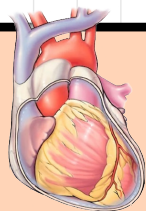
Prominent (Y) → Con. Pericarditis

Cardiac tamponade → X Prominent / Y diminished [P Prominent / diminished]

Kussmaul sign (NOT) seen in CT. [CT / CP]

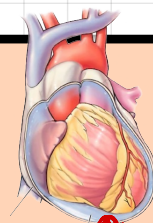
cv wave → TR. [ASD / TS / TR / PR]

[CHB / TR / ASD
Junctional Rhythm
PS / TS / PAH
[CT / CP]



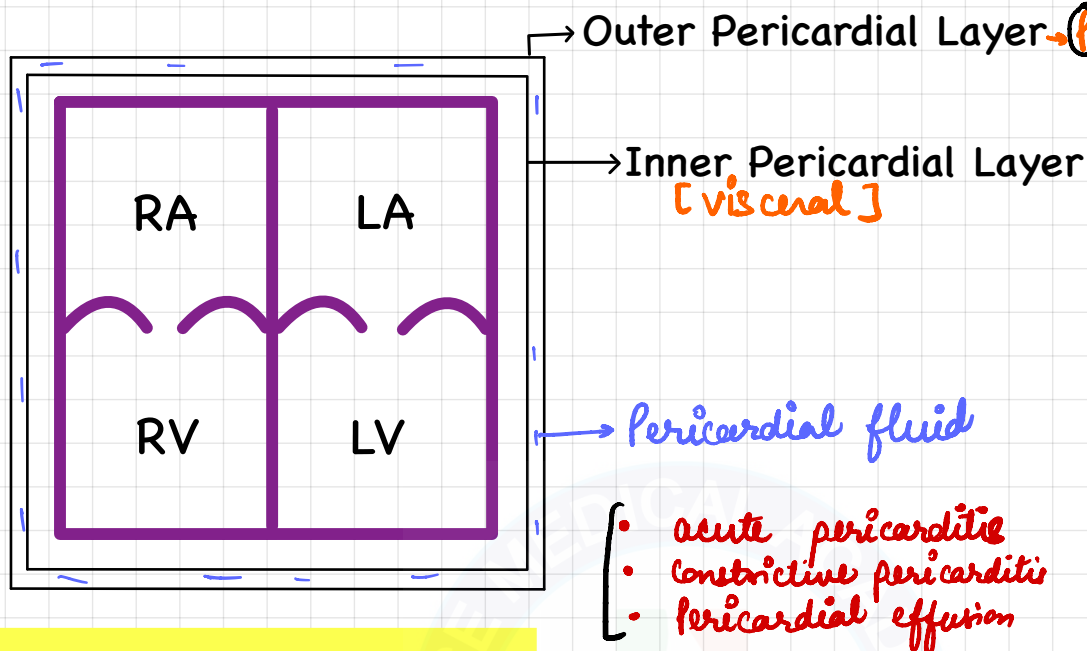
CHAPTER 6

PERICARDIAL DISORDERS



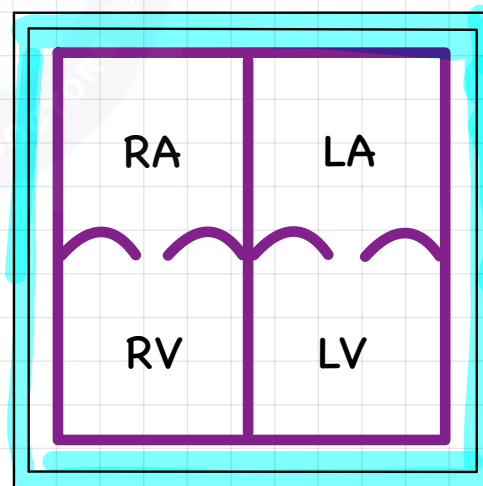
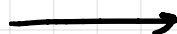
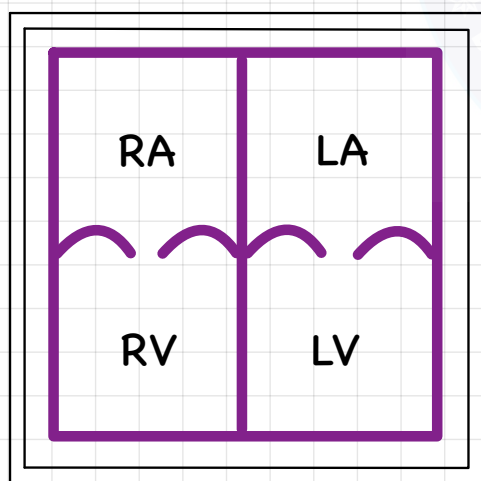
Obstructive

Pain sensitive



ACUTE PERICARDITIS

Etiopathogenesis:

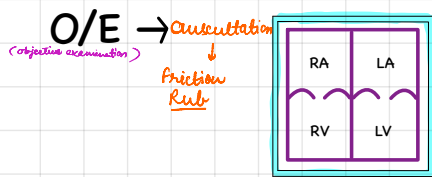


↓ d/t acute inflammation
↓ friction b/w pericardial layer
↓ chest pain
(b/c Pericardium is pain sens.)
→ supine > sitting

Etiology — MCC → Idiopathic

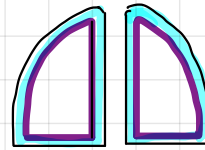
- Other → viral infection (coxsacki)
- Autoimmune → post MI [Dressler syndrome]
- RF (Rheumatic fever)
- drug → Hydralazine, Isoniazid
- Radiation → Uremia

C/F → Chest pain x sharp x [supine > sitting]

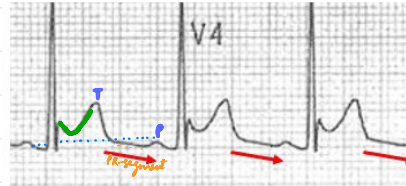
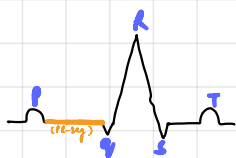


Also know - friction rub in ... Pleuritis ...

But On holding the breath - Absence of friction rub
↓
Pleuritis

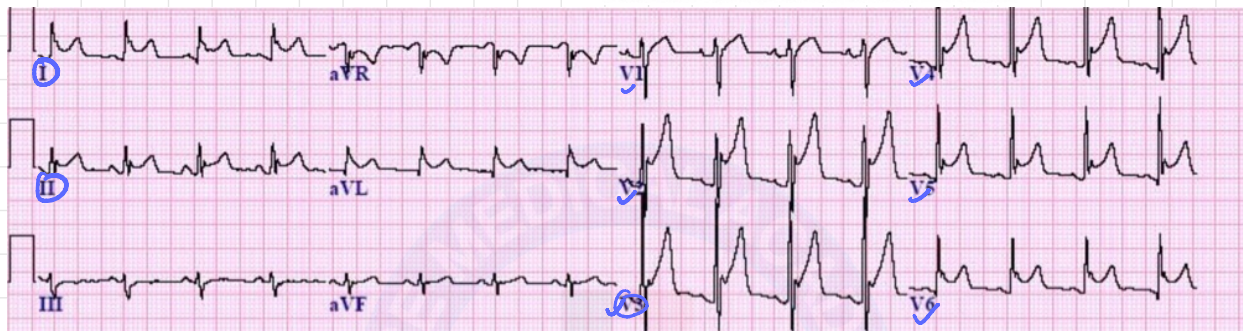


ECG →



PR depression

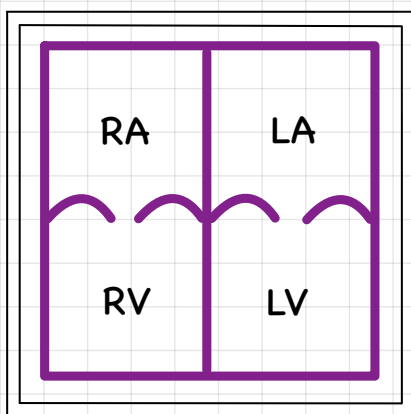
- ST elevation (concave)
- downslowing of TP - spodick sign



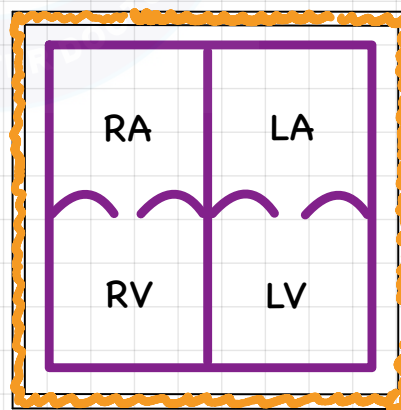
Rx → Steroid

But if acute pericarditis d/t dresler syn → Aspirin (high dose)

CONSTRUCTIVE PERICARDITIS



TB
↓
chronic inflammation in pericardium



Calcification & fibrosis of pericardium

↓
Thick pericardium

↓
[diastolic dysfunction]

↓
[RV > LV]

↓
RVF

Etiology → MICIC → TB^Q
 other causes → Cardiac sx / CKD / Radiation

C/F → same as RVF → distended neck vein
 ↳ SOB
 ↳ abd. discomfort
 ↳ CHEST pain is (not) prominent

O/E → JVP ...↑→ X descent prominent & Y descent prominent → c/a → Friedrich sign
 → kussmaul sign - ...+ve→ low inspiration → ↑JVPJ

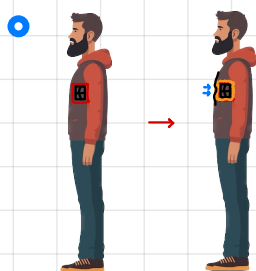
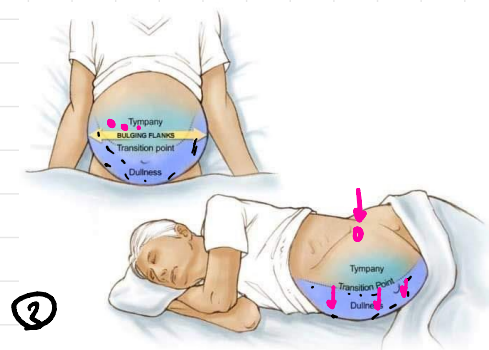
Normally - On inspiration SBP falls But not more than 10 mm of Hg.

If - On inspiration SBP falls But more than 10 mm of Hg c/a - Pulsus Paradoxus^Q

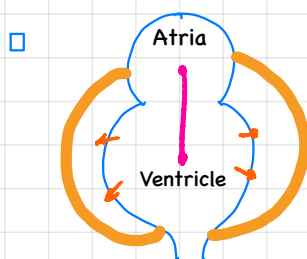
Pulsus paradoxus is also seen in C.P. (Constrictive Pericarditis) & CT (Cardiac Tamponade) & Respiratory Pathology

For ascites ① Puddle test
 ② Fluid Thrill Test
 ③ Shifting dullness Test

• Hepatomegaly

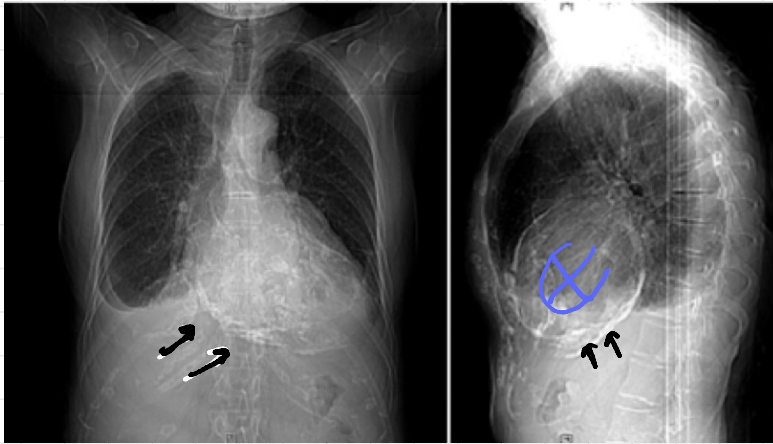


inward pulling of chest wall
 c/a Broad Bent sign

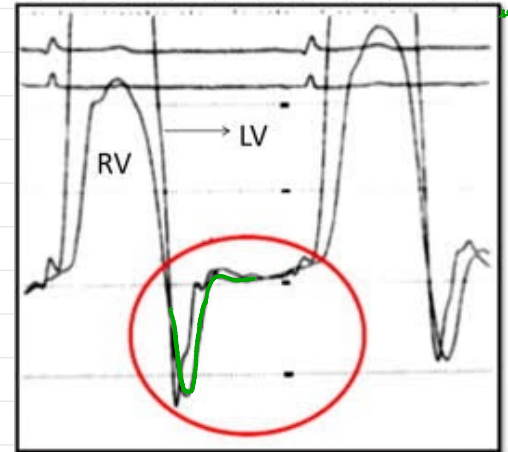


On auscultation - during diastole → pericardial knock

Cxr → egg in cup appearance

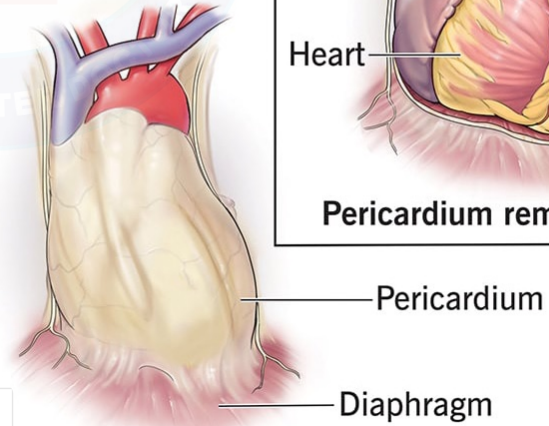
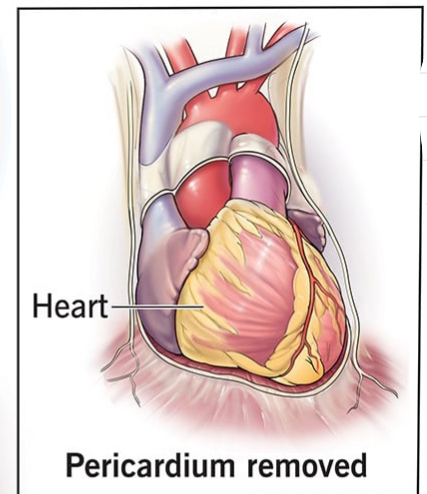
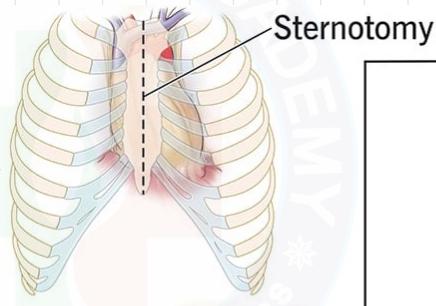


Echo → Thick pericardium
 (LV) pressure → "√" sign (square root sign)
 "not specific" (can also be seen in Restrictive Cardiomyopathy & ALHBP)



Also seen in -

Rx → Pericardiectomy
 if etiology is (TB)
 ↓
"ATT + steroid"



PERICARDIAL EFFUSION

Excess accumulation
of pericardial fluid.

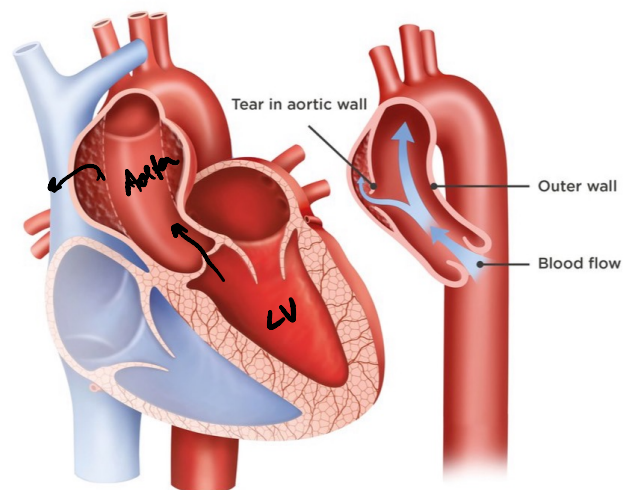
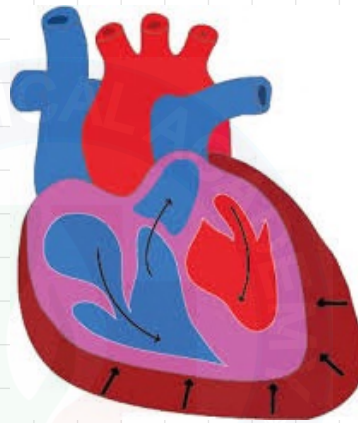
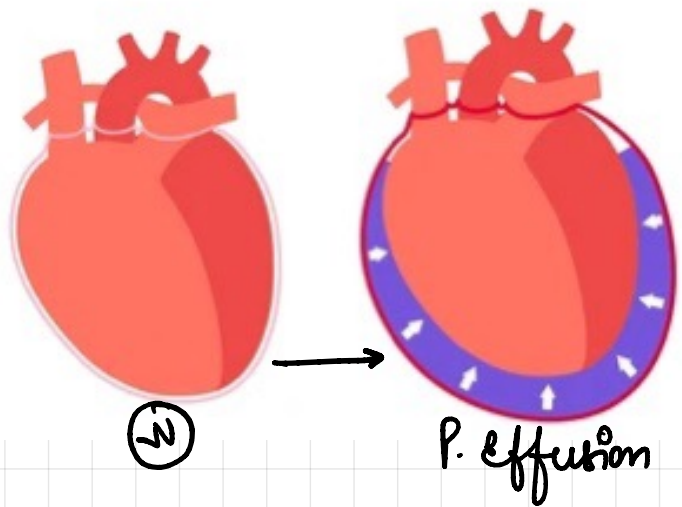
if rapid onset of
P. effusion

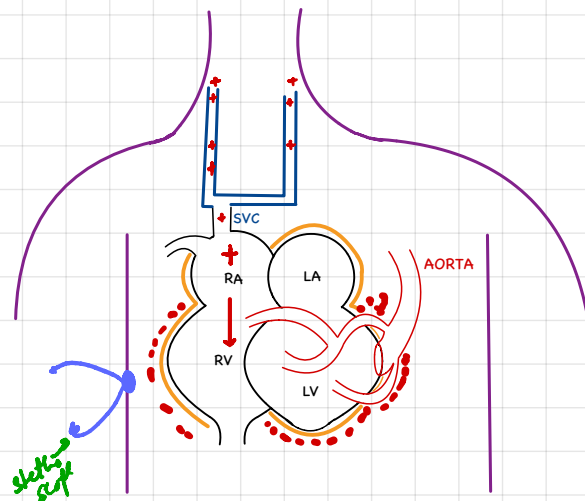
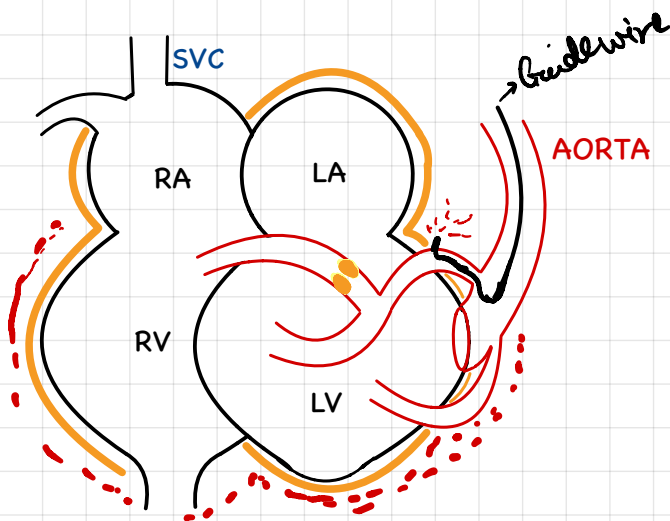
[Hemorrhagic fluid]

C/a

CARDIAC TEMPONADE

Etiology → idiopathic
→ Trauma
→ Malignancy
→ Aortic dissection
→ Iatrogenic
→ Post MI → free wall rupture





C/F
Beck's Triad
↓ BP
distended neck vein
Soft Heart sound

without pulsation

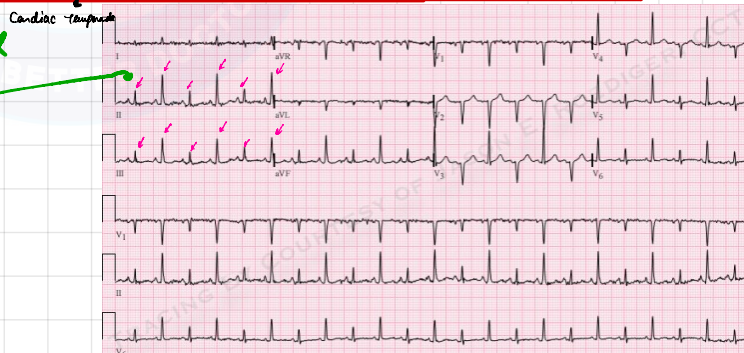


O/E — ● Bulging in epigastric area — Aurenbruger sign

● On inspiration — SBP fall > 10 mm of hg c/a — P. Paradoxeus +nt
But if patient in shock — P. Paradoxeus will not be appreciated

So while taking the BP in CT → patient should breathe normally

ECG — Low voltage complex
Electrical alternance



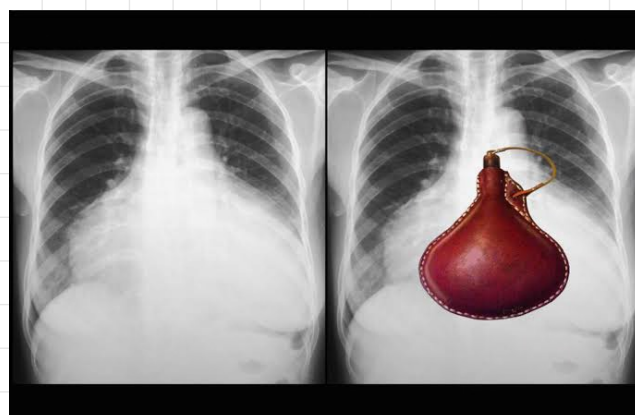
JVP — X descent → prominent

— Y descent → diminished

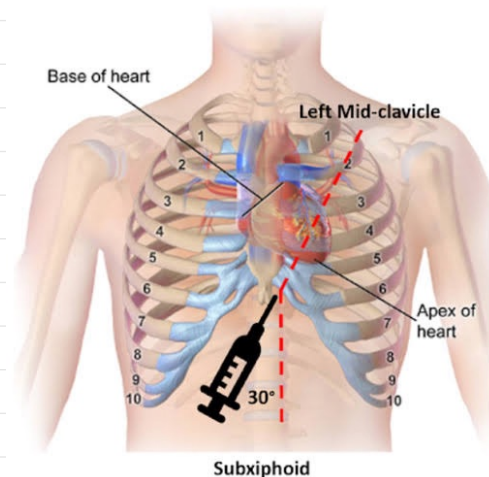
Kussmaul sign — -ve (Absent)

CxR — Money bag appearance or water bottle appearance

ECHO — ① pericardial fluid



Rx — **Pericardiocentesis**
 ↓
 Subxiphoid → 5th ICS → (L)
 ↓
 pointing towards
 (L) mid clavicular point



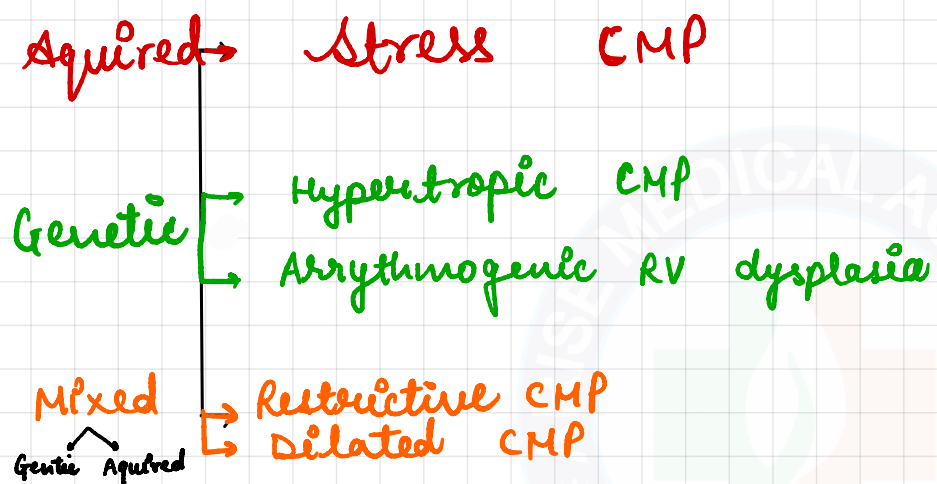
SUMMARY OF PERICARDIAL DISEASE v. imp *

CONSTRUCTIVE PERICARDITIS	CARDIAC TEMPONADE
Etiology TB	Etiology: idiopathic
NOT a common C/F chest pain	C/F - BP ↓ → distended neck vein → muffled heart sound
JVP - X → prominent - Y → prominent	JVP - X → prominent - Y → diminished
Kussmaul Sign +	Kussmaul sign -ve (L)
Pulsus paradoxus +	Pulsus paradoxus +
CxR egg in cup app.	CxR: Money bag app.
Echo - Thick pericardium - ✓ sign	ECG: low voltage electrical alternance
Rx Pericardiectomy	Rx: → Pericardiocentesis

CHAPTER 7 CARDIOMYOPATHY

DEFINITION → 1° disorder of cardiac muscle

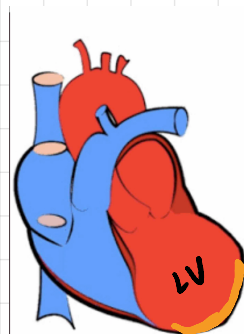
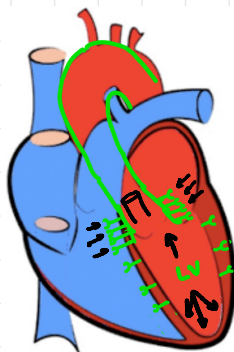
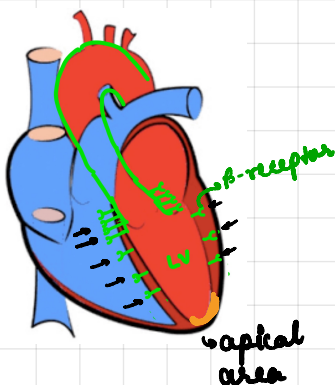
CLASSIFICATION BASED ON ETIOLOGY (CMP)



or → **Apical Balloning Syndrome**

1. STRESS CARDIOMYOPATHY / Broken Heart Syn. ❤️

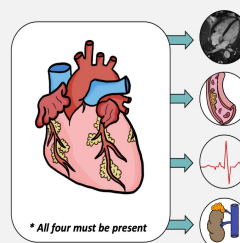
Pathogenesis: $F > M \rightarrow \uparrow \text{stress} \rightarrow \uparrow \text{sympathetic activity}$
 $\downarrow \uparrow \text{NE \& } \uparrow \text{Epinephrine}$
 $\downarrow \uparrow \beta \text{ Receptor}$



C/F: → 50 yr. ♀ → Emotional stress → FIB → Severe chest pain

ECG: → ST elevation + Troponin ↑

Mayo Clinic criteria for Tako-Tsubo cardiomyopathy*



- Revised Mayo Clinic Criteria
- Transient hypokinesia, akinesia, or dyskinesia of the left ventricular segments with or without apical involvement; abnormalities must extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present
 - Absence of obstructive coronary disease
 - New electrocardiographic (ECG) abnormalities usually ST-segment elevations, and/or T wave inversions or a slight increase in cardiac troponin
 - Absence of pheochromocytoma or myocarditis

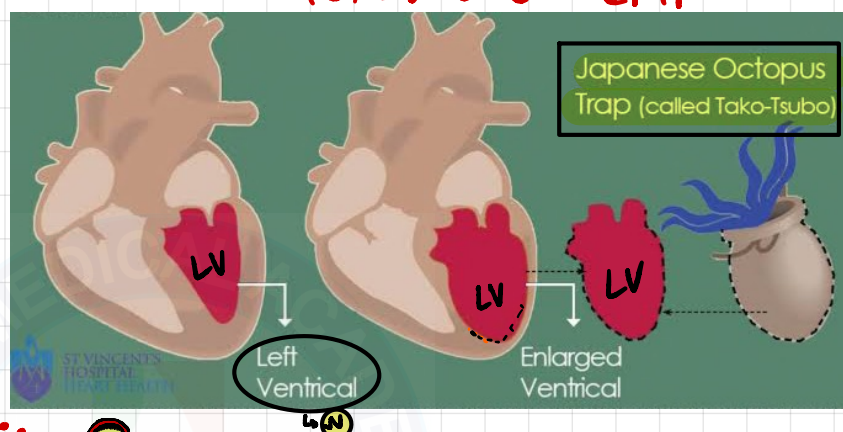
Angiography: → Normal

* Takotsubo CMP

Ventriculogram: → LV shape

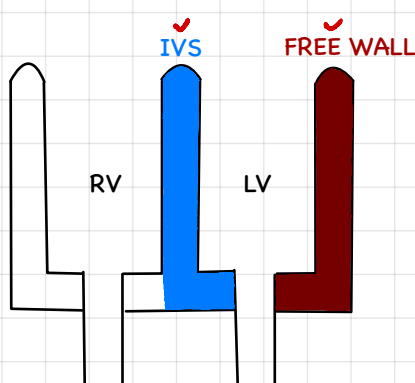
Rx: → β #

Prognosis: → Good prognosis (reversible process)



2. HYPERTROPHIC CARDIOMYOPATHY [H.CMP]

Pathogenesis - multiple mutation are a/w but m/c is → β-myosin Heavy chain mutation.



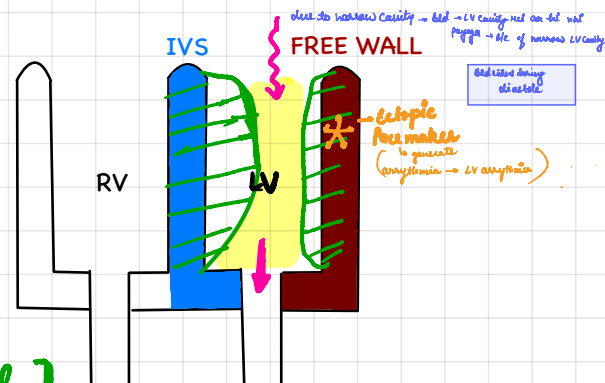
Chr. 14 → Mutation
Autosomal dominant mutation
Asymmetric Hypertrophy

(IVS > Free wall)

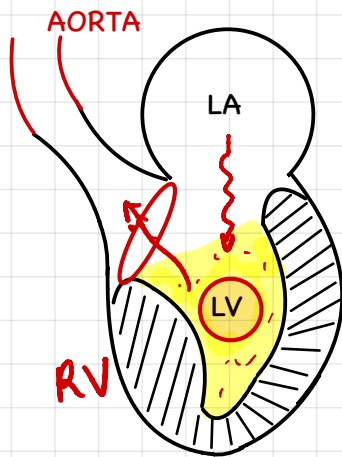
diastolic failure

have V. Arrhythmia

↓
Sudden cardiac death



bid - not during diastole

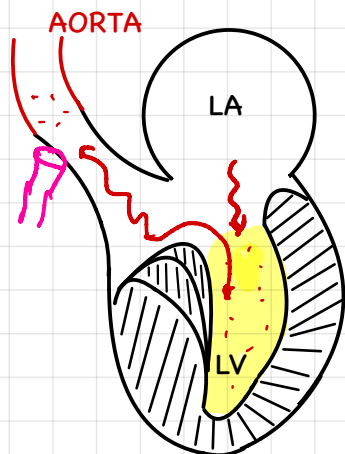


C/F - Asymmetric Hypertrophy

LV cavity size $\downarrow$

..... diastolic Failure $\rightarrow$ c/f $\rightarrow$ SOB on exertion
(due to $\downarrow$ supply of oxygenated bld)
 & LV is small & RV is normal sized)

Progressive increase in hypertrophy leads to



Obstruction..... so c/a H.O.C.M (Hypertrophic obstructive Cardiomyopathy)

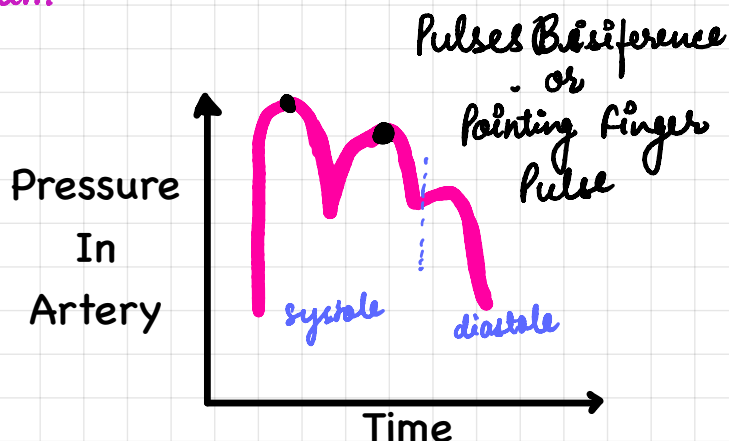
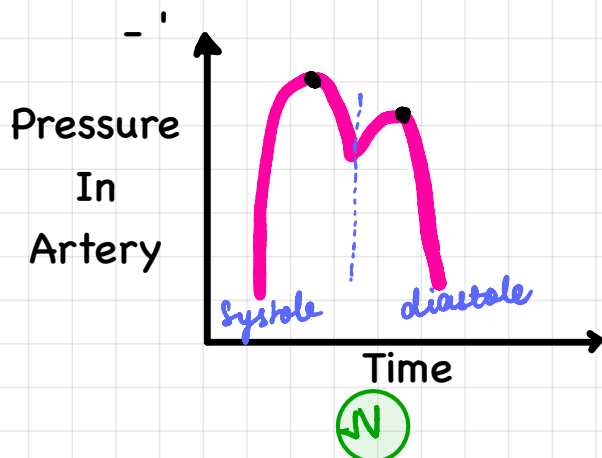
Leads to systolic failure

So stroke volume (blood in aorta) - $\downarrow\downarrow$

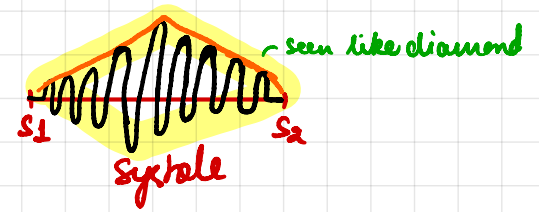
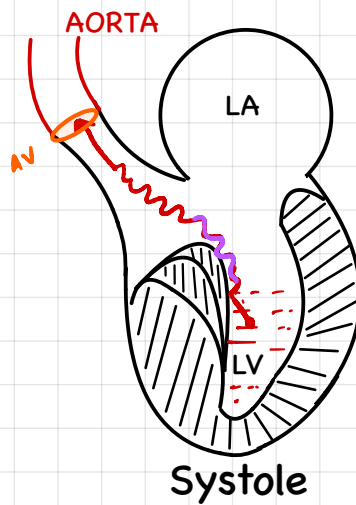
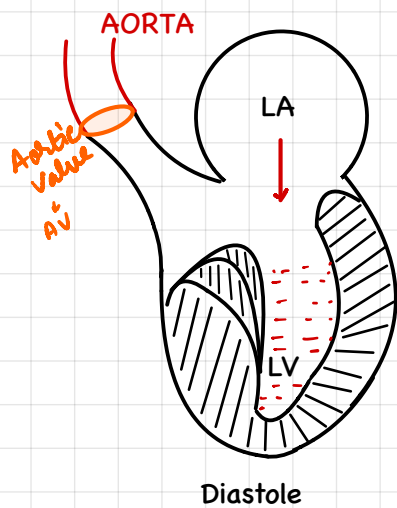
- Blood supply to hypertrophied muscle $\rightarrow$ $\downarrow$ $\rightarrow$ ischemia $\rightarrow$ CHEST pain
- Blood supply to brain $\rightarrow$ $\downarrow$ $\rightarrow$ syncope episode

☞ Hypertrophied ventricular muscle can lead to - V. Arrhythmia.....
 Which can lead to sudden Cardiac death

O/E - S4+ $\rightarrow$ Hypertrophied myocardium

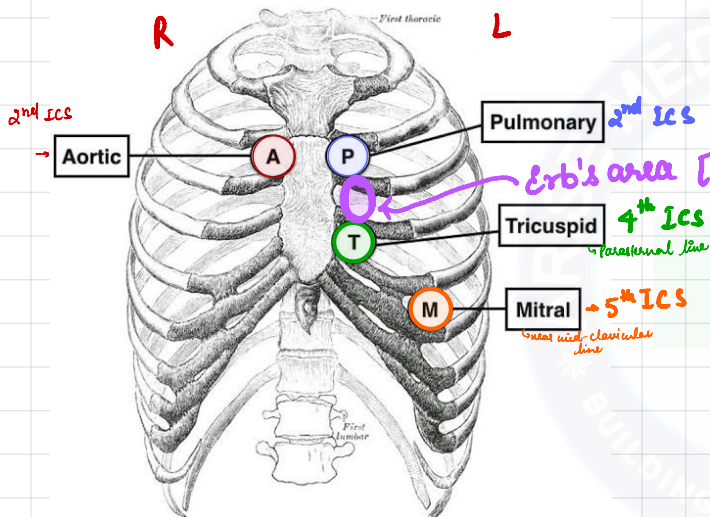


MURMUR → Turbulent bld flow



Crescendo-decrescendo
murmur
or

Diamond shape murmur

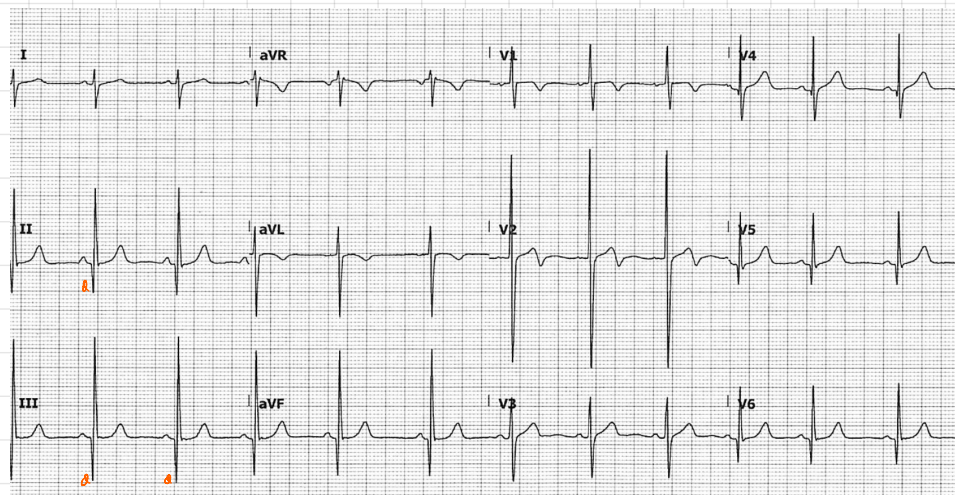


Murmur of HOCM → on standing / Valsalva ↓↓

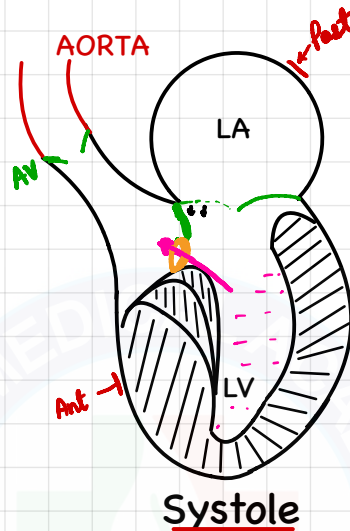
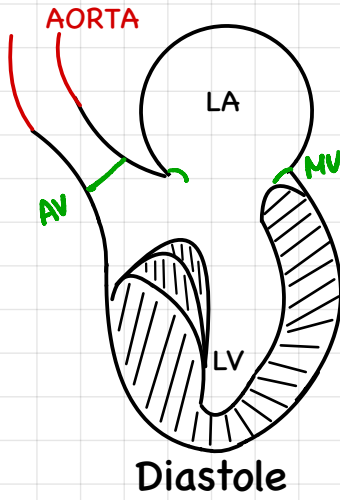
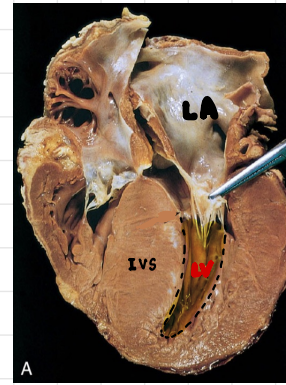
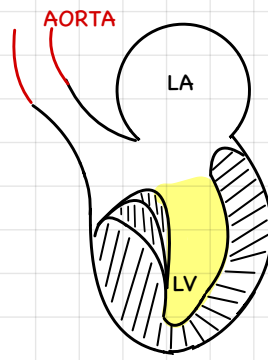
ECG -

→ LVH → $V_1S + V_5R \geq 35$

→ Pathological Q wave → II/III/aVF



Echo — LV cavity size ...↓...
and shape looks like Banana



systolic anterior motion of MV

SAM of MV

dynamic LV outflow obstruction

Rx — To improve diastolic filling — β # $\rightarrow$ $\downarrow$ HR $\rightarrow$ $\uparrow$ diastolic filling

To decrease cardiac remodelling and disease progression — β #

To prevent arrhythmia — Amiodarone $\rightarrow$ S/E $\rightarrow$ Hyperthyroidism
Hypothyroidism
Pulmonary fibrosis

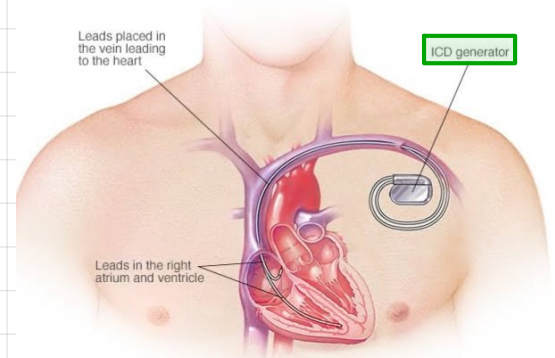
If there is high risk of sudden cardiac death — such as — family history
or LV muscle thickness is > 3 cm

★ Sx $\rightarrow$ Septal artery stenosis
Inj of ethanol for septal ablation (Implantable cardioverter defibrillator) [ICD]

MC S/E of ICD is — unnecessary shocks

pharma connection —

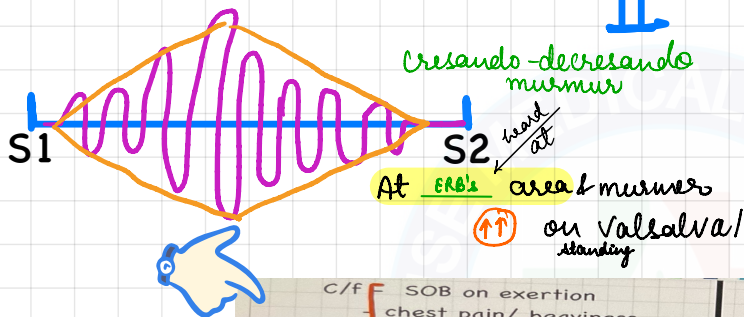
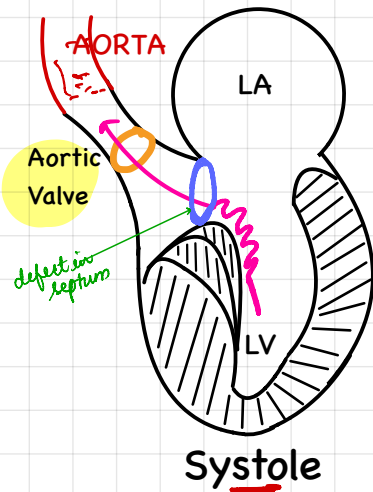
Drug C/I in HOCM — Diuretic
Dilator (nitrate)
Digoxin



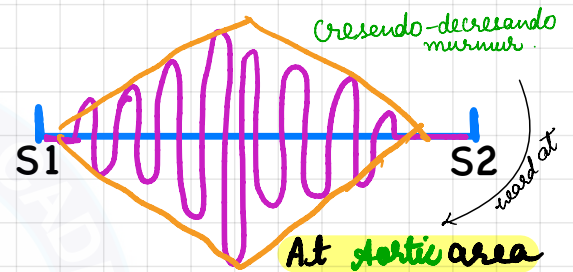
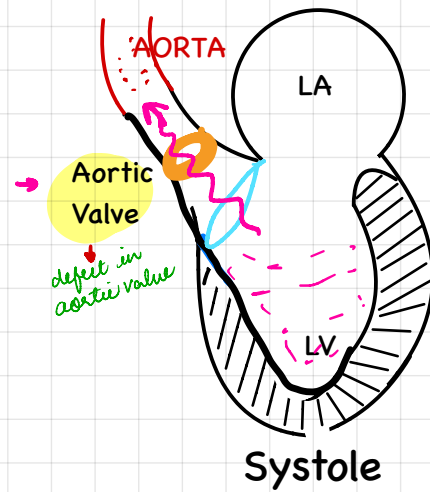
v.v. imp

Extra point

HOCM



Aortic stenosis

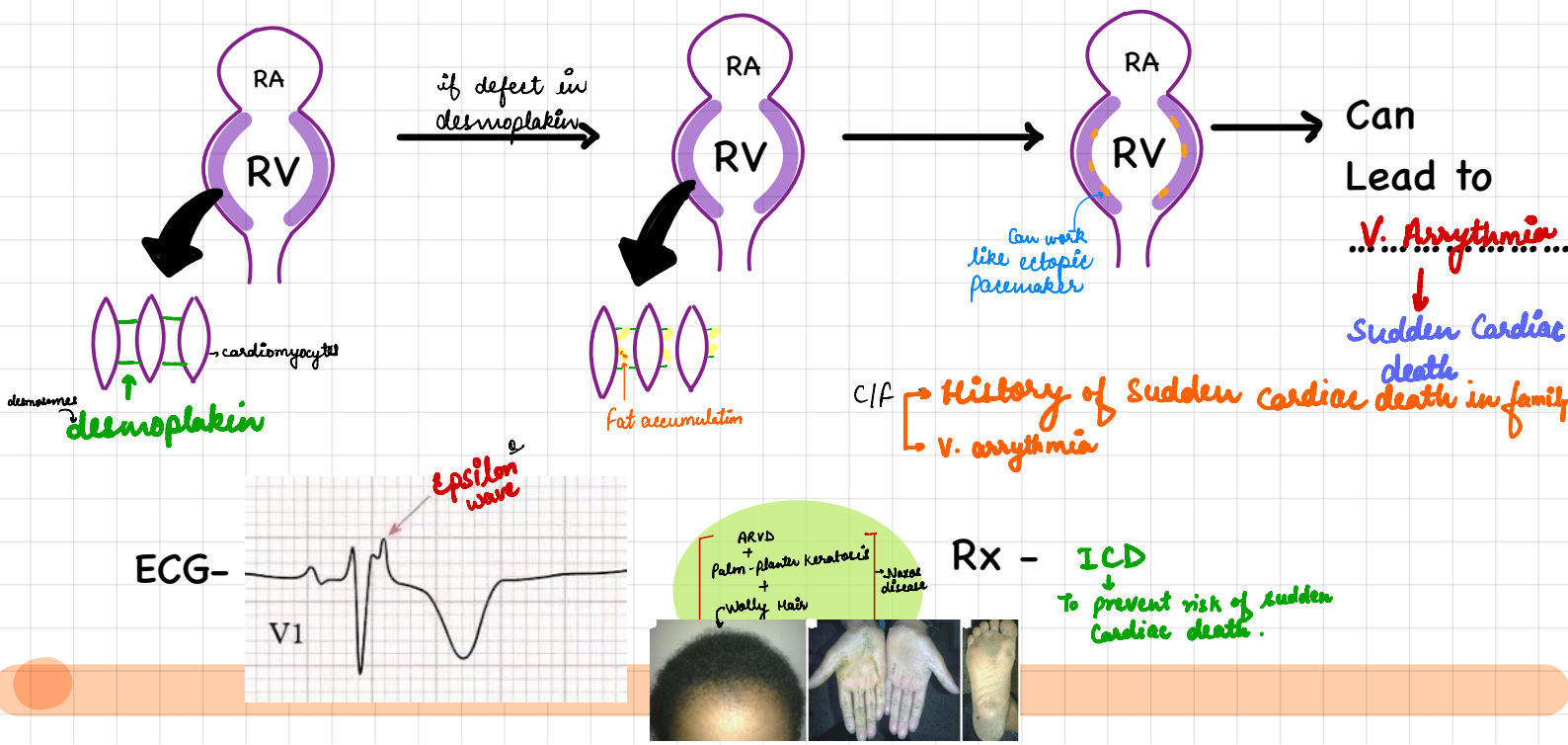


C/f	SOB on exertion chest pain/ heaviness syncope
Age	young
History of sudden cardiac death in family	⊕
S2 Abnormality	⊖ (due to closure of aortic & pulmonary valve)

C/f	SOB on exertion chest pain/ heaviness syncope
Age	old age
History of sudden cardiac death in family	⊖
S2 abnormality	⊕ (due to problem in aortic valve)

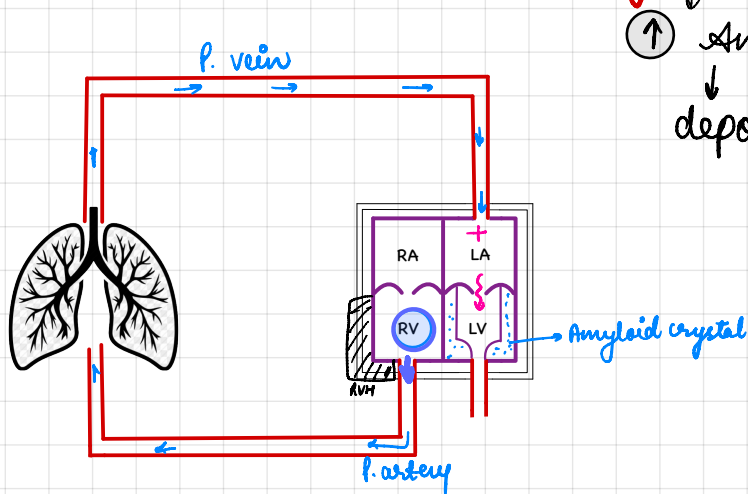
3. ARRYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA [ARVD]

Pathology:



4. RESTRICTIVE CARDIOMYOPATHY

Pathogenesis: → MCC → Amyloidosis



↑ Amyloid

deposite in muscle of (LV)

(LV) stiff

↓ diastole of (LV)

↑ strain on (RV)

↓ (RVF) mainly

MCC — Amyloidosis

Other Causes

- Gaucher disease → deficiency of β -glucocerebrosidase
- Fabry disease → deficiency of α -galactocerebrosidase
- Sarcoidosis
- drug — [Methysergide, Ergotamine]

C/F — same as RVF

O/E → ↑ JVP / Hepatomegaly / Ascites

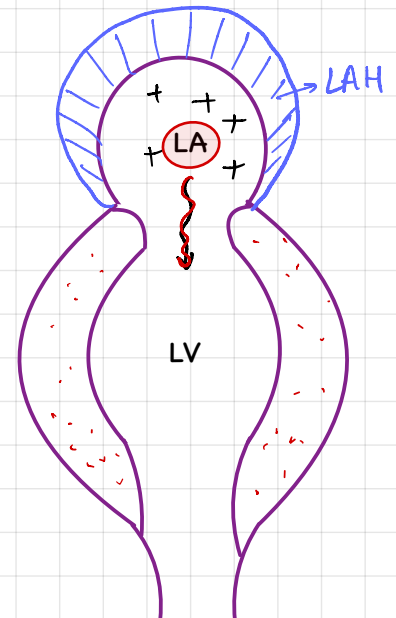
Kussmaul sign → ⊕ [on inspiration → ↑ JVP]

✶ ECHO → LV diastolic dimension → ↓

→ LA size — ↑

→ If Amyloid deposited

→ Glittering myocardium



Rx → DOC → β # → ↑ diastolic filling + ↓ cardiac remodelling

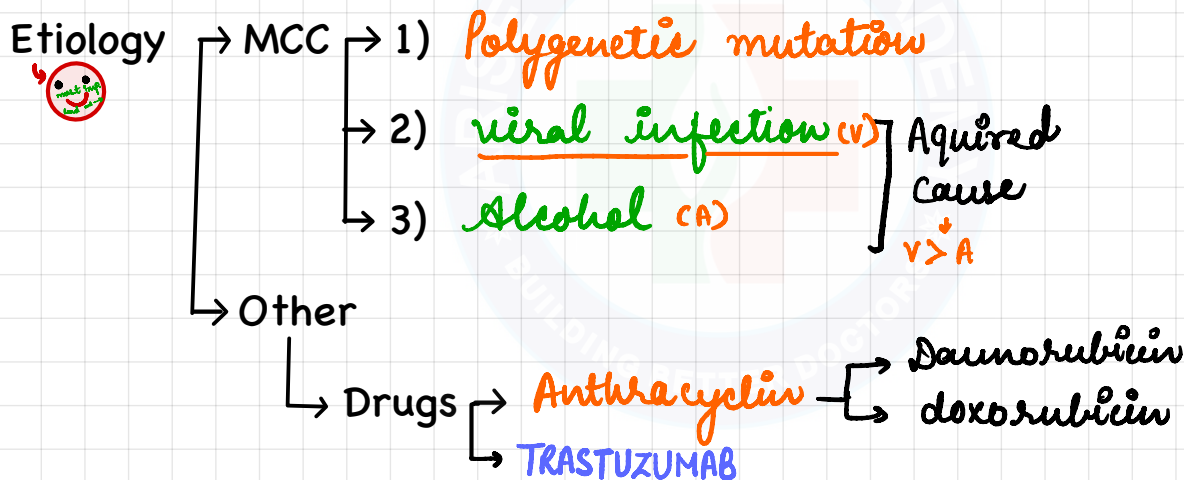
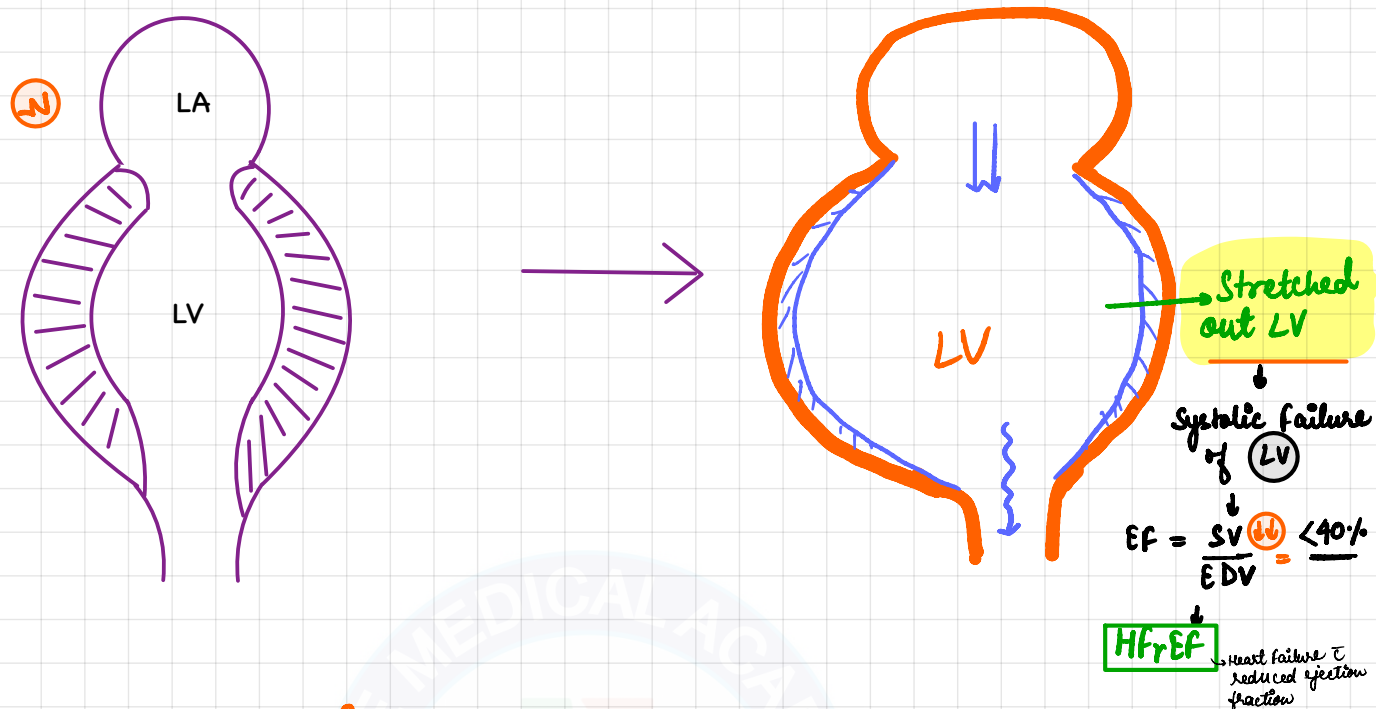
→ C/I ~ Digoxine

→ Treat the underlying cause

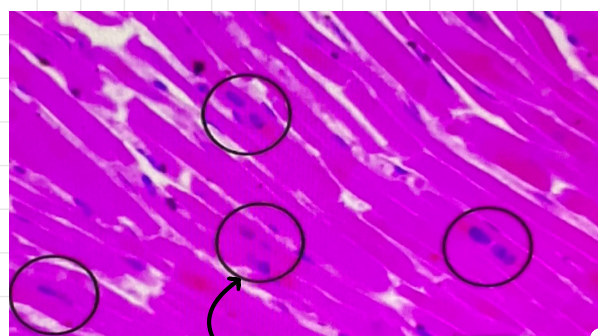
Amyloid	
• stain → Congo Red	
• Alz. disease	Amyloid deposit
• Hemo, dialysis	Amyloid β_2
	Amyloid β_{42} [extraneuronal acc ^m]

5. DILATED CARDIOMYOPATHY [DCMP] → mc Cardiomyopathy

Pathogenesis:



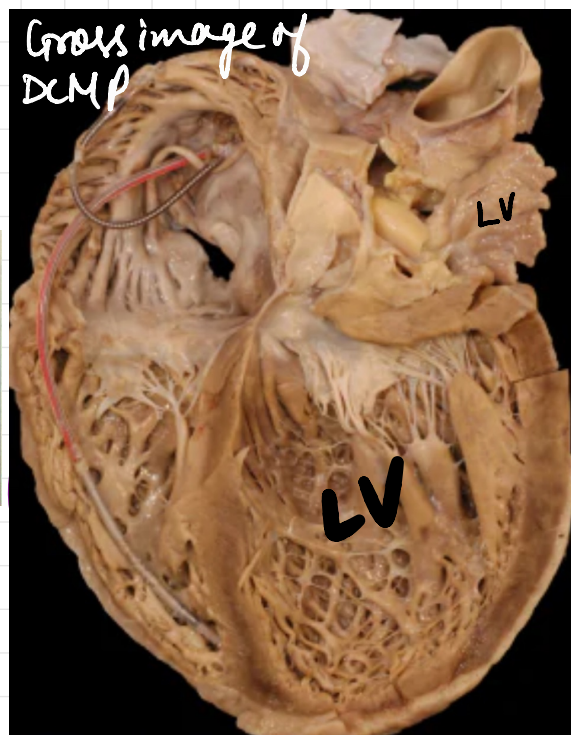
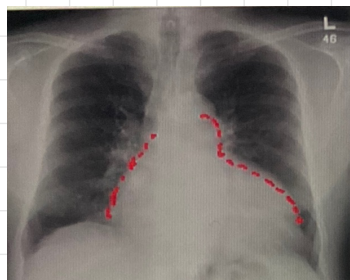
- infection → Para virus / diphtheria
- vitamin deficiency vit (B1)
- Mineral deficiency Selenium → KESHAN disease
- Duchenne muscular dystrophy
- Peripartum → ↑ Prolactin → DCMP
[3rd trimester → 6 month (post partum)]
- Endocrine Cause → PCC → Hyperthyroidism



Multinucleated myocardium

C/F → Same as HF [LVF]
 ↓
 → dyspnea [PND / orthopnea]
 → fatigue

O/E → Same as HF
 ↳ Dicrotic pulse



ECHO - LV cavity size ↑

- SV $\downarrow$
 (Stroke vol^m)

- EF = $\frac{SV}{EDV} \times 100 = <40\%$

Rx → Same as HFrEF - ABCD
 ↳ Treat the underlying cause

AB → ACE # / ARB / ARNI / Aldo. Reop. antagonist
 ↳ β#

CD → ↑ contractility → Digoxine
 ↳ Diuretic / dilator



EXTRA POINT

MCC of sudden cardiac death in young

- 1) HOCM
- 2) ARVD
- 3) Brugada Syndrome

Brugada Syn → Susil → ECG

Pathology → Sodium channel 5A defective [SCN5A]

Feature → asymptomatic (ST) elevation

Inv. ECHO / Electrophysiology study →

Rx → ICD

V₁

V₂

V₃



asymptomatic
 ST elevation

Summary

- * ARVD → desmoplakin defect → fat deposition
- * RCMP → Amyloidosis → (LV) stiff → strain RVF
- * DCM → etiologies → HFrEF
- * Brugada Syn → Defect SCN5A → asymp
↓
ST elevation
[V₁-V₂]

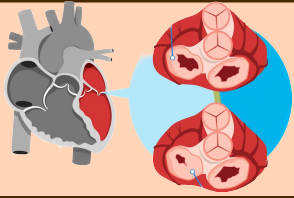
🫀 Stress CMP → a/c/a Taket atheros CMP/ Apical ballooning or broken syndrome.
 → C/F → Trigger → Emotional stress F/b severe chest pain
 → DOC → β #

-  **HOCM**
 - Ch. 14 → β myosin Heavy chain mutation. (AD mutation)
 - Asymmetric Hypertrophy
 - C/F - diastolic failure → ... SOB on exertion
 - ↓ F/b
 - Systolic failure → ... Stroke val^m (↓) → ischemia syncope episode
 - O/E
 - S S4 ⊕ / P. Hypertrophied myocardium
 - crescendo-decrescendo or Diamond shape murmur at Erb's area.
 - Echo
 - LV cavity Banana shape.
 - SAM of MV
 - Rx
 - DOC → β #
 - C/I → Diuretics / Dilator / Digoxin.

Restrictive CMP

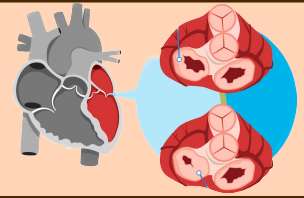
- MCC → Amylotoclosis
- C/F → same as RVF
- ECHO → Glittering myocardium

-  Dilated CMP
 - MCC
 - 1) Polygenetic Mutation
 - 2) viral infection
 - 3) Alcohol
 - Drugs
 - doxorubicin
 - doxorubicin
 - doxorubicin
 - doxorubicin
 - Peri-partum is d/t $\uparrow$ Prolactin.
 - C/F → same as HF HF EF.



CHAPTER 8

RHEUMATIC FEVER

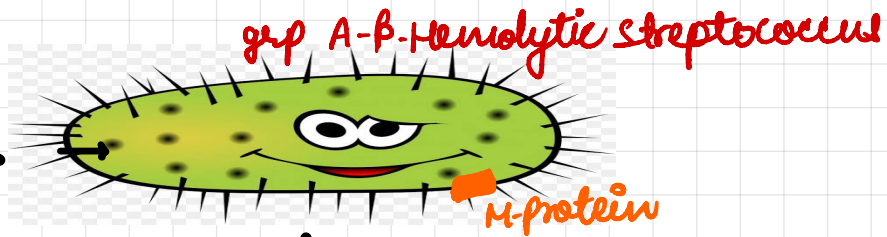


PATHOGENESIS

Age - 5-15 year [M=F]



Acute pharyngitis
↓
Recurrent Acute pharyngitis
↓
Genetic susceptibility

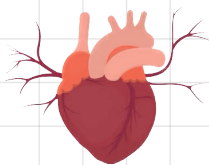


Antibody levels

cross reacting [Molecular mimicry]

Type II Hypersensitive Reacⁿ

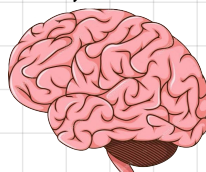
RF



Heart



joint



neuron



Skin



S-C Tissue

if Permanent damage
↓

c/a

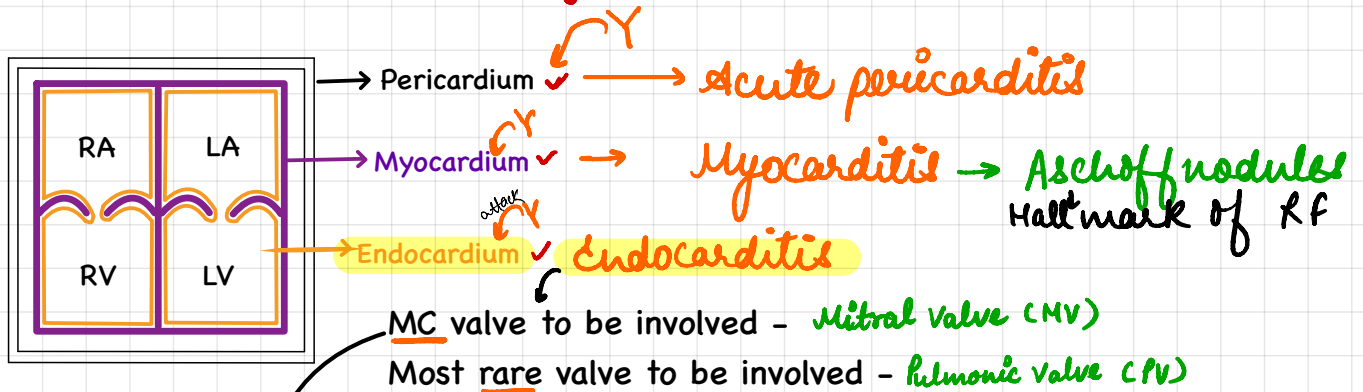
RHD

if permanent joint damage
↓ c/a

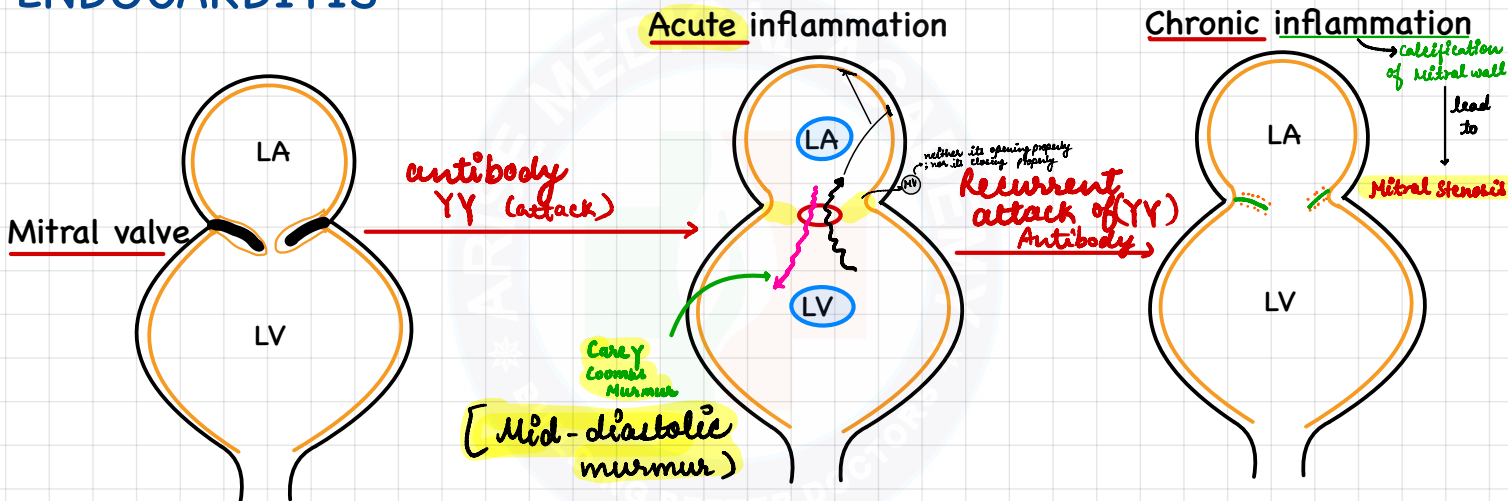
Jaccoud's arthropathy

C/F

a) Cardiac involvement → early & common → Pancarditis

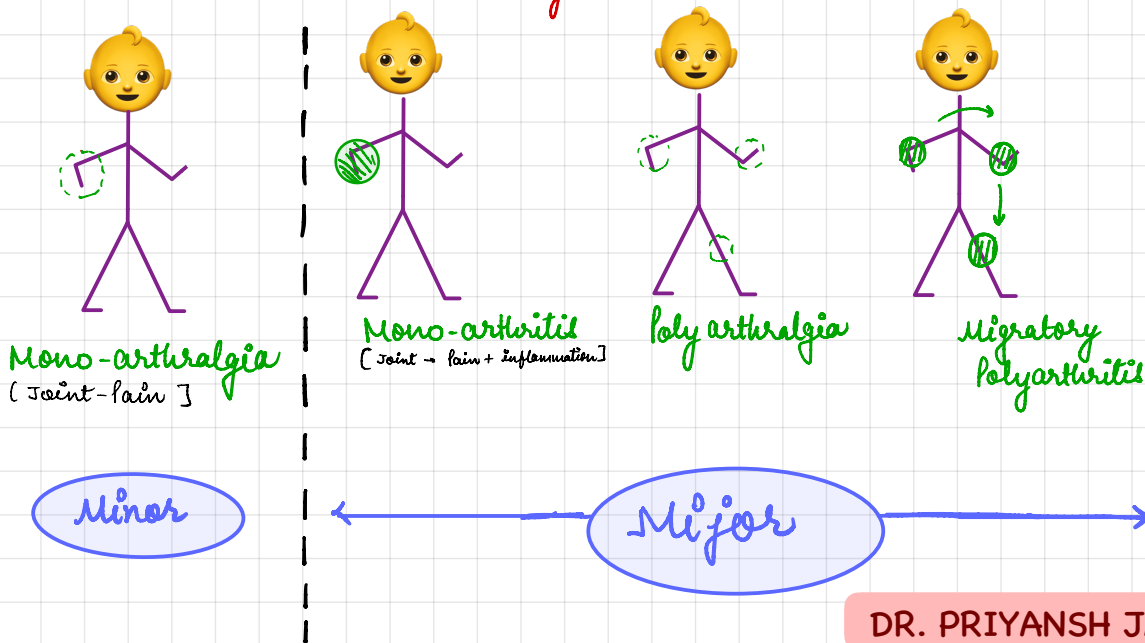


ENDOCARDITIS



Thickened post. wall of LA called as **McCallum's patch**

b) Joint Involvement → early & common

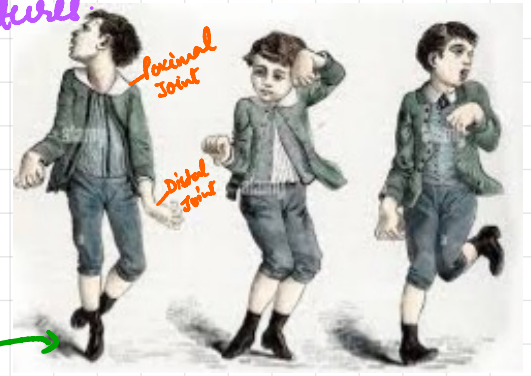


c) Neuronal Involvement → Late & rare features

Involuntary movement, Rapid

[Distal joint > Proximal joint]

ca → Sydenham's Chorea
or
St. Vitus dance



d) Skin Involvement

Red, ring shaped lesion
ca ↓
Erythema Marginatum



e) Subcutaneous tissue involvement

Painless S.C. nodules



INVESTIGATION → ESR ↑ , CRP ↑
→ To find grp-A-streptococcus

- Throat swab culture
- Anti-streptolysin "O" titer [ASO]

CRITERIA FOR DIAGNOSIS

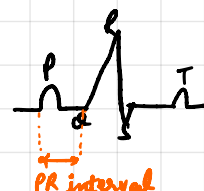
Revised Jones Criteria → Major → ⑤
Minor → ④

② Major or ① major + ② Minor
↓
⊕ evidence of streptococcus
↓
RF

Revised JONES criteria for High Endemic Area

- MAJOR
- 1) **J** → Joint involvement {
 - Mono arthritid
 - Polyarthralgia
 - Migratory Polyarthritids
 - 2) **♥** → Carditis
 - 3) **N** → Neuronal invol^m → Syd. chorea
 - 4) **E** → Erythema Marginatum
 - 5) **S** → S.C nodules

- MINOR
- 1) → ↑ ESR & ↑ CRP
 - 2) → Fever
 - 3) → ↑ PR interval
 - 4) → Mono arthralgia



- Rx — for joint pain → NSAIDS → Aspirin
- for carditis → Steroid
 - for chorea → Self limiting / Na valproate $\xrightarrow{\text{Rx}}$ I/V Ig (Immunoglobulin)
 - for skin rash/ subcutaneous nodule - Self limiting

For prophylaxis of β -H.G.A. streptococcus → inj. of Benzathine Penicillin $\times 21^{\text{st}}$ day

HOW LONG

- RF without cardiac involvement → 5 year @ till the age of 21.
- RF+Cardiac involvement but Recovered → 10 year @ till the age of 21.
- RF+Residual cardiac involvement → 10 yrs @ till the age of 40.
- If allergic to penicillin. - Erythromycin

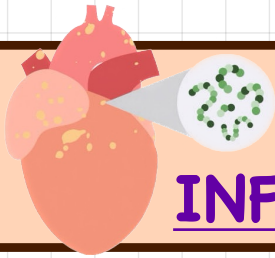


SUMMARY OF RHEUMATIC FEVER

- ♥ Etiology → Infection of → G-A. β -H. Streptococcus / strep. pyogenes
- ♥ Antibody against → (M) protein cross reacts
- ♥ Eg. Of type II Hypersensitivity Reaction
- ♥ Cardiac involvement → Hallmark → Aschoff nodule.
- ♥ Acute endocarditis → leads to → (Mitral Regurgitation → COREY COOMB murmur [Mid-diastolic])
Mitral stenosis
- ♥ Ch. Endocarditis → leads to Mitral Stenosis
- ♥ Skin findings → Erythema Marginatum
- ♥ Neuronal Finding → Syde. Cornea
- ♥ Δ Revised Jones Criteria
- ♥ Prophylaxis → by inj. Ben. penicillin every 21st day.

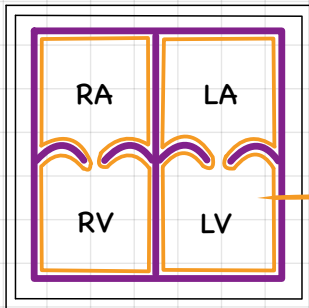
Pharma connection

Inj. Benzathine penicillin is also used for treatment of cardiovascular syphilis - 2.4 million unit once weekly for 3 weeks.



CHAPTER 9

INFECTIVE ENDOCARDITIS



Endocardium

Overall MCC → staph. aureus

Cardiac lesion with → Highest risk of I.E. — M.R. (mitral regurgitation)

→ Lowest risk of I.E. — ASD

* So, ASD patient does not require prophylaxis for IE

I.E. — after valvular Sx (surgery) → < 2 month of surgery → staph. epidermidis

→ > 2 month of surgery → strep. viridans

Sub acute Bacterial
Endocarditis (SABE)

For I/V drug abuser MC valve involved is — TV (Tricuspid Valve) & MC organism is Staph. aureus

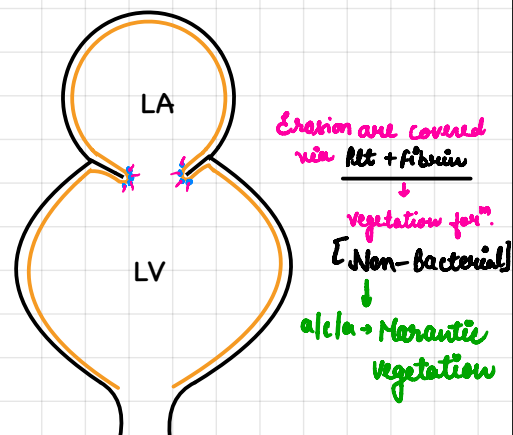
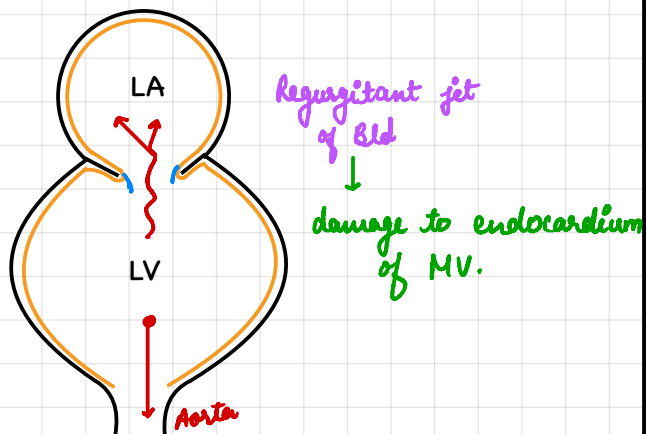
I/V drug abuser if left side heart involved then MCC → Enterococcus

For colon cancer patient MCC — Strep. bovis

V.IMP

PATHOGENESIS

20yr-70yr
HF = MR
Aortic
RND



Bacteremia



Skin wound / dental procedure

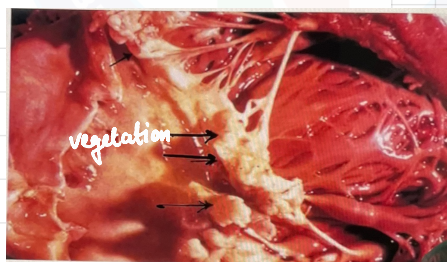
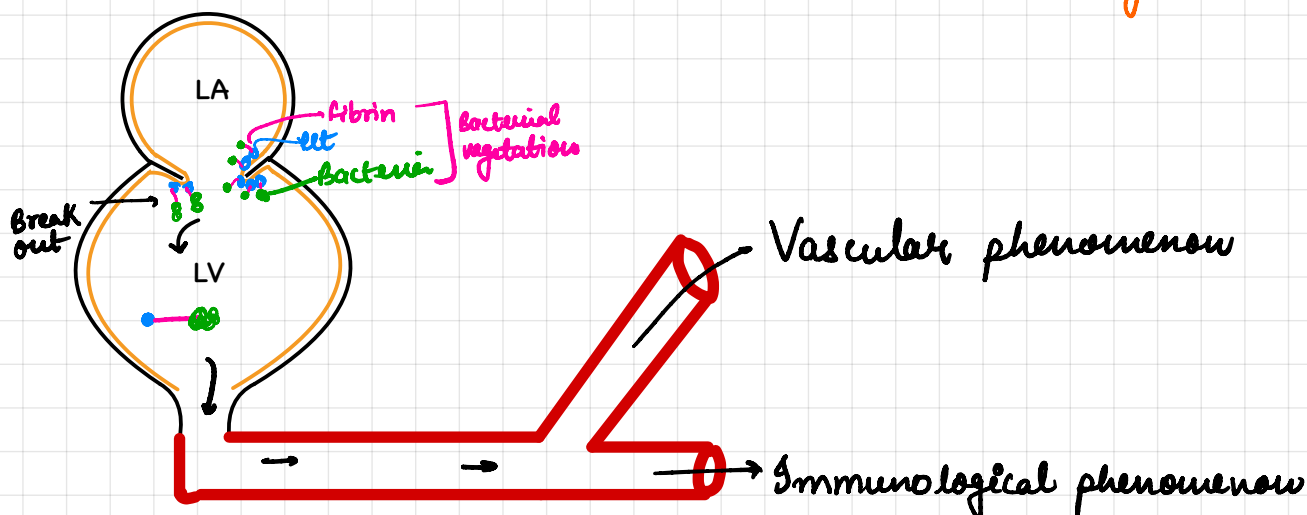
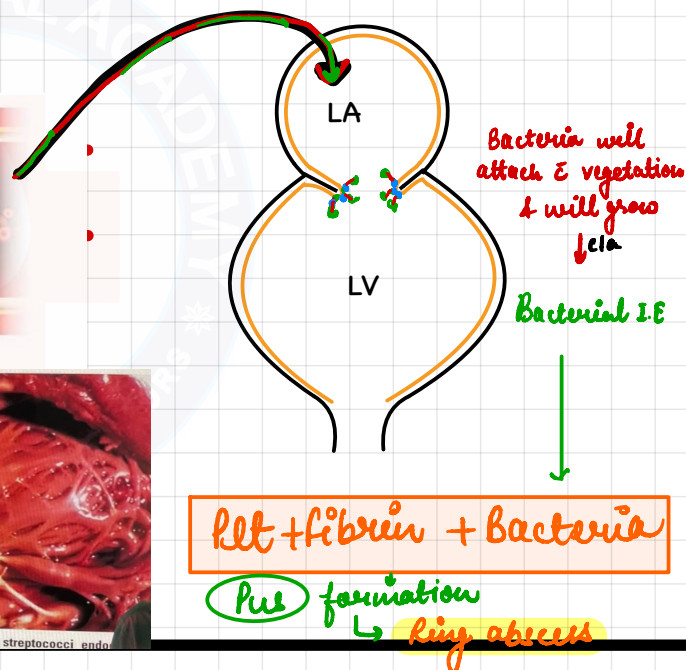
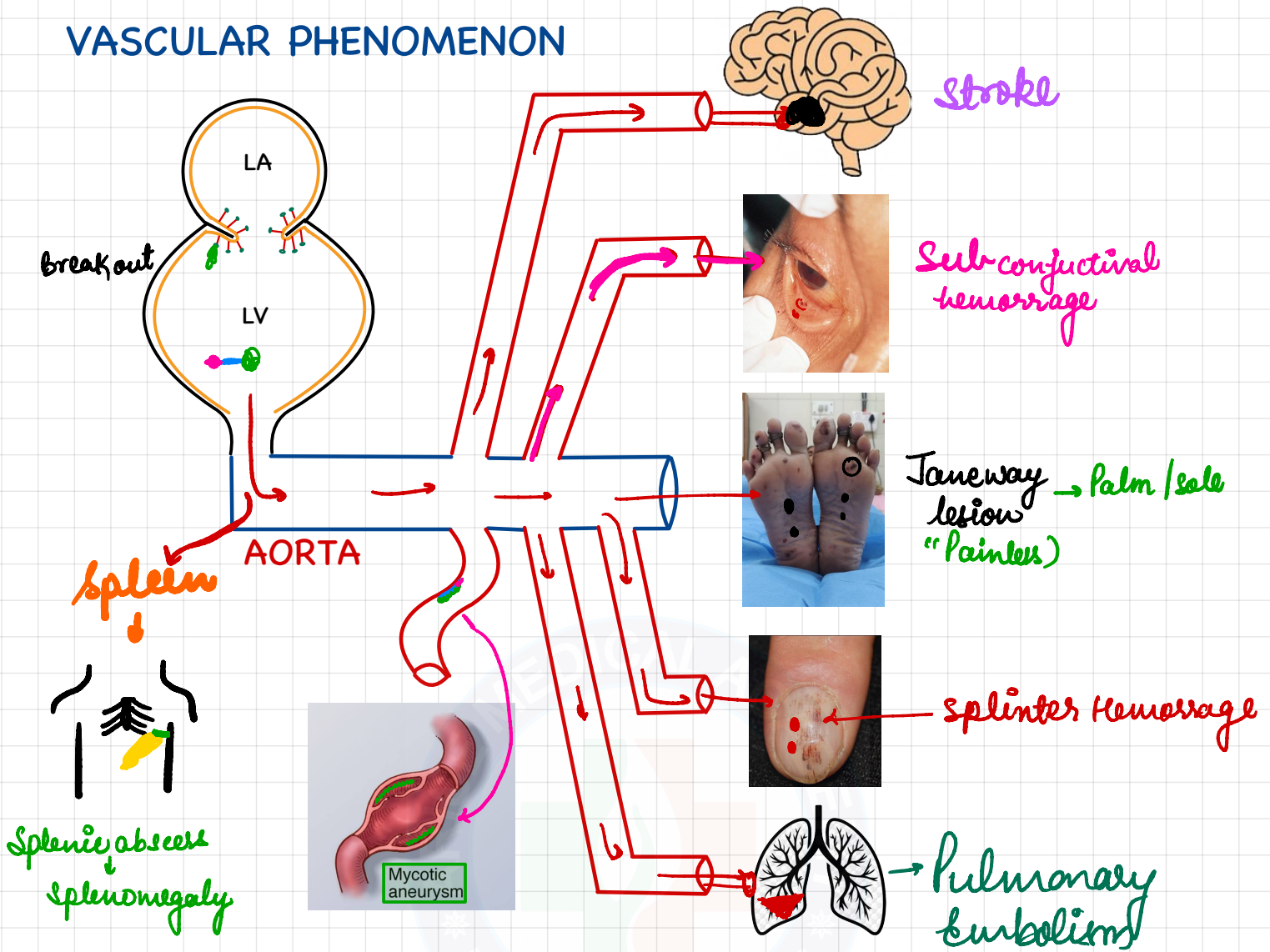


FIGURE 128-1 Vegetations (arrows) due to viridans streptococci endocarditis involving the mitral valve.

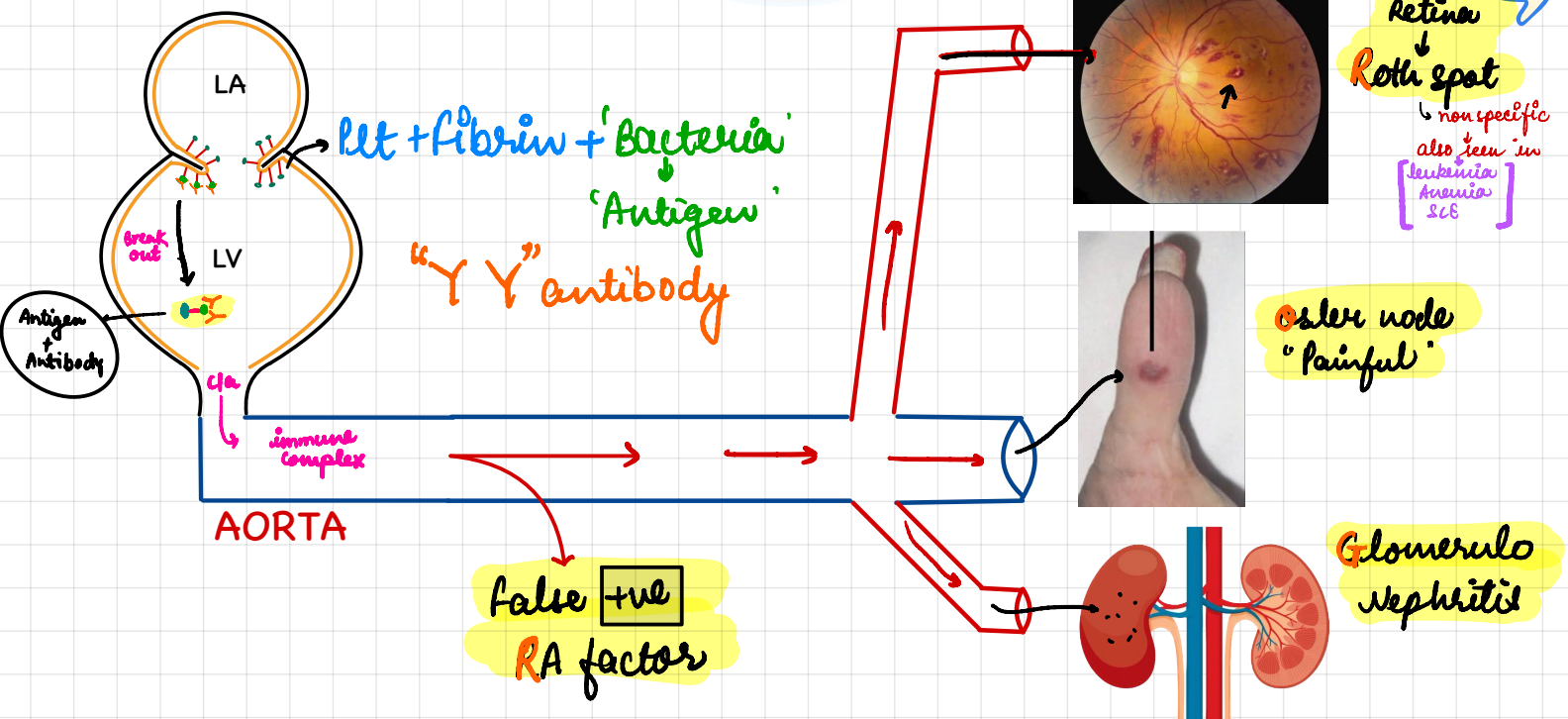


VASCULAR PHENOMENON



IMMUNOLOGICAL PHENOMENON

ROG



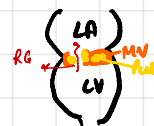
Imp

For - Diagnosis - Modified Duke's criteria.

[Major → ②]
[Minor → ⑤] → 2 Major / 1 Major 3 minor / 5 minor → I.E

MAJOR - 1) ECHO → Vegetation / Ring abscess / New murmur

2) Bld. culture → at least ③ set
[2 bottle] → [1 aerobic / 2 anaerobic] → at least ② set should be +ve for same bacteria
[but for *Coxiella burnetii* → even ① Bld culture + → considered major]



MINOR - 1) Presence of Risk factor → Valvular defect / IV drug abuser
2) → Fever
3) → Any vascular phenomenon
4) → Any immunological phenomenon
5) → if only one Bld culture is +ve [Microbiology evidence]

Rx - Empirical antibiotics → FIB → Change Antibiotic a/c Bld cult -ve report



SUMMARY OF INFECTIVE ENDOCARDITIS

♥ MCC → *Staph. aureus*

♥ Cardiac lesion → Highest risk → MR
→ Least risk → ASD

♥ SABE → Etiology → *Strep. viridans*

♥ Vascular Phenomenon → Nails → *splinter hemorrhage*
→ Palm & sole → *JANWAY lesion* [painless]
→ Spleen → *Abscess*

♥ Immunological phenomenon → Eye → R → *Roth spot*
→ Finger → O → *Osler node* [painful]
→ Kidney → G → *GN*

♥ Δ Modified Duke's criteria.

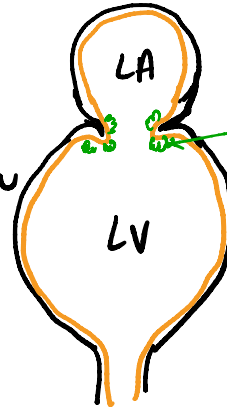
→ Vegetation can also be seen in

SLE

Malar rash + vegetation on both side of MV

Limbic Sac Endocarditis

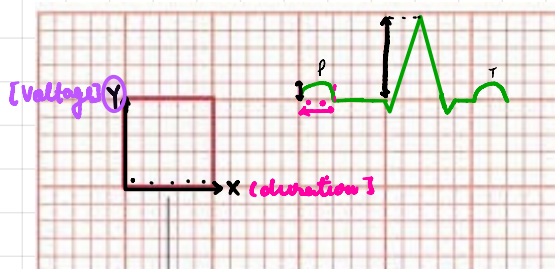
verrucous vegetation or wart like veg.



verrucous or vegetation
wart like vegetation

CHAPTER 10

ECG



1 Big Box = 5 small box in X-axis

1 Small Box = 40 mS.

ECG lead

Chest lead

V₁-V₆

Limb lead

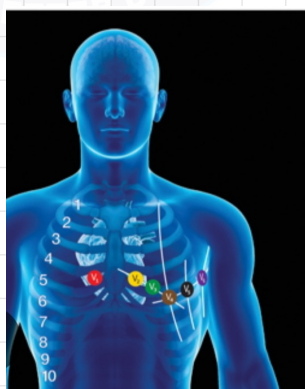
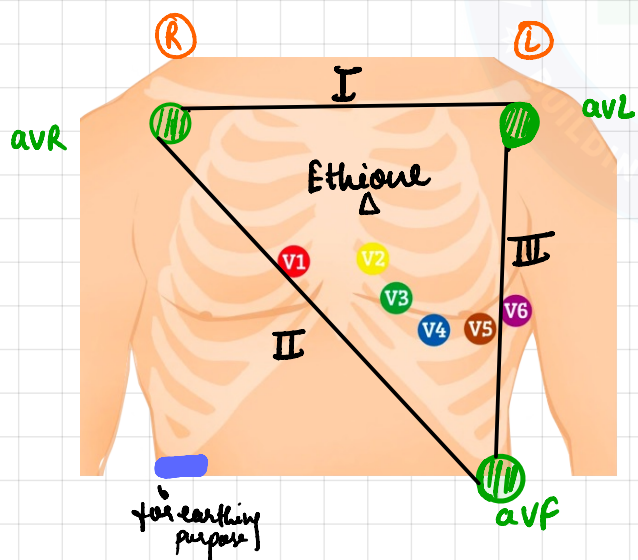
Unipolar

- avR
- avL
- avF

Bipolar

- avR + avL → I
- avR + avF → II
- avL + avF → III

ECG LEADS

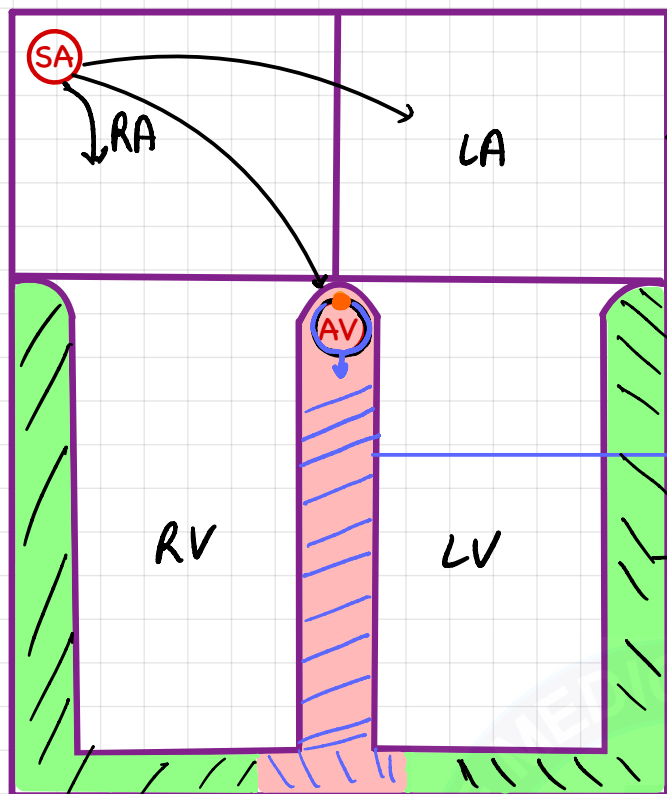
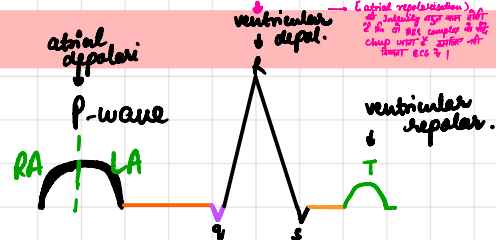


- V₁ 4th intercostal space to the right of the sternum
- V₂ 4th intercostal space to the left of the sternum
- V₃ Directly between the leads V₂ and V₄
- V₄ 5th intercostal space at midclavicular line
- V₅ Level with V₄ at left anterior axillary line
- V₆ Level with V₅ at midaxillary line (directly under the midpoint of the armpit)

* Muscle depolarisation → activate → contraction

* Muscle Repolarisation → Relax

CONDUCTION SYSTEM OF HEART



atrial depolarisation → P wave

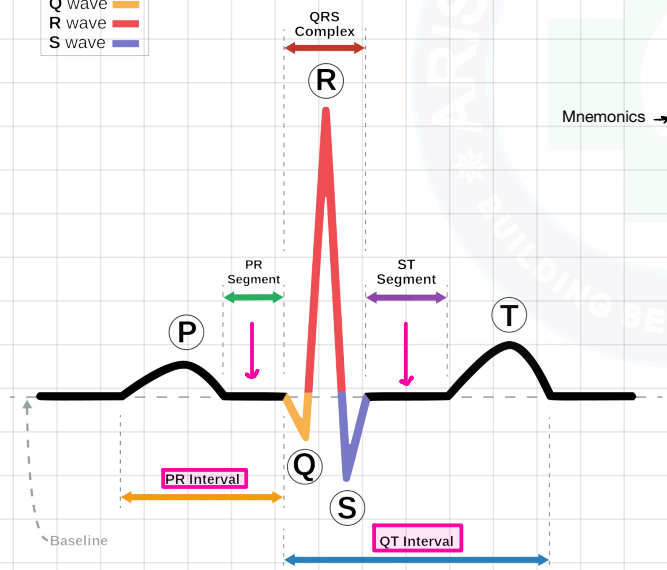
Septal depolarisation → Q-wave

Remaining ventricular depolarisation → RS wave

QRS complex
 Ventricular depolarisation

T wave → Ventricular Repolarisation

Q wave
 R wave
 S wave



Mnemonics →

Q = 1 small box

QRS = 2-2½ small box

P = 3 small box

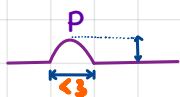
PR = 4 small box

T = 5 small box

QT = 10 small box

ECG NORMS → II lead

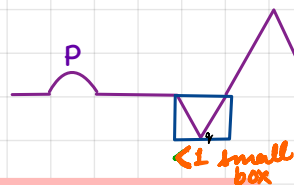
P wave



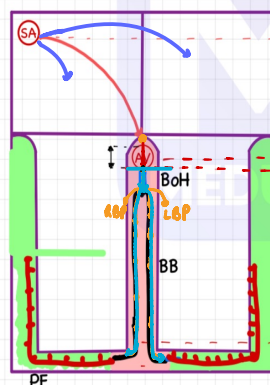
PR interval



q wave



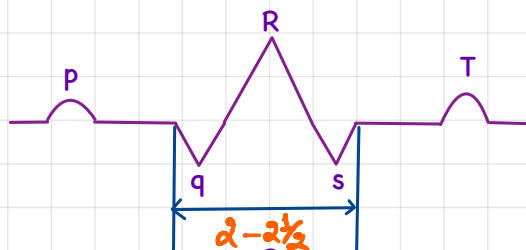
CONDUCTION SYSTEM OF HEART



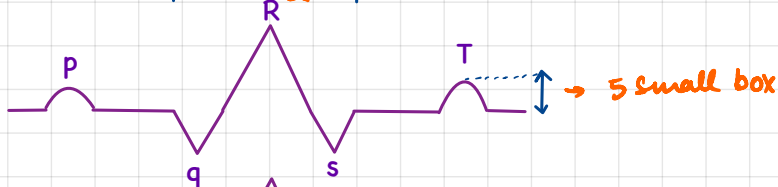
Conduction from SA node till completion of AV node is called as PR interval.

Conduction through bundle of His to Bundle branches is called as HV interval.

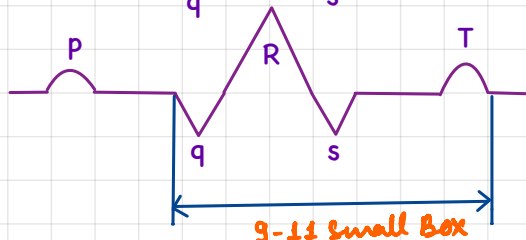
qRS complex



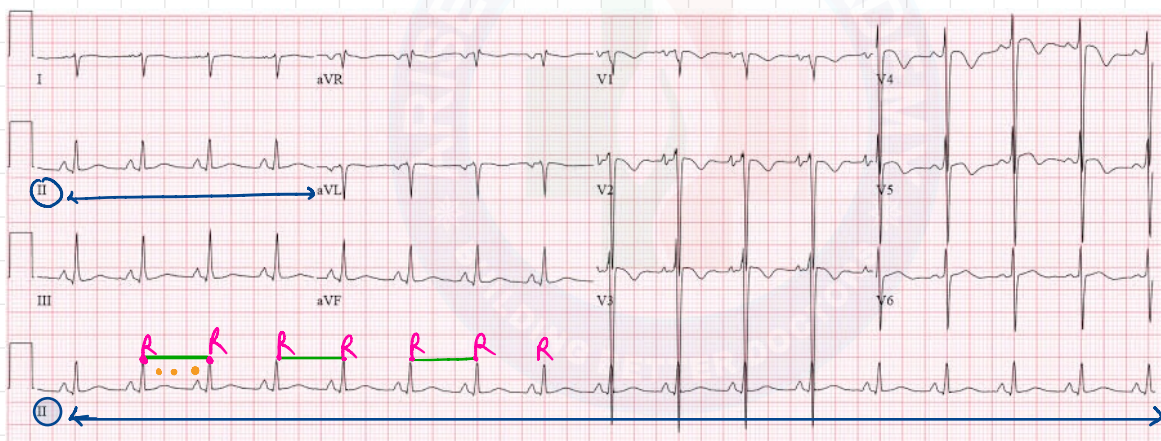
T wave



qT interval



Calculation of Heart Rate



for regular rhythm $\Rightarrow$

HR =

$$= \frac{300}{\text{R-R interval [Big Box]}} = \frac{300}{3} = 100 \text{ HR}$$

$$= \frac{1500}{\text{R-R interval [small box]}}$$

$$= \frac{1500}{15} = 100 \text{ HR}$$



For IR-regular rhythm

$$\text{HR} = \frac{\text{No. of R wave in 30 big boxes}}{30} \times 10$$

in this case $\rightarrow 10 \times 10 = 100$
HR = 100

1 small box = 40 ms

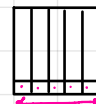
1 big box = $5 \times 40 = 200 \text{ ms}$

200ms = 1 Big Box

1000ms = $1 \times 1000 \div 200 = 5 \text{ Big Box}$

1 sec = 5 Big Box

6 sec = 30 big box

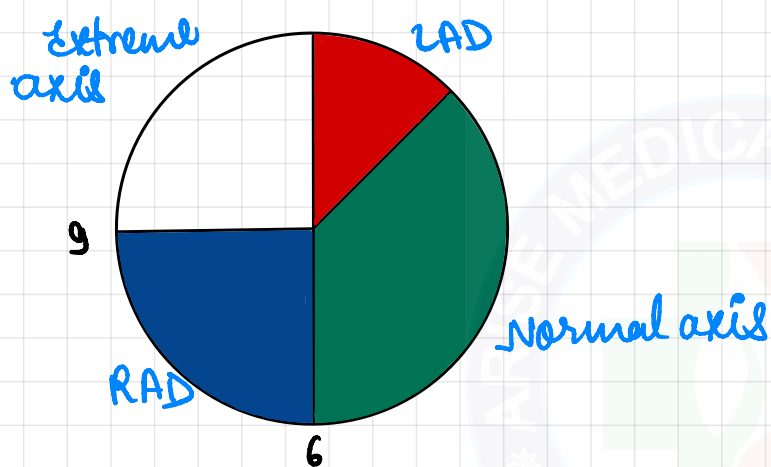


ECG AXIS

Low

Reaching

	Normal	Left axis	Right axis	Extreme Axis
I				
aVF				

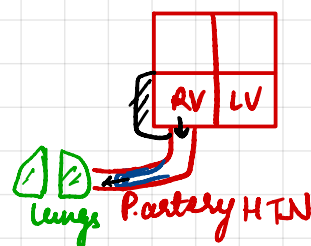
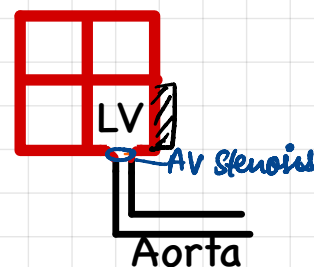


6-9 is Rt line
Mnemonic

So → RVH → RAD
→ LVH → LAD

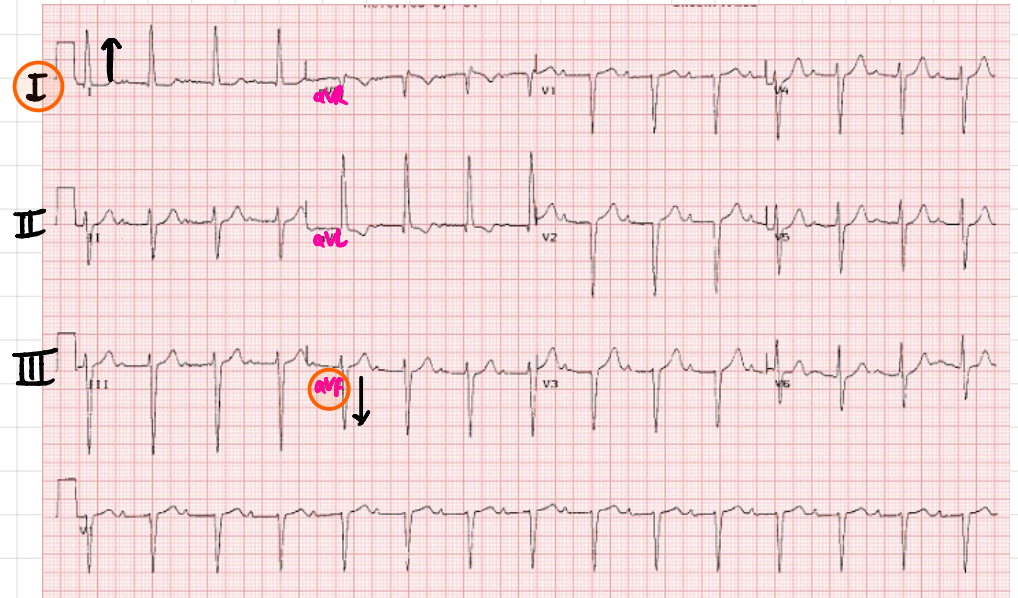
Axis

- Uncontrolled HTN → LVH → LAD
- Aortic Stenosis → LVH → LAD
- Pw. Artery HTN → RVH → RAD
- Ch. Lung Pathology → RVH → RAD
- Tetralogy of Fallot → RVH → RAD

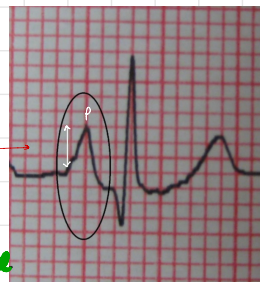
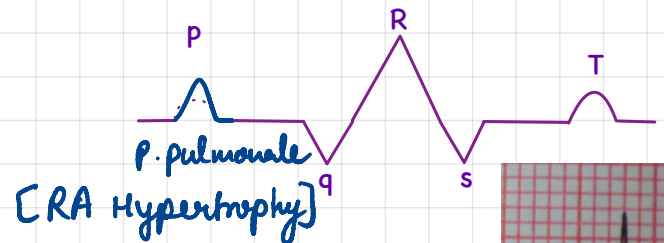
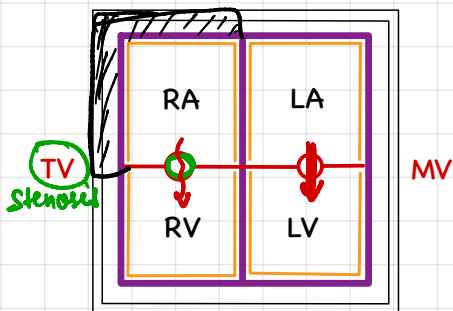
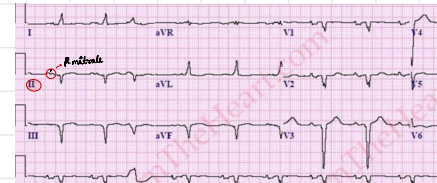
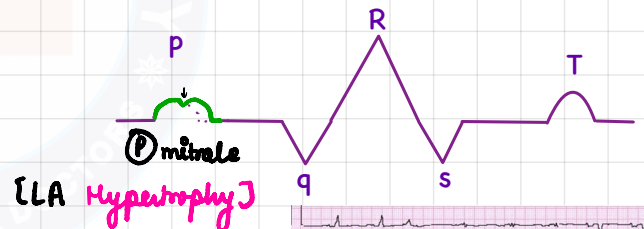
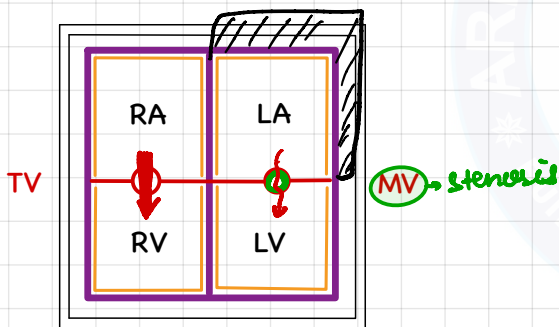
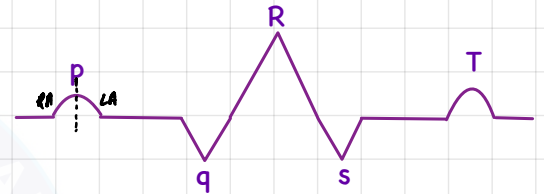


Axis

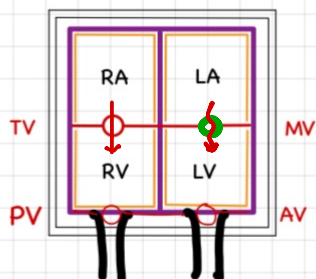
↳ LAD



p wave → atrial depolarisation



If tall P wave — without RA — c/a → Pseudo-P-pulmonale
 Hypertrophy
 seen in Hypokalemia
 [K⁺ < 3.3]

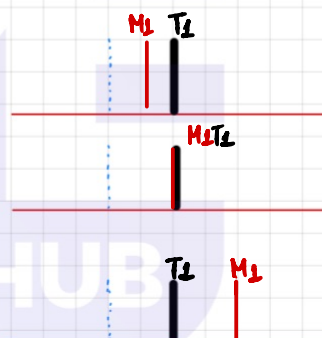


Closure of MV + Closure of TV - (S_1) (normal)

In mitral stenosis -

S1 can be

- narrow
- single
- Reverse split

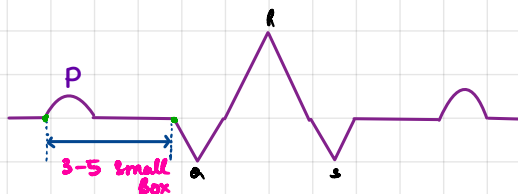


Extra point -

LOUD S1 is seen in - anemia / pregnancy / MS

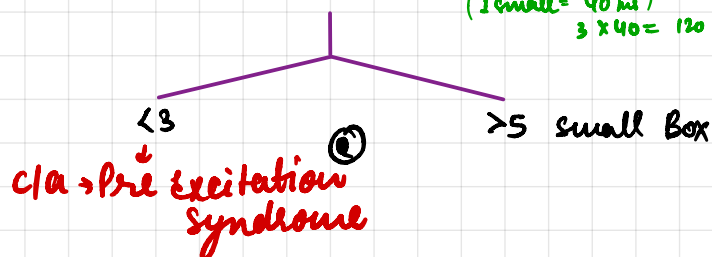
SOFT S1 is seen in - MR / severe calcified MS

PR INTERVAL

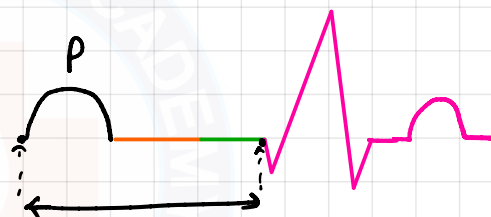
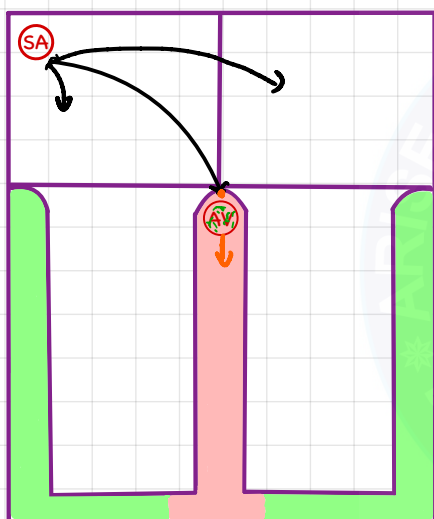


Normal = 3-5 small box [120-200ms]

(1 small = 40ms)
3 x 40 = 120



PR interval = > 5 small box $\xrightarrow{\text{SLO}}$ Defect in (AV) node $\rightarrow$ Heart Block

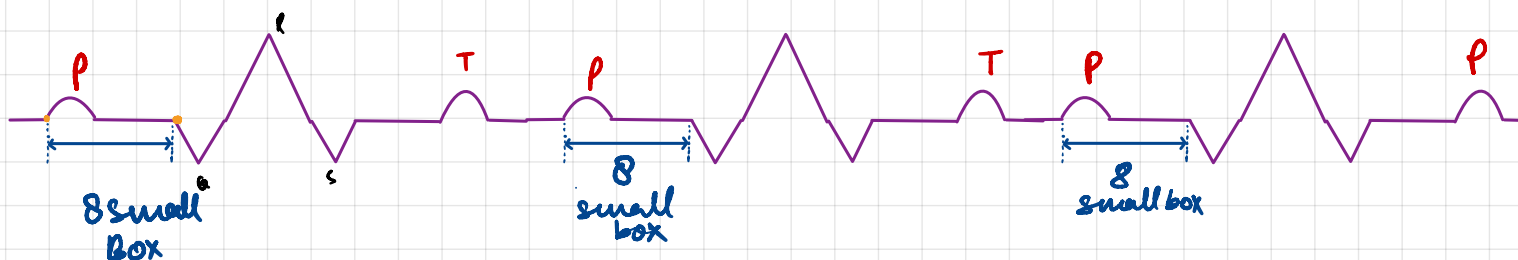


Heart Block $\rightarrow$ $\begin{cases} 1^\circ \\ 2^\circ \\ 3^\circ \end{cases} \rightarrow \begin{cases} Mobitz I \\ Mobitz II \end{cases}$

Etiology — RF, Hypothyroidism, Sarcoidosis,
Lyme disease, Toxoplasma,
Hemochromatosis

1st DEGREE HEART BLOCK

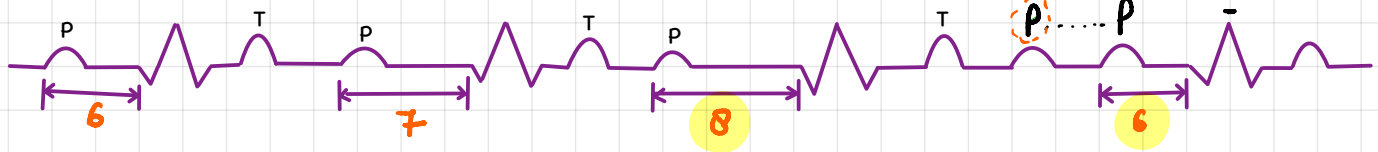
$\rightarrow$ PR-Interval $\rightarrow$ Fixed Prolonged PR interval





IIInd DEGREE → **MOBITZ I** or Wenkebach Phenomenon

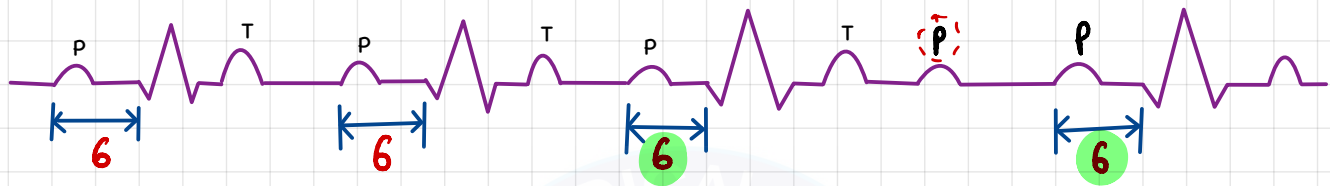
drop beat



PR-interval = Progressive prolongation of PR interval
F/B drop beat.

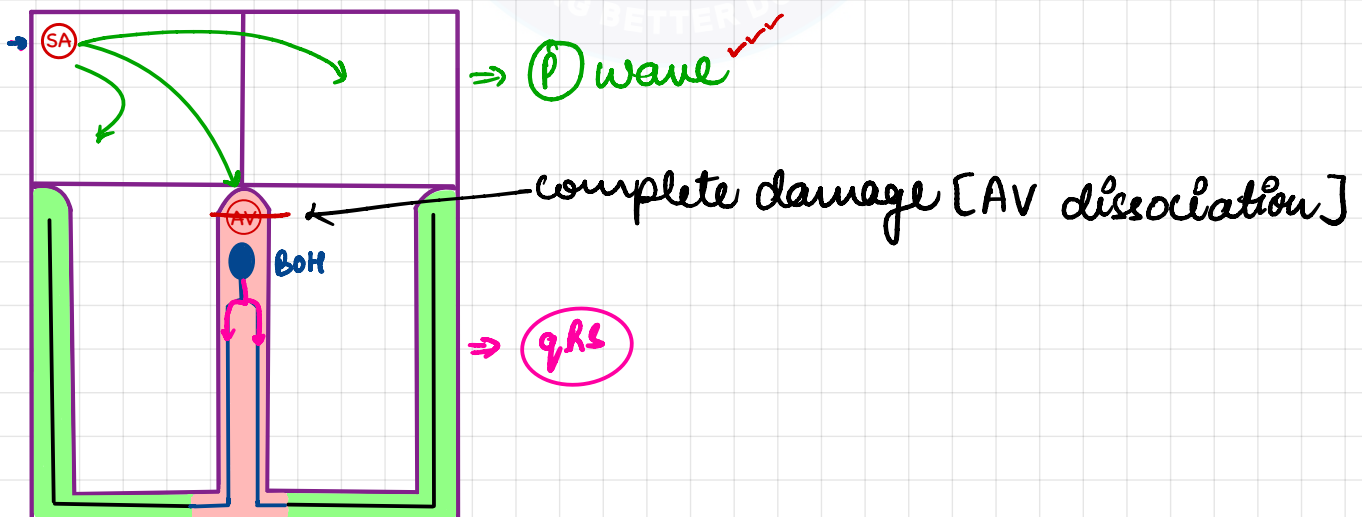
IIInd DEGREE → **MOBITZ II**

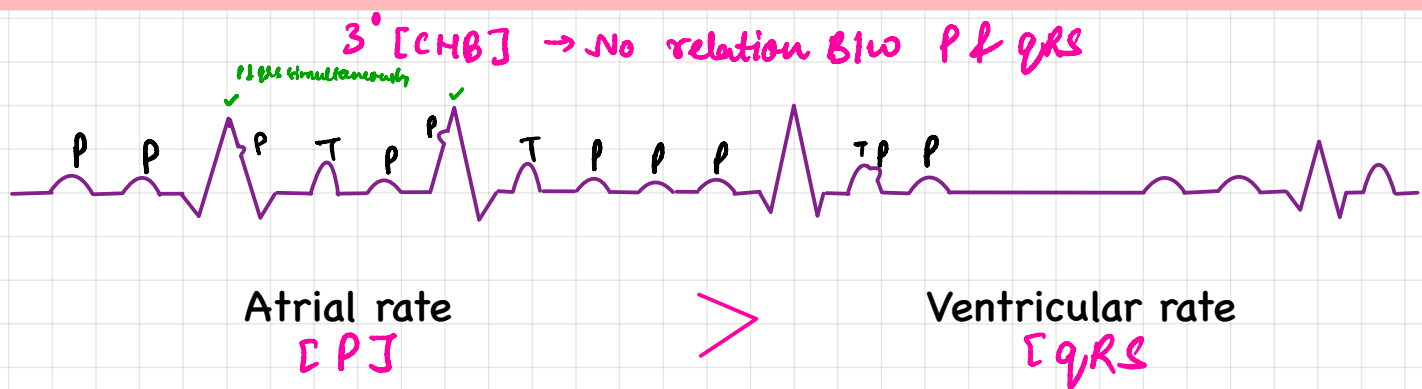
drop beat



PR - Interval = fixed PR interval F/B → drop beat

IIIrd DEGREE [COMPLETE HEART BLOCK]





Clf → Recurrent syncope episode → Clx → stroke adam lym.
JVP → Cannon @ wave

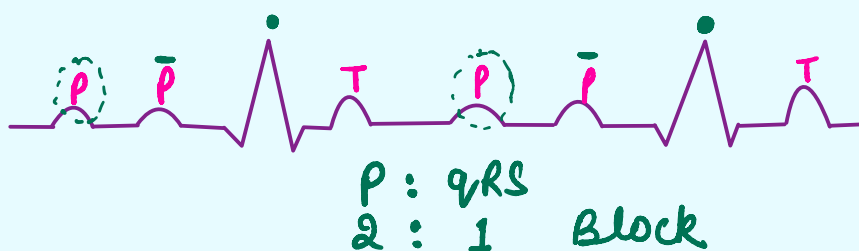


SUMMARY OF HEART BLOCK

	PR Interval	Drop Beat	Rx
1st Degree	Fixed Prolonged	NO	wait & watch
Mobitz I 2°	Progressive ↑ in PR interval	Present	wait & watch
Mobitz II	Fixed PR interval	Present	Pacemaker
3rd Degree	NO Relation b/w P & QRS		Pacemaker



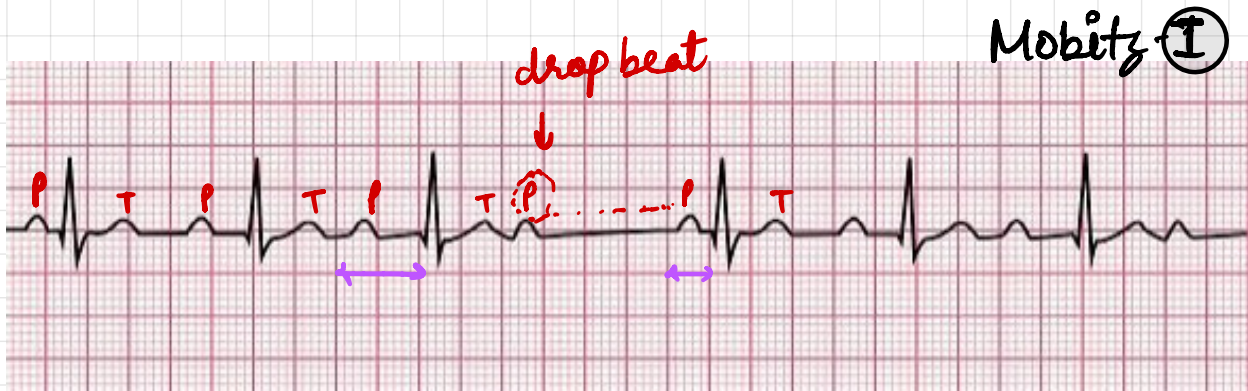
EXTRA POINT



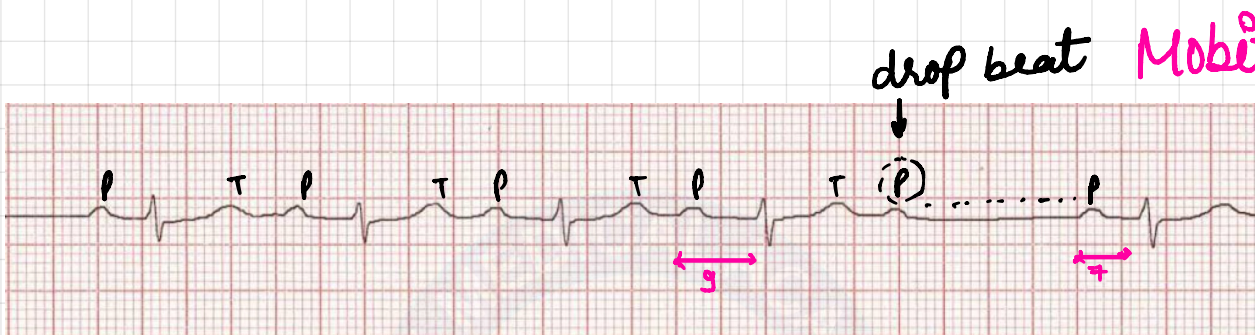
Pharma Pearl :- don't give metoprolol with verapamil as that increase risk of AV node Block hence leads to significant → Bradycardia.

LET'S PRACTICE → 1°/MOBITZ I/MOBITZ II/3° / 2:1

Q1)



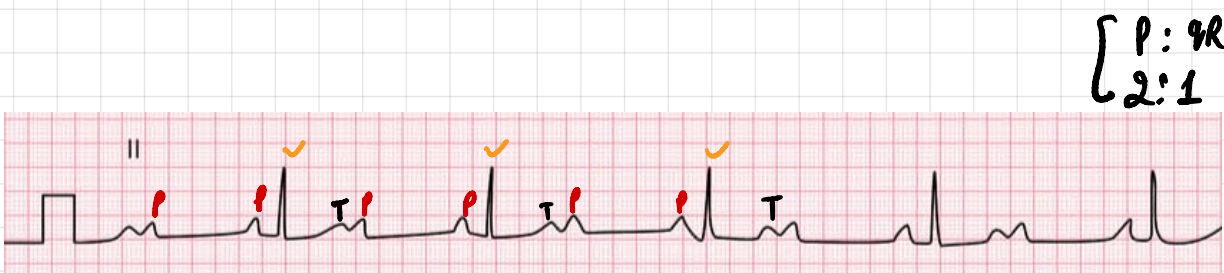
Q2)



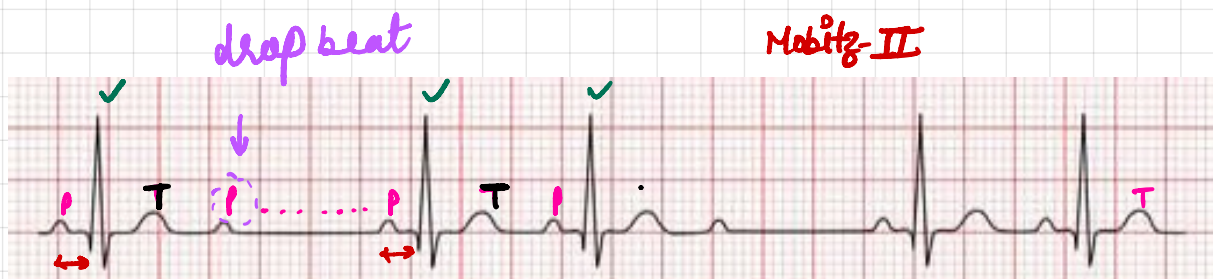
Q3)



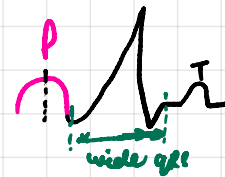
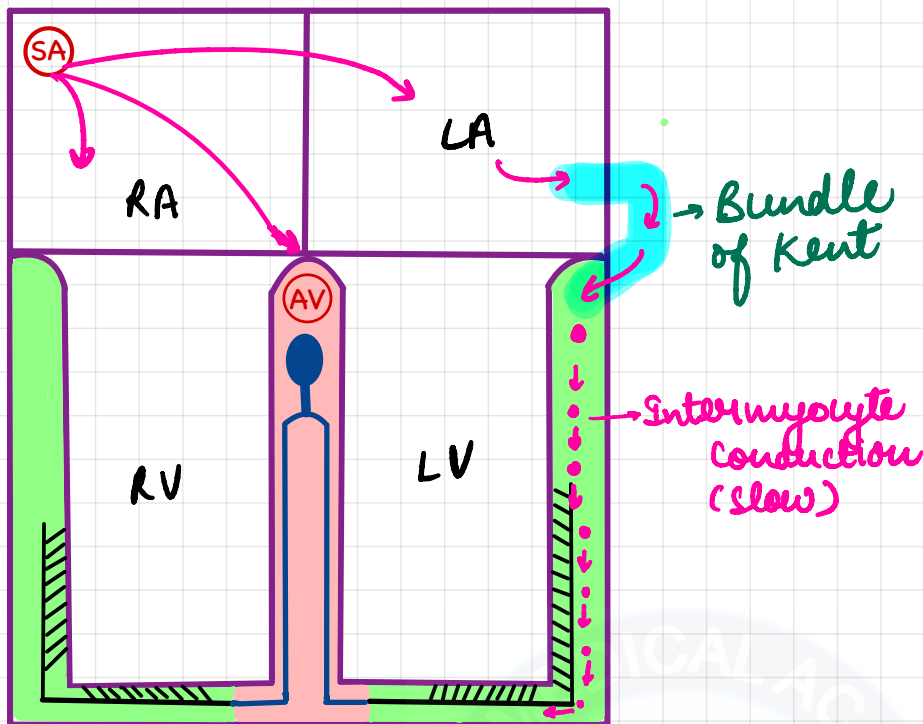
Q4)



Q5)



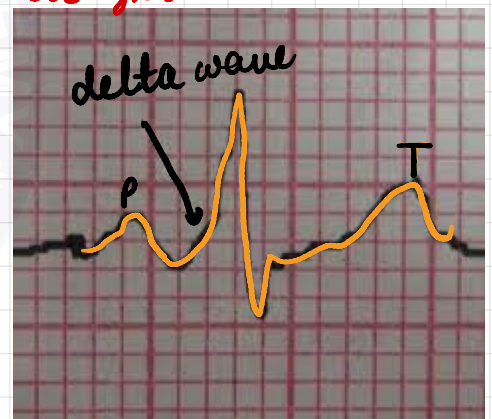
SHORT PR INTERVAL [$< \underline{120}$ mSec]
[< 3 small box]



Take More time to depolarise Ventricle → wide QRS

Q) Wolf - Parkinson white syn / Pre-excitation syn

- So, →
- absent Q wave
 - wide QRS
 - delta wave
 - Short PR interval



WPW Synd — Between

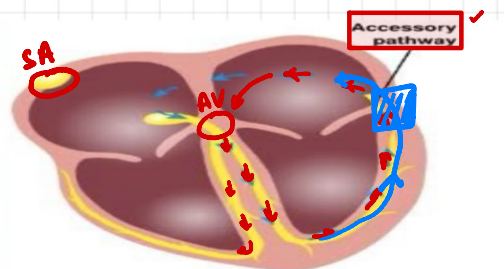
- LA → LV → Type A WPW syn. → MC
- RA → RV → Type B WPW syn.

Accessory Pathway

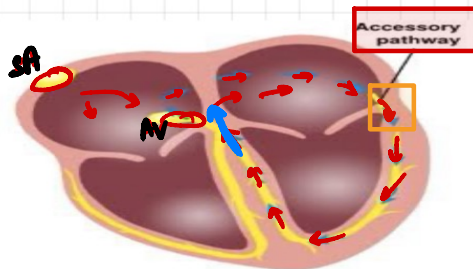
Rx → Procainamide or Flecainide (slow the accessory pathway)

→ Radiofrequency ablation → S/E → Risk of AV nodal damage

* TMT in WPW syn is → avoided



Orthodromic Circular Tachycardia
in a patient with an accessory pathway



Antidromic Circular Tachycardia
in a patient with an accessory pathway



MEDUHUB

q wave — Normally should fit in 1 small box

↓
If bigger

↓
C/a — **Pathological Q wave**

↓
Seen in

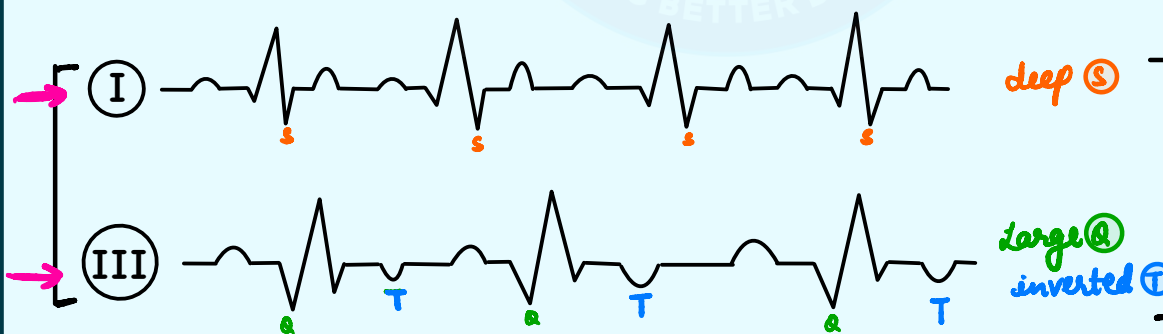
- **old MI**
- **HOCM**
- **pulmonary embolism**



EXTRA POINT

Pul. Embolism → Most common ECG change → **Sinus Tachycardia**

→ Most specific ECG change → **SI QIII TIII" pattern**



Extra point -

Mostly in fever - HR increases but if there is decrease in HR during fever called as - ...**Relative** bradycardia is seen in

Typhoid fever / ...**Coxiella Brunetti** "a fever" / ...**Chlamydia** infection / **Lyme disease**

QRS COMPLEX PATHOLOGY & ARRHYTHMIA

- A) Atrial Fibrillation [Af]
- B) Atrial Flutter [AF]
- C) Multifocal Atrial Tachycardia [MAT]
- D) Paroxysmal Supraventricular Tachycardia [PSVT]
- E) Ventricular Tachycardia [VT]
- F) Ventricular Flutter [VF]
- G) Ventricular Fibrillation [Vf]

SV Arrhythmia

V. Arrhythmia

A) Atrial Fibrillation

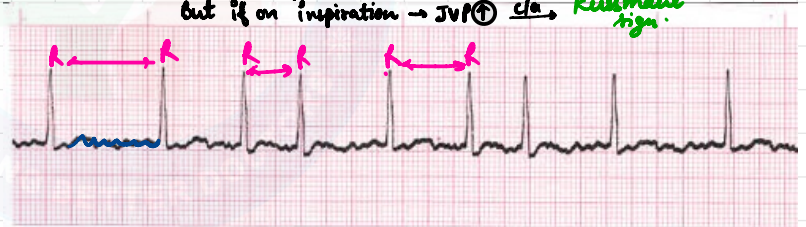


Pathology → Twitching of atria

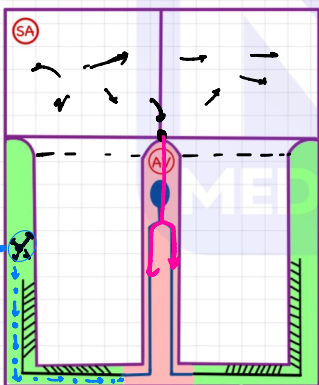
→ C/f → Syncope / Palpitation / asymptomatic
Can be seen in Hyperthyroidism / Hypoatremia / old age

Kussmaul sign seen
① Con inspiration fall in JVP
but if on inspiration → JVP ↑ → Kussmaul sign

ECG → lead → II
→ QRS → narrow
HR → >150/min



Rhythm → Irregularly - Irregular RR interval
P-wave → No identifiable P wave



If Arrhythmia originates from here calls as -
Supraventricular (SV) arrhythmia.

If Arrhythmia originates from here calls as -
Ventricular arrhythmia

Palpatory HR

auscultatory HR



In A. Fibrillation:-

Pulse deficit = Auscultatory HR - Palpatory HR

= >10

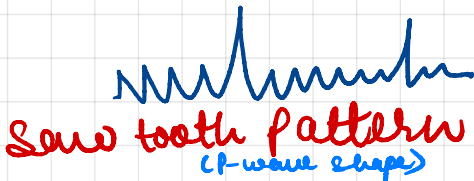
SV arrhythmia - narrow QRS

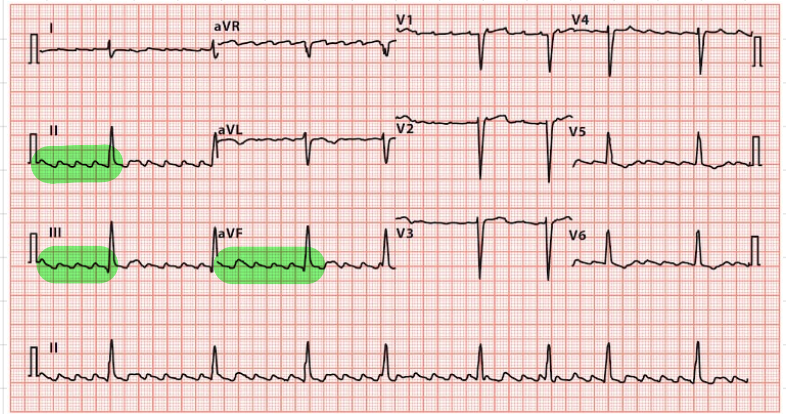
Ventricular arrhythmia - wide QRS

Rx - rate control - B-# → Esmolol
- rhythm control - Ibutilide / Amiodarone better
- if recurrent - then for prophylaxis
Amiodarone

DR. PRIYANSH

B) Atrial Flutter

→ ECG —

 Leads — II / III / aVF



C) MULTI-FOCAL-ATRIAL-TACHYCARDIA [MAT]

• Hint → COPD → Etophyllin → ① Risk of MAT

• ECG → Lead II
 > 3 morphology of P wave + Irregular Rhythm


• Pulse deficit → Present but less than ⑩

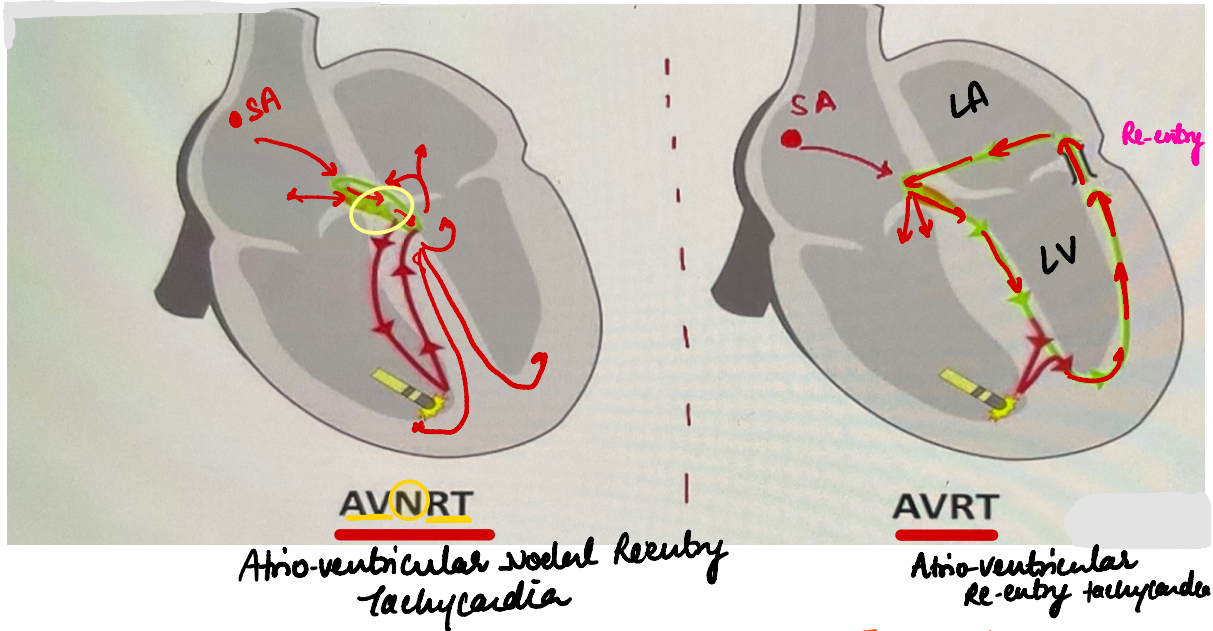
D) PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA [PSVT]

→ C/F → Young female → severe palpitation, impending doom *feeling like washe wali hai*

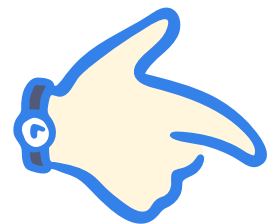
→ ECG → Lead → V2 / V3




 → Narrow QRS
 → HR > 150/min
 → P wave hidden behind QRS



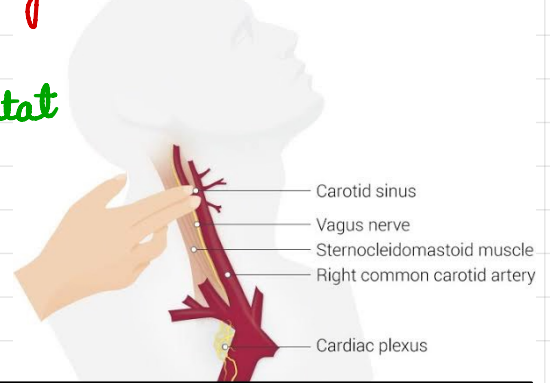
[PSVT = AVNRT = AVRT] → Trick.
to solve quest.



Rx — 1st line Carotid sinus massage / Vagal manueur / Valsalva

DOC :- inj. adenosine 6 mg IV stat

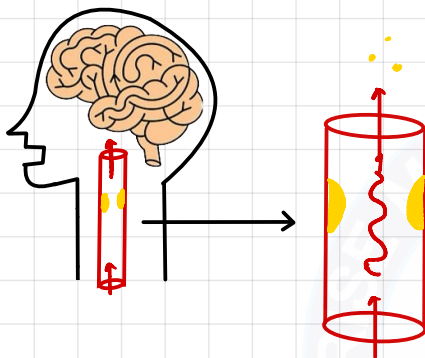
↓
12 mg
↓
12 mg



Other management :- face ice pack.

For Prophylaxis :- Diltiazem / Verapamil

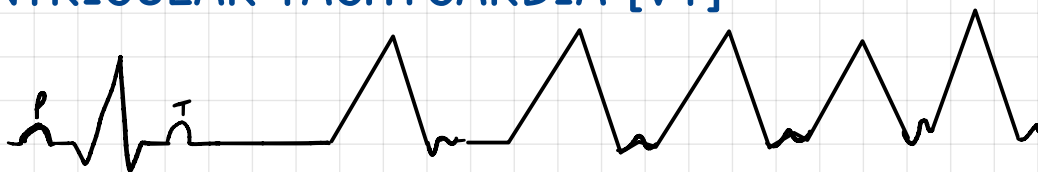
Before **CAROTID SINUS MASSAGE** → auscultation of carotid artery
if carotid Bruit ⊕
↓
sinus massage is C/I



Pharma connection

Adenosine is C/I in — Atrial fibrillation

D) VENTRICULAR TACHYCARDIA [VT]



• Bizarre wide QRS

or

→ ventricular premature complex (VPC)



(VPC) → or extrasystole

✓ VT = ≥ 3 continuous (VPC) + QRS > 100 ms + HR > 100/min.

If - ≥ 3 continuous (VPC) + QRS > 100 ms + HR < 100/min.

↓
c/a → idio-ventricular rhythm.

Ventricular tachycardia (VT)

Shape of VPC

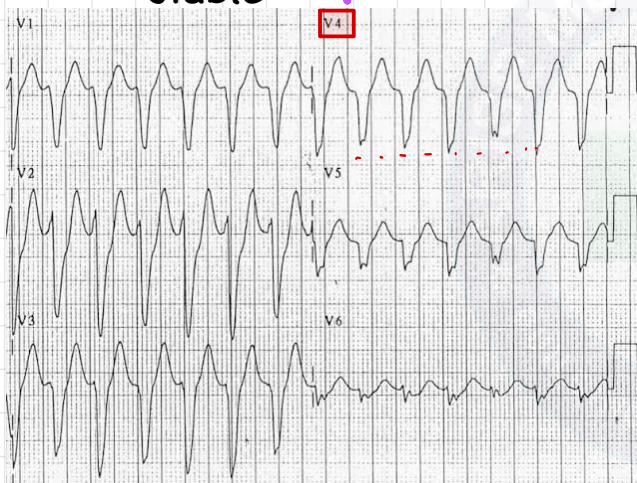
. Same

c/a

Mono-morphic VT

Rx - unstable [↓BP] → DC shock

- stable inj. Amiodarone



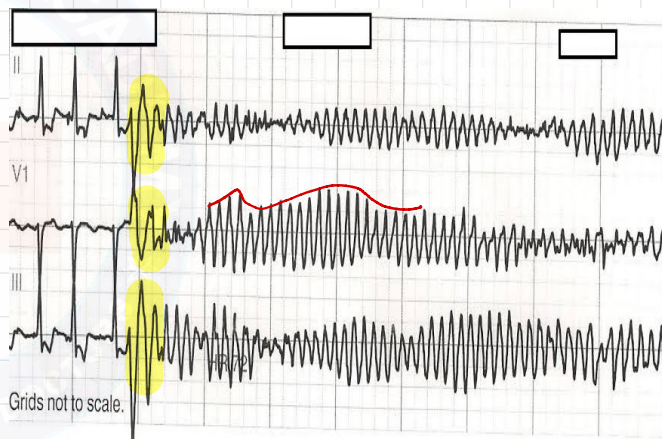
. different

c/a

Poly-morphic VT
or
Torsades-de-Pointes [TDP]

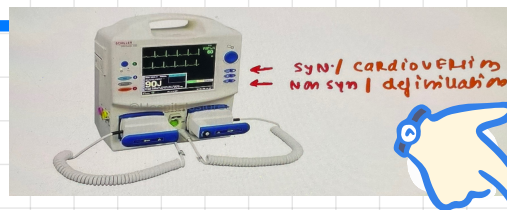
Rx - unstable [↓BP] → DC shock

- stable → inj MgSO4



Trigger of TDP — Hypomagnesaemia
[↑ QT interval]
Ⓚ on ⊕ phenomenon
early after depolarisation

Extra point



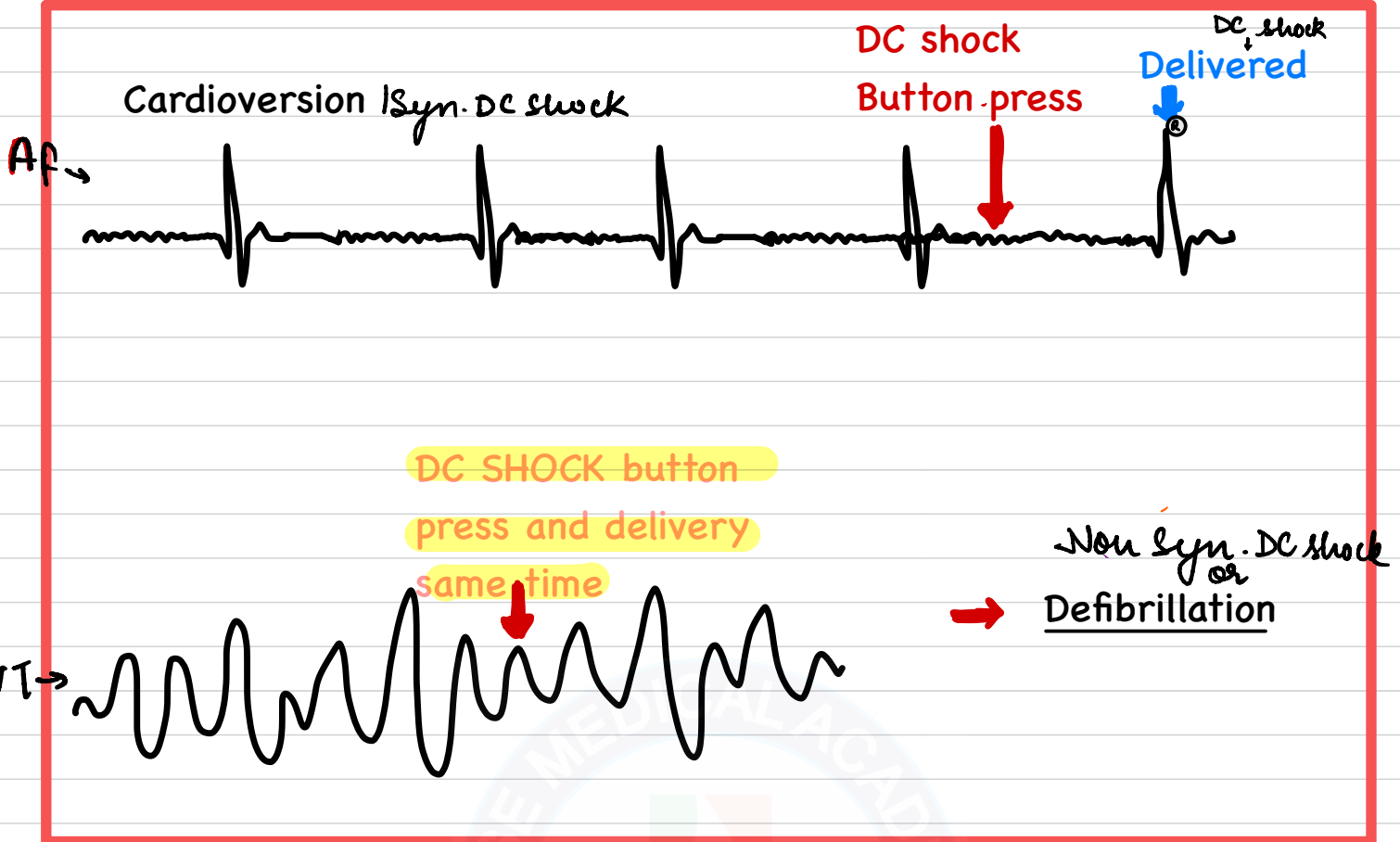
DC SHOCK

Synchronised /
cardioversion

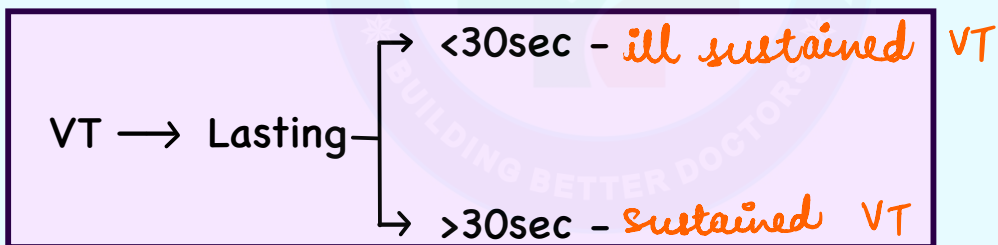
DC shock is sync
with R wave → used for
Supra-ventricular Arrhythmia

Non -Synchronised
/ defibrillation

DC shock is **NOT** sync
with R wave → used for
V. Arrhythmia

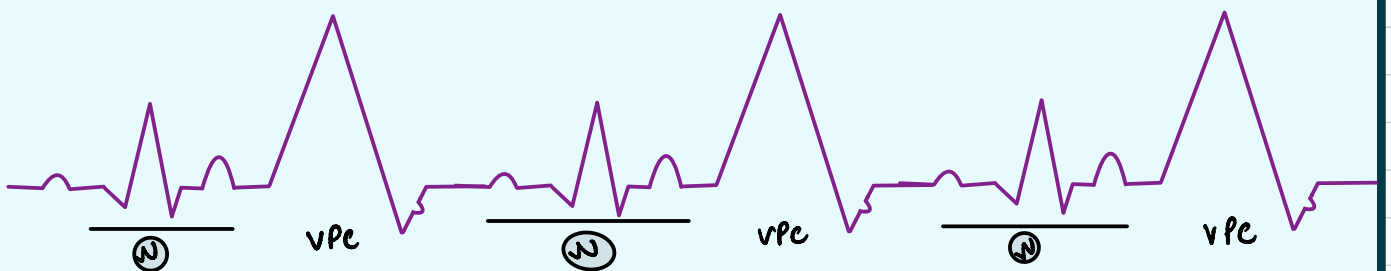


EXTRA POINT



Any Arrhythmia → leading to Hypotension — Rx → DC shock → CPR → Repeat DC shock

A flowchart showing the treatment for any arrhythmia leading to hypotension: 'Any Arrhythmia → leading to Hypotension — Rx → DC shock → CPR → Repeat DC shock'. The text is written in black, orange, and green.



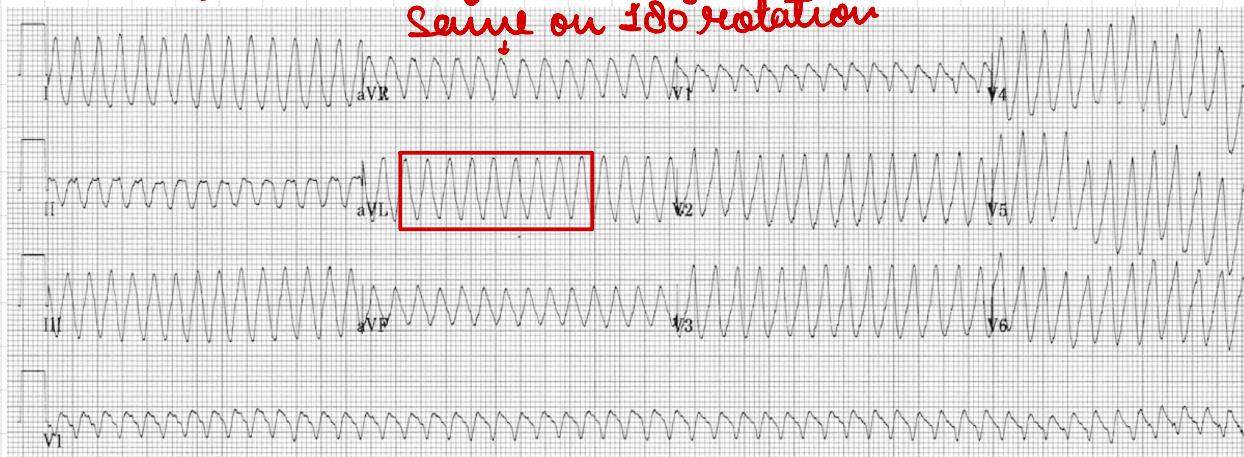
Ventricular Bigeminy → Can be seen in Digoxin toxicity

for the treatment of digoxine induced arrhythmia → *inj Lignocaine*

F) VENTRICULAR FLUTTER [VF] → Extreme form of V. Tachycardia

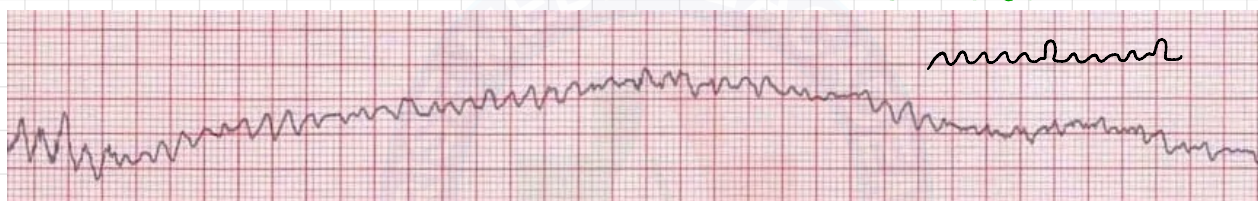
Rx → Same as VT

Same on 180° rotation



G) VENTRICULAR FIBRILLATION [VF] → Random undulation from base line.

Tab death kama wali hote hai use
cuch run pehle hie obkloga



after 1 min

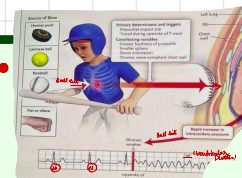
asystole
↓
Don't give
DC shock

for management of asystole → inj Adrenaline



* Blunt Trauma to chest leading to arrhythmia - c/a → Commotio - Cordis

SUMMARY OF ARRYTHMIA



A. Fibrillation → II lead.

↳ IR - IR R-R interval + No Identifiable P wave.



A. Flutter → II/III/aVF leads → sawtooth pattern



PSVT → V2/V3 lead →



↳ Rx → Ist - Vagal manueure

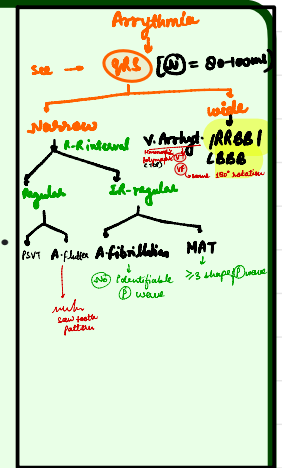
narrow QRS + P wave hidden behind QRS

↳ IInd - DOC → inj adenosine

🍓 **MAT** → lead II → $\geq$ 3 morphology of P wave.
[COPD → ectophyllin]

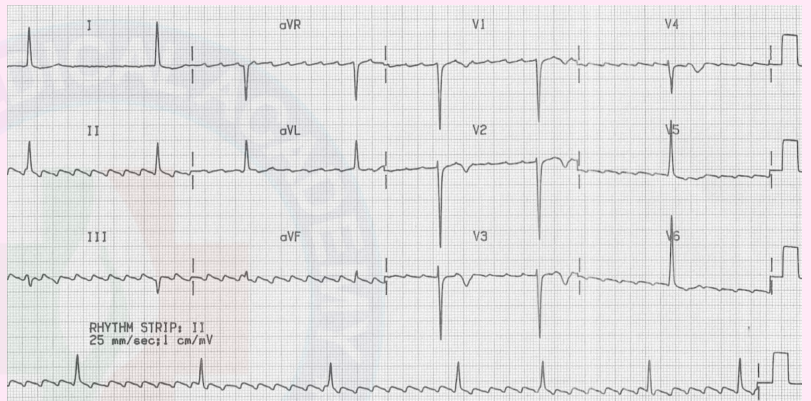
 VT $\rightarrow \geq \underline{3}$ VPC + qRS > 100 mS + HR > 100 /min.

180° Rotation same → Ventricular Flutter



LET'S PRACTICE

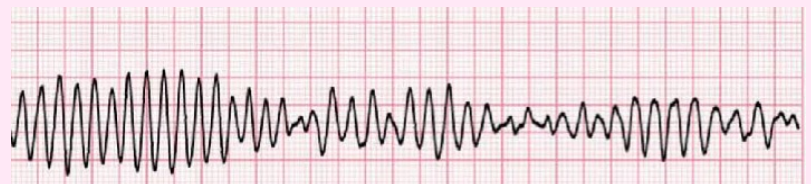
- A. Fibrillation
- A. Flutter
- PSVT
- VT



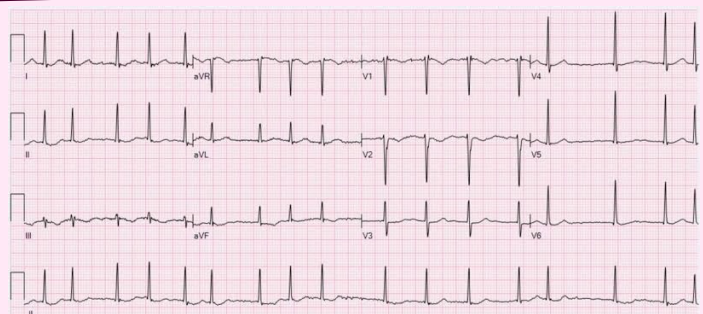
- A. Fibrillation
- MAT
- PSVT
- VT



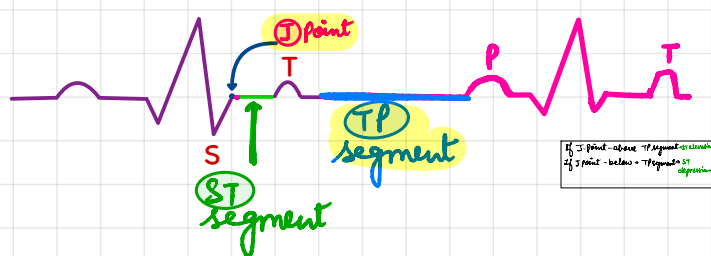
- A. Fibrillation
- Monomorphic VT
- PSVT
- TDP



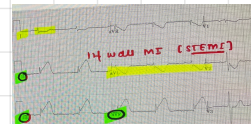
- A. Fibrillation
- MAT
- PSVT
- VT



ST - SEGMENT

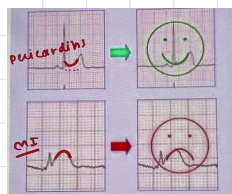


MI → ST-elevation in particular groups of leads.

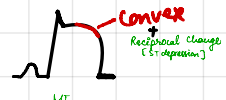


ST Elevation

1) STEMI

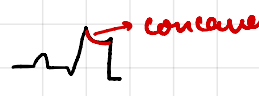


- V1-V4 → anterior wall MI
- I/aVL/V5-V6 → lateral wall MI
- II/III/aVF → inferior wall MI



→ 2) Acute pericarditis → diffuse

→ if ST-elev. in all leads



→ 3) Prinzmetal angina / vasospastic angina

→ 4) stress CMP → 'Takotsubo'

→ 5) LV aneurysm [is post MI complication]

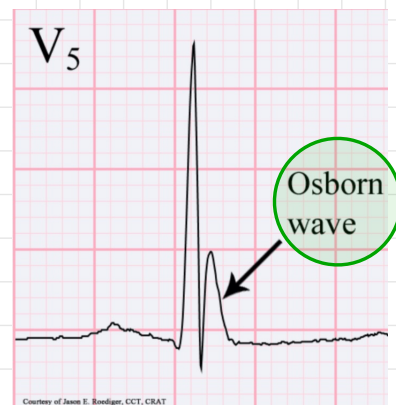
→ 6) ARVD → epsilon wave

→ 7) Brugada syndrome → asymp. ST-elevation → V1 & V2

→ 8) Hypothermia → Osborn wave (J-wave)

→ 9) Hyper

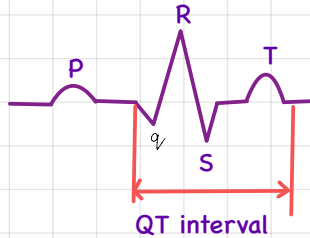
Osborn wave (J wave) can also be seen in hypercalcemia



ST Depression

- Hypokalemia
- Cocaine Toxicity
- Chronic stable angina → TMT ⊕
- NSTEMI [+/-]

QT INTERVAL



⑧ QT → 9-11 small box
= 360 - 440 ms

Prolong QT can lead to → ① Risk of V. Arrhythmia

Congenital prolong QT interval

- with deafness → Jarwell - Lange - Nelson Syn.
- without deafness → Romano - ward Syn.

$$\uparrow \text{QT interval} \propto \frac{1}{\text{S. Ca}^{+2}} \downarrow$$

$$\text{QT interval} \propto \frac{1}{\text{Mg}^{+2}}$$

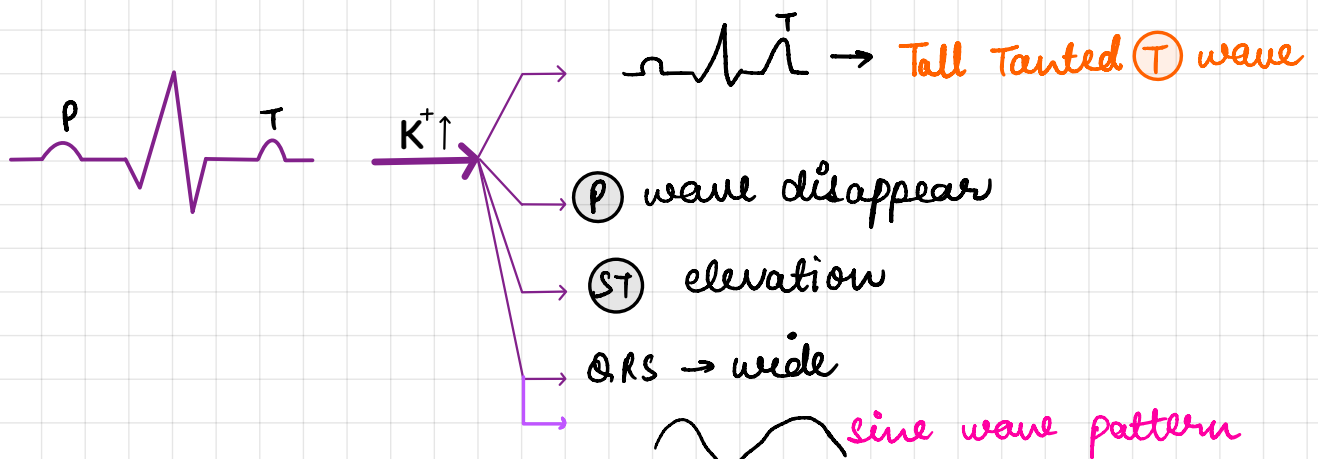


EXTRA POINT

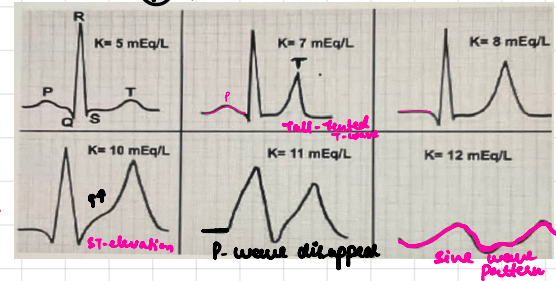
Corrected QT interval [QTc] → =

$$\frac{\text{QT interval}}{\sqrt{\text{R-R interval}}}$$

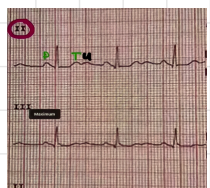
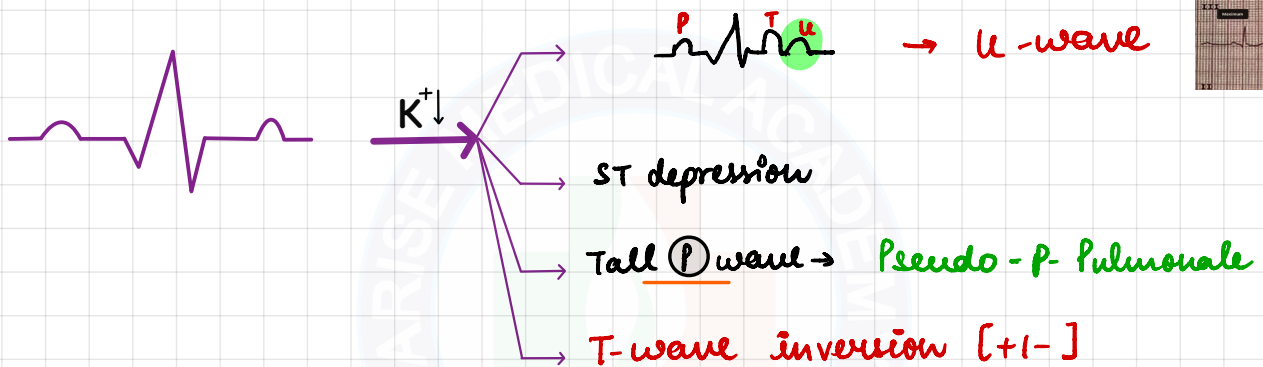
v. v. imp

ECG CHANGES OF HYPERKALEMIA [$\text{K}^+ > 5.5$ mEq]

- Risk factor for Hyperkalemia $\rightarrow$ $\downarrow$ urine output $\rightarrow$ $K^+ \uparrow \rightarrow$ v. arrhythmia
 $[ACE \#]$ $\rightarrow$ $\uparrow K^+$ (lungs) $\rightarrow$ $H^+ \uparrow \rightarrow$ metabolic acidosis
- Rx of Hyperkalemia $\rightarrow$
 - $\uparrow K^+$ $\rightarrow$ To stabilize cardiomyocyte $\rightarrow$ inj Ca²⁺ gluconate (calcium)
 - $\rightarrow$ inj. of **insuline** + dextrose
fastest way to $\downarrow K^+$
 - $\rightarrow$ Nebulization $\bar{c}$ Salbutamol $\rightarrow$ inj Furosemide
 - $\rightarrow$ K^+ binder $\rightarrow$ eg:- Na polystyrene

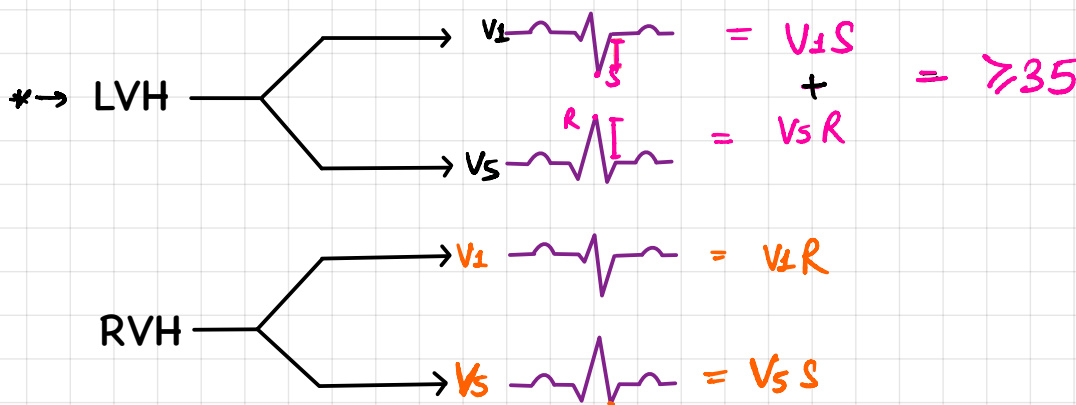


ECG CHANGES OF HYPOKALEMIA [$K^+ < 3.3$ mEq]

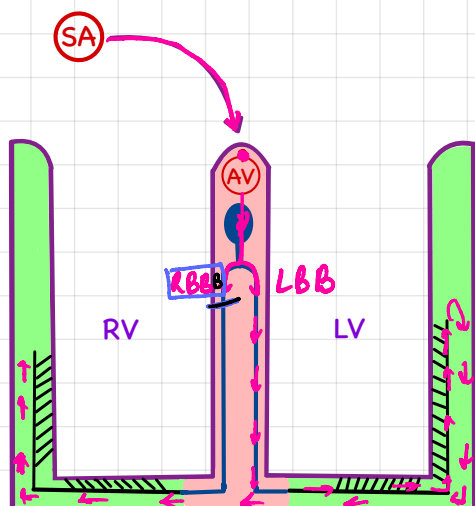


- Risk factor of $K^+ \downarrow \rightarrow$ diuretics [chronic use of diuretics $\rightarrow$ Furosemide]
- Rx $\rightarrow$ inj KCl.
- U wave $\rightarrow$ delayed repolarisation of papillary muscle.

* CHAMBER HYPERTROPHY



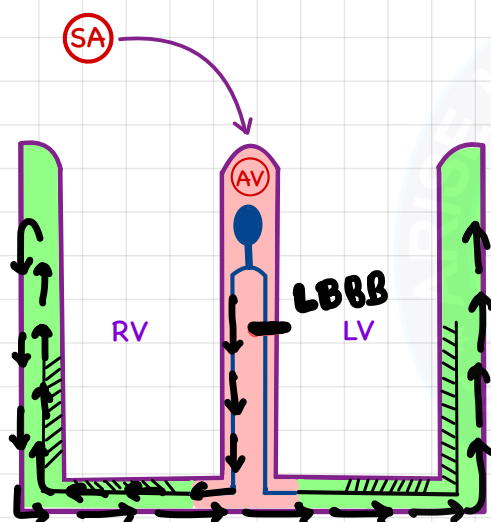
BUNDLE BRANCH BLOCK [BBB]



RBBB
light bundle
Branch Block

→ qRS → wide

→ V1 → 
rRS pattern / Rabbit
ear pattern



LBBB

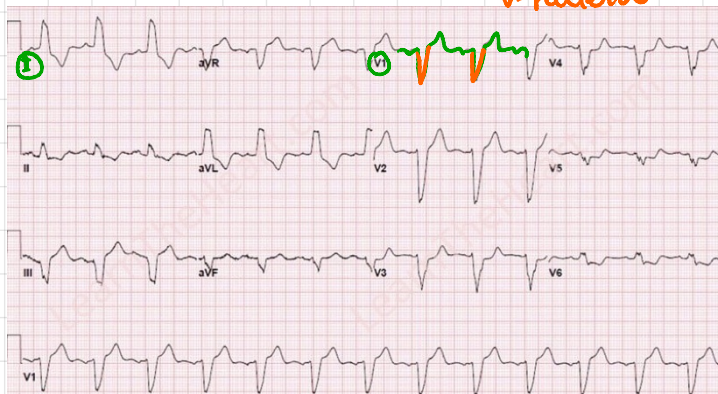
→ qRS → wide

→ V1 → 
V-pattern

Rx - if symptomatic → Cardiac Resynchronization Therapy

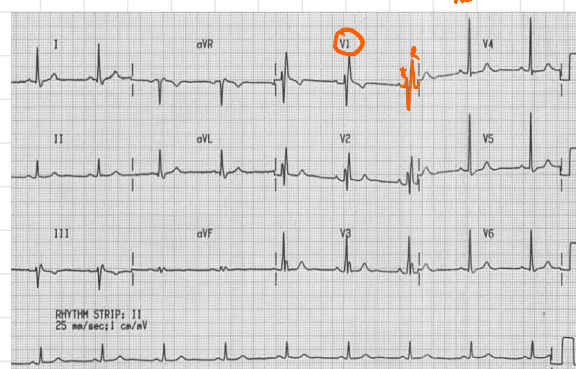
a) RBBB

b) LBBB
v-pattern

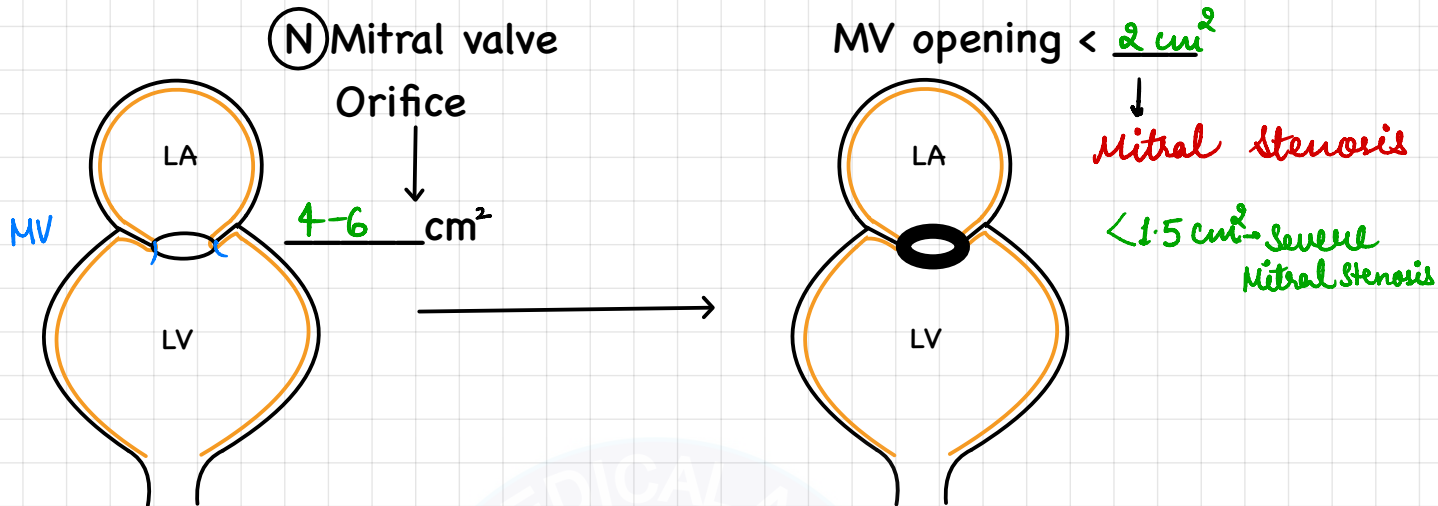


a) RBBB

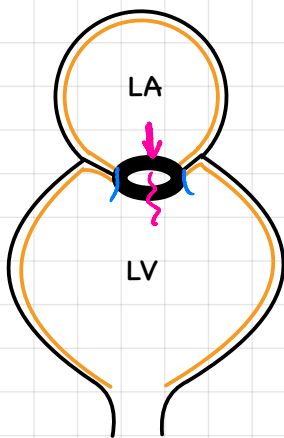
b) LBBB
rRS



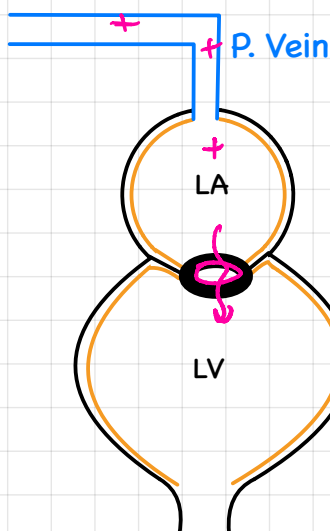
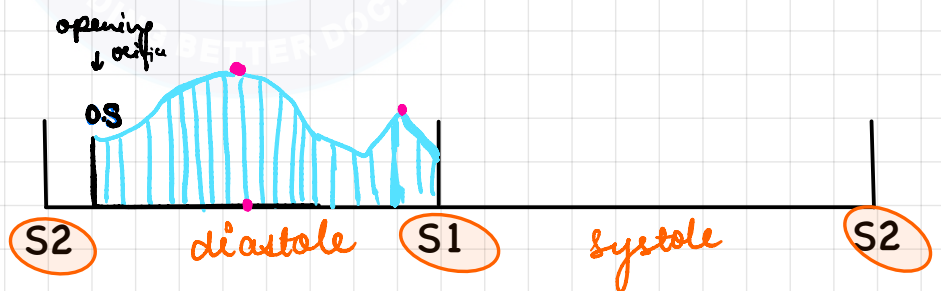
CHAPTER 10 MITRAL STENOSIS



So, during diastole — opening of pathological mitral valve → opening snap.

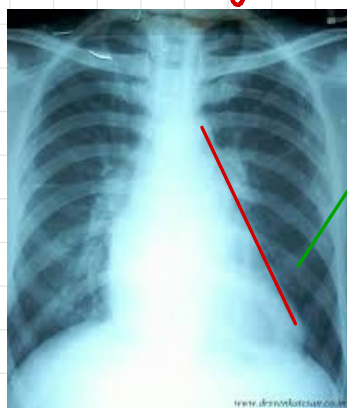


MS → mid-diastolic murmur & presystolic accentuation. (MSD)



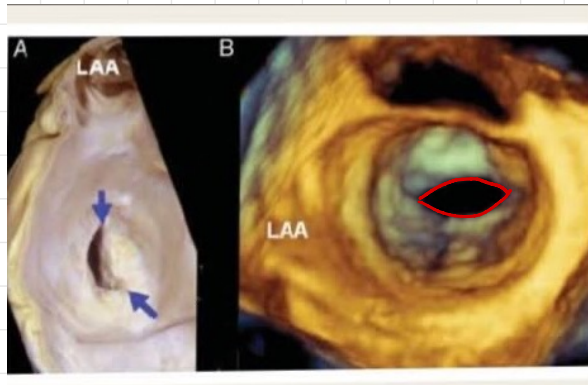
- L.A. → Hypertrophy → ECG → P mitrale.
- P. Vein pressure → ↑
- if MS+ASD → Luntan Bacher syndrome
- S1 → Loud
- [if severe calcified MS → soft S2]

CxR — LA Hypertrophy

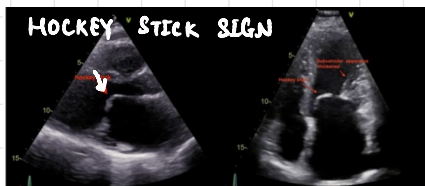


↓
Straight left heart border

Echo — Fish mouth opening of MV.

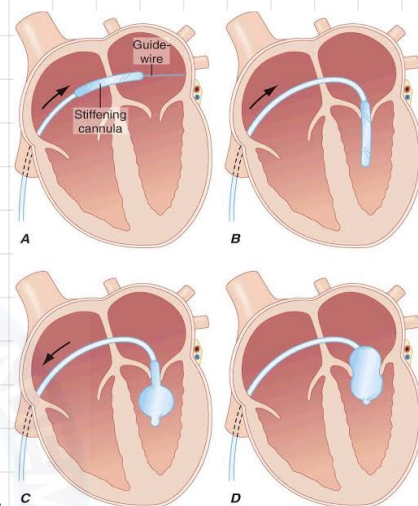


Rx — 1) Percutaneous MV Ballon Valvuloplasty [PMBV]



2) If severe calcified MS →

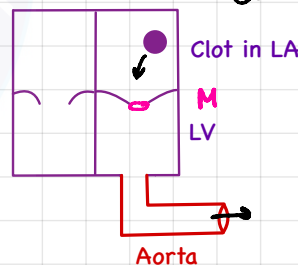
PMBV is CII
Rx → MV Replacement



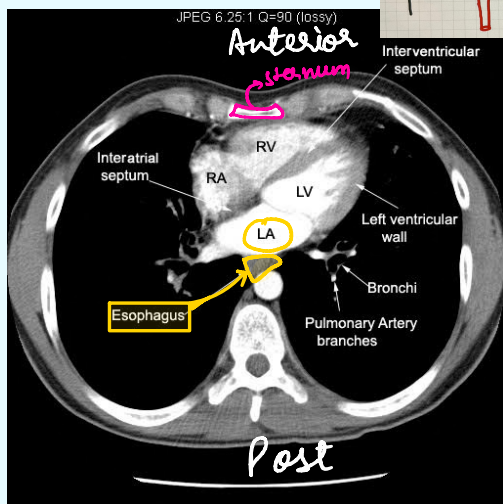
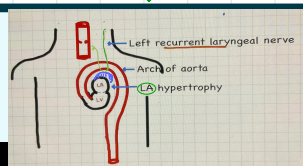
3) If MS is also c A fibrillation then to avoid risk of clot formation Warfarin should be added in treatment.

4) M. Stenosis + Clot in (LA)

→ Removal of clot
MV + Replacement



EXTRA POINT



So, (LA) enlargement can lead

Compression of

Esophageous
↓
dysphagia

① Recurrent laryngeal nerve
↓
Hoarseness of voice

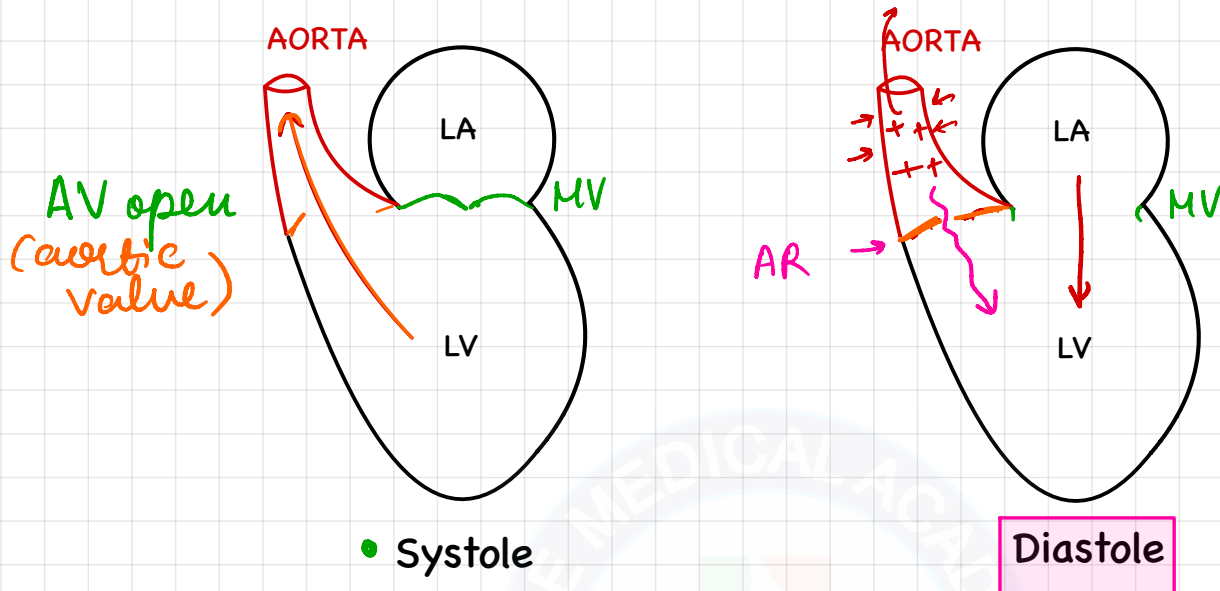
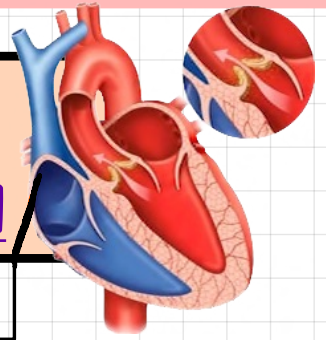
CA → ORTNER SYNDROME

Severity of MS depend on α length of murmur

CHAPTER 11

AORTIC REGURGITATION

Aortic Insufficiency



Etiology → RHD, Aortic dissection, Aortic aneurysm

C/F → ^{imp} Corrigan Pulse / Carotid dance / water Hammer Pulse

→ Head Bobbing → ^(Head continuously moving) de Musset sign

→ Movement in Uvula → Mallet sign

→ Murmur in Femoral Artery → ^Q Duroziez sign

→ Capillary pulsation in nail plate → ^Q Quincke sign

other sign is also seen in AR

lower limb BP is more than U.

Hill sign

* Pulsation in liver → Rosenbach sign

* Capillary pulsation in nail → Osler sign

* change in pupil size → Landolt sign

* Blushing & flushing on forehead → Light Head sign

Pulse Pressure = $SBP - DBP$ → High

Pulse bounding → Rapid runoff

Rx → Aortic valve replacement.

→ Vasodilator → Uses the peripheral resistance so backflow of the blood to LV less hence ↓↓↓ murmur of AR.

* Mild AR murmur is called as - seagull murmur / Austin Flint murmur /

cooing dove murmur

Regurgitant jet of blood is pushing (MV) leaflet

murmur across (MV) blood flow



EXTRA POINT

Aortic dissection

tear in aorta

CF → tearing type of chest pain

→ BP in both arms is different.

→ MRI

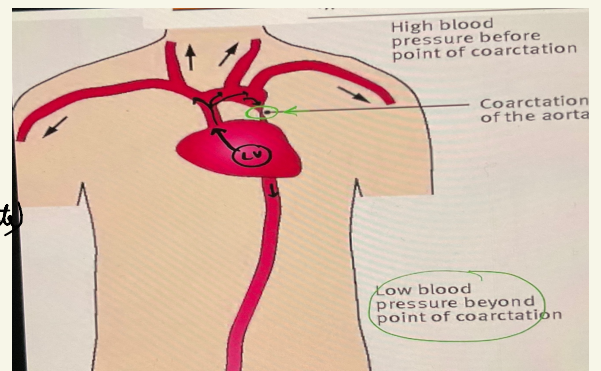
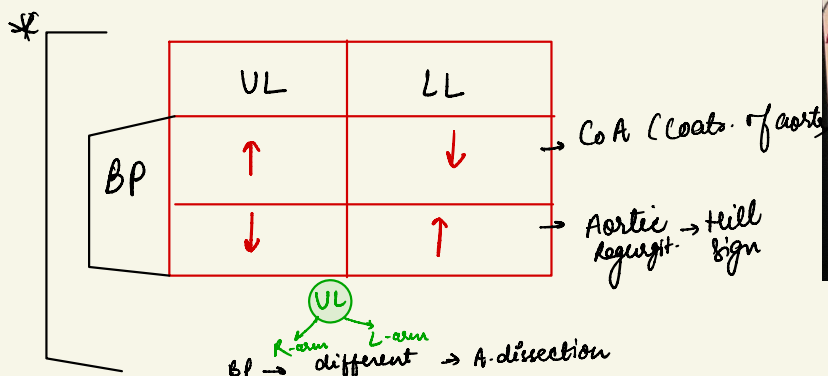
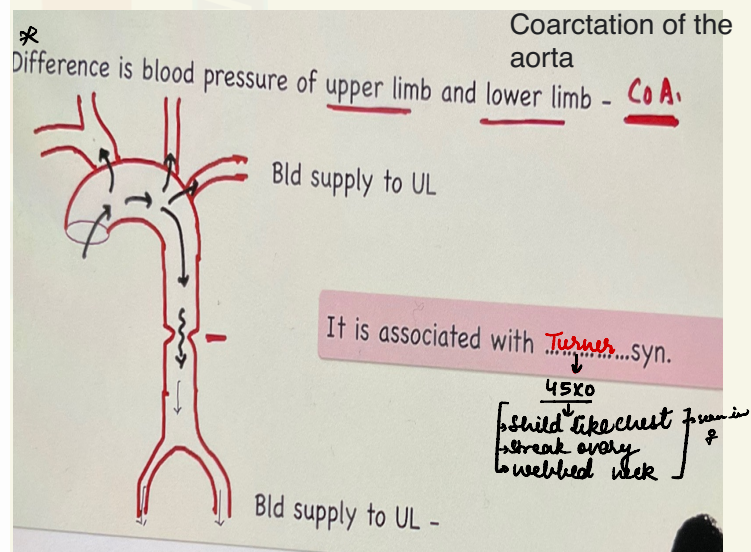
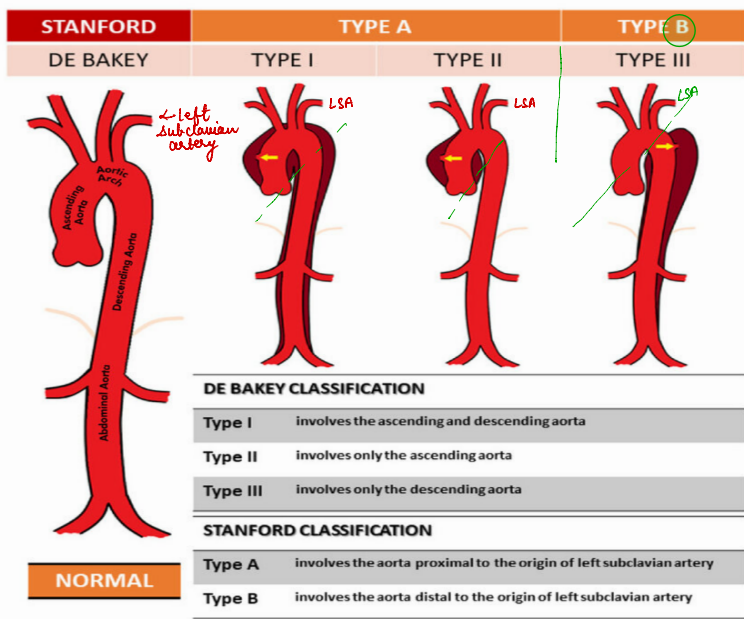
Tennis Ball appearance

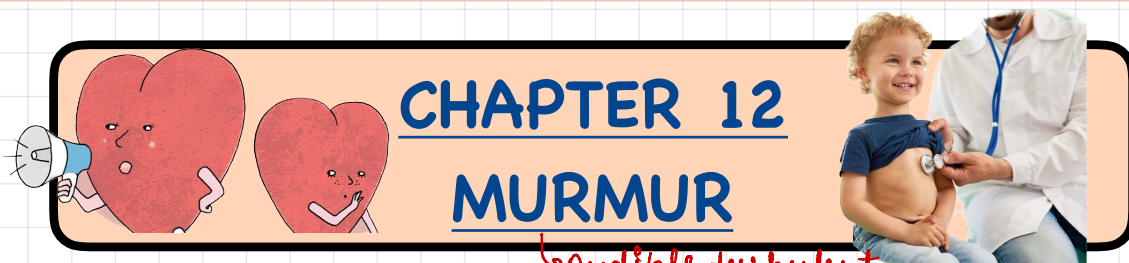


for Aortic dissection we have 2 classification

① DeBakey classification

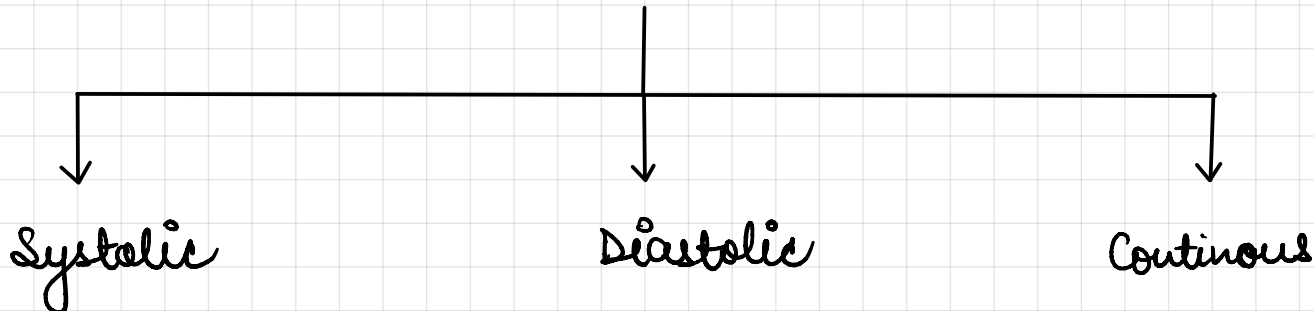
② CLASSIFICATION → STANFORD (A) & (B)



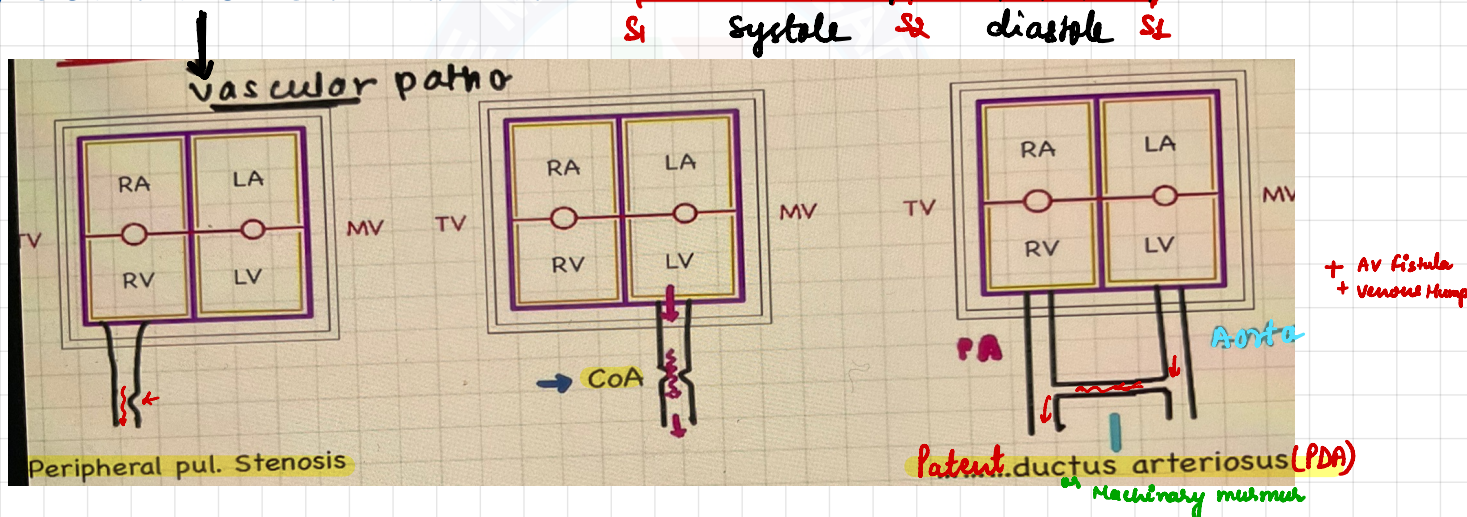
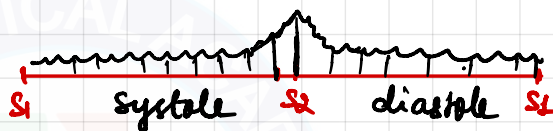


Audible turbulent blood flow

TYPES OF MURMUR



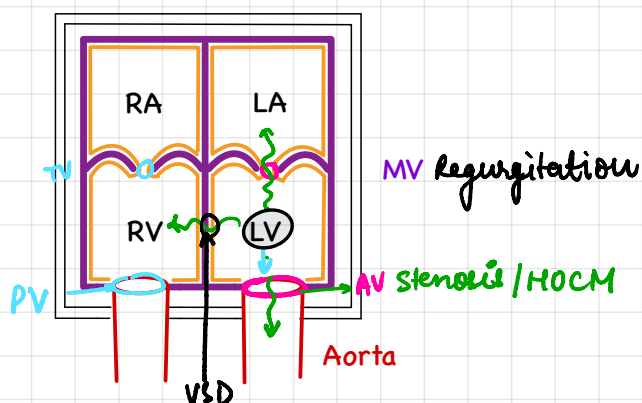
A) CONTINUOUS MURMUR



B) SYSTOLIC MURMUR

Causes →

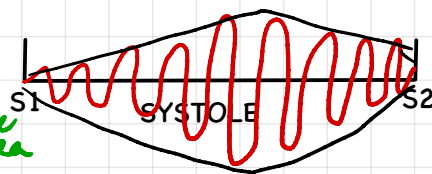
Systolic Left	Right
MR	TR
VSD	
AS	PS
HOCM	



→ Ejection systolic

crescendo-decrescendo
or
diamond shape murmur

AS → at aortic area
HOCM → at Erb's area



→ Pansystolic

↳ MR & VSD

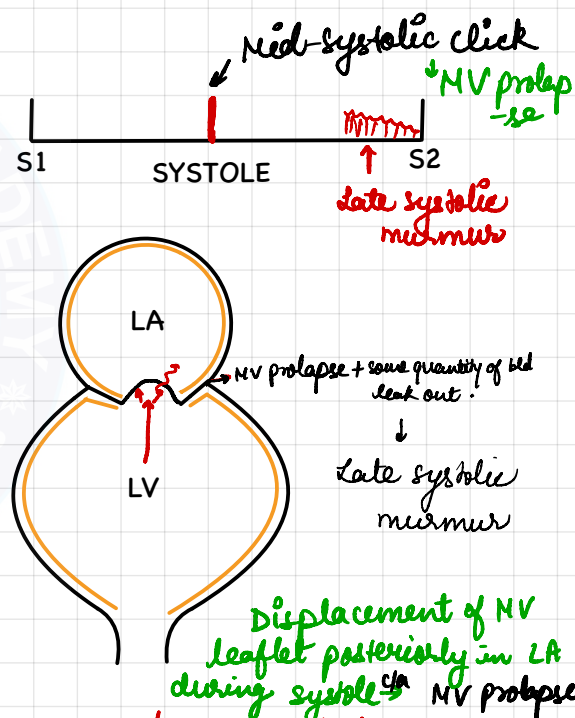
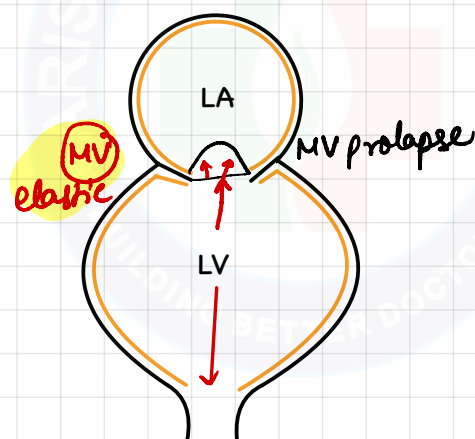
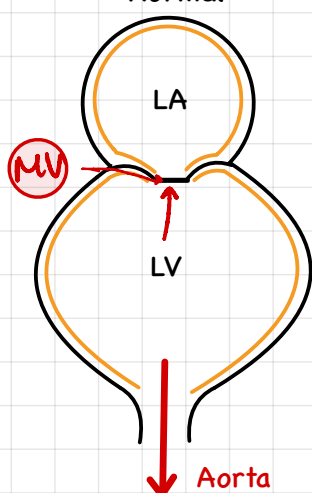
* MR murmur radiates towards - axilla

* Pansystolic murmur & high JVP → TR (Tricuspid regurgitation)



Late Systolic

Normal



Displacement of MV leaflet posteriorly in LA during systole → MV prolapse

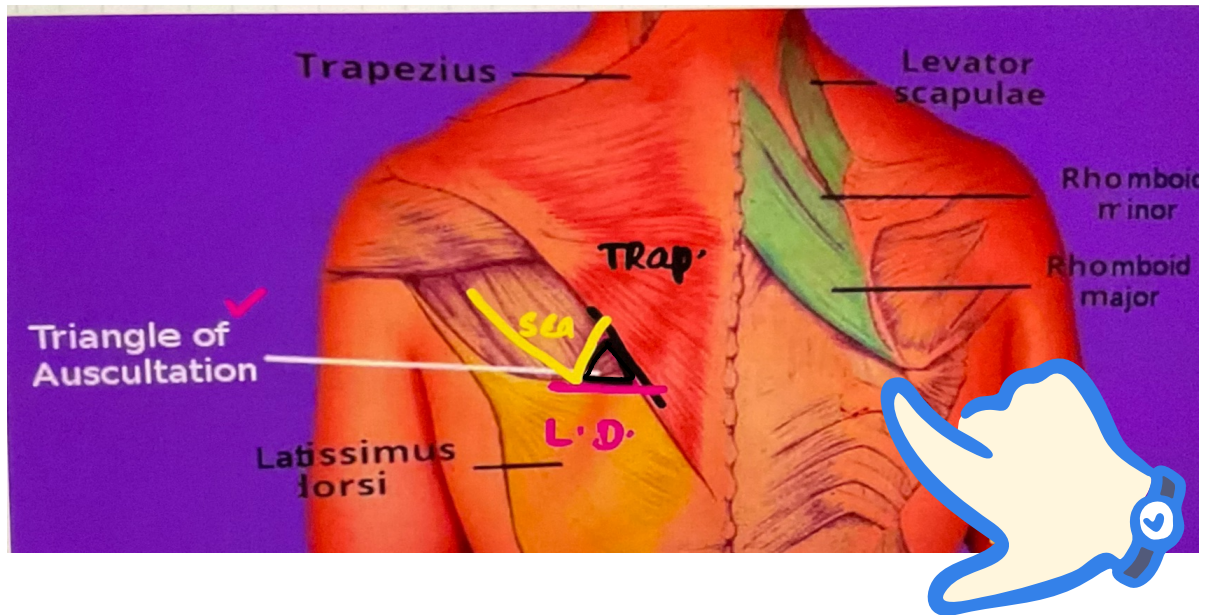
* MVP is a/w EHLer Danlos syn / Marfan syn / Straight back syn.
Contact sports should be avoided in severe MVP.

3) DIASTOLIC MURMUR

- Early → Mild aortic Regurgitation
- Mid-Diastolic → MS / TS
- Pan-Diastolic → AR

Mid diastolic murmur & prominent 'a' wave on JVP → TS

Triangle of auscultation →





EXTRA POINT

HOCM & Mitral valve prolapse murmur

on valsalva

NAMED MURMUR

- 👂 Carvallo Murmur → **TR**
- 👂 Carey Coomb Murmur → **RF** → *acute endocarditis*
- 👂 Austin Flint Murmur → **AR**
- 👂 Graham Steel Murmur → **PR** (*pulmonary Regurgitation*)
- 👂 AS Murmur Radiates towards mitral area [apex]
 - c/a → *gallopericardium* Phenomenon
- 👂 Tumor Plop Sound → **Atrial myx**

NAMED PULSES

- Anacrotic Pulse → **AS**
- OR
- P. Parvus et Tardus
- Dicrotic Pulse → **D** CMP
- P. BisFeriens → **HOCM**
- P. Alternance → **LVF**
- Corrigan / water hammer pulse → **AR**

THE CHAPTER YOU ARE LEARNING TODAY IS
GOING TO SAVE SOMEONE'S LIFE TOMORROW

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