MEDICINE 1
MINI-OSCE COLLECTED SLIDES
- PART 4
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Contents

Cardiology

Ischemic Heart Disease

Ischemic Heart Disease (IHD)

- Caused by atherosclerosis where O2 demand > O2 supply.
- Typical Symptoms: Patients present with Chest pain (Angina), Dyspnea and Diaphoresis.
- IHD is subdivided into Stable Angina & Acute Coronary Syndrome.
- Stable Angina: Normal ECG + Vessels are unable to dilate enough to allow adequate blood flow + Unruptured Plaque + Pain when demand increases / with exertion.
- Acute coronary syndrome is further classified into No ST elevation & ST elevation (STEMI).
- No ST elevation with normal Troponin → Unstable Angina: Ruptured plaque causes partial occlusion + Pain at rest + Subendocardial ischemia but no infarction + ECG either Normal, Inverted T wave, or ST depression.
- No ST elevation with raised Troponin → NSTEMI: Same as unstable angina but Subendocardial Infarction + ECG same as Unstable angina
- STEMI → Complete occlusion causing Transmural infarction + Raised Troponin + ECG shows hyperacute T wave / ST elevation

IHD

- CABG grafts → 1- Lt internal mammary artery. 2- Saphenous vein. 3- Radial artery.
- Revascularization indications → 1- Angina. 2- MI. 3- Systolic Dysfunction.
- Most common cause of Adult's sudden death → CAD (arrhythmia - plaque rupture).
- Most common cause of young Adult's sudden death → HOCM.
- Risk factors for IHD: 1- Prior CAD (major risk factor). 2- DM, PAD, CKD (major risk factors). 3- HTN, Dyslipidemia, Smoking, Obesity.
- For risk factors reduction → Aspirin + Statin + Beta Blocker (prior to infarction).

IHD

- Ischemic Pathological Changes on the Myocardium:

0 - 4 Hours: No changes.

4 - 12 Hours: Grossly → Mottled. / Microscopically → Necrosis, Edema.

12 - 24 Hours: Grossly → Hyperemia. / Microscopically → Infarction of Surrounding Tissue.

5 - 10 Days: Grossly → Central Yellow. / Microscopically → Granulation.

After 7 weeks: Grossly → Gray-white scar. / Microscopically → Scar.

- Complications of Ischemia:

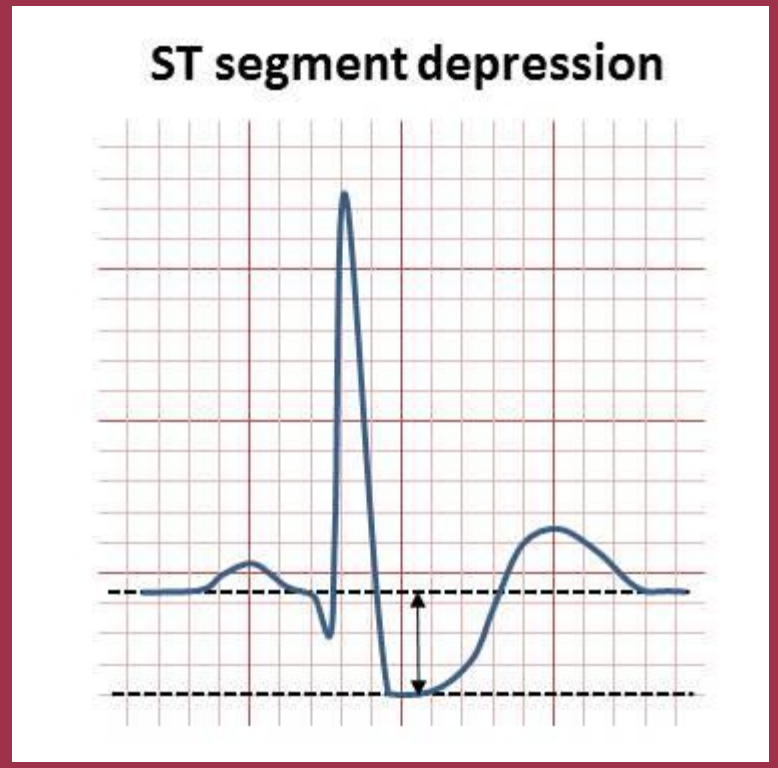
First 4 days → Arrhythmia.

5 - 10 days → Free wall rupture (leading to Tamponade) + Papillary muscle rupture (in inferior MI, may cause MR and HF) + VSD (causing RHF - elevated JVP, edema and Hypotension-) + Pericarditis.

weeks later → Dressler syndrome (Anterior infarction mediated Pericarditis) + Lt ventricular thrombus / CVA + Aneurysm (with Anterior MI) + Pseudoaneurysm (with Inferior MI)

Ischemic ECG Changes

ST Depression

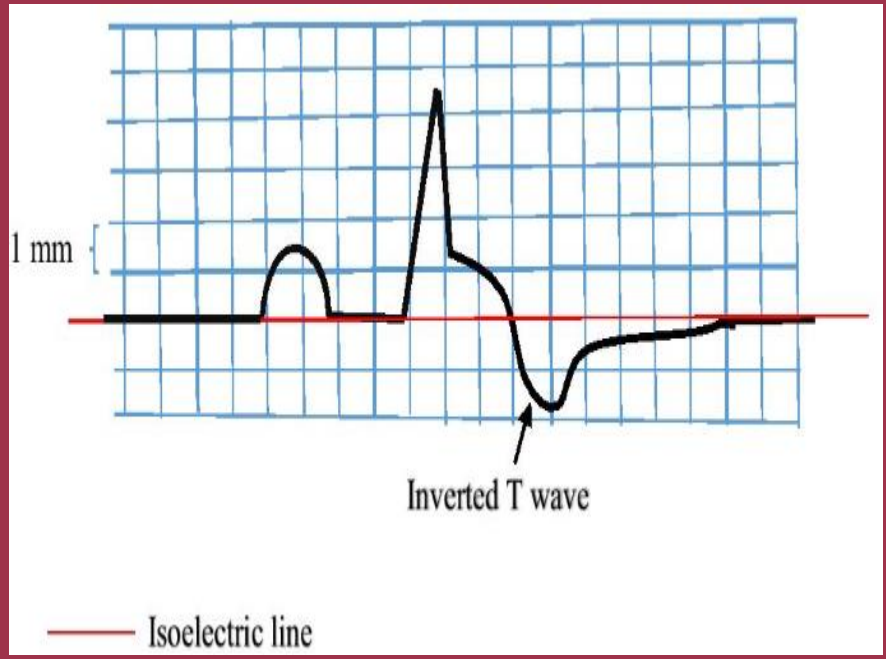


- Indicates Subendocardial Ischemia.
- Note that Ischemic part Depolarizes and Repolarizes First

Ischemic ECG Changes

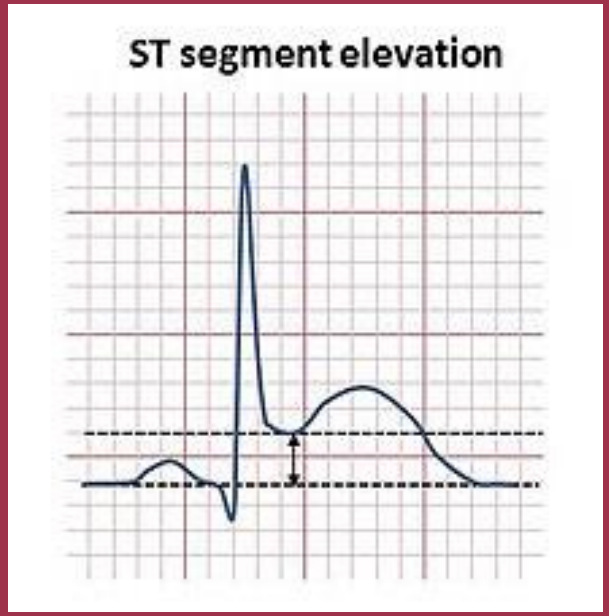
T wave Inversion

- Indicates Subendocardial Ischemia / Resolving Pericarditis / BBB / Ventricular Hypertrophy.
- Since Subendocardial Ischemic part depolarizes first away from the electrode.



Ischemic ECG Changes

ST Elevation

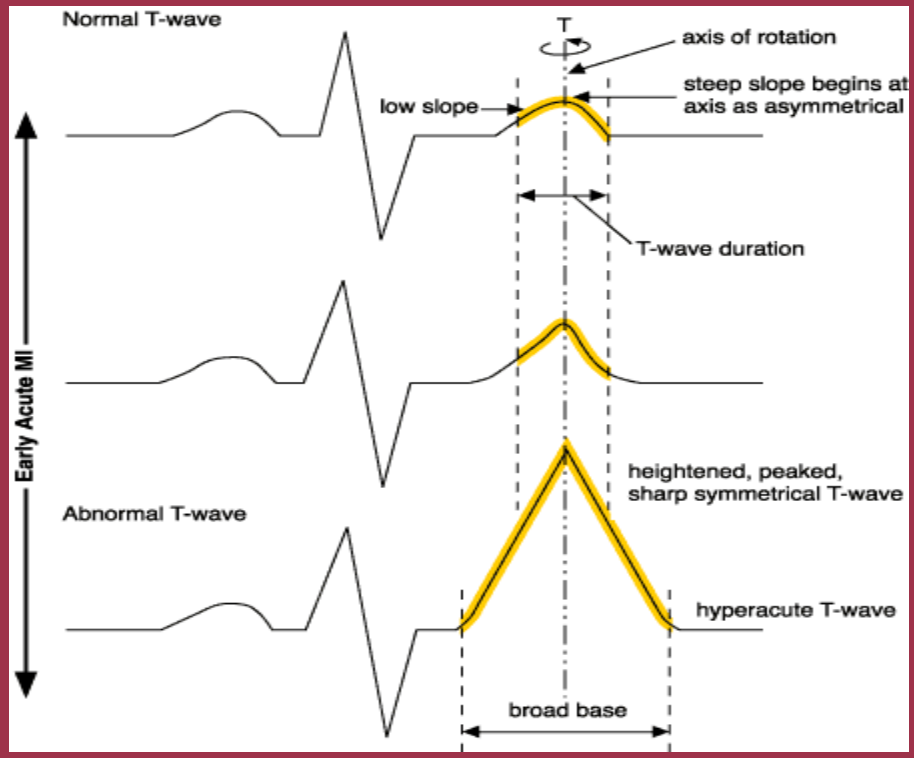


- Indicates Transmural Ischemia.

Ischemic ECG Changes

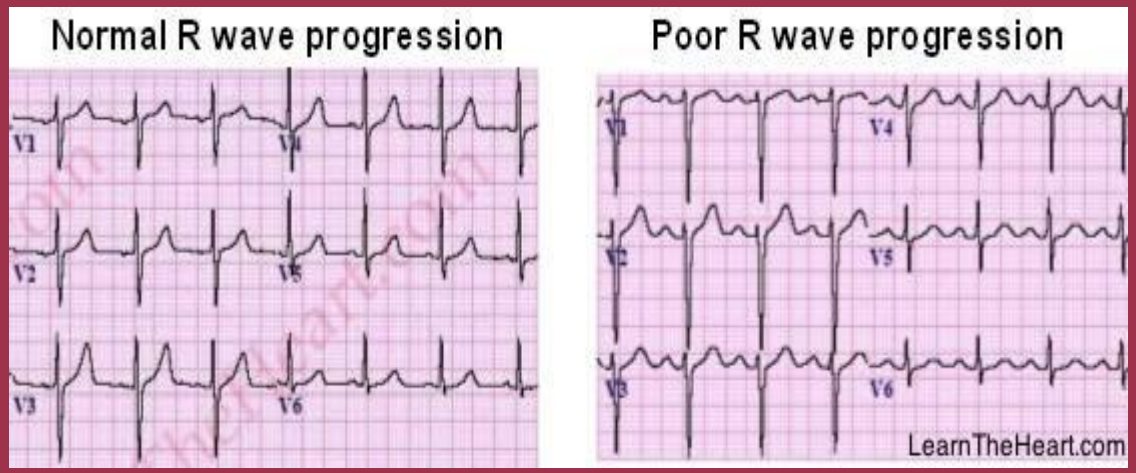
Hyperacute T wave

- Indicates Transmural Ischemia.
- Early sign of ischemia that can be seen even before ST elevation



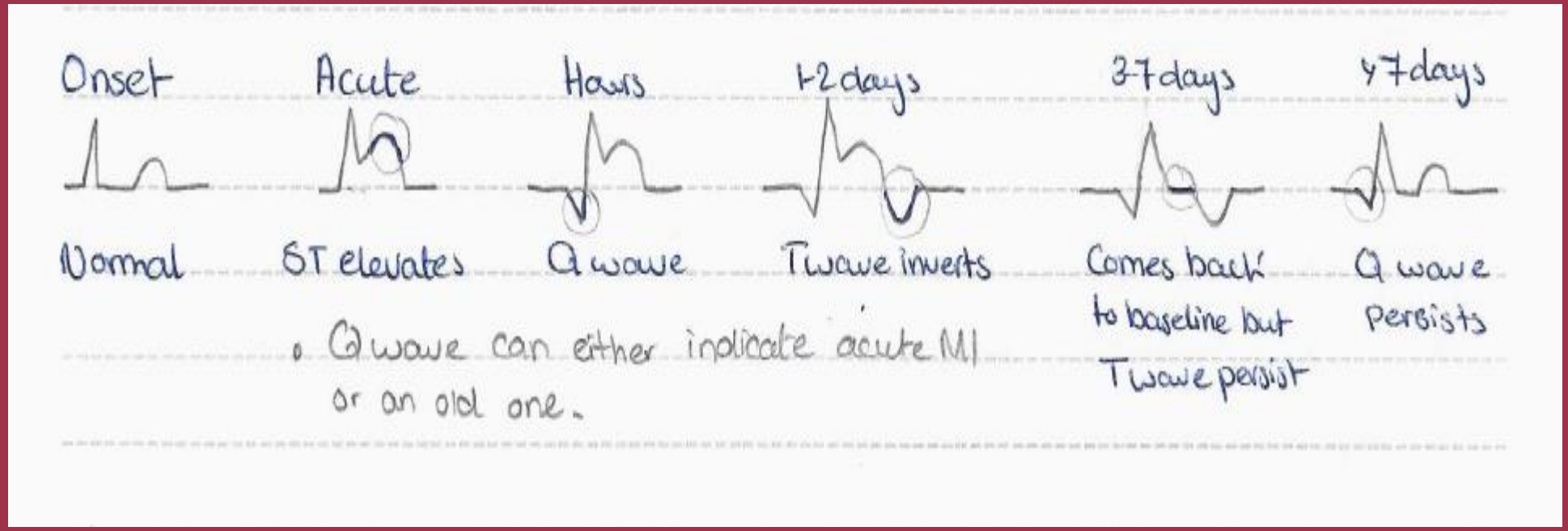
Ischemic ECG Changes

Poor R wave Progression



- Indicates Anterior Ischemia.
- Normally, R wave progress in size in leads V1 - V6, and R > S in lead V3.
- In ischemia, R wave remains tiny by V3 and S wave persists to V6.

Evaluation of EKG Changes in STEMI



IHD

- Stable Angina:
- Ischemic Chest pain with exertion, relieved by rest, no plaque rupture or thrombosis.
- Usually asymptomatic until 75% occlusion.
- Diagnosis: Cardiac Stress test (Treadmill).
- Treatment: Revascularization.

- Unstable Angina:
- Ruptured Plaque → Thrombus formation → Subtotal occlusion <100%.
- Causes ischemic chest pain with normal cardiac biomarkers.
- Diagnosis: From history; chest pain at rest / ECG: ST depression or T wave inversion.
- Treatment: Not an emergency; Aspirin + Heparin + Beta blocker + Angioplasty.

- NSTEMI:
- Ruptured Plaque → Thrombus → Subtotal occlusion <100%.
- Causes ischemic chest pain with abnormal cardiac biomarkers (raised Troponin) → Subendocardial Ischemia.
- Diagnosis: ECG: ST depression, T wave inversion. / Increased Troponins + CK-MB / Isolated increased AST.
- Treatment: Same as Unstable angina.

- STEMI:
- Thrombus causing total occlusion 100% causing ischemic chest pain.
- Transmural ischemia shows on ECG "ST Elevation" on leads:

Left Anterior Descending artery - Anterior Ischemia → V1 - V4
Left Circumflex artery - Lateral Ischemia → I + aVL + V5 + V6
Posterior Descending artery - Inferior Ischemia → II + III + aVF.

- Complications of STEMI: 1- Rt ventricular infarction → Decreased contractility → elevated JVP → Decreased Preload → Hypotension.

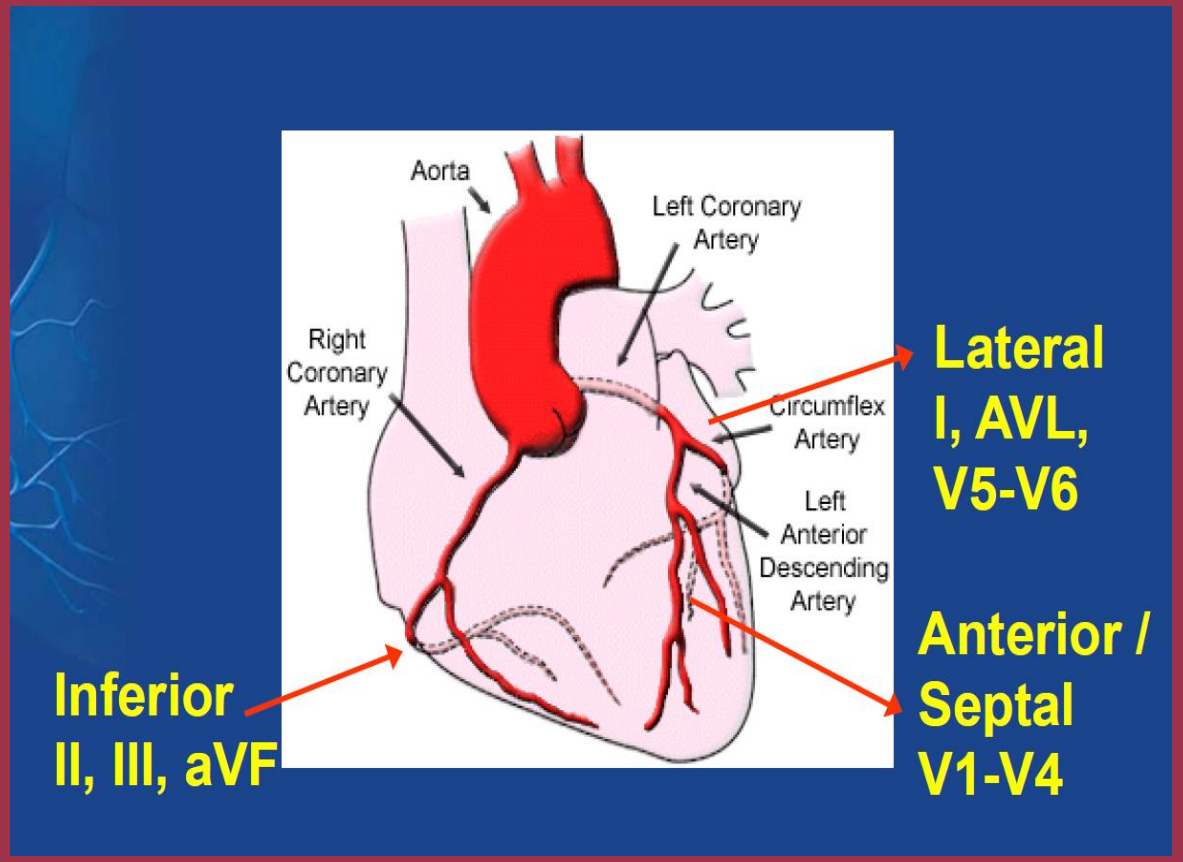
2- Sinus Bradycardia and Heart block → Vagal stimulation from Inf wall ischemia.

- Left main artery occlusion → Massive ischemia + ST elevation on aVR but the rest are depressed.
- PDA occlusion → Absence of ST elevation, only depression on V1-3, Requires special leads V7-9 (show elevation).
- Treatment: Angioplasty <90 mins OR Thrombolysis <30 mins + Supportive Tx (if thrombotic problem → Aspirin and Heparin / if ischemic problem → Beta blockers - to reduce O2 demand- + Nitrates.) + Clopidogrel.
- Caution: Beta blockers are contraindicated in inferior MI → they cause Bradycardia & AV block

Nitrates are contraindicated in Right coronary artery occlusion → Decrease preload leading to hypotension.

Coronary Anatomy

- Left main coronary artery supplies 70% of the myocardium.
- A patient with history of Angina → if he develops MI, he will sustain less Myocardial damage since Angina causes transient ischemia which stimulates opening of the collaterals.
- MI in Inferior leads indicates Rt coronary artery occlusion.
- MI in Anterior leads indicates Left anterior descending artery occlusion.

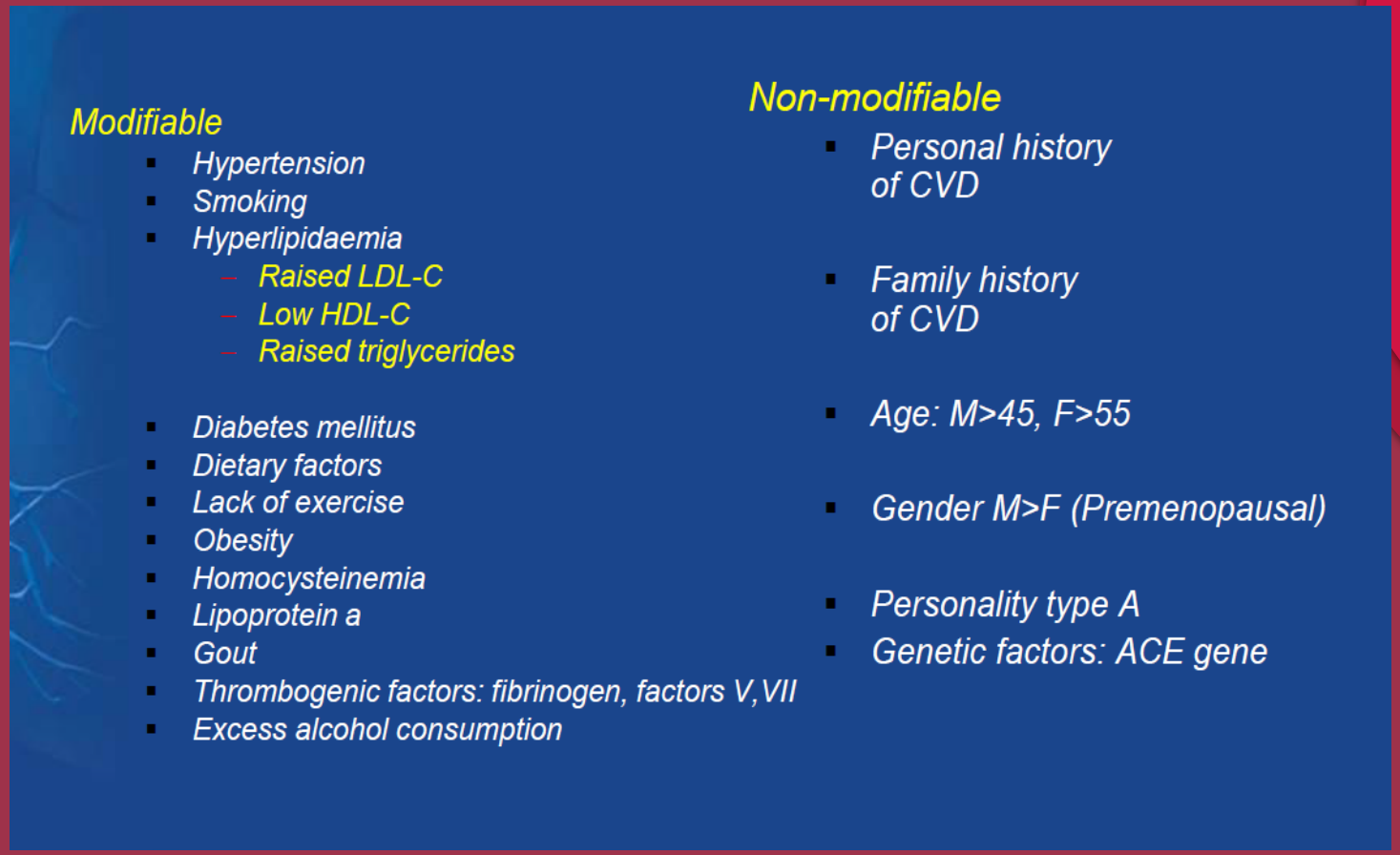


Causes of Coronary Artery Disease

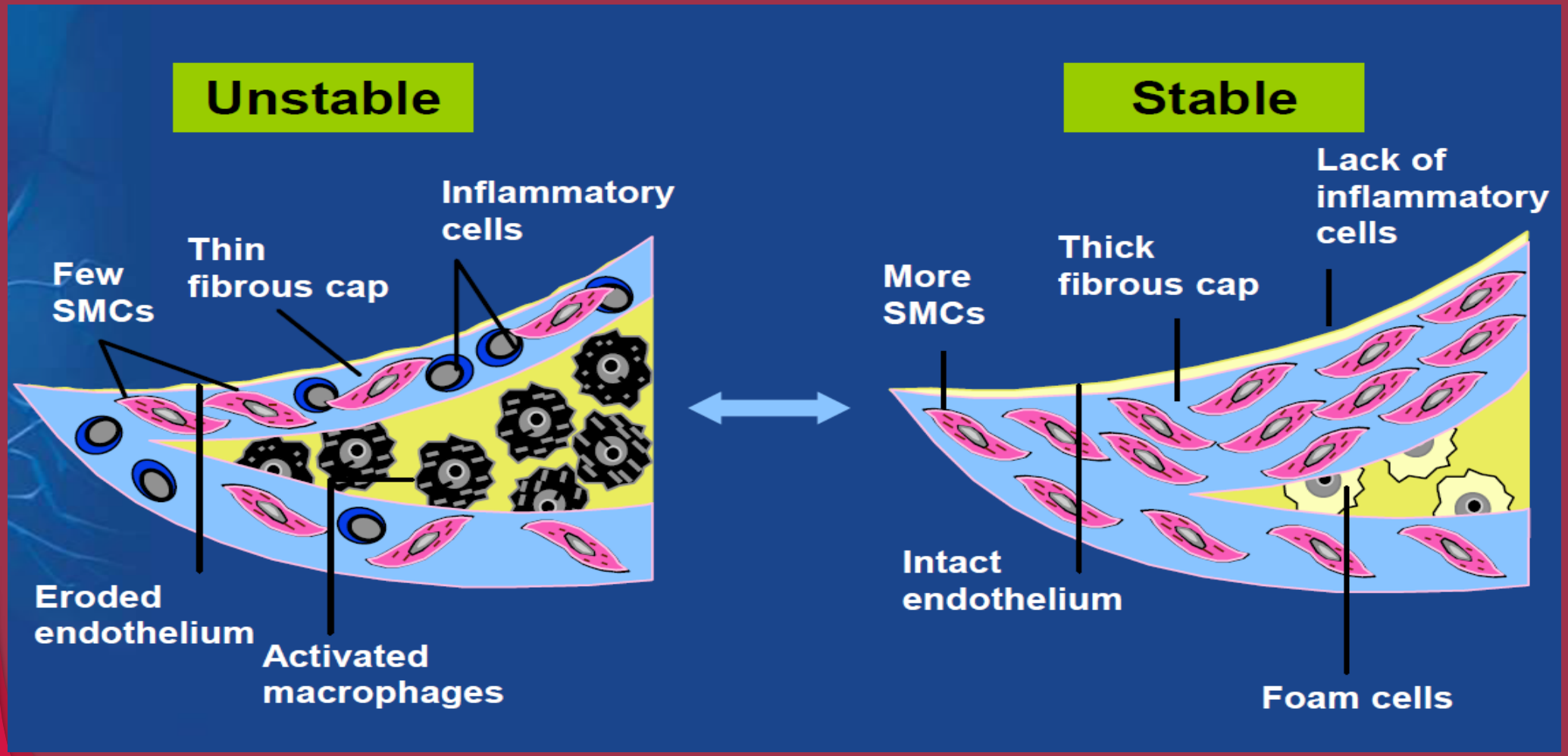
Atherosclerosis 95%	***Nonatherosclerosis***
Risk factors	*1-Arteritis (SLE,RA,Takayasu ,..)* *2-Embolism* *3-Coronary mural thickening (amyloidosis,radiation therapy,..)* *4-Coronary luminal narrowing: coronary spasm, aortic dissection* *5-Congenital coronary artery anomalies*

Risk Factors for Cardiovascular Disease

- Family History is positive if:
  Males < 55 Y/O
  Females < 65 Y/O



Characteristics of Unstable & Stable Plaque



Stable Angina

- Look for an evidence of Atherosclerosis elsewhere.
- Symptoms of Atherosclerosis: like intermittent claudication, history of Angina

Commonest form of angina

Causes: imbalance between demand and supply

Symptom: chest pain

Location: central chest (others)

Radiation: arm(s), neck, jaw

Character : squeezing, pressure, heaviness,..

Duration: 2-10 minutes

Precipitating factors: exertion, emotional upset, heavy meal, sexual intercourse, cold weather

Relieving factors: nitrate, rest

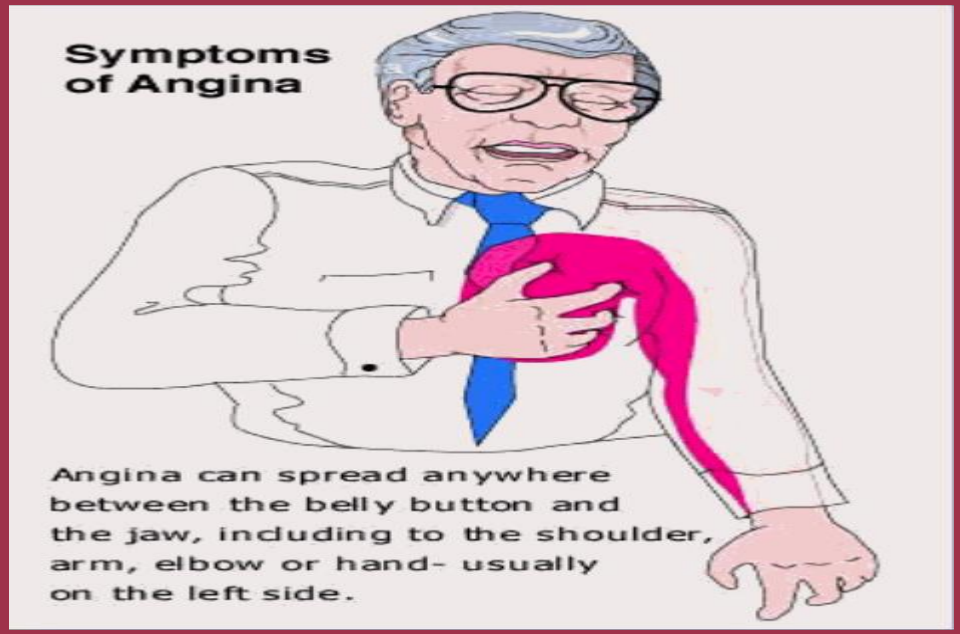
Associated symptoms: dyspnea, diaphoresis, nausea

Classes of angina: 1- 4

Physical Examination: normal, sign of risk factors, peripheral vascular disease

Anginal Pain

- Anginal pain is usually retrosternal, but it could also be in an atypical sites such as the epigastrium, jaw, teeth, and between the scapulae or the shoulders.



NYHA Grading of Cardiac Symptoms (Angina / Dyspnea)

- Important to determine management and prognosis

Grade 1:

Cardiac disease without resulting limitation of physical activity. Ordinary physical activity does not cause chest pain (dyspnea).

Grade 2:

Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity result in chest pain (dyspea).

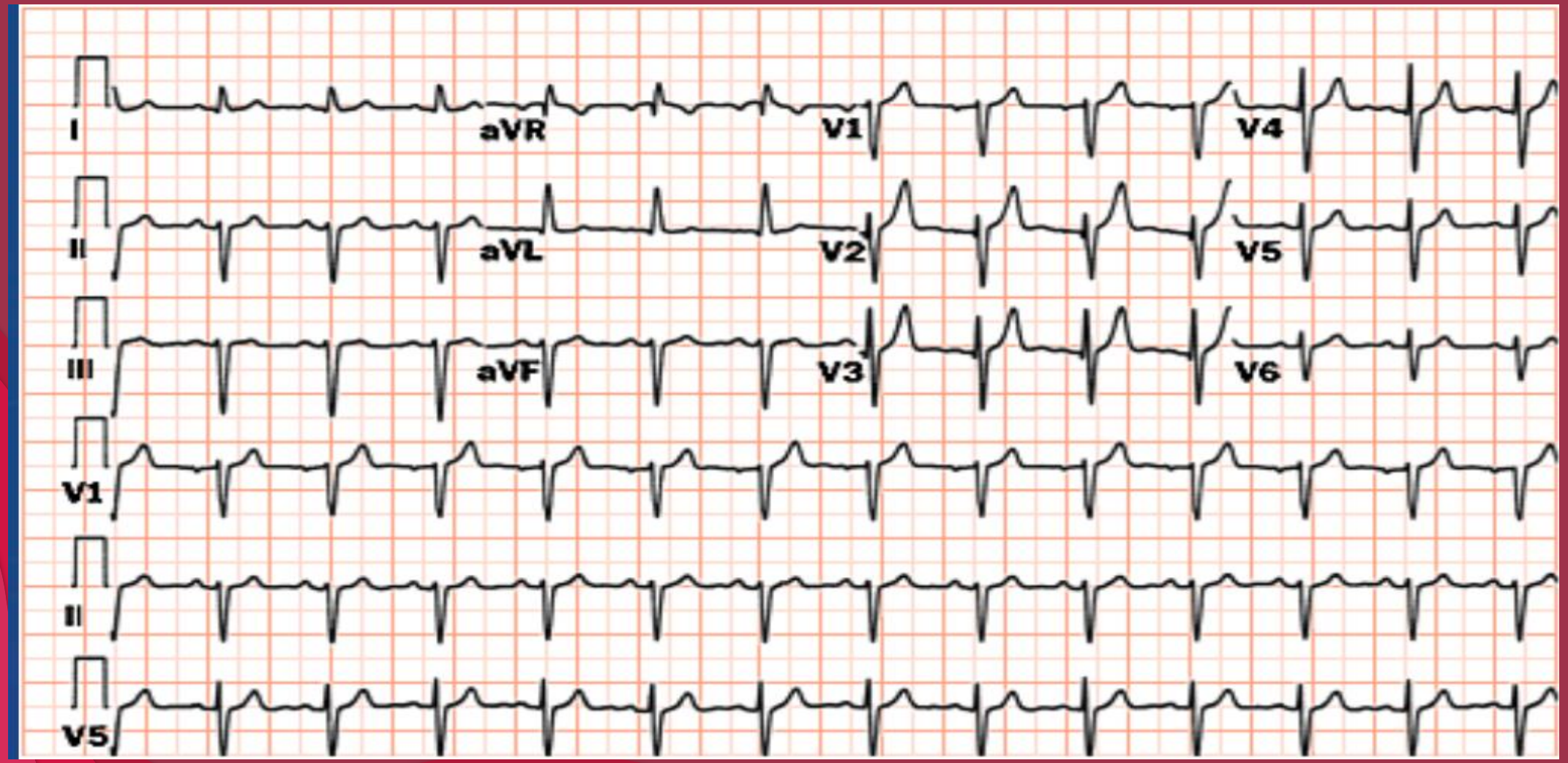
Grade 3:

moderate limitation in physical activity. Comfortable at rest. Less than ordinary activity causes symptoms

Grade 4:

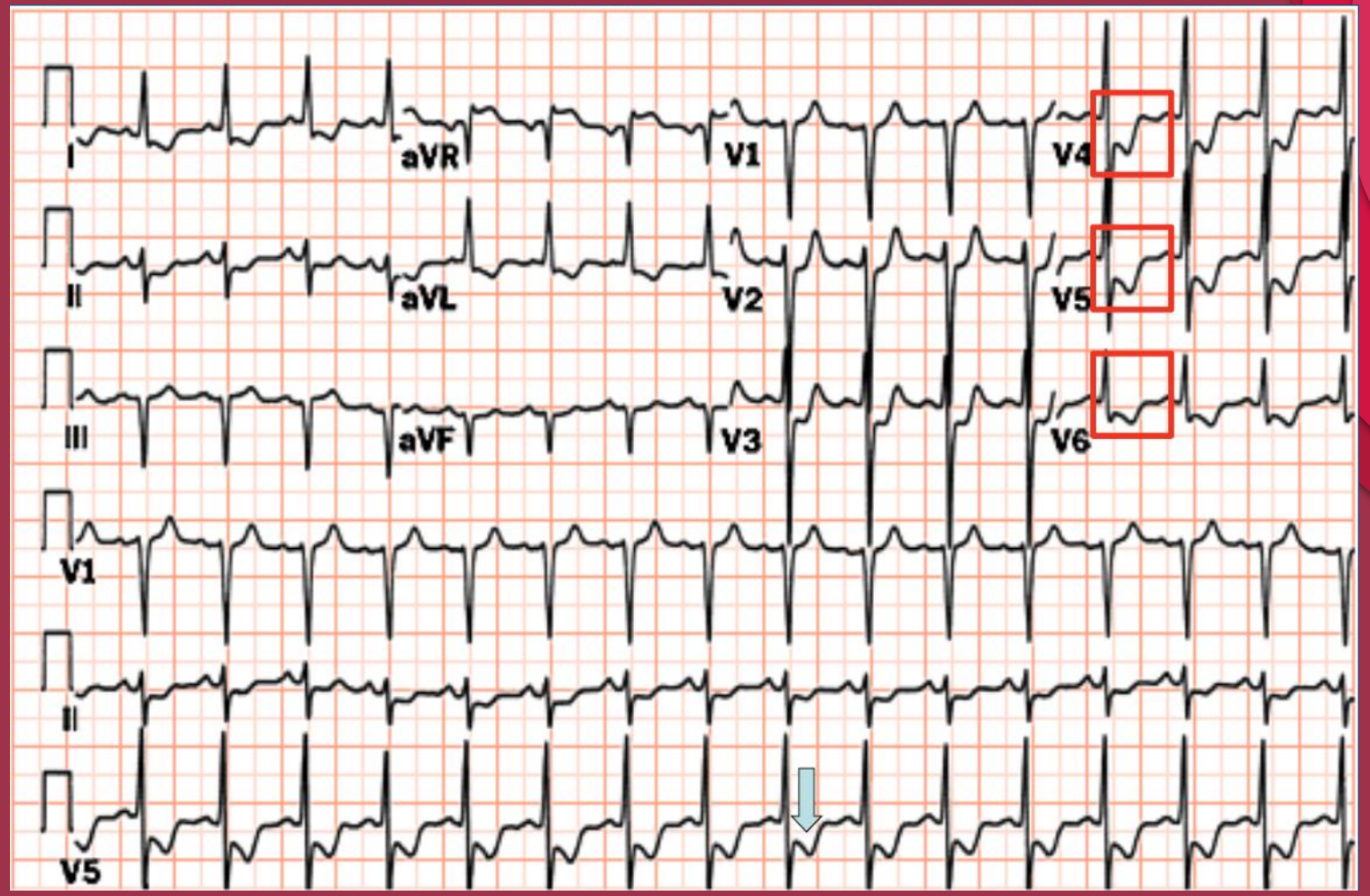
sever limitation: symptoms at rest.

Normal Resting Electrocardiogram



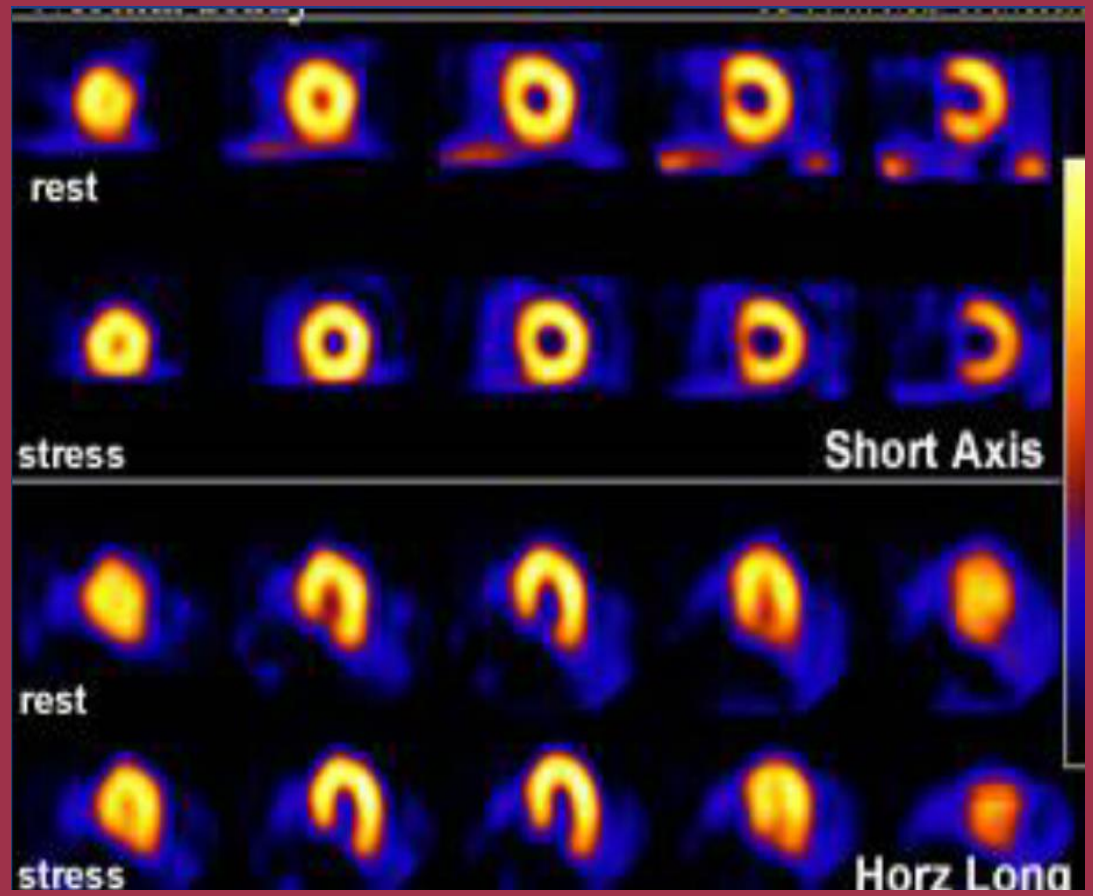
ECG During Exercise

- Leads V4,5,6 show significant ST depression, indicating Ischemia → Positive Exercise Stress Test.



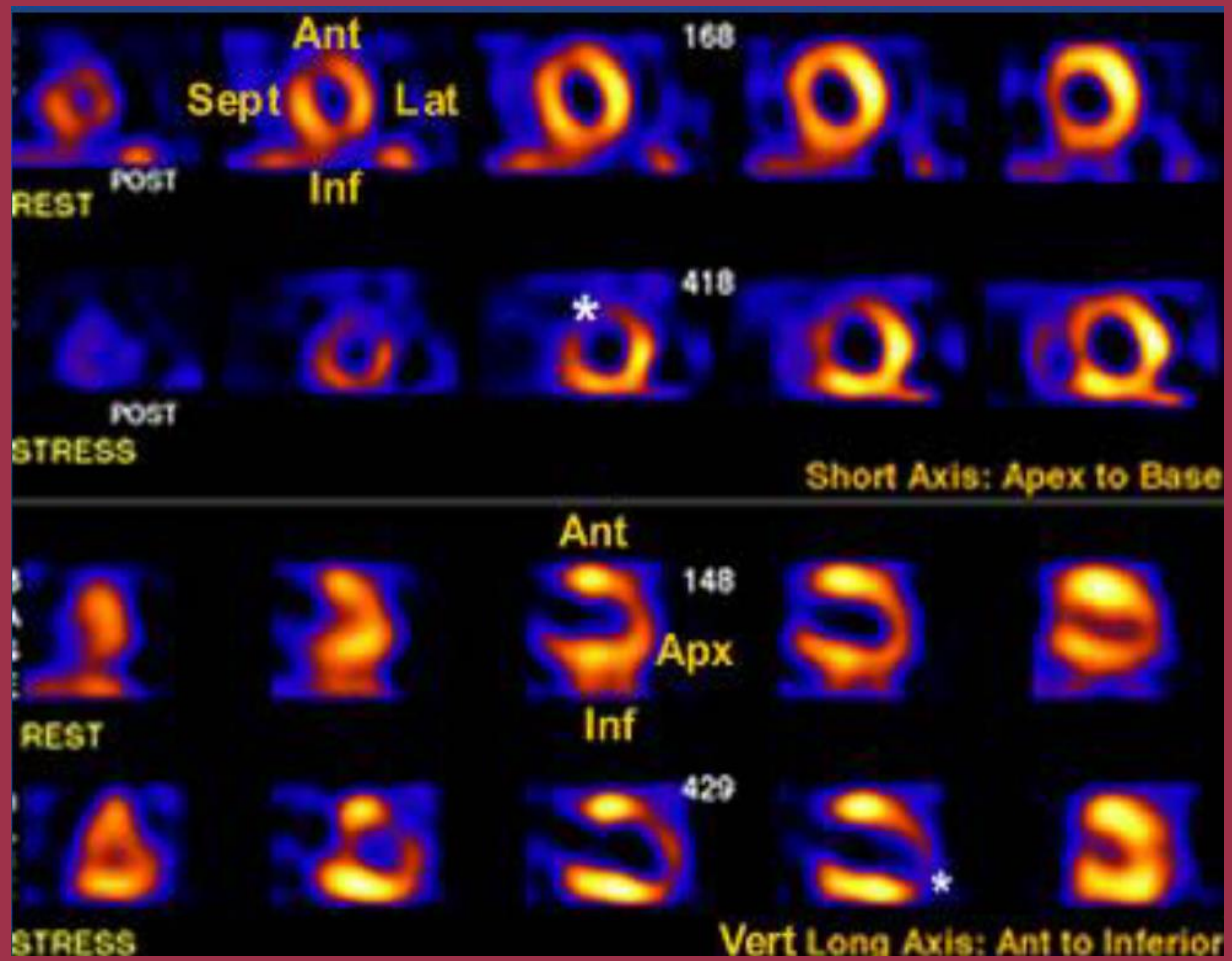
Exercise or Dipyridamole Thallium

- Decrease uptake of the nuclear isotope in ischemic areas during exercise.
- This figure shows normal Myocardial perfusion.



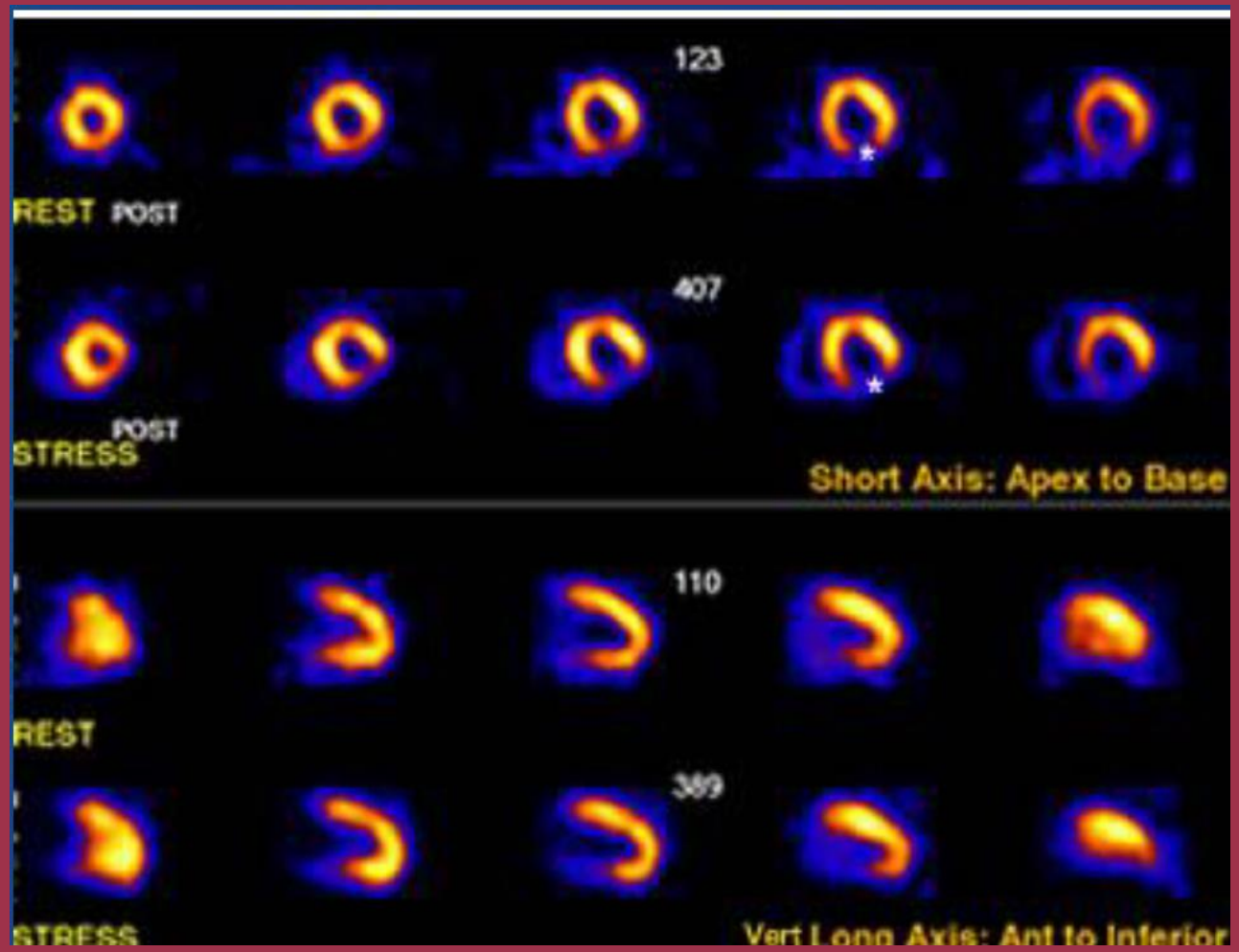
Exercise or Dipyridamole Thallium

- This figure shows Myocardial Ischemia



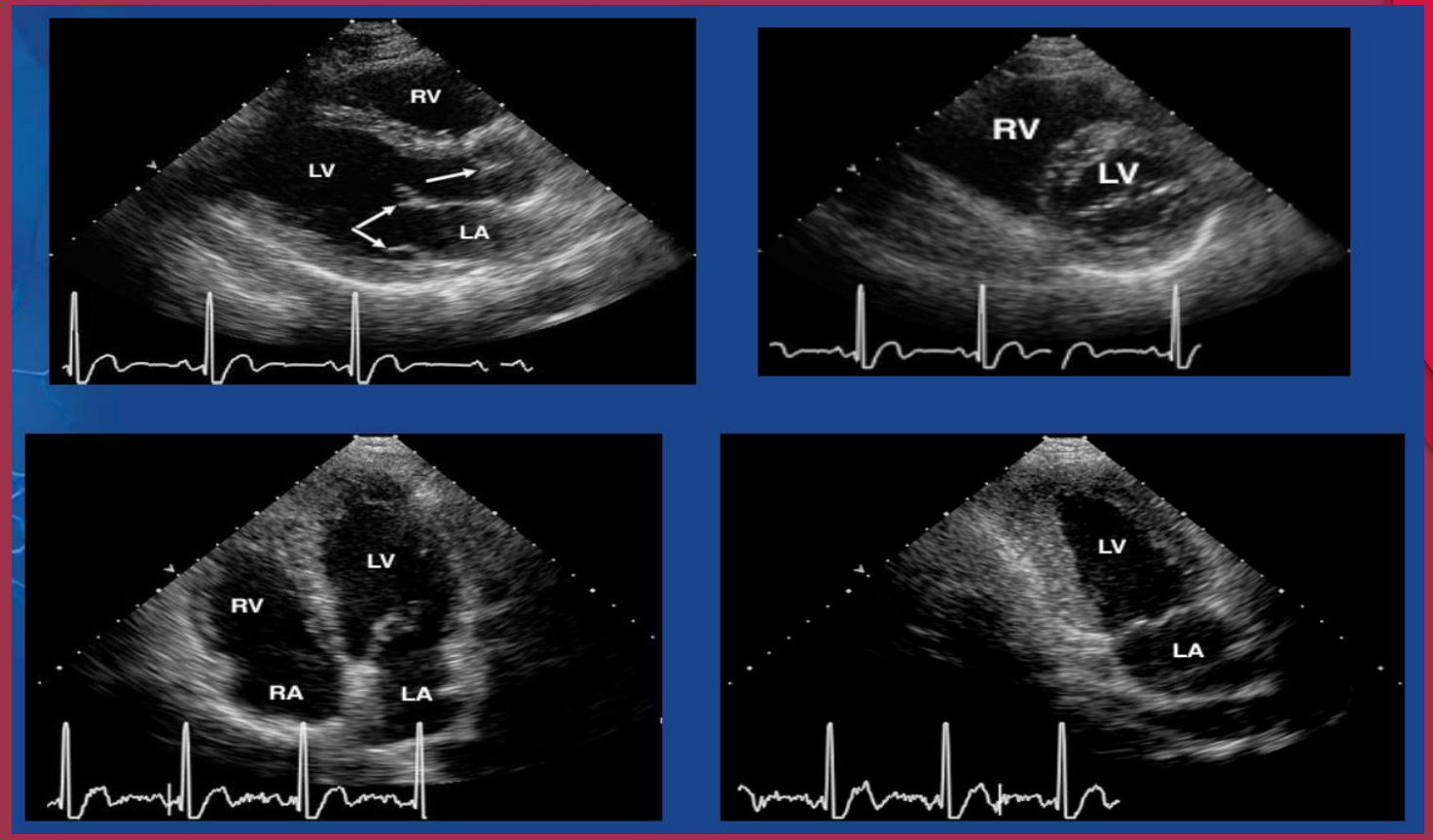
Exercise or Dipyridamole Thallium

- This Figure shows Myocardial Infarction



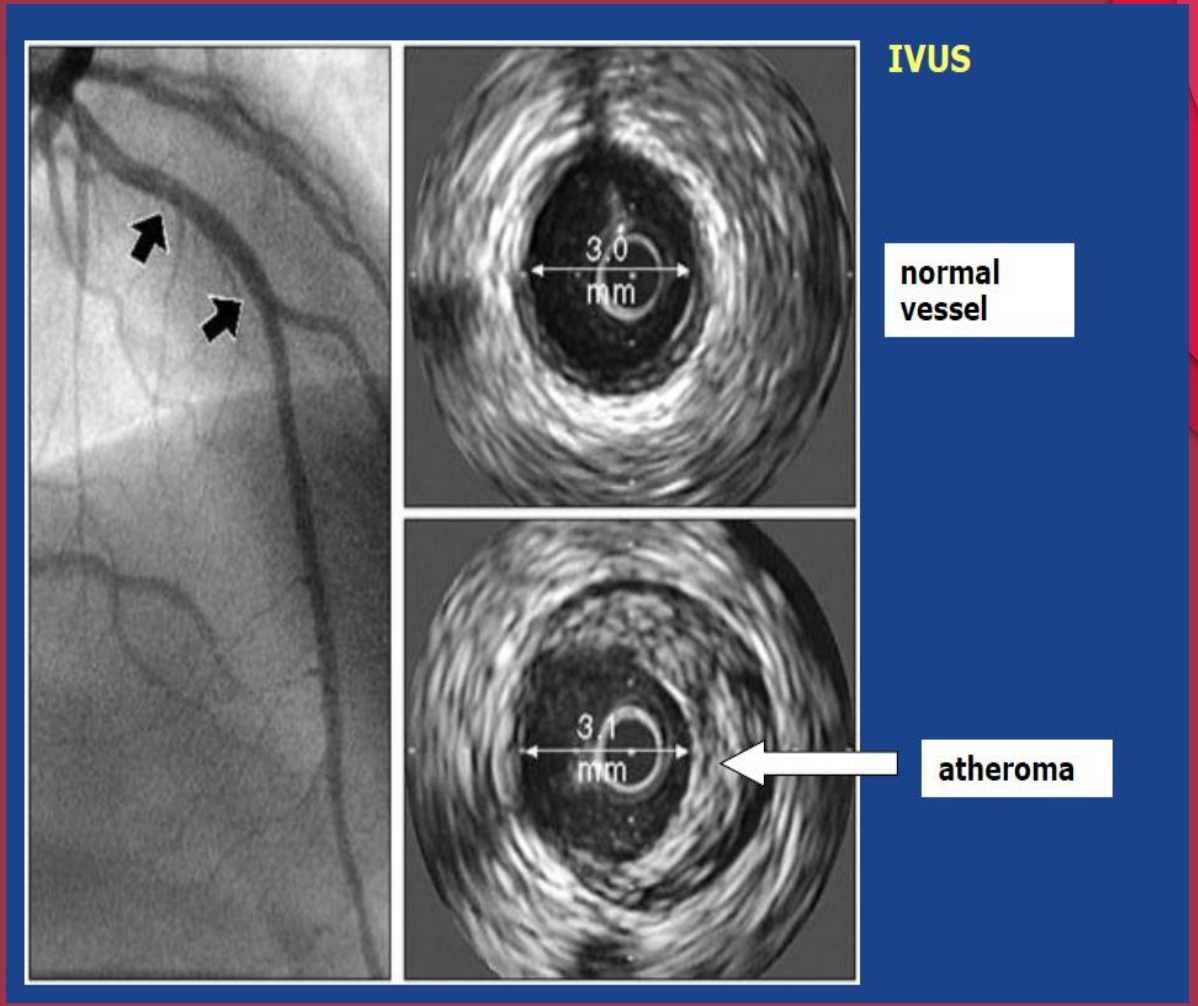
Exercise or Dobutamine Echocardiogram

- It shows wall motion abnormalities.
- Defected (Ischemic) Area will be hypokinetic



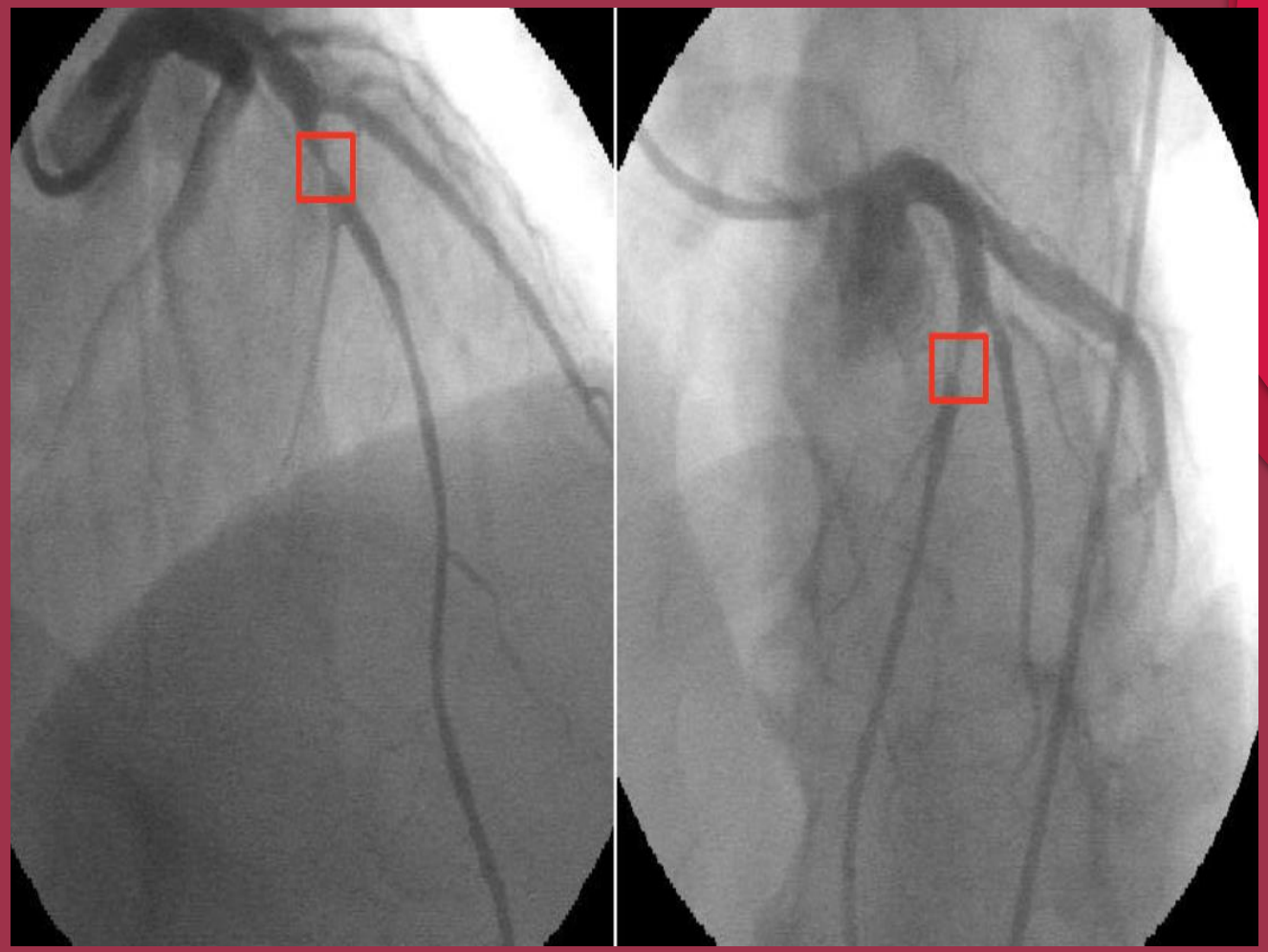
Intravascular Ultrasound (IVUS)

- Invasive imaging technique used to assess atherosclerosis.
- It shows Atheromatous plaques.



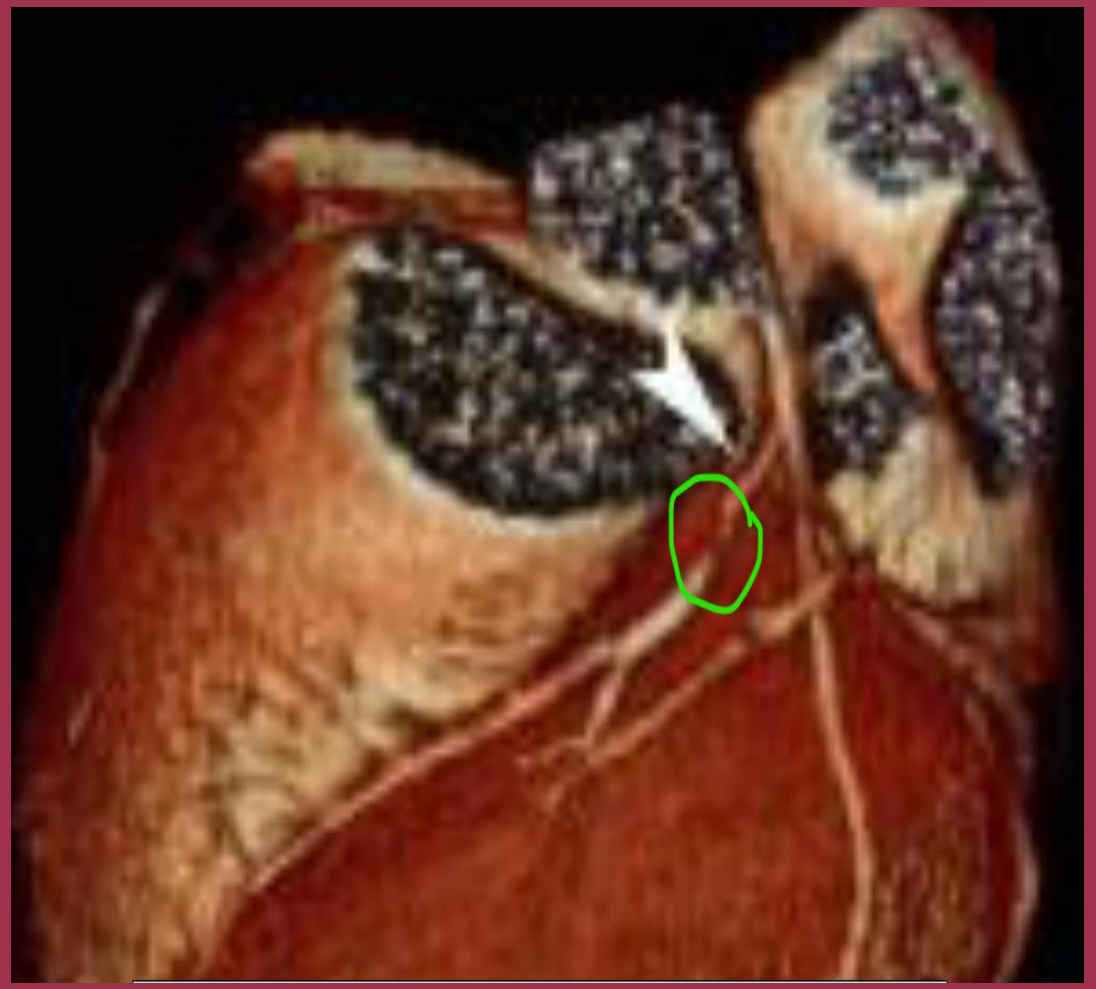
Cardiac CATH

- Red Squares show Subtotal lesions.



Cardiac CT Angiography

- Can show Stenosis



Medical Therapy of Stable Angina

- Beta Blockers are the cornerstone of treatment as they Increase diastolic time and decrease heart rate thus increasing O2 supply and decreasing O2 demand respectively.

Prognostic: Aspirin, Statines, ACEI

Symptomatic: Nitrate,B-,CA-blocker, (nicorandil, ranolazine, ivabradine)

INCREASE O2 Supply

1-Increase diastolic time: B-blocker

2-Decrease coronary tone: nitrate, ca-blocker

3-Decrease LV diastolic pressure: nitrate

4-Correct coronary stenosis: PCI, CABG

5-Increase O2 capacity of blood: transfusion if anemia

DECREASE O2 Demand

1-Decrease heart rate: B-blocker, ca-blocker

2-Decrease contractility: B-blocker, ca-blocker

3- Decrease wall tension (LV pressure and cavity radius): nitrate

4- metabolic: trimetazidine

Treatment in Practice

- General measures include Correction of established risk factors + Weigh reduction + Aerobic exercise (improve functional capacity, well-being sensation) + Treatment of Anemia, Thyrotoxicosis, arrythmias.

1-General measures
2-Aspirin
3-Nitrate: S/L, Oral, dermal
3-B-blocker
4-Statins: LDL>100 mg/dl(70mg/dl)
5-Ca-blocker
6-Angio :PTCA,CABG

Cardiac X Syndrome

- A historic term for Microvascular Angina; Angina with signs associated with decreased blood flow to heart tissues with normal coronary arteries.

Typical, exertional angina with positive exercise stress test

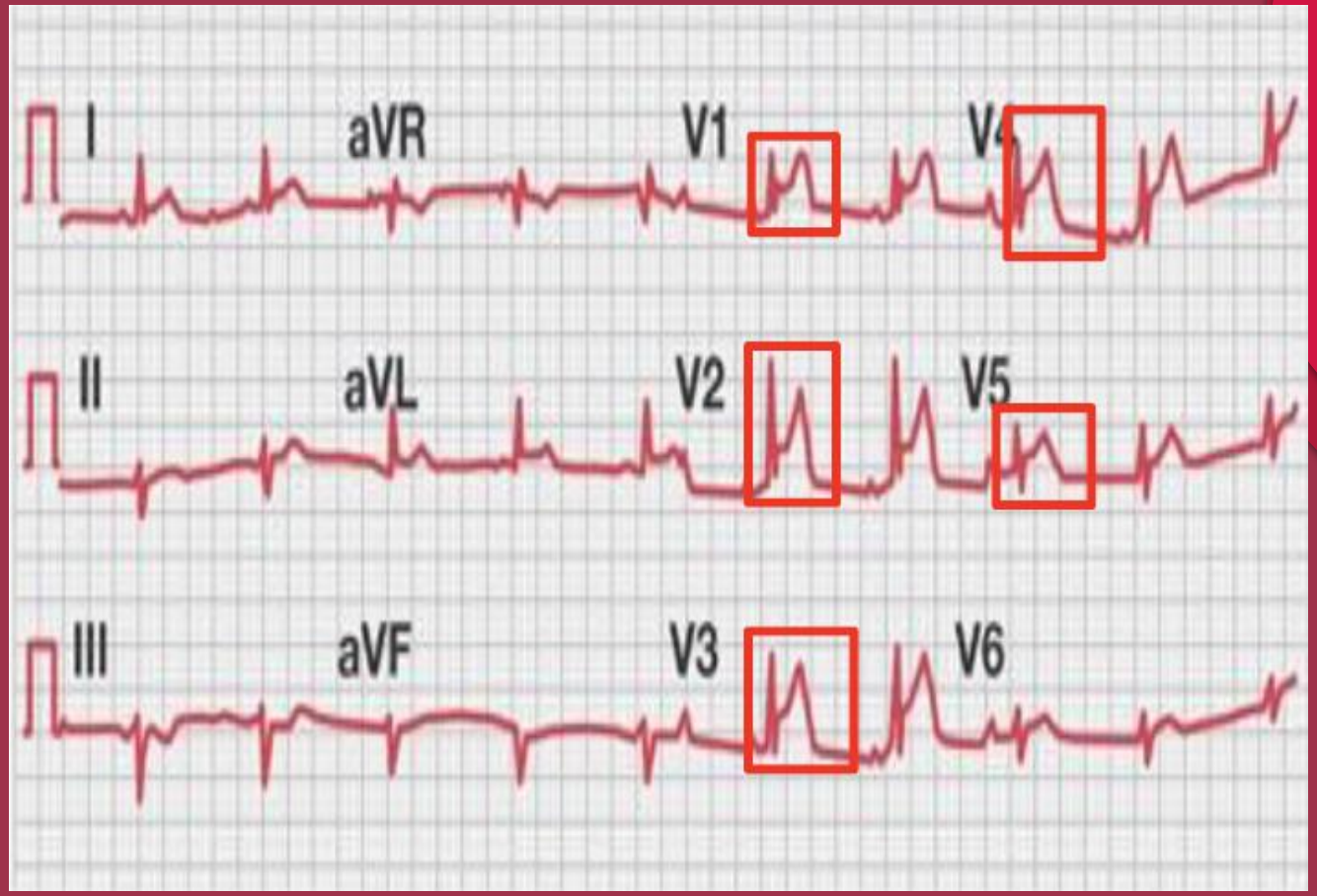
Anatomically normal coronary arteries
Reduced capacity of vasodilation in microvasculature

F>M
Young > Elderly
Excellent prognosis
Antianginal therapy is rarely effective

Long term prognosis very good

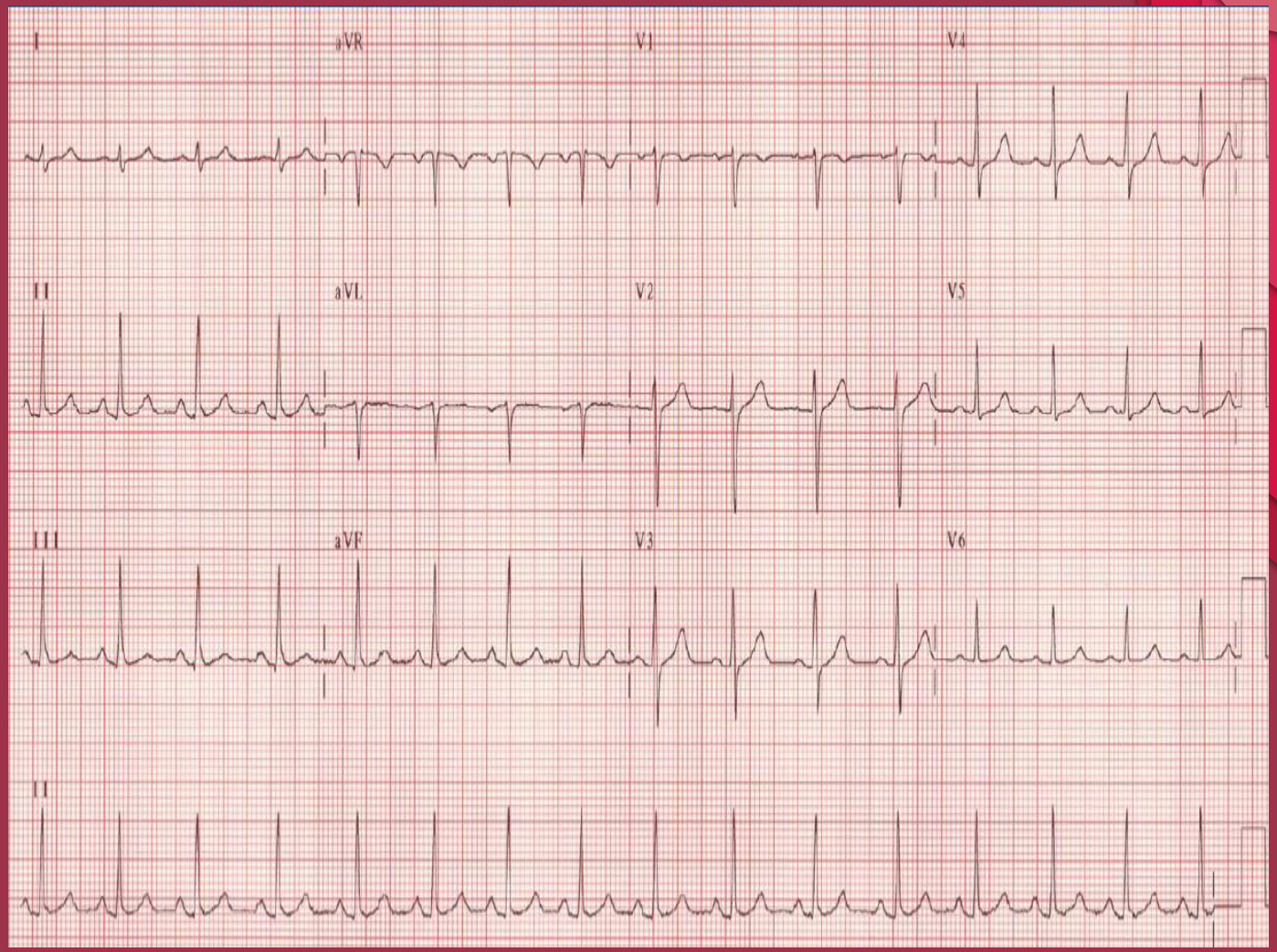
Variant / Prinzmetal Angina

- The figure illustrates the ECG reading during the attack.
- We have ST elevation in leads V1-5.



Variant / Prinzmetal Angina

- This figure illustrates the ECG reading after taking Sublingual Isosorbide Dinitrate.
- ST elevations became normal after nitrates administration.



Variant / Prinzmetal Angina

- Beta blockers are contraindicated because it increases Heart rate and spasms.
- Beta blockers are the cornerstone in treating all ischemic heart disease patients except Variant Angina

Chest pain with ST-Segment elevation
Usually at rest, at night
Troponin: negative
Female > male

Spasm of large epicardial coronary vessel during the attack
transmural ischemia
70% on top of atherosclerosis
Vasospastic symptpms in other organs: Migraine, rhynauds

Can cause arrhythmias and death

Treatment: CA-blocker, Nitrate

B-blocker is contraindicated

Prognosis: 5 year mortality < 5%

Acute Coronary Syndrome

Acute Coronary Syndrome

- When pain duration is >30 mins, we should suspect Acute coronary syndrome (MI).
- Other DDx of acute chest pain: Aortic Dissection, Massive PE, etc.
- Unstable Angina has no elevations in cardiac biomarkers.

The spectrum of clinical conditions ranging from:

- **STEMI (Q-wave MI):** Total occlusion
- **NSTEMI (non-Q wave MI):** Subtotal occlusion
- **unstable angina:** Subtotal occlusion

Characterized by the common pathophysiology of a disrupted atherosclerotic plaque (rupture, erosion, or fissure)

Pathogenesis of ACS

- Any patient with Ischemic Heart Disease must be on long-term antiplatelet therapy to decrease the progression of thrombosis.

Plaque rupture

THROMBOSIS

1- Primary hemostasis: Initiated by platelet

platelets adhesion, activation, and aggregation---platelet plug

2- Secondary hemostasis:

activation of the coagulation system---fibrin clot.

These two phases are dynamically interactive:

Platelet can provide a surface for coagulation enzymes

Thrombin is a potent platelet activator

Acute Myocardial Infarction

- Chest pain is retrosternal, more severe, usually compressing, radiating to the arm, jaw, etc.
- A patient with unexplained Hypotension → Think of MI, it's a bad prognostic sign.
- S4 (common) indicates Left Ventricular stiffness.
- S3 (worst) indicates impending Left HF.
  - Systolic Murmur.
  - Rubs indicate Pericarditis.

THE MOST COMMON CAUSE OF DEATH
RUPTURE ATHEROMATOUS PLAQUE---CORONARY OCCLUSION

***Clinical Manifestation*:**

Chest pain: usually at rest, early morning

> 30 minutes (site, radiation, severity, character, radiation, associated phenomena..)

painless MI (10-15%): DM, elderly

Present as: Hypotension, Heart failure, Arrhythmia

***Physical Examination*:**

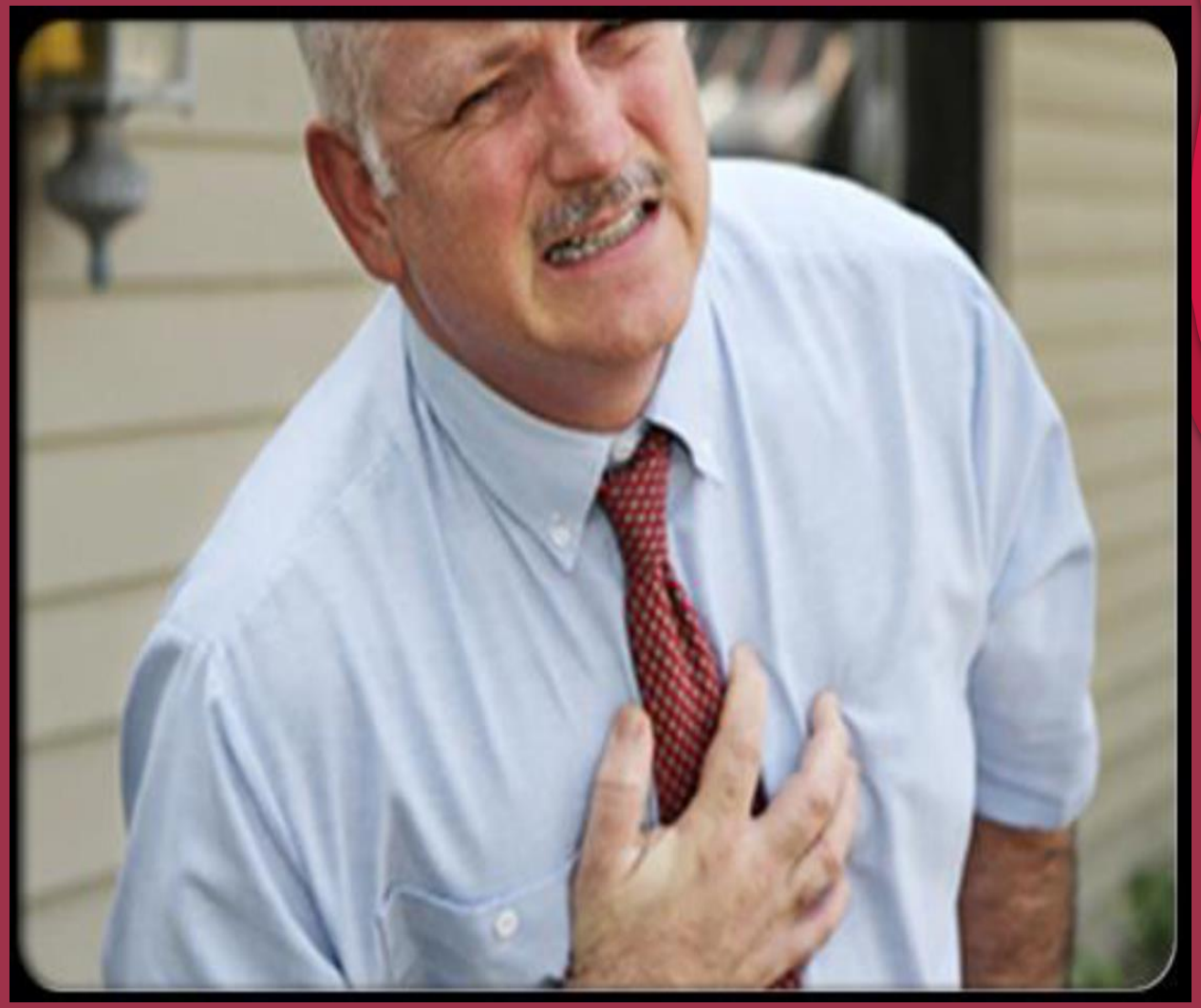
anxious, stressed, sweaty

vital sign: BP, Pulse, Temp

auscultation: S4,S3, Murmure, Rub

Levine's Sign

- A clenched fist held over the chest to describe ischemic chest pain.



Diagnosis of MI

- Normal ECG won't exclude MI; since patients present with normal ECG initially.
- High sensitivity troponin is what we usually use.
- Troponins and CPK together are used to determine the time of MI:
  - Both elevated → within the last 3 days
  - High Troponin, Normal CPK → More than 3 days
- CPK alone is preferred when we need to assess whether a new MI occurred on top of an old one

1-History

2-ECG (Electrocardiogram): STMI and NSTMI

- Hyperacute T wave
- ST-segment elevation
- Q- wave
- T- inversion
- ST-segment depresion

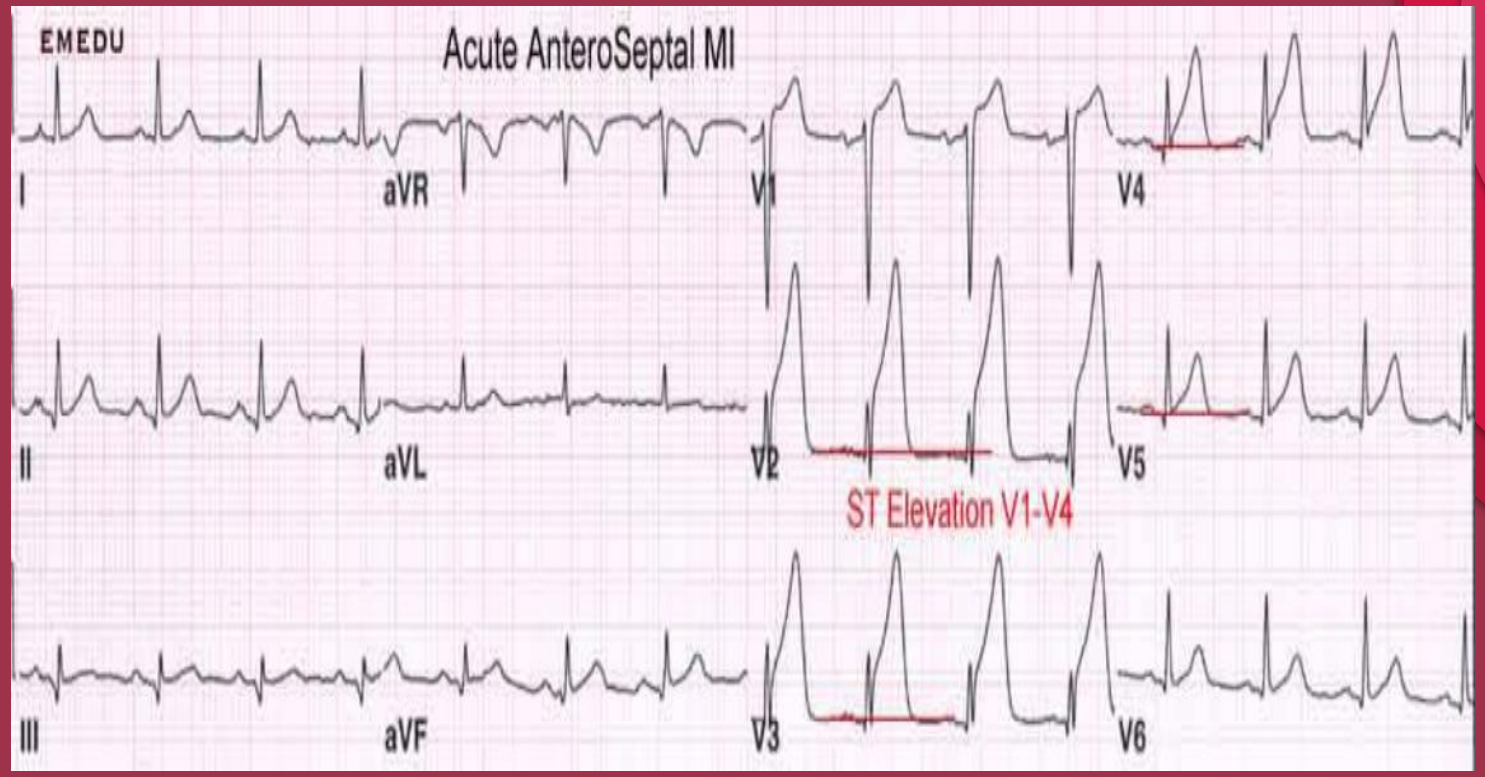
normal ECG will not exclude MI

3-Cardiac Marker: Troponin,CPK, myoglobulin,..

- Troponin T,I: 4-6 Hr (HsT 2-4 hr)
  - last 10-14 days
- CPK:4-6 Hr, peak 17-24hr, normal 72 hr
  - MB(MM,BB)
  - MB2/MB1 >1.5

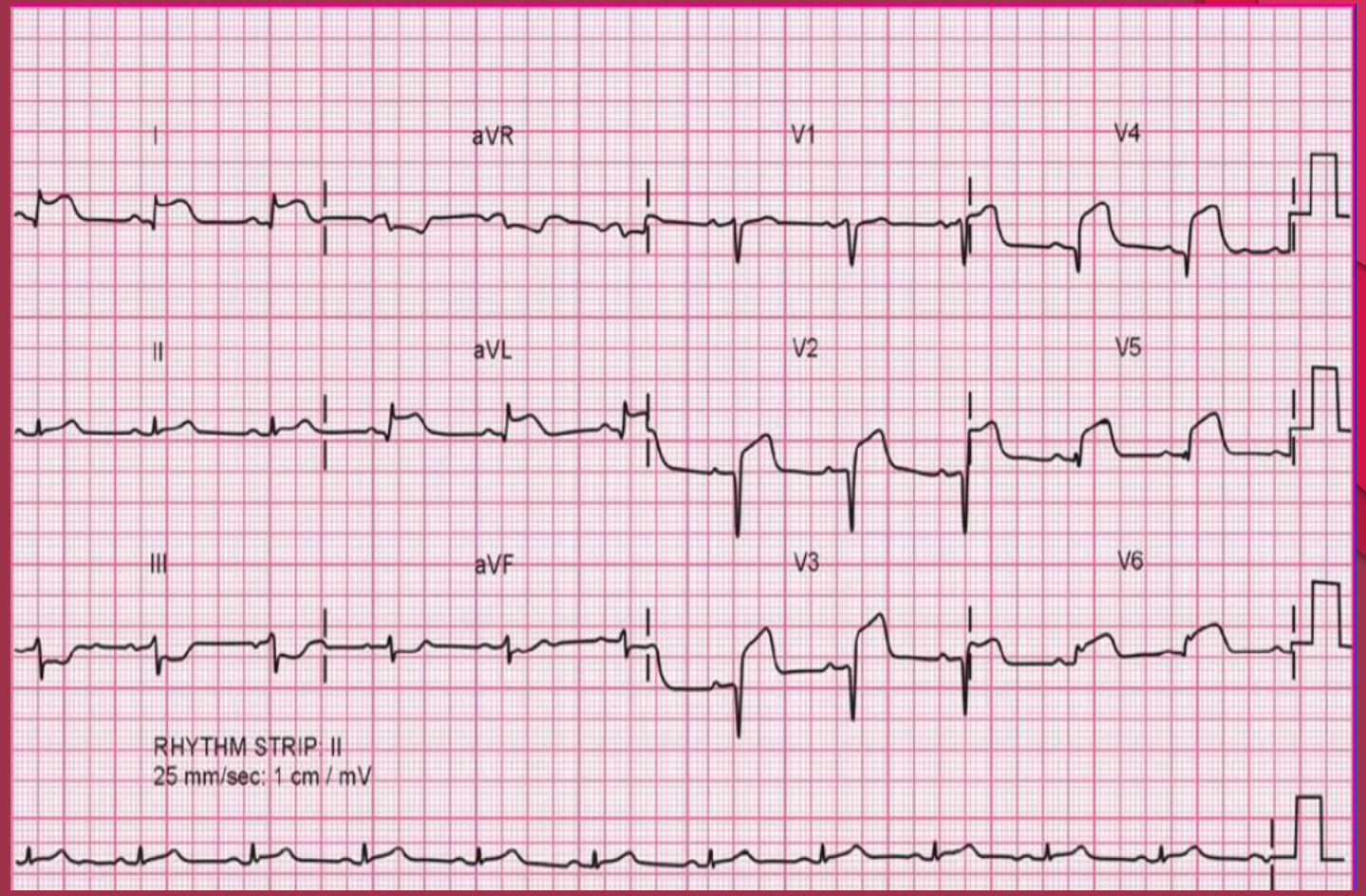
MI ECG

- ST Elevation in Leads V1-4
  → Anterior MI.
  - Hyperacute T waves
    →New MI (within 24 hours).
- So, the diagnosis is: New onset Anterior MI



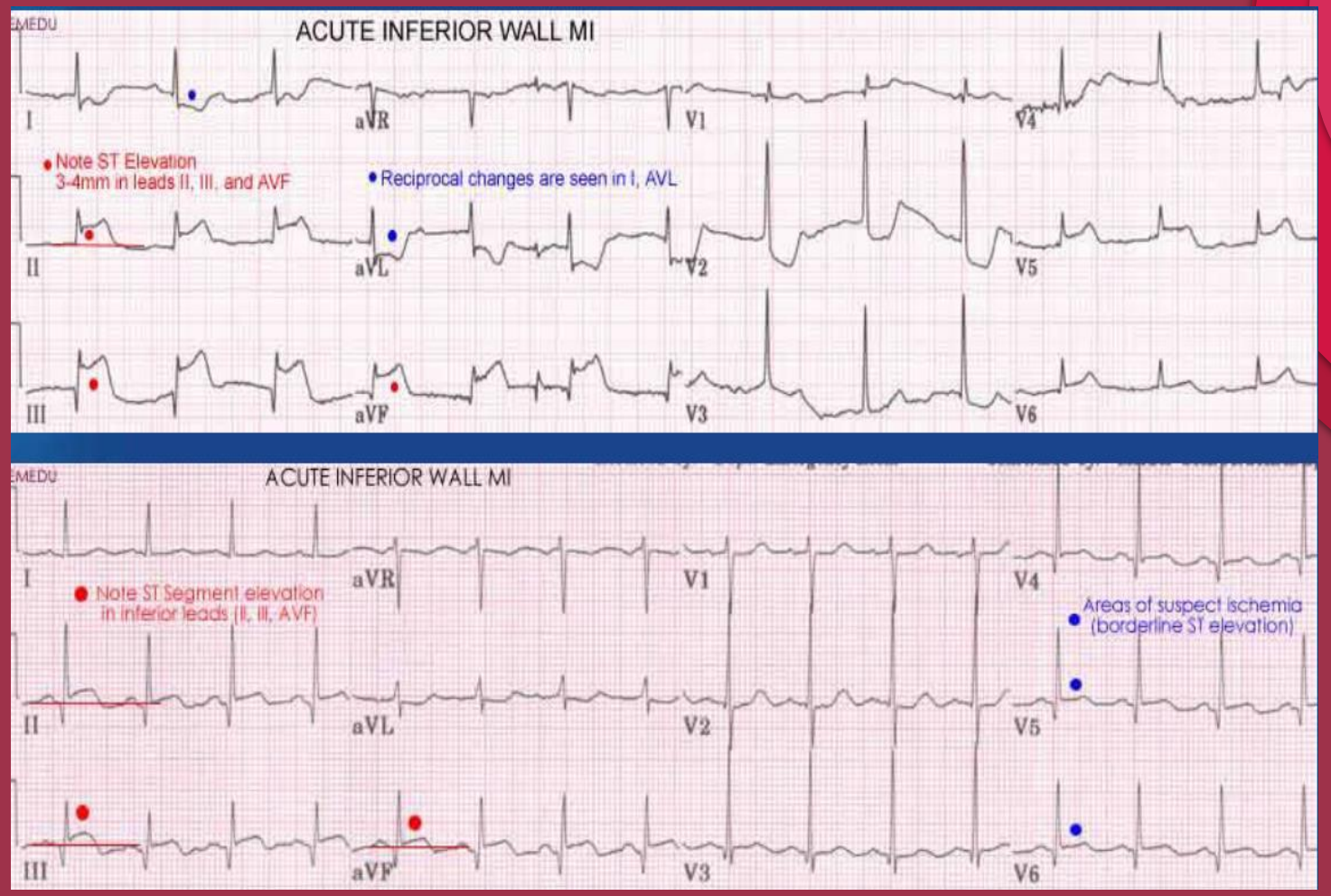
Diagnosis ?!

- Extensive Anterolateral MI



Diagnosis ?!

- Acute Anterior wall MI



ECG Criteria for Significant ST Elevation

- Elevation should be present in 2 adjacent leads.
- In limb leads: elevation should be at least 1 mm regardless of gender + Elevations should be present in at least 2 other adjacent chest or limb leads.

V2-V3 Leads:

Men ≥ 40 years	≥ 2 mm
≤ 40 years	≥ 2.5 mm
Women	≥ 1.5 mm

≥ leads1mm IN at least two other adjacent chest or limb leads

LBBB

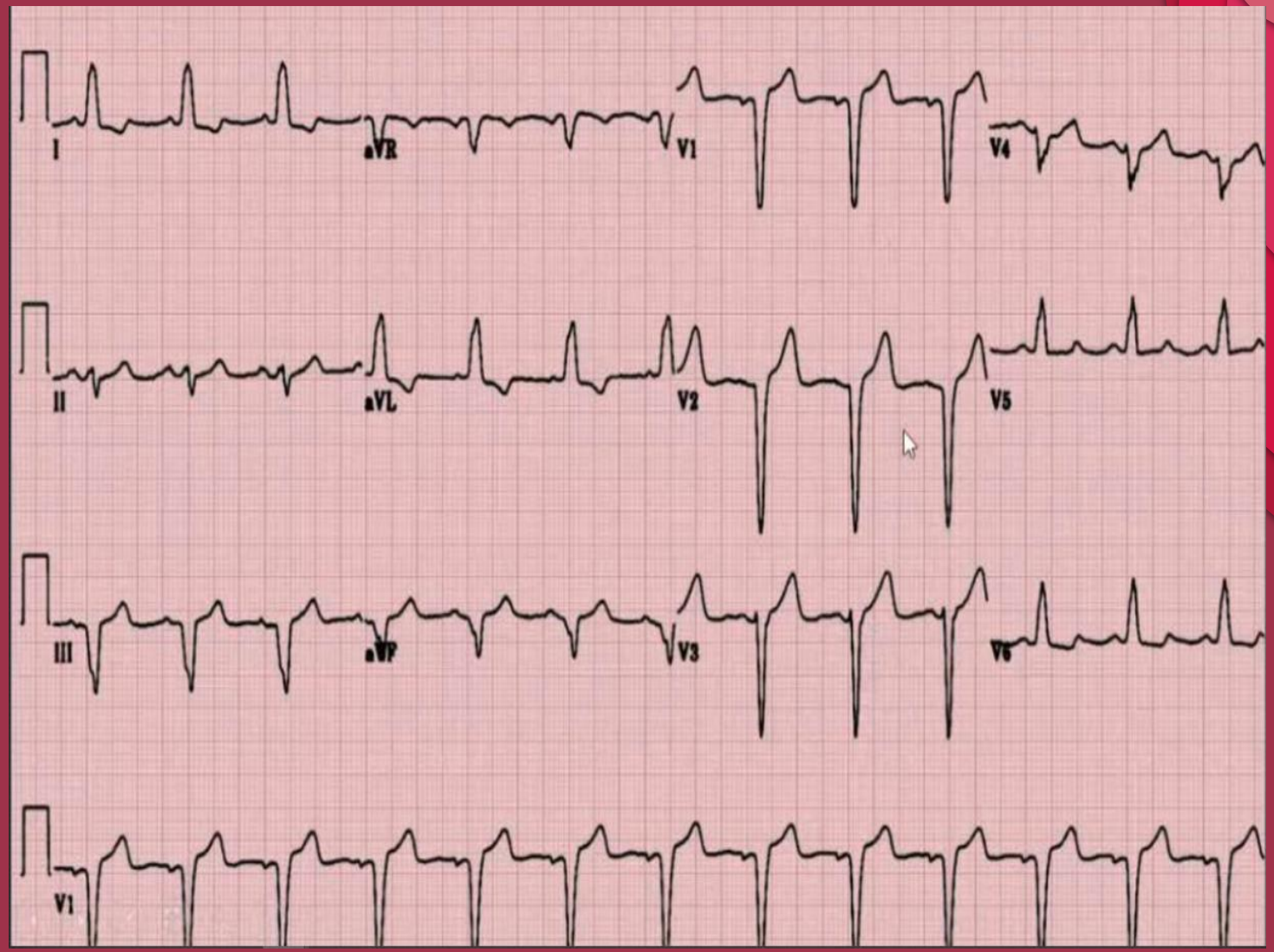
- Can also cause ST elevation.
- ECG:

1- QRS duration >120 ms

2- Absence of Q wave in leads I, V5 and V6.

3- Monomorphic R wave in I, V5 and V6.

4- ST and T wave displacement opposite to the major deflection of the QRS

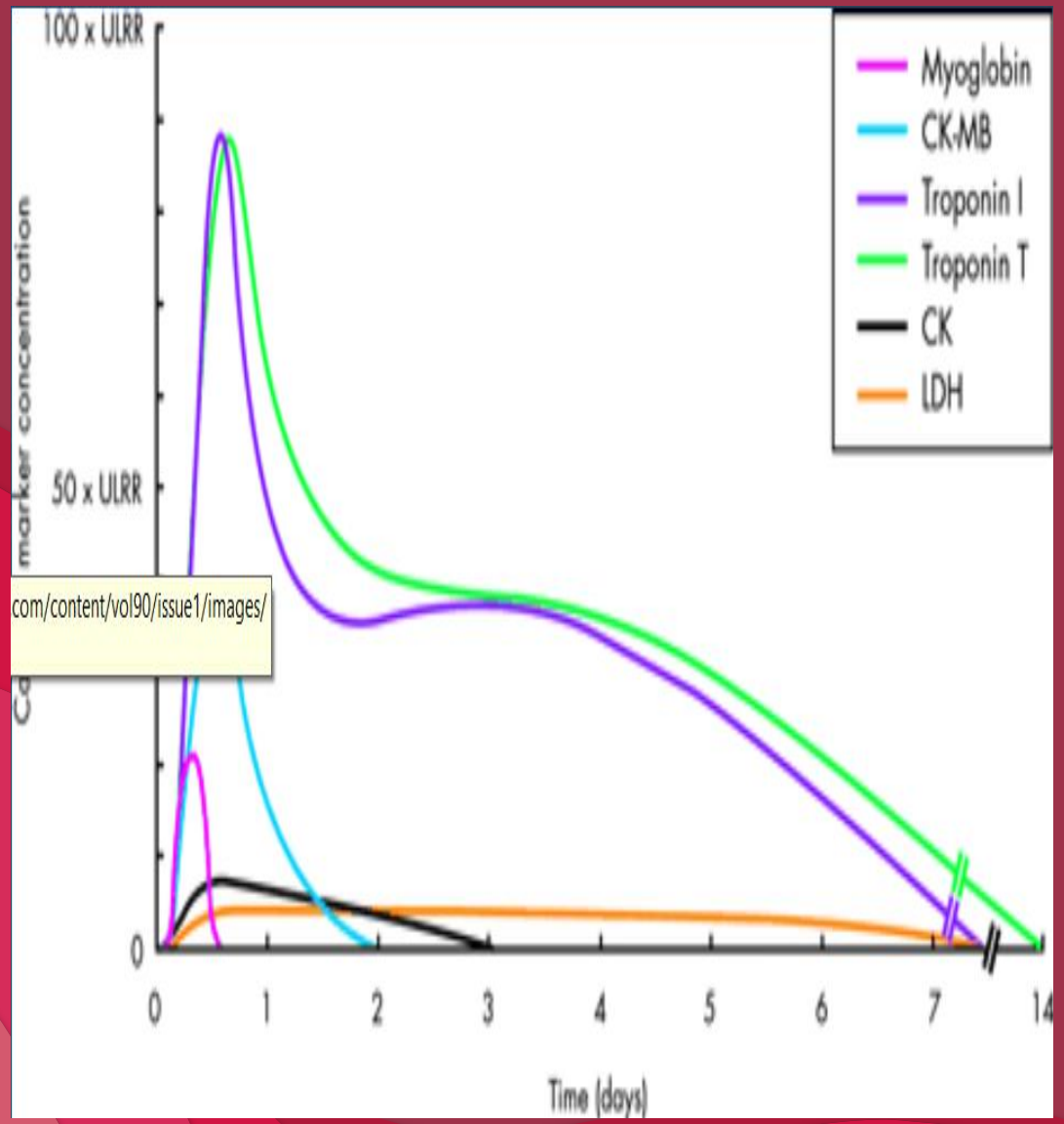


ECG Criteria for the Diagnosis of STEMI in LBBB

- >=3 had specificity of 98%
- Score of 0 doesn't exclude STEMI

Criterion	Location	Points
Concordant ST-segment elevation≥ 1 mm	Any lead with positive QRS deflection	5
Concordant ST-segment Depression≥ 1 mm	V1, V2, or V3	3
Disconcordant ST-segment elevation≥ 5 mm	Any lead with negative QRS deflection	2

Biochemical Markers



Protein	Molecular mass (kD)	First detection	Duration of detection	Sensitivity	Specificity
Myoglobin	16	1.5–2 hours	8–12 hours	+++	+
CK-MB	83	2–3 hours	1–2 days	+++	+++
Troponin I	33	3–4 hours	7–10 days	++++	++++
Troponin T	38	3–4 hours	7–14 days	++++	++++
CK	96	4–6 hours	2–3 days	++	++

Treatment of MI

- Immediate General Management: MONA

1- Morphine 2-3 mg IV: it causes nausea and vomiting; so, give antiemetic.

2- Oxygen 2-4 liters/min: if 02 sat <90%.

3- Nitroglycerin Sublingual or Spray.

4- Aspirin 160-325 mg Chewed, Swallowed or IV.

- In ER we give Clopidogrel or Ticagrelor is Aspirin is contraindicated.
- Reperfusion means opening the blocked vessel; PCI is preferred with success rate about 98%

IN EMERGENCY ROOM:

1-Rapid assessment

2-Establish IV access

3-12 ECG

4- Aspirin 150-300 mg Orally, Clopidogrel or ticagrelor

5-Oxygen

6-Analgesia: IV morphine, diamorphine 3-5 mg

7-Antiemetic: metoclopromide 10 mg IV

8-Sublingual nitrate: if NO hypotension, RV MI

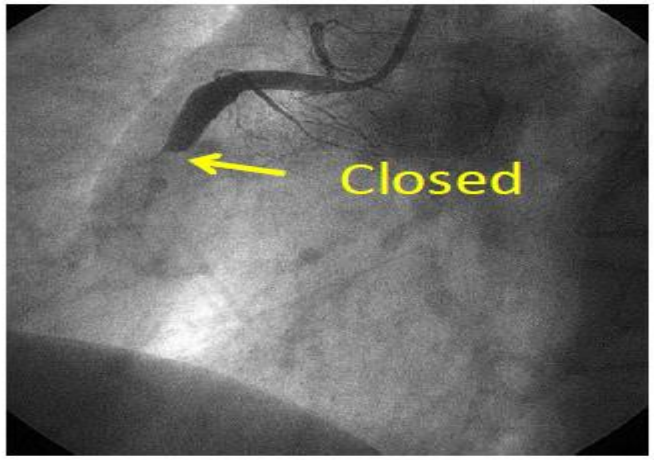
9-ECG monitor

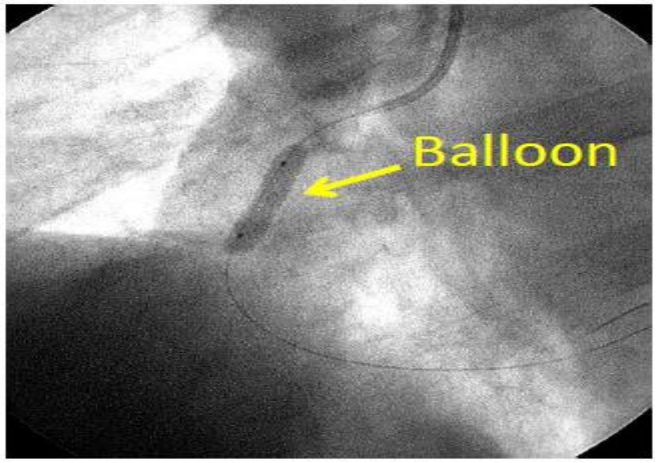
10-Reperfusion: PCI or Thrombolytics, (CABG)

Primary Angioplasty

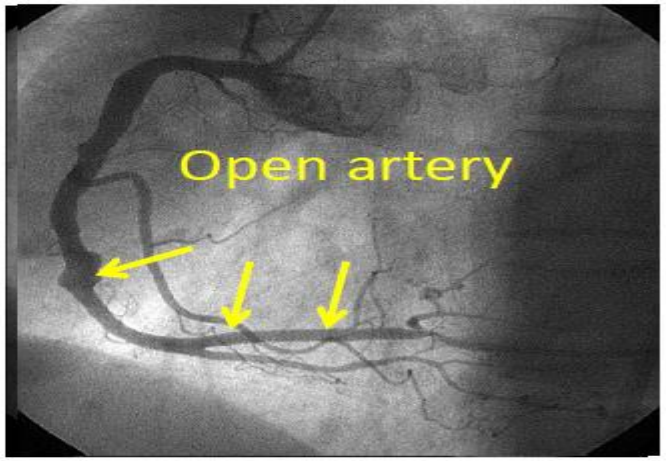
- Coronary arteries: balloon angioplasty

Arrival



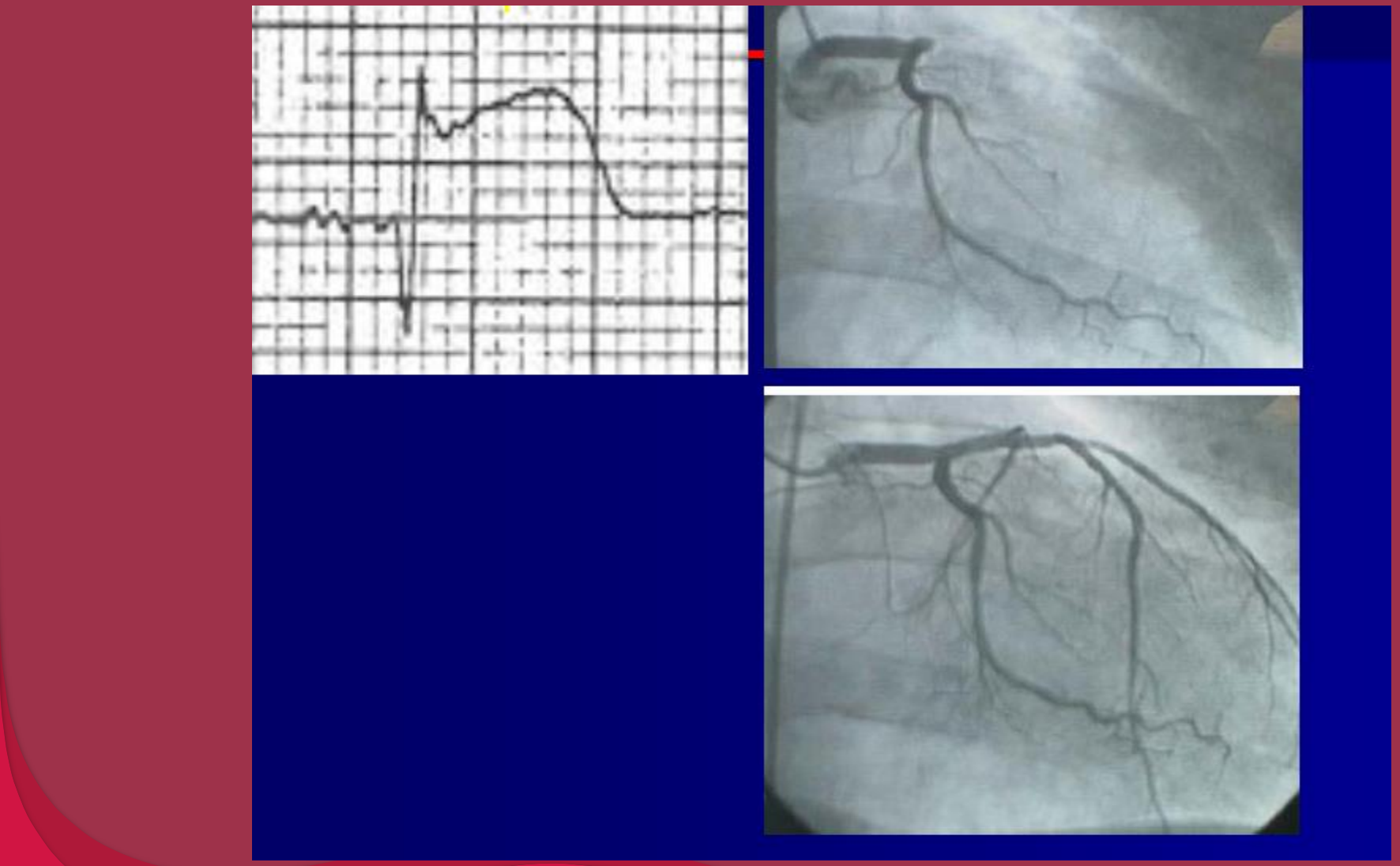


After balloon

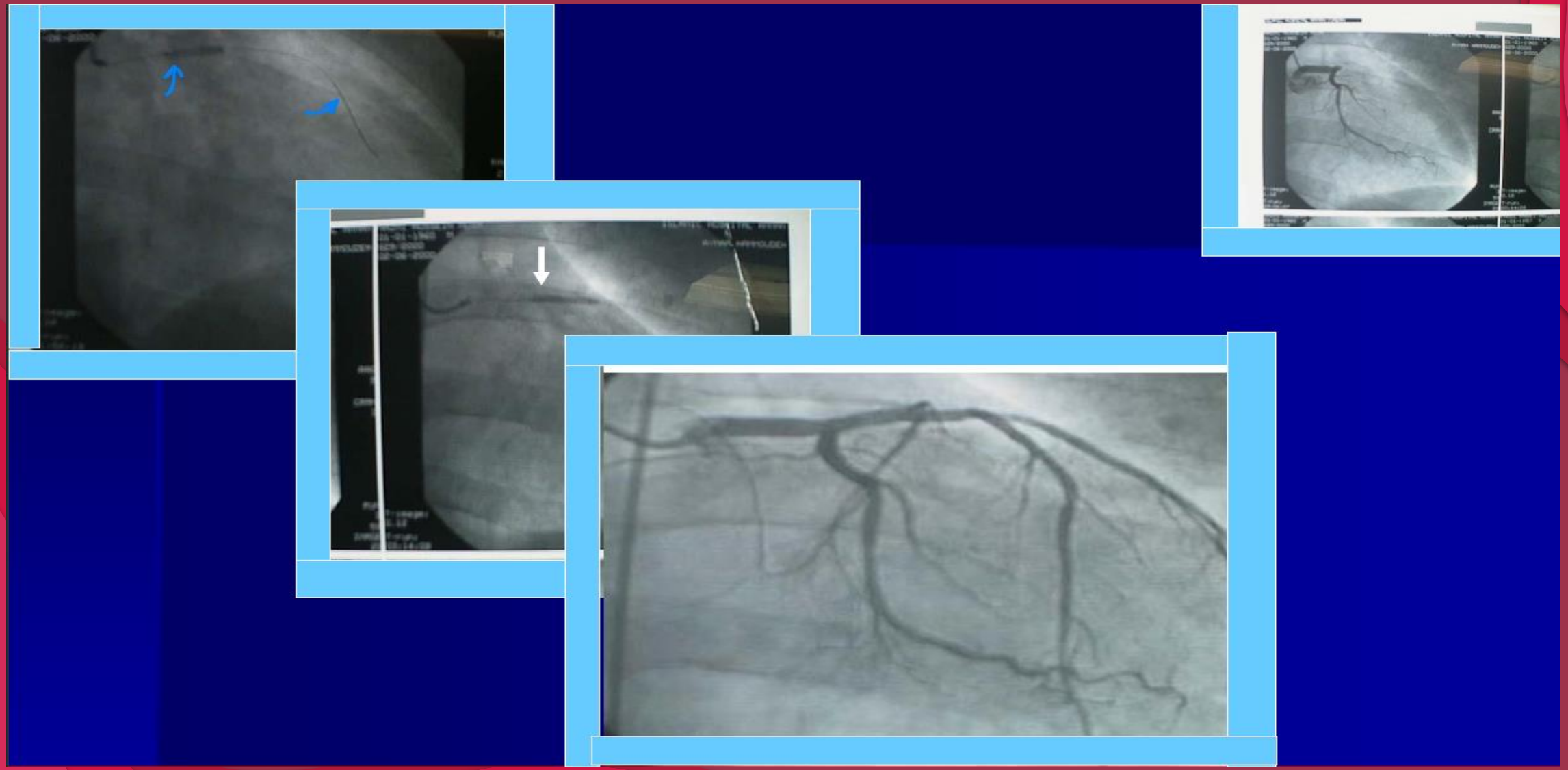


- The European Society of Cardiology (ESC) guidelines recommend primary PCI as the preferred treatment whenever it is available within 90-120 minutes of the first medical contact

Reperfusion in STEMI



Reperfusion: PCI



Indications for Thrombolytic Therapy

1-ST-elevation:

- 2 adjacent leads
- > 1mm in limb leads (L1, L11, L111, AVF,AVL)
- > 2mm in precordial leads (V1-V4)

OR

2- New Left Bundle Branch Block (LBBB)

Common Thrombolytic Regimens for STEMI

- After giving thrombolytic therapy, it should be followed by an ECG + History and Evaluation after 60-90 mins → if ECG remains abnormal with ST elevation, then this means that the vessel is still blocked, and the patient must be referred to any hospital with a Cath Facility to have a PCI and preserve the myocardium

	Initial treatment	Co-therapy	Contraindications
Streptokinase (SK)	1.5 million U in 100 mL 5% dextrose or 0.9% saline over 30–60 min	None or iv heparin x 24–48 hours	Prior SK or anistreplase
Alteplase (tPA)	15 mg iv bolus, then 0.75 mg/kg over 30 min, then 0.5 mg/kg iv over 60 min Total dose not over 100 mg	iv heparin x 24–48 hours	
Reteplase (rPA)	10 U + 10 U iv bolus given 30 min apart	iv heparin x 24–48 hours	
Tenecteplase**** (TNK-tPA)	Single iv bolus 30 mg if <60 kg 35 mg if 60 kg to <70 kg 40 mg if 70 kg to <80 kg 45 mg if 80 kg to <90 kg 50 mg if ≥90 kg	iv heparin x 24–48 hours	

Routine Therapies in Acute STEMI

- Clopidogrel must be continued for at least 1 year

- ASA 150–325 mg (non-enteric coated), Clopidogrel
- Beta-blockers
- Angiotensin-converting enzyme (ACE) inhibitors
- Oxygen
- statines
- Nitrates
- Heparin if indicated
- CCU: 24-48 hr
- Word: 3-5 days
- Home medication: aspirin, B-blocker, statines, ACE I, ? nitrate

TIMI Risk Score in STEMI

Risk factor	Score
1- Age>65	2
2- Age>75	3
3- Hist of angina	1
4- Hist of hypertension	1
5- Hist of DM	1
6- Syst BP< 100	3
7- Heart rate> 100	2
8- Killip II-IV	2
9- Ant M or LBBB	1
10- Delay treat > 4 hr	1

Total Score	Risk of death at 30 days(%)
0	0.8
1	1.6
2	2.2
3	4.4
4	7.3
5	12.4
6	16.1
7	23.4
8	26.8
9-16	35.9

Post-MI Management

1- Risk factors modification (Stop smoking, BP< 140/90, HbA1c<7, Exercise, ..)

2-Aspirin, Clopidogrel or ticagrelor

3- B-blockers

4-Statines

5-ACE-inhibitors

6- Aldosterone antagonist(in presence of heart failure)

Unstable Angina

- Classified into:

1- Acute Vs Subacute

Acute → Rest pain within the last 48 hours

Subacute → No pain within the last 48 hours

2- Primary Vs Secondary

Primary → No secondary causes

Secondary → Severe Anemia, Thyrotoxicosis, HTN, Arrythmia

3- High Risk Vs Low Risk

Definition:

1-New onset angina < 8 weeks

2- Angina at rest or minimal exersion

3-Crescendo angina: patient with chronic angina with increasing frequency, duration, or intensity of chest pain

4-Post MI or Revascularization angina: 2 weeks

Types:

Pathophysiology: plaque erosion or rupture, vasoconstriction, distal embolisation

Diagnosis: Clinical, ECG , *Negative cardiac markers*

High Risk Unstable Angina

1-Rest pain > 20 minutes

2-Accelerating tempo of ischemic symptoms in preceding 48 hr

3-Clinical finding of: pulmonary edema, new S3, new MR, Hypotension, Brady or Tachycardia

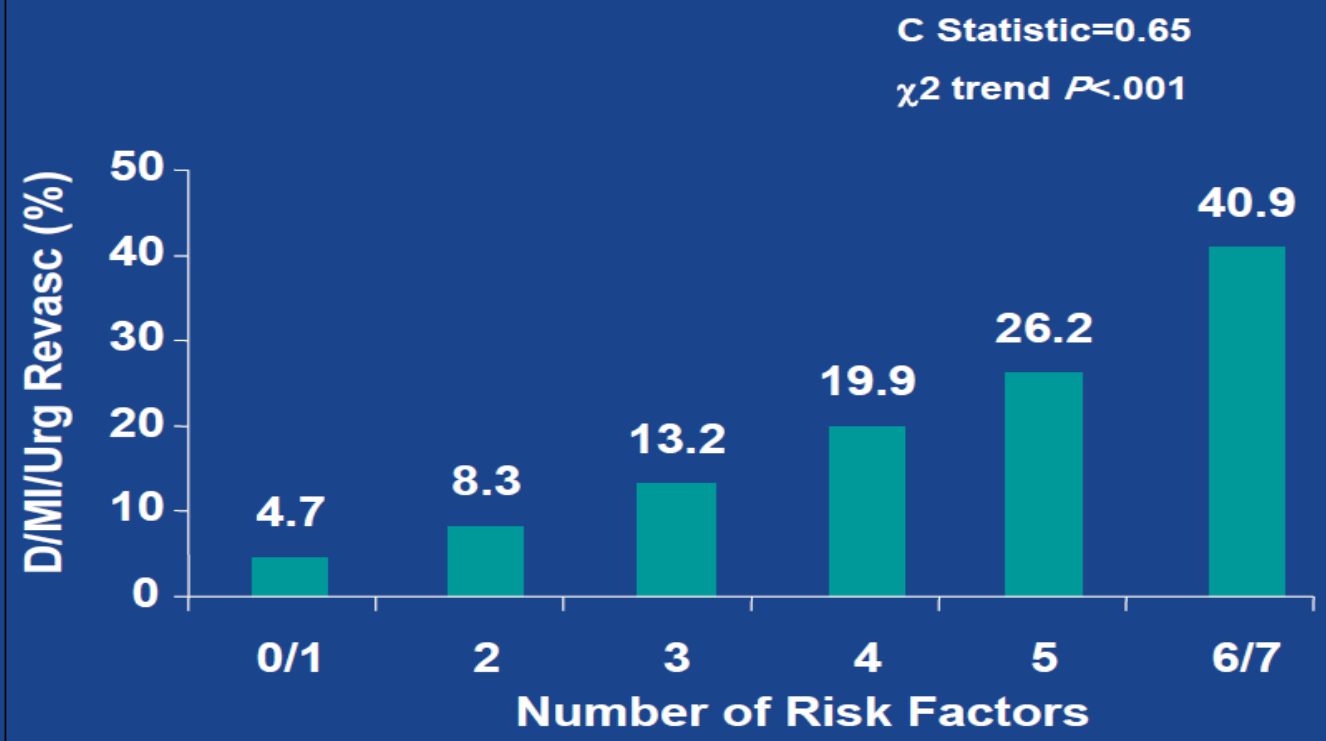
3-ECG changes: transient ST segment changes, BBB, VT

4- DM

TIMI Score for Unstable Angina / NSTEMI

TIMI Risk Score For UA/NSTEMI

- Age ≥65 years
- ≥3CAD Risk Factors
- Prior Stenosis >50 %
- ST deviation
- ≥2 Anginal events ≤24 hours
- ASA in last 7 days
- Elevated Cardiac Markers (CK-MB or troponin)



Population (%):	4.3	17.3	32.0	29.3	13.0	3.4

Treatment of High-Risk Unstable Angina and NSTEMI

- If the patient is stable → Wait 24 hours, then do Cath.
- If the patient is unstable → Do Cath immediately.

1-CCU admission : Treat as MI except for thrombolytics

NO THROMBOLYTICS

2-Aspirin***, Clopidogrel

3-Anticoagulant: heparin (LMWH is superior to unfractionated heparin)***

4- Nitrate (S/L, oral, IV)

5-B-blocker

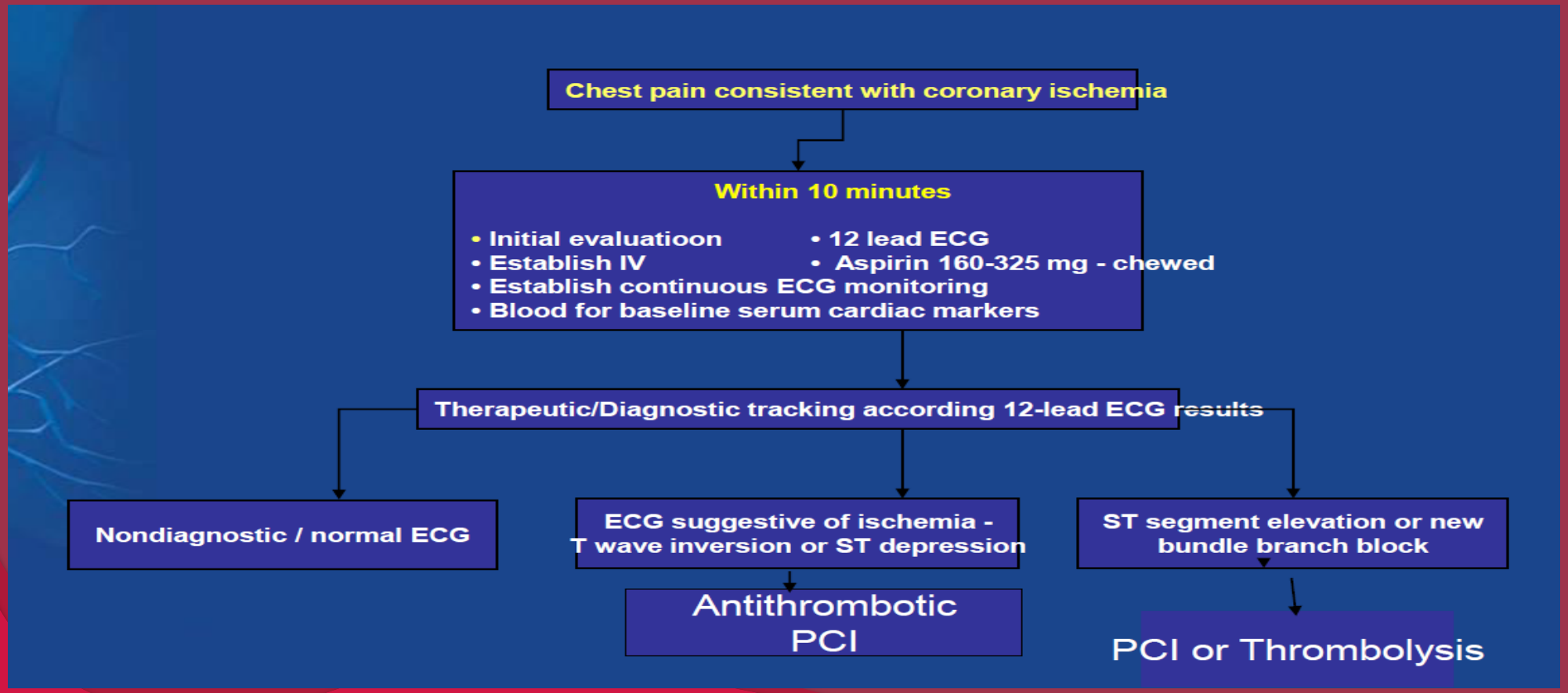
6-clopidogrel, GP 11b,111a--------Cath PCI(angio)

7-Statines

8- Invasive or conservative management

*** *improve prognosis*

Algorithm for Initial Assessment and Evaluation of the patient with Acute Chest Pain



Infective Endocarditis

Infective Endocarditis: Prophylaxis

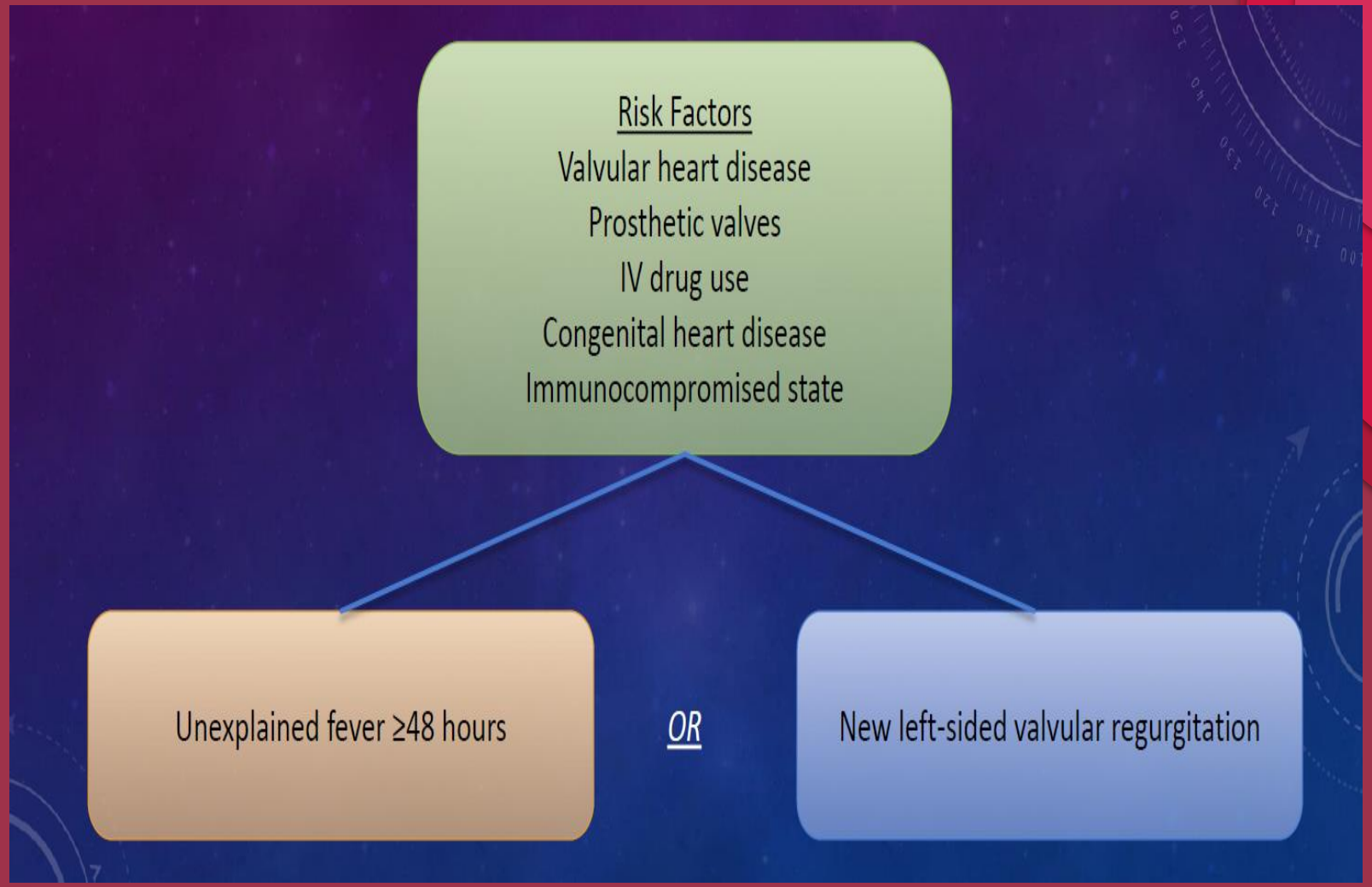
- Prophylaxis should be started in patients at high risk of complications, like Prosthetic Cardiac Valves, Transcatheter valves, Prosthetic material used for valve repair, Previous IE, Transplant recipient with valvulopathy, Congenital Heart disease, Unrepaired cyanotic lesions, Cyanotic lesions with palliative shunts or conduits, Repair <= 6 months ago with prosthetic material, Repaired lesions with residual defects.
- Patients that DON'T require prophylaxis: Mitral prolapse with regurgitation or thickened leaflets, Acquired valvular heart disease, Prior Rheumatic fever, Hypertrophic cardiomyopathy, Bicuspid aortic valve, PDA, VSD, Primum ASD, Aortic Coarctation.
- Procedures that require prophylaxis: Dental Procedures (Manipulation of gingival tissue or root of teeth + Perforation of oral mucosa + Cleaning, Extraction, Root canal), Incision into active skin or soft tissue infection, incision or biopsy in Respiratory tract (Tonsillectomy / Adenoidectomy, Bronchoscopy with biopsy).
- Procedures that DON'T require prophylaxis: Dental injections or X-rays, Placement or adjustment of orthodontic appliances, Bleeding from Trauma to lips or oral mucosa, Shedding of deciduous teeth, Bronchoscopy without biopsy, GI or GU procedures without active infection.

Drugs for Prophylaxis

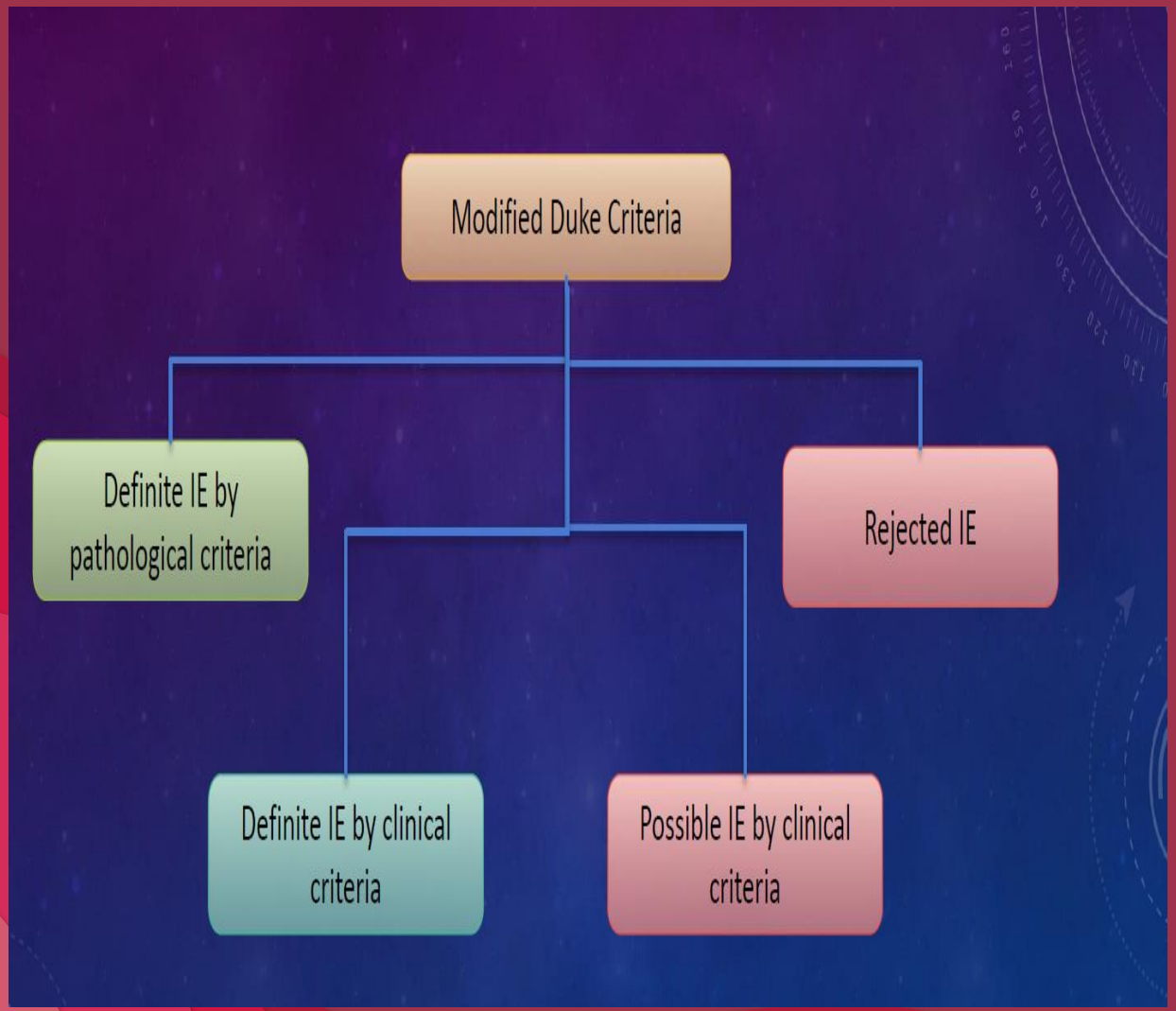
Situation	Drug	Dose
Oral	Amoxicillin	2 grams PO
Unable to take oral	Ampicillin	2 grams IM or IV
	Cefazolin	1 gram IM or IV
	Ceftriaxone	1 gram IM or IV
β-lactam allergy	Clindamycin	600 mg PO
	Azithromycin	500 mg PO
	Clarithromycin	500 mg PO
β-lactam allergy AND unable to take oral	Clindamycin	600 mg IM or IV

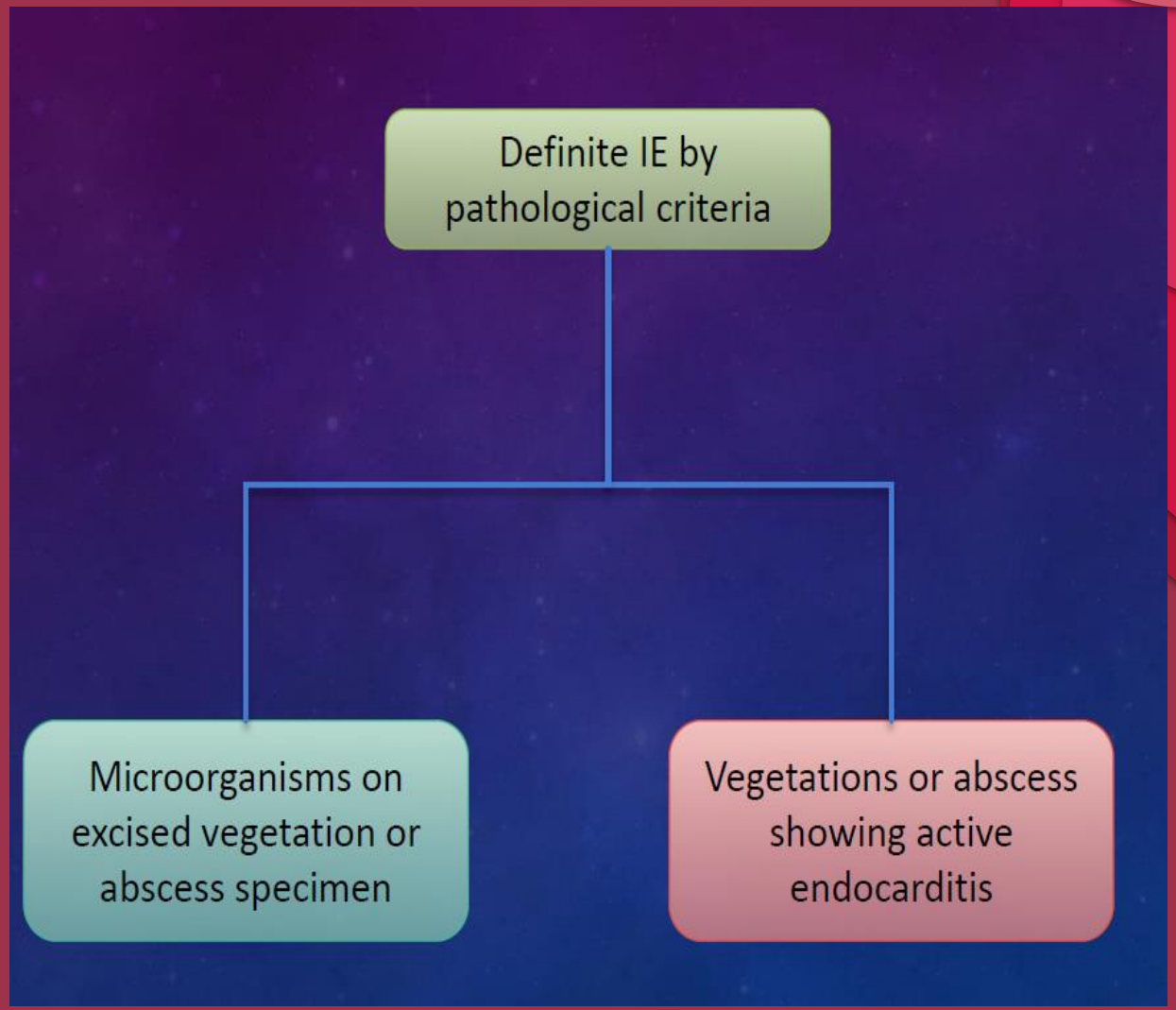
IE: Active Infection

- When should you suspect infective endocarditis ??



IE: Diagnosis



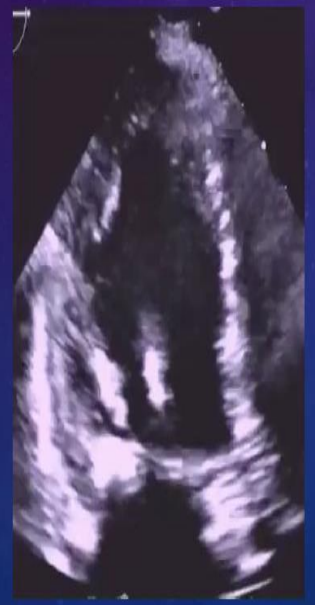


IE: Diagnosis

IE: Diagnosis

Diagnosis

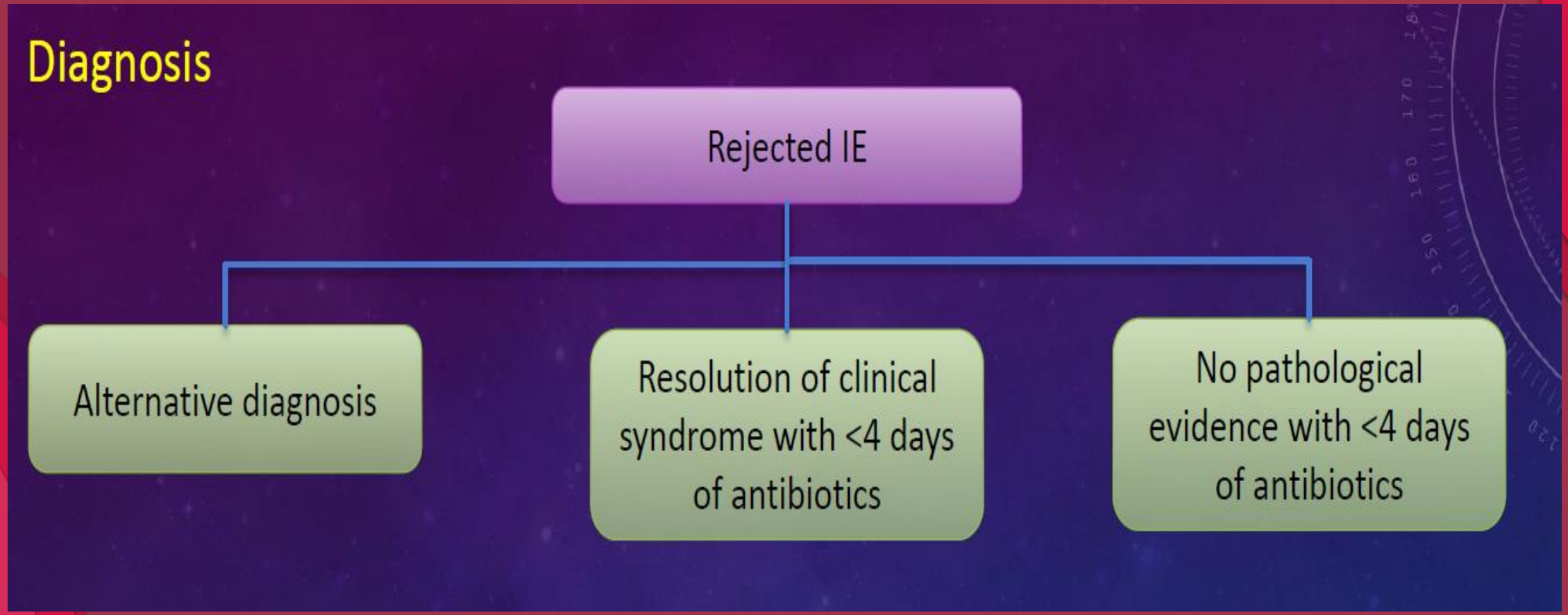
- Major clinical criteria
  - Multiple blood cultures positive
  - Single positive culture for *Coxiella burnetti*
  - Endocardial involvement
    - Vegetation, abscess, dehiscence
    - New regurgitation



Diagnosis

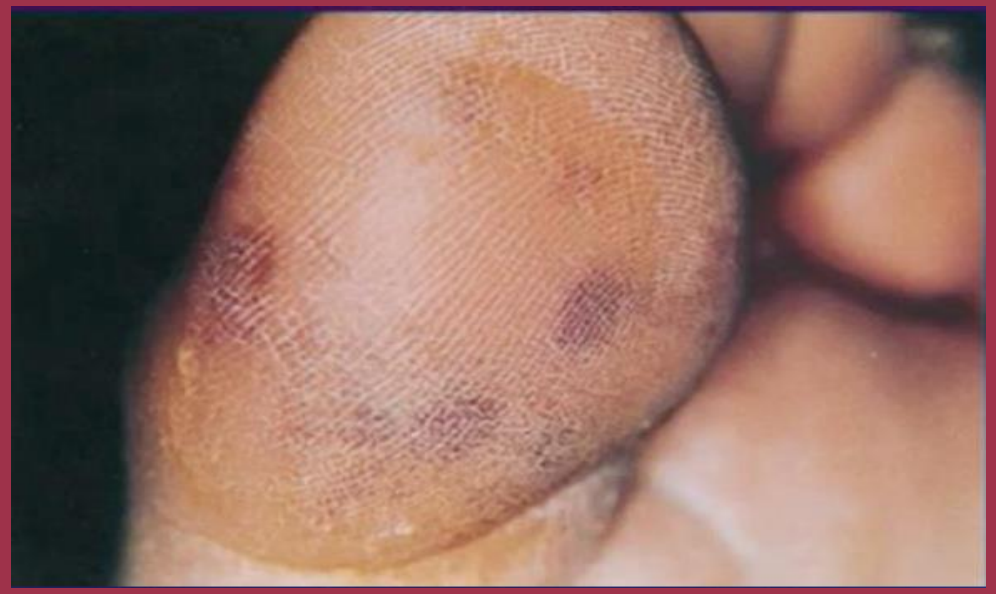
- Minor clinical criteria
  - Clinical predisposition
  - Fever
  - Vascular phenomena
  - Immunological phenomena
  - Blood culture or serological evidence of active infection NOT meeting major criteria

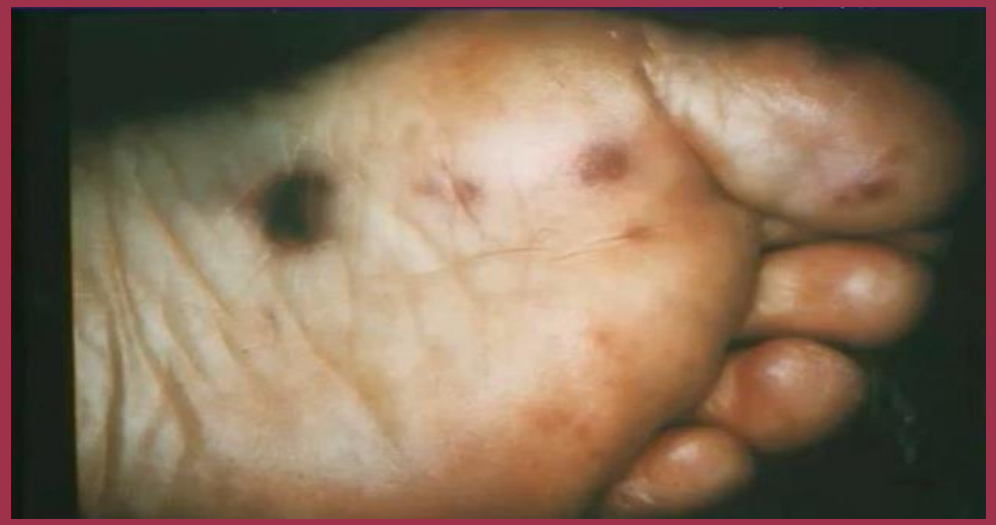
IE: Diagnosis



IE: Vascular Phenomena

- Janeway lesions
- Painless septic emboli
- Irregular, nontender, hemorrhagic macules, located on the palms, soles, thenar and hypothenar eminences of the hands and planter surfaces of the toes.
- They last for Days to Weeks.



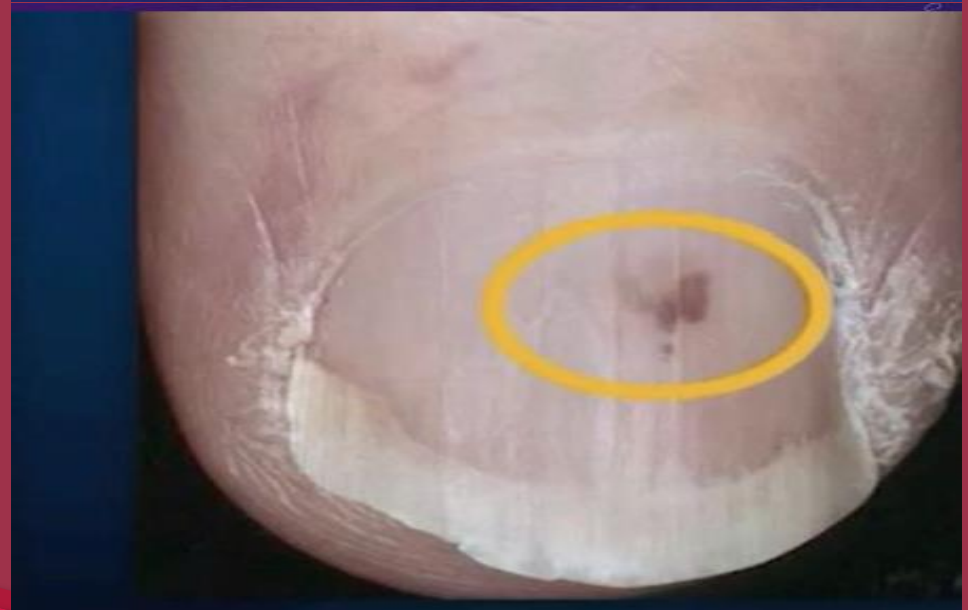


IE: Vascular Phenomena

- Conjunctival Hemorrhage

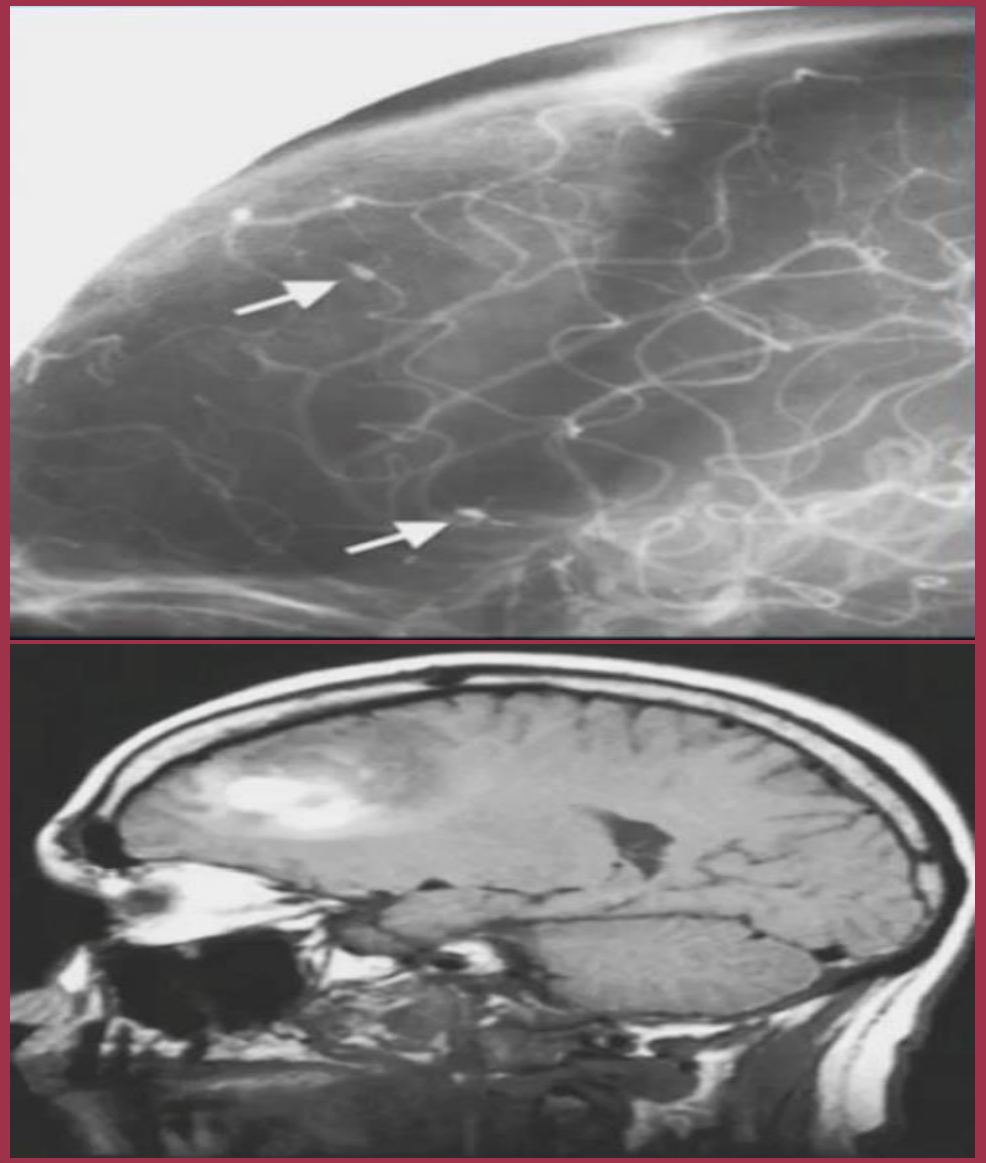
IE: Vascular Phenomena

- Splinter Hemorrhage
- Tiny blood spots that appear underneath the nail.
- It occurs when blood vessels along the nail bed are damaged and burst.
- The first picture shows Traumatic Splinter hemorrhage → At the tip of the nail.
- The second picture shows Embolic Splinter hemorrhage → Anywhere else on the nail.



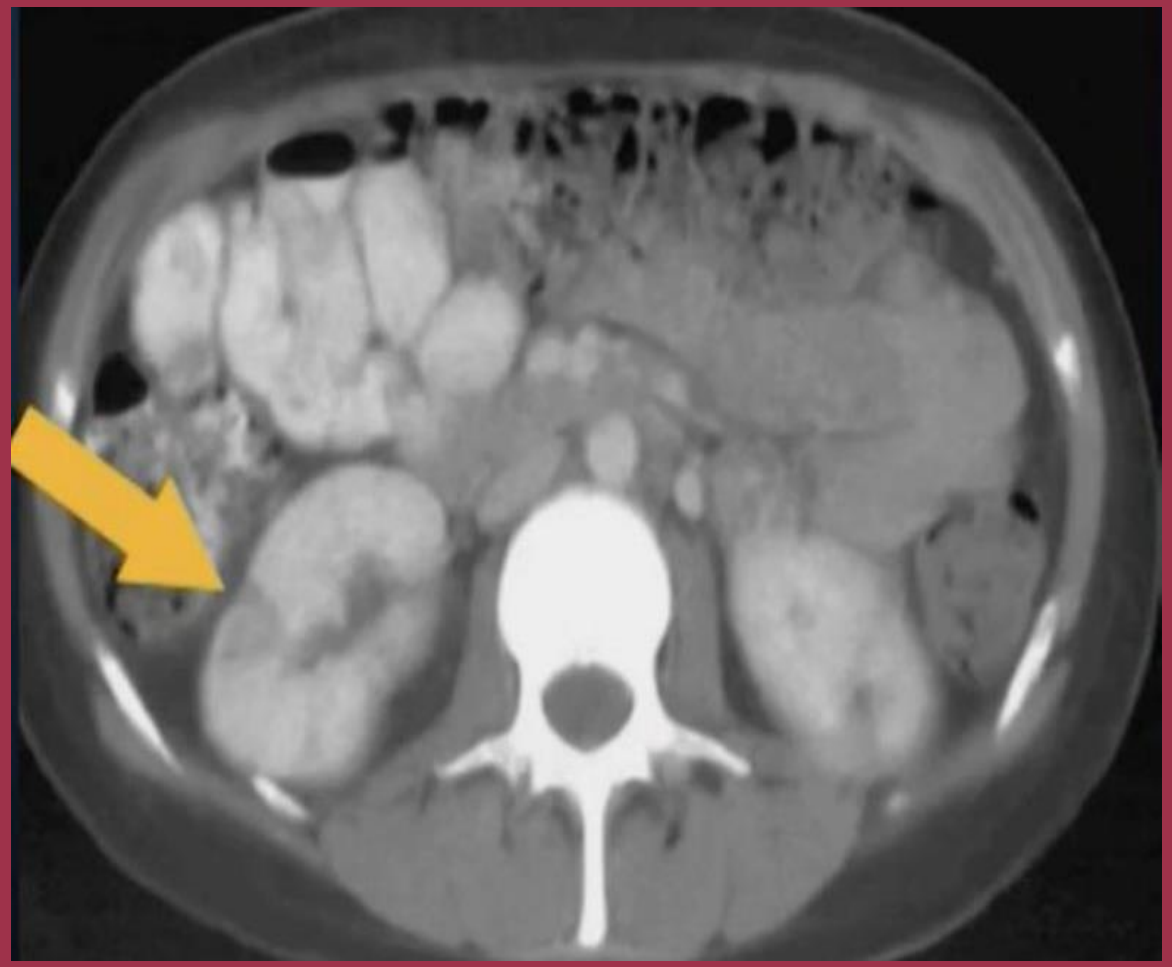
IE: Vascular Phenomena

- Cerebral involvement
- The first picture → Mycotic Aneurysm
- The second picture → Cerebral Hemorrhage.



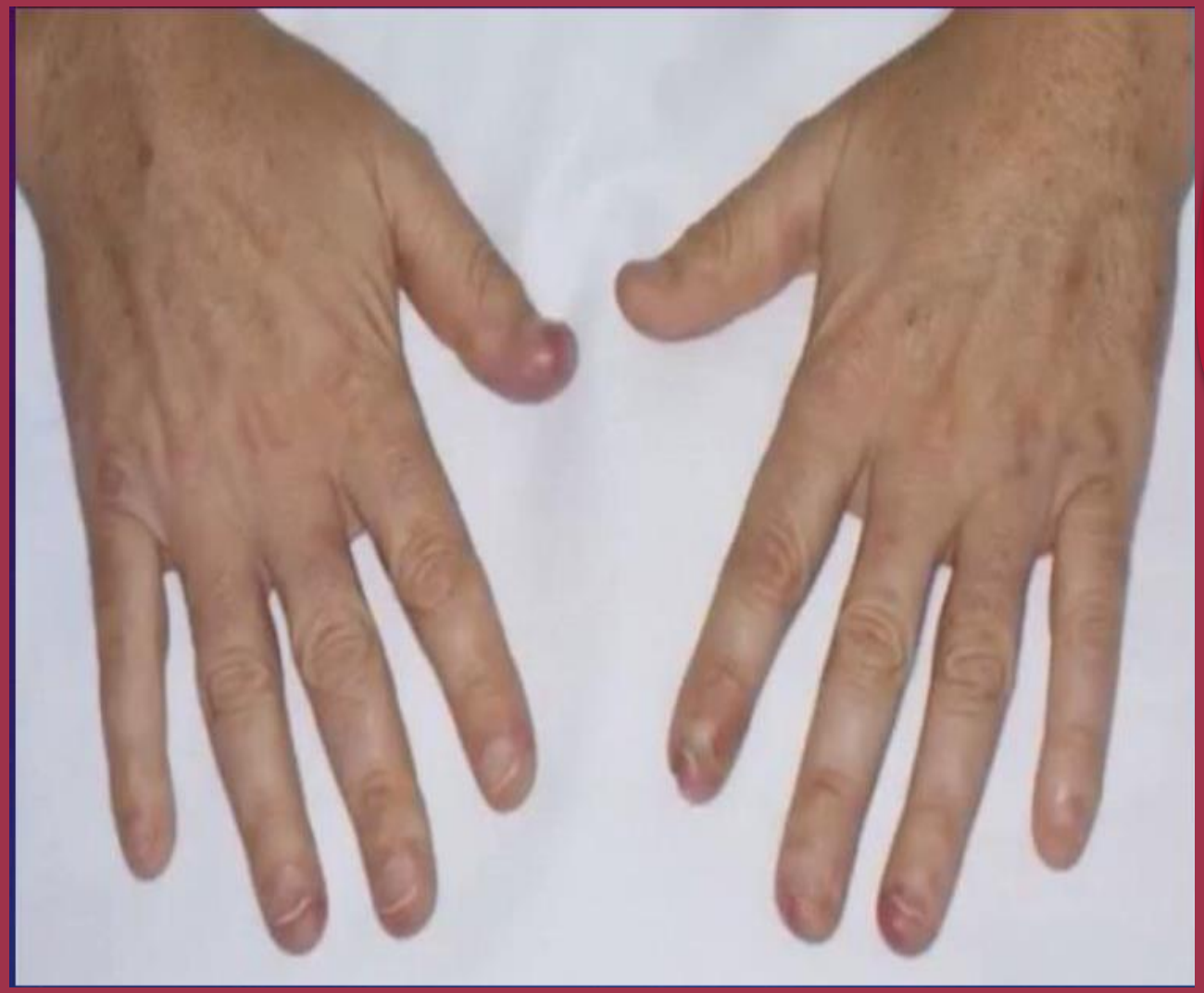
IE: Vascular Phenomena

- Embolic infarcts.
- This CT scan shows Renal infarcts.



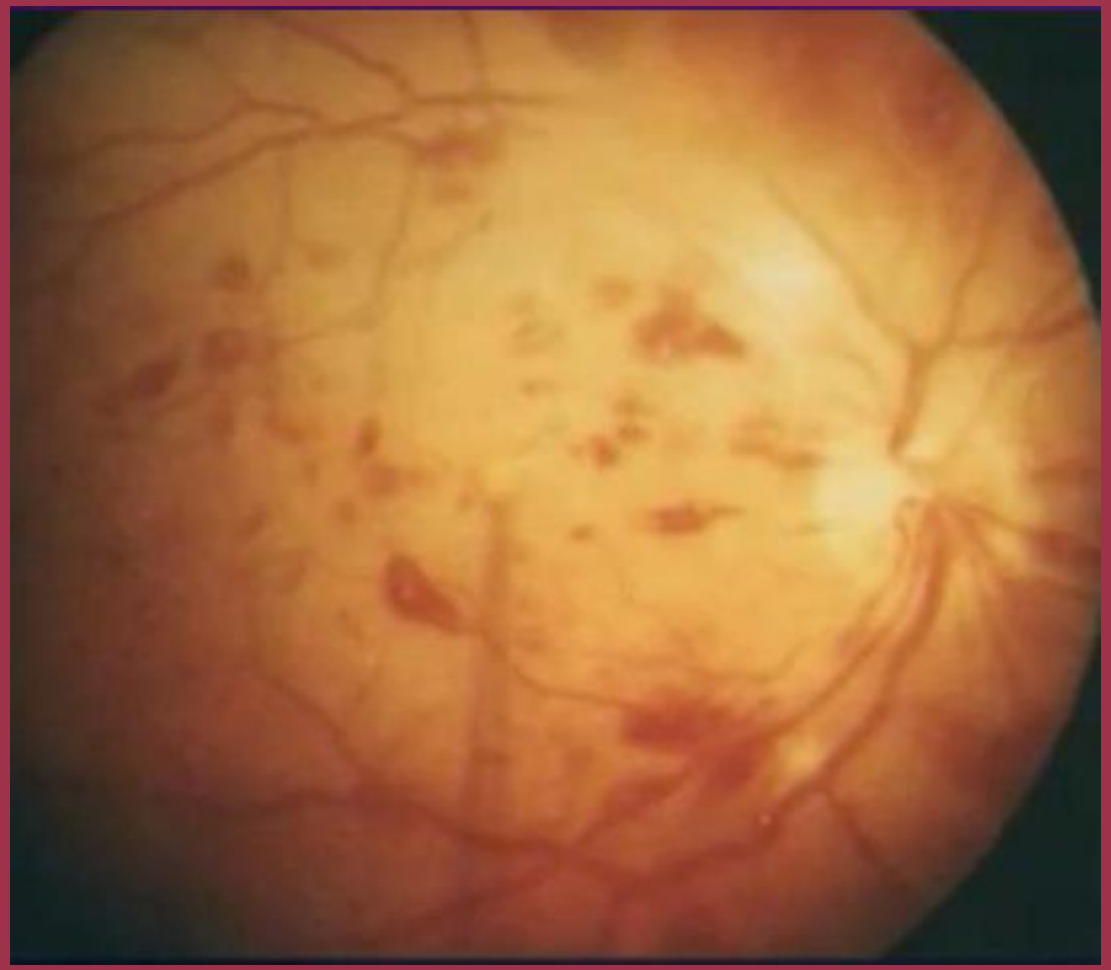
IE: Immunologic Phenomena

- Osler's Nodes
- Small, tender, transient nodules on the pads of the fingers and toes, and the palms and the soles.
- Caused by immune complex deposition leading to Necrotizing Vasculitis.



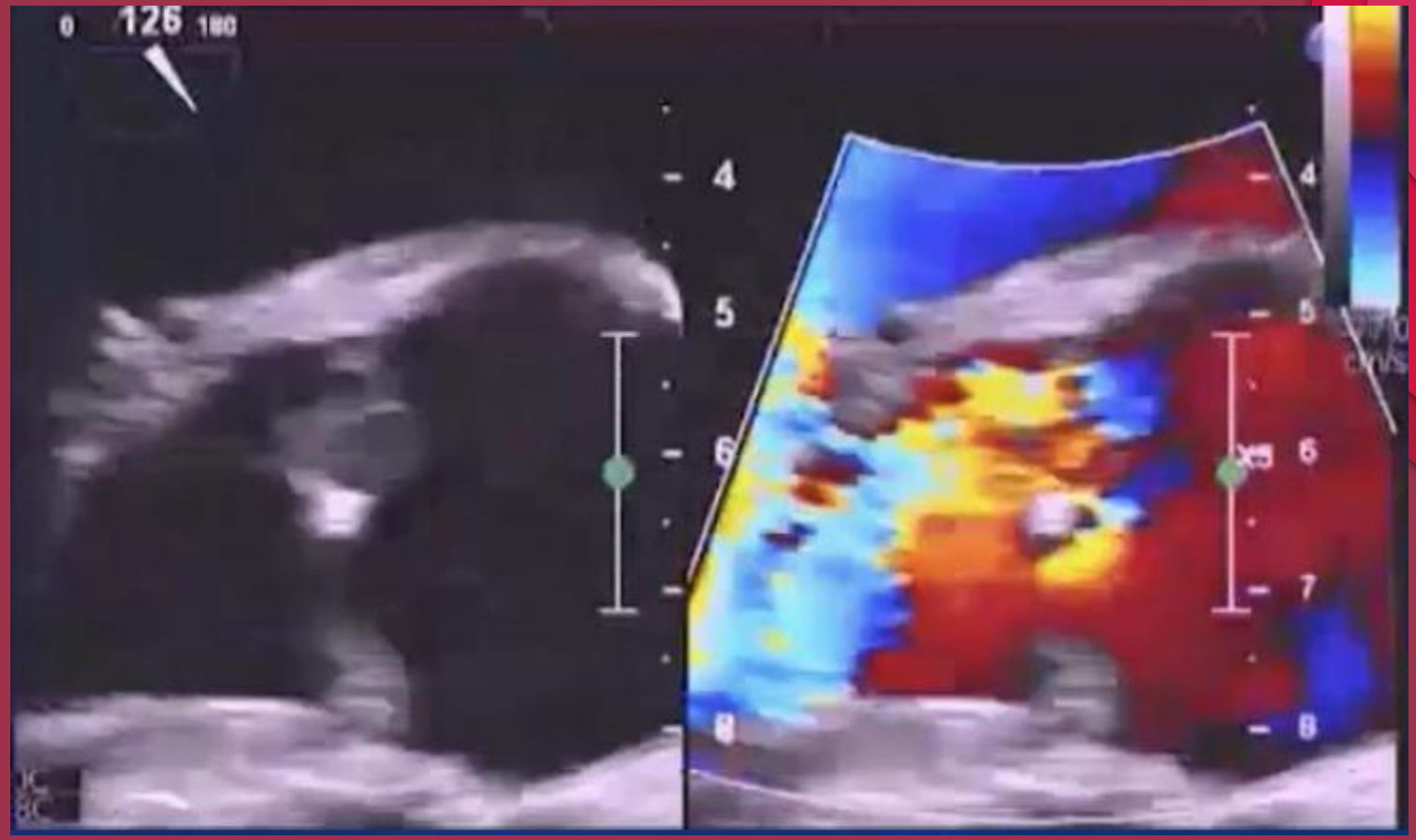
IE: Immunologic Phenomena

- Roth Spots
- Exudative retinal lesions with Pale center.
- Immune-mediated vasculitis



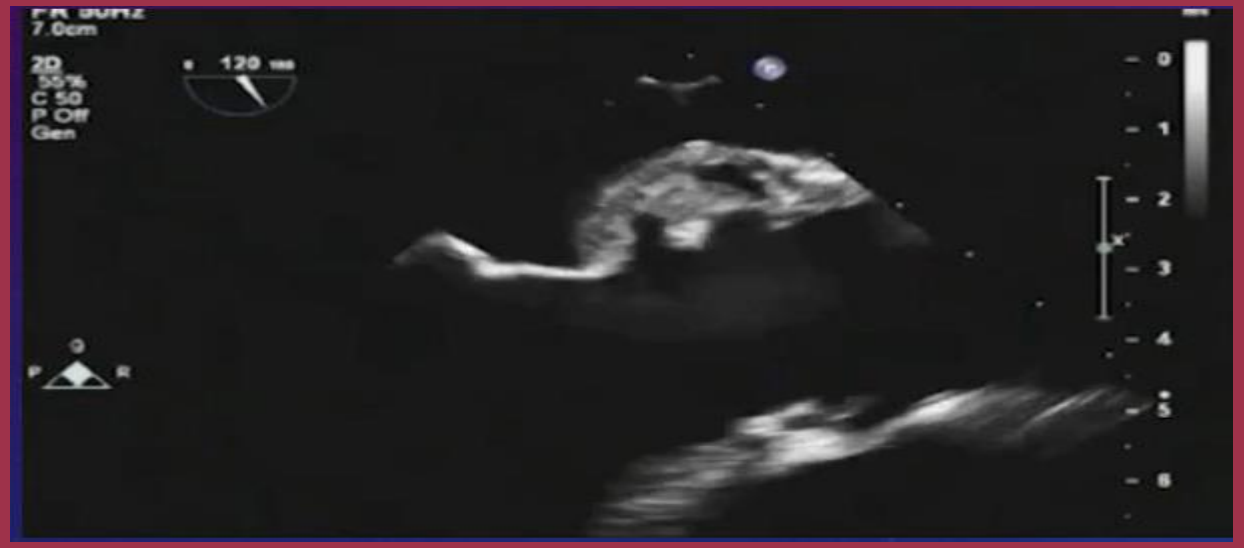
IE: Complications

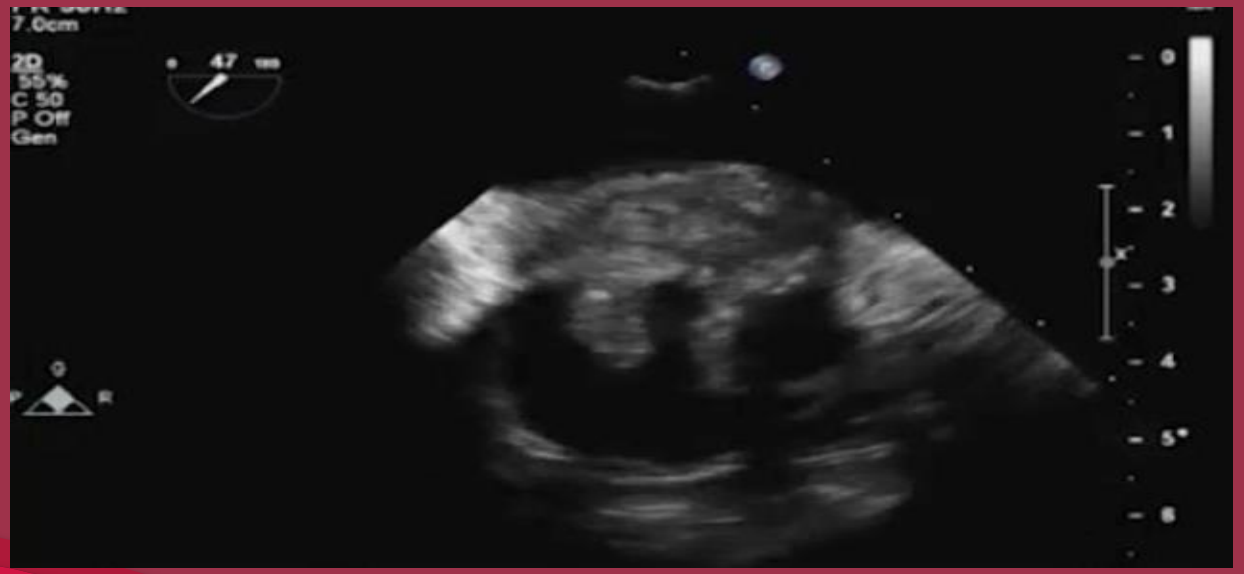
- Valvular Destruction / HF



IE: Complications

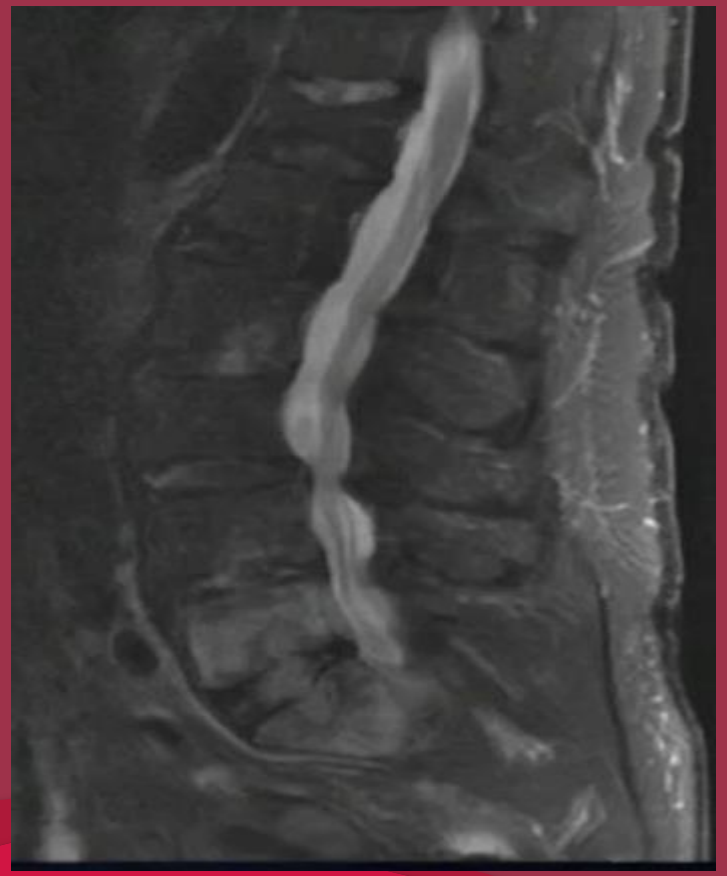
- Perivalvular Extension.

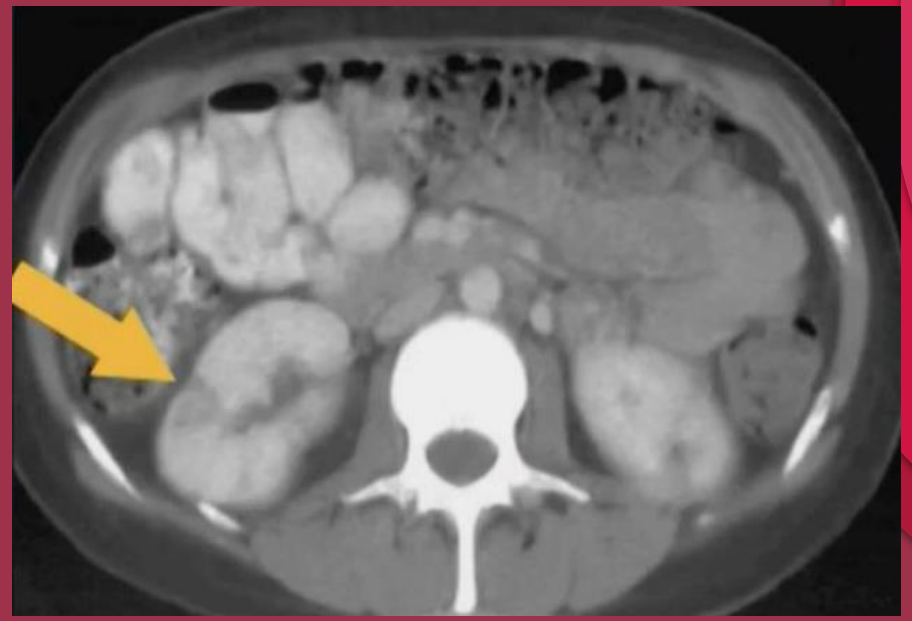




IE: Complications

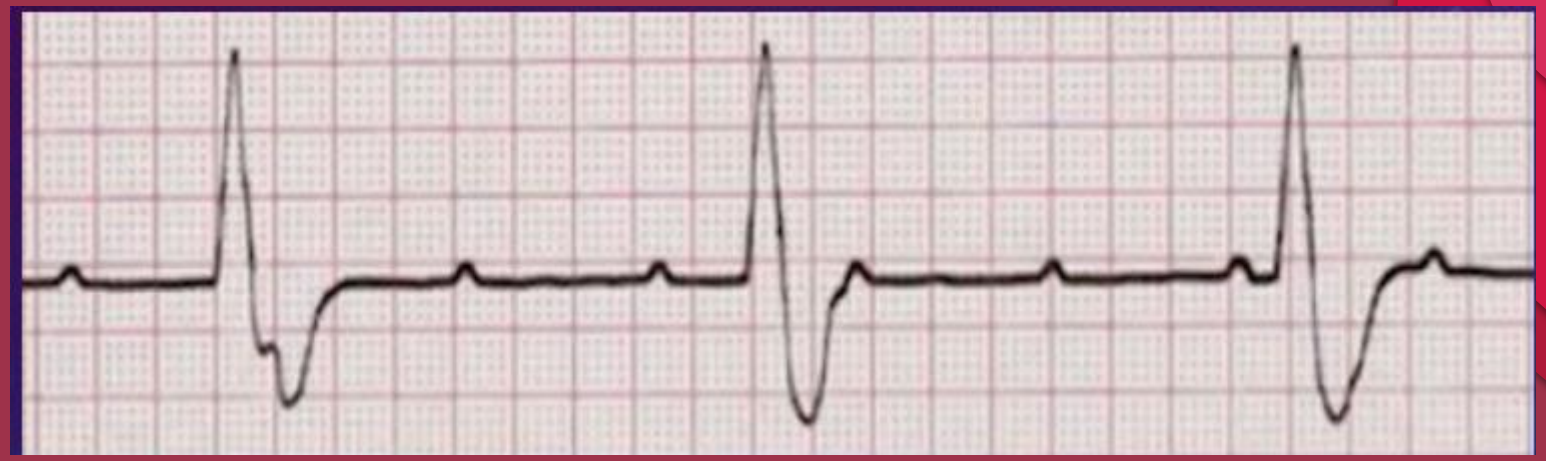
- Embolism / Metastasis





IE: Complications

- Electric Abnormalities.
- This ECG Shows Complete heart block



IE: Antimicrobial Therapy

	Viridans group streptococci		*Staphylococcus*	*Enterococcus*
	Penicillin susceptible	Penicillin resistant		
Native valve	4 weeks Penicillin OR Ceftriaxone 2 weeks Above + Gentamicin	4 weeks Penicillin OR Ceftriaxone AND 2 weeks Gentamicin	6 weeks Naficillin OR Cefazolin OR Vancomycin	4-6 weeks Pen / Amp + Gentamicin 6 weeks Amp + Ceftriaxone
Prosthetic valve	6 weeks Penicillin OR Ceftriaxone ± 2 weeks Gentamicin	6 weeks Penicillin OR Ceftriaxone AND Gentamicin	6 weeks Above + Rifampin AND 2 weeks Gentamicin	6 weeks Pen / Amp + Gentamicin 6 weeks Amp + Ceftriaxone

IE: Anticoagulation

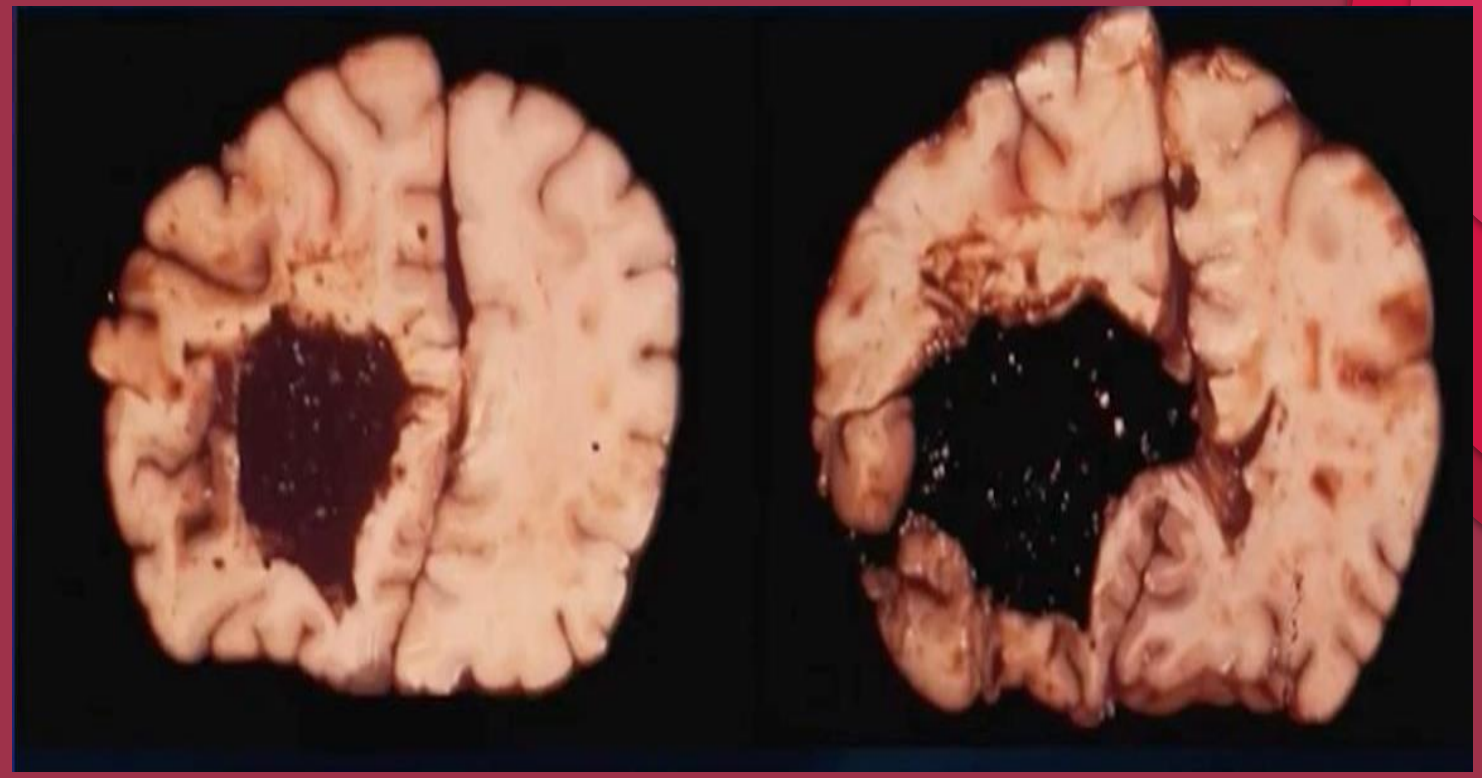
- Neurologic Complications in IE:

Septic Emboli → Ischemia → Hemorrhagic Transformation → Stroke

- Guideline recommendations:

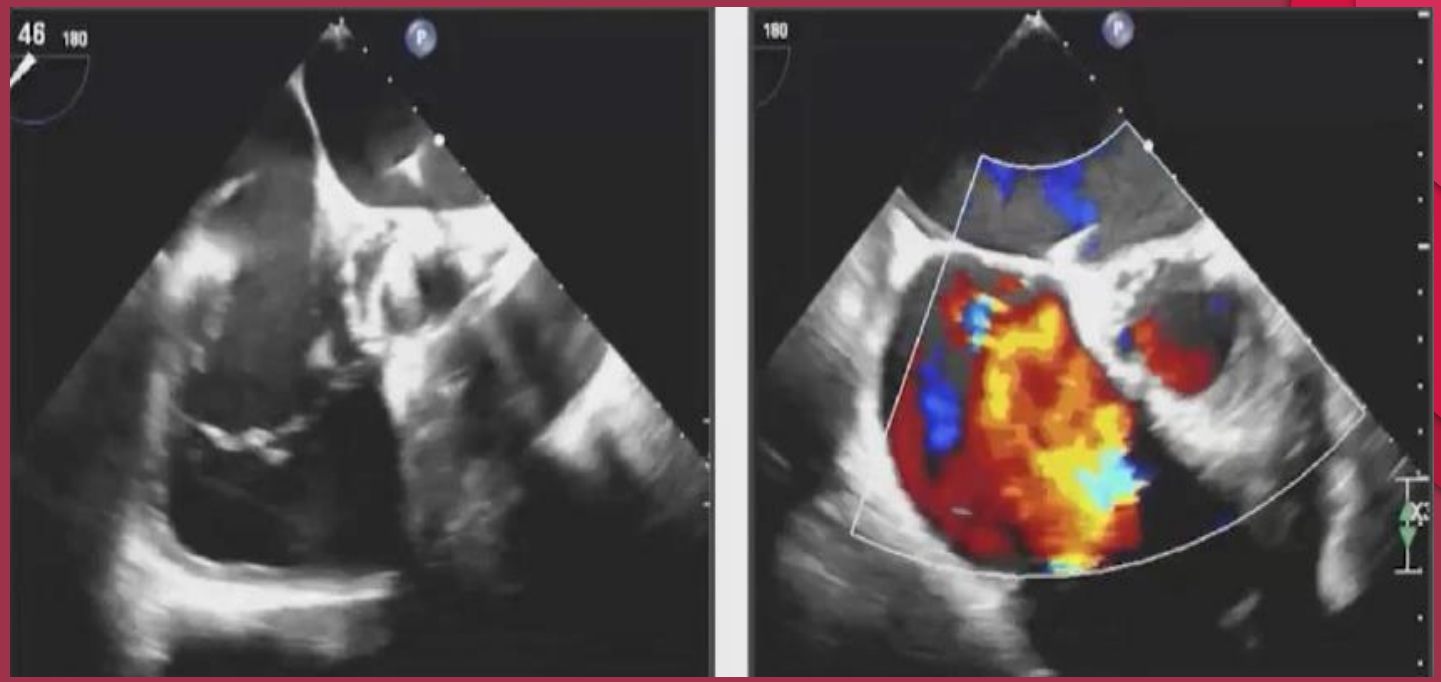
1- Discontinue all forms of anticoagulation in patients with mechanical valve infective endocarditis and CNS embolic event for >= 2 weeks.

2- Don't start aspirin or other antiplatelet agents as adjunctive therapy in IE



IE: Right-sided Endocarditis

- RV IE → S.aureus + IV drug use.
  - Medical :
    - Uncomplicated MSSA → Beta lactam for 2-6 weeks.
    - MRSA → Vancomycin for 6 weeks.
  - Surgical :
    - RV failure + Severe Tricuspid regurgitation + Decreased Treatment response
    - Prolonged infection + Resistant or Fungal organism
    - >= 20 mm vegetation + Recurrent PE despite treatment.



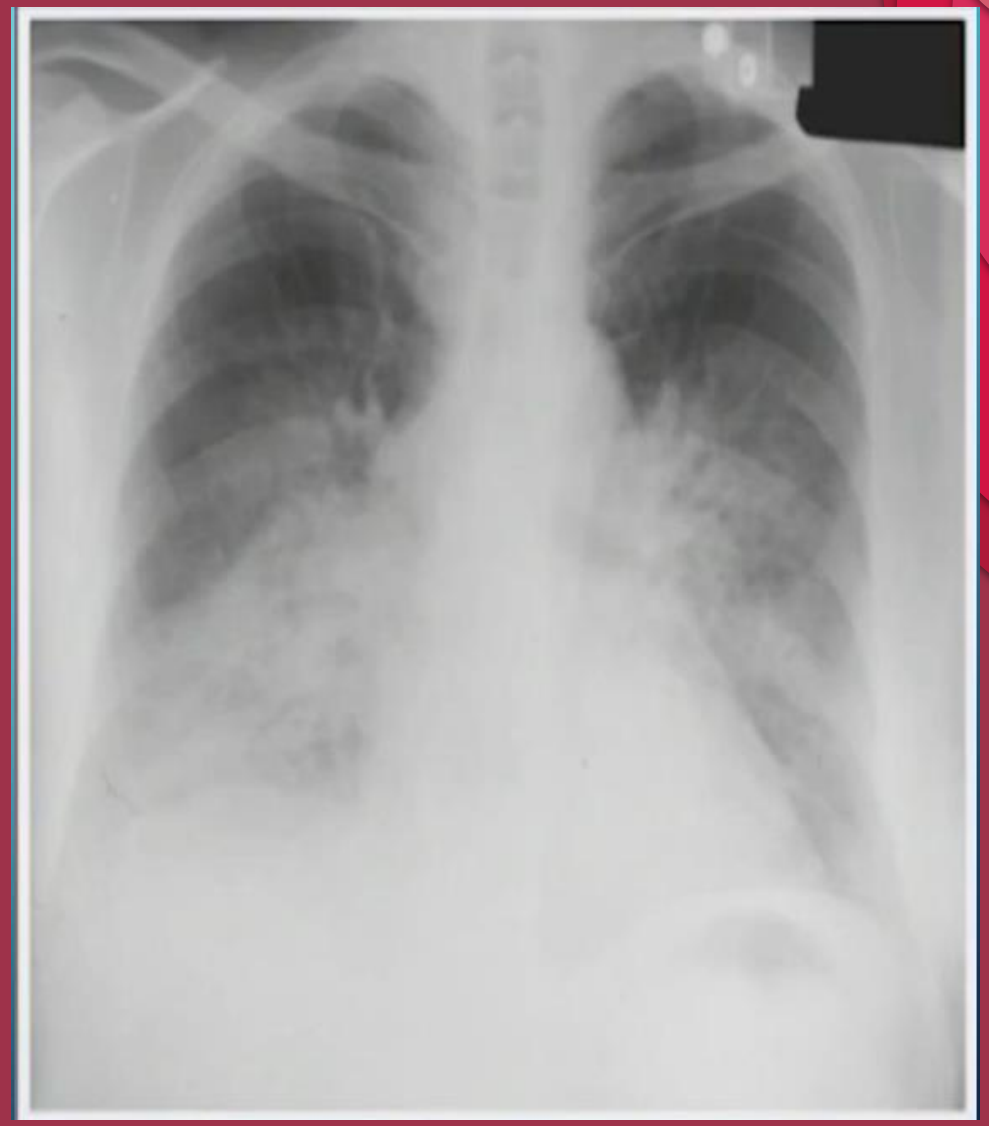
IE: Device Infection

- Account for ~1-5% of Device infections.
- Risk higher with ICDs than Permanent pacemakers.
- Risk factors: Immunosuppression, Co-morbid conditions, Anticoagulation use, Operator inexperience, Amount of hardware, Lack or pre-procedure prophylaxis, Device manipulation.
- Management: Suspect device infection (Fever, High WBCs and ESR, Erythema, Swelling, and Erosions at Generator site), Hx, Physical exam, Device interrogation, Blood cultures followed by antibiotics, Complete removal of the device.
- When might the device remain: Superficial infection at incision site, No pocket involvement, Bacteremia alone with all of the following (Clinical stability, Echo negative for lead involvement, No involvement of pocket or recent manipulation, No valvular involvement or endocarditis, Resolution of bacteremia with antibiotics).
- When can the device be re-implanted: Does the patient need a new device, Select new site preferably contralateral, Wait for negative blood cultures (72 hours after device removal / 14 days if valves involved).

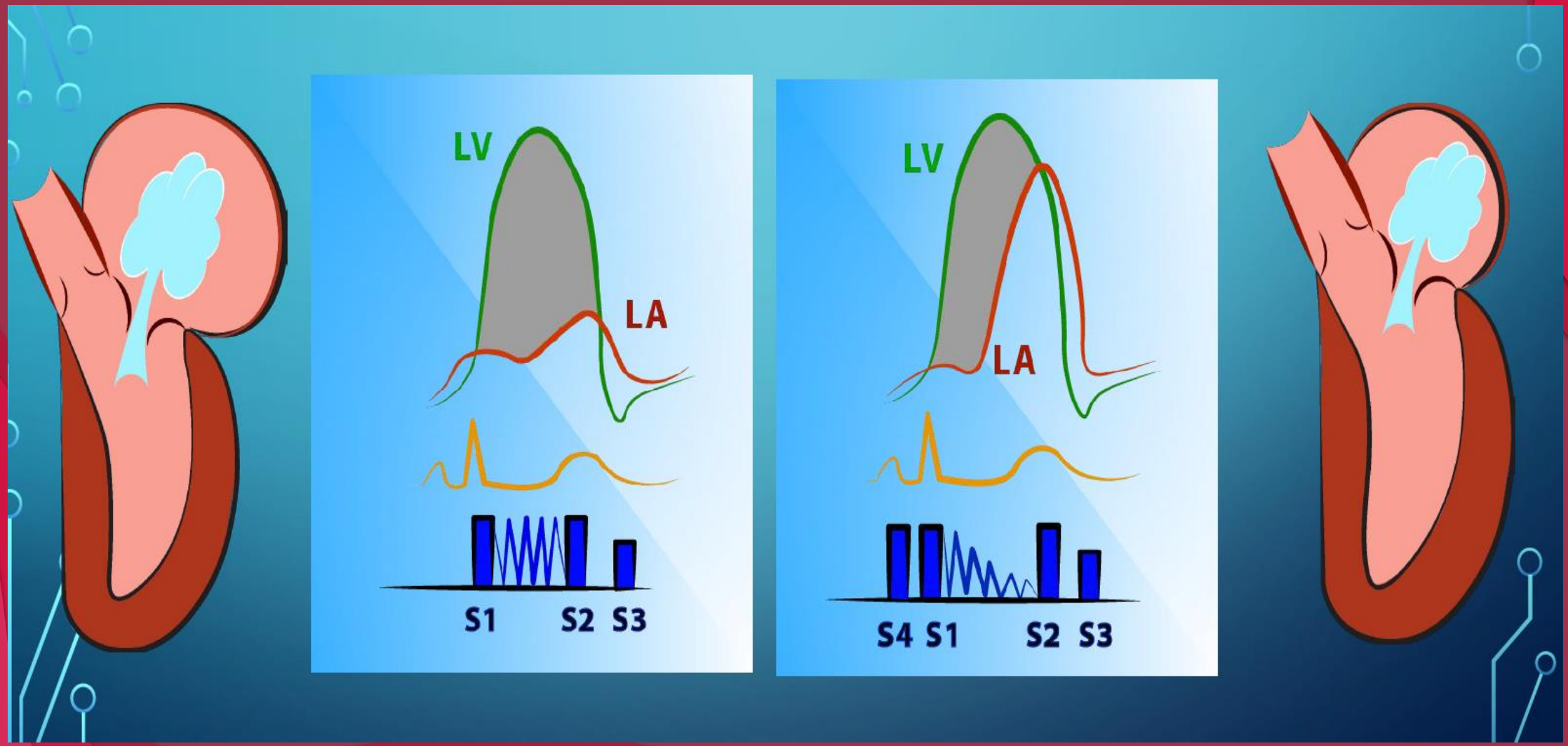
Valvular Heart Disease

Acute Severe Mitral Regurgitation

- *** A condition in which the heart's mitral valve doesn't close tightly, which allows blood to flow backward in the heart.
- Pathophysiology: Abrupt elevation of left atrial pressure in the setting of normal left atrial size and compliance, causing backflow into the pulmonary circulation with resultant pulmonary edema.
- Cardiac output decreases because of decreased forward flow, so hypotension and shock can occur
- Causes: 1- Chordal Rupture. 2- Infective Endocarditis. 3- Ischemic Heart Disease (Papillary muscle rupture)
- Associated with Pulmonary congestion/edema, S3 and S4, and higher mortality than the chronic form
- MR murmur may be soft, short or absent.
- Treatment: 1- Treat with afterload reduction (Vasodilators , Intra-aortic balloon pump). 2- Surgery -dictated by etiology (Papillary muscle rupture, dehisced mitral prosthesis → Operate immediately / Endocarditis → operate if the patient developed HF / Chordal rupture → depends on response to therapy).

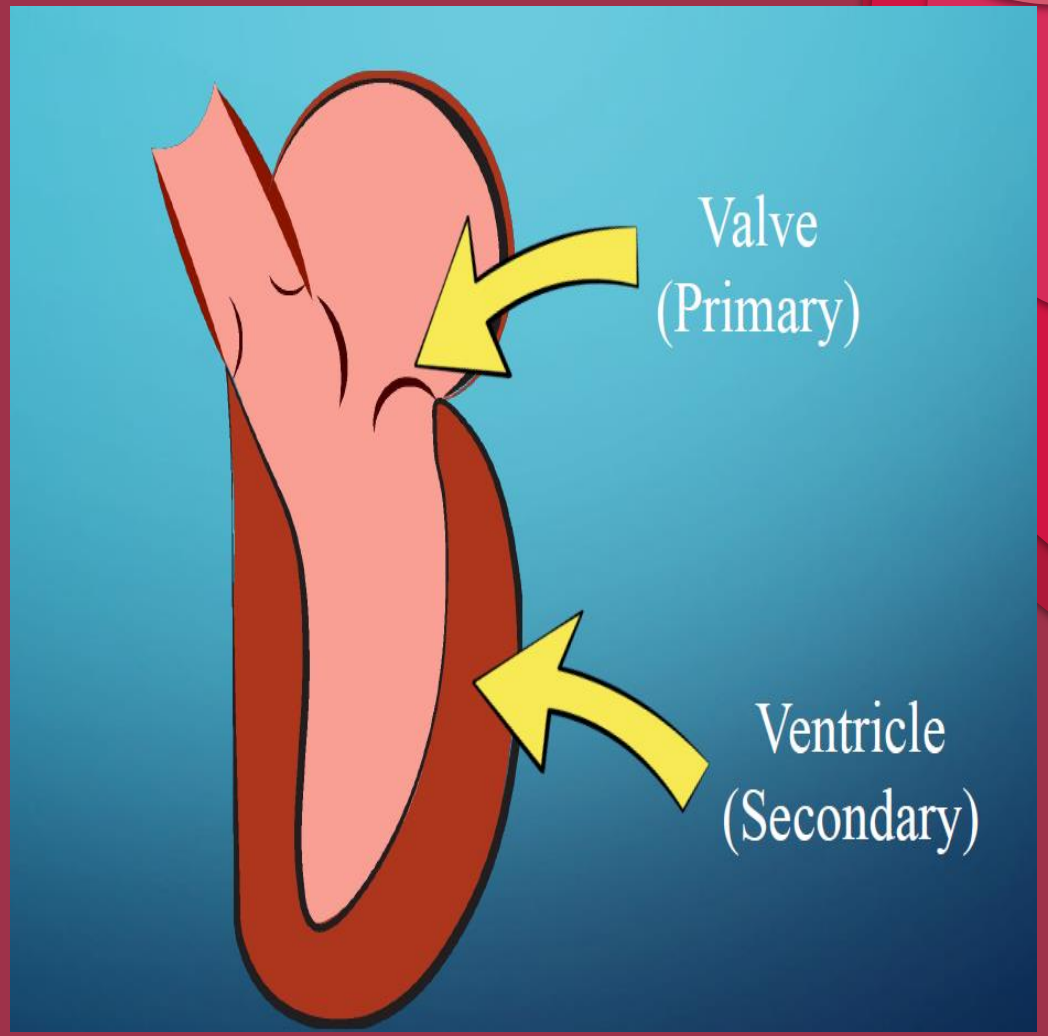


Hemodynamics of MR



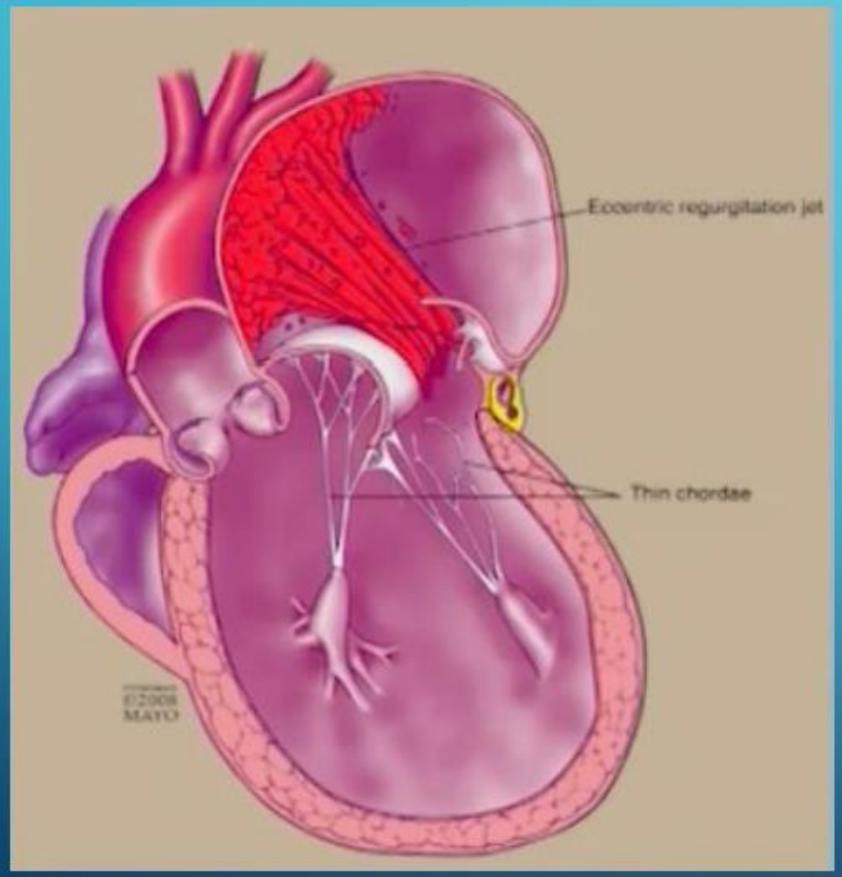
Chronic Mitral Regurgitation

- Pathophysiology: Gradual elevation of left atrial pressure in the setting of dilated left atrium and left ventricle (with increased left atrial compliance). LV dysfunction occurs due to dilation. Pulmonary HTN can result from chronic backflow into pulmonary vasculature.
- Causes: 1- Mitral valve prolapse. 2- Rheumatic fever. 3- Marfan syndrome. 4- Cardiomyopathy causing dilation of mitral annulus.
- Patients present with Exertional dyspnea, PND, Orthopnea, Palpitations, Pulmonary edema, Holosystolic murmur (starts at S1 and continues through S2) at the apex, A fib, Diminished S1, Widening of S2, S3 gallop.
- Diagnosis: 1- CXR findings (Cardiomegaly, dilated LV, Pulmonary Edema). 2- Echocardiogram (MR, Dilated LA and LV, Decreased LV function).

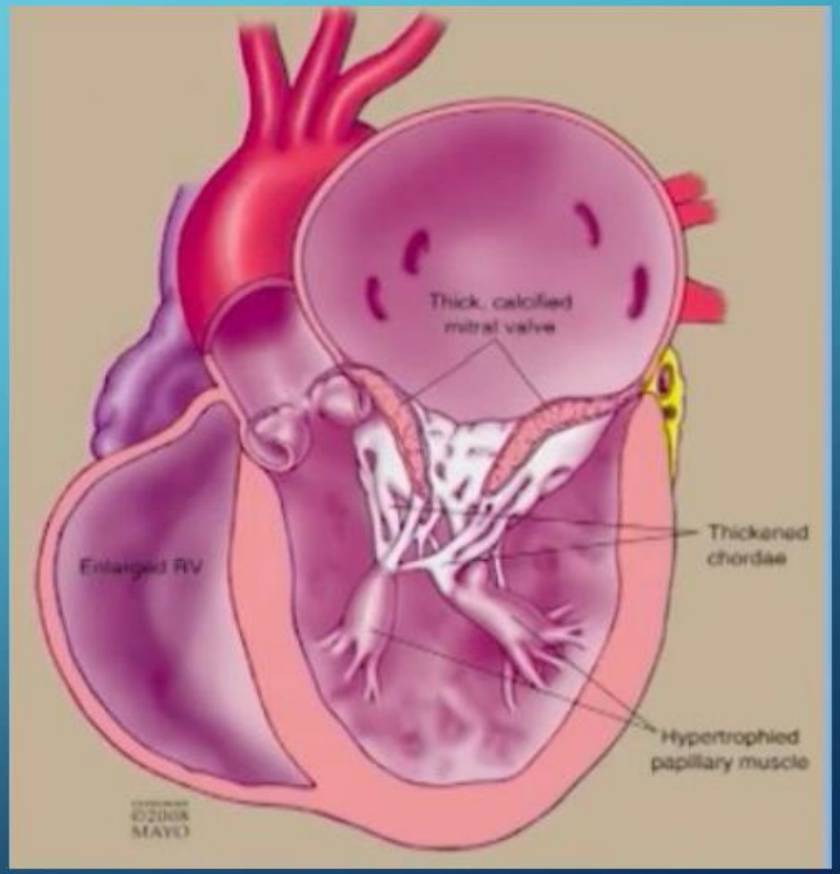


Primary Chronic MR

Degenerative

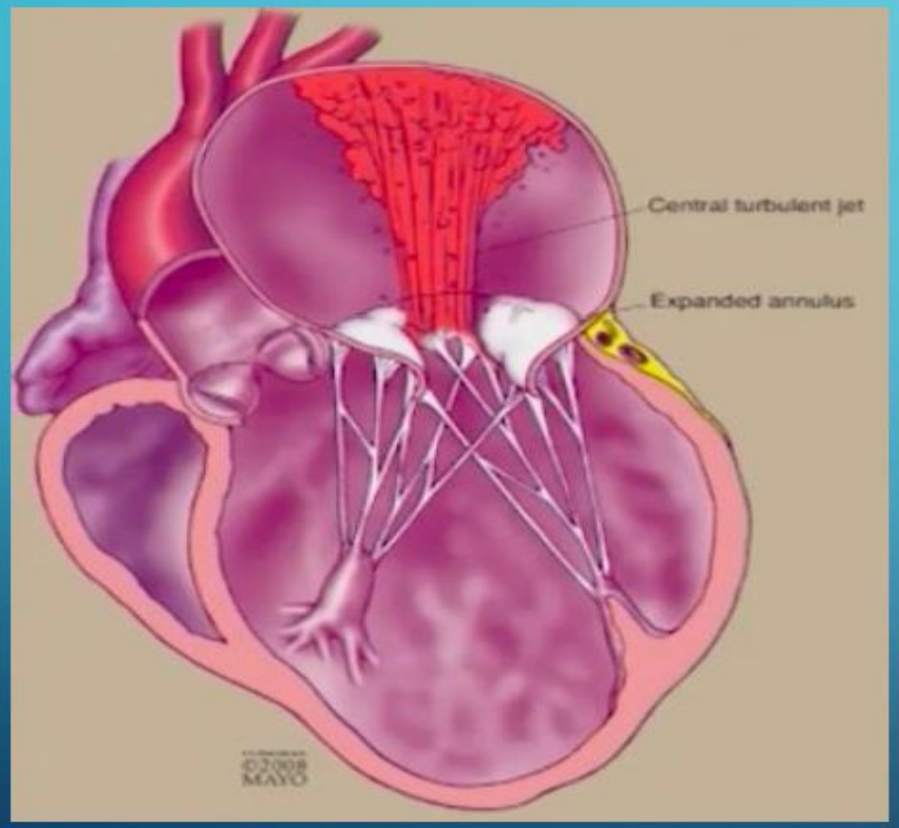


Rheumatic

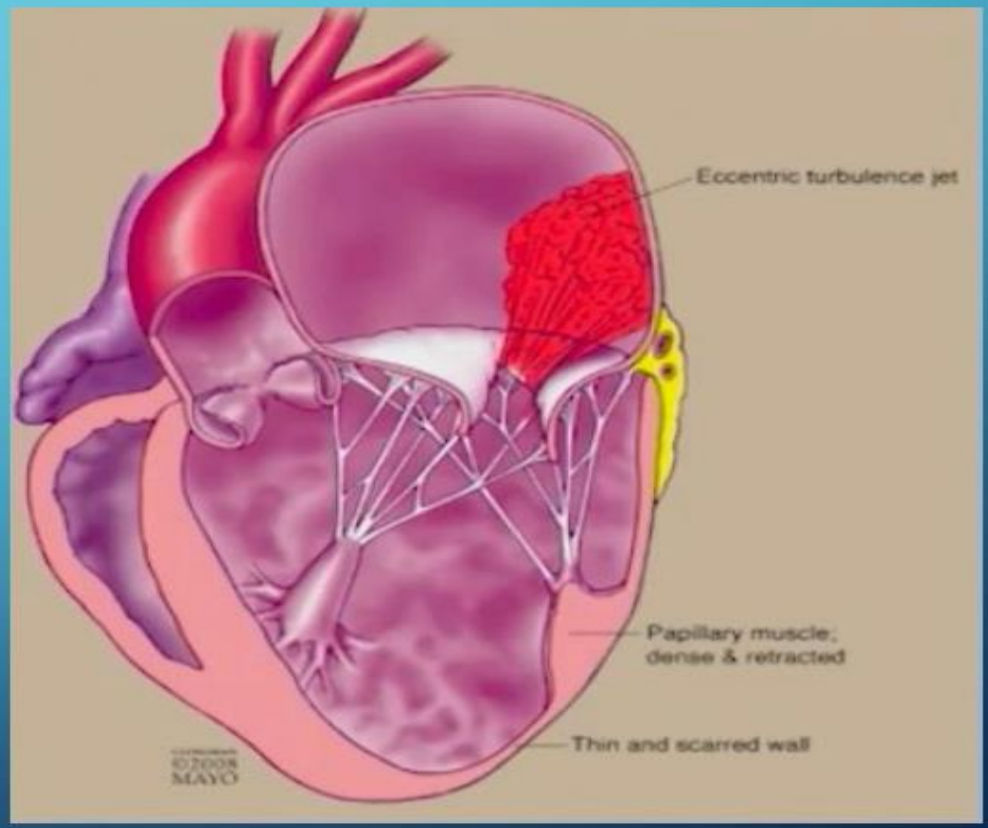


Secondary Chronic MR

Dilated

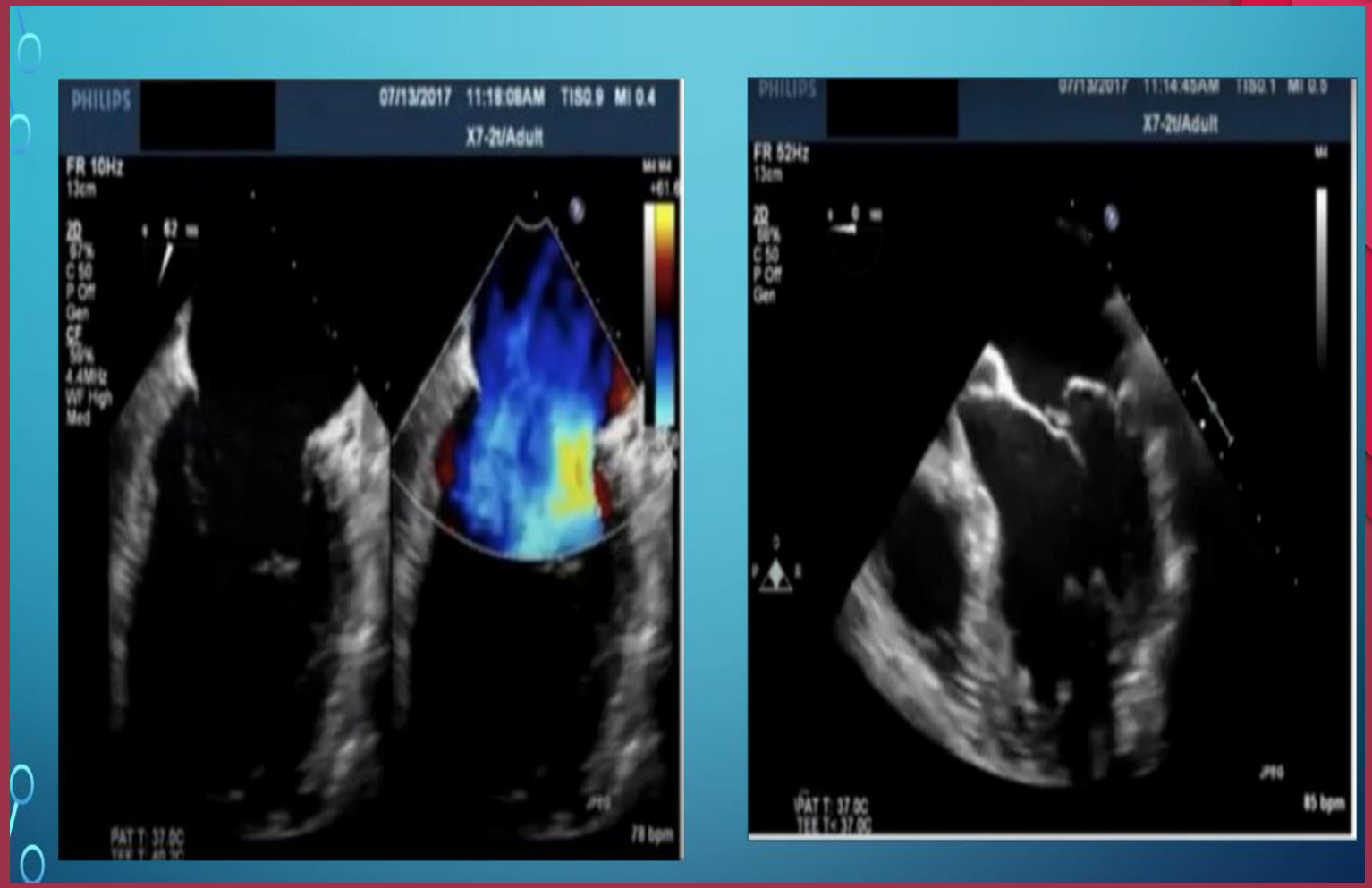


Ischemic



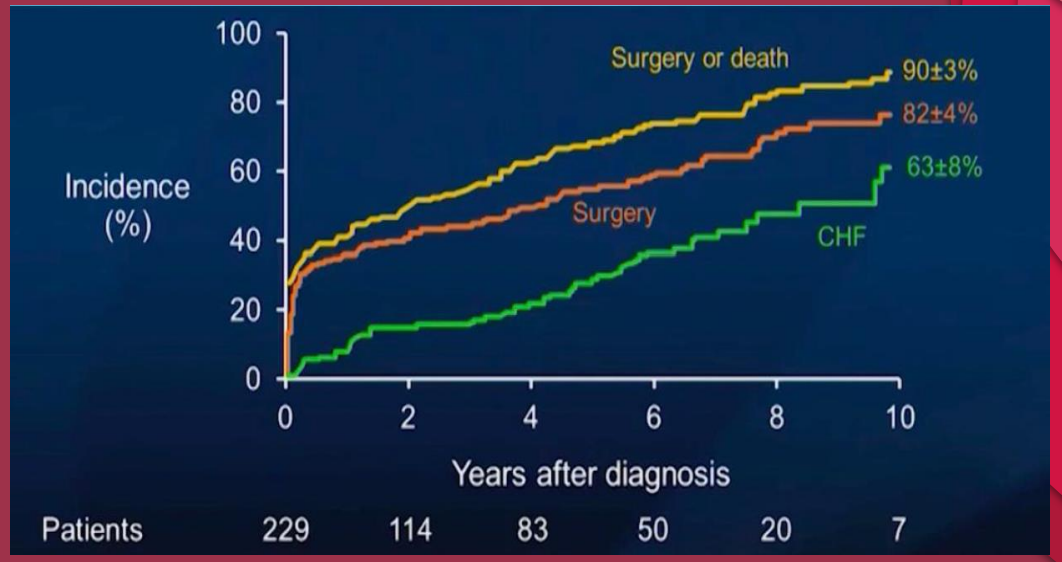
Chronic MR: Assessment

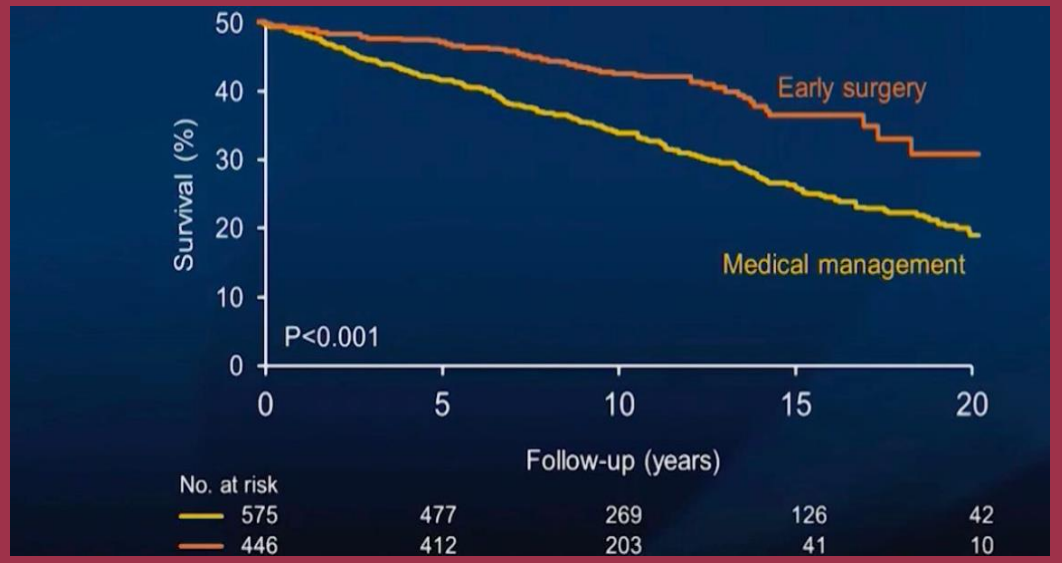
- Echocardiography determines:
  - 1- the severity of the regurgitation.
  - 2- the etiology (Flail leaflets, degenerative disease, Secondary MR, MVP).
  - 3- Evaluation of anatomy for intervention.



Treatment of Chronic Primary MR

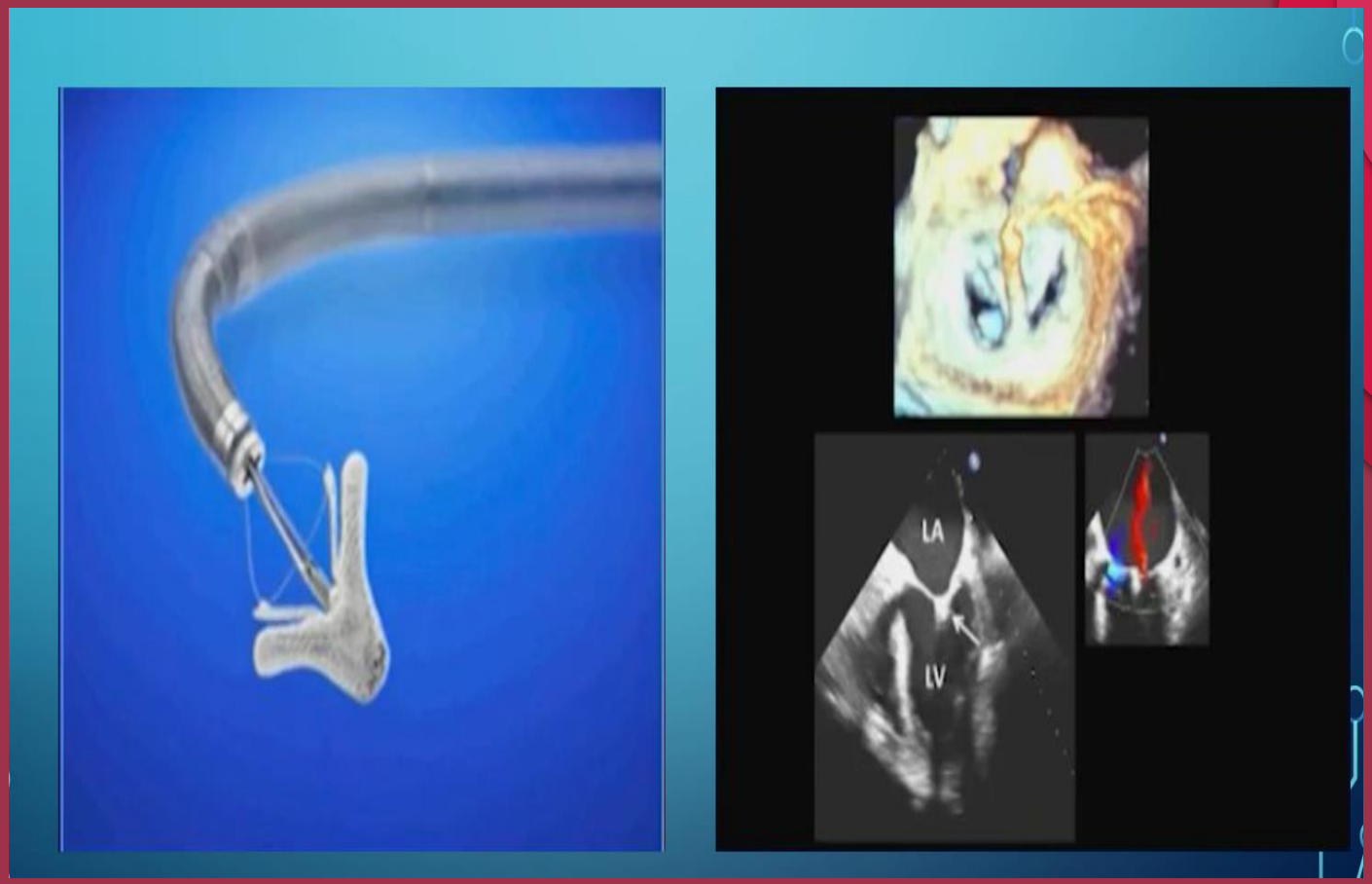
- In the absence of systemic HTN, no indication for vasodilator therapy if asymptomatic and preserved LV.
  - Indications for surgery (MVR or Repair):
    - 1- Severe MR.
    - 2- Any symptoms of HF.
    - 3- LV Dysfunction (EF <60%).
    - 4- LV Dilation.





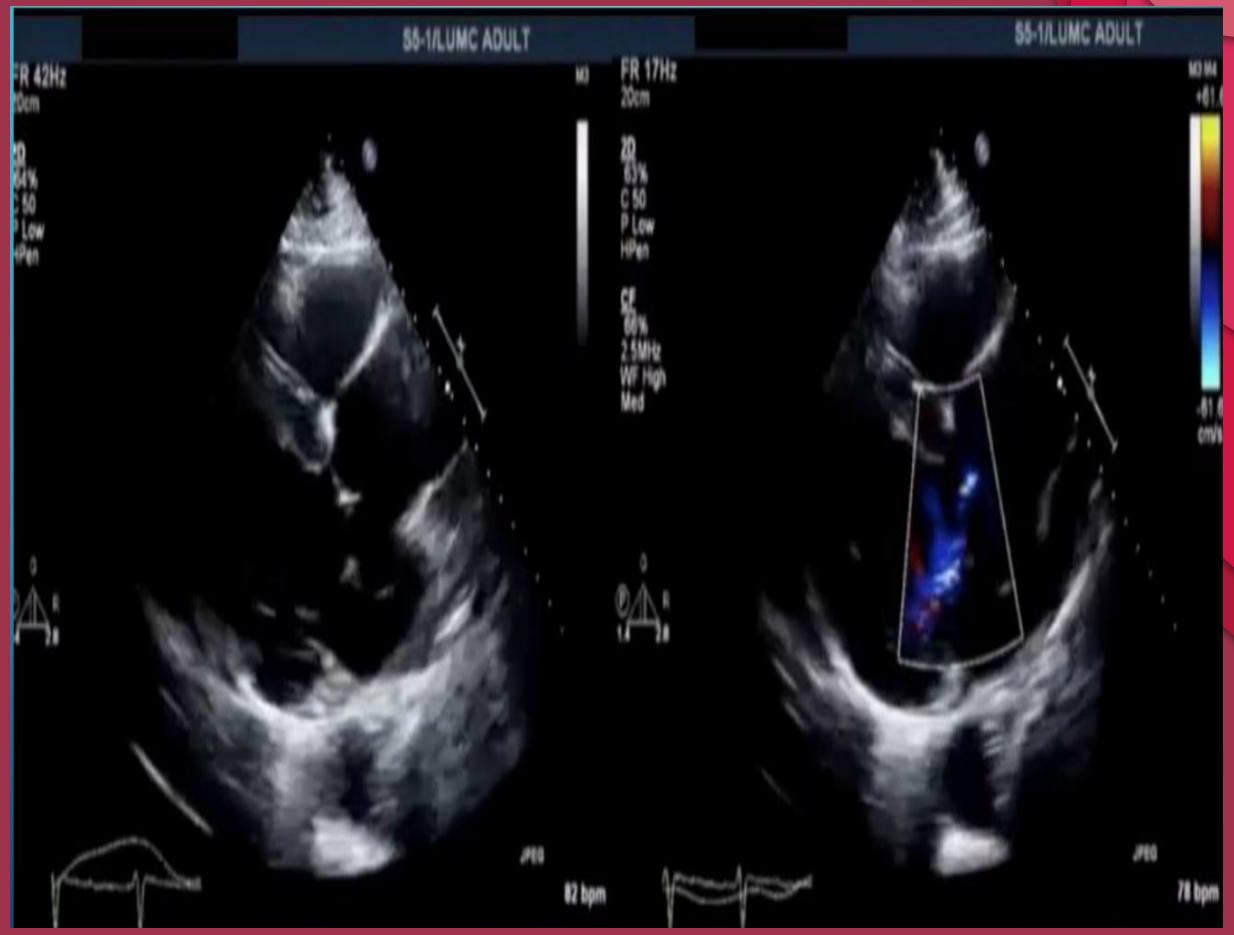
Treatment of Chronic Secondary MR

- 1- If ongoing symptoms with severe MR, consider treatment with Percutaneous Mitral-Clip
- 2- Treatment of chronic HF with guideline-directed medical therapy.
- 3- Treatment of ongoing ischemia if any.



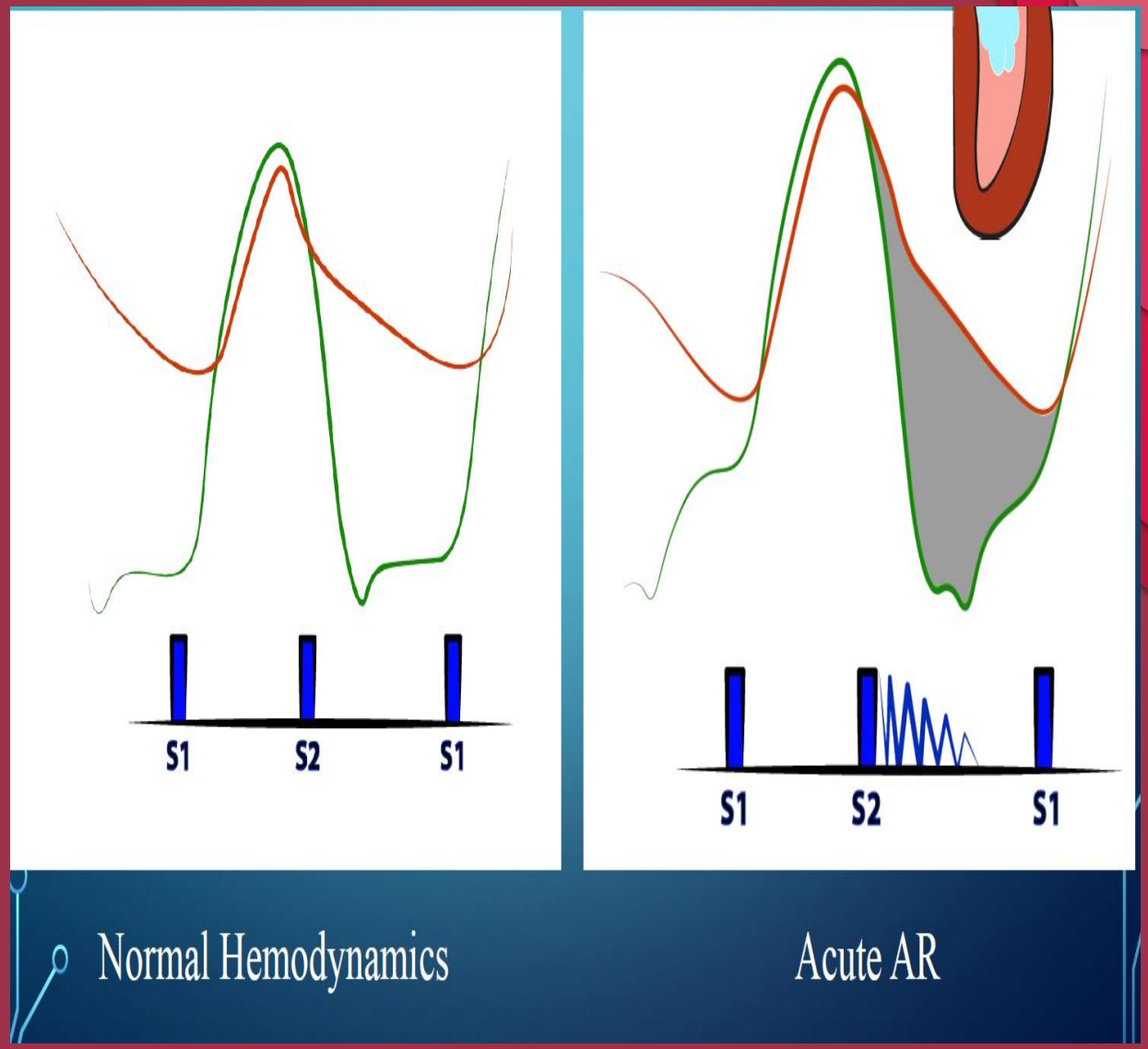
Aortic Regurgitation

- Pathophysiology of AR:
  - Also called aortic insufficiency; this condition is due to inadequate closure of the aortic valve leaflets. Regurgitant blood flow increases left ventricular end-diastolic volume.
  - LV dilation and hypertrophy occur in response in order to maintain stroke volume and prevent diastolic pressure from increasing excessively.
  - Over time, those compensatory mechanisms fail, leading to increased left-sided and pulmonary pressures.
  - The resting LV EF is usually normal until advanced disease.
- Etiologies: 1- Intrinsic Valvular (Degenerative/calcific, Bicuspid, Endocarditis, Rheumatic fever, Valvulitis, Anorexia medications). 2- Ascending Aortic (Degenerative, Type A dissection, Marfan syndrome, Inflammatory, Giant cell Arteritis).
- On PE: Wide pulse pressure (the most consistent finding), Head nodding (de Musset's), Capillary pulsation (Quincke's), Rapid Carotid upstroke / rapid collapse (Corrigan's pulse), "Pistol Shot" femoral (Duroziez's).
- Diagnosis: Echo



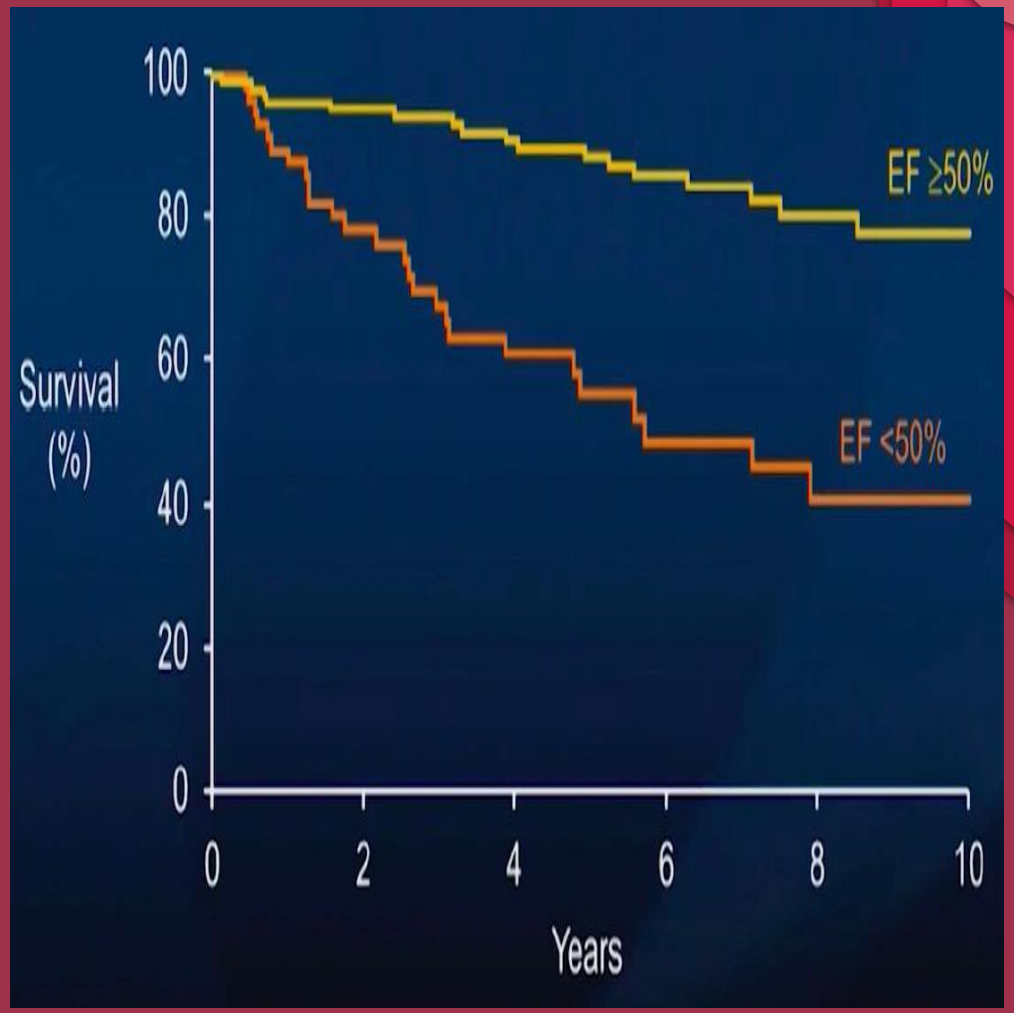
Acute Aortic Regurgitation

- Causes: 1- Root - Dissection. 2- Valve - Endocarditis.
- Associated with Pulmonary congestion/edema, S3 and S4.
- AR murmur may be soft, short or absent.
- May not have bounding pulse.
- Treatment: Urgent surgical intervention, Afterload enhancers are contraindicated, Afterload reducers can be used, Beta blockers contraindicated, Inotropic support can be used.



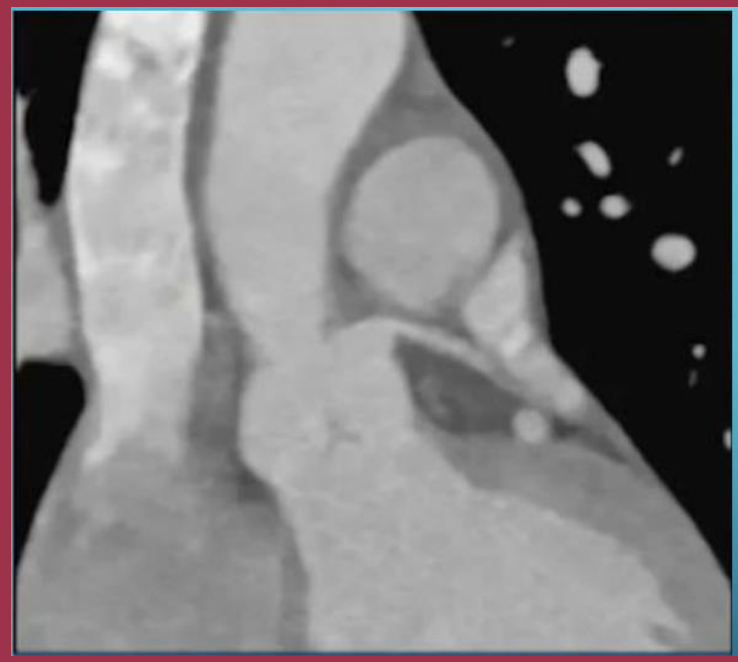
Chronic Aortic Regurgitation

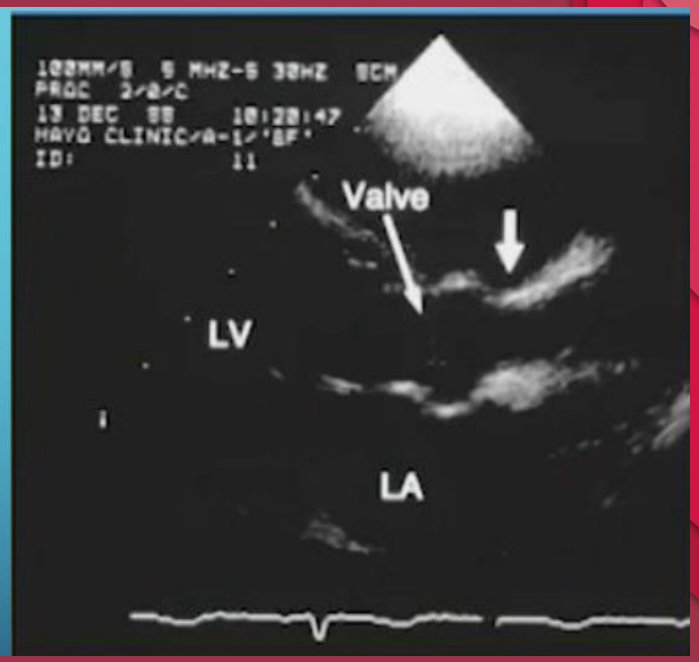
- Causes: 1- Primary valvular (Rheumatic Fever, Bicuspid Aortic valve, Marfan syndrome, Ehlers-Danlos syndrome, Ankylosing spondylitis, SLE). 2- Aortic root disease (Syphilitic Aortitis, Osteogenesis imperfecta, Aortic Dissection, Behcet syndrome, Reiter syndrome, Systemic HTN).
- Treatment: In the absence of systemic HTN, no indication for vasodilator therapy if asymptomatic and preserved LV.
  - Indications for surgery (AVR):
    - 1- Severe AR.
    - 2- Any symptoms of HF.
    - 3- LV Dysfunction (EF <50%).
    - 4- LV Dilation.

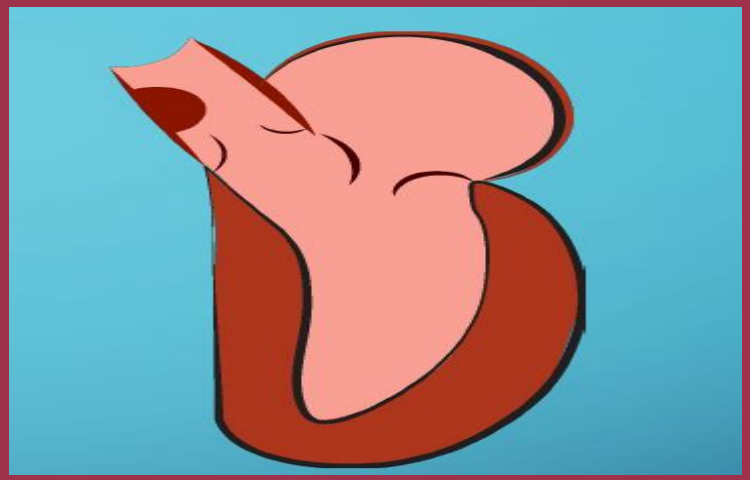


Aortic Stenosis

- Supravalvular Stenosis
- Level of Obstruction → Aorta (Single Discrete narrowing / Long tubular Hypoplasia).
- On PE: Thrill in Suprasternal notch or Rt carotid + Loud A2 + Systolic Murmur over the aortic area.
- Treatment: Surgical - may need conduit if severe.

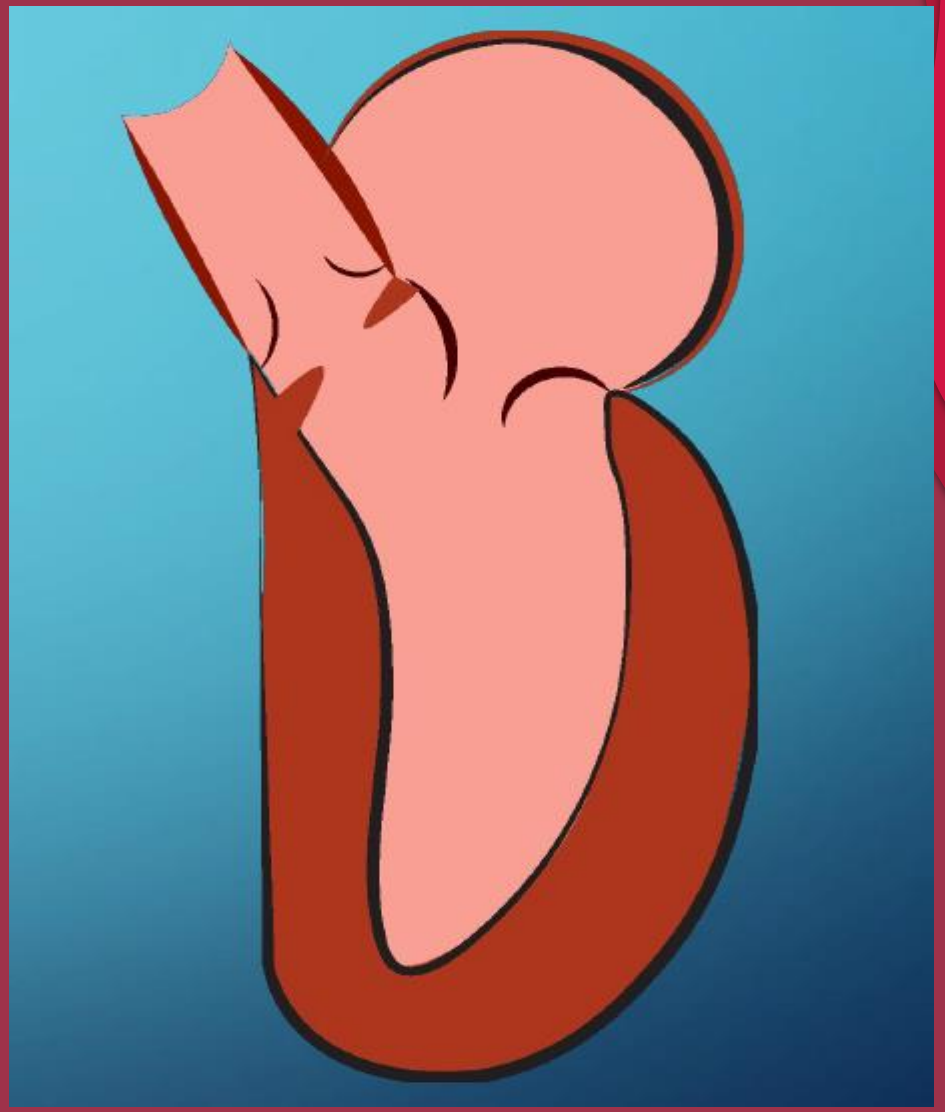






Aortic Stenosis

- Subvalvular Stenosis
- Seen in 10% of patients with AS (Discrete ridge, Tunnel stenosis, Frequently accompanied by AR due to jet on aortic valve).
- Diagnosis: Echocardiography.
- Treatment: Surgical resection especially if severe or there is AR progression



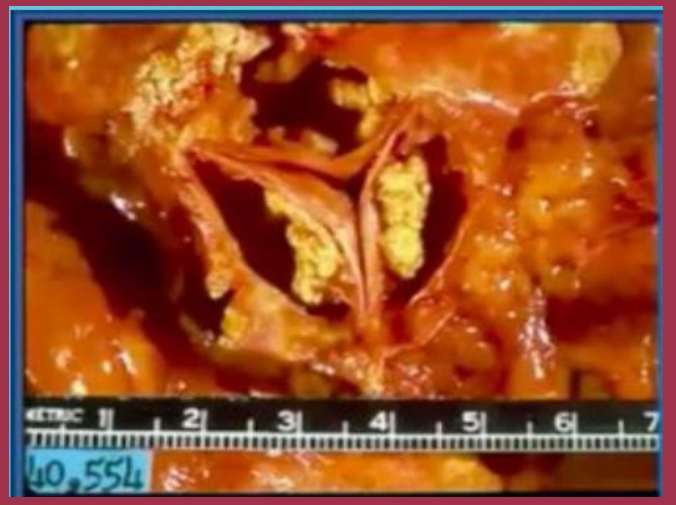
Aortic Stenosis

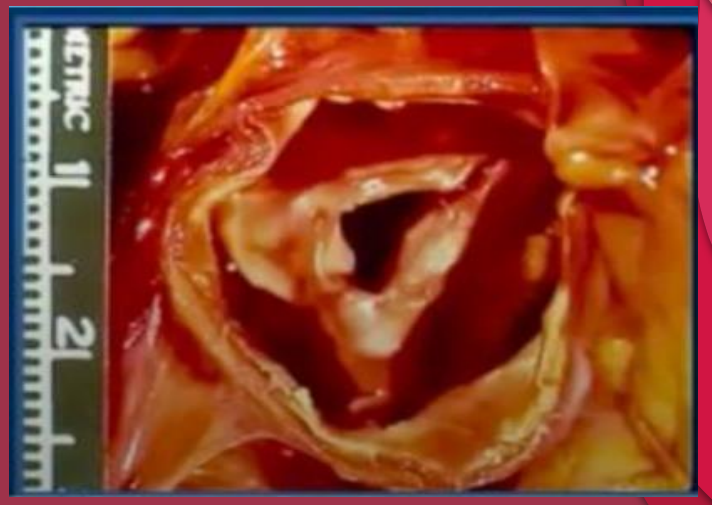
- Valvular Stenosis
- Age related:

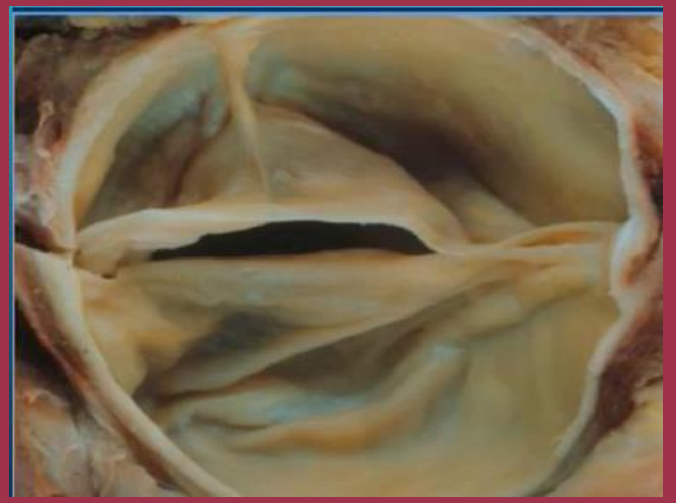
1- <30 → Congenital (Uni/Bicuspid)

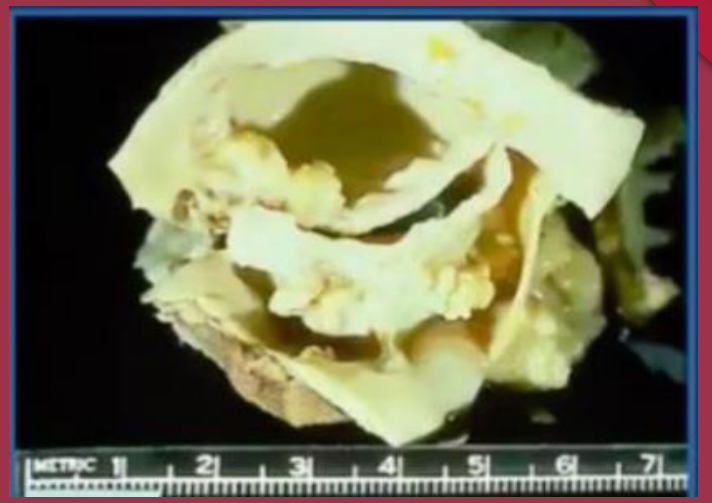
2- 40-60 → Calcified Bicuspid / Rheumatic

3- >70 → Senile degeneration (the most common).



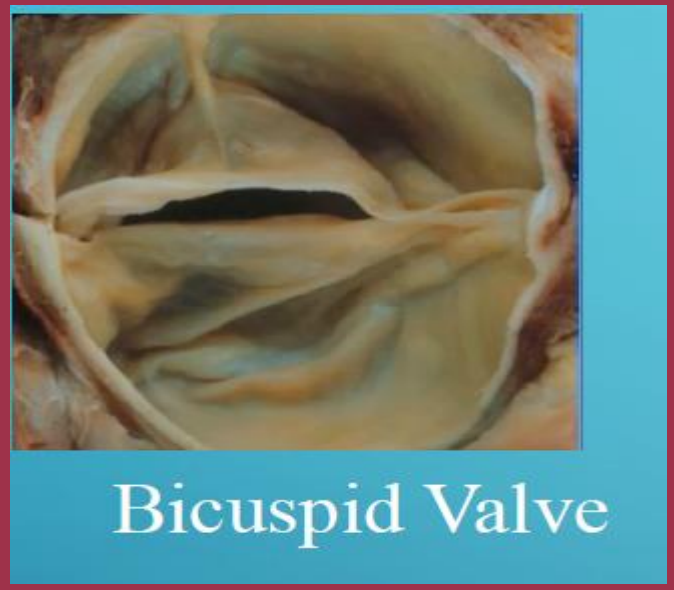




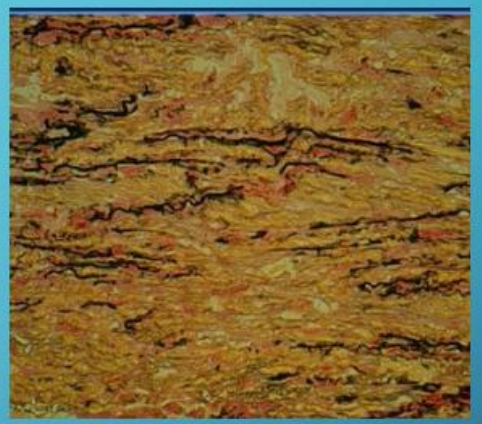


Aortic Stenosis

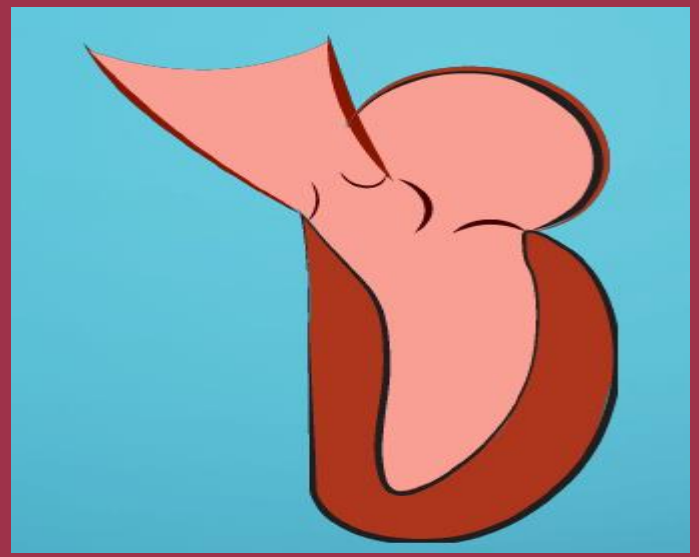
- Bicuspid Valve
- An aortic valve that has 2 cusps instead of 3.
- It may cause narrowed or obstructed aortic valve opening.
- Screen First degree relatives.
- Scan entire aorta.



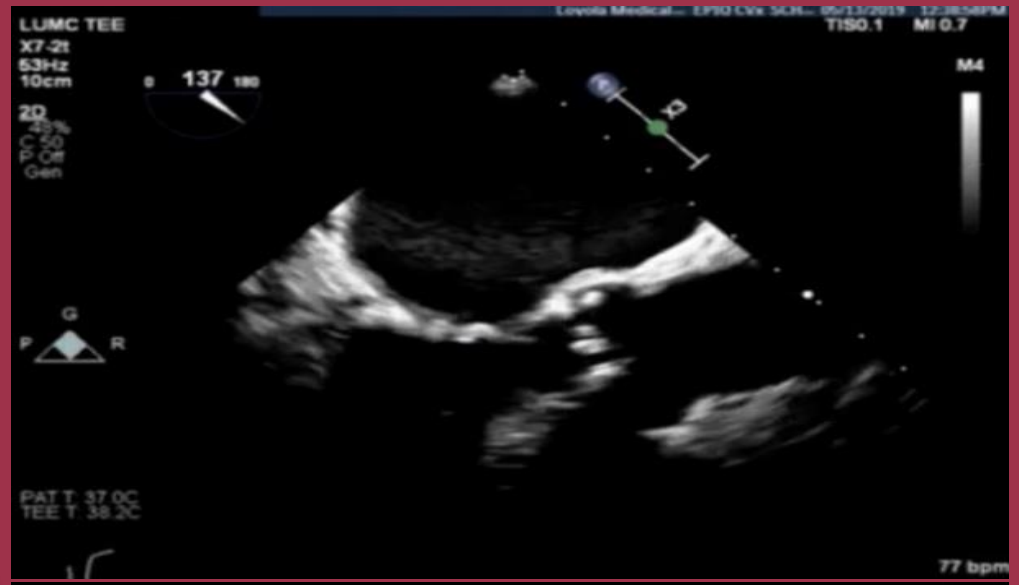
Bicuspid Valve

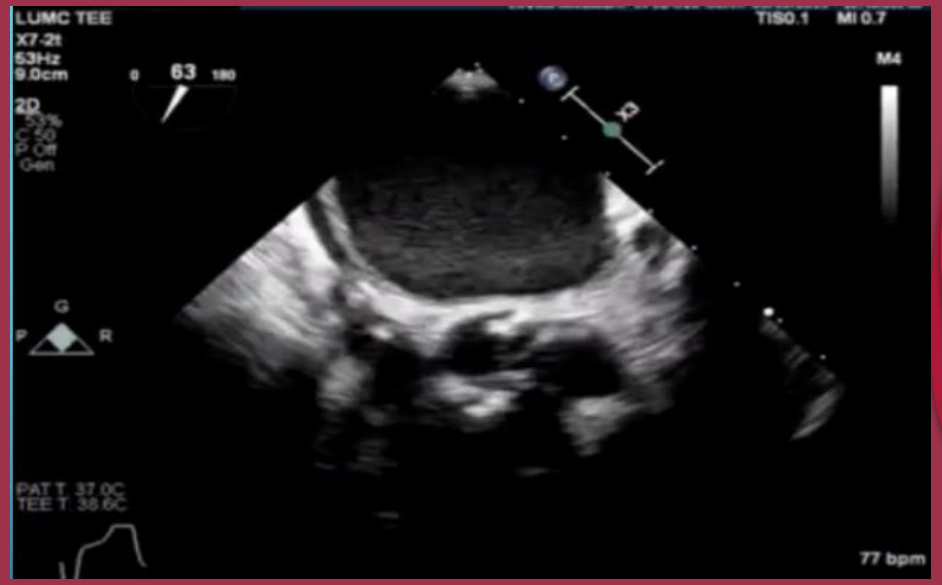


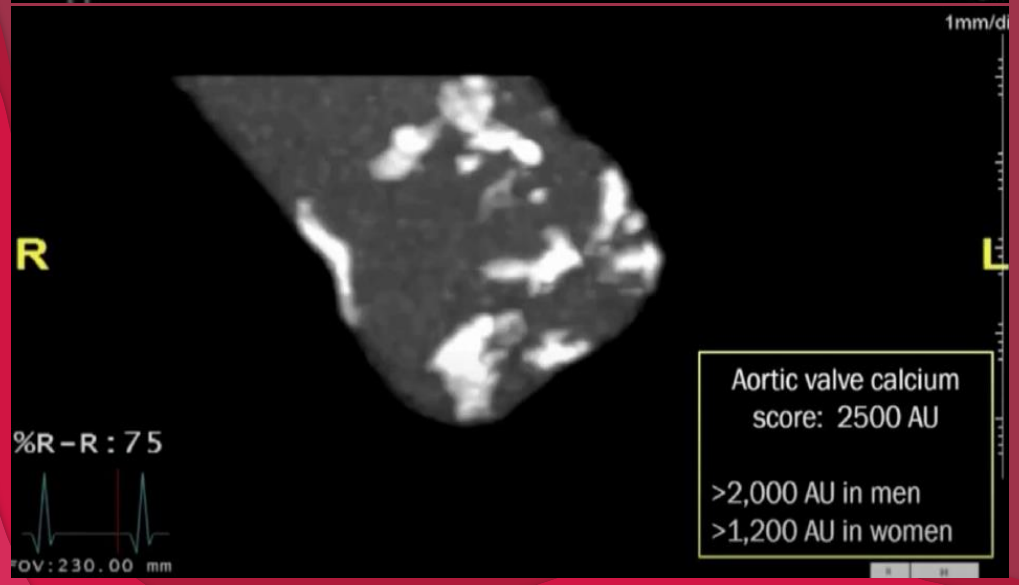
Aortic Medial Changes

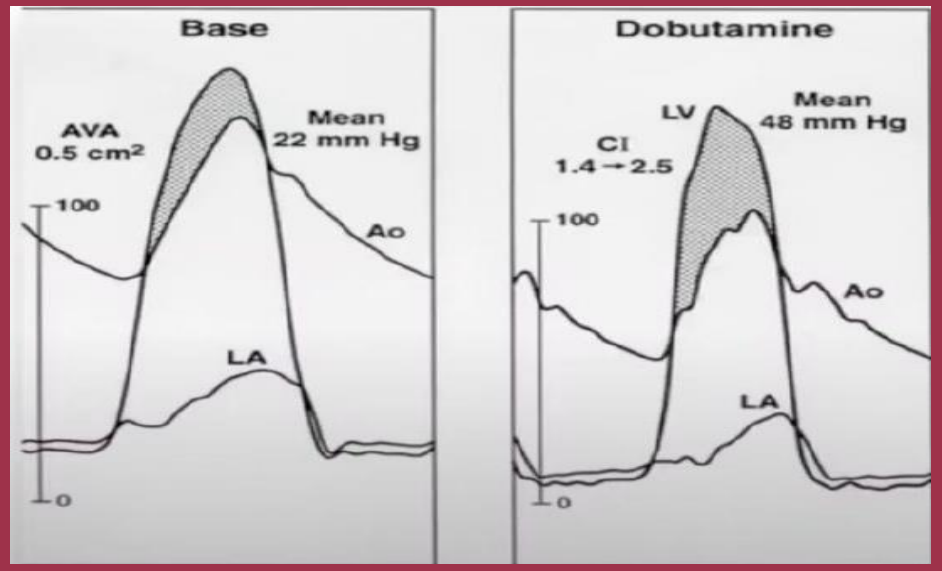


Aortic Stenosis: Diagnosis



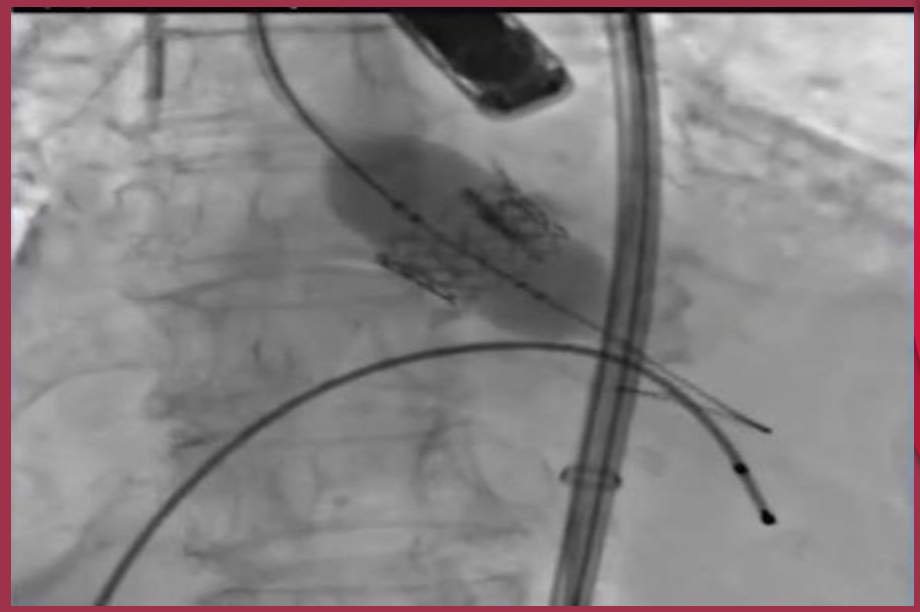






Aortic Stenosis: Treatment

- Aortic Valve Replacement
  - When to operate ?
- 1- Severe AS (with Symptoms, irrespective of LV function, LV dysfunction, Exercise-induced symptoms).
- 2- Moderate-severe AS if planned to undergo other cardiac surgery.
- 3- Asymptomatic Very severe AS.



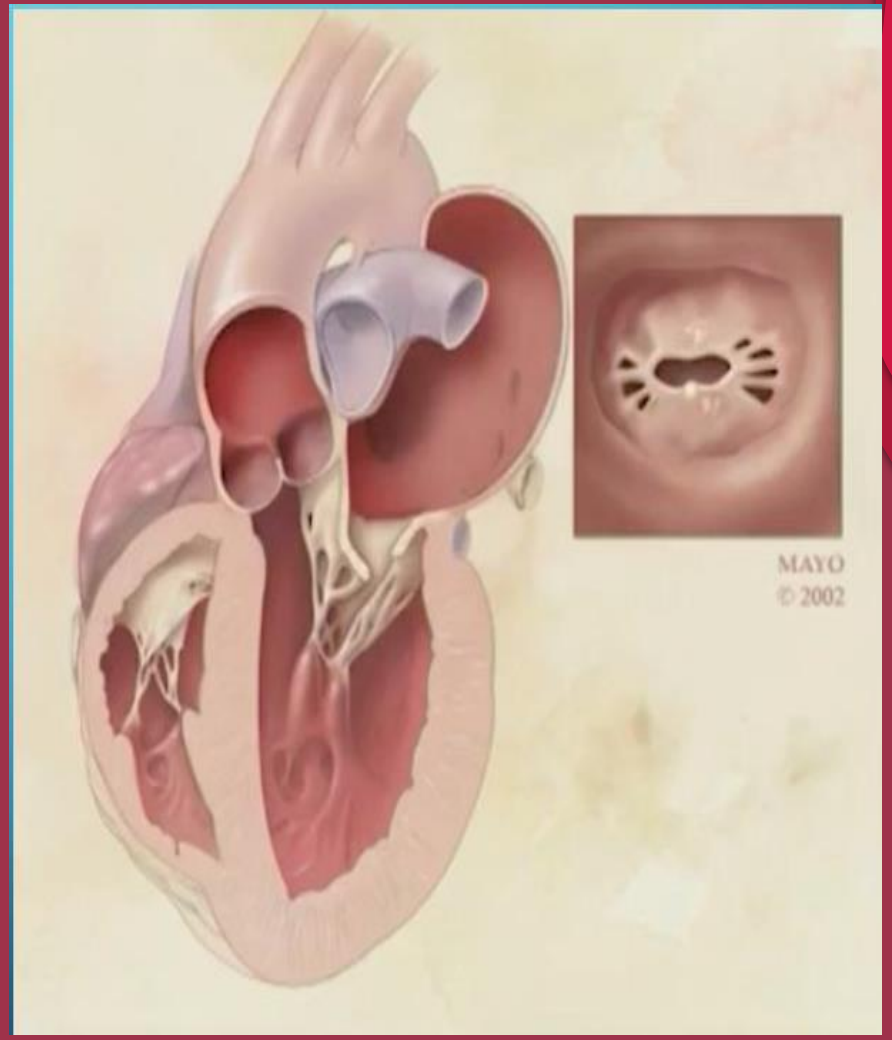
Mitral Stenosis

- Etiologies: 1- Rheumatic (Most common). 2- Degenerative calcification (older age).
- Pathophysiology: 1- Unaffected LV. 2- Elevated LA pressure. 3- Pulmonary HTN. 4- Atrial Arrhythmias. 5- RV failure.
- Patients present with Dyspnea, PND, Orthopnea, Hemoptysis, Palpitations, Emboli
- Indications for Surgery:

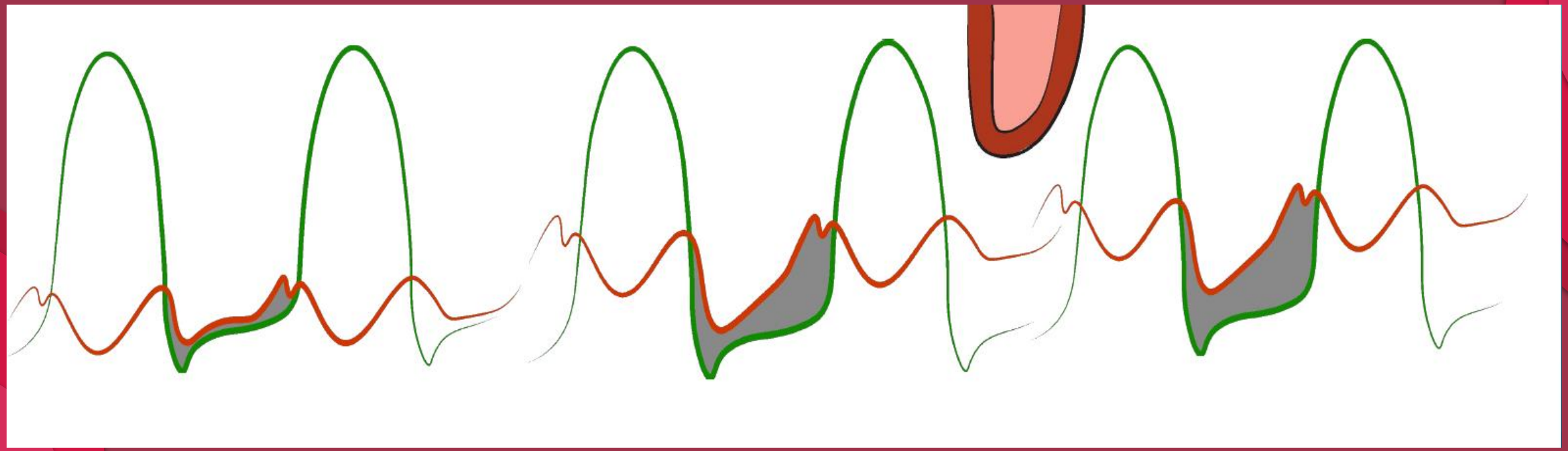
1- Severe symptoms of HF at rest or signs on exercise.

2- Mild symptoms if non-surgical management is feasible (Percutaneous mitral balloon commissurotomy)

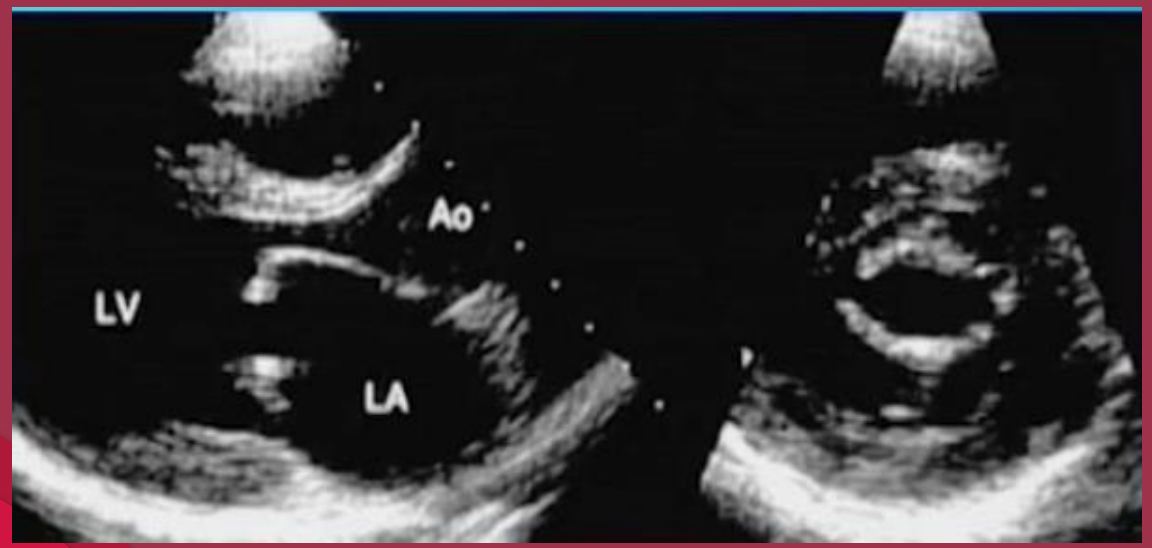
3- Anticoagulation if A fib

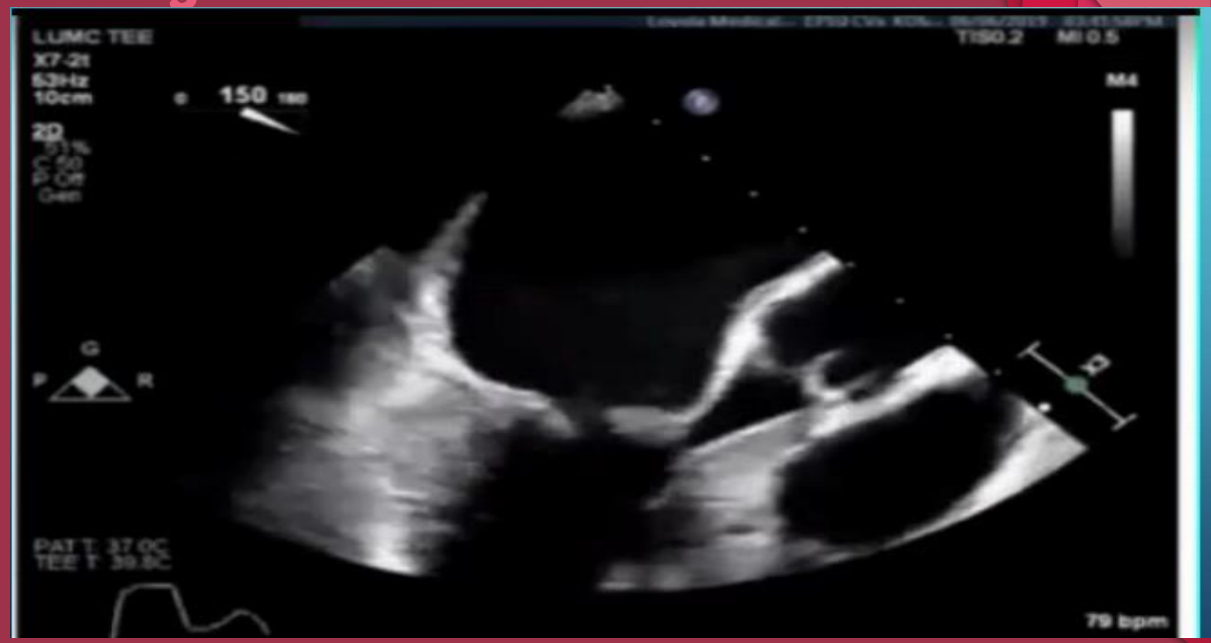


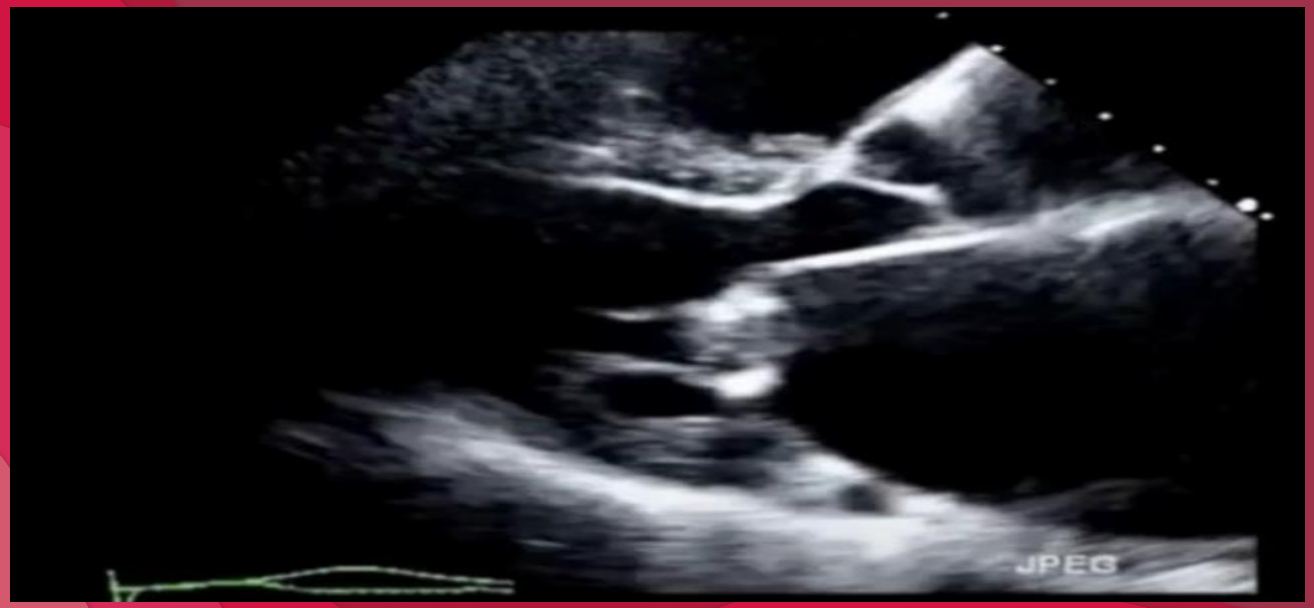
Mitral Stenosis: Pathophysiology



Mitral Stenosis: Diagnosis







Tricuspid Regurgitation

- Etiology: 1- Primary Valvular (Rheumatic, Congenital, Endocarditis, Carcinoid tumor, Pacemaker leads). 2- Secondary (Dilated Cardiomyopathies, Pulmonary HTN, A fib and annular dilation).
- Patients present with Edema, Ascites, and Fatigue.
- On PE: Elevated JVP, Pulsatile enlarged liver, Pansystolic murmur.
- When to operate ?

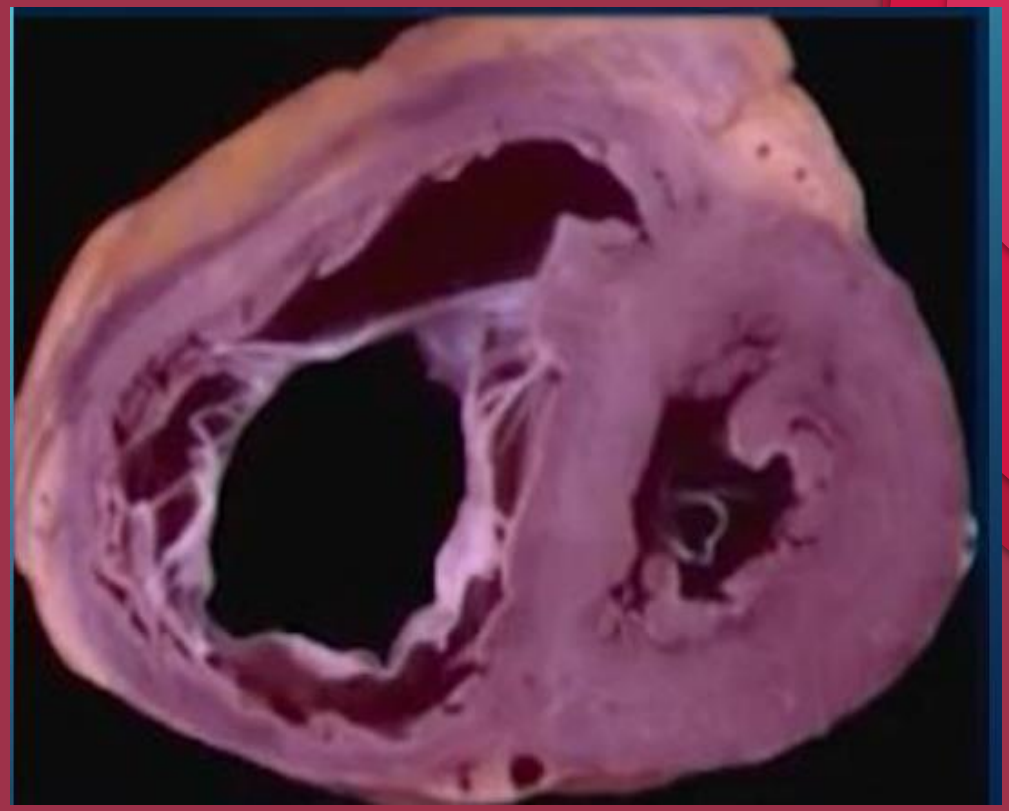
1- Severe symptomatic cases not responding to diuretics.

2- Severe, if left-sided surgery is planned.

3- Moderate, if left-sided surgery and RV is enlarged.

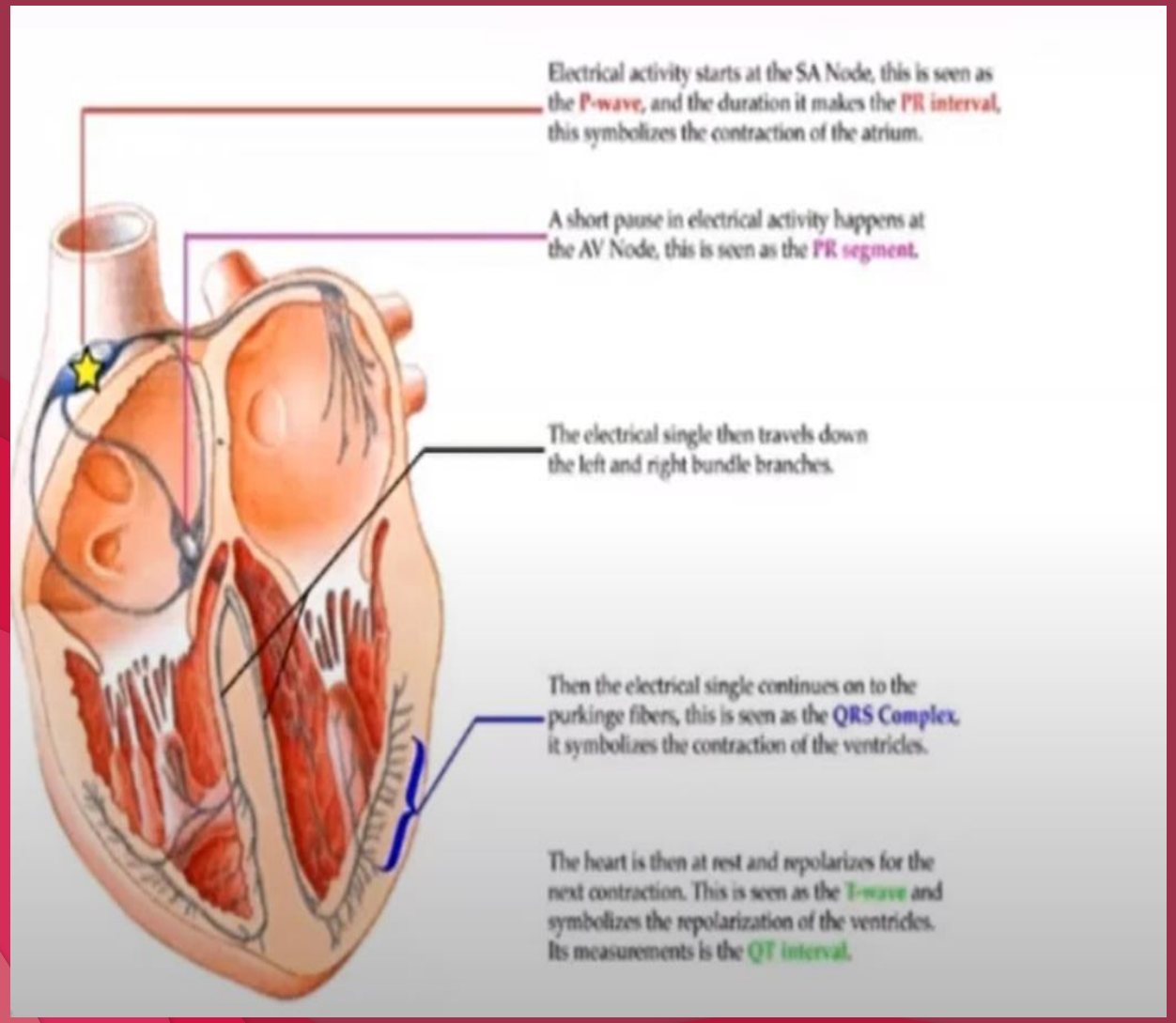
4- If related to pacemaker lead, attempt lead removal +/- TVR.

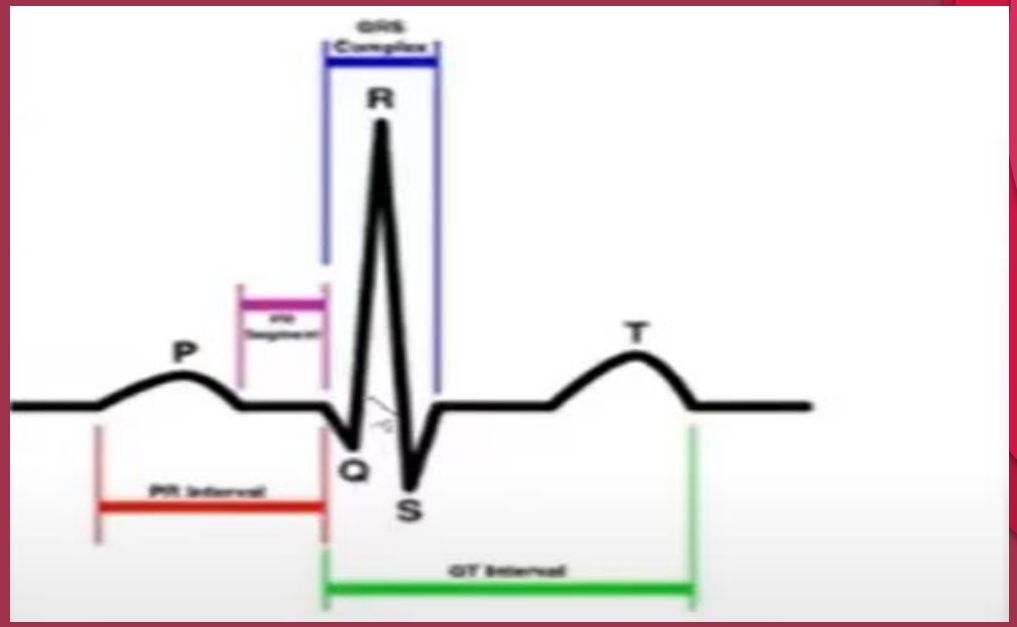
5- If A fib, attempt to return to normal sinus rhythm first.

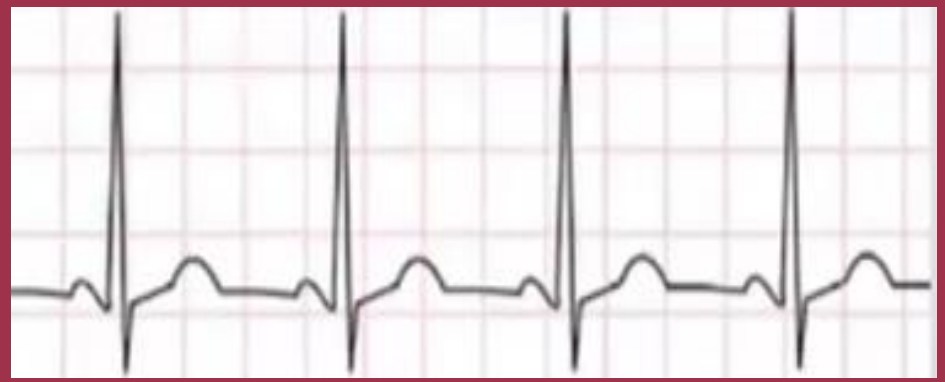


Cardiac Arrhythmias

ECG Basics

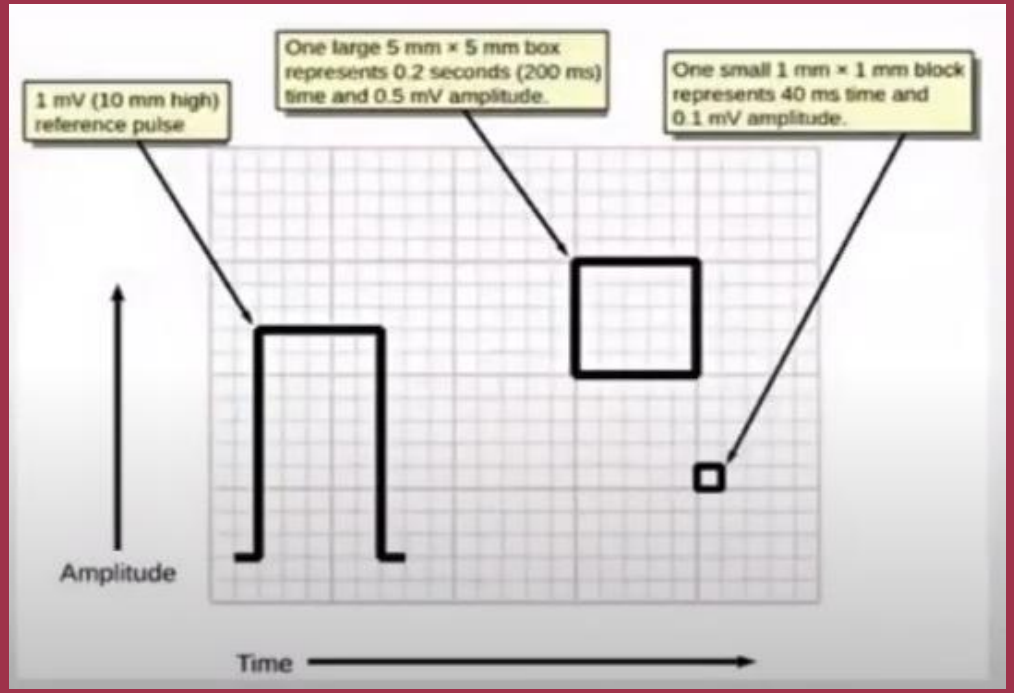

Electrical activity starts at the SA Node, this is seen as the P-wave, and the duration it makes the PR interval, this symbolizes the contraction of the atrium.
A short pause in electrical activity happens at the AV Node, this is seen as the PR segment.
The electrical single then travels down the left and right bundle branches.
Then the electrical single continues on to the purkinge fibers, this is seen as the QRS Complex, it symbolizes the contraction of the ventricles.
The heart is then at rest and repolarizes for the next contraction. This is seen as the T-wave and symbolizes the repolarization of the ventricles. Its measurements is the QT interval.

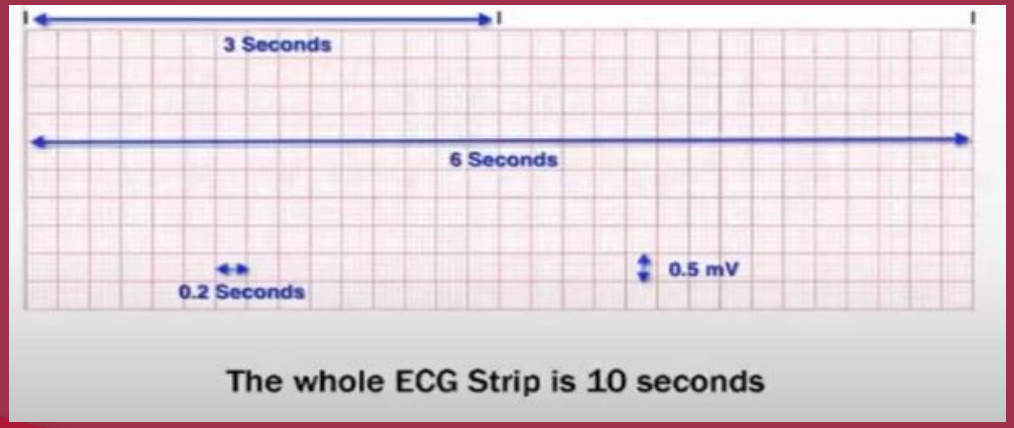

R
P
T
Q
S



ECG Basics

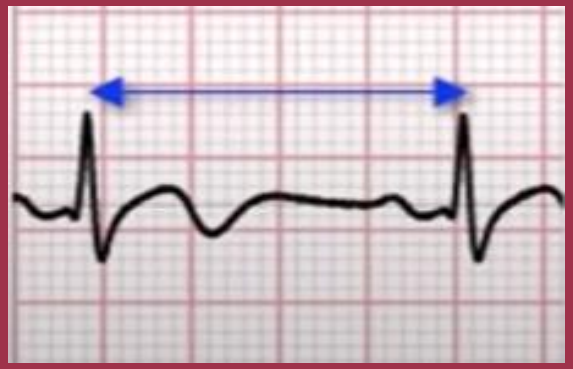
- Approach to Reading ECG:
  - 1- Verify
  - 2- Rate
  - 3- Rhythm
  - 4- Axis
  - 5- Amplitude
  - 6- Intervals
  - 7- Ischemia





ECG Basics: Rate

- Rate = 300 / # of Large boxes between two R waves
  - Rate = 1500 / # of Small boxes.
- Rate = # of QRS complexes in the entire strip X 6.
- The last method only works when we have regular rhythm.



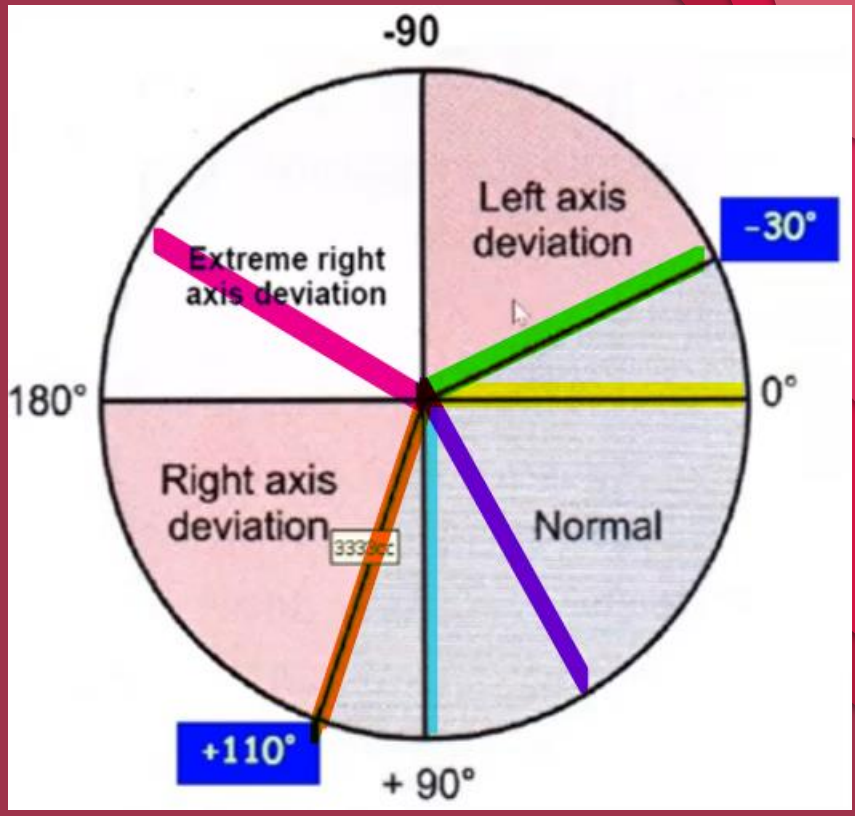
# Large Boxes	HR (BPM)
1	300
2	150
3	100
4	75
5	60
6	50

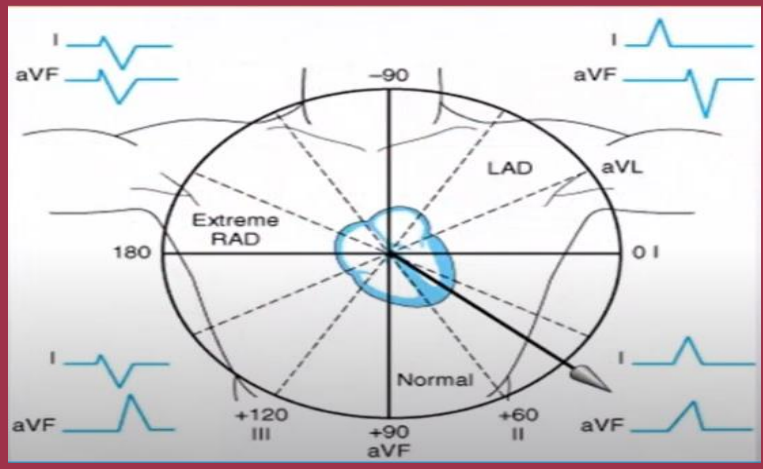
ECG Basics: Rhythm

Approach to Rhythm Questions:	Clinical Significance
1. Is it Tachycardia / Normal Rate / Bradycardia?	Rate
2. QRS is it Narrow or Wide?	Narrow: Rhythm from AVN and above & conduction through normal system Wide: Rhythm below AVN OR Abnormal conduction
3a. Narrow QRS – Is it Regular or Irregular	
3b. Wide QRS – What is Morphology?	Pathophysiology of Wide QRS: Vent. Origin or Aberrant conduction?
4. Look for P-wave (Best place in Lead II and V1)	What is the atria doing?
5. Relationship between the P wave and QRS ?	What is the underlying circuit?

ECG Basics: Axis

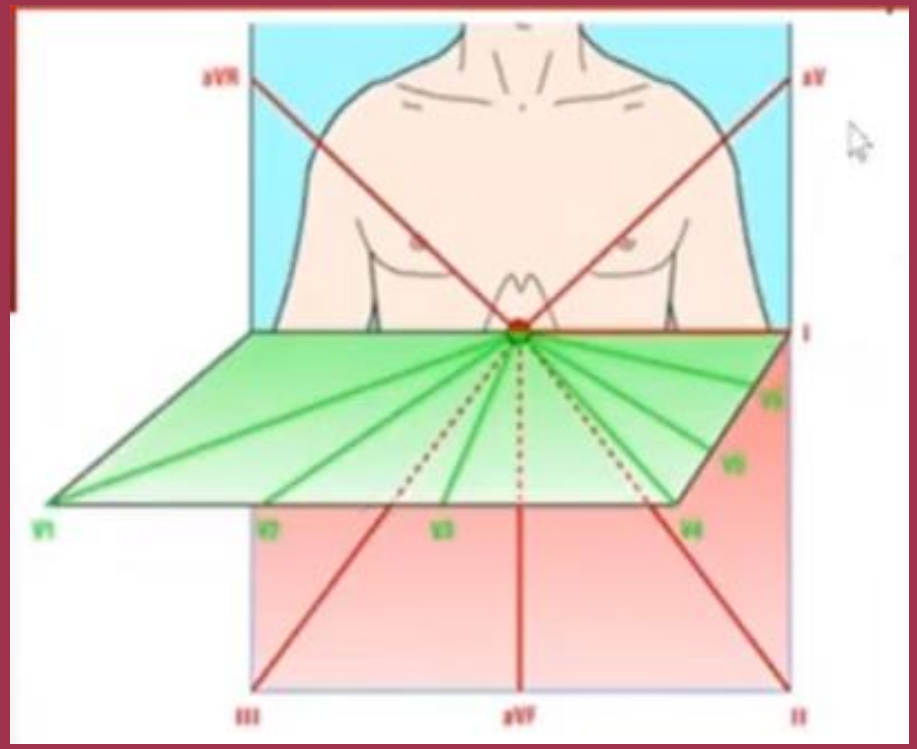
- Yellow Line → Lead I
- Purple Line → Lead II
- Orange Line → Lead III
- Green Line → Lead aVL
- Blue Line → Lead aVF
- Pink Line → Lead aVR
- Now, to determine the axis we look at leads I and aVF :
  - Lead I + / Lead aVF + → Normal Cardiac Axis
  - Lead I + / Lead aVF - → Look at Lead II (if lead II is positive then Normal Cardiac Axis, If lead II is negative then Left Axis Deviation)
  - Lead I - / Lead aVF + → Right Axis Deviation
  - Lead I - / Lead aVF - → Extreme Axis Deviation





ECG Basics: Axis

- Leads I, II, III, aVL, aVR, aVF are in the Red Plane.
- Leads V1-V6 are in the Green Plane.
  - V1-V2 → Septal
  - V2-V3 → Anterior
  - V4-V6 → Lateral.



ECG Basics: Amplitude

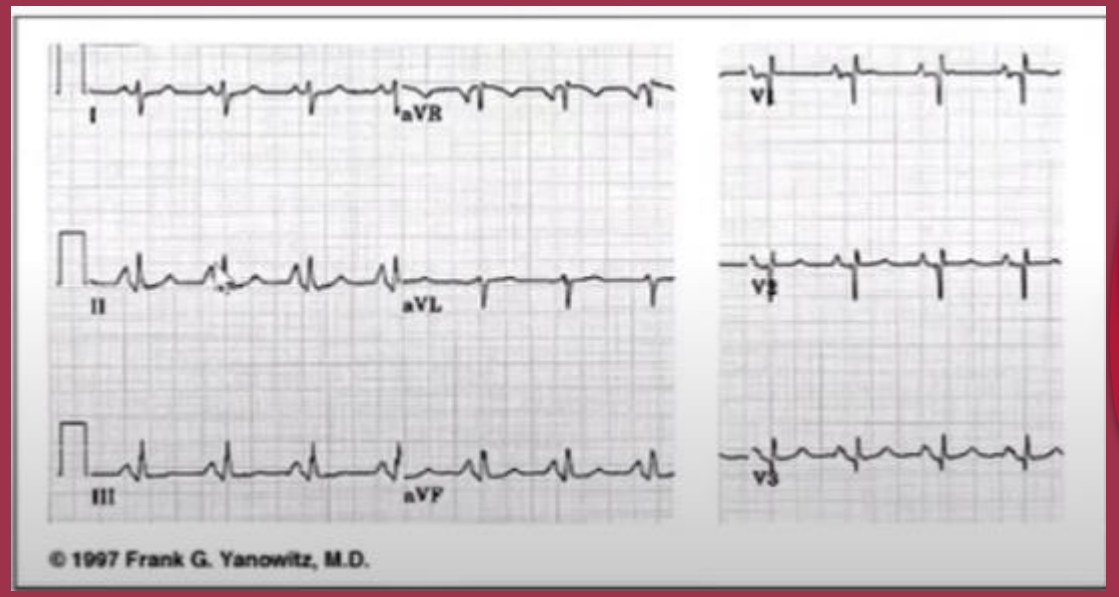
- Low amplitude:
  - Limb leads < 0.5 mV
  - Precordial Leads < 1.0 mV

Component	Amplitude (mV)
P wave	0.2
QRS	1.0
T-wave	0.2 – 0.3

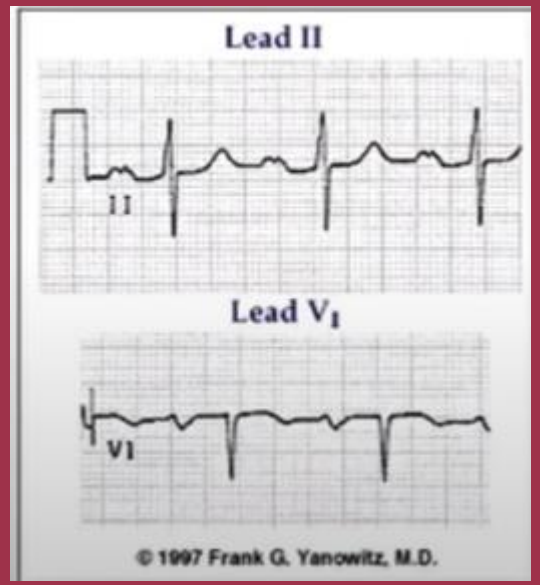
ECG Basics: Amplitude / Hypertrophy

- Figure A → Right Atrial Enlargement → P wave amplitude > 2.5 mm (more than 2 and a half small squares) in lead II and/or >1.5 mm in Lead V1; we call this P-wave: P pulmonale
- Figure B → Left Atrial Enlargement → P wave duration >= 0.12 seconds in frontal plane (usually lead II) + Notched P wave in limb leads with the inter-peak duration >= 0.04s + Terminal P negativity in Lead V1 (i.e. "P-terminal force") duration >=0.04s and depth >=1mm.
- Wider P wave, notched and Biphasic → P mitrale

A



B

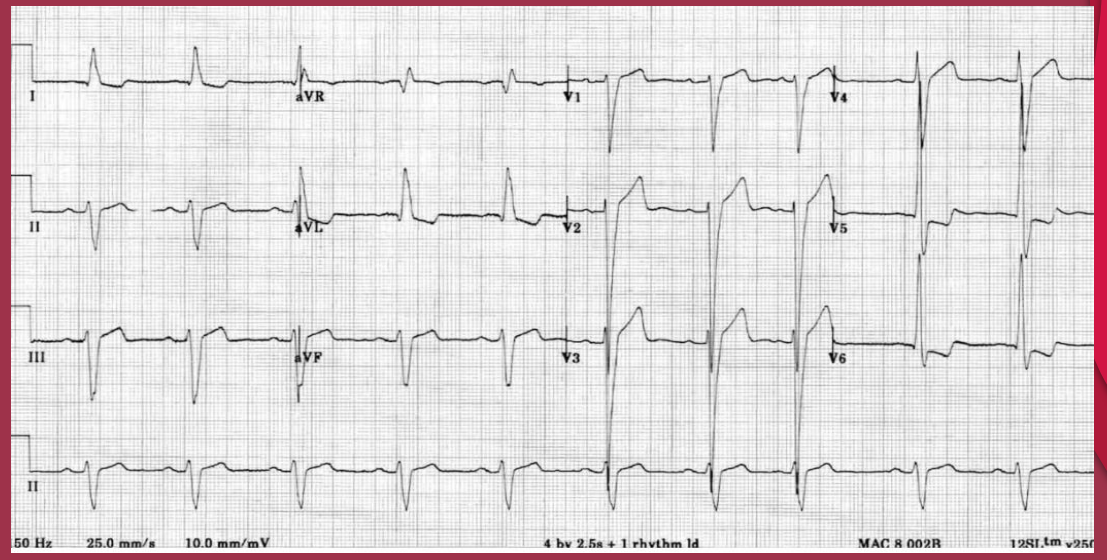


ECG Basics: Amplitude / Hypertrophy

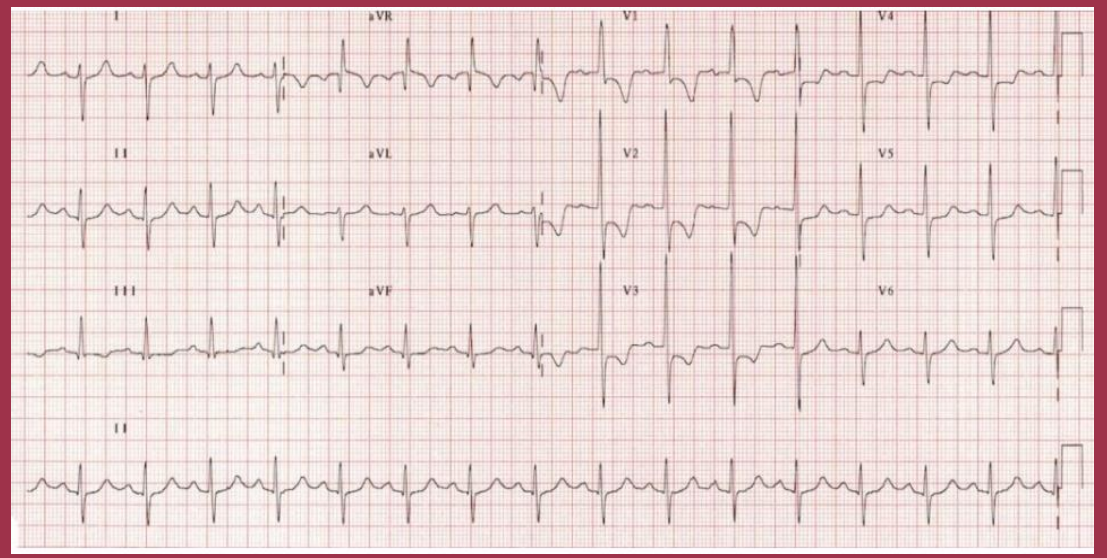
- Left Ventricular Hypertrophy Criteria (Figure A):
- 1- R in aVL >= 11 mm (Limb lead voltage criteria).
- 2- S in V3 + R in aVL > 24 mm in men / 20 mm in women

- Right Ventricular Hypertrophy (Figure B) Criteria:
- 1- Right Axis Deviation > 90 degrees.
- 2- R/S ratio > 1 and negative T wave in Lead V1
- 3- R > 6 mm or S < 2 mm in Lead V1

A

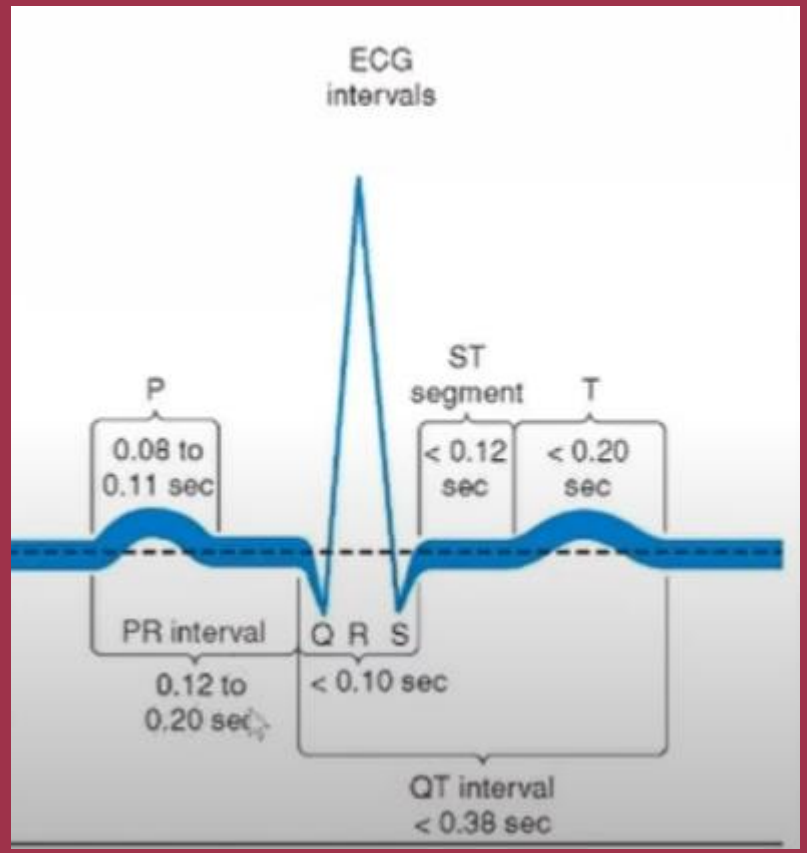


B

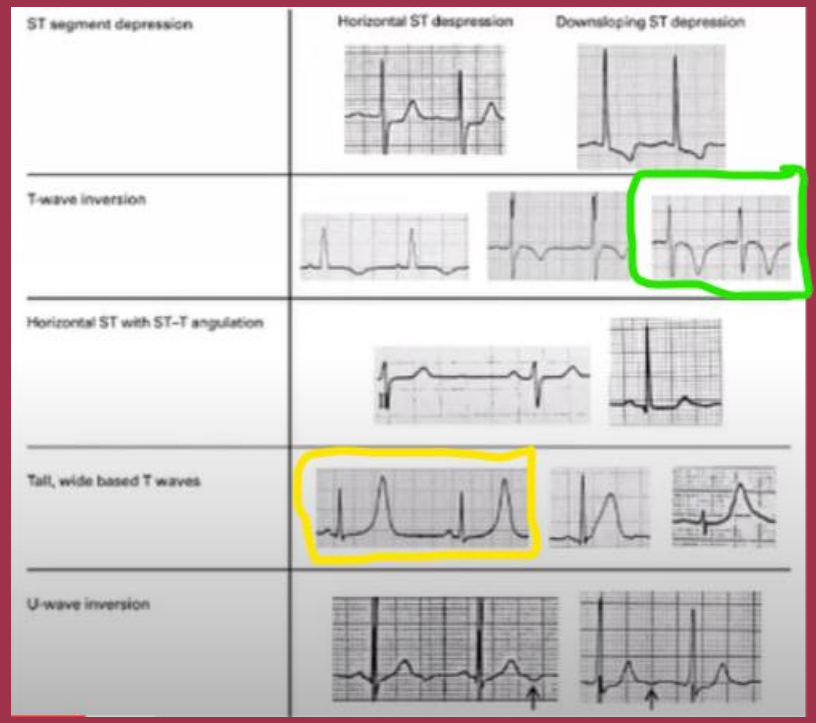


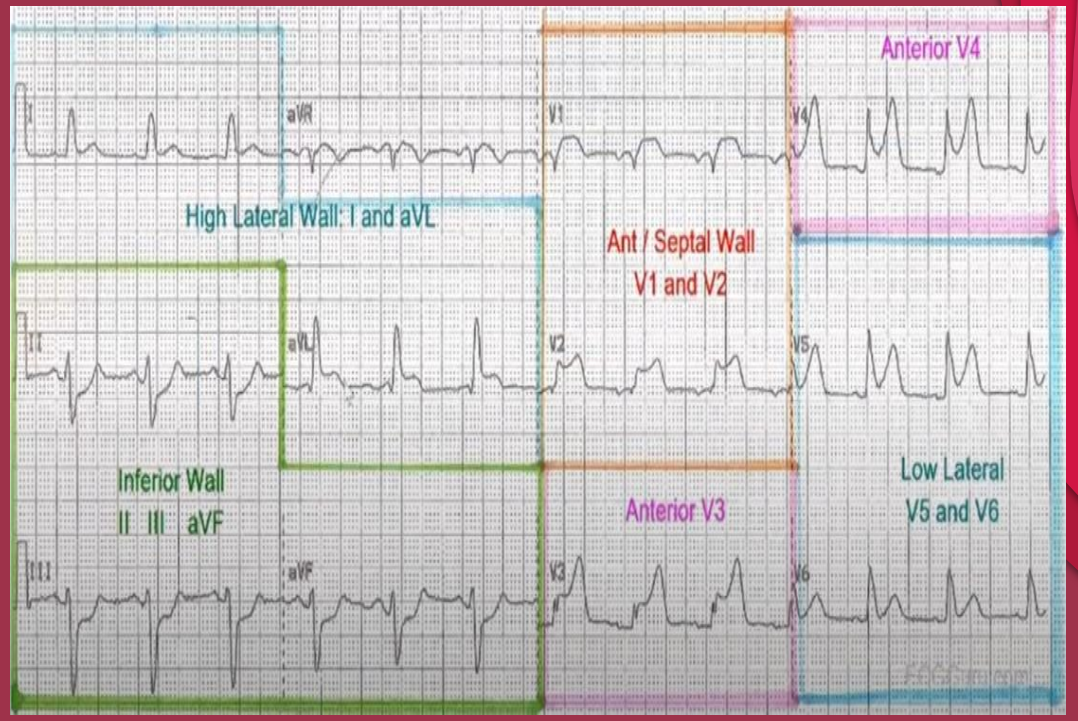
ECG Basics: Intervals

- 0.12s = 3 small squares
- 0.20s = 1 big square.
- QT interval duration varies according to the rate; so we need to correct it by calculating QTc = QT interval / √ RR interval.
- Upper limit of normal QTc:
  - Males: > 460 - 470
  - Females: > 470 - 480.
- An easy way to know it without calculations: Look at the QT interval, if it's longer than ½ RR interval, suspect QT prolongation.



ECG Basics: Ischemia

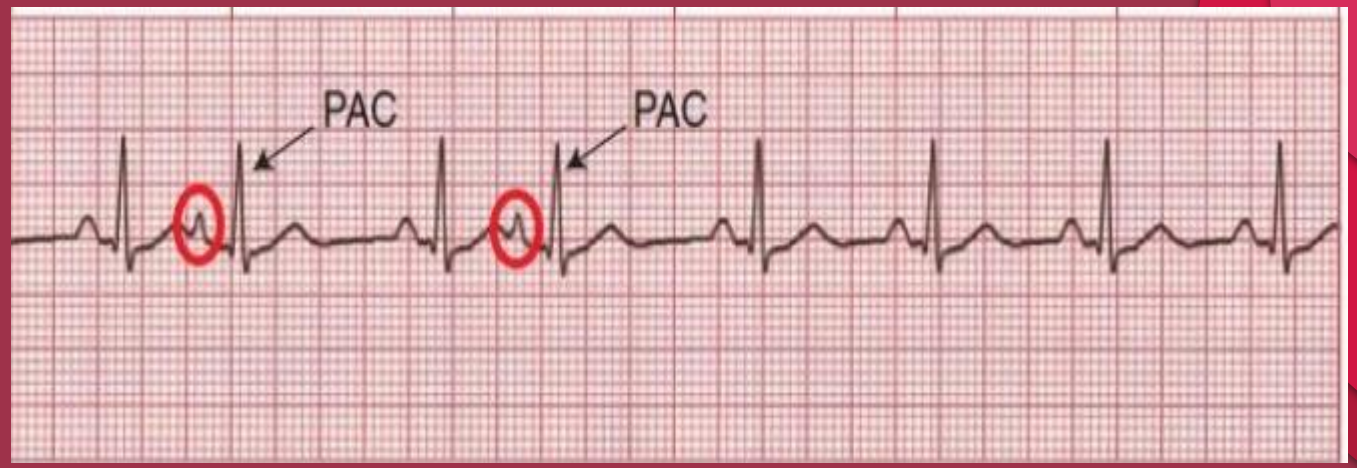




- Green Square → Hyper-Acute T wave inversion (Wellens sign); it indicates Proximal LAD occlusion / Intracranial Hemorrhage.
- Yellow Square → Hyper-acute T wave; indicates Ischemia / Hyperkalemia

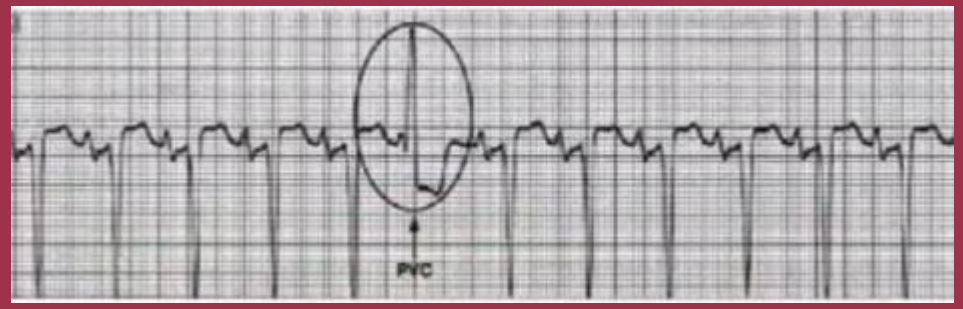
Premature Atrial Contractions / Complexes (PAC)

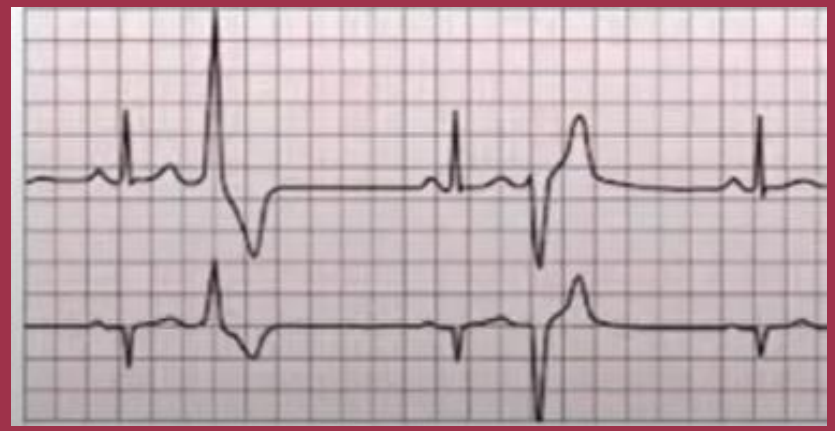
- Very common, may cause palpitations.
  - Causes: Adrenergic excess, Pharmacological, Electrolyte imbalances, Ischemia, Hypoxia, Infection.
- If the patient is asymptomatic → Just observe.
- If symptomatic (Palpitations, Fatigue, Exercise intolerance, Angina, Dizziness, Syncope) → Treat the cause + Give Beta blockers.



Premature Ventricular Contractions / Complexes (PVC)

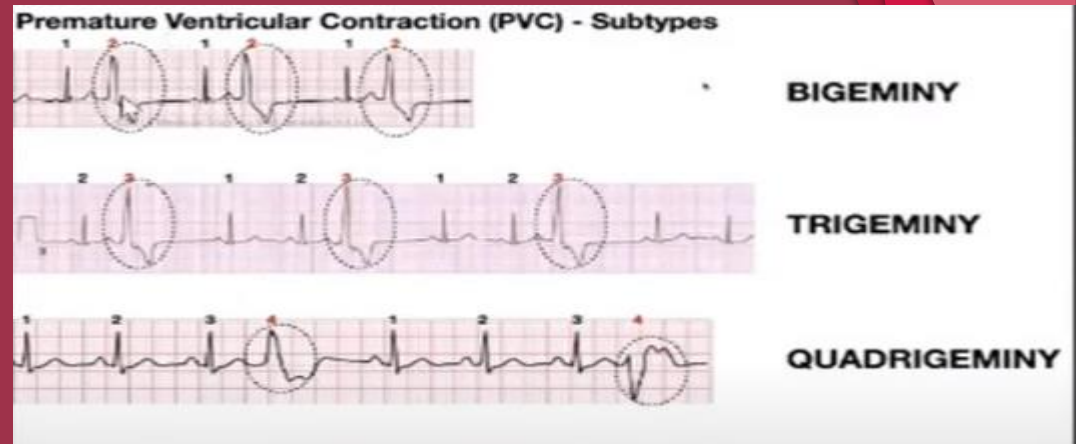
- Common, may cause palpitations.
- Causes: Hypoxia, Electrolyte abnormalities, Pharmacological, Structural Heart disease.
- When you see such an ECG; try to exclude structural heart diseases with an Echo.
- PVCs could be Monofocal (Monomorphic / one shape) or Multifocal (more than one shape).

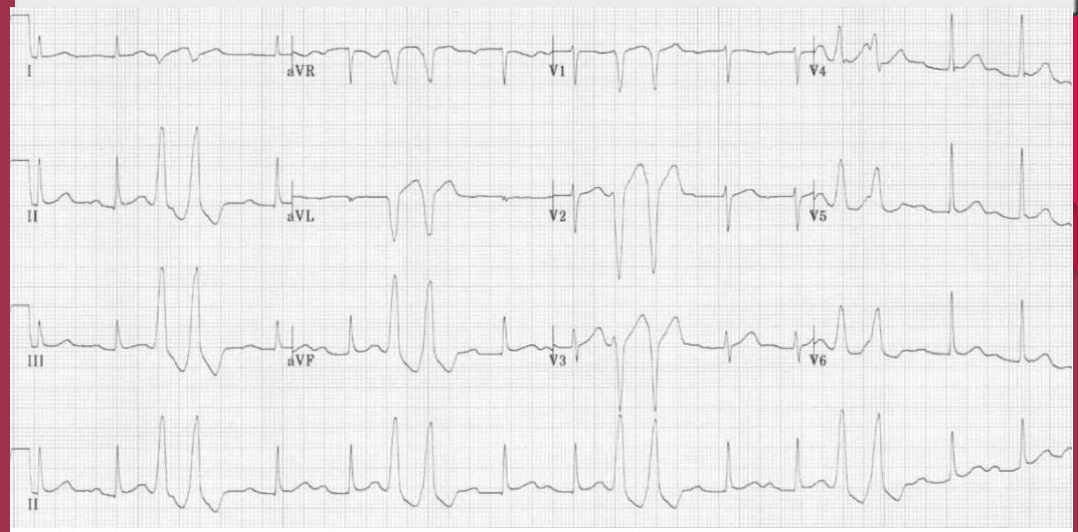


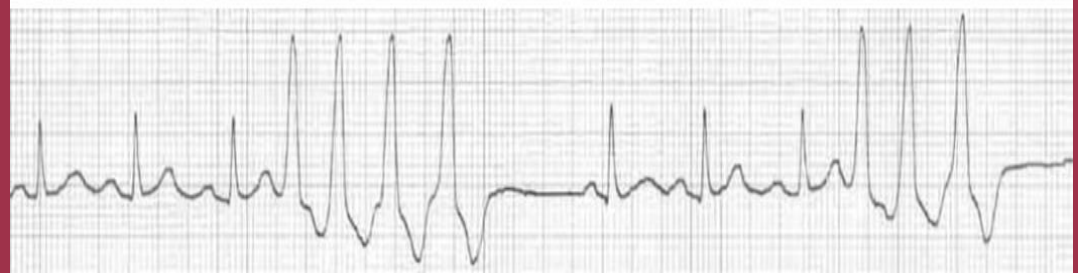


Premature Ventricular Contractions / Complexes (PVC)

- Bigeminy → means one normal beat is followed by one abnormal (ectopic) beat.
- Trigeminy → Every 2 normal beats are followed by an Ectopic beat.
- Quadrigeminy → Every 3 normal beats are followed by an ectopic beat.
- Couplets (the second figure) → one normal beat is followed by 2 ectopic beats
- We don't have triplets, because once we have 3 successive ectopic beats it becomes non-sustained Ventricular contractions (the third figure)
- Treatment:
  - Asymptomatic - Infrequent → Just observe.
  - Asymptomatic - Frequent / repetitive → Rule out Heart disease + Beta blockers + EP study +/- ICD or ablation.
  - Symptomatic (Palpitations, Fatigue, Exercise intolerance, Angina, Dizziness, Syncope) → Treat the cause + Beta blockers



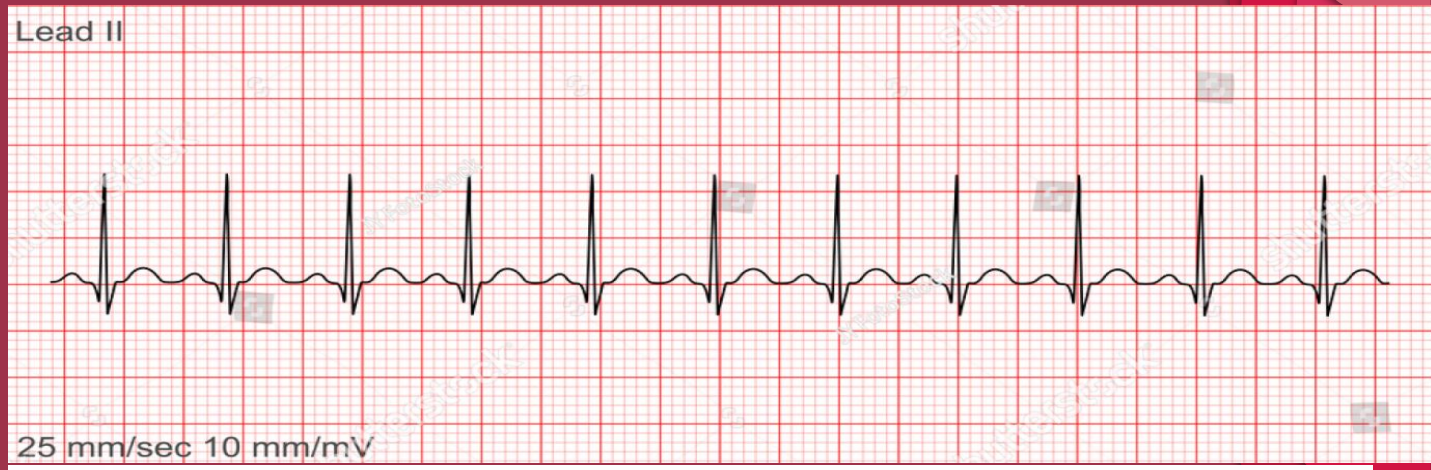




Tachyarrhythmias

Sinus Tachycardia

- Rate > 100 BPM
- Regular Rhythm (P wave is married to the QRS + Narrow QRS)
- It's the most common arrhythmia with Pulmonary Embolism.
- Management: Treat the underlying cause.
- There is an Entity of Sinus Tachycardia where patients have no underlying cause for the tachycardia, we call it Inappropriate Sinus Tachycardia; treated with Beta blockers, Ivabradine, RFA.

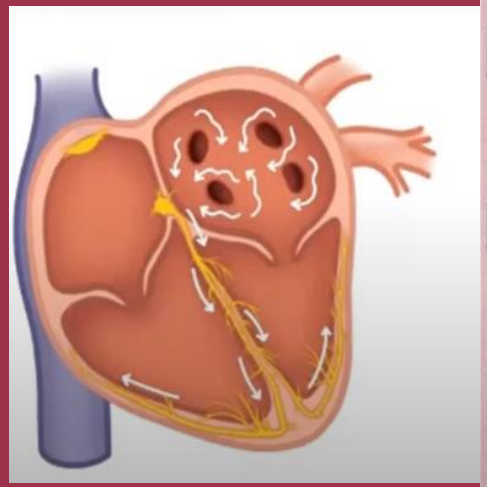


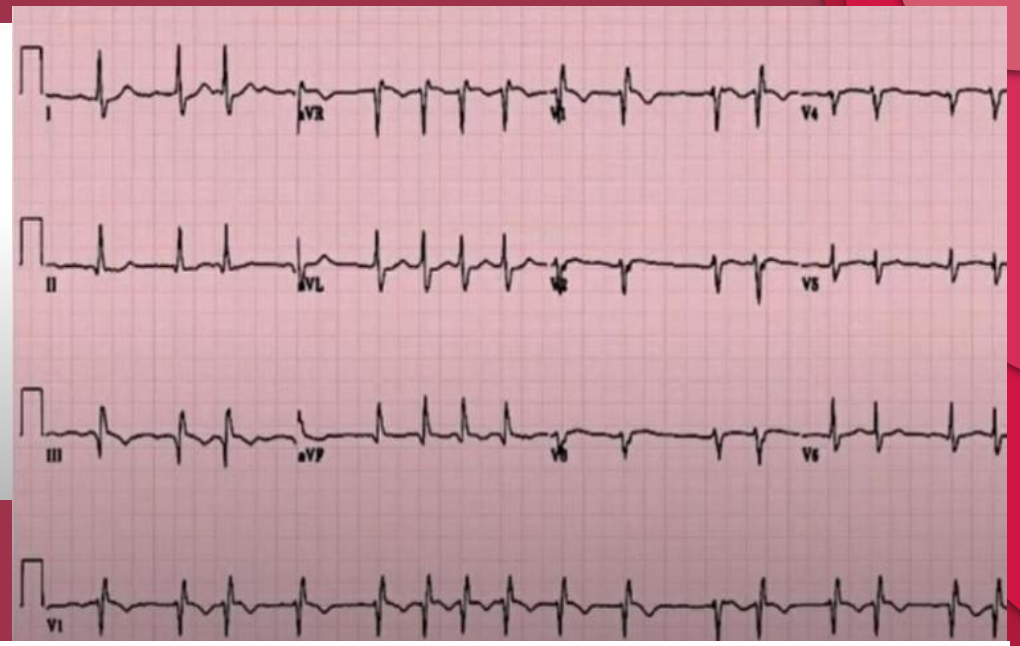
- Causes:

Physiological	Cardiac Conditions	Medical Condition	Pharmacological
• Exercise	• MI	• Shock	• Caffeine
• Emotion	• Cardiomyopathy / HF	• Hypoxia	• Alcohol
• Anxiety	• Acute Valve Disease	• Respiratory Distress	• Tobacco
• Pain	• Pericarditis	• P.E.	• Catecholamines
• Fever	• *Postural*	• Anemia	• B agonist
• Pregnancy		• Infection	• BB Withdrawal
• *Volume Depletion*		• *Dehydration*	• Vasodilators
		• Hyperthyroidism	• Atropine
		• Pheochromocytoma	• Theophylline
		• Cushing's	• Decongestants
		• Hypoglycemia	• Cocaine
		• Panic Attack	• Amphetamines
			• Thyroid Hormones

Atrial Fibrillation

- Very common.
- Notice that we have irregular rhythm, so to calculate the rate: # of R in the entire Strip X 6 = 16 X 6 = 96 BPM.
- Always keep in mind that whenever you see an abnormal ECG with a narrow QRS → then the abnormality is coming from Above the AV node.
- So in this ECG → Irregular Rhythm + Narrow QRS + Absent P wave → A Fib
- Pathophysiology: Micro-reentry around Pulmonary veins cuffs.





- Causes:
  - Heart disease: CAD, MI, HTN, mitral valve disease
  - History of cardiac surgery
  - Pericarditis
  - Pulmonary disease (PE, COPD, Hypoxia)
  - Thyroid disease
  - Pheochromocytoma
  - Systemic illness (e.g. Infection,)
  - Stress (postoperative, pain, anxiety)
  - Hyperadrenergic states
  - Cocaine or methamphetamine use
  - Extremes of activity (sedentary lifestyle, excess exercise such as marathon running)
  - Excessive alcohol intake ("holiday heart syndrome")

Atrial Fibrillation: Management

- 1- Rate control → Beta Blockers + CCBs + Digoxin.
- 2- Anticoagulation → Warfarin + DOACS.
- For Non-valvular A fib we need to determine whether they need anticoagulation or not using CHA2DS2-VASc Score; if the patient has a Score of 0 → Just give Aspirin. If the score was > 1 → Give Anticoagulation.
- We have HAS-BLED Score that is used to determine the possibility of bleeding when giving anticoagulation
- 3- If the patient despite the above therapies remains Unstable / Symptomatic / Rate uncontrolled / or he's young or with new onset → we refer to Rhythm control (Electrical, Pharmacological, Ablation)

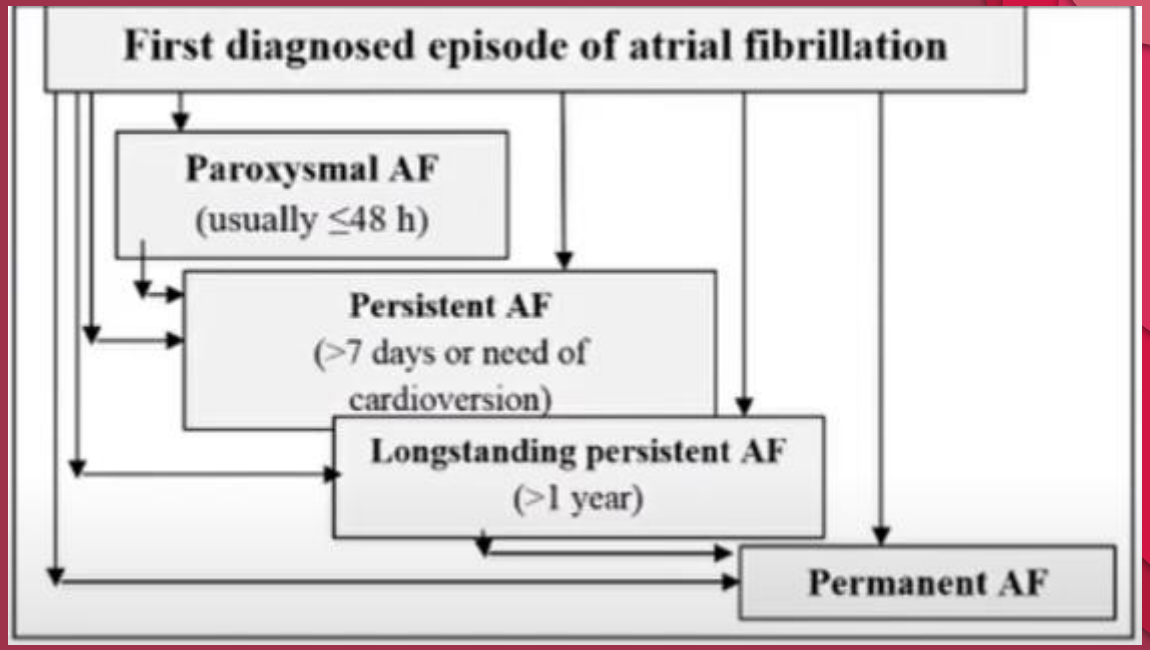
CHA2DS2-VASc Score	
CHF or LVEF ≤ 40%	1
HTN	1
Age ≥ 75	2
DM	1
CVA/TIA/TE	2
Vascular	1
Age 65-74	1
Female	1

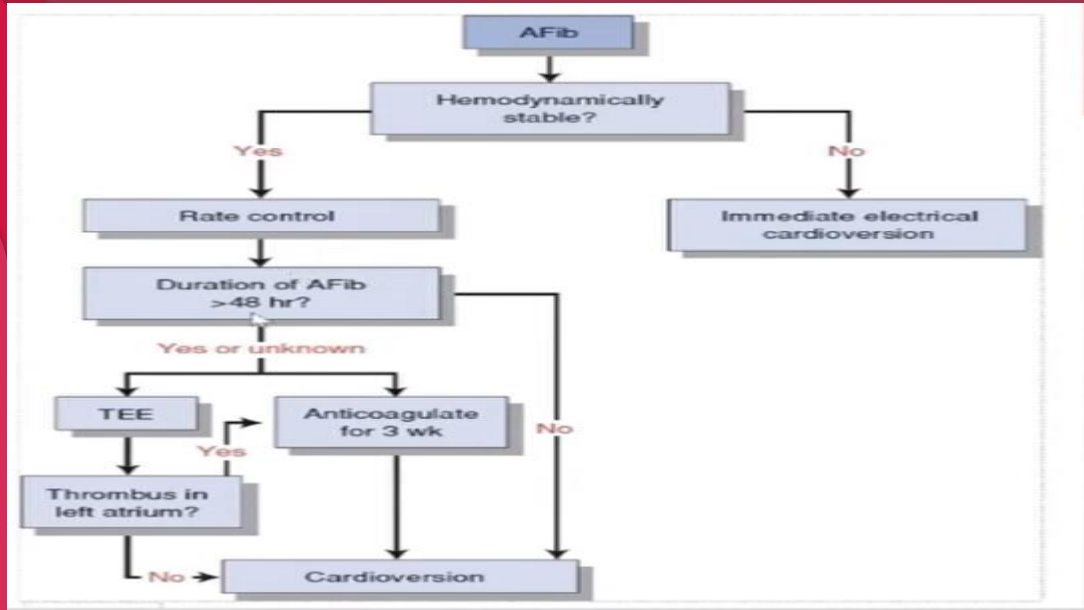
HAS-BLED score

Condition	Points
H - Hypertension	1
A - Abnormal renal or liver function (1 point each)	1 or 2
S - Stroke	1
B - Bleeding	1
L - Labile INRs	1
E - Elderly (> 65 years)	1
D - Drugs or alcohol (1 point each)	1 or 2

Atrial Fibrillation: Pearls

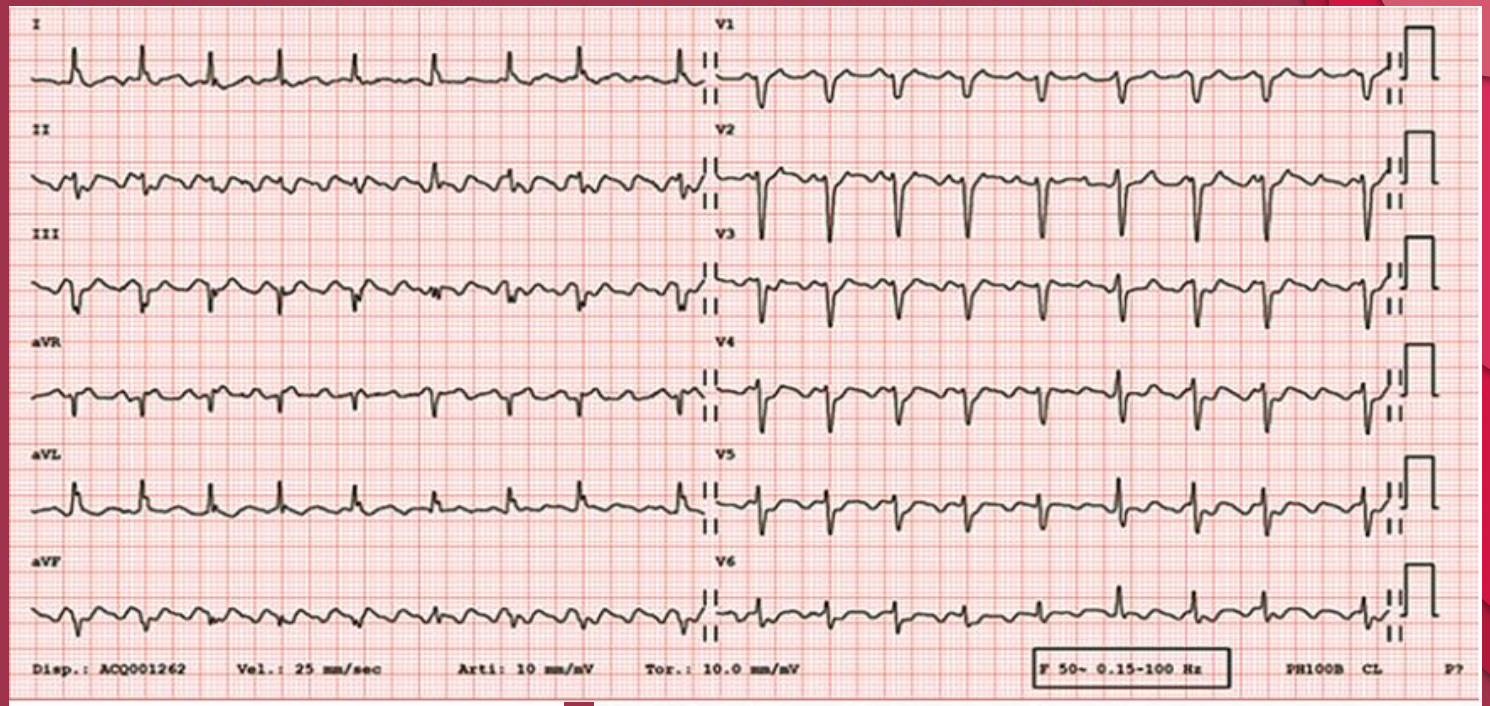
- Paroxysmal A Fib → A fib < 48 hours, it terminates on its own (no need for Cardioversion or medications).
- Persistent A Fib → Lasts more than 7 days or requires Cardioversion
- Longstanding Persistent A Fib → Lasts more than one year.
- Permanent A Fib → Doesn't respond to Cardioversion, may benefit from ablation.

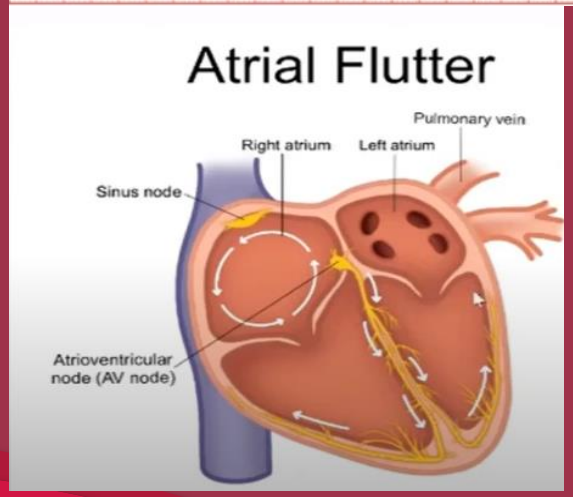


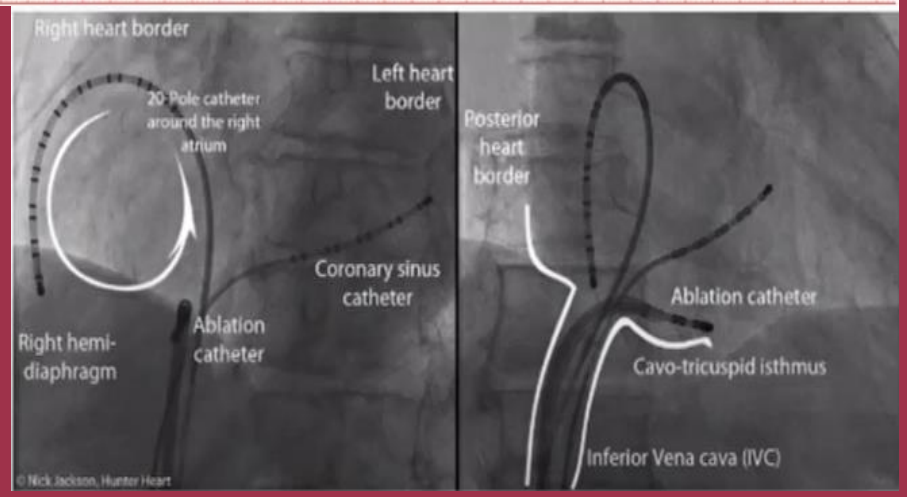


Atrial Flutter

- Common.
- Similar to A Fib except that the P / Flutter wave is more organized.
- ECG has a Saw-tooth appearance.
- Notice on the ECG that there is no normal baseline.
- Unlike A Fib, it's regular; either 2:1 or 3:1 conduction (one beat conducted after 2 or 3 missed beats)
- Management: Exactly the same as Atrial Fibrillation
- There is a small Caveat between A Fib and A Flutter → In A Flutter the reentry isn't microcirculation around Pulmonary veins cuffs, instead it's a microcircuit in the Right Atrium
- We go toward Ablating A flutter more frequently than A fib because it's easy, efficient, has lower risk.

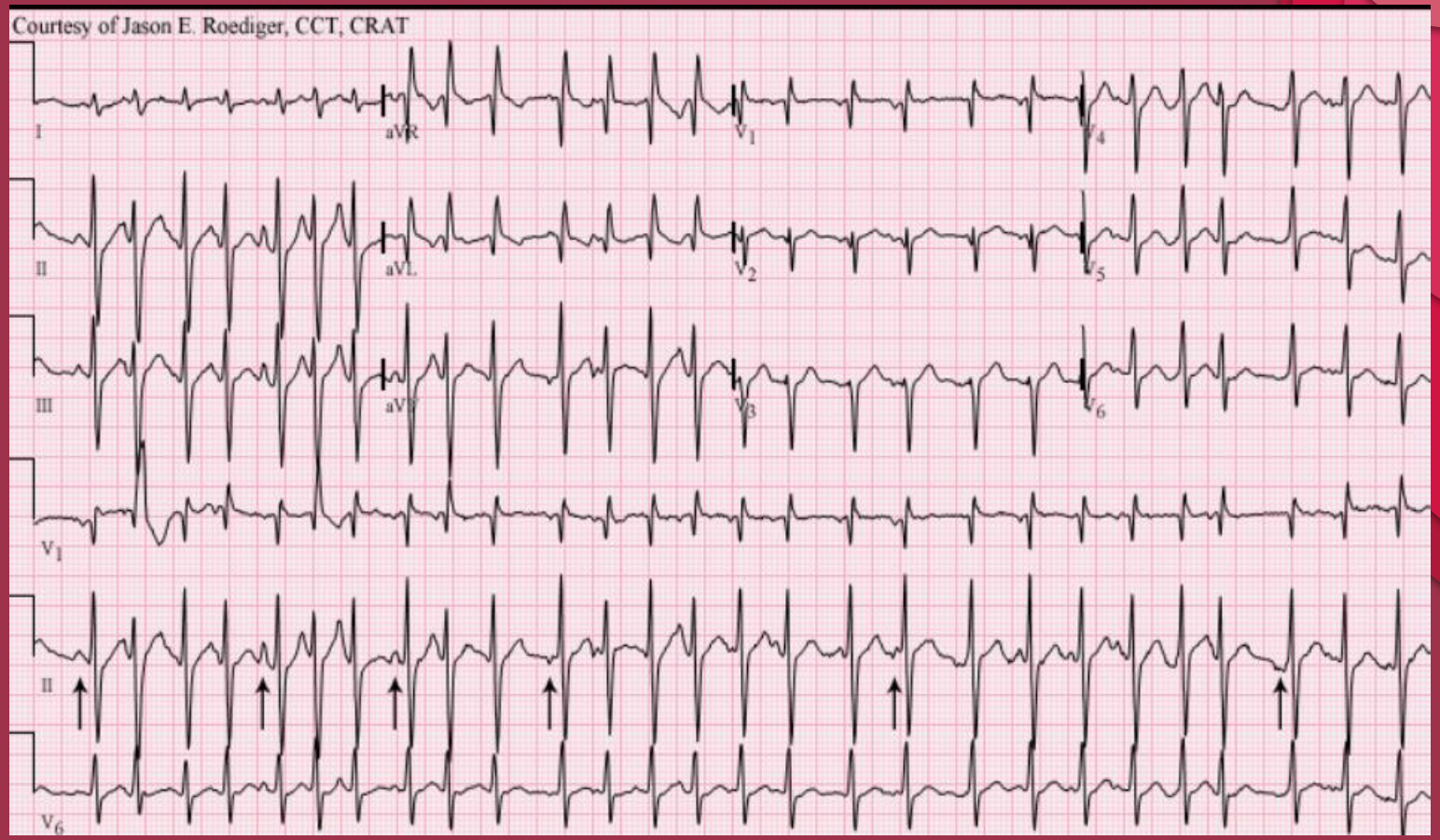






Multifocal Atrial Tachycardia (MAT)

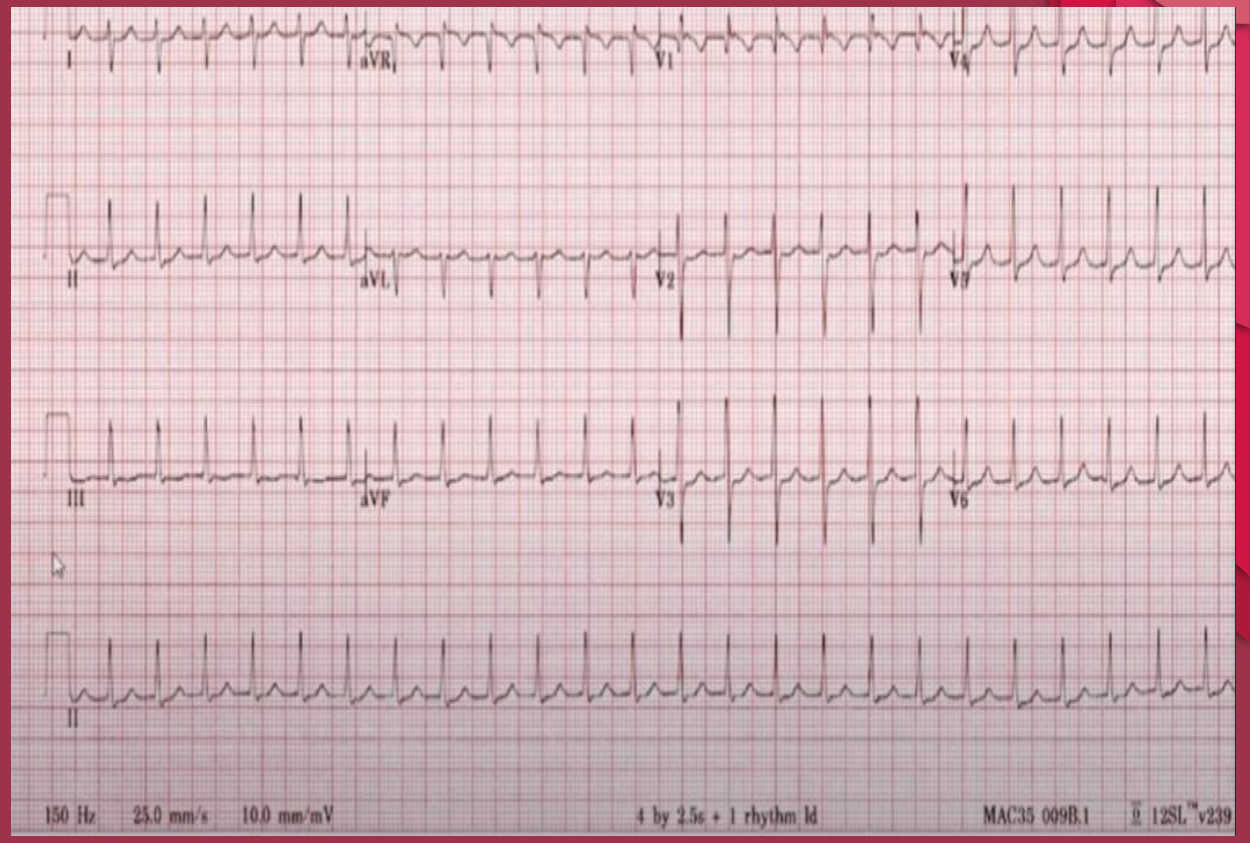
- Common in patients with Severe Pulmonary Disease (e.g., COPD)
- It looks like A Fib (irregular, Narrow QRS, Tachycardic), but if you took a closer look, you'll see that P waves are present.
- Treatment: Improve Oxygenation and Ventilation + Medications: CCBs, Beta blockers, Digoxin, Amiodarone.
- Electrical cardioversion is ineffective and shouldn't be used.

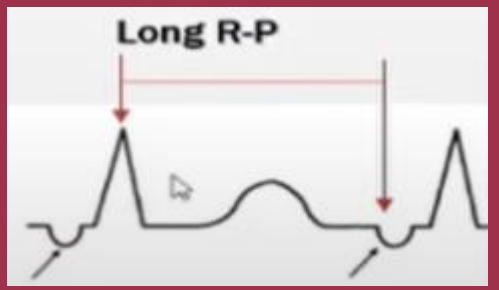


Supraventricular Tachycardia (SVT)

- On ECG: Tachycardia + Narrow QRS complex + Regular; now you have to think of Atrial Flutter OR SVT.
  - A Flutter ECG has saw-tooth appearance.
- So, in this ECG we don't have a saw-tooth appearance → then it's SVT
  - SVT is divided into:

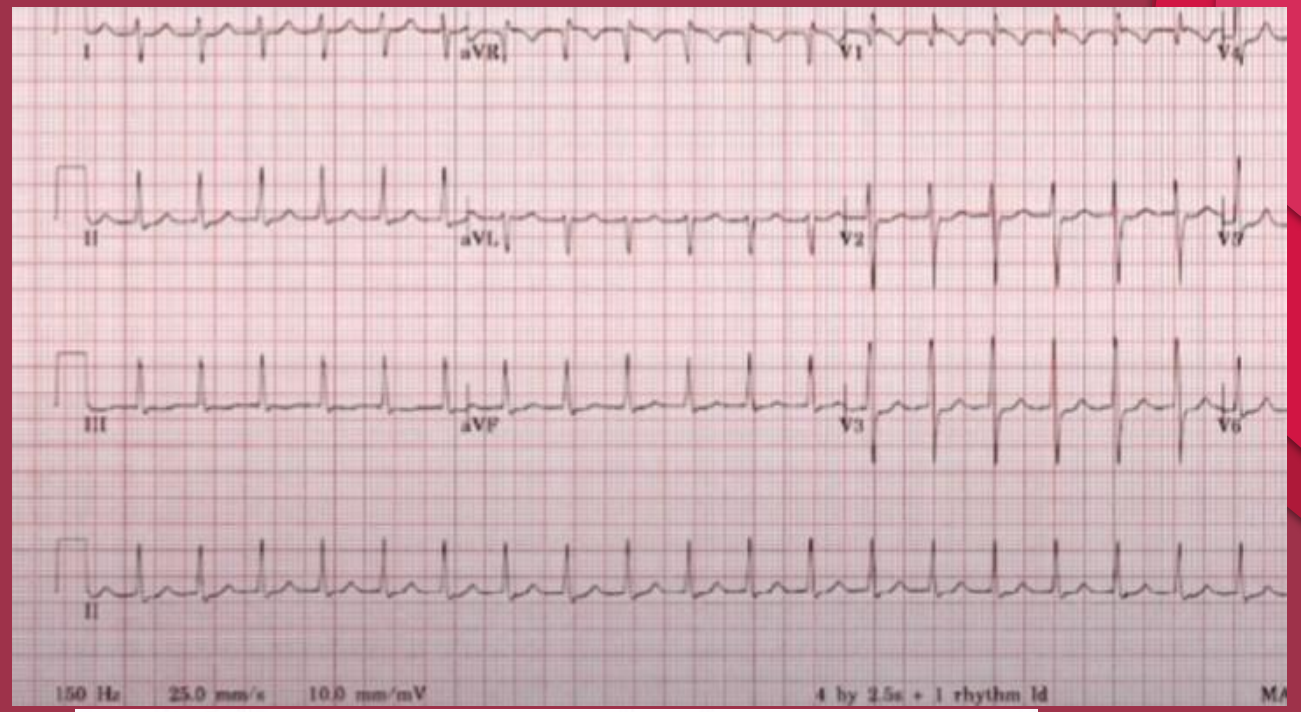
- Short RP Tachycardia: The distance between R-P is shorter than P-R; DDx: AV nodal re-entry Tachycardia (most common cause of SVT) / Junctional Tachycardia / Orthodromic Atrioventricular Tachycardia / Atrial Tachycardia
- Long RP Tachycardia: The distance between R-P is longer than P-R; DDx: Sinus Tachycardia / Atrial Tachycardia / Atypical Orthodromic AV Tachycardia / Atypical AV nodal re-entry Tachycardia / Junctional Tachycardia.

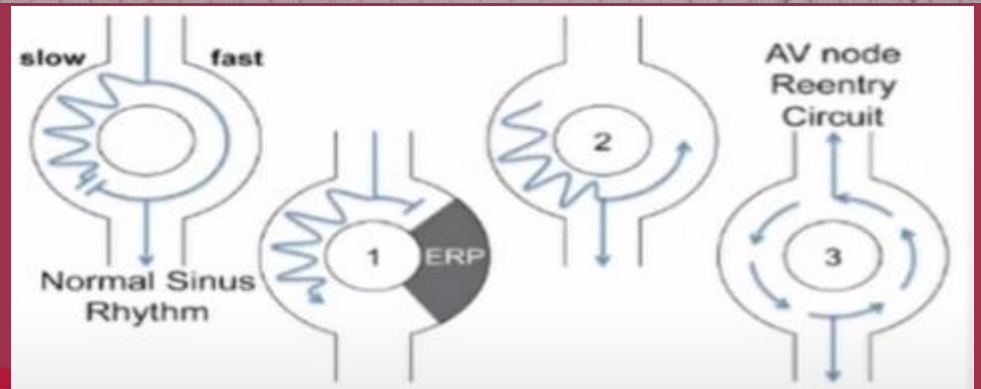




SVT: AV nodal Re-entrant Tachycardia (AVNRT)

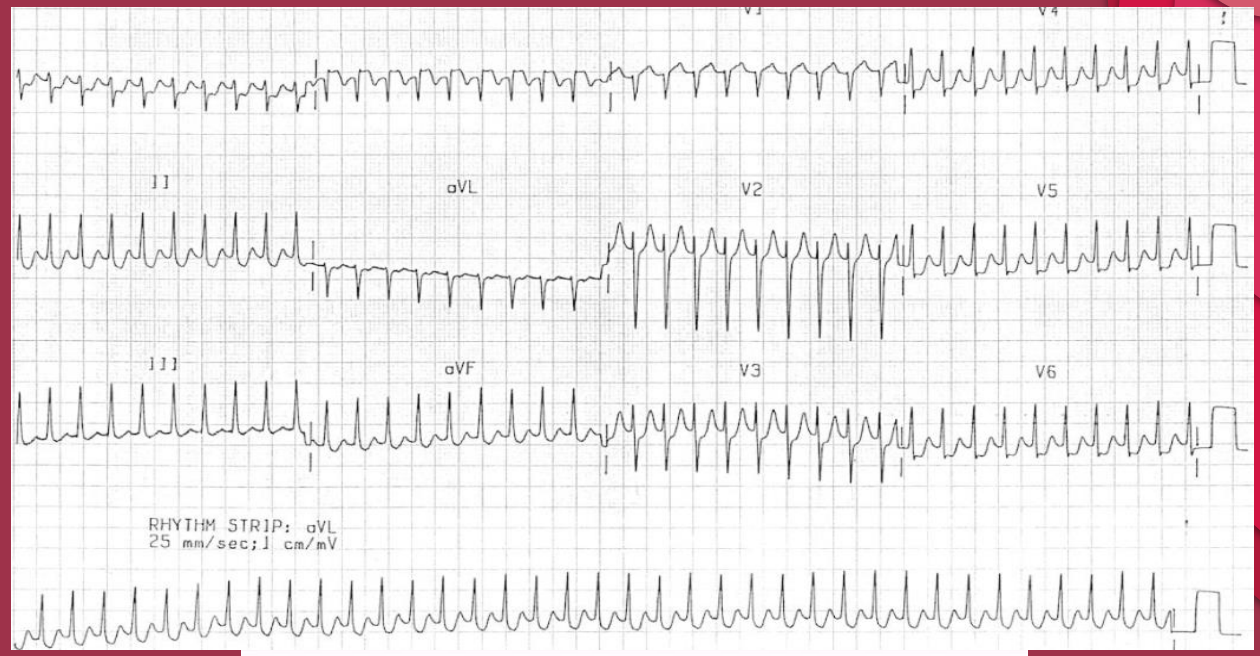
- The most common cause of SVT.
- The Re-entry circuit in this situation is located inside the AV node itself.
- The AV node has 2 physiologies: Fast (repolarizes Slowly) and Slow (repolarizes quickly); so what happens is that every now and then an ectopic beat comes and find the Fast pathway unready (in repolarization) so it enters the short pathway, and by the time it reaches the end of this pathway, it finds the Fast Pathway ready and enters that pathway so there will be an endless circuit.
- Management: Acute management (Vagal Maneuvers / Pharmacologic therapy with IV adenosine or CCBs or Beta blockers / DCCV) + Preventive management (CCBs or Beta blockers / Ablation)

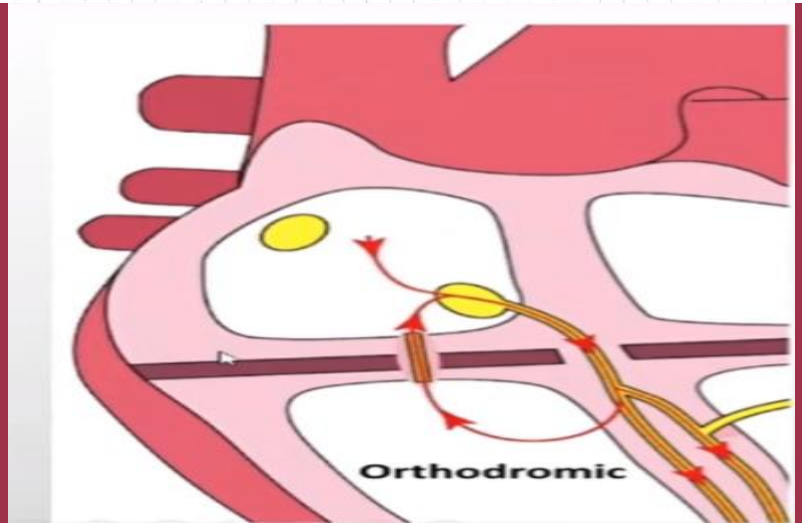




SVT: Orthodromic AV Re-entrant Tachycardia

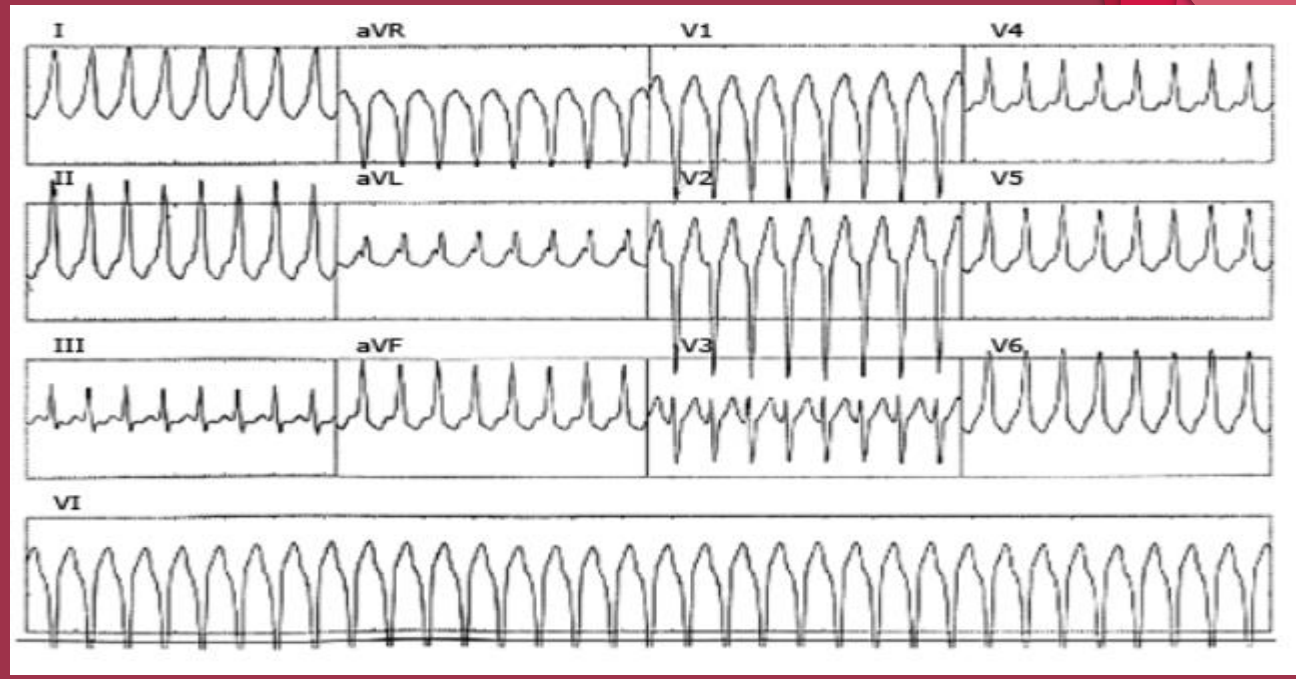
- Here we have normal conduction down the AV nodal system, then the beat enters the accessory pathway, and it goes back up into the atria creating a circuit (Macro re-entry).
  - It's called orthodromic, because the conduction is through the normal conduction system, and it goes back retrogradely in the accessory pathway
- Management: similar to the previous one.

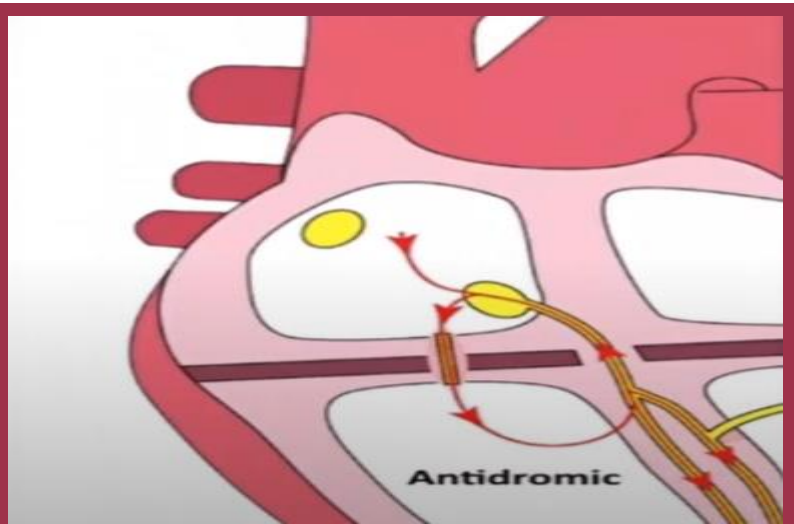




SVT: Antidromic AV Re-entrant Tachycardia (AVRT)

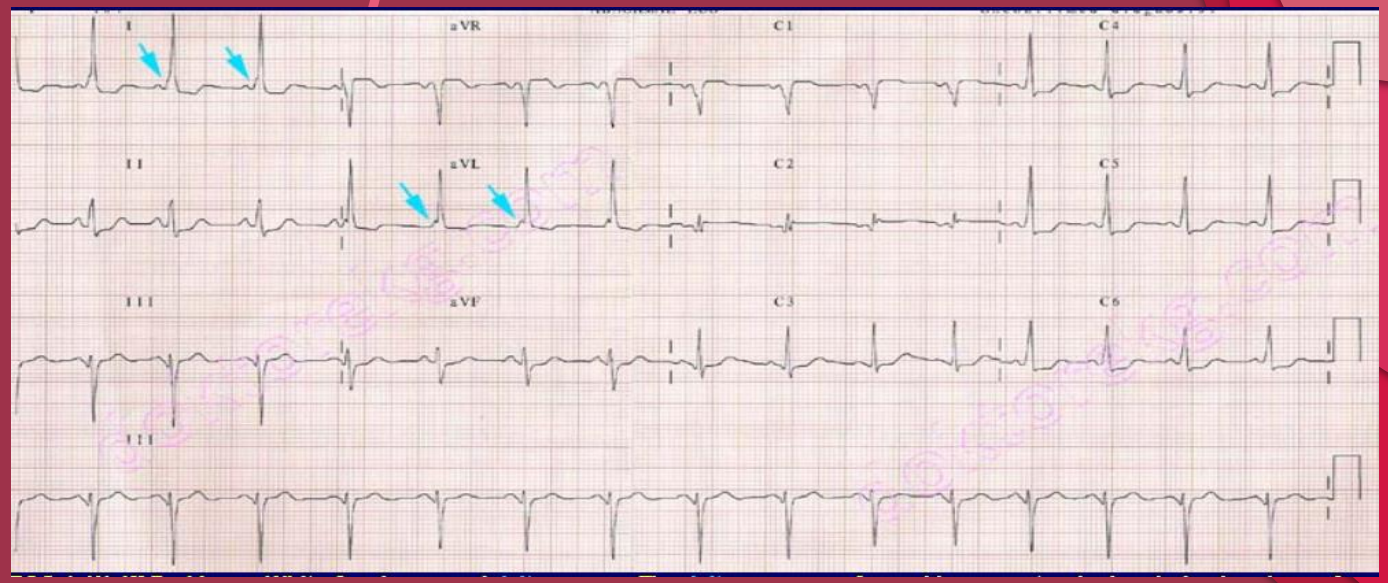
- In this case, instead of conducting through the normal system, it passes antegrade through the accessory pathway, then goes back up through the normal conduction system.
- Because it goes back up in the normal system (not down), it's not a narrow QRS Tachycardia; it's a wide QRS Tachycardia

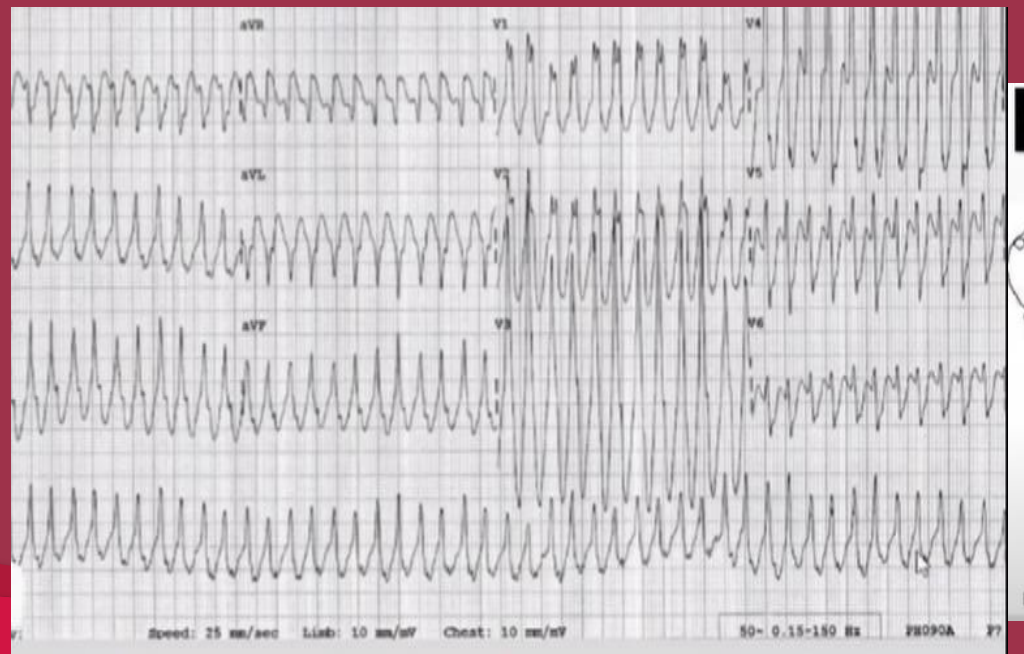


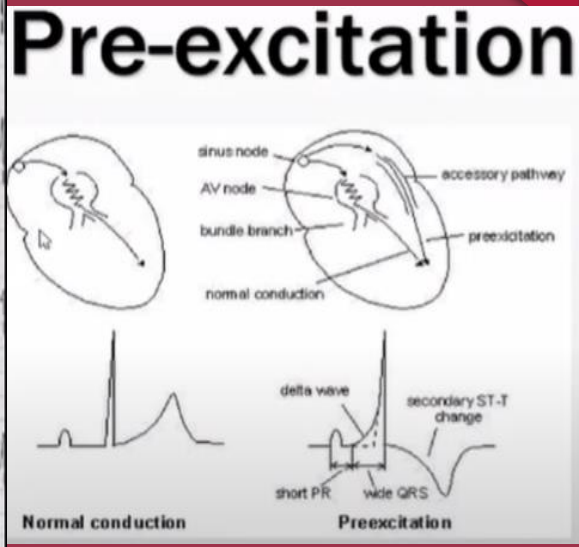


SVT: Wolf Parkinson White Syndrome (WPW)

- This is an Accessory Pathway that conducts Antegrade.
- When the electricity comes from the SA node it has 2 options; either going through the normal pathway or through the accessory pathway → this causes Pre-excitation (on the ECG appears as Delta wave)
- Initially it doesn't cause anything, but sometimes since the patient has an accessory system that can conduct antegrade, the patient may have AVRT.
- So, Pre-excitation + SVT = WPW
- The AV node normally by default slows down the conduction when HR increases, but in the case of pre-excitation where you can conduct everything from top to bottom that can be life threatening.
- For example: if the patient has A fib with Pre-excitation, all the fibrillation activities may pass down causing Ventricular Fibrillation
- Management: Similar to the previous ones, but in this case we don't use CCBs, Beta blockers, or Digoxin (because they slow down the AV node and force everything to go down the accessory pathway and increase the arrythmia); instead we use Procainamide because it slows both pathways.



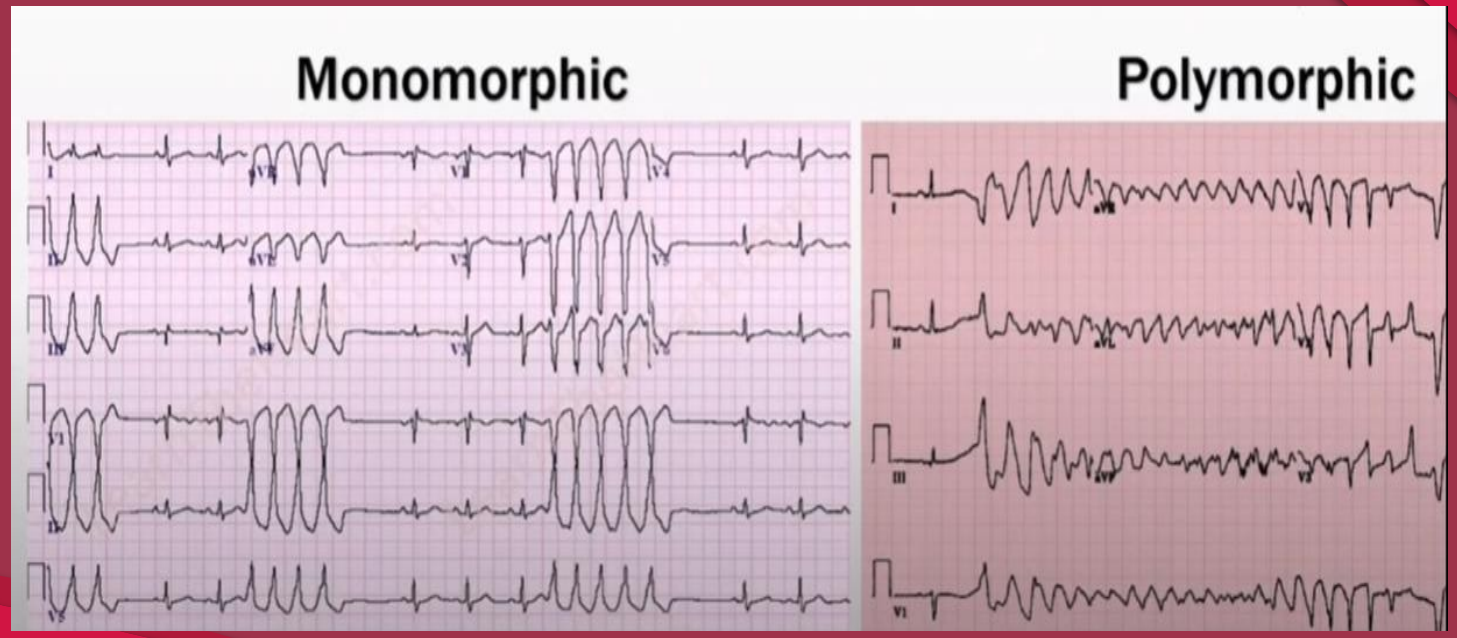




Ventricular Tachycardia

- Non-Sustained Ventricular Tachycardia: < 30 seconds.
  - If more than 30s → Sustained.
  - V Tach can be Monomorphic or Polymorphic (differ in management).
- Polymorphic is more commonly associated with Prolonged QT issues or Hypomagnesemia.
  - If Polymorphic for more than 30s → Torsades de Point

- Causes:
  - Ischemia
  - CAD with prior MI is the most common cause
  - Cardiomyopathies
  - Ventricular scar tissue
  - Congenital defects
  - Long QT syndrome
  - Electrolyte Abnormalities
  - Drug toxicity (antiemetics, antipsychotics, SSRIs, TCAs, macrolide and fluoroquinolone antibiotics)



Ventricular Tachycardia: Sustained VT

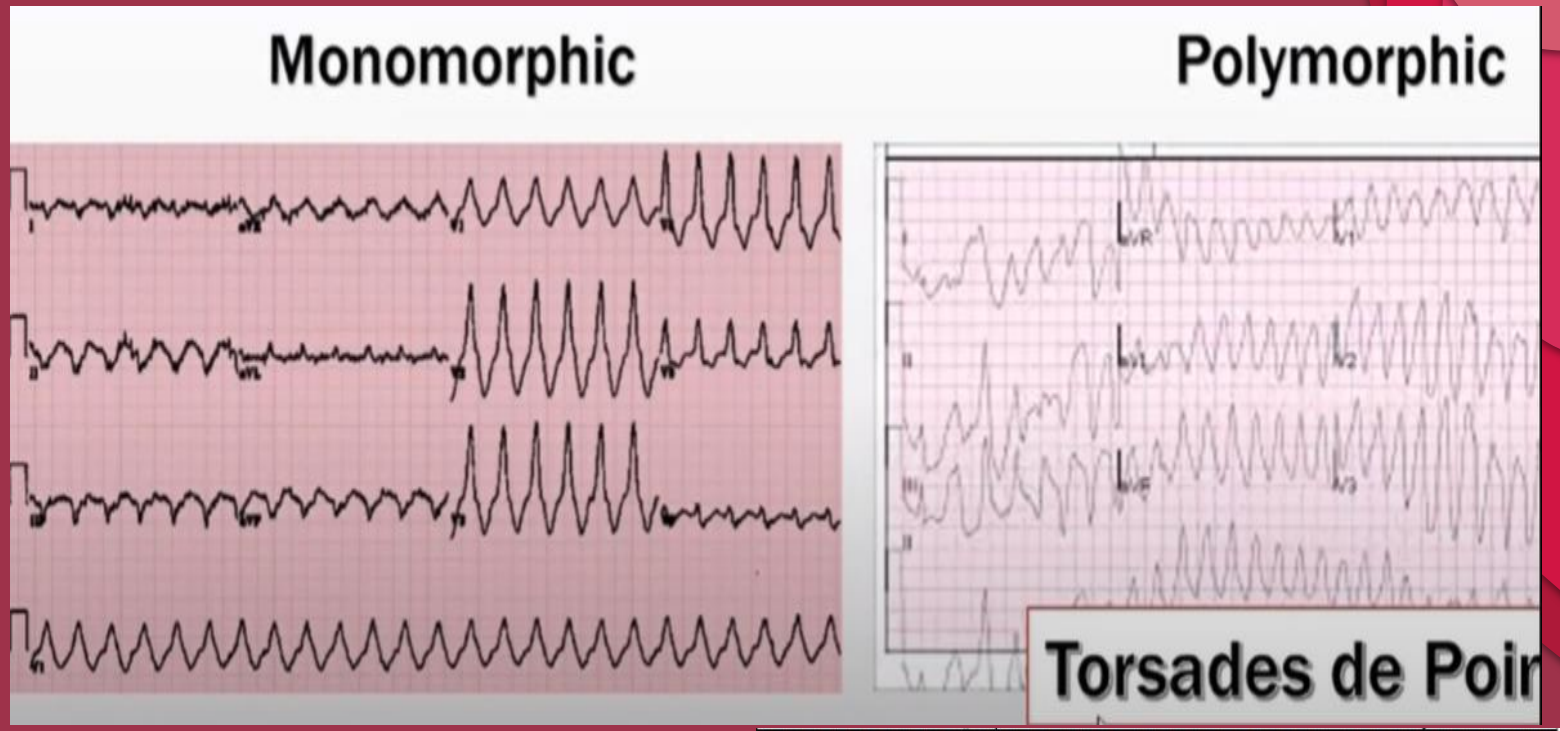
- Torsades de Point: Life threatening, happens before cardiac arrest / Ventricular fibrillation.
- If the V Tachycardia is Stable → Treat with medications

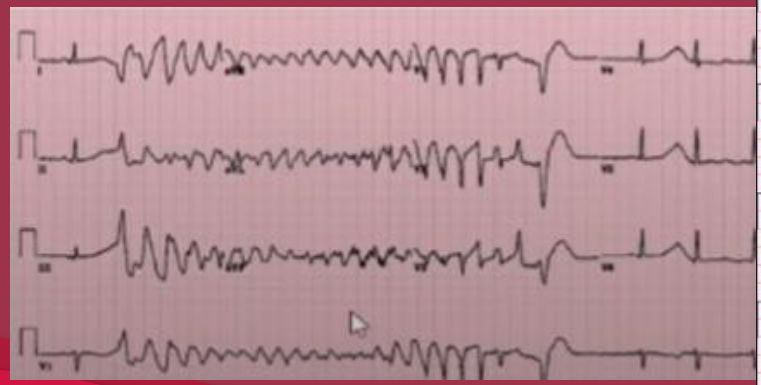
Acute management: Treat the underlying cause (Ischemia, correct electrolytes abnormalities, Remove drug +/- antidote) + IV amiodarone + DCCV

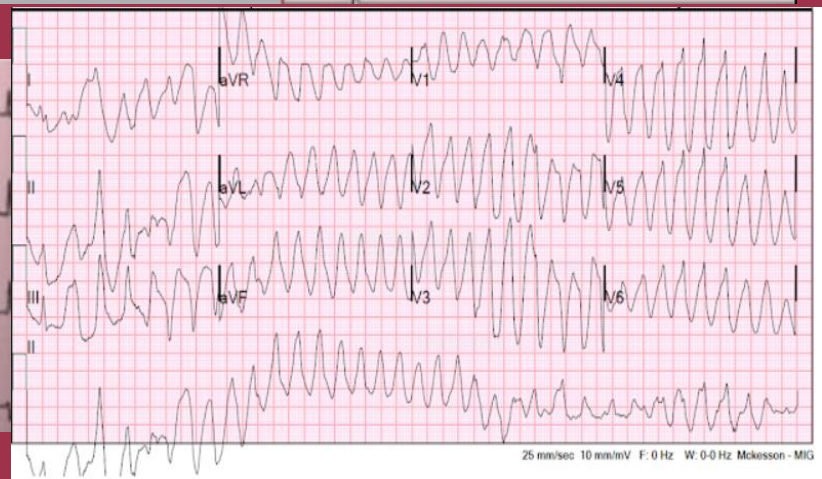
Preventive management: Consider ICD / EPS

If unstable → Cardioversion

The lower most ECGs → Torsades de Point.





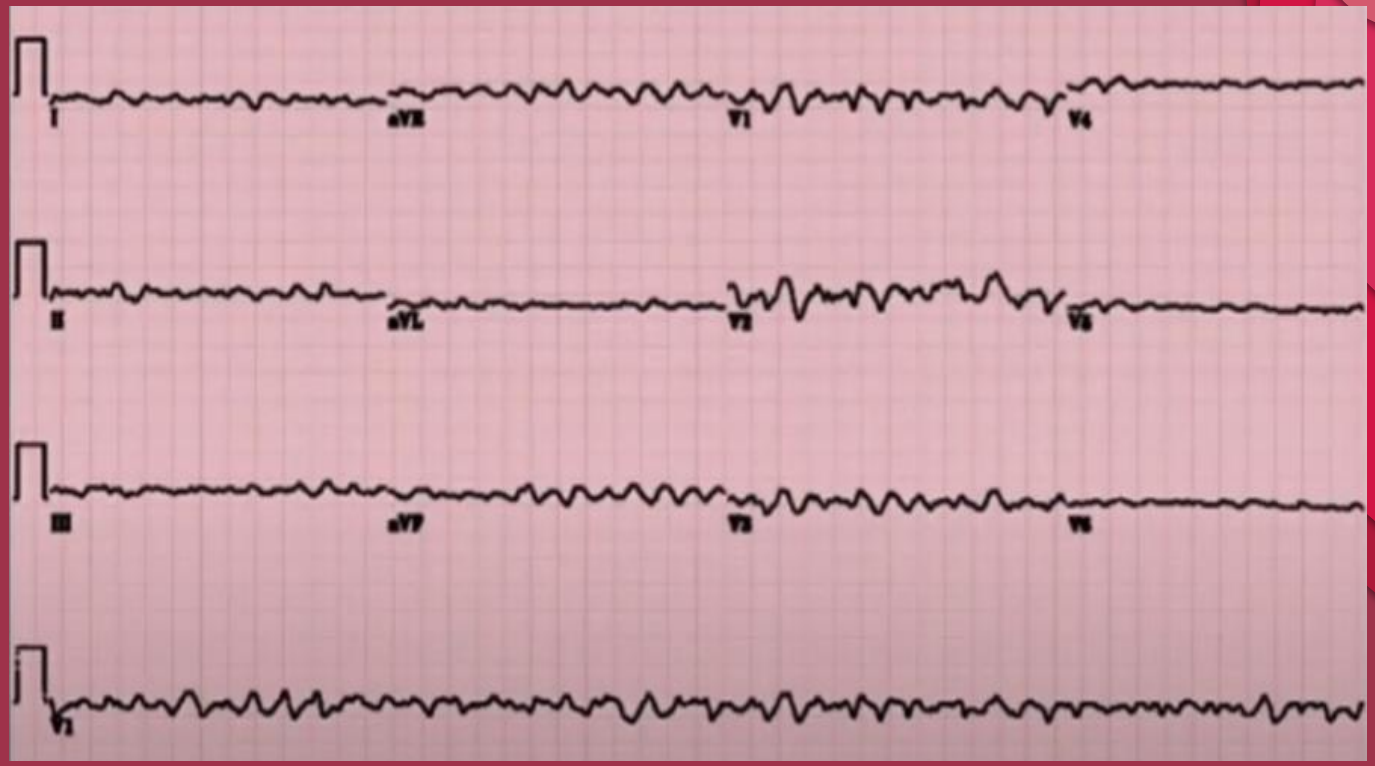


Ventricular Fibrillation

- Code Blue, Medical Emergency, You need to Defibrillate immediately.
- The same thing applies to Unstable Sustained VT.
- The first thing you do when you see such an ECG is checking whether the patient is awake or not, if he is awake then it's an artifact.
- Management:

Acute management, Post ROSC: Treat the underlying cause + Pharmacologic therapy (IV amiodarone / alternatives like Lidocaine, Magnesium, Procainamide) + DCCV

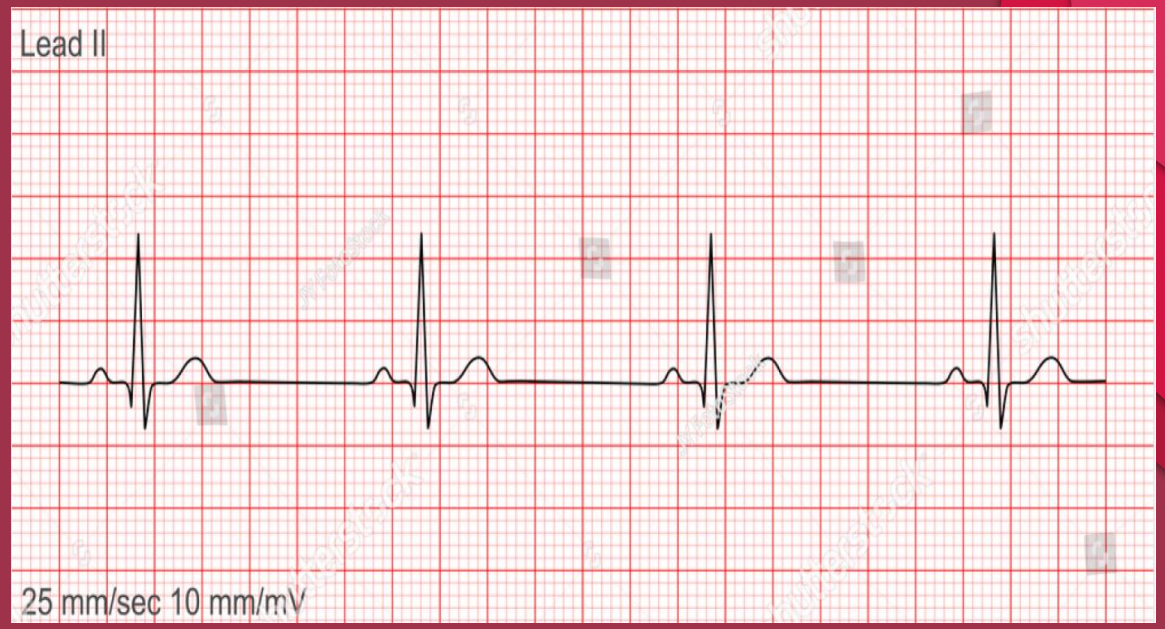
Preventive management: ICS + EPS



Bradyarrhythmias

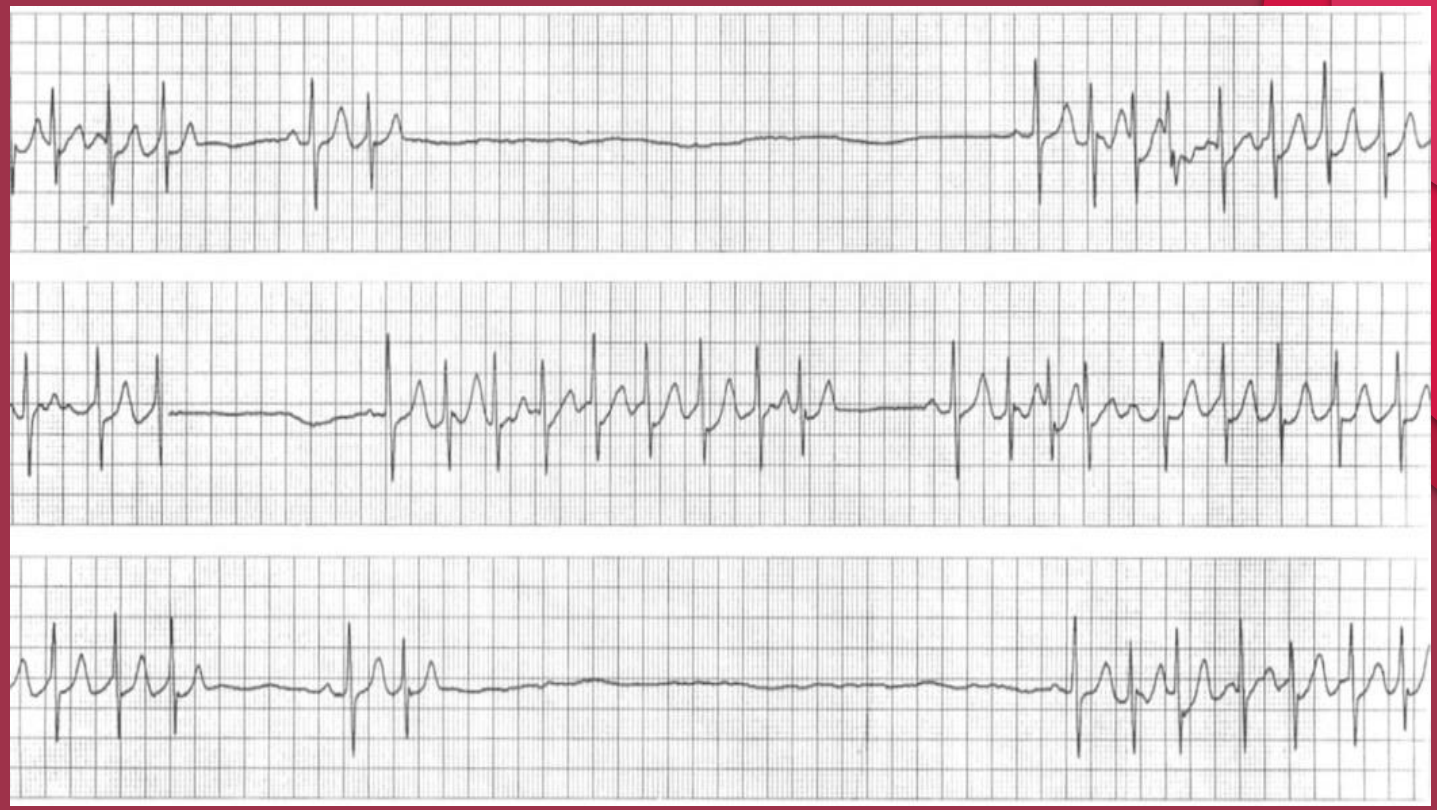
Sinus Bradycardia

- Rate < 60 BPM
- On ECG: Sinus node + Narrow QRS + Regular.
- Causes: Ischemia / Increased Vagal Tone / Structural Heart Disease (infiltrative, IE, ACHD) / Medications / Athletes.
- If Asymptomatic → Just Observe.
- If Symptomatic (Fatigue, Exercise intolerance, Angina, Dizziness, Syncope) → Treat the cause / Atropine or Beta agonist / Pacemaker



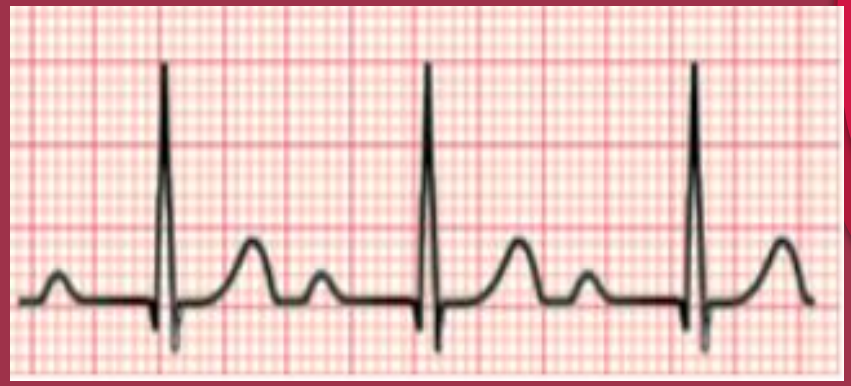
Sick Sinus Syndrome (SSS)

- Sino-nodal Dysfunction → Extra Beats + Pauses.
- Occurs along with Sinus Bradycardia.
- Occurs with advanced age.
- Marked persistent Sinus Bradycardia.
- SA Pauses and Blocks.
- Frequently associated with Tachy-Brady Syndrome (they start slow and after the pause they go fast).
- Usually co-exists with AV nodal disease.
- Treatment: Pacemaker placement.



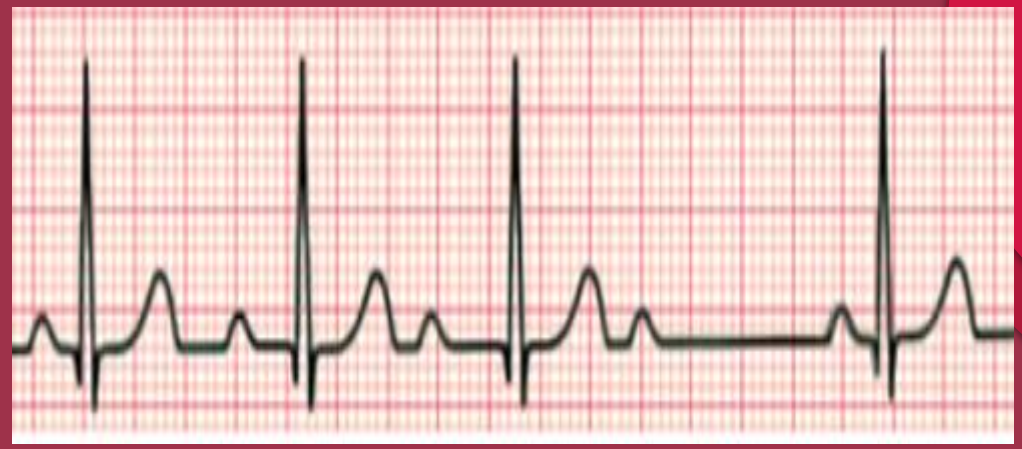
1st Degree AV Block

- Almost normal; the only thing that is abnormal is PR interval duration.
- Prolonged PR interval > 0.2 second (more than 1 large square).
- No dropped beats (No P without QRS).
- Causes: Ischemia, Increased Vagal Tone, Structural Heart Disease, Medications.
- Management:
  - If Asymptomatic → Just observe.
  - If Symptomatic (Fatigue, Exercise Intolerance, Angina, Dizziness, Syncope) → Treat the cause + Atropine / B agonist + Pacemaker.



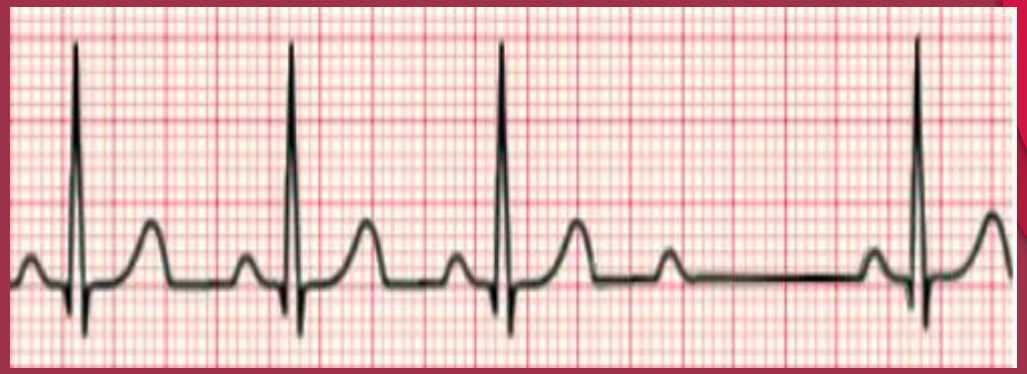
2nd Degree AV Block - Mobitz Type 1 (Wenckebach)

- Progressive PR prolongation followed by a dropped QRS.
- Causes similar to 1st degree block.
- Management similar to 1st degree block



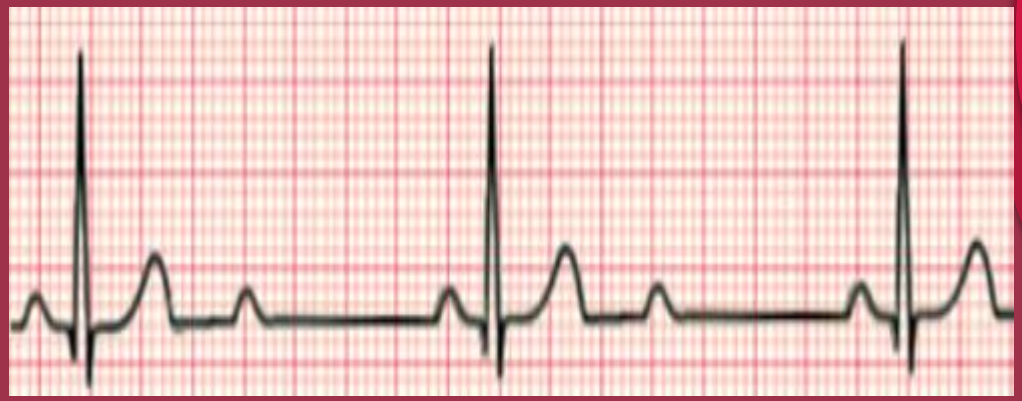
2nd Degree AV Block - Mobitz Type 2

- P waves fail to conduct suddenly without a preceding PR interval prolongation; therefore, the QRS drops suddenly.
- It almost looks like first degree, but it has higher risk (the abnormality arise from below the bundle of His)
- Management: Pacemaker placement is indicated whether symptomatic or asymptomatic



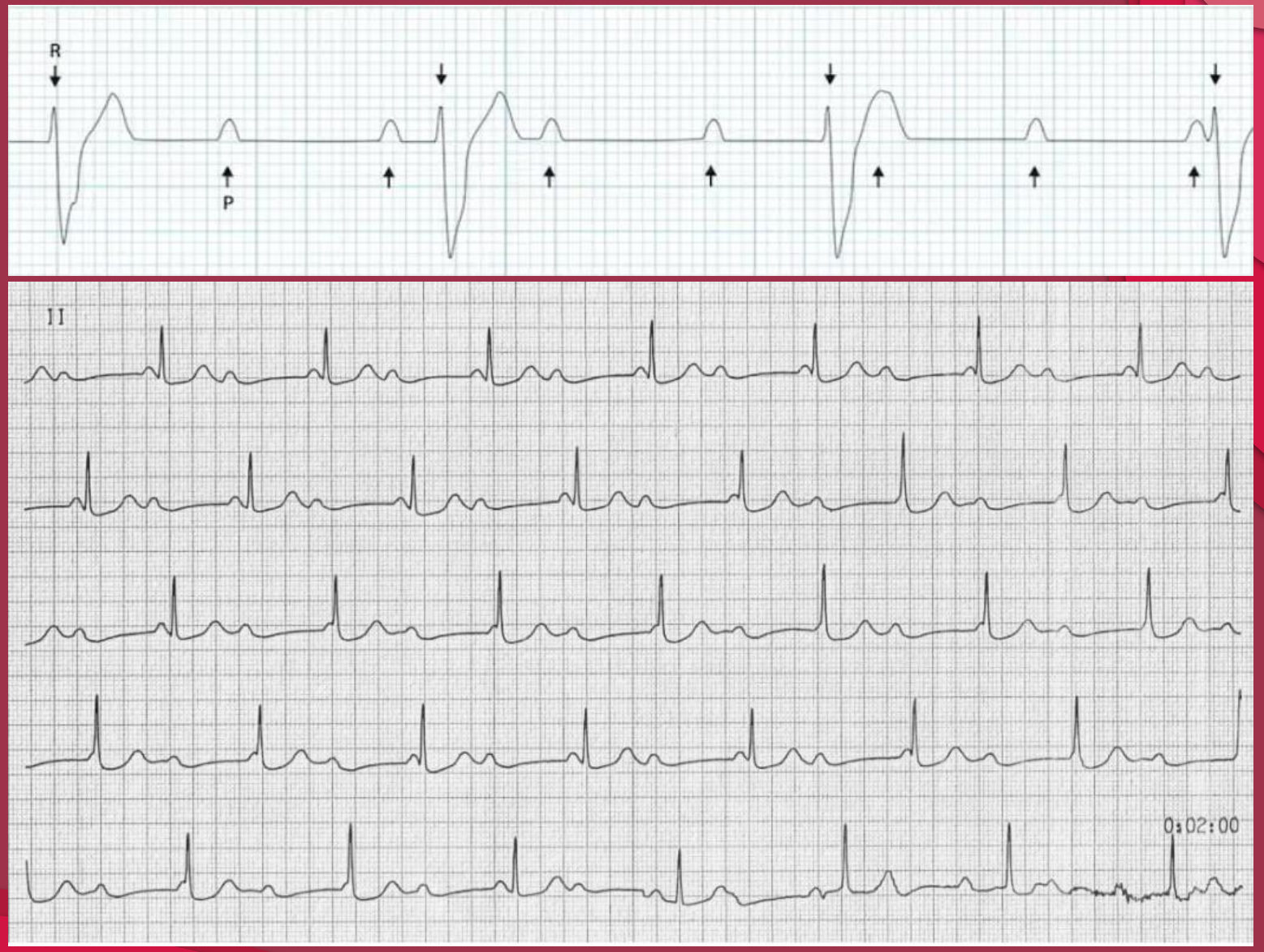
2nd Degree AV Block - (2:1) Block

- Alternating conducted QRS followed by a dropped QRS; i.e., one normal beat followed by an abnormal beat.
- In this case we can't determine whether it's Mobitz type 1 or type 2 because we can't tell if there is a prolongation or not
- Management: The patient needs further evaluation with more prolonged monitoring.



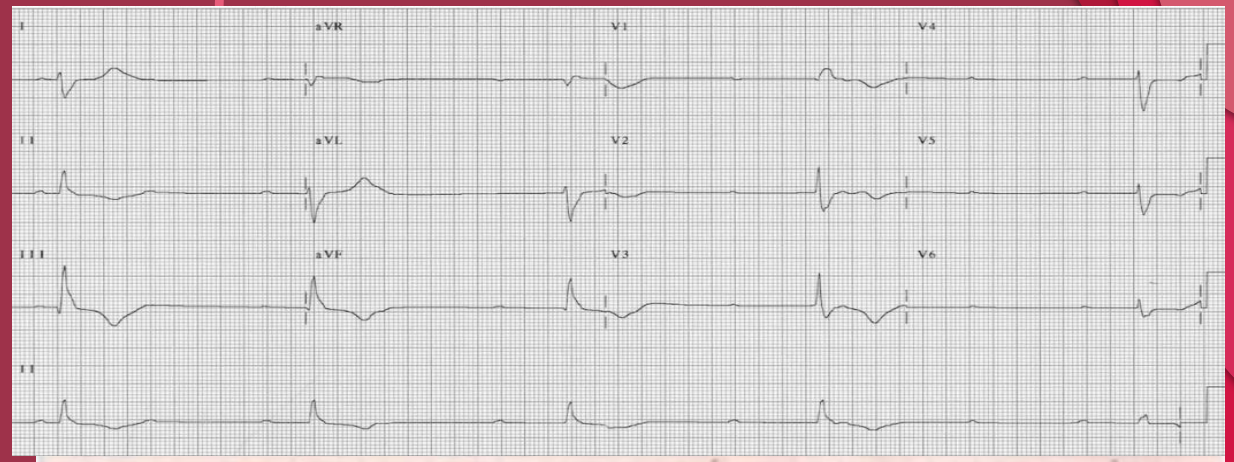
3rd Degree AV Block - Complete Heart Block

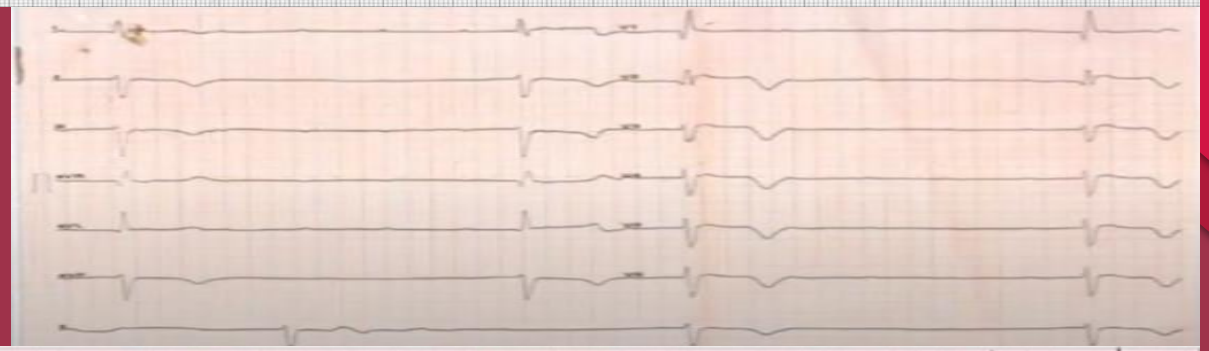
- Complete dissociation between P waves and QRS complexes.
  - # of P waves > # of QRSs
- QRSs could be narrow (if they arise from the junction - bundle of his-) or wide (if they arise from the ventricles)
- Management: A medical Emergency that requires an emergent pacemaker placement.

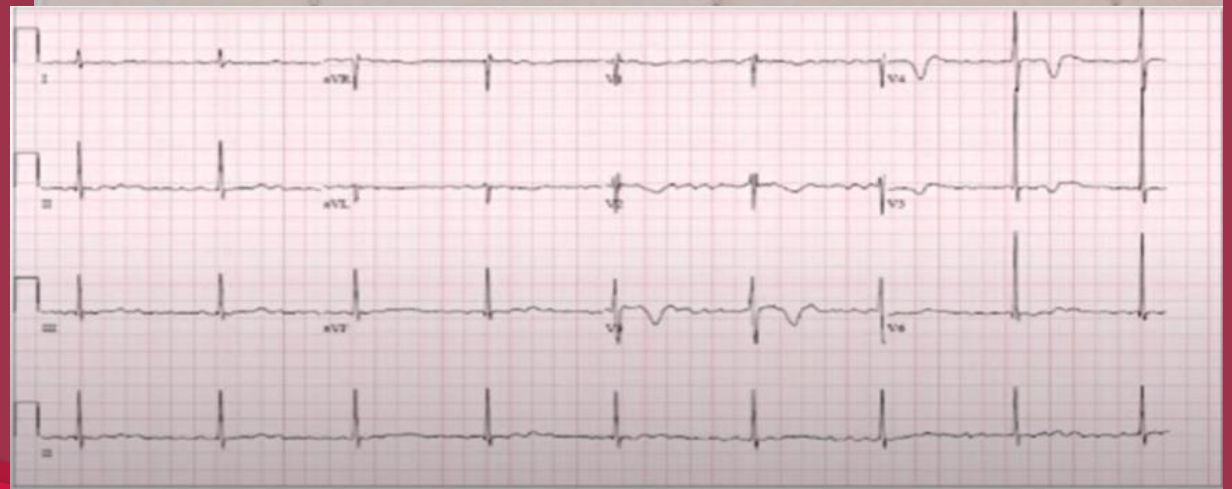


3rd Degree AV Block - Complete Heart Block

- In the third ECG, there are no P waves, and the QRSs are regular → this is a patient who has a baseline A Fib (changed from irregular to regular), then he developed Complete Heart block on top of his A Fib forming Junctional Escape (This is one of the important signs of Digoxin Toxicity).
- One of the things that we do to treat A Fib is giving the patients Digoxin → They may develop Renal Failure and become Dig toxic, so the A fib changes from irregular to regular.

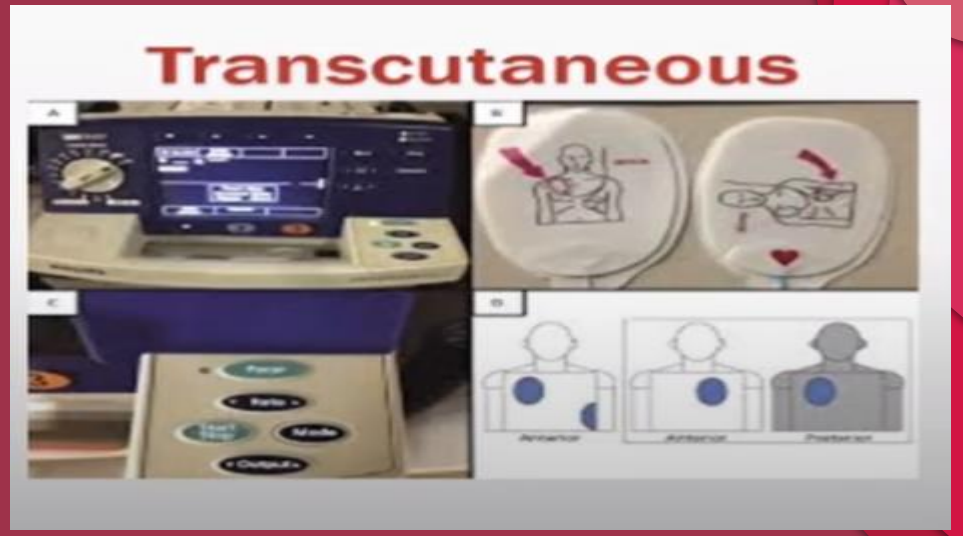


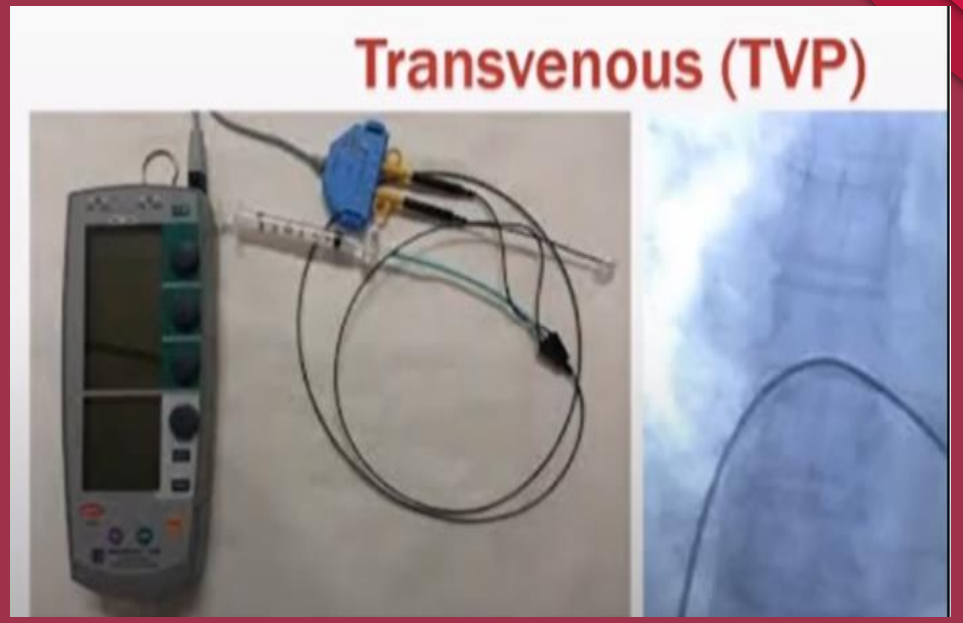




Pacemakers and Cardiac Devices

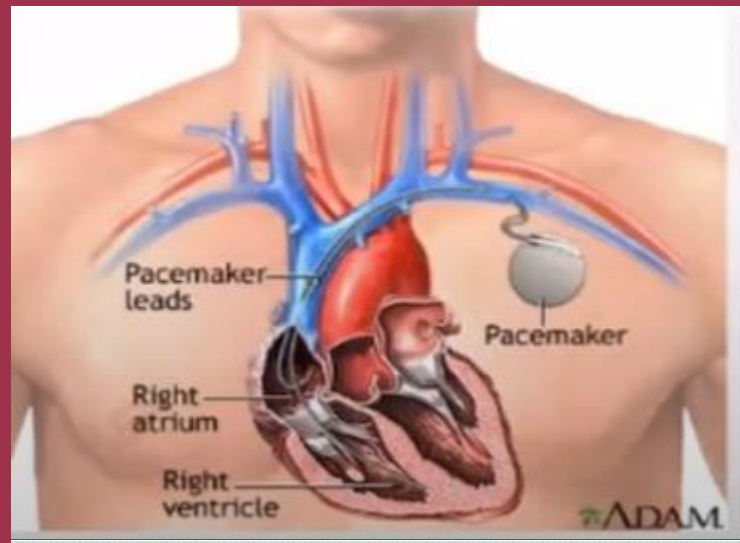
- Pacemakers are divided into 2 major groups: Temporary Vs Permanent.
  - Temporary pacemakers last Hours to Days.
- Transcutaneous Pacemaker: we use pads similar to those used for defibrillation and we change the device mood from Defibrillation to Pacing mode; the only disadvantage is that it's very painful and not reliable
- Transvenous Pacemaker: a wire inserted into the femoral or Internal jugular veins until reaching the right ventricle. Used in emergency cases especially when there is a reversible cause and we want to stabilize the patient until he gets his ultimate solution or until the reversible cause go away.

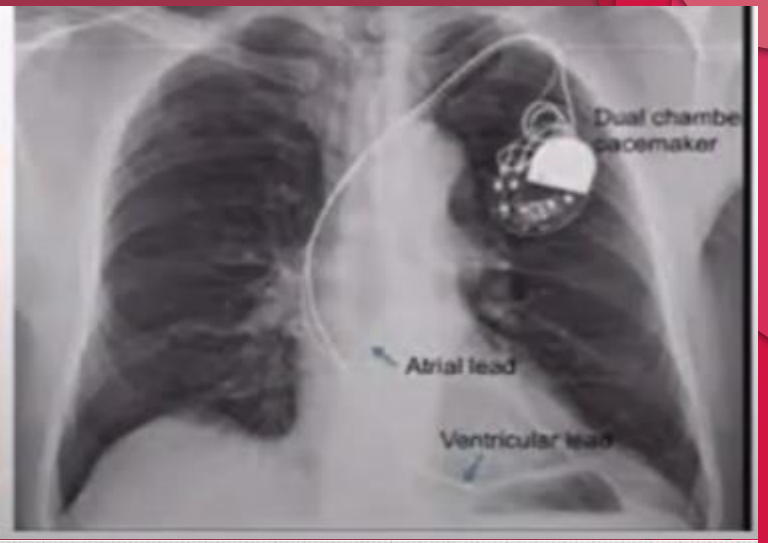


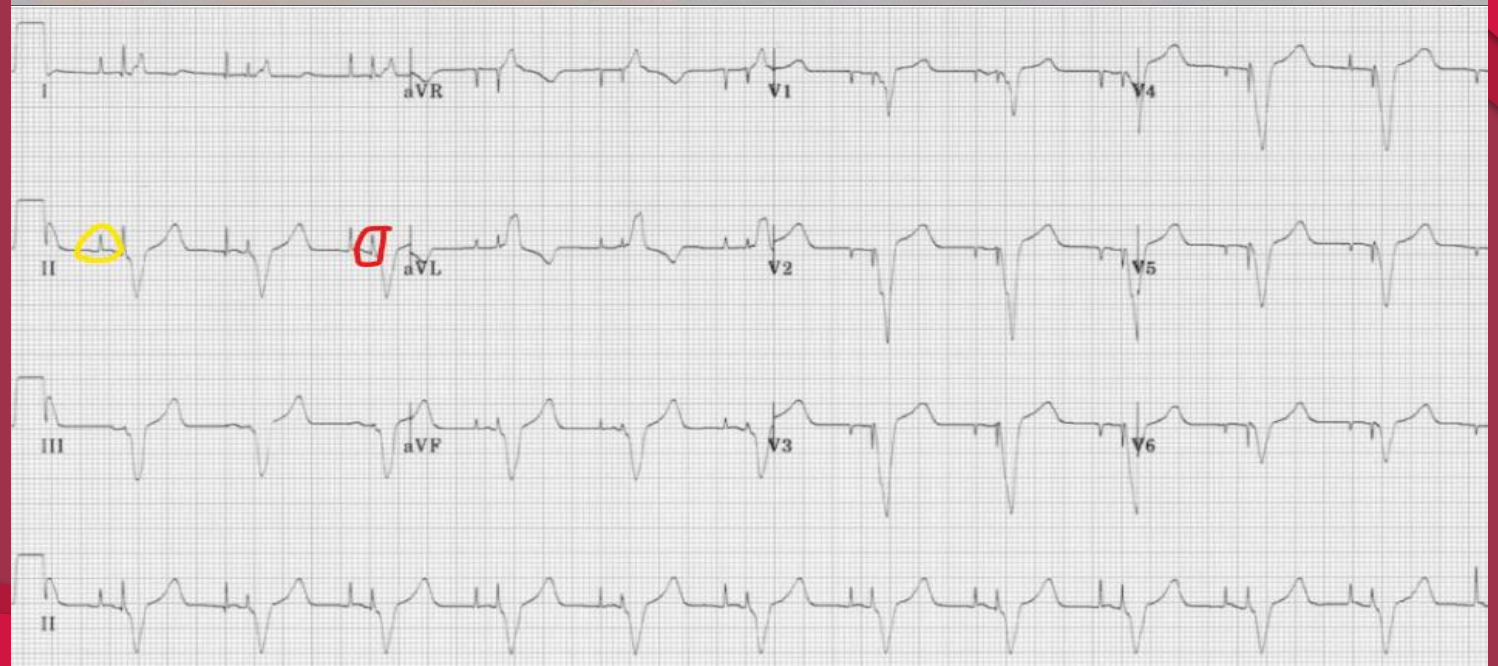


Pacemakers and Cardiac Devices

- In most of the cases you need a permanent pacemaker placement: a small device put in a pouch subcutaneously, then we insert 2 wires in the vein to reach the heart (one in the right atrium and the other in the right ventricle).
- There are 2 types: Single Chamber (goes to either Rt Atrium or Ventricle) or Double Chamber (goes to both of them).
- Permanent Pacemaker ECG: Notice the spike of the Pacer (Yellow → Atrial Pacer / Red → Ventricular Pacer)
- The QRS of the pacemaker is wide → the pacemaker rhythm is a wide rhythm; because it's not conducting through the normal conduction system.

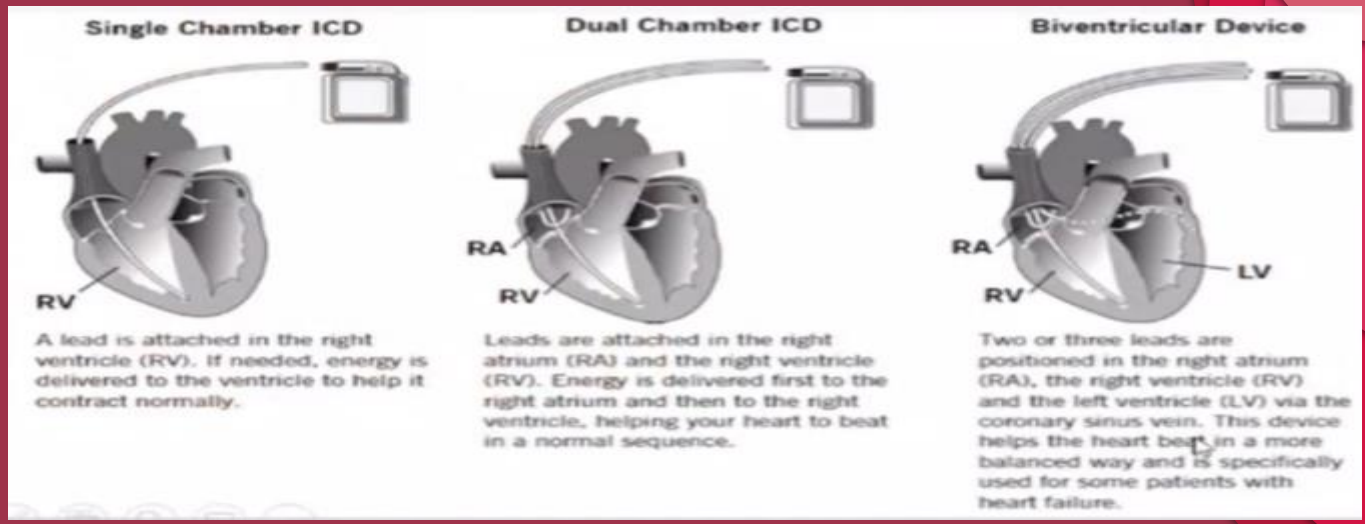


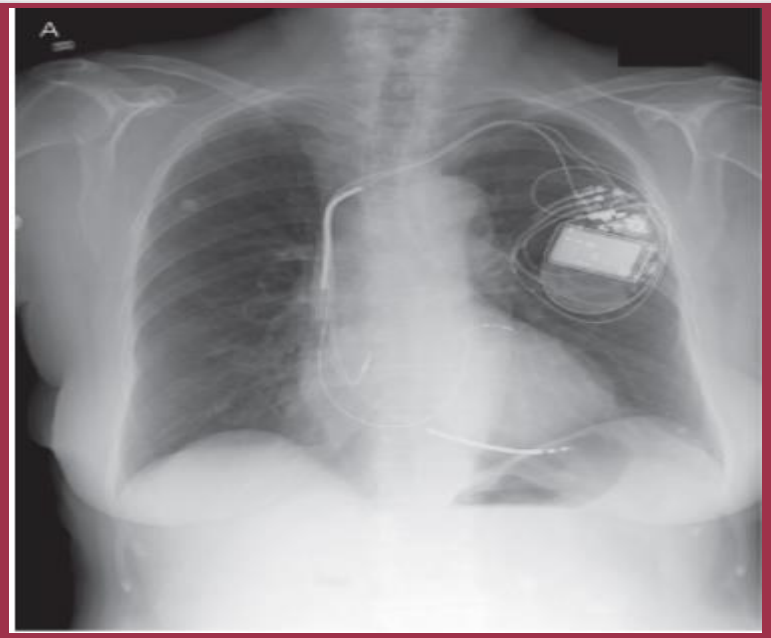




Pacemakers and Cardiac Devices

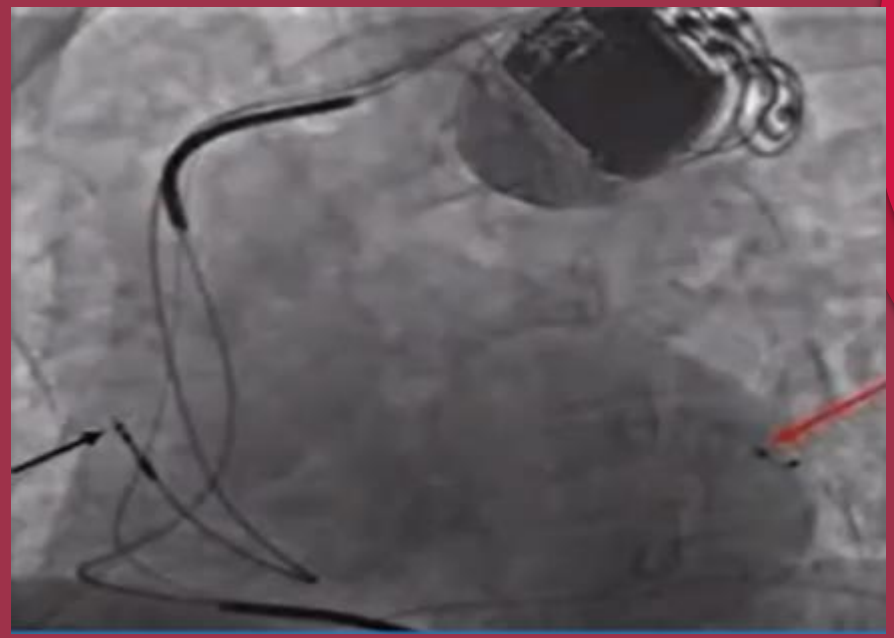
- Implantable Cardiac Defibrillator (ICD).
- It looks like a pacemaker, but its wires are thicker.
- All ICDs have pacemaker activity (they can pace), additionally they watch the rhythm and if the patient goes into ventricular arrhythmia that is life threatening, they shock the heart directly.
- Dual chamber ICD can monitor the type of arrhythmia and gives Anti-Tachycardia Pacing (instead of giving the painful shock, it over-paces the heart trying to reset the heart without shocking it





Pacemakers and Cardiac Devices

- Cardiac Resynchronization Therapy (CRT).
  - Used for HF.
- Three leads are used: One in the right ventricle, the second one in the right atrium, and the third lead pass through the coronary sinus down the cardiac vein to reach the lateral left ventricular wall (coordinates the contractions of 1- the left and right ventricles, 2- the lateral and septal walls of the left ventricle , 3- the Atria and ventricles - Those coordinations are lost in HF-).



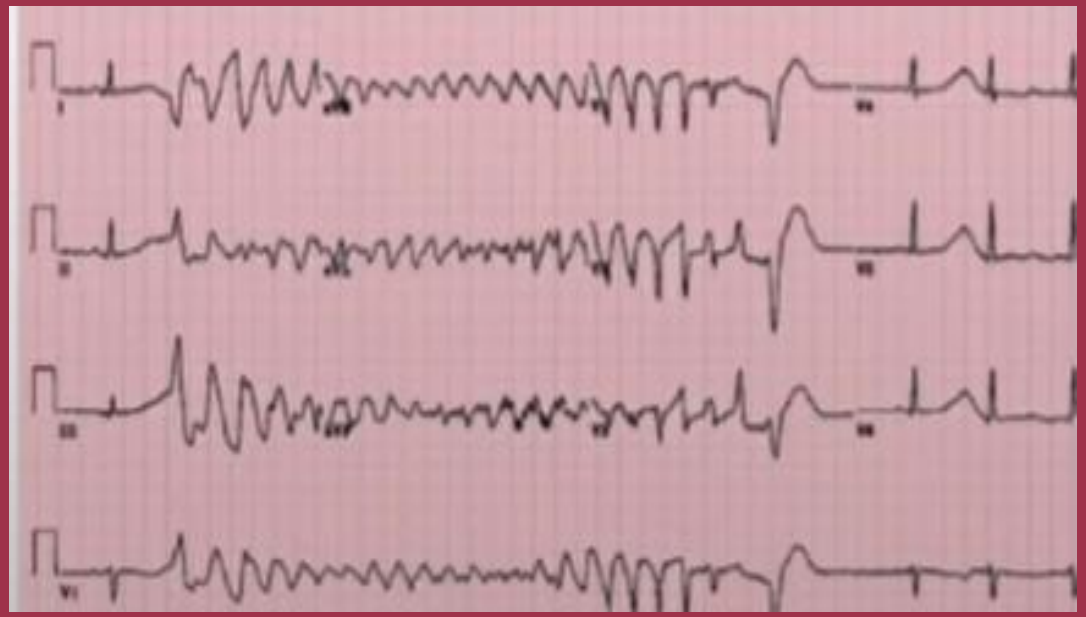
Pacemakers and Cardiac Devices

- Magnet Mode.

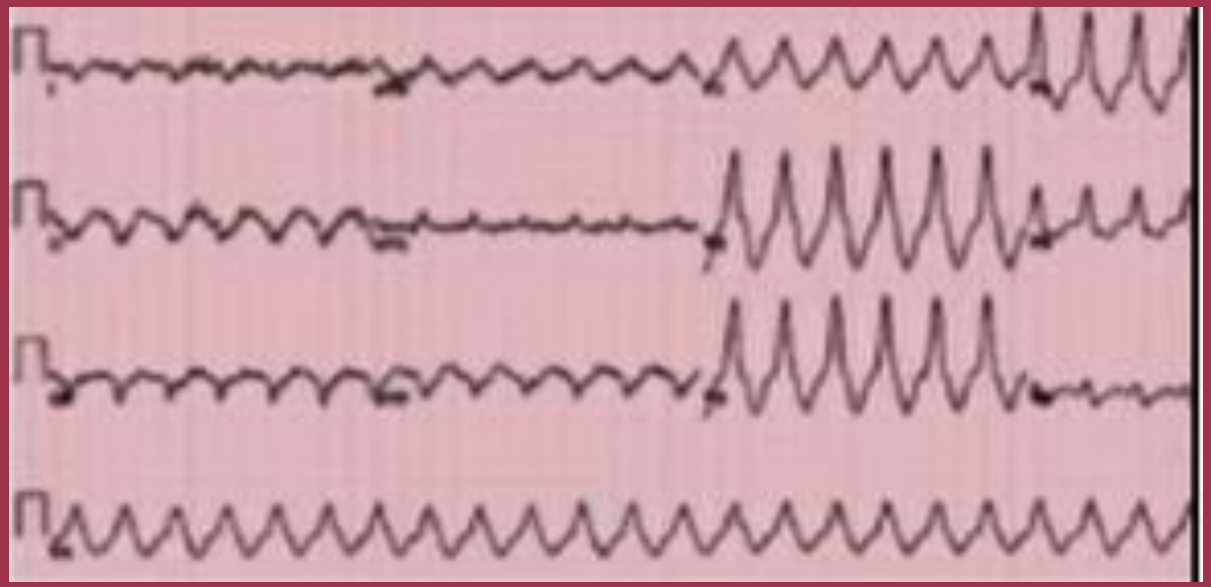
- Let's assume that we have a patient with a pacemaker, and he needs surgery (in surgeries we use electrocautery which confuses the pacemaker and let it cause arrhythmias) so what we do is that we slam this magnet to make the pacemaker continuously pacing without looking at any electricity around it, when you're done you move the magnet, and the pacemaker returns to its normal programming.
- A second use of the magnet is in the case of an ICD (which detects ventricular arrhythmias and shock the patient), but if the patient is in cardiac arrest and we need to resuscitate him (without getting shocked by the ICD), we slam the magnet and turn the ICD off.

Figure 1: 174105-2 Magnet

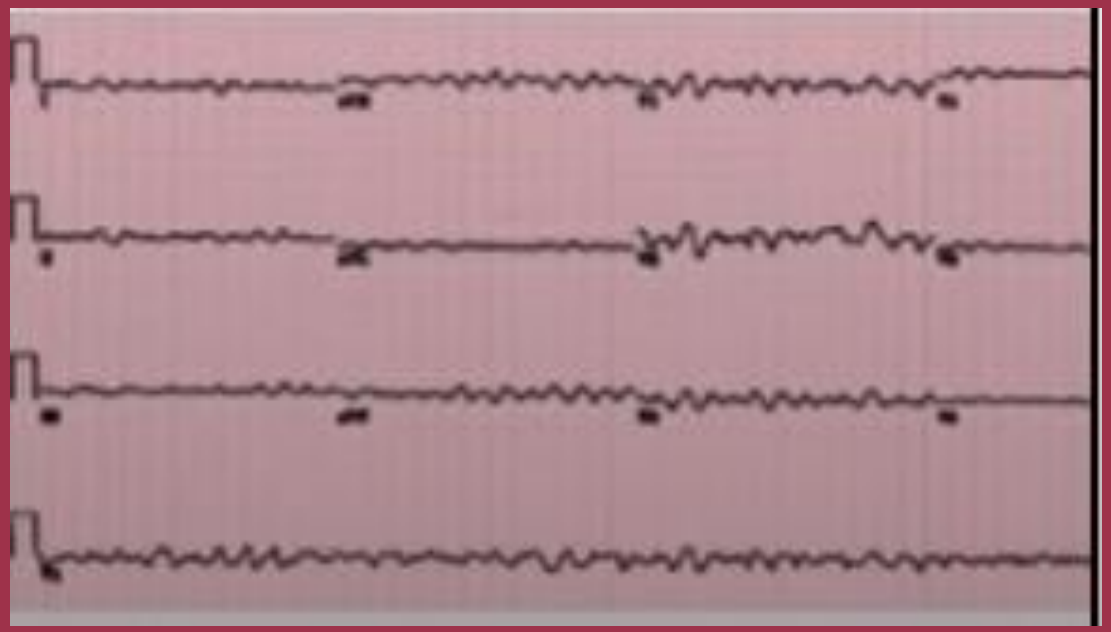
Torsades De Point



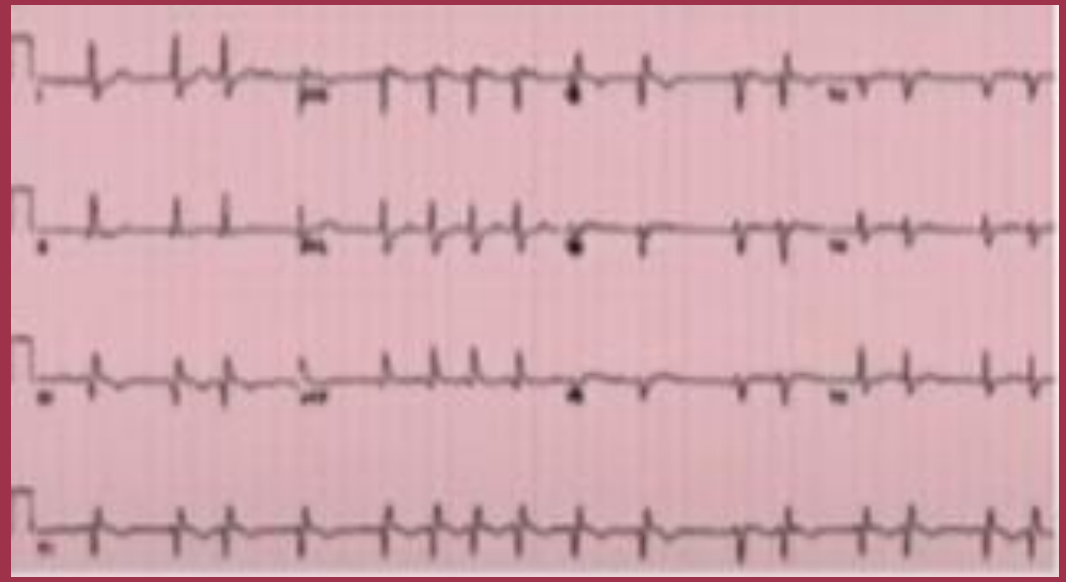
Ventricular Tachycardia



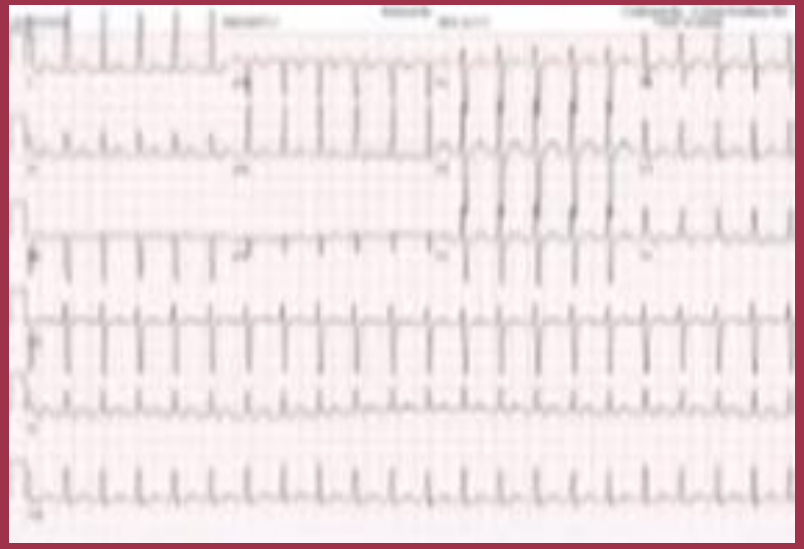
Ventricular Fibrillation



Atrial Fibrillation

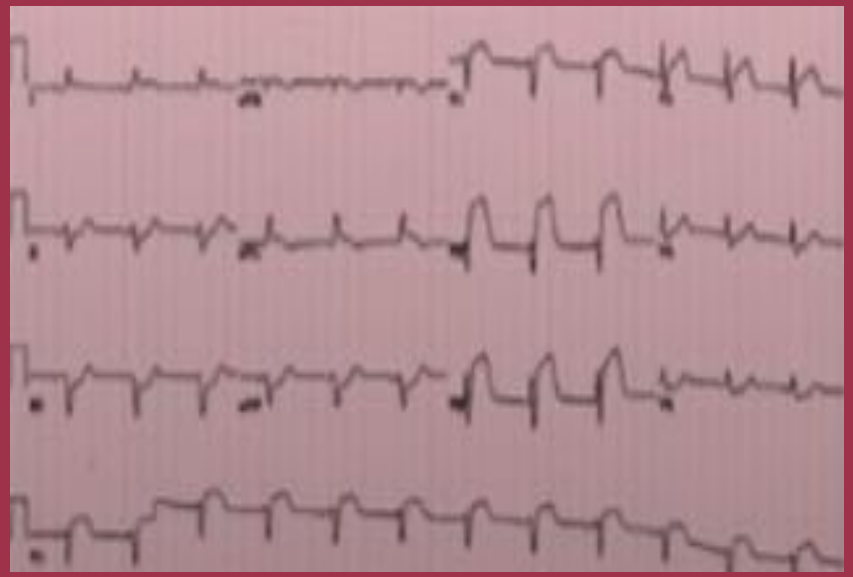


SVT



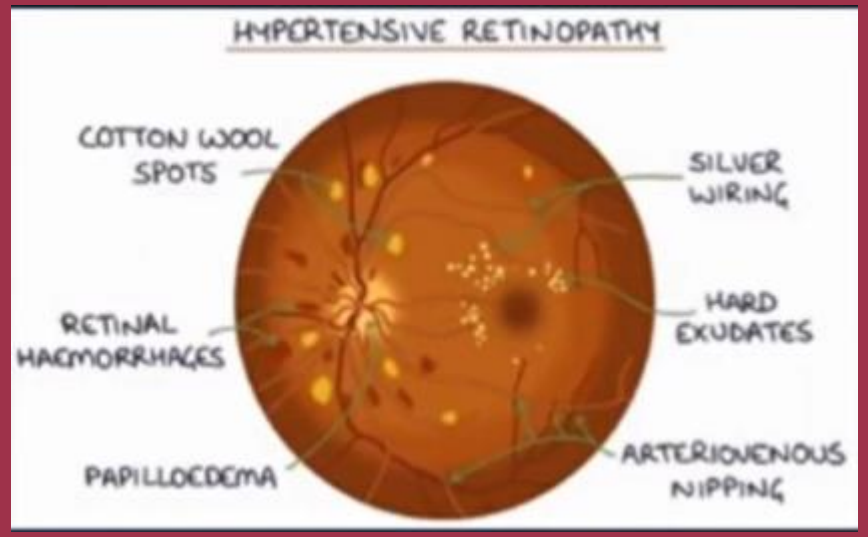
Sorry for the Bad Quality 😢

ST Elevation MI

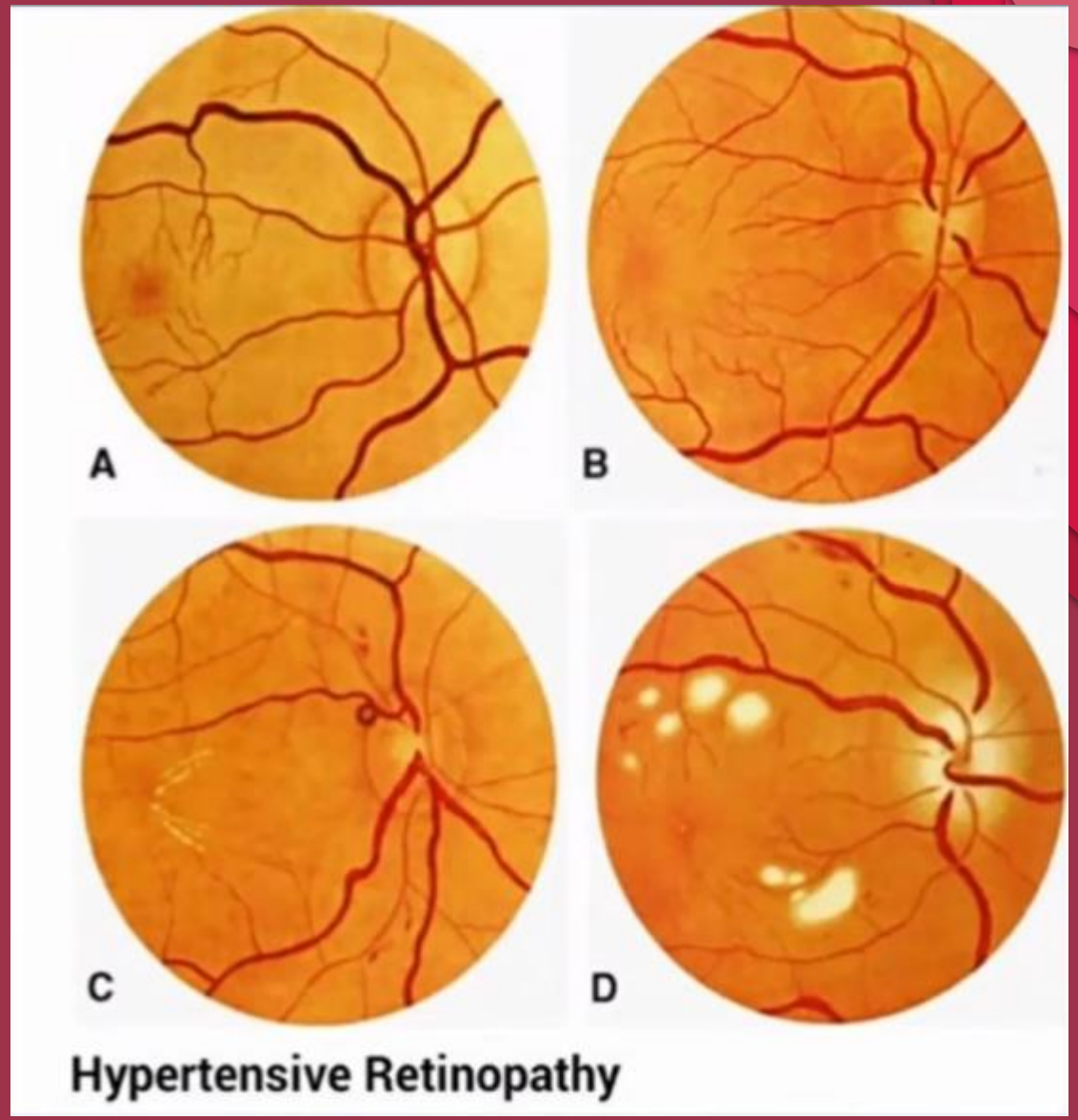


Hypertension

Diagnosis of HTN



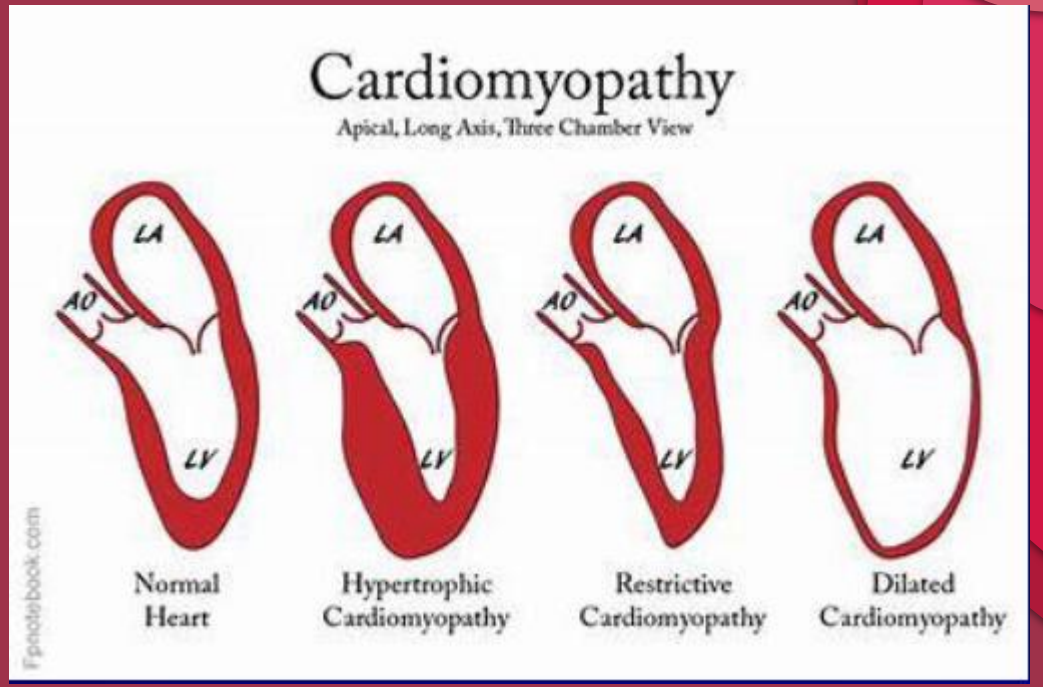
Grade	Classification
Grade I	Mild generalized retinal arteriolar narrowing or sclerosis
Grade II	Definite focal narrowing and arteriovenous crossings Moderate to marked sclerosis of the retinal arterioles Exaggerated arterial light reflex
Grade III	Retinal hemorrhages, exudates and cotton wool spots Sclerosis and spastic lesions of retinal arterioles
Grade IV	Severe grade III and papilledema



Cardiomyopathies

Cardiomyopathies

- Heterogenous group of diseases of the myocardium associated with mechanical and/or electric dysfunction that usually exhibit inappropriate ventricular hypertrophy or dilation and are due to a variety of causes that frequently are genetic.
- Cardiomyopathies are either confined to the heart or are part of generalized systemic disorders.
- MOGES Classification: M → Phenotype (Dilated and Hypertrophic) / O → Organ involvement (with or without extracardiac involvement) / G → Genetic transmission (Autosomal dominant or recessive) / E → Pathogenesis (Genetic with disease gene and mutation, if known) / S → Disease stage.
- WHO Classification (depends on phenotypic images by echo and autopsy): 1- Dilated (Thin ventricular border with dilated chamber - Enlarged, systolic dysfunction). 2- Hypertrophic (Septum is larger than posterior wall - Thickened, diastolic dysfunction). 3- Restrictive (Normal ventricle and chamber size but huge atria - Diastolic dysfunction). 4- Arrhythmogenic RV dysplasia (Fibrofatty replacement). 5- Unclassified (Fibroelastosis, LV noncompaction)



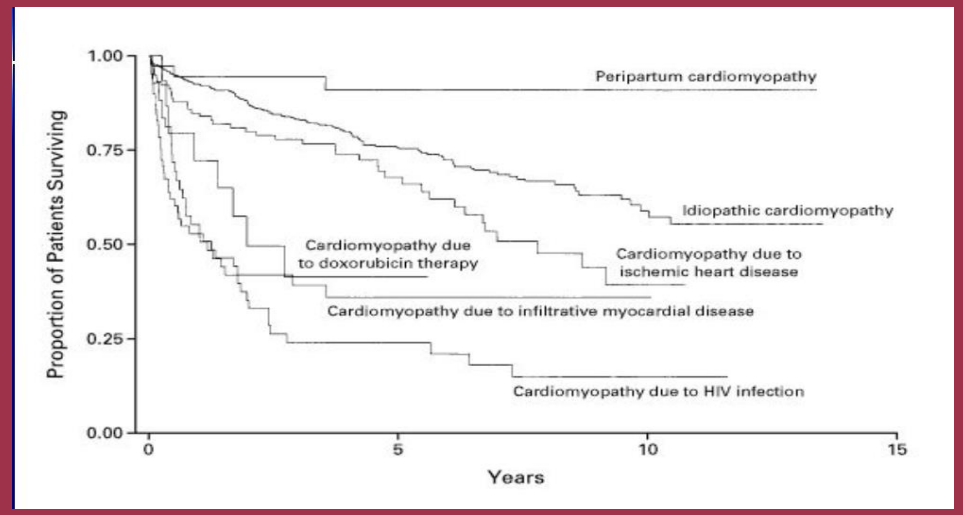
Dilated Cardiomyopathy

- Dilation of the left or both ventricles that is not explained by abnormal loading conditions (MR / AR) or coronary artery disease.
- Characterized by Cardiac enlargement with ventricular walls of approximately normal thickness and varying extents of fibrosis.
- The patient ultimately develops progressive HF with reduced ejection fraction, tachyarrhythmia, increased risk of sudden death.
- Mitral and Tricuspid Regurgitation because of annular dilation are frequent and intensify the hemodynamic burden.
- Causes: Idiopathic, Inherited (Autosomal Dominant, Recessive, X-linked, Mitochondrial - Abnormality in the gene that encodes titin which is the protein that controls the stiffness of the sarcomere), Infectious (Acute viral myocarditis, Cox-B or Echovirus, Self-limited infection in young people), Non-infectious (Inflammation and immune reactions, Allergic reactions to drugs, Kawasaki disease, SLE, Loffler endocarditis, Cardiac MRI (CMR) is a powerful tool for recognition and assessment, Gold standard is biopsy), Toxic (Alcoholic Cardiomyopathy, reversible after abstinence), Peripartum (Echo shows LV dysfunction, Risk factors include African-Americans, Multiple pregnancies, Alcohol, Tobacco).

- This figure shows Large Chamber with Thin Ventricular wall.
- Normal chamber size is up to 5.6 cm

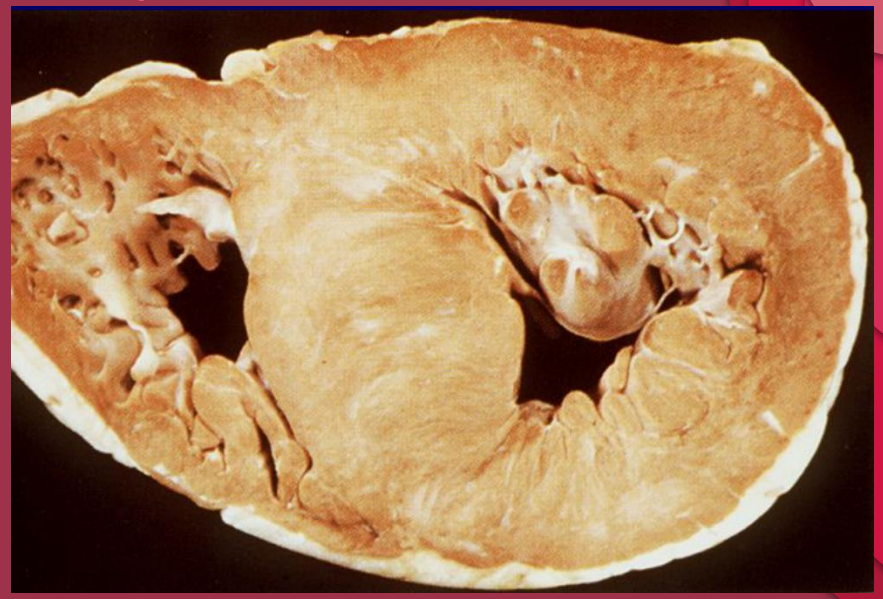
Dilated Cardiomyopathy

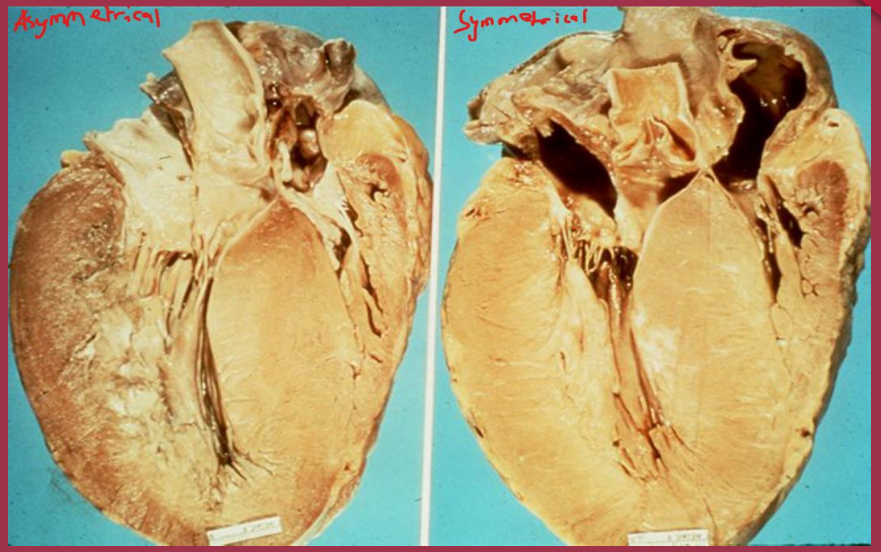
- Prognosis of DCM depends on the Etiology.
  - Cardiomyopathy due to Doxorubicin therapy and HIV infection has the worst prognosis.



Hypertrophic Cardiomyopathy

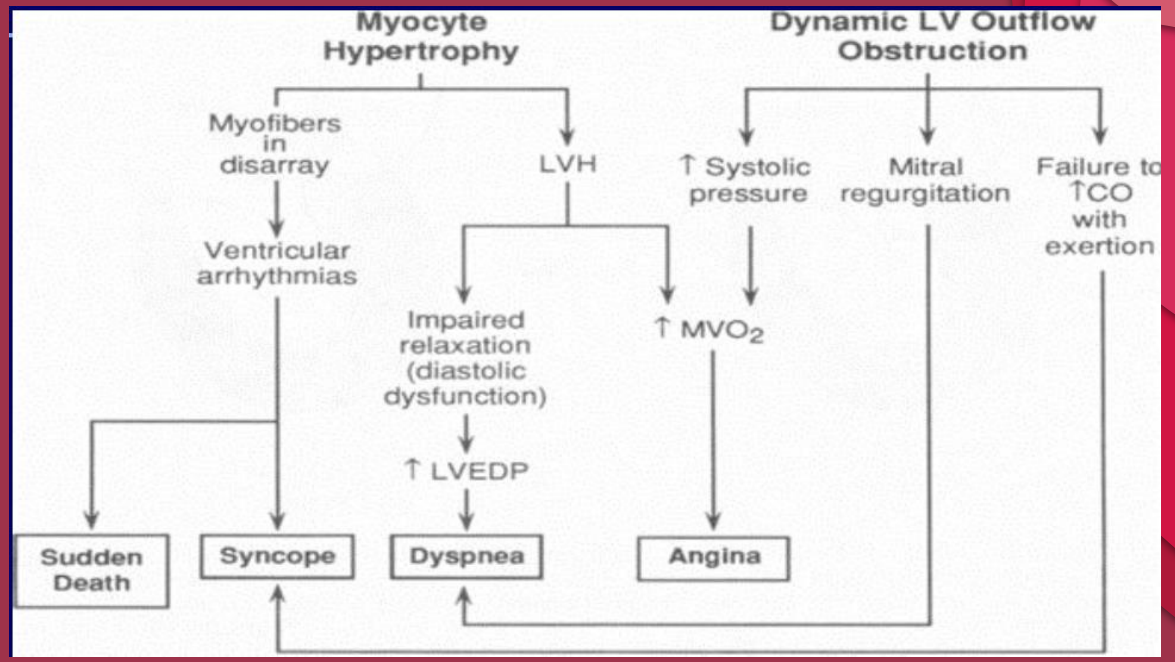
- Left ventricular hypertrophy not due to pressure overload.
- Hypertrophy is variable in both severity and location: 1- Asymmetric septal hypertrophy. 2- Symmetric (non-obstructive). 3- Apical hypertrophy (Japanese variant).
- Vigorous systolic function, but impaired diastolic function.
- Most common cardiomyopathy.
- More common in males.
- Etiology: 1- Familial in most cases (Autosomal dominant). 2- Mutation in one of these genes that encode proteins of cardiac sarcomere (Beta-MHC, Cardiac Troponin T, Myosin binding protein C, Alpha-tropomyosin).
- The figure shows Thick septum compared to the posterior wall.
- Dynamic Left Ventricular Outflow obstruction (may not be present at rest).
- Systolic anterior motion of mitral valve.
- LVOT obstruction → LVOT gradient → increase wall stress → increased MVO2 → Ischemia
- Dyspnea and Angina more related to diastolic dysfunction than to outflow tract obstruction.
- Syncope → LVOT obstruction (failure to increase Cardiac output during exercise or after vasodilatory stress) or Arrhythmia.





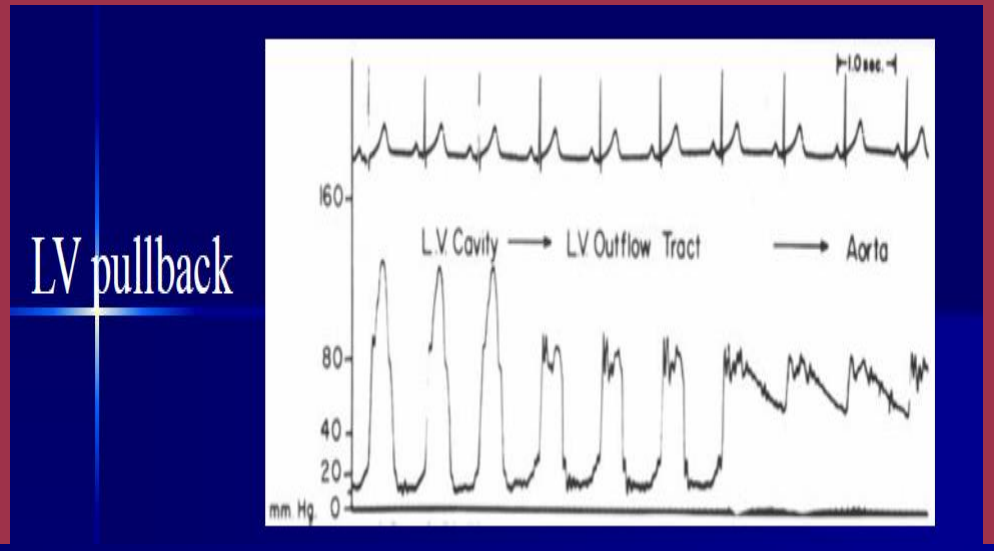
Hypertrophic Cardiomyopathy

- The Adjacent figure illustrates the pathophysiology of HCM.
- On PE: 1- Bisferiens Pulse (double pulse, 2 peaks, spike and dome). 2- S4 gallop. 3- Crescendo/Decrescendo systolic ejection murmur.
- Murmur is increased with decreased preload, because by that the mitral valve and LVOT close so it causes obstruction of the flow.
- Diagnostic Studies: 1- EKG (normal sinus rhythm, Left ventricular hypertrophy, Septal Q waves). 2- 2D-echocardiography (LVH; septum >1.4x free wall, LVOT gradient by Doppler, Systolic Anterior motion of the mitral valve). 3- Cardiac catheterization (LVOT gradient and pullback, Provocative maneuvers, Brockenbrough phenomena).



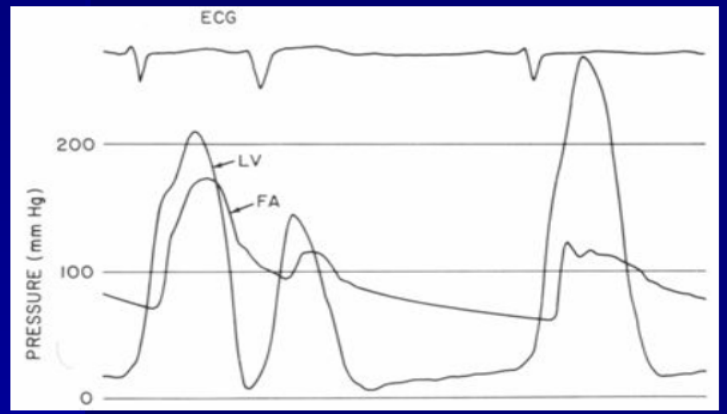
HCM: Cardiac Catheterization

- Brockenbrough sign: FA → Femoral artery to trace the aorta. Pause following PVC followed by forceful contraction leading to extreme gradient within the ventricle and very small gradient within the aorta increasing pulse pressure and causing this sign.
- Amyl nitrate inhalation → lead to further obstruction increasing gradient in LV and small gradient in the aorta.



Brockenbrough-Braunwald Sign

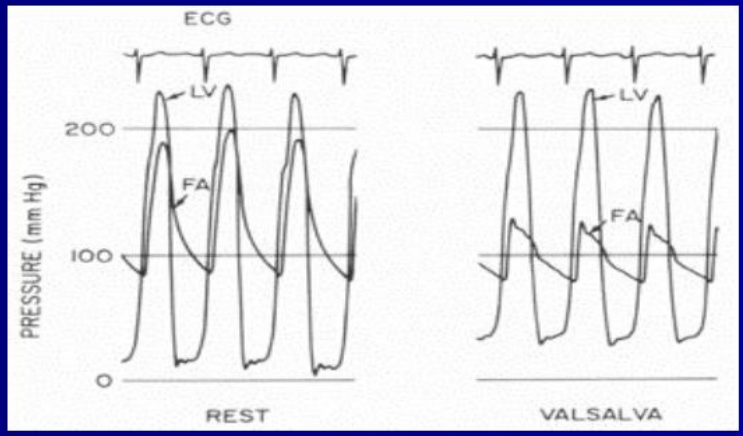
failure of aortic pulse pressure to rise post PVC



Provocative maneuvers:

Valsalva

amyl nitrate inhalation

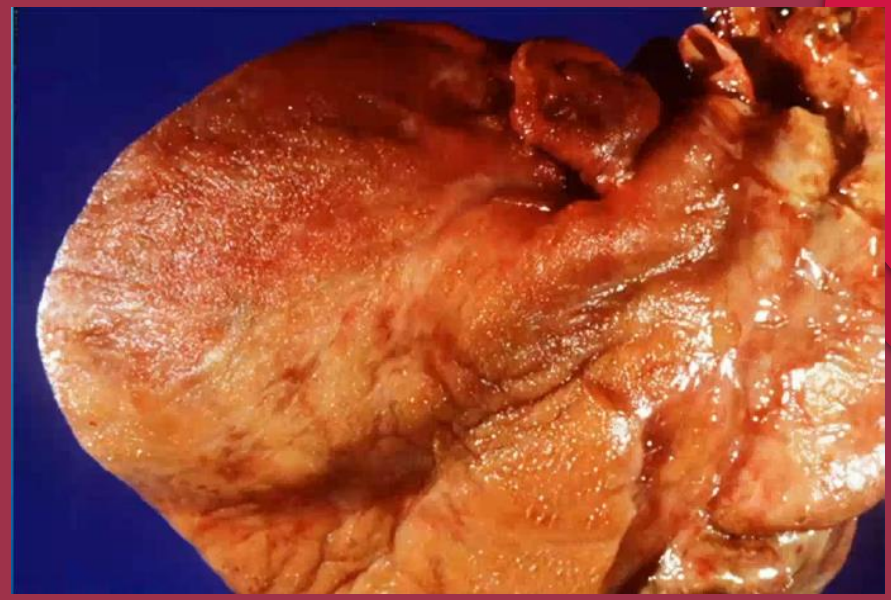


Restrictive Cardiomyopathy

- Characterized by impaired ventricular filling due to an abnormally stiff ventricle + normal systolic function + intraventricular pressure rises precipitously with small increases in volume.
  - Plastic heart
  - Resistant to accept blood.
- Small drop of blood cause increase in intraventricular pressure due to low compliance.
- Causes: infiltration of the myocardium by abnormal substance (Amyloidosis / Sarcoidosis), Fibrosis or scarring of the endocardium.
- The adjacent figure shows Amyloid infiltrative CM (seen with Apple green stain).

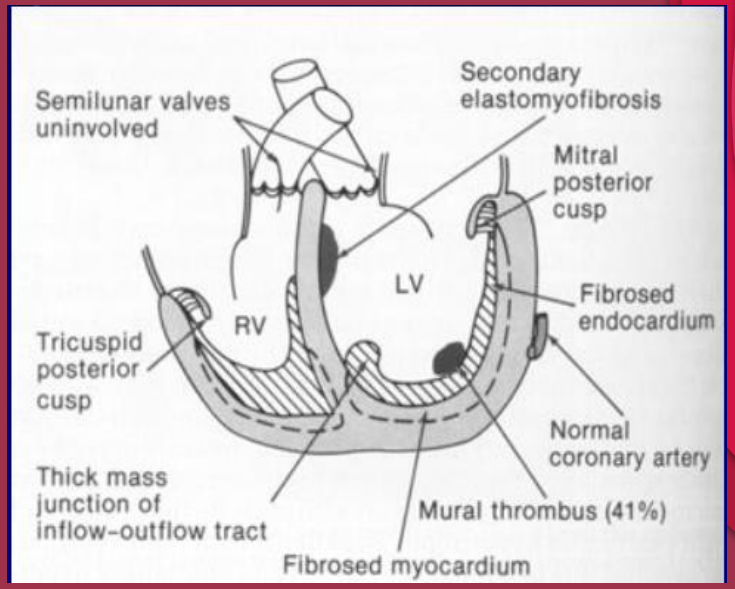
Amyloid Cardiomyopathy

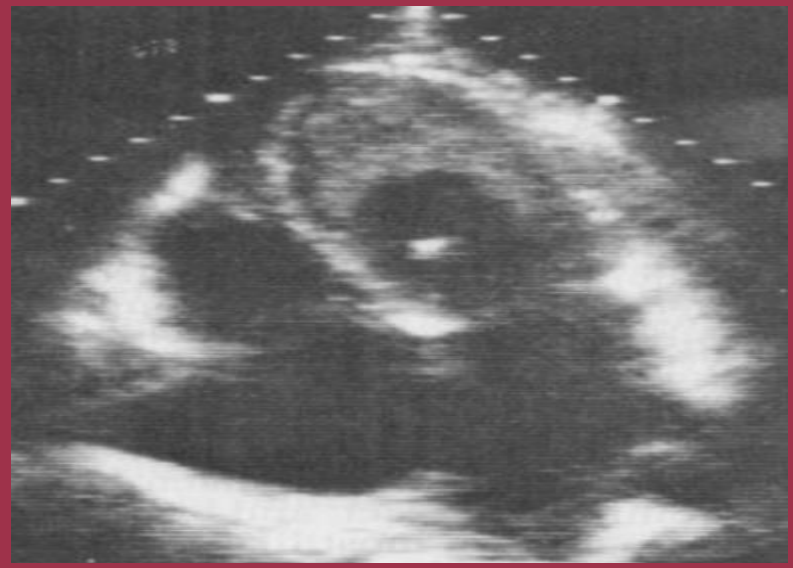
- Amyloidosis is caused by protein misfolding in which extracellular aggregates of the misfolded proteins form fibrils.
  - Primary Amyloidosis → Immunoglobulin light chains - Multiple myeloma.
  - Secondary Amyloidosis → Deposition of protein other than immunoglobulin (Senile, Familial, Chronic inflammatory process).
- Restriction caused by replacement of normal myocardial contractile element by infiltrative interstitial deposits.
  - CMR is a sensitive diagnostic technique.
- Echo → good as a primary test showing scintillating appearance of the heart; if you suspect Amyloidosis then do CMR/PET.
- A definitive diagnosis still requires histological verification (you don't have to take the biopsy from the heart since it's a systemic disease, so take it from other safe areas.
  - Notice the yellowish discoloration of amyloid tissue.
- Treatment: Autologous BM stem cell transplant + Drugs (Dexamethasone, Melphalan, Immunomodulatory agent, Proteasome inhibitor "Bortezomib") + Orthotopic Liver transplant (in wild-type transthyretin amyloidosis).



Restrictive Cardiomyopathy

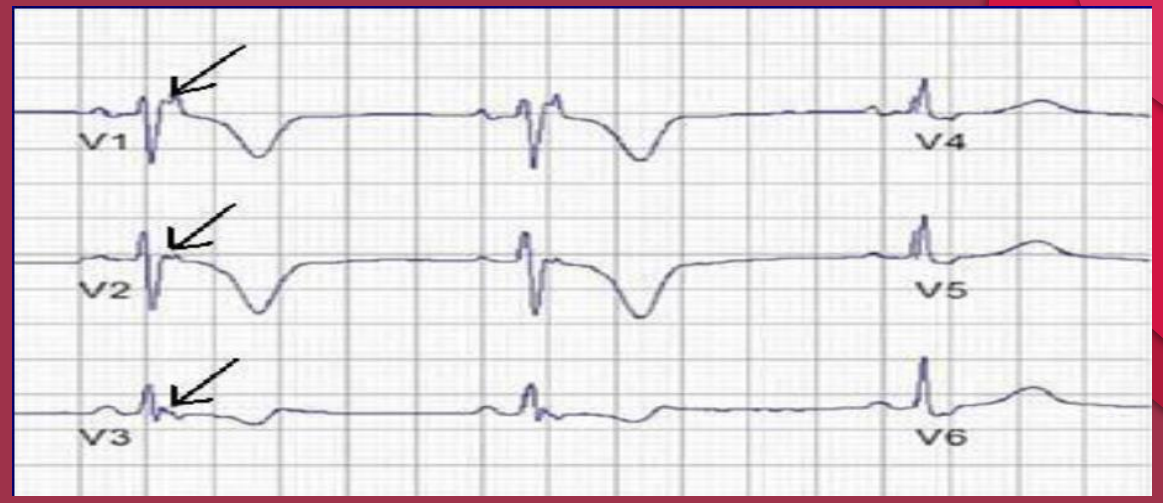
- Endomyocardial fibrosis → Thickening of basal inferior wall, Endocardial deposition of thrombus, Apical obliteration, Mitral Regurgitation.
- Sarcoidosis:
- 1- Now recognized as an inflammatory condition in which non-caseating granulomas involve multiple organs.
- 2- Restriction
- 3- Conduction system disease (AV blocks - complete heart block)
- 4- Ventricular Arrhythmias
- 5- Treatment: Glucocorticoids + other immunosuppressive agents if necessary.
- Treatment of Restrictive Cardiomyopathy: Treat the underlying cause (Amyloid (with melphalan/ prednisone/ colchicine), Endomyocardial Fibrosis (with steroids/ cytotoxic drugs/ MVR), Hemochromatosis (with chelation/ phlebotomy), Sarcoidosis (with steroids)) + Diuretics and other treatment options for HF + Pacemaker for conduction abnormalities + Anticoagulation for thrombus + Transplant (the best option)





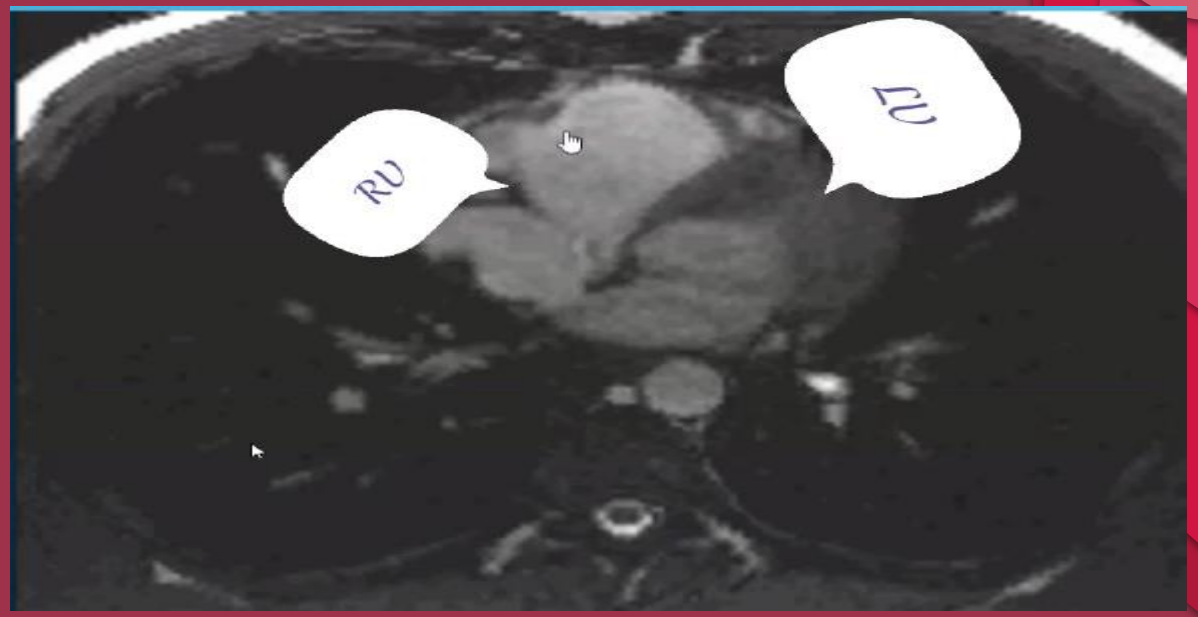
Arrhythmogenic RV Dysplasia

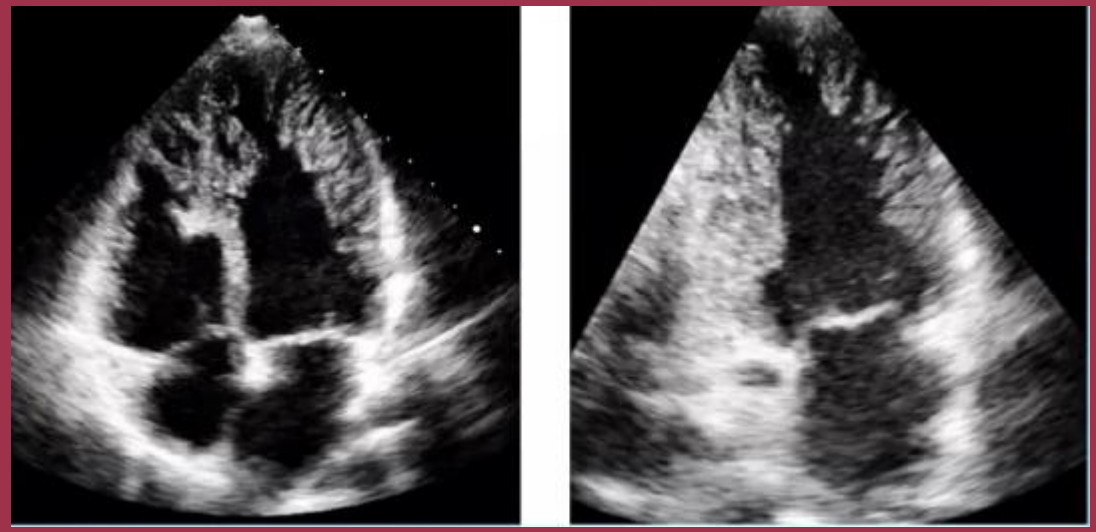
- Myocardium of RV free wall is replaced by Fibrofatty tissue + Regional wall motion / function is reduced.
  - Ventricular arrhythmias → SCD in young.
- Abnormalities in intercellular adhesion molecules "desmosomes", cause cell death and fibrofatty replacement.
  - Caused by mutations in genes like PKP2 and DSP.
    - Inheritance → AD.
- The adjacent ECG shows Epsilon wave seen in ARVD; caused by delayed repolarization following the QRS complex.
  - Contrast enhanced CMR is the best diagnostic tool.
- Treatment: Cessation of heavy physical exertion and competitive athletics.
  - Patients with intractable HF may require cardiac transplant.



LV Non-Compaction

- Diagnostic Criteria → Prominent Trabeculations + Deep recesses in LV apex.
  - Increased risk of CHF, VT/SCD, Thrombosis.
  - Hereditary risk → Screening of offspring.
- Notice in the first figure (MRI) that the RV is approximately the same size as LV with aneurysmal changes.





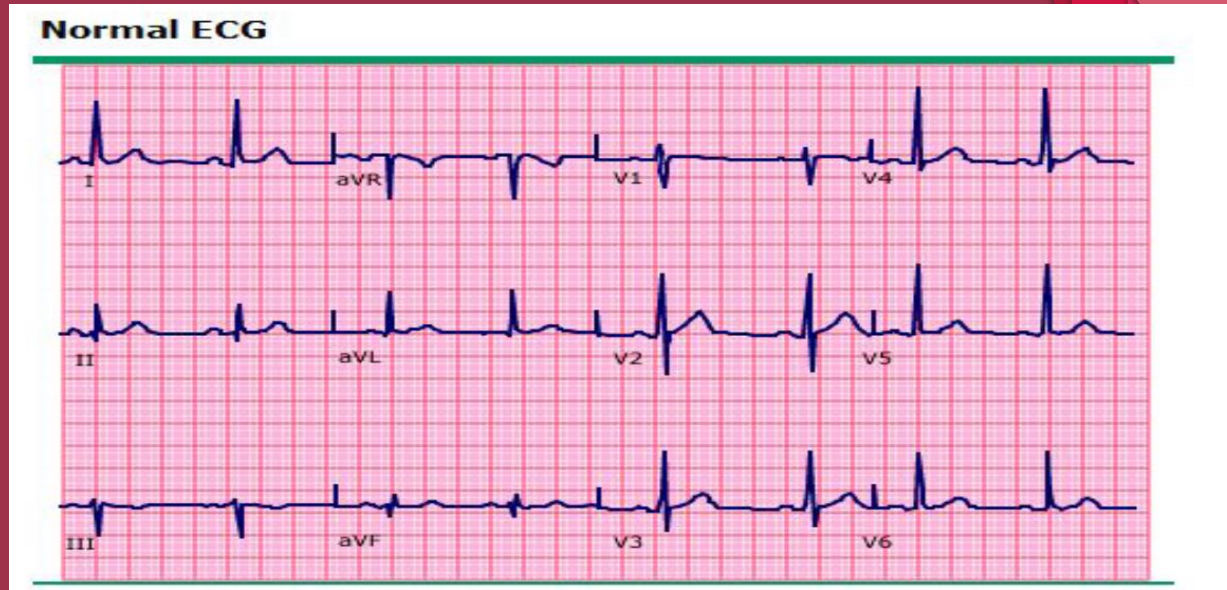
Pericarditis

Pericarditis

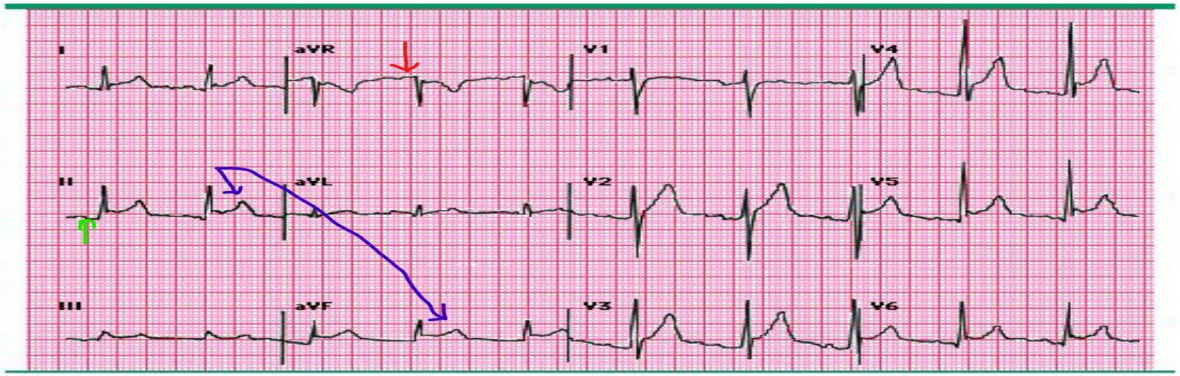
- Perimyocarditis /Myopericarditis: Acute pericarditis that also demonstrate myocardial involvement.
- Most cases of acute pericarditis are considered of possible or confirmed viral origin (Coxsackievirus, Echovirus, Adenovirus).
- Clinical presentation: Patients with an infectious etiology may present with signs and symptoms of systemic infection such as fever and leukocytosis. Viral etiologies may be preceded by "Flu-like" respiratory or gastrointestinal symptoms.
- The major clinical manifestations include: 1- Chest pain (Sharp and pleuritic, improved by sitting up and leaning forward, exacerbated by inspiration or coughing). 2- Pericardial friction rub (a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border). 3- ECG changes (new widespread ST elevation or PR depression). 4- Pericardial effusion.

Pericarditis: ECG Changes

- Stage 1: Seen in the first hours to days. Characterized by diffuse ST elevation with reciprocal ST depression in leads aVR and V1. There is also an atrial current of injury reflected by elevation of the PR segment in lead aVR and depression of the PR segment in other leads.
- Stage 2: Typically seen in the first week, characterized by normalization of the ST and PR segments.
- Stage 3: Characterized by the development of diffuse T wave inversions, generally after ST segment have become isoelectric.
- Stage 4: Represented by normalization of the ECG or indefinite persistence of T wave inversions ("chronic pericarditis").
  - Red arrow → PR segment Elevation
  - Green Arrow → PR segment Depression
  - Blue Arrow → Concave appearance.



Normal sinus rhythm at a rate of 71 beats/min, a P wave axis of 45°, and a PR interval of 0.15 sec.



Electrocardiogram in acute pericarditis showing diffuse upsloping ST segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR segment deviation (positive in aVR, negative in most other leads). ST segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR segment changes are due to an atrial current of injury which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

Pericarditis: Diagnosis

- Acute pericarditis may be associated with increases in serum biomarkers of myocardial injury such as Cardiac troponin, MB fraction of creatine kinase.
- Lab findings: Elevated WBCs + ESR + Serum CRP.

Diagnostic criteria for acute pericarditis and myopericarditis in the clinical setting

Acute pericarditis (at least 2 criteria of 4 should be present)*:
1. Typical chest pain
2. Pericardial friction rub
3. Suggestive ECG changes (typically widespread ST segment elevation)
4. New or worsening pericardial effusion
Myopericarditis:
1. Definite diagnosis of acute pericarditis, **PLUS**
2. Suggestive symptoms (dyspnea, palpitations, or chest pain) and ECG abnormalities beyond normal variants, not documented previously (ST/T abnormalities, supraventricular or ventricular tachycardia or frequent ectopy, atrioventricular block), **OR** focal or diffuse depressed LV function of uncertain age by an imaging study
3. Absence of evidence of any other cause
4. One of the following features: evidence of elevated cardiac enzymes (creatine kinase-MB fraction, or troponin I or T), **OR** new onset of focal or diffuse depressed LV function by an imaging study, **OR** abnormal imaging consistent with myocarditis (MRI with gadolinium, gallium-67 scanning, anti-myosin antibody scanning)
Case definitions for myopericarditis include:
Suspected myopericarditis: criteria 1 plus 2 and 3
Probable myopericarditis: criteria 1, 2, 3, and 4
Confirmed myopericarditis¶: histopathologic evidence of myocarditis by endomyocardial biopsy or on autopsy

* Pericardial effusion confirms the clinical diagnosis but its absence does not exclude it.
¶ In clinical practice a confirmed diagnosis would require an endomyocardial biopsy that is not warranted in self-limited cases with predominant pericarditis.

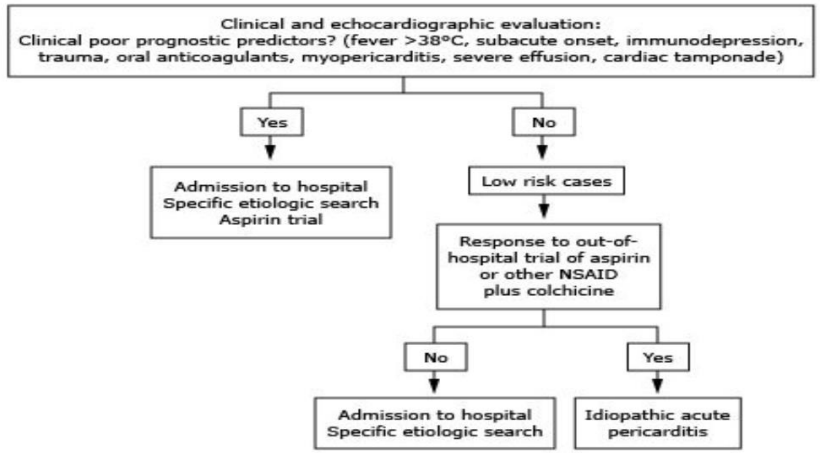
Pericarditis: Treatment

- When dealing with acute viral pericarditis (or if the cause is unknown), then give the patient NSAIDs and Colchicine.
- If other etiologies (Bacterial infection / autoimmune) → treat according the cause.
- Pericarditis following CABG → Aspirin.
- Acute pancreatitis following an MI (Dressler syndrome) → Aspirin + Colchicine.
- Generally, avoid using steroids except if the patient isn't responding to other drugs.
- Steroids given for → Pericarditis due to connective tissue disease + Autoreactive (immune-mediated) pericarditis + Uremic Pericarditis not responding to dialysis + Patients who have contraindications to NSAID therapy.
- Glucocorticoid therapy is also used for patients with idiopathic or viral pericarditis that is refractory to combination therapy with NSAIDs and Colchicine.

Drug therapy in acute pericarditis

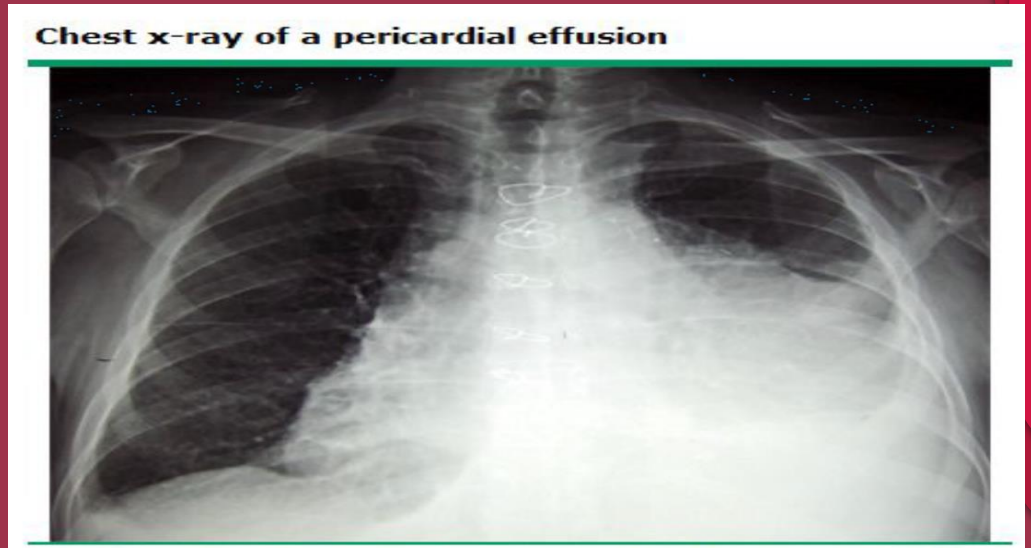
Drug	Dose	Duration of therapy	Tapering
For initial combination treatment of most patients:			
Ibuprofen	400 to 800 mg three times daily	1 to 2 weeks	Decrease the dose weekly
OR			
Indomethacin	50 mg three times daily	1 to 2 weeks	Decrease the dose weekly
PLUS			
Colchicine	0.5 mg two times daily	3 months	Usually not tapered
For initial combination therapy of patients following myocardial infarction:			
Aspirin	650 to 1000 mg three times daily	1 to 2 weeks	Decrease the dose weekly
PLUS			
Colchicine	0.5 mg two times daily	3 months	Usually not tapered
For refractory cases or patients with a contraindication to NSAID therapy:			
Prednisone	0.2 to 0.5 mg/kg/day	2 weeks	Slow tapering, see text
PLUS			
Colchicine	0.5 mg two times daily	3 months	Usually not performed

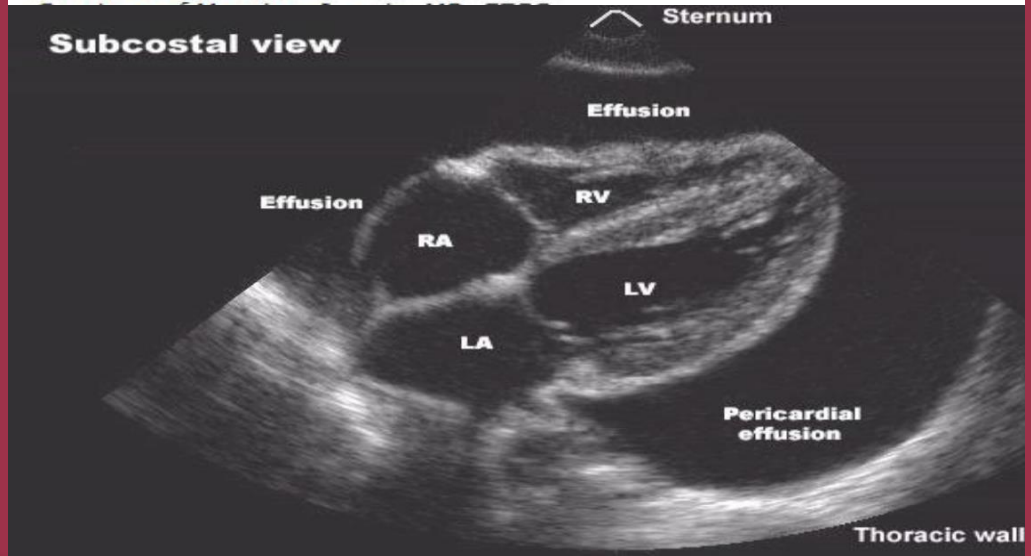
Initial clinical and echocardiographic evaluation of patients with suspected acute pericarditis



Pericardial Effusion: CXR and Echo

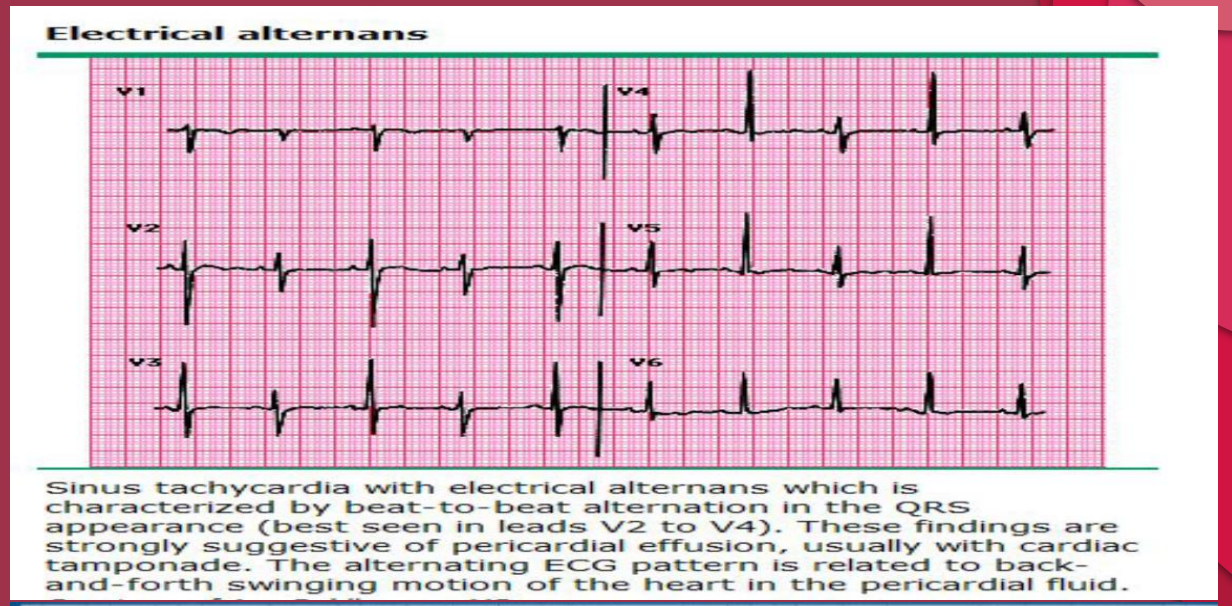
- Echocardiography is often normal in patients with the clinical syndrome of acute pericarditis unless there is an associated pericardial effusion.
- Chest radiography is typically normal in patients with acute pericarditis. Although patients with a substantial pericardial effusion may exhibit an enlarged cardiac silhouette with clear lung fields
- On CXR → Water-bottle / Flask-shaped / Boot-shaped heart
- On Echo → Collapsed Rt ventricle since it's very thin compared to LV so it can't resist pressure.



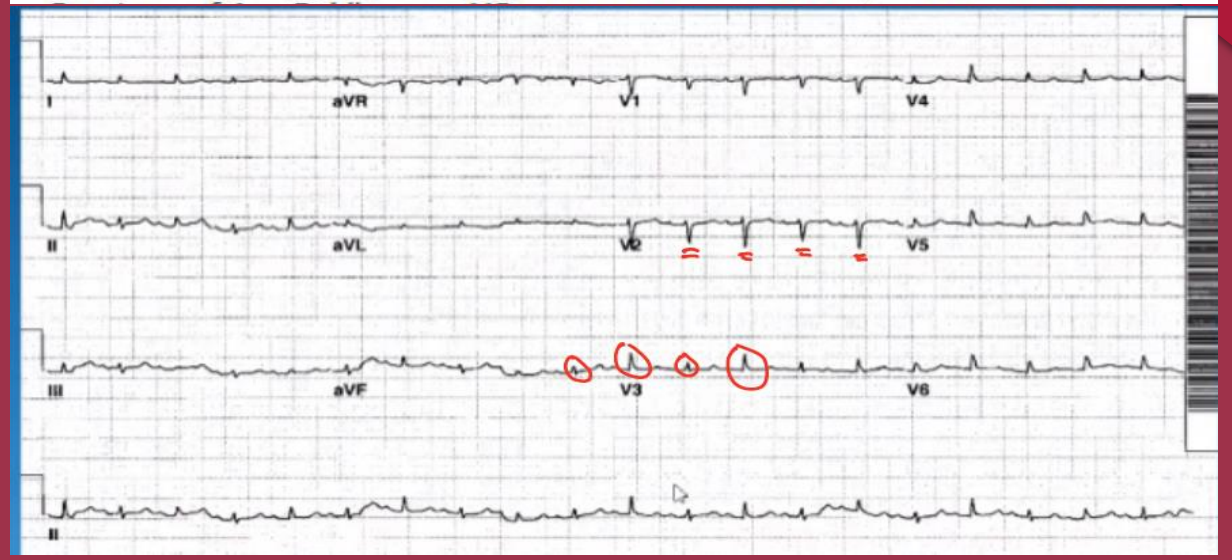


Pericardial Effusion

- Notice on the second ECG that we have low voltages + QRS complex amplitude is alternating between large and small in different beats (check leads V2-V3).

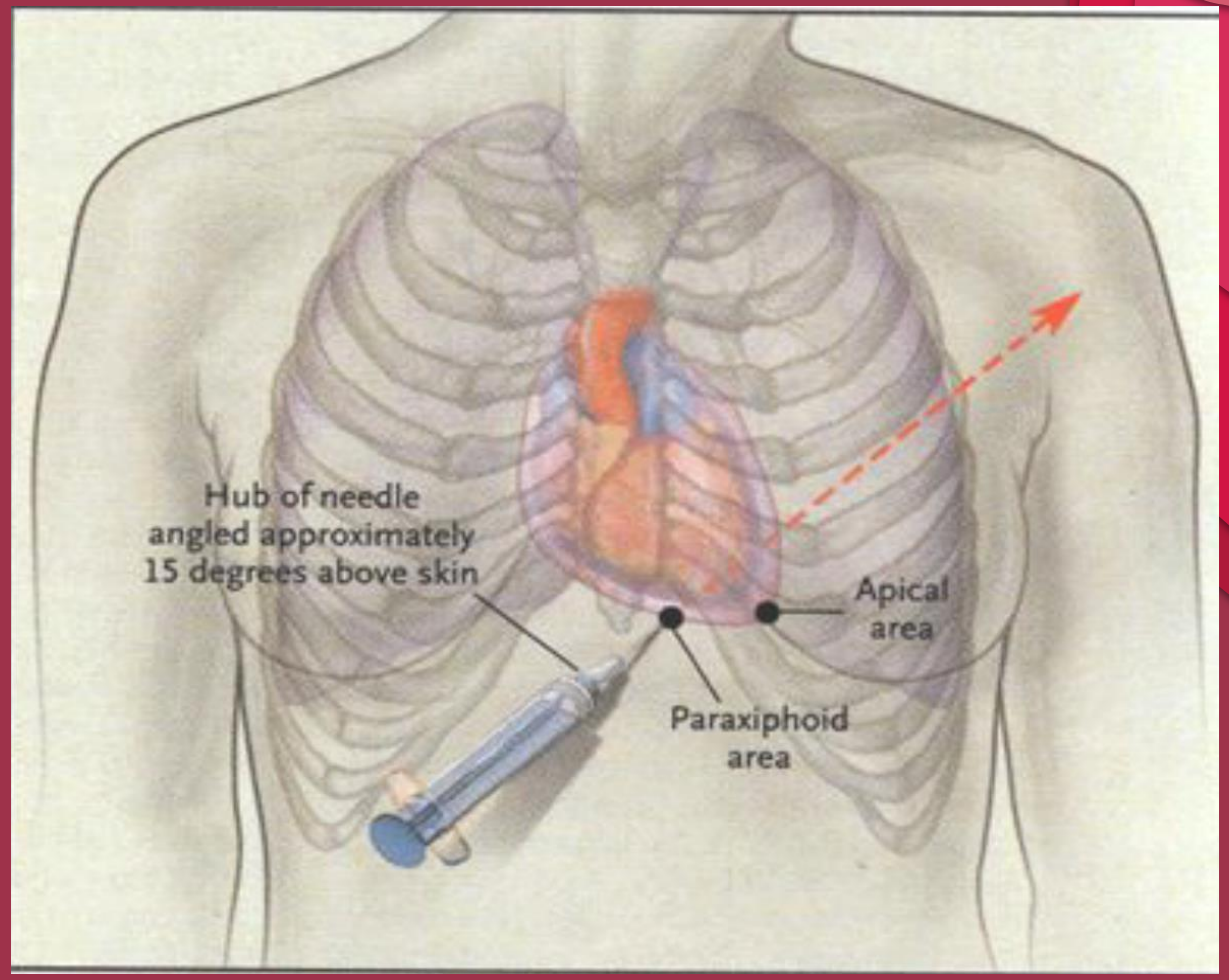


Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with cardiac tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid.



Pericardial Effusion

- Pericardiocentesis: Determine the site of xyphoid protuberance then go just to the left of it to insert the needle at 45 degrees toward the heart gently until reaching the fluid, then connect it to a drain. Along with this, you watch through fluoroscopy in the cath lab and perform Echo at the same time.

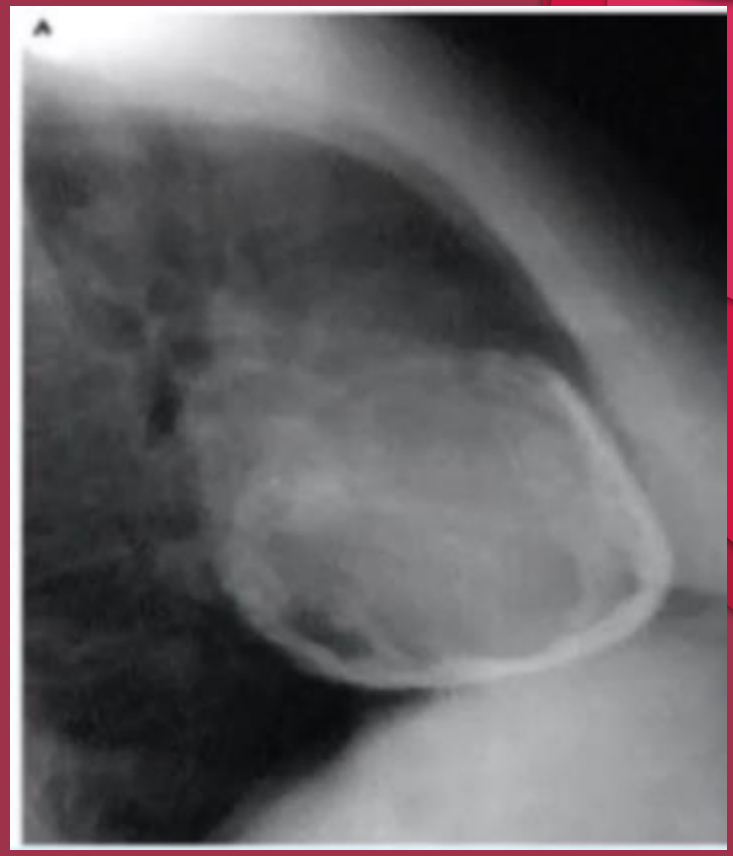


Pericardial Tamponade

- Causes: Trauma, Uremia (cause painless pericarditis and large effusions in some patients), Anticoagulation (warfarin causing bleeding in the pericardium), Neoplasm (most commonly Breast CA, Lymphoma), Infection.
- Clinical signs (Beck's triad): Hypotension + Elevated JVP (due to collapsed Rt ventricle + Muffled heart sound (due to fluid covering the heart) + Pulsus Paradoxus (also found in bronchial asthma and exacerbation of bronchiectasis).
- ECG: Electrical alternans → beat to beat alteration in the amplitude of the QRS complex (QRS is sometimes large and sometimes small since the heart is swinging inside the fluid, when it comes closer to the chest wall then QRS is large, when it's away from the chest then QRS is small) + Low Voltage.
- Echo finding: Effusion + RV diastolic collapse (specific for tamponade).
- Patients also come with Dyspnea, Tachycardia.
- You must do urgent Transthoracic Echo (TTE) → Effusion + Diastolic collapse of Rt-sided chambers + Increased respiratory variation of peak inflow velocities through TV and MV + Dilated IVC without respirophasic variation.
- If the patient is hemodynamically stable: Give IV fluid + Close monitoring + Serial pulsus + Serial TTE + Treat underlying cause.
- If the patient is unstable: Give aggressive IV fluids + Dobutamine if underlying LV dysfunction + Pericardiocentesis or surgery + Intra-aortic balloon pump for refractory hypotension + Minimize PEEP.

Constrictive Pericarditis

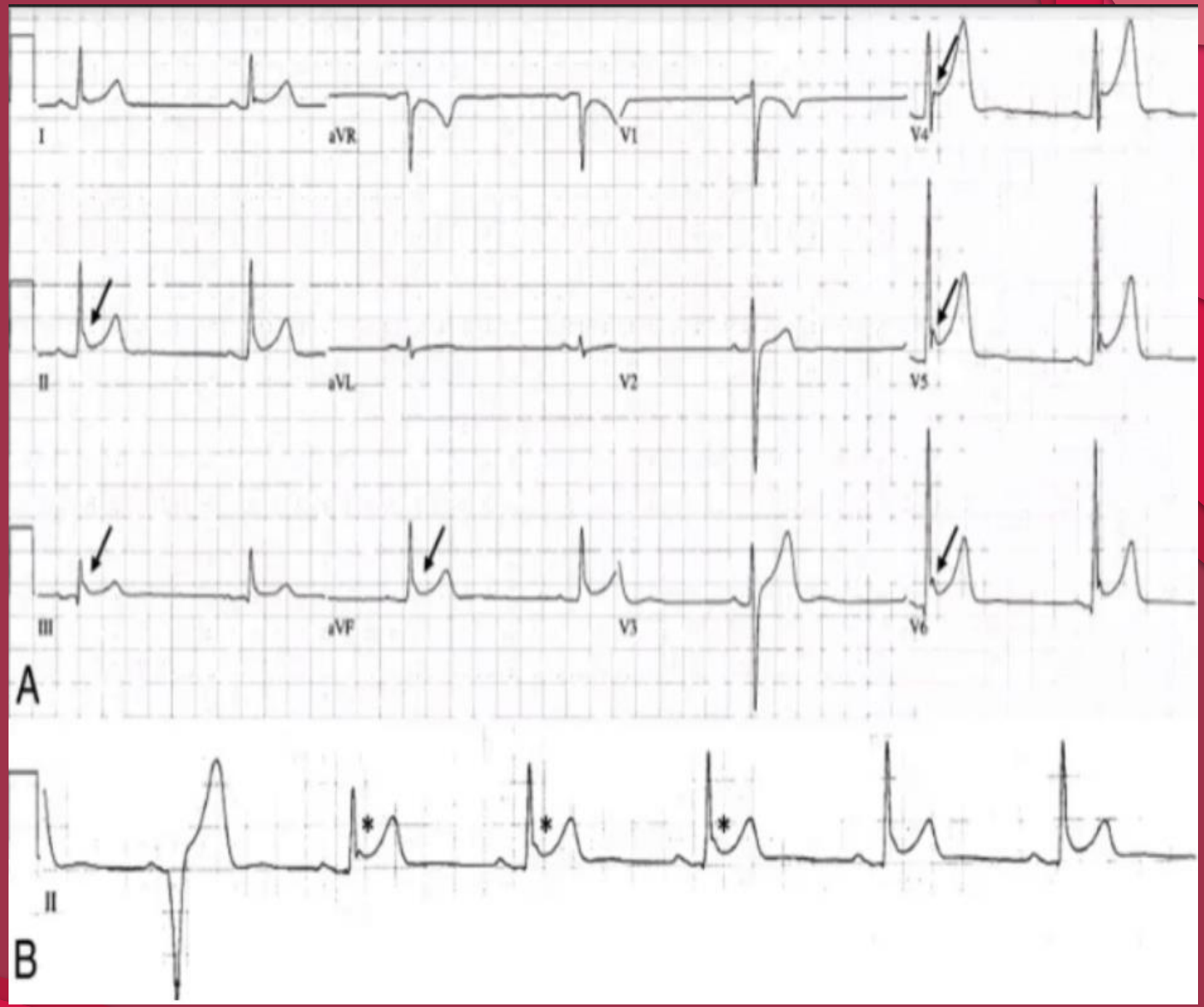
- Thick dense fibrous obliteration with calcification of the pericardial sac encasing the heart.
- Causes: Cardiac surgery + Viral infection + Acute pericarditis + Mediastinal irradiation (more common than TB) + Rheumatoid Arthritis + Connective tissue disease
- Limiting diastolic expansion and restricting cardiac output.
- Not every case include calcification.
- The adjacent figure illustrates a calcified pericardium.
- Evidence of Rt HF
- Kussmaul sign: No fall or even elevation in JVP with inspiration (due to restriction of dilation of the heart so there is no venous filling with inspiration).
- Abnormal Echo
- Patients present with Dyspnea, Fatigue, Elevated JVP, Hepatomegaly and Ascites, Edema of the lower limb, Neck veins distend with inspiration (Kussmaul sign), Pericardial knock (rapid ventricular filling -S3- due to restriction of filling blood. Emptying of atria to ventricle rapidly producing pericardial knock), A Fib in 20%.
- Management: Diuresis with caution + Rate control with caution (if there is A Fib) + 2-3 months trial of conservative measures prior to pericardiectomy + Pericardiectomy.



Case #1

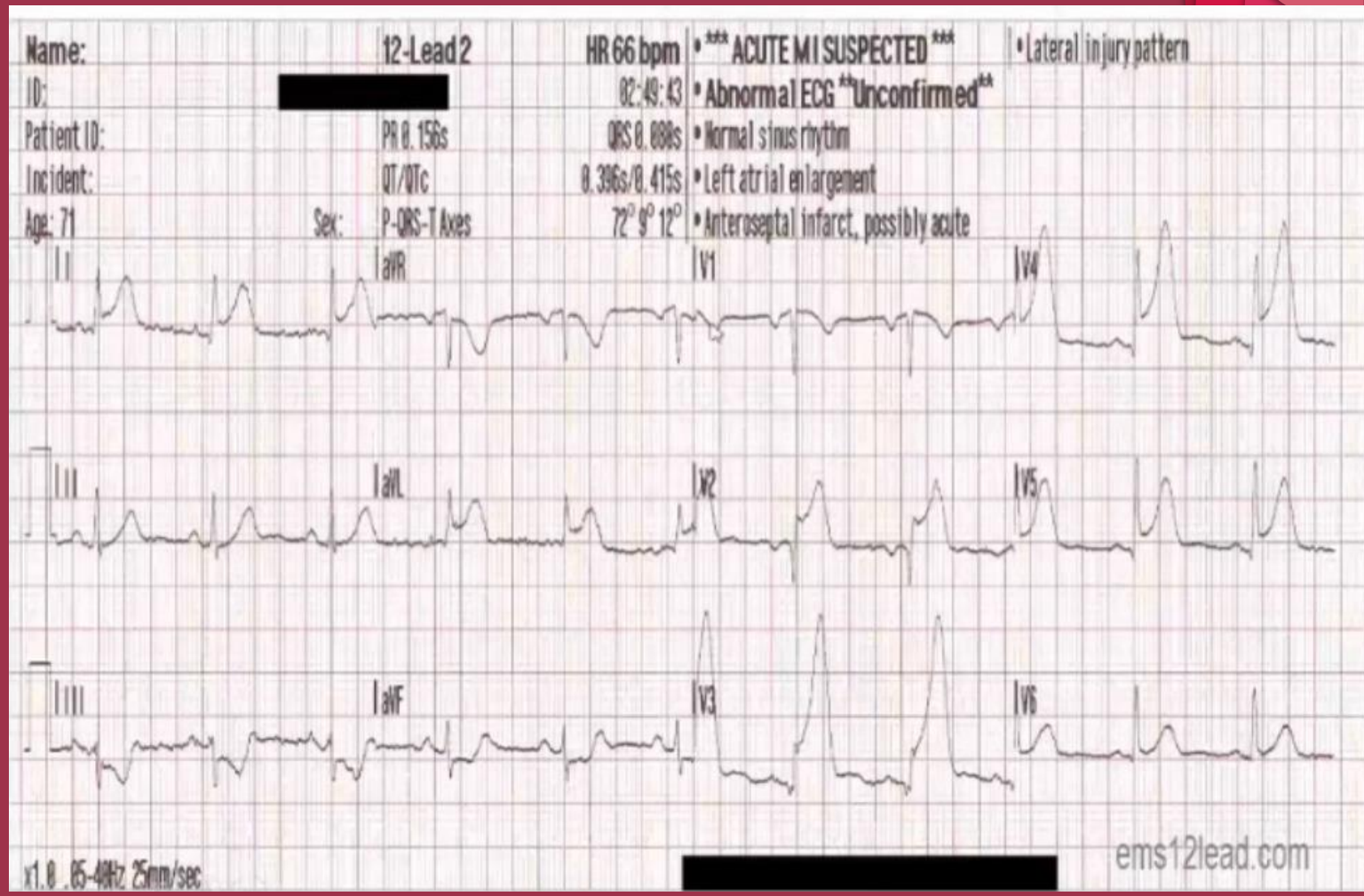
- A 32-year-old male patient presented to your office for an ECG as ordered by his insurance company.

- The ECG shows ST elevations called reciprocal changes found as notching at the end of the QRS indicating repolarization abnormality and it occurs mainly in young, thin individuals.



Case #2

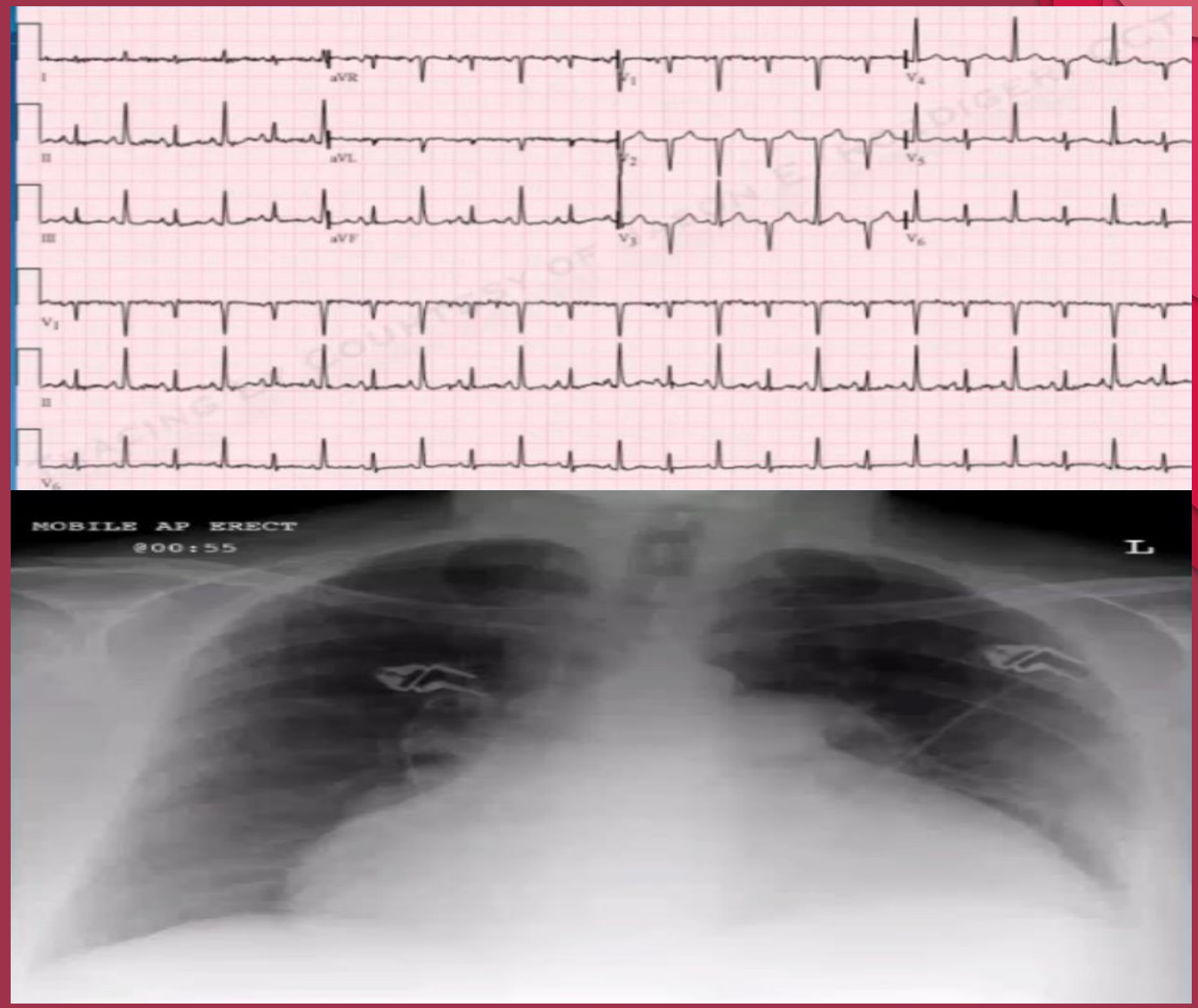
- A 46-year-old male patient with PMHx of HTN and Smoking presented to the ED with chest pain.
- The ECG shows Convex ST elevation in leads I, V2, V3, V4, V5, V6 → Anterolateral STEMI due to proximal LAD occlusion.



Case #3

- A 25-year-old male patient presented with worsening dyspnea to the ED. His HR was 140 bpm and BP 80/40 mm Hg.

- The ECG shows Low voltage + Electrical alternans (QRS amplitude is different) → Pericardial Effusion.



The End

BEST OF LUCK