MANSOURA MANCHESTER PROGRAMME FOR MEDICAL EDUCATION

INTERNAL MEDICINE

CARDIOLOGY

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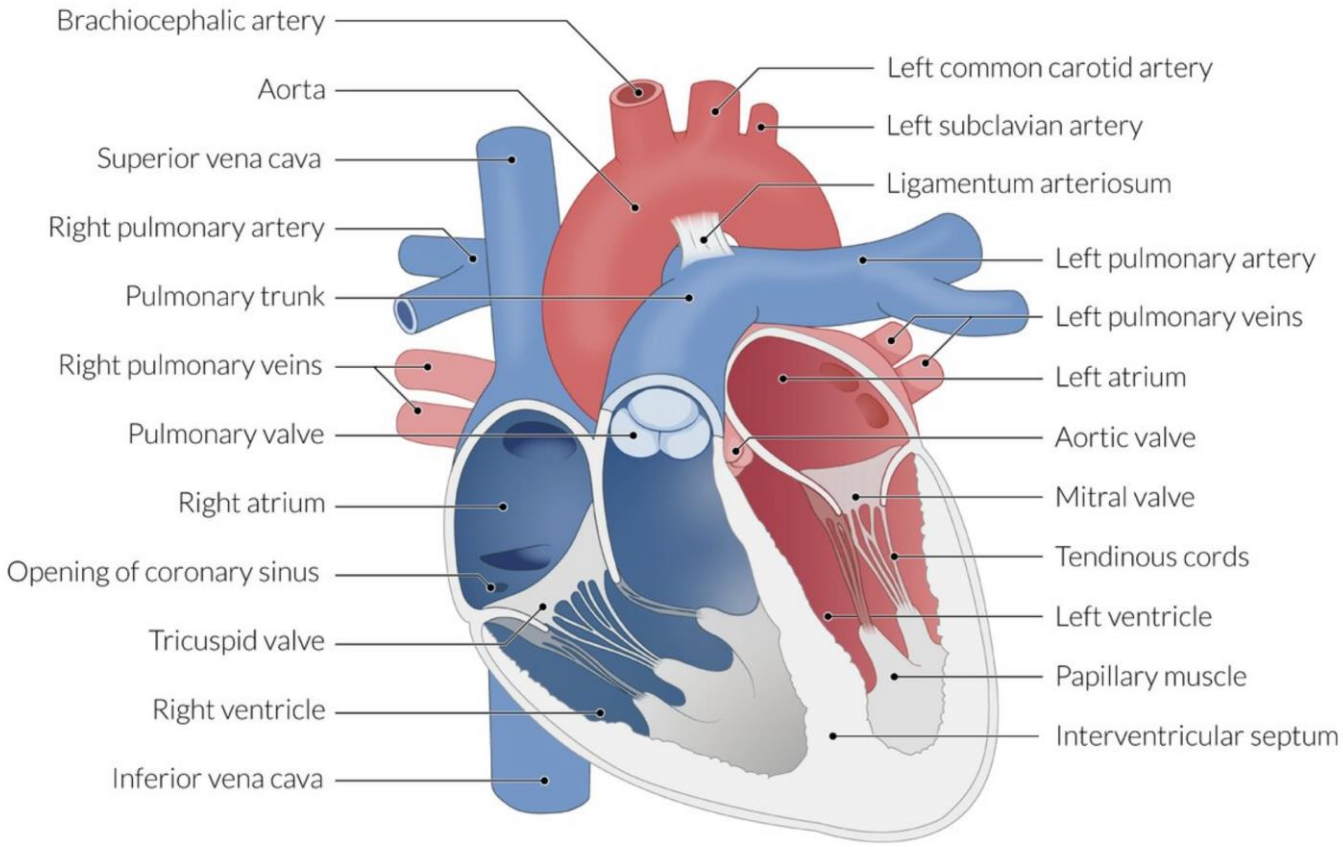
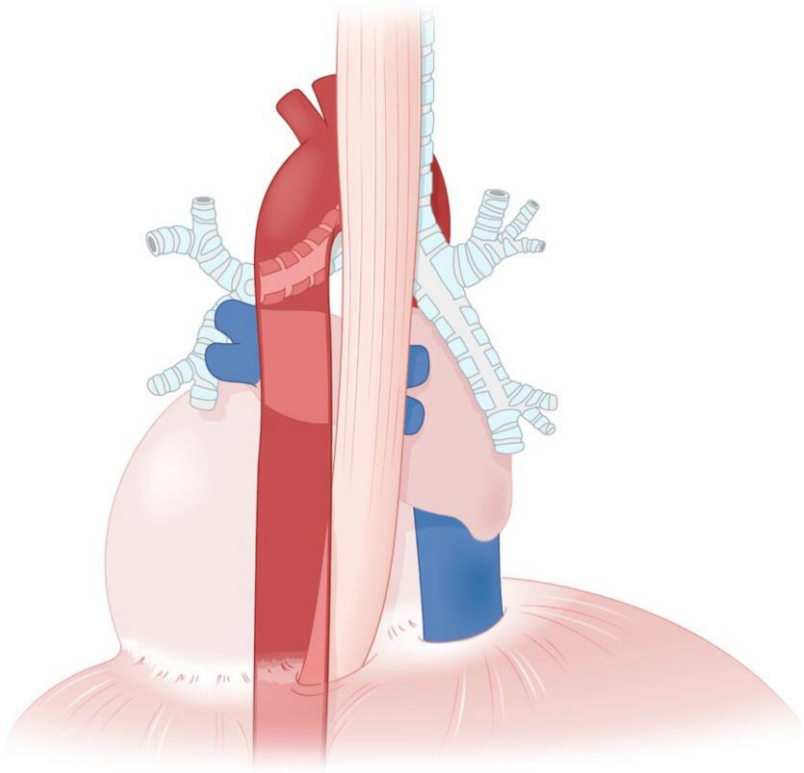
INDEX

INTRODUCTION TO CARDIOLOGY

Anatomy of heart & adjacent structure





Atrioventricular valves

STRUCTURE: leaflets supported by subvalvular apparatus.

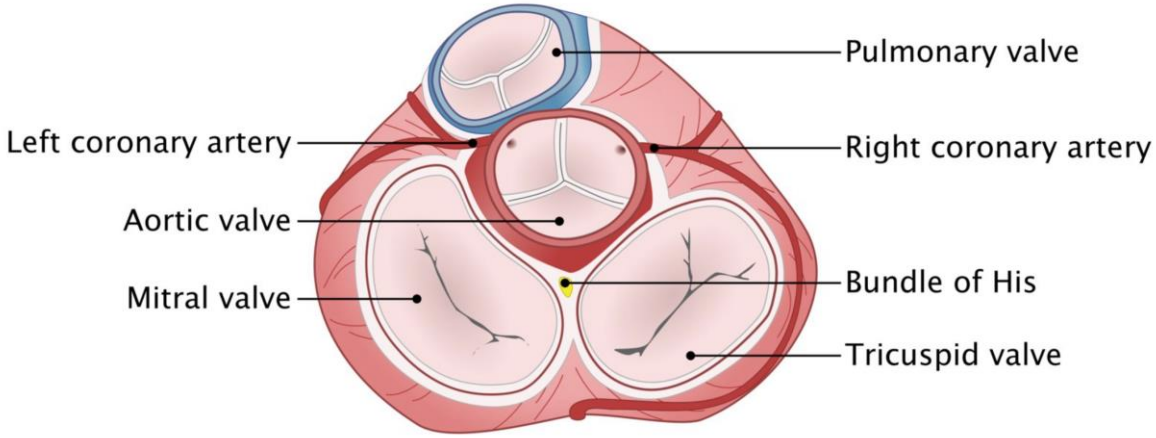
VALVES

	Tricuspid valve	Mitral valve (bicuspid valve)
STRUCTURE	Consists of **three** leaflets	Consists of **two** leaflets
SITE	Between **right** atrium and **right** ventricle	Between **left** atrium and **left** ventricle

SUBVALVULAR APPARATUS :

CHORDAE TENDINEAE	◆ Fibrous cords that **support** AV valves and **connect** them to papillary muscles.
PAPILLARY MUSCLES	◆ **Two** in the left ventricle & **Three** in the right ventricle. ◆ Derive from the **myocardium**. ◆ **Extend from** anterior and posterior ventricular walls and the septum ◆ Have apices that are attached to the **chordae tendineae** ◆ Contract during systole and thereby tighten the chordae tendineae: prevent prolapse of valve leaflets and regurgitation into the atria when pressure rises during ventricular contraction.

- The **tri**cuspid valve has **three** leaflets and is located on the **right** side, as is the **three-lobed right lung.**
- The **bi**cuspid (mitral valve) has **two** leaflets and is located on the **left** side, as is the **two-lobed left lung.**



Semilunar valves

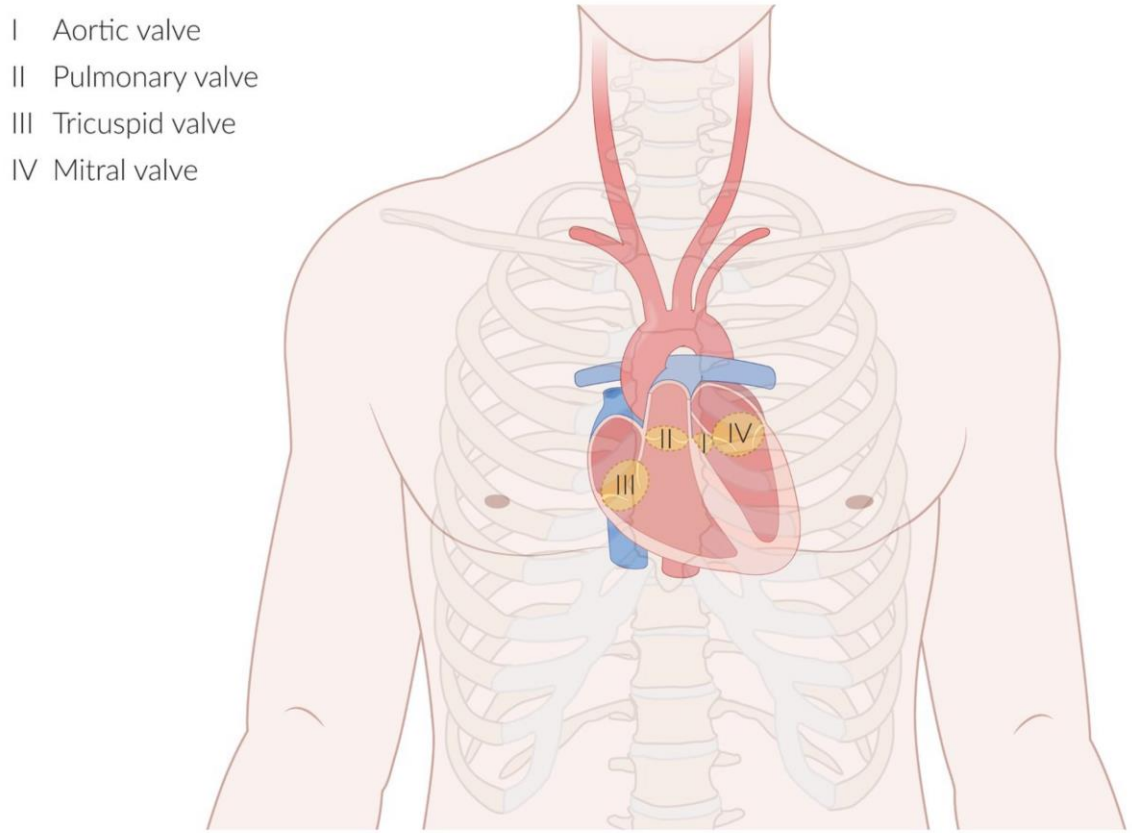
STRUCTURE: three crescent-shaped cusps **without** subvalvular apparatus

VALVES:

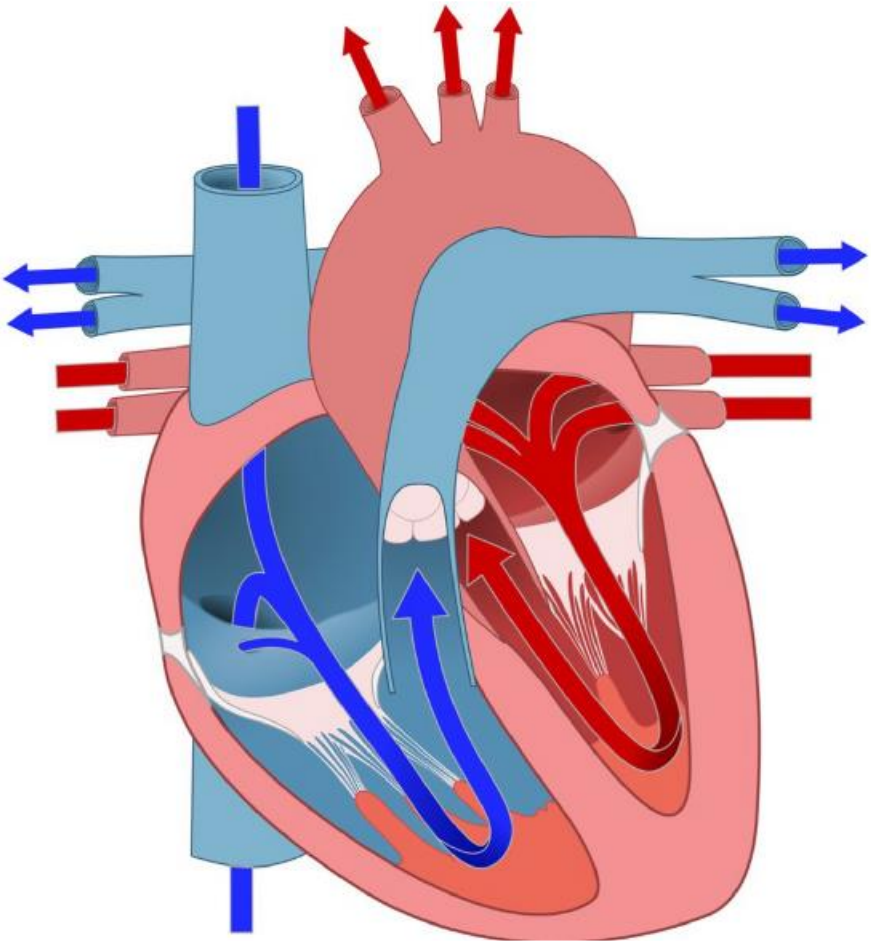
	Pulmonary valve	Aortic valve
STRUCTURE	Consists of **three** leaflets	Consists of **three** leaflets
SITE	Between **right** ventricle and **pulmonary trunk**	Between **left** ventricle and **aorta**

AORTIC VALVE:

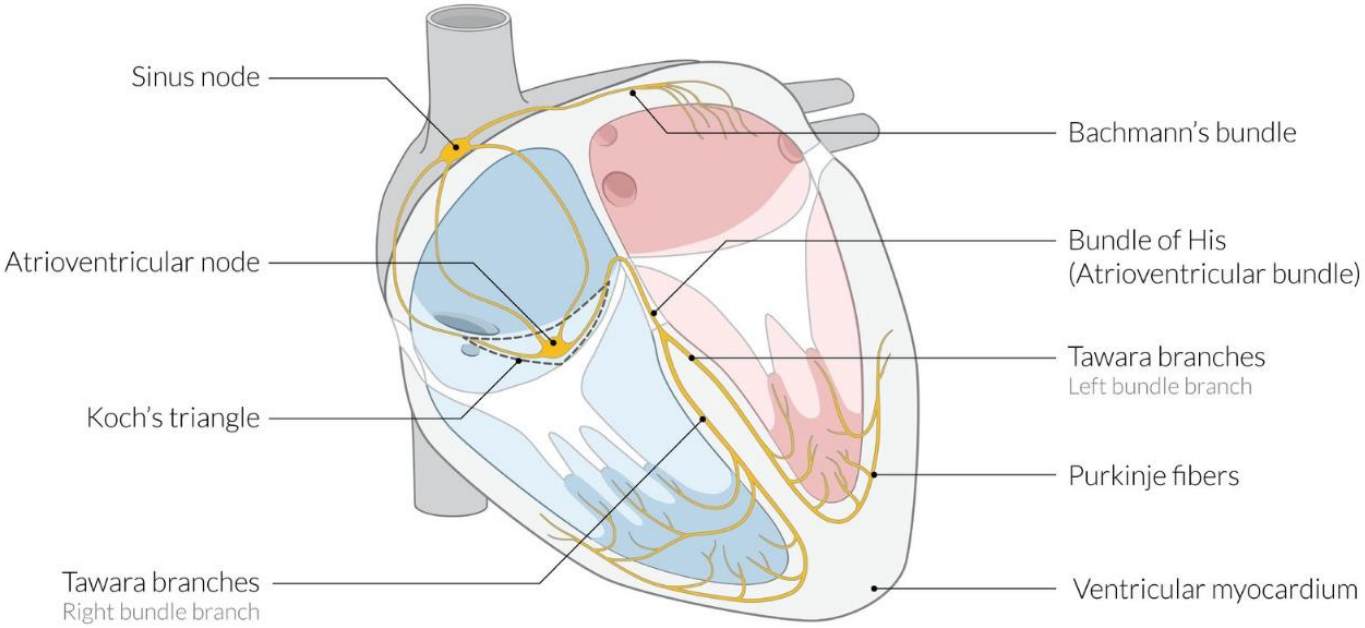
- Consists of three leaflets and the **aortic sinuses** (a space immediately above the aortic valve, between an aortic valve leaflet and the wall of ascending aorta).
  - There are three aortic sinuses (one above each aortic leaflet): the left, right, and posterior aortic sinuses.
  - The left and right aortic sinus give rise to the left and right coronary artery.
  - The three aortic sinuses together with the aortic valve leaflets form a dilation of the root of the aorta called the aortic bulb.



Overview of cardiac physiology



BLOOD FLOW THROUGH HEART.



NORMAL SEQUENCE OF CARDIAC IMPULSE.

Definitions

• HEART RATE (HR)

Definition	◆ The number of heart contractions per minute (bpm)
Normal	◆ 60-100 bpm

• STROKE VOLUME (SV)

Definition	◆ Volume of blood pumped by the left or right ventricle in a single heartbeat
Calculation	◆ SV = end-diastolic volume (EDV) - end-systolic volume (ESV)

• EJECTION FRACTION (EF):

Definition	◆ The proportion of EDV ejected from the ventricle
Calculation	◆ EF = SV/EDV = (EDV-ESV)/EDV
Normal	◆ Normally 50-70%
Imp.	◆ Serves as an index of **myocardial contractility**: eg. ↓ myocardial contractility →↓ EF (seen in systolic heart failure, where EF is < 40%) ◆ Low in systolic heart failure and usually normal in diastolic heart failure

• VENOUS RETURN:

Definition	◆ Rate at which blood flows back to the heart.
Normal	◆ Equals cardiac output

• CARDIAC OUTPUT:

Definition	◆ Volume of blood the heart pumps through the circulatory system per minute
Calculation	◆ **Cardiac output (CO)** = heart rate (HR) x stroke volume (SV). ◆ During physical activity (when SV becomes constant), an increase in cardiac output is mediated by increasing heart rate.
Normal	◆ 5 L/min at rest

• PRELOAD:

- The extent to which heart muscle fibers are stretched before the onset of systole: depends on end-diastolic ventricular volume (EDV), which changes according to:

Venous constriction	• ↑venous tone →↑ venous blood return to the heart →↑ EDV →↑ preload
Circulating blood volume	• ↑circulating blood volume →↑ venous blood return to the heart→↑ EDV →↑ preload

• AFTERLOAD:

- The force against which the ventricle contracts to eject blood during systole
- Afterload is primarily determined by the **mean arterial pressure (MAP)** in the aorta, which is influenced by total peripheral resistance.
- ↑ Afterload →↑ left ventricular pressure→ ↑ left ventricular wall stress

CARDIOLOGY SCHEME

DEFINITION : esp. as regard

- Heart Failure.
- Ischemic heart disease.
- Hypertension.

ETIOLOGY: Enumeration

CLINICAL PICTURE :

SYMPTOMS + SIGNS (GENERAL SIGNS + LOCAL SIGNS)

A) Symptoms :

1- PULMONARY CONGESTION & SYSTEMIC CONGESTION SYMPTOMS :

	Pulmonary Congestion symptoms	Systemic Congestion Symptoms
Etiology	◆ Occur in Lt sided Ht Failure ◆ D2 failure of heart to accept pulmonary venous return	◆ Occur in Rt sided Ht failure ◆ D2 failure of heart to accept systemic venous return
Symptoms	◆ Dyspnea ◆ Cough ◆ Hemoptysis	◆ Confusion ◆ Insomnia ◆ Rt Hypochondrial pain ◆ Anorexia-Nausea-Vomiting ◆ Dyspepsia & epigastric pain ◆ Edema of dependant parts ◆ Abdominal swelling d2 ascites

2- LOW CARDIAC OUTPUT SYMPTOMS :

Etiology	◆ Occur in Lt or Rt sided Heart failure or **both** d2 failure of Heart to eject sufficient CO.
Symptoms	◆ **Brain :** Dizziness – Headache – Syncope ◆ **Eyes :** Blurring of vision ◆ **Heart :** Angina pectoris ◆ **Kidneys :** oliguria ◆ **Muscles :** Easy fatigability , intermittent claudication ◆ **Skin :** Pallor & coldness ,peripheral cyanosis in severe cases

3- PALPITATION :

Definition	◆ It is **unpleasant awareness** of the heartbeat
Etiology	◆ **Change in rate:** Tachycardia / bradycardia ◆ **Change in rhythm:** Arrythmia ◆ **Change In force:** Forceful cardiac contraction ◆ **Psychoneurosis**

4- CHEST PAIN :

◆ **PAIN IN THE CARDIAC ORIGIN MAY ARISE FROM :**

Pericardium	◆ Pericarditis
Myocardium	◆ Angina pectoris & myocardial infarction
Endocardium	◆ Mitral valve prolapsed
Aorta	◆ Aortic aneurysm
Pulmonary	◆ Pulmonary embolism

◆ **YOU MUST COMMENT ON THE FOLLOWING SEVEN ITEMS :**

1. Site?
2. Character?
3. Radiation?
4. Duration?
5. Precipitating factors?
6. Relieving factors?
7. Association?

5- PRESSURE SYMPTOMS :

◆ Occur d2 pressure of cardiac chambers on surrounding structures

◆ **Enlarged Lt Atrium / Aortic aneurysm / Pericardial effusion may compress :**

Esophagus	◆ Dysphagia
Tracheobronchial tree	◆ Dyspnea & cough
Left RLN	◆ Hoarsness of voice
SVC	◆ Swelling of the face & puffiness of eyelids
Spine	◆ Back pain

6- CYANOSIS

◆ **Bluish** discoloration of skin & maybe mucus membranes d2 presence of **more than 5gm** reduced Hemoglobin/100cc of blood

B) Signs :

General Signs

Signs of Pulmonary congestion	◆ Tachypnea ◆ Bilateral fine basal crepitation ◆ Pleural effusion
Signs of Systemic Congestion	◆ **Congested** pulsating neck veins ◆ **Liver :** Enlarged ,tender, +/ – (pulsating – hepatojugular reflux) ◆ **Edema** LL followed by ascites
Signs of low COP	◆ **Pulse :** rapid – weak ◆ **B.P :** low SBP ◆ **Skin :** cold ,peripheral cyanosis
Pallor	◆ **In cardiac disease, pallor may be due to:** a. Anemia. b. Rheumatic activity. c. Infective endocarditis. d. Low cardiac output. e. Shock.
Jaundice	◆ **In cardiac disease, jaundice may be due to:** a. Severe right sided heart failure. b. Pulmonary embolism.
Orthopnea	◆ **Orthopnea :** left-sided heart failure. ◆ **Prayer's position (sitting up & Cleaning forward):** pericardial effusion. ◆ **Squatting :** the Tetralogy of Fallot.
Fever	◆ **In cardiac disease, fever may be due to** a. Rheumatic activity. b. Infective endocarditis. c. Pericarditis. d. Myocarditis. e. Myocardial infarction. f. Pulmonary infarction. g. Pulmonary infection.
Clubbing	◆ **In cardiac disease, it occurs in:** a. Infective endocarditis (pale clubbing). b. Cyanotic cardiac conditions (cyanotic clubbing)

Local Signs

A- INSPECTION & PALPATION

<table>
<tr><td>1- Apex
[SECRT]</td><td>
<table>
<tr><td>Site</td><td>◆ LVE: shifted outwards and downwards
◆ RVE: shifted outwards</td></tr>
<tr><td>Extent</td><td>◆ LVE: localized.
◆ RVE: diffuse</td></tr>
<tr><td>Character</td><td>◆ Pressure overload: heaving
◆ Volume overload: Hyperdynamic
◆ Ms : slapping</td></tr>
<tr><td>Relation e systole</td><td>◆ LVE: syst Bulge
◆ RVE : syst retraction</td></tr>
<tr><td>Thrill</td><td>◆ MS: diastolic thrill
◆ MR: systolic thrill</td></tr>
</table>
</td></tr>
<tr><td>2- Lt parasternal area</td><td>◆ Pulsation: RVE
◆ Thrill: VSD</td></tr>
<tr><td>3- pulmonary area</td><td>◆ Pulsation: Pulmonary hypertension
◆ Thrill: PS</td></tr>
<tr><td>4-Aortic area</td><td>◆ Pulsation : aortic aneurysm
◆ Thrill : AS</td></tr>
<tr><td>5- Epigastric area</td><td>◆ Pulsation:
a) RVE.
b) Hepatic pulsation.
c) Aortic.</td></tr>
</table>

B- AUSCULTATION

1-HEART SOUNDS :

	First heart sound:	Second heart sound:
Cause	◆ closure of mitral and tricuspid valve.	◆ closure of aortic and pulmonary valves.
Timing	◆ beginning of systole.	◆ Beginning of diastole
Increase in	◆ MS , TS, and tachycardia.	◆ systemic and pulmonary hypertension
Decrease in	◆ TR, MR, bradycardia.	◆ AS, AR, PS, PR
Site	◆ Apex and tricuspid areas.	◆ aortic and pulmonary areas
Splitting		◆ Increased in PS, ASD, RBBB ◆ Decreased in AS, PDA, LBBB

	Third heart sound:	Fourth heart sound:
Cause	◆ Increased Blood flow from atrium to ventricle ◆ Flabby myocardium as HF, or ischemic heart	◆ powerful atrial contraction
Timing	◆ in diastole isolated from 2nd heart sound by isometric relaxation	◆ Presystolic, before 1st heart sound
Site	◆ at apex or tricuspid area	◆ at apex or tricuspid.

2. ADDITIONAL SOUNDS :

	Opening snap:	Ejection click:
Cause	◆ Opening of rigid mitral or tricuspid valve as in MS, TS.	◆ Opening of rigid aortic or pulmonary valves as in Opening of aortic and pulmonary valves against high p pulmonary and systemic hypertension
Timing	◆ In diastole being separated from 2"° heart sound by isometric relaxation phase	◆ In systole being isolated from 1st heart sound by isometric contraction phase.
Site	◆ At apex and tricuspid.	◆ Aortic and pulmonary area

3. MURMURS :

- **SITE.**
- **PROPAGATION :**

Lesion	Site Of maximum intensity	Area of propagation
MS	Mitral area; Apex	Local
TS	Tricuspid area	Local
MR	Mitral area; Apex	If anterior leaflet: Axilla If posterior leaflet: back (left scapular region) & sternum
TR	Tricuspid area	Apex
AS	1st aortic area	Apex & carotids
PS	Pulmonary area	Tricuspid area & may be under left
AR	2nd aortic area	Apex
PR	Pulmonary area	Tricuspid Area
VSD	Left parasternal area	Whole precordium
Aortic coartication	Inter scapular area	Anteriorly

- **CHARACTER:**

Soft	◆ any regurge murmur ◆ All incompetent murmurs & now
Harsh	◆ any stenotic murmurs. ◆ VSD, COA& all stenosis murmurs except MS
Machinery	◆ PDA
Rumbling	◆ MS & Ts
Musical "Sea-gun murmur":	◆ due to rupture or valve complex (cusps chordea & papillary muscle).

- **RELATION TO RESPIRATION:**
  - **Left side:** increases with expiration .
  - **Right side:** increases with inspiration.

- **RELATION TO POSITION :**
  - **Any mitral murmur:** increases with lying on the LT lateral position .
  - **Any aortic murmur:** increases by leaning forward.

Posture "Position"	◆ **Mitral murmurs :** best heard in left lateral position
Respiration	◆ **Right (tricuspid& pulmonary) Sided murmurs:** ↑ by inspiration (Carvallo's sign) (due to ↑ VR→↑ blood flow) ◆ **carvallo's sign:** differentiate between TR & MR
Exercise	◆ ↑ MS, MR, & AR

◆ **TiMiNG :**

A. Systolic		
Early systolic	**Ejection systolic**	**Late systolic**
◆ Very small VSD	◆ AS ◆ PS	◆ Mitral valve prolapse (MVP) ◆ Hypertrophic obstructive cardiomyopathy
B. Diastolic		
Pan or holosystolic	**Early diastolic**	**Mid & late diastolic**
◆ MR ◆ TR ◆ VSD	◆ AR ◆ PR (Graham steel mumur)	◆ MS & TS ◆ Carey Coomb's murmur: (functional MS in RF)
C. Continuous (systolic & diastolic murmur):		
◆ Venous hum: jugular vein hum in the neck ◆ Arteria-venous fistula: systemic, pulmonary & coronary ◆ Patent Ductus Arteriosus (loudest in 2nd left interspace) ◆ Double valve lesion: AS/AR - MS/MR ◆ After Block-Taussig operation		

◆ **THRiLL & GRADE :**

- Thrill is Palpable loud murmurs like sensation of purring cat
- Grade 4/6 at least.
- Due to organic & not functional valve disease.

Systolic murmur	Diastolic murmur	Functional murmur
Harsh + Marked thrill	Soft + minimal thrill	No thrill

iNVESTiGATiONS :

X-ray	◆ Chamber enlargement. ◆ Pulmonary congestion in left sided diseases. ◆ Pleural effusion.
ECG	◆ Chamber enlargement. ◆ Detect the cause.
Echo	◆ Chamber enlargement. ◆ Detect the cause. ◆ Paradoxical movement of the myocardium.
Catheterization	◆ Chamber enlargement. ◆ Detect the cause.
Other imaging	◆ radio isotope. CT , MRK
Laboratory	◆ CBC, Lipid profile, blood glucose, liver & renal function tests

NOTiCE : iN THE FOLLOWiNG ADD THE FOLLOWiNG
◆ **In myocardial infarction** → Cardiac enzymes.
◆ **In infective endocarditis** → Blood culture.
◆ **In pulmonary embolism** → Pulmonary angiography ,spiral CT , D dimer.
◆ **In Cardiomyopathy** → Biopsy.

TREATMENT

1- **Treatment of the cause.**
2- **Treatment of precipitating factors**: e.g. hyperlipidemia:
   - Statins : 20 - 80 mg/d SE: myositis.
   - Fenofibrate: 300 mg/d
   - Omega 3 (fish oil)
3- **Specific ttt :** A2 disease.

HEART FAILURE

DEFINITION: "New Universal Definition of HF: 2021"

- **HF is confirmed if a patient has clinical features of HF attributable to structural or functional cardiac abnormalities and either:**
  - Elevated natriuretic peptides.
  - Evidence of cardiogenic pulmonary or systemic congestion.

ETIOLOGY:

	LVF	RVF
Pressure overload	✪ **Systemic** HTN ✪ **Valvular disease:** AS ✪ **Congenital disease:** Coarctation of aorta	✪ **Pulmonary** HTN (e.g. COPD) ✪ **Valvular disease:** PS ✪ **Congenital disease:** PS
Volume overload	✪ Hyperdynamic circulation. ✪ **Valvular disease:** AR, MR ✪ **Congenital disease:** VSD	✪ Hyperdynamic circulation. ✪ **Valvular disease:** PR & TR ✪ **Congenital disease:** ASD
	Renal failure, iatrogenic fluid overload	
↓ Contractility	✪ IHD (AMI) Segmental affection ✪ Cardiomyopathy (DCM), Myocarditis Global affection	
↓ Relaxation (Filling)	✪ **Pericardial disease:** Pericardial effusion, Constrictive pericarditis ✪ **Myocardial diseases:** Cardiomyopathy (HCM & RCM)	
Others	✪ Arrhythmias (Tachy or Brady) ✪ Toxic damage Heavy metals (e.g. Copper, Iron Load), Alcohol, Cocaine	
Most common cause	IHD, Systemic HTN	Pulmonary HTN (esp. gp 2 & 3)

PRECIPITATING FACTORS: "TRIGGERS"

- **THESE FACTORS:**
  - May change a state of Stable (Compensated) HF into Acute Decompensated HF.
- **THESE FACTORS:** if not removed during treatment, HF will become Refractory.
- **MOST IMPORTANT TRIGGERS ARE:**
  - **INFECTIONS:** esp. infective endocarditis, rheumatic activity & chest infection.
  - **IATROGENIC:**
    - Nonadherence to heart failure medication
    - NSAID use
    - Drugs with negative inotropic properties (e.g., nondihydropyridine CCBs)
    - Starting or uptitrating beta blockers
    - Thiazolidinediones
    - Recreational substance use, e.g., cocaine, alcohol, methamphetamines
  - **ANEMIA,** thyrotoxicosis & other causes of hyperdynamic circulation.
  - **ACUTE** Coronary Syndrome (e.g. AMI).
  - **ACUTE** Mechanical cause (e.g. Acute MR 2ry to endocarditis).
  - **ACUTE** Pulmonary embolism
  - **ABRUPT** severe ↑ in systemic BP (Hypertensive emergency).
  - **ARRHYTHMIAS** (e.g. AF or Bradyarrhythmias).
  - **PREGNANCY** & late labor, Physical & emotional stress.
  - **VOLUME OVERLOAD:** due to inappropriate fluid / salt intake or IV fluid therapy.

FAMOUS TRIGGERS :

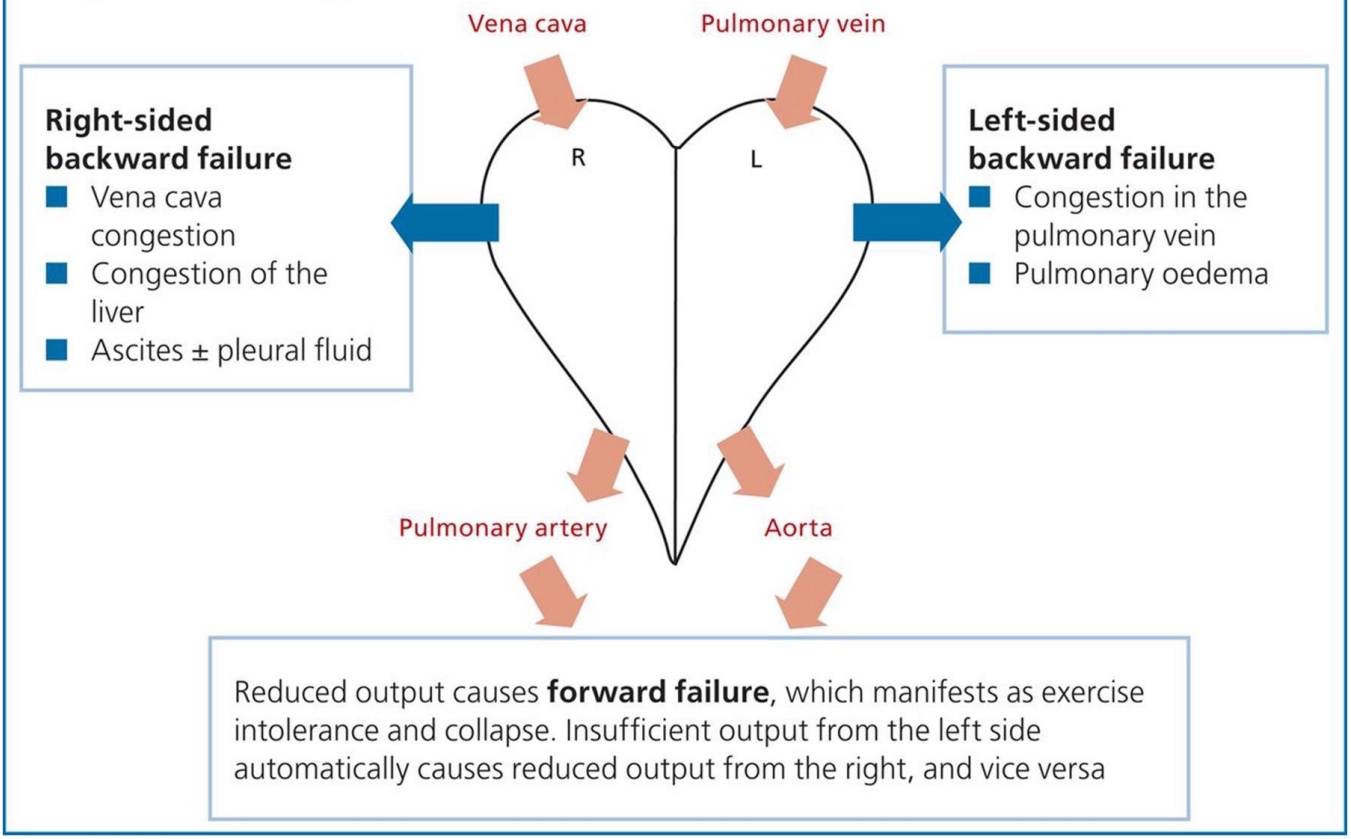
C ⇒ Acute **C**oronary syndrome
H ⇒ **H**ypertensive emergency
A ⇒ **A**rrhythmias
M ⇒ Acute **M**echanical cause
P ⇒ Acute **P**ulmonary embolism

PATHOPHYSIOLOGY:

❶ The clinical manifestations of HF result from:

- **FORWARD FAILURE:** Failure of the heart to eject a sufficient CO ⇒ Low CO.
- **BACKWARD FAILURE:** Failure of the heart to accept the VR ⇒ Congestion.
  - **PULMONARY CONGESTION:** in case of Left - sided HF.
  - **SYSTEMIC CONGESTION:** in case of Right – sided HF.

Diagram showing the clinical features of heart failure



❷ Compensatory mechanisms: (Cardiac Reserve)

- These mechanisms try to restore the Cardiac Output.
- In the short term: They are Adaptive (Beneficial) within limits.
- Over the long term: They may exceed the limits & become Maladaptive (Adverse).

1) Tachycardia:

- **Adaptive (Beneficial) effect:** It ↑ CO.
- **Maladaptive (Adverse) effect:** If it exceeds the limit (HR > 160 bpm) → ↓ V. filling → ↓ CO.

2) Redistribution Of The Blood Flow:

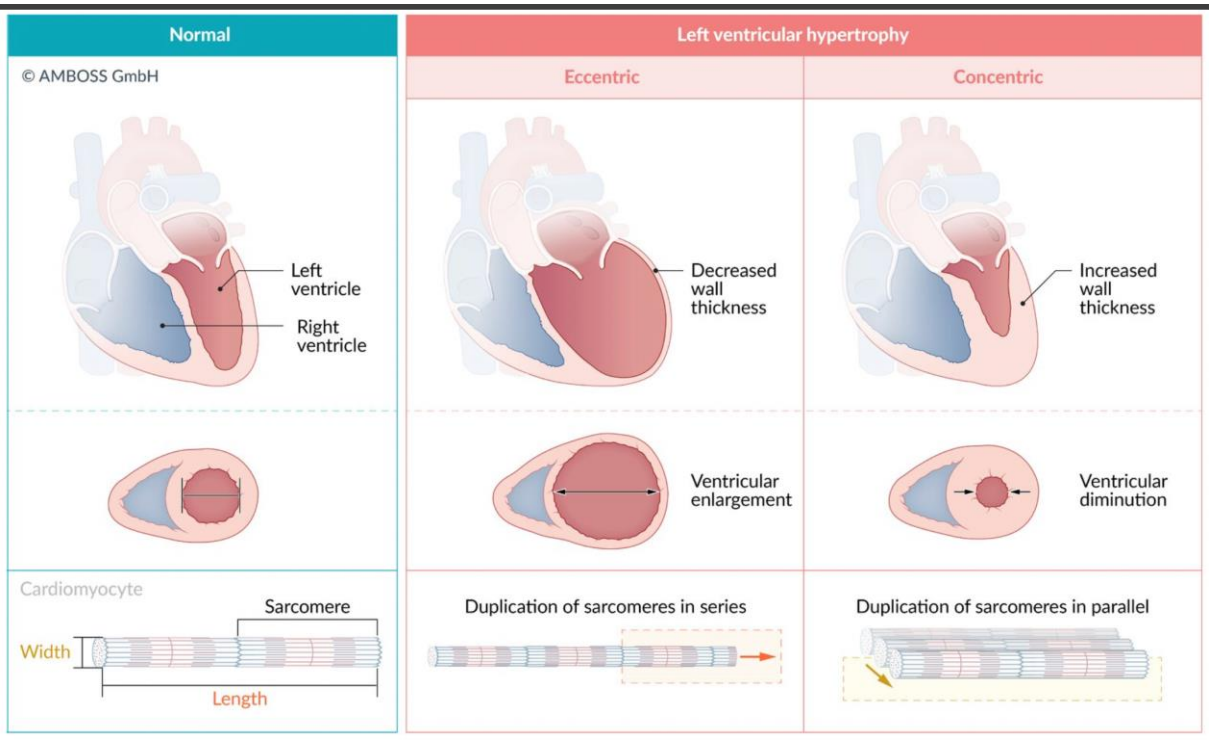
- **From** less vital organs (Skin & Muscles).
- **To** More vital organs (Heart & Brain).

3) Hypertrophy (Concentric Hypertrophy):

- **Increased THICKNESS of cardiac muscle fibre:** in PRESSURE overload.
- **Adaptive (Beneficial) effect:** It ↑ CO (by increasing the force of contraction).
- **Maladaptive (Adverse) effect:** If it exceeds the limit (marked hypertrophy) → ischemic muscle.

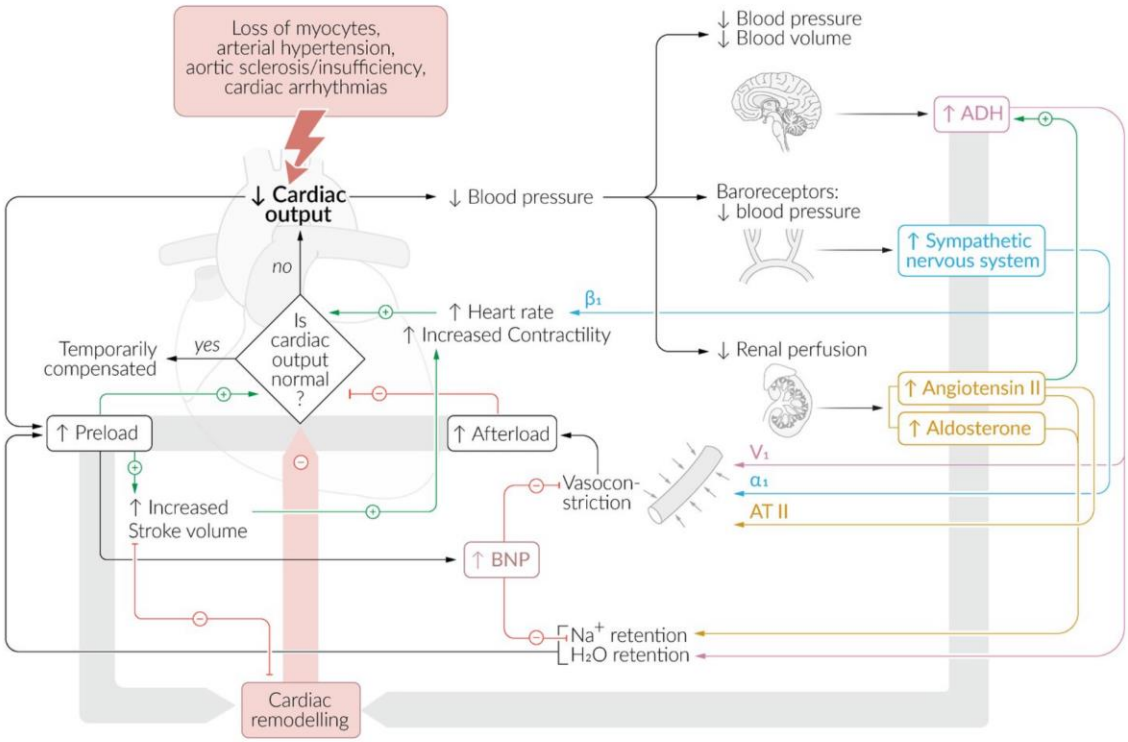
4) Dilatation (Eccentric Hypertrophy):

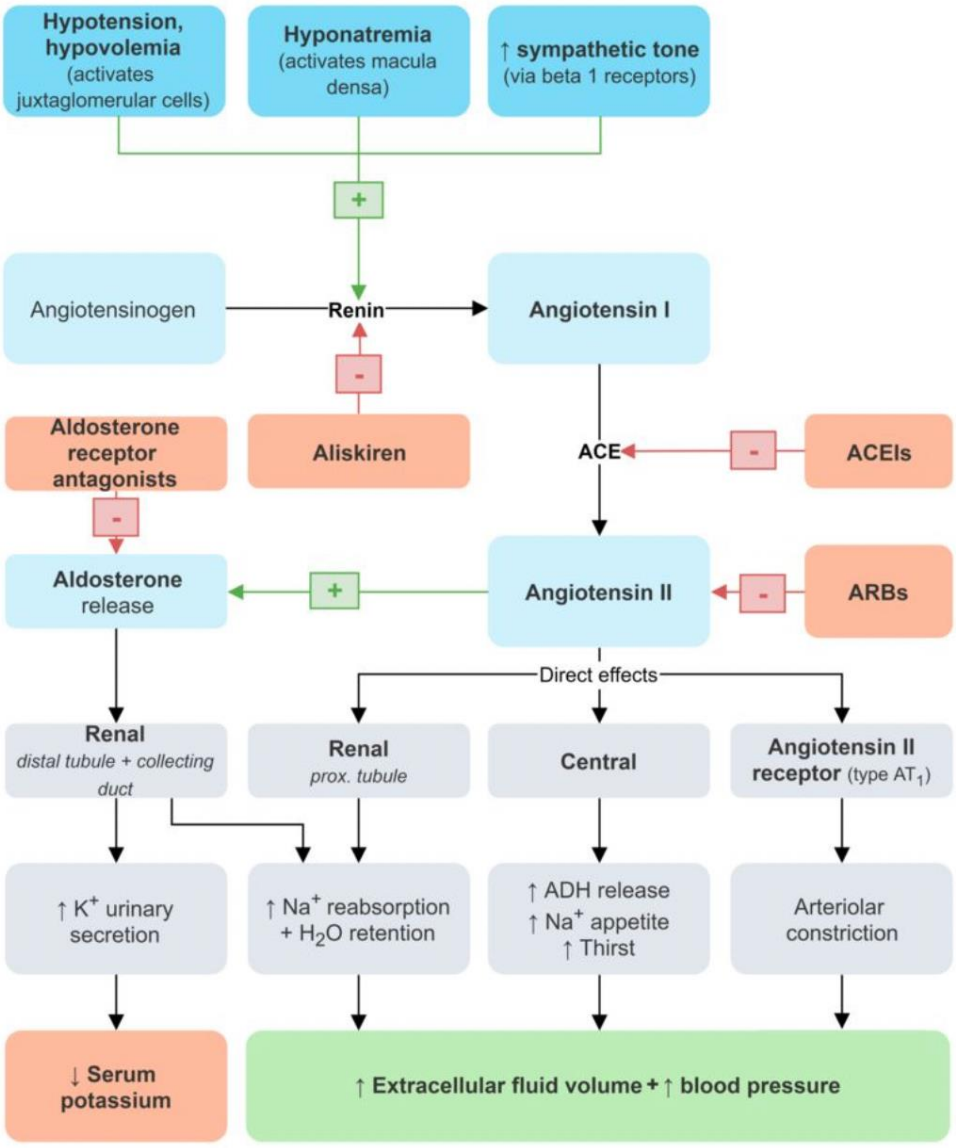
- **Increased LENGTH of cardiac muscle fibre:** in VOLUME overload.
- **Adaptive (Beneficial) effect:** It ↑ CO (according to Starling's law).
- **Maladaptive (Adverse) effect:** If it exceeds the limit (marked dilatation) → ↓ contraction.



5) Neurohormonal (NH) changes:

NH Change	Stimuli	Beneficial effects	Adverse effects	Potential ttt
↑↑ RAAS	• ↓ CO/BP • ↓ Renal blood flow • ↑ SNS	• VC & Na retention→ Maintenance of: BP & perfusion of vital organs	• VC → ↑Afterload • Na retention → • ↑ Preload • Pathologic LV remodeling (Apoptosis & • Hypertrophy)	• RAAS blockers (ACE-I, ARB)
↑↑ SNS	• ↓ CO/BP	• ↑ Contractility (SV) and ↑ HR → ↑ CO	• VC → Afterload • Ischemia • Arrhythmias • Pathologic LV • remodeling • (Apoptosis & • Hypertrophy)	• Beta-blockers
↑↑ Na & H2O retention	• ↑ RAAS • ↑ ADH • ↓ Renal blood flow	• ↑Preload → ↑ SV → ↑ CO	• ↑ Preload → Congestion (Pulmonary & Systemic)	• RAAS blockers (ACE-I, ARB) • Diuretics
↑↑ Natriuretic Peptide	• Volume expansion • Myocyte stretch	• ↓ RAAS & ↓ SNS • VD (Pulm & Syst) • Natriuresis • ↓ Endothelin • ↑ GFR	-----	• Natriuretic Peptides
↑↑ ADH	• ++ Baroreceptors in carotid sinus and aortic arch by the low CO	• H2O retention →↑ Preload → ↑ SV → ↑ CO	• ↑ Preload → Congestion (Pulmonary & Systemic) • Hyponatremia (Dilutional)	• ↓ Fluid intake • Tolvaptan (V2RA)
↑↑ Endothelin	• ↑ RAAS (↑ AgII) • ↑ SNS • ↑ ADH • Hypoxia	-----	• Pathologic LV remodeling (Apoptosis & Hypertrophy)	• Endothelin RAS (Speculative)


Loss of myocytes, arterial hypertension, aortic sclerosis/insufficiency, cardiac arrhythmias
↓ Cardiac output
↓ Blood pressure
↓ Blood pressure
↓ Blood volume
↑ ADH
Baroreceptors: ↓ blood pressure
↑ Sympathetic nervous system
no
Is cardiac output normal ?
yes
Temporarily compensated
↑ Heart rate
↑ Increased Contractility
β1
↓ Renal perfusion
↑ Angiotensin II
↑ Aldosterone
↑ Preload
↑ Afterload
V1
α1
AT II
Vasocon-striction
↑ Increased Stroke volume
↑ BNP
Na+ retention
H2O retention
Cardiac remodelling


Hypotension, hypovolemia (activates juxtaglomerular cells)
Hyponatremia (activates macula densa)
↑ sympathetic tone (via beta 1 receptors)
+
Angiotensinogen
Renin
Angiotensin I
-
Aldosterone receptor antagonists
Aliskiren
ACE
-
ACEIs
-
Aldosterone release
+
Angiotensin II
-
ARBs
Direct effects
Renal distal tubule + collecting duct
Renal prox. tubule
Central
Angiotensin II receptor (type AT1)
↑ K+ urinary secretion
↑ Na+ reabsorption + H2O retention
↑ ADH release
↑ Na+ appetite
↑ Thirst
Arteriolar constriction
↓ Serum potassium
↑ Extracellular fluid volume + ↑ blood pressure

6) Asynchronus ventricular contraction:

✦ **A substantial proportion (= 30%) of HFrEF patients (esp. DCM) may exhibit:**

- Intraventricular conduction delay (BBB, esp. LBBB) → Asynchronus ventricular contraction; therefore, instead of beating simultaneously:
  - ✪ The 2 ventricles beat slightly out of phase.
  - ✪ Also: segmental asynchrony within the LV may occur.
- This asynchrony greatly reduces the efficiency of the ventricles in patients with Heart failure, whose hearts are already abnormal.

CLINICAL PICTURE:

1. Left Sided Heart Failure

<table>
<tr><td>Symptoms</td><td>1) Symptoms of low CO: forward failure.
2) Symptoms of pulmonary congestion: backward failure (esp. PND).
3) When RVF occurs: Symptoms of pulmonary congestion improve.</td></tr>
<tr><td>Signs</td><td>
1) GENERAL:
<table>
<tr><td>Respiration</td><td>◆ Tachypnea.</td></tr>
<tr><td>Pulse</td><td>◆ Tachycardia: except in patients on Digitalis or BB.
◆ Small volume: except in case of High CO.
◆ Pulsus alternans.</td></tr>
<tr><td>Skin</td><td>◆ Coldness & peripheral cyanosis.</td></tr>
<tr><td>Chest</td><td>◆ Pleural effusion & Bilateral basal crepitations.</td></tr>
</table>
2) CARDIAC:
<table>
<tr><td>Precordial exam.</td><td>◆ Signs of LV enlargement.</td></tr>
<tr><td>Auscultation</td><td>◆ Mitral area: S3 gallop (Flabby myocardium), Pansystolic murmer (Functional MR).
◆ Pulmonary area: Signs of Pulmonary Hypertension.</td></tr>
</table>
</td></tr>
<tr><td>Manifestation of cause</td><td>e.g. Typical chest pain in AMI.</td></tr>
</table>

2. Right Sided Heart Failure

<table>
<tr><td>Symptoms</td><td>1) Symptoms of low CO: forward failure.
2) Symptoms of systemic congestion: backward failure.</td></tr>
<tr><td>Signs</td><td>
1) GENERAL:
<table>
<tr><td>Pulse</td><td>◆ Tachycardia: except in patients on Digitalis or BB.
◆ Small volume: except in case of High CO.</td></tr>
<tr><td>Skin</td><td>◆ Coldness & peripheral cyanosis.</td></tr>
<tr><td>Chest</td><td>◆ Pleural effusion.</td></tr>
<tr><td>Prominent signs of systemic congestion</td><td>◆ Neck veins: Congested pulsating neck veins.
◆ Liver: Enlarged tender ± pulsating ± Hepato-jugular reflux.
◆ Lower Limbs: Edema & later on ascites.</td></tr>
<tr><td>Cardiac Cachexia</td><td>◆ ↓ caloric intake (A, N, V & ↓ absorption): due to GIT congestion.</td></tr>
</table>
2) CARDIAC:
<table>
<tr><td>Precordial exam.</td><td>◆ Signs of RV enlargement.</td></tr>
<tr><td>Auscultation</td><td>◆ Tricuspid area: S3 gallop (Flabby myocardium), Pansystolic murmer (Functional MR).</td></tr>
</table>
</td></tr>
<tr><td>Manifestation of cause</td><td>e.g. Chronic cough, Dyspnea & Wheezes in COPD.</td></tr>
</table>

CLASSIFICATION:

1) **LVF VS RVF** (also Biventricular HF) المكان
2) **ACUTE VS CHRONIC** (also Acute on Chronic HF) الزمان

	Acute HF	Chronic HF
	Findings	
LL edema	◆ Absent	◆ Present
Ventricular ++	◆ Absent	◆ Present

3) **HIGH CO VS LOW CO FAILURE:**

	High CO HF	Low CO HF
Cardiac function	✪ Normal	✪ Reduced
Problem	✪ High CO → yet, insufficient for the metabolic needs of tissues	✪ Low CO → insufficient for the metabolic needs of tissues
Incidence	✪ Less common	✪ More common
Etiology	✪ Some causes (Hyperdynamic circulation)	✪ Most causes

HYPERDYNAMIC CIRCULATION

- **PATHO-PHYSIOLOGY :**
  1. Bypass of arteriolar & capillary bed due to: ↓ PR (VD state) and/or
  2. ↑ Tissue needs due to: ↑ metabolism →↑O2 consumption

  ➲ These changes →↑ Co (due to ↑ Velocity of circ, ↑ VR, ↑ HR)
- **GENERAL CAUSES :**
  - **A** ⇒ **A**nemia, **A**V fistula, **A**nxiety, **A**R.
  - **P** ⇒ **P**yrexia, **P**regnancy, **P**aget's disease, **P**DA
  - **E** ⇒ **E**xercise, **E**rythroderma
  - **O**THERS ⇒ Liver cirrhosis, Hyperthyroidism, Beri Beri, Obesity, iatrogenic (VD drugs)

4) CLASSIFICATION ACCORDING TO LVEF:

HF with preserved ejection fraction (HFpEF)	◆ HF with reduced stroke volume, normal or reduced EDV, preserved LVEF (≥ 50%), and evidence of increased LV filling pressures, e.g., increased natriuretic peptides, hemodynamic measurements
HF with reduced ejection fraction (HFrEF)	◆ HF with reduced stroke volume and reduced LVEF (≤ 40%)
HF with improved ejection fraction (HFimpEF)	◆ Previous HFrEF, with a follow-up LVEF measurement > 40%
HF with mildly reduced ejection fraction (HFmrEF)	◆ HF with an LVEF 41-49% and evidence of increased LV filling pressures, e.g., increased natriuretic peptides, hemodynamic measurements

5) CLASSIFICATION ACCORDING TO THE PROGRESSION (STAGES) :

ACC/AHA stages of heart failure

Stage	Definition and criteria	
Stage A: at risk for HF	Asymptomatic	◆ No structural heart disease or abnormal biomarkers ◆ Risk factors for HF, e.g., hypertension, ASCVD, diabetes mellitus, family history of cardiomyopathy
Stage B: pre-HF	Asymptomatic	**Evidence of ≥ 1 of the following:** ◆ Structural heart disease (e.g., reduced ejection fraction, valvular heart disease, ventricular hypertrophy) ◆ Increased filling pressures ◆ Risk factors for HF plus 1 BNP or 1 cardiac troponins with no alternative diagnoses
Stage C: symptomatic HF	◆ Signs and/or symptoms of HF (current or previous) ◆ Structural heart disease	
Stage D: advanced HF	◆ Symptoms of HF that disrupt daily life, with frequent hospitalizations despite GDMT optimization	

Patients with stage C HF will always remain categorized as such, even if they become asymptomatic (i.e., NYHA class I) with treatment.

6) FUNCTIONAL CLASSIFICATION ACCORDING TO SEVERITY (NYHA)

Class I	◆ **No** limitations of physical activity. ◆ **No** symptoms of CHF.
Class II	◆ **Slight limitations** of moderate or prolonged physical activity (symptoms after climbing 2 flights of stairs or heavy lifting). ◆ Comfortable at rest.
Class III	◆ **Marked limitations** of physical activity (symptoms during daily activity like dressing, walking across rooms). ◆ Comfortable only at rest.
Class IV	◆ Confined to bed, discomfort during any form of physical activity. ◆ **Symptoms at rest.**

- **The NYHA classification system:**
  - Used to assess limitations in physical activity and symptoms of patients with symptomatic HF (i.e., ACC/AHA stages C and D).
  - It helps determine treatment eligibility and prognosis.

INVESTIGATIONS:

❶ ECG:

- Chamber enlargement.
- Cause of HF, e.g. CAD, Ppt. factor, e.g. Arrhythmias.

❷ IMAGING :

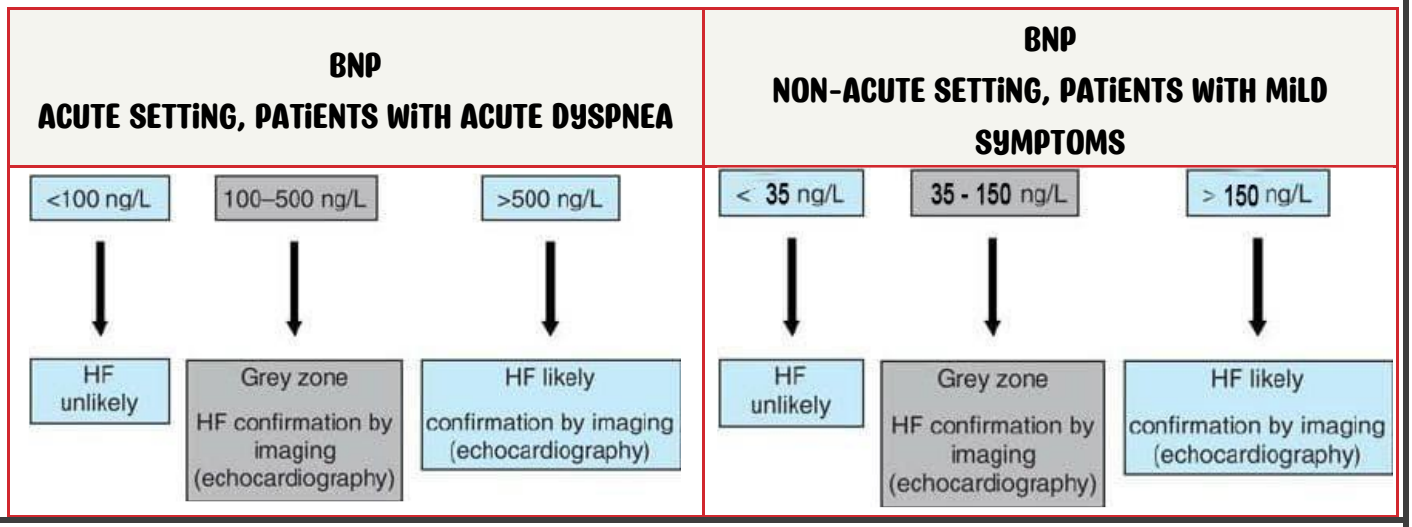
CXR	◆ Chamber enlargement. ◆ Pulmonary congestion in left-sided HF, or Pleural effusion.
ECHO	**(MOST IMPORTANT INVESTIGATION)** ◆ Chamber enlargement. ◆ Cause of HF, e.g. Valvular lesions. ◆ **SYSTOLIC FUNCTION:** Shortening Fraction (SF), Ejection Fraction (EF). ◆ **DIASTOLIC FUNCTION:** Mitral inflow signal [Early & Late filling (E & A wave)]. $EF = \frac{SV = EDV - ESV}{EDV}$
OTHERS	◆ For suspected CAD: ICA, SPECT, PET, CMR (Refer to IHD). ◆ For suspected CM or Myocarditis: CMR (Refer to CM)

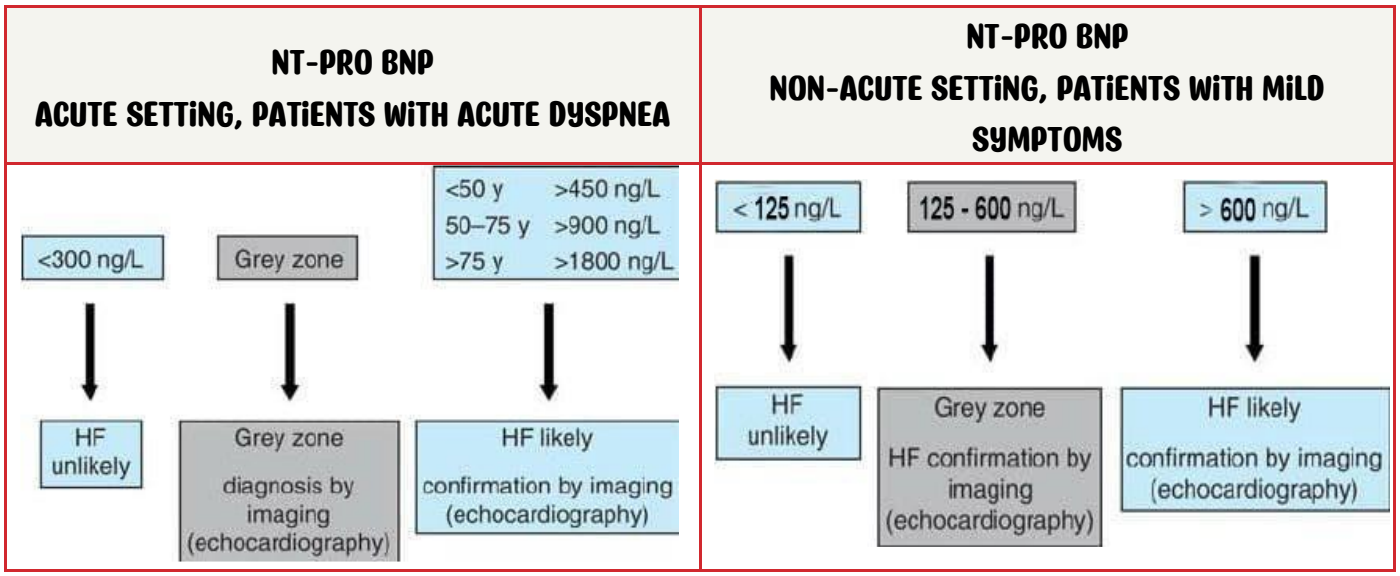
❸ Investigations for the cause & PPT factor:

- Cardiac enzymes (AMI)
- CBC (Anemia)
- Thyroid function (Thyrotoxicosis).

❹ Natriuretic Peptides (NP) (BNP & NT-proBNP):

- NT-proBNP is the precursor of BNP.
- ↑ levels help to differentiate HF from other causes of dyspnea.



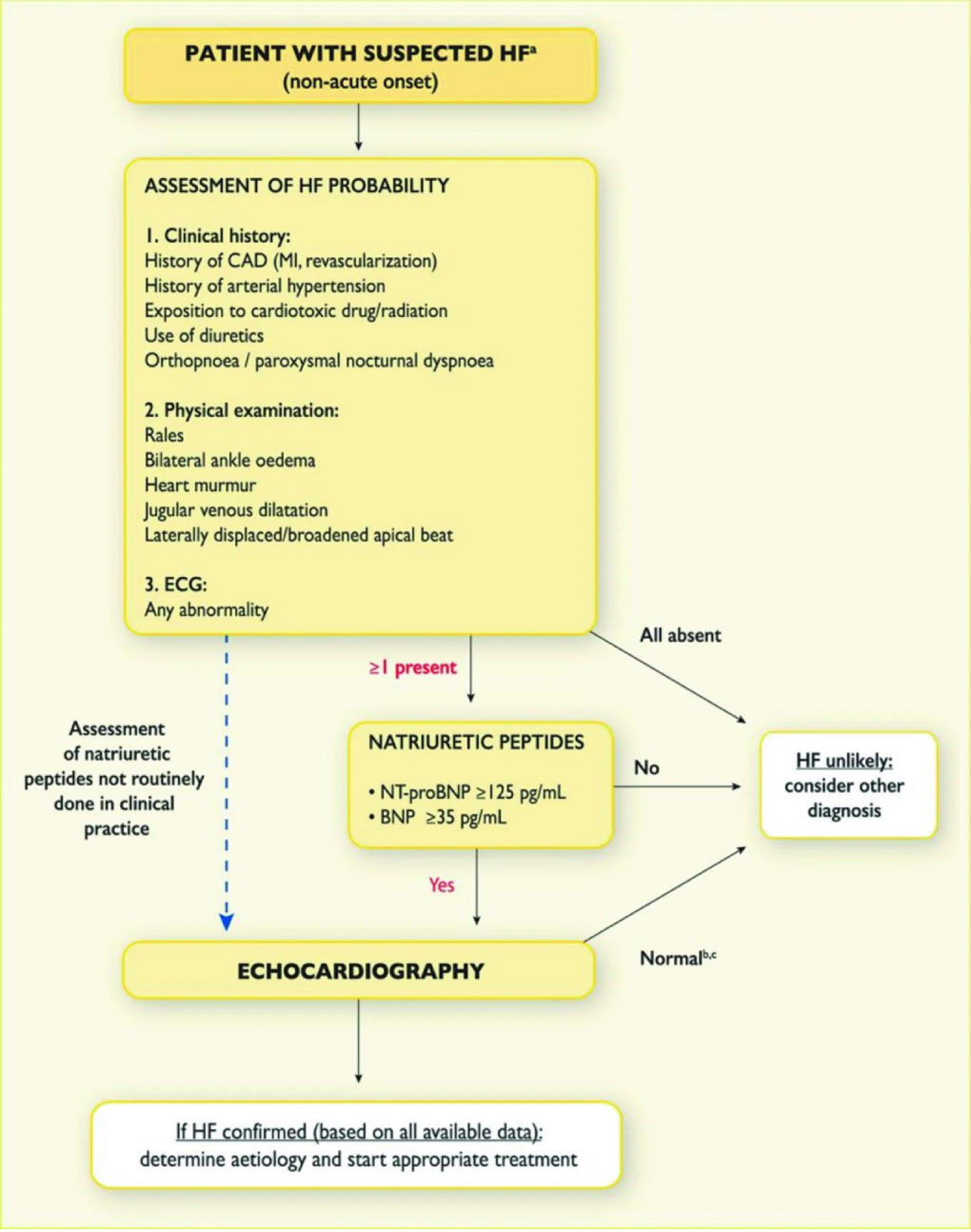


CLINICAL TIPS

- **In acute exacerbation of HF, always check:**
  - Cardiac enzymes to exclude AMI, CXR to exclude infection
- **In the work-up of new-onset HF, always try to identify:**
  - Potentially reversible causes, e.g. Arrhythmias (by ECG).

Cardiac causes of ↑ NP	Non-Cardiac causes of ↑ NP
◆ HF, LVH, ACS	◆ Advanced age
◆ CM (HCM, RCM)	◆ Stroke (Ischemic, SAH)
◆ Myocarditis	◆ Renal dysfunction
◆ Pulm embolism, Pulm HTN	◆ Liver cirrhosis with ascites
◆ Valvular or congenital H. dis.	◆ COPD
◆ Tacharrhythmias (A or V)	◆ Paraneoplastic syndrome
◆ DC cardioversion, ICD shock	◆ Severe infections & burtis
◆ Heart contusion	◆ Anemia
◆ Surgery involving the heart	◆ Metabolic crises (e.g. DKA)

Algorithm for the diagnosis or heart failure



TREATMENT:

1) Treatment of the cause, precipitating factor & any Comorbidity.
2) Treatment of the syndrome of Heart Failure.
3) Treatment of Refractory Heart Failure.
4) Treatment of Acute Heart Failure (Acute Pulmonary edema).

A) Treatment of the syndrome of Heart Failure

- **GENERAL PLAN :**

❶ Decreasing cardiac load:

A) REST: Physical & emotional.

B) REDUCTION OF PRELOAD:

- **D**iet control, especially salt restriction.
- **D**iuretics.
- Vaso **D**ilators (Venous).

C) REDUCTION OF AFTERLOAD: Vasodilators (Arterial).

❷ Decreasing Pathologic LV remodeling:

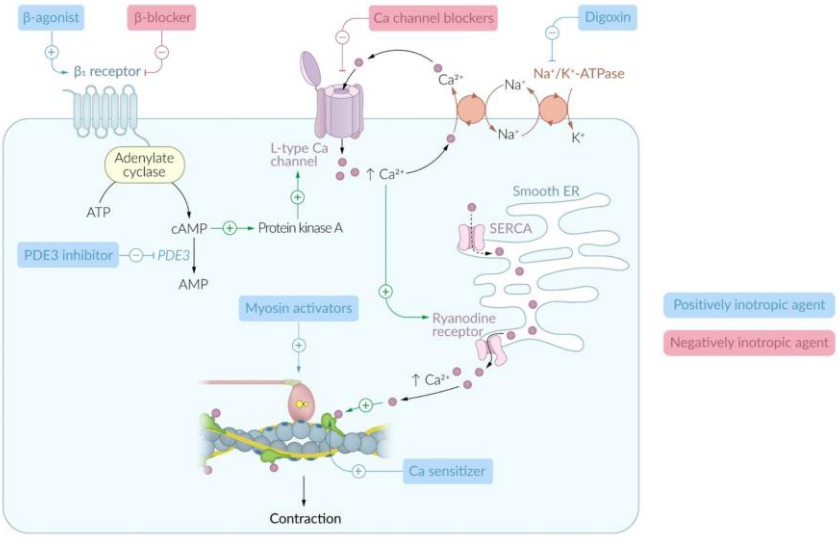
RAAS blockers (ACE-I gr ARB), B-blockers.

❸ Increasing myocardial contractility:

A) DIGITALIS: (For Chronic HF & Acute HF when associated é AF).

B) OTHER INOTROPIC AGENTS: (For Acute HF).

- Sympathomimetic **A**mines: Dopamine, Dobutamine.
- **P**hosphodiestrase inhibitors: Amrinone, Milrinone, Enoximone.
- **C**alcium sensitizer: Levosimendan.



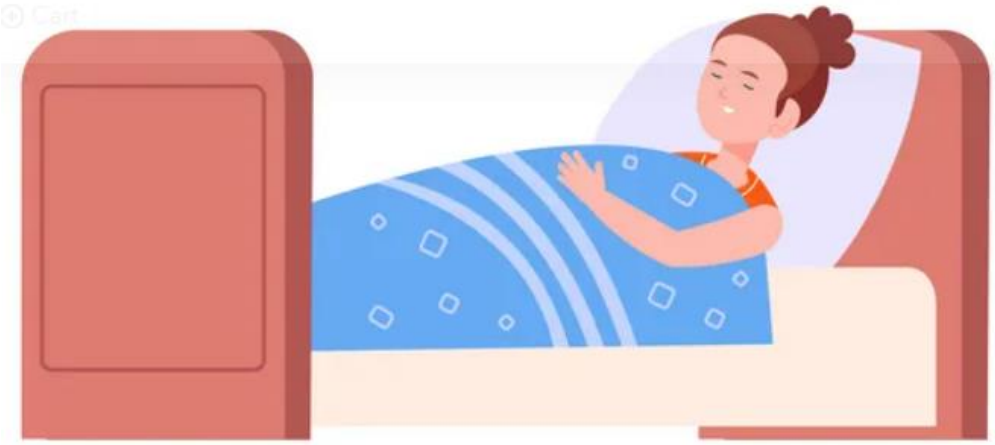
MOA of inotropic drugs

❹ Resynchronizing the ventricles: "CRT"
In patients with HF who have **Intraventricular conduction delay** or **bundle branch block.**
❺ Symptomatic treatment:
A) FOR HYPOXEMIA: oxygen administration. **B) FOR CARDIAC DYSPNEA:** aminophylline administration.
❻ Reducing Hospitalization.

- **RECENT MEDICATIONS :**
  1) **Ivabradine (Funny channel inhibitor)** → ↓ SAN rate → ↑ Ventricular filling.
  2) **Angiotensin Receptor – Neprilysin Inhibitor (ARNI):**
     ARB (Valsartan) → ↓ RAAS + Neprilysin Inhibitor (Sacubitril) → ↑ NP.
  3) **Sodium-GLucose co-Transporter-2 - Inhibitors (SGLT2 - 1):**
     - Dapagliflozin or Empagliflozin are recommended to ↓ the combined risk of HF
     - Hospitalization & CVS death in symptomatic patients é HFrEF, already on Guideline-Directed Medical Therapy (GDMT), regardless of presence of T2DM.

Rest

	PHYSICAL REST
Benefits	◆ Bed rest reduces the metabolic needs of the body & the cardiac load.
Position	◆ **Semi-sitting position** to decrease the venous return.
Complications of prolonged bed rest	◆ DVT & PULMONARY EMBOLISM, ◆ Pneumonia. ◆ Constipation & retention of urine. ◆ Muscle wasting & osteoporosis. ◆ Bed sores. ◆ Psychoneurosis.



	EMOTIONAL REST
Sedation	◆ **Diazepam** 2 – 5 mg tds orally.

Diet control

Salt restriction	◆ ↓ Blood volume & thus ↓ preload.
Fluid restriction	◆ Only needed in severe cases of HF.
Small frequent meals	◆ And encourage healthy eating patterns, e.g., DASH diet, Mediterranean diet.
Weight reduction	◆ In obese patients.
Smoking cessation	◆ And avoidance of alcohol and recreational drug use

Vasodilators

- **CLASSIFICATION:**

	Mechanism & significance:	Example:
Arteriodilators	◆ They dilate the artery leading to decrease ABP (**decrease afterload**)	◆ Hydralazine ◆ Diazoxide ◆ Minoxidil.
Venodilators	◆ They dilate veins leading to decrease venous return (**decrease preload**)	◆ Nitrates
Mixed	◆ They dilate both artery and vein (**decrease of afterload & preload**)	◆ Na nitroprusside ◆ Prazosin ◆ ACEI

	ACEiS
MOA	◆ Unless contraindicated or not tolerated. All patients with symptomatic or asymptomatic HF (EF<40%) should use ACEI • ↓Mortality & ↓morbidity. • Prevent further damage of ventricular fibers.
Members	◆ Captopril (6.25-25 mg/8 hrs) ◆ Enolopril-Lisnopril (once daily) ◆ Prindopril (2-4 mg/day)
Indications	◆ H.F ◆ Hypertension ◆ Diabetic microalbuminuria ◆ 2ry hyper aldosteronism
Side effects (CAPTOPRIL)	◆ **C**ough ◆ **A**ngioedema ◆ **P**otassium excess ◆ **T**aste changes ◆ **O**rthostatic hypotension ◆ **P**regnancy CI/**P**roteinuria ◆ **R**enal failure/ **R**ash ◆ **i**ndomethacin inhibition ◆ **L**eukopenia (rare)
CI (PARK)	◆ **P**regnancy ◆ **A**llergy/**A**ngioedema ◆ **R**enal artery stenosis/**R**enal failure ◆ **K**-hyperkalemia (potassium >5.5)

IN CASE OF CONTRAINDICATION TO ACE-i ARB ⇒ use an alternative good VD (RAS, ↑K, ↑Serum Creatinine >3mg/dl)

- Combination of: Hydralazine (Arteriolar dilator) & Nitrates (Veno dilator).
- H - ISDN = Hydralazine & IsoSorbide DiNitrate

IN CASE OF CONTRAINDICATION TO ACEIS (INTOLERANCE) ⇒ use ARB (esp. Candesartan).

- Intolerance: e.g. dry cough.

Diuretics

- They are very useful in the treatment of HF.
- They promote **loss of salt & water** → ↓ blood volume & 5 preload.

	1. Thiazide - Type diuretics
Action	◆ On the distal tubules, to ↓ reabsorption of Na, H2O, K, Cl.
Preparations	◆ **Hydrochlorothiazide:** 25-100 mg/day. ◆ **Bendroflumethiazide:** 2.5 - 10 mg/day.
Side effects	1. Hypo kalemia. 2. Hypo chloremic alkalosis. 3. Hypo natremia. 4. Hypo magnesemia. 5. Hyper Glycemia. 6. Hyper Lipidemia. 7. Hyper Uricemia 8. Hyper Calcemia.

	Thiazide - like diuretics
Action	◆ On the distal tubules.
Preparations	◆ **Indapamide:** 2.5-5 mg/day. ◆ **Metolazone:** 2.5 - 10 mg/day. ◆ **Chlorthalidone:** 12.5 - 25 mg/day (not commonly used in HF → a first-line diuretic in HTN).
Side effects	◆ Same as Thiazides.

	2. Loop Diuretics
	THEY ARE THE MOST POTENT DIURETICS IN HF.
Action	◆ On the ascending limb of loop of Henle, to ↓ reabsorption of: Na, H2O, K, Cl.
Preparations	◆ **Frusemide:** 40 - 240 mg/day. ◆ **Torsemide:** 10-20 mg/day. ◆ **Bumetanide:** 1 - 5 mg/day.
Side effects	◆ Same as Thiazides (but with hypocalcemia).

◆ **ALDOSTERONE ANTAGONISTS**
- **Spironolactone & eplerenone:** used as add-on therapy to ACE - 1 (Or ARB) in severe HF
- Take care of serum K.

	3. Potassium sparing diuretics
Action	◆ on the distal tubules to ↓ reabsorption of: Na, & H2O. ◆ BUT: They have the advantage of retaining K (they ↓ K secretion).
Uses	**THEY ARE WEAK DIURETICS** ◆ Used to potentiate the action of Thiazide or loop diuretics. ◆ Used to avoid potassium losing effect of Thiazide or loop diuretics.
Preparations	◆ **Spironolactone** (Ald. antagonist = MRA): 25 - 50 mg/day. ◆ **Eplerenone** (Ald. antagonist = MRA): 25 - 50 mg/day. ◆ **Triametrine**, **Ameloride**
Side effects	◆ Hyperkalemia & Metabolic acidosis. ◆ Gynecomastia (with Spironolactone with prolonged use).

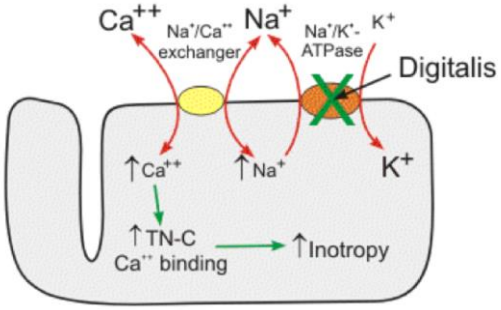
4. Other diuretics	
Natriuretic peptides (Recombinant by infusion)	Carbonic anhydrase inhibitors (Acetazolamide)
◆ Natriuresis . ◆ Aff. a VD, Eff. a VC → ↑ GFR ◆ Peripheral VD	◆ ↓ reabsorption of NaHCO3 in PCT. ◆ Not commonly used in HF.

Digitalis

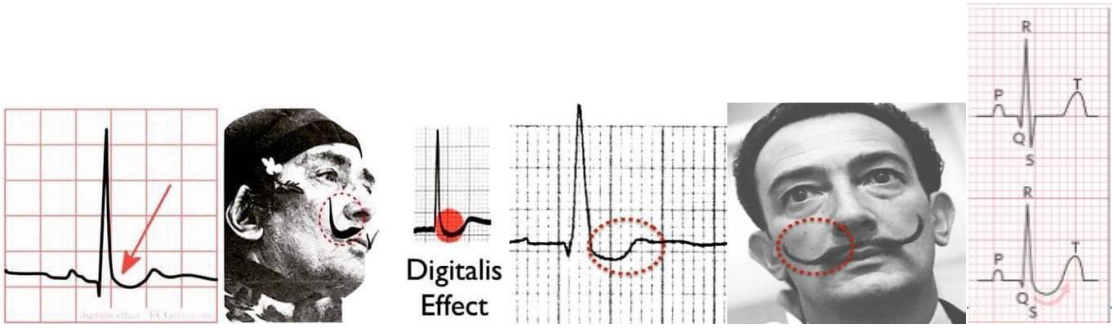
- It is no longer extensively used in the treatment of HF, although it is an effective + ve inotrope

Action

- **INCREASES THE MYOCARDIAL CONTRACTILITY:**
  - Competitive inhibition of NA-K- ATPase → ↑ intracellular Na.
  - Then, Na-Ca exchange occurs & this ↑↑ intracellular Ca.
  - High intracellular Ca promotes sliding of actin & myosin.
  - This leads to increased force of myocardial contractility.



- **IT SLOWS THE HEART RATE:**
  - Vagal stimulation.
  - Direct inhibition of the SAN.
- **IT INCREASES THE EXCITABILITY OF THE ATRIA & VENTRICLES:**
  - In digitalis toxicity, arrhythmias may occur.
- **IT INHIBITS THE CONDUCTION OF THE AVN:**
  - In digitalis toxicity, AV block may occur.
  - Digitalis can be used to protect the ventricle in atrial arrhythmias.
- **ON ECG:**
  - **DIGITALIS EFFECT:** Sagging depression of ST segment, Flat or inverted T wave.
  - **DIGITALIS TOXICITY:** Different types of arrhythmias.



QRS - ST morphology is described as: Sagging or "Salvador Dali's Moustache"

Indications	◆ Systolic Heart Failure. ◆ Rapid atrial arrhythmias: AF, Atrial flutter, SVT.
C/I	◆ **ABSOLUTE CONTRAINDICATIONS:** • Digitals toxicity. • Ventricular tachycardia (Digitalis may change it to fatal VF). ◆ **RELATIVE CONTRAINDICATIONS: "BETTER AVOIDED"** • Incomplete heart block. • Nodal rhythm. • Hypertrophic cardiomyopathy (HCM). • Hypokalemia

DIGITALIS TOXICITY

◆ **WHY DOES A PATIENT ON DIGITALIS DEVELOP DIGITALIS TOXICITY?**

Risk factor	Effect
↑ Ca	◆ SYNERGY: ↑ intracellular Ca already exists
↓ k	◆ Unopposed action of digitalis
↓ Mg	◆ ↑ digitalis uptake by the myocardium
Organ insufficiency	◆ Hepa **T** ic (with digi **T** oxin) ◆ Renal (with digioxin)
Elderly	◆ ↓ renal function or polypharmacy
Discontinuation of digitalis	
Medications: ◆ Amiodarone ◆ Benodiazepines ◆ Cyclosporine, Ca ◆ Diuretics (↓ K), Diltiazem	◆ ↑ digitalis serum levels and / or action

CLINICAL PICTURE:

GIT	◆ **Anorexia:** usually the first symptom. ◆ Nausea, vomiting, Abdominal pain.
CVS	**"DIFFERENT TYPES OF ARRHYTHMIAS & HEART BLOCK"** ◆ **Premature beats:** esp. occurring in bigemini or trigemini. ◆ **Paroxysmal atrial tachycardia:** with heart block. ◆ **Paroxysmal ventricular tachycardia:** most serious.
Neurological	◆ **Mental disturbances**, e.g. Psychosis. ◆ **Pain:** Headache & Neuralgias. ◆ **Vision:** Blurring of vision & Coloured vision (yellow or green).

TREATMENT:

- **Stop digitalis.**
- **Correct hypokalemia, if present:**
  - ✓ Stop drugs causing hypokalemia: e.g. loop diuretics.
  - ✓ Give potassium: Orally or IV.
- **Digitalis specific antibodies:** esp. in life-threatening overdose.
- **TTT of the manifestations of digitalis toxicity:**
  - ✓ **For vomiting:** Anti-emetic drugs, e.g. metoclopramide.
  - ✓ **For arrhythmias:**
    1. Anti-arrhythmic Drugs: especially Lidocaine & Epanutin.
    2. Atropine: for heart block & bradycardia.
    3. DC:
       - ◆ Avoided because it may induce more serious arrhythmias.
       - ◆ Allowed ONLY in case of the fatal arrhythmia: VF.

Beta Blockers

Types	◆ 2nd generation B1 blockers (e.g. Bisoprolol, Metoprolol), or: ◆ 3rd generation B1 blockers (e.g. Carvedilol).
Dose	◆ **Gradually increasing doses :** • Start by extremely low doses (1/8 of the target dose). • Gradually ↑ the dose every 1-2 weeks to reach the target dose. • Target dose: 25 - 50 mg of carvedilol daily.
Indications	◆ Chronic HF. ◆ Moderate HF (NYHA classes II & III).
C/I	◆ Acute HF. ◆ Severe HF (NYHA class IV).

BETA-BLOCKERS iN HF

FROM CONTRAiNDiCATiON TO iNDiCATiON

- In the past: they were contraindicated in HF due to their -ve inotropic effect.
- Now: they are used to treat HF because they were found to:
  - Decrease Pathological LV remodeling induced by catecholamines in HF.
  - Improve the prognosis.
- Medicine is Dynamic …
- The knowledge of Superior Doctors should always be up-to-date .

Cardiac Resynchronization Therapy (CRT)

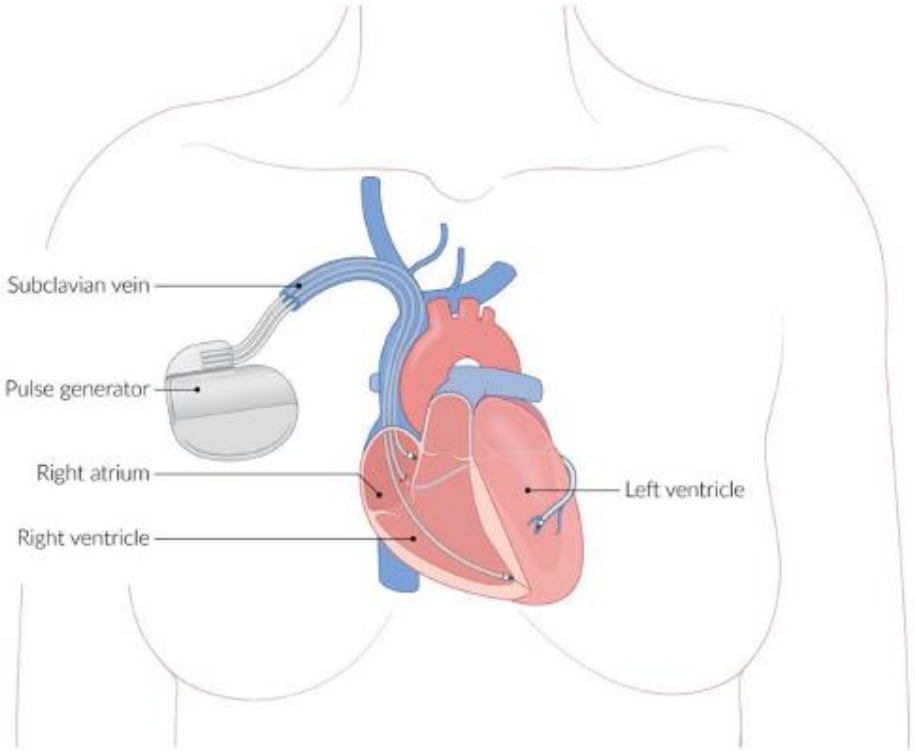
- **iNDiCATiONS OF CRT iN HF**

1) HErEF in Sinus rhythm é LVEF ≤ 35% & severe symptoms despite:

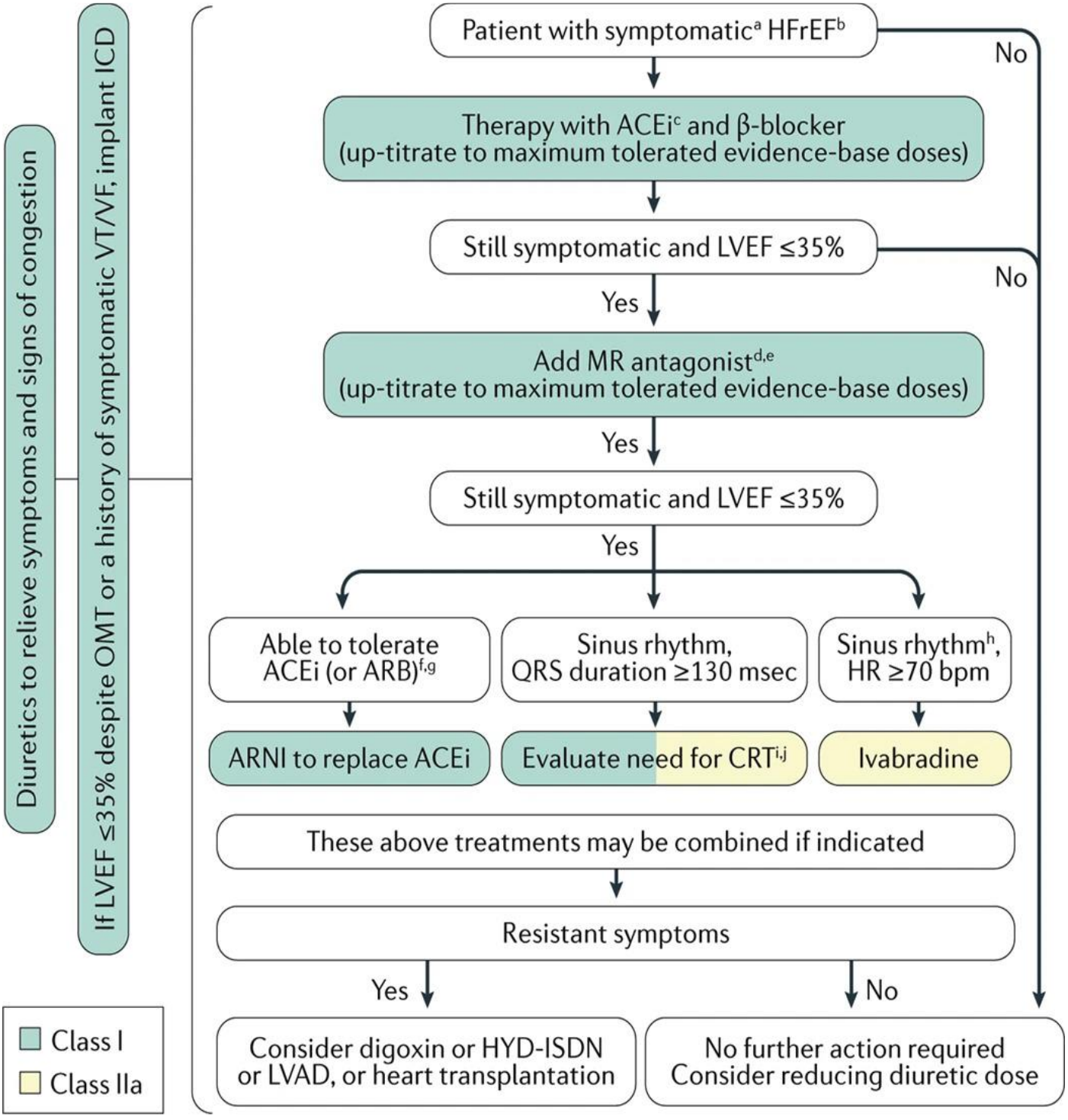
- OMT for at least 3 months (or at least 40 days after AMI).
- Correction of any reversible cause for HF e.g. myocardial ischemia.

Plus

2) QRS duration **≥130 sec,** QRS morphology: **LBBB,** NYHA class II to IV.



HFrEF TREATMENT STRATEGY



HFpEF TREATMENT STRATEGY

- In contrast to HFrEF, there is limited clinical trial evidence guiding the treatment of HFPEF.
- There is no agreed specific strategy for the treatment of HFPEF.
- Treatment is largely directed toward: cause (e.g. HTN) & symptoms (e.g. Oedema).

- **LINES OF TREATMENT:**

❶ Lifestyle Management:

- **Exercise training:** improves exercise capacity & quality of life in HFPEF.
- **Diet control:** salt restriction.
- **Weight loss.**

❷ Treatment of the cause & associated comorbidities :

- HYPERTENSION.
- Myocardial ischemia.
- Arrhythmias: e.g. AF.
- Diabetes, Dyslipidemia
- Obesity.
- COPD.

❸ Conditions To Avoid:

- **Common Precipitating factors of HF:** e.g. Abrupt severe elevations in systemic BP, Arrhythmias.
- **Excessive preload reduction:** Diuretics or venodilators (e.g. Nitrates) must be used é caution.

❹ Pharmacologic Therapy:

- **First-line agents**
  - **SGLT2i** for all patients: e.g., dapagliflozin or empagliflozin
  - **Loop diuretic** for patients with congestion: e.g., furosemide or torsemide

- **Other agents may be considered, e.g.:**
  - **MRA** For patients with clear evidence of HFpEF (including ↑ BNP) who can be carefully monitored for changes in serum K & renal functions.
  - **ARNI or ARB**

B) Treatment of refractory Heart Failure

DEFINITION: (STAGE D = ADVANCED HF):

- Persistence of symptoms of HF (NYHA class III or IV) or Repeated exacerbations of HF despite: Optimal treatment" with drugs of proven efficacy (GDMT).
- **Requires:** Advanced therapies (Consideration for Mechanical circulatory support or transplant).

ETIOLOGY:

1) Presence of a Mechanical factor hindering the cardiac function: e.g.

- Severe valvular disease.
- Pericardial effusion or constrictive pericarditis.

2) Persistence of the CAUSE: e.g. Uncontrolled Hypertension.

3) Presence of a precipitating factor: e.g. Chest infection.

4) Improper management: e.g. Inadequate rest, Inadequate salt restriction & digitalis.

5) End-stage HI: e.g. Advanced DCM.

TREATMENT:

1) Removal of the Mechanical factor: e.g. surgical ttt of valvular disease.

2) TTT of the CAUSE: e.g. proper control of Hypertension.

3) Removal of the precipitating factor: e.g. proper antibiotics for chest infection.

4) Proper management: Adequate rest, Adequate salt restriction, Adequate digitalis.

5) End-stage HR:

A) **DIURETICS:** combination e.g. Loop diuretic & K-sparing diuretic.

B) **VASODILATORS & INOTROPES:** Combination of IV vasodilators such as Na nitroprusside and a potent IV inotrope such as dopamine or dobutamine.

C) **INTERVENTIONAL HF THERAPY:**

- **Fluid removal:** Ultrafiltration.
- **Electrical therapy:** ICD (Refer to VT), CRT (Refer to HFrEF Treatment Strategy).
- **Mechanical Circulatory Support (MCS):**
  a) IABP (Intra Aortic Balloon Pump).
  b) CAFA (Continuous Aortic Flow Augmentation).
  c) LVAD (Left Ventricular Assist Device).
  d) BIVAD (Bi Ventricular Assist Device).
  e) Venoarterial extracorporeal membrane oxygenation.

D) **CARDIAC TRANSPLANTATION:** End-stage HF with severe symptoms.

- No remaining alternative treatment options.

ACUTE HEART FALIURE

DEFINITIONS:

Acute heart failure	◆ Rapid onset of new or worsening signs and symptoms of heart failure
Acute decompensated heart failure (ADHF)	◆ Acute heart failure due to decompensation of preexisting disease/cardiomyopathy (most common)
De novo heart failure	◆ Acute heart failure occurring for the first time in a patient without known cardiomyopathy (~15% of cases)

ETIOLOGY:

Type of acute heart failure	Underlying etiology
De novo heart failure	◆ **Acute myocardial dysfunction** 1. Cardiac ischemia due to acute coronary syndrome (ACS) → **Most common cause.** 2. Myocarditis 3. Drug-induced cardiomyopathy 4. Peripartum cardiomyopathy 5. Thyroid storm 6. Tachycardia-induced cardiomyopathy 7. Takotsubo cardiomyopathy ◆ **Acquired valvular pathology** • Acute mitral regurgitation after ACS • Bacterial endocarditis • Nonbacterial thrombotic endocarditis ◆ **Extracardiac pathologies that affect left ventricular output** • Pulmonary embolus • Pericardial effusion causing tamponade • Aortic dissection
ADHF	◆ Uncontrolled and/or refractory hypertension ◆ New/worsening cardiac ischemia ◆ Arrhythmias (e.g., atrial fibrillation with RVR, complete heart block) ◆ Serious infection/sepsis (e.g., pneumonia)

	◆ **Drugs:** • Nonadherence to heart failure medication • NSAID use • Drugs with negative inotropic properties (e.g., nondihydropyridine CCBs) • Starting or uptitrating beta blockers • Thiazolidinediones • Recreational substance use, e.g., cocaine, alcohol, methamphetamines ◆ Anemia ◆ Renal failure ◆ Volume overload, e.g., due to inappropriate fluid/salt intake or IV fluid therapy ◆ In 40–50% of cases, no trigger is found.

CLINICAL FEATURES:

✦ Clinical features of acute heart failure are commonly classified according to perfusion and the presence of congestion at rest.

	No evidence of congestion (~5% of patients)	Evidence of congestion (~95% of patients)
Adequate perfusion	Warm and dry	Warm and wet
Hypoperfusion	Cold and dry	Cold and wet

✦ **Congestion (most common) :**

Clinical features of left HF	Clinical features of right HF
◆ **Acute dyspnea** and **orthopnea** (i.e., worse when supine) ◆ Signs of increased work of breathing (**WOB**) ◆ **Cough** (occasionally with frothy, blood-tinged sputum) ◆ **Coarse crackles/rales** (and occasionally wheezing) on lung auscultation ◆ **S3 gallop** on heart auscultation ◆ **Severe cases:** central cyanosis	◆ Jugular venous distention ◆ Hepatojugular reflux ◆ Peripheral edema ◆ Ascites

- **Flash pulmonary edema:** Typically manifests with hypertension, pulmonary congestion, and minimal peripheral edema

✦ **Hypoperfusion**

- Weakness, fatigue, altered mental status
- **Signs of poor peripheral perfusion:** (e.g., cold, clammy skin, peripheral cyanosis, skin mottling).

✦ **Blood pressure:**

- May be low, normal, or elevated and should be interpreted in relation to the patient's baseline blood pressure

- The combined presence of jugular venous distention, S3 gallop, and lung crackles/rales makes a diagnosis of acute heart failure highly likely.
- **Assess for clinical features that are suggestive of hypoperfusion** (e.g., narrow pulse pressure, cool extremities, peripheral cyanosis, altered mental status, below baseline blood pressure) to identify patients with or at risk of cardiogenic shock.

DIAGNOSTICS

1. Laboratory studies

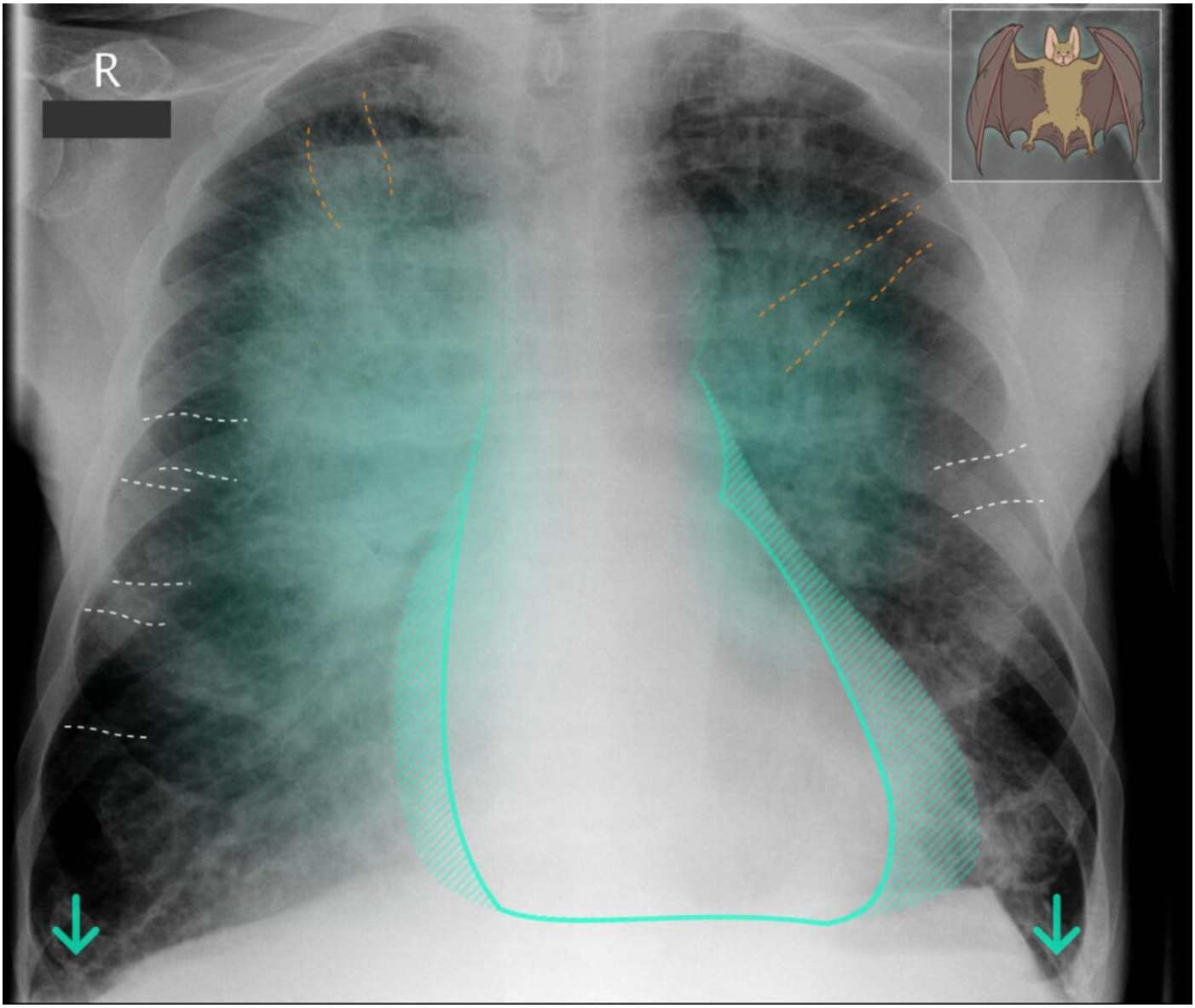
Natriuretic peptide levels in the diagnosis of heart failure		
	Heart failure unlikely	Heart failure likely
BNP (pg/mL)	< 100	> 500
NT-proBNP (pg/mL)	< 300	> 1000

2. Initial Imaging (Chest X-Ray)

✦ **Chest x-ray findings in cardiogenic pulmonary edema :**

1. **Cardiomegaly**
2. **Kerley B lines**
3. **Prominent pulmonary vessels** and perihilar alveolar edema (the hilar shadow has a butterfly or "bat wing" appearance)
4. Basilar interstitial edema
5. Bilateral pleural effusions

ABCDE: Alveolar edema (bat wings), Kerley **B** lines (interstitial edema), **C**ardiomegaly, **D**ilated prominent pulmonary vessels, and **E**ffusions



X-ray chest (AP view)
The cardiac silhouette is enlarged (hatched green overlay) and the perihilar air space opacities (green overlay) have a bat wing, or butterfly, configuration. Linear interstitial opacities representing Kerley A lines (orange dashed lines) radiate from the hila to the apices and Kerley B lines (white dashed lines) are seen in the lateral mid zones. The costophrenic angles are blunted (arrows) from bilateral pleural effusions.
These features are characteristically seen in cardiogenic pulmonary edema.

2. Investigations for the cause

- ECG & Cardiac enzymes (Tropinin) for AMI

MANAGEMENT

1. Initial management

- Perform a rapid ABCDE survey to assess hemodynamic stability.
- Identify and treat any acute underlying cause of AHF for all patients.

Hemodynamically stable patients	✦ **Clinical presentation:** SBP > 90 mm Hg AND no signs of end-organ hypoperfusion; respiratory distress can be present. ✦ **Management:** depends on the classification of AHF ➢ **No evidence of congestion (dry and warm):** • Optimize oral therapy. ➢ **Evidence of congestion (wet and warm):** • Start initial measures for respiratory support in AHF; (e.g., positioning, supplemental O2) as needed. • Start diuretic therapy for AHF if there is volume overload. • Consider vasodilators for AHF, e.g., nitrates. ◆ To remember the management of ADHF, think of "**LMNOP**": **L**oop diuretics (furosemide), **M**odify medications, **N**itrates, **O**xygen if hypoxic, **P**osition (with elevated upper body).
Hemodynamically unstable patients	✦ **Clinical presentation:** can vary ➢ **Cardiogenic shock:** SBP < 90 mm Hg OR signs of end-organ hypoperfusion ➢ **Hypertensive emergency:** hypertension (e.g., SBP > 180 mm Hg) PLUS flash pulmonary edema and hypoxemic respiratory failure ✦ **Management:** depends on the classification of AHF ➢ **Evidence of congestion with shock (wet and cold):** • Prioritize respiratory support for AHF. • Consider inotropic support (e.g., dobutamine, norepinephrine). ➢ **Shock** without evidence of congestion (dry and cold): Consider fluid challenge; add vasopressors and inotropes for shock refractory to fluids. ➢ **Hypertensive emergency** with flash pulmonary edema (wet and warm) • Begin NIPPV and vasodilators for AHF. • Identify and treat the underlying trigger.

2. Ongoing hospital management

Supportive care	✦ **Fluid restriction** does <u>not</u> reduce hospitalization or mortality rates in patients with HF. ✦ **Sodium restriction.** ✦ **Identify and treat comorbidities** (e.g., atrial fibrillation, pneumonia, COPD) and underlying triggers.
Optimizing chronic therapy for chronic HF	✦ **Administer beta blockers cautiously in beta-blocker-naive patients.** ◆ Start at a low dose. ◆ Administer only after stabilization (e.g., after volume status has been optimized and IV diuretics, vasodilators, and inotropic agents have been discontinued). ✦ Initiate, adjust, or continue medical treatment of heart failure as needed. ✦ Optimize blood pressure control.

✦ For patients not previously on beta blockers, use cautiously and only once the patient has been stabilized.

VALVULAR HEART DISEASES

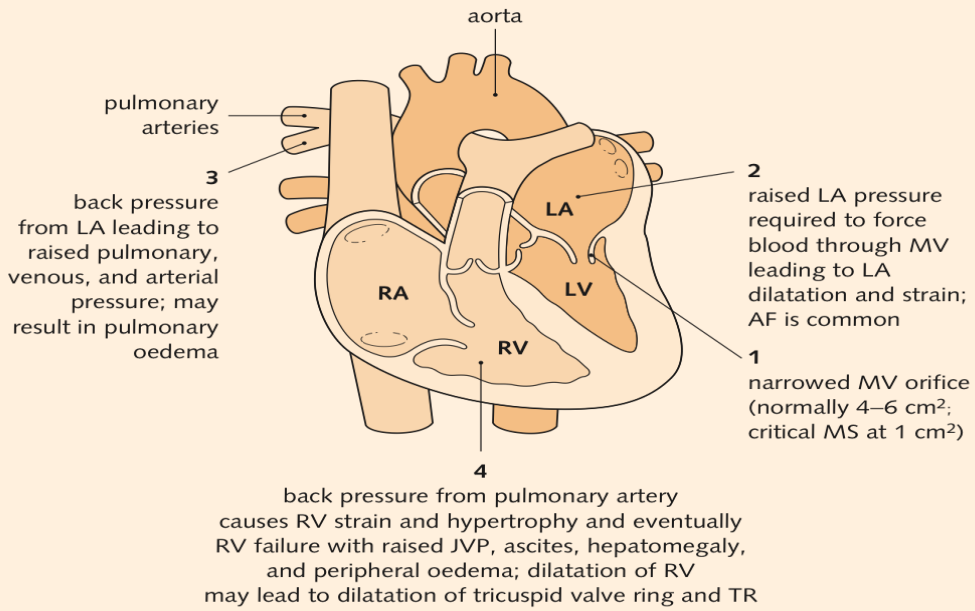
MITRAL STENOSIS

ETIOLOGY :

Organic stenosis	◆ **Rheumatic Fever:** most common cause. MCQ ◆ Calcification of the mitral valve annulus ◆ Autoimmune diseases: systemic lupus erythematosus, rheumatoid arthritis ◆ Congenital ◆ Some conditions may mimic mitral stenosis: bacterial endocarditis of the mitral valve with large vegetation, left atrial myxoma ◆ Radiation-associated valve disease, including MS, is increasingly recognized as late manifestation in survivors of Hodgkin lymphoma that is associated with thickening and ibrosis of the aortic-mitral curtain
Relative (functional) stenosis	◆ **INCREASED BLOOD FLOW** ⇒ through the mitral valve. ◆ **CAREY-COOMBS MURMUR** ⇒ in acute rheumatic valvulitis. ◆ **AUSTIN FLINT MURMUR** ⇒ in severe aortic regurge.

PATHOPHYSIOLOGY :

IN mild cases	◆ The blood flow through the mitral valve remains **normal.** ◆ **No** symptoms occur.
IN severe cases	**(VALVE AREA LESS THAN 2 CM):** ◆ The blood flow through the mitral valve is **decreased.** ◆ Blood stagnates in pulmonary veins **(pulmonary congestion).**
Later on	◆ Vasoconstriction of pulmonary arterioles occurs to ↓pulmonary congestion, but this will lead to **pulmonary hypertension.**
Finally	◆ **RV enlargement** & then **RVF** will occur secondary to pulmonary HTN.



STAGES :

Stage	Pathology	Functional
Stage I	Narrowing of mitral valve	Complete compensation (asymptomatic)
Stage II	Elevated pulmonary **venous** pressure.	Pulmonary congestion
Stage III	Elevated pulmonary **arterial** pressure.	PH + RVH
Stage IV	Systemic venous congestion	Right sided failure

CLASSIFICATION:

AMERICAN COLLEGE OF CARDIOLOGY/ AMERICAN HEART ASSOCIATION STAGE OF MITRAL STENOSIS

Stage	Definition	Symptoms	Mitral valve area	Associated findings
A	At risk for MS	None	Normal	None
B	Progressive MS		> 1.5 cm^2	Mild to moderate LA ++ Normal PASP
C	Asymptomatic severe MS		≤ 1.5 cm^2	Severe LA ++ PASP >50 mmhg
D	Symptomatic severe MS	Exretional dyspnea ↓ exercise tolerance	≤ 1.5 cm^2	severe LA ++ PASP >50 mmhg

CLINICAL PICTURE :

- There is a **latent period** of several years between the initial attack of rheumatic carditis & the development of manifestations of mitral stenosis

A. SYMPTOMS:

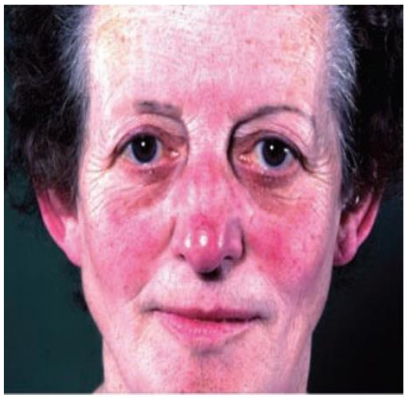
Stage I	• **No** symptoms
Stage II	• Symptoms of **pulmonary congestion** (but pulmonary edema is not common). • Symptoms of **low CO.**
Stage III	• Symptoms of pulmonary congestion: **improve.** • Symptoms of low CO: **increase.**
Stage IV	• Symptoms of **systemic congestion.**

Dyspnea is the most common symptom. MCQ

B. SIGNS

- **GENERAL:**

Stage I	• **No** Signs.
Stage II	• Signs of **pulmonary congestion.**
Stage III	• Signs of **low CO:** malar flush, giant a-wave.
Stage IV	• Signs of **systemic congestion.**



Malar flush

- **CARDIAC:**

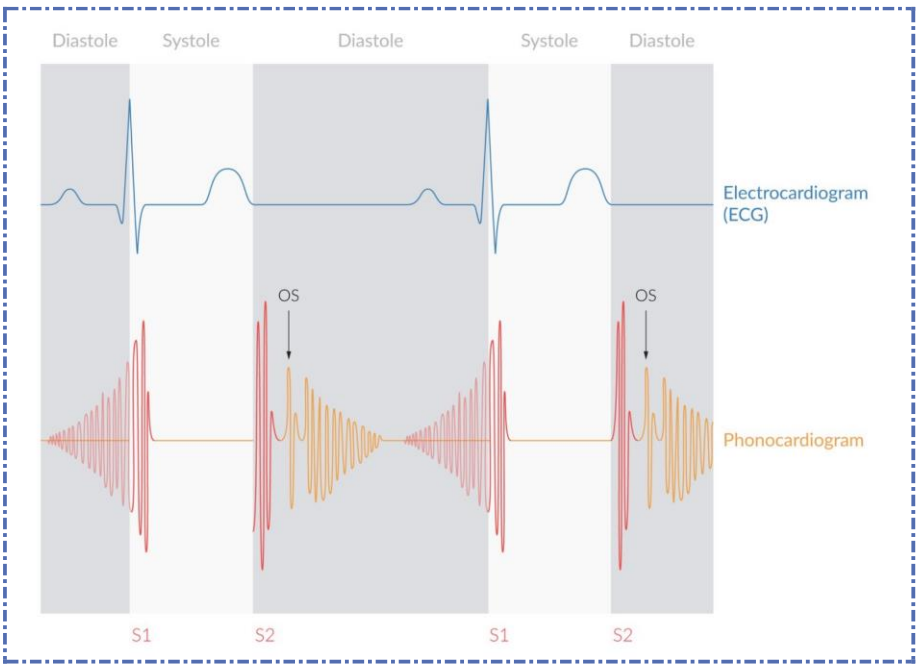
A. PRECORDIAL EXAMINATION:

Stage I & Stage II:	Stage III & IV:

• **Apex:** normal site & slapping character
• **Diastolic thrill:** ending in a palpable S1
	• **The previous findings + Signs of :**
	  • Pulmonary hypertension,
	  • Right ventricular enlargement.

B. AUSCULTATION:

Stage I & Stage II (Over the mitral area):	
Accentuated first heart sound:	◆ **DUE TO:** • Fibrosis of the mitral cusps. • Forcible closure of the mitral cusps because they are displaced downwards due to high LA pressure.
Mitral opening snap:	◆ It is **sharp & snapping** D2 opening of the rigid cusps of mitral valve. ◆ **IT IS HEARD IN THE EARLY DIASTOLE:** • Just after S2 (separated from it by the isometric relaxation phase). • Just before the murmur of mitral stenosis. ◆ Its presence denotes **non-calcificaton** of the mitral valve.
Murmur of mitral stenosis:	◆ **TIMING:** • Mid-diastolic presystolic with presystolic accentuation, • In AF, there is loss of presystolic accentuation . ◆ **CHARACTER:** rumbling. ◆ **SITE:** at or slightly inside the apex. ◆ **PROPAGATION:** not propagated. ◆ **POSITION:** • Best heard with the cone of the stethoscope. • In the **left lateral position.**



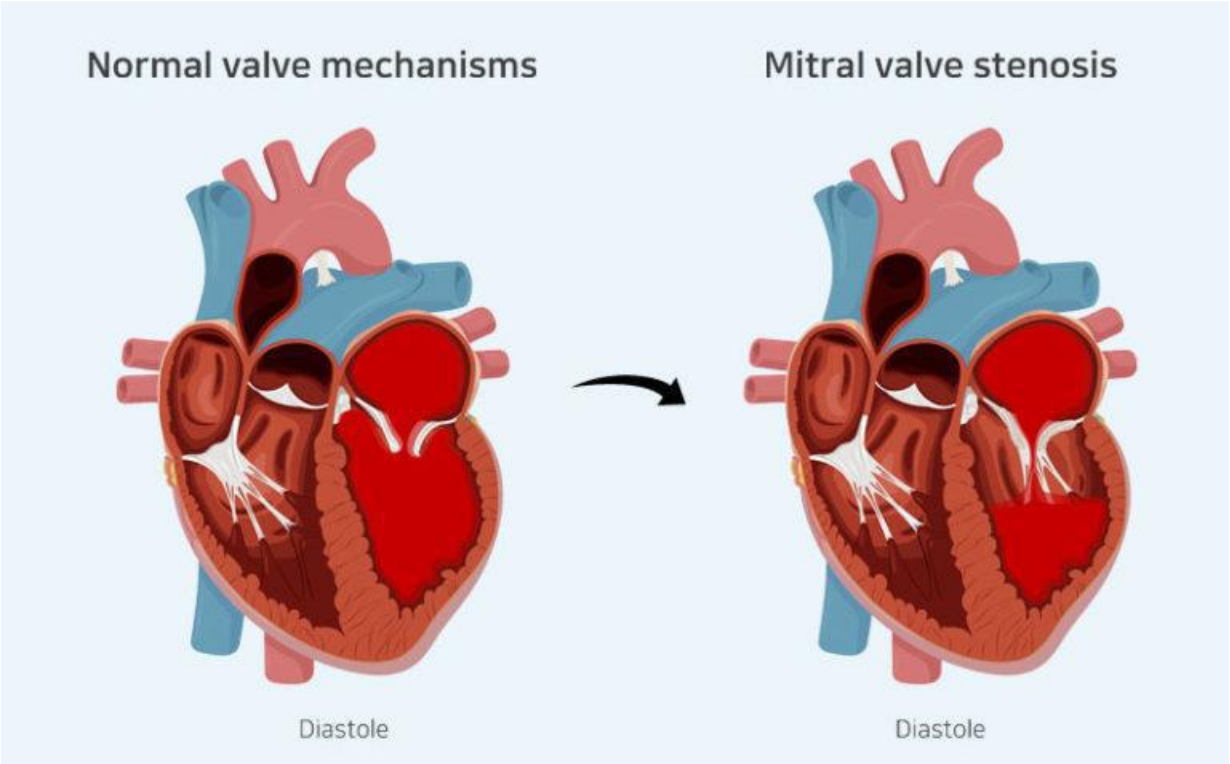
- **SILENT MITRAL STENOSIS: (MS WITH NO MURMUR), DUE TO:**
  - **High LV pressure:** e.g. associated LVF.
  - **Low LA pressure:** e.g.
    a. Severe pulmonary hypertension.
    b. RVF.
  - **In association with ASD.**

Stage III :	
The previous finding +	◆ **AUSCULTATORY FINDINGS OF PULMONARY HYPERTENSION:** • **Over the pulmonary area:** ☺ S_2 is accentuated. ☺ Systolic ejection click. ☺ Systolic ejection murmur. ☺ Soft early diastolic murmur: due to functional PR • **Over the tricuspid area:** S_4.

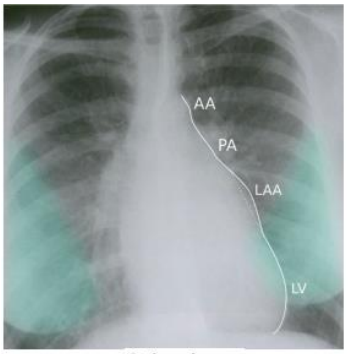
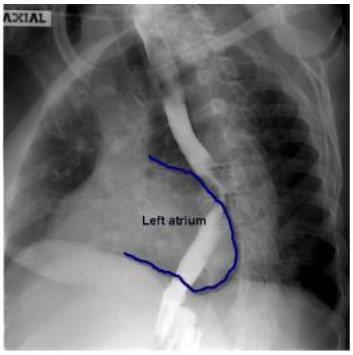
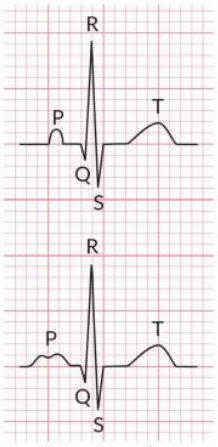
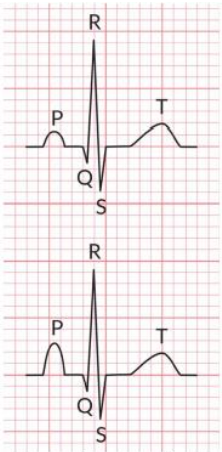
Stage IV :	
The previous finding +	◆ **AUSCULTATION OVER TRICUSPID AREA:** • Pan systolic murmur of functional tricuspid regurge. • RV gallop (S_3) due to RVF.

COMPLICATIONS :

In mitral valve :	◆ Rheumatic activity. ◆ Calcification. ◆ Infective endocarditis.
In left atrium:	◆ **ARRHYTHMIAS: especially AF.** ◆ **LA ENLARGEMENT: causing pressure symptoms:** • **ON ESOPHAGUS :** dysphagia. • **ON LEFT BRONCHUS :** dyspnea & cough. • **ON LEFT RECURRENT LARYNGEAL NERVE:** hoarseness of voice. ◆ **THROMBO-EMBOLIC COMPLICATIONS:** • **Systemic embolization:** cerebral, peripheral, renal. • **Ball & valve embolus:** ⇒ syncope & sudden death.
In right ventricle :	◆ RVF.
In left ventricle :	◆ No LVF in isolated mitral stenosis.
In the lung:	◆ Hemoptysis. ◆ Pulmonary infection. ◆ Pulmonary embolism (2ry to DVT). ◆ Pulmonary edema is not common.
Complications of surgery	



iNVESTiGATiONS :

CXR	◆ **Stage I:** no abnormality. ◆ **Stage II:** (PA view & lateral view with Barium) • LA enlargement. • Pulmonary congestion. ◆ **Stage III & IV:** • Right ventricular enlargement & RA enlargement. • Pulmonary hypertension. • May be calcified mitral valve.  PA view  lateral view
ECG	◆ LA enlargement (P mitrale : m shaped P wave), RVE in late stage. ◆ Pulmonary hypertension (P pulmonale: peaked P wave) ◆ Arrhythmias.  P mitrale  P pulmonale
Echo " most sensitive & specific"	◆ **Detects the severity of stenosis by measuring:** • The valve area. • The pressure gradient across the valve. ◆ **Detects chamber enlargement.**
Cardiac catheterization & angio-cardiography:	

WHEN DO WE SAY TIGHT MITRAL STENOSIS

- **TIGHT MS IS DIAGNOSED:**
  - **A. ACCORDING TO THE STAGE:**
    - ☺ Satge **II** with dyspnea more than grade **II,**
    - ☺ Stage III,
    - ☺ Stage IV.
  - **B. ACCORDING TO ECHOCARDIOGRAPHY:**
    - ☺ Valve area less than 1 cm .
- **TIGHT MS IS AN INDICATION FOR SURGERY .**

TREATMENT :

❶ Medical Treatment :	
Prophylaxis against :	◆ Rheumatic activity. ◆ Infective endocarditis (**uncommon**).
Curative:	1. **Diuretic:** To reduce symptoms of pulmonary congestion. 2. **Anticoagulation with a vitamin K antagonist (VKA) to target INR of 2.5 is indicated if any of the following are present:** • Atrial fibrillation • History of embolic disease • Intracardiac thrombus • Mechanical prosthetic mitral valve • First 3–6 months after bioprosthetic mitral valve implantation ◆ It is controversial whether anticoagulation is indicated for patients with MS who do not have one of the above features. 3. **Antibiotics:** for infective endocarditis. 4. **Prevent/treat atrial fibrillation:** • Digoxin. • b-blockers. • Antiarrhythmic agents. • Anticoagulation.

❷ Surgical Treatment :	
Indications :	◆ Tight mitral stenosis (**valve area is < 1 cm**). ◆ Marked symptoms not responding to adequate medical TTT. ◆ Embolization e no serious deterioration of patient condition . ◆ Pulmonary artery systolic pressure > 50 mmhg. ◆ New onset atrial fibrillation.
Operations types :	◆ **Percutaneous mitral valve balloon commissurotomy (PMBC):** • PMBC is the preferred intervention in most patients with severe MS. • A balloon catheter is advanced percutaneously through the mitral valve and inflated to break open commissural stenosis and increase the mitral valve area. • Contraindicated in presence of lt atrial thrombus or heavy deformed or calcific valve. ◆ **Surgery:** • Surgical interventions include open commissurotomy and mitral valve (mechanical or bioprosthetic) replacement. • **Indications:** for valve replacement 1. Calcification. 2. Associated mitral regurge. 3. Recurrent stenosis after commissurotomy.
Complications :	1. Embolization. 2. Arrhythmias. 3. Mitral incompetence. 4. Re-stenosis. 5. **Post - cardiotomy syndrome:** • Pleuro-pericarditis that may occur 10-15 days following the operation. • Autoimmune response to damaged cardiac tissue. 6. **Complications of artificial valves:** • Infective endocarditis. • Thrombo-embolism. • Mechanical dysfunction. • Hemolytic anemia.

MITRAL REGURGE

ETIOLOGY :

Organic	◆ **RHEUMATIC FEVER:** [MOST COMMON CAUSE] ◆ Congenital. ◆ Prolapse of the mitral valve (MVP). ◆ Papillary muscle dysfunction e.g. CAD (Ischemic MR). ◆ Infective endocartitis. ◆ **Iatrogenic:** following mitral commissurotomy.
Relative	◆ Dilatation of the mitral ring secondary to LV dilatation.

CLASSIFICATION: ACCORDING TO ONSET OF DISEASE:

Acute MR	◆ Acute dysfunction of the mitral valve leads to volume overload and symptoms of acute heart failure.
Chronic MR	◆ To preserve cardiac output, valve dysfunction is initially compensated for by cardiac remodeling. ◆ Over time, remodeling affects LVEF, leading to heart failure.

PATHOPHYSIOLOGY :

Acute MR	◆ ↑ left atrial volume with normal left atrial compliance and ↑ LV end-diastolic volume → rapid ↑ in LA and pulmonary pressures → pulmonary venous congestion → pulmonary edema.
Chronic (compensated) MR	◆ progressive dilation of the LV (via eccentric hypertrophy) → ↑ volume capacity of the LV (preload and afterload return to normal values) → ↑ end-diastolic volume → maintains ↑ stroke volume (normal EF)
Chronic (decompensated) MR	◆ progressive LV enlargement and myocardial dysfunction → ↓ stroke volume → ↑ end-systolic and end-diastolic volume → ↑ LV and LA pressure → pulmonary congestion, possible acute pulmonary edema, pulmonary hypertension, and right heart strain

CLINICAL PICTURE :

1. Chronic Mitral Regurgitation:

A. SYMPTOMS :

1. **NO SYMPTOMS:** in early cases.
2. **SYMPTOMS OF LOW CARDIAC OUTPUT:** in late cases.
3. **SYMPTOMS OF PULMONARY CONGESTION:** in late cases.
4. **PALPITATION.**

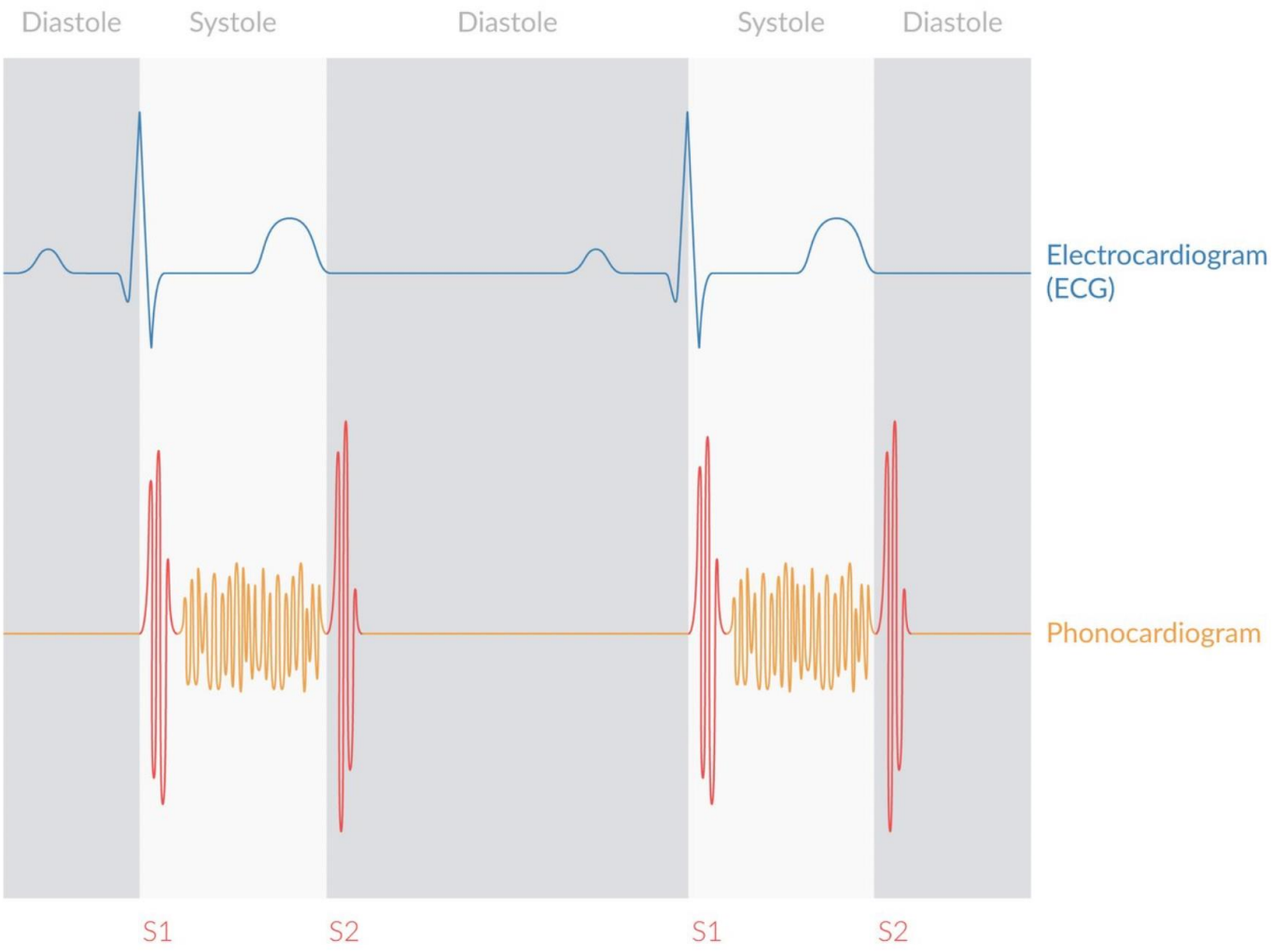
B. SIGNS :

- **GENERAL:**
  - **A. NO SIGNS:** in early cases.
  - **B. SIGNS OF LOW CARDIAC OUTPUT:** in late cases.
  - **C. SIGNS OF PULMONARY CONGESTION:** in late cases.

- **CARDIAC:**
  - **A. PRECORDIAL EXAMINATION:**
    - **SIGNS OF LV ENLARGEMENT:** with hyperdynamic apex
    - **SYSTOLIC THRILL:** over the apex.

  - **B. AUSCULTATION:**

First heart sound	◆ **Weak** (muffled) due to failure of proper mitral closure.
Third heart sound	◆ **Present** due to excessive flow of blood from LA to L
Murmur of mitral regurge :	◆ **TIMING:** pan systolic starting with S_1. ◆ **CHARACTER:** soft or harsh. ◆ **SITE:** over the apex. ◆ **PROPAGATION:** • To axilla. • To base of heart & medially in posterior leaflet disease. ◆ **POSITION:** • Best heard In the left lateral position.



2. Acute Mitral Regurgitation:

Symptoms & Signs	◆ Dyspnea ◆ Symptoms of left-sided heart failure ◆ Signs and symptoms of pulmonary edema (e.g., bibasilar, fine, late inspiratory crackles) ◆ Cardiogenic shock: poor peripheral perfusion, tachycardia, tachypnea, and hypotension ◆ Palpitations
Auscultation	◆ Soft, decrescendo murmur ◆ No murmur in severe regurgitation with LV systolic dysfunction or hypotension ◆ **Potentially:** S3 heart sound

COMPLICATIONS : Same complications of MS, but:

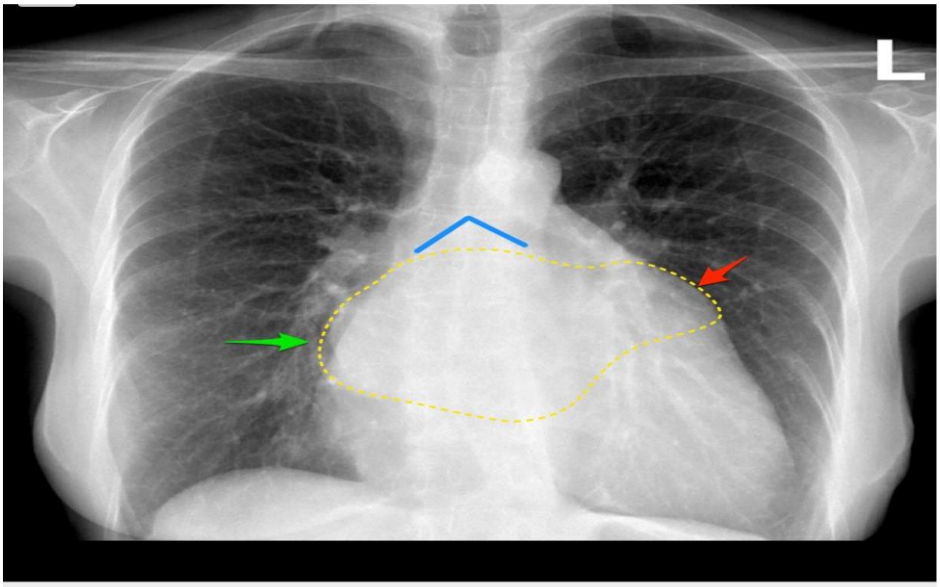
- Infective endocarditis: is **common.**
- Left ventricular failure: occurs.

CLASSIFICATION :

American Heart Association (AHA) Staging for MR

Stage	Extent of mitral regurgitation
A	◆ At risk of MR (Minimal regurgitation)
B	◆ Progressive MR (moderate regurgitation)
C	◆ **Sever asymptomatic MR.** ◆ **Primary MR is further divided into:** • C1: LVEF >60% and LVESD <40mm • C2: LVEF ≤60% and LVESD ≥40mm
D	◆ **Severe symptomatic MR.**

INVESTIGATIONS :

CXR	◆ **Early cases :** no abnormality. ◆ **Late cases :** • LA & LV enlargement. • Pulmonary congestion. ◆ **In double mitral stenosis :** • Calcified mitral valve.  The rough outline of the left atrium (yellow) can be inferred by the presence of a double border on the right (green arrow), splaying of the carina (blue) and prominence of the left atrial appendage (red arrow).
ECG	◆ LA enlargement (P mitrale : broad & bifid). ◆ LV enlargement .
Cardiac catheterization & angiocardiography	◆ Detects the **severity of mitral regurgitation.** ◆ Detects **chamber enlargement.** ◆ Detects the **cause:** e.g. CAD.

Echocardiography

- Detects the **severity of mitral regurgitation.**
- Detects **chamber enlargement.**
- Detects the **cause:** e.g. MVP.

Echocardiographic characteristics of primary MR

Parameter	Acute MR	Chronic MR
Valve movement or function	Abnormal	Abnormal
Aortic valve opening	Decreased	Decreased
Pulmonary vein flow	May be reversed	Generally normal
Left atrium	Normal	Dilated
Left ventricle size	Normal	Increased / remodeled
LVEF	Normal	• Compensated: normal or increased • Decompensated: decreased
Pulmonary artery pressure	Elevated	• Compensated: normal • Decompensated: elevated
Right ventricle ejection fraction	Normal	• Compensated: normal • Decompensated: reduced

TREATMENT :

1. Acute mitral regurgitation

General principles	• All patients with acute primary MR should undergo urgent surgical repair or valve replacement. • While awaiting surgery, any symptoms of heart failure should be managed with medical therapy (e.g., diuretics, nitrates, antihypertensive drugs)
Initial stabilization	• Usually only a temporizing measure while surgery is planned.
Surgery	• Indications: **Acute primary MR:** emergency/urgent surgery • Procedures: **Valve repair:** preferred option.

All patients with acute primary MR should undergo urgent surgical repair or valve replacement.

2. Chronic mitral regurgitation

Medical TTT	◆ As mitral stenosis.
Surgical TTT	◆ **Indications:** severe primary MR with any of the following • Asymptomatic patients with LV systolic dysfunction (LVEF ≤ 60% and/or LVESD ≥ 40 mm) • Symptomatic patients irrespective of LV systolic function ◆ **Procedures:** • **Repair of** mitral leaflets chordae tendinae papillary muscle, mitral annuloplasy for wide rings, or • **Replacement of mitral valve with:** Prosthetic valve, or Homograph.

MITRAL VALVE PROLAPSE

(BARLOW'S SYNDROME - FLOPPY MITRAL VALVE)

EPIDEMIOLOGY

- **Prevalence:** 2–3 % (one of the most common valvular abnormalities in the US).
- The most common cause of mitral regurgitation in developed countries.

ETIOLOGY:

Disease	Mechanism of regurgitation
Primary MVP	
Non-classic	• Isolated prolapse of the mitral leaflet (commonly P2 scallop) • Frequent chordal rupture • Mild annular enlargement
Fome Fruste Barlow disease	• Intermediate features
Classic (Barlow disease)	• Diffusely thickened, redundant mitral leaflets • Chordal elongation/rupture • Severe annular enlargement
Secondary MVP	
Associated with connective tissue disease	• **Associated with** Marfan and Ehlers-Danlos syndromes, osteogenesis imperfecta, and pseudoxanthoma elasticum. Macroscopical and microscopical appearance similar to primary Barlow disease. • Diffusely thickened, redundant mitral leaflets • Chordal elongation/rupture • Severe annular enlargement
Associated with congenital heart disease	• Most common congenital disease associations are **Ebstein's anomaly and atrial septal defect.** • Thickened, redundant mitral leaflets • Chordal elongation/rupture possible
Non-MVP flail mitral leaflet	
Acute myocardial ischemia	• Papillary muscle dysfunction with secondary prolapse/papillary muscle rupture

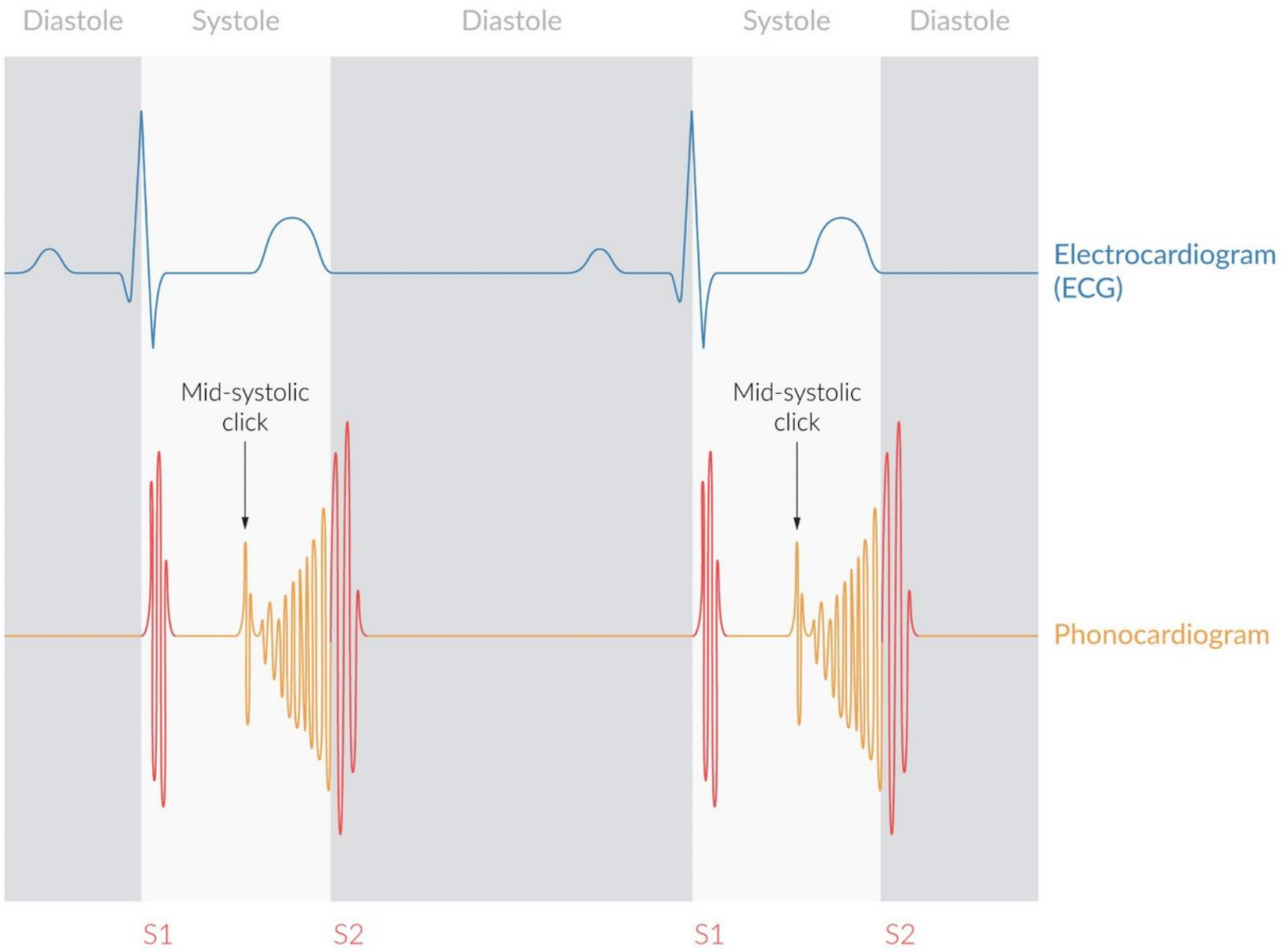
Acute rheumatic fever	◆ Chordal and leaflet destruction by acute inflammatory process
Endocarditis	◆ Chordal and leaflet destruction by infectious process; vegetations
Other	(Trauma, severe mitral annular calcification, hypertrophic cardiomyopathy) ◆ Ruptured chordae. ◆ No myxomatous changes of mitral valve leaflets

PATHOPHYSIOLOGY:

- The most common underlying pathology in the case of mitral valve prolapse is **myxomatous degeneration** (deposition of glycosaminoglycan such as dermatan sulfate) of the mitral valve due to a **primary disease** or **connective tissue disorder**
  - Long, floppy mitral valve leaflets with excessive valvular tissue → the mitral annulus becomes dilated and the chordae tendineae become elongated (and may rupture) → prolapse of one or both mitral valve leaflets into the left atrium during systole
  - The leaflets may also exhibit fibrous thickening at regions where they rub against each other.
- **Mitral valve prolapse sets into motion a vicious cycle of events.**
  - If prolapse happens without the rupture of chordae tendineae → mitral valve leaflets billow into the left atrium → mild to moderate mitral regurgitation
  - If the papillary muscles become severely ischemic and the chordae tendineae rupture → mitral valve leaflets flail about in the left atrium → severe mitral regurgitation

CLINICAL FEATURES:

- Most patients are **asymptomatic.**
- Rarely: atypical chest pain and anxiety
- In case of complications: fatigue, dyspnea, cough, syncope, and palpitations
- **Auscultatory findings :**
  - Mitral valve prolapse click: high-frequency, midsystolic click that is best heard at the mitral region
  - High-frequency, mid-to-late systolic murmur that is best heard at the mitral region and may radiate to the axilla (squatting diminishes the murmur)
  - Patients with severe MR: S3 may be heard as a result of left ventricular overload (especially in the left decubitus position).



COMPLICATIONS:

- **Atrial fibrillation**
- **Ventricular ectopics :** MVP patients with ventricular ectopics should be asked to avoid caffeine; β-blockers may be used for symptomatic relief.
- **Transient ischemic attacks (TIA) and/or stroke :** Patients who experience TIA or stroke will require prophylactic doses of aspirin (also see "Therapy" in stroke)
- **Infective endocarditis :** MVP patients with a past history of infective endocarditis require prophylaxis against infective endocarditis (see prophylaxis for endocarditis).
- **Right heart failure:** in case of progressive mitral regurgitation.

INVESTIGATIONS: **Echocardiography** is diagnostic.

TREATMENT: **No treatment is required in most cases.**

Medical	Surgical
• **Prophylaxis against:** infective endocarditis. • **Anti - arrhythmic therapy.** • **Propranolol:** is effective for chest pain.	**Valve replacement** in cases with severe MR.

AORTIC STENOSIS

DEF:

- **Narrowing** of the aortic valve opening, thus restricting the blood flow from the left ventricle to the aorta.
- **It may be:**

Valvular AS	Narrowing of AV opening.
Subvalvular AS	Stenosis below AV in LV outflow tract (membrane or ridge).
Supravalvular AS	Stenosis above AV in Aorta (narrowing in ascending aorta).

EPIDEMIOLOGY:

- **Most common valvular heart disease** in industrialized countries
- Frequently associated with aortic regurgitation
- **Prevalence :**
  - Increases with age
  - May reach up to 12.4% among individuals ≥ 75 years

ETIOLOGY :

1. **Aortic valve sclerosis:** calcification and fibrosis of aortic valve leaflets
   - Most common cause of aortic stenosis
   - Occurs at an increasing rate as patients age (prevalence is 35% in those aged 75–85 years)
   - Similar pathophysiology to atherosclerosis (see risk factors for atherosclerosis)
2. **Bicuspid aortic valve (BAV):** fusion of 2 of the three aortic-valve leaflets in utero
   - Most common congenital heart valve malformation , predominantly affects males (3:1)
   - Predisposes the valve to **dystrophic calcification** and degeneration
   - Patients present with symptoms of aortic stenosis earlier than in regular aortic valve calcification.
   - Congenital AS is rare and usually features a unicuspid or bicuspid valve.
3. **Rheumatic fever**
   - Rare cause of AS in high-income countries due to consistent use of antibiotics for the treatment of streptococcal pharyngitis
   - Still remains a significant cause of AS in lower-income countries, where antibiotics may be less readily available
   - Stenosis is caused by commissural fusion.

Aortic valve sclerosis occurs without aortic stenosis in its early stages, but can progress to aortic stenosis once signs of LVOT obstruction begin to occur.

PATHOPHYSIOLOGY :

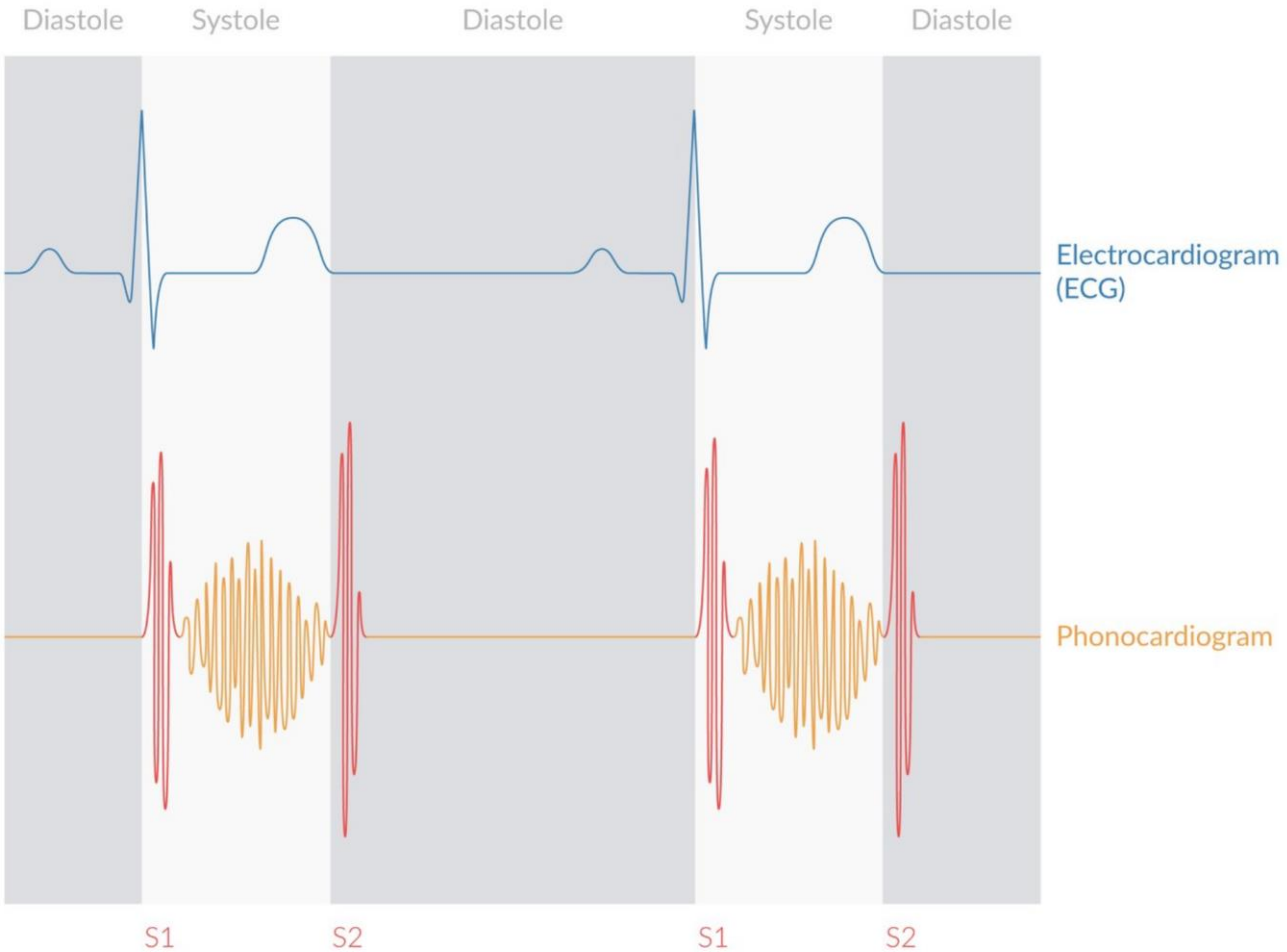
- **During systole:** there is obstruction of blood flow from LV to aorta leading to:
  - Low cardiac output.
  - Pressure overload on LV leading to LVH & LVF.
- **Normally,** the aortic valve area is 3-4 cm2.
- **In severe AS,** it is less than 0.8 cm.

CLINICAL PICTURE :

Symptoms	1. **No symptoms:** in mild cases. 2. **Dyspnea (typically exertional):** Most common symptom. 3. **Angina pectoris:** • Without significant obstructive CAD is caused by subendocaridal ischemia. • Due to increased total oxygen demand secondary to LVH & ↓ Coronary filling due to ↓ COP , shortened diastole. 4. **Dizziness and syncope** (typically exertional). 5. **Symptoms of pulmonary congestion:** due to LVF.
Signs	**A. GENERAL :** ◆ **Pulse:** • **Pulsus parvus et tardus (plateau pulse):** rises slowly, of small volume, returns slowly. • **Pulsus bisferiens:** bifid pulse occurring in double aortic lesion. ◆ **Systolic thrill:** over the carotid arteries. ◆ **Signs of low CO:** in severe cases (esp. low SBP) ◆ **Signs of pulmonary congestion:** due to LVF. **B. CARDIAC :** 1. **PRECORDIAL EXAMINATION:** ◆ **Signs of LVH:** with a heaving apex. ◆ **Systolic thrill:** over 2nd right space → apex & carotid arteries.

2. AUSCULTATION:

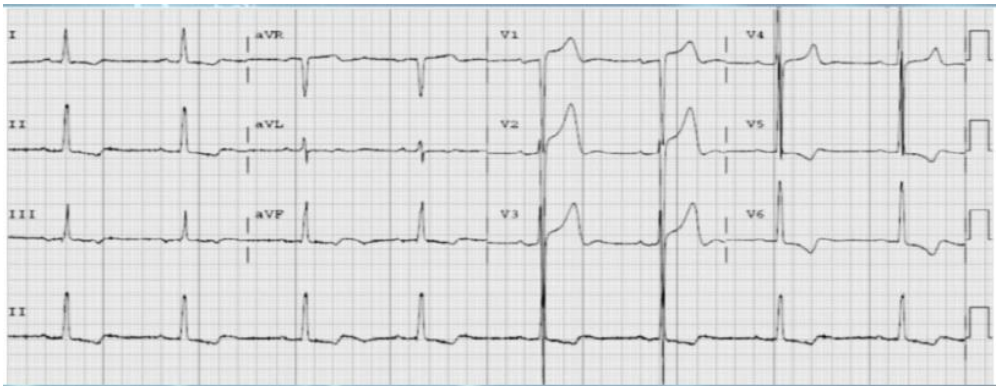
- **Over the aortic area:**
  - **Second heart sound:** weak.
  - **Systolic ejection click:** due to opening of the rigid cusps.
  - **Systolic ejection murmur:**
    - ✓ Midsystolic, Harsh crescendo-decrescendo (diamond shaped)..
    - ✓ Maximum over second right space , radiated to apex & carotid arteries
- **Over the pulmonary area:**
  - Reversed splitting of the second heart sound.
- **Over the mitral area:**
  - **S_4.**
  - **Propagated murmur of AS.**



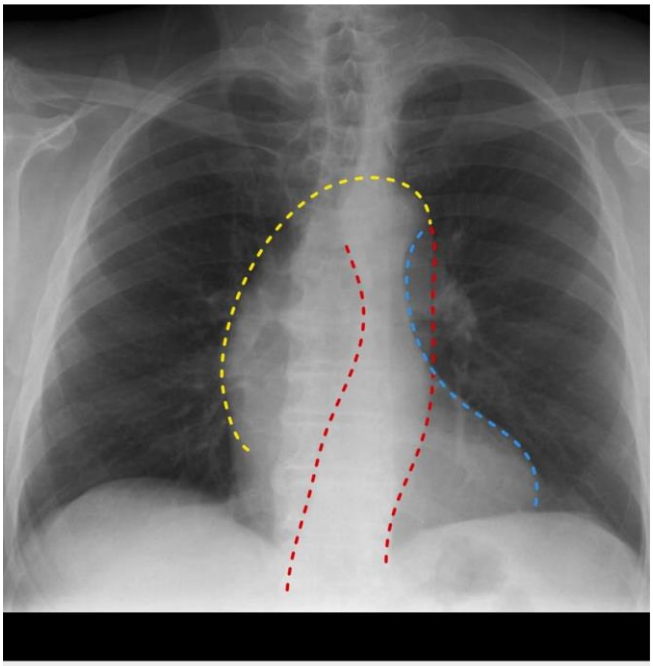
COMPLICATIONS:

1. **LVF.**
2. **Infective endocarditis.**
3. **Sudden death:** usually due to VF.
4. **Heart block:** in calcific AS **due to extension of calcification to the AV bundle**
5. **Rheumatic activity:** in rheumatic AS.

INVESTIGATIONS:

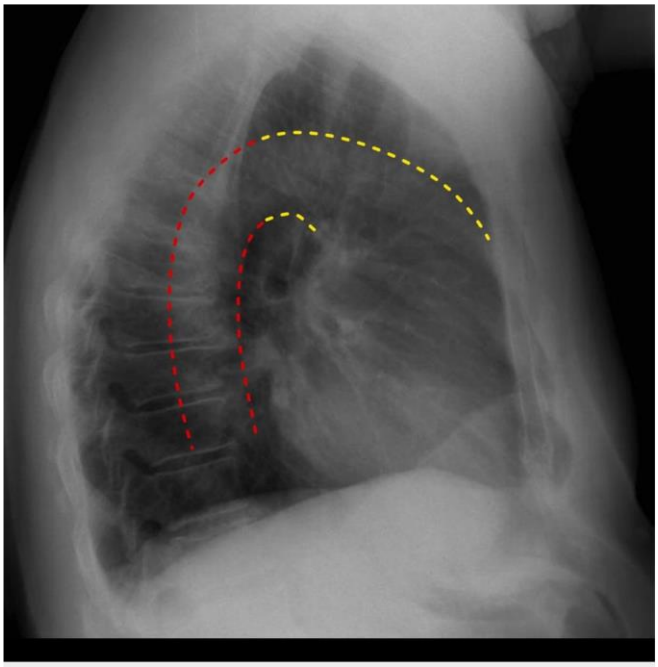
CXR:	◆ **NO ABNORMALITY:** in mild cases. ◆ **LV:** LVH. ◆ **LUNGS:** Pulmonary congestion when LVF occurs. ◆ **AORTA:** • Small aortic knuckle or poststenotic dilatation. • Aortic valve calcification may be seen.
ECG:	◆ May reveal evidence of LVH (typically tall R waves in V5-6 and deep S wave in V1-2) ◆ However, severe AS can occur without significant LVH. 
Echo:	◆ Detects the severity of stenosis by: • **Measurement of valve area.** • **Measurement of pressure gradient across the valve.** ◆ Detects the type of stenosis. ◆ Detects LVH.
Cardiac catheterization & angio-cardiography:	◆ Detects the severity of stenosis by: • Measurement of valve area. • Measurement of pressure gradient across the valve. ◆ Detects the type of stenosis. ◆ Detects LVH.

CXR "AORTiC STENOSiS"



The ascending aorta (yellow dotted line) leading into the arch is dilated, whereas the distal arch and descending aorta (red dotted line) are normal in size. The left heart border (blue dotted line) can be traced upwards along the mediastinum to blend with the aortic arch, explaining why the medial (left) border of the ascending aorta is not visible on x-rays.

Calcified aortic valve (green arrows) noted on CT.



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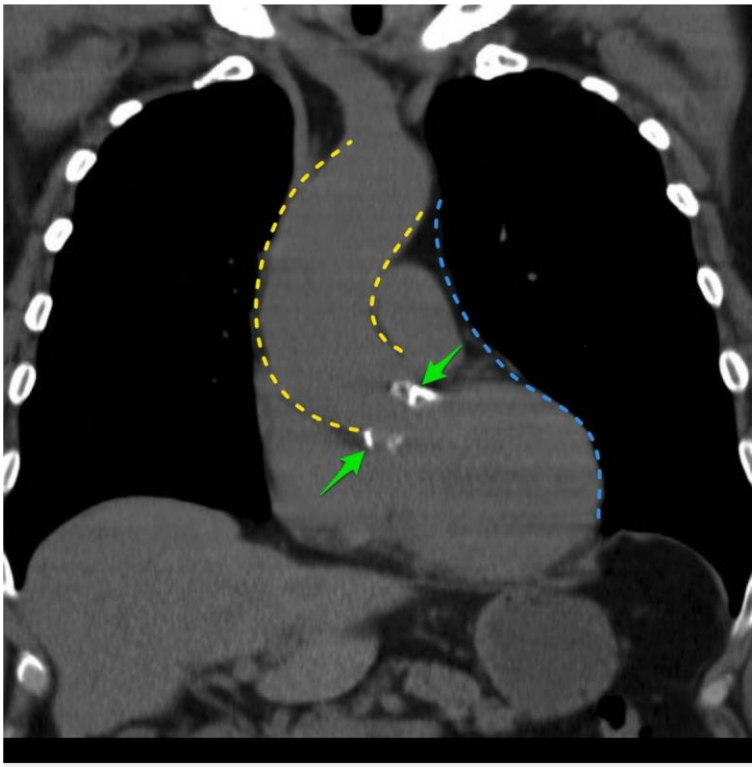

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Calcified aortic valve (green arrows) noted on CT.

CLASSIFICATION:

AMERICAN HEART ASSOCIATION (AHA) / AMERICAN COLLEGE OF CARDIOLOGY (ACC) STAGING SYSTEM

- Used to monitor disease progression and determine the need for intervention.
- Based on echo criteria of valve anatomy and hemodynamics :

Aortic valve area (AVA)	◆ The opening area of the aortic valve measured during systole and an important factor in evaluation of severity of aortic valve stenosis.
Transaortic velocity	◆ The maximum velocity of blood flow measured across the aortic valve during systole, inversely related to aortic valve area.
Mean aortic pressure gradient	◆ The difference in pressure between left ventricle and ascending aorta during systole : • Pressures normally equilibrate relatively rapidly when the valve opens. • Valvular stenosis limits the increase in aortic pressure while increasing LV pressure leading to higher gradient.

Severity		Definition	AVA	Transaortic velocity	Mean aortic pressure gradient
Mild to moderate AS	Stage A	At risk of AS.	3-4 cm2	<2.0 m//sec	<10 mmhg
	Stage B	Progressive AS.	Mild: 1.5-2.9 cm2	2.0 – 2.9 m/sec	10-19 mmhg
			Moderate: 1.0-1.4 cm2	3.0 – 3.9 m/sec	20-39 mmhg
Severe AS	Stage C1	Asymptomatic severe AS (LVEF normal)	≤1.0 cm2	≥4.0 m/sec	≥40 mmhg
	Stage C2	Asymptomatic severe AS with LV dysfunction (LVEF <50%)			
	Stage D	Symptomatic severe AS			

TREATMENT:

Medical	◆ Prophylaxis against infective endocarditis. ◆ Prophylaxis against Rheumatic activity. ◆ Control of HTN, DM & dyslipidemia. ◆ Medical treatment of HF as diuretics.
Surgical	**Aortic valve replacement (AVR) and repair** ◆ **INDICATIONS :** ◆ **Symptomatic** patients with severe, high-gradient AS ◆ **Asymptomatic** patients with severe AS and: ➢ Significantly ↓ LVEF ➢ Undergoing cardiac surgery for other indications ◆ Presence of exertional symptoms (dyspnea on exertion , angina pectoris, syncope) is an indication for surgery. ◆ **PROCEDURE :** 1. **Surgical AVR (SAVR)** is recommended for patients with: • Age < 65 years • Life expectancy > 20 years • Low to moderate surgical risk 2. **Transcatheter AVR (TAVR)** • Recommended for patients with: ➢ Age > 80 years ➢ Life expectancy < 10 years ➢ High or prohibitive surgical risk and predicted survival of > 12 months 3. **Percutaneous balloon valvuloplasty** • May be used in children, adolescents, and young adults • Limited role in older patients

AORTIC REGURGE

ETIOLOGY:

Acute AR	◆ **Infective endocarditis :** • Most common valvular cause of Acute AR. ◆ **Aortic dissection (ascending aorta) :** • Most common Aortic cause of Acute AR. ◆ **Chest trauma.** ◆ **Iatrogenic complications :** After percutaneous aortic balloon dilation or trans-catheter aortic valve replacement (TAVR).
Chronic AR	◆ **Primary valvular defect** • **Congenital bicuspid aortic valve**: most common cause of AR in young adults in **high-income countries** • Calcific aortic valve disease: most common cause of AR in older patients in high-income countries • **Rheumatic heart disease**: most common cause of AR in **lower-income countries** ◆ **Aortic dilatation** • CT disorders (Marfan syndrome, Ehlers-Danlos syndrome) • Chronic hypertension • Aortitis of any etiology (e.g., tertiary syphilis) • Thoracic aortic aneurysm • Ankylosing spondylitis.

PATHOPHYSIOLOGY:

General	◆ **Regurgitation of blood from aorta into left ventricle (LV) leads to:** • Increased systolic blood pressure & decreased diastolic pressure • Widened pulse pressure → **water hammer pulse**
Acute AR	◆ Because LV cannot sufficiently dilate in response to regurgitant blood, LV end-diastolic pressure increases rapidly → pressure transmits backwards into pulmonary circulation → pulmonary edema & dyspnea ◆ ↓ cardiac output if severe → cardiogenic shock & myocardial ischemia
Chronic AR	◆ Initially, a **compensatory increase in stroke volume** can maintain adequate cardiac output despite regurgitation (compensated HF) ◆ Over time, increased left ventricular end-diastolic volume → LV enlargement and **eccentric hypertrophy of myocardium** → left ventricular systolic dysfunction → decompensated heart failure

CLINICAL PICTURE:

1. Acute aortic regurgitation

Signs & symptoms	◆ Sudden, severe **dyspnea** ◆ Rapid cardiac decompensation secondary to heart failure ◆ **Pulmonary edema** ◆ Symptoms related to underlying disease (e.g., fever due to endocarditis, chest pain due to aortic dissection)
Auscultation	◆ Soft S_1 ◆ Soft and short early diastolic murmur

2. Chronic aortic regurgitation

A. SYMPTOMS :

Generalized Body Throbbing	◆ Due to increased arterial pulsations.
Palpitation	◆ Due to forcible LV contraction.
Symptoms of pulmonary congestion	◆ When LVF occurs.
Angina pectoris	◆ **Classic angina of effort:** Decreased DBP: reduces coronary filling. ◆ **Angina of Lewis:** Nocturnal & associated with autonomic disturbances e.g. sweating & tachycardia.

B. GENERAL SIGNS :

"Peripheral signs of aortic regurge" (due to big pulse volume)

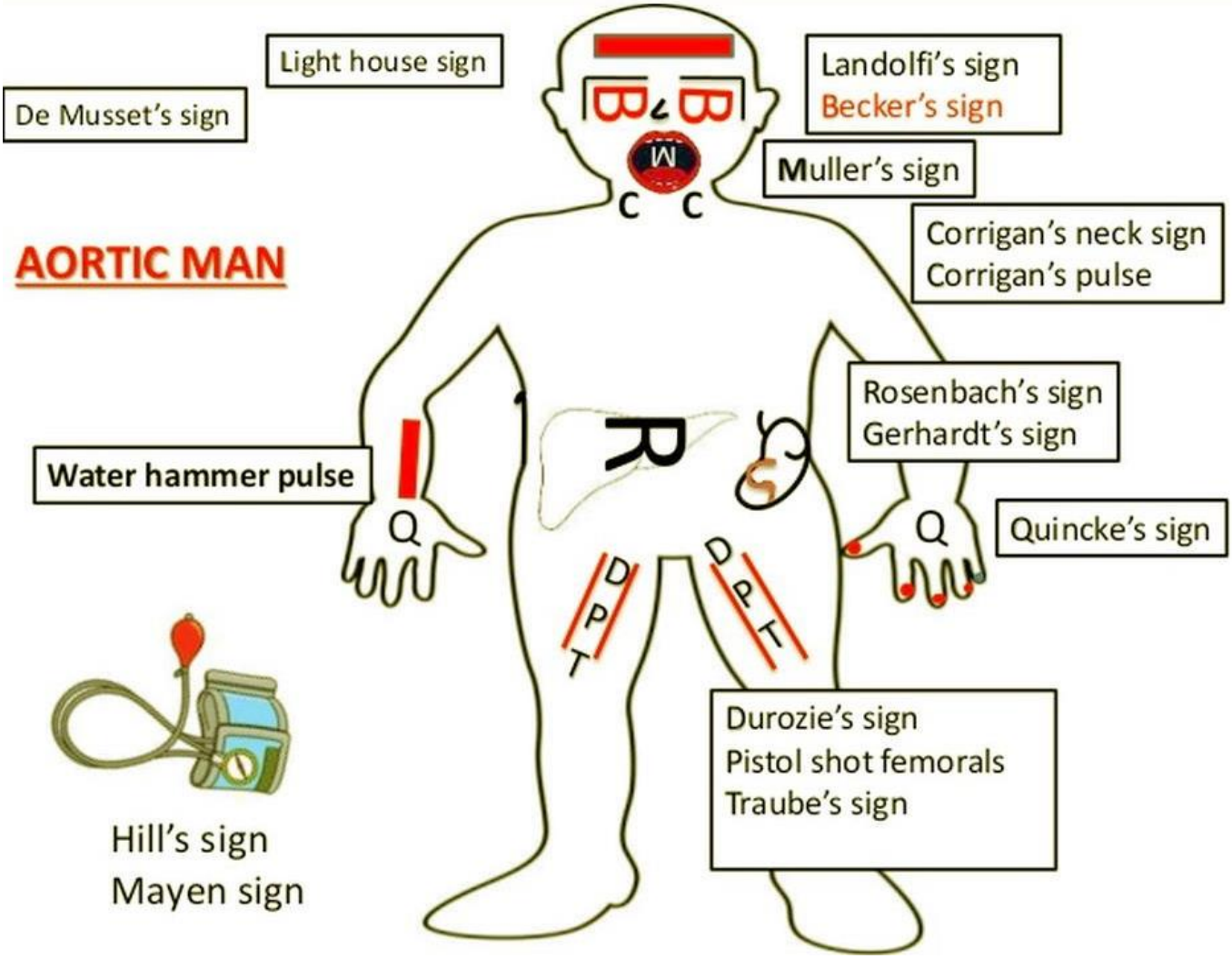
1. Head & Neck	◆ **De Musset sign:** rhythmic nodding or bobbing of the head in synchrony with heartbeats ◆ **Mueller sign:** Systolic pulsations of the uvula. ◆ **Becker sign:** Visible pulsation of the retinal arteries. ◆ **Corrigan's sign:** Marked visible carotid pulsation.
2. Upper Limb:	◆ **BP:** ↑ systolic & ↓ diastolic BP. ◆ **Pulse:** Water hammer pulse. ◆ **Quincke's pulse:** visible capillary pulse when pressure is applied to the tip of a fingernail. ◆ **Mayne sign:** More than 15 mmHg decrease in diastolic BP with arm elevation from the value obtained with the arm in standard position.

3. Abdomen & Lower Limb:	◆ **Traube sign:** pistol shot-like sounds heard over the femoral artery on auscultation ◆ **Duroziez sign:** to-and-fro bruit over the femoral artery that is heard when slight pressure is applied with a stethoscope ◆ **Hill sign:** Popliteal cuff systolic pressure exceeding brachial cuff pressure by more than 20 mmHg with patient in the recumbent position. ◆ **Rosenbach sign:** Systolic pulsations of the liver. ◆ **Gerhard sign:** Systolic pulsations of the spleen.

AR WITH MINIMAL PERIPHERAL SIGNS:

- Mild AR.
- ↓ Systolic BP: MS, AS.
- ↑ Diastolic BP: Systemic hypertension.

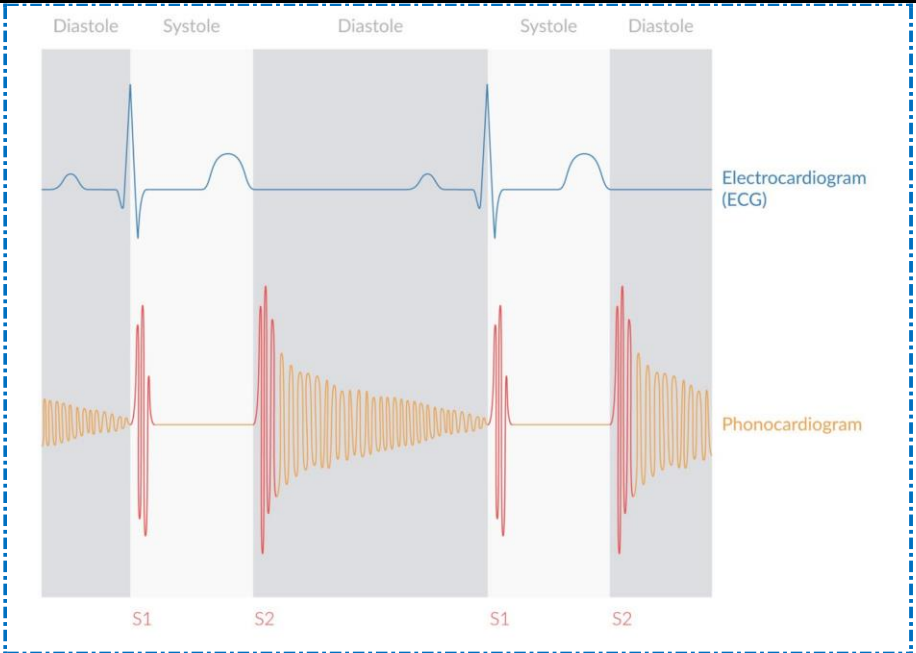
Signs of Aortic Regurgitation



C. LOCAL "CARDIAC" SIGNS :

<table>
<tr><td>Precordial examination</td><td>◆ Signs of LV enlargement: with hyperdynamic apex.
◆ No thrill over the aortic area: in isolated AR.</td></tr>
<tr><td>Auscultation</td><td>
OVER THE AORTIC AREA :
1. Normal second heart sound
2. Murmur of AR:
<table>
<tr><td>Timing</td><td>◆ Early diastolic.</td></tr>
<tr><td>Character</td><td>◆ Soft blowing, decrescendo.</td></tr>
<tr><td>Site</td><td>◆ Maximum over the third space.</td></tr>
<tr><td>Propagation</td><td>◆ To the apex.</td></tr>
<tr><td>Position</td><td>◆ best heard with the diaphragm of the stethoscope while patient is
• Sitting up.
• Leaning forward.
• Holding his breath in forced expiration.</td></tr>
</table>
3. Soft ejection systolic murmur:
◆ Due to ↑ blood flow across the aortic valve (relative AS).

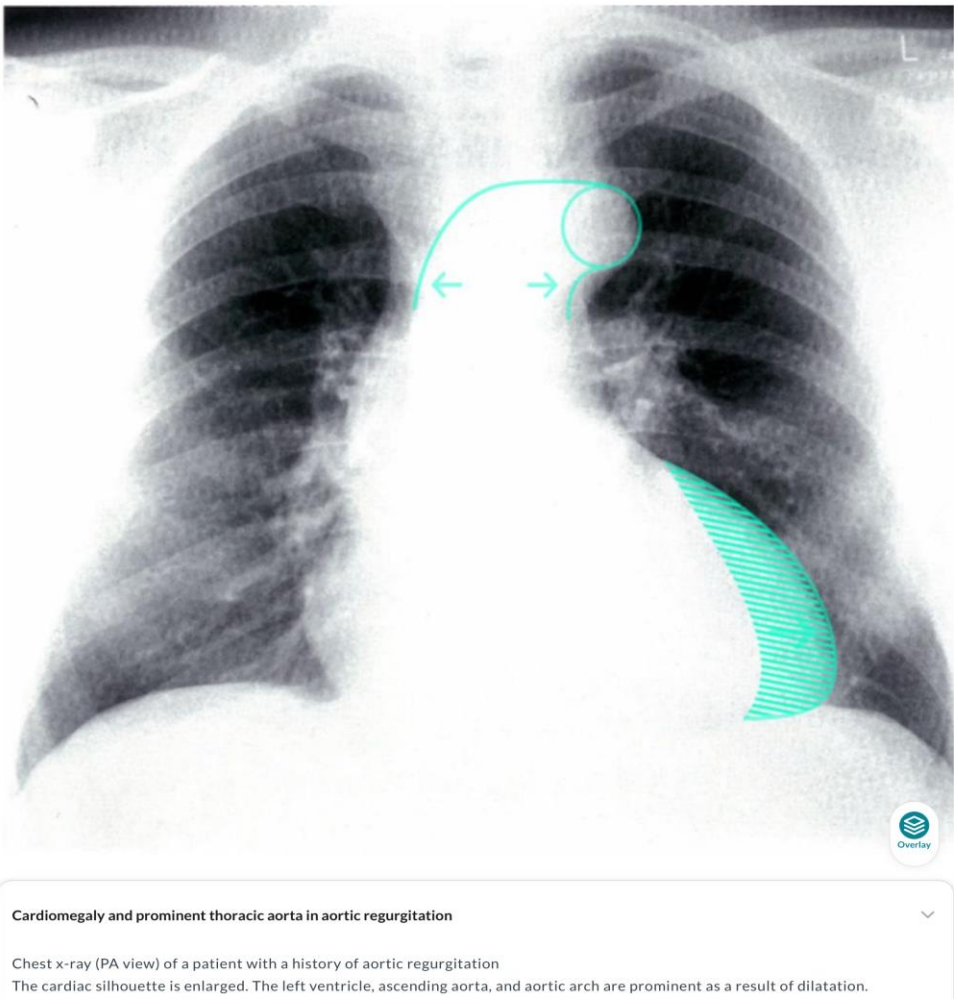
OVER THE MITRAL AREA (APEX):
◆ Mid diastolic murmur of relative MS (Austin - Flint murmur)
◆ Pan systolic murmur of relative MR.
◆ Propagation of ejection systolic murmur of relative or combined AS.
◆ Propagation of early diastolic murmur of AR itself.
</td></tr>
</table>



COMPLICATIONS:

a. Rheumatic activity.
b. Infective endocarditis.
c. LVF.

INVESTIGATIONS:

CXR:	◆ LV enlargement & dilated aorta **"Aortic configuration (Boot-shaped heart)".** ◆ **Lungs:** Pulmonary congestion when LVF occurs.  Cardiomegaly and prominent thoracic aorta in aortic regurgitation Chest x-ray (PA view) of a patient with a history of aortic regurgitation The cardiac silhouette is enlarged. The left ventricle, ascending aorta, and aortic arch are prominent as a result of dilatation.
ECG	◆ LV enlargement.
Echocardiography:	◆ Detects the severity of the lesion. ◆ Detects LV enlargement.
Cardiac catheterization & angiocardiography:	◆ Detects the severity of the lesion. ◆ Detects LV enlargement.

CLASSIFICATION:

AMERICAN HEART ASSOCIATION (AHA) / AMERICAN COLLEGE OF CARDIOLOGY (ACC) STAGING SYSTEM

Severity	Definition	
Stage A	At risk of AR.	
Stage B	Progressive AR.	Mild regurgitation.
		Moderate regurgitation
Stage C1	Asymptomatic severe AR (LVEF >55%)	
Stage C2	Asymptomatic severe AR (LVEF ≤55% or LV dilatation >50 mm)	
Stage D	Symptomatic severe AR.	

TREATMENT:

1. Acute aortic regurgitation

- Severe acute AR requires surgical treatment as soon as possible.
- Medical management of complications (e.g., pulmonary edema) should not delay definitive treatment.

2. Chronic aortic regurgitation

Medical	• Prophylaxis against infective endocarditis, secondary prevention of rheumatic activity. • Diuretics for HF symptoms, Dilators. Good blood pressure control (afterload reduction) with calcium channel blockers and vasodilators (ACE inhibitors).

Surgical	• **INDICATION :**
	  • Acute severe AR
	  • Symptomatic chronic severe AR
	  • Asymptomatic chronic severe AR with one of the following:
	  1. Reduced LVEF ≤ 55%
	  2. Consider if LVESD > 50 mm
	  3. Cardiac surgery for other indications
	• **PROCEDURES:**
	1- AV replacement (mechanical or biological prosthesis) ± Aortic root replacement.
	2- AV repair.

TRICUSPID STENOSIS

ETIOLOGY :

Rheumatic Fever	◆ The most common cause. ◆ Almost always associated with MS.
Carcinoid $	◆ A rare cause.

PATHOPHYSIOLOGY :

- **During diastole, there is obstruction of blood flow from RA to RV leading to:**
  - Systemic venous congestion.
  - Low CO.

CLINICAL PICTURE :

A. SYMPTOMS :

- Systemic congestion.
- low cardiac output.

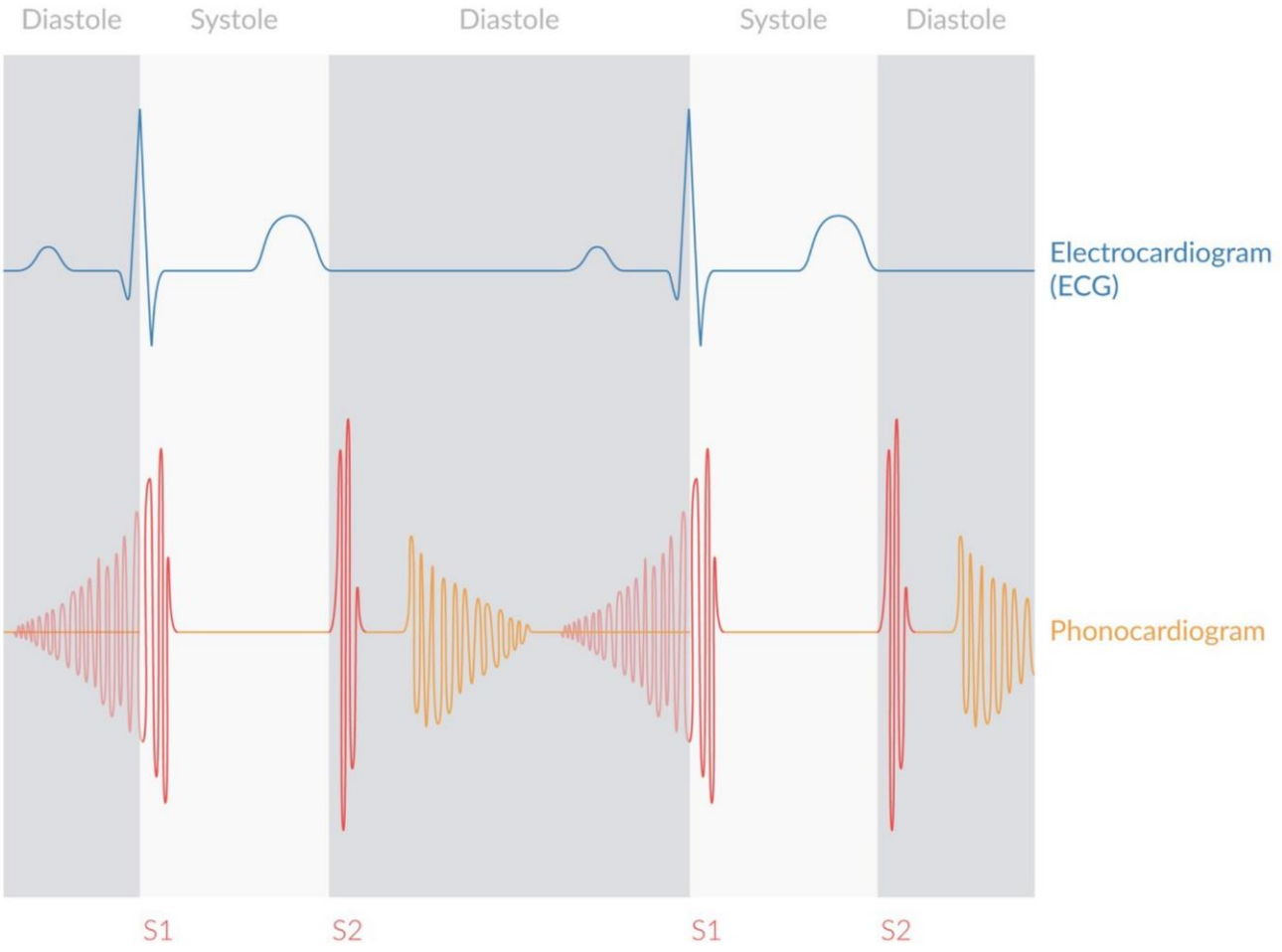
B. SIGNS :

- **GENERAL:**

Systemic congestion	• Congested pulsating neck veins with giant A wave. • Enlarged tender pulsating liver. • Ascites before edema of lower limbs (ascites precox).
Low CO	

- **CARDIAC:**

Precordial examination	• **RA ENLARGEMENT** (dullness to the right border of the sternum). • **RV ENLARGEMENT** (due to frequently associated MS).
Auscultation	• **OVER TRISCUPID AREA:** a. Accentrated first heart sound. b. Opening snap. c. Mid-diastolic murmur that increases by inspiration.



INVESTIGATIONS :

CXR	◆ RA & RV enlargement.
ECG	◆ RA & RV enlargement.
Echocardiography	◆ Diagnostic.
Cardiac catheterization & angiocardiography:	◆ Diagnostic.

TREATMENT:

Medical	Surgical
TTT of right sided - HF.	Valve replacement.

TRICUSPID REGURGE

ETIOLOGY :

FUNCTIONAL	◆ The most common cause due to dilatation of the tricuspid ring secondary to RV dilatation.
ORGANIC "rare"	◆ Rheumatic fever. ◆ Infective endocartitis. ◆ Congenital. ◆ Carcinoid syndrome.

PATHOPHYSIOLOGY:

- During systole, blood regurgitates from RV to RA causing:
  - Low CO.
  - RA enlargement.
  - RV enlargement.
  - RVF (systemic congestion).

CLINICAL PICTURE:

A. SYMPTOMS :

- Systemic congestion.
- low cardiac output.

B. SIGNS :

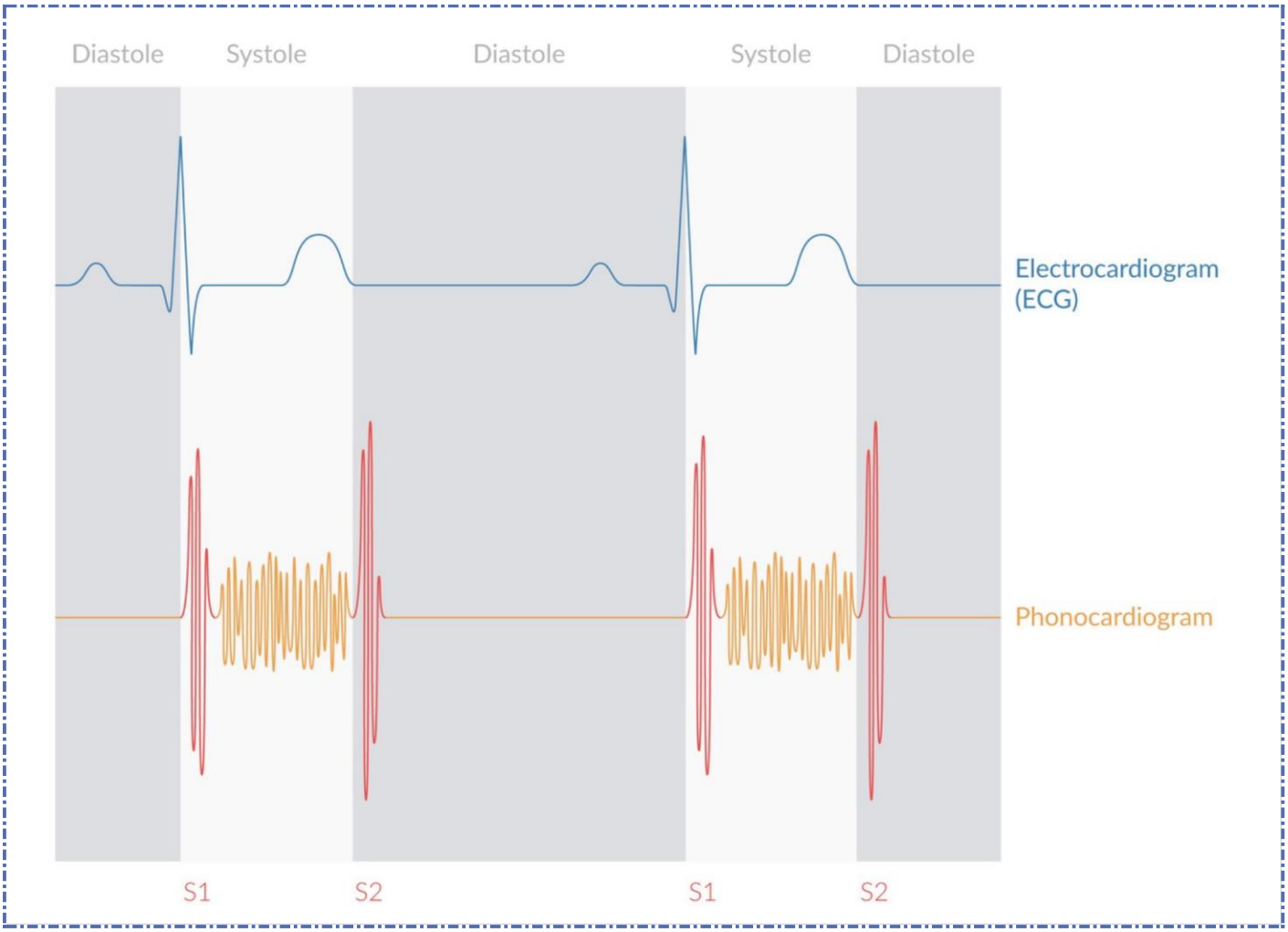
- **GENERAL:**

Systemic congestion	• Congested pulsating neck veins with systolic expansion. • Enlarged tender pulsating liver. • Ascites before edema of lower limbs (ascites precox). • Mild jaundice & peripheral cyanosis (Cyano-icteric face).
Low CO	

- **CARDIAC:**

Precordial examination	• RA & RV enlargement . • Systolic thrill over tricuspid area (rarely).

Auscultation	First heart sound	◆ Weak (muffled).
	Third heart sound	◆ Present.
	Murmur of mitral regurge	◆ **TIMING:** pan systolic. ◆ **CHARACTER:** soft or harsh. ◆ **SITE:** tricuspid area. ◆ **PROPAGATION:** To the axilla & not to the axilla. ◆ **CARAVALOO'S SIGN:** murmur increases with inspiration being of right sided origin.



INVESTIGATIONS :

CXR	◆ RA & RV enlargement.
ECG	◆ RA & RV enlargement.
Echocardiography	◆ Diagnostic.
Cardiac catheterization & angiocardiography:	◆ Diagnostic.

TREATMENT:

Medical	Surgical
TTT of right sided - HF.	Valve replacement.

PULMONARY STENOSIS

ETIOLOGY :

Organic	◆ Congenital: most common cause. ◆ Carcinoid syndrome: rare
Functional	◆ Pulmonary HTN.

PULMONARY REGURGE

ETIOLOGY :

Functional	◆ Mostly due to **PH** which causes dilatation of the pulmonary ring.
Organic	◆ Congenital. ◆ Carcinoid syndrome.

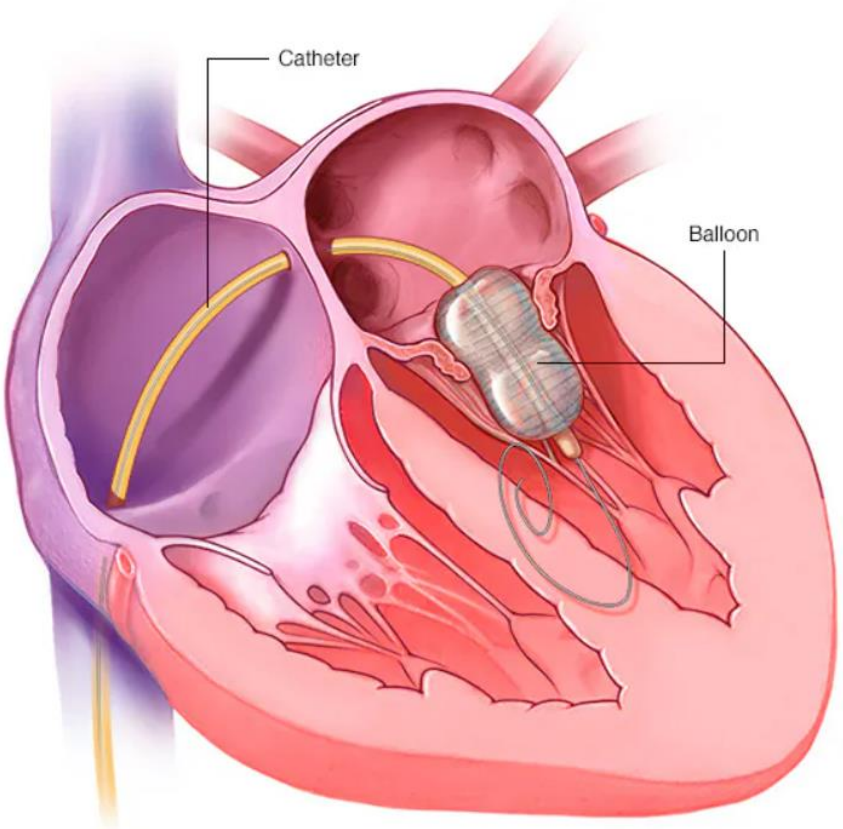
Valve lesion	Most important cause	Most important symptoms	Most important sign	CXR	ECG
MS	Rheumatic	Pulm. Congestion esp. Dyspnea	◆ Accentuated S1 ◆ Mid-diastolic rumbling M over apex ◆ Pulmonary hypertension	LAB Pulm. congestion	P-mitrale P-pulmonale AF
MR	Rheumatic	Palpitation	◆ Pan systolic M & thrill over apex propagated to axilla	LAB LVE	LAB LVE
AS	Rheumatic Calcific Congenital	Low CO esp. Angina & Syncope	◆ Heaving apex ◆ Harsh ejection systolic M & thrill "over second right space → carotid "	LVE Small aortic knuckle Or Post stenotic dilatation	LVE
AR	Rheumatic Syphilitic	Throbbing Palpitation	◆ Peripheral signs ◆ Hyperdynamic apex ◆ Early diastolic soft blowing M " over the third left space "	Boot-shaped heart	LVE
TS	Rheumatic	Systemic congestion	◆ Giant-a-wave ◆ Mid-diastolic rumbling M over tricuspid " ↑with inspiration"	RAE	RAE
TR	Functional	Systemic congestion	◆ systolic expansion in neck veins ◆ Pan-systolic M over tricuspid " ↑with inspiration "	RAE RVE	RAE RVE

	Maximum point	Murmur	Characteristics
Aortic stenosis	• Aortic valve (parasternal 2nd right ICS. • Erb point	• Harsh crescendo-decrescendo systolic ejection murmur	• Radiation to the carotids • Soft S_2 & Possibly ejection click
Aortic regurgitation		• Diastolic murmur with a decrescendo. • Possible addition systolic murmur	• Immediately following 2nd heart sound (immediate diastolic murmur) • Austin Flint Murmur
Mitral stenosis	• Heart apex (midclavicular 5th left ICS)	• Delayed diastolic murmur with a decrescendo	• "Tympanic" 1" heart sound • Mitral opening murmur/opening snap (OS)
Mitral valve prolapse		• Late-systolic crescendo	• Midsystolic high-frequency click (due to tensing of chordae tendinae) • Loudest before S2
Mitral regurgitation	• Heart apex (midclavicular 5th left ICS) • Left axilla	• Holosystolic murmur • 3rd heat sound & Quiet 1st HS	• Blowing • Radiation to axilla.
Pulmonary stenosis	• Pulmonary valve (parasternal 2nd left ICS)	• Crescendo-decrescendo ejection systolic murmur	• Possible radiation into the back • Possible early systolic pulmonary ejection click and/or widely split 2nd HS
Pulmonary regurgitation		• Diastolic murmur with a decrescendo	• Graham Steell murmur high-frequency decrescendo diastolic murmur
Tricuspid stenosis	• Tricuspid valve (parasternal 4th left ICS)	• Delayed diastolic murmur with a decrescendo • Possible pre-systolic crescendo	
Tricuspid regurgitation		• Holosystolic murmur	• Augmentation of murmur's intensity with inspiration **(Carvallo sign)**

SURGICAL ASPECTS OF VALVULAR HEART DISEASES

VALVE REPAIR

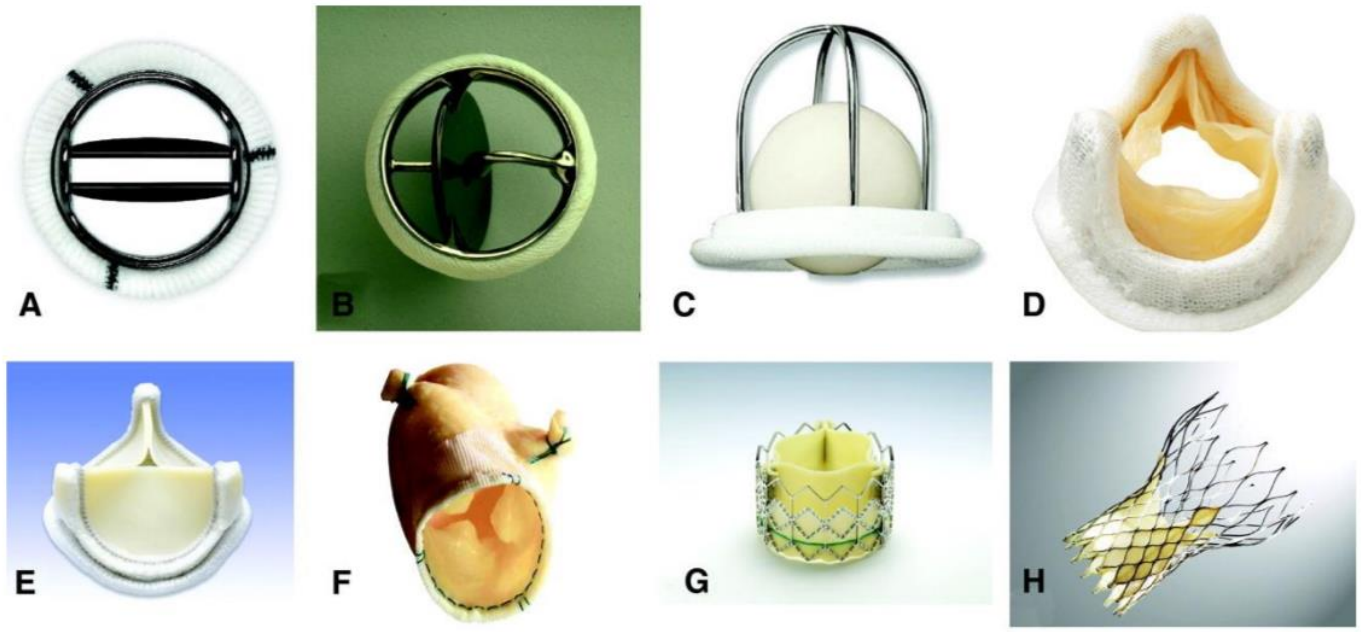
Valve Reconstruction	◆ **Annuloplasty:** • Ring-shaped device is attached to outside of valve opening to reestablish the shape and function of the valve. • Commonly used to treat mitral valve regurgitation ◆ **Leaflet repair:** involves the use of a clip device; may be performed in patients with mitral valve regurgitation
Valvuloplasty	◆ A procedure performed in patients with valvular stenosis (e.g., aortic valve stenosis or mitral stenosis) to separate fused or calcified valve leaflets. ◆ **Approach :** • Percutaneous balloon valvuloplasty: A balloon is advanced into the target valve (either transfemorally or transapically) and inflated, opening the stenotic valve. • Open commissurotomy: open surgical procedure to separate fused and/or calcified leaflets



PROSTHETIC HEART VALVE REPLACEMENT

	Mechanical prosthetic valve	Biological prosthetic valve
Indications	◆ **Younger patients** • Patients < 50 Y who require aortic valve replacement • Patients < 65 Y who require mitral valve replacement ◆ Patients taking **anticoagulants** ◆ Patients with a high surgical risk for **reintervention**	◆ **Older patients** (≥ 65 Y of age). ◆ Patients with high risk of **bleeding** ◆ Patients who currently pregnant or have a desire to carry **pregnancy** in the future
Advantages	◆ Valve has a long lifespan. ◆ Durable. ◆ Available. ◆ Cheap. ◆ Easy implantation, Explanation	◆ Anticoagulation is only necessary for 3 months after surgery. ◆ Better hemodynamic performance. ◆ Non-noisy.
Disadvantages	◆ Life-long anticoagulation is necessary (e.g., with a vitamin K antagonist). ◆ Noisy.	◆ Short life span due to sclerotic degeneration (biological prosthetic valves may need to be replaced every 10 years) ◆ Less durable, expensive. ◆ Difficult in implantation , explanation
Types	1. Bi-leaflet valve. 2. Tilting valve. 3. Ball and cage valve.	**1- Xenograft Valves:** a- Bovine pericardial valve. b- Porcine pericardial valve. **2- Homograft Valves.** **3- Autograft Valves** (Ross and Ozaki valves).

Different types of prosthetic valves



A	Bileaflet mechanical valve (St Jude)	E	Stented pericardial bioprosthesis (Carpentier-Edwards Magna)
B	Monoleaflet mechanical valve (Medtronic Hall)	F	Stentless porcine bioprosthesis (Medtronic Freestyle)
C	Caged ball valve (Starr-Edwards)	G	Percutaneous bioprosthesis expanded over a balloon (Edwards Sapien)
D	Stented porcine bioprosthesis (Medtronic Mosaic)	H	Self-expandable percutaneous bioprosthesis (CoreValve).

REPLACEMENT ROUTE:

- **Surgical heart valve replacement:**
  - May be done in conjunction with a CABG in suitable patients who require both procedures
- **Transcatheter aortic valve replacement (TAVR) :**
  - A minimally invasive, percutaneous procedure that utilizes an endovascular technique to replace the aortic valve.
  - A collapsible replacement valve is inserted via a catheter and placed over the native valve.
  - Once the replacement valve is expanded, it displaces the old valve and assumes its function.
- **Transcatheter mitral valve replacement .**

COMPLICATIONS OF HEART VALVE INTERVENTION:

Patient-prosthesis mismatch	◆ Caused by implantation of a prosthetic valve with a functional area that is too small for the cardiac demand of the patient ◆ Leads to **left ventricle hypertrophy** and impaired exercise capacity ◆ Increased risk of other cardiac events (e.g., arrhythmia, MI) and increased mortality
Prosthetic valve thrombosis	◆ **Etiology:** • Mechanical valves are more prone to thrombosis than biological prosthetic valves. • Insufficient anticoagulatory therapy after valve replacement ◆ **Clinical features:** • Signs of acute heart failure due to valve dysfunction (e.g., dyspnea, fatigue, peripheral edema) • New murmur consistent with valve obstruction and/or regurgitation • Thromboembolic event (e.g., TIA) ◆ **Diagnostics:** • First-line imaging: transthoracic or transesophageal echocardiography. • If echocardiography is negative: cardiac CT to assess leaflet anatomy and motility. ◆ **Treatment:** • Anticoagulation and fibrinolysis. • Surgical valve replacement.
Prosthetic valve dysfunction	◆ e.g., paravalvular leak
Prosthetic valve stenosis or regurgitation	◆ Clinical presentation is similar to native valve disease.
Cardiac complications	◆ Prosthetic Valve Endocarditis, heart failure, pericarditis, high-grade AV block, atrial fibrillation
Vascular complications	◆ Bleeding, femoral artery dissection, hemolytic anemia in patients with a mechanical valve , thromboembolism
Others	◆ Stroke, acute kidney injury

LONG-TERM MANAGEMENT:

1. **Anti-coagulation :**

<table>
<tr><th colspan="2">MECHANICAL VALVE</th><th>BIOLOGICAL VALVE</th></tr>
<tr><td colspan="2">Warfarin for life</td><td rowspan="3"><u>Warfarin</u> for 1st 3-6 months: INR of 2.5 (2-3)
<u>Aspirin</u> 75-100 mg daily lifelong.</td></tr>
<tr><th>AVR</th><th>MVR</th></tr>
<tr><td>INR: 2.5 (2-3)</td><td>INR: 3 (2.5-3.5)</td></tr>
</table>

2. **Endocarditis prophylaxis.**
3. **Clinical follow up.**
4. **Echocardiographic follow up.**

ENDOCARDITIS

DEFINITION:

- Inflammation of the **endocardial** surface of the heart.

TYPES :

A. INFECTIVE ENDOCARDITIS :

1. According to the causative organism:

- Bacterial endocarditis.
- Non - bacterial endocarditis.

2. According to the clinical presentation:

	Subacute bacterial endocarditis (SBE):	Acute bacterial endocarditis (ABE):
Onset	Insiduous	Acute
Heart	Diseased heart	Diseased heart or normal heart.
Organism	Low virulence, e.g. Strept. viridans.	High virulence, e.g. Staph, aureus.
Prognosis	Low mortality	High mortality
Incidence	Common	Rare

B. NON - INFECTIVE ENDOCARDITIS :

- Rheumatic fever.
- SLE.

Infective Endocarditis

DEFINITION :

- Infection of the endocardial surfaces of the heart at one or more heart valves or the mural endocardium.

ETIOLOGY :

COMMON PATHOGENS CAUSING IE :	
Staphylococcus aureus	◆ **35-40%** of native valve IE cases. ◆ **Most common case of acute IE** including **persons who inject drugs** and **patients with prosthetic valve** or **pacemakers/ICDs.** ◆ Typically affects **healthy valves** ◆ Usually **fatal within 6 weeks** if left untreated
Viridians streptococci	◆ **20%** of native valve IE cases ◆ **Most common cause of subacute IE** especially in pre-damaged native valves (mainly mitral valve) ◆ Common cause of IE **dental procedure**, **respiratory tract infections and biopsy.** ◆ Produce **dextrans** that facilitate binding of fibrin-platelet aggregates on heart valves
Staphylococcus epidermidis	◆ **Less than 15%** of native valve IE cases ◆ **Bacteremia** from **Infected peripheral venous catheters** ◆ **Common cause of subacute IE in** patients with **prosthetic heart valves, pacemakers, or ICD.**
Enterococci (Enterococcus faecalis)	◆ **10%** of native valve IE cases ◆ **Multiple** drug resistance ◆ **Common cause of IE** following **nosocomial UTIs** ◆ Cause native and prosthetic valve IE ◆ **Following GIT** or **genitourinary procedures**
Streptococcus gallolyticus subsp. callolyticus	◆ **Less than 15%** of native valve IE case ◆ Associated with **colorectal cancer** ◆ If Sgg is detected, **colonoscopy** is indicated
Gram-negative HACEK group	◆ **Less than 5%** of native valve IE cases ◆ Physiological oral pharyngeal flora ◆ In patients e **poor dental hygiene** and/or **periodontal infection**

Fungal endocarditis (Candida, Aspergillus fumigatus)	◆ **Less than 5%** of native valve IE cases ◆ **At risk groups :** 1. Immunosuppressed patients (patients with HIV or organ transplantation) 2. Persons who inject drugs 3. Patients who have received cardiosurgical interventions 4. Patients with long term indwelling IV Catheter.
Coxiella burneti Bartonella species	◆ **Less than 5%** of native valve IE cases ◆ Gram-negative pathogens responsible for culture-negative endocarditis

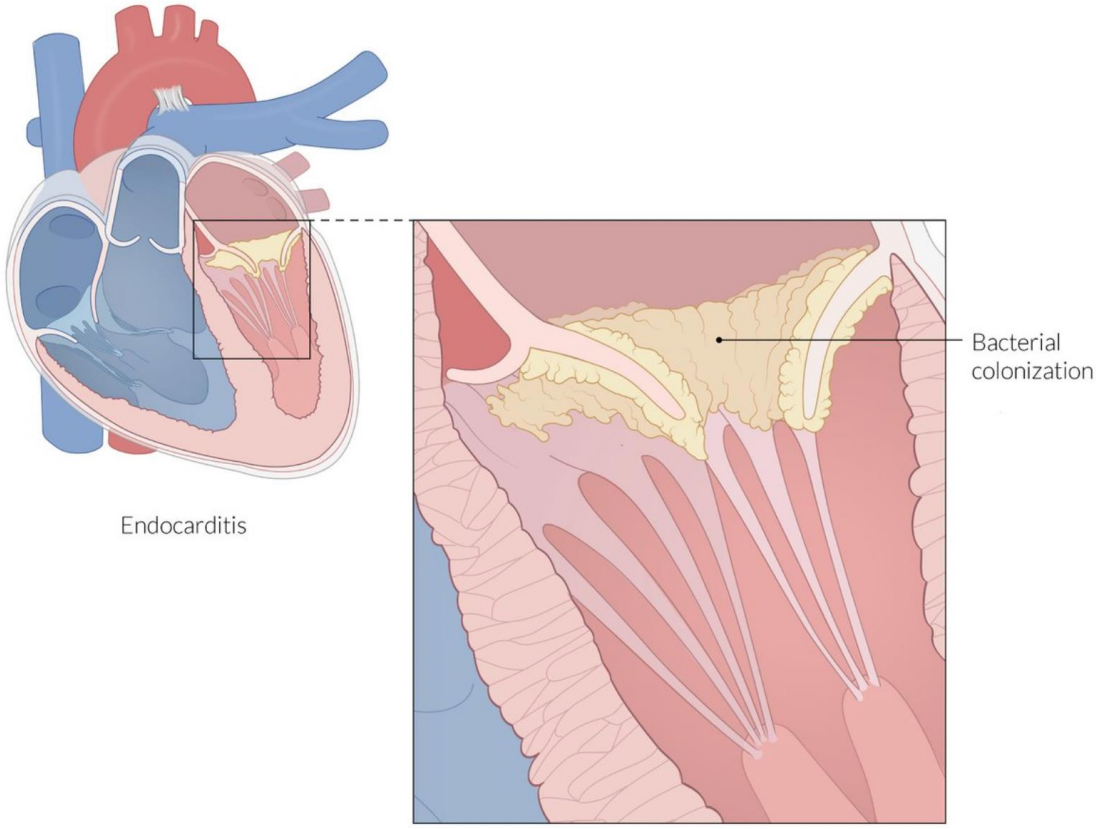
RISK FACTORS FOR INFECTIVE ENDOCARDITIS:

Demographics	◆ Male sex ◆ Age > 60 years
Cardiac conditions	◆ **Acquired valvular disease** (e.g., rheumatic heart disease, aortic stenosis, degenerative valvular disease) ◆ **Prosthetic heart valves** ◆ **Congenital heart defects** (e.g., VSD, bicuspid aortic valve) ◆ Previous IE ◆ Cardiac implantable electronic device (CIED)
Non-cardiac risk factors	◆ **Poor dental status.** ◆ Dental procedures. ◆ Nonsterile venous injections (e.g., in **IV drug use**) ◆ Intravascular devices ◆ Surgery ◆ Chronic hemodialysis ◆ Immunocompromise (e.g., HIV infection, diabetes) ◆ Other bacterial infections (e.g., UTIs, spondylodiscitis, periodontal infection)

PATHO-PHYSIOLOGY:

Pathogenesis	1. Damaged valvular endothelium → exposure of the subendothelial layer → adherence of platelets and fibrin → **sterile vegetation** (microthrombus) 2. Localized infection or contamination → bacteremia → **bacterial colonization** of vegetation → formation of fibrin clots encasing the vegetation → **valve destruction** with loss of function (valve regurgitation)
Valve involvement	◆ Frequency of valve involvement: **mitral valve** > aortic valve > tricuspid valve > pulmonary valve ◆ The tricuspid valve is the most commonly affected valve in persons who inject drugs (associated with *Pseudomonas*, *S. aureus*, and *Candida*).
Clinical consequences	◆ Bacterial vegetation → bacterial thromboemboli → vessel occlusion → infarctions ◆ Emboli can lead to metastatic infections of other organs. ◆ Formation of immune complexes and antibodies against tissue antigens → glomerulonephritis, Osler nodes

"Don't tri drugs for the sake of your tricuspid valves."



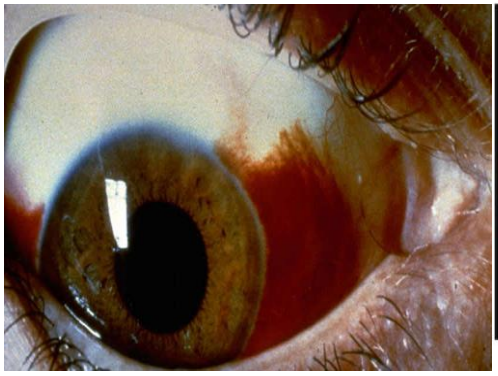
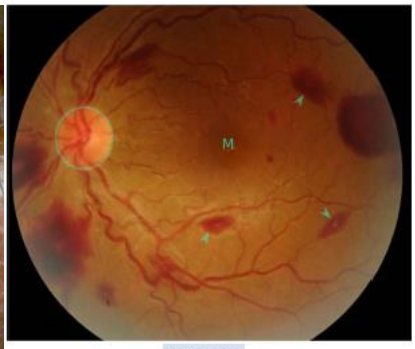
NB: **2 factors** are needed to increase the incidence of infective endocarditis:

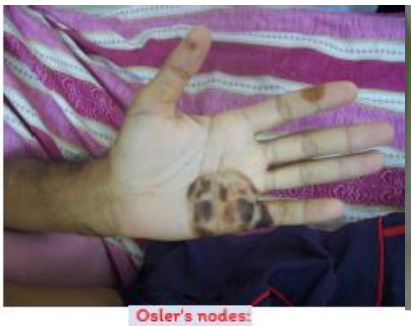
High pressure gradient:	◆ **It** is common on mitral & aortic valves (valves of the left side). ◆ It is rare on tricuspid & pulmonary valves (valves of the right side). ◆ It is more common in MR than in MS. ◆ It is more common in VSD than in ASD. ◆ It is RARE in: AF.
Narrow orifice:	◆ It is more common in small VSD than in big VSD.

CLINICAL PICTURE :

- **Manifestations of the disease are due to:**
  a. Toxemia.
  b. Cardiac damage.
  c. Embolization.
  d. Immune complexes.

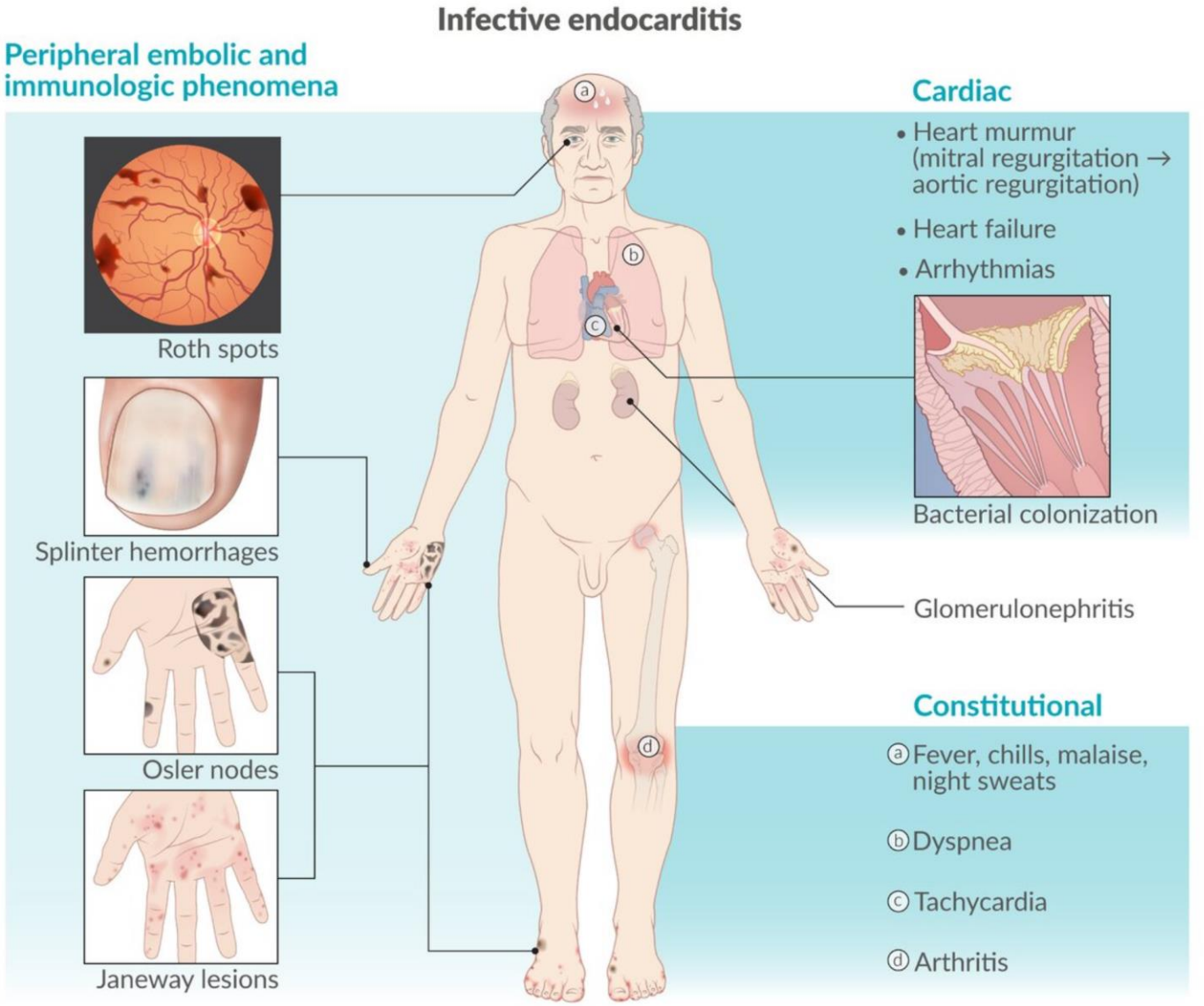
A. GENERAL MANIFESTATIONS: (EXTRA CARDIAC MANIFESTATIONS) :

Face :	◆ **Fever:** (usually low grade & prolonged) : • Any prolonged unexplained fever in cardiac patient is considered and treated as endocarditis until proved otherwise ◆ **Pallor & toxic faces** ◆ **Eye:** • **Subcongunctival hge** • **Roth spots** (area of retinal hge with pale center) • **Sudden blindness:** d2 embolism of central retinal artery.   Roth spots
Upper & lower limbs :	◆ **Pale clubbing:** it occurs with long standing patients ◆ **Osler's nodes:** Small painful red tender raised, in the pulps of the fingers & toes, due to: toxemic endothelial hyperplasia of the capillary . ◆ **Janeway's lesions:** Small painless patches in the palms. ◆ **Splinter hemorrhages:** Linear hemorrhages under nails d2 rupture of capillaries

	◆ **Abnormalities of radial pulse:** a. Tachycardia d2 fever b. Absent pulse: d2 embolization of brachial artery  Janeway's lesions  Osler's nodes:  Splinter hemorrhages:
Kidney :	◆ **Infection:** (immune complex) : it presents as nephritic syndrome, nephrotic syndrome, up to chronic renal failure ◆ **Infarction:** loin pain, hematuria
Spleen :	◆ **Infection:** mild enlarged tender spleen in 80% of case ◆ **Infarction:** stitching pain & splenic rub
CNS :	◆ **Infection:** meningitis, encephalitis, mycotic aneurythm ◆ **Infarction:** embolic hemiplesia , subarachnoid hemorrhage
Lungs :	◆ **Infection:** pneumonia ◆ **Infarction:** due to pulmonary embolism in Rt sided endocarditis

B. CARDIAC MANIFESTATIONS:

- **Development of a new heart murmur or change in a preexisting murmur**
  - **Tricuspid valve regurgitation**
    - Holosystolic murmur that is loudest at the left sternal border
    - Seen in persons who inject drugs, immunocompromised individuals, patients with congenital heart disease, and patients with instrumentation in the right heart (e.g., central venous catheters)
  - **Aortic valve regurgitation:** early diastolic murmur that is loudest at the left 3rd and 4thintercostal spaces and along the left sternal border
  - **Mitral valve regurgitation**: holosystolic murmur that is loudest at the heart's apex and radiates to the left axilla
- **Heart failure:** (e.g., dyspnea, lower limb edema) due to valve insufficiency
- **Arrhythmias:** Suspect a perivalvular abscess in patients with IE who develop a new conduction abnormality (e.g., heart block).



- IE should always be considered as a cause of fever of unknown origin (FUO), especially in the presence of a new heart murmur.
- **"FROM JANE:"** Features of IE include **F**ever, **R**oth spots, **O**sler nodes , **M**urmur , **J**aneway lesions, **A**nemia, **N**ail bed hemorrhage, and **E**mboli.

CLASSIFICATION :

- **IE can be classified by:**
  - Type of affected valve (native vs. prosthetic)
  - Acuity of the infection
  - Location of the infection (left- vs. right-sided).
- Although this is not a definitive classification system, it can help in the approach to management and selection of **empiric antibiotic regimens.**

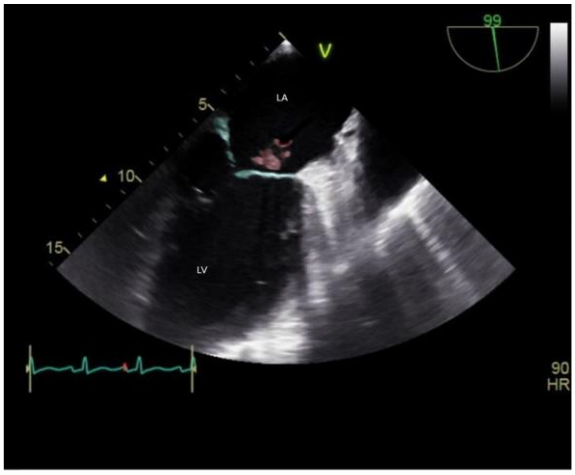
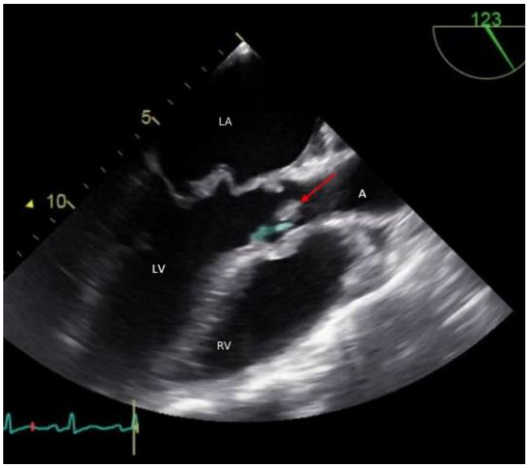
a. Classification by valve type and duration of infection

	Native valve endocarditis		Prosthetic valve endocarditis
	Acute bacterial endocarditis	**Subacute bacterial endocarditis**	
Clinical features	◆ **Acute onset** ◆ Rapid, **fulminant progression** (days to weeks) ◆ Severe constitutional symptoms (e.g., **high fever**)	◆ **Insidious onset** ◆ Slow progression (weeks to months) ◆ Less severe constitutional symptoms (e.g., low-grade fever , malaise, chills, dyspnea, back pain, weight loss)	◆ **Early-onset:** ≤ 1 year after surgery ◆ **Late-onset:** > 1 year after surgery
Main pathogens	◆ Most common: **S. aureus** (associated with large vegetations that can destroy the valves) ◆ Others: group A hemolytic streptococci, S.pneumoniae, N.gonorrhoeae	◆ Viridans streptococci ◆ S. aureus ◆ Enterococci ◆ HACEK	◆ **Early-onset:** Coagulase- negative staphylococci, S. aureus, or gram-negative bacilli (most common) ◆ **Late-onset:** Coagulase- negative staphylococci, S. aureus, Viridans group streptococci (most common)
Affected values	◆ Healthy native valves	◆ Native valves with prior injury or congenital defects	◆ Mechanical valves ◆ Bioprosthetic valves

b. Classification by location of valves involved :

	Right-sided endocarditis	Left-sided endocarditis
Distinguishing clinical features	◆ Bacterial thromboemboli to pulmonary vasculature (e.g., due to lung abscess, pulmonary embolism) ◆ Clinical features of right heart failure (e.g., peripheral pitting edema, abdominal pain from hepatic congestion) ◆ Often associated with venous instrumentation (e.g., IV drug use, indwelling venous catheters)	◆ Emboli to systemic vasculature ◆ Clinical features of left heart failure (e.g., dyspnea, pulmonary edema)
Main pathogens	◆ **S. aureus** (MSSA more often than MRSA) ◆ Streptococci ◆ Coagulase-negative staphylococci	◆ **S. aureus** ◆ Viridans group streptococci ◆ Community-acquired enterococci
Affected valves	◆ Tricuspid ◆ Pulmonic	◆ Mitral ◆ Aortic

iNVESTiGATiONS :

Blood picture:	◆ Anemia. ◆ Leucocytosis ◆ ↑ ESR& CRP ◆ Acute phase reactants.
Blood Culture:	**" MOST iMPORTANT LAB. iNVESTiGATiON"** ◆ It is positive in most of the cases. ◆ At least **3** blood samples are taken during fever. The results are observed from **3** days up to **3** weeks. ◆ Samples are cultured under **aerobic & anerobic conditions.** ◆ If the patient was taking **penicillin, penicillinase enzyme** should be added. ◆ **Culture-negative endocarditis may be due to:** • Prior use of antibiotics. • Infection with other organisms, e.g.: hacek, fungi, rickettsiae.
Urine analysis:	◆ **Hematuria:** Gross or microscopic. ◆ **Proteinuria:** and casts.
Echo :	**A. TRANS-THORACiC ECHO:** ◆ The underlying cardiac lesion. ◆ Any heart complication. ◆ **VEGETATIONS**, however, small vegetations may be missed. **B. TRANS-OESOPHAGEAL ECHO: " MOST iMPORTANT iNVESTiGATiON "** ◆ Detects **small vegetations** missed by trans-thoracic echo.  **Bacterial endocarditis of mitral valve**  **Aortic valve vegetation**
Immunologic tests:	◆ **Circulating immune complexes:** may be detected. ◆ **Rheumatoid factor:** may be positive.

DiAGNOSiS : "MODiFiED DUKE CRiTERiA "

Major criteria	◆ **Positive blood cultures (any of the following):** • Typical organisms from two separate samples: a. Viridans streptococci b. S.aureus c. S.gallolyticus d. HACEK group e. Community-acquired enterococci without a primary focus • Persistently positive cultures with microorganisms consistent with endocarditis : a. ≥2 positive blood cultures drawn >12 hours apart. b. All of 3 or a majority of ≥ 4 separate blood cultures. • One positive culture for coxiella burnetii or an anti-phase 1 IgG titer of ≥ 1:1800 ◆ **Characteristic echocardiographic findings of IE.** ◆ **New valvular regurgitation.**
Minor criteria	I) Predisposing conditions (i.e. predisposing heart condition at high or intermediate risk of IE or PWIDs) II) Fever defined as temperature >38°C III) Embolic vascular dissemination (including those asymptomatic detected by imaging only): • Major systemic and pulmonary emboli/infarcts and abscesses • Haematogenous osteoarticular septic complications (i.e. spondylodiscitis) • Mycotic aneurysms • Intracranial ischemic/hemorrhagic lesions • Conjunctival hemorrhages • Janeway's lesions IV) Immunological phenomena: • Glomerulonephritis • Osler nodes • Roth spots • Rheumatoid factor V) Microbiological evidence: • +ve blood culture but not meet major criterion as noted above • Serological evidence of active infection with organism consistent with IE
Pathological Criteria	◆ Microorganisms demonstrated by tissue culture or histology ◆ Characteristic histologic features of active endocarditis

Definite	Possible	Rejected
◆ 2 major criteria ◆ 1 major and 3 minor criteria ◆ 5 minor criteria ◆ Pathology/histology findings	◆ 1 major and 1 minor criteria ◆ 3 minor criteria	◆ Does not meet criteria for definite or possible at admission with or without a firm alternative diagnosis

TREATMENT

A. Antibiotics :

Empiric antibiotic therapy :

- The goal is to provide broad-spectrum coverage for potential bacterial causes of IE (including multidrug-resistant organisms) until blood culture results are available.
- Common empiric antibiotic regimens
  - **Native valve endocarditis:** vancomycin PLUS a beta-lactam (eg. ceftriaxone, cefepime)
  - **Prosthetic valve endocarditis:** Add gentamicin PLUS rifampin to vancomycin PLUS a beta-lactam (if ≤ 1 year after placement).

VALVE TYPE	CLINICAL PRESENTATION	COMMON REGIMEN
NATIVE VALVE ENDOCARDITIS	Acute bacterial endocarditis (days)	✦ **Vancomycin** ✦ PLUS cefepime
	Subacute bacterial endocarditis (weeks)	✦ **Vancomycin** ✦ PLUS ampicillin-sulbactam
PROSTHETIC VALVE ENDOCARDITIS	≤1 year after valve placement	✦ **Vancomycin** ✦ PLUS gentamicin ✦ PLUS rifampin ✦ PLUS cefepime
	> 1 year after valve placement	✦ **Vancomycin** ✦ PLUS ceftriaxone

Targeted antibiotic therapy :

- Based on **culture** and **sensitivity results** is recommended for all patients with IE.

TARGETED ANTIMICROBIAL THERAPY FOR INFECTIVE ENDOCARDITIS		
Organism	Native valve endocarditis (common regimens)	Prosthetic valve endocarditis (common regimens)
Methicillin-susceptible staphylococci (e.g., MSSA)	✦ **One of the following:** 1. Oxacillin 2. Nafcillin 3. Cefazolin	✦ **One of the following beta-lactams:** 1. Nafcillin 2. Oxacillin 3. PLUS rifampin 4. PLUS gentamicin
Methicillin-resistant staphylococci (e.g., MRSA)	✦ Vancomycin	✦ Vancomycin ✦ PLUS rifampin ✦ PLUS gentamicin
Viridans group streptococci, S. gallolyticus	✦ **One of the following:** Aqueous crystalline penicillin G Ceftriaxone ✦ **PLUS** (if resistant to penicillin) gentamicin ✦ **Alternative:** vancomycin	
Enterococcus spp. (penicillin-sensitive)	✦ **One of the following:** Ampicillin Aqueous penicillin G ✦ **PLUS** gentamicin ✦ OR double beta-lactam therapy: ampicillin PLUS ceftriaxone	
Enterococcus spp. (penicillin-resistant)	✦ Vancomycin ✦ PLUS gentamicin	
HACEK	✦ **First-line:** ceftriaxone ✦ **OR one of the following:** Ampicillin Ciprofloxacin	

- **Duration of therapy:** variable depending on many factors, e.g., drug regimen, affected valve; can be 2–6 weeks or longer after the first sterile blood culture

- **Blood culture-negative endocarditis:** empiric therapy until additional investigations (e.g., serology) yield results

B. Surgical Treatment :

Indications	◆ Prosthetic valve endocarditis ◆ Valve dysfunction leading to heart failure ◆ Uncontrolled infection: eg. enlarging vegetation, persistent bacteremia ◆ Perivalvular extension or complications eg, abscess, pedaneurysm, fistula & heart block ◆ Fungal endocarditis ◆ **High embolic risk:** e, mobile vegetation >10 mm, recurrent embolium
Surgical options	◆ **Valve replacement or valve repair**.

PREVENTION: ENDOCARDITIS PROPHYLAXIS

- **Prophylaxis** is indicated prior to certain procedures with a high risk of bacteremia in patients with high-risk cardiac features.

- **Cardiac risk factors requiring IE prophylaxis** (for procedures that may cause bacteremia)
  - Presence of prosthetic cardiac valve or material
  - History of endocarditis
  - Certain types of congenital heart disease (CHD), e.g., unrepaired cyanotic CHD, repaired CHD (within 6 months of repair), repaired CHD with residual post-operative shunt or regurgitation

- **Procedures requiring IE prophylaxis in patients at risk for IE :**
  - Some dental procedures including tooth extraction and routine dental cleaning
  - Any invasive procedure involving infected tissue (e.g., abscess drainage)
  - Placement of a CIED [40][46]
  - Surgical placement of prosthetic cardiac or intravascular material (e.g., heart valve, intravascular graft) [46]

- **Common regimens** (usually administered 30–60 minutes prior to the procedure):

Prior to dental procedures	◆ No penicillin allergy: amoxicillin OR ampicillin OR cefazolin ◆ Penicillin allergy: a macrolide (e.g., azithromycin) OR doxycycline
Prior to CIED placement	◆ Cefazolin.

- IE prophylaxis is not routinely recommended prior to nondental procedures (including respiratory, skin, musculoskeletal, gastrointestinal, and genitourinary procedures) unless infected tissue is present.

DIFFERENTIAL DIAGNOSIS :

a. **RHEUMATIC FEVER.**
b. **Causes of:** fever in a cardiac patient.
c. **Causes of:** embolization.
d. **Causes of:** GN.
e. **Causes of:** tender splenomegaly.
f. **Precipitating factors of:** heart failure.

	Rheumatic Fever	Infective Endocarditis
Fleeting arthritis	Yes	No
Erythema marginatum	Yes	No
Petechiae	No	Yes
Hematuria	No	Yes
Clubbing	No	Yes
Splenic enlargement	No	Yes
Embolization	No	Yes
Culture	-ve	+ve

DISEASES OF THE PERICARDIUM

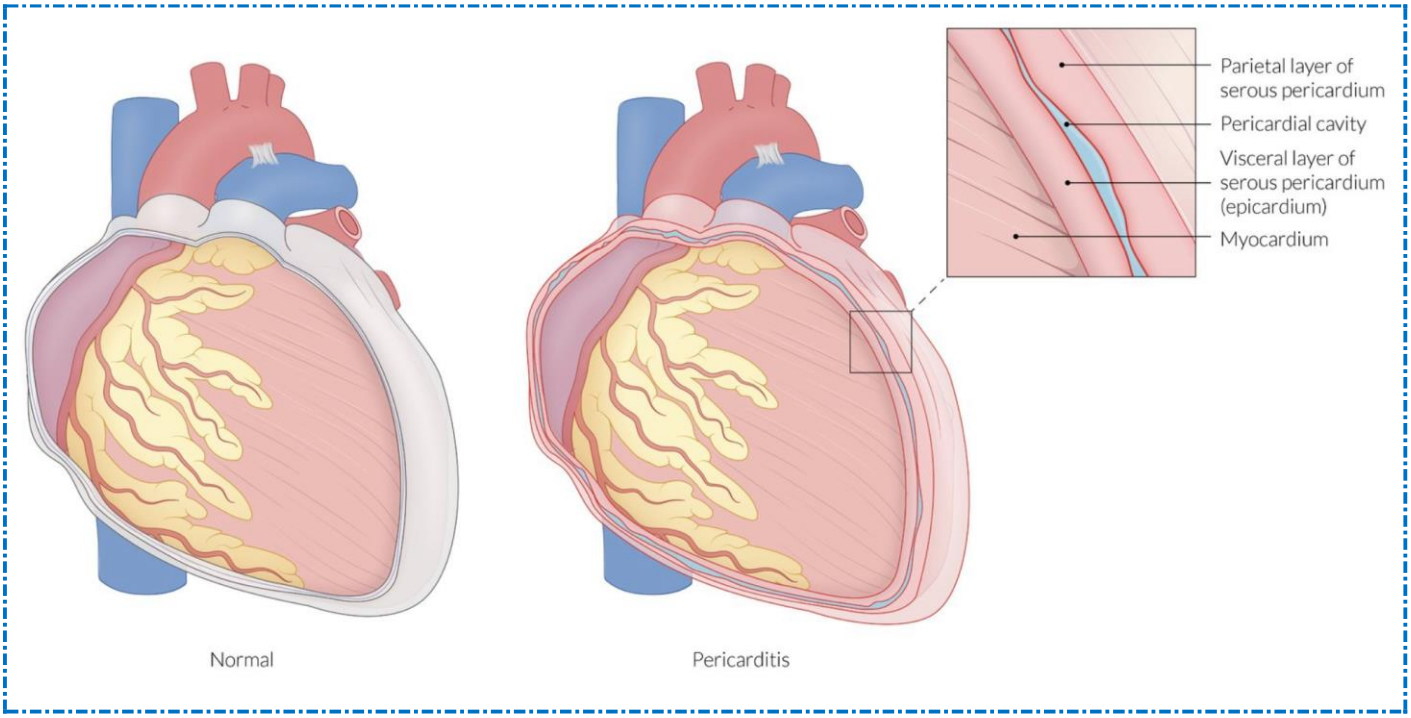
EXAMPLES :

1. Dry pericarditis.
2. Constrictive pericarditis.
3. Pericardial effusion.
4. Adhesive pericarditis.

A. Dry Pericarditis :

DEFINITIONS:

Acute pericarditis	◆ Inflammation of **pericardium** that either occurs as an isolated process or with concurrent myocarditis (myo-pericarditis)
Relapsing/ recurrent pericarditis	◆ Recurrence of symptoms after a symptom free period of 4-6 weeks
Chronic pericarditis	◆ Inflammation of **pericardium** **that lasts > 3 months.**
Peri myocarditis	◆ Condition predominantly affecting myocardium with pericardial involvement.



ETiOLOGY :

- **Idiopathic.**
- **Infectious:**
  - Most commonly viral (e.g., coxsackie B virus).
  - Bacterial (e.g., Staphylococcus spp., Streptococcus spp., or M. tuberculosis)
  - Fungal
  - Toxoplasmosis
- **Myocardial infarction:**
  - Postinfarction fibrinous pericarditis: within 1–3 days as an immediate reaction
  - Dressler syndrome: weeks to months after an acute myocardial infarction
- **Postoperative** (postpericardiotomy syndrome): due to blunt or sharp trauma to the pericardium
- **Uremia:** e.g., due to acute or chronic renal failure
- **Radiation**
  - Exudative pericarditis: develops acutely during or after radiation therapy.
  - Constrictive pericarditis: develops several years after radiation therapy.
- **Neoplasms** (e.g., Hodgkin lymphoma)
- **Autoimmune connective tissue diseases** (e.g., rheumatoid arthritis, systemic lupus, scleroderma)
- **Trauma.**

CLiNiCAL PiCTURE :

Symptoms

A. GENERAL : (FAHM).

B. LOCAL : " CHEST PAIN " :

Due To :	Inflammation of both pericardium & adjacent pleura .
Site :	Pericordial & radiate to shoulders.
Duration :	Continuous.
Character :	Severe Stretching,.
Increase by :	Inspiration & movements of chest wall.
Decrease by:	Sitting up & leaning forward.
Ass. Symptoms :	Pleuritic chest pain

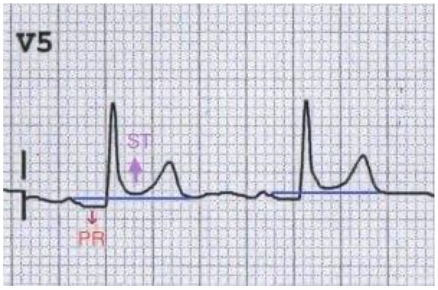
<table>
<tr><td>Signs</td><td>
A. GENERAL : Fever.
B. LOCAL : Pericardial Rub :
<table>
<tr><td>Due To :</td><td>Friction ( ) 2 layers of inflamed pericardium.</td></tr>
<tr><td>Site :</td><td>Best heard over the Base of heart & Bare area.</td></tr>
<tr><td>Timing :</td><td>Systole = Diastole.</td></tr>
<tr><td>Character :</td><td>Stretching.</td></tr>
<tr><td>Increase by :</td><td>Pressing the stethoscope against the chest wall.</td></tr>
</table>
</td></tr>
<tr><td>Complications</td><td>

Pericardial effusion.
Constrictive pericarditis.

</td></tr>
</table>

INVESTIGATIONS :

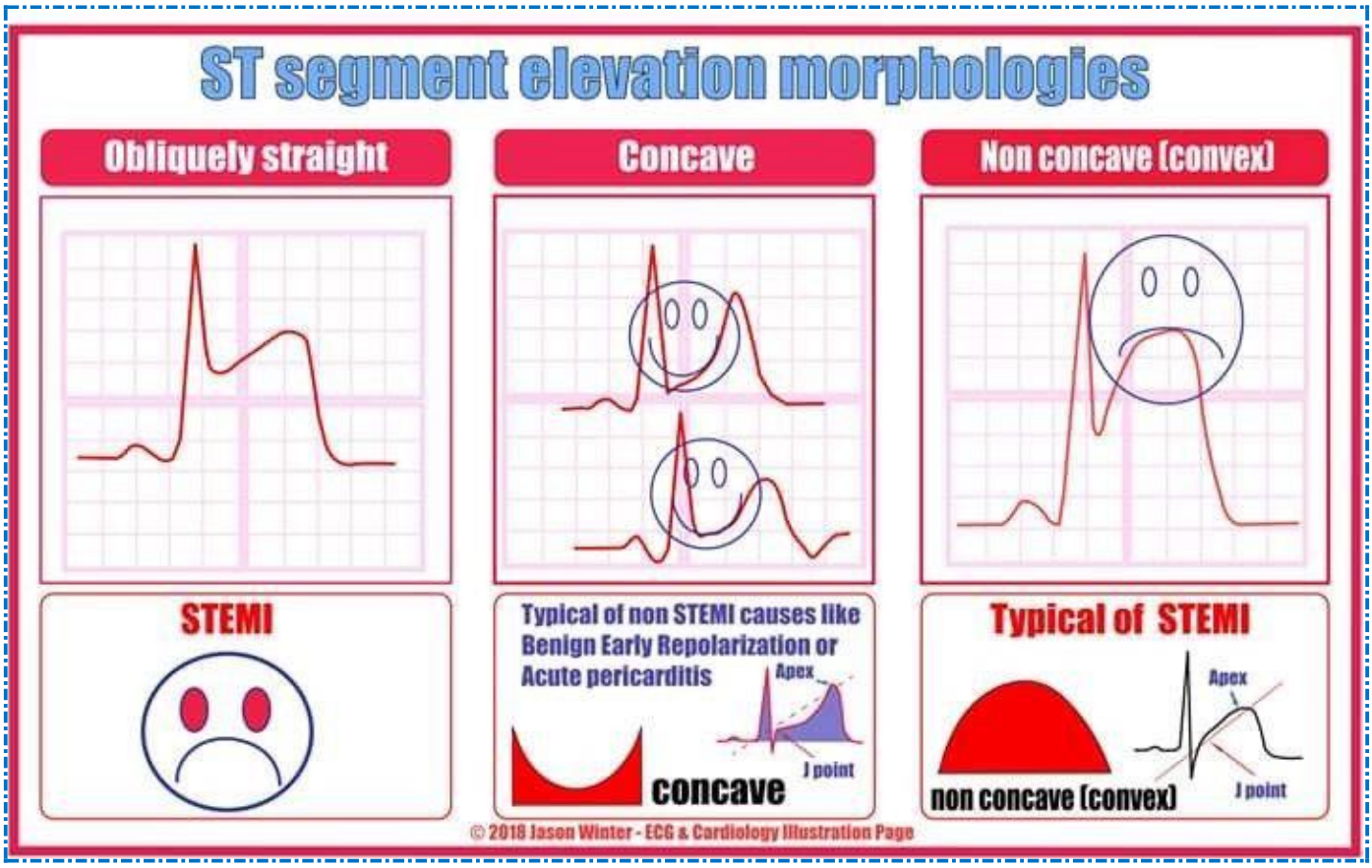
- ECG:
  - Elevated ST segment.
  - Flat or inverted T waves.
- Investigations of the cause.



DIFFERENTIAL DIAGNOSIS :

- DD of the causes of **dry Pericarditis.**
- DD of the causes of **acute chest pain.**
- DD of the causes of **elevated ST segment :**

Items	Acute pericarditis	AMI	Prinzmetal angina
Elevated ST	Concave upwards	Convex upwards	Flat
Elevated ST	In all leads	In some leads	In some leads
Pathological Q wave	Absent	Present	Absent
Cardiac enzymes	Normal	Elevated	Normal



TREATMENT :

- **GOALS OF THERAPY :**
  a. Relief of pain
  b. Treat the underlying disease process
  c. Prevent Cardiac tamponade and constrictive pericarditis.

- **DRUGS :**

NSAIDS	◆ **ASA:** 650 q3-4hr. for 1-2 wk after complete resolution of Sx ◆ **Ibuprofen:** 300-600 q 6-8 hrs x 1-4days.
Steroids	◆ if **no** response after 48hr to NSAID ◆ use concurrent with NSAID
Colchicine	◆ Useful in **chronic cases** +/- NSAID ◆ Useful in **recurrent pericarditis.**
Treatment of cause	◆ e.g :antibiotic or antifungal medication ◆ Most viral infections are **self-limited.**

B. Pericardial Effusion :

DEFINITION:

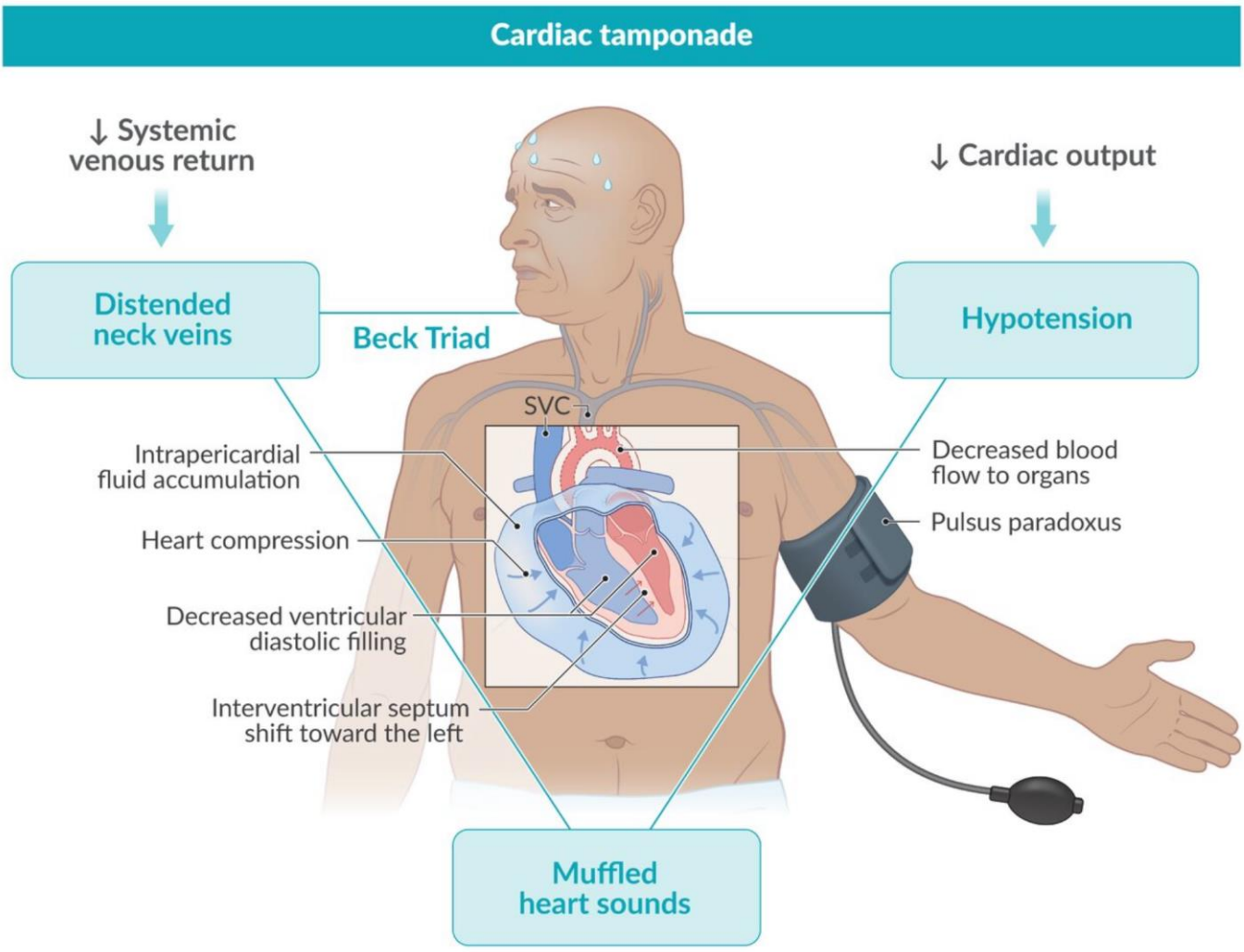
Pericardial effusion	◆ Accumulation of fluid in the pericardial space between parietal and visceral pericardium.
Cardiac tamponade	◆ A patho-physiological process whereby elevated intrapericardial pressure from a pericardial effusion causes compression of the heart (especially right ventricle).

ETIOLOGY :

Sero-pericardium: "exudate"	✦ **It occurs in all conditions that cause dry pericarditis especially:** ◆ **TB: "Most common cause of pericardial effusion in developing countries & in developed countries idiopathic".** ◆ Malignancy. ◆ Viral. ◆ Rheumatic. ✦ **Hemorrhagic pericarditis is a severe form of seropericardium with excessive RB :** ◆ TB. ◆ Myocardial infarction. ◆ Renal failure. ◆ Malignancy.
Pyo-pericardium:	• Severe inflammation of pericardium due to: Infection by pyogenic organisms, e.g. Staph. & Strept
Hydro-pericardium: "transudate"	• Heart failure. • Hepatic failure. • Hypo-proteinemia e.g. Nephrotic syndrome. • Hypo-thyroidism.
Hemo-pericardium:	**The pericardial fluid is frank blood :** • **Rupture of heart:** trauma or infarction • **Rupture of coronaries:** trauma during catheterization • **Rupture of** dissecting aneurysm of the aorta. • **Hemorrhagic blood diseases.**
Chylo-pericardium:	• **Obstruction** of thoracic duct • **Rupture** of thoracic duct

PATHOPHYSIOLOGY :

- **MANIFESTATIONS DEPEND ON :**
  - Amount of fluid.
  - Rate of accumulation.
- **CARDIAC TAMPONADE (SEVERE COMPRESSION) OCCUR IN SEVERE CASES WHEN :**
  - **Amount of fluid:** fills the pericardial reserve volume.
  - **Rate of accumulation** exceeds the rate of stretch of the parietal pericardium.
- **AMOUNT OF FLUID NECESSARY TO PRODUCE CARDIAC TAMPONADE MAY BE :**
  - **As small as 200 ml** when the fluid collects rapidly.
  - **Over 2000 ml** when it collects slowly (allowing adaptation of pericardial sac).
- **CARDIAC TAMPONADE CAUSES ↓CARDIAC RELAXATION & ↓CARDIAC FILLING :**
  - **Systemic congestion:** due to stagnation of blood behind the heart.
  - **Low CO.**



CLINICAL PICTURE:

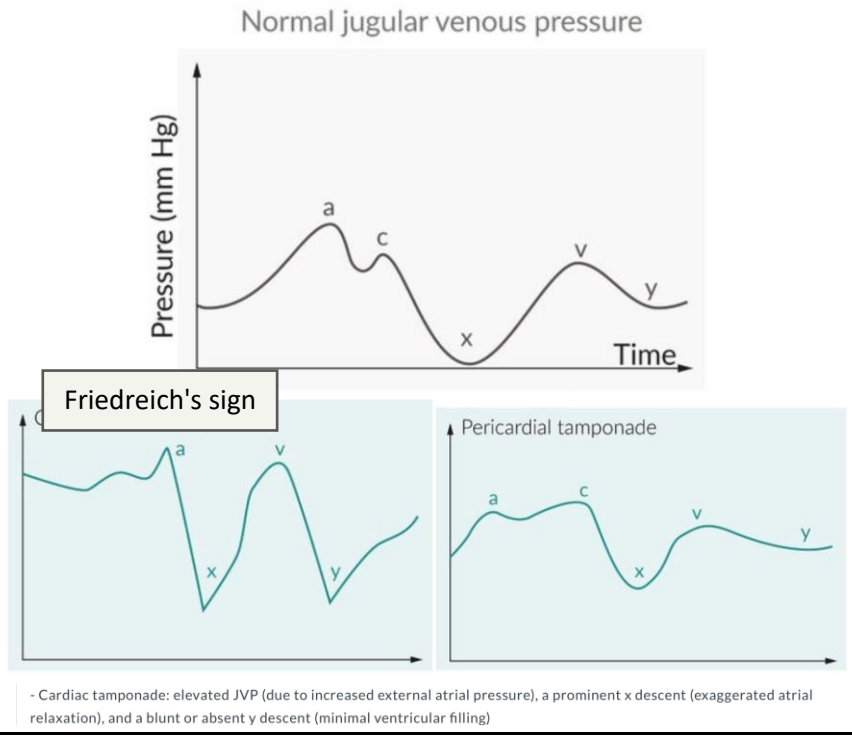
A. Symptoms

1. **Symptoms of** Systemic congestion.
2. **Symptoms of** Low CO.
3. **Pain:**
   - Dull aching pain: due to stretch of the parietal pericardium.
4. **Pressures symptoms:**
   - **On lung:** Dyspnea improves by sitting up - leaning forwards & cough.
   - **On esophagus:** Dysphagia
   - **On left recurrent laryngeal nerve:** hoarseness of voice.
5. **Symptoms of the cause:** **e.g. TB toxemia.**

B. General Signs

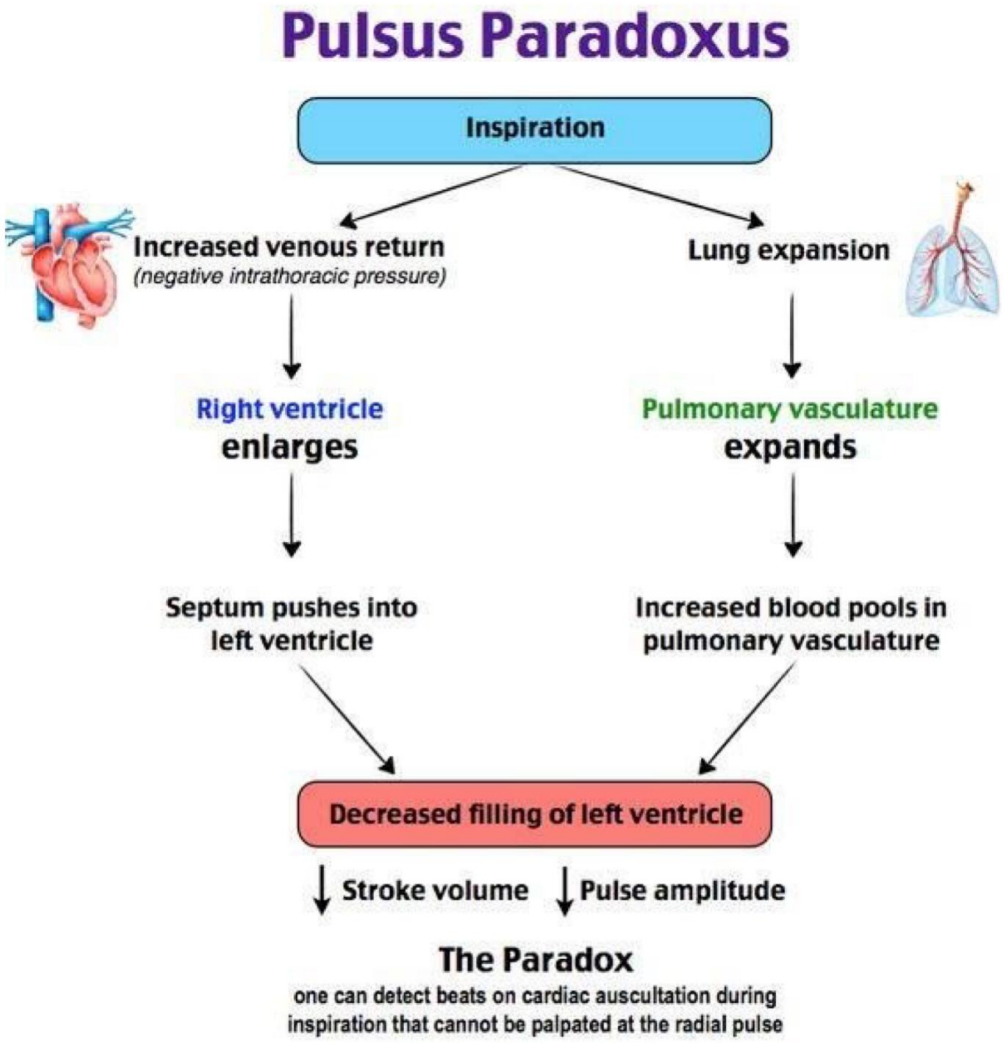
1. **Signs of systemic congestion:**

Neck vein	• Congested neck veins • **KUSSMAUL'S SIGN:** inspiratory filling of neck veins due to failure of heart to accept ↑↑ VR during inspiration • **FRIEDREICH'S SIGN:** rapid deep Y wave
Liver	• Enlarged tender liver.
Ascites precox	**= ASCITES BEFORE EDEMA LL, DUE TO:** • Kinking of hepatic vein • Obstruction of lymphatics passing through central tendon of diaphragm causes accumulation of lymph in peritoneum



- Cardiac tamponade: elevated JVP (due to increased external atrial pressure), a prominent x descent (exaggerated atrial relaxation), and a blunt or absent y descent (minimal ventricular filling)

2. **Signs of LCOP:** cold hand, pallor, low systemic BP, weak pulse.
3. **Prayer's position.**
4. **Pulsus paradoxus:** exaggerated inspiratory fall in BP (>10mmHg)
   - **Normally there is slight fall in BP during inspiration (<10 mmg)**
   - **Notice that pulsus paradoxicus is an exaggerated of the normal & not a paradox**



CAUSES OF PULSUS PARADOXICUS:

1- **A**cute severe asthma
2- **P**ericardial effusion
3- **C**onstrictive pericarditis
4- **C**OPD
5- Restrictive **C**ardiomyopathy

In pericardial effusion During inspiration:

- Increase VR but the compression on the Rt side of the heart prevents the proper filling of ventricle ---- no increase in COP
- Expansion of the lung: taking part of COP of the Rt ventricle

So the net result is massive decrease in COP

C. Local signs	**A] Inspection & palpation:** ◆ **Apical pulsations:** weak or absent. ◆ **Precordial bulge:** may occur in children. **B] Percussion:** ◆ **Dullness** to the right border of the sternum. ◆ **Dullness** outside the apex. ◆ **Dullness** on the pulmonary area (shifting dullness). ◆ **BARE AREA:** ↑ size + stony dullness. ◆ **EWART'S SIGN:** dullness over the left lung base due to its compression collapse by the effusion. **C] Auscultation:** ◆ **Muffled** heart sounds.

COMPLICATIONS :

A. CARDIAC TAMPONADE : severe compression causing ↓, BP, ↑JVP, HF or Shock.

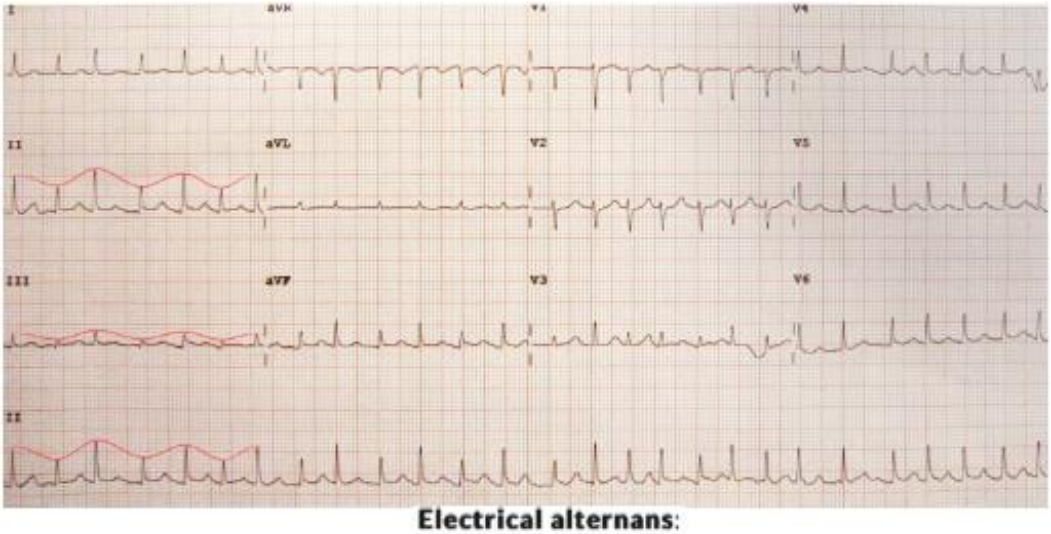
Beck's Triad: ↓ BP, ↑JVP, Muffled heart sounds.

B. CONSTRICTIVE PERICARDITIS.

INVESTIGATIONS:

A. ECG:

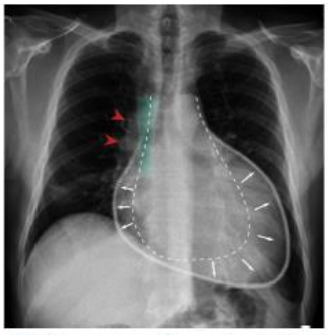
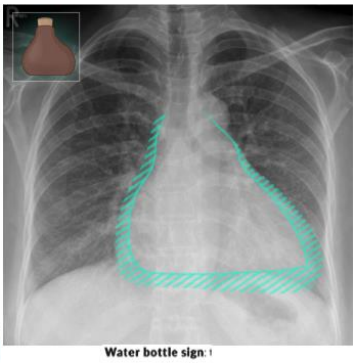
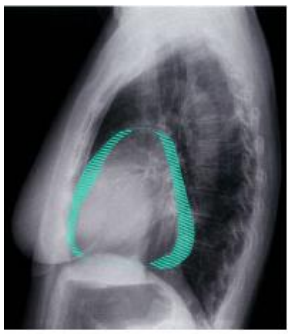
INDICATION	◆ All patients with suspected pericardial effusion ◆ Used to rule out an ischemic cause
FINDING IN :	
◆ **PERICARDIAL EFFUSION**	◆ Normal in smaller effusions ◆ a Low voltage complexes and **electrical alternans** in larger effusions
◆ **CARDIAC TAMPONADE**	◆ Sinus tachycardia ◆ Low voltage QRS complexes ◆ **Electrical alternans:** consecutive QRS complexes that alternate in height due to the swinging motion of the heart when surrounded by large amounts of pericardial fluid ◆ Pulseless electrical activity (PEA) in cardiac arrest.



Electrical alternans:

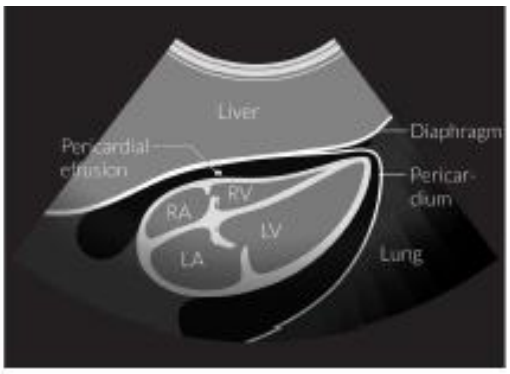
B. CXR :

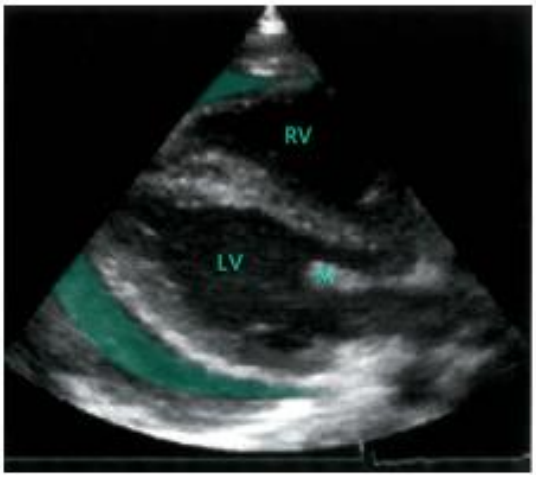
- Not for diagnose pericardial effusion but to exclude other causes of dyspnea.
- 250 ml of pericardial fluid must be present before an effusion is visible on CXR.

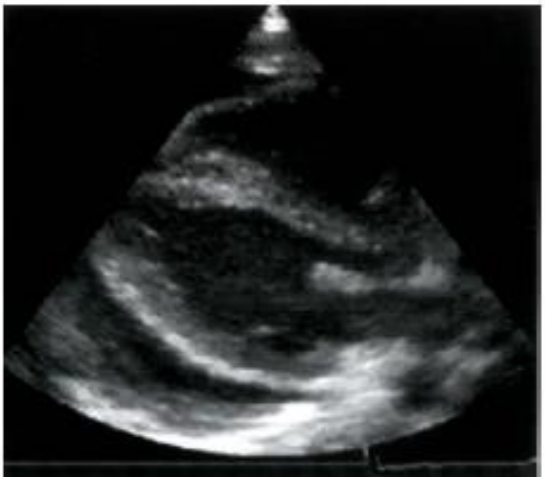
PA VIEW FINDINGS	• Normal in small effusions • Enlarged cardiac silhouette and clear lungs may be seen in moderate effusions • Double countour of the cardiac borders. • Pulmonary oligemia • **Water bottle sign:** the radiographic sign of a large pericardial effusion in which the cardiac silhouette resembles a bottle  Double countour of the cardiac borders.  Water bottle sign:
LATERAL VIEW FINDINGS	• **Posterior inferior bulge sign:** a change in the silhouette of the heart due to a pericardial effusion that collects in the posterior inferior pericardiac recess and expands the pericardium  Lateral view findings

C. ECHOCARDIOGRAPHY :

- The **most sensitive test** for detecting the presence of pericardial effusion & its severity.







D. CARDIAC CATHETERIZATION & ANGIOCARDIOGRAPHY:

- Reveals a gap between the edge of the heart which contains the catheter and the peripheral cardiac shadow.
- Angiocardiography demonstrates the actual size of the heart inside the effusion.

E. OTHER INVESTIGATIONS FOR CAUSE:

- **Pericardiocentesis.**
- **Pericardial biopsy:** e.g. for TB or malignant.
- **Serology:** for SLE.
- **Urea & Creatinine:** for Renal failure.

Pericardiocentesis: (Aspiration of effusion)

Indications

A. DIAGNOSTIC: "May be needed for diagnosis of the cause by analysis of the pericardial fluid"

a. Features of exudate & transudate effusions:

Items	Transudate	Exudate
Proteins Fluid protein / serum protein	<3 gm % <0.5	> 3 gm % >0.5
Specific gravity	< 1016	> 1016
LDH Fluid LDH / serum LDH	< 200 IU / L <0.6	> 200 IU / L >0.6
Cells (WBCs)	< 1000 / cmm	> 1000/cmm

NB Characteristics of TB effusion:

1. **Exudate:** rich in Lymphocytes & RBCs.
2. **TB can be detected by:**
   - Staining: ZN stain.
   - Culture: Lowenstein-Jensen medium or BACTEC
   - Animal innoculation: Guinae pig.
   - PCR for TB DNA.
   - Adenosine deaminase (ADA): ↑Activity of this enzyme.

b. Features of Pyopericardium:

- The fluid is purulent & contains many pus cells.

c. Features of Hemopericardium:

- The fluid is bloody & contains many RBCs.

d. Features of Chylopericardium:

- The fluid is milky white & contains many fat, clears on addition of ether & stains orange with: Sudan III.

B. THERAPEUTIC: **See later.**

C/I

- Bleeding tendency.
- Uncooperative patient.

Sites of aspiration

- From outside the apex.
- From the bare area.

Complications of aspiration

- Infection.
- Arrhythmias.
- Trauma: myocardial rupture, pneumothorax.

DIFFERENTIAL DIAGNOSIS :

1. **Congestive heart failure:**
   - Different types of dyspnea (exertional, orthopnea, PND).
   - Oedema of LLs before ascites.
   - No pulsus paradoxus.
   - Heart is enlarged & there may be murmur & gallop.
2. **Constrictive pericarditis:**
   - Prominent y descent.
   - Pericardial calcification: on Chest x-ray.
3. **Restrictive cardiomyopathy.**
4. **Liver cirrhosis & nephrotic syndrome.**
5. **DD of the cause of effusion.**

TREATMENT :

1. **Treatment of the cause: e.g. TB:**
   - **Anti-TB drugs.**
   - **Corticosteroids:** to ↓inflammation & prevent constrictive pericarditis.
2. **Pericardiocentesis :**

   Withdrawal of only 50 to 75 ml eliminates tamponade (compression) in most cases
   - **In cardiac tamponade :** to relieve dyspnea & compression.
   - **In suppurative effusion :** to drain pus & inject antibiotics.
   - **In malignant effusion :** to inject cytotoxic drugs.
3. **Pericardiodesis: (Pericardial sclerosis with tetracycline)**
   - **In malignant effusion:** to prevent re-accumulation.
4. **Pericardiectomy or pericardio-pleural window:**
   - **For massive & recurrent effusion** not responding to medical ttt.

C. Constrictive Pericarditis :

DEFINITIONS:

Constrictive pericarditis	◆ Characterized by compromised cardiac function caused by a thickened, rigid, and fibrous pericardium secondary to acute pericarditis.
Effusive-constrictive pericarditis	◆ Pericardial effusion occurs in addition to a thickened pericardium, which can lead to **tamponade**

ETIOLOGY: 4 i

- idiopathic.
- INFECTIONS:
  - **Tuberculosis.**
  - Bacterial, Viral, Fungal.
- immunologic: RA (but not Rheumatic Fever).
- irradiation.

- Most common cause in developed countries is idiopathic and post viral infections.
- Most common cause in developing countries in TB.

PATHOLOGY:

- **Dense fibrous tissue** adheres the 2 layers of the pericardium together and constricts the heart.
- Frequently it undergoes calcification.

PATHOPHYSIOLOGY:

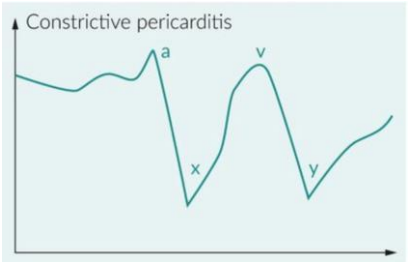
- The rigid pericardium interferes with ventricular filling in late diastole.
- Ventricular filling is not reduced in early diastole, but it gets reduced abruptly when the elastic limit of the pericardium is reached.
- This is in contrast to pericardial effusion where ventricular filling is reduced all through diastole.

CLINICAL PICTURE:

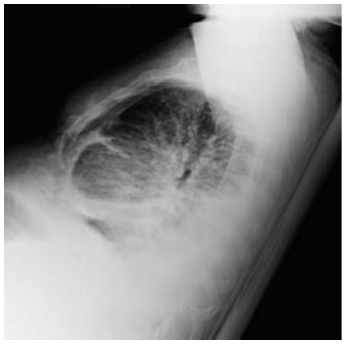
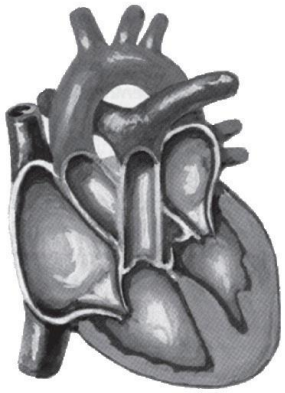
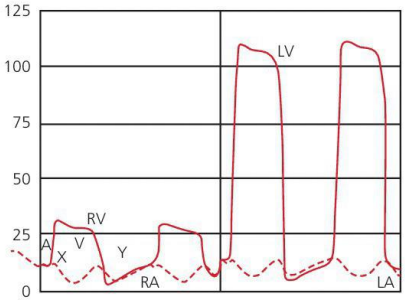
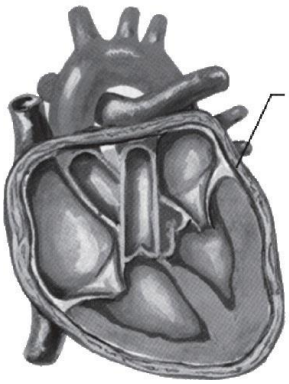
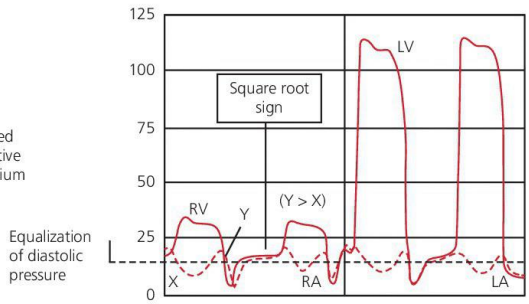
A. SYMPTOMS :

a. Symptoms of Systemic congestion.
b. Symptoms of Low CO.
c. Symptoms of the cause: e.g. TB toxemia.

B. SIGNS :

Signs of CAUSE:	◆ e.g. TB.
General	1. **Decubitus:** No special decubitus. 2. **Signs of:** Systemic congestion: ◆ **Neck veins:** • Congested pulsating neck veins. • **Kussmaul's sign:** Due to failure of the right side of the heart to accept the increased venous return during inspiration. • **Freidreich's sign:** Prominent descent due to rapid emptying of the congested neck veins in a short time after opening of the tricuspid valve.  ◆ **Enlarged tender liver.** ◆ **Ascites before oedema of lower limbs (ascites precox).** 3. **Signs of:** ◆ Low CO: ◆ Low SBP, weak pulse, pallor. ◆ **Pulsus Paradoxus:** in 30 % of the cases. 4. **AF:** in 30 % of the cases.
Cardiac	1. **Precordial examination:** ◆ Apical pulsations: weak or absent. 2. **Auscultation:** ◆ **Muffled heart sounds.** ◆ **Pericardial Knock:** A sharp diastolic sound due to the sudden halting of the relaxing ventricles by the rigid pericardium.

iNVESTiGATiONS :

ECG	◆ Low voltage. ◆ Diffuse nonspecific ST - T changes (Depressed ST, inverted or flat T). ◆ **AF:** in 30 % of cases.
CXR	◆ Cardiac shadow is small. ◆ **Calcification of the pericardium** is diagnostic. ◆ Diminished cardiac pulsations under the screen. ◆ Pulmonary oligemia. 
Echocardiography	◆ Pericardial thickening.
Cardiac catheterization & angiocardiography:	◆ **Square root sign: "Early diastolic dip followed by a plateau"** in: RV & LV ventricular pressure tracings. Normal   Atrial contraction reduces atrial volume and increases atrial pressure (A wave). Ventricular contraction causes initial small C wave and as atrial relaxation ensues, atrial enlargement occurs with pressure decrease (X descent). Passive atrial filling causes V wave until AV valves open and pressure rapidly drops (Y descent) as ventricles relax. Following ventricular systole, an active and passive filling phase follows—pressure lowest in active phase. Constrictive pericarditis   High atrial pressure when the AV valves open results in rapid early filling (rapid Y descent) until filling abruptly stops (square root sign). There is equalization of late diastolic pressures. The RV diastolic is usually > ⅓ RV systolic pressure.
CT & MRI	◆ **Most important investigation.** ◆ Very sensitive in detecting: Pericardial thickening.

DIFFERENTIAL DIAGNOSIS :

- Congestive heart failure.
- Pericardial effusion.
- Restrictive Cardiomyopathy.
- Liver cirrhosis & nephrotic syndrome.
- DD of the cause of constrictive pericarditis.

TREATMENT :

- **Pericardiectomy :** Is the definitive therapy.
- **Treatment of the cause:** Anti - TB drugs **(before & after pericardiectomy).**
- **Medical treatment for the edema and AF.**
- **Diuretics.**

SYSTEMIC HYPERTENSION

DEFINITION OF HYPERTENSION:

- **Based on blood pressure measurement strategy:**

SBP/DBP	Clinic	SMBP	Daytime ABPM	Nighttime ABPM	24-hour ABPM
ACC/AHA Guidelines	≥ 130/80	≥ 130/80	≥ 130/80	≥ 110/65	≥ 125/75
ESC/ESH Guidelines	≥ 140/90	≥ 135/85	≥ 135/85	≥ 120/70	≥ 130/80

- **SBP:** systolic blood pressure
- **DBP:** diastolic blood pressure
- **ACC/AHA:** American College of Cardiology/American Heart Association.
- **ESC/ESH:** European Society of Cardiology/European Society of Hypertension
- **ABPM:** ambulatory blood pressure monitoring
- **SMBP:** self-measured blood pressure.

CLASSIFICATION:

Parameter	ACC/AHA	ESC/ESH
Definition of hypertension	≥ 130/80	≥ 140/90
Normal Blood pressure	✦ **Normal:** <120/80 ✦ **Elevated** 120-129/<80	✦ **Optimal:** <120/80 ✦ **Normal:** 120-129/80-84 ✦ **High normal:** 130-139/85-89
Hypertension Stages	✦ **Grade 1:** 130-139/80-89 ✦ **Grade 2:** ≥140/90	✦ **Grade 1**: 140-159/90-99 ✦ **Grade 2:** 160-179/100-109 ✦ **Grade 3:** ≥ 180/110
Age specific BP targets	✦ **≤ 65 y:** <130/80	✦ **< 65 y:** <130/80
	✦ **≥ 65 y:** <130/80	✦ **≥ 65 y:** <140/80

European Guidelines are universally more acceptable

NB:

- Diagnosis should not be made on a single office visit.
- Usually **2-3 Office visits** at 1-4-week intervals (depending on BP level)are required to confirm the diagnosis of hypertension.
- Diagnosis might be made on a **single Office visit,** if BP is ≥180/110 mm Hg, and there is evidence of cardiovascular disease (CVD).

- Diagnosis is based on: **Office** (Clinic) measurement.
- If possible, diagnosis is confirmed by out-of-office measurement **(ABPM &/or HBPM).**

SUBTYPES AND VARIANTS:

1. White coat hypertension	2. Masked hypertension
✦ Elevated BP readings in a clinical setting (caused by anxiety) but normal readings when measured elsewhere. ✦ **Therefore:** Ambulatory BP monitoring is required).	✦ Normal BP readings in a clinical setting but consistently elevated readings when measured elsewhere. ✦ **Therefore:** Ambulatory BP monitoring is required).

NB: INTERPRETATION OF BLOOD PRESSURE READING :

	In-office blood pressure	Out of office blood pressure
Sustained HTN	Elevated	Elevated
White coat HTN (Isolated clinic HTN)	Elevated	Normal
Masked HTN (Isolated Ambulatory HTN)	Normal	Elevated

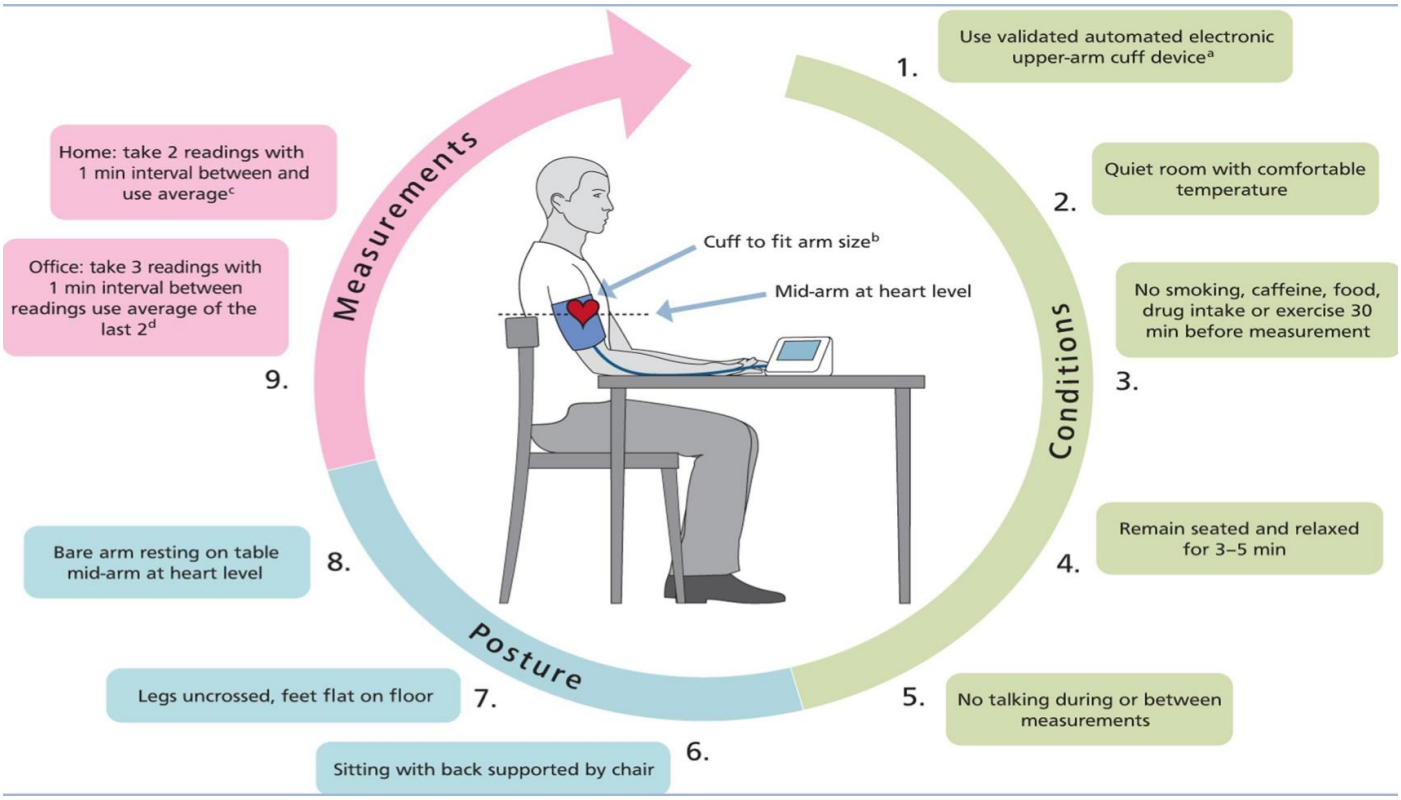
	3. Isolated systolic hypertension
Definition	✦ Elevated SBP (≥ 140 mm Hg) with DBP within normal limits (≤ 90 mm Hg)
Etiology	✦ **Most common:** decreased arterial elasticity and compliance due to aging ✦ May also be secondary to increased cardiac output due to: ✍ Anemia ✍ Hyperthyroidism ✍ Chronic aortic regurgitation ✍ AV fistula

4. Hypertensive Dippers	5. Hypertensive Non-Dippers (Nocturnal HTN ≥120/70)
BP falls at night (This is classic)	**BP does not fall at night (Non-Dippers)** (Nocturnal ↓ in average daytime SBP & DBP of <10%) or even **BP rises at night (Reverse Dippers)**
More common Better outcome	Less common Worse outcome

6. Hypertensive Urgencies	7. Hypertensive Emergencies
Severe form of HTN ≥ 180/120 mm Hg **not associated** with acute HMOD	Severe form of HTN ≥ 180/120 mm Hg **associated** with acute HMOD
HMOD= Hypertension-Mediated Organ Damage	
More common	Less common
May occur: superimposed on preexisting Hypertension (common) or de novo (rare)	

	8. Resistant Hypertension	9. Refractory Hypertension
Definition	◆ **Uncontrolled BP** (>140/90 mmHg) despite being compliant é **3** antiHTN medications, <u>including</u> a diuretic [Confirmed by ABPM or HBPM]	◆ **Uncontrolled BP** (>140/90 mmHg) despite being compliant é ≥ **5** antiHTN medications, <u>including</u> a long-acting thiazide diuretic, such as chlorthalidone, and mineralocorticoid receptor antagonist such as spironolactone.
Incidence	10-20% of treated Hypertensive patients	5% of patients with uncontrolled resistant Hypertension

HOW TO MEASURE THE BLOOD PRESSURE:



- **Measure BP in both arms :**
  - If there is a consistent difference between arms **>10 mm Hg** in repeated measurements, use the arm with the higher BP.
  - If the difference is **>20 mm Hg** → investigations.

EPIDEMIOLOGY:

Prevalence	✦ Hypertension affects between approximately one-third and one-half of adults in the US. • **Primary hypertension:** accounts for ~ **90%** of cases of hypertension in adults and prevalence is increasing in children and adolescents. • **Secondary hypertension:** accounts for ~ **10%** of cases of hypertension in adults ✦ Prevalence increases with age: Approximately 65–75% of adults develop hypertension by 65–74 years of age. ✦ Rates are highest in **African American individuals**, followed by white individuals, and lowest in Asian American and Hispanic individuals. ✦ ~ 60–87% of overweight and ~ 73–95% of obese patients are affected.
Sex	✦ ♂ > ♀ below 65 years of age ✦ After menopause, prevalence increases in women.

ETiOLOGY:

A) Essential (1ry) HTN:

- **Multifactorial** etiology including epigenetic, genetic, and environmental factors
- Directly related to total peripheral resistance and cardiac output

Risk factors:	✦ Nonmodifiable risk factors ◆ Positive family history ◆ Race and **ethnicity** ◆ Advanced age ✦ Modifiable risk factors ◆ Overweight and **obesity** (greatest modifiable risk factor) ◆ Uncontrolled **diabetes** ◆ **Smoking** ◆ Excessive alcohol intake ◆ Diet high in **sodium** and low in potassium ◆ **Physical inactivity** ◆ Psychological stress
Theories :	**A. ACTiVATiON OF THE RAAS** ◆ ↑secretion of renin which converts angiotensinogen to angiotensin I, and then to Angiotensin II which causes hypertension through: ✍ AT II is a very potent VC causing ↑↑ PR. ✍ AT II stimulates aldosterone secretion → salt & water retention. **B. ACTiVATiON OF THE SNS** ◆ ↑ secretion of catecholamines will cause VC & ↑↑PR. **C. VASCULAR HYPERTROPHY & ENDOTHELiAL DYSFUNCTiON :** ◆ ↑ production of certain growth factors → vascular hypertrophy →↑↑ PR & hypertension. ◆ Hypertension → vascular endothelial injury (endothelial dysfunction) →further ↑↑ the production of growth factors → more hypertension. **D. iNSULiN RESiSTANCE: (iR)** ◆ HTN is a member of the cardiometabolic syndrome ◆ Peripheral resistance to insulin occurs → secondary hyperinsulinemia that elevates the BP through: a. Increased Na reabsorption in the renal tubules. b. Increased sympathetic activity

B) Secondary HTN:

A) ETIOLOGY:

Renal causes	**A. RENAL PARENCHYMAL:** ◆ Diabetic nephropathy: Most common. ◆ GN: 2nd most common. ◆ Polycystic Kidney: 3rd most common. ◆ Collagen diseases: SLE & PAN. **B. RENAL VASCULAR:** ◆ Renal artery stenosis (e.g, due to atherosclerosis, fibromuscular dysplasia, polyarteritis nodosa).
Endocrinal causes	◆ **PITUITARY:** Acromegaly. ◆ **THYROID:** Hypothyroidism, or Hyperthyroidism (ISH). ◆ **PARATHYROID:** Hyperparathyroidism. ◆ **ADRENAL GLAND:** • Adrenal medulla: Pheochromocytoma. • Adrenal cortex: Cushing's syndrome & Conn's syndrome.
Neurological causes	◆ ↑ **ICT** (Cushing reflex). ◆ **LESIONS OF:** medulla & hypothalamus.
Drugs	◆ **C**atecholamines. ◆ **C**orticosteroids. ◆ **C**arbenoxolone. ◆ **C**CP ◆ **C**lozapine ◆ **C**ocaine ◆ **C**affeine ◆ **C**ox-2 inhibitors and NSAIDs. ◆ **C**alcineurin inhibitors (CNI): Cyclosporine & Tacrolimus. ◆ Erythropoietin
Miscellaneous	◆ Coarctation of the aorta. ◆ Obstructive sleep apnea (↑ catecholamines during apneic phases → secondary hypertension). ◆ Polyarteritis nodosa. ◆ Polycythemia rubra vera. ◆ Pregnancy: ✍ Gestational HTN (no proteinuria) ✍ Toxemia (with proteinuria).

- **Most common causes in adults:**
  - **< 40 years of age:** thyroid dysfunction, fibromuscular dysplasia, and renal parenchymal disease
  - **40-64 years of age:** hyperaldosteronism, thyroid dysfunction, and obstructive sleep apnea
  - **≥ 65 years of age:** renal artery stenosis
- **Most common causes in children and adolescents (< 18 years of age) :**
  - Renal parenchymal disease and coarctation of the aorta.

B) SIGNS SUGGESTIVE OF SECONDARY HYPERTENSION:

- **Severe hypertension :**
  - Resistant hypertension
  - Target organ damage disproportionate to the degree of hypertension
  - Hypertensive emergency
- **Unusual onset of hypertension :**
  - Abrupt onset
  - Onset at < 30 years of age
  - Onset of diastolic hypertension at > 65 years of age
  - Exacerbation of previously controlled hypertension
  - Drug-induced hypertension
- **Unprovoked or significant hypokalemia.**

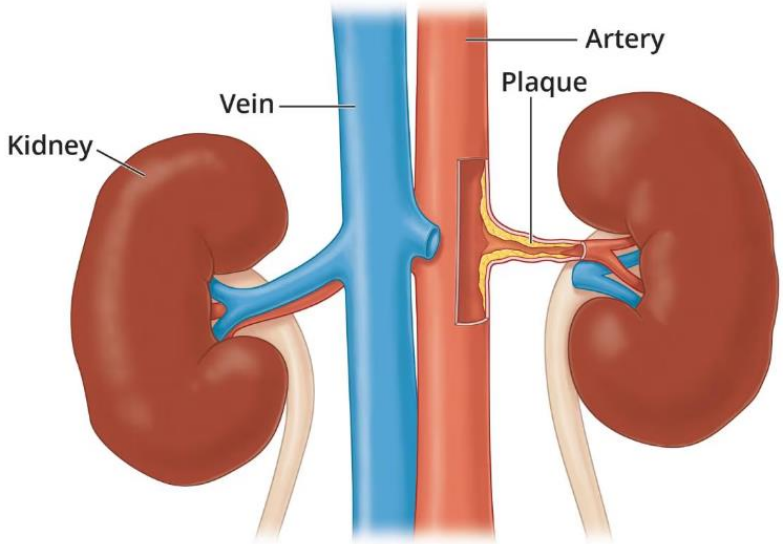
- **Aortic dissection** is a (rare) life-threatening cause of secondary hypertension that may manifest with a blood pressure difference between the right and left arm.

C) SIGNS SUGGESTIVE FOR THE CAUSE OF SECONDARY HYPERTENSION:

I) Renal Hypertension :

	Renal artery stenosis (Reno-vascular HTN)	Renal parenchymal disease
Etiology	✦ Atherosclerosis (~ 90% of cases): occurs more often in men > 50 years of age; increased risk in smokers (3) ✦ Fibromuscular dysplasia (~ 10%): mostly affects women < 50 years of age	✦ Diabetic nephropathy. ✦ GN. ✦ PCKD. ✦ SLE & PAN ✦ CKD.
Potential indications for further workup	✦ Resistant hypertension ✦ Recurrent flash pulmonary edema ✦ Abdominal bruit ✦ ↑ Serum creatinine (by ≥ 50%) within 1 week of starting an ACEI or ARB ✦ Hypokalemia ✦ Asymmetric kidney size	✦ Urinary symptoms ✦ History of excessive analgesic use ✦ Family history of polycystic kidney disease ✦ Abdominal mass (ADPKD) ✦ ↑ Serum creatinine ✦ Abnormal urine analysis (e.g., hematuria, proteinuria)
Workup and findings	✦ Duplex ultrasonography or MRA or CTA of the renal arteries	✦ Renal ultrasound

Renal Artery Stenosis



II) Endocrine Hypertension :

	Potential indication for further workup	Typical findings
Primary hyperaldosteronism (Conn syndrome)	✦ Resistant hypertension ✦ Stroke at < 40 years of age ✦ Family history of early-onset hypertension and/or primary hyperaldosteronism ✦ Adrenal incidentaloma ✦ Possible hypokalemia with consequent metabolic alkalosis	✦ ↑ Plasma aldosterone conc. (≥ 10 ng/dL) ✦ ↓ Plasma renin activity (< 1.0 ng/mL/hour) ✦ ↑ Aldosterone-to-renin ratio on a morning blood sample
Pheochromocytoma	✦ Resistant hypertension ✦ Paroxysmal hypertension ✦ Episodes of headache , palpitations , and diaphoresis ✦ Family history of endocrine tumors ✦ Cutaneous features suggestive of NF type 1	✦ ↑ 24-hour urinary fractionated metanephrines ✦ ↑ Plasma metanephrines
Hypercortisolism (Cushing syndrome)	✦ Weight gain ✦ Osteoporosis ✦ Facial plethora, skin thinning, stria ✦ Muscle weakness ✦ Hyperglycemia	✦ ↑ Serum cortisol following a low-dose dexamethasone suppression test
Hyperthyroidism	✦ Heat intolerance, diarrhea, tachycardia, and/or tremor	✦ ↓ TSH, ↑ free T4
Primary hyperparathyroidism	✦ Typically asymptomatic ✦ ↑ Serum calcium	✦ ↑ PTH level ✦ ↓ Serum phosphates
Acromegaly	✦ Clinical features of acromegaly: • Tumor mass effects like bitemporal hemianopia • Typical facial features	✦ ↑ Serum IGF-1

III) Others :

Coarctation of aorta distal to left subclavian artery	✦ **Potential indications for further workup:** • Blood pressure difference between **upper** and **lower** limbs ✦ **Workup and findings:** • Doppler echocardiography • X-ray chest • CTA or MRA chest and abdomen
Obstructive sleep apnea	✦ **Pathophysiology:** • ↑ catecholamines during apneic phases → secondary hypertension ✦ **Potential indications for further workup:** • Resistant hypertension • Obesity, snoring, and/or daytime sleepiness • Nondipping pattern on 24-hour blood pressure monitoring ✦ **Workup and findings:** • Sleep studies often leads to resolution of hypertension. ✦ Continuous positive airway pressure (CPAP)
Substance-related	✦ **Potential indications for further workup:** • Recreational drug use: amphetamines, cocaine, phencyclidine • Caffeine, nicotine, and/or alcohol use • Use of certain medications: sympathomimetic drugs (e.g., decongestants), corticosteroids, NSAIDs, oral contraceptives ✦ **Workup and findings:** • Urine drug screening • Response to withdrawal of suspected culprit

CLINICAL PICTURE:

A) CLASSIC PRESENTATION (NO CRISIS)

SYMPTOMS	◆ **MAJORITY OF PATIENTS:** asymptomatic = silent killer. ◆ **MINORITY OF PATIENTS:** Headache, tinnitus, dizziness, palpitation & epistaxis.
SIGNS	**A. GENERAL:** ◆ **Pulse:** ↑ pulse volume in ISH. ◆ **Blood pressure:** persistent elevation > 140/90 mmHg (ESC/ESH). ◆ **Fundus examination:** signs of HTN retinopathy. **B. CARDIAC:** ◆ **Precordial examination:** Signs of LVH with a heaving apex. ◆ **Auscultation:** • S2: accentuated. • Systolic ejection click. • Systolic ejection murmur. — OVER THE AORTIC AREA • Soft early diastolic murmur: • S4 — OVER THE MITRAL AREA
FEATURES OF THE CAUSE	e.g. Cushing's syndrome.
FEATURES OF COMP. :	e.g. LVF.

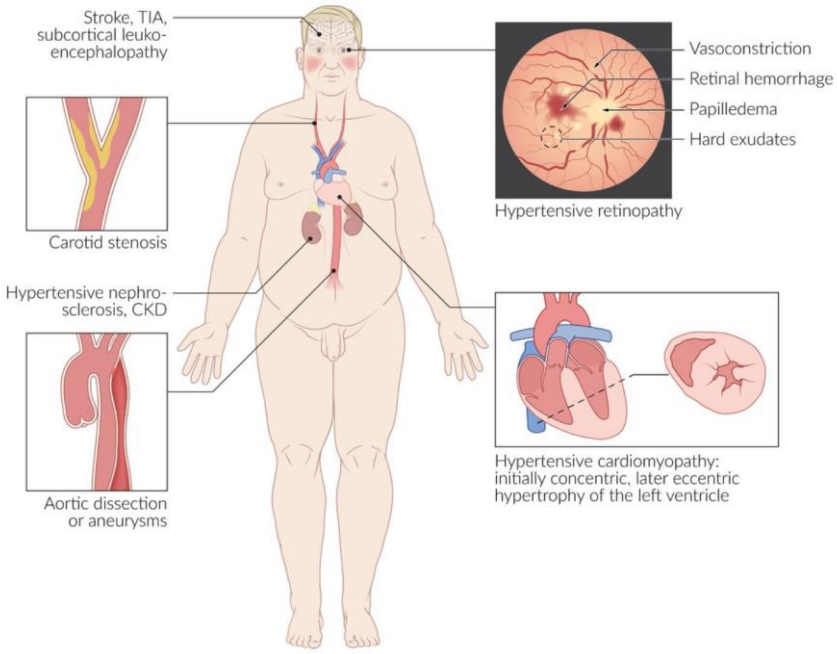
Arterial hypertension

Epidemiology
- Most common cardiovascular risk factor
- Prevalence (US): 1/3 – 1/2 of adults

Etiology
1. Primary hypertension (~ 90%)
2. Secondary hypertension (~ 10%):
- Endocrine causes (e.g., hyperaldosteronism, hyperthyroidism, pheochromocytoma)
- Renal causes (e.g., renal artery stenosis, renal parenchymal disease)
- Coarctation of the aorta
- Obstructive sleep apnea
- Medications (e.g., corticosteroids, sympathomimetic drugs)

Diagnostic criteria
BP ≥ 130/80 mm Hg

Subtypes
- White coat hypertension
- Masked hypertension
- Isolated systolic hypertension (i.e., systolic BP ≥ 140 mm Hg and diastolic BP ≤ 90 mm Hg)

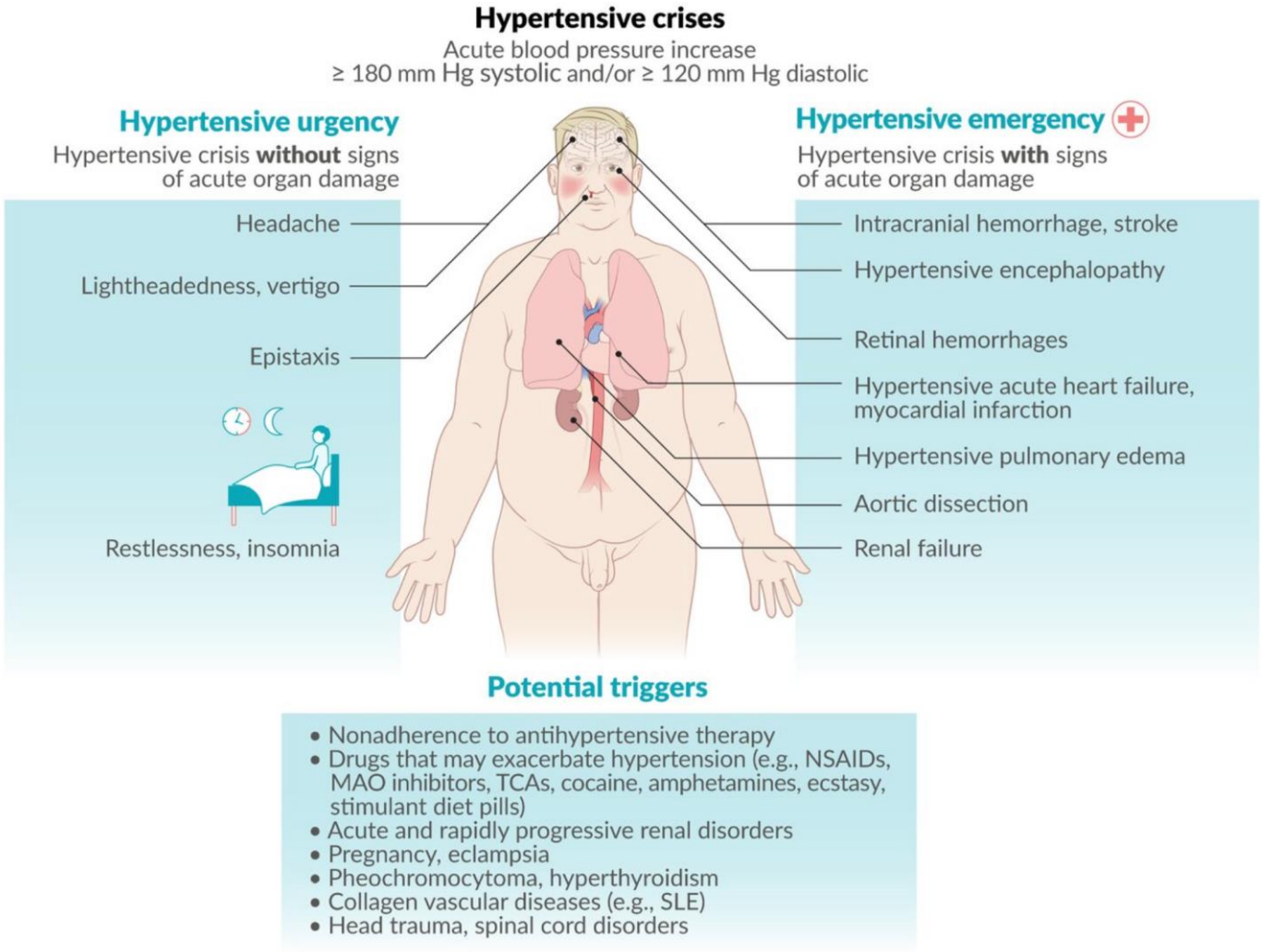


B) HYPERTENSIVE CRISIS

- **HYPERTENSIVE URGENCIES**
  - Severe form of HTN> 180/120 mm Hg **not associated** with acute HMOD.
  - Asymptomatic or isolated nonspecific symptoms (headache, dizziness or epistaxsis)

- **HYPERTENSIVE EMERGENCIES**
  - Severe form of HTN> 180/120 mm Hg **associated** with acute HMOD.
  - **TARGET ORGANS INCLUDE:** retina, brain, heart, arteries & kidneys.
  - **SPECIFIC CLINICAL PRESENTATIONS:**

Malignant Hypertension	◆ **Associated é RETINOPATHY** (flame-shaped hemorrhages & papilloedema); • If associated é (flame-shaped hemorrhages only)→ Accelerated Hypertension. • Malignant HTN & accelerated HTN have similar therapies & outcomes. ◆ **Other Associations:** DIC, Encephalopathy & acute ↓↓ renal function. • The hallmark is: small artery fibrinoid necrosis in retina, brain & kidney.
Hypertensive encephalopathy	◆ Mentioned later
Hypertensive thrombotic microangiopathy	◆ Severe HTN associated é hemolysis & thrombocytopenia in the absence of other causes.
Other presentations	◆ **CVS:** Acute HF, ACS, Acute aortic dissection. ◆ **CNS:** Stroke (Ischemic or Hemorrhagic). ◆ **KIDNEY:** AKI ◆ **PREGNANCY:** Severe preeclampsia & eclampsia.



COMPLICATIONS:

CVS	**A. ATHEROSCLEROSIS:** ◆ CAD (Angina & AMI). ◆ CVD (TIA & Stroke). ◆ PAD. **B. HEART & AORTA: :** ◆ LVH, LVF: Systolic failure (HF_SEF) & Diastolic failure (HF_DEF). ◆ Aortic dilatation (maybe Aneurysm) & aortic dissection. ◆ Arrhythmias: Hypertension is the most common cause of AF
Renal	**A. KIDNEY INJURY:** (Vasculature, Glomeruli, Tubules, Interstitium). **B. CKD, ESKD.**

CNS

1. Cerebral Atherosclerosis: (TIA & Stroke).

2. Hemorrhage: (Cerebral hemorrhage & SAH).

3. Hypertensive Encephalopathy:

Mechanism	◆ Attack of sudden severe ↑ ↑ of BP with generalized arteriolar spasm. ◆ Since the vasomotor tone of the cerebral arteries is weaker than that of the peripheral arteries, therefore the blood will rush to the cerebral circulation & causes cerebral edema (failed auto-regulation)
Clinical Picture	◆ Manifestations of ↑ ↑ ICT: headache, vomiting, blurring of vision & Papilledema. ◆ DCL & Convulsions: with no signs of lateralization.

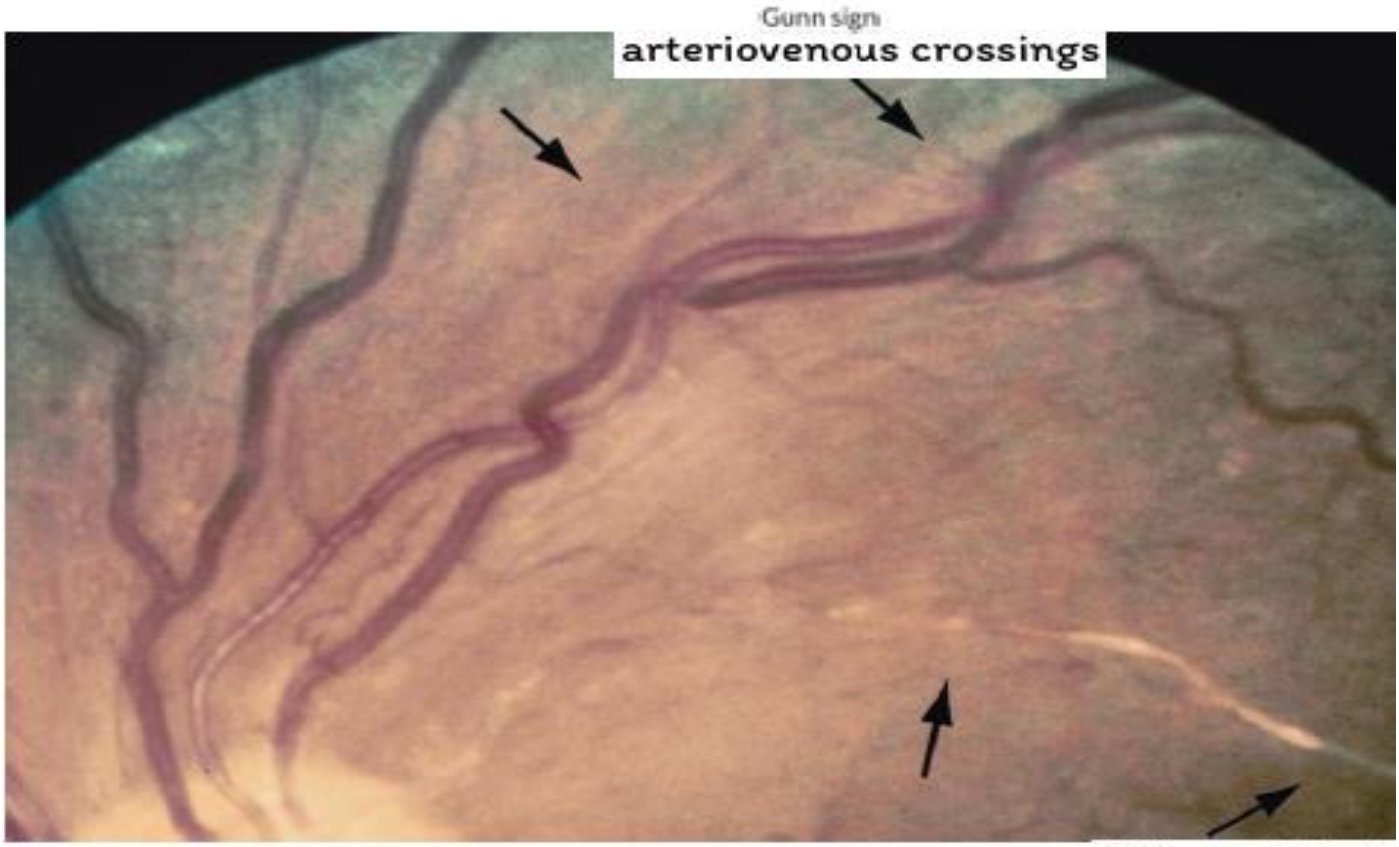
Q: HOW TO DIFFERENTIATE BETWEEN STROKE AND HYPERTENSIVE ENCEPHALOPATHY?	
STROKE	**HYPERTENSIVE ENCEPHALOPATHY**
Signs of lateralization (unilateral)	No signs of lateralization (bilateral)

4. Lacunar infarctions: Most common type of stroke

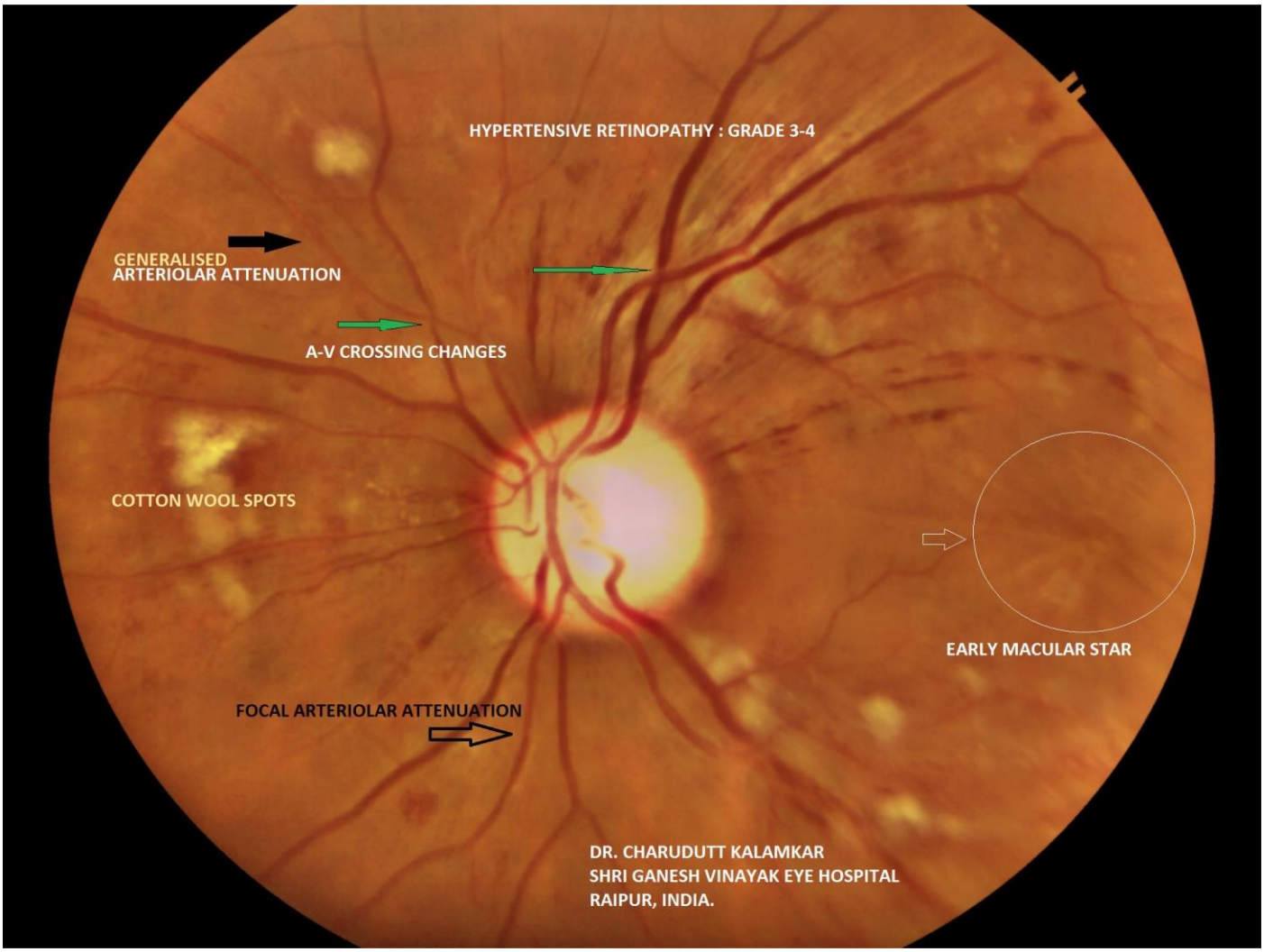
- Multiple small infarcts (in corona radiata, internal capsule, cerebellum or basal ganglia).
- **C/P:** asymptomatic, or mild manifestations (depending on the area affected)

Retinopathy

Grade	Findings	Symptoms
I	**Vessel diameter variation:** arteriolar constriction and tortuosity.	Asymptomatic
II	**Gunn sign** and marked constriction of vessels and scelorosis of arteries **(Silver wiring)**	
III	**Cotton wool exudate**, hard exudate, retinal hemorraghe, retinal edema, macular star formation.	Decreased and/or blurred vision, headache
IV	**Papilledema**, optic atrophy	



(silver wiring).



GRADE iii, iV RETiNOPATHY

iNVESTiGATiONS:

For cause: (in secondary HTN)	**1. Renal investigations:** urine, renal functions, renal imaging. **2. Endocrinal investigations:** lab tests for hormones, imaging. **3. Neurological investigations:** CT, MRI brain. **4. Blood:** CBC.
For complications	**1. Cardiac investigations:** CXR, ECG, Echocardiography. **2. Neurological investigations:** CT, MRI brain. **3. Renal investigations:** urine, renal functions, renal imaging
For detection of associated risk factors of atherosclerosis.	

TREATMENT:

Aim of treatment	◆ **Complications:** To avoid or decrease the development of complications. ◆ **BP goal (ESC/ESH 2018)** • Achieve & maintain BP < 130 / 80 mm Hg in patients <65 Y. • Achieve & maintain BP < 140 / 90 mm Hg in patients ≥65 Y.
Plan of treatment	**a. Primary Hypertension:** 1. Non- pharmacological treatment. 2. Pharmacological treatment: • Hypertension é classic presentation. • Hypertensive crisis (Urgencies & Emergencies). • Hypertension in specific situations. • Resistant Hypertension. **b. Secondary Hypertension:** Treatment of the cause.

I. Non-Pharmacological TTT (Life style modification)

- **Lifestyle measures alone may be trialed for 3-6 months in patients with:**
  - Elevated blood pressure.
  - Stage 1 HTN and 10-year ASCVD risk <10%

- **Lifestyle changes for managing hypertension**

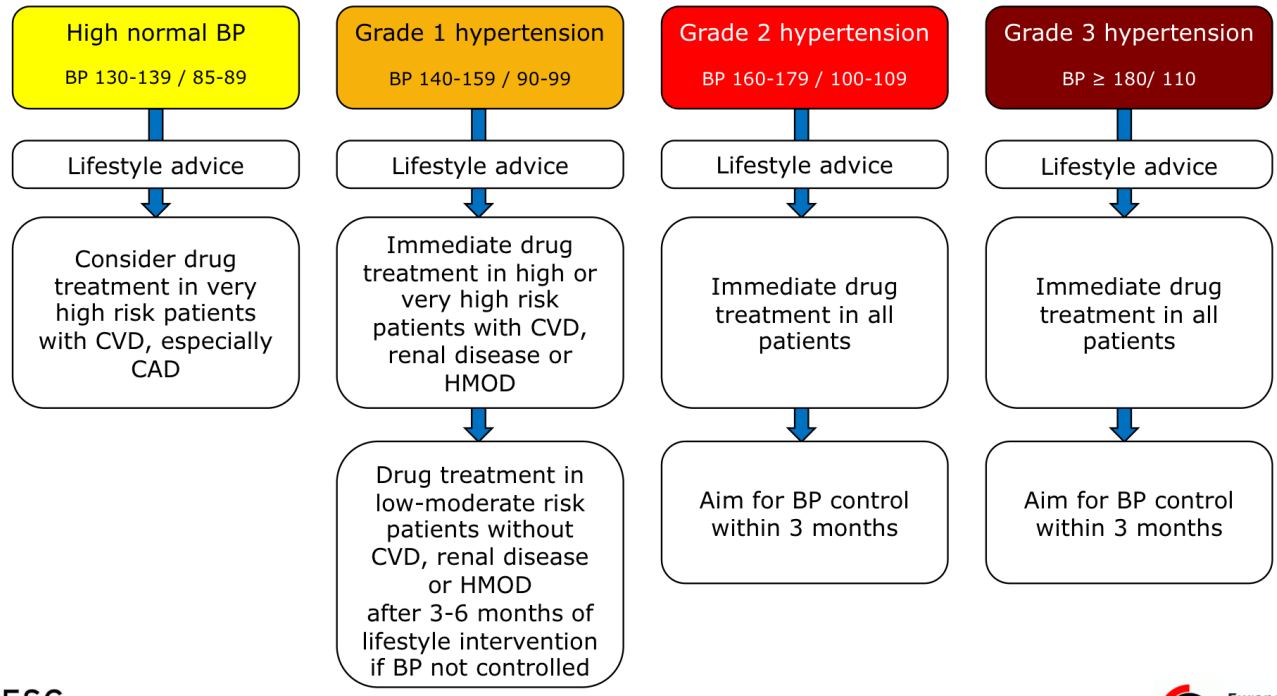
Intervention (in order of effectiveness)		Target	Approximate SBP reduction in hypertensive patients
Weight loss (most effective measure)		Ideal body weight	1 mm Hg per kg reduction in body wt
Diet	DASH diet	Diet rich in fruits, vegetables, and whole grains Low in saturated and trans fats	11 mm Hg
	Decrease dietary sodium	Daily sodium intake < 1500 mg	5–6 mm Hg
	Increase dietary potassium	Daily potassium intake 3.5–5 g (preferably by increasing fruit and vegetable intake)	4–5 mm Hg
	Decrease alcohol intake	♂: ≤ 2 standard drinks daily; ♀: ≤ 1 standard drink daily Provide counseling on alcohol use disorder, if necessary.	4 mm Hg
Exercise	Aerobic	90–150 minutes per week	5–8 mm Hg
	Dynamic resistance (e.g., weight training)	90–150 minutes per week	4 mm Hg
	Isometric resistance (e.g., hand grip exercise)	Three sessions per week	5 mm Hg

- **Smoking cessation** should be advised in all patients to reduce ASCVD risk.
- Consider possible psychosocial factors or social determinants of health that may be contributing to the patient's high blood pressure (e.g., stress, anxiety, lack of access to fresh food) and make appropriate referrals where necessary.
- Increased K intake should not be recommended for patients with **advanced CKD.**

II. Pharmacological Treatment

A) HYPERTENSION & CLASSIC PRESENTATION

Initiation of BP-lowering treatment (lifestyle changes and medication) at different initial office BP levels



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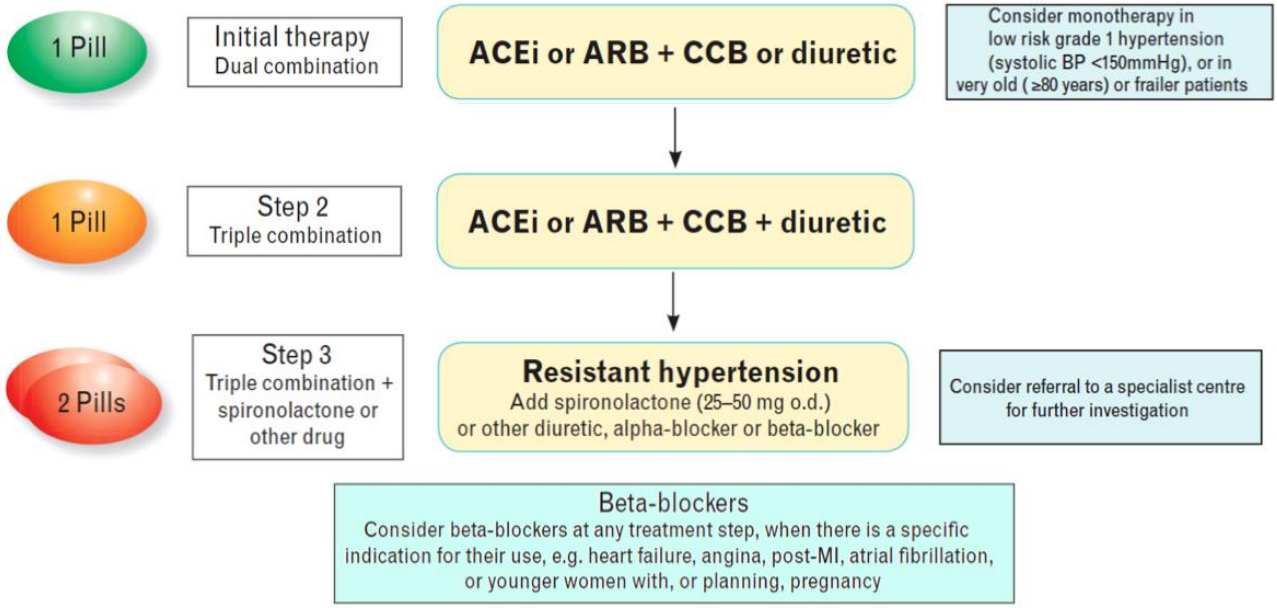
Williams, Mancia et al., J Hypertens 2018;36:1953-2041 and Eur Heart J 2018;39:3021-3104

European Society of Hypertension

Core drug-treatment strategy for uncomplicated hypertension



The core algorithm is also appropriate for most patients with HMOD, cerebrovascular disease, diabetes, or PAD

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Williams, Mancia et al., J Hypertens 2018;36:1953-2041 and Eur Heart J 2018;39:3021-3104

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NB:

- First-line medications for primary hypertension are thiazide, diuretics, ACE, ARBs, and Dihydropyridine CCB
- Do not prescribe an ACE and ARB together or in combination with a direct rein inhibitor. This increases the risk of hyperkalemia and renal function and does not provide additional benefits.

- Patients with CKD potassium >5.5 mE/L and those who take potassium supplements or potassium sparing drugs are at higher risk of hyperkalemia as an adverse effect from pharmacological treatment for hypertension.
- Do not abruptly discontinue beta blockers or alpha 2 agonists. They must be slowly tapered to avoid triggering rebound hypertension.

B) HYPERTENSIVE CRISIS (URGENCIES & EMERGENCIES)

- It is generally unwise to lower the BP too quickly or too much. as ischemic damage can occur in vascular beds that have reset their autoregulatory threshold to the higher level of BP (Hypoperfusion)

HYPERTENSIVE URGENCIES :

Site	• Hospitalization is not needed.
Goal	• 20-25% reduction in MAP or <140 mm Hg SBP.
Duration	• Within 24 hours.
Medications	• Consider a rapid-acting oral antihypertensive agents: a. Clonidine b. Captopril c. Labetalol d. Prazosin

HYPERHENSIVE EMERGENCIES :

➲ **GENERAL TREATMENT FOR ALL EMERGENCY:**

Site	• Hospitalization in an ICU.
Goal	• 20-25% reduction in MAP or **<140** mm Hg SBP. • Exception (Acute Aortic Dissection) → SBP is rapidly to **<120** mmHg (20 min)
Duration	• MAP should be by 10-20% in the 1 hour, then gradually during the next 23 hours so that the final pressure ↓ by 25%.
Medications	• IV Therapy **(See table)**

Hypertensive emergencies requiring immediate BP lowering with i.v. drug therapy

Clinical presentation	Time line and target for BP reduction	First-line treatment	Alternative
Malignant hypertension with or without acute renal failure	Several hours Reduce MAP by 20–25%	Labetalol Nicardipine	Nitroprusside Urapidil
Hypertensive encephalopathy	Immediately reduce MAP by 20–25%	Labetalol Nicardipine	Nitroprusside
Acute coronary event	Immediate reduce SBP to < 140 mmHg	Nitroglycerine Labetalol	Urapidil
Acute cardiogenic pulmonary oedema	Immediately reduce SBP to < 140 mmHg	Nitroprusside or nitroglycerine (with loop diuretic)	Urapidil (with loop diuretic)
Acute aortic dissection	Immediately reduce SBP to < 120 mmHg and heart rate to < 60 bpm	Esmolol AND nitroprusside or nitroglycerine or nicardipine	Labetalol OR metoprolol
Eclampsia and severe pre-eclampsia/HELLP	Immediately reduce SBP to < 160 mmHg and DBP to < 105 mmHg	Labetalol or nicardipine and magnesium sulphate	Consider delivery

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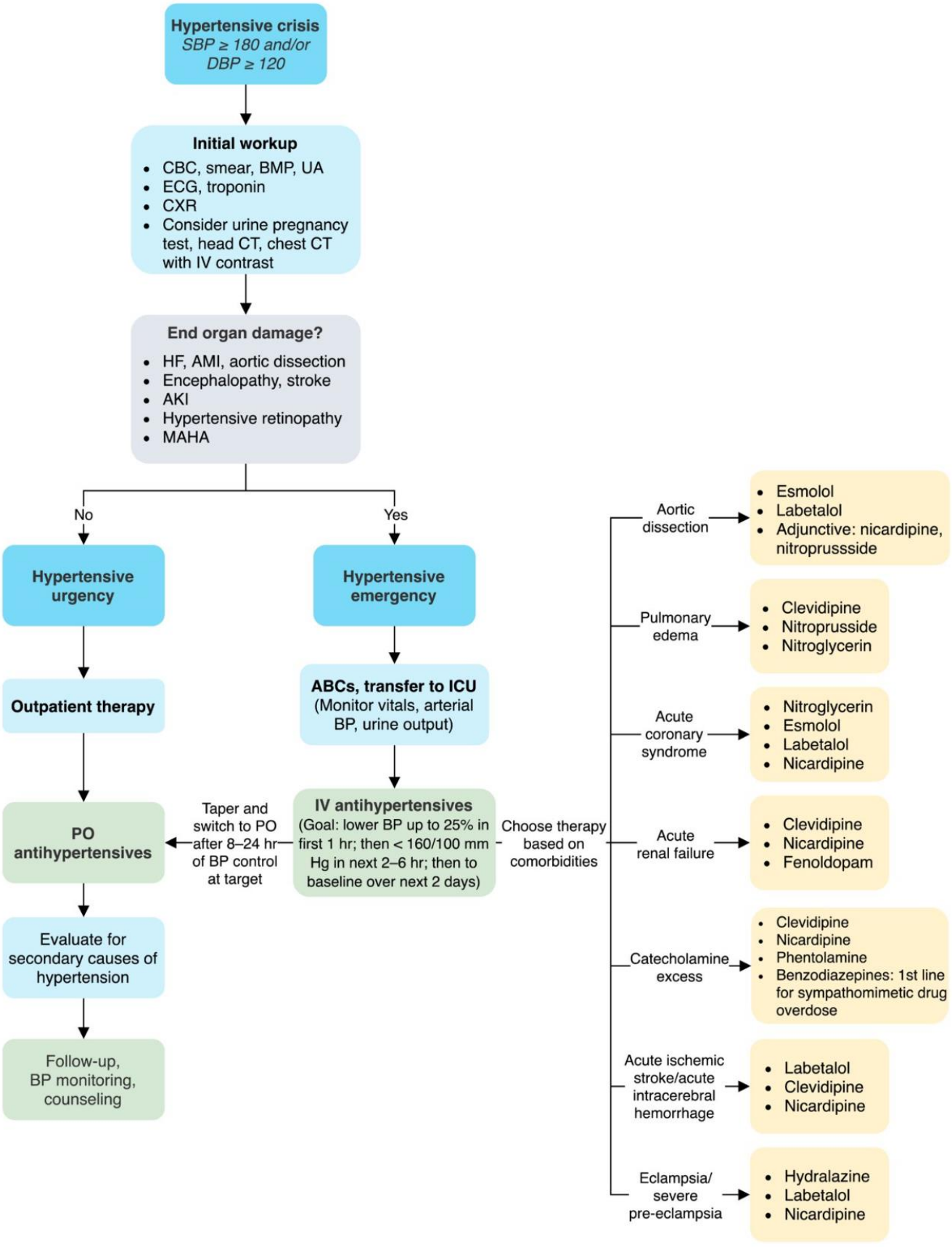
SPECIFIC TREATMENT FOR EACH EMERGENCY

- HTN encephalopathy: Dehydrating measures (conc. mannitol), Anti-convulsants.
- Other emergencies.


Hypertensive crisis
SBP ≥ 180 and/or
DBP ≥ 120
Initial workup
CBC, smear, BMP, UA
ECG, troponin
CXR
Consider urine pregnancy test, head CT, chest CT with IV contrast
End organ damage?
HF, AMI, aortic dissection
Encephalopathy, stroke
AKI
Hypertensive retinopathy
MAHA
No
Yes
Hypertensive urgency
Hypertensive emergency
Outpatient therapy
ABCs, transfer to ICU
(Monitor vitals, arterial BP, urine output)
PO antihypertensives
Taper and switch to PO after 8–24 hr of BP control at target
IV antihypertensives
(Goal: lower BP up to 25% in first 1 hr; then < 160/100 mm Hg in next 2–6 hr; then to baseline over next 2 days)
Choose therapy based on comorbidities
Evaluate for secondary causes of hypertension
Follow-up, BP monitoring, counseling
Aortic dissection
Esmolol
Labetalol
Adjunctive: nicardipine, nitroprussside
Pulmonary edema
Clevidipine
Nitroprusside
Nitroglycerin
Acute coronary syndrome
Nitroglycerin
Esmolol
Labetalol
Nicardipine
Acute renal failure
Clevidipine
Nicardipine
Fenoldopam
Catecholamine excess
Clevidipine
Nicardipine
Phentolamine
Benzodiazepines: 1st line for sympathomimetic drug overdose
Acute ischemic stroke/acute intracerebral hemorrhage
Labetalol
Clevidipine
Nicardipine
Eclampsia/ severe pre-eclampsia
Hydralazine
Labetalol
Nicardipine

C) ANTI - HYPERTENSIVE DRUGS IN SPECIFIC SITUATIONS

Specific Situation	Recommended	Avoided
CARDIOVASCULAR		
IHD	✦ Angina → BB and/or CCB ✦ AMI → BB and RAAS-B	Hydralazine Minoxidil
HF	✦ HFrEF → RAAS-B, ARNI, BB, and/or MRA Diuretics in case of Hypervolemia ✦ HFpEF→ No specific drug, all major agents can be used (e.g. RAAS-B, BB)	Non-DHP CCB DHP CCB (except Amlodipine and Felodipine)
LVH	✦ RAAS-B + (CCB or Diuretic)	
RENAL		
CKD	✦ é Albuminuria ≥ 300mg / g → RAAS - B ✦ é no Albuminuria ≥ 300mg / g → CCB or Diuretic (If eGFR is < 30mL / min / 1.73m2 → loop & not thiazide)	Combination of 2 RAAS-Bs RAAS-B in Bilateral RAS
RESPIRATORY		
ASTHMA COPD	--	BB Diuretics
OTHERS		
DIABETES	✦ RAAS-B, CCB, Diuretic (Thiazide or Thiazide-like)	BB
2ry STROKE PREVENTION	✦ RAAS-B, CCB, Diuretic (Thiazide or Thiazide-like)	--
AF	✦ BB or Non-DHP CCB	--
PAD	✦ TTT is to HTN patients é out PAD (preferably CCB)	--
Pregnancy	✦ Alpha-methyl dopa, Labetalol, CCB (Nifedipine)	RAAS-B

D) TREATMENT OF RESISTANT HYPERTENSION

1) Exclude Pseudoresistant Hypertension (mentioned below)
2) Exclude Secondary Hypertension
3) Reinforce lifestyle measures esp. Na restriction.
4) Add low dose spironolactone or Bisoprolol or Doxazosin.

NB: PSEUDORESISTANT HYPERTENSION

- It is not a true resistant HTN; it appears resistant to ttt but is actually due to some factors:
  1) Poor compliance to treatment.
  2) Poor office BP measurement technique.
  3) Poor adherence to lifestyle measures esp. Na restriction.
  4) White coat hypertension.
  5) Clinician inertia: Failure of Health care providers to initiate or intensify ttt according to current guidelines, e.g. Suboptimal antihypertensive therapy.

ANTIHYPERTENSIVE DRUGS

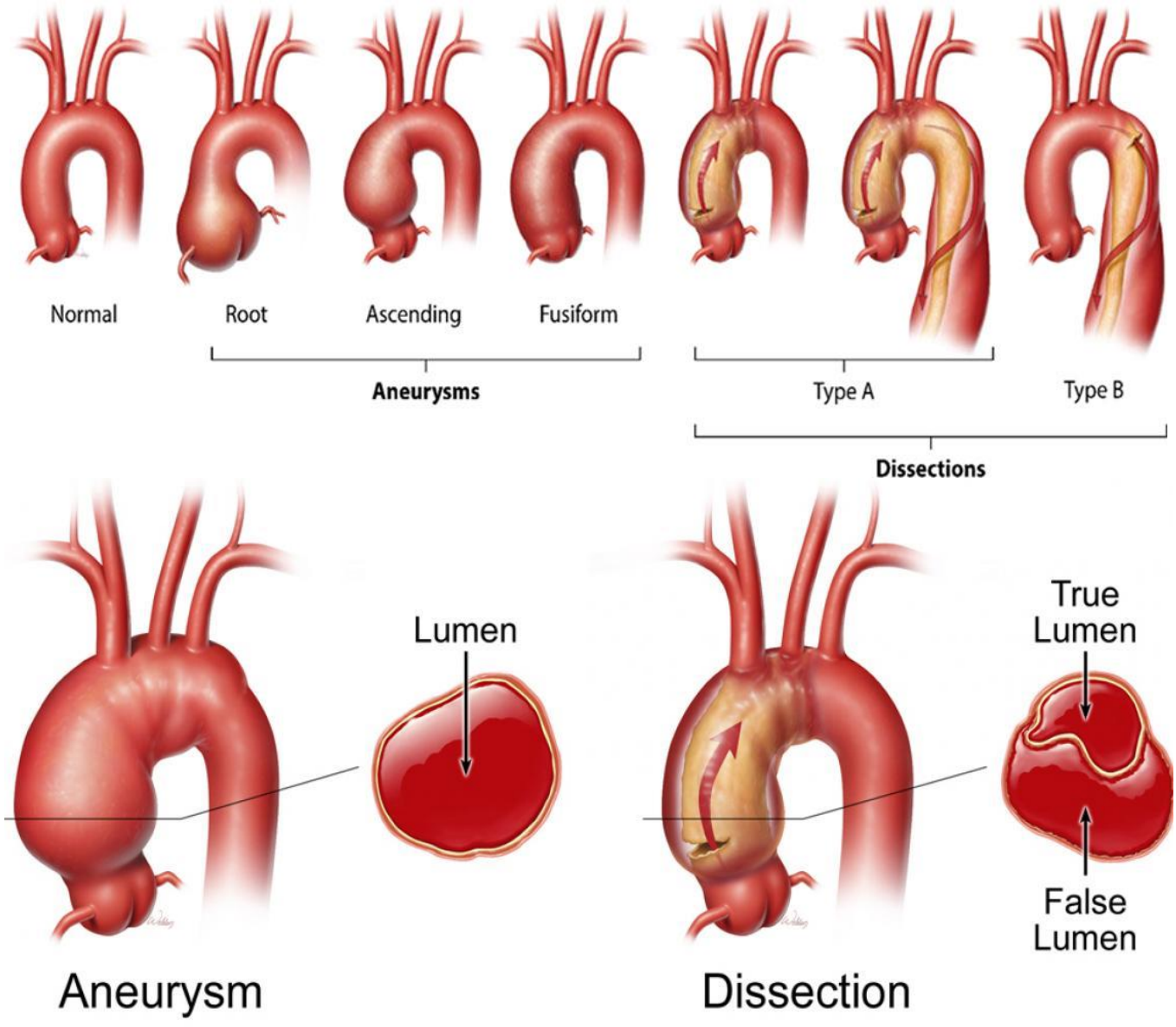
Group	Drug	MOA	Dose	S/E
1.Diuretics	Refer to the chapter of " Heart Failure"			
2. Antiadrenergics				
a) Centrally acting	α-methyl dopa (aldomet)	Central inhibition of sympathetic system	250 -1000 mg tds orally	CAH. HA (AI) Lupus like Ss
	Clonidine		0.1-0.6 Mg bid orally	Postural hypotension Rebound HTN
b) Alpha blockers	Prazocin & Doxazosin	blocks α Receptor	1 - 10 mg bid 1 - 10 mg/d orally	Syncope. Tachycardia
c) Beta blockers	Refer to angina			
d) Combined ββ& αβ	Carvedilol & Labetalol	blocks both alpha & Beta receptors	25-50 mg / day orally	Same as ββ

3. Vasodilators (Direct)			
Hydralazine	Direct smooth muscle VD	10- 40 mg tds orally 10-40 mg IV	Tachycardia Precipitates angina Lupus like syndrome
Minoxidil		2.5 – 50 mg / day orally	Tachycardia Precipitates angina Hirsutism
Sodium nitroprusside		0.5-10 µg /kg /min IV	Thiocyanate poisoning
Diazoxide		300 mg IV Rapid bolus	Hyperglycemia Hyperuricemia
4. Angiotensin converting enzyme inhibitors (ACE I)			
Captopril	Block conversion of Ag I to Ag II	12.5- 50 mg tds orally	Dry cough Angioedema Agranulocytosis Proteinuria Hyperkalemia Renal failure if given in bilateral RAS
Ramipril		5- 10 mg/day	
5. Angiotensin receptor blockers (ARBs)			
Losartan	Block binding of Ag II to its receptors	50 -100 mg/daily orally	Hyperkalemia Renal failure if given in bilateral RAS
Valsartan		80-320 mg/daily orally	
6. Calcium channel blockers (CCBs)			
Refer to angina			

DISEASES OF THE AORTA

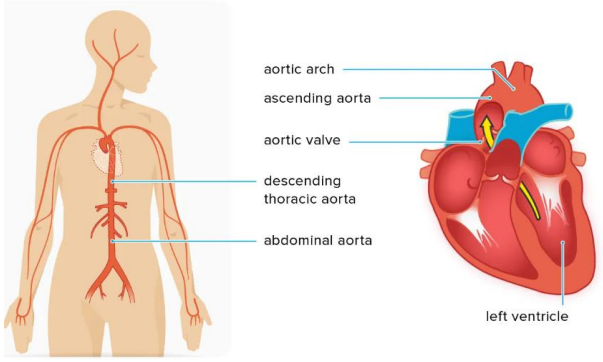
CLASSIFICATION:

1. Occlusive diseases	✦ Aortic coarctation ✦ Atherosclerosis ✦ Takayasu's arteritis
2. Trauma	✦ such as traumatic aortic rupture most often thoracic and distal to the left subclavian artery and frequently quickly fatal
3. Aneurismal diseases	✦ Genetic with marfan syndrome and ehlers-danlos syndrome ✦ Atherosclerosis ✦ Disecting aneurysm ✦ Mycotic aneurysm ✦ Behcet's disease



1. AORTIC DISSECTION

EPIDEMIOLOGY:

Incidence	✦ **Peak incidence:** 60–80 years of age ✦ **In patients with connective tissue disease:** peak incidence 30–50 years of age
Sex	♂ > ♀
Localization	✦ **Ascending aorta: ~ 65%** of cases ✦ **Descending aorta, distal to left subclavian artery:** 20% of cases ✦ **Aortic arch:** 10% of cases ✦ **Abdominal aorta:** 5% of cases 

ETIOLOGY:

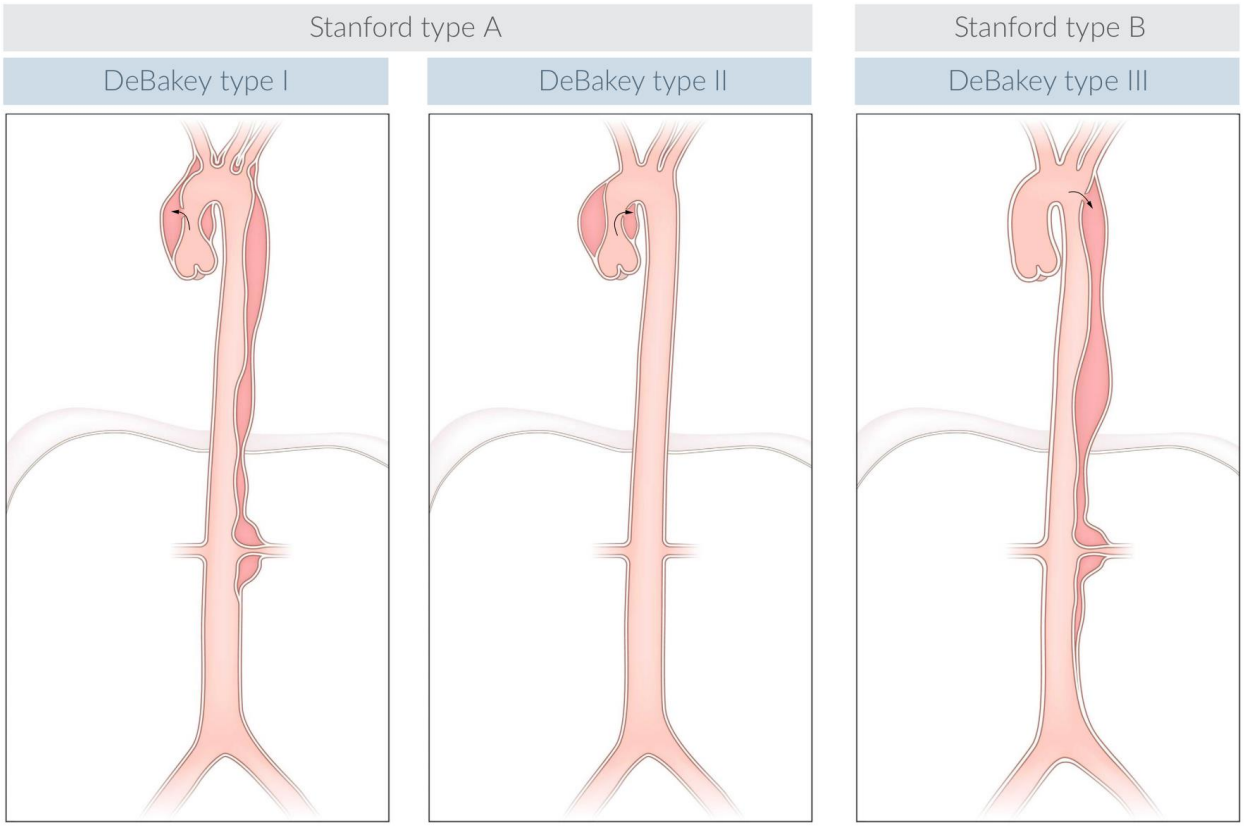
Acquired	◆ **Hypertension** (most common risk factor) • 70% of patients with aortic dissection have elevated blood pressure, which can lead to propagation of the dissection and increases the risk of rupture. • Exception: In patients < 40 years of age, less than 40% of cases are due to hypertension. ◆ **Trauma**, e.g., **deceleration injury** in a motor vehicle collision, or iatrogenic injury during valve replacements or graft surgery (traumatic aortic dissection) ◆ Vasculitis with aortic involvement (**syphilis**, Takayasu arteritis) ◆ Use of amphetamines and **cocaine** ◆ Third-trimester pregnancy (or early postpartum period) ◆ Atherosclerosis
Congenital	◆ Connective tissue disease (**Marfan syndrome, Ehlers-Danlos syndrome**) ◆ **Bicuspid aortic valve** (e.g., in Turner syndrome) ◆ Coarctation of the aorta

CLASSIFICATION:

- There are two classifications of aortic dissection to help direct management.
- Stanford classification groups dissections by whether the ascending or descending aorta is involved.
- DeBakey classification categorizes dissections according to their origin and extent.

A) Stanford classification	
Stanford type A aortic dissection	**Stanford type B aortic dissection**
✦ Any dissection involving **Ascending aorta** (defined as proximal to brachiocephalic artery), regardless of origin: • Can extend proximally to aortic valve and distally to descending aorta • Generally requires surgery • Complications include aortic regurgitation & cardiac tamponade.	✦ Any dissection **not** involving **ascending aorta:** • Descending aorta, originating distal to left subclavian artery • Most cases can be managed with medical therapy (e.g., beta blockers, vasodilators).

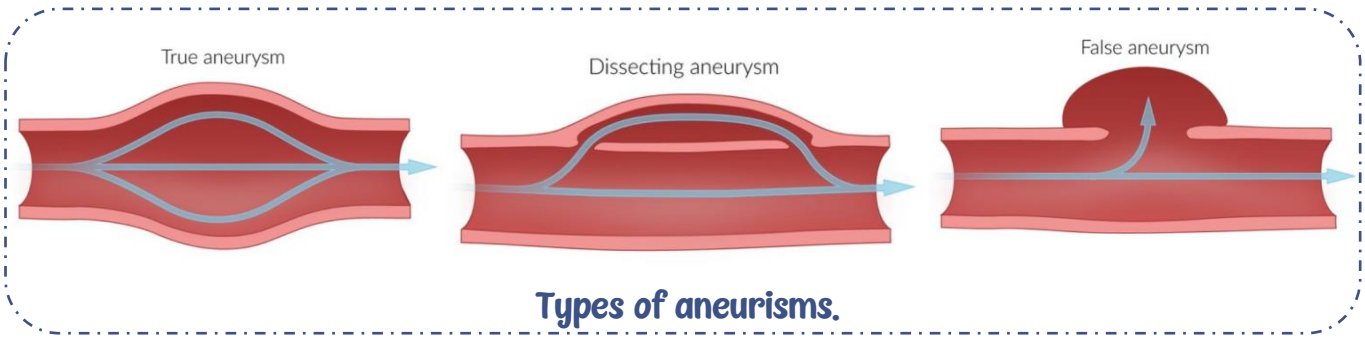
Stanford A = Affects ascending aorta &
Stanford B = Begins beyond brachiocephalic vessels.



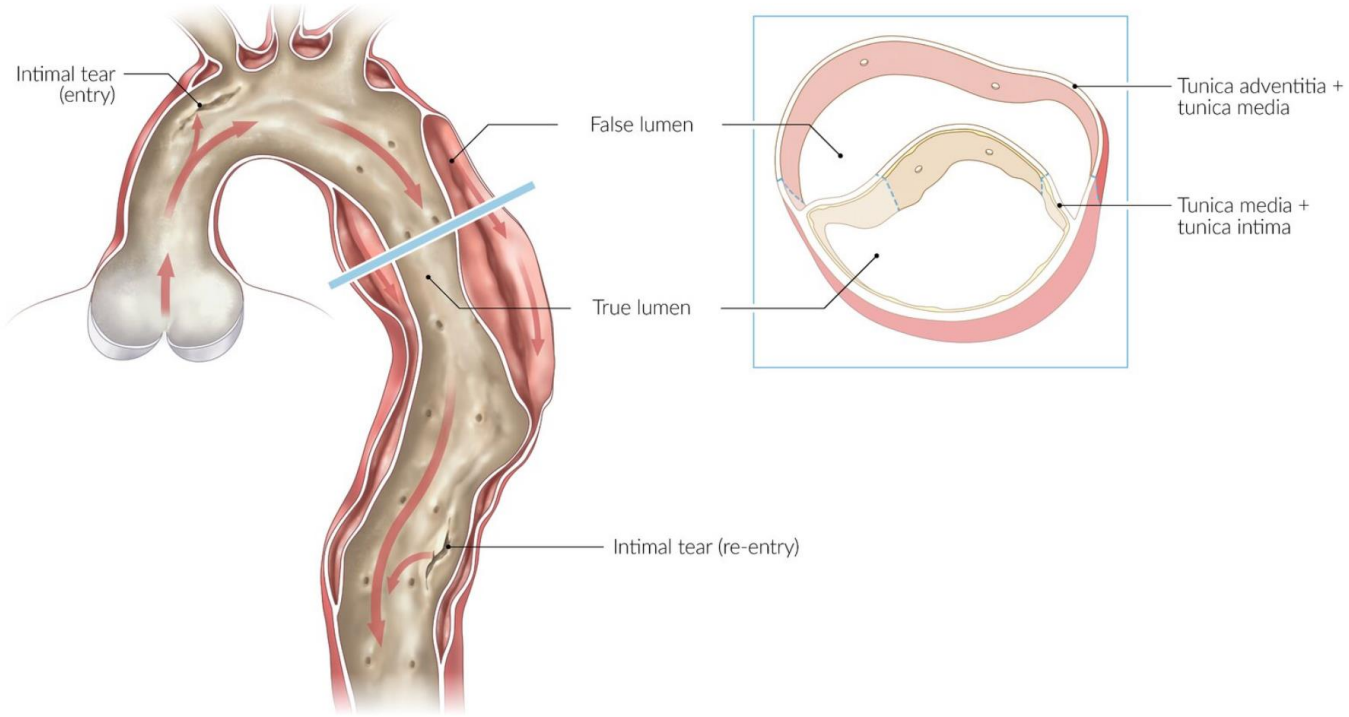
B) DeBakey classification (rarely used)	
Type I	◆ Dissections originate in the **ascending aorta** and **continue** to at least the aortic arch but typically as far as the descending aorta. ◆ Generally requires **surgery**
Type II	◆ Dissections originate in, and are restricted to, the **ascending aorta.** ◆ Generally requires **surgery**
Type III	◆ Dissections originate in **descending aorta** and most often extend distally. ◆ Most cases can be managed by **medical** therapy. ◆ **Can be further subdivided into:** a. **Type IIIa:** limited to the descending thoracic aorta above the level of diaphragm b. **Type IIIb:** extends below the diaphragm

PATHOPHYSIOLOGY:

- **Common anatomic sites of origin :**
  - Above the aortic root
  - Aortic arch
  - Distal to left subclavian artery
- Transverse tear in the aortic intima ("entry") → blood enters the media of the aorta and forms a **false lumen** in the intima-media space → hematoma forms and propagates longitudinally downwards.
  - Rising pressure within the aortic wall → **rupture**
  - Occlusion of **branching vessels** (e.g., coronary arteries, arteries supplying the brain, renal arteries, arteries supplying the lower limbs) → ischemia in the affected areas.
  - A second intimal tear may result in a "reentry" into the primary aortic lumen.



Types of aneurisms.



Aortic dissection

A tear in the tunica intima (entry tear on illustration) allows blood to enter into the tunica media. The column of blood then extends longitudinally, most commonly in the outer half of the tunica media. This creates a false lumen, which may rupture back into the true lumen of the aorta (reentry tear on illustration) or rupture through the tunica adventitia.

CLINICAL FEATURES:

1. **Sudden and severe tearing/ripping pain:**

Location	✦ Anterior chest (ascending) or back (descending) ✦ **Interscapular** or retrosternal pain ✦ Neck and jaw ✦ Abdomen or periumbilical, colicky pain
Character	✦ migrates as the dissected wall propagates caudally

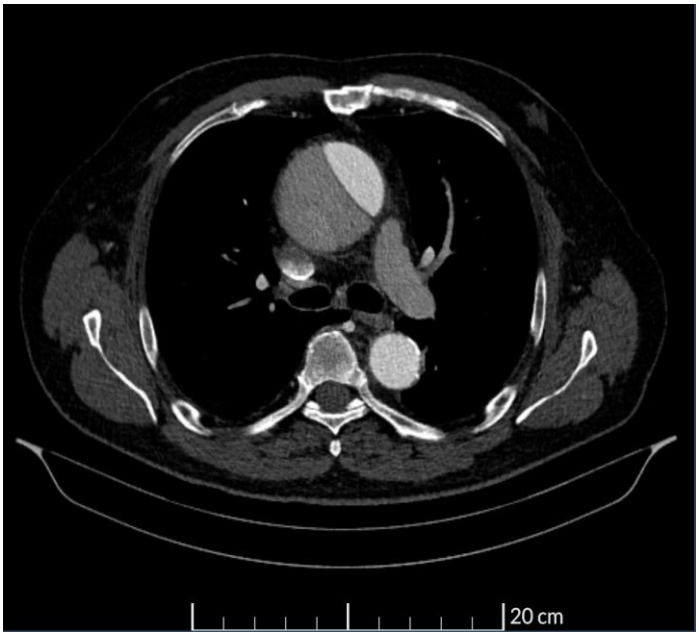
2. **Hypertension** or hypotension
3. **Asymmetrical** blood pressure and pulse readings between limbs
4. **Wide pulse pressure**
5. **Syncope**, diaphoresis, confusion, or agitation

COMPLICATIONS:

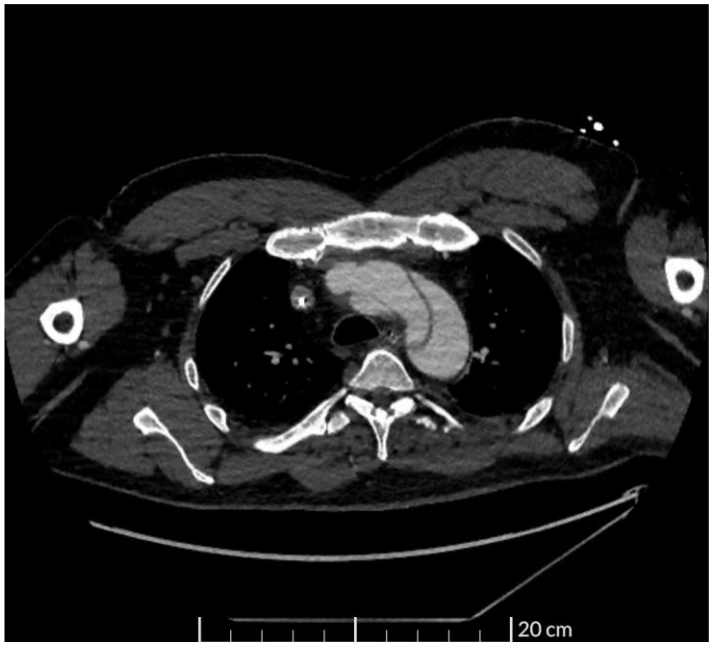
Malperfusion syndrome	✦ A complication of aortic dissection where blood flow to major vascular beds is interrupted, resulting in ischemia and end-organ damage ✦ May affect gut, kidneys, upper or lower limbs, coronary, spinal, or cerebral circulation
Aortic rupture and acute blood loss	✦ Acute back and flank pain (tearing pain), symptoms of shock → indication for emergency surgery
Complications of Stanford type A dissections	✦ **Myocardial infarction** (coronary artery occlusion) ✦ **Aortic regurgitation** (extension of dissection into aortic valve) ✦ **Cardiac tamponade** progressing to cardiogenic shock ✦ Pericarditis, pericardial effusion & pericardial tamponade (slow extension of the dissection into the pericardium) ✦ **Stroke** (extension of the dissection into the carotids)
Complications of both Stanford A & B dissections	✦ Bleeding into the thorax, mediastinum, and abdomen ✦ Arterial occlusion followed by ischemia of the: • Celiac trunk, superior/inferior mesenteric artery → acute abdomen, ischemic colitis • Renal arteries → **acute renal failure** (oliguria, anuria) • Spinal arteries → **weakness of lower extremities or acute paraplegia** • Complete occlusion of the distal aorta → Leriche syndrome (aortoiliac occlusive disease)

INVESTIGATIONS:

Lab	✦ D-Dimer.
ECG	✦ For DD of acute chest pain (to rule out AMI).
CXR	✦ Widening of aortic silhouette (mediastinum).
Cardiovascular Imaging	✦ **HD stable without suspicion of Stanford A:** • CTA (Initial imaging). • MRA (If CTA is equivocal). ✦ **HD unstable or é strong suspicion of Stanford A:** • TEE (Initial imaging). • CTA (If TEE is unavailable).



Aortic dissection (Stanford A)



Aortic dissection (Stanford B)

TREATMENT:

Surgical	✦ **Acute Stanford A (ascending aortic dissection) is:** ⇨ Surgical Emergency
Medical	"IV opioids for analgesia" ✦ **Acute aortic dissection is:** ⇨ Hypertensive Emergency. ✦ **Maintain SBP ( ) 100 & 120 mmHg, HR < 60 bpm:** ⇨ **βB:** Esmolo (IV) or Labetalol (IV inf). ⇨ **VD:** Nicardipine (IV inf) If βB are not tolerated. ⇨ **VD:** Nitroprusside (IV inf) → If SBP remains > 120 mmHg. ⇨ **VD:** Should not be used é out first controlling HR with βB.

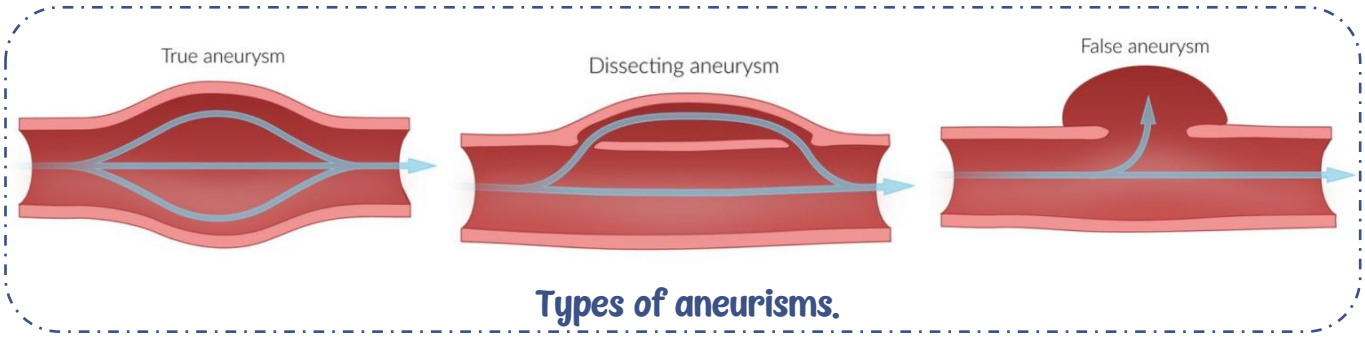
2. AORTIC ANEURYSM

Definition	✦ Localized pathological dilatation of the aorta.
Types	1. Thoracic aortic aneurysm. 2. Abdominal aortic aneurysm.

A) Thoracic Aortic Aneurysm

DEFINITION:

- Dilatation of all three layers of the aortic wall (intima, media, and adventitia) to > 150% of the normal diameter (a true aneurysm)
  a. Ascending aorta: approx. > 5.0 cm
  b. Descending aorta: approx. > 4.0 cm



Types of aneurisms.

EPIDEMIOLOGY:

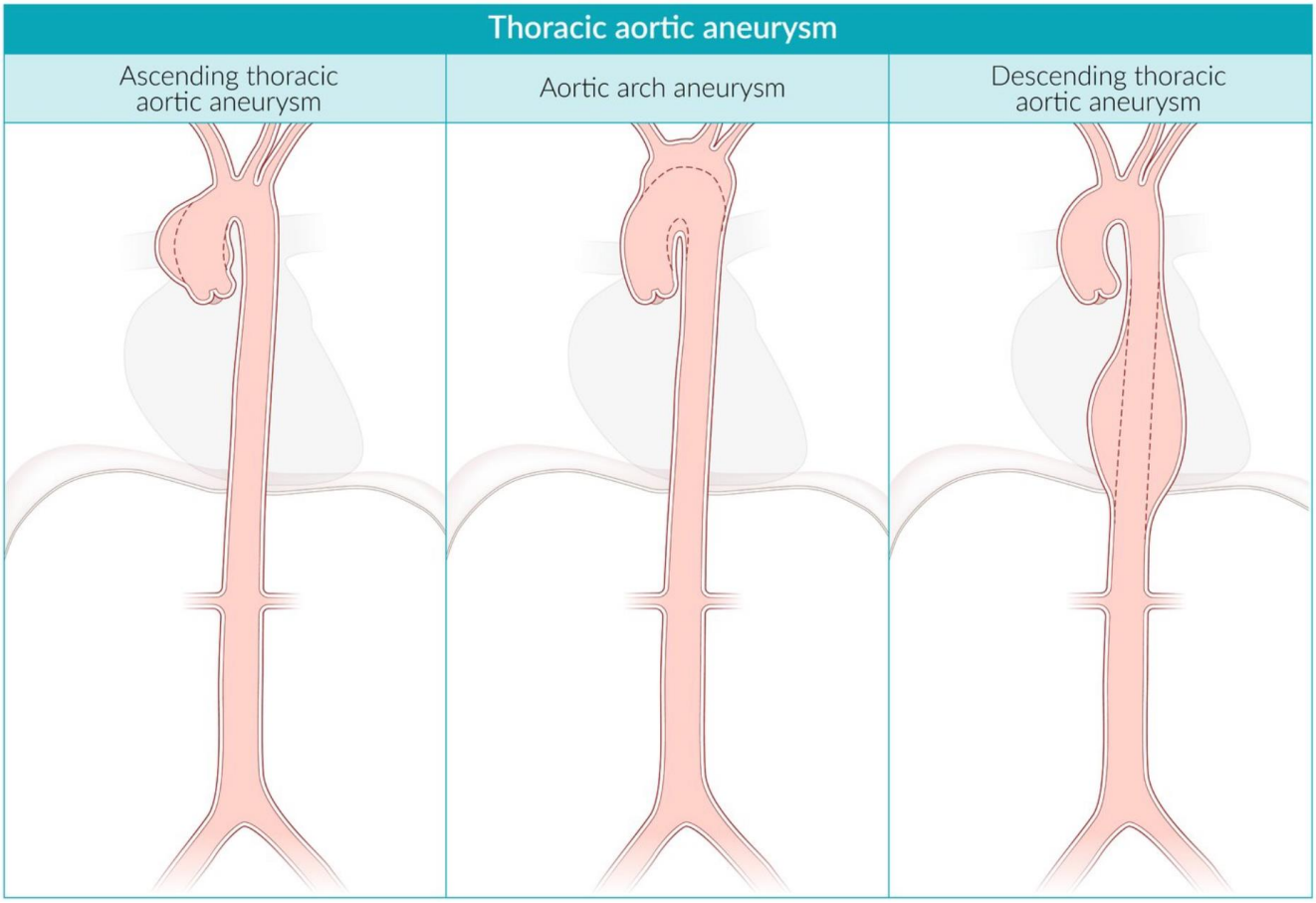
- **Less common** than abdominal aortic aneurysm (AAA)
- **Peak incidence:** 60–65 years
- **Sex:** ♂ > ♀ (~ 3:1)

RISK FACTORS :

- Arterial hypertension
- Advanced age
- Smoking
- Trauma
- Tertiary syphilis (due to obliterative endarteritis of the vasa vasorum)
- Connective tissue diseases (e.g., Marfan syndrome, Ehlers-Danlos syndrome)
- Bicuspid aortic valve
- Positive family history
- **Aortitis:** aortic wall inflammation
  - Noninfectious (most common): giant cell arteritis, Takayasu arteritis, other inflammatory conditions
  - Infectious: bacterial , mycobacterial, fungal

CLASSIFICATION:

1. Ascending aorta (**most common location**)
2. Descending aorta (thoracoabdominal)
3. Aortic arch



Thoracic aortic aneurysm

Ascending thoracic aortic aneurysm: the most common type; can arise from the aortic valve to the brachiocephalic trunk
Aortic arch aneurysm: involves the brachiocephalic vessels
Descending thoracic aortic aneurysm: arises distal to the left subclavian artery

PATHOPHYSIOLOGY:

- **Ascending** thoracic aortic aneurysm: most often due to **cystic medial necrosis**
- **Descending** thoracic aortic aneurysm: typically a result of **atherosclerosis**
- **Inflammation and proteolytic degeneration of connective tissue proteins** (e.g., collagen and elastin) and/or smooth muscle cells in high-risk patients → loss of structural integrity of the aortic wall → widening of the vessel
- The aneurysmatic dilatation of the vessel wall may cause disruption of the laminar blood flow and turbulence.
- Possible formation of thrombi in the aneurysm → peripheral thromboembolism

CLINICAL FEATURES:

- Aortic aneurysms are mostly asymptomatic or have nonspecific symptoms.
- They are often discovered incidentally on imaging.

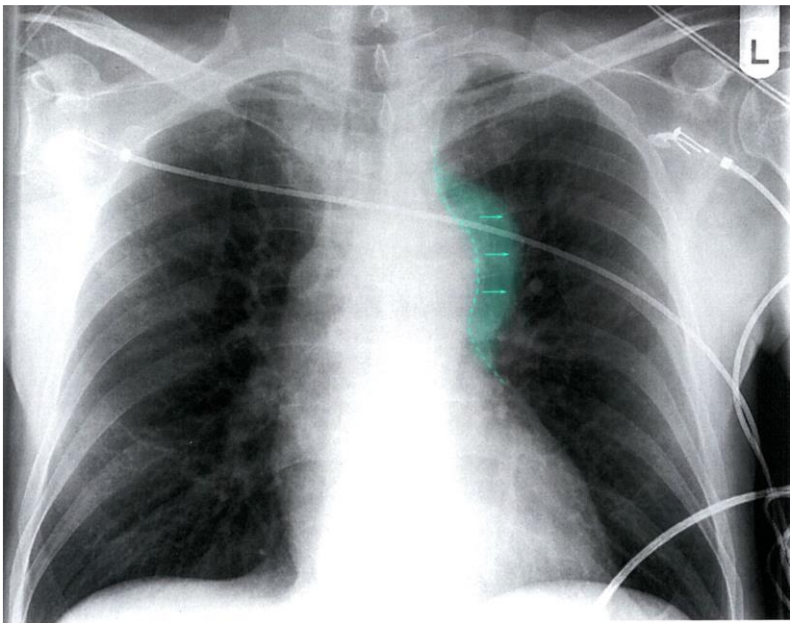
1. **Chest pressure**
2. **Thoracic back pain**
3. Features of mediastinal compression/obstruction, such as:
   - **Difficulty swallowing** (esophagus)
   - Upper venous congestion (superior vena cava syndrome)
   - Hoarseness (recurrent laryngeal nerve)
   - Cough, wheeze, stridor (trachea)
   - Horner syndrome (sympathetic trunk)

COMPLICATIONS:

- **Embolism:** caused by thrombotic material of the aneurysm.
- **Aortic valve regurgitation:** due to aortic root dilation.
- Aortic dissection.
- Thoracic aortic aneurysm rupture.

DIAGNOSTICS (IMAGING):

1. **Chest x-ray :**
   - **Indications:** may be conducted as an initial imaging study in patients with chest pain and/or dyspnea
   - **Suggestive findings:**
     - ⇨ Abnormal aortic contour.
     - ⇨ **Widened mediastinum.**
     - ⇨ Tracheal deviation.



2. CT angiography chest :

- **Indications:** best confirmatory test for TAAs.

3. Additional imaging :

	Indication
MR angiography chest with and without IV contrast	◆ Consider as an alternative to CTA.
Transthoracic echocardiography	◆ Rapid assessment in hemodynamically unstable patients ◆ Evaluation for concomitant heart disease
Transesophageal echocardiography	◆ allows for more accurate assessment than TTE ◆ intraoperative monitoring
Catheter angiography (aortography)	◆ Evaluation and possibly treatment of coexisting coronary artery disease ◆ Assessment of aortic lumen and branch vessels

TREATMENT:

⇨ Approach

- Unstable patients (e.g., in the case of rupture): emergency TAA repair.
- Symptomatic patients: urgent TAA repair
- Asymptomatic patients
  - Aneurysm surveillance
  - Elective TAA repair when size or growth thresholds are passed
- All patients: conservative management with reduction of cardiovascular risk factors

I) Invasive treatment: TAA repair :

⇨ General indications:

- TAA rupture.
- Symptomatic TAA.
- Asymptomatic TAA when size or growth thresholds are passed.

⇨ Indications for asymptomatic patients:

Affected location of the aorta	Aortic diameter
Ascending aorta	• **General threshold:** ≥ 5.5 cm • **Aortic valve pathology or CAD requiring surgical repair:** > 4.5 cm • **Marfan syndrome, Ehlers-Danlos syndrome:** 4–5 cm • **Growth rate:** > 0.5 cm/year
Aortic arch (isolated)	• **General threshold:** ≥ 5.5 cm • **Growth rate:** > 0.5 cm/year
Descending aorta	• **Anatomic requirements for TEVAR are met:** ≥ 5.5 cm • **OSR required:** ≥ 6 cm • **Chronic dissection requiring OSR:** ≥ 5.5 cm • **Increased risk of rupture:** Lower thresholds are reasonable.

⇨ Procedures:

	Open surgical repair (OSR)	Thoracic endovascular aneurysm repair (TEVAR)
Indications	• preferred in young patients with few comorbidities and low surgical risk and patients with connective tissue disorders ⇨ Symptomatic TAAs involving ascending aorta or aortic arch	• Degenerative or traumatic descending aortic aneurysms
Complications	• 40% of all patients experience a perioperative complication ⇨ Paralysis (due to spinal cord injury or ischemia)	• Ischemia of the bowel, kidneys, spinal cord

II) Conservative management

⇨ Reduction of cardiovascular risk factors:

Blood pressure management	✦ **Optimal blood pressure goal to reduce aortic wall stress:** the lowest blood pressure that the patient can tolerate • Less than 140/90 mm Hg in patients without diabetes • Less than 130/80 mm Hg in patients with diabetes or CKD ✦ **Preferred agents:** • Beta blockers (e.g., propranolol, metoprolol) • ACE inhibitor (e.g., lisinopril, enalapril) • Angiotensin receptor blocker (e.g., losartan, candesartan)
Smoking cessation	
Lipid profile optimization	✦ **In patients with atherosclerotic aortic aneurysms** • Target LDL cholesterol: < 70 mg/dL • Preferred agent: statin (e.g., atorvastatin)

III) Aneurysm surveillance

Follow-up frequency for surveillance of thoracic aortic aneurysm or dilatation via CT or MR

Part of the aorta	Maximum diameter of the aorta	Recommended follow-up interval
Ascending aorta	• 3.5–4.4 cm	• 12 months
	• ≥ 4.5 cm	• 6 months
Aortic arch	• 3.5–3.9 cm	• 12 months
	• ≥ 4 cm	• 6 months
Descending aorta	• 4–4.9 cm	• 12 months
	• ≥ 5 cm	• 6 months

B) Abdominal Aortic Aneurysm

DEFINITION:

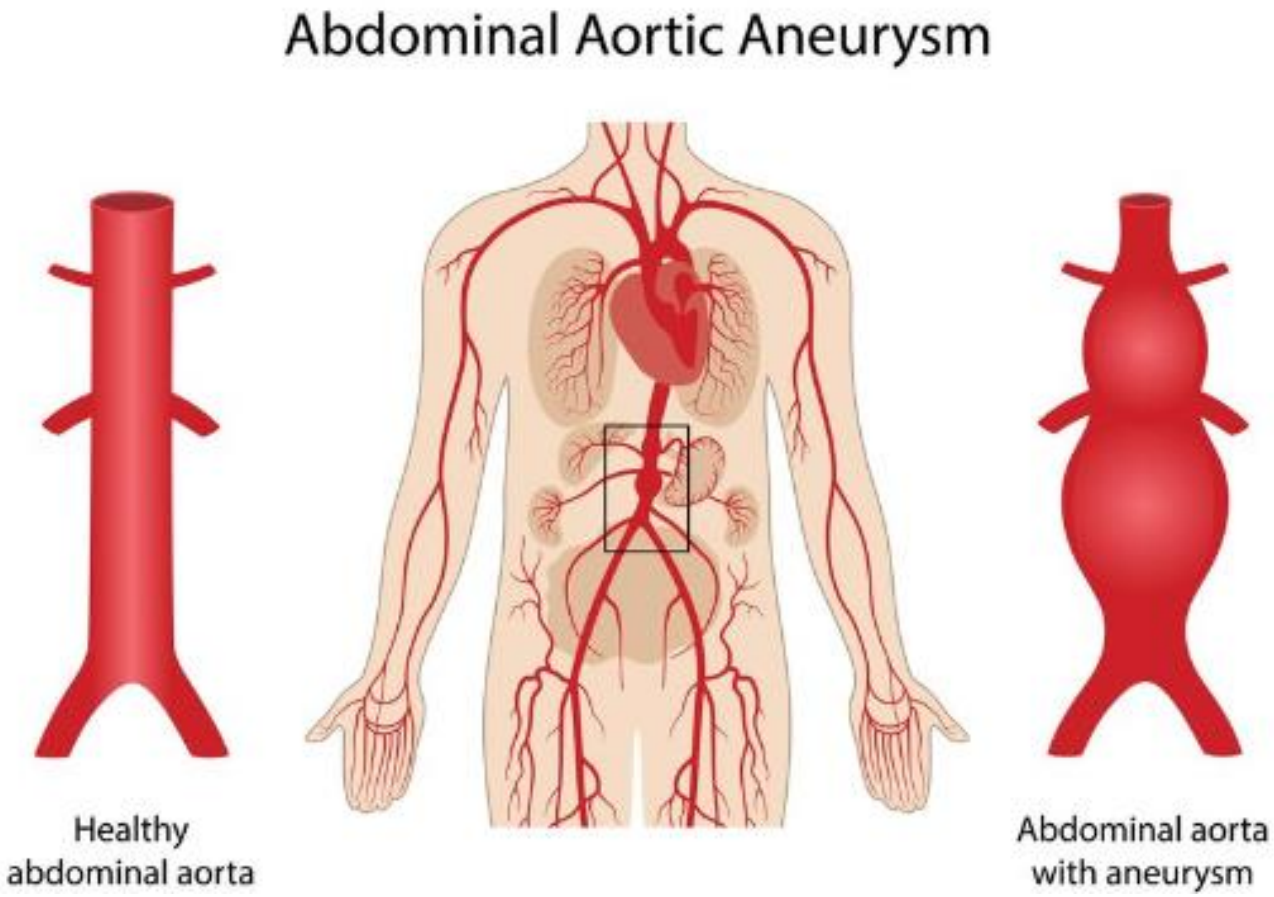
- Localized dilation of all three layers of the abdominal aortic wall (intima, media, and adventitia) to ≥ 3 cm.

EPIDEMIOLOGY:

- **Peak incidence:** 60–70 years (rare in patients < 50 years)
- **Sex:** ♂ > ♀: ~ 2:1

RISK FACTORS:

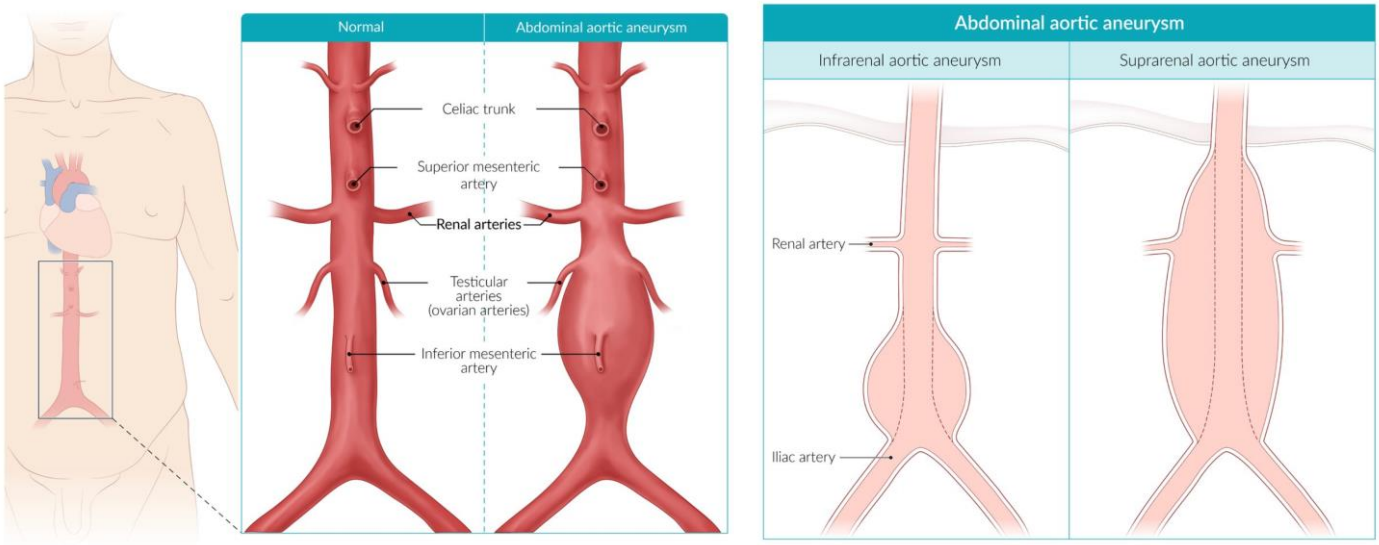
1. **Smoking** (most important risk factor)
2. Advanced age
3. Atherosclerosis (ASCVD)
4. Hypercholesterolemia and arterial hypertension
5. Positive family history
6. Male sex
7. Trauma



CLASSIFICATION:

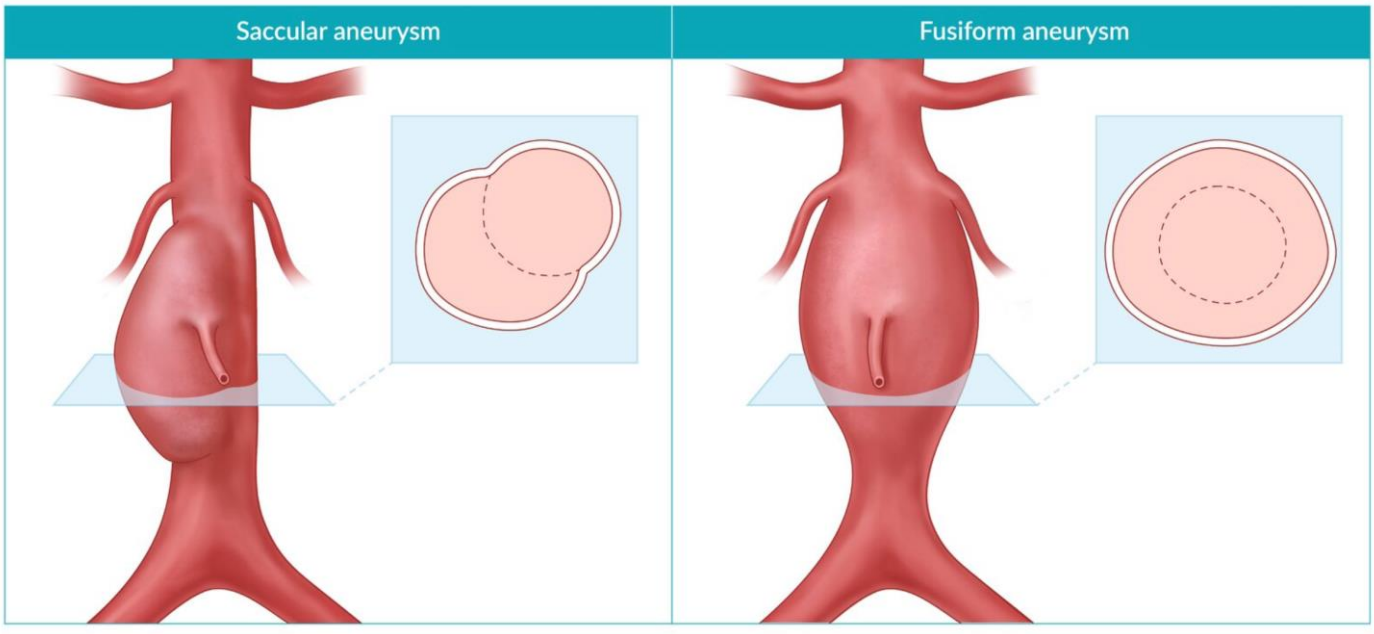
Localization

- **Infrarenal: below the renal arteries**
  - Most common location.
  - One-third of aneurysms extend into the iliac arteries.
- **Suprarenal:** above the renal arteries.



Shape

- Saccular (spherical)
- Fusiform (spindle-shaped)



PATHOPHYSIOLOGY:

- Inflammation and proteolytic degeneration of connective tissue proteins (e.g., collagen and elastin and/or smooth muscle cells) in high-risk patients → loss of structural integrity of the aortic wall → widening of the vessel → mechanical stress (e.g., high blood pressure) acts on weakened wall tissue → dilation and rupture may occur.
- The aneurysmatic dilatation of the vessel wall may cause disruption of the laminar blood flow and turbulence.
- Possible formation of thrombi in the aneurysm → peripheral thromboembolism

CLINICAL FEATURES:

- Aortic aneurysms are usually asymptomatic or have nonspecific symptoms. They are often discovered incidentally on ultrasound or CT scan. Rupture or dissection of the aneurysm is a life-threatening condition.
  1. Lower **back pain**
  2. **Pulsatile abdominal mass** at or above the level of the umbilicus
  3. **Bruit on auscultation**
  4. Peripheral thrombosis and distal atheroembolic phenomena (e.g., blue toe syndrome and livedo reticularis)
  5. Decreased ankle brachial index

COMPLICATIONS:

- **Abdominal aortic aneurysm rupture.**
- **Embolism:** caused by thrombotic material from the aneurysm
- **Aortic dissection.**
- **Postoperative complications:**
  - Ischemia of the bowel, kidneys, and spinal cord
  - Anterior spinal artery occlusion
  - Prosthetic graft infection
  - Aortoenteric fistula
  - Complications following EVAR
    - ⇨ Endoleak
    - ⇨ Access site complications, e.g., bleeding, hematoma, false aneurysm
    - ⇨ Graft limb thrombosis

DIAGNOSTICS:

- ⇨ Diagnosis of AAA is confirmed by imaging showing aortic diameter > 3 cm.
- ⇨ Unstable patients should be taken directly to OR for emergency surgery if ruptured AAA is suspected.
- ⇨ There are no laboratory findings specific to AAA.

	Indication
Abdominal ultrasound (formal ultrasound or POCUS)	◆ **Best initial and confirmatory test in:** • Asymptomatic patients • Patients with abdominal pain and no known AAA or risk factors for AAA
CT angiography abdomen & pelvis	◆ Imaging modality of choice in symptomatic patients and for **preintervention planning** ◆ To help confirm the diagnosis when ultrasound is not possible in asymptomatic patients

TREATMENT:

⇨ **Approach:**

- ✦ **Patients with any symptoms:** immediate vascular surgery consult
  - Suspected or known rupture (regardless of patient stability) : emergency repair .
  - Impending rupture : urgent aneurysm repair, ideally within normal working hours, as this is associated with better outcomes
- ✦ **All patients:** reduction of cardiovascular risk
  - Smoking cessation
  - Appropriate medical management (e.g., hypertension)

I) Invasive treatment: AAA repair

Indications	✦ **Urgent repair:** impending rupture or leaking AAA ✦ **Elective repair:** ◆ Fusiform aneurysm with maximum diameter ≥ 5.5 cm ◆ Small fusiform aneurysm expanding ≥ 1 cm per year
Procedures	✦ **Endovascular aneurysm repair (EVAR)** ◆ Expandable stent graft (older, high risk patients or patients unfit for surgery) ✦ **Open surgical repair (OSR)** ◆ Tube graft or Y-prosthesis (Younger patients).

II) Conservative treatment: AAA surveillance without repair

⇨ Small (< 5.5 cm), asymptomatic AAA can typically be observed with interval surveillance ultrasound.

Follow-up frequency for AAA surveillance	
Maximum diameter of the abdominal aorta	**Recommended follow-up interval**
2.5–2.9 cm	• Repeat ultrasound after 10 years.
3–3.9 cm	• Ultrasound every 3 years
4–4.9 cm	• Ultrasound every 12 months
5.0–5.4 cm	• Ultrasound every 6 months

	Abdominal vs. thoracic aortic aneurysm	
Characteristics	**Abdominal aortic aneurysm**	**Thoracic aortic aneurysm**
Location	✦ Below the renal arteries (most common)	✦ Ascending aorta (most common)
Epidemiology	✦ Advanced age ✦ Predominantly men ✦ More common than TAA	✦ Advanced age ✦ Predominantly men
Etiology	✦ Smoking (most important risk factor) ✦ Atherosclerosis ✦ Hypercholesterolemia and arterial hypertension	✦ Arterial hypertension ✦ Bicuspid aortic valve ✦ Tertiary syphilis ✦ Connective tissue diseases (e.g., Marfan syndrome, Ehlers-Danlos syndrome) ✦ Trauma ✦ Smoking
Clinical features	✦ Pulsatile abdominal mass ✦ Bruit on auscultation ✦ Lower back pain	✦ Feeling of pressure in the chest ✦ Thoracic back pain
Diagnostics	✦ Abdominal ultrasound (best initial and confirmatory test)	✦ Chest x-ray and CTA of chest
Therapy	✦ **Indications for repair** • Diameter: ≥ 5.5 cm • Expansion rate: ≥ 1 cm/year • Symptomatic aneurysm • Complications (e.g., rupture)	✦ **Indications for repair** • Diameter: ascending aneurysm ≥ 5.5 cm & descending aneurysm ≥ 6.5 cm • Expansion rate: ≥ 1 cm/year • Symptomatic aneurysm • Complications (e.g., rupture)

SHOCK

DEFINITION:

- A state of cellular & tissue hypoxia due to ↓ oxygen delivery and/or ↑ O2 consumption or ↓ O2 utilization.
- This most commonly occurs due to circulatory failure (↓ tissue perfusion).
- **Undifferentiated shock:** shock is recognized but the cause is unclear.

CLASSIFICATION:

1. CARDIOGENIC Shock:

⇨ **Intra-cardiac causes** of cardiac pump failure → ↓CO.

CARDIAC MUSCLE DISEASE	◆ AMI (Massive: > 40% of LV). ◆ DCM (é acute exacerbation of HF). ◆ Myocarditis.
ARRHYTHMIA	◆ Tachy-arrhythmia: VT. ◆ Brady-arrhythmia: CHB.
MECHANICAL DISEASE	◆ Severe valvular regurge (AR, MR). ◆ Severe valvular stenosis (AS, MS). ◆ Severe VSD, Rupture myo aneurysm.

2. OBSTRUCTIVE Shock:

⇨ **Extra-cardiac causes** of cardiac pump failure → ↓CO.

PULMONARY VASCULAR	MECHANICAL DISEASE
◆ Acute massive pulmonary embolism	◆ Tension pneumothorax. ◆ Pericardial tamponade.

3. HYPOVOLEMIC Shock:

⇨ **↓ Intravascular volume** (i.e, ↓ preload), →↓ CO.

HEMORRHAGIC (BLOOD LOSS)	NON-HEMORRHAGIC (FLUID LOSS)
◆ **Trauma.** ◆ **GIT bleeding** (Varices, Peptic ulcer). ◆ **Retroperitoneal bleeding** (ruptured aortic a). ◆ **Intraoperative & Postoperative** bleeding.	◆ **Skin:** Burns, ↑↑ sweating ◆ **GIT:** Vomiting, Diarrhea ◆ **Renal:** Osmotic D (DKA), Polyuria (DI) ◆ **3rd space**: Hypoalbuminemia, crush injury

4. DISTRIBUTIVE Shock:

⇨ Severe peripheral VD (Vasodilatory shock) → ↓ **Distribution** of blood flow

SEPTIC SHOCK	✦ Dysregulated host response to infection → life-threatening organ dysfunction. ✦ It is the ***most common cause*** of Distributive shock. ✦ **Responsible pathogens include:** 1. Gm +ve bacteria: Pneumococci, Staphylococci, Streptococci. 2. Gm-ve bacteria: Klebsiella, Pseudomonas, Haemophilus. 3. Fungi: Candida, Aspergillus. 4. Mycobacterium: Mycobacterium TB. ⇨ Associated é Systemic VD due to release of VD Inflammatory Mediators (NO & Cytokines)
NON-SEPTIC SHOCK	1. **Inflammatory shock:** Systemic Inflammatory Response Syndrome (SIRS), assoc. é systemic VD. ✦ Burns, Trauma, Pancreatitis. 2. **Neurogenic shock:** sympathetic tone → Unopposed parasympath response → ↓Vascular tone → VD. ✦ **CNS injury**, especially Spinal cord injury. ✦ **Severe pain** → Stimulation of the Parasympathetic nervous system. 3. **Anaphylactic shock:** Severe, potentially life-threatening allergic reaction (assoc. é systemic VD). ✦ **IgE-dependent immunologic mechanism:** Food (e.g. Fish, milk, egg), Drugs (e.g. Antibiotics). ✦ **IgE-independent immunologic mechanism:** Iron dextran. ✦ **Non-immunologic mechanism:** Radiocontrast agents. 4. **Toxic Shock Syndrome (TSS):** ✦ Potentially fatal due to release of toxins from Bacterial overgrowth. ✦ Streptococcus pyogenes (group A Streptococcus) or Staphylococcus aureus. 5. **Endocrine shock:** ✦ **Addisonian crisis:** ↓ α-1 receptor expression on arterioles 2ry to cortisol deficiency → VD.

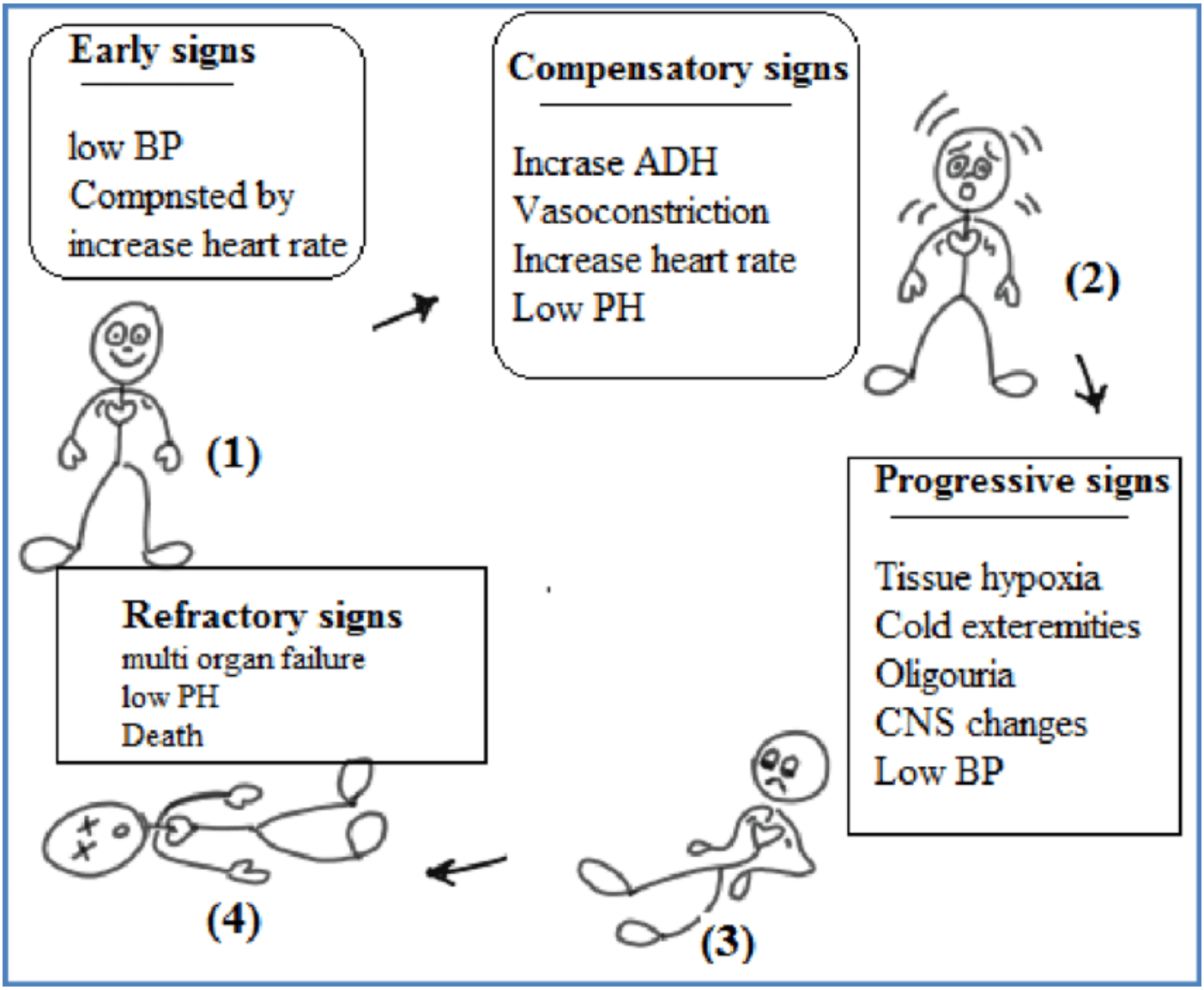
HEMODYNAMIC PROFILE:

TYPE OF SHOCK	What happens?	HEMODYNAMICS				
		Preload (R)	Preload (L)	Pump function	Afterload	Tissue perfusion
		CVP/RAP	PCWP/LAP	CO	SVR	SCO2
Cardiogenic	Heart fails to pump blood out	↑	↑	↓	↑	↓
Obstructive	Heart pumps well, but outflow is obstructed					
Pulmonary Vascular		↑	↓	↓	↑	↓
Mechanical disease		↑	↑	↓	↑	↓
Hypovolemic	Heart pumps well, but there is no enough blood volume to be pumped	↓	↓	↓	↑	↓
Distributive	Heart pumps well, but there is peripheral VD					
Septic		↓	↓	↑	↓	↑
Neurogenic		↓	↓	↓	↓	↓

- ⇨ **R:** right.
- ⇨ **L:** Left.
- ⇨ **CVP/RAP**: Central Venous Pressure/Right Atrial Pressure
- ⇨ **CO** Cardiac Output
- ⇨ **SVR:** Systemic Vascular Resistance
- ⇨ **PCWP/LAP** Pulmonary Capillary Wedge Pressure/Left Atrial Pressure (PCWP measured by pulmonary arterial catheter (Swan-Ganz catheter) provides an indirect estimate of LAP)
- ⇨ **SVO2** Mixed Venous Oxygen saturation (Oxygen content of blood that returns to the heart after meeting tissue needs)

STAGES

Preshock (Compensated)	✦ Compensatory mechanisms, e.g. Tachycardia & peripheral VC in early hypovolemic pre-shock ✦ Body switches from aerobic to anaerobic metabolism
Shock (Decompensated)	✦ Compensatory mechanisms fail ✦ Ss & Ss of organ dysfunction, e.g. Dyspnea, restlessness, diaphoresis, metabolic acidosis, hypotension, oliguria, and cool, clammy skin
End-organ dysfunction	✦ Irreversible organ damage, Multiorgan failure (MOF), e.g. AKI, ARDS, DIC ✦ Impending mortality



CLINICAL PICTURE & TREATMENT:

	BP	HR	RR	Temp	Skin	Urine	Mental status	Others	Treatment
Cardiogenic	↓	↑	↑	~	Pale, Cool, Clammy	↓	Altered	Dyspnea, Syncope ↓ JVP	⇨ Inotropes, ⇨ Mech. assist (IABP)
Obstructive	↓	↑	↑	~				P. paradox, ↓ HS ↓ JVP	⇨ TTT of cause, eg pericardio-centesis
Hypovolemic	↓	↑	↑	~				Thirst Ss of dehydration	⇨ Stop volume loss ⇨ Fluid resuscitation
Distributive									
Septic	↓	↑	↑	↑ or ↓	Flushed, Warm, Dry Then Pale, Cool, Clammy	↑ Then ↓	Altered	Bounding pulse	⇨ Fluid resuscitation ⇨ Antibiotics ⇨ Vasopressors
Neurogenic	↓	↓	↑	↑ or ↓	Flushed, Warm, Dry	↓		Neurologic deficits	⇨ Fluid resuscitation ⇨ Inotropes ⇨ Vasopressors

ANATOMY OF THE CORONARIES

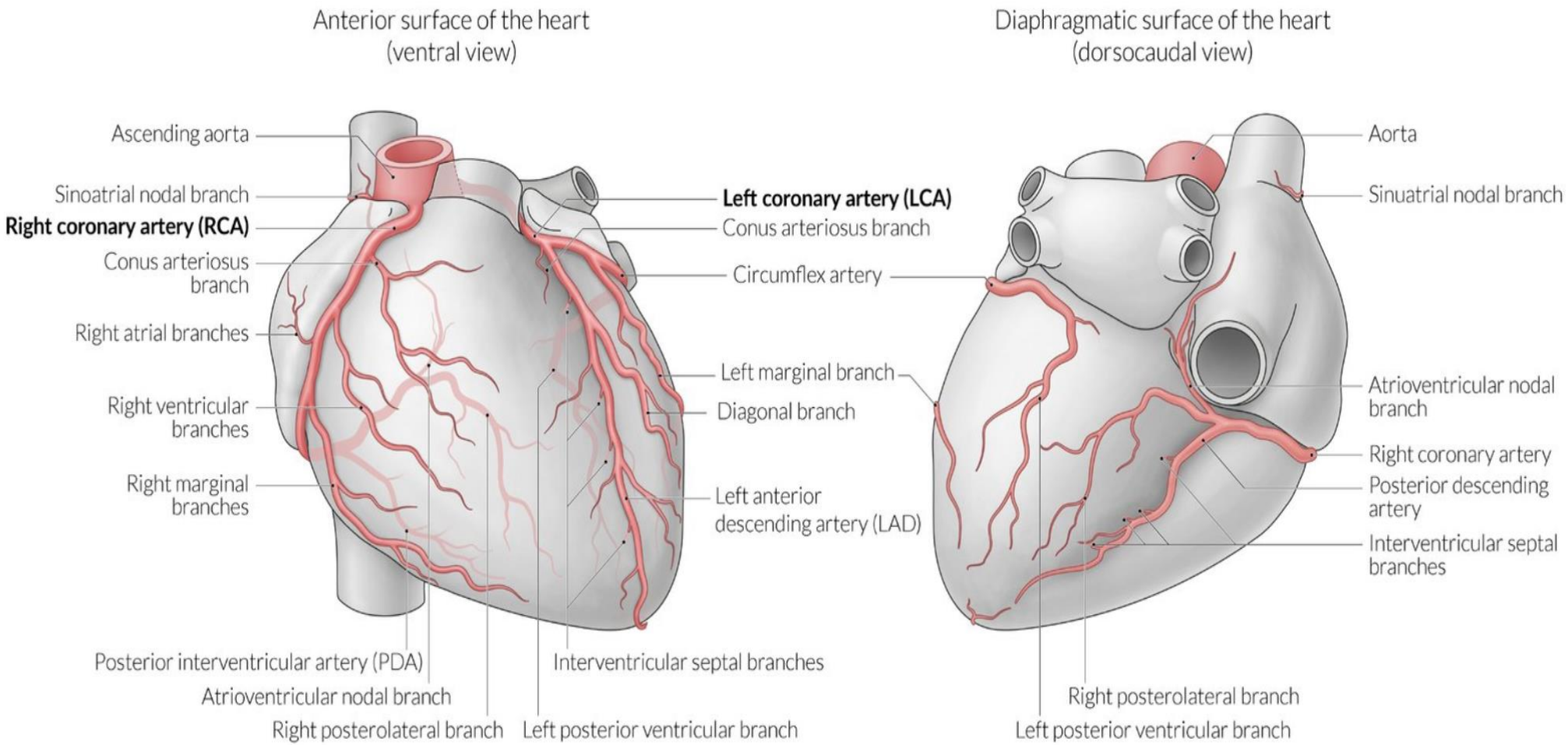
- The left and right coronary arteries arise from the **root of aorta** and supply the heart muscle with arterial blood.

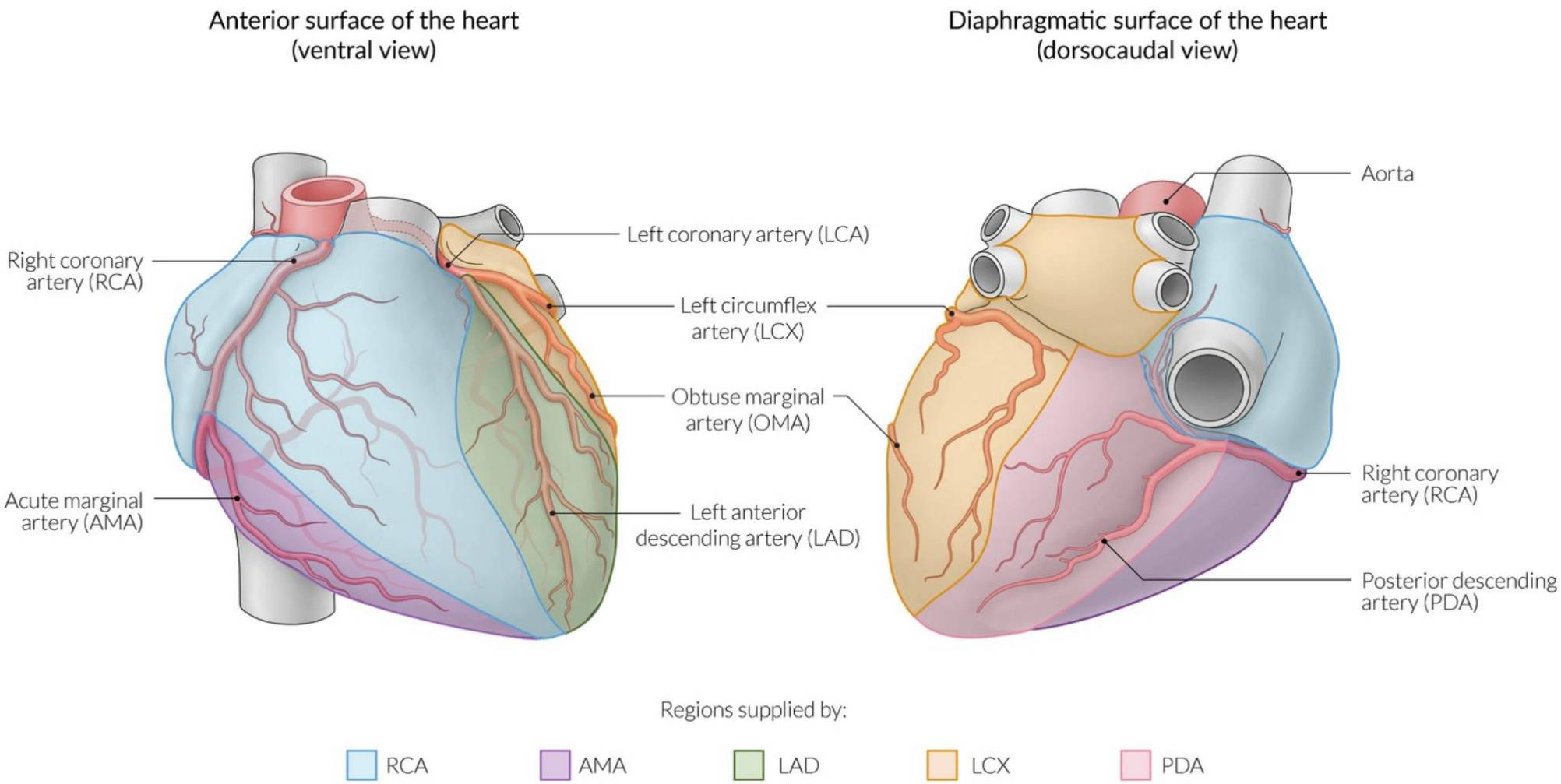
Coronary arteries			
	Source	**Important branches**	**Territory**
Left coronary artery (LCA)	Left aortic sinus of ascending aorta	• **Left anterior descending artery (LAD, LADA):** descends between right and left ventricles on anterior surface of heart (in anterior interventricular sulcus) towards cardiac apex⟶ gives off several diagonal branches on its course	• **>50%** of left atrium and ventricle • **Anterior** aspect of the left ventricle • **Anterior 2/3** of the interventricular septum • **Anterolateral** papillary muscle (also receives blood from LCX) • Cardiac apex
		• **Left circumflex artery (LCX):** courses left around the heart in the coronary sulcus towards the posterior aspect, ending before the posterior interventricular sulcus ⟶ gives off left marginal artery	• Posterolateral left atrium and ventricle • **Anterolateral** papillary muscle (also receives blood from LAD) • **In 40% of the population:** SA node • **In left-dominant and codominant circulation (15% of the population):** gives rise to PDA to supply posterior of interventricular septum, posteroinferior aspect of the heart, and **posteromedial** papillary muscle

	Source	Important branches	Territory
Right coronary artery (RCA)	Right aortic sinus of ascending aorta.	◆ **Various branches**	◆ **Majority of** right atrium and ventricle
		◆ **Right marginal artery:** courses along the diaphragmatic border (acute margini of the heart	◆ **Lateral** right ventricle and cardiac apex
		◆ **Posterior descending artery (PDA):** descends between right and left ventricles on posterior surface of heart (in the posterior interventricular sulcus) towards cardiac apex ◆ Can also originate from the LCX OR LCX and RCA (in left dominant and codominant circulation, respectively)	◆ **Posterior 1/3** of the interventricular septum ◆ **Pasteroinferior** aspect of heart ◆ **Posteromedial** papillary muscle
		◆ **Atrioventricular nodal artery**	◆ **AV node** (in a left dominant circulation, the AV node is supplied by the LCA) ◆ Bundle of His Iminor contribution from the LAD)
		◆ **Sinoatrial nodal artery**	◆ **SA node**

The LAD is the most commonly occluded coronary artery and is often referred to as the "widow maker" due to the high mortality rate associated with LAD infarction.

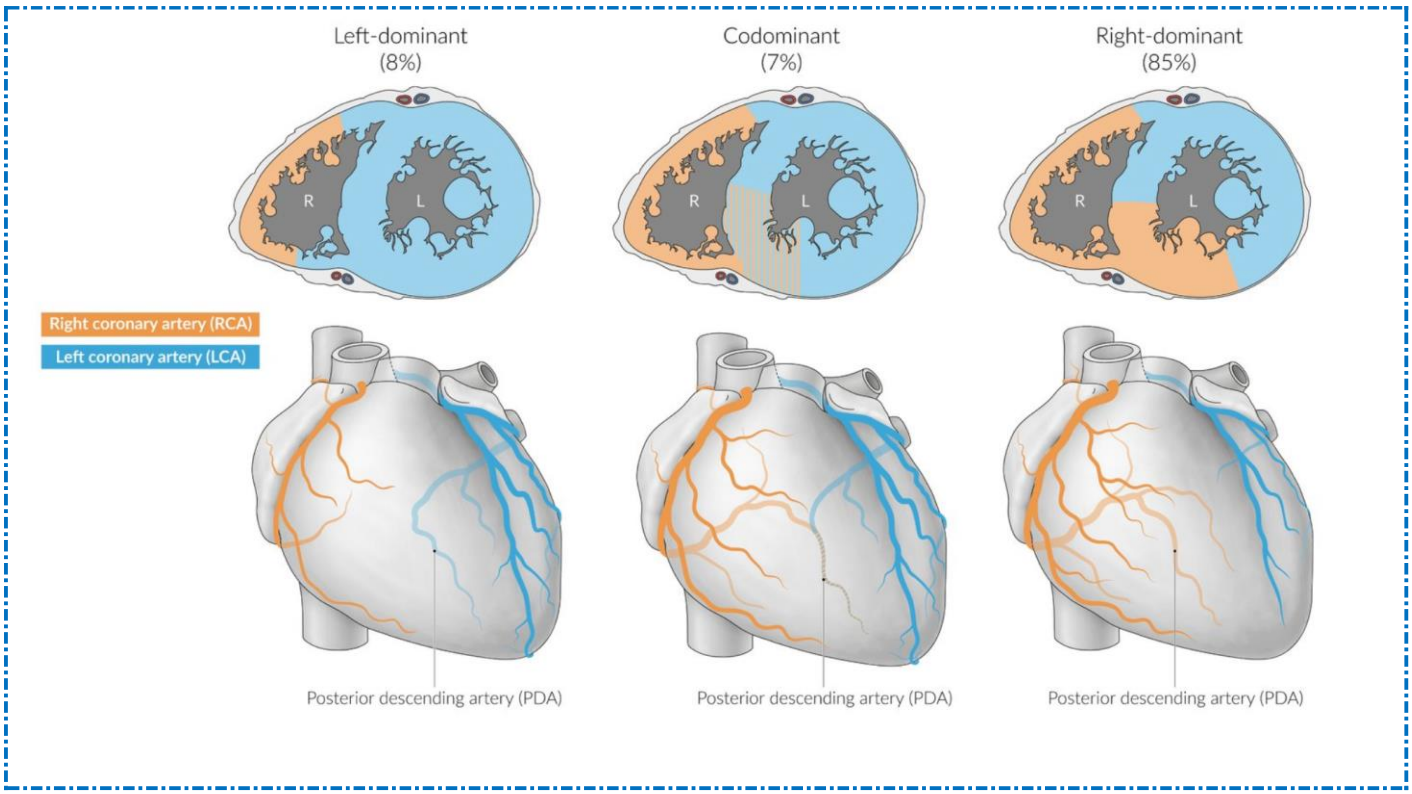
The **RCA** usually supplies the heart's conduction system (**sinus and AV node**) so that stenosis or occlusion of this vessel often leads to cardiac arrhythmias.


Anterior surface of the heart
(ventral view)
Ascending aorta
Sinoatrial nodal branch
Right coronary artery (RCA)
Conus arteriosus branch
Right atrial branches
Right ventricular branches
Right marginal branches
Posterior interventricular artery (PDA)
Atrioventricular nodal branch
Right posterolateral branch
Left coronary artery (LCA)
Conus arteriosus branch
Circumflex artery
Left marginal branch
Diagonal branch
Left anterior descending artery (LAD)
Interventricular septal branches
Left posterior ventricular branch
Diaphragmatic surface of the heart
(dorsocaudal view)
Aorta
Sinuatrial nodal branch
Atrioventricular nodal branch
Right coronary artery
Posterior descending artery
Interventricular septal branches
Right posterolateral branch
Left posterior ventricular branch


Anterior surface of the heart
(ventral view)
Diaphragmatic surface of the heart
(dorsocaudal view)
Right coronary artery (RCA)
Left coronary artery (LCA)
Left circumflex artery (LCX)
Obtuse marginal artery (OMA)
Acute marginal artery (AMA)
Left anterior descending artery (LAD)
Aorta
Right coronary artery (RCA)
Posterior descending artery (PDA)
Regions supplied by:
RCA
AMA
LAD
LCX
PDA

PATTERNS OF CORONARY SUPPLY :

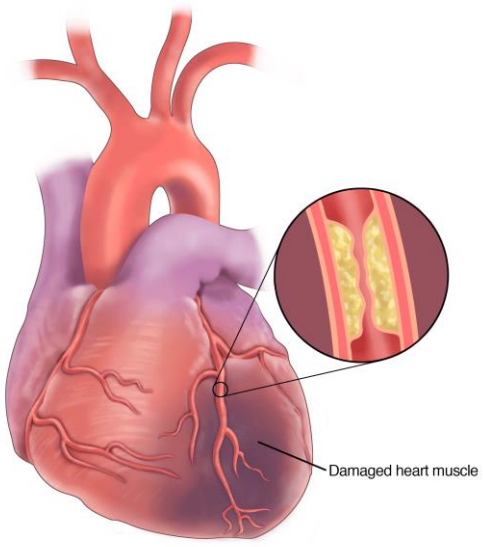
Right dominant	85%	◆ Posterior descending artery (PDA) Supplied by **RCA**
Left dominant	8%	◆ Posterior descending artery (PDA) Supplied by **left circumflex artery (LCX)**
Co-dominant	7%	◆ Posterior descending artery (PDA) Supplied by **both RCA & LCX**



ISCHEMIC HEART DISEASE

ETIOLOGY :

↓↓ myocardial oxygen supply	**A. DECREASED QUANTITY OF CORONARY BLOOD:** ◆ **CORONARY ARTERY DISEASE:** • Coronary atherosclerosis **(MOST COMMON CAUSE).** • Others: ➢ Arteritis e.g. PAN, SLE. ➢ Blood diseases, (Hypercoagulable) e.g. PRV, DIC. ➢ Congenital anomalies. ➢ Dissection. ➢ Emboli. ➢ Spasm of the coronary **(GOOD PROGNOSIS).** ◆ **Non- coronary artery disease (LOW CO) :** • Any cause of low CO, especially: AS. **B. DECREASED QUALITY OF CORONARY BLOOD:** ◆ Anemia. ◆ Cyanotic conditions (hypoxia).
↑↑ myocardial oxygen demand	◆ ↑ **Myocardial contractility:** e.g. Ventricular hypertrophy. ◆ ↑ **Preload:** e.g. Hyperdynamic circulation. ◆ ↑ **Afterload:** e.g. Systemic hypertension. ◆ ↑ **HR:** Tachycardia. ◆ **Severe stress:** physical, mental.



Atherosclerosis

DEFINITION :

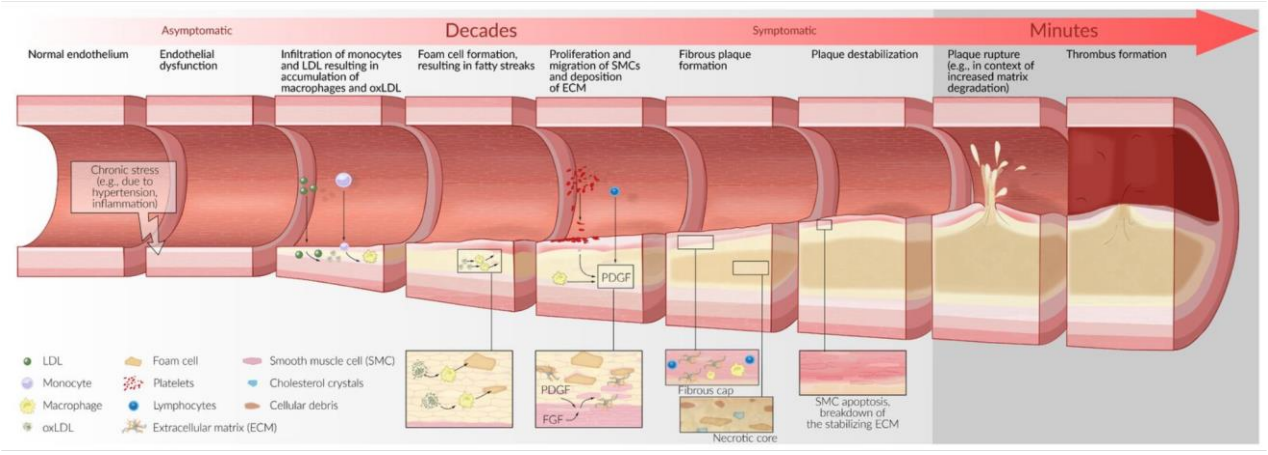
- Process in which lipids (**cholesterol**) are deposited in **intima** causing **atheroma** ⇒ gradual narrowing of arterial lumen ⇒**ischemia** and **necrosis** of the tissues.
- The already present **collaterals** open to bypass the narrow segment.

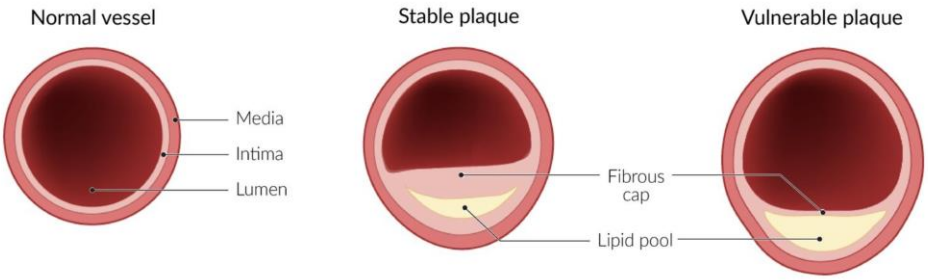
LESIONS OF ATHEROSCLEROSIS :

Early Lesion (fatty streak)	CONTAINS TWO TYPES OF CELLS: ◆ **FOAM CELLS:** macrophages filled with lipids. ◆ **T-LYMPHOCYTES:** and some smooth muscle cells.
Advanced lesion (fibrous plaque)	◆ Thickened cap of dense CT. ◆ Large number of smooth muscle cells.
Far advanced (complicated) lesion:	◆ Degeneration, calcification. ◆ Ulcers, cracks, fissures & **Plaque Rupture**. ◆ Occlusion of the lumen due to: plaque rupture or thrombosis.

COMPLICATIONS :

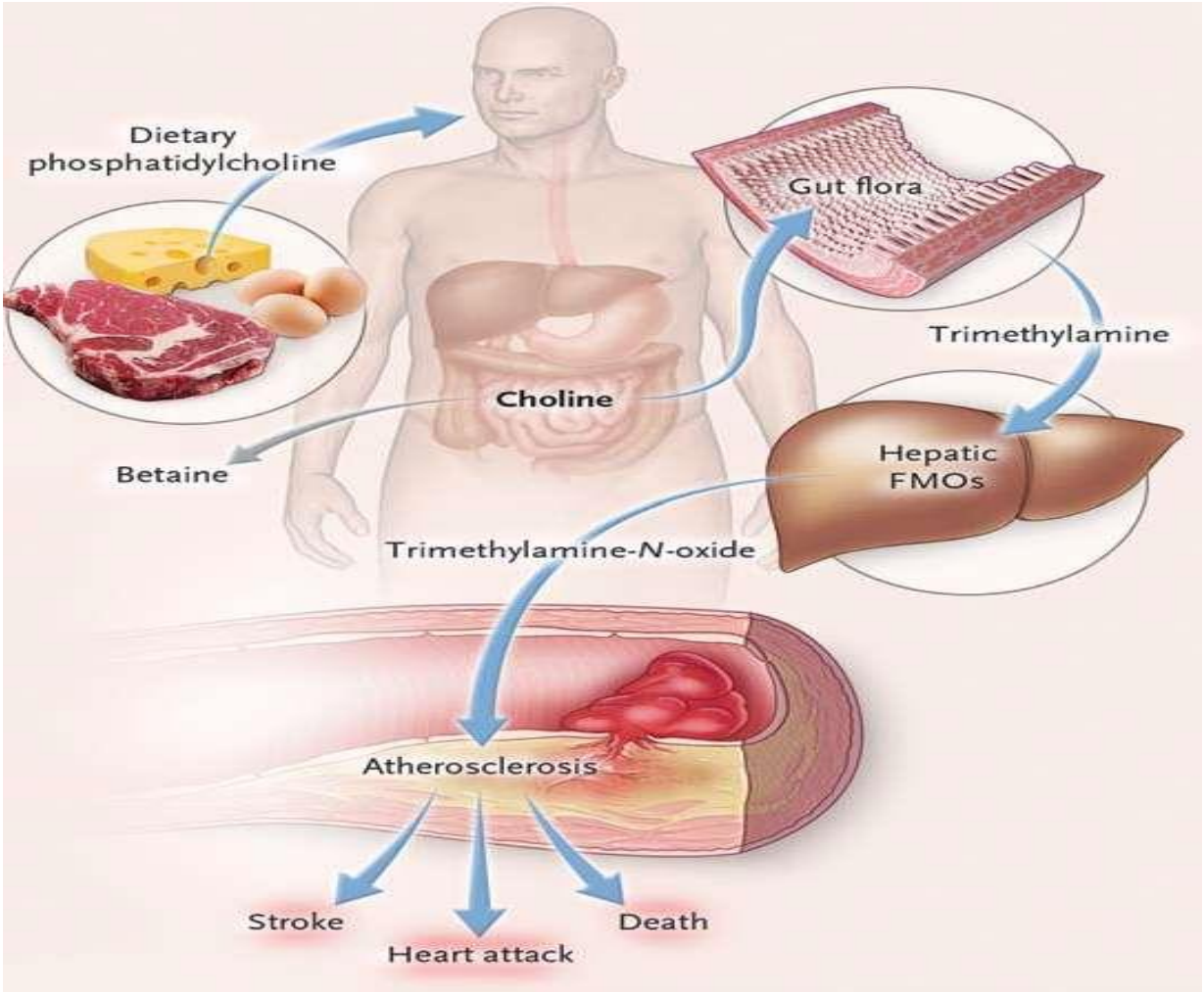
a. Coronary Artery Disease (**CAD**).
b. Cerebro vascular Disease (**CVD**).
c. Peripheral Arterial Disease (**PAD**).





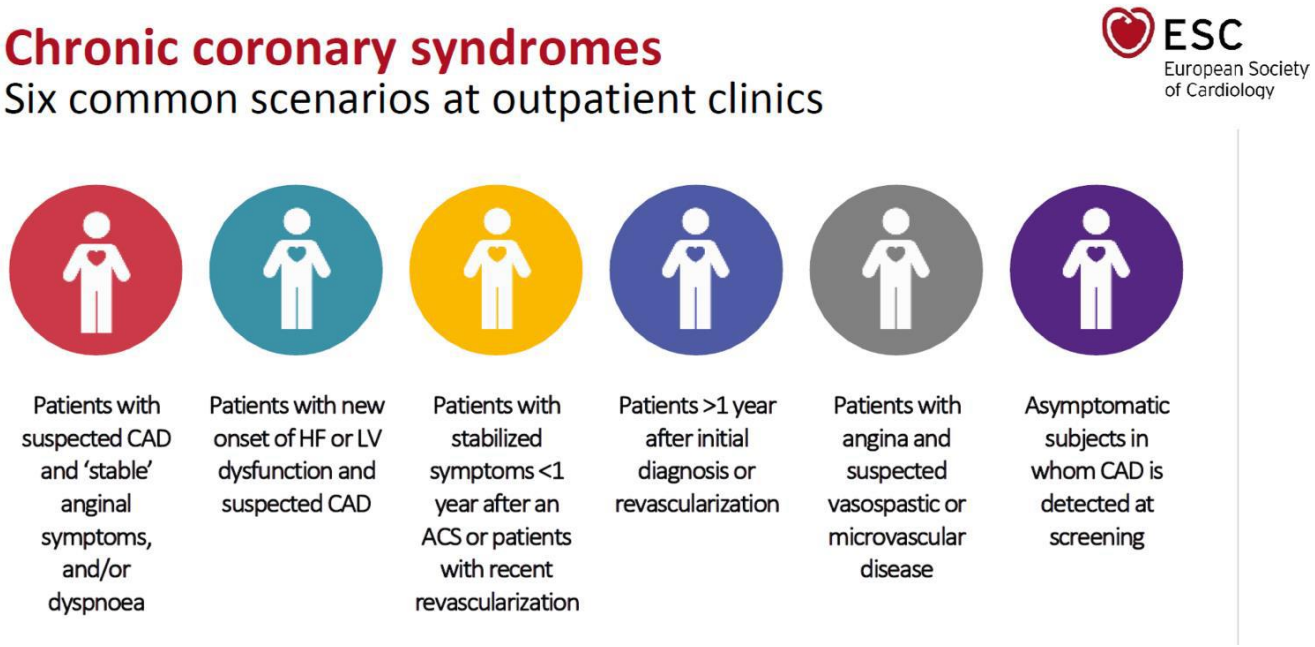
RISK FACTORS :

Non - modifiable	◆ **Age:** above 40 years. ◆ **Sex:** male : female= 5 : 1. ◆ **Type A personality :** Nervous. intellectual. ◆ **Genetic factors:** positive family history.	
Modifiable	**High risk**	**Less risk**
	◆ **Dyslipidemia:** ↑ total cholesterol, ↑ LDL, ↓ HDL. ◆ **Diabetes mellitus.** ◆ **Hypertension:** causes endothelial damaged. ◆ **Cigarette smoking.**	◆ **Obesity.** ◆ **Diet:** rich in saturated fat & cholesterol. ◆ **P**hysical inactivity. ◆ **P**sychological stress. ◆ Contraceptive **P**ills. ◆ **H**yperuricemia. ◆ **H**omocysteinemia. ◆ **H**eavy alcohol intake.

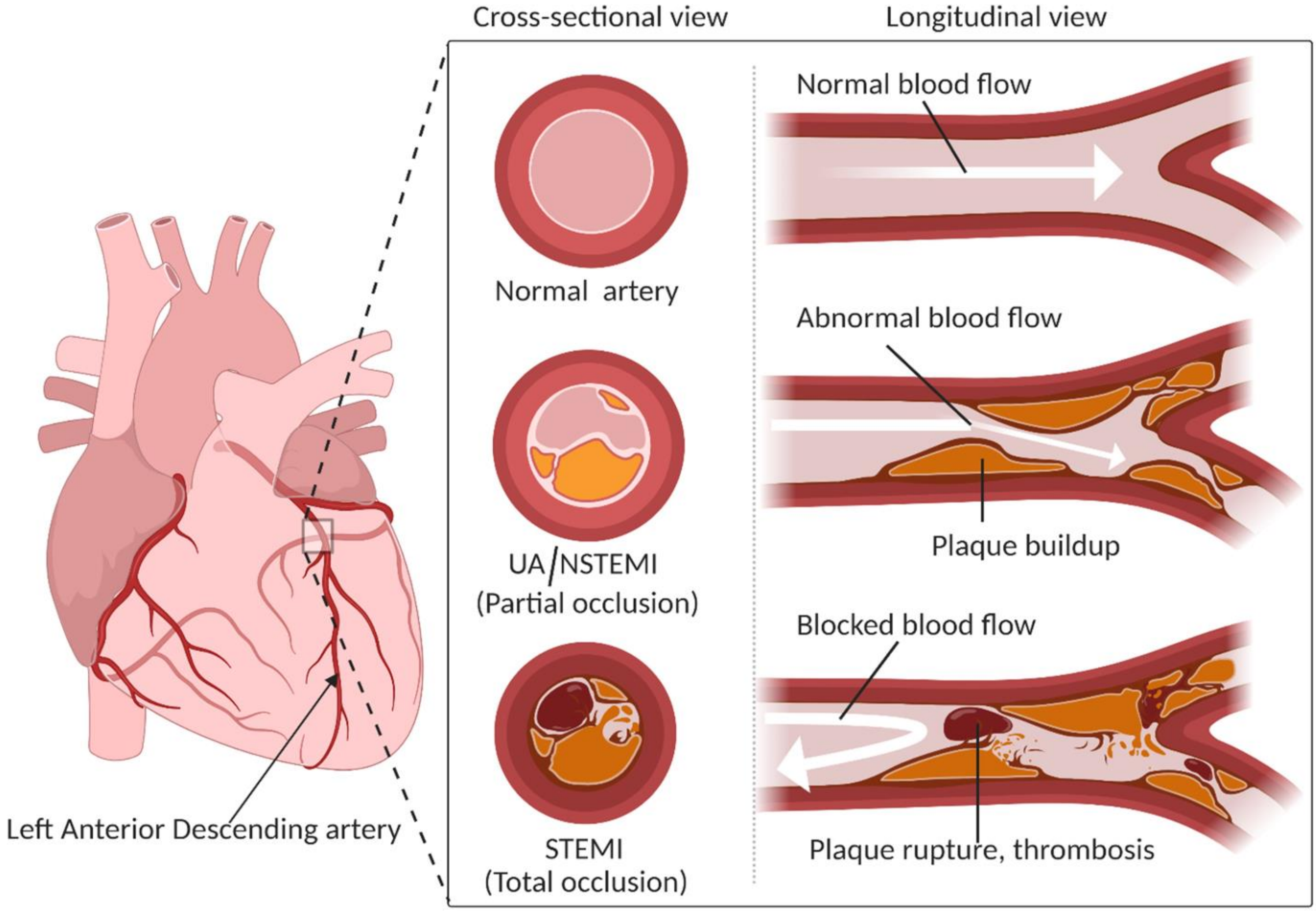


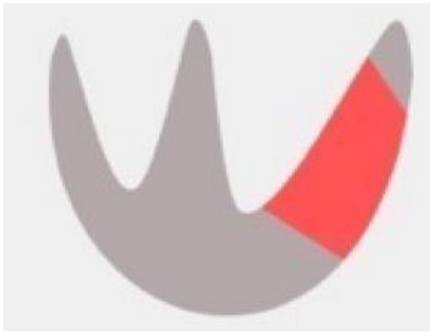
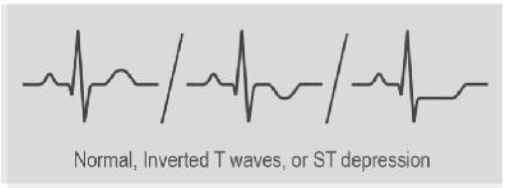
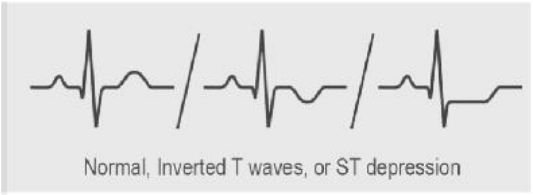
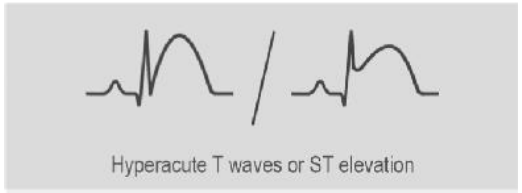
Presentation of IHD

CHRONIC CORONARY SYNDROME (CCS) :



ACUTE CORONARY SYNDROME (ACS) :



	Unstable angina	NSTEMI	STEMI
Patho physiology	Plaque ruptures ⇒ thrombus formed ⇒ Partial vascular occlusion ⇒		Plaque ruptures ⇒thrombus forms ⇒ Complete Vascular occlusion
	Supply ischemia (no infarct)	Subendocardial infarct	Transmural infarct
			
ECG			
Enzymes	Normal	Elevated	Elevated

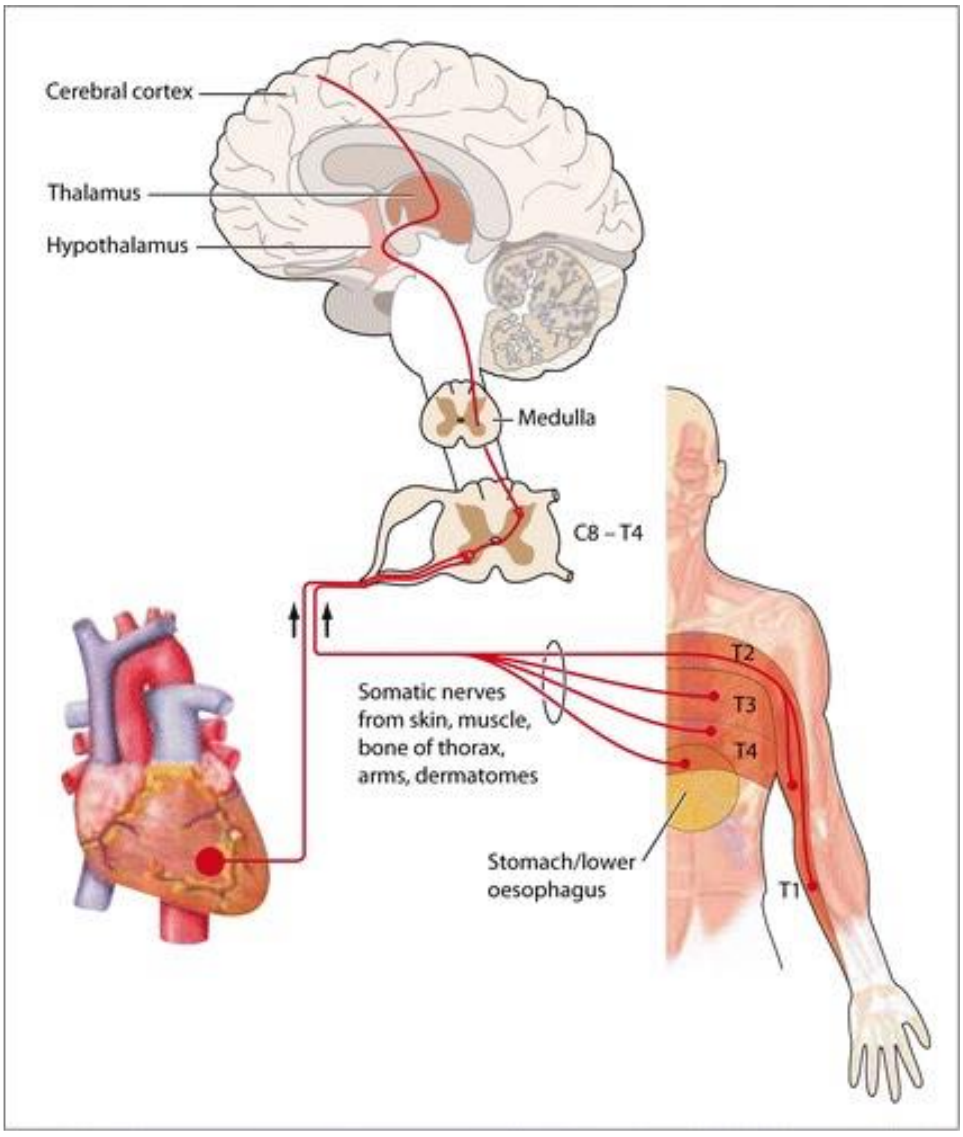
ANGINA PECTORIS

DEFINITION :

- Attacks of **chest pain,** caused by **myocardial ischemia,** usually lasting for several minutes, and **not resulting in myocardial necrosis.**

WHAT CAUSES THE PAIN?

- When myocardial hypoxia occurs, metabolites accumulate & stimulate nerve endings in the myocardium to discharge impulses along sympathetic fibers to the lower cervical & upper thoracic segments of the spinal cord with subsequent radiation to the corresponding dermatomes, producing sensation of pain coming from these areas
- Pain may be **ABSENT** in **DIABETES (Silent ischemia):** This is due to autonomic neuropathy



CLINICAL PICTURE :

A. SYMPTOMS

- **Chest Pain** with the following **7** criteria :

	Common	Less Common	Never
1.SITE	• **Retro sternal** • Often patient **place** his clenched hand over upper sternum	• Any site of chest • Scapular • Infraclavicular • epigastrium	• Left inframammary • Patient never point with his finger
2.CHARACTER	• Compressing • constricting	• Heaviness • Squeezing • Burning • discomfort	• Stitching • Pricking • throbbing
3.RADIATION	• Left shoulder & inner side of lt arm up to little finger • Neck, jaw, or teeth	• Right shoulder • Back • Epigastric area	• Below epigastrium
4.DURATION	• <15 min (1-5) min	• >15 min	• Never sec or hours

5. PRECIPITATING FACTORS:

- Exercise
- Cold weather
- Heavy meals
- Smoking
- Sexual intercourse
- Stress

6. RELIEVING FACTORS:

- **Rest,** but occasionally the pain disappears with continuous exercise (walk through angina)
- Sublingual nitrates.

7. ASSOCIATION:

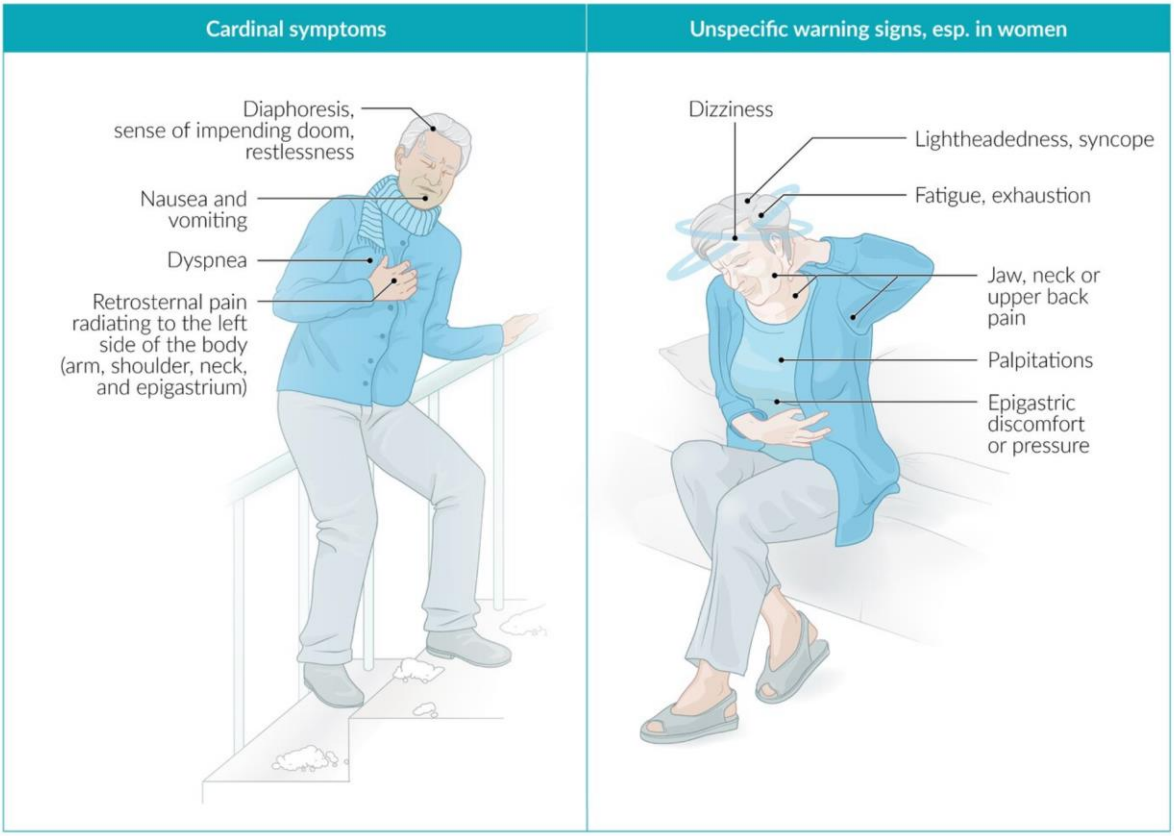
- Sweating
- Dizziness
- Dyspnea: may occur due to LVF
- Fear of death (angina animi)
- Eructation at the end of the attack

TRADITIONAL CLINICAL CLASSIFICATION OF CHEST PAIN :

Typical angina (definite)	◆ **Meets all three of the following characteristics :** • Substernal chest discomfort of characteristic quality & duration. • **Provoked by** exertion or emotional stress. • **Relieved by** rest and/or nitrates within minutes.
Atypical angina (probable)	◆ Meets **two** of these characteristics.
Non-anginal chest pain	◆ **Lacks** or meets **only one** or none of characteristics.

B. SIGNS:

General signs	◆ Pallor, sweating. ◆ Transient tachycardia and hypertension. ◆ **A positive Levine sign :** characterized by the patient's fist clenched over the sternum when describing the pain.
Cardiac signs	◆ **S1 :** Weak. ◆ **S2 :** Reversed splitting. ◆ **S4 :** (due to decreased ventricular compliance by ischemia). ◆ **Signs of complications:** e.g. systolic murmur of MR .



SEVERITY :

CLASS I:	◆ Angina on marked activity, nothing occurs on ordinary activity.
CLASS II:	◆ Slight limitation of physical activity on ordinary effort.
CLASS III:	◆ Marked limitation of physical activity on less than ordinary effort.
CLASS IV:	◆ Angina may be present even at rest.

TYPES:

Stable or Classical (typical) angina	◆ The pain is relatively constant as regards severity, precipitating factors and relief.
Unstable angina	◆ **Worsening angina (crescendo angina):** increased frequency, severity or duration of a preexisting stable angina. ◆ **Angina starting to occur at rest** (or with minimal exertion). ◆ Angina of **RECENT** onset (within 2 months). ◆ Angina **RESISTENT** to therapy. ◆ It carries a bad prognosis as 20% will pass to AMI within several months. ◆ It is considered an **intermediate syndrome** between stable angina and AMI.
Angina of Lewis	◆ In cases of **aortic regurge.** ◆ It is **nocturnal.**
Anginal Equivalent Syndrome	◆ **DEFINITION:** • A symptom of cardiac ischemia (other than chest pain) that is associated with exertion or stress, or relieved by short-acting nitrates or rest ◆ **POSSIBLE MANIFESTATIONS** • **Pain referred to** left arm, neck, jaw. epigastric region, or back • Gastrointestinal discomfort • Dysprea • Dizziness, palpitations Restlessness, anxiety • Autonomic symptoms (e.g diaphoresis, nausea, vomiting, syncope) ◆ Patients with chest paint or anginal equivalents should be evaluated for CAD. Other indications include newly diagnosed heart failure arrhythmia, and syncope.

<table>
<tr>
<td>Variant angina: (Prinzmetal, Vasospastic angina)</td>
<td>

Angina that occur due to spasm of a coronary artery.
DIAGNOSTIC CRITERIA:

<table>
<tr><td>Typical clinical features</td><td>Spontaneous angina with a rapid response to short-acting nitrates and ≥ 1 of the following:
Occurrence at rest (especially at night or early morning
Precipitated by hyperventilation.
Responsive to treatment with CCBs (but not beta blockers)
Reported lower exercise tolerance in the morning</td></tr>
<tr><td>Transient ischemic ECG changes</td><td>One of the following ECG changes in≥ 2 contiguous leads during an anginal episode:
ST elevation or ST depression ≥ 0,1mV (1mm)
New negative U waves</td></tr>
<tr><td>Coronary spasm on angiography</td><td>Coronary artery constriction >90% associated with angina and ischemic ECG changes
Can occur spontaneously during coronary angiography
Can be induced by coronary artery spasm provocation testing</td></tr>
</table>

Definitive vasospastic angina: the presence of typical clinical features as well as documentation of either transient ischemic ECG changes or coronary spasm during coronary angiography
Suspected vasospastic angina: the presence of typical clinical features as well as equivocal or unavailable transient ischemic ECG changes and equivocal coronary artery spasm criteria, Le, changes are seen but do not meet the criteria listed above.

</td>
</tr>
<tr>
<td>Syndrome X</td>
<td>

Typical, exertional angina with positive exercise stress test.
Anatomically normal coronary arteries
Reduced capacity of vasodilation in microvasculature
Long term prognosis very good.
Calcium channel blockers and beta blockers effective.

</td>
</tr>
<tr>
<td>Silent Ischemia</td>
<td>

Very common & Difficult to diagnose
More episodes of silent than painful ischemia in the same patient
Holter monitor & Exercise testing

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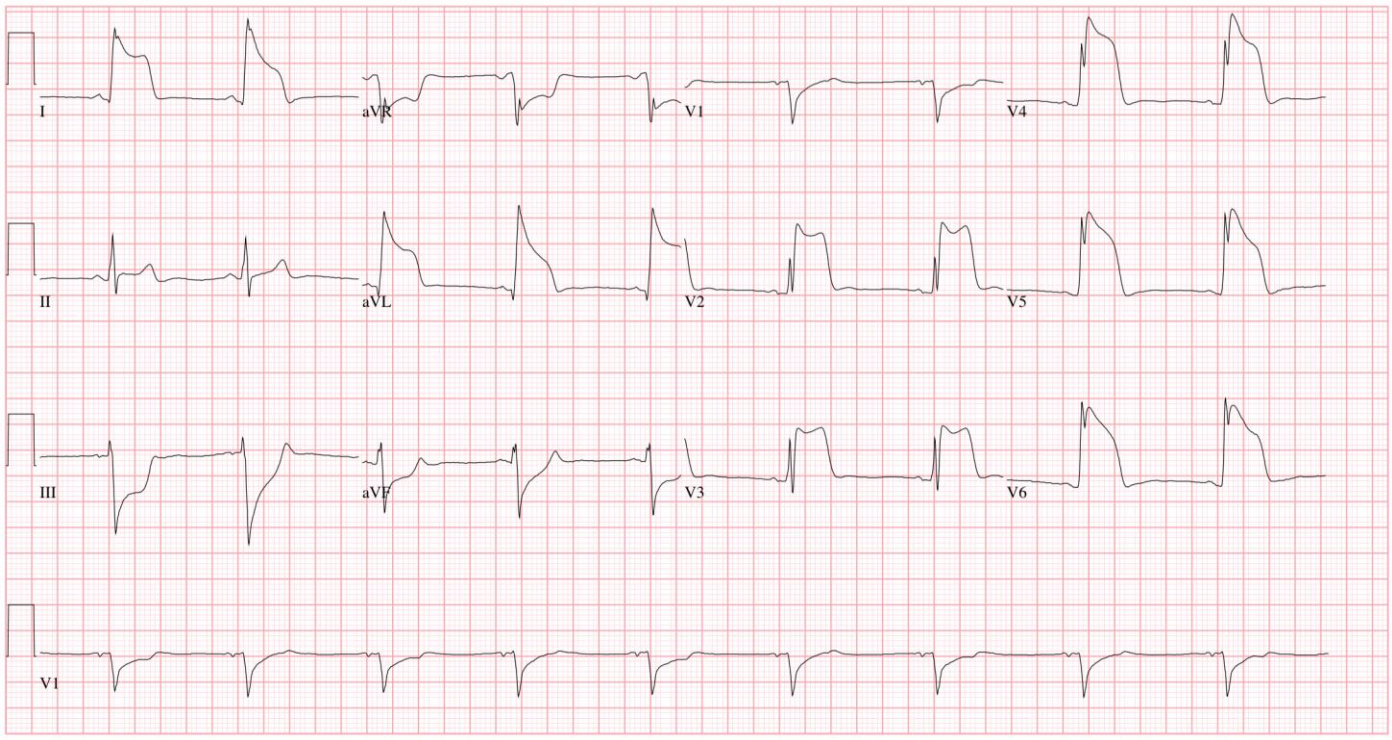


PRINZMETAL ANGINA.

INVESTIGATIONS :

1. ECG:

Resting	◆ **Between attacks:** usually normal. ◆ **During an attack:** • ST segment: depressed (elevated in variant angina). • T wave: inverted. • Arrhythmias or heart block.
Ambulatory	◆ For diagnosis of **variant** angina.
Exercise	◆ Clinical provocation of myocardial ischemia using a treadmill, and recording the ECG changes

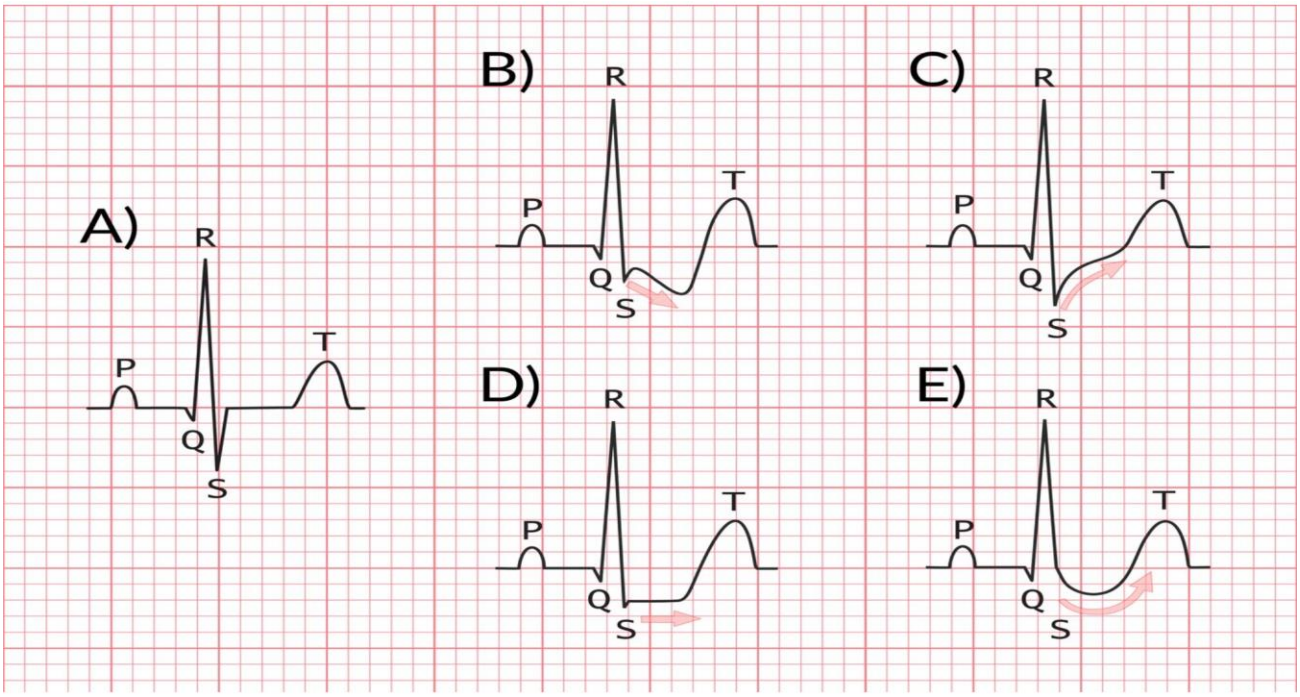
◆ **THE TEST IS POSITIVE FOR MYOCARDIAL ISCHEMIA IF:**

1. Typical anginal pain occurs.
2. ST segment depression occurs.
3. Ventricular arrhythmias occur.

◆ **CONTRAINDICATIONS:**

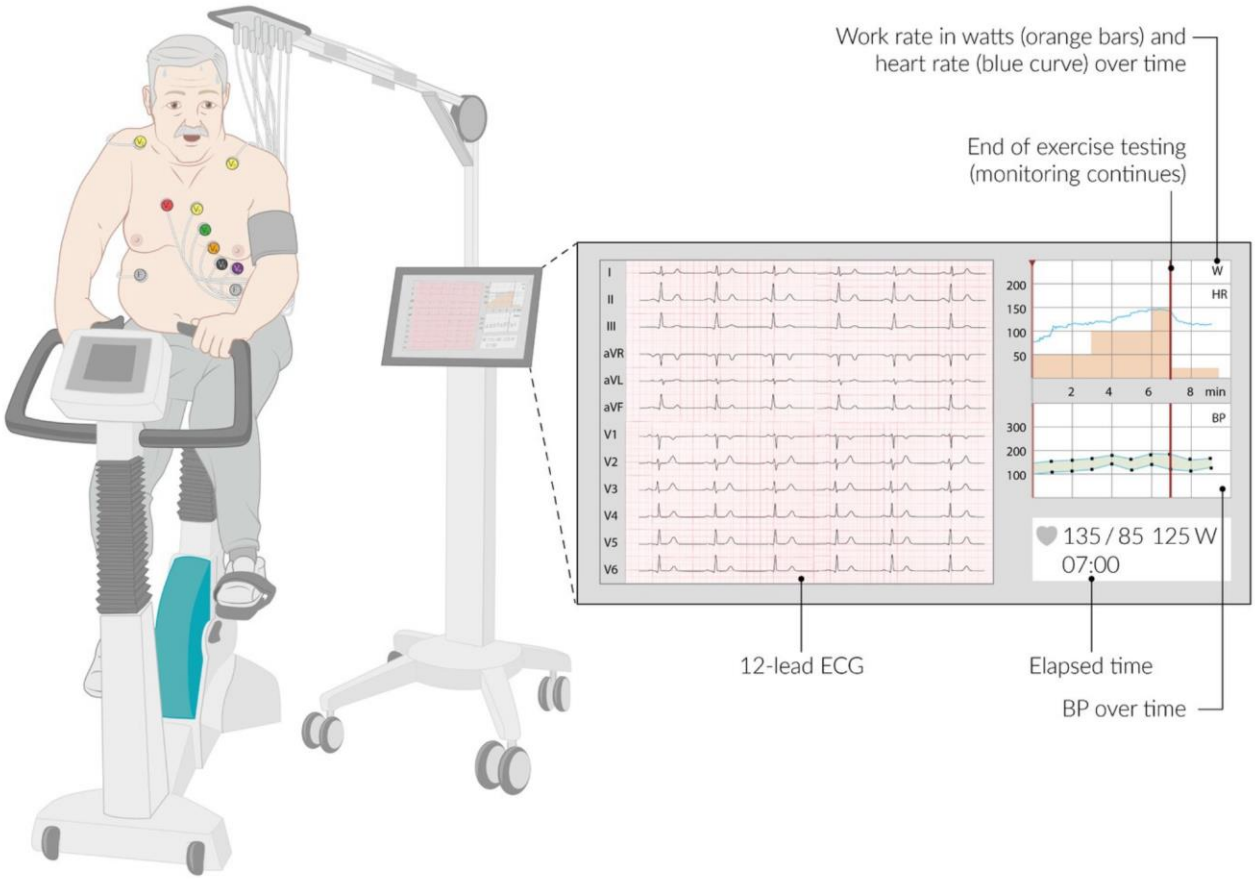
1. Severe aortic stenosis.
2. Severe hypertension.
3. Heart failure.
4. Unstable angina & recent MI (1 week).

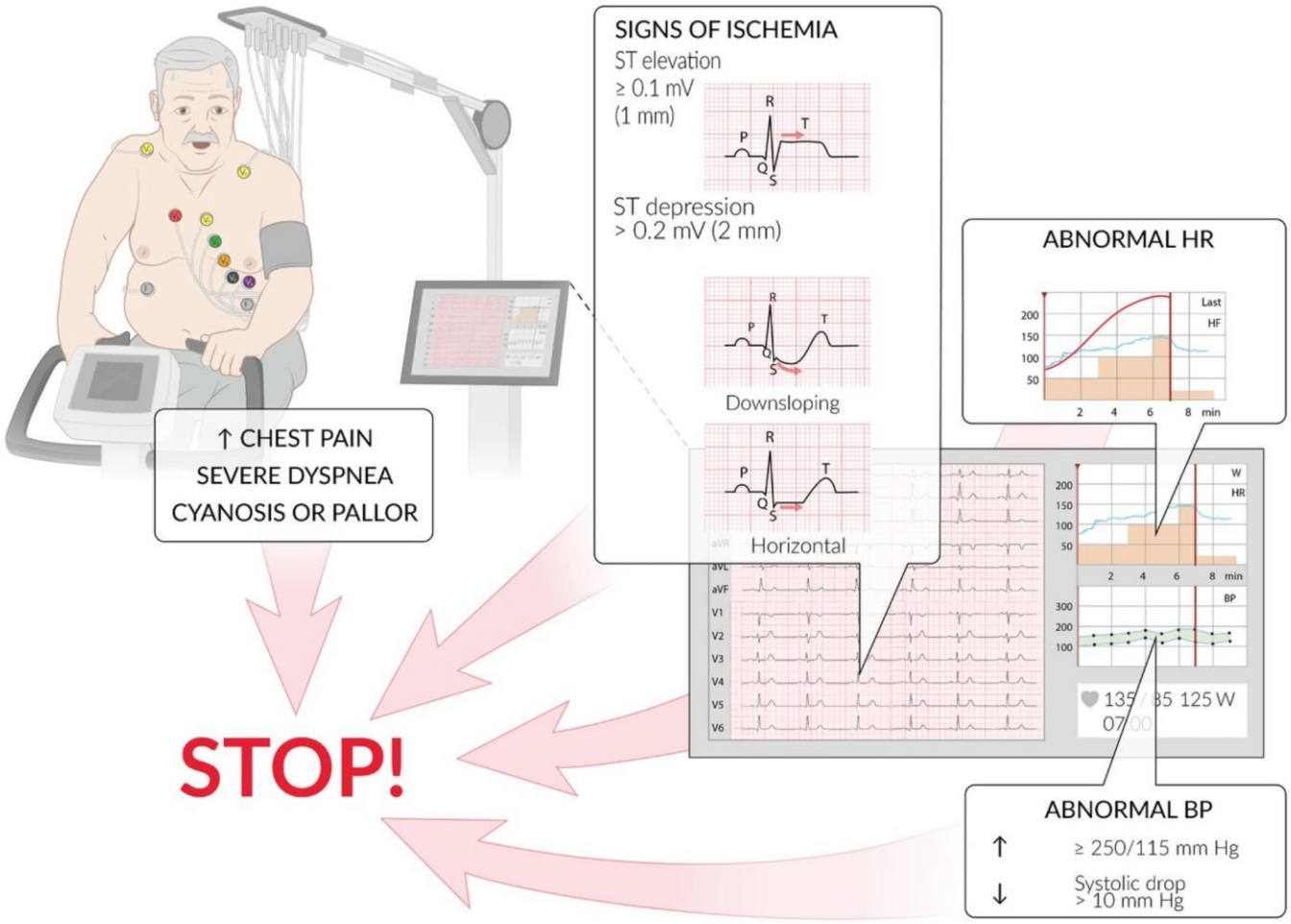
◆ **LIMITATIONS:** "false positivity & false negativity"



Types of ST segment depression

ECG schematic (paper speed: 25 mm/s)
(A) Normal ST segment
(B) Downsloping ST depression (e.g., myocardial ischemia)
(C) Upsloping ST depression (e.g., physical activity, associated with ischemia if prominent t-waves)
(D) Horizontal ST depression (e.g., myocardial ischemia)
(E) Sagging ST depression (e.g., digoxin toxicity)





INDICATIONS FOR EARLY TERMINATION OF CARDIAC EXERCISE STRESS TEST

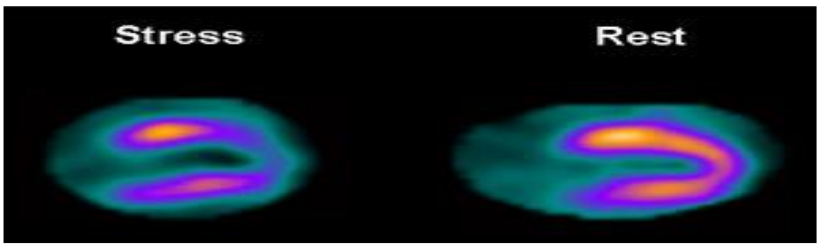
Clinical	◆ Moderate to severe angina, severe dyspnea, cyanosis, pallor ◆ Abnormal BP: a drop in systolic BP of 10 mm Hg compared with baseline or an exaggerated hypertensive response with systolic BP 250 mm Hg or diastolic BP 115 mm Hg
ECG	◆ Exercise-induced ischemia (ST elevations or depressions) ◆ Excessive or delayed increase in heart rate

-

2. MYOCARDIAL PERFUSION IMAGING :

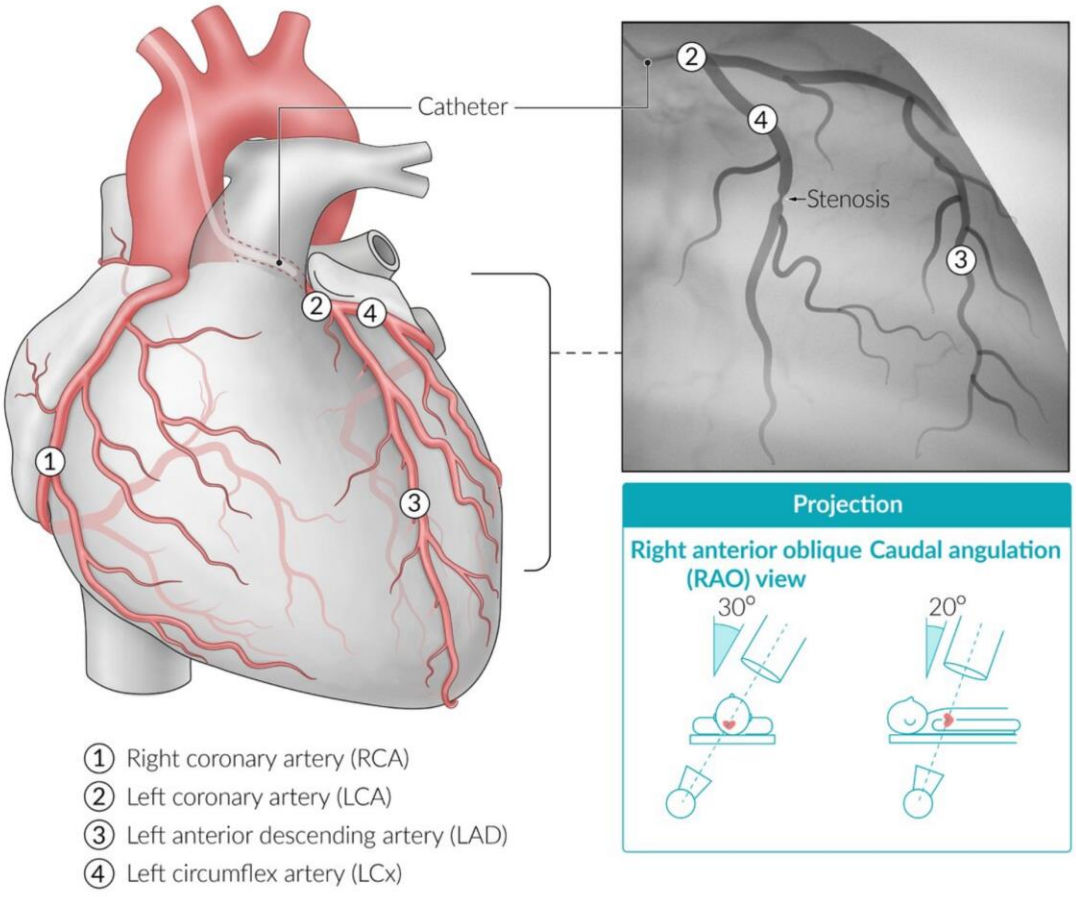
= RADIOACTIVE SCAN = NUCLEAR STRESS TEST

SPECT3	Usual traces include Thallium201 or Technetium-sestamibi99m
PET4	Usual tracer is 18F flurpiridaz
Ischemia appears as a filling defect (Regional ischemia) e stress & disappears at rest	

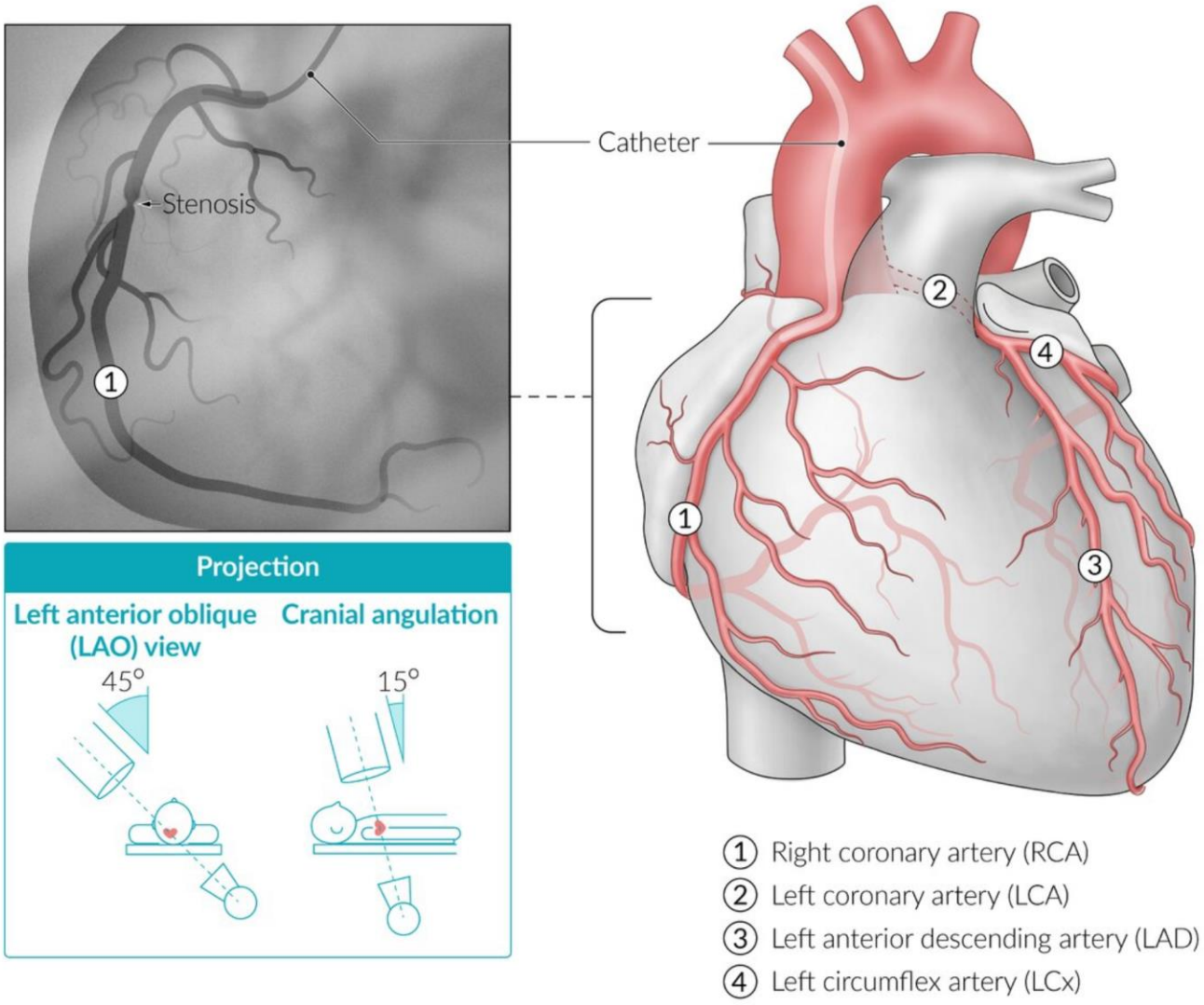


3. CORONARY ARTERIOGRAPHY:

Aim	◆ It demonstrates the site and extent of coronary occlusion
Indications	◆ **Angina which is:** indicated for coronary revascularization. ◆ **Angina which is:** unstable, variant & post-infarction angina. ◆ Recurrent chest pain of uncertain etiology.
Complications	◆ Myocardial infarction. ◆ Embolization. ◆ Arrhythmias. ◆ Arterial dissection.



CORONARY ANGIOGRAPHY (LEFT CORONARY ARTERY).



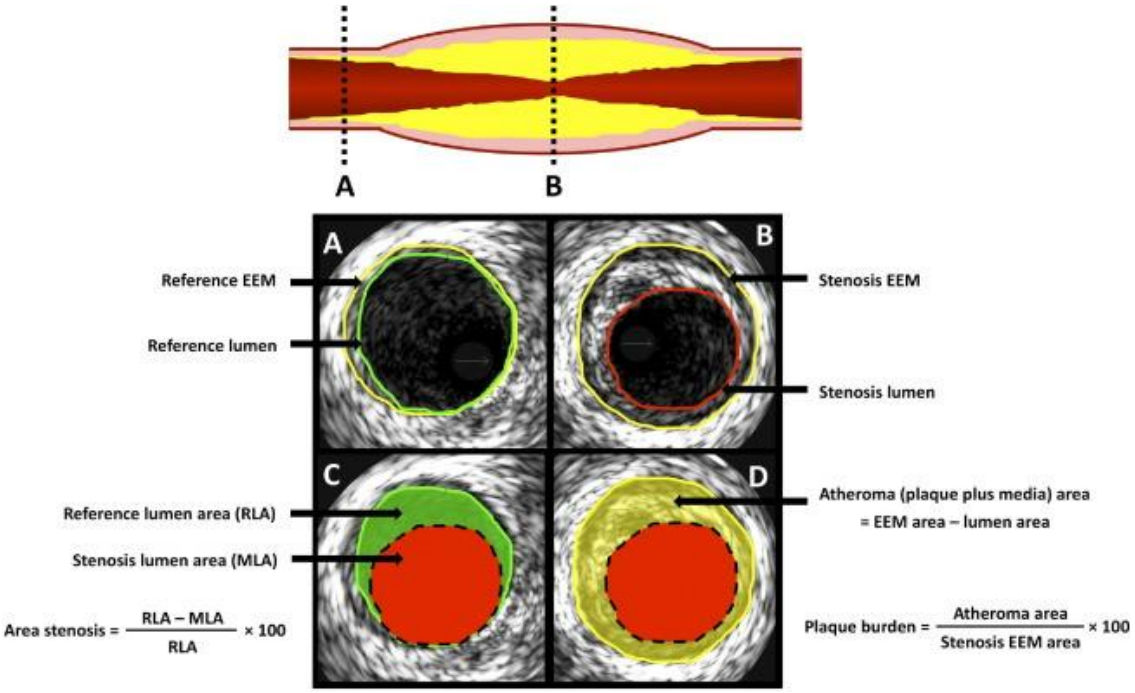
CORONARY ANGIOGRAPHY (RIGHT CORONARY ARTERY).

4. STRESS ECHOCARDIOGRAPHY:

Methods	◆ Exercise. ◆ IV dobutamine: for people who are unable to exercise.
Result	◆ May show RWMA in ischemic areas (Hypokinesia).

5. RECENT iMAGiNG:

- **Multi - slice CT,** Magnetic resonance coronary angiography (MR - CA).
- **IVUS.**

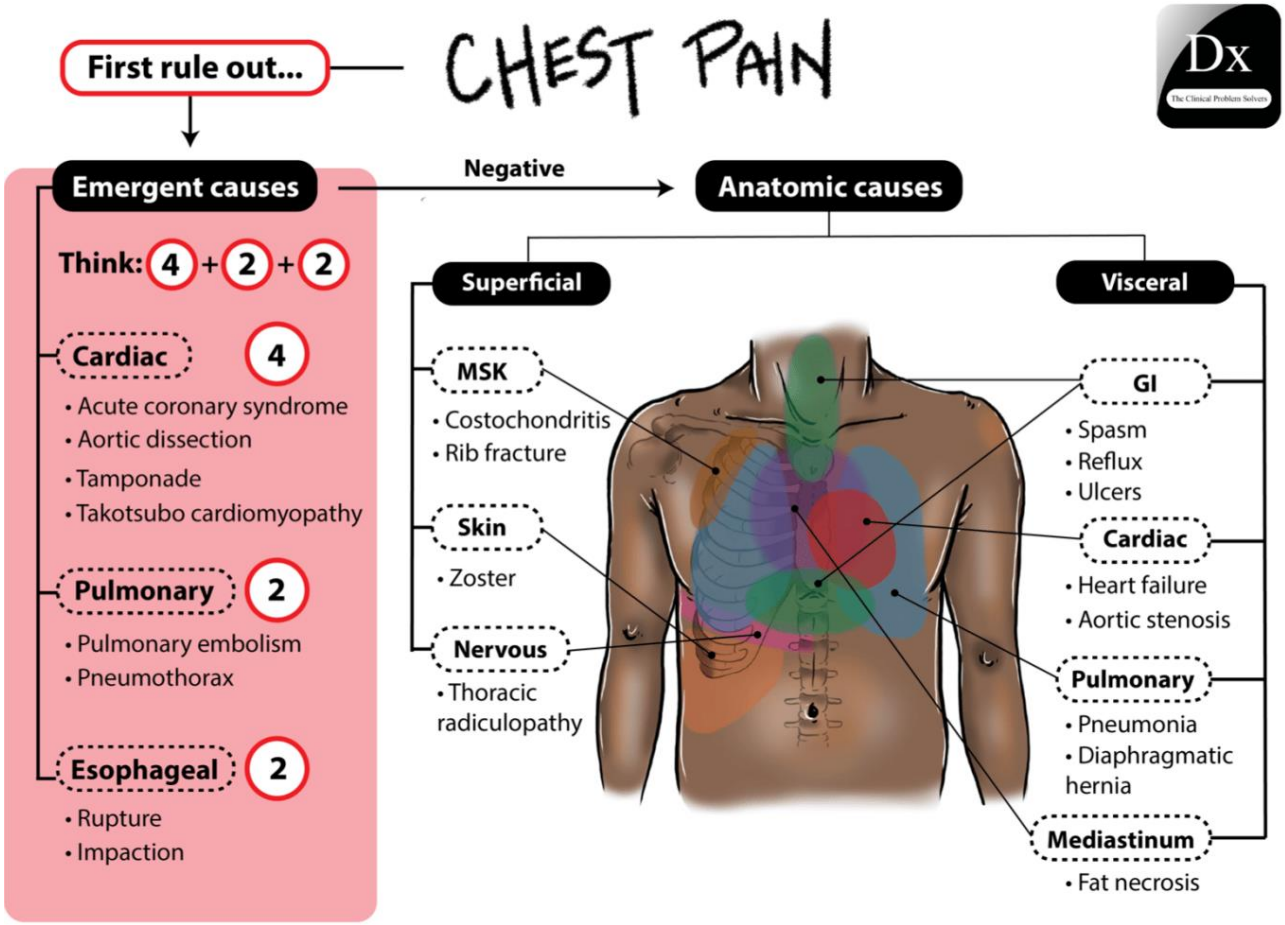


6. BiOCHEMiCAL SCREENiNG: (BLOOD TESTS)

Aim	◆ **For identification of risk factors such as:** 1. **Dyslipidemia:** lipids (TC, LDL, HDL, TG). 2. **Hyperglycemia:** glucose. 3. **Hyperuricemia:** uric acid. 4. **Homocysteinemia:** homocysteine. ◆ **Cardiac enzyme:** normal in angina (↑ in AMI)

DIFFERENTIAL DIAGNOSIS: Causes of acute chest pain

- **Myocardium:** Angina, AMI.
- **Pericardium:** Acute pericarditis.
- **Endocardium:** Mitral valve prolapse.
- **Aorta:** Dissecting aortic aneurysm.
- **Pulmonary:** Acute pulmonary embolism.
- **Pain of cardiac neurosis.**
- **Acute pleurisy.**
- **Acute massive lung collapse** & pneumothorax,
- **Oesophageal pain:** GORD.
- **Peptic ulcer & cholecystitis.**
- **Herpetic pain (Herpes zoster).**
- **Musculo-skeletal disorders** e.g. Tietze syndrome.



Treatment

	DURING THE ATTACK
Rest	◆ **Complete REST**
Sublingual Drugs (Nitrates)	◆ **Examples :** Nitroglycerin 0.5 mg && Isosorbide dinitrate 5 mg ◆ **Mechanism:** • Dilating the coronaries. • ↓ the work of the heart through: venodilator effect.

	INBETWEEN THE ATTACKS
General Measures	◆ **TTT of the cause & correction of the risk factors:** • **Cause:** e.g. Vasculitis, AS, HTN. • **Risk factors:** e.g. Dyslipidemia, Diabetes, HTN.
Non - pharmacologic therapy	◆ **Moderation of life:** Avoid precipitating factors. ◆ **Smoking:** must be stopped. ◆ **Diet control:** Restriction of cholesterol & saturated fat. ◆ **Physical activity:** "REGULAR EXERCISE" • Improves the collateral coronary blood flow. • Improves the lipid profile. • ↓ the heart rate, BP & myocardial O2 demands. • ↓ platelet aggregation, ↑ fibrinolytic activity.

I. Specific measures: "pharmacologic therapy"

AiM:

- **To ↑ myocardial O2 supply** (dilate the coronary arteries),
- **To ↓ myocardial O2 consumption (↓ work of heart),** by using anti-anginal drugs.
- **To guard against** the development of AMI (thrombosis), by using antiplatelets and / or anticoagulants.
- **TTT of Dyslipidemia.**

DRUGS:

Classic therapies	New therapies
◆ Antianginal drugs ◆ Antiplatelets	◆ Medical ◆ Non-medical

I. Classic therapies:

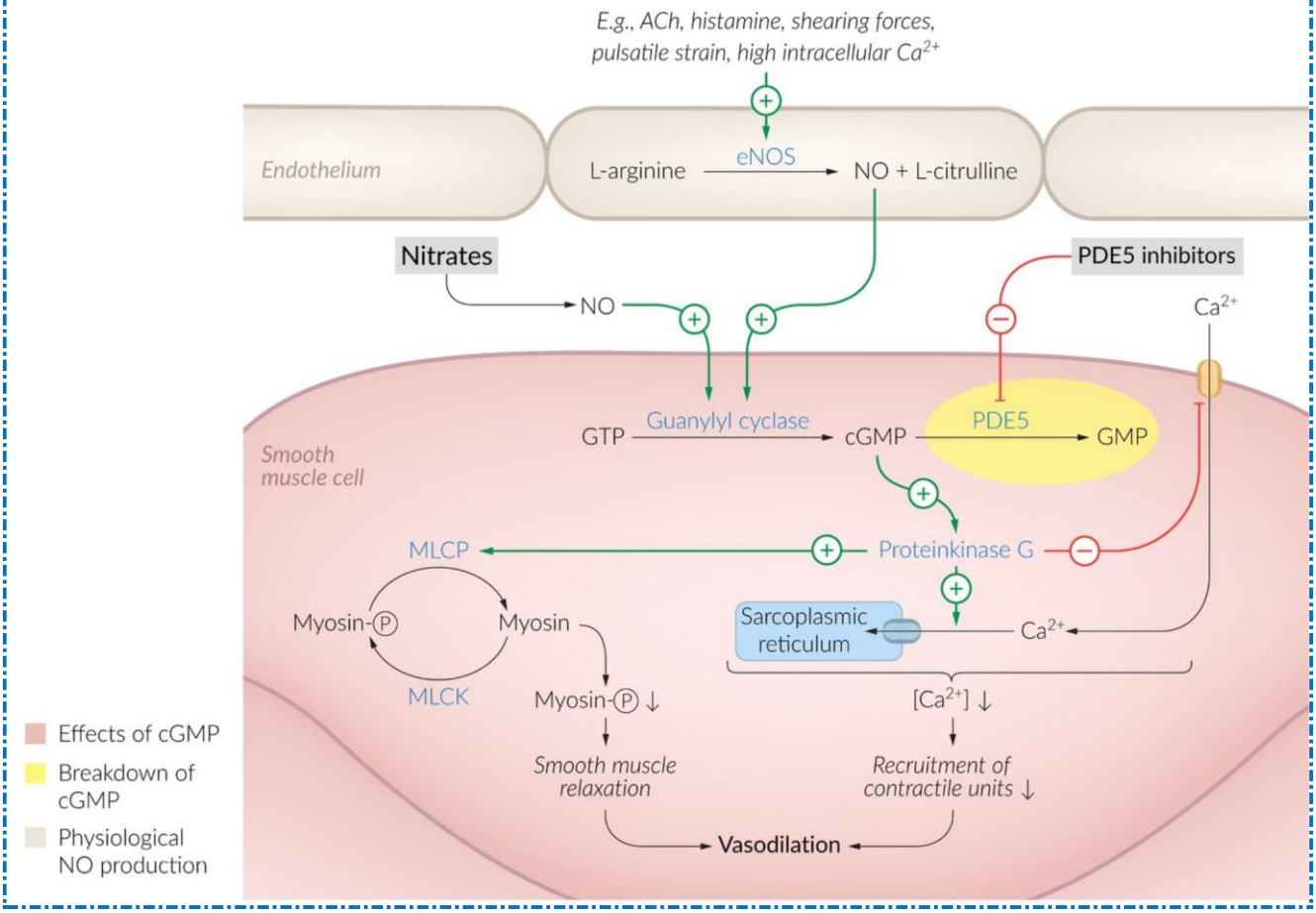
- **Anti-anginal drugs:** "nitrates, beta blockers, ccb
- **Antiplatelets & anticoagulants.**

ANTIPLATELETS & ANTICOAGULANTS:

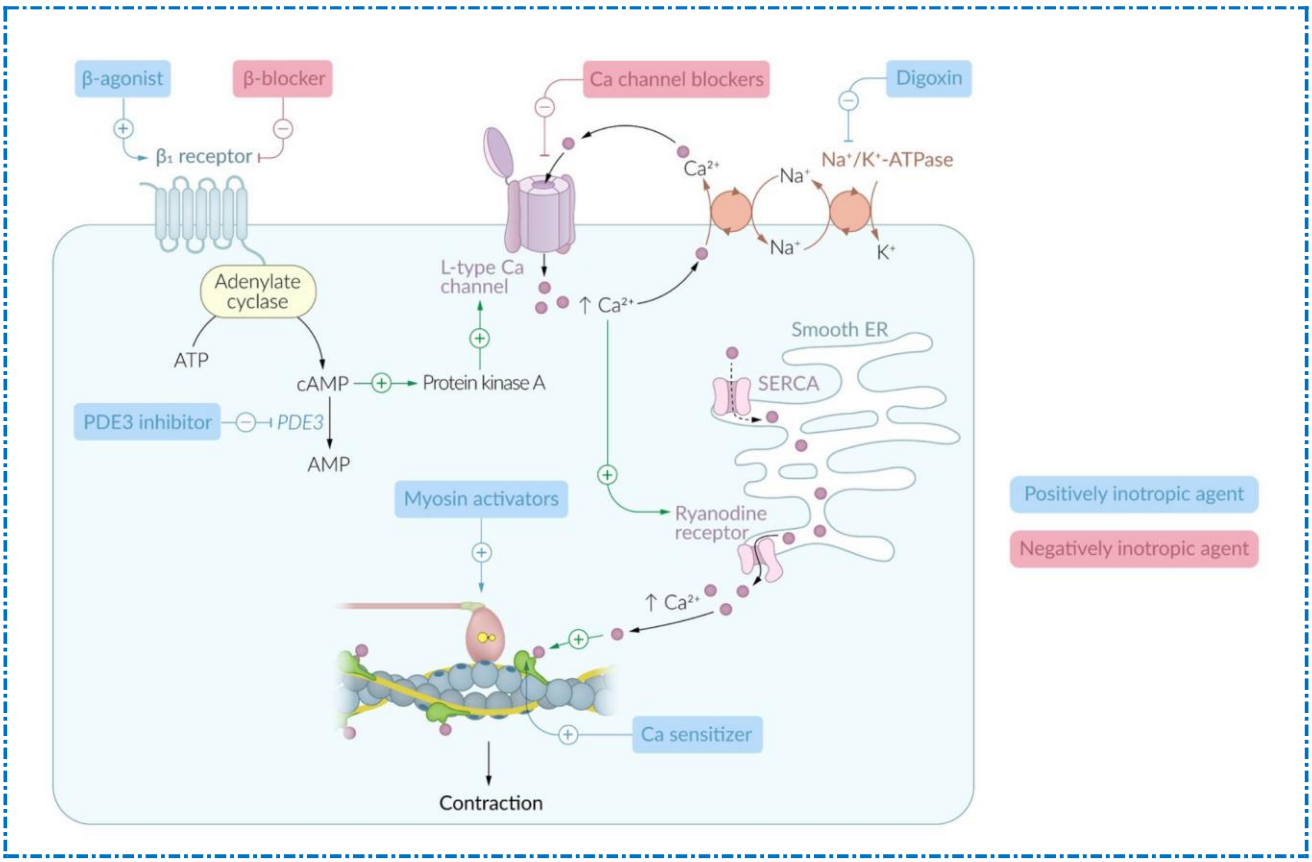
Aspirin (low dose):	75 - 300 mg 1 day (single dose).
Dipyridamole	75 mg twice daily
Ticlopidine	250 mg twice daily.
Clopidogrel	75 mg once daily. protect against AMI & death especially in patients with UA
Heparin	IV heparin (with antiplatelets) is given in UA.

ANTI-ANGINAL DRUGS: "NITRATES, BETA BLOCKERS, CCB

	Nitrates	Beta – blockers	CCBs
Actions	◆ Dilate the coronary arteries. ◆ Reduce preload.	◆ ↓ HR & ↓ BP. ◆ ↓ myocardial contractility.	◆ Dilate coronary arteries (extremely effective in VA). ◆ Reduce after load. ◆ Reduce the myocardial contractility.
Preparations	◆ Nitroglycerine: 2.5 mg / 8 hours orally, or TP. ◆ Isosorbide dinitrate: 20 - 40 mg twice daily, or TP. ◆ Isosorbide mononitrate: 20 mg twice daily, or SR 100 mg	◆ Non-selective: Propranolol, 60 mg / 8 hours. ◆ Cardio-selective: Atenolol, 50-100 mg / day.	◆ Verapamil: 80 mg / 8 hours. ◆ Diltiazem : 60 mg / 8 hours. ◆ Nifedipine : 10 mg / 8 hours. ◆ Amlodipine: 5 mg / day.
Side effects	◆ **Severe headache.** ◆ **Tolerance (hyporesponsiveness):** • Use the smallest effective dose. • Allow a nitrate-free interval (8 h).	◆ Chest: Bronchospasm. ◆ Heart: Bradycardia, Hypotension, Heart block. ◆ Others: • Fatigue, Reversible impotence, • Masking signs of hypoglycemia in diabetes.	◆ Headache & flushing. ◆ Hypotension & aggravation of heart failure. ◆ REVERSIBLE OEDEMA OF LLs. ◆ **Bradycardia:** with verapamil & diltiazem. ◆ **Tachycardia:** with nifedipine.



NITRATE AND PHOSPHO DIESTRASE 5 INHIBITOR



INOTROPIC DRUGS

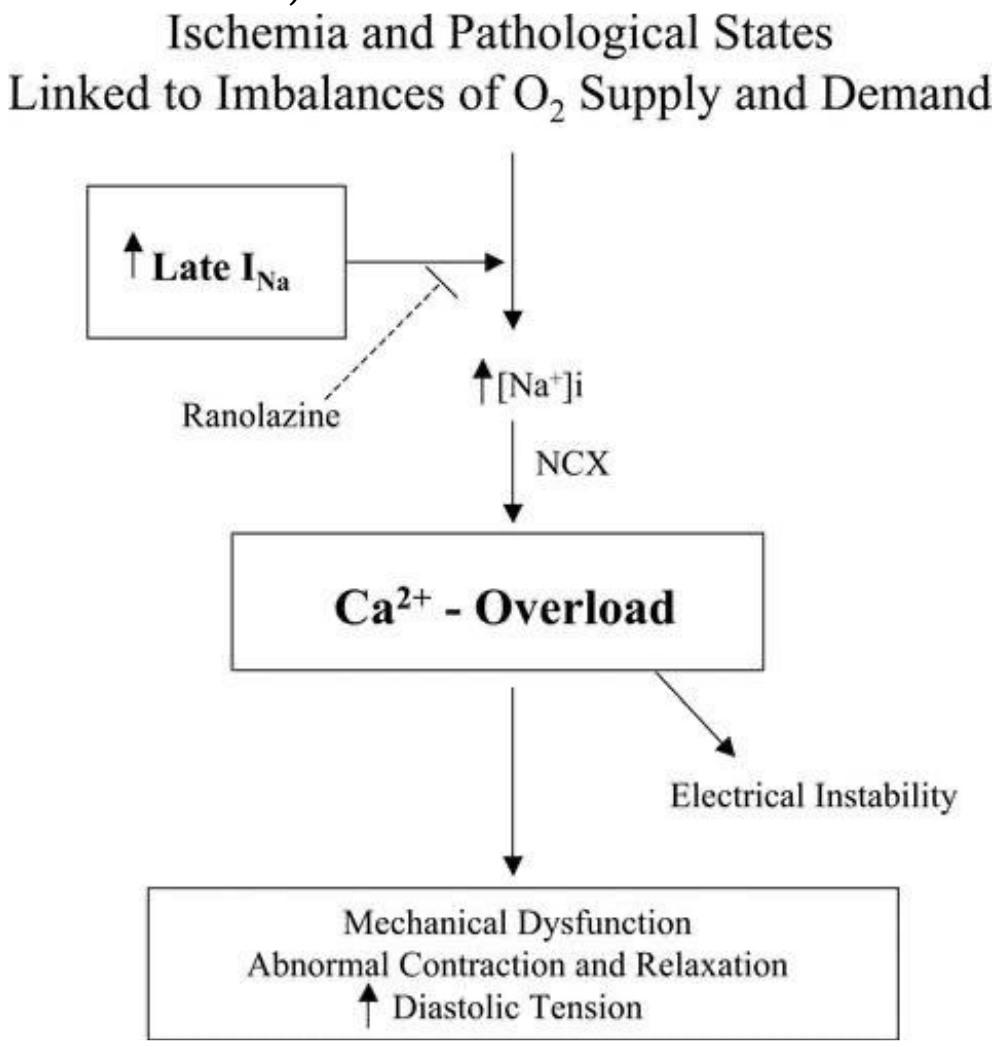
II. New therapies:

A. MEDICAL

1. Ranolazine

Action

- **Improve coronary blood flow :**
  - Inhibits Late I_{Na} (late inward Na channel) W is abnormally activated by ischemia → Prevents Na overload of the cell → Prevents reverse Na-Ca exchange → Prevents Diastolic accumulation of Ca → Improved Diastolic tone (tension)→ improved coronary blood flow
- **Others :** Anti-arrhythmic effect , Glycometabolic effect (↓ HbA1c , unclear mechanism)



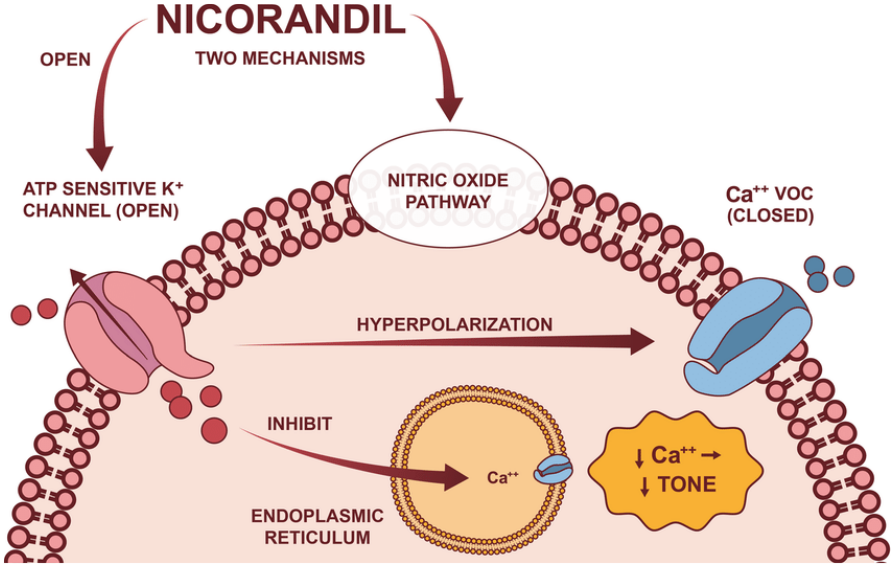
Dose

- 500 – 1000 mg twice / d

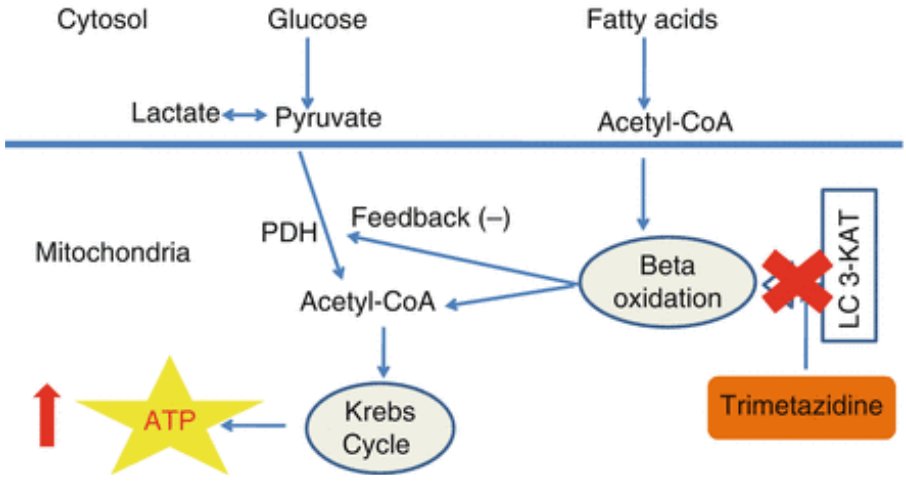
S/E

- ↑ QT interval
- Hepatotoxicity

2. Nicorandil

	K channel activator & Nitrate – Like action
Action	◆ Improve coronary blood flow ◆ Dilates arteries & veins ◆ Mimics natural ischemic Preconditioning → Protects the heart from subsequent ischemic attacks. 
Dose	10-20 mg twice /d
S/E	◆ Similar to nitrates

3. Trimetazidine

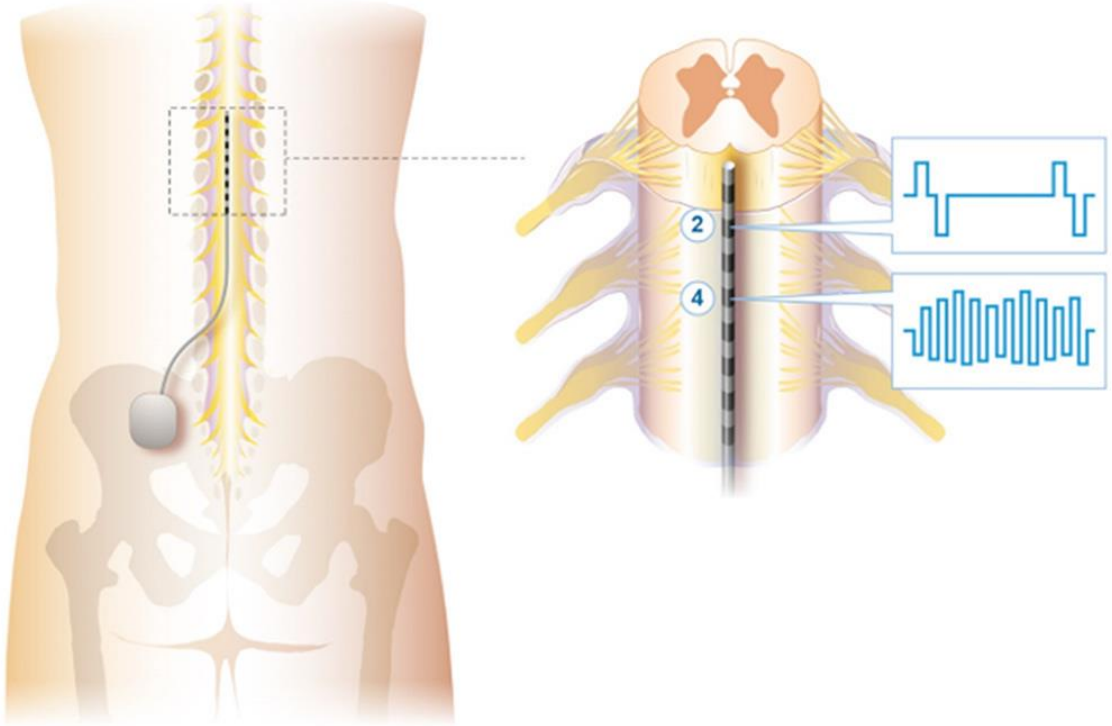
Action	◆ Inhibits β-Oxidation of FFA → ↓ O2 consumption. 

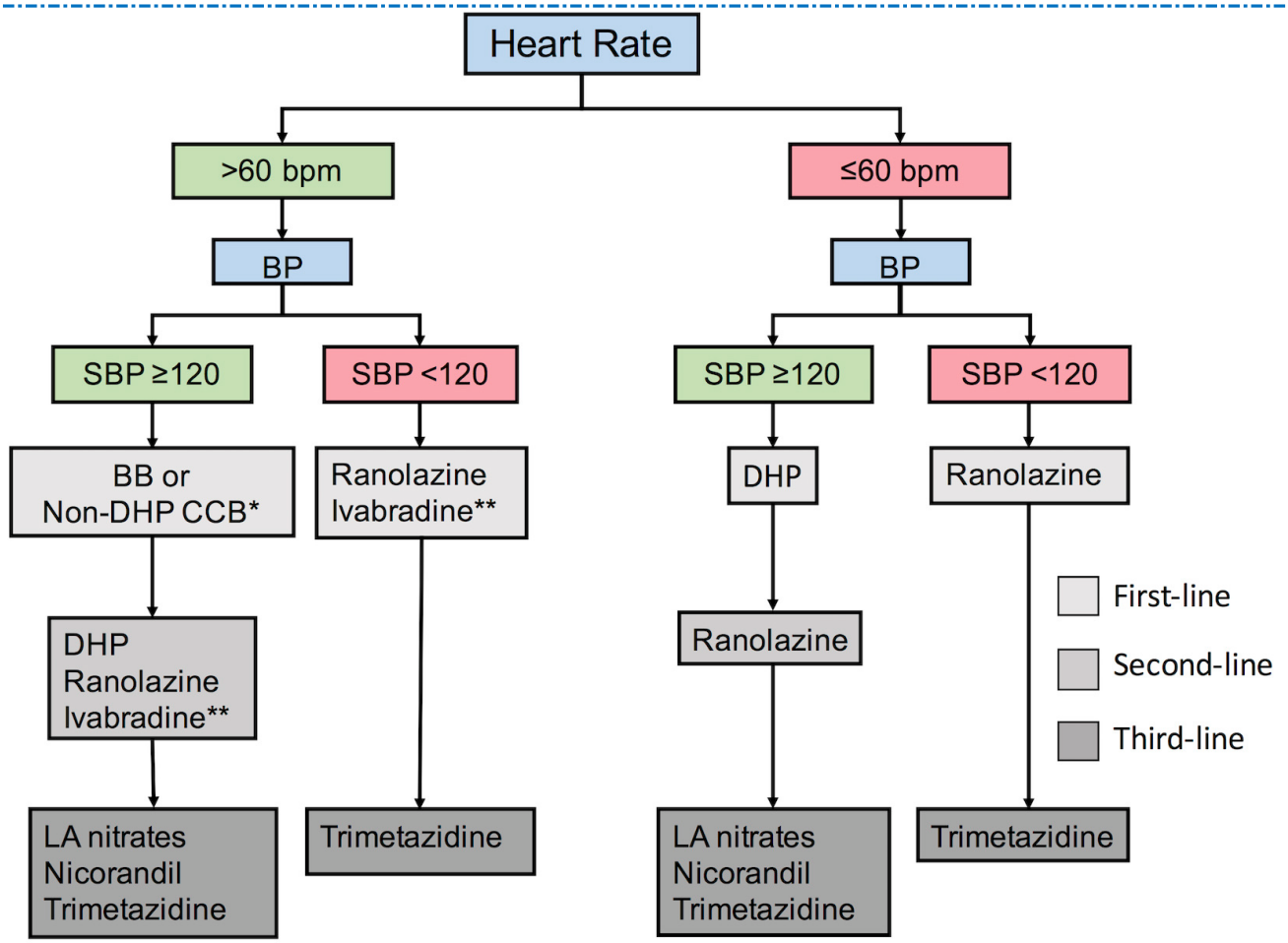
4. Ivabradine

Action	◆ Inhibits selectively cardiac PM current → HR

B. NON-MEDICAL: IN REFRACTORY ANGINA

1. Enhanced External Counterpulsation "EECP"	
Mechanism	◆ Improves Coronary Blood Flow : ↑ aortic blood flow during (diastolic augmentation) ≈IABP
Technique	**Non invasive Method** ◆ Cuffs are wrapped around the patient's calves , thighs & pelvis ◆ Using Compressed air , Sequential pressure (up to 300 mmHg) is applied in the early diastole to propel blood back to the heart and to the coronaries.

2. Spinal Cord Stimulation	
History	◆ Originally used to treat neurogenic pain
Recently	◆ Recently used to ttt of refractory angina
Mechanism	◆ ↓ activity of cardiac neurons during myocardial ischemia ◆ Redistributes myocardial blood flow from nonischemic to ischemic areas
Technique	**Minimally – invasive measure** ◆ Electrode Implanted in epidural space → electrical neuromodulation. 



* normal ejection fraction; ** heart rate > 70 bpm

BB = beta blocker; CCB = calcium channel blocker; DHP = dihydropyridine; SBP = systolic blood pressure

CHOICE OF DRUG THERAPY.

UNSTABLE ANGINA (AND NSTEMI):

1- Acute Management
◆ Patient with UA or NSTEMI should be treated with an early medical regimen similar to that used in an acute STEMI with one Exception : There is no evidence of benefit (and possible harm) from fibrinolysis ◆ Anticoagulant therapy in conjunction e Antiplatelet therapy is recommended for all patients with non-ST elevation ACS (UA &NSTEMI) as soon as possible after the diagnosis irrespective of whether an invasive or a conservative approach is taken

2- Non acute management (Following acute management , further medical ttt includes)	
Antianginal drugs	◆ β B & possibly Nitrates
Antiplatelets	◆ (Dual Antiplatelets Therapy = DAPT) ◆ Aspirin (indefinitely) and a platelet P2Y12 receptor blocker ◆ (e.g Clopidogrel for at least 1 month & up to 1 year)
Lipid lowering drugs	◆ Statins are recommended and the aim is to reduce LDL by ≥ 50% from baseline and to achieve LDL < 55 mg/dL • **If LDL goal is not achieved after 4-6 w despite max. statin dose ,** add Ezetimibe • **If LDL goal is not achieved after 4-6 w Despite max. statin & Ezetimibe ,** add PCSK9-I
ACE-Is or ARBs	◆ Recommended in patients e HFe reduced LVEF (40%) or Diabetes
MRAs	◆ Recommended in patients e HF e reduced LVEF (40%)
PPIs	◆ To reduce the risk of gastric bleeding from antiplatelets

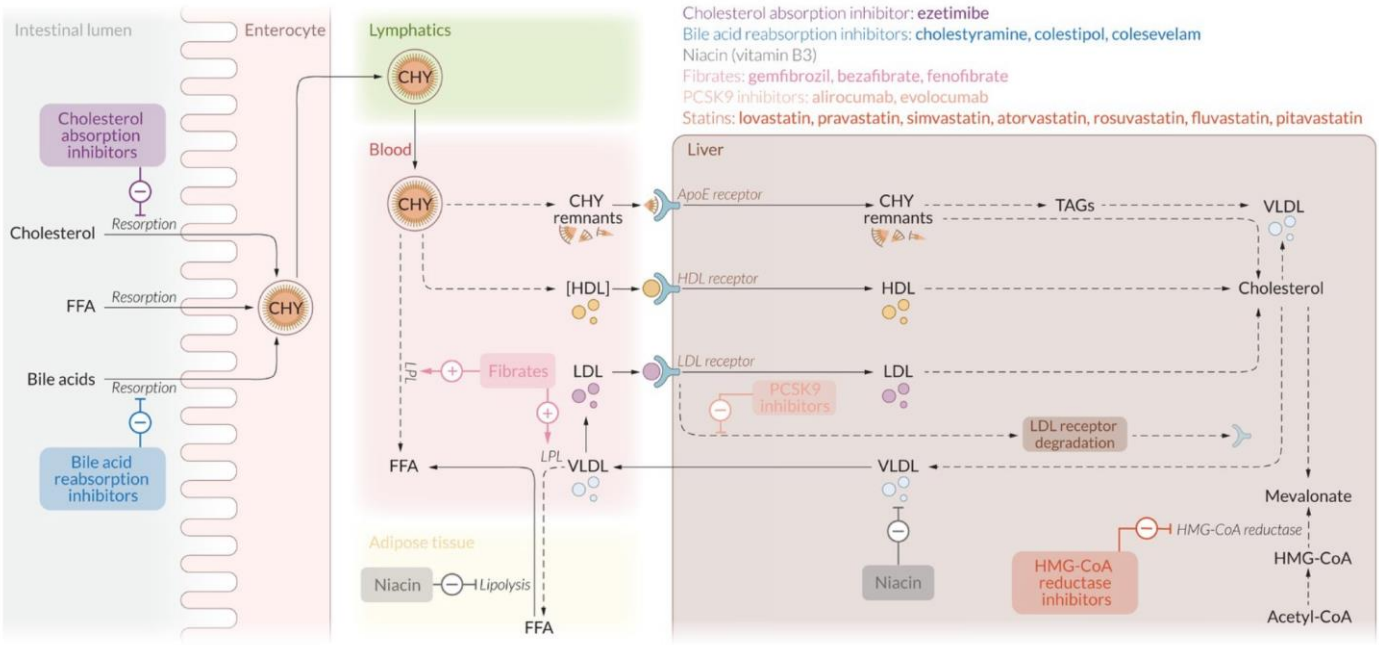
VASOSPASTIC ANGINA

- Nitrates.
- CCBs.

II. Treatment of Dyslipidemia:

Agents	Indications	Mechanism of action	Effects on lipid profile			Adverse effects		C/I	Interactions
			LDL	HDL	TGs				
Statins (e.g., atorvastatin, simvastatin)	✦ First-line lipid lowering agent	✦ Competitive inhibition of HMG-CoA reductase ✦ **Pleiotropic effect:** ↑ Plaque stabilization Antioxidant effect, improved endothelial function of coronary arteries	↓↓↓	↑	↓	Myalgia +	✦ Statin-associated myopathy ✦ ↑ LFTs	✦ Muscle disorder ✦ Pregnancy ✦ Breastfeeding	✦ Other lipid-lowering agents ✦ CYP3A4 inhibitors
PCSK9 inhibitors (e.g., alirocumab, evolocumab)	✦ Add-on therapy for patients who have both of the following LDL ≥ 1.8 mmol/l (70 mg/dL) despite maximally tolerated treatment with statins and ezetimibe Presence of very high-risk atherosclerotic cardiovascular disease	✦ Inhibition of PCSK9					✦ Delirium	✦ History of hypersensitivity	✦ **Statins:** ↓ half-life of alirocumab
Bile acid sequestrants (e.g., cholestyramine, colestipol)	✦ Hypercholesterolemia: combination therapy with statins ✦ Digitoxin overdose	✦ Ion exchange resin binds bile acids in the intestine → interruption of enterohepatic circulation (↓ bile acid absorption and ↑ bile acid excretion)	↓↓	(↑)	(↑)	✦ Nausea, abdominal bloating, and cramping		✦ Hyper-TGs > 300–500 mg/dL ✦ Bowel obstruction	✦ ↓ absorption of warfarin, digoxin, and fat-soluble vitamins
Ezetimibe	✦ C/I or statin intolerance: monotherapy ✦ Insufficient LDL cholesterol reduction resulting from statins: combination therapy (statin and ezetimibe)	✦ Selective inhibition of cholesterol reabsorption at the brush border of enterocytes		↑/↔	↓/↔	✦ ↑ Liver enzymes ✦ Diarrhea		✦ Coadministration with a statin during active liver disease	✦ **Cholestyramine:** ↓ oral bioavailability

Agents	Indications	Mechanism of action	Effects on lipid profile			Adverse effects	C/I	Interactions
			LDL	HDL	TGs			
Niacin	✦ High LDL cholesterol and lipoprotein levels despite statin and ezetimibe therapy	✦ Inhibits lipolysis and fatty acid release in adipose tissue by blockading hormone-sensitive lipase	↓↓	↑↑	↓↓	✦ Flushing and pruritus ✦ Hyperglycemia ✦ Hyperuricemia and gout	✦ Liver failure ✦ Gout	✦ **Alcohol:** ↑ risk of liver damage ✦ Worsening of flushing and pruritus
Fibrates (e.g., bezafibrate, fenofibrate, gemfibrozil)	✦ Second line drug of choice in hyperlipidemia: most effective for lowering triglycerides	✦ Activation of the peroxisome proliferator-activated receptor alpha (PPAR–α)	↓	↑	↓↓↓	✦ Dyspepsia ✦ Myopathy ✦ Cholelithiasis, ↑ LFTs	✦ Renal insufficiency ✦ Liver failure	✦ Enhance effect of other drugs (sulfonylureas, warfarin) by inhibiting hepatic CYP450
Marine omega-3 fatty acids (fish oil)	✦ Add-on therapy	✦ Decreased transportation of free fatty acids to the liver ✦ Inhibition of triglyceride-synthesizing enzymes	(↑)	(↑)	↓ (high doses necessary	✦ Fishy taste ✦ Nausea	✦ Hypersensitivity to ingredients of drug formula	✦ No known severe drug interactions
Lomitapide	✦ A beta lipo-proteinemia	✦ Inhibits microsomal triglyceride transfer protein (MTTP)	↓ ↓ VLDL	↔	↔	✦ Gastrointestinal symptoms ✦ ↑ LFTs	✦ Pregnancy ✦ Hepatic impairment ✦ Concomitant therapy with CYP3A4 inhibiting drugs	✦ **Warfarin:** ↑ plasma concentration → ↑ INR



STATIN THERAPY

INCLUDE	◆ Atorvastatin ◆ Rosuvastatin ◆ Simvastatin.
INDICATIONS	◆ The first-line treatment of choice for patients with high cholesterol and those diagnosed with IHD. ◆ Recommended in all patients with CCS.
BENEFITS	◆ **lowering** cholesterol levels→ (pleiotropic effects) ◆ **Improve** endothelial function, ◆ **Improve** stability of atherosclerotic plaques, ◆ **Reduce** the inflammation and oxidative stress, ◆ **Reduce** platelet aggregation thereby ◆ **Reduce** the risk of a blood clot (thrombus)..

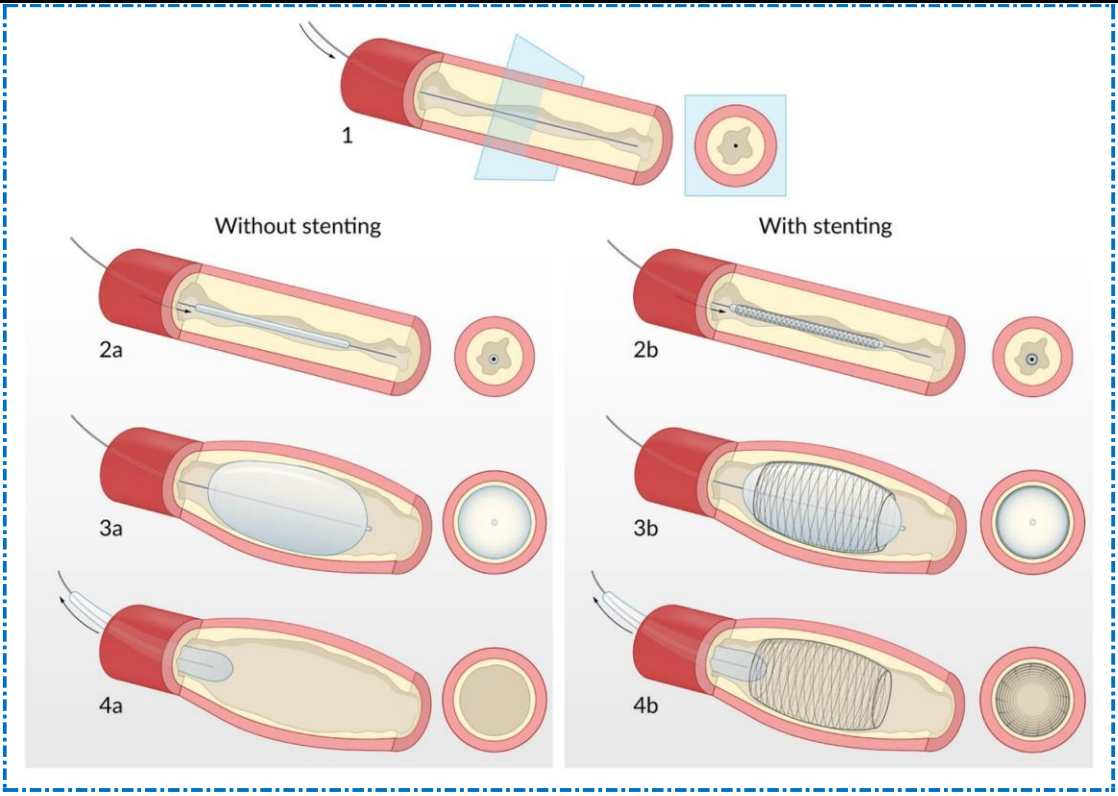
III. Invasive Intervention "Coronary Revascularization":

INDICATIONS:

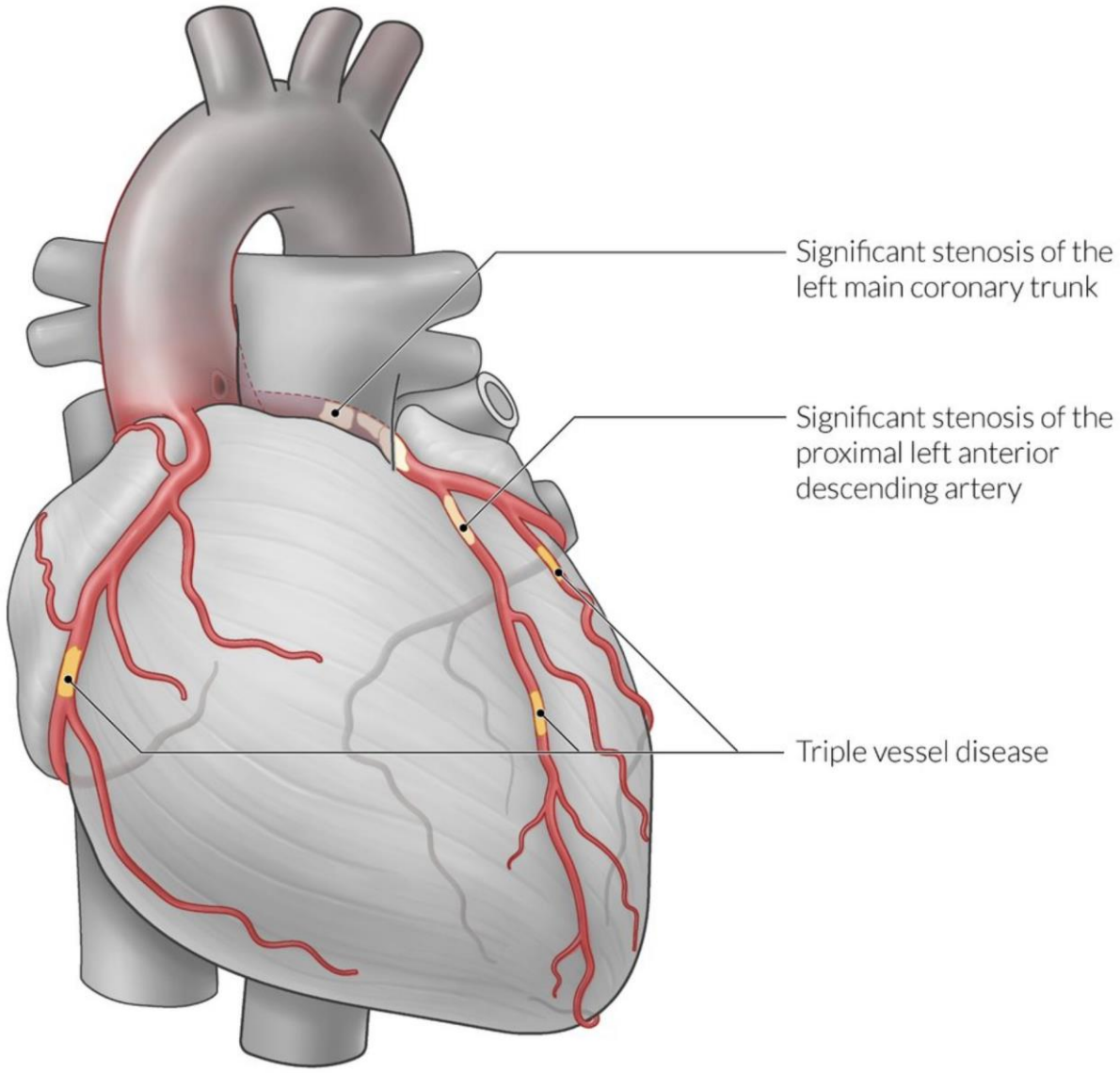
- **General:**
  - Angina which is not responding to medical treatment.
  - Post - infarction angina: to improve the prognosis.
- **Specific to the technique.**

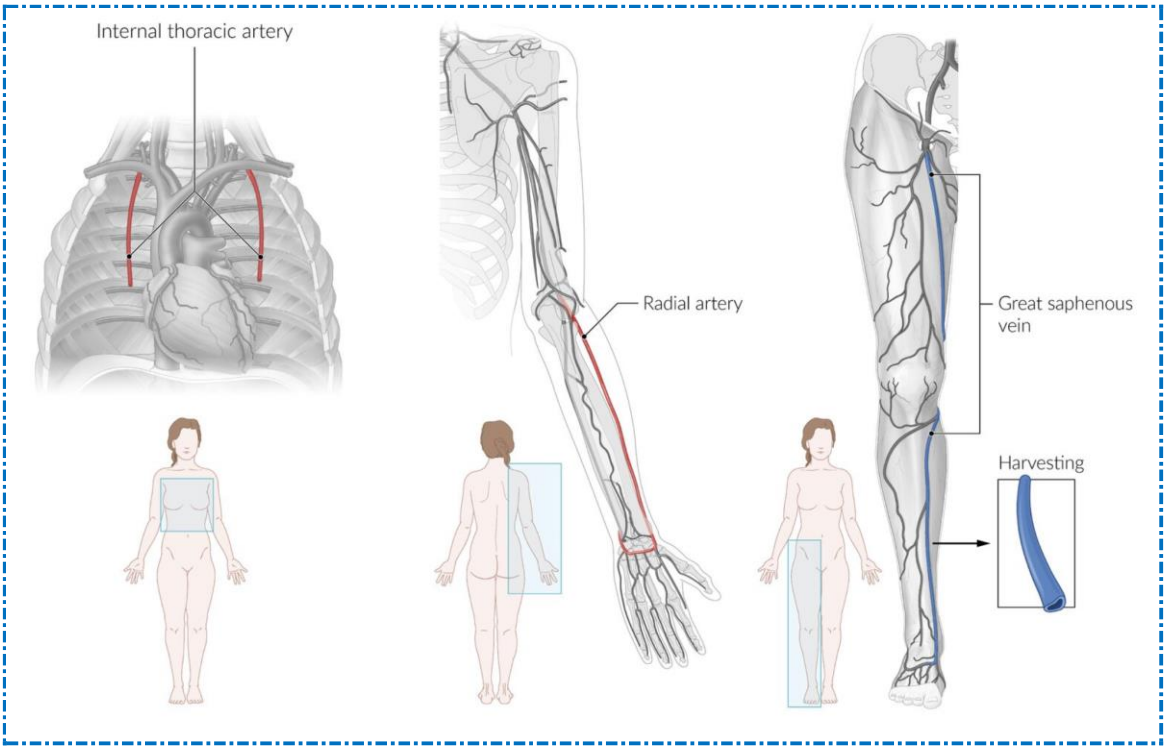
TECHNIQUES:

PCI: (Percutaneous Coronary Intervention) PTCA+ Stent (Percutaneous Transluminal Coronary Angioplasty)	
Principle	↳ Introduction of a balloon to dilate the stenotic artery ↳ Introduction of an intra - luminal Stent to maintain patency of the dilated artery.
Indications	↳ Stenosis of one or 2 vessels only (except left main coronary artery). ↳ Stenosis of bypass grafts following CABG.
Complications	↳ Complications of coronary arteriography. ↳ Acute coronary occlusion. ↳ Restenosis.

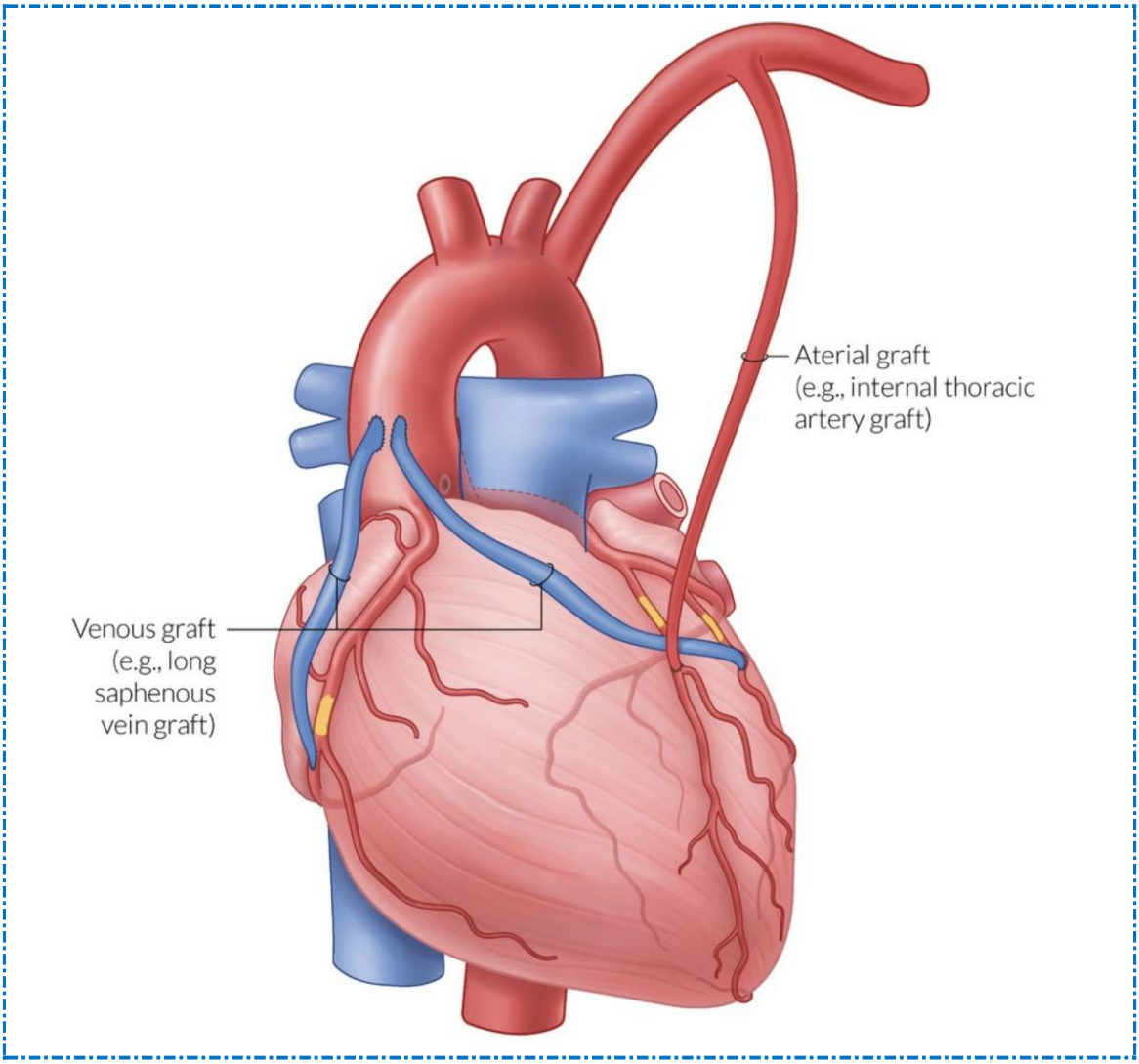


CABG: (Coronary Artery Bypass Graft)	
Principle	↳ Grafting a segment of saphenous vein or an IMA between the ascending aorta & the coronary artery distal to the obstruction.
Indications	↳ Stenosis of 3 or more vessels. ↳ Stenosis of the left main coronary artery
Results	↳ **In 90 % of patients:** Excellent relief of angina. ↳ **In 10 % of patients:** Restenosis of the vein graft in the first year.

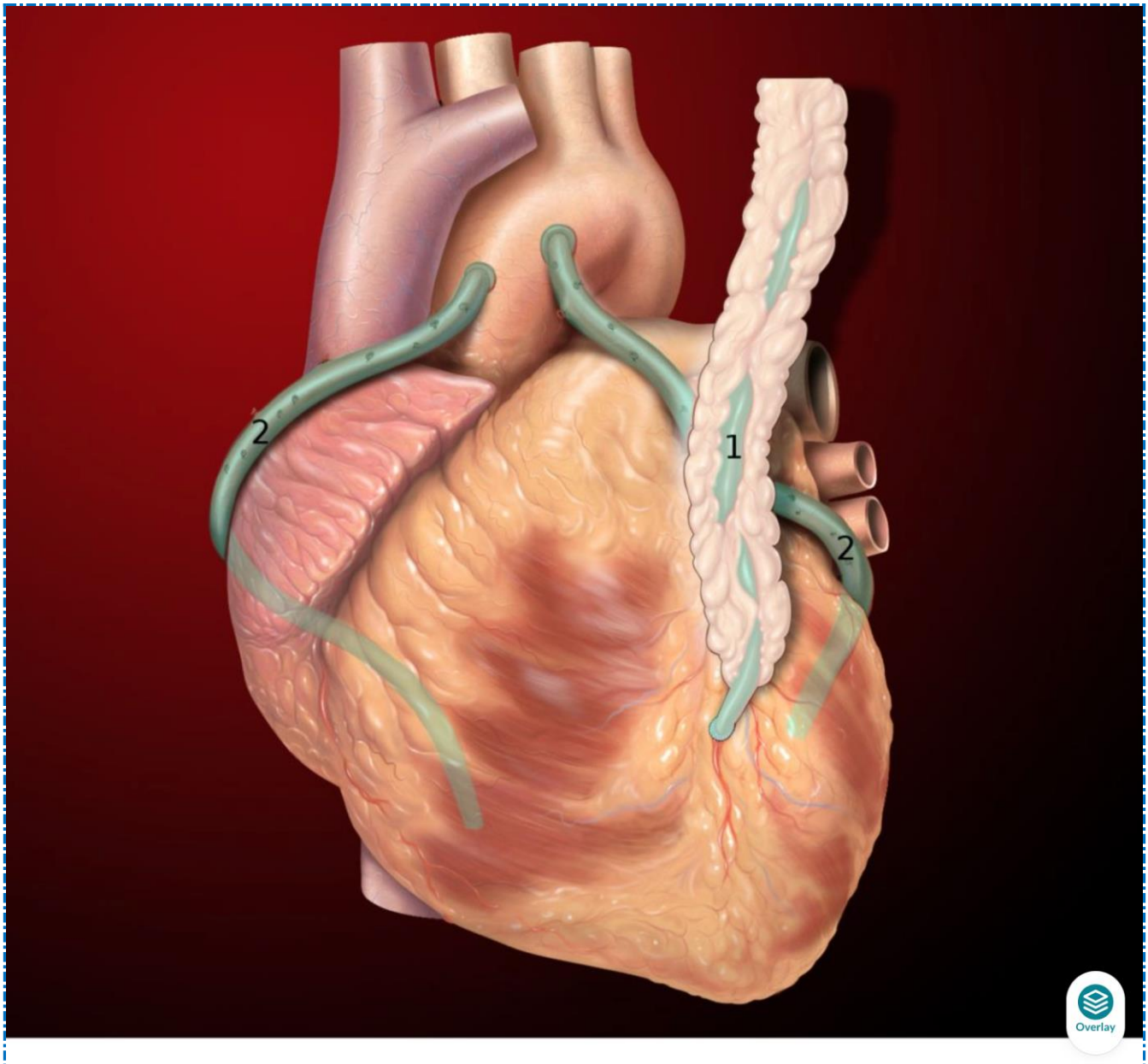




VESSELS COMMONLY USED iN GABG



ARTERiAL AND VENOUS GRAFTS iN GABG



Coronary artery bypass grafts

(1) arterial graft: the left internal mammary artery (LIMA) is grafted onto the left anterior descending coronary artery (LAD) - note the fatty tissue surrounding the artery (white).
(2) venous grafts: veins (usually the saphenous veins are used) are grafted onto the posterior descending artery and left marginal artery - note that the venous grafts are connected directly to the aorta.

ACUTE MYOCARDIAL INFARCTION

DEFINITION :

- **Ischemic necrosis** of part of the myocardium due to **Sudden & persistent cessation of its blood supply "**

TYPES & ETIOLOGY :

Type 1 MI	◆ **Most common form** ◆ MI caused by atherosclerotic plaque disruption or acute coronary thrombosis ◆ Caused by acute thrombosis due to erosion, ulceration, fissuring, dissection, or rupture of an atherosclerotic plaque ◆ ↓ Myocardial blood flow → sudden death of myocardial cells ◆ Usually manifests as STEMI
Type 2 MI	◆ **Less common form (14%)** ◆ MI **secondary** to an oxygen supply/demand mismatch. ◆ Occurs predominantly in women and in individuals with comorbidities (eg, diabetes previous NSTEMI) ◆ **Not due to plaque rupture:** caused by a condition other than coronary artery disease ◆ Ischemia is caused by increased oxygen demand (e.g, anemia or decreased coronary blood supply (e.g, coronary artery spasm)
Type 3 MI	◆ MI resulting in **death** when **biomarker values** are **unavailable**
Type 4 MI	◆ MI related to **percutaneous coronary intervention** : • **Type 4a MI:** ≤48 hours after PCI. • **Type 4b MI:** related to stent thrombosis • **Type 4c MI:** associated with restenosis after PCI
Type 5 MI	◆ MI related to **coronary artery bypass grafting.**
MINOCA	◆ = Myocardial Infarction with No Obstructive Coronary Atherosclerosis. ◆ 5-15% Of cases of MI.

MINOCA

Epicardial Causes

- Coronary Plaque Disruption or Erosion
- Spontaneous Coronary thrombosis/ embolism
- Missed Obstructive Coronary Artery Diease (CAD)

Microvascular Causes

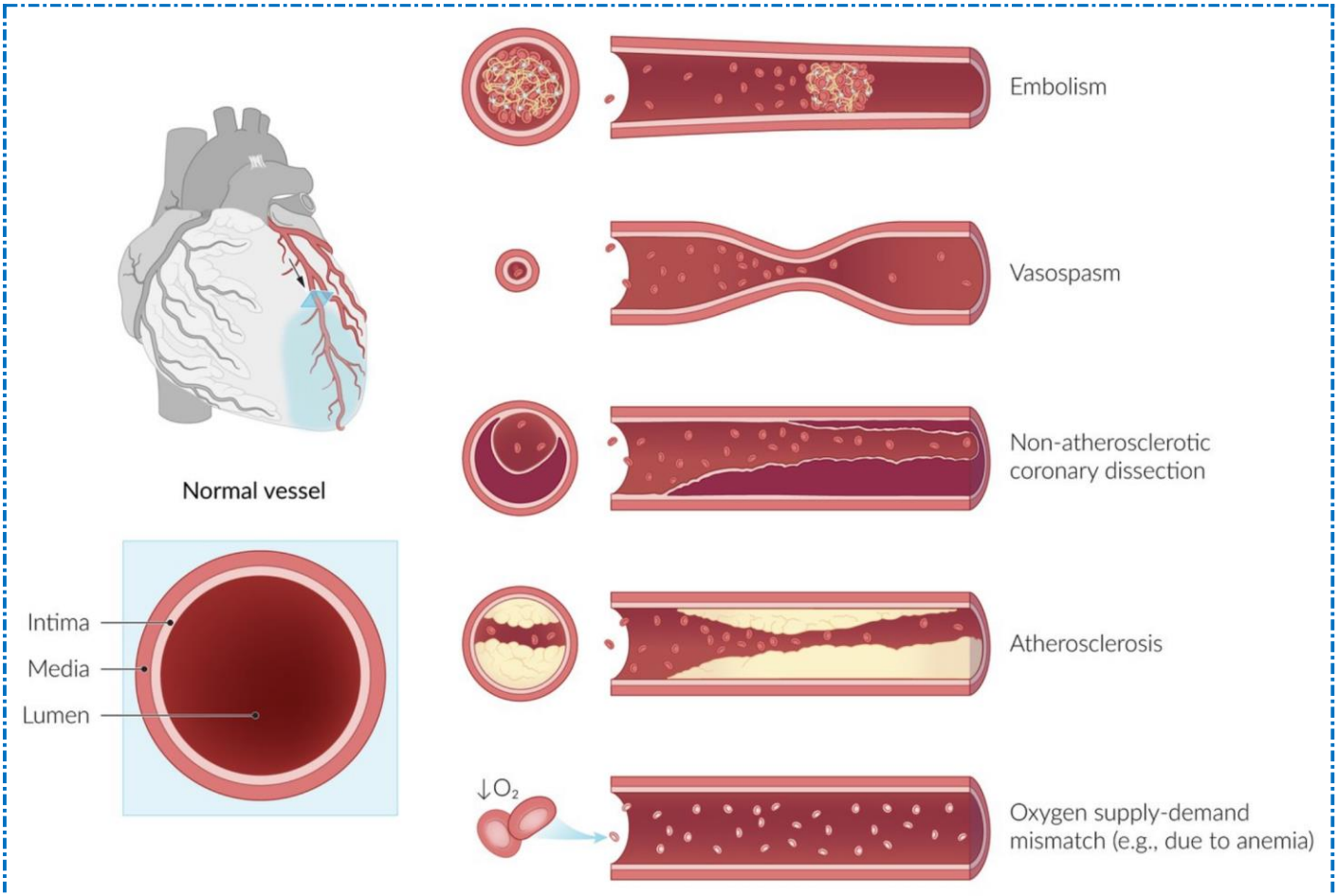
- Myocardial Disorders
  - Myocarditis
  - Takotsubo cardiomyopathy
  - Other cardiomyopathies (DCM*, HCM**)

* DCM-dilated cardiomyopathy ** HCM-hypertrophic cardiomyopathy

INOCA

- Vasospastic Angina (epicardial artery spasm)
- Coronary Microvascular Disorders
  - Coronary slow flow
  - Microvascular angina-*including Mental Stress Induced Myocardial Ischemia (MSIMI)*
  - Microvascular spasm

MiNOCA VS iNOCA

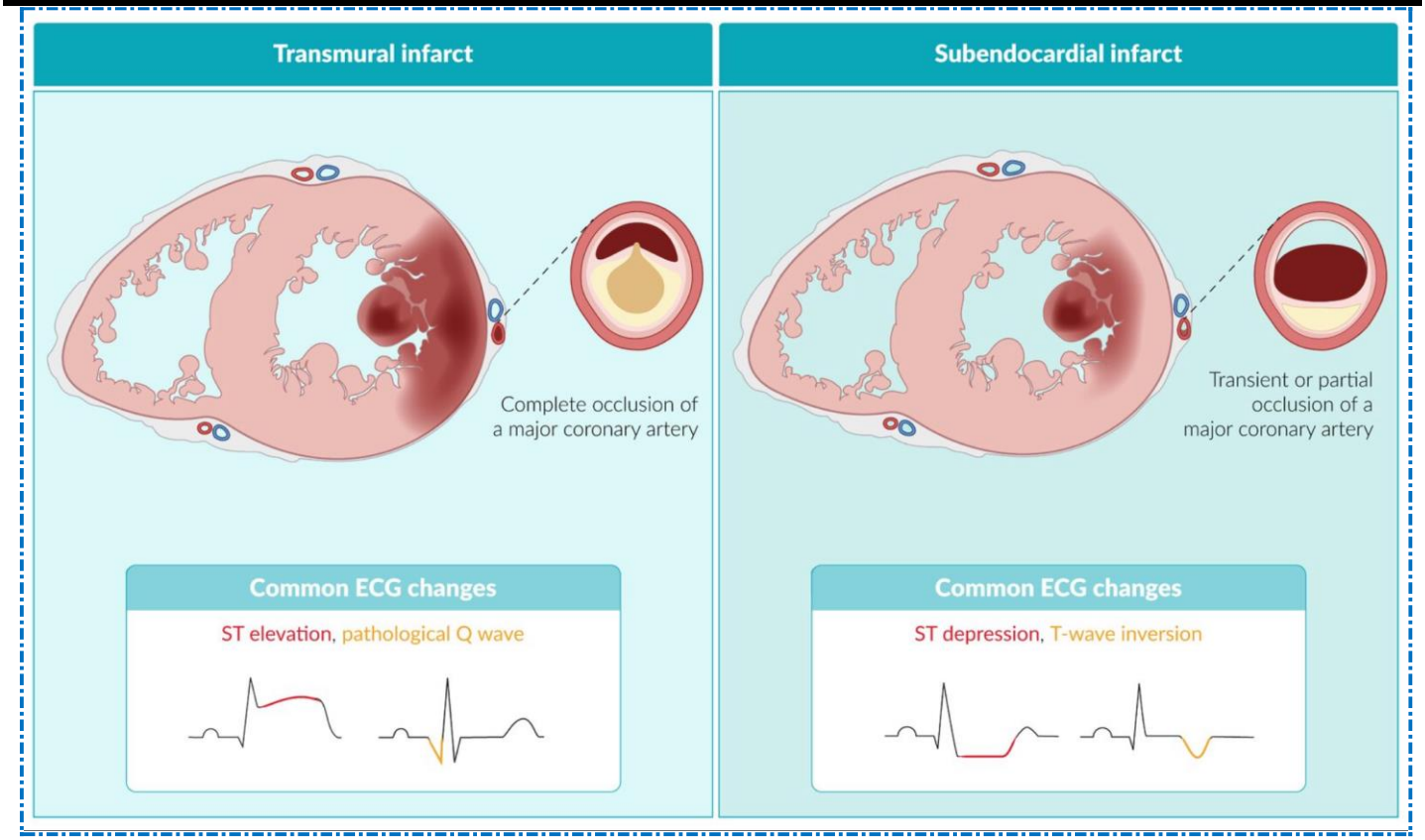


ETiOLOGY OF ACUTE CORONARY SYNDROME

PATHOPHYSIOLOGY:

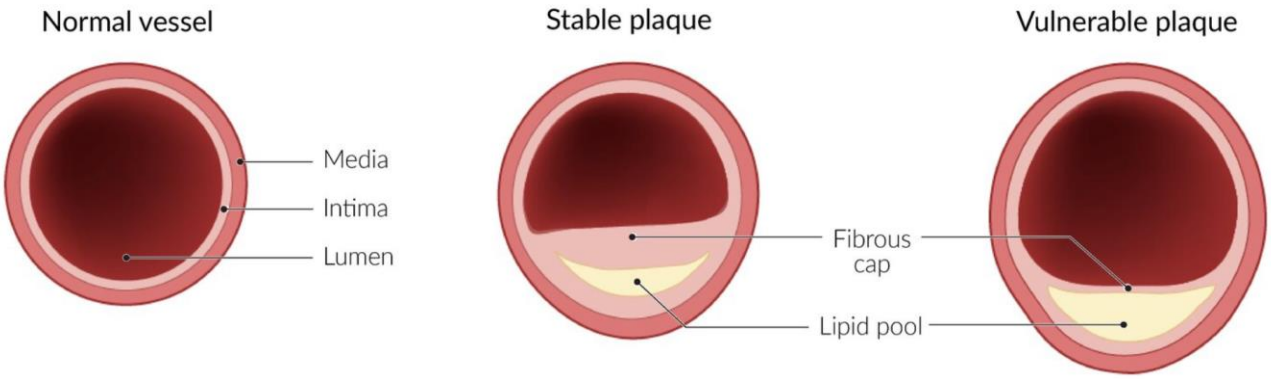
A. CORONARY ARTERY OCCLUSION

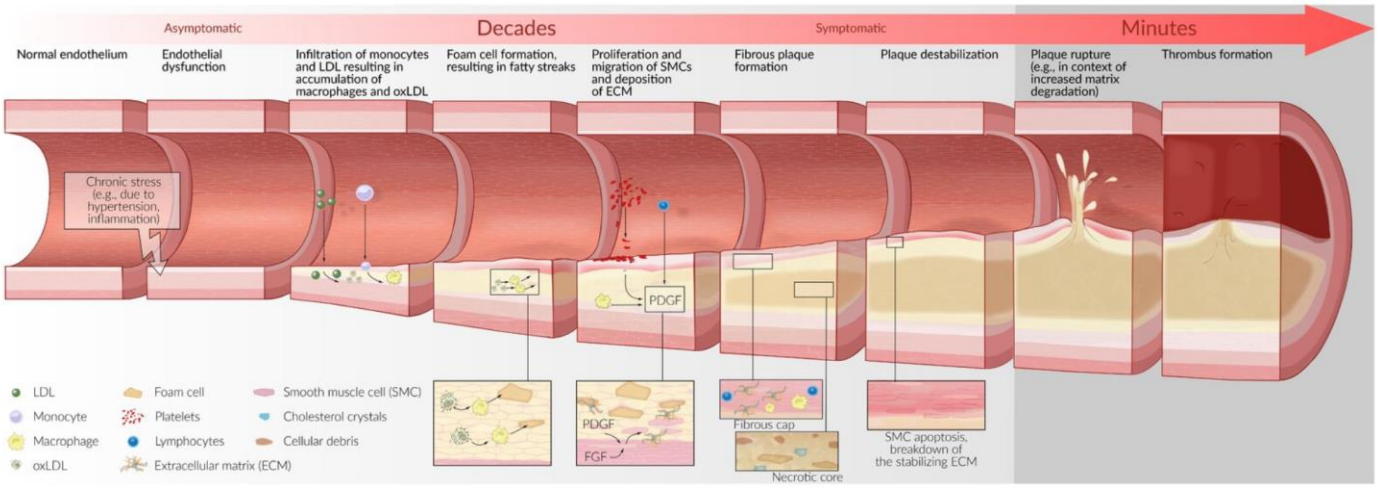
Partial coronary artery occlusion	Complete coronary artery occlusion
◆ **Decreased** myocardial blood flow → supply-demand mismatch→ myocardial ischemia ◆ Usually affects **inner layer of myocardium** (subendocardial infarction) ◆ Typically manifests clinically as **unstable angina and/or NSTEMI**	◆ **Impaired** myocardial blood flow → sudden death of myocardial cells (if no reperfusion occurs) ◆ Usually affects **full thickness of myocardium** (transmural infarction) ◆ Typically manifests clinically as **STEMI**



B. ATHEROSCLEROTIC PLAQUE DISRUPTION (TYPE 1 MI) :

- For **plaque** formation and **Atherosclerosis.**
- **Stable atherosclerotic plaque:** manifests as stable angina (symptomatic during exertion)
- **Unstable plaques** are lipid-rich and covered by thin fibrous caps high risk of rupture and acute coronary syndrome
- **Inflammatory cells in plaque** (e.g. macrophages) secrete matrix **metalloproteinases** → breakdown of extracellular matrix → weakening of the fibrous cap → minor stress → rupture of the fibrous cap → exposure of highly thrombogenic lipid core → thrombus formation→ coronary artery occlusion





C. OXYGEN SUPPLY AND DEMAND MISMATCH (TYPE 2 MI)

- Can occur in patients with or without underlying coronary artery disease
- **Decreased oxygen supply**
  - Occlusion of coronary arteries (eg, coronary dissection, vasospasm)
  - Reduced perfusion (e.g. hypotension, bradycardia, anemia)
- Increased oxygen demand (e.g., sustained tachyarrhythmia)

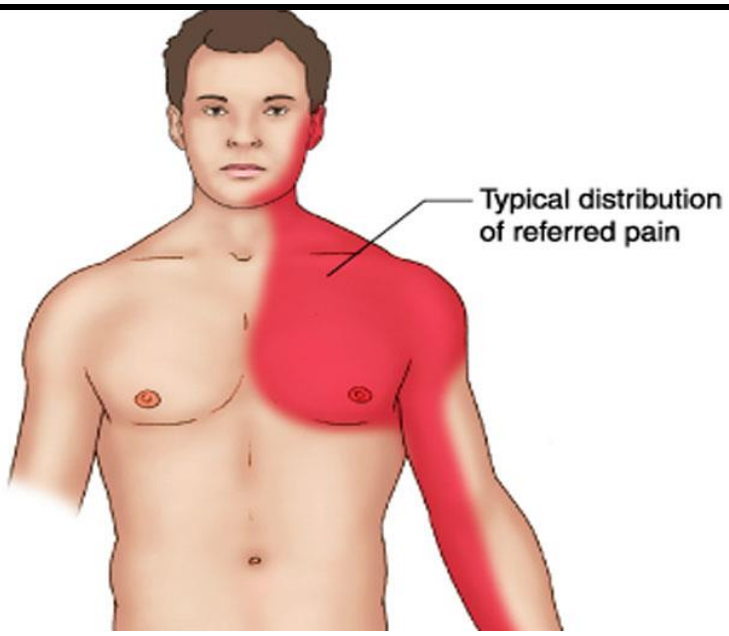
D. NON-ISCHEMIC MYOCARDIAL INJURY :

- Necrosis of myocardial tissue without ischemia (e.g.. in sepsis)
- The pathophysiology of myocardial damage is not completely understood, but potential explanations **include:**
  - Inflammatory cytokines
  - Toxicity of high catecholamine levels

CLINICAL PICTURE :

A) SYMPTOMS:

Onset	**Usually in the early morning due to:** ◆ Rise in plasma catecholamines. ◆ Rise in plasma Cortisol. ◆ Increased platelet aggregation.
Pain (main symptom)	**A. TYPICAL:** as anginal pain, BUT: ◆ More severe, more prolonged, more radiating. ◆ May occur without precipitating factors. ◆ Not relieved by rest or sublingual nitrates. **B. ATYPICAL:** ◆ Sense of indigestion. ◆ Atypical location of pain. **C. ABSENT:** (silent myocardial infarction) ◆ Autonomic neuropathy e.g. **Diabetes**. ◆ **Elderly** patients. ◆ During **anesthesia** or in a transplanted heart. ◆ Disturbed **consciousness** level, heavy sedation, shock. **D. MORE COMMON IN INFERIOR WALL INFARCTION :** ◆ Epigastric pain. ◆ Bradycardia. ◆ Clinical triad in right ventricular infarction (Hypotension, elevated jugular venous pressure, clear lung field).



B) SIGNS:

General

A. GENERAL APPEARANCE: Pale, sweaty, restless.

B. PULSE:

Rate	• **Tachycardia** (sympathetic hyperactivity). • **Bradycardia** (HB or↑vagal tone due to pain).
Rhythm	• Arrhythmias.
Pulse volume	• May be small due to LVF or shock.

C. BLOOD PRESSURE:

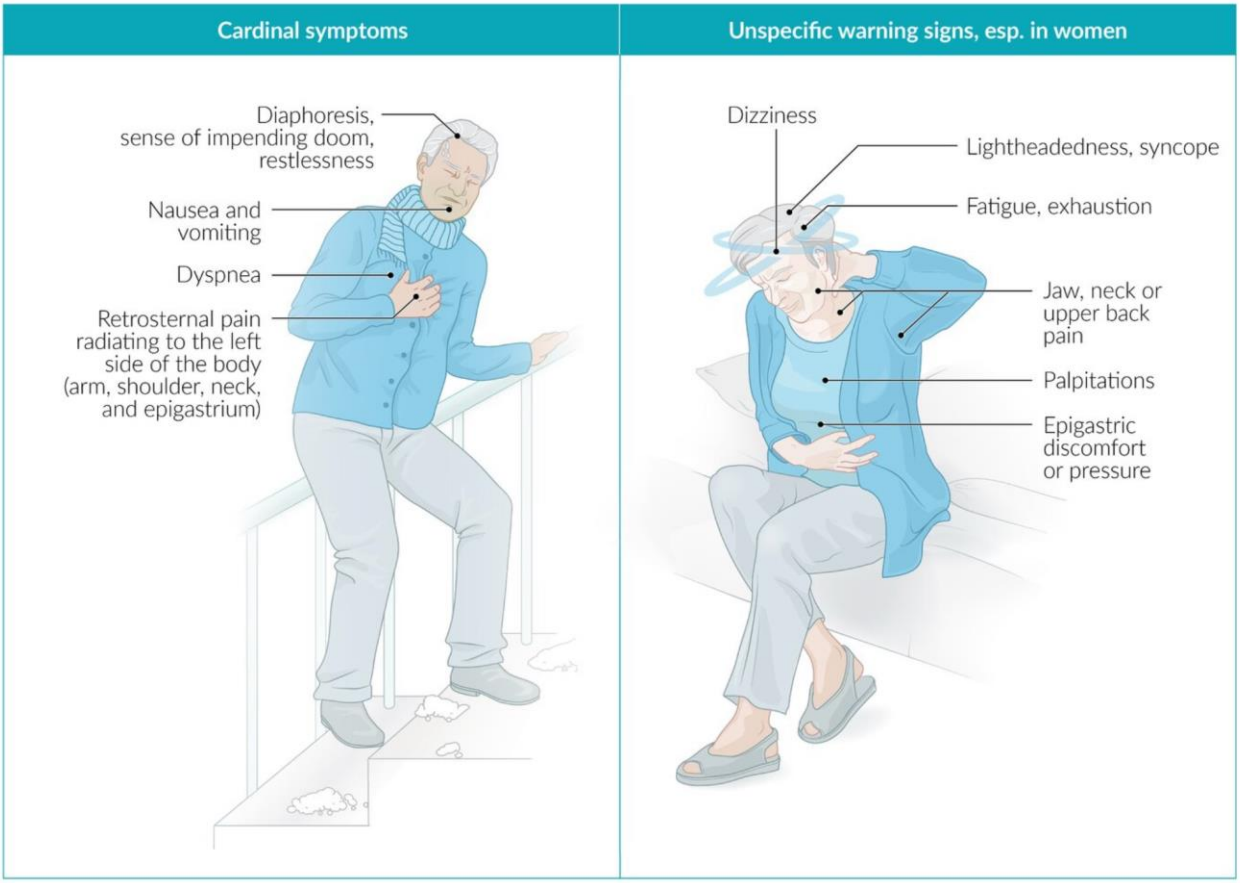
- Initial hypertension (sympathetic hyperactivity).
- Hypotension (LVF or shock or↑ vagal tone due to pain).
- **Decapitated BP:** marked drop of SBP with slight drop of DBP.

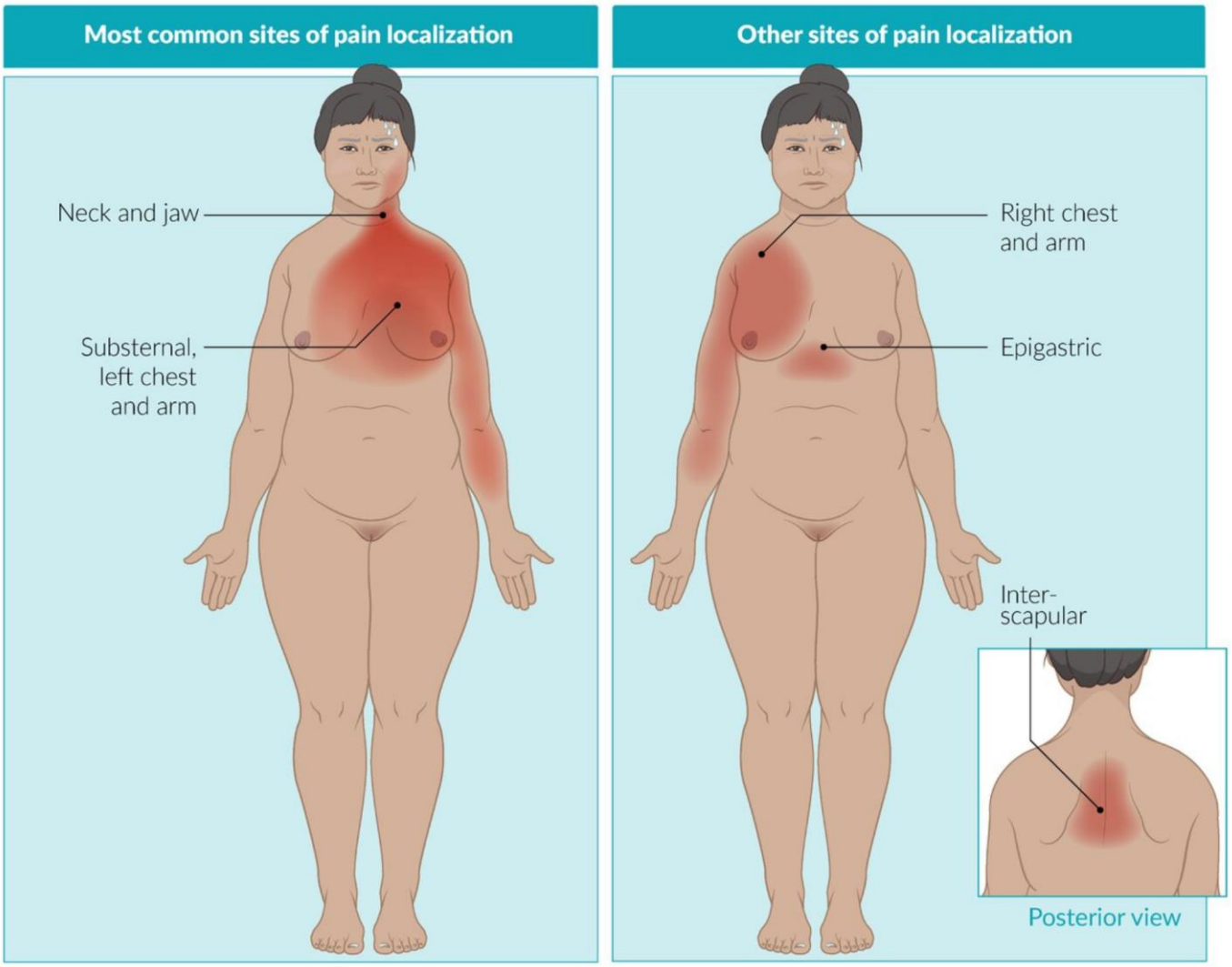
D. FEVER: (NON-SPECIFIC RESPONSE TO TISSUE NECROSIS)

- Moderate rise within 1-2 days of the onset and may last for 1 week.

Cardiac

- **S1**: Weak.
- **S2**: Reversed splitting.
- **S3**: due to flabby myocardium.
- **S4**: due to decreased ventricular compliance by infarction.
- **Signs of complications:** e.g. **systolic murmur of MR** .





COMPLICATIONS :

A. Early Catastrophic Complications: **(first few days)**

Sudden death	**Due to :** ◆ VF. ◆ Rupture of the weak myocardium. ◆ Occlusion of the left main coronary artery.
Cardiac arrhythmias	**All types may occur :** ◆ **The most common are:** **Extrasystoles.** ◆ **The most serious are:** **VT & HB.**
Cardiac failure	◆ Acute LVF.
Pericarditis	◆ Usually dry, but effusion may develop.

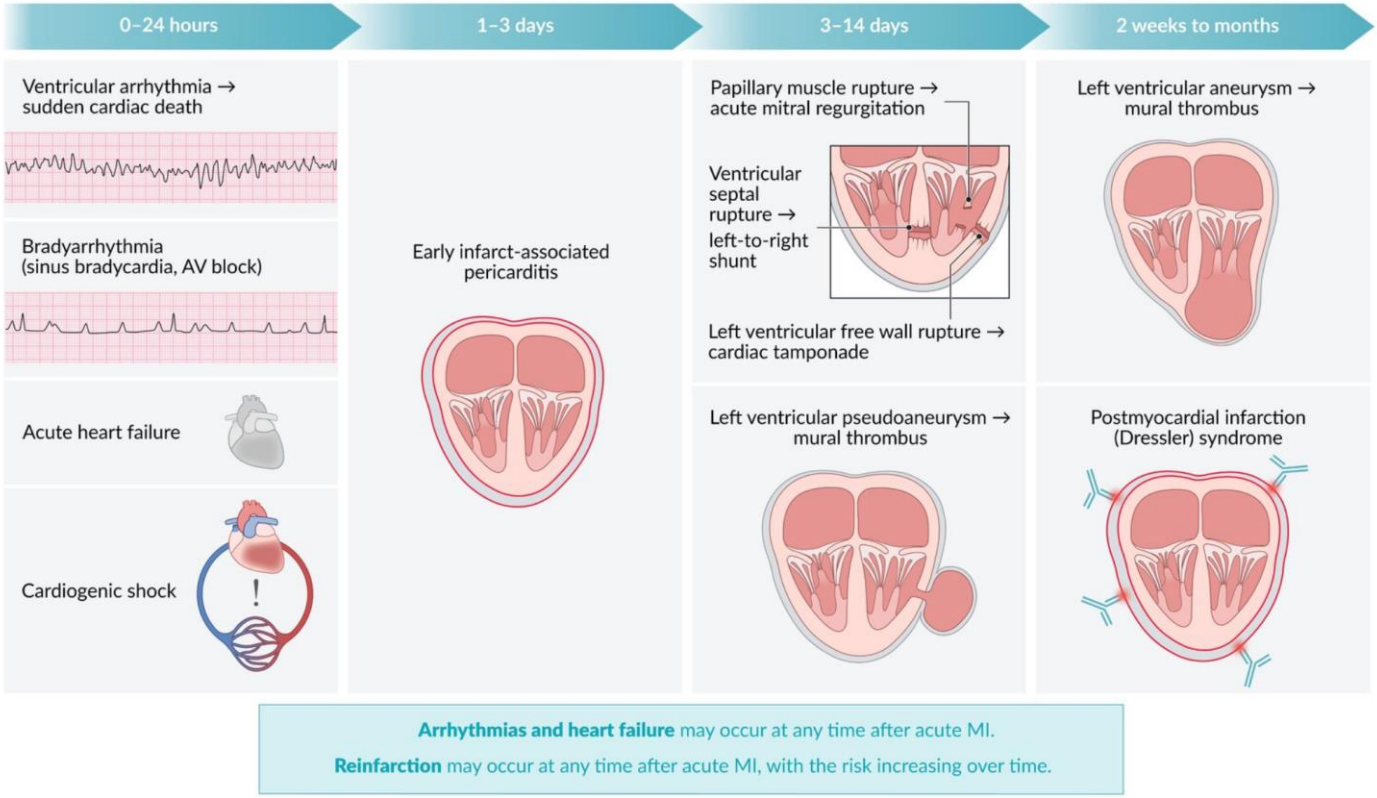
<table>
<tr><td rowspan="4">Shock</td><td></td><td>Cardiogenic</td><td>Neurogenic</td></tr>
<tr><td>Cause</td><td>Massive infarction → pump failure</td><td>severe pain → vagal stimulation</td></tr>
<tr><td>Feature</td><td>Hypotension & Tachycardia</td><td>Hypotension & Bradycardia</td></tr>
<tr><td>Prognosis</td><td>very bad</td><td>good</td></tr>
<tr><td>Myocardial rupture</td><td colspan="3">

Rupture of ventricular free wall → cardiac tamponade &Death.
Rupture of the septum → acquired VSD.
Rupture of the papillary muscles:

<table>
<tr><td>Time</td><td>Usually occurs 2-7 days after MI.
Can lead to acute mitral regurgitation.</td></tr>
<tr><td>Site</td><td>More often: posteromedial papillary muscle rupture due to occlusion of the posterior descending artery (single supply).
Less often: anterolateral papillary muscle rupture due to occlusion of LAD and/or LCx (double supply).</td></tr>
<tr><td>Clinical feature</td><td>New holosystolic, blowing murmer over the 5th ICS on the midclavicular line.
Signs of acute mitral regurgitation: dyspnea, cough, bilateral crackles, hypotension.</td></tr>
<tr><td>Complications</td><td>Mitral regurgitation can lead to severe pulmonary edema and/or cardiogenic shock.</td></tr>
</table>
</td></tr>
</table>

B. Late Complications (after several weeks)

Post infarction angina & recurrent infarction	◆ Due to affection of other coronaries.
Post infarction syndrome (Dressler's syndrome)	◆ **Onset:** developing weeks after infarction. ◆ **Caused by:** autoimmune response to damaged cardiac tissue. ◆ **Responds to:** corticosteroids.
Frozen shoulder (shoulder hand) syndrome:	**"Pain & stiffness of left arm & shoulder"** ◆ **Onset:** developing weeks after infarction. ◆ **Caused by:** reflex arteriolar spasm → ischemia & fibrosis. ◆ **Responds to:** corticosteroids & physiotherapy.
Thrombo - embolic complications	◆ Mural thrombosis (in LV) → systemic embolism. ◆ DVT (from prolonged recumbency) → pulmonary embolism.
Myocardial aneurysm	◆ **Clinical presentations:** • **R**ecurrent embolisation. • **R**ecurrent arrhythmias. • **R**efractory HF. • **R**upture of the aneurysm. ◆ **On examination:** • Double apex. ◆ **Investigations:** • **ECG:** persistent ST segment elevation > 6 weeks. • **Echo:** diagnostic.
Complications of TTT.	



TIMELINE OF MOST COMMON POST MYOCARDIAL INFARCTION COMPLICATIONS.

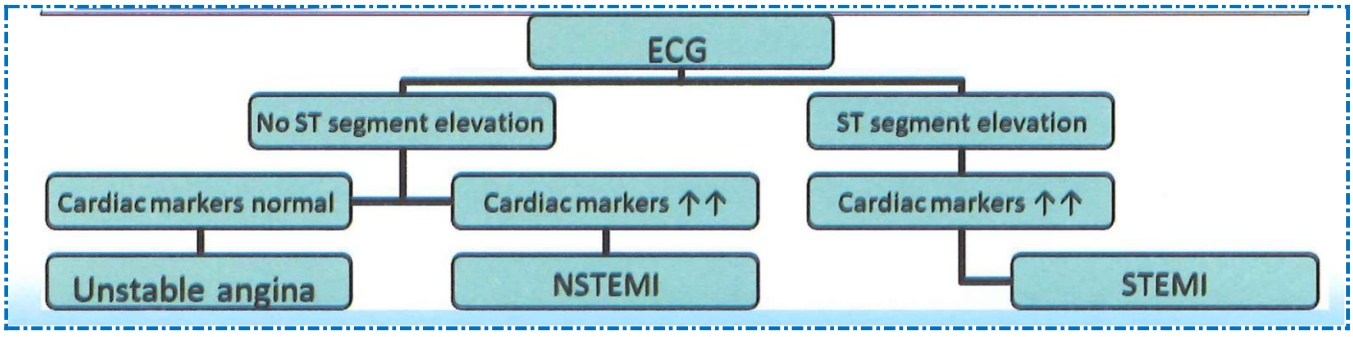
INVESTIGATIONS :

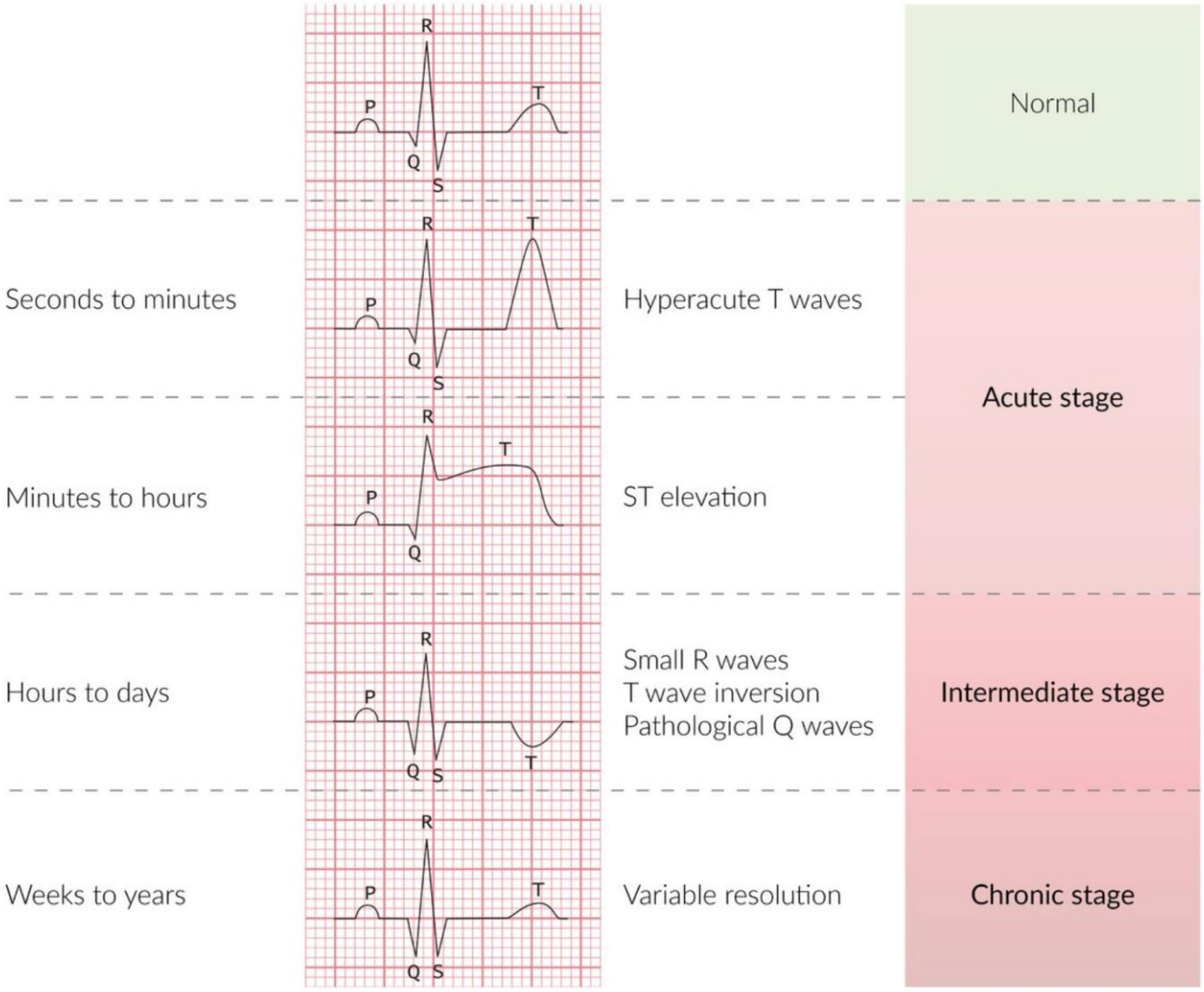
A. ECG

"HYPERACUTE T WAVE IS THE EARLIEST SIGN"

Ischemia	Inverted T wave
Infarction	Pathological Q wave
Recent infarction	Elevated convex ST segment (injury pattern)
MI (Typical ECG pattern)	Recent (elevated ST) Anteroseptal (V 1 - V 4)

A normal ECG does not rule out acute myocardial infarction





TIMELINE OF ECG CHANGES IN STEMI

ACCORDING TO ST SEGMENT, THERE ARE TWO TYPES OF MI

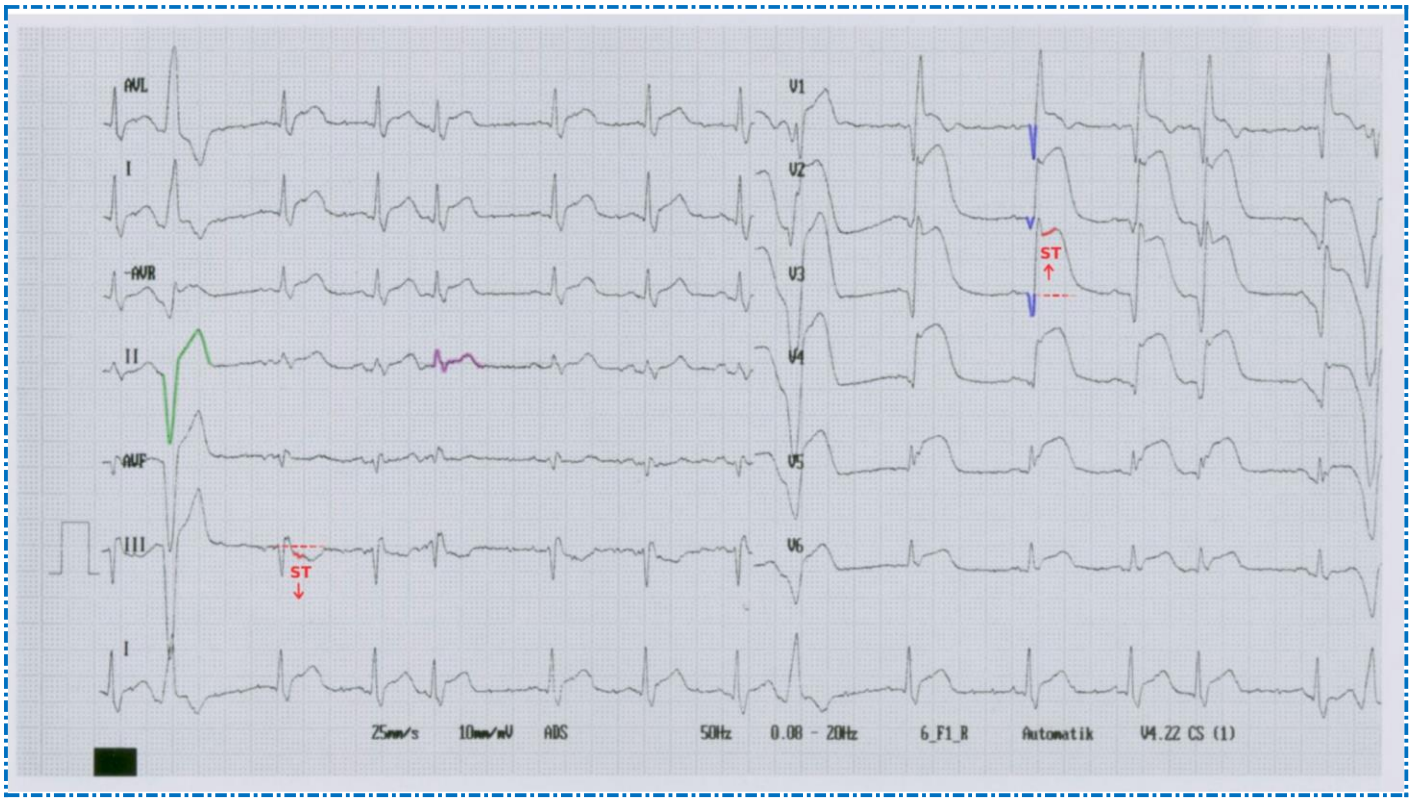
1. **AMI (with ST elevation):** Transmural MI (usually). (Q - wave MI)
   - It is characterized by ischemic necrosis of the full thickness of the affected muscle segment, extending from the endocardium through the myocardium to the epicardium.

2. **AMI (without ST elevation):** Non - Transmural MI (usually). (Non - Q wave MI), diagnosed by ↑↑ enzymes
   - It is characterized by an area of ischemic necrosis that does not affect the full thickness of the muscle segment but is limited to either: the endocardium or the endocardium & myocardium

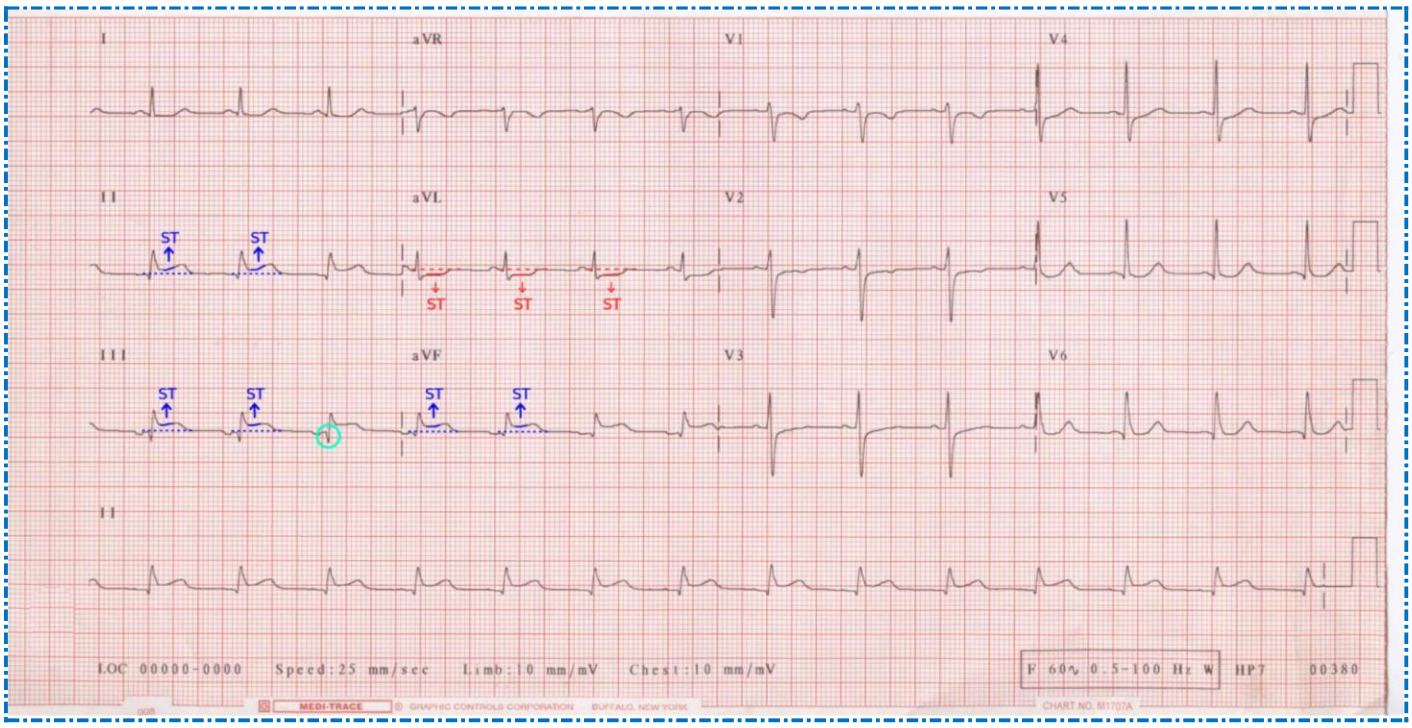
LOCALIZATION OF MYOCARDIAL INFARCT ON ECG:

ECG leads affected	Infarct location	Vessel involved
V1- V6	◆ Extensive **anterior MI** (leads Avl and I may also be affected).	◆ Proximal left anterior descending intery (LAD)
V1- V2	◆ Antero septal MI	◆ LAD
V3- V4	◆ Antero apical MI	◆ Distal LAD
V5- V6	◆ Antero lateral MI	◆ Diagonal branch of LAD ◆ Distal LAD ◆ Left circumflex artery (LCX) ◆ In rare cases, can also be affected by RCA infarct
I, aVL	◆ Lateral MI	◆ Proximal LCX
II, III, Avf V3R – V6R	◆ Inferior MI	◆ RCA (More common) ◆ Distal LCX (Less common)
V7 – V9	◆ Posterior/posterolateral MI	◆ Posterior descend artery from RCA or LCX ◆ Reciprocal ST depression in V1-4.

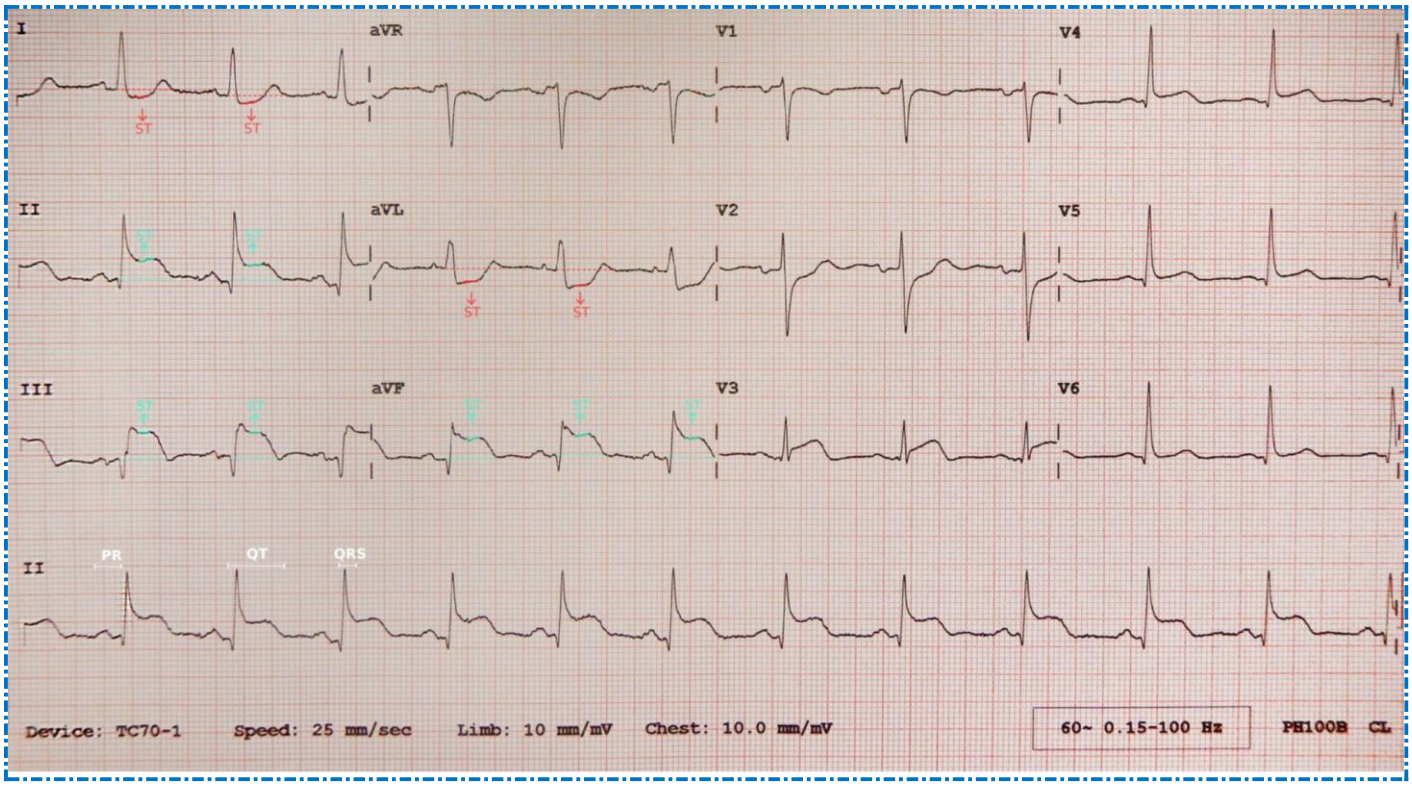
- Infarction of the **anterior wall** is caused by obstruction of the **LAD** or its branches. Depending on the extent of anterior wall infarction, results in ECG changes in the anterior wall leads **(V1-6)** and/or **I and Avl.**
- Infarction of the **inferior wall** is caused by obstruction of the **LCX** or **RCA** or their branches, and ECG changes are seen in leads **II,** III, and **aVF**
- To remember the ECG leads with maximal ST elevation in **anterior** MI think **"SAL"** **S**eptal (V1-2). **A**pical (V3-4), **L**ateral (V5-6L



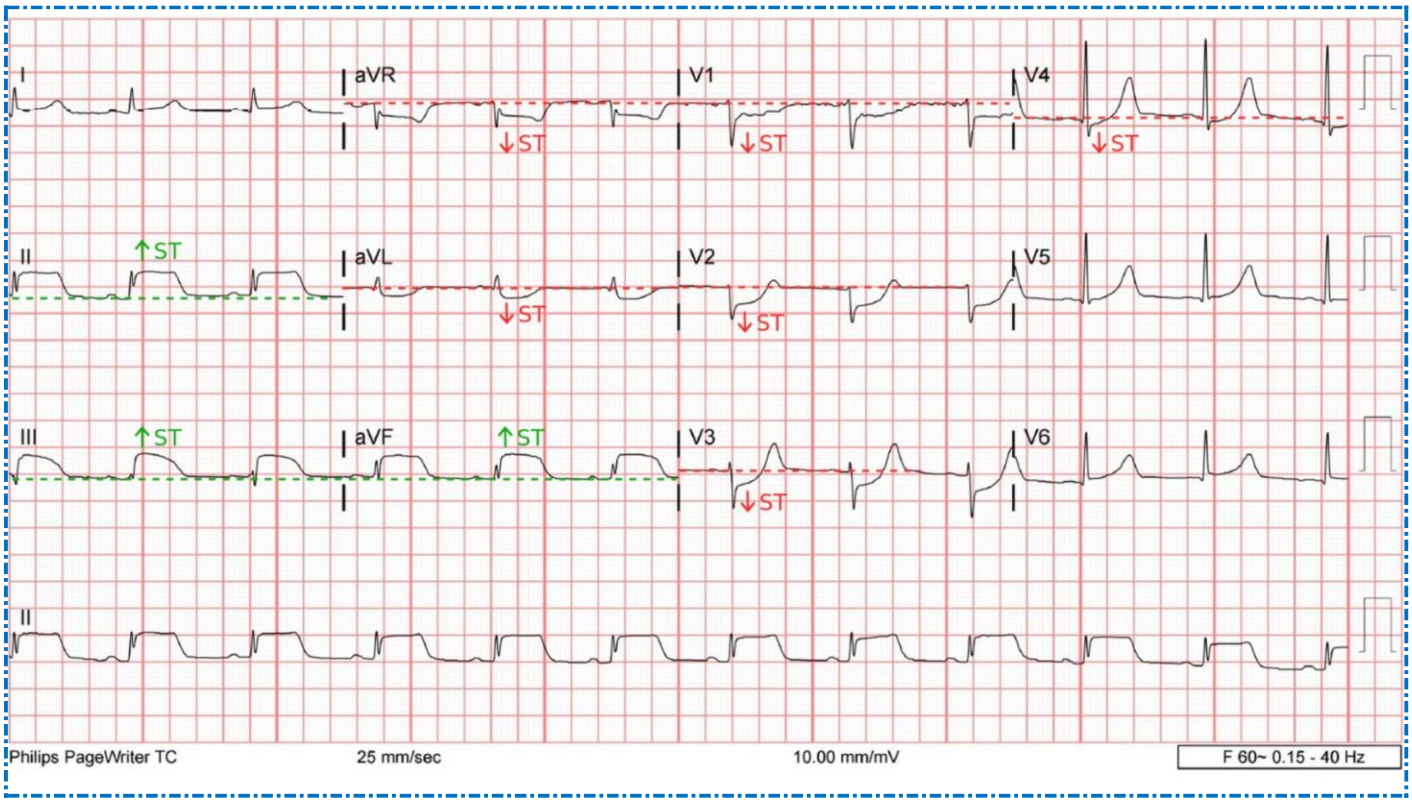
ACUTE ANTERIOR ST ELEVATION MYOCARDIAL INFARCTION (STEMI)



ACUTE INFERIOR ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI)



ACUTE INFERIOR ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI)



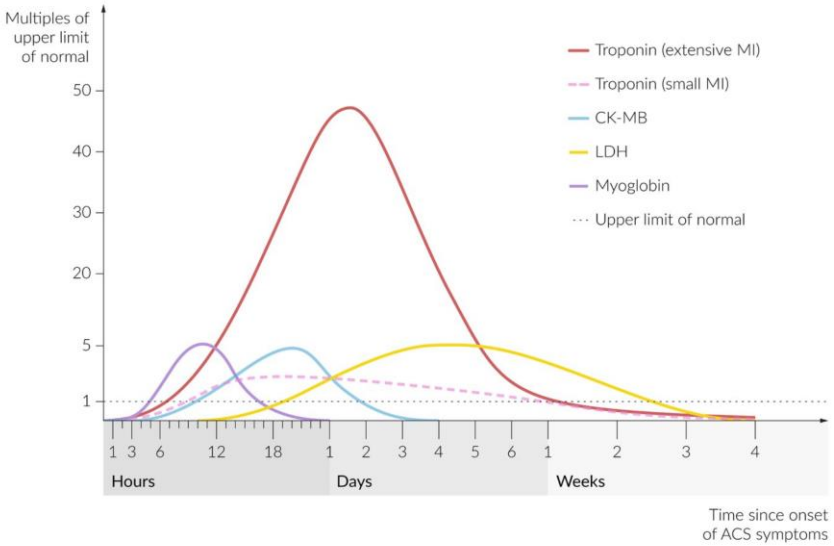
INFERO-POSTERIOR MYOCARDIAL INFARCTION

B. Cardiac Markers

TROPONINS (cTn)	
Most sensitive & specific markers of myocardial injury	
Types	◆ cTn T & cTnI (highly sensitive tests are more accurate: hs-cTnT, hs-cTnI
Level	◆ cTn must be above the 99th percentile of the URL (to diagnose AMI).
Time course	◆ ↑ é in 4-8h (2-3 hé hs test), peak é in 24-48 h, normalize é in 6-10 d.
Specificity	◆ cTn is organ-specific (Myocardium), not disease-specific (not specific to AMI).
Causes of ↑ ↑	◆ **Myocardial injury with ischemia** • Acute Coronary Syndrome (STEMI, NSTEMI) • Other Coronary Ischemia • Arteritis (e.g. SLE), CCS é ↑ O_2 demand (e.g. tachy-), Dissection, Emboli, Spasm • Non-Coronary Ischemia (Shock, Hypoxia, Pulm. emb, Global ischemia) ◆ **Myocardial injury with No ischemia** • Comorbidities (Renal failure, Respiratory failure, Sepsis, Stroke) • Others (Myocarditis, HCM, Heart failure, Cardiac contusion, DC cardioversion)

OTHERS	
CK-MB	↑ é in 4-8 h, peaks é in 24 h, normalizes é in 2-3 d.
Myoglobin	↑ éin 1-2 h, peaks é in 4-12 h, normalizes immediately thereafter
H-FABP 4	↑ é in 1 h, peaks é in 4-6 h, normalizes é in 24 h
Therefore it has 2 advantages: early marker of AMI, a marker of re-infarction	

BIOMARKER	RISE	MAXIMUM	NORMALIZATION	CHARACTERISTICS
TROPONIN T/I	**Regular assays:** 6-8 h **High-sensitive assays:** 1-3 h	12-24 hours	7-10 days	◆ Cardiac-specific marker with high sensitivity for **myocardial necrosis** ◆ The degree of elevation correlates with the size of the infarct and risk of mortality.
CK-MB	4-9 hours	12-24 hours	2-3 days	◆ No longer commonly used clinically; has been replaced by cardiac troponin in the diagnosis of ACS ◆ CK-MB is more specific to cardiac tissue than total CK (but may also be due to skeletal muscle injury). ◆ Can be helpful for **evaluating reinfarction** because of its short half-life but is no longer commonly used ◆ The degree of elevation often correlates with the size of the infarct.
MYOGLOBIN	1 hour	4-12 hours	24 hours	◆ **Nonspecific marker** that is no longer commonly used



C. General evidence of tissue damage

- Leukocytosis
- ↑ ESR
- ↑ CRP (hs-CRP)

→ **ACUTE PHASE REACTANTS**

D. IMAGING

Non-invasive	◆ **Echocardiography:** • Akinesia of the inf arcted area. • Complications, e.g. MR, Myocardial aneurysm, Mural thrombosis. ◆ **Isotopic studies:** • **Thallium imaging:** infracted area appears as cold spot. • **Technitium imaging:** infracted area appears as hot spot.
Invasive	**Coronary arteriography** ◆ To demonstrate: the site of the occluded vessel. ◆ To assess: the condition of the other coronaries
Recent	◆ **Multi - slice CT, MR - CA.** ◆ **IVUS.**

DIAGNOSTIC CRITERIA OF MI (WHO CRITERIA) :

1. **HISTORY:** Ischemic chest pain > 20 minutes **(TYPICAL CHEST PAIN).**
2. **ECG:** Changes in serial ECG tracings **(TYPICAL ECG).**
3. **ENZYMES:** ↑Troponins, CK-MB. **(TYPICAL ENZYMES).**

DIFFERENTIAL DIAGNOSIS:

- From all causes of acute chest pain (refer to **angina**).

BAD PROGNOSTIC CRITERIA IN AMI

Patient	◆ **Age**: old. ◆ **Heart:** originally unhealthy **weak** or **dilated** cardiac muscle. ◆ Associated risk factors of atherosclerosis & CAD: e.g. DM, HTN.
Infarction	◆ **Site:** extensive anterior is worse than lateral & inferior, ◆ **Size:** the larger, the worse the prognosis.
Management	◆ **Delayed**: and inability to apply early thrombolytic therapy. ◆ **Ineffective:** Failure of the patient's condition to stabilize during the first few days of management.
Complications	◆ Development of severe complications: e.g. cardiogenic shock. ◆ Persistent elevation of the ST segment in ECG.

TREATMENT:

First goal	◆ The first goal is to diagnose as soon as possible whether the patient is having an **STEMI** or **NSTEMI** because therapy differs between the 2 types of AMI
Initial treatment	◆ Initial ttt is directed toward restoration of perfusion as soon as possible to save as much of the jeopardized myocardium as possible. ◆ This may be accomplished through: medical or mechanical means (**PCI or CABG**).
Further treatment	◆ **Further treatment is based on the following:** • Restoring the balance between O2 supply & demand to further ischemia. • Pain relief. • Prevention & treatment of any complications that may arise.

1. PRE-HOSPITAL PHASE

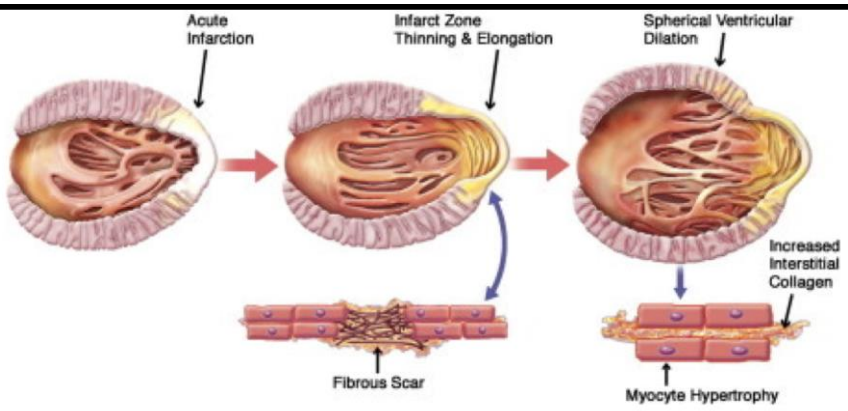
- Approximately **65%** of deaths caused by AMI occur in the first hour.
- In order to reduce this high mortality substantially, we should focus our attention on the prehospital care of this disease.
- An early prehospital ECG & communication with a hospital capable of percutaneous intervention (PCI) by EMS significantly improve the outcome.

a) Analgesics for chest pain:	
Nitroglycerin	◆ sublingually or by spray.
Morphine	◆ 10 mg IV or pethidine: 50 mg IV
b) Prophylaxis & TTT of malignant ventricular arrhythmias:	
Lidocaine	◆ 100 mg IV
c) Treatment of increased vagal tone (causing bradycardia):	
Atropine	◆ 1 mg IV. ◆ If bradycardia persists→ pacing is needed.
d) Prehospital Fibrinolysis:	
Indication	◆ If PPCP cannot be performed within 120 min. from STEMI diagnosis.
Timing	◆ initiate Fibrinolysis immediately, within 10 min of STEMI diagnosis.
e) Antiplatelets:	
◆ Immediate low dose aspirin (162-325 mg) administration further protects the myocardium.	
f) Oxygen inhalation.	

2. HOSPITAL PHASE

1. GENERAL	
Monitoring	◆ Continuous hemodynamic & ECG monitoring
Management of urgent comp. e.g.	◆ **Fatal Ventricular arrhythmias:** DC cardioversion. ◆ **Heart block:** Pacing.
Rest	◆ Physical, Mental (sedatives, e.g. Diazepam 2-5 mg tds)
Diet	◆ Light frequent meals & stool softeners to avoid constipation.
Control of associated DM	◆ Diet control. ◆ Drugs: insulin (small doses of short acting). ◆ Avoid: hypoglycemia.
Prophylaxis against stress ulcer	◆ Proton pump inhibitors (c.g. Omeprazole), or ◆ H_2 receptor antagonists (c.g. Ranitidine).
Oxygen inhalation	

2. Refractory or continuous chest pain	
◆ IV morphine or pethidine.	◆ IV or oral B-blockers.
◆ IV Nitroglycerine (10-200 µg/min).	◆ OXYGEN INHALATION.

3. Reduction of LV Remodeling	
Definition	◆ Changes in the size, shape, structure & function of LV in response to cardiac injury (typically post AMI).
Pathology	
Culprits	◆ Angiotensin II, Aldosterone,Norepinephrine.
Course	◆ Development & progression of LV dysfunction, LVF, arrhythmias and maybe aneurysm formation & LV rupture (poor prognosis).
Drugs	◆ RAAS blockers (ACE-I or ARB), MRA4,βB

4. Reduction of the work of the heart	
Nitrates: (IV nitroglycerine)	◆ Given in the first days following AMI.
B-blockers	Given in the first days following AMI.

5. Medical revascularization "Fibrinolysis"	
Timing	◆ initiate Fibrinolysis immediately, within 10 min of STEMI diagnosis
Fibrinolytic drugs	◆ **Fibrin specific agents** (c.g. Alteplase, Tenecteplase).
After Fibrinolytic drugs	◆ **Antipaletelet** (Clopidogrel plus LDA) & **Anticoagulant** (Heparin), are given with & after fibrinolytic drugs.
Complications	◆ Bleeding. ◆ Reperfusion arrhythmias (esp. AIVR Accelerated Idio-Ventricular Rhythm).
Indications	◆ **Not in:** NSTEMI (no benefit in NSTEMI). ◆ **Only in:** STEMI : "If the patient initially presents to a non PCI-hospital and, while planning to transfer him to a PCI-hospital: PPCI cannot be performed within 120 min. from STEMI diagnosis.
C/I	◆ **Bleeding** { General (platelets < 100,000/mm², coagulopathy), or stroke (ICH)}. ◆ Hepatic failure. ◆ Hypertension (SEVERE). ◆ Aortic dissection. ◆ Active peptic ulcer. ◆ Advanced age: >75 years (relative contraindication). – TIA

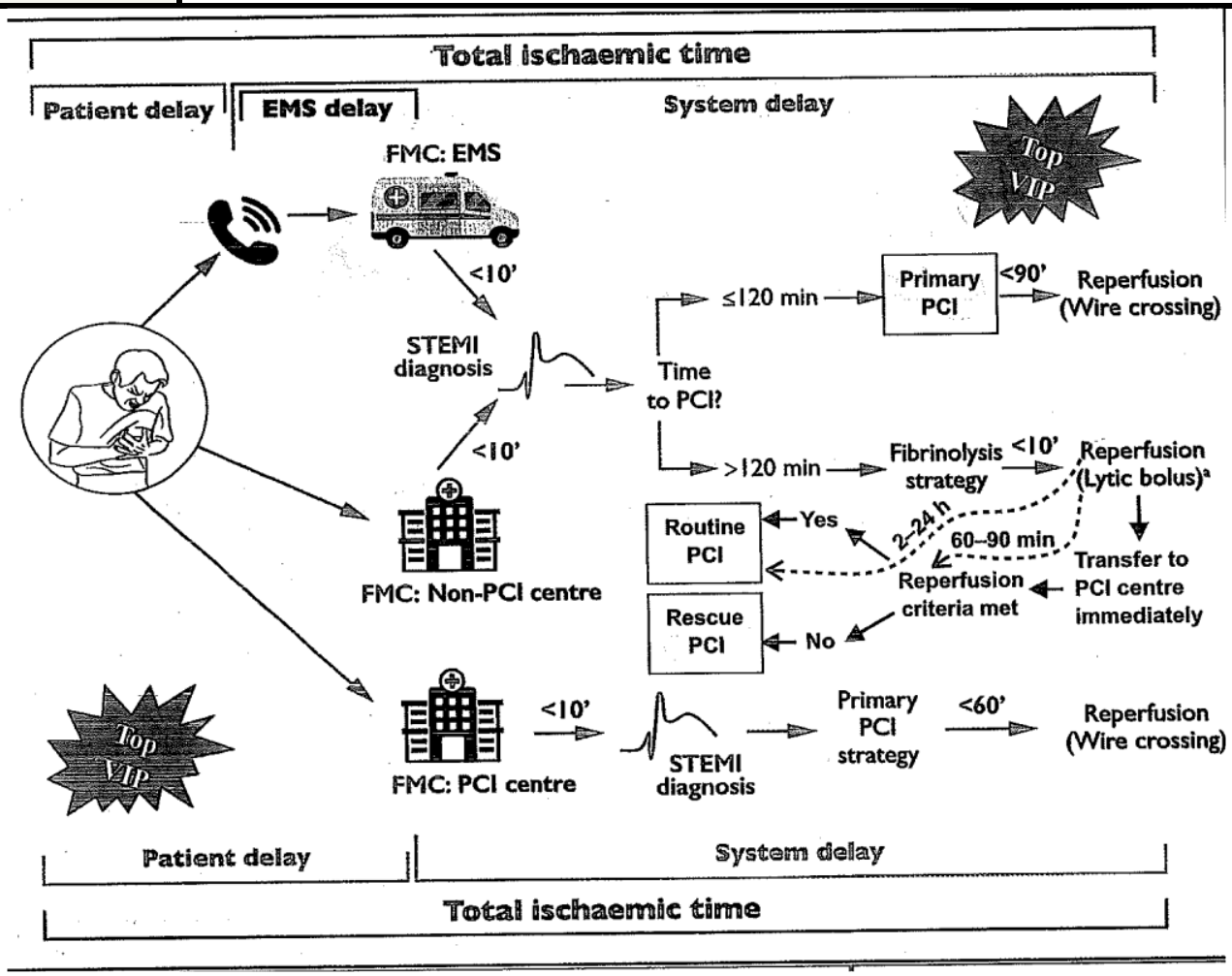
6. Mechanical revascularization

A) PERCUTANEOUS CORONARY INTERVENTION (PCI): (IN A PCI - CENTRE)

1. Primary :	**PCI without prior fibrinolysis** ◆ STEMI when rapid access is possible (Time to PCI < 120 min). ◆ STEMI contraindicated to fibrinolysis (regardless of Time to PCT).
2. Routine	**PCI following successful fibrinolysis** ◆ When Reperfusion criteria are **met** after fibrinolysis. ◆ **Value:** enhances reperfusion, the risk of re-occlusion. ◆ **Adjuvant ttt:** Aspirin, Clopidogrel, Glycoprotein IIb/IIIa inhibitors, and IV heparin to the risk of ischemic complications during the procedure.
3. Rescue	**PCI following failed fibrinolysis** ◆ When Reperfusion criteria are **not met** after fibrinolysis.

B) SURGERY: (CABG)

Indication	**less commonly implemented than PCI or Fibrinolysis** ◆ Failure of PCI. ◆ Sudden occlusion of a coronary artery during catheterization. ◆ In association with surgery for complications, e.g. MR or VSD.



7. Anticoagulants
◆ With & after fibrinolytic therapy. ◆ Thrombo-embolic complications. ◆ Intracardiac thrombi detected by echocardiography

8. Antiplatelets	
Aspirin (low dose)	◆ 75-300 mg/d (single dose).
Clopidogrel	◆ 75 mg once/d.

9. Treatment of early complications	
Medical ttt	◆ e.g. Pump failure, shock, arrhythmias
Surgical ttt	◆ e.g. closure of the VSD.

3. POST-HOSPITAL PHASE = After Discharge

A. TREATMENT OF LATE COMPLICATIONS

- **Medical ttt:** e.g. corticosteroids & physiotherapy for Frozen shoulder syndrome.
- **Surgical ttt:** e.g. aneurysmectomy for Myocardial aneurysm.

B. REHABILITATION

- Gradual return to normal activity.
- Regular exercise.

C. CONTROL OF RISK FACTORS OF ATHEROSCLEROSIS

- e.g. Diabetes, HTN, Dyslipidemia, etc

D. ↓ RISK OF POST-INFARCTION ANGINA & RE-INFARCTION

- Aspirin.
- B-blockers.
- Statins.

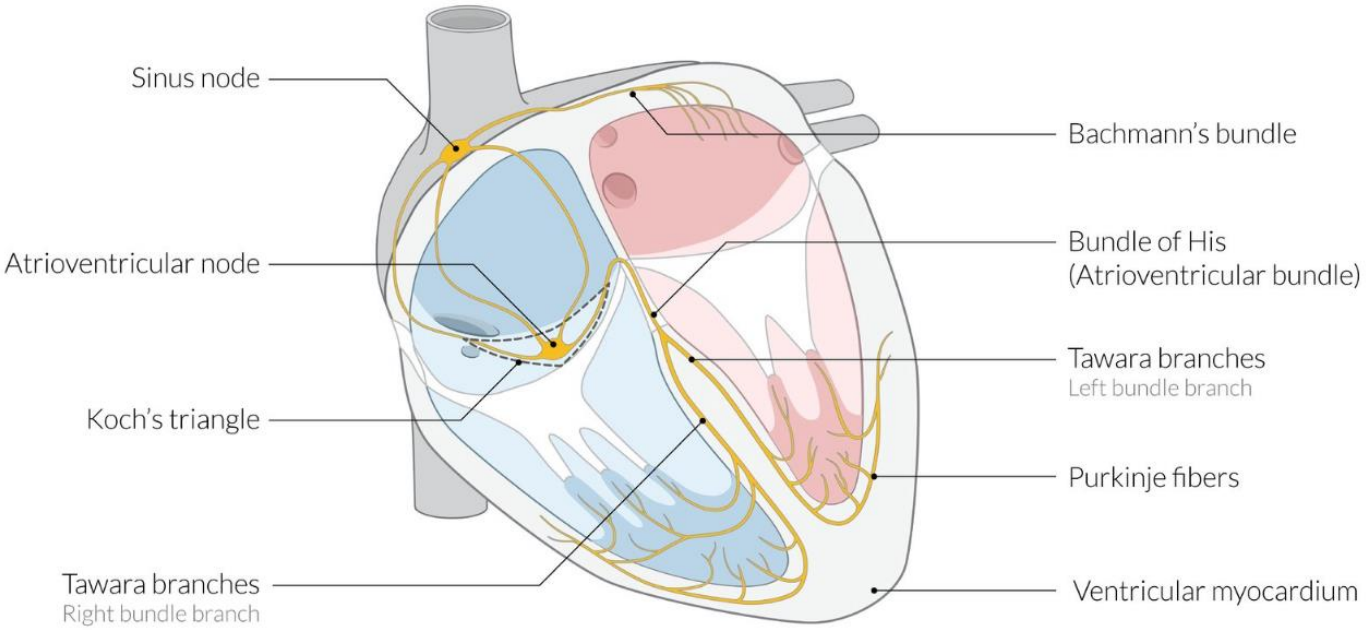
CARDIAC ARRHYTHMIAS

DEFINITION

- **Abnormality** of the cardiac Rhythm or Rate.
- **Normally:** Rhythm is Sinus & Regular, and the Rate is 60-100 beats / min.

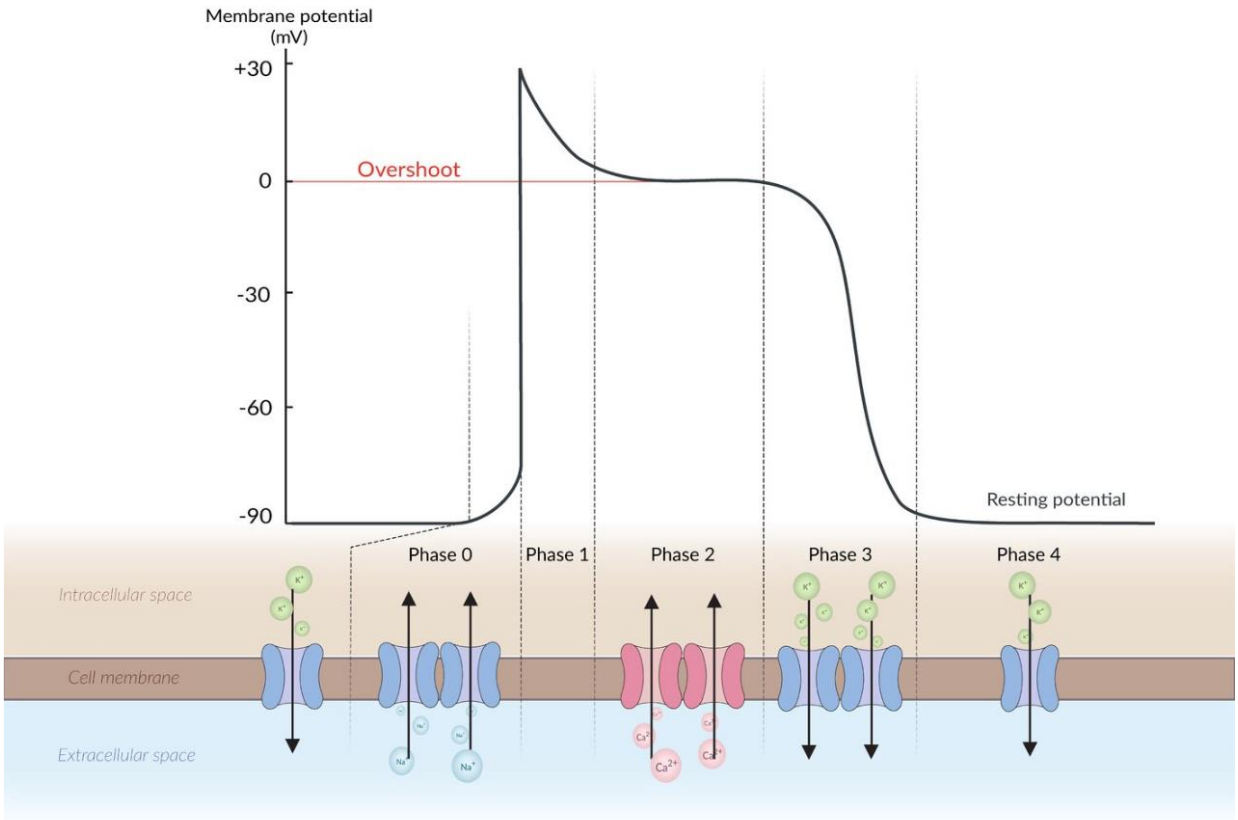
NORMAL SEQUENCE OF CARDIAC IMPULSE :

Sino-Atrial Node (SAN)	• **Cardiac impulse** arises from SAN (pace-maker): SAN discharges at a rate 60-100/min. • **Sympathetic stimulation** accelerates the SAN & **vagal stimulation** slows it.
Atria	• **From** the SAN, the impulses spread to excite both atria→ Atrial contraction. • Atrial contraction is represented by **(a wave in NVS), (p wave in ECG).** • **Sympathetic stimulation** ↑ atrial excitability & **vagal stimulation**↓ it.
Atrio-Ventricular Node (AVN)	• Impulses pass from the atria to the ventricles through the AVN **whose functions are:** 1. Allows passage of impulse from Atria to Ventricles, represented by (PR interval in ECG). 2. Physiological delay. 3. No Retrograde conduction. 1. 4.Surrogate pacemaker. 4. Electric filter. • **Sympathetic stimulation** accelerates conduction in AVN & **vagal stimulation** slows it.
Ventricles	• Impulses pass from the AVN to the Bundle of His in the interventricular septum, then along 2 bundle branches & finally Purkinje fibers to terminate in ventricular myocardium → ventricular contraction. • Ventricular contraction is represented by **(Radial pulse & Si clinically), (QRS in ECG).** • The ventricles are supplied by sympathetic fibers only **(no vagal supply).**



PHASES OF ACTION POTENTIAL

Zero	Rapid Depolarization	Entry on Na+ inside the cell
1	Early rapid Depolarization	Exit of K+ outside the cell
2	Plateau	Exit of K+ outside is equilibrium by Ca++ influx
3	Final rapid Depolarization	Exit of K+ outside the cell
4	RMP (Resting Membrane Potential)	



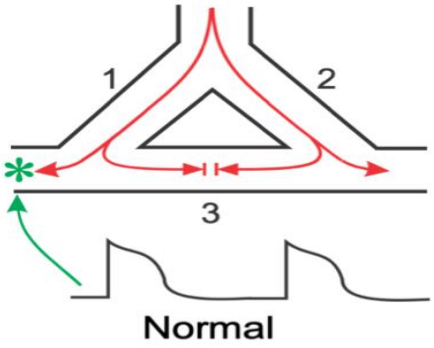
MECHANISMS OF ARRHYTHMIAS :

(A) DISTURBANCE OF IMPULSE FORMATION:

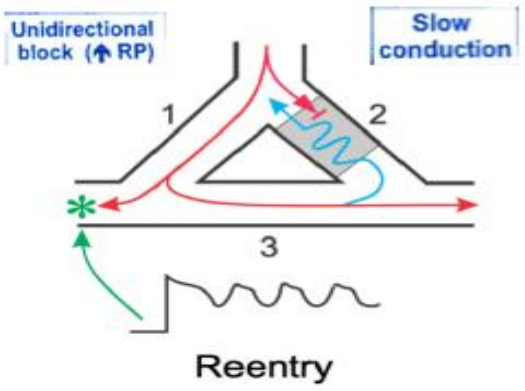
Disturbed normal automaticity:	◆ SAN shows spontaneous depolarization during **diastole (phase 4)** action potential → new action potential when the electric threshold is reached. ◆ Such diastolic depolarization may be accelerated or slowed by: **changing its rate.** ◆ These changes will produce **sinus tachycardia** or **sinus bradycardia**, respectively.
Abnormal automaticity:	◆ **NORMALLY:** • The normal working myocardial cells remain in a resting state at a high negative RMP (-90 mV), depolarizing only when stimulated by SAN. ◆ **IN CERTAIN DISEASES (E.G.ISCHEMIA) :** • RMP of these cells is reduced (-60 mv), Such cells may exhibit spontaneous diastolic depolarization and act as abnormal pace-makers, independent on the SAN; This may lead to: Atrial or Ventricular or Nodal (junctional) arrhythmias.

(B) DISTURBANCE OF IMPULSE CONDUCTION:

Decreased conduction:	◆ There is lost ability of action potential to stimulate myocardial cells → Heart block.
Re-entry:	**"2 conditions are required for re-entry to occur"** ◆ A wave of depolarization may be forced to travel in one direction around a ring of cardiac tissue. A circus movement will result producing a tachycardia.



Normal



Reentry

OPPOSING IMPULSES KILL EACH OTHER
THEREFORE, IMPULSE DIES AFTER STIMULATION

CLINICAL CLASSIFICATION:

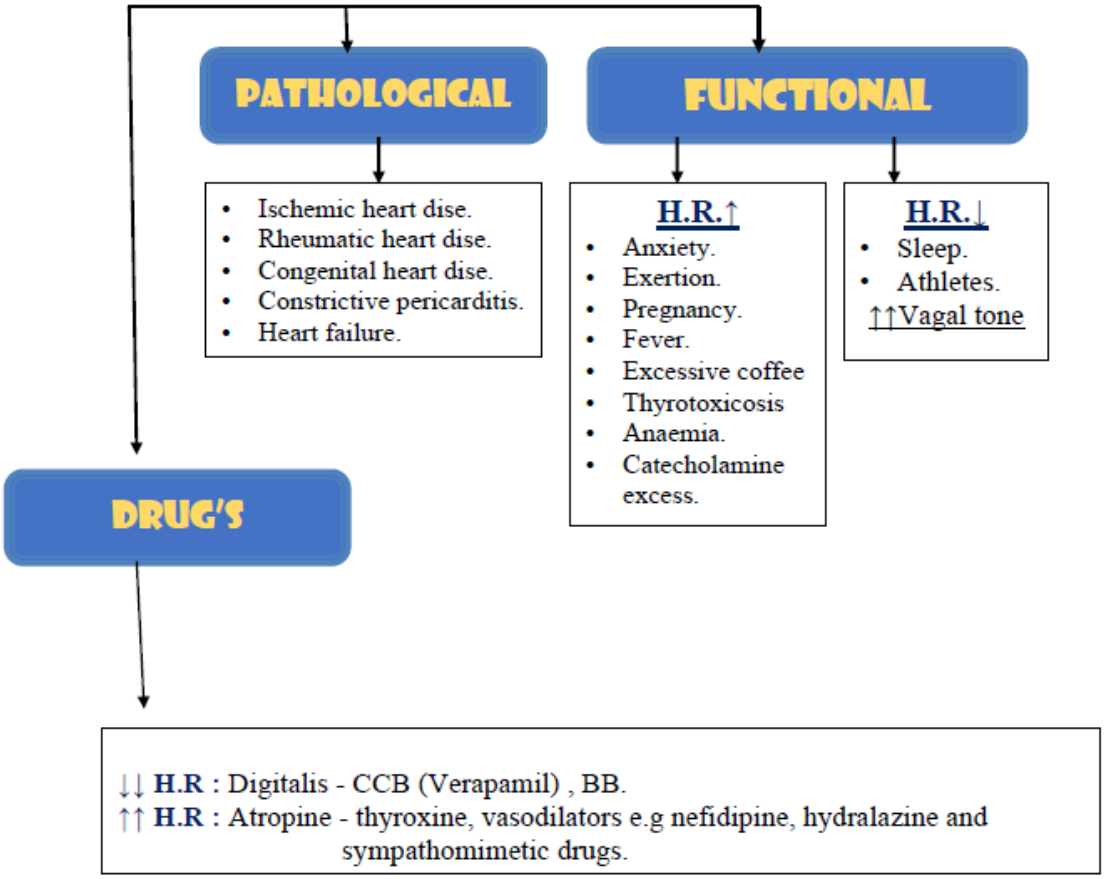
Sinus Rhythm Disturbances	1. Sinus tachycardia. 2. Sinus bradycardia. 3. Sinus arrhythmia.
Pathological Tachy - Arrhythmias:	1. Supraventricular tachycardia (SVT). 2. Atrial flutter. 3. Ventricular tachycardia (VT). 4. Atrial fibrillation (AF).
Pathological Brady - Arrhythmias	1. Nodal (Junctional) rhythm. 2. Heart block. 3. Sick sinus syndrome.
Others	1. Premature beats (Extrasystoles). 2. Pre-excitation syndromes.

General Scheme For Arrhythmia

PATHOPHYSIOLOGY:

- According to the type of the arrhythmia.

AETIOLOGY:



CLINICAL PICTURE :

A) SYMPTOMS: (COMMENT ON THE FOLLOWING):

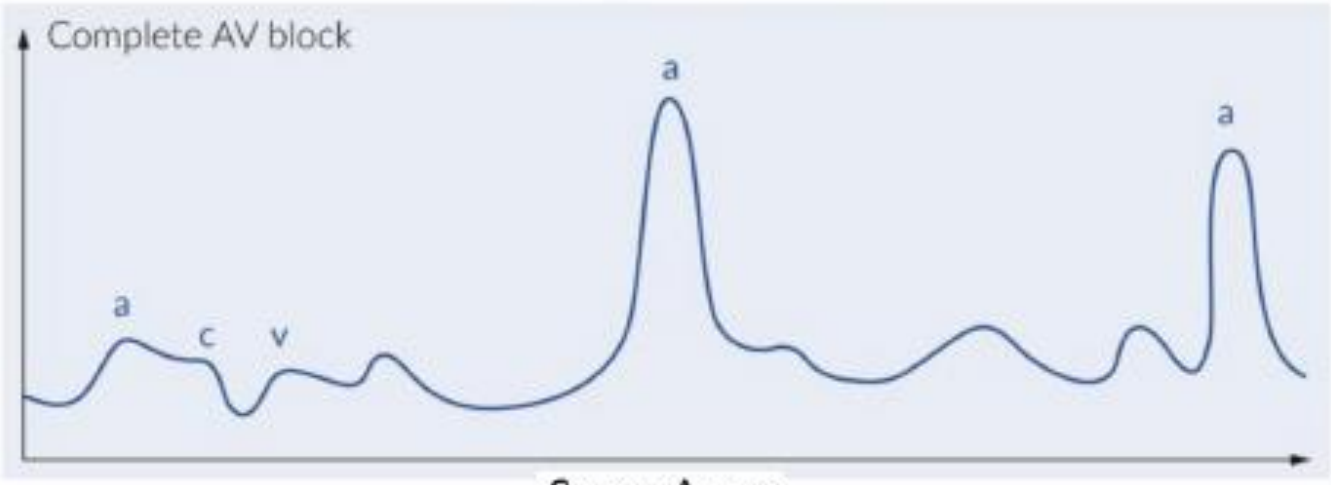
1. asymptomatic
2. Palpitation:
   - Onset.
   - Offset.
   - Regular or irregular.
   - Rapid or slow.
   - Duration.
3. Symptoms of low COP
4. Precipitation of heart failure and angina .
5. Manifestations of the cause:
   - Rheumatic heart disease.
   - Ischemic heart disease.
   - Congenital heart disease

B) SIGNS: (COMMENT ON THE FOLLOWING):

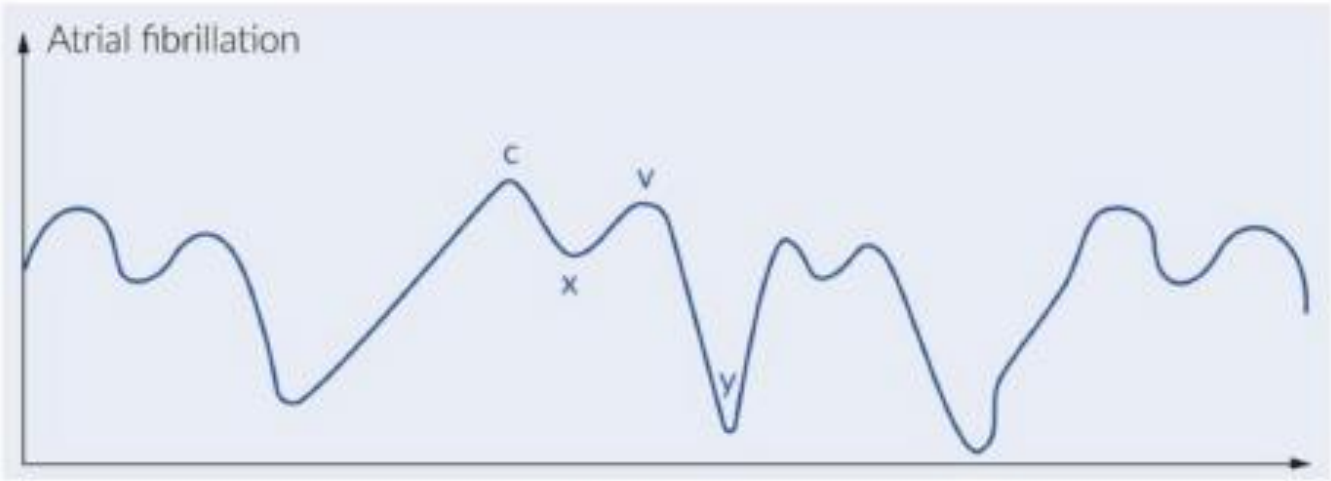
Pulse	a. **rate :** increase in tachy , decrease in bradyarrythymias. b. **rhythm:** all are regular except AF and extra systoles , atrial flutter with variable block and 2nd degree heart block mobitz 2 with variable block. c. **Carotid sinus massage:** Decrease HR in any tachyarrythymia except arrythymias that originate in the ventricle. d. **Respiratory sinus arrhythmia:** negative on all arrythymias except in both sinus tachy and bradyarrythmias. **N.B :** in bradyarrythymias : response to atropine instead
Neck veins	- **Rapid A wave** in atrial tachyarrhythmias. - **Loss of A wave** in atrial fibrillation - **Cannon A wave** in any nodal arrhythmias either : paroxysmal nodal tachycardia or nodal rhythm - **Occasional canon A wave:** ventricular tachycardia and complete heart block (atrio-ventricular dissociation)
Auscultation	- **Accentuated** in any tachycardia - **Weak** in any bradychardia

N.B

- Atrial fibrilation (af) : add thromboembolism
- Ventricular tachycardia (VT) : add sudden death
- Complete heart block : add syncope , sudden death



Cannon A wave



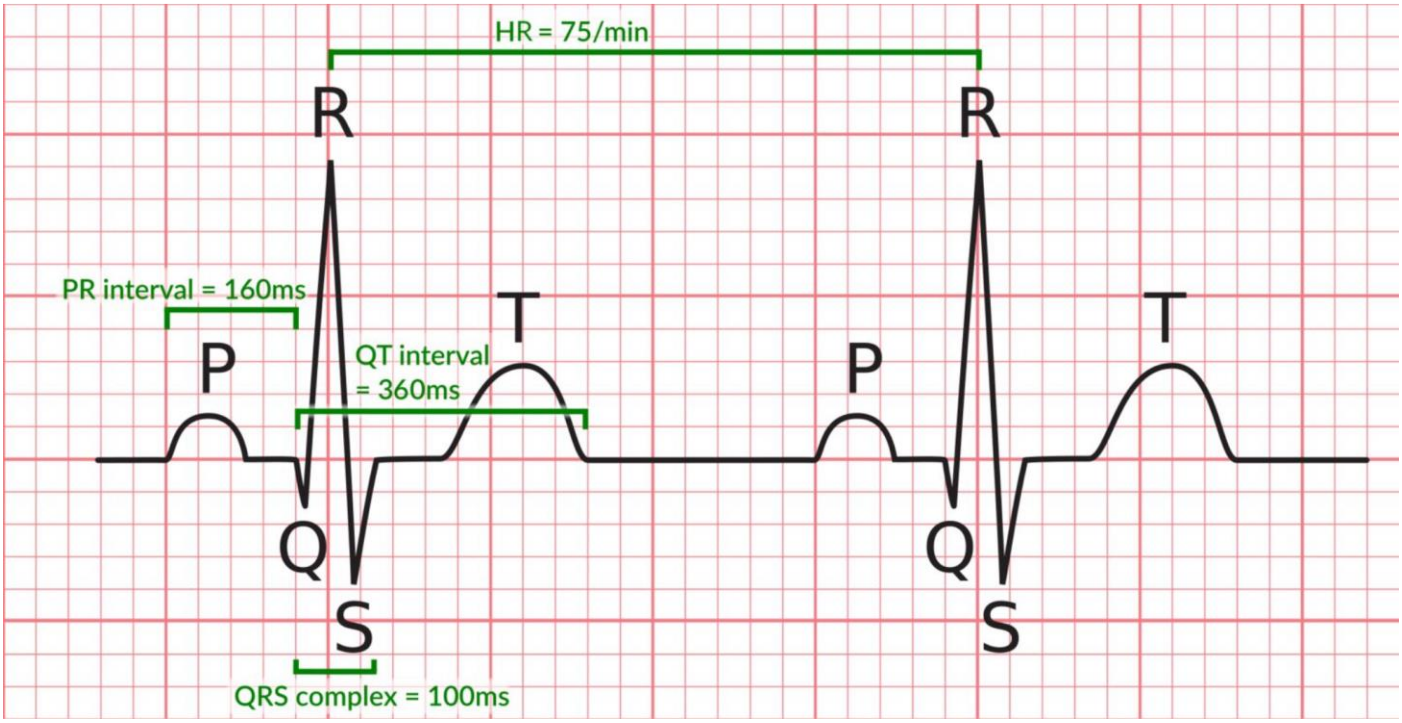
Absent A wave

N.B:

- **VARIABLE S1 IN :**
  - Atrial fibrillation
  - Ventricular tachycardia
  - Complete heart block
- **NODAL RHYTHM** → accentuated S1 inspite of bradychardia .

iNVESTiGATiON:

A. ECG



	REPRESENT	CHARACTER
P WAVE	Atrial contraction	◆ **Normal** in sinus arrhythmias. ◆ **Abnormal** in any other atrial arrhythmias. ◆ **Flutter wave** in atrial flutter. ◆ **Fibrillation waves** or even absent P wave in atrial fibrillation.
PR iNTERVAL	Passage of impulse from atria to ventricles	◆ **Short** in tachycardia. ◆ **Prolonged** in bradycardia. ◆ **AV dissociation** in : VT, CHB
QRS COMPLEX	Ventricular contraction	◆ **Regular** except in AF & extrasystole. ◆ **deformed (bizarre):** in VT & CHB.

B. iNVESTiGATiON FOR THE CAUSE :

ECHO	For detection of the cause as rheumatic heart or congenital heart disease .
LABORATORY	For the cause e.g. (T3, T4, TSH), Hb, serum K, Mg.

TREATMENT: See later

SINUS TACHYCARDIA

DEFINITION :

- The SAN discharges at a rapid rate > 100 / minute.

ETIOLOGY

Physiological	◆ Exercise. ◆ Pregnancy. ◆ Emotions. ◆ Infancy.
Pathological	◆ **Fever.** ◆ **Shock.** ◆ **Hyperthyroidism:** hyperdynamic circulation. ◆ **Hypovolemia:** hypotension. ◆ **Heart failure.** ◆ **Pulmonary embolism**
Pharmachological	◆ **Sympathomimetic drugs**: e.g. Adrenaline. ◆ **Parasympatholytic drugs**: e.g. Atropine. ◆ **CCBs:** e.g. Nifedipine. ◆ **Thyroid** hormones. ◆ Nicotine, Caffeine, Alcohol. ◆ Anemia. Myocarditis.

CLINICAL PICTURE

A) SYMPTOMS:

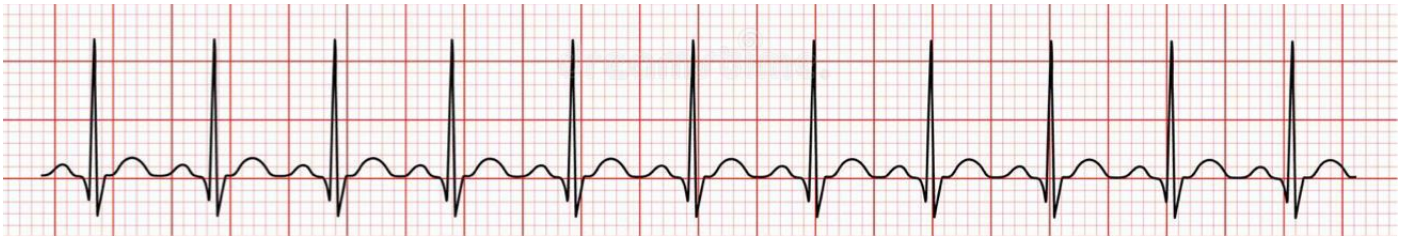
1) **Asymptomatic**.
2) **Palpitation:** Rapid, Regular, Gradual onset, Gradual offset.
3) **Symptoms of** low cardiac output.
4) **Precipitation of** angina & HF in susceptible patients.

B) SIGNS:

Pulse	◆ **Rhythm:** regular. ◆ **Rate:** 100-180/min. ◆ **Response to carotid massage:** • Gradual slowing: of the rate. • Gradual acceleration: to the original rate on stopping massage.
Neck veins	◆ Normal rapid waves with the same rate of pulse.
Auscultation	◆ Accentuated S1.

ECG

QRS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 100-180 /min ◆ **Duration :** normal.	◆ Normal.



TREATMENT:

1) **TTT of the cause:** e.g. antithyroid drugs for hyperthyroidism,
2) **Sedatives:** may be needed.
3) **B-blockers:** e.g. propranolol, in severe cases.

SINUS BRADYCARDIA

DEFINITION: :

◆ The SAN discharges at a slow rate < 60 / minute.

ETIOLOGY:

Physiological	◆ Athletes. ◆ Sleep.
Pathological	◆ Hypothermia ◆ Hypothyroidism ◆ Increased vagal stimulation, e.g. Vasovagal syncope. ◆ Increased intracranial tension, e.g. Brain tumours. ◆ CHOLESTASIS: Obstructive jaundice.
Pharmachological	◆ **Parasympathomimetic drugs**: e.g. Neostigmine. ◆ **Sympatholytic drugs**: e.g. B-blockers. ◆ **CCBs:** e.g. Verapamil & diltiazem. ◆ **DIGITALIS.**

CLINICAL PICTURE:

A) SYMPTOMS:

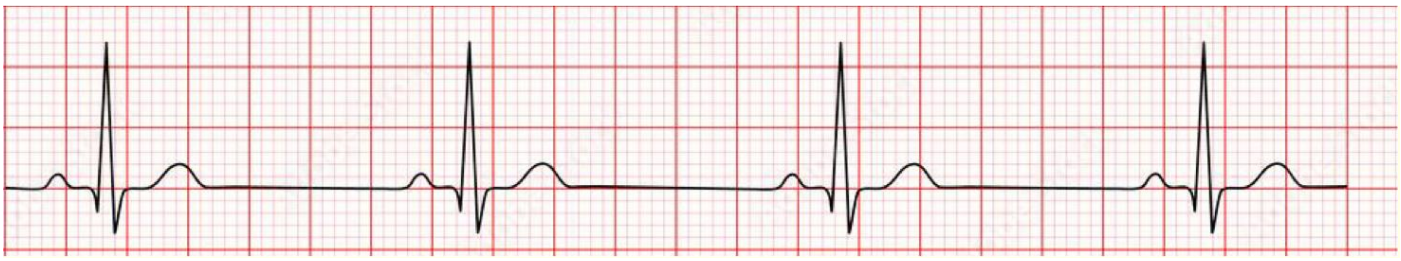
1. **Asymptomatic.**
2. **Palpitation:** Slow, Regular, Gradual onset, Gradual offset.
3. **Symptoms of** low cardiac output.
4. **Precipitation of** angina & HF in susceptible patients.

B) SIGNS:

Pulse	◆ **Rhythm:** regular. ◆ **Rate:** < 60 / minute (50 - 60 / minute, or less) ◆ **Response to carotid massage:** • Gradual acceleration: of the rate. • Gradual slowing: to the original rate on stopping exercise.
Neck veins	◆ Normal slow waves with the same rate of pulse.
Auscultation	◆ Normal or weak S1.

ECG

QRS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 50-60 /min, (or less). ◆ **Duration :** normal.	◆ Normal.



TREATMENT:

1. **TTT of the cause,** e.g. L - thyroxin for hypothyroidism
2. **Atropine:** may be needed.

SUPRAVENTRICULAR TACHYCARDIA

DEFINITION:

- It is an tachy-arrhythmia originating from above the ventricle.

TYPES:

- **Atrial tachycardia:** the arrhythmia originates from the Atria.
- **Nodal tachycardia:** the arrhythmia originates from the AVN.

ETIOLOGY

- It usually occurs in a normal heart, & therefore its presence does **not** always denote an organic heart disease.

Physiological	◆ occurs in a normal heart (no organic heart disease).
Pathological	◆ CAD (especially AMI). ◆ Cardiomyopathy (and myocarditis). ◆ Congenital heart disease. ◆ RHD. ◆ Hypertension. ◆ Hyperthyroidism. ◆ **Pre - excitation sundromes**: e.g. WPW syndrome.
Pharmachological	◆ **Digitalis.** ◆ Sympathomimetic drugs. ◆ Some anti-arrhythmic drugs e.g., class IC.

CLINICAL PICTURE:

A) SYMPTOMS:

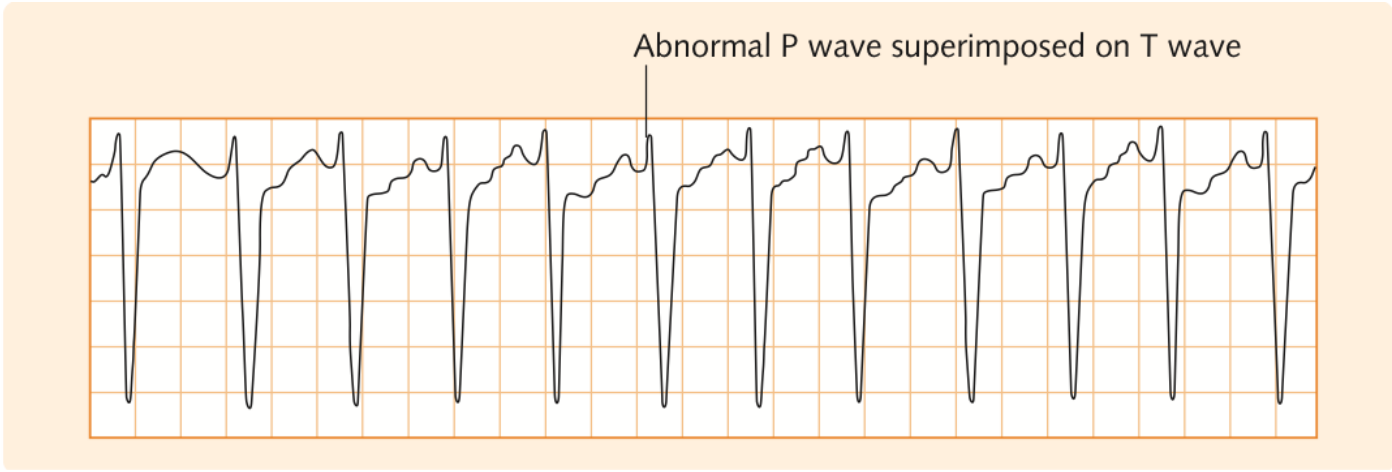
1. **Palpitation:** Rapid, Regular, Sudden onset, Sudden offset.
2. **Symptoms of** low cardiac output.
3. **Precipitation of** angina & HF in susceptible patients.

B) SiGNS:

Pulse	◆ **Rhythm:** regular. ◆ **Rate:** 120 - 250 / minute. ◆ **Response to carotid massage:** • Sudden disappearance: of the arrhythmia may occur.
Neck veins	◆ **In atrial tachycardia:** normal rapid waves with the same rate of pulse ◆ **In nodal tachycardia:** regular cannon waves with the same rate of pulse.
Auscultation	◆ **In atrial tachycardia:** accentuated S1. ◆ **In nodal tachycardia:** regular cannon sounds.

ECG

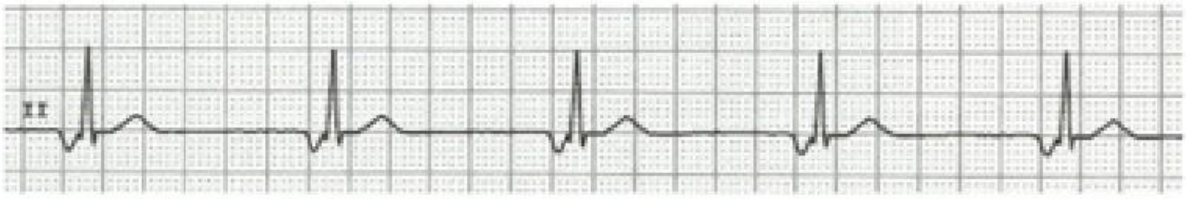
QRS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 120-250 /min ◆ **Duration :** normal.	◆ **In atrial tachycardia:** deformed. ◆ **In nodal tachycardia:** absent or inverted



iN ATRiAL TACHYCARDiA.

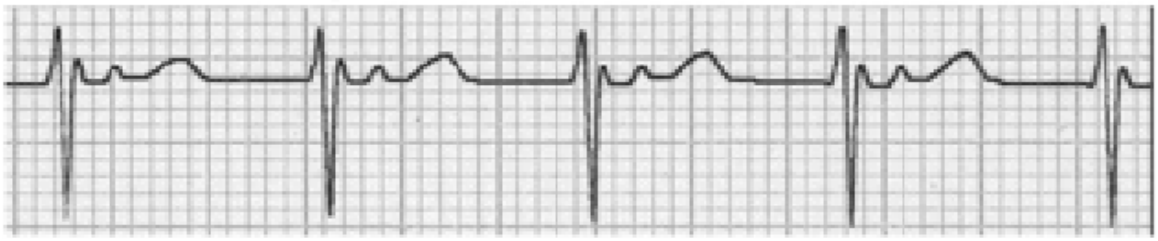
MiD-NODAL.

Junctional rhythm with inverted 'P' waves before QRS



HIGH-NODAL

Junctional rhythm with 'P' waves after QRS



LOW-NODAL

TREATMENT:

A- DURING THE ATTACK:

If the patient is hemodynamically stable:	
Vagal stimulation	Carotid sinus massage.
Slow the ventricular rate:	◆ Adenosine: 6 mg IV. ◆ B-blocker (propranolol): 5 mg IV. ◆ CCB (verapamil): 5 mg IV. ◆ Digitalis: 1 mg IV.
Restore sinus rhythm (Cardioversion):	**(after 1 the ventricular rate)** ◆ **Chemical cardioversion:** Class I A, IC or class III drugs. ◆ **Electrical cardioversion (DC):** if chemical cardioversion fails.
If the patient is hemodynamically unstable:	
DC cardioversion	
Overdrive pacing	◆ The atria are paced at a faster rate than the tachycardia rate. ◆ Sudden cessation of pacing is usually followed by: " restoration of sinus rhythm".

B-PREVENTION OF A FUTURE ATTACK:

Maintenance therapy	◆ **DRUGS: (Oral),** Class IA, IC or class III anti – arrhythmic drugs.
Intervention	◆ **Ablation of focus:** Catheter (Radiofrequency energy) or surgery.
TTT of the cause	

ATRIAL FLUTTER

DEFINITION :

- **Tachycardia** in which the atria discharge at regular rapid rate: 240 - 440 / min.
- A physiological block occurs in the AVN (2:1, or 3:1, or 4:1 block).
- Therefore only 1/2, or 1/3, or 1/4 of the atrial impulses will pass to the ventricles.
- The average ventricular rate will be: 1/2, or 1/3, or 1/4 the atrial rate.
- **The block may be:**
  - Fixed (e.g. 2:1), or,
  - Variable (e.g. changing from: 2:1 to 3:1, to 4:1.....etc....).

TYPES:

1. **Type I (common, typical):** the atrial rate is 240 - 340 / min.
2. **Type II (rare):** the atrial rate is 340 - 440 / min.

ETIOLOGY:

- It uaually occurs in a patient with ORGANIC HEART DISEASE, However, It may occur in a normal heart.

Physiological	◆ Occur in a normal heart (no organic heart disease).
Pathological	◆ CAD (especially AMI). ◆ Cardiomyopathy (and myocarditis). ◆ Congenital heart disease. ◆ RHD. ◆ Hypertension. ◆ Hyperthyroidism. ◆ **Pre - excitation sundromes**: e.g. WPW syndrome.
Pharmachological	◆ **Digitalis.** ◆ Sympathomimetic drugs. ◆ Some anti-arrhythmic drugs e.g., class IC.

CLINICAL PICTURE:

A) SYMPTOMS:

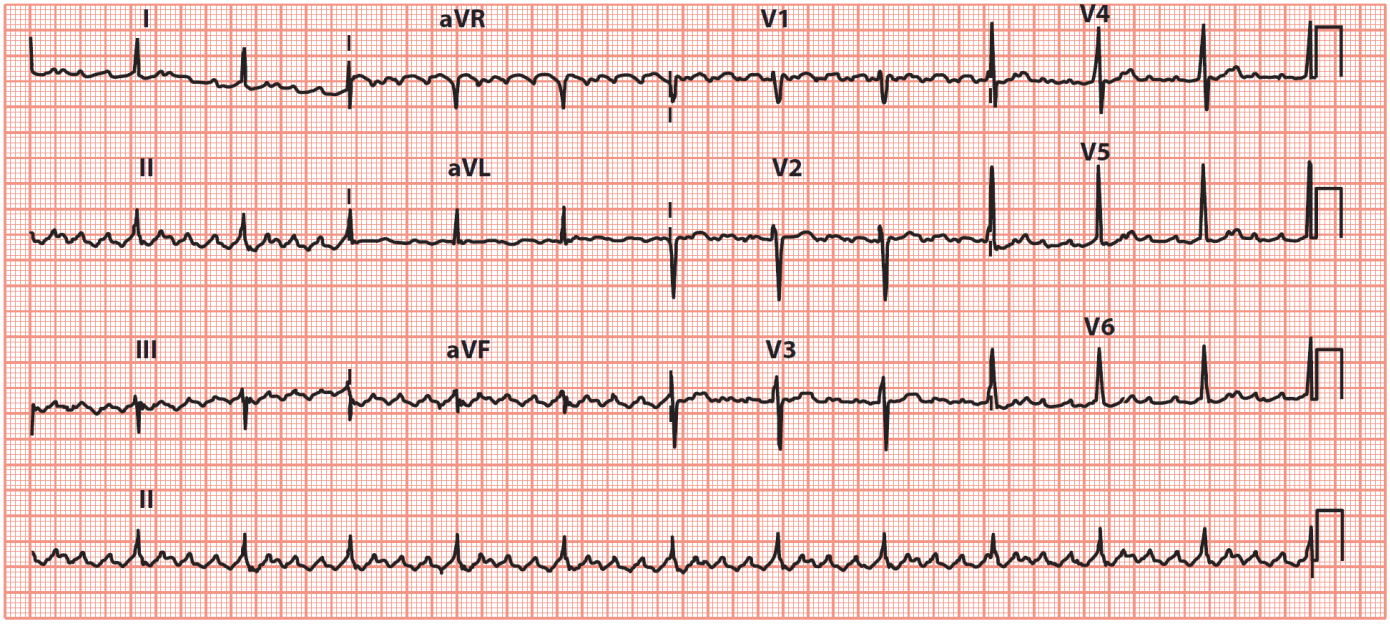
1. **Palpitation:** Rapid, Regular, Sudden onset, Sudden offset.
2. **Symptoms of** low cardiac output.
3. **Precipitation of** angina & HF in susceptible patients.

B) SIGNS:

Pulse	◆ **Rhythm:** regular (fixed block), irregular (variable block). ◆ **Rate:** 150 or 100 or 75 / min. (according to AV block). ◆ **Response to carotid massage:** (↑ AV block from 2:1→3:1 → 4:1) • Stepwise slowing: of the rate (150 → 100 → 75/ min). • Stepwise acceleration: to the original rate on stopping massage.
Neck veins	◆ Multiple "a" waves: double or triple or quadriple the rate of pulse.
Auscultation	◆ Accentuated S1.

ECG

QRS	P wave
◆ **Rhythm:** regular (fixed block), irregular (variable block). ◆ **Rate:** 150 or 100 or 75 / min. ◆ **Duration :** normal.	◆ Replaced by: **multiple "Flutter waves"** at a rate of 240 - 340 / min. ◆ **Typically:** "Saw - tooth appearance".



TREATMENT:

A- DURING THE ATTACK:

If the patient is hemodynamically stable:	
Vagal stimulation	Carotid sinus massage.
Slow the ventricular rate:	◆ Adenosine: 6 mg IV. ◆ B-blocker (propranolol): 5 mg IV. ◆ CCB (verapamil): 5 mg IV. ◆ Digitalis: 1 mg IV.
Restore sinus rhythm (Cardioversion):	**(after 1 the ventricular rate)** ◆ **Chemical cardioversion:** Class I A, IC or class III drugs. ◆ **Electrical cardioversion (DC):** if chemical cardioversion fails.
If the patient is hemodynamically unstable:	
DC cardioversion	
Overdrive pacing	◆ The atria are paced at a faster rate than the tachycardia rate. ◆ Sudden cessation of pacing is usually followed by: " restoration of sinus rhythm".

B-PREVENTION OF A FUTURE ATTACK:

Maintenance therapy	◆ **DRUGS: (Oral),** Class IA, IC or class III anti – arrhythmic drugs.
Intervention	◆ **Ablation of focus:** Catheter (Radiofrequency energy) or surgery.
TTT of the cause	

VENTRICULAR TACHYCARDIA

DEFINITION:

- It is an arrhythmia originating from the VENTRICLE that **presents with:**
  - Three or more successive ventricular premature beats.
  - Rapid Regular tachycardia at a rate of: 120-250/min.
- **Since there is no retrograde conduction in the AVN, there will be AV dissociation:**
  - The ventricles will be controlled by the vent. focus: (VR 120 - 250 / min).
  - The atria will be controlled by the SAN: (AR: 60 - 100 / min).

TYPES:

a. **Sustained:** persists more than 30 sec. or causes hemodynamic instability

b. **Non-sustained:** persists less than 30 sec. with no hemodynamic instability.

ETIOLOGY:

- It is rarely occurs in a normal heart, & therefore its presence almost always denotes an organic heart disease.

Pathological	◆ CAD (especially AMI). ◆ Cardiomyopathy (and myocarditis). ◆ Congenital heart disease. ◆ RHD. ◆ Hypertension. ◆ Hyperthyroidism. ◆ **Pre - excitation sundromes**: e.g. WPW syndrome.
Pharmachological	◆ **Digitalis.** ◆ Sympathomimetic drugs. ◆ Some anti-arrhythmic drugs e.g., class IC.

CLINICAL PICTURE:

A) SYMPTOMS:

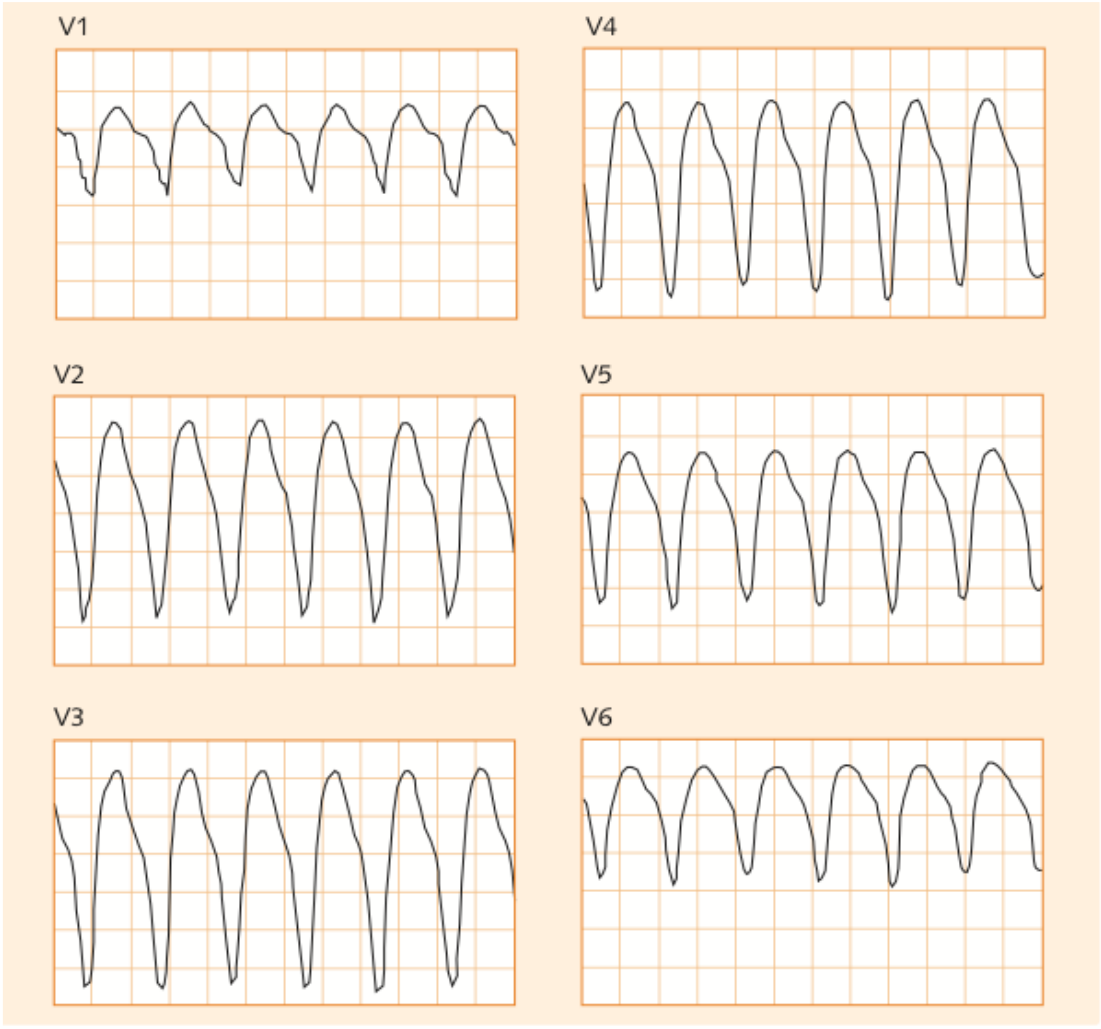
1. **Palpitation:** Rapid, Regular, Sudden onset, Sudden offset.
2. **Symptoms of** low cardiac output.
3. **Precipitation of** angina & HF in susceptible patients.
4. **SUDDEN DEATH:** if converted to Ventricular fibrillation (VF).

B) SIGNS:

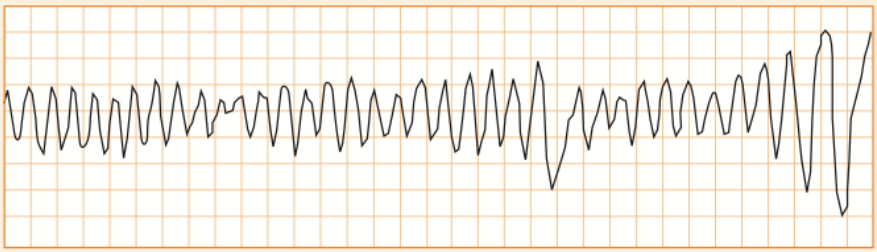
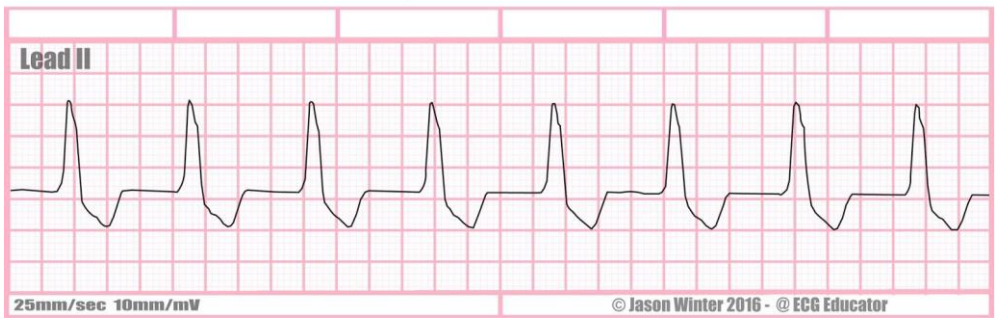
Pulse	◆ **Rhythm:** regular. ◆ **Rate:** 120- 250 / minute. ◆ **Response to carotid massage :** no response .
Neck veins	◆ "a" waves: normal rate (60 - 100 / min) & less than the pulse rate. ◆ **occasional cannon waves.**
Auscultation	◆ Occasional **cannon** sounds. ◆ Variable intensity of S1.

ECG

ORS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 120-250 /min. ◆ **Duration :** Wide	◆ Normal in rate (60 - 100 / minute) & shape. ◆ Comes before, after or is hidden by the QRS (AV dissociation).



SPECIAL TYPES OF VT

Torsade de pointes:	◆ QRS complexes **change continuously & rapidly** & **irregularly** from an upright to an inverted position (twisting of points). ◆ **Causes:** AMI, ↓ K, ↓ Ca. ◆ It is serious & may cause VF & sudden death. 
Accelerated Idio - Ventricular Rhythm:	◆ An **ectopic ventricular pacemaker** discharges at a rate: 60 - 120 / min, and controls the ventricles only resulting in a slow VT. ◆ **Causes:** AMI, post-coronary thrombolysis (reperfusion arrhythmias) ◆ It is **transient** & rarely causes hemodynamic disturbances. 

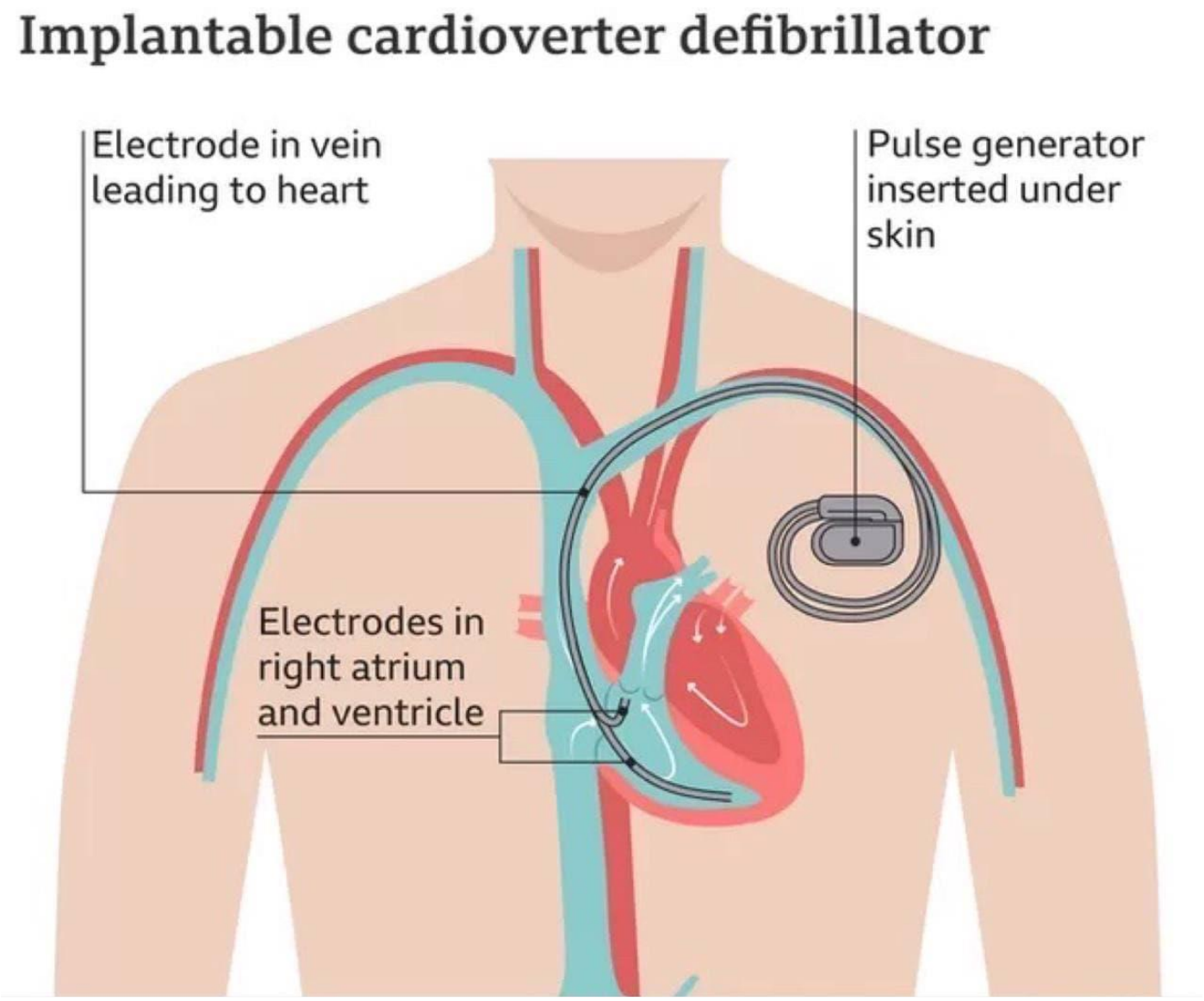
TREATMENT:

A- DURING THE ATTACK:

If the patient is hemodynamically stable:	
Lidocaine	◆ initial bolus of 2 mg / kg IV, followed by maintenance infusion of 1- 4 mg / min. **(first drug of choice).**
Procainamide or Amiodarone	◆ IV (second drug of choice).
Bretylium	◆ IV (in resistant cases).
If the patient is hemodynamically unstable:	
DC cardioversion immediately, followed by IV lidocaine.	

B-PREVENTION OF A FUTURE ATTACK:

Maintenance therapy	◆ **DRUGS:** (Oral), Class I or class III anti - arrhythmic drugs.
Intervention	◆ **Ablation of focus:** Catheter (Radiofrequency energy) or surgery. ◆ **Implantable Cardioverter Defibrillator (ICD):** "Anti - tachycardia pacing"
TTT of the cause	



ATRIAL FIBRILLATION

DEFINITION:

- A form of tachycardia in which the atria discharge at a rate: 400 - 600 / min
- An irregular block occurs in the AVN allowing only some impulses to pass to the ventricles in an IRREG ULAR manner.
- **Therefore, the ventricular beats will be:**

Rhythm	Rate	Force
markedly irregular	100-160/min.	Variable

INCIDENCE:

AF is the most common SUSTAINED ARRHYTHMIA in Adults

ETIOLOGY:

- The exact causes of atrial fibrillation are unknown, but several risk factors have been identified.

CVS risk factors	◆ Advanced age ◆ Hypertension ◆ Diabetes mellitus ◆ Smoking ◆ Obesity ◆ Sleep apnea
Intrinsic cardiac disorders	◆ Coronary artery disease ◆ Valvular heart disease (especially mitral valve disease) ◆ Congestive heart failure (CHF) ◆ Preexcitation tachycardia. eg., Wolff-Parkinson-White (WPW) syndrome ◆ Sick sinus syndrome (tachycardia-bradycardia syndrome) ◆ Cardiomyopathies ◆ Pericarditis ◆ Congenital channelopathies
Noncardiac disorders	◆ Pulmonary disease: COPD, pulmonary embolism, pneumonia ◆ Hyperthyroidism ◆ Catecholamine release and/or increased sympathetic activity ◆ Stress: sepsis, hypovolemia, post-surgical state (especially following cardiac surgery), hypothermia ◆ Pheochromocytoma ◆ Cocaine, amphetamines ◆ Electrolyte imbalances (hypomagnesemia, hypokalemia) ◆ Drugs: eg, adenosine, digoxin

	◆ **Holiday heart syndrome:** irregular heartbeat classically triggered by excessive alcohol consumption but also sometimes by stress, dehydration or lack of sleep ◆ Chronic kidney disease

REVERSIBLE CAUSES OF ATRIAL FIBRILLATION :	
◆ Hyperthyroidism, thyrotoxicosis	◆ Akohol use
◆ Electrolyte imbalances	◆ Excess caffeine
◆ Cardiothoracic surgery	◆ Fever of any cause
◆ Myocarditis	◆ Recreational or pharmacological drug use
◆ Pericarditis	
◆ Myocardial infarction	◆ Pulmonary embolism

Approx. 15% of individuals who develop Afib have none of the above mentioned risk factors (idiopathic/lone Afib).

Remember **PARASITE** to memorize the major risk factors for acute Afib: **P** – **P**ulmonary disease; **A** – **A**nemia; **R** – **R**heumatic heart disease; **A** – **A**trial myxoma; **S** – **S**epsis; **I** – **I**schemia; **T** – **T**hyroid disease; **E** – **E**thanol.

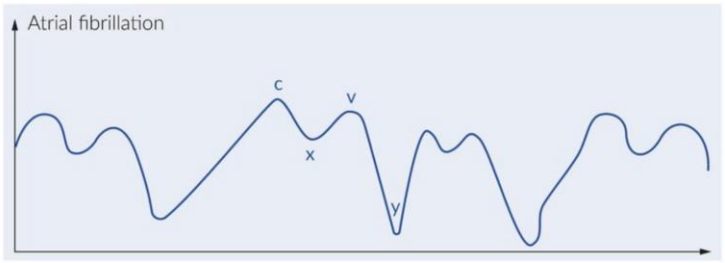
CLINICAL PICTURE:

A) SYMPTOMS:

1. **Palpitation:** Rapid, IRRegular, Sudden onset, Sudden offset.
2. **Symptoms of** low cardiac output.
3. **Precipitation of** angina & HF in susceptible patients.
4. **COMPLICATIONS:** atrial thrombosis & embolization.

B) SIGNS:

Pulse	◆ **Rhythm:** • Marked irregularity (in rhythm & volume). • Pulse deficit: > 10 beats / minute. ◆ **Rate:** 100 - 160 / min. ◆ **Response to carotid massage :** Gradual slowing: due to ↓ AV conduction. ◆ **Response to exercise:** ↑ irregularity: due to ↑ AV conduction.
Neck veins	◆ "a" waves: absent. ◆ Systolic expansion.
Auscultation	◆ Variable intensity of S1.

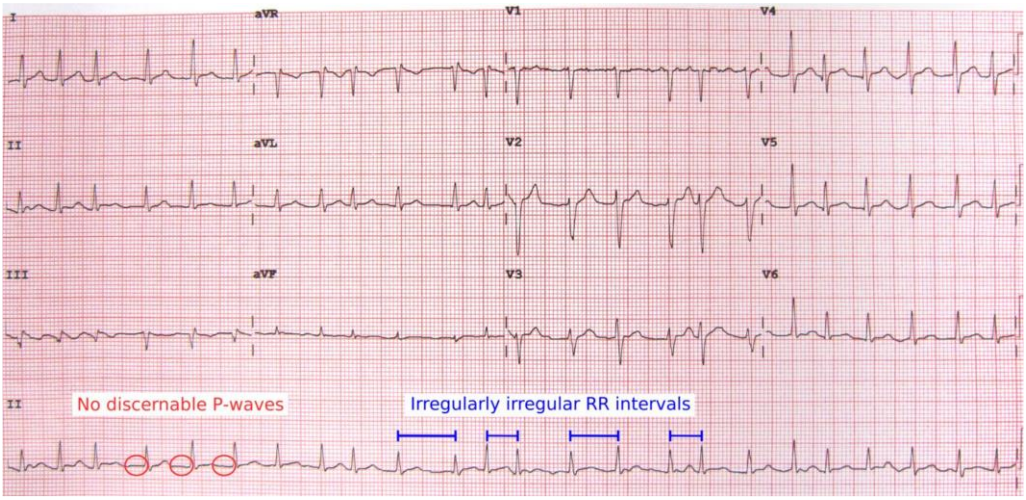


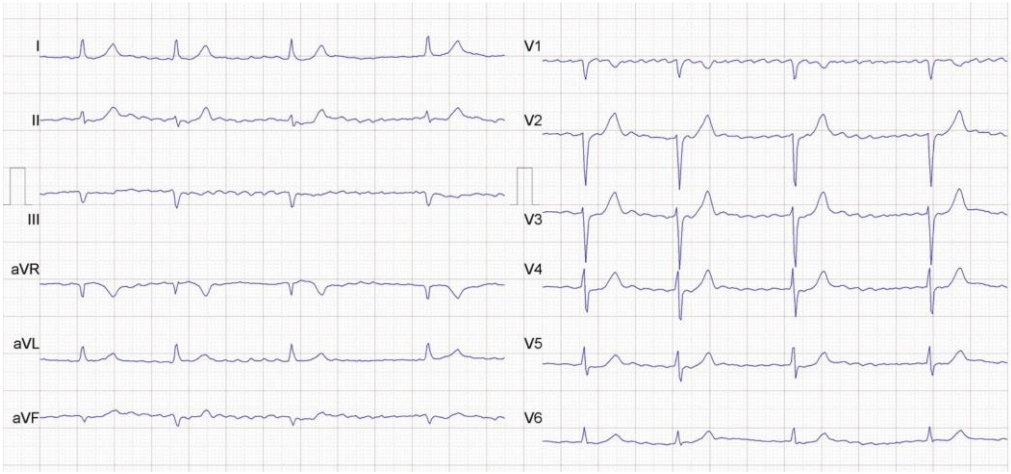
ECG

QRS	P wave
◆ **Rhythm:** irregular. ◆ **Rate:** 100-160 /min. ◆ **Duration :** normal	◆ **"Absent p"** Replaced by: fibrillation waves (irregular vibrations).

CAUSES OF SLOW AF

1. Drugs: Digitalis or B-blocker.
2. Lone AF.
3. Associated heart block.





SLOW AF

CLASSIFICATION

Terminology		Definition
Hemodynamic stability	Unstable Afib	✦ Afib manifesting with signs of hemodynamic instability (e.g., chest pain, altered mental status, acute pulmonary edema, hypotension, or cardiogenic shock)
	Stable Afib	✦ Afib without signs of hemodynamic instability
Ventricular rate	Afib with rapid ventricular response	✦ Afib with a ventricular rate > 100–110/minute (tachycardic Afib)
	Afib with slow ventricular response	✦ Afib with a ventricular rate < 60/minute (bradycardic Afib or slow Afib)
Onset and duration	New onset Afib	✦ New presentation or diagnosis of Afib, regardless of duration
	Paroxysmal Afib	✦ Afib that resolves within 7 days of onset either following treatment or spontaneously; the frequency of recurring episodes may vary.
	Persistent Afib	✦ Continuous Afib for > 7 days
	Long-standing persistent Afib	✦ Continuous Afib for > 1 year
	Permanent Afib	✦ Persistent Afib in which therapeutic attempts are no longer made to convert to or maintain sinus rhythm unless the patient and the treating physician agree to do so
Method of detection	Clinical Afib	✦ Episode of Afib lasting ≥ 30 seconds that is documented on a surface ECG ✦ May be symptomatic or asymptomatic
	Subclinical Afib	✦ asymptomatic Afib not previously detected on a surface ECG that is discovered on implanted cardiac devices and confirmed on intracardiac electrograms
Mitral valve involvement	Valvular Afib	✦ Afib in patients with moderate to severe mitral valve stenosis or an artificial (mechanical) heart valve
	Non-valvular Afib	✦ Afib in patients without moderate to severe mitral valve stenosis or a mechanical heart valve

TREATMENT

A) DURING THE ATTACK:

A. If the patient is hemodynamically stable:	
1. Restore the sinus rhythm: "RHYTHM CONTROL"	
Methods	◆ Pharmacologic cardioversion: Class IA, IC or III drugs. ◆ Electrical cardioversion (DC): If pharmacologic CV fails. ◆ Ablation [Pulmonary VeinIsolation (PVI)]: If pharmacologic CV fails.
Precautions	◆ Oral Anti Coagulation (OAC) should be given: 4 w before & after cardioversion. ◆ Digitalis should be stopped: before electrical cardioversion.
2. Slow the ventricular rate: "RATE CONTROL"	
Drugs	◆ B-blocker (propranolol), CCB (Non-DHP), Digitalis.

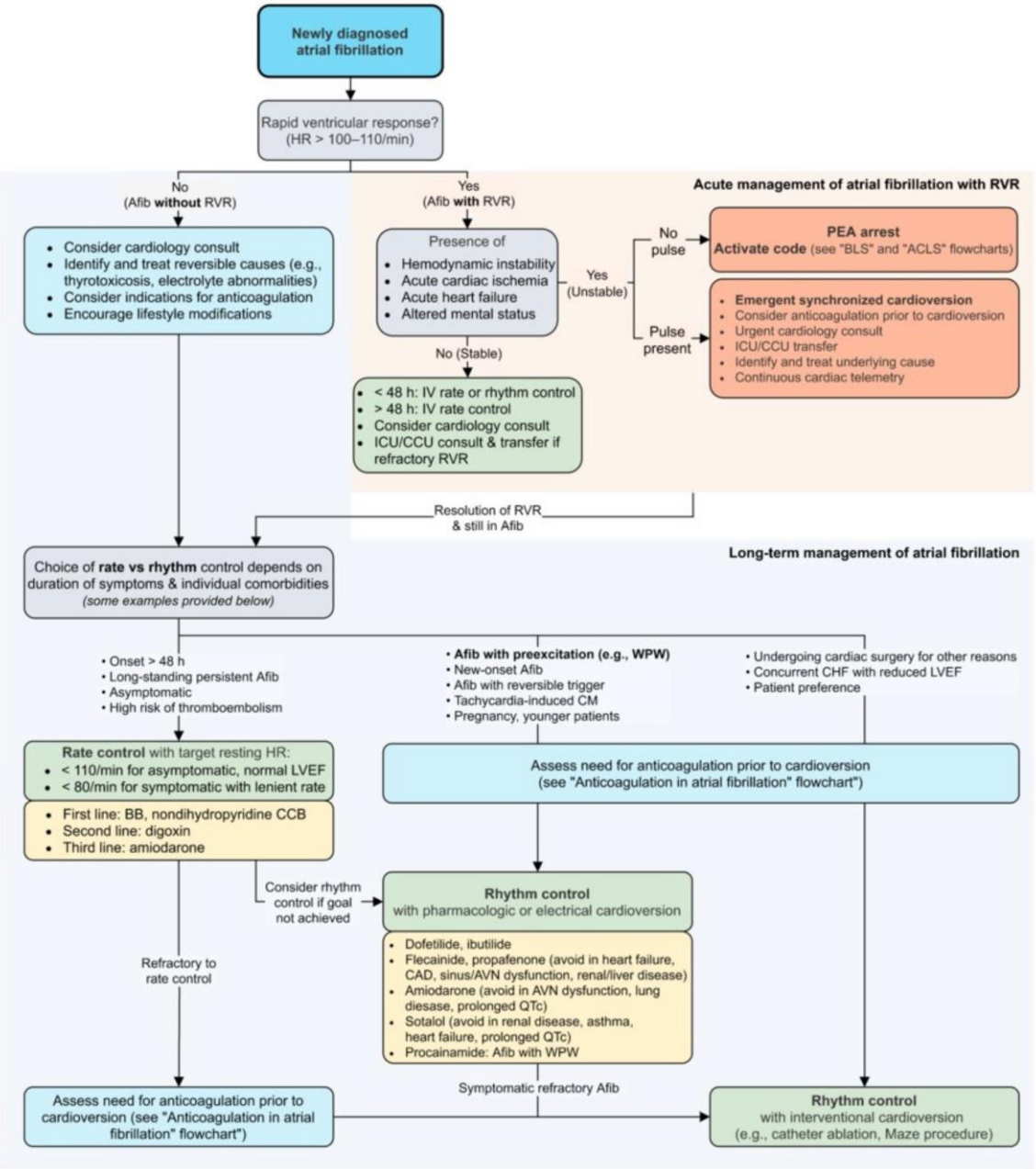
Favours Rate Control	Favours Rhythm Control
Persistent AF	First diagnosed AF or Paroxysmal AF
Less symptomatic	More symptomatic
>65 years of age	<65 years of age
Hypertension	No Hypertension
No History of HF	HF clearly exacerbated by AF
Previous failure of AAD	No Previous failure of AAD (Anti-Arrhythmic Drugs)
Marked ↑ LAVI (> 48 ml/m²)	Normal or mild ↑ LAVI (34 - 42 ml/m²)
Rate control difficult to achieve (failed rate control)	AF precipitated by a temporary event (acute reversible illness), e.g. Hyperthyroidism
Patient's choice	Patient's choice

2012 Canadian Cardiovascular Society: Atrial Fibrillation Guidelines Update
2020 ESC Guidelines for the diagnosis & management of AF (LAVI = Left Atrial Volume Index)

B. If the patient is hemodynamically Unstable:	
DC cardioversion	
Overdrive pacing	◆ The atria are paced at a faster rate than the tachycardia rate. ◆ Sudden cessation of pacing is usually followed by: " restoration of sinus rhythm".

B-PREVENTION OF A FUTURE ATTACK:

Maintenance therapy	◆ **DRUGS: (Oral),** Class IA, IC or class III anti – arrhythmic drugs.
Intervention	◆ **Ablation of focus:** Catheter (Radiofrequency energy) or surgery.
TTT of the cause	



THE iNTEGRATED ABC PATHWAY

A : Anticoagulation Avoid stroke

- **CHA2DS2-VASc score :** estimates risk of **stroke & thromboembolism** in patients e AF

Initials	Risk Factor	Points
C	Congestive HF	1
H	Hypertension	1
A2	Age ≥ 75 y	2
D	Diabetes	1
S2	Stroke (previous stroke , TIA , or Thromboembolism)	2
V	Vascular Disease (previous MI , PAD , or Aortic Plaque)	1
A	Age ≥65 y	1
SC	Sex Category (Female)	1
	MAXiMUM SCORE	9

Total Score	Recommendations
(0) ♂ (1) ♀	**No** Oral AntiCoagulation
(1) ♂ (2) ♀	OAC **should be considered**
(2-9) ♂ (3-9) ♀	OAC **is recommended** (esp **NOACs as 1st** line OAC)

- **HAS-BLED score :** estimates 1-year risk of major bleeding in patients with AF on OAC
  - For high bleeding risk (HAS-BLED : 3-9) :
  - Such high bleeding score shouldn't be used to withhold OAC

Initials	Risk Factor	Points
H	Hypertension (Uncontrolled: SBP>160 mmHg)	1
A	Abnormal Renal or/and Liver function	1 or 2
S	Stroke (Previous Ischemic or Hemorrhagic)	1
B	Bleeding tendency or predisposition	1
L	Labile INR (Unstable/high INR or TTR < 60% in patient on VKA)	1
E	Elderly (Age ≥ 65 y)	1
D	Drugs (Concomitant Antiplatelets or/and excess Alcohol)	1 or 2
	MAXiMUM SCORE	9

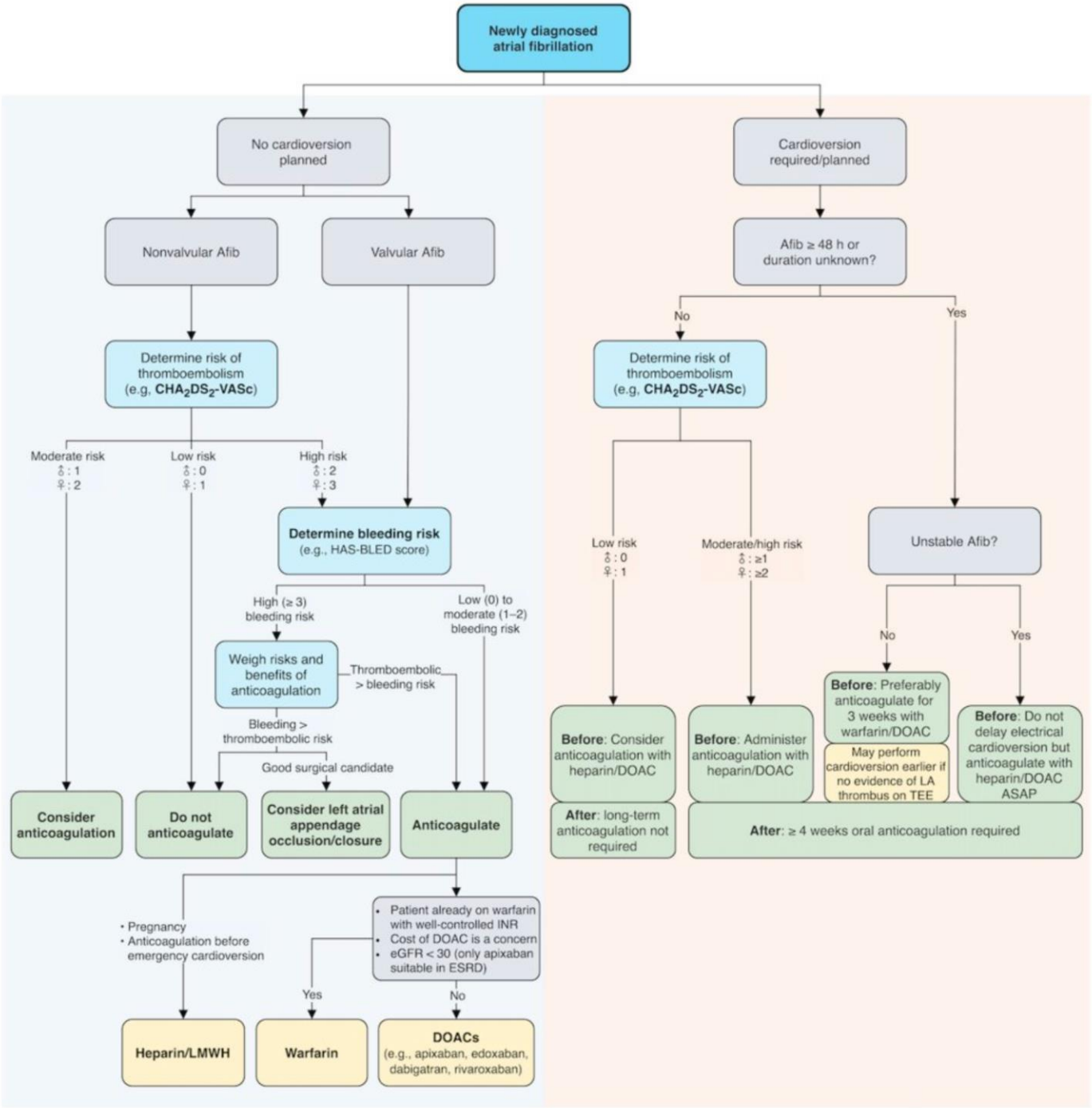
Total Score	Interpretation	Take care
Zero – 2	Low bleeding risk	• 1 point for each of abnormal renal or liver function • 1 point of which platelets or Alcohol • TTR : Time in Therapeutic Range • VKA : Vitamin K Antagonist
3 – 9	High bleeding risk	

B : **B**etter symptom control (Rate control vs rhythm control → mentioned before)

C : **C**oncomitant disease : detection & management (e.g HTN , HF , CAD , DM)

NOVEL ORAL ANTI-COAGULANTS (NOACS)

Drug	Action	Dose	Clearance
Apixaban	Direct factor X a inhibitor	5 mg twice daily	Hepatic
Rivaroxaban		20 mg once daily	Renal
Edoxaban		60 mg once daily	Hepatic
Dabigatran	Direct Factor II a (thrombin) inhibitor	150 mg twice daily	Renal


Newly diagnosed atrial fibrillation
No cardioversion planned
Nonvalvular Afib
Valvular Afib
Determine risk of thromboembolism (e.g, CHA2DS2-VASc)
Moderate risk ♂:1 ♀:2
Low risk ♂:0 ♀:1
High risk ♂:2 ♀:3
Determine bleeding risk (e.g., HAS-BLED score)
High (≥ 3) bleeding risk
Low (0) to moderate (1–2) bleeding risk
Weigh risks and benefits of anticoagulation
Thromboembolic > bleeding risk
Bleeding > thromboembolic risk
Good surgical candidate
Consider anticoagulation
Do not anticoagulate
Consider left atrial appendage occlusion/closure
Anticoagulate
• Pregnancy
• Anticoagulation before emergency cardioversion
• Patient already on warfarin with well-controlled INR
• Cost of DOAC is a concern
• eGFR < 30 (only apixaban suitable in ESRD)
Yes
No
Heparin/LMWH
Warfarin
DOACs (e.g., apixaban, edoxaban, dabigatran, rivaroxaban)
Cardioversion required/planned
Afib ≥ 48 h or duration unknown?
No
Yes
Determine risk of thromboembolism (e.g, CHA2DS2-VASc)
Low risk ♂:0 ♀:1
Moderate/high risk ♂:≥1 ♀:≥2
Unstable Afib?
No
Yes
Before: Consider anticoagulation with heparin/DOAC
Before: Administer anticoagulation with heparin/DOAC
Before: Preferably anticoagulate for 3 weeks with warfarin/DOAC
May perform cardioversion earlier if no evidence of LA thrombus on TEE
Before: Do not delay electrical cardioversion but anticoagulate with heparin/DOAC ASAP
After: long-term anticoagulation not required
After: ≥ 4 weeks oral anticoagulation required

PREMATURE BEATS (EXTRASYSTOLES)

DEFINITION

- They are **ectopic** cardiac impulses occurring before the expected sinus impulse causing premature beats.
- When the normal sinus impulse arises, the heart will not respond because it will be in the: **"Refractory period".**
- **These ectopic cardiac impulses may arise:**
  1. Supraventricular (from the Atria or AVN) or,
  2. Ventricular (from the Ventricles).

ETIOLOGY

Physiological	**may occur in a normal heart (no organic heart disease).** ◆ Emotions. ◆ Excessive: smoking, coffee, tea
Pathological	◆ CAD (especially AMI). ◆ Cardiomyopathy (and myocarditis). ◆ Congenital heart disease. ◆ RHD. ◆ Hypertension. ◆ Hyperthyroidism. ◆ **Pre - excitation sundromes:** e.g. WPW syndrome.
Pharmachological	◆ **Digitalis.** ◆ Sympathomimetic drugs. ◆ Some anti-arrhythmic drugs e.g., class IC.

CLINICAL PICTURE

A) SYMPTOMS:

1) Asymptomatic.
2) PALPITATION: irregular.

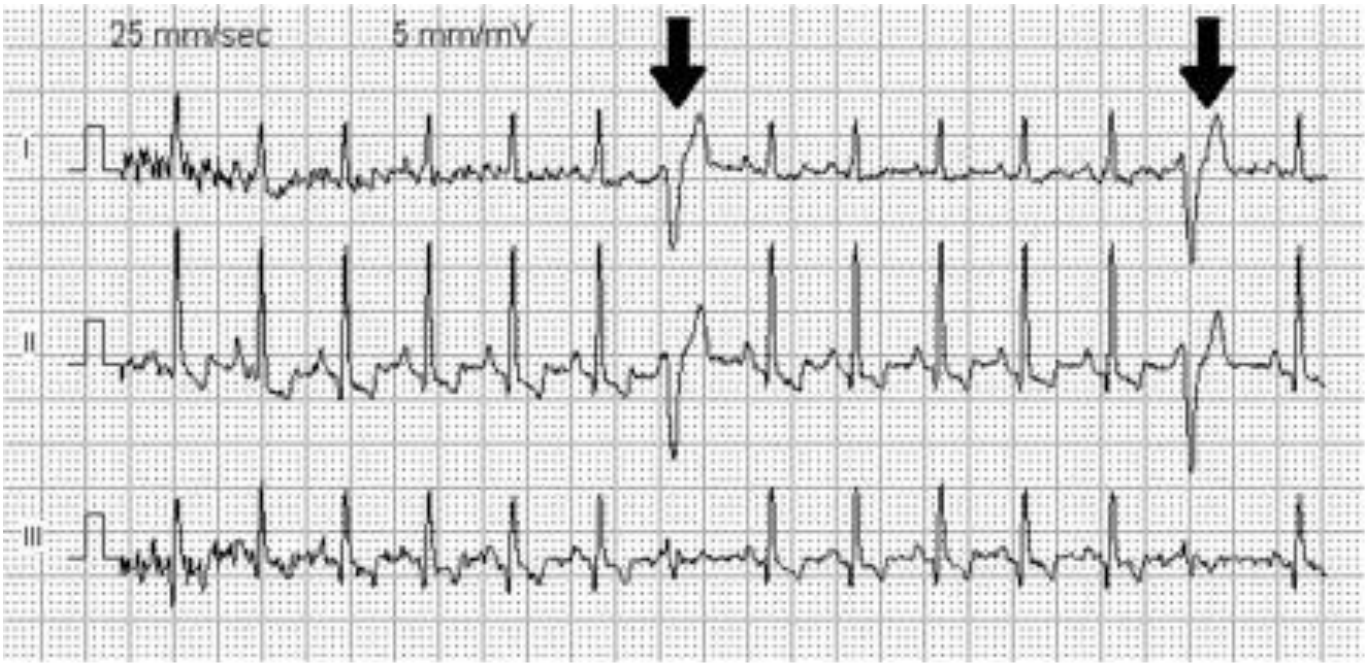
B) SIGNS:

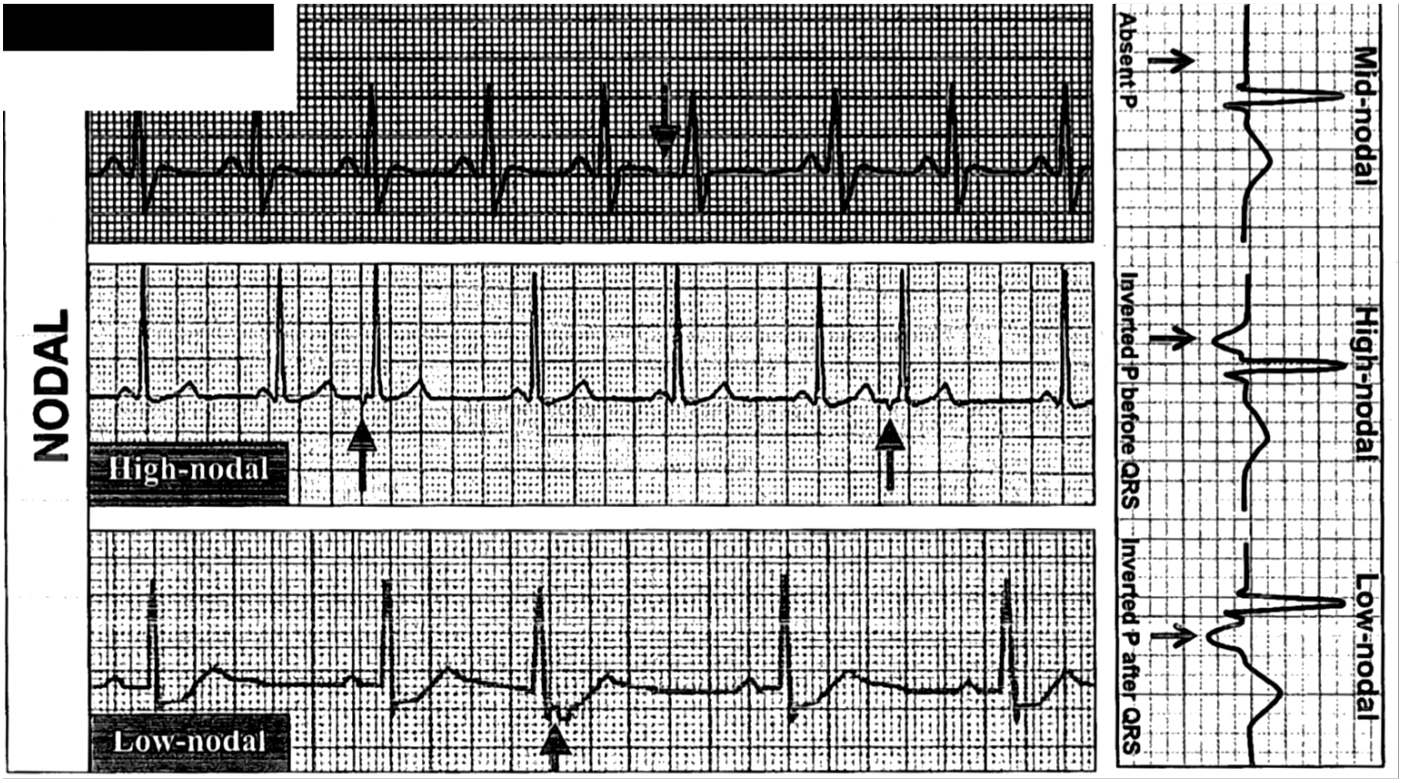
Pulse	◆ **Rhythm:** Occasional irregularity. Pulse deficit: < 10 beats / minute. ◆ **Rate:** VARIABLE according to the sinus rhythm . ◆ **Response to exercise:** "VARIABLE": • ↓ irregularity: due to shortened diastolic period. • ↑ irregularity: due to sympathetic stimulation.
Neck veins	◆ Normal waves with: **Occasional irregularity.**
Auscultation	◆ Normal sounds with: **Occasional irregularity.**

ECG:

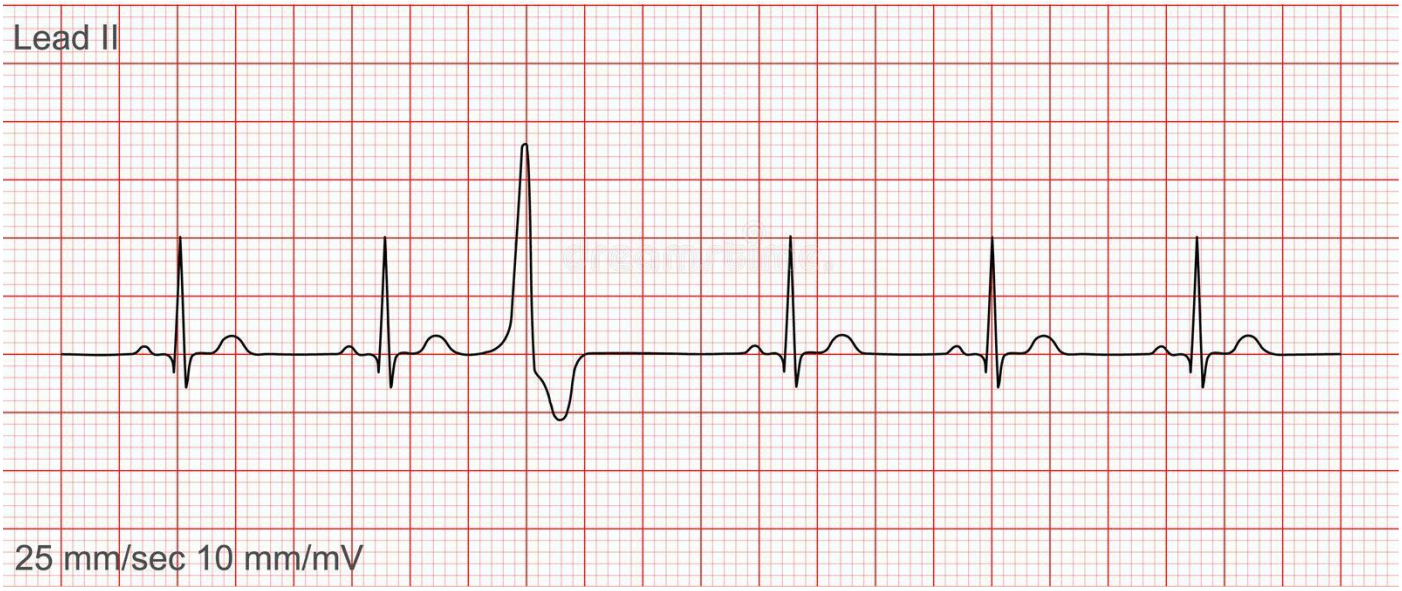
- **RHYTHM:** irregular (Occasional irregularity), but: sinus.
- **THE PREMATURE BEAT COMES EARLY & IS FOLLOWED BY A COMPENSATORY PAUSE:**

Supraventricular (from the Atria or AVN):	
Atrial beats	◆ deformed p - wave followed by ◆ a normal ORS
Nodal beats	◆ absent or inverted p - wave, ◆ a normal ORS
Ventricular (from the Ventricles)	
Ventricular beats	◆ absent p - wave, ◆ wide ORS.





Premature Ventricular Contraction (PVC)



TREATMENT

1) **TTT of the cause.**
2) **Sedatives:** may be needed.
3) **TTT of extrasystoles:**

Supraventricular beats:	◆ They usually need no ttt (asymptomatic). ◆ They may need B-blockers (Symptomatic: causing palpitation).
Ventricular beats:	◆ They usually need no ttt (asymptomatic). ◆ **They may need Anti - arrhythmic drugs if they are:** • Symptomatic: causing palpitation. • Multiple. • Multifocal. • Falling on the preceding T-wave (R on T phenomenon). • Occuring in association with: AMI. ◆ **Anti - arrhythmic drugs are:** • **In emergency conditions:** e.g. AMI, Digitalis toxicity, IV Lidocaine is the drug of choice. • **In stable conditions:** Oral therapy with class I , II , or III drugs

NODAL (JUNCTIONAL) RHYTHM

DEFINITION

- An abnormal heart rhythm, where the AVN initiates electrical activity of the heart.
- The impulses spread up & down to activate the atria & the ventricles simultaneously.

THERE ARE TWO POSSIBILITIES:

Nodal tachycardia:	◆ Abnormal automaticity in the AVN overtakes the normal SAN.
Nodal rhythm:	◆ An escape rhythm in which the AVN becomes the pace – maker of the heart discharging at a rate of 40 - 60 / minute, in cases of: • **Severe bradycardia:** when the SAN discharges at a rate slower than the intrinsic AVN pacemaker. • **Heart block:** conduction problem between the SAN & the AVN.

ETIOLOGY

- It usually occurs in a patient with ORGANIC HEART DISEASE,
- However, It may occur in a normal heart.

Physiological	◆ may occur in a normal heart (↑ ↑ vagal tone during sleep).
Pathological	◆ CAD (especially AMI, inferior). ◆ Cardiomyopathy (and myocarditis). ◆ SICK SINUS SYNDROME.
Pharmacological	◆ DIGITALIS. ◆ B-blockers.

CLINICAL PICTURE

A) SYMPTOMS:

1) Asymptomatic.
2) PALPITATION: regular
3) Symptoms of : Low cardiac output.
4) Precipitation of: angina & HF in susceptible patients.

B) SIGNS:

Pulse	◆ **Rhythm:** regular. ◆ **Rate:** 40 - 60 / minute. ◆ **Response to exercise:** • Reversion to sinus rhythm may occur in some cases.
Neck veins	◆ Regular cannon waves with the same rate of pulse.
Auscultation	◆ Regular cannon sounds.

ECG

QRS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 40-60 /min. ◆ **Duration:** normal	Absent or inverted

TREATMENT

1. **TTT of the cause,** e.g. permanent pacemaker for sick sinus syndrome.
2. **Atropine:** in symptomatic cases.

HEART BLOCK

DEFINITION

- A disease in the Electrical system of the heart.
- Impairment of impulse condution at any of the following sites:
  1) **SAN (Sinoatrial block)** between the SAN & the atria.
  2) **AVN (AV block)** between the atria & the ventricles.
  3) **Bundle branch (BBB)** along the bundle branches.

AV BLOCK

ETIOLOGY

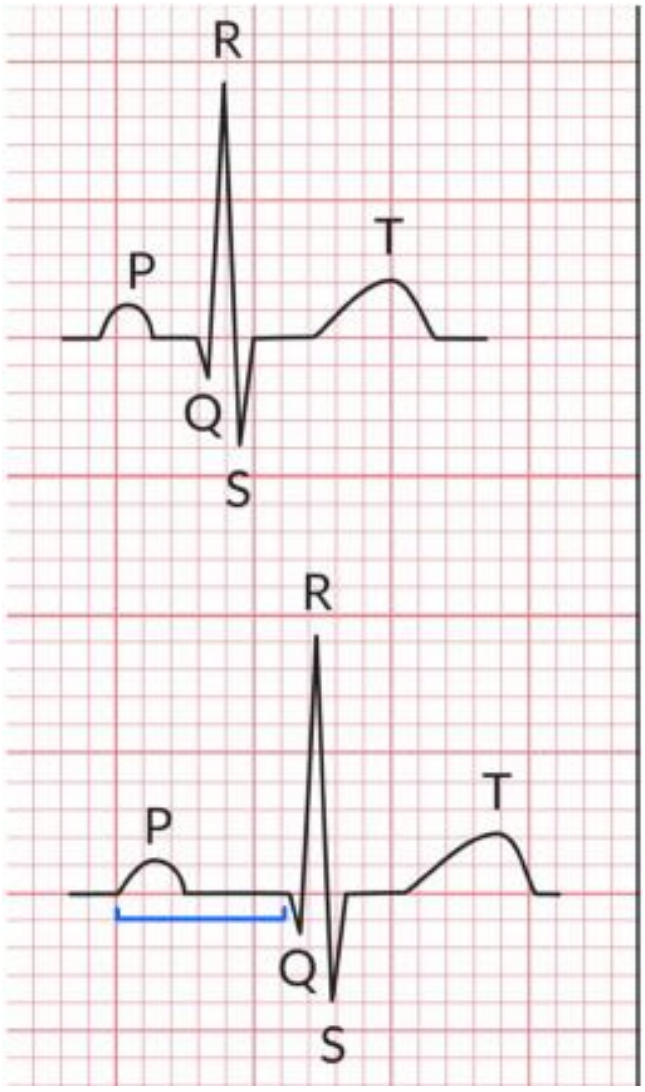
Pathological	◆ CAD (especially AMI, inferior). ◆ Cardiomyopathy (and myocarditis). ◆ Congenital heart disease. ◆ Calcific aortic stenosis. ◆ Conduction system fibrosis. ◆ HYPERKALEMIA.
Pharmacological	◆ DIGITALIS. ◆ B-blockers. ◆ CCBs.

TYPES = GRADES = DEGREES

First degree	Second degree	Third degree
DELAYED conduction of **ALL impulses.**	**NO** conduction of **Some impulses.**	**NO** conduction of **ALL impulses.**

First degree

DEFINITION	◆ Delayed conduction of ALL impulses from the atria to the ventricles. ◆ ALL P waves are followed by a QRS complex.
SYMPTOMS	◆ Asymptomatic. ◆ Symptoms of: the cause
SIGNS	◆ No clinical signs.
ECG	◆ PR: prolonged > 0.2 sec. (> 5 small squares), fixed.
TREATMENT	◆ TTT of: the cause.



SECOND DEGREE (Incomplete HB)

DEFINITION

- No conduction of Some impulses from the atria to the ventricles.
- Some P waves are not followed by a QRS complex.

TYPES:

Mobitz Type I:	**(Wenckebach phenomenon) (Block is in AVN)** • Progressive prolongation of the AV conduction time until conduction fails completely & an atrial impulse is blocked.

Mobitz Type II:	**(Block is below AVN: Infra-Hisian)**
	• Every 2, 3 or 4 atrial impulses, the AVN transmits only one impulse to the ventricles, (so the block will be: 2:1 or 3:1 , or 4:1).
	• The block may be:
	  ◦ Fixed (e.g. 2:1),
	  ◦ Variable (e.g. changing from: 2:1, to 3:1 to 4:1 ..etc....).

CLINICAL PICTURE:

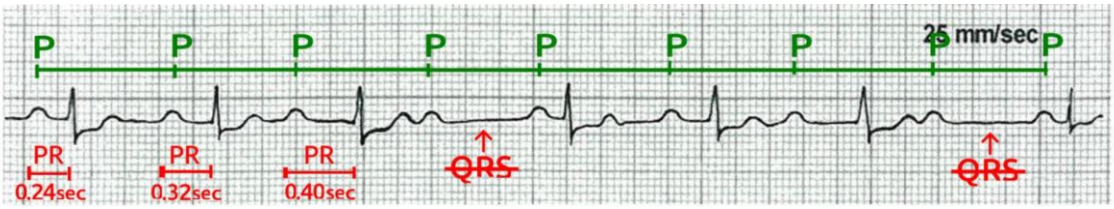
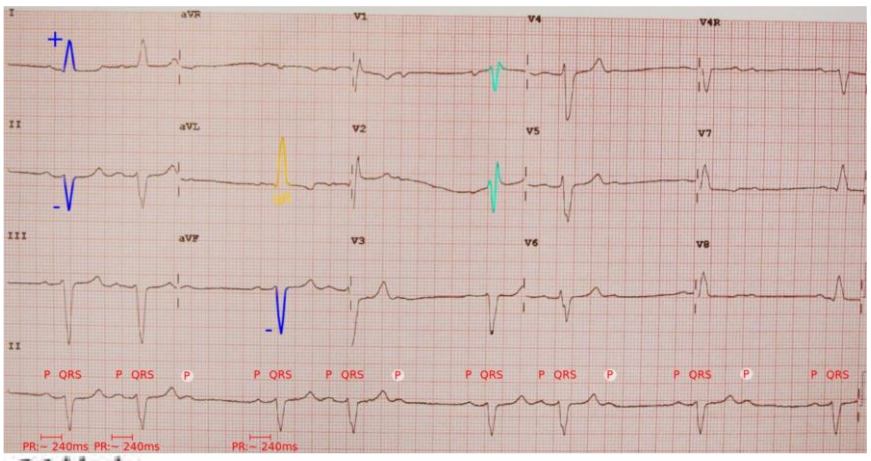
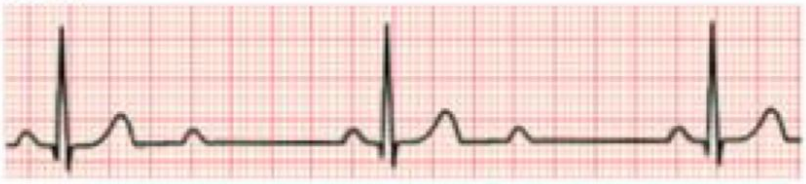
A) SYMPTOMS

1) Asymptomatic.
2) Symptoms of: the cause.
3) PLUS In Mobitz Type IL
   - PALPITATION: regular or irregular.
   - Symptoms of: low cardiac output.
   - Precipitation of: angina & HF in susceptible patients.

B) SIGNS:

Mobitz Type I	• Irregular pulse, Weak S1.
Mobitz Type II	
Pulse	• Rhythm: regular (fixed block), irregular (variable block). • Rate: slow, e.g. 30-50 / min. (according to AY block).
Neck veins	• Multiple "a" waves: double or triple or quadriple the rate of pulse.
Auscultation	• Weak S1.

ECG

Mobitz Type I	
PR	◆ **Progressive prolongation** until a QRS is dropped, i.e. until a p wave is not followed by a QRS complex. Then, the PR interval returns to its normal duration and the sequence is repeated. 
Mobitz Type II	
QRS	◆ **Rhythm:** regular (fixed block), irregular (variable block) ◆ **Rate:** bit slow, e.g. 30-50 / min. (according to AV block) ◆ **Duration**: normal.
P wave	◆ Normal rate. ◆ 2, 3 or 4 P waves occur before each QRS (according to AV block) ◆ **The block may be:** • Fixed (e.g. 2:1), • Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc)  2:1 block 

TREATMENT

1) TTT of the cause.
2) Atropine 1 mg IV.
3) PLUS In Mobitz Type II, Artificial pacemaker.

Third DEGREE (Complete HB)

DEFINITION

- No conduction of ALL impulses from the atria to the ventricles.
- How do the ventricles work ??
- They will be controlled by an: Idioventricular pacemaker.
- No relation between P waves and QRS complexes (AV dissociation).

CLINICAL PICTURE:

A) SYMPTOMS:

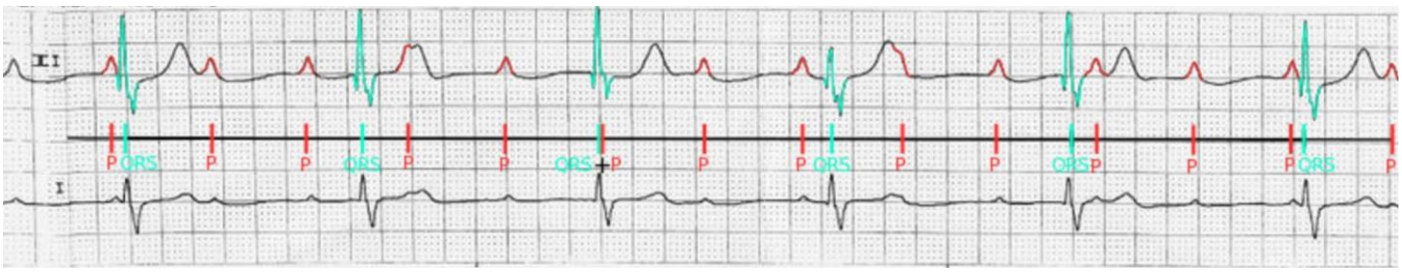
1) Symptoms of the cause.
2) **PALPITATION:** regular.
3) Symptoms of low cardiac output.
4) Precipitation of angina & HF in susceptible patients.
5) **ADAMS – STOKES ATTACKS:**
   - They occur during transmission from one idioventricular focus to another.
   - There is syncope, cyanosis, with absent pulse, BP & heart sounds.
   - If prolonged: convulsions, coma & death may occur.

B) SIGNS:

Pulse	◆ Rhythm: regular. ◆ Rate: slow, 30-40 /min.
Neck veins	◆ "a" waves normal rate (60 - 100 / min) & more than the pulse rate. ◆ Occasional cannon waves.
Auscultation	◆ Occasional cannon sounds. ◆ Variable intensity of S1.

ECG

QRS	P wave
◆ **Rhythm:** regular. ◆ **Rate:** 30-40 /min. ◆ **Duration:** wide.	◆ Normal in rate (60 - 100 / minute) & shape. ◆ Comes before, after or is hidden by the QRS (AV dissociation).



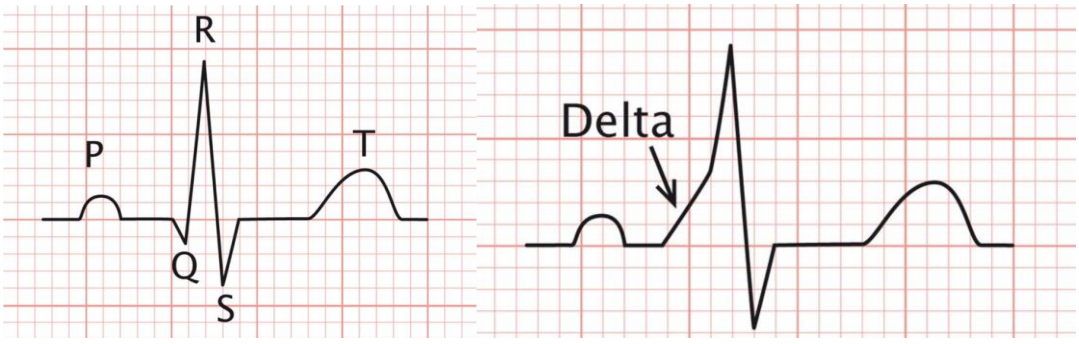
TREATMENT

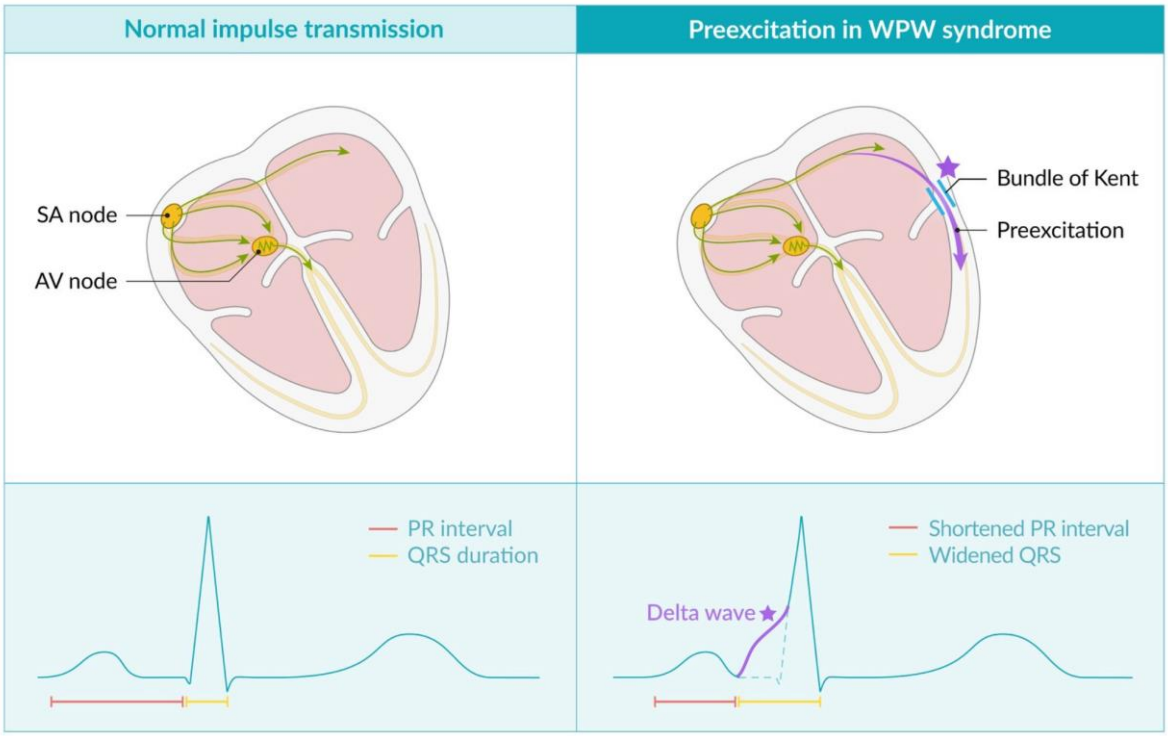
1) TTT of: the cause.
2) Atropine: 1 mg IV.
3) Artificial pacemaker.

SICK SINUS SYNDROME

Definition	◆ This is a clinically symptomatizing SAN dysfunction.
Etiology	◆ Coronary heart disease ◆ Degenerative disease of the SAN.
Clinical Picture	◆ Sinus bradycardia ◆ Sinus arrest , & atrial asystole.
Investigations	◆ Ambulatory (Holter) ECG monitoring.
Treatment	◆ Permanent pacemaker implantation.

PRE-EXCITATION SYNDROMES

Definition	◆ An accessory conduction pathway bypasses the AVN , leading to abnormal early activation of part of the ventricle . ◆ The remaining part of the ventricle will receive the normal impulse from the normal pathway.
Etiology	◆ Congenital.
Clinical Picture	◆ Different arrhythmias: e.g. AF, flutter, or supraventricular tachycardia. ◆ Sudden death.
ECG	◆ Wolff-Parkinson-White syndrome (WPW): "Wide QRS, Short PR interval, Delta wave". 
Treatment	◆ Medical control of the associated tachycardia. ◆ Surgical division of the accessory pathway.



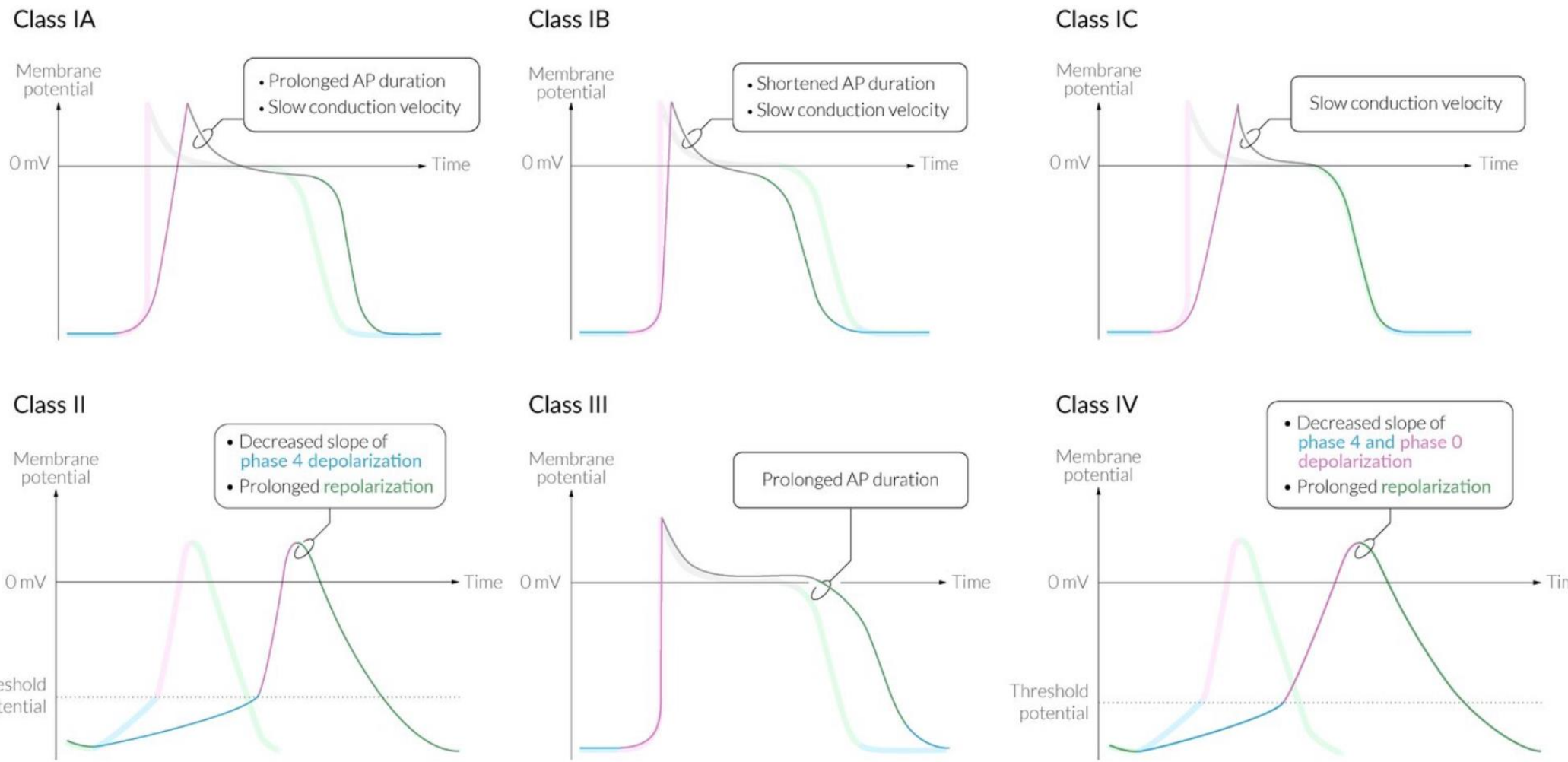
ANTI-ARRHYTHMIC DRUGS

Class	Mode of action	Subclass	Drug	Main uses	Side effects
I	Sodium channel blockers → slowing depolarization phase → depression	IA Moderate phase 0 depression	Ouinidine Disopyramide Procainamide	Broad spectrum	Idiosyncrasy Cinchonism N, V, D Hypotension Anti-cholinergic effects Hypotension Lupus-like synd.
		IB Minimal phase 0 depression	Lidocaine Epanutin	Ventricular arrhythmias	Convulsions Confusion Skin rash BM depression Ataxi
		IC Marked phase 0 depression	Propafenone	Broad spectrum	Pro-arrhythmia

II	B-blockers		Propranolol & Sotalol	ST, SVT Atrial flutter AF Extrasystoles	
III	Potassium channel Blockers slowing the repolarization (prolong the RF)		Amiodarone Bretylium	Broad spectrum Resistant VT	Corneal deposits Thyroid problems Pulmonary fibrosis Hepatotoxicity Hypotension
IV	CCBs		Verapamil	SVT Atrial flutter AF	

DRUGS WITH ANTI-ARRHYTHMIC ACTION:

1. **Adenosine:** it ↓, conductivity in the AV node, so it is indicated in SVT, Atrial flutter.
2. **Digitalis:** it ↓, conductivity in the AV node, so it is indicated in SVT, Atrial flutter, AF.


Class IA
Membrane potential
• Prolonged AP duration
• Slow conduction velocity
0 mV
Time
Class IB
Membrane potential
• Shortened AP duration
• Slow conduction velocity
0 mV
Time
Class IC
Membrane potential
Slow conduction velocity
0 mV
Time
Class II
Membrane potential
• Decreased slope of phase 4 depolarization
• Prolonged repolarization
0 mV
Time
Threshold potential
Class III
Membrane potential
Prolonged AP duration
0 mV
Time
Class IV
Membrane potential
• Decreased slope of phase 4 and phase 0 depolarization
• Prolonged repolarization
0 mV
Threshold potential
Ventricular action potential
Pacemaker cell action potential

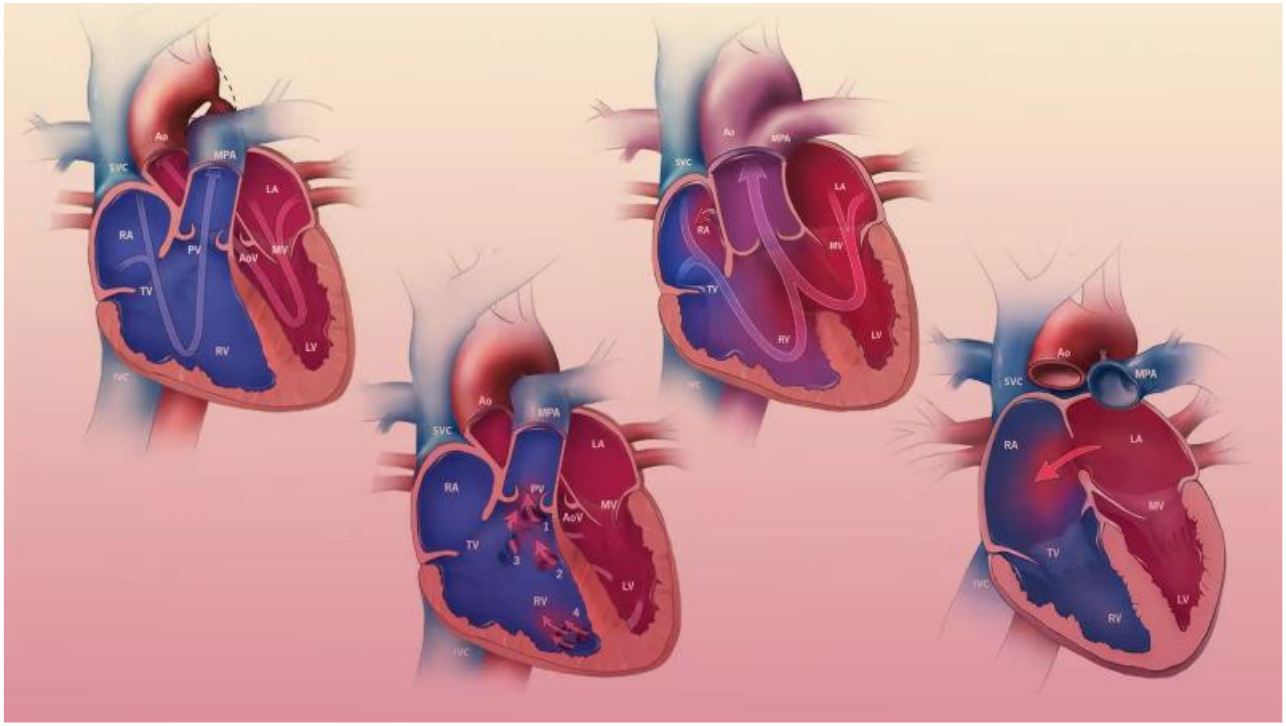
CONGENITAL HEART DISEASES (CHD)

INTRODUCTION :

- It is one of the **commonest congenital defects**, occurring in 0.6–0.8% of newborns.
- Advances in therapy have led to a dramatic improvement in outcome, such that over 85% of infants, even with complex CHD, are expected to reach adolescence and early adulthood.
- As a result of the success of paediatric cardiology and surgery, there are now more adults than children with CHD.

ETIOLOGY :

Environmental factors	✦ Maternal illness (eg, diabetes, rubella, systemic lupus erythematosus) ✦ Maternal intake of teratogenic agents (eg, lithium, isotretinoin, antiseizure drugs) ✦ Maternal age is a known risk factor for certain genetic conditions, especially Down syndrome, that may include cardiac defects.
Numerical chromosomal abnormalities	✦ Trisomy 21 (Down syndrome), trisomy 18, trisomy 13, and monosomy X (Turner syndrome), are strongly associated with congenital heart disease.
Genetic etiology	✦ **No** identifiable genetic etiology is detected in about 72% of patients with congenital heart disease



PATHOPHYSIOLOGY :

- **Congenital heart anomalies are classified as :**
  a. Cyanotic.
  b. Acyanotic (left-to-right shunts or obstructive lesions).

- The physiologic consequences of congenital heart anomalies vary greatly, ranging from heart murmur or discrepancy in pulses in an asymptomatic child to severe cyanosis, heart failure, or circulatory collapse

Cyanotic heart anomalies	✦ Varying amounts of **deoxygenated** venous blood are shunted to the left heart (right-to-left shunt), reducing systemic arterial oxygen saturation. ✦ If there is **> 5 g/dL (> 50 g/L)** of deoxygenated hemoglobin, cyanosis results. Complications of persistent cyanosis include polycythemia, clubbing, thromboembolism (including stroke), bleeding disorders, brain abscess, and hyperuricemia. ✦ Depending on the anomaly, pulmonary blood flow may be **reduced, normal, or increased** (often resulting in heart failure in addition to cyanosis), resulting in **cyanosis** of variable severity. ✦ Heart murmurs are variably audible and are not specific.
Left-to-right shunts	✦ **Oxygenated blood** from the left heart (left atrium or left ventricle) or the aorta shunts to the right heart (right atrium or right ventricle) or the pulmonary artery through an opening or communication between the 2 sides. ✦ **High-pressure shunts** (those at the ventricular or great artery level) become apparent several days to a few weeks after birth; low-pressure shunts (atrial septal defects) become apparent considerably later. ✦ If untreated, elevated pulmonary blood flow and pulmonary artery pressure may lead to pulmonary vascular disease and eventually Eisenmenger syndrome. ✦ **Large left-to-right shunts** (eg, large ventricular septal defect [VSD], patent ductus arteriosus [PDA]) cause excess pulmonary blood flow and left ventricular volume overload, which may lead to signs of heart failure and during infancy often result in failure to thrive. ✦ **A large left-to-right shunt** also leads to lower lung compliance and higher airway resistance. ✦ These factors increase hospitalization in infants with respiratory syncytial virus or other upper or lower respiratory tract infections.

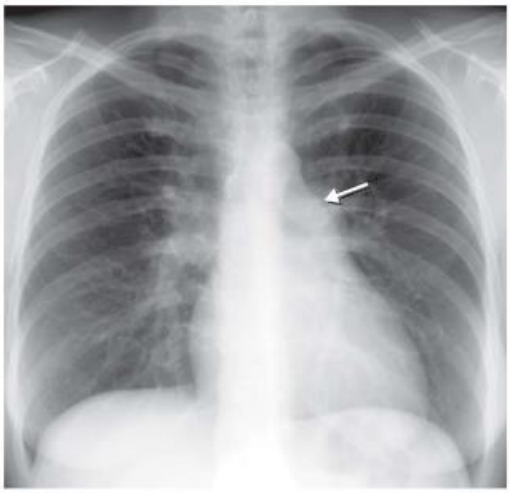
Obstructive lesions	✦ Blood flow is **obstructed** → pressure gradient across obstruction. ✦ The resulting pressure overload **proximal** to the obstruction may cause ventricular hypertrophy and heart failure. ✦ The most obvious manifestation is a **heart murmur**, which results from turbulent flow through the obstructed (**stenotic**) point. ✦ Examples are congenital aortic stenosis, which accounts for 3 to 6% of congenital heart anomalies, and congenital pulmonic stenosis, which accounts for 8 to 12%.

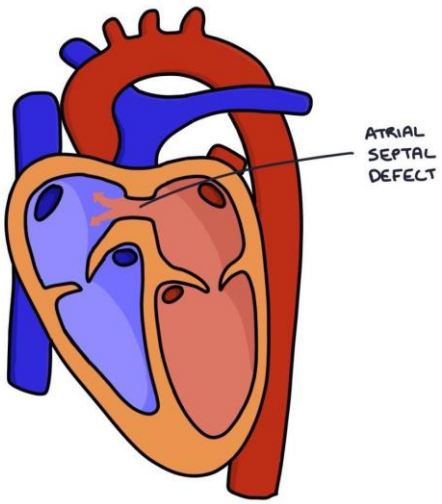
CLASSIFICATION OF CONGENITAL HEART DISEASE IN ADULTS :

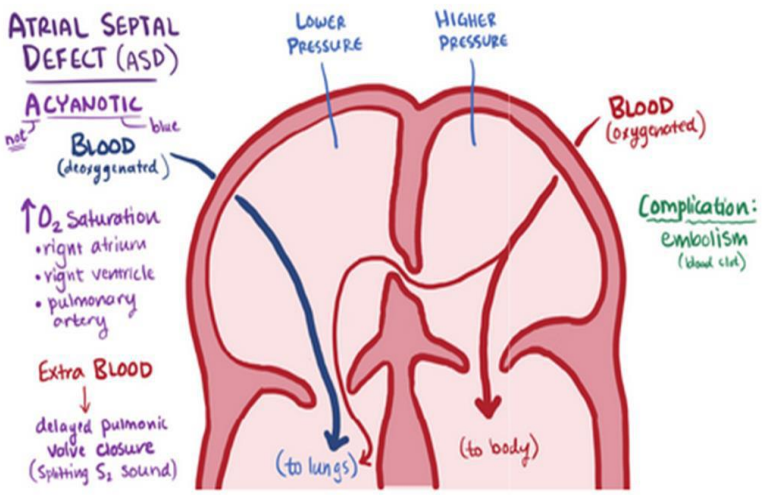
Acyanotic lesions	Cyanotic lesions
✦ Atrial septal defect ✦ Ventricular septal defect ✦ Atrioventricular septal defect ✦ Pulmonary stenosis ✦ Coarctation of the aorta ✦ Patent ductus artreriosus	✦ Transposition of the great arteries ✦ Tetralogy of Fallot ✦ Congenitally corrected transposition of the great arteries

Atrial septal defect (ASD)

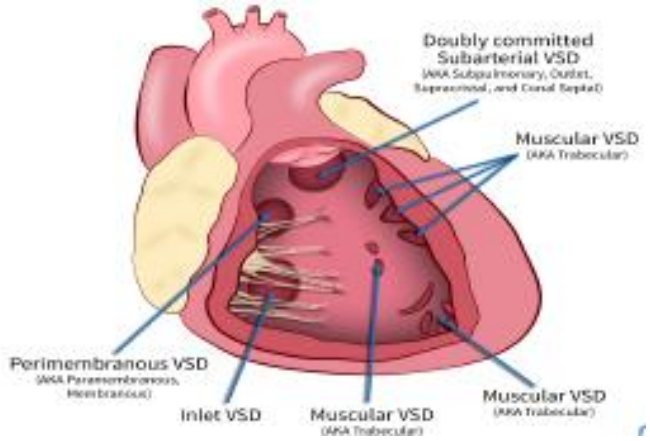
DEF	✦ **Hole** connects the atria. ✦ ASDs are common congenital heart defects in adults
TYPES	✦ **Ostium secundum defects** (high in the septum) are **commonest**. ✦ **Ostium primum defects** (partial atrioventricular canal). ✦ **Sinus venosus defects.**
SYMPTOMS	✦ **Primum ASDS** present **early** ✦ **Secundum ASDS** are often **asymptomatic** until adulthood, as the L-R shunt depends on compliance of the right and left ventricles. The latter decreases with age, so augmenting L-R shunting (hence dyspnea/heart failure, eg at age 40–60). ✦ There may be pulmonary hypertension, cyanosis, arrhythmia, hemoptysis, and chest pain
SIGNS	✦ AF & Increased JVP. ✦ Wide, Fixed Split S2. ✦ Pulmonary ejection Systolic Murmur. Pulmonary Hypertension May Cause Pulmonary or tricuspid regurgitation

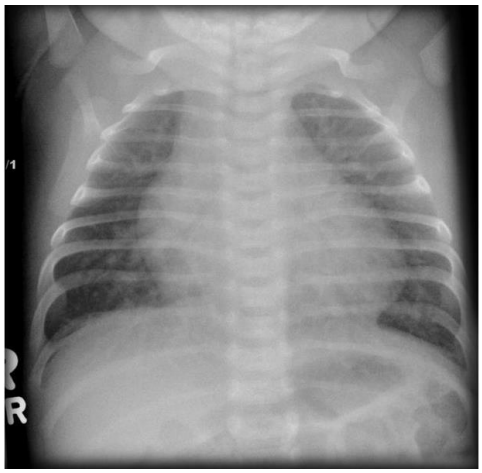
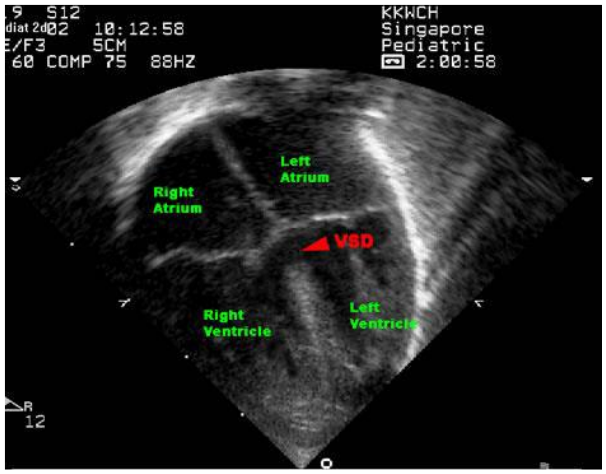
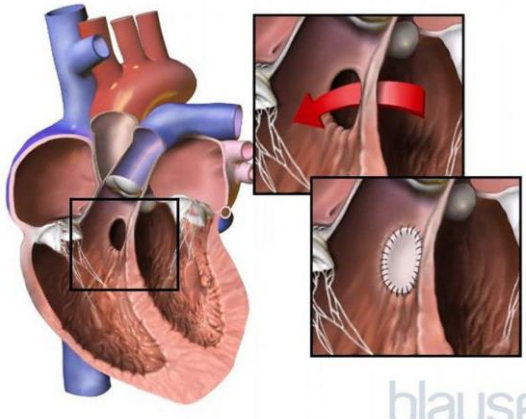
COMPLICATIONS	✦ Reversal of left-to-right shunt, ie **Eisenmenger's syndrome:** initial L-R shunt leads to pulmonary hypertension, hence shunt reversal, causing cyanosis (±heart failure & chest infections). ✦ **Paradoxical emboli** (vein-artery via ASD; rare).
INVESTIGATIONS	✦ **ECG:** RBBB with LAD and prolonged PR interval (primum defect) or RAD (secundum defect). ✦ **CXR:** A prominent pulmonary artery, right ventricular enlargement, and pulmonary plethora are found on chest radiographs ✦ **Echocardiography** is diagnostic.  Chest radiograph showing mild cardiomegaly with prominence of the pulmonary artery (arrow).There are increased pulmonary vascular markings from a left-to-right shunt consistent with a significant atrial septal defect
TREATMENT	✦ **In children:** closure is recommended before age 10yrs. ✦ **In adults:** if symptomatic, or if pulmonary to systemic blood flow ratios of ≥1.5: 1. ✦ Transcatheter closure is now more common than surgical.

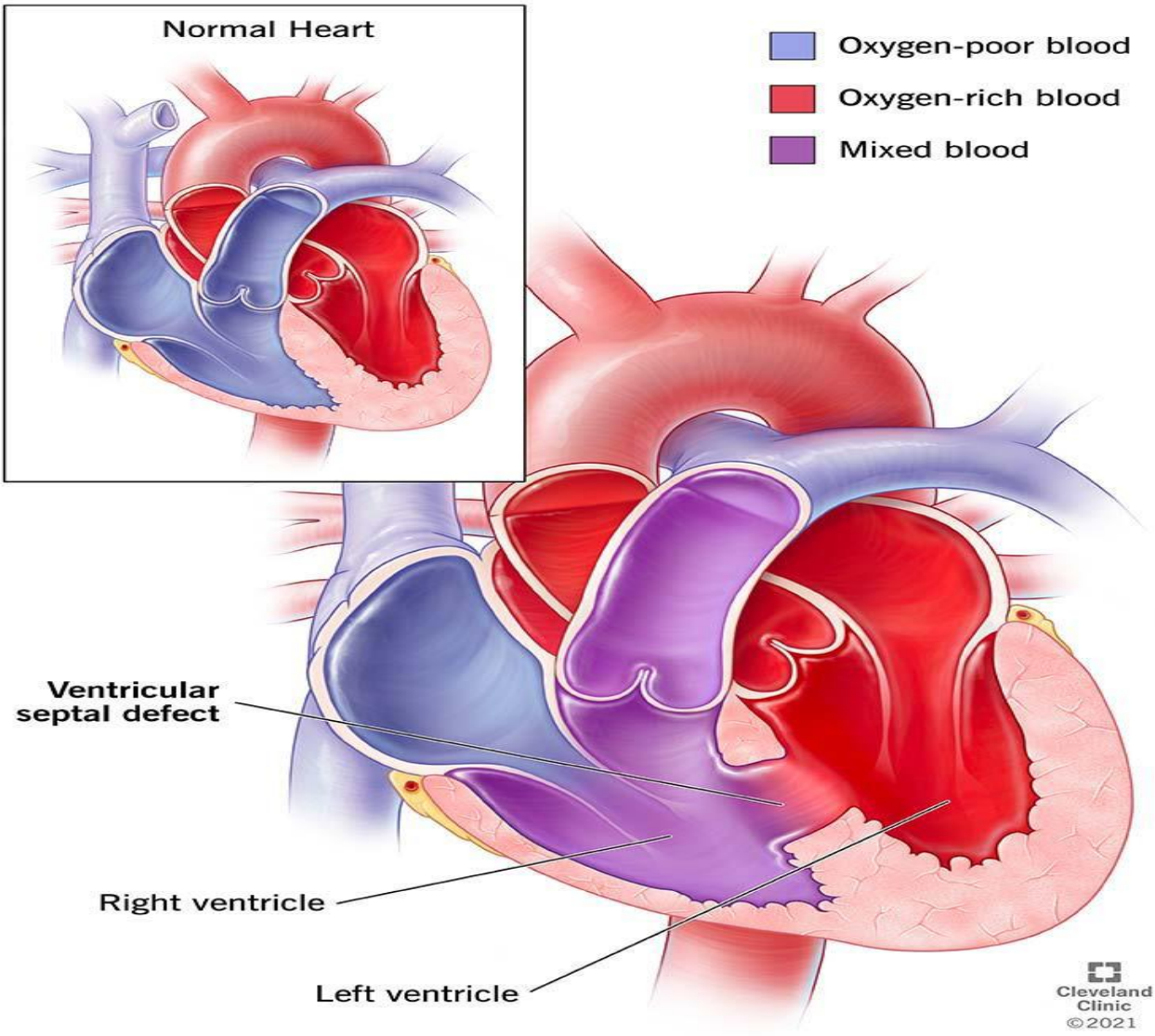


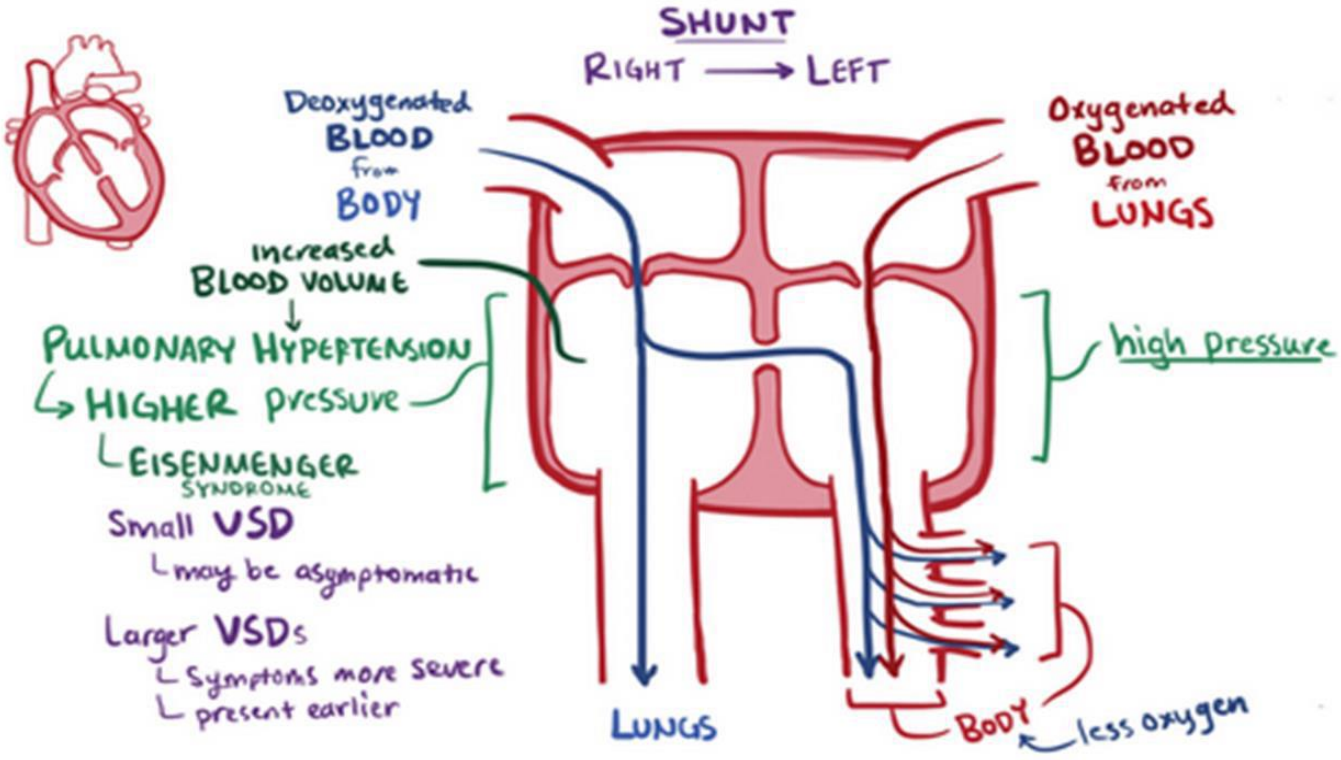


Ventricular septal defect (VSD)

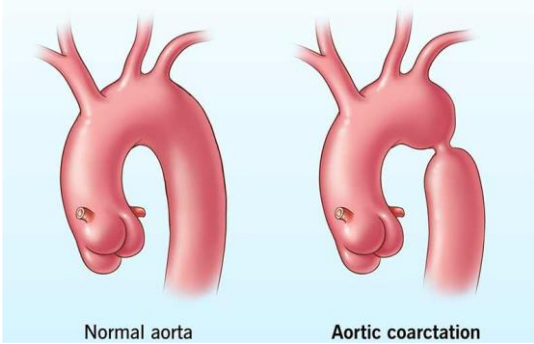
DEF	✦ **Hole** connects the ventricles
TYPES	1. **Membranous:** This is the most common type of VSD and makes up about 80% of cases. These VSDs happen in the upper section of the wall between the ventricles. 2. **Muscular:** These account for about 20% of VSDs in infants, and there is often more than one hole that ' s part of the defect. 3. **Inlet:** This is a hole that happens just below the tricuspid valve in the right ventricle and the mitral valve in the left ventricle. 4. **Subarterial:** occurs in the ventricular septum immediately under the pulmonary valve. These defects are often referred to as supracristal, conoseptal, or doubly committed subarterial defects. 
CAUSES	1. **Congenital** (prevalence 2:1000 births) 2. **Acquired** (post-MI).
SYMPTOMS	✦ **In infants,** moderate to large VSD causes symptoms that look like heart failure. These include: 1. Shortness of breath, including fast breathing or struggling to breathe. 2. Sweating or fatigue during feeding. 3. Failure to thrive (slow weight gain). 4. Frequent respiratory infections. ✦ **VSD in older children and adults can cause:** ✍ Feeling tired or out of breath easily when exercising. ✍ After Eisenmenger syndrome develops, very pale skin or a bluish tinge to skin and lips (a condition called cyanosis) may happen

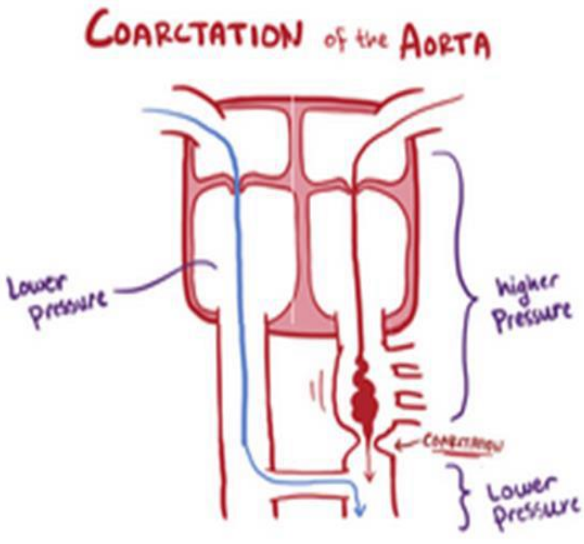
SIGNS	✦ **These depend on size and site:** a. **Smaller holes:** which are haemodynamically less significant, give louder murmurs. Classically, a harsh pansystolic murmur is heard at the left sternal edge, with a systolic thrill, ± left parasternal heave. b. **Larger holes**: are associated with signs of pulmonary hypertension.
COMPLICATIONS	1. AR 2. Infundibular stenosis 3. IE/SBE 4. Pulmonary hypertension and Eisenmenger ' s syndrome.
INVESTIGATIONS	1. **ECG:** normal (small VSD), LAD + LVH (moderate VSD) or LVH + RVH (large VSD). 2. **CXR:** normal heart size ± mild pulmonary plethora (small VSD) or cardiomegaly, large pulmonary arteries and marked pulmonary plethora (large VSD). 3. **Echocardiogram:** Identify the size of the VSD and its exact location. 4. **Computed tomography (CT) scan**  
TREATMENT	✦ This is **medical**, at first, as many close spontaneously. ✦ Indications for surgical closure: failed medical therapy, symptomatic VSD, LV volume overload, SBE/IE. ✦ Endovascular closure is also possible. 

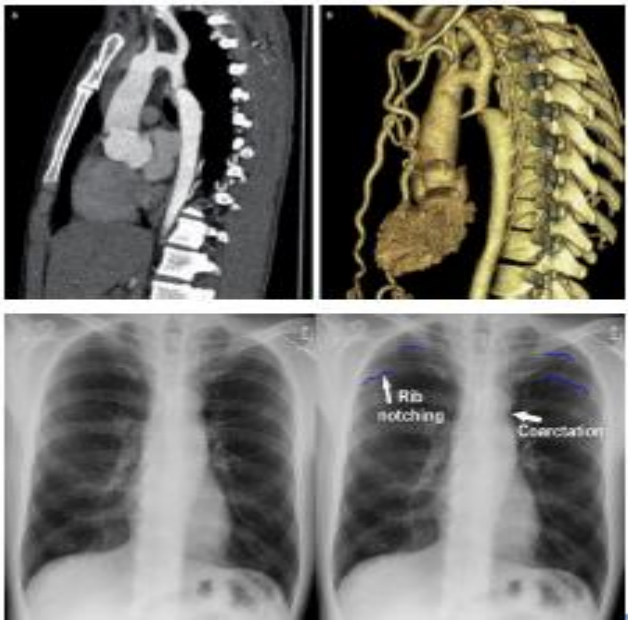

Normal Heart
Oxygen-poor blood
Oxygen-rich blood
Mixed blood
Ventricular septal defect
Right ventricle
Left ventricle
Cleveland Clinic ©2021


SHUNT
RIGHT → LEFT
Deoxygenated BLOOD from BODY
Oxygenated BLOOD from LUNGS
increased BLOOD VOLUME
PULMONARY HYPERTENSION
HIGHER pressure
EISENMENGER SYNDROME
high pressure
Small VSD
may be asymptomatic
Larger VSDs
Symptoms more severe
present earlier
LUNGS
BODY
less oxygen

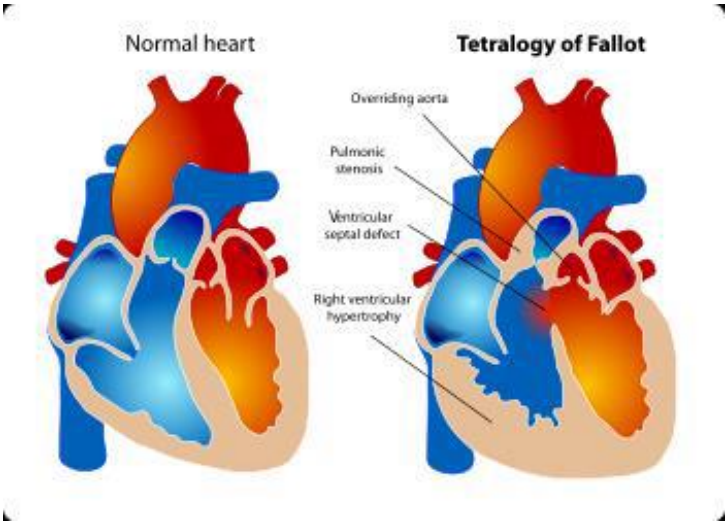
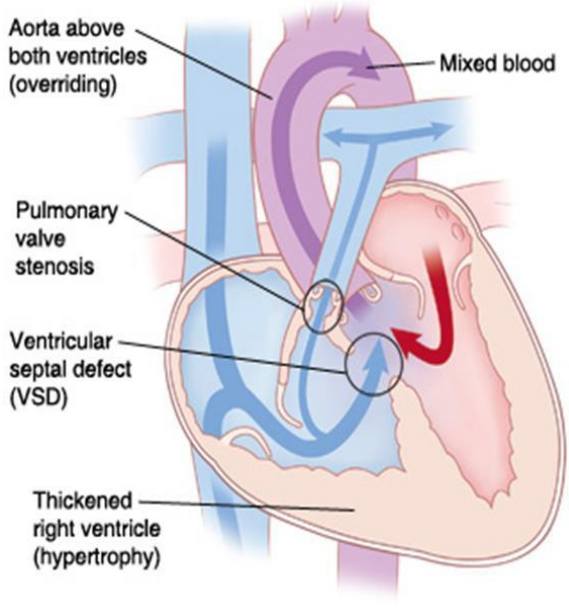
Coarctation of the aorta

DEF	✦ Congenital narrowing of the descending aorta; usually occurs just distal to the origin of left subclavian artery. ✦ **Associations:** 1. Bicuspid aortic valve 2. Turner ' s syndrome. 
SIGNS	1. Radiofemoral delay (femoral pulse later than radial) 2. Weak femoral pulse 3. BP increase 4. Scapular bruit 5. Systolic murmur (best heard over the left scapula).
COMPLICATIONS	✦ Heart failure ✦ Infective endocarditis.
INVESTIGATIONS	✦ CT or MRI-aortogram, CXR shows rib notching.
TREATMENT	✦ Surgery, or balloon dilatation ± stenting

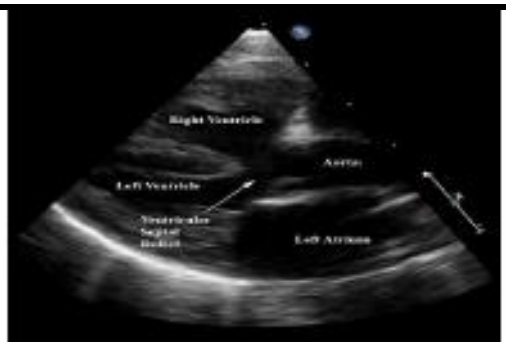
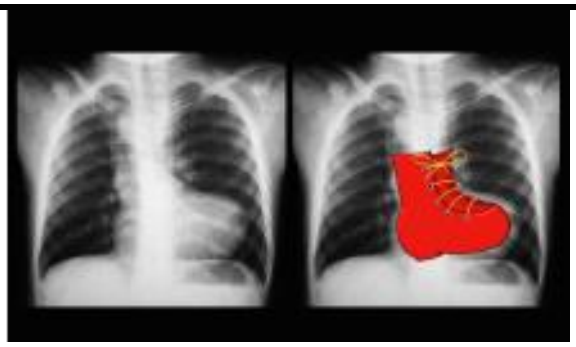




Tetralogy of Fallot (TOF)

INCIDENCE	✦ The most common cyanotic congenital heart disorder 3-6 per 10,000) ✦ The most common cyanotic heart defect that survives to adulthood, accounting for 10% of all congenital defects.
ETIOLOGY	✦ It is believed to be due to abnormalities in separation of the truncus arteriosus into the aorta and pulmonary arteries that occur in early gestation. 
FEATURES	1- Ventricular septal defect (VSD) 2- Pulmonary stenosis 3- Right ventricular hypertrophy 4- The aorta overriding the VSD ✍ Occasionally, a few children also have atrial septal defect, which makes up pentad of Fallot 

PRESENTATION	✦ Severity of illness depends greatly on the degree of pulmonary stenosis. ✦ **Infants** may be acyanotic at birth, with a pulmonary stenosis murmur as the only initial finding. ✦ Gradually (especially after closure of ductus arteriosus) they become cyanotic due to decreasing flow of blood to the lungs as well as right-to left shunt across the VSD. ✦ During a hypoxic spell, the child becomes restless and agitated, and may cry inconsolably. ✦ **Toddlers may squat,** which is typical of TOF, as it increases peripheral vascular resistance and decreases the degree of right to left shunt. Also: difficulty in feeding, failure to thrive, clubbing. ✦ **Adult patients** are often asymptomatic. ✦ In the unoperated adult patient, cyanosis is common, although extreme cyanosis or squatting is uncommon. ✦ In repaired patients, late symptoms include exertional dyspnoea, palpitations, RV failure, syncope, and even sudden death. Children with Tetralogy of Fallot exhibit bluish skin during episodes of crying or feeding.  "Tet spell" A.D.A.M.
INVESTIGATIONS	✦ **ECG** shows RV hypertrophy with a right bundle-branch block ✦ **CXR** may be normal, or show the hallmark of TOF, which is the classic boot-shaped heart. ✦ **Echocardiography:** can show the anatomy as well as the degree of stenosis. ✦ **Cardiac CT and cardiac MRI:** can give valuable information for planning the surgery.

	 
TREATMENT	✦ Give O2. ✦ Place the child in knee-chest position. ✦ Morphine can sedate the child as well as relaxing the pulmonary outflow. ✦ Long-term B-blockers may be needed. ✦ Give endocarditis prophylaxis only if recommended by a microbiologist. ✦ Without surgery, mortality rate is ~95% by age 20. ✦ Surgery is usually done before 1yr of age, with closure of the VSD and correction of the pulmonary stenosis. ✦ 20-yr survival is ~90-95% after repair.