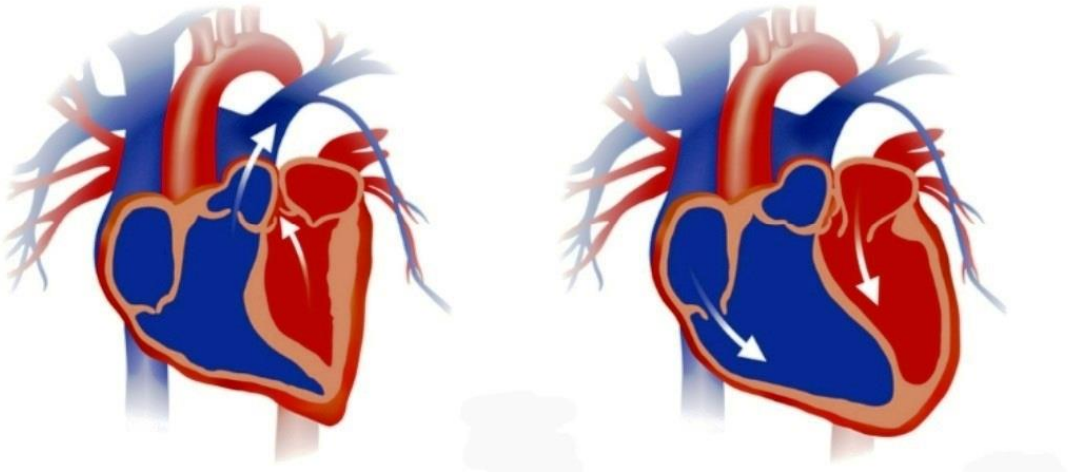
CARDIOVASCULAR SYSTEM

INTERNAL MEDICINE

2024- 2025



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➢ INTRODUCTION:

Anatomy of the Heart:

The Heart Consists of **4 Chambers**
2 Atria & **2 Ventricles**.
Those **4 Chambers** are Separated By **4 Valves;**

Two Right Valves:

➢ **Tricuspid Valve**: Between Right Atrium & Right Ventricle.

➢ **Pulmonary Valve**: Between Right Ventricle & Pulmonary Artery.

Two Left Valves:

➢ **Mitral Valve**: Between Left Atrium & Left Ventricle.

➢ **Aortic Valve**: Between Left Ventricle & Aorta.

Please Remember ✍

Left Vent Thickness **10mm** While Right Vent **3mm**.
Pressure in Lt Vent **4 Times** More Than Rt Vent.

➢ **Stroke Volume** (**SV**) Amount of Blood That Ejected By Ventricles Each **Beat**.
➢ **Cardiac Output** (**COP**): Amount of Blood That Ejected By Ventricles Each **Minute**.
➢ **Ejection Fraction** (**EF**): Percentage of Blood That Ejected From Ventricles, Normally **> 55%**.

Histology of the Heart :

The Heart Consists of 3 Layers:
1- Inner Layer: **Endocardium** Include the **Valves**.
2- Middle Layer: **Myocardium** Containing **Conductive System of the Heart**.
3- Outer Layer: **Pericardium** (Visceral & Parietal).

Blood Supply of the Heart :

Arterial Supply of the Heart Arises From:
Right & Left Coronary Arteries.

Right Coronary Artery:

➢Supply Mainly **Posterior** & **Inferior** Wall of the Left Ventricle (80%).

➢Supply **SA Node** (60%).

➢Supply **AV Node** (90%).

Left Coronary Artery:

Divided Into:

1. Left Anterior Descending (LAD)

➢Supply Mainly **Antero-Septal Wall** of the Heart.

2. Left Circumflex (LCX)

➢Supply Mainly **Lateral Wall** of the Left Ventricle.

Conductive System of the Heart:

1. Sino-Atrial Node (**SAN**): Known as **Peace Maker** Situated in **Right Atrium** Near to the Site of **SVC**. It is **Generate Impulse** ➔ Called **Pulse Generator**, It Can Generate **60** to **100** Beat Per Minute.

2- Atrio-Ventricular Node (**AVN**): It's the **Only** Window Between **Atria** & **Ventricles**. Its Function is **Physiological Delay of Pulse**.

3- Bundle of His.
4- Right & Left Bundle Branch.
5- Purkinje Fibers.

Innervations of the Heart:

Autonomic Nerve Supply, Include:

➢ **Sympathetic Innervations:** By **β1 & β2 Receptors** Increase **Heart Rate** (**Chronotropoic**) and Increase **Contractility** & **COP** (**Inotropic**).

Please Remember ✍
Stimulation of **β2 Receptors** Lead to **Vasodilatation** of **Coronary Blood Vessels**.

➢ **Parasympathetic Innervations:** Vagal Stimulation (Only For **Atrium Not Ventricles**) By **M2 Receptors**. **Decrease** Heart Rate & **Increase** Function of AV Node

➢ HEART FAILURE (HF):

Definition:

It is a Clinical Syndrome Develop When **Heart Can't Maintain Adequate Cardiac Out Put (COP Less Than Tissue Requirement).**
Affect **10%** of People Above **75** Years Old.

COP Depend On **Starling Low**:

- Venous Return (**Pre-Load**): If↑; COP↑.
- Arterial Resistant (**After-Load**): If↑; COP↓.
- Myocardial Contractility: If↑; COP↑.

Please Remember ✍

If **COP Decreased**; Myocardial Contractility Will Be **Increased** Because;
Decreasing of **COP** Lead to **Decreasing** of **Carotid Sinus Activity** Which Will Lead to Increasing of **Sympathetic Activity** and it is Called **Sympathetic Firing**,
This Sympathetic Firing Lead to Increase **Heart Contractility** & Increasing of **Heart Rate**.

Pathophysiology:

According to Definition; in Case of Heart Failure **COP** Will Be Decreased **Less** Than Normal,
Decreasing of **COP** Lead to Decrease of **Renal Perfusion** & That Will Activate **Sympathetic Nervous System** and **Renin Angiotensin Aldosterone System (RAAS):**
Renin Convert **Angiotensinogen** to **Angiotensin I**, **Angiotensin I** Converted Into **Angiotensin II** By **Angiotensin Converting Enzyme (ACE).**

❖ **Angiotensin II (Ag II)** Will Do:

- **Vasoconstriction** Which Lead to ↑ **After-Load.**
- **Stimulate Secretion of Aldosterone** That Will Lead to **Salt & Water Retention** and Result Into Increasing of **Venous Return** Lead to ↑ **Pre-Load.**

❖ **Sympathetic Nervous System** Will Do:

- Stimulation of Cardiac Contractility.
- Vasoconstriction.

This Mechanism Called: **Neuro-Hormonal Activation** That Occur in Case of Decrease of **COP**, Which is **Physiological** in Case of **Healthy Heart** But **Pathological** in Case of **Diseased Heart**, Because It Will Lead to **Deterioration** of Heart Function & **Remodeling** of the Heart.

Classification:

According to **Involved Chamber:**	According to **Cardiac Out Put:**	According to **Cardiac Cycle:**	According to **Onset of Clinical Picture:**
1. Right Side Heart Failure. **2.** Left Side Heart Failure. **3.** Congestive Heart Failure (Biventricular).	**1.** High Cardiac Output Heart Failure (Due to **Hyperdynamic Circulation**). **2.** Low Cardiac Output Heart Failure.	**1.** Systolic Heart Failure (**More Common**). Due to Decrease Myocardial Contraction. **2.** Diastolic Heart Failure (**Less Common**). Due to Decrease Ventricular Filling.	**1.** Acute Heart Failure. **2.** Chronic Heart Failure. **3.** Acute On Top of Chronic Heart Failure.

✵ New York Heart Association (NYHA) Classification of Heart Failure:

Class I: No Limitation of Physical Activity.
Class II: Slight Limitation of Physical Activity.
Class III: Marked Limitation of Physical Activity.
Class IV: Symptoms Occur Even at Rest; Discomfort with Any Physical Activity.

Symptoms:

➢ Right Side Heart Failure:

1. Right Hypochondrial Pain & Jaundice.
(**Cardiac Cirrhosis**).
2. Abdominal Distension, Dyspepsia, Malabsorption & Sever Weight Loss.
(**Cardiac Cachexia**).
3. Bilateral Lower Limb Edema.
4. Generalized Edema (**Anasarca**); Develop Only In **Severe Cases**.

➢ Left Side Heart Failure:

1. Dyspnea, Orthopnia, Paroxysmal Nocturnal Dyspnea (**PND**).
2. Cough & Wheeze (**Cardiac Asthma**).
3. Dizziness, Fatigue, Weakness, Chest Pain, Palpitation, Syncope, Cold Periphery, **Oliguria**, Insomnia, Headache & Intermittent Claudication

Signs:

➢ Right Side Heart Failure:

1. Raised Jugular Venous Pressure (↑JVP).
2. Right Hypochondrial Tenderness with Jaundice, Ascites & Hepatomegaly.
3. Bilateral Lower Limb Edema / Sacral Edema.
4. Generalized Edema (**Anasarca**); Develop Only In **Severe Cases**.

➢ Left Side Heart Failure:

1. Gallop Rhythm (**in Acute Lt Side Heart Failure**).
2. Bilateral Basal Crackles of Lungs, Pleural Effusion (**Decrease** Air Entry By Auscultation & **Dull** By Percussion).
3. Pallor, Cold Periphery, Sweating, Tachycardia, Low Blood Pressure, **Oliguria**, Confusion & Memory Impairment.

Precipitating Factors of Heart Failure:
FAILED HEART

F: Failure to Take Medications **A**: Arrhythmia **I**: Infection **L**: Lung Diseases
E: Endocrine Problem (Pheochromocytoma) **D**: Drugs (Verapamil, Diltiazim, NSAIDS, Steroid & IV Fluid)
H: Hypertension **E**: Endocarditis **A**:Anemia & Alcohol **R**: Rheumatic Fever **T**: Thyrotoxicosis

All Those Factors Cause **Firing of Chronic Heart Failure** & Lead to **Acute On Top of Chronic Heart Failure** Because They Responsible to Change State of Patient with Heart Failure From **Compensated** to **Uncompensated**
➔ **So**... All Patients with **Heart Failure** Should **Avoid** All Those **Risk Factors**.

Please Remember

Left Side Heart Failure Cause Left Ventricular Dilatation & The Dilatation Lead to Functional Mitral Regurgitation
Right Side Heart Failure Cause Right Ventricular Dilatation & The Dilatation Lead to Functional Tricuspid Regurge.

Prolonged Chronic Left Side Heart Failure Cause ➔ **Pulmonary Hypertension**,
Prolonged Pulmonary Hypertension Cause ➔ **Right Side Heart Failure**.
- But Right Side Heart Failure **Doesn't Cause** Left Side Heart Failure.

Causes:

Causes Related to Myocardial Dysfunction:	Causes Related to Ventricular Outflow Obstruction:
3 M:	**AS 3P:**
1. **M**yocardial Infarction (**Most Common**).	1. **A**ortic Stenosis.
2. **M**yocarditis.	2. **S**ystemic Hypertension.
3. **M**yopathies (Dilated Cardiomyopathy DCM).	3. **P**ulmonary Hypertension
	4. **P**ulmonary Stenosis.
	5. **P**ulmonary Embolism.

Causes Related to Ventricular Inflow Obstruction:	Causes Related to Ventricular Volume Overload:
MT Stenosis:	**All Valves Regurgitation:**
1. **M**itral Stenosis.	1. Mitral & Aortic Regurge
2. **T**ricuspis Stenosis.	2. Tricusp & Pulm Regurge
	3. Hyperdynamic Circulation

Causes Related to Diastolic Dysfunction:	Causes Related to Cardiac Rhythm:
CRP:	**Sever Tachycardia & Sever Bradycardia:**
1. **C**onstrictive Pericarditis.	1. Ventricular Tachycardia
2. **R**estrictive Cardiomyopthy.	2. Ventricular Fibrillation
3. **P**ericardial Tamponade.	3. Third Degree Heart Block

Investigations :

1. Echocardiography: (Diagnostic)
Transthoracic Echocardiography (TTE)
Measure Ejection Fraction [EF]; (Normally >50%)
(EF in Sever Heart Failure Less Than 35%).
Detect Valve Lesions (Stenosis or Regurgitation).
Detect Cardiomyopathy & Cardiac Tamponade.

2. 12 Lead Electro Cardiography (ECG):
Detect Ischemic Heart Disease & Arrhythmias.

3. Blood Workup For:
Level of B-Type Natriuretic Peptide (**BNP**) or N-Terminal ProBNP (**NT-ProBNP**) ➔ Elevated Due to Ventricular Dilatation.
Level of Atrial Natriuretic Peptide (**ANP**) ➔ Elevated Due to Atrial Dilatation.

BNP Used Mainly For:
Diagnosis, Prognosis & Follow Up of Heart Failure.
Differentiate Between **Cardiac** & **Non Cardiac Dyspnea**

4. Chest X Ray (CXR):
Detect Pulmonary Venous Congestion.

5. Other Blood Workup
Arterial **B**lood **G**as (ABG) For **Hypoxia**.
Complete **B**lood **C**ount (CBC) For **Anemia** & **Infection**
Thyroid **F**unction **T**est (TFT) For **Thyorotoxicosis**.
Renal **F**unction **T**est (RFT) For **Complications**.
Liver **F**unction **T**est (LFT) For **Liver Congestion**.

Cor-Pumonale Means Any Chronic Lung Parenchymal or Chest Wall Diseases Lead to Isolated Right Ventricular Hypertrophy with or without Heart Failure.

Treatment:

❖ Non Pharmacological Treatment:

Patient Education About Life Style Control Which are:

Decrease Body Weight + Regular Aerobic Exercise + Regular Vaccinations + Salt Free Diet + Fluid Restriction + Small Frequent Meals + Stop Smoking + Stop Alcohol + Avoid Some Drugs as Ca Channel Blockers & NSIDS.

❖ Pharmacological Treatment:

1. Diuretics:

Mechanism of Action:

Decrease Preload (Main Action) Also ↓Afterload

Types:

-Loope Diuretics (furosemide = Lasix): 1st Line

-Thiazide (Metolazone, Bendroflumethiazide).

-Potassium Sparing Diuretic: Spiranolactone & Eplerenone **(Which Improve Survival Rate)**.

2. Angiotension Converting Enzyme Inhibitor (ACE I):

Mechanism of Action:

Decrease Afterload (Main Action).

They **Improve Survival Rate** & Decrease Hospitalization

Types:

Captopril (Capotin): Has Rapid Action.

Enalapril, Lisinopril, Ramipril, Quinapril.

➢ **ACE I Improve Survival Rate.**

3. Angiotension II Receptors Blockers (ARBs):

Mechanism of Action:

Decrease Afterload (Main Action).

Types:

Candisartan, Valsartan, Irbesartan, Losartan

4. β- Blockers

Mechanism of Action:

Moderate Afterload Reduction & Slight Preload Reduction But the Main Action is Protect the Heart Against Sympathetic Stimulation & Inhibit Remodeling & Decrease Risk of Arrhythmias (VT) So Reduce Risk of Sudden Death & Decrease Hospitalization.

Given When the Patient Stabilized By Other Medications

Types:

Carvidolol, Metopolol, Bisoprolol, Nebivolol.

➢ **β- Blockers Improve Survival Rate.**

5. Vasodilators:

Mechanism of Action:

Decrease Preload & Afterload.

Types:

Nitrate, Hydralazine, Nitroprosside Sodium

6. Digoxin:

Mechanism of Action:

Increase Intracellular Calcium That Lead to Increase Myocardial Contractility.

*Also Increase Vagal Activity Leading to Decrease Conduction of Impulse Through AVN

➢ **Digoxin Doesn't Improve Survival Rate.**

7. Amiodaron:

It is a Strong Anti-Arrhythmic Drug which has Little **Negative Inotropic** Effect and May Be Valuable in Patients with **Poor** Left Ventricular Function.

Only Effective in Treatment of **Symptomatic Arrhythmias** & Should **Not** Be Used in Asymptomatic Patient.

8. New Drugs: Ivabradine (Regulate Heart Rate & Decrease Hospitalization), **Neprilysin Inhibitor** (Sacrubitril) Better If It Used with **ARBs** (Valsartan) Known as **Entresto** (Reduce Risk of Death & ↓Hospitalization)

SGLT-2 Inhibitors Which are: **Empagliflozin**, **Dapagliflozin** & **Sotagliflozin** (Reduce Risk of Death & ↓Hospitalization)

❖ Interventional Treatment in Patient with Heart Failure:

1. **I**mplantable **C**ardiac **D**efibrillator **(ICD):** Indicated in Patients with Heart Failure Who Have Had, or Who are at High Risk of **Ventricular Arrhythmia**.

2. **C**ardiac **R**esynchronization **T**herapy Device **(CRT):** Indicated in Patients with **Marked Conduction System Disease**, Especially **Left Bundle Branch Block**.

3. Coronary Revascularization (**PCI** & **CABG**): For Selected Patients with Heart Failure & Coronary Artery Disease.

4. Heart Transplantation: It is the Last & Definitive Surgical Intervention.

About Heart Failure Medications:

➢ Side Effect of Furosemide:

1. Hypotension (Hypovoleamia)
2. Hypocalceamia,
3. Hypokaleamia,
4. Hyponatreamia
5. Hypomagneseamia,
6. **Hyperglyceamia**,
7. **Hyperureceamia**.

➢ Side Effect of Thiazide:

Same as Side Effects of Furosemide Except:

1. **Hypercalceamia** (Not Hypocalceamia),
2. **Hyperlipideamia**.

Please Remember

furosemide & Thiazide Improve Symptoms But **Doesn't Improve Survival**.

➢ Side Effect of Spironolactone:

Hyperkaleamia & Gynecomastia.

Please Remember

Spironolactone **Improve Survival Rate**.

➢ Side Effect of ACE Inhibitors:

1. Dry Cough (**Most Common Side Effect**) in 10%-15%.
2. Firs Dose Hypotension: So In the First Day Should Be Given at **Bed Time** with **Half Dose**.
3. Hyperkaleamia So **Don't** Give It with Spironolactone.
4. Renal Impairment In Patient with **Bilateral Renal Artery Stenosis**: So Renal Function Test Should Be Checked 1-2 Weeks After Starting Treatment.
5. Angioedema.
6. Skin Rash & Leukopenia (Rare).

Please Remember

ACE I are Given to **All Patients** with Heart Failure, Because ACE I **Inhibit Remodeling** of the Heart & **Improve Survival** (Prognosis) & **Decrease Mortality Rate** In Patients with Heart Failure.

About Heart Failure Medications:

➢ Side Effect of Ag II Receptors Blockers:

Same Side Effects of ACE I, Except: **No Cough**.

Please Remember

ARBs **Improve Survival** & **Decrease Mortality** In Patients with Heart Failure (Similar to ACE I) & They are a Useful **Alternative** For Patients Who **Can't Tolerate ACE I Side Effects** as **Cough**.

➢ Side Effects of β-Blockers:

1. Peripheral Vasoconstriction.
2. Mask Symptoms & Signs of Hypoglyceamia.
3. Bronchospasm.
4. Depression & Nightmares.
5. Acute Heart Failure.

Contraindications of β-Blockers:
(BADR Heart Failure)

1. **B**radycardia.
2. **A**sthma.
3. **D**iabetes Mellitus.
4. **R**eynaud's Phenomenon.
5. Heart Failure Grade 4 ➔ (NYHA Class IV).
6. Active Heart Failure (Acute Symptoms).

➢ Side Effect of Digoxin:

Digoxin Toxicity (Digoxin **Not** Given to Patient with **Hypokaleamia**; Because It Lead to **Toxicity**).

Indication of Digoxin Use:

1. Heart Failure with Atrial Fibrillation.
2. Marked Systolic Dysfunction (Severe HF).
3. Persistent of Symptoms Even After Optimal Dose of Diuretics & ACEI.

Complications:

1. Cardiac Arrhythmias:

- Very **Common** and Related to **Electrolytes Disturbance**, Include:
  - Atrial Fibrillation (**Occur in 20%**).
  - Ventricular Tachycardia.
  - Ventricular Fibrillation.
  - Ventricular Ectopic.

2. Organ Failure:

- **Renal Failure:**
  - Due to Poor Renal Perfusion & May **Exacerbated** By **Diuretics**, **ACE I** & **ARBs**.

- **Liver Failure:**
  - Due to Hepatic Venous Congestion & Poor Arterial Perfusion.

3. Thrombo-Embolism:

- Due to Blood Stasis Which Occur Due to Low COP or Patient Immobility, Which Include:
  - **Deep Venous Thrombosis (DVT).**
  - **Pulmonary Embolism (PE).**

4. Electrolytes Disturbance:

- Mostly Due to **Medications Side Effects**:
  - Hyperkaleamia Due to **ACE I** & **Spironolactone**.
  - Hypokaleamia Due to **Furosemide** & **Thiazide** (Also Due to Activation of RAAS).
  - Hyponatreamia Due **Diuretics** and Also Due to **Severe Heart Failure** and It is a **Poor Prognostic Feature**.

5. Sudden Death:

- Occur in 50% of Patients with Heart Failure and Most Often Due to **Ventricular Fibrillation**.

➢ ACUTE PULMONARY EDEMA:

Definition:	Causes:
It is an Acute **Left Side** Heat Failure.	Most Common Cause ➔ Myocardial Infarction. Also Caused By Atrial Fibrillation with Mitral Stenosis

Pathophysiology :

Sudden Left Ventricular Failure Lead to ➔ Increase of **Left Ventricular Pressure**,
Raised Left Ventricular Pressure Lead to ➔ Sudden Increasing of Pressure In **Left Atrium**,

Raised Left Atrial Pressure Lead to ➔ Sudden Increasing of Pressure in **Pulmonary Capillaries**,

Raised Pulmonary Capillaries Pressure Lead to ➔ Sudden **Pulmonary Congestion**,
Pulmonary Congestion Cause ➔ Movement of Fluid **From** Pulmonary Capillaries **To** Alveoli & Lung Interstitium,

Movement of Fluid From Pulmonary Capillaries To Alveoli Cause ➔ **Alveolar Edema** &
Fluid Moves From Pulmonary Capillaries To Lung Interstitium Cause ➔ **Interstitial Edema**
This Known as ➔ **Acute Pulmonary Edema**.

Please Remember

Alveolar Edema:	Interstitial Edema:
Accumulation of Fluid In **Alveolar Space**.	Accumulation of Fluid In **Lung Interstitium** (More at Base).
➢ Clinically Patient Presented with **Symptoms**: **1**. Cough with Frothy Sputum, **2**. Wheeze. **3**. Dyspnea, **4**. Orthopnea, **5**. PND.	➢ Clinically Patient Presented with **Sign**: Bilateral Basal Fine Crackles.

Symptoms:	Signs:
1. Dyspnea. **2**. Orthopnia. **3**. PND. **4**. Cough with Frothy Sputum. **5**. Heamoptysis. **6**. Wheeze.	**1**. Agitation & Distressed. **2**. Pale Periphery & Central Cyanosis. **3**. Hypotension & Tachycardia. **4**. Bilateral Basal Crackles. **5**. Raised Jugular Venous Pressure.

Investigations:

1. Chest X Ray (to Detect Pulmonary Edema).
2. Echocardiography (to Confirm Diagnosis).
3. Cardiac Enzyme (to Detect MI).
4. Arterial Blood Gas (to Detect Hypoxia).

Please Remember 🕊

Features of Pulmonary Edema in Chest X Ray ➔ (**ABCDE**):

A ➔ Alveolar Edema:
Butterfly Opacity.
B ➔ B- Line (Kerley-B Lines):
Due to Interstitial Edema.
C ➔ Cardiomegaly.

D ➔ Dilated Upper Lobe Vessels.

E ➔ Pleural Effusion.

Treatment:

It is an Emergency Case:

- Call For Help + Admission In Coronary Care Unit Patient On Sitting Position + Full Monitoring and Give High Flow Oxygen.

MODN:

1. **Morphine** ➔ (5-10mg IV) Best Choice to Decrease Anxiety & Agitation of the Patient. Also Morphine **Decrease Dyspnea** & Can Lead to **Vasodilatation**.
(Morphine Given with Metolopramide 10mg IV).

2. **Oxygen** ➔ **C**ontinuous **P**ositive **A**irway **P**ressure (CPAP) of 5–10 mmHg By Tight-Fitting Mask.

3. **Diuretics** ➔ Furosemide (50-100mg IV) is the Best Choice, Because It Has Rapid Action.

4. **Nitrate** ➔ IV GTN (10-200Mcg/Min) or Buccal Given Only If Patient Blood Pressure **More Than 110 mmHg**.

Nitrate is Vasodilator Drug (Veno-Dilator) Lead to Dilation of Veins & Decrease Venous Return So **Improve Pulmonary Congestion**.

5. If Previous Measures Failed; ➔ Give **Positive Inotropic Drug** (**Doputamine2.5Mcg/kg/min**).

➢ SYSTEMIC HYPERTENSION (HTN):

Definition:

It is a Condition in Which **Arterial Blood Pressure** is Clinically Elevated More Than **139/89 mmHg** Based on 2 or More Reading in 2 or More Occasions.

Types:

1ry HTN (Essential HTN)
Present in 95% of People, Unknown But May Related to **Genetic Factor** (Most Common) & **Life Style**.

2ry HTN
Present in 5% of People, Caused By: **RED COPA;** **R**enal, **E**ndocrine, **D**rugs, **C**oarctation of Aorta, **O**SA, **P**regnancy, **A**lcohol

Causes:

Primary HTN:

- Genetic (**Most Common 60%**).
- Life Style, Stress, DM Type II.

Secondary HTN: RED COPA

- **R**enal: Renal Artery Stenosis, Glomerulonephritis, Polycystic Kidney Disease.
- **E**ndocrine: Hypothyroidism, Thyrotoxicosis, Hyperparathyroidism, Cushing Syndrome, Conn's Syndrome, Pheochromocytoma, Acromegaly.
- **D**rugs: OCP, Steroids, NSAIDs.
- **C**oarctation of Aorta.
- **O**bstructive Sleep Apnea (OSA) + **O**besity.
- **P**regnancy.
- **A**lcohol.

Target Organs in HTN:

- **Blood Vessels:** Atherosclerosis & Aneurysm.
- **Heart:** Atrial Fibrillation, Left Ventricular Hypertrophy, IHD.
- **Nervous System:** Stroke, Subarachnoid Heamorrhage (SAH), TIA.
- **Kidneys:** Chronic Kidney Disease, Hypertensive Nephropathy.
- **Retina:** Hypertensive Retinopathy.

Treatment:

❖ Non Pharmacological Treatment:

Patient Education About Life Style Control Which are:
Decrease Body Weight + Regular Aerobic Exercise + Salt Free Diet + Stop Smoking + Stop Alcohol + Low Saturated Fat in Diet + Increase Vegetables, Fruits and Omega 3 in Diet.

❖ Pharmacological Treatment:

Diuretics, ACE Inhibitors (ACEI), Angiotensin II Receptors Blockers (ARBs), Calcium Channel Blockers (CCBs), β-Blockers, Vasodilators.

Calcium Channel Blockers Classified Into: **Dihydropiridine**: Nifedipine & Amlodipine (Cause Reflex **Tachycardia**)
Rate Limiting: Verapamil & Diltiazem (Cause **Bradycardia** So Considered Also as an **Anti-Arrhythmic Drugs**).

In Patient > 55 Years or White:
Step I: Give ACEI or ARBs.
If Not Respond; Go to Step II & Add CCBs.
If Not Respond; Go to Step III & Add Diuretics.
If Not Respond; Go to Step IV & Add β-Blockers or Add Other Diuretic.

In Patient > 55 Years or African or Caribbean:
Step I: Give CCBs.
If Not Respond; Go to Step II & Add ACEI.
If Not Respond; Go to Step III & Add Diuretics.
If Not Respond; Go to Step IV & Add β-Blockers or Add Other Diuretic.

➢ ATHEROSCLEROSIS:

Definition:

It is a **Progressive Inflammatory** Disorder of **Arterial Wall**, Characterized by **Focal Lipid-Rich Deposits of Atheroma**.

➢ **Atheroma** It is **Yellow Swelling** Results From Collections of **Fat**, **Smooth Muscle Cells** in Sub-Intimal Surface of the Artery.

Atheroma May Become Large in Size & Cause Impairment of Arterial Perfusion (**Ischemia**) or May Rupture & Leads to Platelets Accumulation Forming **Thrombus** That Cause **Partial** or **Complete Obstruction** of Artery.

Risk Factors:

➢ **Non-Modifiable:**
1. Age (Most Common).
2. Sex (Male > Female).
3. Positive Family History.

➢ **Modifiable:**
1. Alcohol. 2. Smoking.
3. DM. 4. HTN. 5. Diet.
6. Hyperlipidemia. 7. Obesity.
8. Sedentary Life. 9. Stress.

Please Remember ✍

➢ **Atherosclerosis** It Can Affect Any Artery But the Most Common Arteries:
Coronary Arteries Causing ➔ **Ischemic Heart Disease.**
Cerebral Arteries Causing ➔ **Ischemic Stroke.**
Peripheral Arteries Causing ➔ **Intermittent Claudication.**

➢ ISCHEMIC HEART DISEASES (IHD):

Cause:

Atherosclerosis (**Atheroma**) in Coronary Artery.

Types:

1. **Stable Angina** (or Called Angina Pectoris).
2. **Unstable Angina.**
3. **Myocardial Infarction** (Partial + Full Thickness).

Classification of Ischemic Heart Diseases:

Chronic Coronary Artery Disease (CAD):
Stable Angina.

Acute Coronary Syndrome (ACS):
1. Unstable Angina.
2. Partial Thickness Myocardial Infarction.
3. Full Thickness Myocardial Infarction.

➢ STABLE ANGINA (ANGINA PECTORIS):

Definition:	Clinical Pictures:
It is a Clinical Syndrome of **Chest Pain** Due to **Transient Myocardial Ischemia** Caused By **Narrowing** of Coronary Artery Lumen Due to **Atheroma Formation (Atherosclerosis).**	Sudden Onset **Retrosternal** Chest Pain, (**Heaviness or Pressure or Squeezing** in Character), Aggravating By **Exertion**, **Heavy Meal**, **Exposure to Cold** & **Emotional Stress**, Relieving By **Rest** & **Glyceril Tri-Nitrate (GTN)**, Radiating to the **Jaw**, **Neck**, **Left Shoulder**, **Left Arm & Epigastium**, Lasting For **Less Than 10 Minutes (1-5 Minutes).**

Investigations:

1. Stress ECG (Exercise ECG): **Confirmatory Investigation** After Typical History.

Done Via ➔ **Bruce Protocol** By Using **Treadmill** or **Bicycle** & During Exercise Monitor ECG & Blood Pressure & Observe the Patient Clinically for Any Pain or Dyspnea.

According to Bruce Protocol Positive If:
- ECG Shows Arrhythmia or ST Depression.
- Blood Pressure Decrease More than 10mmHg.
- Clinically Patient Has Pain or Dyspnea.

Stress ECG Sensitivity is 75% ➔ So Negative Stress ECG **Does Not** Exclude Stable Angina.
It Can Be **False Positive** in Left Ventricular Hypertrophy, Digoxin Effect, Left Bundle Branch Block & WPW.

Please Remember
Stress ECG Contraindicated in:
1. Acute Coronary Syndrome (ACS).
2. Recent ACS Within 6 Days.
3. Sever Aortic Stenosis.
4. Sever & Uncontrolled Hypertension.
5. Decompensated Heart Failure.
6. Hypertrophic Cardio-Myopathy.

Please Remember
The Usual ECG That Known as:
12 Lead Resting ECG Not Favor in Case of Stable Angina;
Because the Result Will Be **Normal** Due to Patient in Rest Stat
It Can Be Abnormal If ECG Done and Patient Had Attack of Chest Pain.

2. CT Coronary Angiography is the **Imaging Investigation of First Choice**: It Clarifies the Diagnosis and Guides the Use of Anti-Anginal Therapies & Excellent Guides the Use of Invasive Cardiac Catheterization.

Please Remember
CT Coronary Angiography Help in:
1. Diagnose the Cause of Coronary Artery Narrowing.
2. Reveal the Site & Extent Size of Coronary Lesion.
3. Determine the Way of Treatment.

3. Stress Echocardiography: Done For Patients Which Already Has Coronary Atherosclerosis Disease.
Do Echocardiography at Rest, Then Give Patient Dobutamin (Pharmacological Stress) and Repeat Echocardiography;
Echocardiography During Rest Show ➔ **Normal** Myocardial Contractility,
But Echocardiography After Pharmacological Stress (Dobutamin) Show ➔ **Abnormal** Myocardial Contractility.

Please Remember
Stress Echocardiography is the Best Choice For Patient Who **Can Not Tolerate Exercise**
& Highly Sensitive **(78%)** & Highly Specific **(86%)**.
Stress Echocardiography Need Good Expert in Echocardiography Machine (**Operator Depended**).

4. Myocardial Perfusion Scan: Done For Patients Which Already Has Coronary Atherosclerosis Disease.
Give the Patient **Thalium TI-201** or **Technetium 99**, Then Do Isotope Scan at Rest,
Then Give the Patient Dobutamin (Pharmacological Stress) and Repeat Isotope Scan;
Isotope Scan During Rest Show ➔ **Good** Perfusion of Myocardium.
But Isotope Scan After Dobutamin Show ➔ **Abnormal** Perfusion Defect of Myocardium.

Please Remember
Myocardial Perfusion Scan is **Highly Specific (83%)** & **Highly Sensitive (77%)** But It is **Expensive**.

5. Coronary Angiography (**Diagnostic Cardiac Catheterization**):
It is an **Invasive** Test, Done When Other Non-Invasive Tests Have **Failed** to Find the Cause of the Chest Pain.

Indications of Coronary Angiography:

1. Heart Failure with Angina.
2. Post MI or Unstable Angina.
3. MI or Angina in Patient Less Than 50 Years.
4. ECG Changes After Doing Stress ECG.
5. Symptomatic Patient Even After Medications.

Risk Stratification of Stable Angina:

Risk Stratification Help to Determine **Prognosis** & **Treatment:**

Low Risk If:

1. Predictable Exertional Dyspnea.
2. Stress ECG Shows Ischemia at **High** Workload.
3. Echocardiography **Good** Ejection Fraction.
4. Coronary Angiography Shows **Single** Vessel Involvement or Involvement of **2 Vessels**.

High Risk If:

1. Post Myocardial Infarction.
2. Stress ECG Shows Ischemia at **Low** Workload.
3. Echocardiography **Low** Ejection Fraction.
4. Coronary Angiography Shows Left **Main Coronary Artery** Involvement or Involvement of **3 Vessels**.
5. DM & Comorbidity.

Treatment:

❖ Non Pharmacological Treatment:

Patient Education About Life Style Control Which are:

Stop Smoking + Control DM + Control HTN + Control Serum Cholesterol + Decrease Body Weight
Regular Exercise + Low Saturated Fat in Diet + Increase Vegetables, Fruits and Omega 3 in Diet.

❖ Pharmacological Treatment:

1. Nitrates:

Mechanism of Action:

- Coronary **Vasodilatation** So; ↑ Heart O2 Supply.
- Venodilatation So; ↓ Heart O2 Demand.
- Arteriolodilatation So; ↓ Heart O2 Demand.

Types:

1. Sublingual Glyceryl Tri Nitrate (**GTN**):
Has **Rapid Action** (2-3Min) & **Short Duration** (10-30Min).
2. Transdermal GTN (GTN Patch): 5-10mg Daily.
3. Oral Iso-Sorbid Mono-Nitrate (ISMN): 20-60mg Daily
Has Long Duration of Action.
4. Iso-Sorbid Di-Nitrate (ISDN): 10-20mg 3 Times Daily
Has Long Duration of Action.

Side Effects:

1. Severe Headache.
2. Hypotension.
3. Tolerance (Avoided By 6-8 Hours Free of Drug).

2. β– Blockers:

Mechanism of Action:

- **Decrease** Oxygen **Demand** By Reducing Heart Rate, Blood Pressure and Myocardial Contractility.

Types:

Metoprolol (50-200mg), Bisoprolol (5-15mg) Nebivolol, Carvidolol, Atenolol, Propranolol.

β – Blockers Should **Not** Be Withdrawn Sudden; Because this Lead to **Worsening of Angina**, **Arrhythmias** & **Myocardial Infarction**.

Side Effects:

1. Peripheral Vasoconstriction.
2. Mask Symptoms of Hypoglyceamia.
3. Bronchospasm.

3. Calcium Channel Blockers:

Mechanism of Action:

- **Arteriolodilatation** So; ↓ Myocardial O2 Demand.
- **Decrease Cardiac Contractility**.
- **Decrease Heart Rate** Because They Act On SA Node.

Types:

Dihydropyridine: Amlodipine, Nifedipine, Nicardipine.
Rate Limiting: Verabamil, Diltiazem (**Cause Bradycardia**).

4. Anti-Platelets:

Prevent Thrombus Formation By Inhibiting Platelets Aggregation.

Include:

- Aspirin 75mg: (Inhibit COX).
- Clopidogril (Plavix) 75mg: (Inhibit ADP Binding to Platelet Receptors).
- Ticagrelor (Brilinta): Act Like Plavix But More Rapid & Effective.

5. Other Drugs Can Be Used:

1. K Channel Activator: Nicorandil (Vasodilator).
2. IF Channel Antagonist: Ivabradine (Decrease Heart Rate).
3. Ranolazine: Reducing Angina Symptoms.

❖ Interventional Treatment of Stable Angina (Revascularization):

1. Percutaneous Coronary Intervention (PCI):
Use **Balloon Dilatation** Then **Stent.**
It Relieve Symptoms But **Not Prolong Survival**.

Please Remember ➩

*** Post PCI** Give **Strong Anti-Platelets Drugs** to Decrease Risk of **Re-Stenosis**:
1. Statin Long Life.
2. Aspirin + Clopidogrel (**Plavix**) 75mg or Brilinta 90mg (**Ticagrelor)** For **12 Months** Then Continuo with Aspirin Only.

New Antithrombotic Drug: Monoclonal Ab **Abciximab** (Glycoprotein IIB/IIIA Antagonist).

Indications of PCI:
1. Failed Medical Treatment.
2. Single or Two Vessels Disease.
3. Stenosis After **CABG**.
4. Acute Myocardial Infarction as Primary Treatment of Choice (in 1st Two Hours).

Complications of PCI:
1. **Re-Occlusion** of the Vessel: Occur in **Third** of Patients & Prevented By **Stent**
2. **Spasm** of the Vessel Due to **Irritation** of Arterial Wall.

❖❖

2. Coronary Artery Bypass Graft (CABG):
Use Graft From **Great Saphenous Vein** or **Radial Artery** or **Internal Mammary Artery.**
It Relieve Symptoms & **Prolong Survival**.

➢ Grafting From Artery **Better** Than Vein; Because Stay **Longer**.

Please Remember ➩

*** Post CABG** Give:
1. Statin.
2. Aspirin + Clopidogrel (**Plavix**) 75mg or Brilinta 90mg (**Ticagrelor)** For **6 Months** Then Continuo with Aspirin Only. They All Improve Graft Patency.

New Antithrombotic Drug: Monoclonal Ab **Abciximab** (Glycoprotein IIB/IIIA Antagonist).

Indications of CABG:
1. Failed **PCI**.
2. All Three Coronary Arteries.
3. Left Main Coronary Artery.

Complications of CABG:
1. **Angina**: Early or Late.
2. **Infection**: After Surgery.

Post CABG Surgery:
90% of Patient Free From Angina For **1** Year
60% of Patient Free From Angina For **6** Years

50% of Vein Grafts Patent For **10** Years.
80% of Artery Grafts Patent For **10** Years.

➢ UNSTABLE ANGINA (CRESCENDO ANGINA) & ➢ PARTIAL THICKNESS MYOCARDIAL INFARCTION (NON ST ELEVATION MI = Non-STEMI):

Definition:	**Clinical Pictures:**
It is a Clinical Syndrome of **Chest Pain** Due to Myocardial **Ischemia** Caused By **Narrowing** of Coronary Artery Lumen Due to **Thrombus Formation**.	Sudden Onset **Retrosternal** Chest Pain On Minimal Exertion or Even at Rest, **Heaviness** in Nature, Radiating to the **Left Shoulder** & **Left Arm**, Lasting For **More Than 20 Minutes**.

Investigations:

1. 12 Leads Resting ECG: Show ST **Depression** and/or T-wave **Inversion**, **No** Pathological Q wave.
   Don't Forget ECG Findings Appear in **Two Third** of Cases & ECG Appear **Normal** in **One Third**.
2. Cardiac Enzyme Troponin I & CK MB: **Elevated** in **NSTEMI**, But **Not** in Unstable Angina.

Treatment:

It is an Emergency Case:

➢ Call For Help + Admission In Coronary Care Unit + Full Monitoring + Bed Rest.
Immediately Give the Patient **GTN** 500µg Sub-Lingual as **First Aid** + Serial **ECG** Every Half Hour.
If There is No Bradycardia; Metoprolol Can Be Given 50 -100mg Orally (Reduce **Arrhythmia** & Improve **Mortality**).
➢ Give Patient 4 Anti (Anti-Pain, Anti-Platelets, Anti-Coagulants, Anti Anginal Drugs):

Anti-Pain	**Anti-Platelets:**	**Anti-Coagulant:**	**Anti Anginal Drugs:**
(Analgesia): Morphine 10mg IV Or Diamorphine 5mg Given with Anti Emetic Reduce: **Blood Pressure**, **Infarction Size** & **Risk of Ventricular Arrhythmias**.	Aspirin 300mg Orally + Plavix 300mg Orally Given in 1st 12 Hours. Decrease Mortality Rate in 25% of Patients. Continuo Aspirin 75 + Plavix 75mg For 12 Moths.	IV Un Fractionated Heparin or LMW Heparin S/C (Best: **Enoxaprine**) or Fondaparinux 2.5mg Daily S/C 1. Prevent Thrombus to Increase. 2. Prevent Formation of New One. 3. ↓ Risk of Thrombo-Embolism. Continuo 8 Days or Until Discharge.	IV β- Blockers (Atenolol 5-10mg) + IV Nitrate (GTN 0.6-1.2mg/Hr) Calcium Channel Blockers (Nifidipine or Amlodipine) Given If β- Blockers are Contraindicated.

Please Remember
Don't Use **Thrombolytic Therapy** in Patients with: **Unstable Angina** & **Non STEMI**.

➢ After Stabilizing the Patient; Do **Risk Stratification** Via **Grace Score:**

In Case of Low Risk: (Risk of Death < 1%)
➢ Patient Should Do Stress ECG After 4-6 Weeks;
If Stress ECG Negative ➔ Good Prognosis.
If Stress ECG Positive ➔ Do **Coronary Angiography** Followed By **Revascularization** (PCI or CABG).

In Case of High Risk: (Risk of Death > 9%)
➢ Patient Should Do Early **Coronary Angiography** Followed By **Revascularization** (PCI or CABG) within First 48 Hours & Give Glycoprotein IIb/IIIa Receptor Antagonists (Abciximab) IV Infusion.

➢ FULLL THICKNESS MYOCARDIAL INFARCTION (ST ELEVATION MI = STEMI):

Definition:	Clinical Pictures:
It is a Clinical Syndrome of **Chest Pain** Due to Myocardial **Ischemia** Caused By **Persistent** & **Complete Occlusion** of **Coronary Artery**.	Same as Non-STEMI + Associated with: Dyspnea, Nausea, Vomiting, Sweating, Palpitation, Syncope, Epigastric Pain Lasting **More Than 30 Mint** ➢ Chest Pain May Be Asymptomatic in DM + Old Age

Investigations:

1. 12 Leads Resting ECG: (**Confirm Dx**) It Shows ST Elevation, Pathological Q wave, Left Bundle Branch Block.
Within 1st 12 Hours: ST Elevation, Within 1st Days: ST Elevation + Pathological Q Wave + Inverted T Wave, After Months or Years: Pathological Q Wave Only.
ECG Findings Appear in **Two Third** of Cases & Appear **Normal** in **One Third** ; So Normal ECG **Doesn't** Exclude STEMI
Myocardial Infarction May Occur in **Right Ventricle** Which Detected By ➔ **Right Sided ECG**.

2. Cardiac Enzyme (Troponin I/T & CK MB):
Troponin I: **Most Sensitive**, Release in 3-6 Hours, Reach the Peak in 36 Hours, Return Normal After 14 Days.
Creatinin Kinase MB (CK MB): Release in 3-6 Hours, Reach the Peak in 16-30 Hours, Return Normal in 2-3 Days.

3. Echocardiography: Normally Performed **Before Discharge** to Asses **Ventricular Function** & **Complications**.

4. Other Investigations: Chest X Ray, CBC (↑WBC), CRP + ESR (Elevated), Blood Sugar, Lipid Profile.

Treatment:

It is an Emergency Case:

➢ Call For Help + Admission In Coronary Care Unit + Full Monitoring + Bed Rest.
Immediately Give the Patient **GTN** 500µg Sub-Lingual as **First Aid** + Serial **ECG** Every Half Hour.
If There is No Bradycardia; Metoprolol Can Be Given 50 -100mg Orally (Reduce **Arrhythmia** & Improve **Mortality**).
➢ Give Patient 4 Anti (Anti-Pain, Anti-Platelets, Anti-Coagulants, Anti Anginal Drugs):

Anti-Pain (Analgesia):	Anti-Platelets:	Anti-Coagulant:	Anti Anginal Drugs:
Morphine 10mg IV Or Diamorphine 5mg Given with Anti Emetic. Reduce: **Blood Pressure**, **Infarction Size** & **Risk of Ventricular Arrhythmias**.	Aspirin 300mg Orally + Plavix 300mg Orally Given in 1st 12 Hours. Decrease Mortality Rate in 25% of Patients. Continuo Aspirin 75 + Plavix 75mg For 12 Moths.	IV Un Fractionated Heparin or LMW Heparin S/C (Best: **Enoxaprine**) or Fondaparinux 2.5mg Daily S/C. **1.** Prevent Thrombus to Increase. **2.** Prevent Formation of New One. **3.** ↓ Risk of Thrombo-Embolism. Continuo 8 Days or Until Discharge.	IV β- Blockers (Atenolol 5-10mg) + IV Nitrate (GTN 0.6-1.2mg/Hr) Calcium Channel Blockers (Nifidipine or Amlodipine) Given If β- Blockers are Contraindicated.

❖ Specific Treatment of STEMI (Reperfusion):

To Restore Coronary Patency, Improve Survival & Decrease Mortality (25%–50%) Which Include:

Primary Percutaneous Coronary Intervention:

Known as **(1ry PCI),** It's the **Treatment of Choice**.

Best Time Within **1st 2 Hours (120 Minutes)**.

Effective More Than Thrombolytic Therapy.

Decrease Mortality Rate By **50%.**

One Year Survival Rate is More Than **95%.**

Successful **PCI** May Associated with **Transient Arrhythmias**

If 1ry PCI Achieved; Give Glycoprotein IIb/IIIa Receptor Antagonists IV (Abciximab IV Bullos 0.25mg 10 Min Before PCI)

In Hospital Continuo S/C **LMW Heparin** or **Fondaparinux** & Oral **Aspirin** + **Plavix** 75mg, **β– Blockers**, and **ACE I** Therapy

Thrombolytic Therapy:

Done If 1ry PCI Cannot Achieved Within 1st 2 Hours of Diagnosis, Done in **1st 12 Hours**.

Thrombolytic Therapy Include:

1. **Alteplase**, **Tenecteplase** & **Reteplase**: Best Choices, Not Allergic.
2. **Streptokinase**: **Not Favor**, Allergic.

All Patients Who Have Received Successful Thrombolysis Should Be Considered For **Early Coronary Angiography** with a View to **Coronary Revascularization** (**PCI**) Within the **First 24 Hours**

Contraindications of Thrombolytics:

Absolute Contraindication:	**Relative Contraindications:**
1. Active Internal Bleeding.	1. Uncontrolled HTN. 2. Active Peptic Ulcer.
2. Previous Hemorrhagic Stroke.	3. Previous Ischemic Stroke. 4. Pregnancy.
3. Suspected Aortic Dissection.	5. Recent Surgery & Recent Trauma.

❖ Rehabilitation Post STEMI:

If There is No Complications; Patient Can Mobilize After **2nd Day**, Return Home After **2-3 Days**, Resume Car Driving After **1-4 Weeks**, Return in Work After **4 Weeks.**

❖ Secondary Prevention Post STEMI:

Non Pharmacological Prevention:	**Pharmacological Prevention:**
Control DM, HTN, Serum Cholesterol Stop Smoking, Regular Exercise, Decrease Weight, Increase Vegetables, Fruits and Omega 3.	1. Aspirin & Plavix or Aspirin & Brilinta For 1 Year, Then After Year Continuo with Aspirin Only Long Life. 2. ACE Inhibitors Long Life (↓ Risk of Remodeling, ↓ Risk of Heart Failure). 3. Statin (Atrovastatin or Simvastatin) For Long Life. 4. β–Blockers For at Least 2 Years: (↓ Risk of Remodeling).

Complications:

Immediate Complications:	**Early Complications:**	**Late Complications:**
1. **Arrhythmias**: Common But Often **Transient**, Include: Ventricular Fibrillation (in 5-10% of Patients), Ventricular Tachycardia, Ventricular Ectopic, Atrial Fibrillation & Atrial Flutter. 2. **Heart Block**. 3. **Cardiogenic Shock**.	1. **Early Pericarditis:** Occur **2nd-3rd Hour** Relieved By **Opiate** NSAIDs & Steroid **Not Given** (Because ↑Risk of Vent Aneurism) 2. **Acute Heart Failure** (Poor Prognosis) 3. **Left Ventricular Failure**. 4. **Acute Pulmonary Edema**. 5. **Cardiac Rupture:** Occur 2nd-5th Day Rupture of Wall, Ventricular Septum, Papillary Muscles.	1. **Late Pericarditis:** Also Known: **Dressler's Syndrome**; It is as **Autoimmune** Event Occur Within **Weeks** to **Months**, Relived By **NSAIDs** or **Steroid**. 2. **Thrombo-Embolism**. 3. **Myocardial Ischemia**. 4. **Ventricular Remodeling**. 5. **Ventricular Aneurysm** (10%): Characterized By **Persistent ST Elevation**.

➢ ACUTE RHEUMATIC FEVER:

Definition:

It is an **Immune Mediated** Delayed Inflammatory Disease That Result From **Recurrent Untreated Pharyngitis** & **Tonsillitis** By **Group A β-Hemolytic Streptococci**.
Can Affect Heart, **Joints**, **Brain & Skin**.
Develop After **2 – 3** Weeks From Infection
Usually Affect **Children** (**5 – 15 Years**).
It's

Pathophysiology:

Group A β-Hemolytic Streptococcus Has **M-Protein** That Look Similar to Some Proteins in the Heart, Joints, Brain and Skin,
Some Antibodies Synthesized Against Bacterial Antigens (M-proteins) May **Cross React** with of Heart Muscle Cause **Inflammation** of **All Heart Layers**,
Also Cause Inflammation in **Joint**, **Brain** & **Skin**.
Histologically Presence of **Aschoff's Nodules** **Only** in the **Heart** Seen in **Subacute** or **Chronic** Phase of Rheumatic Fever.

Please Remember ✍

Joints are the **Most Common** Site Affected By Rheumatic Fever ➔ **75%**.
While All Layers of Heart ➔ **60%**.
Basal Ganglia ➔ **30%**.
Skin ➔ **10%**.

Clinical Pictures:

First All Patients Have **Streptococcal Pharyngitis**, and Then After **2-3 Weeks** Patients Develops:
1. Chest Pain, Dyspnea & Palpitation (Due to **Pan-Carditis**)
2. Joint Pain with Redness & Swelling (Due to **Poly-Arthritis**)
3. **Erythema Marginatum**, **Sydenham's Chorea & Fever**

But Don't Forget It May Be **Asymptomatic**.

Pan-Carditis:

Its Incidence **Declines** with Increasing Age, Occur in 60% of Patients Include:
1. **Pericarditis:** Causing **Pericardial Effusion**.
2. **Myocarditis:** Causing **Heart Failure**.
3. **Endocarditis:** Involve Valves of the Heart, Causing Mainly **Valves Regurgitation**.

Affect Mainly: **Mitral** & **Aortic** (> 50%)
But Tricuspid & Pulmonary are Rare.

Poly-Arthritis:

Most Common Presentation (75% of Patients);
It is an **Acute**, **Painful**, **Migratory** (Fleeting), **Asymmetrical** Polyarthritis Affect Large Joints of Extremities (Knee, Ankle, Elbow & Wrist) Present with Redness, Swelling & Tenderness Between 1 Day & 4Weeks

Please Remember ✍
This Type of Arthritis Has **Dramatic Response** to **Salicylates (Aspirin)**,
If Not Improve to Aspirin; the Diagnosis is **Doubt**.

Sydenham's Chorea:

Known as ➔ **St Vitus Dance**.
It is **Purposeless**, **Involuntary** Movements of Face & Extremities Occur in **20-30%**, More in Female
It is **Late Neurological Manifestation** Due to Inflammation of Basal Ganglia.
It Appears **3-6** Months After Pharyngitis
Spontaneous Recovery Occur Within Few **Months**.

Erythrma Marginatum:

Non-Tender, **Non-Pruritic**, **Migratory**, **Redness**, with Raised Margin & Pale Center, Mainly On Trunk & Proximal Extremities, But **Not in Face** Occur in Less Than **5%**.

Sub-Cutaneous Nodules:

Firm, **Painless**, **Mobile Nodules** Common in Extensor Surface Appear After 3-6 Weeks of Pharyngitis, Occur in **5-7%**.

Investigations:

1. **Blood Workup: CBC** (↑WBC), **ESR** (Elevated) & **CRP** (Elevated).
2. **Throat Swab For Culture:** Positive in 10-25% of Patients Only.
3. **Serology (Best):** High Anti-Streptolysin-O (ASO) Antibody Titer (>200U in Adult & >300U in Children).
4. **ECG**: For Pericarditis & Heart Block.
5. **Echocardiography**: For Valve Lesions Mainly **Regurgitation** & For **Pericardial Effusion**.

Diagnosis:

By **Revised Jones Criteria** Which are:

- **Major Criteria:**

Poly-Arthtitis, Pan-Carditis.
Sydenham's Chorea, Erythema Marginatum.
Sub-Cutenious Nodules.

- **Minor Criteria:**

Arthralgia, Previous Rheumatic Fever.
Fever, Leukocytosis, High ESR or CRP.
First Degree Heart Block.

- **Evidence of Previous Streptococcal Infection:**

Positive Throat Swab Culture or High ASO Antibody Titer.

❖ **Rheumatic Fever Diagnosed Clinically in Presence of:**

Two Major Criteria + **Evidence** of Previous **Streptococcal** Infection or
One Major + **Two** Minor Criteria + **Evidence** of Previous **Streptococcal** Infection.

Treatment:

- **Bacterial Eradication By Using:**

Single Dose of Benzathine Benzyl Penicillin Injection 1.2 Million Unit IM,
Or Phenoxymethyl Penicillin Orally 250mg 4 Times Daily For 10 Days.
Should Be Given On Diagnosis to Eliminate Any Residual Streptococcal Infection.

- **Symptoms Eradication By Using:**

1. Aspirin For Poly-Arthritis (Response Within 24 Hours Which Help to Confirm Diagnosis), Dose is 100mgxKg/Day & Continuo Until ↓ESR
2. Steroid For Pan-Carditis or Severe Arthritis. Dose is 2mgxKg/Day & Continuo Until ↓ESR.
3. Antipyretics For Fever.

Please Remember

Recurrence of Infection Prevented By Long-Term Prophylactic Penicillin (Oral Phenoxymethyl Penicillin 250 mg Twice Daily or Benzathine Benzyl Penicillin 1.2 Million Unit IM Monthly), Which Given According to:
If Associated with Pan-Carditis with Valve Involvement; Given For 10 Years or Until Age Reach 40 Years Old.
If Associated with Pan-Carditis Without Valve Involvement; Given For 10 Years or Until Age Reach 21 Years Old.
If There is No Pan-Carditis & No Valve Involvement; Given For 5 Years or Until Age Reach 21 Years Old.

➢ CHRONIC RHEUMATIC FEVER

Common in **Female**, Occur in **50%** of Patients with **Rheumatic Fever** with **Pan-Carditis**, Causing **Valvular Heart Disease** Commonly Affect Mitral **(90%)** Then Aortic Then Tricuspid & Pulmonary Isolated Mitral Stenosis Account For About **25%** of All Cases, and an Additional **40%** Have Mixed Mitral Stenosis and Mitral Regurgitation.

➢ VALVULAR HEART DISEASES (VHD):

Mitral Stenosis:

Causes:

1. Rheumatic Fever (**Almost Always**).
2. Senile Degeneration (Heavy Calcification).
3. Congenital (**Rare**)
4. Others: Malignant Carcinoid Disease, SLE, RA.

Symptoms:

Symptoms Appear When Mitral Orifice is < 2cm

1. Fatigue & Cold Periphery (Due to ↓COP).
2. Palpitation (Due to Atrial Fibrillation).
3. Hemiparesis (Due to Stroke).
4. Dysphagia (Due to Compression of Esophagus).
5. Hoarseness of Voice (Due to Compression of Recurrent Laryngeal Nerve Known as **Orthner's Syndrome**).
6. Dyspnea, Orthopnea, PND, Cough with Heamoptysis & Wheeze (Due to Pulmonary Edema).
7. Bilateral LL Edema & Abdominal Distension (Due to Right Side Heart Failure).

Signs:

1. Irregular Pulse (Due to Atrial Fibrillation).
2. Bilateral Lungs Crackles (Due to Pulm Edema).
3. Ascites, Congested Tender Liver & ↑JVP (Due to Right Side Heart Failure).
4. Malar Flushing (Rash).
5. Loud S1 + Opening Snap + Mid Diastolic Murmur

Pathophysiology:

In **Mitral Stenosis** Passing of Blood From Left Atrium to Left Ventricle Depending On **Atrial Contraction**, Which After Long Time It Will Lead to **Left Atrial Dilatation & Hypertrophy**,
Long Time of Dilatation Causes **Atrial Fibrillation** That Leads to Stasis of Blood & Formation of **Thrombus** Which May Detached Causing **Stroke**,
Stasis of Blood in Lt Atrium Lead to **Increase of Pressure** in Lt Atrium Lead to Increase Pressure in Pulmonary Veins Then Pulmonary Capillaries Which Cause **Pulmonary Congestion**,
Long Standing Pulmonary Congestion Lead to **Pulmonary HTN** That Increase Pressure in Pulmonary Artery and Then Rt Ventricle Causing **Rt Ventricular Hypertrophy & Dilatation** Leading to **Right Side Heart Failure**.

Investigations:

1. Echocardiography (**Investigation of Choice**): Confirm Diagnosis and Asses Severity & Complications.
2. ECG: Atrial Fibrillation (Absent P-Wave) or Left Atrial Enlargement (P-Mitral).
3. Cardiac Catheterization: Screening For the Presence of Coexisting Coronary Artery Disease.
4. Chest X Ray (Pulmonary Edema, Lt Atrial Dilatation)

Treatment:

If Patient Asymptomatic: Just **Follow Up** By **Echocardiography** Every **6-12 Months**.
If Patient Has Mild Symptoms (Mild to Moderate Stenosis) ➔ **Medical Treatment**: **Diuretics** (to Control Pulmonary Edema), If Associated with Atrial Fibrillation Give **Anticoagulant** with **Digoxin** or **B-Blockers**
If Patient Has Severe Symptoms (Severe Stenosis): Do **Surgical Intervention** (Balloon Valvuloplasty, Valvotomy, Valve Replacement)

- **Balloon Valvuloplasty & Valvotomy** are Treatment of Choice in Case If: Isolated Mitral Stenosis, No (or Trivial) Mitral Regurgitation, Mobile & Non-Calcified Valve, Lt Atrium Free of Thrumbus.
- **Valve Replacement** is the Best Choice, There are Two Types Prostatic Valve:

Metallic Valve:	**Tissue Valve (Biological Valve):**
Stay Long Life.	Stay For 7-15 Years.
Need Anticoagulant (If Warfarin Target INR 2-3).	No Need For Anticoagulant.
Mostly Done For Young Patients.	Mostly For Old Patients.
Click Sound is Heard.	No Click Sound.

Mitral Regurgitation:

Causes:

1. Rheumatic Fever (**Most Common**).
2. Infective Endocarditis.
3. Mitral Valve Prolapse (Floppy Mitral Valve).
4. Post Myocardial Infarction & Cardiomyopathy.
5. Lt Ventricular Dilatation (Functional Regurge).

Symptoms:

1. Fatigue & Cold Periphery (Due to ↓COP).
2. Palpitation (Due to Atrial Fibrillation).
3. Hemiparesis (Due to Stroke).
4. Dysphagia (Due to Compression of Esophagus).
5. Hoarseness of Voice (Due to Compression of Recurrent Laryngeal Nerve Known as **Orthner's Syndrome**).
6. Dyspnea, Orthopnea, PND, Cough with Heamoptysis & Wheeze (Due to Pulmonary Edema).
7. Bilateral LL Edema & Abdominal Distension (Due to Right Side Heart Failure).

Signs:

1. Irregular Pulse (Due to Atrial Fibrillation).
2. Bilateral Lungs Crackles (Due to Pulm Edema).
3. Ascites, Congested Tender Liver & ↑JVP (Due to Right Side Heart Failure).
4. Displaced Apex Beat, Soft S1 + S3 + Apical Pan Systolic Murmur Radiate to the Axilla.

Pathophysiology:

During Contraction of Left Ventricle, Amount of Blood Will Return Back to Lt Atrium,
This Returned Blood Lead to **Left Atrial Dilatation & Little Increase of Pressure.**
Long Time of Dilatation Causes **Atrial Fibrillation** That Leads to Stasis of Blood & Formation of **Thrombus** Which May Detached Causing **Stroke**,
Stasis of Blood in Lt Atrium Lead to **Increase of Pressure** in Lt Atrium Lead to Increase Pressure in Pulmonary Veins Then Pulmonary Capillaries Which Cause **Pulmonary Congestion**,
Long Standing Pulmonary Congestion Lead to **Pulmonary HTN** That Increase Pressure in Pulmonary Artery and Then Rt Ventricle Causing **Rt Ventricular Hypertrophy & Dilatation** Leading to **Right Side Heart Failure**.

Investigations:

1. Echocardiography (**Investigation of Choice**): Confirm Diagnosis and Asses Severity & Complications.
2. ECG: Atrial Fibrillation (Absent P-Wave) or Left Atrial Enlargement (P-Mitral).
3. Cardiac Catheterization: Screening For the Presence of Coexisting Coronary Artery Disease.
4. Chest X Ray (Pulmonary Edema, Lt Atrial Dilatation).

Treatment:

If Mild Mitral Regurgitation: Just **Follow Up** By **Echocardiography** Every **6-12 Months**.
If Moderate to Severe Mitral Regurgitation ➔ **Medical Treatment:** Give **Diuretics** & **Vasodilators**, **Digoxin** & **Anticoagulant** Given in Atrial Fibrillation, Also **ACE I** or **ARBs** Given in Systemic HTN.
If Severe Mitral Regurgitation: **Surgical Intervention** (Mitral Valve Repair, Valve Replacement).

- **Valve Repair** By Inserting an Annuloplasty Ring, It is a Treatment of Choice For Functional Regurgitation
- **Valve Replacement** is the Best Choice, Done If:
  1. Failed Medical Treatment.
  2. Progressive Cardiomegaly.
  3. Decrease Left Ventricular Function.

Aortic Stenosis:

Causes:

1. Rheumatic Fever (**Most Common in Middle Age**).
2. Congenital (in Infants, Children & Adolescence).
3. Calcification of Bicuspid Valve (Young,Middle,Old)
4. Senile Degeneration (in Middle to Old Age).

Symptoms:

Patients Remain **Asymptomatic** For Many **Years** (Because Stenosis Develops **Slowly**),

Deteriorate Rapidly When Symptoms Develops.
The Symptoms Develops Due to **Exertion**
Which are ➔ **SAD P.S**

S: Syncope (Due to Markedly ↓ of COP).
A: Angina (Due to ↑Myocardial O2 Demand).
D: Dyspnea, (Due to Left Ventricular Failure).
P: Palpitation (Due to Arrhythmia).
S: Sudden Death (Due to Ventricular Tachycardia)

Cardinal Symptoms = SAD (Syncope, Angina, Dyspnea).

Signs:

1. Low Blood Pressure with **Narrow** Pulse Pressure.
2. Small Volume Pulse (**Palsus Parvus**).
3. Slow Rising Pulse.
4. Bilateral Lungs Crackles (Due to Pulm Edema).
5. Soft S2 + Harsh Ejection Systolic Murmur Radiate to the Neck.

Pathophysiology:

During Contraction of Left Ventricle, to Pump the Blood to the Aorta; There is a **Resistance** Through Aortic Valve, Due to **Narrowing of Aortic Orifice**, That Resistance with Time Will Lead to **Left Ventricular Hypertrophy**.
This Left Ventricular Hypertrophy Leads to **Increasing of O2 Demand of Myocardium**,
The Increasing of Myocardial Demand for O2 Lead to **Angina** (Angina with Normal Coronary Artery),
Fixed Outflow Narrowing Lead to **Limit Increasing of COP During Exertion**,
Limiting of COP During Exertion Cause **Exertional Syncope + Exertional Angina + Exertional Dyspnea & Sudden Death**.
Eventually Left Ventricle Will **Fail** (Due to Long-Standing Hypertrophy) Leading to **Pulmonary Edema**.

Investigations:

1. Echocardiography (**Investigation of Choice**): Confirm Diagnosis and Asses Severity & Complications.
2. ECG: Lt Ventricular Hypertrophy, Lt BBB, Down Sloping ST & T-Wave Inversion (Strain Pattern).
3. Chest X Ray: Enlarged Lt Ventricular (Boot-Shape Heart), Pulmonary Edema.
4. Cardiac Catheterization: Screening For the Presence of Coexisting Coronary Artery Disease.

Treatment:

If Patient Asymptomatic (Mild & Moderate Aortic Stenosis): Just **Follow Up** By **Echo** Every **1-2 Years**.
(In Older Patients & Heavy Calcified Aortic Valve Follow Up Echo Every 3-6 Months).

If Symptomatic Severe Aortic Stenosis: **Surgical Intervention** (Balloon Valvuloplasty, Valve Replacement, Tanscatheter Aortic Valve Implantation = TAVI).

- **Balloon Valvuloplasty** Mainly Done For Congenital Aortic Stenosis (But Not For Old Pts & Calcified Valve).
- **Tanscatheter Aortic Valve Implantation (TAVI)** Done For Older Patients & Pts Not Fit For Replacement.
- **Valve Replacement** is the Best Choice For All Severely Symptomatic Patients.

Aortic Regurgitation:

Causes:

1. Rheumatic Fever (**Most Common**).
2. Infective Endocarditis.
3. Dilatation of Aortic Root (Present in Case of: Aortic Dissection & Aneurysm, Aortic Syphilis, Marfan's Syndrome, Ankylosing Spodylitis).

Symptoms:

Patients May **Asymptomatic** or Just Have **Palpitation** Especially in Mild to Moderate Cases.
The Symptoms Develops in **Severe Cases** Which Include:

1. Uncomfortable Awareness of Heart Beat.
2. Dyspnea (But PND Sometimes the 1st Symptom)
3. Angina (Due to ↑Myocardial O2 Demand).
4. Head Nodding:
   (Due to Large Pulse of Carotid Artery).
5. **Lately** Symptoms of Pulmonary Edema (Orthopnea, PND, Cough with Heamoptysis & Wheeze)

Signs:

1. **Wide** Pulse Pressure (>100mmHg) with Low Diastolic Pressure (<60mmHg).
2. Large Volume Pulse: Collapsing Pulse.
3. Femoral Bruit (Pistol Shot): Duroziez's Sign.
4. Capillary Pulsation in Nail Beds: Quincke's Sign.
5. Head Nodding with Pulse: de Musset's Sign.
6. Displaced Apex Beat + S3 + S4 + Early Diastolic Murmur, Austin Flint Murmur (Soft Mid-Diastolic Murmur)

Pathophysiology:

Amount of Blood **Return** to From Aorta to Left Ventricle Which Cause **Increasing of Blood Volume** in **Left Ventricle**,

Then in Next Cycle Left Ventricle Bump **Large Volume of Blood to Entire Body** That Lead to **Increasing of COP 2 or 3 Times More Than Before**,
This High **COP** Lead to **Large Pulsation** of Arteries of the Body,

Recurrence of **Returning** the Blood From Aorta to Left Ventricle Lead to **Increasing of Left Ventricular Pressure**,
Raised Left Ventricular Pressure Lead to **Left Ventricular Dilatation** & **Hypertrophy**.
Eventually Left Ventricle Will **Fail** (Due to Long-Standing Hypertrophy) Leading to **Pulmonary Edema**.

Investigations:

1. Echocardiography (**Investigation of Choice**): Confirm Diagnosis and Asses Severity & Complications.
2. ECG: Lt Ventricular Hypertrophy, LT BBB, ST↓.
3. Cardiac MRI: For Aortic Dilatation.
4. Aortography: For Aortic Dilatation.
5. Cardiac Catheterization: Screening For the Presence of Coexisting Coronary Artery Disease.
6. Chest X Ray: Enlarged Lt Ventricular; (Boot Shape Heart), Pulmonary Edema.

Treatment:

If Patient Asymptomatic: Just **Follow Up** By **Echocardiography** Every **6-12 Months**.

If Patient Symptomatic or the Cause Was Syphilis, Infective Endocarditis ➔ **Surgical Intervention** By Doing:

- **Valve Replacement** (May Need to Be Combined with Aortic Root Replacement & Coronary Bypass Surgery).
  Aortic Root Replacement Usually Necessary If the Cause Was Marfan's Syndrome.
- **If Associated with Systemic HTN Calcium Channel Blockers Like Nifedipine Should Be Given.**

Tricuspid Regurgitation:

Causes:

1. Right Ventricular Dilatation (Functional Regurge)
2. Infective Endocarditis (Drug Abusers).
3. Rheumatic Fever.
4. Carcinoid Syndrome (Combined with Tricuspid Sten)
5. Congenital (Ebstien's Anomaly).

Pathophysiology:

During Contraction of Right Ventricle Amount of Blood Return to **Right Atrium**, Leading to **Right Atrium Dilatation** & **Systemic Congestion**,

During Diastole Large Volume of Blood Passes from Right Atrium to Right Ventricle, leading to **Right Ventricular Dilatation**, **Hypertrophy** & **Failure.**

Symptoms:

Bilateral LL Edema & Abdominal Distension.

Signs:

Ascites, Congested Tender Liver, ↑JVP & Prominent (Giant) **v**-Wave.
Pulsatile Liver During Systole (Epigastric Pulsation)
Displaced Apex Beat + Soft S1 + Pan Systolic Murmur

Investigations:

Echocardiography (**Investigation of Choice**).
ECG: Rt Ventricular Hypertrophy, Dilated Rt Atrium

Treatment:

If the Cause Rt Ventricular Dilatation: Treat Heart Failure (Patient Will Be Improved By Diuretics).
If the Cause Rheumatic Fever: Valve Replacement.

Tricuspid Stenosis:

Causes:

1. Rheumatic Fever (**Common Cause But Not Isolated**).
2. Carcinoid Syndrome (Combined with Tricuspid Regurg).
3. Congenital.

Clinical Pictures:

Bilateral LL Edema & Abdominal Distension (Ascites), Congested Tender Liver, ↑JVP & Prominent **a**-Wave.
Loud S1 + Mid Diastolic Murmur.

Investigations:

Echocardiography (**Investigation of Choice**).

Treatment:

If Isolated Severe Tricuspid Stenosis; Do: Valvotomy or Balloon Valvuloplasty.
If Associated with Other Valve Lesions; Do: Valve Replacement.

Pulmonary Stenosis:

Causes:

1. Congenital (Common): Fallot's Tetralogy.
2. Carcinoid Syndrome.

Clinical Pictures:

↑ JVP with Prominent **a**-Wave.
Ejection Systolic Murmur.

Investigations:

Echocardiography (**Investigation of Choice**).
ECG: Right Ventricular Hypertrophy.

Treatment:

If Isolated Severe Pulmonary Stenosis; Do: Valvotomy or Balloon Valvuloplasty.
If Associated with Other Valve Lesions; Do: Valve Replacement.

➢ INFECTIVE ENDOCARDITIS (IE):

Definition:

It is **Microbial Infection** Affect Endocardium, Cardiac Valves (Even Prosthetic Valve) & Congenital Anomaly.
Commonly Due to Strepto & Staph
More Common in **Male**, in Age > 50 Years.

Risk Factors:

1. Cardiac Diseases (Congenital or Acquired):
   VDS, PDA, Rheumatic Fever (ASD Low Risk).
2. IV Drug Abusers (Common in Rt Side Endoarditis).
3. Prosthetic Valves.
4. History of Myocardial Infarction.

Pathophysiology:

Endocardial Damage Occur Due to Abnormal Blood Flow (Due to Septal Defect) Which Act as a Site That Attract Deposits of Platelets & Fibrin Leading to **Platelets & Fibrin Aggregation** That are Vulnerable to Colonization By Organisms Leading to Formation of **Vegetations.**
Vegetation is Composed of Organisms, Fibrin and Platelets That Grow and May Become Large.

➢ **Fate of Those Vegetations:**

1. Valve Destruction (Valve Regurgitation).
2. Embolus Formation (Pulm Embolism, Stroke).
4. Abscess Formation (Mainly at Root of Aorta).
3. Immune-Complex Deposition (Vasculitis).

Types:

Acute Infective Endocarditis:

Occurs in **Healthy Heart**.
Caused By **Strong Organism**.

More Common with **IV Drug Abusers** & Patients with **Low Immunity**.

Sub-Acute Infective Endocarditis:

Occurs in **Diseased Heart** (VHD, Congenital).

Common at Site of **Pre-Existing Endocardial Damage: (at Site of Congenital Anomaly)**.

Causative Organisms:

Streptococci & Staphylococci:

Streptococcus Viridance:
Normal Flora of Upper Respiratory Tract,
Most Common Cause of **Sub-Acute Infective Endocarditis**

Staphylococcus Aureus:
Normal Flora of Skin, Affect Mainly **IV Drug Abusers**,
Most Common Cause of **Acute Infective Endocarditis**.

Staphylococcus Epidermidis (Coagulase Negative):
Most Common Cause of **Prosthetic Valve Endocarditis**,
Very Common at **First Year** of Surgery.

Others:

Coxiella Burnetti (Q-Fever):
Common in Patients with History of **Farm Animal Contact** (**Mostly Involve Aortic Valve**).

Brucella
Common in Patients with History of **Goats** & **Cattles** (**Mostly Involve Aortic Valve**).

Candida & Asperogillus (Low Immune Pts).

Rickettsia, Chlamydia, Klebsiella, Pseudomonas.

Clinical Pictures & Complications:

Sub-Acute Infective Endocarditis:

Suspected when a Patient with **Congenital** or **Valvular Heart Disease**, Include:

1. General Features:
Fever, Anorexia, Headache, Malaise (FAHM),
(Persistent Fever, Night Sweating, Weight Loss).

3. Embolisation Features:
Cerebral Vessels: Stroke, Brain Abscess.
Retinal Vessels: Sub-Conjunctival Hemorrhage and Blindness.
Peripheral Vessels: Acute Limb Pain.
Coronary Vessels: Retro-Sternal Chest Pain.
Pulmonary Vessels: Pulmonary Embolism Especially in IV Drug Abusers.
Mesentertic Vessels: Acute Abdominal Pain.
Renal Vessels: Non-Visible **Hematuria (Common)**.

2. Valve Destruction Features (**Regurgitation**):
Cardiac Murmurs.

4. Vasculitis Features:
Splinter Hemorrhage: Hemorrhage Under Nails.
Roth's Spots: Retinal Hemorrhage with Pale Center.
Osler's Nodule: Small Painful Intra-Cutaneous Nodules in the Tip of Fingers & Toes.
Glomerular Nephritis: Which Considered as a **Cause of Death** in Infective Endocarditis.

5. Finger Clubbing & Splenomegaly: Late Feature.
(Splenomegaly with Hepatomegaly Common in **Coxiella**)

Acute Infective Endocarditis:

Severe Fever, Cardiac Murmurs & Petechiae.
Embolic Events are Common, and Cardiac Failure or Renal Failure May Develop **Rapidly**.
If Partially Treated; It Will Behaves Like Sub-Acute Infective Endocarditis.
It Can Progress to **Death** Within **Weeks**.

Investigations:

1. Blood Culture (**Identify the Organism**):
Most Important One, Positive in Most of Cases,
At Least Take 3-6 Samples **Before** Giving Antibiotic,
Interval Between Samples 6 Hours or More,
First Two Samples Detect 90% of Organism.

Please Remember
Blood Culture May Negative If:
1. If Patient Taking Antibiotic Before Culture
2. If Organism Was Coxiella Burnetti & Candidal Infection.
Negative Blood Culture Doesn't Exclude Infective Endocarditis; So Need To Be Followed By Echocardiography.

2. Echocardiography (**Vegetations & Valve Lesion**):
Trans-Thoracic Echocardiography (TTE);
Initial Test, Sensitivity ➔ 65%.
Trans-Oseophageal Echocardiography (TOE)
Can Detect Small Vegetations, Sensitivity ➔ 90%.

3. Others:
ECG (May Show Heart Block Due to Aortic Root Abscess)
ESR & CRP (Elevated).
CBC (Leukocytosis, **Normoytic Normochromic Anemia**).
Urine Analysis For Glomerulonephritis,
(Show Proteinuria & Hematuria).
Chest X Ray May Show Evidence of Cardiac Failure & Cardiomegaly.

Diagnosis:

By **Modified Duke Criteria** Which are:

Major Criteria:

1. Positive Blood Culture:

- Typical Organisms From 2 Cultures.
- Persistent Positive Blood Cultures Taken > 12 Hours.
- Three or More Positive Cultures Taken Over > 1 Hour

2. Endocardial Involvement

- Positive Echocardiographic Findings of Vegetations.
- New Valve Regurgitation.

Minor Criteria:

1. IV Drug Abuser Patient.
2. Fever > **38.0**.
3. Embolic Feature.
4. Vasculitis Feature.
5. Blood Cultures Suggestive Organism Grown But Not Achieving Major Criteria.
6. Predisposing Valvular or Cardiac Abnormality.

➢ Infective Endocarditis Diagnosed in Presence of:

2 Major Criteria **or** 1 Major Criterion + 3 Minor Criteria **or** 5 Minor Criteria ➔ **Definitive Endocarditis**.
1 Major Criterion + 1 Minor Criterion **or** 3 Minor Criteria ➔ **Possible Endocarditis.**

Treatment:

1. Empirical Antibiotics:

According to Organism & Natural or Prosthetic Valve Started After Sample & Before Result (Empirical), Duration of Antibiotic Between 2-6 Weeks IV, Monitored By **Clinical Picture** & **CRP**.

Please Remember
Prophylactic Antibiotics Before Interventional Procedures (Dental, Genitourinary or Gastrointestinal Procedures) **No Longer Recommended,** But Considered Only For People With High Risk of **IE**.

Sub-Acute Infective Endocarditis:
Amoxicillin 2g IV 6 Times Daily +/- Gentamycin.

Acute Infective Endocarditis:
Vancomycin 1g IV Twice Daily + Gentamycin 1mgxKg Twice Daily.

Prosthetic Valve Endocarditis:
Vancomycin 1g IV Twice Daily + Gentamycin 1mgxKg Twice Daily + Rifampicin Orally 300-600mg Twice Daily.

2. Symptomatic Treatment (Antipyretics).

3. Surgical Intervention (Cardiac Surgery with Debridement & Valve Replacement) Indicated in:
1. Failure Medical Treatment. **2.** Heart Failure Due to Valve Damage. **3.** Prosthetic Valve Endocarditis.
4. Large Vegetation at Lt Side. **5.** Valve Abscess. **6.** Fungal Endocarditis. **7.** Previous Systemic Emboli.

➢ CARDIOMYOPATHIES

Hypertrophic Cardiomyopathy (HCM):	Dilated Cardiomyopath (DCM):	Restrictive Cardiomyopathy (RCM):
Definition: It is the **Most Common** Type of Cardiomyopathy & It is the **Most Common Cause of Death in Young Athletics**, Characterized By **Marked Left Vent- Hypertrophy** Which May: **Generalized**, **Septal**, **Apical**. Septal HCM Lead to Vent- Outflow Obstruction Known: **Hypertrophic Obstructive Cardiomyopathy (HOCM).** **Causes:** Genetic (Autosomal Dominant). **Symptoms:** Effort Related Symptoms: **SAD P.S** **S:** Syncope **A:** Angina **D:** Dyspnea **P:** Palpitation **S:** Sudden Death. **Signs:** Double Carotid Arterial Pulse Known: **Jerky Pulse**, Other Signs are Similar to Signs of Aortic Stenosis. **Investigations:** **1**.Echocardiography: **Diagnostic**. **2**.ECG: Lt Ventricular Hypertrophy. **Treatment:** Medications **Doesn't** Improve Prognosis **β**-Blockers, Verapamil, Disopyramide ↓ Symptoms & Prevent Syncope. Amiodarone For Arrhythmia. Digoxin & Vasodilators **Contraindicated**. Surgical Intervention; Partial Surgical Resection (Myectomy) or By Iatrogenic Infarction of Septum. **ICD** Done For High Risk.	**Definition:** Symmetrically **Dilatation** of **Left Ventricle** (± Right Ventricle) with **Poor Systolic Contractile Function,** (More Common in **Male**). Dilatation of the **Valve Rings** Can Lead to **Functional** Mitral and Tricuspid **Regurgitation**. **Causes:** **(I AM ASMA)** **I**diopathic (Most Common), **A**lcohol, **M**uscular Dystrophy, **A**utosomal Dominant (25%), **S**LE, **M**yocarditis (Viral-HIV), **A**cromegaly **Clinical Pictures:** Features of Congestive Heart Failure. Thrombo-Embolism, Chest Pain, Palpitation (Due to Arrhythmia), Sudden Death (Due to VT). **Investigations:** **1**.Echocardiography & MRI: **Diagnostic** **2**.ECG: Low Voltage, Lt BBB. **Treatment:** **1.** Treat Congestive Heart Failure. **2.** Anticoagulants. **3.** Ant Arrhythmic Drugs (B-Blockers) **4.** ACE I to Stop Progression. Surgical Intervention; **1.** **ICD** and/or **CRT** For High Risk. **2.** Heart Transplantation (**Definitive**).	**Definition:** It is a Rare Condition Characterized By **Myocardial Stiffness** Due to Fibrosis or Myocardial Infiltration Lead to **Impairment** of **Ventricular Filling** (Usually **Right Ventricle**) Which Cause Increase of Atrial Pressures Leading to **Atrial Hypertrophy** & **Dilatation** & Later Cause **Atrial Fibrillation** May Lead to **Pulmonary Embolism** **Causes:** **1.** Amyloidosis (**Most Common**). **2.** Hemochromatosis. **3.** Sarcoidosis. **4.** Idiopathic. **Clinical Pictures:** Features of Congestive Heart Failure. Thrombo-Embolism. Palpitation (Due to Arrhythmia). Sudden Death (Due to VT). **Investigations:** **1**.Echocardiography & Cardiac MRI are **Diagnostic**. **2**.ECG: Low Voltage, Lt BBB. **3**.Endomyocardial Biopsy (Rare). **Treatment:** Just **Symptomatic Treatment**; Diuretics, Nitrated, Calcium Channel Blockers, Anticoagulants ➢ **Because It is Poor Prognosis So Definitive Treatment is Cardiac Transplantation**.

➢ MYOCARDITIS

Self-Limiting Inflammation of Myocardium That Caused Mainly By **Viral Infection** (Coxsackie, Influenza A&B, SARS)
Other Causes: Bacterial (Mycoplasma Pneumonia), Drugs (Clozapin, Lithium), Autoimmune (SLE, RA, Systemic Sclerosis)
Presented as: Fulminant Myocarditis (Follow Viral Infection), Acute Myocarditis (Present with Heart Failure), Chronic Active Myocarditis & Chronic Persistent Myocarditis

➢ PERICARDIAL DISEASES

Acute Pericarditis:	Pericardial Effusion & Tamponade:	Chronic Constrictive Pericarditis:
Definition: It is an Acute Inflammation of Pericardium Which May Coexist with **Myocarditis**. **Causes:** **(VIRUS MTM)** **1.** Viral (Coxacie B) **Most Common**. **2.** Idiopathic (2nd Common Cause). **3.** Rheumatic Fever (3rd Common). **4.** Uremia. **5.** SLE. **6.** Myocardial Infarction. **7.** Tuberculosis. **8.** Malignancy. **Symptoms:** Acute **Central** Chest **Stabbing** Pain, Increase By Cough & Deep Breathing, Radiating to Shoulders Associated with Palpitation & Fever. **Signs:** Tachycardia. Muffled Heart Sounds. Pericardial Fraction Rub (**Diagnostic**) **Investigations:** **1.**ECG: ST Elevation in All Leads & PR Interval Depression (**Specific**). **2.**Chest X Ray: Pericardial Effusion. **3.**Echocardiography For Monitoring. **Treatment:** NASIDs (**Aspirin, Indomethacine, Colchicine For 3 Months**) Viral Pericarditis Resolve **Spontaneously** Within Few Days to Weeks. Steroid **No Longer** Recommended. **Complications:** **1.** Pericardial Effusion & Tamponade. **2.** Chronic Constrictive Pericarditis	**Definition:** **Pathological** Accumulation of Fluid or Blood In Pericardium. If Effusion Under Tension of Heart Called: **Pericardial Tamponade**. That Lead to Impaired Filling of Cardiac Chambers & Decrease of **COP**. **Causes:** **1.** All Causes of Pericardial Effusion. **2.** Pericardial Metastasis. **3.** Post MI (Due to Vent Rupture). **4.** Cardiac Trauma. **Symptoms:** Same Pericardial Effusion Symptoms. **Signs:** **1.** Same Pericardial Effusion Signs. **2.** Beck's Triad: Hypotension + ↑ JVP + Muffled or Absent Heart Sounds. **3.** **Pulsus Paradoxus**: Dropping of Systolic Blood Pressure During Inspiration **>10mmHg**. **4.**Kussmaul Sign: ↑JVP During Inspiration. **5.** Oliguria. **Investigations:** **1.**Echocardiography: **Diagnostic**. **2.**ECG: **Low Voltage** & Alternate QRS Amplitude (**Electrical Alternans**). Chest X Ray: Flask Shape Heart. **Treatment:** Mainly For Cardiac Tamponade: Immediate **Pericardial Aspiration** (**Pericardiocentesis**) Under Guide of **Echocardiography** + Give Antibiotic.	**Definition:** Diffuse, Progressive **Thickening**, **Fibrosis** & **Calcification** of Pericardium, Then the Heart Become **Encased** in a **Solid Shell** and **Can't** Fill Properly. The Calcification May Extend Into the Myocardium. **Causes:** **1.** Tuberculous Pericarditis (**Most Common Cause**). **2.** Connective Tissue Disease (**Rheumatoid Arthritis & SLE**). **3.** Viral Pericarditis. **Symptoms:** Fatigue, Fever, Features of Heart Failure (**Hall Mark** & **Mainly Rt Side**) Palpitation (Because AF is **Common**) Dyspnea is **Not** Predominant **Signs:** **1.** Signs of Rt Side Heart Failure. **2.** Kussmaul Sign: ↑JVP During Inspiration. **3.** **Pulsus Paradoxus**: Dropping of Systolic Blood Pressure During Inspiration **>10mmHg**. **Investigations:** **1.**Echocardiography (**Diagnostic**) **2.**ECG: Low Voltage. **3.**Chest X Ray & CT: Calcification. **4.**Cardiac Catheterization. **Treatment:** Loop Diuretics & Spironolactone. Surgical Resection (**Pericardiectomy**) Lead to **Dramatic Improvement**.

ATRIAL FIBRILLATION (AF):

Definition:

It is a **Most Common** Sustained Cardiac Arrhythmia Characterized By Multiple Interacting Re-Entry Circuit in Atrium Leading to **Spontaneous**, **Rapid**, and **Ineffective** Atrial Contraction.

Classification:

1. **Paroxysmal AF:** Intermittent Episodes Self-Terminate in 7 Days, Most Common 50%.
2. **Persistent AF:** Prolonged Episodes Terminated By Cardioversion.
3. **Permanent AF:** Prolonged Episodes Difficult to Terminate.

Causes:

1. Systemic Hypertension **(Most Common Cause)**.
2. Valve Heart Disease (Especially Mitral Stenosis)
3. Hyperthyroidism
4. Alcohol
5. Idiopathic (Lone AF)
6. Ischemic Heart Disease
7. Pericardial Disease
8. Cardiomyopathy
9. Pulmonary Embolism
10. Lung Disease.

Clinical Pictures:

- Could Be Asymptomatic (Especially in **Old Age** Patients)
- Palpitation. Dizziness. Dyspnea.
- Symptoms of Underling Cause.
- Thrombo-Embolic Symptoms: Stroke (15%), Renal Infarction, Limb Ischemia.
- Tachycardia with Irregular-Irregular Pulse.
- Absent a-Wave On JVP.
- Signs of Heart Failure.

Investigations:

1. **ECG**: Absent P-Wave, Irregular Rhythm (Irregular RR Interval).
2. **Echocardiography**: to Detect Thrombus.
3. **Thyroid Function Test**: For Thyrotoxicosis.

Treatment:

In Case of Paroxysmal AF: Usually Doesn't Require Treatment But ➔ **B-Blockers** are a Treatment of Choice.

In Case of Persistent & Permanent AF:

1. **Rate Control:** By Using One of **A, B, C, D: A**miodaron, **β**–Blockers, **C**alcium Channel Blockers (Verapamil & Diltiazem), **D**igoxin, (Permanent Pacemaker & Catheter Ablation Done in Poorly Controlled Rate AF).
(B-Blockers & Calcium Channel Blockers **More Effective** in Case of **Mitral Stenosis**).
2. **Restore Sinus Rhythm** By: Cardioversion (Chemical or Electrical By DC Shock): Immediate Cardioversion Done If Present in < 48 Hours, While If Present 48 Hours or More; DC Shock Should Be Avoided & Give Oral Anticoagulant For at Least 4 Weeks Then Do Cardioversion, Then Continuo Oral Anticoagulant For 3 Months.
3. **Prevent Recurrence** By Giving Amiodarone or Flecainide.
4. **Prevent Thromo-Embolism** (Mainly Risk of Stroke) **For All Types of AF** According to **CHA_2DS_2 VASc Score**:
If Score 1 ➔ Give Aspirin (in Male Give Anticoagulant).
If Score 2 or More ➔ Give Anticoagulant: Warfarin & Direct Acting Oral Anticoagulants (**DOACs**).
DOACs are Factor Xa Inhibitors **Rivaroxaban**, **Apixaban** & **Edoxaban**, & Direct Thrombin Inhibitor **Dabigatran**.
DOACs Replaced Warfarin For Stroke Prevention in AF Because They Have Low Risk For Intracranial Hemorrhage

Supraventricular Tachycardia (SVT):

The Term Supraventricular Tachycardia (SVT) Describes a Group of **Regular Tachycardias** That Have a Similar Appearance On ECG.
These are Usually **Narrow-Complex Tachycardias** and are Characterized By a Re-Entry Circuit Involving the Atria.

Types of SVT:

1. **Atrioventricular Nodal Re-Entrant Tachycardia (AVNRT)**
2. **Atrioventricular Re-Entrant Tachycardia (AVRT).**
3. **Atrial Tachycardia.**

➢ Atrioventricular Nodal Re-Entrant Tachycardia:

It is a Type of SVT Caused By Re-Entry in a Circuit Involving the AV Node Produces a **Regular Tachycardia** with a Rate of 120–240b/min, and the Episodes May Last From a Few **Seconds** to Many **Hours**.
Clinically Patient Develop **Rapid** Very **Forceful Regular** Heart Beat Associated with Chest Discomfort, Dyspnea & **Polyuria**.
ECG Shows: Tachycardia with **Normal** QRS Complex.
Treatment Not Usually Necessary But It Can Be Terminated By: Carotid Sinus Massage or Valsalva Maneuver.
In Acute Episode You Can Give: IV Adenosine (3-12mg Rapid Incremental Doses Until Tachycardia Stops) or Verapamil (5mg IV For 1 Minute), B-Blockers & Flecainide Can Be Used.
If Previous Drugs Failed; Do **Synchronized DC Cardioversion**.
Treatment of Choice is ➔ Catheter Ablation.

➢ Atrioventricular Re-Entrant Tachycardia:

In This Condition There is an **Abnormal Band** of Conducting Tissue That Connects the Atria and Ventricles.
This So-Called **Accessory Pathway** If This Pathway Conducts Impulses Antegrade Direction (From Atria to Ventricles)
This Will Lead to a **Premature Ventricular Activation** Causing **Shortening of PR Interval** & Produces a Deflection of QRS Complex Called a **Delta Wave**, the AV Node and Accessory Pathway Have Different Conduction Speeds Lead to Develop a Re-Entry Circuit Causing **Tachycardia,**
This is Known as **Wolff–Parkinson–White (WPW) Syndrome**.
Treated By **Carotid Sinus Massage** or **Valsalva Maneuver**.
Treatment of Choice is ➔ Catheter Ablation.
Prophylactic Treatment as **Flecainide** Use to Slow Conduction

Atrial Flutter:

Definition:

Large Re-Entry Circuit within **Right Atrium** Around **Tricuspid Annulus,**
Atrial Rate Around 300b/Min.
AV Node May Help to Decrease It to 150b/Min.

Causes:

1. Heart Failure.
2. Hypertension.
3. Ischemic Heart Disease.
4. Valve Heart Disease.
5. Cardiomyopathy.
6. Pericarditis & Myocarditis.

Symptoms & Complications:

- Palpitation. Dizziness. Dyspnea. **Polyuria.**
- Symptoms of Underling Cause.
- Thrombo-Embolic Symptoms.

Signs:

Tachycardia (Rate Reach 300 - 150Beat/Minute).

Investigations:

ECG: Multiple **Saw Teeth** P-Wave.

Treatment:

1. Rate Control: By Using One of A, B, C, D: Amiodaron, β–Blockers, Rate Limiting Calcium Channel Blockers, Digoxin.
2. Restore Sinus Rhythm By Using: Chemical or Electrical Cardioversion (DC Shock).
3. Prevent Recurrence By Giving Amiodarone. (**Flecainide is Contraindicated Here**)
4. Catheter Ablation (**Treatment of Choice**): Give More Than **90%** Chance of **Permanent Cure**.
5. Prevent Thromo-Embolism (Give **Anticoagulant**).

➢ VENTRICULAR ARRHYTHMIAS:

Ventricular Ectopic:	Ventricular Tachycardia:	Ventricular Fibrillation
It is an Extra Beat Arising From Ventricle Causing Irregularity Appearing of ECG. Also Known: **Ventricular Extrasystole**. **Causes:** **1.** Ischemic Heart Disease. **2.** Heart Failure. **3.** Cardiomyopathy. **4.** Digoxin Toxicity. **5.** May Physiological. **Investigations:** ECG Show: Wide & Bizzar QRS **Not Proceeded** By P- Wave. **Please Remember** ✍ Ventricular Ectopic Pictures: **1.** Bigemeny VE. **2.** Trigemeny VE. **3.** Couplet VE. **4.** Triplet VE. **5.** Quadriplet VE. **Treatment:** No Need For Treatment For **Asymptomatic** Patients. For Symptomatic Patient Give **β-Blockers** & Treat Underling Cause.	**Causes:** **1.** Ischemic Heart Disease (**Acute Myocardial Infarction Commonest Cause**). **2.** Heart Failure. **3.** Cardiomyopathies. **Types:** **1.** Monomorphic VT. **2.** Polymorphic VT. **Symptoms:** Palpitation, Dyspnea, Syncope (**Not Respond** to Carotid Massage or I.V Adenosine). **Investigations:** ECG Show: **1.** Wide QRS **Not Proceeded** By P- Wave. **2.** Tachycardia (180-240 b/mints). **3.** **Capture** or **Fusion Beats**. **4.** Prolonged QT > 0.4 Second (**Torsades de-Points**): It is a Form of Polymorphic VT Due to **Prolonged** Ventricular **Repolarization** Caused By: ↓K, ↓Mg, ↓Ca & Congenital. **Treatment:** If Systolic BP **Less** Than 90mmHg: Treatment of Choice Electrical Cardioversion (**Synchronized DC Shock**). If Systolic BP **More** Than 90mmHg: Give **Intravenous Bullous Amiodarone** Followed By Contentious Infusion. (Also IV Lidocaine Can Be Used) **Prevention:** **1.** **β**-Blockers with or Without Amiodarone. **2.** **ICD** Insertion (in Poor Vent Function). **Features Confirm VT:** **1.** History of Myocardial Infarction. **2.** Atrioventicular Dissociation + Capture/Fusion Beats (**Pathognomonic**) **3.** **Extreme** Left Axis Deviation. **4.** **Not Respond** to Carotid Massage.	Following Untreated Ventricular Tachycardia. Mainly Following **Polymorphic VT with Torsades de-Points**. **ECG:** Wide, Irregular & Bizzar QRS **Not Proceeded** By P- Wave. **Treatment:** Immediately DC Shock.

➢ HEART BLOCK (HB):

Also Called➔ **ATRIO-VENTRICULAR BLOCK:** It is a Disease of AV Node.

First Degree Heart Block:	Second Degree Heart Block:	Third Degree Heart Block:
It is a Condition in Which AV Conduction is **Delayed** & PR Interval is **Prolonged** (> 0.20 Second). It Rarely Causes Symptoms and **Does Not** Usually Require Treatment **Causes:** **(DR MISC)** **1. D**rugs (β-Blockers, Digixin). **2. R**heumatic Fever. **3. M**yocardial Infarction. **4. I**nfective Endocarditis. **5. S**leep & Athletics (Physiological). **6. C**ongenital. **ECG Show:** **1.** **Fixed** Prolonged **P-R** Interval. **2. P-**Wave Followed By **QRS**. **3.** Narrow **QRS**. **Treatment:** **No Need** For Treatment Except in Symptomatic Patients: Give IV Atropin, (0.6mg Repeated Doses).	Here **Dropped Beats** Occur Because Some Impulses From the Atria **Fail** to Conduct to the Ventricles. **Two Types:** **1. Mobtiz Type I (Wenkebach):** **Causes:** Same as First Degree Block Causes. **ECG Show:** **1.** **Progressive** Prolonged **P-R** Interval. **2.** There is **Drop P-wave** (P- Wave Not Followed By **QRS**). **Treatment:** No Need for Treatment Except in Symptomatic Patients: Give IV Atropin (0.6mg Repeated Doses). **If Failed:** Insert **Temporary Pace Maker** (Most of Cases Block Resolve in 7-10 Days). ❖❖❖❖❖❖❖❖❖❖❖❖❖❖❖❖❖❖❖ **2. Mobtiz Type II:** **Causes:** Same as First Degree Block Causes Except, **Mobtiz II** is **Not Physiological** **ECG Show:** **1.** **Fixed** Prolonged P-R Interval. **2.** There is Drop P-Wave; (**P-Wave Not Followed By QRS**). **3.** Alternate P-Waves are Not Conducted (Called 2:1) May Appear. **Treatment:** Symptomatic or Asymptomatic: Insert **Permanent Pace Maker** as Soon as Possible.	**No Impulse** Conducted From Atrium to Ventricle. (**No Relation** Between Atrial & Ventricular Contraction). = Atrio-Ventricular Dissociation. Ventricular Activity Maintained By **Purkinje System**. **Causes:** **1.**Myocardial Infarction or Ischemia; (**Most Common Cause**). **2.**Infective Endocarditis. **3.**Drugs (β-Blockers, Digoxin). **4.**Idiopathic Fibrosis, Sarcoidosis **5.**Congenital. **ECG Show:** **1.** Atrio-Ventricular Dissociation (**No Relation Between P & QRS**). **2.** Regular **P-P** & **P-R** Interval. **3.** Wide **QRS**. **Clinical Pictures:** **1.** Sever Symptomatic **Bradycardia** Characterized by **Regular** Heart Rate of 25-50 b/mint. **2.** **Cannon Waves** May Seen in **Neck**. **3.** Stokes-Adams Attack: Sudden Loss of Consciousness Due to Transient Asystole. **4.** Brief Anoxic Seizure Due to Cerebral Ischemia Characterized By Rapid Recovery (**Not Epilepsy**). **Treatment:** If Caused By Inferior MI: Give IV Atropin, (0.6mg Repeated Doses). **If Failed:** Insert **Temporary Pace Maker** (Most of Cases Block Resolve in 7-10 Days) If Caused By Anterior MI: Insert **Permanent Pace Maker**

ANTI-ARRHYTHMIC DRUGS:

Drugs:	Main Uses:	Route:	Side Effects:
Class Ia: **Disopyramide**	Prevention & Treatment of **Atrial** and **Ventricular Tachyarrhythmias**.	IV	Myocardial Depression, Hypotension, Dry Mouth & Urinary Retention.
Class Ib: **Lidocaine**	Treatment & Short-Term Prevention of **VT** and **VF**.	IV Oral	Myocardial Depression, Delirium & Convulsion.
Class Ic: **Flecainide**	Prevention & Treatment of **Atrial** and **Ventricular Tachyarrhythmias**.	IV Oral	Myocardial Depression & Dizziness.
Class II: **B-Blockers** Atenolol, Bisprolol & Metoprolol	Treatment and Prevention of **SVT** and **AF** Prevention of **VEs** and **Exercise-Induced VF**.	IV Oral	Myocardial Depression, Bradycardia, Bronchospasm, Fatigue, Depression, Nightmares & Cold Periphery.
Class III: **Amiodarone**	Serious & Resistant **Atrial** and **Ventricular Tachyarrhythmias**.	IV Oral	Photosentivity, Thyroid Dysfunction, Hepatotoxicity, Peripheral Neuropathy, **Torsades de Pointes**, Potentiates Digoxin & Warfarin.
Class IV: **Verapamil**	Treatment of **SVT**. Control of **AF**.	IV Oral	Myocardial Depression, Hypotension, Bradyardia & Constipation.
Others: **Digoxin**	Rate Control of **AF**.	IV Oral	Gastrointestinal Disturbance, Xanthopsia & Arrhythmias.
Adenosine	Treatment of **SVT**.	IV	Flushing, Dyspnea, Chest Pain, & Avoid in Asthma.