

# LECTURE NOTES OF INTERNAL MEDICINE

## Cardiology



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# CARDIOLOGY

## ↪ Anatomy of the heart:

- ♥ The heart is composed of four chambers, two atria and two ventricles. The atria are low pressure capacitance chambers mainly to store blood during ventricular systole and then fill the ventricles with blood during ventricular diastole. The ventricles are high pressure chambers responsible for pumping blood through the lungs and to the peripheral tissues.
- ♥ The most anterior chamber is the right ventricle and the most posterior chamber is the left atrium. The normal heart in chest X ray occupies  $< 50\%$  of the transthoracic diameter. The left border is formed by aortic knuckle, pulmonary trunk, left atrium and left ventricle (from above downward), the right border is formed by the right atrium joined by SVC (from above downward).

## ↪ Coronary circulation:

- ♥ The left main and right coronary arteries (branches from the aorta) arise from the left and right coronary sinuses just distal to the aortic valve.
- ♥ The left coronary gives:



- ◆ Left anterior descending artery, supplies anterior left ventricle, apex and the anterior part of septum.
- ◆ Left circumflex artery, supplies the left atrium and the lateral aspect of left ventricle, (marginal branches).

- ♥ The right coronary gives branches to supply the right atrium, right ventricle, inferior and posterior aspects of left ventricle.

## ↪ Nerve supply of the heart:

- ♥ **Sympathetic:** Supplies atria & ventricles, B<sub>1</sub>-receptors predominate in the heart with positive inotropic and chronotropic effects.
- ♥ **Parasympathetic:** Supplies atria only (vagal escape), cholinergic supply from the vagus supplies the atria via muscarinic receptors, under basal conditions vagal inhibitory effects predominate resulting in slow heart rate.

Cardiac symptoms, examination, ECG and X ray, see the practical parts.



## ↳ Other investigations:

### 1. Echocardiography:

It is similar to other forms to ultrasound imaging to study blood flow, heart structures and the movement of the valves and myocardium.

#### ↳ Value:

- ♥ Assessment of chambers pressure and size.
- ♥ Diagnosis of valve diseases (stenosis - regurge), infective endocarditis (vegetations).
- ♥ Detection of calcification of the valves.
- ♥ Detection of pulmonary and aortic pressure.
- ♥ Diagnosis of pericardial effusion.
- ♥ Diagnosis of cardiomyopathy, septal defects, aortic aneurysm and dissection.

♥ Measures COP, ejection fraction =  $\frac{\text{Stroke Volume}}{\text{End diastolic volume}} = 55-75\%$ .

- ♥ Evaluates the function of artificial valves.
- ♥ Echo doppler detects abnormal direction of blood, blood velocity and pressure gradient across valves.
- ♥ Dobutamine echo for ischemic heart disease, trans-esophageal echo (see later).

### 2. ECG with effort and ambulatory ECG (Holter): See later.

### 3. Cardiac Catheter:

A catheter is inserted into a vein or artery and advanced into the heart under radiographic fluoroscopic guidance.

#### ↳ Value: (Now, the main value is coronary angiography)

- ♥ Measure Pressure: Chambers pressure, Gradient across valves.
- ♥ Measure O<sub>2</sub>: Left side O<sub>2</sub>↑, Right side O<sub>2</sub>↓.
- ♥ Pass through anomaly e.g. VSD, ASD, PDA.
- ♥ Injection of dye showing normal or abnormal pathways e.g. ASD, VSD, PDA.

### 4. Cardiac scan:

Radioisotope is injected IV → Circulation → Gamma camera detects the distribution of radioactivity within the heart.

#### ↳ Value:

- ♥ The Gamma camera detects the amount of isotope emitting blood in the heart during cardiac cycle and can assess the size and function of the heart.
- ♥ Also Gamma camera can detect isotope uptake by the myocardium immediately after injection and with exercise to differentiate between ischaemic areas from non ischaemic areas. Thallium 201 and technetium 99 m are the most used isotopes.

In patients unable to exercise, the heart can be stressed with drugs e.g dipyridamole or dobutamine.



# HEART FAILURE

## Definition:

Failure of the heart to pump sufficient cardiac output to meet the demands of the body, with tissue hypoxia in spite of normal venous return and venous inflow to the heart (normal filling of the heart), this usually occurs with failure of the compensatory mechanisms.

## Causes:

### 1. Left Sided failure:

- **Pressure load:**
  - ➔ Aortic stenosis – systemic hypertension.
  - ➔ Coarctation of aorta.
- **Volume load:**
  - ➔ Mitral incompetence - Aortic incompetence.
  - ➔ Ventricular septal defect.
- **Muscle disease:** Cardiomyopathy, diagnosed by exclusion, confirmed by echo.
- **Ischaemic heart disease.**

### 2. Right sided failure:

- **Pressure load:**
  - ➔ Pulmonary hypertension
    - ➔ Primary P++.
    - ➔ Cor pulmonale.
    - ➔ Secondary to left sided lesion e.g M.S or left failure.
  - ➔ Pulmonary stenosis.
  - ➔ Pulmonary embolism.
- **Volume load:** Atrial septal defect - Tricuspid regurge.
- **Muscle disease:** (Cardiomyopathy.)
- **Ischaemic heart disease.**

- The most frequent cause of right heart failure is secondary to left heart lesion. e.g mitral stenosis or left heart failure.
- Ventricular inflow obstruction can be caused by MS, TS and constrictive pericarditis, so these lesions give picture of heart failure .

## Compensatory mechanisms:

When there is gradual impairment of cardiac function, (i.e in chronic heart diseases) a variety of compensatory changes may take place.

👉 **Aim:** To maintain normal cardiac output.

i.e When the heart is subjected to any load → stimulation of compensatory mechanisms. as below to maintain sufficient COP.



**① Hypertrophy:** "with pressure load"

i.e Increased thickness of cardiac muscle fibers  $\xrightarrow{\text{Late}}$  Ischaemic heart disease.

**② Dilatation:** "with volume load"  $\rightarrow$  increased length of cardiac muscle fibers.

**Starling law:**

**The Force of contraction  $\propto$  initial length of cardiac muscle fiber within limits.**

**③ Increased O<sub>2</sub> extraction**

i.e O<sub>2</sub> dissociation curve shifts to the right  $\rightarrow$   $\uparrow$  O<sub>2</sub> delivery to the tissues.

**④ Tachycardia:** (Sympathetic drive)

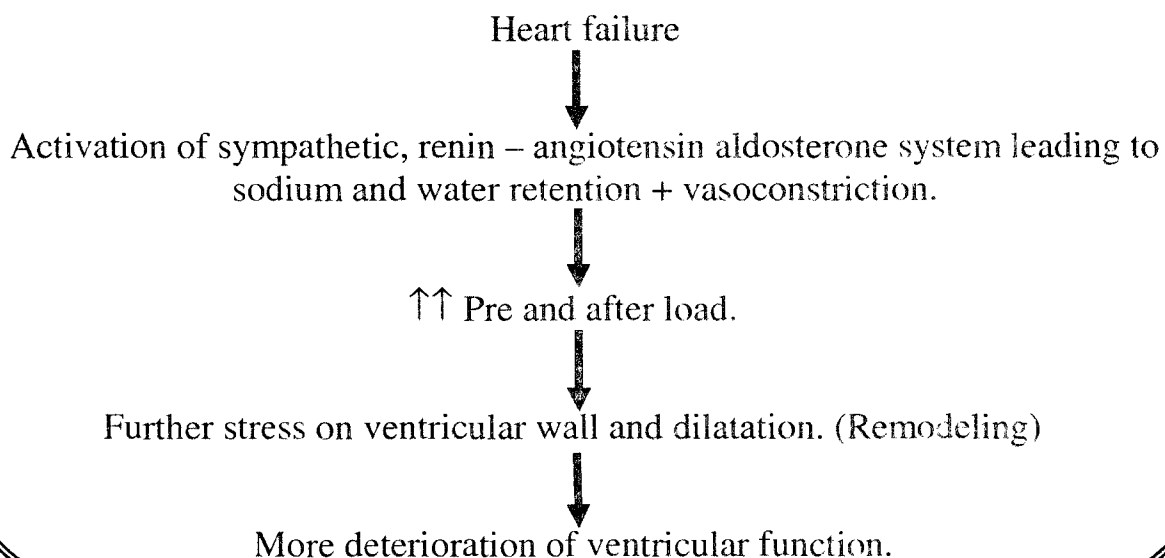
The COP = stroke volume  $\times$  heart rate,

heart failure  $\rightarrow$   $\downarrow$  stroke volume so, this leads to reflex tachycardia to maintain normal COP.

**⑤ Release of atrial nautreic peptide.**

**⑥ Activation of the renin angiotensin aldosterone system.**

**Summery of compensatory mechanisms:**



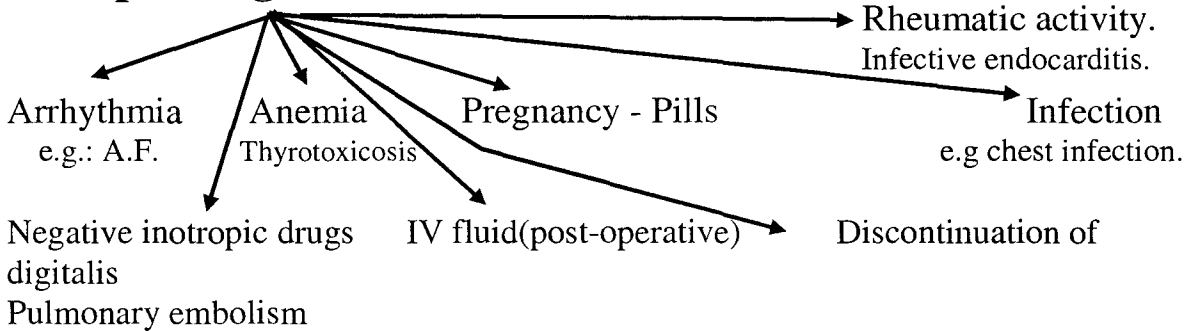
In ventricular remodeling there are changes in the size, mass and configuration of the ventricle as a consequence of hemodynamic changes triggered by myocyte growth, interstitial fibrosis, ischemia and apoptosis  $\rightarrow$   $\downarrow$  the effectiveness of ejection . Mediators that lead to profressive remodeling are angiotensin II, CA, TNF, growth hormone, while counter regulatory mediators are ANP, NO, Bradykinin. ACE inhibitors are helpful drugs to reduce the process of remodeling.



# ↳ Precipitating factors: (Aggravating factors of chronic heart disease)

Example: Patient with MVD  $\xrightarrow{\text{PPT. Factor e.g.: chest infection}}$  decompensated heart.  
 "With compensated heart" or AF (heart failure)

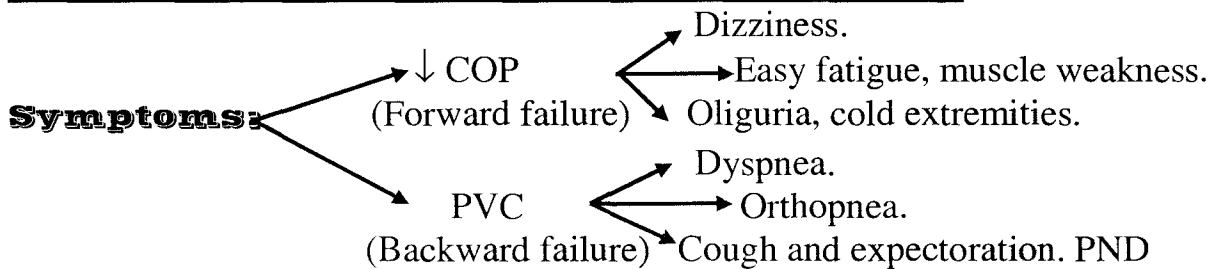
## Precipitating factors:



**Acute left heart failure** may be de novo left failure e.g in cases of acute myocardial infarction or acute left failure on top of chronic heart disease with precipitating or aggravating factors e.g atrial fibrillation on top of MVD. **Chronic heart failure** occurs in progressive chronic rheumatic, congenital heart disease or ischemic heart disease, Compensatory mechanisms are usually occur, so with failure of compensatory mechanisms → heart failure.

# ↳ Clinical Picture of chronic heart failure:

## (A) C/P of chronic left sided heart failure:

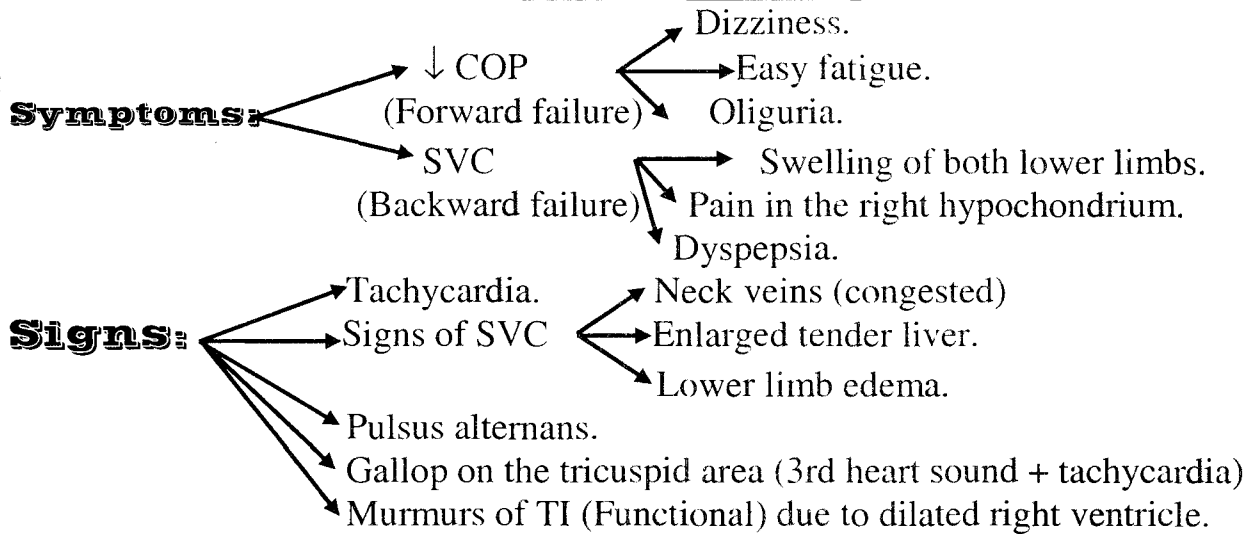


- Signs:**
- ① Tachycardia except in digitalized patient.
  - ② Signs of ↓COP
    - ↳ Low pulse volume.
    - ↳ ↓ Systolic blood pressure.
    - ↳ Cold extremities and peripheral cyanosis
  - ③ Signs of PVC (bilateral fine basal crepitations)
  - ④ Pulsus alternans.
  - ⑤ Gallop on the apex (3rd heart sound+ tachycardia= ventricular gallop)
  - ⑥ Murmur of MI. (MI may be a cause of heart failure or a result due to left ventricular dilatation).

- The term congestive heart failure is best restricted to cases where right heart failure results from pre-existing left heart failure.



**(B) C/P of chronic right sided failure:**



**Complications of heart failure**

- ☹ Uremia (Prerenal failure) ☹ Hypokalemia (diuretics and ↑ aldosterone).
- ☹ Hyponatremia (diuretics). ☹ Impaired liver function (↓COP + SVC).
- ☹ Thromboembolism. ☹ Arrhythmias. ☹ Cardiac cachexia.

**Cardiac cachexia:** (loss of lean (non edematous) body mass).

Chronic heart failure is sometimes associated with marked weight loss caused by a combination of anorexia and impaired absorption due to gastrointestinal congestion, poor tissue perfusion due to ↓ COP and skeletal muscle atrophy due to immobility. Also increased circulating levels of tumour necrosis factor have been found in patients with cardiac cachexia.

**Investigations:** (diagnosis of heart failure is mainly clinical)

1. **X-ray:** Cardiomegally (dilated heart), Left sided failure (PVC)
2. **ECG:**
  - It records electrical activity of the heart & not the mechanics. (No specific findings for heart failure).
  - It detects chamber enlargement, tachycardia or ischaemia.
3. **Echo:**
  - Measures COP, this reflects ventricular function.
  - Measurement of ejection fraction =  $\frac{\text{Stroke Volume}}{\text{End diastolic volume}}$   
It is an accurate assessment of ventricular function, if < 40-45% = systolic dysfunction.
4. **Cardiac scan.**
5. **Other investigations:** e.g.: serum creatinine, blood urea, serum Na and K, Hb and liver enzymes, bilirubin.
6. **Natriuretic peptide:** Normal level can exclude heart failure !?





# ↪ Treatment of chronic heart failure:

## 1. Rest:

- ☆ Rest until clinical improvement.
- ☆ Rest increases renal blood flow and help diuresis.

### Q. Complications ➔ DVT.

- of prolonged rest:
- ➔ Pulmonary embolism.
  - ➔ Constipation, osteoporosis.

## 2. Diet and other measures:

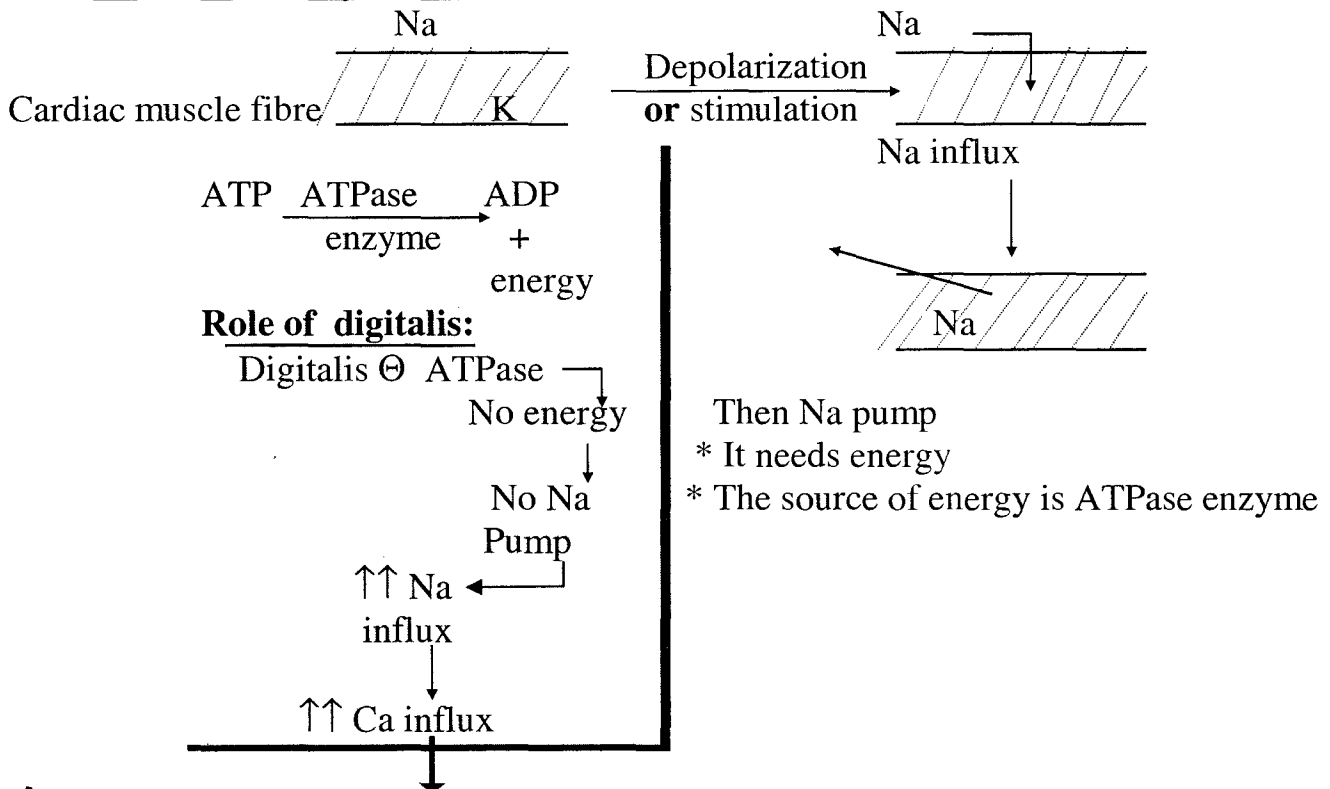
- ☆ Salt restriction (sodium intake about 2 gm/d).  
KCl is a salt containing no sodium.
- ☆ Fluid restriction: "**Fluid chart**" ➔ to avoid volume overload, with monitoring of urine output, also to avoid hyponatremia.

Required fluid = 500 ml + the volume of urine output in the previous day.

- ☆ Avoid heavy meals, avoid alcohol as it has a negative inotropic effect.
- ☆ Weight reduction in obese patients to reduce the cardiac load.
- ☆ It is better to give Influenza and Pneumococcal vaccine, stop smoking.

## 3. Digitalis:

### Mechanism of action:



- ☆ Increase muscle contraction by sliding of actin on myosin.
- ☆ K Inhibits the action of digitalis on ATPase. So, ↓ K → digitalis toxicity. also, we use K in treatment of digitalis toxicity.



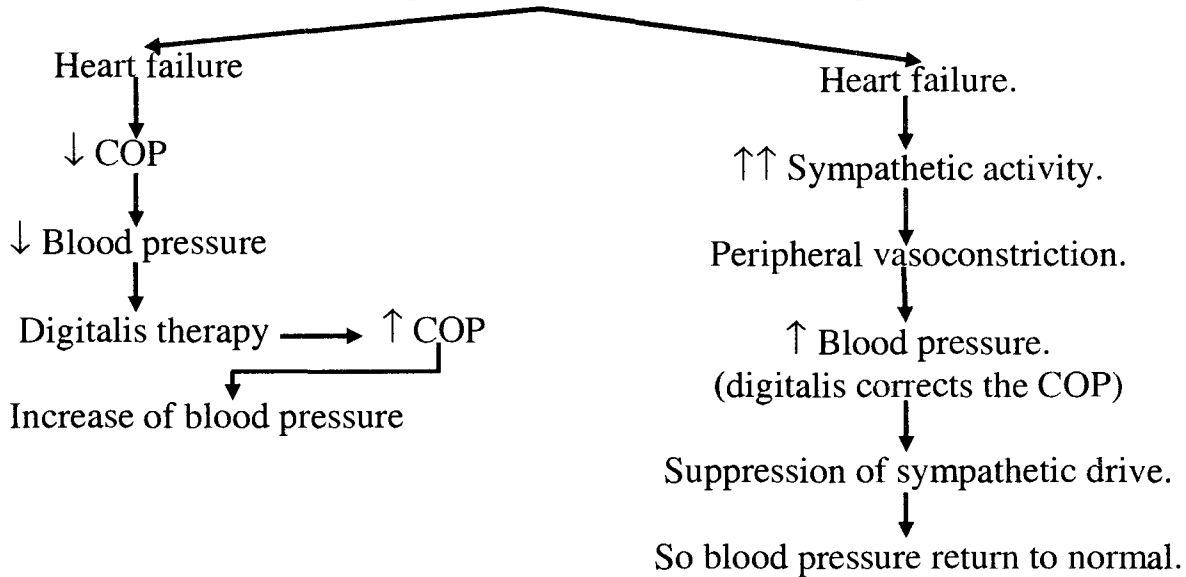
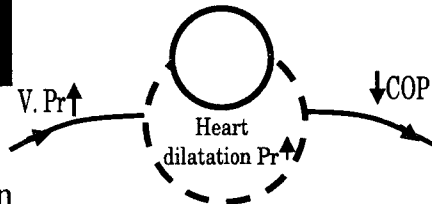
**Pharmacological actions:** **Electrical actions**  
**Mechanical actions**

## Electrical actions:

The heart rate is decreased due to A-V block. Digitalis is also excitatory on atria and ventricles !?.

## Mechanical actions:

- ↑ Contraction of the ventricles.
- ↑ COP.
- ↓ Size of the heart i.e heart dilatation
- ↓ Venous pressure (shift of blood from venous to arterial side).
- Improvement of coronary supply secondary to ↓↓ heart rate.
- Effect on blood pressure (It normalizes blood pressure)



**Uses:**

- ① Heart failure, the use of digitalis is essential with associated AF.
- ② Arrhythmias:
  - Atrial fibrillation.
  - Atrial flutter.
  - Supraventricular tachycardia.

**Dose:**

### Digoxin:

- ★ 85 % excreted in the urine, 15 % through biliary excretion.
- ★ Therapeutic level will be achieved after 5 days of daily maintenance therapy (cumulative method).

**Cumulative method:** (maintenance dose from the start)

- ★ 0.125 - 0.25 mg / day.      ★ Response after about 5 days.
- ★ Tablet = 0.25 mg (lanoxin or cardixin).

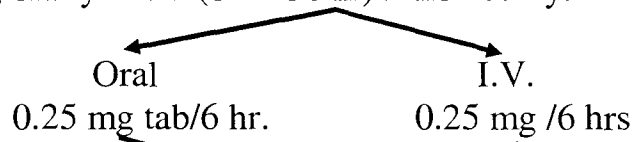


**👉 Rapid digitalization:** (loading method)

**Example:**

Loading dose is about 1-1.5 mg over 24 hours

Give 0.25 – 0.5 mg orally or IV (over 30 m) followed by:



Then 0.125 – 0.25 mg/day as maintenance dose.

**💣 Q. Indications of IV digitalization:**

- Severe left ventricular failure.
  - Heart failure with rapid atrial tachyarrhythmia (aggravating factor)
    - Rapid AF
    - Supraventricular tachycardia
- This is to get the benefit of A-V block also.

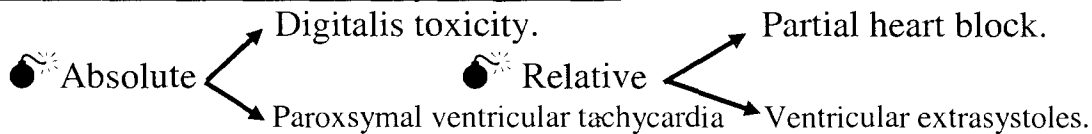
**👉 Digitoxin:**

Half life is 5 days, metabolized mainly in the liver, only 15 % excreted in the kidney, to reach steady state it must be taken for about 3 weeks.

**👉 Quabain:**

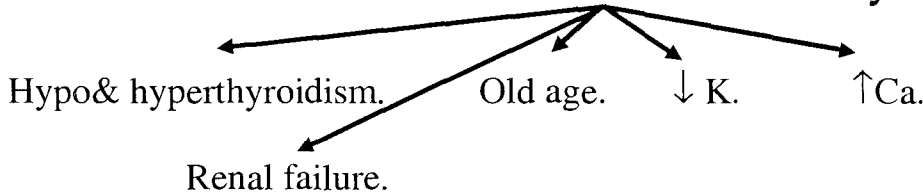
It is rapidly acting, onset of action 5-10 minutes, peak 60 minutes after IV injection, excreted through the kidney.

**📝 Contraindications of digitalis:**



**💀 DIGITALIS TOXICITY:**

**💀 Patients liable to toxicity**



**💀 To avoid toxicity:**

- ↳ Decrease the dose (give half the dose)
- ↳ Drug holiday.

- Digitalis may lead to arrhythmias in cases of ischaemic heart disease, hyperthyroidism and myocarditis.
- The effect of digitalis in cases of cor pulmonale is poor !?



☠ **Clinical picture:**

- ☒ Vomiting, diarrhea.
- ☒ Arrhythmia (e.g extrasystole) specially bigeminy, ventricular tachycardia and heart block (see ECG).
- ☒ Blurring of vision, altered colour vision (xanthopsia).
- ☒ ECG → arrhythmia, digitalis effect (sagging of S-T segment)

☠ **Diagnostic:** The desired therapeutic serum level is 0.5 – 2 ng/ml, it is > 2ng/ml in case of digitalis toxicity.

☠ **Treatment:**

- ⇒ Stop digitalis.
- ⇒ Give K.
- ⇒ Digitalis Ab: Fab fragments of digitalis Ab → Fab fragments digitalis complex → excreted through the kidney.
- ⇒ Haemofusion → adsorption of digitalis
- ⇒ Stop diuretics.
- ⇒ Treatment of arrhythmia. (See later)

## 4. Diuretics:

**Aim:**

Sodium & water excretion.

Decrease Sodium retention.      Fluid loss with reduction of heart load.  
Decrease venous pressure, this leads to relief of PVC & SVC.

### Furosemide:

- Acts on loop of Henle:
- \* It is a venodilator of pulmonary veins.
- \* ↓↓ PVC and SVC.
- \* Dose: ( 40-160 mg/d ) oral or injection.
  - Tablet 40 mg
  - Amp: 20 mg , 40 mg IV, I.M.

😊 **High ceiling loop diuretics:**

- ☒ Bumetanide (burinex).
- ☒ Furosemide.
- ☒ Ethacrynic acid.

High ceiling diuretics i.e. their action increases with increase of the dose.

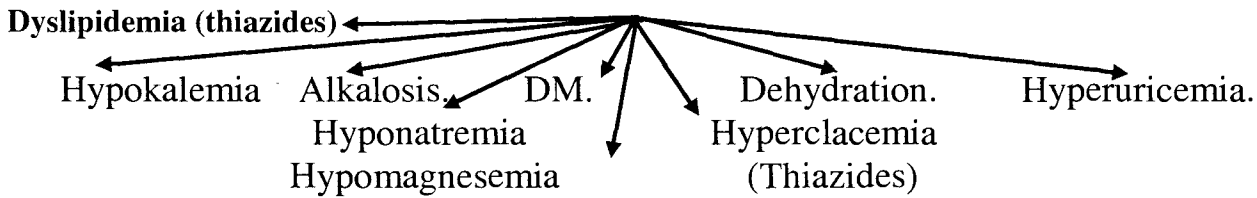
### Thiazides:

- Act mainly on distal tubules:
- \* Dihydrochlorothiazide 25 - 50 mg/d.
- \* Chlorothalidone 25-100 mg/d. (Long acting)

Thiazides in combination with loop diuretics have a synergistic action and greater diuretic effect.



## Side effects of Lasix and thiazides:



### 💣 Q. Uses of diuretics in medicine:

- |                               |                                    |
|-------------------------------|------------------------------------|
| ➔ Hypertension, heart failure | ➔ Ascites, nephrotic \$.           |
| ➔ Lasix                       | ➔ Glaucoma (diamox)                |
| ➔ SIADH                       | ➔ Thiazides in DI.                 |
| ➔ Brain edema.                | ➔ Conn's syndrome (spironolactone) |

### **K- Sparing diuretics:**

They can be combined with lasix and thiazides.

- \* Spironolactone acts through aldosterone antagonism in the distal tubules. (Tab. 25 mg) we give up to 200 mg/day. Hyperkalemia and gynaecomastia are side effects. Spironolactone may reduce the process of remodeling.
- \* Other K sparing diuretics. e.g triamterene, amiloride (5-20 mg/d). They act directly on ion transport in the distal tubules with no aldosterone antagonism, (inhibit Na channel) so they inhibit reabsorption of Na and secretion of K ions.

### **Osmotic diuretics:** (contraindicated in heart failure)

- \* E.g. mannitol.
- \* They do not markedly influence Na, Cl excretion.
- \* They usually not used in heart failure as they cause initial hypervolemia leading to volume overload.

### **Carbonic anhydrase inhibitors:**

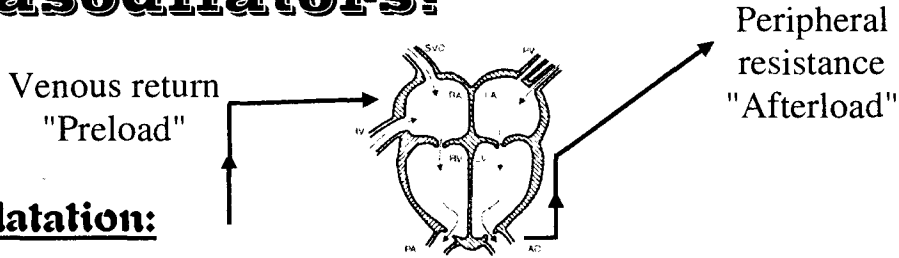
- \* E.g. acetazolamide used in glucoma only.

### **Aminophylline:**

- \* Oral, suppositories, IV.
- \* It is usually used in cases of heart failure with superimposed bronchospasm.
- \* IV injection must be very slowly to avoid arrhythmia.
- \* It is bronchodilator.
- \* It is + ve inotropic.
- \* It has a diuretic effect (due to ↑ renal blood flow).



## 5. Vasodilators:



### Venodilatation:

Value: decrease venous pressure, this will relieve SVC and PVC.

↓ Venous return → ↓↓ Preload

### Arterial vasodilatation:

Value: ↓↓ Peripheral resistance → ↓↓ afterload, this will improve myocardial efficiency So → ↑ COP

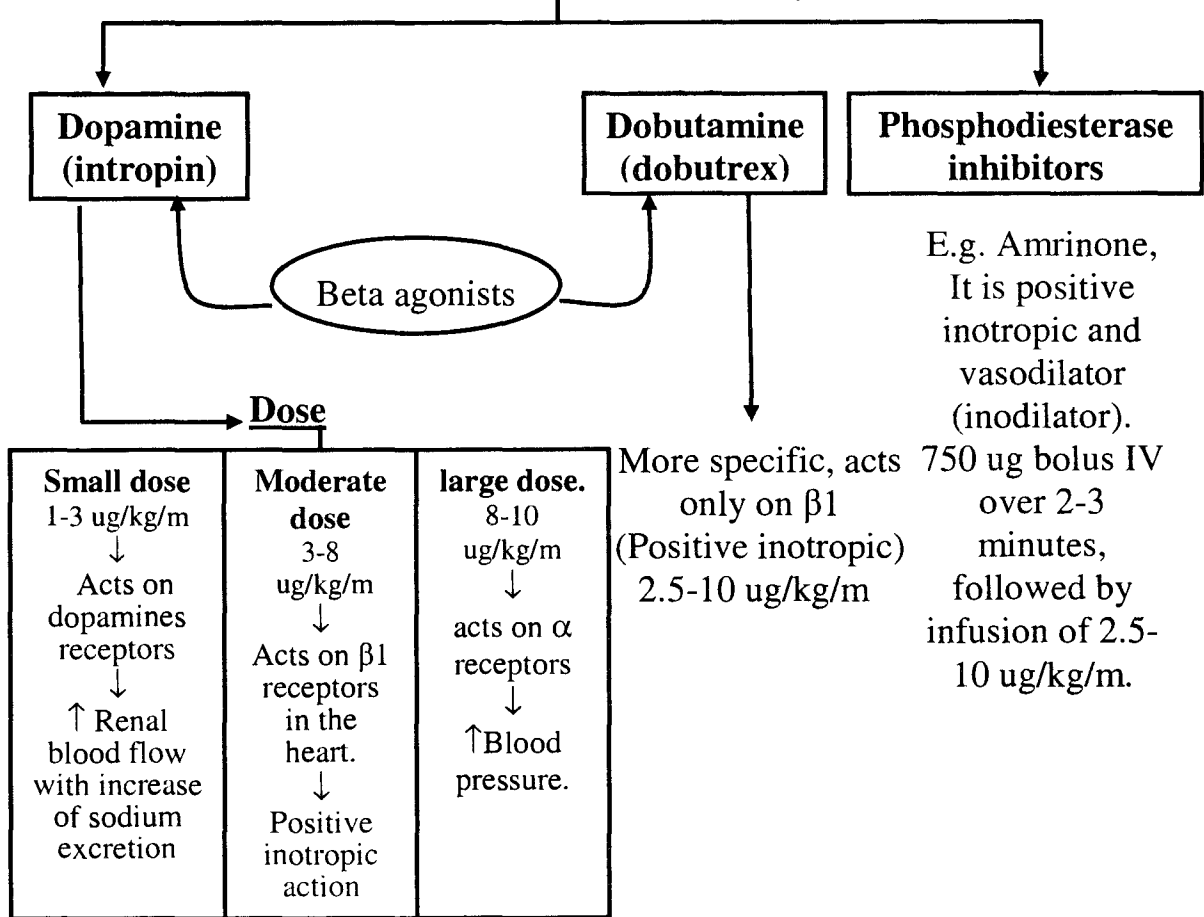
\* ↑↑ After load → ↓ COP.  
 \* ↑↑ Preload → ↑↑ PVC, ↑ SVC.

☆ **So veno & arterial vasodilators** must be used e.g ACE inhibitors.

ACE inhibitors are the best vasodilators in cases of heart failure. Captopril (12.5 mg/8hr. up to 50 mg /8hr.), ramipril (2.5 mg/12hr up to 5 mg/12hr) or enalapril (2.5-10 mg/12 hr). To get the benefit of these vasodilators, keep the systolic blood pressure above 100 mmHg. ACE inhibitors also lead to reduction of the process of remodeling.

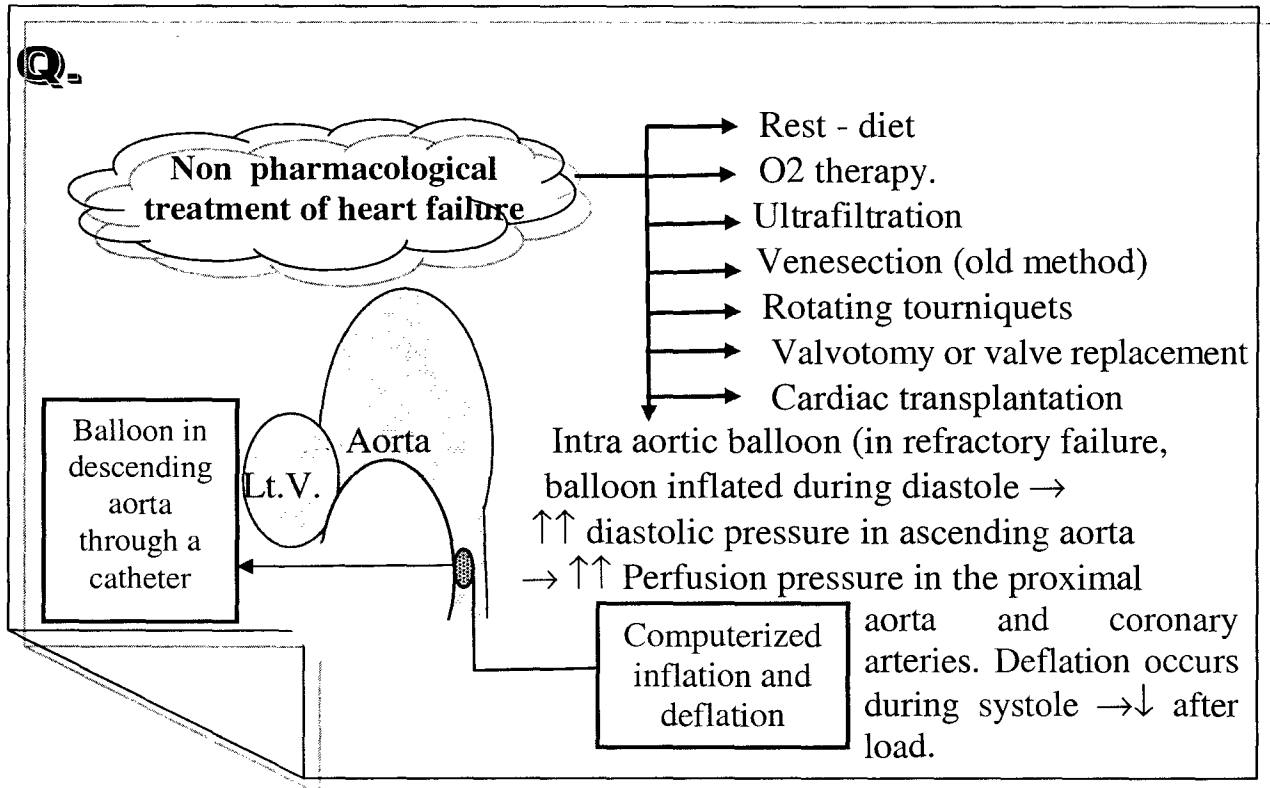
## 6. Potent inotropic: (used in intractable or refractory failure)

by IV infusion





**7- Treatment of precipitating factors, surgery e.g.: Valvotomy, ultrafiltration and intraortic balloon.**



**8. Treatment of complications of heart failure:**

- Anticoagulants for patients with atrial fibrillation or with history of thrombo-embolism and in cases with dilated cardiomyopathy.
- Antiarrhythmic drugs e.g for atrial fibrillation, ventricular tachycardia (arrhythmia may lead to deterioration of symptoms). Electrolyte disturbances and digitalis toxicity must be diagnosed early and treated.
- **Cardiac resynchronization:** many patients with HF developed LBBB which delays and discoordinates contraction, so insertion of pacemaker to resynchronize contraction improves both the hemodynamics and symptoms.
- Monitoring of kidney functions and urine output to avoid renal failure.

**CARDIOGENIC PULMONARY EDEMA**

**(ACUTE LEFT HEART FAILURE)**

**Causes:**

- 1- Acute left ventricular failure. e.g extensive MI, myocarditis.
- 2- MS with aggravating factors e.g AF.
- 3- Acute mitral incompetence (backward failure), acute AI.

Pulmonary capillary pressure above 20 mmHg leads to interstitial edema. Alveolar edema occurs when the capillary pressure exceeds the total oncotic pressure (approximately 25 mmHg).



## C/P:

- ❖ Manifestations of the cause.
- ❖ Marked dyspnea, orthopnea and haemoptysis (frothy pink sputum).
- ❖ Sense of impending death with marked irritability.
- ❖ Bubbling crepitations and rhonchi all over the chest.

## Investigations:

- ❖ **Chest x ray:** Showing butterfly opacity (**Bat wing appearance**)
- ❖ **Echo:** Showing decline of ejection fraction of the left ventricle or any valve lesion.

## Treatment of acute cardiogenic pulmonary edema or acute left heart failure

- \* Hospitalization & rest in bed in sitting position, O<sub>2</sub> therapy with high concentration (60%, 100%).
- \* Treatment of precipitating factors & the cause.
- \* Morphine 2-5 mg IV → ↓↓ Venous pressure & sedation, naloxone must be available, metoclopramide 10 mg IV to prevent emesis.
- \* Furosemide is a potent venodilator and decreases pulmonary congestion before its diuretic action. An initial dose 20-40 mg IV given over several minutes and can be increased, to a maximum 200 mg in subsequent doses.
- \* Venous vasodilators e.g. nitroglycerin 5-10 µg/m (rapid, effective).
- \* Na nitroprusside (20-30 µg/m in hypertensive patients, keep the systolic blood pressure > 100 mmHg).
- \* Powerful positive inotropic → dopamine or dobutamine (see before).
- \* IV digitalization if needed e.g. with rapid AF.
- \* Aminophylline, 5mg/kg IV infusion over 10 minutes.
- \* Tracheobronchial aspiration.
- \* Ultrafiltration, rotating tourniquets, Intra aortic balloon as before.

## Recent role of BB in treatment of heart failure

- Activation of the sympathetic system may initially maintain cardiac output through an increase in myocardial contractility, heart rate and peripheral VC. However prolonged sympathetic stimulation leads to cardiac myocyte apoptosis (cell death), hypertrophy and focal myocardial necrosis.
- BB may help to counteract the deleterious effects of enhanced sympathetic stimulation and may prevent arrhythmia and sudden death. We can start with small dose, bisoprolol (concor) 1.25 – 2.5 mg/d with gradual increase of the dose according to need with monitoring of patients.
- Abrupt administration of large doses of BB can intensify HF, specially acute HF.





## Different classifications of heart

### i **A. Systolic failure:**

As before.

### **B. Diastolic dysfunction:**

This means ↓ the ventricular compliance with ↓ ventricular filling. Blood accumulate in the atrium, this is common in systemic hypertension, ischemic heart disease, restrictive cardiomyopathy → mild PVC → dyspnea, orthopnea.

#### Treatment:

(Ca channel blockers- β Blockers)

### ii **A. ↓ COP failure:**

As before.

### **B. ↑COP failure:**

As in thyrotoxicosis, beri beri, and anaemia where there is ↑COP, but the heart is unable to meet the demands of tissues due to the hypermetabolic state.

#### Treatment:

It is treated by treatment of the cause

### iii **A. Left sided failure**

### **B. Right sided failure**

### IV **Backward heart failure**

i.e HF presented mainly with SVC or PVC or both.

### **Forward heart failure**

i.e HF presented mainly by ↓ COP

### V **A. Acute heart failure**

As before

### **B. Chronic heart failure**

As before

- **Refractory heart failure** i.e non responding heart failure despite adequate ordinary medical therapy i.e (Digitalis, diuretics and vasodilators) we can use IV dopamine, dobutamine, amrinone. Intraaortic balloon insertion, treatment of PPT factors, surgery e.g valvotomy, valve replacement and lastly cardiac transplantation.



# SYSTEMIC HYPERTENSION

## Definition:

- ✓ Persistent elevation of blood pressure above normal values on three different occasions under mental & physical rest.
- ✓ Blood pressure > 140/90 diagnosed as hypertension.
- ✓ About 15% of population can be regarded as hypertensive!?

## Isolated Systolic Hypertension

- ✓ It means systolic blood pressure  $\geq$  140 mm Hg, with diastolic blood pressure below 90 mmHg. Grade 1 (140-159), grade 2 ( $\geq$  160).

## Causes:

- ♥ Atherosclerosis.
- ♥ Thyrotoxicosis.
- ♥ Complete heart block.
- ♥ AI – PDA.
- ♥ Coarctation of Aorta.
- ♥ Anxiety, fever.

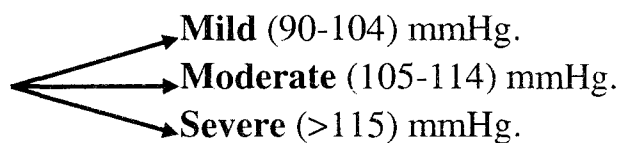
## Treatment:

- ✓ Treatment of the cause.
- ✓ Isolated systolic hypertension with high pulse pressure is associated with high incidence of cerebrovascular stroke.
- ✓ Antihypertensive can be given for elderly with atherosclerosis with isolated systolic hypertension e.g calcium channel blockers, ACE inhibitors.

## Diastolic Hypertension

Diastolic hypertension = diastolic blood pressure  $\geq$  90 mm Hg, this is usually associated with elevated systolic blood pressure.

## Grades:



## Recent staging of hypertension:

Category	Systolic in mmHg	Diastolic in mmHg
Normal:	< 130	<85
High normal: (or prehypertension)	130 – 139	85 – 89
Hypertension:		
★ Stage I (mild):	140 – 159	90 – 99
★ Stage II (moderate):	160 – 179	100 – 109
★ Stage III (severe):	$\geq$ 180	$\geq$ 110



**Labile hypertension:** It is considered in patients who sometimes, but not always have arterial pressures in the hypertensive range, these patients are often considered to have borderline hypertension and must be observed.

## ➡ Causes of diastolic hypertension:

**Primary hypertension  
"Essential"**

- ★ Age: usually between 35 – 55 years.
- ★ No apparent causes.
- ★ Family history is usually positive.

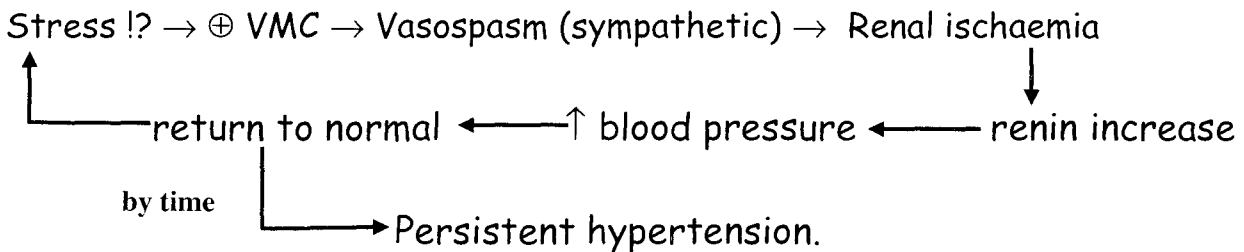
It is usually benign hypertension in most cases (slowly progressive with remote complications), but it may turn malignant.

### ➡ Theories of primary hypertension:

1. Renal Theory: There is increase of renin secretion.
2. Increased adrenal gland activity → ↑↑ Aldosterone secretion.
3. Increased activity of VMC → ↑↑ sympathetic discharge

↓  
↑ Blood pressure.

4. Multifactorial theory:



5. Barroreceptors resetting.
6. Impaired pressure natriuresis.
7. ↑ COP - ↑ P.R.
8. Genetic factors, hypertension tends to run in families and children of hypertensive parents.
9. Insulin resistance, obesity.
10. Alcohol intake, excessive sodium intake or salt sensitivity.

**Secondary hypertension**

- ★ Age: usually < 35 or > 55 years.
- ★ Causes: e.g renal or endocrinal diseases.
- ★ Family history is usually negative.

It may be malignant hypertension (rapidly progressive with early complications) specially with renal hypertension.



**Causes of secondary hypertension:**

1. **Renal:**
  - Glomerulonephritis.
  - Chronic tubulointerstitial nephritis.
  - Diabetic nephropathy.
  - Obstructive uropathy.
  - Renal artery stenosis.
  - Polycystic kidney disease.
  
2. **Endocrinal:**
  - Cushing syndrome and Conn's disease.
  - Pheochromocytoma, Acromegally.
  - Hyperparathyroidism, Myxodema.
3. **Neurological:** → ↑ ICT → reflex ↑↑ in blood pressure (**cushing reflex**).
4. **Cardiovascular:** → Coarctation of aorta.
5. **Pregnancy:** → Preeclampsia.
6. **Blood disease:** → Polycythemia → hyperviscosity → ↑ blood pressure.
7. **Drugs:**
  - Corticosteroids.
  - Oral contraceptives containing oestrogen.
  - NSAID.
  - Ephedrine, Erythropoietin, Cyclosporine, Carbenoxolone and Sympathomimetic agents.

} → Salt & H<sub>2</sub>O retention.

**C/P of hypertension:**

**Symptoms**

(It is mostly asymptomatic)

- Asymptomatic
  - Occipital headache
  - Blurring of vision,
  - Easy fatigue
- } non specific
- Symptoms of complications.
  - Symptoms of the cause (2ry H.).

**Signs**

- Elevated blood pressure
- Signs of the cause (2ry H), see later.

**Manifestations of the cause of hypertension:**

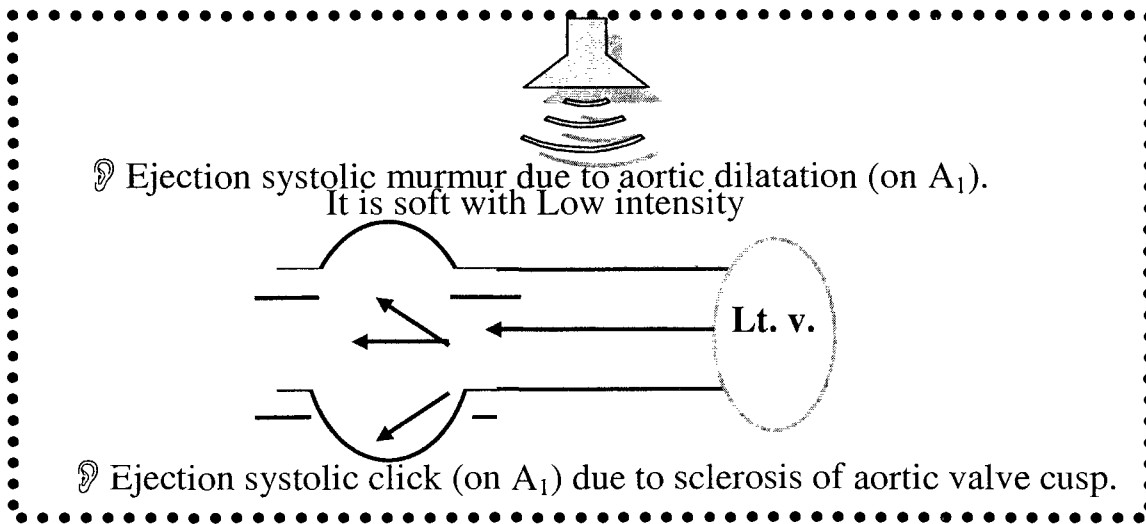
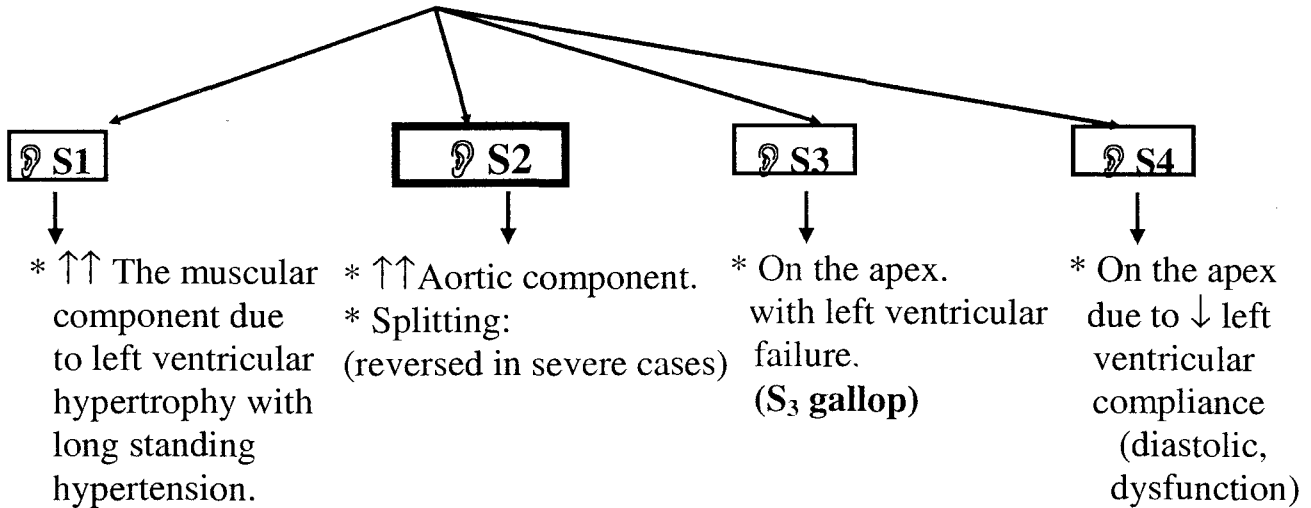
- e.g.:
- ★ Radiofemoral delay (Coarctation of aorta)
  - ★ Enlarged kidneys (polycystic kidney)
  - ★ Moon face (Cushing's syndrome)
  - ★ Troussaue's sign (Conn's syndrome)
  - ★ Acromegaly, thyrotoxicosis (see endocrine).
  - ★ Bruits in flanks due to renal artery stenosis.

**Examination of the heart (may be normal, but):**

(a) **Inspection and palpation** may show sustained apex, pulsating aortic area with aortic dilatation.



## (b) Auscultation



### Malignant Hypertension

- It is a rapidly progressive hypertension (diastolic pressure usually > 130-140 with papilloedema), there is fibrinoid necrosis of the vessel wall with early complications, causing end organ damage

Cerebral hemorrhage. (see neurology)      Acute or rapidly progressive renal failure      Left heart failure.

- **C/P:** as above plus:

- ☛ Pallor: Vasospasm.
- ☛ Fundus examination (Macular star + Papilloedema)

### Q Accelerated hypertension?

It is similar to malignant hypertension with no papilloedema or end organ damage. Systolic blood pressure > 200 mmHg and/or diastolic pressure > 120 mmHg. If untreated it will progress to malignant phase.



## Complications: (Target organ damage)

1. Heart
  - Systolic dysfunction or left sided heart failure
  - Ischemic heart disease.
  - Diastolic dysfunction.
  - Atrial fibrillation.
2. Neurological
  - Stroke (cerebral hemorrhage – Lacunar infarction).
  - Hypertensive encephalopathy.
3. Kidney → Renal failure
  - ☆ Chronic renal failure in benign essential hypertension.
  - ☆ Acute or rapidly progressive renal failure in malignant hypertension.
4. Eye → Retinopathy
5. Aortic aneurysm and dissection.
6. Side effects of the antihypertensives.

## Investigations:

1. ECG & X-ray: → Left ventricular hypertrophy.
2. Echocardiography:
  - "Long standing hypertension"
  - Left ventricular hypertrophy.
  - Diastolic dysfunction.
  - Ejection fraction (it declines late).
3. Fundus Examination:
 

If it is positive , this indicates long standing hypertension

(Grades):

  - 1- Silver wiring of arterioles.
  - 2- Artery - Venous nipping +1.
  - 3- Haemorrhage - exudate +2.
  - 4- Papilloedema + 3 (with malignant H).
4. Investigations for the Cause:
  - Urine analysis for protein, casts. Kidney function tests, renal angiography and duplex scan on renal arteries.
  - Cortisol level (Cushing \$), growth hormone level
  - Blood picture e.g for polycythemia.
  - VMA in urine- Plasma renin level in blood (↑ in 10% of cases)
  - Na, K (Hypokalemic hypertension) → see later – Lipid profile.

## Treatment:

### Life style modifications: (non pharmacological therapy).

- (1) Rest in bed during exacerbations.
- (2) Stable cases:
  - \* Moderation of life:
  - \* Avoid stressful conditions.
  - \* Avoid straining.
- (3) Diet
  - \* Salt restriction with high potassium diet, excessive fruit and vegetables.
  - \* Low fatty diet (to ↓ cholesterol)
  - \* Low carbohydrate diet (to ↓ body weight)
- (4) Weight reduction (BMI < 25), aerobic exercises.
  - ✓ In overweight patients this leads to a true fall in blood pressure.



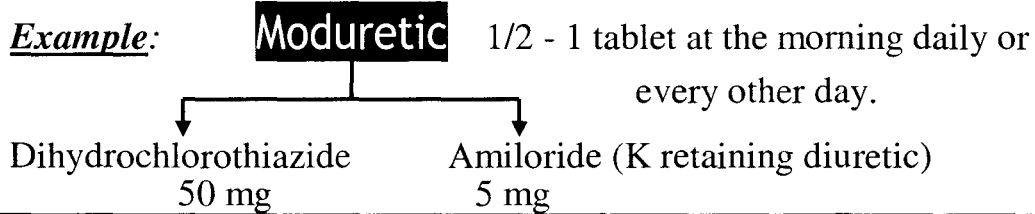
# DRUG THERAPY

## I. Diuretics

Value: Na excretion → decrease reactivity of blood vessels to catecholamines, reduction of blood and extracellular fluid.

### Thiazides:

Value: Diuresis, vasodilatation – can be tolerated for long time.  
Dose: Dihydrochlorothiazide 12.5mg/d up to 50 mg/d.



Indapamide (Natriliq) is related to thiazides 2.5 mg tablet/d. It has little effect on potassium, glucose, uric acid excretion. It has a vasodilator effect as it block calcium influx in blood vessels.

### Furosemide:

(it is not routinely used in stable cases of hypertension).

Indications:
 

- Emergency e.g. H. encephalopathy.
- Salt & water retention e.g. nephritic syndrome.
- GFR < 25 ml/min.

Dose 20-80 mg/d

### Spiroinolactone:

(Aldosterone antagonist).

- ◆ Used in Conn's syndrome. (Primary hyperaldosteronism)
- ◆ Dose : 400 mg/day.

*Amiloride or spiroinolactone are not effective when used alone, with the exception of spiroinolactone in primary hyperaldosteronism.*

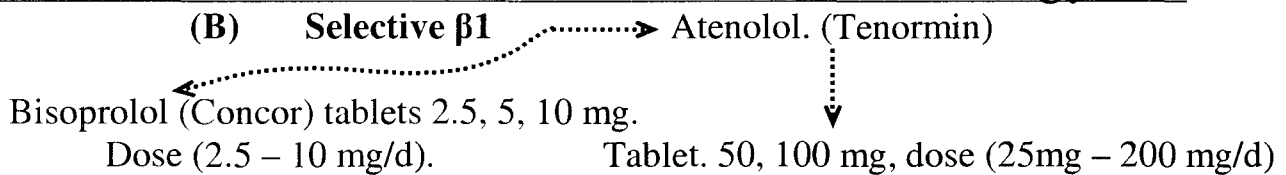
## II. Beta blockers

Mech
 

- Negative inotropic .....
- Negative chronotropic..... → ↓ Cop → ↓ blood Pressure.
- Inhibit renin release

Types: (A) NON selective β1 & β2

- Inderal , at least 80 mg - 120 mg /d !?
- Carvedilol 12.5-25 mg/d "Dilatrend" It is non selective with vasodilator properties, it can be used in patients with peripheral vascular disease!?



### III. $\alpha$ - Blockers

#### **Prazosin:** (Minipress)

- ⊗ **Action** Arterial vasodilator and venodilator.
- ⊗ **Uses**
  - Heart failure.
  - Hypertension.
  - Peripheral vascular diseases.
- ⊗ **Dose** 1– 10 mg/day
- ⊗ **Side effects**

- ✓ **First dose phenomenon (with 2 hours of the first dose):**  
i.e first dose → marked vasodilatation → syncope or postural hypotension.  
(To avoid, start with low dose, at home and on going to bed, also withhold diuretics and BB).  
Other  $\alpha$ -blockers e.g doxazosin (cardura) 1-4 mg/d, or terazosin (Itrin) 2-5 mg/d, they mainly used for senile prostate.

Labetalol (100-600 mg/12 hr) is an agent that has combined  $\alpha$  and B blocking properties, it can be used in hypertension with pregnancy. Bronchospasm and vomiting are side effects.

### IV. Centrally acting drugs

They are agonist to  $\alpha_2$  adrenoceptors in the brain leading to  $\downarrow\downarrow$  sympathetic discharge →  $\downarrow$  Blood pressure.

#### **$\alpha$ - methyl dopa):** (Aldomet), it is still widely used despite central, hepatic and haematological side effects.

- ⊗ **Uses**
  - Hypertension.
  - It is save with pregnancy.
- ⊗ **Side effects**
  - Depression.
  - Extra pyramidal manifestations.
  - Autoimmune.
    - Hemolytic anemia.
    - Chronic hepatitis.
- ⊗ **Dose** 250 - 1500 mg /d.

#### **Clonidine:** (Catapress), it is an imidazoline.

- ⊗ **Dose:** Tablet 150 ug (1-2 tab/d).

Side effect: →

Salt & water retention so, give diuretics. Rebound hypertension on withdrawal may occur.





**Reserpine:**

(It has no role in recent medicine)

♦ It depletes adrenergic nerve of noradrenaline stores.

♦ e.g.  $\begin{matrix} \longrightarrow \\ \longrightarrow \end{matrix}$  ♦ Serpasil.  
 ♦ Brinerdine (reserpine +  $\alpha$  blocker + thiazide related).

⊗ Side effects  $\begin{matrix} \longleftarrow \\ \longleftarrow \\ \longleftarrow \end{matrix}$  Depression.  
 Extra pyramidal manifestations.  
 Nasal congestion.

**V. Vasodilators**

**Arterial vasodilators:**

**Hydralazine:**

(Apresoline). Now it has little place in the routine oral therapy.

⊗ Used with pregnancy, it can be used in hypertensive encephalopathy by infusion. It leads to reflex  $\rightarrow \uparrow$  HR  $\rightarrow \downarrow\downarrow$  coronary filling.

⊗ Dose is up to 100 mg/day, in hypertensive emergency (see later).

⊗ Side effects  $\begin{matrix} \longrightarrow \\ \longrightarrow \end{matrix}$  SLE like.  
 Tachycardia – Headache – flushing.

**Calcium channel blocker:**

(Ca Channel blockers), see angina  $\begin{matrix} \longrightarrow \\ \longrightarrow \\ \longrightarrow \\ \longrightarrow \end{matrix}$  Nifedipine 10-20/8hr  
 Amlodipine 5-10mg/d.  
 Diltiazem 60-180mg/12hr.  
 Verpamil 120-480 mg/d

**Minoxidil:**

(Not used now)

♦ It leads to hypertrichosis

**Diazoxide:**

(Arterial vasodilator)

Inhibits insulin release.  
 (Used in insulinoma)

IV 1-3 mg/kg in cases.  
 of hypertensive encephalopathy  
 it is given rapidly

Very potent.

**Venodilators:**

**Nitrates** ..... (See angina)

♥ They can be used by infusion in case of hypertensive encephalopathy i.e glyceryl trinitrate (Tridil) 0.3-1 ug/kg/m.



**Arterial and Venodilators:**

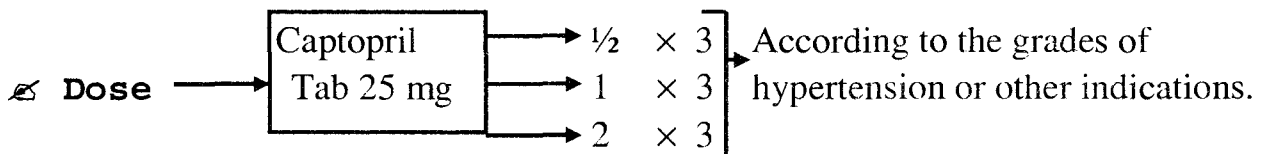
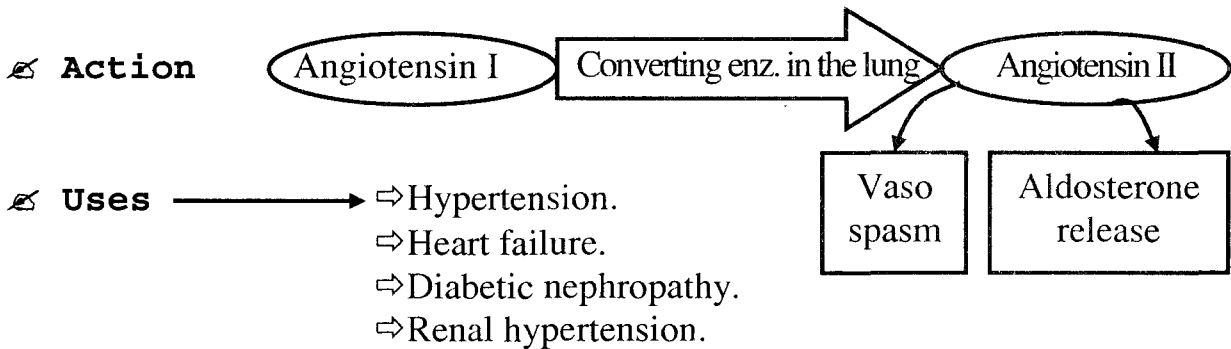
**Na Nitroprusside:** (Niprid)

- ◆ Very potent.
- ⚡ **Used:** In emergency.
  - H. encephalopathy.
  - Cardiogenic pulmonary edema.
- ⚡ **Dose:**
  - ◆ 50 mg vial, start with 0.3-1 ug/kg/m, the control of blood pressure is established at 0.5-6 ug/kg/m, maximum dose is 10 ug/kg/m.
- ⚡ **Side effects:**
  - ◆ Hypotension, cyanide or thiocyanate toxicity.

Na nitroprusside converted to cyanide which is converted to thiocyanate by the liver, thiocyanate excreted in urine.  
 Liver disease: → Cyanide toxicity. "Pink color, dilated pupil"  
 Renal disease: → Thiocyanate toxicity. "Tinitus, skin rash"

**α - blocker:** (see before)

**Angiotensin converting enzyme inhibitors**  
**"ACE inhibitors"**



- Side effects**
- Hyperkalemia, skin rash.
  - Nephrotic \$ (Membranous GN)
  - Dry cough due to accumulation of bradykinin in the lung.

ACE inhibitors reduce the process of ventricular remodeling.



☛ ACE inhibitor contraindicated in bilateral renal artery stenosis.

**OTHER ACE INHIBITORS:**

- ☛ **Ramipril** 2.5-10 mg/d (Tritace).
- ☛ **Lisinopril** Long acting 10-20 mg/d (Zestril).
- ☛ **Enalapril** 10-20 mg/day oral (Ezapril), it can be given IV.
- ☛ **Trandolapril** 1-4mg/dav.

**↪ Angiotensin II receptor antagonists: (ARBs)**

This group shares many of actions of the ACE inhibitors, but they do not cause cough. So, they are used for patients who can not tolerate ACE inhibitors. They include losartan 50-100 mg/D (Cozar) and valsartan 80-160 mg/D (Tareg)

♣ ***Schematic approach to the treatment of hypertension in patients with no need for specific therapy or emergency instead of the old stepped care approach***

1. Start with low dose, ACE inhibitor (12.5 mg/12hr captopril) or Ca channel blocker (60mg diltiazem/12hr) or (25 mg atenolol/day).
2. If no response → double the dose.
3. If no response add low dose thiazides.(12.5 mg – 25 mg/day)
4. If no response give full dose of the initial drug + thiazides.
5. If no Response add Clonidine, hydralazine, α methyl dopa.

♣ **Hypokalemic hypertension**

1. Conn's syndrome: → ↑ Aldosterone → ↑ Blood pressure.  
→ ↑ K excretion.
2. Cushing syndrome: → ↑ Cortisol → ↑ Blood pressure.  
→ ↑ K excretion.
3. Renal artery stenosis: → Renin ↑ → ↑ Aldosterone.  
→ ↑ Angiotensin.

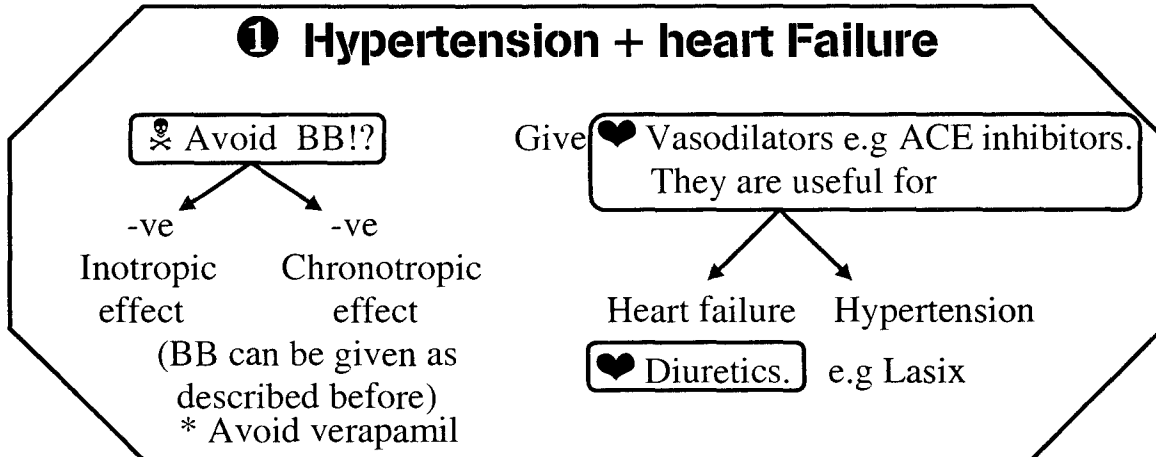
**Adjuvant drug therapy of hypertension:**

- \* Antiplatelet (Aspirin) to reduce cardiovascular risk, it may cause intracranial haemorrhage in small number of patients, the benefits of aspirin therapy are thought to outweigh the risks in patients with will controlled blood pressure.
- \* Lipid lowering drugs (statins, see later).

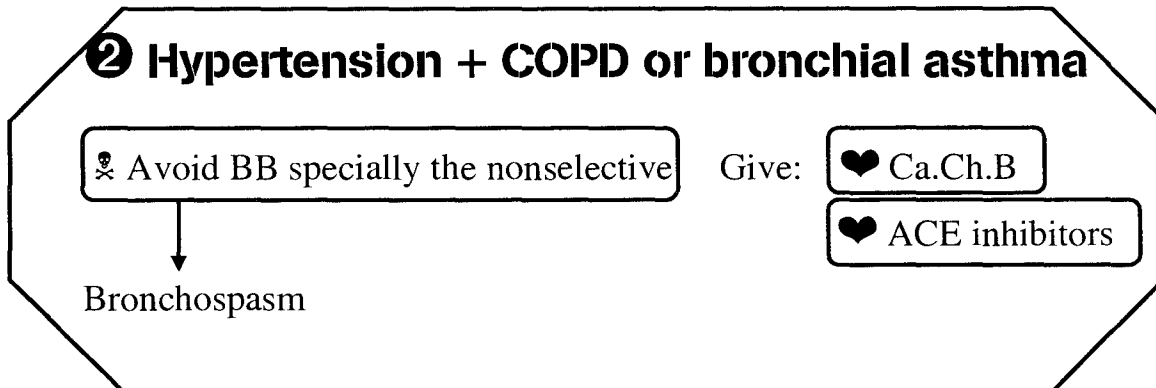


# Special problems with hypertension

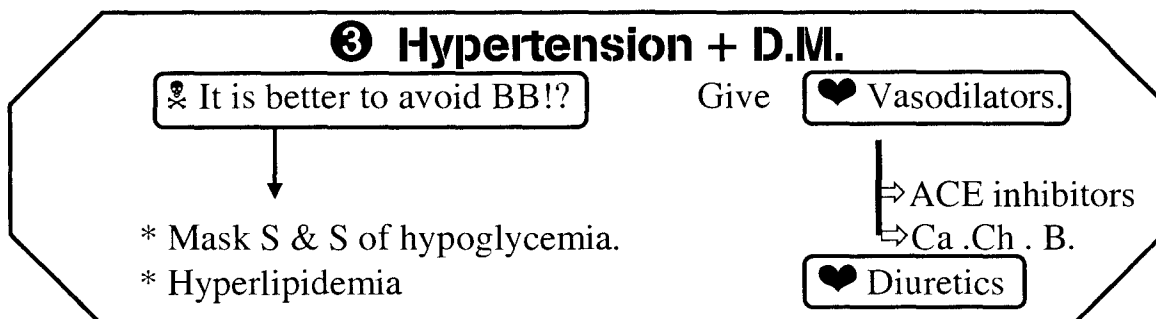
## 1 Hypertension + heart Failure



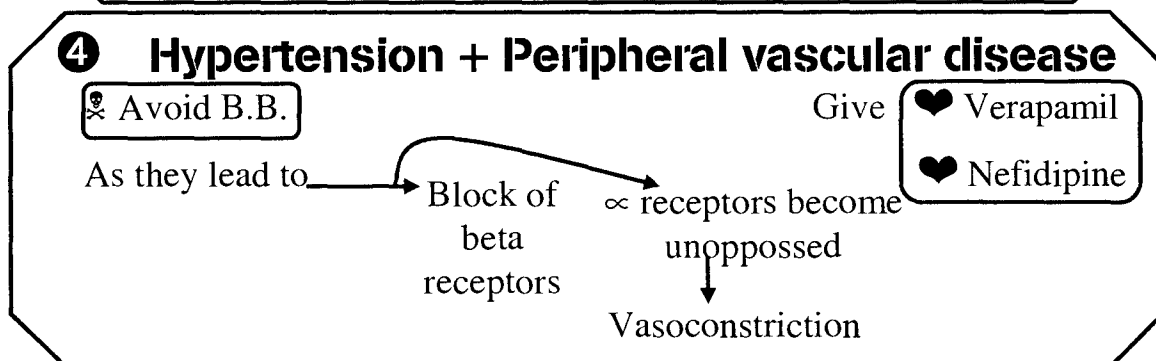
## 2 Hypertension + COPD or bronchial asthma



## 3 Hypertension + D.M.



## 4 Hypertension + Peripheral vascular disease



∴ 1, 2, 3, 4 Avoid BB , give vasodilators.



### 5 Hypertension + IHD

☠ Avoid Hydralazine:

As it ↓↓ coronary filling  
due to ↑↑ heart rate.  
Avoid nifedipine (alone).

Give ♥ (B.B. or Ca.Ch.B)

They are useful for: → Hypertension.  
→ Ischemic heart disease.  
Also we can give ACE inhibitors.

### 6 Hypertension + Pregnancy

\* Avoid

☠ Propranolol

→ Foetal bradycardia.

☠ Diuretics

→ ↓↓ placental blood flow.

☠ ACE inhibitors

\* Give

♥ α methyl dopa.

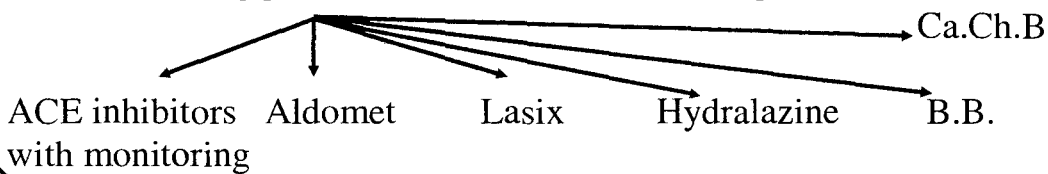
♥ Hydralazine.

♥ Atenolol.

♥ Ca.Ch.B.

♥ Labetalol

### 7 Hypertension + Renal impairment



### 8 Malignant hypertension and accelerated phase of hypertension

- ◆ It is unwise to lower blood pressure too quickly, because this may compromise tissue perfusion due to altered auto regulation and may lead to cerebral damage, coronary or renal insufficiency a controlled reduction, to level of about 150/90 over a period of 24-36 hrs is ideal.
- ◆ In most patients it is possible to give oral drug therapy with BB, Ca.Ch.B, Lasix and ACE inhibitors with bed rest.
- ◆ In critical cases we can use:
  - Glyceryl trinitrate 0.6-1.2 mg/hr.
  - Sodium nitroprusside 0.3-10 ug/kg/m.
  - Hydralazine 1.5 – 5 ug/kg/m.
  - Labetalol 20 mg/m to a maximum of 200-300 mg.

### 9 Hypertension in elderly

Give ACE inhibitors or Ca.Ch.B.  
It is better to avoid BB and thiazides if possible.

**10 Hypertension with diastolic dysfunction give BB, verapamil or diltiazem**



# Hypertensive encephalopathy

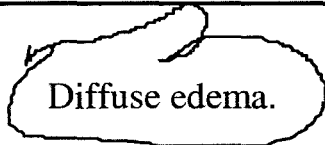
**Definition:** Sudden, marked elevation of blood pressure ⊕ abnormal cerebral state. i.e brain oedema, that is potentially reversible with control of blood pressure.

**Pathology:** Sudden and marked increase of blood pressure → disturbance of the cerebrovascular autoregulatory mechanisms → brain edema.

- Diagnosis:**
- ⊗ Very high blood pressure, headache, visual blurring, drowsiness, convulsions.
  - ⊗ Coma → without lateralizing signs.
  - ⊗ Fundus examination showing papilloedema and retinal hemorrhages. CT, MRI show diffuse cerebral edema.

**D.D:**

**H. encephalopathy**



Diffuse edema.  
 ↳ Diffuse edema.  
 ↳ No lateralizing signs

**stroke**



Focal lesion  
 e.g.: cerebral hge or infarction  
 ↳ +ve lateralizing signs

It is unwise to lower the blood pressure too quickly in hypertensive encephalopathy because this may compromise tissue perfusion due to altered autoregulatory mechanisms → cerebral damage.

**Treatment:**

Anti hypertensive drugs

- Glyceryl trinitrate 0.6-1.2mg/hr.
- Sodium nitroprusside 0.3-10 ug/kg/m
- Hydralazine 1.5-5 ug/kg/m.
- Labetalol 20 mg IV over 2 minutes, the dose can be doubled every 10 m until control of blood pressure(maximum dose 200-300 mg).

Convulsions → Diazepam IV.

Brain edema → Frusemide is effective.

**Q- Refractory hypertension**  
 The commonest causes are non compliance, inadequate therapy and failure to recognize the cause as: renal artery stenosis

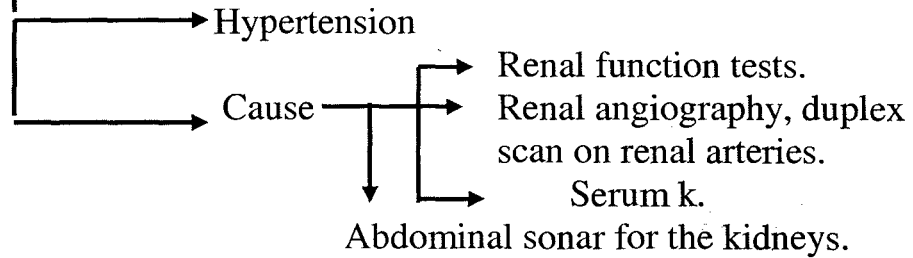


## Q- Renal hypertension

⌚ **Aetiology:** see before.

⌚ **C/P:** C/P of hypertension - C/P of the cause.

⌚ **Investigations for:**



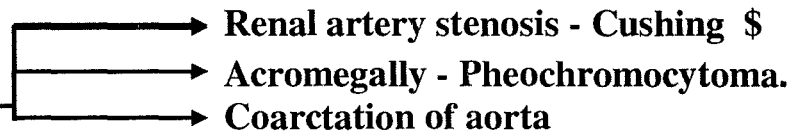
⌚ **ttt:**

- Medical → drugs (as before)
- Surgical, according to the cause as renal artery stenosis.

## Q- Curable hypertension

It is a surgically curable hypertension, it is a secondary hypertension.

### Causes



**C/P:** Hypertension + the cause

**Investigations** \* for hypertension  
\* for the cause

**ttt**

- Control blood pressure
- Surgery according to the cause

## Hypertensive crises (BP > 200/120 mmHg) are subclassified into:

### \* Hypertensive urgency:

Severely elevated blood pressure with no evidence of ongoing target organ damage i.e no neurologic, cardiovascular or renal deterioration and fundoscopic abnormalities. Blood pressure should be controlled within 24-48 hours. It can includes the term accelerated H !?

### \* Hypertensive emergency:

As above but with evidence of ongoing target organ damage. It should be treated immediately with parenteral medications, with reduction of the mean blood pressure by 25% within 1 hour of presentation. It includes hypertensive encephalopathy, LVF and acute renal failure, aortic dissection, subarachnoid hemorrhage, and cerebral infarction or haemorrhage. It can includes the term! Malignant H.



# ISCHAEMIC HEART DISEASE (IHD)

Myocardial ischaemia occurs when there is an imbalance between the supply of oxygen (and other nutrients) and the myocardial demands.

## Causes:

A) Decrease coronary blood flow by mechanical obstruction.

- 1-Atherosclerosis.
- 2-Coronary spasm.
- 3-Coronary thrombosis or embolism.
- 4- Vasculitis
  - Young female (SLE) →
  - ← Young male (Poly arteritis nodosa)
- 5- Syphilitic coronary osteal obstruction.

B) Decrease the flow of oxygenated blood to the myocardium.

- 6-Hypoxia / anaemia.
- 7-Hypotension and decreased COP.

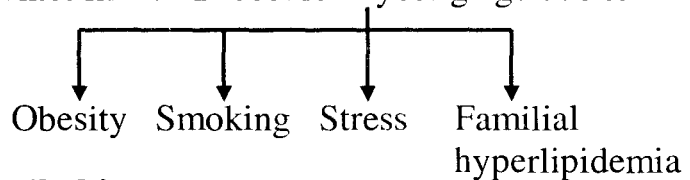
C) Increased oxygen demand

- 8-Increased demands e.g left ventricular hypertrophy
  - ← Hypertension
  - ← Aortic stenosis

## Risk Factors:

(for coronary atherosclerosis)

1. **Age** → Usually above 40, sometimes IHD occurs in young age due to



2. **Sex:** male > female , positive family history.

3. **DM** with hyperinsulinism.

4. **Hyperlipidaemia** with ↑ LDL

5. **Obesity**, lack of exercise (sedentary lifestyle).

6. **Dietary factors:**

Diets deficient in fresh fruit, vegetables and polyunsaturated fatty acids. Low levels of vitamin C, vitamin E and other antioxidants may enhance the production of oxidised LDL. Low dietary folate and Vit B<sub>12</sub> can elevate homocysteine levels.

7. **Stress**

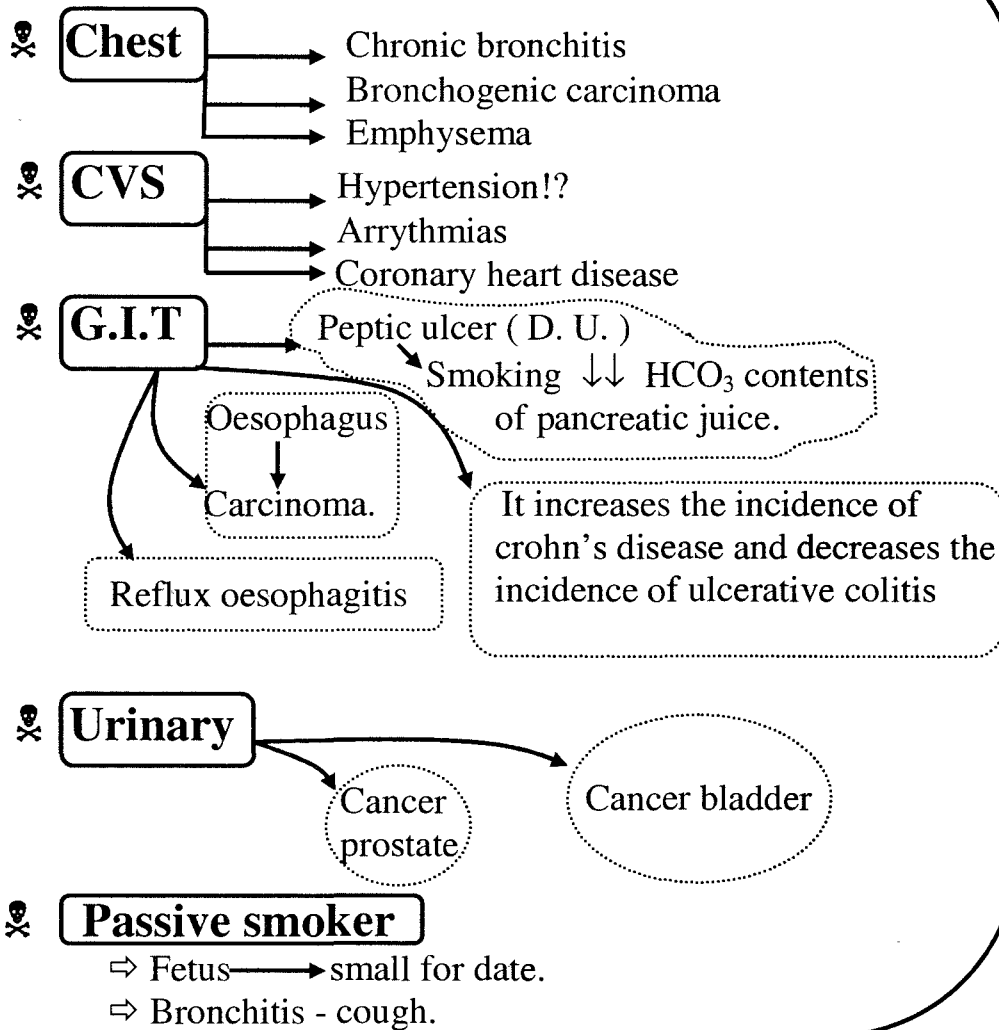
8. **Recent associations:**

- ⇒ High Plasma homocysteine.
- ⇒ High CRP
- ⇒ High fibrinogen.
- ⇒ Chlamydia pneumoniae.



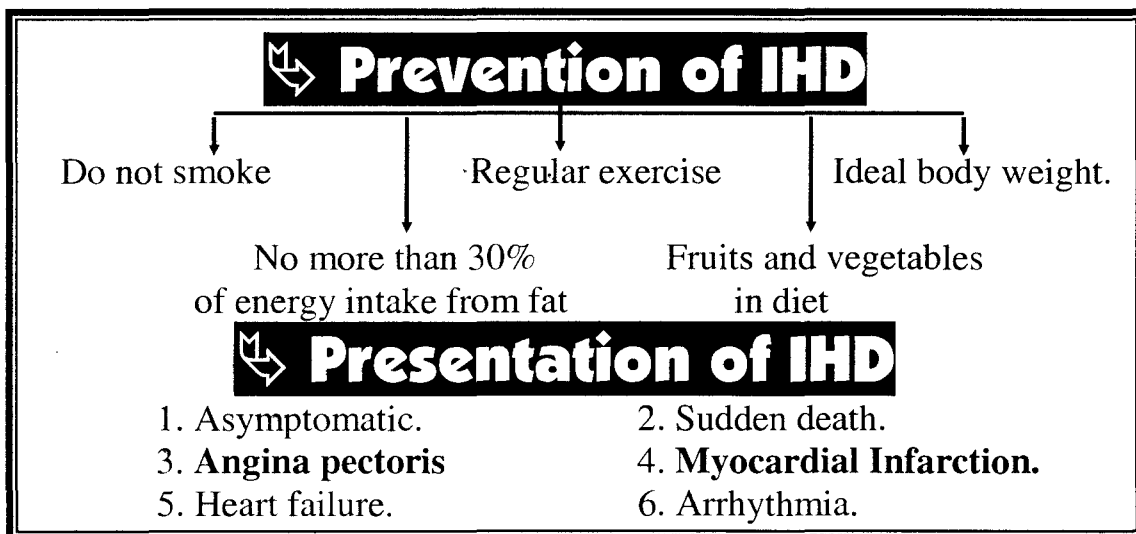


**9. Smoking:** The hazards of smoking are:



**10- Heavy alcohol consumption, contraceptive pills and cox-2 NSAID.**

The above risk factors 1, 2 are (fixed risk factors), but the other risk factors are (potentially changeable with treatment).

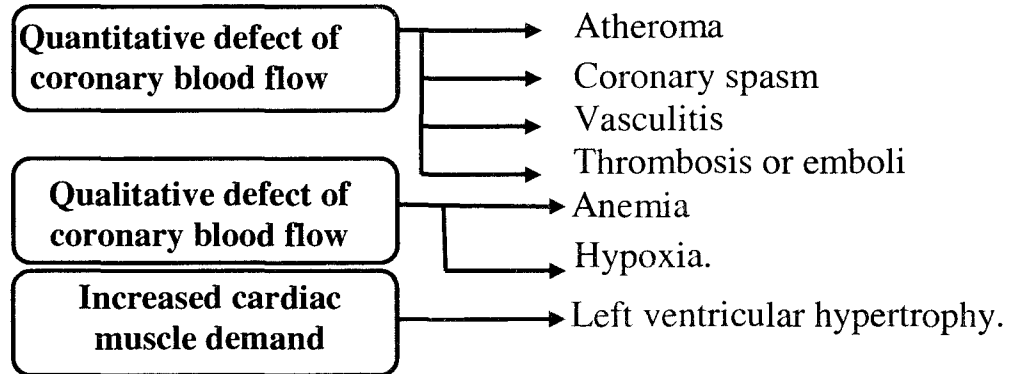




# ANGINA PECTORIS

**Def** Episodic clinical syndrome due to transient myocardial ischemia characterized mostly by chest pain with no cardiac tissue damage.

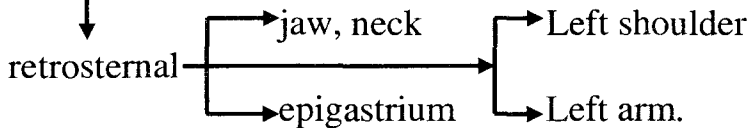
**Causes**



**C/P**

## Symptoms

① **Chest pain:** (heaviness, ↑ by exertion, ↓ by rest or nitrates)



② **Risk factors are positive** (see before)

⇒ **Anginal pain never to be:**

① Localized. ② Stitching or throbbing. ③ < 30 sec. , > 30 min (except unstable angina)

⇒ **The anginal pain PPT by:**

- ❖ Exertion
- ❖ Cold exposure
- ❖ Heavy meals
- ❖ Vivid dreams (nocturnal angina)

③ Angina may be presented with dyspnea, fatigue, faintness (**angina equivalents**).

## O/E

1. **The heart examination is mostly normal**
2. **Heart sounds** → S4 due to decreased ventricular compliance (Apex).
3. **Murmur of MI** due to ischaemic papillary muscle may be present.
4. **Other manifestations** e.g xanthelasma in hyperlipidemia, anaemia.

**Investigations**

**1. E.C.G.**

- ◆ S-T segment depression.
  - ◆ T- wave inversion.
- (During the attack)

Resting ECG usually normal.  
 ⇒ If resting ECG is normal, stress test will be required (treadmill test), 75% of patients with significant coronary artery disease will give a positive test.



**2. Cardiac scan (Thallium, Technetium)**

Thallium IV → Uptake by the heart. → reflect coronary perfusion.

- ✍ Thallium with exercise is more accurate.
- ✍ The heart also can be stressed with dobutamine in patients unable to do exertion.

**3. Echocardiography and dobutamine echo.**

✍ To assess ventricular function (ejection fraction). Also to detect wall motion abnormalities reflecting ventricular damage. Dobutamine echo is very useful in diagnosis of IHD (it is technically difficult).

**4. Coronary angiography (coronary catheter).**

**Indications:**

1. Stable angina refractory to medical therapy
2. Unstable angina
3. Strongly positive stress test
4. Post infarction angina
5. Unexplained / significant chest pain, where the diagnosis of angina is uncertain

**5. TLC / ESR/CK enzyme**

✍ Normal / No tissue damage.

**6. Lipid profile, serum homocysteine, CRP, blood sugar.**

**↳ Clinical types of Angina:**

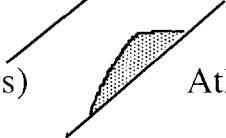
**I- Stable Angina:**

It occurs when coronary perfusion is impaired by fixed stable atheroma of the coronary arteries.

- ⇒ **Aetiology:** Atherosclerosis.
- ⇒ **Risk factors:** Are usually present.
- ⇒ **Criteria of pain**
  - Short duration (5-10 minutes)
  - Induced by exertion, emotional stress.
  - Relieved by rest and nitrates.

Some patients suffer anginal pain during the start of walking then disappear despite greater effort.(start – up angina).

- ⇒ **ECG**
  - S-T segment depression occur in ECG with effort or during anginal attacke.
  - Resting ECG often normal or may show (positive stress test) or previous MI.

- ⇒ **Angiography** → Fixed lesion (stable atheromatous) plaque  Atheroma.



# Categories of stable angina

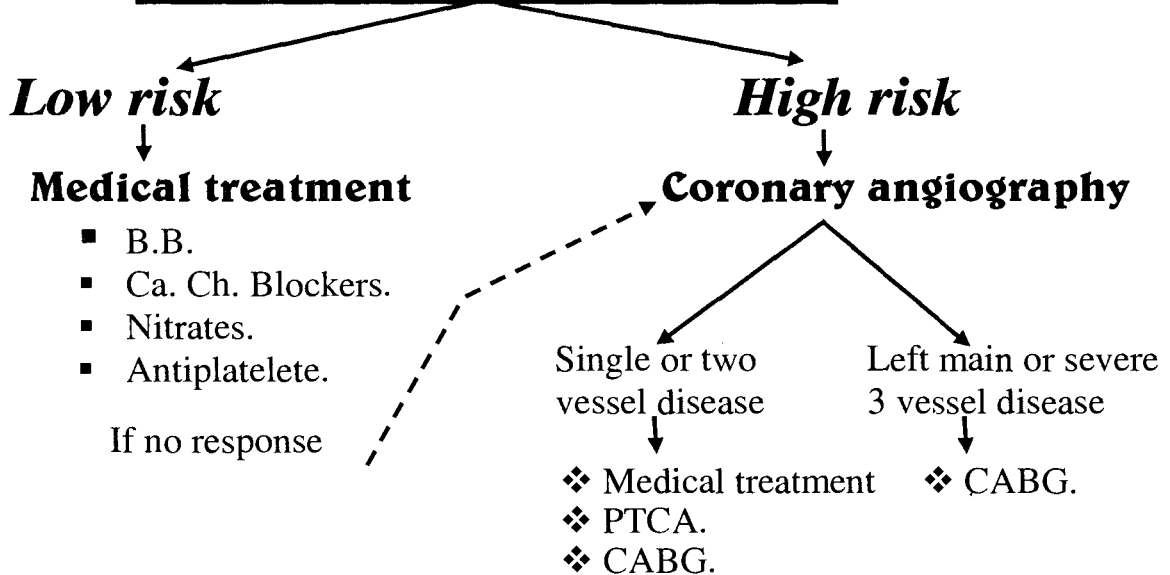
## High risk:

- ✓ Post infarction angina.
- ✓ Ischemia at low work load.
- ✓ Poor left ventricular function.
- ✓ Poor effort tolerance.
- ✓ Left main or three vessel disease.

## Low risk:

- ✓ Predictable exertional angina.
- ✓ Ischemia only at high work load.
- ✓ Single vessel or minor two vessel disease.
- ✓ Good effort tolerance.
- ✓ Good left ventricular function.

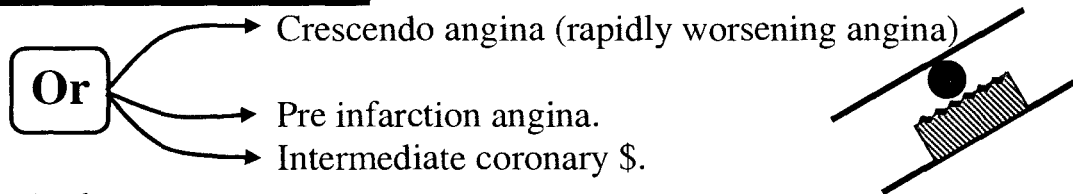
## Treatment of stable angina



**CABG** (Coronary artery bypass grafting)  
**PTCA** (percutaneous transluminal coronary angioplasty)

Advice to patients with stable angina: No smoking, ideal body weight, regular exercise, avoid severe unaccustomed exertion, sublingual nitrates before exertion that may induce angina.

## II- Unstable Angina:

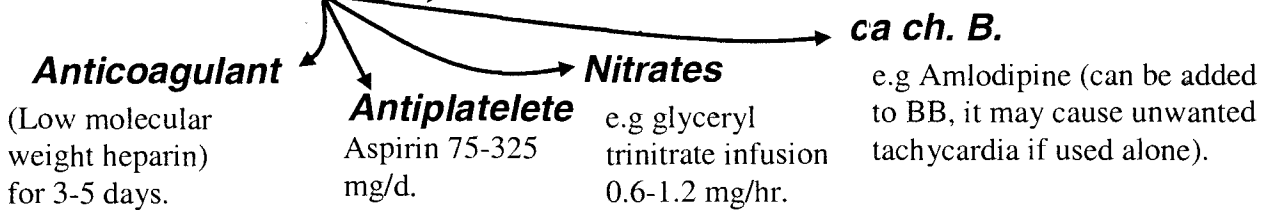


- ⇒ **Aetiology** as above. (But with complicated atheromatous plaque)  
 i.e a complex ulcerated or fissured atheromatous plaque ± local coronary spasm.
- ⇒ **Risk factors** as above.
- ⇒ **Criteria of pain:** Prolonged, at rest, frequent, poor response to nitrates.
- ⇒ **ECG** → as with stable angina, changes during discomfort may be more prominent.
- ⇒ **Angiography** → Fixed lesion (atheromatous plaque) ± coronary spasm.



⇒ **Treatment**, It is a medical emergency:

- ① **Hospitalization:** To exclude infarction (by enzymes), also there is high risk of MI or death.
- ② **Initial ttt** → **B.B.** e.g atenolol 50-100 mg/12 hr. (verapamil is an alternative)



Then angiography must be done, with planning for:

Or

**Coronary Artery Bypass Grafting (CABG):**  
Using saphenous vein or internal mammary artery

**Coronary angioplasty:**  
By balloon catheter (percutaneous transluminal coronary angioplasty) = PTCA.  
The main acute complications are occlusion of the target vessel by thrombus or dissection (2%), mortality (1%).

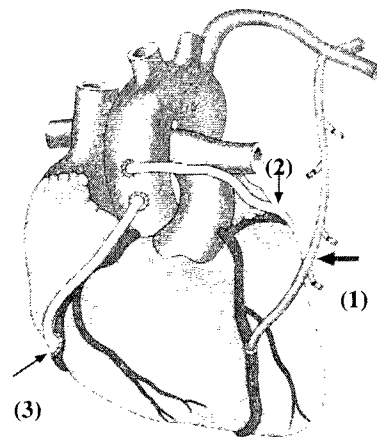
⇒ **Indications of CABG** (according to results of angiography):

- ✓ Three vessels coronary artery disease (left anterior descending branch, circumflex and right coronary)
- ✓ Two vessels disease involving the proximal left anterior descending branch.
- ✓ Left main stem artery disease.
- ✓ Symptomatic patient despite optimal medical treatment and whose disease is not suitable for PTCA.

⇒ **Indications of PTCA:**

- ✗ Single or two vessel disease!?
- ✗ Recent trials have demonstrated that PTCA is also feasible in patients with three vessel disease or left main coronary!?

- *PTCA can be done with or without stent insertion, aspirin plus ticlopidine are given following stent insertion.*
- *A triple coronary artery bypass graft, we use reverse saphenous vein graft for the circumflex and right coronary, and left internal mammary artery for the left descending branch or left main coronary (see the figure).*
- *Diffuse or distal disease diagnosed by angiography treated medically.*
- *Right internal mammary can be used for right coronary*



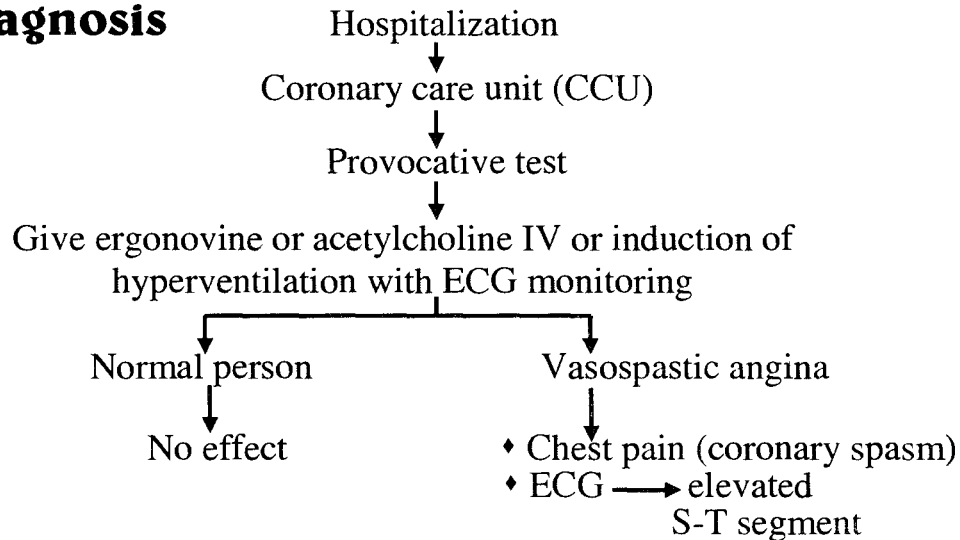
(1) Internal mammary artery  
(2) , (3) Saphenous vein.



### III- Variant angina (Prinzmetal's angina):

A) Pure vasospastic angina i.e in the presence of angiographically normal coronary arteries (dynamic coronary obstruction).

- ⇒ **Pathology** Coronary spasm.
- ⇒ **Age** Young age.
- ⇒ **Risk factors** are negative
- ⇒ **Not related to exertion.**
- ⇒ **Diagnosis**



- ⇒ **Treatment:** \* B.B. are contraindicated (may induce coronary spasm).
- \* Give  $\begin{cases} \rightarrow \text{Ca Ch. Blockers.} \\ \text{Or} \\ \rightarrow \text{Nitrates.} \end{cases}$

\* Aspirin may exacerbate the vasospastic angina.

B) Prinzmetal's angina (coronary spasm) can occur at the site of an atheromatous plaque.

#### Q Decubitus Angina:

Is that occurring on lying down.

It usually occurs with association impaired left ventricular function.

#### Q Nocturnal Angina:

Is that occurring at night and may wake up the patient from sleep.

It may be provoked by vivid dreams.

#### Q Cardiac syndrome X:

Refers to patients with a good history of angina, positive exercise test with normal angiography.

This is due to abnormalities of the coronary microcirculation.

#### Q Angina with normal coronary arteries i.e. (normal angiography)

1. Coronary spasm. (< 1% of all cases of angina)
2. Cardiac syndrome X.

**Q Acute coronary syndromes are myocardial infarction and unstable angina, as there are similar pathophysiologic mechanisms.**



## ↪ Treatment of Angina Pectoris

### ✎ During the attack:

- I. Rest, O<sub>2</sub> therapy.
- II. Nitrates sublingual tablets (see below)
- III. Reassurance and sedation.

### ✎ In between the attacks:

- I. Diet → ↓ fat, ↓ CHO, ↓ salt.
- II. Stop smoking, moderation of life, aspirin 75-150 mg/d.
- III. Treatment of risk factors: DM – Hypertension - Hyperlipidemia.
- IV. Drug therapy.

## Drug therapy

### I- Nitrates:

(converted to nitric oxide → vasodilatation.)

★ **Action:** → Venodilators → ↓ VR → ↓ preload  
 ↓  
 ☞ Coronary vasodilatation?!, dilate the larger conductance arteries.  
 ↓↓ Ventricular wall tension.

### ★ Other uses:

- ① Oes spasm and achalasia.
- ② Relieve of pulmonary venous congestion
- ③ Myocardial infarction.
- ④ Biliary colic and hypertensive encephalopathy.

★ **Side effects:** Headache – Hypotension

### ★ Routes:

1. Sublingual tablets 300 or 500 ug (glyceryl trinitrate). It can relieve the anginal attack within 3 minutes, the dose can be repeated, sublingual spray can be used (glyceryl trinitrate 400 ug/puff).
2. Oral isosorbide dinitrate 10 - 20 mg/8 hrs (Dinitra)
3. Amyl nitrate ampule for inhalation.
4. Ointment (2% nitroglycerin ointment) at night.
5. IV used in myocardial infarction and unstable angina.
6. Transdermal patches → long acting transdermal nitroglycerin (it can be applied in the morning and removed at bed time).

- ⇒ Chronic use of nitrate produces tolerance.
- ⇒ Long acting nitrates (Isosorbide mononitrate) are preferred, 20-60 mg once or twice/d e.g effox tablets 20 mg.
- ⇒ Sildenafil (Viagra) should not be given to patients taking nitrates.

(Sublingual tablets or spray are used during the attack, or prior to performing activities that will provoke angina).



**II- B.B.:**

★Action:

- 1. Negative inotropic
  - 2. Negative chronotropic
- ] ↗ ↓↓ O2 consumption.  
] ↘ ↑↑ the time of coronary filling.

★Types:

**1. Non selective (First generation)**

- Propranolol = (Inderal) 40 – 320 mg/d.
- Nadolol (Corgard) 80 mg/d.

**2. Selective (Second generation)**

- Atenolol = (Tenormin) 25 - 200 mg/d,
- Metoprolol (Betalock) 50 - 100 mg/d,
- Bisoprolol (Concor) 5 - 10 mg/d.

**3. Third generation with additional vasodilatation effect**

- E.g. Carvidilol (Dilatrend 25 mg/d)

**Lipophilic B.B** (lipid soluble):

They are well absorbed but undergoes extensive hepatic metabolism, this leads to short half life. They cross BBB e.g.: Propranolol.

**Hydrophilic B.B** (water soluble):

They are less absorbed and slowly eliminated, this leads to sustained Concentration. They can't pass BBB e.g.: Atenolol, Nadolol.

★Uses of BB:

⇒Cardiovascular uses:

- 1. Hypertension.
- 2. Angina.
- 3. Arrhythmia.
- 4. Cyanotic spells (F4).
- 5. Mitral valve prolapse.

⇒Non cardiovascular uses:

(usually, we use propranolol in these cases)

- 1. Thyrotoxicosis.
- 2. Anxiety.
- 3. Portal hypertension.
- 4. Familial tremors.
- 5. Parkinsonism.
- 6. Migraine.
- 7. Glaucoma (**Timolol**).

★Side effects:

- ♦ Bradycardia.
- ♦ Heart failure.
- ♦ Depression.
- ♦ Fatigue.
- ♦ Bronchospasm (with non selective BB).
- ♦ Heart block.
- ♦ Night mares.
- ♦ Sudden withdrawal →angina.
- ♦ Impotence.





### III- Calcium channel Blockers:

#### Ca.Ch. Blockers are classified into:

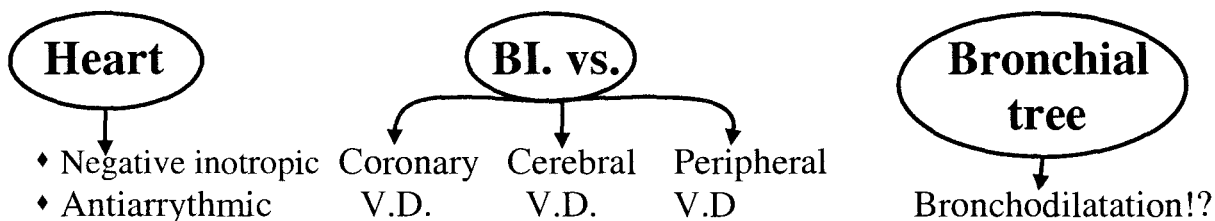
\* Dihydropyridines:

- Nifedipine, Nicardipine. (Short acting)
- Amlodipine. (Long acting)
- Cinnarizine (stugeron), it is cerebral vasodilator.
- Nimodipine (Nimotop), it is used in cases of subarachnoid hemorrhage.

\* Non Dihydropyridines:

- Verapamil.
- Diltiazem.

✎ **Actions** of Ca.Ch.B they ⊖ Ca Influx in:



✎ **Nifedipine:** (Adalat) 10 - 20 mg t.d.s.

1. Vasodilatation  Peripheral.  
 Coronary.
2. No negative inotropic effect.
3. No negative chronotropic effect.

- Can be used in ischemic heart disease with heart block.
- Nifedipine can cause undesirable reflex tachycardia when used alone, so it is better to be combined with BB.

★ **Side effects** ♦ Headache - tachycardia  
 ♦ Peripheral edema

✎ **Nicardipine:** As nifedipine

✎ **Verapamil:** (Isoptin) 80 mg / 8 hr or 240 mg slow released tablets once or twice daily.

1. Mainly antiarrhythmic.
2. Negative inotropic
3. Coronary vasodilator.

So, it can be used in IHD with arrhythmia and not used with heart failure !?

★ **Side effects:** HF – H block – Constipation.



### 🦋 **Diltiazem:** (Delaytiazem)

- ⇒ Dose: 60 - 180 mg / 12 hrs
- ⇒ It is a coronary vasodilator more than verapamil and -ve inotropic less than verapamil.

Slow-release formulations e.g amlodipine can be used once daily with no significant effect on the heart rate and no significant negative inotropic effect.

Nimodipine is a calcium channel blocker, which can be useful in cases of subarachnoid hemorrhage (Nimotop 30 mg tab, 1 – 2 tab/day, 10 mg vial for infusion)

### **Common good drug combinations in treatment of IHD:**

- ➔ Beta blocker + Nitrates.
- ➔ Beta blocker + Amlodipine.
- ➔ Verapamil + Nitrates.
- ➔ Deltiazem + Nitrates.

Nifedipine + Nitrates → Tachycardia + hypotension.

Verapamil + Beta blocker → Heart block + heart failure.

(Verapamil should not be used with BB)

### **IV- Other medications in treatment of IHD:**

- \* Antiplatelet drugs:
  - Aspirine 75-100 mg/12 hrs.
  - Ticlopidine 250 mg/12 hrs.
- \* Hypolipidemic drugs e.g statins (see later).
- \* Treatment of associated anaemia, obesity, DM.

### **Intractable angina**

- ♥ Some patients remain symptomatic despite medication and are not suitable for further revascularization.
- ♥ **Trans myocardial laser revascularization (TMR)**  
i.e Laser is used to form channels in the myocardium to allow direct perfusion of the myocardium from blood present in the ventricular cavity.



# Myocardial Infarction (MI)

It is a complete cessation of coronary perfusion due to formation of occlusive thrombus at the site of rupture or erosion of an atheromatous plaque leading to ischemic necrosis of a localized area of the myocardium.

**Cause:** As angina i.e. atherosclerosis, but there is ruptured atheromatous plaque with superimposed thrombosis

**Risk Factors:** As angina

**C/P:** As angina (with the following differences):

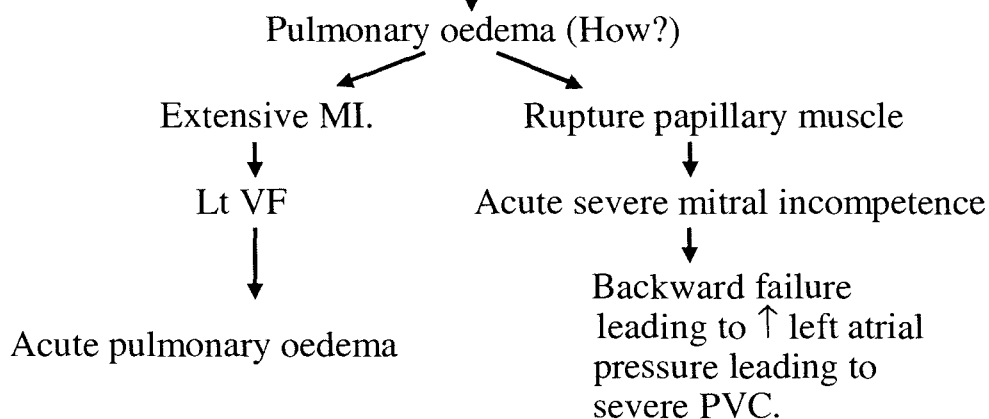
1. **Chest pain** (similar to angina but it is):
  - Severe.
  - At rest – prolonged.
  - Not responding to nitrates.
2. Anxiety (fear of impending death)
3. Sympathetic stimulation → pallor, sweating, ↑ HR.
4. Vagal stimulation → vomiting and bradycardia, this is common with inferior wall infarction. Nausea and vomiting may also due to opiates.
5. Hypotension "may occur specially with the use of nitrates", Sinus tachycardia, fourth H.S and raised jugular venous pressure are common, i.e myocardial dysfunction.
6. Manifestions of complications e.g.: Heart failure + arrhythmia.

**So, sudden onset of chest pain, hypotension, arrhythmia, dyspnea or loss of consciousness direct our attention to MI in patient with positive risk factors.**

## Q- Causes of Painless infarction

- \* Diabetic neuropathy
- \* Infarction during coma
- \* Infarction during anesthesia
- \* Infarction with pulmonary oedema.
- \* Elderly.
- \* Transplanted heart (denervated).

## Q - Patient with myocardial infarction





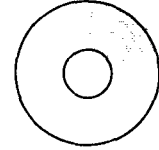
## Pathology:

"The commonest cause is atherosclerosis with ruptured atheromatous plaque with superimposed thrombosis"

### Types:

1- **Transmural infarction** (with superimposed thrombosis)

i.e Infarction of full thickness of the ventricular wall.



2- **Subendocardial infarction** (can occur without superimposed thrombosis)!

I.e.: limited to the inner one third to one half of the ventricular wall. It can be precipitated by hypotension, hypoxia (e.g during anaesthesia).

### Sites:

➤ **Anterior wall infarct** : Occlusion of anterior descending branch.

➤ **Lateral wall infarct** : Occlusion of left circumflex artery.

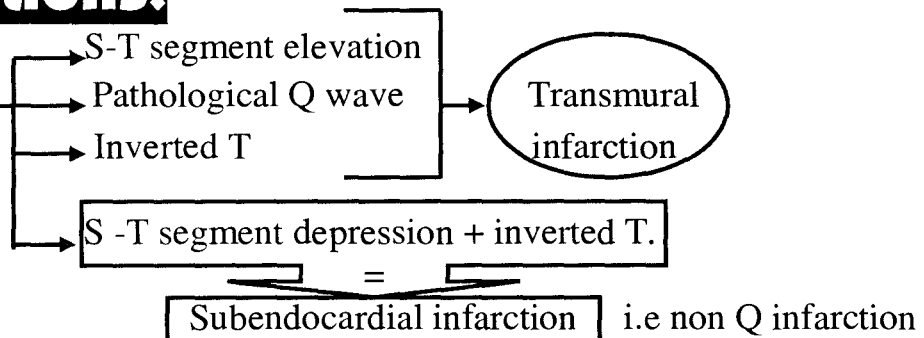
➤ **Inferior wall infarction** : Occlusion of right coronary artery.



## Investigations:

### 1. E.C.G

(It gives changes after 6 hrs)



### 2. Enzymes and other markers:

- ☒ **CK** (Creatine kinase): Onset ( 4-6 hours), Peak (12 hours), Duration (2-3 days). CK may ↑↑ with intramuscular injection and muscle disease (Polymyositis). So, ask for the specific CK enzyme for MI (CK-MB fraction).
- ☒ **AST** (onset 12 hrs, peak 1 day, duration 3 days) and **LDH** enzyme (onset 12 hr, peak 2 days duration 1 week) also elevated in MI.
- ☒ **Cardiac troponins T and I** are very specific for cardiac injury. They are released early (4-6 hours) and can persist for up to 7-14 days.
- ☒ **Myoglobins** also is a recent marker, it can be detected within 2 hours of the onset of MI and remains for 24 hrs!?

3. **TLC** ↑ /**ESR** ↑ /**CRP** ↑ as there is tissue damage.

4. **X ray** May show pulmonary edema.

5. **Echo** Showing VSD, ruptured interventricular septum, pericardial effusion, mitral incompetence. Also it detects ejection fraction, which has a prognostic value.



# ↩ Treatment of myocardial infarction:

## I- ttt of uncomplicated M.I:

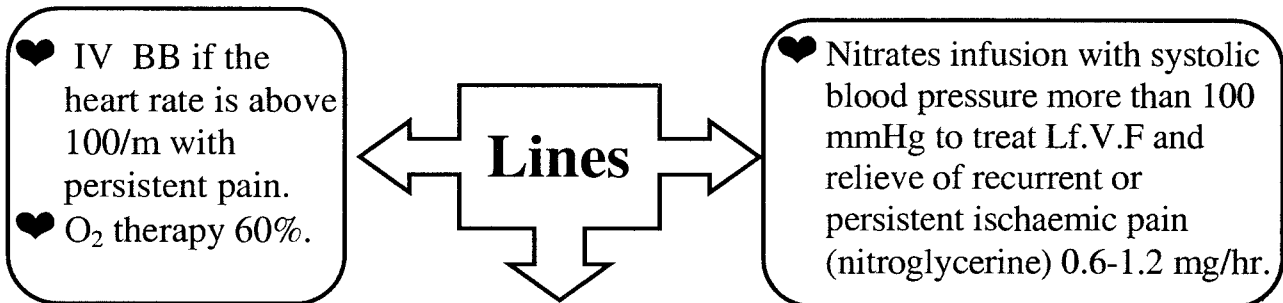
Uncomplicated myocardial infarction means that there is no associated arrhythmia or heart failure. i.e. haemodynamically stable patient.

### Treatment:

- First aid:** ✂ Rest, Reassurance. ✂ O<sub>2</sub> therapy.  
✂ Sublingual nitrates. ✂ Sedation – analgesia.

### **In hospital:**

1. C.C.U. (coronary care unit)                      2. ECG monitoring.
3. Sedation, analgesia by morphine 5-10 mg IV, morphia may lead to bradycardia, heart block, or depression of respiration so, lanoxone must be available.
4. IV cannula with infusion of glucose 5% IV drip very slowly (just to keep patent cannula, avoid excessive infusions).
5. Mini dose heparin or antiplatelets or both, low molecular weight heparin is safe.
6. Metoclopramide IV if required.
7. O<sub>2</sub> 2-4 L/m to maintain O<sub>2</sub> saturation > 90%.
8. Measures to limit the size of infarction or to reverse it within the first 6 hours, as infarction is usually established after 6 hours !?.



### Thrombolytic therapy "Reperfusion":

- ★ **Streptokinase** 1- 1.5 million units in 100 ml saline IV over 1 hour.  
The drug is antigenic and may cause allergy and hypotension.
- ★ **Urokinase**, it is not antigenic and seldom causes hypotension, bolus dose of 15 mg then 0.75 mg/kg over 30 m and then 0.5 mg/kg over 60 m.
- ★ **Recombinant tissue plasminogen activator.**
- ♥ Thrombolytic therapy should be given to patients presenting 6-12 hours of onset of symptoms. After 12 hrs, there is no clear benefit.
- ♥ Heparin infusion should be given 48 -72 hours following the thrombolysis..
- ♥ Aspirin improve the survival with thrombolytic therapy.
- ♥ Circulating streptokinase antibodies are formed after therapy and persist for 5 years or more, this can render subsequent infusion of streptokinase ineffective.  
**PTCA can be done when the patient is stabilized, primary PTCA can be done (see later).**



Primary PTCA without thrombolysis is safe and an effective alternative therapy to thrombolytic therapy in experienced centers especially for patients in whom the hazards of thrombolytic therapy is high.

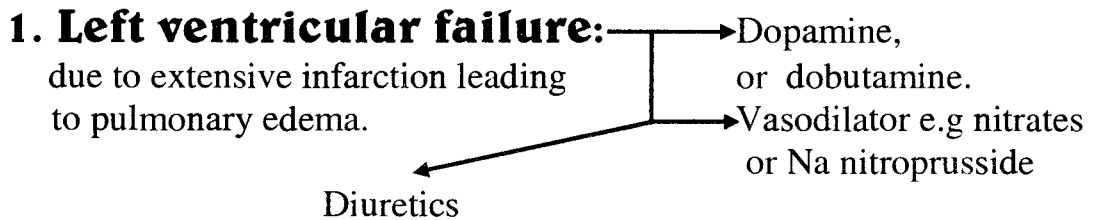
9. ACE inhibitors for reduction of ventricular remodeling after infarction.
10. Adjust serum Mg to reduce risk of arrhythmias.

**☠ Contraindications of thrombolytic therapy:**

1. Major surgery within the previous 2 weeks.
2. Active bleeding from GIT or other non compressible sites.
3. Allergy to the thrombolytic therapy.
4. Recent cerebrovascular stroke.
5. Proliferative diabetic retinopathy.
6. Refractory hypertension (Systolic blood pressure > 180 mm Hg).

**II- ttt of complicated infarction:** (complications of MI and their treatment)

As above + ttt of following complications



**2. Rupture papillary muscle** → acute mitral I → pulmonary edema.

Treated as above + intraortic balloon + valve replacement when the patient is stabilized.

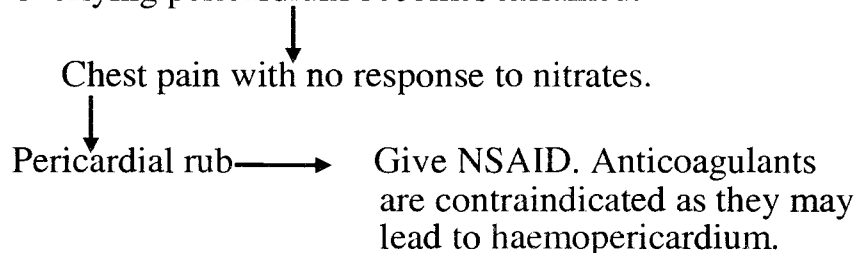
**3. Rupture interventricular septum** → Acute VSD.

Treated as above + intraortic balloon + surgery when the patient is stabilized.

(Rupture IV septum tends to cause right heart failure rather than pulmonary edema !?)

**4. Early Pericarditis** (within 2-3 days)

The overlying pericardium becomes inflamed.



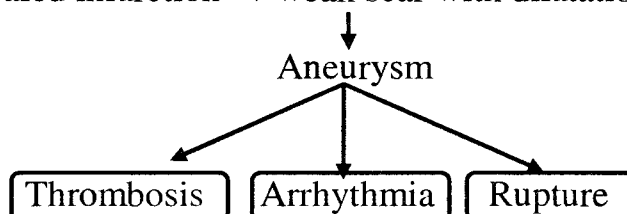
**5. Extension of infarction**

I.e.: Pain↑↑ after initial stabilization, this can be detected by elevation of a new marker of myocardial damage e.g myoglobin, as it can be detected within 2 hours.

**6. Myocardial aneurysm and remodeling**

Diagnosed by persistent S-T segment elevation >2wks.

Pathology: Healed infarction → weak scar with dilatation.





- ttt Anticoagulants.
- Antiarrhythmics.
- Surgery aneurysctomy.

N.B.: Early use of ACE inhibitors reduce the incidence of aneurysm.

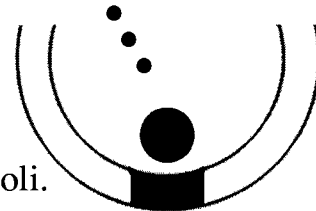
## 7. Rupture of the ventricle may lead to cardiac

**Tamponade**, it is usually fatal although, it may be possible to support a patient with an incomplete rupture until emergency surgery is performed.

## 8. Mural thrombus:

Infarction → Rough surface

└─→ Thrombosis → Emboli.



ttt Anticoagulant.

## 9. Arrhythmia (see later)

- └─→ Ventricular extrasystoles
- └─→ Ventricular tachycardia or fibrillation.
- └─→ AF-Heart block.

10. **DVT** → Pulmonary embolism (see later).

## 11. Late pericarditis (Dressler's syndrome)

This occurs weeks or months after myocardial infarction, the damaged pericardium and myocardium leading to escape of pericardial and myocardial cells to the circulation, this will lead to an autoimmune response → triggering antibodies release → attack the pericardium

ttt Steroids – Anticoagulants are contraindicated, see before.

## 12. Post infarction angina.

## 13. Frozen shoulder:

ttt Physiotherapy.

- 1, 2, 3, 4, 5, 7, 8, 9 are early complications.
- 6, 10, 11, 12, 13 are late complications.

Treatment of  
Post infarction  
Or secondary  
prevention of  
M.I

- ★ Avoid risk factors. e.g. smoking, control blood sugar.
- ★ Nitrates e.g isosorbide mononitrate 20 mg/12h.
- ★ BB e.g metoprolol 50 mg/12hr
- ★ Antiplatelets e.g aspirin 75-150 mg/d.
- ★ ACE inhibitors used with patients with left ventricular dysfunction also useful to reduce remodeling e.g ramipril 2.5 mg/12hr.
- ★ Hypolipidemic drugs e.g simvastatin 20-80 mg/d.
- ★ Coronary angiography must be done for patients with post infarction angina for the possibility of revascularization.



# Cardiac Arrhythmias

## Definition:

It is an abnormality of heart rate or irregularity of the cardiac rhythm, it is usually presented with palpitation, dizziness, sudden death or it may be asymptomatic.

## Notes:

- 1. Normal Heart rate:**
- 60 - 90 /min.
  - < 60 (Bradycardia).
  - > 100 (Tachycardia).

**2. S.A.N = (pace maker):**

It is approximately 1 ½ cm long & 2 - 3 mm wide and supplied by the sinus node artery from right coronary (60 %) or left circumflex (40%).

It has the highest rhythm = (120/min)  $\xrightarrow{\text{Vagal effect}}$  So, the heart rate is around 60-90 /M

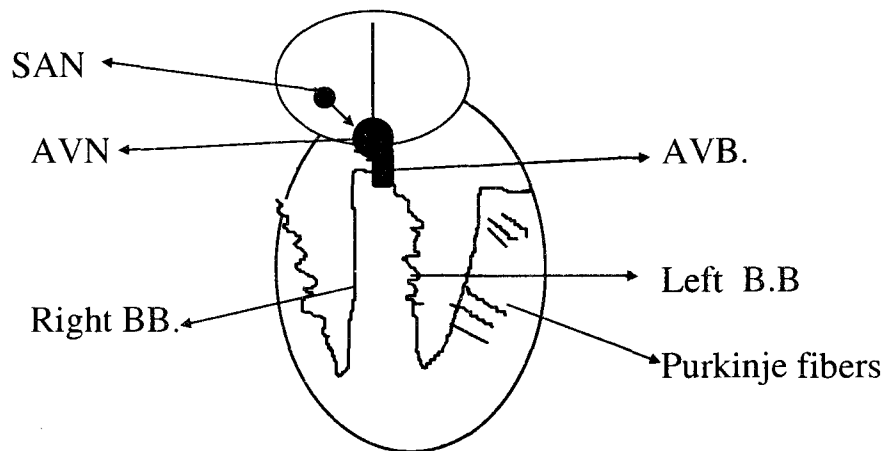
**(SAN) characterized by:**

Automaticity, i.e Ability to generate impulses.

So, nerve supply of the heart aims at regulation of heart rate & not initiation of rhythm.

**3. A.V.N:**

It has a physiological delay, to give chance of the atrium to contract before the ventricle. This physiological AV block is useful to protect the ventricle from any tachyarrhythmia arising from the atrium.



Impulses from SAN  $\rightarrow$  excite atria & then to  
 $\downarrow$   
 AVN (slow conduction)  $\rightarrow$  AVB  $\rightarrow$  Rt & Lf BB  $\rightarrow$   
 purkinje fibres  $\rightarrow$  ventricular wall.

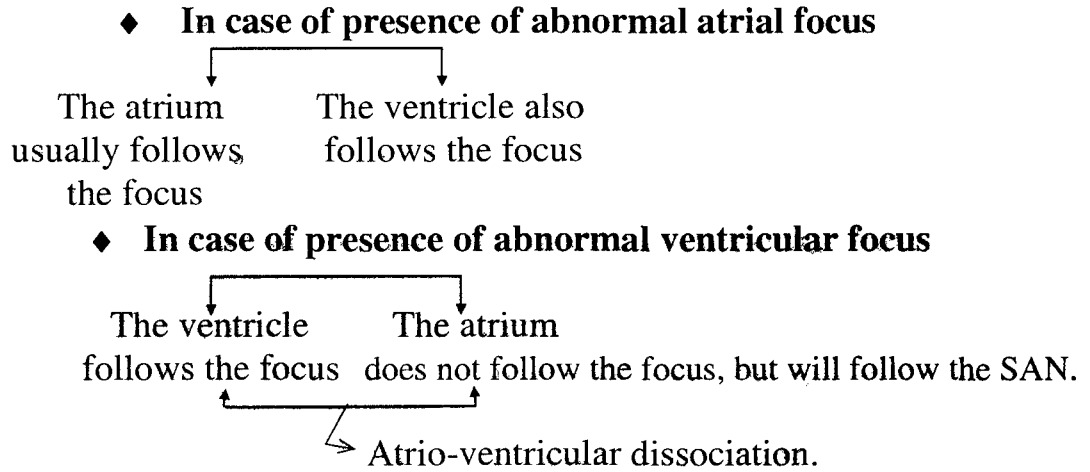




**AVN:**

- 1 Does not allow > 160 - 180 impulse/m to pass to ventricles !?
- 1 Digitalis increases AV block, so it is useful for atrial tachyarrhythmias

**5. Usually there is no retrograde conduction:**



**6. Respiratory sinus arrhythmia:**

- \* Inspiration → VR ↑ → ⊕ SAN → HR ↑.
- \* This is a physiological process indicating that the pace maker is the SAN

**7. Carotid sinus massage:**

Vagal stimulation

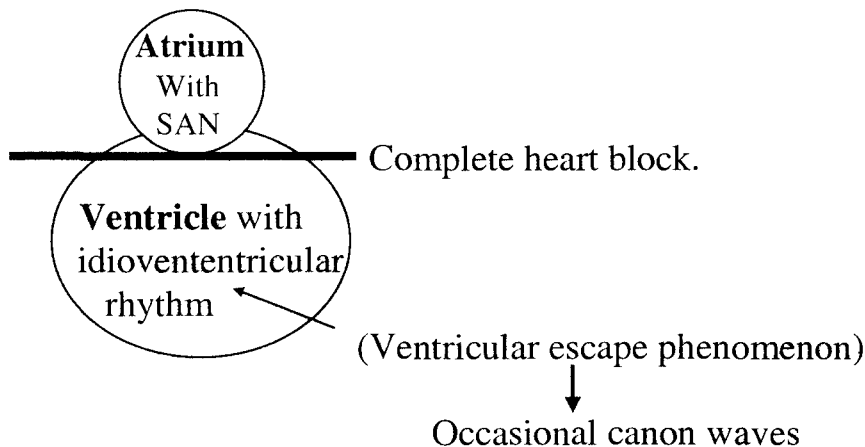
So, if arrhythmia disappears with carotid massage, this indicates that the focus is within the atrium because the atrium is supplied by the vagus.

**8. Cannon wave:**

It is due to atrial contraction during ventricular systole.

This occurs when there is complete atrio-ventricular dissociation.  
e.g. Complete heart block

The atrial contraction during ventricular systole occurs occasionally.





**9. The awareness of heart beats is variable:**

Some patients with atrial fibrillation may be asymptomatic

Some patients feel every extrasystole.

**Mechanisms of arrhythmia**

**① Accelerated automaticity**

Normally there is slow depolarization during diastole. This mechanism leads to increase the rate of diastolic depolarization.

This occurs in sinus tachycardia, escape rhythm (Idioventricular rhythm) and accelerated AV nodal rhythm.

**② Triggered activity by digitalis, catecholamines or myocardial damage. this occurs in ventricular arrhythmia or atrial tachycardia induced by digitalis toxicity.**

**③ Re - entry (circus movement) in the form of wave of depolarization forced to travel around a ring of cardiac tissue. This occurs in cases of paroxysmal atrial tachycardia, junctional tachycardia, atrial flutter and fibrillation.**

**General Scheme for arrhythmia**

**\* Patho-physiology:**

According to the type of the arrhythmia

**\* Aetiology:**

**Pathological**

- ♦ Ischemic heart disease.
- ♦ Rheumatic heart disease.
- ♦ Congenital heart disease.
- ♦ Constrictive pericarditis.
- ♦ Heart failure.

**Functional**

- |   |  |
|---|--|
| <p>H.R. ↑</p> <ul style="list-style-type: none"> <li>♦ Anxiety.</li> <li>♦ Exertion.</li> <li>♦ Pregnancy.</li> <li>♦ Fever.</li> <li>♦ Excessive coffee</li> <li>♦ Thyrotoxicosis.</li> <li>♦ Anaemia.</li> <li>♦ Catecholamine excess.</li> </ul> | <p>H.R. ↓</p> <ul style="list-style-type: none"> <li>♦ Sleep.</li> <li>♦ Athletes.</li> </ul> <p>↑↑ Vagal tone</p> |
|---|--|

**Drugs**

⏏ ↓↓ H. R. → Digitalis - Ca. Ch. B. (Verapamil)  
→ BB.

⏏ ↑↑ H. R. → Atropine - thyroxine, vasodilators e.g nefidipine, hydralazine and sympathomimetic drugs.



**\* C/P:**

***Symptoms:*** (comment on the following):

**1 Palpitation:**

- ♦ Onset.
- ♦ Duration.
- ♦ Offset.
- ♦ Regular or irregular.
- ♦ What increase and what decrease.

**2 ↓ COP symptoms during arrhythmia:**

↓ COP symptoms up to syncope may occur if there is

- Severe tachycardia (> 160 / m)
- Heart block (see later)

**3 Manifestations of the cause:**

- Rheumatic heart disease.
- Ischemic heart disease.
- Congenital heart disease.

***Signs:*** (Comment on the following)

\* **Pulse rate, rhythm:**

- Regular irregularity (certain types of extrasystoles)
- Irregular irregularity (AF)

\* **Blood pressure:**

- Low blood pressure (↓ COP), this occurs usually with severe tachycardia or heart block.
- Systolic hypertension in complete heart block.

\* **Carotid sinus massage:** if +ve (effective), this means that the source of arrhythmia is the atrium.



\* **Respiratory sinus arrhythmia:** if present, this means that SAN is the pace maker.

\* **Neck veins:**

- ✓ Cannon wave = A - V dissociation.
- ✓ Loss of A wave in atrial fibrillation.

***Local examination of the heart***

- Signs of the cause → e.g. Murmur of MVD or AVD.
- Cannon sounds in cases of A-V dissociation.
- First heart sound is usually loud with tachycardia.

**Investigations:**

**1 ECG:** showing tachyarrhythmia or bradyarrhythmia or irregularity.

If the arrhythmia is paroxysmal → ask for ambulatory ECG (**Holter**).

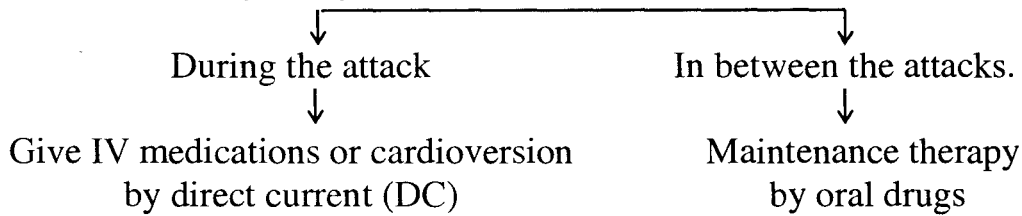
**2 Echo** for detection of the cause as rheumatic heart or congenital heart disease..

**3 Laboratory investigations** for the cause e.g. (T3, T4, TSH), Hb, serum K, Mg.



**\* Treatment:**

**1. If the arrhythmia is paroxysmal:**



**2. Non paroxysmal** → maintenance oral therapy from the start.

**Tachyarrhythmia**

**1. Sinus tachycardia**

**↳ Patho-physiology:**

SAN discharges impulses > 100 / minute, usually around 100 – 160/m.

**↳ Aetiology:**

**1. Functional:**

> Stress, pregnancy, fever, anaemia, thyrotoxicosis, excessive coffee, smoking

**2. Pathological:**

> Heart failure. (Compensatory tachycardia)

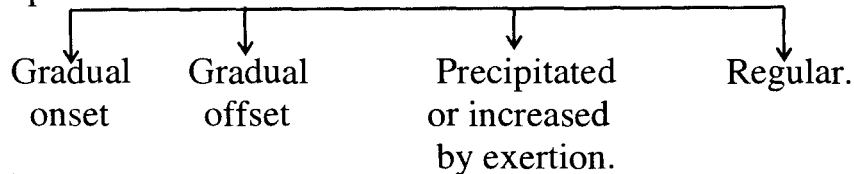
**3. Drugs:**

- Atropine.
- β<sub>2</sub> agonist.
- Thyroxine.
- Vasodilators.
- Sympathomimetics

**↳ C/P:**

**↳ Sympt:**

**① Palpitation:**



**②** ↓↓ COP symptoms are usually not present.

**③** Manifestations of the cause e.g anaemia, thyrotoxicosis.

**↳ Signs:**

1. Tachycardia ↑ > 100/m with regular rhythm.
2. Blood pressure is almost normal.
3. Carotid sinus massage is positive (effective) → ↓ HR.
4. Respiratory sinus arrhythmia is present. "As the SAN is the pace maker."
5. Neck veins:
  - Rapid - normal.
  - No cannon waves.



## Local exam:

1. HS: loud first heart sound, it is muffled with HF.
2. Signs of heart failure, for example S<sub>3</sub> gallop on the apex.

## Investigations:

1. ECG (sinus rhythm, HR > 100/m).
2. (T3, T4, TSH), Hb.
3. Echo.

## Treatment:

- \* Usually no required treatment (it should not be treated as a primary arrhythmia).
- \* You can give BB (If the patient is markedly symptomatic)
- \* In cases of heart failure you can give BB very gradually as before.
- \* Treatment of the cause.

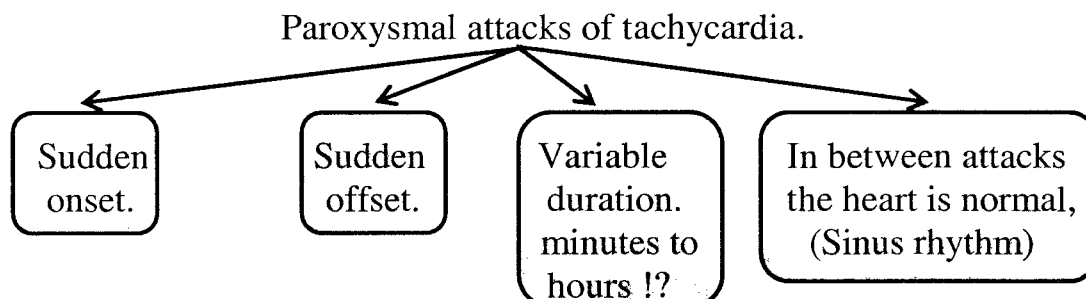
# 2. Paroxysmal atrial tachycardia (PAT)

(Supraventricular tachycardia)

## Patho-physiology:

Due to abnormal focus in the atrium ↻  
 Which discharges impulses more than SAN up to 200/m or more (regular).  
 ↓  
 The heart neglects the SAN and follows the focus.  
 ↓  
 Since, this focus is unstable So, it will disappear after a time.  
 ↓  
 Then the heart returns again to follow the SAN.

So the net result:



## Aetiology:

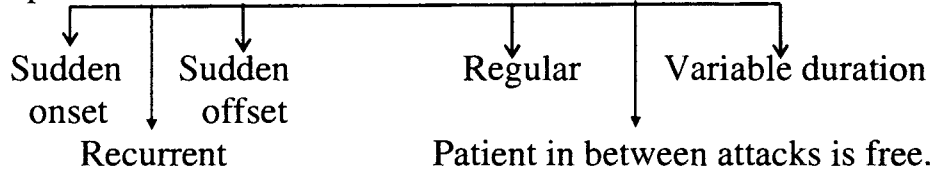
1. Functional: e.g stress, smoking, excessive coffee, thyrotoxicosis.
2. Pathological: as before e.g rheumatic heart disease.
3. Drugs causing tachycardia e.g. sympathomimetics.



**C/P:**

⇒ **Symptoms:**

① Palpitation:



② ↓↓ COP usually present.

③ Manifestations of the cause.

④ Polyuria may occur after the attack (release of ANP !?).

⑤ Chest pain may occur due to ↓ coronary filling.

⇒ **Signs:**

- ♦ Tachycardia up to 200 /m or more, regular
- ♦ Blood pressure may be low due to decreased heart filling
- ♦ Carotid sinus massage: may be effective (focus in the atrium) → ↓ HR.
- ♦ Respiratory sinus arrhythmia is not present (SAN is not the pace maker)
- ♦ Neck veins: Rapid venous pulsations, no cannon waves.

⇒ **Local exam:** First heart sound is loud.

**Investigations:**

① ECG: Abnormal P wave and ↑ HR up to 200/m or more (during the attack).

② Holter ECG. ③ Echo-cardiography. ④ (T3, T4, TSH), Hb.

**Treatment:**

(The arrhythmia is paroxysmal)

① During the attack:

1. Vagal stimulation by:
  - ↗ Carotid sinus massage.
  - ↘ Pressure on eye globe.
  - ↘ Gaging reflex.
2. Drugs: →
  - IV verapamil, 5-10 mg IV with saline by infusion very slowly (under ECG monitoring).
  - \* Side effects: hypotension, give antidote, (Ca gluconate), it is also negative inotropic.
  - Recently adenosine IV injection (advantage: No -ve inotropic effect)**
3. DC shock: it is rarely used in this case.

Also we can use during the attack

- BB IV (second choice after verapamil and adenosine).
- Digitalis IV (slower onset of action, usually it is not used during attack!?)

② In between attacks: treatment of the cause plus one of the following drugs:

Select according to the patient condition

- ① Digitalis is good with associated systolic dysfunction.
- ② BB is good if it is stress related.
- ③ Verapamil is good with associated IHD.



## Junctional or AV nodal tachycardia

**Causes:** similar to PAT.

- C/P:**
- ◆ Palpitation (sudden onset, sudden offset)
  - ◆ Chest pain (↓ coronary filling).
  - ◆ Polyuria, as tachycardia → ↑ atrial pressure → release of ANP.
  - ◆ HR is about 140 – 200 /m or more
  - ◆ Regular canon waves.
  - ◆ ECG (P waves are usually obscured)

**TTT:** As PAT.

## 3. Paroxysmal ventricular tachycardia (PVT)

### ↳ Pathophysiology:

Abnormal focus in the ventricle, discharge impulses more than SAN rate up to 200/min or more, (Regular). Since, the focus is within ventricle, and there is no retrograde conduction. So, ventricle will follow the focus and atrium will follow the SAN.



Atrio-ventricular dissociation.

The focus appears suddenly and disappears suddenly so, the heart returns back to sinus rhythm.

### ↳ Aetiology:

\* Diseased heart is usually present.

Ischemic heart disease  
e.g myocardial infarction.

Congenital heart disease

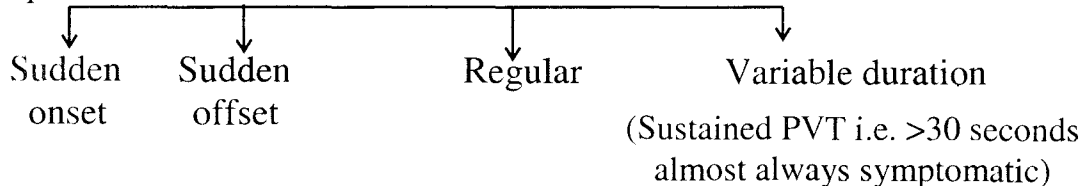
Rheumatic heart disease

\* Digitalis toxicity

### ↳ C/P:

#### ⇒ Symptoms:

① Palpitation:



② Low COP usually present.

③ Manifestations of the cause, e.g IHD.

Cardiac arrest may occur i.e. PVT converted to ventricular fibrillation.



### ⇒ Signs:

- ♦ Tachycardia up to 200/m or more (Regular)
- ♦ Blood pressure is low due to decreased COP.
- ♦ Carotid sinus massage: it is not present.
- ♦ Respiratory sinus arrhythmia: it is not present.
- ♦ Neck veins: Occasional cannon (A-V dissociation)

⇒ **Local exam:** Canon sounds.

### ⇒ Investigations:

- ① ECG: (regular, rapid bizarre shaped complexes)  
Holter, ECG monitor can be used.
- ② Echocardiography.
- ③ Serum K (for ↓ k) and Mg (for ↓ Mg).

### ⇒ Treatment: Ventricular tachycardia is paroxysmal so:

#### During the attack

1. DC if the patients is hemodynamically Compromised e.g (hypotension, pulm edema).
2. Lignocaine 50-100 mg IV then 4 mg/m for 30 m, then 2 mg/m for 2 hrs, then 1 mg/m for 24 hrs. It can be used if DC is not available or if the arrhythmia is well tolerated (hemodynamic stability).  
Procainamide can be used as an alternative.

#### In between attacks

1. Cause.
2. Amiodarone (see later).
3. Implantable cardioverter defibrillator.

Torsades de pointe is a form of polymorphic PVT (see ECG), it may degenerate into v. fibrillation.

## 4. Atrial Flutter

**Patho-physiology :** Ectopic focus in the atrium → discharge rapid & regular impulses 250- 350 /min. So the presentation will be according to the A -V block.

i.e (The ventricular response).

Atrial flutter is a short lived rhythm often degenerating into atrial fibrillation, or may return to sinus rhythm.

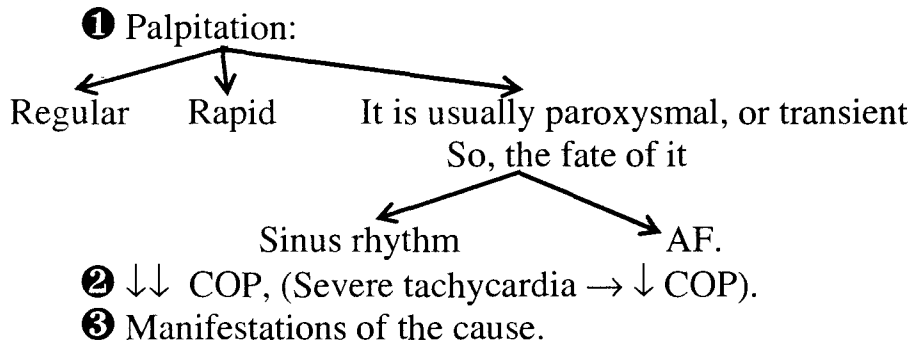
⇒ **Aetiology:** Usually organic heart disease, Typically, the ventricular rate is half the atrial rate i.e 150/m (2 : 1 AV block).





**C/P:** According to A-V block.

⇒ **Symptoms:**



Although atrial flutter is associated with lower risk of embolisation than AF, prophylactic anticoagulant therapy is essential.

⇒ **Signs:**

- ♦ Regular tachycardia.
- ♦ Blood pressure is usually low with severe tachycardia.
- ♦ Carotid sinus massage is effective.
- ♦ Respiratory sinus arrhythmia: is negative.
- ♦ Neck veins:
  - ☞ Venous pulsations are rapid, multiple A waves before each V wave.
  - ☞ No cannon waves.

⇒ **Local exam:** Loud first heart sound. Murmur of heart lesion.

⇒ **Investigations:** - ECG → Sawtooth appearance (Flutter waves).  
- Echo .

⇒ **Treatment:** ➤ DC shock.  
➤ Verapamil, BB or digitalis to decrease the ventricular response.

**Q. Atrial tachyarrhythmia**

- |                        |                        |
|------------------------|------------------------|
| ♥ Atrial fibrillation. | ♥ Atrial tachycardia.  |
| ♥ Atrial flutter.      | ♥ Atrial extrasystole. |

**Q. Ventricular tachyarrhythmia**

- |                             |                            |
|-----------------------------|----------------------------|
| ♥ Ventricular extrasystole. | ♥ Ventricular tachycardia. |
| ♥ Ventricular fibrillation. |                            |

# 5. Atrial fibrillation (AF)

⇒ **Definition:**

- ♦ It is an irregularly irregular arrhythmia in which there is no ordered contraction of the atria producing an irregular ventricular response, the rate of which depends on the A-V conduction.
- ♦ It is the most common sustained cardiac arrhythmia (0.5 % of adult population).



## ↳ Patho-physiology:

Multiple foci in the atrium discharge impulses, very rapid 400-600/m.(irregular)



The degree of A-V block determines the ventricular response.

So, in A F. heart rate: ➤ May be rapid.

➤ May be slow ( slow AF= AF with HR < 60/min), this may occur in digitalized patient (digitalis toxicity).

- Ineffective atrial contraction and left atrial dilatation predispose to stasis and may lead to thrombosis and systemic embolism.
- Loss of atrial contraction leading to decrease of ventricular filling → ↓ COP, also it leads to rise of atrial pressure with aggravation of PVC with development of pulmonary edema in cases of MVD.

## ↳ Aetiology:

1. Rheumatic heart disease, especially MS.
2. IHD.
3. Congenital heart disease e.g. ASD.
4. Constrictive pericarditis.
5. Thyrotoxicosis.
6. Hypertension.
7. Lone AF.
8. Pulmonary embolism.
9. Infective endocarditis.
10. Normal heart e.g (stress following surgery).
11. Alcohol abuse (holiday heart \$)
12. Wolff Parkinson white syndrome.

## ↳ C/P:

① Palpitation:

Irregular      At rest      Increased with exertion.

② ↓ COP may be +ve.

③ Manifestation of the cause.

## ↳ Signs:

- ♦ Pulse: irregular irregularity, persistent with exertion.
- ♦ Pulse deficit (Apical- radial) is usually > 10 /min.
- ♦ Blood pressure is variable (measure the blood pressure 3 times and take an average).
- ♦ Carotid sinus massage is negative.
- ♦ Respiratory sinus arrhythmia is negative.
- ♦ Neck veins: Absent A wave.

## ↳ Local examination:

- First H.S is of variable intensity
- Murmur of valve lesion.

## ↳ Investigations:

- ① ECG: Irregular rhythm - absent P wave
- ② Echo-cardiography for heart lesions e.g MVD.
- ③ T3, T4, TSH.



### **Types of AF.:**

1. **Paroxysmal AF:** i.e. discrete self terminating episodes.
2. **Persistent AF:** i.e. prolonged episodes that can be terminated by electrical or chemical cardioversion.
3. **Permanent or chronic A.F:** i.e. sinus rhythm can not be restored
  - ★ Permanent AF is usually preceded by bouts of paroxysmal AF and one or more episodes of persistent AF.
  - ★ 50 % of paroxysmal AF can occur in normal heart.
  - ★ 20 % of persistent or permanent AF can occur in normal heart.

### **Complications of AF:**

- (1) Thromboembolism.
- (2) Angina due to  $\uparrow$  HR and  $\downarrow$  COP.
- (3) It is a precipitating factor of heart failure and pulmonary edema in presence of pre-existing heart disease.

### **Treatment:**

#### **1. Paroxysmal AF:**

- ★ Occasional attacks that are well tolerated do not necessarily require treatment.
- ★ BB or flecainide can be used if symptoms are troublesome, BB may have particular advantages in patients with underlying structural heart disease or hypertension and in those who develop AF during exertion or stress.

#### **2. Persistent AF:**

- ★ DC cardioversion after the administration of IV heparin is appropriate if AF has been present for less than 48 hours. Also IV flecainide is a safe and an alternative to electrical cardioversion.
- ★ It is better to do cardioversion after the patient becomes on warfarin for at least 3 weeks (initiate with heparin and then switch to warfarin).
- ★ Anticoagulation should be maintained for at least 1 month and ideally for 6 months following successful cardioversion.

Attempts to restore sinus rhythm are most successful if AF has been present for less than 3 m, with young patient and with no structural heart disease.

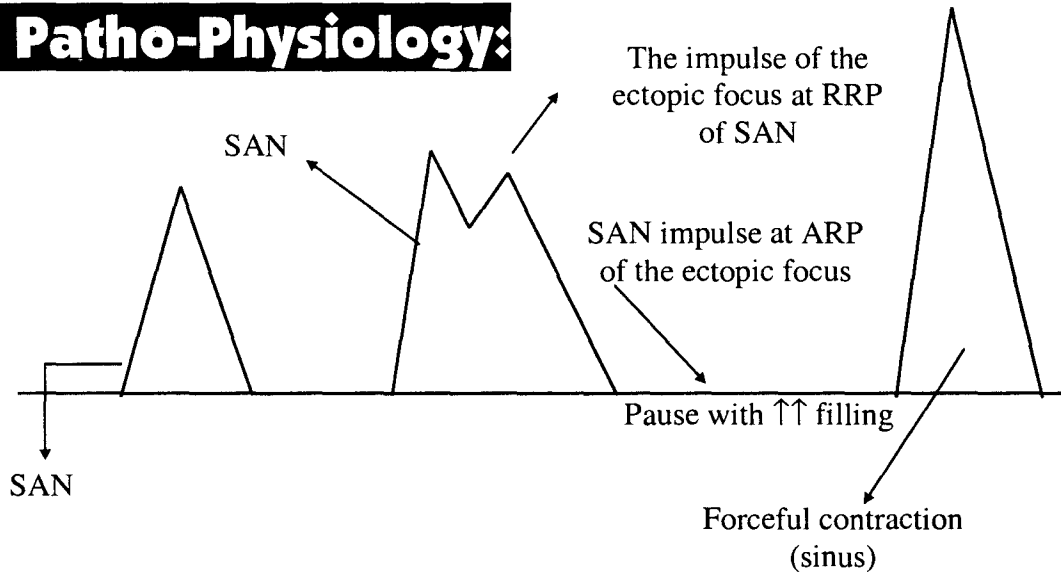
#### **3. Permanent or chronic AF:**

- ★ Digoxin, BB, or Ca Ch B e.g. verapamil or diltiazem to maintain appropriate heart rate ( $\downarrow\downarrow$  ventricular response by  $\uparrow$  the degree of AV block). BB and the above Ca Ch. B. are more effective than digoxin in controlling the heart rate during exercise and have additional benefits in hypertension and structural heart disease.
- ★ Anticoagulant to prevent thromboembolism.
- ★ Combination therapy (digoxin + atenolol) can be used !?



# Extrasystoles (premature beats)

## Patho-Physiology:



Ectopic beats produce a low stroke volume (short time of diastole with ↓ filling) so, ventricular contraction is premature and ineffective.

### \* C/O:

- \* Extra beat.
  - \* Dropped or missed beat.
  - \* Strong beat.
- } occasionally

## Aetiology:

1. Functional e.g stress, smoking, excessive coffee, ↓ Mg.
2. Pathological e.g rheumatic heart disease.
3. Drugs: e.g sympathomimetics, digitalis toxicity.

## C/P:

### ⇒ Symptoms:

#### ① Palpitation:

Irregular    At rest    The irregularity disappears with exertion.

#### ② Manifestations of the cause.

### ⇒ Signs:

1. Pulse:
  - Irregular, (occasional irregularity), regular irregularity in certain types e.g pulsus bigeminy or trigeminy.
  - The irregularity disappear with exertion.
  - Pulse deficit is usually < 10/m.
2. Carotid sinus massage: → insignificant.



3. Neck veins: normal waves with occasional irregularity.

⇒ **Local examination:** S1 variable intensity.

⇒ **Investigations:**

- ECG showing: extrasystole which is either
  - ↘ Atrial.
  - ↘ Ventricular.
- Echo.
- T<sub>3</sub>, T<sub>4</sub>, TSH – K – Mg.

⇒ **Treatment:**

⇒ **Atrial extrasystoles:**

- Most cases are asymptomatic, treatment is not required.
- When they cause palpitations or trigger PAT, treatment may be useful.
- Elimination of the precipitating factors e.g alcohol, tobacco or adrenergic stimulants.
- Mild sedation or the use of BB can be tried.

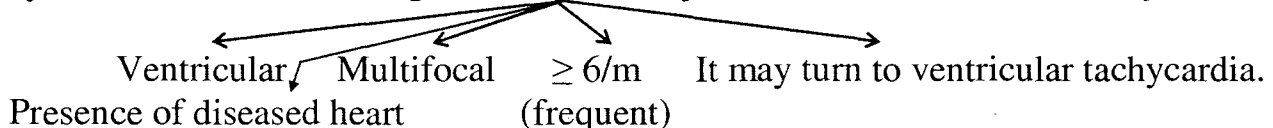
⇒ **Ventricular extrasystoles:**

- In absence of cardiac disease with isolated asymptomatic cases, no treatment is required.
- Symptomatic cases can be treated with sedatives if no response, BB can be used especially with extrasystoles induced by stress or due to thyrotoxicosis or mitral valve prolapse. Also it is important to eliminate alcohol, tobacco,....
- Frequent, multifocal ventricular extrasystoles with organic heart disease must be treated e.g by BB, verapamil. Amiodarone or procainamide are occasionally necessary.

**Important terms in extrasystoles:**

- ✓ **Unifocal extrasystoles** (identical beats arising from a single ectopic focus)
- ✓ **Multifocal extrasystoles** (varying morphology with multiple foci)
- ✓ **Couplet and triplet:** are terms to describe two or three successive ectopic beats.
- ✓ **Bigeminy** means a run of alternate sinus and ectopic beats.

**Q - What is the dangerous extrasystole (ttt is mandatory)**



**Q - Importance of neck veins examination in cases of arrhythmia.**

- |                        |                          |
|------------------------|--------------------------|
| 1- AF – Atrial fultter | 2- Extrasystoles.        |
| 3- V. Tachycardia.     | 4- Complete heart block. |



# Bradyarrhythmia

## 1. Sinus bradycardia

### Patho-physiology:

The SAN discharges, impulses < 60/m.

### Causes:

- 1- Physiological:
  - During sleep.
  - Athletes.
- 2- Obstructive Jaundice. → Bile salts → ⊖ SAN or causing AV block.
- 3- Hypothyroidism, ↑ ICT.
- 4- Drugs:
  - B.B.
  - Ca. channel blocker. e.g Verapamil.
  - Digitalis.

### C/P:

- 1- It is usually asymptomatic.
- 2- Manifestations of the cause.

### O/E:

- ♦ Pulse < 60 / m, regular. Blood pressure is normal.
- ♦ Neck veins showing no abnormality (just slow waves).
- ♦ Respiratory sinus arrhythmia is present.

### Investigations:

- ECG → ↓ HR + sinus rhythm.
- T<sub>3</sub>, T<sub>4</sub>, TSH.

### Treatment:

- Treatment of the cause.
- Symptomatic cases e.g hypotension is an indication of treatment.
  - ✓ Vagally mediated bradycardia may respond to atropine.
  - ✓ B-agonist isoproterenol is also effective.
  - ✓ A temporary pace maker is rarely required until treatment of the cause.

No drugs are effective and safe for the long term treatment of this disorder, so permanent pace maker is the treatment of choice in cases with chronic, symptomatic sinus bradycardia.

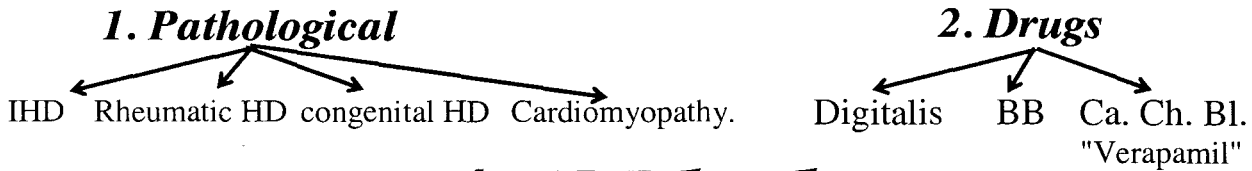
## 2. Heart block

### Types:

- 1- Sino atrial block → Failure of SAN to stimulate the atrium.
- 2- B.B.B
  - Right BBB. "Wide splitting of S2, ECG findings." (See ECG), asymptomatic
  - Left BBB. "Reversed splitting of S2, ECG findings." (See ECG), It is caused by AS, hypertension, IHD.
- 3- A-V Block → Three degrees (see below)



## Causes of A-V block



# A-V Block

### **First degree heart block (A-V block)**

It is an ECG finding (prolonged P-R interval, > 0.2 second)

- ★ All impulses from SAN can be conducted to the ventricles.
- ★ It is not dangerous and may occur physiologically during sleep and in persons with high vagal tone e.g.: athletes. It is asymptomatic.

👉 **Significance of diagnosis:**

1. Direct your attention to the cause → See before
2. Avoid some medications !? in these patients, such as:
  - \* Digitalis.                      \* BB.                      \* Ca.Ch. blocker.

### **Second degree heart block (A-V block)**

It is a partial heart block, So, some impulses fail to pass from atrium to ventricle.

👉 **Types:**

- |   |                  |
|---|------------------|
| <ol style="list-style-type: none"> <li>① Mobitz type I.</li> <li>② Mobitz type II.</li> </ol> | <p>→ See ECG</p> |
|---|------------------|

Mobitz Type I	Mobitz Type II
Progressive lengthening of P-R interval followed by dropped complex (Wenckebach's phenomenon). This may occur physiologically e.g during sleep in athletes.	The number of P waves > QRS. The PR interval of the conducted impulses remains constant but some P wave are not conducted.

👉 **C/P:**

\* Bradycardia.\* ↓ COP.\* It may be asymptomatic.\* Adam's stock attacks

👉 **Adam's stock attacks:** (Episodes of ventricular asystole)→recurrent syncope

- This occurs in cases of 2<sup>nd</sup> degree heart block mobitz type II and in cases of sinoatrial block. (i.e. no impulses will pass from atrium to the ventricles) So, this leads to episodes of ventricular asystole "recurrent syncope."
- When the heart starts beating again the patient regains his consciousness. These attacks can also occur in complete heart block as the idioventricular rhythm is unstable.



**Investigations:**

ECG, the types, (see above).

**Treatment:**

- \* Cause.
- \* Pace maker.
- \* Atropine → enhance conduction

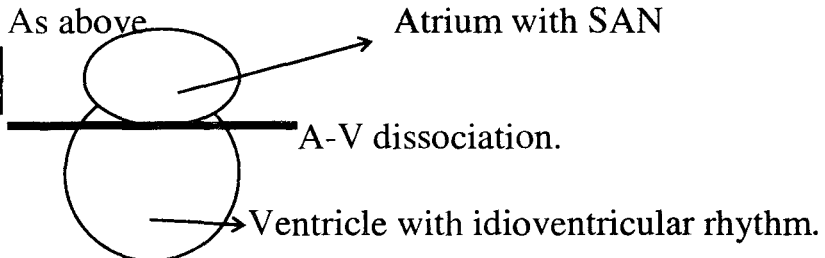
**Complete heart block**

**Causes:**

As above

Atrium with SAN

**Pathology:**



**C/P:**

- Manifestations of the cause.
- Palpitation. (Regular - slow)
- ↓ COP - Adam's stock attacks (recurrent syncope).

**Signs:**

- ♦ Bradycardia, the pulse rate is 30-40/mg, regular.
- ♦ Systolic hypertension may occur (according to the myocardial state); this is due to compensatory increase in stroke volume up to occurrence of functional murmur (high flow murmur)
- ♦ Neck veins: Cannon waves (occasional)

**ECG:**

- Regular bradycardia.
- A-V dissociation.
- QRS Complex less than P waves.

**Treatment:**

- ⊕ Cause.
- ⊕ The best: → Pace maker
- ⊕ Atropine.

**Wolff Parkinson white \$:**

- There is abnormal band of myocardial tissue which connects the atria and ventricles and can by pass the AVN (additional pathway).
- In normal sinus rhythm conduction takes place partly through AVN and through the additional pathway → short P-R interval. Paroxysms of tachycardia may occur. AF is potentially a very dangerous arrhythmia in these patients and may cause collapse.
- **Treatment:** indicated in symptomatic patients by amiodarone as it decreases conduction through the accessory pathway. Digitalis and verapamil increase conduction through the accessory pathway so they are not used.
- It can be treated by radio frequency ablation of the abnormal pathway.





## Sick sinus syndrome: (sinoatrial disease):

- ⊕ It is a disease of SAN due to degenerative changes, ischaemia, it may lead to:
  - Sinus bradycardia.
  - Sinus arrest (syncope).
  - Paroxysmal tachycardia.
  - Paroxysmal AF.
- ↪ **C/P.** Palpitation, dizziness or syncope i.e Adams-stokes attacks.
- ⊕ Treatment: Pace maker.

## *Drugs and other Managements Available For Arrhythmias*

**Class I**  
(Sodium  
channel  
blockers)

- ↘ a- Quinidine - procainamide
- ↘ b- Lignocaine - phenytoin.
- ↘ c- Flecainide.

**Class II** → Beta blockers.

**Class III** → Amiodarone (It prolongs the action potential).

**Class IV** → Calcium channel blocker (verapamil), prolongs action potential.

## (Rules for use of Antiarrhythmic drugs)

### 1. Calcium Channel Blocker: "Verapamil"

- ⇨ It is good for atrial arrhythmia (it blocks the atrial foci and causing heart block), especially in supraventricular tachycardia. It can be used for ventricular ectopic. It should not be used with BB.
- ⇨ In cases of supraventricular tachycardia give 5-10 mg IV during attack, also it can be used between attacks (see before).

### 2. Lignocaine:

- ⇨ It is used mainly for ventricular tachycardia.
- ⇨ Dose : see before.
- ⇨ Side effects:
  - Convulsions.
  - Confusion.

### 3. Phenytoin: "antiepileptic"

- ⇨ As lignocaine.
- ⇨ But it is the drug of choice in treatment of V tachycardia induced by digitalis



#### 4. **B.B.:**

- ⇨ Used in stress, exercise or thyrotoxicosis induced arrhythmia. e.g. Sinus tachycardia, extrasystoles, and supraventricular tachycardia.
- ⇨ Atenolole (25-200 mg/d), bisoprolole (5-10 mg/d).
- ⇨ BB can be given IV for supraventricular tachycardia.

#### 5. **Quinidine:**

- ⇨ Used in atrial or ventricular arrhythmia.
- ⇨ Used in AF.
- ⇨ Side effects are allergy, cinchonism (nausea, vomiting, tinnitus).

#### 6. **Amiodarone:**

- ⇨ It is a broad spectrum antiarrhythmic drug.
- ⇨ **Mechanism:** It blocks:
  - Ca channels.
  - K channels.
  - Na channels.
- ⇨ **Action:**
  - Antiarrhythmic.
  - Coronary vasodilator.
- ⇨ **Uses:**
  - Supraventricular tachycardia.
  - Ventricular tachycardia.
  - Extrasystoles.(it is indicated in multifocal frequent ventricular extrasystoles) to avoid the development of ventricular tachycardia specially in diseased heart.
- ⇨ **Dose:**
  - Oral 600-1200 mg within 24 hrs initially, then 200 mg/d.
  - I.V 5 mg/kg over 20-120 m, then up to 15 mg/kg/24 hrs.
- ⇨ **Side effects:**
  - Corneal deposits.
  - Disturbance of thyroid function.
  - Pulmonary infiltrates.

#### 7. **Adenosine:**

- ⇨ For SVT give 3 mg IV over 2 seconds, followed if necessary by 6 mg, then 12 mg at intervals of 1-2 minutes.
- ⇨ It blocks automaticity and A-V conduction. (Transient A-V block like carotid sinus massage)
- ⇨ It is contraindicated in heart block.
- ⇨ Flushing, dyspnea, chest pain are common side effects.
- ⇨ Also no -ve inotropic action so, it can be used with heart failure.
- ⇨ It terminates supraventricular tachycardia.



Adenosin causes transient AV block in AF and atrial flutter but in ventricular tachycardia gives no response so, it can be used to differentiate these conditions.

## ➤ Pace makers

♥ External energy sources can be used to stimulate the heart, when disorders in impulse formation and or transmission causing symptomatic bradyarrhythmia

### ♥ Types:

♥ Temporary.

♥ Permanent.

### ♥ Indications:

♥ Complete heart block.    ♥ Sick sinus S.    ♥ Second degree heart block.

(Programmed pace makers are available)

## ➤ Implantable automatic cardioverter-defibrillator:

Which can recognize ventricular tachycardia or fibrillation and automatically delivers pacing or a shock to the heart to cause cardioversion to sinus rhythm.

## Q. Non drug therapy of arrhythmias!?

- ① External defibrillator (DC) examples.....
- ② Implantable cardioverter – defibrillators e.g. in ventricular tachycardia.
- ③ Radiofrequency catheter ablation to interrupt a re–entry circuit by selective damage of endocardial tissue.
- ④ Artificial cardiac pace makers
  - ✓ Temporary.
  - ✓ Permanent.

# Cardiac Arrest

**Definition:** It is sudden & complete loss of cardiac function.

(No pulse with loss of consciousness, respiration ceases immediately)

It may be due to ventricular fibrillation, ventricular asystole (standstill) or electromechanical dissociation.

## (1) Ventricular Fibrillation

✍ This is the commonest cause of cardiac arrest & the most easily treatable

### ↪ Causes:

- ✓ Myocardial ischaemia.
- ✓ Hypokalemia.
- ✓ Structural heart disease e.g congenital, rheumatic, ischemic heart disease.
- ✓ Electrocutation.

### ↪ Diagnosis

- ✓ The arrhythmia produces rapid ineffective movement of the ventricles.



**ECG (QRS complexes are):**

- ✓ Broad.
- ✓ Bizarre.
- ✓ Irregular.

**↪ Treatment:**

- ✓ Defibrillator 200 - 360 joules (It can be repeated 3 times)
- ✓ If no response, adrenaline 1mg IV or intracardiac during cardiopulmonary resuscitation (CPR), see later. Then defibrillator 360 Joules can be repeated.
- ✓ If refractory, internal defibrillation can be used.

Under other circumstances the circulation must be maintained by the resuscitation procedures described below (CPR).

***(2) Ventricular Asystole***

This occurs when there is no electrical activity of the ventricles which may be due to failure of the conducting system or massive ventricular damage.

**↪ Cause:**

- ✓ Adam's stock attacks.
- ✓ Extensive myocardial infarction.

**↪ Treatment:**

- ✓ DC (200 – 360 joules), It can be repeated.
- ✓ If no response give adrenaline 1mg IV or intracardiac, also atropine 2 mg IV can be tried.
- ✓ Then pace maker must be considered when the electrical activity becomes evident.

- If the cause is massive infarction the treatment will be difficult.
- Any IV medications used in cases of cardiac arrest must be given with CPR.

***(3) Electromechanical dissociation***

i.e no effective COP despite the presence of normal electrical activity, also it is called pulseless electrical activity.

**↪ Causes:**

- 1- Tension pneumothorax.
- 2- Cardiac tamponade.
- 3- Massive pulmonary embolism.  
(Rarely, it may break up during resuscitation)
- 4- Cardiac rupture.

**↪ Diagnosis:**

ECG (QRS without palpable pulse).

**↪ Treatment:**

- ♦ Treatment of the cause.
- ♦ CPR.
- ♦ Adrenaline 1 mg IV can be tried.



# "Cardio Pulmonary Resuscitation" (CPR)

- ♦ Lay the patient on hard object.
- ♦ Clear airway from any secretions, extend the neck & raise the chin, then breathing mouth to mouth or to mouth through tube + cardiac massage 1:4.

- The most common cause of cardiac arrest is coronary heart disease.
- Potentially reversible causes of cardiac arrest:
  - ☆ Hypoxia.
  - ☆ ↑K, ↓K.
  - ☆ Hypothermia.
  - ☆ Tension pneumothorax.
  - ☆ Tamponade.
  - ☆ Drug induced.
  - ☆ Thromboembolic.

## *Anticoagulants*

### ↩ **Indications:**

- ☆ Recent MI – Unstable angina, AF.
- ☆ Cerebro-vascular insufficiency.
- ☆ Patients predisposed to thrombosis, (Thrombophilia) see haematology.
- ☆ DVT, pulmonary embolism.
- ☆ Patients with artificial cardiac valves.

### ↩ **Contraindications:**

- 1- Liver cirrhosis, hemorrhagic diseases.
- 2- GIT ulcers.
- 3- Infective endocarditis for fear of cerebral hemorrhage.
- 4- MI + pericarditis for fear of haemopericardium.
- 5- Severe uncontrolled hypertension.

### ↩ **Types:**

- ☆ Heparin.
- ☆ Oral anticoagulants.

### ☞ **Heparin**

★ **Action:** Antithrombin (it potentiates the action of antithrombin)

★ **Dose:**        ✧ 1000 units / hr IV infusion or 20 units/kg/hr (we can start with loading dose 5000-10000 units IV).

Or        ✧ 5000 - 7500 units / 6 hrs. I.V.

Or        ✧ 10.000 units / 8 hrs. s.c.

★ **Controlled by:** PTT, It must be 1.5 – 2.5 times the control time.

★ **Antidote:** ✧ Protamine sulphate.  
                   ✧ Fresh blood.



Low molecular weight heparin used with low incidence of hemorrhage (Clexane), it can be given without monitoring.

**Dose:** 60-80 mg/12h SC (Therapeutic dose).

## ✋ **Oral anticoagulants**

- ★ **Action:**
- ♦ ↓ Synthesis of prothrombin.
  - ♦ ↓ Synthesis of factor VII, IX, X.

- ★ **Dose:**
- ♦ Phenandione (Dindevan) 50 mg tab, it is an old drug.
  - ♦ Warfarin 5 mg tab (Marevan). We can start with 10 mg as a loading dose, then adjust the subsequent daily dose according to INR.

★ **Controlled by:** PT, It must be about 1.5-2 of the control.  
INR is more accurate, it must be 2.5-3.5 according to the condition (see haematology).

★ **Antidote:** Vitamin K.                              ♦ Fresh blood.

### **Side effects of anticoagulants:**

- 1- Hemorrhage.
- 2- Sudden withdrawal —————→ Thrombosis.
- 3- Phenandione —————→ Jaundice.
- 4- Heparin —————→ Allergy & platelets↓↓.

In treatment of thrombotic conditions e.g DVT, pulmonary embolism, heparin is started concurrently with warfarin for 3-5 days until warfarin reaches a therapeutic level, warfarin is then continued for 3-6 m in patients no longer at risk for recurrent thrombosis, but it should be continued indefinitely in patients in whom the predisposing condition persists.

- Antithrombin deficiency → Lack of heparin response.
- Factor VII declines after 6 hrs, factor IX declines after 24 hrs, factor X and prothrombin decline after 48 hrs from the start of anticoagulant therapy.

- The prophylactic dose of heparin is 5000 unit SC /12 hr (Minidose heparin) or 20 – 40 mg SC /12 hr for low molecular weight heparin (Clexane).



# Rheumatic Fever

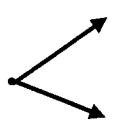
## Definition:

- ♥ Rheumatic fever is an inflammatory disease following infection with group A streptococci.
- ♥ It affects the heart, skin, joints and central nervous system. So, it is a multi-system disease.
- ♥ It is mostly due to autoimmune reaction triggered by the infecting streptococcus.
- ♥ The condition is not due to direct infection of the heart or to a production of a toxin.

## Etiology:

### 1- Allergic theory:

- ♥ Due to cross sensitivity between antigen of streptococci & cardiac tissue. (cardiac myosin and sarcolemmal membrane protein)
- ♥ This leads to production of antibodies against cardiac tissue.

So..  Rheumatic fever occurs 2-3 weeks after infection with streptococci. (until production of antibodies)  
ASO titre is high.

A cell wall constituent (N acetyl glycosamine) of the group A streptococci shares the antigenic properties with heart valve glycoprotein.

### 2- Auto immune theory: ( Old theory and not accepted)

Fixation of toxins of the streptococcus



Render cardiac tissue antigenic



Production of auto-antibodies

## Predisposing factors:

- 1- Age: 5 – 15 years, (it is rare below age of 4 years or more than 25 years).
- 2- Low socioeconomic status.
- 3- Over crowding.
- 4- Its prevalence in Europe and North America has progressively declined to very low levels, but it remains common in parts of Asia, Africa, South America and Eastern Europe.
- 5- In developing countries rheumatic fever is still the most common cause of acquired heart disease.



**Pathology:** (Rheumatic fever bites the heart but licks the joints)

Two types of reactions are identified

**Exudative reaction**

- Affecting:
- ⇒ Pleura.
  - ⇒ Peritoneum.
  - ⇒ Pericardium.
  - ⇒ Synovial membrane of joints.

The exudative reaction heals mostly by resolution.

**Proliferative reaction**

- Aschoff nodules
- Inflammatory cells



Central fibrinoid necrosis.

♥ These Aschoff nodules, heal by fibrosis.

♥ Aschoff nodules affect:

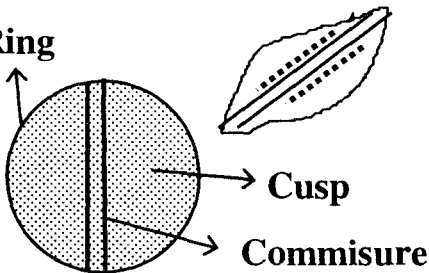
- Endocardium.
- Myocardium.

**Macroscopic:**

The valve cusps are

- Swollen.
- Edematous.
- Vegetations along the line of closure of the valve. They are very adherent so, no embolization. They may lead to some degree of valvular regurgitation...!?. Late fibrosis of the valves occurs with development of stenosis or regurgitation of the valves (organic valve lesions).

Ring



Cusp

Commissure

Fibrosis with shrunken cusps → regurge  
 Fibrosis of the commissures → stenosis.

**Clinical picture:**

*Revised Jones criteria*

**Major**

CACSE

- ◆ Carditis.
- ◆ Arthritis.
- ◆ Subcutaneous nodules.
- ◆ Chorea.
- ◆ Erythema marginatum.

**Minor**

- ◆ Arthralgia.
- ◆ Previous rheumatic fever.
- ◆ First or second degree heart block in ECG.
- ◆ ↑ ESR or CRP.
- ◆ ↑ TLC.
- ◆ Fever.





- ♣ It is important for evidence of preceding streptococcal infection e.g ↑ ASO or positive throat swap culture.
- ♣ Evidence of streptococcal infection is particularly important if there is only one major criteria.
- ♣ The diagnosis is made on the basis of two or more major criteria or one major plus two or more minor criteria.

## Arthritis

- Big joints with asymmetrical involvement.
- Each joint affected for about one week.
- Course of arthritis is about 6 weeks.
- The joints are swollen, red and tender.
- Flitting, (as the inflammation in one joint recedes, another becomes affected)
- Good response to salicylates.

## Carditis

- Affect mainly children.
- Pancarditis

### A. Myocarditis

- Tachycardia
- Arrhythmia e.g-extrasystole
- Muffling of heart sounds. (Tic-Tac rhythm.)
- Heart failure with dilatation of the heart. may results from severe myocarditis.
- Prolonged P-R interval.
- MI due to dilated heart

### B. Pericarditis

- Pericardial rub
- It's presence indicates severity
- It is usually dry but effusion is rare.

### C. Endocarditis

- Affecting (mitral – aortic) but tricuspid and pulmonary valves are rarely affected.
- Carey - Coomb's murmur. i.e transient diastolic murmur on mitral area which present during rheumatic activity, due to swollen mitral cusps. (Mitral valvulitis)
- Fulminant rheumatic fever may lead to MI or AI.

Later, chronic rheumatic valvulitis well developed either in the form of stenosis or regurge.

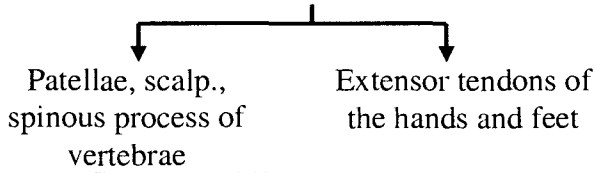


**Rheumatic chorea** (St vitus dance) may manifest late after the initial infection, see neurology. It is self limiting after about 6 months.

**Subcutaneous nodules**

(associated with severe carditis)

- Usually occurs over



- Small (pea-sized)
- Firm
- Not tender
- Not adherent to skin.

**Erythema marginatum**

(20% of cases)

- Rounded macules
- Reddish or pink
- Central pallor
- Affects trunk and proximal extremities.
- Not painful or itchy.

It appears for hours and disappears, then recurs, it usually does not affect the face.

**Erythema nodosum**

Dusky red raised papules or nodules, on front of the shins; they are not common and non specific.

**Investigations:**

(There is no specific laboratory test to indicate the presence of rheumatic fever)

1. **Blood picture:** → TLC ↑↑ (Neutrophilia)
2. **E.S.R.:** ↑↑
3. **C.R.P.:** ↑↑ (It is a globulin reacts with the capsules of pneumococci in vitro)

**(A) Evidence of systemic disease.**

4. **ASO titre is high:**
  - Normally it is 0-150 Todds.
  - If it is > 250 in adult, or > 333 in children = positive test
  - Very high titre or raising titre is diagnostic.
  - It indicates recent streptococcal infection.

5. **Throat swap for the streptococci.**

6. **Other Antibodies**

20 % of patients & those with rheumatic chorea have borderline ASO titre so, ask for:

- Anti-DNase or Anti-hyaluronidase.
- Anti streptozyme test (ASTZ), it is a haemagglutination reaction to concentrate of extracellular streptococcal antigens adsorbed to red blood cells.

**(B) Evidence of preceding streptococcal infection.**



- 7. **X ray** → Cardiomegally, PVC (myocarditis).
- 8. **Echo** → Valve lesions – ventricular dysfunction (ejection fraction).
- 9. **ECG** → First or second degree heart block.

↑

**(C) Evidence of carditis.**

Acute phase reactants

- |               |                    |
|---------------|--------------------|
| 1. CRP.       | 4. Platelets.      |
| 2. C3 .       | 5. Serum ferritin. |
| 3. Globulins. |                    |

These phase reactants usually increase within the few hours in most infectious diseases, tissue damage, or inflammatory conditions.

**Q. Signs of rheumatic – activity:** Tachycardia, fever.

- ... ↑E.S.R.
- ... TLC↑↑ - CRP↑.
- ... Muffling of heart

sounds.

**D.D.**

- ◆ Infective endocarditis.
- ◆ Juvenile rheumatoid (juvenile chronic arthritis).
- ◆ Fever of unknown etiology.
- ◆ Blood disease due to the presence of (pallor - fever - bony aches)

**Treatment:**

**\* General measures:**

- Rest till → S&S↓ - ESR↓ - CRP↓
- Diet. → ↑Carbohydrate, ↓ salt, also avoid heavy meals.

In patients who have had carditis, it is conventional to continue bed rest for 2-6 wks after ESR and temperature have returned to normal

**\* Prophylaxis of rheumatic fever**

(A) Primary prevention:

- Proper hygiene, avoidance of over crowding, good nutrition to prevent streptococcal throat infection. Early detection and treatment of streptococcal throat infection.

(B) Secondary Prevention:

- Long acting penicillin. (1.2 million units/2weeks) or oral penicillin V (250 mg / 12hr) or erythromycin (250-500 mg/12 hrs) till age 25years or for 5 years after the last attack in adults.
- Recurrences are more common when cardiac lesion is present and if so, prophylaxis may be required until age 30 years or for life !?

(C) Tertiary prevention::

- Prevention of infective endocarditis (see later).



## \*Active treatment:

### 1. Penicillins: (to eliminate any residual streptococcal infection)

- › Phenoxy-methylpenicillin (oral) 250-500 mg/6 hrs for 1 week.
- or
- › Benzathine. P → 916 mg (1.2 million units), IM.
- or
- › Ampicillin or amoxicillin → 500 mg/ 6 hours for 1 week.

### 2. Salicylates:

- 5-6 gm/d oral (60-120 mg/kg/d) divided into six doses.
- It will relieve symptoms of arthritis rapidly within 24 hours.
- It should be continued until ESR has fallen and then gradually tailed off to avoid rebound arthritis.

- ☞ Value
- Anti inflammatory.
  - Analgesic.
  - Antipyretic.

☞ Side effects

- Bleeding tendency
- Respiratory alkalosis & then metabolic acidosis (toxicity).
- Salicylism. (vertigo, tinnitus)
- GIT irritation.
- Bronchospasm.
- Nephrotoxicity.

### 3. Steroids:

☞ Indications:

- \* Severe carditis
- \* If the salicylates → is not effective (severe arthritis).  
→ or is not tolerated.

☞ Dose:

- ◆ Prednisolone 1-2 mg/kg /d for about 4 weeks.
- ◆ It should be continued until ESR has fallen and then tailed off, salicylates can be added during steroid withdrawal to avoid rebound arthritis.

#### **Treatment of heart failure in cases of rheumatic fever (myocarditis)**

- = Low salt diet - Diuretics.
- ▶ Steroids for myocarditis.
- ▶ Digitalis is better to be avoided or used with caution !?,  
as it may lead to:
  - Arrhythmia.
  - Heart block.
- ▶ Dopamine, dobutamine, vasodilators, can be used.



# INFECTIVE ENDOCARDITIS

## Etiology

- \* Infective endocarditis is due to microbial infection of a heart valve (native or prosthetic), the lining of a cardiac chamber or blood vessel, or a congenital anomaly (e.g septal defect).
- \* The causative organism is usually a bacterium, but may be a rickettsia (*coxiella burnetii* or Q fever endocarditis), chlamydia or fungus.
- \* The term infective endocarditis is better than bacterial endocarditis because not all the infecting organisms are bacteria
- \* Virulent organisms may infect normal valves, especially when the patient is generally debilitated or immunologically incompetent

## Pathology

- ⊙ Bacteria with abnormal jet of blood or turbulence e.g (valve lesions or septal defect)



Implantation of organisms in the endocardium.

- ⊙ It is more common on the left side, in the IV drug addicts the valves in the right side are usually affected.

### I- Underlying cardiac lesion:

(Abnormal jet of blood or turbulence)

#### λ Congenital

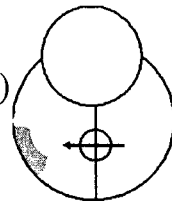
- VSD
- ASD → rare (low pressure gradient)
- PDA.

#### λ Rheumatic

MVD (uncommon in MS) -AVD.

#### λ Prosthetic valve

→ prosthetic valve endocarditis



### II- Bacteremia:

- |                          |                                   |
|--------------------------|-----------------------------------|
| • Endoscopy              | • Tooth extraction                |
| • Catheter               | • Chewing candy                   |
| • Minor or major surgery | • IV cannulae or cardiac catheter |
| • Cardiac surgery        | • Tooth brushing (daily activity) |

### Organisms:

- |  |                             |
|--|-----------------------------|
| • Streptococcal viridans.                        | • S. Faecalis, enterococci. |
| • Staphylococci - Pneumococci.                   | • Chlamydia.                |
| • Rickettsia ( <i>coxiella burnetii</i> )        |                             |
| • Fungal ( <i>Candida</i> , <i>Aspergillus</i> ) |                             |

(*Staph epidermidis* is common in IV drug addicts)

### Pathogenesis of infective endocarditis.

- Infections tend to occur at sites of endothelial damage because there is a deposit of platelets and fibrin in these areas → colonization. The avascular valve tissue and the presence of fibrin help to protect the proliferating organism from host defense mechanisms.



- When the infections is established, vegetations (composed of organisms, fibrin and platelets) grow and may become large enough to cause obstruction, they may break into septic emboli. **So:**
  - (A) Damage of endocardium of right ventricle, in VSD for example is due to the abnormal jet of blood. → Rough surface → Vegetation + organism → Septic friable thrombus → Thromboembolism.
  - (B) Lesion occurs in the left atrium due to mitral incompetence, lesion occurs in the pulmonary artery in cases of PDA.
  - (C) Lesion occurs in the left ventricle in case of AI.

- ◆ Subacute endocarditis gives full picture as it may be of long time enough to produce symptoms and signs.
- ◆ Acute endocarditis, the clinical signs of subacute endocarditis are usually absent.
- ◆ Post operative endocarditis may similar to subacute or acute endocarditis according to the virulence of infecting organism.
- ◆ Endocarditis is more common on the left side (MVD and AVD).
- ◆ In IV drug addicts the valves in the right heart are usually affected e.g tricuspid valve → TI.

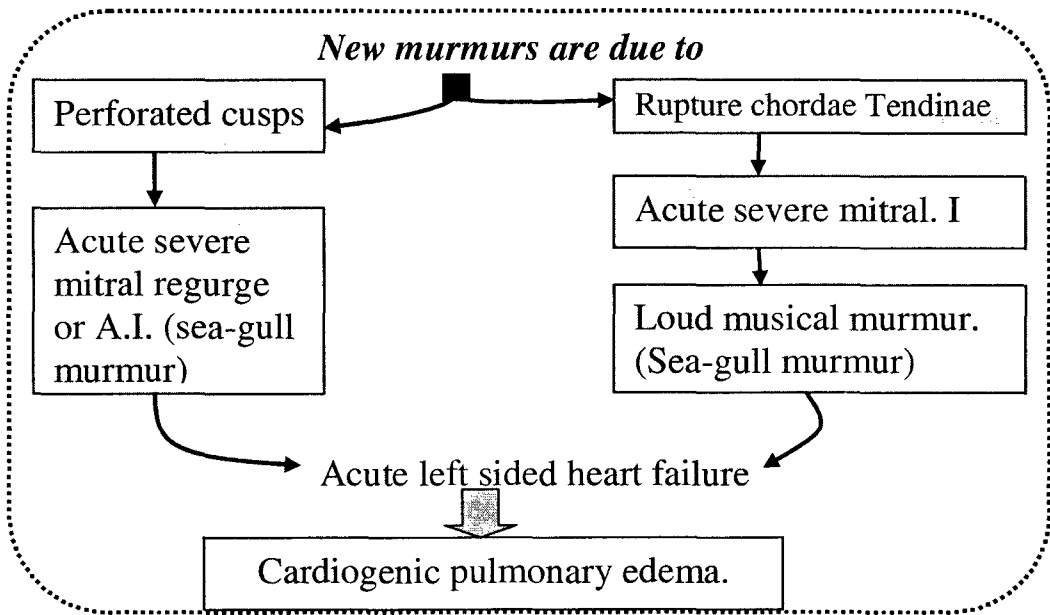


**1. Toxic manifestations**

Fever. Anorexia. Headache. Malaise.

**2. Original cardiac lesions**

± new additional lesion.





Acute mitral incompetence leads to sudden backward failure with sudden rise of the left atrial pressure → sudden severe PVC leading to pulmonary edema.

### 3. Thromboembolism

- CNS → Stroke (cerebral infarction) → hemiplegia.
- UL – LL → Ischaemia.
- Mesenteric occlusion → acute abdomen.
- Mycotic aneurysm occurring at points in the artery wall that have been weakened by infection in the vasa vasorum or where septic emboli have lodged → fibrosis → aneurysm → rupture → subarachnoid haemorrhage.

### 4. Renal

- Infarction → Gross haematuria.
- Immune complex GN (membranoproliferative) →
- Flea beaten kidney due to capillaritis.

Microscopic haematuria and proteinuria.

### 5. Eye

1. Roth's spot by fundus examination (vasculitis)
2. Subconjunctival haemorrhage (vasculitis)
3. Sudden blindness (central retinal artery occlusion)

### 6. U.L

#### • Osler's nodules (vasculitis)

- \* Tender.
- \* Small nodules
- \* Intracutaneous.
- \* Over the bulbs of the fingers.

#### • Clubbing

- \* (Pale clubbing or toxic clubbing), it occurs with long standing cases, So it is common in subacute rather than acute cases.

#### • Splinter haemorrhage (vasculitis)

- \* Longitudinal.
- \* Under nails.

#### • Ganeway lesions or spots

- \* Reddish or hemorrhagic patches on the palms or the soles.
- \* They are painless.

### 7. Spleen++

- It occurs with longstanding cases.
- Soft.
- Mild enlargement.
- Tenderness.
- Friction rub usually present with splenic infarction.



**8. Arthritis or arthralgia** of big joint is frequently seen.

**9- Petechial hemorrhages** of the skin due to toxic capillaritis.

- The extracardiac manifestations result either from embolisation or due to deposition of immune complexes.
- Myocardial infarction can result from coronary emboli, also pulmonary infarction may occur if right sided lesions embolize.
- Hepatosplenomegaly occurs in infective endocarditis caused by *Coxiella burnetii*.

## Investigations

- 1. Blood culture:** (it is the crucial investigation).  
(Three samples for aerobes & anaerobes, special culture may be necessary.)
- 2. Echo** for vegetation:  
(Small sized vegetation i.e few mm can be seen by transoesophageal echo).
- 3. ↑ ESR - TLC↑ - ↑ CRP**, (normocytic normochromic anaemia).
- 4. Urine analysis for:** Proteinuria and haematuria.
- 5. Serum immunoglobulins:** are increased, but total complement and C3 are decreased due to immune complex formation. Also circulating immune complexes (CIC) are present in more than 70% of cases.
- 6. Positive rheumatoid factor.**
- 7. ECG:** Conduction defects with abscess formation and occasionally myocardial infarction due to emboli may occur.

## Treatment

### Curative

Once you suspect give empirical antibiotic combination for G+ve and G-ve organisms:

- ☛ For G +ve. → Penicillin G (25 million unit IV /d.) or vancomycin 1g/12hr.
- ☛ For G -ve. → Gentamycin 80mg/12hrs IM or 3rd G cephalosporins 2-4 IV gm/d.

When the culture is available give antibiotic according to culture for 6 weeks.

- ☛ **Coxiella burnetii** → Rifampicin + doxycycline
- ☛ **Strept. Viridans** → Penicillin + gentamycin
- ☛ **Staphylococci.** → Methicillin 4 gm/d  
or Vancomycin 1gm/12hr IV.
- ☛ **Pseudomonas** → Carbenicillin 10gm/d+ Gentamycin 80mg/12 hrs  
or Azlocillin + tobramycin
- ☛ **Strept Faecalis** Gentamycin + penicillin





**Persistence of fever with appropriate antibiotic therapy may be due to abscess formation or large vegetations (septic).**

⇒ **Surgery**

- Indications:**
1. Replacement of infected prosthetic valve.
  2. Abscess formation.
  3. Large vegetations (septic).

**Recent trends for antibiotic regimens for dental, respiratory procedures and GIT, urinary procedures**

Procedure	Antibiotic regimen
Dental or upper respiratory tract procedures under local anaesthetic If allergic to or received penicillin in last month	Amoxicillin 3 g orally 1 hr before  Clindamycin 600 mg orally 1 hr before
Dental or upper respiratory tract procedures under general anaesthetic If allergic to or received penicillin in last month	Amoxicillin 1 g i.v. at induction plus amoxicillin 0.5 g orally 6 hrs later  Vancomycin 1 g i.v. infusion over at least 100 mins. Plus gentamicin 120 mg i.v. at induction.
<b>Special-risk patients</b> , i.e. prosthetic valve or previous endocarditis Genitourinary procedures If allergic to or received penicillin in last month	Amoxicillin 1 g i.v. Plus gentamicin 120 mg i.v. at induction Plus amoxicillin 0.5 g orally 6 hrs later.  Vancomycin 1 g i.v. infusion over at least 100 mins. Plus gentamicin 120 mg i.v. at induction.
<b>Obstetric and gynaecological procedures or gastrointestinal surgery/instrumentation (as for special-risk patients above).</b>	

**The prophylactic long acting penicillin for rheumatic fever does not prevent infective endocarditis.**

**Q Culture – negative endocarditis:**

This accounts for 5-10% of endocarditis cases. the usual cause is antibiotic therapy, or due to fastidious organisms that fail to grow in usual blood cultures e.g. *Coxiella burnetii*, *Chlamydia* and *Legionella*. PCR may be needed to diagnose such cases.



## ***Q Duke criteria for the diagnosis of infective endocarditis (IE)***

### **(1) Major criteria.**

- Positive blood culture: \* Expected microorganisms for IE from two separate blood cultures.
  - \* Persistently positive blood culture (blood cultures that are obtained more than 12 hours apart).
- Evidence of endocardial involvement by echo:
  - Oscillating intracardiac mass.
  - Abscess.
  - New valvular regurgitation.

### **(2) Minor criteria.**

- Fever  $> 38^{\circ}\text{C}$ .
- Immunological phenomena e.g Roth's spots, osler nodules, GN or +ve rheumatoid factor.
- Predisposition to IE e.g heart condition or IV drug abuse.
- Vascular phenomena e.g arterial embolic, mycotic aneurysm janeway spots, conjunctival haemorrhages.
- Echo finding consistent with IE, but major criteria as stated above are not met.
- Microbiological evidence of IE (blood cultures are positive but major criteria are not met as previously described, or serological studies support an infection consistent with the diagnosis of IE.

**Diagnosis of IE can be made if two major criteria, one major and three minor, or five minor criteria are present.**



# PERICARDIAL DISEASES

## The Pericardial Sac:

- The pericardium has two protective covering layers, parietal and visceral layers.
- It lubricates the surface of the heart and acts as a barrier to the spread of infection; it also limits distention of the heart. The pericardial sac contains up to 50 ml of fluid.
- The visceral layer is not sensitive to pain, but the parietal layer is sensitive.
- The parietal layer may be insensitive, and the pain that occurs with pericarditis is due to irritation of the pleura.
- Absence of the pericardium does not appear to result in significant clinical or functional limitation.

## I. Acute Pericarditis

### Causes

1. Viral: coxsackie - echo - influenza- measles -mumps
2. Purulent e.g.
  - ♦ Staphylococci → blood born (septicaemia)
  - ♦ Streptococci → penetrating trauma.
3. TB.
4. Rheumatic fever
5. Uremia.
6. Malignancy
  - ↙ Carcinoma of the breast
  - ↘ Carcinoma of the lung
7. Radiation.
8. Collagen disease.
9. Myocardial infarction
10. Drugs e.g hydralazine.

### C/P

1. Pain →
2. Toxemia
3. Symptoms of the cause
4. Pericardial rub.

- ♥ Stitching.
- ♥ Retrosternal.
- ♥ Radiates to shoulders and neck.
- ♥ Increased by breathing, movement and swallowing.

### Investigations

- ⇒ ECG
  - ♦ Raised S-T segment (diffuse with upward concavity)
  - ♦ Inverted T if there is a degree of myocarditis.
- ⇒ Echo: for development of effusion.
- ⇒ Lab: Cardiac enzymes may be elevated if there is associated myocarditis.

### Treatment

1. Cause
2. NSAIDs. e.g (Indomethacin 25mg / 8hrs)
3. Systemic steroids may be needed (controversy), infective causes!?
4. Anticoagulants must be avoided because of the risk of haemopericardium.

✎ A pericardial rub is a high pitched superficial scratching or crunching noise , it is heard during systole and may also be audible in diastole and has to-and - fro quality.

✎ Viral pericarditis may be relapsing (relapsing pericarditis)



## II. Pericardial Effusion

### Fluid in pericardium

- ♥ **Bloody (hemopericardium):** ♦ Trauma. ♦ Rupture aneurysm of aorta.
- ♥ **Hemorrhagic:** ♦ Malignant. ♦ TB. ♦ CRF.
- ♥ **Exudate:** ♦ Viral. ♦ TB. ♦ Malignancy.
- ♥ **Transudate:** ♦ Part of generalized edema e.g nephrotic \$.
- ♥ **Chylous:** ♦ Fluid is milky white, rich in fat.  
With Sudan III it becomes orange.  
With Ether it becomes clear.

### ↪ Haemodynamics

It depends on the amount & rate of accumulation of effusion.

- ⇒ Small amount or large amount + rapid onset → Tamponade..
- ⇒ Large amount + gradual onset may gives mild S & S.

Significant effusion (**Tamponade**) i.e effusion interfering with the mechanics of the heart with impaired filling of the heart and decreased cardiac output → picture of heart failure (↓ COP, PVC, SVC).

### ↪ C/P

### Symptoms

- 1. Retrosternal oppression**
- 2. Manifestations of the cause**
- 3. PVC:** as effusion compresses pulmonary veins & left atrium (posterior structure)
- 4. SVC:** Pain in the right hypochondrium and swelling of both LL.
- 5. ↓ COP:** Dizziness, fainting, blurring of vision.
- 6. Position :** Prayer position → shift of fluids away from pulmonary veins & left atrium → ↓ PVC.

### Signs

- General:
- ① Tachycardia.
  - ② ↓ Cop with hypotension.
  - ③ **Pulsus paradoxus.**
  - ④ **Neck veins:**
    - Inspiratory filling (**Kussmaul's sign**).
    - Diastolic collapse (**Friedreich's sign**) i.e deep y descent.
  - ⑤ **Ascitis precox.**



## Heart examination

- Inspection and palpation: ♥ Apex is not visible or palpable.
- Percussion: ♥ Dullness outside the apex.  
♥ Wide bare area.  
♥ Shifting dullness at the 2nd left space !?  
♥ **Ewart's sign:** (Dullness in the left infrascapular area due to compression of the left lung → (left basal lung collapse)
- Auscultation: ♥ Weak or distant heart sounds.

## Investigations

- **X- ray:** (Flask shaped heart with sharp border).
- **Echo:** It is the best; it diagnoses also severity of effusion.
- **ECG:** Low voltage i.e amplitude of QRS complex in all limb leads < 5 mm.
- **Pericardiocentesis:** It may leads to arrhythmia or injury of coronary vessels.
  - ⇒ Diagnostic      ♦ Culture & Z.N.      ♦ Cytology      ♦ Chemistry
  - ⇒ Therapeutic      ♦ To prevent fibrosis      ♦ To ↓↓symptoms

Site of aspiration: (echocardiography guided).

At the angle between xiphoid process & left costal margin, and direct it towards the direction of the left shoulder.

- \* Cardiac tamponade describes picture of heart failure due to compression of the heart by a large or rapidly developing effusion leading to PVC, SVC and COP.
- \* SVC obstruction is common in constrictive pericarditis than pericardial effusion as constrictive pericarditis is a chronic disease but effusion with SVC obstruction = tamponade (emergency).

## Treatment

- ☛ Cause.
- ☛ Pericardiocentesis.
- ☛ Pericardial window.
- ☛ Steroids to ⊖ adhesions !?.
- ☛ Pericardiectomy.

## III. Constrictive Pericarditis (Pick's Disease)

☛ **Etiology** TB - Viral - Purulent – Haemopericardium – Irradiation.

## ☛ Pathology

The two layers of pericardium adherent to each other.



Interference with mechanics of the heart i.e poor filling of the heart due to the catching effect of the fibrous pericardium.



Also, the calcification may extend to the myocardium → impaired myocardial contraction.

Constrictive pericarditis should be suspected in any patient with a picture of unexplained right heart failure with a small heart.



**C\P**

As effusion but there are:

- ❖ Ascites precox.
- ❖ Size of the heart is smaller.
- ❖ AF due to decreased ventricular filling → ↑ atrial pressure → atrial dilatation → AF.
- ❖ Pericardial knock (3rd heart sound).
- ❖ P. Paradoxus.
- ❖ Kussmaul's sign and Friedreich's sign of neck veins.



**Investigations**

1. X-ray: → small sized heart – calcification of the pericardium.
2. ECG showing low voltage .
4. Echo shows thickened calcified pericardium.
3. CT scan or MRI → can detect pericardial calcification.



**D.D.**

Restrictive cardiomyopathy, to be differentiated by catheter and echo-doppler.



**Treatment**

- Antituberculous drugs.
- Pericardiectomy (Improvement may be delayed after pericardiectomy)

## IV. Adhesive Pericarditis



**Etiology**

It is a rare complication of rheumatic fever!?



**Pathology**

Adhesions of both layers of pericardium & between parietal pericardium & surrounding structures in the mediastinum.



**C\P**

1. Associated valve lesions.
2. Fixed apex, and systolic intercostal retraction (**Broadbent's sign**).
3. Manifestations similar to constrictive pericarditis e.g PVC,  
↓ COP, SVC.



**Investigations**

- ♦ Fluoroscopy screen (Ba swallow + screening) showing kinking of oesophagus with cardiac beats .



**Treatment**

Treatment of any associated valvular lesions + surgery for the pericardium.



# CARDIOMYOPATHY

## Definition:

The cardiomyopathies are diseases that primarily affect the myocardium and are not the result of hypertension, congenital or rheumatic heart disease, coronary or pericardial abnormalities.

## Clinical types:

### 1. Dilated Cardiomyopathy

#### Aetiology:

- ❖ Alcohol abuse.
- ❖ Hemochromatosis
- ❖ SLE.
- ❖ Polymyositis.
- ❖ Friedreich's ataxia.
- ❖ Selenium deficiency
- ❖ Adriamycin.
- ❖ Idiopathic

#### C/P:

- ① All chambers are dilated with systolic dysfunction.
  - ♥ Biventricular dilatation.
  - ♥ Picture of congestive heart failure.
    - Left sided failure "see before"
    - Right sided failure "see before"
- ② Embolic manifestation, AF may occur.

#### Investigations

- \* X-ray, echo → dilated heart.
- \* ECG showing non specific changes.

#### Treatment

- ACE inhibitors.
- Diuretics.
- Anticoagulant therapy for AF or history of embolization.

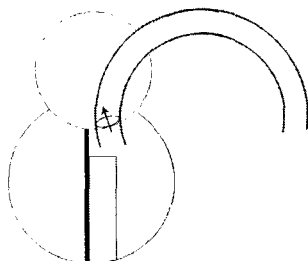
Reversible dilated cardiomyopathy may occur with pregnancy, alcohol abuse, selenium deficiency, hypothyroidism and hyperthyroidism.

- Corticosteroids for inflammatory cases.
- Digitalis for cases with AF.
- Treatment of arrhythmias.

**D.D:** Ischemic heart disease.

### 2. Hypertrophic obstructive cardiomyopathy (HOCM)

There is ↑↑ thickness of the interventricular septum → encroachment on the aortic opening → picture of AS (subvalvular) i.e left ventricular outflow tract obstruction.



- ❖ It is an autosomal dominant disease.
- ❖ There is mainly diastolic dysfunction.
- ❖ Mitral incompetence may occur in this type of cardiomyopathy as the thickened septum leads to abnormal movement of the mitral valve during systole.



**C/P:**

- Picture of aortic stenosis, with chest pain and dyspnea (diastolic dysfunction) on effort.
- Syncope commonly with exertion, ventricular arrhythmia.
- Sudden death typically occurs during or just after vigorous physical activity, HOCM is the most common cause of sudden death in young athletes. Ventricular arrhythmias are thought to be responsible for many of these deaths.
- Jerky carotid pulse because of rapid early ejection, followed by an obstruction and then by another emptying during systole (it indicates severity).
- There is also left ventricular hypertrophy with palpable 4th heart sound, which gives double impulse at the apex.

**Murmurs of HOCM:**

- Ejection systolic murmur at the base: It can be increased by valsalva or standing and decreased by squatting.
- Pansystolic murmur due to mitral incompetence secondary to systolic anterior motion (SAM) of the mitral valve.

**Investigations**

Echocardiography.   
 ↳ Left V hypertrophy involving the septum.   
 ↳ Systolic anterior motion (SAM) of the mitral valve.   
 ECG: Left V++, Deep wide Q waves (pseudo-infarct pattern).

**Treatment**

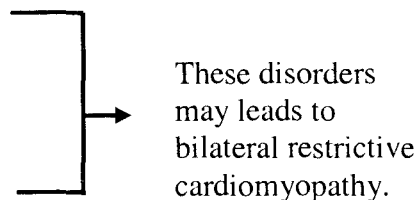
- Vigorous exertion should be avoided (see before).
- ♥ **Beta blocker and verapamil** (↓↓ outflow obstruction), they can help to relieve the angina and sometimes prevent the syncopal attacks.
  - ♥ **Digitalis** is better avoided as it ↑↑ the outflow tract obstruction.
  - ♥ **Amiodarone for arrhythmias.**
  - ♥ **Vasodilators** → ↑ outflow tract obstruction (so they are not used)
  - ♥ **Surgery** → myomectomy to ↓↓ thickness of the septum.
- Also, non surgical ablation of the septum can be done.

**3. Restrictive cardiomyopathy**

The ventricular filling is impaired because the ventricles are stiff.

**Causes**

- Amyloidosis
- Haemochromatosis.
- Sarcoidosis



**C/P**

- \* Diastolic dysfunction with restriction of ventricular filling giving picture similar to constrictive pericarditis.   
 ↳ Elevated venous pressures (SVC or PVC) i.e congestive heart failure.
- \* Fourth heart sound





- \* AF as there is decline in ventricular filling (stiff ventricle)  
→ ↑ atrial pressure → atrial dilatation → AF.
- \* Congested neck veins with kussmaul's sign and Friedreich's sign.

### Investigations:

- \* Echo → diastolic dysfunction.
- \* Endomyocardial biopsy.

### Treatment (No specific treatment)

- ♥ Symptomatic treatment e.g to relieve SVC and PVC by salt restriction, diuretics and to treat embolic manifestations.
- ♥ Treatment of the cause.
- ♥ Cardiac transplantation.  
(The disease may recur in cases of cardiac amyloidosis)

## MYOCARDITIS

### Etiology:

- Coxsackie virus.
  - Influenza virus
  - SLE, Rheumatoid disease.
  - Rheumatic fever.
  - Sarcoidosis.
- } → Myocarditis may occur several weeks after the initial viral infection.

### C/P:

- Picture of biventricular failure.(see before)
- Inappropriate tachycardia, muffling of H.S.
- Manifestations of the cause.

### Investigations:

- Echocardiography for ejection fraction – ECG → ST and T wave abnormalities.
- Manifestations of the cause e.g viral serology.
- Increased Troponine and cardiac enzymes.

### Treatment:

- Treatment of the cause.
- Diuretics.
- ACE inhibitors.
- Steroids (controversy).
- ECG monitoring for arrhythmias.

In cases of viral myocarditis there is controversy as regard the use of steroids. It may be harmful or with no benefit.



# DEEP VENOUS THROMBOSIS (DVT) & PULMONARY EMBOLISM

## Causes of DVT:

Virchow's triad (See hypercoagulable state in haematology)

- |                                 |                                   |
|---------------------------------|-----------------------------------|
| ❶ Slow venous circulation e.g.: | ❖ Heart failure.                  |
|                                 | ❖ Prolonged recumbency.           |
| ❷ Intimal injury                | e.g.: ❖ Phlebitis.                |
| ❸ Hypercoagulable state         | e.g.: ❖ Polycythaemia rubra vera. |
| (thrombophilia see below)       | ❖ Protein C, S deficiency.        |
|                                 | ❖ Antiphospholipid \$.            |
|                                 | ❖ Antithrombin deficiency.        |

## Thrombophilia

⇒ It is a term describing inherited or acquired defects of haemostasis leading to venous or arterial thrombosis e.g protein C,S deficiency.

(See coagulation abnormalities in haematology)

⇒ Thrombophilia should be considered in patients with:

- ❶ Recurrent DVT.
- ❷ Venous thrombosis under age of 40 years.
- ❸ Family history of venous thrombosis.
- ❹ Arterial thrombosis in absence of arterial disease.

## Sites of DVT:

☛ Lower limb: Ilio femoral or popliteal veins.

### ↙ C/P

1. Edema of lower limb.
2. Tender calf muscle.
3. Tenderness along the course of the affected vein.
4. Positive Homan's sign i.e pain on the calf on dorsiflexion of the foot, it is not diagnostic and may occurs with all lesions of calf muscles.

### ↙ D.D.

of DVT in LL (Or DD of tender calf muscle):

- |                         |                        |
|-------------------------|------------------------|
| 1. DVT                  | 2. Cellulitis.         |
| 3. Rupture plantaris.   | 4. Rupture baker cyst. |
| 5. Peripheral neuritis. | 6. Osteomyelitis.      |

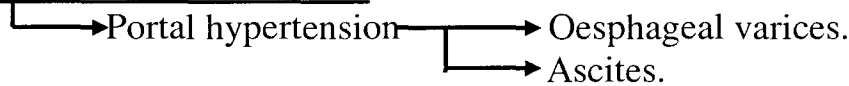
## ↙ Investigations:

1. **Lab investigations:** To diagnose thrombophilia (See haematology).
2. **Duplex scan**
3. **Venography.**
4. **Radio isotope scan:** Injection of radioactive fibrinogen, the site of DVT can be detected by Gamma camera (fibrinogen scanning).



## Other sites of DVT

### 1- Portal vein thrombosis



### 2- Renal vein thrombosis

- ◇ Common in membranous GN.
- ◇ Dehydration
- ◇ Blood disease C/P
  - ↳ Loin pain and tenderness.
  - ↳ worsening of kidney function?.
  - ↳ Proteinuria.

### 3- IVC thrombosis

- Cause    ❶ Typhoid.                      ❷ Behcet's disease.
- C/P            ☆ Edema of both lower limbs.
- ☆ Ascites.
- ☆ Collaterals on the abdominal wall.

### 4- SVC Thrombosis

- Cause            ☆ Constrictive pericarditis ♦ Mediastinal mass.
- C/P                ☆ Facial edema.
- ☆ Congested non pulsating neck veins.
- ☆ Chest collaterals directed from above downward.

### 5- Axillary Vein Thrombosis

- C/P                • Edema of the arm.
- Pain, tenderness along the course of axillary vein.

## PULMONARY EMBOLISM

### Sources of pulmonary embolism:

1. DVT.
2. Infective endocarditis of right side of the heart.
3. Fat embolism. e.g bone fracture.
4. Amniotic fluid embolism.
5. Air embolism.

### C/P of pulmonary emboli: (According to the size of embolism)

#### A- Small sized embolism

**C/P**

- No symptoms, but cough, dyspnea or chest discomfort may occur.
- Recurrent embolization with obliteration of >2/3 of pulmonary vascular bed.

↓

Thromboembolic pulmonary hypertension

↓

Right sided failure (cor pulmonale) i.e. subacute cor pulmonale.

**B- Moderate sized emboli (Pulmonary infarction)****C/P:**

1. Cough
2. Blood tinged sputum
3. Chest Pain (pleuritic)
4. Dyspnea
5. Fever.

**Local examination:**

- a- Pleural rub.
- b- Crepitations may present over the involved area.
- c- Blood stained effusion may developed (exudative effusion).

The lung parenchyma has three sources of O<sub>2</sub> i.e pulmonary vessels, bronchial vessels and air within alveoli so, the hemodynamics of the lung must be compromised for a lung infarction to occur on top of pulmonary embolism.

**Investigations****► Radiological Signs of Pulmonary Embolism**

1. Normal X – ray.
2. Triangle (wedged shaped opacity) i.e pulmonary infarction.
3. Pulmonary oligemia = massive embolism.
4. Pleural effusion.
5. Pulmonary edema.
6. Dilated pulmonary artery.

**► Blood tests:** ↑ TLC, ↑ ESR, ↑ LDH,

- ECG**
- ♥ Normal except for sinus tachycardia, AF may occur.
  - ♥ Right ventricular strain may occur (inverted T in V1, V2)
  - ♥ Right axis deviation.
  - ♥ Right BBB.

**► Pulmonary angiography**, it is diagnostic but invasive.**► Lung scan:****Ventilation Scan**

Patient inspires (Xenon) gas with radioactive material



We detect the distribution of the radioactive material by gamma camera within lung tissue, this reflects ventilation.

**Perfusion Scan**

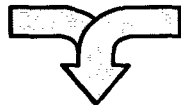
IV injection of radioactive material (Tc)



Lung uptake occurs by pulmonary arteries, this can be detected by gamma camera



This reflects pulmonary vascularity.



**Normal ventilation scan + abnormal perfusion scan highly suggestive for pulmonary embolism.**

In pulmonary fibrosis, there is abnormal ventilation scan with abnormal perfusion scan.

**► Spiral CT with IV contrast (CT pulmonary angiography).**



## C- Massive pulmonary Embolism

It is pulmonary embolism obstructing > 50 % of pulmonary vasculature.

### **C/P**

1. **Chest Pain** (similar to anginal pain) due to:

- ♥ Hypotension or shock ( $\downarrow$  cop)
  - ♥ Hypoxia.
  - ♥ Rapid distention of pulmonary artery.
- } → Anginal pain

2. **Shock** due to:

- ♥ Marked decreased of blood flow to lung  $\rightarrow \downarrow \downarrow$  VR to left atrium  
 $\rightarrow \downarrow$  COP  $\rightarrow$  shock.

3. **Cyanosis** (hypoxia), tachypnea and tachycardia.

4. **Acute right sided heart failure**

- ♥ Lower limb edema.
- ♥ Enlarged tender liver.
- ♥ Congested neck veins, with prominent a wave.
- ♥ S3 gallop on tricuspid area.

### **Investigations**

As above, **blood gases** showing hypoxia.

## Treatment of pulmonary embolism & D.V.T

### **I. Prophylactic measures:**

- ① Avoid prolonged post operative recumbency specially in pelvic surgery (e.g hysterectomy, hip surgery, prostatectomy).
- ② In risky patients subjected to surgery give:  
mini dose heparin or low molecular weight heparin (post operative).
- ③ Treatment of the cause

### **II. Resuscitation** (for massive embolism)

1. O<sub>2</sub> therapy.
2. Analgesics  $\rightarrow$  Pethidine
3. Treatment of shock e.g with dobutamine, this will improve the right ventricular efficiency.
4. Treatment of cardiac arrest if occurs.

### **III. Thrombolytic therapy** (for right ventricular failure and hemodynamic instability)

- Streptokinase.
- Urokinase.
- Recombinant tissue plasminogen activator.

- Value
- ☞ Relief the obstruction of the pulmonary vasculature.
  - ☞ Improvement of right ventricular efficiency.
  - ☞ Correction of the hemodynamic instability.



## IV. Anticoagulant therapy (for DVT and pulmonary embolism)

### Heparin

Give 5000 - 10.000 units IV as a loading dose.

Then 1000 units / hr IV infusion drip.

Heparin infusion is the best

Why?

↳ Low incidence of haemorrhage.  
↳ Maintained therapeutic level all over the day.

### Other methods of heparin therapy:

☛ 5000 - 7500 units IV / 6hrs

☛ 10,000 units SC / 8hrs.

Duration of heparin therapy → 7-10 days or till clinical improvement.

Follow up by PTT is adjusted to be (1.5 - 2.5) of the control value

Then start oral anticoagulant (Warfarin) for 3-6 months (as below).

### Warfarin

2.5- 7.5 mg/d.

The dose is adjusted according to PT to be (1.5 - 2) of the control value or to reach an INR value (2-3.5) according to the need.

The insertion of filter into IVC can be done if the anticoagulant or fibrinolytic therapy is contraindicated or fails to inhibit thromboembolism. So high risk patients with contraindications of anticoagulant therapy can be treated by insertion of a filter in the IVC above the level of the renal veins to prevent embolisation.

Pulmonary embolism should be suspected in patient with new onset of unexplained cough, dyspnea, or hyperventilation, chest pain, haemoptysis, AF, or with signs of P++ if no other cause can be found



# DISEASES OF AORTA

## 1. Aneurysm of Ascending Aorta

### Etiology:

- ☛ Syphilis (rare)
  - ☛ Cystic medial degeneration mainly in the elderly.
- This is the aneurysm of signs.
- ☛ AI (see later).
  - ☛ Dullness of aortic area.
  - ☛ Pressure on S.V.C.
  - ☛ Pulsating aortic area.

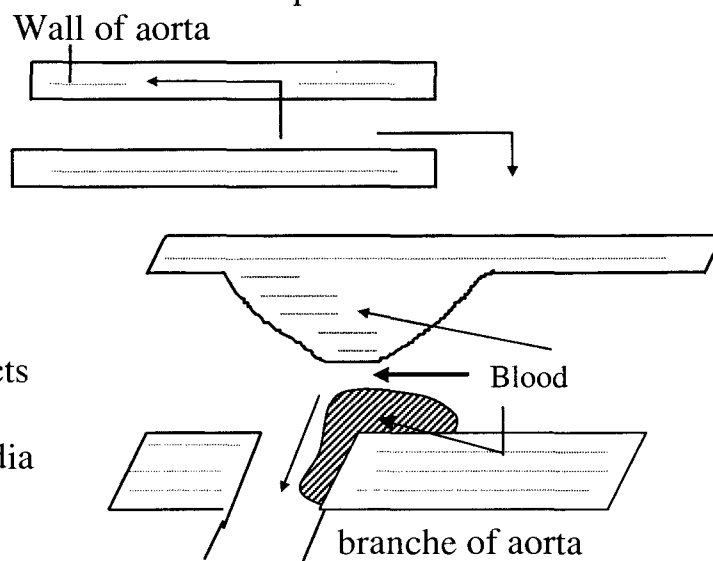
## 2. Aneurysm of the Arch (Aneurysm of symptoms)

See chest (mediastinal \$), it is usually due to atherosclerosis.

## 3. Dissecting Aortic Aneurysm

### Pathogenesis:

Tear in the intima of aorta → Blood bursts into the media of the aorta (dissection) which is then split into two layers, the aortic valve may be damaged and the branches of the aorta may be compromised.



Notice that the blood dissects its way through the media leading to narrowing of the lumen with obstruction of branches of aorta at the site of dissection

### C/P: (Old male, hypertensive with chest pain)

- ☛ Shock, 50% of patients may be hypertensive.
- ☛ Severe chest pain (tearing) radiating to the back.
- ☛ Obstruction of the opening of the branches from aorta → ischaemia.

Occlusion of aortic branches may cause a variety of complications including myocardial infarction (coronary), paraplegia (spinal), mesenteric occlusion (caeliac), renal failure (renal) and acute limb ischaemia.



- ⇨ Unequal pulse volume of both upper limbs or both lower limbs.
- ⇨ Acute AI may occur due to widening of aortic root.

### Types of dissecting aortic aneurysm:

- ♥ Type I → Proximal ascending aorta to descending.
- ♥ Type II → Confined to ascending aorta.
- ♥ Type III → Descending aorta and distally.

### ⇨ Etiology:

1. Hypertension.
2. Atherosclerosis.
3. Collagen disease e.g. Marfan & Ehler Danlos \$.

### ⇨ D.D.: Acute chest pain + shock.

- Extensive MI.
- Massive pulmonary embolism.
- Tension Pneumothorax.
- Dissecting aortic aneurysm.

### ⇨ Investigations:

- ⇨ CT scan is diagnostic.
- ⇨ MRI is highly accurate.
- ⇨ Chest X ray → broadening of mediastinum.

### ⇨ Treatment:

- ♥ Control blood pressure specially with B- blockers to lessen shear forces.
- ♥ Surgery with replacing the affected part with a Dacron graft, aortic valve replacement may be needed.

### Diseases of aorta:

- Aortic aneurysm and its types.
- Takayasu's disease (Aortic arch \$).
- Coarctation of aorta.
- Aortitis e.g in rheumatoid disease and sero negative arthropathy → AI.





# CONGENITAL HEART DISEASE

(0.8 % of live births)

## When you suspect congenital heart disease?

- 1 - Age < 5 years.
- 2 - Hypertension in a child.
- 3 - Positive prenatal history.
- 4 - Associated congenital anomalies.
- 5 - Cyanosis since birth.
- 6 - Negative history of rheumatic fever.
- 7 - Thrill over the base (AS-PS) or left parasternal (VSD)

## Causes (factors)

- 1- Genetic abnormalities e.g congenital heart block, ASD.
- 2- Irradiation.
- 3- Maternal rubella (PDA, Pulmonary stenosis)
- 4- Premature infants.
- 5- Chromosomal abnormalities e.g.:
  - ❖ Down's syndrome → VSD.
  - ❖ Turner's syndrome → coarctation of aorta
- 6- Maternal alcohol abuse (septal defects).

## Classification

### • Cyanotic

- ☞ Fallot's tetralogy
- ☞ Fallot's triology

### • A cyanotic

- ☞ Coarctation of aorta.
- ☞ PS- AS.
- ☞ Dextrocardia

### • Potentially cyanotic

- ☞ VSD.
- ☞ PDA.
- ☞ ASD.

Q. The commonest congenital heart diseases are bicuspid aortic valve and VSD.

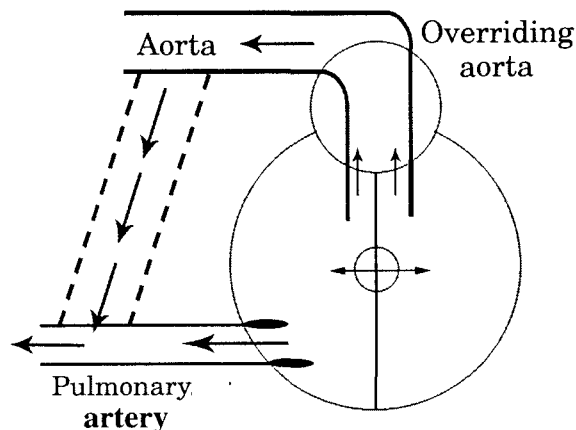
Q. The commonest congenital cyanotic heart disease is Fallot's tetralogy

## FALLOT'S TETRALOGY

It is the most common cyanotic anomaly in those who do survive and is commonest amongst adults.

## Components

- ◆ Pulmonary stenosis (PS)
- ◆ VSD.
- ◆ Right ventricular hypertrophy.
- ◆ Overriding aorta.

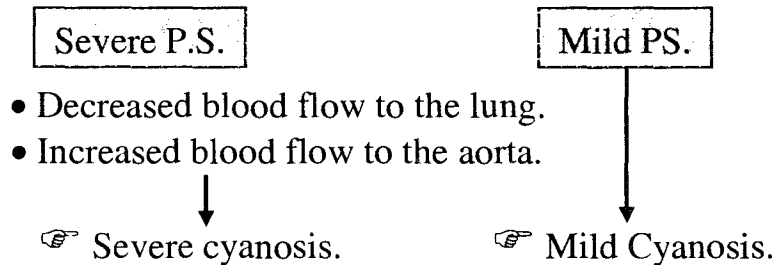




# 1. P.S

**It is the most important lesion that determines the severity of F4**

- ☉ It is infundibular = subvalvular due to abnormal tissue deposition, (right ventricular outflow obstruction).
- ☉ The subvalvular tissue is supplied by adrenergic receptors, so, the RV outflow obstruction is dynamic and may increase suddenly under adrenergic stimulation.
- ☉ PS is the most important lesion as it determines the amount of blood ejected to the lung.



## Cyanotic spells (Fallot's spells)

Exertion → ↑ sympathetic discharge with adrenergic stimulation of the subvalvular tissue  
 → spasm of the infundibulum → ↑ PS → ↑ blood flow to the aorta  
 → severe cyanosis & hypoxia → hypoxic syncope (cyanotic spells)

### Treatment

- ☉ O<sub>2</sub> therapy.
- ☉ Beta blocker IV or diamorphine to relax the right ventricular outflow obstruction.
- ☉ Squatting position.

# 2. V.S.D.

It is silent (no murmur) → why?:

- ♥ As it is a wide VSD.
- ♥ Both ventricles are subjected to the same aortic pressure !?

# 3. Overriding Aorta

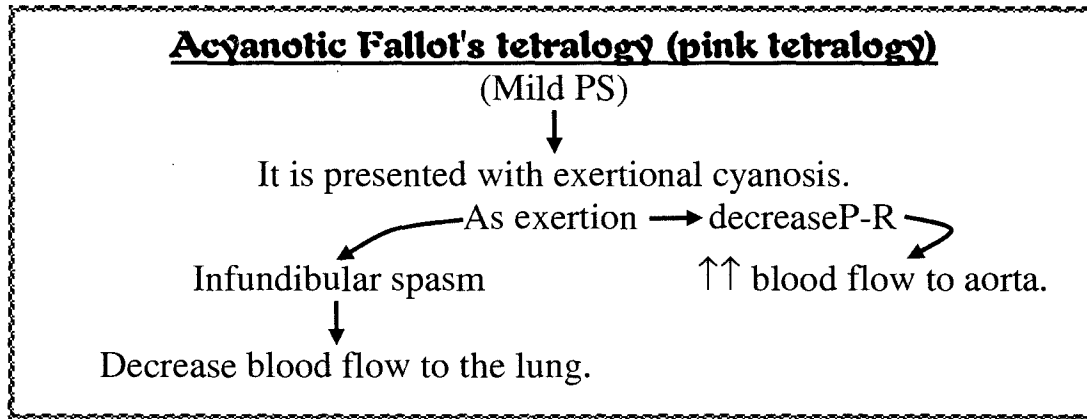
➡ This leading to central cyanosis.

# 4. Right ventricular hypertrophy

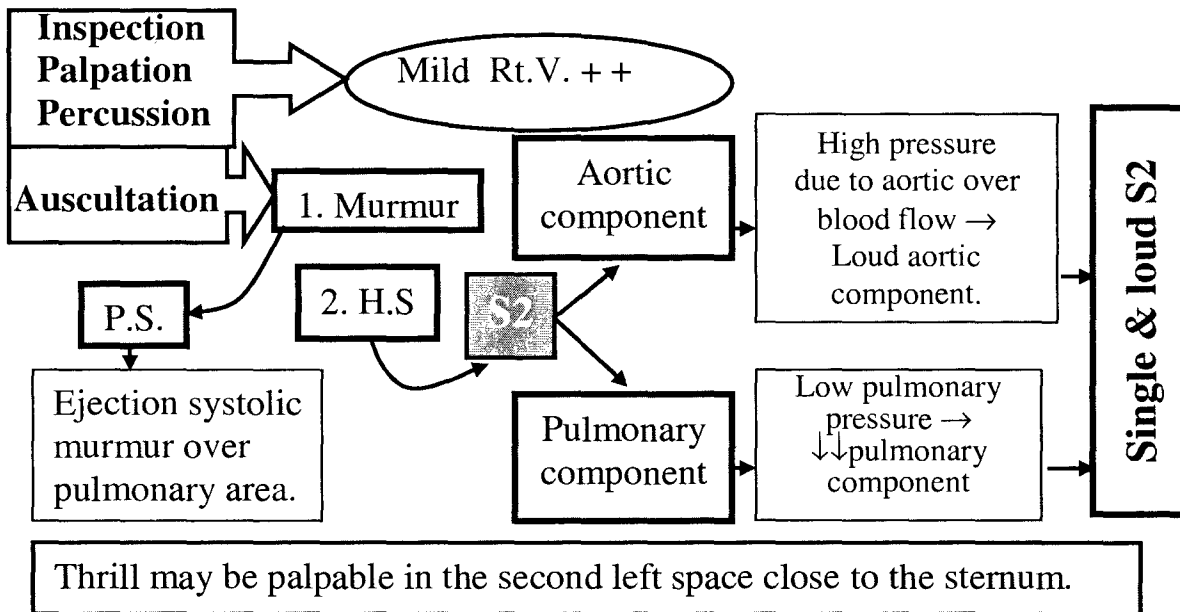
➡ It is mild → because it has 2 pathways: ① Pulmonary artery. ② Aorta.

### C/P

- |   |   |
|---|---|
| <ul style="list-style-type: none"> <li>① Cyanosis since birth or when the ductus closes, the time of it's closure is very variable i.e it may close late, even after one year.</li> <li>③ Clubbing of fingers.</li> <li>⑤ Stunted growth</li> </ul> | <ul style="list-style-type: none"> <li>② Secondary polycythemia usually present.</li> <li>④ Squatting position</li> </ul> |
|---|---|



**O/E**



**Investigations**

1. X-ray      • Coeur en sabot heart (Rt V++)
2. ECG        • Right ventricular hypertrophy.
3. Echocardiography
4. Catheter    • Measures pressure → high right ventricular pressure.  
                   • Measures O<sub>2</sub> → Low O<sub>2</sub> of aorta .  
                   • Passes through anomaly (through VSD)  
                   • Injection of dye → reveal the abnormality.



# Treatment:

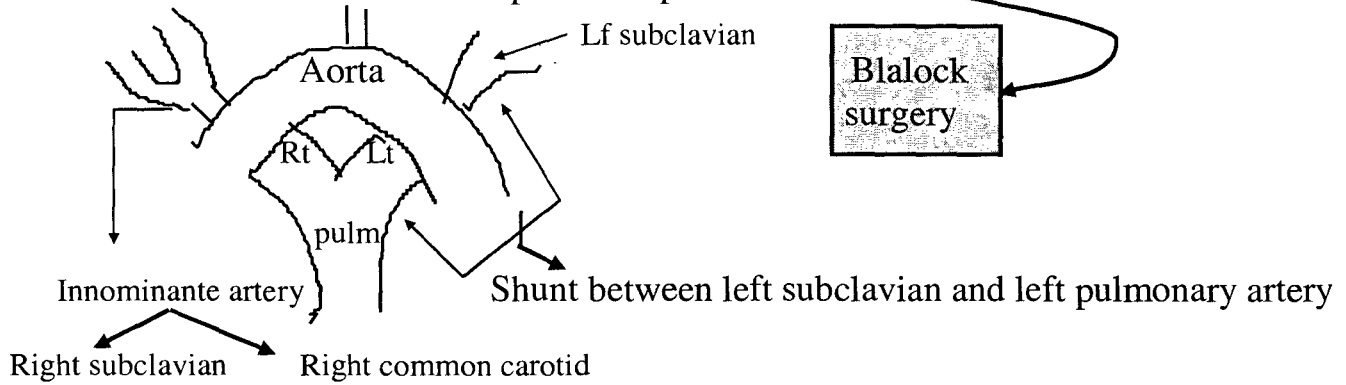
(A) **Medical:** Treatment of Fallot's spells and prophylaxis for endocarditis.

(B) **Surgical:**

## I- Artificial patent duct

It is a palliative procedure called

**Blalock surgery**



**VALUE:**

- = Improvement of cyanosis and general condition.
- = Improvement of the vasculature of the attenuated pulmonary vessels.
- = Sometimes it is done in cases of too hypoplastic pulmonary arteries without total correction specially in later life.

## II- Total correction

(the Best)

Complete surgical correction is possible even in infancy, before the occurrence of hypoplasia of pulmonary arterioles.

**Q =**

	<b>F-TRIOLOGY</b>	<b>F-TETRALOGY</b>
<p>Huge Rt.V</p>	<p>ASD + PS + Huge RT.V</p> <ul style="list-style-type: none"> <li>☛ Huge right V.</li> <li>☛ Congested neck veins.</li> <li>☛ Cyanosis is delayed</li> <li>☛ S2 is weak with wide splitting</li> </ul>	<ul style="list-style-type: none"> <li>☛ Mild RtV++.</li> <li>☛ Neck veins is not congested.</li> <li>☛ Cyanosis Since birth.</li> <li>☛ Single loud S2</li> </ul>
		<p>* The reversed shunt → cyanosis, this is not Eisenmenger's syndrome because there is no P++</p>

F. Pentalogy (F4+ASD).



## PULMONARY STENOSIS

### ↻ Aetiology

- It is almost always congenital ↗ Valvular stenosis
- ↘ Subvalvular (infundibular stenosis)
- Carcinoid \$
- Rheumatic P.S. is rare.

### ↻ Hemodynamics

➤ PS → pressure load on Rt V → Rt V++ → Rt V failure.

### ↻ C/P (Mild case is asymptomatic)

- ↓ COP (stenotic lesion).
- Right ventricular failure → systemic venous congestion (pain in right hypochondrium and swelling of both LL)
- Chest pain (atypical) due to ↓ COP and Rt V++.

### ↻ O/E

➤ Rt V++, left parasternal heave, Rt V failure ➤ Thrill (on pulmonary area).

### Auscultation:

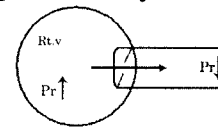
- 👂 S2 → pulmonary component ↓↓ with wide splitting.
- 👂 S3 → with failure over the tricuspid area (gallop).
- 👂 S4 → tricuspid area.
- 👂 Murmur
  - Ejection systolic on pulmonary area propagated.
  - lower down with infundibular stenosis (like VSD !?)
  - It is harsh and increased with inspiration.

Valvular PS	Subvalvular PS
<ul style="list-style-type: none"> <li>• Ejection systolic click</li> <li>• Post-stenotic dilatation</li> </ul>	<ul style="list-style-type: none"> <li>- ve.</li> <li>- ve.</li> </ul>

### ↻ Investigations

Echo or catheter to measure pressure gradient across pulmonary valve.

If the pressure gradient > 50 = severe PS.



- ttt • Mild to moderate cases dose not require treatment (low risk of endocarditis)
- Valvotomy (balloon or surgical valvoplasty) in valvular type.
- Resection for subvalvular type.

**Q DD of Functional PS** or functional murmurs on pulmonary area:

- \* Innocent PS.
- \* Pulmonary hypertension.
- \* On top of ASD.
- \* Hyperdynamic circulation.

**Q P.I.:** It is rarely isolated and usually occur with pulmonary dilatation due to P++ (Graham steell murmur).



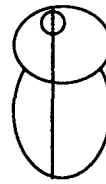
# ATRIAL SEPTAL DEFECT (ASD)

## Types

**Low ASD**  
(Ostium Primum)

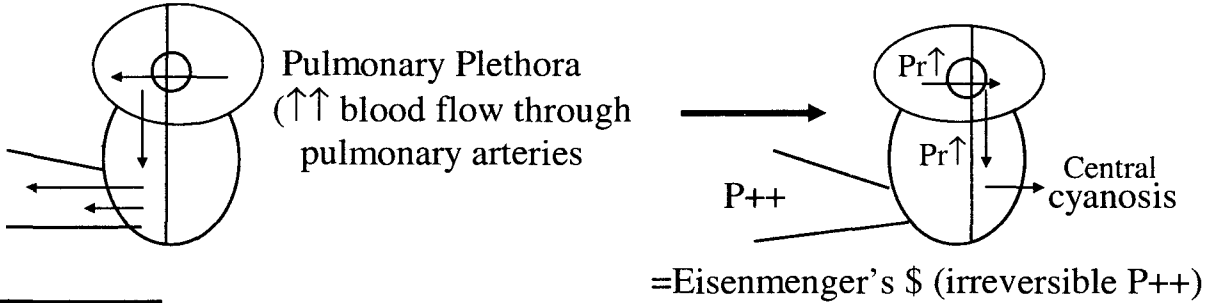


**High ASD**  
(Ostium secundum)  
It is the most common



♥ Low ASD is usually associated with mitral or tricuspid regurgitation.

## Hemodynamics

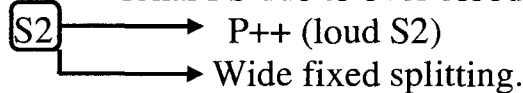


**C/P** (It is often diagnosed in adulthood, female > male)

- ① Recurrent chest infection, as plethora compromises alveolar macrophages.
- ② Right ventricular failure, Eisenmenger's syndrome occurs late.
- ③ Palpitation due to AF, (it is late with development of pulmonary hypertension).

**O/E**

- 👂 Left parasternal pulsation, pulsation of pulmonary area.
- 👂 Murmur: functional PS due to over blood flow through pulmonary valve.



## Investigations

- ① Catheter
  - 👉 Pressure is high in right atrial pressure
  - 👉 O<sub>2</sub> is high in right atrium.
  - 👉 Pass through anomaly.
  - 👉 Dye injection reveals the defect
- ② Echo
  - 👉 It is usually abnormal if significant defect is present, Transoesophageal echo may be necessary (more accurate).
- ③ Chest X-ray
  - 👉 Reveals prominent pulmonary artery & pulmonary plethora.

## Treatment

- 👂 Mild → follow up.
- 👂 Severe → i.e. pulmonary to systemic shunt ratio greater than (1.5:1) to (2:1), it should be repaired before age of ten years or as soon as possible if diagnosed in adulthood (to avoid occurrence of Eisenmenger's \$), closure by implantable closure devices with catheter.

### ECG in ASD

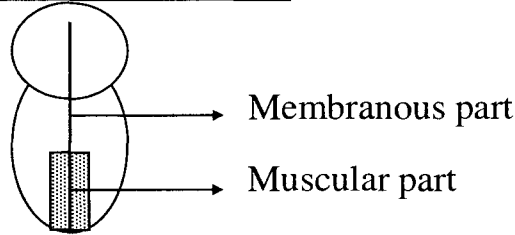
- Ostium primum → Left anterior hemiblock + LAD.
- Ostium secundum → RBBB + RAD.



## VENTRICULAR SEPTAL DEFECT (VSD)

VSD is the most common congenital heart disease (1 in 500 live births). It may be isolated or associated with other anomalies

### Parts of the Interventricular Septum:



### Types

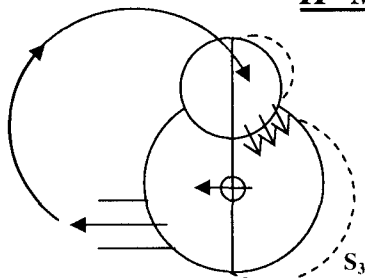
#### I- V.S.D in muscular part (Roger's disease)

It usually closes during systole so, It is haemodynamically insignificant.

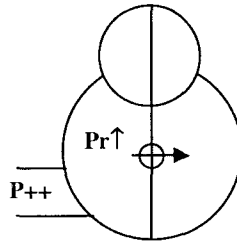
**C/P** → No chamber ++ (mild defect)  
→ Systolic murmur.

**ttt** → Usually it is closed spontaneously.  
→ Prophylaxis against endocarditis.

#### II- Membranous V.S.D



Pulmonary plethora



Eisenmenger's \$ with cyanosis

Plethora with ↑↑ blood flow to the left side

**Q-**

Chamber enlargement in VSD?

- ⊗ Left ventricle (volume load) → left ventricular dilatation.
- ⊗ Left atrium → left atrial dilatation.
- ⊗ Right ventricle (P++ → pressure load) → right ventricular hypertrophy.

**C/P**

- ☞ Recurrent chest infections.
- ☞ Palpitations (volume load).
- ☞ ↓ COP (severe VSD).
- ☞ Cyanosis
  - Exertional before the development of Eisenmenger's \$.
  - Eisenmenger's \$ at 2nd - 3rd decade
  - Associated PS.
    - ♦ No P++
    - ♦ cyanosis occurs at age of 3-5 years.

**O/E**

- ☞ Prominent apex with palpable thrill at the lower left sternal edge.
- ☞ Pulsations of pulmonary area.
- ☞ Murmur, pansystolic -loud – harsh – along the left sternal border (tearing character).
- ☞ Functional MS.
- ☞ Third HS (on the apex) -S2 wide splitting.

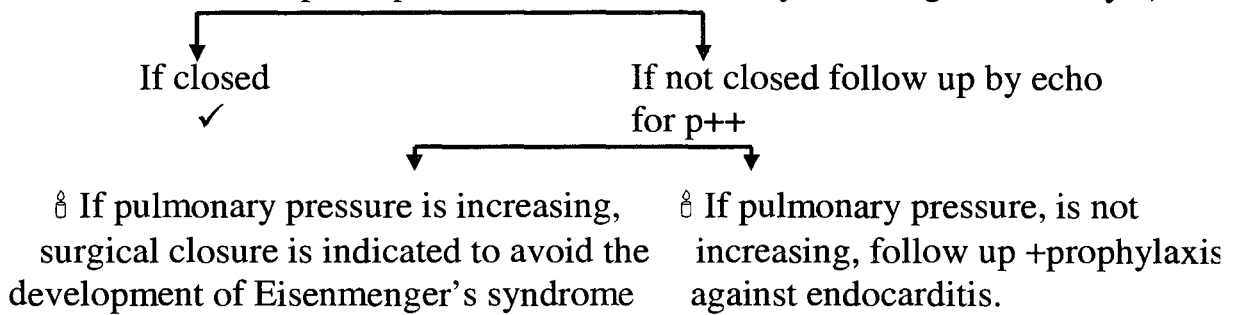


## Investigations

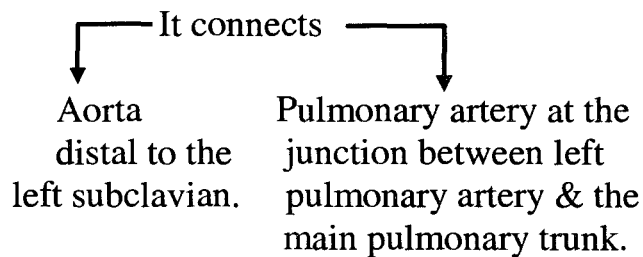
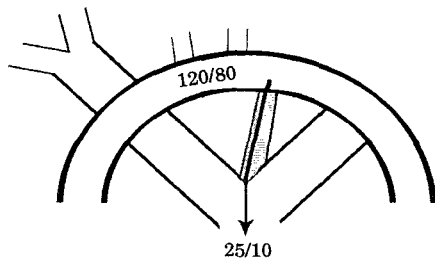
- ➔ ECG                   ♥ Left ventricular and left atrial enlargement.
- ➔ X-ray                 ♥ Pulmonary plethora- Chamber ++.
- ➔ Echo                 ♥ Chamber ++ & severity.
- ➔ Catheter           ♥ Pressure ↑↑ in right ventricle. ♥ O<sub>2</sub> ↑↑ in right ventricle.
- ♥ Passes through anomaly.           ♥ Dye injection showing the defect.

## Treatment

- 1- Moderate and large VSD should be surgically repaired before the development of pulmonary hypertension. Closure device through cardiac catheter are being developed. Prophylaxis against bacterial endocarditis should be advised.
- 2- Mild VSD needs: follow up till spontaneous closure (usually within age around 4 yrs)

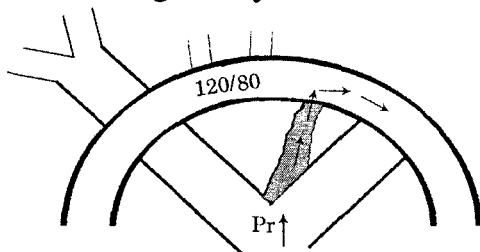


## PATENT OR PERSISTENT DUCTUS ARTERIOSUS



## Haemodynamics

Increase of blood flow to pulmonary arteries → pulmonary plethora → P++ → Eisenmenger's syndrome.



- Normal O<sub>2</sub> of upper 1/2
- Hypoxia of Lower 1/2

= Differential cyanosis

## C/P

- 1- Recurrent chest infections.
- 2- Hyperdynamic circulation (like P signs of AI).
- 3- Left sided failure in severe cases, Eisenmenger's \$ with differential cyanosis.





The hyperdynamic circulation is due to increase left heart blood flow and the decompression of the aorta into the pulmonary artery.

**O/E**

• *Murmur* → Machinery murmur left infraclavicular.  
 • *S2* → Reversed splitting.

**Investigations:**

**Catheter:** • Passes through anomaly. • Dye injection showing the anomaly  
 • Pressure → ↑ pulmonary pressure. • O<sub>2</sub> → ↑ Pulmonary O<sub>2</sub>.

**Echocardiography** (Echodoppler)

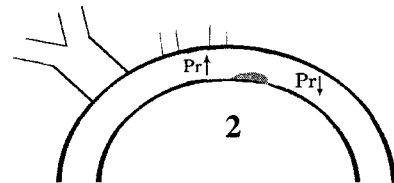
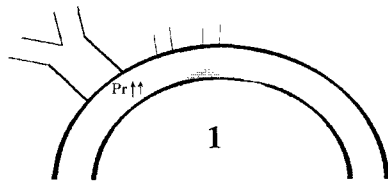
**Treatment:**

- Since there is serious complication (endocarditis) and the surgery is easy, so surgery should be as soon as possible and not be late to the age of 5 years. (Treatment by surgical ligation or by angiographically occlusive device)
- Premature infants can be treated medically with endomethacin which inhibits PG and stimulates duct closure.

**COARCTATION OF AORTA**  
(NARROWING OF AORTA)

**Types**

1- **Infantile type:** → incompatible with life, the narrowing is proximal to left subclavian. → very high pressure in head & neck → cerebral haemorrhage



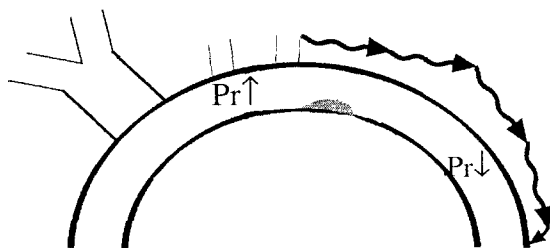
2- **Adult type:**

- The narrowing is just distal to the origin of the left subclavian artery (the isthmus)
- Coarctation of aorta is common in females than males; it may be associated with Turner's syndrome and bicuspid stenotic aortic valve.
- Coarctation of aorta is often asymptomatic for many years.

**Haemodynamics**

Upper 1/2 with ↑ pressure  
 Lower 1/2 with low pressure

collaterals to by pass the obstruction



Development of collaterals:

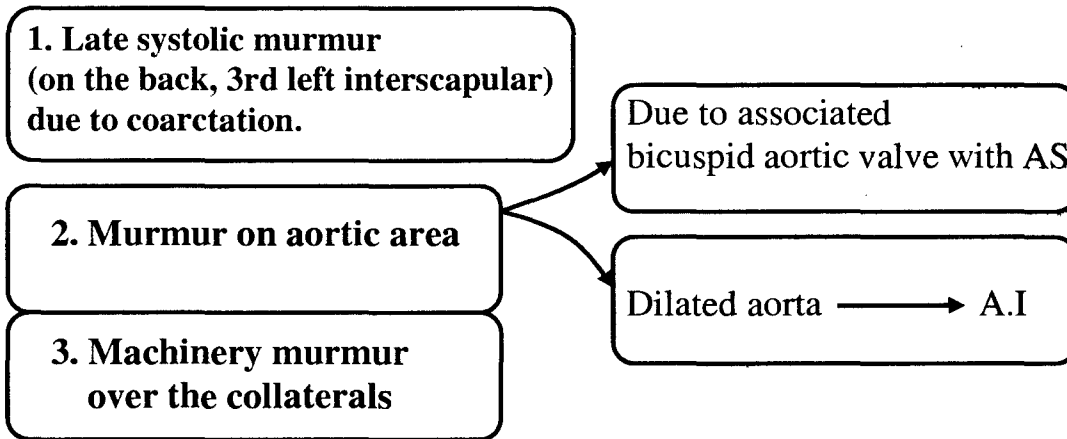
- The intercostal arteries.
- The prescapular arteries.



**C/P:** Associated anomalies: → Aneurysm of the circle of willis (Berry's aneurysm)  
 → Bicuspid aortic valve (80%).  
 → Turner's \$.

1. Blood pressure is high in UL and low in LL , radiofemoral delay.
2. Headache, epistaxis.
3. LL ischaemia with claudication pain.
4. **Suzman sign** (pulsating intercostal arteries in the back)

**5. Murmurs in cases of Coarctation:**



Decreased renal perfusion can lead to development of systemic hypertension that is usually persists even after surgical correction.

**Investigations** (MRI is diagnostic)

1. X-ray: (indentation of the descending aorta, 3 sign) i.e post-stenotic dilatation.
2. Aortography.
3. Catheter → measures pressure gradient across the coarctation
4. **Rosler's sign:** Notches in the lower parts of ribs due to pressure of the intercostal collaterals.

**Treatment**

Surgery in early childhood is better to avoid persistent hypertension.  
 ➔ Resection & anastomosis.  
 ➔ Prosthetic vascular graft may be needed.

When surgery is performed in early childhood, hypertension usually resolves completely. However, when the operation is performed on adolescents or adults the hypertension persists in 70% because of previous renal damage.

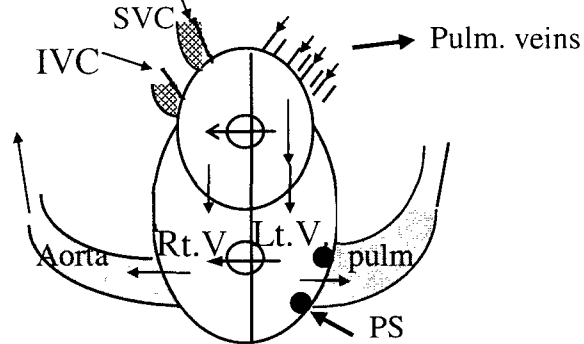
**Acquired Coarctation may occur due to trauma or Takayasu's disease.**



# TRANSPOSITION OF THE GREAT VESSELS

Aorta arises from the right ventricle & pulmonary artery from the left ventricle .

This leads to  
**2 separate circuits**



☛ To maintain life, communication between pulmonary & systemic circulation should be exist through → ♦ ASD ♦ VSD ♦ PDA

☛ PS (infundibular) Increases pressure in left side → Forces the oxygenated blood to the right side through shunt → aorta → cyanosis since birth.

## EBSTEIN'S ANOMALY

↙ ↘  
Huge right atrium      Cyanosis

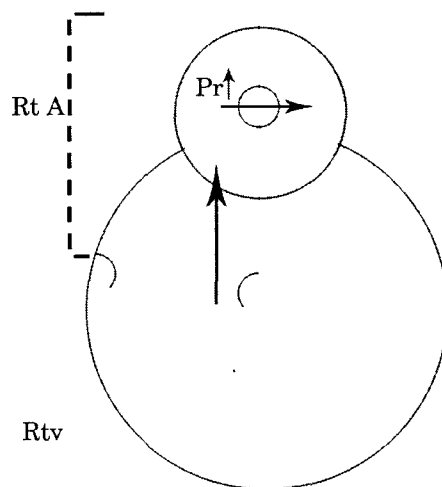
A rare congenital anomaly of the tricuspid valve in which the posterior leaflet of the valve is attached lower down to the mass of the right ventricle → TI, also there is ASD

**So there are:**

- ☛ Enlarged right atrium (Atrialization of right ventricle).
- ☛ Central cyanosis results from blood shunting through ASD (reversed shunt).
- ☛ Congenital tricuspid regurge.

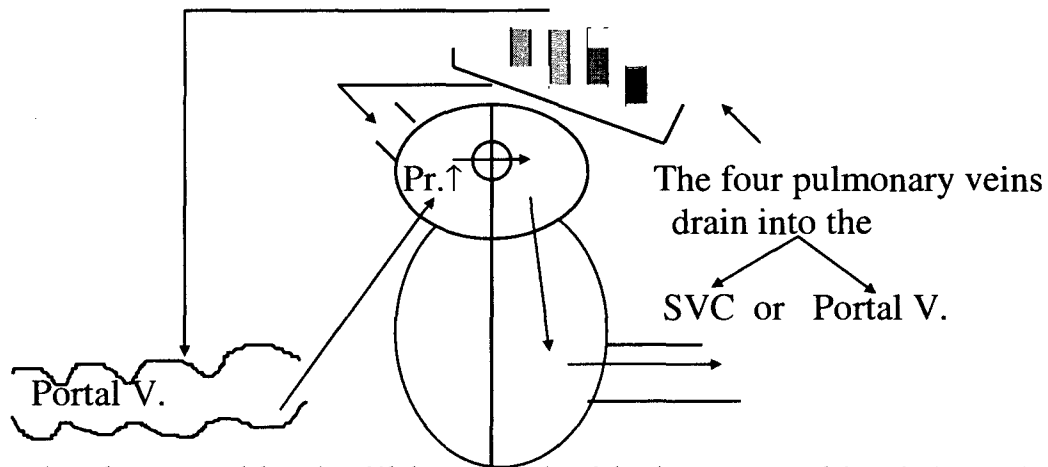
**C/P** ☛ Central cyanosis since birth. ☛ Murmur of TI.

**Treatment** Valve replacement & closure of ASD.





## TOTAL ANOMALOUS PULMONARY VENOUS DRAINAGE



- \* Oxygenated pulmonary blood will be mixed with the venous blood through ASD → left atrium → central cyanosis.
- \* Pulmonary veins are narrow due to fibrous bands → PVC.

## MALPOSITIONS OF THE HEART

- **Isolated dextrocardia:** Mirror like position of the heart, the apex is in the right side of the chest.
- **Situs inversus totalis:** Mirror like position of the heart as above and other viscera i.e. Traub's area on the right and liver dullness is present in the left side.
- **Levocardia:** Left sided apex with situs inversus.
- **Mesocardia:** Midline apex.
- **Dextroversion:** Simple displacement of the heart to the right side, it may be congenital or acquired due to right sided pulmonary fibrosis.

## EISENMENGER'S SYNDROME

- ♥ It occurs in about 20 % of cases of left to right thunts especially VSD.
- ♥ The development of this syndrome is genetically determined due to persistence of the fetal pattern of the pulmonary arterioles!?

**C/P**

- ☞ Cyanosis, dyspnea, angina of effort, exertional syncope and arrhythmia.
- ☞ Clubbing. ☞ P++ → ↓ COP.
- ☞ Signs of P++
  - Pulsating pulmonary artery.
  - Dullness on pulmonary area.
  - Loud S2 (pulmonary component).
- ☞ Raised venous pressure.
- ☞ No murmurs from the septal defect (low flow across the defect) i.e the pressure gradient becomes lower.
- ☞ Murmur due to P++ and murmur of TI due to right ventricular enlargement.
- ☞ Ankle edema with right ventricular failure.



**D.D.:**

- ◆ Fallot's tetralogy-primary P++ - Transposition of great vessels.
- ◆ Interstitial pulmonary fibrosis with patent foramen ovale.

**Investigations**

1. X-ray → P++.
2. E.C.G. → P- Pulmonale.
3. Echocardiography & cardiac catheter.

**Eisenmenger's complex:**

Originally described by Eisenmenger, which is pulmonary hypertension with reversed shunting through VSD.

**Eisenmenger's syndrome:**

It includes pulmonary hypertension with reversed shunting through VSD, ASD or PDA.

**Treatment**

- ⇒ Surgery is of no value as the shunt acts as a safety valve.
- ⇒ Diuretics for right ventricular failure, digoxin for A.F, Antibiotics for invasive procedures and for infective endocarditis, anticoagulants for thrombo- embolism.
- ⇒ Heart & lung transplantation.

**Complications of congenital heart diseases:**

- ☠ Infective endocarditis e.g in VSD & PDA.
- ☠ Secondary polycythaemia
- ☠ Eisenmenger's S and its complications e.g. right ventricular failure, sudden death, polycythaemia, infective endocarditis and paradoxical embolism.
- ☠ Heart failure e.g huge VSD.
- ☠ Arrhythmia
- ☠ Paradoxical emboli → i.e DVT

↓  
Emboli to the right side of the heart

↓  
The emboli are shunted to the left side of the heart through the shunt

↓  
Systemic embolization.

- ☠ Renal failure with marked low COP.
- ☠ Exertional syncope in cases of Fallot's tetralogy, also this occurs with cardiac shunts with high pulmonary vascular resistance as systemic vascular resistance fall on exercise but pulmonary vascular resistance remains high → syncope (hypoxic syncope).

## **Q, Presentations of congenital heart disease :**

1- Birth and neonatal period .

- Cyanosis .
- Heart Failure .

2- Infancy and childhood

- Cyanosis - Heart failure - Arrhythmia - Failure to thrive.

3- Adolescence and adulthood :

- Hypertension (Coarctation of aorta). - Arrhythmia .
- Cyanosis . ( Eisenmenger s syndrome) - Heart failure.

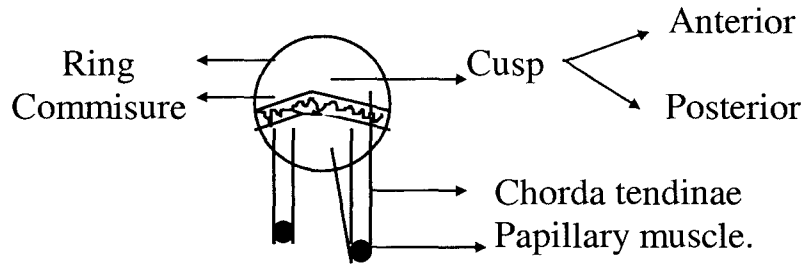
## **Cardiovascular disease with pregnancy**

- Pregnancy is associated with hemodynamic stress to the patient with pre-existing heart disease e.g rheumatic or congenital heart disease.
- During normal pregnancy , plasma volume increases with increase in stroke volume ,heart rate so ,  $CO \uparrow$  .
- During labour uterine contractions  $\longrightarrow$  increase of up to 500 ml of blood in the central circulation .
- symptoms and signs that may mimic cardiac disease often accompany these hemodynamic changes e.g fatigue , LL edema , distention of neck veins , S3 and functional systolic murmurs .
- Hypertension during pregnancy include chronic hypertension , gestational hypertension and toxemia .
- Peripartum cardiomyopathy is a form of dilated cardiomyopathy .
- About 50% of aortic dissections that occur in women younger than the age of 40 are associated with pregnancy . It is postulated that hemodynamic and hormonal changes associated with pregnancy may weaken the aortic wall.
- Many pregnant patients with known cardiac disease can complete a normal pregnancy and delivery without significant harm to the mother or fetus.



# MITRAL STENOSIS (MS)

## Anatomy of mitral valve:



### Valve area

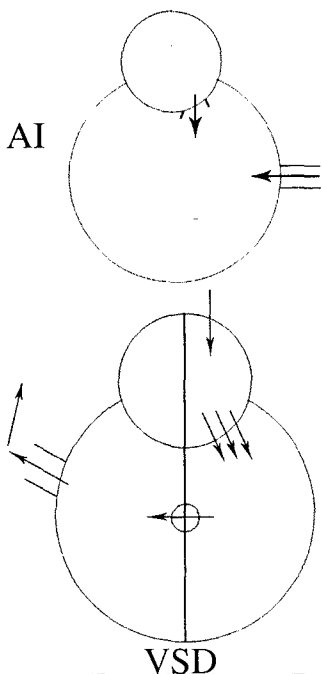
- It is about 4 - 5 cm
- If it is < 1 cm = tight mitral stenosis.

## Causes

### Organic

- MS is almost always **rheumatic**, so search for other valve lesions. (because rheumatic fever usually leads to multi valvular lesions).  
MS is the most common single valve lesion due to rheumatic fever so, isolated rheumatic MS may occur.
- Other causes of organic MS.
  - Calcific MS in elderly, congenital Ms (rare).
  - Lutembacher's \$ (acquired MS + ASD).
  - Carcinoid tumours metastasizing to the lung, or primary bronchial carcinoid.

### Functional



### Carey Coomb's murmur:

It occurs during rheumatic activity (edematous cusps).  
It is reversible so, it is a functional MS.

### Austin flint murmur:

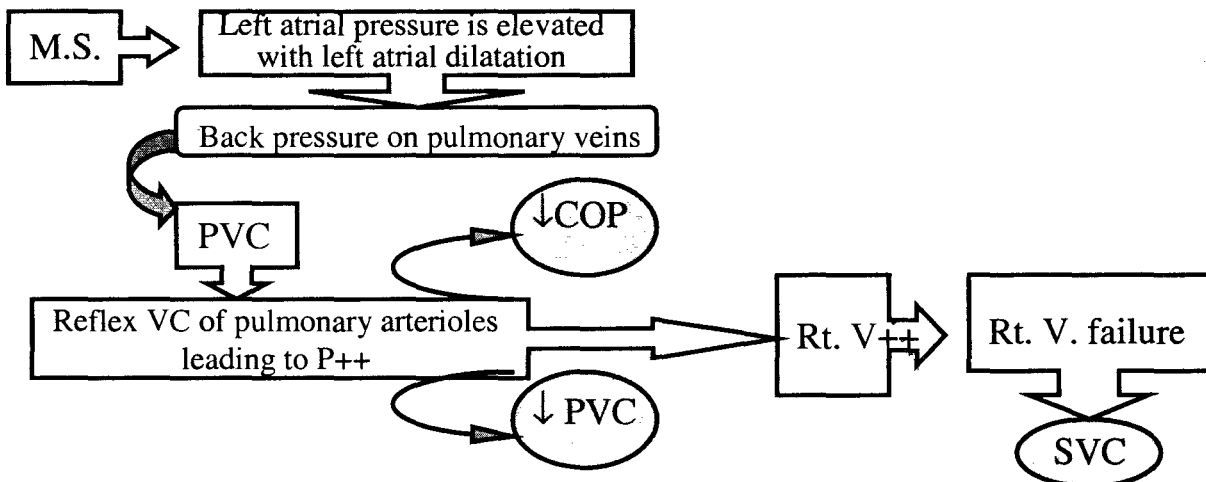
Occurs on top of severe AI specially syphilitic AI.  
The regurged blood interferes with the full opening of mitral valve.

### VSD → Functional MS as it leads to

Over blood flow across mitral valve.



## Haemodynamics



- Any increase in heart rate will shorten the diastole (the time of mitral valve opening), this leads to further rise in the left atrial pressure so AF, exercise and pregnancy are poorly tolerated.
- The onset of atrial fibrillation may precipitates pulmonary oedema because the tachycardia and loss of atrial contraction lead to marked haemodynamic deterioration with a rapid rise in the left atrial pressure.

- ⊛ Patient with rheumatic heart with P ++ → Suspect MS.
- ⊛ Patient with rheumatic heart with SVC → Suspect MS.
- ⊛ Rare types of M.S are congenital M.S, calcific MS in elderly.
- ⊛ Lutembacher's \$ is the combination of acquired rheumatic MS. with ASD

## C/P

**(USUALLY NO SYMPTOMS UNTIL THE VALVE ORIFICE HAS AN AREA OF 2 CM.)**

- Stages**
- ♥ Asymptomatic MS.
  - ♥ PVC (congestive MS).
  - ♥ P++ ⊕ Rt V ++.
  - ♥ SVC (right sided failure).

- Symptoms**
- ★ Symptomes of PVC, ↓ COP late SVC.
  - ★ ↓ COP symptoms are usually exertional so, they don't occur early because of dyspnea, So with development of P++ → ↓ PVC → relieve of dyspnea → exertion → ↓ COP symptoms become manifested !?.

### Signs

#### ⇨ General

- ♥ **Malar flush** (mitral facies)  
Dusky pink discoloration over cheeks due to A-V anastomoses and vascular stasis !?
- ♥ **PVC** → Bilateral fine basal crepitations on the lung.





- ♥ ↓ COP:
  - Low systolic blood pressure.
  - Weak pulse volume.
  - Pallor.
  - Peripheral cyanosis.
  - Cold extremities.
- ♥ Right sided failure
  - Congested neck veins (prominent a wave).
  - Enlarged tender liver.
  - Lower limb edema.
- ♥ Late AF may occur.

⇨ Local

**Inspection & palpation for:**

- ♥ Chamber enlargement.
- ♥ Thrill.
- ♥ Palpable sounds.

**Stage of PVC**

- ♥ Chamber ++ i.e left atrial dilatation.
- The dilated left atrium push the heart forward, so right ventricle become nearby chest wall → (left parasternal pulsation).
- ♥ Diastolic thrill on the apex in left lateral position.
- ♥ Palpable S1 with weak apex amplitude i.e slapping apex.

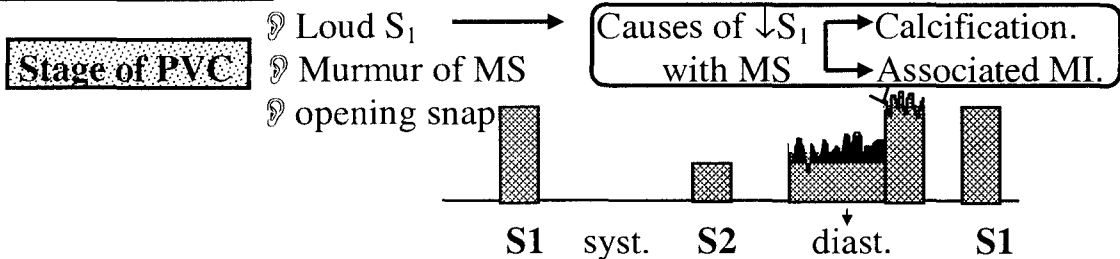
**Stage of P++:** (pulmonary hypertension)

- ♥ Pulsating (pulmonary area).
- ♥ Palpable S2
- ♥ left parasternal heave (Rt v++)
- due to dilated pulm. artery. (diastolic shock).

**Stage of right sided failure:**

Signs of P++, right ventricular and SVC.

**Auscultation**



☞ Murmur → mid diastolic rumbling with presystolic accentuation.

Q Causes of Silent MS i.e (↓↓ blood flow through mitral valve → no murmur).

**1. Associated ASD:**

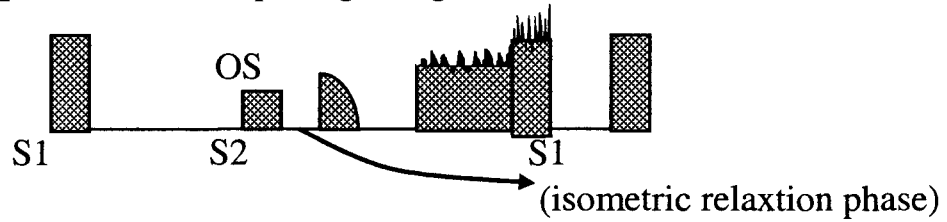
ASD + MS = Lutembacher' \$

**2. P++:** (Pulmonary vasoconstriction → ↓ blood flow to the lung → ↓ blood flow to the left side of the heart).

**3. Right sided heart failure.**



**Opening snap** It is due to opening of rigid mitral valve

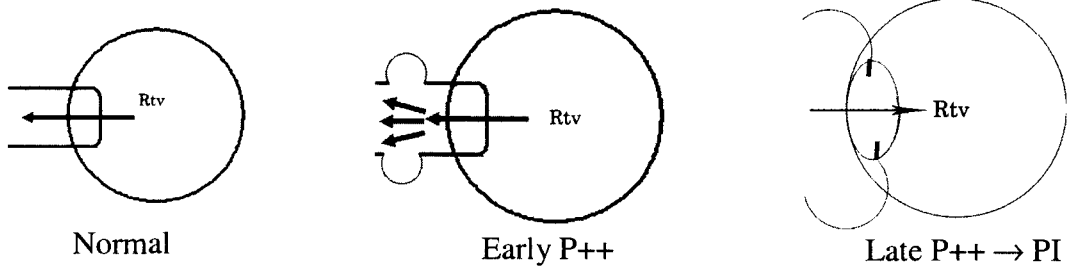


**Value:**

- Diagnosis of organic MS
- Pliable mitral valve.
- It is absent in calcified MS.
- Severe MS → high left atrial pressure → open the mitral valve early  
→ OS becomes near by S2.

**Stage of P++**

- S2 ↑↑ (pulmonary component) + closed splitting of S2
- S4 due to rise of right ventricular pressure → Vigorous right atrial contraction → S4 (on tricuspid area)
- Systolic murmur on pulmonary area due to dilated pulmonary artery.
- Late dilatation of pulmonary valve ring → PI (Graham Steell murmur).



**Stage of right sided failure**

P++ → Right V failure → Gallop on tricuspid area (3rd H.S.+ tachycardia)

**Investigations**

1. X-ray ☉ Left atrial ++ ☉ Right V + ☉ Pulmonary venous congestion.
2. ECG ☉ Lt atrial + ☉ Rt V +
3. Echo ☉ Valve lesion. ☉ Calcification. ☉ P ++.  
☉ Valve area. ☉ Chamber ++.  
☉ Pressure gradient across mitral valve.
4. Catheter: ☉ left atrial pressure is high.

$$\text{MS index} = \frac{\text{COP}}{\text{Left atrial pressure}} \times 100 = \frac{5}{5} \times 100 = 100\%$$

MS leading to  $\frac{\downarrow \text{COP}}{\uparrow \text{left atrial pressure}} \rightarrow \downarrow \downarrow$  index, if < 25% = Tight MS

We can measure MS index by echo doppler.



## Treatment

### ⇒ Mild cases with mild PVC e.g mild dyspnea.

#### Medical & follow up

- ☞ Prophylaxis for rheumatic fever.
- ☞ Prophylaxis for endocarditis, (it is uncommon with MS)
- ☞ PVC can be relieved by diuretics → ↓ venous pressure.
- ☞ Atrial fibrillation treated with digoxin and anticoagulants to prevent thromboembolism.
- ☞ Prompt therapy of attacks of chest infections.

### ⇒ Severe cases with P++ or PVC not responding to medical ttt

#### Surgery

☞ According to echo finding:

- ☛ Calcified MS or associated MI → valve replacement
- ☛ Pure MS → **Valvotomy**

**Balloon catheter**

**Valvotomy**

**Surgery**

i.e. Balloon valvuloplasty

Open valvotomy

Closed Valvotomy

A balloon catheter is introduced into the right atrium via the femoral vein. The inter-atrial septum is then punctured and the catheter advanced into the left atrium and then to mitral valve.

It is preferred to closed valvotomy.

The fused cups are forced apart by a dilator introduced through the apex.

- ✓ It is better to operate early as long as the patient is symptomatizing e.g dyspnea and orthopnea
- ✓ Early operation before the development of P++ → good results.
- ✓ Late operation → irreversible P++ even after surgery.
- ✓ If there is P++ , we can do test of reversibility of P++, measure pulmonary blood pressure by echo before and after intake of Ca.Ch. B, if the pulmonary pressure ↓ → good prognosis!?
- ✓ Prophylaxis against endocarditis is essential after valvotomy and follow up every year is essential as restenosis may occur. Prophylaxis is essential after valve replacement.

### Criteria for mitral valvuloplasty:

- No or mild MI.
- Non calcified valve.
- Left atrium is free of thrombus.

### Complications & sequelae of M.S

1. P++
2. Pulmonary edema
3. DVT & pulm embolism
4. AF
5. Infective endocarditis
6. Rt sided heart failure
7. Calcification of mitral valve
8. Systemic embolization



## Tight MS

### Diagnosis

- ✎ The murmur is prolonged.
- ✎ OS is nearby S2.
- ✎ Echo valve area < 1 cm.
- ✎ P++.
- ✎ MS index < 25%.

### Treatment

Surgery (valvotomy or valve replacement according to echo findings, as before).

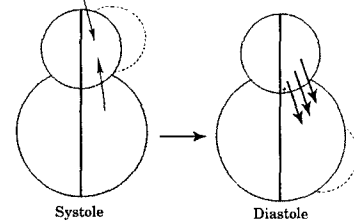
## Mitral incompetence (MI)

### Causes:

- Organic:** ① Rheumatic ② Infective endocarditis. ③ Mitral valve prolapse (see later)  
 ④ Ischaemic i.e. ischaemic papillary muscle. ⑤ HOCM. ⑥ SLE.

### Functional:

☞ Due to dilatation of left ventricle e.g on top of AI, heart failure or dilated cardiomyopathy.



### Haemodynamics:

Longstanding M.I → regurge to LA with little increase in left atrial pressure due to left atrial dilatation → ++ blood flow to Lt V → Volume overload on Lt V → Lt V dilation → late failure will occur → PVC.

In acute MI the normal compliance of the left atrium does not allow much dilatation and the left atrial pressure rises, see later (backward failure).

### Q. Patient with MI ⊕ PVC How !?

- ☞ Associated MS.
- ☞ MI → LtV failure, it is late because:  
 the blood has 2 pathways → to the low pressure of the left atrium & to the high pressure of aorta
- ☞ MI → regurged blood to left atrium → mild P.V.C.

### Symp:

1. Palpitation: due to volume overload.
2. PVC as a direct result of MI or due to left ventricular failure
3. ↓ COP, especially with severe cases.
4. Late symptoms of right sided failure will developed.

### O/E:

☞ local

### ☞ Inspection and palpation:-

- ① Left V ++
- ② Hyperdynamic apex.
- ③ Systolic thrill on the apex.



**Auscultation** **S1** is muffled.

The murmur is pansystolic,  
Soft blowing,  
maximum  
intensity on the apex  
(Anterior leaflet MI).

**S2** PVC. → P++ → Loud S2

**S3** Lt VF (Late) i.e. Gallop, it may be present also  
due to overflow of blood to the Lt V.

**S4** Over tricuspid area due to P++ , late.

Posterior leaflet MI, the murmur  
maximum intensity on the apex and  
propagates to left parasternal area.

Signs of P++, AF and right sided failure may develop later in the disease.

**Investigations:**

- ☉ X-ray - ECG      ⚫ Lt V ++, left atrial dilatation.
- ☉ Echo              ⚫ Diagnosis of MI, chamber ++.
- ⚫ Severity of MI.

**Treatment:**

**Mild MI** i.e (mild symptoms, the left ventricle is not dilated, no third heart sound and by echo), it is treated by prophylaxis against rheumatic fever and endocarditis, diuretics to ↓ PVC , digoxin and anticoagulant for AF, ACE inhibitors (vasodilators) may decrease the severity of regurge and the remodeling process !?

**Severe MI** Valve replacement or mitral valve repair.

- Acute severe mitral regurgitation may occur due to ruptured chorda tendinae or perforated cusps due to infective endocarditis or due to rupture of papillary muscle on top of myocardial infarction. With severe MI the compliance of the left atrium doesn't allow much dilatation, and the left atrial pressure rises → PVC → Pulmonary oedema, this is called backward failure.
- Chronic MI → gradual dilatation of the left atrium with little increase of atrial pressure.
- In double mitral lesion, what is the dominant lesion!?
- ❖ Dominant MS.: S1↑, P ++, weak apex in the 5<sup>th</sup> space, opening snap.
- ❖ Dominant MI.: S1 ↓ , hyperdynamic apex, dilated left ventricle (apex is below the 5<sup>th</sup> space).

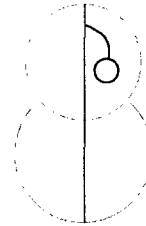


## Left Atrial Myxoma

Benign tumor from the interatrial septum encroaches on the left atrium.

### C/P

- Young female.
- Recurrent syncope.
- Embolism
- Positional MS (rumbling), loud S1 and a tumor plop (S3 is produced as the pedunculated tumor comes to an abrupt halt)



**Investigations:** Echo - ↑ ESR

**Treatment:** Resection.

- ☞ Primary cardiac tumors are rare but the heart and mediastinum may be the site of metastases.
- ☞ Most tumors are benign e.g. fibroma, lipoma and haemangioma.
- ☞ Myxoma is the most common primary tumour of the heart (benign).

**Incompetence = Regurge = Insufficiency = Reflux.**

## Mitral Valve Prolapse

**= (Click murmur \$) = (Floppy mitral valve)**

Degenerative disease of mitral valve cusps (myxomatous) → Prolapse of mitral valve cusps into left atrium (mostly posterior leaflet). The cause is unknown but it may be associated with marfan's syndrome and thyrotoxicosis.

### Pathophysiology:

During ventricular systole, a mitral valve leaflet (commonly the posterior leaflet) prolapses into the left atrium leading to abnormal ventricular contractions, papillary muscle strain and sometimes mitral incompetence.

### C/P

- Young female.
- Atypical chest pain.
- Recurrent arrhythmia with palpitation.

### Diagnosis

- ✓ Mid systolic click.
- ✓ Late systolic murmur.
- ✓ Echo.

### Treatment

- Beta-blockers (prophylaxis against arrhythmia and for the chest pain).
- Prophylaxis against endocarditis with significant M.I.
- Valve replacement for severe regurge.

- Mild mitral valve prolapse is so common that it may be regarded as a normal variant!?
- Mitral valve prolapse sometimes associated with marfan's syndrome and thyrotoxicosis.

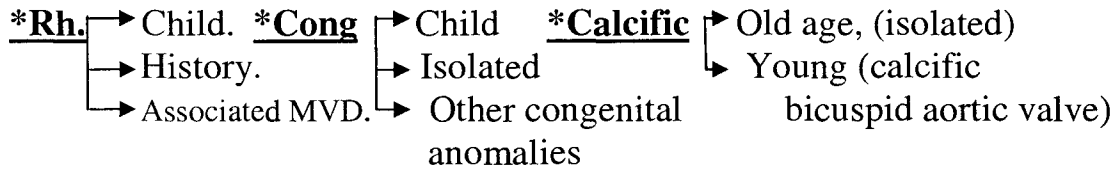


# Aortic stenosis (AS)

## Causes:

### A) Organic Causes:

#### 1. Valvular:



#### 2. Subvalvular AS: with HOCM.

3. Supravalvular AS: with elfin facies, mental retardation (william's syndrome), in which there is fibrous diaphragm above aortic valve.

### B) Functional causes : Severe AI – hperdynamic ciculation

## Haemodynamics

A.S → presure load on left V → left V hypertrophy → ischaemia → heart failure.

- In contrast to mitral stenosis which tends to progress slowly, patients with AS typically remain asymptomatic for many years but deteriorate rapidly when symptoms develop.
- Rh. AS is almost always associated with MVD.

## C/P: Triad

(The symptoms are usually extertional)

- ☞ Low COP e.g dizziness, fatigue and syncope.
- ☞ Chest pain due to → ↓ cop and left ventricular hypertrophy.
- ☞ Left ventricular failure in advanced stages.

### ⇒ ↓ COP (syncope)

☞ At rest: Syncope at rest may occur in calcific AS in old age due to calcification in conductive system (AVB) → heart block (Adam's stock attacks).

☞ Exertional: —————→ occurs in any type of AS.

### ⇒ Chest pain:

\* May be due to 
}

 Low COP.  
 Left V hypertrophy.

\* Or associated coronary atherosclerosis with calcific type (old age)

### ☉/☞ Platuae pulse (small volume with slow-rising)

## ⇒ Inspection & palpation:

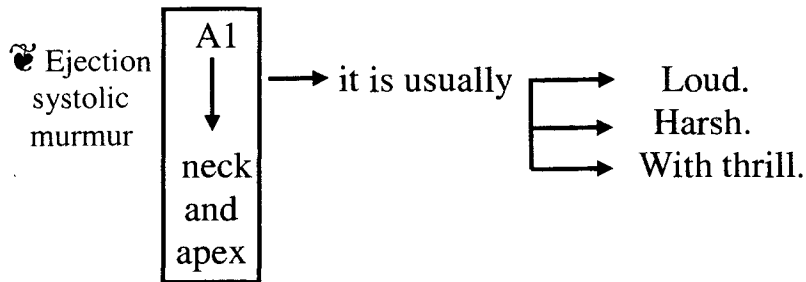
☉ Left ventricular hypertrophy, apex is sustained (thrusting apex) and localized at 5<sup>th</sup> space due to concentric hypertrophy of left ventricle, shift of the apex is a late finding.



➔ Thrill (base → neck) = organic aortic stenosis.

➔ Auscultation

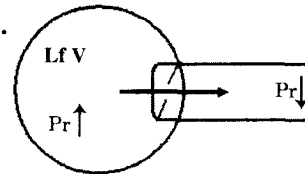
👂 **Murmur**



- 👂 **S1** : (Left ventricular hypertrophy) → ↑ muscle component of S1.
- 👂 **S2** : ↓ Aortic pressure → ↓ S2 (aortic component), reversed splitting of S<sub>2</sub>.
- 👂 **S3** : Left ventricular failure (late) (S3 on the apex) i.e. Gallop.
- 👂 **S4** : AS → Left ventricular diastolic dysfunction → ↑ diastolic pressure of the LV → vigorous atrial contraction (S4 on the apex).

➔ **Investigations**

- 1) ECG, X-ray : → Left V ++ with strain in severe cases.
- 2) Catheter to measure pressure gradient across aortic valve, Pressure Gradient > 50 mm. Hg = severe AS.
- 3) Echo : valve area < 0.7 cm<sup>2</sup> (severe AS), normally it is 2.5 cm<sup>2</sup>.



Also it can measure pressure gradient across aortic valve

➔ **Treatment:**

(No role for medical treatment, digitalis diuretics , BB may ↓ symptoms.!?)

- ➔ Mild AS, follow up with echo to detect the progression of stenosis.  
Angina best treated with BB. Vasodilators or nitrates are better avoided as they aggravate the exertional syncope.
- ➔ Surgery: Valve replacement.  
Indications (all symptomatic patients, pressure gradient >50), any delay in surgery → irreversible deterioration of ventricular function with risk of sudden death.
- ➔ Aortic balloon valvoplasty → transient improvement.  
It is useful in congenital AS (non calcific).

The intensity of the murmur is not a good guide to severity of aortic stenosis because it is decreased by reduced COP So, in severe cases the murmur may not heard.

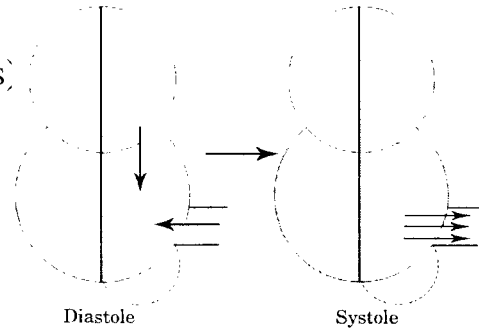




# Aortic incompetence (AI)

## Causes:

- 1) Rheumatic fever.
- 2) Syphilis → aneurysm of ascending aorta → AI.
- 3) Post-valvotomy
- 4) Systemic diseases:
  - i. Ankylosing spondylitis (aortitis)
  - ii. Marfan's
  - iii. Rheumatoid disease (aortitis).
- 5) Endocarditis.
- 6) Dissection of the aorta (acute AI)



## Hemodynamics:

- ✓ There is regurgitation of blood to the left ventricle during diastole → LV dilatation.
- ✓ The systolic pressure is increased due to ↑ stroke volume, (the stroke volume and COP of the left ventricle may be doubled or tripled).
- ✓ The diastolic pressure is decreased due to the aortic runoff during diastole, this will lead to wide pulse pressure (hyperdynamic circulation).
- ✓ As the disease progresses, left ventricular diastolic pressure rises especially with exercise → dyspnea, left ventricular failure occurs late.

## C/P:

• Mild cases may be **asymptomatic except for palpitation.**

• **Chest pain** + **palpitation**

↓  
 Due to ↓↓ diastolic pressure                      Due to volume load

↓  
 ↓ Coronary filling

- **Arrhythmias** are relatively uncommon.
- **Dyspnea** due to ↑ left ventricular diastolic pressure.
- Late → **sympt of heart failure.**

Mild AI → Palpitation.  
 Severe AI → Dyspnea, angina and palpitation

## Peripheral signs of A.I (Due to hyperdynamic circulation)

- 1) **Corrigan's sign:** Marked prominent carotid pulsation.
- 2) **De-Musset sign:** Nodding of the head with each heart beat.
- 3) **Systolic thrill over the carotid:**  
 Due to rapid flow of blood (carotid shuddering).
- 4) **Water hammer pulse.**



**5) B.P.:** → Systolic ↑  
Diastolic ↓ } → Wide pulse pressure.

**6) Capillary pulsation:**

(Quinke's sign) may be detected in nails, also it can be detected in lips, lobule of ear or uvula (Muller's sign).

**7) Pistol shot femoralis:**

It is a loud sound heard with each pulse beat over the femoral artery (Traube's sign).

**8) Duroziez's sign:**

Systolic & diastolic murmurs are heard over the femoral artery with a slight pressure applied with a stethoscope.

**9) Hill's sign:**

Normally blood pressure in LL is higher than in UL by about 20 mmHg, in AI the difference may be > 40 mmHg.

⇒ **Local examination:**

- ✓ Lt V ++ (dilatation).
- ✓ Hyperdynamic apex.
- ✓ ↓ S2 aortic component, fourth heart sound (Apex).
- ✓ Murmur:

A2  
Apex. } → Soft, high pitched decrescendo character.

**Murmurs over the apex in case of A.I.**

- ☞ AI.
- ☞ Functional MI due to Lt V dilatation.
- ☞ Austin flint murmur (MS) as the regurgitation impinge on the anterior mitral valve cusp.

**\* Causes of absence of peripheral signs in cases of AI?**

- MS.
  - AS.
  - Lt VF.
  - Mild A.I.
- ↓ COP → ↓ systolic blood pressure → narrow pulse pressure

**\* (Peripheral signs if present, means that the left ventricle is compensated)**

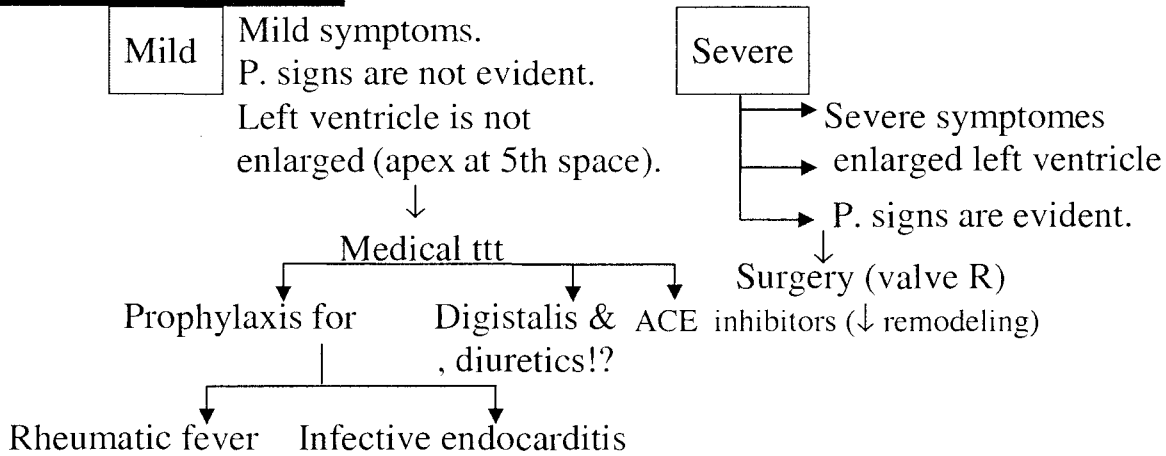
**\* Acute AI or acute MI lead to left sided heart failure → pulm.edema**

⇒ **Investigations**

- ECG, → Lt V++.
- Echo, x ray → Lf V ++ with dilatation of the ascending aorta.



**Treatment:**



Surgery is the best even in mild cases especially with cardiomegally or decline in the left ventricular function. ACE inhibitors prevent progressive left ventricular dilatation and are recommended for asymptomatic patients.

**Q Double aortic valve lesions**

What is the dominant lesion!?

- ❖ AI is dominant if the apex is hyperdynamic, also if there is wide pulse pressure with evident peripheral signs.
- ❖ AS is dominant if the apex is sustained, narrow pulse pressure with absence of peripheral signs.

**Tricuspid Stenosis (TS)**

**Etiology:**

- ✓ It is usually rheumatic in origin and usually associated with mitral or aortic valve disease.
- ✓ Carcinoid \$

**C/P**

Usually the symptoms of the associated mitral and aortic valve disease predominate.

- ☛ Low COP.
- ☛ SVC.
- ☛ If there is associated MS, TS will decrease PVC, and P++.
- ☛ Congested neck veins + presystolic hepatic pulsations.
- ☛ Mid diastolic rumbling murmur on tricuspid area, signs of SVC.

**Investigations**

👁 Echo showing right atrial enlargement, stenosis of tricuspid valve.

**ttt**

- Diuretics with salt restriction.
- Valvotomy (occasionally possible).
- VR (often necessary).
- Other valves also need replacement because TS is rarely an isolated lesion.



# Tricuspid Regurge (TR)

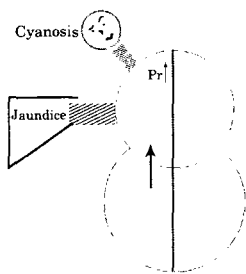
## Causes

- ✓ The commonest cause is functional due to Rt. V++.
- ✓ Rheumatic – congenital (Ebstein's anomaly).
- ✓ Endocarditis especially in IV drug abusers (staph).

## C/P

- ☞ Neck veins are congested. ☞ Cyanoicterus.
- ☞ Ascites precox !?
- ☞ Positive hepatojugular reflux, systolic expansile pulsations of the liver.
- ☞ Pansystolic murmur at the left sternal edge. It increases in intensity with inspiration, it can be heard to the right of the sternum.

## ttt



- ☞ Treatment of the underlying cause reduce the severity of TR i.e. relieve of right ventricular load (e.g P++) by mitral valvotomy or mitral valve replacement, or with the use of diuretics and vasodilators.
- ☞ Tricuspid valve annuloplasty or plication with severe functional TR, it can be done for example during mitral VR.
- ☞ Valve replacement for organic TR.

## Complications of Rheumatic heart disease

- Infective endocarditis.
- Heart failure, DVT.
- Systemic embolisation.
- Rheumatic activity.
- Arrhythmia.
- Pulmonary embolism.

# Heart Transplantation

## Indications:

- (1) Refractory end stage heart failure.
- (2) Heart disease grade 3,4 (symptoms at mild exertion or at rest)

## Exclusion criteria:

- Irreversible p++
- Active infection
- Advanced liver or kidney disease.
- Malignancy
- DM with end organ damage.

## Medications after transplantation:

- ① Cyclosporine
- ② Corticosteroids.
- ③ Azathioprine.

## Complications of cardiac transplantation:

- Rejection
- Infections e.g CMV; pneumocystis.

Cardiac denervation → High resting heart rate.



# Pulmonary Hypertension

The pulmonary vascular bed is normally of low pressure, the systolic pressure is about 25mmHg and the diastolic about 10mmHg.

## Causes of P++:

- ① Hyperkinetic P++ (overflow): ASD, VSD, PDA.
- ② Passive P++: LFV or MVD
- ③ Obstructive P++
  - ☛ Embolic, Schistosomiasis
  - ☛ Vasculitis, Sickle cell disease.
  - ☛ Primary pulmonary hypertension.
- ④ Reactive P++ e.g.: COPD- High altitude – Sleep apnea, (hypoxia → Pulm. VC)

## ♥ The causes of pulmonary hypertension can be divided into:

- **Precapillary:** (in the pulmonary arteries and arterioles).
  - Primary pulmonary hypertension.
  - Congenital heart with Eisenmenger's S.
  - Thromboembolic.
- **Capillary disorders:** (causing damage to the alveolar capillary mechanisms):  
Parenchymal lung diseases e.g emphysema and pulmonary fibrosis → hypoxia → vasoconstriction → P++, also they lead to decrease surface area of pulmonary vascular bed.
- **Postcapillary:** (passive) i.e lesions distal to the pulmonary capillary bed e.g:
  - MVD
  - Left sided failure
  - Pulmonary venoocclusive disease.

♥ Mediators of pulmonary hypertension are endothelin, angiotensin II and TA2, they act as vasoconstrictors and growth factors lead to cell proliferation, fibrosis and smooth muscle hypertrophy, this will lead to increase of the pulmonary vascular resistance.

## C/P:

- ① Cause.
- ② Rt V++, P++.
- ③ Rt sided failure.

## Investigations:

☉ Cause    ☉ Echo → P++, Rt V+    ☉ ECG → Rt V++ with or without strain.

## Treatment:

- ♥ Treatment of the cause.
- ♥ Treatment of right sided heart failure e.g with diuretics.



## Primary Pulmonary Hypertension

This is a rare condition (female > male) especially children and young adults.

### ↪ Aetiology:

It is a primary pulmonary arteriopathy with medial hypertrophy of pulmonary arterioles.

### ↪ C/P:

- ♥ Dyspnea, angina (right ventricular ischemia).
- ♥ Signs of P++.
- ♥ Right ventricular failure - Low COP.

### ↪ ttt:

- Vasodilators have little efficacy, Ca.Ch.blockers e.g diltiazem (120-900 mg/d) or nifedipine (30-240 mg/d) or Amlodipine (5-20 mg/d) can be used usually with the high doses. Recently endothelin receptor antagonists are available. Sildenafil (Viagra) can be used.
- Heart - lung transplantation should be considered early.

## Hyperlipidaemia

### Serum lipoprotein by electrophoresis

#### 1. Chylomicrons:

Which transport dietary (exogenous) triglycerides from intestine to tissues.

#### 2. VLDL:

Transports endogenous triglycerides from the liver to tissues. It also contains cholesterol.

#### 3. LDL:

Transports 60% of plasma cholesterol from liver to tissues.

#### 4. HDL:

Transports 40% of plasma cholesterol from tissues to liver.

### I. Classification of primary Hyperlipidaemia

#### ↪ Type 1 (↑ Chylomicrons) (Due to ↓ lipoprotein lipase)

- Creamy plasma - Eruptive xanthomas.
- High chylomicrons - High triglycerides → pancreatitis.
- TTT by fat free diet.

#### ↪ Type II (↑ LDL)

- A- with normal triglycerides
- B- with high triglycerides

- It is a familial hypercholesterolemia.
- Due to ↓↓ LDL receptors.
- Liable to IHD.
- Xanthelasma.



- **TTT** by reducing cholesterol rich diet & by drugs e.g. cholestyramine & HMG CoA reductase inhibitors (statins), HMG CoA i.e. (hydroxymethyl Glutaryl coenzyme A).

### ↪ **Type III (↑ cholesterol, ↑ Triglycerides, ↑ VLDL)**

- Hypercholesterolemia & triglyceridemia.
- Liable to IHD – Palmar xanthomas.
- **TTT** by ↓ cholesterol in diet & by fibrates.

### ↪ **Type IV**

- ↑ VLDL, ↑ triglycerides.      ➤ Liable to IHD & pancreatitis.
- Eruptive xanthoma.
- **TTT** ↓↓ fat, ↓CHO in diet & nicotonic acid.

### ↪ **Type V (↑ Chylomicrons, VLDL and Triglycerides)**

- It is a familial hypertriglyceridaemia.
- Plasma triglycerides ↑ → pancreatitis      ➤ Eruptive xanthoma.
- Associated with obesity.      ➤ Liable to pancreatitis.
- **TTT** by ↓fat, ↓CHO, weight reduction & nicotinic acid (triglycerides lowering agents).

## II. Causes of 2ry Hyperlipidaemia

- 1) DM → ↑ VLDL, IDL, triglycerides.
- 2) Alcohol excess → ↑ VLDL, triglycerides.
- 3) Chronic renal failure → ↑ VLDL, ↓ HDL, ↑ triglycerides.
- 4) Thiazides → ↑ VLDL, ↑ triglycerides.
- 5) Non selective BB → ↑ VLDL, ↑ triglycerides.
- 6) Hypothyroidism → LDL ↑, ↑ cholesterol.
- 7) Nephrotic \$ LDL ↑, ↑ cholesterol.

### **Drugs used in the management of hyperlipidaemia**

- ✓ **Fibrates** e.g. bezafibrate (Bezalip), fenofibrate (lipanthyl). Their action mainly is decrease of triglycerides and increase of HDL.
- ✓ **Statins** (HMG CoA reductase inhibitors) e.g simvastatin (Zocor), atorvastatin (lipitor). Their main action is lowering of LDL.
- ✓ **Resins**, they are bile acid sequestration drugs and can lower LDL.
- ✓ **Nicotinic acid**, it reduces triglycerides and increase HDL.
- ✓ **Propucol** reduces HDL.
- ✓ **Omega 3** reduces triglycerides.

#### **Doses:**

- ✓ Fibrates: Bezafibrate 200 mg tab/8hrs, fenofibrate 300 mg/d.
- ✓ Statins: Atorvastatin 10-80 mg/d, simvastatin 10-40 mg/d.



## **The lipid - Lowering diet.**

- ◆ **Reduce the total fat intake:** Dairy products and meat are the principal sources of saturated fat in diet.
- ◆ **Substitution with monounsaturates and polyunsaturates:** Monounsaturated oils e.g olive oil and polyunsaturated oils such as sunflower oil, corn oil should be used.
- ◆ **Reduce dietary cholesterol:** Liver should be avoided, eggs are rich in cholesterol but can still be a part of a balanced lipid lowering diet.
- ◆ Increase intake of dietary fibres.
- ◆ Reduce alcohol intake.
- ◆ Ideal body weight.

## **Atherosclerosis**

It is a progressive inflammatory disorder of the arterial wall characterized by formation of focal lipid rich deposit of atheroma → ischaemia.

### **↳ Pathogenesis** (sequences of events)

- 1)** Endothelial injury → adherence of blood macrophages or monocytes which imbibe LDL and actively enter the intima and become (foam cells).
- 2)** Active macrophages release free radical that oxidize the LDL, the oxidized LDL is toxic to endothelium causing endothelial loss and exposure of the subendothelial collagen → platelets aggregation.
- 3)** Platelets will release mitogenic factors → migration of smooth muscles into the intima.
- 4)** Also activated macrophage secretes cytokines e.g platelet growth factors, T.N.F, fibroblast growth factor and IL1.
- 5)** The smooth muscle cell & macrophages accumulate LDL from the plasma, this is enhanced by ↑ LDL in blood.

### **↳ Pathologic feature of atherosclerosis**

#### **A- THE FATTY STREAKS**

- Thin flat yellow streaks in the intima, they consists of macrophages and smooth muscles cells whose cytoplasm distended with lipids (foam cells).
- They are present very early in life, they ↑↑ in number until about age of 20 years and then remain static or decreased !?.

☞ There is controversy about whether some fatty streaks progress into fibrous atheromatous plaque or whether they are independent of atherosclerosis !?.





## B- FIBROUS ATHEROMATOUS PLAQUE

It consists of fibrous cap under the endothelium & lipid zone consists of lipid laden macrophages.

## C- COMPLICATED PLAQUE

Complicated plaque is calcified and fissured or ulcerated → rupture plaque → thrombosis.

### Risk factors of atherosclerosis & IHD.

Variable

- ☞ LDL: HDL > 5: 1.
- ☞ ↑↑ Level of HDL seem to be protective.
- ☞ HDL may allow elusion of cholesterol out of coronary vessels.
- ☞ Smoking – DM – Obesity – Diet – Stress – Haemostatic factors.

Fixed

- ☞ Age – Male sex – FH.

### Ch. Ch. of vulnerable plaques:

Lipid rich core - thin fibrous capsule with ↑ inflammatory cells → fissuring, rupture → thrombosis.

### Q Ch. Ch. of stable plaques:

Small lipid core, thick fibrous capsule, lipid lowering therapy help to stabilize vulnerable plaques.

## Atherosclerosis with chronic Peripheral Arterial Disease

Male > female, strongly associated with smoking and affects the leg more than the arm. Patients are usually over 50 years.

### Sympt:

- ☞ Intermittent claudication.
- ☞ Cold feet or legs.
- ☞ Peripheral cyanosis.
- ☞ Late, rest pain.

### Signs:

- ☞ Peripheral pulse is weak.
- ☞ Leg ulcers, absence of hair
- ☞ Finally frank gangrene.

### Investigations:

- ✓ X-ray → Calcification of arteries.
- ✓ Doppler and duplex imaging.
- ✓ Magnetic resonance angiography (MRA)..
- ✓ Angiography for revascularisation.

### ttt:

- ☞ Regular exercise e.g walking – cycling or swimming for 20 minutes 3 times/week have a protective effect due to:
  - ☞ ↑ HDL.
  - ☞ ↓ Blood pressure.



- ☛ ↓↓ Blood clotting
- ☛ Improve the collateral circulation.
- ☛ Medication has a little role.
- ☛ Balloon angioplasty for iliac or femoral stenosis.
- ☛ Calcium channel blockers.
- ☛ Amputation.

Management of atherosclerotic peripheral vascular disease	
G. measures	Indications of revascularization
<ul style="list-style-type: none"> <li>☛ Stop smoking.</li> <li>☛ Weight reduction.</li> <li>☛ Stop vasoconstrictors.</li> <li>☛ Control DM &amp; hypertension.</li> <li>☛ Exercise to improve the collaterals.</li> <li>☛ Avoid infection, trauma.</li> <li>☛ Vasodilators and anticoagulants are unhelpful.</li> </ul>	<ul style="list-style-type: none"> <li>☛ Chronic ischaemia with impaired skin and tissue viability.</li> <li>☛ Disabling symptoms due to severe stenosis.</li> </ul>

**N.B.:**

Atherosclerosis is also presented with coronary heart disease and cerebrovascular disease, (See their chapters).

## **Prevention of atherosclerosis**

### ***1. Primary:***

i.e. to prevent atherosclerosis in apparently healthy persons by:

- Regular exercise.
- No smoking.
- Fresh fruits and vegetables.
- Ideal body weight.
- Lipid lowering drugs.
- TTT of hypertension and DM.

### ***2. Secondary prevention:***

i.e.: to prevent or decrease the incidence of another vascular event in patients who already has evidence of atheromatous vascular disease by:

- Stop smoking.
- Control blood pressure.
- Lipid lowering agents.
- Aspirin.
- Ca.Ch.B and ACE inhibitors.



# Peripheral vascular disease

## **(I) Peripheral arterial disease:**

(a) Atherosclerotic chronic LL ischaemia (see before).

(b) Acute LL ischaemia:

**Causes:** Embolic or thrombotic disease

**C/P:**

- Pain, pallor, paraesthesia, paralysis and coldness of the affected limb.

- Pulses are diminished or absent.

**Investigation:** Doppler and duplex imaging – MRA.

**TTT:** Thrombolysis, embolectomy.

(c) Buerger's disease in heavy smokers..

(d) Aortic aneurysms (see before).

(e) Takayasu's disease (see rheumatology).

(f) Raynaud's phenomenon or disease:

Raynaud's phenomenon consists of spasm of the digital arteries precipitated by cold and relieved by heat. If there is no underlying cause it is called Raynaud's disease.

**C/P:**

- Vasoconstriction causes skin pallor, followed by cyanosis due to sluggish blood flow, then redness secondary to hyperaemia..

- The duration of the attacks is variable but they may last for hours.

- Numbness, burning sensation and severe pain occur as the fingers warm up.

**TTT:**

- Avoid cold – wearing gloves – stop smoking.

- Avoid BB – give Ca.Ch.B.

- Sympathectomy or prostacycline infusion for severe cases.

## **(II) Peripheral venous disease:**

(a) Varicose veins.

(b) DVT (see before).

(c) **Superficial thrombophlebitis:** Commonly involves saphenous veins, axillary veins may be involved. There is painful, tender, cord like structure with redness and swelling. It is treated by rest – elevation of the limb – NSAID – Anticoagulants are not necessary because embolization does not occur.



# Shock

## (Acute circulatory failure)

This term is used to describe acute circulatory failure with inadequate tissue perfusion resulting in generalized cellular hypoxia.

### ↳ Causes of shock:

#### ① Hypovolemic shock:

A condition causing major reduction of the blood volume e.g. internal or external hemorrhage, severe burns & dehydration (e.g. diabetic ketoacidosis)

#### ② Cardiogenic shock:

Any form of severe heart failure. e.g. extensive myocardial infarction & acute mitral regurgitation.

#### ③ Obstructive shock:

Obstruction of the blood outflow. e.g. massive pulmonary embolism, cardiac tamponade & tension pneumothorax.

#### ④ Neurogenic shock:

It is caused by major brain or spinal injury, producing disruption of brain stem & neurogenic vasomotor control.

It may be associated with neurogenic pulmonary edema.

#### ⑤ Anaphylactic shock:

Due to inappropriate vasodilatation triggered by an allergen e.g.: shellfish, drugs or bee sting.

#### ⑥ Septic shock / SIRS:

(Systemic inflammatory response syndrome):

Infection or any other causes of a systemic inflammatory response that produce widespread endothelial damage with vasodilatation, arterio-venous shunting, microvascular occlusion & tissue edema, resulting in multi-organ failure.

### ↳ Haemodynamic changes in shock:

#### ↳ Hypovolemic shock:

- Low central venous pressure (CVP).
- Low cardiac output (COP).
- Increase systemic vascular resistance.

#### ↳ Cardiogenic shock:

- CVP is high.
- COP is very low.
- Increase systemic vascular resistance.





### ③ *Obstructive shock:*

- A. Cardiac tamponade:
  - A. Pulsus paradoxus & distant heart sounds.
  - B. Kaussmaul's sign (increase jugular venous pressure on inspiration)
- B. Pulmonary embolism:
  - See chapter of pulmonary embolism

### ④ *Anaphylactic shock:*

- ❖ Signs of profound vasodilatation.
  - A. Warm extremities.
  - B. Low blood pressure.
- ❖ Erythema, urticaria, angio-edema.
- ❖ Bronchospasm, rhinitis.
- ❖ Edema of the face, pharynx and larynx.
- ❖ Nausea, vomiting, abdominal cramp.

### ⑤ *Septic shock:*

- ❖ Fever (hypothermia is unusual).
- ❖ Warm extremities, bounding pulse due to vasodilatation.
- ❖ Hypotension
- ❖ Occasionally signs of cutaneous vasoconstriction.
- ❖ Rapid capillary refilling.



## **Monitoring of patients in shock**

#### 1- Clinical indices of tissue perfusion:

- a. Pale cold skin, delayed capillary refilling & absence of visible veins in the hands & feet indicate poor perfusion.
- b. Urinary output is a sensitive indicator of renal perfusion & haemodynamic performance.

#### 2- Blood pressure.

#### 3- Central venous pressure (CVP).

#### 4- Wedged pulmonary artery pressure, by Swan-Ganz

catheter. It is inserted centrally or through the femoral vein or the antecubital vein → major veins → right atrium → right ventricle → pulmonary artery & into the wedge position.

### **Complications of Swan-Ganz catheter:**

- Arrhythmia.
- Valve trauma.
- Pulmonary infarction.
- Balloon rupture / leakage / embolism.
- Sepsis.
- Thrombosis, embolism.
- Pulmonary artery rupture.

#### 5- Cardiac output.



## Investigations:

- Complete blood picture.
- Coagulation profile.
- Blood gases, acid base state.
- Liver biochemistry.
- Echocardiography.
- Blood glucose.
- Blood culture.
- Kidney functions, electrolytes.
- FDPs.

## Treatment of shock:

Delay in making the diagnosis and in initiation of treatment and inadequate resuscitation leads to the development of multiple organ failure (MOF).

### (A) General measures:

- Patent airway.
- Oxygen therapy.
- Fluid therapy either:  
Crystalloid solution e.g saline or colloidal solutions e.g.  
Dextrans and hydroxyethyl starch  
(More increase in plasma volume)

The fluid therapy is given according to the need and according to (CVP, type of shock)

### (B) Specific measures: "according to the type of shock"

#### 1. Hypovolaemic shock:

- Control haemorrhage.
- Blood transfusion.

#### 2. Septic shock:

- Treatment of infection by antibiotic (IV)
- Surgical drainage for any collections elsewhere.

#### 3. Anaphylactic shock:

- Adrenaline IM.
- Antihistaminics.
- Hydrocortisone IV.

#### 4. Massive pulmonary embolism:

- Thrombolytic therapy.
- Anticoagulants.

#### 5. Cardiac Tamponade:

- Pericardiocentesis.

#### 6. Cardiogenic shock:

- Dopamine, Dobutamine.

### (C) Treatment of complications:

e.g. renal failure, coagulopathy and ARDS.

## **Cardiovascular involvement in systemic diseases**

- 1- SLE → pancarditis, RhD → myocarditis, pericarditis and AI.
- 2- Seronegative arthropathies. → AI.
- 3- Vasculitis → Coronary heart disease, hypertension .
- 4- Scleroderma → Cardiomyopathy, polymyositis. .
- 5- DM → Coronary heart disease.
- 6- Thyrotoxicosis → Tachy - arrhythmia.
- 7- Hypothyroidism → ↓ HR, heart block . and cardiomyopathy
- 8- Sarcoidosis, Amyloidosis and Haemochromatosis → Cardiomyopathy
- 9- Chronic renal failure → pericarditis, hypertension and arrhythmia.
- 10- Liver cell failure → hyperdynamic circulation .
- 11- Leukaemia , Lymphoma , polycythaemia, HIV, CMV, IMN and disseminated TB or malignancy can affect the heart (see chapters).

**Rheumatic fever is a systemic disease mainly affects the heart.**

### **Extracardiac manifestations (organs involvement) of cardiovascular diseases.**

- 1- Heart failure → see its complications
- 2- Systemic hypertension → see target organ damage .,
- 3- Rheumatic and congenital heart disease → pulm embolism or systemic embolization
- 4- AF → embolization .
- 5- Atherosclerosis → cerebral , renal, peripheral ischaemia (see before) .
- 6- Shock → multiorgan failure.
- 7- Dissecting aortic aneurysm → renal, peripheral ischaemia
- 8- Infective endocarditis → renal , cerebral , mesentric occlusion (see before).

### **Immune mediated cardiovascular diseases.**

- Rheumatic fever .
- Some manifestations of IE are immune mediated (see before).
- Graft rejection.
- Cardiac involvement in SLE, Rh D, Vasculitis (see rheumatology).

### **Cardiovascular emergencies**

- 1- Hypertensive emergencies see before .
- 2- Cardiogenic pulmonary edema .
- 3- Massive pulmonary embolism .
- 4- Paroxysmal arrhythmia.
- 5- Cardiac arrest .
- 6- Shock (acute circulatory failure)



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