○ Large arteries of aorta :- leading to ;

- Unequal radial pulse.
- Unequal blood pressure.
- Unilateral clubbing.
- ◆ Ischemic manifestations as; Pallor, pain, coldness, parasthesia.

Pressure on nerves :-

- Sympathetic chain :- Horner's syndrome (mention).
- ☐ Left recurrent laryngeal nerve :- Hoarseness of voice, stridor, bovine cough & paralysis of vocal cord.
- Vagus:- Arrhythmia & dyspeptic manifestations.
- → Phrenic nerve: Hiccup & Diaphragmatic paralysis.
- Spinal nerves: Brachial plexus → Brachial neuralgia, Intercostal nerves → Intercostal neuralgia.

Pressure on bones :-

- Sternum :- Sawing pain & Pathological fracture.
- Ribs: Pain, erosion & pathological fractures.
- Clavicle: Pain & pathological fractures.
- ⊃ Spine: Pressure necrosis & Collapse of bodies → pressure on nerves → Paraplegia.
- dorsal spine in children, denoting mediastinal mass as lymphadenopathy, Normally, it is not heard.

4. Systemic symptoms and syndromes :-

- Fever, anorexia, weight loss and other systemic manifestations.
- <u>Syndromes:</u> Cushing's, Hypoglycemia, Hypertension, Hypercalcaemia & Gynaecomastia.
- Thymoma: Myasthenia gravis, Red cell aplasia, Myocarditis, Hypo & hyperglobulinaemia.
- 5. Pneumo-mediastnum :- which is presented by;
 - <u>Pain:</u> (commonest); dull aching pain due to stretching of mediastinal structures & aggravated by breathing and changing position.
 - Dyspnea, dysphagia, dysphonia with characteristic hot potato voice.
 - Associated subcutaneous crepitations.
 - Mediastinal crunch .
 - <u>Acute mediastinitis:</u> sub-sternal pain, chills, high fever and prostration; they are treated by antibiotics and surgical drainage.



☆ Differential diagnosis of Mediastinal masses :-

Anterior compartment	Middle compartment	Posterior compartment
1. Thymus:-	1. Pericardium :-	1. Neural :-
- Hyperplasia.	- Cysts .	a. Peripheral nerves:
- Cyst.	- Diverticulae .	- Neurofibroma.
-Thymoma:- benign or malignant.	2. Vascular :-	- Malignant tumor.
2. Germ cell tumors :-	- Aneurysm of arch.	b. Autonomic nerves:
- Benign cyst (dermoid).	- Anomalous vessels.	- Ganglioneuroma.
- Malignant :-	3. Heart :-	- Neuroblastoma.
* Seminoma.	- Lt vent. aneurysm.	c. Paraganglioma.
* Non-seminoma.	- Cardiomeyally.	d. Meningeocele.
	4. Trachea and main bronchi :-	2. Oesophageal masses.
	- Bronchogenic cysts .	3. Diaphragm :-
3. Hodgkin's lymphoma.		- Hernia.
4. Thyroid:-		- Tumors.
- Retrosternal goiter.		4. Aortic aneurysm.
- Entopic thyroid.		
5. Parathyroid adenoma	radio to	
6. Morgagni's hernia	Company Mannager Mannager (1997) and the company of	

* Masses may be found in more than one mediastinal compartment :-

1. Lymph node masses :-

- Lymphoma :- Hodgkin's or non-Hodgkin's lymphoma .
- Metastasis.
- · Sarcoidosis.
- TB.
- Castleman's giant follicular hyperplasia.
- 2. Reduplication cysts: Bronchial & Gastroenteric.
- 3. Vascular: Aneurysm of the aorta and its branches.
- 4. Connective tissue :- Lipomatosis & Malignant fibrous histiocytoma.
- N.B.: The commonest mediastinal mass is L.N., due to; T.B., bronchial cancer, pneumoconiosis, leukaemia, lymphoma & Sarcoidosis.

☆ Investigations of a case with mediastinal mass:-

A) Imaging techniques:-

- <u>Schest X-ray:</u> (postero-anterior and lateral).
- © Computed tomography (CT).
- ∽MRI.
- **Radio-active iodine studies :- in case of thyroid swelling .
- S Contrast studies: Ba Oesophogography, Angiography, Selective angiography & Myelography.

B) Biopsy techniques:-

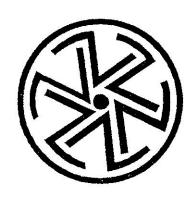
- Trans tracheal and trans-bronchial aspiration biopsy.
- Percutaneous aspiration guided by C.T or ultrasonography.
- Mediastinoscopy and anterior mediastinotomy.

C) General approach to diagnostic investigation of mediastinal masses :-

- Most mediastinal lesions require tissue diagnosis e.g.;
 - ⇒ Thyroid swelling; cleared by 1131 scan.
 - ⇒ Granuloma; it is densely calcified.
 - ⇒ Mediastinal lipomatosis; diagnosed with C.T..
 - ⇒ Lymphoma or other malignancies; diagnosed by lymph node biopsy.
 - ⇒ Pulmonary TB and sarcoidosis; appropriate tissue biopsy.

A Treatment of mediastinal masses:-

- All mediastinal masses either benign or malignant must be surgically removed, as;
 - They may enlarge and compress mediastinal structures.
 - Bleeding, rupture or infection.
 - Malignant degeneration.
- ☆ C.T or MRI must be dune for pre- and post-operative evaluation.





PULMONARY EMBOLISM

A Definition of Pulmonary embolism:-

• The pulmonary arteries become occluded by thrombus or other obstructive materials .

N.B.: - Venous thromboembolism (VTE) includes DVT & pulmonary embolism.

* Major risk Factors for venous thromboembolism :- [60]

- Goperative: abdominal, pelvic, bone surgery, Pregnancy and/or postpartum.
- Goncology: cancer pancreas, stomach, lung lymphoma and leukaemia.
- ☞ Old age.
- GOral contraceptive pills.
- @ Obesity .
- *Gothers*:- [2 C]
 - → Congestive heart failure and post-myocardial infarction.
 - - Deficiency of anti-thrombin III, protein C and protein S.
 - Defective plasminogen . (or release of plasminogen activator) .
 - Increased circulating coagulation protein factors V & VII.
 - Hyper-reactive platelets ...

* Predisposing factors to venous thrombosis (Virchow's triad):-

- Venous stasis: CHF, Prolonged bed rest, Post-operative & V.V..
- Hyper-coagulability:- Dehydration, Polycythemia, After major operations or MI.
- Endothelial injury :- Thrombo-phlebitis, Trauma & Infusion with concentrated glucose

☆ Sources of the emboli:-

Peripheral veins :- as ;

- → Thrombi of deep veins: ilio-femoral or calf vein DVT (>90%).
- → Fat Embolism :- after fracture of long bones .
- → Air embolism :- after air insufflation or neck surgery .
- Amniotic fluid embolism :- in pre-mature separation of placenta.
- → Malignant emboli :- as from renal carcinoma.
- → Parasitic emboli :- as Ascaris.

Right & Left side of the heart :- as in ;

- → Thrombi:- either on:-
 - Rt side :- RA thrombi (HF, AF) & RV thrombi (Septal infarction).
 - + Lt side :- Paradoxical emboli → pass through defect as ASD or VSD to Rt side of heart.
- Tumours:- Rt or Lt atrial myxoma.
- → Vegetations: in case of Infective endocarditis.



* Patho-physiological consequences of Pulmonary embolism: depends on;

- Degree of pulmonary arterial obstruction .
- Pre-existing cardio-pulmonary Status.
- Secondary effects e.g. local release of neuro-humoral substances.

* Patho-physiological changes in Pulmonary embolism :-

Circulatory changes :-

1. Haemodynamic changes:-

- Small and few emboli → no haemodynamic effects.
- * Large emboli or a multiple small emboli → Systemic hypotension & Decrease in COP
- 2. Ventilation perfusion imbalances.
- 3. Increased dead space ventilation.
- 4. Increase in alveolar ventilation.
- 5. Anatomic venous admixture: due to opening of intra-pulmonary A-V shunts.
- 6. Systemic arterial hypoxaemia: due to Venous admixture & Patent foramen ovale.
- Hypocapnea: due to Hyperventilation.
- Widening of alveolar arterial difference in PO2.

A Categories of Pulmonary embolism (PE):-

- Acute massive PE:- large embolus lodge in the center of the pulmonary arterial tree.
- Sub-massive PE:- without pulmonary infarction [the most common type]
- PE associated with pulmonary infarction.
- Chronic thrombo-embolic disease
 → severe pulmonary hypertension.

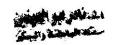
* Clinical manifestation of DVT:-

- Unilateral swelling with duskiness of the leg.
- Pain over calf muscles on dorsiflexion (Homan's sign).

* Clinical picture of PTE: there are many forms of manifestations;

A) Asymptomatic: due to;

- Small emboli.
- Fibrinolysis in pulmonary circulation.
- Double blood supply of the lung.



B) Sudden death: dt massive embolism (> 85 % of pulmonary vascular bed is obliterated).

C.) Acute Cor-pulmonale (Large embolus):- (> 50 % is occluded), presented by;

- ◆ Sudden sever retro-sternal pain :- due to ;
 - ✓ Mechanical distention of pulmonary artery .
 - ✓ Reflex coronary spasm.
 - ✓ Low COP (↓ pulmonary BF → ↓ coronary BF).
 - ✓ High RA pressure (2^{ry} to high pulmonary BP) → resist emptying of sinus venosus → \downarrow coronary blood flow.
- ◆ Shock & Syncope may occur rapidly due to marked ↓ in COP, ↓ BP & rapid weak pulse.
- Dyspnea .
- * Central cyanosis :- due to ;
 - ✓ Opening of pulmonary A-V shunt secondary to sudden ↑ in pulmonary BP.
 - ✓ Non-uniform constriction of distal arterial ways → under-ventilation of lung units with respect to their perfusion.
 - ✓ Lack of surfactant by non perfused lung.
- * RSHF manifestations: except oedema L.L. as the period is too short for it to develop.

D) Pulmonary infarction: (Medium sized embolus), presented by;

- In first day: Dyspnea, Pleural rub & Pleural pain (DRP).
- In second day: Cough, fever, Haemoptysis, haemolytic anaemia & haemorrhagic pleural effusion (blood).
- Signs of pulmonary consolidation.

E) If Small embolus but repeated:-

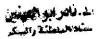
- It leads to gradual obliteration of pulmonary vascular bed → Pulmonary HTN & chronic cor-pulmonale.
- F) If infected embolus: leads to → pneumonia & lung abscess.

G) General Collective signs of PE:-

- ☆ Tachypnea (RR > 20 / min)
- ☆ Tachycardia (pulse > 100 / min)
- ☆ Blood pressure:- there may be shock state.
- ☆ Elevated jugular venous pulse.
- ☆ Right ventricular gallop or murmur.

- ☆ Rales (crackles).
- ☆ Accessory muscle use .
- ☆ Pleural friction rub.
- ☆ Central cyanosis
- ☆ Signs of pleural effusion.

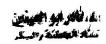
N.B. Unexplained dyspnea associated with anxiety, tachypnea & tachycardia which persist during sleep should arouse suspicion of Pulmonary Embolism.



☆ Investigations of Pulmonary Embolism :-

A) Basic tests :-

- 1) Chest X-ray: show the following findings:
 - Normal chest radiography.
 - Elevated cupola of diaphragm on affected side .
 - Evidence of pleural reaction or effusion .
 - Parenchymal infiltrates :
 - Fleischner's hump: pleural-based wedge shaped parenchymal lesion → Pulmonary infarction.
 - Fleischner's lines: atelectatic bands at lung base.
 - * Westemark's sign: unilateral increased density due to hypoperfusion of one lung.
 - In case of acute cor-pulmonale :- there will be;
 - ✓ Signs of pulmonary HTN :- e.g. Prominent central pulmonary artery & unequal size of pulmonary arteries .
 - ✓ Signs of RAH & RVH .
- 2) ECG: It is IMP to exclude MI as a cause of dyspnea & chest pain, it shows:
 - Sinus tachycardia:- It is the most common pattern.
 - * Atrial flutter.
 - Classic pattern S₁Q₃T₃ is only present in 10-15 % of pulmonary embolism.
 - Rt axis deviation & Rt ventricular enlargement.
 - T wave inversion in right chest leads may be present in severe cases.
 - Prolongation of QRS complexes due to recent Rt BBB.
- 3) Arterial blood gases: (supportive for diagnosis of PE)
 - It shows: Hypoxaemia & Hypocapnia.
 - Normal ABGs do not exclude PE3.
 - A normal alveolar arterial PO2 difference is more specific evidence against PE.
 - Finding of unexplained hypoxaemia should raise the possibility of PE.
- 4) Catheterization: it shows 1 in RV & pulmonary artery pressure.
- 5) Laboratory data: they are not specific;
 - ↑ LDH, ↑ serum bilirubin & normal SGOT → Pulmonary infarction.
 - ↑ LDH, Normal CPK & Normal HBD → to exclude MI.
 - ullet 1 level of fibrinogen degradation products after pulmonary embolism .



B) Lung imaging:

- 1) Helical (spiral) CT:- It has the following advantages in PE;
 - Rapid and non-invasive.
 - Fewer potential complications.
 - Sensitivity 95% and specificity 97%.
 - Diagnosis of diseases other than PE.
 - Less cost and time than lung scan or conventional pulmonary angiography...

2) Ventilation / perfusion lung scan (V/Q scan):- it involves 2 scans;

Pulmonary perfusion scan: -

- It uses ⁹⁹Tc & Macro-aggregated albumin (MAA).
- It reveals areas of hypo-perfusion (cold areas).
- ◆ It is highly sensitive, but it is not specific as it is +ve in pneumonia, atelectasis & malignancy.

Dentilation scan :-

- It uses Xenon (¹³³X) inhalation.
- It reveals areas of hypo-perfusion (Cold areas).

N.B. :- If cold areas of perfusion scan concise with that of ventilation scan \rightarrow Pulmonary embolism.

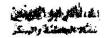
Dundamental facts underlying the use of V/Q scan:

- It helps in identification of high risk patients.
- I ventilation & I perfusion (matched defect) → parenchymatous lung disorder (pulmonary embolism or infarction).
- 1 ventilation & normal perfusion (reversed mismatch) → airway obstruction or atelectasis.

3) Pulmonary angiography:-

- It is the most accurate diagnostic study for evaluating pulmonary embolism.
- Disadvantages of pulmonary angiography:-
 - ✓ Invasive and expensive .
 - ✓ Technical complexity.
 - ✓ Limited availability.
 - ✓ Mortality rate of about 1%.
- Pulmonary angiographic findings in acute PE:-
 - ✓ Primary findings: Abrupt cut-off of the vessels & Intraluminal filling detect.
 - ✓ Secondary findings: Absent, decreased or delayed filling of lung zone.

4) MRI.



C) Leg imaging:

- Ultrasonography (colored Doppler).
- MRI venography.
- Ascending contrast venography (not preferred).

D) Echocardiography:-

- It will show Right ventricular enlargement.
- For evaluation of severity of core pulmonale and follows up of improvement.

☆ Diffrential diagnosis of PE:- from;

- Other causes of acute chest pain .
- Other causes of Haemoptysis.
- Other causes of HF.
- Other causes of acute cor-pulmonale.
- Lung absess .

* Management of Pulmonary embolism :-

1. Identification of the high risk patients .

2. Prophylaxis to prevent DVT.

3. Diagnostic approach for patients with suspected DVT.

4. Treatment of acute PE:-

A) Prophylaxis against PE:-

- ◆ Non Pharmacologic :-
 - ✓ Graded compression stockings (TED stockings).
 - ✓ Intermittent pneumatic compression boots.
 - ✓ Vena cava filters (e.g. Greenfield filter).
- Pharmacologic agents:-
 - ✓ Low molecular weight heparin.
 - ✓ Mini-dose warfarin.
 - ✓ Antiplatelet (aspirin).

B) Treatment of diagnosed acute pulmonary embolism:-

1) Heparin:-

- Loading dose 100 lU / kg given as an intravenous bolus.
- Maintenance dose 500-600 IU / kg (total daily dose of 30.000 IU for a 60kg patient)
- Adjust infusion rate until the APPT = 1.5-2.5 that of the control (45-75 sec).
- ◆ It prevents extension & recurrence of thrombo-emboli & enhances fibrinolytic dissolution

2) Oral anticoagulants "warfarin":-

Adjust the dose international normalized ratio (INR) to be [] 2 & 3 that of the control.

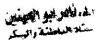
3) Intravenous thrombolysis with streptokinase or urokinase:-

- Its use carries high risks and should be used only when indicated.
- Bolus (250000 U IV) followed by infusion of (100000 U/h) for 12-24 h.
- * Indications :-
 - ✓ Life threatening PE especially in presence of haemodynamic instability (shock)
 - ✓ Evidence of right heart dilatation on ECHO.
 - ✓ Deterioration symptoms and signs in spite of heparin therapy.

4) Additional drug therapy: - according to the condition;

- Anti-shock measures: with vaso-pressors to support circulation.
- Treatment of pain: by analgesics as Pethidine (avoid morphine → suppress RC)
- Treatment of dyspnea :- Oxygen & respiratory stimulators as Coramine .
- Treatment of HF: IV Digitalis & Aminophyline infusion (avoid IV fluid → ↑ HF)
- Atropine :- Vagal reflexes → ↑ coronary & pulmonary artery spasm.
- Anti-biotics :- for infection .
- Treatment of the causes & associated conditions.





C) Surgical treatment:-

◆ It has the following indications :-

1- Documented iliofemoral vein thrombosis, with;

- ✓ NO improvement after 2 hours of medical treatment.
- ✓ Contraindications to anticoagulants .
- ✓ Documented PE during full coagulation .
- ✓ Free floating thrombus.
- ✓ High-risk for fatal PE.

2- No iliofemoral vein thrombosis, but;

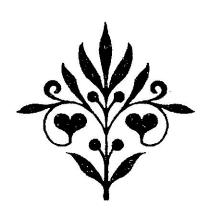
- ✓ Long-term prophylaxis is necessary (e.g. paraplegia).
- ✓ High-risk for both PE and haemorrhage .

Methods:-

- ✓ Pulmonary embolectomy (Trendlenberg's operation) using cardiopulmonary bypass.
- ✓ Insertion of a suction catheter (Green field catheter) through femoral vein & used to remove the thrombus .

D) Prevention of further emboli:-

- Anticoagulants (see before:).
- IVC interruption :- either;
 - ✓ Surgically:- by tent or occlusion.
 - ✓ Non surgically:- by introduction of a ballon device attached to catheter into IVC through neck or upper limb.



PULMONARY HYPERTENSION

** Definition :- Systolic pulmonary arterial pressure > 30 & mean pulmonary artery pressure > 20mmHg

N.B. normal pulmonary artery pressure = 25/10 with mean 15 mmHg.

* Mechanisms and types :-

- Pulmonary blood pressure = pulmonary flow & pulmonary vascular resistance
- Pulmonary hypertension occurs when there is increase in flow and / or resistance.
- There are 3 mechanisms for pulmonary hypertension:-

A) Increased pulmonary blood flow: as in;

- Left to right shunt:- hyperdynamic pulmonary hypertension.
- Left sided heart failure :- passive pulmonary hypertension.

B) Increased pulmonary Vascular resistance:-

- Hypoxaemia: vasoconstrictive (passive & hyperdynamic) pulmonary HTN.
- Intravascular thrombosis & Embolism :- obstructive pulmonary hypertension.
- Pulmonary fibrosis, syphilis & Vasculitis: obliterative (Chronic) pulmonary HTN.

C) Idiopathic pulmonary Hypertension.

A Causes of pulmonary hypertensive diseases:-

A) Primary pulmonary hypertension:-

- * Arteriolar (Precapillary):- Dietary & familial primary pulmonary hypertension.
- Venous:- Pulmonary veno-occlusive disease.
- Capillary: Pulmonary capillary haemangiomatosis.

B) Secondary pulmonary hypertension:-

- * Pulmonary causes :-
 - ✓ Airway diseases :- COPD .
 - ✓ Parechymatous diseases: Interstitial pulmonary fibrosis & collagen diseases.
 - ✓ Vascular diseases: Pulmonary embolism & Bilharziases.
 - ✓ Chest wail diseases: Kyphoscoliosis & obesity.

◆ Cardiac diseases :-

- ✓ VSD ASD PDA → Hyperdynamic pulmonary HTN.
- \checkmark Mitral and aortic valve diseases \rightarrow Passive pulmonary HTN .
- ✓ Left ventricular failure .
- ✓ Cardiomyopathy .
- ✓ Left atrial myxoma.

* Clinical manifestations :-

≥ Symptoms :-

- * Asymptomatic until sever.
- * Symptoms due to underlying pulmonary or cardiac disease .
- Dyspnea on exertion is the most common symptom of pulmonary hypertension .
- * Chest pain :-
 - ✓ Angina like chest pain in patients with severe pulmonary hypertension .
 - ✓ It is due to right ventricular overload and myocardial ischemia.
- * Syncope :- dt fixed low COP & vagal stimulation (associated with bradycardia)
- *Hoarseness of voice: dt pressure of Lt recurrent laryngeal n. by dilated Lt pulmonary A.
- * Haemoptysis: 2^{ry} to pulmonary venous congestion (e.g. mitral stenosis).

Physical examination :-

- In mild to moderate pulmonary hypertension → no signs.
- Signs of the causes: mention examples.
- Neck veins: Prominent or giant A wave.
- Signs of RVF.
- * Cardiac signs of pulmonary HTN:-
 - ✓ Inspection & palpation :- (Precordial examination)
 - ⇒ RV apex → Heaving . . .
 - **⇒** Signs of pulmonary artery dilatation .
 - Pulsation in pulmonary area .
 - ⊃ Signs of right ventricular hypertrophy .
 - ➡ Diastolic shock over the second left space.

✓ Percussion :-

Dullness on pulmonary area .

✓ Auscultation :-

- Wide splited second sound with accentuated pulmonary component.
- ➡ Ejection systolic murmur due to relative pulmonary stenosis .
- ⊃ Early diastolic (Graham-Steell) murmur is heard [associated PR] .
- ⊃ Pulmonary systolic ejection click .
- Over tricuspid area :- we can hear;
 - ⇒ Pan-systolic murmur (relative TR).
 - ⇒ Pre-systolic gallop (Forth HS).

- * Complication of Pulmonary HTN:- as those of RSHF (Mention).
- ★ Investigations: (they are mentioned in details from lesson of → MS)
 - 1- Electrocardiogram.
 - 2- Chest x-ray.
 - 3- Echocardiography.
 - 4- Cardiac catheterization.

*Treatment of Pulmonary HTN:- It aims to :-

- 1- Treatment of the cause.
- 2- Treatment of the heart failure.
- 3- Decreasing the workload of the right ventricle.
- 4- \(\text{Pulmonary HTN} :- \text{ by using the following medications (ANPIR);} \)
 - ACEIS.
 - Nifedipene & Amlodepine → CCBs.
 - ⇒ Prostacycline .
 - ⊃ Isoprenaline (β-agonist).
 - Regetine (α-blocker)
- 5- Improve life style of the patient; through the following:-
 - → Pulmonary vasodilators: Oxygen therapy (most effective).
 - Inhaled nitric oxide.
 - Diuretics.
 - ⊃ Anti coagulant :- in primary pulmonary hypertension .
 - ightharpoonup Phlebotomy:- when the HCV > 55 to 60 %.
- 6- If the cause is established: No treatment but, Lung with or without heart transplantation.
- N.B.: If the question says → Discuss pulmonary HTN: you should add bilharzial cor-pulmonale on the lesson of pulmonary HTN.



COR-PULMONALE

★ Definition: It is right ventricular enlargement with or without failure resulting from pulmonary diseases after exclusion of LSHF & congenital heart diseases.

* Causes: The same causes of pulmonary hypertension except the cardiac causes (Mention)

* Types:- It is classified according to the onset of its presentation into:-

A) Acute cor-pulmonale :- due to;

- Acute massive pulmonary embolism .
- Tension pneumothorax.
- Acute massive collapse.

B) Subacute cor-pulmonale: due to;

- Lymphangitis carcinomatosis (intrapulmonary metastasis).
- Recurrent minor pulmonary embolism.

C) Chronic cor-pulmonale: due to;

- COPD (most common cause especially blue bloater type).
- IPF.
- Thoracic wall deformities (Kyphoscoliosis).
- Pickwickian syndrome (obesity hypoventilation syndrome).

* Pathogenesis of cor pulmonale :-

- In acute rise of pulmonary pressure, right ventricle 1 its COP until pressure in Pulmonary artery reach 50 mmHg then the ventricle fails.
- 🖎 In chronic rise of pulmonary pressure, the RV can tolerate higher pressure before failure.

A Clinical picture of cor-pulmonale:-

- Anorexia 🖎
- > Discomfort in the right upper quadrant of the abdomen due to hepatic engorgement.
- Example 20 Congested pulsating neck veins with prominent (V) wave appears in the jugular pulse.
- 🖎 Pedal oedema.
- > The liver often shows expansile pulsations which are synchronous with the heart beats.
- > Hydrothorax and ascots are uncommon, even after RVF has occurred.
- A palpable cardiac impulse near the left sternal border and in the epigastrium.
- > Tricuspid insufficiency manifested by a pan-systolic murmur,etc.
- Arrhythmias: precipitating by Anxiety, Bronchodilators, † sympathetic activity, Hypokalaemia & Respiratory failure with or without disturbances in acid-base balance

* Diagnosis of cor-pulmonale :- It requires the following;

- 🖎 Diagnosis of pulmonary HTN by the characteristic signs, symptoms and investigations.
- Exclusion of LSHF and congenital heart diseases.
- 🖎 Diagnosis of the cause of cor-pulmonale.

☆ Prognosis :-

It is much better in patients with chronic bronchitis and emphysema in who blood gases can be maintained at near-normal levels.

A Differences between Bilharzial and hypoxic cor-pulmonale:-

Parameter	Bilharzial cor pulmonale	Hypoxic cor pulmonale
• Aetiology of P.HTN :-	bilharzial GranulomaEnd Arthritis bitterns	[*] 2 ^{ry} to hypoxic V.C. of pulmonary blood vessels.
Symptoms :-	• Low COP.	Hypoxia & hyper apnea .
◆ Complexion :-	• Pale .	Cyanosis .
◆ Pulse :-	Thread .	• Pounding .
◆ Hand :-	• Cold .	• Worm .
• Spleen :-	- Enlarged .	Normal or associated enlargement.
◆ Manifestations of P.HTN:-	• Evident .	 Masked by emphysema.
◆ Chest shape :-	• Normal .	Barrel shaped .

☆ Treatment of cor-pulmonale:-

- makes Treatment of the cause and the underlying pulmonary disorder.
- ≥ Oxygenation & Oxygen therapy ⇒ long term oxygen therapy (LTOT) in COPD.
- > Treatment for infection .
- 🖎 Bed rest.
- ≥ the workload of the right ventricle by decreasing pulmonary arterial pressure :-
 - + Pulmonary vasodilators :-
 - ✓ Calcium tumor blockers.
 - ✓ Prostacycline → powerful VD, short half-life (continuous infusion in acute cor pulmonale)
 - Nitric oxide: currently inhaled nitric oxide is used in ARDS.
 - Other measures :- carbonic anhydrate inhibitors
 - ✓ It is used to correct the alkalaemia induced by excessive diuresis .
 - ✓ Side effects: metabolic alkalosis.
- Diuretics.
- > Phlebotomy: standard treatment for the polycythemia if HCV > 55 to 60 %.
- Anti-coagulants (Warfarin is the drug of choice):- used in;
 - Management of primary pulmonary hypertension.
 - Low COP to 1 incidence of venous thrombosis caused by venous stasis.
- > Lung transplantation: in case of primary pulmonary hypertension.

PERICARDIUM

Acute Dry Pericarditis

Aetiology:- (PIRD)

- = 3 P:- Post traumatic, Post-cardiotomy & Post-commisurotomy.
- G 6 I: Idiopathic, Infection (viral → Coxaki, bacterial → TB), Infiltration, Irradiation, Infraction (transmural, Dressler's syndrome) & Iatrogenic.
- 3 R:- Rheumatic fever, Rheumatoid arthritis & Renal failure (Painless & terminal).
- CD:- Collagen diseases (SLE) & Drugs (Procainomid, Menoxidile, INH,).

☆ Pathology :-

Fine fibrinous adhesion [] 2 layers of pericardium → rub lay frication with lung motility.

☆ Clinical Presentation:-

A) Symptoms:-

- ★ General constitutional symptoms:- fever,
- * Chest pain: Retrosternal, radiate to shoulder & neck, Stitching, 1 by sitting & leaning forward, 1 with movement, lying down & respiration because pericardium is attached to central tendon of diaphragm.

B) Signs:-

- ★ General:- fever.
- * Local Cardiac signs :-
 - ✓ Pain: ↑ by pressure with stethoscope (lower part of external surface of parietal pericardium is the only pain sensitive part of it).
 - ✓ Pericardial rub: to and fro (pathgnomonic), lathery sound, no fixed relation to HS, ↑ by pressure with stethoscope & not disappear with holding respiration (DD from pleural rub which disappear).

☆ Investigation :-

- ☞ ↑ ESR.
- Leucocytosis.
- FCG: universal (in all leads) elevated concave ST segment upwards & inverted T-wave.

☆ Treatment :-

Analgesic.

Antibiotic.

Treatment of cause.

Follow up: for fear pericardial effusion.

Pericardial Effusion

Aetiology:-

A) On top of pericarditis: The effusion may be;

- Exudate: the same causes of dry pericarditis.
- Purulent:- pyogenic organisms:
- Haemorrhagic: TB, malignancy & infraction.

B) Effusion without pericarditis:- It maybe;

- Transudate: due to generalized oedema with;
 - High venous pressure in systemic congestion.
 - Hypoproteinaemia (NS or liver cirrhoses).
 - Myxedema.
- Exudate: which is either;
 - Heamo-pericardium :- due to rupture of aorta, heart or coronary.
 - Chylous effusion (lymph):-
 - ✓ Due to thoracic duct obstruction or injury .

✓ Characters:-

- ⇒ Milky white in colour .
- ⇒ Rich in fat so cleared by addition of ether.
- ⇒ Stained orange by sudden III .

☆ Haemodynamics:-

☐ In diastole:-

- It interferes with diastolic expansion, specially if rate of accumulation is rapid → systemic congestion & some pulmonary congestion due to obstruction of pulmonary veins → Low COP.
- It affects Rt > Lt side due to high pressure on Lt side.
- It affects veins > arteries due to thick wall of arteries.



A Clinical Presentation :-

Symptoms Symptoms

- 1- Systemic congestion symptoms (mention).
- 2- Dyspnea: due to lung congestion & mechanical pressure.
- 3- Low COP symptoms.
- 4- Pressure symptoms.
- 5- Pain (sense of heaviness):-
 - Due to stretch of infero-lateral parts of parietal pericardium & referred to shoulder by phrenic nerve.
 - It is due to separation of the 2 layers of pericardium.

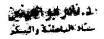
🗷 Signs

A) General signs :-

- ★ Decubitus: Prayer sign → lean forwards.
- * Low COP signs :- mention.
- ★ Systemic congestion signs :- e.g.
 - Congested neck veins: early (pulsating), late (non pulsating).
 - Kaussmaul's sign: Inspiratory filling of neck veins.
 - Fredrich's sign :- Rapid deep Y-wave of neck veins .
- * Pulse: pulsus paradoxicus.
- * Ewart sign: dullness or bronchial breathing on left lung base due to collapse of left lobe of lung by pericardial effusion.

B) Local cardiac signs :-

- * Invisible, impalpable apex.
- * Dullness outside apex.
- ★ Dullness over effusion.
- * Dullness in 2nd left space.
- * Shifting dullness: done over the heard during & before inspiration.
- ★ Wide bare area.
- ★ Distant weak heart sounds.



☆ Complications:-

- 1- Constrictive pericarditis.
- 2- Acute cardiac tamponade :- due to rapid rate of accumulation (discussed later).

☆ Investigations :-

1-X-ray:-

- Enlarged cardiac shadow .
- Change of cardiac size from day to night.
- Flask shaped (obtuse) or onion shaped (acute) heart.
- Change shape from position to other.
- Short broad supra-cardiac shadow.
- Obtuse cardiophernic angle → Rotché sign.
- Double contour :- heart & effusion.
- Stenciled border :- no separation [] chambers & borders.

2- ECG :-

- Low voltage (amplitude).
- Changes in ST segment & T-wave ⇒ Electerical alternance.
- 3-ECHO:- Free space [] myocardium & pericardium (effusion), because ECHO is reflexed on tissues, except in fluid → passes through it.

4- Cardiac Catheterization:-

- Early high pressure in right side (& even in Lt side).
- Pressure of space outside tip of catheter.
- 5-Angiography:- There is shadow outside opacified heart due to dye.
- 6- Aspiration: from bare area & out side apex, left sub-scapsular area (with +ve Ewart sign) & left side of subcostal angle.

7- Fluoroscopy (screen):-[Continuous X-ray]

Wide shallow without or with \$\diam\text{ pulsations of shadow border}\$; Normally pulsations of heart appear in fluoroscopy.

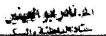
Differential Diagnosis :-

- 1- Constrictive Pericarditis.
- 2- Other causes of Low COP.
- 3- DD of aetiology.
- 4- Other causes of systemic congestion :- e.g. Cardiomegally (due to HF);

Parameter	Pericardial effusion	Cardiomegally
☞ Apex	✓ In-palpable, invisible:	✓ Palpable , visible
	✓ Dullness outside apex & Shifting dullness	✓ Not
 Auscultation 	✓ Weak, distant HS	✓ Gallop
Investigations:-		
• ECG :-	✓ as before.	✓ high voltage .
◆ X-ray :-	✓ as before	✓ Not
◆ ECHO :-	✓ as before .	✓ Not
Catheterization :-	✓ as before.	✓ Not
• Fluroscopy :-	✓ as before	✓ Not

5- From T.B. & Rheumatic fever :-

Parameter	TB pericarditis	Rheumatic pericarditis
★ Onset :-	◆ Gradual → chronic	• Acute .
★ Toxaemia :-	Moderate .	• Marked .
★ Degree :-	Marked effusion .	Moderate effusion
★ Valvular lesion :-	◆ No	◆ +ve .
★ Other C/P :-	Other TB manifestations.	• other rheumatic or
	, .	valvular C/P .
★ Fate :-	Constrictive Pericarditis	• Not.
★ Treatment :-	• Anti TB, steroid (if affect	Salicylate, Steroid but
	serous membrane).	not anti TB.
★ Tuberculin :-	• +ve .	◆ -ve



* Treatment of pericardial effusion :-

- 1- Treatment of cause.
- 2- Aspiration:-
 - * Types :- It is either;
 - Diagnostic :- show for criteria of each type .
 - Therapeutic: to relieve tamponade.

- It is done under screen to avid touching myocardium → AF & to avoid entrance
 of lung air to pericardium → hydro-pneumo-pericardium.
- It is done at sites:- previously mentioned...
- 3- If recurrent :- we do Pleuro-pericardial window;
 - > Fluid is drained in Mediastinum or pleura so; fluid spread over large surface area
 - → absorbed by lymphatics.

CARONIC PAROCURAGE

- 🗅 Definition :-
 - Rapid (acute) accumulation of 300 c.c. fluid in pericardium.
 - Accumulation of 1 L in any duration (Sub-acute).
- → Causes :-
 - Trauma → Cardiac perforation
 - T.B.
 - All acute causes of pericardial effusion.
- ⊃ უypes :-
 - A) Acute:- It is diagnosed by [Boeck's triade]; which is composed of;
 - Rising venous pressure → congested neck veins.
 - Falling arterial blood pressure.
 - Small quiet heart → No auscultated heart sound.
 - B) Sub-Acute: Pericardial Effusion.
- C/P, Investigations & D.D.: as Pericardial effusion.
- ⊃ Treatment :-
 - Treatment of HF.
 - Aspiration :- Explain .

Constrictive Pericarditis

[Pick's Disease]

* Aetiology:- Idiopathic, Infection (TB) & ?On top of Pericardial Effusion.

* Pathology & Clinical presentation :-

A) Symptoms:-

- Fibrosis [] layers of pericardium → pressure on right & left sides → interfere with
 diastolic expansion → systemic congestion & Low COP, <u>BUT</u>; heart is not
 enlarged so, no pressure symptoms are seen & patient can lie flat.
- Lung oligaemia → lung congestion → dyspnea which 1 by conversion of pericardial effusion to constrictive pericarditis by compression on pulmonary artery.

B) Signs:-(4 No, sign, sound)

- No dullness out side apex & No shifting dullness.
- No Ewart sign & No prayer decubitus.
- Freidrich's sign :- more apparent .
- Pericardial chock :-
 - \checkmark It is Diastolic sound, heard at the same site of S₃.
 - ✓ Cause: Due to sudden arrest of ventricular expansion during diastole as there is NO
 space for this expansion → turbulence of blood & production of sound.

C) Complications: - AF due to MS, ASD & Constrictive Pericarditis.

☆ Investigation :-

- 1-X-ray: Globular heart, 50% show calcifications.
- 2-ECG:-changes in ST segment & T-wave.
- 3-ECHO:- No free space.
- 4- Angiography: no space outside opacified heart.
- <u>5- Catheterization</u>:- high equal pressure in all champers, i.e., end diastolic pressure is equal in all chambers (pathognomonic).

☆ Treatment :-

A) Pericardiectomy:-

- * Avoid myocardial injury to guard against arrhythmia & AF
- Begin with left side first, because if started by right side → ↑ blood flow to lung then to the obstructed left side → lung oedema.

B) Anti-TB drugs :-

- Because it is the most common cause
- * Taken for 6 m before operation & 1 year after it

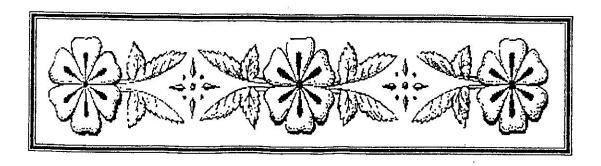
Adhesive Pericarditis

Example 2 There is Fibrous tissue between heart & surrounding structures as ;

- Sternum.
- Oesophagus.
- Mediastinum.

🛎 Findings in Adhesive Pericarditis:-

- Fixed, shifted Cardiac apex.
- Broad Bent sign: Retracting of lower end of sternum & intercostal space with each systole
- Kinking of barium filled esophagus.



Cardiomyopathy

** Definition :- It is a disease of cardiac muscles in absence of known primary cardiac aetiology (the cause may be systemic)

☆ Causes :-

A) Primary: Post partum (purpeurial) ⇒ No cause.

B) Secondary (systemic Aetiology):-(ETMCNI)

- 1- Endocrinal:- hypothyroidism, Acromegally & Addison's disease.
- 2- Toxic: alcohol & Emitine.
- 3- Metabolic: DM, amyloidosis & Glycogen storage diseases.
- 4- Collage disease :- Vasculitis.
- 5- Neurological: Freidrich ataxia, Myotonia atrophica & Duchenne myopathy. (do ECG).
- 6- Infection.

☆ Clinical Presentation: (Clinical types)

- 1-Dilated (Congestive) cardiomyopathy:- It occurs in the following conditions,
 - Congestive heart failure (RSHF or RSHF on top of LSHF) .
 - Dilated left & right ventricle with MR, TR.
 - Thrombo-embolism (common).
 - Arrhythmia.
- 2- Obstructive Cardiomyopathy: in case of IHSS & Fredrich's ataxia.

3- Restrictive cardiomyopathy:-

- It is due to infiltration of heart as in amyloidosis.
- * C / P:- constrictive pericarditis (if right side) or HF (if left side).
- It is liable to thrombo-embolism.
- # Investigations: X-ray, ECG, ECHO (all as HF) & Biopsy → infiltrated myocardium.
- * Differential diagnosis:- from Rh.HD, CHD, HTN, Coronary HD & cor-pulmonale.
- * Treatment: Definitive treatment is Cardiac transplantation; but;
 - In dilated type :- Treatment of HF & arrhythmia , give Anti-coagulants .
 - In obstructive type :- β -blockers & CCB .
 - In restrictive type: No treatment (for fibrotic endocardium) → exercise & treatment of
 HF & Thrombo-embolism.

Aortic Aneurysm

** Aetiology:- Congenital, Traumatic, Inflammatory (syphilitic & mycotic) & atherosclerosis.

** Site:- Ascending aorta (most), Aortic arch (less) & Descending aorta (rare).

A Clinical presentation:

A) Aneurysm of ascending aorta: (many signs & minimal symptoms)

Inspection & palpation :-

- Systolic expansile pulsation over A1, 1st ICS & supra-sternal notch.
- Palpable S2 & systolic thrill over aortic area.
- Apex normal or displaced down & out due to associated AR \rightarrow LVH & LV is pushed down by aneurysm.
- Percussion: dullness over aortic area.
- Auscultation :- (over aortic area)
 - Loud rising S2.
 - Ejection systolic murmur (relative AS).
 - Early diastolic murmur (AR).
- <u>FLarge aneurysm:</u> gives the picture of mediastinal syndrome (write in brief).

B) Aneurysm of arch of aorta: (many symptoms & minimal signs).

- Pressure on surrounding :- clinical picture of mediastinal syndrome.
- Supra-sternal pulsation .
- Rupture of aneurysm: either in;
 - In trachea → fatal haemoptysis.
 - In oesophagus → fatal heamatesis.
 - In pericardium → fatal heamopericardium.
 - In outside → fatal external haemorrhage.
 - In pleura or Mediastinum → rare.
- <u>Gracheal Tug:</u> upward suspension of larynx show → conducted pulsation from aneurysm.

☆ Complications:-

- Rupture.
- Thrombus formation.
- Pressure on surrounding → mediastinal syndrome.
- Dissection → Dissecting aortic aneurysm.

☆ Investigation :-

- Plain X-ray (screen):-
- Fusiform or saccular aneurysm with marked pulsation.
- Irregular calcification in its wall...
- Aortography.
- Wasserman's test: for syphilis.
- GCT.
- ⇒ ECHO.

☆ Treatment :-

- Medical: Bed rest, antibiotics (if the cause is infection).
- Surgical:-
 - Excision & graft.
 - In dissecting aneurysm → use composite graft
 - In case of AR → use graft contain aortic valve.

N.B.:- Chest pain in aortic aneurysm is of 2 types:-

- Aoralgia: Retrosternal sawing pain due to stretch of surrounding sympathetic Fibers around aorta.
- O Neuralgia: brachial or intercostal pain due to compression on roots of spinal nerves.

Dissecting Aortic Aneurysm

☆ Clinical presentation :-

As Aortic aneurysm .

It gives clinical picture similar to that of Acute MI but with the following differences;

Parameter	Dissecting Aortic aneurysm	Acute MI
◆ Chest pain :-	✓ start acute .	✓ Start acute.
* Character of pain :-	✓ Tearing pain.	✓ Compressing.
	✓ Maximum from the start.	✓ Building up pain
* History of :-	✓ Atherosclerosis .	✓ Anginal attack.
	✓ Sever HTN .	
<u>.</u>	✓ Marfan's syndrome .	
* X-ray :-	✓ Widening of aortic lumen .	✓ Not effective.
	✓ Ca sign :- > 1cm thick	
	aortic wall .	
◆ECG:-	✓ Free , except LVH .	✓ Infarction changes .
◆ Cardiac enzymes :-	✓ Insignificant.	✓ High enzymes.
<i>◆ ECHO :-</i>	✓ Diagnostic .	✓ Diagnostic .

Syncope

★ Definition: Sudden transient loss of consciousness due to sudden transient generalized brain ischemia.

* Aetiology:-(H, 2C, 2V)

A) Hypoxia:- 1 O2 supply to brain.

- Hypoxic cor-pulmonale.
- Sever anaemia.
- Congenital Cyanotic HD ⇒ Tetralogy of Fallot (F4).

B) Cardiac :-

- ◆ ↓ Filling:- sever tachycardia & cardiac tamponade.
- ◆ ↓ Pumping :- acute HF , sever bradycardia & cardiac arrest .
- Obstruction :- stenotic valve .
- Cyanotic spills :⇒ central cyanosis...
- Adam's stoke attack

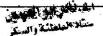
C) Cerebral :-

- Hypertensive encephalopathy .
- Hyperventilation → CO₂ wash → respiratory alkalosis & cerebral VC.
- Thrombosis & embolism.

D) Vasomotor: [orthostatic, postural hypotension]

- During transmission from supine to upright position, pressure $\downarrow > 30$ mmHg as in;
 - ✓ Brain stem or spinal cord lesion (factors control diameter of b.v. → VMC).
 - ✓ Hypovolaemia → Addison's disease.
 - ✓ Neuritis (DM , alcohol) & lumbar sympathectomy .
 - ✓ Ganglion blockers.
 - ✓ Huge varicose veins.
 - ✓ Flabby muscles in prolonged recumbency.
 - √ α-Blockers.
- In all these conditions, there is failure of normal VC of LL vessels to shift blood to brain during change of position from spine to upright position.

E) Vasovagal :-



• In females :-

- ✓ Sudden pain, bad news or sights due to reflex vagal stimulation.
- ✓ Diagnosis: Bradycardia.
- ✓ Treatment :- SC Atropine.

• In males :-

- ✓ Sensitive carotid sinus syndrome as in shaving & tight tie
- ✓ Trauma to sensitive area (trigger zone).

☆ Causes of exertional syncope :-

- AS → Commonest .
- MS .
- * Infective endocarditis.
- Obstruction of outflow of LV → hypertrophic obstructive cardiomyopathy.
- Sever pulmonary HTN: due to bilharzial cor-pulmonale.

☆ Treatment :-

A) Emergency treatment :-

- Place patient with face up on bed or floor.
- Lift the LL.
- * Loosen clothes.
- Drash water on face .
- Check pulse & blood pressure :- Tachycardia → Shock or bradycardia → vasovagal attack .
- Cardio-pulmonary resuscitation .
- If there is cardiac arrest → treatment of cardiac arrest.

B) Treatment of the cause :-

- Vaso-vagal :- Atropine :
- Hysterical: sedation or irritation (saline or alcohol SC).

N.B. :-

- If u found a person in unconscious state, test pulse (brady or tachy?).
- Adam's stoke attack :-
 - $\checkmark C/P$:- No pulse, no blood pressure, syncope, cyanosis \pm convulsions.
 - ✓ Time :-
 - Arrest during conversion from idio-ventricular focus to another one .
 - Transmission from 2nd to 3rd degree heart block.
 - ✓ Treatment :- I.V. ephedrine (keep V.rate at 35-40), No lactate, Mixture (glucose + Insulin + K), Temporary pace maker & Perminant pacemaker if all methods fail.

