

* Treatment of Atrial flutter :-

1-Electeric cardioversion :- (It is the treatment of choice)

◆ Sudden total depolarization & repolarization → give the chance for SAN to take the upper hand once again.

2- Medical Cardioversion :- by ;

- A) Digitalis:- It \(\psi\) ventricular rate, leading to one of the following;
 - Patients remain in atrial flutter state.
 - Patient regain his sinus rhythm.
 - ◆ Patient become AF → it may disappear if digitalis stop.

B) Quinidine:-

- It is vagolytic in action, so it leads to;
 - ✓ ↑ Atrial refractory period → stop circus movement & SAN acts.
 - ✓ ↓ Automaticity → ↓ Ability to form impulses i.e. it inhibits ectopic focus.
- It must be used in caution :- because;
 - \checkmark It is vagolytic († conduction) so , you must use digitalis before it .
 - ✓ We must use anti-coagulants with it to avoid embolization.
 - ✓ Dose: -0.2 gm for sensitivity, give in the 2 tab. In first day, 3 tab. In third day & 4 tab. In forth day → each 2 hours up to 6 doses.

☆ Treatment of Atrial Fibrillation:-

1- Electric Cardioversion :-

- AS Atrial flutter.
- Contraindications of DC:-
 - ✓ History of thrombo-embolism.
 - ✓ Old AF.
 - ✓ Persistent Aetiology...
 - ✓ Loan AF.
 - ✓ Heart failure.

2- Medical Cardio-version :-

- A) Digitalis: It ↓ Ventricular rate (not to normal rhythm) as, it atrium contract by regular rhythm → destruction of thrombus in LA ↑ Embolization.
- B) Quinidine.
- C) Verapamil.
- D) β-Blockers.

N.B. :-

* Medical Cardio-version is indicated in :

- ✓ Recent AF.
- ✓ NO history of embolization.
- ✓ Corrected actiology of AF.
- ✓ Normal size & function of the heart.

* Causes of slow AF :-

- ✓ Digitalis toxicity, β-Blockers.
- ✓ Heart block, Loan AF.
- ✓ Myxedematous degeneration .
- * Causes of occasional Cannon S1:- as occasional Cannon (a) wave.
- * Causes of prolonged Q-T interval :- Acute hypoglycaemia & Hypocalcaemia .

C) Heart Block

* Definition: Slowing or interruption of spread of cardiac impulses at any part of conducting system.

☆ Normal Physiology :-

SAN can produce impulses at rate of 120 b/m, but due to vagal control impulses are released at a rate of 70-80 b/m.

Once impulse is released → activation of atrium → reach AVN (at which there is physiological delay) → Ventricles contract after atrial contraction.

- Coarse of impulses: It start at AVN → bundle of Hiss → 2 bundles (under endocardium) → Lt. bundle divides into anterior & posterior fascicle → All right bundle & 2 Fasicles of Left bundle give → Purkinje Fibers for myocardium
- All heart is supplied by vagus except ventricles (Vagal escape phenomenon).

* Types of heart block: - According to the site of interruption;

- ⊃ At SAN → Sino-Atrial block.
- ⇒ Inside Atrium → Intra-atrial block.
- ⇒ At AVN → AV nodal block (will be discussed).
- ⊃ At bundle of Hiss → Infra-nodal block.
- ⇒ At Right bundle → Right bundle branch block (Rt. BBB)
- ⇒ At Left bundle → Left bundle branch block (Lt. BBB).
- ⇒ At Anterior or posterior fascicle → Hemi-block.
- ⇒ At Perkinje fibers → Intra-ventricular block.

Atrio-Ventricular Block (AV Block)

* Definition :- Slowing or interruption of cardiac impulses at AVN.

Aetiology :-

- ◆ Congenital heart diseases.....
- Calcification & Fibrosis of conductive system .
- Cardiomyopathy & Myocarditis .
- Calcified AS.
- Drugs:- Verapamil, Indral, Digitalis toxicity.
- ◆ Physiological Heart block :- e.g. Athelets (High vagal tone). Mobiz type II

★ Degrees of heart block:-

- 1- First degree :- delay conduction from atrium to ventricles.
- 2- Second degree :- It has 2 types :-

* Mobitz type I (Wenkebach's phenomenon):-

 It shows progressive prolongation of AV conduction time until conduction fails completely → Block of atrial impulses at the level of AVN.

Mobiz type I

☆ Mobitz type II :-

• Intermittent failure of AV conduction (2:1,3:1) i.e.; every 2 impulses activating atrium → only one activates the ventricle.

3-Third degree (Compete heart block):-

- All atrial impulses are not conducted to the ventricles & so ventricles beat by Idio-ventricular rhythm (30 b/m) & Atria beta by SAN impulses at rate of 80 b/m.
- There is AV dissociation → Occasional cannon, i.e. It is similar to ventricular tachycardia but slower & with regular rhythm.



N.B.:-

High grade AV block means combination of 2 types of block

• First degree AV block.

• Lt. BBB.

Causes of variable S1:-

 $\star AF$.

Third Degree Heart Block

• All causes of occasional Cannon wave :- Complete HB & Ventricular tachycardia .

All causes of long PR interval → weak S1 & vice versa.

← In case of sinus Bradycardia → there is normal PR interval, but in the first degree HB, there is prolonged PR interval.

A Clinical Presentation & Investigations:-

Parameter	First degree	Second degree	Third degree
A Symptoms:-	Asymptomatic	• Asymptomatic .	• Asymptomatic (if
(19) 5		• Low COP.	pacemaker is in
2	5	• Syncope.	bundle of Hiss).
40			• Low COP.
☆ Signs :-			
→ Neck veins :-	Normal a & cv wave	◆ Type I :- Gradual	• Number of a wave >
	• Prolonged a-c interval.	prolongation of a-c interval	pulse.
	¥	till one a wave is not	Occasional cannon a
		followed by cv wave.	wave.
		◆ Type II :- 2 or 3 a waves	
	5	are followed by one cv	
		wave.	÷
→ Auscultation :	• Weak S ₁ .	◆ Variable S₁ in some	Variable S ₁ .
		cycles.	
☆ Investigations :-	Normal P & QRS .	◆ Type I :- gradual ↑ in	• Fixed P-P interval at
→ ECG :-	◆ Long PR interval >	PR interval till P	rate of 80 b/m.
	0.22 second.	wave is not followed	• Fixed R-R interval at
	(Normal PR = 0.2 sec).	by QRS .	rate 30 b/ m .
		◆ Type II :- 2 or 3 P	• No relation [] P &
		waves are followed by	QRS complex.
	ses officering directly for the following	one QRS.	Variable PR interval.
			• Wide QRS complex .
☆ Rate :-	• Bradycardia .	Bradycardia .	Tachycardia .
☆ Rhythm:-	• Regular .	• Irregular .	• Regular .

* Treatment of heart block :-

- 1- Treatment of the cause.
- 2- Parasympatholytics: to ↓ vagal tone → ↓ conduction, e.g. Atropine, 0.5-1 mg, S.C..
- 3- Sympathomimetics:- as Isoprenaline.
- 4- Artificial pacemaker.

Cardiac Arrest

★ Definition: No blood come out from the heart.

☆ Types:-

- 1- Ventricular fibrillation: Lack of effective pumping → fibrillation waves.
- 2- Asystole :- Cardiac standstill → Complete stoppage.
- 3- Electro-mechanical dissociation: There is electrical activity but NO contraction or relaxation.

Aetiology :-

A) Medical causes :-

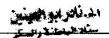
- Drug anaphylaxis.
- · Acute MI.
- Acute pulmonary embolism :
- Coronary HD.
- Cyanotic congenital HD .
- Electric Shock.
- Valvular HD: specially AS.
- Drugs: Digitalis, Procainamide, K & Sympathomimetics.

B) Surgical cause:-

- Anaphylaxis to anaesthesia.
- Hypoxia :- Acidosis during operation .
- Excessive manipulation of viscera → stimulate vagus.

☆ Clinical presentation:-

- Absent pulse, BP & HS.
- Cyanosis.
- Convulsions & coma.
- · Death.



☆ Treatment :- (Cardio-pulmonary resuscitation)

- 1- Put patient on hard object.
- 2- Direct thumbing of sternum in case of VF.
- 3- Cardiac massage :- It is of 2 types ;
 - External: 70 / m, for 15 minute (Cardiac massage: Breathing = 4:1).
 - Internal (intra-operative).
- 4- Artificial respiration :- either by;
 - Mouth to mouth (16 b/m).
 - Umbo bag as pump of air connected to oxygen tube .
 - Endo-tracheal tube + oxygen inhalation.
- 5- NaHCO₃:- for acidosis during arrest.
- 6- Special line of treatment for each type:-
 - VF:- they are;
 - ✓ Intra-cardiac adrenaline, 0.5-1 ml (1/1000), in bare area.
 - ✓ Direct current cardio-version .
 - ✓ Defibrillation.
 - Asystole: Artificial pacemaker. (Immediately)
 - Electro-mechanical Dissociation :- I.V. fluids for hypovolaemia & Atropine .

☆ Prognosis :-

- If arrest for short duration :- Recovery is the role .
- If for long period; It may lead to: Permanent damage & even Death.

N.B. :-

- Conditions for resuscitation :-
 - Correction of brain oedema by Mannitol.
 - It should be continued till full recovery.
 - Correction of hypotension by I.V. fluids & Dopamine.
- In case of VF, if Adrenaline fails, give;
 - Lidocaine: 75 mg/IV/5 min/3 doses.
 - Phenytoin: -100 mg/5 min/3 doses, I.V..
 - * After sinus rhythm is restored, Lidocain is continued by I.V. infusion at a rate of 2-4 mg/min for at least 48-72 hour to prevent recurrence.

GORONARY HEART DISEASES

Anatomy of coronary blood vessels:-

- There is anterior & posterior atrio-ventricular groove between atria & ventricles .
- There is anterior & posterior inter-ventricular groove between the 2 ventricles over septum.
- There are 3 cusps of aorta above each, there is dilatation called ⇒ "Sinus of Valsalva".
- Gross anatomy of Right coronary artery :-

✓ Course :-

- → It arises from right anterior sinus of valsalva.
- → It passes on right side of pulmonary artery till reach AV groove & pass in side it → till right border of heart, then turn backwards in post AV groove crossing the septum.

✓ Branches :-

- → Conus artery to pulmonary.
- ⇒ Branches to → right atrium, right ventricle, SAN (55 %) & AVN (95 %) posteriorly.
- Marginal artery which runs along lower border of the heart to supply both surface of RV.
- → Posterior descending artery → runs in posterior inter-ventricular groove supplying

 1/3 of inter-ventricular septum...
- Gross anatomy of Left coronary artery:---

✓ Course :-

- → It arises from left sinus of valsalva.
- → It passes on left side of pulmonary artery till reach AV groove where it ends by dividing into 2 branches:-
 - Anterior descending branch: which passes in anterior IV groove till apex then turn backwards in posterior IV groove to meet posterior descending artery.
 - © Circumflex artery:- which passes to left side till margin of heart where it turn backwards in post AV groove to meet the right coronary.

✓ Branches :-

- → Anterior descending artery: gives conus artery to pulmonary artery, branches to SAN (45%), RV, LV & anterior 2/3 of IV septum.
- → Circumflex artery: gives branches to LV & gives Marginal artery.

* Patterns of circulation :-

A) Balanced circulation :- in which;

- Rt coronary supply ⇒ RA, RV & posterior 1/3 of IV septum.
- Lt coronary supply ⇒ LA, LV & anterior 2/3 of IV septum.

B) Right coronary predominance:-

• In which Rt coronary supply ⇒ its normal sites + posterior wall of RV.

C) Left coronary predominance:-

• In which left coronary supply ⇒ its normal sites + posterior wall of RV + posterior 1/3 of IV septum.

N.B:- Atherosclerosis affect left branch more therefore, it is commonly obstructed. So, in patient with Lt coronary predominance \rightarrow heart block occurs early as it supply whole septum.

☆ Collaterals of coronary arteries:-

≥ Criteria of coronary collaterals :-

- Coronary arteries are not end arteries.
- They are potential collaterals ⇒ act on need.
- They open on gradual obstruction of coronaries.
- If obstruction occur suddenly → infarction of part below.

≥ Examples :-

- Branches [] branches of one coronary and those of the other ...
- Branches [] branches of one coronary & each other
- Branches [] branches of one coronary & direct cavity (atrium or ventricle).
- Branches [] branches of one coronary & extra cardiac artery.

A General clinical presentation of coronary heart diseases:-

- Silent: coma, syncope, anaesthesia, & DM (dt diabetic neuropathy), so in DM do ECG/6 min.
- Sudden death: due to cardiac arrest.
- Angina:- pain for minutes.
- > Infarction: pain of hours.
- \simeq Pre-infarction syndrome: Prolonged pain (1-2 h) without infarction \to MI within weeks.
- 🖎 Arrhythmia
- \cong HB \rightarrow e.g. Left BBB.
- ≥ Ischemic Cardiomyopathy.

* Aetiology of coronary heart diseases (CHD):-

A) Causes:-

- Atherosclerosis (99 % of cases).
- Syphilitic coronary osteal stenosis.
- Embolism & Thrombosis.
- Dissecting aortic aneurysm .
- Vasculitis (collagen disease):- e.g. Kawasaki disease.
- Spasm in coronary → angina.
- Abnormal origin of Lt coronary from Lt pulmonary artery → infarction in young age.

B) Risk factors:-

Un-Avoidable risk factors :-

- Positive family history.
- ◆ Age > 50 year (common).
- Sex :- male > female (due to endogenous oestrogen & progesterone) then male = female (post menopausal).

Avoidable risk factors :-

- Syndrome X:- hyperlipidaemia, hyperglycaemia, HTN, Hyperuricaemia,

 Hyperinsulinaemia & NASH (Non Alcoholic Steato-Heapatitis).
- smoking :- Carboxy Hb & Atherosclerosis .
- Sedentary life.
- Stress.

C) Contributing factors:-

- \downarrow O₂ due to advanced lung disease.
- 1 HB due to advanced anemia.
- · Advanced aortic disease.
- ◆ Gall bladder disease & colonic disturbance → Reflex coronary spasm → chest pain (Cardiac link) .

الله فالتواجع المهندين مناوع بعدالة والهداد

D) Precipitating factors:-

سى السيد راجع من السّنعل

- Effort.
- Heavy meals.
- Exercise after meal.

- * Emotions.
- Hypoglycemia (stress).
- ه انعنول د خل فی الدینه
- Cold path → ↑ catecholamines

N.B. :-

* A driver complains of chest pain after driving in cold night, you should suspect coronary HD or myocarditis.

* Causes of Hyperlipidaemia:-

- ✓ Primary :- Genetic & Familial .
- ✓ Secondary: Hypothyroidism, DM, Nephrotic syndrome, Cushing's & Drugs (Corticosteroids, oral contraceptives).

* Fasting serum lipids :-

- ✓ No chylomicrons.
- \checkmark ↑ VLDL \rightarrow ↑ TGs.
- ✓ ↑ HDL → ↓ cholesterol (Protective).
- \checkmark ↑ LDL \rightarrow ↑ cholesterol (Atherogenic).

* Discuss Atherosclerosis :-

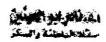
- ✓ Mention risk factors.
- ✓ C/P: Ischemic heart, Cerebral stroke, renal artery stenosis & PVD.
- ✓ Investigations :- Serum lipids (explain).
- ✓ Treatment: Diet control & Hypo-lipidaemic drugs.

☆ General Investigation for CHD:-

1- ECG :-

★ In angina :-

- During attacks :- it shows;
 - ✓ Straight flat depressed ST segment except in prinzmetal angina (elevated ST segment).
 - ✓ Flat or inverted T wave .
 - ✓ Dysrrhythmia .
- In between attacks: normal or show ischemic changes in sever cases.
- * In infarction:- It occurs only in leads representing the part in infarction
 - First sign :- Raised convex (coved) ST segment.
 - Late pattern (evolving):- inverted T wave is added.
 - Then; Appearance of Q-wave.
 - On recovery: all changes disappear except Q-wave persists (stigmata of infraction).
- N.B.: In Pericarditis, there is Concave upwards (raised) ST segment) & It appears in all leads (all heart surfaces).



- 2-Stress ECG:- It is done by ergometer or treadmill; Ask about chest pain ???
 - * Take care of HR, BP, ECG changes in this patient.
 - * Results :-

🖙 In angina :-

- Done [] attacks to precipitate an attack of angina when ECG is free.
- * BP & HR → No increase → Ischemia (supposed to ↑).
- ECG: Appearance of ischemic changes.
- <u>In infarction</u>:- It is done 6 weeks after acute infarction to assess exercise tolerance & improvement before discharge.
- ★ It is considered +ve if ⇒ chest pain, Dyspnea, Fatigue, claudication & depressed ST segment > 1 m.volt.
- 3- ECHO: During attack of angina & infraction, It shows;
 - * Abnormal wall motion :-
 - Hypokinesia → weak movement.
 - Dyskinesia → paradoxical movement.
 - Akinesia → no movement.
 - * It helps in evaluation of ventricular function by ejection fraction.

4- Stress ECHO :-

- ★ In angina: done in between attacks.
- * In infraction :- done after 6 months.

5- Isotopic studies :-

* In angina :-

- Using Thalium²⁰¹, IV \rightarrow go to heart \rightarrow taken by healthy parts, then use γ -camera which show cold spot that means either dead area (MI) or area of hypoperfusion (Angina).
- After 4 hours, take another picture;
 - \checkmark If the cold spot is absent \Rightarrow it was Angina.
 - ✓ If it persist ⇒ old infraction (fibrous tissue).
- $★ In fraction :- Using Tc⁹⁹ pyrophosphate, IV → go to heart → taken by necrotic tissue → gives a hot spot <math>\Rightarrow$ it indicates recent infraction.
- 6- Stress Isotopic studies: Use Thallium in angina between attacks & in infarction.

7- Coronary Angiography:-

* Indications :-

- *In angina:- in 2 cases;
 - ✓ Persistent or recurrent angina with –ve investigations.
 - ✓ Narrowing of > 50 % of coronary lumen.
- * In infarction: It is done pre-operative & if there is occlusion of one artery.
- If there is concomitant AS.
- Pre-operative e.g. before any bypass operation .
- If there is persistent chest pain.

* Disadvantages :- It maybe normal in the following cases;

- Coronary artery spasm; in 5-10 %.
- Syphilitic osteal stenosis.
- Cardiac syndrome X:- obliteration of small arterioles.

8- Evidence of tissue damage :-

- * They are :- ESR, WBCs count, Cardiac enzymes & Iso-enzymes, Cardiac troponin.
- * Results :-
 - In angina :- ↓ ESR , leucopenia , ↓ Cardiac enzymes .
 - * In infarction :- † ESR, leucocytosis, † Cardiac enzymes & † Troponin ...
- * Details on each cardiac enzyme & Isoenzymes :-

Enzyme	Normal level	Rises within	Return normal within
1) CPK	0 - 4 U/ml	6 h	3 days
2) AST	8 - 40 U/ml	12 h	7 days
3) LDH	150 - 250 U/ml	24 h	10 days
4) HBD	50 - 250 U/ml	48 h	12 days

N.B.:- these cardiac enzymes are named as follows;

- CBK :- Creatine phosphokinase.
- *AST:- Aspartate amino-transferase (It is also named SGOT).
- ◆ LDH :- Lactate dehydrogenase.
- * HBD :- Hydroxy Butyrate dehydrogenase .

5) Iso-enzyme:-

- They are fraction of the same enzyme having the same catalytic action but differ in some physical properties that help in their isolation.
- Examples :-
 - ✓ CBK:- CBK-BB → brain, CBK-MM → skeletal muscle & CPK-MB → heart.
 - ✓ LDH: $LDH_1 \rightarrow heart \& LDH_5 \rightarrow liver$.
- 6) Cardiac troponin: It increase within one hour of infarction (most sensitive & specific).

9- Investigations for risk factors :-

- * Blood sugar :- To exclude DM.
- * Wasserman test:- to exclude syphilitic osteal stenosis.
- * Uric acid :- to exclude Hyperuricaemia.
- ★ Lipids in blood :-
 - Cholesterol: \uparrow in 50 % of cases (N = 150 250 mg %).
 - Lipoproteins: normal LDL: HDL ratio = 2:1, when it becomes > 2.5:1, it means atherosclerosis.



★ Definition: It is a clinical syndrome due to myocardial ischemia characterized by attacks of chest pain which is precipitated by effort & relieved by rest & sublingual nitrates.

* Aetiology:- (the same precipitating, contributing & risk factors). Causes are the same +

Low COP :-

- ✓ Quantitative (↓ COP):- ↓ filling, ↓ pumping & obstruction.
- ✓ Qualitative (↓ oxygen) :- Anemia & central cyanosis .
- ◆ ↑ demand :- ventricular hypertrophy & Thyrotoxicosis.

\$\times Clinical presentation :-

A) In between attacks: - Patient is completely free.

B) During attack:-

1- Symptoms (PADE)

≯ Pain :-

- For 3 30 min (never seconds, never hours).
- Site: retro-sternal (behind upper & middle 1/3), Rt chest, epigastrium, back & never infra-mammary on left.
- Radiation: Lt shoulder, medial aspect of forearm, little finger, epigastrium, Rt shoulder, back, jaw & tongue
- Characters: Constricting, compressing, oppressing maybe burning, staping or heaviness & Never; stitching (pericarditis), bricking (neurosis) or throbbing (abscess).
- ↑ by effort & ↓ by rest & sublingual nitrites.
- * Angor enemi :- fear of emending death .
- * Dyspnea :- Transient ischemia → LVF → lung congestion.
- * Eructation :- at end of attack; may be autonomic.

2- Signs

- * No signs maybe present.
- * Pallor & cold skin.
- * THR & BP:- due to sever pain.
- * Auscultation: Weak HS, Paradoxical splitting of S2 due to delayed function.
- * Paradoxical pulsation of ischemic area..
- * Papillary muscle dysfunction → MR.
- * Investigation :- As mentioned + negative tissue damage.
- * Differential diagnosis :- of acute chest pain .
- ☆ Types of angina:-
 - 1- Stable angina: angina of effort (angina pectoris), pain is relieved by rest.

2- Unstable angina:-

- Any recent angina within 8 weeks or recent change in pain of stable angina.
- → Examples :-
 - ✓ Post infarction angina .
 - ✓ Pre-infection syndrome: attack of angina prolonged for hours with normal cardiac enzymes → develop MI in hours.
- → Treatment of pre-infarction syndrome :- Anti-coagulant & admission to ICU or CCU.
- 3- Silent angina: Angina with NO pain, due to;

4- Prinzmetal angina (Angina inversa or Variant angina):-

- → It occurs at fixed time during / day due to coronary spasm.
- → ST segment is elevated then it become depressed again.
- Treatment: CCB (amlodepine) & NOT β-blockers (as it → unopposed α-action → ↑ coronary artery spasm).

5- Other types :-

I] Linked angina:-

- Diseased GB + diseased coronary → anginal pain felt at gall bladder site.
- So; in gall bladder pain → Do ECG to exclude this cardiac link.

II] Angina of lewis :- (NARA)

- It is Nocturnal: during sleep → ↓ HR → ↑ diastolic period → ↑ regurgitant
 blood form aorta → ↓ coronary blood flow.
- It is also; $\underline{\mathbf{A}}$ utonomic, $\underline{\mathbf{R}}$ esistant to treatment & occur in $\underline{\mathbf{A}}R$.

III] Angina decubitus :-

- Pain occur on lying down & relieved by sitting.
- Caused by LVF: lying down → ↑ VR → ↑ congestion → ↑ failure → ↓ COP → ↓ coronary filling.
- It is of bad prognosis as it develop infarction within weeks.
- Treatment :- Digitalis (0.25 mg).

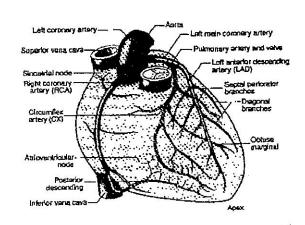
IV] Status angiosus :-

- It can occur even at rest.
- There is ↑ in frequency & duration → bad prognosis.
- It is resistant to treatment.

☆ Treatment of angina :-

A) During the attack :-

- 1- Rest.
- 2- Short acting vasodilators :- (Nitrates), as;
 - → Nitroglycerine (Angised):-
 - It has rapid action & it bypass the liver.
 - It is taken sublingual, transdermal, ointment or IV.
 - → Isosorbide dinitrate (Isordil) :- only sublingual .
- <u>Action of nitrates</u>:- Coronary VD & ↓ preload → ↓ heart work.



B) Between attacks:-

- 1- Treatment of the cause, Contributing factors & Precipitating factors.
- 2- Treatment of risk factors: especially atherosclerosis by;

→ *Diet :-*

- ↓ Total calories, ↓ fat to 30 %, ↓ cholesterol < 300 mg / day.
- † polyunsaturated oils (fish oil & vegetable oil).

₩ Drugs :-

- ↑ cholesterol & LDL → use cholestyramine.
- ↑ TGs & VLDL → use clofibrate.
- ↑ both → use nicotinic acid.
- 3-Bed rest:-till attack relieved in case of unstable angina.
- 4- Exercise: It has the following criteria;
 - Graduated & not Isometric (no muscular contraction) as it 1 cardiac load .
 - Value: -↓ HR, ↓ BP, ↑ mechanical efficiency of skeletal muscle, sense of well-being & open the collaterals.
- 5- Sedation :- e.g. Valium.

6- Anti-anginal drugs:-

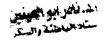
- * Long acting vasodilators: as Nitrite which are VD → ↓ pre-load → ↓ demand.
 - → Nitroglycerine (Nitromag):- oral, 2.5 mg.
 - ➡ Isosorbide dinitrate (Isosordil):- oral; 10, 20, 40 mg,

* β-Blockers :-

- used in all types except prinzmetal angina.
- → Mechanism :- ↓ Cardiac work → ↓ HR → ↑ diastolic period → ↑ coronary filling.
- ⇒ Example: Propranolol (160-240 mg/d) & Atenolol (50-100 mg/d).
- N.B. :- If no response to β -blockers \rightarrow use Prehexiline (pixid) \rightarrow 1 cardiac work .

* Calcium Channel Blockers (CCB):-

- \rightarrow used when β -blockers are ineffective & in prinzmetal angina .
- → Example: Verapamil (80 mg t.d.s) & Deltiazem.
- → Mechanism :- ↓ HR & Cardiac work → prevent V.C → coronary VD.



- 7- Anti-coagulants: as; Heparin, 5000 U/6h, in pre-infarction & unstable angina.
- 8- Family education: about management during attack.
- 9- Anti-platelets: (in stable angina), as;

 \rightarrow Aspirin: - not > 325 mg (if > 325 mg \rightarrow analgesic).

- → Dipyridamole (persantin):-75 mg t.d.s., it causes shift of blood from diseased area to healthy one ⇒ Coronary steal phenomenon.
- → Ticlopedine.

10-Surgical treatment:-

- * It depends on results of cardiac catheterization;
 - → If coronary stenosis → Percutaneous transluminal coronary angioplasty (PTCA).
 - → If coronary occlusion → Coronary Aorta bypass grafting (CABG);
- * In CABG, we can use:-
 - Graft of great saphenous vein which is not under autonomic supply of the heart & liable for occlusion.
 - → Graft of internal mammary artery (recent).
- * Precautions of CABG:-
 - → No major risk factors.
 - → Normal heart size.
 - \rightarrow < 65 years old.

- → Normal LV functions.
- → Localized disease.

Myocardial Infarction

→ Definition: Necrosis of cardiac muscles due to sudden, complete & persistent stoppage of blood supply.

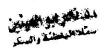
N.B. :- If this stoppage is transient \Rightarrow Prinzmetal angina.

A Actiology of Myocardial Infarction (MI):-

- → Risk, contributing & precipitating factors:- as in angina.
- Cause: as in angina but; No 1 in requirements & there's a spasm on top of thrombus.

☆ Pathology :-

- → Coagulative necrosis after 1 week → granulation tissue formation which is after 5-8 weeks is replaced by fibrous scar.
- → Consecutive frequency of affection :- Anterior descending artery → Circumflex artery
 - → Rt coronary artery.



* Pathological types of infarction :-

- 1- Transmural infraction (associated with Pericarditis).
- 2- Subendocardial infraction.
- 3- Non transmural infraction.

N.B.:-

- rightharpoonup Transmular infraction
 ightharpoonup necrotic tissue which may reach pericardium
 ightharpoonup pericarditis
 ightharpoonup Pericardial rub.
- This type is dangerous as it is liable to rupture → cardiac tamponade except if it is closed by clotting.
- <u>SO</u>; In a case of pericardial rub, there is absolute contraindication to use heparin as if it is transformed into infarction, it will lead to cardiac tamponade which is fatal.

☆ Clinical presentation :-

A-Symptoms

1- Pain :-

- Site:- as angina but radiate more.
- Character: as angina but more sever.
- Duration :- more than angina .
- Precipitating factors :- as angina BUT not relieved by rest or sublingual nitrates .

2-Shock :-

- Neurogenic due to pain :- good prognosis + bradycardia...
- Cardiogenic due to massive infraction :- bad prognosis + tachycardia .

3- Heart failure :-

- Especially; LVH (anterior descending artery) with dyspnea & lung congestion → cardiac asthma & pulmonary edema.
- It maybe due to neurogenic shock.

4- Complications: enumerate (mentioned later).

N.B. :- Causes of painless infarction :-

- ≥ Diabetic neuropathy .
- ≥ Tabes dorsalis.
- Shock.
- 🗷 Syncope.
- ≥ Pulmonary oedema .
- 🗷 During anaesthesia .

B-Signs

I] General signs :-

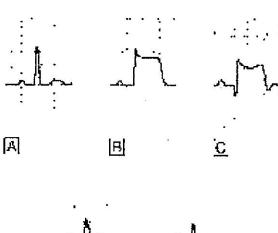
- 1- General appearance: in agony (pain), dyspnea (HF), collapsed (pallor & sweating) due to shock.
- 2- Closed fist sign (Le Veen sign):- patient put his closed fist on sternum to support it as if it will burst out.

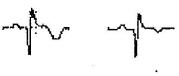
3- Vital signs :-

- * Pulse :-
 - Rate: either Normal, Bradycardia (HB, neurogenic shock) or tachycardia (pain & cardiogenic shock).
 - → Rhythm:- Arrhythmia.
- * Blood pressure :- either;
 - → Normal.
 - → Hypotension: due to shock.
 - → Hypertension :- due to pain .
 - → ↓ systolic with normal diastolic :- in previous HTN (decapitation) [patient was 180/100 & when get MI becomes 130/100]
- * Temperature: Fever in 2-6th day due to absorption of necrotic material.
- * Respiration: Rapid & Shallow (shock).

II] Cardiac signs :- as angina + pericardial rub.

★ Investigation: As mentioned, urine examination → oliguria (shock).





* Complication of Myocardial Infarction :- [layers of the heart]

A) Endocardial complications:-

- 1- Thrombosis.
- 2- Vegetations → Infective endocarditis.

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B) Myocardial complications:-

- 1- Shock :-
 - * Neurogenic shock :- leads to heart failure → Low COP.
 - * Cardiogenic shock :- 85 % mortality, treatment is cardiac transplantation.

2- Arrhythmia:-

- * Tachycardia: due to electrical instability of ischemic heart.
- * Bradycardia:- due to damage of conducting system.
- * Heart block.

3- localized complications in infarction:-

- * Rupture of infarction :- in;
 - → Cardiac muscles → Haemopericardium.
 - → Septum → VSD.
 - → Papillary muscle → MR.

* Dilatation of infarction scar (Myocardial Aneurysm):-

- → Suspected in :-
 - ✓ Double apex (on palpation)
 - ✓ Weak first heart sound on apex.
 - ✓ ECG: Persistent elevation of ST segment (it is supposed to return to normal after few days).
 - ✓ Echocardiography & Angiography :- Diagnostic .
- → Complications of Myocardial aneurysm: Rupture aneurysm, Resistant HF, Resistant arrhythmia, IE & thrombus formation.

C) Pericardial complications:-

- 1- Dry Pericarditis.
- 2- Haemorrhagic Pericarditis.
- 3- Heamopericardium.
- 4- Dressler's syndrome :- (3P+F)
 - * Absorption of necrotic tissue of infarction → act as antigen → stimulate Abs formation & after 2-6 weeks → Pleurisy, Pericarditis, Pneumonitis & Fever.
 - * It is autoimmune Pericarditis, so it is treated by Corticosteroids.

D) Miscellaneous complications:-

- 1- Depression .
- 2- Complications of prolonged bed rest.
- 3- Complication of anticoagulants (will be mentioned later).
- 4- Frozen shoulder: Stiffness with limitation of movements, due to
 - * Pain.:- reflex spasm & ischemia, treatment is analgesics & physiotherapy.
 - * Immune reaction: Auto-immune capsulitis, associated with auto-immune Pericarditis, treatment is corticosteroids.

N.B.: - Frozen shoulder occur in Myocardial ischemia & DM.

* Differential diagnosis of MI:- From causes acute chest pain.

* Treatment of Myocardial infarction:-

A) Pre-hospital treatment: for;

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1- Pain :-

- * Morphine: 5-10 mg IV, SC; not used in bradycardia as it † vagal tone.
- * Pethidine: 50-100 mg, IM.
- 2- Arrhythmia: (prophylactic as death occur in 1st 2.5 hours)
 - * If Ventricular extra-systole (PMB):- Lidocaine, 50-100 mg, IM in deltoid muscle.
 - ★ If bradycardia or HB: use atropine, 0.5-1 mg, IV or SC.

B) Hospitalization:-

- 1) Patient admitted to coronary care unit (CCU); where there is must be,
 - * Well trained doctors & nurses.
 - * Continues monitoring by ECG.
 - * DC cardioversion.
 - * Pacemakers.

2) Thrombolytic drugs: - within 1st 5 hours;

- ★ Streptokinase; 1.5 mU.
- * Urokinase.
- ★ Tissue plasminogen activator (TPA):- at site of thrombus.
- 🖎 All of them breakdown the pre-formed thrombus but don't prevent formation of new one .
- 3) Anti-coagulants: they prevent formation of new thrombus;
 - * As heparin, warfarine, (discussed later)
 - * Given for all patients specially extensive infarction, extending infarction, shock, HF & DVT.

4) Symptomatic treatment:-

- * Treatment of pain :- analgesics.
- ★ Treatment of arrhythmia.
- ★ Diet: 1st day (fluids), 2nd day (semi-fluid) & 3rd day (gradual building of diet).
- * Bowel care, Oxygen, stop smoking, laxative & Catheterization.

5) Treatment of complications:-

- * Treatment of cardiac asthma.
- * Treatment of arrhythmia: anti arrhythmic drugs.
- * Treatment of heart block :- Pace-maker.

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6) Surgical treatment:-

* For coronary arteries :-

- → PTCA.
- → CABG.
- Remove atheroma by rotablator or by laser.

* For complications :-

- → VSD :- Closure.
- → Papillary muscle rupture → Artificial valve.
- → Aneurysm → Aneurysmectomy.

C) Post discharge care:-

- ≥ Discharge patient after 7-10 days, if uncomplicated & after 14-20 days if complicated.
 - 1- Vasodilators: nitrites (describe from angina).

2- Anti-platelets :-

- * Aspirin :- (Rivo; 325 mg & aspocid; 75 mg).
 - ightharpoonup If large dose ightharpoonup acts on thromboxane $A_2
 ightharpoonup \uparrow$ thrombus .
 - \rightarrow If small dose \rightarrow act on PG \rightarrow \downarrow thrombus.
- * Dipyridamole (Persantine):-75 mg, 3/day, before meal by 1 hour.
 - → ↑ Perfusion to ischemic area.
 - → ↓ Platelet adhesion.

3- Rehabilitation :-

- ★ Guide for graduated muscular exercise.
- ★ Full activity not allowed before 12 weak.



Anti-Coagulants



** Types:- Heparin & oral anti-coagulants (Warfarin).

Parameter	Heparin	Oral anti coagulants
★ General :-	Carry positive charge:	Groups:-
9 *		• Dindevan; 50 mg tablets.
		• Warfarin; 10 mg tablets.
★ Onset :-	→ Of immediate action.	GOf delayed action, 48-72h.
★ Action :-	→ Neutralize co-agulation	Inhibit vit-K dependant
	factors (-ve charge).	co-agulation factors in liver
		(II, III, IV, X), by
	↓ co-agulation factors (X).	competitive inhibition.
★ Dose :-	= 5000 IU / 6h, SC or IV but	= 1 st day (3 tablet).
	not IM as it cause	= 2 nd day (2 tablet).
	haematoma with heparinase	⊂ 3 rd day (1 tablet).
	enzyme.	
★ Control factor:-	Co-agulation time & APTT.	- Prothrombine
	Must be used till they are 3 times	concentration, which must
	normal, not more (bleeding) &	↓ to 30% of basal level.
	not less (ineffective).	
★ Antidote :-	Fresh blood .	Fresh blood.
	Protamine sulphate (1 mg for each	
5. ·	l mg).	
:	Protamine zinc insulin + glucose.	

☆ Indications :-

- Recent AF, AF+MS.
- * Angina (unstable), Pre-infraction syndrome & Myocardial infraction.
 - After vascular surgery (DVT, pulmonary embolism), Artificial valve.
 - TIAs, Embolic hemiplegia in adults, Stoke in evolution.

* Complications (Side effects of prolonged usage):-

- · Bleeding.
- Hyper-coagulability on sudden stoppage .
- For oral:- Teratogenic, Hepatitis (Dendivan).
- For Heparin :- (if exceeds 10 days use)
 - ✓ Alopecia.
 - ✓ Thrombocytopenia.
 - ✓ Allergy:- asthma, pruritis & anaphylaxis.
 - ✓ Osteoporosis.

N.B :- Begin with heparin & oral anti coagulants till they act then use oral drugs only.

* Contraindications :-

Cardiac :-

- ✓ Infective endocarditis: as embolization + drug → cerebral Haemorrhage.
- ✓ Dissecting aortic aneurysm: prevent closure of bleeding point in aorta by haematoma.
- ✓ Infraction with pericardial rub: If transmural type (see before).
- Chest: T.B → Haemoptysis.

• GIT :-

- ✓ Liver cell failure .
- ✓ Peptic ulcer: It prevents closure of perforation if occur → haemo-peritoneum.
- Blood :- haemorrhagic blood diseases e.g. haemophilia .

<u>N.B.</u>

≥ Elevation of ST segment occurs in 2 conditions :-

1- Myocardial infarction :-

- ◆ No S-wave.
- ◆ Convex ST segment .
- ◆ Deep Q-wave .
- * Appears in one, 3 or 3 leads according to area of infarction.

2- Pericarditis :-

- ◆ No Deep Q-wave .
- ◆ Concave ST upwards .
- Appears in all leads \Rightarrow Universal.

THE MEDIASTINUM

* Definition:- It is the space [] the 2 lungs & pleural cavities including heart & other chest viscera.

Anatomy of the Mediastinum:-

- 1. Superior Mediastinum: above an imaginary line extending from the sternal angle to the

 4th dorsal inter-vertebral disc.
- 2. Inferior Mediastinum :- It is divided by the heart into :-
 - Anterior Mediastinum: anterior to the heart.
 - <u>Middle Mediastinum</u>:- contains the heart and the pericardium.
 - <u> Posterior Mediastinum :-</u> posterior to the heart.

\$ Boundaries :-

- → Laterally:- parietal pleura and the lungs.
- → Anteriorly :- sternum.
- Posteriorly:- para-vertebral gutters and ribs.
- → Superiorly:- thoracic inlet.
- → Inferiorly :- diaphragm.

A Contents :-

A. Contents of the superior Mediastinum :-

- * Aortic arch and its 3 large arteries (innominate, Lt common carotid & Lt subclavian).
- Upper 1/2 of the superior vena cava (SVC) and its 2 innominate veins.
- Trachea and esophagus.
- Thoracic duct & superior sympathetic ganglia.
- Nerves: phrenic, vagus, cardiac & left recurrent laryngeal.

B. Contents of the posterior Mediastinum:

- Spinal nerve roots and the sympathetic chain.
- Vagus and the splanchnic nerves.
- Descending aorta.
- The azygos and hemiazygos veins.
- Thoracic duct & Oesophagus.
- C. Contents of the anterior Mediastinum :- Thymus gland & fat .

D. Contents of the middle Mediastinum :-

- Heart and pericardium.
- Ascending aorta, Pulmonary artery and its main branches.
- ◆ Lower 1/2 of SVC and part of the azygos vein.
- Pulmonary veins & Inferior vena cava.
- Lower part of the trachea and its bifurcation.
- · Phrenic nerves.

NB:- The 3 components contain lymph nodes, connective tissue, lymphatics and nerves.

* Diseases that affect the Mediastinum :-

1. Inflammation :- mediastinitis.

2. Fibrosis.

3. Pneumomediastinum.

4. Masses :- benign or malignant.

☆ Definition of Mediastinal Syndrome :-

It is a syndrome of compression due to space occupying lesion in Mediastinum causing pressure manifestation.

A Clinical manifestations of mediastinal masses:

- 1. Asymptomatic masses: 50 % of all mediastinal masses are Asymptomatic (50 % of them are benign)
- 2. Clinical presentation of the causes: Mention some examples.
- 3. Clinical presentation of compression: (dt compression or invasion of the adjacent structures)
 - Pressure on tubes :- (4 tubes);
 - Trachea:- leading to;
 - Stridor :- inspiratory.
 - Cough: brassy with metal tone.
 - Dyspnea:- especially on lying backwards, so patient prefers leaning forwards (
 Prayer's position).
 - **⊃** Bronchi :- leading to either ;
 - Complete obstruction → Atelectasis:
 - Incomplete obstruction → Emphysema & Broncho-pneumonia .
 - Oesophagus :- leading to Dysphagia & Regurgitation.
 - → Thoracic duct: leading to chylous effusion at pleura, pericardium & peritoneum.

Pressure on vessels:-

- **⊃** SVC :- leading to ;
 - Congested non pulsating neck veins .
 - Oedema & cyanosis of face & upper limb.
 - Dilated veins over the chest.
- - Engorged veins over upper part of chest filled from above downwards.
 - Right pleural effusion.