

HEART FAILURE

HEART FAILURE

- It is a **complex clinical syndrome** of **failure** of the heart **to maintain normal cardiac output** due to any structural or functional cardiac disorder
- Worldwide- HF- **incidence** 2 per 1000
- **Age related disease** which increases up to 90 per 1000 after 85 years old
- **51.5% HF-DEATH** within 5 years- more than death from cancer
- **5% of hospital admission** from HF
- After age of 65 Y most common cause of hospital admission
- **Frequent** hosp. admission carry **poor prognosis**
- **COST** of HF- TREATMENT in UK > 1 BILLION dollar per year

Main mechanisms involved in HF

1-Reduced ventricular contractility- IHD-MI

2-Ventricular- pressure outflow obstruction

- LV- High-pressure -Afterload

HTN- Calcific-Degenerative – AS – Bicuspid-AS- HOCM-
CONG.Sub-Aortic Membrane- Coarctation of Aorta

- RV- PS- Pulmonary embolism -PUL.HTN

3-Ventricular -inflow obstruction: PRELOAD- MVS- TVS

4-Ventricular -increased VOLUME- overload

- AI – PI-MVR-TVR- AV-SHUNT--VSD- ASD- PDA

- High metabolic demand- TACHYCARDIA

- THYROTOXICOSIS - ANEMIA - PAGET S DISEASE

Main mechanisms involved in HF

5-ARRHYTHMIA

- Tachyarrhythmia- AF-SVT
- Bradycardia - Complete heart block - Sick Sinus Syndrome
- Arrhythmia does not give adequate time for good ventricular filling so will reduce cardiac output
- **Tachyarrhythmia Induce Cardiomyopathy**

6-IMPAIRED VENTRICULAR DIASTOLIC FILLING

- Pericardial Diseases- Compressing The Heart
- Pericardial Tamponade
- Chronic Constrictive Pericarditis
- Restrictive Heart Disease
- Amyloidosis – Haemochromatosis

7- TOXIC- Alcohol-cardiomyopathy- Chemotherapy

8- INFECTION - Myocarditis- SBE- Sarcoidosis- Chagas's Disease

9- CKD- Cardio-renal Syndrome

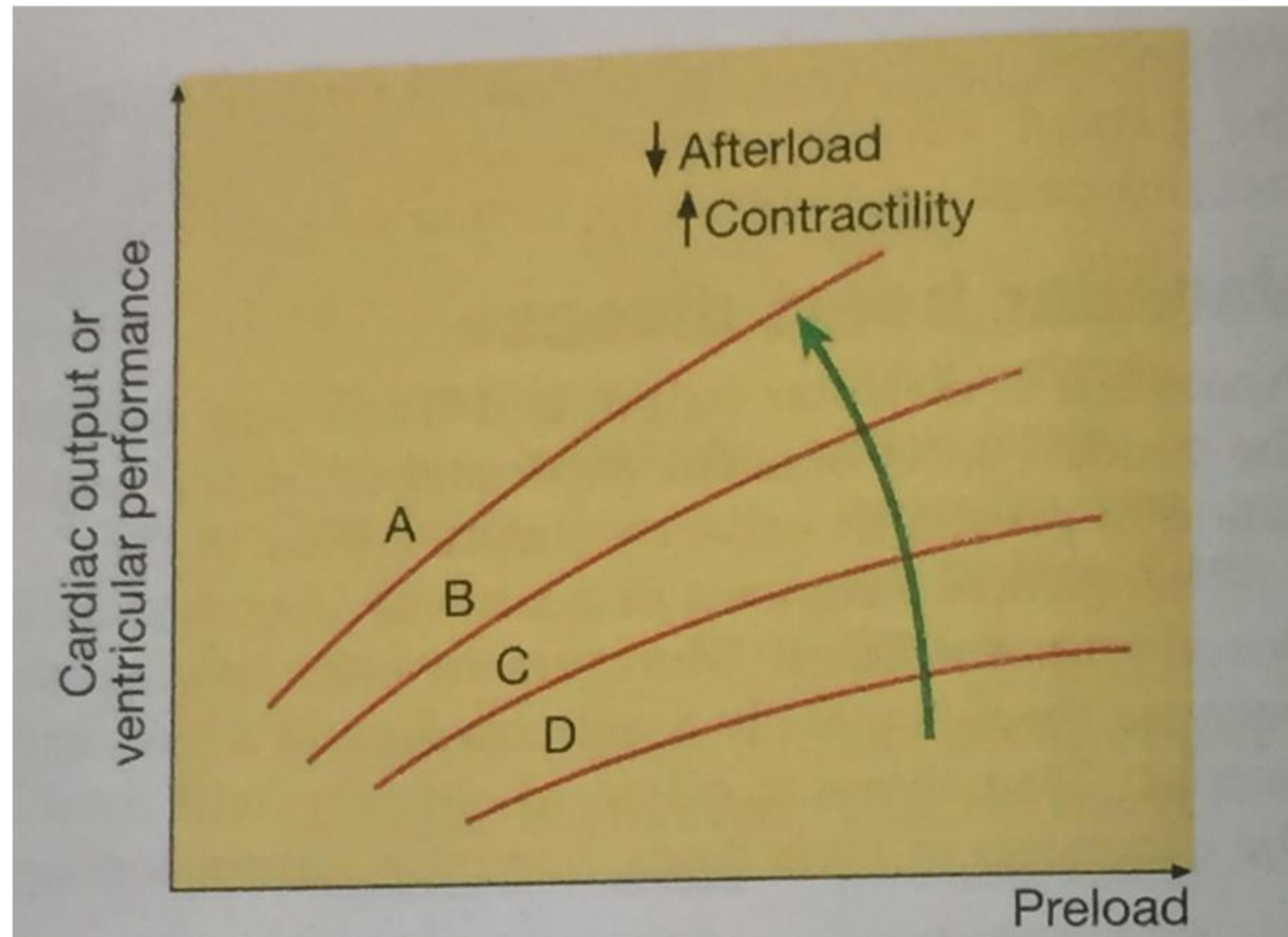
Dm- Diabetic Cardiomyopathy

PATHOPHYSIOLOGY

- STARLING S LAW
- **PRELOAD**- means volume of blood and pressure in the ventricle at the end of diastole
- **Increase** in the **PRELOAD** venous return will increase, myocardial FIBERS stretching which will enhance ventricular performance and cardiac output but over ventricular wall stretching due to increase pre-load will cause marked deterioration of cardiac output ending in **HF- pulmonary edema- SOB**

PATHOPHYSIOLOGY

- LAPLACE S LAW
- AFTER LOAD
- Myocardial Wall Tension; blood volume and pressure in the ventricle during systole
- Cardiac output related to intra-cardiac Pressure (P) multiplied ventricular radius (R)
- Increase in the afterload will increase LV- End-Diastolic Volume; ventricular dilatation; Cardiomegaly, to compensate by increase the cardiac output on expanse of increased -LV-Volume and LV- dilatation.



PRIMARY CARDIAC ABNORMALITY IN HF

- Ventricular function impairment
- Myocardial cells loss – apoptosis – necrosis - myocardial hyaline deposition – fibrosis - cytokines release and free radicals - Low cardiac output - hypotension - neuro-hormonal activation

1-SYMPATHETIC STIMULATION

Noradrenaline release – Tachycardia – Vasoconstriction – High Renin – Increase BP and Afterload – more myocardium work – more myocardial cell damage and loss – decrease cardiac output – vicious cycle.

2- RENIN ANGIOTENSINE ALDOSTERONE SYSTEM

- Na-water retention increase intravascular > Increase blood volume and BP increase both cardiac PRELOAD and AFTERLOAD > more myocytes damage and loss > decreases cardiac output- vicious cycle

PRIMARY CARDIAC ABNORMALITY IN HF

3- HIGH BNP-ANP-

- Secretion by **stretch volume receptors stimulation** in ventricles and atrium - Natriuresis – Vasodilatation
- High BNP level - HF-NT- pro BNP
- Level >300pg/ml- sinus rhythm- or > 900 pg/ml in AF

4- ANTIDIURETIC

- High ADH –Vasopressin – H – Water Retention
- ISADH - cause **hyponatremia** in sever HF- **poor prognosis**

CLASSIFICATION OF HF

1- NORMAL EF- variable - 52%-55%

- HFrEF- Systolic HF-Low EF

2-HFpEF-DYSTOLIC HF- NORMAL EF

- Myocardial **Inflammation**- Fibrosis- Stiffness
- **Impaired** LV Diastolic **Filling**
- **Insulin resistance**- OBESITY- DM-
- **No real treatment of HF** only treat underlying causes

3-HFmrEF- EF-40%-<55% = Medium EF

4-HF-IM-EF

TYPES OF HF

1-Left Side – HF: SOB- PND- PULMONARY EDEMA

2-Right Side – HF: LEG EDEMA- ASCITIS- ANASARCA- High JVP

3-Biventricular – HF: Congestive HF

4-High Cardiac Output – HF: THYROTOXICOSIS- ANEMIA- Beri-Beri

5-Chronic – HF: COMPENSATED- DECOMPENSATED

6- Acute – HF:

- ACUTE PULMONARY EDEMA
- ACUTE INFECTIVE ENDOCARDITIS
- ACUTE MYOCARDITIS-VIRAL
- ACUTE RUPTURE CHORDETENDENI-MR- IN -MVP-MARPHAN SYN.
- ACUTE SEVER ACCELERATED AND MALIGNANT HTN
- ACUTE MI
- ACUTE VALVULAR DESTRUCTION
- ACUTE DISSECTING AORTIC ANEURYSM

TYPES OF HF

7-ACUTE ON CHRONIC HF

- DECOMPENSATED HF
- UNCONTROLLED -HTN-DM
- ANEMIA
- ELECTROLYTES IMBALANCE- K- Mg- Ca- Phos.
- ACUTE CORONARY SYNDROME -ACS- ACUTE DECOMPENSATED- IHD
- ACUTE MR-PAPILLARY MUSCLE DYSFUNCTION.
- ACUTE ONSET ARRHYTHMIA- AF
- ACUTE PULMONARY EMBOLISM
- POOR DRUGS COMPLIANCE
- INFECTION-SEPSIS
- THYROID DISEASE

NEW YORK HEART ASSOCIATION- NYHA

Classification of HF

Class-1- No Limitation of Physical Exercise

Class-2- Mild Limitation of Physical Exercise

Class-3- Moderate Limitation of Physical Exercise

Class-4- Symptoms of HF at rest – Orthopnea – PND – Pulmonary edema

NEW CLASSIFICATION

1- Pre-HF: risk factors; structural or functional factors

2- Asymptomatic-HF

3- Symptomatic-HF

4- Advanced-HF

NEW YORK HEART ASSOCIATION- NYHA

CLASS-1-2- MILD cardiac dysfunction:

- **CARDIAC OUTPUT= stroke volume * heart rate**
- Is compensated and maintained by increased venous return and diastolic ventricular filling pressure and increase heart rate.

CLASS-3- SEVER cardiac dysfunction:

- Cardiac output only maintained by marked sinus tachycardia and large increase in venous return at the expense of Pul. interstitial and alveolar fluid accumulation causing SOB on mild exertion but not at rest.

CLASS-4- VERY SEVER HF:

- Cardiac output at rest is severely depressed, despite high venous return and pressure and high heart rate; tachycardia – SOB at rest – Orthopnea - acute pulmonary edema

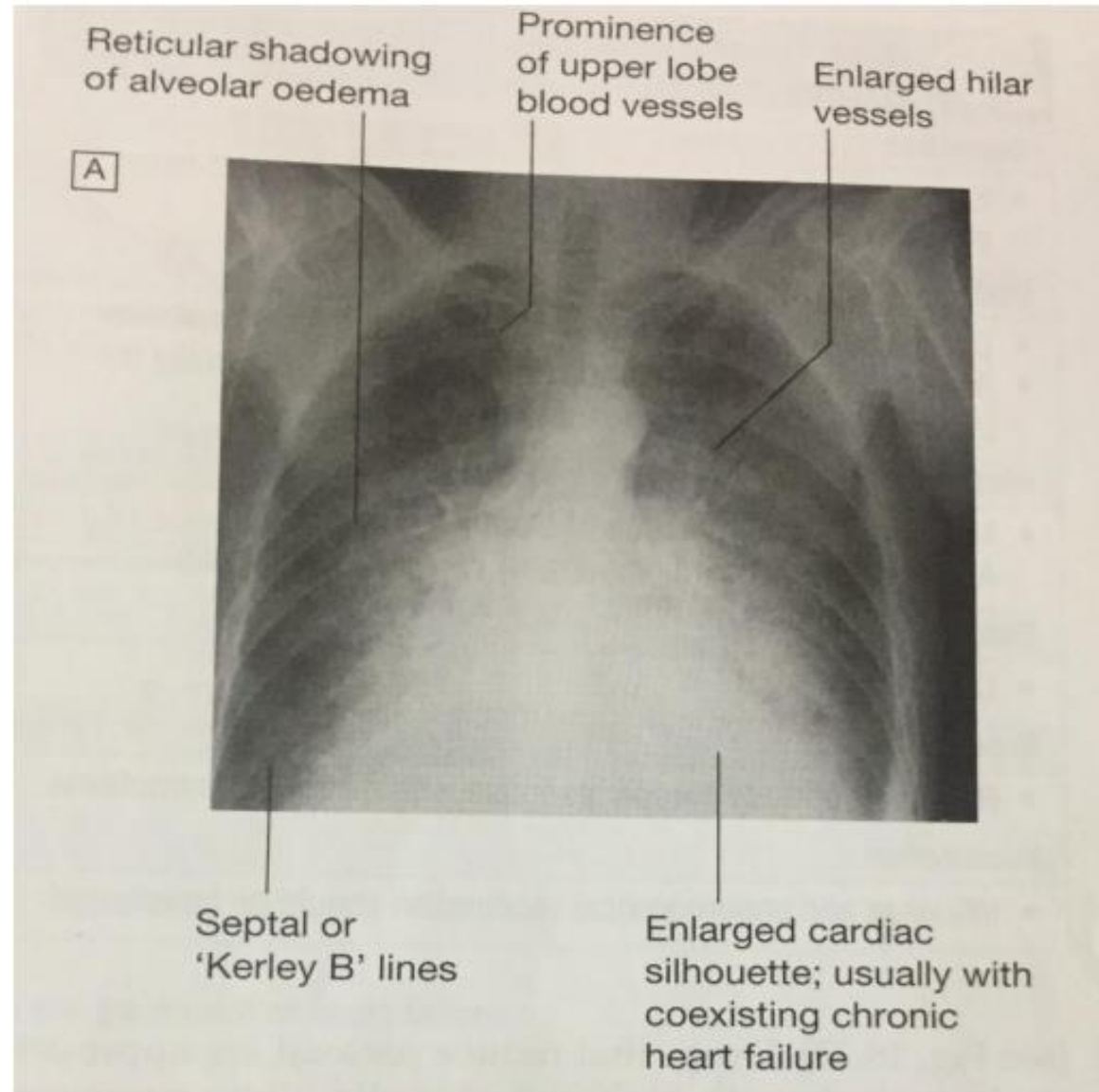
SYMPTOMS AND SIGNS OF HF

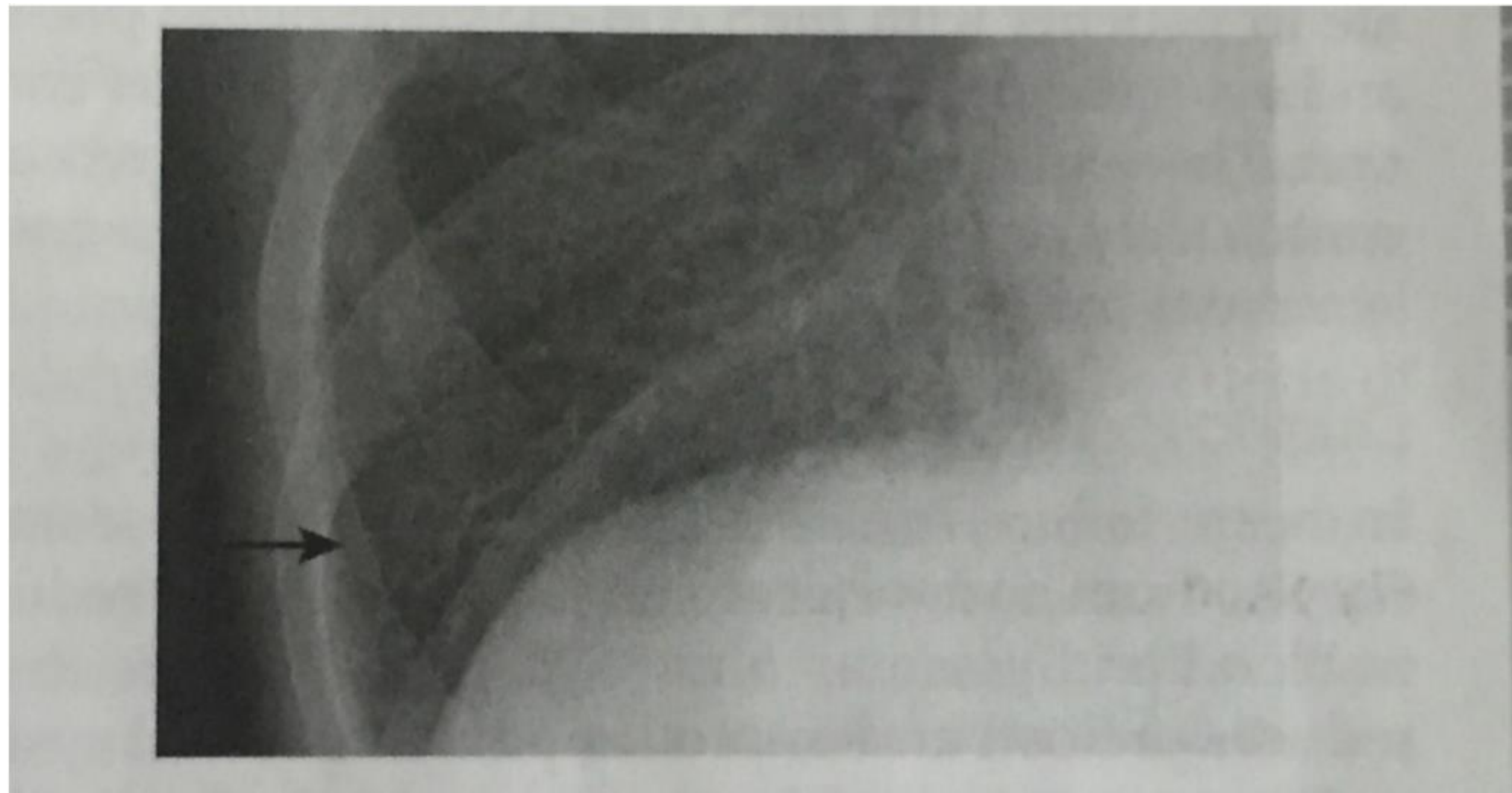
1- SOB – Palpitation

- On Exertion
- Orthopnea on lying flat-at rest.
- PND- SOB-during sleep.
- Acute pulmonary edema

2-Fatigue – Pallor

- Low cardiac output; **blood flow maintained only to the vital organs (brain-kidneys)** at the expense of the muscle and skin.



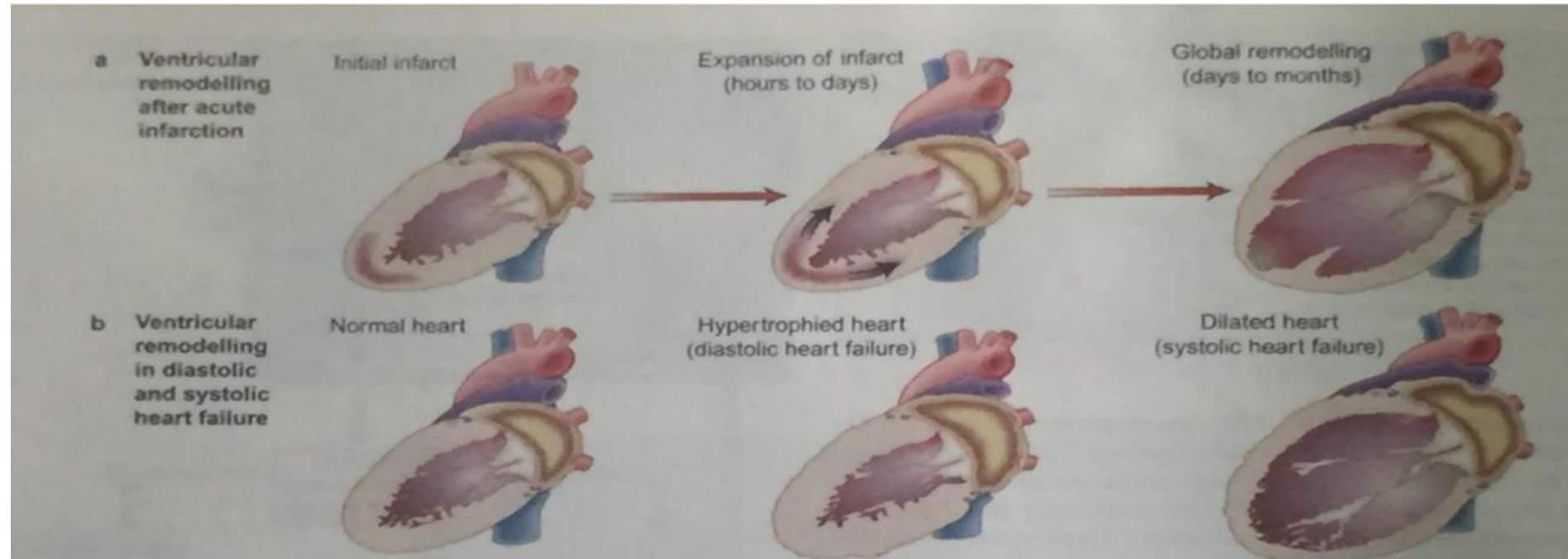


SIGNS OF HF

- 1-High JVP - Central and Peripheral Cyanosis**
- 2-Bilateral ankle oedema- ascites- anasarca**
- 3-Hypotension- dizziness- vertigo- pre-syncope- syncope**
- 4-Tachycardia- Sinus Tachycardia- Atrial Flutter- AF**
 - Atrial or Ventricular ectopics – Extra-Systole**
 - VT-VF- Cardiac Arrest- Sudden Death**
 - Bradycardia- Complete Heart Block**
- 5- 3rd / 4th Heart Sounds- gallop rhythm**
 - systolic murmur-MR- Bilateral lung basal crackles**
- 6-Tender Hepatomegaly- Congested Liver- Jaundice**
- 7-WT Loss- Cardiac Cachexia**
- 8-CX- Cardiomegaly- Pleural effusion- Pulmonary edema**

MYOARDIAL REMODELLING IN HF

- Progressive alteration in ventricular **size, shape and function**
- **Thinning** of the **wall** and **ventricular dilatation**
- Following myocardial **cells damage** ending in progressive -HF





INVESTIGATIONS

1-ECG

2-CX

3- BLOOD TESTS- Cardiac Enzymes

- NT- proBNP level >300pg/ml-sinus rhythm or >900pg/ml –AF
- BIO. FBS-HBA1C- Urea- Creatinine- Lipids- TFTs- CBC- LFT- PT-INR

4- ECHO- Transthoracic- Transesophageal

5-HOLTER STUDY- Ambulatory 24-hour ECG Monitoring

6-NUCLEAR ISOTOPE CARDIAC STUDY

7-CARDIAC CATHETERIZATION

- Both **Diagnostic- Therapeutic**- IHD-PCI
- AS-TAVI- MITRAL VALVE-VALVOTOMY-REPAIR CLOSER VSD-ASD- PDA

8- MRA-CT-ANGIO-CARDIO

MANGMENT

- Advice
- Change lifestyle
- Influenza and corona vaccination
- Treat the underlying etiology

A- PHARMOCOLOGICAL TREATMENT

1- DIURETICS- Loop Diuretics- Frusemide- Hydrochlorothiazide

MRA-K- Sparing Diuretics

Aldosterone Receptors Antagonist: Spironolactone – Eplerenone

2- ACEI- Enalapril- Lisinopril- Ramipril

ARBs- Valsartan- Losartan- Candesartan

3- BETA BLOCKER- AV- Node Blocker- Decrease HR- keeping HR- < 70/min

Metoprolol- Bisoprolol- Nebivolol- Carvedilol

MANGMENT

4- SA- NODE BLOCKER- Ivabradine

5- DIGOXINE-HF-with AF

6- VASODILATORS- Isosorbide- Dinitrate- Hydralazine

7- ARNI- Entresto (Sacubitril/Valsartan)

[ANGIOTENSIN-RECEPTOR –NEPRILYSIN-INHIBITORS]

8- SODIUM GLUCOSE TRANSPORTER INHIBITOR-SGLT2

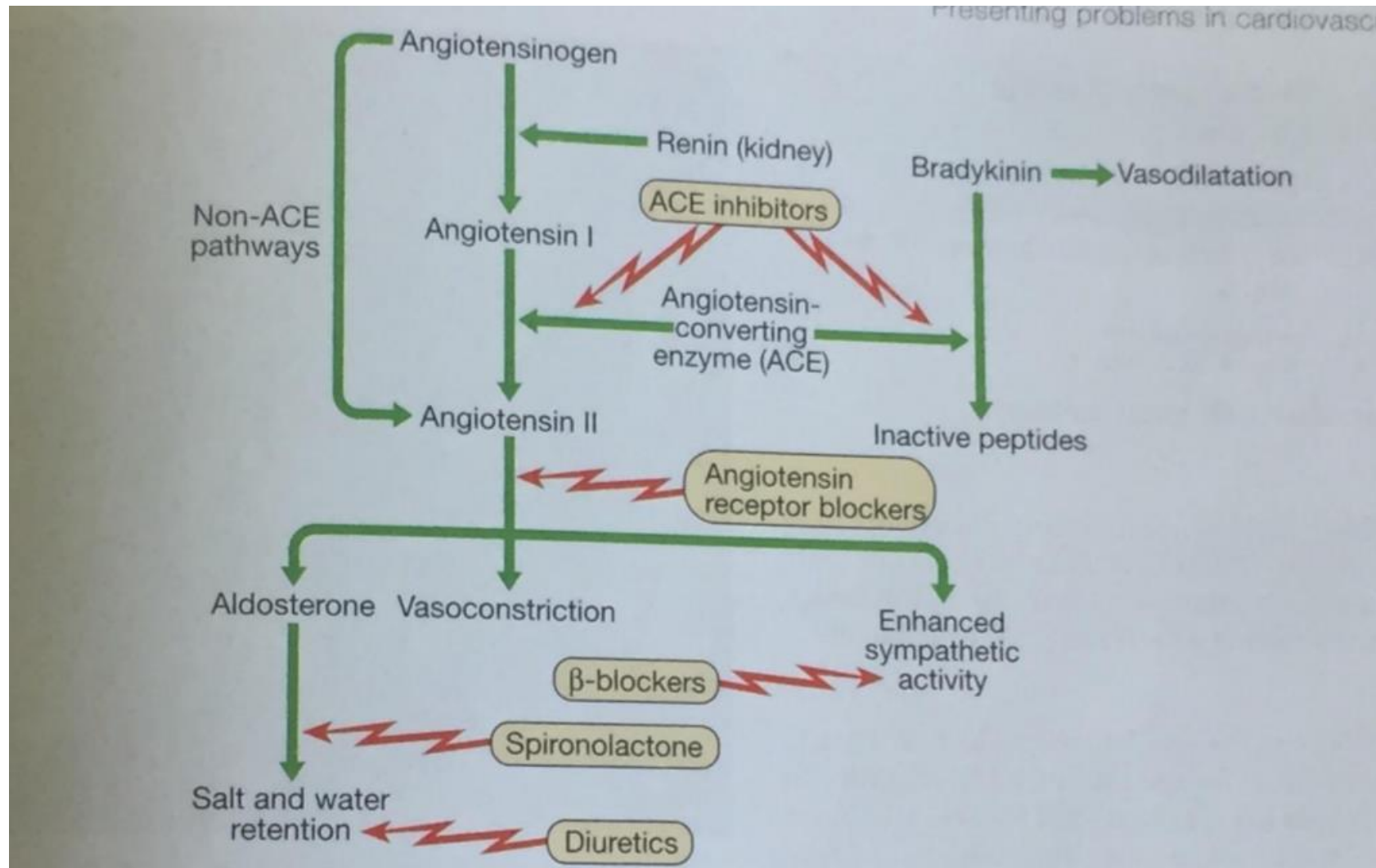
Empagliflozin- Dapagliflozin- Canagliflozin

9- TREAT ANEMIA- ferritin level $<100\text{ng/ml}$ -IV-IRON INFUSION

10- TREAT SECONDARY MR- PCI-IHD- AS-TAVI

Pharmacological treatment start slowly- 4 weeks - 6months

• 1-DIURETIC-2-ARNI-2-SGLT2-3-BB-IVABRADINE-4-MRA-5-VASODILATORS



MANGMENT

B- ELECTRICAL TRAETMENT

1-ICD

2-CRT-Cardiac Resynchronization Therapy

- Improve ventricular resynchronization – shorten ventricular activation time
biventricular pacing-BIVP- bundle of HIS-PACING in LBBB-better result

C- MECHANICAL TREATMENT

IABP- Cardiogenic Shock- enhance Coronary blood flow in Diastole

D- VENTRICULAR-ASSISTANT DEVICE- BRIDGE TO HEART TRANSPLANT

- SURGICAL-VAD - HF- young patients-refractory to medical treatment
- LV-RV- Biventricular- pulsatile- continuous
- Wide blood flow device
- COMPLICATIONS- infections-thrombosis-CVA
- CABG-MV- Repair and Replacement- AS-TAVI- HEART TRANSPLANT