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.
- World wide- HF- incidence 2 per 1000 .
- Age related disease which increases up to 90 per 100O- 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 -AFTER - LOAD-

  HTN- CALCIFIC-DEGENERATIVE-AS- BICUSPID-AS- HOCM-

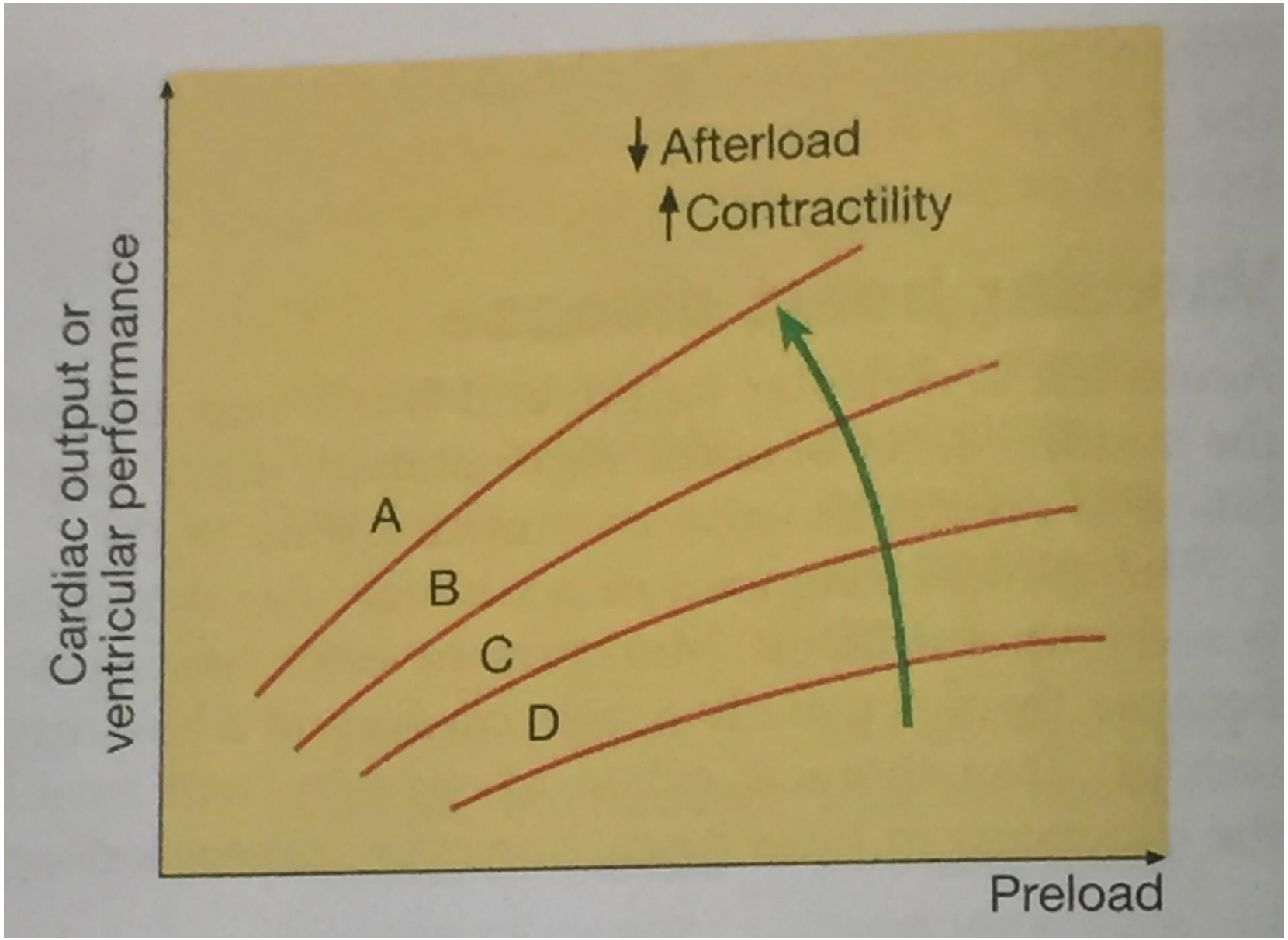
  CONG.SUB-AORTIC MEMBRAN- COARCTATION OF AORTA
- RV- PS- pulmonary embolism -PUL.HTN.
- 3-Ventricular -inflow obstruction- PRE -LOAD- MVS- TVS .
- 4-Ventricular -increased VOLUME- overload-
- AI –PI-MVR-TVR- AV-SHUNT--VSD- ASD- PDA.
- high metabolic demand- TACHYCARDIA-
- THYROTOXICOSIS- ANEMIA- PAGET S DISEASE.

- 5-ARRHYTHMIA- - TACHYARRYTHNIA- 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 DYASTOLIC FILLING-
- PERICARDIAL DISEASES- COMPRESSING THE HEART-
- PERICARDIAL TAMPONADE-
- CHRONIC CONSTRICTIVE PERICARDITIS.
- RESTYRICTIVE HEART DISEASE-
- AMYLOIDOSIS - HAEMOCHROMATOSIS.
- 7- TOXIC- ALCOHOL-CARDIOMYOPATHY- CHEMOTHERAPY-
- 8- INFECTION - MYOCARDITIS- SBE- SARCOIDOSIS-CHAGA S DISEASE.
- 9- CKD- CARDIO-RENAL SYNDROME-
- DM- DIABETIC CARDIOMYOPATHY.

PATHOPHSIOLOGY-

- 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.

- 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 VOL- ventricular dilatation- CARDIOMEGALY.
- to compensate by increase the cardiac output-
- on expanse of increased -LV-VOL. and LV- dilatation.


Afterload
Contractility
Cardiac output or ventricular performance
A
B
C
D
Preload

PRIMARY CARDIAC ABNORMALITY IN- HF-

- VENTRICULAR FUNCTION IMPAIRMENT-
- Myocardial cells loss- apoptosis- necrosis- myocardial hyaline deposition
- fibrosis- cytokines release-and free radicals.
- LOW CARDIAC OUT- 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 myocyte damage and loss
  decreases cardiac output- vicious cycle.
- 3- HIGH BNP-ANP-
- secretion by stretch volume receptors stimulation in
- ventricles and atrium- NATRIURESIS-VASODILATATION-
- high BNP- level - HF-NT- proBNP
- level >300pg/ml-sinus rhythm- or >900 pg/ml in AF.
- 4- ANTIDIURETIC – high-ADH. –VASOPRESSIN-H-WATER RETENSION.
- ISADH- cause hyponatremia in sever HF- poor prognosis.

CLASSIFICATION OF HF

- 1- NORMAL EF- variable- 52%-55%
- HFrEF- SYSTOILC HF-LOW EF

- 2-HFpEF-DYSTOLIC HF- NORMAL EF
- MYOCARDIAL INFLAMMATION- FIBROSIS- STIFNESS-
- IMPAIRED LV DIASTOLIC FILLING.
- insulin resistance- OBESITY- DM- NO REAL TREATMENT OF HF-
- ONLY TREAT UNDERLYING CAUSES.

- 3-HFmrEF- EF-40%-<55% = MEDIUM 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 MI-
- ACUTE INFECTIVE ENDOCARDITIS-
- ACUTE VALVULAR DESTRUCTION
- ACUTE RUPTURE CHORDETENDENI-MR- IN -MVP-MARPHAN SYN.
- ACUTE MYOCARDITIS-VIRAL
- ACUTE SEVER ACCELERATED AND MALIGNANT HTN-
- ACUTE DISSECTING AORTIC ANEURYSM

- 7-ACUTE ON CHRONIC HF-
- DECOMPENSATED HF-
- POOR DRUGS COMPLIANCE-
- UNCONTRLLED -HTN-DM
- INFECTION-SEPSIS
- ANEMIA
- THYROID DISEASE
- ELECTROLTES IMBALANCE- K- Mg- Ca- phos.
- ACUTE CORONARY SYNDROME -ACS- ACUTE DECOMPENSATED- IHD
- ACUTE MR-PAPILLARY MUSCLE DYSFUNCTION.
- ACUTE ONSET ARRHYTHMIA- AF
- ACUTE PULMONARY EMBOLISM

NEW YORK HEART ASSOCIATION- NYHA-

- CLASSIFICATION OF HF.
- CLASS-1- NO LIMITATION OF PHYSICAL EXERCISE.
- CLASS-2- MILD LIMIATION OF PHYSICAL EXERCISE.

CLASS-3- MODERATE LIMITATION OF PHYSICAL EXERCISE.

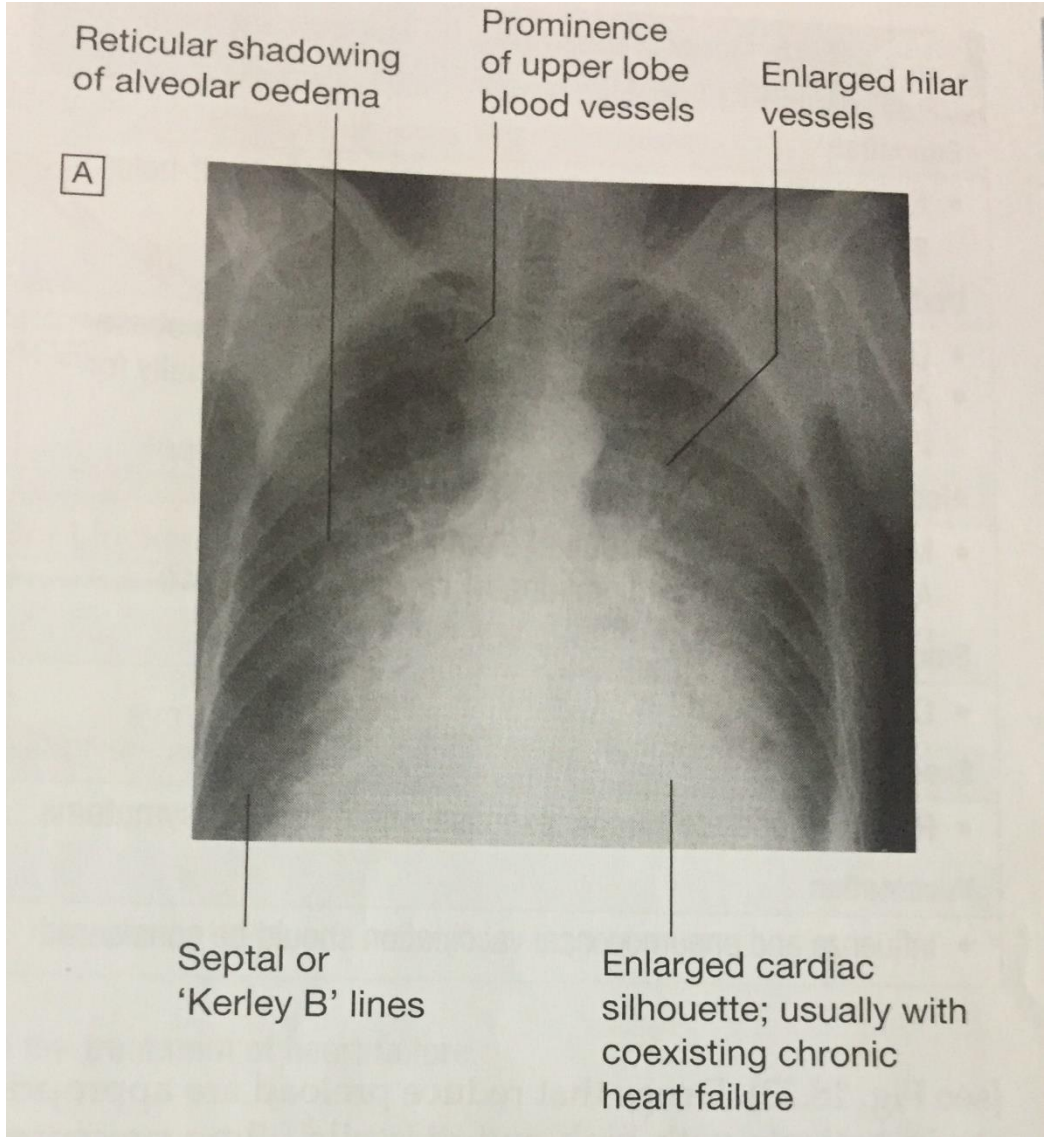
- CLASS-4- SYMTOMES OF HF-AT REST- ORTHOPNIA-PND-PUL.EDEMA.

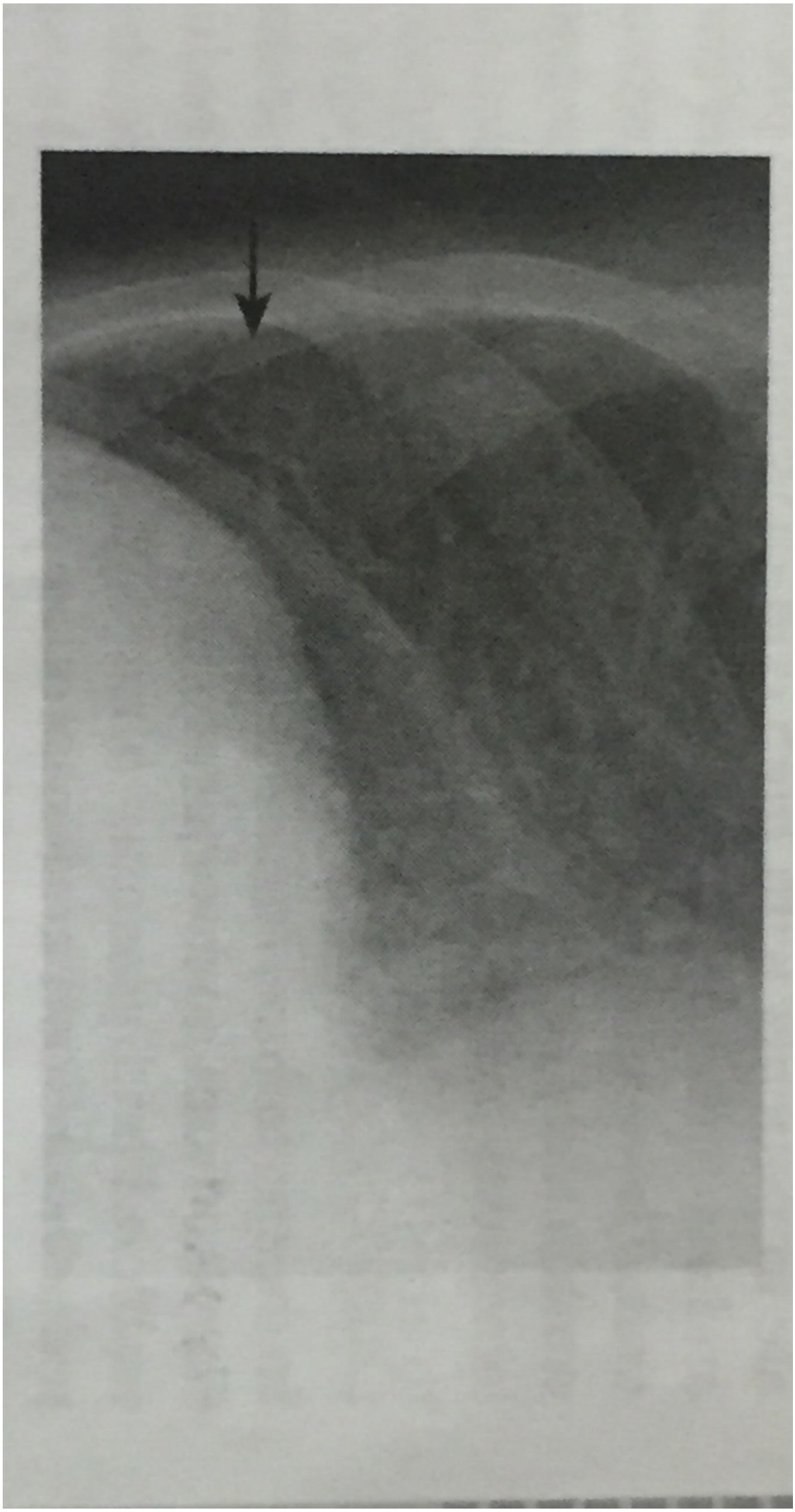
- CLASS-1-2- MILD cardiac dysfunction-
- CARDIAC OUTPUT= stroke volume by heart rate –
- is compensated and maintained by increased venous return and
- diastolic ventricular filling pressure and increase heart rate.
- CLASS-3- SEVER cardiac dysfunction-
- cardiac output only maintained by marked sinus tachycardia
- and large increase in venous return at the expanse 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 –ORTHOPENIA-acute pulmonary edema.

SYMTOMES OF HF

- 1-SOB- PAPITATION
- ON EXERTION-
- ORTHOPNEA ON LYING FLAT-AT REST.
- PND- SOB-DURING SLEEP.
- ACUTE PULMONAY EDEMA

- 2-FATIGUE- PALLOR-
- LOW CARDIAC OUT PUT- blood flow maintained only to the vital organs-BRAIN-KIDNEYS- at the expanse of the muscle and skin.


Reticular shadowing of alveolar oedema
Prominence of upper lobe blood vessels
Enlarged hilar vessels
A
Septal or 'Kerley B' lines
Enlarged cardiac silhouette; usually with coexisting chronic heart failure

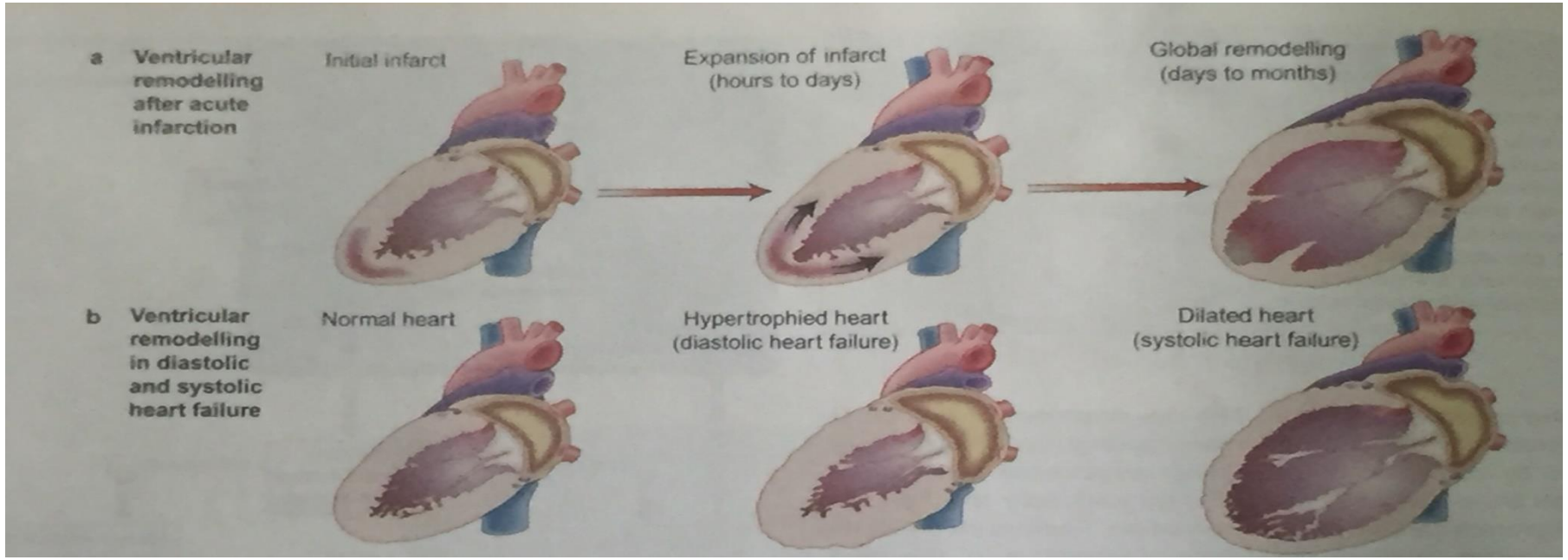


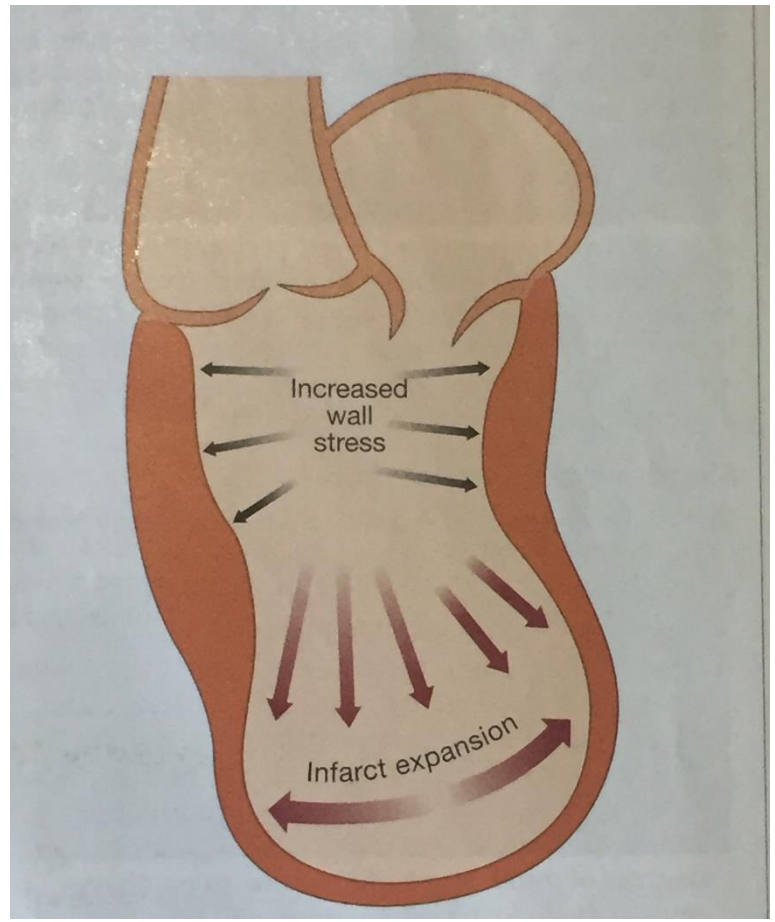
SIGNS OF- HF

- 1-HIGH-JVP
- 2-BILATERAL ANKLE- ODEMA- ASCITIS- ANASRCA.
- 3-HYPOTENSION- DIZZNESS-VERTIGO-PRE-SYNCOPY- SYNCOPY.
- 4-TACHYCARDIA- sinus tachycardia-atrial flutter- AF-
- atrial or ventricular ectopics – EXTRA-SYSTOLE
- VT-VF- CARDIAC ARREST- SUDDEN DEATH.
- BRADYCARDIA- COMPLETE-HEART BLOCK.
- 5-CX-CARDIOMEGALY-
- PLEURAL EFFUSION- LUNG CONGESTION- PULMONARY EDEMA.
- 6- 3rd-4rd – heart sounds-gallop rhythm-
- STSTOLIC MURMUR-MR- Bilateral lung basal crackles.
- 7-TENDER HEPATOMEGALY- CONGESTED LIVER- JAUNDICE.
- 8- WT.LOSS- CARDIAC CACHEXIA.

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.




Increased
wall
stress
Infarct expansion

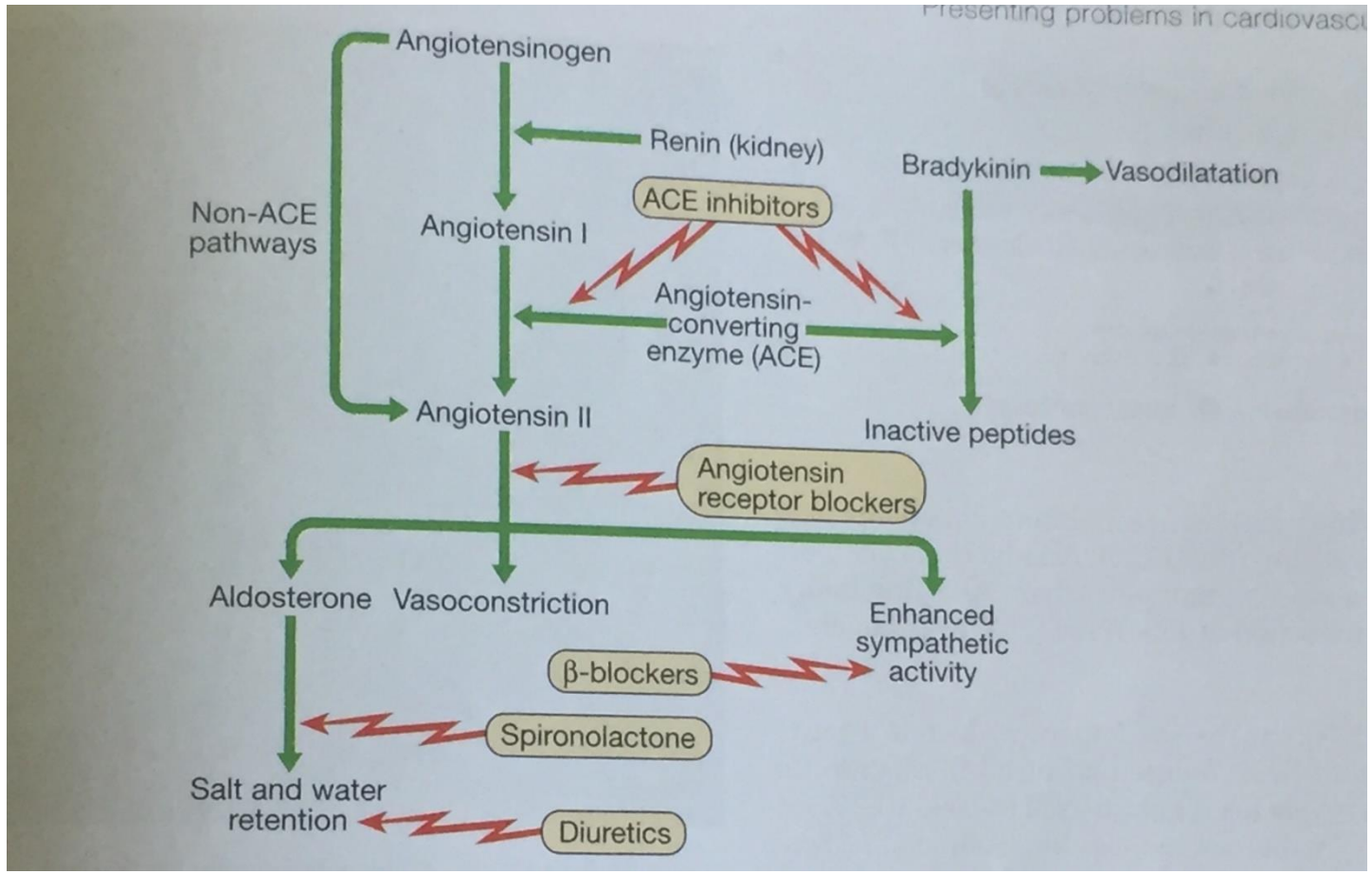
INVESTIGATIONS

- 1-ECG
- 2-CX
- 3- BLOOD TESTS- CARDIAC ENZYMES-
- NT-proBNP level >300pg/ml-sinus rhythm-or >900pg/ml –AF
- BIO. FBS-HBAIC- UREA-CREATININE-LIPIDS-T3-T4-TSH-CBC-LFT-PT-INR.
- 4- ECHO-TRANSTHORASIC- TRANSESOPHAGEAL.
- 5-HOLTER STUDY-AMBULATORY 24-hour ECG-MOINTERING
- 6-NUCLEAR ISOTOPE CARDIAC STUDY
- 7-CARDIAC CATHETERIZATION
- WITH OR WITHOUT INTERVENTION- BOTH DIAGNOSTIC-
- THERAPUTIC- IHD-PCI- AS-TAVI- MITRAL VALVE-VALVOTOMY-REPAIR
- CLOSER VSD-ASD- PDA.
- 8- MRA-CT-ANGIO-CARDIO.

MANGMENT

- ADVICE-
- CHANGE LIFE STYLE-
- INFLIUNZA- AND KORONA- VACCINATION
- TREAT THE UNDERLYING AETIOLOGY
- A- PHARMOCLOGICAL TREATMENT
- 1- DIURETICS- LOOP DIURETICS- FRUSEMIDE- HYDROCHLORTHIAZIDE-
- MRA- K-SPAIRING DIURETICS-
- ALDOSTERONE RECEPTORS ANTIGONIST-
- SPIRONLACTONE-EPLERENONE.
- 2- ACEI-ENALOPRIL-LISNOPRIL-RAMIPRIL
- ARBS-VALSARTAN-LOSARTAN-CANDESRTAN-

- 3- BETA BLOCKER- AV- NODE BLOCKER- DECREASE HR-
- METOPROLOL-BISOPROLOL- NEBIVOLOL- CARVIDOLOL-
- KEEPING HR- < 70/min.
- 4- SA- NODE BLOCKER- IVABRADINE
- 5- DIGOXINE-HF-with AF
- 6- VASODILATORES- ISOSORBIDE-DINITARATE- HYDRALAZINE
- 7- ARNI- ENTERSTO- SACUBITRAL/VALSARTAN-
- ANGIOTENSINE-RECEPTAR –NEPRILYSIN-INHIBITORS.
- 8- SODIUM GLUCOSE TRANSPORTER INHIBITER-SGTI2-
- - EMPAGLIFLOZEN- DAPAGLIFLOZEN-CANAGLIFLOZIN
- 9-TREAT ANEMIA- ferritin level <100ng/ml-IV-IRON INFUSION.
- 10-TREAT SECONDARY MR- PCI-IHD- AS-TAVI- .
- -PHARMCOLOGICAL -TREATMENT-START-SLOWLY-4-WEEKS - 6MONTHS-
- 1-DIURETIC-2-ARNI-2-SGTI2-3-BB-IVAPRADINE-4-MRA-5-VASODILATORS


Angiotensinogen
Renin (kidney)
Bradykinin
Vasodilatation
ACE inhibitors
Non-ACE pathways
Angiotensin I
Angiotensin-converting enzyme (ACE)
Angiotensin II
Inactive peptides
Angiotensin receptor blockers
Aldosterone
Vasoconstriction
Enhanced sympathetic activity
β-blockers
Spironolactone
Salt and water retention
Diuretics

- B- ELECTRICAL TRAETMENT
- 1-ICD
- 2-CRT-CARDIAC RE-SYNCHRANISATION THERAPY-
- improve ventricular re-synchranisation-shorten ventricular activation time-biventricular pacing-BIVP- bundle of HIS-PACING in LBBB-better result
- C- MECHANICAL TREATMENT
- IABP-CARDIOGENIC SHOCK-ENHANCE COR.BLOOD FLOW IN DIASTOLE
- D- VENTRICULAR-ASSISTANT DEVICE- BRIDGE TO HEART TRANSPLT
- SURGICAL-VAD - HF- young patients-refractory to medical treatment.
- LV-RV- BIVENTICULAR-PULSATILE-CONTINOUS.
- WIDE BLOOD FLOW DEVICE-
- complications-infections-thrombosis-CVA.
- CABG-MV- REPAIR AND REPLACEMENT-AS-TAVI-HEART TRANSPLANT