Heart Failure

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Definition & Types

AHA defines heart failure as complex clinical syndrome that results in structural or functional impairment of ventricular filling or ejection of blood, this leads to cardinal clinical manifestation such as dyspnoea and fatigue, edema, rales.

Types

HFrEF AKA (Systolic Failure)

HFpEF AKA (Diastolic Failure)

Etiology HFrEF

TABLE 252-1 Etiologies of Heart Failure

Depressed Ejection Fraction (<40%)	
Coronary artery disease	Nonischemic dilated cardiomyopath
Myocardial infarction[a]	Familial/genetic disorders
Myocardial ischemia[a]	Infiltrative disorders[a]
Chronic pressure overload	Toxic/drug-induced damage
Hypertension[a]	Metabolic disorder[a]
Obstructive valvular disease[a]	Viral
Chronic volume overload	Chagas' disease
Regurgitant valvular disease	Disorders of rate and rhythm
Intracardiac (left-to-right) shunting	Chronic bradyarrhythmias
Extracardiac shunting	Chronic tachyarrhythmias
Chronic lung disease	
Cor pulmonale	
Pulmonary vascular disorders	

Index Event

Pathogenesis

Loss of function of myocytes

Inability of the Myocardium to generate force

Compensatory Mechanism
RAAS

Increased Myocardial contractility
Vasodilatory Molecule

LV Remodelling

LV Remodelling

It refers to the changes in LV Mass, Volume, Shape & Composition after a cardiac injury

High End Diastolic Wall Stress

Myocyte Hypertrophy

Alteration in Contractility

Progressive loss of myocyte

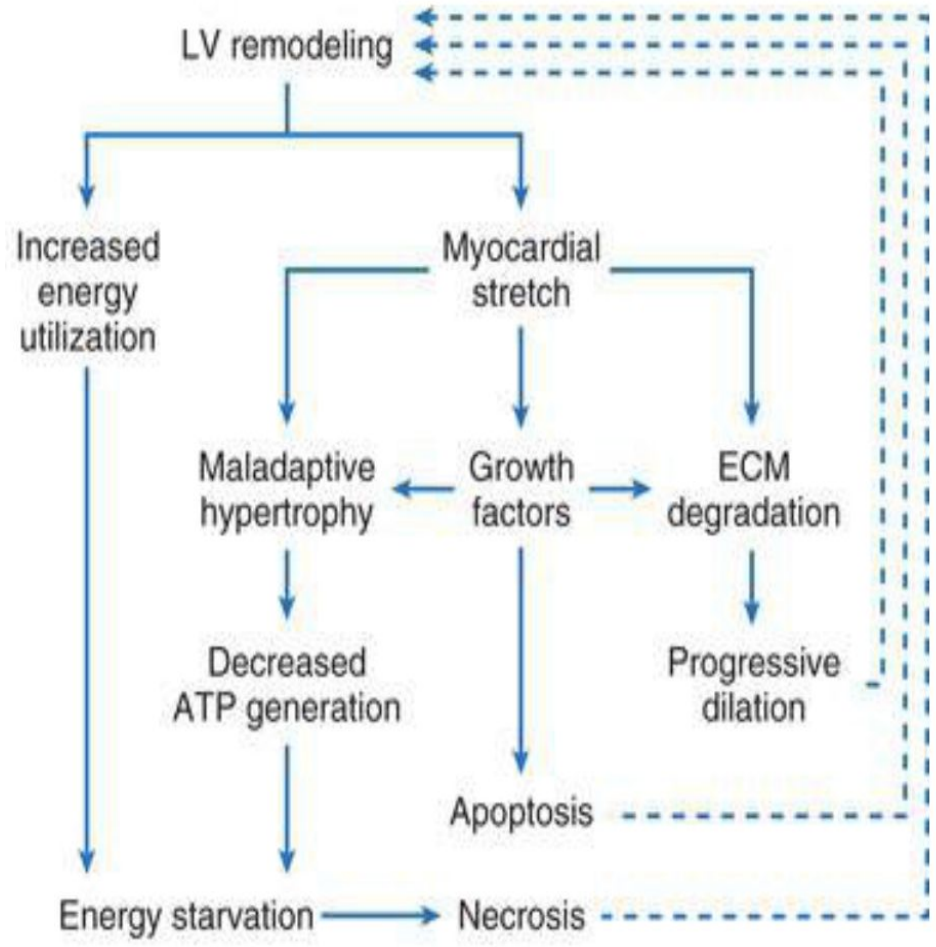
Alteration in Energy Mechanism

Extracellular matrix reorganization

TABLE 252-3 Overview of Left Ventricular Remodeling

Alterations in Myocyte Biology
Excitation-contraction coupling
Myosin heavy chain (fetal) gene expression
β-Adrenergic desensitization
Hypertrophy
Myocytolysis
Cytoskeletal proteins
Myocardial Changes
Myocyte loss
Necrosis
Apoptosis
Autophagy
Alterations in extracellular matrix
Matrix degradation
Myocardial fibrosis
Alterations in Left Ventricular Chamber Geometry
Left ventricular (LV) dilation
Increased LV sphericity
LV wall thinning
Mitral valve incompetence

LV Remodelling



After Effects

Increased Wall Stress

Afterload Mismatch

Episodic Subendocardial Hypoperfusion

Increase in oxygen demand

Functional Mitral regurgitation

Worsening Overload

Maladaptive Signals

Maladaptive Gene Expression

Clinical Features

Symptoms

- Fatigue
- Dyspnoea, Tachypnoea
- PND, Orthopnoea
- Cough
- Edema
- Abdominal Distension
- Somnolence

Signs

- Low volume Pulse
- Pulses Alternans
- Cold Peripheries
- Anasarca
- Tachy, APC, VPC
- Tachypnoea
- JVP
- Crepts or Wheeze
- Down & Out, Sustained AI
- Parasternal Lift
- Cardiac Extrasound (S3)
- TR or MR
- Hepatomegaly

Edema

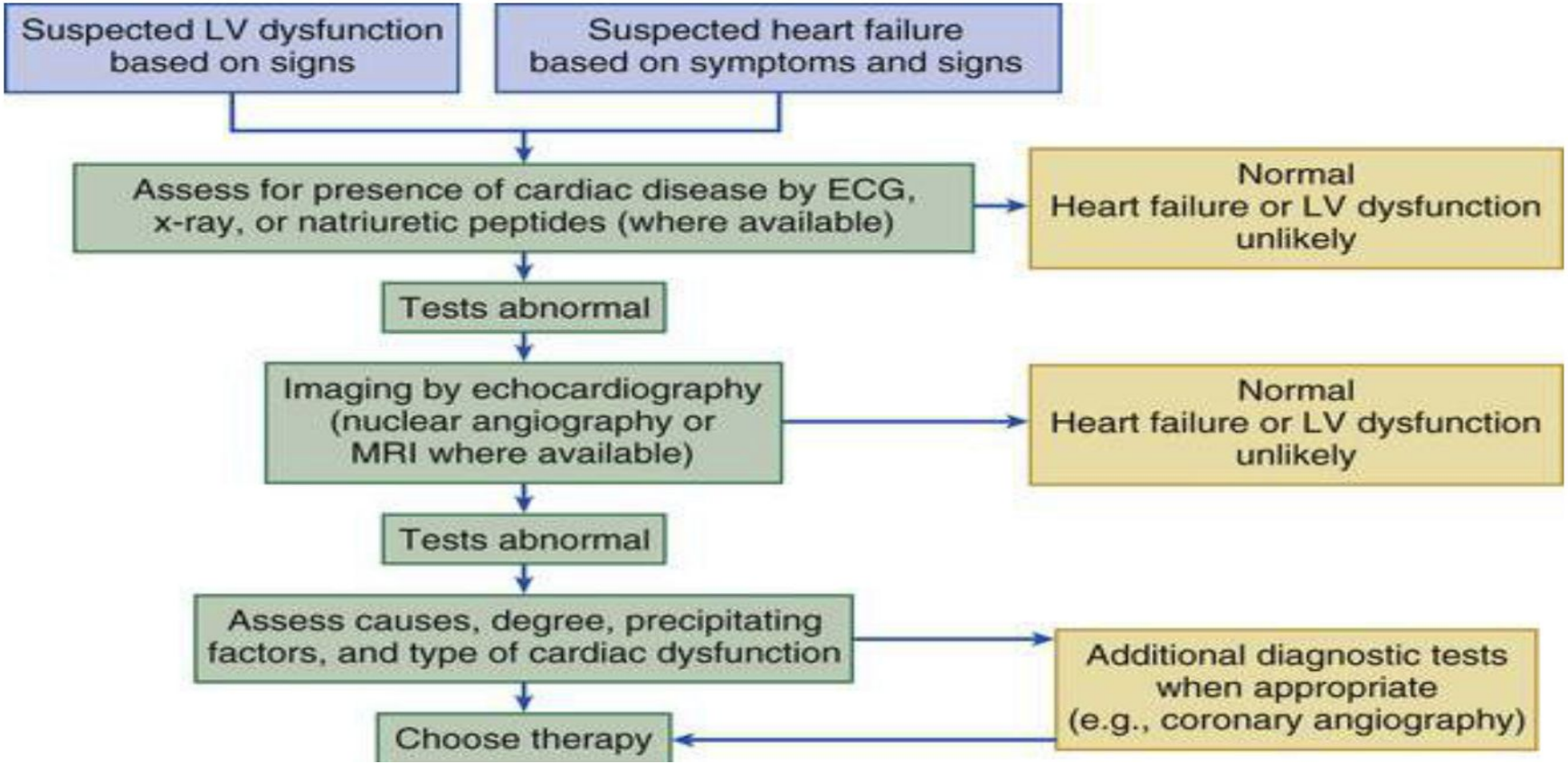
- Ascites
- Presacral
- Pedal
- Chronic venous stasis

Diagnosis

FRAMINGHAM CRITERIA		
Major Criteria	**Minor Criteria**	**Major or Minor Criteria**
Paroxysmal nocturnal dyspnea or orthopnea Neck vein distention Rales Cardiomegaly Acute pulmonary edema S_3 gallop Increased venous pressure >16 cm H_2O Hepatojugular reflux	Ankle edema Night cough Dyspnea on exertion Hepatomegaly Pleural effusion Vital capacity decreased One-third from maximal capacity Tachycardia (rate >120/min)	Weight loss >4.5 kg in 5 days in response to treatment

- 2 Major or 1 Major or 2 Minor is required for diagnosis of Heart failure.
- In absence of any other systemic illness

Diagnosis


Suspected LV dysfunction based on signs
Suspected heart failure based on symptoms and signs
Assess for presence of cardiac disease by ECG, x-ray, or natriuretic peptides (where available)
Normal
Heart failure or LV dysfunction unlikely
Tests abnormal
Imaging by echocardiography (nuclear angiography or MRI where available)
Normal
Heart failure or LV dysfunction unlikely
Tests abnormal
Assess causes, degree, precipitating factors, and type of cardiac dysfunction
Additional diagnostic tests when appropriate (e.g., coronary angiography)
Choose therapy

Bio-Marker

Inflammation[*][‡]
C-reactive protein
Tumor necrosis factor
Fas (APO-1)
Interleukins 1, 6, and 18
Oxidative Stress[*][†][§]
Oxidized low-density lipoproteins
Myeloperoxidase
Urinary biopyrrins
Urinary and plasma isoprostanes
Plasma malondialdehyde
Extracellular Matrix Remodeling[*][§]
Matrix metalloproteinases
Tissue inhibitors of metalloproteinases
Collagen propeptides
Propeptide procollagen type I
Plasma procollagen type III
Neurohormones[*][†][§]
Norepinephrine
Renin
Angiotensin II
Aldosterone
Arginine vasopressin
Endothelin
Myocyte Injury[*][†][§]
Cardiac-specific troponins I and T
Myosin light-chain kinase I
Heart-type fatty acid protein
Creatine kinase MB fraction
Myocyte Stress[†][‡][§][¶]
B-type and N-terminal pro–B-type natriuretic peptide
Midregional proadrenomedullin
ST2
New Biomarkers[†]
Chromogranin
Galectin 3
Osteoprotegerin
Adiponectin
Growth differentiation factor-15

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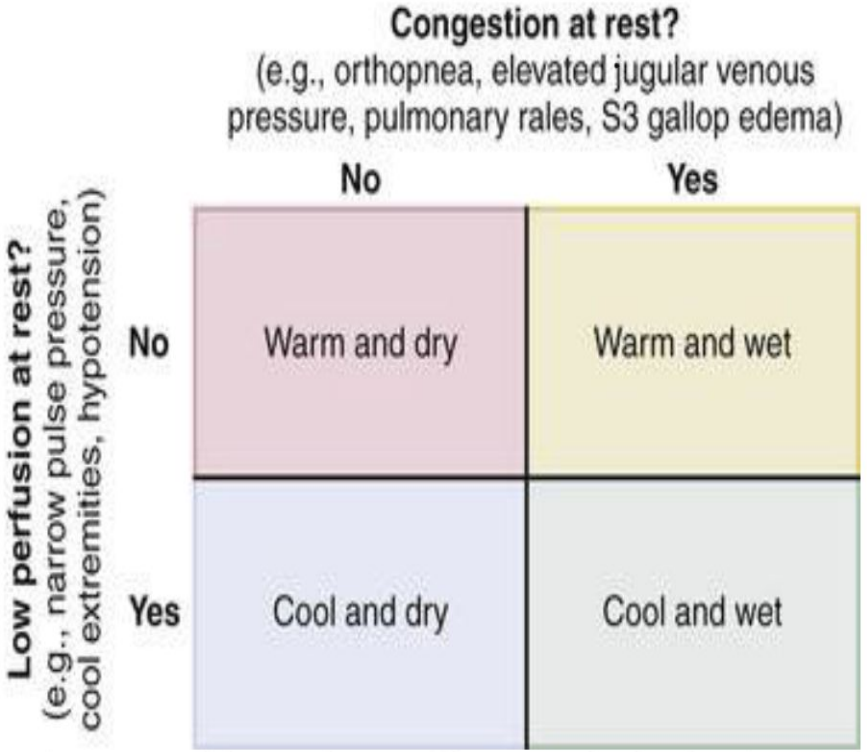
Functional Classification, Clinical Classification

TABLE 252-2 New York Heart Association Classification

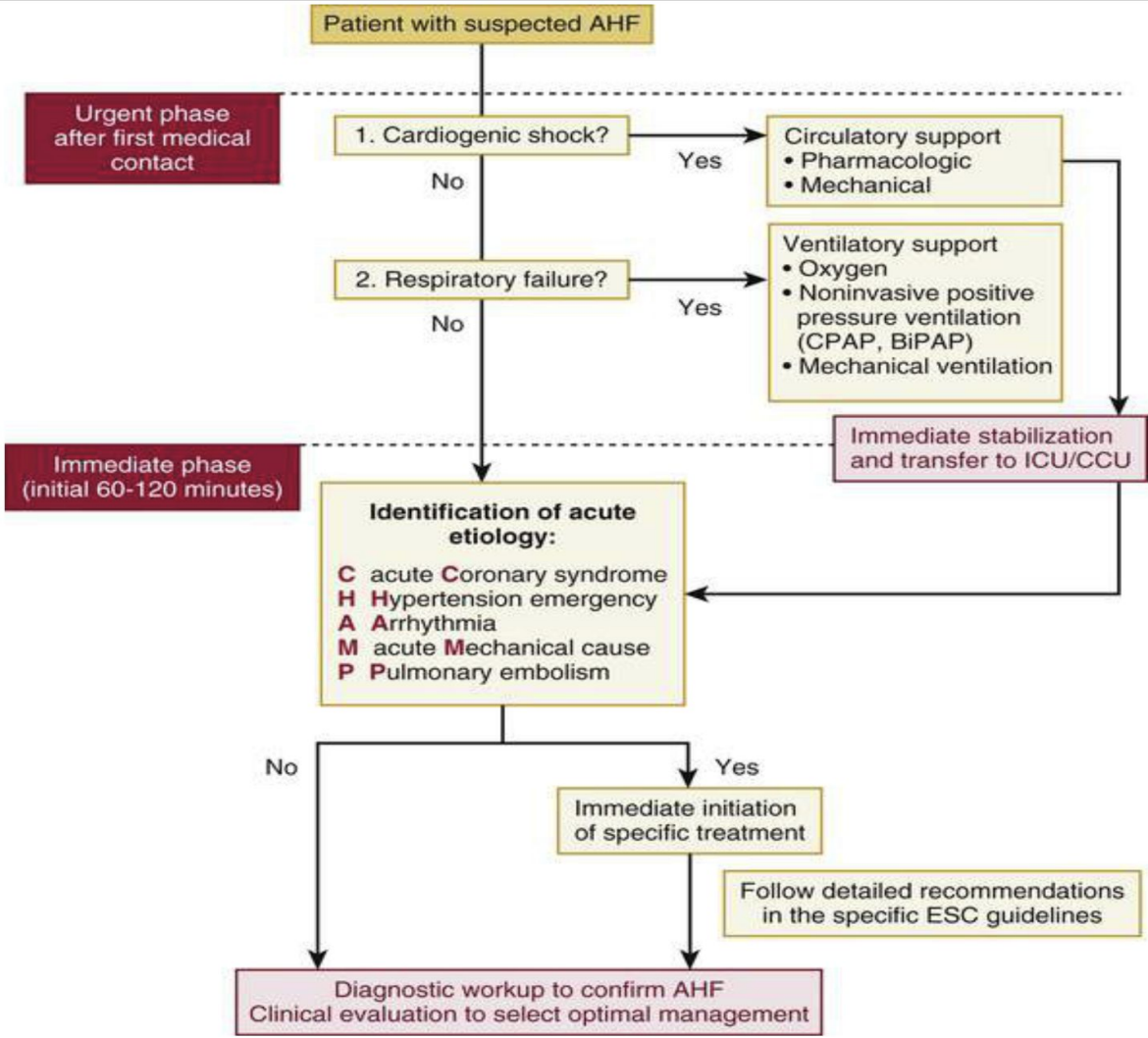
FUNCTIONAL CAPACITY	OBJECTIVE ASSESSMENT
Class I	Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, dyspnea, or anginal pain.
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.



Management

Cause of Acute Heart Failure

- Decompensated HF
- Acute hypertensive failure
- Cardiogenic shock



Pharmacological

- Diuretics
- Vasodilator Therapy
- Inotropes and Inodilators
- PDE Inhibitors
- Vasopressors
- Others
    - Digoxin,AVP Antagonist, CCB
- Novel agents

Non Pharmacological

- Ultrafiltration
- Hypertonic Saline

Diuretics

Furosemide (20 to 160 mg; 5 to 40 mg/hr)

Bumetanide(0.5 to 4 mg; 2 to 4 mg/hr)

Torsemide(20 to 100 mg; 5 to 20 mg/hr)

If refractory

HCTZ(25 to 50 mg BD)

Metolazone(2.5 to 10 mg OD)

Spironolactone(25 to 50 mg)

Vasodilators

1. **NTG**(20 to 400 mcg/min)
2. **ISDN**(1 to 10 mg/kg)
3. **Nitroprusside**(0.3mcg/kg to 4 mcg/kg)
4. **Nesiritide**(2 mcg/kg; 0.010-0.030 mcg/kg/min)
5. **Noval Agents**
    a. Serelaxin,
    b. Ularitide,
    c. Aliskarine,
    d. Tazosetan,
    e. Cinciguat, Variciguat

Inotropes

- Dobutamin (2 to 20 Mcg/min/kg) [B 1&2, Var A]
- Dopamine(2 to 4 mcg/min/kg; 5 to 20 mcg/kg/min)[D, B, A]
- Epinephrine(0.05 to 0.5 mcg/kg/min) [B]
- Nor-Epinephrine(0.2 to 1.0 mcg/kg/min) [B1,A1,b2]

Phosphodiesterase

- Milrinone(25 to 75 mcg/kg bolus; 0.10 to 0.75 mcg/kg/min)
- Enoximone(1.25 to 7.5 mcg/kg/min)
- Levosimendan(12 to 24 mcg/kg; 0.5 to 2 mcg/kg/min)

Noval

Omecamtiv mecarbil, Istaroxime

Other Pharmacological Therapy

Digoxin

AVP Antagonist

- Tolvaptane
- Conivaptane

CCB (Nicardipin, Clevidipine)

Non Pharmacological Therapy

- Ultrafiltration
  - Its a method of removing Na & H2O
  - Greater salt removal
- Hypertonic Saline 3%
- NIV

Heart Failure with Reduced Ejection Fraction

Methods to decrease volume overload

- Diet & Fluid
- Diuretics
  - Loop, Thiazid, MRA, Carbonic anhydrase inhibitors, SGLT2I, AVPI

Prevention of disease progression

- ACEI, ARB, ARNI, MRA, Beta blocker,
- Others
  - Hydralazine/ISDN
  - Digoxin
  - Ivabradine

Devices

Diet

- Sodium 2 to 3 gms
- Fluid restriction to be considered in
  - Hyponatremia
  - Despite diuretics, difficult to control fluid status
- Cardiac cachexia
  - Caloric supplementation
    - Caloric support and nitrogen balance
  - Steroids not used

Diuretics

Loop Diuretics

Furosemide, Bumetanide and torsemide

Thiazides

Chlorthalidone, Chlorothiazide, Hydrochlorothiazide, Metalazone

Mineralocorticoid Receptor Antagonist

Spironolactone, Eplerenone

Novel agents

Non steroidal - Finerenone

Finerenone	ARTS HF	30% NT PRO BNP decrease than Eplerenone.

Other diuretics - Amiloride, Triamterene

Carbonic Anhydrase inhibitor (acetazolamide)

SGLT2 I(Cana, Empa, Dapa)

Vasopressin Antagonist(tol, lixi, sata, Coni)

Prevention Of Progression

ACEI(Capto, Enala, Lisno, Rami, Fosino, Quina, Trandolapril)

ARB(Val, Cande, Losartan)

ARNI(Valsartan/sacubitril)

Beta Blocker(Carvedilol, Bisoprolol, Metaprolol, Nebivolol)

MRA(Spironolactone, Eplerenone)

Others

- Hydralazine/ISDN
- Digoxin
- Ivabradine

ARNI

Combo of AT1 Receptor Antagonist & Neprilysin Inhibitor

Decreases NP, Bradykinin and adrenomedullin degradation

Enhances Diuresis, Natriuresis, Myocardial Relaxation

Decreases Renin and Aldosterone Levels

Angiotensin Receptor Neprilysin Inhibitor		
Sacubitril/valsartan	24 mg/26 mg twice	97 mg/103 mg twice

FUNNY CURRENT CHANNEL inhibitor

SHIFT	Ivabradine	DEC in CV death and HF hospitalization by 18%
BEAUTIFUL	IVABRADINE	Didn’t meet primary end point

Renin Inhibitor

Failed to improve outcome in ASTRONAUT, ATMOSPHERE

Symptomatic after Treatment

Glycosides(NaKATPase, Inc Intracellular Ca)
N-3 PUFA

Digoxin Side Effect
Toxicity (High levels, Low Levels due to hypokalemia or hypomagnesemia)
- Arrhythmias(Ectopic, High grade AV Block)
- Neuro - Visual disturbance, Disorientation, Confusion
- GIT - Anorexia, Nausea, Vomiting

Treatment - Fab fragment

Device Therapy

Dyssynchrony

Its is defined by a prolonged QRS complex (>120ms) on surface ECG mostly due to BBB

Alteration in timing and pattern of ventricular conduction, Paradoxical septal motion

Reduction in ventricular contractility, prolong MR

TYPES

ICD (Intracardiac Defibrillator)

CRT (Cardiac Resynchronization therapy)

VAD (Ventricular Assist Devices)

CRT

INDICATIONS(Revised CRT guidelines)

Mod - LVef < 35%, Sinus Rhythm, LBBB with QRS>120ms, NYHA 2, 3, 4.
Strong - QRS>150 with or without HF with LBBB

LIMITATION

Success rate 88 to 92%, Risk of dissection or Perforation of coronary Sinus

ICD

INDICATIONS

Non ischemic DCM LVEF < 35%,
IHD(after 40 days) LVEF < 35%,
NYHA 2 or 3 Symptomatic after OMT

Terminology Describing Characteristics of Mechanical Circulatory Support (MCS) Devices

Pump Location
Extracorporeal: Pump located outside the body
Paracorporeal: Pump located outside but adjacent to the body
Intracorporeal: Pump implanted within the body
Orthotopic: In the normal position of the heart (TAH)
Ventricle Supported
LV support (LVAD)
RV support (RVAD)
Biventricular support (BiVAD)
Biventricular replacement (TAH)

INDICATION

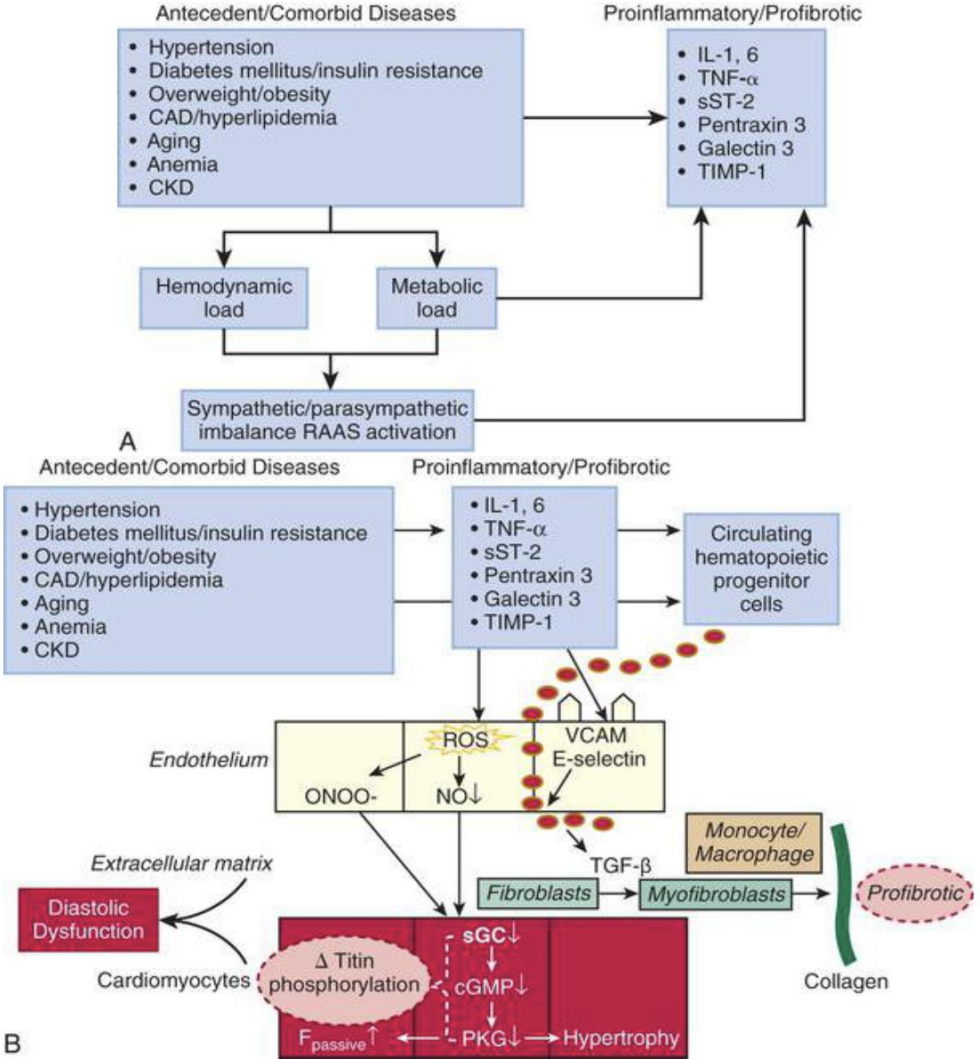
- Bridge to Therapy
- Bridge to Transplant
- Destination therapy

No particular hemodynamic criteria
But guideline Recommendation
BP<90
Resting Tachycardia
PCWP>20
Oliguria
Rise in creatinine and SGOT, SGPT
Mental state changes
Cool Extermities

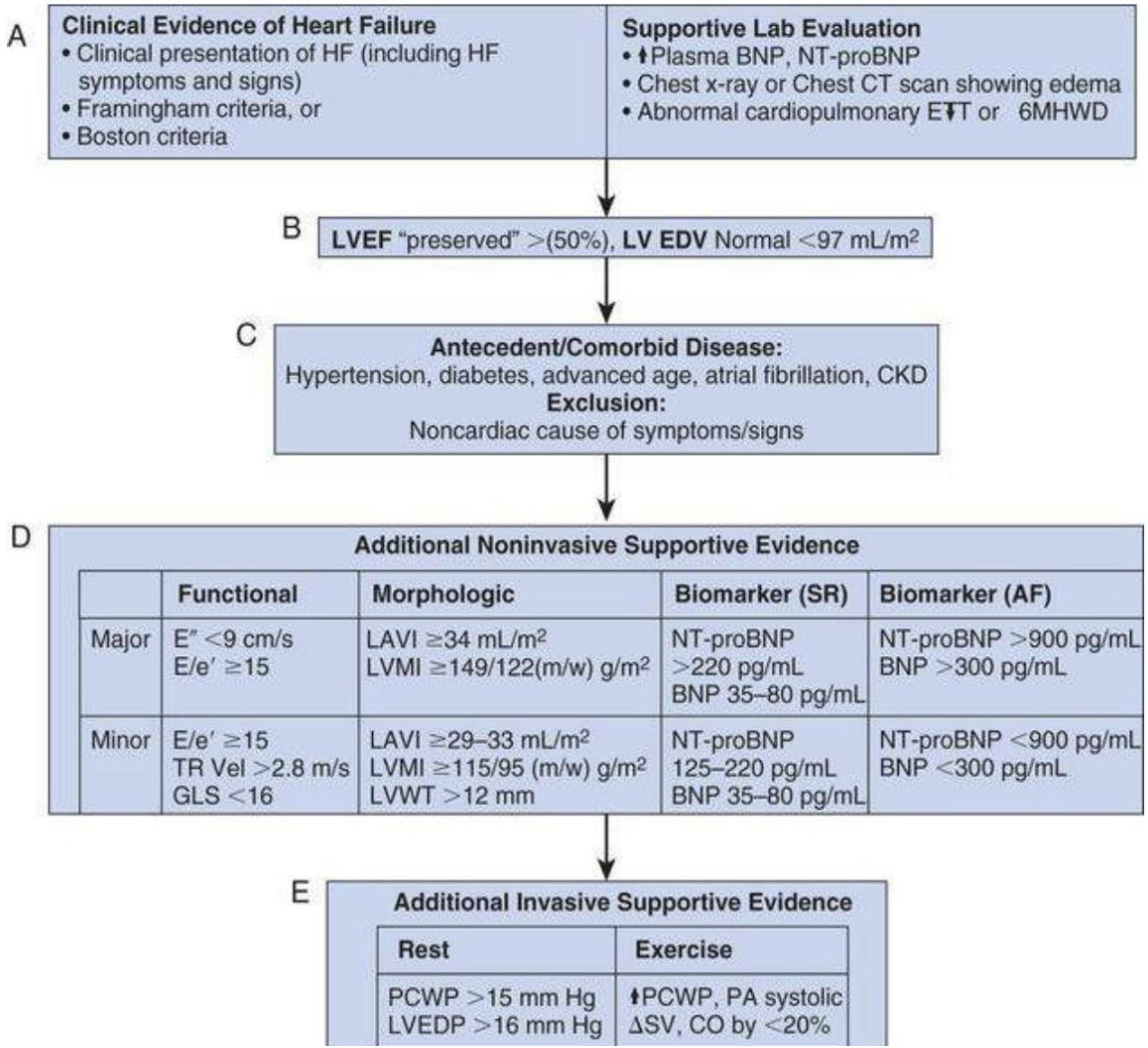
HFpEF Etiology

Preserved Ejection Fraction (>40–50%)	
Pathologic hypertrophy	Restrictive cardiomyopathy
Primary (hypertrophic cardiomyopathies)	Infiltrative disorders (amyloidosis sarcoidosis)
Secondary (hypertension)	Storage diseases (hemochromatosis)
Aging	Fibrosis
Endomyocardial disorders	

Pathogenesis



Diagnostic Criteria



Therapy

DIG	Digoxin	No alteration in HF hospitalization, CV mortality
CHARM	Candesartan	No impact on mortality and morbidity
PEP-CHF	Perindopril	No change in primary endpoint Results
I PRESERVE	Irbesartan	No effect on mortality and morbidity
SENIORS	Nebivolol	No mortality benefit
TOPCAT	MRA	No significant change in patient parameters
RELAX	Sildenafil	No exercise capacity improvement
NEAT	Nitrates	Decrease in quality of life

Assisted Devices

Implantable hemodynamic monitor
It helps in monitoring the
- Volume status
- Heart rate
- Rhythm
- Sympathetic tone
- Serum and Plasma Biomarker

RCT
- COMPANION
- Estimated Pul Art Diastolic pressure
- 152 % DECEASE IN PAP
- 52% REDUCTION IN HOSPITALIZATION

Surgical Management

CABG
VALVE REPLACEMENT
VENTRICULAR RECONSTRUCTION

Transplant

Indication

Cardiogenic Shock on high dose vasopressin or on VAD

Stage D refractory HF on maximal therapy

Recurrent life threatening Arrhythmias despite max intervention and implanted defib

Refractory angina not able to revascularize

Stemcell therapy