*"He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all"*
William Osler

# Medicine

# 430

# Heart Failure

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# Heart Failure

## Definition:

Heart failure is a complex clinical syndrome in which an abnormality of cardiac structure or function is responsible for the inability of the heart to eject or fill with blood at a rate corresponding with the requirements of the metabolizing tissues.

## Prevalence:

Since HF is more common in the elderly, its prevalence is likely to continue to increase as the population ages.
1/3 of HF cases in KSA have diastolic HF

## Causes:

Cardiac causes:

- Pericardium
  - Effusion (quick filling of heart could cause cardiac tamponade)
- Myocardium:

1) MI:
   Dyskinetic segments contracting poorly>Distorts contraction and relaxation patterns
2) Acute Myocarditis (fever-severe pain)
   Usually preceded by flu-like illness>Inflammation>Progressive ventricular dilatation - theory: Receptors present in the myocardium in some individuals are already genetically susceptible to this so when they get a viral infection (ex coxsackie virus), they develop HF.
3) Cardiomyopathy

- Endocardium:
  - Endocarditis: Infection in valve – vegetations – degenerative calcification of aortic valves in elderly (AS) – valvular heart disease

Cardiomyopathy:

a. -Dilated CM: Heart is enlarged but when catheterization is performed the coronary arteries are found to be normal – hormonal response. Could be related to pregnancy – preceded by viral infection
b. -Restricted CM: The heart is not enlarged. The problem lies in the amount of interstitial tissue in between the myocytes. Ex: Sarcoidosis (CT disease) in which there's ↑ deposition of fibrous tissue in myocardium causing restriction on the heart to fill properly (DHF)
c. -Hypertrophic CM: Could be congenital in which the heart is thicker than normal.

Non-Cardiac causes:

- Pulmonary (Rt Sided HF):
  - Interstitial Lung Disease
  - Pulmonary Embolism
  - >>Cor Pulmonale: Rt sided HF secondary to lung diseases – describes no problems in Lt side of the heart – Rt sided affected (sleep apnea- snoring)

Most cases of Rt sided HF are due to Lt sided HF

High Output Heart Failure Causes:

- Chronic Edema
- Pregnancy
- Hyperthyroidism
- AV fistulas
- Wet beriberi (caused by thiamine "vit. B1" deficiency
- Paget's disease of the bone
- Mitral Regurgitation
- Aortic Insufficiency

*There conditions rarely cause heart failure by themselves. However, if these conditions develop in the presence of underlying heart disease, heart failure can result quickly

- High-Output Heart Failure
- Thyroid:
  - In severe cases
- Uncontrolled HTN
- Renal:
  - Artery stenosis
- Arrhythmia:
  - Tachycardia: not enough time for the ventricle to fill – low CO
  - Complete heart block: limits CO even if SV is normal

MOST COMMON CAUSES:

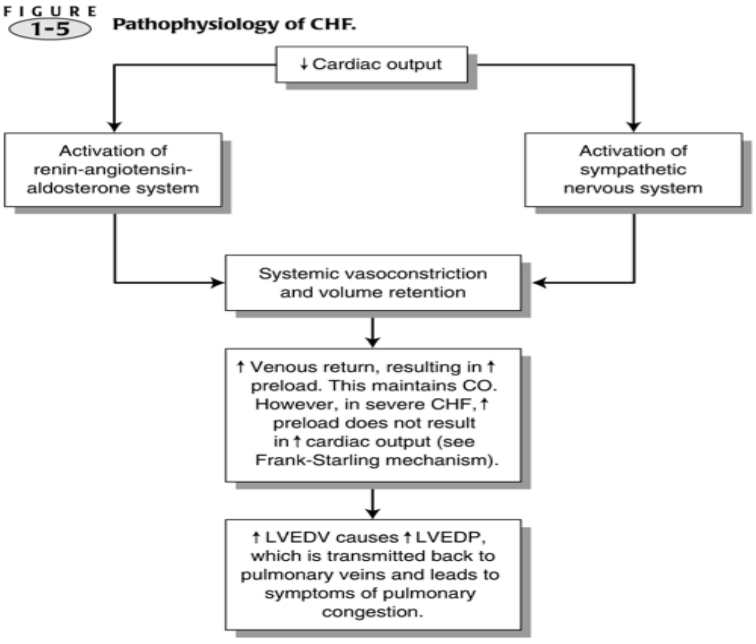
1) CAD/IHD "MI"
2) Uncontrolled HTN
3) Valvular HD

Most common cause of Lt sided HF: IHD
Most common cause of Rt sided HF: Lt sided HF

## Pathophysiology:

The heart requires adequate blood supply (Coronary arteries), proper electrical conduction and valve function. So any problems in any of the following could eventually lead to HF.

Any fall in CO activates counter-regulatory neurohumoral responses (compensatory) that would support cardiac function in physiologically normal conditions. However, if these mechanisms occurred in the setting of impaired ventricular function they will lead to deleterious increase in both preload and afterload. This will initially be beneficial to the heart but in the long run, it becomes harmful because any additional fall in CO will further stimulate this neurohumoral response and ↑ peripheral vascular resistance, thus, creating a vicious cycle.




(Basis of Starling's Law) Cardiac Output is a function of:

- Preload- Volume and pressure before contraction (at end of diastole)
- Afterload- Volume and pressure after contraction (during systole)
- Myocardial contractility

## Compensatory Mechanisms:

1) Stimulation of RAAS: (mediated by angiotensin 2)
    - Salt & water retention
        - Aldosterone
        - Endothelin-1
        - ADH (in severe cases): Lower Limb edema, pulmonary edema
    - VC: preserving even more fluids
    - Sympathetic nervous system stimulation (because of ↓ CO & SV so heart compensates by ↑ HR, CO=SV x HR)
        - ↑ myocardial contractility
        - ↑ HR
        - Peripheral VC

        This initially maintains cardiac output. However, prolonged sympathetic stimulation leads to cardiac myocyte apoptosis, hypertrophy and focal myocardial necrosis.

2) Atrial Natriuretic Peptides: (released in response to atrial stretch)
    - Act as antagonists to fluid conserving effect of Aldosterone

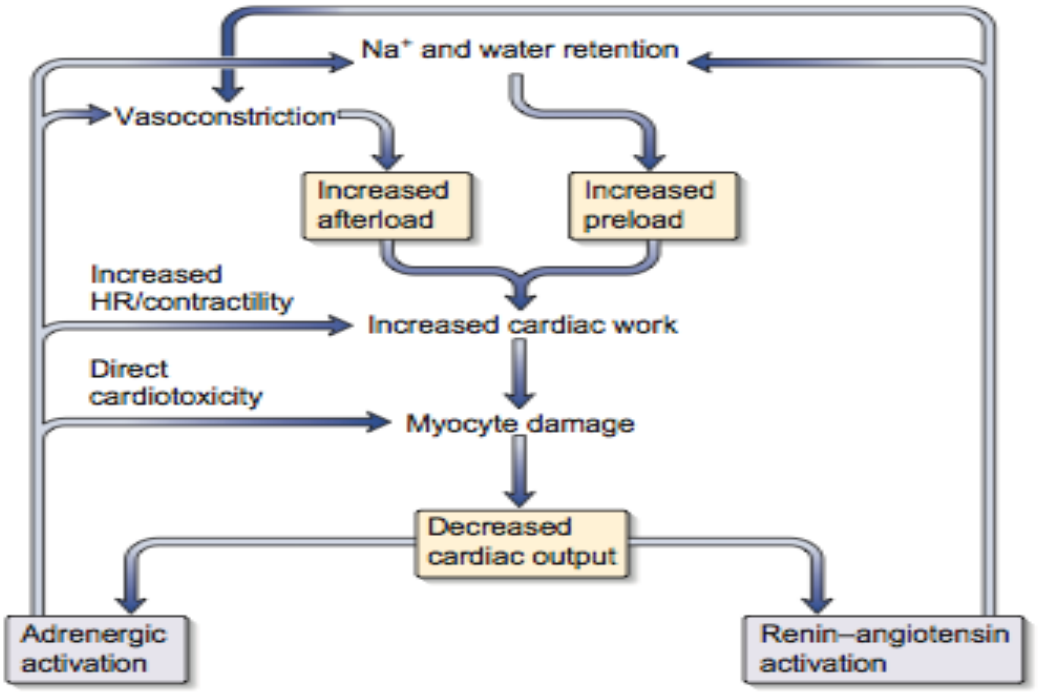



***Fig. 13.57*** **The compensatory physiological response to heart failure.** Chronic activation of the renin–angiotensin and adrenergic systems results in a 'vicious cycle' of cardiac deterioration that further exacerbates the physiological response.

## Cardiac Remodelling:

- After MI:
  - Infarcted Segment>Dilated, thinned and expanded
  - Non-Infarcted Segment> Hypertrophied

This leads to further deterioration in ventricular function and worsening HF.

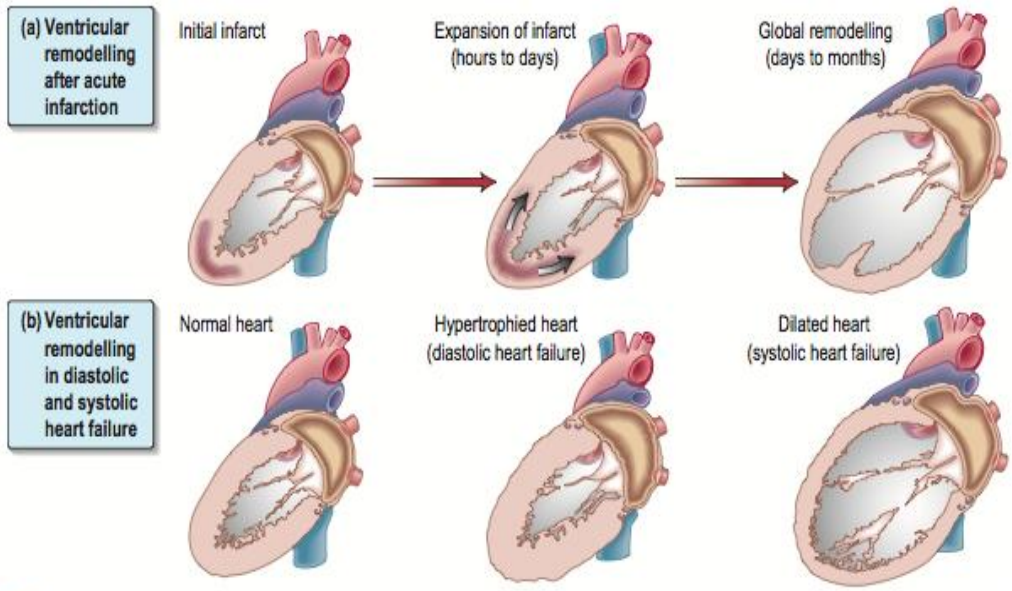



***Fig. 13.58*** **Ventricular remodelling in (a) ischaemic (MI) and (b) non-ischaemic heart failure**, e.g. in cardiomyopathy. From Jessop M, Brozana S. *New England Journal of Medicine* 2003; **348**: 2011, with permission. Copyright © 2003 Massachusetts Medical Society. All rights reserved.

## Mechanisms of HF:

1) ↓ Ventricular Contractility
2) Ventricular Outflow Obstruction (Pressure Overload)
3) Ventricular Inflow Obstruction
4) Ventricular Volume Overload ( ↑ output failure? Severe anemia, large AV shunt, Beri beri, thyrotoxicosis)
5) Arrhythmia
6) Diastolic Dysfunction

# Types & Forms of HF

## Left Heart Failure

- Lt atrial pressure↑- if acute manifests as pulmonary congestion (edema)- if gradual (ex MS) it manifests as pulmonary hypertension due to reflex pulmonary VC which protects against the edema.

## Right Heart Failure

- Dilated cardiomyopathy/IHD: affects both ventricles – Disease of LT ventricle>chronic ↑of Lt atrial pressure> pulmonary hypertension> Rt HF

## Biventricular HF:

- Dilated cardiomyopathy/IHD: affects both ventricles – Disease of LT ventricle>chronic ↑of Lt atrial pressure> pulmonary hypertension> Rt HF

## Systolic:

- Impaired myocardial contractility – Ex: Infarction> loss of muscle mass> ↓SV> ↓ CO> Systolic HF
- Ejection Fraction < 40%

## Diastolic:

- Abnormal Ventricular Relaxation> poor ventricular filling + ↑filling pressures> stiff non-compliant ventricle/Lt Ventricular hypertrophy (so hypertrophy is the result not the cause)
- Ejection Fraction > 40%

## Acute:

- de novo – as acute episode of decompensation on chronic HF background (Acute-On-Chronic HF)

## Chronic:

- adaptive changes take place (compensatory mechanisms) and prevent development of overt HF

## Clinical Features:

### Symptoms of Lt-sided HF:

Fatigue, orthopnea, shortness of breath, paroxysmal nocturnal dyspnea, wheezing (cardiac asthma), crackles

Sudden onset of dyspnea at rest> rapidly progresses to acute respiratory distress, orthopnea and prostration – peripheries cool and clammy – rapid pulse – inappropriate bradycardia or excessive tachycardia – ↑ BP or ↓ (if in state of shock) – ↑ JVP (especially in fluid overload + Rt sided HF) – no time for ventricular dilatation and apex displacement

### Signs of Lt-sided HF:

- Edema> Lower limb, sacral (in bedridden patients) or generalized (anasarca)
- Patient looks tachypnic using accessory muscles
- Vital signs: ↑ BP (as compensation) but sometimes ↓ BP – tachycardia
- ↑ JVP
- Crackles or crepitations (or wheezing or could have both)
- S3 ( volume overload) – triple gallop
- S4 (stiff ventricle)
- Abdominal: ascites- hepatomegaly (Rt sided HF in particular> underfilling of Lt ventricle> ↓ blood supply to body tissue) > stimulating the neurohormonal response
- Cardiomegaly

### Symptoms/Signs of Rt-sided HF:

- Peripheral pitting edema
- Nocturia
- JVD
- Hepatomegaly/Hepatojugular reflex
- Ascites
- Rt Ventricular heave

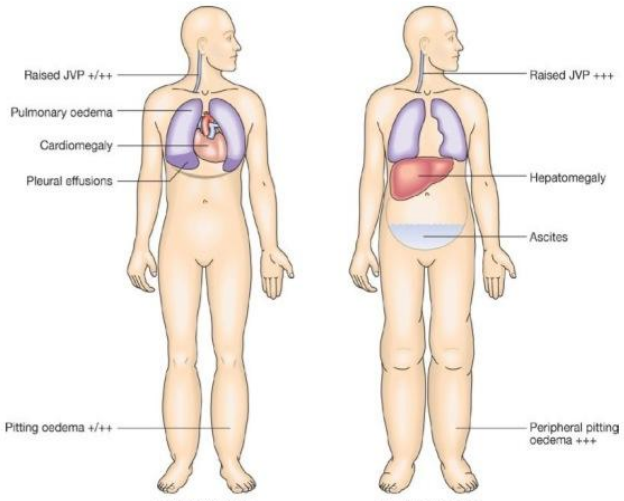


Figure 18.24 **Clinical features of left and right heart failure.** (JVP = jugular venous pressure)

### Chronic HF:

- ↓ CO > fatigue & ↓ effort tolerance + cold peripheries ( ↓ BP) + perfusion is diverted away from the skeletal muscles causing fatigue and weakness and the kidneys resulting in oliguria and uremia
- Congestive HF associated with marked weight loss (cardiac cachexia) due to anorexia, impaired absorption due to gastrointestinal congestion, poor tissue perfusion and skeletal muscle atrophy due to immobility

**TABLE 216-1 Framingham Criteria for Diagnosis of Congestive Heart Failure[a]**

| |
|---|
| ***MAJOR CRITERIA*** |
| Paroxysmal nocturnal dyspnea |
| Neck vein distention |
| Rales |
| Cardiomegaly |
| Acute pulmonary edema |
| $S_3$ gallop |
| Increased venous pressure (>16 $cmH_2O$) |
| Positive hepatojugular reflux |
| ***MINOR CRITERIA*** |
| Extremity edema |
| Night cough |
| Dyspnea on exertion |
| Hepatomegaly |
| Pleural effusion |
| Vital capacity reduced by one-third from normal |
| Tachycardia (≥120 bpm) |
| ***MAJOR OR MINOR*** |
| Weight loss ≥4.5 kg over 5 days' treatment |

[a] To establish a clinical diagnosis of congestive heart failure by these criteria, at least one major and two minor criteria are required.

*Source*: KKL Ho et al, Circulation 88:107, 1993.

**This functional classification which assesses exercise tolerance carries prognostic significance.**

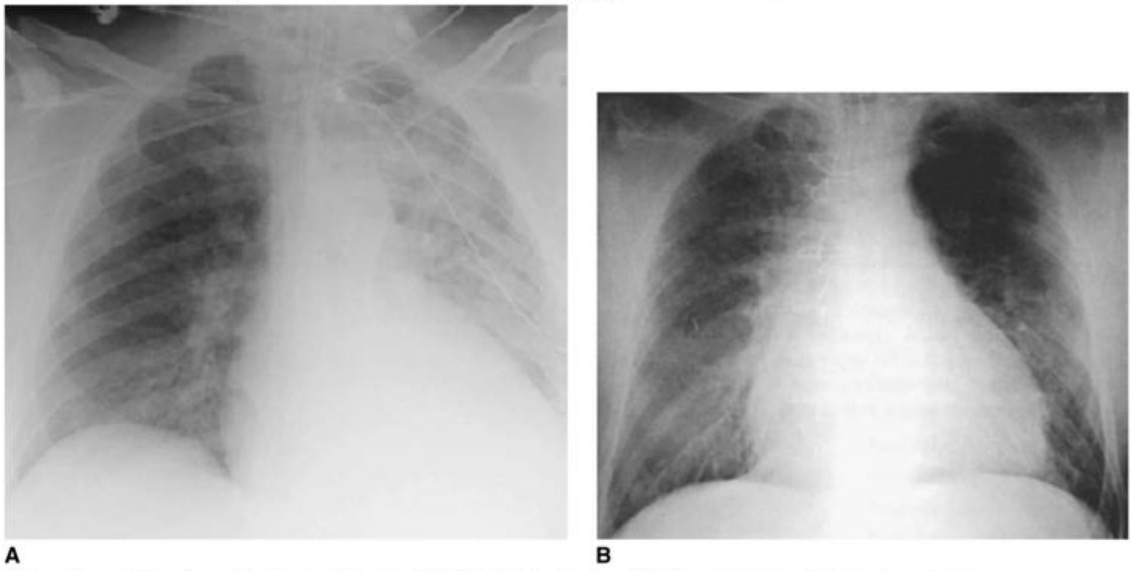
**Table 13.21 New York Heart Association (NYHA) Classification of heart failure**

| | |
|---|---|
| Class I | No limitation. Normal physical exercise does not cause fatigue, dyspnoea or palpitations |
| Class II | Mild limitation. Comfortable at rest but normal physical activity produces fatigue, dyspnoea or palpitations |
| Class III | Marked limitation. Comfortable at rest but less gentle physical activity produces marked symptoms of heart failure |
| Class IV | Symptoms of heart failure occur at rest and are exacerbated by any physical activity |

## Diagnosis:

- **CXR**
  - Cardiomegaly
  - Kerley B lines (short horizontal lines near periphery of the lung)> Pulmonary congestion 2ary to dilatation of pulmonary lymphatic vessels
  - Prominent interstitial markings
  - Pleural effusion

FIGURE 1-7 **A. Chest radiograph showing cardiogenic pulmonary edema. B. Another example of cardiogenic pulmonary edema; note cardiomegaly (patient had CHF).**



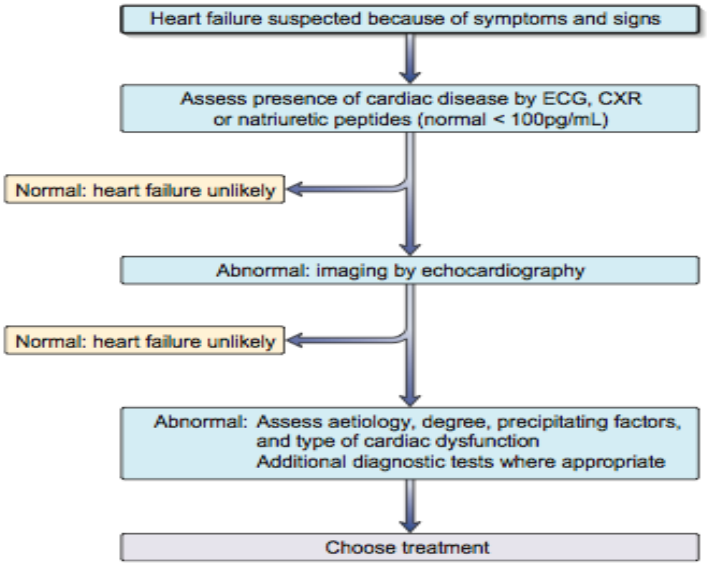
A B

(**A** from Mergo PJ. Imaging of the Chest–A Teaching File. Philadelphia: Lippincott Williams & Wilkins, 2002:50, Figure 24A.)
(**B** from Stern EJ, White CS. Chest Radiology Companion. Philadelphia: Lippincott, Williams & Wilkins, 1999:38, Figure 5-4.)

- **Echocardiogram**
  - Initial test of choice whenever HF is suspected
  - Determines whether systolic or diastolic impairment predominance of left or right ventricle
  - Determines etiology (also unsuspected valvular heart disease)
  - Identifies patients who will benefit from long-term therapy with drugs such as ACE inhibitors
  - Estimates EF: it's very important to distinguish between patients with systolic dysfunction (EF<40%) from those with preserved left ventricular function (EF>40%)
  - Shows chamber dilation and/or hypertrophy

- **ECG**
  - Nonspecific but can be used to detect chamber enlargement & presence of IHD or prior MI

- **BNP levels**
  - Released from ventricles in response to ventricular volume expansion and pressure overload
  - May be useful in differentiating between dyspnea caused by CHF and COPD
  - If >100 pg/ml, this strongly correlates with the presence of decompensated CHF – can be used to R/O HF
  - Prognostic feature: the higher the BNP the higher the cardiovascular and all-cause mortality




**Exacerbating Factors:** (>Congestive HF)

What can cause the heart to decompensate?

- Salty food (junk food, canned food, paste..)
- Uncontrolled HTN
- Acute MI (ischemia)
- Smoking
- Non-compliance to diet or medications (most common) or inappropriate reduction of therapy
- Administration of drugs that cause fluid retention (NSAIDs, negative inotropes)
- Severe prostate involvement can cause fluid retention
- Infection (ex pneumonia) causing stress> tachycardia + fever> HF
- Arrhythmia (most common type is Atrial Fibrillation)
  - Diastole: 75% is passive (which will already be impaired because of ventricular filling problems) and the rest of the 25% is due to atrial contraction. Once this atrial "kick" is lost, acute HF will occur.
- Pulmonary embolism
- ↑ metabolic demand (pregnancy, thyrotoxicosis, anemia)
- IV fluid overload

## Management:

Acute HF (>Sever pulmonary edema):

The three major treatment goals for patients with chronic HF:

- Relief of symtoms:
  - Dietary Na restriction
  - Loop diuretics
  - Digoxin
- Preventing disease progression:
  - Reversible causes should be aggressively sought and treated
- Reduction in mortality risk:
  - ACEIs or ARBs
  - Beta-blockers
  - If intolerant to ACEIs and ARBs > Hydralazine with nitrates

Any factor that is aggravating the failure should be identified and treated. Similarly, the underlying cause of HF must be determined and corrected. So basically we:

- Treat the cause
  - Thrombolytics > MI
  - Antiarrhythmics > Atrial Fibrillation (shock him if severe HF, particularly if he's in shock)
- Treat exacerbating factors
  - Anemia, thyroid disease, infection, ↑BP
- Avoid exacerbating factors
  - NSAIDs > Fluid retention
  - Verapamil > -ve inotrope

### General Measures:

- Explanation of nature of disease, treatment and self-help strategies
- Diet: Sodium restriction: <4 g/day (initially) + weight reduction for the obese
- Exercise
- Smoking and alcohol cessation
- Vaccinations (pneumococcal and influenza)

Drug Therapy:

- Systolic Dysfunction:

- **Diuretics:**
  - Used for symptomatic relief of dyspnea and improve exercise tolerance by ↓ preload.
    1. Loop Diuretics:
       - Furosemide (Lasix) most potent/Bumetanide
       - Risk of ↓k > monitor renal function and serum electrolytes

    2. Thiazide Diuretics:
       - Hydrochlorothiaziade

    3. Spironolactone
       - Has K sparing effect
       - Added if K<3.2mmol/L, predisposition to arrhythmias, concurrent digoxin therapy (↓K increases risk of digoxin toxicity) or pre-existing K losing conditions.
       - Trials have shown that they do ↓ morbidity and mortality in patients with class 3 or 4 HF (because it inhibits Ald.)
       - Contraindicated in renal failure

- **ACE Inhibitors:**
  - Captopril, enalapril, lisinopril
  - They improve survival in all functional classes (NYHA 1-4) and are recommended in all patients at risk of developing HF = initial drugs of choice in treating CHF
    - Delay onset & progression of HF in pts with asymptomatic LV dysfunction
    - ↓ cardiac remodeling
  - They ↓ preload & afterload > ↓SVR and prevent remodeling.
  - They block RAAS by inhibiting the conversion of angiotensin I to angiotensin II > VD, ↓ Na and water retention
  - Main S/E: Hypotension, cough, ↑ K and renal dysfunction

- **ARBs:**
  - 2nd line therapy in pts intolerant of ACEIs (angioneurotic edema, cough)
  - Not as effective though so ACEIs are still preferred if the patient isn't contraindicated
  - Contraindicated in bilateral renal artery stenosis

- **Beta Blockers:**
  - They're –ve inotropes so they will be working on ↓ HR
  - CO (↓) = SV (↓) x HR (↑ by compensating) > so if we take out the compensatory mechanism (↑HR) and undo it by giving him beta blockers, he will go into shock. This is why they're only used in stable chronic conditions
  - They will prevent hypertrophy by blocking RAAS thus improving remodeling.

- **Digoxin: (Digitalis)**
  - +ve inotrope agent
  - Used as add-on therapy in symptomatic HF patients already receiving ACEI and beta-blockers
  - Indicated in patients in atrial fibrillation with HF
  - Digoxin toxicity:
    - GI: Nausea, Vomiting, Anorexia
    - Cardiac: Ectopic (ventricular) beats, AV block, AFib
    - CNS: Visual disturbances, Disorientation

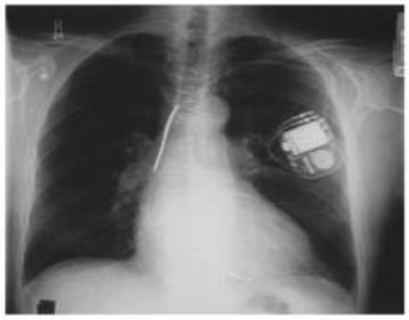
- **Vasodilators: (Nitrates & Nitrites)**
  - Combination of hydralazine and nitrates ↓ afterload and preload (clears pulmonary congestion) and is used in patients intolerant of ACEI or ARA.
  - This combination has demonstrated improvement in survival of patients with chronic HF
  - SE of Hydralazine: drug-induced lupus

- <u>Diastolic Dysfunction</u>:
  Few therapeutic options available - Symptomatic treatment

Non-pharmacological treatment of HF:

- ICD
  - Implantable Cardiac Defibrillators
  - Indicated in pts with symptomatic ventricular arrhythmias and HF

- Heart transplantation
  - Successful form of treatment for patients with intractable HF
  - CAD and dilated cardiomyopathy are the most common indications



(a)

**Prognosis:**

- Progressive: If end stage HF, patient has 3-5 years left to live unless ICD devices or transplants are done thus prolonging this period
- Median survival rate depends on underlying cause
- Overall 5 year mortality for all CHF patients is about 50%

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## References:


- Davidson's Principles & Practice of Medicine
- Step up to Medecine
- Kumar & Clark's Clinical Medicine
- 429 medicine team notes

## Extra diagrams:

**TABLE 216-2** *Stages in the Evolution of Heart Failure/Recommended Therapy by Stage*

| Stage A | | Stage B | | Stage C | | Stage D |
|---|---|---|---|---|---|---|
| At high risk for heart failure but without structural heart disease or symptoms of HF<br>Patients with hypertension coronary artery disease diabetes mellitus *or*<br>Patients using cardiotoxins with FHx CM | Structural heart disease | Structural heart disease but without symptoms of HF<br>Patients with previous MI LV systolic dysfunction asymptomatic valvular disease | Symptoms of HF develop | Structural heart disease with prior or current symptoms of HF<br>Patients with known structural heart disease shortness of breath and fatigue, reduced exercise tolerance | Refractory symptoms of HF at rest | Refractory HF requiring specialized interventions<br>Patients who have marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions) |
| ***THERAPY*** | | | | | | |
| Treat hypertension<br>Encourage smoking cessation<br>Treat lipid disorders<br>Encourage regular exercise<br>Discourage alcohol intake, illicit drug use<br>ACE inhibition | | All measures under stage A<br>ACE inhibitors in appropriate patients<br>Beta-blockers | | All measures under stage A<br>Drugs for routine use:<br>Diuretics<br>ACE inhibitors<br>Beta-blockers<br>Digitalis<br>Dietary Salt restriction | | All measures under stages A, B, and C<br>Mechanical assist devices<br>Heart transplantation<br>Continuous (not intermittent) IV inotropic infusions for palliation<br>Hospice care |

***Abbreviations***: HF, heart failure, FHxCM, family history of cardiomyopathy; ACE, angiotensin-converting enzyme; MI, myocardial infarction; LV, left ventricular; IV, intravenous.

***Source***: Modified from S Hunt: J Am Coll Cardiology, 38:2101, 2001, with permission.

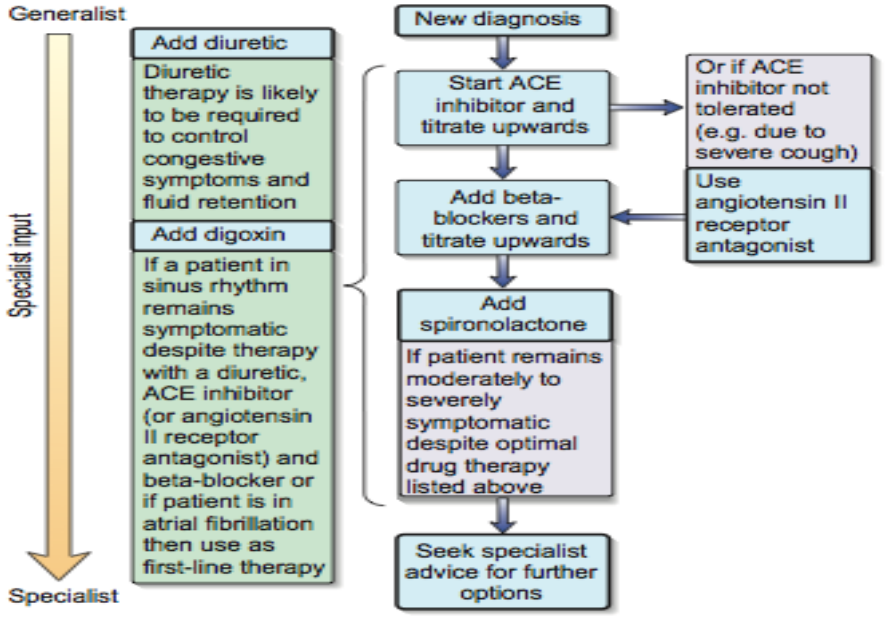


***Fig. 13.60*** Heart failure treatment guidelines. From NICE. 'Algorithm for the pharmacological treatment of symptomatic heart failure due to left ventricular systolic dysfunction' in CG5 chronic heart failure: management of chronic heart failure in adults in primary and secondary care. London: NICE, 2003. Available from www.nice.org.uk/CG005. Reproduced with permission.

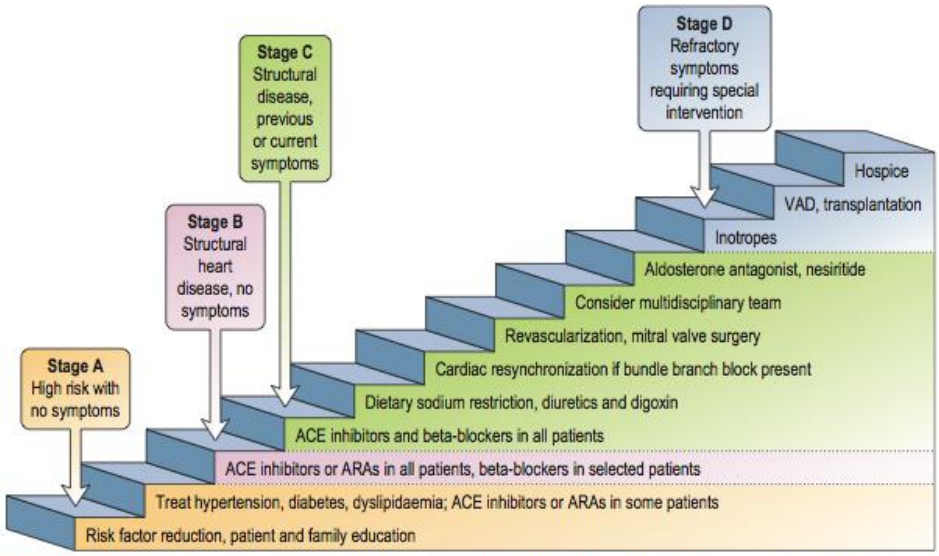


***Fig. 13.61*** **Stages of heart failure and treatment options for systolic heart failure.** ARA, angiotensin II receptor antagonist; ACE, angiotensin-converting enzyme; VAD, ventricular assisted device. From Jessop M, Brozana S. *New England Journal of Medicine* 2003; **348**: 2013, with permission. 