Heart Failure

Etiology And Diagnosis

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Definition:

Heart failure (HF) is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.

Prevalence

- Prevalence 0.4-2% overall, 3-5 % in over 65s, 10% of over 80s
- Commonest medical reason for admission
- Annual mortality of 60% over 80s
- > 10% also have AF
- Progressive condition - median survival 5 years after diagnosis

- REMEMBER LEFT VENTRICULAR FAILURE IS A TRUE LIFE THREATENING EMERGENCY

Etiology

- *It is a common end point for many diseases of cardiovascular system*
- It can be caused by :

  -Inappropriate work load (volume or pressure overload)

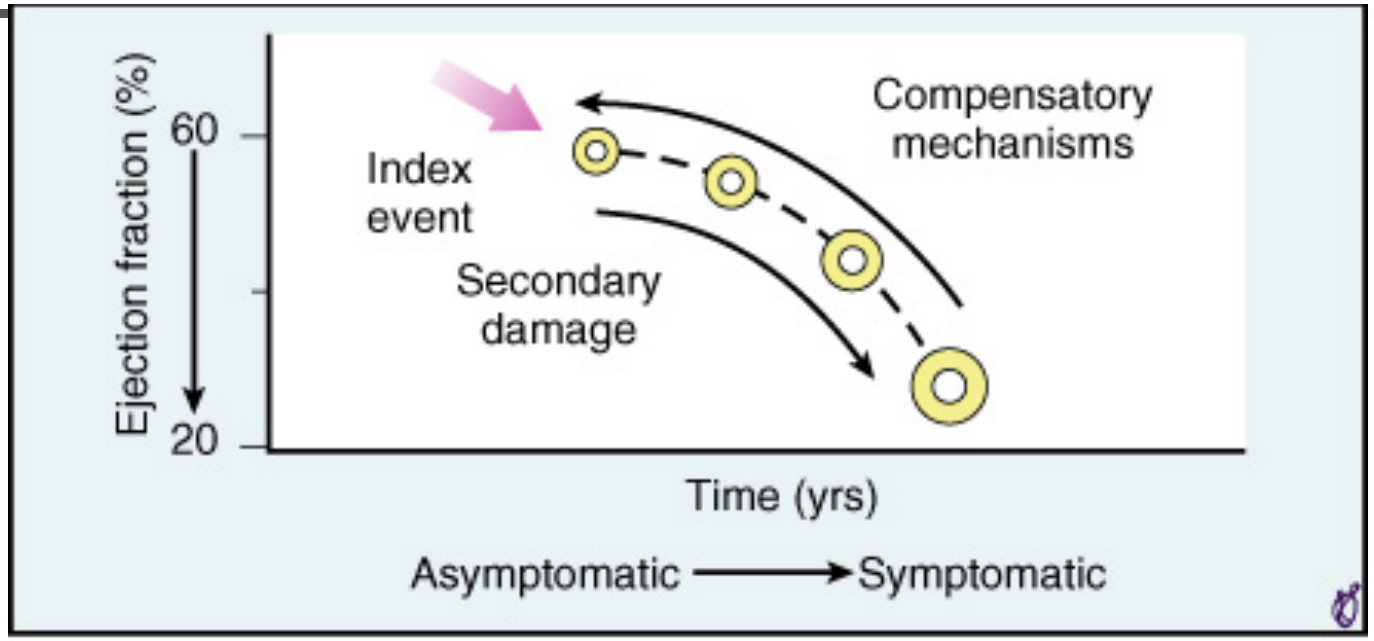
  -Restricted filling

  -Myocyte loss

Causes of left ventricular failure

- ***Volume over load***: Regurgitate valve
  High output status
- ***Pressure overload:*** Systemic hypertension
  Outflow obstruction
- ***Loss of muscles:*** Post MI, Chronic ischemia
  Connective tissue diseases
  Infection, Poisons
  (alcohol,cobalt,Doxorubicin)
- ***Restricted Filling:*** Pericardial diseases, Restrictive cardiomyopathy, tachyarrhythmia

Background



- Heart failure pathophysiology
  - Index event
  - Compensatory mechanisms
  - Maladaptive mechanisms

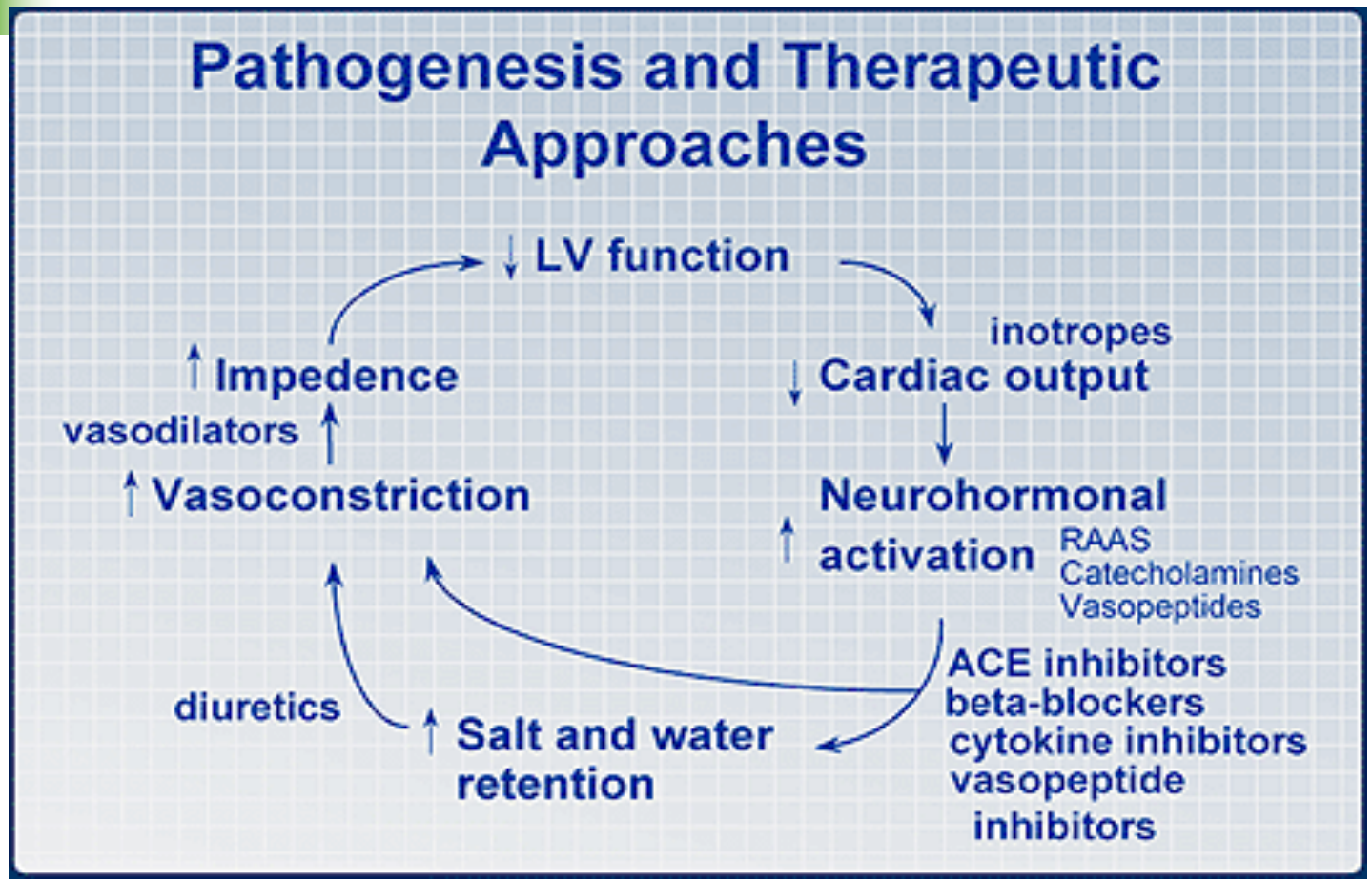
Pathophysiology

- Hemodynamic changes
- Neurohormonal changes
- Cellular changes

Hemodynamic changes

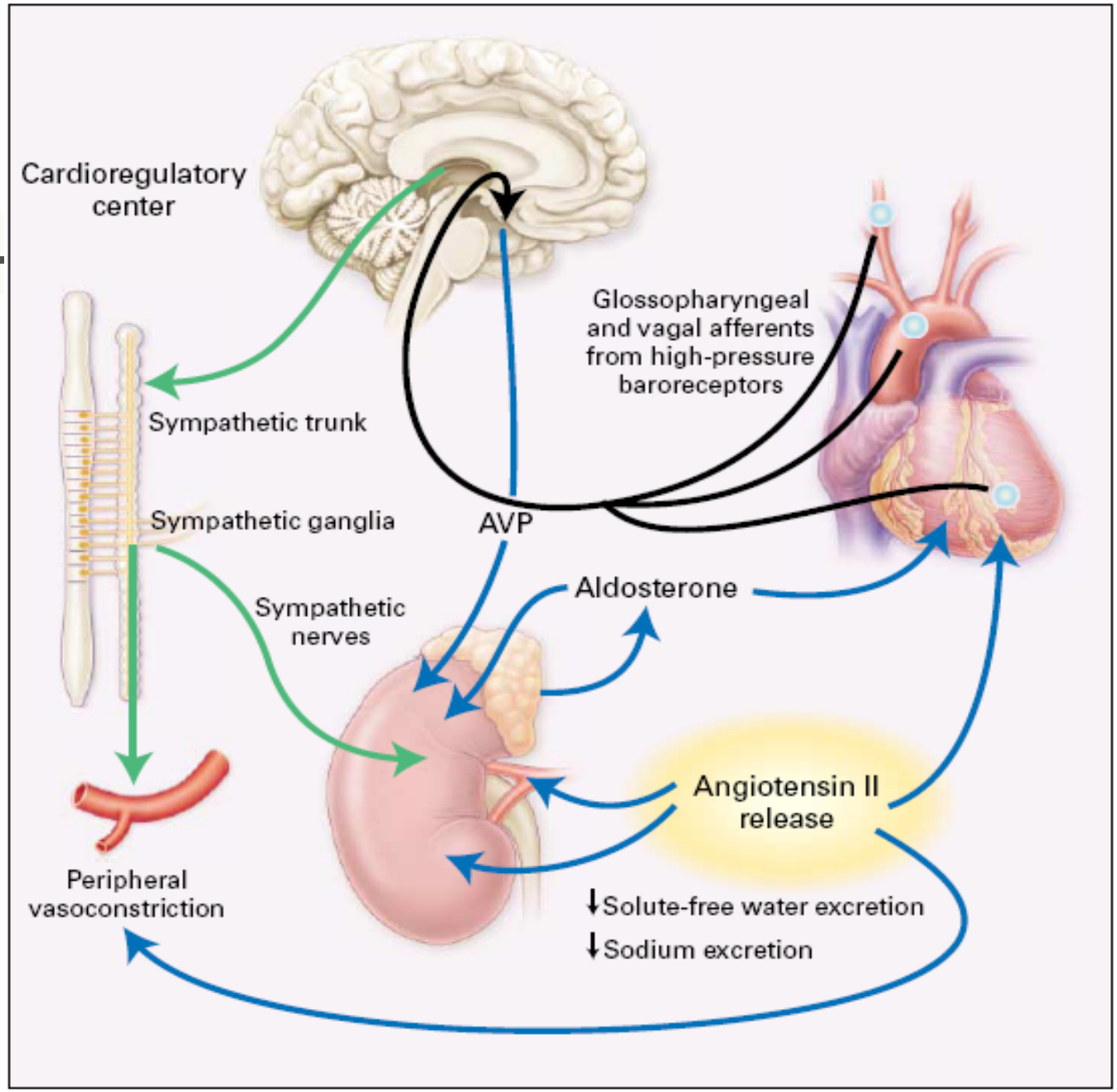
- *From hemodynamic stand point HF can be secondary to systolic dysfunction or diastolic dysfunction*

Pathogenesis and Therapeutic Approaches



Neurohormonal changes

N/H changes	Favorable effect	Unfavor. effect
↑ Sympathetic activity	↑ HR ,↑ contractility, vasoconst. → ↑ V return, ↑ filling	Arteriolar constriction → After load →↑ workload →↑ O_2 consumption
↑ Renin-Angiotensin – Aldosterone	Salt & water retention→↑ VR	Vasoconstriction → ↑ after load
↑ Vasopressin	Same effect	Same effect
↑ interleukins &TNFα	May have roles in myocyte hypertrophy	Apoptosis
↑Endothelin	Vasoconstriction→↑ VR	↑ After load


Cardioregulatory center
Glossopharyngeal and vagal afferents from high-pressure baroreceptors
Sympathetic trunk
Sympathetic ganglia
AVP
Aldosterone
Sympathetic nerves
Angiotensin II release
Peripheral vasoconstriction
↓Solute-free water excretion
↓Sodium excretion

Cellular changes

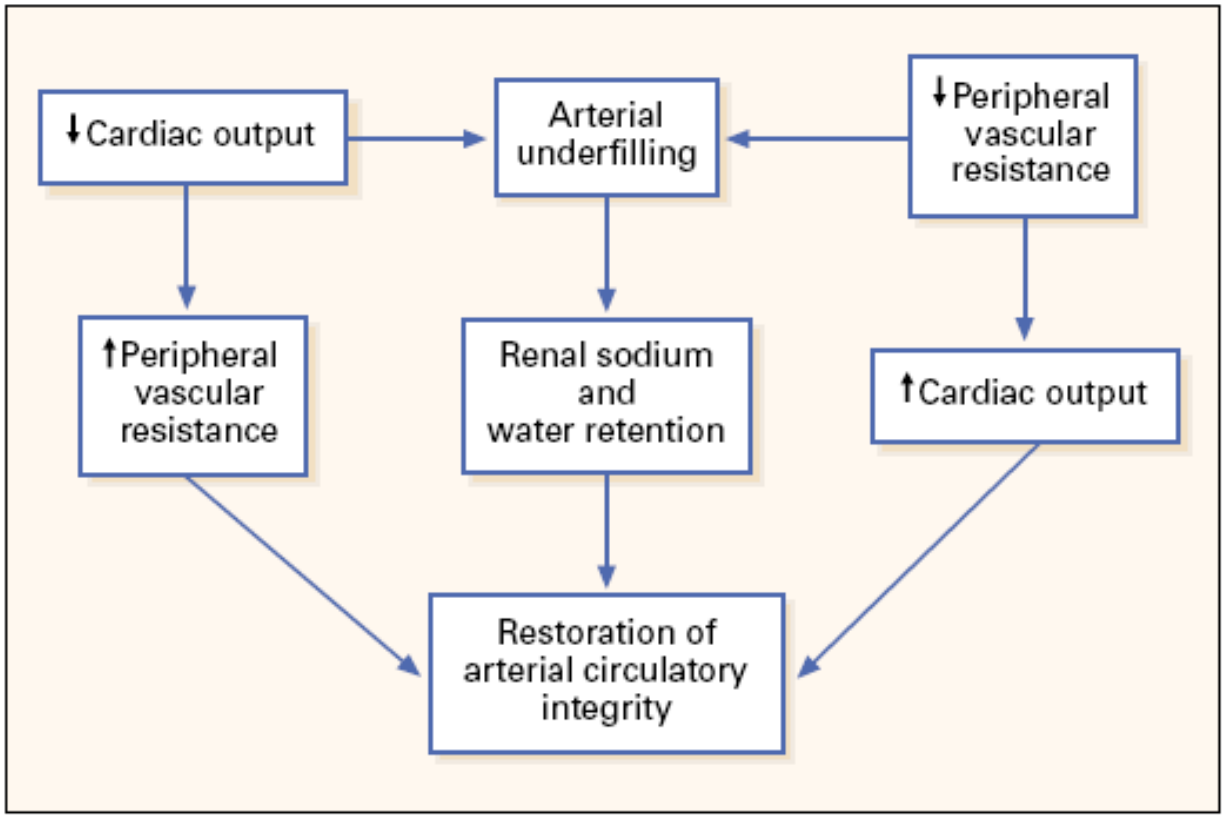
- ***Changes in Ca^{+2} handling.***
- ***Changes in adrenergic receptors:***
  - Slight ↑ in α_1 receptors
  - β_1 receptors desensitization → followed by down regulation
- ***Changes in contractile proteins***
- ***Program cell death* (Apoptosis)**
- ***Increase amount of fibrous tissue***

Body-Fluid Volume

- Renal Na and water excretion
  - Dependent on arterial circulation
  - Cardiac output and peripheral resistance
- Decrease in circulation leads to ***arterial underfilling***
  - Decreased effective circulating volume
- ***Neurohormonal*** reflexes are triggered

Arterial Underfilling

- Causes and consequences
- Counter-regulation

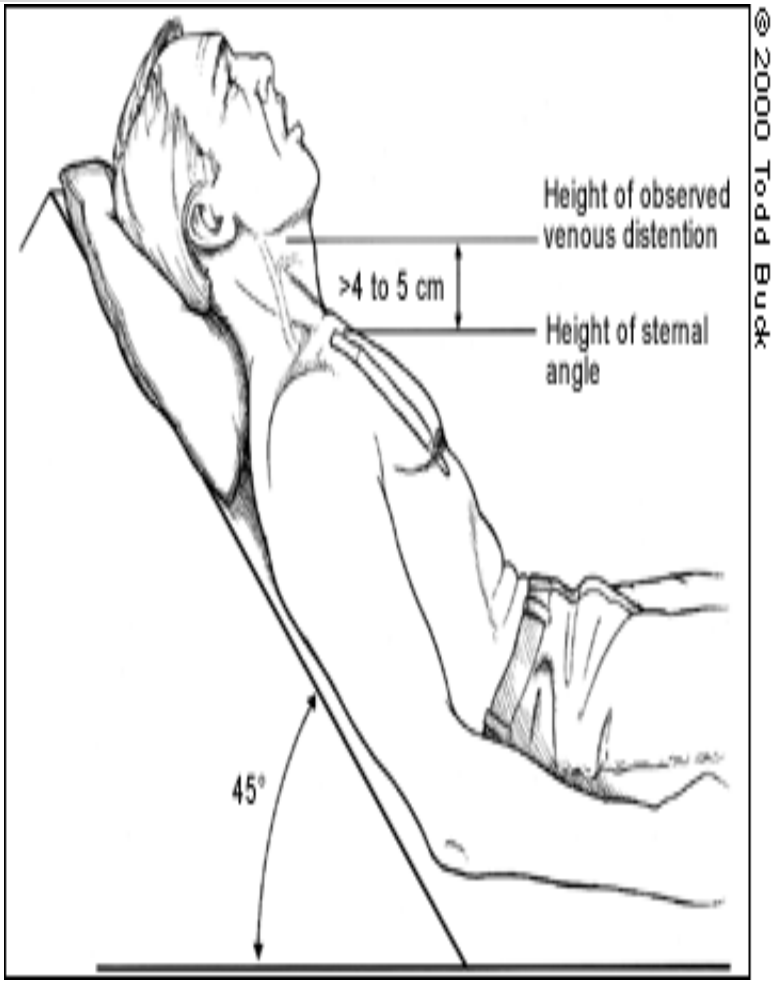


Symptoms

- **SOB, Orthopnea, paroxysmal nocturnal dyspnea**
- **Low cardiac output symptoms**
- **Abdominal symptoms:** *Anorexia,nausea, abdominal fullness, Rt hypochondrial pain*

Physical Signs

- High diastolic BP & occasional decrease in systolic BP (decapitated BP)
- JVD
- Rales *(Inspiratory)*
- Displaced and sustained apical impulses
- Third heart sound – *low pitched sound that is heard during rapid filling of ventricle*



Physical signs (cont.)

- Mechanism of S_3 sudden deceleration of blood as elastic limits of the ventricles are reached
- Vibration of the ventricular wall by blood filling
- Common in children

Physical signs (cont.)

- **Fourth heart Sound (S_4)**

  - Usually at the end of diastole
  - Exact mechanism is not known

  Could be due to contraction of atrium against stiff ventricle

- **Pale, cold sweaty skin**

Framingham Criteria for Dx of Heart Failure

- Major Criteria:
  - PND
  - JVD
  - Rales
  - Cardiomegaly
  - Acute Pulmonary Edema
  - S_3 Gallop
  - Positive hepatic Jugular reflex
  - ↑ venous pressure > 16 cm H_2O

Dx of Heart Failure (cont.)

- **Minor Criteria**

LL edema,

Night cough

Dyspnea on exertion

Hepatomegaly

Pleural effusion

Tachycardia 120 bpm

Weight loss 4.5 kg over 5 days management

Forms of Heart Failure

- **Systolic & Diastolic**
- **High Output Failure**
  - Pregnancy, anemia, thyrotoxisis, A/V fistula, Beriberi, Pagets disease
- **Low Output Failure**
- **Acute**
  - large MI, aortic valve dysfunction---
- **Chronic**

Forms of heart failure (cont.)

- **Right vs Left sided heart failure:**

Right sided heart failure :

Most common cause is left sided failure

Other causes included : Pulmonary embolisms
Other causes of pulmonary htn.
RV infarction
MS

Usually presents with: LL edema, ascites
hepatic congestion
cardiac cirrhosis (on the long run)

Differential diagnosis

- Pericardial diseases
- Liver diseases
- Nephrotic syndrome
- Protein losing enteropathy

Laboratory Findings

- Anemia
- Hyperthyroid
- Chronic renal insuffiency, electrolytes abnormality
- Pre-renal azotemia
- Hemochromatosis

Electrocardiogram

- Old MI or recent MI
- Arrhythmia
- Some forms of Cardiomyopathy are tachycardia related
- LBBB→*may help in management*

Chest X-ray

- Size and shape of heart
- Evidence of pulmonary venous congestion (dilated or upper lobe veins → perivascular edema)
- Pleural effusion

Echocardiogram

- Function of both ventricles
- Wall motion abnormality that may signify CAD
- Valvular abnormality
- Intra-cardiac shunts

Cardiac Catheterization

- When CAD or valvular is suspected
- If heart transplant is indicated

In conclusion, congestive heart failure is often assumed to be a disease when in fact it is a syndrome caused by multiple disorders.

TREATMENT

- Correction of reversible causes
  - Ischemia
  - Valvular heart disease
  - Thyrotoxicosis and other high output status
  - Shunts
  - Arrhythmia
    - A fib, flutter, PJRT
  - Medications
    - Ca channel blockers, some antiarrhythmics

Diet and Activity

- Salt restriction
- Fluid restriction
- Daily weight (tailor therapy)
- Gradual exertion programs

Diuretic Therapy

- The most effective symptomatic relief
- Mild symptoms
  - HCTZ
  - Chlorthalidone
  - Metolazone
  - Block Na reabsorbtion in loop of henle and distal convoluted tubules
  - Thiazides are ineffective with GFR < 30 --/min

Diuretics (cont.)

- **Side Effects**
  - Pre-renal azotemia
  - Skin rashes
  - Neutropenia
  - Thrombocytopenia
  - Hyperglycemia
  - ↑ Uric Acid
  - Hepatic dysfunction

Diuretics (cont.)

- **More severe heart failure → loop diuretics**
  - **Lasix** (20 – 320 mg QD), Furosemide
  - **Bumex** (Bumetanide 1-8mg)
  - **Torsemide** (20-200mg)

Mechanism of action: Inhibit chloride reabsortion in ascending limb of loop of Henle results in natriuresis, kaliuresis and metabolic alkalosis

Adverse reaction:

pre-renal azotemia
Hypokalemia
Skin rash
ototoxicity

K^+ Sparing Agents

- **Triamterene & amiloride** – acts on distal tubules to ↓ K secretion
- **Spironolactone** (Aldosterone inhibitor)

  recent evidence suggests that it may improve survival in CHF patients due to the effect on renin-angiotensin-aldosterone system with subsequent effect on myocardial remodeling and fibrosis

Inhibitors of renin-angiotensin-aldosterone system

- Renin-angiotensin-aldosterone system *is activation early in the course of heart failure and plays an important role in the progression of the syndrome*
- Angiotensin converting enzyme inhibitors
- Angiotensin receptors blockers
- Spironolactone

Angiotensin Converting Enzyme Inhibitors

- *They block the R-A-A system by inhibiting the conversion of angiotensin I to angiotensin II → vasodilation and ↓ Na retention*
- *↓ Bradykinin degradation ↑ its* level → *↑ PG secretion & nitric oxide*
- Ace Inhibitors were found to improve survival in CHF patients
  - Delay onset & progression of HF in pts with asymptomatic LV dysfunction
  - ↓ cardiac remodeling

Side effects of ACE inhibitors

- Angioedema
- Hypotension
- Renal insuffiency
- Rash
- cough

Angiotensin II receptor blockers

- Has comparable effect to ACE I
- Can be used in certain conditions when ACE I are contraindicated (angioneurotic edema, cough)

Digitalis Glycosides (Digoxin, Digitoxin)

- The role of digitalis has declined somewhat because of safety concern
- Recent studies have shown that digitals does not affect mortality in CHF patients but causes significant
  - Reduction in hospitalization
  - Reduction in symptoms of HF

Digitalis (cont.)
Mechanism of Action

- +ve inotropic effect by ↑ intracellular Ca & enhancing actin-myosin cross bride formation (binds to the Na-K ATPase → inhibits Na pump → ↑ intracellular Na → ↑ Na-Ca exchange
- Vagotonic effect
- Arrhythmogenic effect

Digitalis Toxicity

- Narrow therapeutic to toxic ratio

- Non cardiac manifestations

  Anorexia,

  Nausea, vomiting,

  Headache,

  Xanthopsia sotoma,

  Disorientation

Digitalis Toxicity

- **Cardiac manifestations**
  - Sinus bradycardia and arrest
  - A/V block (usually 2nd degree)
  - Atrial tachycardia with A/V Block
  - Development of junctional rhythm in patients with a fib
  - PVC's, VT/ V fib (bi-directional VT)

Digitalis Toxicity
Treatment

- Hold the medications
- Observation
- In case of A/V block or severe bradycardia → atropine followed by temporary PM if needed
- In life threatening arrhythmia → digoxin-specific fab antibodies
- Lidocaine and phenytoin could be used – try to avoid D/C cardioversion in non life threatening arrhythmia

β Blockers

- Has been traditionally contraindicated in pts with CHF
- Now they are the main stay in treatment on CHF & may be the only medication that shows substantial improvement in LV function
- In addition to improved LV function multiple studies show improved survival
- The only contraindication is severe decompensated CHF

Vasodilators

- **Reduction of afterload** by arteriolar vasodilatation (hydralazin) → reduce LVEDP, O_2 consumption,improve myocardial perfusion, ↑ stroke volume and COP
- **Reduction of preload** By venous dilation (Nitrate) → ↓ the venous return →↓ the load on both ventricles.
- Usually the maximum benefit is achieved by using agents with both action.

Positive inotropic agents

- These are the drugs that improve myocardial contractility (β adrenergic agonists, dopaminergic agents, phosphodiesterase inhibitors),

  dopamine, dobutamine, milrinone, amrinone
- Several studies showed ↑ mortality with oral inotropic agents
- So the only use for them now is in acute sittings as cardiogenic shock

Anticoagulation (coumadine)

- Atrial fibrillation
- H/o embolic episodes
- Left ventricular apical thrombus

Antiarrhythmics

- Most common cause of SCD in these patients is ventricular tachyarrhythmia
- Patients with h/o sustained VT or SCD → ICD implant

Antiarrhythmics (cont.)

- Patients with non sustained ventricular tachycardia
  - Correction of electrolytes and acid base imbalance
  - In patients with ischemic cardiomyopathy → ICD implant is the option after r/o acute ischemia as the cause
  - In patients wit non ischemic cardiomyopathy management is ICD implantation

New Methods

- **Implantable ventricular assist devices**
- **Biventricular pacing** (only in patient with LBBB & CHF)
- **Artificial Heart**

Cardiac Transplant

- It has become more widely used since the advances in immunosuppressive treatment

- Survival rate
  - 1 year 80% - 90%
  - 5 years 70%

Prognosis

- Annual mortality rate depends on patients symptoms and LV function
- 5% in patients with mild symptoms and mild ↓ in LV function
- 30% to 50% in patient with advances LV dysfunction and severe symptoms
- 40% – 50% of death is due to SCD