



## HEART FAILURE

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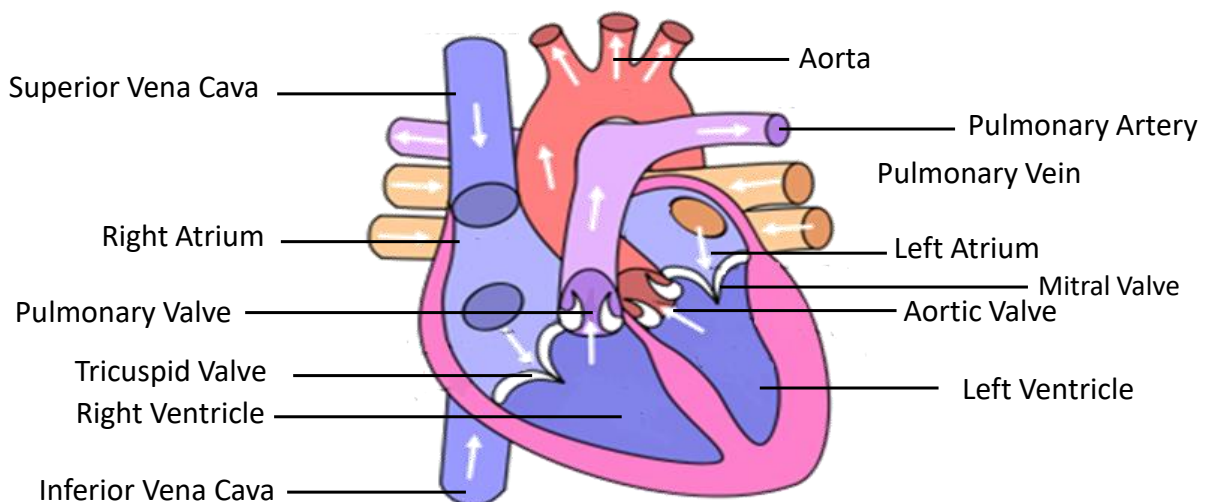
## OBJECTIVES

- Understand and explain the pathophysiology of HF
- Discuss various mechanisms that compensate for the failure
- Differentiate signs and symptoms of left-sided HF and right-sided HF
- Identify the goals of care
- Understand most common interventions

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## ANATOMY OF THE HEART



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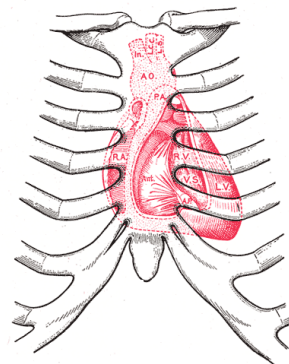
## DEFINITIONS

**Heart Failure (HF):** occurs when heart cannot meet the body's oxygen demand

- Reduced pumping efficiency of the left ventricle: ↓ Cardiac Output
- As output decreases, backflow occurs

Classified as:

- Left ventricular failure (left-sided failure) or Right ventricular failure (right-sided heart failure) or both
- Acute or Chronic



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## DEFINITIONS

**Acute HF:** sudden deterioration of signs and symptoms of HF.

**Chronic HF:** a long-term condition of the heart that is characterized by the decreased pumping power of the heart muscle.

**Cor Pulmonale:** enlargement of right side of heart due to pulmonary congestion.

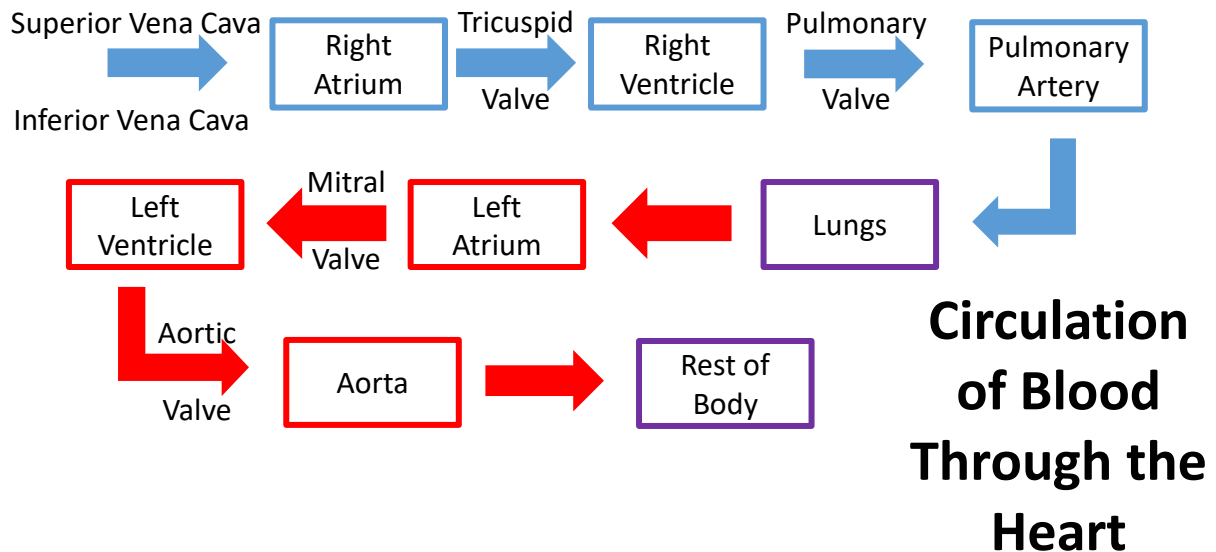
**Cardiac Output (CO):** volume of blood ejected from ventricles/minute.

**Stroke Volume (SV):** volume of blood ejected during systole.

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## PATHOPHYSIOLOGY



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## PATHOPHYSIOLOGY

In order to compensate for reduced CO, body responds with **four compensatory mechanisms**:

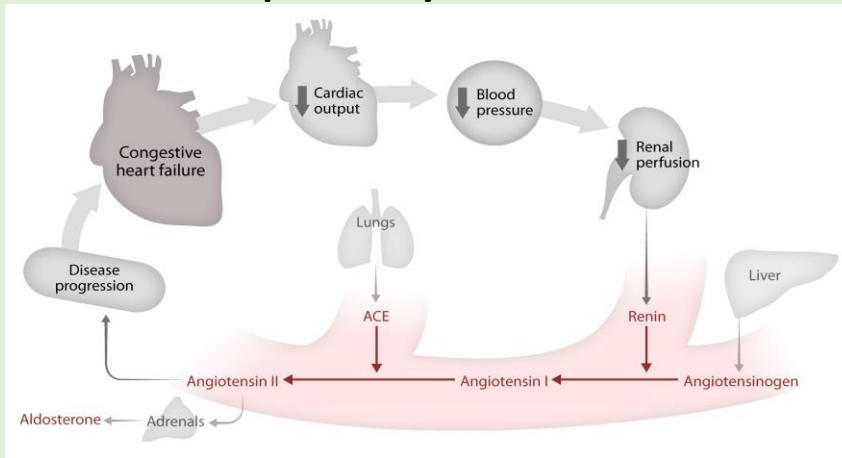
1. **Stimulation of RAAS** (renin-angiotensin-aldosterone system), which causes water retention and constriction of blood vessels in order to maintain blood perfusion (increases afterload and preload).
2. **Increase in SV** in order to increase CO (increase preload).
3. **Increase in heart rate** in order to increase CO (increases preload and afterload).
4. **Enlarging ventricular walls** to generate a greater force of contraction (remodeling).

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# PATHOPHYSIOLOGY - RISK FACTORS

## Compensatory Mechanism



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# ETIOLOGY - RISK FACTORS

## Non-Modifiable

- Increasing age
- Cardiomyopathy (viral, alcohol, anthracycline use)
- Rheumatic heart disease
- Valvular heart disease

## Modifiable

- Hypertension
- Coronary artery disease
- Diabetes mellitus
- Smoking
- Hyperlipidemia
- Obesity



- **Most common cause of HF:** coronary heart disease (70%) and hypertension
- **HF is preventable – risk is 50% reduced by managing HTN alone**

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## ETIOLOGY

These **precipitating factors** can cause acute decompensation from chronic HF (CHF) to acute HF

- **Uncontrolled hypertension/Non-compliance with BP medications**
- High fluid/sodium intake
- Cardiac events (ex. ischemia/infarction, arrhythmia)
- Anemia, stress, infections, thyrotoxicosis
- Medications (NSAIDs and Beta Blockers)



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## DIAGNOSTICS

**Jugular Venous Distention (JVD):** most reliable sign of fluid overload

- >4cm distention of JVD when client is seated at a 45 degree angle

**Electrocardiogram:** detects coronary artery disease

**Echocardiogram:** determines wall thickness, chamber size

- Most sensitive test for HF diagnosis

**Brain Natriuretic Peptide (BNP):** biomarker for diagnosis of HF or HF exacerbation

- Detects when either ventricles are being stretched from increased volume
- Helps differentiate dyspnea is from HF or not

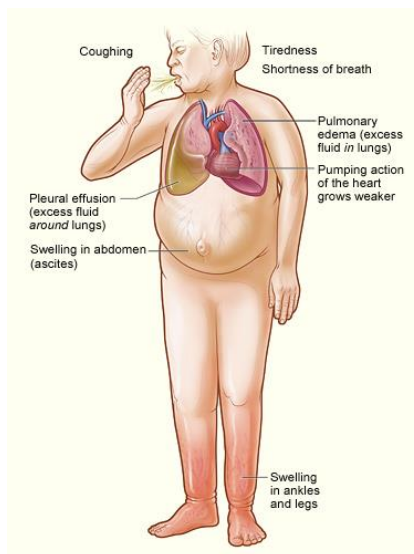
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# CLINICAL MANIFESTATIONS

## Left-sided HF

- Fatigue
- Tachypnea and dyspnea
- Crackles
- ↑BP if fluid overload
- ↓BP if impaired pump
- Dry cough
- Pink frothy sputum
- Cyanosis
- Orthopnea



## Right-sided HF

- Peripheral edema
- Weight gain
- Hepatomegaly with RUQ pain
- Ascites
- Splenomegaly
- Jugular vein distention

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# CLINICAL MANIFESTATIONS

## Pulmonary Congestion/Edema

- Crackles (from the bases moving upwards), SOB, tachycardia, ↓urine output, pink frothy sputum, lethargy, and restlessness, cool, clammy, or cyanosis

## Peripheral Edema (weight gain > 1lb overnight)

## Systemic Congestion

- Jugular venous congestion, hepatomegaly, splenomegaly, ascites, N/V/abdominal pain
- Right side of heart backing up systemic circulation

## Hypotension, Fatigue, Cyanosis, Confusion, Chest pain

- Left ventricle unable to supply adequate CO to body

### NURSING ALERT!



Pulmonary edema can emerge very rapidly and requires immediate attention. Report any changes that may suggest fluid overload to the health care provider



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## PULMONARY EDEMA INTERVENTIONS

- ✓ Place in high Fowler's
- ✓ Administer oxygen
- ✓ Quickly assess lung sounds
- ✓ Sublingual nitroglycerin if SBP above 100 to reduce afterload and preload (q5mins up to 3 doses)
- ✓ Initiate IV access
- ✓ Diuretics such as Lasix to increase output
- ✓ If BP stable, IV morphine
- ✓ Frequent VS (especially BP and Respiratory rate)

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## GOALS OF CARE

- Control risk factors including HTN
- Manage fluid balance
- Manage cardiac demands



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## INTERVENTIONS - NON-PHARMACOLOGICAL

### Diet:

- Salt restriction: 1.5-2g Na / day
- Eliminate high salt foods (deli meats, processed foods)
- Fluid restriction – 2L/day from all sources

### Frequent weight monitoring

**Control** HF risk factors (**hypertension**, etc.)

No more than one alcoholic drink /day



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## INTERVENTIONS - PHARMACOLOGICAL

**Angiotensin Converting Enzyme Inhibitors (ACE-I):** reduce preload by blocking angiotensin II and hence sodium and water retention.

- **Adverse effects:** chronic cough, hypotension, hyperkalemia, and **angioedema (serious)**
- **Contraindication:** pregnancy
- **1<sup>st</sup> line for all clients including asymptomatic**

**ACE-I**  
Ramipril,  
Enalapril,  
Lisinopril

**Angiotensin Receptor Blockers (ARBs):** reduces preload by reducing water and sodium retention.

- Indicated when ACE-I not well tolerated
- **Contraindication:** pregnancy

**ARBs**  
Candesartan  
Valsartan

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## INTERVENTIONS - PHARMACOLOGICAL

**Beta Blockers (BBs):** antagonizes sympathetic nervous system: ↓ HR, inhibits activation of RAAS and therefore sodium/water retention.

- **Adverse effects:** ↓ BP and bradycardia/heart block
- **Drug interaction:** insulin and calcium channel blockers

### BBs

Bisoprolol,  
Carvedilol,  
Metoprolol

**Vasodilators – Nitrates:** ↓ preload, slightly ↓ vascular resistance and ↑ blood flow.

- **Side effects:** headaches and hypotension
- **Drug interaction:** antihypertensives
- **Contraindication:** nitrates with sildenafil (Severe hypotension)



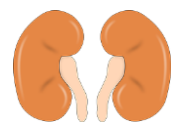
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## INTERVENTIONS - PHARMACOLOGICAL

**Diuretics:** ↓ preload (fluid retention), ↓ edema + pulmonary congestion.

- **Side effects:** hypokalemia, hypotension, and other electrolyte imbalances
- **Drug interactions:** antihypertensives and NSAIDs  
↑ renal toxicity
- Monitor daily weight and renal function regularly to avoid dehydration



### Loop diuretics

Furosemide  
Bumetanide

### Thiazide diuretics

chlorthalidone,  
hydrochlorothiazid

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## INTERVENTIONS - PHARMACOLOGICAL

**Aldosterone antagonists:** Inhibition of aldosterone receptor helps ↓preload + ↓edema

- Side effects: hyperkalemia, nausea, and dizziness
- **Drug interaction:** hyperkalemia with other potassium sparing medications
- **Contraindication:** baseline K<sup>+</sup> levels > 5mmol/L

**Aldosterone antagonists:**  
Spironolactone,  
Epleronone

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## INTERVENTIONS - PHARMACOLOGICAL

**Digoxin:** positive inotropic and ↓ reabsorption of Na in kidneys

- **Drug interaction:** medications that waste potassium (i.e. potassium wasting diuretics)
- **Contraindication:** ventricular arrhythmias and heart block
- Do not administer if HR < 60bpm
- Periodic drug levels drawn to prevent toxicity

### **NURSING ALERT!**



Monitor for digoxin toxicity. Classic signs include: N/V diarrhea, irregular pulse, dysrhythmias and vision changes (seeing halos). Notify HCP immediately.



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## SUMMARY

- Heart failure occurs when the heart becomes unable to adequately pump out blood to meet the needs of the body
- Uncontrolled HTN is the number one cause of HF
- Manifestations depend on the side of the heart that is failing
  - Left-sided HF: pulmonary manifestations
  - Right-sided HF: systemic manifestations
- Diagnosis is based on visualizing the heart for its size and thickness
- Goal of care is to manage hypertension, fluid balance and cardiac output demands

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