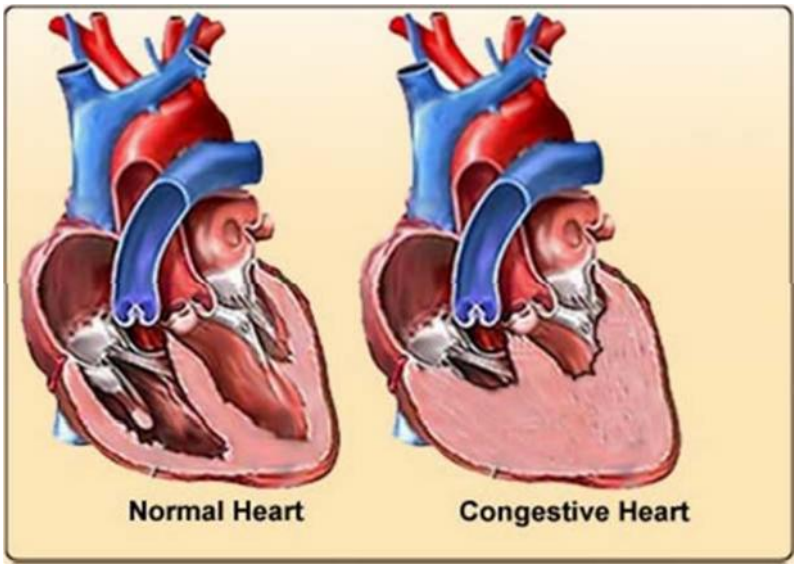
CONGESTIVE CARDIAC FAILURE

- A clinical syndrome
- Result from any disorder that impairs the ability of the ventricle to fill with or eject blood
- Rendering the heart unable to pump blood at a rate sufficient to meet the metabolic demands of the body
- Heart failure can result from any disorder that reduces ventricular filling (diastolic dysfunction) and/or myocardial contractility (systolic dysfunction)



Etiology

Systolic dysfunction (decreased contractility)

- Reduction in muscle mass (e.g. myocardial infarction)
- Dilated cardiomyopathies
- Ventricular hypertrophy
- Pressure overload (e.g. systemic or pulmonary HT, aortic or pulmonic valve stenosis)
- Volume overload (e.g., valvular regurgitation, shunts, high-output states)

Diastolic dysfunction (restriction in ventricular filling)

- Increased ventricular stiffness
- Ventricular hypertrophy (e.g. hypertrophic cardiomyopathy)
- Infiltrative myocardial diseases (e.g. amyloidosis, sarcoidosis, endomyocardial fibrosis)
- Myocardial ischemia and infarction
- Mitral or tricuspid valve stenosis
- Pericardial disease (e.g. pericarditis, pericardial tamponade)

Types of Heart Failure

- Acute and chronic heart failure
- High output and low output HF
- Left sided, right sided and biventricular HF
- Forward and backward HF

- Systolic and diastolic HF

Acute heart failure

- Heart is not able to pump the blood effectively
- Also called as Decompensated heart failure
- Compensatory mechanisms of human body cause increase in CO by stimulation of β_1 receptors and also RAA-system
- Both mechanism leads to vasoconstriction
- Treatment with **Inotropic** drugs becomes necessary

Chronic heart failure

- Failure of compensatory mechanism
- Heart needs to undergo surgery for its repair
- Further controlled by administration of drugs

High output HF:

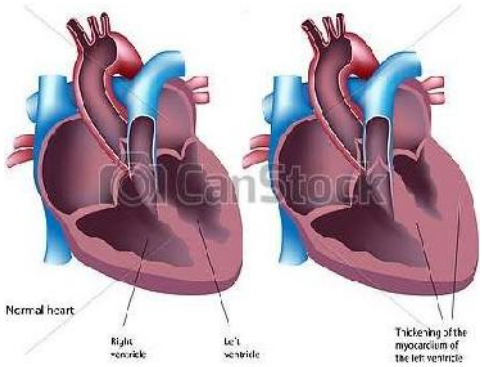
- There is high demands of the body, which are not met even with increased cardiac output
- e.g.: anemia, pregnancy

Low output HF:

- There is decreased contractility of heart leading to decreased cardiac output
- e.g.: cardiomyopathy, valvular disease

Left sided (left ventricular) HF

- Excess fluid accumulates upstream
- Reduction in left ventricular output
- Increase in left atrial pressure
- Increase in pulmonary venous pressure



➢ Acute increase in left atrial pressure causes pulmonary congestion and pulmonary edema e.g.: MI

➢ Gradual increase in left atrial pressure causes reflex pulmonary hypertension but no pulmonary edema

e.g. : aortic stenosis

Right sided (right ventricular) HF:

- Excess fluid accumulates upstream behind the failing right ventricle

- Reduction in right ventricular output
- Results in systemic venous congestion

Ex: pulmonary valvular stenosis, multiple pulmonary emboli

Systolic HF:

- Characterized by an abnormality of ventricular contraction
- As seen in ischemic heart failure and dilated cardiac myopathy

Diastolic HF:

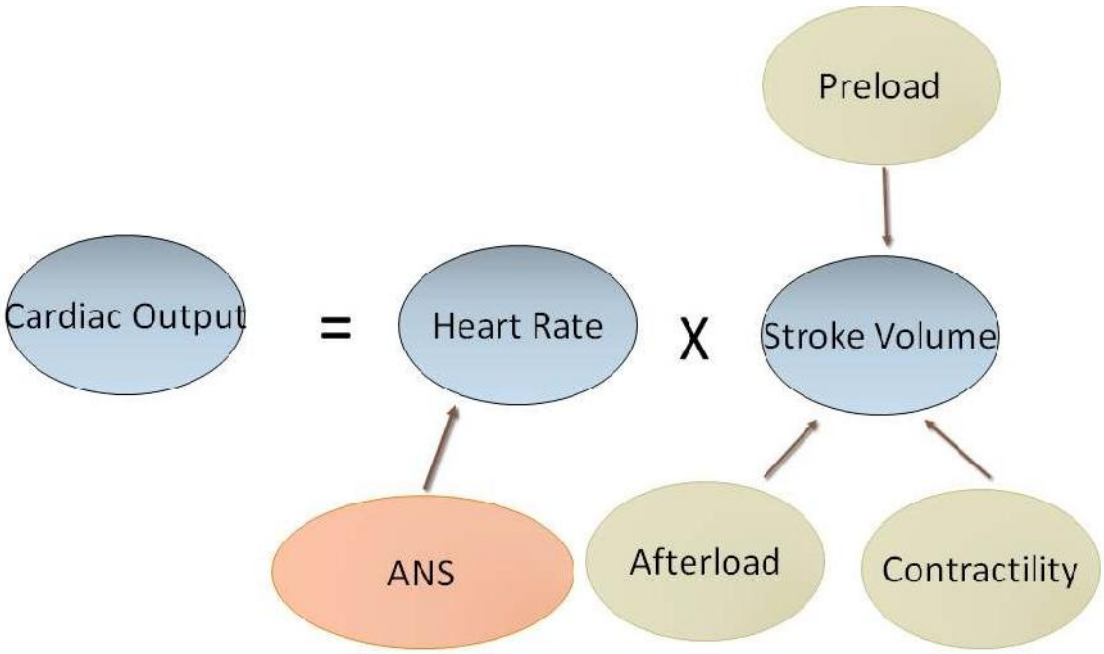
- Characterized by an impaired ventricular relaxation
- Increased ventricular stiffness resulting in diastolic dysfunction

e.g.**:** ischemia, left ventricular hypertrophy

PATHOPHYSIOLOGY

Preload: Pressure that fills the left ventricle during diastole

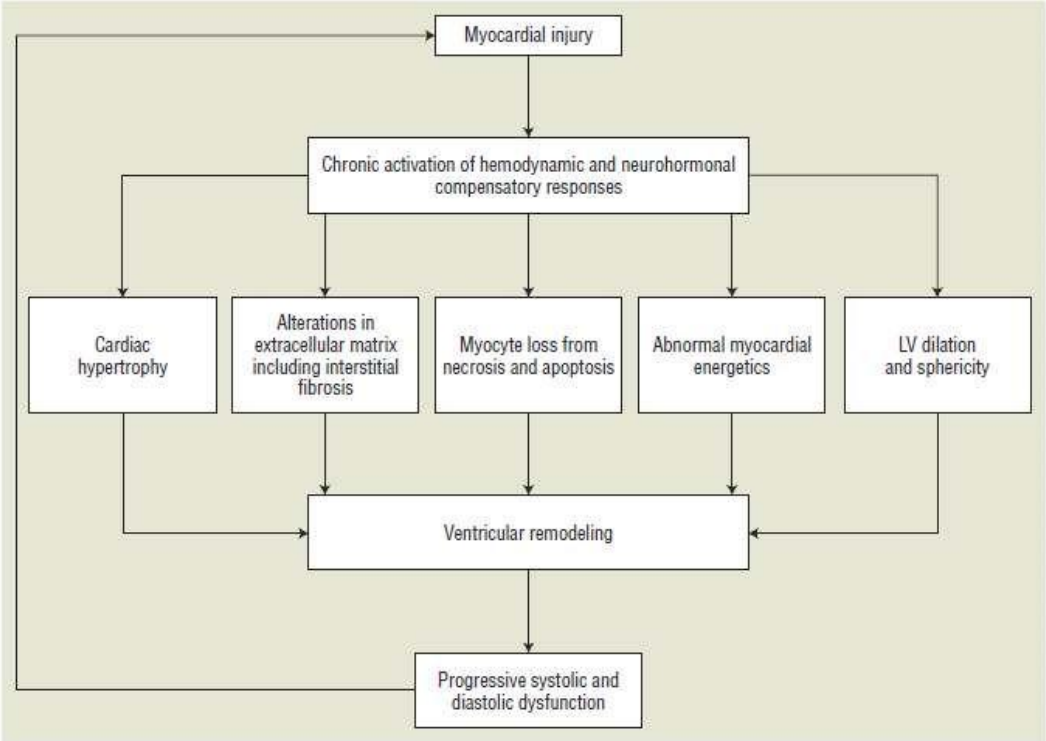
- **Main Determinant**- left ventricular compliance and venous return
- Small increase in end-diastolic volume
- large increase in cardiac output
- Primary compensatory mechanism in normal heart
- Ability of heart to alter the force of contraction depends on preload



Afterload: Pressure against which the left ventricular contracts and is measured as the mean aortic pressure

- Main determinants - total peripheral resistance and left ventricle size
- Left ventricular dysfunction - an inverse relationship exist between afterload and stroke volume
- An increase in afterload causes a decrease in stroke volume

Key components of the pathophysiology of cardiac remodelling



Compensatory Mechanisms

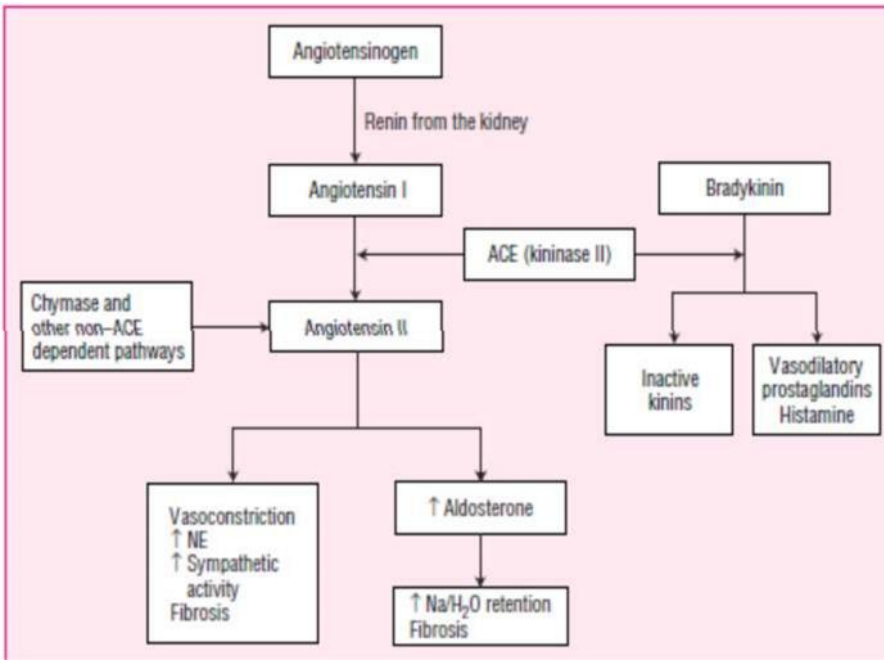
Cardiac compensatory mechanism:

- Ventricular dilation
- Ventricular hypertrophy

Peripheral compensatory mechanisms:

- Increased sympathetic activity
- Activation of renin angiotensin aldosterone system
- Increased release of arginine vasopressin

Renin Angiotensin Aldosterone system



Clinical Presentation – Signs

- Pulmonary edema
- Pleural effusion
- Tachycardia
- Cardiomegaly
- Peripheral edema
- Jugular venous distension
- Hepatojugular reflux
- Hepatomegaly

SYMPTOMS

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Exercise intolerance
- Tachypnea
- Ascites, Mental status changes
- Cough
- Fatigue
- Nocturia
- Hemoptysis
- Abdominal pain, Anorexia, Nausea, Bloating

SUMMARY

- CHF is a clinical syndrome that impairs the ability of the ventricle to fill with or eject blood
- Heart is unable to pump blood at a rate sufficient to meet the metabolic demands of the body
- It can be classified as: Acute and chronic heart failure,
- High output and low output heart failure,
- Left sided, right sided and biventricular heart failure,
- Systolic and diastolic heart failure