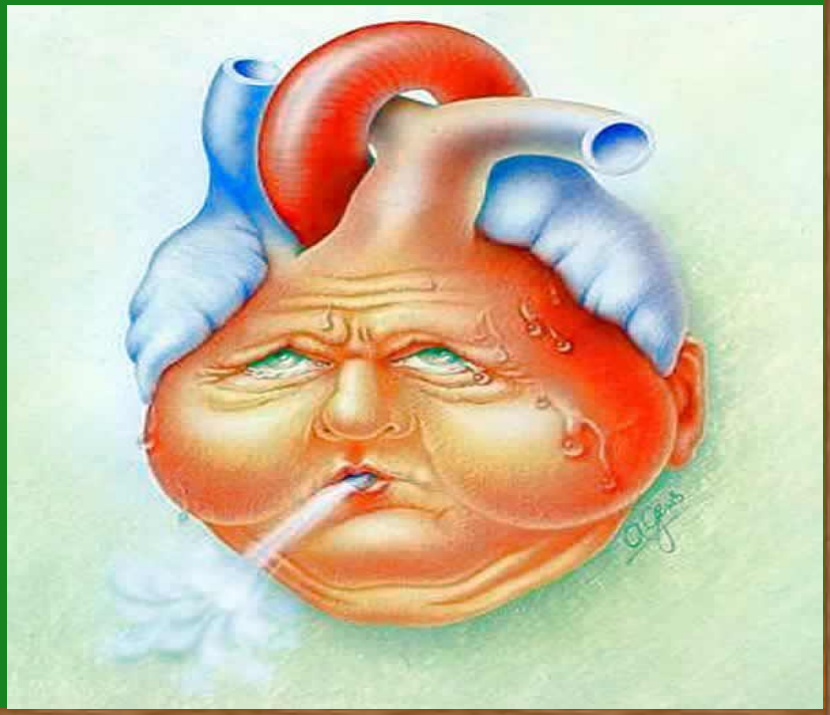
Heart failure

by Dr. swathi swaroopa. B



- Definition
- Etiology
- Symptoms
- Types of heart failure
- Pathophysiology

Definition

- Heart failure (HF) is a clinical syndrome caused by the inability of the heart to pump sufficient blood to meet the metabolic needs of the body.

- Cardiac output (CO) is defined as the volume of blood ejected per unit time (L/ min)and is the product of heart rate (HR) and stroke volume (SV):

$$CO = HR \times SV$$

Control of HR

Autonomic nervous system

Control of SV

preload

contractility

afterload

Preload

Volume of blood stretching the heart muscle at the end of diastole is normally determined mainly by venous return to heart.

After load

It is the force that the contracting muscle must generate to eject the blood from the filled heart.

Myocardial contractility

Contractile performance of heart or ability of contractile elements (actin and myosin filaments) of the heart muscle to interact and shorten against load.

Types of Heart failure

- Systolic dysfunction
- Diastolic dysfunction

- Left ventricular dysfunction
- Right ventricular dysfunction

- High output
- Low output

- Systolic dysfunction---- pulmonary congestion
- Diastolic dysfunction----pulmonary congestion

- Right sided heat failure---peripheral edema

Etiology

- HF can result from any disorder that reduces ventricular filling (diastolic dysfunction) and/or myocardial contractility (systolic dysfunction).

- Systolic dysfunction (decreased contractility)

- Reduction in muscle mass
- Dilated cardiomyopathies
- Ventricular hypertrophy

  Pressure overload

  Volume overload

Etiology ……

- **Diastolic dysfunction (restriction in ventricular filling)**

- Increased ventricular stiffness
  - Ventricular hypertrophy
  - Infiltrative myocardial diseases
  - Myocardial ischemia and infarction
- Mitral or tricuspid valve stenosis
- Pericardial disease

Others

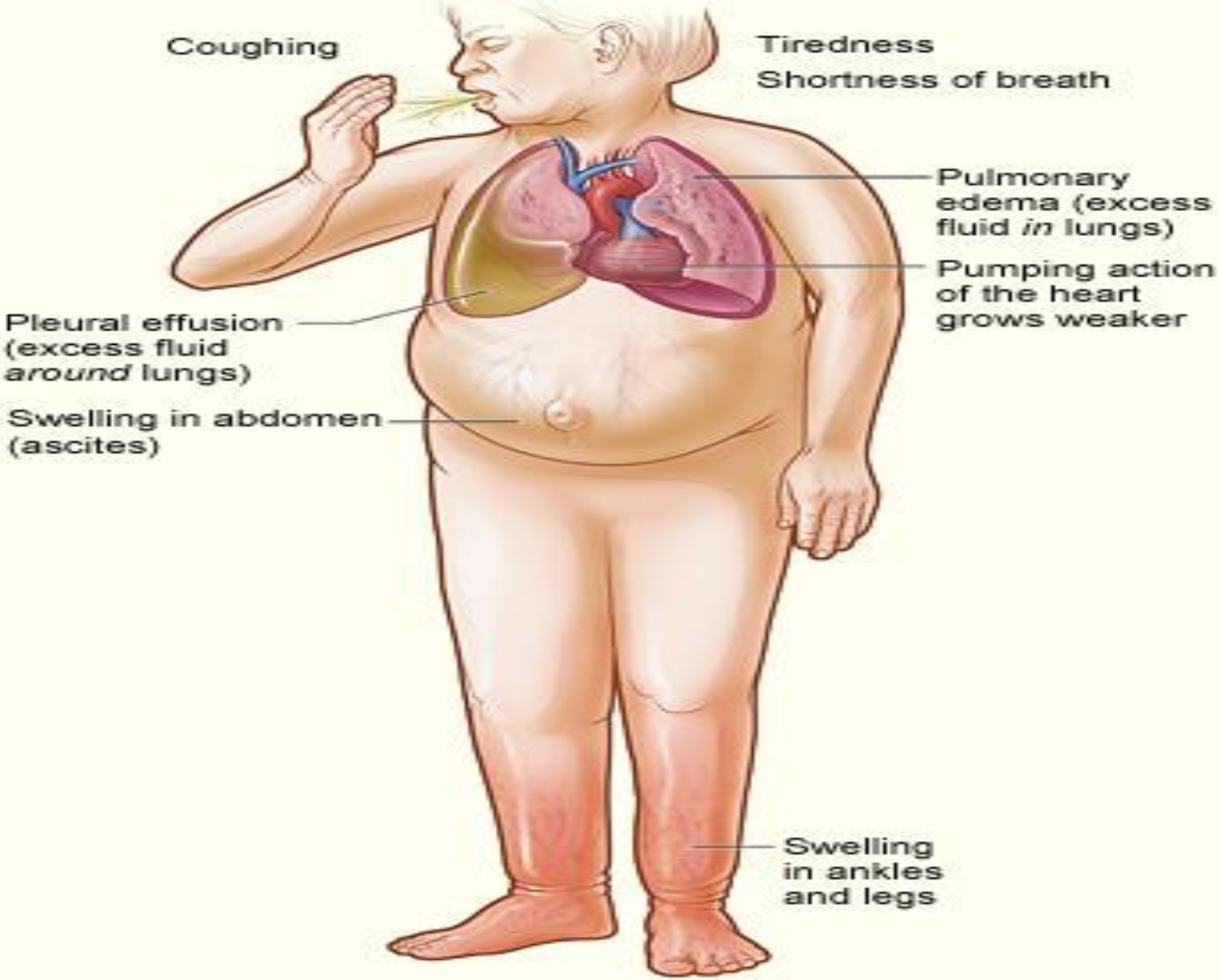
Pulmonary Heart Disease

Symptoms of left-side heart failure

- Fatigue
- Dyspnea
- Othopnea
- nocturnal dyspnea
- Pulmonary edema
- Cough up with blood-tinged sputum

Symptoms of right-side heart failure

- Peripheral Edema
- Hepatomegaly
- Ascites
- Nocturia


Coughing
Tiredness
Shortness of breath
Pulmonary edema (excess fluid *in* lungs)
Pumping action of the heart grows weaker
Pleural effusion (excess fluid *around* lungs)
Swelling in abdomen (ascites)
Swelling in ankles and legs

New York Heart Association classification of heart failure based on severity of the condition.

Class I: No limitations

Class II: Slight limitation of physical activity

Class III: Marked limitation of physical activity

Class IV: inability to carry on any physical activity without discomfort; symptoms of cardiac insufficiency or chest pain possible even at rest

ACC/AHA heart failure staging system.

Stage A

Patients at high risk for developing heart failure

Stage B

Patients with structural heart disease but no HF signs or symptoms

Stage C

Patients with structural heart disease and current or previous symptoms

Stage D

Refractory HF requiring specialized interventions

Pathophysiology

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Index event i.e. Damage of heart muscle, with a resultant loss of functioning cardiac myocytes, or alternatively disrupts the ability of the myocardium to generate force, thereby preventing the heart from contracting normally.

Force of contraction of heart decreases

Decreases cardiac output and stroke volume

Venous return remains un altered

End diastolic volume is increased

The decrease in the heart's pumping capacity results in the heart to rely on compensatory responses to maintain an adequate cardiac output.

Longterm activation of these compensatory responses results in development and progression of CCF.

Compensatory responses include

(a) Tachycardia and increased contractility through sympathetic nervous system (SNS) activation,

(b) The Frank-Starling mechanism,

(c) Vasoconstriction, and

(d) Ventricular hypertrophy and remodeling

Tachycardia and increased contractility

Norepinephrine (NE) released from adrenergic nerve terminals.

Cardiac output increases with heart rate until diastolic filling becomes compromised

Increases myocardial oxygen demand.

If ischemia is induced, both diastolic and systolic function impairs, and stroke volume drop precipitously.

2.

The higher calcium concentration

Greater filament interaction during systole

Generating more tension.

↑ contractility

Fluid Retention and Increased Preload

preload

- The Frank-Starling law-Stretching the myocardial fibers during diastole by increasing end-diastolic volume (preload)→ ↑force of contraction during systole.

- As myocardial sarcomere length is stretched, the number of cross-bridges between thick and thin myofilaments increases, resulting in an increase in the force of contraction.

Reduced renal perfusion, increased sympathetic tone

Activation of the renin–angiotensin–aldosterone system (RAAS)

Renin

ACE

Angiotensin I → angiotensin II

aldosterone release

sodium and water retention

preload increases

Increase in preload will increase force of contraction, stroke volume to certain point.

- Further increases in preload will only lead to pulmonary or systemic congestion, a detrimental result.

Vasoconstriction and Increased Afterload

- Vasoconstriction occurs in patients with heart failure to redistribute blood flow away from nonessential organs to coronary and cerebral circulations

NE, angiotensin II, endothelin-1, and arginine vasopressin (AVP).

↓

Vasoconstriction

↓

Increase afterload

↓

Worsen HF state

Ventricular Hypertrophy and Remodeling

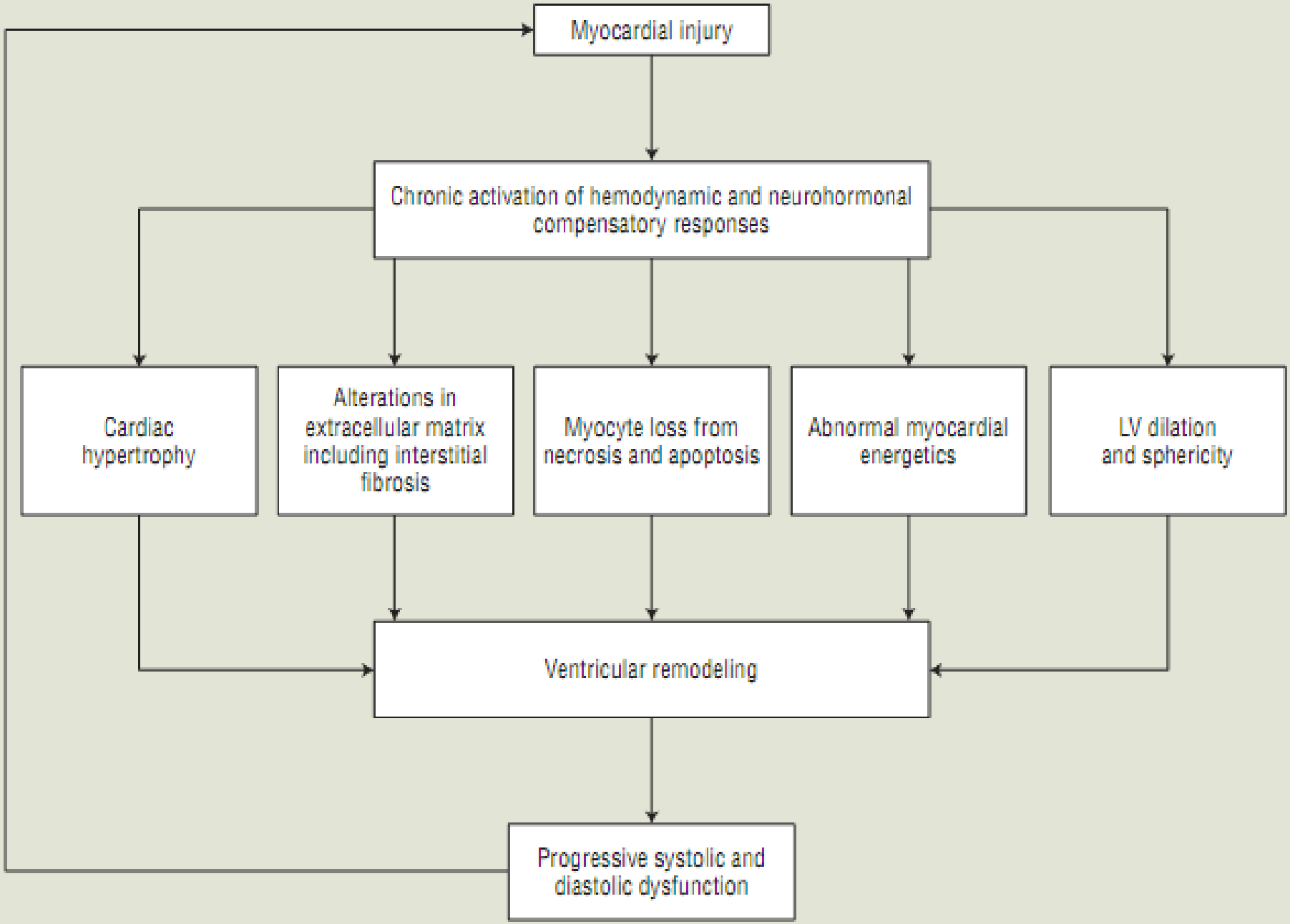
- **Ventricular hypertrophy** is a term used to describe an increase in ventricular muscle mass.
- **Cardiac or ventricular remodeling** is term describing changes in both **myocardial cells** and **extracellular matrix** that result in changes in the size, shape, structure, and function of the heart.

- Ventricular hypertrophy and remodeling can occur in association with any condition that causes myocardial injury including **MI, cardiomyopathy, hypertension, and valvular heart disease**.

Angiotensin II, NE, endothelin, aldosterone, vasopressin and numerous inflammatory cytokines,

activated both systemically and locally in the heart initiating the

signal–transduction cascade responsible for ventricular remodelling


Myocardial injury
Chronic activation of hemodynamic and neurohormonal compensatory responses
Cardiac hypertrophy
Alterations in extracellular matrix including interstitial fibrosis
Myocyte loss from necrosis and apoptosis
Abnormal myocardial energetics
LV dilation and sphericity
Ventricular remodeling
Progressive systolic and diastolic dysfunction

Compensatory Response	Beneficial Effects of Compensation	Detrimental Effects of Compensation
Increased preload (through Na^+ and water retention)	Optimize stroke volume via Frank-Starling mechanism	Pulmonary and systemic congestion and edema formation Increased MVO_2
Vasoconstriction	Maintain BP in face of reduced CO Shunt blood from nonessential organs to brain and heart	Increased MVO_2 Increased afterload decreases stroke volume and further activates the compensatory responses
Tachycardia and increased contractility (due to SNS activation)	Helps maintain CO	Increased MVO_2 Shortened diastolic filling time β_1-Receptor downregulation, decreased receptor sensitivity Precipitation of ventricular arrhythmias Increased risk of myocardial cell
Ventricular hypertrophy and remodeling	Helps maintain CO Reduces myocardial wall stress Decreases MVO_2	Diastolic dysfunction Systolic dysfunction Increased risk of myocardial cell death Increased risk of myocardial ischemia Increased arrhythmia risk Fibrosis