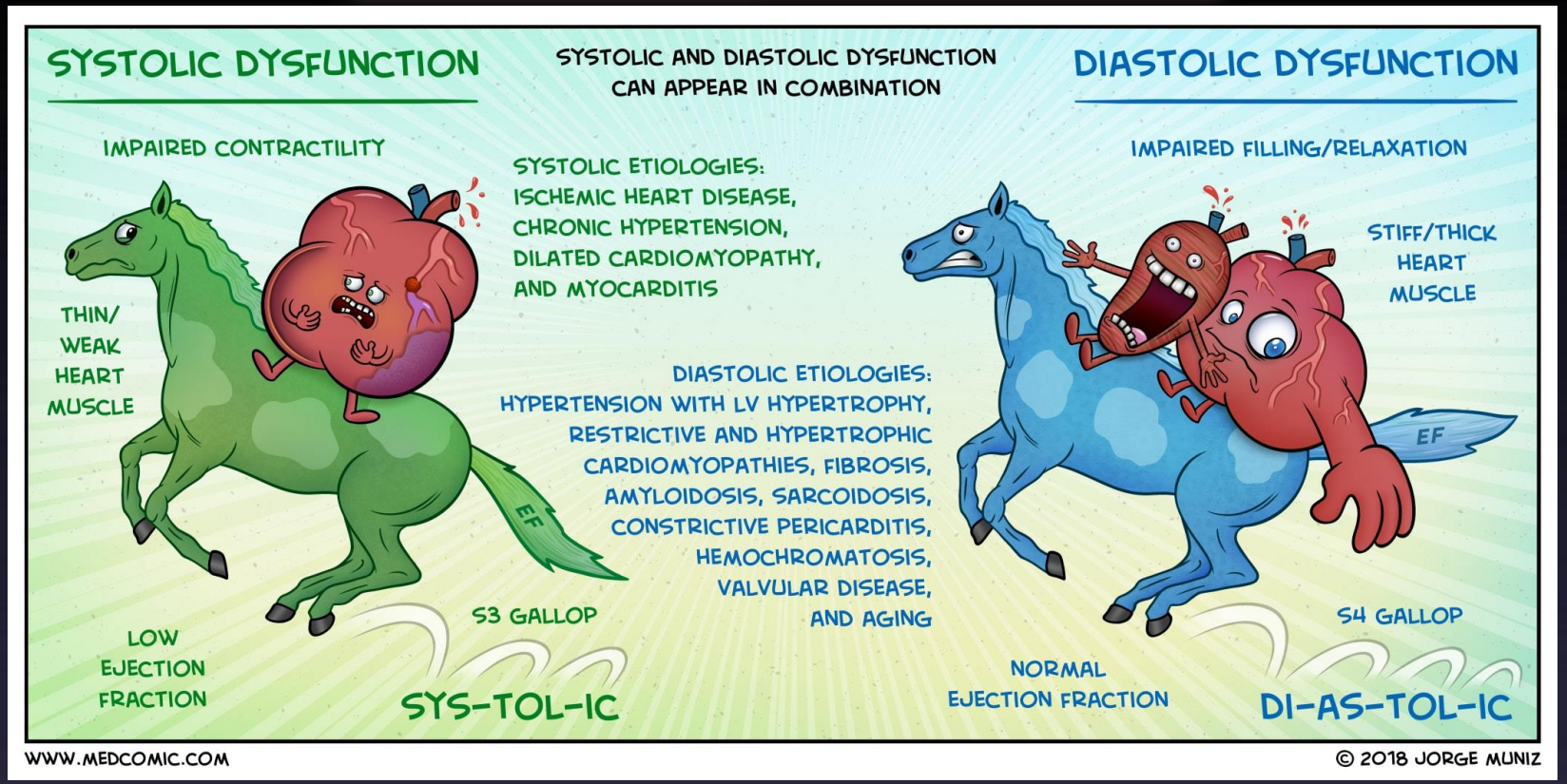
CARDIOVASCULAR SYSTEM

# JUGULAR VENOUS PULSE & HEART FAILURE



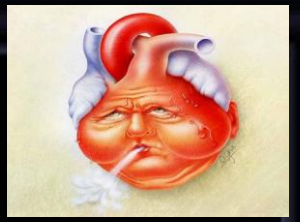

**Prof. Shahid Habib**
Dept of Physiology
King Saud University

# OBJECTIVES

## At the end of the lecture you should be able to …

- Describe Normal pattern of the jugular venous pulse
- Define and classify heart failure.
- Describe the causes and pathophysiological consequences of acute and chronic heart failure.
- Explain how left-sided failure leads to right-sided failure & congestive heart failure.
- Discuss the compensatory mechanisms in heart failure.
- Summarize clinical picture of left-sided and right-sided failure.
- Interpret and draw Starling curves for healthy heart, acute failure, and failure treated with digoxin.

# Heart Failure



- It is the pathophysiological process in which the heart as a pump is unable to meet the metabolic requirements of the tissue for oxygen and substrates despite the venous return to heart is either normal or increased.
- Thus, the resting CO may be low, normal or even elevated, despite the presence of heart failure as long as this level is inadequate for body organs need of blood and $O_2$.

Heart failure can involve the left or right side of the heart or both.

Usually the left side is affected first.

Manifested mainly by:

- Inadequate cardiac output.
- Build-up of blood in veins behind left heart or right heart (increased venous pressure).

# Heart Failure

# Causes

## 1- Impaired cardiac function

- Coronary heart disease
- Cardiomyopathies (muscle disease)
- Rheumatic fever
- Endocarditis
- Cardiac arrhythmias: e.g., complete heart block

## 2- Increased cardiac workload

- Hypertension
- Valvular disorders
- Anemias
- Congenital heart defects

## 3- Acute non-cardiac conditions

- Volume overload
- Hyperthyroidism, Fever, Infection

# Heart Failure

## Systolic failure

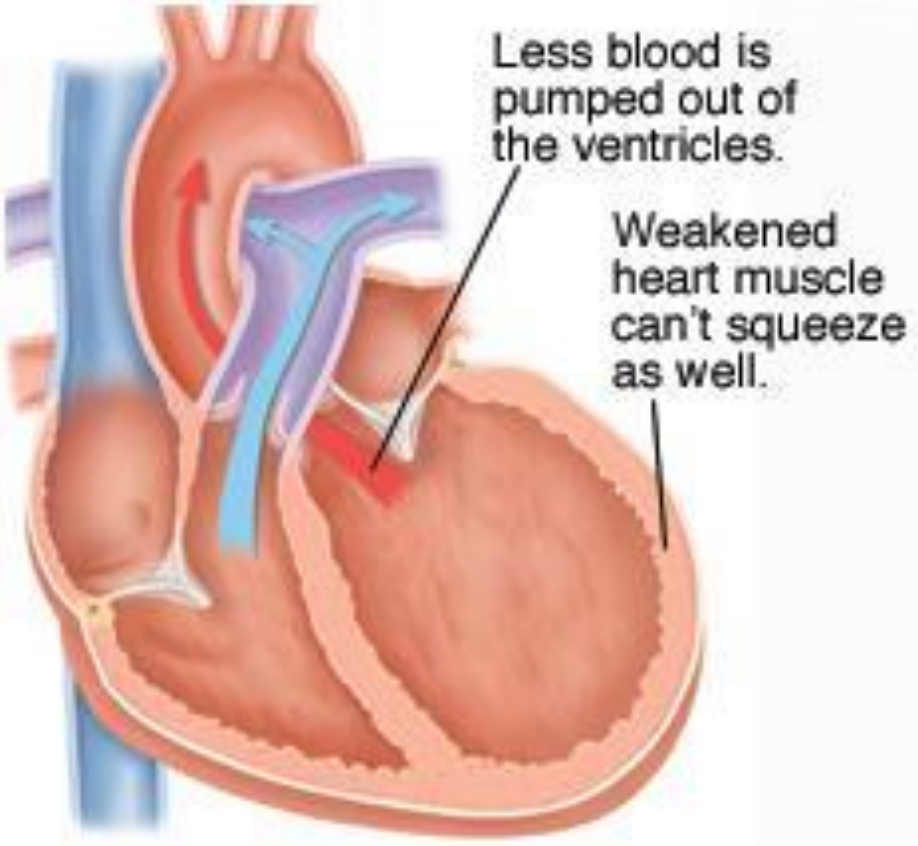
- The heart looses it's ability to contract or pump blood into the circulation

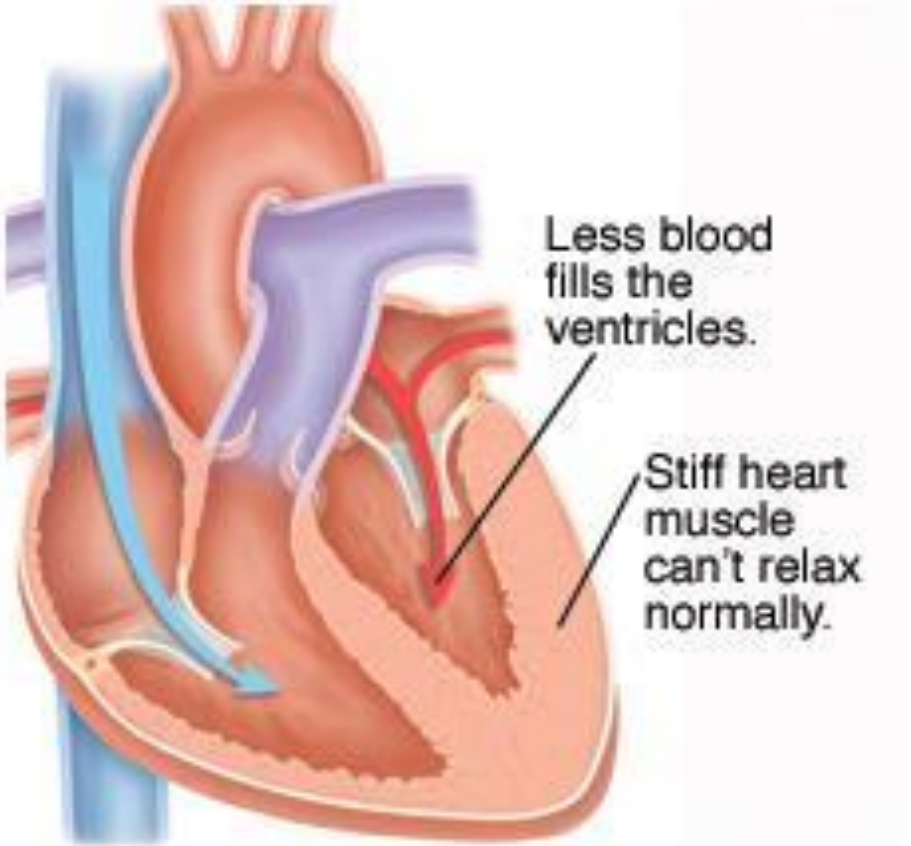
## Diastolic failure

- The heart looses it's ability to relax because it becomes stiff
- Heart cannot fill properly between each beat

| Characteristic | Diastolic Heart Failure | Systolic Heart Failure |
|---|---|---|
| Age | Frequently elderly | All ages, typically 50–70 yr |
| Sex | Frequently female | More often male |
| Left ventricular ejection fraction | Preserved or normal, approximately 40% or higher | Depressed, approximately 40% or lower |
| Left ventricular cavity size | Usually normal, often with concentric left ventricular hypertrophy | Usually dilated |
| Left ventricular hypertrophy on electrocardiography | Usually present | Sometimes present |
| Chest radiography | Congestion with or without cardiomegaly | Congestion and cardiomegaly |
| Gallop rhythm present | Fourth heart sound | Third heart sound |

**Congestive heart failure**
**Chronic left HF results in secondary pulmonary hypertension and right HF**




**Systolic heart failure.** The heart muscle becomes weak and enlarged. It can't pump enough blood forward when the ventricles contract. Ejection fraction is lower than normal.

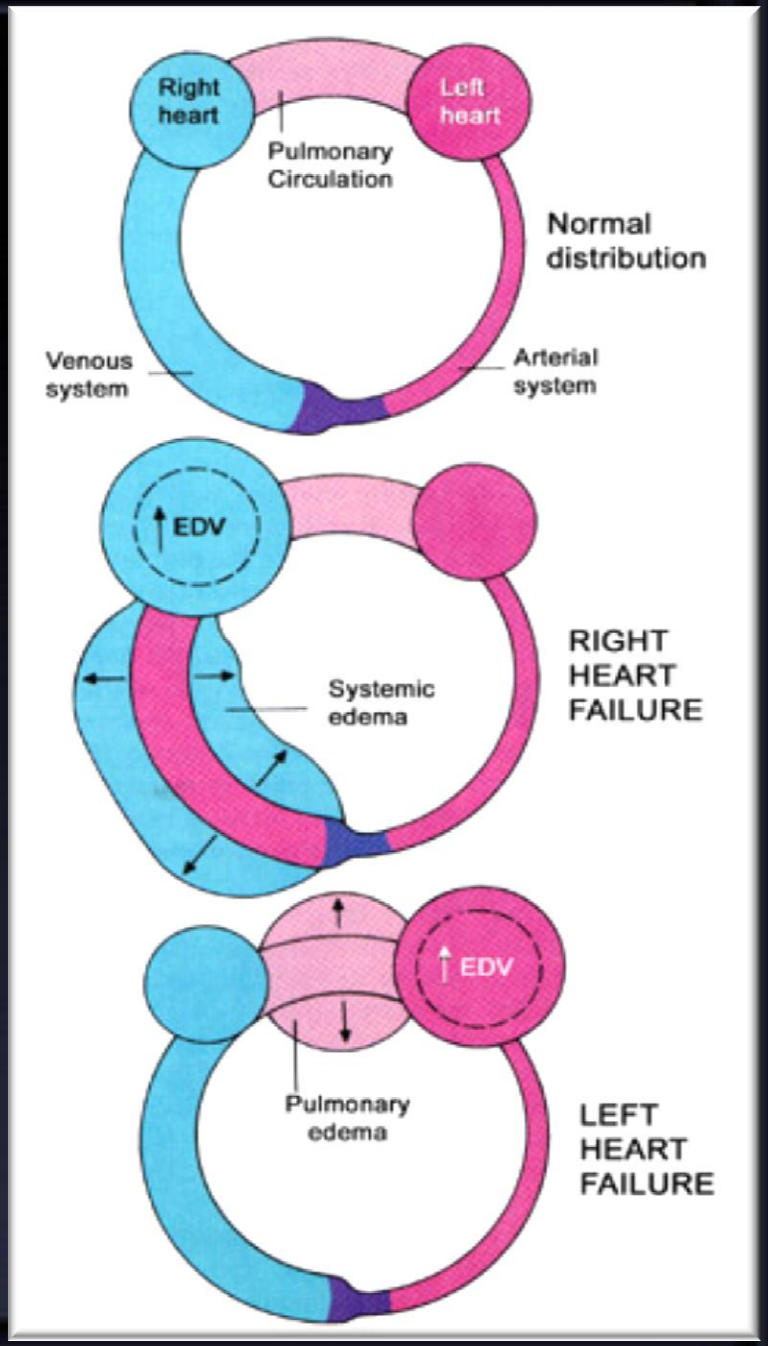
**Diastolic heart failure.** The heart muscle becomes stiff. It doesn't relax normally between contractions, which keeps the ventricles from filling with blood. Ejection fraction is often in the normal range.

# Left vs. right heart failure:

- In left-sided failure, VR pulmonary circulation is not pumped out by the failing LV → blood accumulates in pulmonary circulation → ↑ the pulmonary capillary pressure → pulmonary edema

- In right-sided failure, VR from systemic circulation is not pumped out by the failing RV → blood accumulates in systemic circulation increasing the systemic capillary pressure → systemic edema.

- Though each side of the heart can undergo failure separately, dysfunction of one side may lead to a sequence of events that make the opposite side also to fail.



# LEFT HF ⟵ Causes ⟶ RIGHT HF

## Systolic Dysfunction

- **Impaired Contractility**
  - Myocardial infarction
  - Transient ischemia
  - Chronic volume overload
  - MR/AR
- **Increased Afterload**
  - Aortic Stenosis
  - Uncontrolled HTN

## Diastolic Dysfunction

- **Obstruction of LV filling**
  - Mitral Stenosis
  - Pericardial constriction or tamponade
- **Impaired ventricular relaxation**
  - Hypertrophic or restrictive cardiomyopathy
  - Transient ischemia

## Cardiac Causes

- **Usually occurs as a result of left HF**
- **Pulmonary stenosis**
- **Right ventricular infarction**

## Pulmonary Vascular Disease

- **Pulmonary emobolism**
- **Pulmonary HTN**
- **Right ventricular infarction**

## Pulmonary Parenchymal disease

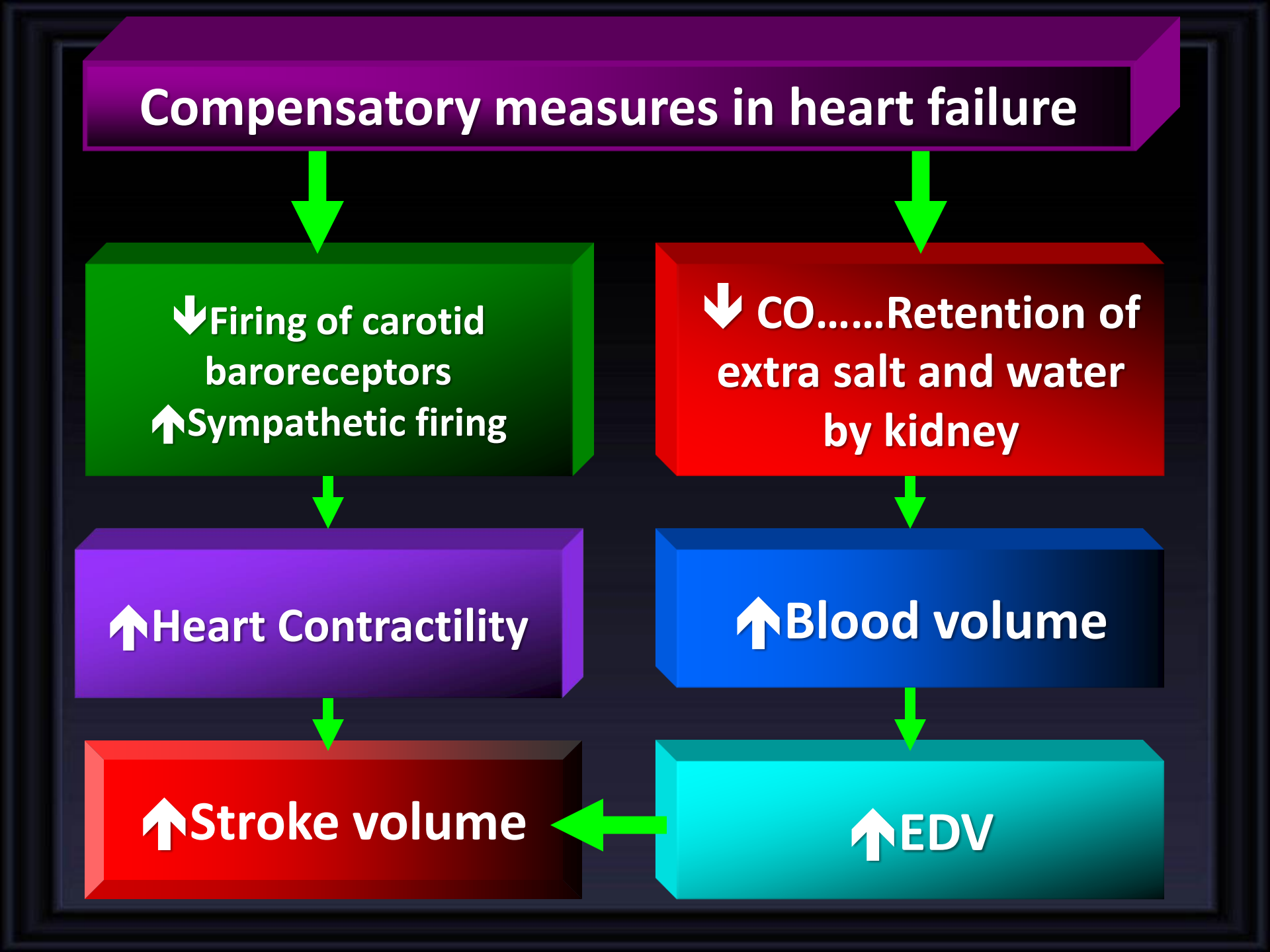
- **COPD**
- **Interstitial lung disease**
- **Chronic infections**
- **Adult respiratory distress syndrome**

**COR PULMONALE**
**Right HF due to chronic lung disease**

# Acute vs chronic heart failure:

Sudden serious abnormalities of the heart (e.g., massive infarction, arrhythmias, valve rupture; sepsis) → acute heart failure (hours / days) ……. can be life threatening because the heart does not have time to undergo compensatory adaptations. [usually left-sided]…….Cardiogenic shock develops following acute failure if the heart became unable to pump enough to even keep tissues alive.

Chronic heart failure is a long-term condition (months/years) …. It is associated with the heart undergoing adaptive responses (e.g., dilation, hypertrophy)……Which can be deleterious.


Compensatory measures in heart failure
↓Firing of carotid baroreceptors
↑Sympathetic firing
↓ CO……Retention of extra salt and water by kidney
↑Heart Contractility
↑Blood volume
↑Stroke volume
↑EDV

## Physiological adaptation to CHF

# compensatory mechanisms

## *can be deleterious?*

1-Decreased firing of carotid sinus baroreceptor → increased sympathetic stimulation:

→ vasoconstriction of arterioles (increased afterload),

→ vasoconstriction of veins (increased preload).

→ increased HR and force of contractility.

→ increased CO and increased BP.

ANP and BNP are major antagonizing agents of the renin–angiotensin–aldosterone system.

2-Decreased renal perfusion → Activation of RAA system

3-Decreased effective circulating blood volume → posterior pituitary releases ADH (vasopressin) → increased $H_2O$ reabsorption.

# Complications of progressive heart failure
# Factors contributing to decompensation

1) Prolonged sympathetic activation to the heart: down regulation of the myocardial adrenergic receptors → ↓ the myocardial adrenergic receptors density and sensitivity to catecholamines. Consequently, the inotropic and chronotropic reponses of the heart cannot be elevated in parallel to increased body requirements.
2) Vasoconstriction of the arterioles (under enhanced sympathetic activity): This increases resistance, thus the cardiac afterload.
3) Hypertrophied heart: → imbalance between the $O_2$ supply and need → deterioration of the ability to generate force.
4) Excessive salt and water retention:
5) Over-distended ventricle: Has to consume more energy and generate more wall tension to develop the required ejection pressure (Laplace law).

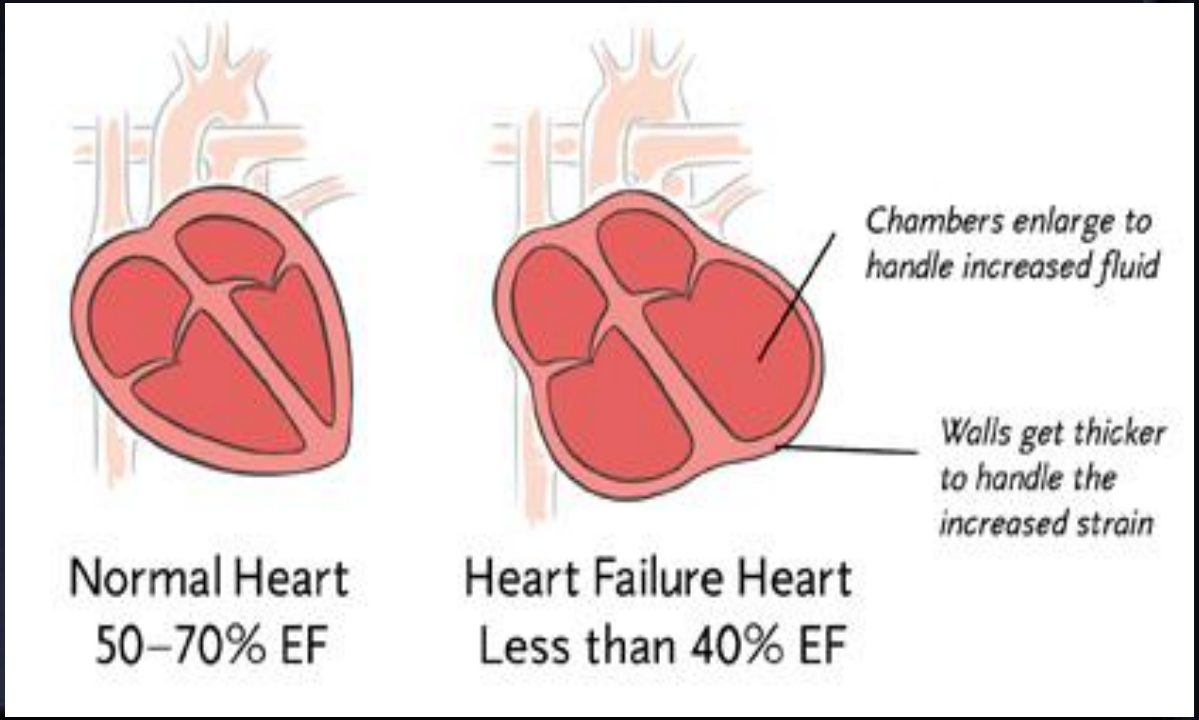
# INDICATOR FOR DIAGNOSING HEART FAILURE

## Ejection Fraction (EF)

- Ejection Fraction (EF) is the percentage of blood that is pumped out of your heart during each beat

## Fractional Shortening….

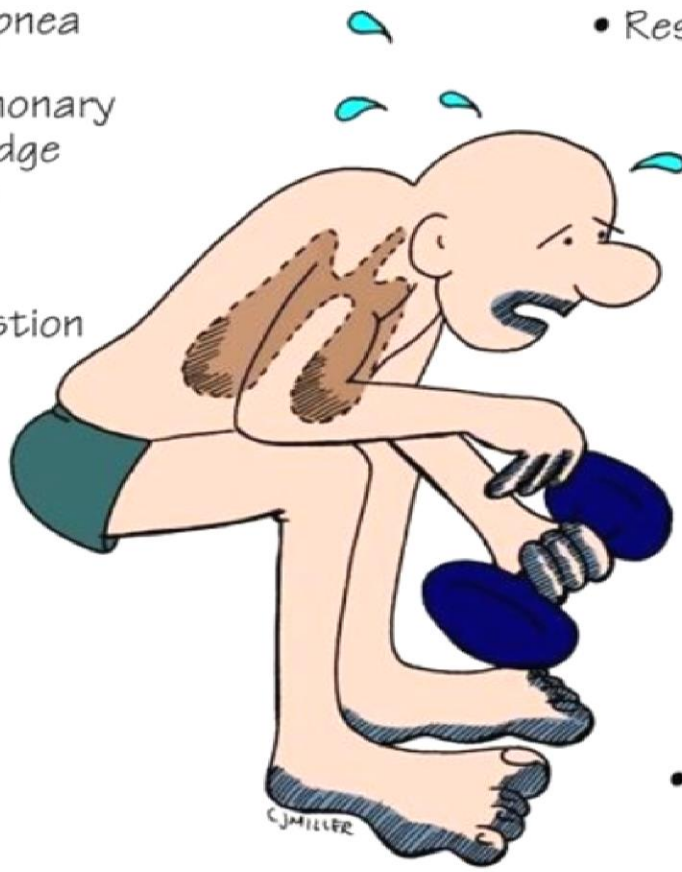
Fractional Shortening. Fractional Shortening: This is one of the most basic measures in adult functional echocardiography. It simply looks at the degree of shortening of the left ventricular diameter between end-diastole and end-systole.



# HOW HEART FAILURE IS DIAGNOSED

- Medical history is taken to reveal symptoms
- Physical exam is done
- Tests
  - Chest X-ray
  - Blood tests
  - Electrical tracing of heart (Electrocardiogram or "ECG")
  - Ultrasound of heart (Echocardiogram or "Echo")
  - X-ray of the inside of blood vessels (Angiogram)

# Clinical picture of left-sided failure

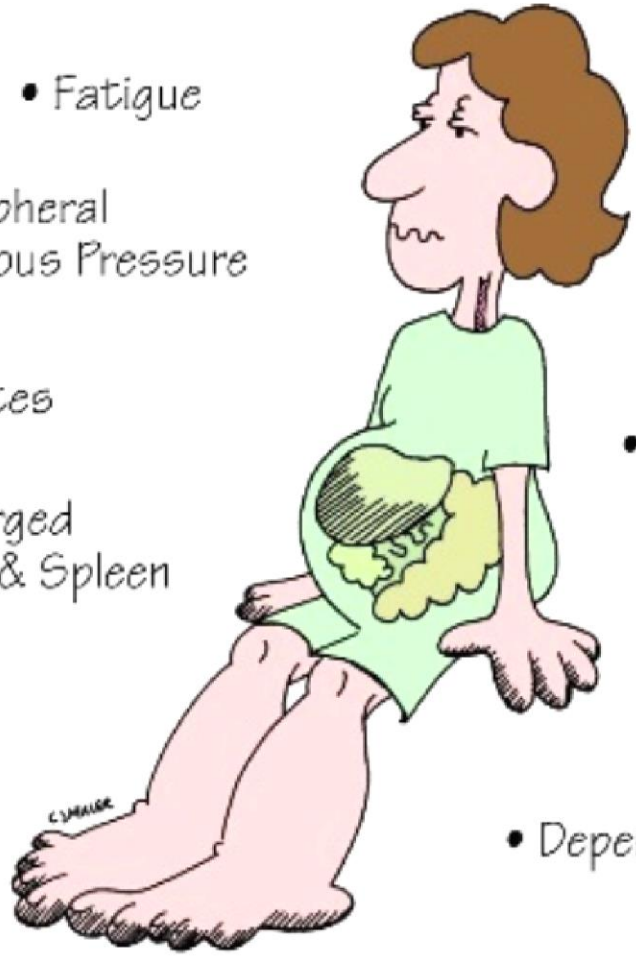



**Orthopnoea is shortness of breath (dyspnea) that occurs when lying flat, causing the person to have to sleep propped up in bed or sitting in a chair.**

**Paroxysmal nocturnal dyspnea refers to attacks of severe shortness of breath and coughing that generally occur at night. It usually awakens the person from sleep, and may be quite frightening.**

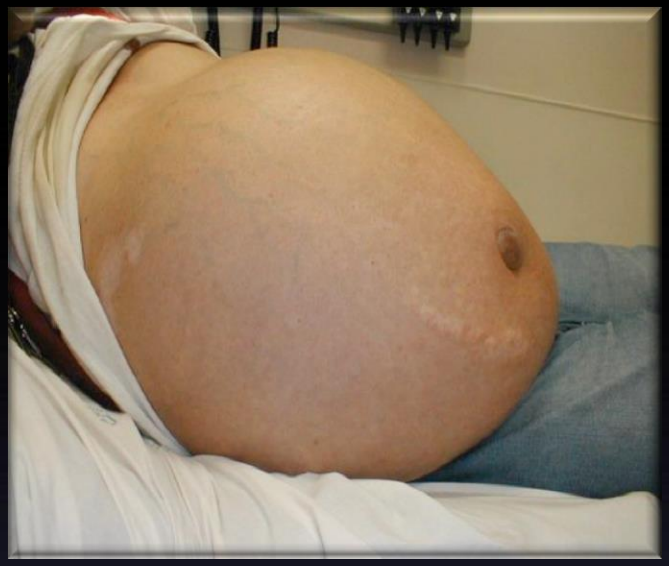
# Clinical picture of right-sided failure

(Cor Pulmonale)

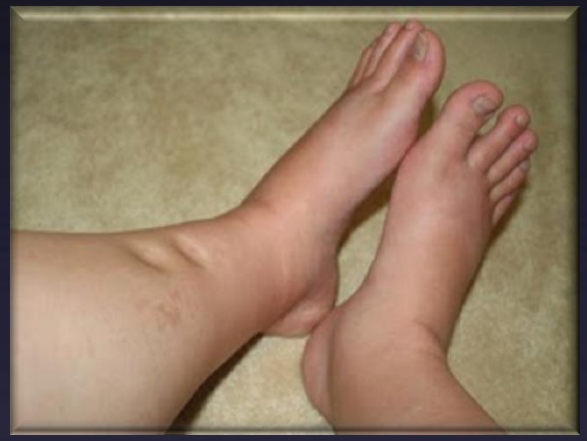


- Fatigue
- ↑ Peripheral Venous Pressure
- Ascites
- Enlarged Liver & Spleen
- May be secondary to chronic pulmonary problems
- Distended Jugular Veins
- Anorexia & Complaints of GI Distress
- Swelling in Hands & Fingers
- Dependent Edema

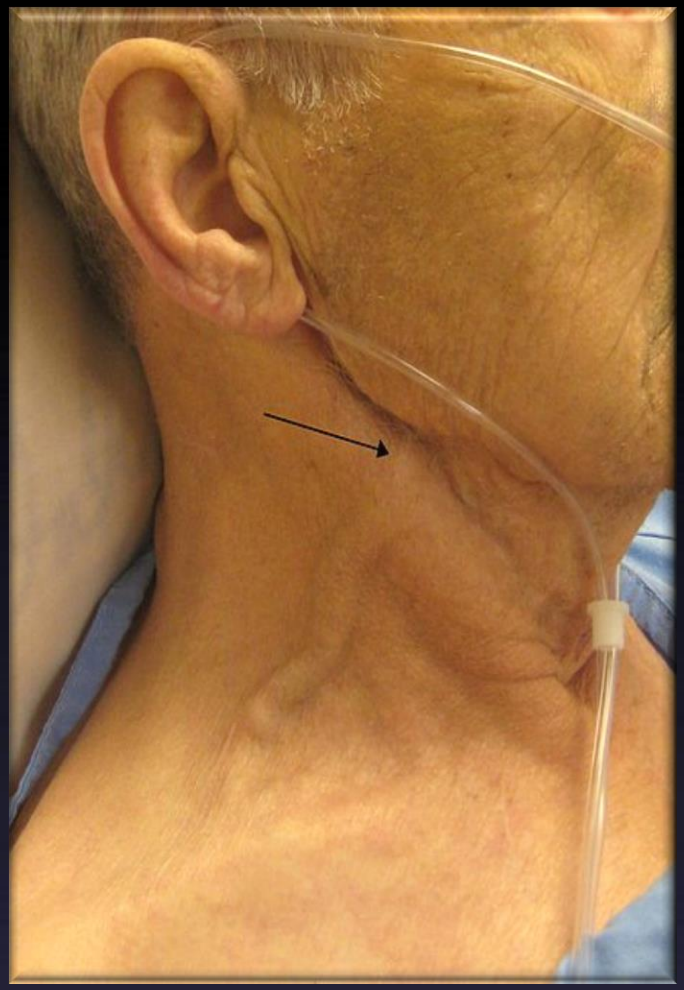
# Clinical picture of right-sided failure



(Ascites)



(Pitting edema)



(Elevated JVP)

# Abnormalities of jugular venous pulse

**A- Low jugular venous pressure**

- Hypovolemia.

**B- Raised Jugular Venous Pressure**

- Increased right ventricular filling pressure e.g in heart failure fluid overload.
- Obstruction of blood flow from the right atrium to the right ventricle e.g tricuspid stenosis.
- Superior vena caval obstruction e.g retrosternal thyroid goiter.
- Positive intrathoracic pressure e.g pleural effusion, pneumothorax.

**The JVP usually drops on inspiration along with intrathoracic pressure**

| Clinical picture | Left-sided failure | Right-sided failure |
|---|---|---|
| Pitting edema (legs, hands) | Mild to moderate | Moderate to severe |
| Fluid retention | Pulmonary edema (fluid in lungs), and pleural effusion (fluid in the pleural cavity) | Abdomen (ascites) |
| Organ enlargement | Heart | Liver. Mild jaundice may be present |
| Neck veins | Mild to moderate elevation in JVP | Sever elevation in JVP. Neck veins are visibly distended |
| Shortness of breath | Prominent dyspnea, paroxysmal nocturnal dyspnea, and orthopnea | Dyspnea is present but not as prominent |
| GIT symptoms | Present but not as prominent as in right-sided failure | Loss of appetite, bloating, constipation. Symptoms are significantly more prominent than in left-sided failure |

# Treatment of cardiac failure

Aims at

Reduction of salt & water retention

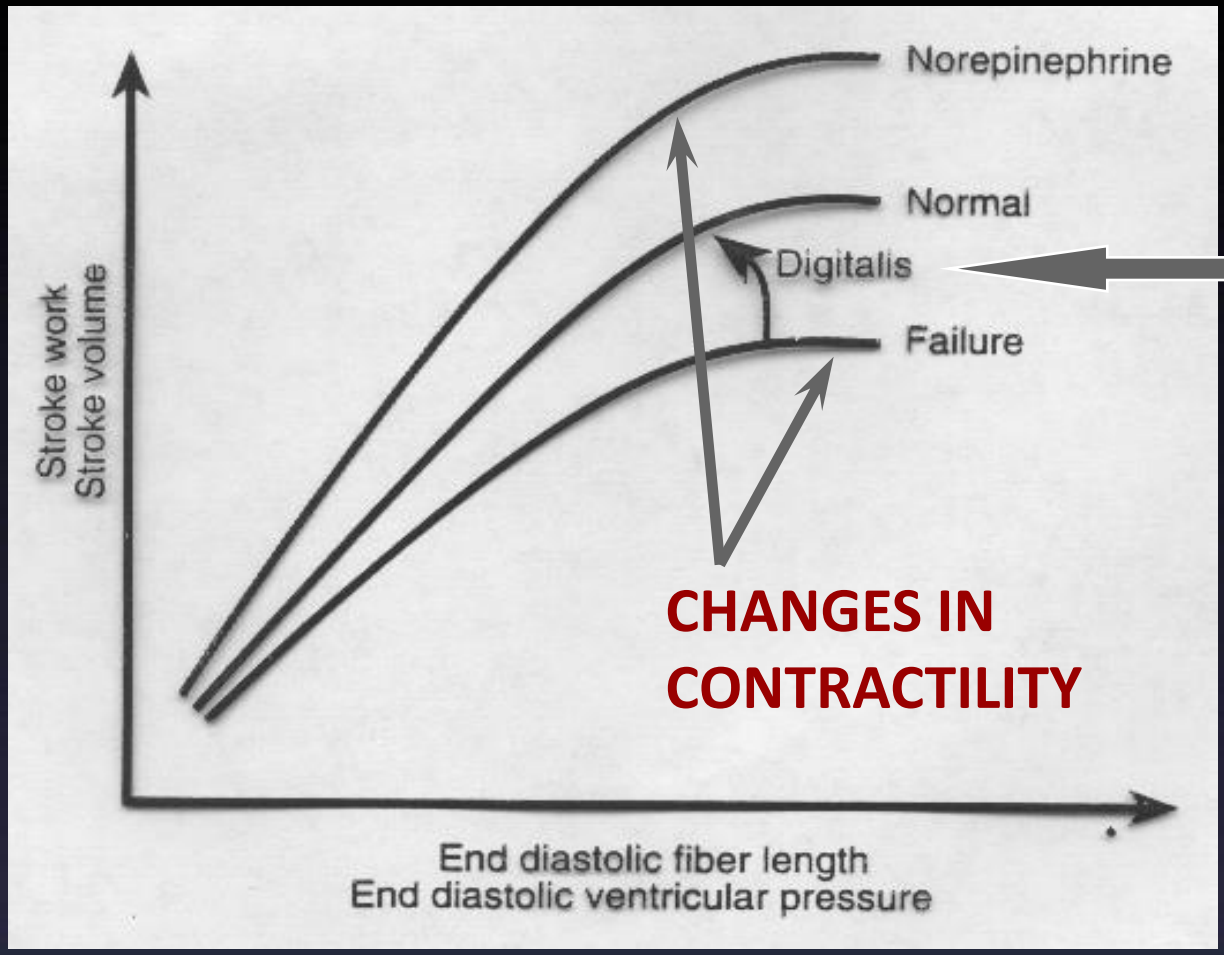
↑ in the pumping activity of failing heart

Diuretics

Digitalis

Digitalis improves pumping activity of heart by increasing cytosolic $Ca^{++}$

# Starling curve in heart failure



# SYSTOLIC DYSFUNCTION

**Impaired ventricular contraction**

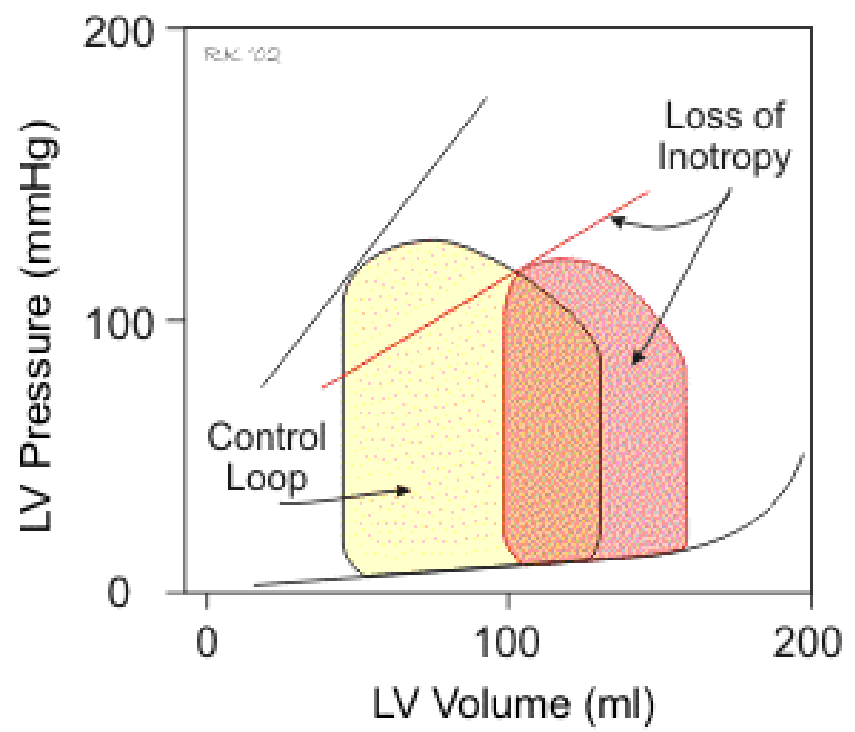



***Figure 2.*** Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

↓ slope of ESPVR i.e. ↑ ESV

Compensatory rise in preload i.e. ↑ EDV

↓ SV

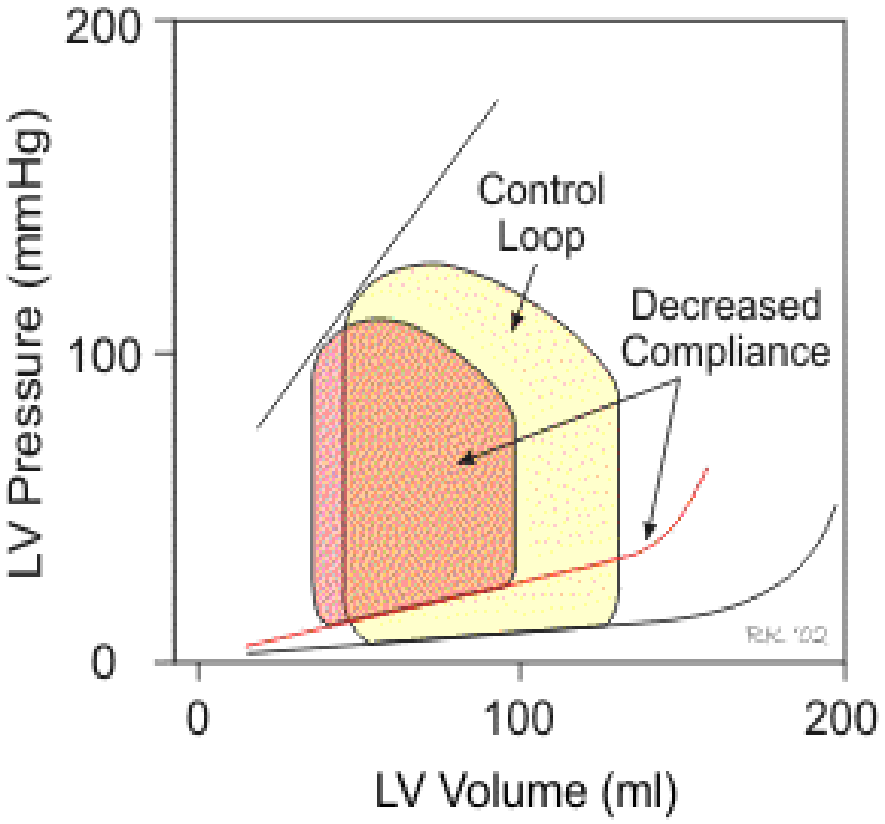
↓ EF

↓ Work Output of Heart

↑ EDP

end-systolic pressure-volume relationship (ESPVR)

# DIASTOLIC DYSFUNCTION




Effects of left ventricular diastolic failure caused by decreased ventricular compliance (e.g., hypertrophy) on left ventricular pressure-volume loop. Heart rate, inotropy and systemic vascular resistance are unchanged.

**Reduced venous return / compliance / relaxation (lusitropy)**

**↓ EDV**

**↓ SV**

**↓ or no change EF**

**↓ Work Output of Heart**

**↑ EDP**