

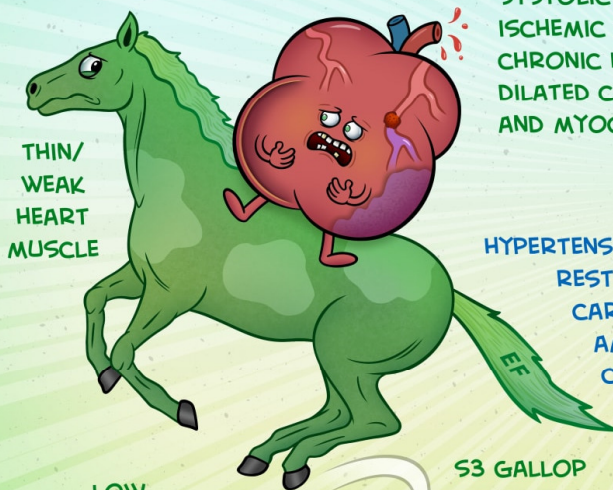
CARDIOVASCULAR SYSTEM

JUGULAR VENOUS PULSE & HEART FAILURE

SYSTOLIC DYSFUNCTION

IMPAIRED CONTRACTILITY

THIN/WEAK HEART MUSCLE



LOW EJECTION FRACTION

S3 GALLOP

SYSTOLIC AND DIASTOLIC DYSFUNCTION CAN APPEAR IN COMBINATION

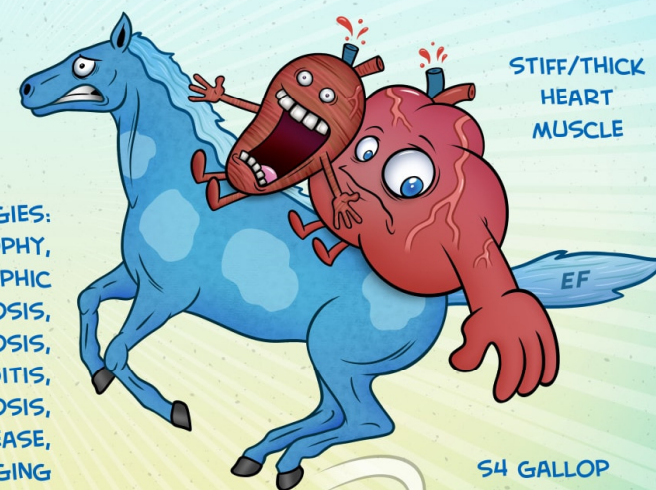
SYSTOLIC ETIOLOGIES:
ISCHEMIC HEART DISEASE,
CHRONIC HYPERTENSION,
DILATED CARDIOMYOPATHY,
AND MYOCARDITIS

DIASTOLIC ETIOLOGIES:
HYPERTENSION WITH LV HYPERTROPHY,
RESTRICTIVE AND HYPERTROPHIC
CARDIOMYOPATHIES, FIBROSIS,
AMYLOIDOSIS, SARCOIDOSIS,
CONSTRICTIVE PERICARDITIS,
HEMOCHROMATOSIS,
VALVULAR DISEASE,
AND AGING

DIASTOLIC DYSFUNCTION

IMPAIRED FILLING/RELAXATION

STIFF/THICK HEART MUSCLE



NORMAL EJECTION FRACTION

S4 GALLOP

DI-AS-TOL-IC

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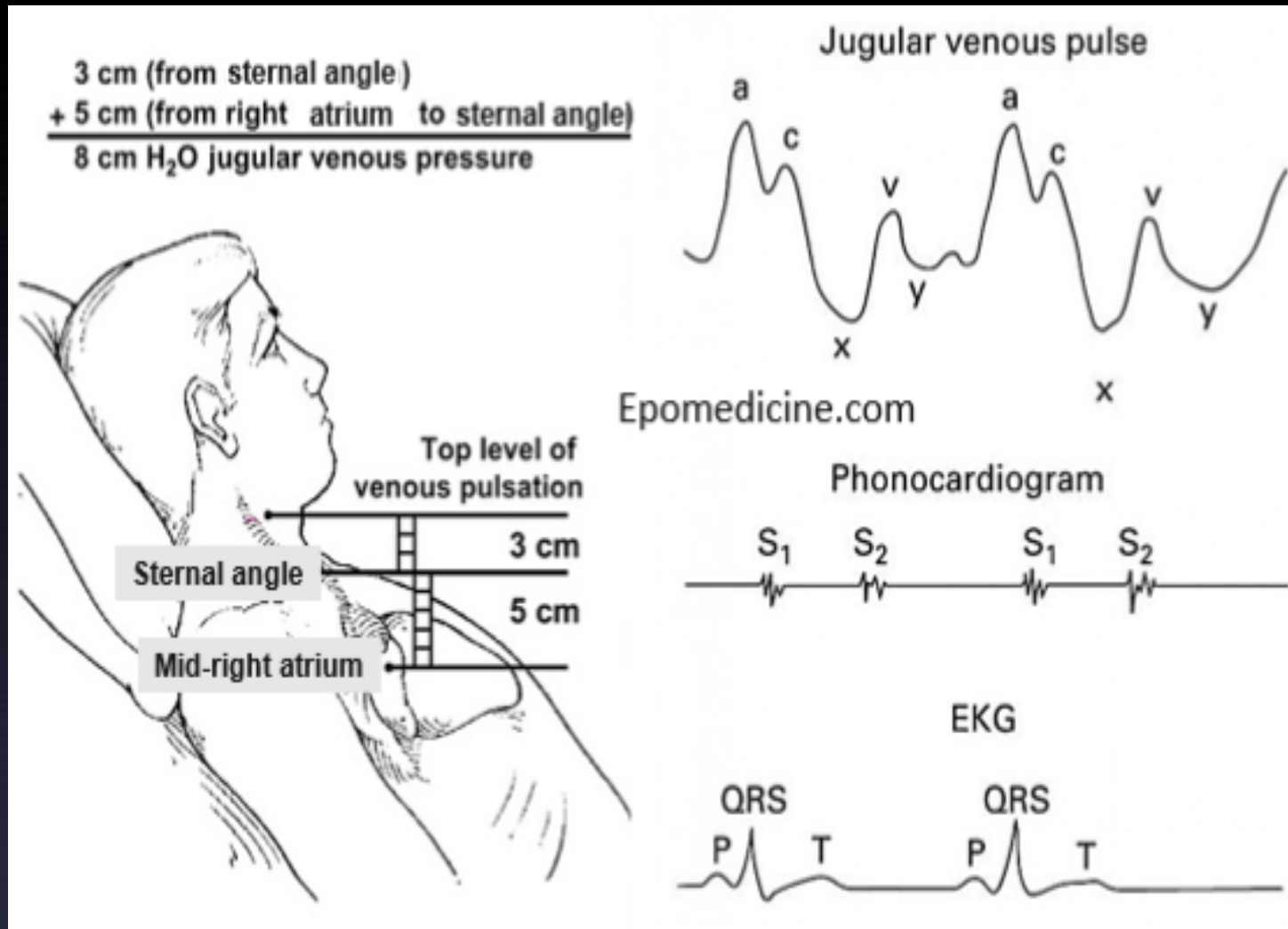
OBJECTIVES

At the end of the lecture you should be able to ...

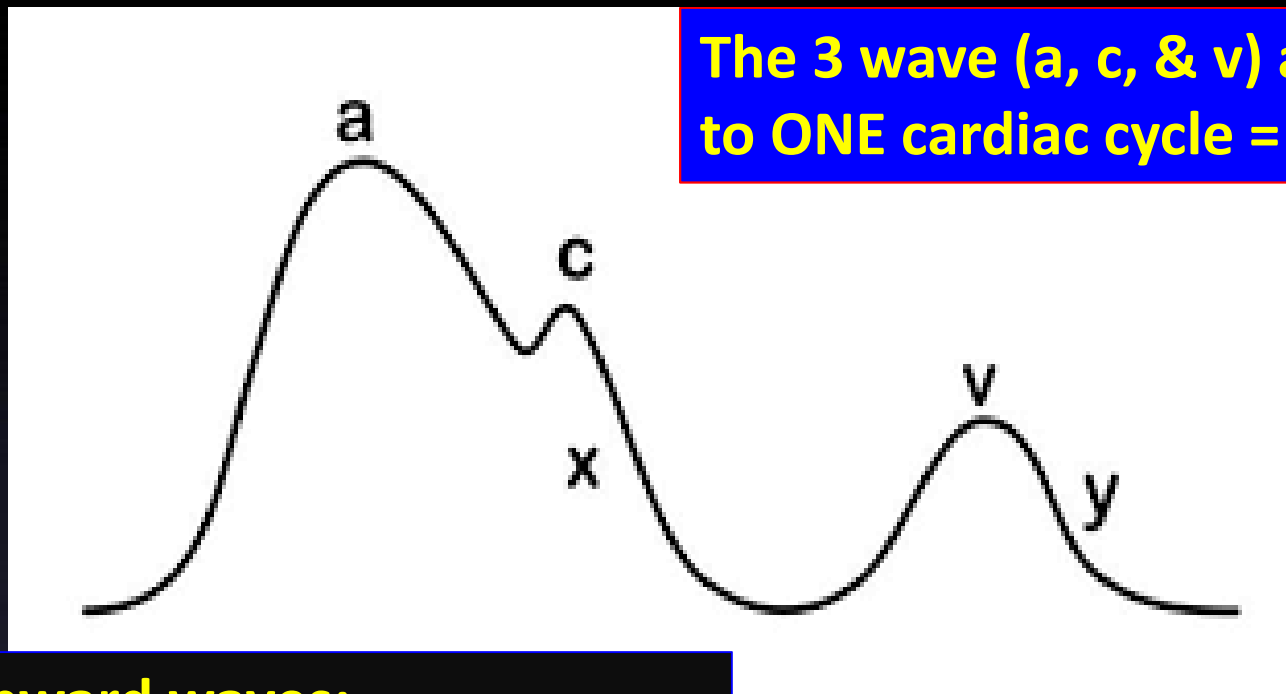
- Describe Normal pattern of the jugular venous pulse
- Define and classify heart failure.
- Describe the causes and pathophysiological consequences of acute and chronic heart failure.
- Explain how left-sided failure leads to right-sided failure & congestive heart failure.
- Discuss the compensatory 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.

Atrial pressure changes during the cardiac cycle

THE JUGULAR VENOUS PULSE (JVP)



Atrial pressure changes during the cardiac cycle



The 3 wave (a, c, & v) are equal to ONE cardiac cycle = 0.8 sec

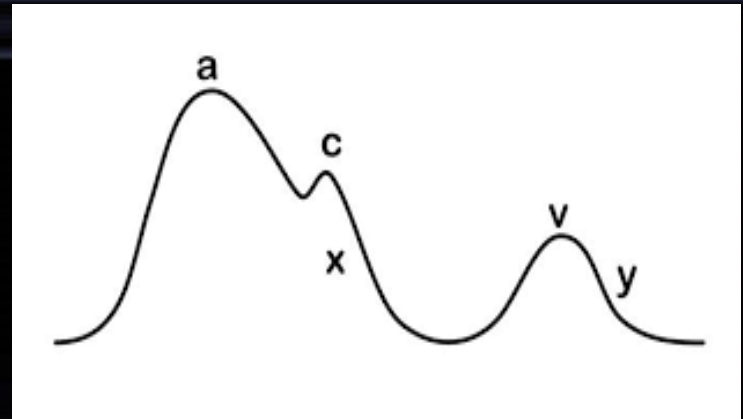
3 upward waves:

- a, c, & v waves
- 2 components in each wave: +ve (\uparrow pr), -ve (\downarrow pr)

2 downward deflection (waves):

- x & y waves

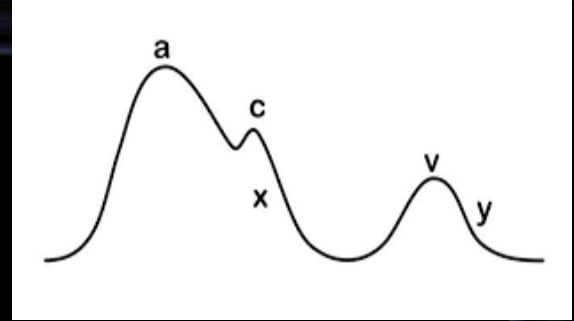
Atrial pressure waves



- ❑ 'a' wave: Atrial systole:
 - ↑ atrial pressure during atrial contraction

- ❑ 'c' wave: Ventricular systole
 - ❑ +ve as a result of bulging of AV valve into the atria during 'isovolumetric contraction phase'
 - ❑ -ve as a result of pulling of the atrial muscle & AV cusps down during 'rapid ejection phase', resulting in ↓ atrial pressure

Atrial pressure waves



- ❑ 'v' wave: Atrial diastole
 - ❑ +ve: atrial pressure \uparrow gradually due to continuous VR
 - ❑ -ve as a result of \downarrow atrial pressure during 'rapid filling phase'
- ❑ 'x' descent:
 - ❑ Downward displacement of AV valves during 'reduced ejection phase'
- ❑ 'y' descent:
 - ❑ \downarrow atrial pressure during 'reduced filling phase'

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

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 O₂.

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

Systolic failure

- ❑ The heart loses its ability to contract or pump blood into the circulation

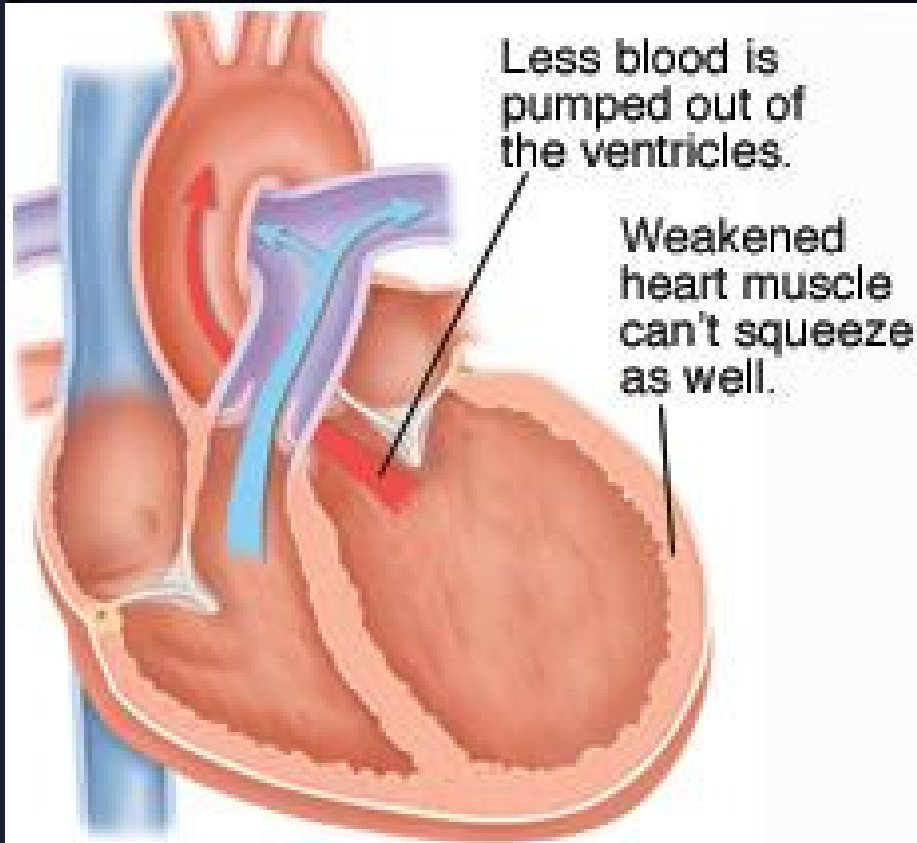
Diastolic failure

- ❑ The heart loses its ability to relax because it becomes stiff
- ❑ Heart cannot fill properly between each beat

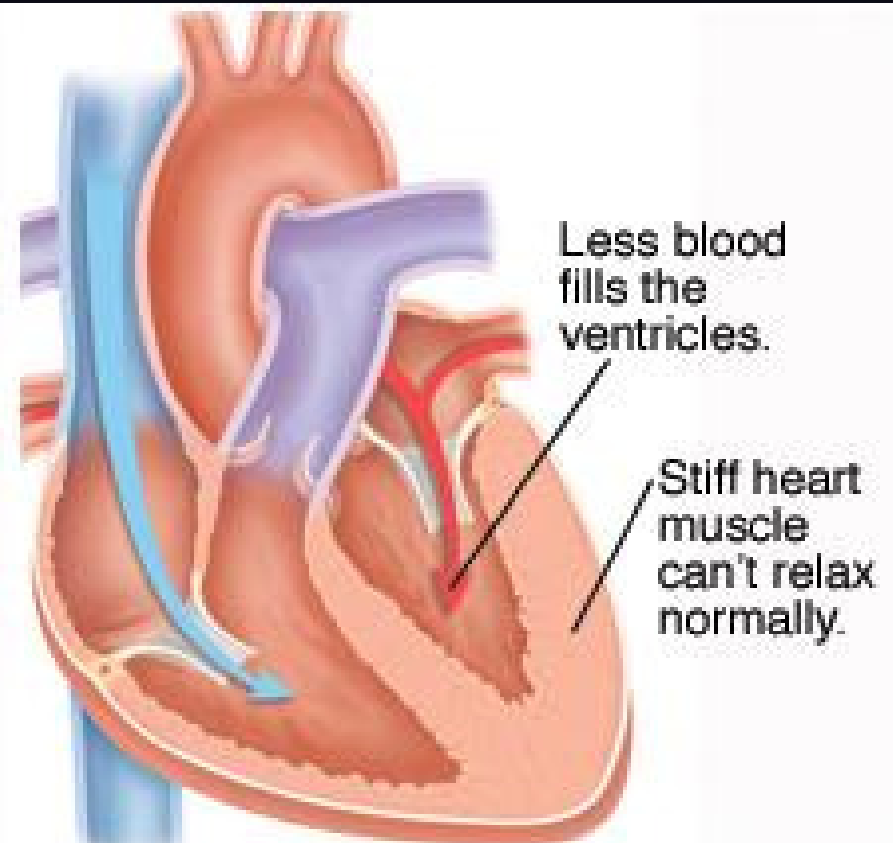
Characteristic	Diastolic Heart Failure	Systolic Heart Failure
Age	Frequently elderly	All ages, typically 50–70 yr
Sex	Frequently female	More often male
Left ventricular ejection fraction	Preserved or normal, approximately 40% or higher	Depressed, approximately 40% or lower
Left ventricular cavity size	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.

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

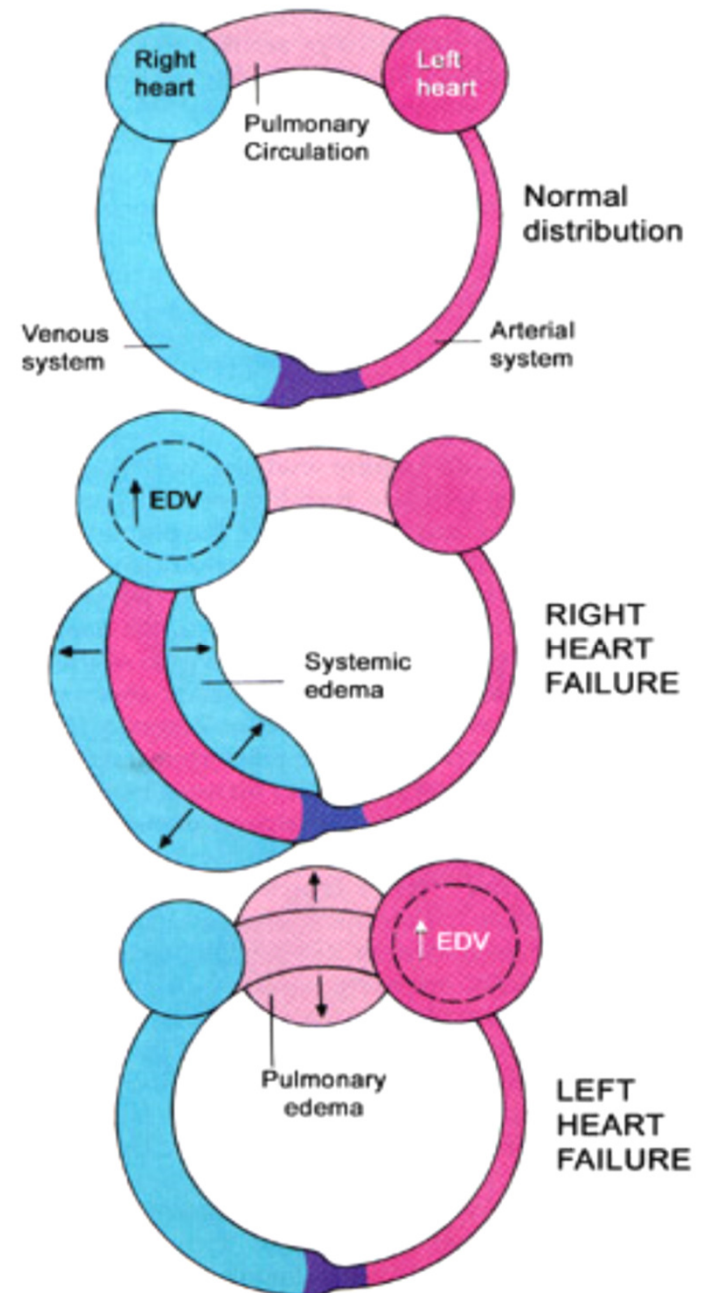
- **Hypertension**
- **Valvular disorders**
- **Anemias**
- **Congenital heart defects**

3- Acute non-cardiac conditions

- **Volume overload**
- **Hyperthyroidism, Fever, Infection**

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 embolism
- 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

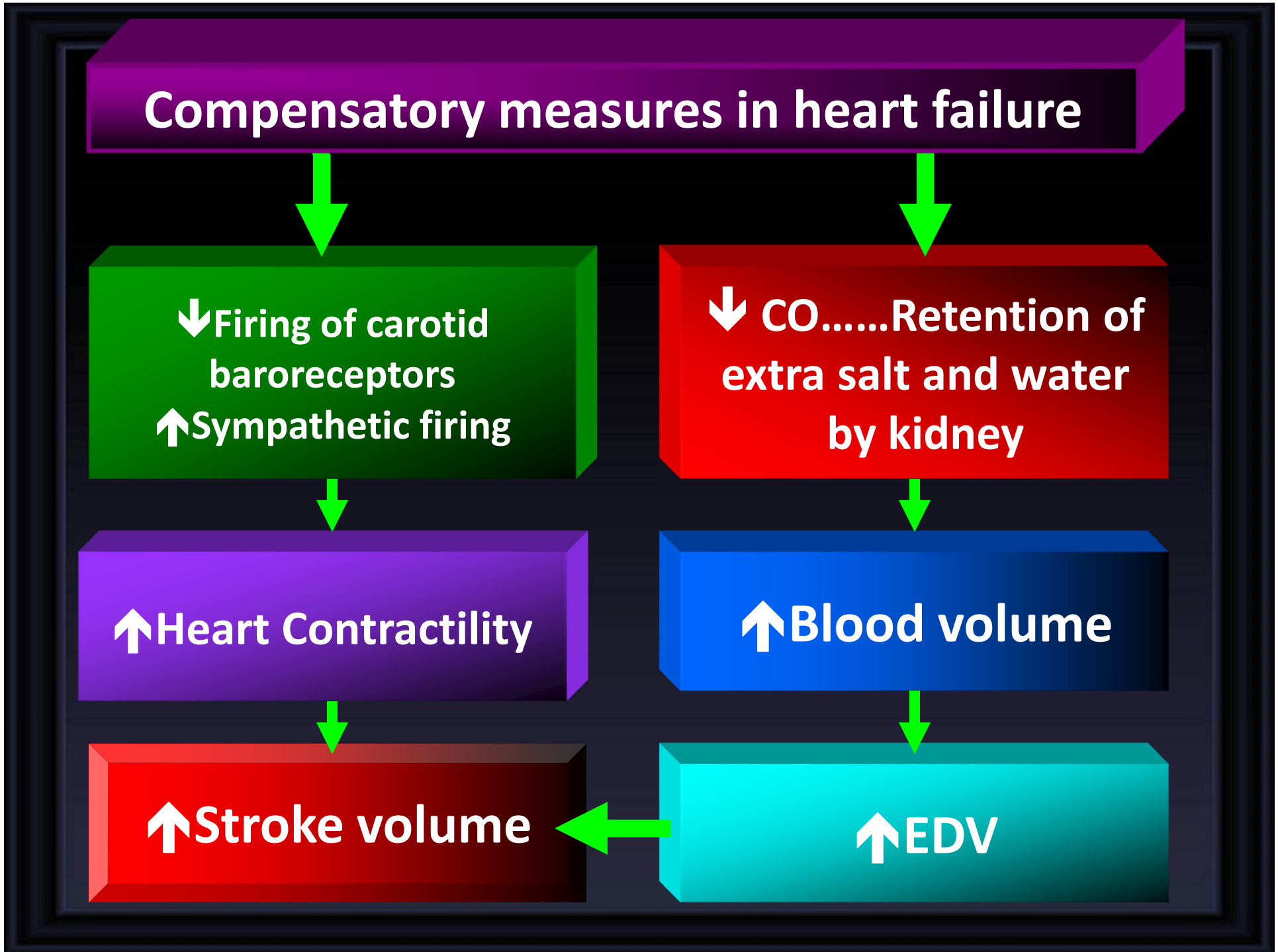
↑ Heart Contractility

↑ Stroke volume

↓ CO.....Retention of extra salt and water by kidney

↑ Blood 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 H₂O 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 responses 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 O₂ 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).

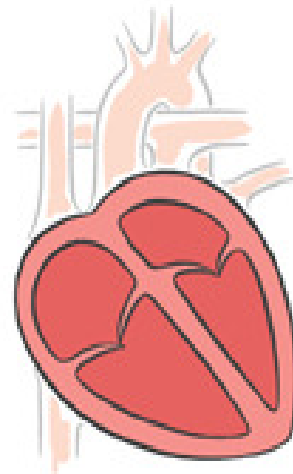
INDICATOR FOR DIAGNOSING HEART FAILURE

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.



Normal Heart
50–70% EF



Heart Failure Heart
Less than 40% EF

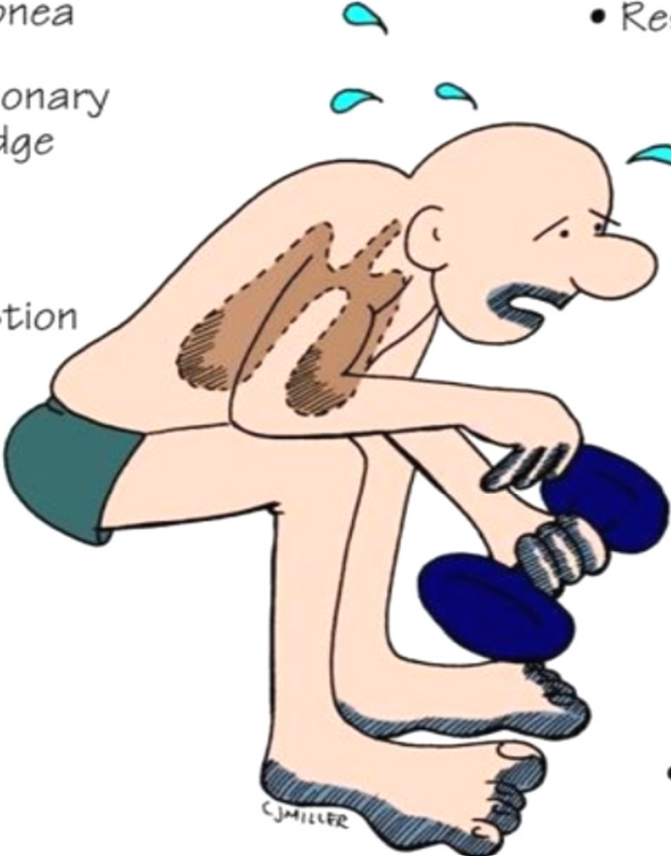
Chambers enlarge to handle increased fluid

Walls get thicker to handle the increased strain

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

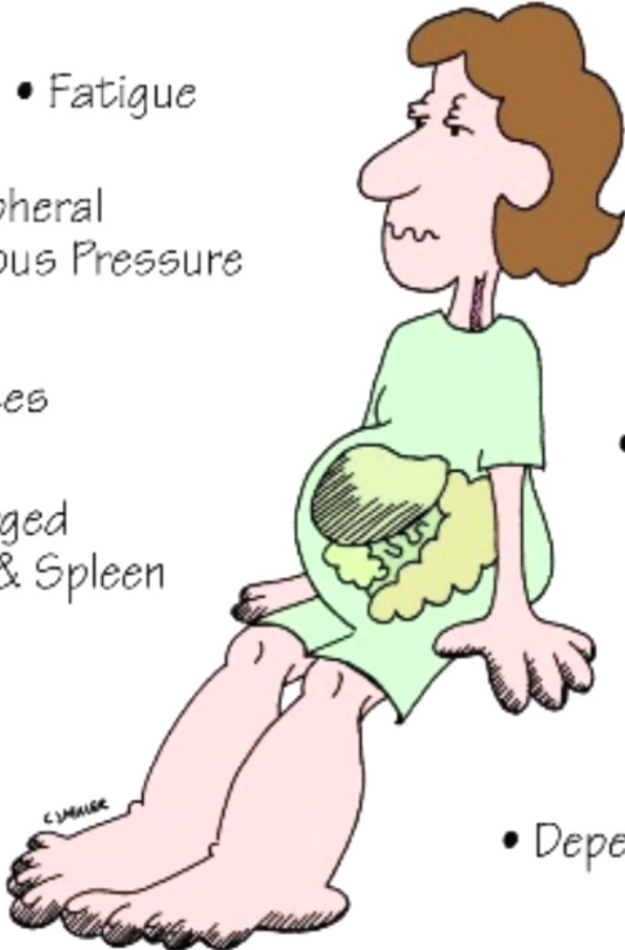
- Paroxysmal Nocturnal Dyspnea
 - Elevated Pulmonary Capillary Wedge Pressure
 - Pulmonary Congestion
 - Cough
 - Crackles
 - Wheezes
 - Blood-Tinged Sputum
 - Tachypnea
 - Restlessness
 - Confusion
 - Orthopnea
 - Tachycardia
 - Exertional Dyspnea
 - Fatigue
 - Cyanosis
- 

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)

Clinical picture	Left-sided failure	Right-sided failure
Pitting edema (legs, hands)	Mild to moderate	Moderate to sever
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

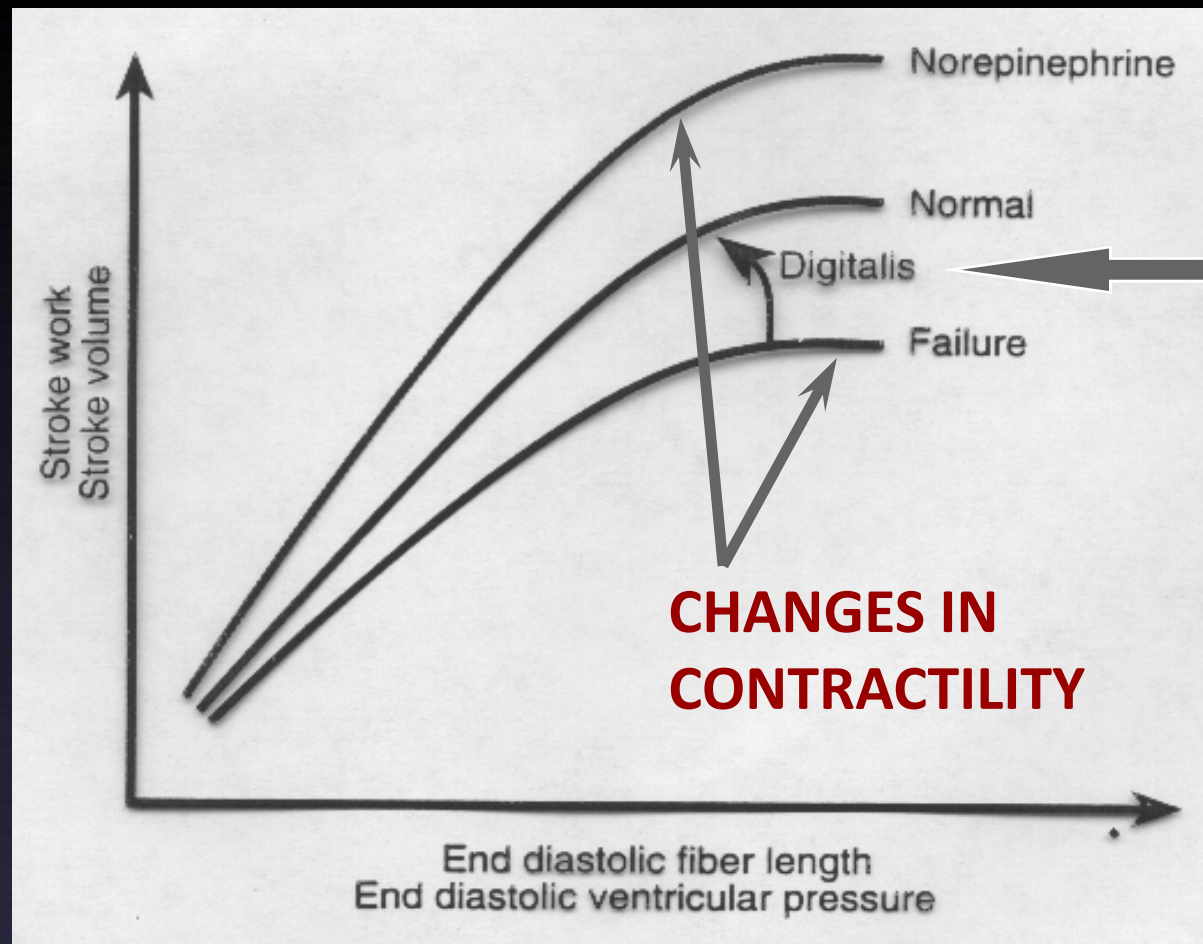
Diuretics

↑ in the pumping
activity of failing heart

Digitalis

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

Starling curve in heart failure



SYSTOLIC DYSFUNCTION

Impaired ventricular contraction

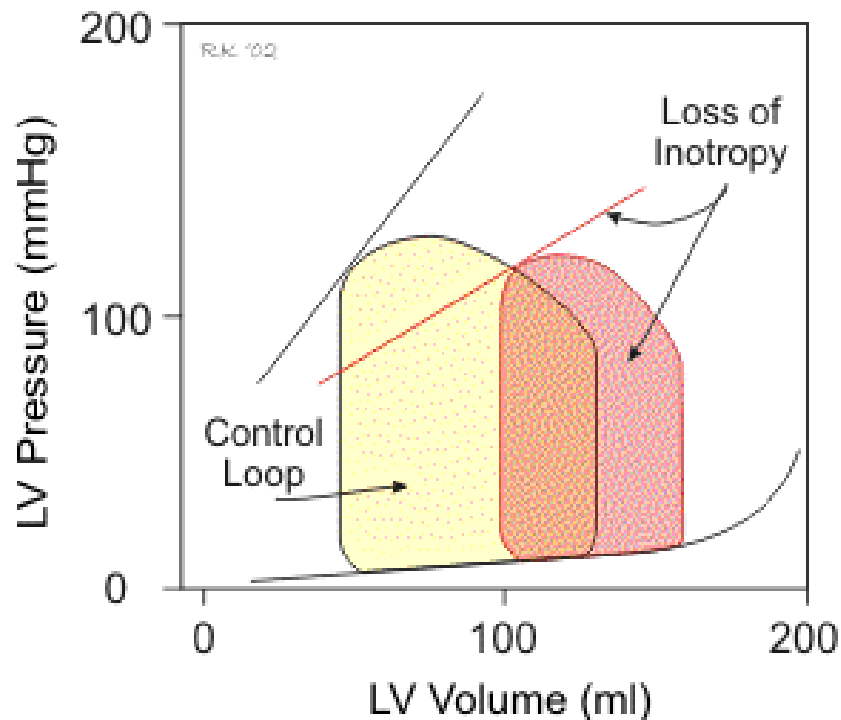


Figure 2. Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

↓ slope of ESPVR i.e. ↑ ESV

Compensatory rise in preload i.e.

↑ EDV

↓ SV

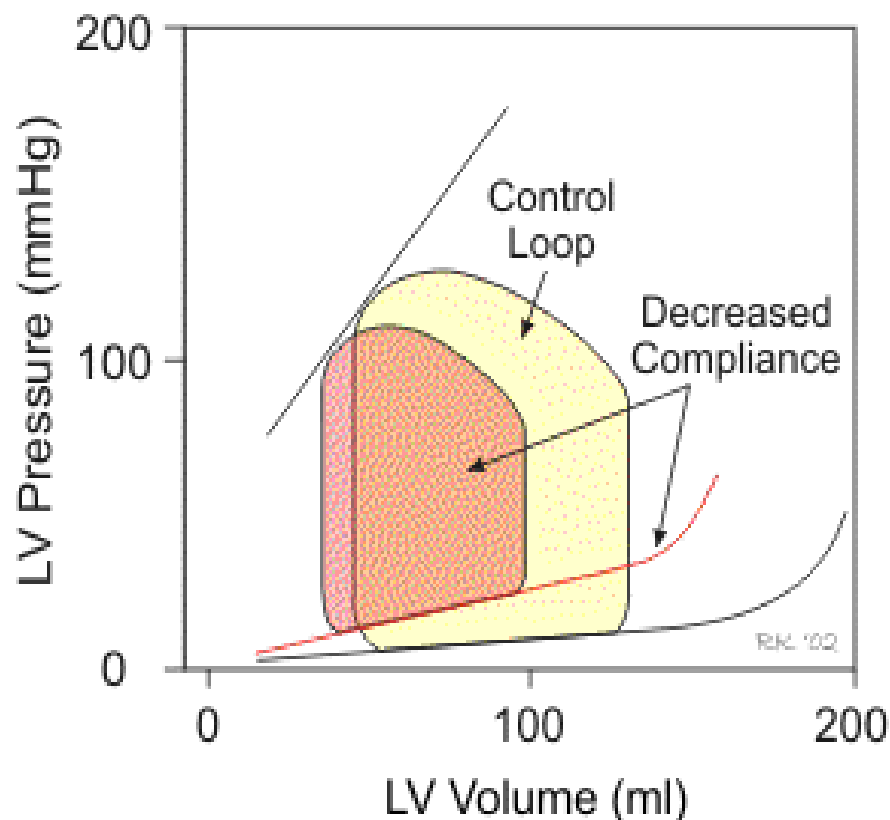
↓ EF

↓ Work

↑ 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 = EF

↓ Work

↑ EDP