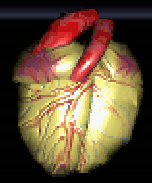
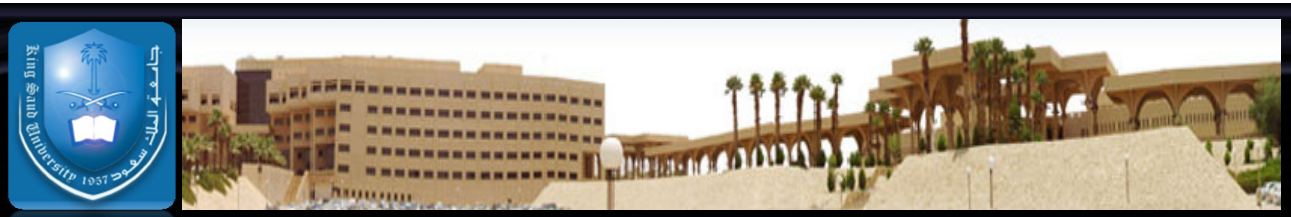
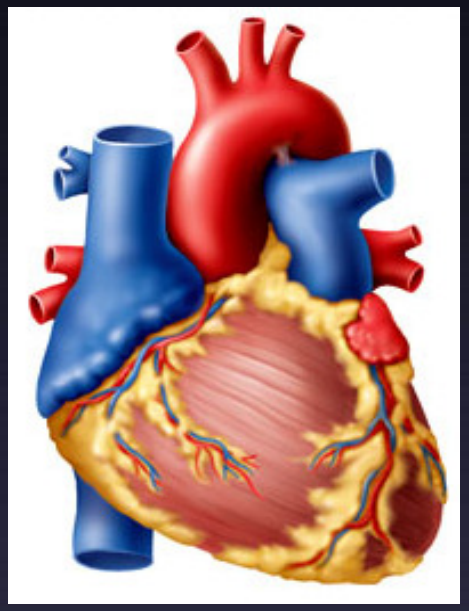
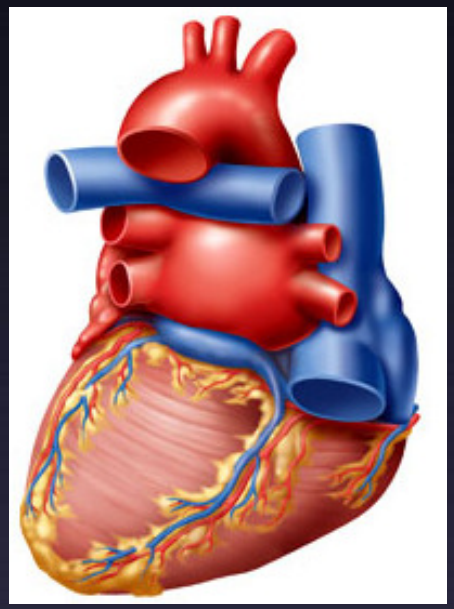




CARDIOVASCULAR SYSTEM

Regulation of stroke volume (preload, contractility &afterload) & heart failure





Prof. Shahid Habib
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OBJECTIVES

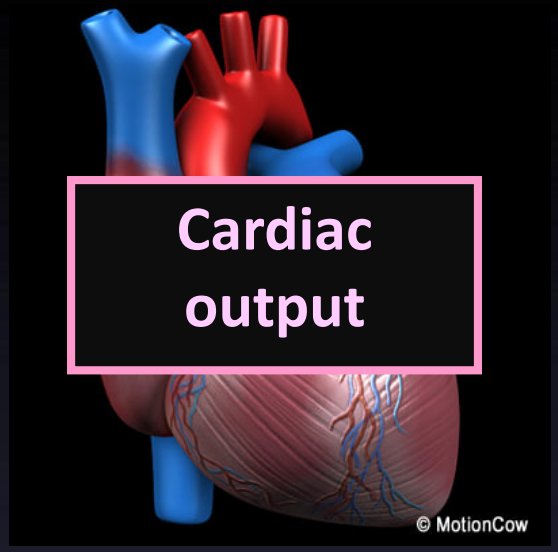
❖ At the end of the lecture you should be able to …..

- ❑ Explain how cardiac contractility affect stroke volume.
- ❑ 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.
- ❑ Compare and contrast compensated and decompensated 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.

Determinants of the CO

Afterload

Preload



Myocardial Contractility (Inotropic state)

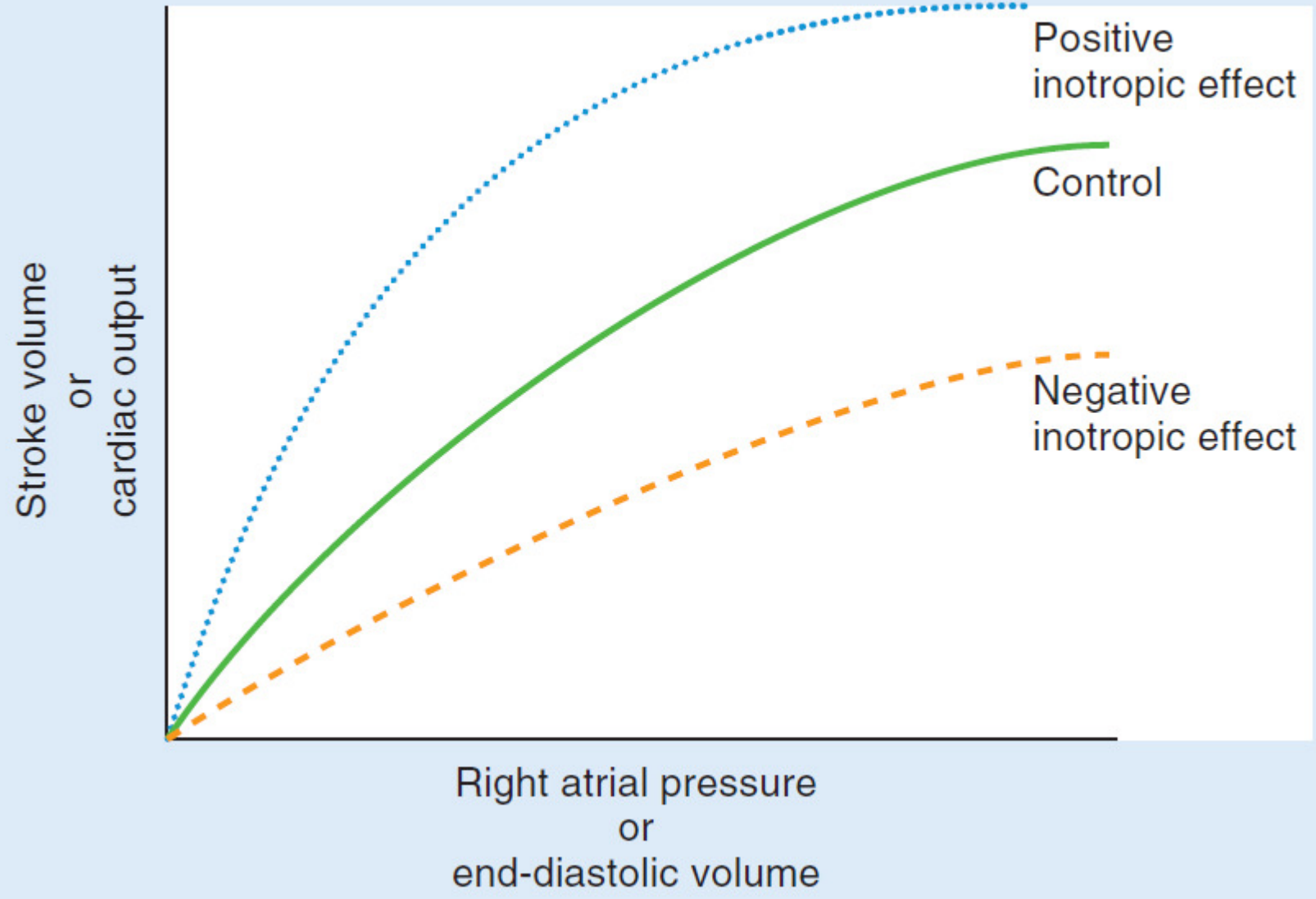
Heart rate (Chronotropic state)

EJECTION FRACTION (EF) is the percentage of ventricular end diastolic volume (EDV) which is ejected with each stroke.

$$EF = \frac{SV \text{ or } (EDV - ESV)}{EDV} \times 100$$

$$\frac{75}{120} \times 100 = 62.5\%$$

Normal ejection fraction is about 60 – 65 %.
Ejection fraction is good index of ventricular function.


Positive
inotropic effect
Control
Negative
inotropic effect
Stroke volume
or
cardiac output
Right atrial pressure
or
end-diastolic volume

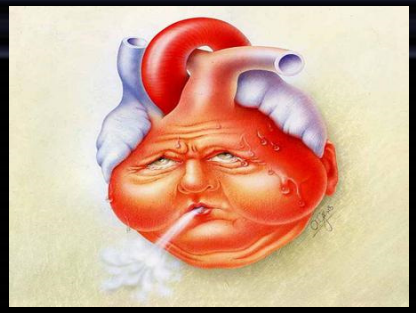
Preload & Afterload

Preload: It is the amount of blood that returns to the heart from veins.

Is end-diastolic volume, which is related to right atrial pressure. When venous return increases, end-diastolic volume increases and stretches or lengthens the ventricular muscle fibers

Afterload: It is the resistance against which the ventricles contract.

For the left ventricle is aortic pressure. Increases in aortic pressure cause an increase in afterload on the left ventricle and for the right ventricle is pulmonary artery pressure. Increases in pulmonary artery pressure cause an increase in afterload on the right ventricle.



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

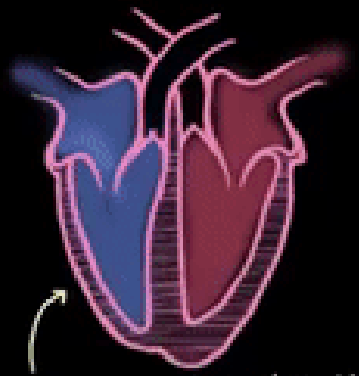
Systolic failure

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

Diastolic failure

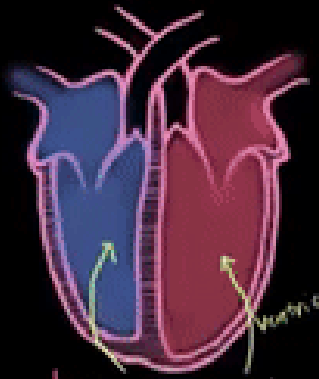
- The heart looses it's ability to relax because it becomes stiff
- Heart cannot fill properly between each beat

Healthy Heart



Systole : contracts & ejects blood

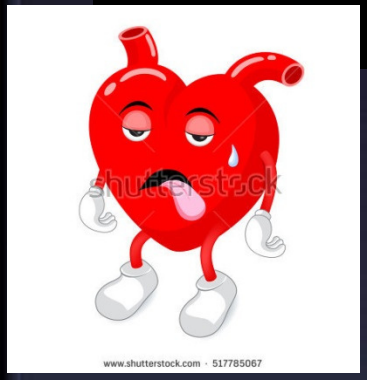
Systolic Heart Failure

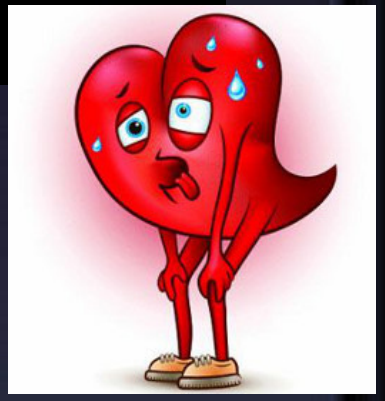


↓ Force of contraction

↓ Ventricles

↓ Ejection





Heart Failure

Causes

- Intrinsic myocardial causes (These result in reduction in ventricular contractility):
  - myocardial infarction (death of cardiac myocytes due to blockage of the coronary arteries)
  - Cardiomyopathy
  - Myocarditis
- Cardiac arrhythmias: e.g., complete heart block
- Extrinsic causes (These make it more difficult to eject blood into aorta):
  - systemic hypertension
  - aortic stenosis

Heart Failure

Causes

1- Impaired cardiac function
- Coronary heart disease
- Cardiomyopathies (muscle disease)
- Rheumatic fever
- Endocarditis

2- Increased cardiac workload
- Hypertension
- Valvular disorders
- Anemias
- Congenital heart defects

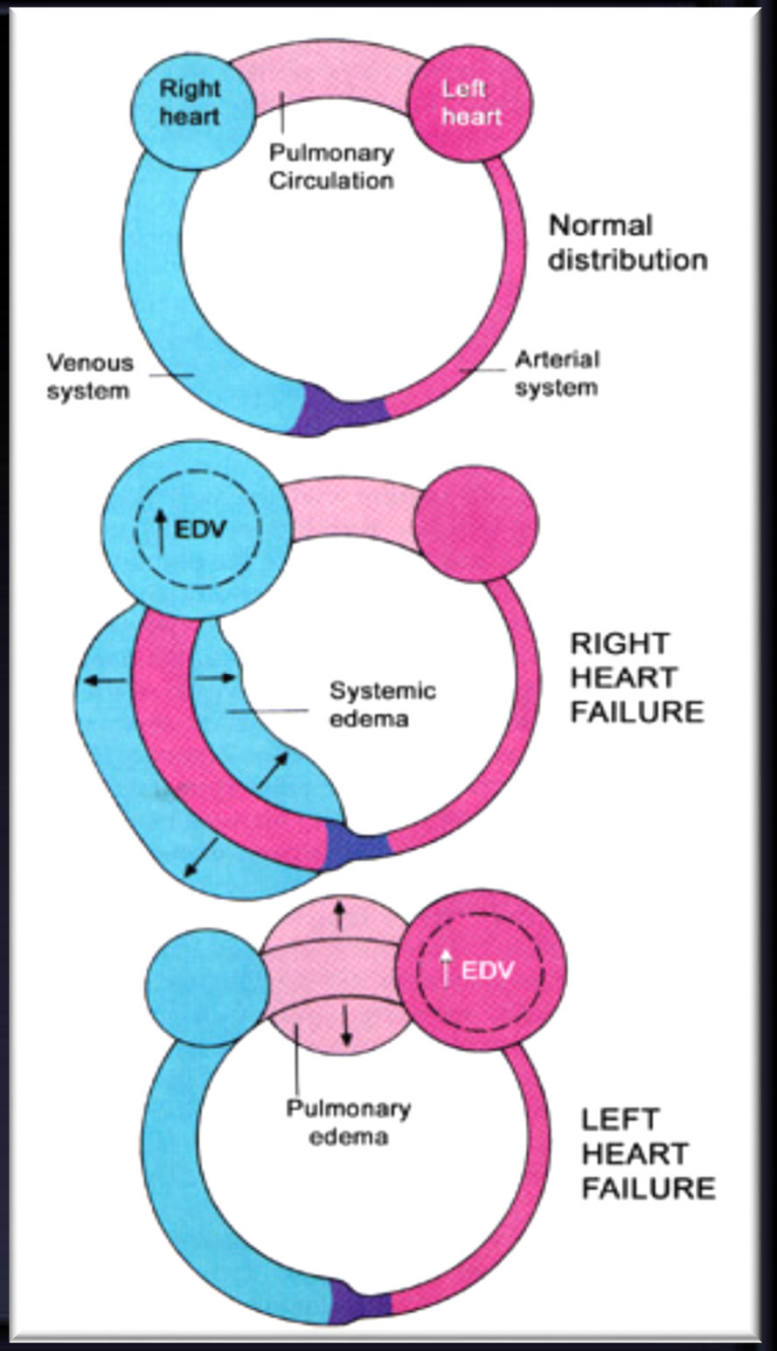
3- Acute non-cardiac conditions
- Volume overload
- Hyperthyroidism, Fever, Infection

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.

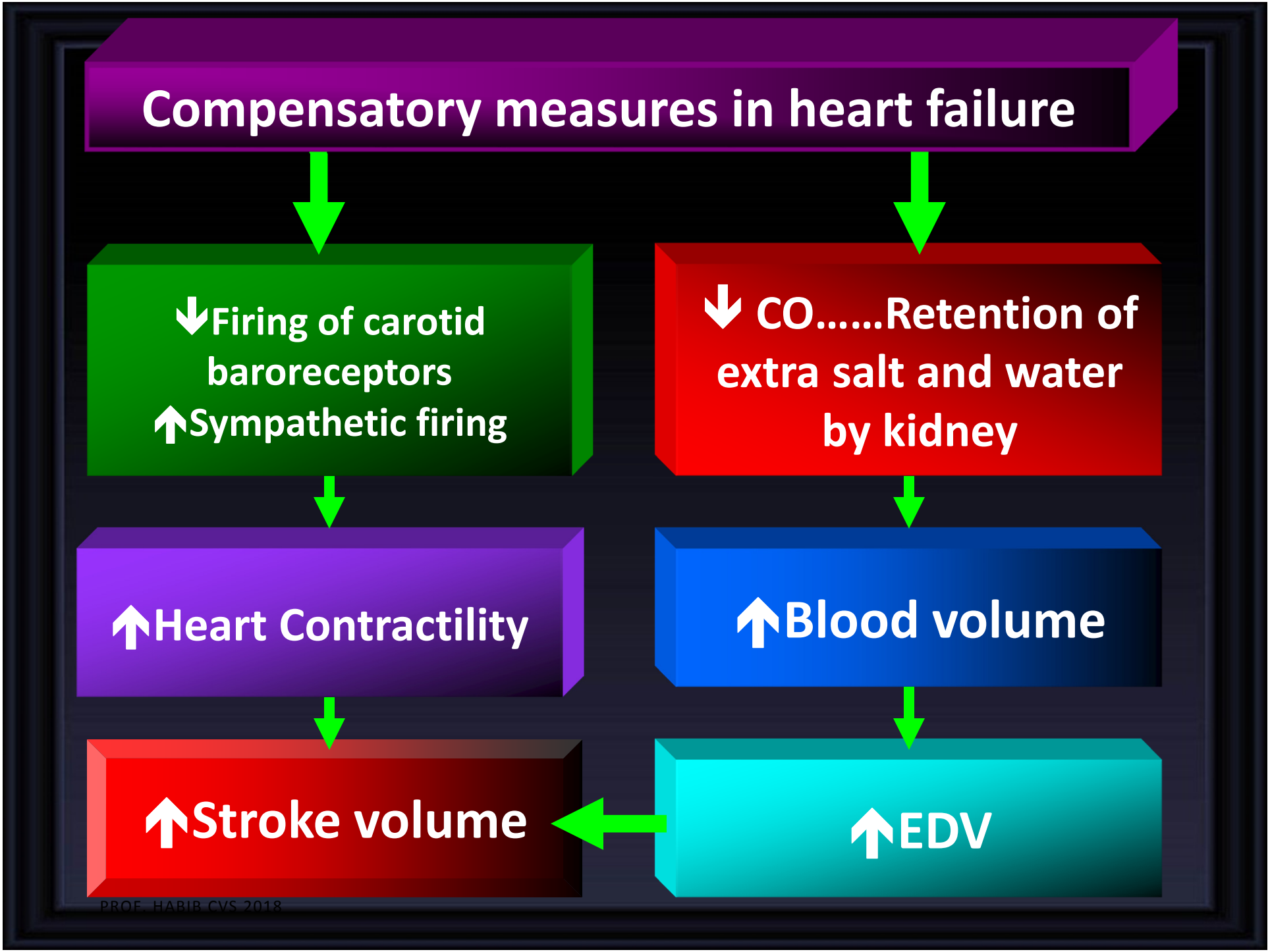
- Though each side of the heart can undergo failure separately, dysfunction of one side may lead to a sequence of events that make the opposite side also to fail.



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
PROF. HABIB CVS 2018

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).

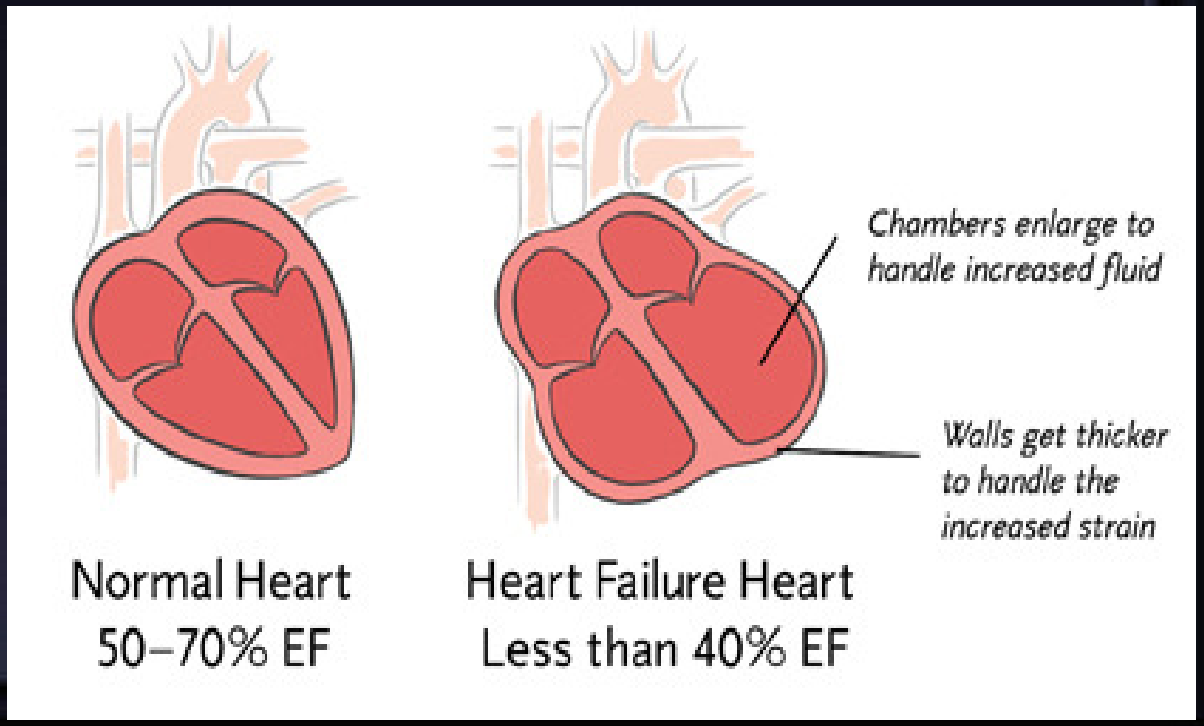
INDICATOR FOR DIAGNOSING HEART FAILURE

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.

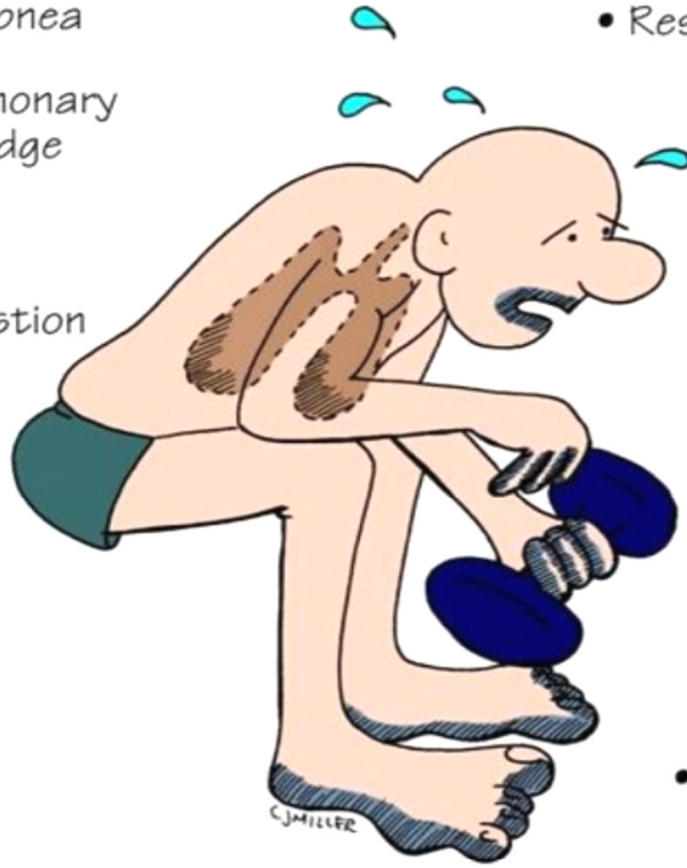


HOW HEART FAILURE IS DIAGNOSED

- 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

- 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.

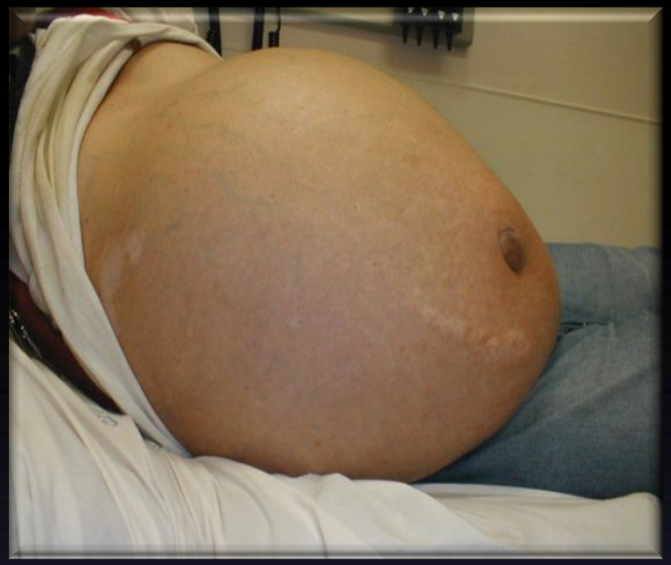
Paroxysmal nocturnal dyspnea refers to attacks of severe shortness of breath and coughing that generally occur at night. It usually awakens the person from sleep, and may be quite frightening.

Clinical picture of left-sided failure

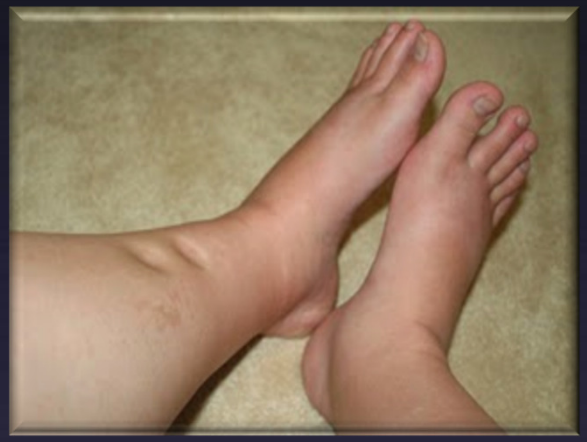
(Cor Pulmonale)

- 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

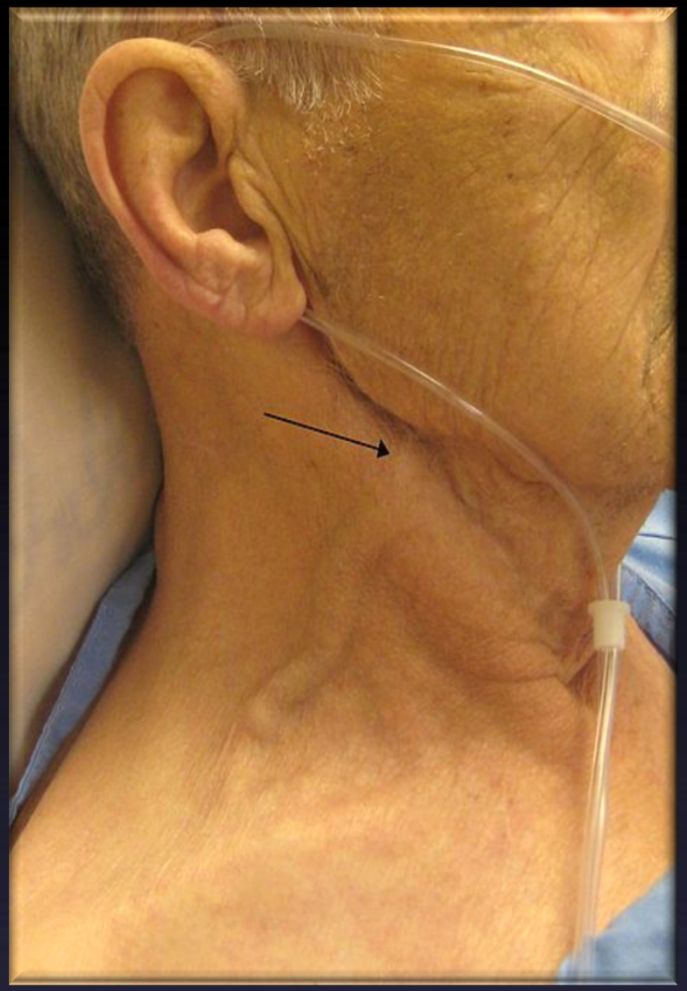
Clinical picture of right-sided failure



(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

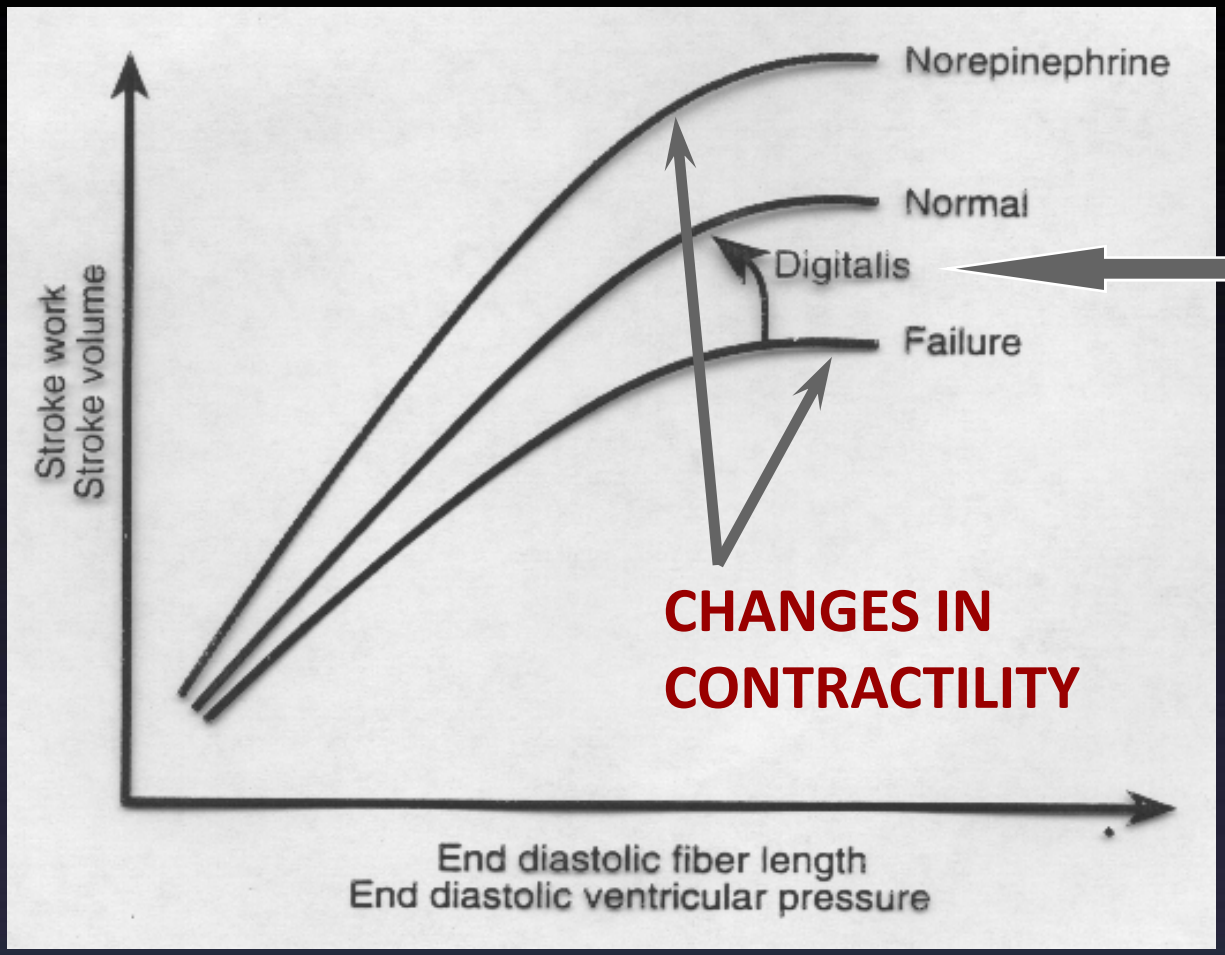
Treatment of cardiac failure

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**

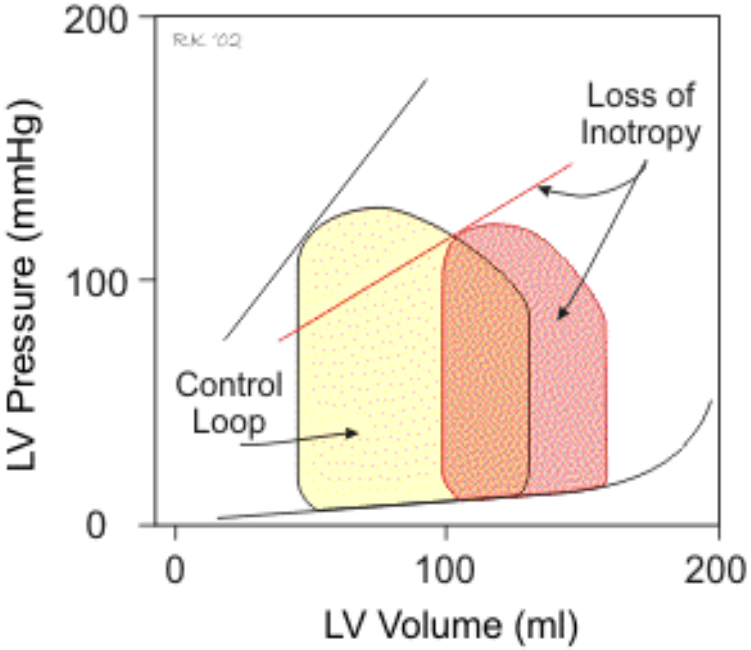


Figure 2. Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

↓ slope of ESPVR i.e. ↑ ESV

Compensatory rise in preload i.e. ↑ EDV

↓ SV

↓ EF

↓ Work

↑ EDP

FORCE- VELOCITY RELATIONSHIP

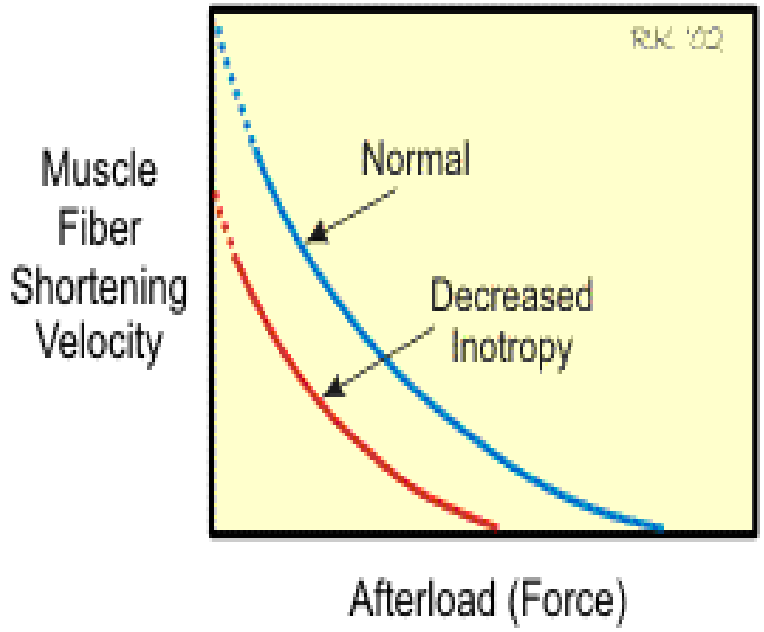
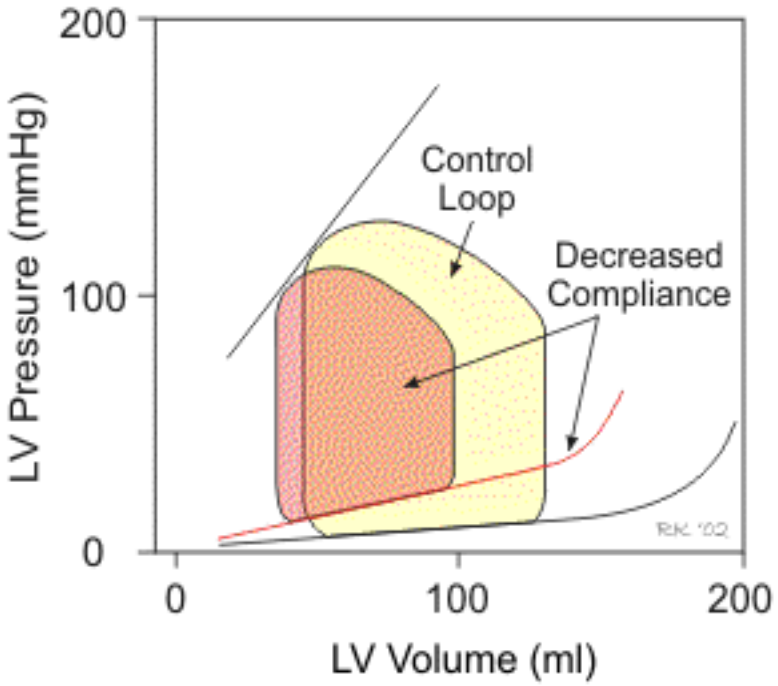


Figure 3. Effects of ventricular failure (decreased inotropy) on the force-velocity relationship. Decreased inotropy decreases velocity of fiber shortening at any given afterload.

At any given preload and afterload, a loss of inotropy results in decrease in shortening velocity of the cardiac fibres.

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

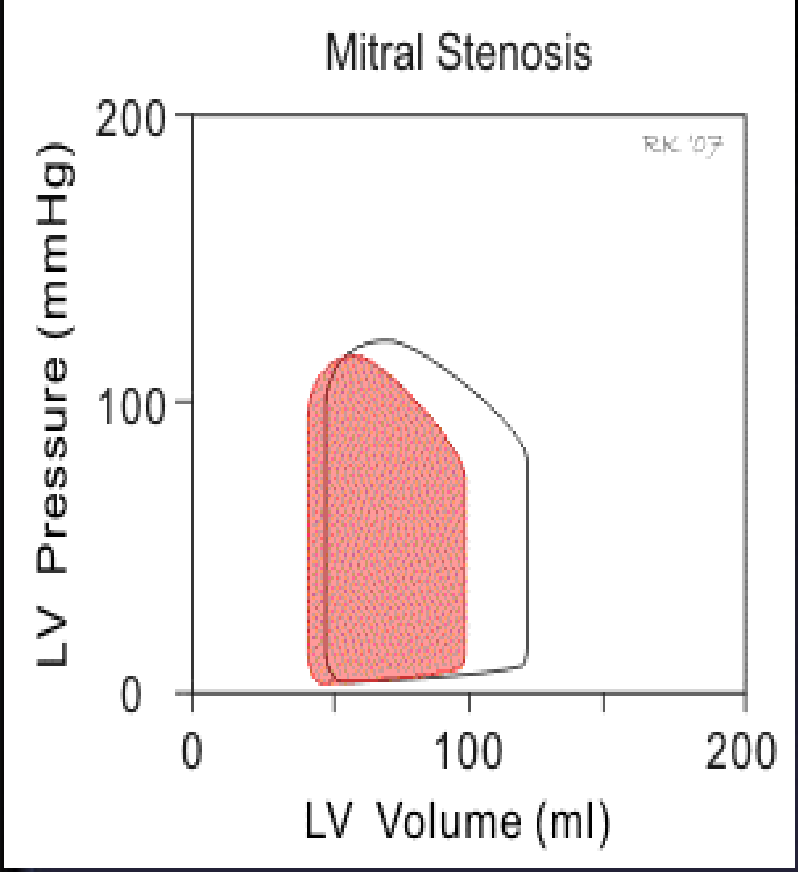
↓ SV

↓ or = EF

↓ Work

↑ EDP

MITRAL STENOSIS



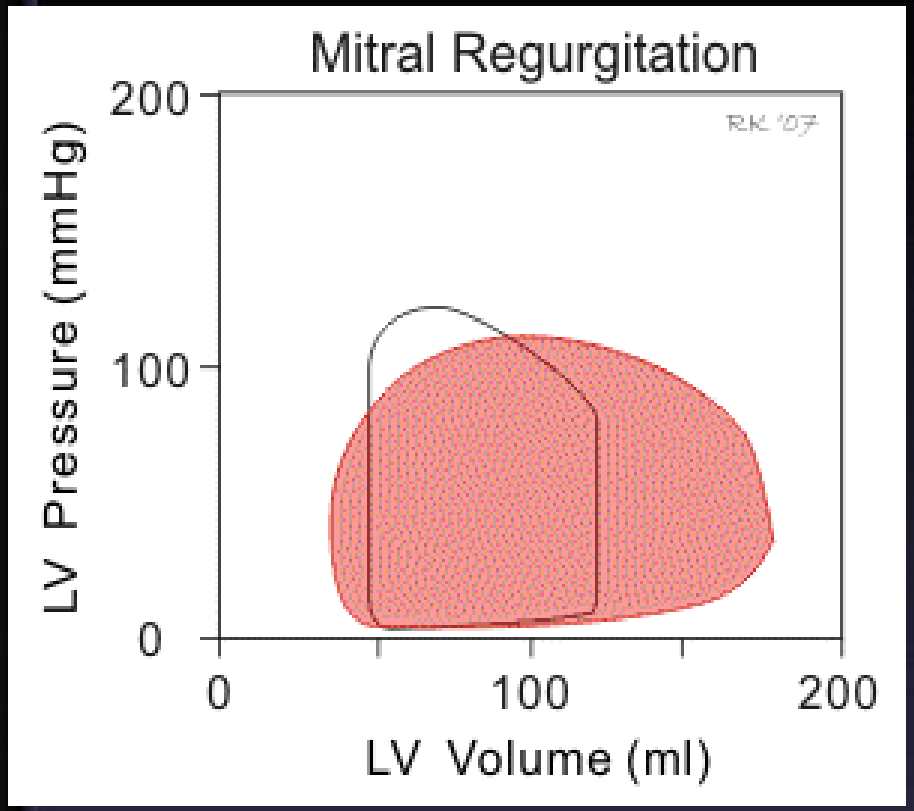
Impaired LV filling

↓ EDV

↓ afterload ; ↓ ESV

↓ SV and CO

MITRAL REGURGITATION



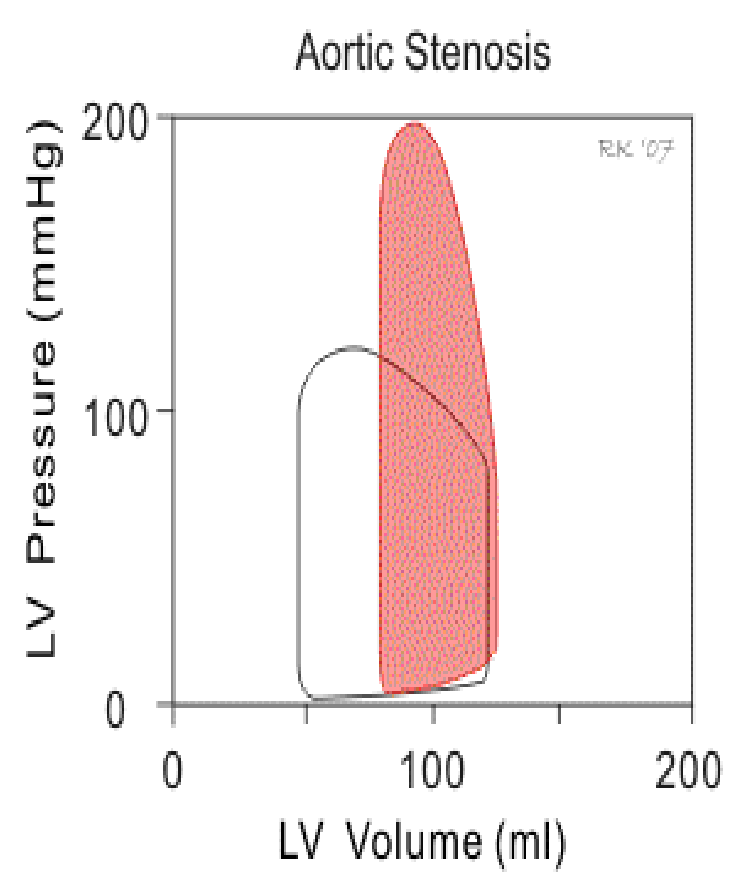
Afterload on LV ↓ Outflow resistance is ↓

EDV and EDP ↑

↑ SV

↓ EF

AORTIC STENOSIS



High outflow resistance; LV emptying impaired

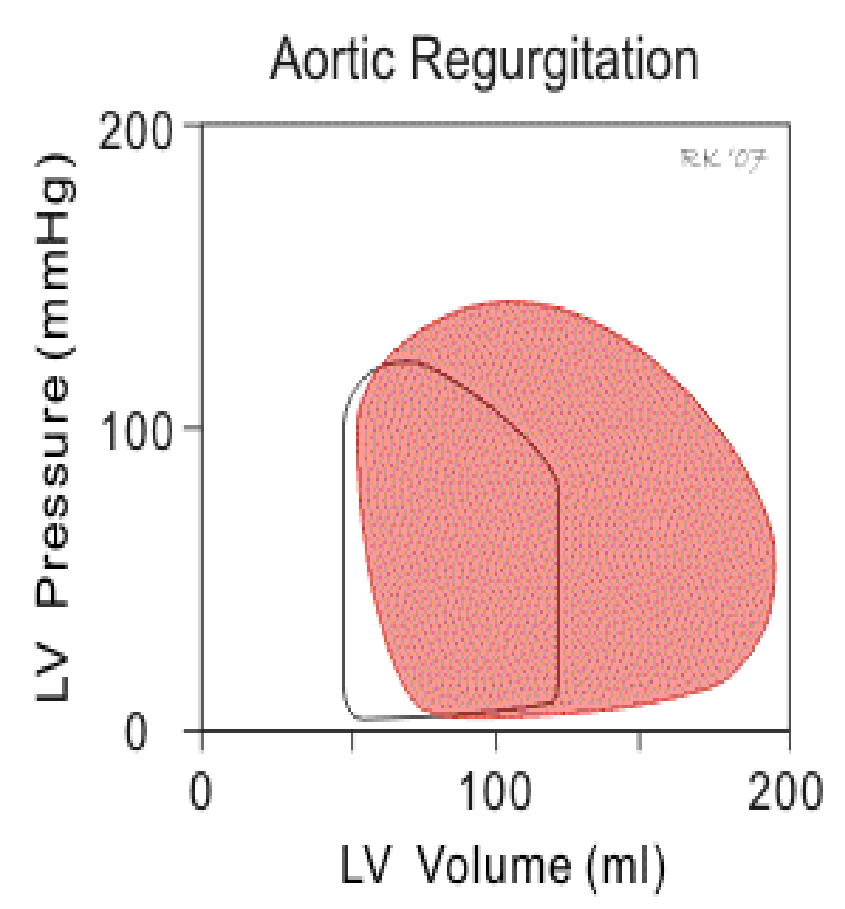
↑ Peak systolic pressure; ↑ afterload

↓ SV

↑ ESV

↑ EDV

AORTIC REGURGITATION



No true isovolumetric relaxation

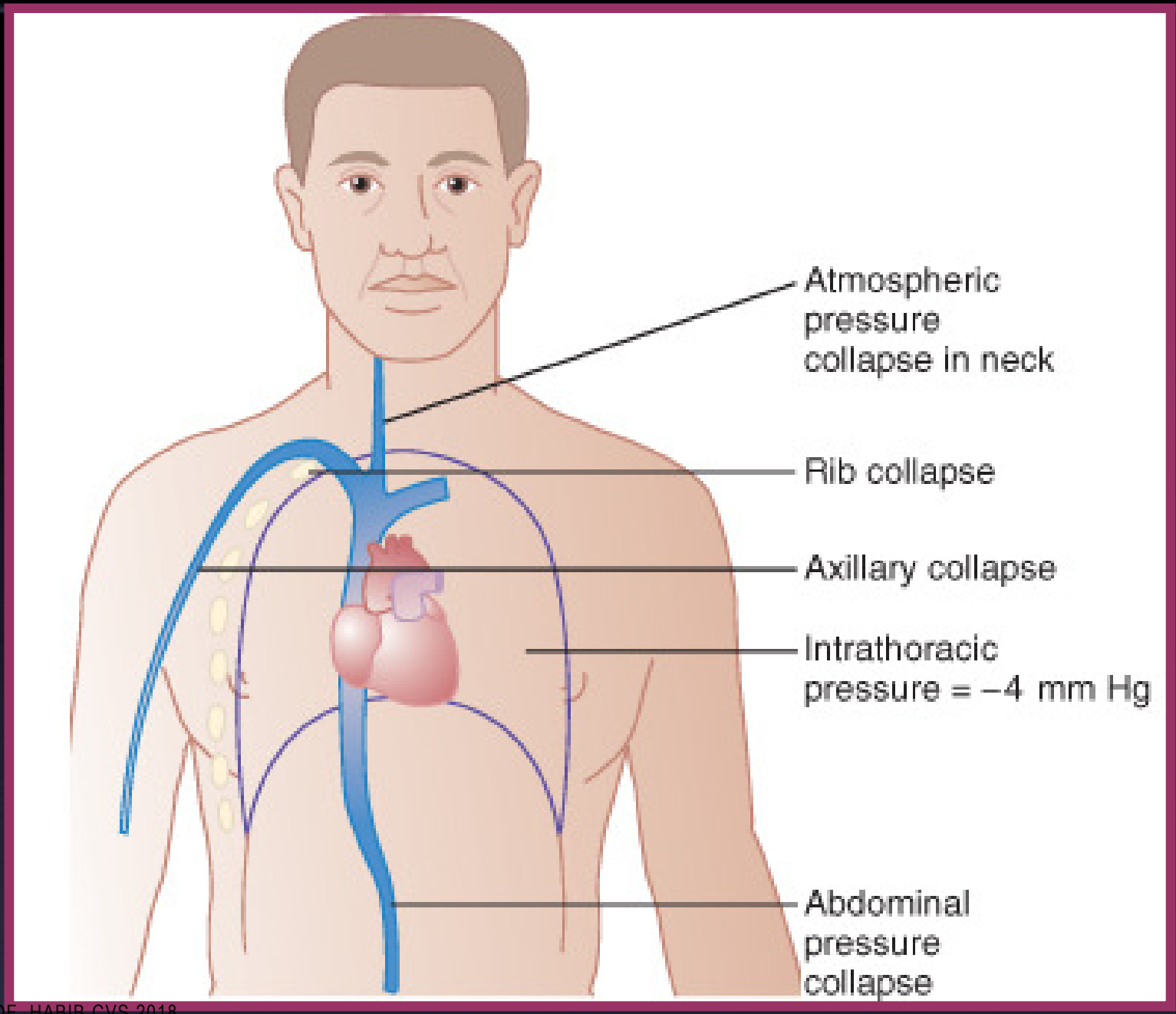
Blood from aorta to ventricle throughout diastole

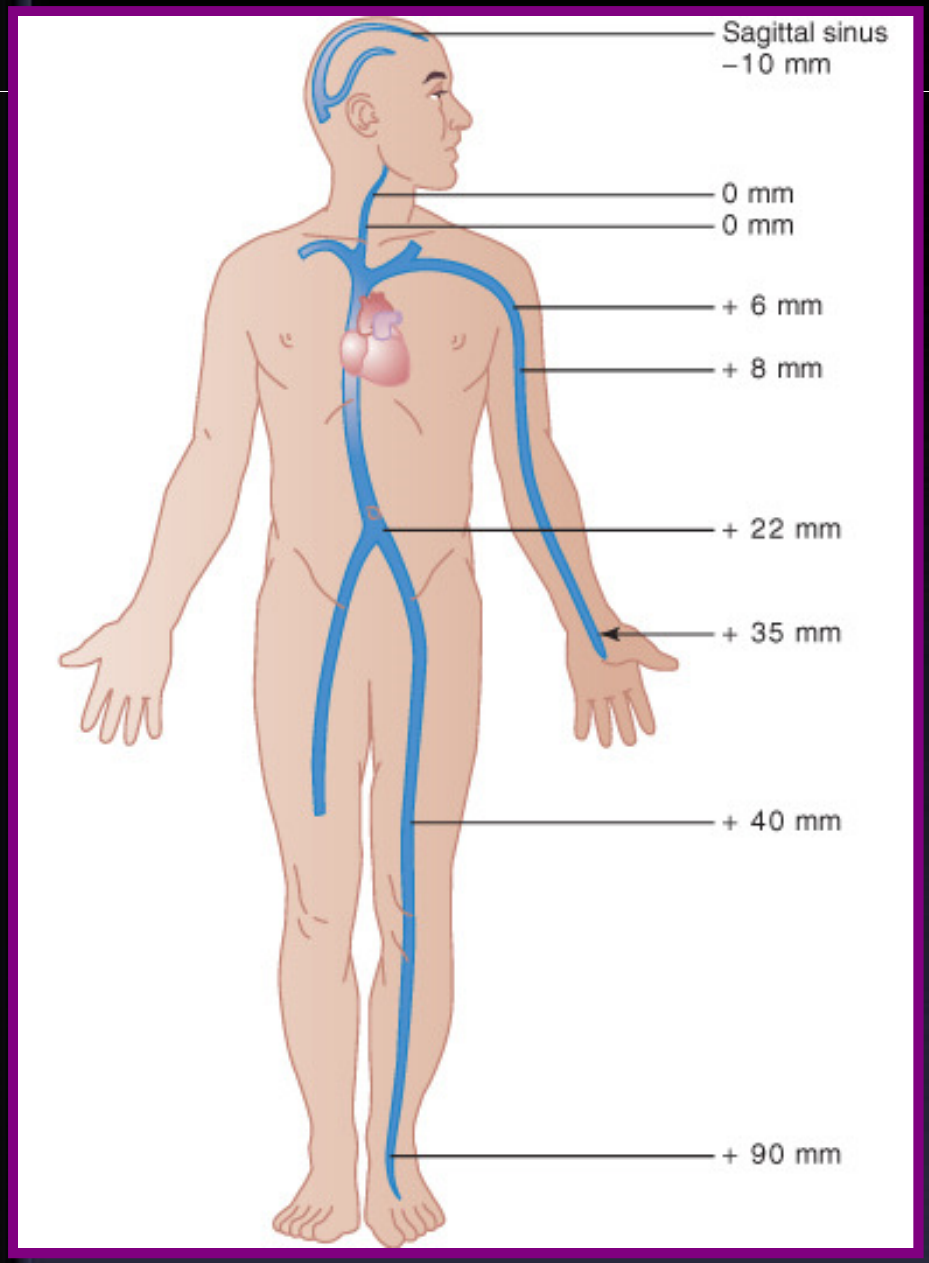
↑ EDV

↑ SV (if no failure)

↑ ESV and ↓ SV in failure

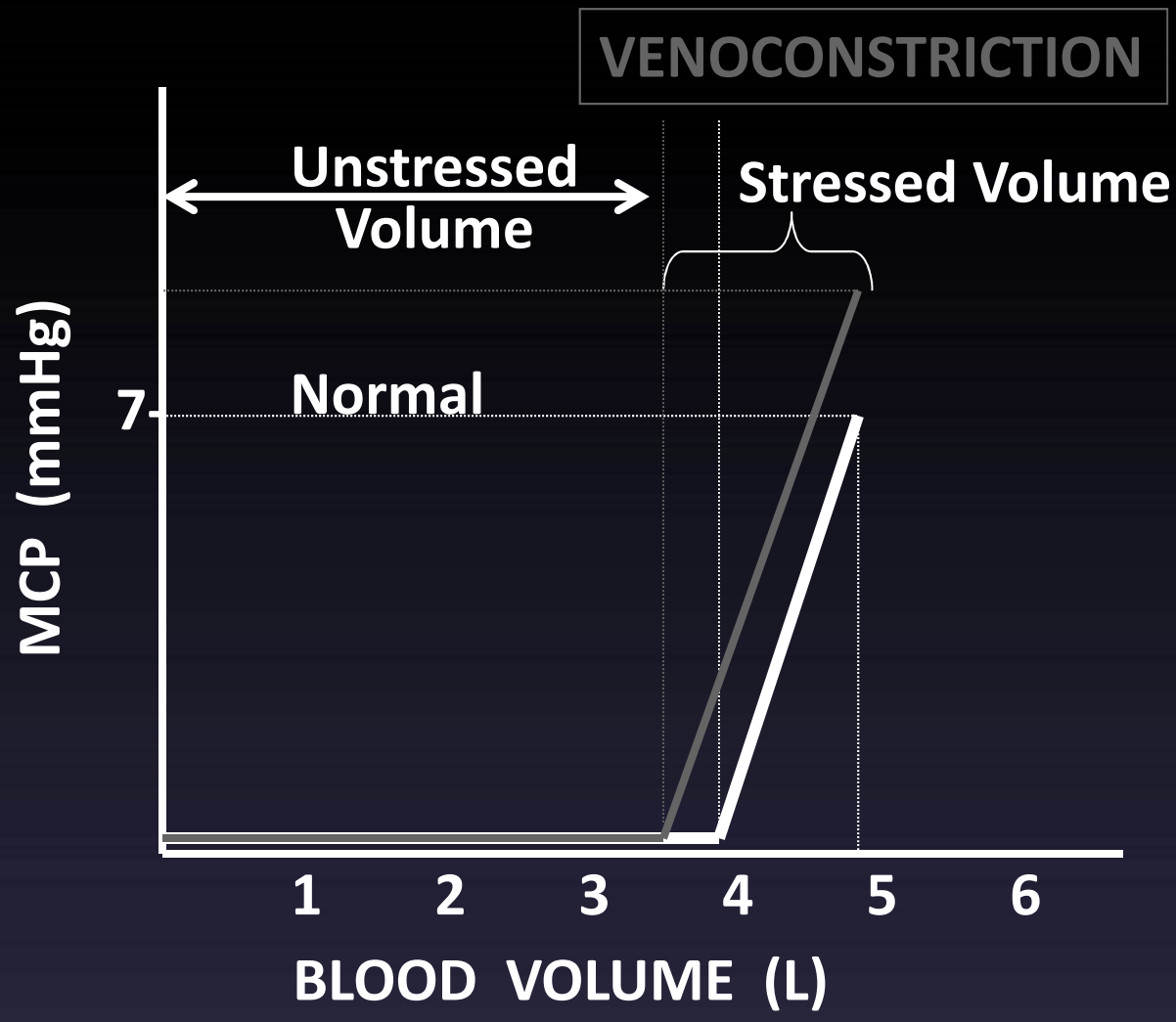

THANKS


Atmospheric pressure collapse in neck
Rib collapse
Axillary collapse
Intrathoracic pressure = −4 mm Hg
Abdominal pressure collapse



Effect of Hydrostatic Pressure on Venous Pressures

Mean circulatory pressure; MCP



Mean circulatory pressure; MCP