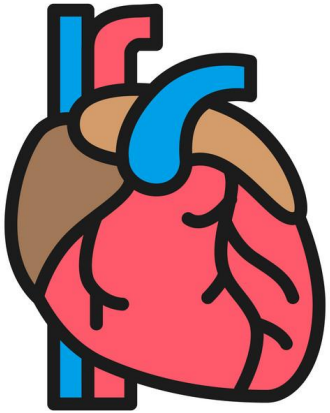
Revised & Reviewed by:
Abdulaziz & Bahammam
Faye Wael Sendi

MED441
KING SAUD UNIVERSITY



Jugular Venous Pulse & Heart Failure

Color Index:

- Main text
- Important
- Girls Slides
- Boys Slides
- Notes
- Extra

Editing File

Helpful Videos

JVP: 

Video

HF: 

Video

HF in Animation: 

Video

Objectives

- Identify the jugular venous pressure
- Know the method of examination of the internal venous pressure
- Normal pattern of the jugular venous pulse
- What are the abnormalities of jugular venous pulse
- Define heart failure
- Identify types of heart failure
- Describe the causes and pathophysiological consequences of acute and chronic heart failure.
- Indicators for diagnosis of 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 heart failure, and heart failure treated with digoxin.

Girls doctor played it

Video

Female lecture start from here

Males lecture start from heart failure slide 14

Jugular Venous Pulse and pressure definitions.

Jugular Venous Pulse:

JVP is the oscillating top of vertical column of blood in right internal jugular vein.

It reflects pressure changes in right atrium during the cardiac cycle.

Jugular Venous Pressure:

Vertical **height** of oscillating column of blood in right internal jugular vein.

Why Right Internal Jugular Vein (IJV)?

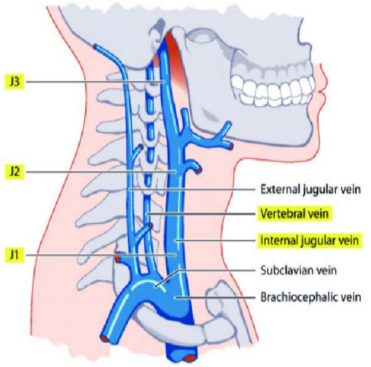
Female slides

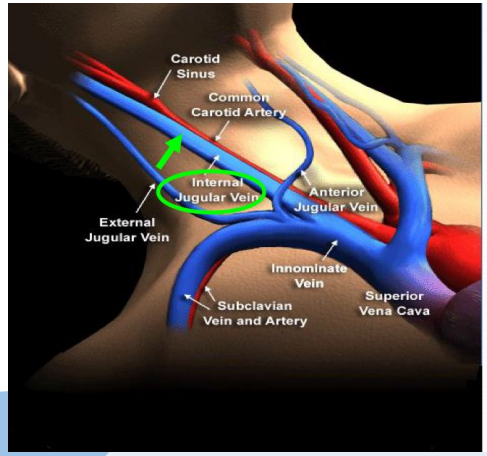
1. Right internal jugular veins (IJV) extend in an almost **straight** line to superior vena cava and has a direct course to RA, thus favoring transmission of the hemodynamic changes from the *right atrium.*

2. IJV is anatomically **closer** to RA.

3. IJV has **no valves** (valves in EJV prevent transmission of RA pressure)

The **left innominate vein** is not in a straight line and may be kinked or compressed between aortic arch and sternum, by a dilated aorta, or by an aneurysm.





Method Of Examination

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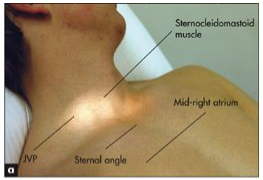
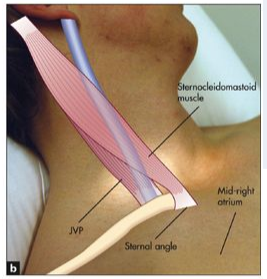
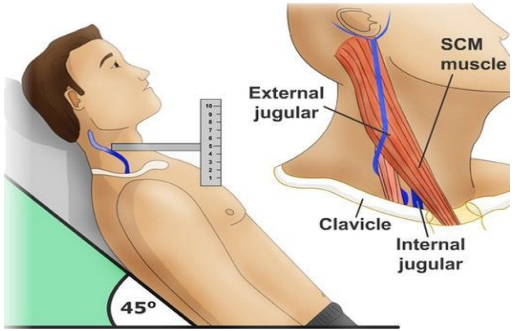




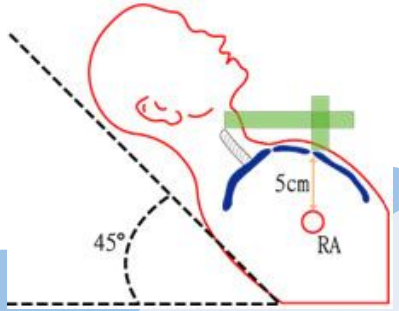
- The patient should lie comfortable during the examination.
- Clothing should be removed from the neck and upper thorax.
- Neck should not be sharply flexed.
- Examined effectively by shining a light across the neck.
- There should not be any tight bands around abdomen.
- Patient reclining with head elevated 45 °



Then Observations is Made

Observe :

1. The level of venous pressure.
2. The type of venous wave pattern.



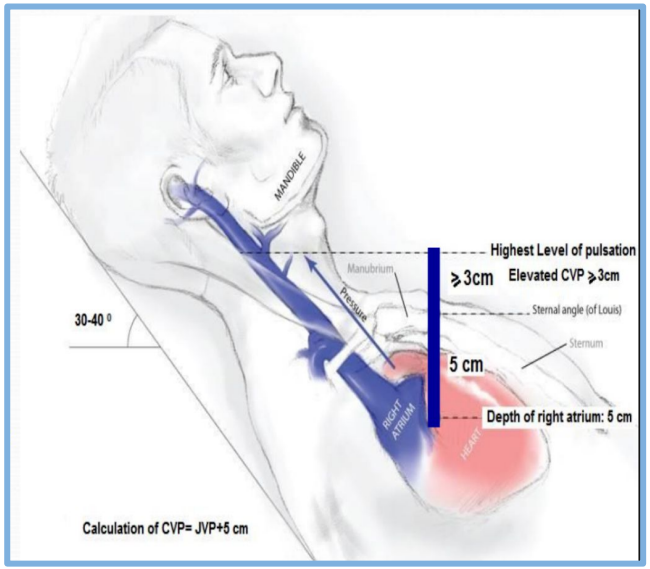
Female slides

The level of venous pressure.

JVP

Using a centimeter ruler, measure the vertical distance between the angle of Louis (sternal angle) and the highest level of jugular vein pulsation.

The upper limit of **normal is 3 cm above the sternal angle.**



JVP= jugular venous pressure
CVP= Central venous pressure

CVP

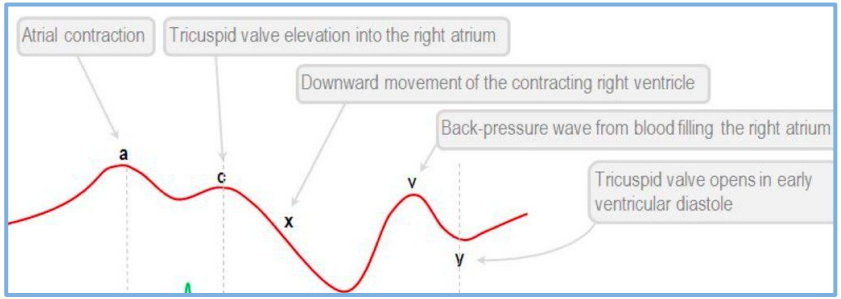
Add 5 cm to level of venous pressure measure **central venous pressure (CVP)** since right atrium is 5 cm below the sternal angle.

Normal CVP is < 8 cm H2O

The type of venous wave pattern.

Female slides

Normal pattern of the jugular venous pulse is the (Atrial pressure changes during the cardiac cycle.)



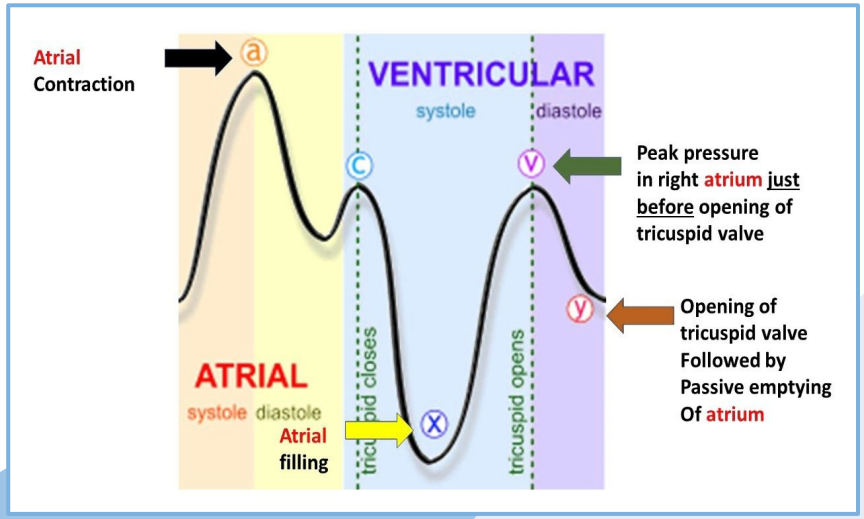
- ❖ The normal JVP reflects phasic pressure changes in the right atrium and consists of:
- Three positive waves (a, c, & v waves).
- Two negative descents (x&y waves).

- ❖ These 3 waves are equal to:

ONE cardiac cycle = 0.8 sec

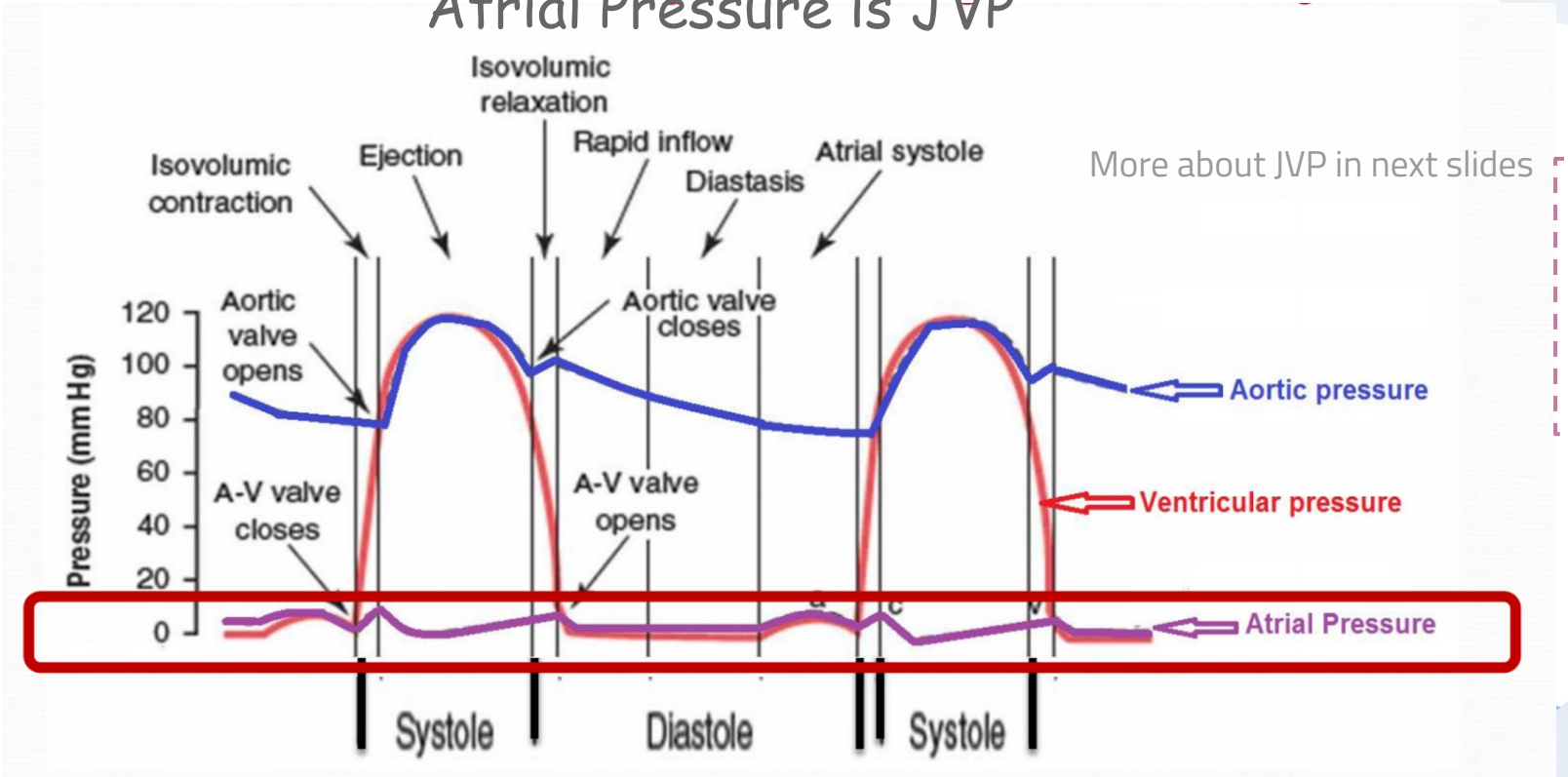
- ❖ components in each wave:

+ve (↑atrial pressure),
- ve (↓atrial pressure).



Atrial Pressure Changes During Cardiac Cycle

Atrial Pressure is JVP



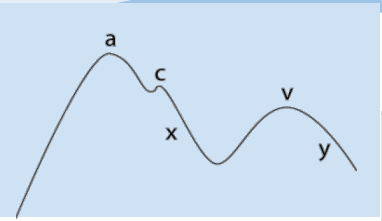
More about JVP in next slides

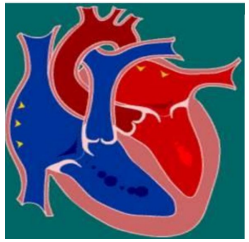
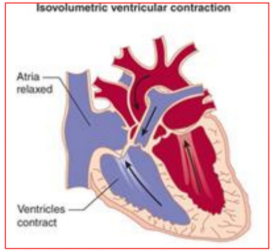
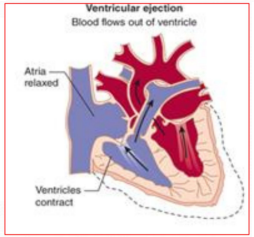
Female slides

See how important cardiac cycle is ??

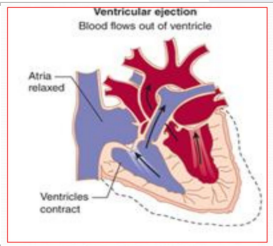
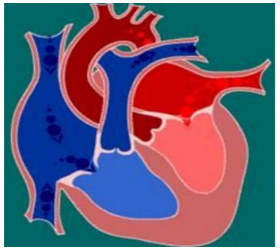
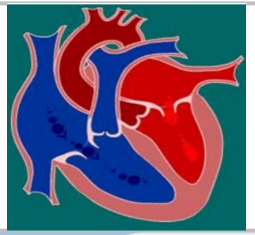
The waves

Female slides



a wave during Atrial systole	**+ve**, venous distension due to RA contraction and retrograde blood flow into SVC and IJV **-ve** due to blood passage into ventricles.	
c wave during Ventricular systole	**+ve** due to ventricular contraction and resulting bulging of tricuspid valve into the right atrium during isovolumetric contraction. (bulging ⟶ ↓ atrial capacity ⟶ ↑ atrial pressure ⟶ ↑ JVP).team 39 **-ve** due to the pulling down of the atrial muscle & A-V cusps during 'rapid ejection phase', resulting ↓in atrial pressure.	 

Female slides

X descent during Ventricular systole	It is due to atrial relaxation and downward displacement of the tricuspid valve during 'reduced ejection phase.'	
v wave during Ventricular diastole	**+ve** due to↑ venous return rising right atrial pressure when blood flows into the right atrium during atrial diastole while the tricuspid valve is shut. **-ve** due to entry of blood into ventricles when the tricuspid valve reopens during 'rapid filling phase.'	
y descent during Ventricular diastole	It is due to decline in right atrial pressure due to entry of blood into ventricles during 'reduced filling phase.'	

Female slides

Abnormalities of Jugular Venous Pulse

Raised JVP

- Increased right ventricular filling pressure (↑ afterload on atria) e.g in heart failure, fluid overload (hypervolemia).
- Obstruction of blood flow from the right atrium to the right ventricle e.g tricuspid stenosis → cannon wave.
- Superior vena caval obstruction e.g retrosternal thyroid goiter (enlargement of thyroid gland).
- Positive intrathoracic pressure e.g pleural effusion, pneumothorax.
- *N.B: The JVP usually drops on inspiration along with intrathoracic pressure (↓ intrathoracic pressure → ↓ JVP). normal condition*

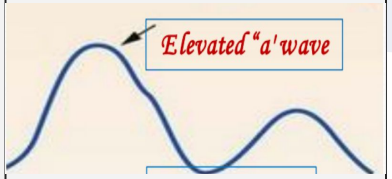
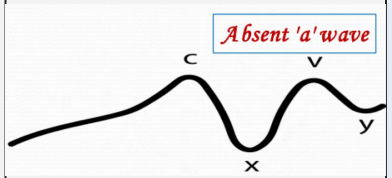
Lowered JVP

- Hypovolemia (anything causing ↓ VR).

Gray text was taken from lab physiology

Abnormal "a" wave

Female slides

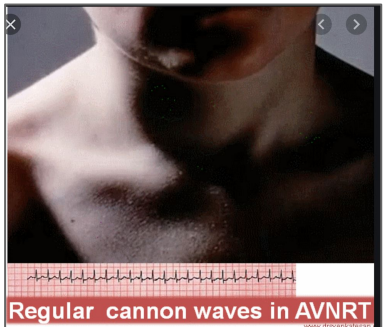
Elevated 'a' wave	Signifies increased right atrial pressure	• Tricuspid stenosis • Decreased ventricular compliance • ventricular failure, • valves stenosis • Pulmonary hypertension • Pulmonary stenosis • Right heart failure	 
Cannon 'a' wave	It occurs when the right atrial pressure becomes very high usually secondary to right atrial contraction against a closed tricuspid valve	• Atrial-ventricular asynchrony (atria contract against a closed tricuspid valve) • Complete heart block(irregular) Third degree heart block • Following premature ventricular contraction • Ventricular tachycardia(regular) • Ventricular pacemaker • Atrial flutter	
Absent 'a' wave	The right atrial pressure does not increase due to failure of proper contraction	Atrial fibrillation or atrial flutter	

Female slides

Cannon wave

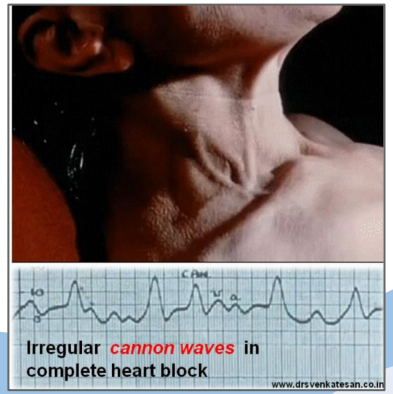
Regular

Regular cannon waves occurs in atrioventricular nodal reentrant tachycardia, when atria and ventricular contractions occur nearly simultaneously.



Irregular

Irregular cannon waves occur in complete heart block.



Males slide start from here
Within females slides

Heart Failure

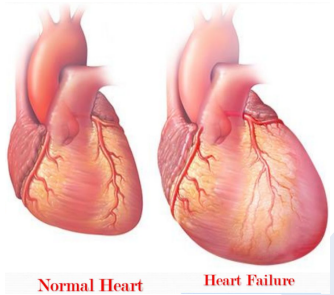
Definition

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 substances despite the venous return to heart is either normal or increased.

So the heart might be receiving blood properly, but it can't pump properly.team 39

How Fast Does Heart Failure Develop?

- Usually a chronic disease,so usually happens over time (chronic) but may happen all of a sudden (acute).
- The heart tries to compensate for the loss in pumping function by:
  - Developing more muscle mass
  - Enlarging
  - Pumping faster

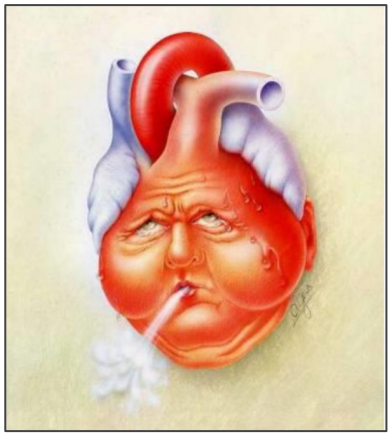


Heart assumes a more spherical shape, enlargement of all 4 chambers

Only in males lecture

Help in understanding physiology of heart failure

- Thus, the resting CO may be low (not contracting properly), normal (the heart is normally pumping but the tissue needs more O2, and the heart cannot do it)or even elevated (in some diseases such as Sepsis, which cause intense vasodilation, so the heart is pumping with great force, but the blood pressure is still falling) , 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:

1-Inadequate cardiac output.

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

Heart Failure causes

1- Impaired cardiac function
The heart muscle gets weak

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

2- Increased cardiac workload
A problem with the workload.

- Hypertension.
- Valvular disorders
- Anemias. to compensate with O2 the heart has to pump more.
- Congenital heart defects

3- Acute non-cardiac conditions

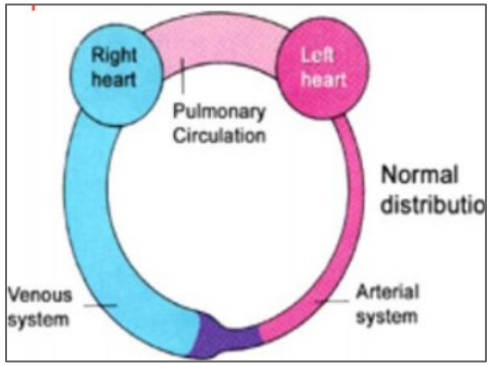
- Volume overload
- Hyperthyroidism, Fever, Infection

Only in males lecture

Heart Failure

Heart failure can involve the left or right side of the heart or both (Congestive heart failure) CHF

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.



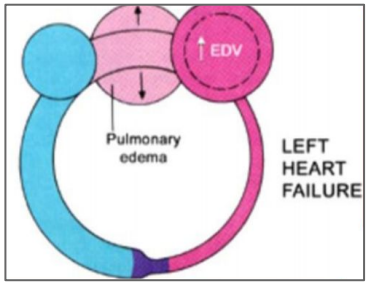
Normal

Types of Heart Failure

Left sided heart failure

Inadequate output of LV causing decreased CO to body and back pressure to the lungs (pulmonary edema).
>The left side of the heart is usually where heart failure begins.

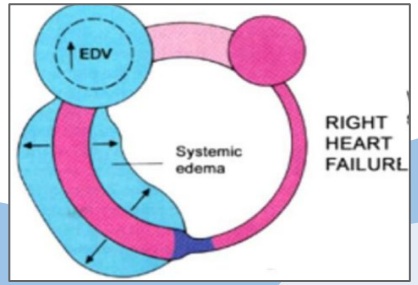
venous return (VR) from pulmonary circulation is not pumped out by the failing LV → blood accumulates in pulmonary circulation → ↑ the pulmonary capillary pressure→ the blood will leak→ **pulmonary edema**



Right side heart failure

Inadequate output of RV causing decreased CO to lungs and back pressure to venous system (systemic edema).
>It may occur alone but is usually a result of left-sided failure.

venous return (VR) from systemic circulation is not pumped out by the failing RV → blood accumulates in systemic circulation → ↑ the systemic capillary pressure → **systemic edema** (ascites,lower limbs in coming slide).



Types of Heart **Dysfunction** that Leads To Hf

Causes of HF in general

1

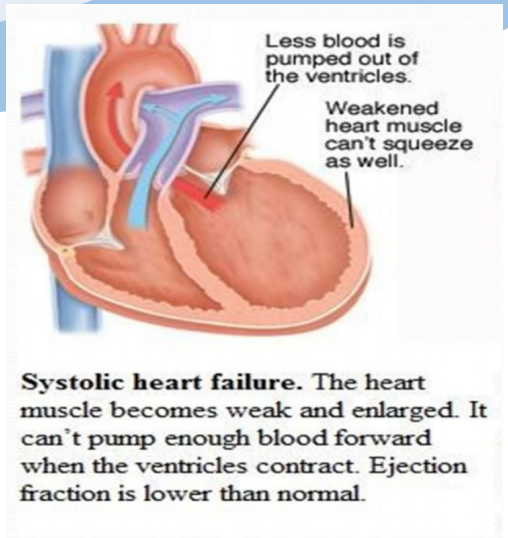
Systolic (or squeezing) heart failure

- This is the most common cause of HF
- The muscle of ventricle is weak and enlarged and loses some of its ability to contract or pump the amount of oxygenated and nutrient-filled blood them body needs into the circulation.
- (i.e. ejection fraction is lower than normal).

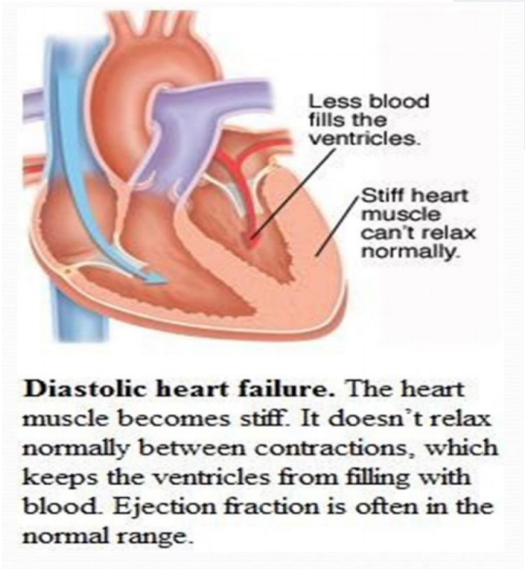
2

Diastolic (or relaxation) heart failure

- The heart loses its ability to relax because it becomes stiff. The walls of the heart thicken, and the size of the chamber may be normal or reduced.
- As a result, the affected chamber cannot fill properly with blood during the rest period that occurs between each heartbeat. So the heart cannot fill properly between each beat
- Ejection fraction is often in normal range.



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.

Congestive heart failure

A Chronic left HF results in Secondary pulmonary hypertension and Right HF and Pulmonary edema. So having right and left hf in the same time .

Characteristic	Diastolic heart failure	Systolic heart failure
Age	Frequently elderly	All ages, typically 50-70yr
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

Causes of *left* Sided HF

Systolic Dysfunction

Impaired Contractility :
➔ Myocardial infarction
➔ Transient ischemia (→fibrous tissue →can't relax or contract)
➔ Chronic volume overload (hypervolemia).
➔ MR/AR (mitral/aortic regurgitation)

Increased Afterload:
➔ Uncontrolled HTN (prolonged hypertension).
➔ AS (Aortic Stenosis)

Diastolic Dysfunction

Obstruction of LV filling:
➔ MS mitral stenosis
➔ Pericardial constriction or tamponade (blood or fluid in pericardial cavity → heart can't relax & expand).

Impaired ventricular relaxation:
➔ Hypertrophic or restrictive cardiomyopathy (stiff).
➔ Transient ischemia

▪ *In both types, blood may "back up" in the lungs causing fluid to leak into the lungs (pulmonary edema)*

▪ *Fluid may also build up in tissues throughout the body (edema)*

Causes of *Right* Sided HF

Pulmonary Parenchymal Disease

-COPD

-Interstitial lung disease

-Chronic infections

-Adult respiratory distress syndrome

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

COR PULMONALE:
Right HF due to chronic lung disease

Acute vs Chronic Heart Failure

Acute Heart Failure:

- Sudden serious abnormalities of the heart (e.g., massive infarction, severe arrhythmias, valve rupture; sepsis) → acute heart failure (hour/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.

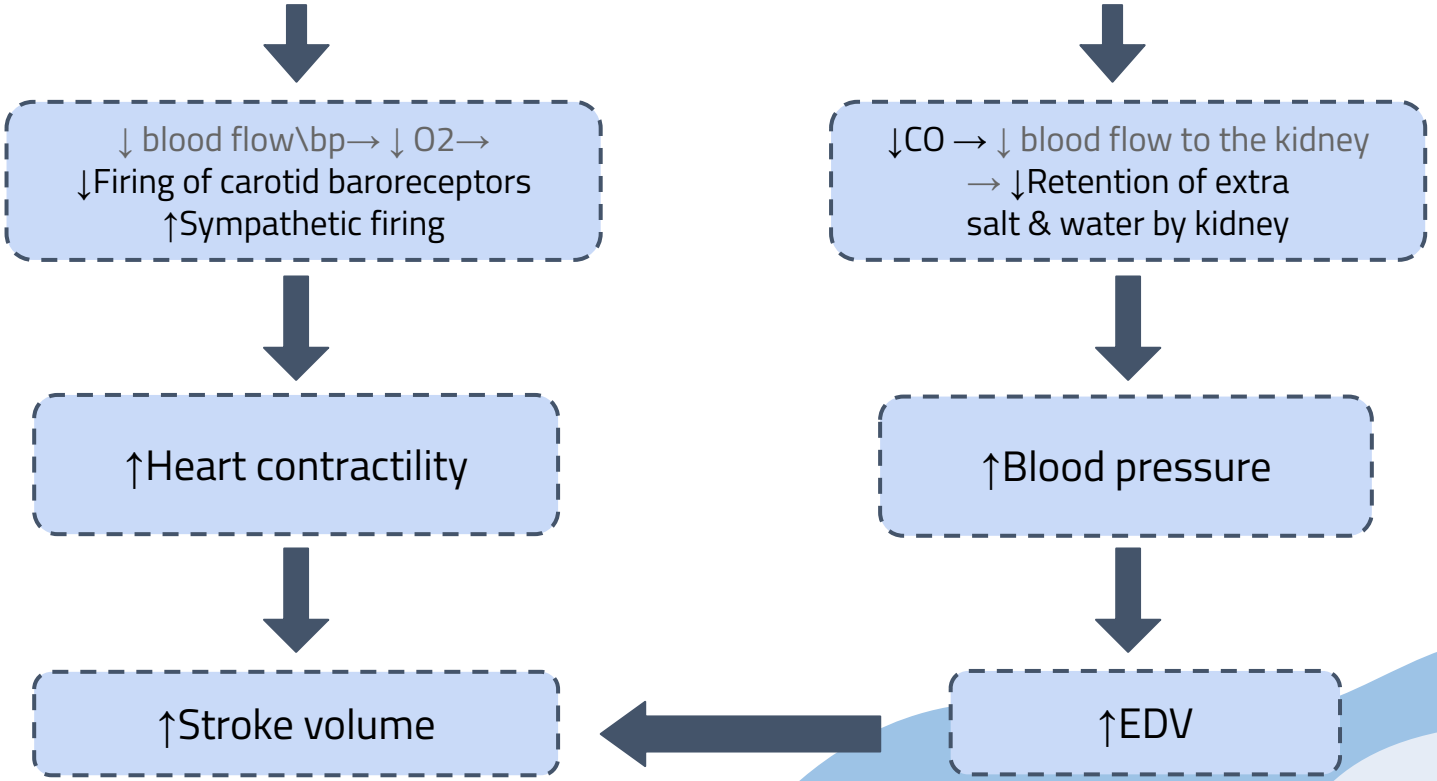
Chronic Heart Failure:

- Chronic heart failure is a long-term condition (months/years).

- It is associated with adaptive responses in the heart, hypertrophy)
- Which can be deleterious.

-carotid baroreceptors: monitor O2 & CO2 content in the blood.
- activation of carotid baroreceptors → ↓ sympathetic activity

Compensatory measures in heart failure



Compensatory Mechanism in CHF

Decreased effective circulating blood volume	Decreased renal perfusion	Decreased firing of carotid sinus baroreceptor
• posterior pituitary releases ADH (vasopressin). • Increased H2O reabsorption.	• Activation of renin-angiotensin-aldosterone system (angiotensin→ vasoconstriction) (aldosterone→ NA & water retention).	• **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 (Atrial natriuretic peptide & Brain natriuretic peptide) **are major antagonizing agents of the renin–angiotensin- aldosterone system.** (will be discussed in Endocrine Block)

Complications of progressive heart failure: Factors contributing to decompensation

- 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.

- Vasoconstriction of the arterioles (under enhanced sympathetic activity): This increases resistance , thus the cardiac afterload. peripheral resistance is determined by arterioles

- Hypertrophied heart : → imbalance between the O2 supply and need (Hypertrophied heart needs more O2 & because of heart failure CO to the heart is less than normal) → deterioration of the ability to generate force.

- Excessive salt and water retention.

- Over-distended ventricle (↑diameter): Has to consume more energy and generate more wall tension to develop the required ejection pressure **(Laplace law).**

Clinical Picture of HF

Poor Cardiac Output → Poor perfusion

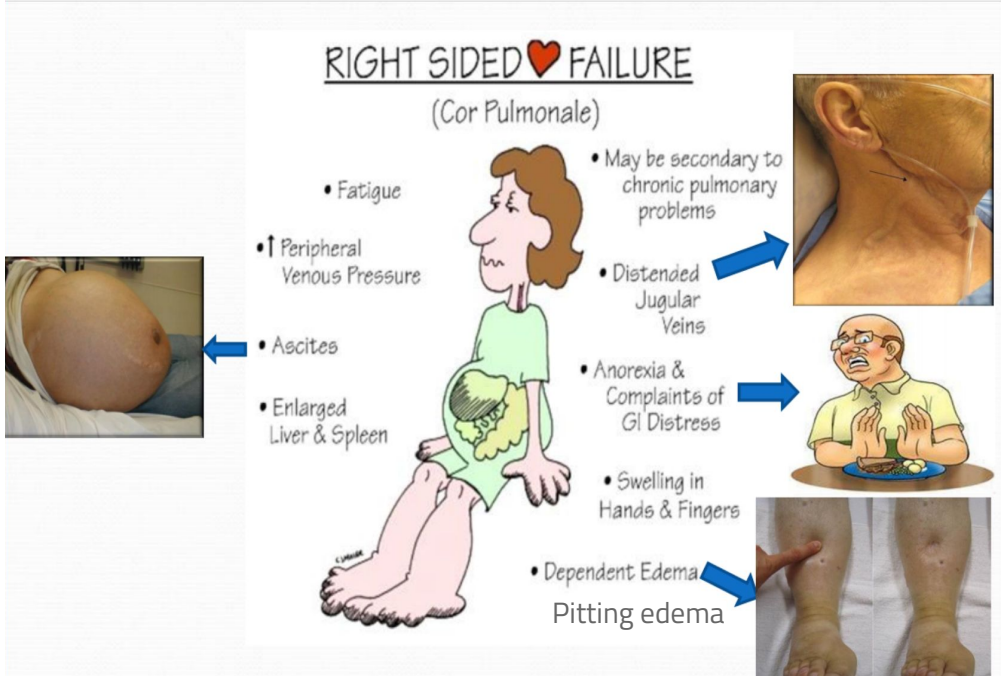
Increased filling Poor Cardiac pressures → Congestion

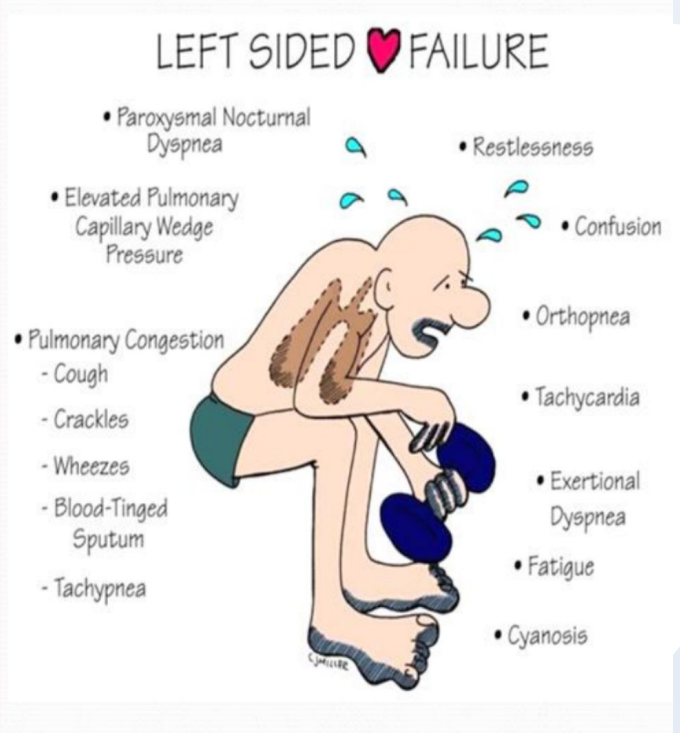
Clinical picture of Right sided heart failure

- Fatigue (↓ blood flow to muscles).
- Ascites. (accumulation of fluid in the abdomen as a result of systemic congestion).
- Enlarged liver & spleen.
- Distended (elevated) jugular veins.
- Anorexia & complaints of GI distress (↓ blood flow to GIT).
- Swelling of hands & Feet.
- Dependent edema.(pitting edema)

Clinical picture of Left sided heart failure

- Tachypnea (↑ rate of respiration), shortness of breath (dyspnea).
- Orthopnea: 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: attacks of severe shortness of breath and coughing at night. It usually awakens the person from sleep, and may be quite frightening.
- Cough, rales (crackles) due to pulmonary edema.
- Restlessness, confusion and fatigue.
- Pallor, cyanosis.
- Tachycardia (compensatory mechanism).


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
Pitting edema


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

Comparison between clinical picture of right & left sided HF

Clinical Picture	Left sided Failure	Right sided Failure
Pitting edema (hands & legs)	**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 of JVP**	**Severe elevation in JVP,** Why? because it's directly connected. **Neck veins are visibly distended**
Shortness of Breath	**Prominent dyspnea. Paroxysmal nocturnal dyspnea and orthopnea.**	**Dyspnea is present but not as prominent**
GIT symptoms: loss of appetite, bloating, constipation.	**Present but not as prominent as in right-sided failure**	**Significantly more prominent than in left-sided failure**

How heart failure is diagnosed?

Tests:

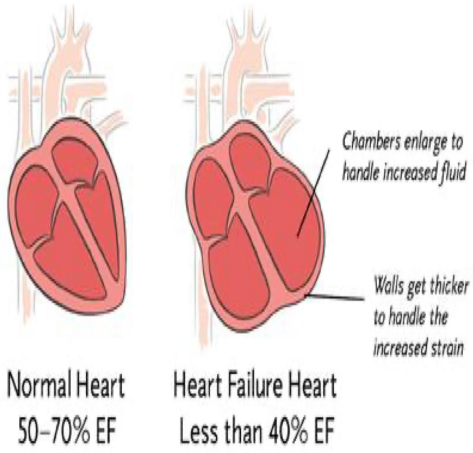
- Chest X-ray.
- Electrical tracing of heart (ECG).
- Ultrasound of heart (Echocardiogram or "Echo").
- X-ray of the inside of blood vessels (Angiogram).

Physical Examination

Medical history is taken to reveal symptoms

A key indicator for diagnosing heart failure **Ejection Fraction**

Ejection Fraction (EF):	is the percentage of blood that is pumped out of the ventricle during each beat. If it is less than 50% then it's heart failure. What mechanism is used to measure EF? Fractional Shortening
Fractional Shortening:	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. It's simply the difference between the most dilated state and most contracted state of the heart, the higher it is the better it is.



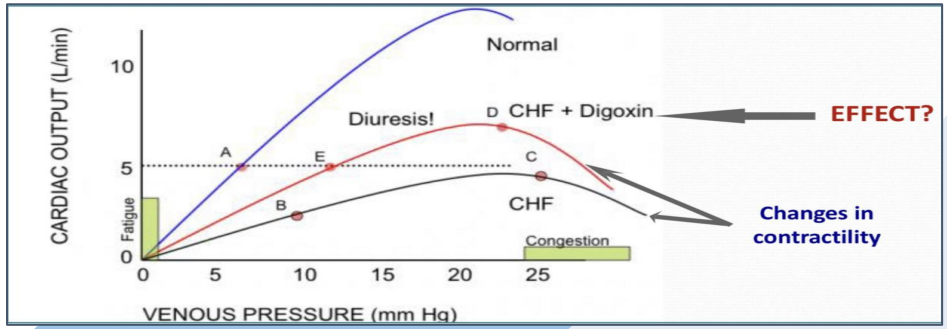
Treatment of Cardiac Failure

Aims at

- Reduction of salt & water retention. (which will decrease preload). → **Diuretics**
- ↑In the pumping activity of failing heart → **Digitalis**

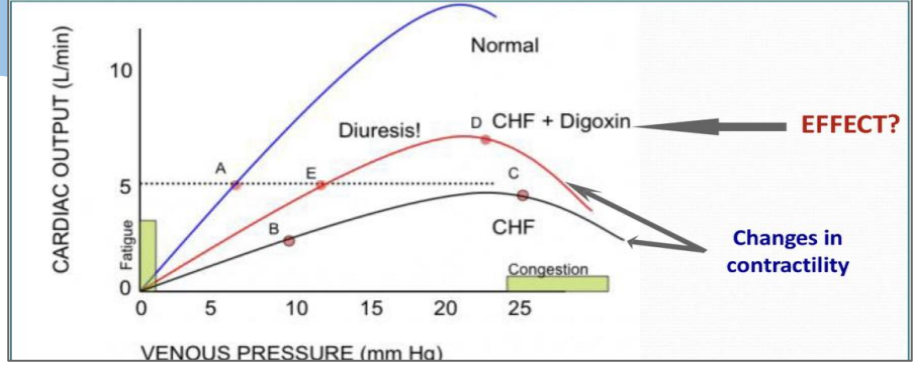
★ Digitalis improves pumping activity of heart by increasing cytosolic Ca++

- **Effects of congestive heart failure & digoxin on Frank-starling curve**



Explained in more details next slide

Special Thanks to med439



Digitalis (Digoxin): will shift the curve to the left and Up.

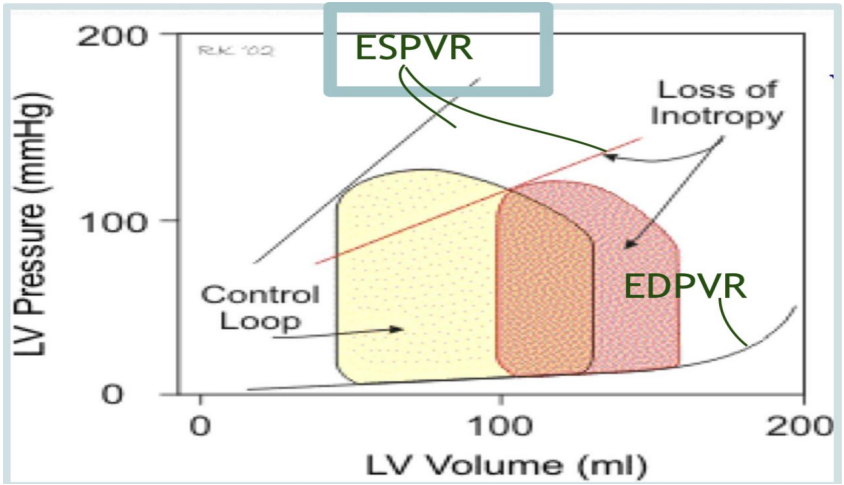
Explanation:

Effect of heart failure and digoxin on the Frank-Starling relationship. The normal set point required to maintain adequate tissue perfusion is a cardiac output of 5 L/min. During heart failure, the relationship between cardiac output and venous pressure becomes shifted down and to the right (patient moves from point A to B). Sympathetic activation and increased fluid retention result in an increased venous pressure (preload) which acts to increase cardiac output by increasing the stretch of cardiac fibers (patient moves from B to C). If cardiac output remains below 5 L/min, the kidney continues to retain fluid, and venous pressure continues to rise, until either a 5 L/min cardiac output is achieved, or the patient "drowns in their own fluids" (e.g. due to pulmonary congestion).Digoxin can shift the curve upwards and to the left by a mechanism different from sympathetic stimulation (so that the patient ideally moves from point C to D). The resulting increase in blood flow to the kidney results in a diuresis (patient moves from D to E) with an associated reduction in venous pressure due to reduced venous volume.

Mechanism of Action

Digoxin exerts its positive inotropic action primarily by binding to and inhibiting the Na/K ATPase in cardiac cell membranes. The Na/K ATPase enzyme acts as a pump for the outward transport of Na+ in exchange for the inward transport of K+. The Na/K ATPase contains a receptor for digitalis glycosides, as well as for intracellular Na+ and extracellular K+. Digoxin's inhibition of the Na/K pump results in an increase in intracellular [Na]. Due to the presence of a Na/Ca antiporter, a rise in intracellular [Na+] also results in a rise in a consequent rise in intracellular [Ca2+] (see Figure X). Most of this rise in [Ca2+] is taken up into the sarcoplasmic reticulum (SR), and then released into the cytoplasm upon stimulation by an action potential. This larger Ca release

Effects of left ventricular ***systolic*** failure on left ventricular pressure volume loop.

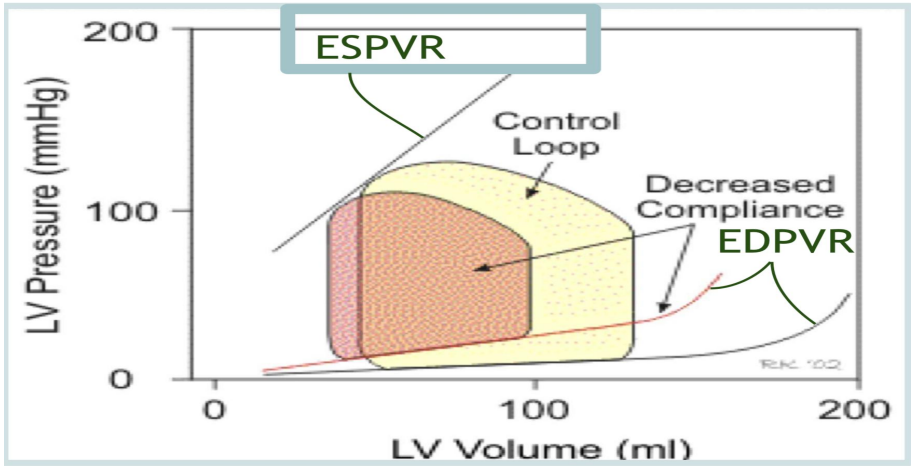


↓Slope of End-systolic pressure- volume relationship (ESPVR)
i.e.
↑ESV compensatory rise in preload sense the ventricle is not pumping well there will
be more blood left
↑EDV
↓SV, because the ventricle is not pumping properly
↓EF
↓ external Work (work done by the heart) (not CO)
↑EDP, because the EDV has increased.
Heart rate is unchanged.

remember PV loop from cardiac cycle?

work → shaded area

Effects of left ventricular ***diastolic*** failure on left ventricular pressure volume loop.



↓Ventricular compliance/ relaxation (lusitropy).
↓EDV
↓SV
↓or no change in EF
↓ external Work
↑EDP
↑ slope End-systolic pressure- volume relationship (ESPVR)
Heart rate, inotropy and systemic vascular resistance are unchanged.

Team Leaders

Rand aldajani

Nawaf Alshehri

QBank Team

Sub Leader

Samiah AlQutub

Team Members

Sahar alghamlas

Saad Alasmari