# Jugular venous pulse & Heart Failure

Physiology Team 439

MED439
KING SAUD UNIVERSITY

Black: in male / female slides

Red : important

Pink: in female slides only

Blue: in male slides only

Green: notes

Gray: extra information

**Helpful videos:**
**JVP :** click here
**HF :** click here
**HF Animation:** click here

Editing File

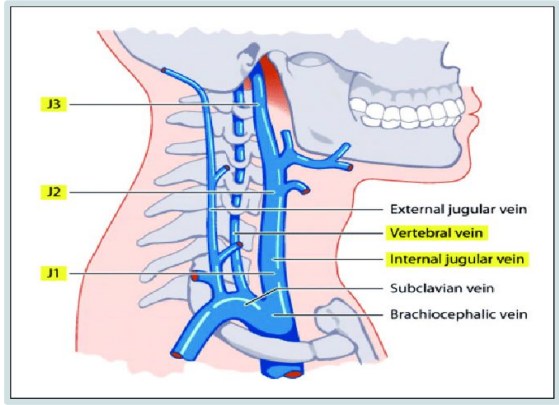
# Objectives

- 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, and 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 failure, and failure treated with digoxin.

# Definition of:

- **Jugular Venous Pulse**:
  Defined as the oscillating top of vertical column of blood in right internal jugular vein. It reflects pressure changes (pressure waves) in right atrium during the cardiac cycle.

- **Jugular Venous Pressure**:
  Vertical height of oscillating column of blood in right internal jugular vein.
  significance? helps to estimate the degree of increase in pressure in the right atrium.




## Why Right Internal Jugular Vein (IJV)?

- 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 haemodynamic changes (pressure changes) from the right atrium
- IJV is anatomically closer to RA.
- 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.

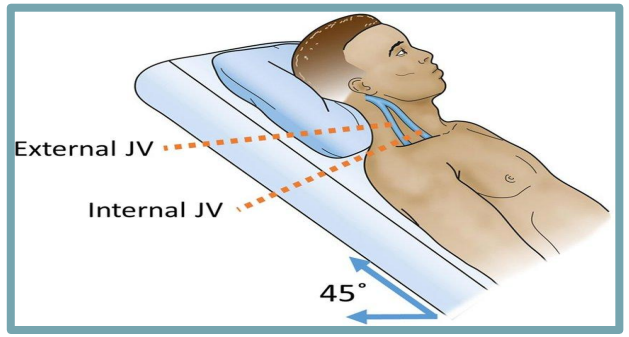
# Method Of Examination:

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

# Observations Made:

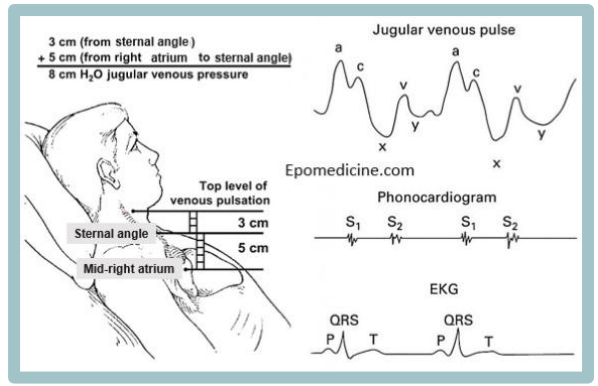
**When the patient reclining with head elevated 45 °, observe:-**

1. The level of venous pressure.
2. The type of venous wave pattern **(jugular venous pulse).**



# The level of venous pressure

- Using a centimeter ruler, measure the vertical distance between the angle of Louis and the highest level of jugular vein pulsation.
- The upper limit of normal is 3 cm above the sternal angle.
- Add 5 cm to measure central venous pressure since right atrium is 5 cm below the sternal angle. **(more than 3 above sternal angle = ↑ JVP = ↑ pressure in the right atrium.)**
- Normal CVP is < 8 cm H2O.

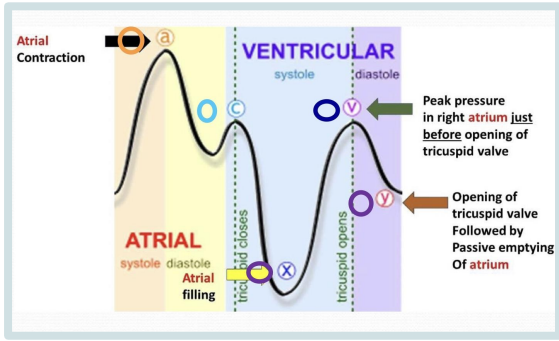



## Normal pattern of the jugular venous pulse Atrial pressure changes during the cardiac cycle

**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).

2 components in each wave:
+ve ( ↑atrial pressure), - ve ( ↓atrial pressure).
These 3 waves are equal to ONE cardiac cycle = 0.8 sec




Each wave explained next slide

| The Wave | Description |
| --- | --- |
| **The "a" Wave: Atrial systole:** | - +ve, venous distension due to RA contraction and retrograde blood flow into SVC and IJV (because they don't contain valves → few mmHg increase of pressure).<br>- -ve due to blood passage into ventricles. |
| **The "c" wave: 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).<br>▪ -ve due to the pulling down of the atrial muscle & A-V cusps during 'rapid ejection phase', resulting in ↓atrial pressure. |
| **The "x" descent:** | It is due to atrial relaxation and downward displacement of the tricuspid valve during 'reduced ejection phase.' |
| **"v " Wave** | +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.<br>-ve due to entry of blood into ventricles when the tricuspid valve reopens during 'rapid filling phase.' |
| **"y" Descent** | It is due to decline in right atrial pressure due to entry of blood into ventricles during 'reduced filling phase.' |

# 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

## Lower JVP

- ➔ Hypovolemia (anything causing ↓ VR).

# Heart Failure

**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 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.

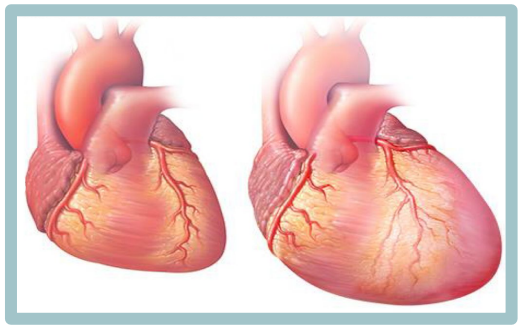
- Thus, the resting CO may be low (not contracting properly), normal (the heart is normally pumping but the tissue needs more O2, and the heart cannot do it)or even elevated (in some diseases such as Sepsis, which cause intense vasodilation, so the heart is pumping with great force, but the blood pressure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

## How Fast Does Heart Failure Develop?

Usually a chronic disease.

The heart tries to compensate for the loss in pumping function by:

- Developing more muscle mass (hypertrophy)
- Enlarging
- Pumping faster



**Normal Heart** **Heart Failure**

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

Male slide only

# 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

# Types of Heart Failure

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

## Left sided heart failure

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

In left-sided failure, venous return 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 sided heart failure

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

In right-sided failure, 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).

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.

# Types of Heart Dysfunction That Lead To Heart failure

## 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 (→ ↑ EDV) the amount of oxygenated and nutrient-filled blood the body needs into the circulation.

(i.e. ejection fraction is **lower than normal**)

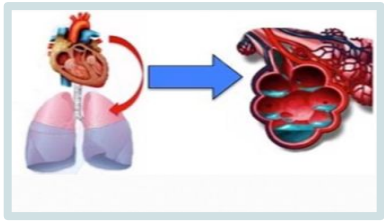
## Diastolic (or relaxation) heart failure

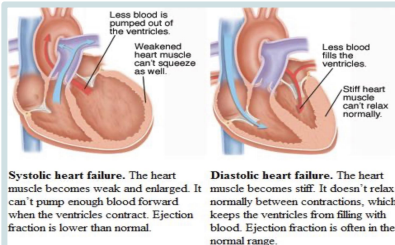
- The heart loses its ability to relax because it becomes stiff (pumping power is normal). As a result, the affected chamber cannot fill properly with blood during the rest period that occurs between each heart beat (→ ↓ EDV → ↓ SV ).
- Ejection fraction is often in **normal** range ( because the heart is normally contracting, but it's not receiving, and both EDV & SV decreases).
- Often the walls of the heart thicken, and the size of the chamber may be normal or reduced. If the heart can't relax properly then it can't pump properly.

## Congestive heart failure

Chronic left HF results in:-

- Secondary pulmonary hypertension
- Right HF
- Pulmonary edema





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| 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).
- ➔ 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

**Pulmonary Vascular Disease**
-Pulmonary embolism
-Pulmonary HTN
-Right ventricular infarction

**Cardiac Causes**
-Usually occurs as a result of left HF
-Pulmonary stenosis
-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.

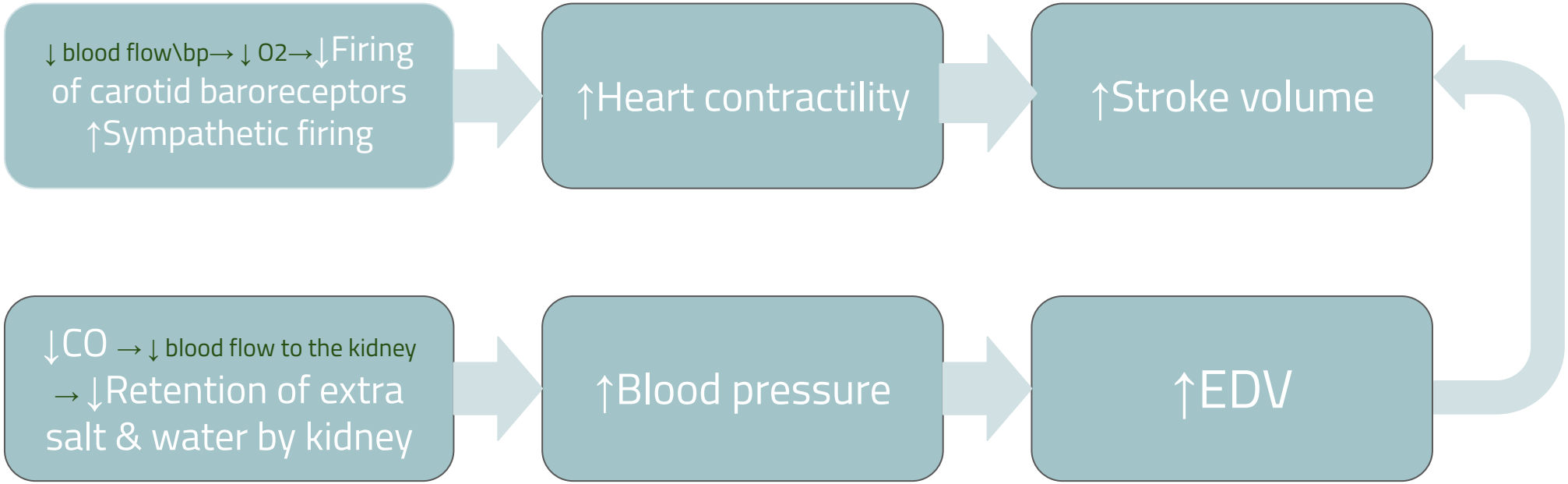
## Chronic heart failure

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

# Compensatory measures in heart failure

carotid baroreceptors: monitor O2 & CO2 content in the blood.

- activation of carotid baroreceptors → ↓ sympathetic activity.



# Compensatory mechanism in CHF

## Decreased effective circulating blood volume

- posterior pituitary releases ADH (vasopressin).
- Increased $H_2O$ reabsorption.

## Decreased renal perfusion

- Activation of renin-angiotensin-aldosterone system (angiotensin→ vasoconstriction) (aldosterone→ NA & water retention).

## 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** (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

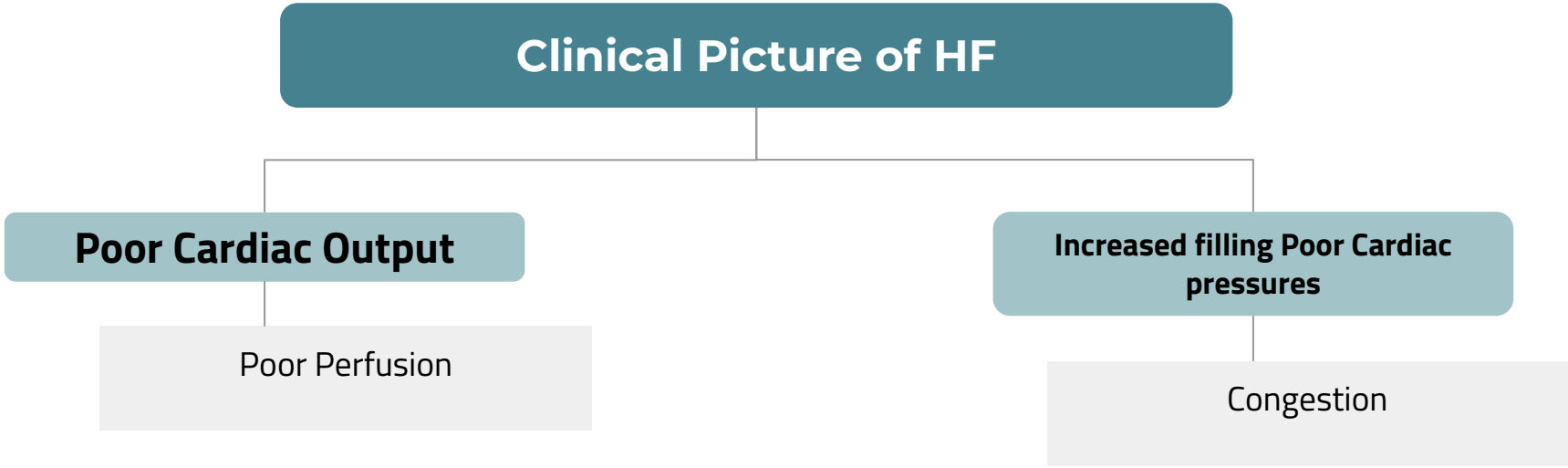
- Prolonged sympathetic activation to the heart: down regulation of the myocardial adrenergic receptors → ↓ the myocardial adrenergic receptors density and sensitivity to catecholamines. Consequently, the inotropic and chronotropic responses of the heart cannot be elevated in parallel to increased body 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).




**symptoms & signs depend on the organ with ↓ perfusion**

## 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.

## 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).

# 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

**Diagnosis of the Heart failure**

**Tests**

**Physical examination**

**Medical history is taken to reveal symptoms**

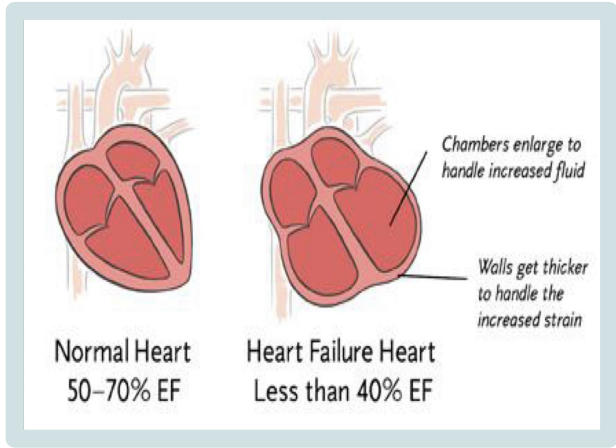
- Chest X-ray.
- Electrical tracing of heart (ECG).
- Ultrasound of heart (Echocardiogram or "Echo").
- X-ray of the inside of blood vessels (Angiogram).

# 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.



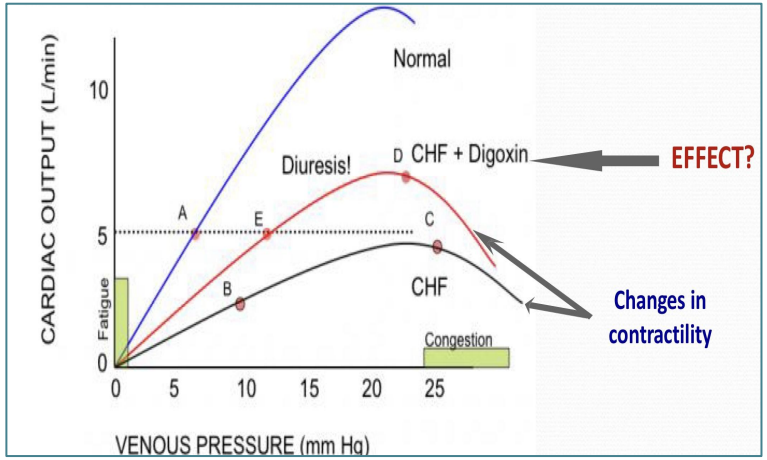
# Treatment of cardiac failure

**Aims at**

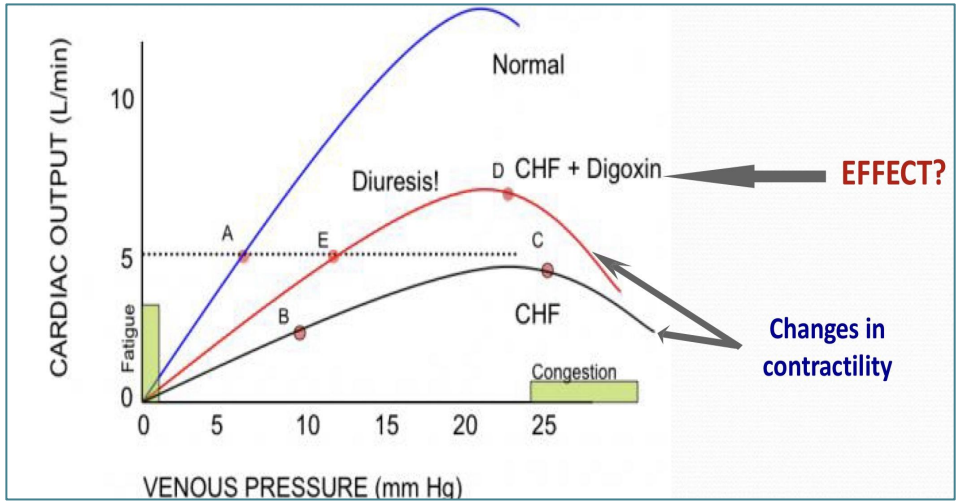
- Reduction of salt & water retention. which will decrease preload. → **Diuretics**
- **↑In the pumping activity of failing heart** → **Digitalis**

**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




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.

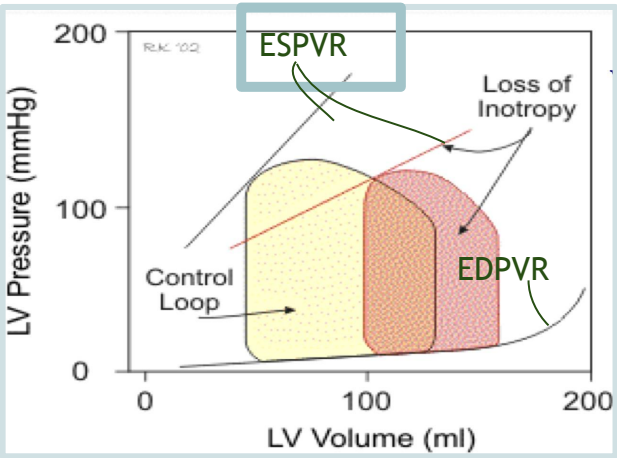
**Mechanism of Action**

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.

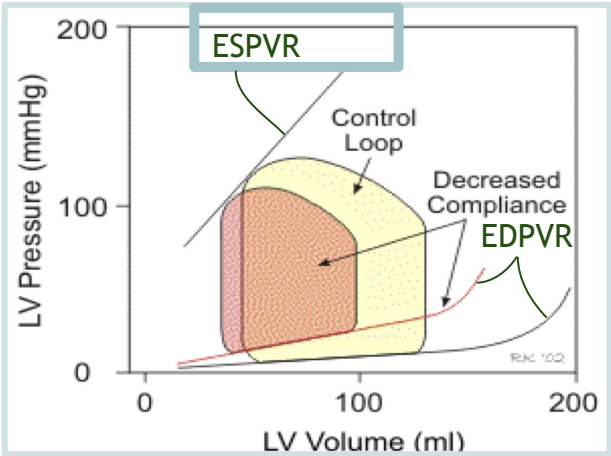
work → shaded area

**remember PV loop from cardiac cycle?**




↓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.

# 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.

# Quiz:

**1-Which of the following is a Characteristic of JVP?**
A) Increased during diastole
B) Increased in right sided heart failure
C) Decreased in right sided heart failure
D) Decreased during systole

**2-In right-sided heart failure, the organ that gets enlarged is:**
A) Lungs
B) Liver
C) Kidney
D) Heart

**3-Bulging of the tricuspid valve causes which of the following waves?**
A) C- wave
B) A- wave
C) V- wave
D) X- wave

**4-which of the following is a cause of diastolic dysfunction in left sided HF?**
A) myocardial infarction
B) mitral stenosis
C) aortic stenosis
D) uncontrolled HNT

**5-heart failure can occur?**
A) in left side then right side
B) only in the left side
C) only in the right side
D) all of the above

**6-Raised jugular venous pressure can be due to?**
A) Increased right ventricular ejection pressure
B) Obstruction of blood flow from the right atrium to the right ventricle
C) Hypovolemia
D) negative intrathoracic pressure

**7-Right sided heart failure causes?**
A) aortic stenosis
B) pulmonary stenosis
C) pulmonary emboli
D) B&C

**8-Which of the following is compensatory mechanism of CHF**
A) Increased effective circulating blood volume
B) Increased firing of carotid sinus baroreceptor
C) Deactivation of renin-angiotensin-aldosterone system
D) Decreased renal perfusion

**9-Which of the following cause raised jugular venous pressure?**
A) Superior vena caval obstruction
B) decreased right ventricular filling pressure
C) Hypovolemia
D) A & B

**10- which of the following is a clinical sign of left sided heart failure?**
A) ascites
B) anorexia
C) orthopnea
D) edema

**Answer Key:** 1.B 2B. 3. A- 4B - 5D
6B - 7D - 8D - 9A - 10C

# Quiz:

**1- definition of heart failure?**

**2- How Heart failure can be diagnosed?**

**3-What is the definition of ejection fraction?**

**A1:** 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.

**A2:** 1- taking history to reveal symptoms 2- physical examination 3-tests (Chest X-ray, ECG, Echocardiogram, angiogram).

**A3: EF** is the percentage of blood that is pumped out of the ventricle during each beat.

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