جامعة الملك سعود
King Saud University

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

غيداء آل مصمع
عبدالرحمن الحيسوني
Revised by

Index:

Red: important Grey: extra information Green: doctor's notes yellow: numbers

Purple: only in female slides Blue: only in male slides

Physiology Team437

MED437
KING SAUD UNIVERSITY

Objectives

- Define stroke volume, cardiac output, venous return, cardiac index & cardiac reserve
- Understand the concept of preload and afterload
- Describe the factors affecting the SV & CO
- Explains how cardiac contractility & rate affects SV
- Describe and explain the pressure-volume loop
- 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
- Know how fast does heart failure develop
- Identify types of heart failure
- Discuss the causes of heart failure
- Know the symptoms & signs of heart failure
- Indicators for diagnosis of heart failure

Definitions

- End-diastolic volume (EDV):
  Volume of blood in ventricles at the end of diastole = 110-130 mL.

- Stroke volume (SV):
  Amount of blood ejected from ventricles during systole (per each beat) = EDV-ESV = around 70 ml in an average adult at rest = 70-80 mL/beat

  - End Diastolic Volume and End Systolic Volume determine the Stroke Volume.

- End-systolic volume (ESV):
  Amount of blood left in ventricles at the end of systole = 40-60 mL.

- Ejection fraction (EF):
  Fraction of end-diastolic volume ejected during a heart beat =stroke volume/end diastolic volume = 60-65 %.

Cardiac Output

IF the amount of blood pumped by the left ventricle per beat = Stroke Volume (≈70ml).

THEN

What is the amount of blood pumped by the left ventricle per minute?

= Stroke volume (SV) X heart rate (HR) = Cardiac output (CO) = 70 x 70 ≈ 5L/min

- Cardiac Output: Volume of blood ejected by each ventricle in each minute. (around 5 liters in an average adult at rest)
- Since the normal adult blood volume is about 5 L, the entire blood supply passes through the body once each minute.
- Cardiac Output = Stroke Volume x Heart Rate

ONLY in female slides

Normal Values of Cardiac Output at Rest

- Resting cardiac output:
  - The average cardiac output for the resting adult is 5L/min.
  - CO vary with size of individual.
  - Children have smaller CO than adults.
  - Women have smaller CO than men.
  - For men, CO ≈ 5.6L/min.
  - For women, ≈ 4.9L/min.

- Factors affecting the Cardiac Output:
  - Body metabolism
  - Exercise
  - Hyperthyroidism
  - Pregnancy
  - Increased body temperature

Cardiac Index and Cardiac Reserve

- Cardiac Index: is Cardiac Output per Square Meter of Body Surface Area.
  - CI = CO/m2
  - It relates the cardiac output to body surface area. (CO/min/m2 of body surface area)
  - Thus relating heart performance to the size of the individual.
  - Normal Cardiac index= (3.2 Liters/min/m2 body surface area).
- Venous Return: is the Quantity Of Blood Flowing from the Veins into Right Atrium each Minute.
  - CO = VR
- Preload: is the amount of blood presented to the Ventricles.
- Afterload: is the resistance against Which the ventricles contract.

- Cardiac Reserve: The difference between the resting CO and the maximum volume of blood the heart is capable of pumping per minute.
- During exercise, the CO can increase to 20 - 25 liters/min and to as high as 35 - 40 liters/min in well trained athletes.

ONLY in female slides

Regulation of CO

- CO is crucial since it is also the amount of blood that flows into the circulation and is responsible for transporting substances to and from the tissues.
- Thus, the body has strict control mechanisms that maintain adequate CO.
- There are 2 major factors which determine CO:
  - Stroke Volume (SV)
  - Heart Rate (HR)
- And Stroke Volume = End Diastolic Volume (EDV) - End Systolic Volume (ESV).
- So any factor that affects these parameters (EDV, ESV and HR) will affect CO.

ONLY in male slides

- Determinants of Cardiac Output:
  - Afterload
  - Preload
  - Myocardial Contractility (Inotropic state)
  - Heart rate (Chronotropic state)

Determinants of CO

1. Heart rate (HR)

- Since the CO is = SV . HR, as the heart rate increases, CO increases.
- At heart rates up to about 180, ventricular filling is adequate as long as there is enough venous return, and cardiac output per minute is increased by an increase in heart rate.
- However, at very high heart rates, filling may be compromised to such a degree that cardiac output per minute falls.
- The heart rate has an influence on cardiac contractility as well (Frequency-force relationship).

ONLY in male slides

Determinants of the cardiac output

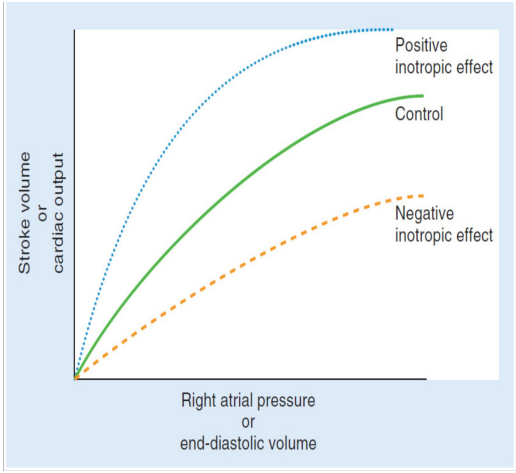
- Afterload
- Preload
- Myocardial contractility (inotropic state)
- Heart rate(chronotropic state)

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.vv



1-Effect of ions & temperature:

ONLY in male slides

In the following We will discuss about:
1- Effect of ions & temperature
2- Factors affecting contractility
3- Factors affecting myocardial contractility (inotropic factors)

1-increasing in K+ :

Increasing of K in the extracellular fluids causes the heart to become dilated and flaccid and also slows the heart rate

Increasing of K decreases the resting membrane potential in the cardiac muscle fibers

the intensity of the action potential also decreases, which makes contraction of the heart progressively weaker.

2-increasing in CA_{2+} :

Ca increase causes spastic contraction. This is caused by a direct effect of calcium ions to initiate the cardiac contractile process

Ca decreasing causes flaccidity of the muscle.

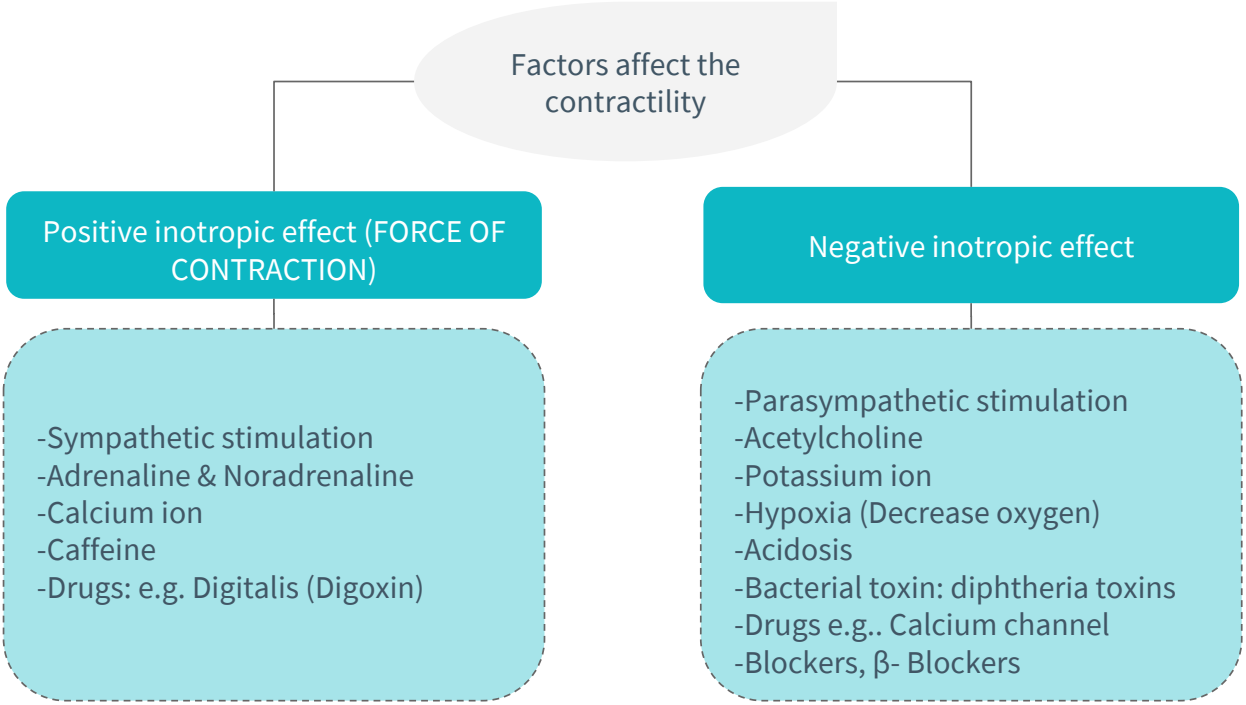
3- Temperature :

Increase the temp. causes a greatly increased heart rate, sometimes to as fast as double normal [increase permeability to ions that self-excitation].

Decreased temperature causes a greatly decreased heart rate, falling to as low as a few beats per minute.

ONLY in male slides

2-Factors affecting the contractility:



ONLY in male slides

3-Factors affecting the myocardial contractility: (inotropic factors)

1. **End-Diastolic Volume** [Starling's law of the heart]
2. **Cardiac innervation** [SNS > increase , PNS > decrease]
3. **Oxygen supply** [Hypoxia > decrease the contractility]
4. **Calcium increase & potassium ions decrease contractility**.
5. **Physical factors** [Warming > increase , Cooling > decrease]
6. **Mechanical factors** [syncytium, cannot be tetanized]
7. **Hormonal & chemical factors** (drugs).

-What is the influence of heart rate on myocardial contractility?
Frequency-force relationship; Increasing heart rate increases contractility.

-This frequency dependency of force generation in the heart is probably due to accumulation of Ca^{2+} ions within the myocytes as a result of:
1-The increased in number of depolarizations/min → more frequent plateau phases → more Ca^{2+} entry.
2-The magnitude of Ca^{2+} current is also increased → increases the intracellular Ca^{2+} stores.
3-Both effects enhance the release and uptake of Ca^{2+} by the sarcoplasmic reticulum, thus Ca^{2+} availability to the contractile proteins with more force generation through cross-bridge cycling.

Determinants of the CO
2. Stroke volume (SV)

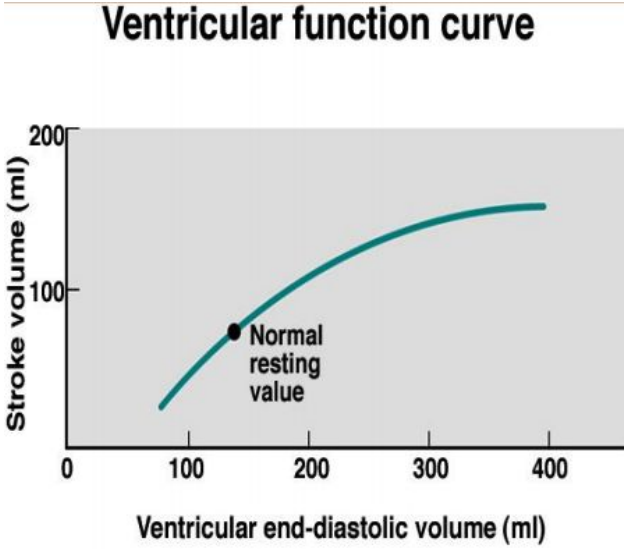
CO = SV × HR

SV = End-diastolic volume - End systolic volume

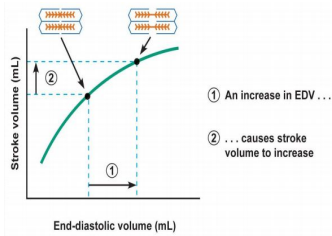
CO = (EDV - ESV) × HR

Any factor that affects these parameters will affect the CO.

The **intrinsic relationship** between EDV and SV is known as Starling's Law of the heart. It reflects the ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return.



Frank – Starling's Law



- The Frank Starling principle is based on the **length-tension** relationship within the ventricle.
- If EDV (preload) is increased, the ventricular fiber length is also increased, resulting in an increased 'tension' of the muscle (i.e. **the stroke volume increases in response to increase of the end diastolic volume).**
- This is called the Frank-Starling mechanism (or Starling's Law of the heart).
- Within physiological limits, the heart pumps all blood comes to it without allowing stasis of blood in veins.

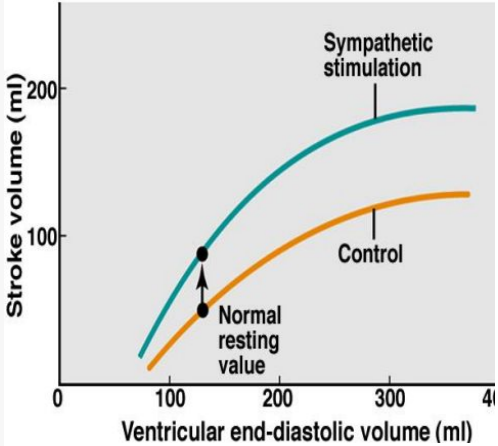
(Note:If the venous return increase beyond physiological limits, the heart will get stretched out and will not contract properly.)

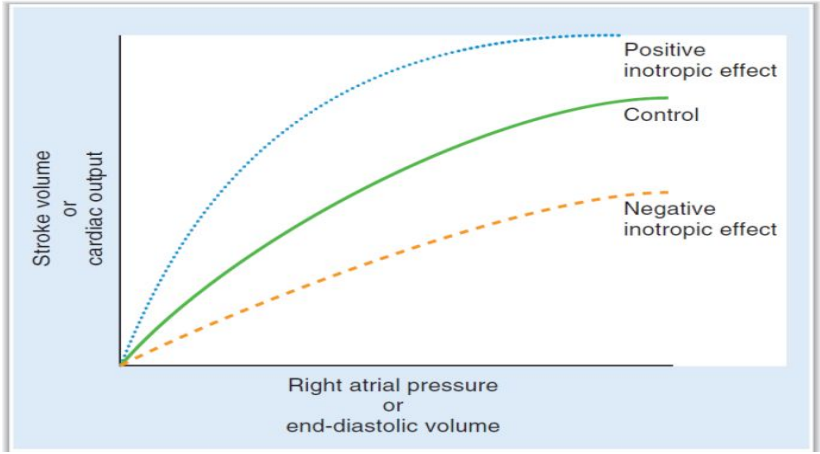
- The greater the stretch of the cardiac muscle, the greater would be the force of contraction.
- " The energy of contraction is proportional to the initial length of the cardiac muscle fibers" and for the muscle is proportional to the End Diastolic Volume.

Because Actin and Myosin filaments are brought to more optimal degree of sliding, therefore increase force of contraction. (only in males slides).

ONLY in male slides

- When the mean RAP is about 0 mmHg (note that RAP normally fluctuates with atrial contraction and respiration), the cardiac output in an adult human is about 5 L/min.
- Because of the steepness of the cardiac function curve, very small changes in RAP (just a few mmHg), can lead to large changes in cardiac output.



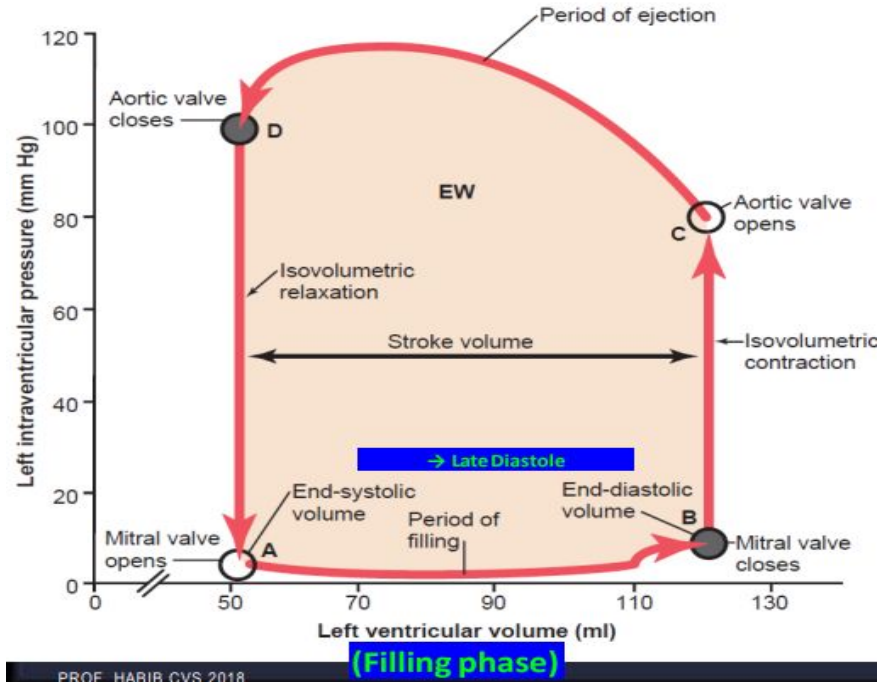


ONLY in male slides

Ventricular Pressure - Volume Loop

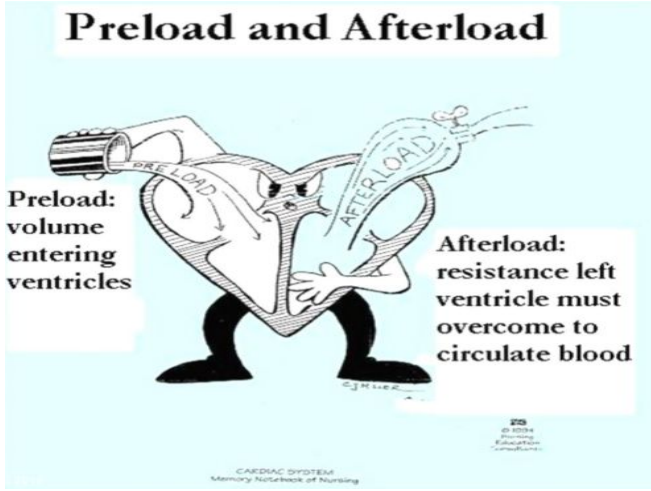
Plots LV pressure against LV volume through one complete cycle .

- **Systole:** divided into early systole and late systole.
- **Diastole:** divided into early diastole and late diastole.



Preload and Afterload

- **Preload:** it is the amount of blood that returns to the heart from veins. (end diastolic pressure)
- Preload is the load on the muscle in the relaxed state.
- 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.
- Increased afterload= increased cardiac workload.
- 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.



Female slides only	Preload	Afterload
Increased in :	Hypervolemia Heart failure	Hypertension Vasocon-striction

ONLY in male slides

Preload and Afterload

a. **Increased preload:** refers to an increase in end-diastolic volume and is the result of increased venous return.

Causes an **increase in stroke volume**

based on the Frank-Starling relationship ..

Reflected <u>increased width</u> of the

pressure-volume loop.

b. **Increased afterload:** refers to an increase in aortic pressure. The ventricle must eject blood against a higher pressure, resulting in a decrease in stroke volume… is reflected in <u>decreased width</u> of the pressure-volume loop. The decrease in stroke volume results in an increase in end-systolic volume.

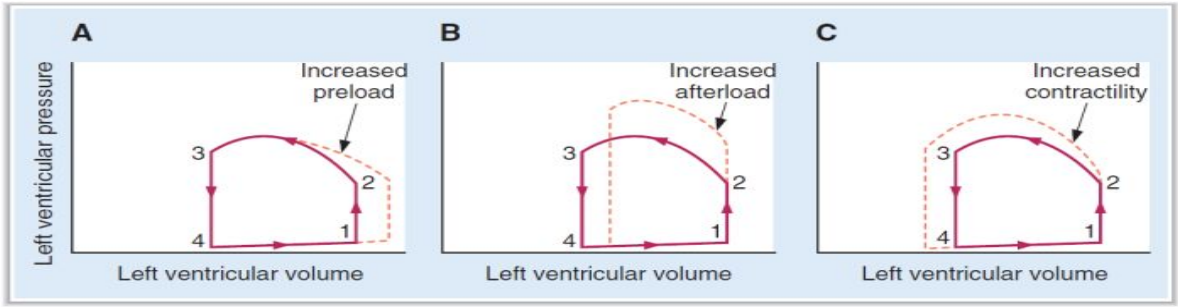
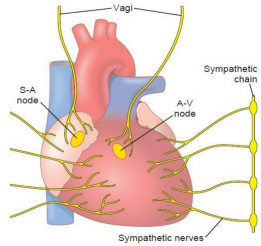


FIGURE 3-10 Effects of changes in **(A)** preload, **(B)** afterload, and **(C)** contractility on the ventricular pressure–volume loop.

Regulation of heart rate: Effect of Sympathetic & Parasympathetic Nerves

ONLY in male slides

Nerve Type	Sympathetic	Parasympathetic
Site of innervation	Innervate the whole heart.	Innervate the: SA node, AV node, the Purkinje system and Atria.
Affection	Increases heart rate and contractility.	Slows the heart but has a little effect on contractility.
Mechanism of action	Release noradrenaline (adrenaline), which stimulates heart β1 - receptors.	Release ACh that stimulates muscarinic (M2) receptors.



The SA node, atria, and AV node have vagal innervation, but the ventricles do not.

ONLY in male slides

Definitions

Inotropic : Affects the force of cardiac contraction

Positive inotropic agents produce an increase in contractility.

Negative inotropic agents produce a decrease in contractility

Chronotropic : Affects the heart rate

Dromotropic : Affects conduction velocity through the conducting tissues of the heart

Inotropic: From Greek in-, meaning fibre or sinew.
Chronotropic: From chrono-, meaning time, and tropos, "a turn".
Dromotropic: From the Greek word "dromos", meaning running, a course, a race.

Parasympathetic effects on heart

Negative inotropic effect:

- ACh via muscarinic receptors decreases the force of contraction in the atria by decreasing the inward Ca2+ current during the plateau of the cardiac action potential.

Negative chronotropic effect:

- Decreases heart rate by decreasing the rate of phase 4 depolarization.
- Fewer action potentials occur per unit time because the threshold potential is reached more slowly .
- The mechanism is decreased If, the inward Na+ current that is responsible for phase 4 depolarization in the SA node.

Negative dromotropic effect:

- Decreases conduction velocity through the AV node.
- Action potentials are conducted more slowly from the atria to the ventricles. increases the PR interval.
- The mechanism is decreased inward Ca2+ current and increased outward K+ current.

Sympathetic effects on heart

Positive chronotropic effect:

- Increases heart rate by increasing the rate of phase 4 depolarization.
- More action potentials occur per unit time because the threshold potential is reached more quickly .
- The mechanism is increased If, the inward Na+ current that is responsible for phase 4 depolarization in the SA node.

Positive dromotropic effect:

- Increases conduction velocity through the AV node.
- Action potentials are conducted more rapidly from the atria to the ventricles, and ventricular filling may be compromised. decreases the PR interval.
- The mechanism of the positive dromotropic effect is increased inward Ca2+ current.

Positive Inotropism effect:

Increases the force of contraction by two mechanisms:

1. It increases the inward Ca2+ current during the plateau of each cardiac action potential.
2. It increases the activity of the Ca2+ pump of the SR (by phosphorylation of phospholamban); as a result, more Ca2+ is accumulated by the SR and thus more Ca2+ is available for release in subsequent beats.

Effect Of Sympathetic and Parasympathetic Stimulation on Prepotential (Pacemaker Potential)

The ß1 -adrenoreceptors on SA node cells cause:

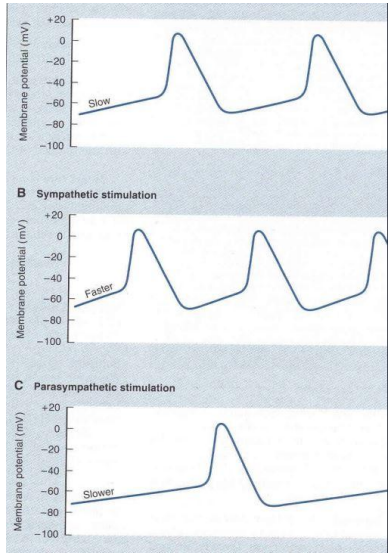
- Opening of Na+ and Ca2+ channels → speed up the rate of depolarization and hence the heart rate.
- Decrease in K+ permeability by accelerating inactivation of the K+ channels. Thus, fewer positive potassium ions leave the cell → the inside of the cell becomes less negative → depolarizing effect.

- **The net effect is:** Swifter drift to threshold → greater frequency of action potential → increase the heart rate.

M2 (muscarinic-2) ACh receptors cause:

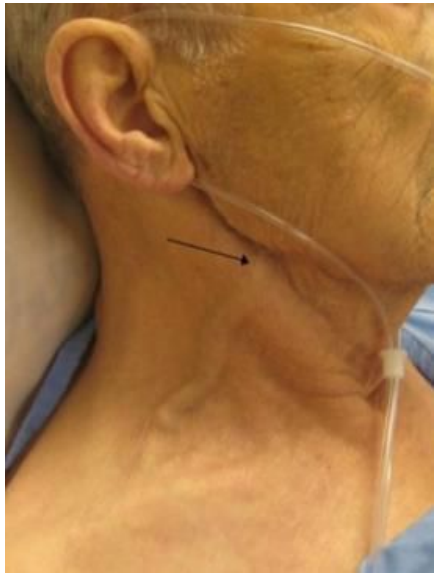
- closure of Na+ and Ca2+ channels.
- opening of potassium channels in the cell membrane → hyperpolarises the cell and makes it more difficult to initiate an action potential.

- **The net effect is:** Slower drift to threshold → lesser frequency of action potential → decrease the heart rate.



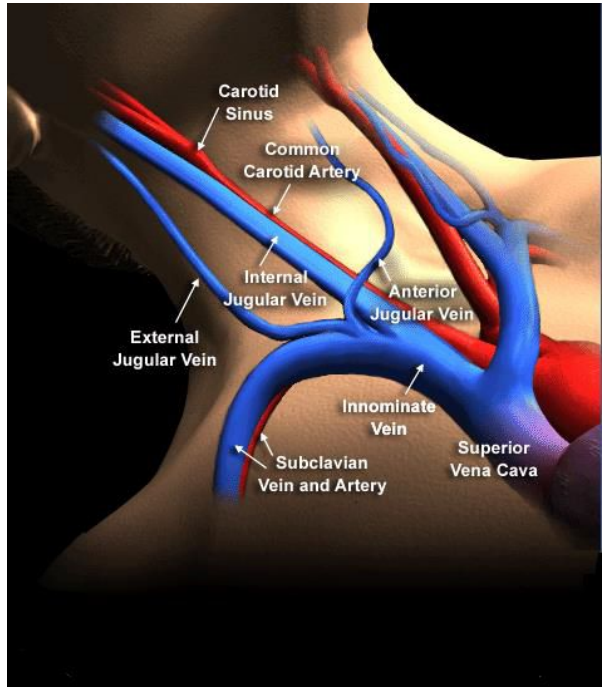
Definition of :

- Jugular Venous Pulse: Defined as the oscillating top of vertical column of blood in right internal jugular vein. It reflects pressure changes in right atrium in cardiac cycle.
- Jugular Venous Pressure: Vertical height of oscillating column of blood.



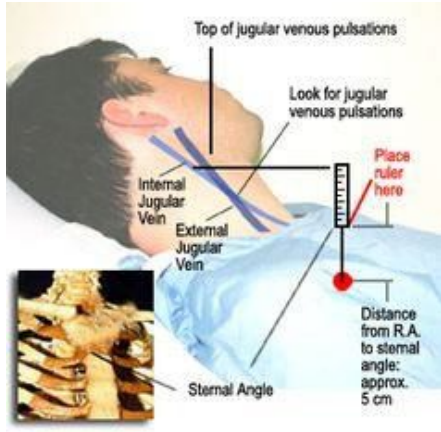
Why Right Internal Jugular Vein (IJV)?

- 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 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.



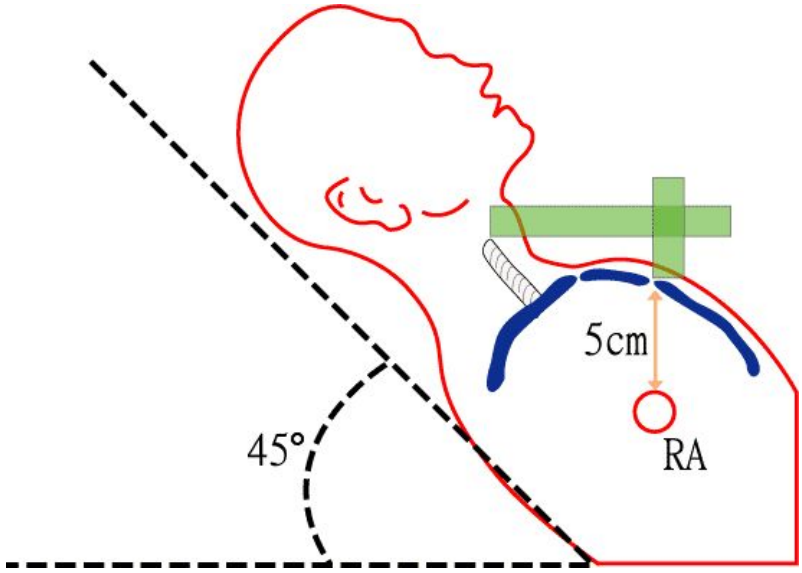
Method Of Examination

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



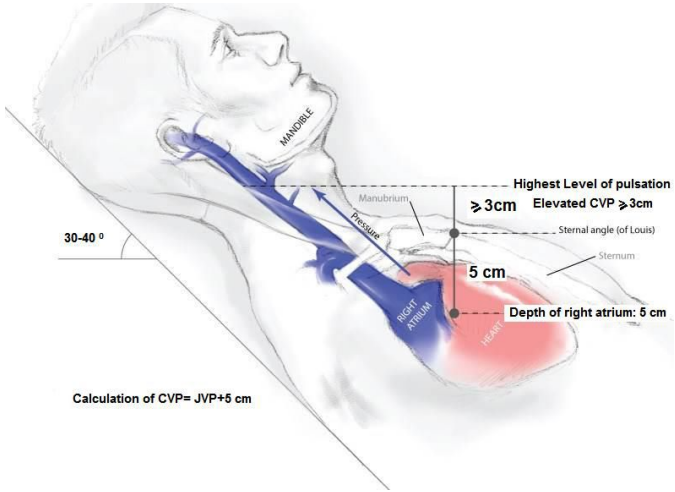
Observations Made

- When the patient reclining with head elevated 45 °, observe:-
- The level of venous pressure.
- The type of venous wave pattern.



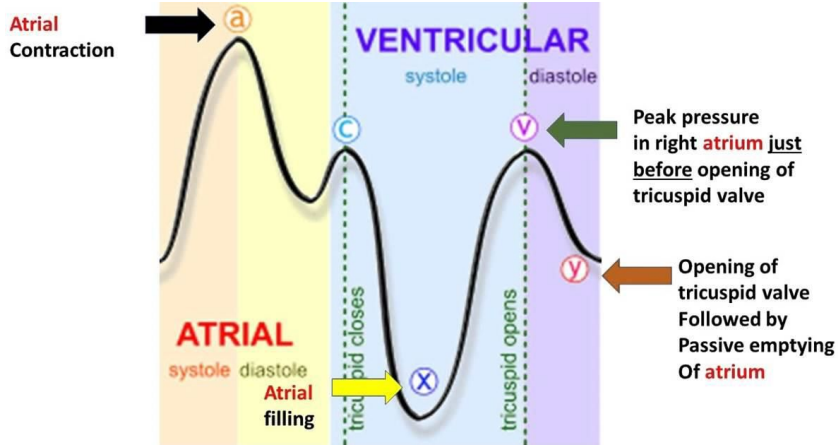
The level of venous pressure

- 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.
- Normal CVP is < 8 cm H2O



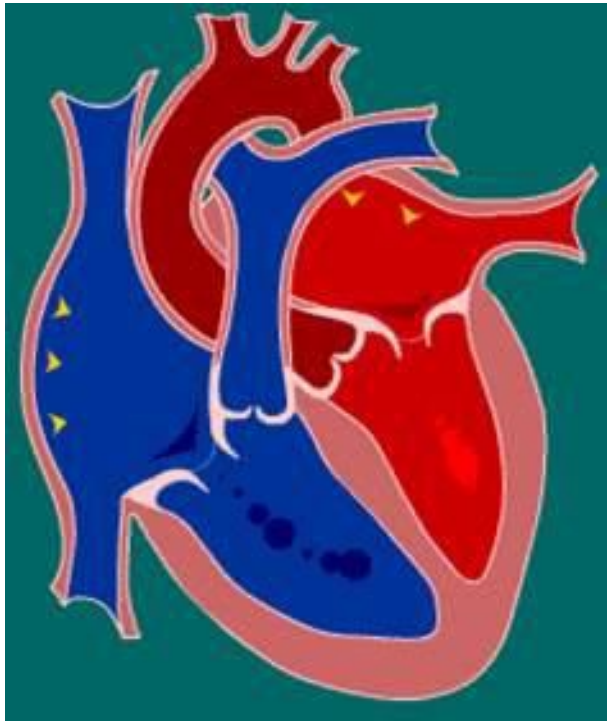
Normal pattern of the jugular venous pulse

- The normal JVP reflects phasic pressure changes in the right atrium and consists of:
- Three positive waves
- Two negative descents.



The “a" Wave: Atrial systole

- +ve, venous distension due to RA contraction and retrograde blood flow into SVC and IJV
- -ve due to blood passage into ventricles.



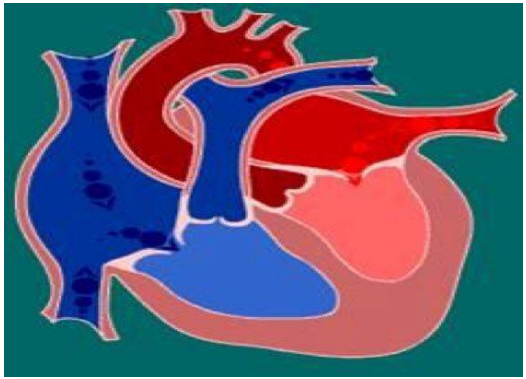
The “x” descent:

- It is due to atrial relaxation and downward displacement of the tricuspid valve during ‘reduced ejection phase.’

The “c” wave: Ventricular systole

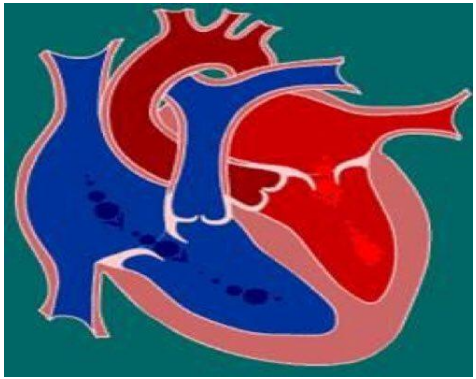
- +ve due to ventricular contraction and resulting bulging of tricuspid valve into the right atrium during isovolumetric contraction.
- -ve due to the pulling down of the atrial muscle & A-V cusps during ‘rapid ejection phase’, resulting in atrial pressure.

“v” Wave:



- +ve due to high venous return using atrial pressure when blood flows into the right atrium during atrial diastole while the tricuspid valve is shunt.
- -ve due to entry of blood into ventricles during ‘rapid filling phase’.

“y” Descent:



- It is due to decline in right atrial pressure due to entry of blood into ventricles when the tricuspid valve reopens during ‘reduced filling phase’.

Abnormalities of Jugular Venous Pulse

- Low jugular venous pressure
  - Hypovolemia.
- 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.
- N.B: 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

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

This is the most common cause of HF

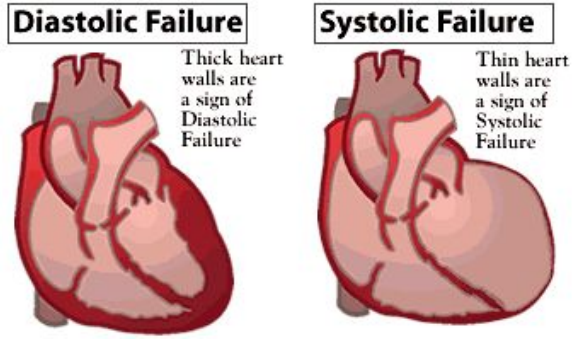
The heart loses its ability to contract or pump blood into the Circulation

In turn, it may not have the muscle power to pump the amount of oxygenated and nutrient-filled blood the body needs into the circulation

Diastolic failure

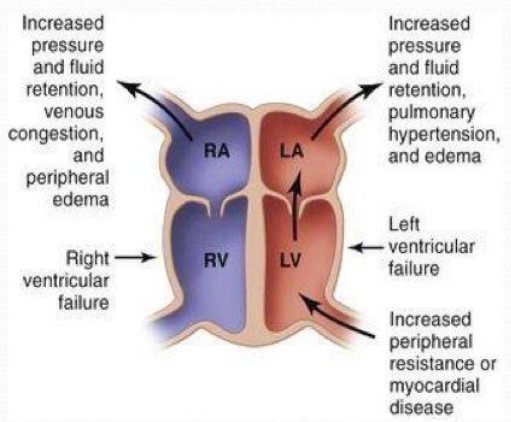
The heart loses its ability to relax because it becomes stiff
Heart cannot fill properly between each beat.

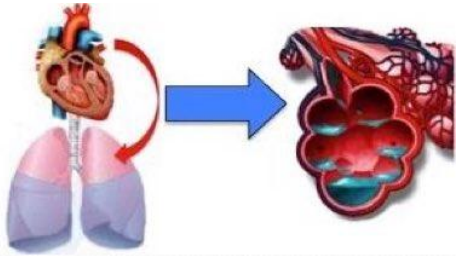
Often the walls of the heart thicken, and the size of the chamber may be normal or reduced.



Types of heart failure

- Left sided heart failure
  - Inadequate output of LV causing decreased CO to body and back pressure to the lungs. The left side of the heart is usually where heart failure begins.
- Right sided heart failure
  - Inadequate output of RV causing decreased CO to lungs and back pressure to venous system. It may occur alone but is usually a result of left- sided failure.
- Congestive heart failure
  - Chronically, left HF results in secondary pulmonary hypertension and right HF.





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

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

Causes of left Sided HF

1. Systolic Dysfunction

Impaired Contractility

- Myocardial infarction
- Transient ischemia
- Chronic volume overload
- Mitral/Aortic Regurgitation

Increased Afterload

- Atrial Stenosis
- Uncontrolled HTN

2. Diastolic Dysfunction

Obstruction of LV filling

- Mitral Stenosis
- Pericardial constriction or tamponade

Impaired ventricular relaxation

- Hypertrophic or restrictive cardiomyopathy
- 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

Cardiac Causes

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

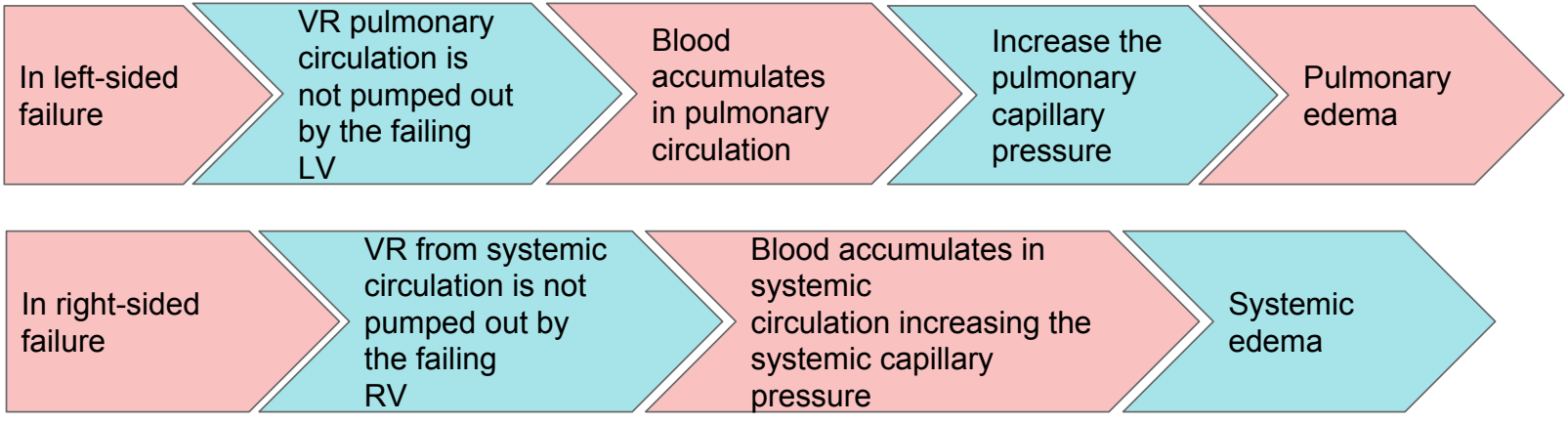
Pulmonary Vascular Disease

- Pulmonary embolism
- Pulmonary HTN Right ventricular infarction

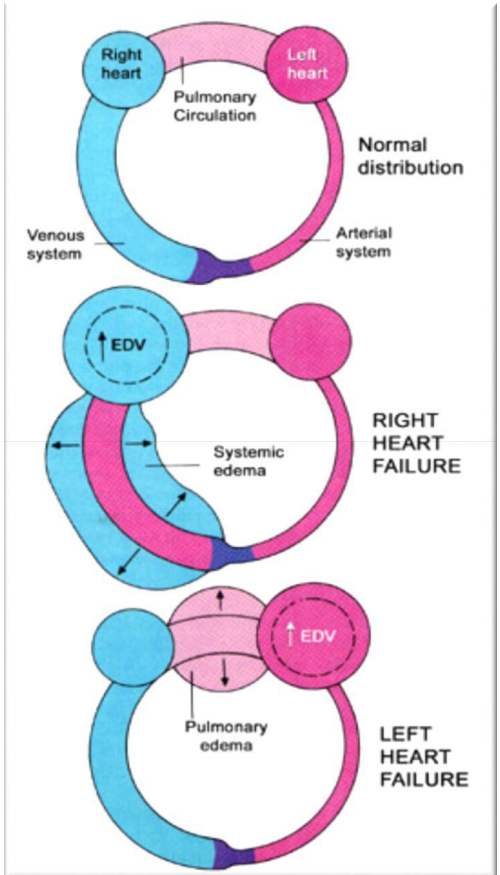
Pulmonary Parenchymal disease

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

Left vs right heart failure



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


Right heart
Left heart
Pulmonary Circulation
Normal distribution
Venous system
Arterial system
EDV
RIGHT HEART FAILURE
Systemic edema
EDV
Pulmonary edema
LEFT HEART FAILURE

Acute vs chronic heart failure

- Acute :
  - 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 :
  - 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

compensation:The body tries to correct

↓ Decrease	↑Increase	↑Increase	↑ **Increase**	↑Increase	↓ Decrease	& ↑ increase
cardiac output lead to retention of extra salt and water by kidney	blood volume	EDV	**stroke volume**	Heart Contractility	Firing of carotid baroreceptors	Sympathetic firing

Physiological adaptation to CHF compensatory mechanisms

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

2-Decreased renal perfusion → activation of RAA system((renin–angiotensin–aldosterone system))

- ANP((Atrial natriuretic peptide)) and BNP((Brain natriuretic peptide)) are major antagonizing agents of the renin–angiotensin–aldosterone system.

3-Decreased effective circulating blood volume → posterior pituitary releases ADH (vasopressin) → increased H2O reabsorption.

Complications of progressive heart failure Factors contributing to decompensation

Decompensation : failure of compensation

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.

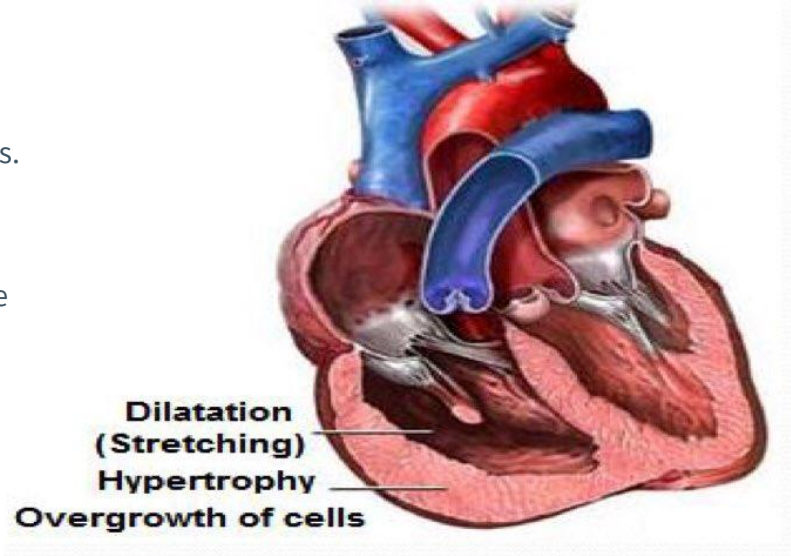
3) Hypertrophied heart: → imbalance between the O2 supply and need → deteriora:on of the ability to generate force.

4) Excessive salt and water retention:

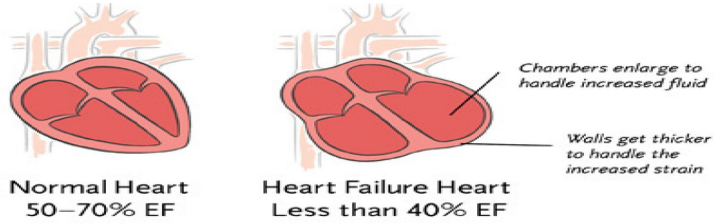
5) Over-distended ventricle: Has to consume more energy and generate more wall tension to develop the required ejection pressure (Laplace law).

Can a person have RVF without LVF? (COR PULMONALE)

- Cor pulmonale, or right-sided HF, is an enlargement of the right ventricle.
- It is due to high blood pressure in the lungs.
- It is usually caused by chronic lung disease



Indicator for Diagnosing Heart Failure



Ejection Fraction (EF)

- is the percentage of blood that is pumped out of your heart during each beat.

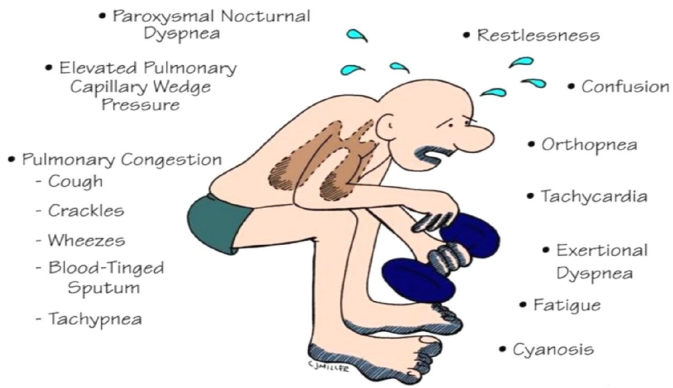
Fractional Shortening

- 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)
  - . Biomarkers

Clinical picture of left-sided failure

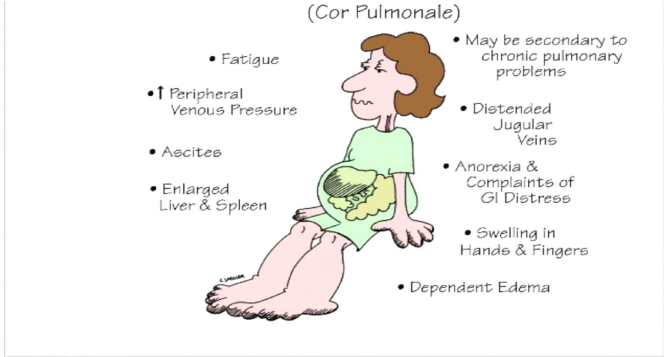


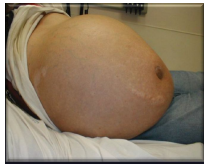
Orthopnoea is shortness of breath (dyspnea) that occurs when lying flat, causing the person to

have to sleep propped up in bed or sitting in a chair.

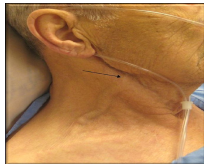
Paroxysmal nocturnal dyspnea refers to attacks of severe shortness of breath and coughing that generally occur at night. It usually awakens the person from sleep, and may be quite frightening.

Clinical picture of Right-sided failure

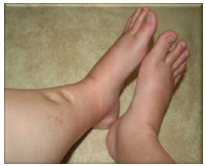




Ascites

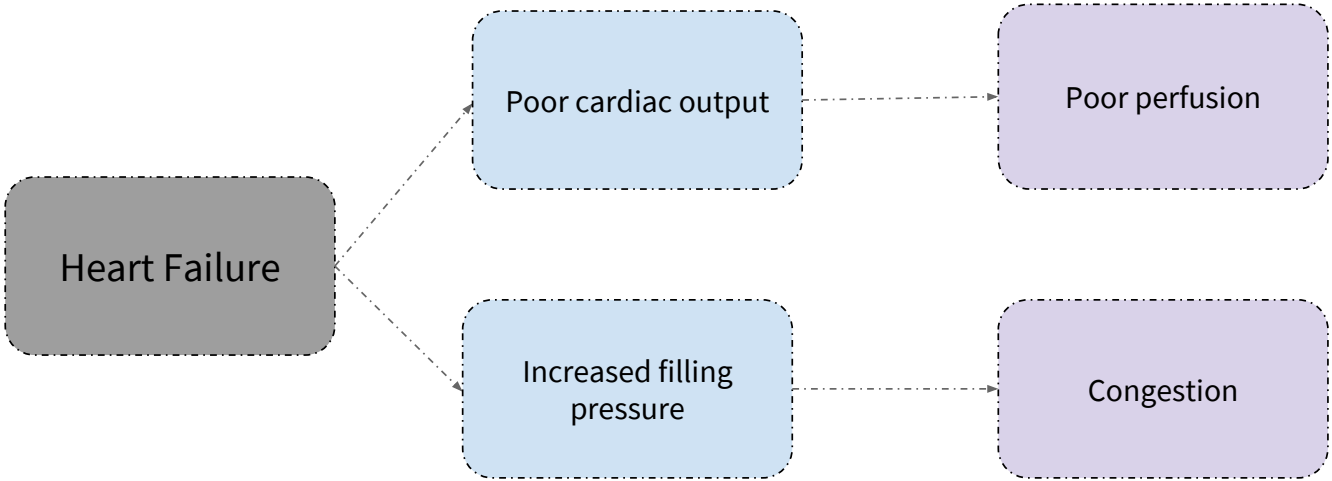


Elevated JVP



Pitting edema

Symptoms and Signs of HF



Signs and symptoms of congestive heart failure

- Symptoms:
  - Shortness of breath.
  - Leg swelling (edema).
  - Orthopnea.
  - Fatigue.

Explanation of some signs and symptoms of HF

“Persistent cough or wheezing”

- Why?
  - Fluid “backs up” in the lungs.
- Symptoms:
  - Coughing that produces white of pink blood-tinged sputum.

ONLY in female slides

Symptoms explanation

Tiredness, fatigue

WHY?

Heart can't pump enough blood to meet needs of bodies tissues Body diverts blood away from less vital organs (muscles in limbs) and sends it to the heart and brain

SYMPTOMS Constant tired feeling Difficulty with everyday activities

Lack of appetite/ Nausea

WHY?

The digestive system receives less blood causing problems with digestion

SYMPTOMS Feeling of being sick or full stomach

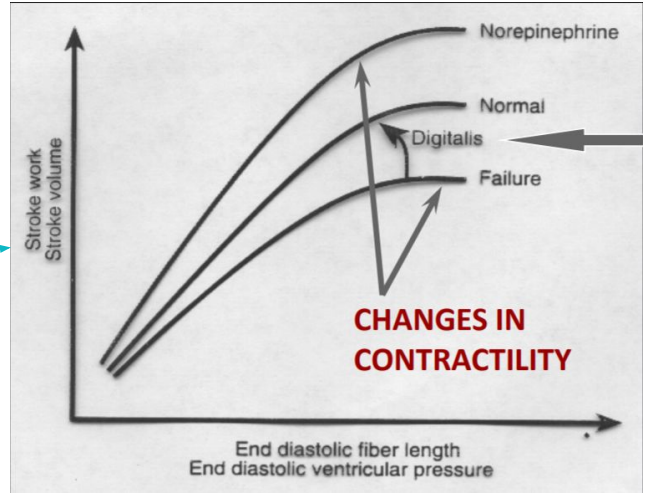
Clinical picture	Left-sided failure	Right-sided failure
Pitting edema (legs, hands)	Mild to moderate	Moderate to sever
Fluid retention	Pulmonary edema (fluid in lungs), and pleural effusion (fluid in the pleural cavity)	Abdomen (ascites)
Organ enlargement	Heart	Liver. Mild jaundice may be present
Neck 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

ONLY in male slides

Treatment of cardiac failure

If we want to reduce salt & water retention, we use Diuretics.

If we want to increase the pumping activity (contractility) of failing heart, we give Digitalis, since it improves contractility of heart by increasing cytosolic Ca++



As we can see, the increase in contractility solves the patient's problem. But if the problem is not in contractility, it will worsen the case.

ONLY in male slides

Systolic dysfunction

Dr.shahid:these curves are not coming in the exam except the systolic and diastolic curves. So focus on them

impaired ventricular contraction

↓ slope of ESPVR i.e. ↑ ESV

Compensatory rise in preload i.e. ↑ EDV

↓ SV ↓ EF ↓ Work ↑ EDP

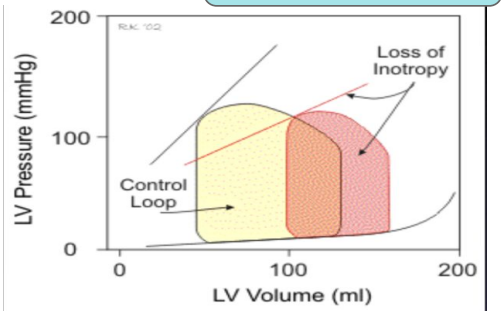
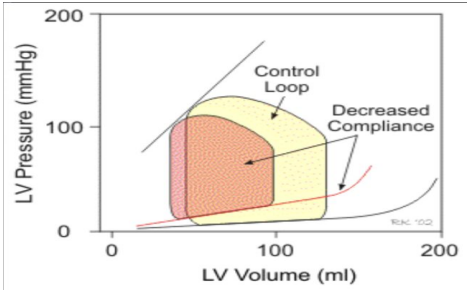


Figure 2. Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

Diastolic dysfunction

Reduced venous return / compliance / relaxation (lusitropy)

↓ EDV ↓SV ↓or=EF ↓ Work ↑ EDP



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.

ONLY in male slides

FORCE-VELOCITY RELATIONSHIP

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

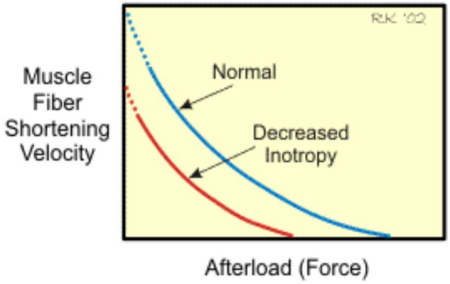
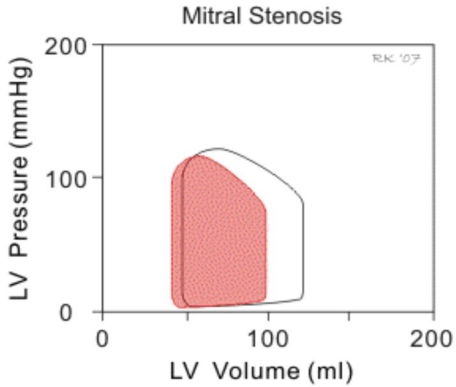


Figure 3. Effects of ventricular failure (decreased inotropy) on the force-velocity relationship. Decreased inotropy decreases velocity of fiber shortening at any given afterload.

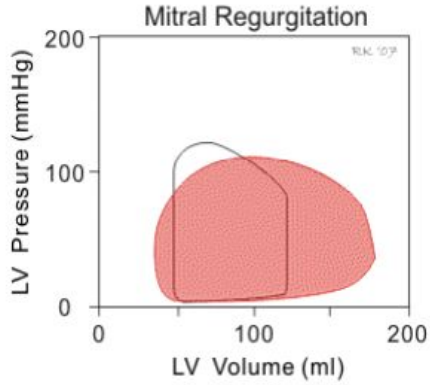
MITRAL STENOSIS

Impaired LV filling
↓ EDV
↓ afterload ;
↓ ESV
↓ SV and CO

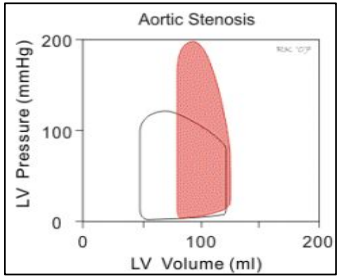
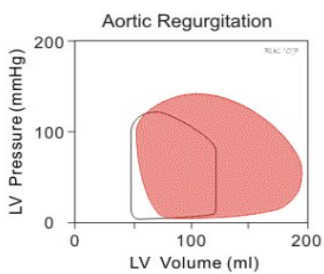


MITRAL REGURGITATION

↓ Afterload on LV
↓ Outflow resistance
↑ EDV and EDP
↑ SV
↓ EF



ONLY in male slides

AORTIC STENOSIS	AORTIC REGURGITATION
High outflow resistance; LV emptying impaired	**No true isovolumetric relaxation**
↑ Peak systolic pressure; ↑ afterload	**Blood from aorta to ventricle throughout diastole**
↓ SV	**↑ SV (if no failure) , ↓ SV in failure**
↑ ESV	**↑ ESV**
↑ EDV	**↑ EDV**
	

Summary

Cardiac Output	Volume of blood ejected by each ventricle in each minute. (5L)	CO = HR X SV
Stroke Volume	Volume of blood ejected by each ventricle per each beat. (70 ml)	SV = EDV - ESV
Cardiac Index	Cardiac Output per Square Meter of Body Surface Area.	CI = CO/m^2
Venous Return	The Quantity Of Blood Flowing from the Veins into Right Atrium each Minute.	CO = VR
Preload	The amount of blood presented to the Ventricles.	
Afterload	The resistance against which the ventricles contract.	
Cardiac Reserve	The difference between the resting CO and the maximum volume of blood the heart is capable of pumping per minute.	

Summary

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 substrates despite the venous return to heart is either normal or increased

Types

- Left Sided Heart Failure
- Right Sided Heart Failure
- Congestive Heart Failure

Causes

- Impaired Cardiac Function
- Increased Cardiac Workload
- Acute Non-cardiac Conditions

Signs/Symptoms

- Poor Cardiac Output (Poor Perfusion)
- Increased Filling Pressure (Congestion)

Diagnosis

- Medical History
- Physical Exam
- Tests:
  - Chest X-ray
  - ECG
  - Echocardiogram
  - Biomarkers

Quiz

1-When Ach binds to Muscarinic receptor(M2) in the heart, it will……..

A-increase the force of contraction.

B-Decrease the force of contraction.

C-Decrease Heart rate

D- Both B&C

Answer:D

2-increasing the afterload will shift "starling curve" toward:

A-up and right

B- up and left

C-Down and right

D- down and left

Answer:c

3- According to the Frank-Starling mechanism of the heart:

A- the left ventricle ejects a larger volume of blood with each systole than the right ventricle.

B- the intrinsic rate of the heart's pacemaker is 100 beats/min.

C-cardiac output increases with increased heart rate.

D-stroke volume increases with increased venous return

Answer:D

4- A 65-years-old man, suffering from dyspnea, anxiety and he has a high blood pressure and heart rate. Also, he has Cheyne strokes breathing with cyanosis?

A- Left ventricular failure

B- Right ventricular failure

C- Cor pulmonale

D-Congestive heart failure

Answer:A

5-Elevated jugular venous pulse is prominently seen in

A-Left sided heart failure.

B- Right sided heart failure.

C-None.

Answer:B

Thank you for checking our work

Team Leader:

العنود سلمان

Male Team:

أنس السويداء
أنس السيف
خالد شويل
ريان الموسى
سعد الهداب
سعود العطوي
سيف المشاري
عبدالجبار اليماني
عبدالرحمن آل دحيم
عمر الفوزان
فهد الحسين
نايف المطيري
نواف اللويمي
محمد الحسن
هشام الشايع
خالد العقيلي
سعد الفوزان
عبدالله الزيد
نواف اللويمي
عبدالمجيد الوردي
يزيد الدوسري

Female Team:

الآء الصويّغ
رناد المقرن
رهف الشنيبر
روان التميمي
روان مشعل
ريم القرني
ليلى الصباغ
فلوة السعوي
نورة بن حسن
نورة الحربي
نورة العثيم
مجد البراك
لينا العوهلي
عهد القرين
مها النهدي
مها بركة
سارة الفليج
هند العريعر
ريناد الغريبي
عائشة الصباغ
ميعاد النفيعي
سمية العقيفي
سارة البليهد

Any questions?

Contact us at

- twitter:@physio437
- physiologyteam437@gmail.com