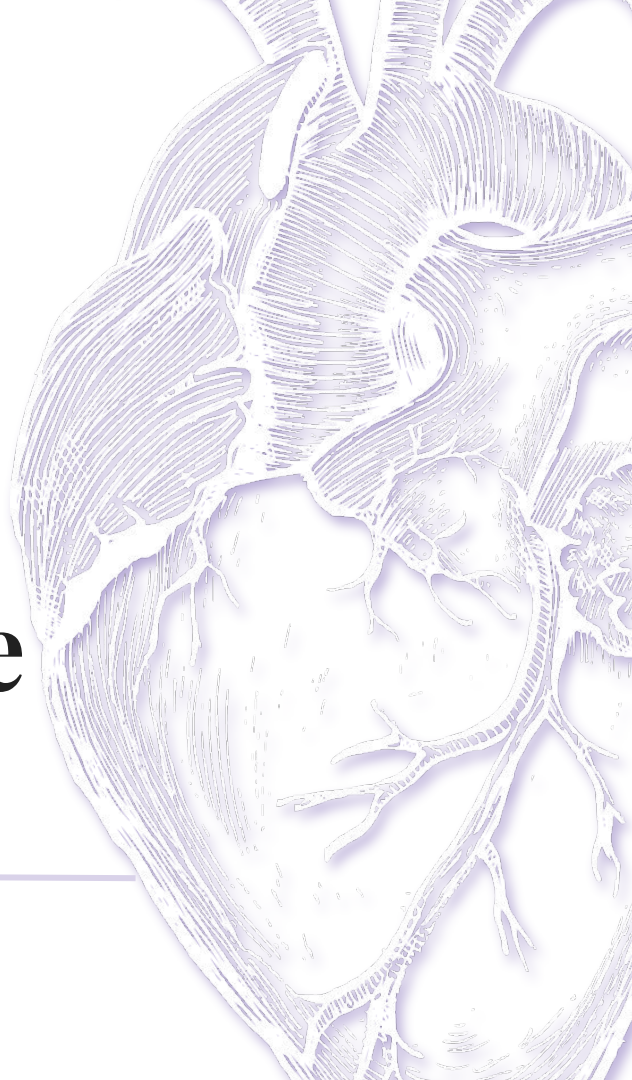
MAWEEN ALRUWAIDY . JOOD ALJUFAN . KHALID ALQHTALI . MESHARI ALHARBI

Physiology team

MED444
KING SAUD UNIVERSITY



Jugular venous pulse & heart failure

Editing File

Main text Female's slide Male's slide Important text Doctor's note Extra

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 & classify types of HF
- 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.

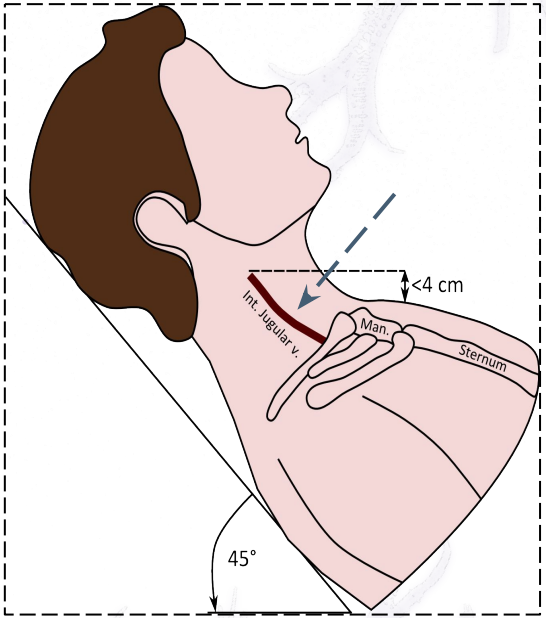
Jugular Venous Pulse and pressure definitions

1

Jugular Venous Pulse:is the oscillating top of vertical column of blood in right internal jugular vein.

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

The pulse in the jugular vein is a reflection of the heartbeat. When the heart beats, it creates a pressure wave that travels through the arteries and into the veins. This pressure wave causes the jugular vein to expand and contract, producing a pulse that can be felt or seen.
(other veins normally do not have a pulse)

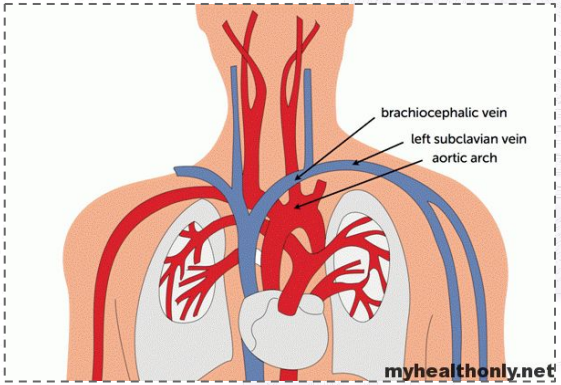


2

Jugular Venous Pressure: Vertical height of oscillating column of blood in right internal jugular vein.

Why Right Internal Jugular Vein (IJV)?

- Pressure changes in RA can be recorded from IJV
- 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.
- IJV is anatomically closer to RA.
- IJV has no valves (EJV has valves and these valves prevent transmission of RA pressure that's why it can't be relied also obstructed by facial & muscular layers through which it passes)
- The left innominate vein (innominate vein = brachiocephalic 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 °
   Then observations are made by:
   - The level of the Venous pressure
   - The type of the Venous wave pattern
4. Neck should not be sharply **flexed**,head is turned slightly to the left.
5. Examined effectively by shining a **light** across the neck
6. There should not be any tight bands around abdomen.

Female's slide

The level of venous pressure

JVP= jugular venou pressure

CVP= Central venous pressure

JVP

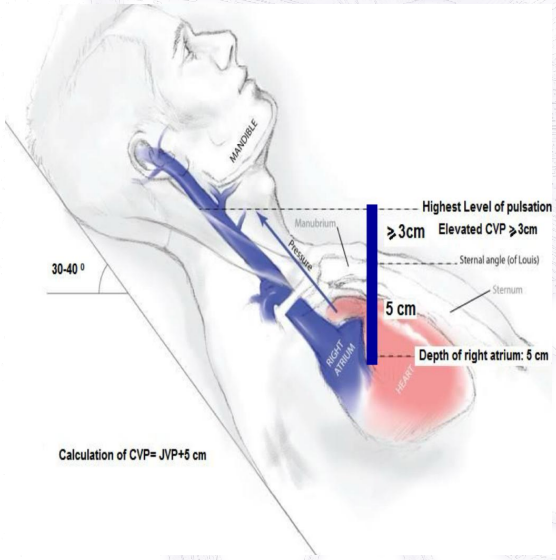
Using a centimeter ruler, measure the vertical distance between the angle of Louis (sternal angle) and the highest level of jugular vein pulsation.

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

CVP

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

Normal CVP is < 8 cm H_2O

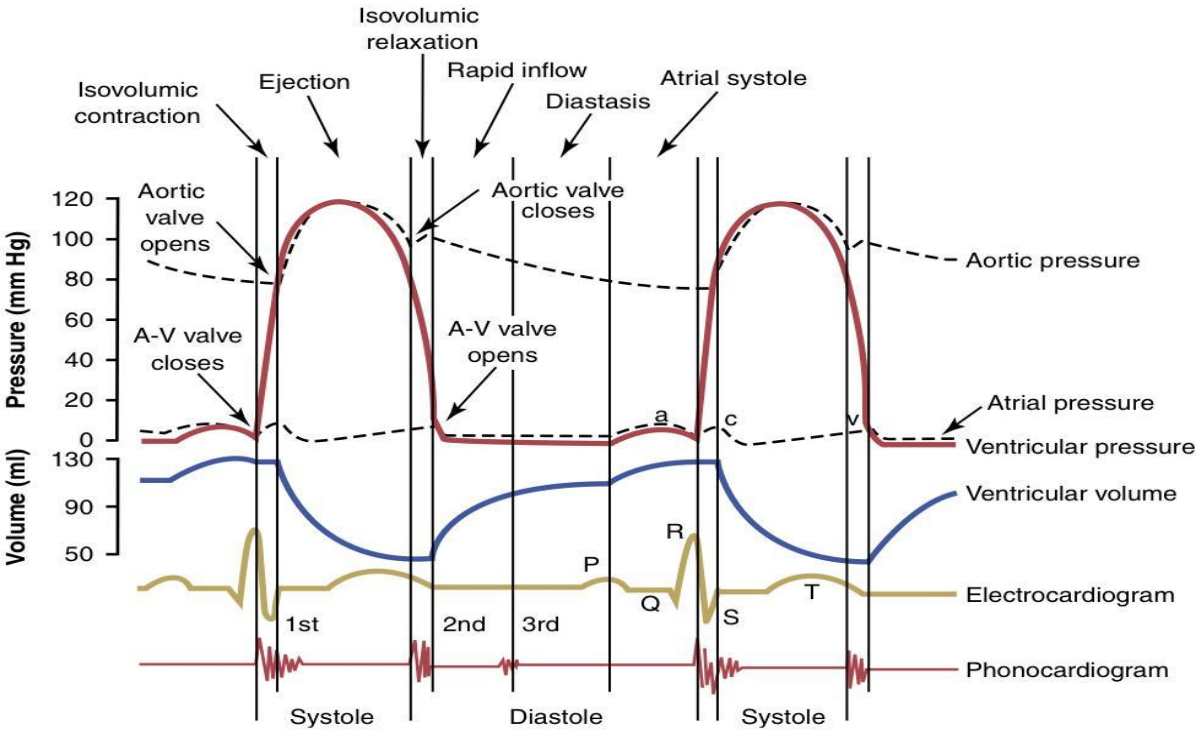


Male's slide

JVP ↑ in:

1. Rt. Sided heart failure
2. Fluid overload.

Atrial Pressure Changes During Cardiac Cycle



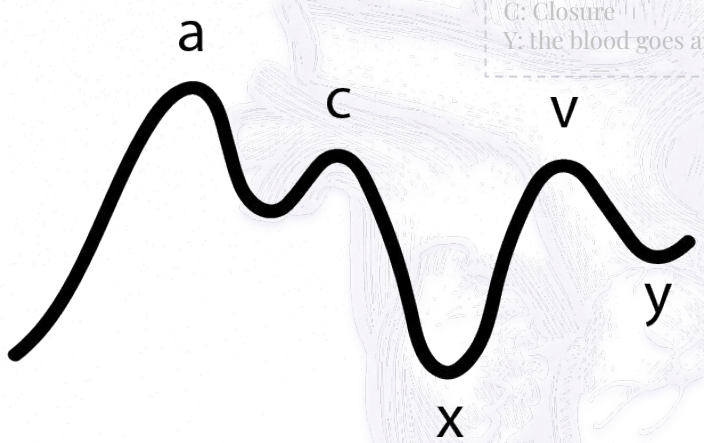
JVP is the "Atrial pressure"

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

Video for JVP: Waveforms & Their Abnormalities From female's doctor

A: Atrial contraction
X: relaX
V: Volume
C: Closure
Y: the blood goes awaY

"a" wave	Atrial contraction (pressure raising)
"c" wave	Pressure rise and valve closes and ventricle contracts
"x" descent	decrease in atrial pressure (JV) due to atrial filling / relaxation
"v" wave	peak pressure in RA just before opening of tricuspid valve. volume of blood increase due to venous return the atria start raising in pressure
"y" wave	opening of tricuspid valve followed by passive emptying of atrium. Pressure drop because blood volume is decreasing in atria (JV)



The normal JVP reflects phasic pressure changes in right atrial pressure (RAP) and consists of:

Three positive/upward waves (a, c, & v waves) = +ve (↑atria pressure)

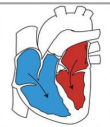
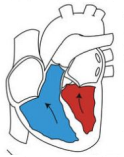
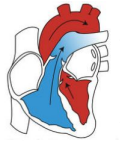
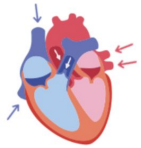
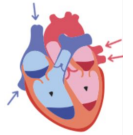
Two negative/downward deflection descents or waves (x&y waves) = -ve (↓atrial pressure).

These 3 waves (a , c ,v) are equal to: ONE cardiac cycle = 0.8 sec

JVP WAVES:

Helpful video

Important slide

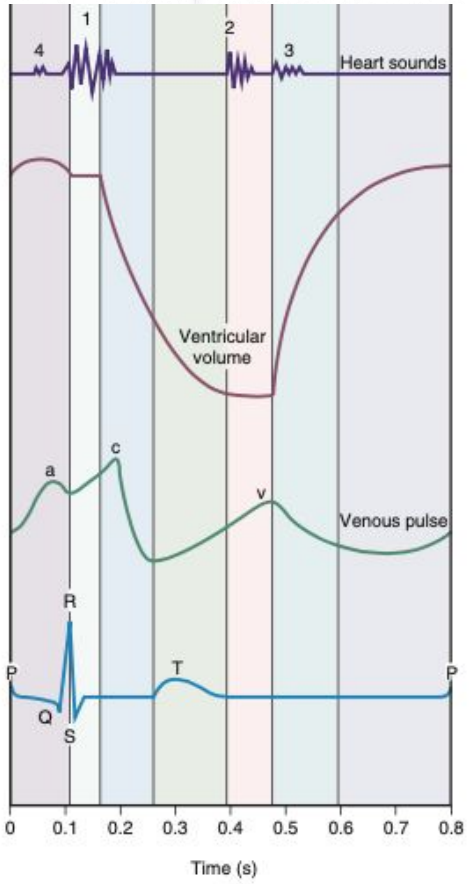
'a' wave **Atrial systole**	It represents the increase in right atrial pressure secondary to/during right atrial contraction occurring at the end of ventricular diastole (atrium systole)	
'c' wave **ventricular systole**	+ve: It represents the increase in right atrial pressure as a result of bulging of AV/tricuspid valve into the atria (right atrium) during 'isovolumetric ventricular contraction phase'. Or due to transmitted carotid pulsations. -ve as a result of pulling of the atrial muscle & AV cusps down during 'rapid ejection phase', resulting in ↓ atrial pressure	
'x' descent ventricular systole	It represents the decrease in right atrial pressure due to atrial relaxation and downward displacement of AV/tricuspid valve secondary to contraction of papillary muscles during ventricular systole (ejection phase).	
'v' wave **atrial diastole**	+ve: Atrial pressure ↑ gradually due to continuous VR It represents the increase in right atrial pressure gradually as it fills with blood returning from the great veins against a closed tricuspid valve during atrial diastole (continuous VR) -ve: as a result of ↓ atrial pressure during 'rapid filling phase' " semilunar valves are closed during this wave"	
'y' descent ventricular diastole	It represents the fall in right atrial pressure as blood flows out of the right atrium into the right ventricle upon opening of the tricuspid valve during the (reduced filling phase)	

Correlation of JVP with ECG & Heart Sounds

Female's slide

Important slide

- The 'P' wave in the ECG occurs just before the 'a' wave of JVP.
  Electrical activity always comes before mechanical activity
- The 'R' peak of the ECG happens between the peak of the 'a' wave and the peak of the 'c' wave of JVP. or before the 'c' wave
- S1 occurs at the beginning of the 'c' wave
- S2 occurs at the beginning of the 'v' wave
- The 'T' wave of the ECG occurs before the 'v' wave of JVP



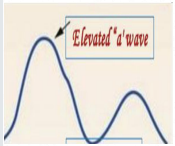
Abnormalities in Jugular Venous Pulse

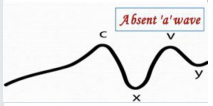
Raised jugular venous pressure	Low jugular venous pressure
Increased right ventricular filling pressure e.g in heart failure, fluid overload (hypervolemia).	Hypovolemia (anything causing ↓ VR).
Obstruction of blood flow from the right atrium to the right ventricle e.g tricuspid stenosis.	JVP usually drops on inspiration along with intrathoracic pressure (↓ intrathoracic pressure ↠ ↓ JVP), normal condition.
Superior vena caval obstruction e.g retrosternal thyroid goiter (enlargement of thyroid gland)	
Positive intrathoracic pressure e.g pleural effusion, pneumothorax.	

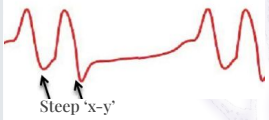
Important slide

Waves abnormalities

‘a’ wave	Elevated, prominent giant	- Signifies increased RA pressure - uniform, every beat	- Right atrial / Ventricle hypertrophy (↑ resist of filling) - Tricuspid stenosis - Decreased ventricular compliance (right ventricular failure, pulmonary valve stenosis or pulmonary hypertension)
	Absent	- The right atrial pressure does not increase due to failure of proper contraction	- Atrial fibrillation or atrial flutter, or Tricuspid regurgitation (TR)
	Cannon	–	- Discussed in next slide
‘v’ wave	Large	–	- Atrial septal defect, Tricuspid regurgitation, Constrictive pericarditis
	Diminished	–	- Hypovolaemia
‘C’ wave	absent/prominent	prominent in TR	- absent In Constrictive pericarditis (stiffer and thicker pericardium)
‘x-y’ descends	Steep (Friedreich’s sign)	–	- Constrictive pericarditis as it reduces elasticity of pericardial sac, raises arterial pressure and limiting ventricular filling in early diastole
’y’ descend	Attenuated	–	- Cardiac tamponade as it impedes ventricular filling







Important slide

Waves abnormalities (cannon ‘a’ wave)

cannon ‘a’ wave

Definition

- It occurs when the right atrial pressure becomes very high usually secondary to right atrial contraction against a closed tricuspid valve

- intermittent, various height

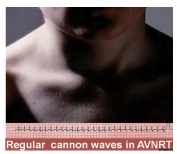
Types

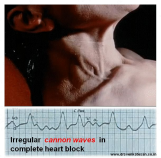
1. Regular:

occurs when atria and ventricular contractions occur nearly simultaneously (as in atrioventricular nodal reentrant tachycardia).

2. Irregular:

occur in complete heart block





Seen in

- Atrial-ventricular asynchrony (atria contract against a closed tricuspid valve)

- Complete (AV) heart block (irregular)

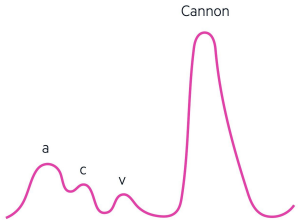
- premature ventricular contraction (asynchrony)

- Ventricular tachycardia(regular)

- Ventricular pacemaker

- atrial flutter

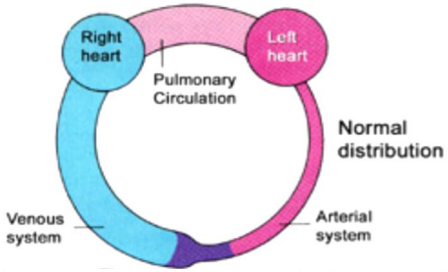
Pic



Heart failure (HF)

Highly recommend video

1. 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.



2. How Fast Does Heart Failure Develop?

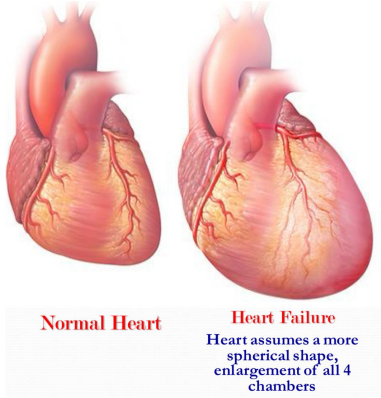
1. Usually a chronic disease so usually happens over time (chronic) but may happen all of a sudden (acute)"442".

2. The heart tries to compensate for the loss in pumping function by:
- Developing more muscle mass
- Enlarging
- Pumping faster

3. - Heart failure can involve 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.

- Usually the left side is affected first.



Male's slide

Heart failure causes

Impaired cardiac function	Increased cardiac workload	Acute non-cardiac conditions
Coronary heart disease	Hypertension	Volume overload
Cardiomyopathies (muscle disease)	Valvular disorders	Hyperthyroidism
Rheumatic fever	Anemias	Fever
Endocarditis	Congenital heart defects	Infection
Cardiac arrhythmias e.g., complete heart block	—	—

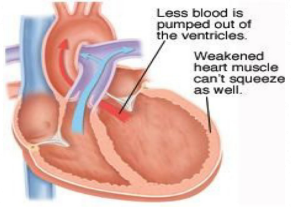
HF is Manifested mainly by:

1. Inadequate cardiac output.

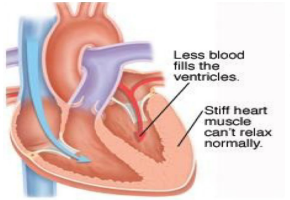
2. Build-up of blood in veins behind left heart or right heart (increased venous pressure).

Types of Heart Dysfunction that Leads To HF

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 the body needs into the circulation. N.B ejection fraction is lower than normal. <40%
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. N.B Ejection fraction is often in normal range.



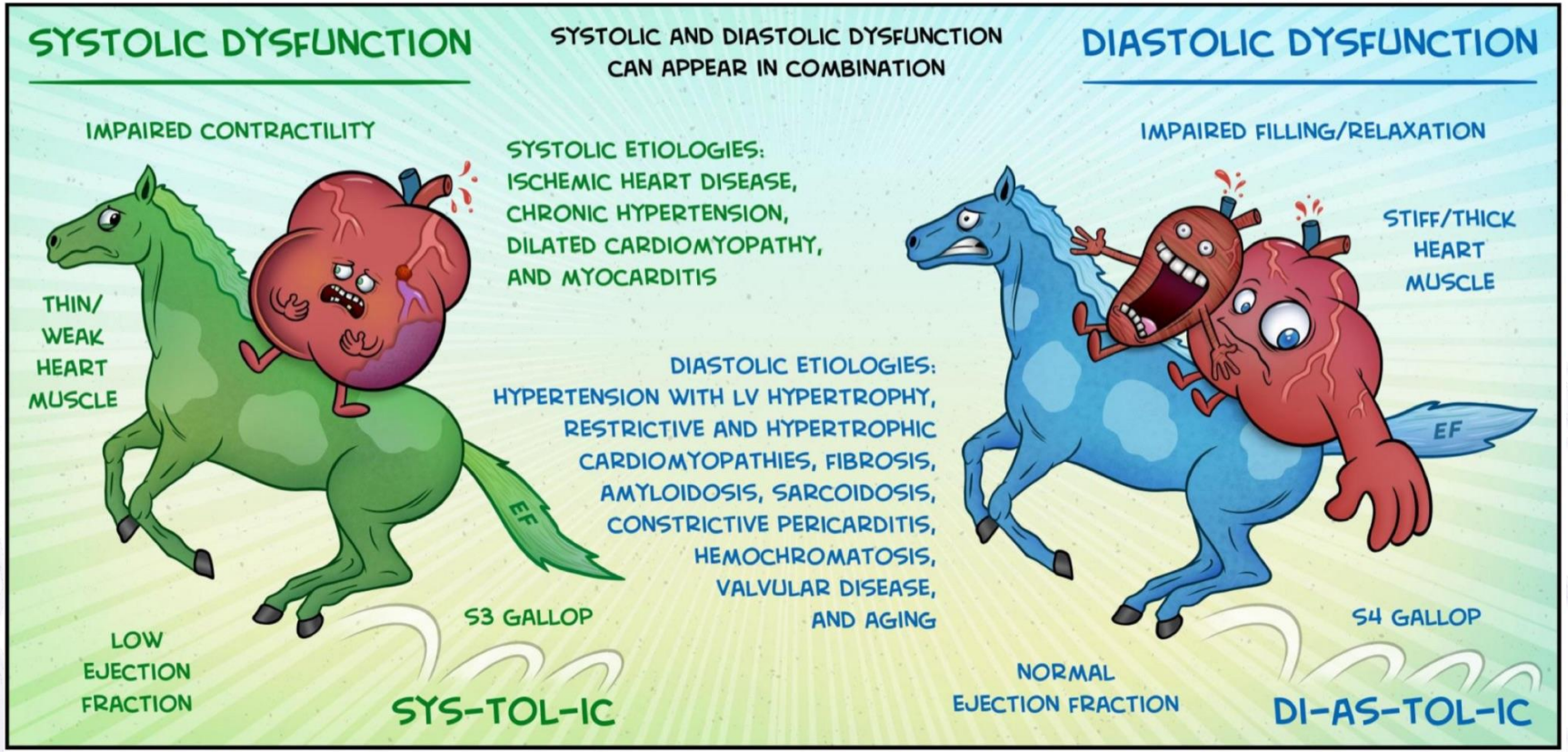
Systolic heart failure. The heart muscle becomes weak and enlarged. It can't pump enough blood forward when the ventricles contract. Ejection fraction is lower than normal.



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.

Female's slide

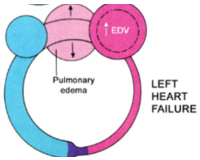
Types of Heart Dysfunction that Leads To HF



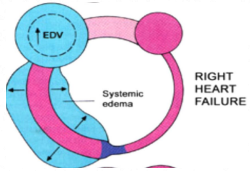
Types of Heart Dysfunction that Leads To HF

Characteristics	Diastolic heart failure	Systolic heart failure
Age	Frequently elderly	All ages, typically 50–70 yr
Sex	Frequently female	More often male
Left ventricular EF	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 radiology	Congestion with or without cardiomegaly	Congestion and cardiomegaly
Gallop rhythm present	Fourth heart sound	Third heart sound

Types of HF



Types



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 sided 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
-if someone have pulmonary embolism the first side will be affected is right side HF

Causes of left Sided HF

Systolic Dysfunction		Diastolic Dysfunction	
Impaired Contractility	**Increased Afterload**	**Obstruction of LV filling**	**Impaired ventricular relaxation**
Myocardial infarction	Aortic Stenosis	mitral stenosis	Hypertrophic or restrictive cardiomyopathy
Transient ischemia (↛fibrous tissue ↛can't relax or contract)	Uncontrolled HTN (prolonged hypertension)	Pericardial constriction or tamponade (Heart can't expand)	Transient ischemia
Chronic volume overload (hypervolemia)	-	-	-
MR/AR (mitral/aortic regurgitation)	-	-	-

- 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	Pulmonary Vascular Disease	Pulmonary Parenchymal Disease
Usually occurs as a result of left HF	Pulmonary embolism	COPD
Pulmonary stenosis	Pulmonary HTN	Interstitial lung disease
Right ventricular infarction	—	Chronic infections
—	—	Adult respiratory distress syndrome

Combination of both right and left HF

Congestive heart failure :

Chronic left HF results in:

Secondary pulmonary hypertension

↓

Pulmonary edema

↓

Right HF ↠ ↑ the systemic capillary pressure ↠ systemic edema

COR PULMONALE: Right HF due to chronic lung disease

Acute vs Chronic Heart Failure

Acute heart failure

1. Acute HF is a short-term condition (hour/days) due to sudden serious abnormalities of the heart (e.g., massive infarction, severe arrhythmias, valve rupture; sepsis) [usually left-sided] ↠ acute heart failure

2. Can be life threatening because the heart does not have time to undergo compensatory adaptations.

3. Cardiogenic shock develops following acute failure if the heart became unable to pump enough to even keep tissues alive.

Chronic heart failure

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

2. It is associated with adaptive responses in the heart, (Dilation , hypertrophy) which can be deleterious.

Compensatory Mechanism in chronic HF

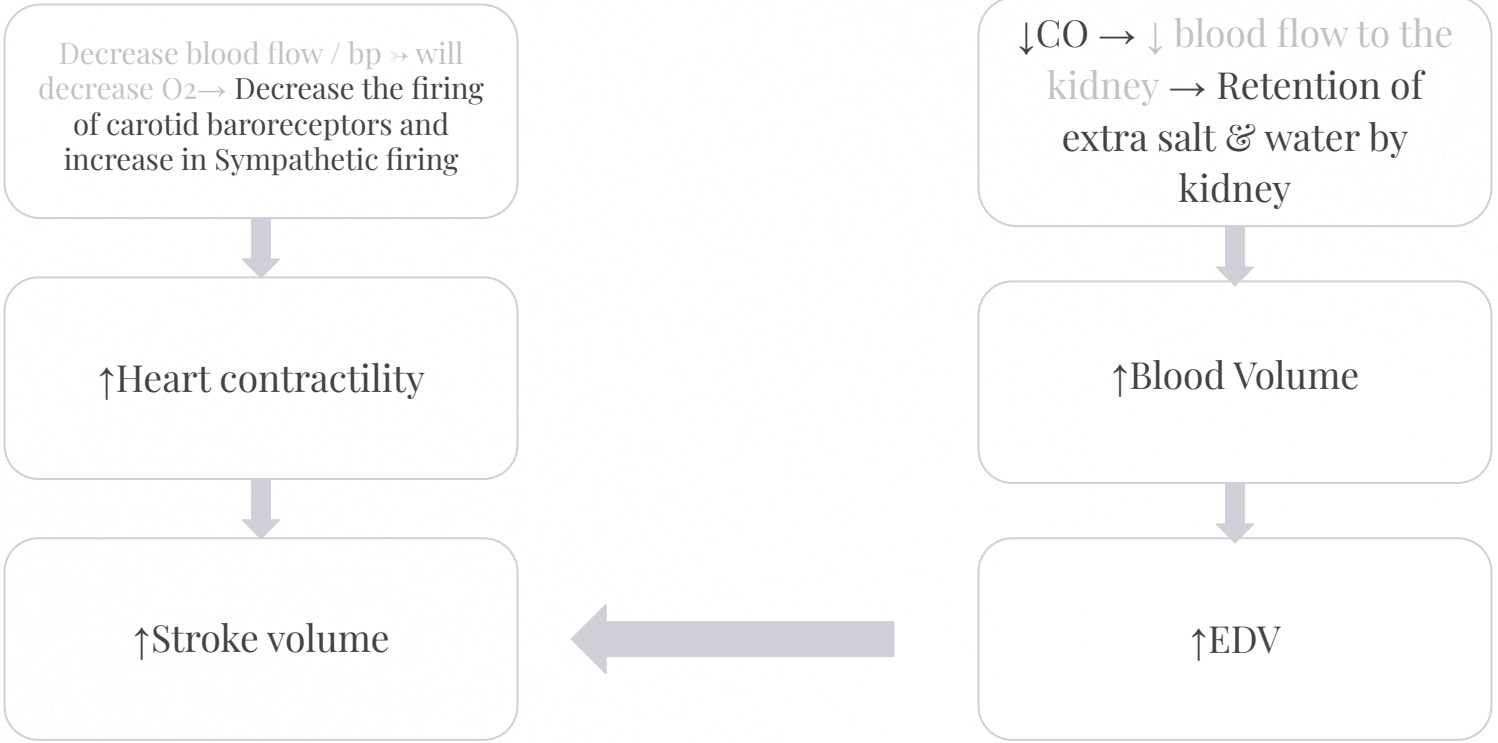
1. ↓ firing of carotid sinus baroreceptor → ↑sympathetic stimulation → vasoconstriction of arterioles (increased afterload) & vasoconstriction of veins (increased preload). ↠ ↟HR & force of contractility. → ↑CO &↑BP.

2. ↓renal perfusion → Activation of renin– angiotensin-aldosterone system (RAAS)→ retention of extra salt & water by kidney. (angiotensin↠ vasoconstriction) (aldosterone↠ NA & water retention)

3. ↓ effective circulating blood volume → posterior pituitary releases ADH (vasopressin) → ↑H_2O reabsorption.

4. 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).

Compensatory measures in heart failure



Complications of progressive heart failure: Factors contributing to decompensation

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

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

3. Hypertrophied heart → imbalance between the O_2 supply and need → deterioration of the ability to generate force (Hypertrophied heart needs more O_2 & because of heart failure CO to the heart is less than normal)

4. Excessive salt and water retention ↠ edema

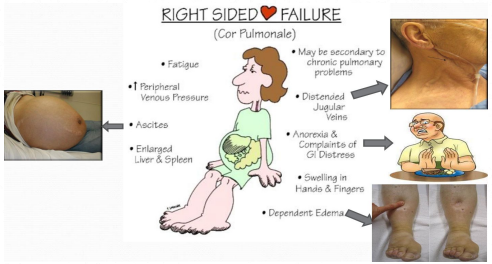
5. Over-distended ventricle (↑diameter): Has to consume more energy and generate more wall tension to develop the required ejection pressure (Laplace low).

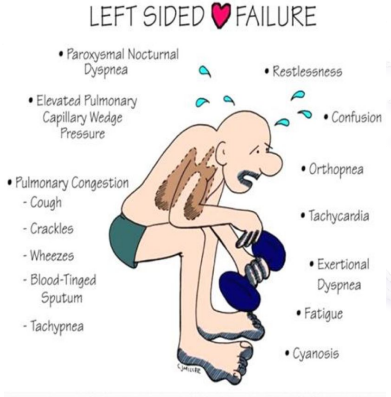
Clinical Picture of HF

Poor Cardiac Output ↠ Poor perfusion

Increased filling pressures ↠ Congestion

Right sided heart failure	Left sided heart failure
Fatigue (↓ blood flow to muscles)	Tachypnea (↑ rate of respiration)
Ascites (accumulation of fluid in the abdomen as a result of systemic congestion).	Orthopnea: 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. (due to pulmonary edema)
Enlarged liver & spleen	shortness of breath (dyspnea)
Distended (elevated) jugular veins	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
Anorexia & complaints of GI distress (↓ blood flow to GIT)	Cough, rales (crackles) due to pulmonary edema
Swelling of hands & Feet	Restlessness, confusion and fatigue
Dependent edema.(pitting edema)	Pallor, cyanosis
–	Tachycardia (compensatory mechanism)





Comparison between clinical picture of right & left sided HF

Clinical picture	Left sided heart failure	Right sided heart 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 (bilirubin)
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?

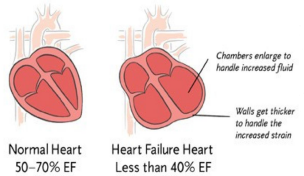
1. Medical history is taken to reveal symptoms

2. Physical Examination. Such as BP, JVP, Palpitation

Tests

3. - Chest X-ray.
   - Electrical tracing of heart (ECG).
   - Ultrasound of heart (Echocardiogram or "Echo").
   - X-ray of the inside of blood vessels (Angiogram).
   - Blood tests (BNP and pro BNP)

4. Ejection fraction (EF) is a Key Indicator for Diagnosing Heart Failure, it's the percentage of blood that is pumped out of the ventricle during each beat.
   If it is less than 40% then it's heart failure. What mechanism is used to measure EF? Fractional Shortening



5. 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 to

Reduction of salt & water retention.
(which will decrease of preload).

↓

Diuretics

↑ In the pumping activity failing heart

↓

Digitalis

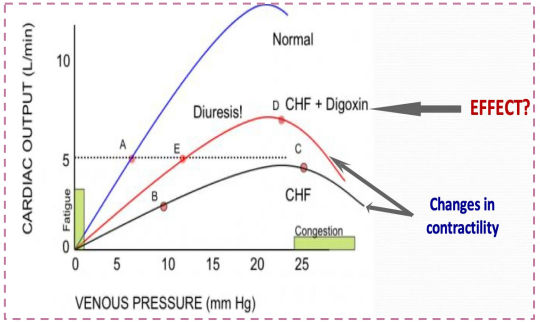
Digitalis improves pumping activity of heart by increasing cytosolic Ca++

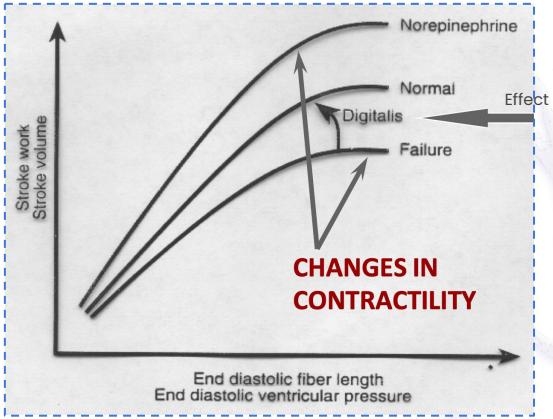
Effects of congestive heart failure & digoxin on Frank-starling curve

Thx Med443

Explanation: 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.

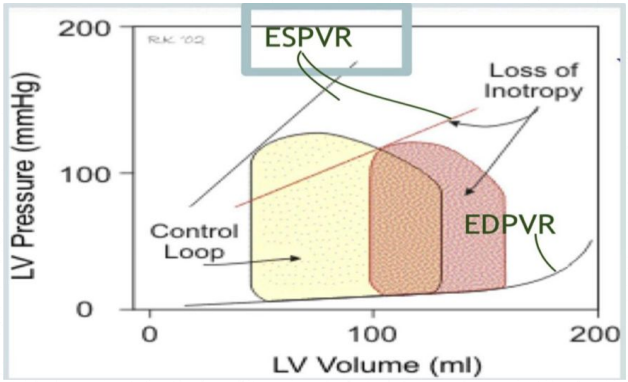




Female's slide

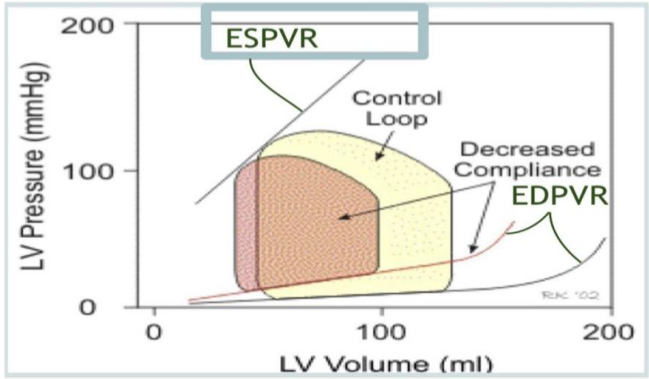
Effects of left ventricular systolic and diastolic failure on LV pressure volume loop

systolic



- ↓ 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.

Diastolic



- ↓ 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.

Check here for our summary
Highly recommended !!!!!

Sorry but if you will not cheack it راحت عليك المليون

MCQs:

Answers

For more question check our summary file!

1/B
2/D
3/C

1 If ECG shows complete heart block. Which one of the following could be observed in her jugular venous pulse curve?

A	Absent of “a” wave	B	Cannon “a” wave	C	Enlarged “c” wave	D	Giant “v” wave

2 in right sided HF which organ is enlarged ?

A	Kidney	B	Liver	C	Spleen	D	Both B&C

3 Absent ‘a’ wave is an indication of:

A	RH failure	B	Tricuspid stenosis	C	Atrial fibrillation	D	Valves stenosis

MCQs:

Answers

For more question check our summary file!

4/C
5/D
6/C

4

What is the mechanism underlying orthopnea and dyspnea in left sided heart failure?

A	↓ RA pressure	B	↓ ventricular EDV	C	Pulmonary congestion	D	Systemic venous congestion

5

heart failure can occur?

A	Only in right side	B	Only in left side	C	In left side then right side	D	All of the above

6

which of the following is a clinical sign of left sided heart failure?

A	Ascites	B	Anorexia	C	Orthopnea	D	Edema

SAQ

How heart failure can be diagnosed?

Explain briefly what's the cause of each JVP wave and in which cardiac cycle occurs each wave?

What are the JVP Waves abnormalities and give an example for each one:

Answers..

1. Slide 28
2. Slide 9
3. Slide 12

Finally you have arrived , we have been waiting for you !!

Meet our team !

Team leaders

Rimaz Alhammad

Noreen Almaraba

Rayan Alshehri

Omar Albaqami

Aljoharah Alyahya

Heroes of the lecture :

Faris Alturaiki

Nora Alturki

Elaaf Albadi

Did you like the lecture ? we mean our work :)

Contact with us! physiology.444ksu@gmail.com