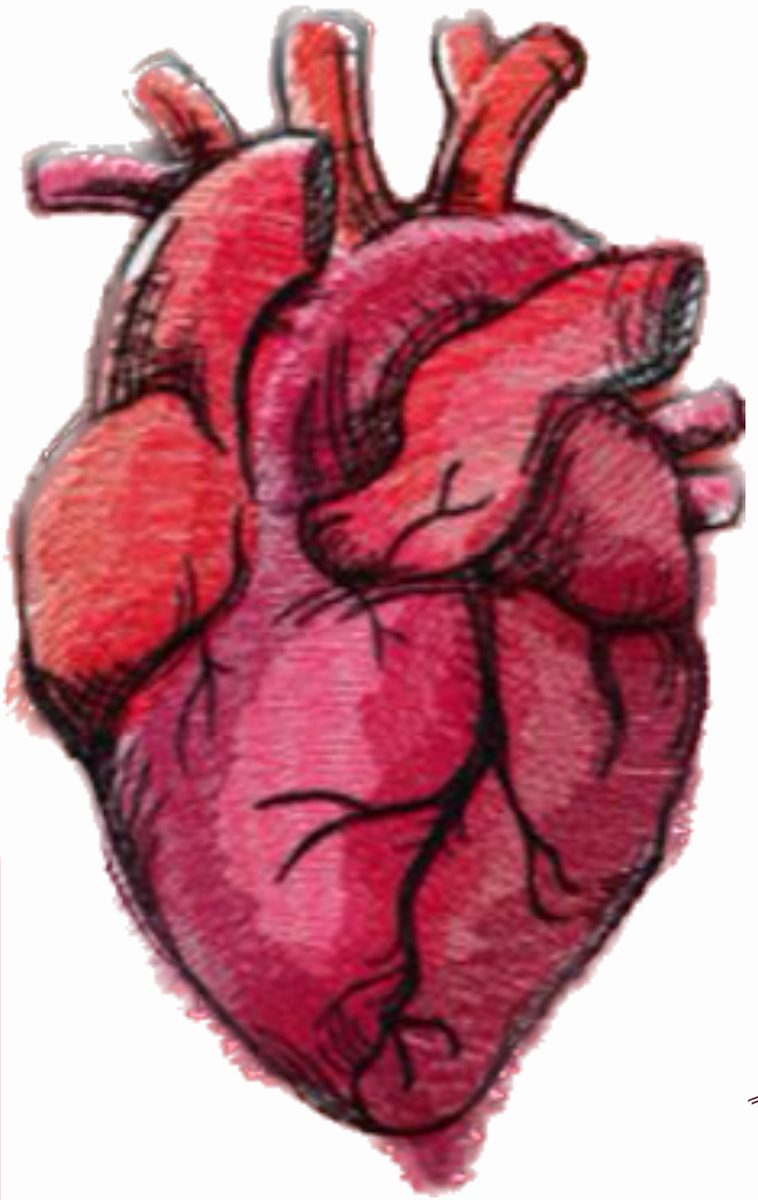

النادي الطبي

CARDIOVASCULAR SYSTEM

SUBJECT : physiology

LEC NO. : 15

DONE BY : Abdullah Bani Mustafa

وَقُل رَّبِّ زِدْنِي عِلْمًا

SCAN ME!

Heart Failure

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Lectures Objectives:

1. Define heart failure, and the difference between low output and high output heart failure.
2. Describe etiology and basic pathophysiology of chronic heart failure (CHF).
3. Explain terminology related to CHF.
4. Describe signs and symptoms of CHF.
5. Outline therapeutic goals of treating CHF.

Heart failure → no treatment so we aim to stop the progress of it instead of curing it

that doesn't mean that CO is small during rest, at the first stages of heart failure the CO during resting is normal & no symptoms during resting → symptoms occur when there is increase in demand for O_2 so it won't be able to increase CO during to heart failure

Definition: The inability of the CO to keep pace with the body's demand for supplies and removal of wastes. Heart failure is a **progressive syndrome** due to a variety of disease processes. Sometimes heart failure is represented as increase in CO at rest for eg. 8 instead of 5 /min so the heart start to fail after a period of time due to increase of CO at rest also it cannot increase the CO if needed

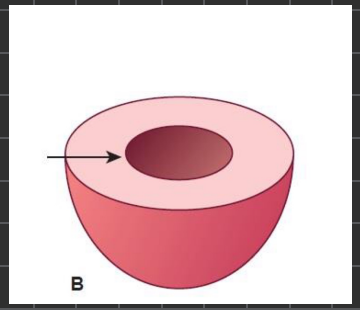
Pathogenesis: It is mainly due to;

1. Prolonged pumping against a chronically elevated afterload (BP).
   - On the left side; ischemic and hypertensive heart disease → increase of after load of LV → ↑ resistance → uncontrolled cause heart failure
   - On the right side; cor pulmonale that is a complication of chronic lung disease, multiple pulmonary emboli, and primary pulmonary hypertension. started with the lung then effect the heart ↳ silent killer ↑ pressure in pulmonary artery → ↑ after load in front of RA ↳ no reason causing ↑ pressure in pulmonary artery
     - increase resistance of blood flow in the lungs
     - after multiple occlusion it will be hard to pump blood from the heart to the lungs cause ↑ in pulmonary pressure
2. After myocardial infarction load is increased on remaining viable cells. large part of one of the ventricles suffer from myocardium infarction causing the death of large number of cells so the rest functioning cells won't be able to compensate → causing "cardiogenic heart failure"
3. Variety of other disease processes such as valvular heart disease, congenital heart disease, and cardiomyopathies.
   - eg. valve diseases → Tetralogy of Fallot
   - "unknown causes"
   - ↳ causing murmur → left untreated cause heart failure
   - eg. aortic valve stenosis → LV can't pump blood effectively to aorta ↓ ↑ after load ↓ failure of LV
   - ■ ↑ Afterload → Concentric hypertrophy of myocytes with little hyperplasia & cardiac dilation & failure.
     - ↳ increase thickness of ventricles but the heart cavity stay the same
     - ↳ due to increase of after load (explained in next slide)
   - ■ ↑ Preload or ↓ afterload → ↑ CO → Eccentric hypertrophy → Heart failure.
     - explained in next slide
     - in case of incompetence ↓ pump 70 ml to the aorta but part of it 20 ml would be back to the heart ↑ EDV → causing chronic increase in EDV ↑ contractility → until it can't

the thickness of the ventricles walls are increased while the cavity volume / EDV is the same with slight decrease in so, the thickness increased so it could be able to overcome the after - load → it's explained by Laplace's law

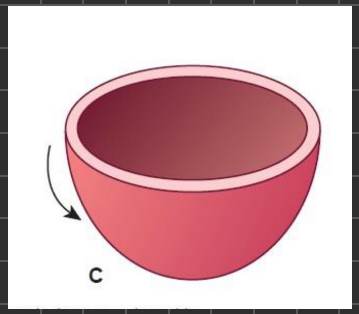
→ tension = $\frac{\text{Pin chamber} \times \text{Radius}}{\text{wall thickness}}$

if the pressure is increased in LV to overcome the increase of after load → ↑ tension the heart try to reduce the increase in tension by increase the wall thickness so the tension would be back to normal by hypertrophy with same radius this is called "concentric hypertrophy" which then end with heart failure with time



Now lets talk about the increase of EDV / pre load

eg. aortic incompetence → ↑ EDV causing chronic decrease in after load due to frank starling law "the heart pump what it receive" → so the heart now is pumping 100 but through the valve 30ml come backs & to circulate then 70 is re-added by venous return ≈ 100 causing increase in LV volume / EDV & decrease in thickness of the ventricle wall this is called "Eccentric hypertrophy" which then end with heart failure with time



marathon runners have large heart because they pump 100 ml SV → while u $\frac{70}{5}$ CO = 5

↳ it's not pathological because it's balanced with the thickness of the wall

70 ml x 70 heart beat/minute

normal 70 x 70 = 5000 ml/minute

athleats 50 x 100 = 5000 ml/minute } same cardiac output

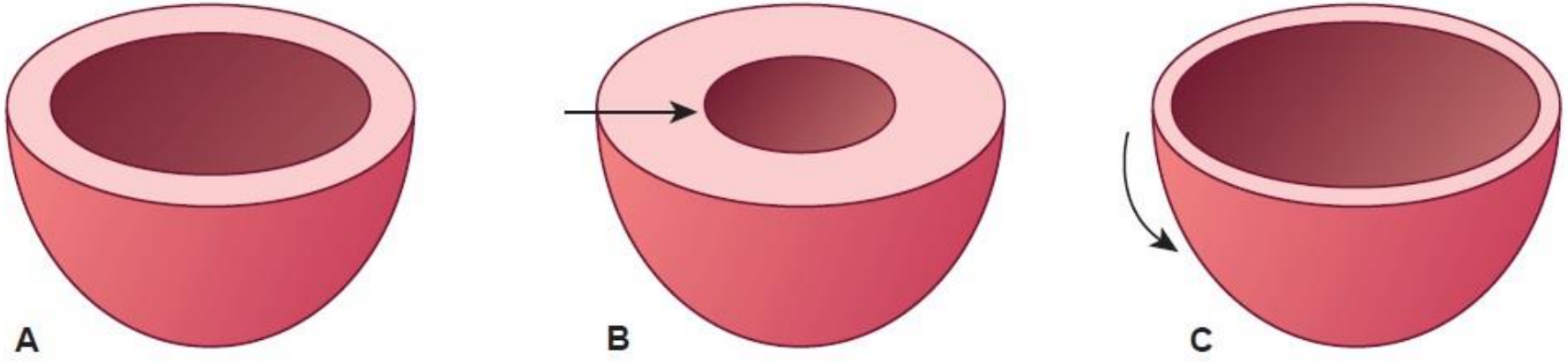


FIGURE Different types of myocardial hypertrophy. (A) Normal symmetric hypertrophy with proportionate increases in myocardial wall thickness and length. (B) Concentric hypertrophy with a disproportionate increase in wall thickness. (C) Eccentric hypertrophy with a disproportionate decrease in wall thickness and ventricular dilation.

* B & C could end up in heart failure if they remain untreated.

Pathophysiological mechanisms of heart failure:

it might be as result of both which is the worst type

1. Systolic dysfunction describes ejection abnormalities that slow the rate of pressure rise during isovolumic contraction and the rate and extent of ejection

   The systolic dysfunction = ↓ vent. contraction and ↓ SV and ↑ ESV.

   ↳ contractility is reduced → but with normal diastole so the EDV → but can't pump norma SV due to the weak contractility

   → which we can conclude that ejection fraction ↓ ($\frac{SV}{EDV}$)

   So if we see reduced ejection fraction it's systolic HF

   Causes of systolic dysfunction include left ventricular damage caused by myocardial infarction, dilated cardiomyopathies, viral myocarditis, and toxic and metabolic abnormalities.

2. Diastolic dysfunction describes impaired ventricular filling by slowing the rate of pressure fall during isovolumic relaxation, decreasing the rate of filling, or increasing stiffness throughout diastole

   → normal ejection fraction so normal ejection fraction doesn't rule out HF

   Diastolic dysfunction = ↓ vent. elasticity + ↑ vent. Pr. + ↓ vent. Filling → ↑ venous congestion and edema.

   before contraction ↑ due to decrease in area volumes

   the heart can no longer expand to receive the full amount of venous return → ↓EDV & ↓SV

   in LV → Pulmonary hyper tension

   RV → edema & hepatomegaly

   accumulation of fluid in interstitial fluid due to increase blood volume in veins

Pathophysiological mechanisms of heart failure (cont.):

Diastolic dysfunction can be caused by wall thickening and reduced cavity volume in patients with hypertension, myocardial ischemia, or hypertrophic cardiomyopathies. And restrictive diseases like amyloidosis, fibroelastosis, and pericardial disease.

3. In systolic dysfunction ejection fraction % *decreases* from normal of 65% (range 55%-70%) to as low as 20%. However, ejection fraction could be *normal* in diastolic dysfunction. so again normal ejection fraction doesn't rule out HF
4. CO decreases initially only during exercise, but then it decrease at rest.
   ↓ late stages of HF
   ↓ at first stages of HF

Note: systolic and diastolic dysfunction commonly occur together, which makes it difficult for any hemodynamic measurement to identify which of these mechanisms is responsible for impaired pump function.

↳ symptoms at rest occur when this mechanism fail

Compensatory mechanism: why at the beginning of HF doesn't effect the patient at rest?
can succeed initially as there is;

1. ↑ Sympathetic activity to the heart. explained in the next slide
   the Sympathetic stimulation will effect the kidney increasing reabsorption of Na+ & water to ↑ blood volume
2. Kidney retains salt & water to increase EDV to pump back normal CO at a longer cardiac muscle fiber length (heart looks bigger in chest X-ray). As the disease progresses, the heart reaches a point at which it is no longer able to pump out a normal SV. The mechanism is as such;

hypervolumea can be detected by chest x ray

on chest x ray
عدى القلب لازم ما يزيد عن ½ عرض الصدر

The decrease in BP → ↓ baroreceptor reflex → ↑ sympathetic activity + renal vasoconstriction + ↑ renin secretion (and aldosterone secretion) → Na^+ and water retention.

Note: People with heart failure often use their cardiac reserve at rest. Therefore, just climbing a flight of stairs may cause shortness of breath because they have exceeded their cardiac reserve.

need more CO can't be achieved by compensatory mechanism → Symptoms appear

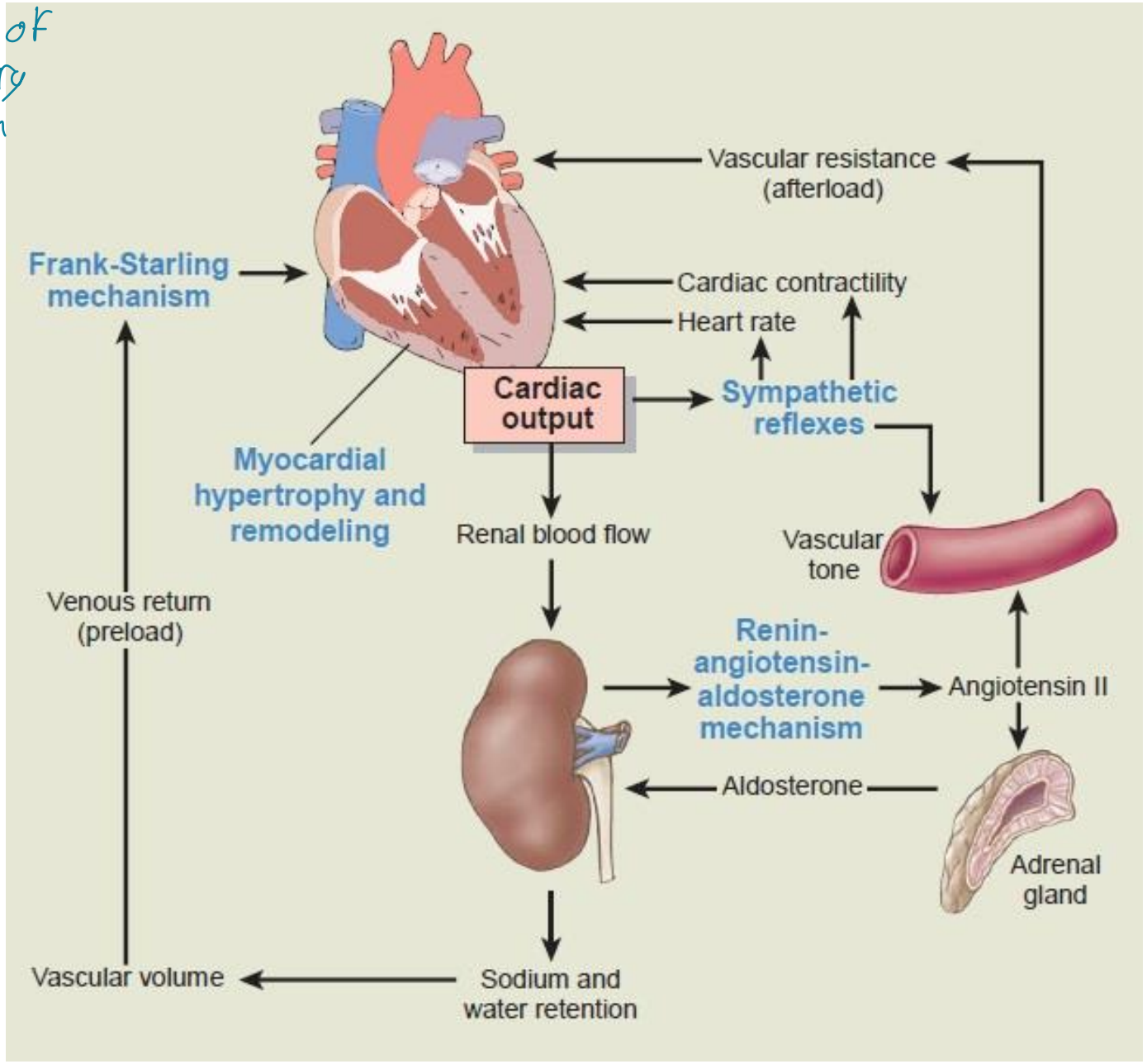
1) increase sympathetic activity of the heart

at the beginning of HF BP is reduced (still in the normal range) but baroreceptor defect that small decrease in BP cause reflex sympathetic stimulation → ↑ noradrinaline reaching to the heart → ↑ B1 receptors stimulation → ↑ contractility + sympathetic stimulation will affect the kidney so renin will cause increase in angiotensin 1 production → angiotensin II → causing aldosterone production which increase water-salt retention these mechanism ↑ venous return ↑ CO

Angiotensin II functions:

1) vasoconstriction
2) stimulate to produce aldosterone

summary of compensatory mechanism



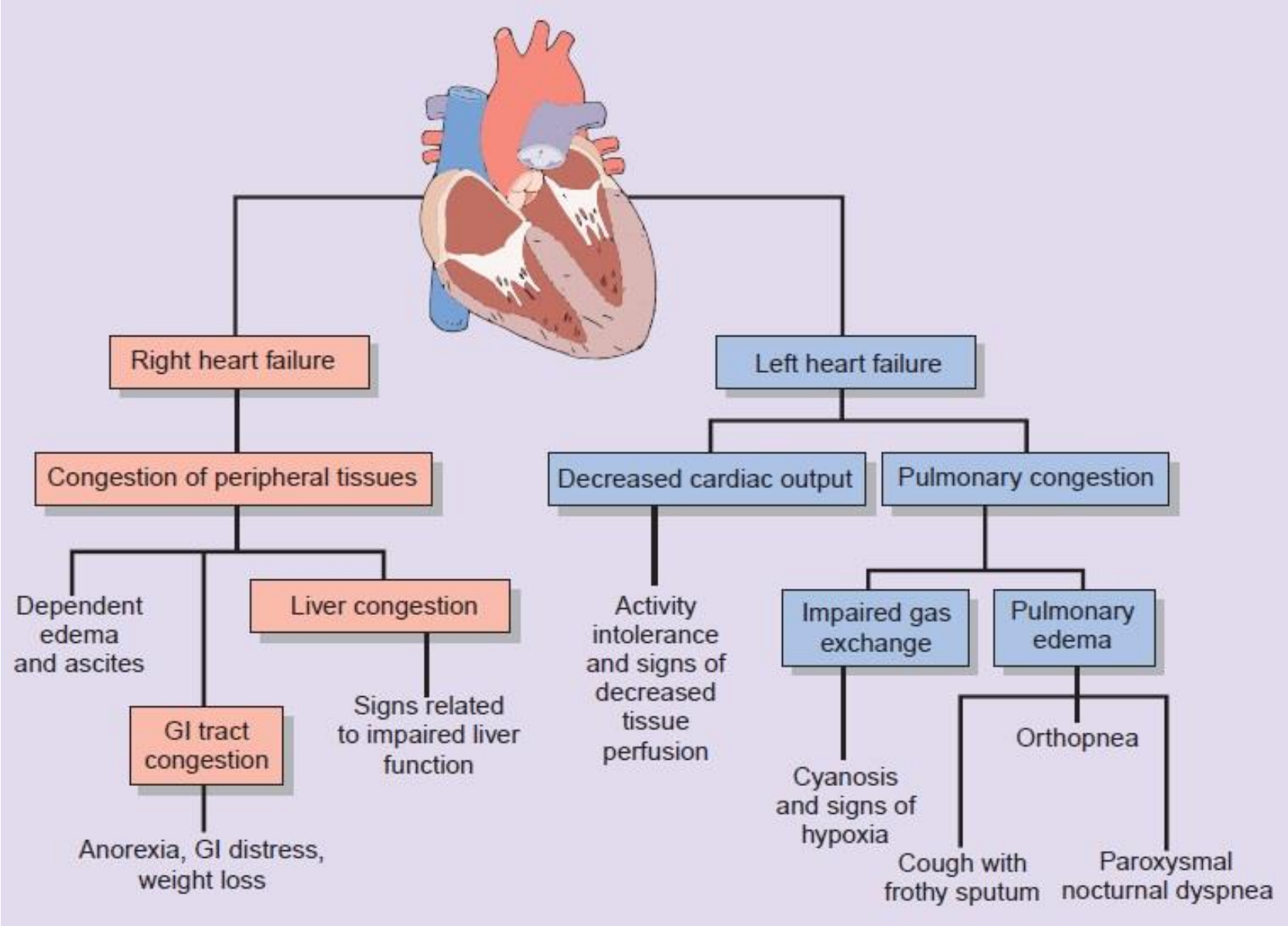
Compensatory mechanisms in heart failure

Types:

1. Low-output failure = normal metabolism + ↓ CO
2. High-output failure = ↑ metabolism or ↓ afterload + ↑ CO but less than demand (e.g. thyrotoxicosis; severe anemia; arterio-venous fistula; vit. Thiamine deficiency or Beriberi)

Manifestation of congestive H. failure:

1. Overt heart failure may be precipitated by conditions such as infection, emotional stress, uncontrolled hypertension, or fluid overload.
2. Sign and symptoms of fatigue + Dyspnea on exertion, and in severe cases orthopnea (= dyspnea precipitated by lying flat)
3. Cardiac enlargement
4. Edema of the dependent portions of the body, ascites, and pleural effusions in right heart failure
5. Case progresses to pulmonary edema (left heart failure)
6. Prolonged circulation time
7. Hepatomegaly + distention of neck veins



Manifestation of left and right-sided heart failure

Treatment:

* increase HR doesn't work because it's already done by the compensatory mechanism
* increase contractility by increasing the stimulation of β_1 receptors in the heart doesn't work either same reason
* increase blood volume to increase venous return to increase CO No → kidney already done it !!

The aim is directed to reverse fluid retention by the kidneys, which is the major cause of edema, and reduce peripheral arteriolar vasoconstriction, which lowers cardiac output and decreases cardiac efficiency; the only solution is to decrease the heart after load by decreasing angiotensin effect because

1) angiotensin cause vasoconstriction & increase water salt retention by ↑ aldosterone so by decreasing it we will cause vasodilation & ↓ blood volume which will decrease after load

1. Increase contractility (by digitalis) to treat systolic dysfunction. inhibit Na^+-k^+ pump ↑ intracellular Na^+ ↑ Ca^{2+} ↑ contractility but cause cellular edema
2. Decrease the load on the heart by decreasing the production of angiotensin II by ACE inhibitors (or by blocking the effect of angiotensin II on AT1 receptors). Such measurement reduces circulating aldosterone level and ↓ blood pressure. Recently aldosterone receptor blockers seem promising.

best solution

The load on the heart can also be reduced by drugs that decrease venous tone; such as nitrates, hydralazine, and diuretics. These drugs ultimately lower the preload.

Note:

1. ATP and phosphocreatine levels are significantly reduced in overloaded and failing hearts. This is because energy production in failing hearts is usually decreased.
2. The use of β blockers was found recently to reduce mortality and morbidity in heart failure as they are energy-sparing. help the heart to increase ATP consumption in effective way
3. Digitalis is especially useful in heart failure that is associated with atrial fibrillation as it slows the ventricular rate. Digitalis is of little benefit in patients who are in sinus rhythm.

Test Question: مش متاكد

Q. Which of the following has the greatest impact on mortality with regards to chronic therapy of heart failure with reduced ejection fraction?

systolic HF weak contractility

A. Beta blockers → No already weak contractility
B. Mineralocorticoid receptor antagonists
C. Angiotensin converting enzyme inhibitors
↳ cause vasodilation which will cause ↓ venous return → progress the problem the heart can't pump so we need to reduce blood volume not increase it
D. Angiotensin II receptor blockers
E. Hydralazine (a potent arteriolar dilator)