PASSION ACADEMIC TEAM YU - MEDICINE

Cardiovascular System

Sheet# 12

Lec. Date :

Lec. Title : Cardiac shock & heart failure

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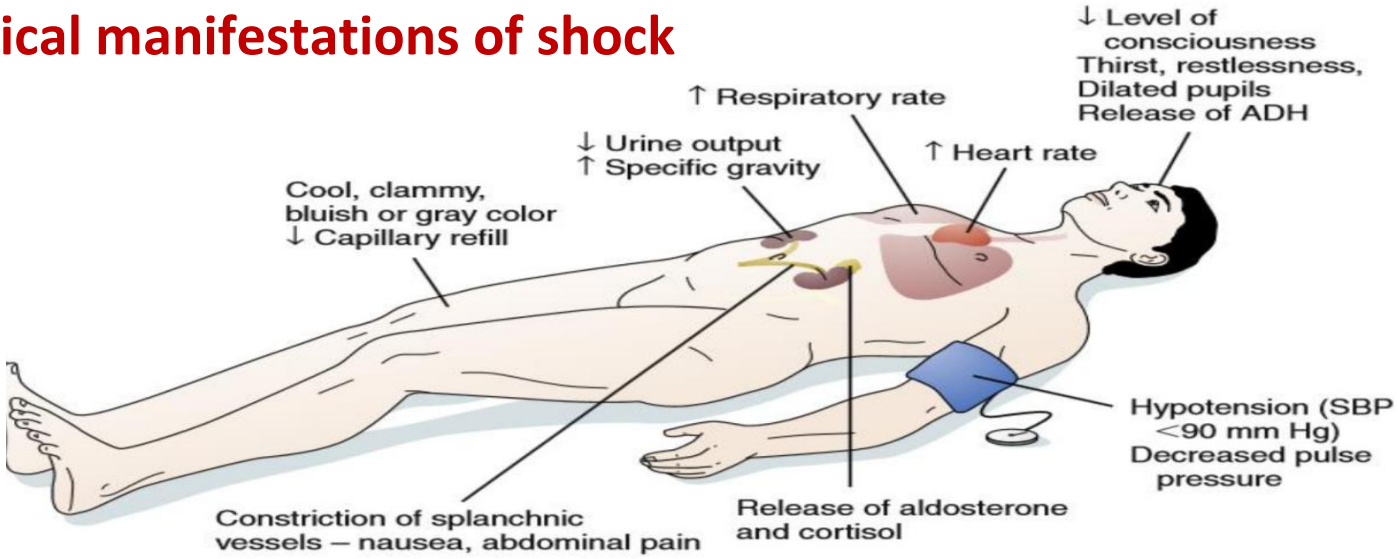
DEFINITION:

- **Shock** is a general term that refers to the depression or suppression of body functions produced by any disorder.
- **Circulatory shock** refers to the shock developed by inadequate blood flow throughout the body or by reduction in cardiac output due to different reasons , due to vessels (vasogenic shock) ,due to heart defects(Cardiogenic shock), due to blood volume defects (Hypovolemic shock)

And so on the circulatory shock is a life threatening condition & it result in death if the effected patient is not treated immediately

✓clinical manifestations of shock



Cardiac output (C.O) reduction → leads to reduction in arterial BP (bc there is direct relationship bw the mean arterial BP and the C.O)

MAP = Q (C.O) × TPR

And so on when C.O is decreased the arterial pressure drops down ..

Low BP produces reflexes → causes **tachycardia** and **vasoconstrictions** (to adjust the BP bc it decreased) these reflexes produced in Baroreceptors

- **tachycardia** → increased HR =decrease cardiac cycle duration→ decreases diastolic phase →filling of the heart reduces → decrease end diastolic volume (preload) →reduction in force of contraction(shift in frank starling curve downward & to the right)→ decrease stroke volume and systolic pressure

- **C.O decrement** → HYPOXIA occur → the skin become pale and cold→also CYANOSIS occur along with hypoxia in many parts of the body , particularly the ear loops & finger tips.

- **reduced C.O &BP** →constriction in renal blood vessels (afferent arterioles in renal system)→ decrease blood flow to glomerulus → **the glomerular filtration rate** and **urinary output reduced**

- Metabolic activity of the myocardium are accelerated, due to reduction in blood flow and increasing in HR but with no enough C.O nor oxygen which leads to → ANEROBIC respiration for ATP generation→causes accumulation of lactic acid →metabolic acidosis

Metabolic acidosis : reduction in PH that **changes the electrolytes concentration** which is imp for generating a current or A.P →myocardial efficiency and pumping action of the heart inhibited leading to further reduction in S.V and C.O

In this case the blood flow to VITAL organs is severely affected for ex. decreasing blood flow to brain tissue leads **to ISCHEMIA** , results in fainting & brain damage.

Finally, damaged of brain tissue and cardiac arrest kill the patient.

All clinical features of all types of shock are similar with minor differences

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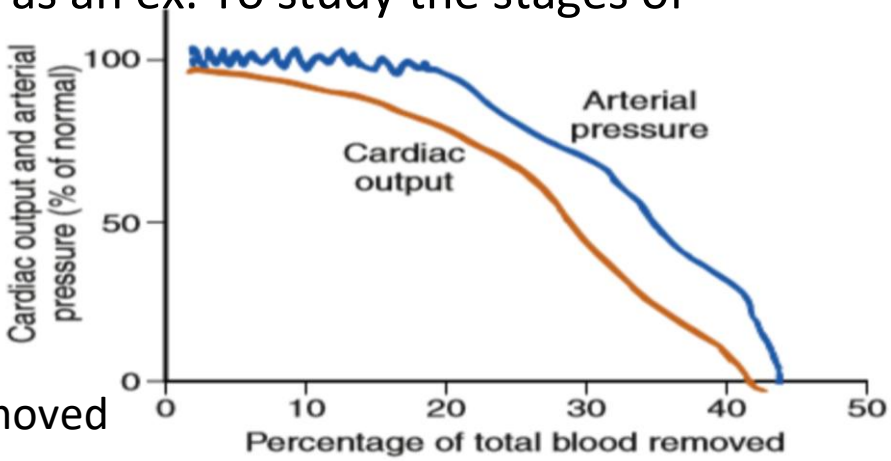
Circulatory shock occurs in three stages:

- First stage or non-progressive stage (also known as compensated.
- Second stage or progressive stage
- Third stage or irreversible stage or refractory shock

Hypovolemic shock (hemorrhagic shock) the most common shock and we will take it here as an ex. To study the stages of the shock .



Y-axis →arterial BP & C.O

X-axis→blood volume that is removed

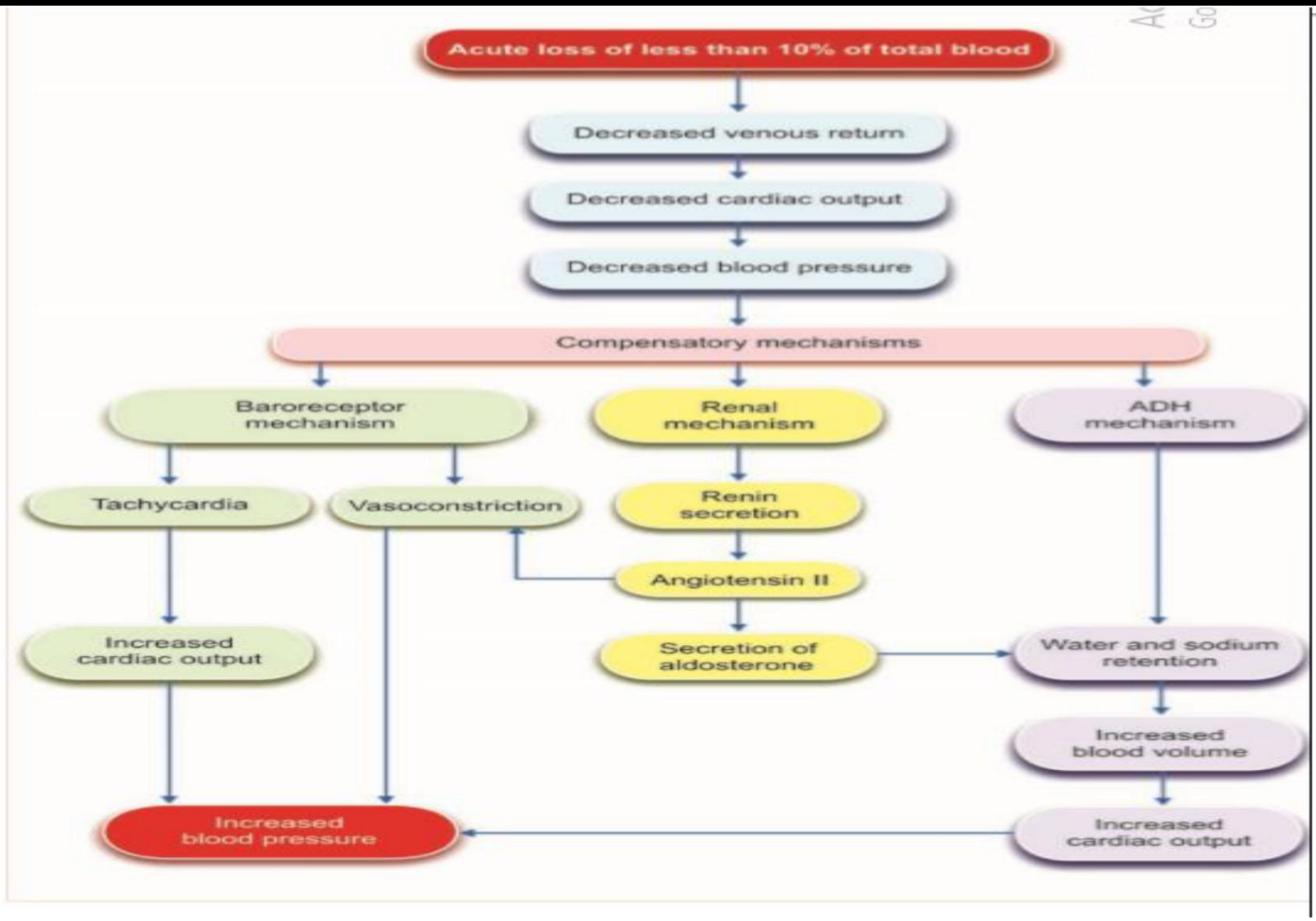
In early stages of Hypovolemic shock, the BP is stable (100) although the cardiac output is Falling, WHY?

✓ Due to compensatory mechanisms that adjust BP and keep it constant even thought the blood flow (C.O) is falling (FIRST stage which is compensated)

BUT when the blood volume loss = 25% or more (as u see on X-axis) →the BP & C.O decrease significantly. In this case (SECOND stage) the compensatory mechanism don't work well to keep the BP stable (not efficient alone), we need treatment along with these compensatory mechanisms

At THIRD stage the compensatory mechanism and treatment both won't be efficient well.

FIRST STAGE OR COMPENSATED STAGE



First stage in hypovolumic shock

If there is blood volume loss but less than 10% of total blood volume, BP decreases ONLY moderately.

Compensatory mechanism in this case can work successfully to re-establish normal BP and keep the BP stable & keep normal blood flow through the body

So on the shocks becomes non progressive and the patient recover.

The compensatory mechanism: is a negative feedback mechanism, includes 3 mechanisms:

1. Baroreceptors mechanism

If BP reduced activates the Baroreceptors will initiate a strong sympathetic stimulation which causes Vasoconstriction & tachycardia (affect SA node , firing of A.P faster , increase HR ,affect vessels(constriction), increase resistance which finally leads to increase BP)

2. Renal mechanism

The glomerular cells releases large amounts of renin →increases angiotensin II formation →produces intense vasoconstriction &increases the release of aldosterone from adrenal cortex (which promote water & salt retention by the kidney) which results in restoration in blood volume & more venous return → more C.O → MORE BP

3. ADH mechanism

Anti-diuretic hormone, released from posterior pituitary gland and increases water retention by the kidney

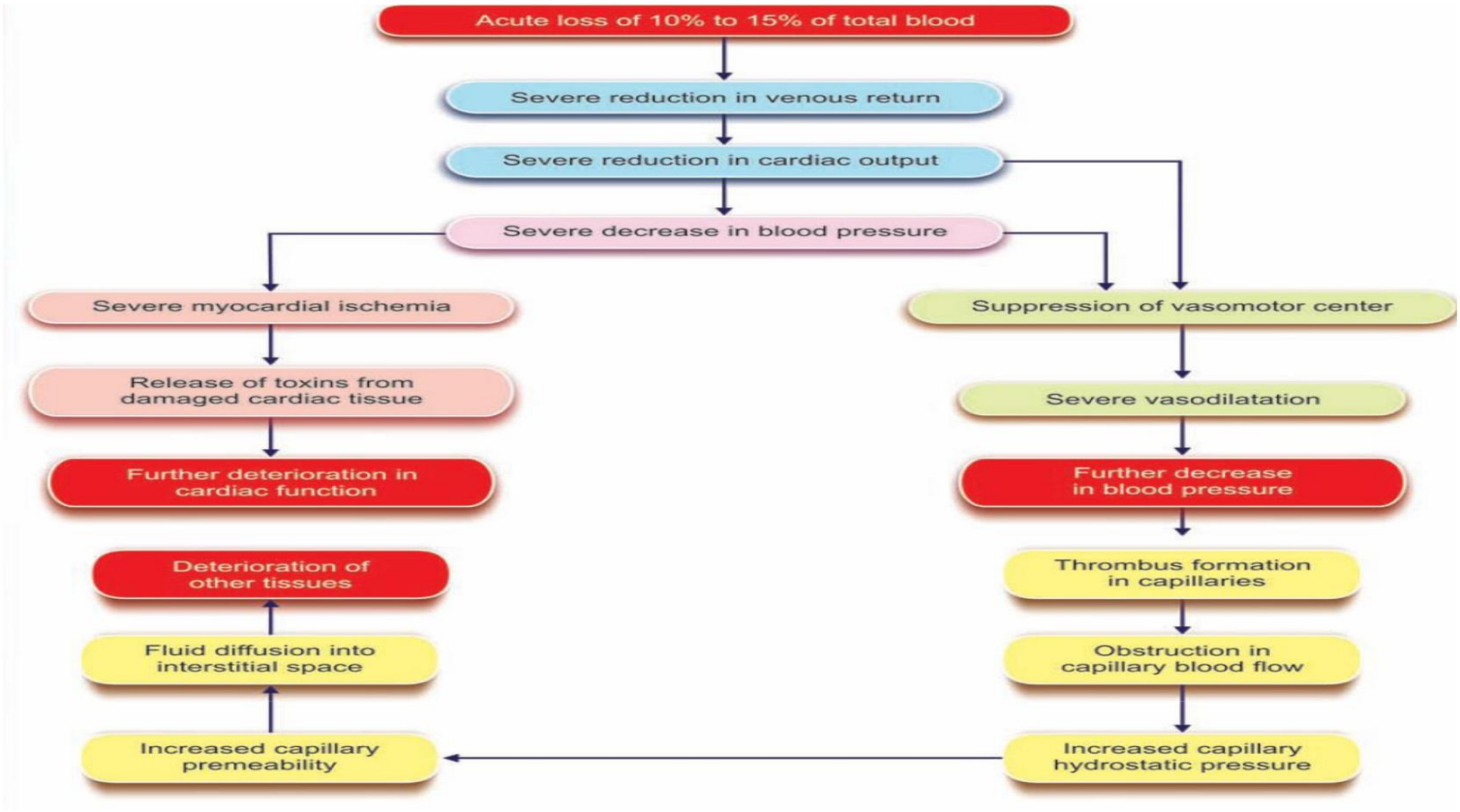
Also ADH enhances vasoconstriction.

All these 3 mechanism will result in:

Water retention, shift in interstitial fluid into capillaries due to vasoconstriction AND increase resistance

All together increase BP and keep it stable

SECOND STAGE OR PROGRESSIVE STAGE



Second stage (progressive stage or decompensated)

When the shock is severe which means the total blood loss **at least 15% of total blood volume**, there won't be negative feedback system BUT **positive feedback** system develops that result in vicious cycle (means the compensatory mechanism result in MORE reduction in BP)

And So on at this stage there must be an immediate and appropriate treatment to reverse this shock (bc of vicious cycle it make the state worse but with treatment we can reverse it)

Due to reduction in blood flow and BP to a low level→leads to inadequate blood flow to cardiac muscle → cardiac muscle start deteriorating due to lack of nutrition and oxygen

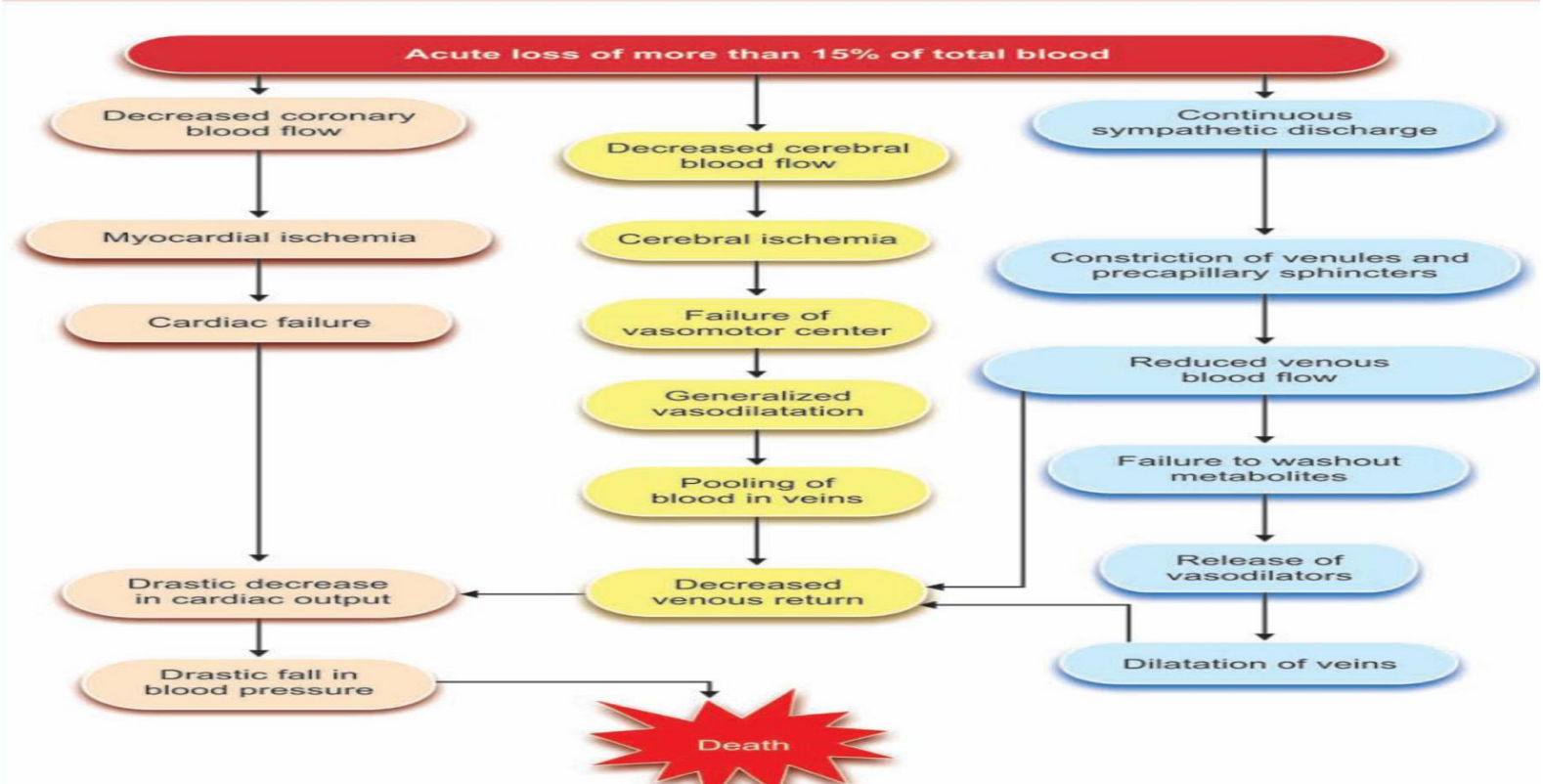
So on TOXIC sub released from damaged tissue & then theses toxins will suppress the myocardium further

ALSO **suppression in vasomotor system** occur due to reduction of blood flow to this system &reduction in BP →which causes suppression of sympathetic system that causes further decrease in BP →less blood flow to cardiac muscle (vicious cycle) more lack of nutrition & O2 , more toxins ….leads to severely damaged myocardium ..

Thrombosis start in small blood vessels which causes increase in capillary hydrostatic pressure = more permeability and more filtration of fluid → shift of fluid from blood vessel into interstitial space, which decreases blood volume…

Due to all prev. causes the severe symptoms start appearing and the shock move to irreversible stage but with treatment it can be reversed

THIRD STAGE OR IRREVERSIBLE STAGE



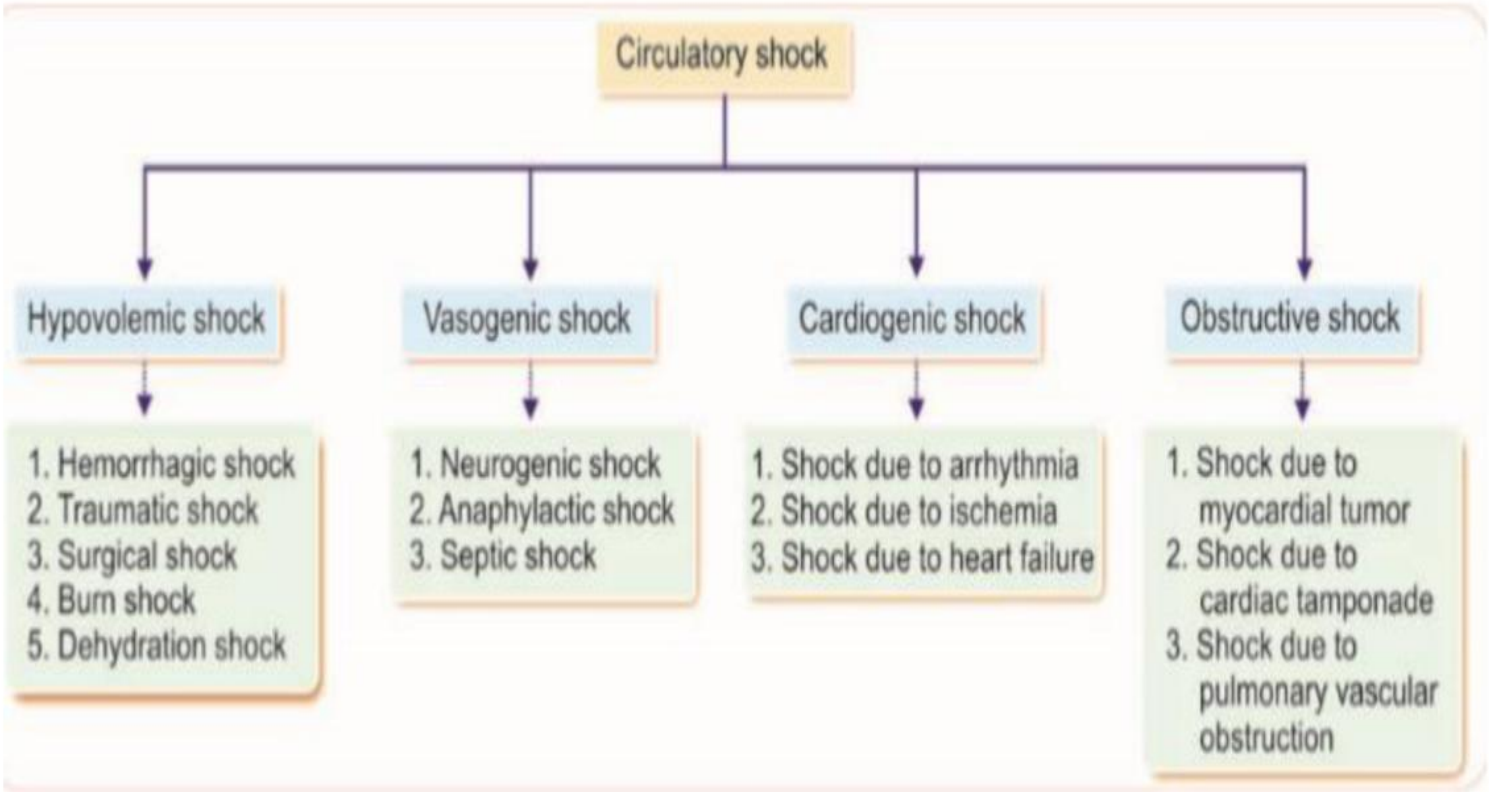
Third and the last stage before collapse.

this stage leads to DEATH regardless of treatment offered to the patient (irreversible even with treatment) bc the brain fails to function normally due to severe cerebral ischemia (severe lack of O2 & nutrition to brain tissue)

ALSO BP falls drastically (largely) even infusion of blood to patient will fail to save him.

Cardiac fail, severe damage to myocardial activity, reduce arteriolar tone all resulting in death.

TYPES AND CAUSES OF CIRCULATORY SHOCK



There are 4 types of shock :

1. Hypovolemic shock: due to decreased blood volume, occur when there is acute blood loss, at least 10-20 %

Different causes for losing blood, Due to:

- Acute hemorrhagic : we call it hemorrhagic shock
- Injury : called traumatic shock
- blood loss during a surgery :surgical shock
- Due to burns : called burn shock
- Due to alteration in metabolism (metabolic diseases like diabetes or adrenal insufficiency)
- Severe diarrhea or vomiting : called dehydration shock

2. Vasogenic shock: due to increased vascular capacity, when neural reflexes or toxic sub causes:

Excessive vasodilatation within vascular system (which means there is storage of fluid and blood in capacitance vessels) which result in reduction in venous return and C.O although the blood volume is NORMAL, no decrease in volume unlike Hypovolemic shock.

Vasogenic shock is 3 types:

✓ **Neurogenic shock :** occurs due to 2 types of nervous effect , BOTH causes massive vasodilation and reduction in C.O :

A. marked reduction in sympathetic vasomotor tone (no tonic vessels which results in significant decrease in BP), we must have resistance downstream , because it causes reduction in BP and increase in ΔP (pressure gradient)

B. marked increase in vagal tone

- **Anaphylactic shock**: due to **allergic reaction** (large quantities of histamine & histamine-like sub causes marked vasodilation→ reducing peripheral resistance & so on reduce BP...
  And marked increase in capillary permeability (shift of fluid from the capillary into the interstitial fluid) which causes fluid loss → adding Hypovolemic shock to anaphylactic (loss of blood volume) .

- **septic shock** : In which **bacteria** circulate and multiply in the blood AND form toxic sub which result in vasodilation and reduction in BP

3.cardiogenic shock : a shock due to cardiac diseases (the source of the shock is the HEART)

occur due to decrease pumping ability of the heart , although the vascular blood volume is NORMAL , but the cardiac output is insufficient due to reduced pumping.

MI, Congestive heart failure (CHF) all leads to Cardiogenic shock

4. Obstructive shock: due to obstruction of blood flow , and due to impairment of ventricular relaxation → impaired filling during diastole , which occurs due to many reasons :

The most imp is due to external pressure on the heart , like:

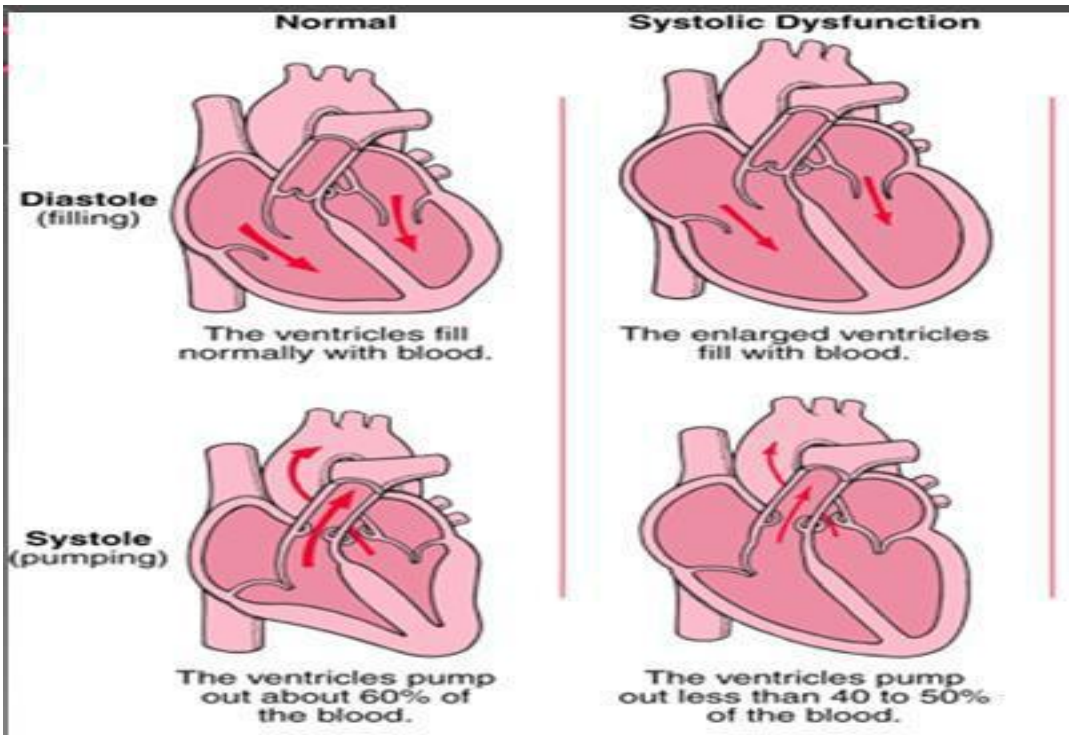
- epicardial effusion or bleeding in pericardium that create external pressure on the hearts & prevent the heart to relax normally
- Tension pneumothorax
- Pulmonary embolism leads to obstructive shock, HOW? (question for you to look for the answer)

HEART FAILURE

- ✓ Heart failure or cardiac failure is the condition in which the heart looses the ability to pump sufficient amount of blood to all parts of the body.
- ✓ Heart failure may involve left ventricle or right ventricle or both (more common in left ventricle and then move to right ventricle)
- ✓ It occurs either due to:
  - decreased myocardial contractility
  - an increased pressure overload (after load, hypertension for ex.) or volume overload(increase in end-diastolic volume , preload).
- ✓ The usual physiological alterations are:
  - a decreased stroke volume (in forward failure)
  - Damming (accumulation) of blood in the venous compartment (in backward failure).

TYPES OF HEART FAILURE

- ✓ **Systolic Heart Failure**



- Heart failure due to decreased ability of heart to contract
- May involve right or left ventricles or BOTH .
- Different causes, all due to muscular weakness & valvular defects.

In the figure, in normal heart there is normal diastole filling and normal pumping in systole (pumping out 60% of blood) which is **normal ejection fraction**

Systolic dysfunction , we can see **enlarged ventricles** , although contains **normal blood volume**(no problem in filling) the problem is that the heart can't pump this volume out with sufficient force & so on reduction in stroke volume → reduction in ejection fraction about **20%** (amount of blood ejected to the lungs and the body is decreased and more blood stay in ventricles)

Accumulation of blood & fluid in the ventricles, means more amount of blood accumulated in lungs or in systemic veins or both of them, leads to pulmonary edema or peripheral edema

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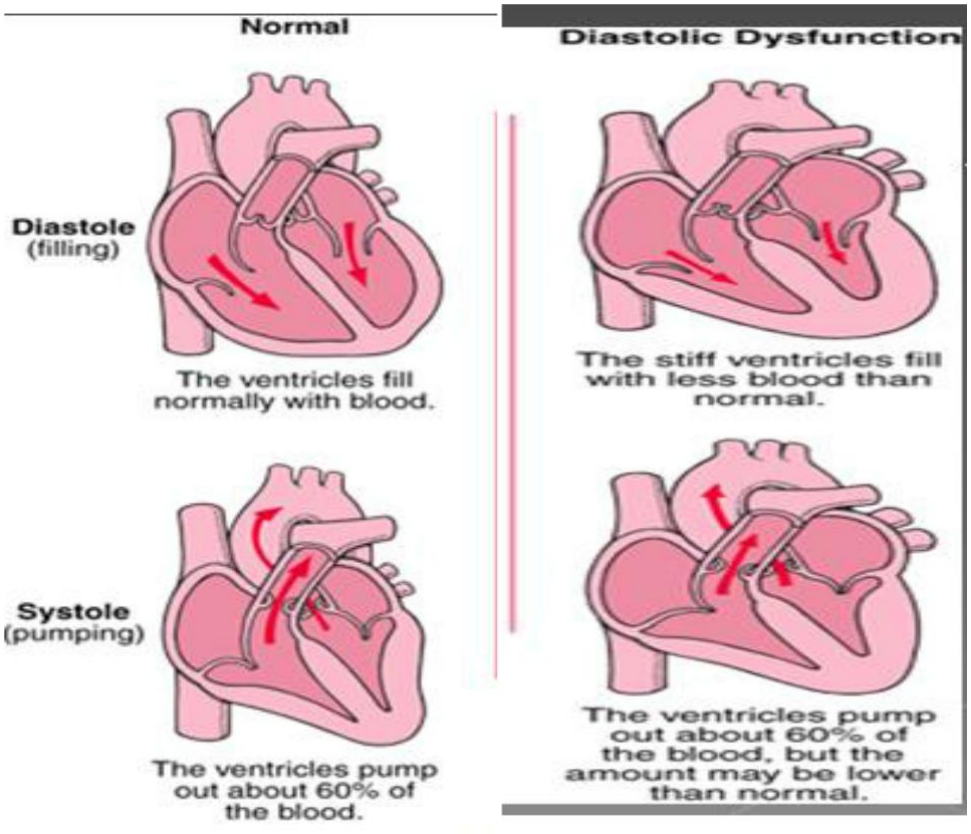
✓ Diastolic Heart Failure



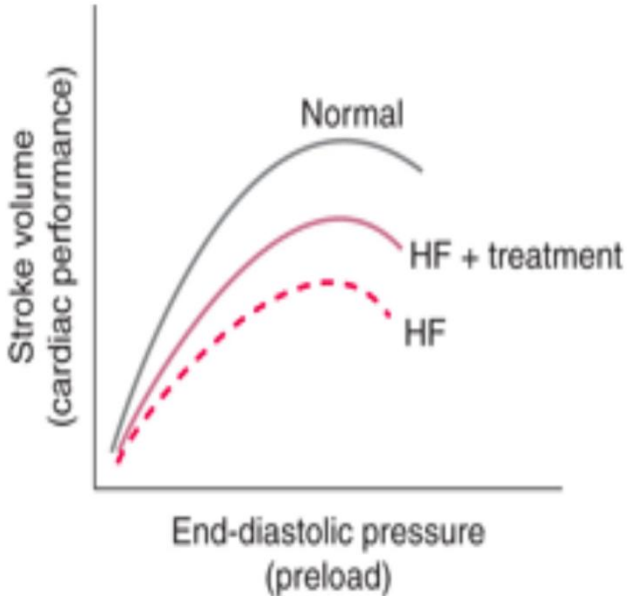
Diastolic dysfunction: hypertrophy of cardiac wall, abnormal stiffness of ventricles and the compliance of the ventricles reduced….
All results in a reduced ability to fill sufficiently at normal diastolic filling pressure(less diastolic volume, less preload than normal)

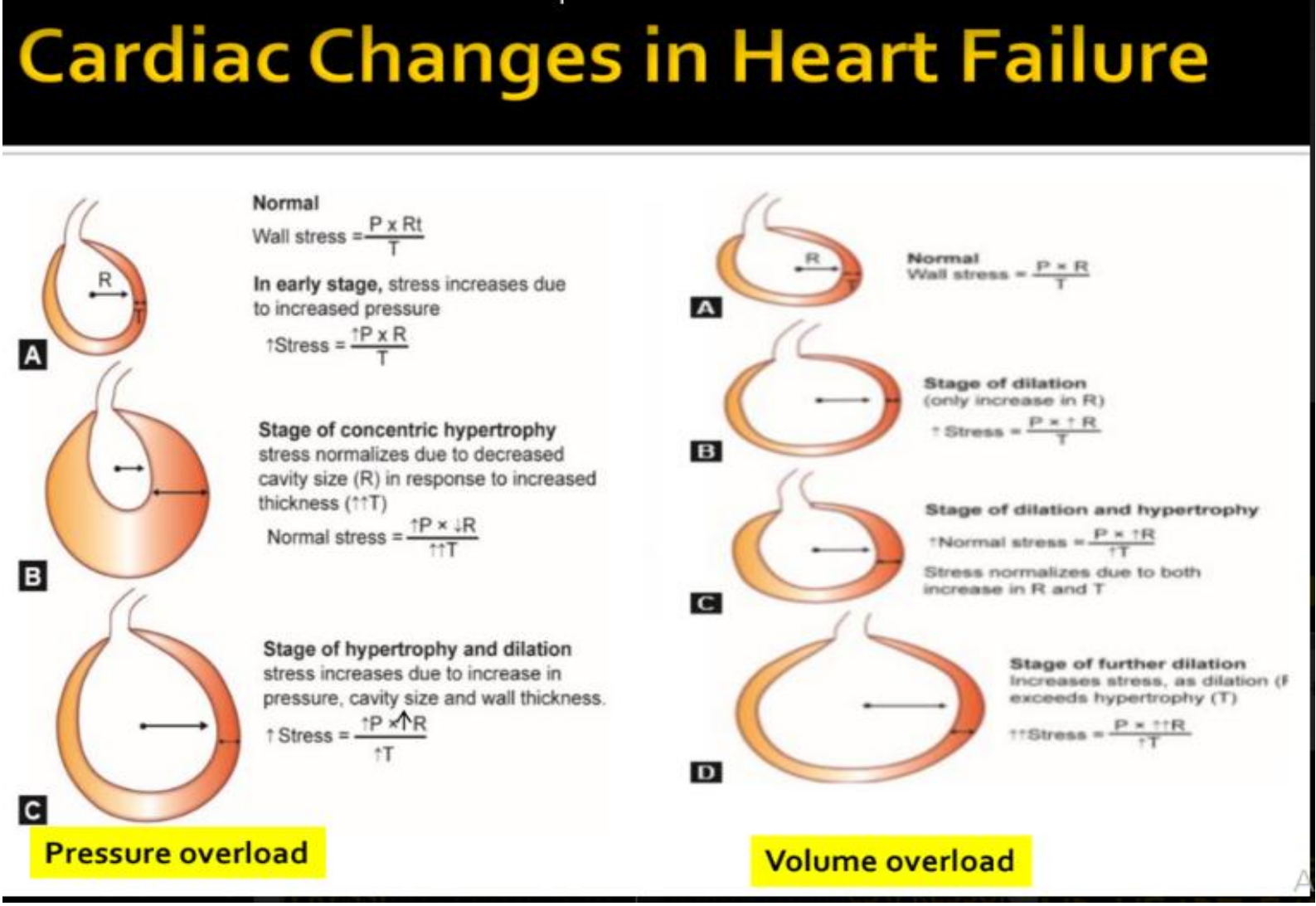
Although the compliance of ventricles decreased, but ventricular contractility still normal, force of contraction is normal so the ventricles can eject the same normal ejection fraction (no weakness of ventricular contractility)

(Normally more **end diastolic volume** = the more **stroke volume**) but here the contractility is high, although end diastolic volume in abnormal but the ejection fraction is normal bc the contractility is normal

If continuous progress occurs in this defect it will result in reduced ejection fraction and reduced stroke volume as the figure below (frank starling curve shift to right and downward)

This also leads later to pulmonary edema on left side and peripheral edema on the right side





The modification that occurs in heart failure:

The heart tries to Overcomes the failure by <u>increasing HR</u> or <u>increasing the thickness of cardiac wall</u> (hypertrophy)

✓ The changes in **<u>pressure overload</u>** heart failure:

Finally causes ventricular **systolic dysfunction** (systolic heart failure ...

In normal heart the wall tension(stress) of ventricles : is the product of the distending pressure and radius of the ventricles divided by thickness of ventricular wall (Laplace law) **stress= P*R/T**

at A in early stages of pressure overload , ventricular pressure increases (due to more afterload) and so on the wall stress(tension) increases…

Increasing in wall stress (no efficient heart function) so compensation occur → which is concentric hypertrophy (thickened wall) → **normalization of the wall stress** due to greater increase in the wall thickness that decreases the radius

If this situation became chronic with no treatment, it will reach to stage of failure at C (stage of hypertrophy& dilation) wall stress increases again due to proportional increase in pressure, thickness and radius, so on dilatation in heart occurs… Warning the heart of systolic heart failure

Figure at the right:

✓ The changes in **volume overload**

in volume overload finally leads to **diastole** dysfunction(diastolic heart failure) if not treated

A is normal heart.

B stage of dilation, in early stages of volume overload ventricles dilate for ventricular to accommodate more blood, dilatation causes increase in cavity diameter (radius) which leads to more increase in wall stress

C (compensation stage) extrinsic hypertrophy of ventricles occurs → normalize the wall stress due to proportionate increase in radius & thickness and the heart work normally

But if no treatment applied, the condition become chronic and inter into stage D

D Stage of failure (further dilation) →wall stress increase again due to increase in ventricular cavity size as result of further dilation which warn of diastolic heart failure.

Good, better, best

Never let it rest.

Till your good is better

And your better is best...