



CVS

PHYSIOLOGY



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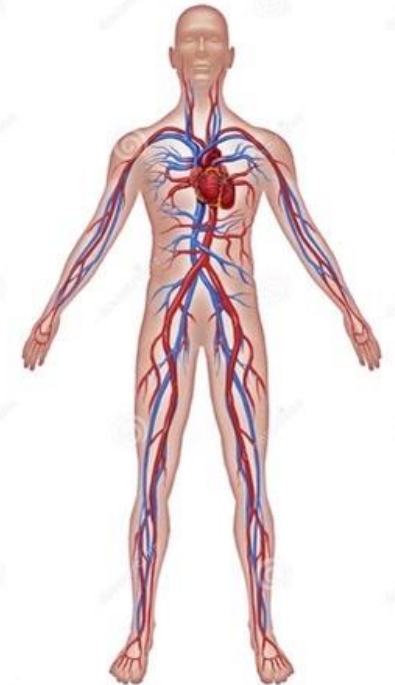
Cardiovascular Physiology

Fatima Ryalat, MD, PhD

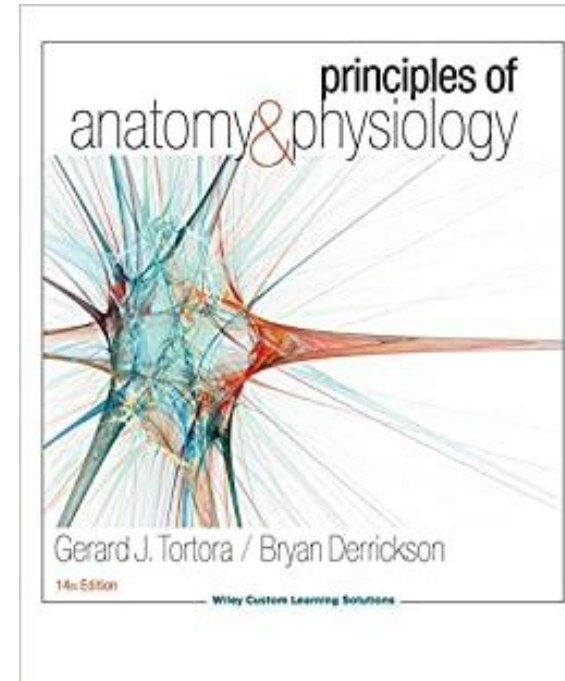
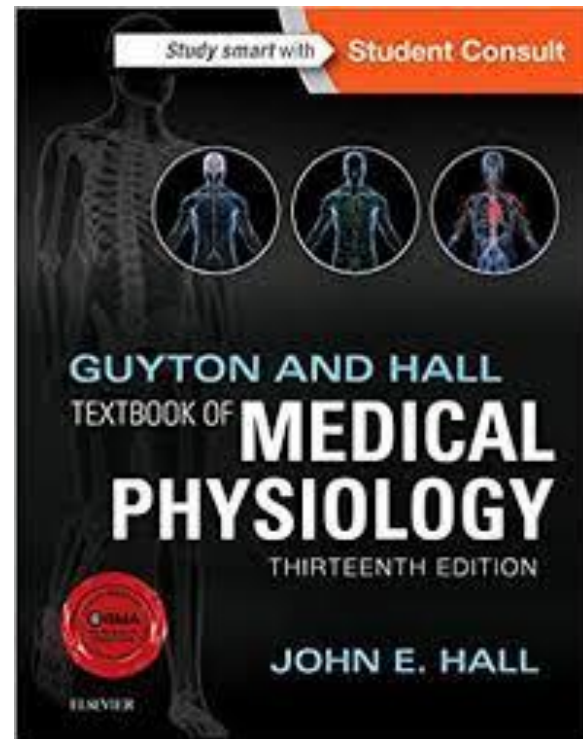
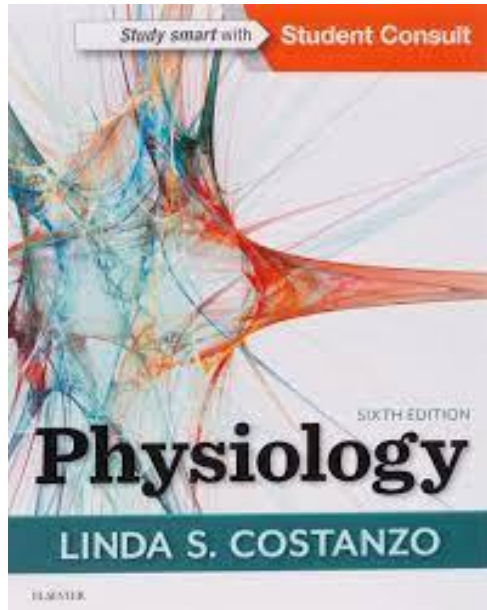
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Color code

■	Slides
■	Doctor
■	Additional info
■	Important



References



9TH
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Human Physiology From Cells to Systems

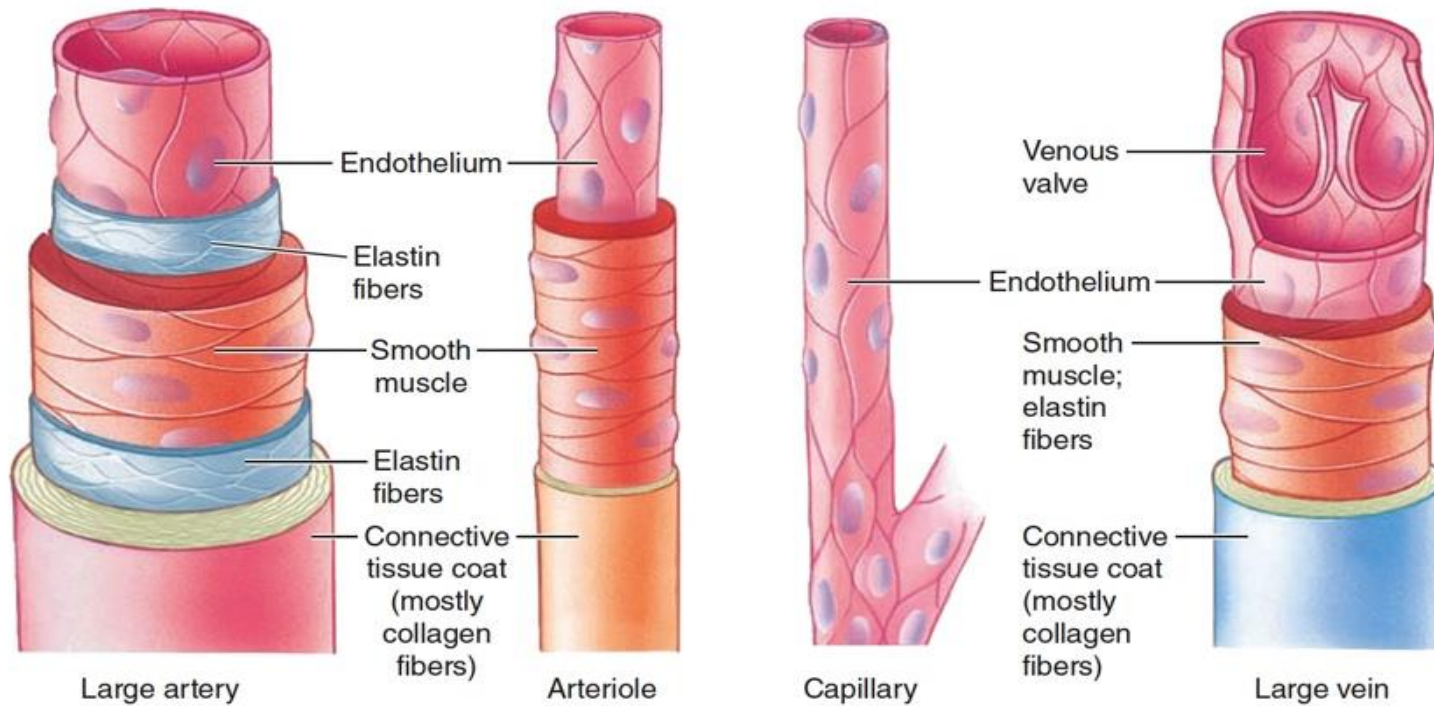
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Veins



- Large veins are characterized by their wide diameter and thin walls. They contain a thin layer of smooth muscle, which is innervated by the sympathetic nervous system. However, the contraction of this muscle is not as strong as that seen in arterioles.
- Veins have a property known as “compliance,” which allows them to accommodate large volumes of blood with minimal changes in pressure.

• Unlike elastic arteries, veins lack elastic recoil or elasticity. Instead, they function as capacitance vessels, meaning they can store blood within their walls until needed by the circulatory system. This unique property allows veins to act as a blood reservoir. For example, if you lose up to 20% of your blood volume, the veins’ capacitance helps prevent significant hemodynamic changes.

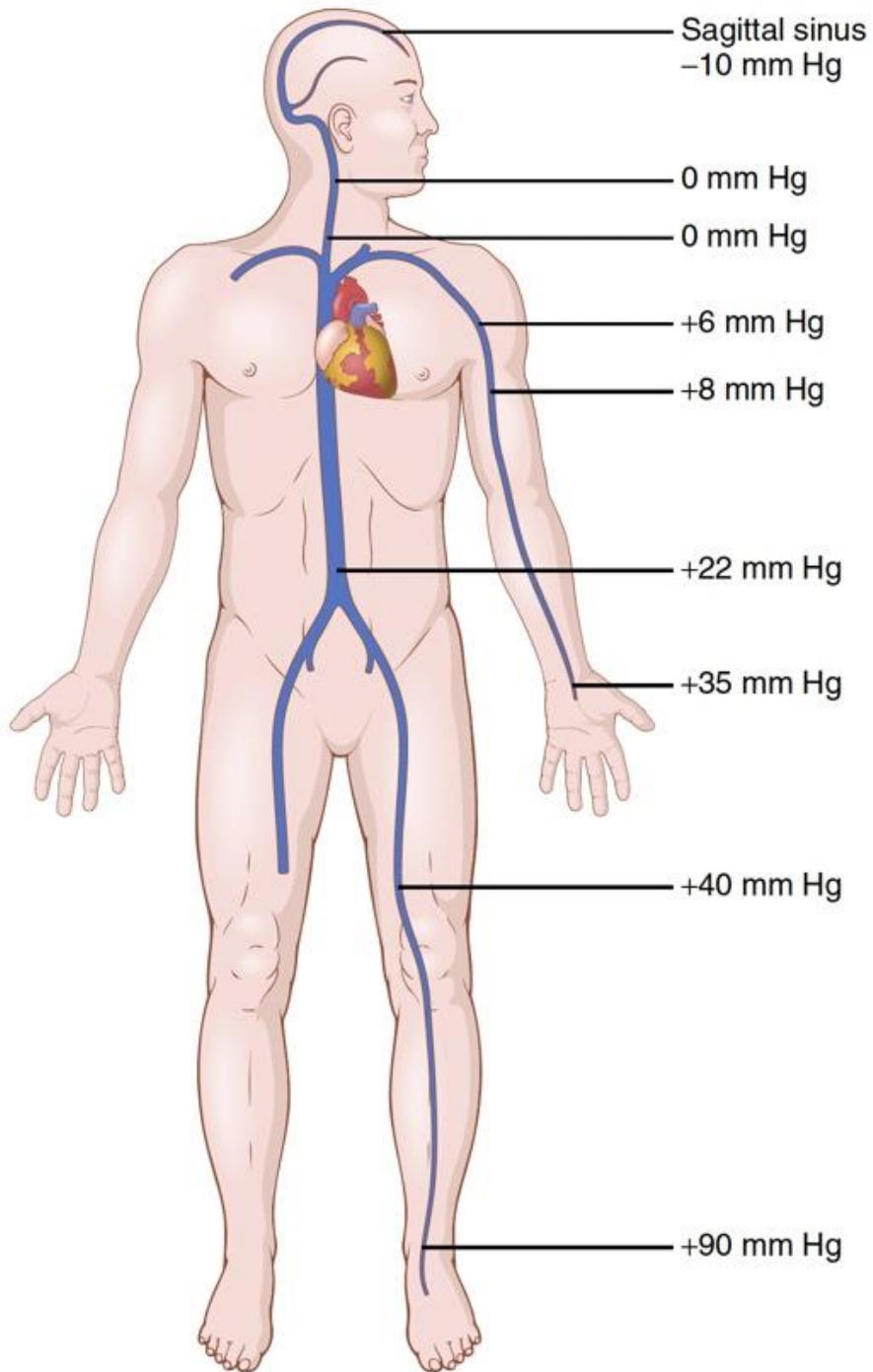
• Sympathetic stimulation induces vasoconstriction (venoconstriction), reducing the diameter of veins. As the diameter decreases, venous capacitance also decreases, leading to an increase in venous return. This process mobilizes the blood stored in the venous walls, directing it toward the heart.

Veins as blood reservoir

- more than 60% of all the blood in the circulatory system is usually in the veins.
- For this reason, and also because the veins are so compliant, the venous system serves as a blood reservoir for the circulation.
- Even after as much as 20% of the total blood volume has been lost, the circulatory system often functions almost normally because of this variable reservoir function of the veins.

Venous capacity

- Venous capacity (the volume of blood that the veins can accommodate) depends on the distensibility of the vein walls (how much they can stretch to hold blood) and the influence of any externally applied pressure squeezing inwardly on the veins.
- At a constant blood volume, as venous capacity increases, more blood remains in the veins instead of being returned to the heart.
- Such venous storage decreases the effective circulating blood volume, the volume of blood being returned to and pumped out of the heart.
- Conversely, when venous capacity decreases, more blood is returned to the heart and is subsequently pumped out.
- Thus, changes in venous capacity directly influence the magnitude of venous return, which in turn is an important determinant of effective circulating blood volume.



- So, veins function to store blood to be used when needed, and to bring back this blood to the heart (Right Atrium).
- Remember that VR (venous return) is the volume of blood that's brought back to right atrium by the veins per unit time, so it is a Flow.
- Remember that flow depends on the Pressure gradient as well as the Resistance.
- In the venous system, venules have a pressure of approximately 17 mmHg, while the pressure in the right atrium is close to 0 mmHg. Although this creates a pressure gradient, it is relatively low compared to the arterial system, where the pressure in the aorta is about 100 mmHg and decreases to around 37 mmHg on the arteriolar side.
- The venous side has a low-pressure gradient, and veins offer low resistance due to their large diameter. To enhance venous return (VR) to the right atrium, the sympathetic nervous system plays a crucial role.
- As mentioned earlier, sympathetic stimulation induces venoconstriction, which helps increase VR. However, it's important to note that venoconstriction in veins is less potent compared to the vasoconstriction observed in arterioles.

Important note:

Sympathetic stimulation has different effects on veins and arterioles:

- **In veins**, sympathetic stimulation causes vasoconstriction, reducing their capacity to store blood. This leads to an **increase in venous return (VR) to the heart**, which subsequently raises the volume of blood circulating in the system.
- **In arterioles**, sympathetic stimulation causes vasoconstriction, reducing the amount of blood flowing to the tissues. As a result, less blood reaches the veins, which ultimately **decreases venous return (VR) to the heart**.

Sympathetic stimulation of the veins

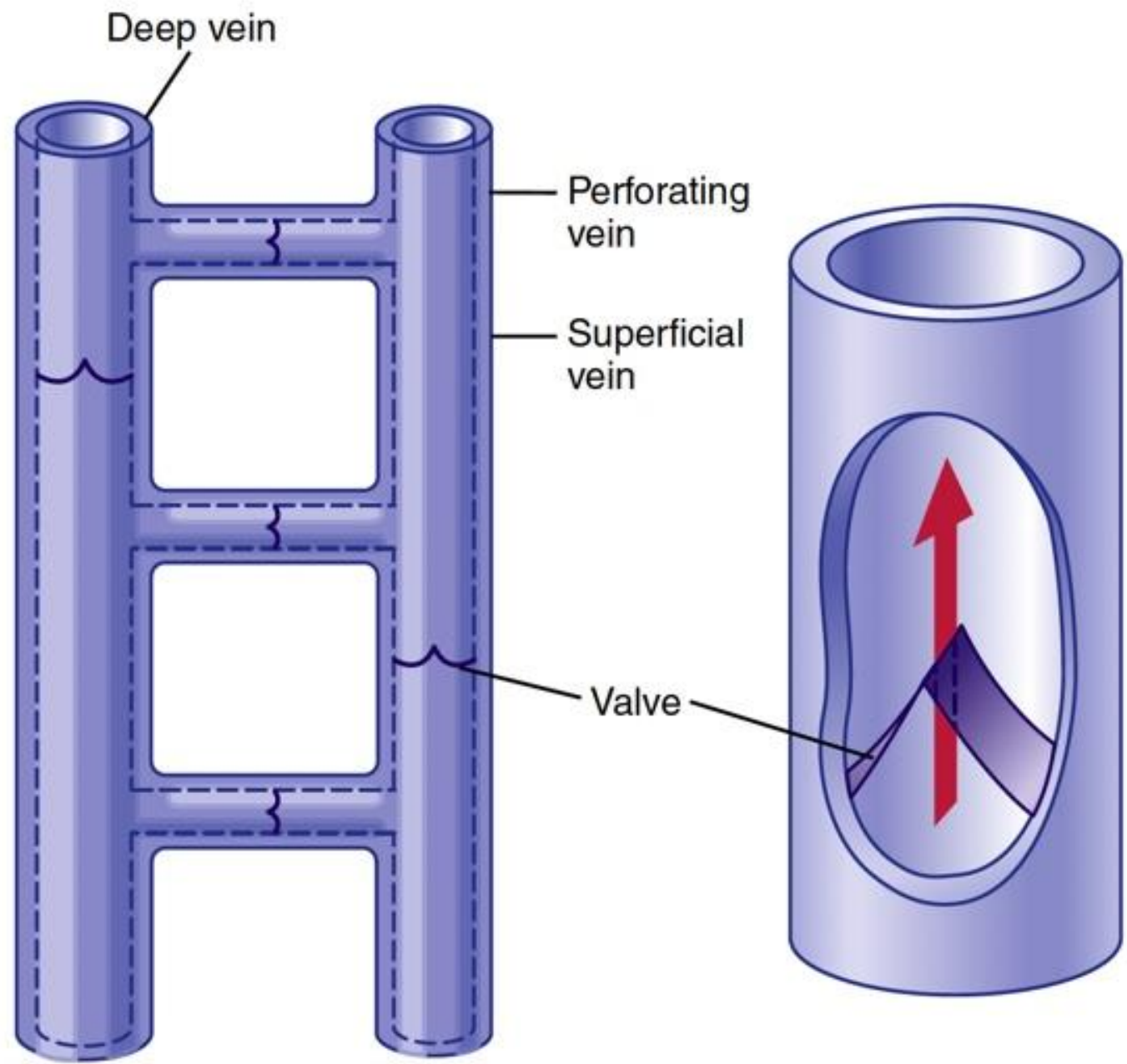
- Veins are not very muscular and have little inherent tone, but venous smooth muscle is abundantly supplied with sympathetic nerve fibers.
- Sympathetic stimulation produces venous vasoconstriction, which modestly elevates venous pressure; this, in turn, increases the pressure gradient to drive more of the stored blood from the veins into the right atrium, thus enhancing venous return.
- Veins normally have such a large radius that the moderate vasoconstriction from sympathetic stimulation has little effect on resistance to flow.
- Even when constricted, veins still have a relatively large radius and are still low-resistance vessels.

Arteriolar vs venous vasoconstriction

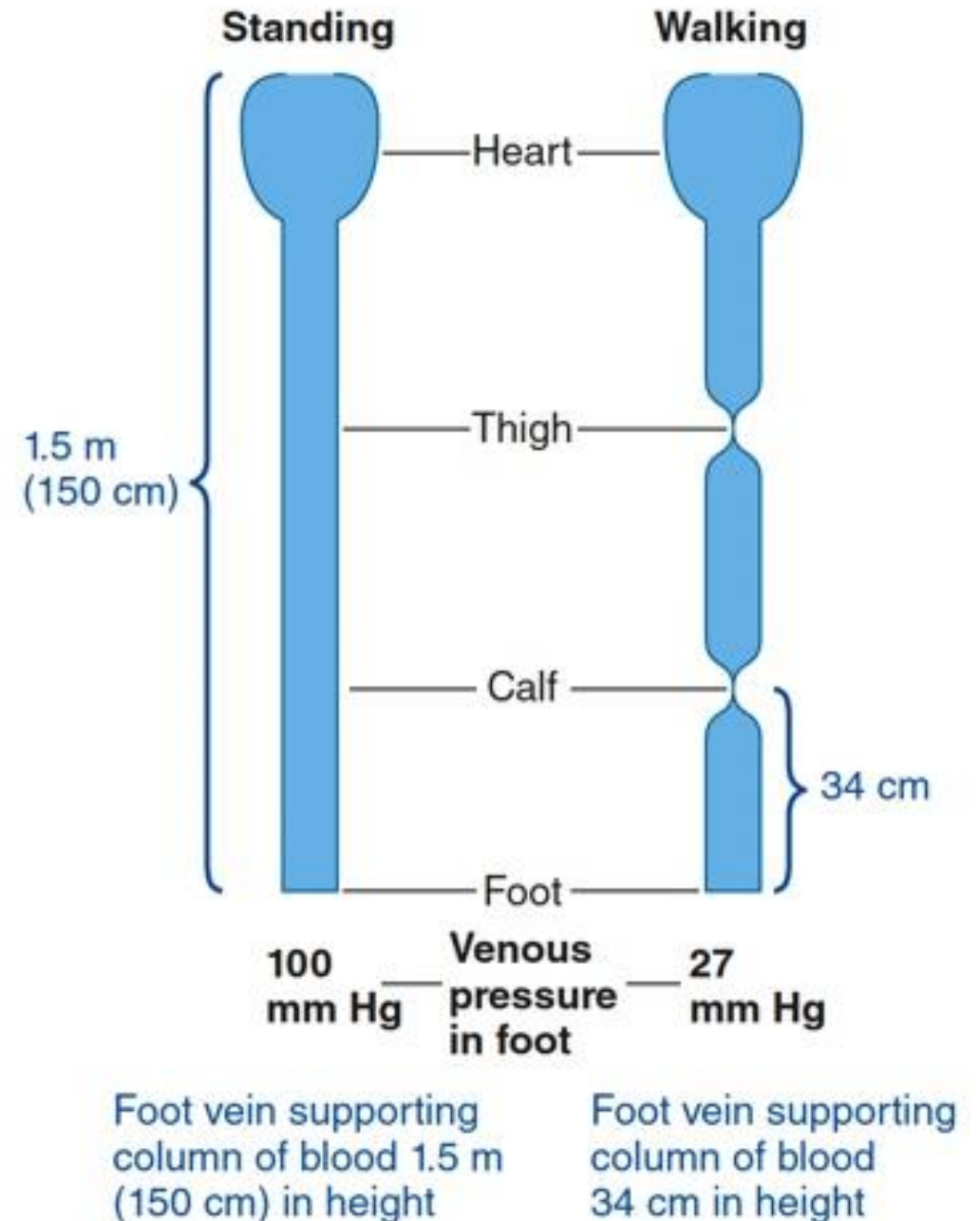
- Arteriolar vasoconstriction immediately reduces flow through these vessels because of their increased resistance (less blood can enter and flow through a narrowed arteriole), whereas venous vasoconstriction immediately increases flow through these vessels because of their decreased capacity (narrowing of veins squeezes out more of the blood already in the veins, increasing blood flow through these vessels).
- In addition to mobilizing the stored blood, venous vasoconstriction sustains increased venous return. With the filling capacity of the veins reduced, less blood draining from the capillaries remains in the veins but continues to flow instead toward the heart.

Venous valves

- the valves in the veins are arranged so that the direction of venous blood flow can only be toward the heart.
- Consequently, every time a person moves the legs or even tenses the leg muscles, a certain amount of venous blood is propelled toward the heart.
- This pumping system is known as the venous pump or muscle pump, and it is efficient enough that under ordinary circumstances, the venous pressure in the feet of a walking adult remains less than +20 mm Hg.



- Most of veins are located below the level of the heart, So they have to overcome the effect of gravity in order to bring the blood back to the heart. This will be hard, especially for the lower parts of the body, because, as you know, Hydrostatic pressure depends on the weight of the blood in that column, so the higher the weight, the more the pressure in the vein. For example: in the foot, the pressure is about 90 mmHg, less in the calf, thigh...etc. so as you go up (closer to the level of the heart) the pressure will be lower, But this increase of pressure will increase the capacitance of the veins by distending the wall more.



Venous valves

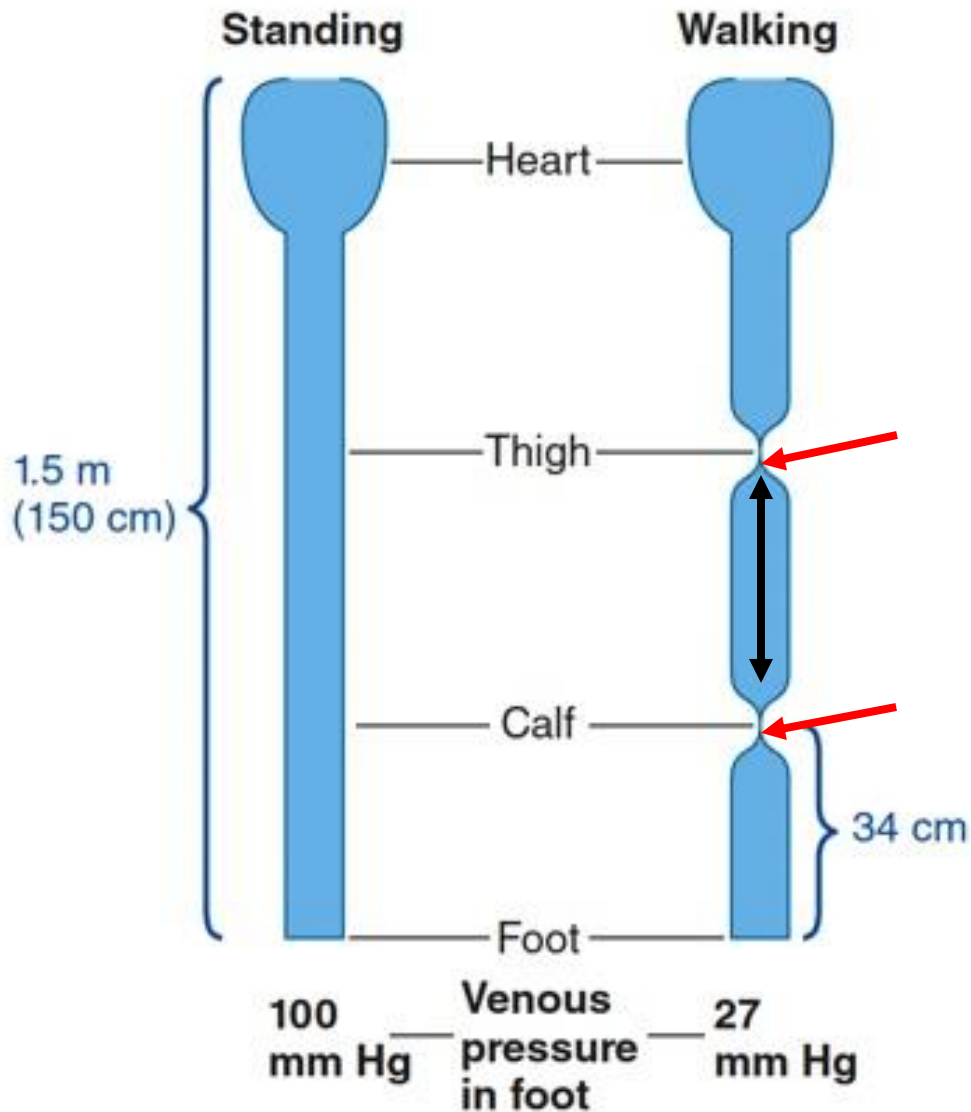
- If a person stands perfectly still, the venous pump does not work, and the venous pressures in the lower legs increase to the full gravitational value of 90 mm Hg in about 30 seconds.
- If you are standing for a long time, you are keeping distending the wall of vein, this kind of distension causes pulling of the valves, so they become incompetent, more blood will back flow, increasing the pressure down in the lower extremities causing edema.
- The pressures in the capillaries also increase greatly, causing fluid to leak from the circulatory system into the tissue spaces.
- As a result, the legs swell, and the blood volume diminishes; 10% to 20% of the blood volume can be lost from the circulatory system within the 15 to 30 minutes of standing absolutely still.

Varicose veins

- Stretching the veins increases their cross-sectional areas, but the leaflets of the valves do not increase in size.
- Therefore, the leaflets of the valves no longer close completely. With this lack of complete closure, the pressure in the veins of the legs increases greatly because of failure of the venous pump, which further increases the sizes of the veins and finally destroys the function of the valves entirely.
- Thus, the person develops what are called varicose veins, which are characterized by large bulbous protrusions of the veins beneath the skin of the entire leg, particularly the lower leg.
- Also, Genetic predisposition plays a role in development of varicose veins.

Varicose veins

- Whenever people with varicose veins stand for more than a few minutes, the venous and capillary pressures become very high, and leakage of fluid from the capillaries causes constant edema in the legs.
- The edema, in turn, prevents adequate diffusion of nutritional materials from the capillaries to the muscle and skin cells, so the muscles become painful and weak, and the skin may even become gangrenous and ulcerate, also this stagnation of blood may increase the risk of clotting.
- The best treatment for such a condition is continual elevation of the legs to a level at least as high as the heart. So the solution is to walk or keep stretching and contracting the skeletal muscles (especially the lower limb).
- Tight binders or long compression stockings on the legs also can be of considerable assistance in preventing the edema and its sequelae.



Foot vein supporting column of blood 1.5 m (150 cm) in height

Foot vein supporting column of blood 34 cm in height

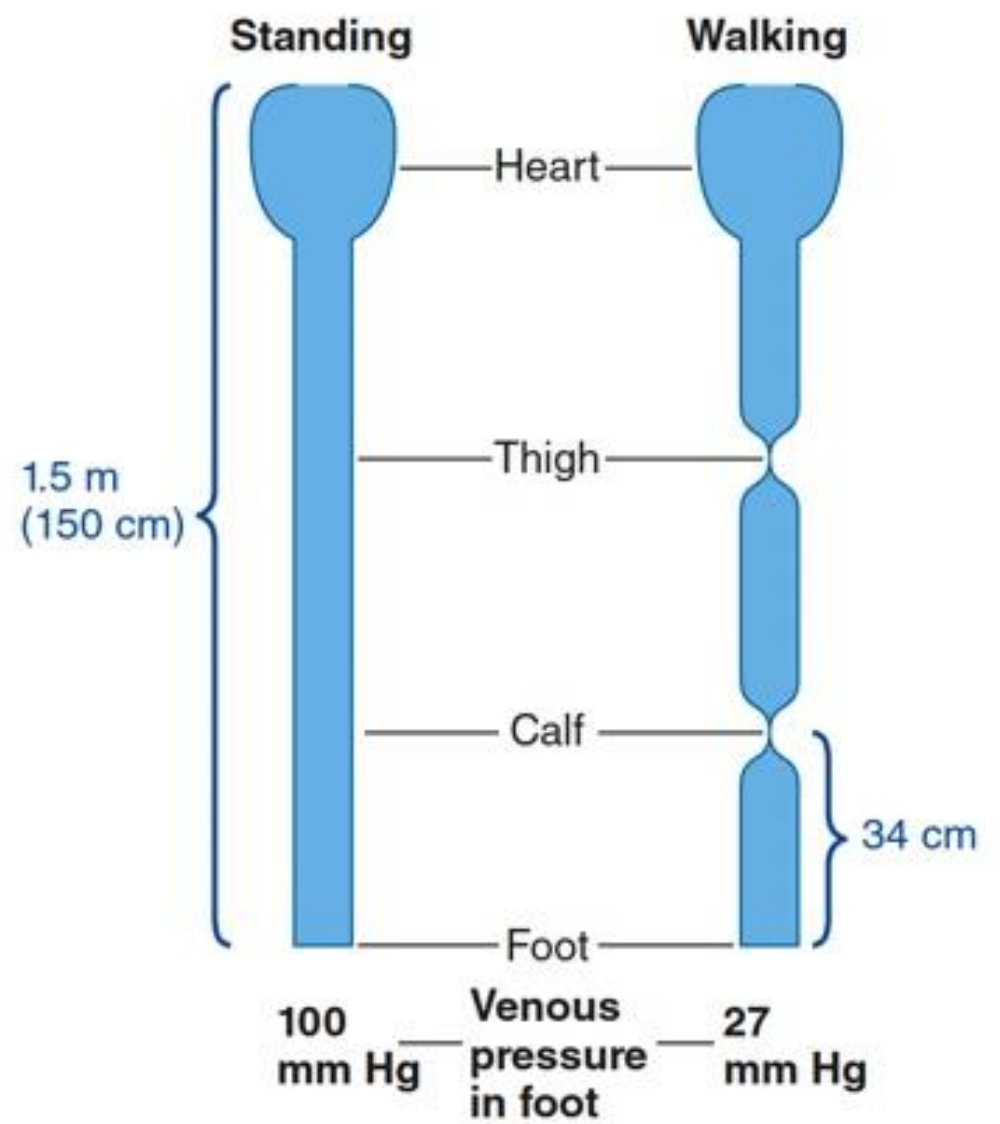
- The solution of edema is to walk or keep contracting the muscles of the lower limbs. When these muscles are contracting, they help squeeze the vein at different points (the red arrows), by this the height of the blood column decreases (the black arrow). This decrease of column height causes decrease in the Hydrostatic Pressure.
- When you are squeezing a tube from the middle, you are forcing the fluid to move in both directions, but this does not happen in the veins, Why? **Because of the presence of one way valve.**

Factors enhancing venous return

- In addition to the driving pressure imparted by cardiac contraction, five other factors enhance venous return: sympathetically induced venous vasoconstriction, skeletal muscle pump, venous valves, respiratory pump, and cardiac suction.
- respiratory pump, and cardiac suction act as a suction of blood.

Skeletal muscle pump

- When a person stands up, however, gravitational effects are not uniform. In addition to the usual pressure from cardiac contraction, vessels below heart level are subject to pressure from the weight of the column of blood extending from the heart to the level of the vessel.
- This increased pressure has two major consequences. First, the distensible veins yield under the increased hydrostatic pressure, further expanding so that their capacity is increased (increased distention of the wall). Even though the arteries are subject to the same gravitational effects, they are not nearly as distensible and do not expand like the veins. Much of the blood entering from the capillaries tends to pool in the expanded lower-leg veins instead of returning to the heart. Because venous return is reduced, CO decreases and the effective circulating volume shrinks. Second, the marked increase in PC (**capillary hydrostatic pressure**) resulting from the effect of gravity causes excessive fluid to filter out of capillary beds in the lower extremities, producing localized edema.



Foot vein supporting column of blood 1.5 m (150 cm) in height

Foot vein supporting column of blood 34 cm in height

Skeletal muscle pump

- Two compensatory measures normally counteract these gravitational effects.
- First, the resultant fall in MAP that occurs when a person moves from a lying-down to an upright position triggers sympathetically induced venous vasoconstriction, which drives some of the pooled blood forward.
- Second, when a person moves around, the skeletal muscle pump “interrupts” the column of blood by completely emptying given vein segments intermittently so that a particular portion of a vein is not subjected to the weight of the entire venous column from the heart to that portion’s level

Respiratory pump

- As a result of respiratory activity, the pressure within the chest cavity (**intrathoracic pressure**) averages 5 mm Hg less than atmospheric pressure. As the venous system returns blood to the heart from the lower regions of the body, it travels through the chest cavity, where it is exposed to this subatmospheric pressure. Because the venous system in the limbs and abdomen is subject to normal atmospheric pressure, an externally applied pressure gradient exists between the lower veins (at atmospheric pressure) and the chest veins (at less than atmospheric pressure). This pressure difference pushes blood from the lower veins to the chest veins (**from the area of high pressure to the area of low pressure**), promoting increased venous return. This mechanism of facilitating venous return is called the respiratory pump because it results from respiratory activity. Increased respiratory activity, the skeletal muscle pump, and venous vasoconstriction, all enhance venous return during exercise.

Cardiac suction effect

- The extent of cardiac filling does not depend entirely on factors affecting the veins. The heart plays a role in its filling. Remember that the VR will go to the Right atrium, then to the Right ventricle, During ventricular contraction, the AV valves are drawn downward (and that will increase the intra atrial volume), enlarging the atrial cavities. As a result, atrial pressure transiently drops below 0 mm Hg, thus increasing the vein-to-atria pressure gradient so that venous return is enhanced. In addition, rapid expansion of the ventricular chambers during ventricular relaxation creates a transient negative pressure in the ventricles so that blood is “sucked in” from the atria and veins—that is, the negative ventricular pressure increases the vein-to-atria-to-ventricle pressure gradient, further enhancing venous return. Thus, the heart functions as a “suction pump” to facilitate cardiac filling.

Please watch this video to understand the factors that enhance the VR:

<https://youtu.be/tgkB2B3oGV8?si=AAULz7REjk8ggqDuW>

And this video to understand the varicose veins:

<https://youtu.be/fpiyGin5nGk?si=n7oLPY0WQKDLsLYI>

Venous return

- **factors** that can increase this venous return and thereby increase the right atrial pressure are as follows: (1) increased blood volume; (2) increased large vessel tone throughout the body with resultant increased peripheral venous pressures (venoconstriction); and (3) dilation of the arterioles, which decreases the peripheral resistance and allows rapid flow of blood from the arteries into the veins.

Remember that venoconstriction → decrease in the diameter of the veins → decreasing the capacitance → increasing the pressure inside the veins → more venous return to the right atrium.

- Remember that constriction of the arterioles will decrease the blood flow to the veins → less venous return.

All the factors affecting the venous return will also affect the right atrial pressure.

↑ Venous return → ↑ CVP

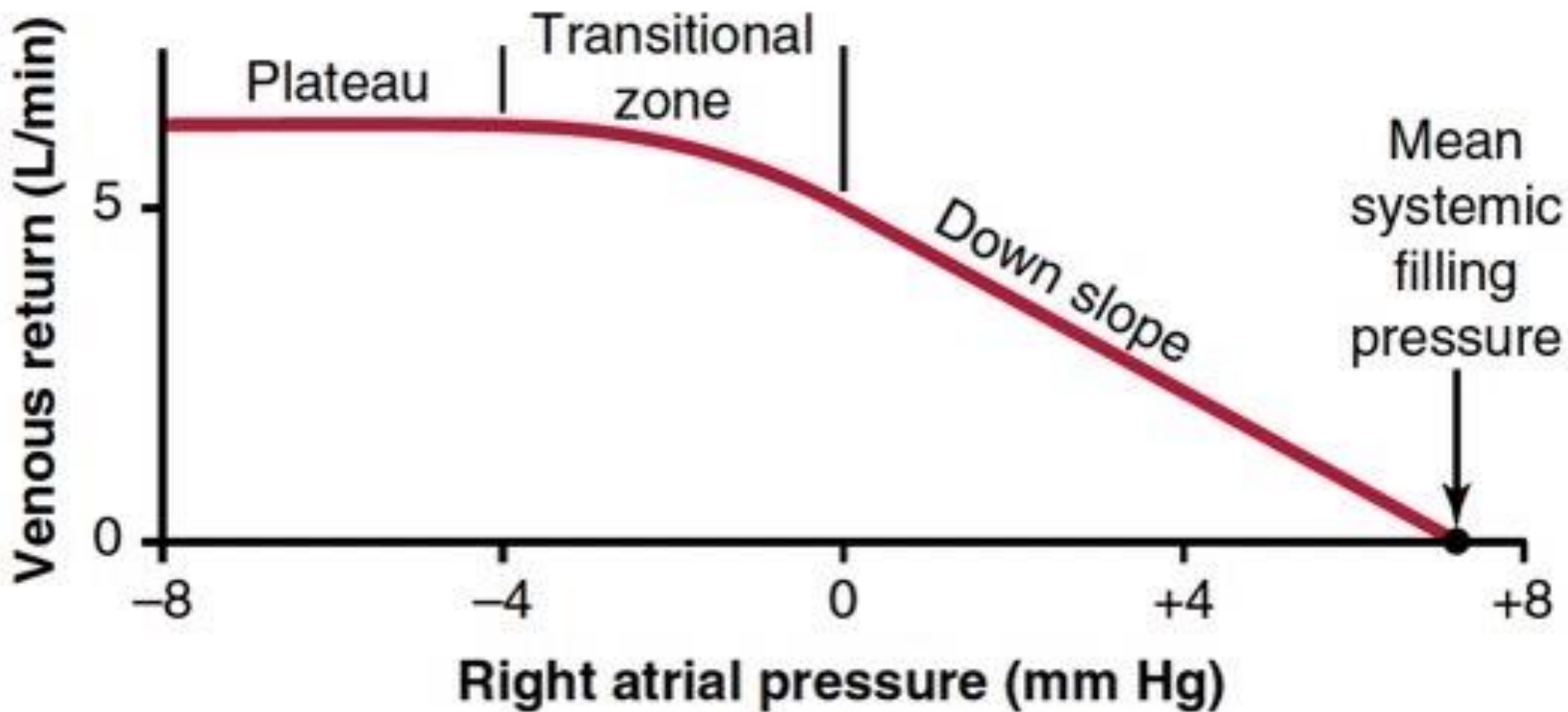
Right atrial pressure

Central Venous Pressure (CVP)

- Veins are capable of constricting and enlarging and thereby storing small or large quantities of blood and making this blood available when required by the remainder of the circulation.
- Blood from all the systemic veins flows into the right atrium of the heart. That's why it is called Central Venous pressure (central collection point).
- Therefore, the pressure in the right atrium is called the central venous pressure. The normal right atrial pressure is about **0 mm Hg**, which is equal to the atmospheric pressure around the body.
- Right atrial pressure is regulated by a balance between (1) the ability of the heart to pump blood out of the right atrium and ventricle into the lungs (contractility of the heart) and (2) the tendency for blood to flow from the peripheral veins into the right atrium (venous return).
- The stronger the contractility of the heart, the more emptying of the right atrium, which leads to decrease in the right atrial pressure → higher pressure gradient.

$$VR = \frac{\Delta P}{R} = \frac{MSFP - RAP (CVP)}{RVR}$$

- MSFP: Mean systemic filling pressure. / RVR: resistance to venous return.
- What is MSFP? Imagine the heart stopped pumping for some time → there is **no blood flow** → there is no pressure gradient → the pressure almost everywhere in the circulation should be **equal!** this blood volume pressure that is filling the circulation is called MSFP. (the average pressure of the blood when it's not moving).
- It is normally around 7 mmHg.
- It reflects the venous pressure more because two thirds of the circulation blood volume is within the venous side.
- mean systematic filing pressure is different than mean circulatory filling pressure, the circulatory one will also include the pulmonary circulation.



- This curve (the previous slide) shows how the venous return (VR) is affected by changes in the right atrial pressure (RAP), this curve is called venous function curve (VFC) or venous return curve.
- Let's start with the point 0 RAP which is the normal RAP, if we increased the RAP (positive pressure) → it is a backward force against the venous return → we will expect a decrease in the VR.
- If we keep increasing the RAP, we will reach a point where there is no venous return → no blood flow! This point is called MSFP (mean systemic filling pressure). (this happens in the x intersection point).
- If we decreased the RAP (negative pressure) → we will expect increase in the VR because of the increased ΔP , right? **This is not actually the case!** There will be some increase in the transitional phase followed by a plateau! Why? Decreasing RAP to this negativity will cause **collapse** in the veins, and if we combine both ($\uparrow \Delta P$ + the collapse), this will result in a plateau.

Venous return curve

- three principal factors that affect venous return to the heart from the systemic circulation:
 - 1. Right atrial pressure, which exerts a backward force on the veins to impede flow of blood from the veins into the right atrium.
 - 2. Degree of filling of the systemic circulation (measured by the mean systemic filling pressure), which forces the systemic blood toward the heart (this is the pressure measured everywhere in the systemic circulation when all flow of blood is stopped, discussed in detail later).
 - 3. Resistance to blood flow between the peripheral vessels and the right atrium.
- These factors can all be expressed quantitatively by the venous return curve

Vascular function curve or venous return curve

- The inverse relationship between venous return and right atrial pressure is explained as follows:
- Venous return back to the heart, like all blood flow, is driven by a pressure gradient.
- The lower the pressure in the right atrium, the higher the pressure gradient between the systemic arteries and the right atrium and the greater the venous return.
- Thus as right atrial pressure increases, this pressure gradient decreases and venous return also decreases.

Vascular function curve

- The knee (flat portion) of the vascular function curve occurs at negative values of right atrial pressure.
- At such negative values, the veins collapse, impeding blood flow back to the heart. Although the pressure gradient has increased (i.e., as right atrial pressure becomes negative), venous return levels off because the veins have collapsed, creating a resistance to blood flow.
- The slope of the vascular function curve is determined by total peripheral resistance (TPR).

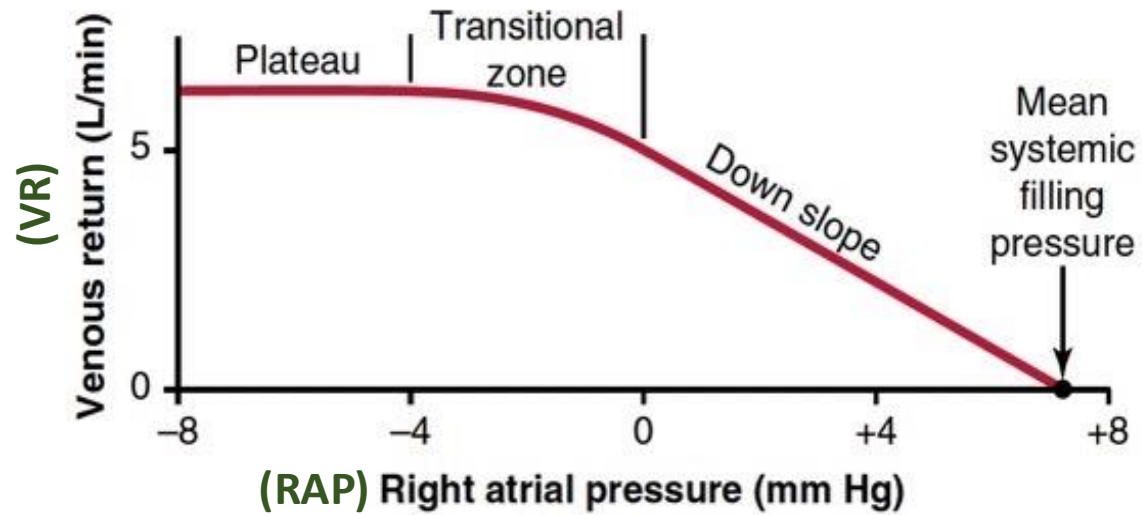
Mean systemic filling pressure

- Two factors influence the value for mean systemic pressure: (1) the blood volume and (2) the distribution of blood between the unstressed volume and the stressed volume.
- In turn, the value for mean systemic pressure determines the intersection point (zero flow) of the vascular function curve with the X-axis.
- increased blood volume and decreased compliance of the veins produce an increase in mean systemic pressure and shift the vascular function curve to the right. Decreased blood volume and increased compliance of the veins produce a decrease in mean systemic pressure and shift the vascular function curve to the left.

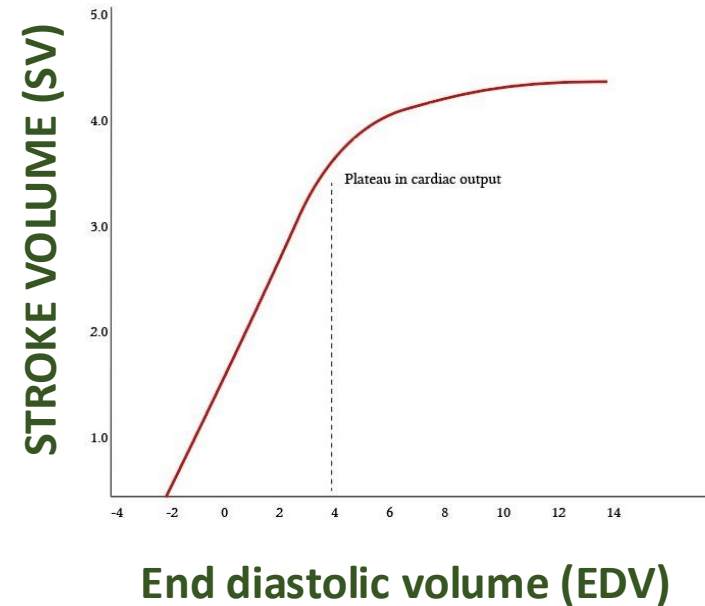
Combining cardiac and vascular function curves

- In the steady state, cardiac output and venous return are, by definition, equal at the point of intersection.
- Combining these curves provides a useful tool for predicting the changes in cardiac output that will occur when various cardiovascular parameters are altered.
- the system will move to a new steady state.

Venous return curve



Cardiac output curve

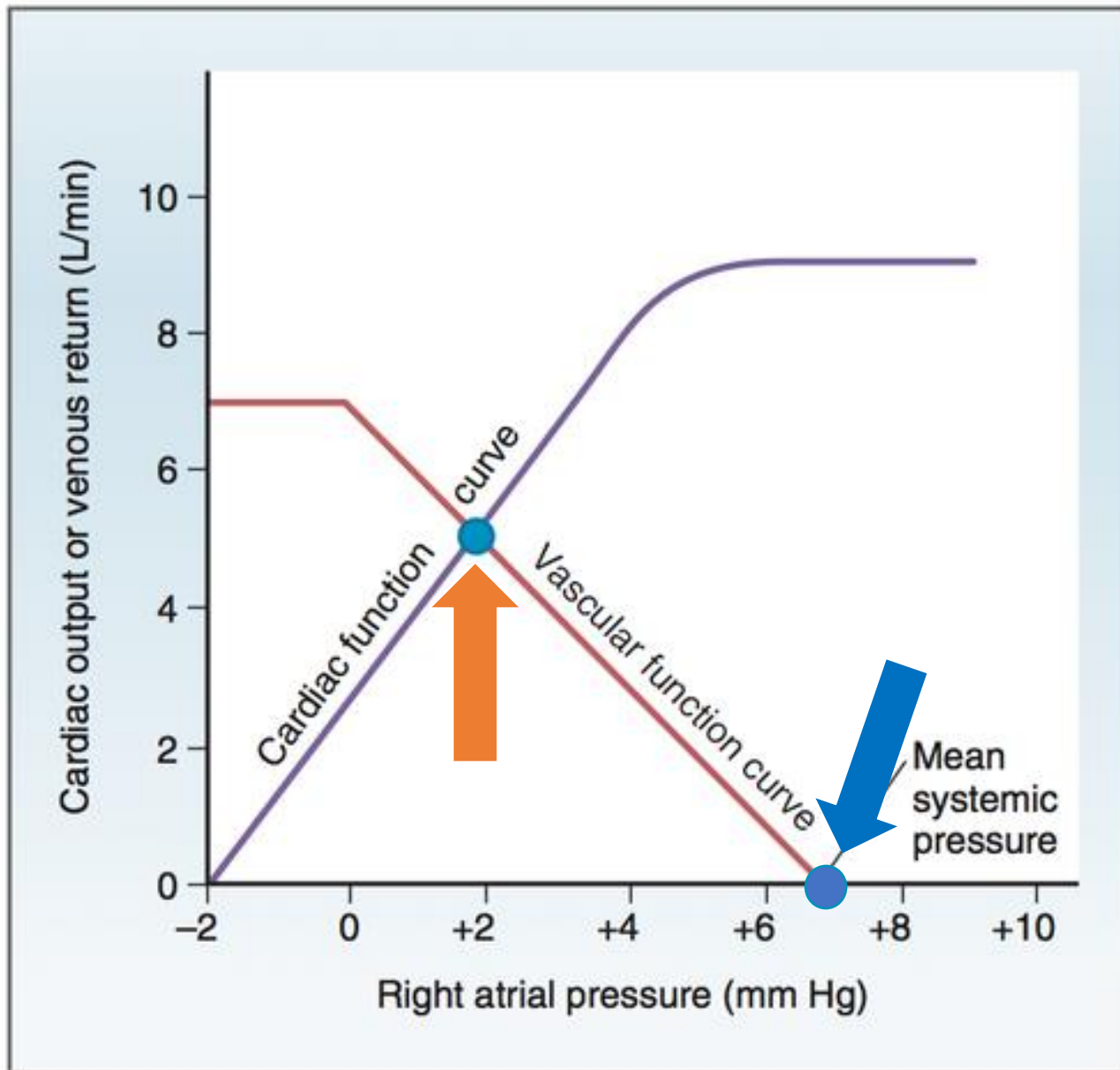


Above there is two curves: the **venous return curve** and the **cardiac output curve**.

Remember: end diastolic volume (EDV): The volume of blood in the ventricle at the end of diastole.

Stroke volume (SV): The amount of blood pumped out of each ventricle with each contraction.

- EDV reflects the RAP, so on the cardiac output curve we can replace the (EDV → RAP) and replace (stroke volume → cardiac output).
- We can combine both curves together in one curve.
- Venous return curve represents vascular curve, cardiac output curve represents cardiac curve.



- The orange arrow represent **equilibrium point (steady state point)**, which is the point where the venous return curve intersects the cardiac output curve (the venous return = cardiac output).
- The blue arrow represent the **MSFP (mean systemic filing point)**.

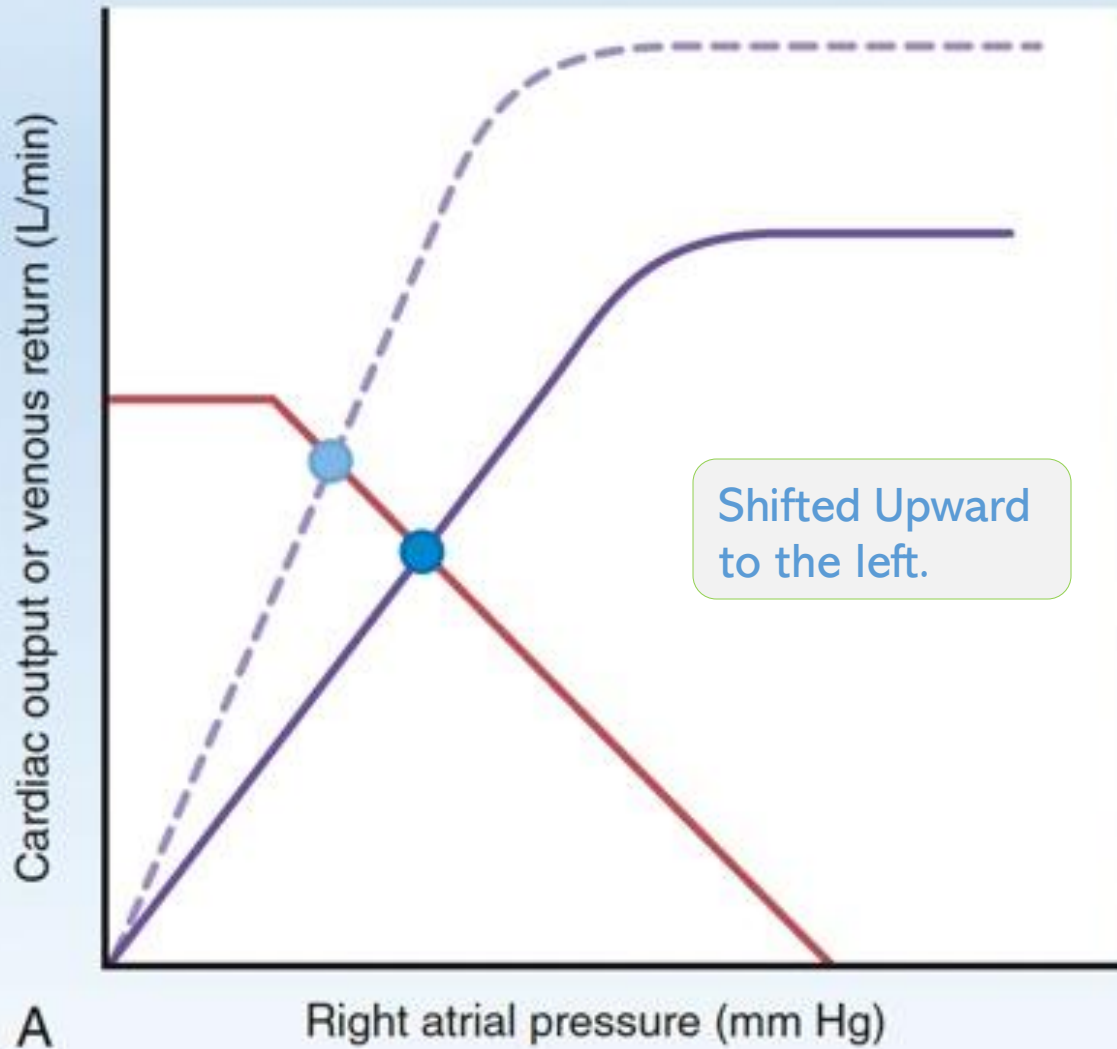
Any change in the circulation will affect this curve differently, we will see now the effect of three different factors:

- 1) inotropic agents
- 2) Blood volume changes
- 3) TPR (total Peripheral resistance)

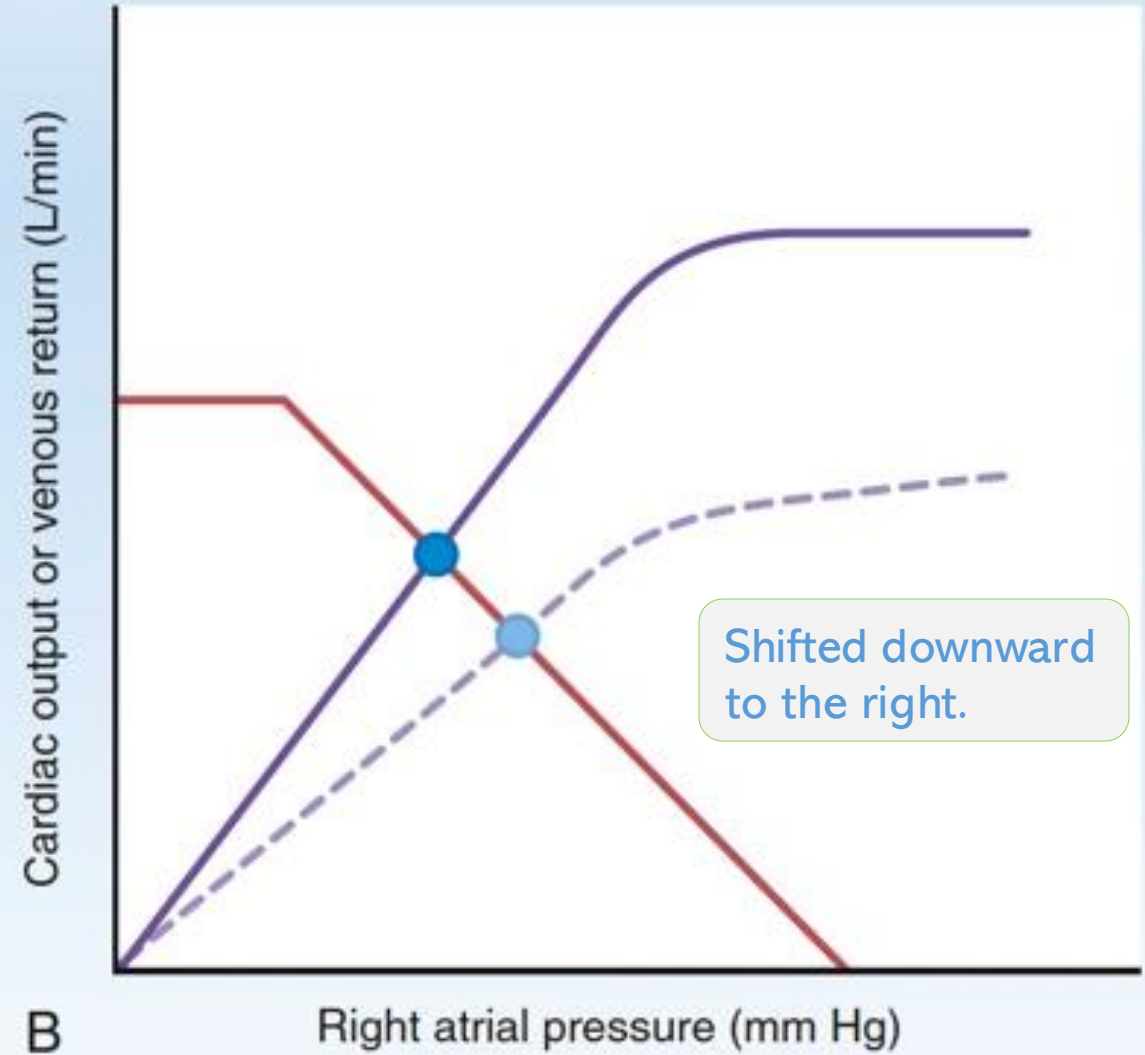
Inotropic effect

- Positive inotropic agents **such as digitalis** produce an increase in contractility, an increase in stroke volume, and an increase in cardiac output for any level of right atrial pressure.
- Thus the cardiac function curve shifts upward, but the vascular function curve is unaffected.
- The point of intersection (the steady state point) of the two curves now has shifted upward and to the left.
- In the new steady state, cardiac output is increased and right atrial pressure is decreased.
- The decrease in right atrial pressure reflects the fact that more blood is ejected from the heart on each beat as a result of the increased contractility and increased stroke volume.
- **affect only cardiac curve.**
- Negative inotropic agents will make downward shift to the cardiac curve, lower cardiac output, But higher RAP.

Positive inotropic effect



Negative inotropic effect



Higher CO, but lower RAP



Equilibrium point
(steady state point)

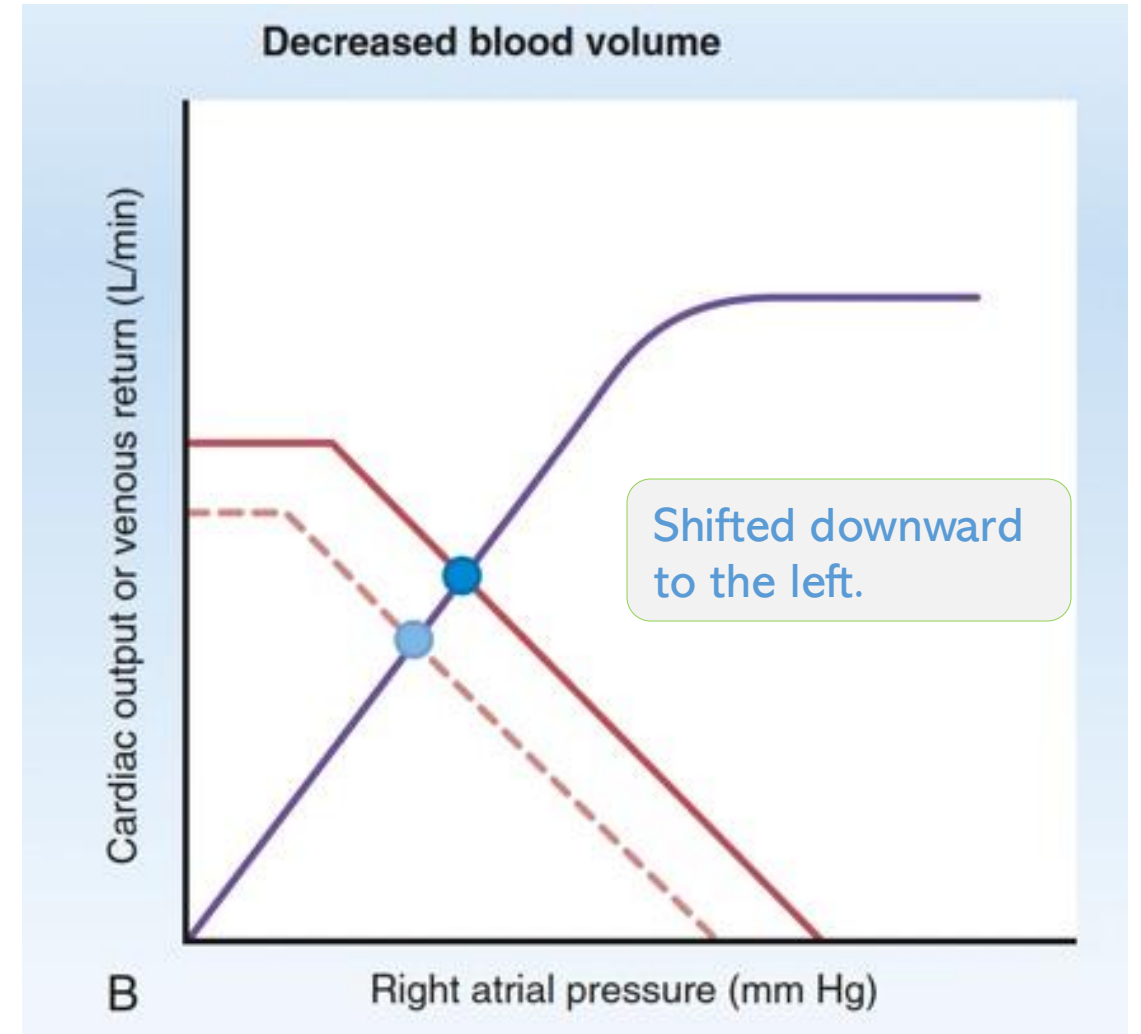
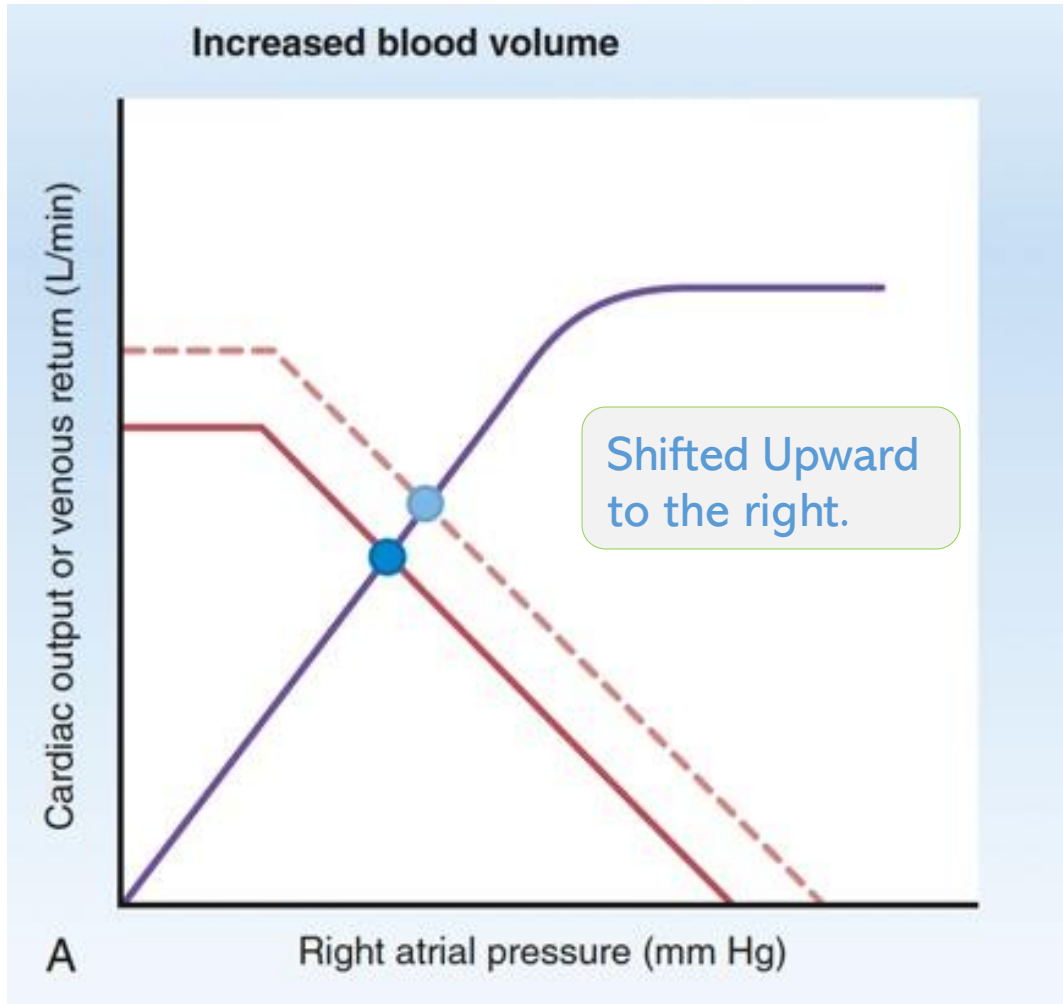


lower CO, but higher RAP

Blood volume effect

- Increases in blood volume increase the amount of blood in the stressed volume and therefore increase the mean systemic pressure (MSFP).
- Mean systemic pressure is the point on the vascular function curve where venous return is zero.
- Increases in blood volume shift this intersection point to the right and therefore shift the curve to the right in a parallel manner. (The shift is parallel because there is no accompanying change in TPR, which determines the slope of the vascular function curve.)
- In the new steady state, the cardiac and vascular function curves intersect at a new point at which cardiac output is increased and right atrial pressure is increased.
- **Affect only vascular curve.**
- Decrease in the blood volume will result in downward left shift in a parallel manner, with lower CO and RAP

the shift is in a parallel manner because there is no change in the TPR



Higher CO and RAP.

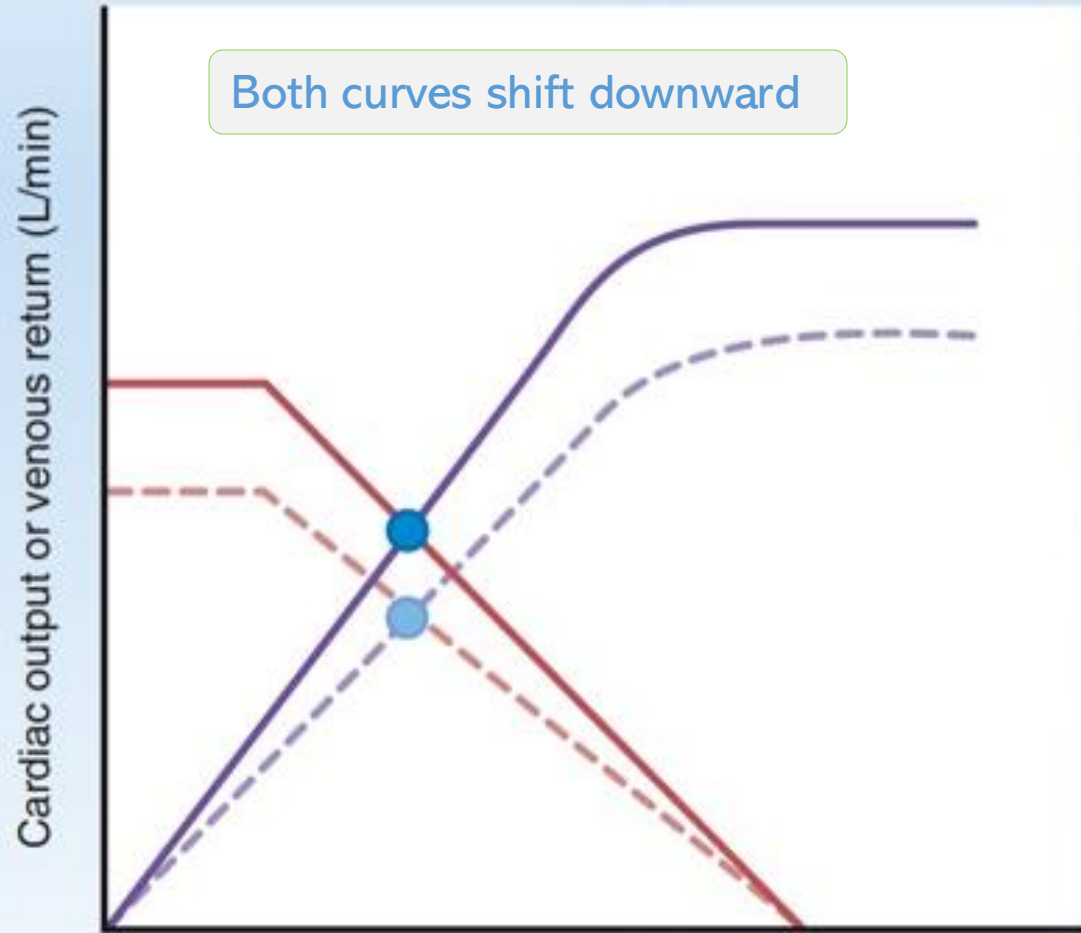
← Equilibrium point (steady state point) →

Lower CO and RAP.

TPR effect

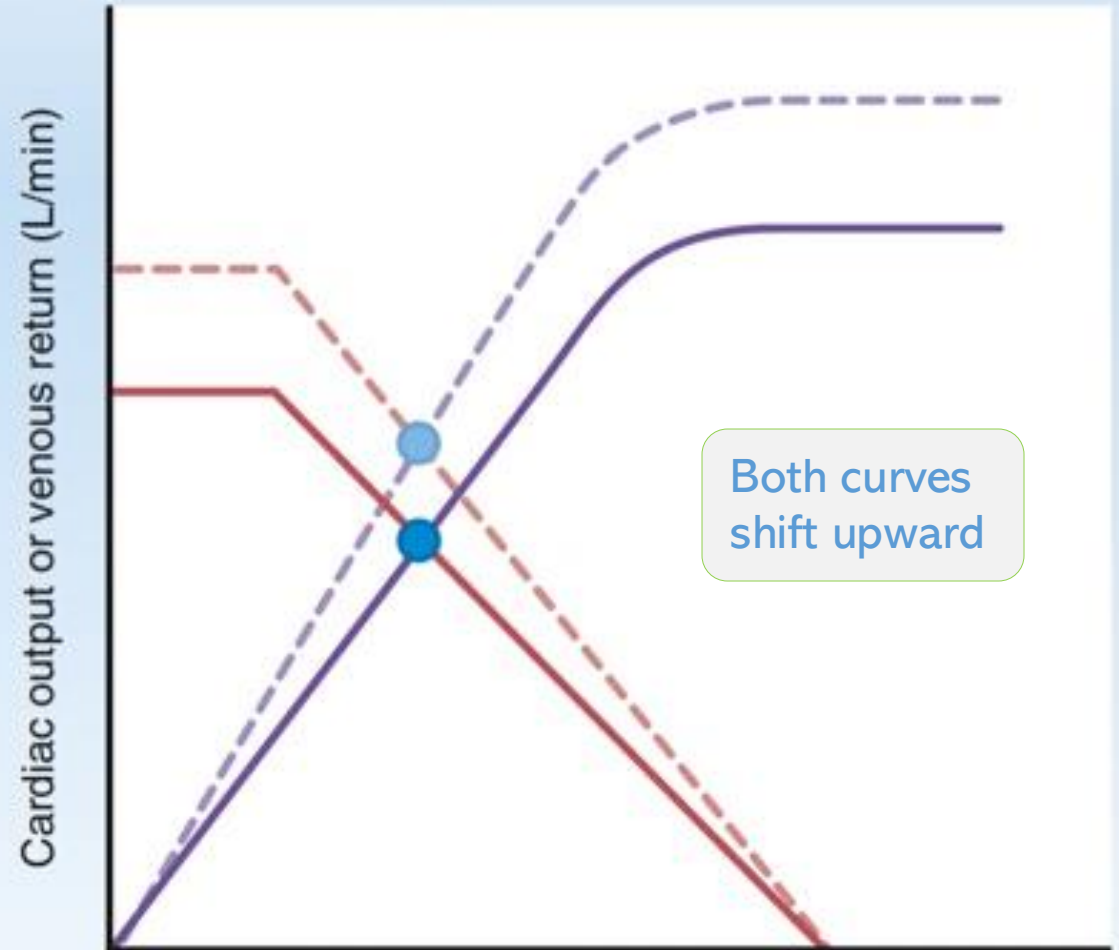
- Increases in TPR cause an increase in arterial pressure by “holding” blood in the arteries.
- This increase in arterial pressure produces an increase in afterload on the heart, which decreases cardiac output.
- The cardiac function curve shifts downward as a result of the increased afterload.
- This means that less blood returns to the heart for a given right atrial pressure—venous return is decreased. (shifts downward)
- The curves intersect at a new steady state point at which both cardiac output and venous return are decreased.
- **affect both cardiac and vascular curves.**
- Decrease in TPR → less afterload → more cardiac output → upward shift in CO curve, and also it will increase venous return which will shift the venous function curve upwards.

Increased TPR



Both curves shift downward

Decreased TPR



Both curves shift upward

Lower CO / VR, no change in RAP



Equilibrium point
(steady state point)



higher CO / VR, no change in RAP

RAP is almost the same, but this is not always the case!

Finally, the doctor said that these curves are a lot more complicated than this because CO, VR and RAP are not independent factors, they are interdependent, so it is much more complicated than this, but for now this is enough.

Thank you

فِي بَضْعِ سِنِينَ قَلِيلٍ لِلَّهِ الْأَمْرُ مِنْ قَبْلُ وَمِنْ بَعْدُ وَيَوْمَئِذٍ
يَفْرَحُ الْمُؤْمِنُونَ ﴿٤﴾

اللهم لك الحمد كما ينبغي لجلال
وجهك وعظيم سلطانك

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2→V3			



امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!