

Venous return & factors affecting it

Editing File

Objectives:



Discuss functions of the veins as blood reservoirs.



Know the pressure variations in systemic blood vessels.



Define venous return, mean circulatory filling pressure and right atrial pressure.



Describe measurement of central venous pressure (CVP) and state its physiological and clinical significance.



Describe vascular and cardiac function curves under physiological and pathophysiological conditions.



State determinants of venous return and explain how they influence it.



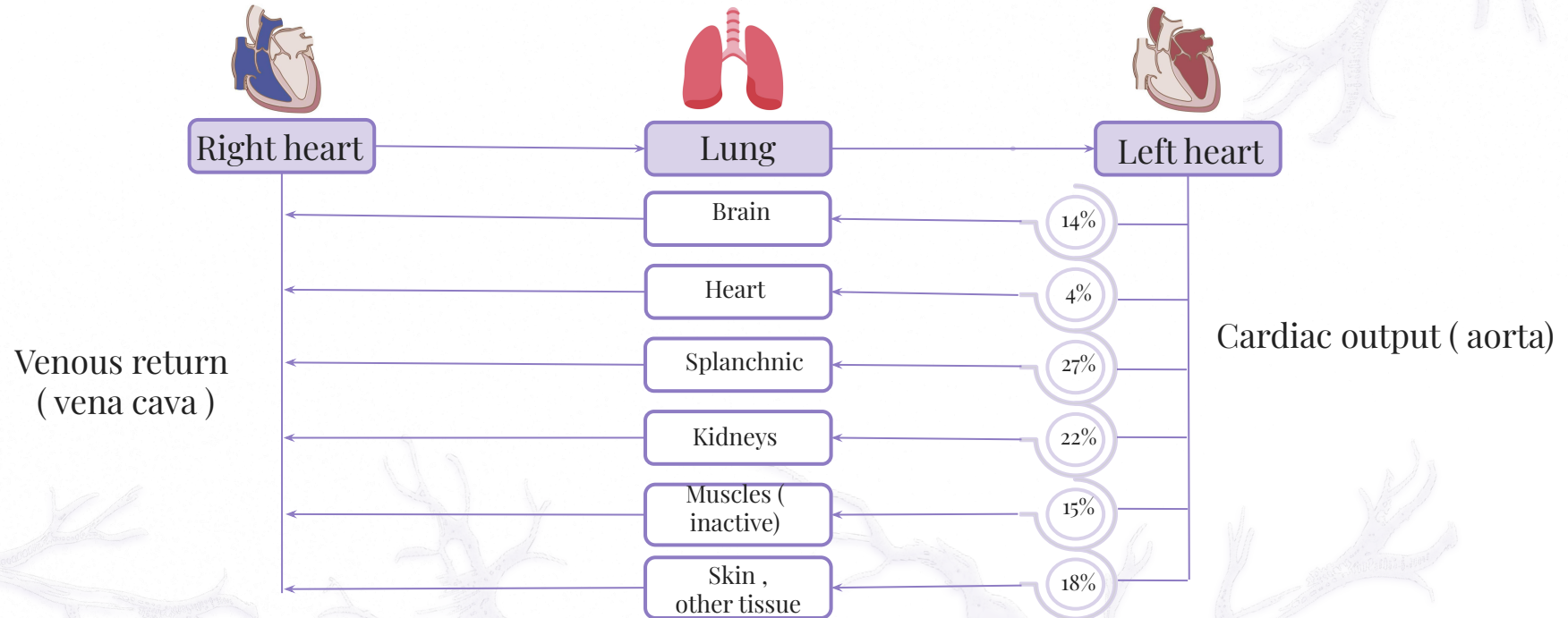
Explain the effect of gravity on venous pressure and explain pathophysiology of varicose veins.



Recommended video by Alila Medical Media Notes

Principle

Cardiac output = Total Tissue blood flow



the percentages is not important

About veins

1

Veins hold most of blood in body (60-70%).

2

They are called capacitance vessels

3

They have thin walls & stretch easily to accommodate more blood without increased pressure (= higher compliance)

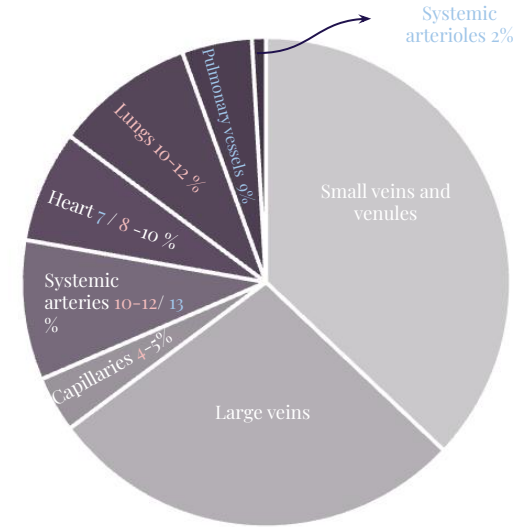
4

They have only 0-10 mm Hg pressure

Systemic veins
60/64 - 70 %

Female's dr : the percentages is not important

Distribution of blood



- Small veins and venules
- Large veins
- Heart
- Lungs
- Capillaries
- Systemic arteries
- Pulmonary vessels
- Systemic arterioles

Veins Are Blood Reservoirs

1

When the body is at rest many of the capillaries are closed, the capacity of the venous reservoir increased as extra blood bypasses the capillaries and enters the veins » stretches the veins

2

When this extra volume of blood stretches the veins » increase their total cross sectional area » blood moves forward through the veins more slowly. Therefore, blood spends more time in the veins.

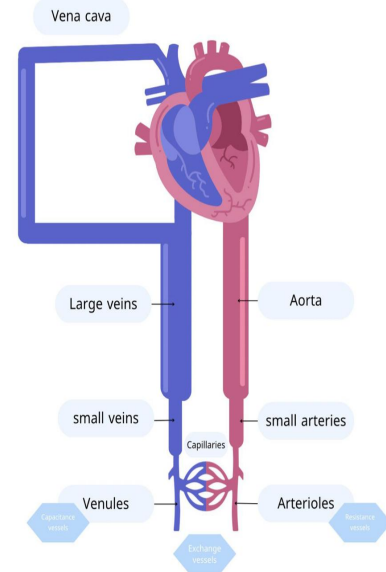
3

When the stored blood is needed, during exercise, **extrinsic factors** reduce the capacity of the venous reservoir and drive the extra blood from the veins to the heart so that it can be pumped to the tissues.



سُبْحَانَ رَبِّكَ رَبِّ الْعَزَّةِ

Thanks to Lama 443



Made by : Lama

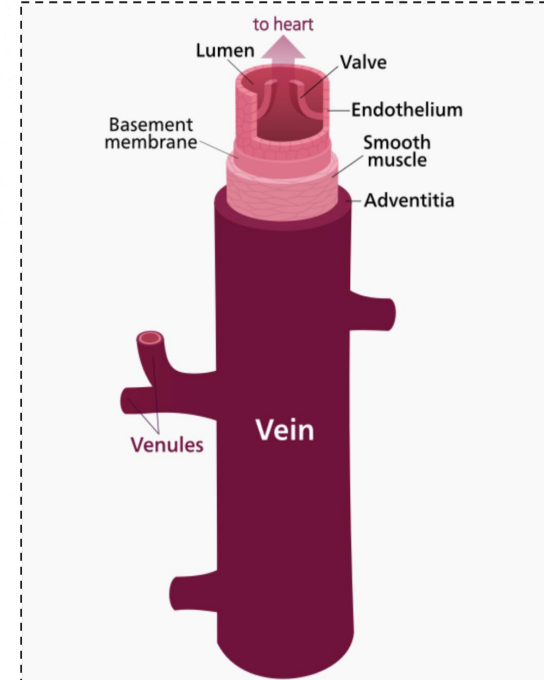


Structures of veins

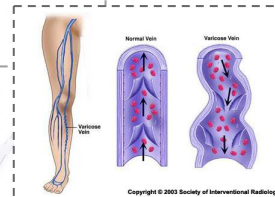


Varicose veins

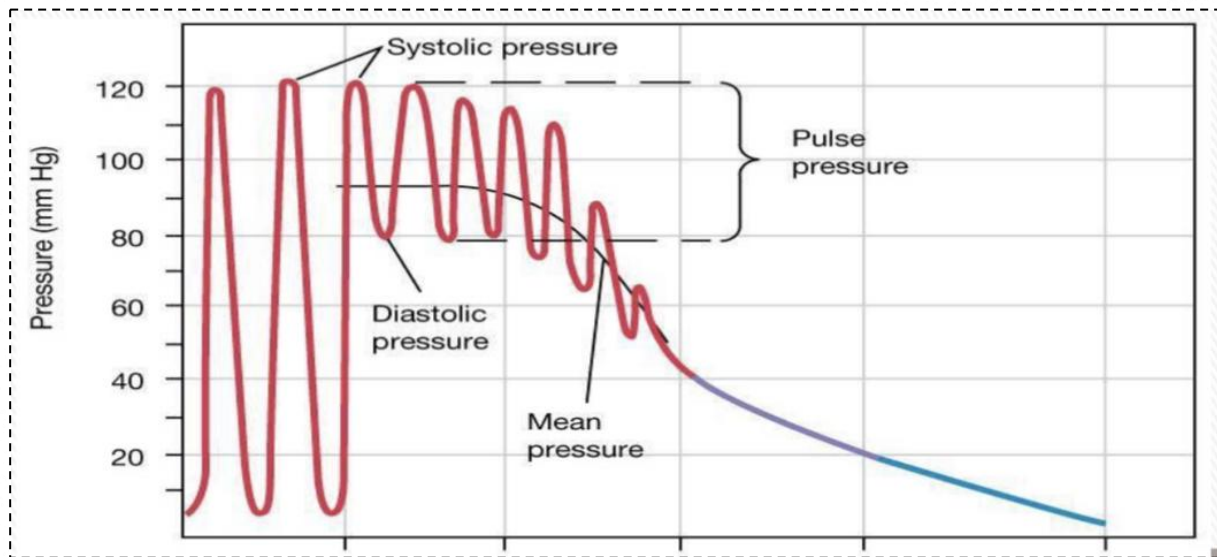
| | |
|--------|---|
| Layers | All 3 layers are present, but thinner than in arteries of corresponding size (external diameter). |
| Valves | Veins in lower extremities have paired semilunar, bicuspid valves to restrict backflow. |



In varicose veins, blood pools because valves fail causing venous walls to expand.



Pressure variation in systemic blood vessels



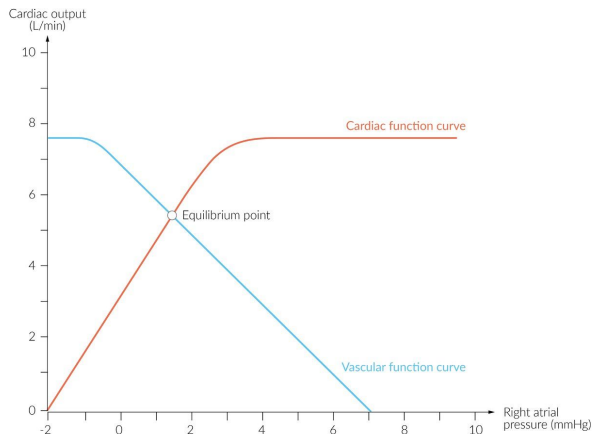
| Left ventricle | Arteries | Arterioles | Capillaries | Venules veins | Right atrium |
|--|---|--|--|---|--------------|
| Pulsatile pressure, high systolic and low diastolic pressure (never 0) | Pulsatile pressure, high systolic pressure and higher diastolic then left ventricle | Pulsatile pressure , lower systolic pressure and lower diastolic then arteries | Notice the highest pressure drop happens within arterioles. (resistance vessels) | No pulsatile pressure and the pressure keeps decreasing | Around zero |

Summary and explanation for the first half of the lecture (9-20)

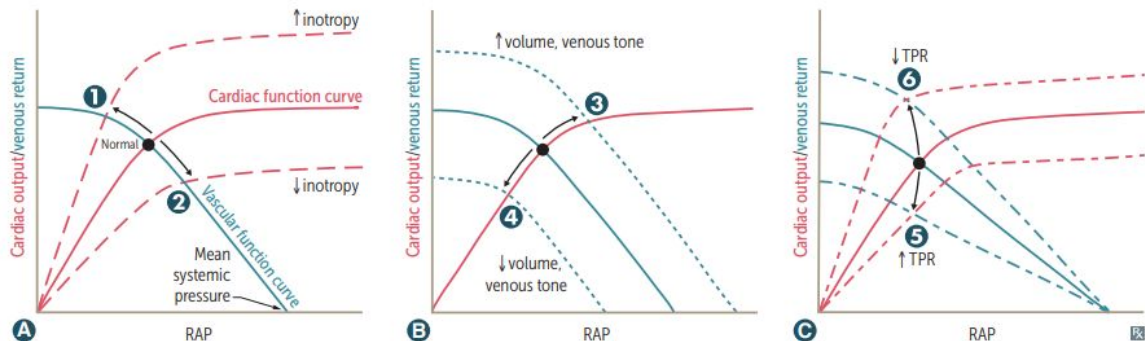
If you understand it inshallah you won't need to read the slides, only skim them.



Very nice explanation By: Khalid Alohal
And THX to him for this amazing slide!!
والشرح موجود بنسختين در ايف ويوتيوب !!



Cardiac and vascular function curves



Intersection of curves = operating point of heart (ie, venous return and CO are equal, as circulatory system is a closed system).

| GRAPH | EFFECT | EXAMPLES |
|--------------------------------------|---|---|
| A Inotropy | Changes in contractility → altered SV → altered CO/VR and RA pressure (RAP) | <ol style="list-style-type: none"> 1 Catecholamines, dobutamine, milrinone, digoxin, exercise ⊕ 2 HF with reduced EF, narcotic overdose, sympathetic inhibition ⊖ |
| B Venous return | Changes in circulating volume → altered RAP → altered SV → change in CO | <ol style="list-style-type: none"> 3 Fluid infusion, sympathetic activity, arteriovenous shunt ⊕ 4 Acute hemorrhage, spinal anesthesia ⊖ |
| C Total peripheral resistance | Changes in TPR → altered CO Change in RAP unpredictable | <ol style="list-style-type: none"> 5 Vasopressors ⊕ 6 Exercise, arteriovenous shunt ⊖ |

Changes often occur in tandem, and may be reinforcing (eg, exercise ↑ inotropy and ↓ TPR to maximize CO) or compensatory (eg, HF ↓ inotropy → fluid retention to ↑ preload to maintain CO).

Venous return

1

Venous return (VR) is the flow of blood from the periphery to the heart (Rt atrium)

2

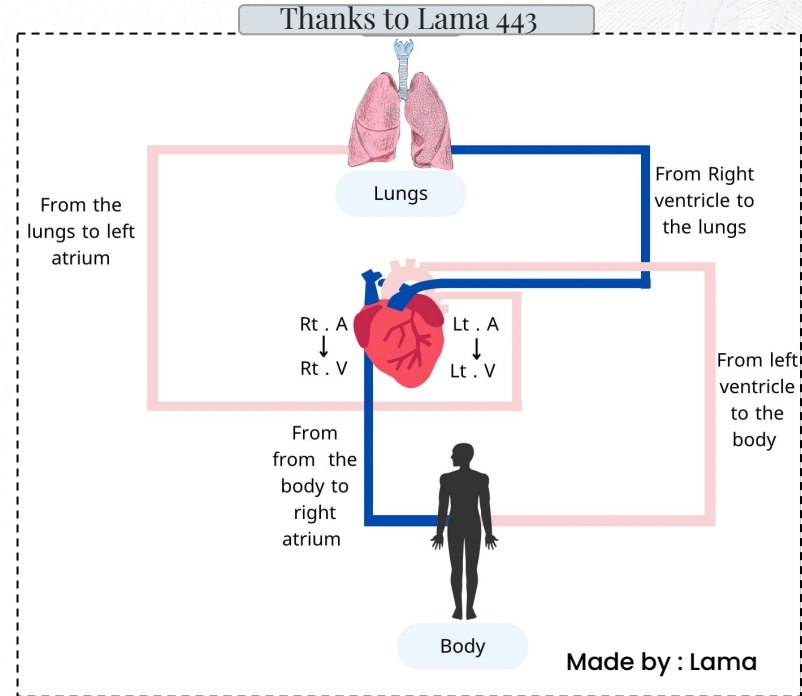
Normally VR must equal CO when averaged over time because the CVS is essentially a closed loop. Otherwise, blood would accumulate in either the systemic or pulmonary circulations.

3

Venous return is determined by the difference between the venous pressure nearest to the tissues (mean systemic filling pressure (Psf) , mean circulatory pressure(MCP)) and the central venous pressure nearest to the heart (CVP).



$$VR = MCP - CVP$$



CVP & MCP

When heart pumping is stopped by shocking the heart with electricity to cause ventricular fibrillation or is stopped in any other way, flow of blood everywhere in the circulation ceases a few seconds later. Without blood flow, the pressures everywhere in the circulation become equal, this equilibrated pressure level is called the mean circulatory filling pressure.

Venous Return

Central Venous Pressure (CVP)

CVP: is the pressure in the right atrium and the big veins of thorax (right atrial pressure (RAP)= JVP).

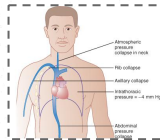
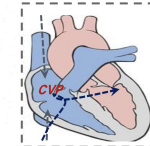
CVP is measured with a catheter inserted in SVC (superior vena cava).

The normal range of the CVP = 0 - 4 mmHg. increased with hypervolemia, decreased with hypovolemia

It is the force responsible for cardiac filling.

CVP is used clinically to assess hypovolemia and during IV transfusion to avoid volume overloading.

CVP is raised in right-sided heart failure.



Mean Circulatory Pressure (MCP)

MCP: It is the pressure nearest to the tissues, and is about 7 mmHg.

The value for right atrial pressure at which venous return is zero is called the mean systemic filling pressure. **IT IS AFFECTED BY:**

Blood volume: it is directly proportional to blood volume.

Venous capacity: it is inversely proportional to the venous capacity.

↑ Blood volume → ↑ MCP

Venoconstriction → ↑ MCP

↓ Blood volume → ↓ MCP

Venodilation → ↓ MCP

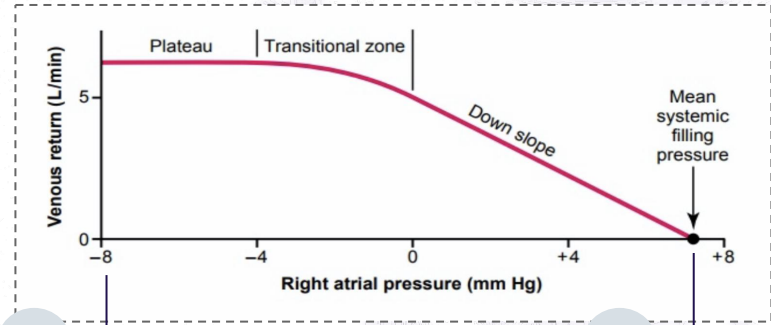
The Venous Return Curve

(The Vascular Function Curve)

1 The curve relates VR To Right Atrial Pressure (RAP)

2 Mean systemic filling pressure (Psf) is the point at which the vascular function curve intersects the X-axis, (i.e VR is zero and RAP is at its highest value, Psf = 7 mm Hg).

1 The curve depends on the difference between MCP and CVP

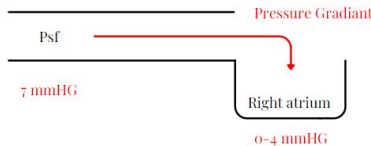


2 If the pressure is highly negative then a compression force causes the veins to close, so the venous return stop increasing

3 The Right atrial pressure increase causes decrease in venous return until it reaches a point where MCP-CVP = zero

Extra :

When the pressure in Right atrium rises to equal the Psf, there is no longer any Pressure difference Between peripheral vessels and the right atrium.



The Venous Return Curve

(The Vascular Function Curve)

****Possible SAQ!

1

There is an inverse relationship between venous return and right atrial pressure (RAP).

2

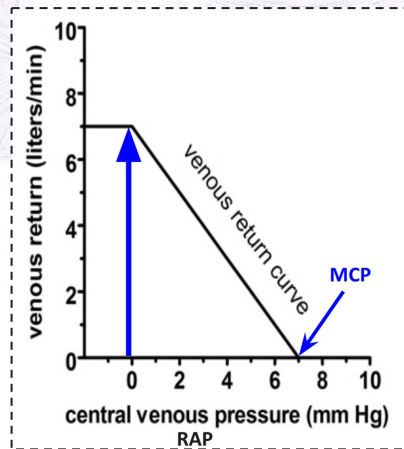
Venous return back to the heart, like all blood flow, is driven by a pressure gradient. The lower the pressure in the right atrium, the higher the pressure gradient the greater the venous return.

3

Thus as RAP increases, this pressure gradient decreases and venous return also decreases.

4

The knee (flat portion) of the vascular function curve occurs at negative values of RAP. At such negative values, the veins collapse & impedes VR in spite of high pressure gradient.



Cardiac Function Curve

1

The curve relates CO (pumping of blood by the heart) To Right Atrial Pressure (RAP).

2

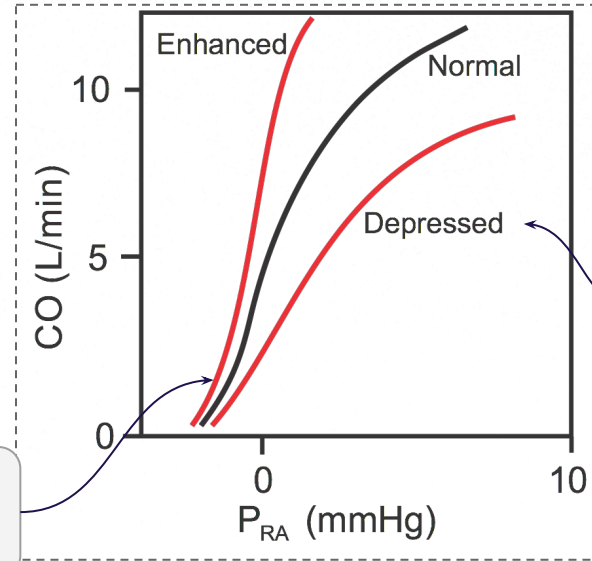
When the mean RAP is about 0 mmHg, the CO in an adult is about 5L/min.

3

Normally, Rt atrial pressure (RAP) fluctuates with atrial contraction and respiration.



Example : Inspiration → ↓ RAP → ↑ VR



Not frank-Starling curve but similar to it.

The Frank-Starling curve is named after the pioneering physiologists Otto Frank and Ernest Starling, who first described the relationship between ventricular filling and stroke volume in the heart. The term "cardiac function curve" is a more general term that is used to describe any curve or graph that represents the relationship between cardiac function and some other physiological parameter, such as heart rate, blood pressure, or cardiac output.

Effect of RAP Changes On Function Curve

Because of the steepness of the cardiac function curve, very small changes in RAP (just a few mmHg), can lead to large changes in cardiac output.

Factors That Cause a Hypereffective Heart:

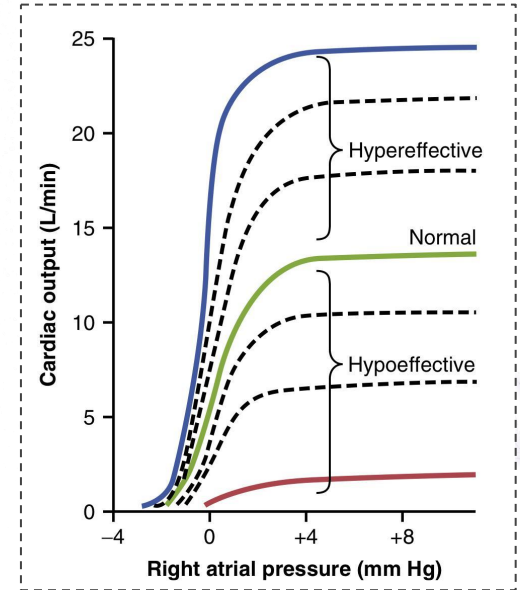
Two general types of factors that can make the heart a stronger pump than normal which are nervous stimulation and hypertrophy of the heart muscle.

Factors That Cause a Hypoeffective Heart:

Any factor that decreases the heart's ability to pump blood causes hypoeffectivity. Some of the factors that can decrease the heart's ability to pump blood are the following:

- 1- Increased arterial pressure against which the heart must pump, such as in severe hypertension
- 2- Inhibition of nervous excitation of the heart
- 3- Pathological factors that cause abnormal heart rhythm or rate of heartbeat
- 4- Coronary artery blockage, causing a heart attack

- 5- Valvular heart disease
- 6- Congenital heart disease
- 7- Myocarditis, an inflammation of the heart muscle
- 8- Cardiac hypoxia



Cardiac Function Curve & Vascular Curve

Important slide

Female's slide

Cardiac Function

The cardiac function curve or cardiac output curve, shown in **Figure 4.26**, is based on the **Frank-Starling relationship** for the left ventricle. The cardiac function curve is a plot of the relationship between cardiac output of the left ventricle and right atrial pressure.

Again, recall that right atrial pressure is related to venous return, end-diastolic volume, and end-diastolic fiber length: As venous return increases, right atrial pressure increases, and end-diastolic volume and end-diastolic fiber length increase. Increases in end-diastolic fiber length produce increases in cardiac output. Thus in the steady state the volume of blood the left ventricle ejects as cardiac output equals or matches the volume it receives in venous return.

Increases in end-diastolic volume (i.e., right atrial pressure) produce increases in cardiac output by the Frank-Starling mechanism. However, this “matching” occurs only up to a point: When right atrial pressure reaches a value of approximately 4 mm Hg, cardiac output can no longer keep up with venous return and the cardiac function curve levels off. This maximum level of cardiac output is approximately 9 L/min.

Vascular Curve

The vascular function curve or venous return curve, shown in **Figure 4.26**, depicts the relationship between venous return and right atrial pressure. Venous return is blood flow through the systemic circulation and back to the right heart. 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.

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.

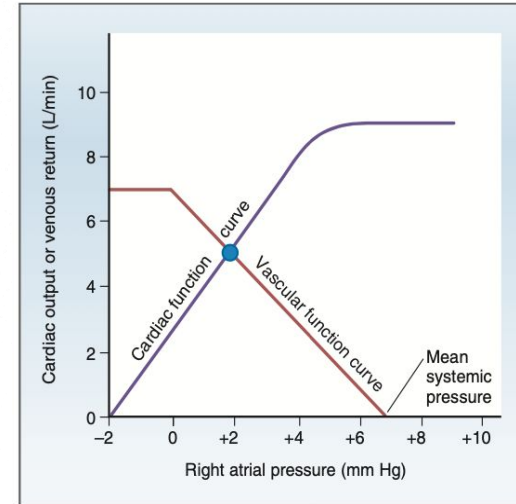
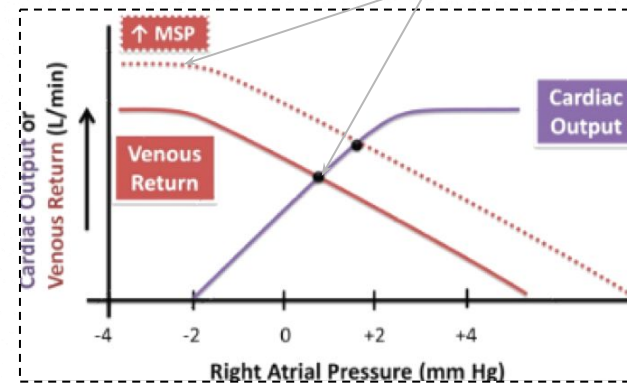


Fig. 4.26 Cardiac and vascular function curves. The cardiac function curve is cardiac output as a function of right atrial pressure. The vascular function curve is venous return as a function of right atrial pressure. The curves intersect at the steady state operating point (filled circle) where cardiac output and venous return are equal.

Combining cardiac and vascular function curves

Effects of changes in blood volume

Increase in blood volume increase mean systemic pressure (MSP) while Cardiac Function curve is not changed



1

Increases in blood volume as a result of transfusion of a large fluid volume into the circulation increase the amount of blood in the stressed volume and, therefore, increase the mean systemic pressure.

2

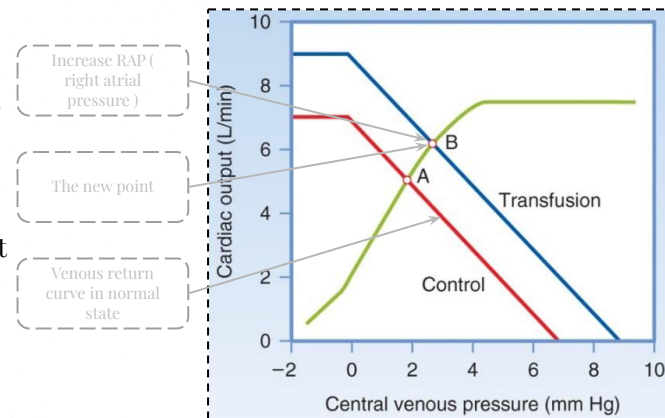
This results in shifting of the vascular function curve to the right and up

3

The cardiac function is not altered with changes in blood pressure) volume.

4

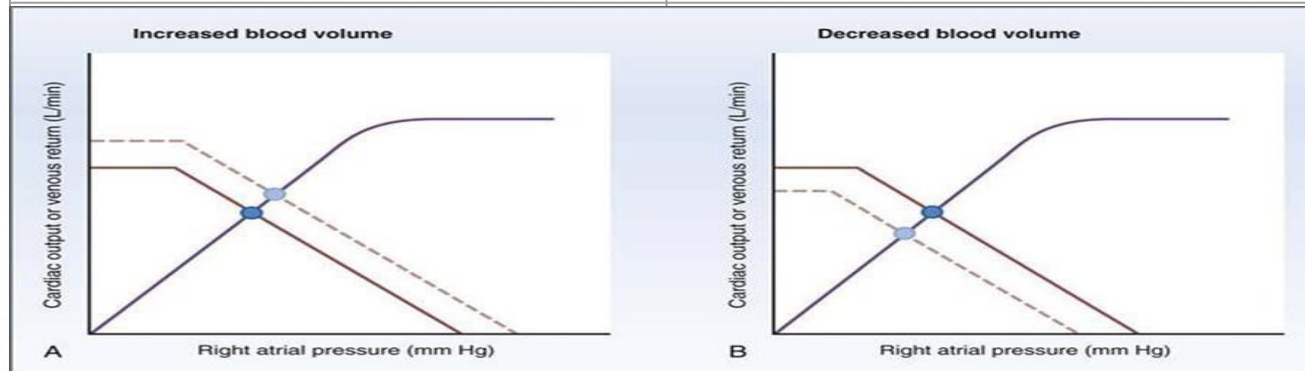
In the new steady state, the cardiac and vascular function curves intersect at a new point at which venous return and the cardiac output are increased. The RAP is increased.



Combining cardiac and vascular function curves

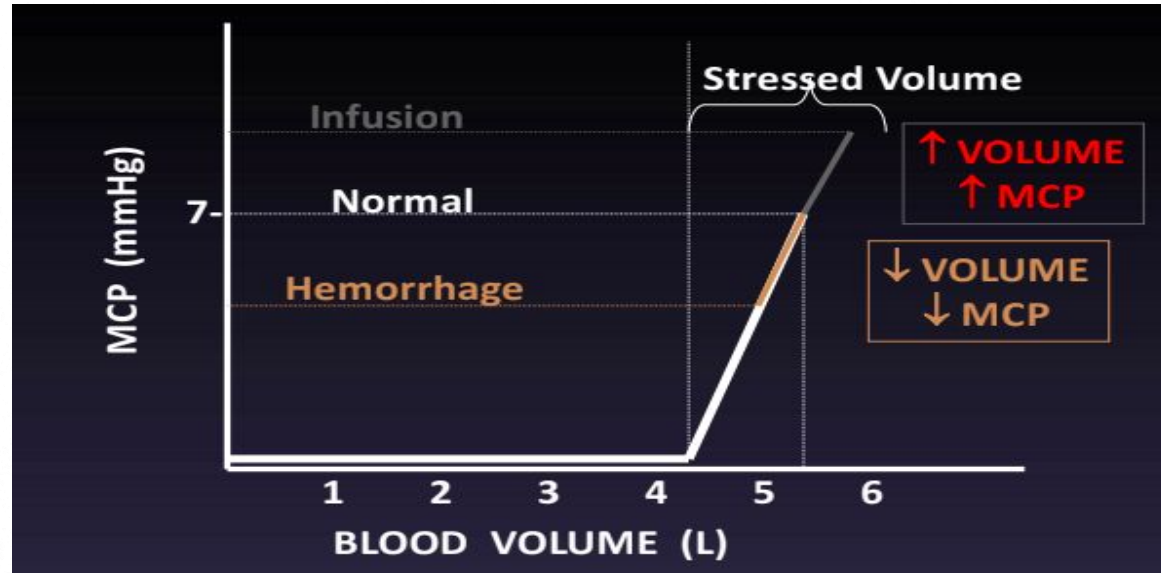
Blood volume effects

| Increase blood volume | Decrease blood volume |
|--|--|
| <p>E.g. transfusion, shifts the venous function curve up and to the right</p> <ul style="list-style-type: none"> ⤴ cardiac output ⤴ RAP ⤴ Mean systemic pressure | <p>E.g. hemorrhage, shifts the venous function curve down and to the left</p> <ul style="list-style-type: none"> ⤵ cardiac output ⤵ RAP ⤵ Mean systemic pressure |



Mean circulatory pressure; MCP

MCP is determined by blood volume and venous capacity



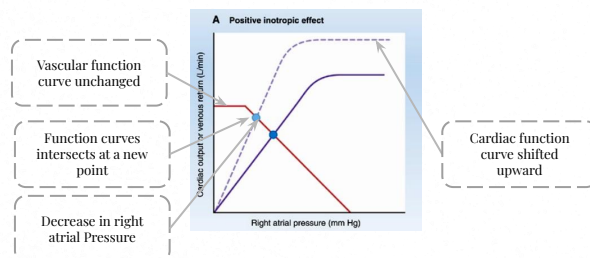
Combining cardiac and vascular function curves

Inotropic effects

Inotropic effects

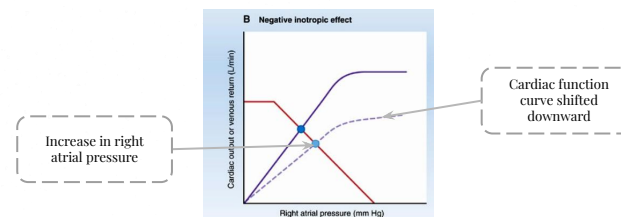
Positive Inotropic effects

- Positive inotropic agents produce an increase in contractility, an increase in stroke volume, and an increase in cardiac output for any level of RAP. **Thus, the cardiac function curve shifts upward (counter-clockwise), but the vascular function curve is unaffected.**
- Thus, there will be substantial increases in the cardiac output and venous return, while the RAP is decreased..



Negative Inotropic effects

- the opposite is true with negative inotropic agents.
- decrease contractility and stroke volume
- Decrease in cardiac output
- The cardiac function curve is shifted downward
- Increase in right atrial pressure



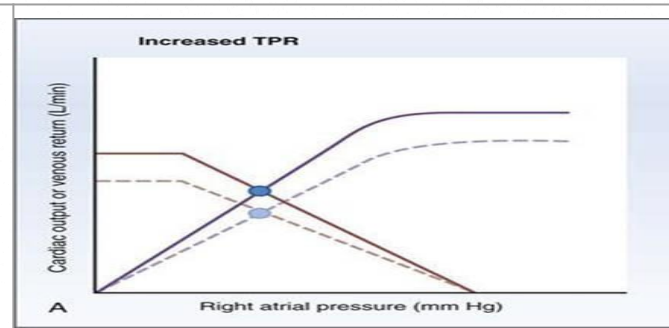
Combining cardiac and vascular function curves Effects of changes in TPR (Total peripheral resistance)

Changes in TPR alter both curves. The cardiac function curve changes because of a change in afterload (arterial blood pressure), and the vascular function curve changes because of a change in venous return.

Increase TPR

I.e. constriction of the arterioles

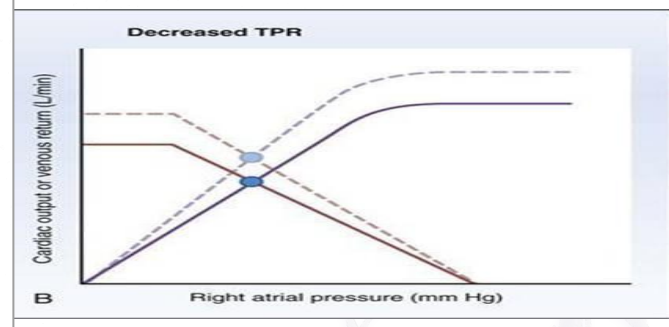
With increased TPR, there is a substantial decrease in the cardiac output and venous return with the almost no change in the RAP



Decrease TPR

I.e. dilation of the arterioles

The opposite is true when the TPR is decreased



Basic Principles

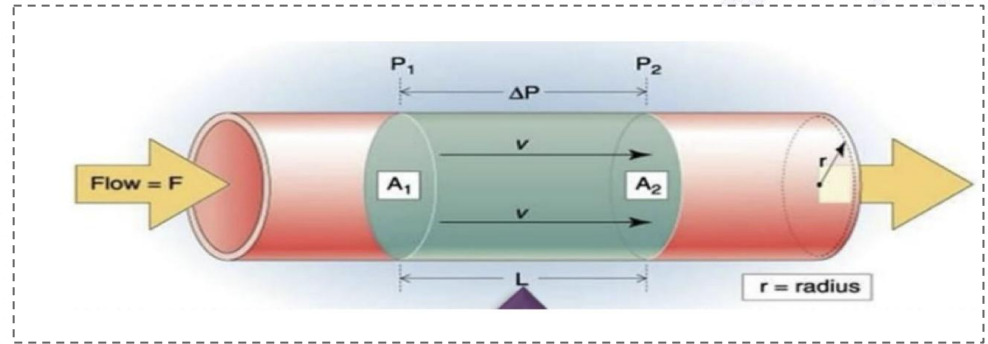
Flow of any fluid (blood) through a tube (vessel) depends on :

1

The pressure difference between the two ends (Pressure gradient), Blood flows from **high pressure to low pressure**

2

The resistance to blood flow through the vessel, Controlled by the diameter of the Vessel, and **blood viscosity**.



Determinants of venous return:

Summary for the last half of the lecture (23-34)



Nice summary
by Jood Aljufan



1 Pressure gradient

\pm RAP \rightarrow pressure gradient \rightarrow VR \rightarrow (inverse relationship between VR and RAP(CVP)).

\pm difference between (psf)+RAP \rightarrow VR
 \pm PsF \rightarrow (VR curve) shift upward right
 \pm Psf \rightarrow (VR curve) shift down left

2 Blood volume

Increase Blood volume shift vascular curve upwards and to the right
 Decrease BV shift the curve downwards and to the left.

note:At constant venous capacity

3 Venous capacity

Venous Capacity = volume in veins

\pm VC = venodilation = \pm MCP \rightarrow \pm VR, curve down and left.
 \pm VC=venoconstriction= \pm MCP \rightarrow \pm VR, curve upward to right

note :At a constant blood volume

4 Sympathetic activity

\pm (SNS) activity \rightarrow venoconstriction \rightarrow \pm VC \rightarrow modest \pm MCP \rightarrow \pm VR.

5 Total peripheral resistance

For a given RAP :

\pm TPR \rightarrow \pm VR

\pm TPR \rightarrow \pm VR

6 Skeletal muscles activity

Skeletal muscle contraction \rightarrow external venous compression \rightarrow \pm venous capacity \rightarrow \pm VR
 like pump

7 Venous valves

Increase VR (counteract gravity)

8 Respiratory activity (respiratory or thoracic pump)

Inspiration = increase VR

Less thoracic pressure and more abdominal pressure (diaphragm)

Pressure gradient moves upwards.

9 Effect of gravity

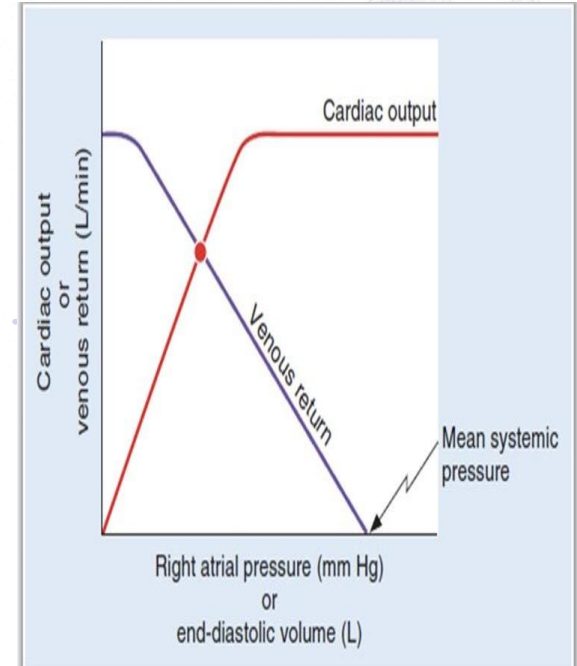
Less VR \rightarrow blood pooling down (orthostatic hypotension)

Determinants of venous return

1

Pressure gradient

1. VR back to the heart like all blood flow is driven by a pressure gradient. $VR = MCP - RAP$ (CVP)
2. There is an inverse relationship between VR and RAP (CVP).
3. The lower the RAP, the higher the pressure gradient and the greater the VR.
4. Thus as RAP \uparrow , pressure gradient \downarrow and VR also \downarrow .
5. When the RAP falls more below zero (at highly negative values of RAP, no further increase in VR and a plateau (the knee, flat portion) of the vascular function curve is reached.
6. At such negative values cause: collapse of the veins entering the chest. This impedes VR in spite of high pressure gradient.



Determinants of venous return

1

Pressure gradient (cont):

1. The greater the difference between the mean circulatory filling pressure (psf) and the RAP, the greater becomes the VR

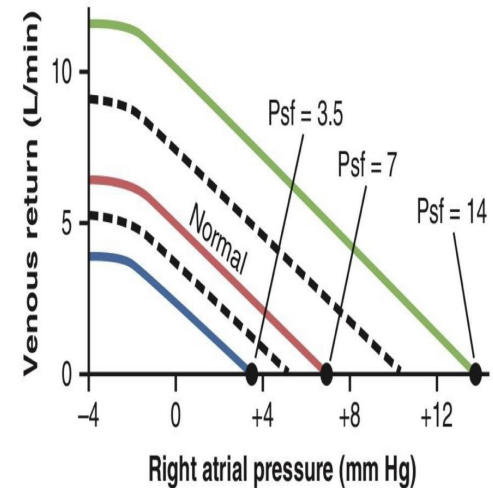
2. The greater the Psf the greater the “tightness” with which the circulatory system is filled with blood), the more the VR curve shifts upward and to the right. (green line)

3. The lower the Psf, the more the curve shifts downward and to the left. (blue line)

4. When the RAP = Psf, there is no longer any pressure difference between the

peripheral vessels & the Rt atrium. Resulting in? \longrightarrow No venous return.

a cessation of flow from the peripheral vessels into the right atrium, as there is no pressure gradient to drive the flow. This is known as a "zero pressure gradient" state, In this state, the balance between venous return and cardiac output is achieved, and the heart is able to maintain an adequate blood flow to the body's tissues.



More explanation

Determinants of venous return

2

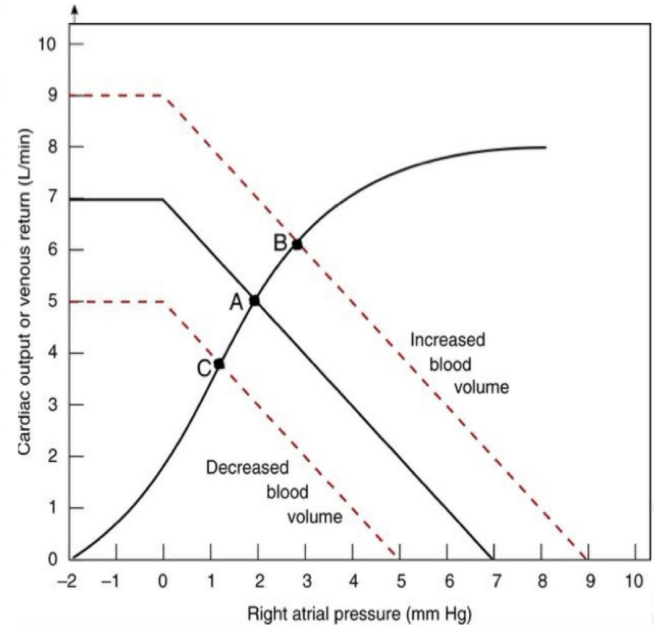
Blood volume

- At constant venous capacity: (on the curve)

A. Normal

B. \uparrow blood volume \Rightarrow \uparrow MCP \Rightarrow \uparrow VR,
The intersection point of the vascular function curve shifts upwards and to the right.

C. \downarrow blood volume \Rightarrow \downarrow MCP \Rightarrow \downarrow VR,
The intersection point of the vascular function curve shifts downwards and to the left.



Determinants of venous return

3

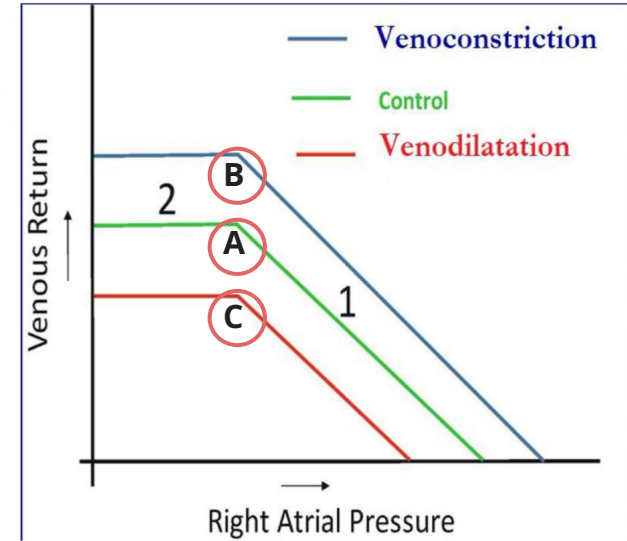
Venous capacity

- Definition: the volume of the blood that the veins can accommodate.
- At a constant blood volume : (on the curve)

A. Normal (Green line)

B. ∇ venous capacity (venoconstriction) \Rightarrow \uparrow MCP \Rightarrow \uparrow VR, The intersection point of the vascular function curve shifts upwards and to the right. (Blue line)

C. \uparrow venous capacity (venodilation) \Rightarrow ∇ MCP \Rightarrow ∇ VR, The intersection point of the vascular function curve shifts downwards and to the left. (Red line)



لَقَدْ خَلَقْنَا الْإِنْسَانَ فِي أَحْسَن تَقْوِيمٍ

Determinants of venous return

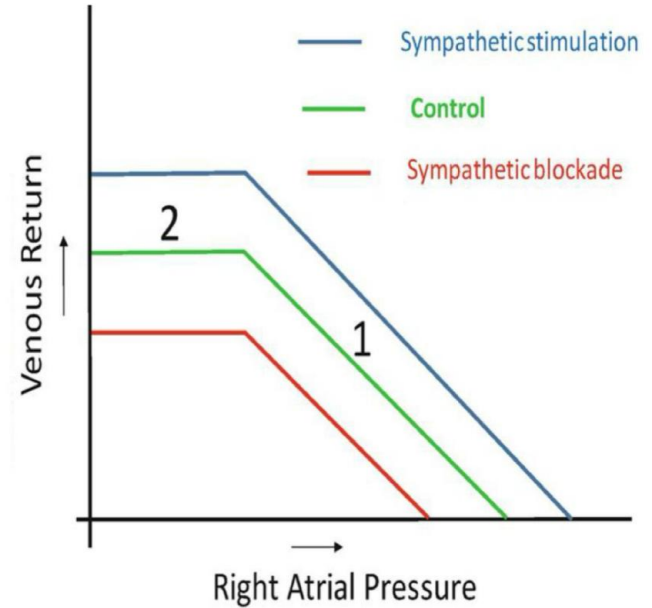
4

Sympathetic activity

A. Venous smooth muscle is profusely supplied with sympathetic fibers. (Green line)

B. \uparrow Sympathetic nervous system (SNS) activity \Rightarrow venoconstriction \Rightarrow \downarrow venous capacity \Rightarrow modest \uparrow MCP \Rightarrow \uparrow VR. (Blue line)

C. The veins normally have such a large diameter that the moderate vasoconstriction accompanying sympathetic stimulation has little effect on resistance to flow. (Red line)



Determinants of venous return

5

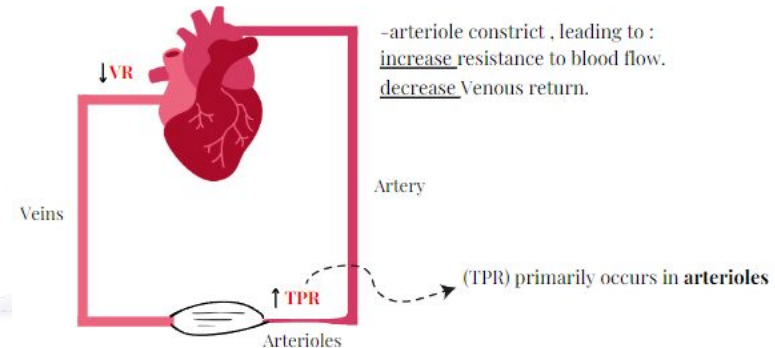
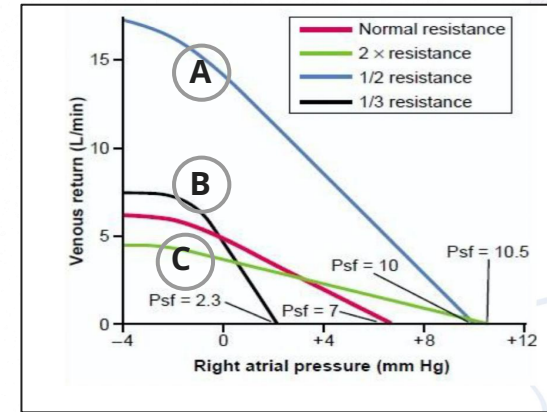
Total peripheral resistance (TPR)

- For a given RAP :

vasoconstriction \Rightarrow \uparrow TPR \Rightarrow \downarrow VR

A&B. \downarrow TPR \Rightarrow \uparrow VR, decreased resistance of the arterioles makes it easier for blood to flow from the arterial to the venous side of the circulation and back to the heart.

C. \uparrow TPR \Rightarrow \downarrow VR, increased resistance of the arterioles makes it more difficult for blood to flow from the arterial to the venous side of the circulation and back to the heart.

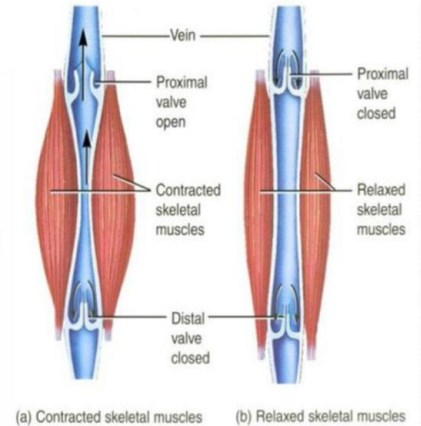


Determinants of venous return

6

Skeletal muscle activity

- This is known as skeletal muscle pump.
- Skeletal muscle contraction \rightarrow external venous compression \rightarrow \downarrow venous capacity \rightarrow \uparrow VR
- Skeletal muscle activity also counter the effects of gravity on the venous system. Like Calf muscle pump (2nd heart) promotes venous return from the lower extremity

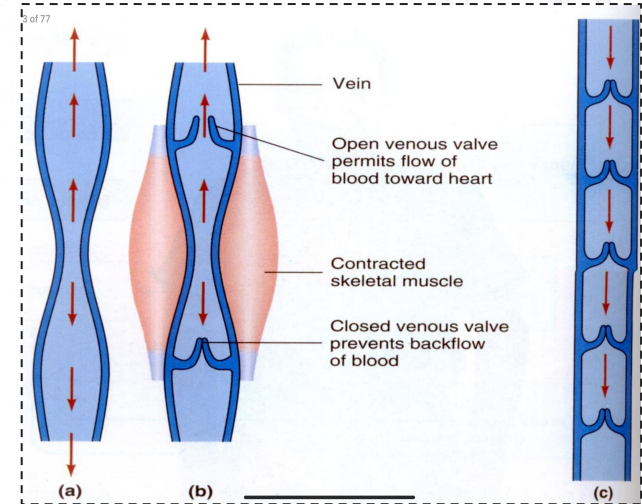


Determinants of venous return

7

Venous valves

- These valves play a role in counteracting the gravitational effects of the upright posture.
- These valves permit blood to move forward towards the heart but prevent it from moving back toward the tissues.
- **Skeletal muscle pump is ineffective when the venous valves are incompetent.**
- **permitting unidirectional flow enhances venous return**
- Chronically raised pressure in the veins leads to pathological distention of the veins (**varicose veins**) الدوالي .
- **Increased capillary filtration leads to swelling (edema) with trophic skin changes and ulceration (venous ulcers).**



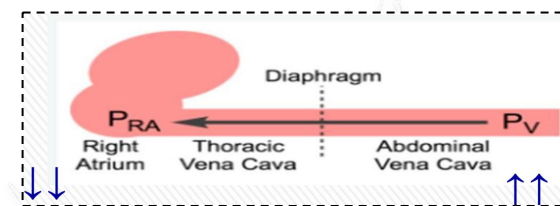
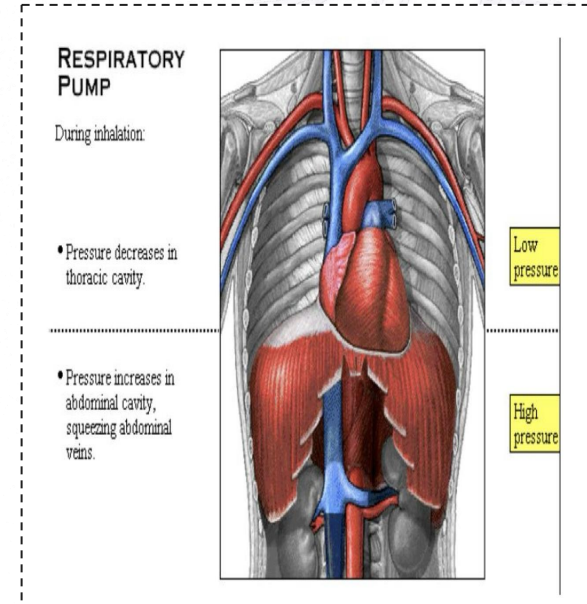
Determinants of venous return



8

Respiratory activity (respiratory or thoracic pump):

1. As the venous system returns blood to the heart from the lower regions of the body, it travels through the chest cavity
2. The pressure in the chest cavity is 5mmHg less than atmospheric pressure.
3. The venous system in the limbs and abdomen is subjected to normal atmospheric pressure.
4. Thus, an externally applied pressure gradient exists between the lower veins and the chest veins, promoting VR (respiratory pump).



Determinants of venous return



9

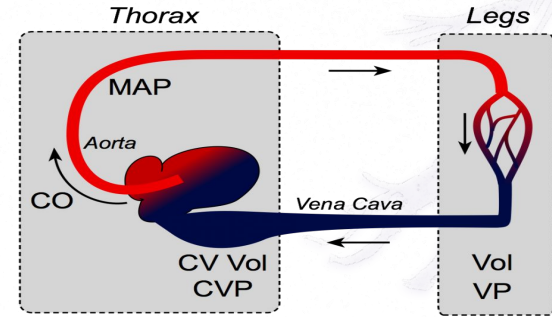
Effect of gravity

1. In standing, venous pressure \uparrow in the lower limbs :

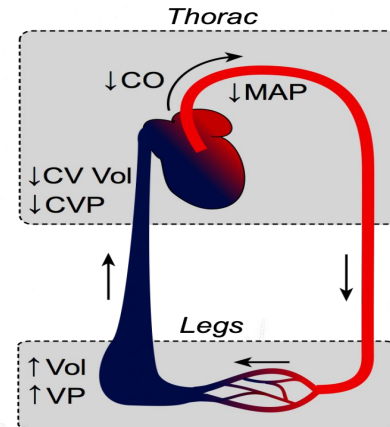
- $\rightarrow \downarrow$ thoracic venous blood volume and \downarrow CVP
- $\rightarrow \downarrow$ Rt ventricular filling pressure
- $\rightarrow \downarrow$ Rt ventricular SV by the Starling mechanism.
- $\rightarrow \downarrow$ reduced pulmonary VR
- $\rightarrow \downarrow$ Lt ventricular SV
- $\rightarrow \downarrow$ CO and mean arterial pressure (MAP)

2. If MAP falls significantly upon standing, this is termed **orthostatic** or **postural hypotension** $\rightarrow \downarrow$ cerebral blood flow to the brain and person might experience syncope (fainting) إغماء!

3. On the transition from sitting to standing, blood is pooled in the lower extremities as a result of gravitational forces.



Supine



Standing

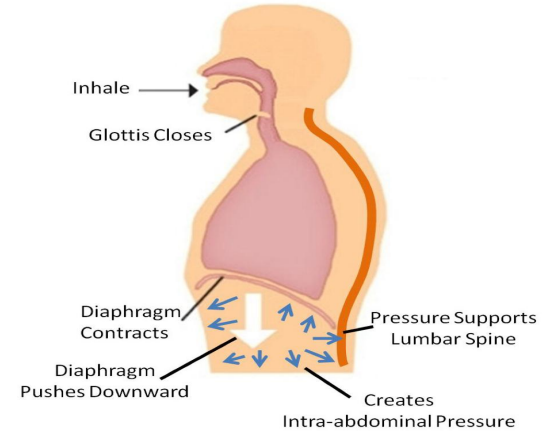
The effect of Valsalva Maneuver on VR

Valsalva maneuver

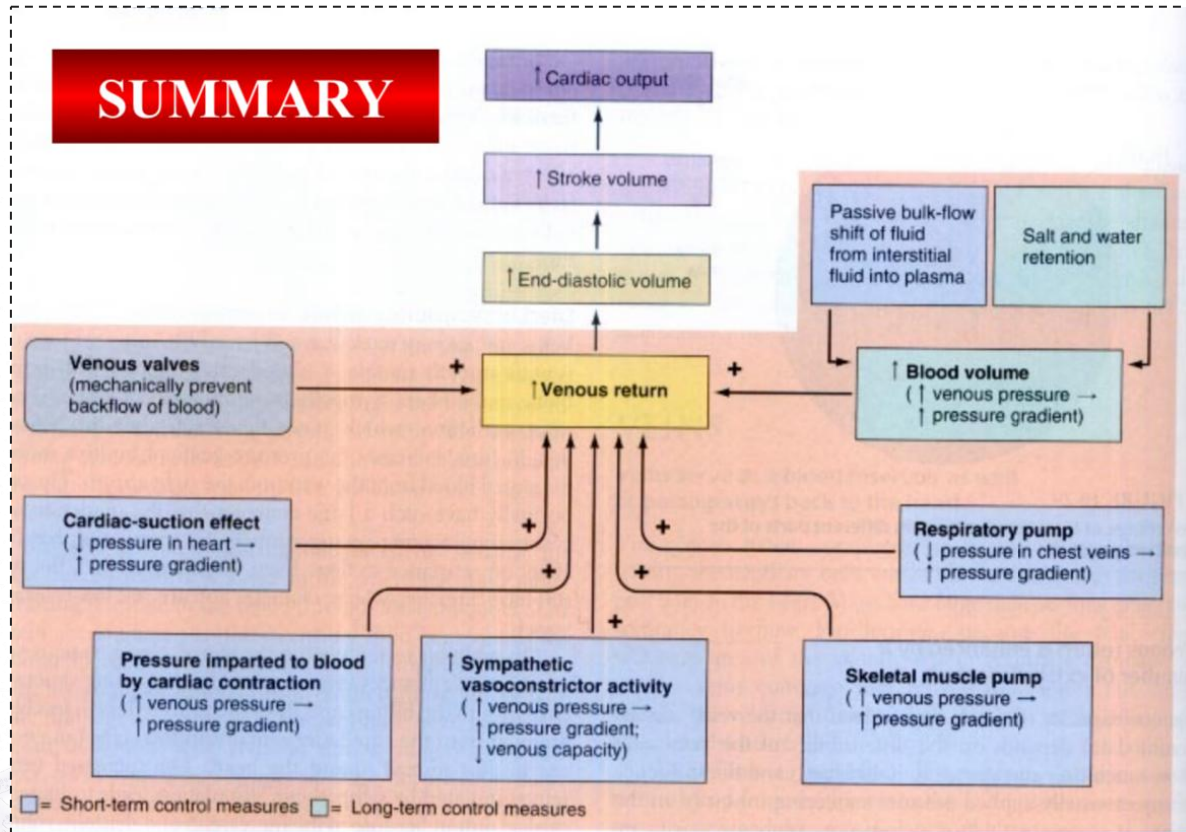
It is forceful expiration against a closed glottis

the effect on VR

Intrapleural pressure become positive which is transmitted to the large veins in the chest (persistent increase in thoracic cavity) \Rightarrow \Downarrow VR.



Summary



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



Sorry but if you will not check it راحت عليك المليون

MCQs:



Answers

For more question check our summary file!

1/C
2/A
3/C

1 Venous return (VR) is the flow of blood from the periphery to the heart , at what location?

| | | | | | | | |
|---|----------------|---|------------------|---|---------------|---|-------------|
| A | Left ventricle | B | Right ventricle. | C | Right atrium. | D | Left atrium |
|---|----------------|---|------------------|---|---------------|---|-------------|

2 What of the following causes an increase in RAP in cardiac & vascular function curve ?

| | | | | | | | |
|---|---------------|---|----------------|---|---------------|---|--------------|
| A | -ve inotropic | B | +ve inotropic. | C | Increase TPR. | D | Decrease TPR |
|---|---------------|---|----------------|---|---------------|---|--------------|

3 What is the function of venous valves ?

| | | | | | | | |
|---|------------------------|---|--|---|---|---|------------------------------|
| A | Maintains blood volume | B | Counteract gravitational force while lying | C | Aid skeletal muscle in directing blood toward heart | D | Promotes gravitational force |
|---|------------------------|---|--|---|---|---|------------------------------|

MCQs:



Answers

For more question check our summary file!

4/A
5/C
6/B

4 What type of blood vessels are known as resistance vessels?

| | | | | | | | |
|---|------------|---|-------|---|-------------|---|---------|
| A | Arterioles | B | Veins | C | Capillaries | D | Venules |
|---|------------|---|-------|---|-------------|---|---------|

5 Chronically raised pressure in the veins leads to pathological distention of the veins named as:

| | | | | | | | |
|---|----------------|---|---------------|---|------------------|---|-------------------|
| A | Venous Ulcers. | B | Venous Edema. | C | Varicose Veins . | D | Valsalva maneuver |
|---|----------------|---|---------------|---|------------------|---|-------------------|

6 The highest blood distribution is found in:

| | | | | | | | |
|---|----------------------|---|----------------|---|--------------------|---|-------------------|
| A | Systemic arterioles. | B | Systemic veins | C | Pulmonary vessels. | D | Systemic arteries |
|---|----------------------|---|----------------|---|--------------------|---|-------------------|

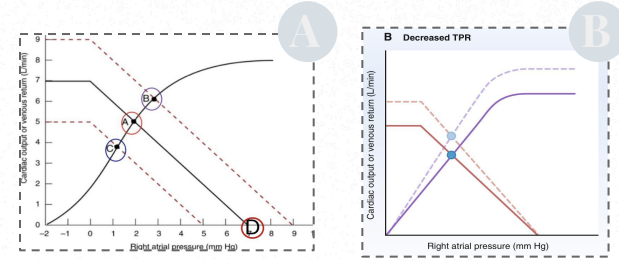
SAQ

What does the curve “A” represent and identify the point D ?

What is the causes that can shift the curve “A” to point B, C ?

What it's the effect of decrease TPR?

Use curve B to answer this question



Slide 15

Slide 24

Slide 20

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 :



Rayan Almutairi

Ritaj Alsubaie

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



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