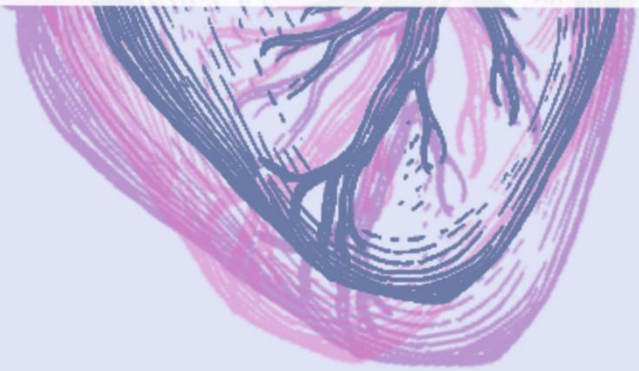
Cardio-Vascular System

11+12

Physiology



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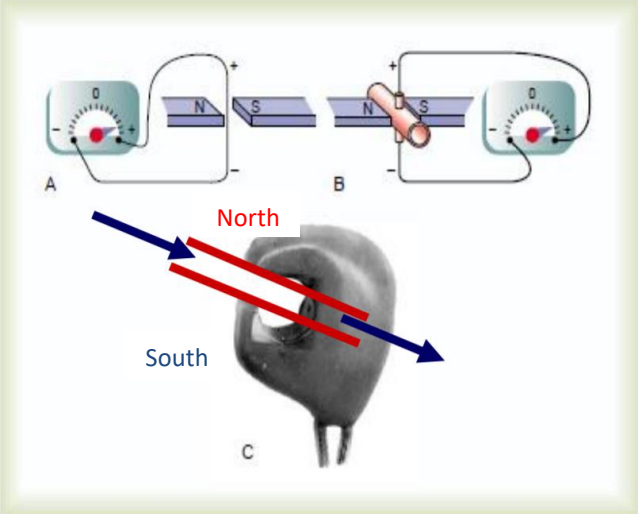
Cardiac Output Measurements

*We can measure the CO by either **Direct methods** or **Indirect methods**

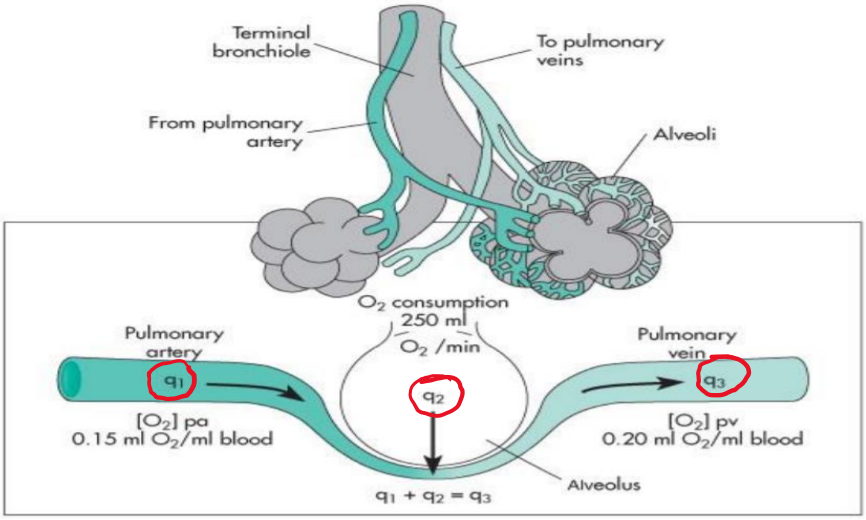
1-Direct method: we make the aorta bleed and collect the blood but it's not practical and not used

2-Indirect methods:

A- Electromagnetic flowmeter: We have two poles of a magnet (north and south). When a charged flow runs between the two poles, a current is thus formed and can be calibrated up by a galvanometer. Because blood is full of electrolytes, it is a charged flow, and the current that would be formed between the poles of the magnet is proportional to the flow. If we measure this flow per min, we can calculate the cardiac output. This method can be used around any artery, and many times. It is usually used in cardiac surgery.



B- Oxygen Fick method:



*The O_2 content of the pulmonary vein **=** the O_2 content of the pulmonary artery **+** the O_2 that has been taken up from the lung.

*The amount of oxygen that enters the lung per min. through the pulmonary artery (q1) = cardiac output × oxygen concentration in the systemic **venous** blood

q2= the amount of oxygen taken by the lungs from air through alveoli.

q3= the amount of oxygen that enters the pulmonary veins: This equals the cardiac output × **arterial** oxygen concentration.

Remember that the pulmonary vein carries arterial blood, while the pulmonary artery carries venous blood

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Now that we know that q3 = q1 + q2:

$$CO \times arterial\ O2\ conc. = CO \times venous\ O2\ conc. + oxygen\ uptake$$

$$\text{Oxygen uptake} = CO \times \{C_{AO2} - C_{VO2}\} \rightarrow CO = \frac{Oxygen\ uptake}{\{C_{AO2} - C_{VO2}\}}$$

To sum things up.. Cardiac output = (oxygen consumption in 1min) / (arterial concentration of oxygen – venous concentration of O2). **Pay attention to the units ^^**

Some Q&As

How to find the oxygen consumption?

- We measure it using a spirometer. You let the patient breathe and you measure concentration of inspired O_2 after one or two minutes.

How to find the arterial and venous concentrations of oxygen?

- All arteries have the same concentration of oxygen because the exchange happens only at the level of the capillaries. That's why you can take a sample from any artery to measure the concentration of oxygen in arterial blood.

However..

We can't take venous sample from any vein because the concentration of oxygen differs according to the metabolic needs. So we put a special catheter called **Swan-Ganz** catheter in the pulmonary artery or the right ventricle (using the cubital vein). This blood found there is called **central venous blood**; as if we mix all venous blood and take the sample.

Test your understanding:

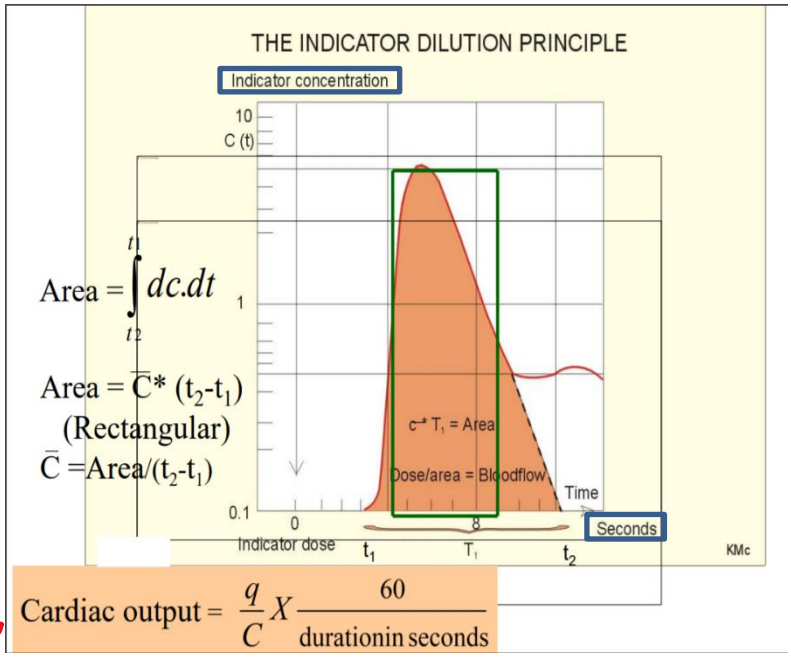
O_2 Fick Problem

- If pulmonary vein O_2 content = 200 ml O_2/L blood
- Pulmonary artery O_2 content = 160 ml O_2 /L blood
- Lungs add 400 ml O_2 /min
- What is cardiac output?
- Answer: 400/(200-160) =10 L/min

Again, pay attention to the units (mL, L, etc)

Some Qs may require conversions.

C- Indicator Dilution principle:



We use an indicator that is non-toxic, distributed well and colorful. E.g. ***Cardiogreen.*** We inject a certain amount of this dye (call it q) in the right ventricle (using Swan Ganz catheter we mentioned before). **Briefly:** we draw this curve and we calculate the area (either by integration or by rectangles), dividing it by time duration to get the mean concentration (C). Using this equation we can get the cardiac output.

D- Thermodilution principle:

We inject cold saline with a known temperature and volume (again using Swan Ganz catheter). **Notice that this special catheter has a thermistor that measures temperature.**

$$AREA = \int_{t1}^{t2} dT.dt$$

$$\frac{Area \times 60}{Tavg \times duration}$$

Now using a special computer program we can calculate the cardiac output through integrating temperature changes over time. (or we can use an equation similar to the last method's)

The advantage of this method is that you can use it multiple times to have multiple readings and take the average

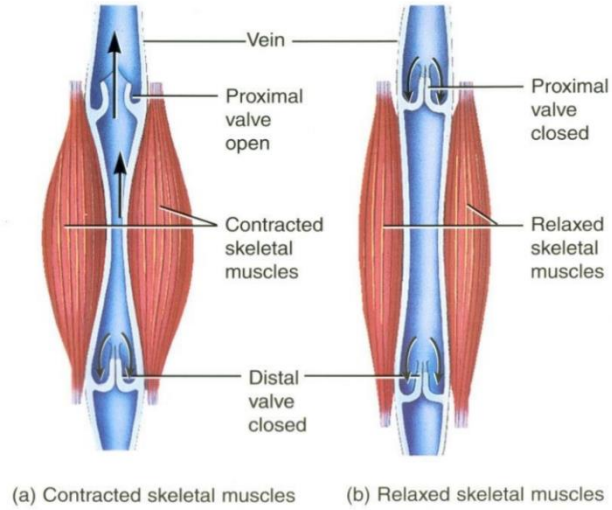
Venous Return (VR)

- ✓ It is the amount of the blood that returns to either atria per minute. It equals the cardiac output in normal conditions according to Frank-Starling (What comes to heart must leave).
- ✓ Venous return is a flow and thus based on Ohm's law.

Ohm's law:

$$VR = CO = \frac{\Delta P}{R} = \frac{\text{Venous pressure} - \text{Rt.Atrial pressure}}{\text{resistance to venous return}}$$

- ✓ The veins are embedded within skeletal muscles that when contract squeeze the veins and push the blood upward toward the heart.



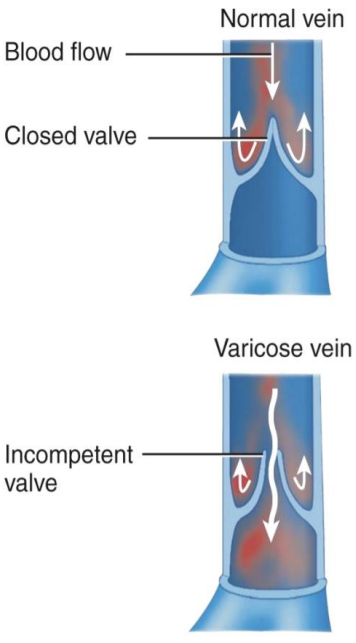
- ✓ Veins have valves. When these valves are normal, they prevent blood from going down due to gravity. They close and keep little amount of blood. Because of this the blood column is discontinuous and the pressure in the veins is maintained low.

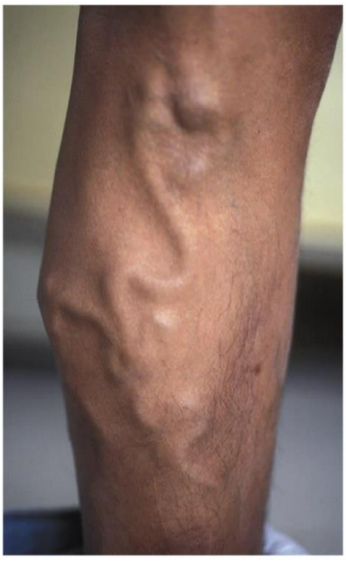
Clinical application:
When someone stands for a long time, blood will be collected around the valves. This causes the valve to slightly open and the blood column will become continuous. By time, the valve will become incompetent and this increases the pressure in the affected veins leading to varicose veins. These veins are congested, dilated, tortuous and blue.

Why does that happen?

Let's assume that the right atrium is our zero point and from there until the toe we have a continuous blood vessel that has a length of 136 cm, what is the pressure at the lowest point?
- Height from the right atrium = 136cm
- Pressure = blood column/density of mercury= 136/13.6= 10 cmHg= 100 mmHg

The pressure down there is normally much lower than that, so here lies the problem of standing still for a long time.
* Abdominal pressure also tends to increase venous pressures in the legs.




Dilated and twisted appearance of varicose veins in the leg

Central Venous Pressure (CVP):

- ✓ It is the pressure in the right atrium
- ✓ In the right atrium CVP is usually 0mmhg, but it might reach 20mmhg and it's still considered normal
- ✓ Increase in right atrial pressure causes blood to back up into the venous system (venous return decreases) thereby increasing venous pressure
- ✓ To monitor the right atrial pressure we use a Catheter "central venous line".
- ✓ CVP should be monitored in heart failure patients because increased CVP may lead to sudden death, especially when giving IVF fast and haphazardly
- ✓ Right atrial pressure (RAP) is determined by the balance of the heart pumping blood out of the right atrium and flow of blood from the large veins into the right atrium. When pumping decreases or the venous return increases, right atrial pressure will increase.

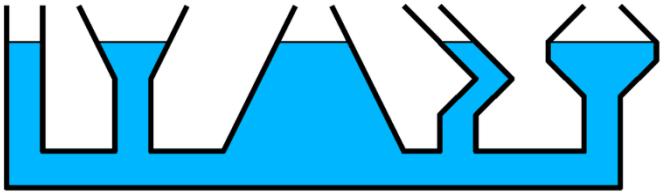
Factors that increase RAP:

1- **Increased blood volume**: Pressure increases everywhere including in the right atrium.
2- **Increased venous tone**: more venoconstriction → pushes more blood to atria → increased pressure in right atrium
3- **Dilation of arterioles**: Dilation of arterioles → less resistance → more blood flow→ increased RAP
4- **Decreased cardiac function**: When pumping increases → End systolic volume (ESV) decreases→ RAP decreases because RAP reflects how much blood is found in the ventricle. On the other hand, when cardiac functions decrease RAP increases. That's why it should be monitored in patients with heart failure.

Factors that facilitate Venous Return: (Cardiac output increases in all scenarios)

1. **skeletal muscle contraction**: When muscles contract → increased venous pressure → increased gradient between the veins and right atrium →increased venous return
2. **Veno-constriction** → increased venous pressure → increased gradient → increased venous return
3. **Blood volume**: increased blood volume means more pressure → increased venous pressure → more gradient between veins and right atrium → more venous return
4. **Respiration**: Inspiration → volume in the chest increases → pressure inside alveoli decreases (according to boyle's law) → less pleural pressure → less right atrial pressure → more venous return.
Expiration: The other way around ^^
5. **Cardiac function**: if it increases → ↓ESV→ RAP↓ → increased gradient → increased venous return → This is called cardiac suction.
6. Venous Return is decreased if **valves** are incompetent.

Venous Return Curve:



We can understand it by using the concept of communicating vessels. (if we stop the heart and just pour the blood in the vessels, the level of blood will be parallel in all these vessels (just like the figure) and if we add more blood the level will rise in all vessels by the same amount. If we lowered the pressure in one of them, water would flow to this vessel from all other vessels (from high to low pressure), until they are all equal and happy again.

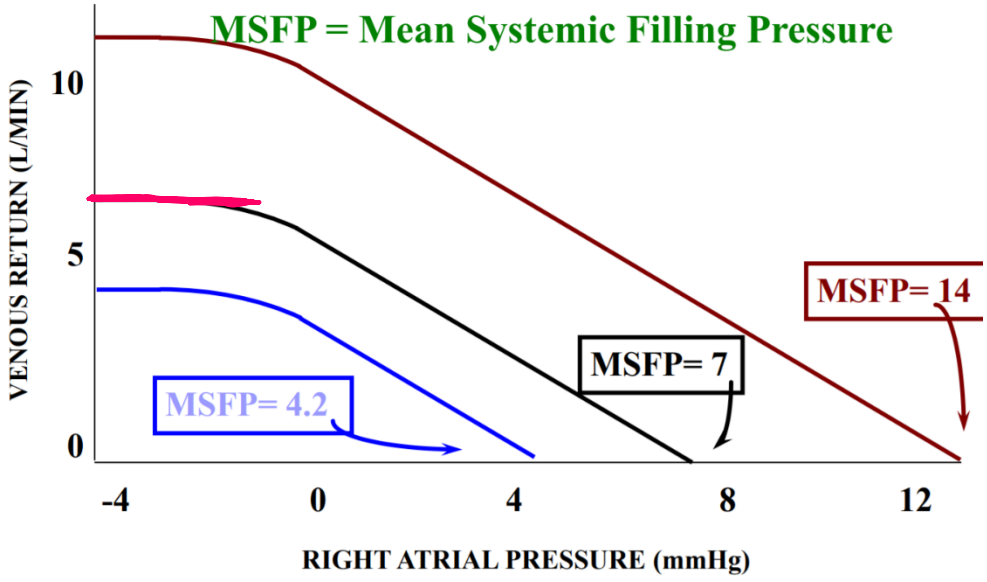
Now that all vessels are equal, the arterial pressure in every single one of them is called mean systemic pressure because it is the same pressure in all vessels of the systemic circulation. Makes sense, right?

History Class:
The first person to measure the mean systemic pressure was Guyton. He first measured it in dogs, then tried to do so in humans and found out that it was about 8 mmHg

Now let's consider the right atrium a part of the game. It has the same pressure as other vessels and no blood is flowing anywhere. If right atrial pressure decreases below 8mmHg, a gradient will form between right atrium and systemic circulation.

If pressure in right atrium is 6mmHg there's a little flow. When it's 5mmHg, flow will increase "more" and so on. That's why we can call the mean systemic pressure (MSPH or mean systemic **filling** pressure) because the difference (gradient) between MSFP and atrial pressure is what causes its filling.

When the atrial pressure reaches zero, the venous return will be max at 5L/min. When pressure in the right atrium becomes negative, however, vessels will collapse and venous return will no longer increase (plateau in the figure below).

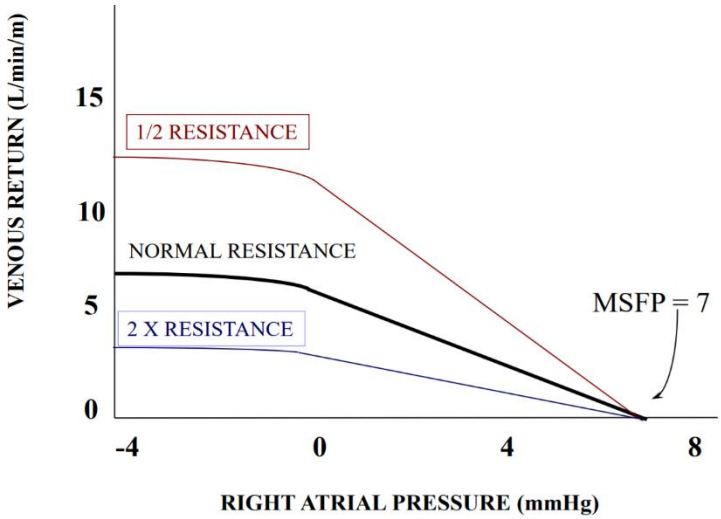


What does affect MSFP?

1. **IV fluid infusion**: increased fluid or blood volume increases venous return→ shift the curve to the right and upward. That's why when someone has hypotension, we give him/her IV fluid.
2. **Sympathetic stimulation:** It causes veno-constriction and consequently higher venous pressure. Sympathetic inhibition goes other way around ^^
3. **Hemorrhage and dehydration**: means low blood volume → shift the curve to the left and downward

Effect of Resistance:

increasing or decreasing TPR (total peripheral resistance) has no effect on MSFP because resistance is mainly in the arterioles, and arterioles contain little amount of blood, so it won't affect the MSFP. However, venous return can change. When the resistance is decreased, venous return will increase and vice versa.



Other factors affecting venous return:

1- Thiamine deficiency (Beriberi): the artery becomes flexible → Arteriolar dilatation → ↓RVR (resistance to venous return) → ↑venous return

Remember: VR = (MSFP - RAP) /RVR

2- AV fistula: An arteriovenous fistula is an abnormal connection or passageway between an artery and a vein. It may be congenital or surgically created for hemodialysis treatments. It shunts blood from an artery to a vein bypassing the capillary network → resistance decreases → increase in VR.

* Hemodialysis means that blood is taken from an artery to be filtered in a machine and then returned to a vein. Making an AV fistula makes this procedure easier.*

3- Hyperthyroidism → O_2 consumption increases → Blood vessels relax in response → ↓RVR → ↑venous return

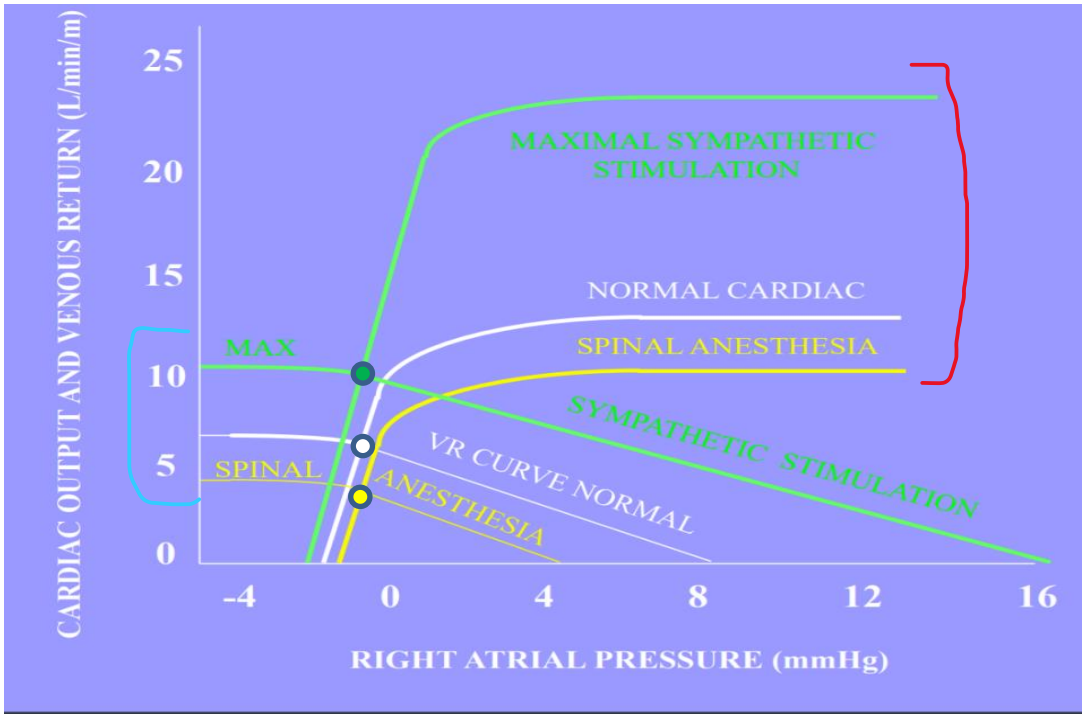
4- Anemia: decreased number of RBCs → decreases viscosity → ↓RVR → ↑venous return

5- Venous compliance (muscle contraction): As we said before, veno-constriction increases venous pressure. However, muscle contraction also increases RVR. So we have two opposing factors. If the increase in venous pressure was higher than the increase in RVR, venous return increases, and vice versa.

6- Obstruction of veins → ↑RVR → less venous return

Note: Please understand and don't just memorize
Reversing any of the factors mentioned above would lead to the opposite effect. For example: Increasing blood volume increases MSFP and venous return, so decreasing it would decrease venous return. Makes sense, right?

This figure shows curves of both Cardiac output and Venous return in different conditions. As you can see (hopefully) the points of intersection are the 'working cardiac output' or the cardiac output that's found in our body. So, in normal conditions it is about 6L (white curves). In the case of sympathetic stimulation, the venous return curve is shifted upward and to the right as we said earlier. The cardiac output is also shifted upward so now we have a new intersection point, and the working cardiac output would become higher (10L for instance).



Good Luck!!