Cardiovascular System

Sheet 9

Subject | physiology

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Last lecture we started talking about **the cardiac output and venous return.** And we said that the Cardiac Output is the sum of all tissue flows and is directly proportional to tissue O2 and proportional to 1/TPR when AP is constant.

- **CO = MAP/TPR**
- **MAP=CO*TPR**

- CO: cardiac output
- MAP: mean arterial pressure
- TPR: total peripheral resistance

- MAP of any person should always stay constant (homeostasis), if it increases our system will try to decrease it and vice versa.

- From the previous equation we can conclude that we can change the MAP by changing CO or by changing Total peripheral resistance:

Also we said that the cardiac index is used to compare the cardiac output between different individuals (cardiac index equals the cardiac output divided by body surface area, so cardiac index unit will be L/min/$m2$).

- You should know that there are **differences** in the basic cardiac output values between individuals, since it is affected by the **size and weight** so we use cardiac index to compare between people.

Question:

How comes the CO equals 15L/min and the whole blood volume equals 5L??

^^ This means that the blood circulates faster than normal, so if the CO is 15L/min; the 5L circulate 3 times each minute.

-We also said that Cardiac output = Stroke volume* Heart Rate, and discussed the factors affecting the CO.

Now let's talk about the cardiac output curve:

The cardiac output curve relates

the **right atrium pressure** to the **cardiac output**,

and it is a mathematical representation of

Frank-starling law.

-The right atrium pressure is an indicator for EDV, the higher the EDV is, the higher the right atrium pressure.

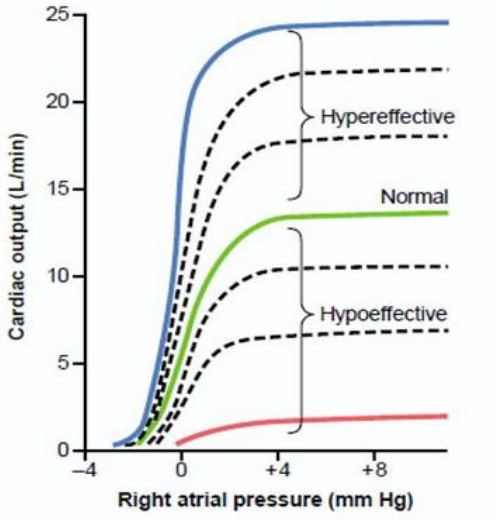
-The cardiac output is an indicator for the intraventricular pressure

- **Normally**, when the right atrial pressure equals zero, the CO will be 5 L/min.

- when the right atrial pressure increases due to an increase in EDV (Frank-starling) the CO increases, until it reaches its optimal (Maximum value) which is 15 L/min, we will observe a brief plateau in this curve.

SO we can increase the CO up to 15L/min without any autonomic effect.

- If we increased the right atrial pressure more and more, the cardiac output will decrease, leading to heart failure.

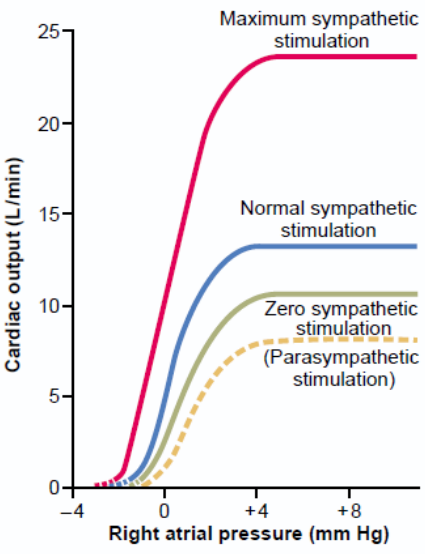


- **Hyper-effective** heart, an increase in the CO above normal and only 2 factors can make the heart a better pump:

1. Sympathetic nervous stimulation: increases contractility, which means **fixed end diastolic volume** (fixed right atrial pressure-RAP) and **increased stroke volume**, so the CO when RAP equals ZERO will be higher than 5L, SO the curve is shifted to the **left and upward**.

2. hypertrophy of the heart muscle (athletes).

When the heart muscle goes under hypertrophy, it becomes stronger and pumps more blood at the same EDV.



^^ The optimal (maximum) value of cardiac output in a hyper-effective heart in non-athletic individuals is about 25 L/min. However, in athletes, the max. value for CO may reach **35 L/min,** so at rest their stroke volume about 100 and their heart rate about 50 bpm.

- **Hypo-effective** heart, a decrease in the hearts ability to pump blood, can be caused by many factors, such as coronary artery blockage, Myocardial infarction, **sympathetic block (inhibition**) etc.

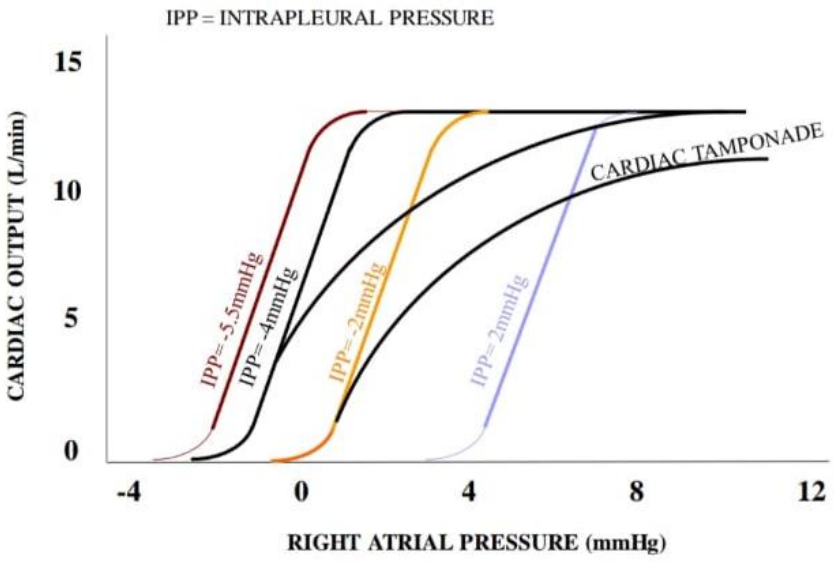
the curve will be lower as the CO will be decreased (the curve will be shifted **downward and to the right**). The value of the CO when the right atrial pressure is zero may reach only **2L**.

At rest, the sympathetic nervous system is always working at a basal rate, and the importance of this is that we can have a positive or a negative control over SNS, for example in the vessels inhibition of SNS (negative control) causes vasodilation. However, if the rate of SNS at rest was ZERO we would not be able to have a negative control over it.

Effect of the intra-plural pressure on the cardiac output:

The heart is surrounded by the lungs which are surrounded by the pleura.
-Normally, the pressure inside the pericardium is equal to the pressure inside the pleura.
- The intra-plural pressure is always **negative**** around **-5 mmHg** that's why the lungs are **distended** (like if there is a vacuum around them).

- According to the figure:



1- Let's consider the **normal** intrapleural pressure is **(-4 mmHg)** at this value the right atrium pressure (RAP) will be **zero** and the cardiac output at that pressure equals 5L and the maximum cardiac output is 15L.

2- When the intrapleural pressure is decreased to **5.5mmHg** the pressure in the right atrium will be reduced to **-1.5mmHg** and the cardiac output at that pressure will be equal to 5L and the maximum CO will be 15L. **(shifted to the left)**.

3- When the IPP increases to **2 (**like in the case of pleural effusion**),** this increase in the intra-pleural pressure will be reflected on the heart and the pressure will change in the right atrium from **zero to +6** and this means that **in order to fill the atrium we will need higher pressure gradient** (more power to fill the heart). This will shift the cardiac output curve to the right.

^^ It is **important** to notice that in case of increase/decrease in IPP the whole curve will be shifted to the right/left and the **maximum cardiac output according to Frank-Starling will be the same**.

^^ **In the case of cardiac tamponade (pericardial effusion):**

The increase in the pericardium pressure will **directly** affect the heart muscle, and it will prevent filling of the blood, thus **reaching the maximum CO will become very difficult** (we need very high atrial pressure to reach the maximum CO).

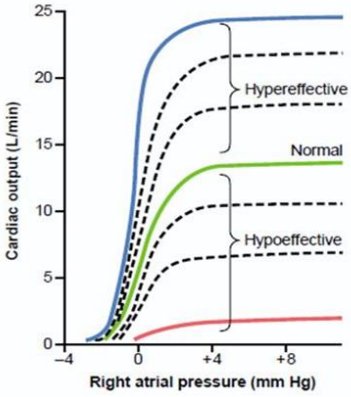
* Shifts the curve to **the right** and sometimes **downward.**

For example, when the interpleural was -4 mmHg and there was pericardial effusion we needed almost **8 mmHg** to reach the maximum CO. "more severe than pleural pressure".

- If the cardiac tamponade becomes **very severe**, the cardiac output **might not reach the maximum CO** and **it might reach 0** since the heart is unable to pump blood anymore and this eventually leads to **heart failure**.

- So cardiac tamponade is an emergency case and it needs to **be relieved by cutting through the chest using a sharp object.**

NOW the plateau of CO curve is determined by the strength of the heart (contractility + HR), and some of the factors that affect the plateau are:



1- sympathetic raises the plateau up and shifts the curve to the left. (hyper effective heart)

2-parasympathatic mostly does not have any significant effect.

3-Heart hypertrophy raises the plateau up and shifts the curve to the left. (hyper effective heart)

4- Myocardial infarction lowers the plateau and shifts the curve to the right due to mass decrease. (Hypo effective heart)

5- Valvular disease lowers the plateau and shifts the curve to the right due to decrease in the cardiac output, examples are **stenosis or regurgitation**.

Stenosis: narrowing of the valve opening, thus an increase in the resistance to blood flow due to the narrowing of the orifice, and this leads to decrease in stroke volume and eventually decrease in cardiac output.

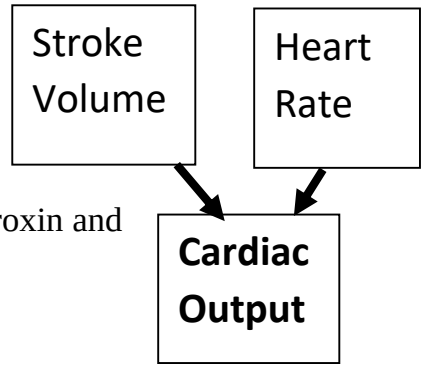
Regurgitation (valve incompetence): some of the blood goes back from the ventricle to the atrium (AV valve), or from the aorta to the ventricle (semilunar valve), and thus decreasing the CO.

6- Myocarditis lowers the plateau as it decreases muscle mass (decreases contractility).

7- Cardiac tamponade (pericardial effusion) will prevent the filling of the ventricle, so it decreases cardiac output and thus lowers the plateau.

8- Metabolic damage also lowers the plateau.

Factors that affects cardiac output:



CO = Heart rate * stroke volume

Heart rate is controlled by the Autonomic nervous system (sympathetic or parasympathetic) or Hormones (Thyroxin and catecholamines).

Stroke volume = EDV - ESV

EDV is mainly affected by:

1-The venous return (equals CO): how much blood return back to the heart. Increase in venous return increases EDV, increases **preload,** (and this is Frank-Starling).

2- Filling time, which is affected by the heart rate, increasing the heart rate (tachycardia) leads to **decrease in the cardiac output** due to the decrease in the **duration of ventricular diastole and this reduces the filling time of the ventricle**.

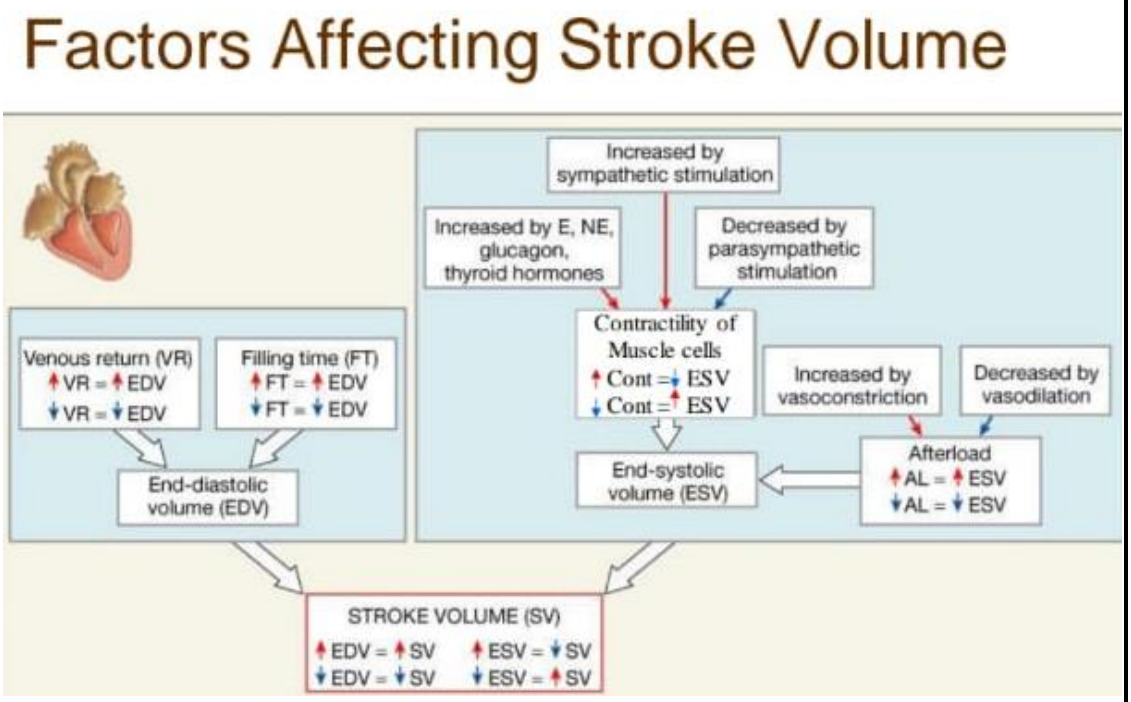
During exercise the heart could reach its maximum rate (200 bpm) and this is dangerous, you should **not exceed 80% of your maximum heart rate**, because the increase in heart rate leads to reduced stroke volume and thus decrease in cardiac output and eventually might lead to myocardial ischemia.

ESV is affected by:

1- Contractility: Decrease in contractility (negative inotropic) increases ESV

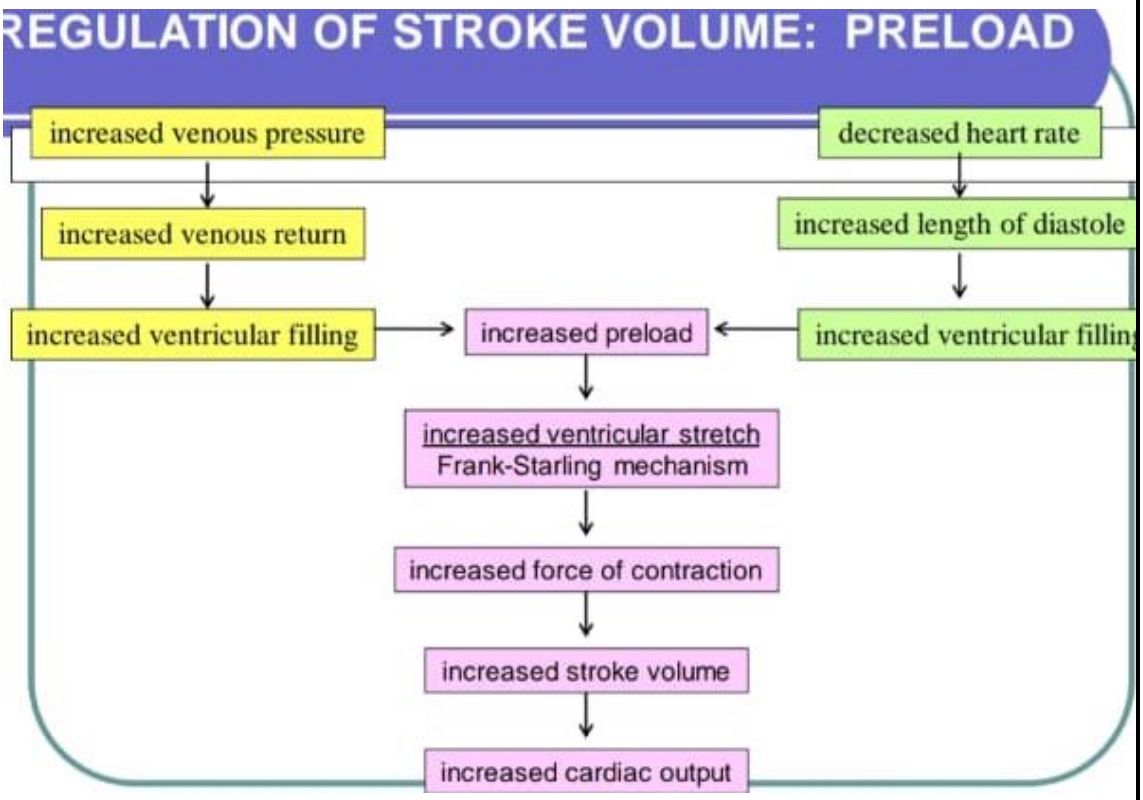
2- Vasoconstriction, increases the pressure, increases the resistance and thus decreasing the blood flow (cardiac output) by decreasing stroke volume. Also causes an increase in the afterload and increase in the ESV.

Pay attention to **Glucagon** which is considered a **positive inotropic agent.** And because Glucagon only has an effect on contractility it is used instead of Epinephrine, Norepinephrine to avoid any increase in the Heart Rate.



Preload: is the initial stretching of the myocytes prior to contraction (related to ventricular filling).

An increase in the venous pressure means increase in Δp: The difference between the venous pressure and the right atrium pressure.



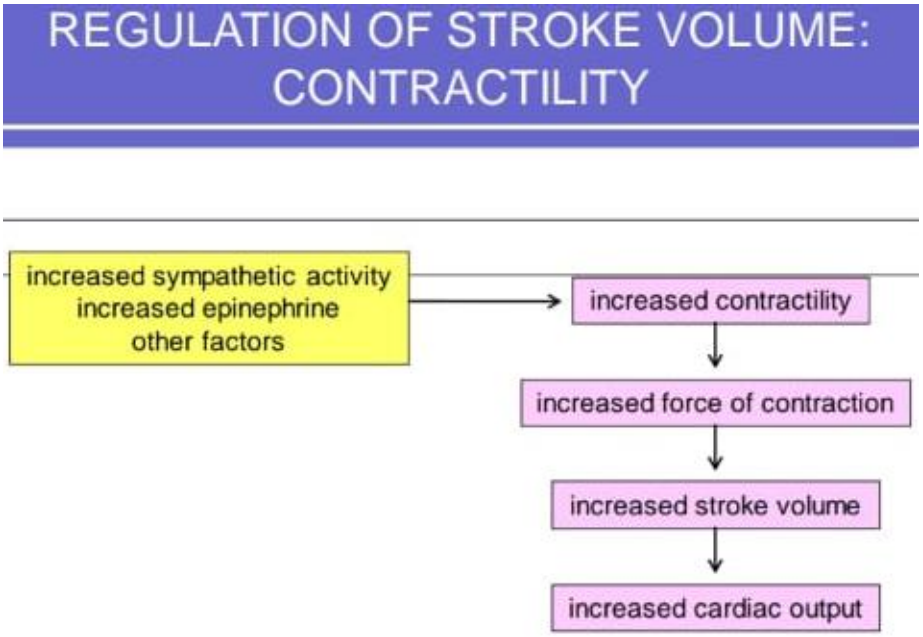
- Contractility is mainly affected by sympathetic stimulation (positive inotropic).

- Excess Ca++ increases contractility.

-Excess K+ (hyperkalemia) decreases contractility.

- The best measure for **Cardiac contractility** is **Ejection fraction:**

the maximum change in pressure per time (dP/dt).



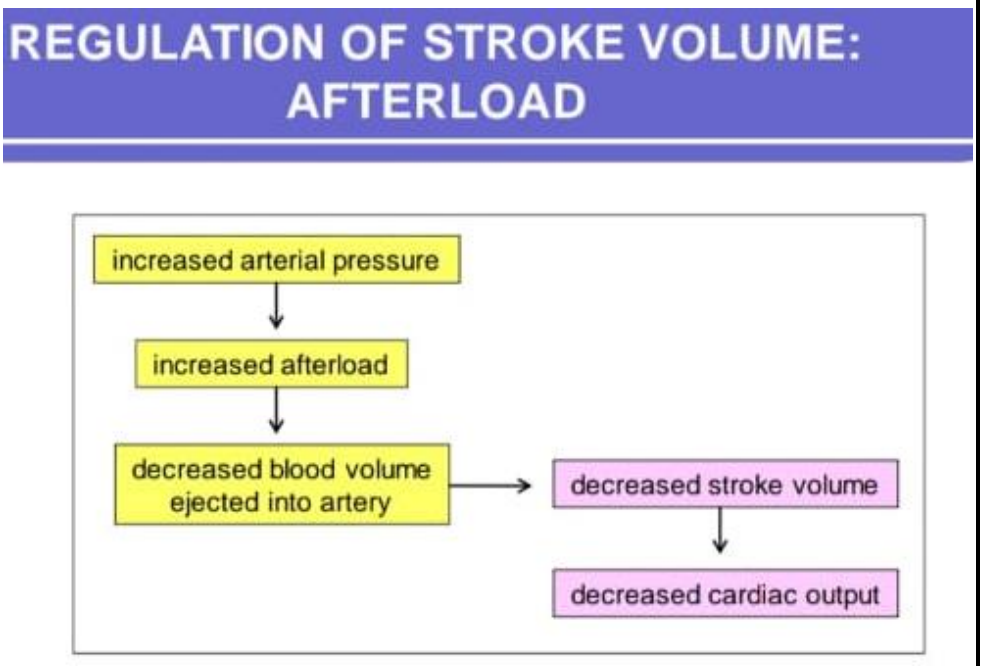
However, it is not easy because **the pressure of the ventricle** during the isovolumic contraction **increases very fast**, so we use special machines to measure it.

Note that (dP/dt) **is not an accurate measure,** because it increases with increasing preload and afterload. **(dP/dt)/P ventricle** is better (**P ventricle** is instantaneous ventricular pressure).

Afterload is the force against which the heart has to contract to eject blood (related to aortic or pulmonary pressure during ventricular systole).

- Vasodilation decreases the afterload.

- Hypertension increases the afterload.



Measurement of cardiac output:

- **Direct methods**: Used in animals, you cut the aorta, collect the blood ejected per min, this gives you cardiac output. Obviously, we can't do this in humans.

- **Indirect methods:**
  1- Electromagnetic flowmeter it is indirect but is done directly in heart surgery.
  2- Indicator dilution (dye such as cardiogreen)
  3- Thermal dilution
  4- Oxygen Fick Method (CO = (O2consumption / (A-V O2 difference)).

Electromagnetic flowmeter: We put two poles of magnate (north and south) around the aorta. When a charged flow passes between the two poles, an electrical current is formed and it is proportional to the flow, the current can be followed up by a **calibrated galvanometer**.

Because blood is full of electrolytes, It is a charged flow, and the current that would be formed (we can also call it voltage difference or potential difference) between the magnate 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, (**only used during cardiac surgery**, invasive method).

Fick Method (oxygen consumption):

-The amount of blood that comes from the right ventricle to the lungs through the pulmonary artery per min is called **Cardiac output**.

- The blood that come to the left ventricle by pulmonary veins is **equal to CO**.

- The **amount of oxygen that comes to the lung per minute (Q1)** equals cardiac output multiplied by oxygen concentration in the venous blood.

- The **amount of oxygen that come to left ventricle (Q3)** from the lungs by pulmonary veins = the cardiac output* the concentration of oxygen in the arterial blood.

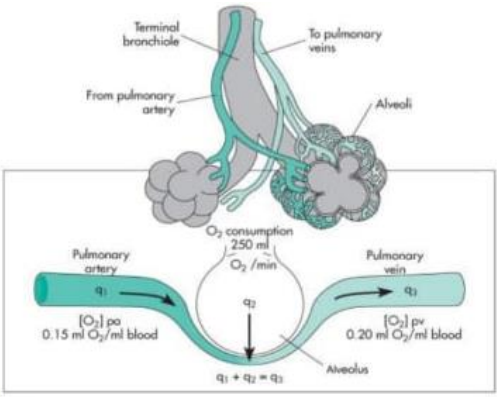
The O2 content does not differ between the blood in pulmonary veins and any other artery, since the exchange of gases happens in capillaries, so if we use any artery we get the same O2 content in PV.

^^ the concentration of O2 in arterial blood = O2 in veins + O2 uptake by the lungs.

Q1= Cardiac output*concentration of O2 in venous blood.

Q2= amount of Oxygen uptake by the lungs = CO *(C arterial O2 – C venous O2).
^^ O2 uptake --- measured by spirometer.

Q3= Cardiac output * the concentration of O2 in arterial blood = Q1 + Q2.



$q_1 = CO * C_{VO2}$
q_2=amount of Oxygen uptake by the lungs
$q_3 = CO * C_{AO2}$ and equals $= CO * C_{VO2} + O_2$ uptake
Oxygen uptake $= CO\{C_{AO2} - C_{VO2}\}$
CO=Oxygen uptake/$\{C_{AO2} - C\ V_{O2}\}$

Cardiac output = Oxygen uptake / (C arterial O2 – C venous O2).

Oxygen uptake = CO*(CAO2-C VO2)

^^ When it comes to the O2 content of venous blood we cannot just simply take a sample from any vein, as O2 content differs greatly, due to different O2 requirement by different tissues.

SO **How to get a sample from pulmonary artery?**

Using a catheter inserted from antecubital vein → axillary vein → subclavian → brachiocephalic → SVC → right atrium → right ventricle (Pulmonary Artery).

In the **right ventricle** and **Pulmonary Artery,** the blood is called mixed venous blood---- sample from all veins.

An example of a catheter used is the **Swan-Ganz catheter**.

QUESTION:

If the pulmonary vein O2 content = **200 ml O2/L blood**

and Pulmonary artery O2 content = **160 ml O2 /L blood**

Lungs add **400 ml O2 /min**

What is cardiac output?

Answer: 400 / (200-160) =**10 L/min**