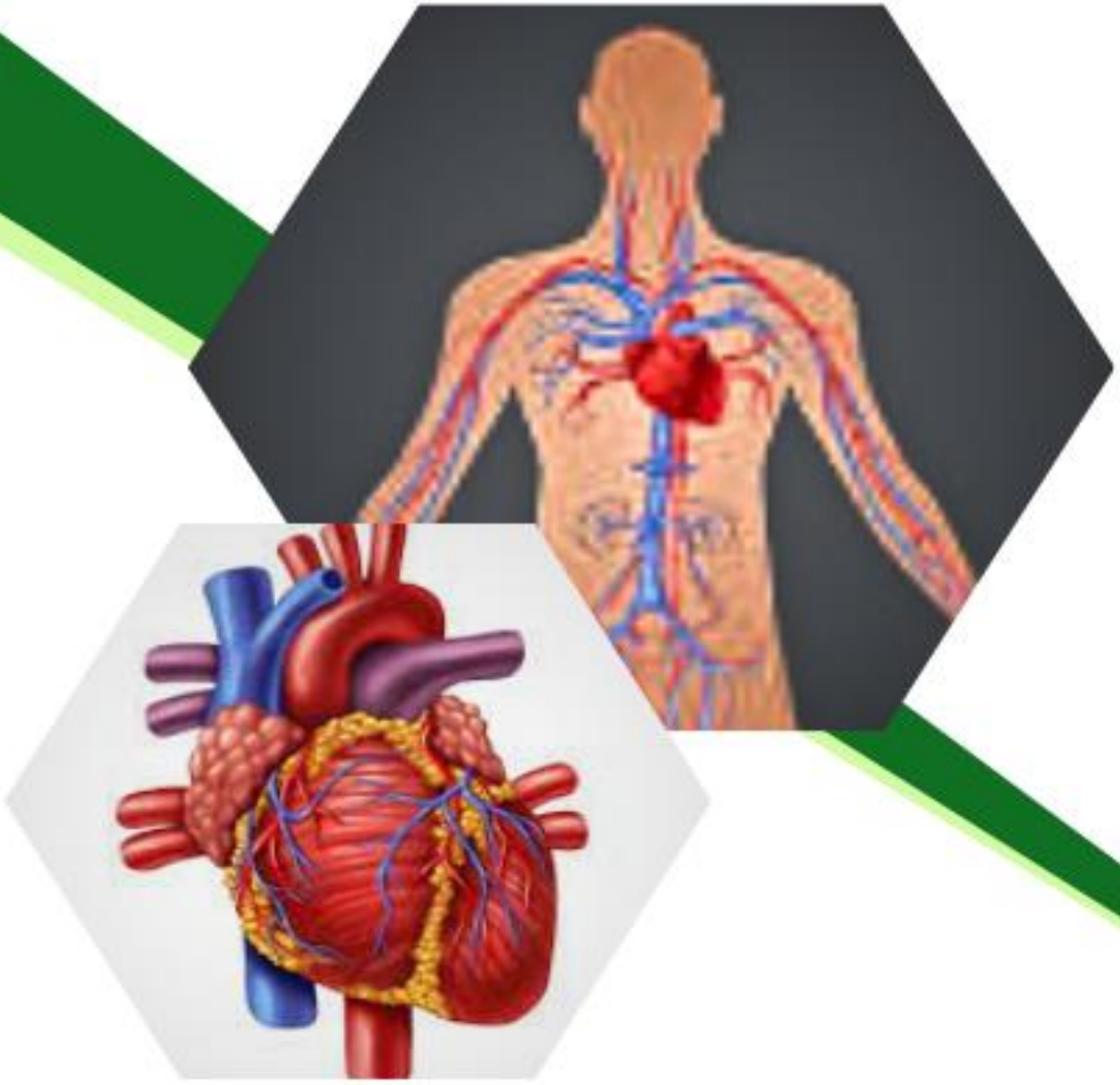
Doctor.021
no.

CVS
PHYSIOLOGY



Writer:

Corrector:

Doctor:

Regulation of Heart Rate

- Positive chronotropic factors increase heart rate.
- Negative chronotropic factors decrease heart rate.

Regulation of Heart Rate: Autonomic Nervous System

- Sympathetic nervous system (SNS) stimulation is activated by stress, anxiety, excitement, or exercise.
- Parasympathetic nervous system (PNS) stimulation is mediated by acetylcholine and opposes the SNS. (stimulated by drugs or by rest & digest).
- PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone.

Atrial (Bainbridge) Reflex

- a sympathetic reflex initiated by increased blood in the atria.
- Causes stimulation of the SA node
- Stimulates baroreceptors in the atria, causing increased SNS stimulation.

For example, someone with too much venous return or we gave him excess fluid, that leads to expansion of the right atria that stimulates the barorecptors on the right atria increase the HT (via increased SNS stimulation) which increases the cardiac output, to get rid of the excess blood inside the atria.

Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate.

Pheochromocytoma patients for example their cells always secrete epinephrine, so they always have high HR & BP.

Hyperthyroidism and Grave's disease also secrete hight amount of thyroxine in contrast to hypothyroidism.

- Intra- and extracellular ion concentrations must be maintained for normal heart function.

Ca++, K+…

Important Concepts About Cardiac Output (CO) Control

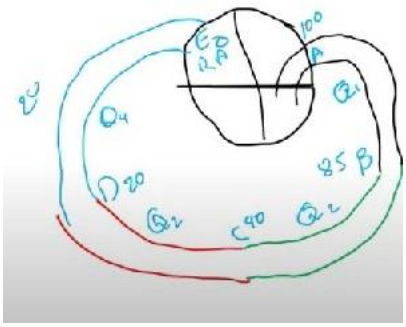
- Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m2 (surface area in m2).

cardiac index is the CO/body surface area.

- CO is proportional to tissue O2 use.
- CO is proportional to 1/TPR when AP is constant.
- CO = (MAP - RAP) / TPR.

We know that the cardiac output in all our vascular system is the same (CO in arteries = CO in arterioles etc…)

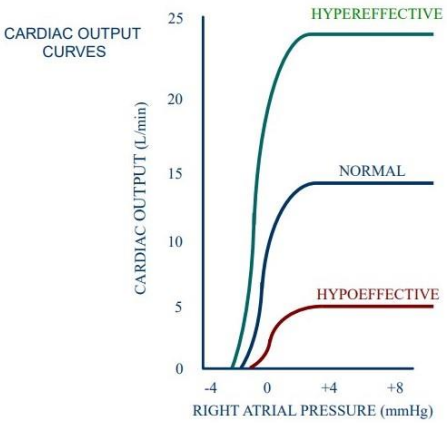
Q1(CO)= Pa – Pb/ Ra – Rb R=resistance



➢ SUMMARY OF DOCTOR'S EXPLANATION:

Using the equation above knowing that Q is equal in all of our vascular system, we can measure how much force we need to push the blood in each segment (which is the pressure difference) & that's how we concluded that the arterioles have the greatest resistance.

- hypereffective in +inotropic or sympathetic stimulation



Ventricular Pressure and Volume Curves

- During the latter part of the ejection phase how can blood still leave the ventricle if pressure is higher in the aorta? Momentum of blood flow
- Total energy of blood = P + mV2 /2 = pressure + kinetic energy
- Total energy of blood leaving ventricle is greater than in aorta.

Ejection Fraction

ΦEnd diastolic volume = 125 ml
ΦEnd systolic volume = 55 ml
ΦEjection volume (stroke volume) = 70 ml
ΦEjection fraction = 70ml/125ml = 56% (normally 60%)
ΦIf heart rate (HR) is 70 beats/minute, what is cardiac output?
ΦCardiac output = HR * stroke volume = 70/min. * 70 ml = 4900ml/min

Ejection Fraction as a tool to classify heart failure: Depending on the percentage measured, and other tests, heart failure may be classed as the following:
•heart failure with preserved ejection fraction (HFpEF) - >50%)
•heart failure with mildly reduced ejection fraction (>40% - 49%)
•heart failure with reduced ejection fraction (<40%)

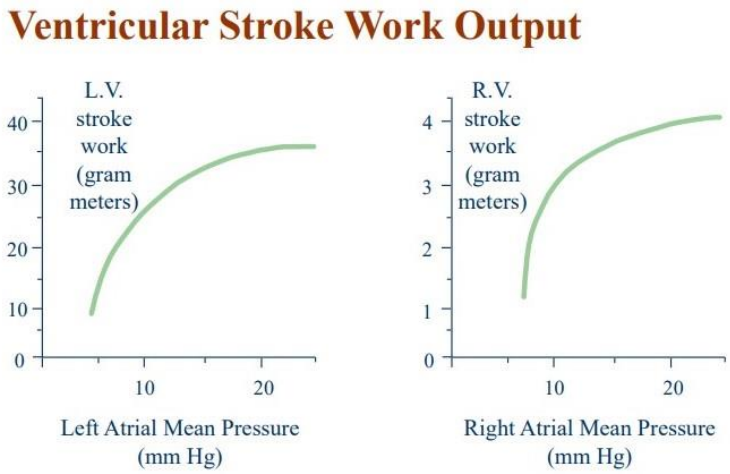
- If HR =100, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is cardiac output?
- C.O. = 100/min. * 160 ml = 16,000 ml/min.
- Ejection fraction= 160/180%=~ 90%

Aortic Pressure Curve

- Aortic pressure starts increasing during systole after the aortic valve opens.
- Aortic pressure decreases toward the end of the ejection phase.
- After the aortic valve closes, an incisura occurs because of sudden cessation of back-flow toward left ventricle.
- Aortic pressure decreases slowly during diastole because of the elasticity of the aorta.

Frank-Starling Mechanism

- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.
- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.



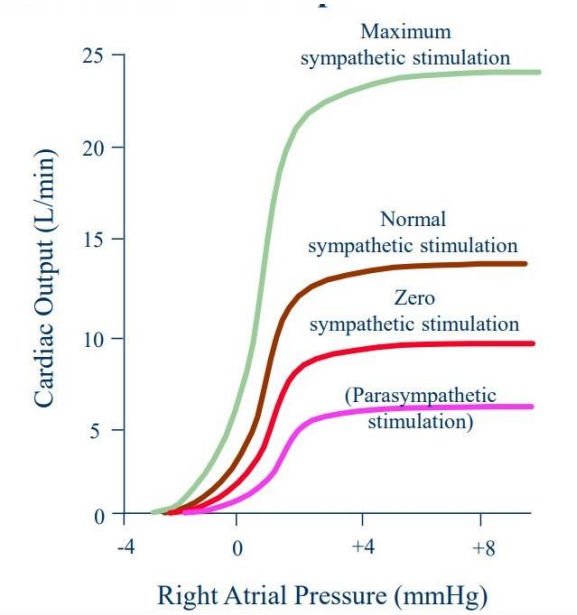
Autonomic Effects on Heart

- Sympathetic stimulation causes increased HR and increased contractility with HR = 180-200 and C.O. = 15-20 L/min.
- Parasympathetic stimulation decreases HR markedly and decreases cardiac contractility slightly. Vagal fibers go mainly to atria.
- Fast heart rate (tachycardia) can decrease C.O. because there is not enough time for heart to fill during diastole.

Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output

To know which nervous system dominates the other we block the receptor chemically (beta1 blockers & m2 blockers). the same thing if we did a cardiac transplant to a patient, The HR is going to be 100 in both cases which is the intrinsic beating rhythm of the SA node.

in normal conditions the HR is 75 so we know the parasympathetic dominates.



Cardiac Contractility

- Best is to measure the C.O. curve, but this is nearly impossible in humans.
- dP/dt is not an accurate measure because this increases with increasing preload and afterload.
- (dP/dt)/P ventricle is better. P ventricle is instantaneous ventricular pressure.
- Excess K+ decreases contractility.
- Excess Ca++ causes spastic contraction, and low Ca++ causes cardiac dilation.

**the doctor skipped most of the slides saying we already talked about it.

CARDIAC OUTPUT AND VENOUS RETURN:

Important Concepts About Cardiac Output (CO) Control

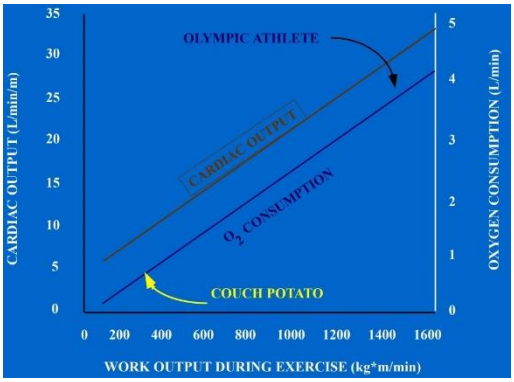
- Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m2).
- CO is proportional to tissue O2. use.
- CO is proportional to 1/TPR when AP is constant.
- F=ΔP/R (Ohm's law)
- CO = (MAP - RAP) / TPR, (RAP=0) then
- CO=MAP/TPR; MAP=CO*TPR

CO=(MAP-RAP)/TPR and as we said RAP=0 and MAP=Pa so:

Pa=Q*TPR(Q:cardiac output) => Q=Pa/TPR

So, if TPR increases cardiac output decreases and vice versa

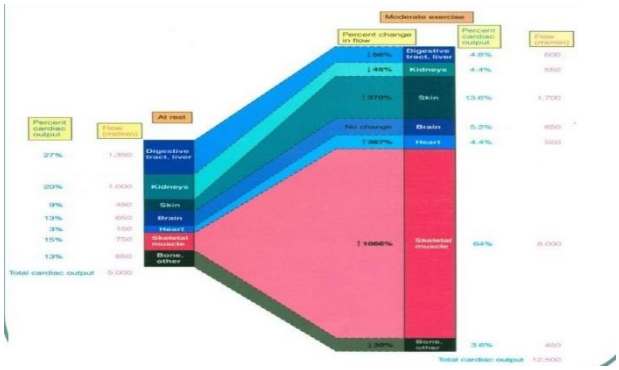
❖ Cardiac output increases depending on oxygen consumption, for example O2 comsumption can increase up to 15 folds from 250ml/min (as we said) to3.5L during exercise.

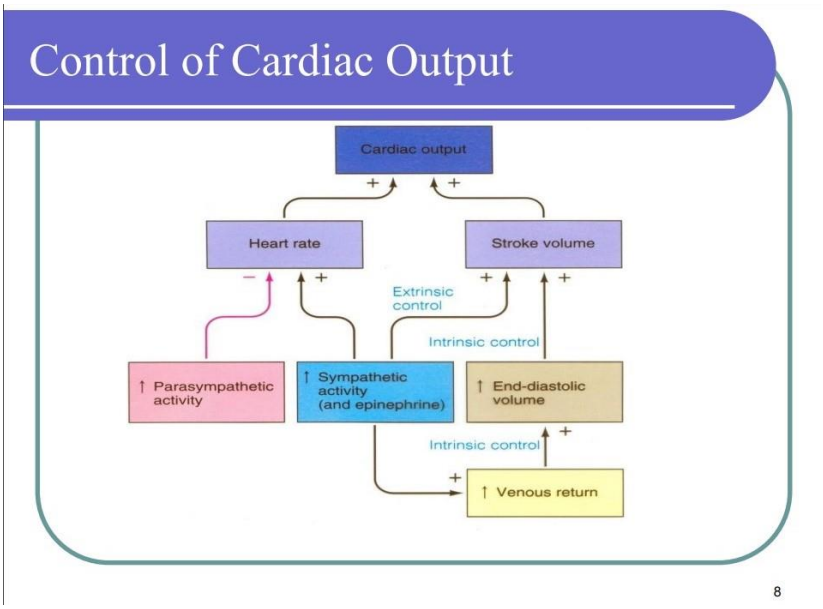


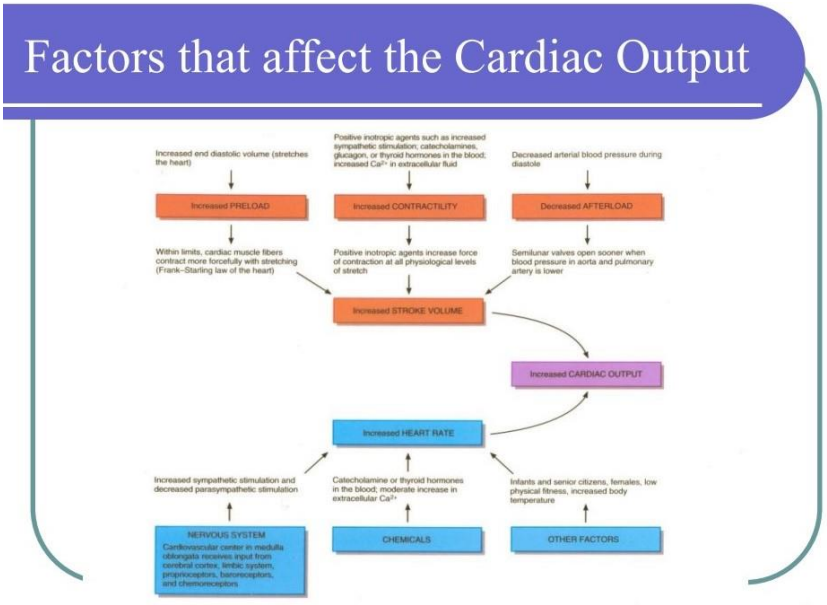
Blood flow (ml/g/min)

Tissue	Blood flow (ml/g/min)	A-V difference (Vol %)	Flow ml/min	O_2 consumption ml/min
Heart	0.8	11	250	27
Brain	0.5	6.2 (25-30% Extraction)	750-900	
Skeletal Muscle	0.03	6	1200	70
Liver	0.6	3.4 Reconditioner organ		
SKIN	0.1			
Kidney	4.2	1.4	1250	18
Carotid bodies	20	0.5	0.6	

- But the cardiac output does not increase 15 folds on its own, both Extraction ratio and blood flow increase, to provide the oxygen needed in exercise
- Blood flow the kidneys as well as GU tract decreases during exercise, while it increases to the heart.

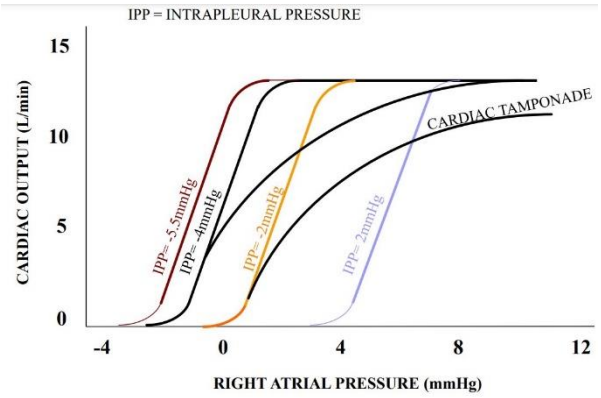






Normal IPP (intraplural pressure)=-4

VR=Q=5L (venous return= cardiac output) and its defined as the blood that returns to the heart per minute.



- There are 6 factors that affect VR
  1- Pressure in the veins is more than the pressure in the right atrium. So if the right atrial pressure this decreases the VR, while if the venous pressure increases (blood volume increases for example), also venous constriction increases the venous pressure cause Pv=F/A(pressure=force/area)
  2- Valves in the veins allow blood to go in one direction toward the heart
  3- Deep veins are surrounded by muscles, and the contraction of these muscles squeezes blood to go toward the heart
  4- Sympathetic stimulation.
  The doctor didn't mention the other two factors

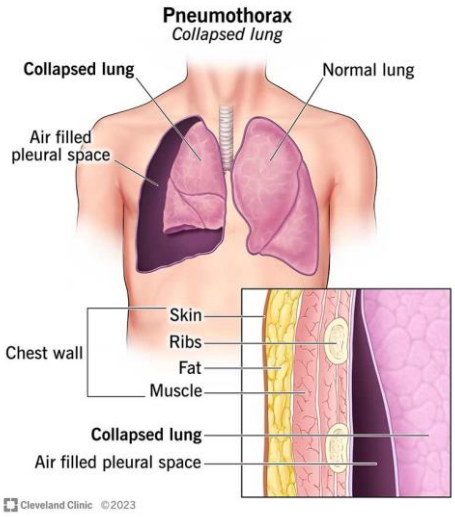
Plateau of CO curve determined by heart strength (contractility + HR)

- Sympathetic ⇒ plateau
- ↓ Parasympathetic (HR) ⇒ (? plateau)
- Plateau
- Heart hypertrophy's ⇒ plateau
- Myocardial infarction ⇒ (? plateau)
- ↓ Plateau

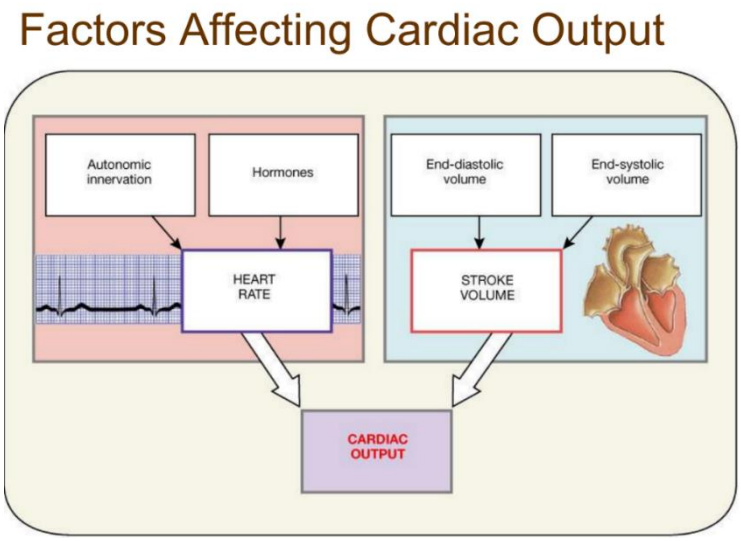
Valvular disease ⇒ ↓ plateau (stenosis or regurgitation)

- Myocarditis ⇒ ↓ plateau
- Cardiac tamponade ⇒ (? plateau)
- ↓ Plateau
- Metabolic damage ⇒ ↓ plateau

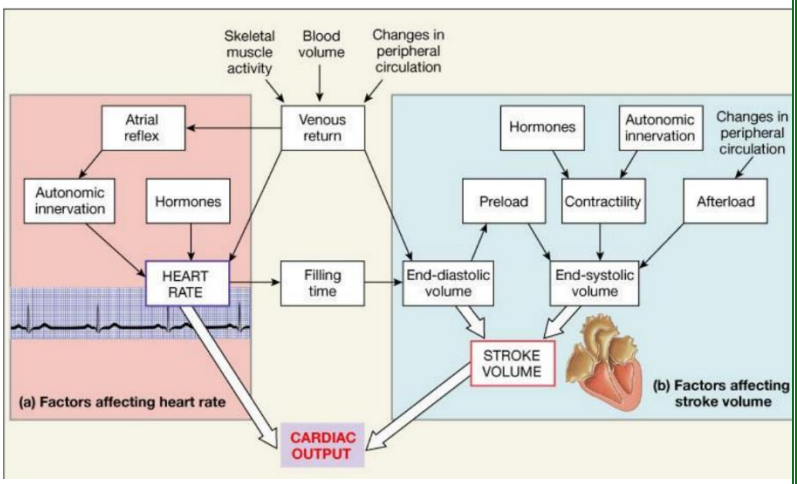
- Now let's clarify the concept of inspiration and expiration, during inspiration, the abdominal cavity become smaller and the thoracic cavity becomes larger to accommodate the air inside the lungs, this compression in the IVC distally (in the abdomen) will increase the difference in pressure so more blood flow toward the right atrium.
- In the case of pneumothorax, there is an abnormal collection of the air in the pleural space between the lungs and chest wall, increasing the pressure inside that space will disrupt the normal negative pressure inside the space which is needed for its normal function, this pressure will compress IVC distally and alter the pressure difference, making it equal to zero, so no blood flow, so venous return will decrease significantly, as we talk in pneumothorax, you have increased IPP (intrapleural pressure), how is that reflected in the right atrium pressure-CO diagram? The curve will be shifted to the right in order to have

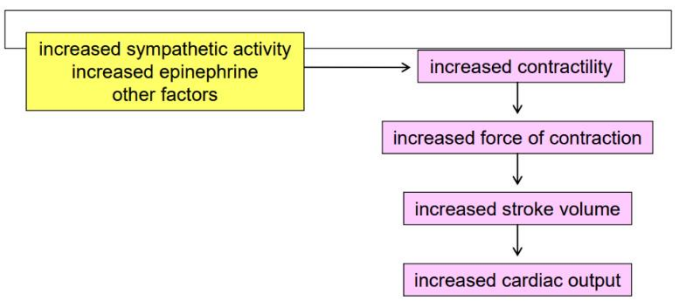


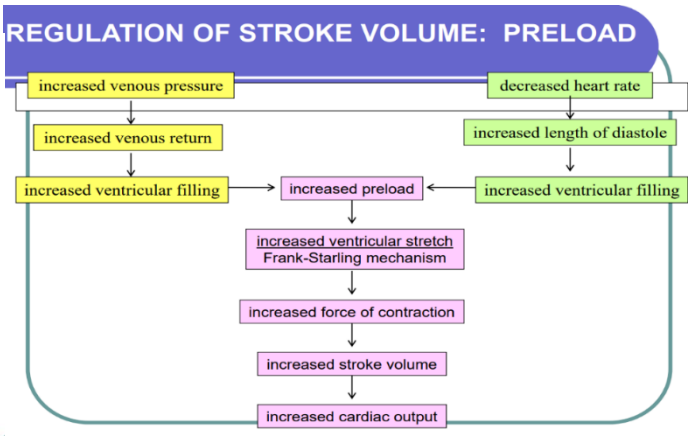
the same CO meaning that a higher level of atrial pressure should be reached in order to attain the normal functional level, in contrast, cardiac tamponade will shift the curve downward.



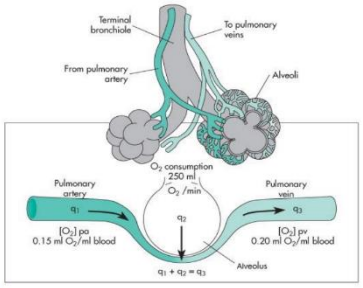
- Quick recap: as you know from this equation CO = SV × HR, so it is affected by two factors, HR which is affected by sympathetic and parasympathetic and hormones. SV which is affected by end-diastolic volume, preload, afterload, end systolic volume.







Explanation for this pic :



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

Q1 represents blood that came from the right ventricle and pulmonary art.
→ q1 = CO * Cvo2

So according to the equation:

*Q1=5 L *15 ml o2 →750ml of o2

Q3 represents blood going to left heart and arterial system.

→ q3 = CO * Cao2 = q1 + q2

So according to the equation:

CO*Cao2=CO*Cvo2+oxygen up take

Bring the CO to same side of equation you will get →CO(Cao2-Cvo2)=oxygen up take

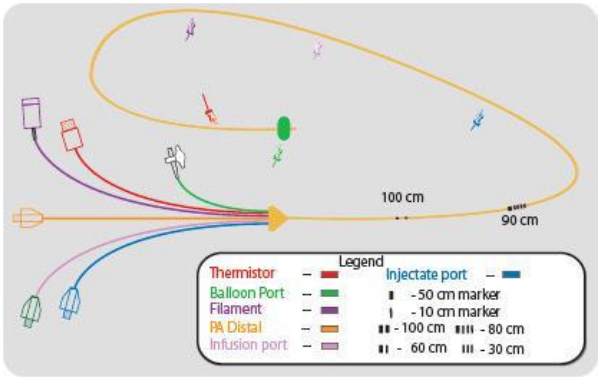
Let's make CO ALONE IN ONE SIDE OF THE EQUATION→

CO=oxygen uptake /(Cao2-Cvo2)

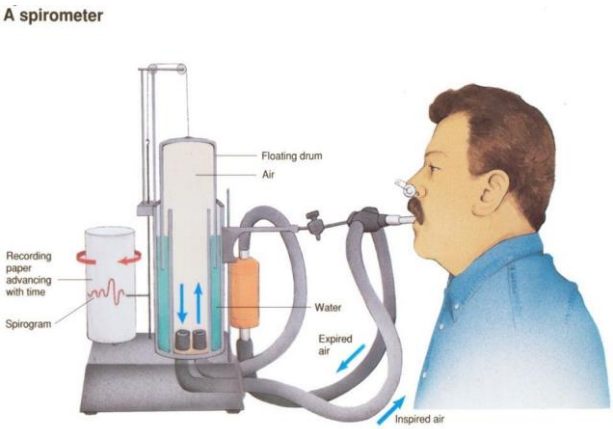
And now u can see the slides it say the same and solve the problem after that test your self

q1=CO*CVO2
q2=amount of Oxygen uptake by the lungs
q3= CO* CAO2 and equals = CO*C VO2+ O2 uptake
Oxygen uptake = CO{CAO2-C VO2}
CO=Oxygen uptake/{CAO2-C VO2}

- so how we can measure the CO?
  - Swan-Ganz catheterization is a choice (also called right heart catheterization or pulmonary artery catheterization) is the passing of a thin tube (catheter) into the right side of the heart and the arteries leading to the lungs, Thermodilution methods can be used to estimate cardiac output. Cold saline is injected through a port in the catheter, and the change in blood temperature is detected as it passes through the pulmonary artery. This information is used to calculate cardiac output.



  - Another way to calculate CO by using spirometer, first of all, the pulmonary artery that is going to lungs for taking oxygen and being saturated then goes back to the left atrium, q1 refers to the concentration of the oxygen before saturation, q2 refers to the concentration of oxygen taken up by the lungs and will be added to the q1 to form q3, by using spirometer which is used to measure the volume of air inspired and expired by a person in a given time, enabling you to measure the oxygen content in both, then by calculating the oxygen difference (oxygen uptake), by using fick principle you will get the result.



O2 Fick Problem

If pulmonary vein O_2 content = 200 ml O_2/L blood

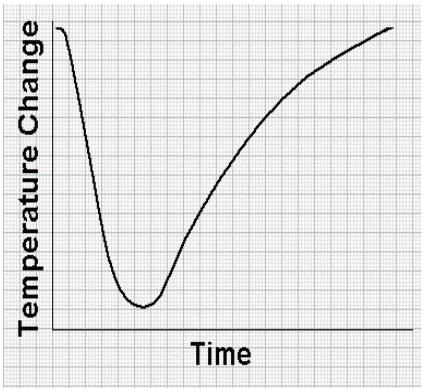
Pulmonary artery O_2 content = 160 ml O_2 /L blood

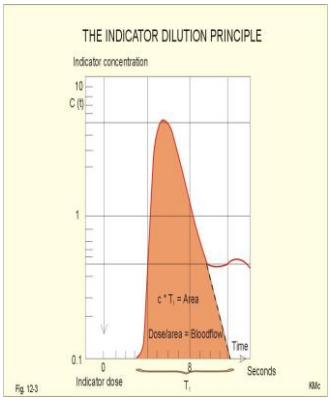
Lungs add 400 ml O_2 /min

What is cardiac output?

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

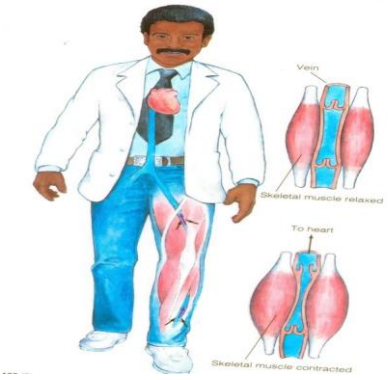
The thermodilution principle not required (the next two pics)

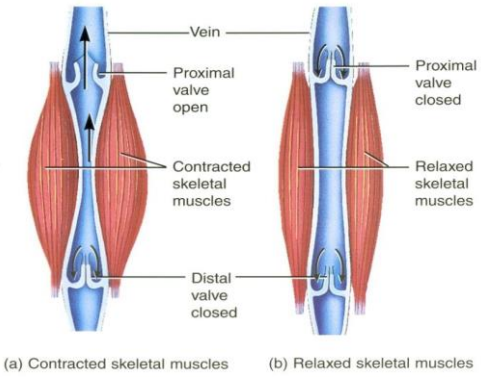




Effect of Venous Valves

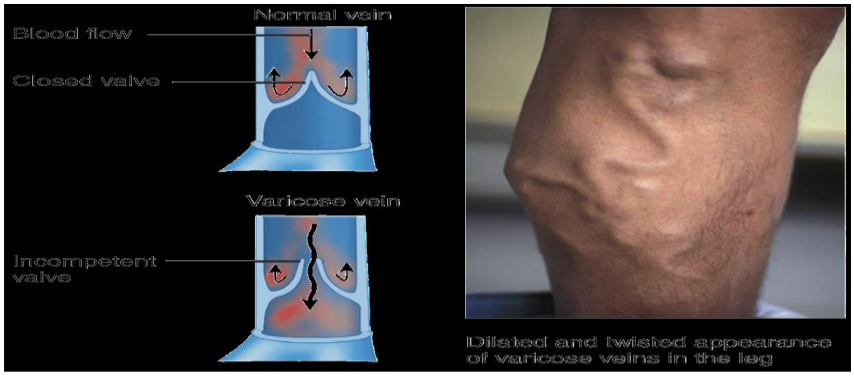
the valves are found in veins and not the arteries and they insure the one direction movement of the blood





Effect Of Gravity on Venous Pressure

During the pregnancy of the woman the presence of the baby within the uterine may cause compression to the veins and may cause edema (the doctor said u took this in pathology … smile)

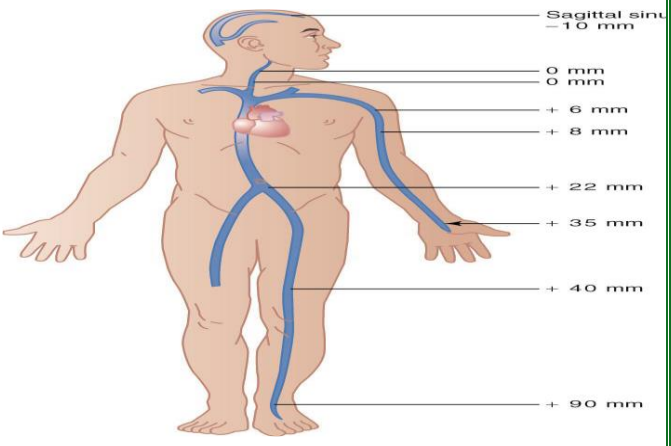


The doctor said to read the last slides alone

Good luck

Venous Pressure in the Body

- Compressional factors tend to cause resistance to flow in large peripheral veins.
- Increases in right atrial pressure causes blood to back up into the venous system thereby increasing venous pressures.
- Abdominal pressures tend to increase venous pressures in the legs.

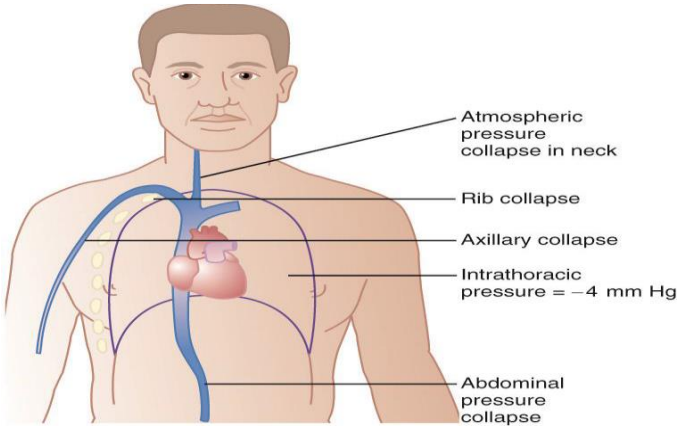


Central Venous Pressure

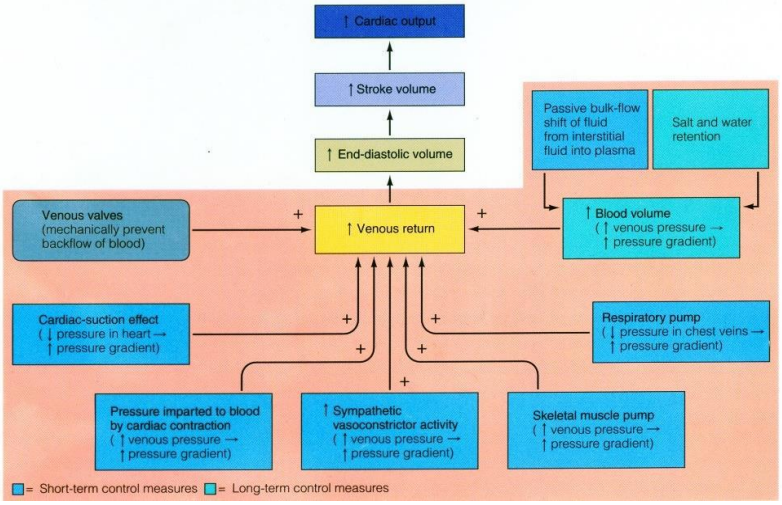
- Pressure in the right atrium is called *central venous pressure*.
- *Right atrial pressure* is determined by the balance of the heart pumping blood out of the right atrium and flow of blood from the large veins into the right atrium.
- Central venous pressure is normally 0 mmHg, but can be as high as 20-30 mmHg.

Factors affecting Central Venous Pressure

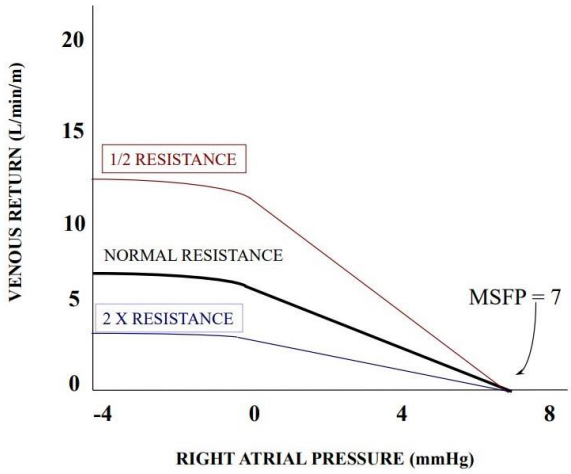
Right atrial pressure (RAP) is regulated by a balance between the ability of the heart to pump blood out of the atrium and the rate of blood flowing into the atrium from peripheral veins.
Factors that increase RAP:
-increased blood volume
-increased venous tone
- dilation of arterioles
-decreased cardiac function

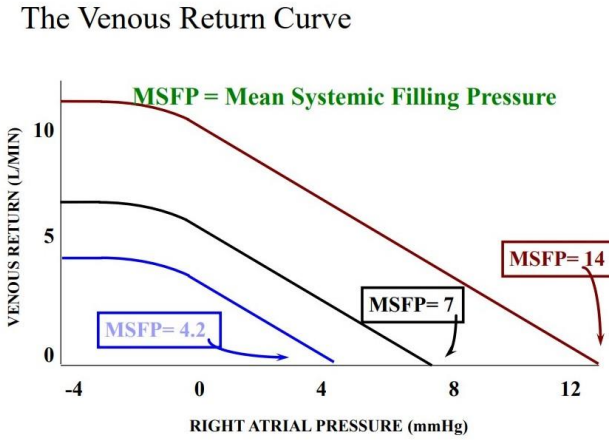


Factors that Facilitate Venous Return



The Venous Return Curve





VR

- Beriberi - thiamine deficiency → arteriolar dilatation → decrease RVR
- (RVR= resistance to venous return) because VR = (MSFP - RAP) /RVR (good for positive RAP's)
- A-V fistula → (? RVR)
  Decrease RVR
- C. Hyperthyroidism → (? RVR)
  Decrease RVR
- Anemia ⇒ ↓ RVR (why?)
- Sympathetics ⇒ MSFP
- Blood volume ⇒ MSFP + small
  ↓ in RVR
- ↓ Venous compliance (muscle contraction or venous constriction) ⇒ (? MSFP)
  MSFP

Factors Causing ↓Venous Return

- Blood volume ⇒ ↓ MSFP
- ↓ Sympathetics ⇒ (? v. comp. and MSFP)
- Venous compliance and ↓MSFP
- Obstruction of veins ⇒ (? RVR)
  RVR