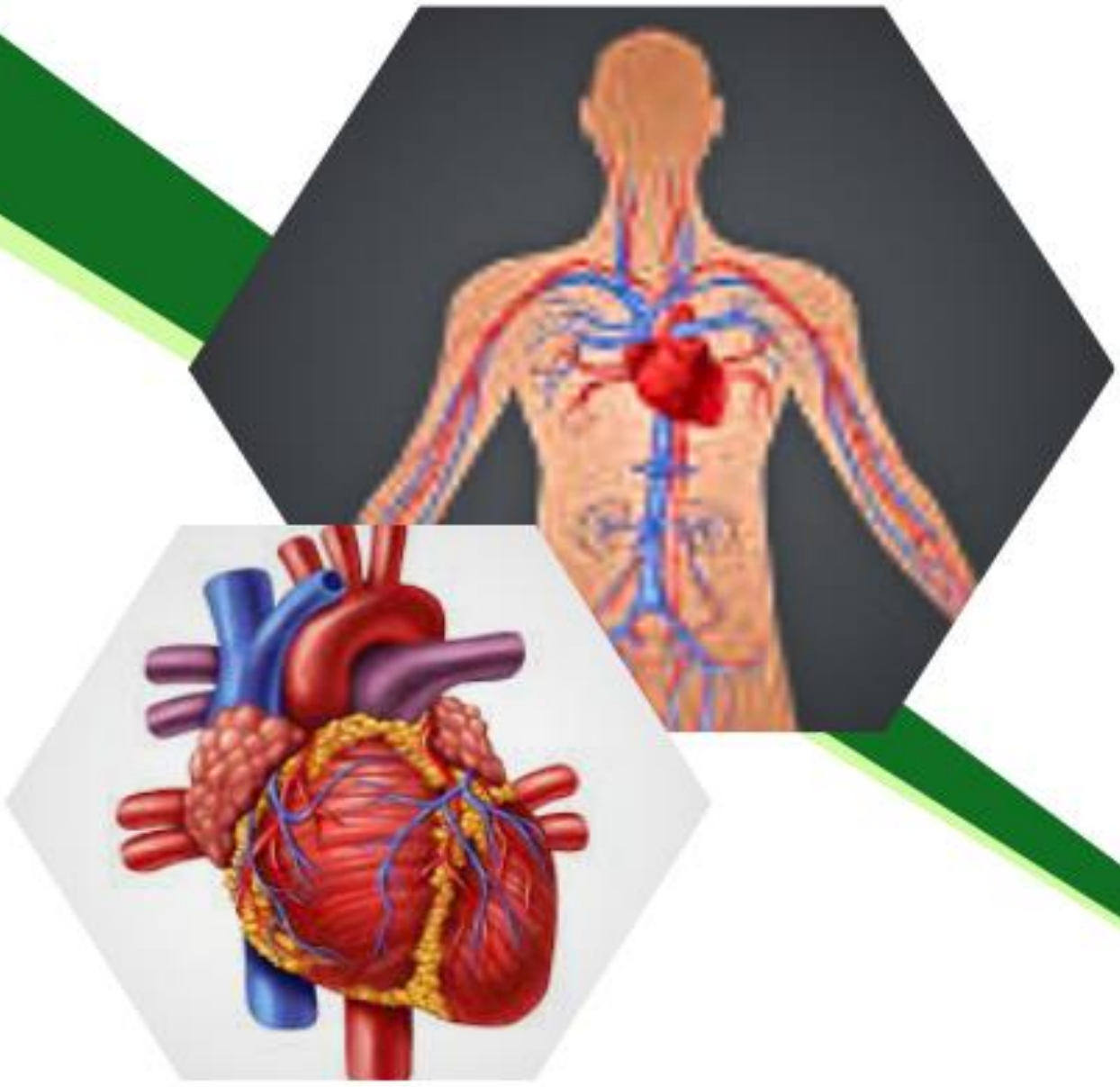
Doctor.021

no. 11

CVS

PHYSIOLOGY



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Cardiac Cycle: (**7phases**)

- Atrial systole 0.1 second
- Atrial diastole 0.7 second
- Ventricular systole 0.3 second

1 ▪ Isovolumic contraction 0.01 seconds
2 ▪ Rapid ejection period
3 ▪ Slow ejection period

- Ventricular diastole 0.5 seconds

4 ▪ Isovolumic relaxation 0.02 seconds
5 ▪ Rapid filling
6 ▪ Slow filling (Diastasis)
7 ▪ Atrial contraction.

HR	Systole	Diastole
60	0.3	0.7
75	0.25	0.55
200	0.19	0.11

Each person has his own optimal maxHR.

As you note: when the HR increased, the diastolic time(filling time) will be expensed(decreased), as a result the SV and the CO will decrease.

Ejection fraction = SV/EDV ***100%**

End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)

SV X heart rate (HR) = cardiac output (CO)

Phases of the Cardiac Cycle

- Ventricular filling – mid-to-late diastole

– Heart blood pressure is low as blood enters atria and flows into ventricles.

– AV valves are open, then atrial systole occurs (the atrial contraction) it is the last step in diastole.

● Ventricular systole

– Atria relax

– Rising ventricular pressure results in closing of AV valves

– Isovolumetric contraction phase

– Ventricular ejection phase opens semilunar valves

-when the atria contract the ventricles will be relaxed.

(atrial systole spends 0.1sec, while the atrial diastole spends 0.7sec)

● Isovolumetric relaxation

– early diastole

– Ventricles relax

– Backflow of blood in aorta and pulmonary trunk closes semilunar valves.

Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves.

Remember: the valves closure is audible.

Changes during Cardiac cycle

● Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.

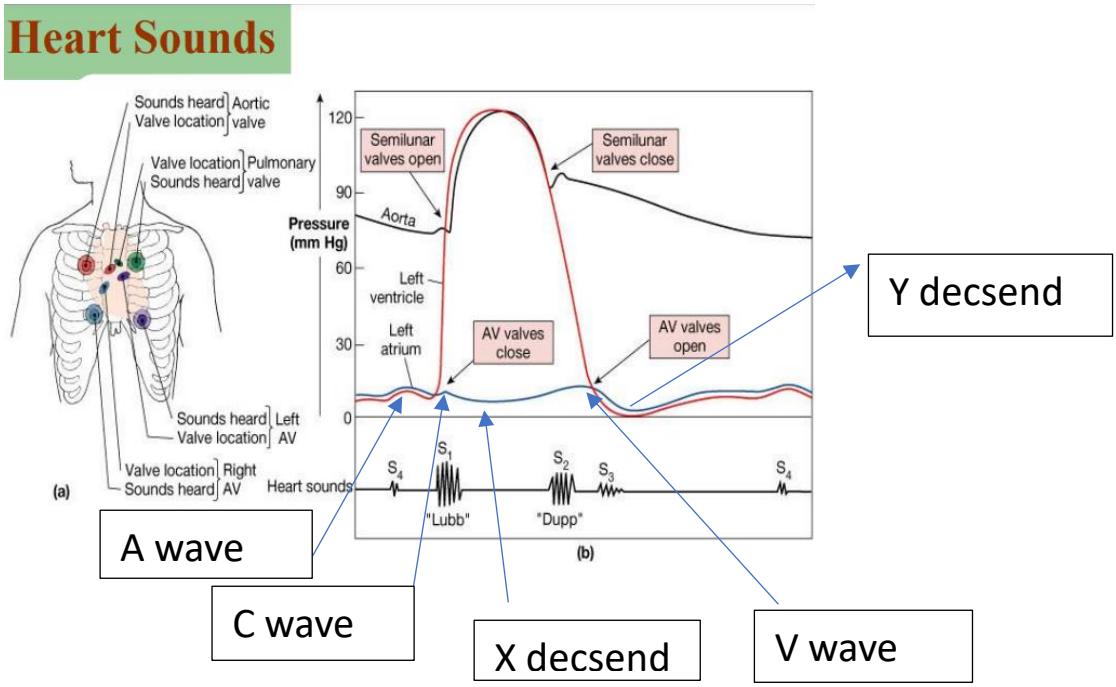
● Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic

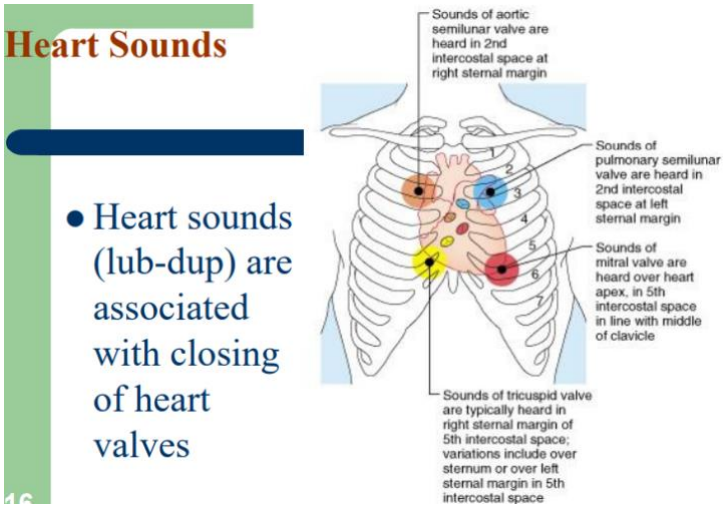
● Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.

● Atrial pressure: A wave =atrial systole**(contraction)**, C wave= ventricular contraction (AV closure), V wave= ventricular diastole **because of venous return** (Av opening).

-a-c-v waves. Bcs there is no valves between the Rt atrium and the SVC, the pressure waves in the Rt atrium are transmitted to the central veins which are distensible. They reflect a window inside the heart. a-wave at the end of the ventricular diastole “a” stands for atrial contraction, c-wave “c” stands for cusps, “v” stands for venous filling the atrium with blood.

(a-c-v waves) we can call them atrial waves or venous waves because we can see them in the internal jugular veins.





These the locations where we can listen to the heart sounds to detect if there are murmurs or not. (the murmurs are 2 types diastolic or systolic)

Heart sounds

- Auscultation – listening to heart sound via stethoscope
- Four heart sounds: Closure the valves make noise (S1 and S2). Opening of valves is silent
- S_1 = turbulence of blood around a closed AV valves, S_2 = turbulence of blood around a closed semilunar valves.
  - S_1 – "lubb" caused by the closing of the AV valves
  - S_2 – "dupp" caused by the closing of the semilunar valves
  - S_3 – a faint sound associated with blood flowing into the ventricles. During the rapid filling phase in adults and after exercise S3 might be heard (due to rapid venous return). This sound might be physiological or might be pathological.
  - S4 – another faint sound associated with atrial contraction. In stiffed ventricles, the hypertrophic atrium has to work harder and pump blood against a stiffed ventricle, atrial contraction phase might produce S4. This sound is always pathological.

S_3: Early diastole during rapid filling → turbulent flow→S3. It is normal in children where they have hyper dynamic state of blood flow. It is seen in mitral incompetence due to increase blood flow to the left ventricle. In heart failure it gives a characteristic **gallop rhythm (triple rhythm خبب)**, caused by ventricular dilation.
Ventricular filling is normally silent. This heart sound is normal in children; but is often pathological in adults and caused by ventricular dilation.
Seen in 1. Childern, 2. HF, and 3. mitral incompetence
S_4: This can only be recorded by phonocardiogram. We cannot hear it even in pathological conditions. It is recorded at late diastolic phase duo atrial contraction. It is inaudible to the naked ear. Is caused by vibration of the ventricular wall during atrial contraction. Generally, it is noted when the ventricle compliance is reduced ("stiff" ventricle) as occurs in ventricular hypertrophy and in many older individuals

-S3 can be physiological or pathological sound (sometimes in heart failure we can sound the **gallop** (tribble sound))

-while S4 is always pathological sound.

Factors Affecting Stroke Volume

- Preload – amount ventricles are stretched by contained blood.

the greater the size of EDV is, the more elongation it exerts on sarcomere providing stronger contractile behaviour and therefore the ejected volume.

- Afterload – back pressure exerted by blood in the large arteries leaving the heart.
- Contractility– cardiac cell contractile force due to factors other than EDV and afterload.

Contractility describes the how efficient the mechanical pumping behaviour the ventricular myocytes exhibit upon contraction given fixed preload (EDV) and therefore sarcomere length, and afterload (Intra-aortic pressure provided by blood forcing vessel walls, this demands additional work by the heart).

- Key role controller of contractility is the calcium availability, and sensitivity of Troponin C towards it.
- Here we give rise to a question: which is healthier, efficient-pumping, stronger heart, child or adult one.
  Despite having greater CO of 5L among adults compared to 3L CO among youngers, this is not enough to have the clear

answer, instead we refer to the standard cardiac index, we divide the CO by the BSA (individual body surface area) in litres. So, in our example, supposing an adult BSA of 2 L, and chid BSA of 1 L, so we end by a greater cardiac index and more forceful child heart with CI (cardiac index) of 3 degrees compared to the adult CI of 2.5 degrees.

Frank-Starling Law of the Heart

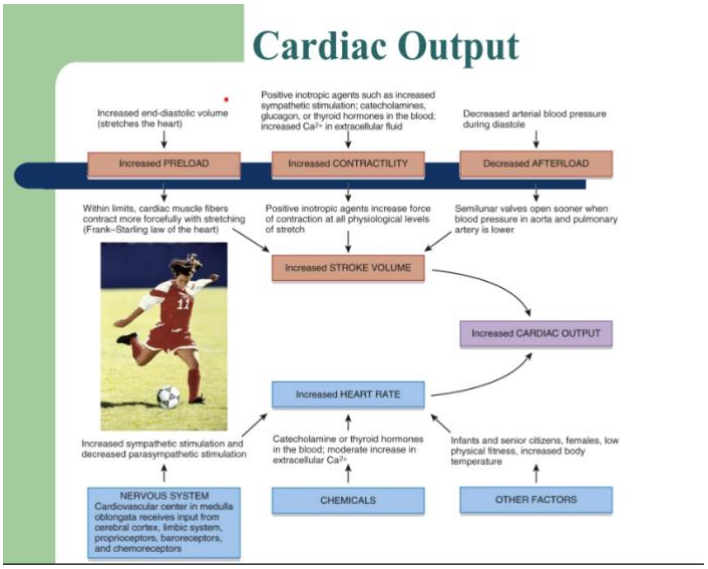
- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume.
- Slow heartbeat (prolonged diastolic filling phases and larger EDV and longer sarcomeres, finally more SV) and exercise increase venous return to the heart, increasing SV.

Marathoners have SV of 100 mLpB and HR of 50 BpM , therefore CO of 5000 mLpM at rest

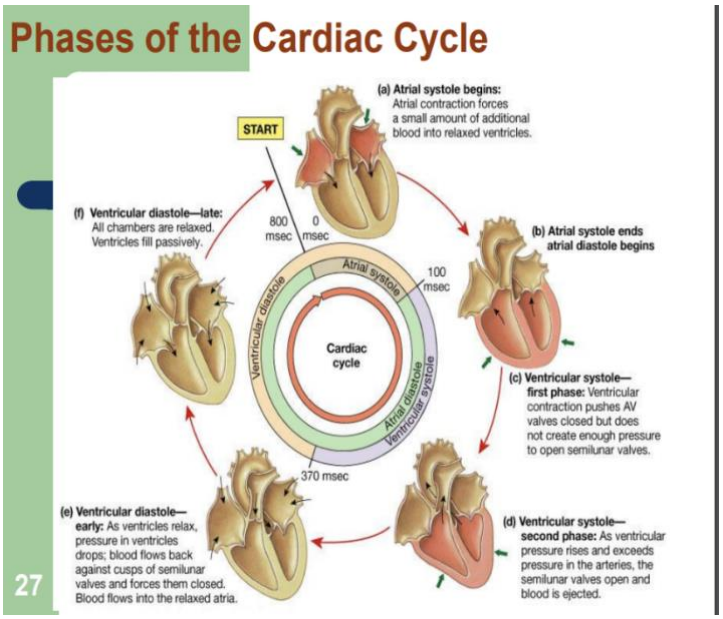
- Blood loss and extremely rapid heartbeat decrease SV

Again, by the role of Diastolic time and EDV.

- Notice that the positive inotropic effect induced by (sympathetic stimulation, digoxin, etc) increases SV by intracellular Ca^{2+} holding, beside the positive chronotropic effect (on HR) which ultimately increases CO.



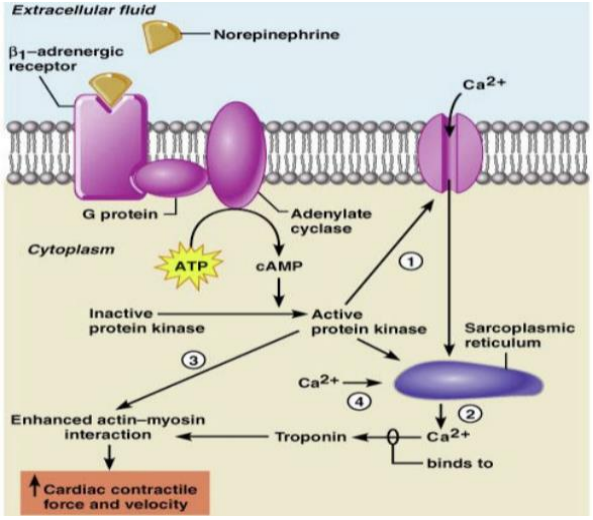
Again, and again ☺
Remember that atrial-ventricular diastolic filling is predominantly (by its 80%) a passive procedure, whereas arial active contraction contributes to only 20% in the last phase of diastole.
We survive without this active filling phase and the condition is called Atrial fibrillation; this is when diastolic period is reduced to compensate with the dramatic rate of (350-600)BpM.
However, patient should be kept under treatment with B-blockers (atenolol) and Ca^{2+} channel blocker.
Atrial flatter is characterized by tachyarrhythmic rate btwn (250-350) BpM .



Extrinsic Factors Influencing Stroke Volume

- Contractility is the increase in contractile strength, independent of stretch and EDV.
- Increase in contractility comes from: – Increased sympathetic stimuli – Certain hormones (T3, T4, Glucagon) – Ca2+ and some drugs.
- Agents/factors that decrease contractility include: – Acidosis (high H^+) – Increased extracellular K+ – Calcium channel blocker.

The picture beside represents epinephrin-mediated intracellular mechanism, which is responsible for achieving sympathetic cardiac effect, NOT REQUIRED.



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	

- The total circulating ejected blood volume per minute also called systemic blood flow or the cardiac output (is 5 L, it is distributed on organs based on their rest levels of demands as illustrated in the previous table:

Normally, Brain and skeletal muscle and kidney each consumes 1 L. The remaining systemic 2 L are supplied to GIT (1.25 L), heart (0.25 L).

Artero-venous O_2 difference ($O_{2(a)}$-$O_{2(v)}$) of a tissue is an indication of its Oxygen consumption.

In the same context the average total body O_2 consumption (metabolic indicator) can be measured as the difference between A: any arterial points, as parallel vessel distribution ensures similar blood composition all through the body before supplying organs.

B: the most accurate is mixed venous blood in pulmonary artery which allows us to scan the whole-body demand of O_2 by examination of terminal O_2 left over in the venous systemic blood drained by caval system, taking in consideration also the drainage of:

1)heart by coronary (thebesian) sinus to the right atrium towards Pulmonary trunk. (So that we can also have a look to the Cardiac O_2 consumption, drainage of the heart can end in its left also.

- however, we can refer in this left-over venous composition to previous cardiac points, right atrium, or right ventricle. But for sure, the later the venous measuring point is, the more accurate our scan of whole systemic and cardiac O_2 consumption (that's why we prefer pulmonary trunk that further ensures the consideration of:

2) Deoxygenated venous bronchial drainage, which is normally through systemic veins, to the right atrium, but with intrapulmonary structural connection between pulmonary and systemic circulation deoxygenated blood may leak to oxygenated one, blood is then called polluted.

As you know the right atrium receives blood from the SVC and IVC (the two differ in their composition) then the blood goes to the right ventricle and then to the pulmonary trunk, and if I take a sample of the blood in the pulmonary trunk it will surely represent the average of the venous blood in the body, and it's called **mixed venous blood ($\bar{v}$)** and its conc. is 15ml O2/dl.

O2 $\bar{v}$: is the average arterial blood, taken from the limbs and it contains 20ml O2/dl (the average conc. of the arterial blood).

Mixed venous blood conc. - average arterial blood conc.=

20ml O2/dl - 15ml O2/dl = 5ml O2/dl (taken by the capillaries to cells, the amount of oxygen used by our bodies for metabolism)

- Arterial oxygen concentration is 20ml O2/dl (constant for all organs).
- Mixed venous oxygen concentration is 15ml O2/dl.
- Capillaries' oxygen concentration is 5ml O2/dl.

So, the capillaries extracted 5ml O2 from the arterial blood (20ml o2), and **the extraction ratio** is 5/20 or 25%, meaning that from every dl blood, 5ml O2 is extracted.

If we want to calculate the **oxygen consumption ($\dot{V}O_2$)** of the body, we multiply the cardiac output (CO= 5 litres/min or 5000ml blood/min) by the oxygen consumption concentration of the capillaries (5 ml O2/dl or 5ml o2/ 100ml).

So, **oxygen consumption ($\dot{V}O_2$) =** 5000ml blood/min * 5ml O2/100ml = 250ml O2/ min (which is amount of oxygen our body consumes per min).

Another way to calculate it; 5 litres = 50dl/min (CO) and from every dl, 5ml o2 is taken, **oxygen consumption ($\dot{V}O_2$)=** 50dl blood/ min * 5ml O2/ ml= 250ml O2/ min.

But how to know the oxygen consumption per min for a specific organ or tissue?
We calculate the amount of oxygen that enters the organ and then subtract from it the amount of oxygen that exits that organ by the veins.

- To know the amount of <u>oxygen</u> that entered the organ, first I need to know the amount of arterial <u>blood</u> that enters the organ (**Qc)**, (which is the same as the amount of venous blood that exists that organ, the difference is in the composition, O2 decreases) then we multiply it by arterial oxygen concentration

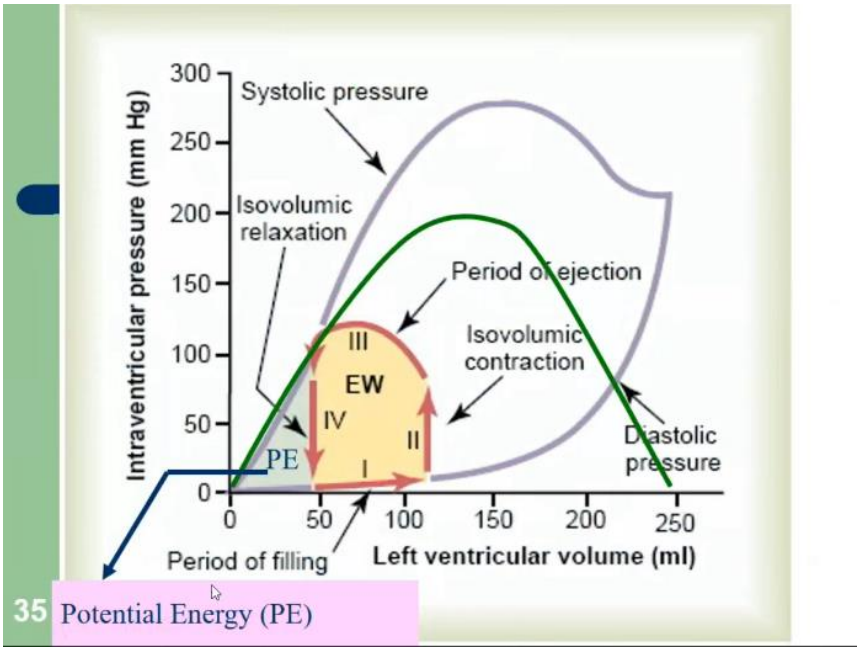
which is constant (20ml O2/dl) , by this we know the amount of oxygen that enters that organ.

- And we do the same to know the amount of oxygen that goes out of that organ, Qc (the same one we used before) * venous oxygen concentration of that organ.
- Then we subtract the veinous oxygen content of that organ from the arterial oxygen content for the organ to get the oxygen consumption for it. And this is called the **arteriovenous oxygen difference [a-v]$_{O2}$,**

If the arterio-venous oxygen difference is small, like in the carotid bodies, where the arterial oxygen concentration is 20 (constant) and the venous oxygen concentration is 19.5, the difference is .5ml O2/ min, this means that the blood which goes to the carotid bodies is much more than it need, so the blood goes there not to just supply the carotid with oxygen, it rather goes for other purposes. (the doctor said he doesn't want to mention it now •ᴗ•)

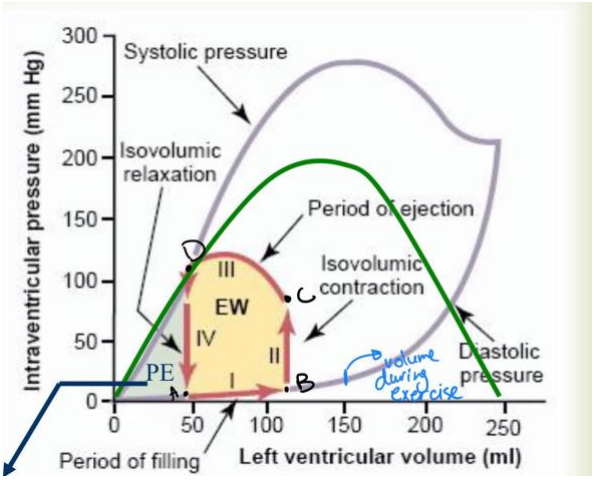
- The carotid bodies and other organs with small arterio-venous oxygen difference are called **reconditioning organs or tissue** because of its role in regulation and homeostasis of blood, another example is the kidneys their arterio-venous oxygen difference is 1.4ml O2/min.
- The skeletal muscles' arterio-venous oxygen difference at rest is 5ml O2/min (venous oxygen concentration is 15), while during exercise skeletal muscles extract more oxygen 10-20 (if it was 20 it would have extracted 100% of the oxygen remember the arterial oxygen concentration itself is 20).
- The heart (our most important example)
  In the heart the arteriovenous O2 difference is 11 (9 ml/dl O2 left in the vein after it was 20 in the coronary arteries), what does that mean ? If a slight reduction in O2 supply happens or increase in oxygen demands for any reason it will subject the heart to hypoxia then ischemia, and that's unlike reconditioning

organs which get more than their oxygen needs already so a decrease in supply (to some extent) wont affect them, for example we have the kidney if its oxygen supply goes down to half it wont become ischemic.



- Why do we care for oxygen consumption ?
  Because it will gives an idea of how much work the heart has to preform, more oxygen consumption -> more work

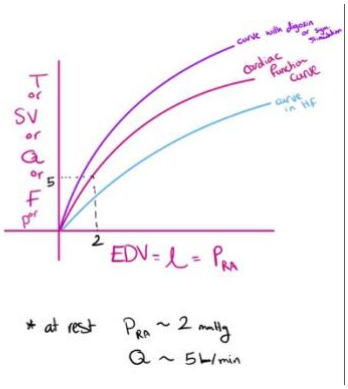
  Work (W) = P*V
  If volume increases work increases (the area under the curve is increased)
  P=T(tension)/r —> T=Pr/W
  In conclusion tension reflects oxygen consumption, dilation increases tension which increases oxygen demand and sequentially blood demand, if blood profusion didn't increase it will lead to ischemia.
  The same principle applies on hypertensive patients the heart tries to protect itself from hypoxia by increasing the ventricular thickness two to three times the normal to reduce the tension



Now looking In the diagram above, the curve (in grey) is generated by diastolic function (below) and systolic function (above), if we put A,B,C,D, d is derived from the systolic curve , now if we imagine a person who is doing exercise , know actually during exercise the skeletal muscle contraction will push more blood in the veins , so here the venous return is increasing , and this will increase the SV if there is no other changes happens but actually that's not the situation inside our bodies , its more complicated and many factors will change at the same time :)

Now the doctor continued the lecture on his own drawings so I draw them , please check the doctor's record for better explanation .

here its multiaxial diagram , you can choose any two factors you will find the same relationship !



T : tension , SV: stroke volume , Q: cardiac output , F: force of contraction.

EDV: end diastolic volume, L: length of sarcomere, P_{RA} : right atrial pressure

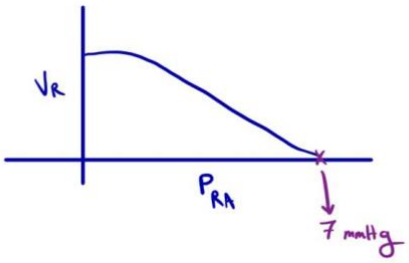
this is called cardiac function curve , if we have failure it will shifted to the right , and it will shifted to the left with sympathetic stimulation or digoxin intake.

we have 4 cardiac chambers , one of them is the right atrium , from the previous diagram we know that increasing P_{RA} will increase the contractility .

P_{RA} depends on the blood that comes from veins , or what's called venous return (VR).

VR: is the volume of blood that returns to the heart per minute .

It depends on the pressure difference btw veins and right atrium , if this difference decreases their will be small or no return , so the relation btw P_{RA} and VR is inverse relationship as shown in this picture.



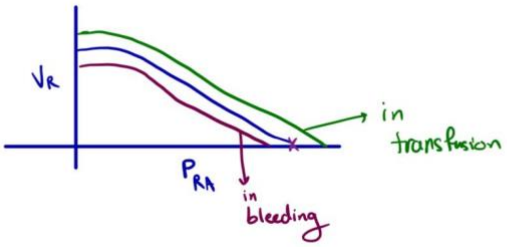
VR= Pv -P_{RA}/ Rv

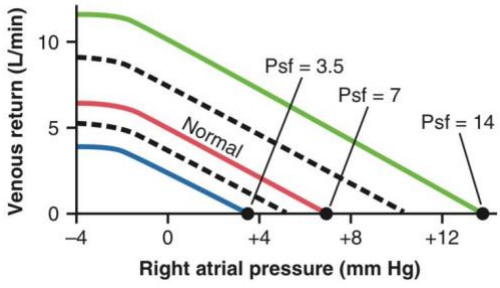
Pv : venous pressure

Rv: venous resistance

If P_{RA} = Pv their will be no return, when VR is zero means that no circulation (P_{RA}=Pv=Pa), this pressure is called mean systemic filling pressure or mean circulatory pressure , it's the pressure in CVS when their is no flow , in normal person its around 7mmHg.

The P_{sf} increases in transfusion and decreases in bleeding.





This pic from the book

كان من دعاء النبي صلى الله عليه وسلم " اللّهُمَّ نجِّ المُستضعفينَ من المؤمِنينَ" فلا تنسوهم من دعواتكم

V1