

The Cardio-

VASCULAR

System

- Anatomy
- Histology
- Pathology
- Pharmacology
- Physiology
- Microbiology

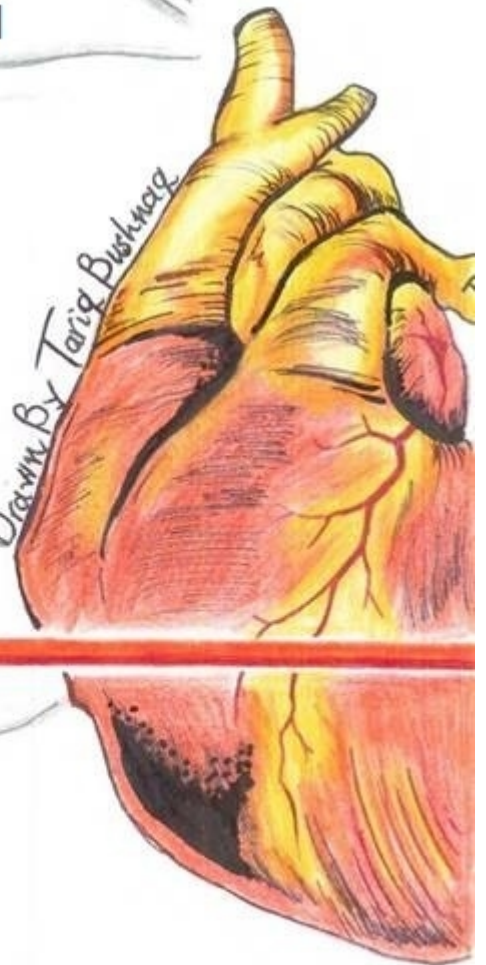
Lec #: 15

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- Handout
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Drawn by Tariq Bushnaq



Hemodynamics & Introduction to Blood pressure regulation

Yesterday we talked about flow and it's relation with resistance, length, radius and viscosity, how resistance can be parallel or in series, and how to calculate the total resistance depending on that.

Today we start with Viscosity (η), so how does the viscosity change?

1. **Hematocrit:** higher hematocrit means higher viscosity and vice versa, therefore in anemia viscosity is lower and in polycythemia viscosity is higher.

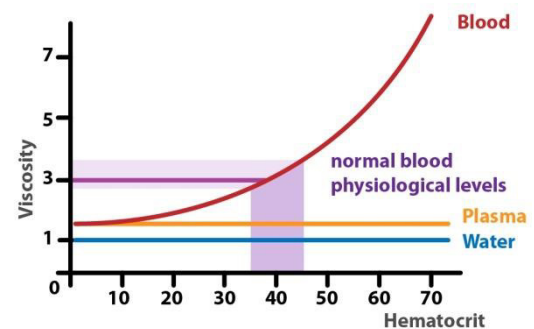


Figure 1

-Notice that an increase of hematocrit in plasma doesn't give a higher viscosity (because plasma proteins are the same), in contrast in blood an increase in hematocrit gives a curvilinear (not linear) increase in viscosity (fig. 1), and that's what happens in polycythemia where the number of RBCs increase, therefore the viscosity increases drastically and this leads to an increase in resistance of flow, which may end in an increase of the chances of thrombus formation and embolism. As well as decreased tissue oxygen due to sluggish circulation, so these are the dangers of polycythemia.

2. **Plasma protein:** mainly albumin, fibrinogen and globulin.
3. **Diameter of blood vessel** (Note that: this doesn't apply in capillaries, because although they are small, they

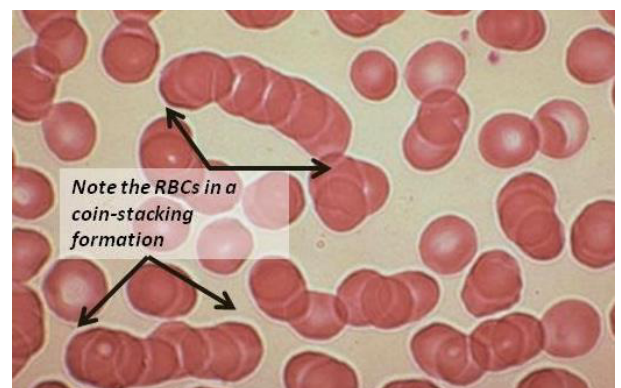


Figure 2

have the same viscosity as that of large vessels and that is due to rouleaux formation of RBCs) (fig. 2)

4. **Temperature:** an increase in temperature causes a decrease in viscosity and vice versa.
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-Since the mean arterial pressure (MAP) is almost constant, we can change the flow by changing the **resistance**, as it increases, the flow decreases and vice versa, so how do we change the resistance? We control the resistance by changing the diameter, either by vasoconstriction (increase the resistance) or vasodilation (decrease the resistance).

-**Critical closing pressure:** is the point at which there is **no** flow although there is some pressure, and this is due to very high resistance. (fig. 3) (We will elaborate on this point in the respiratory system).

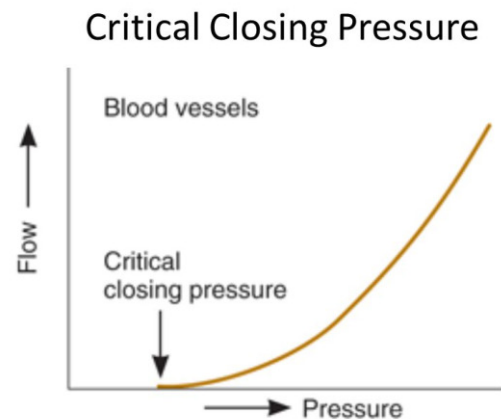


Figure 3

Laplace's Law:

states that the larger the vessel radius, the larger the wall tension required to withstand a given internal fluid pressure given by the equation: $T = P * r$

where: T= Tension, P= Distending Pressure
& r=Radius

Now we discuss 2 important terms.

1- **Distensibility (or elasticity)**: the proportional increase in volume per increase in pressure.

This is a fraction so we divide by the original volume.

And this is the ability of the vessel to distend or in other words how much the vessel is elastic.

$Distensibility(D) = \frac{\Delta V}{\frac{\Delta P}{V^o}}$. Where ΔV = change in volume, ΔP = change in pressure & V^o = original volume.

So if we have an original volume of 100ml, and to increase the pressure of this vessel by 1 ml Mercury, we have to add 10ml, what is the distensibility?

$\frac{\frac{10}{1}}{100} = 0.1$ Or 10%, remember it's unitless as this is a fraction.

-The veins are 6 times more distensible than the artery.

2- **Compliance (or capacitance)**: total change in volume per unit change in pressure.

$Compliance(C) = \frac{\Delta V}{\Delta P}$ Where ΔV = change in volume & ΔP = change in pressure.

Compliance = Distensibility * the volume (derived from the above equation)

-The veins are **24 times** more compliant than the arteries and that is because the total volume in the veins is 4 times more than the arteries and the veins are 6 times more distensible than the arteries ($6*4=24$). Which means in arteries a small increase in volume yields a significant increase in pressure unlike veins (compliant vessels) which can store large amounts of blood while maintaining a low pressure.

Pulse pressure:

-It is the difference between systolic and diastolic pressure, so it is normally $120-80=40$.

-It is responsible for the movement of the pulse through the arteries (however if you feel a pulse in a vein or a capillary it's an abnormality).

-The pulsations are generated by the movement of the walls of the arteries and not by the flow of fluid through them.

-Now since the pulsations are through the walls, the compliance alters how fast the pulsations move, imagine the vessel as a carpet if you have a thin carpet and you start creating waves with it, the waves will have higher crests and lower troughs so its velocity decreases as in A, while if the carpet is thick the wave will have smaller crests and troughs and therefore a higher velocity as in B. (Check fig. 4).

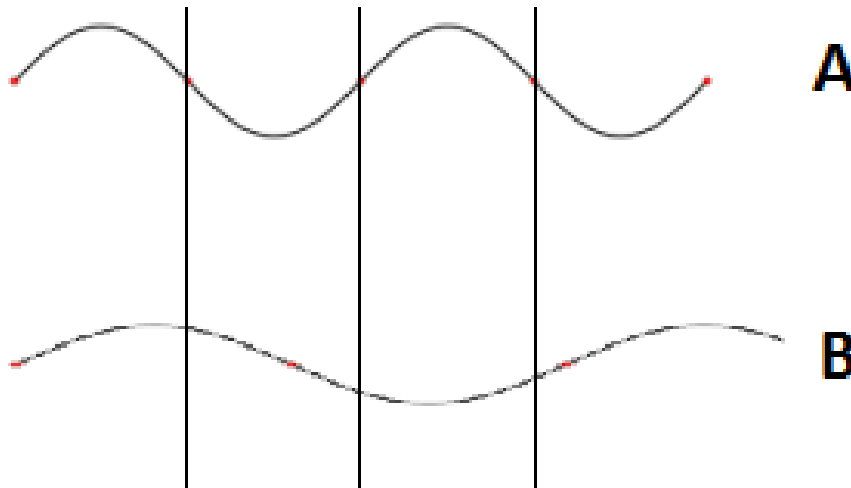


Figure 4

Now the thick carpet (B) represents the vessels (medium sized arteries or arterioles) which have a lower compliance, and the thin carpet (A) represents the vessels (the aorta) which have a higher compliance. So as the

compliance increases, the speed of propagation of the pulse decreases.

The speed of propagation in the aorta is 5m/s, while in medium sized arteries 10m/s and in some arterioles it may reach 40m/s. that's why when you feel the radial pulsation it will be felt almost at the same time as the first heart sound (due to the very high velocity).

Note: the compliance in the aorta is higher than the compliance in the subsequent arteries, however it's much lower than that of the veins (which as we said are 24 times more compliant), so don't confuse things up.

What affects the pulse pressure?

1- Systolic pressure 2- diastolic pressure 3- compliance.

Systolic is affected by stroke volume; the more blood that gets pumped through the vessel the more pressure it applies on the wall, increasing the systolic pressure, thus increasing the pulse pressure.

On the other hand **compliance** also affects pulse pressure, a blood vessel with a low compliance means a small addition of volume gives a higher pressure, so the lower the compliance the higher the systolic pressure and therefore a higher pulse pressure and vice versa.

So we can conclude that the pulse pressure depends on the ratio of stroke volume over the compliance ($\frac{SV}{C}$).

So if we increase the SV, but at the same time increase the compliance the ratio will stay the same and **the pulse pressure won't change.**

Since Mean Arterial Pressure(MAP) = $\frac{1}{3}$ systolic + $\frac{2}{3}$ diastolic, we can change the equation to diastolic + $\frac{1}{3}$

pulse pressure, as pulse pressure is systolic – diastolic, so we will get diastolic + 1/3 systolic – 1/3 diastolic, which ends up as 2/3 diastolic + 1/3 systolic.

Conclusion MAP = diastolic + 1/3 pulse.

(Check fig. 5 for maths)

$$\begin{aligned} \text{Mean Arterial Pressure (MAP)} &= \frac{2}{3} \text{ diastolic} + \frac{1}{3} \text{ systolic} \\ &\text{also diastolic} + \frac{1}{3} \text{ pulse} \\ &= \text{diastolic} + \frac{1}{3} (\text{systolic} - \text{diastolic}) \\ &= \text{diastolic} + \frac{1}{3} \text{ systolic} - \frac{1}{3} \text{ diastolic} \\ \text{MAP} &= \frac{2}{3} \text{ diastolic} + \frac{1}{3} \text{ systolic} \therefore \end{aligned}$$

Figure 5

Now we study 3 pathological cases which affect the pulse pressure:

1-Aortic regurgitation:

Manifests when some blood flows back to the ventricle from the aorta due to incompetency of the semilunar aortic valve, so the end diastolic volume (EDV) increases, so the stroke volume increases and more blood will be pumped causing the systolic pressure to increase, also since blood flows back to the heart,

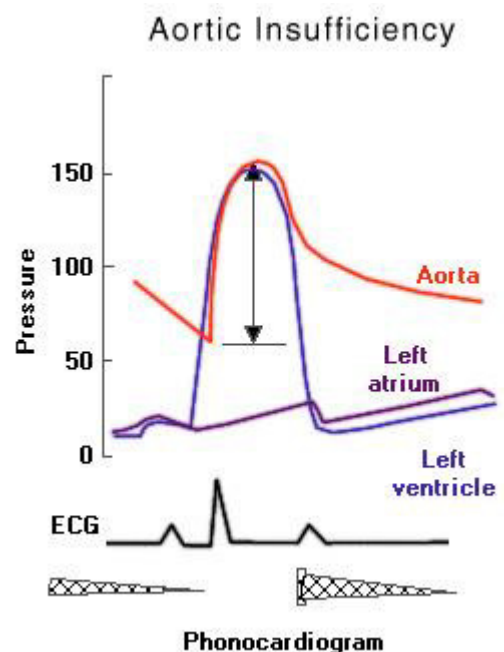


Figure 6

the diastolic pressure decreases, therefore the pulse pressure increases drastically, so you can diagnose aortic regurgitation by measuring blood pressure. (In some severe cases diastolic pressure can reach 0).

-studying the aortic pressure curve of a patient with aortic regurgitation (fig. 6), we find that the systolic pressure is increased, also the diastolic pressure decreased, but one key finding will be the **absence** of the dicrotic notch (insisura).

2- Patent ductus arteriosus:

Blood will be shifted from going from the aorta to going to the pulmonary artery, and will end up in the left ventricle increasing the End Diastolic Volume, therefore also increasing the Stroke Volume and the systolic pressure, also the diastolic pressure will decrease but not to the same extent as that of a regurgitating aortic valve.

3- Atherosclerosis:

The walls of the arteries will thicken, decreasing the compliance (same volume, leads to higher pressure) so the systolic pressure increases. So you should expect to see high blood pressure in some old patients due to this.

Regulating Blood Pressure:

some general points:

- there are 2 kinds of regulators: 1- short term regulators and 2- long term regulators.

When you stand up from a supine position, you expect your blood pressure to decrease, but this doesn't happen due to the action of short term regulators, in elderly

people these regulators have diminished function, so when they stand up they feel dizzy because not enough blood reaches their brain. Short term regulators usually do their function by signals from nerves.

While long term regulators' function is to bring back pressure to its normal value if it has been deviated from normal for a long time. Long term regulators usually do their function by signals from hormones.

We measure blood pressure using a device called **sphygmomanometer** (fig. 7) & a **stethoscope** (fig. 8).



Figure 7



Figure 8

A sphygmomanometer used to contain mercury, but they later replaced it with other materials because mercury is toxic and hard to handle safely.

How to measure the blood pressure?

1-Raise the pressure above the systolic pressure, so that no flow passes through the arteries, therefore you will not hear any sound from the stethoscope placed on the arterial system.

2-start decreasing the pressure, till you hear the first sound, this sound demarcates the systolic pressure.

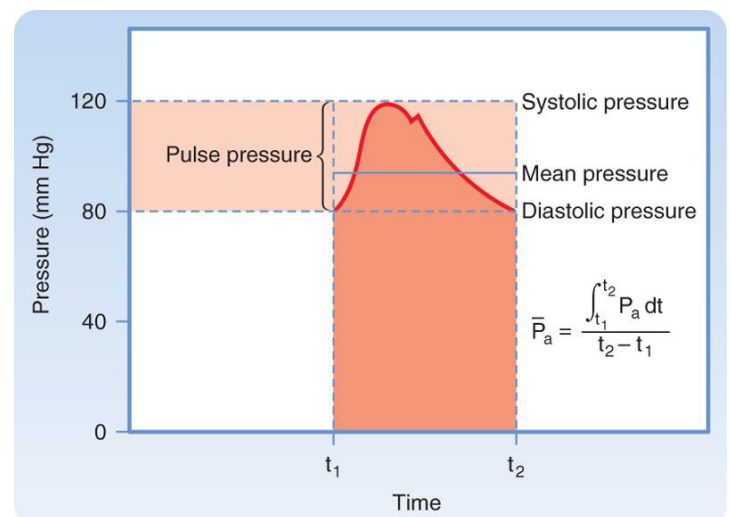
3-then you continue decreasing till you can no longer hear any sound and this demarcates the diastolic pressure.

-Now what do the presence and absence of a sound resemble? The sound you hear through the stethoscope is produced due to turbulence inside the blood vessel, now when the pressure is above the systolic pressure, there will be no flow inside the vessel and hence no sound, when you reach the systolic pressure blood will start passing but with turbulence, that's why you hear a sound, then after the pressure decreases below the diastolic pressure blood will flow inside the vessel in a laminar fashion, producing no sound.

-Now this whole procedure is called **auscultation** method, because you're using a stethoscope to hear the sounds, sometimes you don't have a stethoscope around, here you can use your fingers to detect the radial pulse, when the pressure is above the systolic you won't feel a pulse, once you feel a pulse that's your systolic pressure, however using this method you can only measure the systolic, the diastolic can't be measured this way.

Mean arterial pressure:

Now to calculate the mean arterial pressure, we have to find the area under the curve and divide it by the time, we can calculate the area using integration (fig. 9), and then dividing it by the time (t_2-t_1), but it isn't



Koepfen & Stanton: Berne and Levy Physiology, 6th Edition.
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Figure 9

feasible to do this every time, if we use a machine it will automatically use integration to calculate the MAP accurately, but without a machine we refer to the approximation of $MAP = 2/3 \text{ diastolic P} + 1/3 \text{ systolic P}$ or as mentioned earlier $MAP = \text{diastolic P} + 1/3 \text{ pulse P}$.

To change the MAP we either change the CO or the TPR or both.

To change the CO we either change the SV or the HR or both.

Questions made by me (not past papers) so they may contain mistakes:

1- Which of the following regarding viscosity is FALSE:

- A-Blood viscosity decreases in case of anemia.
- B-In hypoalbuminemia plasma viscosity decreases.
- C-In a patient with Hypothermia blood viscosity increases.
- D-Plasma viscosity increases in polycythemia.
- E- All the above are true.

2-Which of the following regarding compliance and distensibility is TRUE:

- A-Arteries are 24 times more compliant than veins.
- B-Veins are 6 times more compliant than arteries.
- C-In a vein with a distensibility of 0.2 and original volume 100, you will need to add 2ml to raise the pressure by 20mmHg.
- D-Capillaries have a lower compliance than veins.
- E-More than one of the above is true.

3-Which of the following regarding aortic pressure curve is FALSE:

- A-The decrease in diastolic pressure in aortic regurgitation is more significant than that of a patent ductus arteriosus.
- B-A dicrotic notch is present in a patent ductus arteriosus.
- C-The pressure difference around the aortic semilunar valve increases in atherosclerosis.
- D-Using your fingers to feel the radial pressure in addition to a sphygmomanometer, the measured quantity will be lower in aortic regurgitation.
- E-In some severe cases of aortic regurgitation the diastolic pressure can reach 0.

4-Regarding blood pressure regulation which of the following is FALSE:

- A-Laminar flow produces no sound in a stethoscope.
- B-When the pressure in the sphygmomanometer is above the systolic pressure, no sound is produced because of laminar flow.
- C-As long as the sphygmomanometer pressure is between systolic and diastolic pressure, a sound is heard due to turbulent flow caused by vasoconstriction of the artery.
- D-Short term regulators return pressure back to normal when standing up from supine position.
- E-The stethoscope has a Bell & diaphragm.

1	2	3	4
D	D	D	B

This sheet is dedicated to: **الأوزة الرمادية, الضفدع الثوري و الجزيرة الطويلة الكهربائية.**
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