4

Physiology sheet

Doctor 2022 -أثر- I medicine I MU

DONE BY:

Doha Al Thunibat

Bayan Qudah

CORRECTED BY:

Ibrahim Amjad

DOCTOR

Dr. Arwa Rawashdeh

Resistance and Capacitance

To figure out any relationship between X and Y variables in the heart- related lectures you have ONLY either of two ways, if not both: memorizing the laws and/ or understanding the relationships to make sense of them. Make sure to cover both; you never know.

Isometric and Isotonic Contractions:

- **A quick revision: Muscles contractions are either 1) isometric contractions or 2) isotonic contractions with its two subdivisions: a) eccentric and b) concentric.**
- **Isotonic contraction → Shortening of the muscle without stretching, same tension, doing work by moving and it is 25-30% or 20-25% of the overall contraction. Concentric vs. eccentric? In a concentric contraction, the muscle shortens when its tension is greater than the force opposing it, such as your biceps does when perform an arm curl. In an eccentric contraction, the force is greater than the muscle tension, causing the muscle to elongate; this happens when going downstairs or sitting down in a chair, as the effects of gravity add to the opposing force.**
- **Isometric contraction→ has stretching but no working, increase tension but no change in length.**

- **In Frank Starting (Pre-load) has Isometric contraction.**
- **Isotonic 25% contraction, remaining energy takes place in balancing human body, heat production and other things in homeostasis process (very important). That being said, not all effort (energy) go to contraction.**
- **Can isometric contraction become isotonic contraction?**
- **Yes, after Pre- loading we have pumping out. So, we need sliding actions between actin & myosin microfilaments → we need shortening (which happens in isotonic contractions only). Isometric= 0 and Isotonic= 20-25% → Only the game in town**
- **So, not just moving muscles, body metabolic processes balancing also needs energy. If balance is not done → some diseases might happen.**

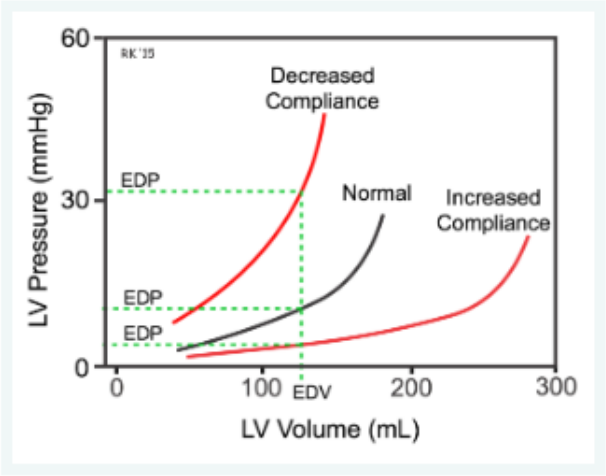
- **Stroke volume (SV) is around 60 ml/ beat. However, we do not measure SV but we measure ejection fraction (EF) where:**

$$\text{EF}= \frac{Stroke\ Volume\ (SV)}{End{-}Diastolic\ Volume\ (EDV)}\ x\ 100$$

- **So, if EDV was 100 ml/ beat; EF=** $\frac{60}{100}\ x\ 100 = 60\%$**. So, working or mechanical efficiency of this heart= 60% (W (work)= F (force). D (distance) or Mechanical efficiency= W/ energyX100). Note that: EF= 60-50% is the normal range, 50>- 40% is below normal and <40% may indicate heart failure. So, the 60% calculated above is normal → his heart is pumping enough blood. (anything less than that→disease or at least abnormality)**

Ventricular Compliance:

- **Note that:** Conductance= capacitance= elasticity= compliance. **In the figure:**
- Let the independent variable (on x- axis) be left (L) ventricular (LV) volume in mL (or EDV) and the dependent variable (on y- axis) be LV pressure in mmHg (or EDP).
- **As the ventricle fills with blood, the pressure and volume that result from filling are determined by the compliance of the ventricle. Normally, compliance curves are plotted as the change in volume (ΔV) over the change in pressure (ΔP). Therefore, the slope of the relationship is the reciprocal of the compliance, which is sometimes referred to as ventricular "stiffness."**



- **As the ventricle fills with blood and its volume increases, the pressure within the ventricular chamber passively increases (see the Normal filling curve in the figure). The relationship is not linear, particularly at higher volumes, because the compliance of the ventricular wall decreases ("stiffness" increases) the more the ventricular wall is stretched. This occurs in most biological tissues.** At maximum pressure → no any kind of stretching (contractions) → actin & myosin separated.

- In the case of heart- related problems (for example: dilated cardiomyopathy → ↓ amount of oxygen (O2) used) which is characterized by thinning of ventricles' muscles walls and it is a very common disease in ages 20- 60. Causes of it include: genetic, endocarditis (inflammation of heart's endocardium) and general infections. EDV is the amount of blood pumped into ventricles just before the heart contracts → widening of ventricular walls → stretching. In case of dilated cardiomyopathy, no matter the amount of stretching done (due to increase of the volume), the increase in pressure will be minimal (i.e. pressure and volume are not linear (approximately) like before → curvy), This is called **increased compliance (see its curve)**→ (changes in the volume are much greater than changes in the pressure, parallel?). **In a disease state such as dilated cardiomyopathy, the ventricle becomes very dilated without appreciable thickening of the wall. This dilated ventricle will have increased compliance as shown in the figure; therefore, although the EDV may be very high, the EDP may not be greatly elevated.**

- Another case is **decreased compliance (see its curve)**→ any increase in volume will cause dramatic (huge) elevations in pressure. In other words, pressure will be higher than that resulted from EDV normally → there is a lot of pressure inside the ventricle → mostly seen in cases of thicker than usual ventricular walls (hypertrophy of ventricles) → ventricles' walls are much enlarged (accordingly, more O2 is needed than usual). Such causes of enlargements include: stenosis of valve (narrowing), heart murmur or hypertension (HTN). However, it could be

acute for normal causes such in exercising where lots of contractions are needed. Note that both ↑ and ↓ compliance affect SV. **in ventricular hypertrophy the ventricular compliance is decreased (i.e., the ventricle is "stiffer") because the thickness of the ventricular wall increases; therefore, ventricular end-diastolic pressure (EDP) is higher at any given end-diastolic volume (EDV).**

- **To sum up:**
  - **Any increase in volume should increase the pressure in a correct manner; normal compliance (not more, not less). If not → something is wrong.**
  - **Normally: ↑ in LV volume (EDV) → ↑ in LV pressure (EDP)**
  - **Increased Compliance: ↑↑↑ in LV volume (EDV) → ↑ in LV pressure (EDP) (as in dilated cardiomyopathy)**
  - **Decreased Compliance: ↑ in LV volume (EDV) → ↑↑↑ in LV pressure (EDP) (as in ventricular hypertrophy)**

Blood Pressure:

- **We can simply define blood pressure as the force exerted by blood against the walls on a specific area and it is related to cardiac output. ↑ force (velocity) → ↑ pressure. Mathimatically:**

$$\text{Pressure (P)} = \frac{Amount\ of\ Force\ (F)}{Surface\ Area\ (SA)}$$

Or

$$BP = CO\ X\ TPR$$

- **Where: BP: Blood pressure, CO cardiac output and TPR total peripheral resistance.**
- **First, we want to decide what CO and TPR is, then we get to the right meaning of BP**
- **Cardiac output (Flow)= Heart rate X Stroke volume**
  - $CO\ (F)$= HR X SV
  - ml/min= Beat/min X ml/ Beat
- **SV (the volume of blood pumped from LV / beat) has the following relationships:**
  1. **Directly with → pre- load (EDV, the more blood in the ventricle during diastole → the more blood pumped during systole) and consequently → with CO.**
  2. **Indirectly (inversely) with → contractility (indicated by End- systolic volume (ESV); or HR) → think of it as: if the heart beats faster (↑ HR→ no time for filling LV → ↓ volume in LV →↓ amount to be pumped by LV→ ↓ SV). Same as the volume of blood left in LV after each systole; ESV. Remember, heart rate value of 60 – 90 beat/ min (the normal range) means 60- 90 cardiac cycles are done during one minute.**

- **Assignment Question: Most of the athletes have blood pressures as low as 40 beats/ min, is it normal? Explain your answer.**
- **MY ANSWER: structural modifications of heart muscle (myocardium), particularly hypertrophy, happens in athletes with routine exercising. According to that, they**

require lower efforts to pump the same amount of blood in normal people → they tend to have lower than usual HR. Moreover, exercising consistently for 3- 4 weeks is enough to make your heart rate drop by 5 to 10 points than its usual, in a healthy none- pathological way.

Resistance and Blood flow:

- **How to relate TPR to blood pressure?**
- $F = \Delta P/R$ **Ohm's Law** F: force, ΔP: pressure gradient and R: resistance
- $CO = \Delta P/TPR$ CO: cardiac output and TPR: total peripheral resistance

- $R = 8nl/\pi r^4$ **Poiseuille's law** n: blood viscosity, l: BVs length, r: BVs radius
- $n \alpha R$
- $n =$ viscosity
- ↑blood volume inside chamber→ ↑blood pressure → ↑stretching (tension). That being said, there are direct relationships between BP and each of: CO, SV (as it has a direct relationship with CO which has itself a direct relationship with BP) and tension (stretching). The relationship between tension, pressure and radius:

$$Tension = Pressure \ x \ Radius$$

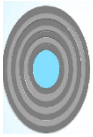
- Total Peripheral Resistance (TPR) or Systemic Vascular Resistance (SVR) or shortly resistance (R); is simply the amount of force exerted on circulating blood by the vasculature of the body, in other words, force exerted by BVs on our blood. There is a relationship between CO, BP and TPR:

$$BP = CO \ x \ TPR$$

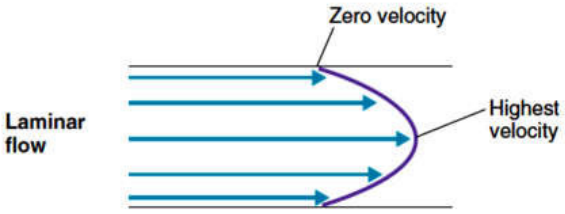
- There is 1) an indirect (inverse) relationship between TPR and CO (blood flow) and 2) a direct relationship between BP and resistance → HTN= ↑↑↑ resistance. So, when we talk about any kind of heart disease referring to resistance (almost 90% of heart diseases are caused from increase of resistance). This increase in resistance may cause heart muscles to tear.
- We use Δ P rather than P in Ohm's Law and CO law because we mean by pressure here a difference; from the first point the blood enter the vena cava until it was pumped; so, it is always a difference between the highest and the lowest.

- **How to relate TPR to blood pressure?**
- $F = \Delta P/R$
- $CO = \Delta P/TPR$
- $R = 8nl/\pi r4$ Poiseuille's law
- $n \alpha R$
- $n =$ viscosity
- Viscosity (resistance of a fluid to flow) → it ↑ friction between blood flowing and wall of blood vessel. With normal viscosity blood flow won't have any problems →

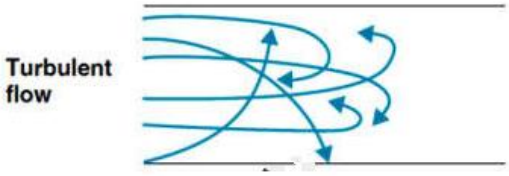
normal blood flow → laminar flow (in parallel lines). **Laminar flow: normal blood flow in the blood vessels (physiological):**



- As you go toward the edges the velocity the blood is going to be slower and the velocity in the middle is highest
- So, imagine you are looking to blood vessels as a circle, and you are looking at the flow from the back you are going to notice that is flow is very concentric and this type of flow is silent



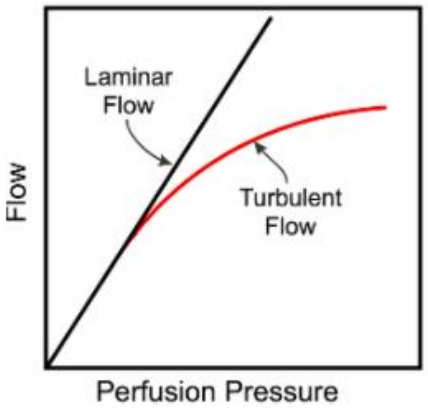
- Polycythemia (high Hct)α n ; a lot of friction between the layers, because whenever blood is flowing it flows in layers when there is a lot of fri ction rubbing up against between those layers because increase in viscosity and slow the flow down. If the viscosity was ↑ as seen in ↑RBCs total count (polycythemia vera) → ↑ viscosity (as the blood is formed of liquid plasma and solid cells, ↑ the cells will definitely ↑ viscosity) → ↑ resistance → ↓ elasticity → blood flow is not parallel anymore → turbulent flow. **Turbulent flow: pathological and physiological one**:
  - Inside our heart you have a valves mitral valve and aortic valve whenever blood is being pumped upward right it can hit mitral valve as it hits mitral valve it can develop turbulent flow
  - Imagine a blood vessels and plaques inside ; as the normal flow gets to the occlusion it start developing a turbulence and that gives a lot of heat and changes the action of perfusion pressure and produce what called brutes and can be heard at carotid artery so if you take a stethoscope and put it over carotid artery you can hear it as actual sounds that caused by turbulent flow. It also can produce murmurs

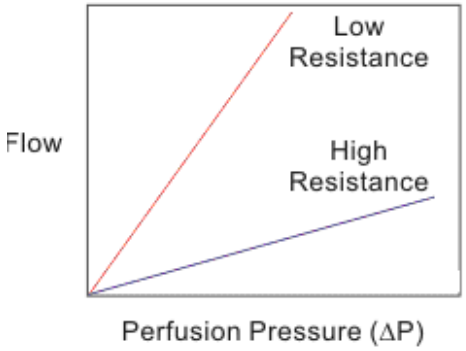


- Anemia $\frac{1}{\alpha}$ n: In anemic patients usually there is a ↓ in blood viscosity → ↓ in resistance.

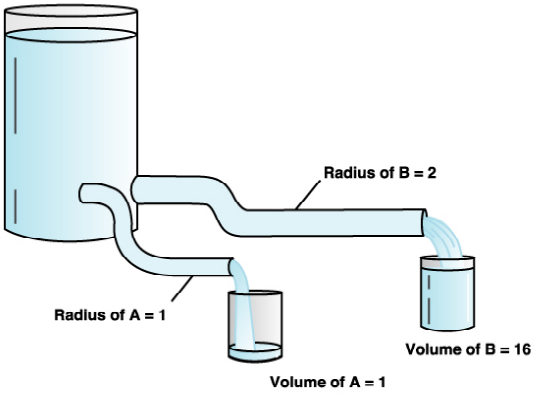
- *(the following part will be explained next lecture). If you look at the graph here*; as you increase the pressure the flow is increasing in laminar or turbulent flow, but you get to the point where the flow veers off and the flow start decreasing as the perfusion pressure start increasing

- **If there is a turbulent flow it decreases the actual flow the volume of blood that circulating through an area of blood vessel per a minute and increase the perfusion pressure and the resistance is going to be very high**





- **In the following figure: Effect of radius on resistance and blood flow**
- **Length and weight (of people) both have direct relationships with resistance. While radius of a BV has an indirect (inverse) relationship with resistance. The main factor affecting resistance is radius as it's to the power of 4. For example, ↑ the radius from 1 to 2, ↓ the resistance from 1 to 1/16 and consequently ↑ the flow from 1 to 16.**

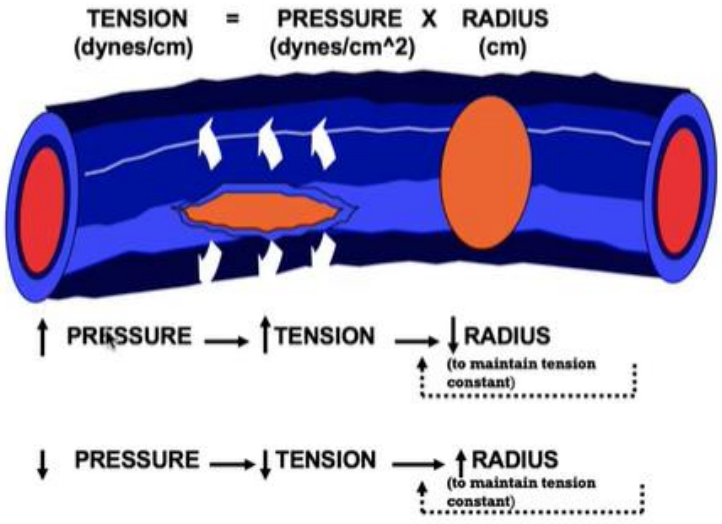


Resistance ~ $\frac{1}{\text{radius}^4}$	
Tube A	Tube B
R ~ $\frac{1}{1^4}$	R ~ $\frac{1}{2^4}$
R ~ 1	R ~ $\frac{1}{16}$

Flow ~ $\frac{1}{\text{resistance}}$	
Tube A	Tube B
Flow ~ $\frac{1}{1}$	Flow ~ $\frac{1}{\frac{1}{16}}$
Flow ~ 1	Flow ~ 16

- **Note the following relationships:**
  1. **Resistance and radius: indirect.**
  2. **Resistance and blood flow: indirect.**
  3. **Blood flow and radius: direct.**

- **In the following figure: If blood flow ↑ for any reason dramtically → pressure ↑ in our bodies beyond its stretching abilities → our BVs will explode! It won't happen as our bodies ↓ TPR by causing vasodilation → ↑ r to maintain constanst blood pressue. $L \alpha R$ and increase in Weight and height increases in L. $r = 1/\alpha R$ the most important factor that affecting the R because it is raised to power 4.**
- **Vasodilation increases in r and Vasoconstriction decreases in r**
- **T= (PXr)/H**



- Laplace Law (will be explained better in the next lecture): ↑ blood inside BVs, ↑ pressure and ↑ tension → affects resistance and radius. On dilation → ↑ radius → ↓ resistance.

- What type of BVs are responsible for controlling resistance? Arteries (our bodies change radius of arteries).

- To sum up, tension (stretching) has direct relationships with pressure and radius but indirect (inverse) relationships with thickness of BVs walls.

"Conductance" of Blood in a Vessel and Its Relation to Resistance:

- **Conductance (C_L) is a measure of the blood flow through a vessel for a given pressure difference.**

$$C = \frac{\Delta V}{\Delta P}$$

- **This is generally expressed in terms of milliliters per second per millimeter of mercury pressure, but it can also be expressed in terms of liters per second per millimeter of mercury or in any other units of blood flow and pressure.**
- **It is evident that conductance is the exact reciprocal of resistance in accord with the following equation:**
- **Conductance= 1/Resistance**
- **The vascular compliance is proportional to the vascular distensibility and vascular volume of any given segment of the circulation. The compliance of a systemic vein is 24 times that of its corresponding artery because it is about 8 times as distensible, and it has a volume about 3 times as great.**

Cardiac Output

- **HR:**
- **- PSNS** (parasympathetic nervous system → rest or digest → ↓ HR → ↑ SV).
- **+ SNS** (sympathetic nervous system → fight or flight → ↑ HR → ↓ SV).
- **+ Hormones (EPI, NE)** (mostly linked to sympathetic nervous system → same effect).
- **IONS: Ca++, Na+ , K+ dependents on their level increase or decrease** (most effect are due to: Na, Ca then K). They tend to have direct relationships → ↑ HR → ↓ SV.

- **SV:**
- **+ Preload ; Increase the blood volume returns increase diastolic volume**
- **+ Contractility ; SNS (EPI,NE+), Hormones (glucagon,T3 and T4), IONS like Ca++** → ↑ those factors → ↑ contractility → ↑ SV → ↑ CO.
- **- Afterload; Hypertension, Atherosclerotic plaques** (Cholesterol occlusion of BVs), **TPR** → ↑ after- load (End systolic volume) → ↓ SV → ↓ CO.
- Compliance (or any of its synonyms) has indirect (inverse) relationship with resistance.
- Compliance of veins is more than that of arteries by 24 (to 30) times.

- **Ejection Fraction = 60/100=60%**

لم ندخل الطب لنفشل. لم ندخله إلا لنكون سبب حياة. لنكون سبب ابتسامة. لنعيد ترتيب ميم الألم ولام الأمل. لم نأت لنجني درجات أو نحصد تصفيقات. جئنا نبحث عن العلم. عن المعرفة. عن تلك السطور التي قد ننقذ حياة أحدهم بها يوما ما بإذن الله 🍎🦋