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Physiology sheet

Doctor 2022 -أثر- I medicine I MU

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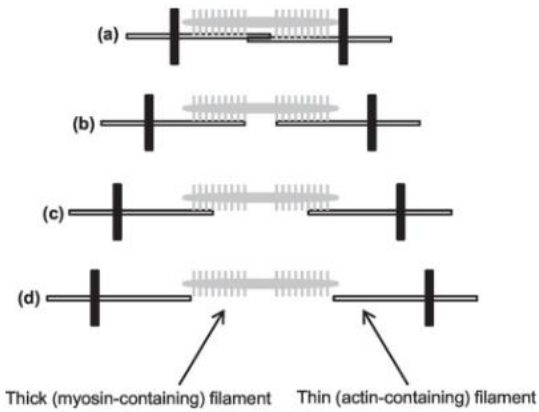
Frank- Starling Mechanism and Mechanical Efficiency

Frank Starling Mechanism:

- **How can we measure working efficiency of the heart? By comparing blood received (volume before beating) to blood pumped (volume after beating).**
- **Pumping blood from atrium to ventricle is called (diastole) and the corresponding volume is called (diastolic volume) → relaxation of ventricular muscles to take a huge amount of blood → also called preload volume, pre-beat volume or pre-pumping volume → it's linked to stretching actions.**
- **The process of pumping blood out of the left (L) ventricle to all body parts is called: beating, after- load or systole→ it is linked to pumping actions and contractions.**
- **At (@) first, to "welcome" the huge amounts of blood to the heart, contractions must happen to push this blood → lots of cross- bridges between actin and myosin must happen (remember, actin and myosin are microfilaments responsible for excitation- contraction coupling mechanism, where sliding action happens).**
- **Then, stretching (increasing the length) must occur to the myofibers. The wider the cross- bridging → the more the distance→ more blood will flow. Until we reach a stage of maximum cross- bridging → no more contractions→ maximum stretching was attained. Note that myofibrils in general have high elasticity (can stretch and get back to its original shape afterwards [called recoil]).**
- **The following figure:**
  - **a) The beginning of the process of pumping blood is called early diastole. the actin filaments overlap with each other due to the lack of muscle lengthening, but there is contact between the myosin heads and the actin filaments → maximum amount of cross- bridges → maximum contractions.**



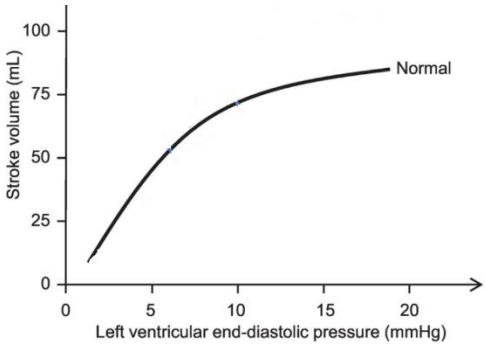
Tension developed at different cardiac sarcomere lengths.

  - **b) It is called the middle diastolic. the volume of blood increases, which leads to the pumping of a lot and a lot of blood into the ventricle and the atrium. There is a gradual increase in the volume of blood inside the heart and there is an increase in the length of the muscle (all are stretched and actin filaments moving away) but there is still an overlap between the actin filaments and the myosin head → Stretching happened→ increase in length. However, there is still contractions even if it is lower than before.**
  - **c) Continue to pump blood into the heart. actin filaments and the myosin heads still binding, and the process of increasing the length (stretching) continues,**

which leads to an increase in the distance between actin filaments → contractions are still there yet lower.

d) The maximum volume of blood inside the heart so that the heart does not receive any type of blood, too much stretching, so there is no connection between actin and myosin due to increased distance. (Tension: Force inside the cell). → no more contractions happen here as there is no more cross- bridging.

- **The next figure shows** the relationship of pressure to volume; the greater the volume:
  - → The greater the contraction force
  - → The greater the tensile force (stretching force)
  - → The greater the cardiac output



- Cardiac output (CO) is an increase in blood volume, either on the R or L sides.
- Stroke volume (SV) means amount of blood outside a heart chamber (venous blood return); remember: the amount of blood on the R side must be equal to the amount of blood on the L side
- Frank- Starling Mechanism → the more stretching in ventricles (ventricular muscles) →the more diastolic volume in ventricles → the more systolic volume the → more stroke volume.

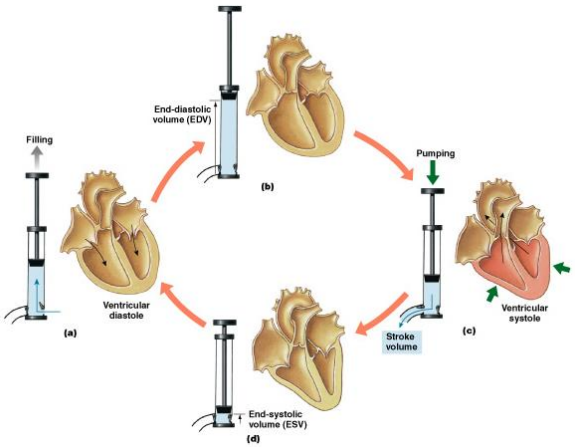
Frank- Starling Principle:

- **The Frank–Starling law of the heart (also known as Starling's law and the Frank–Starling mechanism BUT not Starling forces; those are different) represents the relationship between stroke volume and end diastolic pressure and consequently volume.**
- **End diastolic volume (EDV): The amount of blood that remains in the ventricle just before ventricular early systole while as End systolic volume (ESV): The amount of blood that remains in the ventricle at the end of ventricular systole and Stroke Volume (SV): volume pumped out of the left ventricle of the heart during each systolic cardiac contraction. That being said, Frank- Starling Law is:**

$$SV = EDV - ESV$$

- Note that: EDV is directly proportional to SV while as ESV is indirectly (inversely) proportional to SV.
- **This principle illustrates the relationship between cardiac output and left ventricular end diastolic volume. What is the relationship between SV and cardiac output (CO)? a direct relationship.**

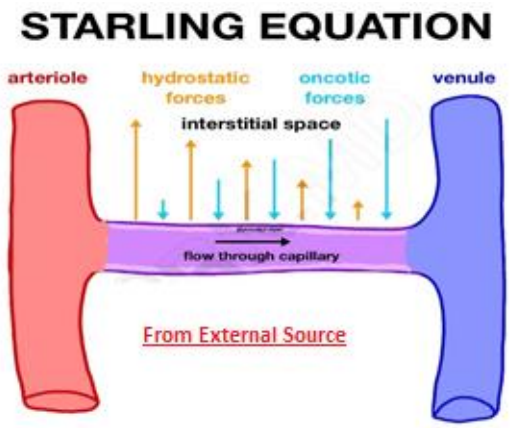
- **The law states that the stroke volume of the heart increases in response to an increase in the volume of blood in the ventricles, before contraction (the end diastolic volume), when all other factors remain constant.**
- **As a larger volume of blood flows into the ventricle, the blood stretches the cardiac muscle fibers, leading to an increase in the force of contraction.**
- **The Frank-Starling mechanism allows the cardiac output to be synchronized with the venous return, arterial blood supply.**
- **The physiological importance of the mechanism lies mainly in maintaining left and right ventricular output equality.**
- **If this mechanism did not exist and the right and left cardiac outputs were not equivalent, blood would accumulate in the pulmonary circulation (were the right ventricle producing more output than the left) or the systemic circulation (were the left ventricle producing more output than the right).**
- **If during the pumping process to pulmonary artery (from R side of the heart) or to Aorta (from L side of the heart) we faced a problem in the valve → the pumping process will be harder → not all the blood will leave ventricles →↑ volume in ventricles → ↑ pressure in ventricles → ↑ ESV → ↓ SV. If the problem is in the R side it's called <u>right heart failure</u> which is due to L ventricular failure → not able to contract or pump all the blood.**



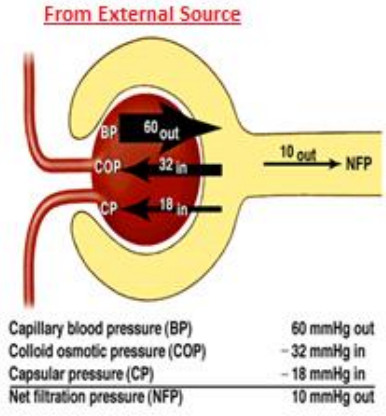
Starling Forces:

- **We studied in the <u>renal system</u> that: <u>Capillary blood pressure (hydrostatic pressure) (BP):</u> a pressure exerted by fluid on a wall againt it. <u>Colloid osmotic pressure (COP):</u> an attracting pressure exerted by solutes (proteins mostly) towards it. <u>Capsular Pressure (CP)</u> is the hydrostatic pressure resulted from the fluids inside Bowman's <u>Capsule</u>. Since Bowman's Capsule doesn't have any proteins NORMALLY, its COP is close to zero.**

- **Look @ the following figure,** the normal blood flow -in any organ-: arteries (O2 rich) → arterioles → **arteriolar end of the capillaries O2** → capillaries → **venular end of the capillaries CO2**→ venules → veins (CO2 rich).



- As blood flows, it will be filtered to the interstitium → ↓ in volume → ↓ in BP. Consequently, oncotic forces (COP in this case) will dominate more and more.
- COP is <u>constant</u> along all the regions (proteins amount won't be affected), that being said, the <u>Net Balance of Forces</u> will determine the net movement: @Arteriolar End: BP> COP, BP dominates while @ Venular end: COP> BP, COP dominates. That being said; **Hydrostatic capillary and oncotic interstitial: favor filtration** while **Oncotic capillary and hydrostatic interstitial: oppose filtration**
- Now back to our specifc case of the kideny (**Check the glomerulus figure**), we add the forces in the same direction (60 + 0) together and then we subtract those of opposite directions (32 +18) to yeild <u>Net Filtration Pressure NFP</u> of 10 mmHg out (favoring BP direction → filtration occurs!); **Kidney's net filtration (10 mmHg) out of capillaries and favoring Bowman"s capsule** (in its direction).



- Edema (morbid accumulation of fluids in interstitium → ↑ interstitial fluid volume). It can be generalized (all the body's limbs are bigger than usual, swollen and warm) or localized (one limb only). How does our hearts prevent edema from happening? hydrostatic pressure of blood from heart is very high, when moving along ciruclation it will decrease gradually with the increase of oncotic pressure (as we go from arteries to veins) → fluid pooling inside veins → prevents accumulation of fluids in interstitium. When does edema happen? When blood vessels (BVs) leaks fluids abnormally for any cause.
- How can we assure having a healthy heart? Circulation with highest blood in artery and allowing O2 passage to cells and lowest blood in veins while allowing CO2 to be taken. So, perfusion will be done.
- <u>Hypoproteinemia</u> (↓ in blood plasma proteins [<u>albumin</u> is the main one])→ oncotic pressure ↓ (depending on osmolarity) → hydrostatic pressure ↑ → ↓ pooling inside veins → ↑ blood pushing out of veins → ↑ accumulation of fluids inside interstitium → ↑ edema (generalized, localized or both). Hypoproteinemia

is usually linked with: 1)hyperproteinuria (↑ amount of proteins secerted in urine), which is characterized by foamy urine and 2) ↑ risk of thrombus formation (blood clots). The normal level of protein in urine is 300 mg/ day, an increase >300 mg/day is called nephrotic syndrome. It can increase due to multiple causes such as: 1) Nephrotic Syndrome mostly due to problems in glomeruli: proteins to be found in urine like albumin, lipoprotein and anti- thrombosis factor 3 (III), those pateints are usually having gout (↑ in blood uric acid levels) and 2) ↑ in blood cholesterol levels. 2) malnutrition as in starvation, 3) Chronic liver disease can't produce proteins as in liver cirrhosis (a type of fibrosis), 4) Severe burns (2nd or 3rd degrees) and 5) protein malabsorption.

- For the proteinuria tests to be conclusive (diagnostic), urinalysis (urine testing) must be done many types and always give abnormal results.

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

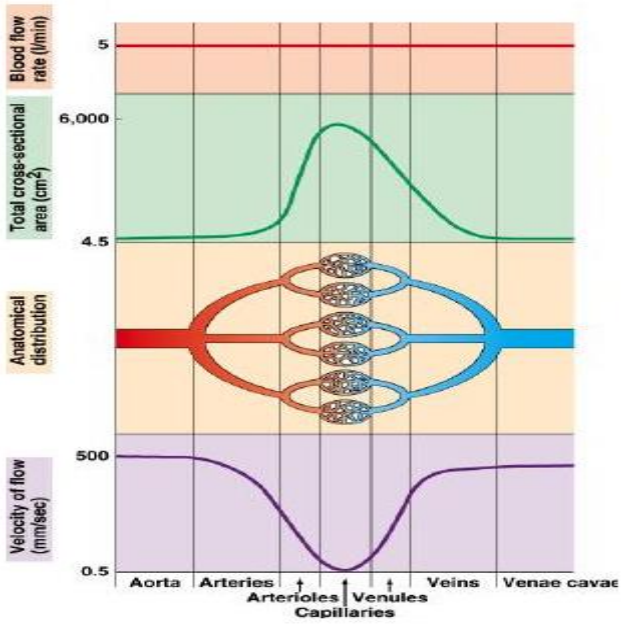
Cardiac Output (the part in page# 5 is duplicated in the next lecture; it will be explained better there)

- HR:
- PSNS –
- SNS +
- Hormones (EPI, NE) +
- IONS: Ca++, Na+ , K+ dependents on their level increase or decrease
- SV:
- + Preload ; Increase the blood volume returns increase diastolic volume
- + Contractility ; SNS (EPI,NE+), Hormones (glucagon,T3 and T4), IONS like Ca++
- - Afterload; Hypertension, Atherosclerotic plaques , TPR

- Another formula relates to CO: 1 ml= 1 Cm3 and Flow = Cm3/min
- Another formula relates to flow: Velocity (Cm2/min?)= Flow (cm3/min)
- Cross sectional area (Cm2)
- V (or P)= F/A
- How to relate this to cardiac output?
    - Increase Flow (CO)→ Increase V
    - Cross sectional area; measured in units of Pi r square because the blood vessels are cylinder in shape
    - A (or SA) (πr^2); Increase A→ Decrease V
    - Imagine a hose and water coming out of the hose at a nice pace then I put my thumb on the edge of the hose and I make A smaller the flow of the water is going to shooting out that means the velocity increases
- Cross- sectional area ≠ radius.

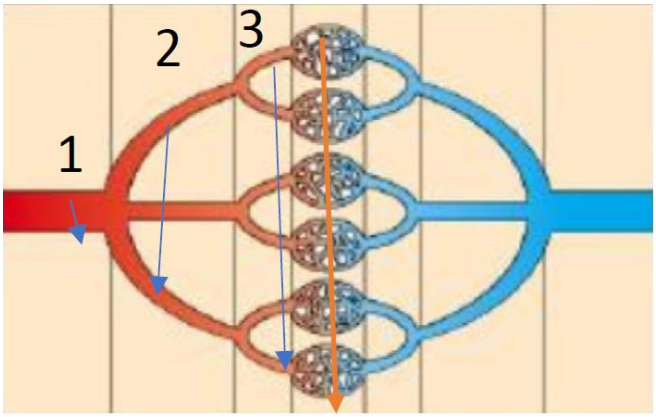
Velocity and Cross- Sectional Area:

- The cross-sectional area for the aorta is going to be very very small as you start to move toward arterioles to capillaries it is going to start rising
- As you get towards the venules it starts decreasing again and comes back down
- You have noticed so far that the aorta and arteries don't change much they change a just little bit
- But once you hit the arterioles that's when the actual specifically the cross-sectional area increases
- But we have said that the aorta has a very big diameter??
- We are going to compare between each one of these vessels
- We are going to take these numbers and correlate what we are going to talk in the next slide



- This big one here is aorta (1) then the aorta splits it gives off arteries (2) then arterial branches (arterioles) (3) and then capillary branches ten to hundred per capillary bed (4) and after drain from the capillary bed then they go to what called venules (veins- related branches) (5) and from the venules they come eventually into the veins (6) and again to vena cava system

- **compare the cross- sectional area of the capillary and cross-sectional area aorta and velocity:**



- As per cross- sectional area: the highest point is # 3 that describes capillaries, the lowest points are #1 (aorta) and #5 (vena cava); so, cross sectional area moving away from the aorta (and vena cava) becomes larger and as we going away from the capillary it's starting to going down (becomes smaller). However, radius is definitely the biggest in aorta and vena cava.
- As per velocity (which is the same as force or pressure): Aorta and then vena cava have the highest (strongest) while capillaries have the lowest (weakest). That is self- explanatory; as higher forces are needed to pump blood and receive it (function of aorta and vena cava) while low- to no forces are needed for gas exchange (function of blood capillaries).
- **As you increase the cross-sectional area the velocity decrease**
- **The velocity is the slowest in the capillaries and faster in the aorta**
-
- In the following matches; is the relationship direct or indirect (inverse)?

a. CO and P: direct
b. P and SA: indirect
c. CO and SA: indirect
d. F and SA: indirect
e. SV and EDV: direct
f. SV and ESV: indirect
g. A and flow: direct
h. V and F: direct
i. SA and V: indirect
j. CO and BP: direct
k. CO and HR: direct
l. SV and HR: indirect

لا عليك يا صاحبي. لا عليك.

هذه التي أتعبتك هي في نهاية المطاف دنيا! هكذا سماها خالقها كي لا نغتر بها.. فاستجمع قواك إن لك أن تمشيه وعلى كاهلك أمانة عليك أن تؤديها. وقد كان شعارك دوما: لا أبرح حتى أبلغ! فلا تبرح!