Physiology Sheet (Lecture 23)

❖ Blood pressure **must** be controlled.

❖ **The control of blood pressure is controlled through three major mechanisms:**

1. **Neural** (very fast)
2. **Hormonal** (intermediate speed)
3. **Renal** (slow)

Neural Mechanism

* It can bring blood pressure back to 75% normal and it will **not** replace the lost blood; the lost blood is lost.
* It starts from detecting the problem, which is change in the blood pressure, so if we have an increase in blood pressure in the walls of major arteries (specifically, *Cartoid* and *the Aortic*). The more the blood pressure the more the number of impulses reaching the brain, the less the pressure the less the number of impulses reaching the brain.
* ***Baroreceptors*** transmit the information to the brain in no time, and the brain responds in no time as well, that’s why the neural response is very fast, but you need to guarantee three things: **the efferent fiber** conducts impulses with a high velocity and high velocity conduction is proportional to the radius (the larger the nerve axon, the higher the velocity.), it also has to do with **the myelination**, if the fiber is myelinated, it can conduct impulses faster, but if the myelin sheath was removed as in some diseases that attack the myelin sheath and cause **demyelination**, here, the baroreceptor reflex will take a longer time to reach the brain. (This is very bad, very dangerous! :O)
* The baroreceptors are the first line of defense against fluctuations of blood pressure. If blood pressure drops, the number of impulses reaching the *Madulla* decreases, the *Madulla* will process this piece of information and understand that there’s something wrong –the blood pressure has decreased, therefore, it will fire on **the sympathetic** and inhibit **the parasympathetic**. The sympathetic goes to three places: the heart, the arteries and the veins. The sympathetic works normally as a mass production, whereas, the parasympathetic works by divergence, more by convergence; you can stimulate the stomach without stimulating the heart, for example. The sympathetic fires everywhere! Everywhere in **the Cardiovascular system** means:

**THE HEART**, in the heart, it goes to three places: **1. SA Node**, to increase the heart rate. **2. AV Node**, to increase the conduction velocity. **3. Ventricles**, to increase contractility. (By increasing the force of contraction, the stroke volume increases.)

**THE ARTERIES**, it cause them to decrease their diameter, thus, causing arterial constriction that will result in increasing pressure. (*P=F/A*)

**THE VEINS**, it causes **vasoconstriction**; squeezing the veins, which pushes the blood back to the heart, because in order for the heart to eject blood it must receive blood from the veins.

* Sympathetic stops parasympathetic; which **ALMOST** works inversely. Parasympathetic system has no effects on veins and ventricles, slightly affect the arteries, and affects SA and AV nodes.
* **Peripheral circulation**, starts from *the Aorta* and ends in the *Vena Cava*; blood flows from **the left ventricle** to **the right atrium**.

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* Blood won’t flow from A to B unless, the presser in A (Pa) is larger than the pressure in B (Pb). (*Ohm’s law*, the flow is directly proportional to the driving force.)
* Flow = (Pa-Pb)/Ra (Ra: resistance in the arterial system.)
* Blood flows in the arteriolar system only if the pressure in B is larger than the pressure in C.
* Pa = 100 (main arterial blood pressure) = 860 mmHg.
* Pe = 0 (pressure in the right atrium) = atmospheric pressure = 860 mmHg.
* **Question:** what is the volume of blood that flows in one minute from:

**A to E** 5 L/min

**A to B** 5 L/min

**B to C** 5 L/min

And that equals **the cardiac output**.

* **Q (A to E) = (Pa – Pe) / TPR**

**TPR: Total peripheral resistance,** resistance in series, the resistances the blood will face upon going from the *Aorta* until reaching the *Vena Cava*.

* Since Pe = 0, Pa is the driving force. (if Pe increases, that might cause a problem.)
* **Q: Cardiac output.**

**Pa: Arterial blood pressure.**

* Pa = Q \* TPR = HR (heart rate) \* SV (stroke volume) \* TPR. **Very Important!**
1. Resistance in a blood vessel (tube) is directly proportional to **the radius forth power**, that is a **slight** change in the radius will cause the resistance to change **significantly**. How to change the radius? By constriction and dilatation –sympathetic stimulation causes constriction in the arteries which increases resistance, thus, increasing the pressure. For the manipulation of the arterial blood pressure, what can be done? You can change the heart rate, stroke volume or the radius in order to manipulate the arterial blood pressure.
2. **The length of the tube** is directly proportional to the resistance, and it can only be changed by the formation of new arteries. When? Too much fat causes the formation of new arteries, which increases the length and the resistance as a result, thus, increasing the blood pressure. Note that under normal physiological conditions the length of the artery is not under control.
3. **Viscosity (type of fluid)**; the more viscous the fluid the harder to push it forward.

Control of Blood Pressure

* P = F / A ( F: Force: Blood volume in the arterial system = 750 mL)
* If you want to cure a high blood pressure patient, you probably aim to decrease the blood volume, but how?

Blood is composed of **45% cells** and **55% plasma** (92% water, 8% others, thus, plasma is basically water.)

So, you give the patient **Diuretics** (most common: *Lasix*) to decrease the blood volume **slightly**.

* **The blood pressure decreases, the number of impulses** -from *the Cartoid* (more important than *the Aortic* because it’s capable of detecting small changes in the pressure.) carried by *the Glassopharyngeal* nerve; the 9th cranial nerve, and *the Aortic* arch by *the Vegus* nerve; the 10th cranial nerve- **decreases**, and by firing the sympathetic system, increment in the heart rate and stroke volume occurs, and that also cause vasoconstriction (veino-constriction increases blood coming back to the heart to guarantee a stroke volume.) and finally **increasing the blood pressure**.
* **Sympathomimic**: (receptor) it interferes with the metabolism of **the norepinephrine** (first messenger.)
* **Sympathoagonist**: (before-receptor) it interferes with the same receptor of the norepinephrine and binds to it, preventing the norepinephrine from binding to it
* **Sympatholytic**: (-lysis: degradation) it breaks down norepinephrine, norepinephrine is not functioning here.
* **Sympathoantagonist**: prevent the original substance (e.g. hormone) from acting.
* **Sympatholytic and sympathoantagonist may both give the same effect**.
* A patient with high blood pressure: sympathoantagonist, why? Because we need to decrease the blood pressure.
* **High affinity** means if the concentration of the hormone is **too low** the receptor still can capture it.
* **The heart contains beta-receptors.**

To prevent Beta-receptors from functioning, **Beta-antagonist** must be given; **Beta-blockers**, which block **adrenergic receptors**; **norepinephrine receptors.**

* Beta-blocker is the most important drug used to lower high blood pressure, it blocks the binding site of the norepinephrine, thus, the heart rate does down, the blood pressure as well. (Norepinephrine is responsible for a high heart rate.)

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