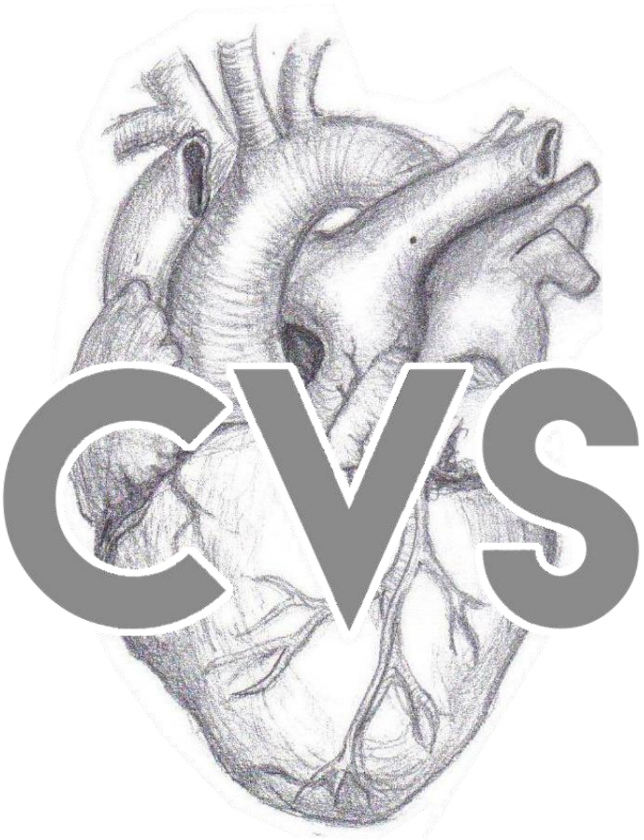

CVS

PHYSIOLOGY

☑ Sheet ○ Slide ○ Handout

Number

16

Subject

Blood Pressure Regulation

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This sheet was written according to the record from section 2.

Blood pressure regulation

Regulators of blood pressure:

1. **Short term regulators** are <u>fast</u> regulators. For example, if you were lying down and you suddenly stood up, the pressure will drop really fast, but there are very fast regulators that regulate this pressure to prevent the pressure from falling, otherwise the blood flow in the brain will be compromised and you will fall down. These short term regulators work through the nervous system (because neurons work the fastest).

2. **Long term regulators** include: epinephrine, antidiuretic hormone (ADH), Renin–Angiotensin–Aldosterone system, atrial natriuretic peptide (ANP), and kidney-body fluids.

3. **Intermediate term regulators** (will be discussed in the next lectures).

Mercury sphygmomanometer

This type of sphygmomanometers is no longer used because it might break, and the mercury is very toxic. Nowadays we use other types like <u>aneroid or digital</u> sphygmomanometers.

Whatever the type, the principle is the same.

- How to measure the blood pressure using a sphygmomanometer:

1. You put a cuff around the arm.
2. <u>You raise the pressure above the systolic pressure (120);</u> at this point you won't hear any pulsation because there's no blood flow (no sound).
3. <u>Then you decrease the pressure in the sphygmomanometer to a value that is equal to the systolic pressure</u>, at this point there is blood flow during the systole. However, because there is a constriction, the type of blood flow is **turbulent** flow, so you would hear tapping sound called **(Korotkoff sound**).

> **Note**:
> In general, the blood flow is **laminar**, which means it flows linearly and smoothly in adjacent layers, but under pressure or high flow, like in the ascending aorta the laminar flow becomes chaotic, disrupted and **turbulent**.

4. Upon decreasing the pressure in the sphygmomanometer you keep hearing these Korotkoff sounds, until there's no more compression in the artery, that's when you reach the diastolic pressure.
   At this point the flow changes from turbulent to laminar again, and the laminar flow has no sound, so the Korotkoff sounds disappear.
5. The first encounter with Korotkoff sounds marks the systolic pressure, and then the absence of the sound marks the diastolic pressure.

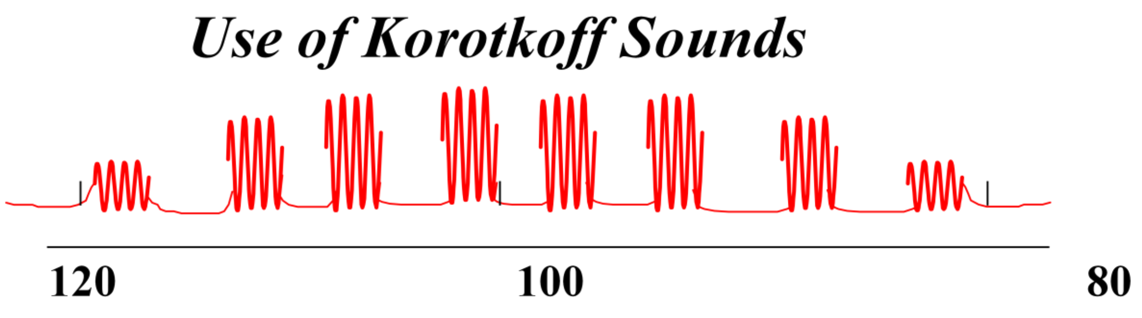
Notes:

- This method that requires sphygmomanometer with stethoscope to measure the blood pressure is called **(Auscultatory method**).
- Sometimes, if you don't have a stethoscope, you can use your hands to feel the pulse. this is called the (**Pulpatory method**) , you put your hands at the site of the brachial artery lower than the cuff , and you sense the pulse , upon rising the pressure in the sphygmomanometer above the systolic , you won't feel any pulsation as there's no blood flow in the artery , and that would mark the systolic pressure **. BUT, in** this method you can't measure the diastolic pressure, as the pulse won't disappear upon reaching the diastolic pressure. So this is only used to measure the systolic pressure.

- the effect of cuff pressure on brachial blood flow

Cuff pressure > 120	**NO FLOW**
Cuff pressure < 80	**FREE FLOW**

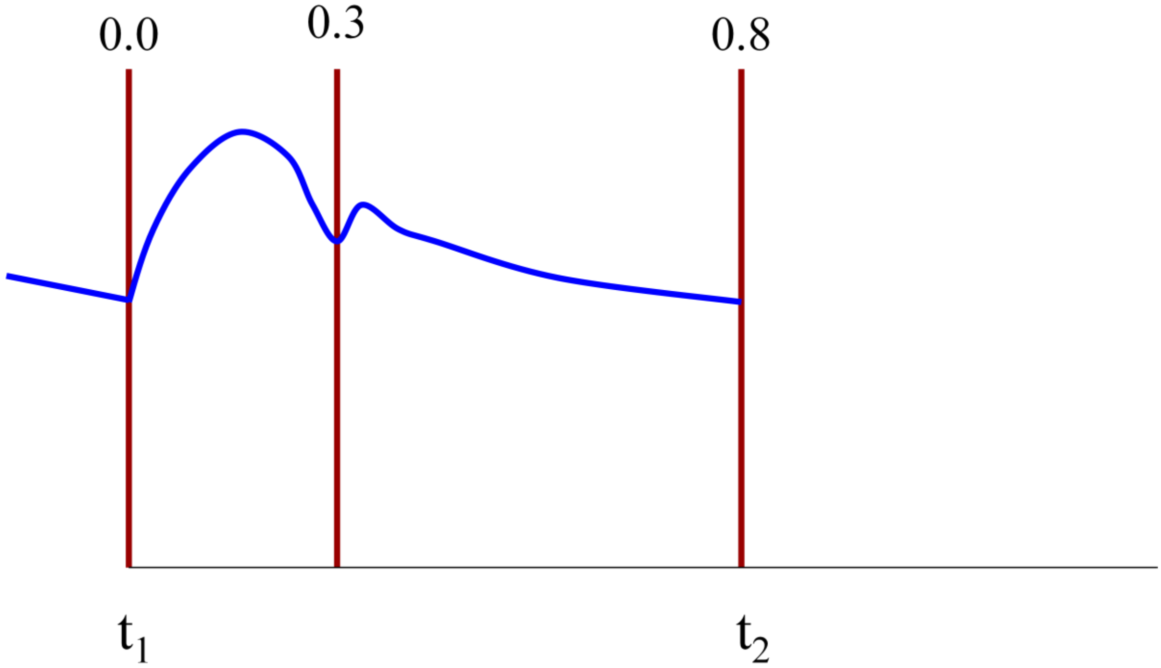
Korotkoff Pressure



In the diagram above, notice that Korotkoff sounds begin at 120 mm Hg, and disappear at 80 mm Hg because there's no compression anymore and the blood flow becomes laminar again.

Mean Arterial Pressure (MAP)

- It is the pressure that pushes the blood in the circulation.
- MAP is closer to the diastolic pressure than the systolic pressure, that's because the diastolic time is longer than the systolic time.
- It equals two thirds of the diastole, plus one third of the systole.



The figure shown above graphically represents the changes in the aortic pressure

How is the area under the scale calculated?

By an integration from t2 to t1

$$\boxed{MAP = \int_{t2}^{t1} dp.dt}$$

How to calculate the MAP from the scale?

We divide the area by the time interval (t2 – t1)

$$\boxed{MAP = \int_{t2}^{t1} dp.dt/(t_2 - t_1)}$$

This method of calculation is usually automated; you just connect the machine to a transducer that measures the pressure, and it will automatically calculate the MAP.

How can the MAP be calculated manually?
We use the following equations:

$$\boxed{MAP = 1/3 \text{ systolic pressure} + 2/3 \text{ diastolic pressure}}$$

Or

$$\boxed{MAP = \text{diastolic pressure} + 1/3 \text{ pulse pressure}}$$

Remember that: (Pulse pressure = systolic – diastolic)
Both equations are the same and give the same answer.

Factors Affecting MAP

CO = MAP / TPR
MAP = CO * TPR

To alter the MAP, we either change the <u>cardiac output</u> or the <u>total peripheral resistance.</u>

Remember:

- **The vessels are only supplied by the sympathetic** nervous system, and they are **NOT** supplied by the **parasympathetic** nervous system.
- **There is a basal amount of supply from the sympathetic to these vessels,** so the vessels normally are a little bit constricted, this is called **(the basal tone**), and it's similar to the skeletal muscle tone that we previously studied, which makes the muscle a little bit constricted (muscle tone). You can find the muscle tone in normal living individuals. However, you don't find this basal tone in dead bodies.
- The smooth muscles in the myocardium around the vessels are constricted a little bit. This is called the basal tone, and it's supplied by the sympathetic nervous system.
- **This basal tone provides a positive and a negative control,** which means it give the vessels the ability to constrict or dilate, but if this basal tone didn't exist, then the vessel can only constrict but not dilate.

- **The parasympathetic only supplies the atrial part of the heart.** Therefore, this means that the parasympathetic innervation has some <u>control over the heart rate.</u>

Parasympathetic >> controls heart rate (controls heart function only)
Sympathetic >> controls heart rate and contractility and the resistance in the vessels (controls both heart function and the circulation)

- **All vessels are supplied by sympathetic innervations except the capillaries (including precapillary sphincters and metarterioles)** because they don't have smooth muscles.
- Innervation of small arteries and arterioles allows the **sympathetic nerves to increase vascular resistance** when they are stimulated.
- **Parasympathetic nervous system is mainly important for controlling the heart rate via the vagus nerve.**

1. Baroreceptors

Let's assume that the blood pressure is always changing. If the blood pressure goes down it should be elevated to normal pressure, and if it goes up it should be dropped to normal pressure.
How does the system know that blood pressure is rising or dropping?
Baroreceptors or **pressoreceptors** sense any **change in the blood pressure** whether it was up or down, and accordingly they serve their purpose.
They are also called **high pressure regulators** because they work in areas of high pressure such as the aortic arch.

Where are they located?
If they were located in the leg, the person would die before they sense any change in the blood pressure. So logically:

1. <u>They should be very close to the heart</u>, so that they would sense any change in the pressure.
   They are called **aortic Baroreceptors** and they are found in the **aortic arch.**
2. <u>They should be found close to the brain</u>, because the brain is a vital organ and any compromised blood flow to the brain would affect the brain.
   These receptors are called **carotid baroreceptors** and they are found in the **carotid sinus** – because that's where the blood flows to in the brain.

Mechanism of action:

- The **aortic receptors** are innervated by a branch from the **vagus** nerve (cranial nerve number 10), and they send impulses that are transmitted to the cardiovascular center in the medulla oblongata in the brain stem.
- The **carotid receptors** are innervated by **a branch of the glossopharyngeal nerve** (cranial nerve number 9), AKA **Hering's nerve**. This nerve sends the impulses from these carotid baroreceptors to the cardiovascular center in the medulla oblongata in the brain. The carotid sinus is found just after the bifurcation of the common carotid artery in the internal carotid artery.
- <u>These baroreceptors are stretch receptors</u> which are found in <u>the wall</u> of the artery, so if the wall stretches they are stimulated.
- **When do we see more stretch?**
  When the <u>pressure increases</u>, the vessels are stretched. Therefore, when there's too much pressure, the <u>impulses from these receptors increases</u> and goes to the cardiovascular center.

- **The cardiovascular center is made up of two parts**, the <u>cardiac</u> and <u>the vascular</u> parts.

  1. **The cardiac part contains two parts**, <u>cardio-acceleratory</u> and <u>cardio-inhibitory</u>.
     - **The cardio-acceleratory** part sends its impulses down to the heart via sympathetic fibers to <u>increase the heart rate and contractility</u> which increases the stroke volume (positive chronotropic and positive inotropic).
     - **The cardio-inhibitory** part sends its impulses through the vagus nerve (parasympathetic) which decreases the heart rate and decreases contractility.

  2. **The vascular part is called the vasomotor center (VMC)** which is located bilaterally in the reticular **substance of the medulla oblongata** and is made up of **3 areas**: the <u>vasoconstrictor area</u>, the <u>vasodilator area</u>, and <u>the sensory area</u>.
     - The area that receives the impulses from the baroreceptors is the **sensory area**, and the sensory area <u>distributes these impulses to the rest of the areas in the vasomotor center and also to the cardiac part of the cardiovascular center</u>.
     - **The only area that transmits it's impulses down to the vessels is the vasoconstrictor area**. It sends its impulses through the sympathetic nervous system (the impulses go down to the spinal cord, then through a

sympathetic ganglion and finally exit the ganglion through a sympathetic fiber) and to the vessels. Thus, if this area is stimulated, it results in vasoconstriction and if it is inhibited then vasodilation will occur.

- **The vasodilator area and the sensory area work upward,** meaning that they send their impulses to the CNS. The vasodilator area sends its impulses through the vagus nerve (cranial nerve X).

CASE A

- Suppose there was an **increase in the blood pressure.**
- An increase in pressure causes **more stretch** of the wall of the carotid and the aortic arch.
- Stretching the arterial wall causes **more streams of impulses** to the cardiovascular center.
- This increase in the stream of impulses to the cardiovascular center **inhibits the vasoconstrictor area** (fewer impulses to the vessels which causes vasodilation, less resistance, decrease in the MAP) and **stimulates the vasodilator area**.
- The impulses also reach the **cardio-acceleratory** area through the sensory area and **inhibit it**. This means that the cardio-acceleratory area will send <u>less sympathetic stimulation to the heart</u> (less HR→less contractility→less SV→less CO and when the CO goes down, the MAP goes down), and it **stimulates the cardio-inhibitory** area, which <u>sends more parasympathetic stimulation to the heart</u> (less HR→less CO→decrease in MAP).

CASE B

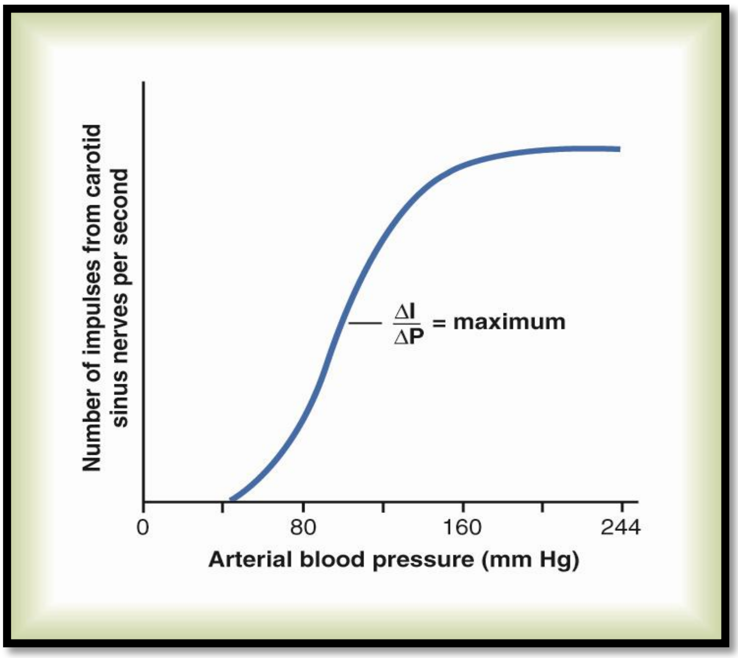
- Suppose there was a **decrease in the blood pressure.**
- The **number of impulses sent from the baroreceptors decreases**.
- When the number of impulses decreases, the **cardio-acceleratory area** is not inhibited anymore, so it is **stimulated** (more sympathetic stimulation to the heart→more HR→more contractility→more SV→more CO→increase in the MAP), and the **cardio-inhibitory area in inhibited**.
- The inhibition on the vasoconstrictor area is also removed because there are fewer impulses, so the **vasoconstrictor area is stimulated** (causes vasoconstriction in the vessels, increases the total peripheral resistance, and hence increases MAP).

Arterial Baroreceptor Reflex

- This reflex is <u>initiated by stretching in the walls</u> of the <u>large systemic arteries</u> which stimulates the **stretch receptors** (AKA baroreceptors, or pressoreceptors) that exist in the walls of these arteries.
- **These receptors will send impulses to the VMC**, which works to <u>decrease the arterial pressure</u> back to **normal** (by the mechanisms mentioned previously).
  - **Note**: these baroreceptors are considered **buffers** for the blood pressure, as <u>buffers always maintain things in their normal range</u> and just like the buffer system, which depends on pKa in its function, the **arterial baroreceptor reflex works best around 100 mmHg.**
- Baroreceptor reflex is **most sensitive at 100 mmHg**, which means that the reflex <u>changes easily with any small increase or decrease in the arterial blood pressure</u> around 100 mm Hg, but if the change in pressure is much higher or much lower than 100, then the reflex doesn't change drastically.
- Carotid sinus baroreceptors respond to pressure **between 60 and 80 mmHg.**
- Baroreceptor reflex is most sensitive around 100 mm Hg which is the MAP.

Observe the following sigmoid curve:

ΔI = change in impulses
ΔP = change in pressure



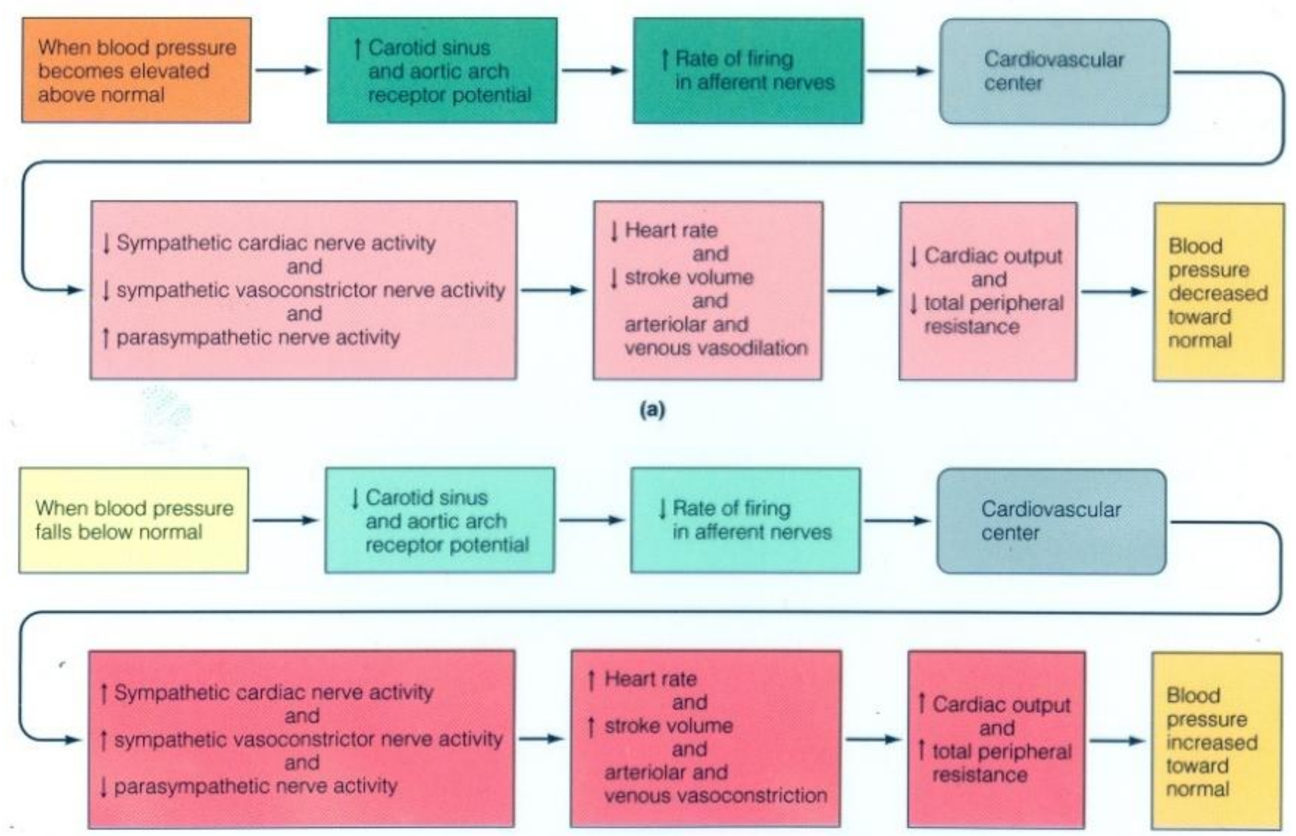
✓ **ΔI / ΔP** indicates the point where <u>any small change in pressure up or down causes a huge change in the number of impulses per second</u>. **This point is 100 mmHg**, which means baroreceptors are most sensitive around 100 mmHg.

✓ Notice that if the pressure increases or decreases too much away from 100, then **the sensitivity decreases drastically**, and no matter what the change in the pressure was, the **number of impulses won't change.**

Notes:

- Rate of firing = impulses sent from the baroreceptors
- The reflex works to maintain the normal blood pressure
- If BP increases, the reflex decreases→the BP returns **back to normal.**
- If BP decreases, the reflex increases→the BP returns **back to normal.**

Baroreceptor Reflexes to Restore Blood Pressure to Normal



What happens when we stand?

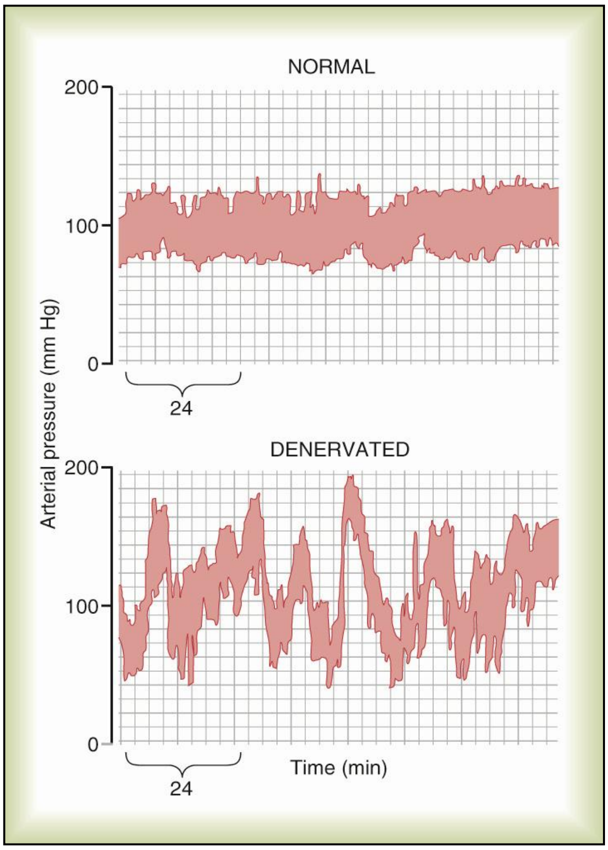
- When we stand, the <u>venous return to the heart is decreased</u> → CO and the arterial pressure decrease.
- The baroreceptors sense this change in the arterial pressure, and send impulses to increase the sympathetic stimulation to the heart to increase the CO and the arterial pressure.
- <u>This process happens really fast in young individuals.</u>
- <u>In older people</u>, the baroreceptors **are less sensitive** because of the atherosclerosis. This makes them feel dizzy once they stand up after they've been lying down, so older people are advised to stand slowly to avoid feeling lightheaded.

Functions of Baroreceptors

1. **Maintain relatively constant pressure despite changes in body posture**
   - If we wanted to measure the change in the blood pressure after denervating the baroreceptors (cutting their innervations), what would it be?
   - Provide the patient with a pressure transducer for 24 hours which will measure the blood pressure many times a day.
   - When the **baroreceptors are innervated** (normal), the readings would be around 100 mm Hg most of the time.
   - However, **if the baroreceptors were denervated**, we would find 25 readings that give 100, and the remaining 75 readings would be split in half: one part with readings higher than 100, and the other part with readings lower than 100. In this case, the range and the variations are much higher (not part of homeostasis), while in the innervated baroreceptors the range will be very close to 100, which means that these receptors are part of the homeostasis and they maintain the BP around 100.

2. **Oppose either increases or decreases in the arterial pressure thereby reducing the daily variations arterial pressure.**
3. **They are insignificant in long term control of arterial pressure because the baroreceptors adapt.**

The figure on the right shows variation in the arterial pressure in normal and denervated baroreceptors.



Remember:
- CO = SV × HR
- SV increases by increasing the end diastolic volume, or by increasing the sympathetic stimulation.
- The end diastolic volume increases by increasing the venous return.
- The heart rate increases by the sympathetic stimulation and decreases by the parasympathetic stimulation.

How do the sympathetic and parasympathetic nerves affect the cardiovascular system?

1. **Parasympathetic**: only works on the heart→it decreases HR→less CO→decrease in BP.
2. **Sympathetic**: works on three parts:
   i. Heart: it increases contractility which increases the SV + HR. They both increase the CO, which causes an increase in the BP.
   ii. Arterioles: it causes vasoconstriction, which increases the total peripheral resistance, thus increasing the BP.
   iii. Veins: causes venoconstriction → increase in the **mean systemic filling pressure MSFP** (that's because veins contain 2/3 of the blood volume) → increases the venous return→higher SV→more CO→increase the BP.

2. Low Pressure Regulators

- These are located in low pressure areas, in the **right atrium** and **the right ventricle** (and sometimes in the pulmonary arteries).
- These are sensitive to **changes in volume.**
- If there is **an increase in blood volume** → increase in venous return → increase CO → increase in MAP → which causes a **decrease in the pressure in the right atrium**
- In order to fix this decrease in pressure, we should bring the other factors (CO, MAP, venous return) down, or increase the venous return to the right atrium, this is done by low pressure baroreceptors that are found in the right and left atria.

Atrial Hypothalamic Reflex

- In this reflex, the right atrium and the hypothalamus communicate.
- When there’s an increase in right atrial pressure, the receptors send impulses to the hypothalamus, and **the hypothalamus reduces the secretion of ADH.**

- **ADH has two important jobs:**
  1. Water reabsorption from the collecting ducts in kidneys. This retained water will contribute to the extracellular volume (ECV). If ECV increases then blood volume increases, and the mean systolic filling pressure will be increased→more venous return→more end-diastolic volume→higher SV→higher CO.
  2. Vasoconstriction
     If ADH is decreased → less vasoconstriction and more vasodilation.

- **That's why ADH has two names**; one for every function. The first name is antidiuretic hormone, and the second is vasopressin.

In summary, if the BP is low in the right atrium, more venous return is needed so ADH is stimulated. If the BP in the atrium is high, less venous return is needed so ADH is inhibited.

Atrial Renal Reflex

- In this reflex, the right atrium and the kidney communicate.
- An increase in the volume of the right atrium means high BP in the right atrium.
- Right atrium sends impulses to the kidney to dilate the afferent arterioles that supply the nephron; this increases the blood flow to the nephron→increases the glomerular filtration rate (GFR).
- When GFR increases this means that more water is lost through the kidney→ECV decreases→the venous return decreases→lower SV→lower CO→lower MAP.
- On the other hand, if there was a decrease in the blood volume in the right atrium that would cause vasoconstriction in the afferent arterioles and less water is lost through the kidneys.
- Affecting the GFR means affecting the urine volume.

3. Chemoreceptors

- These receptors are **located at the same areas as the baroreceptors** (in the aortic arch and the carotid sinus).
- They are called the **aortic bodies** and the **carotid bodies**.
- The blood flow around these receptors is very high to an extent where the O_2 and the CO_2 concentrations around these areas are the same as the O_2 and CO_2 concentrations in the arteries.
- These receptors are sensitive to pO_2 (partial pressure of O_2), pCO_2, and the hydrogen ion concentration (pH).
- These receptors are called **peripheral chemoreceptors**
- The peripheral chemoreceptors are highly sensitive to:
    1. **Low** O_2 concentration
    2. **High** CO_2 concentration
    3. **High** H concentration

- **High BP**→High flow→high pO_2 and low pCO_2 and low H concentration (high pH)→receptors respond to these changes→send impulses to the vasomotor center and inhibit the vasoconstrictor area→vasodilation→the pressure decreases back to normal.
- **Decrease in the BP**→minimal flow to the chemoreceptors→high CO_2, low O_2, and high H ions (lower pH)→chemoreceptors are stimulated→they will send more impulses to the vasoconstrictor area→vasoconstriction in the vessels→increase the TPR→increase BP.
- **Peripheral chemoreceptors are highly sensitive to a <u>decrease in oxygen concentration.</u>**
- **Central chemoreceptors are highly sensitive to an <u>increase in CO_2 concentration</u>.**
- Both peripheral and central chemoreceptors are very important in **respiratory control.**

Peripheral Chemoreceptors in Chronic Smokers

- The peripheral chemoreceptors are very important to chronic smokers who have **respiratory failure**.
- In respiratory failure, the central chemoreceptors have **adapted** to high CO_2 concentrations (adaptive receptors), so they **are not sensitive** to changes in CO_2 concentration anymore. In this case, the main effectors on the respiratory control are the peripheral chemoreceptors, because they are highly sensitive to any reduction in O_2 concentration. The concentration of O_2 in chronic smokers is very low while the CO_2 concentration is very high.
- Chronic smokers who have respiratory failure **depend on the stimulation** of the respiratory center through the impulses that come **from the peripheral chemoreceptors only.** *Therefore, these patients cannot be given high oxygen concentrations through a mask because this would over-stimulate the peripheral chemoreceptors (which are highly sensitive to pO_2) to give rapid impulses to the respiratory center. Such patients should be provided with oxygen gradually through nasal tubes.*

Forgive me for any mistakes.

Revised and Edited by: Amer M. Sawalha