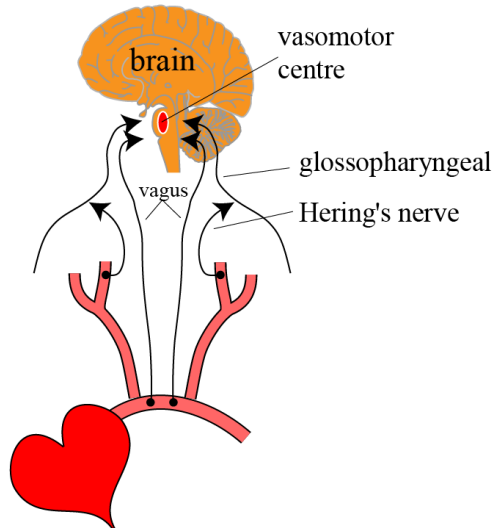


B.6.2. Short-term Regulation

Introduction:

1. Short-term regulation of the blood pressure includes those regulators that work very fast, from seconds to minutes. There are also long-term regulators but these take days to weeks to work (see next page).	2. There are several short-term regulators: 1) Baro reflex 2) Chemo reflex 3) Vascular fluid shift
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A. Baro-reflex:

1. This is a real nerve reflex. This means it has a reflex arc . In general, reflex arcs consists of: 1. a sensor 2. afferent nerves 3. a centre in the Central Nervous System (= CNS) 4. efferent nerves 5. an effector.	2. In the case of the baro-reflex, these components are as follows: Sensors: the baro-receptors, which are located in the internal carotid arteries (located in the carotid sinus) and along the aortic arch. These are stretch sensors . When they are stretched (because of higher blood pressure), they will send more action potentials to the CNS. If the blood pressure is decreased, then the firing frequency is decreased. Therefore, they 'sense' the blood pressure.
3. Afferent nerves; the nerves from the aortic receptors "run" through the vagus nerve. The nerves from the carotid sinus go through Hering's nerve to the glossopharyngeal nerve.	The efferent nerves of the Baro reflex:  BasicPhysiology.org
4. Both nerves end in the cardiovascular centre (= vasomotor centre) in the medulla of the brain.	
5. The efferent nerves are: a) the vagus (the para-sympathetic system) and b) the sympathetic system.	
6. The effectors are: a) the heart b) the arteries c) the veins	

A. How does the Baro reflex work?

Step 1: Suppose the blood pressure decreases suddenly.	
Step 2: The baro-receptors will sense this and respond by producing less action potentials	
Step 3: The Vasomotor centre will respond by influencing two systems: - the Parasympathetic system - the Sympathetic system	Function diagram of the Baro-reflex <pre> graph TD 1[1 arterial blood pressure ↓] --> 2[2 stretch on baroreceptors ↓] 2 --> 3[3 firing of action potentials to the vasomotor center ↓] 3 --> 4a[4a Parasympathetic drive ↓] 3 --> 4b[4b Sympathetic drive ↑] 4a --> HR[heart rate ↑] 4b --> HR 4b --> C[1. heart rate ↑] 4b --> 2C[2. contractility ↑] 4b --> 3C[3. constriction of arterioles and thus increase in peripheral resistance ↑] 4b --> 4C[4. constriction of veins: a. decrease in blood capacity ↓, b. increase in venous return ↑] HR --> ABP[arterial blood pressure ↑] 3C --> ABP 4C --> ABP </pre>
Step 4A: The parasympathetic system is then inhibited: this will increase the heart rate (by increasing the frequency of the cardiac sinus node).	
Step 4B: The sympathetic system is stimulated. This has multiple effects: 1) increase in heart rate (= chronotropic) 2) increase in contraction force of the heart (= inotropic) 3) vasoconstriction of the arterioles (this leads to an increase in the peripheral resistance). 4) vasoconstriction of the veins: this has two effects: a) decreasing the capacity of the veins b) increasing the venous return	4b) Increase in the venous return. This will make the heart beat: a. faster (sinus node stretch). b. stronger (Frank-Starling effect).
4a) The capacity of the veins is decreased (remember that the veins are also being used as a buffer, a capacity or reserve). If these large veins constrict more, more blood will be pushed into the circulation, effectively increasing the circulating blood volume.	

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C. Wait! There is something strange here!

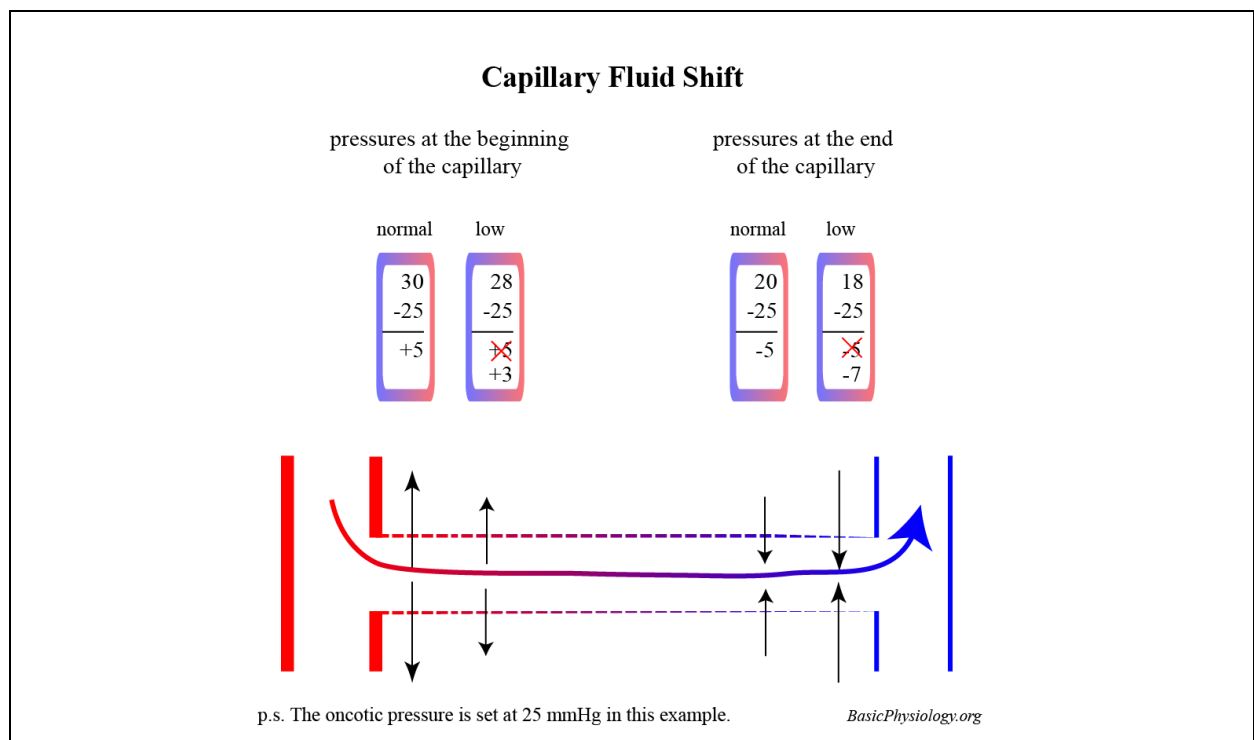
1. In the above diagram, I said that the parasympathetic system is inhibited and that this will increase the heart rate??	2. Till now, every time we have stimulated nerves (in muscles, heart etc.), we got more activity, not less. So, how is inhibiting a nerve going to increase the firing rate in the heart??
3. Good! This is an important point, which I will now try to explain.	4. First of all, you must know that the heart, by itself, without any interference of nerves, hormones or whatever, would beat automatically and at a higher rate than we are used to; typically at about 110 beats/min. This is called the intrinsic rate .
5. But in the body, at rest, the heart rate is much lower, at about 60-70 beats/min. Why is this?	6. Because, at rest, the parasympathetic nerves (i.e., the vagus) is active and is inhibiting the sinus node. This will decrease the normal heart rate.
intrinsic rate 110 normal heart rate at rest ~ 70 90 130 150 — heart rate during exercise parasympathetic activity sympathetic activity BasicPhysiology.org	
7. And, if necessary, for example during exercise, inhibition of the parasympathetic system, will 'release' the brake on the sinus node that will, by itself, then produce more action potentials.	8. In my class, I always compared this with the behaviour of a sports car in front of a red light! You may have seen something like this: you see the car shaking because the driver is at the same time pushing on the gas pedal and on the brake (and smoke coming out of the exhaust!).
9. When the light turns green, the driver releases the brake and his car (<i>it is always a 'he'!</i>) suddenly roars ahead of all the other cars.	10. The same happens in the heart. Normally, the active parasympathetic system 'brakes' the heart. When the brake (=inhibition) is released, the heart jumps quickly to a higher rate. That's the trick!

D. Chemo reflex:

1. The chemo reflex is very similar to the baro reflex. The only difference is that the sensors are not sensitive to stretch but sensitive to blood concentrations of oxygen , carbon dioxide and pH .	2. These chemoreceptors are also located in the aortic bodies and the carotid bodies, close to the corresponding stretch receptors.
3. Because the receptors are a bit slower than the stretch receptors, this reflex works slower.	4. Because they measure blood gasses, their function is much more important in the regulation of the respiration .

E. Vascular Fluid Shift:

1. Another way to regulate, in the short term, the blood pressure is not by influencing the heart but influencing, at the other end of the circulation, the capillary system!	
2. As you may remember, in the capillary, some of the fluid in the blood filtrates out of the capillary thereby carrying oxygen and nutrients to the cells. At the end of the capillaries, most of this fluid is reabsorbed back into the circulation.	3. But this system, to some extent, is sensitive to the arterial blood pressure. And this system tends to minimize the increase or decrease in blood pressures. This can be seen in the following example:



4. In the normal situation, the hydrostatic (=blood) pressure at the beginning of the capillaries is typically 30 mmHg and at the end of the capillaries 20 mmHg.	5. Suppose that the blood pressure has decreased a little. Therefore, the pressure at the beginning of the capillary will also, slightly, decrease to, let's say, 28 mmHg.
6. The oncotic pressure has not changed of course as the blood still has the same amount of albumin.	7. Therefore, the net filtration pressure at the beginning decreases from +5 to +3 mmHg. This means that less fluid will leave the capillary.
8. At the end of the capillary, the hydrostatic pressure will also have decreased, from 20 to 18 mmHg leading to a net filtration pressure of -7 mmHg. This means that more fluid will be reabsorbed into the blood.	9. This therefore causes a shift of fluid , from the interstitial space to the vascular space. This increase in blood volume will then increase the blood pressure .
10. The opposite of course happens when the blood pressure increases. Then, more fluid will go out of the capillary and this will reduce the blood pressure.	11. The amount of fluid shift is actually very little, about 5-10% of the plasma volume. Remember that the plasma volume is about 50% of the total blood volume. Therefore, these shifts typically only use 100 to 200 ml plasma.

F. The stupid Physiologist!

1. There was once a physiologist who thought he had a clever idea.	2. This idea, he thought, was so good he would become famous, win the Nobel prize and earn a lot of money.
3. As you know, hypertension is a big problem and difficult to treat.	4. Our soon-to-become world-famous physiologist, with the help of the Baro reflex, invented a clever way to treat hypertension.
5. His idea was to stimulate the Hering's nerve with an artificial (implanted) pacemaker!	6. In this manner, the vasomotor centre would think that the blood pressure was too high and would react by exciting the parasympathetic system and inhibiting the sympathetic system to decrease the blood pressure.

7. In his first group of hypertensive patients, his plan worked!! Indeed, the blood pressure decreased as soon as the pacemaker was switched on!	8. But after a few weeks, the blood pressure started to creep back to hypertensive levels, even while the pacemaker was switched on. In fact, when the pacemaker was switched off, the blood pressure became even higher than before the implantation of the pacemaker!
9. The poor physiologist, instead of treating his patients, was actually making things worse. Gone were his dreams of the Nobel prize, richness and fame.	10. Why had he failed? Why did his idea not work? Because stimulating through Hering's nerve is a short-term solution and NOT a long-term solution. Hypertension is a problem with the long-term regulation of the blood pressure (see next page).
Additional note: In recent years however, new technologies are being developed to treat hypertension by influencing the baro reflex! However, the point of this new potential treatment is not to influence the short-term regulation of the baro reflex but, instead, by stimulating the vagus nerve, thereby influencing the set point in the vaso-motor centre in the brain. This could then reduce the blood pressure in patients. To be followed ...!	