

Control of heart rate and Blood Pressure Revision Sheet.

Action potentials originate in the sinoatrial node and travel across the wall of the atrium to the atrioventricular node on the right side of the heart. This passes slowly giving time for the atria to contract and empty all the blood into the ventricle. This is then passed along the atrioventricular bundle into the interventricular septum along the bundle of His. This bundle separates into two at the apex (bottom) of the ventricles and goes upwards along each of the ventricle walls. The action potentials travel along this pathway and is carried deeper into the ventricular walls by the Purkinje fibres. This provides unison of a strong contraction by both the ventricles and allowing the ventricles to empty properly.

Chemoreceptors, located in the aorta and the carotid artery, monitor the CO_2 levels, O_2 levels and pH levels in the blood. When the receptor picks up the stimulant it transfers it to the sensory neurone to the medulla oblongata (also known as medulla) which also has chemoreceptors. The impulse then travels along the sympathetic neurone or parasympathetic neurone depending on the stimuli to the sinoatrial node. The effector, the cardiac muscle, can have two responses depending on the stimulus and route it takes:

- If there are low levels of O_2 and pH with high levels of CO_2 then the impulse will go through the sensory neurone to the medulla and through the sympathetic neurone releasing noradrenalin from the sinoatrial node (an excitatory neurotransmitter) which makes the heart rate faster.
- If there are high levels of O_2 and pH with low levels of CO_2 then the impulse will go through the sensory neurone to the medulla and through the parasympathetic neurone releasing acetylcholine from the sinoatrial node (an inhibitory neurotransmitter) which makes the heart rate slower. This is so the levels of O_2 and pH can lower and CO_2 levels can be raised.

Baroreceptors, located in the carotid artery and aorta only, detect changes in the pressure in the blood. The routes are exactly the same for which the nerve impulses take:

- If there is high blood pressure the route it follows is through the parasympathetic neurone which releases acetylcholine from the sinoatrial node slowing the heart rate down to lower the blood pressure.
- If there is low blood pressure the route it follows is through the sympathetic neurone which releases noradrenalin from the sinoatrial node making the heart rate faster to raise the blood pressure.

Control of Blood Pressure Summary

If blood pressure within the aorta or the carotid sinus increases, the walls of these arteries stretch and stimulate increased activity within the baroreceptor. This information is then sent via nerves to the cardio regulatory center within the medulla, which responds by initiating mechanisms that decrease the blood pressure to a normal level. Let's take a look at what happens to bring your blood pressure back down to a normal level when it gets too high.

To lower blood pressure, we first see a decrease of sympathetic input and an increase in parasympathetic input to the heart. We previously learned that the sympathetic nervous system can increase heart rate and stimulate the heart muscle to pump with more force. We also learned that the parasympathetic nervous system can decrease the heart rate. Therefore, by shutting off the sympathetic stimulation and boosting the parasympathetic stimulation, we decrease the heart rate and stroke volume, which decreases the cardiac output and decreases blood pressure. Second, if the baroreceptors are detecting that blood pressure is too high, the cardio regulatory center of the medulla will also decrease sympathetic input to the blood vessels. This causes vasodilation, which decreases total peripheral resistance and decreases blood pressure.

The opposite happens when the baroreceptors of the aorta or carotid sinus detect a drop in blood pressure. A decrease in blood pressure causes a decrease in action potentials sent to the cardio regulatory center of the medulla. Therefore, to raise blood pressure, the body will first cause an increase in sympathetic nerve activity to the SA node, causing it to fire more frequently, which increases the heart rate. The heart muscle is also stimulated to pump with more force, and this increases the stroke volume. When heart rate and stroke volume increase, we see an increase in cardiac output. As we learned, an increase in cardiac output causes an increased blood pressure, restoring blood pressure back to a normal level. Second, this causes an increased sympathetic input to the blood vessels, which stimulate the smooth muscle to contract, causing vasoconstriction, which increases total peripheral resistance and increases blood pressure.

The Renin-Angiotensin System and Blood Pressure Control

The renin-angiotensin system or RAS regulates blood pressure and fluid balance in the body. When blood volume or sodium levels in the body are low, or blood potassium is high, cells in the kidney release the enzyme, renin. Renin converts angiotensinogen, which is produced in the liver, to the

hormone angiotensin I. An enzyme known as ACE or angiotensin-converting enzyme found in the lungs metabolizes angiotensin I into angiotensin II.

Angiotensin II causes blood vessels to constrict and blood pressure to increase. Angiotensin II stimulates the release of the hormone aldosterone in the adrenal glands, which causes the renal tubules to retain sodium and water and excrete potassium.

Together, angiotensin II and aldosterone work to raise blood volume, blood pressure and sodium levels in the blood to restore the balance of sodium, potassium, and fluids. If the renin-angiotensin system becomes overactive, consistently high blood pressure results.