Course: Electrophysiology of Heart

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Lecture 14: Heart rate & Blood pressure- Baroreflex pathway

Hello everyone. So, today we will start our next topic that is heart rate and heart rate and blood pressure the baroreflex pathway we will discuss today. So, the concepts will cover what are the regulatory mechanisms or for blood pressure that is the components of regulation of blood pressure. We will talk mainly about the short term regulation the baroreceptors and the chemoreceptors. The baroreceptor reflects 2 reflex we will mainly talk that is the baroreceptor reflex and the bridge reflex. Now when we talk about blood pressure blood pressure is usually defined as the lateral pressure which is exerted by the column of blood on the wall vessel wall.

So, this lateral blood pressure has got various entities like systolic blood pressure, the maximum pressure at the systole which is liable to fluctuations. And the main important points to be remembered in systolic blood pressure that means, it what are the factors which affect the systolic blood pressure the stroke volume and the compliance. The compliance is inversely related to the systolic blood pressure. Then we have the diastolic blood pressure.

Diastolic blood pressure is the minimum blood pressure which is present during the diastole. So, here elastic recoil of ota and total peripheral resistance these 2 are the important factors which govern the diastolic blood pressure you have to remember. Then we have come across an another entity known as pulse pressure. The pulse pressure is usually nothing, but the difference between the systolic blood pressure and the diastolic blood pressure. So, what is the systolic blood pressure around 120 millimeter of mercury if we take if we are taking the average reading I am not talking about the range.

Diastolic blood pressure 80 millimeter of mercury. So, this pulse pressure will be 120 minus 80 that is 40 millimeter of mercury. The second thing is the mean atrial pressure. Now, this mean atrial pressure is the average or the mean pressure which is usually present throughout the cardiac cycle to ensure adequate perfusion of all the organs. So, mean atrial pressure is not the arithmetic mean of diastolic blood pressure or systolic blood pressure had it been arithmetic arithmetic mean then it would have been systolic

blood pressure plus diastolic blood pressure divided by 2, but it is not that.

We can say it is a weightage mean two third of the diastolic blood pressure and one third of the systolic blood pressure that will around a count to 93 millimeter of mercury. Now, why is it so up two third of diastolic blood pressure and one third of systolic blood pressure it is mainly because of the weightage is mainly because of the period or the duration of the ventricular diastole or the ventricular systole. We know in a cardiac cycle of 0.8 seconds duration 0.5 seconds it is ventricular diastole and 0.

3 second is ventricular systole. So, the maximum duration is of ventricular diastole and the maximum filling occurs in the ventricular diastole. So, that is why the mean atrial pressure is two third of diastolic blood pressure and one third of systolic blood pressure. So, with this basics of blood pressure there are various you know factors which occurs changes in the blood pressures setting positions standing positions running positions in pathetic activities and all. So, those I am not going through that you can go through your book the concepts which is very important is the regulation of the blood pressure.

Now, how blood pressure is regulated the blood pressure is regulated mainly based on three mechanisms depending on the time period. Within seconds I if I want to compensate my blood pressure for that short term or rapid mechanism is important which occurs within seconds. In case of hormonal mechanisms or short term mechanisms or intermediate mechanisms that usually occurs minutes or days it usually takes minutes or days to bring the blood pressure back to normal. And here usually we have catecholamines vasopressing angiotin two mediated actions, then we have capillary fluid shift mechanism, then we have stress relaxation of the blood vessels. So, these are the various mechanisms by which the intermediate blood pressure regulatory system is done.

The long term mechanisms means days and months it requires various days or months for the blood pressure to bring back to normal. And that is usually done by the most important system that is renin angiotensin aldosterone system. But we are concerned here since our topic is cardiac autonomic tone. So, we are mainly concerned with the short term regulation of blood pressure. Short term regulation of blood pressure we will be talking about the baroreceptor reflects the chemoreceptor reflects mainly and the last resort is the CNS or the ischemic response.

So, the baroreflex pathway mainly consists of the baroreceptors. Now, what are these baroreceptors and chemoreceptors where are they situated? We have the common carotid artery, when the common carotid artery bifurcates into internal carotid artery and external carotid artery. The internal carotid artery shows a bulging that bulging consists of the

baroreceptor that is carotid sinus that group of cells which is present in that bulge of the internet carotid artery that is carotid sinus. And carotid sinus consisting of group of cells means it will consist of nerve endings. Another body is present which is present in between the external carotid artery and the internal carotid artery mainly that is carotid body.

Carotid body is a chemoreceptor, carotid sinus is the baroreceptor. Now, when I am talking about artery that means, it is a blood vessel it must be having its layers like tunica intima, tunica media, tunica adventacia that means, this baroreceptors are present in which layer. So, this baroreceptors are present in the tunica adventacia layer to be very specific. And this baroreceptors are usually mechanosensitive or stretch sensitive. So, whenever there is a stretch in the blood vessels this baroreceptors will get activated.

This baroreceptors usually act in the range of blood pressure, this usually act in the change in the blood pressures. Now, the changing the blood pressure which type of pressure, which type of pressure entities we have systolic blood pressure, we have diastolic blood pressure, we have pulse pressure, we have mean arterial pressure. So, the baroreceptors are usually sensitive to pulse pressure and mean arterial pressure. The next important thing is the mean arterial pressure ranging or the pulse pressure ranging I mean the blood pressure ranging will be how much that this baroreceptors will act. The baroreceptors will act in a blood pressure range of usually 50 millimeter to 200 millimeter of mercury.

So, this much you have to remember in case of baroreceptors. And the carotid sinus besides carotid sinus in the internal carotid artery, we have baroreceptors also present in the wall of aorta that is aortic arch which consist of aortic body. So, this is also the baroreceptors. So, the baroreceptors if we conclude it is carotid sinus and aortic body. So, this is the baroreceptors.

Then we have the chemoreceptors also as I had already told you the carotid body which is present in the bifurcation of the external carotid artery and the internal carotid artery more towards the external carotid artery that is the chemoreceptors carotid body. The chemoreceptors usually they are subjective to the change in the acidic at the pH whether the H plus ions. And also the partial pressure of oxygen the partial pressure of carbon dioxide. Now, this baroreceptors the impulses from the baroreceptors are carried to the center for the reflex pathway to occur. These are mainly carried by the nerves.

The nerves which are getting carried from the carotid sinus they are known as sinus nerve or that is the glossopharyngeal nerve. The nerve which is getting carried from the aortic body or the wall of the aorta that is the baroreceptors of aortic body that is the

vagus nerve or mainly the tenth cranial nerve. So, we have two nerves over here the glossopharyngeal nerve which is also termed as sinus nerve and the vagus nerve. So, these are the afferents from the baroreceptors which take the signals to the center. The center of this reflex baroreflex pathway is medulla.

So, the center we will see this is the medulla. The simplified diagram which is I will show is the baroreceptors the signals are getting transferred via tenth nerve and ninth nerve glossopharyngeal and tenth cranial nerve to the medulla. Now in medulla we have nucleus tractus solitarius. This NTA stands for nucleus tractus solitarius which acts like a relay center. So, this nucleus tractus solitarius will give positive input or glutaminergic fibers to the vasomotor center.

The one of the vasomotor center is CVLM. CVLM is Cordoventrolateral medulla. This is nothing but the depressor area. So, Cordoventrolateral medulla is depressor area actually. So, this Cordoventrolateral area will send negative input to the Rostroventrolateral medulla.

Now positive input means here we have glutaminergic fibers excitatory neurotransmitter and negative input means here we have inhibitory neurotransmitter that is GABA. So, impulses coming from the baroreceptors. The baroreceptors afferent impulses through the sinus nerve and the vagus nerve going to the nucleus tractus solitarius. From nucleus tractus solitarius the input is going to the Cordoventrolateral medulla. The excitatory input and Cordoventrolateral medulla is suppressing the Rostroventrolateral medulla.

Otherwise this Rostroventrolateral medulla is usually tonically active which means if there is no inhibition from the Cordoventrolateral medulla this Rostroventrolateral medulla would have been tonically active. And from here the sympathetic outflow is gone usually goes to the heart. That means there will be increase in the heart rate there will be increase in the contractility and the vasoconstriction. Now another pathway is there from nucleus tractus solitarius which usually gives inputs to the cardiac inhibitory center or cardiac vagal center. This cardiac inhibitory center means which keeps a check on the cardiac which keeps a check on this particularly the parasympathetic domain.

So, the cardiac inhibitory center or the cardiac vagal center means it will keep the heart under inhibition. So, this when it will get stimulated it will cause inhibition more inhibition. That means decrease in the heart rate if it is less stimulated or if there is inhibition of the cardiac inhibition center. That means there will be increase in the heart rate. So, the vice versa signals is passed through the parasympathetic fibers.

So, that is the simplified diagram and this is nothing but the baryoreceptor reflects the

pathway which is shown in the figure. Nucleus tractus solitarius RVLM, CVLM and the glutaminergic fibers the GABA fibers already been told you. And the pathways are sympathetic fibers and the parasympathetic fibers which will going to the myocardial and the blood vessels. Now we will see how this baryoreceptors are acting. Now suppose there is a baryoreceptor firing.

Now baryoreceptor firing suppose there is baryoreceptor firing means whenever there is a increased in the sinus pressure. That increase in the sinus pressure can be done if I do carotid sinus messaging. When I do carotid sinus messaging if I massage the area of the carotid sinus there will be increased sinus pressure. So, increased sinus pressure will cause increased baroreceptor firing. So, whenever there is increased baroreceptor firing what will happen it will go to the vasomotor center, vasomotor center will be finally inhibited.

And on the other end cardiac inhibitory center will be stimulated. So, cardiac inhibitory center stimulated means there will be decrease in the heart rate. The cardiac I am doing more inhibition to the heart and decrease in the conduction velocity which is mainly seen in case of polyformic SVTs to treatment. Usually we do carotid sinus massage and there is decrease in the heart rate which is seen as a response. And whenever there is inhibition of the vasomotor center there will be decreased blood pressure or the decreased contractility or vasoconstriction or there will be vasodilatation.

So, this is seen in case of carotid sinus messaging. Now whenever there is no baroreceptor firing that means whenever already I am having decreased blood pressure. Whenever my blood pressure is low that means there is no stimulation of the baroreceptors. So, at that time there will be no stimulation or there will be no inputs to the nucleus tractors solitarius to inhibit the vasomotor center. The vasomotor center will not remain under inhibition from the caudoventrolateral medulla.

So, what will happen it will remain tonically active and when there will be tonically activity there will be increase in the sympathy activity and there will be decrease in the vagal activity. So, there will be increase in the heart rate increase in the cardiac contractility and there is increase in the blood pressure. So, this is all about how baroreceptors act and the same mechanism is seen when a person is undergone through various autonomic function test whether the person is taken from lying down to I mean supine position to standing positions or in the head up till test usually this mechanisms are seen baroreflex pathway. Now what if we do the ligation of this carotid the baroreceptors or the carotid sinus it depends on which path we are doing the ligation either we are distally ligating or whether we are approximately ligating. So, whenever we are ligating proximity that means, this is the carotid sinus and here we are ligating.

So, whenever we are doing proximal ligation that means, there is no sinus pressure there is decrease in the sinus pressure. So, whenever there is decrease in the sinus pressure that means, there is decreased baroreceptors firing the baroreceptors is not getting any inputs. So, what will happen the vasomotor center will remain tonically active and hence there will be rise in the blood pressure and rise in the heart rate. When there is a ligation distally that means, there is distal ligation and this is the carotid sinus, the carotid sinus is already full that is the sinus pressure is already increased. So, whenever the sinus pressure is increased the opposite will happen there will be stimulation of the baroreceptor firing, there will be inhibition of the vasomotor center and hence there is decrease in the blood pressure.

So, this is all about your baroreceptors pathway and various conditions like carotid sinus messaging and ligation of the carotid sinus at various levels. Now, we come to the chemoreceptors, now as I already told you the chemoreceptors the peripheral chemoreceptors usually act in response to partial pressure of oxygen, partial pressure of carbon dioxide and also hydrogen ion concentrations. But what about the central chemoreceptors, the center for central chemoreceptors is nothing but medulla. The center for chemoreceptors the central chemoreceptors that is medulla. So, today we will be talking about the central chemoreceptors.

So, chemoreceptors is mainly involved with the respiratory pathway. So, when we talk about the peripheral chemoreceptors we have to talk about the respiratory pathway, but since we are dealing with the cardiovascular mechanisms. So, here is the central chemoreceptors we will play an important role. Now, chemoreceptors will only act whenever there is a decrease in the blood pressure of less than 60 millimeter of mercury. And this usually occurs because of the hypoperfusion of the glomer cell, the glomer cell is nothing but type 1 glomer cells which are present in the that is mainly the chemoreceptors.

Now, the chemoreceptors will act directly on the vasomotor center, it will stimulate the vasomotor center. So, whenever there will be stimulation of the chemoreceptors, there will be stimulation of the vasomotor center, there will be stimulation of the cardiac inhibitory center or cardiac vagal center. Which means on mild stimulation of chemoreceptors there is bradycardia, because there is stimulation of the cardiac inhibitory center. So, obviously, the parasympathetic domain is getting activated, so obviously, there will be decrease in the heart rate. But, whenever there is more and more stimulation or severe or intense stimulation of chemoreceptors at that time a separate pathway, there is intense stimulation of the chemoreceptors, what will happen there will be increased ventilation.

Now, intense stimulation of the chemoreceptors will cause there is increased ventilation, now this increased ventilation will cause the lung stretch receptors to get activated. And this lung stretch receptors will indirectly cause inhibition of the cardiac inhibitory center or the cardiac vagal center. So, when I am inhibiting or I am inhibiting the cardiac inhibitory center or vagal center that means, I am not inhibiting the parasympathetic system or there is parasympathetic withdrawal happening, which means the heart rate will increase. So, severe stimulation of chemoreceptors will cause tachycardia, so this you have to remember. And this is actually one of the pathway, where lung stretch receptors are inhibiting the cardiac inhibitory center, which is causing increase in the heart rate.

This is actually one of the pathway which mechanism seen in sinus arrhythmias that is physiological sinus arrhythmia. So, this is the role of the chemoreceptors, the chemoreceptors also play an important role in important phenomena, whenever there is progressive loss of blood, whenever there is a progressive loss of blood there will be decrease in the blood pressure. So, whenever there is progressive loss of blood, there will be massive loss or massive decrease in the blood pressure. So, that blood pressure suppose this is the blood pressure, this is time and this is pressure, this is 60 millimeter of mercury. And suppose here this is time and this is pressure, here we have mean arterial pressure, say the mean arterial pressure value is 100 millimeter of mercury.

So, when there is a loss of blood severe loss of blood, your blood pressure is falling from 100 millimeter of mercury to 60 millimeter of mercury. So, I told you the at the point when the blood pressure will become less than 60 millimeter of mercury, the chemoreceptors will get activated. Whenever the chemoreceptors will get activated, there will be stimulation of the vasomotor center. So, direct stimulation of vasomotor center will occur and there will be rise in the blood pressure. So, this blood pressure will rise, rise, rise and it will reach to the mean arterial pressure, it might cross the mean arterial pressure also.

The moment it will reach the mean arterial pressure, the baroreceptors will try to sense the change in the mean arterial pressure. As I had already told you, it senses the change of pressure between 50 to 200 millimeter of mercury. So, the baroreceptors will cause, there is a change in the blood pressure, it will try to decrease the blood pressure by baroreceptors firing. So, again the baroreceptors firing will occur, again the blood pressure will get decreased, it will get decreased to around 60 millimeter of mercury. Again at the 60 millimeter of mercury, the chemoreceptors will again get activated and cause the blood pressure to rise.

So, this cycles will keep on repeating, so at 60 millimeter of mercury, the chemoreceptors got activated, at the level of mean arterial pressure the baroreceptors got activated. So, because of this dual activation, the sinusoidal wave which is occurring, this is nothing but the Meyer's wave or Vassomotor wave. This is typically seen in case of severe hemodynamic instability or whenever there is a progressive loss of blood. So, this we have to understand the Meyer's wave. Now, coming to the Bainbridge reflex, as I told you the Bainbridge reflex is mainly done by the low pressure receptors, the baroreceptors are the high pressure receptors.

The low pressure receptors are the other cardiopulmonary receptors, this low pressure baroreceptors are usually present at the level of right atrium, more at the level of right atrium compared to left atrium, pulmonary artery and also mainly at the veno atrial junction. And this low as I told you that the high pressure baroreceptors, they are usually sensitive to the stretch, they are mechanosensitive. The low pressure receptors, they are usually sensitive to increased venous return or increased ECF or volume of blood. So, they are usually sensitive to increased venous return or increased volume of blood. So, what happen when there is an inspiration, whenever we inspire or say whenever there is an increased venous return.

So, there is increased venous return which is occurring, this increased venous return can occur either because of the inspiration or because of infusion. Suppose, I have infused lots of fluid to a person, infusion of fluids, what will happen, this will stimulate the lower pressure baroreceptors. Lower pressure baroreceptors with the help of vagus nerve will stimulate the medulla and there will be inhibition of the cardiac inhibitory center or cardiac vagal center. Whenever there will be inhibition of the cardiac inhibitory center or cardiac vagal center, it will give rise to increase in the heart rate or tachycardia. The same is actually occur in case of inspiration, there is increased venous return, there is stimulation of the stretched receptors also that is the lung stretched receptors.

And there is inhibition of the vasodilator area, there is decrease in the vagal tone and there is tachycardia. So, Bainbridge reflex is one of the mechanism for sinus arrhythmia. The second mechanism is the lung stretched receptors, which is causing direct stimulation of the vasomotor center, sorry it is not the direct stimulation of the vasomotor center, which is causing inhibition of the cardiac inhibitory center or cardiac vagal center. And because of which there is increase in the heart rate, so this accounts around 15 percent. Bainbridge reflex accounts around 70 to 75 percent, these are the mainly reasons for the respiratory sinus arrhythmia.

So, with this the summary is, whenever there will be hypovolemia that means, whenever there is decrease in the electro cellular fluid, there is hypovolemia. Because of this

hypovolemia, there is decreased baroreceptors firing and hence because of this decreased baroreceptor firing, there will be stimulation of the vasomotor center. And hence there will be any less inhibition of the cardiac inhibitory center and that will give rise to tachycardia or increased heart rate. But, whenever there is increased ECF at that time the baroreceptors would not act at that time the main the receptors, which will act as the low pressure baroreceptors that is the Bainbridge reflex. At that time the Bainbridge reflex will act, here it is the baroreceptor, here it is the Bainbridge reflex that will act.

And this will cause with the help of Bainbridge reflex increase in the heart rate, with the help of low pressure baroreceptors. So, this you have to remember and if anybody ask you what is reverse Bainbridge reflex that is nothing but, when the baroreceptors are taking role while increasing the heart rate in case of hypovolemia. This is known as your reverse Bainbridge reflex. So, this with this we would like to conclude today's topic these are the references, which is very important for the baroreflex pathway. Thank you.