Course: Electrophysiology of Heart

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Lecture 7: Conducting system of heart

Hello everyone. So, today we will start conducting system of heart. So, in this topic we will study what are the sub topics we have. We will cover different parts of the conducting system of the heart. The AV nodal delay, explain various mechanisms of the AV nodal delay, the conduction velocity in different cardiac tissues and how the electrical impulses propagated along the conducting system of heart. Now, generally the muscles of heart consist of two types of myocardium, the normal myocardium and the modified myocardium.

The normal myocardium which is striated in nature and consist of actin and myosin filaments specifically myosin 2. This myocardium is mainly responsible for the contractile properties of the heart when the heart contracts. But when we talk about modified myocardium, this is mainly responsible for the conduction that means conducting the electrical impulse from one path to the other. So, we have basic differences between this two types of myocardial cells.

Now, normal myocardial that is atrial and ventricular myocytes they are usually singly nucleated they have one nucleus. They are connected each other with intercalated disc. Now, we had already studied in the previous lectures that each cardiac myocytes the two cardiac myocytes are usually connected electrically as well as mechanically. So, electrical connection is through the gap junctions that is electrical window. And this gap junctions are nothing but they are the electrical synapse which is made up of connexin proteins.

Also we have the mechanical connections through desmosomes and this help the cardiac cells to contract as a whole and hence this is known as the functional syncytium. Now, if we talk about the modified myocardial system which plays a very important role in conduction it has got less actin and myosin filaments because the main role is not contraction conduction. So, it should have abundance of P cells P cells or pacemaker cells which is mainly responsible for generating its own rhythm that means generating its

own beat. So, this principle is known as automicity. So, this are the two main differences between the myocardial cells.

Myocardium we have atrial and the ventricular myocardial fibers. Modified myocardium is present in the internodal pathways the nodal tissues we have bundle of his and the purkinje fibers. So, first we will talk about the SA node or the sinoatrial node. Now, sinoatrial node is the primary or the main pacemaker tissue of heart. Why it is called the pacemaker tissue of heart because it contains abundant of P cells.

Now, this P cells are present everywhere in the heart, but the proportion to which it is present it varies. So, SA node or the sinoatrial node consists of the maximum number of P cells. And SA node where it lies SA node usually lies close to the opening of the superior vena cava. It usually lies in the right atrium right side. So, right atrium at the opening of the superior vena cava.

So, this sinoatrial node has got a diameter of 0.5 centimeter and the action potentials from sinoatrial node are of slow time. Now, we also know the resting membrane potential we had studied of different muscles are different in cardiac muscles also. We have the cardiac myocytes of resting membrane potential ventricular myocytes minus 90 milli volt, but nodal tissue specifically SA node consist of RMP of minus 50 milli volt. So, the action potentials in case of SA node is slow type which is mainly because of the calcium ion influx.

And the velocity of the conduction of this impulse is also slow that is 0.05 meter per second. Now this is the sinus node or the SA node and this we have the AV node. Now, AV node is the atroventricular node this lies just above the atroventricular ring and the fiber diameter is also small in case of AV node. And the rate of impulse or the conduction is also slow that means it is slower than that of the SA node also.

Usually it ranges between 0.02 meter per second to 0.05 meter per second. So, this is the conduction speed of the AV node. Now, why the conduction at the AV node is slow when it is also a one of the nodal tissue as just like that of the SA node.

Now, if I draw the AV node simply. So, this AV node consist of various cells. So, this AV node also consisting of various P cells. The number one the first thing is in AV node the gap junctions, gap junctions are the electrical window which connect the two cells. These are comparatively less that is why the conduction is slow.

The second thing is because of the fiber diameter the nodal fiber diameter is small. Now, the smaller the diameter the resistance to the flow of anything will be high. So,

whether it is impulse or whether it is blood flow, whether it is air flow it is always high the smaller the diameter of the lumen or the diameter of the fiber. The third thing is the resting membrane potential in case of AV node is minus 60 milli volt. So, normally we see the ventricular myocytes is having resting membrane potential of minus 90 milli volt.

Now, depolarization means influx of positive ions or cations. So, the more the negative the charge it will be the more negative the membrane potential it will be. So, there will be more attraction or more rushing of the positive ions towards it. So, since minus 60 milli volt is also negative potential, but minus 90 milli volt in the ventricular muscle these that is further more negative. So, resting membrane potential of AV node is comparatively less negative to the other ventricular myocytes.

So, these are the very important features of AV node which makes the AV nodal conduction very slow. And there is a particular significance of this delay in the AV nodal conduction. And this AV nodal conduction because of this slow conduction that there is usually a delay of 100 milliseconds or 0.1 seconds. And this delay is very much significant because whenever there is a passage of impulse from SA node to AV node the impulse is passing from SA node to AV node.

So, whenever there is an impulse passage from SA node to AV node depolarization is occurring. So, depolarization is the electrical event the mechanical event that will proceed that will occur after the depolarization is the contraction. So, AV nodal delay is very much required to ensure the atria are into maximum contracted state. So, that the ventricles are fully filled otherwise there will be half filling of the ventricles and there will be less stroke volume and so the cardiac output. So, to ensure the maximum contraction of the atria and proper filling of the ventricles there is slow conduction or the there is AV nodal delay of 100 milliseconds.

Now, this again will come later. So, before that there are three nodal pathways between SA node to AV node. Initially it was thought that the impulse from SA node to AV node traverses across the atrial myocytes. But later the ultra structure has said there are three nodal pathways between SA node and the AV node. The first one is anterior internodal pathway, the middle internodal pathway and the posterior internodal pathway.

So, this is anterior internodal pathway, this is middle internodal pathway and this is posterior internodal pathway. Now, anterior internodal pathway rises from the anterior surface of the SA node to the anterior surface of the AV node. Now, it also gives another branch to the left atrium, direct branch it gives to the left atrium. So, internodal pathway, anterior internodal pathway is giving two branches. The first branch is directly to the AV node and the another branch it directly gives to the left atrium.

That branch is known as tract of back man or back man tract. Now, the middle internodal pathway also known as tract of Benke back generally supplies the inter atrial septum. And the posterior internodal pathway is the tract of thorol which usually traverses from the posterior surface of the SA node to the posterior surface of the AV node. So, this is all about the internodal pathways between the SA node and OAV node. So, the impulse traverses from SA node to AV node, this signifies nothing but atrial depolarization.

So, when the impulse is traversing from SA node to AV node, this is atrial depolarization. That means, now atria will contract, only the atria will contract because atrial myocardium and the ventricular myocardium are separated by annulus fibrosis except the AV bundle where already the depolarization is slow. So, initially the atria will contract. Now, coming to the rate of automicity, this rate of automicity is very important because SA node is the primary or the main pacemaker in our heart. But, AV node and his bundle or Purkinje fibers, these are known as latent pacemaker.

Now, why this is known as latent pacemaker and why SA node is the main pacemaker because SA node conduction is the conduction in the SA node takes place in such a way because of the presence of many number of P cells, 60 to 80 beats per minute it generates, that is the rhythm of the heart. Generally, our average heart rate is 72 beats per minute, this we know. So, this is the average heart rate. So, SA node is the sinatrial node which controls our beats and it ranges between the 62 or it sends the impulses in between 60 to 80 beats per minute. Maximum, it can go up to 100 beats per minute.

Then, coming to the AV node, it could beat AV node as well as bundle of his at the rate of 40 to 60 beats per minute, the range I am talking about 40 to 60 beats per minute. And Purkinje fibers, they beat at 15 to 40 beats per minute. Now, this is because of the number of P cells which are present in this tissues. Now, SA node usually inhibits or keeps a check on the other nodal tissues, whether it is AV node or his bundle or Purkinje fibers. So, this is known as overdrive suppression.

Why it is known as overdrive suppression and because of this overdrive suppression, SA node is considered to be the main pacemaker of our body because it inhibits the other tissues from generating its own rhythm and it only allows its own rhythm to carry forward. So, because of the overdrive suppression. But, suppose there is a heart block at the level of his bundle, whenever there is a complete heart block at the level of his bundle. So, what will happen, the impulses are not getting transferred from SA node to AV node. So, what the ventricles initially in this person, what will happen, the ventricles will take its own rhythm.

That means, the ventricles will generate its own rhythm and the rhythm of the ventricles nothing but the Purkinje fibers actually that is 15 to 40 beats per minute. So, SA node inhibition is usually removed in cases of gets removed in cases of heart block or abnormal situations. Like I told you at the level of his bundle, whenever there is a complete heart block at that time the ventricles will generate its own rhythm. So, when that rhythm which ventricle it will generate that is at the rate of 15 to 40 beats per minute that is known as idio ventricular rhythm. So, normally the question is asked what is the rate of this idio ventricular rhythm which we had already discussed that is the Purkinje fibers and 15 to 40 beats per minute is the rate of the idio ventricular rhythm.

So, this is the role of automicity. Now, already I had discussed that there is a delay of 0.1 second which occurs in the AV node due to various reasons and this is known as AV nodal delay. Now, the ability of this AV node to slow down the electrical impulses which are coming from the SA node to the ventricle is known as decremental conduction. Now, this decremental conduction is very important because suppose in case of abnormal situations suppose atrial fibrillation any sort of arrhythmia related to atria where there is atrial fibrillation or atrial flutter. The atria suppose it is beating at the rate of very fast heart rate suppose 300 beats per minute.

Now, if 300 beats per minute is the net heart rate of a person you can imagine that person will immediately go into heart failure there will be certain cardiac arrest. So, what it does our AV node is the gatekeeper at this time. It will slow down the electrical impulses it will slow down the heart rate whichever it is getting from the atria that is from 300 to approximately 100 to 150 beats per minute. And the person will generate its heart rate as 100 to 150 beats per minute. So, decremental conduction is very important in slowing down the transmission of very rapid impulses from SA node to ventricles.

Next, come to coming to the Hiss bundle. So, Hiss bundle what happens Hiss bundle is also a connective tissue and this septum rises from the AV node and terminates at the level of the Purkinje fiber. So, the pecs of the ventricle. So, Hiss bundle on entering the interventricular septum divides into usually right bundle branch and left bundle branch. Now, right bundle branch is longer and thinner because right ventricle is comparatively the thickness is less compared to the left ventricle. This is right ventricle and this is left ventricle whereas, the left bundle branch this usually gives rise to two branches.

One is anterior fascicle the other one is posterior fascicle. It gives an anterior branch and the posterior branch. Interior branch now right bundle branch as I told you it usually supplies the right ventricle whereas, left bundle branch it usually supplies the anterior fascicle supplies the anterior part of the left ventricle and the posterolateral portion of the

left ventricle is supplied by the posterior branch of the left bundle branch. Now, this left bundle branch while supplying to the ventricular walls gives rise to various fibers. You can see over here these are giving rises to this giving rises to various fibers.

These fibers are nothing but, the Purkinje fibers. These are very fast fibers and this Purkinje fibers these branches are given off at the sub endocardial layers of the myocardium. Sub endocardial means this is the wherever it is giving the branches this is the inner side of the myocardium and here we have the apex of the ventricle. So, this is the outer side of the myocardium. So, towards the inner side of the myocardium the Purkinje cells or this fast conducting fibers are given branches.

So, next we come to the Purkinje fibers. So, Purkinje cells are the largest cells in the heart. The Purkinje cells are usually discovered by the scientist Johan Purkinje and this person has discovered Purkinje fibers in the heart as well as Purkinje cells in the cerebellum. So, this is a network of fast conducting fibers that is present in the sub endocardial regions of the right and the left ventricle. Numerous gap junctions are present this is one of the reasons for the fast conduction of the Purkinje fibers. Now, why the conduction is fast? The first point is gap junctions are present in large amount.

The second point is the fast action type of potential, fast action potential because the ventricular muscle has got a resting membrane potential of minus 90 milli volt. So, this action potential will be fast which is usually carried out by the sodium influx. And the third point is the fiber, the diameter of the Purkinje fibers that is large what we had seen in the AV node it was small. So, since the diameter of the Purkinje fibers are large. So, there will be less resistance to the flow and so the rate of impulse conduction is quite high in the Purkinje fibers the rate is usually 4 meter per second.

And the action potentials which are generated over here is of fast type. So, we have seen various conducting fibers rate 4 meter per second is very important the maximum speed of conducting fibers that is the Purkinje fibers in the heart. And very slow minimum we have in the AV node that is 0.

02 to 0.05 to be very specific 0.02 meter per second. So, this is the conduction velocity S naught 0.05 meter per second internal pathways 1 AV node this you have to remember 0.02 to 0.05 Purkinje fibers 4 and other 1 meter per second. So, even if you do not remember all the maximum and the minimum is very important that is Purkinje fibers consist of the maximum conduction velocity while AV node consist of the minimum conduction velocity.

Now, coming to the conducting system of heart how the electric impulse gets conducted

from S a node to the ventricles. That means, we will see we have seen the electrical events that is the how action potential is generated there is a depolarization phase there is a repolarization phase. We will see that how it occurs in the conducting system how that depolarization wave or repolarization wave is conducted throughout the heart. Now, the impulse generation has already told that is sinus node. So, sinus node has generated its impulse and the impulse direction is towards AV node.

Now, since sinus node is situated on the right atrium. So, the impulse will be directed towards left side downward. So, the AV node is also downward. So, sinus node the impulse coming from the sinus node or sinuetrial node is left side downwards. So, this is the impulse this is nothing but the atrial depolarization. And as I told you that this AV node is very slow conducting pathway.

And this AV node will have a delay of 100 milliseconds or 0.1 seconds delay. And the conduction velocity at this AV node is 0.02 meter per second which is very slow to ensure full contraction of the muscles and proper filling of the ventricles. So, this AV nodal delay has been told. Now, there is a range given in the Harrison of adenodal delay that is 60 to 120 milliseconds of the AV nodal delay.

If a specific answer is not asked of 100 milliseconds or 0.1 milliseconds the range is sometimes given or asked that is 60 to 120 milliseconds. So, after this AV node Purkinje fibers has got maximum conduction velocity. Now, after this AV node purkinje the passage of the impulse to the AV node or the AV bundle then it will pass to the his or the bundle of his. So, this bundle of his and this level of bundle of his the time it is taken at the bundle of his is 40 milliseconds. Now, before that I would tell what is the time taken for the impulse to travel from SA node to the tip of AV node that is 30 milliseconds.

So, 30 milliseconds SA node to AV node then 100 milliseconds at the level of AV node which has got an AV nodal delay then we have 40 milliseconds at the level of his bundle. So, these are the time taken if we start with 0 from here. So, generally at the level of his bundle it has taken around 130, 40 and 30 around 170 milliseconds. That means, the depolarization will start at from the level of 170 milliseconds. So, from 170 milliseconds till the apex of the ventricles the depolarization will reach at 200 milliseconds which means now the depolarization occurs in various phases.

The main three phases of depolarization which takes place the first one is septal depolarization. The initially there will be septal depolarization that means, from left bundle branch towards the right bundle branch. So, from the left side to right side, there will be septal depolarization. The next depolarization that is the whole ventricular musculature whole ventricular depolarization. And the last portion of the heart to get

depolarized that is the basal portion of the heart.

The basal portion of the heart gets lastly depolarized. So, this three steps of depolarizations occurs one by one. So, after septal depolarization or the onset of septal depolarization start at the level of 170 milliseconds. That means, when it reaches the his bundle. And at the apex that means, the ventricular depolarization takes place at the level of 200 milliseconds.

Till now the whole ventricles have not got complete depolarization. It will only get completed when the basal portions of the heart also gets depolarized. So, the basal portion of the heart takes another 20 milliseconds. And hence the whole ventricle depolarization occurs at 220 milliseconds. This time intervals are very important because these are usually asked. So, the whole depolarization takes at 220 milliseconds, which means the Purkinje fibers takes only 20 to 20 minus 200 if we do 20 milliseconds to reach from this ventricular wall to the base of the heart.

So, which means you can understand what could be the speed how fast the conducting fibers are the Purkinje fibers are. So, the apex of the ventricles 200 milliseconds. So, 200 milliseconds apex of the ventricles and the whole ventricular depolarization takes place at 220 milliseconds. The rest part is the repolarizations. We already know the action potential it ranges between 250 to 300 milliseconds in case of cardiac action potential.

So, this is all about the conducting system of the heart, the seconds the time intervals which are also important. Now, the slower conductor conduction velocity in AV node is already I had told you because of the small size and the slow up stroke of the action potential which occurs due to the slow voltage calcium channels. That means the slow action potential because of the resting membrane potential which is of minus 50 to minus 60 milli volt and weak electrical coupling because of the present of relatively few gap junctions. The importance of AV nodal delay allows proper ventricular filling. So, that to ensure proper stroke volume and maximum cardiac output it maintains low ventricular rate that is the decremental conduction to prevent higher impulses higher rate impulses travelling from atria to ventricles.

Also there are effects of drugs which reduce the heart rate partly by acting at the level of AV node by delaying the or promoting the AV nodal delay. That means increasing the AV nodal delay. So, these are the few examples digitalis and digitalis or digoxin or beta blockers. Now, digitalis and digoxin mechanism of actions are already been discussed that is it is a blocker of the sodium potassium ATPS pump.

And beta blockers they act on the beta 1 receptors present in the heart. So, by blocking

the beta 1 receptors they will cause decrease in the cyclic AMP and hence in decrease in the calcium influx. So, these are the effects of the drugs also vagal stimulation. Now, this AV nodal delay is subjective to change. Now, whenever there is a sympathetic stimulation suppose there is an epinephrine secretions or fear anxieties whenever there is a sympathetic stimulation this AV nodal delay with will get shortened. Whenever there is a parasympathetic stimulation that means vagal stimulation because of the acetylcholine.

So, release of the acetylcholine. So, this AV nodal delay will get prolonged. So, this causes AV nodal block in subsequent constant vagal stimulation. So, coming to the conduction the direction of the conduction in the heart the first as I already told you the SA node to AV node it is directed which from upper from up towards the lower side. That means, the direction is towards the lower side laterals lower and left side and this usually contributes for the atrial depolarization. At the level of AV node there is AV nodal delay of 100 milliseconds. Then moving to the his bundle then at the level of the his bundle it divides into right bundle branch and left bundle branch at the bundle at the interventricular septum there will be septal depolarization first.

So, septal depolarization first. Now, the septal depolarization is from left to right side because it has been said in some books that the septal the ventricular septum which is present and here the left bundle branch there is a connection with the left bundle branch. So, the left bundle branch is connected towards the it has got some connections with the septum and so the depolarization takes place from left to right. After the septal depolarization there will be depolarization of the ventricular muscle from this endocardium to the epicardium. Now, endocardium to the epicardium means from the inner side of the myocardium towards the outer side from inner towards the outer side. Because, the purkinje cells which are present over here these are the maximum conducting fibers and this conducting fibers are present at the level of sub endocardial surface.

Now, sub endocardial surface means it is towards the inner myocardium and so the depolarization wave will pass from the inner myocardium to the outer myocardium. So, inner myocardium towards the outer myocardium if this is the apex and so this is the inner myocardium and this is the outer myocardium or epicardium. So, the depolarization will be from inner side towards the outer side that means from endocardium to the epicardium and the last portion to gets depolarized that is the basal portion of the heart. So, posterobasal portion of the hearts are the last portion to get depolarized. So, this the start is the septum that also which part of the septum middle part of the septum left side is left and the portion is middle.

So, left middle portion of the septum will get depolarized first. Direction is left to right and the last portion of the heart to get depolarized that is the base of the heart that is left posterobasal region of the heart and the depolarization ventricular depolarization is from endocardium to epicardium. The opposite occurs in case of repolarization. Remember the last side to get depolarized is the first side to get repolarized. Why it is so? Because whenever there is a contraction depolarization means the contraction will occur the muscles contraction will occur.

So, whenever ventricles will contract to its maximum. So, what will happen the inner portion of the blood vessels will get constricted there will be restriction to the flow of the blood, but the outer blood vessels the outer myocardial blood vessels will remain sort of unaffected. So, the repolarization starts at the epicardial layer or the outer myocardium. So, the repolarization of the apex starts from the epicardium to endocardium. So, that is why this is the repolarization which starts from the epicardium to endocardium.

So, this we have to remember the action potentials from SNO are of slow type. And in case of portinger fibers fast type there is a AV nodal delay of 0.1 second which is very important for the ventricular proper ventricular filling ensuring proper cardiac output. Depolarization occurs always from inner to outward that is endocardium to epicardium while repolarization occurs from outer to inner that is from epicardium to endocardium. So, these are the concluding topics and these are the references. So, we conclude today's lecture. Thank you.