Course: Electrophysiology of Heart

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Lecture 11: Abnormal ECG-1

Hello everyone. So, today we will start our next topic that is abnormal electrocardiography that is abnormal ECG. Till now so far we have discussed all about the normal ECGs, the calibrations, the technical aspects and the interpretations of normal ECGs. So, based on that today we will see what are the abnormal rhythms, cardiac rhythms. So, first we will start with the cardiac arrhythmia, what are the types of cardiac arrhythmia, what do we understand by bradycardia or bradyarrhythmia, different causes, different types and finally, case discussion. Now, whichever arrhythmias we are discussing, whichever abnormalities we are discussing, we will discuss only the pathophysiological basis, the management of this diseases, cardiac arrhythmias or any abnormalities that comes beyond the scope of this course.

So, what do you understand by cardiac arrhythmias? Now, any abnormality of the cardiac rhythm is known as cardiac arrhythmia. Now, we have the normal sinus rhythm. So, if normal sinus rhythm as per what we know, it should have a particular heart rate, it should beat within 60 to 100 beats per minute, it should have a certain conduction velocity and pathway, conduction pathway. So, beyond this if anything happens that is cardiac arrhythmias.

Now, this cardiac arrhythmias suddenly present with palpitations, a feeling of anxiety, syncope, hypotensions and sometimes it could present with no symptoms at all. So, the normal sinus rhythm what we understand by normal so far that is the origin of the impulse should be from the primary pacemaker that is acinode. Here acinode is the primary pacemaker we have already discussed. Then, we have a normal conduction pathway, normal conduction pathway means the impulse should travel from acinode to AV node, then there should be a conduction delay in the AV node, then there it should pass to the bundle of his, then the branches, then purkinje fibers, then the ventricles. Now, this is the normal conduction pathway, so the impulse should travel through this normal conduction pathway.

Then it should have a normal velocity like for example, the AV nodal delay is of certain significance it is of particular importance, we know why it is very important. So, it should AV node should have a slow conduction and purkinje fibers on the other hand they have a faster conduction. So, you can understand if the reverse happens by any means, so that becomes a pathological condition. So, again the impulse should travel through normal velocity and the rate, the rate at which the impulse is been generated. So, the 60 to 100 beats per minute is usually the normal rate.

So, this if goes below 60 it is bradycardia or bradyarrhythmia, if this goes above 100 we call it as tachycardia or tachyarrhythmia. Now, we have different causes for bradyarrhythmias and tachyarrhythmias, but what is actually I am meaning to say is this these are the normal criteria's to say that yes this is a normal sinus rhythm. If any deviation occurs out of any of this four, we usually consider that as cardiac arrhythmias. That means, if the generation of the impulse is not from SA node rather it is from some other ectopic focus. If the conduction pathway is not the normal conduction pathway, there is an any accessory conduction pathway.

If the velocity as I told it gets interchanged, if the velocity differs in the cells and if the rate is different it does not fall within the normal range that is 60 to 100 beats. So, we call all this at cardiac arrhythmias. Now, what are the types of cardiac arrhythmias? As I told you bradyarrhythmias means the beating should be less than 60 beats per minute. Tachyarrhythmias means it should be 100 beats per minute. In general bradycardia there is sinus bradycardia, there is sinoatrial block and there is AV block which we generally in layman term we call it as heart block.

So, there is AV nodal block or AV atroventricular block or heart block. So, there are different types of blocks are present we will discuss that. Then we come if we come to the tachyarrhythmias, we have supraventricular tachycardia, ventricular tachycardia, flutters and fibrillation. Now, supraventricular tachycardia means any tachycardia which is originating above the level of ventricles, it can be AV nod, it can be atria. So, this is supraventricular tachycardia, then we have the ventricular tachycardia, then we have flutter both atrial as well as ventricular, then fibrillations both atrial as well as ventricular.

So, the rates are different in each of this. So, today we will discuss about the bradyarrhythmias. Now, sinus arrhythmia what do we what do we understand by sinus arrhythmia? Sinus arrhythmia remember it is a normal physiological phenomenon, it is not pathological. Sinus arrhythmia means there is a difference in the rhythm rate and rhythms of our heartbeat along with our respiratory cycles, which means on inspiration our heart rate increases, on expiration our heart rate decreases. This is not abnormal, this

is absolutely normal and physiological.

Why? Because in the last class we have discussed that while inspiration while we inspire there is increase in the venous return as well as the stimulation of the lung stretch receptors. There is increased in the venous return as well as stimulation of the lung stretch receptors. Now, this increase in the venous return will cause stimulation of the volume receptors that is low pressure baroreceptors, which are mainly present at the level of atrium. So, this will get stimulated and this will cause activation of the medulla via vagus nerve or tenth cranial nerve. Here this will further inhibit the parasympathetic fibers.

Now, vagal activity is inhibited means our heart rate is increased, the sympathetic activity is increased. So, parasympathetic fibers are inhibited from the medulla and also the lung stretch receptors which were stimulated they directly inhibit the cardiac vagal center or cardiac inhibitory center. This is also present at the level of medulla. So, when cardiac inhibitory center or cardiac vagal center is inhibited again the parasympathetic activity or the vagal activity is decreased and so the heart rate will increase. So, this happens in case of inspiration which means the opposite will occur in case of expiration.

So, whenever there will be expiration suppose there is an expiration the reverse things will occur. The lung stretch receptors would not get stimulated, there would not be increased venous return, there will be decreased venous return. So, the low pressure receptors would not get stimulated and finally, there will be the parasympathetic activity which will remain unaltered. So, heart rate will decrease. So, expiration causes decrease in the heart rate, inspiration causes increase in the heart rate.

So, that is what we see in this ECG. We can see this and this if you just consider this two RR intervals and if you just consider this and this RR interval. We can see here this RR interval is prolonged that means the heart rate here is decreased that means this is an expiratory phase, here expiration is going on. Here we can see the RR interval is shortened that means here the heart rate is increased that means inspiration is going on. So, this is a normal physiological phenomenon which we see in the ECG that means on inspiration the heart rate increases, on expiration the heart rate is decreased.

Now coming to the sinus bradycardia. Sinus bradycardia is the decrease in the heart rate which is less than 60 beats per minute. It can occur physiologically, it can occur pathologically also. So, first we will see what are the physiological causes of sinus bradycardia. Now a physiological variation occurs mainly in case of sleep, then in case of athletes who are trained persons athletes they are usually having strong vagal tone.

So, this strong vagal tone causes their heart rate to be decreased below 60 beats per minute. So, the heart rate can go as low as 50 to 40 beats per minute. Now this is because of mainly the strong vagal tone. This is physiological. Now pathological variations of the causes of bradycardia is mainly hypothermia, hypothyroidism, increased intracranial pressure or there can be action of some antiarrhythmic drugs.

Antiarrhythmic drugs means the drugs which causes decrease in the heart rate like beta blockers, calcium channel blockers, digitalis, then sodium channel blockers etc. So, here we can see if we are asked to calculate the heart rate, we take any RR interval and we count the number of boxes. So 1, 2, 3, 4, 5, 6. Six boxes are there. So 1500 divided by 6 into 5, 30 small squares.

This is the way which we usually calculate our heart rate for a regular rhythm. So this is 50 beats per minute. So what we observe over here is heart rate is slow, heart rate is decreased, but it is regular. It is not irregular. So this is the condition which we see in case of sinus bradycardia.

So this is 50 beats per minute heart rate. Now what we see over here, this is the normal sinus rhythm where the beats is usually ranging between 60 to 100 beats per minute. If we calculate it is 1, 2, 3, 3 and more or less 4 if we take. So 1500 divided by 20. So it will give around 75 beats per minute which is normal.

Bradycardia here we can see the beats is less than 60 beats per minute. This is tachycardia, the beats is more than 100 beats per minute. If we take this 2 RR interval, then we can see this 2 that means 10, then approximately 15. So 1500 divided by 15 is 100. So it will be around 100 or 105 beats per minute.

Then there is a syndrome where bradycardia is ongoing. Along with that suddenly we get tachycardia. So that syndrome is known as tachybrady syndrome. Here we can see the RR interval is prolonged. So RR interval is prolonged or lengthened that means here bradycardia is going on.

But suddenly we see there is shortening of the RR interval. So shortening of the RR interval means heart rate is increased. So at one point the same ECG we are getting bradycardia as well as tachycardia. This is known as tachycardia syndrome or sick sinus syndrome. There is a abnormality in the sinus node that is why it is also known as sick sinus syndrome.

Any person having the sick sinus syndrome usually they should undergo pacing. So chronic symptomatic sick sinus syndrome requires permanent pacing along with the use

of the anti-arrhythmic drugs. Now next we come to this junctional rhythm. Junctional rhythm usually occurs in two conditions. The first one is whenever there is sinus arrest.

The second one is whenever there is complete heart block. Now sinus arrest means there is a dysfunction or abnormality in the sinus node or sinus impulse transmissions or SA node. So when the impulse is not getting transmitted properly from the SA node to the AV node after some time the AV node usually generates its own rhythm which means the AV node usually escapes the overdrive suppression. This term which I have already used whenever the SA node keeps a check on the other tissues it means it is suppressing the other tissues to generate its own rhythm. So SA node usually generates its own rhythm because of its fastest automaticity but whenever this rhythm would not be generated by the SA node and the other nodal tissues will escape this overdrive suppressions they will beat on its own rhythm and this is known as escape rhythm or junctional rhythm.

When this happens at the level of AV node or AV nodal junction it is known as AV junctional rhythm. Now this beating will be definitely regular and when it is beating at the level of AV node so it will be around 40 to 60 beats per minute. Now the second condition where junctional rhythm is occurring that is complete heart block. So suppose there is a block between anywhere between the AV node and the bundle of his that means there is no conduction of impulse from the AV node to the bundle of his and so the distal cells which will lie distally to the bundle of his the cells lying distal to the bundle of his they will generate its own rhythm and when they will generate its own rhythm the ventricular rate or the rate that will also be called as a junctional rhythm or the escape rhythm and that will also beat at the rate of around 40 beats per minute. And when the rhythm is generated distal to the bundle of his at the level of ventricles we would not get usually P waves because the origin is at the level of beyond the bundle of his that means at the level of ventricles so usually we do not get P waves.

So that is what we see in the ECG this is a junctional rhythm which we are getting and you see we are not getting any P waves over here. The beats is 46 beats per minute and we are not getting any P wave but sometimes we can get the retrograde P waves also here we are getting retrograde P waves also. So regular ventricular rate at the level of 40 to 60 beats per minute and P waves are usually absent and we can get inverted at also we can get P waves after the QRS complex is based on the timing of the depolarization of the atria and the ventricles. So generally junctional rhythm we get with hidden or no P waves.

The next we have the sinoatrial block. Sinoatrial block means any block which is causing the delay or no conduction of impulse from SA node to AV node that means

atrial depolarization is not occurring properly there is delay or there is no conduction or there is hindrance in the conduction of the impulses from sinoatrial node to atrioventricular node. So there is various types of sinoatrial block. We have first degree sinoatrial block. We have second degree sinoatrial block which is again divided into one and two types. Then we have the third degree sinoatrial block.

Now first degree sinoatrial block is very difficult to identify on the surface conventional ECG. Why? There is a delay in the conduction from SA node and difficult to detect in the conventional ECG because it appears usually normal. Now suppose this is the P wave generally we will be looking at the P wave. So what happens here in the sinoatrial block degree one suppose this is the level where SA node is getting activated or discharging the impulse. And this is the level where atrial depolarization is occurring.

Usually this does not happen. Here atrial depolarization is occurring that means here only the SA node is generating the impulse and so the atrial depolarization is occurring. So it is very difficult on the ECG to detect this prolongation or this delay between the sinus node discharge and the depolarization of the atria. So here SA block first degree is delay in the conduction from SA node. Now we have two intra-cardiac studies are to be done for that. Now SA node second degree in the second degree there is a delay in the conduction from SA node throughout the internodal pathways.

So there will be gradual increase in the delay till one impulse is blocked. Till one impulse is blocked means we can see over here in the diagram this is the sinus impulses this is the second degree usually. In the second degree there is a blockage of the impulses sporadically. There can be blockage of one impulse, there can be blockage of two impulse, there can be blockage of three impulses. So in the second degree type one which is also known as Wienkebach phenomenon.

So second degree type one there is delay in the conduction from SA node through the internodal pathways and this delay is usually gradual which means the P wave atrial depolarization which is occurring in suppose 0.2 seconds. In the next rhythm it will occur in around 0.4 seconds, in the further rhythm it will occur in around 0.

6 seconds. In this way finally at one point it will come when you will not get any rhythm. So there is a gradual increase in the delay in the conduction from SA node to Avino till one impulse is blocked. So when one impulse is blocked which means we will not get the P wave as well as the QRS complex over here. In the second degree block as I told you the type two of the second degree block we usually get the sporadic blockage of the impulses. There can be one blockage, there can be two blockage, there can be three blockage.

Here we can see this is the P wave, this is the P wave, this is the P wave but here we do not see any P wave or any rhythm or QRS complexes. So again we see P wave, here is a fusion, here again we see P wave, again we see P wave but here again we have lost the impulse. So this is the second degree block, the blockages, the degree and the nomenclature occurs based on the number of P waves which you are getting and number of blocks you are getting. And finally the third degree SA block there is a complete blockage of the impulses from the SA node to Avino node. At that time there has to be presence of escape rhythm which is usually at the rate of 40 to 60 beats per minute.

So this is all about your sinoatrial block. Now here we can see this is the second degree again, this is the P wave, this is the P wave but here we get usually we are missing two impulses. We are not getting any impulses, again we get a P wave, again we get a P wave. So this is also the second degree sinoatrial block. Now AV block, AV block is nothing but atrioventricular block. Now atrioventricular block means there is delay at the level of AV node, AV nodal conduction is getting delayed which means impulses not getting conducted or delay or not getting conducted from AV node to bundle of his.

Now in the ECGAP could remember where do we see this AV nodal conduction or the bundle of his conduction. This we usually see at the level of PR interval. So this is P, this is R. So this PR interval usually tells about our AV nodal conduction and the bundle of his conduction which means any blockage or any delay at the level of atrioventricular node will cause prolongation of the PR interval. This PR interval will get prolonged and of course the QRS complexes, the depolarizations will get affected.

So there will be abnormal morphology of the QRS complexes. So QRS complexes abnormal morphology you can get. So AV block what we will see the first degree AV block, second degree AV block and third degree usually the blocks nomenclature is same as that of the sinoatrial block. In the first degree AV block the PR prolongation is constant the same which we had seen in case of the first degree sinoatrial block which is difficult to trace in the ECG. But this is not usually difficult to stress in the ECG in AV block because usually the PR prolongation is regular.

In every bit you will get PR prolongation. The PR interval is prolonged in every bit. In case of second degree hard block or AV block there are various types that is Mobitz 1, Mobitz 2 and 2 is to 1 block. Now usually the impulses are getting conducted but since there is a delay or since there is a hindrance to the conduction so the impulse does not get conducted to the from the AV node to the bundle of hints and hence the ventricular depolarization fails to occur. So you would not be getting QRS complex over here and that is nothing but the dropped beats. So P waves will be present but you would not be

getting QRS complexes.

Then further it is divided into Mobitz 1, Mobitz 2, 2 is to 1 based on the number of P waves and the QRS complexes you were getting. For example you were getting 2 P waves and 1 QRS complex it is 2 is to 1. If you are getting 3 P waves and 2 QRS complex it is 3 is to 2. So Mobitz 1 is usually the same as that of the sinoatrial block Wenke back phenomenon but usually we do not use this Wenke back phenomenon name in case of Mobitz 1 type of hard block since we are using it in the sinoatrial block. And third degree there is total P wave and QRS dissociation or atrioventricular dissociation in other terms.

So this is we will see the first degree AV block. This is a normal conducting pathway. This is the AV node. This is the his bundle of his. Then right bundle and left bundle.

This is supplying the right ventricle. This is supplying we have the posterior fascicle. We have the interior fascicle and with the Purkinje fibers they are supplying the left ventricle. So this is the normal conducting pathway. Now in the first degree AV block if suppose any block is present there will be delay in the conductions. It is not that the impulses would not pass but the delay of AV nodal which is usually 0.

1 second it will get increased. So that will cause prolongation of the PR interval. So here we see the first degree AV block. Generally if I ask you what is the normal PR interval we usually have 5 boxes.

5 boxes means 5 into 0.04 that means 0.2 seconds. 0.2 to 0.22 seconds should be the normal PR interval. But when it is getting more than that that means the PR interval is prolonged and that is what we are seeing. This is the P wave. This is the R wave. We can see in every cases the P wave and R wave is present regularly and the PR interval is prolonged also at regular intervals.

So this is the first degree AV block. It is quite easy to determine in the ECG. The second degree AV block is the Mobitz 1 block where all the P waves are not conducted. All the sinus impulses are not conducted to the ventricles.

And so what happens we lose the QRS complexes. We only have the P wave. So it is also we have type 1 and type 2. In type 1 there is a gradual prolongation of the delay of the PR interval.

Here this is the P wave. This is the P wave. This is the P wave. And we can see here we have the P wave but quite prolonged PR interval. So this is the PR interval.

This is the PR interval. This is the PR interval. And this is the PR interval. We can see there is a gradual prolongation of PR interval with starting from suppose 1 and half boxes we are getting this as 2 boxes. We are getting this as 3 boxes. And this we are almost getting as 3.5 boxes.

So there is a gradual and finally we will lose the QRS complex in the next bit. So that means there is exhaustion of the conduction capacity. The AV node exhaustion is so much that for the next conduction of the next impulse the conduction fails and we do not get the QRS complex. So there is progressive PR prolongation and finally disappearance of the impulse that is known as dropped bit. Now this is second degree atroventricular block Mobitz 1.

This is nothing but what we had shown in the ECG. This is the PR. This is the PR. This is the PR interval. This is the PR interval. And finally no QRS complex is present over here.

Only we could see the P wave. So this is Mobitz type 1 block. Now Mobitz type 2. Mobitz type 2 impulses are blocked sporadically. There can be 1 impulse blocked. There can be 2 impulses blocked. There can be 3 impulses blocked.

Now very importantly Mobitz type 1 block is usually seen at the level of AV node. Mobitz type 2 block is usually seen at the level of bundle of his or bundle branches. So 80% of the block is taking place in bundle branches or 20% of the bundle of his. So in Mobitz type 2 the PR interval will remain constant. We can see the PR interval will remain constant but there will be loss of QRS complexes.

So here we can see this is the PR interval. This is the PR interval. This is the PR interval. This is the PR interval. So the PR interval is not getting prolonged but we are having many dropped beats at a random fashion. So here we have the P wave but no QRS complexes.

Here we have the P wave but no QRS complexes. So this is the second degree AV block 2 is to 1 pattern. When 2 is to 1 pattern means we have 2 P wave and 1 QRS complexes. As I told you this will depend on the number of P waves and the QRS we are getting. So here we have the P wave and we are getting 1 QRS complexes.

So 2 P waves we are getting and 1 QRS we are getting. So this is the 2 is to 1 block. So this is second degree AV block. Now third degree AV block. Third degree AV block we see no relation of the P waves and the QRS complexes. The atria and the ventricles will

beat on its own rhythm. There will be electrical dissociation between atria and the ventricles and this is known as AV dissociation.

The escape rhythm occurs depending on the site from which the impulse is generating. So we can see the constant P-P intervals. This is the P wave. So we can see number of P waves which are at constant spacing or constant interval. Also we can see the R waves which are also at constant spacing. That means P-P interval is there, RR interval is there but PR interval as such there would not be present.

So there is a different atrial rate and there is a different ventricular rate we get over here. So here we get atrial rate different, here we get ventricular rate different. So this is third degree AV block. Now coming to the bundle branch block. Bundle branch block means the blockage is occurring at the level of the bundle branches. We know the conduction in the bundle branches and Purkinje fibers, they usually are depicted as the QRS complexes in the ECG which means any abnormality in the depolarization of the ventricles because of the abnormality in the conduction will result in wide QRS complexes.

The QRS complexes will be more than 0.12 seconds. And of course the morphology of the QRS complexes will get changed. There will be Bisel morphology of the QRS complexes. So this is what we can see and the bundle branch block usually is detected at the horizontal plane. That means how the precordial leads or the chest leads are presented.

So the best chest leads to determine this bundle branch block is V 1 and V 6. We can also use V 2 and V 5. So V 1, V 2, V 5, V 6, this usually determine the, which predict the bundle branch block easily. These leads are important we have to remember. Now we will see what happens in case of left bundle branch block. So in case of left bundle branch block, first we have to remember whether it is right bundle branch block or left bundle branch block.

The bundle branch which is getting blocked and which is supplying the other, the respective ventricle, they would not get depolarized easily. So there will be abnormal depolarization going on there. So the unaffected ventricle will get depolarized fast. Then that will cause the further depolarization of the affected ventricles.

Here the ventricles, right ventricle and the left ventricle, they are not depolarizing at the same time. So we will get the left, whichever ventricles is affected, they will depolarize later. First the unaffected ventricle will get depolarized. So what we see in case of left bundle branch block, SN out to atria is okay normal, atria to AV node conduction is

normal, AV node to bundle of his conduction is normal. Now there is a block in the left bundle branch. So there will be conduction through the right bundle branch and there will be depolarization of the right ventricle and then there will be depolarization of the left ventricle.

The right ventricle will depolarize first in case of left bundle branch block and then the left ventricle will depolarize. And this will cause the difference in the electrical vectors, the current vector. So here we can see, this is the V 1 and this is the V 6, V 2, V 5 also you can take. So in V 1 and V 6 what we can see, these are the vectors we are getting. Initially this is the septal vector, in the septal vector what we are getting, this is the positive deflections and in case here this we are getting the negative deflections.

And then this is the ventricular depolarization of the right ventricle and this is the ventricular depolarization of the left ventricle. So in case of left bundle branch block, so right ventricular depolarization away from the, this is the negative deflections and then left ventricular depolarization, this is the positive deflection. In case of V 6, this is what, there will be actually if we see this V 5 it will be better because this is the deflection of the right ventricle and this wave is the deflection of the left ventricle. So we actually get this rabbit ear or bunny ear appearance in case of V 5 or V 6, here also it is not present because it is merged, but usually we get the rabbit ear or the bunny ear appearance in case of left bundle branch block.

So this and obviously the QRS duration is more than 0.12 seconds. Similarly we see in case of right bundle branch block. In right bundle branch block what we see, this is the right, this is the left, this block is there. So there will be no conduction of the impulse to the right bundle branch. So left ventricle will depolarize initially and then the depolarization will pass on to right ventricle.

So that is what is happening. The septal depolarization is occurring first. This is the septal vector. Then we have the left ventricle depolarization. Then we have the right ventricular depolarization. And based on that we have this V 5 or V 6 analysis, vectorial analysis and this is the V 1 analysis. So this is the wave is mainly small r. This is mainly because of the depolarization of the left ventricle and then big R that is mainly because of the depolarization of the right ventricle.

So in this way we have seen the various bundle branch block and the dissociations in the current. So this is the case summary. A 70 year old man came to emergency with slow pulse. Generally all the arrhythmic patients will come with palpitations or slow pulse, syncope, hypotensions, attacks of dizziness, etc.

So this is his ECG. What we can see over here. Now this ECG if we just see the rhythm strip we can analyze. This is a rhythm strip of complete heart block or AV dissociation. We can see this is a P wave, this is the P wave, this is the P wave, this is the P wave and this is the R wave. So again we can see this is a P wave, this is a P wave, this is a P wave, this is a P wave and this is the R wave. Same P wave, P wave, P wave and this is the R wave.

So this ECG is of complete heart block or third degree AV block where we can see there is an AV dissociation. The atrial rate is different and the ventricular rate is different. If we just calculate the atrial rate it will be different. If we calculate the ventricular rate it will be different.

If we just 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13. If we 13 into 5, 15. The ventricular rate will be around 26 or 27 beats per minute and atrial rate will be obviously it will be around 1500 divided by 13 small boxes I can see. So it will be around 130 beats per minute. So the atrial rate and the ventricular rates are different.

So this is the AV dissociation, this is an ECG of complete heart block. So these are the references of today's topic. With this I conclude today's lecture. Thank you.