Course: Electrophysiology of Heart

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Lecture 12: Abnormal ECG-2

Hello everyone. So, today we will move on to the next part of our abnormal ECG. So, till now we have learnt the various types of bradyarrhythmias. So, today we will learn the mechanisms and various types of techie arrhythmias. So, what do you understand by techie arrhythmia? Techie arrhythmia means techiecardia where the rate is more than 100 beats per minute. So, usually there are various types of techie arrhythmia based on the rates, based on the anatomical location from where this arrhythmia is occurring.

So, generally we divided into two broad classification. The first one is supraventricular techie arrhythmia and the second one is ventricular techie arrhythmia. Now, supraventricular techie arrhythmia as per the name suggests, this means above the level of ventricles. Supraventricular means above the levels of ventricles.

That means this techie arrhythmia can arise at the level of atria, at the level of AB node, anywhere this techie arrhythmia can occur. So, ventricular arrhythmia means this techie arrhythmia will occur at the level of ventricles. So, what are the mechanisms of this techie arrhythmias? Generally, there are three important mechanisms we come across. The first one is increased or abnormal automaticity. So, what do you understand by increased or abnormal automaticity? Now, autorhythmicity is the primary feature of the primary pacemaker that is SA node, because SA node has got the fastest automaticity.

So, this is the primary pacemaker. So, the origin of impulse should always be from the SA node. If besides this SA node, anywhere else any cells other than the SA node is causing the generation of the impulse, then that is increased automaticity or abnormal automaticity. Suppose AB node is generating the impulse, suppose the bundle of his is generating the impulse, suppose atria cells, atrial cells are generating the impulse, the ventricular cells are generating the impulse, any cells rather than the SA node who are generating the impulse that is known as increased automaticity or abnormal automaticity. Now, because of this increased automaticity what happens, there is the increased automaticity, there is the depolarization of the cells.

So, there is depolarization of the cells means this increased automaticity usually happens in case of pathological conditions. In physiological conditions it would not happen, because physiological condition is the primary pacemaker is SA node. So, whenever any ischemia of the cell suppose what happens in that ischemic cells, there will be increased automaticity. Then we have triggered activity, then we have reentrant mechanism. So, we will come one by one.

In increased automaticity as I told you, any rhythm which is escaping the overdrive suppressions of the SA node and that escape rhythm is taken over by the latent pacemakers. Now, pathological conditions usually I told you occurs because of the ischemia. Now, the very main important principle for ischemia generating automaticity is ischemia will cause hypoxia. That means, there is decrease in the concentration of oxygen. The cells will be having hypoxic condition.

So, in this hypoxic condition we know there are various important channels, we are already discussed in the voltage gated channels. There are the first one is the potassium ATP channels, potassium ATPS channels, then we have the primary active transport that is sodium potassium ATPS pump. These all act under the concentrations of oxygen. So, this hypoxic condition will deteriorate the functions of primary active transport as well as the potassium ATPS channels. So, this will cause depolarization of the cells.

Now, depolarization of the cells will occur because the if suppose the primary active transport is not functioning. That means, the cell is not coming to the resting membrane potential of minus 90 milli volt, which means the cells is having loading of cations. Because it is not able to throw out the sodium inside and it is not able to take the potassium inside. 3 sodium is 3 is to 1 is the ratio coupling ratio, 3 sodium is thrown out and 2 potassium is taken inside and that maintains the negativity of the resting membrane potential across the cell. So, this will not be possible.

So, because of the loading of the cations and in case of potassium ATPS channels usually what happens this channels open whenever there is the hypoxic conditions. So, whenever this hypoxic conditions will occur, the channels will open there will be more potassium in the extracellular. So, extracellular fluid if it contains more potassium because of the more flux of the potassiums because of the more opening of the channels, what will happen because of the increased potassium concentrations that is outside hyperkalemia that will also cause depolarization in the cell membrane depolarization. So, all this will cause membrane depolarization. Suppose if the normal cell is having minus 90 milli volt, the ischemic cell will have the depolarization potential.

That means, it will be minus not minus milli volt it will be around minus 60 or minus 50 milli volt. It will be less electronegative, which also means this potential will be close to resting membrane potential or threshold potential. So, these cells will be very much active. This the cells this ischemic cells will be very much liable to the increased automicity or triggered activity. So, this is the basics of this is the basis of the generation of the extracerestral or arrhythmia because of increased automaticity of the cells.

So, starts firing extracerestral and because of there is a change in the resting membrane potential towards depolarization. The same thing happens in case of triggered activity or after depolarization. After depolarization per se is not after depolarization it means the depolarization is occurring after or during the repolarization phase. Now, as we had already been come across the terms about absolute refractory period and relative refractory period. So, in case of early after depolarization means when the depolarization is occur during the late repolarization that means during the refractory period late refractory period.

So, at that time what happens the strong stimulus is enough to trigger another action potential. Similarly, when the repolarization is already over at that time if the action if the trigger is occurred a strong stimulus is given then also we will get another action potential. So, triggered activity is usually after depolarization that means depolarization which is occurring during repolarization or soon after the repolarization a strong stimulus can activate and generate an action potential and produce extra systoles or extra beats. Now, we come to the re-entrant phenomenon or the other name is circus movements. Now, what happens in case of re-entrant phenomenon generally the impulse will move in a cell in this directions.

Generally there because the impulse conduction is regular the normal impulse conduction is regular fast uniform without any hindrance in the pathway, but what happens whenever there is a hindrance in the pathway. Now, suppose there is a scar tissue now suppose there is a scar tissue what will happen the impulse will come and it will pass in this fashion. Now, this portion it may not go to this portion because this portion might be refractory. So, whenever it will pass here after that whenever it will come back to this region if this portion the portion which was refractory earlier that might become excitable and hence the circling motion of this current goes on impulse goes on. So, we can see depolarizing impulse arrives to an area that displays the characteristics of the necessary to cause re-entry phenomenon.

So, here it is entering you can say this is the scar tissues the impulse encounters a block this is the black circle and manages to get around it is getting around it. Now, this impulse would not be coming to this part because the cells here are not excitable. Now,

for the re-entry phenomenon to occur the very important thing is the cells should be excitable. So, that the current could be conducted in that cells if the cells are not excitable then the current would not get conducted through those cells. So, here the impulse gets blocked because of the refractoriness because of the non-excitability of the cells.

So, the impulse passes here generally the impulse should travel here, but in spite of travelling the impulse runs around the block because by the time it moves here this cells becomes excitable and finally, the blue cell will also become excitable. So, by the time the impulse arrives to the previously blocked area this area has become excitable the circus movement has been established and the wave front current encounters the excitable tissue and this re-entry loop is created. Now, this if it is happens one it is all right, but if it is happening constantly like this it will throw numerous depolarizing waves it will throw numerous depolarizing waves towards the out to the myocardium. And so, this is the re-entry circuit which gives rise to various types of arrhythmia. Now the only thing to remember is the excitable cells are necessary for re-entrant phenomenon that is why termination of this arrhythmias are done with electrical shock or disease shock.

So, whenever we give the electrical shock or cardioversion to the patients who are coming with arrhythmia we depolarize all the excitable cells. This shock is given to depolarize all the excitable cells when the electrical shock will depolarize all the excitable cells. So, the re-entry circuit will die out. Generally, this re-entry loop is classified into anatomical and functional. Anatomical the most important example is atrial flutter.

This atrial flutter usually happens around the tricuspid valve. So, there is a presence of an anatomical tissue which is usually not excitable the valvular tissues are usually non excitable. So, this non excitable this valvular tissues they generally they do not generate any impulse. So, around the valvular tissue this re-entrant phenomenon occurs and this is anatomical. But when the block is can be present anywhere and we are not supposed to define where the block is present at that time this is known as functional re-entrant loop which usually happens in case of atrial fibrillation atrial fibrillation ventricular fibrillation.

So, these are the functional re-entrant loop. Now, coming to the Wolf-Parkinson-White syndrome. Now this Wolf-Parkinson-White syndrome is mainly because of the AV re-entrant tachycardia phenomenon. Now, we will see the first picture that is a normal atroventricular AV means atroventricular. So, normal atroventricular impulse transmission is occurring.

This is S a node and here we have the AV node. So, normally these are the internodal pathways through which the S a node transmits its impulse to the AV node and finally, this because of the slow conduction of the AV node the impulse transmission occurs which means the atria cannot transmit the impulses directly to the ventricles right. But what happens if there is an accessory pathway which connects the atria to the ventricles. So, at that time this conduction the impulse conduction which is happening from S a node to AV node that would not happen because through that accessory pathway the impulse will get transmitted directly to atria to ventricles already the impulse is getting a shortcut. So, why it will take the root of AV node and why it will go under the AV nodal delay.

So, that is exactly what happens in case of pre excitation or the wool parkinson white syndrome where there is an accessory pathway and this accessory pathway is nothing but the bundle of kind. Now, whenever we are talking about the accessory pathway which means we are having problem in the AV nodal delay. So, accessory pathway is transmitting the impulse fast which means the AV nodal delay would not be present. So, an AV nodal delay in the ECG is usually marked with the PR interval. So, which means the normal PR interval would not be present in case of wool parkinson white syndrome.

Besides that what will happen see here the pre excitation is occurring. So, when the impulse will get transmitted directly from the atria to the ventricles with the help of this accessory pathway it will meet the myocardium at some point of time. Soon it will meet the myocardium ventricular myocardium and the ventricles will get depolarized earlier there will be early depolarization of the ventricles. So, whenever there will be early depolarization of ventricles. So, what will happen the point where the accessory pathway and the normal pathway is meeting that will give rise to a wave that is known as delta wave.

So, here we are getting this upward sloping this is nothing, but the delta wave. So, there will be early depolarization of the ventricles formation of the delta wave the PR interval will be very short and of course, the QRS prolongation will be there you can see the QRS duration is usually less than 0.12 seconds. But here the QRS prolongations will be more than the interval will be more than 0.

12 seconds. So, if this is the ECG been asked when a patient is presented with dyspnea chest pain palpitations what are the findings in the ECG. The ECG paper this is usually rolled at the speed of 25 millimeter per second for better accuracy 50 millimeter per second speed is taken. So, here we can see there is no at all PR interval. So, if we say this is the P wave this is the P wave. So, we can see the PR interval is very shortened.

So, PR interval is very shortened. So, QRS complex here we have 1, 2, 3, 4 around 4 boxes of QRS complexes ideally it should have 2.5 boxes. So, 4 boxes of QRS complexes that gives around more than 0.16 seconds. So, QRS complexes wide, then we have PR interval very shortened very short PR interval.

And we can see this slope this is nothing but the delta wave, we can see this slope this is nothing but the delta wave delta wave is present. So, this is nothing but the pre excitation reentrant phenomenon or ABRT that is atrioventricular reentrant tachycardia very specifically you can say it as wool-partinson-wight syndrome what we get. So, the next we come to premature atrial contractions very short form we use PAC back. So, premature atrial contraction as the name suggests in heart the atria there must be some ectopic focus in the atria which is generating the impulse. The ectopic focus can present anywhere at the level of atria.

So, that will give rise to premature atrial contraction that will give rise to premature atrial beat which is a P wave which will which should always precede the normal sinus P wave. The premature P wave appears before the normal sinus P wave. Now this P premature bead should reset the sinoatrial node the sinoatrial node should be reset. So, that the next bead for the next sinus bead to occur.

So, that is why that resetting takes some time. So, that is why we get an income like incomplete compensatory pause which we do not which we get actually complete compensatory pause in PVC that is premature ventricular contraction. So, what we see over here this is a sinus bead this is a sinus bead and this is a premature atrial bead. So, premature atrial bead is having a P wave we can see a P wave a Q R is complex and a T wave all the waves are present then we get another sinus bead. Now, this premature atrial bead we can see the duration this duration between the bead and the sinus bead is more than the duration of between the normal sinus beads. And to be very specific before the duration which is present which is present before this bead that is the duration from previous sinus bead to the next sinus bead is usually less than 2 R R, R R is the normal sinus cycles.

Now, these are the few differences we see in case of premature ventricular cycle beads contraction also. So, here the premature P wave is present which should always precede the P wave should always appear before the sinus P wave. And the interval between the premature bead and the next sinus bead will be longer than one normal sinus interval. So, this is about the premature atrial contraction. We will now see having keep like keeping this in mind we will see the premature ventricular contraction.

Now, premature ventricular contraction means the ectopic focus is lying anywhere in

the ventricles. The ectopic focus is lying anywhere at the level of the ventricles. So, whenever the ectopic focus at the level of ventricles will generate an impulse generally we do not get the P wave because it is generating at the level of ventricles not at the level of atria. So, here premature ventricular contraction would not be having P wave in preceding to it the bead.

So, this is the premature ventricular contractions. Here we can see the normal sinus bead and this is the premature ventricular contractions with the T wave inversions. Now, this is mainly because of the abnormal repolarization. We can get to see the T wave changes because of the abnormal repolarization due to the abnormal depolarization. The second most important thing is the duration. Now, premature ventricular contraction usually does not set the sinuatrial node.

So, that is why it usually gives a complete compensatory pause. That means, the next bead the next sinus bead will occur at it is own schedule. This is one R R interval and from here to the next sinus bead we get the two R R interval cycle. This is the PVC. The sinus bead after a PVC occurs on schedule due to the complete compensatory pause.

So, this much you have to remember in premature ventricular contraction. Now, this sort of ECG's you are supposed to remember you should not forget. What is this ECG where you can at least identify the P waves and the waves and what is this ECG where you get we are getting visor irregular small waves. So, one is the atrial flutter the other one is the atrial fibrillation.

In one you get where the waves are very much visible. Those waves are known as flutter waves similar to that of the P waves and the irregular waves which you are getting those in the other ECG those are fibrillatory waves. Flutter waves denoted by capital F, fibrillatory waves denoted by small f. Now, atrial flutter the mechanism already had told you the reentry phenomenon occurs at the level of anatomical location that is usually around the tricuspid bulb. You can see this is the tricuspid bulb the reentrant phenomenon here can occur anticlockwise can occur clockwise. Based on the reentry circuit whether it is occurring anticlockwise or clockwise we get the appearance of the flutter waves positive or negative in the respective leads.

So, generally the rate is usually 250 to 300 beats per minute. Now since the atrial rate is around 300 beats per minute. So, AV conduction is because slow it will only impart 150 beats to the ventricles and the ventricle rate will be 150 beats per minute which is regularly irregular. The flutter waves very specifically we can see the base line is giving saw tooth appearance. Now, these are the flutter waves we can see the flutter waves here 1 and 2 these are the negative deflection these are the negative in lead 2.

This is the most common type of atrial fibrillation which is usually occurring because of the anticlockwise or counterclockwise motion around the tricuspid bulb. In lead 2 it is occurring negative. So obviously, in lead V 1 it will be positive the flutter waves are positive this typically they appear as the P wave with shorter duration. So, atrial flutter next we come to the atrial fibrillation we should remember atrial flutter 300 beats per minute atrial rate and the ventricular rate is 150 beats per minute which is regularly irregular and we get saw tooth like appearances. Now, atrial fibrillation with fibrillatory waves this is small f waves these are irregular waves there is absence of P waves we do not get to find any P waves over here.

And the f waves morphology if you see they are beating irregularly irregularly irregular. So, 300 to 600 beats per minute. So, ventricular rate is 120 per minute around and this is baseline fibrillatory waves we get in atrial fibrillation. Atrial tachycardia, atrial flutter, atrial fibrillation the rate actually increases. The tachycardia is more than 100 over here and atrial flutter the atrial rate the atrial tachycardia the atrial flutter is around here is 250 to 300.

The atrial fibrillation is obviously, 300 to more than that 300 to 500 or 600 beats per minute. So, this is all about your atrial tachycardia's. Now, we come to the atrioventricular nodal reentry tachycardia. This is not to be confused with atrioventricular reentry tachycardia. This is nodal reentry tachycardia that was Wolff Parkinson syndrome that is AVRT.

This is occurring at the level of junction and AVRT was occurring at the level of atria that because of the internodal pathway accessory pathway. So, this is usually supraventricular in origin it is also called as PSVT. Paroxysmal supraventricular tachycardia since it is occurring at the level of AV node. So, that is why it is supraventricular paroxysmal because it happens suddenly the onset is sudden the ending is sudden.

So, that is why we called is as PSVT. Now atrial impulses the mechanism will see this is the avinotal junctions the atrial impulses from ectopic focus reaches the AV node. The ectopic focus is anywhere at the supraventricular level it reaches the AV node. Now the two pathways in AV node is there one is refractory the other one is where it will get conducted. So, it gets conducted through the other pathway and reach the ventricles where the ventricles get depolarized, but by the time it reach the ventricles the other cells becomes excitable. So, as I told you the reentry phenomenon needs the excitable cells to occur otherwise it will die out.

So, the refractory pathway now gets excitable impulse get conducted through the same it gets back to the AV node and the reentry circuit. So, what is happening this reentry circuit is moving and moving. So, there is a wave front forming at the level of AV node. So, that is why it is known as atrioventricular nodal reentry tachycardia and it is usually not accompanied with the P wave. So, this P wave is not present sometimes you can get an inverted P wave or the wave just after the QRS complex, but that is actually pseudo S waves.

So, this usually occurs because of the retrograde travel of the impulse from ventricles to the bundle of his to the AV node and then to the atria. So, because of this you might get otherwise generally we do not get the P wave in case of this supraventricular tachycardia. Now coming to the ventricular tachycardia the most dangerous arrhythmias. So, ventricular rate usually ranges between 100 to 150 beats per minute that is slow ventricular tachycardia.

150 to 250 beats per minute that is ventricular tachycardia. More than 250 beats per minutes that is ventricular flutter, more than this we have the ventricular fibrillation and then a systole. So, wide QRS complexes you will get in ventricular tachycardia is one of the important features of ventricular tachycardia. Besides that the classification can range based on the anatomical location from where the tachycardia is occurring, duration of the tachycardia based on that. So, sustained V T or sustained ventricular tachycardia means it is occurring for more than 30 seconds, less than 30 seconds non-sustained V T. Monomorphic ventricular tachycardia means only one ectopic focus is there in the ventricles from where reentry phenomenon is occurring.

Generally the fascicular V T, fascicular ventricular tachycardia usually monomorphic V T. The polymorphic V T which is further more dangerous that is many ectopic foci are there, too many ectopic foci are there we do not know from where the impulses are originating. The most common example is long Q T syndrome, then we have Brugada syndrome. Now, in case of long Q T syndrome the polymorphic V T which is occurring that is also known as Torsades de Pointes. So, this is the polymorphic ventricular tachycardia which is occurring in the long Q T syndrome.

Now, this is the ventricular typical picture of a C G which is seen in ventricular tachycardia. Here you can see the P waves, you can see the P waves, you can see the P waves, we can see the wide Q R S complexes. What we can see is the A V dissociation that means, there is A V dissociation P waves are seen usually and Q R S complexes are seen separately. So, there is an A V dissociation, A V dissociation is very much confirmatory in case of V T, whenever A V dissociation is occurring that means, it is ventricular tachycardia. Next we have the Q R S complex which is wide and the normal

between the ventricular beats and the normal beats there, so that is a fusion beat.

Now, fusion beat also occurs in case of premature ventricular contraction. Suppose, the atrial impulse and the premature ventricular contraction impulse they coincide with each other I mean they are occurring at the same time. So, at that time fusion of this two beats will occur, so that is also known as fusion beats. So, this is very typical that is ventricular fibrillation, we can see irregular waves and there is no P wave, no Q R S and no T waves. So, these are fibrillatory waves, this fibrillatory waves are seen in case of ventricular fibrillation. And ventricular is a progression of ventricular tachycardia ventricular tachycardia, this is a progression of ventricular tachycardia to ventricular fibrillation, after this what will happen a flat line in the ECG.

This flat line in the ECG is nothing but a systole or also known as pulseless electrical activity. That means, ECG is not showing any electrical activity. So, this is known as this is the usual transformation if the ventricular tachycardia is not promptly managed, then that can further progress to ventricular fibrillation which is life threatening, because it can go to cardiac arrest. So, what is this ECG which is shown over here. Now, this is the ECG we can see some artifact over here, this artifact these are nothing but pacing stimulus artifact.

That means, pacemaker is placed in the heart. Now, where this pacemaker is placed, if this pacemaker is occurring before the Q R S complex, which means it is a ventricular pacing has been done. If this artifact would have been before the P wave that means, atrial pacing would have been done. So, in this case this is the ventricular pacing which is done. So, now coming to the summary of today's topic that is tachyarrhythmias, Q R S complex is less than 0.

12 seconds usually S V T, if it is more than 0.12 we take it as S V T as well as bundle branch block or any electrolyte disturbances. It can be pre excitation syndrome that is accessory pathway, Wulff Parkinson white or AVRT or ventricular tachycardia. So, pre ventricular tachycardia usually they are not that much dangerous, but in case of the Wulff Parkinson syndrome or any ischemic heart disease present with the supra ventricular tachycardia proper care should be taken. And ventricular tachycardia is always concerning because it can progress to ventricular fibrillation and cardiac arrest if not treated well. Lastly, but not the least the management of the tachycardia or tachyarrhythmias not only depends on the ECG findings, it also depends on the symptoms of the patients as well as the hemodynamic status of the individual.

For example, if a person is coming to you with arrhythmic symptoms, but the person is hemodynamically stable, the blood pressure is all right, the person is not getting

hypotension. So, you will take the ECG recording and you will try to find the diagnosis based on the ECG findings and treat accordingly. But if the patient is not hemodynamically stable and coming with the arrhythmias that means, the patient is already having deteriorating blood pressure, there is some electrolyte imbalances also. So, at that time the patient should be generally treated with the thumb rule that is electro cardioversion is done, electrical shock is given to stop the arrhythmia. Because if that arrhythmia pauses further progresses, the patient will go into cardiac arrest ventricular fibrillation.

So, hemodynamic status should be corrected first and then the further diagnosis of the several types of tachycardia should be made. The treatment for the long term follow up of this patients usually we go with the anti arrhythmic drugs first and then the other ablation therapies are there and pacemaker pacing, artificial pacemakers or implantable cardiac defibrillators are there. But before going on to this therapies one should always start with the anti arrhythmic drugs. The patients compliance if it is not good with the anti any classes of the anti arrhythmic drugs, then they should go for the ICD or the catheter ablation therapy. With this I would like to conclude today's topic. Thank you. .