Course: Electrophysiology of Heart

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Lecture 13: ECG and Myocardial Infarction

Hello everyone. So, today we will discuss our next topic that is ECG and Myocardial Infarction. So, so far we have ah discussed about various abnormalities which we see in ECG. We will have a brief recap of that, then we will classify what are the acute coronary syndromes the diagnostic criteria of myocardial infarctions in ECG and physiological basis of ECG changes in MI and how you can locate or localize ah a particular MI on the ECG. So, ah arrhythmia generally we classify into normotopic and ectotopic. So, ectopic means ah the any arrhythmia which is arising from the away from the normal site.

So, normotopic we have sinus arrhythmia, sinus bradycardia and sinus tachycardia. Now, in all these there is difference in the ah heart rate with respect to either with respect to respiratory cycles or there is decrease in the heart rate there is increase in the heart rate. But generally the difference of the the increase or decrease in the variations in the heart rate this is regular in nature. And generally we have PQRS T waves which are normal, we do not get to see any abnormality or any morphological changes in the PQRS T waves.

So, regular ah rhythm and PQRS T normal. Now, ectopic arrhythmia we have heart block, extracestals, paroxysmal tachycardia, others like atrial flutter, atrial fibrillation, then we have ventricular fibrillation etcetera. So, heart block generally we have sinoatrial block and the atrioventricular block. Sinoatrial block means there is delay or block in the conduction of impulses from SNO to AV node. So, in this case generally the dropped bit will be PQRS T waves.

So, ah that is sinoatrial block. Now, we have the atrioventricular block where there is a delay or con delay in the conduction or there is a block in the conduction of impulses along the AV node. So, either the PR interval could be prolonged, so that is first degree AV block, QRS can be dropped, so that is second degree AV blocked that can be impulses can be dropped sporadically or there can be atrioventricular dissociations where P and QRS complexes are electrically dissociated. So, this is third degree AV block. So,

based on the certain features we can easily identify which type of block is present in the ECG.

Now, the paroxysmal tachycardia, paroxysmal means which is arising suddenly and ending ah also suddenly. So, tachycardia is another way of classification of this tachycardia is based on the narrow QRS complexes and wide QRS complexes. So, narrow QRS complexes means if it is less than 0.12 seconds, wide QRS complexes means if it is more than 0.12 seconds.

So, usually narrow QRS complexes when we tell we mean it is a supraventricular usually in origin. Generally, we see narrow QRS complexes in supraventricular origin and wide QRS complexes generally we see in the ventricular origin. Now, they again this features can be regular and irregular. Narrow QRS complexes which are regular in nature generally that is AV nodal reentrant tachycardia or that is SVD, supraventricular tachycardia, paroxysmal supraventricular tachycardia where generally we do not have any P waves. Then normal atrial tachycardia and also atrial flutter where usually the ah atrial rate is between 250 to 300 beats per minute.

Now, if this tachycardia in narrow QRS complexes is irregular in nature we go for atrial fibrillation. Flutter we see get to see the flutter waves the capital F waves and fibrillation we have the small fibrillary waves very irregularly in nature. Wide QRS complexes again regular and irregular. Regular we tend to see in case of monomorphic ventricular tachycardia monomorphic BT for example, fascicular BT and also we tend to see it in atrioventricular reentrant tachycardia or Wolf Parkinson White syndrome. And irregularly white QRS complexes we usually see in case of polymorphic BT like Long QT syndrome, Brugada syndrome and ventricular fibrillation.

And extracistals are mainly of two types that is premature atrial beats and premature ventricular contractions. So, premature atrial beats as there is a premature atrial contraction. So, there will be a premature P wave before the sinus wave and in case of premature ventricular contraction since the origin is from the ventricles. So, there will be no P wave as well as there will be a compensatory pause and also there can be fusion beats. When the atrial impulses and the premature atrioventricular impulses fuse together.

So, there can be fusion beats. So, this is the general layout of different types of arrhythmia. So, based on this brief we can easily look out what are the changes in the ECG. Now, say this is the first example of ECG, we will discuss what are the abnormalities in this ECG. So, first we are supposed to take about the as per the ECG rule that is the rate.

So, here the heart rate will be now based on this ECG we are supposed to follow the 10 seconds rule. So, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36. So, in 10 seconds we are getting we have counted the beats. So, it is 36 beats into 6 we are supposed to do it will ah come around 200 ah 10. So, approximately it will be around 200 ah 10 and I am not going into the absolute calculation 210 beats per minute.

So, it is more than 200 beats per minute. So, heart rate ah if we just go 36, 3, 18, 216. So, 216 beats per minute. Now, next we are supposed to see what is the ah axis the rhythm is actually regular over here the rhythm is regular. Axis we define by the ah we will take the quadrant approach that is lead 1 and AVF which is both are directing positive sides.

So, axis is normal. Now, we will see the QRS complexes the waves over here first and foremost we do not get to see ah any P waves over here. In any of the rhythm we are not finding any P waves you can see we are not finding any P waves over here. So, which means there are no P waves. Second if we take the QRS complexes we tend to see the QRS complexes are narrow.

So, QRS complexes are narrow. So, ah it is less than two boxes. So, we get narrow QRS complexes usually normally we get it around 2.5 boxes. So, narrow to QRS complexes.

So, so far we have got no P waves ah axis is normal regular rhythm rate obviously, it is tachycardia it is beating around 216 beats per minute and normal QRS complexes. So, ah this ah this is as per our classification it should go normal QRS complexes no P waves, tachycardia, regular QRS complexes this is regular. So, this should go around paroxysmal supraventricular tachycardia or atrioventricular nodal reentrant tachycardia. So, ideally if this ah if there has been changed with the ah QRS complexes like if it if it is a wide QRS complexes we would have made some other the diagnosis. So, this ECG is of ah atrioventricular nodal reentrant reentrant tachycardia or paroxysmal supraventricular tachycardia.

Now, next we will see this ECG. So, first we will calculate again the rate the sinus rate. So, here the sinus rate is here we can go with the ah ah boxes the 1500 boxes. So, it is around ah 5, 5, 10 and 15. So, approximately 1500 by 15 that is 100 beats per minute.

So, here the heart rate is around 100 beats per minute. Now, ah second thing is ah the regularity it is regular, then we have ah the QRS complexes and the P waves ah. First and foremost we could see this beat we can say this beat very characteristics of

ventricular premature complexes and this is not accompanied with P wave you can see no P wave. So, this is nothing, but sinus rhythm with ventricular extracistals or ventricular premature complexes. So, we have no P waves also what we get in case of ventricular ah ah like ventricular premature complexes and the QRS complexes obviously, that is normal QRS complexes is normal and the axis also if you see it is also positive axis.

So, normal axis. So, that is why we did not go into any other causes except the ventricular premature complexes or ventricular extracistals. So, with this we will move on to our next topic that is classification of the acute coronary syndromes. Now, there can be several risk factors for example, obesity ah adverse eating habits like eating several junk foods, then we have ah diabetes chronic illnesses like diabetes mellitus, hypertension, then dyslipidemia, increased level of cholesterol, lack of physical activity, stress anxiety there can be various ah and of course, aging. So, there are various risk factors which usually give rise to inflammation in the coronary arteries. Now, whenever there will be inflammation in the coronary arteries there will be along with calcification there will be atherosclerotic plaque formation.

Now, this plaque will either occlude or rupture. So, whenever this rupture of the plaque or occlusion occurs that means, it is it is occluding the coronary vessels. So, the person will come with this severe chest pain severe sudden chest pain because of the occlusion of the blood vessels because of the obstruction of the coronary blood flow. Now, if this occlusion of this blood vessels is complete then generally it gives rise to a transmural ischemia. If this occlusion of the blood vessel is incomplete usually complete occlusion means throughout the endocardium ah from endocardium to epicardium there is occlusion.

So, that is transmural ischemia and partial occlusion means the coronary arteries partially blocked. So, that is usually sub endocardial ischemia. Now, in this two cases if there is transmural ischemia and we generally get to see ST elevations in the ECG we call it as STE that is ST elevation acute coronary syndrome or STEMI that is ST elevation myocardial infarction. Now, if there is no significant ST elevations, but still there is chest pain we usually call it as non ST elevations or non STEMI that is ACS that is acute coronary syndrome. Now, that is the usual typical feature in cases of sub endocardial ischemia.

Now, besides this ECG findings there are cardiac biomarkers mainly the troponins. This troponins will confirm the diagnosis of the myocardial infarction generally in case of both ah STEMI as well as non ST elevations ah MI we generally tend to see this troponin level is increased. If the troponin level is normal, but the patient is presenting with the

above symptoms then you usually call it as unstable angina. So, this is what ah your acute coronary syndrome. Now, the clinical uses of ECG in MI is usually we do have to see the 12 lead ECG.

We cannot see only the chest leads or the limb leads because as I told you lead is be a behaving like a camera. A camera is seeing your heart from various directions. So, 12 cameras are placed 12 cameras will see your various portions of the heart and that is very ah important for myocardial infarction because myocardial infarction is a very dynamic process. So, ECG during the chest pen generally you ah tend to see the ST changes in the MI. Now, during the chest pen means the person is presenting in the acute stage that we call as acute myocardial infarction.

So, in acute myocardial infarction usually ST and T changes will be present. Now, the T wave inversion which is usually present in case of ah myocardial infarction we get to see. This isolated T wave inversion is there then it is not pathognomic of myocardial infarction. We can tell this of acute myocardial infarction. If only isolated T wave inversion is present without any significant STT changes.

So, isolated T wave ah inversion is generally means post myocardial infarction that means, after the injury it has been ah the ECG recording has done post the ah myocardial injury. So, stable angina cannot be diagnosed with resting ECG or stable angina you have to ask the person for exercise stress test like cardiac ah like exercises ah we do treadmill test we do. And we have to check the previous ECGs also continuous monitoring of ECG is required. So, in this diagram we see the various biomarkers which are usually raised in case of cardi ah myocardial infarction. Generally ah we take the highly specific and sensitive troponin levels we have cardiac T and I troponins.

These are very much sensitive ah for the myocardial infarctions they usually get raised within they usually get rest within 2 to 3 hours of the ah myocardial infarction and subside within 7 days. So, 2 to 3 hours of the injury troponin T and I will rise and within 7 days it will subside that means, the level will become normal that is why we ah during this period we tend to ah check for different levels number of samples of troponins we collect. Now, the other biomarkers which are present like creatinine ah and myoglobin. So, these are usually non specific in nature because they are also present in the skeletal muscles. So, that is why we tend to emphasize troponin T and troponin I.

Now, what is the natural course of occlusion of this ah myocardial infarction? Now, whenever there is an occlusion in the coronary vessels immediately ischemia will start occurring. Now, this ischemia will start at the sub endocardial region, the infarction commences in the sub endocardial region. Now, it will give a time period of 20 to 30

minutes why because whenever there the cells are ischemic in nature they will ah get to to remain in viable state the cells will go under anaerobic metabolism. So, cells to remain viable viable they will undergo anaerobic metabolism, but this occurs only for 20 to 30 minutes. Within this 20 to 30 minutes if the blood flow is restored two condition can occur either the blood flow that means occlusion is over blood flow is restored.

So, what will happen that ischemic area will heal with a scar and there will be slow ventricular remodeling. The next important thing is if the blood flow is not restored that means there is progression of the occlusion blood flow is not restored. If the blood flow is not restored this ischemia will progress very fast from the sub endocardial region to the epicardial region like a wave front. Like a wave front it will spread from the sub endocardial region to the epicardial region and this tissue will become necrotic it will die the ischemic myocardium dies and this ah tissue will become necrotic and this occlusion is not if it is solved within 2 to 12 hours the entire ischemic area will become necrotic. The 12 hours time is being given because usually we get ah because of the collateral coronary circulations.

So, that is why ah this 12 hours time we get. So, 2 to 2 ah 12 hours if the blood flow is not ah reversed the occlusion is not resolved then the area will become necrotic. Now, why this necrosis or why this ischemia is occurring in the sub endocardial region because sub endocardial region is very prone for ischemia. This is very prone to ischemia because sub endocardial region is lying away from the ventricular cavity to enjoy the oxygen from the ah ventricular cavity. Also ah we know the coronary blood flow is from epicardium to endocardium. So, that is why the sub endocardial region ah is very prone to ischemia.

I ischemia occurs in the sub endocardial region it will spread to the epicardial region. So, so this is the natural course of occlusion. Now, we will see what are the changes we see in the ECG that is the ST and T changes. Now, normally ST changes means I am reflecting the plateau phase of the action potential in the contractile cell. So, the I am reflecting the ah plateau phase there is some abnormality in the plateau phase which is occurring that is causing the disturbance in the ST segments.

So, normal findings ah what we see with the direction with the T wave this you must remember that is lead 1 lead 2 V 5 and V 6 this should display positive T waves and lead AVR should display a negative T wave. This is the normal ECG we get AVR all negative waves and lead 1 and lead 2 and the V 5 V 6 the lateral leads ah that display usually inferior and the lateral leads that that usually display the positive ah T waves. So, the next thing is very important now the magnitude of the ST depression or elevation that is measured with reference to the J point. Now, I had already told you this is the J

point. So, whatever depression or elevation is occurring you should be measuring it with reference to the J point.

Suppose this is ST elevation which is occurring this is the base line and this is the J point. Now, whenever there will be elevation or depression the J point will also shift. So, that is why we measure with reference to that. So, this is the ah 4 squares means 4 into 1 millimeter that is 4 millimeter ST elevation we have got. Similarly, in case of depression this is the base line and this is the J point.

So, 3 small boxes equal to 3 millimeter of ST depression we have got. So, now, normal versus ischemic ST depressions in cases of physiological conditions generally ST depressions we tend to see ah we tend to see an up sloping ah ST segment. Now, this up sloping ST segment is usually seen in case of hyperventilation or exercise it is not ah ischemic it is not seen in ischemic conditions. So, this is a physiological till there is no inversion of the T wave. So, there is an here we can see ah upward deflections of T wave that is T wave is positive and there is upward sloping of the ST segment with that ST segment depression this is normal.

What is not normal is whenever there is a flat or horizontal ST segment or there is a down sloping of the ST segment associated with T wave changes there can be T wave upward deflections or there can be negative T wave ah deflections that means, T wave inversions. So, these are typical of ischemia the horizontal ST changes and the downward sloping of the ST changes. So, the current guideline for ischemic ST segment depression is horizontal or down sloping of the ST depressions more than equal to 0.5 millimeter at 2 contiguous leads.

So, this is the current guideline. Next, we see what is the ah normal versus ischemic ST elevation. So, whether ST elevation is convex these are all ST elevations is a baseline this is the J point this is the J point in every cases this is the J point J point J point. So, we can see the ST elevations it can be convex it can be straight up sloping it can be straight horizontal it can be straight down sloping all are abnormal. There is ST elevation can be convex it can be straight it can be up sloping it can be down sloping all are considered to be abnormal. So, the current guideline is the ST elevations should be present in at least 2 contiguous anatomical leads.

So, whether the age is more than 40 years less than 40 years irrespective of the gender male or female generally the elevation should be more than 2 millimeter in V 2 and V 3 and more than 1 millimeter in other leads. So, this is very specific there can be difference of 0.5 millimeter in the V 2 and V 3 leads ah interior chest leads, but yes there has to be elevation ST elevation of more than 1 millimeter in other leads. So, these are strongly

suggestive of myocardial infarctions. Now, when we are having ST elevations we get to see ST depressions in the reciprocal or the in the other leads this is nothing, but called as reciprocal ST depressions.

Reciprocal ST depressions are nothing, but the mirror images or the mirror reflections of the ST elevations which is seen in the other leads because elevation or depression we get to see based on the ST vector and the exploring electrode. So, this is the normal ah this is the ischemic ST elevations we get to see depression we have seen and elevation we have seen. Now, we will come to the normal versus ischemic T wave pattern this is the normal T wave pattern. Now, in case of hyperkalemia generally we get ah tall T waves. Now, tall T waves is usually also present in case of ischemia hyper acute tall T waves we get the difference is there has to be some STT changes in case of ah ischemia.

And also we get to see short base in case of hyperkalemia this is the short base and in case of broad base I mean in case of ischemia we get to see broad base in the T wave. Besides this is the T wave isolated T wave inversion that is post ischemic in case of acute ischemia we get to see ah inversion of T wave as well as significant ST and T changes. So, these are very pathognomic of myocardial infarctions as this I already told you without any ST and T changes only isolated T wave inversions we are getting. So, that is post ischemia that means, the recording has been taken post sometimes after post injury. T wave inversion with ST deviations that is acute ischemia and this T wave inversion should be more than 1 millimeter into contiguous leads.

So, this is the current diagnostic criteria of STT changes and the T wave changes. So, what is the ECG course of this STT changes and the T waves morphology changes in the myocardial infarctions. Now, initially whenever the occlusion is occurring there is a hyper acute T wave formations this we can see the T wave is tall there is hyper acute T wave formations without any STT changes. Now, then acute conditions ah generally within minutes to hours there will be ST elevations we can see the this is ST elevation this is ST elevation sub acute means ah when certain hours has been already progressed this is ST elevations this is ST elevations. Now, in all these stages usually patient presents with severe chest pain this is the time if the patient is coming ah with severe chest pain in an emergency this features usually we get in the ECG.

That means, they are coming in this phase the acute to sub acute phases with T wave morphological changes and the significant ST elevations ah changes I am talking about STEMI. Now, next we get to see when already the injury has occurred ah for some days the progression has occurred we tend to see pathological Q waves. Generally pathological deep Q waves Q waves we tend to see in case of ah old lesion. So, here we see Q wave here we also we see Q wave. So, Q wave is seen pathological Q wave is seen

along with T wave inversions and later T wave inversions in chronic cases when it is ah old in fact, is staying for years usually the ah morphology comes to comes back to normal in case of T wave, but you get to see deep Q waves.

So, this is how ECG progression takes place in case of MI. Initially tall T waves then ST elevations during the acute phases then T wave ah inversion occurs then you get to see the Q waves and the T wave inversions usually resolves with the chronicity. So, this is a 60 year old male with retrosternal chest pain he has come with this ah in the emergency and we get to see certain changes in the ECG. First and foremost we get to see ah these are the Q waves these are nothing, but the Q waves we get to see in case of lead 2 ABF and lead 3 here we see Q waves. Second we see ST segment elevation. So, ST segment elevation we also see in case of lead 2 ABF and of course, this is that is lead 3.

We also see ST segment ah depression in case of AVL and I so that means, this is the reciprocal actually ah depressions we are seen ST segment depressions these are reciprocal depressions which we are seeing in the opposite lids. Now, lead 2 lead 3 and AVF if you just remember these are the inferior leads and we are seeing Q waves. So, which means this is the ECG of old inferior myocardial infarction this ECG is of old myocardial infarctions ah inferior myocardial infarctions because we have seen the Q waves as well as the ST segment elevations. Now, the localizations of acute MI how you will state where is the occlusion the culprit how to find it out. Now, this is the normal conducting pathway sinoatrial node is usually supplied by the ah right coronary artery similar is that of right coronary artery supplied ah the atrioventricular node and right bundle branch usually getting the supply from the left anterior descending artery and the posterior descending artery from the collaterals of right coronary artery either a right coronary artery or circumflex artery whichever is the dominant one.

Then his bundle is also getting the supply from the right coronary artery left bundle branch is getting supply from the left anterior descending arteries similar is that of the posterior fascicle and anterior fascicle they also ah get the supply from the septal branches of the left anterior descending artery. So, if we just see the lead over here the hexaxial system this is lead 1, this is lead 2, this is lead 3, then we have the ah AVL, AVF, AVR, then we have the chestlets that is V 1, V 2, V 3, V 4, V 5, V 6. So, these are the chestlets we have. So, what are the locations this leads are giving. So, septal is definitely V 1 and V 2 this is definitely the left anterior descending artery locations as we can see the septal portions is looking the left anterior descending artery.

The anterior leads is V 2 ah V 3 the anterior lead is V 3, V 4 mainly V 3 and V 4 that is the anterior leads. So, anterior leads are also saying the left anterior descending artery to some extent it is also saying the left circumflex artery also. Inferior leads means we have

lead 3, we have AVF, we have lead 2 these are the inferior leads. So, this inferior leads are saying the right coronary artery. Lateral leads usually V 5, V 6 actually V 5, V 6 ah ah depicts the ah apex of the heart.

So, they can be collectively called as apical portions they can see the apical portions sometimes we also ah denote them in the lateral portions also. So, we have V 5 and V 6, we have lead 1, we have AVL and right lateral is AVR. So, this is also seeing the ah LAD, this is also seeing the left anterior descending artery and some portions of the the left circumflex artery. So, in this way we can find out the location of the MI and the reason that means, where the occlusion has taken place ah in the coronary artery.

So, the physiological basis of ischemic changes is very important. Now, generally whenever there is a tissue ischemia the cell is undergoing ischemic process there is hypoxia because the oxygen concentration is decreased. So, there will be decreased activity of sodium potassium ATPS pump as well as decreased activity of the potassium ATPS pump ah ATPS channels. So, because of this decreased activity of sodium potassium ATPS pump the coupling mechanism that means, the throwing out of ah 3 sodium outside and taking in of 2 potassium inside this will get affected which will cause cations loading, loading of cations. So, in the cell and potassium ATPS channels also this will open because of the reduced ATP this will cause a flux of potassium ions to the extracellular fluid which will cause hyperkalemia which will in turn cause membrane depolarization.

That means, the cell will have reduced ah membrane potential. So, that is what we can see in case of diastole that means, when the cell is at rest this is the ischemic cell and this is the normal cell usually we get to see minus milli ah 90 milli volt throughout the cell electronegativity, but in case of ischemic cells it will have reduced membrane potential the potential potential will be towards the depolarization. So, that is why the depolarization vector extracellular depolarization vector will be from ischemic to the normal cell. But in case of systole we see the depolarization in case of the ischemic cells is not that act as that of the normal cells because the sodium channels which is mainly important for the depolarizations they gets inactivated faster. So, that is why we get to see the ischemic cells having lower depolarization potential compared to the normal cells that is why during systole the extracellular vector will be from the normal cell towards the ischemic cell. So, the main three reasons which we get for the ischemic changes are reduced membrane potential, delayed depolarization and early ah repolarization because of the ah reduced action potential duration.

So, first T wave inversion what how wide T wave inversion occurs now whenever there is an ischemia suppose sub endocardial ah ischemia has occurred ischemic sub

endocardial cells. We can see this cells will tend to have a depolarized resting membrane potential they will have reduced membrane potential or depolarized resting membrane potential. So, they will tend to ah have a ah ah decreased action potential durations. So, due to this decreased action potential durations during repolarization the direction will get ah reverse that will reverse the direction of the repolarization which we get to see in case of normal ventricular cells repolarization. In normal ventricular cell repolarization starts from epicardium to endocardium this is normal, but here because the strong vector is directed from the sub endocardial cells ischemic cells towards the exploring electro ah towards the we have the exploring electrode and this vector is the repolarization vector.

So, repolarization to occur from sub endocardium to epicardium repolarization wave towards the positive electrode will give negative deflection of the T wave. Generally what we get the normal is repolarization wave we get away from the positive electrode that is why we get positive deflection of T waves. So, here we are getting negative deflection of the T wave because of the repolarization wave directing towards the positive electrode. Now, the ST segment depressions and the ST segment elevation, depression occurs in the sub endocardial ischemia ST segment elevation will see in the transmural ischemia. Now, in case of ST segment depressions or in ah ST segment elevations this ST segments usually coincides with the plateau phase, but this depression and elevations are not occurring during the plateau phase these are occurring in the resting repolarized ventricular cell.

So, in a resting repolarized ventricular cell the ischemic cells again will have a reduced membrane potential or towards the depolarized membrane potential and that is actually occurring in case of sub endocardial ischemia. So, whenever this reduced ah membrane potential occur it is causing a depolarization vector. So, there will be a depolarization ah wave which will occur between the depolarized and the repolarized tissues it has been denoted by this arrow. And you can see this arrow I mean this wave is towards the recording electrode, that is why there will be a positive voltage deflection and this deflection will be from the base line. So, there whenever there will be positive voltage deflections the waves will move upward there will be an upward shift of the waves from the base line, but we will tend to see as if there is an ST segment depression.

So, baseline voltages are elevated and there is an appearance of ST segment depression. The reverse occur in case of ST segment elevation in case of transmural ischemia when this whole cell the whole endocardium to epicardium this myocardium is totally destroyed I mean there that is ischemic. So, the vector the depolarization will be away from the recording electrode. Now, when this will be away from the recording electrode it will tend to deflect a negative voltage and since it is tend ah reflecting a negative voltage there will be shift downward from the base line. So, this downward shift from

the ah this negative voltage shift downwards from the base line will give appearance of ST segment elevation in case of transmural ischemia. So, these are the physiological changes we tend to see in myocardial infarctions whenever any ECG is given to you, you are supposed to see which region of the heart is involved besides the ah sinus rhythm PQRS complexes normal axis culprit or the which ah including vessel is there and age of the infarct whether it is acute subacute or old infarct. So, with this I would like to conclude today's topic. Thank you.