- Welcome to Mayo Clinic's ECG segment Making Waves Continuing medical education podcast. Join us for a lively discussion on the latest and greatest in the field of Electrocardiography. We'll discuss some of the exciting and innovative work happening at Mayo Clinic and beyond with the most brilliant minds in the space, and provide valuable insights that can be directly applied to your practice.

Dr. Anthony Kashou: Welcome to Mayo Clinic's ECG segment making waves. In this episode, we continue our conversation with Dr. Ken Grauer, picking up where we left off on the critical role of history and having a systematic approach in ECG interpretation. We're shifting our focus to the topic of acute coronary occlusion in the ECG errors that could mean the difference between detection and amiss. We're fortunate to have Dr. Ken Grauer back with us today. Dr. Grauer is Professor Emeritus in Family Medicine following his residency training in family medicine. He worked for two years in a busy emergency department in South Florida before moving to Gainesville, Florida, where he was full-time faculty in the University of Florida Family Medicine residency program for his 30 year career until he retired from his academic practice in 2010. Dr. Grauer has written over 10 books on ECG and arrhythmia interpretation, presented hundreds of talks in workshops locally and nationally on ECG interpretation and other cardiology topics over his career. And he's been active as ever since retiring with over 3 million views on his own ECG blog. He's the associate editor and active contributor to Dr. Steven Smith's ECG blog, as well as he continues to answer numerous questions, daily queries that are addressed to him on so many international ECG internet forums that he contin continues to contribute to. Dr. Grauer. Thank you so much for coming back with us. I'm really excited about this episode here.

Dr. Ken Grauer: Thank you, Anthony. It's a pleasure to be here.

Dr. Anthony Kashou: Dr. Grauer, you know, in the previous episode, we covered the importance of incorporating the history routine use of a systematic approach to both rhythm and 12 lead interpretation. In this episode, we wanted to shift gears as we mentioned earlier, from the ECG errors and how those errors can be occur in the setting of acute coronary occlusion. And I wanted you, because you've been studying and looking at this so much, what do you have planned for us today and and where are we gonna take this?

Dr. Ken Grauer: Well, as you mentioned, Anthony, in your introduction, one of the main functions of the electrocardiogram is simply to assess the patient with chest pain and or other symptoms, or even if no symptoms, but to assess the patient for the possibility of acute ischemia and infarction. So I wanted to address a number of points that relate to this, that relate to errors that are made regarding this assessment and some simple things we can do to improve our interpretations, make them more time efficient, make them more accurate. And the first error that I would start with is that all too many clinicians in 2023 are still stuck in the STEMI paradigm. And that paradigm is simply that you need a certain number of millimeters depending upon the age of the patient and the sex of the patient and the lead that you're looking at. You need a certain number of millimeters before you qualify as having an acute STEMI with quotes around that. So this first error is that too many clinicians are still stuck in the

STEMI paradigm. And this brings up the question of what do we really care about when we're looking at the ECG of a patient who presents with new symptoms, especially new chest pain? And the answer is that we wanna know is there an acute coronary occlusion? Because these are the patients that we could do the most of if we have an acute infarction because of acute coronary occlusion, which is almost always the case. These are the patients who by reperfusion opening up the occluded artery, either by PCI, by thrombolytics, we can benefit by salvage of significant amounts of myocardium. So the problem is that the STEMI paradigm in 2023 is wrong and it's outdated. Smith and Myers have shown that at least 30%, if not significantly more than that, at least 30% of all patients with an acute coronary occlusion. And the abbreviation for this that is now being used more and more is an acute omi that's an occlusion based myocardial infarction. So at least 30% of patients who have an acute omi, you're gonna miss these patients if you're stuck on waiting for a certain number of millimeters of ST elevation. Now, how many of you have encountered this? How many of you find that when you contact your interventionist they say, well, we can't really do a cath at this point because the ECG does not yet show a stemi. There's not enough millimeters of ST elevation. Now, some of these patients eventually develop a stemi. This could be minutes or more often hours later. And yes, you finally get a STEMI and you take 'em to the cath lab and you do angioplasty or you start your thrombolytic, but hours later you've lost part of the ball game. You've lost a significant amount of myocardium that you could have salvaged if you took them to the cath lab earlier. Other patients with acute OMI acute, they never developed the STEMI criteria despite having acute coronary occlusion that may result in extensive damage with troponins that go up into the many thousands could be that their ST segment elevation was before they got to the hospital before EMS arrived on the scene. So you never saw it. So error number two, many of these patients who have troponins in the thousands, they never have an ECG that satisfies the millimeter based STEMI criteria. What do they get diagnosed as having they get diagnosed as having an N stemi? That's a non ST segment elevation mi. And I will say that at least in my experience, a majority of patients who are diagnosed as having an N STEMI actually had acute coronary occlusion, but they never met STEMI criteria. So to me, this is error number two, almost error number one. It's not appreciating that N stemi, it's really practically speaking, a useless term probably ought to be abandoned. That's in my opinion, simply because it just states that at the time that one or more ECGs were done, that there never was enough millimeters of ST elevation to qualify as a stemi. It does in no way rule out the possibility that there was acute coronary occlusion. And again, as I emphasize, Smith and Myers have shown at least 30% of the time, if not significantly more, you're gonna miss OMI if you're stuck on this definition. So error number three, this is not appreciating the ECG findings to look for when you don't meet STEMI criteria. So the new paradigm, and again, lots of credit ongoing to Robert Herman, who you had on your podcast earlier to Dr. Smith and Meyers. The new paradigm is to look for other ECG findings apart from enough ST segment elevation to qualify as an acute occlusion myocardial infarction. So in a patient with new chest pain, what are some of these other findings? Now I'm gonna first list four or five of these, and then I wanted to go over each one by giving a couple of pointers with them. So the first non enough ST elevation criteria is the presence of hyperacute T waves. Then there's what I call a magical reciprocal relationship between the ST segments. This is the mirror image opposite relationship between lead three and lead A VL with an acute inferior infarction. The third one is, is there a posterior infarction? And the last one that I'll go over today, there are a few others, but the last one is, are there dynamic ST segment T wave changes on serial ECGs? There's a lot contained in that last one. So let me start by going over. The first other criteria apart from having enough ST elevation, are there hyper QT waves. And I've never seen strict written criteria of whether or not you have quote Hyper QT waves. It's kind of like recognizing a

face. I know you Anthony, I recognize your face, but if you ask me to describe you other than to say how handsome you are, I couldn't do it in terms of words, but I recognize you. And it's the same thing. The more practice you get with looking at ST segments and t waves you get more comfortable recognizing an appearance of the ST segment and or T wave that just shouldn't be there, which in a patient with new chest pain is data. So a pearl that I would give out is if you have a patient who presents with a cardiac sounding new chest pain and you see one or two leads, doesn't have to be more, that has what you recognize as this just shouldn't be there, they're hyper QT waves. Now how do you define that? Well, I look at T waves that are what I call taller than they should be taller than expected with consideration to the QRS complex. Within that lead, for example, you'll look at a lead V2 and the R wave is usually pretty small in lead V2. So if I see a T wave of seven or eight millimeters that's taller than the R wave and the S wave is not very deep, only a couple millimeters, it is disproportionately tall, much taller than it should be. I look for T waves that are, and I usually put quotes around this, not to offend anyone fatter at their peak than they should be. They are taller than they should be. They're wider at their base, then they should be for what is a normal repolarization T-wave. And again, the more you do this, the more comfortable you get with recognizing these. Now couple of points with this. One is prior tracings, you know, is this, it looks a little abnormal. The history, it's not that definitive, but there are some new symptoms. Can I find an old ECG? And if you find that the T-wave was previously flat or even depressed and now it's somewhat elevated, that's hyperacute. And if you get in leads like the inferior leads especially, which are often low QRS amplitude, the T-wave does not have to be real tall. It's just compared to the QRS complex, it's taller than it should be than you would expect it to be. And particularly if you see it in neighboring leads. So if I see lead three is definitely abnormal, I'm gonna look at neighboring leads, I'm gonna look at leads two and A VF, and they may not be very abnormal. It may be that if I only looked at lead two or aVF alone that I wouldn't really be convinced. But in the context of new chest pain and lead three is definitely abnormal, then any slight abnormality in other leads may become significant, particularly if it is in neighboring leads. If I look at lead three, for example, and I'm concerned about the T wave there, I'm gonna look at lead two and lead V6. And if V4 has a little bit taller than it should be, I'll look at V5. Let me move on to the magical what I call, I've labeled this the magical reciprocal relation. I've developed the mirror test. I don't know if I was the first one I'd began popularizing this. 1983 was my first publication and I put it in all my books and blogs. It's what I call the mirror test, but not only for posterior infarction that I'll get to in a moment, but also for other reciprocal changes. You can see a mirror image. The heart is a cylinder, or at least the left ventricle is cylindrical in shape, such that if you have ST segments going up in one of the walls, really almost all of the other walls for a cylinder are gonna be opposite. And instead of ST segments going up, the ST segments may go down. And the shape of that ST segment is a mirror image. You just flip it up. If you have an electrocardiogram, you can flip it up and hold it up to the light if you wanted to with inferior infarction. Dr. Smith has emphasized this concept. Also, when you have an acute inferior infarction, there is this magical relationship between leads three and aVL. Now, if you think of it in the frontal plane, lead three is at about 120 degrees in the frontal plane lead. AVL is at minus 30 degrees. There are almost directly opposite. And with acute inferior infarction lead AVL often, almost always, I should say, manifest an mirror image opposite shape of the ST segment in lead three. So not only elevated, but if you've got coving, a little bit of curving and then an upright fatter than expected T-wave peak, you will see a mirror image opposite picture in lead AVL. Almost always. And this is helpful because oftentimes we ask ourselves, is this a normal repolarization variant that should not give you a mirror image opposite shape? Now you may normally sometimes get T-wave inversion in lead aVL. That's not what I'm talking about.

I'm talking about the whole ST segment, T-wave mirror image opposite shape in AvL compared to lead three. That's what I look for if I'm considering acute inferior omi. How about posterior myocardial infarction? Posterior omi? How do we determine this? And in my experience, this is one of the most, if not the most commonly overlooked diagnosis. And the reason it's so commonly overlooked is dependence on posterior leads. What you do is you could do a, you rotate the patient, you do a V seven, V eight, V nine, you almost have to have, you have to have the patient lying face down in the bed to get to V nine. And that gives you a look at what the posterior wall of the left ventricle might look at. But to get that view, how much of the thick back musculature does the electrical activity have to go through? And basically that's the reason if you look at posterior leads, sure they may show some ST elevation V seven, V eight, V nine, but the amplitude of this is a whole lot less in almost all cases than what you have with what I call my mirror test. With my mirror test. Basically I'm looking at anterior leads usually V two, V three, V four, I mean you could start out even without the mirror test, you could say if you have maximal ST depression in lead V two and or V three and or V four, if it's maximal in one or more of those leads in a patient with new chest pain, it's a posterior OMI until you prove otherwise. So you can say that. Now you have to keep in mind if the ST segments are depressed, not only in those V two, V three, V four leads, but diffusely, maybe the patient has what's known as diffuse sub endocardial ischemia. Maybe they have diffuse coronary disease, not an acute posterior OMI. Now they could have both. So just because they have a lot of ST depression in V two, V three, V four, they could also have severe coronary disease. The point I'm making is if it's maximal in V two or V three and or V four, then think posterior infarction. And if you have a positive mirror test, that is the shape of the ST segment in lead V two, V three or V four is the mirror image opposite of what looks like an acute infarction shape with Q waves and ST elevation. That's a positive mirror test. Okay, so that's posterior OMI. Now most of the time you're gonna see acute inferior MI when you have an acute posterior OMI. But sometimes you don't. You can have an isolated posterior OMI, in which case if you depended on using posterior leads, you're gonna miss it. You're gonna miss it because the amplitude is a whole lot less. And in my experience, I've seen posterior OMI in which my mirror test gives a positive diagnosis and you don't see anything in the posterior leads. Okay, we're up to dynamic ST and T wave changes on serial ECGs. So your patient has chest pain. The first ECG really doesn't show much. Maybe non-specific ST segment flattening, maybe a little ST segment depression, nothing overly remarkable. When you repeat the ECG, you now see that there has been some changes with respect to what that first ECG showed. This is data. You had a patient with chest pain and in front of your eyes over the minutes or longer you've had a change. Now let me emphasize, you know, when do you repeat the ECG? A lot of times people wait too long. If you have an active acutely evolving only, you may see changes not in an hour, not in 20 minutes. I've seen them in less than 10 minutes in five minutes, depends on the setting. So basically if your first ECG is non-specific, non-diagnostic, but you have a high prevalence situation, what we talked about, patient has what sounds like cardiac sounding chest pain. You wanna be repeating ECGs serially often until you know they are or are not having an acute omi. Now you can look at troponins, don't fall into the trap that the first troponin, even if it's high sensitivity, was normal. Because even acute OMI sometimes potentially evolving large OMI can have a normal high sensitive troponin for the first troponin result. So don't stop there. You want to keep doing things for your patient. You are clinically by the history suspicious of until you have a definitive answer. Okay, dynamic changes. So we can repeat the ECG, we can find a prior tracing. Now when you find a prior tracing, so often I have, here's the prior ECG, and my question is what was going on with the prior ECG? The person forgot to look, they forgot to look and see, oh the patient had their first myocardial infarction at the time of this prior ECG. That's not a baseline,

that's an ECG when the patients have an acute ischemia and an infarction. So it's good to get whatever you can for prior ECGs, but continue to look at the prior chart and find out what was the history at the time of the prior ECG that you're showing me there. The history. So often I see this is the first ECG, this is the repeat ECG. And I asked what was happening at the time of the first ECG. Was there still chest pain or was it that the patient had crushing the worst chest pain ever for three hours at home and then it went away and the first ECG was when the chest pain went away. That is critical. So I would suggest that you write down on the actual ECG what the history was at the time that ECG was done and put it in the chart because otherwise it's gone. And that is critical data and that is so rarely done. So that gets into the series of my next errors, not paying attention to serial ECGs. And this includes a series of problems that that occur. And the reason for this I think is not appreciating the pathophysiology of what happens with an acute omi, an acute occlusion myocardial infarction. 'cause the process itself is dynamic in probably over 90% of cases with acute infarction. There's an acute coronary occlusion, acute omi. So that's the first thing that occurs. Whatever causes that to occur, whether there was underlying plaque, how severe the plaque is, there's an acute occlusion of that vessel. Myocardial damage can be limited if we can determine what the culprit artery is. Do an acute cardiac catheterization and open by angioplasty within a timely period that culprit artery or institute if don't have access to prompt cath 24 7. If you do thrombolytic therapy in a timely manner, you can also open the culprit artery. So reperfusion could be accomplished either way by PCI or by thrombolytics. And if you reperfuse the culprit artery in a timely manner, then you can limit the damage. And what you'll find is as the vessel reuses, usually, I mean nothing's a hundred percent, you can even have acute infarctions without chest pain. But most of the time the patient's symptoms will decrease and usually, or often they will go away as the culprit artery is reperfused. So what happens to the ST segment changes if there was ST elevation at the time of the chest pain and the acute occlusion and you open the vessel as we talked about earlier, we said, well there could be this pseudo normalization stage as the ST segments come down to baseline. And then reperfusion is usually seen as T-wave inversion. Now it'll be the opposite for posterior infarction by my mirror test. Instead of I said there's st depression and the T wave, the inverted T wave is deeper. You're gonna see an upright T wave that gets taller with posterior reperfusion changes. So those are reperfusion changes. The history, this is the key point, can give you a key clue to whether or not there's been reperfusion. And the point that many people don't realize is, sure PCI on cardiac catheterization can reperfuse, thrombolytics can reperfuse, but you can often see this spontaneously before the patient gets to the hospital, I had terrible chest pain for three hours. It's gone spontaneous reperfusion, your body heals itself in a sense and can open up, maybe it's still an 80% lesion and it still may cause some future problems, but it's open and there's perfusion and your chest pain may decrease or even go away. And the ST segments go down. That is data that's often lost because people don't correlate each and every ECG to the history at the time that you're looking at it. So when I talk about dynamic changes, okay, the patient had severe chest pain at home, but it was gone by the time EMS arrived. So the ECG was non-specific, non-diagnostic, non-specific ST segment flattening at the time. First ECG is done, the paramedics get there and then you do another one that's in the hospital and the chest pain, it's coming back a little bit. And you look and the ECG might not be that dramatic until you compare it to the ECG that showed flat ST segments that are now starting to rise at the same time that the patient is starting to get new chest pain. This is dynamic. You don't have to stop for go, you don't have to get troponin values basically, you don't have to repeat cardiograms if it's a posterior infarction, you don't have to get posterior leads. You need to reperfuse that patient. Why? Well, what spontaneously occluded might just as easily spontaneously occlude. And sometimes what you have in the history in serial ECGs can tell you

this is sometimes you got a culprit vessel that occluded it opened a little, it occluded again, chest pain came back a little bit. ST segments started to go up. No, they went away. Now came back and forth and back and forth sometimes multiple times until finally the body adjusts at its final equilibrium, which if it happens to be with total coronary occlusion, you're in trouble. So that's why even when the patient says, Hey, I feel better. I'm in the emergency room, you tell me my cardiogram now looks normal, I'm going home. How often have you seen that scenario? And that's a patient who needs to go to the cath lab. And sometimes often in my experience from following all these cases, both on Dr. Smith's ECG blog and the internet with cases I've seen is those patients are not treated. Okay. They're called a non stemi, maybe you missed the ST elevation in the first place. So those are patients that need to be reperfused to prevent spontaneous reocclusion. I'm down to my last little point just about done with covering this topic. The last point that I would bring up is not learning from our cases. And we all know this, from whatever you're training, if you're a paramedic, nurse, clinician, cardiologist, emergency physician, you gotta get follow-up. Now I understand, and this happens on the internet all the time with paramedics, they don't see the patient again. But if it's possible to get follow-up next time to go to that hospital, ask the clinicians, see if you are able to get from the chart. Get follow up. This is how I learn. So this is the first point that Anthony had asked me, how did I get to be so good? Well, I followed everything up in the days when digoxin was used, I would follow up serum dig levels to what the ECG looked at. I would follow up serum potassium levels as the patient was treated and the hypo or hyperkalemia was corrected. What happened to the ECG as the arrhythmia was treated? What happened to what I thought was occult retrograde P waves on the electrocardiogram. This is how we learn. Now sometimes I learn from mistakes. Hopefully I didn't make too many mistakes that costs were too costly. But you gotta learn from what we're doing. That's the reality. After the fact, after the case, go back and look at things. And this is particularly true with the ECG interpretation. I'll give the example of a patient I'll never forget with lung cancer, this was an outpatient unfortunate case and basically, you know, the patient ended up having this severe lung cancer and I had done a chest x-ray, it was I think a year or something ago. And I went back and I looked at the X-ray after I found out he had this terrible spread of his lung cancer. And I felt at least a little bit better that his lung cancer was not obvious to me or to other providers a year ago, but, but knowing that this is the area that he subsequently developed this huge cancer, I say, yes, there is something there and you can do the same thing with an electrocardiogram. So I think learning from our cases, painful as it may be, that's how we get better.

Dr. Anthony Kashou: That was amazing and thank you so much. There's so many key points and main points that you've highlighted and we've highlighted really key aspects of 12 lead interpretation. We talked about acute myocardial ischemia and really focused on acute coronary occlusion. We conveyed the concept of occlusion, myocardial infarction or OMI emphasizing appreciation of how the STEMI paradigm can miss at least 30% of acute coronary occlusions then could be picked up using this OMI OMI paradigm. Furthermore, we cited ECG findings and Dr. Grower took us through a number of them. How do we recognize hyperacute T waves? Look back, make sure to look at that in relation to the QRS complex. Look for the mirror image STT wave changes that can be seen as reciprocal changes as you mentioned posterior MI and watching out for those optimizing use of serial ECGs, making sure how to correlate not only the ECG and when it was taken as a baseline, but those correlated symptoms at the time. Dr. Grauer, there was so much that you talked about us here and I'm really enjoying this and we've gone through two episodes and hopefully those will go back and listen to the previous one where we

talked about common errors. We're so grateful for your ongoing support, your countless contributions to the field and so much that you continue to do for us learners. We hope you'll join us again.

Dr. Ken Grauer: Thank you so much, Anthony, be my pleasure to join you again.

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