Announcer: 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 explore the importance of clinically correlated ECGs, including the optimal techniques for comparing ECGs, the role of clinical history in this process, and the applications of comparing tracings for cardiac arrhythmias. We're fortunate to have Dr. Ken Grauer back with us today who is Professor Emeritus in Family Medicine following his residency training and family medicine. He worked for two years in a busy emergency department in South Florida. Then he moved to Gainesville, Florida where he was full-time faculty at the University of Florida's Family Medicine residency program and he spent 30 years there until he retired in 2010. Dr. Grauer has written well over 10 books on ECG and arrhythmia interpretation. He's an expert teacher. He's presented hundreds of talks in workshops locally and nationally on ECG interpretation and other cardiology topics. And he's been active as ever since retiring. He hasn't stopped. He continues his own ECG blog. You better go look for Dr. Grauer's ECG blog. I learned so much on that. He's the associate editor, one of them and contributor to Dr. Smith's ECG blog and he continues to answer many questions on international forums. My emails, my questions, he's nonstop and we're so grateful to have Dr. Grauer back with us today.

Dr. Ken Grauer: Thank you Anthony.

Dr. Anthony Kashou: Thinking about all we've looked at, we've talked about common errors in a previous podcast. You know, we recently talked about AI and what that field looks like. You know, today a really important skill that I, I wanted to discuss with you because you do this and you answer so many questions for so many of us. What can we learn from comparing one ECG to another and you know, what is the optimal technique because you do this so much, how do you make it look so easy? And what is your approach?

Dr. Ken Grauer: Well, from what I've observed, potential benefits of comparison tracings are not optimally appreciated by all too many clinicians of all experience levels. And even when clinicians do appreciate the benefits of comparison tracings, their technique for comparison is lacking. So to note this applies both to the use of comparison of 12 lead ECGs as well as the use of comparison of tracings for arrhythmia detection. And let me start first with comparison of 12 lead ECGs. As to the benefits of comparison of 12 lead ECGs, I think of two types of comparison tracings. The first type is with a prior 12 lead tracing on the patient, done at some time in the past. And the second is with serial ECGs. And most commonly this is gonna be done on the patient who presents with new symptoms, new chest pain when you're trying to assess for acute coronary occlusion. So key point for both types of 12 lead comparisons. What are you comparing? And by this I mean if you're using for example, prior tracing, how long ago was the prior tracing? So often I see people say this is a prior ECG that I found in the chart. And this is

especially important if you're assessing a patient with new chest pain. I've seen clinicians compare a quote baseline tracing from five years earlier with the initial ECG of a patient with new chest pain without any mention that since this baseline done five years ago, the patients had a myocardial infarction. Or perhaps that at the time that this comparison tracing found in the chart was done, the patient was also having acute chest pain. So this is essential information that so often is overlooked and then you're trying to compare things. And when he used to when he or she used to have chest pain five years ago and is now having chest pain, you see st elevation on both of them. No change when in fact it's a very different situation. So now to fully acknowledge, oftentimes it is not easy to figure out what was going on at the time of the prior tracing. If you don't know say. So if you do know when it was done, say so. But if you have an extra moment and you have the clinical chart available, take a look, try and figure things out. Get an idea as to what was going on at the time of the last tracing and writing it down. Because it's essential to know if there's an acute change. I often suggest writing on the chart or writing on the actual ECG, what's going on. 'cause if you don't write it then you're never gonna write it and then it's hard to put it together after the fact. Now regarding serial ECGs, as I mentioned in my podcast talk on omi. In a patient you are evaluating for new chest pain. You wanna know if the culprit vessel is open or closed at the time you're looking at the patient at the time that a given ECG was done. So it's essential to write this information down somewhere, either on the ECG and or on the chart, the time each ECG was done and whether or not the patient did or did not have chest pain. And whether or not the chest pain was increasing, decreasing or the same, a scale of one to 10 is probably the easiest way to do that. That's because if chest pain is decreased or gone at the same time that the ECG shows that the ST elevation you saw an hour ago is now gone, this tells you that the culprit vessel has now opened. But if the patient's chest pain returns at the same time that the repeat ECG shows ST elevation and reciprocal ST depression and other leads at the same time it shows this is returning, then there has been occlusion. And this is fundamental, it's the basic pathophysiology. And I can't tell you how often it gets ignored by everyone including unfortunately lots of cardiologists clinically critical information. So how many times have clinicians said all is well, I mean the patient had chest pain but now when they're being seen chest pain's gone and the ECG shows some non-specific ST segment flattening. So nothing needs to be done. So the clinical point is what's our goal? Our goal with patients with new symptoms who we are evaluating for possible acute myocardial infarction evolving for possible acute coronary occlusion. Our goal is to see if there was or was not acute occlusion. And if so, we wanna open the vessel. We wanna salvage as much myocardium as we can by opening the vessel as soon as we can. And we can do this with PCI. We can do this with thrombolytics if you don't have PCI available. But the point to emphasize, and this is still overlooked by too many folks, is that coronary vessels, it's not an all or none phenomenon and that there can be spontaneous reperfusion. The body on its own can help generate processes that can spontaneously open a previously occluded vessel. And with some patients this occurs on and off over a period of time until ultimately you get the final state of affairs. So if you have somebody with acute chest pain, ST elevation, acute occlusion, and then on the way to opening the vessel and having that ST segment elevation evolve to deep T wave inversions of reperfusion T waves, what happens? Well there is a pseudo normalization stage in between ST elevation and T-wave inversion. So the ECG may look non-specific and clinically you can tell this and this is just so essential and it's the fundamental principle of comparison tracings. So write down indicate whether the patient had chest pain. Look at the comparison tracing. Just because the chest pain is gone and the ECG looks non-specific at the time you look at that first tracing doesn't mean that everything is well and what spontaneously reopened might just as easily spontaneously occlude. So those patients still need an intervention, be it thrombolytics and

or ideally prompt cat with ECI. Okay, how often then should you be repeating the ECG in a patient with new chest pain? And the answer, my answer to this is as often as you need to repeat the ECG until you become certain of what your diagnosis is. Now you may become certain before you can convince your interventionist that they need to take the patient to the cath lab. Depends on what criteria they are using. So realize a couple of things. Number one, first of all, even if the ECG never shows any acute changes, if the patient has acute ischemic sounding symptoms that persist, even if the ECG is unremarkable, that in itself is potential indication for taking the patient to the cath lab. They shouldn't have that unrelieved symptoms. And that's another reason for not going ahead with morphine until you know what you're gonna do with the patient. Okay? The other point is remember that an acutely evolving event may evolve quickly. I mean sometimes it takes an hour for the s st segments to go up, but I've seen a bunch of cases within minutes and sometimes within less than 10 minutes. So a couple of points. First, if your symptoms, if your symptoms change at all in someone you're evaluating for potential acute omi, repeat the ECG, repeat the ECG every few minutes in someone you are concerned about until you have a particular answer. So repeat that first. ECG if you're uncertain within 10 to 20 minutes, not one to two to three hours as all too often is the case. You wanna repeat the ECG and repeat it frequently until you have a particular answer. And if you have changes that that correlate with what we've just talked about in terms of the presence and relative severity of chest pain and association with what the ST segments and t waves are doing at that particular time, if that changes, those are dynamic ST and t-wave changes in a patient with new symptoms, that in and of itself is an indication for taking the patient to the cath lab. So what it then is the optimal, the quotes around optimal technique for comparing ECGs. And I would say that in my experience of overseeing this for decades, the most common era I see by far is that clinicians are in a rush. So they look at one ECG and it doesn't look like much. There's just some non-specific ST segment flattening, maybe a tiny bit of ST depression, nothing to write home about. They look at one ECG in its entirety and then they look at the other ECG in its entirety and it also doesn't look like much. So they say there's no problem with that, but they are not going lead by lead. And I can't tell you how often when if you looked at one ECG in its entirety and then the other in its entirety, how often you're gonna miss things instead of going lead by lead by lead, by lead. I mean look at lead one on one tracing, then look at lead one in the other tracing, is there any change? I'll often look at the same areas together. I'll look at the high lateral leads one and AVL together on one tracing, and then I'll look together on the other tracing. And then I'll look at two, three AVF, the three inferior leads at the same time on one and then the other tracing. And you'll pick up differences much, much better if you do th that way than if you're just looking in its entirety and it does not repeated, it does not take more time, probably takes less time because you don't have to keep going back trying to remember what you looked at. You're going lead by lead. Now I have an expression that I like. Are you comparing apples with apples or apples with oranges? And basically what I mean by this is many folks are unaware of the fact that if you change the elevation of the bed, you might affect the appearance of the frontal leads. And that's been shown. So your patient has acute heart failure, they can't lie flat. And then later on they're doing better 'cause they got diuresed or whatever and now they are lying flat. That may affect the frontal plane axis. So another point, as an aside, if your patient's at 40 degrees when you do the first ECG, write that on the chart because that might explain why the frontal plane axis is different. Now when you're comparing one lead to the other, you wanna know two things. One frontal plane axis, apples with apples. If you've got the same or similar frontal plane axis than any small change in ST segments and T waves elevated or depressed between one tracing and the next is significant. If on the other hand there's a change in the frontal plane axis for whatever reason, then it's harder to compare and you must use

your overall gestalt. But you can't necessarily say, I mean what you want to exclude is that the change in frontal plane axis rather than ischemia is the reason for the difference. So you wanna be looking at that. And I like it when the frontal plane axis is the same 'cause then I can really hone in. Same thing with precordial lead placement. Say the excellent EMS squad did an ECG and then your excellent emergency department technicians did an ECG, but they're different people. So and we've shown this, I mean it's amazing how precordial lead placement and we, we looked at examples of excellent quotes around it. Precordial lead placement on the internet that showed precordial lead placement that was far from being accurate. So basically you wanna be sure that our wave progression is similar. And if it is, great then small differences in ST segments and T waves are very significant. And if it's different then you have to take that in account. Doesn't mean that you can't compare but you, it's not a strict comparison. So apples with apples versus apples with oranges a word about artifact. So I call these technical misadventures and there are really all too common. I don't have time to go into all of them at this point, but I'll just say in my experience they often go unrecognized. So a couple of points. Number one, as a general rule, if the ECG complexes of a particular tracing look funny, they look odd, unusual, unusual shapes, they look geometric or too rounded or something. Or for example, you have a, one of the standard leads lead one, two or three is a flat line. That's not normal. And what I've seen all too often is even when people say, Hey, that's a funny looking complex, but I just did the ecg, I'm not gonna repeat it. You are evaluating the patient in a multi-thousand dollar workup and evaluation for acute chest pain. If you think there may be artifact, check the leads and immediately repeat the ECG. 'cause otherwise you cannot have a valid assessment of those leads with potential artifact. So you wanna validate that and repeat the ECG if there's a potential problem with that. Okay, another point lead one, lead one should never be all negative T-wave QRS and T wave or the QRS. I mean you may have a Q wave in lead one, but if you have a predominant Q wave and the P and the T are also negative, that's not normal other than rare Dextrocardia, that's almost always some form of lead misplacement. I'm just gonna mention one type of artifact that we've been seeing more of. Now it's interesting when I say this is a medical phenomena that in recent months or years quote we've been seeing more of. It's not that all of the years of medical history, it never necessarily occurred. It could be we just didn't know about it. One of my favorite talks that one of my colleagues used to give every year in family medicine was five things that I wish I knew last year. I mean I learned all the time with things I've learned so much from being exposed to things. This is pulse tap artifact that I'm sure was around for many, many years. But until the past couple years for me, I never realized it exists. And now we're seeing it. Why? Huh? 'cause we're realizing it and we're realizing how to recognize it. Pulse tap artifact. So it's a good thing to be aware of. If you have one of the four extremity electrodes that come into a good or a strong contact with a pulsating artery underneath where the electrode lead is placed, you may get artifact from that. And it's interesting with this artifact it's gonna be one of the four leads, okay? And it's gonna be with a fixed relationship to the QRS complex because it's related to the pulse. And you're gonna see a usually geometric like unusual, bizarre. Although I've seen some of these that for all the world look like while there's ST elevation, tremendous ST elevation in two three A VF, it looks kind of bizarre. But there it is. But it's a little bit rounded and it turns out to be pulse tap artifact. So you wanna be aware of it. And there are a couple of easy ways to recognize this. Number one Einthoven's triangle, more than a hundred years old now gave us some mathematical relationships of the electro leads of how we do an electrocardiogram. So what you see if there's pulse tap artifact, which affects one of the four extremity electrode leads, is in this three standard limb leads, leads one, two, and three. Two of those three leads are gonna have maximal and equal artifact of a geometric looking complex. And the third is gonna look normal. Why?

Einthoven's triangle. So if it's the left foot that's involved, you're gonna see maximal artifact in lead two and three. That looks a little bizarre and you're gonna see a flat line in lead one. It's the flat line and one of the three leads that says, hey, what's going on? And then if you look in the augmented leads which are derived, it turns out, and this is the easy way to recognize what's the culprit extremity makes you look really smart within two seconds, that's pulse tap artifact and it's that augmented lead that shows maximum artifact almost equal in amplitude to what you see in leads two and three here. If you see it in AV F, you can immediately say it's the left foot that gives this and the other two augmented leads because of the geometric relationship Einthoven's triangle show approximately half that amplitude of artifact. So those relationships allow you to recognize this immediately. That's 12 leads. What I wanna do now is briefly look at cardiac arrhythmias add. A lot of times we say comparison tracings, we don't think of cardiac arrhythmias, but there are some specific very useful ways of employing repeat or serial or comparison tracings comp compare comparison rhythm strips, depending upon what you are looking for with cardiac arrhythmias. So let's assume that for your single or double lead rhythm strip, you've already gotten a 12 lead. Okay? Advantages 'cause a lot of times I used to see this when I would stroll around the hospital and make my early morning 5:00 AM rounds 'cause that's the fastest way to make rounds. And I'd have telemetry folks give me all of these tracings to look at. They'd give me an unusual single lead rhythm strip. I'd say show me the 12 lead. So if you're just looking at rhythms in a single lead of repeat or comparison tracing is to get the 12 lead, which gives you 11 more chances to find atrial activity to tell. Is the QRS really narrow or am I seeing one of the leads that's on the baseline so that it looks narrow, but in reality is wide. I wrote a chapter and one of my earlier books, 12 leads are better than one. Just to show people, hey you look at the single lead, it's a narrow qrs. No it's wide. You're looking for super ventricular arrhythmias. Now the best lead for looking for atrial activity is lead two. Because if you see an upright P wave, particularly with a constant PR interval in lead two you've got a sinus rhythm. The second best lead for looking for atrial activity tends to be lead V one, which anatomically is located over the atria. And then I find my other three quote, go, go-to leads are leads three AVF and aVR as other leads where it's often really helpful, particularly with flutter activity leads three and AVF, oftentimes these right-sided leads give me the best indication of flutter activity. And then I look at the other seven leads. But those are my five leads, V two V one, three AVF and AVR. The other advantage of always getting a 12 lead for a problematic arrhythmia that you're not sure what's going on is you've got 12 leads to tell if the QRS is wide or narrow. If you're dealing with super ventricular activity, you wanna look for atrial activity. So we talked about the best leads to have and you might get a single 12 lead and you might not be sure of what atrial activity is doing. And practically speaking, if you are in an emergency setting and you have a regular supraventricular tachycardia, it's a narrow QRS and you're not sure what atrial activity is doing, practically speaking, your initial treatment and evaluation measures gonna be fairly similar. If you're in the emergency department, you might use adenosine, you might use vagal maneuvers both or some other AV nodal blocking drug depending upon where you are, be it verapamil, diltiazem or beta blockers. I think it helps if you can figure out what the rhythm is. So if we think about a regular SVT rhythm, and let's say the rate is about 150 or 140, 160 a minute, so you cannot tell for sure what's going on. If I got a regular SVT and the rate is 200, that's too fast for sinus tachycardia, that's too fast for two to one atrial flutter. But if the rate is 150 we're thinking of four basic items, sinus tach, atrial tachycardia, the most commonly overlooked SVT regular SVT is atrial flutter. Or it could be a reentry SVT such as A-V-N-R-T or A VRT if there's an accessory pathway. What is atrial activity doing? So if it's a reentry, supra ventricular tachycardia is there retrograde atrial activity. And you can tell this on a 12 lead and you might be able to tell this by comparison. ECG, you can get a prior 12 lead ECG during sinus

rhythm and you look at the QRS complex and then you look at the QRS complex during the SVT or if you successfully treated the SVT and you wanna know after the fact what was the rhythm? 'cause you should always be getting a post-conversion sup, ECG to tell what atrial activity was doing was there retrograde atrial activity. So you should see a negative deflection in the inferior leads if there's retrograde atrial activity and you can tell regarding what we call our key prime intervals. How far is this negative deflection from the preceding QRS complex? You tend to have the typical AVNRT that's a reentry supraventricular tachycardia contained totally within the AV node, which means that there's a short distance to travel over the other pathway and therefore you have a very short retrograde P wave that tends to notch the end of the QRS complex. Or if you have a negative P wave that has a slightly longer RP prime interval, it occurs within the ST segments that often suggest you're dealing with an accessory pathway because that's outside of the AV node takes a little bit longer. Or if you have the uncommon, what we call fast slow reentry, in which case the reentry impulse is traveling backwards over the slow pathway and therefore you have the negative deflection really with a very long RP prime interval that almost occurs near the next P wave would normally be. So retrograde atrial activity can give you a clue to what you're dealing with. Let me finish up by looking at regular wide complex tachycardias. How does a comparison ECG help you with this? So we could take the simple approach patient presents with a regular wide complex act, excuse me, with a regular what I call WCT or wide complex tachycardia. We all know that if the patient's at all unstable shock first, then figure out what the rhythm is afterwards. 'cause it doesn't matter if it's super ventricular WPW related or ventricular tachycardia, if they are unstable because of, because of a wide complex tachycardia, you've gotta give electricity basically. But I think it helps particularly with acute management with management over the next bunch of hours long before you do an EP study and with perhaps long-term management. 'cause some of these patients might be discharged and managed in an outpatient setting after their emergency department presentation. They do not all need to go to EP after their first presentation. It depends on what the particular rhythm is. So you wanna know, was this a super ventricular tachycardia due to either a preexisting bundle branch block or rate related to aberrant conduction or is it vt? And if it's vt, is this an idiopathic VT that's an otherwise younger adult without heart disease versus an ischemic ventricular tachycardia in a patient that does have underlying heart disease? And sometimes you look at the wide complex tachycardia and you can't tell for sure despite all of the criteria that you're looking at. So what can we do? How can comparison tracings help you? Well, if you have a wide tachycardia that looks totally typical for some typical form of bundle branch block, a totally typical in all 12 leads, left bundle branch block tracing or right bundle branch block tracing, you know, then it's more likely to be something aberrant or preexisting bundle branch block. And if it's an ugly looking QRS, then it's more likely to begin from the ventricles of BV tac. Unless say the patient used to have a left bundle branch block and then they had a couple of infarcts on top of this, then they are nice, neat left bundle branch block might have Q waves, unusual QRS deviations with this. So you wanna look to see if there's a prior 12 lead ECG assuming you have time to do so. You might not if the patient's crashing in front of you, but once you have a moment of time, it helps to know was there an identical looking QRS complex 12 leads in past? Say you don't have that, you shock the patient, they're out if it. If you wanna get a post-conversion tracing, now you wanna look and make sure the patient didn't have an acute OMI as the cause of their ventricular tachycardia. And the other thing that's often ignored is say they were in this wide tachycardia and you get 'em out of it into a narrow complex rhythm in which you see normal sinus P waves and then you have a couple of wide beats, particularly if these wide beats do not have a premature P wave in front of them, they're PVCs. So you wanna look to see if in all 12 leads, the morphology of these post-conversion wide premature beats

is the same as the QS morphology during the regular wide complex tachycardia. And then you can know, hey, this was ventricular tachycardia the whole time.

Dr. Anthony Kashou: Well, Dr. Grauer, that was incredible. Another clinic, I feel like that's one I can certainly listen to. Again, it, it's just really, I enjoy listening to you and you could tell we don't even have an EKG in or an image you're putting up in the way you explain it. It's just so natural. Thank you so much. In this episode, we explored the significance of clinically correlated ECGs examining the best practices for ECG comparison. We showed how Dr. Grauer does it lead by lead. Make sure you're doing that. We underscore the pivotal role of clinical history. The history matters, especially with the baseline ECG. What was the current state when that ECG was recorded, we highlighted the applications of comparing ECGs, both the 12 leads and then we looked at cardiac arrhythmias. Dr. Grauer again, what a clinic you just put on for us. We really appreciate your unwavering support, your numerous contributions to the field, to myself, my own learning. Thank you so much for being with us today and I hope you'll join us again.

Dr. Ken Grauer: Thank you, Anthony. My pleasure.

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