PVC Localization and Management

Announcer: Welcome to Mayo Clinic's ECG Segment: Making Waves, Continuing Medical Education podcast. Join us every other week 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. Kashou: Welcome to Mayo Clinic's ECG segment: Making Waves. We are so glad you could join us. Today we have a fascinating episode planned for you as we discuss premature ventricular complexes or PVCs. It is perhaps one of our more practical episodes. We're fortunate to have an experienced and expert clinician in this field to discuss and help us better understand this important clinical topic. So let's get started. Premature ventricular complexes or PVCs are commonly encountered in clinical practice, especially in cardiology. While many can be benign in nature, it's important to be aware of cases that might be more concerning for the patient. In this episode, we're going to discuss how to identify alarming features when asked to evaluate PVCs, what ECG features can point us to underlying structural heart disease or an increased risk, which patients warrant a trip to the electrophysiology or EP lab, and how to use the 12 lead ECG to locate PVCs and integrate such findings during mapping in the EP lab. We'll be discussing all this today with one of the leading experts in the field. And with that said, let me introduce you to our special guest today, Dr. William Stevenson. Dr. Stevenson is Professor of Medicine at Vanderbilt University Medical School. He received his medical degree from Tulane University in Louisiana, and he completed his medicine in cardiology training at UCLA Center for the Health Sciences. In 1993, Dr. Stevenson joined the faculty at Brigham and Women's Hospital, where he became Professor of Medicine at Harvard Medical School and the Director of the Cardiac Arrhythmia Program. In September of 2017, he was recruited to Vanderbilt University Medical Center in Nashville, Tennessee. Dr. Stevenson specializes in the diagnosis in management of cardiac arrhythmias and prevention of sudden death. His research focuses on the treatment of complex heart rhythm disorders with catheter ablation, his methods of cardiac mapping incorporating entrainment mapping to guide catheter ablation are widely used today. Dr. Stevenson has published well over 300 articles and chapters. He has supervised post-doctoral trainings of over 50 cardiology electrophysiology fellows, and he's the co-director of the longstanding annual advanced ablation course. He's the chair of the International Advisory Committee to the Canadian Arrhythmia Network, and he is co-editor to the text Cardiac Electrophysiology: From Cell to Bedside. We're so excited to have him. I just wanna mention some of his awards that he's also earned, and this is only a few. Dr. Stevenson's earned the Heart Rhythm Society, Distinguished Teacher Award, the Michelle Moroski Lectureship Award from John's Hopkins Medical School, the Prystowsky Lectureship Award from the Heart Rhythm Society and so many more. We're so excited to have him. And I wanna mention for those that are in the field of research, he is the Founding Editor of Circulation, Arrhythmia, and Electrophysiology of the American Heart Association. Dr. Stevenson, what a true honor, there's so much more we could say about you, but thank you so much for joining us today.

Dr. Stevenson: Well, thank you very much, it's a pleasure to be here and thank you for that warm introduction.

Dr. Kashou: No, there's so much more, like I said, we can mention of all your work throughout your career and we're so lucky. And what I wanna get into is the field that, not only you spent a lot of time in, but you've essentially mastered and have taught at least me so much from your prior lecture here at Mayo Clinic, but maybe you could tell us a little bit about when we think of premature ventricular complexes, what are things that should be more initial concerns when we're consulted on a patient for this issue?

Dr. Stevenson: Yeah, so premature ventricular beats can be the initial manifestation of underlying structural heart disease, which is probably the major concern. Although most commonly, when we see that patient in the clinic with PVCs they're idiopathic or not necessarily associated with structural heart disease. So, sorting that out, who's the patient at risk is where this is a sign of underlying structural heart disease versus those without structural heart disease. And then we need a third category, which is the patient who doesn't have structural heart disease, but has some sort of electrical or ion channel disease, which puts them at risk. So that's our initial consideration when we approach a patient, who's found to have premature ventricular beats.

Dr. Kashou: Really interesting. And so how do you put them in those buckets, to make sure if I get consulted on a patient, what are more of those alarming features I should be keeping in mind? You know, you mentioned the structural heart disease or those that increased risk. What should we be thinking about?

Dr. Stevenson: Yeah, so we start with a systematic approach. So, the history, does a patient have any arrhythmia symptoms? Certainly if they've had palpitations that might be benign. If they've had syncope, that's a big red flag or episodes of sustained rapid palpitations with symptoms and then any other symptoms that point towards underlying heart disease. So, exertional dyspnea, angina. So we start there. Then our family history is extremely important for assessing the risk in a patient with PVCs, family history of sudden death can be a sign that there's an underlying predisposition to heart disease, such as coronary artery disease, or it may be a sign of an underlying genetic abnormality present in the family. And the genetic abnormality may be associated with structural heart disease for example, a genetic cause of nonischemic dilated cardiomyopathy is very important to consider. And PVCs are sometimes the initial manifestation of genetic cardiomyopathies of patients who we see who have a non-ischemic cardiomyopathies. So, no coronary artery disease have PVCs for which they're referred to as and have ventricular dysfunction with some degree of left ventricular dysfunction, 30 to 40% of those folks will have an abnormal gene identified if we do genetic testing. So this has become a very important group of patients and we're beginning to better characterize the prognosis of those folks as well. Then the physical examination focuses on any cardiac abnormality. So, if they have a murmur, perhaps this is the initial presentation of aortic stenosis or hypertrophic cardiomyopathy or mitral valve prolapse. So any physical exam abnormality involving the heart or cardiovascular system can be a clue. Some of the genetic cardiomyopathies are syndromic and have other manifestations. So, neurologic manifestations, for example, with muscular dystrophies that can also affect the heart. So, a good physical examination is important. And then the next thing that we look at after that is we scrutinize the electrocardiogram and we look not only at the PVCs, but also the sinus rhythm conducted beats. Are there any abnormalities on that EKG? Is the QRS complex completely normal? Is repolarization completely normal? Does any abnormality suggest the possibility that this is an underlying cardiac disease rather than an idiopathic PVC? And then finally, after all of

that, then we get to looking at the PVCs and the most common location for idiopathic PVCs is up in the outflow tract of the, either the left ventricle or the right ventricle. And so those PVCs, and we'll get to this a little later, have a characteristic ECG morphology. If we see PVCs that are not in the outflow tract, then we worry a little bit more about structural heart disease, particularly if the PVCs come from the left ventricle. And if there are multifocal PVCs, PVCs that have multiple origins, that's more likely to be associated with underlying structural heart disease. So, our initial evaluation focuses on those aspects and often provides a clue as to how worried we should be. Then the next step, once we've done that, just about everybody who presents with PVCs warrants some cardiac imaging, and it's easiest probably to start with an echocardiogram and that'll show us if there's any ventricular dysfunction, it'll show us if there's mitral valve prolapse, any valvular abnormalities. If we're a little more worried than that, or if an echocardiogram has been say borderline in that perhaps it shows a question of an abnormality or maybe mild or upper normal ventricular left or right ventricular size. An MR cardiac study is very useful, very sensitive for displaying areas of fibrosis manifest as regions of late gadolinium enhancement. And most cardiomyopathies that are associated with ventricular arrhythmias often will have areas of fibrosis that are detectable on MR imaging.

Dr. Kashou: And so it's interesting. It's almost like what we learned in medical school, from the history of the physical and then the diagnostic testing, they're all important in the evaluation of are these actually concerning things that we should look out for from the ECG and the imaging? And forgive me, but how about the Holter more prolonged, You know, we hear about PVC burden, is that something you do before after the ECG, before imaging or how do you use that in your evaluation?

Dr. Stevenson: Yeah, so long term recordings of the rhythm are very important, particularly if the patient has symptoms that are intermittent and you want to see if the symptoms correlate with some arrhythmia, then ambulatory monitoring, and the question there is well for how long? If the patient has symptoms every day, well, you can probably do a 24 or 48 hour Holter, if they have symptoms several times a week, you're likely to catch it in monitoring for a week or so, if the symptoms are less frequent than that, you may have to try and do a longer period of monitoring. And then the second thing you get from the ambulatory monitoring is the density of the arrhythmia. So we're talking about PVCs and it used to be 20 years ago or so, if I saw a patient in the office with PVCs and they were mildly symptomatic and they had normal ventricular function and no other features suggesting an underlying disease process and the benign family history, I would reassure them that, oh, we don't need to worry about this, come back if they start to bother you more, and there are things that we can consider doing, but now we recognize that some people will develop a decrease in their ventricular function over time, which we feel is likely related to the frequency of the PVCs. And it usually takes more than 15 to 20% burden of PVCs. So if you figure that on average, we have about 100,000 heartbeats a day. So somebody who has more than 15 to 20,000 PVCs on their 24 hour ambulatory monitor, we get a little concerned that that person is at some risk for developing ventricular dysfunction over time. And the risk is probably in the range of 10 to 15% of patients over five years, but the data are somewhat limited. So, that ambulatory monitoring, if you get somebody who has got more than 10% or so PVCs, we do worry a little bit, this is someone that probably should be followed longitudinally for development of ventricular dysfunction if they don't have any evidence of ventricular dysfunction at this point, and who may warrant therapy down the road. So, even if a

patient is asymptomatic, if they have enough PVCs in your office, that you're seeing several on a 12 lead electrocardiogram in that ten second span, they may have a pretty good density of PVCs and some ambulatory monitoring to quantify that and make an assessment for how detailed their follow up needs to be is quite reasonable. And then the third thing that you might see on monitoring, which we usually don't see is something scary, right? So, we're talking about PVCs here, and those usually are pretty benign, but PVCs that are associated with non-sustained VT, which is unusually fast, more than 200 beats a minute, polymorphic and fast. Those can be signs of potentially life threatening arrhythmia that may initiate ventricular fibrillation. And while it's very uncommon to encounter that on routine ambulatory monitoring, if you did see it, it would make you very concerned regarding the possibility of an underlying genetic process that may be associated with sudden death or myocardial ischemia or hypertrophy or some other underlying process that puts the patient at increased risk.

Dr. Kashou: You know, and so we're looking at the kind of the evaluation of who's at increased risk in all these almost adding up to that pretest probability of who should we be concerned about from the history, the physical, the family history, some of the diagnostic testing, including the ECG imaging, and Holter, I guess when do you pull the trigger to who warrants that trip to the EP lab for assessment or ablation? When do you make, how do you make that call?

Dr. Stevenson: The electrophysiology lab is most useful in this situation for treating the PVCs. So somebody that has symptomatic PVCs and they want them gone, usually we will have started with a beta blocker, something very safe and easy, and which will not abolish the PVCs, but may diminish the symptoms to the point that they're quite manageable. And if that fails, occasionally a non dihydro purine calcium channel blocker will be helpful, not very often, but occasionally, and it's something that's very safe that can be tried. If those two things don't work, then you're two membrane active antilithic drugs, such as flecainide and propafenone the sodium channel blocking drugs that can be very effective at suppressing PVCs, but which we don't like to give to patients with structural heart disease, because we know that when they were given to patients who'd had prior myocardial infarction, that it was associated with an increased risk of death. So, usually if I've tried a beta blocker and maybe a calcium channel blocker, and that hasn't alleviated symptoms, I'm usually having a discussion about the risk benefit of catheter ablation of the PVCs with the patient. If there's any degree of ventricular dysfunction, and we think it may be related to the PVCs, my preference is ablation for those rather than trying to suppress those with an antirrhythmic drug for two reasons, one is then I avoid the toxicities of antirrhythmic drugs in case there is underlying structural heart disease, and secondly, at the time of the EP study, we'll also do programmed ventricular stimulation to determine whether there is any VT, which is inducible, which would be a sign that there is likely an underlying structural heart disease, particularly if there's an inducible VT with a morphology, that's not typical of an idiopathic ventricular tachycardia. So, a patient who has underlying structural heart disease and PVCs, or where we're concerned about it, someone that has late gadolinium enhancement in an area on their MR imaging there, I do worry, are they at increased risk? Do they have the potential for scar related reentrant arrhythmias? And we can try and sort that out with program stimulation in the electrophysiology laboratory. This has not encountered all that commonly in large series of patients it's in the order of less than 10% of your PVC patients will be found to have a scar related reentrant VT, those who are selected to come to the electrophysiology laboratory, but it's clearly a very important group because they are at risk of having sustained VT and sudden death.

Dr. Kashou: And so you think of, you mentioned against two groups, the structural heart disease that could be and often are at more of that, that increased risk if they have this burden and those refractory to medical therapy, is there ever the case where say you have, say a young person, maybe a family history, the high pretest probability that maybe there's some channelopathy or ion disorder, do you ever go from that to be skipping the medical therapy to right to the EP lab, is there an indication for that?

Dr. Stevenson: Yeah, occasionally, so that can come up with some of the ion channelopathies, you know a lot of those aren't particularly PVC producing interestingly, but things that we can potentially discover in the EP lab, one of the things that comes up is Brugada syndrome. So, most patients with Brugada syndrome, PVCs is not a prominent part of their clinical picture, but occasionally they do have some and if you have an EKG, which is suspicious, but not diagnostic for Brugada syndrome, then giving a challenge of a sodium channel blocker, we usually use intravenous procainamide and would prefer to do that in the electrophysiology laboratory. Also most patients who have Brugada syndrome who have had a cardiac arrest, so, not the patients we're talking about, but the ones who've had a cardiac arrest, most of them will have inducible polymorphic VT VF in the EP lab. And so while that finding of inducible polymorphic VT VF is controversial in terms of its prognostic significance in this situation, it is one more thing that makes me more worried about that patient with Brugada pattern. And here we're talking about largely the asymptomatic patient where trying to decide what to do with this abnormal EKG. The other thing that comes up is arrhythmogenic right ventricular cardiomyopathy. So now there, those folks often do have PVCs. Most of them will have some subtle EKG abnormalities with T-wave inversions in the anterior pre cordial leads V1 to V3, and the disease can have mild involvement structurally of the right and or left ventricles, and it can even escape detection on an MRI. So if we have somebody who's got PVCs that we think come from the right side of the heart, and they're not up in the outflow tract, then ARVC is one of our considerations and in the electrophysiology laboratory administering isoproterenol to see what that does to the arrhythmia and many, ARVC people produce multiple morphologies of PVCs. And to see if they have inducible sustained VT, that also typically comes from the areas of scar where the heart is involved typically, or most commonly the right side of the heart. That information is very useful in assessing these patients.

Dr. Kashou: It is so fascinating. You know, you're almost leading us into our next question that I wanted to ask. And you mentioned PVCs originating from the right ventricle, looking at maybe polymorphic nature of some of PVCs in comparison, comparing them to the sinus beats. What is your approach to working to localize these PVCs on ECG?

Dr. Stevenson: Yeah, I have a very simple approach, kind of a three step approach basically. So, first thing is to look at lead V1. So V1 of course sits just at the second right intercostal space and over the anterior chest fall, and a vector that is moving away from V1 that gives you a dominant S wave in V1 will produce a negative deflection in that lead and tells you that you're depolarizing the anterior aspect of the heart. So the ventricle that sits right underneath the chest wall there is the right ventricle. If you go straight back from that, you encounter the septum. So if you have a dominant S wave in V1, you've got initial depolarization of the right ventricle or the inter ventricular septum. In contrast, if you've got a big dominant R wave in V1, which would be

referred to as a right bundle branch block like pattern that tells you that the posterior ventricle, which is the left ventricle is being activated first. So, from V1 you have a pretty good idea, is this right ventricular or left ventricular? Then you look at the frontal plain axis and a wavefront which is going from high to low from cranial towards the diaphragm, produces large R waves in the inferior leads two, three, and AVR. Whereas if you depolarize the diaphragmatic surface of the heart first, you get dominant S waves in the inferior leads. So that gives you your position high or low. And then to get your position in the base to apex frame of reference of the heart, you look at the leads that are over the apex, leads V3 and V4 and away front, which is going away from V3 and V4. So big S waves in those leads tells you that you're depolarizing the heart out near the apex of the heart, whereas a wavefront, which is coming at V3 and V4 throughout the cardiac cycle, so dominant R waves in those leads tells you that initial depolarization originated towards the base of the heart. And from those three steps, you can nail down the quadrant of the ventricle that's involved in likely producing that arrhythmia pretty quickly.

Dr. Kashou: I love that approach three simple steps, and I'm learning and listening as we go. But you almost localize it in almost that 3D dimension, V1 again, right or left ventricle, the X is the frontal plane, you know, high or low, and then base to apex V3 and V4 I love that approach. And that's three simple methods, thank you. I will remember that as we leave here. And so you say that can localize different PVCs and where they're coming from. Some that are, is there an area that we should be looking for that maybe are maybe more alarming PVCs? You mentioned the V1 for ARVC. Maybe you can mention one that we should remember, or maybe the benign one, the more common one.

Dr. Stevenson: Right, so the easy thing is to remember, well, I'll give you, the most common benign ones. So the most common benign ones are from the outflow tract. And so from the right ventricular outflow tract, you would expect left bundle branch block, and the outflow tract is the highest portion of the heart, so it's going to have an inferior axis and the outflow tract region, by that we mean back around the aortic and the pulmonic valve rings, so that's towards the base of the heart. So, those mid pre cordial leads V3 V4 will be dominant R waves. So, left bundle branch block, inferior axis, monophasic dominant R waves in V3 and V4, that's the right ventricular outflow tract. If you move just a little posterior to that, you encounter the aorta. And as you move posterior, you increase the forces that are moving from base to apex. So now even V2 begins to grow a little bit more of an R wave, and it becomes a little broader. So now you've got an R wave prominent in V2, V3, V4, and that suggests you're more into the left ventricular outflow tract, most likely.

Dr. Kashou: So fascinating. So it's that transition you're saying in those leads that helps to, if it's earlier the earlier transition, more posterior. Interesting, great.

Dr. Stevenson: If you wanna remember one more, the third group of idiopathic PVCs are those that come from the left ventricular papillary muscles. So, that's your right bundle branch block configuration, and it's a mid ventricular structure, so it's not way back at the base, it's not way out at the apex. So the mid pre cordial leads, V3 V4 typically have RS complexes. So, a right bundle with an RS complex in V3 and V4, it's probably a left ventricular papillary muscle. And anything else you should get more concerned about the possibility of underlying structural heart disease. And of course, in structural heart disease, you can also have arrhythmias that come from the

outflow track and from along the valve annular as well, those are common locations for fibrosis. So, we still need to fall back on those other things we talked about looking for other signs of underlying heart disease.

Dr. Kashou: Yeah, this is great, I'm probably gonna be the first one to listen to this again, to remember all these and just be able to apply these in practice, you know, from the 12 lead and using your approach, how do you translate those to mapping during an electrophysiology test?

Dr. Stevenson: You know, there's so many variables that influence the nuances of the EKG. So the patient's body habits, the position of their heart in the chest, whether there is any underlying structural heart disease that may influence how the wavefront spreads through the heart. So the 12 lead EKG gives us a pretty good guide, we can predict pretty well the quadrant, the general region of the heart from which the PVCs originate. But if we're taking the patient to the EP lab for ablation, we've gotta do quite a bit better than that, we've gotta get it down to millimeters. And we can't do that from the surface, from the standard surface EKG, but it's a good place to start that informs where we start mapping. And then what we will generally do very early in the case is let's say that we're approaching a right ventricular outflow tract PVC. Well, that's the right and the left ventricular outflow tracks are very close together, those PVCs can look very similar on just the aortic side versus the pulmonic side of that outflow area. So, we'll put the catheter into the region where we think the PVC originates from, and we'll pace and look at the pace QRS morphology that you obtain doing that. And if it looks like what we expect, hopefully it looks something like the PVC then, okay, the heart position, probably isn't having a major influence to disrupt our assumptions, and we proceed with the mapping. If we get something that looks very different then, well, the first thing we do is check and make sure the EKG leads are appropriate in the EP lab, it wouldn't be the first time that somebody reversed the limb leads, and then that can really value up. But if that's not the case and you get a QRS morphology somewhat different than what you expect, then you know that there's something else patient's position to the heart in the chest, perhaps some areas of scar that are influencing how the heart is activated and that the QRS morphology can be potentially misleading a bit for where the arrhythmia is, and you're really going to have to, to map and be prepared to map in locations that you, where you didn't think you necessarily needed to look to determine where the origin of that arrhythmia is located.

Dr. Kashou: That's great, thank you so much. Premature ventricular complexes, PVCs are commonly encountered in clinical practice and understanding of the more concerning associated features can aid in proper expert referral in management decisions. Even more we learn today how the simple 12 lead ECG approach by Dr. Stevenson can help us to localize PVCs and aid during mapping in the EP lab. Dr. Stevenson, what incredible work you have done, and this is only a little bit, I know you've done so much more. You continue to contribute so much to the field of electro cardiology. I'm grateful for this opportunity to talk to you on this important topic. On behalf of our team, thank you for taking time out of your day to join us, it's been a true pleasure.

Dr. Stevenson: Thank you very much, it's been a pleasure.

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