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## PVC Ablation Procedures for Ventricular Tachycardia

Narrator:

Welcome to **Medical Breakthroughs** from Penn Medicine, advancing medicine through precision diagnostics and novel therapies.

Dr. Caudle:

This is ReachMD and I am your host, Dr. Jennifer Caudle, and joining me today is Dr. Francis E. Marchlinski. He is the Director of the Electrophysiology Laboratory and the Richard T. and Angela Clark President's Distinguished Professor at the University of Pennsylvania. We'll be discussing PVC ablation related to ventricular tachycardia. Dr. Marchlinski, welcome to the program.

Dr. Marchlinski:

Thank you. Thanks for having me.

Dr. Caudle:

Well, let's begin. Can you first begin by discussing what are PVCs and where exactly in the heart do they come from?

Dr. Marchlinski:

Sure. Well, PVCs stand for premature ventricular contractions. So, they're extra beats or premature beats that originate from the ventricles, that is the two bottom chambers of the heart, and they can originate from different locations, but very uniquely they actually typically originate from the region surrounding the valvular structures and, occasionally, from the anatomic structures that extend from the inside of the heart, the endocardium, that are called the papillary muscles in part of the Purkinje network. So, there seems to be an added anatomic predilection that really is still poorly understood, but it's very common to have the arrhythmias originate from these anatomic sites.

Dr. Caudle:

Great. And can you tell us why do these occur?

Dr. Marchlinski:

Well, our understanding is incomplete. Some people, obviously if they have structural heart disease like prior heart attacks or nonischemic cardiomyopathy from a viral illness, may develop scarring in the heart and some of the arrhythmias originate from the

scarring, but the majority of ventricular premature beats that occur actually originate -- they're referred to as being idiopathic, that is we don't know the actual etiology. We believe that maybe there are subclinical viral processes that inflame the heart a bit and then in areas that are susceptible, leave some of the cells uncoupled and predisposed to leaking current and firing early and causing these premature beats. So that's the hypothesis but it's still not well worked out. We refer to them as being idiopathic because we don't know the exact mechanism for their origination. We do know, as I said, that there appears to be some method in the madness, in terms of where they originate, that is, the perivalvular structures of the heart.

Dr. Caudle:

Great. And can you discuss what kind of problems that PVCs can cause and also what kind of symptoms do patients with PVCs experience?

Dr. Marchlinski:

I want to start by saying many patients are totally asymptomatic. So they may have frequent ventricular premature beats without being aware that they're having them. And that's very common. We don't quite understand why some people are dramatically symptomatic and others having the same frequency, the same coupling in terms of the degree of prematurity, are not, but suffice it to say many patients are asymptomatic; however, many patients do develop pretty severe symptoms when they have these premature ventricular contractions. The premature ventricular contractions occur, and then typically there's a pause, and the next beat that occurs is a normal beat, but it has a greater intensity in terms of its contraction, and as a result patients will feel this is as a sudden fullness in their chest, or a discomfort that jumps up to their throat. They may, if the beats are recurring, they may describe palpitations. If they continue over a period of time they actually produce some fatigue; they can produce some shortness of breath or dyspnea. Importantly, some patients actually can get dizzy with the extra beats. Some of this is a sudden onset of these extra beats in a bigeminal pattern, that is every other beat being the abnormal or extra beat, suddenly decreases the effective heart rate and the extra beat is actually not produced because, or doesn't create perfusion, because it occurs early and as a result, there is an effect of halving of the heart rate and patients experience some dizziness. So these are the symptoms that patients can experience. There's one other important point, that is, patients can experience what's referred to as a PVC-induced cardiomyopathy. So, over time, particularly if patients have a high PVC burden, typically more than 10,000 PVCs in a day, they become at risk for developing a weakening of the contractility of the left ventricle, and if allowed to progress, the ventricle can dilate and eventually patients can develop symptoms of heart failure and congestive heart failure, so that it's important to recognize the possibility if patients are having frequent PVCs. As I said, the cut off that we frequently begin to be concerned about is at a level of 10,000 PVCs per day, but if they have more than that we worry and we monitor the patients with repeated echocardiograms, typically every 3 months to 6 months initially, and then once we have a comfort level that the PVC burden is stable and the cardiac function is stable, maybe less frequently, on a yearly basis. But there is that potential and it's of concern because patients may not be aware of the PVCs and their first manifestation of having a high PVC burden will be breathlessness and the development of heart failure from left ventricular dysfunction.

Dr. Caudle:

If you are just tuning in, you're listening to Medical Breakthroughs from Penn Medicine on ReachMD. I'm your host, Dr. Jennifer Caudle, and I'm speaking with Dr. Francis E. Marchlinski who is the Director of the Electrophysiology Lab at the University of Pennsylvania. So, can you tell us how do you treat PVCs?

Dr. Marchlinski:

If the patient's asymptomatic and the PVC burden is low, that is they're having less than 5,000 to 10,000, then frequently we won't treat the patients. We'll just reassure them and indicate that they'll need some ongoing followup. But if patients have symptoms, or if they have a decrease in cardiac function, then they obviously need to have treatment initiated and there are two forms of treatment; there's pharmacologic and nonpharmacologic. The pharmacologic therapy that we usually employ is...consists of beta-blockers. They're the mainstay of treatment. And we also can use in selected patients, calcium channel-blockers. We reserve the more potent membrane-acting drugs for patients that don't respond to the beta-blocker or calcium channel-blocking therapy. The problem with medical therapy, however, is that patients tend not to be very responsive. At best, it is reported in the literature, only 25 to, at most, 50%, if you really try a lot of drug treatment strategies, will respond, and patients, therefore, will remain symptomatic and the PVC burden may continue to

contribute to the risk of cardiomyopathy. So, the second way of handling these arrhythmias is by doing what's referred to as catheter ablation. We know now from experience that these extra beats come from very specific anatomic sites and we've gotten quite good at looking at the 12-lead ECG pattern, when the patient's experiencing the extra beat, and being able to predict within a dime-sized area of the heart where these extra beats are coming from. This allows us to position a catheter that can deliver radiofrequency energy at the right spot, confirm the location of and the source of the abnormal beating, and apply radiofrequency energy, and eliminate the source, and eliminate the PVCs. It's a very successful technique that typically, at least 95% of patients get an initial response and over 95% during longterm have their arrhythmias eliminated. Only in patients where the PVCs end up being in very precarious positions, sometimes they're located under the coronary arteries, and sometimes they're located linked to the Purkinje network, that we are unable to eliminate the arrhythmias because of the risk. But it's a very effective form of therapy and it's a good alternative when patients don't respond to drugs.

Dr. Caudle:

So, is the heart muscle weakening reversible when one gets rid of the extra beats?

Dr. Marchlinski:

Yes. So, it's important to get rid of the majority of the extra beats. So, as I indicated, the threshold for weakening of the heart looks like 10,000 per day, and so the number of PVCs have to be reduced to a minimum of less than 5,000 per day to see a benefit either with pharmacologic therapy or with catheter ablative therapy, and for many of the patients with catheter ablative therapy we can eliminate the PVCs completely. It takes time to recover from the dysfunction that's occurred. I always tell our patients that it may take as long as it's taken to develop. The weakening of the heart muscle will reverse, but the same time-period over which it took to develop the cardiomyopathy, you may have that same duration in terms of the reversal of the cardiomyopathy. But the heart muscle dysfunction can reverse and that's been very exciting, to see a patient with pretty severe left ventricular dysfunction have a completely reversible disease process by eliminating the PVCs. In fact, in some of our patients, they initially looked like candidates for implantable defibrillators because of the severity of the dysfunction and we've been able to reverse the dysfunction completely by eliminating the PVCs and eliminate the need for the defibrillator.

Dr. Caudle:

That's wonderful. So, does this work in people who have structural heart disease?

Dr. Marchlinski:

Yes. It's, I think it's very important to note that not only does it work in people who start out with what's referred to as idiopathic VPBs who develop this cardiac dysfunction or have palpitations, but in people who have had a prior heart attack or weakening of the heart muscle from a viral cardiomyopathy. Those patients start out with depressed function and the PVCs actually can make the depressed function even worse. So, it's very nice and helpful in those patients because they've been marginalized already to get rid of the PVCs and see the dysfunction improve by 5 or 10%, in terms of the, what's referred to as the ejection fraction, the left ventricular ejection fraction. It represents the amount of blood that gets squeezed out of the heart for every heart beat and normally it's 55% and if you can get an extra 10% when you've had a prior heart attack and starting out at 35% and can get it up to 45%, you're really going to have an improvement in cardiac function. So, I think it's important to note that these techniques, whether it's an effective drug, or effective ablation, can work to improve the heart dysfunction in patients who've had some damage and also have frequent PVCs.

Dr. Caudle:

Great, and then, finally, are there any other circumstances or situations where eliminating PVCs is particularly important?

Dr. Marchlinski:

Yes. I think though there's been now a movement to treat patients who have structural heart disease and who have what's referred to

as left bundle branch block on their ECG with a pacing device that paces both chambers and what is referred to as resynchronization of the heart. Well, even a small amount of, number of PVCs, can make these devices less effective and so patients who get these devices and don't seem to respond to the promise of resynchronization therapy, the first thing one should do as a physician is assess how much in the way of PVC burden the patient's experiencing. And if it is even more than a 1000 PVCs per day, it creates a situation where the device won't work properly and just by going in and eliminating the PVCs, either with ablative therapy or adding the appropriate drug therapy to produce the same effect, you can improve the function of the device therapy and patients again benefit quite a bit.

Dr. Caudle:

Wonderful. Well, Dr. Marchlinski, thank you so much for joining us today and sharing your insights on PVCs.

Dr. Marchlinski:

My pleasure. Thank you for the opportunity.

Narrator:

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