Kendal Williams (Host): Welcome everyone to the Penn Primary Care podcast. I'm your host, Dr. Kendal Williams. So if you're like me, atrial fibrillation has become very confusing. When I graduated from residency, we would measure the left atrial size, decide whether a patient was eligible for cardioversion. If they weren't and many weren't, we would put them on Digoxin and Coumadin and go about our day and figure out how to manage the INR levels. Since then, it's gotten a lot more complicated, largely because of the success of various interventions and increasing evidence around the best forms of anticoagulation as well as antiarrhythmics and so forth. So, I invited an old friend, Dr. Benjamin D'Souza, on the program to talk to us about all this. Ben is a cardiologist at Penn, an electrophysiologist primarily based at Presbyterian Hospital. He's an Associate Professor of Clinical Medicine at Penn. He did his residency at Penn in Med Peds, and then also Cardiology fellowship and EP fellowship here. Ben, thanks for coming.

Ben D'Souza, MD: Thanks for having me, Kendal. You know, it's funny, I went from the most generalized residency to the most subspecialized people, so go figure. I took a couple of left turns. And so, I'm happy to chat with you and cover pretty much everything about AFib that we can during our time.

Host: I remember Benny was one of my most stellar residents, and we worked together a couple times. At that time, you were going to go into congenital heart disease. You were going to transfer your Med Peds training into congenital heart disease, but then it went another direction, huh?

Ben D'Souza, MD: Sometimes it's the mentors and the people that you interact with during your training more so than the field that you think you're going to go into. And if you asked me when I started residency that I was going to be an adult electrophysiologist and not a pediatric cardiologist, I'd say, "You're crazy." But go figure. You know, always keep your eyes and ears open. You never know who you'll interact with and decide what you are going do. But I couldn't be happier in what I do now, but you're right, Kendal. From the days when we were rounding at Presby together to doing this now, it's changed significantly, but in a good way.

Host: So, let's talk about atrial fibrillation. This is obviously very common. Twenty-five percent of patients over 40 will have atrial fibrillation. You know, we know that the primary concern with AFib is stroke risk, and we spend a lot of our efforts thinking about that. But, you know, there's also prolonged AFib, uncontrolled AFib that leads to tachycardia-induced cardiomyopathy. I'm not sure we even knew that when I was in residency. And then, there's also the symptomatic piece that we want to be attentive to and control. Ben, I'm just

going to start with AFib generally and get your thoughts on the complexity as it exists now, sort of from an overview perspective.

Ben D'Souza, MD: Sure. I tell my patients, atrial fibrillation, it's the most common abnormal heart rhythm in America. Millions of Americans have it. It's the most common reason that people come and see me. It's not a life-threatening heart rhythm. So, it's usually the first thing I tell patients because they're very worried. They're meeting, you know, a specialist who deals with life-threatening heart problems, but it can be life-altering certainly. And as you had mentioned, there're sort of different stages of atrial fibrillation. And largely treatment-based options, which we'll spend more time talking about, can be related to symptoms and obviously stroke prevention, sort of the two most important things.

But as we've done better and better at treating patients, keeping them alive longer and having more octogenarians and nonagenarians in the world, we're going to see more and more AFib. So, it doesn't really matter what your subspecialty is, whether you be an electrophysiologist, an internist, an ER doc, we will all take of patients with atrial fibrillation. So, it's important that we understand what it is and how to treat it and not ignore it essentially. And so, I joke with my patients, it'll put my kids through college because I'm going to be caring for millions upon millions of patients with it and they're just going to continue to grow. So, it behooves us to make sure that we know how to treat this properly. Because quite frankly, the majority of the people who treat this are not electrophysiologists, they're primary care physicians. And so, that's very important for them to understand. And also, kind of talk about some things that were lore within primary care and internal medicine for a while with AFib that are actually not true, and that we've disproven over the last few decades.

Host: Let's start with the basic breakdown of AFib into valvular AFib and non-valvular AFib. You know, our literature basically starts with non-valvular AFib because that's what we're largely managing ourselves as opposed to valvular AFib. When we say valvular AFib, Ben, we're talking about not just valve replacement, but also valve disease associated with AFib, right?

Ben D'Souza, MD: Right. So, most clinical trials that looked at valvular AFib, and we're talking about largely anticoagulation trials, were based on patients who had moderate to severe mitral stenosis. So, the mitral valve is the one that's particularly troublesome in terms of patients who have atrial arrhythmias. It's sort of a different mechanism and a different way of treatment. But also, patients who have other valvular disease, tricuspid, aortic, that's not necessarily part of the definition. So, if you've had a bio AVR, aortic valve replacement or, again, non-mitral disease, it's not really indicative.

The reason that this is important is that the mechanism of blood clot and stroke forming in patients who have mitral stenosis or mechanical mitral valves in particular is completely different. And so, it really changes our treatment and that's kind of the basic difference between valvular and non-valvular AFib. And you're correct, Kendal, the majority of clinical trials and treatment options we're talking about is in non-valvular atrial fibrillation, meaning non-mitral valve disease. But that is the majority of the patients anyway that have that.

Host: And it's still true that for the most part, from an anticoagulation perspective, for valvular AFib, Coumadin still has the dominant role, right?

Ben D'Souza, MD: Correct. And so, you know, it's interesting when they did initial trials looking at the novel anticoagulants or the DOACs, things like Eliquis (apixaban), Xarelto (rivaroxaban) or Pradaxa (dabigatran). Patients who had mechanical valves and/or moderate to severe mitral stenosis, they developed clots like very high risk of it and had strokes. And so, the indications in ACC/AHA guidelines are very clear that for those patients, they should be on warfarin or Coumadin. But the majority of the other patients, which is almost everyone, can be on the novel anticoagulants. But that's correct. Those patients should very much only be on warfarin or Coumadin.

Host: So several months ago, I had a patient come to see me who was older, 70s I believe, but really hadn't seen a doctor in a long period of time, and came into the office and was asymptomatic, had actually biked in. He was very physically active. And we did an EKG because he was a new patient. He was in AFib. So, we had this question, "Okay, so what do we do now? " His heart rate was relatively controlled at that point. But the first decision for me is really about an anticoagulation decision. And then, a question of, "Do I need to control the heart rate with something? Do I need to get that under control?"

So before we get into issues of rate versus rhythm control and other things, those are the most basic elements of all AFib management, I think, are rate control and anticoagulation. So Ben, it's changed a little bit in terms of the urgency with which you would start anticoagulation. By that, I mean, you know, we used to admit people. That patient would've been admitted to the hospital in my residency program and placed on a Heparin drip. I think we've backed off on that a little bit, but we still want to start anticoagulation whenever we recognize it, right?

Ben D'Souza, MD: No, I agree. And, you know, I think that with things like the Apple Watch and a device called KardiaMobile and there are many other ways that you can actually get an EKG, we're identifying even more patients

with atrial fibrillation than we did before. But even today, I had a patient come in who went to their primary care doctor for their regular visit, had an EKG, and it showed atrial fibrillation, completely asymptomatic. So, I think it's incredible to me that we have patients that are so symptomatic with AFib that they go on heart failure or can't tolerate even a little bit of it. And then, you have patients who have no idea that they have it. And so, that spectrum sort of runs the gamut. I see patients of all age ranges with atrial fibrillation. And it's more common as we're older, but I see it in 20-year-old marathon runners. And so, it really is fascinating from that concept.

In general, the patients who are older tend to be the more asymptomatic and that's likely because the atrium is a little bit more stiff as we get older. And that actual squeezing of the atrium makes less of an importance to those patients. That's the concept. And so, in terms of anticoagulation, it is paramount. It's kind of the number one important thing. Because as I mentioned earlier, it's the top two cause of stroke in America along with hypertension. And so, really, that's the majority of our patients. And I just rounded on a patient yesterday in the hospital who had a stroke from AFib who was asymptomatic and she's doing okay, but the family are devastated. I mean, strokes are probably the worst thing that can happen to a person. There's multiple surveys that ask patients whether they'd rather die or have a stroke, and the answer is die. That's how bad strokes are. And unfortunately, strokes from AFib tend to be more debilitating than non-AFib strokes. So, it's an even worse scenario.

It's interesting that in terms of the answer to anticoagulation, yes, now that we have novel anticoagulants, meaning Pradaxa or Xarelto, Eliquis or even edoxaban, there are multiple options for patients. You know, warfarin or Coumadin's time tested. It's been around forever. Some call it rat poison because it was used for that. And so, unfortunately, warfarin has a couple of issues that many of you have dealt with, is that it's very difficult to a patient to get them therapeutic on INR. It interacts with a lot of medications and foods. It's a vitamin K antagonist. So, it really is difficult for patients to take, but it is cheap. So, the other novel anticoagulants, depending on the choice of medicine, tend to be more expensive, but they work within about two hours of when you take them. So if you have a patient in the office and you prescribe it for them, by the time they take it and go to the pharmacy, they're therapeutic. And it's really wonderful from that standpoint. And all of them have been found to be at least non-inferior, if not superior, to Warfarin. So, I'm very biased in that those drugs are much better in terms of preventing stroke and ease for patients to be able to take.

Interestingly though, even with the novel anticoagulants, there's up to a third of patients who are not anticoagulated who have atrial fibrillation. And so, that's an important thing to recognize. And you'll have patients come into your office who have chronic atrial fibrillation and not anticoagulated. And a lot of times they'll come in and see me and I say, "Why?" And sometimes you have to tease that out in a little bit more detail. But it is very important that we figure out why. If the answer is that they can't be, then we can talk about it, you know, moving forward in terms of alternatives to anticoagulation. But it's the number one most important thing in terms of treating patients who have atrial fibrillation, is the stroke prevention..

Host: You know, the highest risk category for stroke risk, I think is now 18%, I believe. So, 18% per year. You're talking about anticoagulants dramatically reducing that risk, not to zero, but bringing it down significantly to the point where it is the highest priority whenever you recognize patients.

Ben D'Souza, MD: That's exactly right. And what sometimes people don't recognize about either CHADS or CHA2DS2-VASc score is that it's a cumulative risk. So when I tell my patients, we're not talking about 50-year-old, like next year, but over the 30 years. If you do that math, it's pretty profound in terms of stroke risk. And so, I had mentioned the CHADS score, which is the sort of the general score calculated to decide if patients need anticoagulation. But I will say that everyone should be using the CHA2DS2-VASc score.

So, the CHA2DS2-VASc score was developed after the CHADS score, and it basically looked at retrospectively which patients were we missing? Who was having a stroke that had AFib? And the answer was women and people with vascular disease. So, those are the ones that traditionally were missed from the CHADS score. Now, we think that women is an interesting one because we're not sure if it was actual biases within decisions. If you actually look across the board, we tend to prescribe anticoagulation more for men than women, even for any other risk stratification and offer advanced therapies like an ablation or otherwise. And this is biases within medicine that we always have to pay attention to. But also, there's some thought that there may be actual menopausal differences in hormonal differences within blood clot formation that might also contribute. We don't really know, to be honest. But we know that women were under prescribed anticoagulation and had a higher risk of stroke. And patients with vascular disease, so peripheral vascular disease, coronary artery disease, disease elsewhere, that increased their risk of a blood clot forming. And so, the CHA2DS2-VASc calculator, which you can get on MDCalc or really anything, you really should calculate for every patient who has atrial fibrillation, and I

document that in every one of my notes because I think it's important I tell that patients what their risk is.

There's also a bleeding score, calculator, it's called HAS-BLED. And that actually tells you your risk of bleeding. But I want to be clear that your CHA2DS2-VASc and your HAS-BLED score don't tell you which one to choose. So if your HAS-BLED score is really high and your CHA2DS2-VASc score is moderately high, it doesn't mean, "Okay, don't do anticoagulation." It turns out that the higher your HAS-BLED score is, the higher your CHA2DS2-VASc score is because you're sicker. And so, it is just a sort of a natural progression. And so, I use those score calculators every single day. Again, they're pretty easy to calculate on the patients. Honestly, if your CHA2DS2-VASc score is one or above, I do anticoagulate. One is kind of a borderline depending on the European or the US guidelines. But I hate strokes, so I really push to anticoagulate these patients more so than not. And again, just another caveat for the CHA2DS2-VASc score, if female is your only risk factor, that is not a risk factor. It's female with age or female with diabetes, heart failure, previous stroke, et cetera.

Host: So, I don't calculate it as much because I think a lot of my patients, I automatically know that they need anticoagulation because as I've sort of looked through the score, it's almost like these patients that we used to refer to as lone AFib, I don't know if you still use that term, but basically are young people with no other risk factors, no other comorbidities who just happen to have atrial fibrillation out of the blue and who may not need anticoagulation. But beyond that, it seems like there's a very small category of people with AFib that they can actually opt out of it.

Ben D'Souza, MD: You're right. And honestly, because we tend to take care of just sick people in general, not just in Philadelphia, but across the board. You mentioned lone atrial fibrillation. So, let's talk about sort the different categories. So, lone atrial fibrillation is a single, isolated episode of AFib that never occurs again. Less than 10% of patients are lone AFib. It's quite uncommon, generally will happen in the setting of having surgery or sepsis or some other clear inciting factor.

So then, you move to what's called paroxysmal atrial fibrillation, so atrial fibrillation, which your heart goes in and out of it on its own. By definition, again, terminates without requiring a cardioversion or within seven days. And so, that's what's referred to as paroxysmal. Patients will progress to persistent atrial fibrillation, which is where you're in atrial fibrillation for longer than seven days, require a cardioversion. About 20% of patients progress within a

year. So, one in five is what I quote my patients, which is what the data shows in terms of going from paroxysmal to persistent. The reason that's important is that atrial fibrillation begets atrial fibrillation, sort of a common mantra in our field, meaning it causes damage to the heart. And we can talk about that moving forward.

When you get from persistent atrial fibrillation to what's called longstanding persistent atrial fibrillation, which is more than a year of being in atrial fibrillation, that's the next category. And then, chronic atrial fibrillation, previously referred to as permanent atrial fibrillation is when we've made a decision that this patient will not ever try to get out of atrial fibrillation and they'll stay in it permanently. And we can talk about the consequences of that or the benefits and risk of that certainly as well.

So, those are the different classifications. The progression to each is variable in the patient, but is progressive. It's a progressive disease. It does not generally go away on its own. And for patients who have one episode and they assume that they're not going to have another, that's extremely uncommon.

And interestingly, and this is a boards question, the question is your risk of stroke higher in paroxysmal versus persistent atrial fibrillation? And the answer is it's the same. Most of us think, "Oh, it's PAF. It's going away within a day or two." The common thought was within six to 12 hours, a blood clot would form in the heart. But I want to caution people that say, well, they have PAF, not persistent AFib, so I'm not prescribing them a novel anticoagulant, that's not the correct thing to do. And that those patients should be based on not the chronicity of atrial fibrillation, but on their CHA2DS2-VASc score. And so, it's a very common misconception and a common mistake that people make in terms of not prescribing or even offering anticoagulation for patients who need it.

Host: Ben, I just have one question I want to go back to on the patient who seems to be low risk and has an episode of AFib, do you monitor them?

Ben D'Souza, MD: Yeah. That's a good question. You know, and the patient, regardless of whether you start anticoagulation or not, I'm a big fan of monitoring, and there's a couple of options in terms of what that monitoring is. So if you're talking about a patient that's paroxysmal, you know, Holter monitor monitors for 24 hours. The only time I really prescribe a Holter monitor is for patients who have chronic atrial fibrillation, I want to make sure they're under rate control, right? The data largely shows that if you keep the heart rate below a hundred, you'll decrease the risk of, as you had mentioned earlier, Kendal, a

cardiomyopathy. And so, there's actually some data that shows that aggressive beta blockade or calcium channel blockade, meaning getting the heart rate between 60 and 80 is probably not that important. If it's between 80 and 100, it's ok. But 120 and 130, you'll more likely develop a cardiomyopathy.

The next option in terms of monitoring is either a mobile telemetry or an event monitor. Again, these are longer monitors, like two to four weeks, largely to look for patients who have symptoms that are less sporadic. You know, I mentioned KardiaMobile and the Apple Watch. And so, the data actually shows that those devices are actually quite sensitive, but not specific. Actually, they're more than 98-99% sensitive. They're very good at reading normal. You have to wear it, obviously. And so if you're not wearing it, that's a problem.

But there's some studies. There was a study that was published where they took patients who were wearing the watch who were in the hospital on telemetry and they looked between the two. And so, the problem is I mentioned it's not specific. So, premature atrial beats, artifact, PVCs, other reasons, noise. I can't tell you how many times I've had patients that said, "Oh, doc," said, "I had possible AFib on my Apple watch." And I look at, "I'm like it's normal." You know, very frequently, I'll have patients upload their EKGs to myPennMedicine and then send to me. And then, I'll take look at them just to make sure that everything's ok. But those are actually pretty decent technologies to be able to monitor your heart rhythm.

And then, the option that I use very frequently in patients, especially if we're going to talk about stopping their anticoagulation, is an implantable monitor. So, implantable loop monitor, which is about the size of the pen cap goes underneath the skin, monitors every heartbeat for like four years. There's actually some pretty sophisticated AI algorithms for me to be able to monitor your heart rhythm and it transmits actually through your smartphone. So every day, it sends us information. I run all the remote monitoring at Penn for our patients. We follow like 5,000 patients that have devices and we have a whole team of people that just watch it. And we're not looking at it in real time. But every day, we get data on a patient and then we look at it and it helps us to make decisions.

Just to give you an example, I have a very close friend of mine. He's actually my best friend. He lives down in Somers Point, New Jersey. He's 41 years old and he has AFib. So, he has an implantable monitor and I'm kind of keeping an eye on him to make sure that he's doing okay, because if his burden starts to get worse, I'm going to start to push him to do more aggressive things than being on anticoagulation, rate control and on an antirrhythmic. He's actually on flecainide

currently. So, he's my best friend, so I'm not thrilled about having to take him in to do an ablation. So, I'm trying to get him to do appropriate lifestyle modification to be able to help with his atrial fibrillation, which there are options for it.

Host: The patch monitoring has been useful in primary care, just a non-intrusive option. I imagine you probably are doing so many implantables that you don't order as many patch monitors.

Ben D'Souza, MD: That's true. The nice thing about the patch monitor is it records the data. It's really easy to wear. It's waterproof, patients can shower with it and stuff like that. The only caveat, and you need to be careful, is that if you're looking for something dangerous, that patch monitors don't transmit in real time, so you don't find out what the data is until after they return it. So if you have a patient that has syncope or you are worried about something, but AFib is actually a good indication for that as long as they're anticoagulated obviously. And so, I don't use as much of that, but I tend to sway towards wearables, like KardiaMobile or an Apple watch or an implantable. But I do all of it pretty much. This is very tailored to the patient depending on what you're looking for. But it is important to look for progression of atrial fibrillation because when you move from paroxysmal to persistent, success rates of everything we're going to talk about moving forward go down. And so, you want to offer the best success rate for your patient.

But the biggest hurdle that we have in electrophysiology is that referrals to us for symptomatic AFib are quite late in the disease state. And so, I always thought it was fascinating that if you had a patient in your office today who had chest pain, you wouldn't even let them leave the office. You'd admit them to the hospital and make sure that they don't have anything significant. But AFib will kick the can down for years. We'll basically say, "You're fine. You're not going to die." But then, we don't offer them the best therapy for rhythm restoration that we could have and we decrease their success rates because we did that. And that's largely because of trials like AFFIRM, which I'm happy to talk about in more detail. And things that us in primary care feel like, "Oh, well, you're not going to die from this," that doesn't mean we have to do anything further. Well, we're doing a disservice to the patient by doing that.

Host: So, let's talk about rate versus rhythm control, because I can give you a little bit of the primary care perspective on this. You know, I sort of felt like I knew what I was doing with atrial fibrillation. And it seemed that, you know, maybe in the early 2000s, we would constantly get in the New England Journal new studies, trying to show that rhythm control was better than rate control.

And I would flip through them and flip through the abstract and it would say it failed to make any improvements over rate control. And I'd say, "Oh, good, that's good for me. I don't have to do anything different. But there has been a lot of change since then. And I think many of us maybe have missed that. and that's maybe what you're referring to, Ben. You know, that you people are not getting to you early enough. So, let's actually go back for a second and talk about the effects of AFib on the heart, not just uncontrolled AFib, but just AFib as well.

Ben D'Souza, MD: Sure. So atrial fibrillation, obviously beyond the symptoms, what happens is, and there's very interesting animal models where they took animals and they put them into rapid AFib and saw what happened to the atrium. So, the atrium over time scars. And in fact, at Penn, we did the initial studies to figure out what's the normal voltage of the atrium. We actually had patients sign up and we put catheters in their heart and we figured out what the normal-- we at Penn discovered this. And so, we actually know what normal voltage is in the heart. And what happens is over time a scar starts to form. And I describe it to patients as like scar on your skin. And the left atrial size, as you alluded to earlier, Kendal, is kind of a marker of that. It's not perfect, but it's sort of a rough marker of basically dilation and damage that occurs to the heart. And so, essentially what happens is that scar pattern starts to develop over time and gets worse over time. And that is what leads to not just the symptoms of AFib, which is palpitation, shortness of breath, fatigue, exercise intolerance. I can't tell me how many times I talk to patients and I say, "Do you have any symptoms related to AFib?" And they say no. And then if the common symptom you're talking about is palpitations, they don't necessarily have that. But then, when I tease it out a little bit more, they're like, "Well, I used to run. Now, I walk. I used to do this, but I'm getting older and it's related to that." It has nothing to do with the age. It has to do with that your pump is not pumping in unison, which is what I tell my patients. But some patients, because of that scar patterns, develop heart failure. And so, we may not say, and there is no trial that's shown a mortality benefit from restoration of sinus rhythm in atrial fibrillation, morbidity is significant, and so whether it be symptoms, admissions for heart failure.

And very interestingly, over the last few years, there's some clear data that shows that leading patients in atrial fibrillation, even on anticoagulation, increases your risk of vascular dementia and early senile dementia. And so, we think that even on anticoagulation, you're probably throwing microemboli to the brain and causing infarcts that we don't see. But it's been very clear. And so if you have, like the patient you were referring to, Kendal, a 50-year-old who comes into your office in new-onset AFib, who's asymptomatic, one, I would say really tease out symptoms, especially if they don't know the duration of AFib. And two, the mantra in EP is always give someone at least one attempt at

sinus rhythm, whether that just be a cardioversion or a medication or a combination.

There is long-term data that shows that remaining in atrial fibrillation for years or decades is detrimental in certain ways. And so, we've never really acknowledged that until somewhat recently that we're probably causing harm to at least a subset of patients. Look, if you have a nine-year-old that comes in your office, asymptomatic AFib, talk to them about anticoagulation, make sure the rates are controlled and leave it at that. But if you have a 50-year-old who comes in in atrial fibrillation, even if they're asymptomatic, I would still prove that they're asymptomatic. Get them out of AFib and see how they do, and almost everyone feels better. But also, explain to them there are some consequences to leaving you in atrial fibrillation for forever, especially in younger patients.

Host: So before we deal with cardioversion, I want to ask a scenario, let's say a patient presents to you that their CHA2DS2-VASc is high enough that they would be on anticoagulation, they have paroxysmal AFib, they've been documented, maybe they're in an ED. But now, they come to you and they're not in AFib. What do you do with that patient?

Ben D'Souza, MD: This is a symptomatic patient or asymptomatic patient?

Host: They were symptomatic when they had it.

Ben D'Souza, MD: But haven't since.

Host: Haven't since and they self-resolved, auto-converted.

Ben D'Souza, MD: So even after the first episode of atrial fibrillation, I do recommend anticoagulation, I think, as I had mentioned, that lone atrial fibrillation is so uncommon and the risk of a stroke is there, assuming they're not a CHA2DS2-VASc of zero. Typically, what we'll do is look for any potential reversible risk factors. Those are things like thyroid disease. Sleep apnea is a huge unidentified risk factor. And we've actually started a program at Penn where we're doing home sleep studies, to try to figure out in patients-- these are patients who have documented AFib-- clearly shown that attempt at rhythm restoration is improved by helping sleep apnea.

And weight loss, obesity is another epidemic we have in this country. And it is clearly shown that weight loss decreases burden of atrial fibrillation. There was a paper that was published in JACC a few years ago that clearly showed that.

Now, it's easier said than done. I mean, I think that more and more I've tried to educate my patients on weight loss and try to get them to exercise and diet and do the appropriate things. So, diet, exercise, modifying any other risk factors, whether it be excessive amounts of caffeine and alcohol, those are all potential things that should be done regardless. And then, we generally will put a patient on, in addition to anticoagulation, some sort of rate control agent, whether it be a beta blocker or a calcium channel blocker. I tend to sway more towards beta blockers. So, there's no rhyme or reason per se, depending on the patient.

And then if they have recurrent atrial fibrillation, now you've sort of gone to the point where you're going to prescribe an anti-arrhythmia medication or recommend an ablation. And when you get to that point, you should refer the patient to a cardiologist or, honestly, a cardiac electrophysiologist. And I think that what happens is, even now, you guys are the gatekeepers of these patients. You see them much more than we do. But the delay in getting them to us is again decreased in terms of success rate. And I'm happy to walk through what we currently use in terms of antirrhythmic medications and talk more about ablation.

Host: Before we get to ablation, let's just talk about the anti-arrhythimia medications because the other scenario I was just ready to propose to you is a patient who is in AFib when they come in to see you. And, you know, they're rate controlled, maybe they're on metoprolol, they've seen their internist, they're on apixaban, say, but they're in AFib. You've got an echo I'm sure by the time they get to you generally. And you're going to make a decision about cardioversion. What's going into that decision?

Ben D'Souza, MD: I mean, you want to confirm that they're not going in and out of atrial fibrillation versus persistently in it, whether that be with a monitor or one of those mobile devices that I'd mentioned before. In terms of anti-arrhythimia medications, unfortunately, amiodarone is the easiest drug to prescribe. It's the most commonly prescribed anti-arrhythimia medication in America, and it has the most side effects. You know, whether it be thyroid, liver or lung toxicosis, if any of you have taken care of some of these patients. I just saw a patient earlier today who has really bad thyroid disease because of amiodarone and sometimes it's irreversible.

The other problem with amiodarone is it absorbs in the fat tissue. And so basically, the halflife of amiodarone, depending on the patient, can be up to 50 days for one halflife. So, this drug stays on for a very long period of time, especially if it's been chronically used. Well, the problem is if you've already used amiodarone, every other drug that I might try to prescribe to the patient,

which are flecainide or propafenone, which are the 1C agents or medicines like dofetilide, Tikosyn or sotalol, which are the class 3 agents, class 1C agents affect the sodium channels, the class 3 agents affect potassium channels, you don't need to necessarily understand that part, but I can't prescribe those if they've already been on the drug amiodarone. So if you've already decided this patient's going to be on amiodarone and they've been on it for a few months, there's a black box warning to prescribe any of those other drugs. So, you've already kind of pigeonholed the patient that they have to be on this drug for life. And like I said, for older patients, it's perfectly fine. But for younger patients, I think you're doing this patient a disservice by just putting them on amiodarone. And if that decision is made, the question is, "Why didn't you pick any of those other drugs?" And if the answer is, "I'm not familiar with prescribing any of those other drugs," well, then send them to a doctor that does do that for a living, which is me.

And so, I think that cardioversion is very much an option for patients certainly. Cardioversion I described to patients as like resetting the fuse box in your house. It'll get you out of atrial fibrillation, but it won't keep you out of atrial fibrillation. And so, I have patients that might last a day, a week, a month, a year. So, that prediction is variable. But in the absence of starting another drug or talking about ablation, the likelihood of recurrence is exquisitely high. And so, I would pretty much guarantee a patient that they're going to go back into AFib if we just do a cardioversion. And it's perfectly fine to do it. And in general, if you've been on anticoagulation for a month and sort of that category and been compliant or INR's therapeutic, then you don't need a transesophageal echocardiogram or a TEE. And then, so for anyone who's ordering it and/or prescribing it, if it's new-onset AFib and they haven't been anticoagulated, then do a TEE. If you're not sure about compliance, do a TEE, because you don't want to miss blood clot, right? Because then you have a stroke and it's devastating. But if they've been compliant, we routinely will not do a TEE in a patient who's been on it for more than, you know, four weeks.

Host: So, a patient that comes to you and they're on metoprolol and apixaban, they've been on it for six weeks, you're comfortable starting an antirrhythmic right at that first office visit. I actually want to highlight this, I didn't know this. So if you've been on amiodarone, you can't use the other agents, right?

Ben D'Souza, MD: For at least a month. You have to wash out the amiodarone and then start the other drug. And depending on the drug, you have to hospitalize them.

Host: Flecainide has a bad reputation, somewhat infamous because it was actually, you know, in my circles, which was evidence-based medicine, it taught us a lot about the value of randomized controlled trials. And it was always used as the example of randomized controlled trials because, when it was used to suppress V-tach, it actually had increased mortality. But then, it got new life in the AFib universe, right? And maybe you could just speak to flecainide, because that's probably the one most of us know a little bit about and now are seeing it more on our patients.

Ben D'Souza, MD: So, the trial you're referring to, Kendal, is called the CAST trial. And so basically, a drug like flecainide called encainide, was used in patients who had had a myocardial infarction, were having a lot of PVCs, we're having a lot of non-sustained ventricular arrhythmias. And we said, "Well, we should probably suppress those. It's probably a bad idea that they're having them, so let's put them on a drug that suppresses them." Well, the drug did a great job of suppressing the PVCs, but they died more. And so, it was associated with mortality. The drug was pulled from the market. And then, kind of what happened was later drugs like flecainide and propafenone or Rythmol were developed and are the same agents. But now, we excessively are protective with our patients and not using these drugs, meaning if they have a structurally abnormal heart, if they've had a history of certainly a myocardial infarction and reduced ejection fraction. However, we kind of generalize it to all patients, even if that's not correct, meaning cardiomyopathy otherwise, stable CAD, really anything that's abnormal.

The biggest concern that I have, and this is again a relative contraindication, is if you have conduction system disease on your EKG, meaning a bundle branch block or bifascicular block. The reason is that these drugs actually work on the QRS, the sodium channels, and it actually can increase your risk of heart block. And so, the majority of the patients that probably die who take these medicines, it's because of that, it's cause of bradyarrhythmias.

So, what I would say is if you're comfortable prescribing these meds, I think that's perfectly fine. But if you're getting to the point where you're doing that, they're probably should be following with at least a cardiologist, if not a cardiac electrophysiologist. And so, not that you can't prescribe it, but they're complicated and they have drug interactions and they have side effects. And this is literally what I spend my life doing. And so, I think that if you're getting to that point, then that's where a referral to, you know, a specialist for AFib is important. So, propafenone and flecainide are in the same class, which is 1C and they have very similar side effects. Then, the other class of drugs that we

usually use for this are the class 3 agents, which are sotalol or dofetilide, Tikosyn.

Host: So, you said earlier that everybody probably deserves a chance at least one cardioversion, if I heard you correctly.

Ben D'Souza, MD: That's correct, yes. That's one chance at sinus rhythm.

Host: Yeah. And is that chemical or electrical or just one of those options?

Ben D'Souza, MD: Yeah. So, the likelihood of a conversion to sinus with drugs, so amiodarone is quite uncommon. I'd say maybe 10-15%. Flecainide and propafenone, maybe a little bit higher, like 20%. The drugs that actually have the best chance of cardioversion chemically are dofetilide, Tikosyn or sotalol that work on the potassium channels. And those drugs, they can prolong the QT interval, which is the reason that we hospitalize those patients. But they're about a 30-40% chance of chemical cardioversion. But I also want to be clear, especially for those who round in the hospital, if you start any of these drugs on patients and you don't know their duration of anticoagulation, if they convert to sinus, they have the same risk of having a stroke as if you electrically cardiovert them. So, you just have to be careful. Patient comes in with new-onset AFib, unknown duration, you start on my amiodarone in the ER or on the floor, and they convert to sinus and have a stroke, you basically cardioverted them. And so, you have to make sure that you're not putting your patients at undue risk. In fact, I've seen that happen. The cardioversion that I'm largely referring to is an electrical cardioversion in terms of getting patients out of AFib.

There is a drug that we don't really use is called ibutilide that you can use to IV cardiovert somebody. But I don't even think it's on formulary at Penn to be honest. So, you know, we talk about cardioversion. I can't tell you how many times, by the way, patients come in and they say, "I came into the ER. I got a medicine. IV metoprolol, IV Lopressor, IV diltiazem. And it converted me to sinus." It did not. Those drugs do not cause a restoration of sinus rhythm. Your heart went out of it on its own. All it did was control the heart rate until we did that. So, those drugs just control heart rate. They don't control heart rhythm. It's a common misconception. And I try to explain that to the patients. And so, those medicines don't do that. All they do is prevent the rapid ventricular rates or RVR, make the patients somewhat more, you know, less symptomatic until their heart goes out of it longer term.

Host: So, if you fail cardioversion, then they're in your office and this whole question of catheter ablation comes up. I understand we are still doing the maze procedure. Can you take us through that? So, you have a patient who's in AFib, has symptoms, you know, you don't want to leave them in their current state and you want to do a procedure.

Ben D'Souza, MD: Yeah. So, I joke with all my patients, if I knew what caused atrial fibrillation, I'd have the biggest house here on the main line and I wouldn't, you know, have to be working most weekdays. But unfortunately, I and nobody knows what causes atrial fibrillation. But interestingly, we do know where in the heart it comes from. So, the majority of atrial fibrillation actually comes in the left atrium. So, the pulmonary veins where they insert to the left atrium embryologically are different than the actual left atrial body. And we think that that anatomic difference is why we get atrial fibrillation in that area. We think.

There's a couple of other theories that there are nerve ganglion that are on the outside of the heart that are there as well. We don't really know to be honest. There's lots of different theories and lot of research that is being done among other places. But what we found was that the Cox-maze procedure was a very successful procedure to be able to keep patients out of atrial fibrillation. It is a surgical procedure in which the surgeons would put stitches and basically compartmentalize the entire left and right atrium. It actually was very successful. It was like more than 90% successful, but at a high rate of complications, especially when they did the stitches on the right side of the heart, because they caused sinus node dysfunction and heart block, and a lot of those patients needed pacemakers.

So, catheter ablation was essentially developed to recreate that surgical procedure, but without an open heart surgery. And so, you're correct, Kendal. We still do the maze procedure. Surgeons, if they're going to the hospital or operating room for valve surgery otherwise and have AFib, they'll do it. We don't routinely do it as a de novo procedure, meaning send the patients for open heart surgery because we can do it with a catheter procedure.

So, catheter ablation has sort of taken off over the course of the last few years. And again, it's a catheter procedure in which we place catheters in the groin, go up and cross over from the right to the left atrium and basically, electrically isolate that abnormal tissue around the pulmonary veins. That's why it's referred to as pulmonary vein isolation. There's two main ways to do it. There's actually a third way that's brand, brand new called pulse field ablation, which is like the new hot topic in EP. But essentially, radiofrequency ablation, which is burning

or cryoablation, which is freezing, different ways to basically isolate electrically abnormal tissue. And so, that's pretty much what we do in the EP lab. Success rate of drugs for PAF are about 40-50%, the ones that we talked about earlier, flecainide, propafenone, Tikosyn or amiodarone, et cetera. Success rate of catheter ablation for paroxysmal atrial fibrillation is about 80-90%. So, it's about twice as good. It's not a hundred percent, but it's pretty good.

Now, persistent atrial fibrillation is a little bit of a different story and success rates of that go down. Depending on the clinical trial and the patient, it can be anywhere between 60-70% depending and sometimes requires multiple procedures or additional medications. So, my point earlier is letting your patient wait until they get to persistent atrial fibrillation has caused them a disservice in terms of what I can offer them or anyone can offer them in terms of ablation. And so, many people looked at ablation as sort of a black box and that it didn't work, and that would've had a high risk of complications.

Well, CABANA was a trial that came out fairly recently. It was a randomized trial that looked at AFib ablation versus drugs. It did not meet the endpoint of mortality because the very first thing I said, atrial fibrillation is not life-threatening. So if you're trying to get a mortality benefit out of AFib, you're not going to get it because the patients are not dying from it. But it did show that catheter ablation was superior to drugs in terms of quality of life, heart failure, hospitalizations and side effects actually. The side effect profile from meds is significantly higher. I generally will quote between a 1 and 3% risk of anything happening with catheter ablation. It's a safe procedure. In fact, I and the folks at Penn were the first ones to send patients home the same day from this procedure. I send 90% of the patients home the same day, come in the morning, have the procedure, go home that afternoon. And they do quite well with it. And I think it is an option for patients. But it's a class 1A indication for patients who have PAF who have failed a drug. And despite that, the majority of the patients that are sent to us have tried multiple drugs or are persistent. And we're not, again, doing our patients the best of what we can by offering them this therapy. And so, it is very much the future.

I very briefly mentioned pulse field ablation. So, I was lucky enough to be the first one in the state of Pennsylvania to do a PFA trial on a patient. And so, pulse field ablation is a technology that actually was invented for cancer patients. So, it's basically non-thermal energy that's delivered to the heart. And it actually helped to shrink cancer tissue. But it turns out that cardiac myocytes are very sensitive to this. So, it's really fascinating because ablation, meaning when you burn or freeze tissue, there are collateral structures that you can damage. Behind the heart is the esophagus. In front of the heart is the phrenic.

You damage the phrenic nerve, you lose one of your diaphragms, it's not good. If you damage the esophagus, you can have something that is a very life-threatening complication of ablation called an atrial-esophageal fistula, AE fistula. It's absolutely horrible. So, this therapy may completely make that go away and it's actually really fascinating. I think sometimes in medicine when we apply technology or something from a different field to ours, that's how we really revolutionize the field. So anyway, it's still early on. We just finished the FDA trials, and we were a part of it. It's really, really neat and cool and exciting to be a part of it, which is why I love working in a place like where we work.

My point is that technology continues to expand. So if you use the AFFIRM trial to make your decision as to what you're going to do, you're taking a trial that's now 20 years old, 2003, I believe, and making decisions when technology has been revolutionized over the course of the 20 years. And I think that it's important that you continue to either keep up with the data or send them to the people who literally just do this for a living to make sure that they're getting, you know, the best therapy possible. It's very much an option for patients who have symptomatic AFib who've either failed a drug or don't want to be on medicines forever.

Host: So when we talk about 70-90% effective, that's what I had read and I think you quoted something similar for catheter ablation, those patients are effectively cured of atrial fibrillation?

Ben D'Souza, MD: Yeah. That data is more than one year post without any medications. Now, it's hard to ever use the word cure, but yes. There's actually an interesting trial that's going on right now where we implant loop monitors in patients post AFib ablation and stop their anticoagulation. The current guidelines say that you're supposed to stay on anticoagulation even after catheter ablation based on your CHADS score.

Well, the number one reason that patient has come to me, number one or two, is can I stop my NOAC or Coumadin if I have an ablation? And the answer is maybe. So, I think eventually it will make it into the guidelines. The trial's ongoing right now, it's really fascinating. But I do do that in clinical practice and many docs do do that. And I put loops in either at the time of the ablation or in the future and stop pretty much all their medicines. And so, that's exactly right.

Host: So, patients will leave your ablation lab off anticoagulation with an implantable--

Ben D'Souza, MD: No. Good question. The first three months is the highest risk of a stroke forming after a catheter ablation, just because there's catheters in the heart, they're stunning in the heart, even more so than after a cardioversion, right? So, cardioversion, the risk of stroke is not just because of the blood clot that's there in the left atrial appendage, which is where more than 90% of blood clots form. It's that the atria are kind of stunned. They're not squeezing quite yet. And then, the blood stagnates, you know, Virchow's triad. And then, it goes to the brain and that's how you have a stroke.

But again, because 90 plus percent of blood clots form in the left atrial appendage, there's also another procedure that we do called left atrial appendage closure or a Watchman procedure. There's actually more companies that developed it. It's an option for patients who have non-valvular atrial fibrillation who have an elevated CHADS score who can't be on anticoagulation, so stroke or whatever, but GI bleeding, bleeding anywhere essentially, thrombocytopenia, cancer, falls due to Parkinson's, et cetera.

Interestingly, we ran two clinical trials at Penn where we basically randomized patients to preference, anticoagulation or left atrial appendage closure. And in the future, it is possible, and more than likely, the two trials were called OPTION and CHAMPION, I ran them both at Penn, that patients will get to choose. They'll say, "I'm either going to stay on Eliquis for the rest of my life," or "I'm going to have this procedure called a Watchman procedure." And so, that to me is very interesting. And there may be patients that decide, "I don't want to be on anticoagulation." In fact, the majority of people don't want to be on anticoagulation. And so, I think that's a procedure that I do as well for patients. You know, I just did a couple of them the other day, patients that just can't be on anticoagulation. And so if you have a patient that can't be on anticoagulation, just saying, well, just accept your whatever percentage risk of stroke, there's actually an option for those patients, and that's a left atrial appendage closure device like a Watchman procedure.

Host: Those patients remain in AFib with Watchman, right? Like you're not curing the AFib like you potentially are with ablation. They're remaining in AFib. It's just that you're basically-- This is a simple way of thinking about it-- I guess you're keeping the clots from leaving the left atrium and blocking them, right?

Ben D'Souza, MD: Yeah. No, that's exactly right. Basically, if you look at the device, it looks like an umbrella or a parachute and you're exactly right. What happens is the body endothelializes that front of the fabric, which is made of Gore-Tex or PTFE. And essentially, it's like the left atrial appendage never

existed. Left atrial appendage, it's a vestigial organ. It's kind of like your appendix, but it's in your heart. It serves no purpose that we know of. But that's where the majority of the blood clots form. If you ever looked at it, it's very trabeculated. It's actually a beautiful place for a blood clot to form. And so, that's why we basically seal it off. But you're exactly right, Kendal, the number one patient who's referred to me for Watchman, they say, "Well, this is going to fix my AFib, right?" And the answer is no, it's not treating your AFib. It's just preventing blood clots in patients who can't be on anticoagulation.

We're only recently looking at the possibility of doing both procedures in the patient at the same time, combination of AFib ablation and left atrial appendage closure. Because then, you get them off of all their medicines, you treat their AFib, you prevent the progression of AFib, prevent strokes. We, again, were one of the first ones to do this in the country at Penn, and we're looking at that. And unfortunately, the main reason that we don't do it is the insurance companies will only pay for one procedure in a patient at a time. And so, the hospital actually gets stuck with a bill if we do that. But for a subset of patients, it's the right thing to do. And I mentioned one of those clinical trials called the OPTION trial, which data will come out hopefully in the near future and show that it'll be the right thing to do and Medicare will pay for it and we'll be able to offer it to our patients. Because that's really a huge future for our patients, not to have to be on any medicines at all for AFib, including anticoagulation. But typically, we kind of stratify this. Symptoms of AFib, treat the symptoms of AFib, stroke prevention, treat, stroke prevention.

Host: I want to go back to the ablation question in anticoagulation. You had mentioned that three months out they have to be on it.

Ben D'Souza, MD: Minimum, yeah.

Host: And then, there is a thinking that you can do monitoring with no anticoagulation,

Ben D'Souza, MD: Right. And this is kind of a controversial part of the field because it's not technically in the guidelines. So if you ask five EPs, they'll give you five different answers. So, many of my patients, they're going to have an AFib ablation, but they also need another surgical procedure or something else.

I say, if there's a timing, make sure you do that other procedure first, because at a a very minimum, six weeks after the procedure, I'm going to say you cannot interrupt your anticoagulation at all. And in fact, this is probably the number one question I get asked. I just saw a bunch of patients today, "I'm having a

dental procedure. I need to hold my anticoagulation for how long? You know, how long is it okay?" The answer is there's not a right answer. We don't really bridge patients anymore for AFib, unless they have a mechanical valve.

So, the data's actually in, and it's in the ACC/AHA guidelines, is that we don't bridge patients with Lovenox or heparin who are going to have a surgical procedure. But I typically will say, "You can hold the med for up to three days." Some people will say that they told me to hold it for seven days for teeth cleaning. Well, the halflife of the drug gets completely washed out within 48 hours, certainly within 72. And I do tend to clear my patients to have it done. It is always a little bit of a risk anytime we do that. It's not a huge risk, but it's there. There's some data that suggests in some of the novel anticoagulants, when you stop it, there's a rebound prothrombotic effect. And so, you do have to keep that in mind. That being said, you have to weigh the risk of bleeding versus anticoagulation. For something like a teeth cleaning, it's probably not higher risk. Obviously, for biopsies from a colonoscopy or prostate biopsies or something where it potentially is a higher risk, you have to weigh that.

Now, the surgeon who's going to do their procedure, they care about bleeding risks. I care about stroke risk. And so, it's sort of a competing factors. So, I just wanted to bring that up because it's a very common question that my patients ask about. But you're a right, Kendal, is that we don't stop any coagulation in the first few months generally and close monitoring is required. If you're going to stop. I typically wait till close to a year. Because it's a nice clinical timeframe that says, "You've done well, you haven't had anything. Let's talk about this." And some patients are like, "I'm terrified of having a stroke. I want to stay on my NOAC." It's fine with me. It's all patient based.

Many of my patients ask, "Which is the best NOAC?" There's not a good answer to that question because there's never been a head-to-head study of all of them. Eliquis probably has the best decreased hemorrhagic stroke risk, and it's tend to be used. But honestly, I tell my patients whichever one's covered by your insurance. So, Xarelto or rivaroxaban costs $20 and the other one costs 200, choose the cheaper one because there's no data that suggests that one is better than the other. Pradaxa or dabigatran will be the first drug that will be generic. And so, we're getting to the point based on FDA approval that that'll be the first one. Pradaxa causes the most dyspepsia or GI symptoms. So, it's a little bit harder for the patients today, but I pretty much recommend any of the other drugs, you know, over warfarin or Coumadin if at all possible.

Host: So, I want to go back to these procedures. I'm a little bit focused on these procedures. I understand catheter ablation. You're basically curing them. I

understand the Watchman procedure. This is for patients that are in atrial fib. They may have failed catheter ablation, but they want to get off or they can't be on anticoagulation. And so, there's this option and it sounds safe and effective.

The next procedure that I think is just worth talking about is AV nodal ablation with pacemaker insertion, which always seemed to me to be the most like the biggest commitment of all. So,

can you talk to us about that?

Ben D'Souza, MD: Yeah. Let's talk about that. And so, traditionally when AFib ablation was not as successful as it is now, many docs would provide this. In general, the way that I would think about it is if you've exhausted every other option that we've talked about earlier, or if you're too sick to have any of these options, right? You have a 90-year-old sweet patient who has fib with RVR despite beta blockers, calcium channel blockers, digoxin, amiodarone, et cetera, and is symptomatic with it. Then, an AV node ablation and a pacemaker is an option.

What are the risks? They're generally low, but you have now made a patient pacemaker-dependent, right? And so, God forbid, if anything happens with that pacemaker, it is life-threatening. That's rare. That is quite uncommon. The patients I'm referring to are older, sicker people. The ablation procedure I'd referred to earlier, an AFib ablation, you do have to go under general anesthesia for that, and you do have to be able to tolerate it. Depending on the procedure, it could take a few hours. And so, you have to have a patient that's healthy enough to tolerate it. You also have to have a patient that can tolerate anticoagulation. So if you have a patient that's truly contraindicated, brain bleed that neurologist says they can't be on heparin for even, you know, a few minutes, and they can't be a candidate for left atrial appendage closure, then this may be an option. But I want to be clear that doesn't obviate their stroke risk. They'll still be at a stroke risk because they're in AFib. Many people think that if you get an AV node ablation and a pacemaker, you don't have a stroke risk anymore because you're synchronizing, but you're not. You're synchronizing the ventricle, not the atrium. The atrium's still in AFib and so, you're still at risk of blood clot or stroke. So, we don't do too many AV node ablations in pacemakers. They're really for refractory, chronic atrial fibrillation or permanent atrial fibrillation, we again use chronic inserted permanent, that we don't have any other options for. So, you have a patient that's on the ACE unit and we can't control them otherwise, and they're having heart failure, they're having issues, then we'll go ahead and put in a pacemaker.

And actually interesting, more recently, we've been doing something called physiologic pacing, or left bundle pacing, which is instead traditionally when a lead went into the heart and went to the artery apex, the bottom of the heart. Well, the electrical conduction actually starts at the base of the heart and goes down to the apex. And so now, for the last few years, we actually screw that lead into the base and it kind of delivers more physiologic pacing. Because sometimes, depending on the pacemaker type, you can develop a cardiomyopathy. But this is a rate control strategy, not a rhythm control strategy in patients who we just don't have any other options. So, we do it less and less frequently than we used to, but still an option for patients.

Host: so, Ben, back in the day, atrial flutter was considered different from atrial fib. I believe everything we've said about atrial fib probably translates to a flutter as well, but educate us on that.

Ben D'Souza, MD: Yeah, of course. So, 30% of patients who have atrial fibrillation have atrial flutter. I tell people they're like cousins. And so, the stroke risk is the same. The anticoagulation indication is the same. However, atrial fibrillation is even more amenable to catheter ablation, atrial flutter that is, than atrial fibrillation. Generally, when we refer to flutter, we're referring to CTI flutter, cavotricuspid isthmus-dependent flutter, right-sided flutter. The concept is the IVC has no electrical tissue. The tricuspid valve has no electrical tissue. But the area in between them, meaning the CTI, does. So, you set up a sort of a circuit in the heart that we all have, but for some reason it's the perfect way to set up what's called a reentry rhythm or basically a spinning around in the circle on the right side. Now, if you've had a heart surgery or a previous ablation, that can occur anywhere, not just on the right side.

But in general, it is a class 1A indication for first-line ablation for atrial flutter, meaning over cardioversion for two reasons. One, the risk of recurrence of atrial flutter is even higher than with atrial fibrillation. Just whenever you have that circuit, you get locked into it. And two, and it is in the guidelines, you could stop anticoagulation in patients who have had an atrial flutter ablation. We typically do the same like two to three months after. But to commit a patient to lifelong anticoagulation after you cardiovert them from flutter or you could cure it-- and I use the word cure with flutter-- and then get them off of anticoagulation. Again, I still do monitoring those patients because 30% of them can have AFib. You don't want to miss that. I just had a patient that I did their flutter ablation. I put a loop monitor in. He's two months out and he has a little bit of AFib. He doesn't feel it, so we're not going to worry about it, but I'm not stopping his Eliquis because he could have had a stroke, not because of flutter, because of AFib.

And when you see atrial flutter on an EKG, that is a get-them-to-one-of-us because we can cure that. And they're even harder to control with medications. Rate control is almost impossible in those patients, because they're kind of locked into a macro-reentry circuit, and it's not going away. And so, anticoagulation is the same. Medication choices are the same, but catheter ablation is much more amenable, to the point where if you see a patient in the hospital, call me about them and I'll do their ablation before they even leave the hospital, because I can cure it for him. It's a very straightforward 30-minute procedure, same day, that kind of stuff.

So, I think the technology has made us improve so much with this. I just want to share a very brief story. The person who invented a lot of the mapping systems that we do, it's called eletroanatomic mapping. He was a physician and an Israeli soldier, and he actually figured out that we can track soldiers out in the field without seeing them. How do we do that in the heart? And we put these little magnets on catheters and we put patches on the patient's chest magnet and it revolutionized our field. Things that used to take eight hours, take less than one hour. Things that took hundred minutes of x-ray, use no x-ray now.

And so, we're lucky and blessed that we have all these new toys and fun stuff at Penn that we can do all of this with. But also, it's changed the field in terms of treatment options. And I think that for folks who think of this from when it first started, say 20 plus years ago, it's not even close to the same options and treatment. And it's going to continue to grow. I was just at the Heart Rhythm Society last week and I'm learning new stuff everyday, which is exciting and it's a good time to be in the field, but it's complicated. And so, it makes it hard to know all of this stuff, especially if you don't do it for a living. Even if you do it for a living, it's hard to do.

Host: I'm glad you said it's complicated because that's the reason why I invited you on because I was like, "This is starting to get really complicated." Ben, this is great. We're up against the time. I actually want to bring you back at another time because I do want to talk about some of the ventricular arrhythmia stuff. Maybe we can get into some of the SVT stuff. But just to talk about also, I had a final question I was going to pose to you about congestive heart failure and atrial fibrillation. But I think it's probably just going to take us down a place that we need to have another show.

Ben D'Souza, MD: Sure. Well, I'll say a very brief answer to that is that patients who have heart failure and AFib, they really don't tolerate AFib. And the data is actually clear in a mortality standpoint that you should really be aggressive about treating those patients. So if you have a patient with either

HFpEF or HFrEF, reduced ejection fraction or preserved ejection fraction AFib, don't sit on those patients. You're really, really doing them a disservice. So, that's the short version. But yes, we can go down the rabbit hole next time.

Host: And that's a great place to end. Thanks, Ben, so much for coming. I really appreciate it. I'm sure the audience appreciates it. This is really educational and very helpful. I imagine we'll have some followup questions. Thank you to the audience for joining the Penn Primary Care podcast. Please come back again next time.