SPEAKER 1: Thanks. Great. So I'm going to be talking to you about atrial fibrillation, which is a large percentage of my practice. I would imagine it's a large percentage of all of our practices, but it's become a real focus in electrophysiology. And so much has changed in a-fib compared to what when we were training or what we have to offer patients even 10 years ago.

So it feels like a really exciting area of electrophysiology. What do I need to point to that, this? Is this the advance?

[SIDE CONVERSATION]

So I have probably more slides than I need today, so I'm going to move through some of the ones that you're already pretty familiar with quickly. We know it's the most common rhythm disorder, and it affects something like 15% of the population over the age of 70. It causes very significant morbidity and mortality. And it impairs life, quality of life by the palpitations of the teeth and the dyspnea, and it's independently associated with the risk of stroke, heart failure, and death.

You've probably seen this graph a million times basically just showing-- I'm just curious to see, is that the little light there-- basically just showing that as our population ages, we get a huge spike in a-fib pretty centered around the '70s and '80s, and I think that's all of our experiences in our offices. Depending on which study you look at, the calculations for how many people are going to have a-fib by the time we get up to 2040 and 2050 depends on which study you pick, but anywhere from 5.6 to 15.9 million people with atrial fibrulation. So this is a growing, huge issue in the United States-- actually, worldwide.

There are a couple interesting findings. For one thing, a-fib is much more prevalent in men than it is in women. We don't really know why that is, and it's also more prevalent in whites than it is in African Americans.

And again, we don't really know why that association exists, but it does. As an electrophysiologist, one of the first things that I do when I'm meeting a patient with a-fib is I write it in the chart, or I characterize it in my mind. But I want to know what stage of a-fib they're in. And for electrophysiologists, there are four phases.

The first is the paroxysmal stage. These are people who have brief, self-limited episodes. They don't require a cardioversion. They don't require a drug.

The episodes come and go. That's the most favorable stage, in terms of any treatment that we want to do. They're the most responsive.

The second stage is called persistent. And persistent fibrulators, once they go into fib, they're stuck. They need something to get them out, either a drug as an intervention or a cardio version.

The third stage is a new category. We're calling it longstanding persistence. So these are people who have gone into fib, and they're staying in fib. It's an arbitrary decision.

It hasn't been a year. So if they're in a-fib continuously, but it hasn't been a year, we'll call them a longstanding persistent. And once they're chronic, then it pretty much means they're refractory to anything we might try.

So once somebody's been in it for five years, they're not going to very successful at getting the person out, whether you've given ablation or you've used drugs. And usually, I've heard electrophysiologists characterize chronic as when they feel like they've exhausted all their possibilities. I don't know.

Are you familiar with the Zio patch? It's my favorite monitor. We've had a lot of issues trying to get it through various insurances, but I think we've resolved that now.

The patients love it. It's basically a big Band Aid that we stick over their left side of the chest. They sleep with it. They shower with it.

There are no wires. There's nothing attached, just a little bubbble to the center, where the middle of it's raised. And they can actually click that, if they're feeling any symptoms.

But basically, they work for 14 days, and then they peel it off and mail it back. It's a 14 day holter, and the tracings are really clear.

So this is a fantastic way to try and capture what's going on. And I don't know about you, but when a patient says to me they're having palpitations, I don't know what rhythm that is. And I don't trust [INAUDIBLE].

If they say, oh, just a couple skipped beats, then I get a monitor, and they're billing 180 all the time, and they don't know it, or when they say, that's absolutely terrible, and then I get the monitor back, and there's three frequencies. So you can't go by symptoms. You need a recording.

So that's a fantastic way to record. I think it's much more difficult than the asymptomatic patient. And I say fully 50% of the a-fib patients I see have almost no symptoms. It's just discovered coincidentally at a doctor's office visit.

For those people, I don't know if you've heard about our new little implanted monitor, this guy. It's called The LINQ. It's incredibly tiny.

So this was an implanted recorder we've been using for years. But in order to implant that, it was a little bit more like a pacemaker surgery. You have it in the pocket and sew it in.

This guy is injected, so I simply clean the skin, numb it up, just make a little nip in the skin and inject it, kind of like what was it, [INAUDIBLE]? It's a little cylinder. It works for three years. It's wireless.

They have a transmitter at home. And basically, every 24 hours, if they walk by the transmitter once, so we have to put it in their bedroom, it will download any data that's in the plate, and it will send it to the website. So every morning, when we come in, we open up the website, and we can see who's had a-fib.

So for us, it's a fantastic way to track all the asymptomatic people, because otherwise, if you're relying on their reporting, you really don't know, has the drug been successful? Has your operation been successful? This thing is amazing, and the patient doesn't have to worry about trying to activate it when they have symptoms.

And as I said, three years. They can take the transmitter with them, if they go on vacation.

What's it called?

It's called a LINQ, L-I-N-Q. But they call these implantable loop recorders, or ILRs. But this one is super tiny. Only Medtronic is making this really small one right now.

So things that are usually worked out before I even meet the patient, whether it's something that's secondary causes that look for. Of course, thyroid function tests-- although, it's a fairly rare cause. Usually, the primary problem in the US is hypertension. And if that seems difficult to diagnose, or there is a suggestion via echo-- in other words, hypertrophy or a big left atrium. But no obvious hypertension, maybe an ambulatory blood pressure cup would be a way to go.

Sleep study in appropriate patients-- this is turning out to be a really interesting cause of a-fib. It's pretty significant. And when I get to some of the new techniques that they're doing, I'll show you something that is unique to people with sleep apnea. And if that's appropriate, maybe a sleep study.

Everyone using echo, it would be really helpful if you're going to make a referral, to order the echo fib, because that's what we're going to order first visit, because we need to know about structural heart disease. I need to know about left atrial size. You need to know about LB function. No decision that I make can happen, until I have the echo.

Stress test-- only if that's appropriate-- so risk factors, a chest pain. You don't need that in every one. And then in light of how complicated it's become, because of ablation, because of anti-arrhythmc drugs, it might be appropriate to consider a specialist referral.

After I decided the stage, I've made the diagnosis by recording. I've gotten my quick look for secondary causes. One of the first things I'm going to determine is, what is their stroke risk?

So on my office charts, I have the CHADS-VASc score just running down the side. I had this before Epic, so I don't know if I can get that put into Epic. But it's pretty helpful. So you can just add the score right when the patient's in the room. Based on the score, we're going to select either aspirin, coumadin, or one of the novel agents, very rarely dual platelet therapy if there's [INAUDIBLE] to a more traditional anticoagulant.

This is a comparison of the CHADS score and the CHADS-VASc score. People towards CHADS-VASc. Traditionally, we just use CHADS, and anything greater than 1 was considered to be a significant [INAUDIBLE] stroke, and that's something to think about-- Coumadin, or Pradaxa, or Xarelto, or Eliquis-- whatever your choice was.

With the CHADS-VASc score, it has not been as easily defined. Like, you can see that CHADS score 1, gives you a 2.8% risk. CHADS-VASc score 3, gives you a 3.3% risk. So we kind of use a CHADS-VASc 3 as the break point for when you go over there, including a lot of novel agents.

So, strokes are obviously the most dreaded complication of AFib, and it's been determined that about 15% of all strokes are due to AFib. I actually thought it was higher that. I'm somewhat surprised at that number, but they are very significant, and it turns out that strokes that are due to AFib are some of the worst strokes. Because the Clots are pretty big, so you get a pretty big middle cerebral artery, typically, stroke.

This is two different neurology scores I'm not so familiar with. The NIHSS score is an assessment they hop out of patients just when they arrive. And you can see, if you compare the lacunar strokes, atherothrombotic strokes, and cardioembolic strokes, these patients present with much more deficit, and they also have-- this is an mRS score that determines their outcome-- and you can see that lacunar strokes have a fairly good outcome, but the cardioembolic strokes often leave the hospital with a burden-- significant deficit. So it's a large clot if it occurs, and we would just like to have it not happen at all.

When I gave the grand rounds, Dr. Gershony wanted me to really focus a lot on the different novel agents. I'm going to kind of go through them fairly quickly. These are all New England Journal of Medicine's studies that, the least of all of these drugs, dabigatran, or Pradaxa, was the RE-LY study. And you can see that they compared to Coumadin in gray, dabigatran at a low dose, and dabigatran at a high dose, 150 milligrams. But 110 milligrams was never released in the US, so they only really have it in Europe. So we use 150 milligrams. dabigatran's the only direct thrombin inhibitor. The other two drugs are Xa inhibitors. So we have rivaroxaban-- same kind of graph-- and Eliquis, essentially the graph. So all of them were significantly better than Coumadin in terms of preventing strokes.

The issue has been fleeting, and in part, whenever a new drug comes out, there's always a lot laser beam focus. You have a lot of hemorrhages on Coumadin too. The problem with Coumadin is we often have those people that get too high and then have a hemorrhage, or people that get too low, and clot. You're constantly trying to adjust. If they affect anything with their [INAUDIBLE], you could have a three- or four-week period in which you don't really know if their I amount is low or their I amount is high. So I really, strongly prefer the novel agents if I can get it through the insurance company.

I pretty much never use Pradaxa, dabigatran, mainly because of its side effects with your PI, so when we started using it, when it's the first drug that was available, I kept getting these calls from patients that they were having chest pain. I couldn't figure it out. Everybody was having chest pain. But once it became clear that the vehicle that Pradaxis is built into is very acidic, it's GI. It's all acid reflux symptoms. I can't use that because I'm doing ablation, and I'm trying to be very careful about the esophagus, which I'll pop about seven. For us, Pradaxis is kind of out.

So we use either Xarelto, rivaroxaban, or we use apixaban. I'm kind of leaning towards apixaban. And the patient-- they may have a little renal insufficiency. I lean towards Xarelto if somebody's sort of saying I just want once a week, or a little more [INAUDIBLE]. But they're pretty similar, in my opinion.

All the agents showed a very significant decrease in intracranial hemorrhages compared to Coumadin. Only apixaban also showed a reduction in all major bleeding as compared to Coumadin. T Even though that's sort of not the buzz, right? The buzz from the hospital, that says, oh my god, that sounds so horrible. Everybody has terrible GIBs. And I think we saw that a lot-- just when Pradaxa [INAUDIBLE] now, but I think if you really use big trial data, there isn't an increase in [INAUDIBLE] GI hemorrhage with these strokes.

They are potent in the fibrins, and they work very quickly. You have to be very careful to make sure you're discontinuing somebody's aspirin. The combination of the novel agent and aspirin definitely is a dangerous [INAUDIBLE]. And the drug that gets discontinued the most is Pradaxa because of the GI side effects that are just removed.

The big argument is, well, there's no antidote. And there isn't. There is no FDA-approved antidote. We can reverse Xarelto and Eliquis by using Prothrombin Coagulation Complex, PCC. They do have it at the hospital. It's really expensive. For the most part, these drugs have such a short half-life that you normally don't need to actually reverse. You can give it 24 hours, and it will recover. If there's a car accident and their spleen is hemorrhaging, we're going to have to do surgery, no matter what.

If there is a situation that's a little life-threatening, of course, we can get the PCC. And we actually usually put that in our room when we're doing ablation, in case after bed, you perforate something, you are ready, but we usually just return it to the pharmacy unopened, because I think it will be somewhere between $75,000 and $100,000 per dose. Vitamin K will not work. Yeah?

AUDIENCE: So will the [INAUDIBLE].

SUSAN EISENBERG: No, not approved. There is no approved.

AUDIENCE: So they're just using it--

SUSAN EISENBERG: Because a couple of studies have come out that there's nothing FDA's proved for that indication. And Brad Lewis, who is a good hematologist, was over at my building on Grant Street, talking to the primary character there, and he also talked about PCC. So to know amongst hematologists is a way to reverse, but it is not approved.

AUDIENCE: It's not common?

SUSAN EISENBERG: No, I would say that almost nobody has used it in the hospital. But it is there. We do have it if we need it, but usually if you can give it 12 to 24 hours, there's just not enough [INAUDIBLE] to be a concern. I don't know if you have seen this, like with surgeons who are asking you, what do I do with the blood thinner? They're holding it for five days. Totally unnecessary. And I think I already told them that it's gone at 24 hours. You don't need five days. You're just exposing the patient to surplus during that period of time, and as soon as you start it, you will be [INAUDIBLE]. So if this is any kind of spinal procedure, like a steroid injection, whatever, you probably shouldn't start it that night [INAUDIBLE].

OK. For a while, there was no study published on doing a cardioversion on these drugs. So were you allowed to? But yes, there have now been a couple of studies showing that there was no increase in risk, but only if you used a novel agent for Coumadin, if you were doing a cardioversion or doing an ablation. So we can use them all interchangeably.

So that's it for blood thinning, but the next decision point for me is am I going rhythm control, or am I going rate control? And a lot of people quote this fairly old study, the AFFIRM study-- 2002-- in which they compared a rhythm strategy to a rate strategy. And they said, see, there's no difference-- there's no difference in mortality. And I think that has been used as an argument for many years why we don't really need to do anything except control the rate.

And I really disagree with that. I think it's great to know that there's no difference in mortality. If we're controlling the rate, and the person's still [INAUDIBLE] they will live a nice long life if they're appropriately anticoagulated. And similarly, they will if they're doing the rhythm control arm.

But what this study didn't even address is simplest. So to me, this study is irrelevant when I'm talking to an active person-- an active person in their 60s or 70s who still goes out and exercises. They are not going to feel as well in fifth as they feel in sinus rhythm. So of course, I have a bias. I want sinus rhythm. If you send me the 85-year-old person home patient who really just lives in a wheelchair, no, we don't need to get in the whole rhythm control strategy. At that point, rate control would be important.

So, as I just said, rate control typically are older, more asymptomatic patients. They have to have more advanced fibrillation by the time they show up. More structural heart disease-- they've perhaps had previous other attempts at restoring sinus rhythm but have been successful, or they have other comorbidities-- cancer, what have you-- that are more relevant and getting involved in, and they push for sinus rhythm.

The one thing I will say about AV nodal blockers is that I don't think there's any rope with digoxin. None. It's a terrible AV nodal blocker, and then their rate is controlled sitting [INAUDIBLE] in an office, I can take it they'll walk out of the office, and they'll go down the stairs, and their heart rate will be high. So the drug is completely overwhelmed by any surge of adrenaline. It's a poor rate control drug, and if you keep up on your dose, they have toxicity. So my preference is always for calcium channels or beta blockers-- either/or.

If we're going to go with a rate control strategy, which is perfectly fine, I do think you're obligated to demonstrate that you have good heart rate control. So again, what they have sitting in the office is probably not relevant. So once you've decided on that, you've chosen your AV nodal blocker, put on a Holter. Put it on and see the impact-- something to show that when they're out there walking, their heart rate isn't super high all the time.

If you really can't control rate-- and sometimes we get into this scenario, again, with the older patients-- if we get the heart rate controlled adequately, then they start having pauses, or their blood pressure is too low to tolerate the drug. Sometimes you're just stuck. As much as you're trying to control the rate, you cant.

And then in that scenario, think about putting in an AV nodal ablation pacemaker. It's very straightforward. You put in a pacemaker. It's 40 minutes. And then we basically heat the AV node-- the ablation catheter-- for about 60 seconds, and that gives them, basically, a complete heart pump. Then they're never going to race. We don't need to use any nodal blocker. The pacemaker will give you all the heart rate variability you want. You still have to anticoagulate, but you've controlled the heart rate. Is that familiar to everybody?

Now, what about the rhythm control arm? This is going to be probably our younger fibrillation patients. Less structural heart disease. They often start very paroxysmally, or have early persistent. Usually, the left atrium is not huge. It might be moderately enlarged. And these are patients are going to be willing to accept trying a drug, having a cardioversion-- maybe it doesn't work. Try another drug, have another cardioversion. Oh, now, let's go on to ablations. So it's a commitment. It's definitely a commitment.

In terms of anti-arrhythmic drugs, my way of thinking is that there are three groups of two. The first two drugs are the IC drugs, so flecainide and propafenone. [COUGH] Sorry. Those are only drugs that can be used, quote unquote, "pill-in-the-pocket." So that means that if you have a patient who has a rare episode of fib, you even give them some flecainide and other beta blocker, or calcium channel blocker. Make them carry it around like their emergency pills. And when they have an episode, they immediately pop 200 of flecainide plus a beta blocker, or, depending on which preparation of propafenone you're using, 300 milligrams of propafenone, and a beta blocker, and usually they'll convert within 30 minutes to an hour.

I tried really hard to have none of my [INAUDIBLE] AFib patients show who we are. So they all have this strategy. They all know what to do. If it doesn't work after an hour and a half, they call. You might repeat it once. And then if they're still fibrillating and they feel poorly, then obviously, they would go to the emergency room.

All the IC drugs, their effect is most potent at a high heart rate. So you know the issue with anti-arrhythmic drugs is that you might fix the fib, but what else are you going to provoke because they're all so tricky? So with flecainide and propafenone, what you might provoke is reheat. So the way we screen for that is once the patient's product at the dose, if they're taking it regularly, then I have them come in and run on the treadmill. I get their heart rate up as high as I can get it, and I look at the QRS because what happens with flecainide or propafenone toxicities, is it more or less gets wider, and wider, wider, and wider. So we basically measure, and if I'm really seeing a lot of widening, you want to stop their drug-- get off that drug.

Another important consideration is if your patient has ever had flutter, which is like the son of fibrillation, you cannot give a IC, because with flutter, which typically does 300, if you give a IC, which is a potent, slower, conduction, you might slow the flutter rate to 200. So at 300, your AV node knows not to conduct down the ventricle at 300-- it can't. But if you slow the flutter to 200, the AV node can do that. So oddly enough, you could give somebody flecainide, they go into flutter, and they go faster, because now they conduct 1:1. So once they've had flutter, IC [INAUDIBLE] happening. And of course-- remember your CAST trial-- you would never use it in anybody who's had an MI or has a cardiomyopathy. Has to be a nice, healthy heart. Does that seem clear? ICs?

The calcium channel blockers, sotalol and dofetilide-- these drugs, you have to be actually hospitalized to initiate. It's a huge turn-off for patients. So nobody wants to spend two days in the hospital getting a drug, and having [INAUDIBLE] after every dose. But what we do is we measure QT interval, because there are some people who genetically respond oddly to those drugs, and if their QT really prolongs, they're at risk for [INAUDIBLE]. So I don't use them a lot because it's a pain in the neck to admit somebody, and because I kind of feel like, what am I doing? I'm giving somebody a drug that can cause [INAUDIBLE] when all they had was AFib-- it's just not the best change I'd like to make.

The only time I use those drugs a lot is if they have a device. So if they have a pacemaker-- or a defibrillator, I should really say, but a pacemaker-- so that I know that at least they're never going to get bradycardia, because if QT prolongation is greatest, it'll lower their heart rates. So unlike the treadmill, these people, you want to see their QT numbers see to predict toxicity. And it's renally cleared, so if your patient as a known insufficiency, you can't use it.

The last category of two is the amiodarone-dronedarone combination. These are drugs that have multiple effects. Amiodarone, of course, is our most effective drug, no doubt about it, but what patient isn't a little upset when they go to the pharmacy and gets a-- you have a 12-page printout of toxicities, and side effects, and-- I often am trying to convince patients that they can use it in the short term, and that really, there are very few side effects short-term. It's a tenure issue for the pulmonary toxicity.

So if I'm keeping somebody on amiodarone long-term, then every six months, I'm going to check liver function. I'm going to check thyroid function. And once a year, I'm going to do the fusing capacity to make sure we're not [INAUDIBLE], which is the worst complication, and may or may not reverse when we stop the drug. So short-term use is great. Long-term use, not so good. There is our very few patients over the age of 85 who can handle amiodarone. They get unsteady, get really bad balance problems, and they fall. And now you've got them on a blood thinner, they fell, so it's either going to be a-- so I'm not a fan in the older patients.

Multaq, or dronedarone, is supposed to be amiodarone, but without the side effects, or without the toxicities. And it is. It's just hugely less effective. But it's very safe, so I don't mind trying Multaq at all. But it just doesn't work for a bad fibrillator. But it's a similar mechanism to amiodarone.

Any other questions or any questions on drugs before I move on to ablation? Yeah?

AUDIENCE: Ablation. [INAUDIBLE]. Is that the same with the novel agents? The [INAUDIBLE]

SUSAN EISENBERG: So I got rid of hypercoagulability with Coumadin. I hear about it all the time with novel agents. But again, I think that's for [INAUDIBLE]. So I think these are people who have high CHADS scores. When you stop whatever anticoagulation you've stopped, they're likely to have a stroke if you're leaving them unprotected. But there isn't anything that makes you unusually coagulable after stopping any of them.

The reason that beta came out was the Xarelto trial. We took really high CHADS score patients, like 3 and higher. And then they stopped the Xarelto at the end of the study, and switched them to Coumadin. You know, that takes like, two weeks. So these people were basically unprotected, and they had a lot of events during that changeover time, and so people start saying hypercoagulable. But we don't believe that you're attacked. You're hypercoagulant.

AUDIENCE: If you want to switch while slightly [INAUDIBLE] how many days would you have it moving this way?

SUSAN EISENBERG: Yeah, there's no really good answer to that question, so I'd budget a little, you know, depending on how high their CHADS score is. So if this is somebody I really won't want to ever leave uncovered, then I might go two days off of Coumadin and then start the novel agent. If it's somebody I feel like I can wait a little longer-- three days, sure. Let me know if their INR is not too high to begin with. Two days is usually adequate, but will be a little more overlap. There will be three.

OK, so let's talk a little bit about ablation. Who is a good candidate? We have typically said symptomatic patients. That's a little bit of a misnomer because there are an awful lot of patients that say, no, I don't feel it. I don't feel it at all. And then you get them in sinus rhythm, and they say, oh, I feel really different. So they have much better exercise capability. They know much where to graph. They just have a better sense of well-being.

So there aren't that many people that I meet that are truly asymptomatic. And if we're not sure, sometimes I give a drug in cardiobart, and that person feels sinus rhythm for a few weeks, and they come back. And if they say, oh, much better, then we might want to think about going on to the ablation.

These typically are people who don't have a lot of structural heart disease, although we're beginning to move into more and more [INAUDIBLE] patients, which we weren't doing a few years ago. They have to, for most insurances, have failed at least one anti-arrhythmic drug. And we want to catch them before the disease is so advanced that we just can't succeed. So by the time they're chronic, we really don't have anything to offer them. So if you're even thinking about it with a patient, try and catch it when it paroxysmal or early persistent. But once they are in AFib continuously for more than a year, their success rate really drops. And I'll screen one.

This was the paper-- incredibly, now 25 years ago-- Michel Haissaguerre in Bordeaux, France. They're the ones that figured out that the pulmonary veins seem to be the source of triggering cells that initiate AFib. So prior to that, we never had any particular concept how we could avoid A. fib. But what they discovered is if you go into the mouth of the pulmonary veins, right where they bug into the left atrium, there's a ring of cells right in that intersection zone that are very irritable, and when they're exposed to stretch, and as they are when there's hypertension, that stretches the left atrium. And When they get pulled on, they begin to throw out PACs. So storms of PACs will typically initiate the AFib.

So this is very overly detailed CT scan. Forget the pulmonary arteries above. This is the back of the left atrium. Right superior, right inferior, left inferior, left superior vein. So that's where we really focus our energy in an ablation, and we're looking at this zone right where the vein is connecting into the left atrium.

This is a sly comeback, kind of pivotal study. This is a catheter sitting in the right inferior pulmonary vein. There's a sinus beat on top, and then you see one premature beat, and it initiates the AFib. In the left superior vein, we get a premature firing. From the pulmonary vein, there's no p-wave. The pulmonary vein fires, but it didn't start beating. And now on the next slide, we get a sinus beat pulmonary vein fire, and it starts the AFib. So it's all about middling the couple interval, or the [INAUDIBLE] state of the patient at any one given time.

So this idea that AFib initiates in the posterior wall of the left atrium has sort of dominated ablation for the last 10 years, and so we really focused on the pulmonary veins. Here's a nice section of those pulmonary veins. If you look at his superior vein, you can see that there is a ring of muscle. And then going around here, whereas this interior vein doesn't have a lot of muscle. There has to be muscle to fire these premature beats.

So our concept with this cartoon is to go make a ring around the base of the vein, right where it's joined. And we used to do that with a hot-tipped ablation catheter--the regular frequency catheter. The problem with that is it's kind of a dot, dot, dot, approach. You're walking around and making dots in order to make a circle. And you could easily imagine that there could be little gaps between your dots that maybe don't appear acutely, but show up when the swelling goes down.

So the problem with this technique was for currents. So we could fix it all that day, and they got home, and they have AFib again. So something like 30% or 40% of people had to come back for a touch-up procedure. This is the technique that we would use. We'd go all-venous, so we come from the groin, lead our catheters up into the right atrium. We have to cross the interatrial septum. Kind of looks like an eardrum that was actually open when we were all in uterus. So there is some kinds of piggy [INAUDIBLE], but usually not, so we make a little puncture in that [INAUDIBLE], and then feed our catheters into the left atrium.

We typically would use two catheters-- one that was circular that had all these recording sites on it, and then the hot-tipped ablation catheter. The circular catheter would guide us so we could see all cells that were firing, and then we would burn while working our way around the pockets. And that's what it looks like on X-ray. You can see the number of electrodes, and the ablation catheter right underneath.

One of the key things that we do is we do get a CT before we do an ablation because the anatomical variation is mind-blowing. So there's supposed to be just four veins, and sometimes they're just wacky. So this guy kind of has a common right and a common left. This one has this big bulging, antrum to both veins on the left. This person has a fifth vein. This person's anatomy is relatively normal. So I get the CT, I get that nice 3D reconstruction, burn it on a CD, and we load it into our computer while we're doing the ablation.

So here's that CT image, and then we've overlaid it with our ablation marks. This is when we are doing radiofrequency. So they were walking around this vein, isolating it. There's the circular catheter sitting in the left inferior vein, and the little green-tipped ablation catheter. And you can see the circular catheter, which is inside the vein, isn't recording any electrical activity. So that thing is isolated. And then you go on to the next.

One of the cool things is that we can turn that in which any way we want in the room, and that red, vertical catheter is an electrode sitting in the esophagus. And we do that because the esophagus is really close to the left atrium, and if we don't know where it is, and we heat on the back wall of the atrium, we could be heating the esophagus. And one of the most dreaded complications-- thank god we've never seen that at John Muir-- is you can actually make a fistula. It's usually fatal. It's an error, and [INAUDIBLE] heart blood into the esophagus. So we always know where the esophagus is. We monitor its temperature, and we never let it get hot or cold, depending on what technology we're using.

So here's a cool example of somebody in sinus rhythm on top, but the catheter that's inside the vein is showing me AFib. It just can't get out anymore because you've isolated the vein. So that's how we used to do it. Now we use this balloon. We place it in the mouth of the vein and fill it with nitrogen. Goes down to about minus 60. And it kind of stamps the whole vein all at once. So it makes a beautiful circle instead of dot, dot, dot, dot, dot. Hugely reduced our current [INAUDIBLE]. Huge lift.

That's what it looks like. It sits in the mouth of the vein. It has a little recording catheter on the inside. We can inject a little guide, make sure we really occluded the vein, and then we freeze it. And we can follow that on ultrasound-- is the balloon sitting there? Is the balloon sitting there? This is what it looks like on X-ray. You can see balloon here. We'd inject a dye beyond that, so you can see that the vein's lit up with contrast and material, and that's we know we've occluded.

The newest thing that we're doing-- and this will be my last couple of sides-- is the vein isolation thing is great for people with early fib, but it doesn't do a lot if you have bad fibrillation, like persistent, or a really big left atrium. And we used to just do bad fib. Some patients do it-- isolate the vein, and the vast success rate we've had would be about 60%.

So we used to look for these rapid signals. We would ablate those. Didn't matter what we did. We never got much better than about 60%, 65% success. And these are results from different studies showing the kind of crappy results single procedure if you had persistent AFib. Same here.

So now we're using this new technique. We are one of only 20 medical centers in the whole country to have this equipment. The company's called Topera. It's a basket catheter that we place in the right atrium, and then in the left atrium. It has 64 recording sites on it-- all down these wires-- and you can see it sitting here on X-ray. It's horizontally oriented. You can see the little strands. You can see it sitting, by ultrasound, in the left atrium. And basically, it records during AFib, and it looks for what are called rotors, which are areas of circular spinning activity.

I explain it to patients by saying it kind of looks like a hurricane. So it's electricity that's spinning in a circle. That appears to us as a color-coded map. Where we find these circular activity areas, we ablate right in the eye of the hurricane, and that whole storm goes away. On average, people have two or three. Some might be in the right atrium, not the left atrium. And that's interesting-- it seems like a lot of sleep apnea people have it in the right atrium.

So this is brand-new. We just got it in May. Here, in this particular example, you can see two areas of spinning that were identified. You heat in the center, and boom, the AFib terminates.

The success rate with this technique is much higher. This is the study data with implanted monitors, conventional ablation-- we call this technique FIRM-guided. 84% success. And since May, every patient I've done with this technique is still on sinus rhythm, and that would never have occurred before. Never.

So success really depends on how bad your fib is, but with the rotor mapping, we're looking at 85%. The chronic patients still are not that great. The redo rate-- it really depends on the underlying structural heart disease, but it seems to be much lower. Maybe I should leave it there so we have a minute or two for questions. I really don't have too much else anyway. Yeah?

AUDIENCE: Can you speak to the row of the violation or [INAUDIBLE]

SUSAN EISENBERG: PVCs-- we used to think of it as a very benign problem and we didn't recommend doing much of anything for it. It turns out it's not quite so benign. There are people, if they have a very high density of PVCs that can help a cardiomyopathy. So when I'm seeing a patient with frequent PVCs, I always get an echo, so I know if they already have affected their LB function, and a monitor that would show me the frequency of PVCs.

You need a lot of PVCs to cause that. So you're kind of looking for 20% of all your beats being PVCs. Below that, it probably won't cause a cardiomyopathy, and getting we can occasionally go in and try to break them, but if they don't have enough, we can't really map them, because you're sitting there with your catheter, waiting for a PVC, and-- there's one. Then you try and place it, and then you wait for another 20 minutes. So they have to have a high density. But if they do, we should treat it, because there are people who have developed cardiomyopathy as a result. Yes?

AUDIENCE: Reaching anticoagulation with novel agents-- you don't need [INAUDIBLE]

SUSAN EISENBERG: I don't.

AUDIENCE: But then with more prep, you [INAUDIBLE] other people. These would not bother with ligatures and bridge everybody during the procedure.

SUSAN EISENBERG: I bridge based on-- well, if it's an ablation procedure, I don't interrupt any [INAUDIBLE] at all. So you're going go speak again, adapt to a procedure. But for other things-- the gallbladder, or whatever-- they have to be bridged if their CHADS score is high, but if their CHADS score is low, you can probably just have a window of time without the anticoagulation. So I would say, CHADS score of 2 and higher, you're going to have to use a bridge. And we typically use Lovenox, but frankly, there's not a whole lot of difference between using Lovenox and using Xarelto, so I prefer to use Xarelto or Eliquis isn't good. Then you don't have [INAUDIBLE] the cells.

SPEAKER 1: Thank you very much.

SUSAN EISENBERG: My pleasure.

[APPLAUSE]

[SIDE CONVERSATIONS]