Welcome to this OR Live webcast presentation, live Montefiore Einstein Heart Center in New York. During the program it’s easy for you to make referrals, make appointments or request more information. Just click on the buttons on your webcast screen and open the door to informed medical care. OR Live, the vision of improving health.

Good evening. Tonight we are broadcasting live from the Montefiore Einstein Heart Center. Today I am privileged to sit on a panel with my two esteemed colleagues, Dr. John Fisher and Dr. Eugen Palma.

The title of our program tonight is “Left atrial catheter ablation for persistent atrial fibrillation.” The goal of our program is to provide the viewer with an in-depth understanding of cutting-edge treatment of atrial fibrillation and catheter ablation to cure atrial fibrillation.

Due to time constraints, we were able to prerecord our procedure at an earlier date, approximately two week ago. The procedure normally takes four to eight hours, depending on the type of atrial fibrillation was encountered, and based on the fact that we only had one hour to broadcast, we had to do some editing. So without further adieu, I’m going to present some introductory slides just to give some background information.

One must understand that there are different types of atrial fibrillation. Patients that have paroxysmal atrial fibrillation have palpitations and irregular heart rhythms that come and go. They resolve on their own. They can last for hours or for days, but when they resolve, they resolve without medical treatment. Patients with persistent atrial fibrillation have atrial fibrillation and palpitations, which is usually sustained beyond a seven-day period and usually necessitates some sort of treatment from a physician, either medicines or electrocardioversion. Yet a third type of atrial fibrillation is called “longstanding persistent atrial fibrillation,” which is defined as continuous AF, or atrial fibrillation of greater than one-year’s duration.

When we speak about chronic atrial fibrillation, which is mentioned in the title of this presentation, it combines patients with longstanding persistent atrial fibrillation and patients with what’s called “permanent atrial fibrillation.” It essentially means the atrial fibrillation is difficult to conquer and to restore sinus rhythm. As these definitions go, patients with paroxysmal atrial fibrillation tend to do the best when it comes to catheter ablation treatment. Those with persistent atrial fibrillation do quite well, and when you have longstanding persistent atrial fibrillation, like the patient we’ll present tonight, the success rates are still quite good but less successful than those that have paroxysmal AF.

In the next slide we’ll talk a little bit about who is affected by atrial fibrillation. It is the most common arrhythmia in clinical practice, and certainly between the U.S. and Europe there’s almost six million people that have this arrhythmia. We know that most patients that have AF are elderly and that AF is associated with an increased risk of stroke, heart failure, and all cause mortality. In fact, in the next decade, we expect AF to be the next great epidemic, and an estimated 16-million people will be diagnosed with atrial fibrillation.
This slide simply depicts the mortality associated with atrial fibrillation. This is data taken from the Framingham Cohort 5,000 patients. Those patients with atrial fibrillation have a greater mortality over a ten-year period as compared to patients without AF.

There are many different ways to treat atrial fibrillation, and when speaking about rhythm control therapy, this algorithm for treating atrial fibrillation gives various different options. You can see that depending on the comorbidities or the diseases that you have alongside of atrial fibrillation, one is eligible to receive various different medications, and in the end, everything comes down to if you fail medical therapy to catheter ablation, no matter what type of atrial fibrillation you have.

Of course catheter ablation is not without complications. Complication rates range from two to five percent, with patients who need permanent pacemakers being a complication more of Maze procedure and less of catheter ablation.

Moving onto our case history, talking about our patient that you’ll see tonight, is a 66-year-old gentleman who had a history of a nonischemic cardiomyopathy, a weak heart muscle, high blood pressure, and an injection fraction or left ventricular function that suggests how much blood is being ejected from the heart of 35 percent. The patients had previously had a defibrillator implanted to prevent sudden cardiac death.

Approximately one year after defibrillator implantation, the patient presented with multiple shocks. It was determined that the shocks were due to atrial fibrillation causing his heart rate to race, which was detected by the defibrillator and resulted in inappropriate shocks. He was treated with AV nodal blocking agents and antiarrhythmic agent called “Dofetilide” and returned several months later with even more shocks. After failing antiarrhythmic therapy as well as rate control therapy, he was referred to our center for a left atrial catheter ablation.

Drew, Aside from the weak injection traction and the recurrent shock, was this patient feeling anything else?

Well the patient was suffering from dyspnoea exerion. He had shortness of breath when he was exercising. He was also complaining of fatigue quite often, and when we had seen him, he had been in persistent atrial fibrillation for a number of months.

I think that’s an important symptom that you brought up that I think many patients don’t recognize or even many physicians actually don’t recognize that fatigue is a very common symptom of atrial fibrillation. It’s not just palpitations and shortness of breath, but sometimes fatigue is an important symptom of atrial fibrillation.

That’s an excellent point. The crux of helping somebody with atrial fibrillation is to make sure that they actually have symptoms. There are some people who don’t have symptoms with their atrial fibrillation. But if you dig deep enough and you speak to your patients you find that many of them are quite symptomatic and have sort of changed their lifestyle to accommodate to their atrial fibrillation. They’re less active. They are more prone to cancel long trips. And those are the patients who we really can stand to help by curing their atrial fibrillation.

Again, to flip back to our slides, I just want to show this slide. Once we get started with our presentation it’s going to be a little difficult to orient people who are watching this, the viewers, to exactly what we’re looking at.

Just so you have an idea, and we’ll go through this on a heart model shortly, on the left panel of your screen is an intracardiac ultrasound image depicting the left atrium with a catheter at the top part of the screen passing through the intra-atrial septum, and then the two pulmonary veins sort of juts out at the bottom of your screen at about 5:00 and 6:00 o’clock, those are the left pulmonary veins.
On the right panel, you can see a three-dimensional map of the left atrium and the ultrasound view sort of slicing right through. We’re going to use or intracardiac ultrasound catheter to make a map of the left atrium and to help us pinpoint the critical areas for delivering ablation therapy to the left atrium and curing the patient of their atrial fibrillation.

And I think our viewers actually will realize or be able to have a better understanding of what those images that they were seeing on the screen when you go through the procedure.

Absolutely. One more thing that I’d like to do is I’d like to show on this heart model exactly what we’re going to do tonight. This is the human heart. These chambers down here are the ventricles. We can open them up, and you can see the heart’s got four chambers; two ventricles, which are the main pumps, and two atrium, which are at the top part of the heart.

There’s a right and left side of the heart. We’re going to be focusing on the rear portion of the heart and the left atrium, which is this structure, this yellow structure here. You can see that the aorta is just behind it. The esophagus is just behind it. And what we’re going to be doing tonight is we’re going to be drawing a line of electrical block, surrounding the pulmonary veins, those are these tubes that are entering the left atrium. We’re going to draw a line across the roof of the left atrium, connecting the two and circling the lesions around each set of pulmonary veins. That line is going to go from here to here. And then down at the area of the mitral valve, which is the area that connects the left atrium to the left ventricle, we’re going to draw a line from the left inferior pulmonary vein down to the ventricle. That’s called the “mitral isthmus line.”

Why would you draw a line around these veins?

So that’s an excellent question, Dr. Fisher. The terminology “draw” is what it looks like on our computer screen. What we’re actually doing is we’re creating a line of electrical block. We’re creating a small minute scar that is a linear scar that prevents electrical circuits from perpetuating and causing the patient to have palpitations.

I like to think of it as you’re creating barricades or barriers to the electrical signals that are coming from the pulmonary veins so that they don’t get out to the rest of the heart and start atrial fibrillation that way.

I think without any further adieu, we’re going to jump to the OR here and go to the Montefiore Heart Center electrophysiology laboratory. Our patient is brought into a specially-equipped room designed for complex ablation. We use biplane fluoroscopy, intracardiac echocardiography, and three-dimensional anatomic mapping to assist us during the procedure. The patient is draped and prepped in a sterile fashion, and we place introducer sheets into each femoral vein. We do this via a standard Seldinger technique that you’ll see depicted shortly.

That was just local anesthesia that a dentist might use that they were giving?

Correct. Prior to doing this, the patient is sedated. We sometimes use general anesthesia, and oftentimes we use conscious sedation. Either way, patients feel virtually nothing. In this particular case, we used conscious sedation, and the patient has no recollection of any pain or any suffering during this procedure, which did take several hours.

Here you can see, we’re inserting a guide wire through a hollow board needle into the vein, and over that guide wire, we’ll introducing an introducer sheath that has a one-way valve and will allow us to exchange catheters throughout the procedure.

Right now you can see one of our technicians who is feverishly working to create a shell of the left atrium, which is from a previously-obtained CAT scan of the heart. That’s going to serve as a road map. It’s going to show us where the pulmonary veins are, and it’s going to show us what
the left atrial anatomy is like that we can more accurately and carefully place our lesions and avoid critical structures like the internal portions of the pulmonary veins and even the esophagus.

As you can see here, we’re just inserting one of the introducer catheters. Once the introducers have all been inserted we place an intracardiac ultrasound catheter into the heart. And we do a survey of the left and right atrium looking for blood clots and defining our anatomy. We define all four pulmonary veins and the esophagus.

Here is an example of the intracardiac ultrasound catheter screen. You can see the right atrium, right ventricle, and the aorta coming off of the right. This is what our CAT scan looks like. You see a three-dimensional view with the rear end of the left atrium facing us and the four pulmonary veins jutting out to each corner of the screen.

Back to our ultrasound screen you can see the left pulmonary veins. At about 5:00 o’clock is the left superior vein, and at about 6:00 o’clock is the left inferior pulmonary vein. That large stripe behind the left atrium is the aorta.

So we’re seeing blood actually flowing from the pulmonary veins into the left atrium; is that correct?

Correct. We actually use Doppler measurements and get baseline pulmonary venous flows prior to beginning our procedure. As you can see by the tracings here, the patient is in atrial fibrillation as we begin. This is a fluoroscopic image, an x-ray image that we use. As you can see here, it is an RAO image. There are a number of catheters in the heart. To the left of your screen you can see an intracardiac ultrasound catheter placed in the right atrium. There’s a coronary sinus catheter passing diagonally across the screen, and you could see in that last view the patient’s indwelling defibrillator wire with its tip in the right ventricular apex.

Once we have set ourselves up and done our survey of the right and left atrium, we begin to create our map with a system called “CartoSound.” This software allows us to make a shell -- a real-time shell of the left atrium and the pulmonary veins using the ultrasound images, and it takes us about 20 to 25 slices and 20 to 25 ultrasound pictures to create a shell.

What you see now is our technician tracing the inner border of the atrium and adding it to the shell. It’s extremely important during this process that we define accurately exactly where the pulmonary veins begin and where the atriums begin.

So the advantage of this, Drew, is basically you’re converting an image which was two-dimensional, like fluoroscopy or even ultrasound, and converting it into a three-dimensional image that helps you visualize the anatomy; is that correct?

Exactly correct. We oftentimes will combine the three-dimensional image created with the ultrasound and CartoSound with the CT image.

This is a particularly important ultrasound picture. You can see there’s a stripe just behind that black circle, which represents the left atrium. That stripe is the esophagus. It’s very important that we view the esophagus at all points in time throughout the procedure because we need to avoid the application of radiofrequency energy. We need to avoid burning in areas that the esophagus is nearby, because one of the complications that is extremely rare to this procedure involves damage to the esophagus. This system helps us really maintain the esophageal location throughout the procedure, and it is well known that the esophagus can shift over a multi-hour procedure. It looks like we’re almost at the point here where we’ve got a three-dimensional map of the left atrium.

How long does it usually take to do the reconstruction itself, Drew?
To do the construction at the beginning of the procedure, it’s about 30 minutes taking images. We really check and double check the pulmonary vein locations. Oftentimes you spend a little time registering your CAT scan images so that you can really have a nice accurate three-dimensional view.

You can see here this gray structure is actually our three-dimensional reconstruction of the left atrium. We’re going to add in the mitral valve, which is the valve that connects the left atrium and left ventricle in just a second here. And once that’s done we’re going to proceed with getting access into the left atrium and actually putting a catheter in the left atrium.

So the structure that you’re seeing now, Drew, it’s a virtual reconstruction based on your ultrasound images?

Correct. We have defined the esophagus and the pulmonary veins and the mitral valve and the various walls, the anterior wall, the posterior wall, the inferior wall, and the lateral walls. The nice thing about this system is we are not in the left atrium. No catheters have been placed into the left atrium just yet, and we already have a three-dimensional view. You can see now our transseptal puncture needle being depicted on your screen with a guide wire being passed in and out.

We pass along sheathe through the vein up into the heart, and we’re getting ready to enter the left atrium. In order to do that, we actually have to poke a small hole through the intra-atrial septum and get right into the left atrium.

So it’s important because normally catheters or the wires that we use only stay in the right atrium and they can’t go into the left.

Correct.

Unless you do this maneuver.

Correct. So you’re going to see that we’re going to move our long sheathe downward or quarterly, and it’s going to fall onto the intra-atrial septum right about here. You should see it move to the center of your screen right there. We’ll flip to an aerial view of the fluoro, which will show that our catheter is in a posterior position, and then we’ll look at the ultrasound screen and see that there’s some tenting or an indentation of that intra-atrial septum being caused by our catheter.

And I think that’s how intracardiac echo really helps, because you can actually see where your catheter is going so that you don’t unnecessarily cause damage to other structures.

Correct. While you were speaking, Dr. Palma, we just saw that we crossed over into the left atrium. We have extended a guide wire out into the left superior pulmonary vein, and now we’re passing our long guide sheathe up into the left atrium. Through this guide sheathe, which a steerable sheathe, which will help us maneuver nicely throughout the body of the left atrium, we put an ablation catheter, which is an irrigated Catheter.

And that's what we're seeing now.

That's what we're seeing now, the jet of saline that sprays while we're applying radiofrequency energy.

The purpose of the saline or salt water helps to keep the tissue from being too hot?
Correct. Having the saline spraying at the interface where the catheter is prevents charring and sometimes blood-clot formation and improves safety while it minimizes lesion time and gives us a nice deep lesion.

What you’re seeing now is a second catheter that was placed into the left atrium. That’s our lasso catheter. The lasso catheter has electrodes around its perimeter and allows us to check our work and make sure that we’ve truly electrically isolated or disconnected the pulmonary veins. Here on the ultrasound screen you’ll see the lasso catheter being positioned into the left pulmonary veins.

That white stripe that we see is the lasso catheter.

Correct. Now this is our recording system. This is perhaps the most important but most confusing system that we have. There’s probably 35 different leads running through here, but I’ll try to explain it rapidly. The first four leads are surface ECG leads. The next two leads, which are in yellow, are recordings that we get from our ablation catheter. The green leads are the leads that are being recorded from the perimeter of the lasso catheter, and the white leads that are down at the bottom of your screen are recording atrial electrograms from the coronary sinus, the multicolored coronary sinus catheter, which is being pushed up towards the right side -- right-hand portion of your screen right this moment. So we’re going to seat our catheter just at the os of the left superior pulmonary vein. That’s one of the left veins.

You say “os.” You mean where the vein enters into the atrium?

Correct. That is right at the entrance. And we’re going to take a look on our recording system at the green recordings and see exactly what sort of electrical activity we have. And as you can see, the patient is in atrial fibrillation. There is a lot of rapid irregular activity in those green recordings, and that represents a lot of electrical activity at the interface between the vein and the left atrium.

Our goal here is to eliminate those electrical signals. And what we’re going to do on our map, which we have added some colorful tubes to represent the pulmonary veins entering the left atrium, we’re going to create a wide encircling lesion, basically a big circle of scar around each set of pulmonary veins. The gray structures that you see here are the pulmonary veins as depicted on ultrasound, and the tubular structures, the colorful tubes that you see are the pulmonary veins that we have been able to place on the map by placing our catheter into the vein and dragging it backwards. The computer registers this on our three-dimensional map.

Once we get our catheter in the correct position, we will begin to ablate and create our lesion set. During ablation we try to keep certain temperature and wattage settings or power settings to prevent damage or perforation to the atrial wall. When we’re on the posterior wall, we use 25 watts, and when we’re on the anterior side we usually use 35 watts. The posterior wall is a very thin structure and adjacent to structures like the esophagus, and we want to be extremely careful when we’re ablating along the posterior wall.

So, John, you’ve have had tremendous amount of experience with RF ablation. In fact, pioneered the ablation here at Montefiore. Do these scars cause permanent or damage in any way?

Well you actually want to cause permanent damage because you must permanently isolate the pulmonary veins. It’s a little bit like touching a hot stove. You don’t a blister that heals without a scar. You want a permanent block or blockade or corralling or whatever you want to call it. But it is, as Drew was saying earlier, it’s a very thin area of scar, and it does not really affect heart function or the function of other nearby organs.
What you’re seeing now on our screens are the recordings coming from the left superior pulmonary vein. We’re continuing to make our lesion set, and we’ve made almost a complete encircling lesion around the left pulmonary veins here. The only portion that we have left out is the area what we call the “famous ridge area,” which is a ridge between the left superior pulmonary vein and the left atrial appendage.

Here you see an ultrasound shot of our ablation catheter and our lasso catheter, and if you pay attention to the green recordings on your screen, you’ll start to see that low and behold we’re beginning to isolate the left superior pulmonary vein. You will start to see that the little hash marks on the green leads start to slow done and eventually disappear.

And I think that’s a very nice position for your lasso because it’s right where the border of the left atrium meets the pulmonary vein itself because you don’t want to be too far in and at the same time, you don’t want to be -- you want to be able to record pulmonary vein potentials as well.

So you’ll see we slowed down significantly. We have what appears to be entrance block into the left superior pulmonary vein. And in a moment when we flash back to our recording system, once we put in these last few lesions, we’re right at the carina, the area between the left superior and left inferior pulmonary vein. You can see that we have almost completely disassociated this vein.

So there are lots of squiggles on the white lead, but not so many more on the greens.

Correct. The white leads represent recordings from the coronary sinus, and the left atrium is still very much fibrillating. But we have isolated this little area around the left superior pulmonary vein, and we have then positioned our lasso into the left pulmonary vein just at the os. And you can see it there on the ultrasound quite nicely, and we’ll take a look at our recording system and see if there is any electrical activity there. Since we have encircled both veins, we would hope for no significant electrical activity, and that’s what we have here. So we’ve isolated both veins.

That’s what you mean by “entrance block,” because entry of electrical signals is blocked from going into the pulmonary vein.

Exactly. We’re still fibrillating in the atrium at large, but that rapid electrical activity cannot get into the veins. We’re assuming that by having entrance block we also have exit block, since we know that atrial fibrillation is usually initiated by rapidly firing foci coming from the pulmonary veins, and we’ll be able to check for exit block later in the procedure. It’s essential that you do both. You can’t just have entrance block and not look for exit block.

So keeping the lasso catheter at the os site -- or at the border of where the left atrium or the top left part of the heart meets the pulmonary veins, I think is critical. And I think it’s one of the advantages of using intracardiac echo, because you can actually see its position quite clearly.

Absolutely. We’re going to take a break now from the OR and try and answer some questions that have come through. With regard to Eugen Palma’s last point, it’s very important to have the ultrasound working throughout the procedure. It gives you real-time information and shows you exactly where your catheter is in relation to your vein, in relation to your esophagus, and it allows you to safely navigate throughout the left atrium.

Just briefly, Drew, why would somebody undergo a procedure four to eight hours instead of just taking a pill? What are the advantages?

It’s funny you should ask that, because that’s one of our questions from the Internet here that I was just handed. The question is, “What is the advantage? Why bother with a long complex procedure when someone can just take a pill.” And I think the answer to that question is really dependent upon the patient at large.
Some patients don’t have any problems taking medicines. The problem with taking medicines is that antiarrhythmic medicines are oftentimes only 40 to 60 percent effective. By doing a catheter ablation procedure we have the opportunity to prevent a cure, to prevent palpitations, to get patients off of medications, and to improve their quality of life in a significant way. What do you think, Eugen?

I just have, I think one -- I’m almost hesitant to use the word “cure” always because we don’t know what will happen 25 years from now, because this procedure, you know, only started ten years ago. In fact, it’s just the tenth anniversary this year. So I hesitate to use that word. But at the same time, I think the advantage of this procedure is that some trials now, and very scientifically conducted trials, have shown that this procedure works better than just taking pills alone. And when we say “better,” we mean that patients feel better, which is actually the most important part, and that they are able to not take pills, and that their, what we call “quality of life” actually improves.

Patients with atrial fibrillation can be very, very symptomatic, and some studies have shown that they’re almost as sick as patients who have just had a heart attack, for example. So I think this procedure allows patients who feel their atrial fibrillation and makes their life quite miserable, and we can make a dramatic difference in those types of patients.

I think that’s an excellent point. I think the focus of our program here at the Montefiore Einstein Heart Center is to provide the patient with quality of life, and that a cure, in my mind, is not necessarily complete eradication of atrial fibrillation but rather a tremendous change in the quality of life for the patient, significant reductions in palpitations, significant reductions in the number of medications that they have to take, and significant reduction in hospitalization and recurrent trips to the emergency room. I think that makes all the difference, and it’s an important point.

And I think just one of the important points also about this particular patient that I’d like to bring up is that this patient had a very weak pump or a very weak ejection faction. It’s almost half of what normal is. And nothing predicts how long a person will live as much as the strength of the left ventricle or the pump of the heart. And I think if by doing this procedure we find out three months down the road that his pumping function is actually improved, then I think, as it has in some of the patients that I’ve actually done and what’s been reported in the literature, then I think that’s another advantage to doing this procedure that is being under recognized.

One more question that was just handed to me. “What is the harm in leaving a patient in atrial fibrillation without restoring sinus rhythm?”

I guess there are lots of things that can go wrong. There is an increase in hospitalization. There’s an increase overall in mortality. There is a decrease in quality of life, increase in risk of stroke, a reduction in the patient’s ability to do things. They tend to get short of breath more if they are in atrial fibrillation. As you were saying, they gradually may contract their activities, reduce their activities. So there are a lot of disadvantages, some of them subtle and some of them not so subtle, to being in atrial fibrillation.

What would you do, Eugen, in a patient that comes to you, referred to you that is completely asymptomatic, on minimal medications, 65 years old, and they have adequate rate control. They don’t seem to have much fatigue. What would you say to that patient?

Yeah, I think that’s a very good question, and I think that’s difficult to answer. One of the things that I think we need to do right at the beginning is make sure that this patient is really asymptomatic. I have oftentimes performed a cardioversion in a patient who claims, “I feel great” and found out how much better they felt when they were in a normal rhythm than when they were in atrial fibrillation. So I think that’s the first thing.
And whether it’s by trial of frustration of normal rhythm through a cardioversion or by just having them exercise or walk up and down and time how much they measure exactly the amount of exercise they do. I think it’s important in finding out how truly asymptomatic they are. I think the second part of the question is, are there any other factors that we need to consider, such as other things which prevent you from doing atrial fibrillation in this patient.

So if they have serious heart conditions other than atrial fibrillation or heart failure, if they have other lung diseases or kidney diseases, then I think we should, you know, think long and hard before recommending a procedure that does take a long time to do, and is not without complications.

And then I think the lastly, I think it’s a dialogue with the patient on what their expectations are. Does he expect to come off blood thinners. If that’s the reason that he’s doing this, then, you know, I tend to discourage those patients, because we just don’t know at this point in time whether or not blood thinners can be stopped completely in patients who have very many risk factors.

In patients who only have high blood pressure or just one other risk factor, maybe there’s enough evidence now to say that, yes, we can just put these patients on aspirin alone. But otherwise, I think there’s just not enough data to have that as the only reason to do an atrial fibrillation ablation.

I completely agree. I’m getting a signal that we should cut back to the OR here. You’re going to see very much the same procedure that we did in the first part. We’re going to be making an encircling lesion around the right pulmonary veins and looking for isolation in those veins. And because we’re speaking about such an interesting topic when it comes to atrial fibrillation, everybody wants to know about anticoagulation or thinning the blood.

So for the viewers who don’t know this, when you have atrial fibrillation and you do have risk factors for stroke; that is, constructive heart disease, heart failure, hypertension, previous history of stroke, or diabetes, you must be maintained on anticoagulation therapy, either Coumadin or some sort of heparin, but usually Coumadin, this is really problematic, and it really affects patient’s quality of life because they real must get their blood checked on a regular basis, sometimes weekly or every two weeks to make sure that their blood is not too thin and that their blood is thin enough to prevent stroke. Because Coumadin prevents stroke in patients that have atrial fibrillation with risk factors.

So here’s, I think, an important question from the audience, Drew. “Do you know the percent of patients who undergo catheter ablation and completely get rid of their atrial fibrillation?”

That’s an excellent question. I can’t say that I have an answer to that since there are so many centers. In general, and the question is --

What about in your experience?

What does completely mean? You know, in general and in our experience, patients that come for catheter ablation have an 85-percent success rate of restoration of sinus rhythm. Now some of those patients have to do this procedure two times, about a quarter of them, and some of those patients do require long-term antiarrhythmic medications and even Coumadin. But a large majority of those patients are able to be taken off of their antiarrhythmic medications, and some of them can be taken off of Coumadin depending on their risk factors.

Does it make a difference whether they have the paroxysmal type that comes and goes and an otherwise normal heart versus the patient like this one who had a poor heart?
Correct. It absolutely does. As we alluded to earlier, patients with paroxysmal atrial fibrillation, the on-again off-again all on its own tend to have the best outcomes and really have tremendous response to this procedure. We have seen some dramatic cures in many patients.

Patients that have chronic or longstanding, what we call “longstanding persistent atrial fibrillation,” are patients that tend to have less robust outcomes. And we have seen in this population about a 50-percent redo rate and maybe a 60-percent maintenance of sinus rhythm. But most of those patients require some sort of antiarrhythmic medication. I would say the majority, and usually because they all have structural heart disease, I’m very slow to take any of those patients off of anticoagulation.

You know, in this particular patient, he had several hospital admissions because his defibrillator was giving many multiple shocks, and the point of this procedure is to prevent that from happening in the future and to prevent hospital admissions.

So what’s happening here, Drew? What are you doing?

So, again, we are doing what we like to call the “reach around,” where we take our catheter and reach around the left atrium to get to the left inferior portion of the left atrial septum. We’ve drawn an encircling lesion around the superior portion of the right veins, and we’re trying to get to the bottom part of the right inferior vein.

Inferior meaning it’s not as good?

No. Inferior meaning it’s just as good as the superior, but meaning that it’s below the superior vein. Because our catheter comes from the septum, the veins that are sort of coming off the septum, the right veins, are a little bit harder to reach because we have to curl our catheter back around. And, in fact, the right inferior vein, which is just as good as the others, is just a little more difficult to get to. It’s infamous for being a little harder to isolate. And we’re struggling right now just getting our catheter into the right position to complete this lesion set.

What are those polka dots again?

So those red polka dots represent points where we have laid down lesions, places where we’ve cauterized the left atrium. Each place that we put a polka dot is a place where we have applied radiofrequency energy and noticed that the electrograms at that site have diminished significantly, and we believe that we will not have to go back to those areas to ablate. That being said, we frequently go back and forth over our old lines to look for gaps that we may have missed to ensure that we have a completion lesion. And you’ll notice, you can see here actually, we just flipped back to our monitor, we have isolation, what I believe electrical isolation of the right superior pulmonary vein, and we’re pulling our catheter back now, the lasso catheter back and looking at the right inferior vein.

So just a couple of quick questions, Drew, from the Internet.

Sure.

How long is it take to recover after you have had this procedure?

It usually taking a couple of days. Generally speaking, we can send the patient home the following day or two or three days later. The impediment to sending a patient home is generally we need to get their blood thinned again, and sometimes patients don’t like to inject themselves with Lovenox. And if that’s the case, we leave them on an IV heparin and keep them in the hospital for a day or two. I generally tell people to take three, four days off of work. If you do the procedure on a Wednesday, you can go back to work the next Monday. Your groins may be a little bit sore, but in general, the recovery is quite rapid.
But also I think it depends on the length of the procedure also, I think, Drew. If you have had an extensive procedure that lasts, you know, eight hours, then I think there is a bigger chance that patients will feel it for a longer period of time. And, you know, I have had some patients -- it’s three weeks and they still feel that they’re tired a little bit.

And I think it’s important to also -- one of the questions is a patient -- or I assume a patient had an ablation a year ago. I don’t think this is one of our patients, and she said that she still gets palpitations. Is it true that there’s a period of time wherein the palpitations don’t mean the procedure failed?

So palpitations are a very subjective symptom. And what we often do in patients that have recurrent palpitations is we send a home monitor, transtelephonic monitor to assess, when you have your palpitations what is your heart rhythm actually doing?

Some patients have palpitations but they’re not in atrial fibrillation. Some patients have palpitations when they are in atrial fibrillation and sometime when they’re not in atrial fibrillation, and you can get a mixture of results. But if you’ve been out a year and you’re still having palpitations after an ablation, and that’s documented to correlate with atrial fibrillation, well then there may be some touchup lesions that may need to be done. You may need to go for a repeat procedure.

We do know that 25 percent of patients require a second time around because of recovery of conduction. You can see that all of these lesions that we’re making, it’s not beyond the imagination to fathom that one of these lesions might develop a gap down the road and that you can have a little bit of leak of conduction. And any little leak will potentially perpetuate atrial fibrillation.

I’m just going to jump back to the video here, because we’re putting in a roof line connecting those two sets of encircling lesions. While we have been talking we’ve isolated the right veins and we have shown that there’s entrance block into each right vein. And now we’ve created the roof line and actually just flipped down to the mitral isthmus line where we’re connecting the mitral valve with the left inferior pulmonary vein lesion set.

So the reason you’re doing these lines is based on the type of atrial fibrillation in this patient.

Correct. When you have longstanding persistent atrial fibrillation, there’s been a lot of evidence to suggest that these linear lesions help to debulk the atrium, help to shorten wavelength and prevent multiple circuits from occurring. And they also help to prevent atrial flutters.

Earlier on you were saying that the atrial fibrillation came from the pulmonary veins. Does this mean that in the patients with actual heart disease or serious heart disease or long-lasting atrial fibrillation, that it comes from the atrium itself and not just from the pulmonary veins.

Certainly. There is an entity called “atrial electrical remodeling” where there are numerous factors that occur as the atrium dilates and it continues to fibrillate, which perpetuates atrial fibrillation, and, in fact, any of the thoracic veins can be triggers for atrial fibrillation in addition to atrial scarring, decreased expression of atrial gap junctions, and all of the things that go on with atrial electrical remodeling.

Right here -- I’m going to flip back to the screen -- you can see complex fractionated electrograms that we’re ablating. It was sort of fleeting there. But we are looking for actual atrial tissue that may be a driver of atrial fibrillation. And in order to find those areas that may be driving fibrillation, we search around the atrium looking for areas that have what’s called “complex fractionation” or rapidly firing areas. Typically, they are less than 120 milliseconds in cycle length
for those people that understand what that means. And they usually show continuous squiggles or continuous electrical activity.

You can see on the right-hand panel there are some pink dots around the anterior portion of the left atrium and at the base of the left atrial appendage. Those dots represent areas that we found fractionated electrograms, and we added some RF energy to those particular areas.

Going back to what Dr. Palma's question was, the question that he was involved with a few minutes ago about is there a timeframe in which you worry about things? Some people have suggested that the irritation from the ablation process itself, where you're making lesions on the heart muscle, could cause rhythms that are like atrial fibrillation, which may heal up then as the ablation lesions heal.

And I think that's important to remember, especially the referring or the physicians who are managing these patients or co-managing them with you is that I think the first three months after an ablation can be difficult sometimes. They have just undergone a long procedure. They do have some palpitations that may or may not be atrial fibrillation. And it's during these -- but it's gratifying to note that a large majority of these patients actually quiet down after about three months, and I think that's when we decide whether or not this patients needs a second procedure or not.

That's an important point. And we always talk about the blanking period, which is this period where one should not --

Oh, and we missed it, but I think very nice, Drew. I think you've just created normal rhythm here on this patient.

All right. So now we're in sinus rhythm. We need to check all of our work just like we showed that we has entrance block into the veins when we were fibrillating, in sinus rhythm, we can actually pace from within the veins.

In the sinus rhythm we just had an occasional hash mark, as you said, on both the yellow and the green and the white lines.

So the yellow represents our probe that's recording in the atrial depolarization or an atrial signal, and the white lines are in the coronary sinus. The green lines right now, you can see what's called the "pacing artifact." And we actually have our lasso catheter in the left superior pulmonary vein right at the os, and we're pacing circumferentially from around the lasso, seeing if we can capture where we can depolarize the left atrium. If we cannot, that would suggest that we have exit block.

Electricity is not able to come from the pulmonary vein to get to the left atrium.

Correct. Now we're positions our lasso at the left inferior vein, and we're going to do the same thing, making sure that the area around the antrum of the left inferior vein is not connected any longer to the left atrium. And you can see that nicely if you are looking at the recording system here. We'll do that with the rights and then we'll go to check some of our linear lesions.

We just placed our lasso catheter in the left appendage, so you can see some very large signals in green. And we're going to paste the left atrial appendage and use it, because it sits in a strategic location for testing some of the linear lesions or the long lesions we created. So the roof line and the mitral line, exactly.

What you'll see here is on the right-hand panel we're going to point out the left atrial appendage there and we're going to tilt the -- there's the left atrial appendage, and we're going to tilt down or image here. And we're going to paste from that left atrial appendage and watch the sequence of
electrical activation in the left atrium. What we hope to see as the electricity sort of bounces up against that roof line and doesn’t cross it, and we’re all to do that by putting a catheter just behind the roof line and making sure that there’s significant delay going from the left atrial appendage all the way back under our encircling lesions and back to a caudal/cranial direction up the posterior wall.

So why do you have to verify that your lines are complete?

So if you don’t verify that you lines are complete, you’re at risk of having a gap. And if you have a gap, it’s a perfect recipe for something called “atrial flutter” or “atrial tachycardia,” and that sort of goes back to what we spoke about just before. In the blanking period there are many patients that may develop a more regular rhythm called an “atrial flutter” or an “atrial tachycardia” that can sometimes even cause palpitations that are worse than the palpitations they felt when they were in atrial fibrillation because the heart may sometimes beat a little bit faster.

Oftentimes those patients do quiet down over the three-month period, and there are subset of patients that do require a repeat procedure. And when you get them back on the table and you look, you may find a gap in the line at the roof. You may find a gap around one of the encircling lesion sets that you put around the veins. And that’s why it’s very important to make sure while you there the first time that you have a complete lesion set.

We’re just spinning around our 3D-model, looking at our lesions, looking to see if there’s any gaps that we need to fill in. And we’re now going to focus on our mitral isthmus line and make sure that is intact.

You can see now, if you’re familiar with LAO fluoroscopic view, the X-ray view, that out ablation catheter is placed next to the multipolar coronary sinus catheter. By putting our ablation catheter in the coronary sinus, we actually have access to the epicardial or the outside portion of the heart. We made all of our lines and all of our lesions on the inner portion of the heart, but we’re now in a vein that courses around the outer portion of the heart.

There’s sometimes a sleeve of muscle that connects the left atrium to the mitral valve annulus, a sleeve of atrial tissue. And without ablating that particular area you oftentimes cannot get complete block at the mitral isthmus line, and so that’s what we’re trying to do here. We’re pacing from the left atrial appendage in green, and we’re looking at those white electrograms that are coming down at the bottom of your screen. And it looks like the very bottom of the screen comes early, and the very top of the white recordings come later. And basically we’re on one side of our mitral isthmus line trying to see if we have block going around the mitral isthmus.

Right now we do not have block and we’re going to apply some radiofrequency energy, and you will see the pattern in white will change when we get back to the recording system. So we’re in the coronary sinus giving short pulses of energy, radiofrequency energy, to create complete block across the mitral isthmus line.

When we come back to the recording system, you will see that our activation pattern is going to change, which will be consistent with block. And then before our eyes, it will actually recover and we won’t have block anymore until we touch up the lesions. So now there’s a change in activation pattern.

So these are the white squiggles.

The white squiggles are -- the highest white squiggle is early, and the lowest white squiggle is late. And you’ll see that that pattern is going to change very rapidly such that the sequence changes right there.

There it is.
Right there. So now we had some block, but it looks like we had recovery of conduction. So we’re going to just give a little more radiofrequency energy, and you’ll see the pattern will reverse, consistent with conduction block across the mitral isthmus. And we’re ablating now and back to complete block. And we’ll make sure that we have block 30 minutes after this is completed.

I think that’s to be congratulated, Drew, because I think that line, that mitral line is one of the most difficult lines to complete.

It can be quite challenging. It really can be. After this, we go back to the right atrium and we usually do a cavitricuspid isthmus line in patients with longstanding atrial fibrillation. I think that pretty much wrap up the intraoperative procedures and the computer screens from the AP laboratory.

One writer -- questioner writes in, “What’s the difference between this procedure and one to fix something called ‘SVT,’ because a relative had that procedure and it only took an hour or two?”

That’s a great question. SVT is a generic term. It’s an umbrella term, and it stands for “supraventricular tachycardia.” In other words, a rapid heart rhythm coming from anywhere above the ventricles. So you can group atrial fibrillation, atrial flutter, AVNRT, which is AV nodal reentrant tachycardia, and others under the umbrella of SVT. SVT is generally a single circuit that has a discrete point where you can ablate it and eliminate the SVT.

Atrial fibrillation involves multiple circuits, at least four veins, and depending on the type of atrial fibrillation you have, you can do this procedure in a matter of a couple of hours, versus many, many hours. If you’re the type of person that has paroxysmal atrial fibrillation with a clear trigger, it becomes a much shorter procedure than if you have chronic longstanding chronic persistent atrial fibrillation. And that is the difference between your relative and this particular procedure.

I think it’s also important for patients to realize that, yes, they may have had a relative who had it done in an hour or two, but going into the case, you never know exactly. And even an SVT could take a lot longer. And at least from the point of view of electrophysiologists, we really just work until the job is done. There’s no set number of hours in most cases.

I think also one of the points that I just wanted to make a comment on is that patients who paroxysmal atrial fibrillation can become persistent, and they go back and forth in the beginning of the disease process. But as time goes on, a fair number become what we call “longstanding persistent” or even “permanent.” And I think emerging data -- and it’s still controversial -- but I think there’s emerging data that suggests that maybe if we are able to eliminate the disease process early, we may prevent progression to longstanding persistence, and that may be one of the reasons to sort of intervene earlier rather than wait later. Because doing longstanding persistent atrial fibrillation is harder to do than paroxysmal.

That’s a question that we struggle with all the time. Patients come and ask us that very question. “If I do this now, is it better than if I wait until it gets worse?” And, again, we’re still waiting for some hard data. There are no clear answers. We just do know that it’s much easier to take care of paroxysmal atrial fibrillation than chronic persistent atrial fibrillation for various reasons, not the least of which you have atrial electrical remodeling and total changes in the structure of the atrial tissue.

Ashley writes in that you mentioned trials. “Are these results recorded somewhere or is there a procedure report, or what are these trials you talk about?”

Excellent question. One can look in “PubMed” or go to the Heart Rhythm Society site or the American Heart Association websites and you can find numerous trials. Any cardiology journal these days is chalked full of randomized control trials. Some not so randomized and not so
controlled, but many randomized controlled trials that have shown benefit for atrial fibrillation ablation. We’re expecting a large randomized control trial called the “Cabana Trial” to be releasing results in the near future. And we hope that we’ll see some promising results.

Now when we talk about trials, what are we talking about? Well we’re taking patients that have antiarrhythmic medications and are being treated for atrial fibrillation and comparing them with patients who go for catheter ablation and seeing which group of patients does better from a standpoint of quality of life, from a standpoint of long-term morbidity, mortality, and from a standpoint of complications.

And time and time again in small trials, we have seen that catheter ablation has tremendous promise. We still need to get some large clinical trials out there to prove this point. And still there are some remaining questions. How does catheter ablation affect morbidity -- mortality, excuse me? How does catheter ablation stack up when you look 20 years out? Do patients all redevelop their atrial fibrillation because of an ongoing process that created the atrial fibrillation in the first place? We don’t know the answers to those questions.

Do you see any promising new therapies that are ongoing that are being researched or --

So the field of atrial fibrillation ablation is constantly moving, so you have to stay on the cutting edge at all times. Yesterday’s procedure is no longer worth anything because there is now a new catheter or a new computer software system that’s better than what you were just using. Because we’re aware of this, we were actually part of the Stop AF trial, which was looking at balloon catheters and a cryoballoon catheter system that you can actually insert into the pulmonary vein and have it inflate and freeze the entire os in a circular fashion so as to prevent dragging your catheter around and making those polka dots that you saw on a three-dimensional map.

Just sort of make frostbite all around the --

Yeah, instead of a thermal burn, you get a frostbite burn, and by doing that you can actually isolate the veins in a more rapid, probably if not as safe, safer -- in a safer mode. We have seen some promising results, and we’re waiting for the official results to be released. Although we know that in Europe this has been approved and is in wide use. So we’re looking forward to balloon catheter technology, and we expect that this will really make procedure time shorter and procedures safer in general, and we’ll be able to offer this procedure to more and more patients.

Are there any other questions?

I think we can give some final thoughts. I think before we do wrap up, I think that what our audience should know at home is that when you do enter into an atrial fibrillation ablation is that there are many different parties that are involved. You have a radiologist who does your CAT scan. You have a team of nurses, nurse practitioners, and physician extenders that are involved on the day-to-day basis answering your phone calls, scheduling, dealing with palpitations before and after the procedure and really helping you along. And I don’t think that any of us could be up here without all of our physician extenders including Bridgette Murcaldi [PH], Debbie Johnston [PH] [PH], Gail Scaven [PH], Lisa Caroza [PH], Julie Thomas [PH], and Melissa Harding [PH]. Without our whole team here, we would really be up a tree. Any comments?

I think just also that I think we have a very experienced team of nurses and technicians and just that we have just recently started doing this at Montefiore’s other campus, the Einstein campus and the East campus. And I think there also, I would like to give a lot of kudos to the staff that is working there.

Overall it looks to me like atrial fibrillation has become a focus of attention in the world of heart rhythm disorders or arrhythmias. And in the past, other rhythms that have been targeted or
focused upon have gradually been conquered, and how do you see atrial fibrillation then in the future?

I’m sorry, somebody was whispering in my right ear during that. How do I see atrial fibrillation in the future?

Do you think that atrial fibrillation will eventually be a routinely accomplished kind of ablation?

I believe so. I believe with the advent with this balloon catheter technologies, I think it’s going to become as routine as let’s say the person who asked the question about SVT. As soon as we can do this in a rapid safe manner, patients will -- benefits and risk will change, and so you’ll have great benefit with very low risk, and there will be no reason not to do these procedures as opposed to thinking about if your patient is symptomatic enough to do the procedure. I think that this will allow patients to have a greater quality of life and avoid several hospitalizations before they actually commit to going for an ablation.

Okay. I think that concludes our program this evening. I want to thank my two panelists and thank everybody at home for watching.

Pleasure being here.

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