ELIJAH H. BEATY: Thank you very much to familiar faces. Thank you for the shouts like I was somebody famous to my lab staff. So we'll go ahead and get started. Today's talk is on inpatient and outpatient management of atrial fibrillation. So I have nothing to disclose, not famous enough yet.

So the objectives today are going to be address the three main components of management of atrial fibrillation, that being stroke prevention, rate control, as well as rhythm control, if somebody has symptoms. We'll also discuss the risk and benefits of anti-coagulation options, both medication-wise, as well as device-based therapy. We'll also talk about the rate control, whether that be medical or device-based therapy for rate control.

And also, we'll talk about the details of an AFib ablation. Sometimes, you may refer patients for AFib ablation, and it's just this nebulous thing that nobody knows about, it just gets done. So AFib is the most common arrhythmia that we have, affecting about six million people in the Us currently. But as the population ages, the likelihood for a patient of yours to have AFib will go up, and it's estimated that about 16 million patients will have AFib by 2050.

The thing about AFib based on that chart, it is linked with an increased risk for mortality. And the greatest component of that mortality risk is the stroke. So once we take care of stroke, then it's more of a quality of life issue. So as far as the pathophysiology of AFib, there are two types of components of AFib, both the trigger, as well as the substrate.

If you take a look at that image to the right, you see those red starbursts. Those are representative of the pulmonary veins. The pulmonary veins can have extensions of atrial myocardial that can fire on their own. When they start firing, they then get out into the atrium with multiple signals running into each other, running into small micro scars, creating atrial fibrillation.

In addition to those pulmonary veins that are firing, there are two components of the autonomic nervous system, both vagal input, as well as these localized ganglionic plexi, which help either trigger AFib, or promote the continuation of AFib once it starts. As you can see from these ganglionic plexi, they are extremely close to the os of the pulmonary veins. And when we actually do an ablation, if you keep this picture in mind for one of our later slides, you'll see that when we do an ablation, we're not only isolating the pulmonary veins, but also affecting these ganglionic plexi.

So some of the hemodynamic effects of AFib includes a decreased stroke volume, a shortened diastole, increased left atrial pressure volume, as well as aortic valve regurgitation, and then the irregular rapid ventricular rate. All of these increase the symptoms of AFib. Patients may present with a flip-flopping in their chest, shortness of breath, lightheadedness, just general fatigue, sometimes chest pain.

Some of the risk factors for development of atrial fibrillation include structural heart disease, whether that be heart failure, valvular myopathies, hypertension, age, diabetes, being male, as well as hyperthyroidism. So when we classify atrial fibrillation, we break it down into different components. After that first episode of AFib with the recurrence, that's when we determined what type of classification we had. So if an AFib episode lasts less than seven days-- usually less than 48 hours-- then it's considered a paroxysmal. Or if you cardiovert somebody within 48 hours, it's considered paroxysmal.

If you last more than seven days, it's considered persistent. Longstanding persistent AFib is anything that lasts for more than a year. And then if we have decided that we're not going to try to return somebody [INAUDIBLE], then we will label it as permanent AFib. So once again, I'm going to keep driving this home. The three mainstays of treatment: stroke prevention, rate control, and rhythm control. And we'll approach them in that order, pretty much by order of importance.

So with stroke prevention, we can use either medications or device-based therapy. When we determine somebody's risk factors for stroke, we end up using our CHADS VASc score. Our CHADS VASc score is a non-point scale that determines somebody's risk for stroke. The C stands for congestive heart failure, H stands for hypertension-- that can be either hypertension that is uncontrolled, or if somebody is on medications. If somebody is over the age of 75, they get two points.

As you look a little bit further down the line, if they're over the age of 65, then they get one point. Going back up, if they have diabetes, they get a point. A stroke automatically gives you two points. If you have any evidence of vascular disease, whether that be a prior MI, peripheral vascular disease, any kind of aortic plaque that may be seen on a transesophageal echocardiogram. And then if you're female, you get a point as well.

Adding all of those up, the higher your risk score, the higher your risk for stroke. What I usually quote to my patients is that if they have a CHADS VASc score between two and four, then their risk for stroke while being off of any anti-coagulation is about 2% to 4% per year. And it goes up from there exponentially. You see the different shades of red. Anybody who has two or more is considered high risk. If they have one point, they're considered an immediate risk. And if they have zero, they are low risk with the same risk as the general population for developing stroke.

In addition to the risk for stroke, we also evaluate their risk for bleeding. This is the HAS-BLED score. Each of these letters corresponds to a certain risk factor. If you have a score of three or more on this scale, then that puts you at increased risk for bleeding. As you can see, these two scales kind of overlap. So if somebody has hypertension, prior stroke, or they're elderly, then they're going to be at increased risk for stroke, as well as increased risk for bleed. The main determining factor would be that CHADS VASc score. But you do have to keep in the back of your mind what their risk for bleeding is based on the HAS-BLED score.

So when we utilize anti-coagulation regimen, we can either use Coumadin-- which is an age old-- or some of the newer agents, whether that be the dabigatran, rivaroxaban, apixaban, or edoxaban. Now, the newer agents are typically what we tend to use. But if somebody has been on Coumadin for a while, their INR is between two and three, and they're consistently between two and three, usually, I don't make a change in those patients. However, if they are extremely labile or if they've had any kind of bleeding complications from Coumadin from INR greater than three, then I strongly consider switching over to one of the NOACs.

These NOACs are great, because they do not have to be monitored on a routine basis. You do have to watch out for them whenever somebody has renal insufficiency, because that may either require a dosing adjustment or cessation of the NOAC altogether. So like I said before, when I usually start a new patient on an anti-coagulation regimen, I tend to go with the NOACs. But there is another one of those scores that I don't honestly don't use this one too much, which is the SAMe-TTR score.

In short, if you take a look at that chart to the right, the higher your score on this, the higher the risk for either bleeding complications or thrombotic complications while on Coumadin. And the higher your score on this scale, the less likely Coumadin is to be effective, and the more likelihood you should have to switch over to a NOAC. As you can see, if you have a high TTR score which is greater than two, then you're going to be at increased risk for labile INRs, you're going to be at increased risk for stroke, as well as bleeding, and ultimately, an increased risk for death.

So when deciding what kind of anti-coagulation to use, the first thing you should determine is their CHADS VASc score. So if somebody has a CHADS VASc score of zero, they do not need anti-coagulation. If a score of one, then they should either be on high dose aspirin, or full anti-coagulation. I tend to lean towards full anti-coagulation at this time, because I've found the bleeding risk on high dose aspirin as well as the NOACs are about the same.

So as far as those bleeding and side effects, of course, you've seen enough ads on TV and your patients have seen enough ads on TV to ask you about each of the NOACs, as well as Coumadin. You can see that the risk for major bleeding is about 3% across the board. So we're always looking for ways to decrease the risk for stroke without increasing the risk for bleeding. If NOACs or Coumadin are not an option, one of the reasons for not being on a NOAC is that there was a fear of non-reversibility. Whereas Coumadin could be given vitamin K for reversibility, the other NOACs did not have a reversing agent until now.

So this is based on a study in the *New England Journal of Medicine* for idarucizumab for dabigatran or Pradaxa reversal, the brand name being Praxbind. This is the first reversal agent for any of the NOACs. This is based on a relatively small study called the reverse AD study. It evaluated 90 patients who either had a higher risk for bleeding or were undergoing an urgent procedure. The dosing is in two different 2.5 gram infusions, and they evaluated the maximum reversal of the anti-coagulant effect.

So this is a very small chart, so we won't focus on this too much. But in summary, if you take a look at these four images, on the left hand side of each of the image, it represents basically, activity of anti-coagulation effect. That dotted line that's on each one represents pretty much normal activity as if the anti-coagulant was not on-board. And within 10 minutes of the first administration of this idarucizumab, all of the clotting times went back to normal.

So when they evaluated the outcomes, there was a complete reversal effect in about 90% to 98% of patients who had elevated clotting times. There was normal hemostasis obtained prior to the urgent procedures. And for those who were actively bleeding, cessation of bleeding happened within 11 hours. You would think with the reversal of any kind of blood thinning agent, that there may be an increased risk for a thrombotic event. That risk was only a 1% risk during this trial.

So in addition to the novel anti-coagulants, we also have device-based therapy for preventing stroke, one of these being the WATCHMAN Device, which is placed here at Wake. This is a nitinol parachute-type frame that is placed in the left atrial appendage. This is done by our EP staff under general anesthesia. It is a relatively quick procedure, though. And we'll get into a video of that in a second. And it only takes a one day hospital stay just to monitor for any kind of growing complications.

So what are the indications for a WATCHMAN Device? So if somebody has a high CHADS VASc score but they are suitable for warfarin, at least, temporarily. And they have a reason to seek this non-pharmacologic alternative to warfarin, they may be a candidate for the WATCHMAN Device. So after we've set somebody up for the procedure, we put them temporarily on warfarin for about 45 days after the procedure. Then, we continue aspirin and Plavix for about six months afterwards. And then aspirin alone, even a baby aspirin can be used.

So here's an example of the procedure itself. In the upper left hand corner of the screen, we do this under transesophageal echocardiogram guidance. We first get a transseptal puncture. That's a picture of the transseptal needle. And if you look at the live screen now, under this guidance, we then deployed the WATCHMAN Device into the left atrial appendage.

Once it's placed, then the video will reset. But we don't reset everything. But once it's placed, we then evaluate the left atrial appendage. For example, in the lower left hand corner, that is a similar view of the left atrial appendage with the WATCHMAN Device in place. So we have a color flow Doppler on right now, and it shows that there is no leakage around this device. Once this is in place, the reason why we put them on anti-coagulation is that you still have this prosthetic device in place. And we have to wait that six months before it becomes endothelialized, and essentially becomes part of the left atrial wall.

The study that evaluated this WATCHMAN Device was called the PROTECT AF study. It compared WATCHMAN with Coumadin. There were about 700 patients in this cohort. And the evaluation was a primary endpoint of stroke, thromboembolism, or cardiovascular death. And as you can see, when compared head-to-head, the rate of endpoint was about the same, once again at about 3%. And this was deemed to be non-inferior, meaning that it was the equivalent both efficacy, as well as safety as Coumadin.

When breaking it down into risk for stroke as well as all cause mortality, this Kaplan-Meier curve shows that there is no significant difference, even out to four years. And when they broke it down by subgroup analysis, if anything, there was a trend towards improved efficacy, comparing it to Coumadin. In the intention to treat analysis, it was definitely non-inferior, but as stated before, there's a trend towards efficacy with that risk ratio being less than one, but still spanning one.

So in addition to the WATCHMAN Device, there are other devices that are in use, including the Lariat-Epicardial left atrial appendage closure, which utilizes an epicardial access. What we do is we go epicardially, as well as endocardially. This is not done here because it's still kind of in trial technique. Two magnets form together to create a rail, and then a lasso is advanced over the left atrial appendage. A balloon is then blown up into the left atrial appendage. The Lasso is then tightened down. The balloon is deflated. And as the balloon gets pulled out, that lasso becomes fully tight.

Then, from that venogram, you can see that the left atrial appendage is completely walled off and there's no residual left atrial appendage. Over time, this becomes essentially a vestigial structure, and this 3D reconstruction of the left atrial appendage shows a before and after. In the before image, you can kind of make an outline of that chicken wing formation of the left atrial appendage. And then afterwards, on repeat evaluation, there's no presence of an appendage whatsoever.

So in addition to the stroke prevention, we also focus on rate control. That may be with medications, and we'll kind of fly through these, because everybody utilizes these medications both for blood pressure control, as well as rate control. So rate control is a good option for those who are asymptomatic with their AFib. As long as their rate is under control, and their stroke is prevented by anti-coagulation, then you don't have to move on to rhythm control. The AFib is still going to be at increased risk for stroke, so they still need to be on anti-coagulation.

So the AFFIRM trial, which is an older trial from 2002, evaluated both rate control as well as rhythm control for an outcome of mortality. There was actually no difference in mortality with rhythm versus rate control. And this is the evidence of why we say if somebody is asymptomatic with their AFib and their rate is under control, we stop there.

In addition to that, if somebody has a normal EF and they are asymptomatic, then we can be relatively lenient as far as the rate control goes, as long as their heart rate is consistently less than 110 beats per minute, then their risk for death from any kind of cardiovascular cause, worsening heart failure, stroke, embolism, or major bleeding is about the same as if we tried strict control.

So some of the options for rate control include beta blockers, calcium channel blockers, digoxin, as well as amiodarone. If somebody has no evidence of cardiovascular disease the beta blockers and calcium channel blockers will be options. If they have any evidence of LV dysfunction, you would not want to use the calcium channel blockers. If they have COPD, you'd want to try to avoid amiodarone as well.

So amiodarone is typically used as a second line agent, if the beta blockers or calcium channel blockers are not easily used, whether that be a limitation by blood pressure, or if they are acutely ill. So with the beta blocker usage, you can use pretty much any beta blocker that you're comfortable with for both acute control. You can give IV pushes of beta blockers, but then you can switch over to oral control later. Same thing with the calcium channel blockers. You have both IV, as well as p.o. forms. Usually, once we have somebody under rate control with something such as a Diltiazem drip, we switch over to short-acting calcium channel blockers as an inpatient. Once we are able to get to the 24 hour requirement, we then switch over to long-acting calcium channel blockers for outpatient use.

So as far as the indications for it, so a beta blocker/calcium channel blocker should be used for rate control. It can be used in an acute setting. You want to keep the ventricular rate within a physiologic range when exercising. If they have a heart rate of less than 80, that's a decent goal for symptomatic patients. If somebody is critically ill and they are hypotenstive or septic, amiodarone is a very valid choice, at the slight risk for acute conversion of about 25%.

So also, you can use AV node ablation if patients are not rate controlled with just medications. But that should not be a choice, if you have not already tried rate-controlling medications. Also, as I stated before, calcium channel blockers should not be used in decompensated heart failure. And dronaderone or Multaq should not be used in permanent AF because of the increased risk for death.

So I talked about dig as a possible use. It is a good use short-term, but long-term, I tend to stay away from the use of digoxin for rate control. And this is based on the TREAT A-F study, which is a large VA database study that evaluated patients who had non-valvular AFib. About 23% of those patients were on digoxin. And over the course of time, there was an independently associated increased risk for mortality with the use of digoxin.

So this is my little PowerPoint illustration. I apologize for this. This is very crude, very, very crude. That's all you can do on PowerPoint. That took maybe 30 minutes the first time I tried doing that. So this is AFib with RVR. So with the drugs that we use, once we get it under control, then the ventricular response is lower. We look for the lower ventricular response to decrease the symptom.

If medications do not work, then we can choose an AV node ablation and pacemaker. So the AV node ablation cuts out the communication between the top and bottom chambers of the heart. The AFib continues to occur in the atrium, but the ventricle is not responding whatsoever. When you don't have that response, then the ventricle usually beats at about 30 beats per minute. That is not good for sustaining a good quality of life, or even sustaining life. So that's why we have to put in a pacemaker.

So pacemakers can be transvenous. This is an example of a transvenous pacemaker. If somebody is in permanent AFib, we typically only put one lead down into the ventricle if they have paroxysmal AFib. But those paroxysms lead to a rapid ventricular response, and they have dual chamber pacemakers. But one thing that Wake does do-- and we were the first ones to place this in the region-- is the leadless pacemaker.

This is the Micra transcatheter pacemaker. It's placed through groin access into the right ventricle. It allows pacing. Everything is right there in that component. So there's no transvenous leads. The pacemaker, the battery, everything is right there. And it can last for up to 12 years at a time. There are specific indications for this. Can we play that? There are specific indications for this. The specific indications are if somebody has an indication for a single chamber pacemaker, then they can have this device.

So our common indication is somebody who has chronic atrial fibrillation, but either slow response themselves, or if we're about to do an AV node ablation, then this will be a good choice going forward. So as I said, this is a video outlining what we do. We end up getting access into the femoral vein. After running a wire up into the heart into the SVC, we then get our introducer sheath into place. This is about a two minute video, so we'll take a small break.

So once we get the introducer sheath into place, this creates our guide for putting the actual delivery system into the heart. The delivery system goes up through the introducer sheath. We then cross the tricuspid valve with deflection. We then aim for the apical septum or the apex of the right ventricle. We then inject contrast to confirm that we have a good position. And then once we've confirmed that we have good position and a good amount of pressure on the wall, we then deploy the actual device.

The device is still held on to by a tether system. Here's a direct image in the cadaver lab. So it has four little nitinol springs that hold into either the myocardium, or wrap around the trabeculae. As you can see in that image, the right ventricle has a lot of trabeculations, and we don't really appreciate that too much because we're not looking directly into the right ventricle too much. Now, once we confirm that all the numbers look good, then we release those tethers, clip the tethers free, and then the device is left there to work for the next 12 years.

So if somebody has low EF, then they probably wouldn't be a good candidate for this. And this is based on the Block HF trial. The Block HF trial evaluated patients who had heart failure and an EF less than or equal to 50%. And everybody had a BiV pacing capability, but they were randomized to RV pacing or biventricular pacing, and they evaluated at time of the death, heart failure or a drop in EF. As you can see from the Kaplan-Meier trial, even at one year, those who had biventricular pacing had better outcomes than those who had RV-only pacing. So somebody who has a low EF and we are expecting to pace their ventricle more than 40% of the time, they would typically get a BiV pacer.

So after rate control, there's rhythm control. As I talked about before, if somebody is still symptomatic after rate control, then rhythm control is our choice. There are two different classes of antiarrhythmic medications, including class 1c agents, including flecainide and propafenone. These are both great first line therapies, but they are contraindicated for obstructive heart disease, as well as heart failure.

The other class includes a class III. These are potassium channel blockers, including sotalol, amiodarone, dofetilide, dronedarone, well as ibutilide. Ibutilide is typically used for acute conversion, the others are used for more long term control. So when we make a decision on which type of antiarrhythmic to use, if somebody has no evidence of structural heart disease-- just hypertension-- then they can use pretty much anything they want to use.

As you can see from the chart, amiodarone is typically reserved as a second line therapy, just because of the long term side effects. If somebody does have coronary artery disease-- and that pretty much wipes out the class 1c agents-- you're limited to sotalol, dronaderone, or dofetilide. And if somebody has heart failure, dofetilide, and amiodarone are the medications of choice. But as you can see, if you failed one antiarrhythmic medication, then catheter ablation is on the table.

So if antiarrhythmic medications don't work on their own, then you can do an acute cardioversion. And a cardioversion is where we put pads on the front and back of the chest. If somebody has been in AFib for less than 48 hours, then we can anti-coagulate and perform a cardioversion without a preceding TEE or transesophageal echocardiogram. However, if they have been in for more than two days, the safest thing to do is to perform a TEE first; TEE being the transesophageal echocardiogram, which goes down in the esophagus. It has a close unobstructed view of the left atrial appendage, where 90% of left atrial clots come from. And if the appendage is clear, then we proceed with the cardioversion. The main risks for this procedure include aspiration as well as esophageal perforation. The risk for that is less than 1 in 500.

If we do see a clot such as that clot seen in this picture, then the patient would need to be anti-coagulated for another three weeks. They'd need a repeat TEE to make sure that that clot has resolved, and then proceed with the cardioversion. The reason why we do that repeat TEE is that sometimes that clot doesn't resolve after three weeks. I've had one patient where it took eight months of repeat TEEs before that clot had fully resolved before she could return to sinus rhythm.

So if antiarrythmia medications do not work, if you failed one oor if you've been intolerant to one medication, then they can be referred to EP for an ablation. The goals of ablation are to eliminate the trigger, as well as eliminate that substrate like we talked about at the beginning of the talk, and therefore, improve symptoms. This is more of a quality of life issue, not a quantity of life issue. It It isn't used as an indication for getting off of anti-coagulation, either. So we talked about this, and I'm going to drive this home. If somebody has failed or intolerant to one anti-arrythmic medication, then AFib ablation is on the table.

So with our procedures, we usually get a pre-op CT scan that shows the left atrial anatomy. Sometimes, there can be variations to the left atrial anatomy. Typically, you have two left and two right pulmonary veins, but you can have varying anatomies which may modify our approach. So once we place the catheters, we perform a transeptal puncture. We do 3D mapping to create a 3D shell of the left atrium, and then we perform our ablation. There are different types of ablation lesion sets. But as I said, we are mainly focusing on isolating the pulmonary veins and decreasing the atrial mass to decrease the likelihood for perpetuation of AFib.

So our typical way of doing it is a wide area circumferential ablation, which is in the upper left hand corner of the chart. We ablate around the antrums of the pulmonary veins and block the left and right atrium. But sometimes, it does take ablating within the carina between the two, as seen in that lower left hand lesion. Some other institutions may target just these fractionated atrial electrograms, but the most widespread use is the wide area circumferential ablation.

So this is a 3D reconstruction of the inside of the heart, with both the left and right pulmonary veins. Those dotted lines with those arrows pretty much show the trajectory and what we're trying to accomplish, by completely isolating the veins that traps the electrical activity inside. So therefore, those triggers cannot affect the rest of the atrium.

We test this by placing a lasso catheter, which is a multielectrode kind of ring-tip catheter inside the veins that pushes up against the wall and lets us know what the atrial activity or what the vein activity is before the ablation. And we look to make sure that we have complete entrance and exit blocks. So at baseline, an active pulmonary vein that is communicating with the atrium will have a sharp signal. But once we've completed that line to isolate the pulmonary veins, the pulmonary veins may fire on their own-- as shown in that middle picture-- independent of the atrium or the rest of the heart.

As you can see, you see the two arrows in the middle picture showing the pulmonary vein firing. But if you take a look at the top three lines, you can see QRS complexes just marching through that. We also test exit block by pacing around the pulmonary vein, so that if we see capture and it does not get out to the rest of the atrium, that has proven both entrance and exit block. We also give adenosine challenge to open up any small gaps that we may have missed on the first go around.

So for a long-standing persistent AFib, we tend to go in a stepwise fashion. But recent studies have shown that if you just focus on that wide area circumferential ablation, it's just as good as more extensive lesion sets. So we try to avoid doing too many lines, because that increases the risk for atrial flutters and atrial tachycardias in the future.

So in order to decrease the amount of radiation that we use during AFib ablations, we use 3D mapping systems. This is an example of our most commonly used 3D mapping system, which is the Carto-3 base system. This allows us to drop radiation down drastically. I think before we had the widespread use of the 3D systems, patients undergoing AFib ablations may undergo 120 minutes of flouro time. Now, we've cut that down to about 3 to 10 minutes, just depending.

So in addition to a radiofrequency ablation, there's also the cryoablation. This is a double layered balloon that pretty much contains liquid nitrogen that evaporates, causing ice crystals to form in the cells, and creates a scar tissue by directly damaging those cells.

This is an example of the cryo balloon ablation. This is known as an Arctic Front ablation. I don't have to have the sound with it. So with the Arctic Front ablation, what we do is we do the same transseptal technique to get over from the right atrium to the left atrium. And this is typically used for those who have paroxysmal AFib. Here, you see an active pulmonary vein. Once we get that transseptal access, we get that lasso catheter into the vein, just to see the electrical activity inside the vein. It also provides a rail system, so that we get a good direct entryway into the vein.

The balloon is expanded, and it's fairly compliant. So we can push that balloon fairly deep into the vein to pretty much plug up the entire vein. And we confirmed good plugging by injecting contrast to make sure that none of the contrast leaks out into the atrium. That's when we have a good tight seal. Once we have confirmed that, we turn on the ablation catheter and it ablates in a hemispheric fashion through eight jets. This freezes the tissue to about negative 60 degrees Celsius.

So that freezing and thawing technique-- which, the freezing lasts for about three minutes-- then creates a scar. That white line that you see there, it kind of represents a scar and the trapped electrical activity. So you say, well, which one is better for my patient? Honestly, both of them have about the same efficacy. And this was proven in the Fire and Ice trial. It paired both the radiofrequency ablation with the balloon catheter ablation. It evaluated over 750 patients who had symptomatic paroxysmal AFib-- so no persistent AFib-- who were refractory to antiarrhythmic therapy. And they evaluated a time-to-analysis event. So if they've had a recurrence of AFib lasting more than 30 seconds, any kind of atrial flutter, atrial tachycardia needing antiarrhythmic medications to control, or needing a repeat ablation.

And when compared head-to-head between the two, there was no significant difference in the outcome going forward. They did break down into different types of both RF and ablation catheters, the newer generation of cryoablation catheters worked a little bit better, because they now have a hemispheric freeze, as opposed to an equatorial freeze. And this was built as a non-inferiority trial. So it reached non-inferiority. There was no trend either way, as far as efficacy. As far as safety, there was a slight increased risk for damaging the phrenic nerve with the Cryoballoon when compared to the radiofrequency ablation.

So I know that this looks to be a low success rate, but this is once again, the combination of multiple things including recurrent AF, as well as the use of antiarrhythmic therapies. A big win for any kind of ablation is if we don't need to use antiarrythmics, that's a big win. A partial win is if the combination of ablations and antiarrythmics maintain sinus rhythm. And then if we do not reach either of those endpoints, then we consider a repeat ablation.

But long term, the success rate of antiarrythmic therapies can be anywhere between 20% and 40%. Amiodarone is kind of on that higher end of 40% to 50%, but the success rate for an ablation is I quote about 70% to 80%. And that's why if somebody has had one ablation, I always tell them they expect to have a second ablation at some point in time in the future. So long-term follow-up, like I said, there's no cure for AFib. There's still a decent recurrence rate of about 25% at five years. But like I said, once we get their risk for stroke under control, it's more of a quality of life issue going forward.

So since we talk about ablations, we do have to talk about the potential complications. So you may see some of these patients in follow-up, so there are certain things to look for after an AFib ablation, including tamponade. The risk is about 6%. This may be a slow or sudden fall in blood pressure that is not explained, that can respond somewhat to fluid-based therapy. This image right here is an image of an intracardiac echo that shows this pericardial effusion.

The pericardial effusion-- I only have one pointer and multiple screens, so I don't know what screen to point to. All right. I'm at a loss. Here's a better one that has labels. So this is a surface transthoracic echocardiogram in left peristernal lung view that shows both the right ventricle, left ventricle, and then behind the left ventricle, you see that PE that stands for the pericardial effusion. So it can be seen both during the procedure, as well as afterwards. So if you have any suspicion, please order a limited echo to see whether or not there's a pericardial effusion.

The treatment for this being a pericardiocentesis, usually in the subxiphoid area, once we drain this pericardial effusion, usually, it seals off whatever hole is there seals off. And we can remove the drain shortly thereafter. Other potential complications include pulmonary vein stenosis. When we first started ablating, the pulmonary veins would ablate within the pulmonary veins. And this would create this stenosis, but that's why we tend to stay in the atrium around the antrum of the pulmonary veins to limit the risk for a stenosis.

Some of the presentations-- and this can happen weeks to months later-- you can have increased chest pain, shortness of breath, just recurrent lung infections, that should increase your suspicion for possible pulmonary vein stenosis. If it's just a mild amount, less than 50% in the diameter, usually not a problem. They're usually only symptomatic once they get more than 70% stenosis, just about like with coronary artery disease. And the treatment is about like coronary artery disease too, whether that be balloon angioplasty or a stent in the pulmonary vein.

One of the most dangerous complications to this procedure-- and this is the reason why we try antiarrhythmics first-- is esophageal injury, especially atrial esophageal fistula. The risk of this is extremely, extremely low. I have not had this, but we have seen this transfer into our hospital at points in the past. The risk is less than 1 in 1,000. But if this does happen, where the atria and the esophagus communicate, then that's a greater than 80% mortality.

So this can manifest usually about two to four weeks after the procedure, a patient may come in with problems with swallowing, fevers or chills. Or if they have recurrent embolic events that are unexplained, then that may be an atrial esophageal fistula. You do not try to diagnose this with a TEE or endoscopy, the best way to do this is with a contrasted CT or MRI of the esophagus.

Other potential complications, we talked about this as being a potential complication with the Cryoballoon, includes phrenic nerve injury. It more commonly happens when we try to ablate the right-sided pulmonary veins. The phrenic nerve runs anterior to the right-sided pulmonary veins, put posterior to the superior and inferior vena cava. This is the nerve that controls the diaphragm. So if you damage this, then the diaphragm is not working. And the patient can be short of breath. The good thing is that if this does happen, all but about 1% recover after one year.

Other complications include stroke and TIAs, thromboembolisms can happen in 0% to 7% of the cases. We prevent this by temporarily putting somebody on extra doses of heparin to keep their blood thin, to prevent a clot from forming on the catheter. Once the catheter is pulled back to the right atrium, we reverse that with protamine. And there a less than 1 in 1,000 chance for death.

So some of the take home points that I want to drive home are that AFib ablation-- since that's what we do-- is a class I indication of somebody's failed antiarrhythmic therapy. It's a class IIa if they choose ablation as a first line therapy. If somebody has a higher risk score of stroke-- so a CHADS VASc score of greater than two, preferably greater than or equal to one, they should be on anti-coagulation. And also, know the potential complications of AFib ablation, and make sure that you inform your patients about these risks and what to look for after an ablation.