● 特別講演

Update on Radiofrequency Catheter Ablation of Atrial Fibrillation

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I would like to spend the first half of this lecture by discussing our efforts that were directed toward therapy for chronic atrial fibrillation. Catheter ablation of atrial fibrillation demands that we have at least an understanding of the electrophysiologic mechanisms which we target for ablations. In the classic understanding, atrial fibrillation was thought to be due to the presence of multiple wavelets of reentry within the right and the left atria. This hypothesis was first forwarded by Dr. Moe in the early 1900 s. Our understanding has advanced very little since that initial hypothesis, and if in fact this hypothesis is true, that atrial fibrillation is due not to a fixed anatomic abnormality, around which reentry occurs, but rather migrating wavelets of reentry, then an ablation procedure designed to cure that dysrhythmia must be based on delivery of ablation energy in a linear fashion. On the other hand, if we take the stance that atrial fibrillation is possibly due to a more focal mechanism, either due to abnormal automaticity or various forms of reentry on either a micro or macro reentrant basis, then it is possible that a procedure based on delivery of discrete ablation lesions could in fact be successful in eliminating atrial fibrillation.

Beginning in 1993, we elected to begin a protocol to investigate chronic atrial fibrillation and ablation of that dysrhythmia. We selected patients with the following clinical characteristics. The patients by and large had underlying cardiac pathology, including but not limited to coronary disease, valvular heart disease or primary cardiomyopathic states. In the presence of these underlying disease processes, patients had objectively enlarged atria. On echocardiographic evaluation, each of the patients presented with left atrial enlargement. In addition, on the very few examples of 12 electrocardiograms obtained during sinus rhythm, those patients also presented with impaired intra-atrial conduction；that is, P-wave duration which was prolonged or P-wave morphologies which were abnormal. These patients have chronic or persistent atrial fibrillation. The mean duration of atrial fibrillation in these patients was 4 years；that is, atrial fibrillation without documented sinus rhythm for 4 years in this patient population. The patients were addressed with a procedure that was adapted from the original surgical approach to atrial fibrillation, the so-called Maze procedure which was developed by Dr. Cox and his associates at Washington University in St. Louis. Here depicted is the original concept of the Maze. As you can see, that procedure is based on surgical incision in both the right and the left atria. These incisions were designed and strategically placed so as to permit activation of both the right and the left atrium, but limit that activation to such small segments that

reentry could not occur. A key portion of this procedure was total isolation of the pulmonary venous structures with this horseshoe-shaped incision placed posteriorly in the left atrium. Now, this incision is probably the most critical to the Maze procedure, but also serves as perhaps the weakest link in the Maze procedure, in that this horseshoe-shaped incision excludes-it isolates-approximately 35 to 40% of the left atrium. That is, the entire posterior aspect of the left atrium becomes dead tissue, and therefore atrial transport is markedly impaired. In an effort to avoid that limitation of the Maze procedure, the catheter ablation approach was devised to separate the pulmonary venous structures with incisions that are shaped like a cross. These incisions, or I should say linear ablations, were placed in addition to a line which proceeded from the top or roof of the left atrium laterally to the mitral anulus. This lesion was positioned so that it would pass just anterior to the left superior pulmonary vein. Also depicted are the right atrial lesions along the crista terminalis and posteriorly from the superior vena cava to the inferior vena cava. In addition, right atrial lesions were placed from the tricuspid anulus to the inferior vena cava orifice, a lesion which you now know is successful in treating Type I atrial flutter. A lesion was also positioned along the interatrial septum, proceeding from the limbus of the crista terminalis superiorly to the superior vena cava. This original procedure was performed in a stepwise fashion, so that the right atrial ablation portion was always accomplished first. We were hopeful that limiting ablation to the right atrium could in fact successfully eradicate atrial fibrillation.

Unfortunately, what we found was that with ablation in the right atrium along the crista terminalis posteriorly from the superior to inferior vena cava, and also along the subeustachian recess, from tricuspid anulus to inferior vena cava, these ablation lines were not successful in any of the first 15 patients. They were not successful in eliminating atrial fibrillation. Instead, what we observed was marked organization of right atrial activation. Here are very brief examples of rhythm prior to any right atrial ablation and following completion of the right atrial ablation lines.

In the high right atrium, there is fractionated and variable morphology on the electrograms. Also, at the region recorded from the ablation catheter, we see nearly continuous activation, variable morphology and a quite fractionated appearance. Following ablation, notice that the morphology in both the high right atrium and now with the ablation catheter repositioned in the posterior lateral portion of the subeustachian recess, there is regularization of the morphology and nearly regular intervals of activation, and this phenomenon is observed anywhere in the right atrium with the exception of the interatrial septum.

It was not until we proceeded to ablation in the left atrium that we observed organization and finally elimination of atrial fibrillation, and this occurred in a stepwise fashion. Here, following delivery of the initial left atrial ablation line from the roof of the left atrium to the mitral anulus, there is regional organization of electrograms as recorded in the coronary sinus, but there is still atrial fibrillation on the surface ECG. With isolation, then, of the pulmonary veins with that cross-shaped incision, we see further organization of intracardiac activation so that there now is a one-to-one relationship between left atrium and the right atrium, and on the surface ECG there is also the appearance of an unusual variety of atrial flutter. Finally, with

completion of the left atrial ablation, and here during radio frequency application, sinus rhythm is restored. Although the initial procedure was highly successful in restoring sinus rhythm, we found a very high percentage of patients to have recurrence of dysrhythmia in the form of very unusual atrial tachycardias. In this particular case, a patient two months following initial ablation of atrial fibrillation presented with an incessant atrial tachycardia, and the intercardiac activation sequence suggests a left atrial origin, and in fact with mapping in the left atrium near the left superior pulmonary vein orifice, we see very unusual electrograms that are low amplitude, fractionated, and these multiple fragmented electrograms continue throughout the entire cardiac cycle. With discrete ablation at the left superior pulmonary vein orifice in this patient, sinus rhythm was restored.

In response to the very high percentage of recurrent tachycardia due to reentry around pulmonary veins, we modified the ablation procedure beginning with the 16th patient. Beginning with the 16th patient, the left atrial ablation lines were modified to incorporate each of the pulmonary vein ostia so as to prevent reentry around these fixed anatomic obstacles. Notice that with the reorganization of the lines of ablation, we still permitted activation posteriorly of the left atrium, and anteriorly this incision did not proceed all the way to the AV groove so that activation could proceed anteriorly into the appendage. Once again, the entire left atrial activation sequence is preserved, but we have now provided for a procedure that prevents reentry around each of these pulmonary veins. Also, beginning with the 16th patient, we avoided the right atrial ablation lines.

We began the procedure as a left atrial ablation procedure, and what we found in the 16th through 36th patient, which was our final case, was that ablation in the left atrium alone is sufficient to eliminate atrial fibrillation. We no longer required ablation along these lines depicted. The only right atrial ablation that was required was that which is necessary for cure of Type I atrial flutter, from the tricuspid valve anulus to the inferior vena cava orifice, because in each of our patients Type I atrial flutter was inducible on follow-up electrophysiologic studies. With the elimination of atrial fibrillation, each patient underwent extensive programmed stimulation, and during those programmed stimulation studies Type I atrial flutter was universally inducible, and subsequently we ablated it. The atrial fibrillation procedure is now solely based within the left atrium.

I would like to show you a series of radiographs that now depict the current version of the catheter ablation procedure. Here you see right anterior oblique and left anterior oblique views of the initial left atrial ablation line. In the right anterior oblique, you can see that our ablation catheter is delivered to transseptal at this area, and it proceeds laterally, then posteriorly, to the origin of the right superior pulmonary vein. You can appreciate that in a left anterior oblique view. Here the catheter is delivered laterally along the mitral anulus. It proceeds superiorly, then medially, toward the right superior pulmonary vein orifice. With the maintenance of a tip temperature at 50 degrees centigrade, this catheter then is pulled back in 30-second increments. Every 30 seconds the catheter is pulled back two to three millimeters, and in that method linear ablation is accomplished along this entire line. At this position we pass by the left superior pulmonary vein orifice, and at this position, very low near the mitral anulus,

we pass by the left inferior pulmonary vein.

The second ablation line is produced posteriorly within the left atrium. Again, we have right anterior oblique view and left anterior oblique view. In the right anterior oblique view, you can see that the catheter is delivered superiorly in the left atrium, then travels posteriorly and inferiorly to rest approximately one centimeter above the mitral anulus. That catheter, as before, then maintaining a tip temperature of 50 degrees centigrade, is progressively pulled back until a complete line of ablation is produced to the right superior pulmonary vein. At approximately this position, the right inferior pulmonary vein is also bisected. With these two catheter passes, then, we have connected the right inferior, right superior, left superior and left inferior pulmonary veins. A rectangular incision has been made to incorporate or bisect each of the pulmonary veins.

The third ablation line starts from near the mouth of the left superior pulmonary vein. It proceeds anteriorly and then medially to rest very near the root of the aorta. That is a region that I refer to as the left fibrous trigone. In this LAO position it is difficult to tell how anterior the catheter is. But as it is pulled back it will then proceed over the left atrial roof and then to the left superior pulmonary vein. You will appreciate how anterior this catheter is.

When it is passed anteriorly, the catheter is in a position to avoid entry into the left atrial appendage, which is approximately here. But you can see how anteriorly it is directed. Here is the first ablation line. You can see it is about 90 degrees opposed from that first ablation line. The left superior pulmonary vein is at approximately this position.

In rare cases, when the left atrium is extremely tall or cylindrical shaped, a fourth ablation line is necessary. And that is a line that proceeds from the right inferior pulmonary vein posteriorly. You can see the posterior application of the catheter, and it proceeds laterally. And we stop short of the left inferior pulmonary vein, so as to allow activation into this rectangle back here. But in rare cases where the left atrium is very long or cylindrical in shape, this ablation line is also necessary for elimination of atrial fibrillation.

When these catheters are pulled in that order, we can see an electrogram sequence as follows. Here is a baseline tracing in a patient with mitral stenosis and recurrent bouts of pulmonary edema requiring intensive care unit admission because of rapid atrial fibrillation. She was refractory to approximately five medical regimens including amiodarone. That baseline, as you can see in the electrograms, is very consistent with atrial fibrillation. Here the ablation catheter rests near the right superior pulmonary vein. It is situated to begin our first ablation line. With completion of that first ablation line, there is a very marked change in the character of the intracardiac activation sequence. Here in the coronary sinus you can see very regular activation and a very regular morphology. Even on the ablation catheter, when it is repositioned back near the right superior pulmonary vein, a very regular activation sequence. With application of the third ablation line, we now see that there is a transition from the regular appearance of intracardiac activation but atrial fibrillation on the surface ECG to a regular atrial tachycardia on the surface ECG and a one-to-one relationship between right atrium and left atrium as recorded in the coronary sinus. So again, we are seeing a very gradual progression-organization and progression to sinus as the catheter approaches the left superior pulmo-

nary vein. With this third ablation line, as we approach the left superior pulmonary vein orifice, the atrial tachycardia is terminated and sinus rhythm is restored.

Now, in spite of the modification of our procedure to prevent reentry around the pulmonary veins, we still frequently see troublesome areas of reentry around the left superior pulmonary vein or right superior pulmonary vein. In this case the catheter is positioned at the entry to the left superior pulmonary vein. In this LAO view how high we are off of the mitral anulus. This is the right anterior oblique view. In the left anterior oblique view you can see that we are very near the cardiac silhouette.

At that particular catheter position in this patient, we found electrograms that presented with nearly continuous activation. This is a patient in whom all of the left atrial ablation lines had been performed, yet we were still in atrial tachycardia, which was quite organized, organized with respect to both right and left atrial activation. It appears as a very unusual form of atrial flutter on the surface ECG. The negative appearance of the atrial activation in the limb lead 1, isoelectric in the limb lead 2, positive in 3 and positive in V 1. This unusual variety of atrial flutter was in fact reentry around the left superior pulmonary vein orifice. In this particular patient, therefore, it was application of linear ablation that uncovered a very rapid reentry around the left superior pulmonary vein. With discrete ablation at the left superior pulmonary vein orifice, tachycardia was terminated. I would like to leave the pulmonary veins for right now, but please remember the pulmonary veins. They are troublesome not only for chronic atrial fibrillation, but they also prove to be intriguing structures when we consider paroxysmal atrial fibrillation.

Any ablation procedure for atrial fibrillation can be measured as a successful procedure only if we are able to restore atrial transport function. So our initial investigation was not just to investigate the possibility of restoring sinus rhythm in those who had chronic atrial fibrillation, but also to investigate the quality of that sinus rhythm in terms of atrial transport. Immediately following ablation, it was easy to become disillusioned because none of our patients had measurable atrial transport. Here, it is interrogation of the left ventricular inflow. You see passive filling, then with a good imagination, perhaps some active filling, but very, very poor. On the next cycle there was no active filling. On this cycle again, perhaps minimal. But this is insignificant on a clinical basis. There is no left atrial transport immediately after restoration of sinus rhythm.

Upon following these patients, we observed that there was, over time, a restitution of atrial transport function. This phenomenon is very similar to that observed following direct current cardioversion of atrial fibrillation. Initially, there is very poor or immeasurable atrial transport, but with time atrial transport is restored to a normal value. And here in the same patient, we have serial interrogation of left ventricular inflow. This is immediately post-three, six and twelve months after ablation, the progressive improvement in left atrial transport was observed.

Here is another example of left atrial transport one year following ablation of chronic atrial fibrillation, the left atrial transport with active filling velocities was exceeding six-tenths of a meter per second. These are normal values. Not only is there grossly normal left atrial

transport, but if you evaluate atrial transport on a regional basis and look at left atrial appendage function, here is a transesophageal echo which interrogates the appendage-the left atrial appendage-very high velocity emptying out of the left atrial appendage on a very consistent basis. We see a very consistent ejection from the left atrial appendage. If you also evaluate left atrial mechanics in terms of pulmonary venous flow, you see a very normal appearance of reversal in the pulmonary veins, reversal of blood flow as a result of atrial mechanical function. And you can see that very consistently on a beat-to-beat basis in these patients during their chronic follow-up period. These transesophageal echos were performed one year following ablation.

This is to emphasize that not only is there a return of normal a wave or active atrial transport with velocities exceeding one-half of a meter per second, but the ratio of active to passive filling also returns to normal with a ratio of less than 2. Interestingly, with restoration of mechanical transport, there is also significant anatomic remodeling. There is normalization of left atrial volume. We have observed a reduction in left atrial volume of approximately 30%. Not only does left atrial transport return to normal, but also does right atrial transport. Here is the right ventricular inflow interrogation, and there is passive filling followed by very healthy and vigorous active filling, and that is consistent on a beat-to-beat basis.

This returns also to normal relative ratio of active to passive filling below 1.5 at twelve months following ablation. So what have we learned as a result of this rather extensive investigation for ablation of chronic atrial fibrillation? We have learned that the patients have improved left ventricular function from a baseline of 49% to 56%. We have learned that they have sustained sinus rhythm 80% of our patients. With now follow-up of two years, they have maintained sinus rhythm. These numbers are not accurate but the percentage is 80%. We now have 36 patients. They have increased left ventricular ejection fraction, as I have shown. They have restored atrial mechanical function, and I have to emphasize that it is both right and left atrial mechanical function. There is normalization of left atrial volume. The patients do not require chronic antiarrhythmic medication following the ablation procedure. On a subjective status, they all enjoy improved functional status.

Unfortunately, this procedure is not practical in its current form. It requires an extensive, an exhaustive, physical effort to do this procedure. It requires attendance at the cath table for twelve and a quarter hours. It requires fluoroscopy support of over 100 minutes. And over half of the patients require a second ablation procedure to address atrial tachycardia, usually reentry around a pulmonary vein or occasionally around the mitral anulus. That second procedure is not a procedure which requires linear ablation, but rather focal ablation at a specific site responsible for the reentry.

The procedure is not safe in its current form. The reason I make that statement is that in these 36 patients I have observed more complications than in my prior ten years of ablation experience. Two patients have suffered from stroke two out of 36. The very first patient that we enrolled in the protocol suffered a stroke from which he fully recovered. A second patient with a rare antithrombin 3 deficiency syndrome had a stroke as well. We do not routinely screen, of course, for antithrombin 3 deficiency. Pericardial effusion which did not require

intervention was observed in one patient. Adult respiratory distress syndrome because we do this procedure under general anesthesia, and many of the patients have been subjected to chronic corterone or amiodarone therapy, so adult respiratory distress syndrome has actually been seen in two patients. Pericarditis which occurred one week following ablation in one patient required only initiation of nonsteroidal antiinflammatories. Urinary tract infection from Foley catheter in two patients and gastrointestinal bleeding from the use of high-dose heparin and reinitiation of coumadin therapy in two patients. This did not require any emergent medical intervention for the GI bleeding. It was from internal hemorrhage in both cases. Now that I have told you about a procedure which cannot be considered clinically relevant in its current form, I would like to move on to perhaps a more exciting procedure that addresses not chronic atrial fibrillation, but atrial fibrillation which occurs on a paroxysmal basis. That is fibrillation which has been referred to as focal atrial fibrillation. I do not know whether it is due to automaticity or perhaps reentry, but regardless of the mechanism it is amenable to delivery of discrete ablation lesions.

I would first like to outline the clinical characteristics of a patient who might benefit from discrete ablation for paroxysmal atrial fibrillation. These is a group of patients who have normal cardiac anatomy as well as normal cardiac function. That is to say, their echocardiogram is normal. They do not have underlying disease such as coronary disease, valvular heart disease or primary cardiomyopathies.

On the twelve leads electrocardiograms during sinus rhythm, they have evidence of normal interatrial conduction. This is a very crude assessment, but on the surface ECG they have normal P-wave duration and normal P-wave morphology. In spite of that, they have very frequent and often complex forms of atrial premature depolarizations. I would like to emphasize that these patients will have frequent atrial premature depolarizations. They occur on nearly an incessant basis. They have well documented paroxysmal atrial fibrillation which is medically refractory, and if you evaluate them extensively with ultramonitors, you will often identify periods of quite regular supraventricular tachycardia, a very regular, narrow QRS, complex tachycardia.

This is a typical patient with paroxysmal atrial fibrillation occurring on a focal basis. The baseline cardiac rhythm that you see is one of sinus rhythm with frequent and often multiple atrial premature depolarizations. Here is a sinus beat, two premature atrials, another sinus beat, a sinus beat followed by yet two more atrial premature depolarizations. This is very typical for the patient with focal atrial fibrillation.

When investigating the source of atrial premature depolarizations in these patients, we observed that there was activity isolated to the pulmonary venous structures, almost always the right superior pulmonary vein or the left superior pulmonary vein as shown in this case. In the pulmonary veins you will see isolated discharge which you would be tempted to discard or just ignore if it occurred once or twice. But if you leave the catheter in a stable position, as you see here in a 30-degree LAO, the catheter is positioned well beyond the cardiac silhouette. In a 30-degree RAL view you can appreciate that it is indeed in the left superior pulmonary vein. When that catheter is permitted to reside in the pulmonary vein in a stable fashion, as you will

repeatedly observe in this patient, we repeatedly observed this focal discharge.

Not only did it occur in isolation but it also occurred prior to each atrial premature depolarization, and here you see an example of both forms of focal discharge. Here the focal discharge is followed by an atrial premature depolarization. But here the focal discharge does not result in a global atrial response. That discharge is isolated to the pulmonary veins. It does not exit to the left atrium.

Furthermore, if you continue to observe, you will see that each and every atrial premature is in fact preceeded by these premature discharges. Here is a case where each atrial premature is preceded by these high frequency discharges which are isolated to the pulmonary vein. So it is possible to see the discharges without response in the left atrium, but you will always observe these discharges before atrial premature depolarizations.

If you continue to observe, you will also see that these discharges become nearly continuous during periods where multiple atrial premature depolarizations are recorded. Here is a series of three atrial premature depolarizations, and during that period of time you see very high-density activity within the pulmonary venous structure. This is very, very classic.

Here is yet another example, with repetitive atrial discharges. There is nearly continuous discharge at very, very high rates within the pulmonary vein. I would like to emphasize that this activity is not just a few milliseconds before atrial activation, but hundreds of milliseconds, typically 100 to 160 milliseconds prior to any atrial electrogram that you record elsewhere. Here is an example of that, with the ablation catheter resting in a pulmonary vein. You see this focal discharge. The earliest atrial electrogram that we see anywhere else is the low septal atrium followed very closely thereafter by high right atrium and coronary sinus. In fact, the coronary sinus and His activation may in fact be identical. This is a case where the activation was occurring in a right superior pulmonary vein, but it was occurring literally 100 milliseconds prior to activation of either the low septal atrium or coronary sinus. This is not a subtle finding. It is always 100 or 150 or even greater milliseconds. If one is lucky enough to observe this spontaneous onset of atrial fibrillation, here you see a discharge without atrial response, and here a discharge produces atrial fibrillation, and that atrial fibrillation will continue as long as focal discharge occurs in the pulmonary vein. It is extremely important to observe these phenomena during sinus rhythm.

In the next you will see why that is important. During atrial fibrillation you cannot distinguish focal atrial fibrillation from the more classic form of reentrant atrial fibrillation. Here is a case of atrial fibrillation caused by right superior pulmonary venous discharge, and the intercardiac electrograms from the coronary sinus from the His bundle, from the high right atrium, are indistinguishable from any electrograms that you might record from a patient who is in atrial fibrillation for the past six years. It is important to see these patients and to see these phenomena during sinus rhythm. I know of no way yet to discern the mechanism of atrial fibrillation, may be it focal or the classic macro-reentry. I know of no way to discern the mechanism when atrial fibrillation is present. Had we not observed the onset of this episode of atrial fibrillation, we would not be able to tell what its mechanism is.

One of the hallmarks, as I previously stated, of focal atrial fibrillation is its ability to take

on a characteristic of superventricular tachycardia-that is, a regular and narrow complex tachycardia. Here is an example of that, spontaneous conversion from atrial fibrillation to a regular-appearing tachycardia. This particular tachycardia has a one-to-one, A to B relationship. If you look very closely at where the ablation catheter is positioned in the right superior pulmonary vein, there is no difference in the local electrogram characteristics. Whether the patient is in atrial fibrillation or whether they are in this organized-appearing tachycardia, the right superior pulmonary vein activity is identical. This shows a detail of the superventricular tachycardia. And in this detail you can see the ablation catheter with high rate of discharge and a two-to-one exit to the left atrium. So there is a fixed two-to-one exit block during this period of time. This now permits the left and right atrial activation to appear quite organized, and this produces a very mundane or normal-looking superventricular tachycardia. Here we can see that tachycardia degenerate back to atrial fibrillation as exit block disappears. Exit block from where the ablation catheter rests in the right superior pulmonary vein has now given way to one-to-one conduction out of the right superior pulmonary vein and now gives the appearance of atrial fibrillation.

Another mechanism of superventricular tachycardia is reversion to Type I atrial flutter. This is seen very commonly actually. Here a patient is seen with the onset of atrial fibrillation. The ablation catheter is resting in a pulmonary vein. You can see very low amplitude, fractionated atrial electrograms beginning here all the way over to here. We see atrial fibrillation on the surface ECG now organized to Type I atrial flutter. Type I atrial flutter is very commonly found in these patients, so not only do they require focal ablation within a pulmonary vein, but they usually require delivery of Type I atrial flutter ablation lesion from tricuspid anulus to inferior vena cava.

So the atrial fibrillation can in fact be cured with a discrete ablation. But to render the patient completely free of dysrhythmia also requires ablation of Type I atrial flutter in many of these patients.

Now, as I said, the most common area in which we identify this focal discharge is in the right superior pulmonary vein. Here we see a right anterial oblique and left anterial oblique view of a catheter positioned at the origin of a focal atrial fibrillation. It is not a subtle finding. This catheter is well beyond the posterior aspect of the left atrium. This is a full centimeter beyond the left atrial posterior edge. And you can see here we have gone trans-septal, but now we are wrapping all the way posteriorly behind the right atrium, where the ablation actually takes place. This is not ablation within the left atrium. It is not even ablation at the orifice of the right superior pulmonary vein. This is ablation within the pulmonary vein itself.

What we have found is that the pulmonary veins are in fact quite complex. Here is an angiogram of the right superior pulmonary vein. It is in a right anterior oblique view. Here you can see the sheath as it goes trans-septal, then points posteriorly and superiorly toward the orifice, right here, of the right superior pulmonary vein. If one maps the right superior pulmonary vein, you see electrical activity well into the small tributaries of the main vein. In fact, ablation is commonly performed in these small tributaries to the right superior pulmonary vein proper. During angiography you actually see contraction within the right superior pulmo-

nary vein. That large body actually contracts. This is probably better described as part of the left atrium rather than as the right superior pulmonary vein. This is part of the left atrium both mechanically and electrophysiologically.

Here is an example of a catheter position where a left superior pulmonary vein fibrillation was ablated. Again, you can appreciate that the catheter extends well beyond the cardiac silhouette. The left superior pulmonary vein angiography demonstrates the complexity of this structure. Here you can see the sheath is actually directed into the pulmonary vein, which is seen here with its various tributaries, and applies to the left atrium at about this region. The sheath itself is actually already a full centimeter into the pulmonary vein. But you can see that ablation and mapping within these structures can be an incredibly challenging exercise. But if you are willing to tackle it, you will be encouraged by the results. And this means that you will cure atrial fibrillation using very standard equipment. This does not require anything special. It requires a routine ablation catheter and a routine transseptal approach to the left atrium. Here is an example of application of radio-frequency energy during a period of time when the atrial fibrillation had actually organized to a supraventricular tachycardia with a two-to-one atrial ventricular ratio. This type of tachycardia behaves identical to a Wolfe-Parkinson-White patient in that if the catheter is properly positioned with application of energy, the tachycardia will terminate within several seconds.

The lessons that we should take home from these experiences are as follows. First and foremost, atrial fibrillation. The term describes a family of dysrhythmias. Atrial fibrillation is not a single dysrhythmia. There are many, many types of atrial fibrillation. They look identical on the surface ECG, but they are different dysrhythmias. These different dysrhythmias require different ablation approaches. In 1997 ablation should be considered the therapy of choice for atrial fibrillation which is occurring on a focal basis ; that is, high-frequency discharge within pulmonary venous structures. I must emphasize, however, that chronic atrial fibrillation, which probably is on the basis of multiple wavelet reentry, cannot be ablated in a clinically relevant fashion. Do not attempt the catheter Maze procedure unless it is being performed under approved investigational protocols. It is a truly investigational procedure and will remain investigational until tremendous advances in technology become available.

Unfortunately, when a patient comes to the electrophysiology laboratory, there is no way to discern whether we are dealing with focal or multiple wavelet fibrillation, so that standard catheter-based recording modes are insufficient during atrial fibrillation to determine what the underlying mechanism is. The patient must be in sinus rhythm before standard catheter based recording modes and you can differentiate the type of fibrillation that your patient suffers from. And with that I will conclude our lecture.

<本稿は本研究会での講演速記録より作成したものである>