Catheter Ablation of Supraventricular Arrhythmias:
State of the Art

FRED MORADY

From the Division of Cardiology, Department of Medicine, University of Michigan, Ann Arbor, Michigan

Introduction

The era of catheter ablation for the treatment of arrhythmias began in 1981 when Dr. Melvin Scheinman performed the first atrioventricular junction ablation using direct current shocks in a patient with drug refractory atrial fibrillation and an uncontrolled ventricular rate.[1] Because of the barotrauma associated with intracardiac, high energy shocks, direct current catheter ablation played only a limited role in the treatment of arrhythmias. With the advent of radiofrequency catheter ablation in the late 1980s, the range of arrhythmias amenable to catheter ablation and the number of patients that could be treated safely by catheter ablation vastly expanded. In the past decade, radiofrequency catheter ablation has become firmly established as first line therapy for paroxysmal supraventricular tachycardia and typical atrial flutter. Furthermore, although still in an evolutionary phase, catheter ablation to eliminate atrial fibrillation has been demonstrated in the past 5 years to be feasible and clinically useful. The intent of this article is to review the current status of catheter ablation for the treatment of paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation.

Paroxysmal Supraventricular Tachycardia
Atrioventricular Nodal Reentrant Tachycardia

The most common mechanism of paroxysmal supraventricular tachycardia is atrioventricular nodal reentrant tachycardia. When radiofrequency catheter ablation first was used to eliminate atrioventricular nodal reentrant tachycardia, the fast atrioventricular nodal pathway was targeted. Fast pathway ablation was associated with a success rate in the range of 80–90%, and with a risk of high grade atrioventricular block ranging from 0 to as high as 21%.[2-4]

Fast pathway ablation was replaced in short order by slow pathway ablation because of its greater efficacy and lower risk of high grade atrioventricular block.[3,5-7] In the initial reports, the success rate of slow pathway ablation ranged from 98 to 100%, and the incidence of high grade atrioventricular block was only 0–1%.[5,6]

To ablate the slow pathway, radiofrequency energy is applied at the posteroseptal right atrial sites near the ostium of the coronary sinus. An anatomic approach can be used to create a line of block in the posterior septum, or target sites can be selected based on electrogram characteristics.[3,5-7] Discrete potentials between the atrial and ventricular electrograms in the posterior septum of the right atrium, which may represent activation of the slow pathway, were found to be a useful guide to ablation.[5,6] The most efficient technique for identifying slow pathway ablation sites may be a combined anatomic-electrogram mapping approach, in which energy is applied at posteroseptal sites where there is a small atrioventricular electrogram ratio and a fragmented or multicomponent atrial electrogram.[8] Because electrograms with these characteristics can be recorded near the tricuspid annulus at sites that are distant from the slow pathway,[9] the region that is explored with the ablation catheter should be limited to the posterior septum, near the coronary sinus ostium.

When a conventional, 4–mm-tip ablation catheter is used with a temperature feedback system, typical settings consist of a maximum power of 50 W and a maximum temperature of $55°C–60°C$. Effective slow pathway ablation sites almost always display an irregular, accelerated junctional rhythm during an application of radiofrequency energy.[10] The junctional ectopy is not specific to successful ablation sites, and it also occurs commonly during ineffective applications of radiofrequency energy in the posterior septum.[10]

It is important to monitor ventriculoatrial (VA) conduction during the junctional ectopy. High grade atrioventricular block is heralded by VA block, and atrioventricular block usually is avoided by the immediate termination of radiofrequency energy delivery on the first instance of VA conduction delay or block.[10]

Another technique for avoiding atrioventricular block is to deliver the radiofrequency energy during atrial pacing at a cycle length that is short enough to override the junctional rhythm. The atrioventricular interval is monitored during applications of radiofrequency energy, and energy delivery is aborted if the atrioventricular interval

Address for reprints: Fred Morady, M.D., Cardiology, TC B1 140, 1500 E. Medical Center Dr., Ann Arbor, MI, 48109-0311. Fax: (734) 936-7026; e-mail: fmorady@umich.edu

prolongs. If atrial pacing at a cycle length short enough to override the junctional ectopy results in atrioventricular Wenckebach block even before the onset of radiofrequency energy delivery, isoproterenol or atropine can be used to shorten the atrioventricular block cycle length and maintain 1:1 atrioventricular conduction during pacing.

If slow pathway ablation cannot be achieved in the posteroseptal right atrium, it may be worthwhile to deliver radiofrequency energy at sites in the proximal coronary sinus, close to the ostium. Rarely, successful slow pathway ablation may require an application of energy on the left side of the posterior septum, along the mitral annulus.[11]

The optimal endpoint for slow pathway ablation is the complete elimination of slow pathway conduction. However, complete slow pathway ablation may not be necessary to achieve a successful clinical outcome. If atrioventricular nodal reentrant tachycardia was inducible before ablation, residual slow pathway function with a single atrioventricular nodal reentrant echo is an acceptable endpoint.[6,12] Although there may still be evidence of dual atrioventricular nodal pathways, the recurrence rate of atrioventricular nodal reentrant tachycardia during follow-up is no higher than when there is complete slow pathway ablation, provided there is no more than a single atrioventricular nodal echo beat, even during infusion of isoproterenol.[12]

Some patients with atrioventricular nodal reentrant tachycardia have a first-degree atrioventricular block during sinus rhythm, suggesting the possibility of absent anterograde fast pathway conduction and a high risk of complete atrioventricular block if the slow pathway is ablated. However, in patients with first-degree atrioventricular block, the atrial-His interval usually remains stable or shortens after slow pathway ablation.[13,14] The fast pathway may be affected by an electrotonic interaction with the slow pathway, and elimination of this effect by slow pathway ablation often shortens the effective refractory period of the fast pathway.[14]

The initial studies of patients undergoing slow pathway ablation were published in the first half of the 1990s.[3−7] Since that time, several years of experience have accrued in large numbers of patients. To obtain an accurate view of the outcomes obtained in contemporary practice, several electrophysiological laboratories (see *Acknowledgment*) were surveyed for their results of slow pathway ablation during the 5-year interval of 1997–2002. In a pooled sample size of 8,230 patients with atrioventricular nodal reentrant tachycardia, a repeat ablation procedure was necessary in 1.3% of patients, and the long-term success rate in eliminating atrioventricular nodal reentrant tachycardia

was 99%. High grade atrioventricular block requiring implantation of a pacemaker occurred in 0.4% of patients. These results confirm the highly favorable risk to benefit ratio of radiofrequency slow pathway ablation.

Recent studies have demonstrated that slow pathway ablation can be performed by transvenous catheter cryoablation.[15,16] Two potential advantages of cryoablation over radiofrequency ablation are catheter stability during ablation and the reversibility of atrioventricular block.[15] However, given the high success rate and low risk of radiofrequency slow pathway ablation, it may be difficult to demonstrate a clinical advantage of cryoablation over radiofrequency ablation of atrioventricular nodal reentrant tachycardia.

Because of a favorable risk to benefit ratio, radiofrequency catheter ablation of atrioventricular nodal reentrant tachycardia has become the treatment of choice for most patients with atrioventricular nodal reentrant tachycardia who are symptomatic enough to require therapy. Many patients may elect to undergo radiofrequency catheter ablation as first line therapy to avoid bothersome symptoms of tachycardia and/or the need for antiarrhythmic drug therapy.

Orthodromic Reciprocating Tachycardia

The second most common mechanism of paroxysmal supraventricular tachycardia is orthodromic reciprocating tachycardia using an accessory pathway in the retrograde direction. In 1984, in the first report of catheter ablation of an accessory pathway, a 200-J shock was delivered near the ostium of the coronary sinus to ablate a posteroseptal accessory pathway.[17] A long-term success rate of 67% was achieved using direct current shocks to ablate posteroseptal accessory pathways.[18] Direct current ablation of left- and right-sided accessory pathways also had only a modest success rate and was associated with a significant risk of serious complications like myocardial rupture.[19−23] Therefore, direct current ablation of accessory pathways was usually limited to drug refractory patients who had severe symptoms. When radiofrequency energy was substituted for direct current shocks, catheter ablation of accessory pathways in all locations became feasible and relatively safe, and the selection criteria for the procedure greatly broadened.

Accessory pathways most often are located in the free wall of the left ventricle. These accessory pathways can be mapped and ablated using a retrograde aortic or transseptal approach. Advantages of the transseptal approach include better maneuverability of the mapping/ablation catheter and avoidance of the risk of arterial complications, like dissection of a coronary artery.[24,25] However, the

transeptal approach introduces the risk of complications like coronary air embolism,[26] and may not allow firm tissue contact as easily as with the retrograde approach. In patients with a prosthetic aortic valve or a diseased aorta, the transeptal approach clearly is the preferred technique. However, in studies that have compared the retrograde and transeptal approaches, the overall success rates with both approaches have been similar,[24,27,28] and in most cases the primary determinant of which technique to use is operator preference.

Approximately 20% of accessory pathways are posteroseptal in location, and these pathways are most often ablatable using a right-sided approach with delivery of radiofrequency energy along the posteroseptal aspect of the tricuspid annulus or within the proximal portion of the coronary sinus. In approximately 20% of patients, the accessory pathway is located on the left side of the posterior septum, and these pathways are ablatable using a transeptal or retrograde aortic approach, according to operator preference. Findings that suggest the need for a left-sided approach consist of an R:S ratio > 1 in lead V_1 (when there is overt preexcitation), a 10–25-ms increase in the VA interval in association with a left bundle branch block during orthodromic reciprocating tachycardia, and a ΔVA > 25 ms during orthodromic reciprocating tachycardia.[29]

The next most common site of accessory pathways is along the right free wall. These accessory pathways can be approached from the inferior or superior vena cava. When the inferior vena cava approach is used, long guiding sheaths that extend into the right atrium and that have distal configurations tailored to specific locations along the tricuspid annulus provide support for the mapping/ablation catheter and improve catheter stability.[30]

Approximately 2% of accessory pathways are anteroseptal or mid-septal, and these accessory pathways may be challenging to ablate because of their proximity to the atrioventricular junction. Because cryothermal ablation minimizes the risk of atrioventricular block, this approach may be preferable to radiofrequency ablation of accessory pathways that are located close to the atrioventricular conduction system.[31] In a series of 20 patients with an anteroseptal or mid-septal accessory pathway who underwent cryothermal catheter ablation, a successful outcome was achieved without injury to the atrioventricular conduction system in all patients, although a second ablation procedure was required in 20% of patients.[32]

Target sites for ablation of accessory pathways are identified by early ventricular activation relative to the delta wave onset (for manifest accessory pathways), by the earliest atrial activation in the retrograde direction (for any accessory pathway that conducts retrogradely), and/or by the presence of a high frequency electrogram consistent with an accessory pathway potential.[33,34] Free-wall accessory pathways often have an oblique course, and the site of earliest anterograde ventricular activation may differ from the site of earliest retrograde atrial activation.[35]

Decremental anterograde conduction through a bypass tract occurs when there is a decrementally conducting atrioventricular accessory pathway[36] or an atriofascicular accessory pathway.[37] Target sites for ablation of the former are identified in the same way as for standard accessory pathways. Atriofascicular accessory pathways are most reliably localized by identification of an accessory pathway potential during sinus rhythm or antidromic tachycardia, usually along the lateral, posterolateral, or anterolateral tricuspid annulus.[37]

For most free-wall accessory pathways, complete bidirectional block can be achieved with a conventional 4–mm-tip ablation catheter, using a power setting of 50 W and a temperature setting of 60°C. If the conduction block is transient, permanent accessory pathway block may be easier to achieve with an 8–mm-tip ablation catheter or with an irrigated-tip ablation catheter.[38]

Between 5 and 17% of posteroseptal and left posterior accessory pathways have been reported to be epicardial and ablatable only within a branch of the coronary sinus (most commonly the middle cardiac vein), on the floor of the coronary sinus at the orifice of a venous branch, or within a coronary sinus diverticulum. These pathways may consist of connections between the muscle coat of the coronary sinus and the ventricle.[39] In the presence of a coronary sinus-ventricular accessory pathway, ventricular activation within a branch of the coronary sinus precedes endocardial activation, and coronary sinus muscle extension potentials usually are recorded in the venous branch, producing a pattern similar to an accessory pathway activation potential.[39]

A conventional ablation catheter may completely occlude a branch of the coronary sinus, preventing cooling of the ablation electrode and resulting in high impedance when radiofrequency energy is delivered. This markedly reduces the amount of power that can be delivered and may result in adherence of the ablation electrode to the wall of the vein. An externally saline-cooled ablation catheter allows more consistent delivery of radiofrequency energy with less heating at the electrode to tissue interface. However, before delivering radiofrequency energy near or within a branch of the coronary sinus, coronary
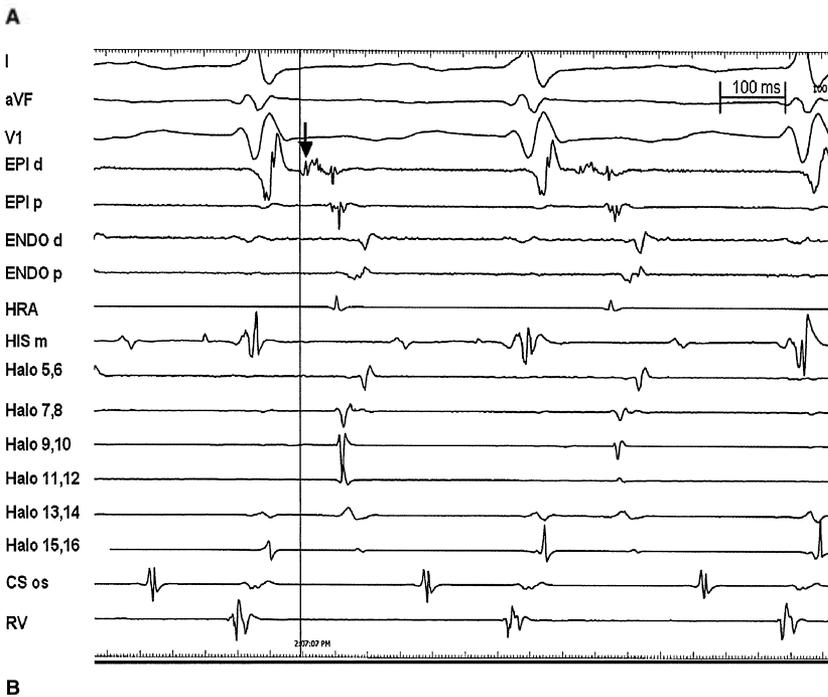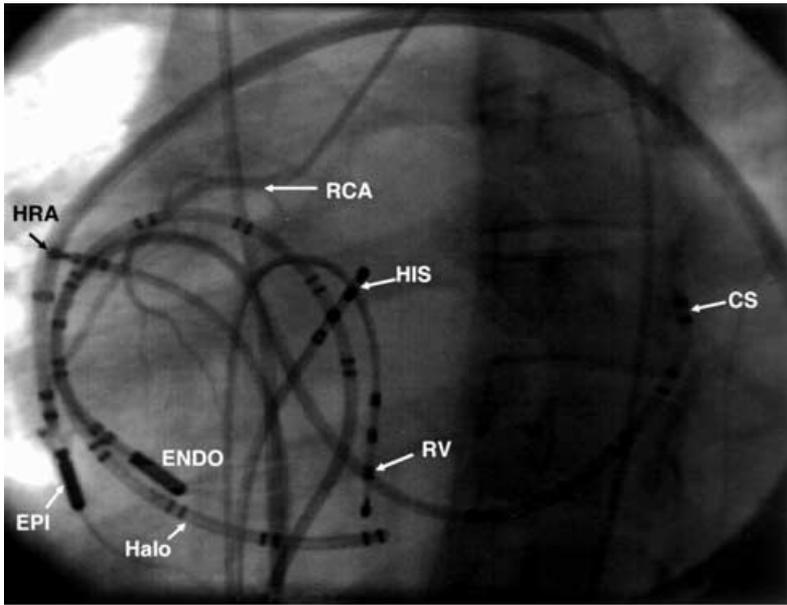
Figure 1. *A right-sided epicardial accessory pathway ablated using a transcutaneous pericardial approach. (A) A 45-degree left anterior oblique fluoroscopic view of the catheters in the heart (CS, halo, HIS, HRA, ENDO, RV) and pericardial space (EPI), and of the right coronary artery (RCA). The pericardial catheter (EPI) is at the posterolateral aspect of the tricuspid annulus. (B) Intracardiac electrograms recorded by the catheters shown in A during orthodromic reciprocating tachycardia. A high frequency potential compatible with an accessory pathway potential (arrow) was recordable only with the pericardial catheter positioned at the posterolateral aspect of the tricuspid annulus. Radiofrequency ablation at this pericardial site abolished accessory pathway conduction. The accessory pathway could not be ablated with endocardial applications of radiofrequency energy. (Figures courtesy of Dr. Kalyanam Shivkumar, University of California, Los Angeles, CA, USA.) CS = coronary sinus; HRA = high right atrial; ENDO = endovascular; RV = right ventricular.*

angiography should be performed to determine if there are any branches of the right or left coronary artery in proximity to the ablation site. If there is a branch of the right coronary artery within 2 mm of the ablation site, there may be a high risk of coronary artery injury if radiofrequency energy is delivered (Dr. Warren Jackman, personal communication, July, 2003). In this situation, it may be that cryoablation can be performed with little or no risk of coronary artery injury.[31,40]

A small percentage of left free-wall accessory pathways also may be epicardial, requiring ablation from within the coronary sinus.[41,42]

Other types of unusual accessory pathways that cannot be ablated with a standard endocardial approach at the annulus also have been described. These include accessory pathways that connect the right atrial appendage to the right ventricle, successfully ablated using a transcutaneous pericardial approach,[43] or by endocardial ablation over a large area,[44] and accessory pathways closely associated with the ligament of Marshall, ablated by targeting this ligament.[45] Rarely, a transcutaneous pericardial approach is required to ablate epicardial atrioventricular accessory pathways that are posteroseptal or right-sided (Fig. 1).

In early reports, the acute success rate of accessory pathway ablation ranged from 89 to 100%, with a recurrence rate of 3–9% and a long-term success rate of 85–100%.[28,33,46–50] The complication rate ranged between 2 and 3%, with the most common complications being cardiac tamponade and atrioventricular block.[28,33,46–50] In a trial of a temperature-controlled radiofrequency ablation catheter conducted at 18 institutions between 1992 and 1995, successful ablation was achieved in 93% of 500 patients with an accessory pathway, with the success rate being higher for left-sided (95%) than for posteroseptal (88%) or right-sided (90%) accessory pathways.[25] There was one death in that study (caused by dissection of the left coronary artery),[25] yielding a fatality rate of 0.2%. A survey of five university electrophysiological laboratories in 1998 indicated that a fatal complication had occurred in 0.08% of 3,856 patients who had undergone radiofrequency ablation of an accessory pathway.[51] This mortality rate compared favorably with the annual risk of sudden death in the Wolff-Parkinson-White syndrome, estimated at 0.05–0.5%.[52]

A more contemporary view of the risk to benefit ratio of accessory pathway ablation is available by pooling the results of ablation between 1997 and 2002 in the electrophysiological laboratories surveyed for this article. In a total of 6,065 patients, and with a repeat procedure necessary in 2.2%, a long-term success rate of 98% was achieved. A serious complication (cardiac tamponade, atrioventricular block, coronary artery injury, retroperitoneal hemorrhage, or stroke) occurred in 0.6% of patients, with one fatality (0.02%). Therefore, the one-time risk of catheter ablation is considerably lower than the cumulative annual risk associated with the Wolff-Parkinson-White syndrome, and it is clear that the treatment of choice for patients with the Wolff-Parkinson-White syndrome who may be at risk of life-threatening arrhythmias is catheter ablation. In addition, the highly favorable risk to benefit ratio justifies the use of catheter ablation as first line therapy for any patient with accessory pathway dependent tachycardia that requires treatment.

Atrial Tachycardia

Atrial tachycardias may be focal or macroreentrant. Focal atrial tachycardia may be caused by abnormal automaticity, triggered activity, or microreentry,[53,54] and is characterized by centrifugal spread of activation away from a discrete site of origin, without electrograms spanning all of diastole.[55] In contrast, in macroreentrant atrial tachycardia, electrograms are present throughout diastole. Macroreentrant atrial tachycardias, including incisional or scar related tachycardia, are much more closely related to nonisthmus dependent atrial flutter than to focal atrial tachycardia and, therefore, are included in the discussion of atrial flutter.

Focal atrial tachycardias most commonly arise in the right atrium along the crista terminalis, near the tricuspid annulus, or near the coronary sinus ostium.[56,57] Approximately 5% of focal atrial tachycardias arise in the left atrium, and these have been reported to occur most commonly along the superior or inferior aspect of the mitral annulus,[58,59] or near the ostia of the pulmonary veins (Fig. 2).[58] Recent experience gained during catheter ablation of atrial fibrillation has indicated that focal tachycardias also may arise in the pulmonary veins,[60] superior vena cava,[61,62] vein of Marshall,[63] or inferior vena cava.[64]

Target sites for ablation of focal atrial tachycardias are identified by activation mapping or pacemapping. It often is difficult to discern small differences in P wave morphology during pacemapping, and the activation sequence recorded by several multipole catheters is a useful surrogate for P wave morphology.[65] Mechanical interruption of an atrial tachycardia by the ablation catheter also has been demonstrated to be helpful in identifying an effective ablation site.[66]

Identification of the earliest site of atrial activation during a focal atrial tachycardia may be facilitated by the use of various tools like a basket catheter,[67–69] an electroanatomic mapping system (Fig. 2),[58,70] or a noncontact mapping system.[71]

Because focal atrial tachycardias are relatively uncommon, there are no large series in the literature describing the results of catheter ablation of focal atrial tachycardia. In seven studies that included a total of 112 patients with focal atrial tachycardia, the short-term success rate of radiofrequency ablation was approximately 90%, 7% of patients had a late recurrence, and no serious complications were reported.[54,65,66,72–75] However, these studies used neither the three-dimensional mapping systems nor the 8–mm-tip or cooled-tip ablation catheters that are widely available today. With these sophisticated mapping tools and with ablation catheters capable of creating larger lesions than conventional ablation catheters, clinical failures more often are related to the emergence of new atrial foci than to the inability to ablate a particular atrial tachycardia.

Given the high probability of successful ablation and the low risk of serious complications, it is appropriate to consider the option of catheter ablation for any patient with an atrial tachycardia that is clinically significant and requires therapy. However, pharmacologic therapy is more appropriate if multiple atrial foci are present. In these patients, even if all of the atrial tachycardia foci are mapped
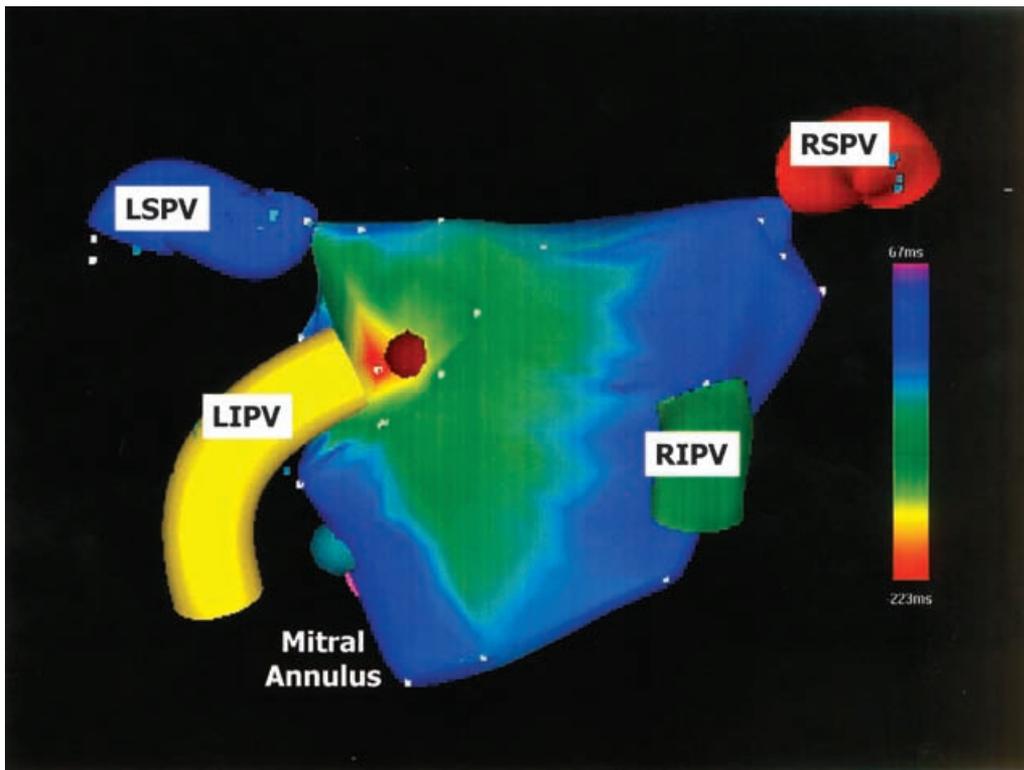
Figure 2. *Activation map of a focal atrial tachycardia created with a three-dimensional electroanatomic mapping system. A posterior view of the left atrium is shown. Note that there is centrifugal spread from a focal area of early activation (red). The site of origin was near the ostium of the left inferior pulmonary vein. The dark red tag indicates the successful ablation target site. RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein.*

and ablated, new foci may later emerge. In older patients with pleiomorphic atrial tachycardia who are highly symptomatic and drug refractory, atrioventricular junction ablation/pacemaker may be more appropriate than attempts to ablate the multiple foci.

Atrial Flutter

Isthmus Dependent Atrial Flutter

The most common type of atrial flutter is isthmus dependent atrial flutter in which reentry is confined to the right atrium with the wavefront progressing in a counter-clockwise or clockwise direction across the cavotricuspid isthmus. In the initial reports of radiofrequency catheter ablation of isthmus dependent atrial flutter, conventional ablation catheters were used and movement of the ablation catheter along the cavotricuspid isthmus was guided only by fluoroscopy. The endpoint of ablation in the early reports was termination of atrial flutter and/or the inability to induce the flutter by pacing. The acute success rates in these stud-

ies ranged from 77 to 100%, but recurrence rates usually were in the range of 20–40%.[50,75−77]

The recurrence rate of atrial flutter in the initial reports of radiofrequency ablation was high probably in large part because termination and noninducibility of atrial flutter are unreliable endpoints. Contributing to the more reliable outcome of catheter ablation of isthmus dependent atrial flutter in contemporary practice are more accurate endpoints for the procedure. Recognition of complete isthmus block has been enhanced by several techniques: a change in the atrial activation sequence along the lateral right atrium during coronary sinus pacing;[78,79] differential pacing;[80] double potentials separated by at least 90–110 ms along the entire ablation line;[81−83] measurement of transisthmus conduction intervals;[84] and a change in electrogram polarity in the region of the isthmus during coronary sinus pacing.[85] In addition, although conventional catheter techniques suffice, complete isthmus block can be confirmed with the use of three-dimensional mapping systems.[86,87]

In addition to more accurate endpoints for complete isthmus block, more precise localization and tracking of the ablation catheter along the cavotricuspid isthmus is now possible with a variety of nonfluoroscopic localization tools that facilitate the creation of ablation lines devoid of gaps.[86–92] Also available is phased-array intracardiac echocardiography,[93] which allows visualization of the pouches and recesses that commonly are present along the cavotricuspid isthmus and that sometimes make a complete line of block in the isthmus difficult to create.[93–95]

Advances in catheter design also have enhanced the ability to create complete lines of block in the cavotricuspid isthmus. Because of the pouches, recesses, ridges, and trabeculations that may occur in the isthmus,[94] it often is advantageous to create lesions that are larger than those created with conventional 4–mm-tip ablation catheters. Radiofrequency ablation catheters that have an 8-mm distal electrode or a cooled- or irrigated-tip electrode allow the creation of larger lesions in high and low flow regions. Several studies have demonstrated that complete isthmus block is more reliably achieved with an 8–mm-tip catheter[96–100] and with a cooled- or irrigated-tip catheter[99,101–104] than with a conventional ablation catheter. In a randomized study that compared the 8–mm-tip and cooled-tip catheters, complete isthmus block was achieved in 99% of 100 patients, with the efficacy of the two catheters being equally high.[105]

Complete isthmus block also can be achieved by cryothermal ablation.[106] Although there is no reason to believe that cryothermal ablation will be more effective than radiofrequency ablation of the cavotricuspid isthmus, cryothermal ablation has the advantage of being less painful.[106]

The cavotricuspid isthmus can be ablated using a purely anatomic approach[107–112] or an electrogram mapping approach that targets single potentials or narrowly split double potentials during atrial flutter or coronary sinus pacing.[81,113] In a randomized comparison, the electrogram mapping approach was found to be more efficient than the anatomic approach if the first ablation line did not result in complete isthmus block.[114]

With the availability of precise catheter locator systems, more effective ablation tools, and accurate endpoints for ablation, the inability to successfully create complete isthmus block and to eliminate recurrences of isthmus dependent flutter is unusual in contemporary practice. This is reflected in the pooled ablation results of the electrophysiological laboratories surveyed for this article. In a total of 7,071 patients with isthmus dependent atrial flutter who underwent radiofrequency catheter ablation, a repeat ablation procedure was performed in 4% of patients and the long-term success rate in preventing recurrent atrial flutter was 97%. The overall incidence of serious complications was 0.4%, with the most common complication consisting of atrioventricular block in 0.2% of patients. Other rare complications that have been reported include ventricular tachycardia[115] and acute occlusion of the right coronary artery.[116]

In light of the high efficacy and safety of radiofrequency ablation for isthmus dependent atrial flutter, catheter ablation is an appropriate therapeutic option for drug refractory patients, and also for patients who prefer curative therapy to pharmacologic therapy. However, a large proportion of patients with atrial flutter also previously had or will develop atrial fibrillation.[117–120] If treatment with a class I or III antiarrhythmic drug suppresses atrial fibrillation but not atrial flutter, a hybrid approach that combines catheter ablation of the atrial flutter with continued antiarrhythmic drug therapy often is effective in preventing recurrent atrial fibrillation.[121–123] However, in patients who have both atrial flutter and atrial fibrillation prior to ablation, the atrial fibrillation usually does not resolve after atrial flutter ablation.[117–120] Therefore, additional therapy for atrial fibrillation, pharmacologic or catheter ablation, may still be necessary after atrial flutter is eliminated.

Other Atrial Flutters

Atrial flutters, other than isthmus dependent atrial flutter, consist of right atrial flutters that are not isthmus dependent, left atrial flutters, and incisional atrial flutters. Because the pathophysiology and treatment of nonisthmus dependent atrial flutter and macroreentrant atrial tachycardia are identical, they are discussed together.

Right atrial flutter that does not involve the cavotricuspid isthmus is unusual, particularly in patients without a history of open heart surgery. In a series of 160 patients with right atrial flutter, only 4% were mapped to the right atrial free wall and did not use the cavotricuspid isthmus.[124] These atrial flutters were successfully eliminated by creation of an ablation line from a mid-lateral line of conduction block to the inferior vena cava.[124]

Macroreentrant left atrial tachycardia and flutter also are unusual in patients without prior surgery or left atrial catheter ablation. In the largest series of left atrial flutters, detailed mapping in 17 patients, most of whom had structural heart disease but no history of heart surgery, demonstrated that there most commonly was macroreentry around the mitral annulus, pulmonary veins, and an electrically silent area.[125] Successful ablation was achieved with ablation lines between the mitral annulus and a pulmonary vein or an
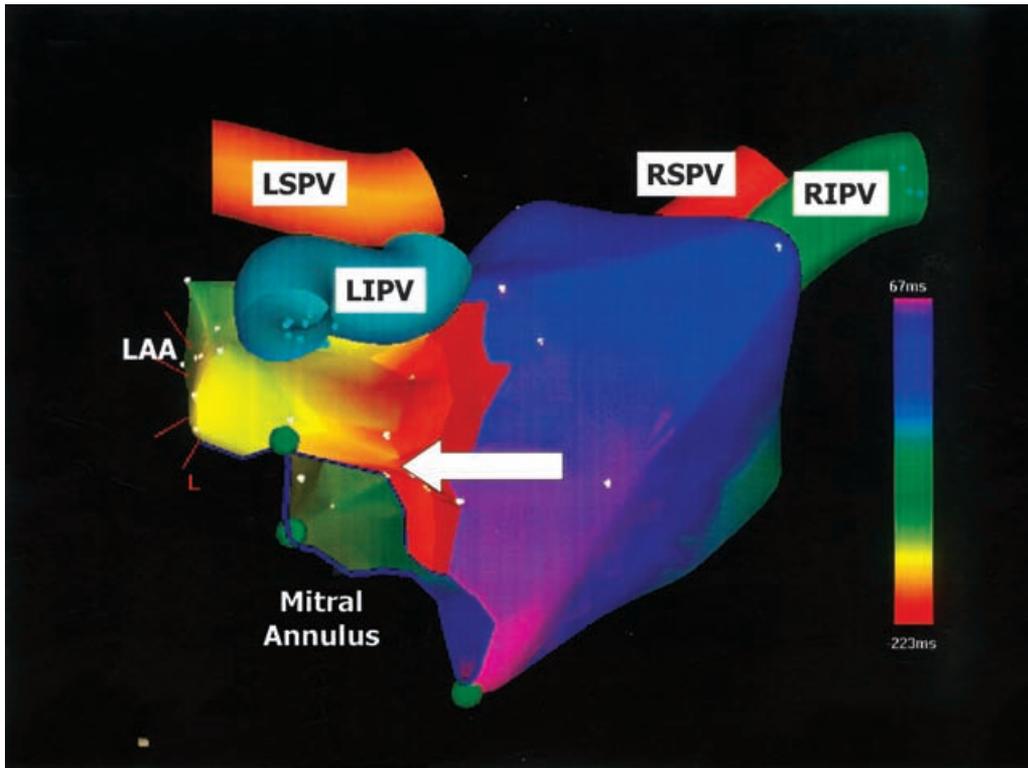
Figure 3. *Activation map of a left atrial flutter created with a three-dimensional electroanatomic mapping system. A left posterior oblique view of the left atrium is shown. Note that the sites of earliest (red) and latest (purple) atrial activation are adjacent to each other, in the isthmus between the mitral annulus and the left inferior pulmonary vein. The white arrow indicates the direction of the wavefront across the mitral isthmus. An ablation line created in this isthmus successfully ablated the atrial flutter. LAA = left atrial appendage; other abbreviations as in Figure 2.*

electrically silent area (Fig. 3).[125] Left atrial macroreentry also has been reported to involve the muscular coat of the coronary sinus and to be treatable by circumferential radiofrequency ablation within the coronary sinus.[126]

Atypical right atrial flutter that occurs as a consequence of a surgical scar or suture line has been referred to as "incisional" flutter or tachycardia. Entrainment mapping and the postpacing interval are helpful in identifying critical components of the reentrant circuit.[127] These macroreentrant circuits usually can be abolished by transecting a critical isthmus with an ablation line extending from an area of scar to a naturally occurring anatomic barrier. The most common locations of the ablation lines used to treat incisional flutter/tachycardia are between a right lateral atriotomy scar and the inferior vena cava, superior vena cava, or tricuspid annulus.[127,128] Success rates of 80–85% were reported in early studies that did not use three-dimensional mapping systems.[127,128] A later study used a three-dimensional electroanatomic mapping system to

demonstrate that macroreentrant right atrial tachycardias after surgery for congenital heart disease often use relatively narrow channels between scars.[129] Detailed voltage mapping allowed identification of the channels and elimination of the tachycardias, often with only a single application of radiofrequency energy.[129]

In another study, the cavotricuspid isthmus was found to be part of the reentry circuit in approximately 70% of patients with postoperative intraatrial reentrant tachycardia.[130] Therefore, it is worthwhile to first evaluate the role of the cavotricuspid isthmus in an incisional flutter before embarking on detailed activation or voltage mapping of the right atrium.

Patients who have undergone cardiac transplantation with an atrial anastomosis may develop an atrial tachycardia attributable to atrioatrial conduction across the suture line separating the donor and native right atria.[131] In addition to primary ablation of the arrhythmia in the native atrial remnant, these arrhythmias also may be eliminated by ablation of atrioatrial conduction.[131]

Left atrial flutter may be a proarrhythmic complication of surgery or left atrial catheter ablation for atrial fibrillation. Many of these left atrial flutters use the isthmus between the mitral annulus and the left inferior pulmonary vein and can be eliminated by creating a line of block in this isthmus.[132]

With the current availability of three-dimensional mapping systems, it is reasonable to expect a success rate of at least 90% when attempting to ablate nonisthmus dependent atrial flutters. Therefore, catheter ablation is a reasonable option for drug refractory nonisthmus dependent atrial flutter or when it is the sole arrhythmia in a patient who prefers to avoid antiarrhythmic drug therapy. If the nonisthmus dependent atrial flutter occurs following a surgical or atrial-based catheter procedure for atrial fibrillation, it is appropriate to treat the flutter pharmacologically before proceeding with catheter ablation, because these flutters may spontaneously resolve within 3–4 months after the surgical or catheter procedure.

Atrial Fibrillation

In the past several years, the most exciting developments in catheter ablation of supraventricular arrhythmias have occurred in the field of atrial fibrillation. The proof-of-concept for catheter ablation of atrial fibrillation was provided by Swartz et al.,[133] who demonstrated that linear ablation lines made by contiguous applications of radiofrequency energy with a conventional ablation catheter in the left and right atria could eliminate atrial fibrillation. However, because of the technically challenging nature of the procedure and a high complication rate, this type of ablation procedure was not adapted into clinical practice. Simpler and safer linear ablation procedures were attempted, generally with poor results.[134−138]

Role of Pulmonary Veins and Pulmonary Vein Isolation

A landmark study by Haissaguerre et al.[60] in 1998 focused attention on the importance of the pulmonary veins in the generation of atrial fibrillation. It was found that the premature depolarizations that trigger atrial fibrillation often arise in the muscle sleeves that surround the pulmonary veins. This seminal observation led to the technique of focal ablation within the pulmonary veins to eliminate the triggers of atrial fibrillation.[139−141] However, because of the multifocal nature of the triggers, the inability to identify all triggers in the course of a catheter ablation procedure, and the later emergence of new triggers, focal ablation within the pulmonary veins was associated with a high recurrence rate.[139] Another problem with the procedure was that it ne-

cessitated ablation within the pulmonary veins, creating pulmonary vein stenosis in 4–42% of patients.[140,141]

The next major step in the evolution of catheter ablation of atrial fibrillation was the demonstration that the pulmonary veins could be electrically isolated from the left atrium by ostial applications of radiofrequency energy guided by pulmonary vein potentials.[142] Pulmonary veins can be electrically isolated by circumferential ablation at the ostium,[143] but when ostial ablation is guided by pulmonary vein potentials, complete isolation often is achieved with ablation at < 60% of the ostial circumference.[144] This is because the muscle fibers that conduct impulses between the muscle sleeve and the atrium are not evenly distributed throughout the circumference of the ostium. Electrical isolation of a pulmonary vein by segmental ostial ablation eliminates the need for identification and mapping of individual triggers and serves as a safeguard against the future emergence of new triggers from a vein. It also reduces the risk of pulmonary vein stenosis by limiting the amount of ostial ablation needed to isolate a vein.

Pulmonary vein potentials are best recorded with a catheter that has 10–20 electrodes in a distal ring configuration, or with a multitined basket catheter. Appropriate target sites for ablation are identified based on the earliest bipolar electrogram, or on the unipolar electrogram that has the steepest and largest intrinsic deflection.[145,146] A randomized study showed that complete isolation of the pulmonary veins is achieved more quickly and efficiently when guided by unipolar electrograms than when guided by bipolar electrograms (Fig. 4).[147]

Ostial pulmonary vein potentials may be buried within atrial electrograms recorded at the ostia of the pulmonary veins, and this may hinder the identification of appropriate target sites for ablation. The pulmonary vein potentials usually can be separated from the atrial electrograms by pacing within the coronary sinus or left atrial appendage (in the case of the left-sided pulmonary veins),[148,149] or by the introduction of an atrial extrastimulus during atrial pacing (Fig. 5).[150]

The left atrial appendage often lies in close proximity to the left-sided pulmonary veins, and a potential that originates in the appendage may mimic a pulmonary vein potential. Pulmonary vein potentials can be distinguished from left atrial appendage potentials by pacing within the left atrial appendage.[148,149]

The endpoint of segmental ostial ablation is the elimination of all pulmonary vein potentials with complete conduction block in and out of the pulmonary vein (Fig. 6).
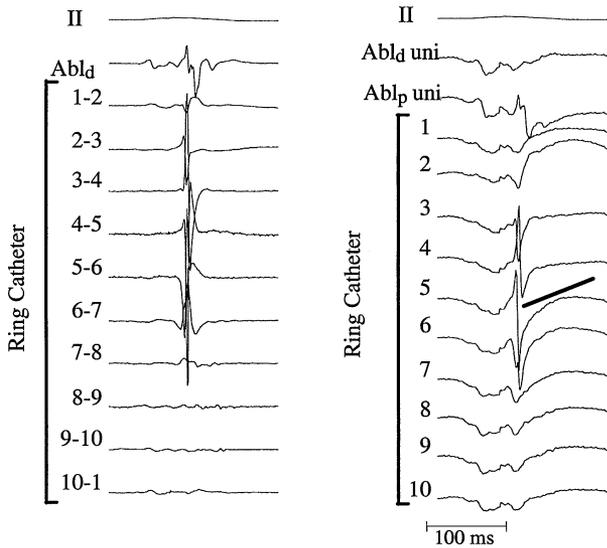
Figure 4. *Bipolar (left panel) and unipolar (right panel) electrograms of pulmonary vein potentials recorded at the ostium of the right superior pulmonary vein with a ring catheter. Shown are lead II, bipolar and unipolar (uni) electrograms recorded with the ablation catheter (Abl), and multiple electrograms recorded with a ring catheter positioned at the ostium of the vein. In the bipolar recordings, the most appropriate ablation site is unclear. In the unipolar recordings, the most appropriate target site is easily identified to be at electrode 5 of the ring catheter, where the largest, steepest, and earliest intrinsic deflection is recorded (arrow). d = distal; p = proximal.*

In initial studies, when an arrhythmogenic pulmonary vein was isolated, the short-term success rate in eliminating paroxysmal atrial fibrillation ranged from 55 to 93%, depending on the number of arrhythmogenic pulmonary vein foci that were present.[80] However, as many as 54% of patients needed a repeat procedure because of residual triggers arising in other pulmonary veins or locations.[80]

In addition to being a major source of premature depolarizations that trigger atrial fibrillation, the pulmonary veins may also be important in maintaining an episode of paroxysmal atrial fibrillation. Intermittent bursts of rapid electrical activity in the pulmonary veins may serve as at least one of the drivers of atrial fibrillation.[151,152] These bursts of pulmonary vein tachycardia have a high prevalence in the left superior, left inferior, and right superior pulmonary veins during episodes of paroxysmal atrial fibrillation, and this led to the approach of systematically isolating at least three pulmonary veins during the first ablation procedure, whether or not triggers were found to be arising in each vein.[144,152] With this approach, symptomatic paroxysmal atrial fibrillation was eliminated, without the need for antiarrhythmic medications, in 70% of patients, and the percentage of patients requiring a repeat procedure dropped to 9%.[144]

Pulmonary vein isolation has been performed with a conventional 4–mm-tip catheter, an 8–mm-tip catheter, or a saline-cooled or irrigated-tip catheter.[153,154] In a study that compared the three types of catheters, the best results were obtained with the 8–mm-tip catheter.[154] However, in other studies, pulmonary vein isolation has been achieved reliably with a conventional 4–mm-tip catheter.[144,152,155] It is possible that the deeper lesions created with an 8–mm-tip catheter may increase the risk of pulmonary vein stenosis, and the type of ablation catheter that is associated with the most favorable risk to benefit ratio is unclear. When

Figure 5. *The use of an atrial extrastimulus to separate atrial and pulmonary vein potentials recorded with a ring catheter at the ostium of a left superior pulmonary vein. During coronary sinus (CS) pacing at a cycle length of 450 ms, the atrial and pulmonary vein potentials are fused (asterisk). A premature atrial extrastimulus (S2) results in separation of pulmonary vein potentials (arrows) from the atrial potentials. Abl = ablation catheter; d = distal.*
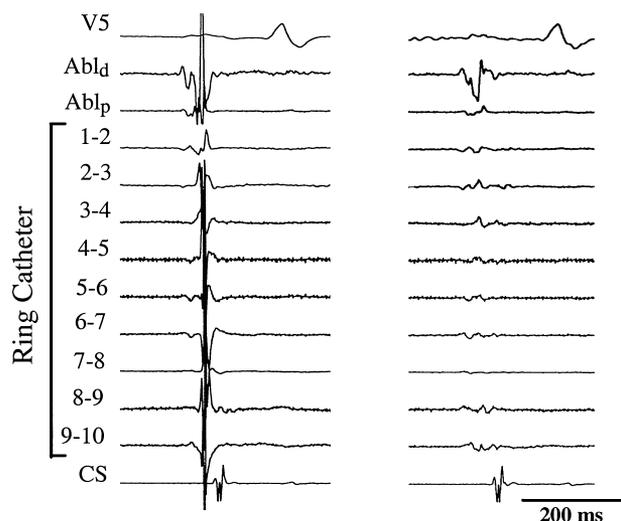
Figure 6. *Endpoint of segmental ostial ablation. The ring catheter was positioned near the ostium of a left superior pulmonary vein, and multiple bipolar electrograms are shown before (left panel) and after (right panel) several ostial applications of radiofrequency energy. Note that all pulmonary vein potentials were eliminated. Abbreviations as in Figures 4 and 5.*

a cooled-tip or 8–mm-tip catheter is used, monitoring of microbubble formation by intracardiac echocardiography during applications of radiofrequency energy may reduce the risk of pulmonary vein stenosis and improve efficacy.[156]

The triggers or driving mechanisms for atrial fibrillation may arise in other thoracic veins, including the vein of Marshall, superior vena cava, and coronary sinus, or in the left or right atrium.[60,61,139,157−161] The ectopy that initiates atrial fibrillation has been demonstrated to originate outside of the pulmonary veins in as many as 28–47% of patients with paroxysmal atrial fibrillation.[162,163] Therefore, if an ablation strategy for paroxysmal atrial fibrillation is limited to pulmonary vein isolation, it may be unrealistic to expect a long-term clinical success rate greater than approximately 70%, regardless of how reliably the pulmonary veins are isolated.

The pulmonary veins appear to play a much less important role in the generation of chronic atrial fibrillation than paroxysmal atrial fibrillation. This explains why pulmonary vein isolation by itself eliminates chronic atrial fibrillation in only 25% of patients.[164] Another problem with pulmonary vein isolation is that it is associated with a 3–5% risk of moderate to severe pulmonary vein stenosis.[165−167] Because the ostium of a pulmonary vein may have a funnel configuration and may not be clearly definable, radiofrequency en-

ergy may inadvertently be delivered within the proximal portion of a pulmonary vein. Therefore, although it was an important step in the evolution of ablation techniques for atrial fibrillation, pulmonary vein isolation by itself has significant shortcomings.

Alternative Ablation Strategies

The limitations of pulmonary vein isolation have been addressed in several different ways. One approach has been to add an ablation line in the left atrium to segmental ostial ablation of the pulmonary veins. For example, the addition of an ablation line in the mitral isthmus, between the left inferior pulmonary vein and the mitral annulus, improved the success rate of pulmonary vein isolation for paroxysmal atrial fibrillation to 82% at a mean of a 6-month follow-up.[168] Furthermore, with the addition of one more ablation lines across the left atrial roof, chronic atrial fibrillation was successfully eliminated in 60% of patients at a mean follow-up of 7 months.[169] A second approach has been to first isolate the pulmonary veins, then to search for and ablate triggers arising in other sites.[163,170] In one study, this technique resulted in freedom from atrial fibrillation at 1-year follow-up in 80% of 75 patients, 88% of whom had paroxysmal atrial fibrillation.[170] However, in another study that used a strategy of targeting all ectopic foci within and outside of the pulmonary veins, the success rate at the 22-month follow-up was a more modest 63%. A third approach has been to extend the targets for ablation from the ostia to the atrial tissue surrounding the pulmonary veins. With this approach, approximately 80–90% of patients were reported to have no further symptomatic atrial fibrillation at the 14-month follow-up.[156]

**Left Atrial Ablation to Encircle
the Pulmonary Veins**

Another alternative to segmental ostial ablation has been to limit ablation to the left atrium. This technique involves the creation of circumferential ablation lines around the left- and right-sided pulmonary veins, 1–2 cm from the ostia of the veins, guided by a three-dimensional electroanatomic mapping system (Fig. 7).[171,172] Ablation lines in the posterior left atrium and mitral isthmus often are also created (Fig. 7). The technique of circumferential left atrial ablation was first described by Pappone et al.[171] In a group of 589 patients with paroxysmal or chronic atrial fibrillation, approximately 85% were free from atrial fibrillation at a mean of 900 days after circumferential left atrial ablation.[173] The most common serious complication of circumferential left atrial ablation has been pericardial tamponade, which
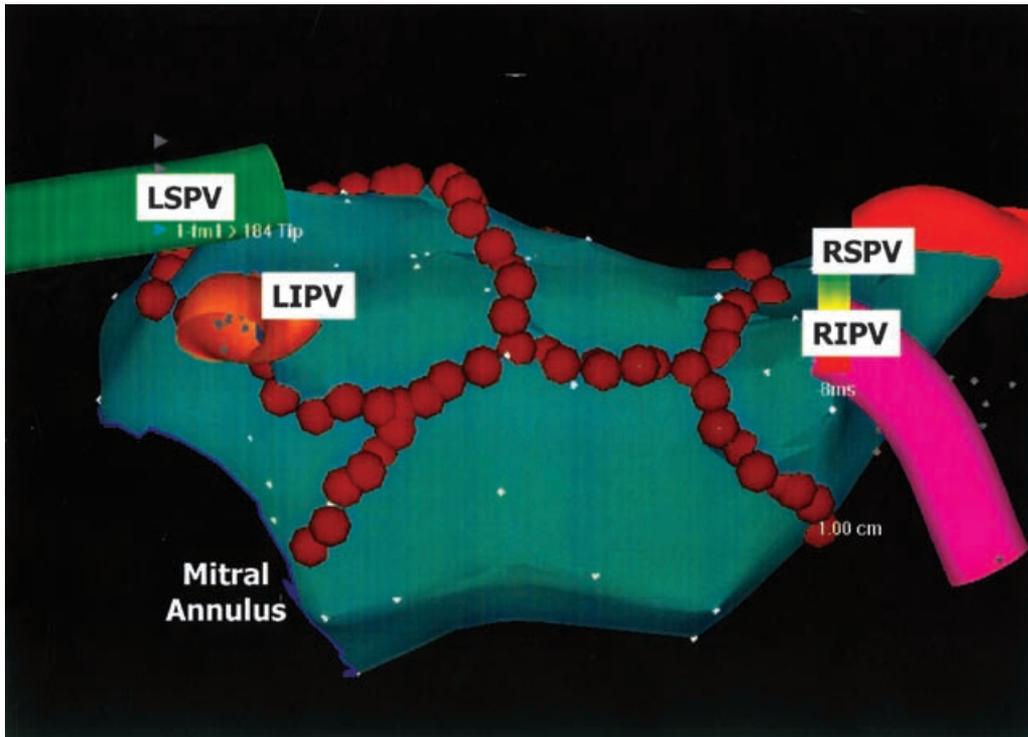
Figure 7. *A circumferential left atrial ablation strategy. Shown is the posterior aspect of the left atrial shell created with an electroanatomic mapping system. The dark red tags indicate ablation sites. The left- and right-sided pulmonary veins were encircled. Additional ablation lines were created in the posterior left atrium and across the isthmus between the mitral annulus and the inferior portion of the circumferential ablation line surrounding the left-sided pulmonary veins. Abbreviations as in Figure 2.*

has occurred in 1% of patients. Proarrhythmia in the form of left atrial flutter may occur within a few days of left atrial ablation in 5–10% of patients, (unpublished data) and these flutters may require ablation if they do not resolve spontaneously within 3–4 months after the procedure.

Segmental ostial ablation to isolate the pulmonary veins was compared to the circumferential left atrial ablation strategy (Fig. 7) in a randomized study of 80 patients with paroxysmal atrial fibrillation.[174] The endpoint of the study was freedom from symptomatic atrial fibrillation in the absence of drug therapy after a single ablation procedure. The circumferential left atrial ablation strategy was found to be significantly more effective than segmental ostial ablation, with success rates at 6 months being 87% and 67%, respectively. The average procedure duration with both approaches was approximately 2.5 hours.

It is logical that an extensive ablation procedure that has several possible mechanisms of action would be more effective than an ablation strategy that is limited to segmental ostial ablation. It is likely that segmental ostial ablation eliminates

atrial fibrillation only by eliminating the triggers and drivers of atrial fibrillation that arise in the pulmonary veins. The circumferential left atrial ablation strategy, including ablation lines in the posterior left atrium and mitral isthmus, has several possible mechanisms of action: (1) pulmonary vein isolation, at least to some degree; (2) elimination of anchor points for "mother waves," or rotors that may generate atrial fibrillation, at or near the left atrial pulmonary vein junction;[175–177] (3) ablation of other potential trigger sites, like the vein of Marshall and the posterior left atrial wall;[163] (4) ablation of right-left atrial connections that may play a role in generating atrial fibrillation; (5) atrial debulking, to provide less space for circulating wavelets; and (6) atrial dennervation. Some or all of these factors may explain why the circumferential left atrial ablation strategy is more effective than segmental ostial ablation for both paroxysmal and chronic atrial fibrillation.

Because atrial fibrillation is an independent predictor of mortality, it is conceivable that the elimination of atrial fibrillation may improve survival. The Atrial Fibrillation Follow-Up

Investigation of Rhythm Management (AFFIRM) trial demonstrated that a rhythm control strategy conferred no survival advantage over rate control in patients with atrial fibrillation who had risk factors for stroke.[178] However, drug inefficacy and/or adverse drug effects easily could account for the absence of a survival benefit of rhythm control. In a recent study of 1,171 patients with symptomatic atrial fibrillation that were treated with medical therapy or left atrial ablation, left atrial ablation was associated with a 55% reduction in major morbid events, like heart failure and stroke, and a 50% reduction in mortality. Because treatment allocation was not randomized, the results of this study cannot be considered definitive. Nevertheless, the study does suggest that the elimination of atrial fibrillation may improve not only quality-of-life, but also survival.

Current Role of Catheter Ablation of Atrial Fibrillation

Catheter ablation techniques for atrial fibrillation are still in an evolutionary phase, and the long-term risk to benefit ratio is as yet unsettled. At this time, it seems appropriate to limit catheter ablation of atrial fibrillation to symptomatic patients whose quality-of-life has been impaired and who cannot be successfully managed with pharmacologic therapy. As the experience with catheter ablation of atrial fibrillation grows over the next few years, it is likely that the selection criteria also will expand.

Future Directions

After more than a decade of experience with radiofrequency catheter ablation of atrioventricular nodal reentrant tachycardia, accessory pathways, monomorphic atrial tachycardias, and atrial flutter, it seems safe to say that the vast majority of patients with these types of supraventricular tachycardia can be cured with little risk of a serious complication. With success rates often in the upper half of the 90–100% range, one might question if there is any need for further improvements in the catheter techniques used to treat these arrhythmias. However, until success rates reach 100%,
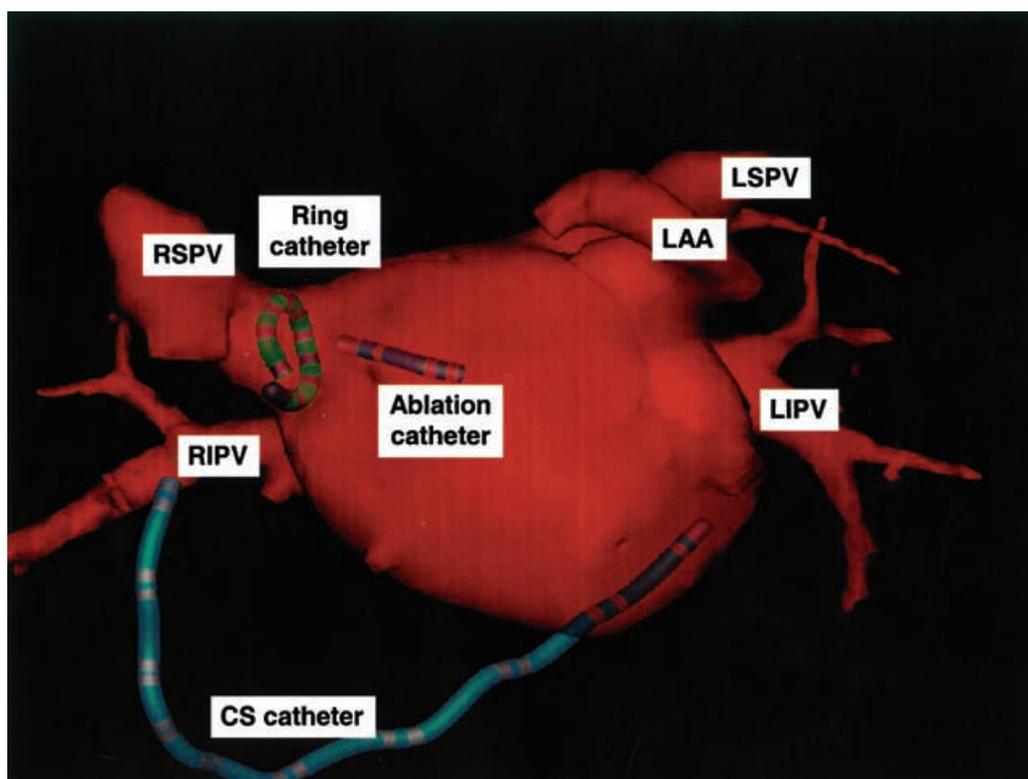

Figure 8. *An example of "digital fusion" of an MRI of the heart into a three-dimensional catheter localization system (NavX). Shown is an anterior-posterior view of the left atrium, with a ring catheter in the right superior pulmonary vein, the distal portion of an ablation catheter in the left atrium, and a 20-pole catheter in the coronary sinus. (Figure courtesy of Dr. Jasbir Sra, Milwaukee Heart Institute. NavX DIF currently under investigation.) CS = coronary sinus; MRI = magnetic resonance image; other abbreviations as in Figures 2 and 3.*

there will continue to be room for improvement in ablation tools and refinement in ablation techniques. For example, cryoablation may prove to be a useful alternative to radiofrequency ablation for epicardial accessory pathways that lie close to a coronary artery, or for mid- or anteroseptal accessory pathways that lie close to the atrioventricular conduction system. Whether other energy sources like laser or ultrasound will also find a niche in the electrophysiological laboratory is less clear, but possible.

High intensity focused ultrasound (HIFU) provides the potential for ablating specific targets in the myocardium or atrioventricular conduction system from outside of the heart and potentially from outside of the thorax.[179] There are several technical problems that need to be overcome, but it will be interesting to see how the application of HIFU to ablation of arrhythmias develops over the next few years.

In the past few years, fluoroscopy techniques that limit radiation exposure have become commonplace,[180] and this has been beneficial to patients and electrophysiologists. Furthermore, the widespread availability of three-dimensional mapping tools has reduced our dependence on fluoroscopy and contributed to a reduction in radiation exposure, while at the same time improving the ability to map and ablate complex arrhythmias. Future developments in the area of imaging, like real-time magnetic resonance imaging and "digital fusion" (the integration of a magnetic resonance image or computed tomography) of the heart into a three-dimensional mapping system to provide an anatomically accurate image of a chamber (Fig. 8) are likely to further reduce the need for radiation exposure to patients and operators.

A new approach to catheter ablation that currently is under development is the remote control of an ablation catheter using a magnetic guidance system in which a magnetic field is used for precise catheter navigation to an ablation target.[181] The concept is intriguing and initial studies have demonstrated that the remote control of an ablation catheter is feasible.[182] The degree of incremental value of this new technology in clinical practice should become clear in the near future.

In regards to atrial fibrillation, by far the most complex and challenging supraventricular arrhythmia faced by interventional electrophysiologists, improvements in catheter ablation techniques will depend to a great degree on a better understanding of the mechanisms of this arrhythmia. The goal should be to learn how to eliminate atrial fibrillation with the minimum amount of ablation. But because there are several possible mechanisms of atrial fibrillation, it is likely that there will be no minimum left atrial lesion set that will be effective in all patients with atrial fibrillation. Furthermore, although atrial fibrillation is curable in most patients by ablation in the left atrium, the atrial fibrillation at times may be generated in the right atrium and require ablation of gaps in the crista terminalis.[183] The challenge will be to learn how to recognize the various subtypes of atrial fibrillation and to tailor the ablation therapy accordingly. The development of ablation catheters that facilitate the creation of lines of block in the atria also may improve the ability to cure atrial fibrillation.[184]

References

1. Scheinman MM, Morady F, Hess DS, et al. Catheter-induced ablation of the atrioventricular junction to control refractory supraventricular arrhythmias. JAMA 1982; 248:851–855.

2. Lee MA, Morady F, Kadish A, et al. Catheter modification of the atrioventricular junction with radiofrequency energy for control of atrioventricular nodal reentry tachycardia. Circulation 1991; 83:827–835.

3. Jazayeri MR, Hempe SL, Sra JS, et al. Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. Circulation 1992; 85:1318–1328.

4. Langberg JJ, Leon A, Borganelli M, et al. A randomized, prospective comparison of anterior and posterior approaches to radiofrequency catheter ablation of atrioventricular nodal reentry tachycardia. Circulation 1993; 87:1551–1556.

5. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry, by radiofrequency catheter ablation of slow-pathway conduction. N Engl J Med 1992; 327:313–318.

6. Haissaguerre M, Gaita F, Fischer B, et al. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. Circulation 1992; 85:2162–2175.

7. Kay GN, Epstein AE, Dailey SM, et al. Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia. Evidence for involvement of perinodal myocardium within the reentrant circuit. Circulation 1992; 85:1675–1688.

8. Kalbfleisch SJ, Strickberger SA, Williamson B, et al. Randomized comparison of anatomic and electrogram mapping approaches to ablation of the slow pathway of atrioventricular node reentrant tachycardia. J Am Coll Cardiol 1994; 23:716–723.

9. Niebauer MJ, Daoud E, Williamson B, et al. Atrial electrogram characteristics in patients with and without

atrioventricular nodal reentrant tachycardia. Circulation 1995; 92:77–81.

10. Jentzer JH, Goyal R, Williamson BD, et al. Analysis of junctional ectopy during radiofrequency ablation of the slow pathway in patients with atrioventricular nodal reentrant tachycardia. Circulation 1994; 90:2820–2826.

11. Altemose GT, Scott LR, Miller JM. Atrioventricular nodal reentrant tachycardia requiring ablation on the mitral annulus. J Cardiovasc Electrophysiol 2000; 11:1281–1284.

12. Hummel JD, Strickberger SA, Williamson BD, et al. Effect of residual slow pathway function on the time course of recurrences of atrioventricular nodal reentrant tachycardia after radiofrequency ablation of the slow pathway. Am J Cardiol 1995; 75:628–630.

13. Sra JS, Jazayeri MR, Blanck Z, et al. Slow pathway ablation in patients with atrioventricular node reentrant tachycardia and a prolonged PR interval. J Am Coll Cardiol 1994; 24:1064–1068.

14. Strickberger SA, Daoud E, Niebauer M, et al. Effects of partial and complete ablation of the slow pathway on fast pathway properties in patients with atrioventricular nodal reentrant tachycardia. J Cardiovasc Electrophysiol 1994; 5:645–649.

15. Skanes AC, Dubuc M, Klein GJ, et al. Cryothermal ablation of the slow pathway for the elimination of atrioventricular nodal reentrant tachycardia. Circulation 2000; 102:2856–2860.

16. Rodriguez LM, Geller JC, Tse HF, et al. Acute results of transvenous cryoablation of supraventricular tachycardia (atrial fibrillation, atrial flutter, Wolff-Parkinson-White syndrome, atrioventricular nodal reentry tachycardia). J Cardiovasc Electrophysiol 2002; 13:1082–1089.

17. Morady F, Scheinman MM. Transvenous catheter ablation of a posteroseptal accessory pathway in a patient with the Wolff-Parkinson-White syndrome. N Engl J Med 1984; 310:705–707.

18. Morady F, Scheinman MM, Kou WH, et al. Long-term results of catheter ablation of a posteroseptal accessory atrioventricular connection in 48 patients. Circulation 1989; 79:1160–1170.

19. Bardy GH, Ivey TD, Coltorti F, et al. Developments, complications and limitations of catheter-mediated electrical ablation of posterior accessory atrioventricular pathways. Am J Cardiol 1988; 61:309–316.

20. Fisher JD, Brodman R, Kim SG, et al. Attempted nonsurgical electrical ablation of accessory pathways via the coronary sinus in the Wolff-Parkinson-White syndrome. J Am Coll Cardiol 1984; 4:685–694.

21. Ward DE, Rowland E, Camm J. Transvenous ablation of anomalous conduction. J Electrophysiol 1987; 1:197–203.

22. Ruder MA, Mead RH, Gaudiani V, et al. Transvenous catheter ablation of extranodal accessory pathways. J Am Coll Cardiol 1988; 11:1245–1253.

23. Warin JF, Haissaguerre M, Lemetayer P, et al. Catheter ablation of accessory pathways with a direct approach. Results in 35 patients. Circulation 1988; 78:800–815.

24. Lesh MD, Van Hare GF, Scheinman MM, et al. Comparison of the retrograde and transseptal methods for ablation of left free wall accessory pathways. J Am Coll Cardiol 1993; 22:542–549.

25. Calkins H, Yong P, Miller JM, et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: Final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. Circulation 1999; 99:262–270.

26. Lesh MD, Coggins DL, Ports TA. Coronary air embolism complicating transseptal radiofrequency ablation of left free-wall accessory pathways. PACE 1992; 15:1105–1108.

27. Natale A, Wathen M, Yee R, et al. Atrial and ventricular approaches for radiofrequency catheter ablation of left-sided accessory pathways. Am J Cardiol 1992; 70:114–116.

28. Deshpande SS, Bremner S, Sra JS, et al. Ablation of left free-wall accessory pathways using radiofrequency energy at the atrial insertion site: Transseptal versus transaortic approach. J Cardiovasc Electrophysiol 1994; 5:219–231.

29. Chiang CE, Chen SA, Tai CT, et al. Prediction of successful ablation site of concealed posteroseptal accessory pathways by a novel algorithm using baseline electrophysiological parameters: Implication for an abbreviated ablation procedure. Circulation 1996; 93:982–991.

30. Morady F, Strickberger A, Man KC, et al. Reasons for prolonged or failed attempts at radiofrequency catheter ablation of accessory pathways. J Am Coll Cardiol 1996; 27:683–689.

31. Skanes AC, Yee R, Krahn AD, et al. Cryoablation of atrial arrhythmias. Card Electrophysiol Rev 2002; 6:383–388.

32. Gaita F, Riccardi R, Hocini M, et al. Safety and efficacy of cryoablation of accessory pathways adjacent to the normal conduction system. J Cardiovasc Electrophysiol 2003; 14:825–829.

33. Jackman WM, Wang XZ, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991; 324:1605–1611.

34. Calkins H, Kim YN, Schmaltz S, et al. Electrogram criteria for identification of appropriate target sites for radiofrequency catheter ablation of accessory atrioventricular connections. Circulation 1992; 85:565–573.

35. Otomo K, Gonzalez MD, Beckman KJ, et al. Reversing the direction of paced ventricular and atrial wavefronts reveals an oblique course in accessory AV pathways and improves localization for catheter ablation. Circulation 2001; 104:550–556.

36. de Chillou C, Rodriguez LM, Schlapfer J, et al. Clinical characteristics and electrophysiologic properties of atrioventricular accessory pathways: Importance of the accessory pathway location. J Am Coll Cardiol 1992; 20:666–671.

37. McClelland JH, Wang X, Beckman KJ, et al. Radiofrequency catheter ablation of right atriofascicular (Mahaim) accessory pathways guided by accessory pathway activation potentials. Circulation 1994; 89:2655–2666.

38. Nakagawa H, Yamanashi WS, Pitha JV, et al. Comparison of in vivo tissue temperature profile and lesion geometry for radiofrequency ablation with a saline-irrigated electrode versus temperature control in a canine thigh muscle preparation. Circulation 1995; 91:2264–2273.

39. Sun Y, Arruda M, Otomo K, et al. Coronary sinus-ventricular accessory connections producing posteroseptal and left posterior accessory pathways: Incidence and electrophysiological identification. Circulation 2002; 106:1362–1367.

40. Gaita F, Paperini L, Riccardi R, et al. Cryothermic ablation within the coronary sinus of an epicardial posterolateral pathway. J Cardiovasc Electrophysiol 2002; 13:1160–1163.

41. Haissaguerre M, Gaita F, Fischer B, et al. Radiofrequency catheter ablation of left lateral accessory pathways via the coronary sinus. Circulation 1992; 86:1464–1468.

42. Langberg JJ, Man KC, Vorperian VR, et al. Recognition and catheter ablation of subepicardial accessory pathways. J Am Coll Cardiol 1993; 22:1100–1104.

43. Lam C, Schweikert R, Kanagaratnam L, et al. Radiofrequency ablation of a right atrial appendage-ventricular accessory pathway by transcutaneous epicardial instrumentation. J Cardiovasc Electrophysiol 2000; 11:1170–1173.

44. Goya M, Takahashi A, Nakagawa H, et al. A case of catheter ablation of accessory atrioventricular connection between the right atrial appendage and right ventricle guided by a three-dimensional electroanatomic mapping system. J Cardiovasc Electrophysiol 1999; 10:1112–1118.

45. Hwang C, Peter CT, Chen PS. Radiofrequency ablation of accessory pathways guided by the location of the ligament of Marshall. J Cardiovasc Electrophysiol 2003; 14:616–620.

46. Kuck KH, Schluter M, Geiger M, et al. Radiofrequency current catheter ablation of accessory atrioventricular pathways. Lancet 1991; 337:1557–1561.

47. Lesh MD, Van Hare GF, Schamp DJ, et al. Curative percutaneous catheter ablation using radiofrequency energy for accessory pathways in all locations: Results in 100 consecutive patients. J Am Coll Cardiol 1992; 19:1303–1309.

48. Calkins H, Langberg J, Sousa J, et al. Radiofrequency catheter ablation of accessory atrioventricular connections in 250 patients. Abbreviated therapeutic approach to Wolff-Parkinson-White syndrome. Circulation 1992; 85:1337–1346.

49. Swartz JF, Tracy CM, Fletcher RD. Radiofrequency endocardial catheter ablation of accessory atrioventricular pathway atrial insertion sites. Circulation 1993; 87:487–499.

50. Kay GN, Epstein AE, Dailey SM, et al. Role of radiofrequency ablation in the management of supraventricular arrhythmias: Experience in 760 consecutive patients. J Cardiovasc Electrophysiol 1993; 4:371–389.

51. Morady F. Radio-frequency ablation as treatment for cardiac arrhythmias. N Engl J Med 1999; 340:534–544.

52. Hogenhuis W, Stevens SK, Wang P, et al. Cost-effectiveness of radiofrequency ablation compared with other strategies in Wolff-Parkinson-White syndrome. Circulation 1993; 88(Suppl. II):II-437–446.

53. Engelstein ED, Lippman N, Stein KM, et al. Mechanism-specific effects of adenosine on atrial tachycardia. Circulation 1994; 89:2645–2654.

54. Chen SA, Chiang CE, Yang CJ, et al. Sustained atrial tachycardia in adult patients. Electrophysiological characteristics, pharmacological response, possible mechanisms, and effects of radiofrequency ablation. Circulation 1994; 90:1262–1278.

55. Saoudi N, Cosio F, Waldo A, et al. A classification of atrial flutter and regular atrial tachycardia according to electrophysiological mechanisms and anatomical bases: A statement from a Joint Expert Group from the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J 2001; 22:1162–1182.

56. Poty H, Saoudi N, Haissaguerre M, et al. Radiofrequency catheter ablation of atrial tachycardias. Am Heart J 1996; 131:481–489.

57. Kalman JM, Olgin JE, Karch MR, et al. "Cristal tachycardia": Origin of right atrial tachycardias from the crista terminalis identified by intracardiac echocardiography. J Am Coll Cardiol 1998; 31:451–459.

58. Hoffmann E, Reithmann C, Nimmermann P, et al. Clinical experience with electroanatomic mapping of ectopic atrial tachycardia. PACE 2002; 25:49–56.

59. Kistler PM, Sanders P, Hussin A, et al. Focal atrial tachycardia arising from the mitral annulus. Electrocardiographic and electrophysiologic characterization. J Am Coll Cardiol 2003; 41:2212–2219.

60. Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med 1998; 339:659–666.

61. Tsai CF, Tai CT, Hsieh MH, et al. Initiation of atrial fibrillation by ectopic beats originating from the superior vena cava: Electrophysiological characteristics and results of radiofrequency ablation. Circulation 2000; 102:67–74.

62. Dong J, Schreieck J, Ndrepepa G, et al. Ectopic tachycardia originating from the superior vena cava. J Cardiovasc Electrophysiol 2002; 13:620–624.

63. Hwang C, Wu TJ, Doshi RN, et al. Vein of marshall cannulation for the analysis of electrical activity in patients with focal atrial fibrillation. Circulation 2000; 101:1503–1505.

64. Scavee C, Jais P, Weerasooriya R, et al. The inferior vena cava: An exceptional source of atrial fibrillation. J Cardiovasc Electrophysiol 2003; 14:659–662.

65. Tracy CM, Swartz JF, Fletcher RD, et al. Radiofrequency catheter ablation of ectopic atrial tachycardia using paced activation sequence mapping. J Am Coll Cardiol 1993; 21:910–917.

66. Pappone C, Stabile G, De Simone A, et al. Role of catheter-induced mechanical trauma in localization of target sites of radiofrequency ablation in automatic atrial tachycardia. J Am Coll Cardiol 1996; 27:1090–1097.

67. Schmitt C, Zrenner B, Schneider M, et al. Clinical experience with a novel multielectrode basket catheter in right atrial tachycardias. Circulation 1999; 99:2414–2422.

68. Rodriguez E, Callans D, Kantharia B, et al. Basket catheter localization of the origin of atrial tachycardia with atypical morphology after atrial flutter ablation. PACE 2000; 23:269–272.

69. Zrenner B, Ndrepepa G, Schneider M, et al. Basket catheter-guided three-dimensional activation patterns construction and ablation of common type atrial flutter. PACE 2000; 23:1350–1358.

70. Wetzel U, Hindricks G, Schirdewahn P, et al. A stepwise mapping approach for localization and ablation of ectopic right, left, and septal atrial foci using electroanatomic mapping. Eur Heart J 2002; 23:1387–1393.

71. Seidl K, Schwacke H, Rameken M, et al. Noncontact mapping of ectopic atrial tachycardias: Different characteristics of isopotential maps and unipolar electrogram. PACE 2003; 26:16–25.

72. Walsh EP, Saul JP, Hulse JE, et al. Transcatheter ablation of ectopic atrial tachycardia in young patients using radiofrequency current. Circulation 1992; 86:1138–1146.

73. Kay GN, Chong F, Epstein AE, et al. Radiofrequency ablation for treatment of primary atrial tachycardias. J Am Coll Cardiol 1993; 21:901–909.

74. Goldberger J, Kall J, Ehlert F, et al. Effectiveness of radiofrequency catheter ablation for treatment of atrial tachycardia. Am J Cardiol 1993; 72:787–793.

75. Lesh MD, Van Hare GF, Epstein LM, et al. Radiofrequency catheter ablation of atrial arrhythmias. Results and mechanisms. Circulation 1994; 89:1074–1089.

76. Feld GK, Fleck RP, Chen PS, et al. Radiofrequency catheter ablation for the treatment of human type 1 atrial flutter. Identification of a critical zone in the reentrant circuit by endocardial mapping techniques. Circulation 1992; 86:1233–1240.

77. Cosio FG, Lopez-Gil M, Goicolea A, et al. Radiofrequency ablation of the inferior vena cava-tricuspid valve isthmus in common atrial flutter. Am J Cardiol 1993; 71:705–709.

78. Nakagawa H, Lazzara R, Khastgir T, et al. Role of the tricuspid annulus and the eustachian valve/ridge on atrial flutter. Relevance to catheter ablation of the septal isthmus and a new technique for rapid identification of ablation success. Circulation 1996; 94:407–424.

79. Cauchemez B, Haissaguerre M, Fischer B, et al. Electrophysiological effects of catheter ablation of inferior vena cava-tricuspid annulus isthmus in common atrial flutter. Circulation 1996; 93:284–294.

80. Haissaguerre M, Jais P, Shah DC, et al. Electrophysiological end point for catheter ablation of atrial fibrillation initiated from multiple pulmonary venous foci. Circulation 2000; 101:1409–1417.

81. Shah DC, Haissaguerre M, Jais P, et al. Simplified electrophysiologically directed catheter ablation of recurrent common atrial flutter. Circulation 1997; 96:2505–2508.

82. Tada H, Oral H, Sticherling C, et al. Double potentials along the ablation line as a guide to radiofrequency ablation of typical atrial flutter. J Am Coll Cardiol 2001; 38:750–755.

83. Shah D, Takahashi A, Jais P, et al. Local electrogram-based criteria of cavotricuspid isthmus block. J Cardiovasc Electrophysiol 1999; 10:662–669.

84. Oral H, Sticherling C, Tada H, et al. Role of transisthmus conduction intervals in predicting bidirectional block after ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2001; 12:169–174.

85. Tada H, Oral H, Sticherling C, et al. Electrogram polarity and cavotricuspid isthmus block during ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2001; 12:393–399.

86. Nakagawa H, Jackman WM. Use of a three-dimensional, nonfluoroscopic mapping system for catheter ablation of typical atrial flutter. PACE 1998; 21:1279–1286.

87. Leonelli FM, Tomassoni G, Richey M, et al. Usefulness of three-dimensional non-fluoroscopic mapping in the ablation of typical atrial flutter. Ital Heart J 2002; 3:360–365.

88. Shah D, Haissaguerre M, Jais P, et al. High-density mapping of activation through an incomplete isthmus ablation line. Circulation 1999; 99:211–215.

89. Kottkamp H, Hugl B, Krauss B, et al. Electromagnetic versus fluoroscopic mapping of the inferior isthmus for ablation of typical atrial flutter: A prospective randomized study. Circulation 2000; 102:2082–2086.

90. Schneider MA, Ndrepepa G, Zrenner B, et al. Noncontact mapping-guided ablation of atrial flutter and enhanced-density mapping of the inferior vena caval-tricuspid annulus isthmus. PACE 2001; 24:1755–1764.

91. Schreieck J, Ndrepepa G, Zrenner B, et al. Radiofrequency ablation of cardiac arrhythmias using a three-dimensional real-time position management and mapping system. PACE. 2002; 25:1699–1707.

92. Schneider MA, Ndrepepa G, Dobran I, et al. LocaLisa catheter navigation reduces fluoroscopy time and dosage in ablation of atrial flutter: A prospective randomized study. J Cardiovasc Electrophysiol 2003; 14:587–590.

93. Morton JB, Sanders P, Davidson NC, et al. Phased-array intracardiac echocardiography for defining cavotricuspid isthmus anatomy during radiofrequency ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2003; 14:591–597.

94. Cabrera JA, Sanchez-Quintana D, Ho SY, et al. The architecture of the atrial musculature between the orifice of the inferior caval vein and the tricuspid valve: The anatomy of the isthmus. J Cardiovasc Electrophysiol 1998; 9:1186–1195.

95. Heidbuchel H, Willems R, van Rensburg H, et al. Right atrial angiographic evaluation of the posterior isthmus: Relevance for ablation of typical atrial flutter. Circulation 2000; 101:2178–2184.

96. Tsai CF, Tai CT, Yu WC, et al. Is 8-mm more effective than 4-mm tip electrode catheter for ablation of typical atrial flutter? Circulation 1999; 100:768–771.

97. Kasai A, Anselme F, Teo WS, et al. Comparison of effectiveness of an 8-mm versus a 4-mm tip electrode catheter for radiofrequency ablation of typical atrial flutter. Am J Cardiol 2000; 86:1029–1032, A1010.

98. Rodriguez LM, Nabar A, Timmermans C, et al. Comparison of results of an 8-mm split-tip versus a 4-mm tip ablation catheter to perform radiofrequency ablation of type I atrial flutter. Am J Cardiol 2000; 85:109–112, A109.

99. Marrouche NF, Schweikert R, Saliba W, et al. Use of different catheter ablation technologies for treatment of typical atrial

flutter: Acute results and long-term follow-up. PACE 2003; 26:743–746.

100. Ventura R, Willems S, Weiss C, et al. Large tip electrodes for successful elimination of atrial flutter resistant to conventional catheter ablation. J Interv Card Electrophysiol 2003; 8:149–154.

101. Jais P, Haissaguerre M, Shah DC, et al. Successful irrigated-tip catheter ablation of atrial flutter resistant to conventional radiofrequency ablation. Circulation 1998; 98:835–838.

102. Jais P, Shah DC, Haissaguerre M, et al. Prospective randomized comparison of irrigated-tip versus conventional-tip catheters for ablation of common flutter. Circulation 2000; 101:772–776.

103. Jais P, Hocini M, Gillet T, et al. Effectiveness of irrigated tip catheter ablation of common atrial flutter. Am J Cardiol 2001; 88:433–435.

104. Atiga WL, Worley SJ, Hummel J, et al. Prospective randomized comparison of cooled radiofrequency versus standard radiofrequency energy for ablation of typical atrial flutter. PACE 2002; 25:1172–1178.

105. Schreieck J, Zrenner B, Kumpmann J, et al. Prospective randomized comparison of closed cooled-tip versus 8-mm-tip catheters for radiofrequency ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2002; 13:980–985.

106. Timmermans C, Ayers GM, Crijns HJ, et al. Randomized study comparing radiofrequency ablation with cryoablation for the treatment of atrial flutter with emphasis on pain perception. Circulation 2003; 107:1250–1252.

107. Kirkorian G, Moncada E, Chevalier P, et al. Radiofrequency ablation of atrial flutter. Efficacy of an anatomically guided approach. Circulation 1994; 90:2804–2814.

108. Fischer B, Haissaguerre M, Garrigues S, et al. Radiofrequency catheter ablation of common atrial flutter in 80 patients. J Am Coll Cardiol 1995; 25:1365–1372.

109. Chen SA, Chiang CE, Wu TJ, et al. Radiofrequency catheter ablation of common atrial flutter: Comparison of electrophysiologically guided focal ablation technique and linear ablation technique. J Am Coll Cardiol 1996; 27:860–868.

110. Poty H, Saoudi N, Nair M, et al. Radiofrequency catheter ablation of atrial flutter. Further insights into the various types of isthmus block: Application to ablation during sinus rhythm. Circulation 1996; 94:3204–3213.

111. Schwartzman D, Callans DJ, Gottlieb CD, et al. Conduction block in the inferior vena caval-tricuspid valve isthmus: Association with outcome of radiofrequency ablation of typeI atrial flutter. J Am Coll Cardiol 1996; 28:1519–1531.

112. Schumacher B, Pfeiffer D, Tebbenjohanns J, et al. Acute and long-term effects of consecutive radiofrequency applications on conduction properties of the subeustachian isthmus in type I atrial flutter. J Cardiovasc Electrophysiol 1998; 9:152–163.

113. Chen J, de Chillou C, Basiouny T, et al. Cavotricuspid isthmus mapping to assess bidirectional block during common atrial flutter radiofrequency ablation. Circulation 1999; 100:2507–2513.

114. Tada H, Oral H, Ozaydin M, et al. Randomized comparison of anatomic and electrogram mapping approaches to ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2002; 13:662–666.

115. Ramanna H, Derksen R, Elvan A, et al. Ventricular tachycardia as a complication of atrial flutter ablation. J Cardiovasc Electrophysiol 2000; 11:472–474.

116. Ouali S, Anselme F, Savoure A, et al. Acute coronary occlusion during radiofrequency catheter ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2002; 13:1047–1049.

117. Paydak H, Kall JG, Burke MC, et al. Atrial fibrillation after radiofrequency ablation of type I atrial flutter: Time to onset, determinants, and clinical course. Circulation 1998; 98:315–322.

118. Fischer A, Mehta D. Atrial fibrillation after atrial flutter ablation. J Interv Card Electrophysiol 2002; 6:181–182.

119. Hsieh MH, Tai CT, Chiang CE, et al. Recurrent atrial flutter and atrial fibrillation after catheter ablation of the cavotricuspid isthmus: A very long-term follow-up of 333 patients. J Interv Card Electrophysiol 2002; 7:225–231.

120. Loutrianakis E, Barakat T, Olshansky B. Early versus late atrial fibrillation after atrial flutter ablation. J Interv Card Electrophysiol 2002; 6:173–180.

121. Nabar A, Rodriguez LM, Timmermans C, et al. Radiofrequency ablation of "class IC atrial flutter" in patients with resistant atrial fibrillation. Am J Cardiol 1999; 83:785–787, A710.

122. Schumacher B, Jung W, Lewalter T, et al. Radiofrequency ablation of atrial flutter due to administration of classIC antiarrhythmic drugs for atrial fibrillation. Am J Cardiol 1999; 83:710–713.

123. Tai CT, Chiang CE, Lee SH, et al. Persistent atrial flutter in patients treated for atrial fibrillation with amiodarone and propafenone: Electrophysiologic characteristics, radiofrequency catheter ablation, and risk prediction. J Cardiovasc Electrophysiol 1999; 10:1180–1187.

124. Kall JG, Rubenstein DS, Kopp DE, et al. Atypical atrial flutter originating in the right atrial free wall. Circulation 2000; 101:270–279.

125. Jais P, Shah DC, Haissaguerre M, et al. Mapping and ablation of left atrial flutters. Circulation 2000; 101:2928–2934.

126. Olgin JE, Jayachandran JV, Engesstein E, et al. Atrial macroreentry involving the myocardium of the coronary sinus: A unique mechanism for atypical flutter. J Cardiovasc Electrophysiol 1998; 9:1094–1099.

127. Kalman JM, VanHare GF, Olgin JE, et al. Ablation of 'incisional' reentrant atrial tachycardia complicating surgery for congenital heart disease. Use of entrainment to define a critical isthmus of conduction. Circulation 1996; 93:502–512.

128. Baker BM, Lindsay BD, Bromberg BI, et al. Catheter ablation of clinical intraatrial reentrant tachycardias resulting from previous atrial surgery: Localizing and transecting the critical isthmus. J Am Coll Cardiol 1996; 28:411–417.

129. Nakagawa H, Shah N, Matsudaira K, et al. Characterization of reentrant circuit in macroreentrant right atrial tachycardia after surgical repair of congenital heart disease: Isolated channels between scars allow "focal" ablation. Circulation 2001; 103:699–709.

130. Chan DP, Van Hare GF, Mackall JA, et al. Importance of atrial flutter isthmus in postoperative intra-atrial reentrant tachycardia. Circulation 2000; 102:1283–1289.

131. Saoudi N, Redonnet M, Anselme F, et al. Catheter ablation of atrioatrial conduction as a cure for atrial arrhythmia after orthotopic heart transplantation. J Am Coll Cardiol 1998; 32:1048–1055.

132. Scharf C, Oral H, Chugh A, et al. Acute effects of left atrial radiofrequency ablation on atrial fibrillation. J Cardiovasc Electrophysiol 2003; (In press).

133. Swartz JF, Pellersels G, Silvers J, et al. A catheter-based curative approach to atrial fibrillation in humans. (abstract) Circulation 1994; 90(Suppl. I):I-335.

134. Haissaguerre M, Jais P, Shah DC, abstract.. Right and left atrial radiofrequency catheter therapy of paroxysmal atrial fibrillation. J Cardiovasc Electrophysiol 1996; 7:1132–1144.

135. Gaita F, Riccardi R, Calo L. Atrial mapping and radiofrequency catheter ablation in patients with idiopathic atrial fibrillation. Electrophysiological findings and ablation results. Circulation 1998; 97:2136–2145.

136. Jais P, Shah DC, Takahashi A. Long-term follow-up after right atrial radiofrequency catheter treatment of paroxysmal atrial fibrillation. PACE 1998; 21:2533–2538.

137. Garg A, Finneran W, Mollerus M, et al. Right atrial compartmentalization using radiofrequency catheter ablation for management of patients with refractory atrial fibrillation. J Cardiovasc Electrophysiol 1999; 10:763–771.

138. Natale A, Leonelli F, Beheiry S, et al. Catheter ablation approach on the right side only for paroxysmal atrial fibrillation therapy: Long-term results. PACE 2000; 23:224–233.

139. Jais P, Haissaguerre M, Shah DC, et al. A focal source of atrial fibrillation treated by discrete radiofrequency ablation. Circulation 1997; 95:572–576.

140. Chen SA, Hsieh MH, Tai CT, et al. Initiation of atrial fibrillation by ectopic beats originating from the pulmonary veins: Electrophysiological characteristics, pharmacological responses, and effects of radiofrequency ablation. Circulation 1999; 100:1879–1886.

141. Shah DC, Haissaguerre M, Jais P. Catheter ablation of pulmonary vein foci for atrial fibrillation: PV foci ablation for atrial fibrillation. Thorac Cardiovasc Surg 1999; 47(Suppl. 3):352–356.

142. Haissaguerre M, Shah DC, Jais P, et al. Electrophysiological breakthroughs from the left atrium to the pulmonary veins. Circulation 2000; 102:2463–2465.

143. Natale A, Pisano E, Shewchik J, et al. First human experience with pulmonary vein isolation using a through-the-balloon circumferential ultrasound ablation system for recurrent atrial fibrillation. Circulation 2000; 102:1879–1882.

144. Oral H, Knight BP, Tada H, et al. Pulmonary vein isolation for paroxysmal and persistent atrial fibrillation. Circulation 2002; 105:1077–1081.

145. Shah DC, Haissaguerre M, Jais P, et al. Electrophysiologically guided ablation of the pulmonary veins for the curative treatment of atrial fibrillation. Ann Med 2000; 32:408–416.

146. Tada H, Oral H, Wasmer K, et al. Pulmonary vein isolation: Comparison of bipolar and unipolar electrograms at successful and unsuccessful ostial ablation sites. J Cardiovasc Electrophysiol 2002; 13:13–19.

147. Tada H, Oral H, Knight BP, et al. Randomized comparison of bipolar versus unipolar plus bipolar recordings during segmental ostial ablation of pulmonary veins. J Cardiovasc Electrophysiol 2002; 13:851–856.

148. Shah D, Haissaguerre M, Jais P, et al. Left atrial appendage activity masquerading as pulmonary vein potentials. Circulation 2002; 105:2821–2825.

149. Tada H, Oral H, Greenstein R, et al. Differentiation of atrial and pulmonary vein potentials recorded circumferentially within pulmonary veins. J Cardiovasc Electrophysiol 2002; 13:118–123.

150. Tada H, Oral H, Ozaydin M, et al. Response of pulmonary vein potentials to premature stimulation. J Cardiovasc Electrophysiol 2002; 13:33–37.

151. Oral H, Ozaydin M, Tada H, et al. Mechanistic significance of intermittent pulmonary vein tachycardia in patients with atrial fibrillation. J Cardiovasc Electrophysiol 2002; 13:645–650.

152. Oral H, Knight BP, Ozaydin M, et al. Segmental ostial ablation to isolate the pulmonary veins during atrial fibrillation: Feasibility and mechanistic insights. Circulation 2002; 106:1256–1262.

153. Macle L, Jais P, Weerasooriya R, et al. Irrigated-tip catheter ablation of pulmonary veins for treatment of atrial fibrillation. J Cardiovasc Electrophysiol 2002; 13:1067–1073.

154. Marrouche NF, Dresing T, Cole C, et al. Circular mapping and ablation of the pulmonary vein for treatment of atrial fibrillation: Impact of different catheter technologies. J Am Coll Cardiol 2002; 40:464–474.

155. Oral H, Chugh A, Scharf C, et al. Pulmonary vein isolation to prevent atrial fibrillation: Long-term safety, efficacy, and predictors of outcome. Submitted 2003; (abstract) J Am Coll Cardiol (In press).

156. Marrouche NF, Martin DO, Wazni O, et al. Phased-array intracardiac echocardiography monitoring during pulmonary vein isolation in patients with atrial fibrillation: Impact on outcome and complications. Circulation 2003; 107:2710–2716.

157. Hwang C, Karagueuzian HS, Chen PS. Idiopathic paroxysmal atrial fibrillation induced by a focal discharge mechanism in the left superior pulmonary vein: Possible roles of the ligament of Marshall. J Cardiovasc Electrophysiol 1999; 10:636–648.

158. Wu TJ, Ong JJ, Chang CM, et al. Pulmonary veins and ligament of marshall as sources of rapid activations in a canine model of sustained atrial fibrillation. Circulation 2001; 103:1157–1163.

159. Chen PS, Chou CC. Coronary sinus as an arrhythmogenic structure. J Cardiovasc Electrophysiol 2002; 13:863–864.

160. Chen PS, Wu TJ, Hwang C, et al. Thoracic veins and the mechanisms of non-paroxysmal atrial fibrillation. Cardiovasc Res 2002; 54:295–301.

161. Oral H, Ozaydin M, Chugh A, et al. Role of the coronary sinus in maintenance of atrial fibrillation. J Cardiovasc Electrophysiol 2003; (In press).

162. Schmitt C, Ndrepepa G, Weber S, et al. Biatrial multisite mapping of atrial premature complexes triggering onset of atrial fibrillation. Am J Cardiol 2002; 89:1381–1387.

163. Lin WS, Tai CT, Hsieh MH, et al. Catheter ablation of paroxysmal atrial fibrillation initiated by non-pulmonary vein ectopy. Circulation 2003; 107:3176–3183.

164. Oral H, Knight BP, Tada H, et al. Pulmonary vein isolation for paroxysmal and persistent atrial fibrillation. Circulation 2002; 105:1077–1081.

165. Scharf C, Sneider M, Case I, et al. Anatomy of the pulmonary veins in patients with atrial fibrillation and effects of segmental ostial ablation analyzed by computed tomography. J Cardiovasc Electrophysiol 2003; 14:150–155.

166. Kato R, Lickfett L, Meininger G, et al. Pulmonary vein anatomy in patients undergoing catheter ablation of atrial fibrillation: Lessons learned by use of magnetic resonance imaging. Circulation 2003; 107:2004–2010.

167. Saad EB, Marrouche NF, Saad CP, et al. Pulmonary vein stenosis after catheter ablation of atrial fibrillation: Emergence of a new clinical syndrome. Ann Intern Med 2003; 138:634–638.

168. Jais P, Hocini M, Sanders P, et al. Importance of epicardial ablation through the coronary sinus to complete mitral isthmus block in ablation of paroxysmal atrial fibrillation. (abstract) PACE 2003; 26:1006.

169. Jais P, Weerasooriya R, Scavee C, et al. Catheter ablation of chronic atrial fibrillation using a combination of left linear lesions and pulmonary vein isolation. (abstract) PACE 2003; 26:962

170. Marchlinski FE, Callans D, Dixit S, et al. Efficacy and safety of targeted focal ablation versus PV isolation assisted by magnetic electroanatomic mapping. J Cardiovasc Electrophysiol 2003; 14:358–365.

171. Pappone C, Rosanio S, Oreto G, et al. Circumferential radiofrequency ablation of pulmonary vein ostia: A new anatomic approach for curing atrial fibrillation. Circulation 2000; 102:2619–2628.

172. Pappone C, Oreto G, Rosanio S, et al. Atrial electroanatomic remodeling after circumferential radiofrequency pulmonary vein ablation: Efficacy of an anatomic approach in a large cohort of patients with atrial fibrillation. Circulation 2001; 104:2539–2544.

173. Pappone C, Rosanio S, Augello G, et al. Mortality, morbidity, and quality of life after circumferential pulmonary vein ablation for atrial fibrillation. Outcomes from a controlled, nonrandomized, long-term study. J Am Coll Cardiol 2003; 42:185–197.

174. Oral H, Scharf C, Chugh A, et al. Catheter ablation for paroxysmal atrial fibrillation: Segmental pulmonary vein ostial ablation vs. left atrial ablation. Circulation 2003; 108:2355–2360

175. Arora R, Verheule S, Scott L, et al. Arrhythmogenic substrate of the pulmonary veins assessed by high-resolution optical mapping. Circulation 2003; 107:1816–1821.

176. Mandapati R, Skanes A, Chen J, et al. Stable microreentrant sources as a mechanism of atrial fibrillation in the isolated sheep heart. Circulation 2000; 101:194–199.

177. Jalife J, Berenfeld O, Mansour M. Mother rotors and fibrillatory conduction: A mechanism of atrial fibrillation. Cardiovasc Res 2002; 54:204–216.

178. Wyse DG, Waldo AL, DiMarco JP, et al. A comparison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med 2002; 347:1825–1833.

179. Strickberger SA, Tokano T, Kluiwstra JU, et al. Extracardiac ablation of the canine atrioventricular junction by use of high-intensity focused ultrasound. Circulation 1999; 100:203–208.

180. Kuon E, Glaser C, Dahm JB. Effective techniques for reduction of radiation dosage to patients undergoing invasive cardiac procedures. Br J Radiol 2003; 76:406–413.

181. Faddis MN, Lindsay BD. Magnetic catheter manipulation. Coron Artery Dis 2003; 14:25–27.

182. Faddis MN, Blume W, Finney J, et al. Novel, magnetically guided catheter for endocardial mapping and radiofrequency catheter ablation. Circulation 2002; 106:2980–2985.

183. Liu TY, Tai CT, Chen SA. Treatment of atrial fibrillation by catheter ablation of conduction gaps in the crista terminalis and cavotricuspid isthmus of the right atrium. J Cardiovasc Electrophysiol 2002; 13:1044–1046.

184. Avitall B, Helms RW, Koblish JB, et al. The creation of linear contiguous lesions in the atria with an expandable loop catheter. J Am Coll Cardiol 1999; 33:972–984.