Catheter Ablation of Atrial Fibrillation
Roderick Tung, MD; Eric Buch, MD; Kalyanam Shivkumar, MD, PhD

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and is associated with significant morbidity and cost.1 AF is seen across a wide spectrum of patients, from lone AF without structural heart disease, to the postoperative setting, to patients with significant left ventricular dysfunction and advanced heart failure. AF is clinically classified as paroxysmal (<7 days), persistent (>7 days or requiring intervention to restore sinus rhythm), longstanding persistent (>1 year), or permanent, when restoration of sinus rhythm is no longer pursued.2

The presence of AF is associated with increased mortality, and treatment of patients requires symptom relief and prevention of thromboembolism. Optimal antithrombotic therapy is determined on the basis of stroke risk as predicted by the CHADS2 scoring system (a system that awards 1 point each for congestive heart failure, hypertension, age ≥75 years, and diabetes mellitus and 2 points for prior stroke or transient ischemic attack).3,4 More recently, a modified scoring system, CHA2DS-VASC2, has been shown to improve risk prediction for patients previously thought to be at low risk for thromboembolic events.5 Large-scale randomized trials have not shown mortality reduction with a rhythm control strategy compared with a rate control strategy6; however, many patients continue to have symptoms despite adequate rate control. For these patients, 3 therapeutic options are available for AF: (1) Antiarrhythmic drug therapy, (2) catheter ablation, and (3) surgical intervention (Maze procedure). Antiarrhythmic drug therapy has limited efficacy (40%–60%) and important side effects and has been shown to be less effective than catheter ablation in multiple comparative studies.7 In this Clinician Update, we illustrate current ablation strategies used to treat paroxysmal and persistent AF in the electrophysiology laboratory.

Case 1: Paroxysmal AF With Superior Vena Cava Triggers
A 57-year-old man with a history of hypertension presented with palpitations for 2 years, which had worsened during the previous 4 months. An event monitor demonstrated that his symptoms were correlated with episodes of paroxysmal AF that lasted from 15 minutes to 3 hours. Antiarrhythmic therapy with dronedarone and flecainide did not control his symptoms. An echocardiogram demonstrated a structurally normal heart with left ventricular ejection fraction of 60% and left atrial dimension of 42 mm. He was referred for elective catheter ablation. Preprocedural magnetic resonance imaging demonstrated normal left atrial dimensions and conventional pulmonary vein (PV) anatomy.

After induction of general anesthesia, transesophageal echocardiography was performed to exclude left atrial appendage thrombus. Double transseptal access was obtained by puncture under intracardiac catheter-based echocardiographic guidance after administration of intravenous heparin for systemic anticoagulation. A circular mapping catheter was placed in or near each PV ostium, and circumferential ablation was performed on the atrial side of each PV with an open-irrigated ablation catheter until electric isolation of all PVs was demonstrated. Isoproterenol (20 μg/min) was administered intravenously, and frequent PACs were seen. Activation mapping revealed the superior vena cava as the origin of the trigger. Ablation was performed at the junction of the right atrium and superior vena cava until electric isolation or abolition of all venous potentials was achieved.
The patient was seen in follow-up at 1 month and every 3 months after, with 1-week event monitors obtained at these time points. Warfarin was initiated after ablation for 3 months with a low-molecular-weight heparin bridge. Aspirin monotherapy was then resumed given his CHADS2 risk score of 1. At 1 year, the patient remained free of AF recurrence.

Catheter ablation for paroxysmal AF involves an anatomic approach aimed at permanently interrupting electric conduction between the PVs and left atrium. PV potentials are seen as high-frequency signals arising from muscular sleeves that extend from the venous structure to the atrium. The use of a circular mapping catheter shows near simultaneous circumferential electric activation of pulmonary venous muscle sleeves and adjacent left atrial tissue. Ablation is only performed outside the vein to avoid the risk of PV stenosis. The position of the ablation catheter relative to the PV ostium is verified with intracardiac echocardiography, fluoroscopy, electric impedance, and a 3-dimensional electroanatomic mapping system.

The electrophysiological end point is complete elimination of PV potentials on the circular catheter, representing entrance block from the left atrium to the PVs during sinus rhythm. Bidirectional block is demonstrated when pacing from the circular catheter on the venous side of the ablation line results in local PV but not left atrial capture (Figure 1).

After PV isolation is achieved, other triggering premature atrial complexes that may initiate AF can be induced with infusion of isoproterenol, up to 20 μg/min. Potential non-PV triggers can originate from the superior vena cava, inferior vena cava, coronary sinus, left atrial appendage, vein of Marshall, and interatrial septum. Ablation that targets these triggers in addition to PV isolation has been shown to decrease AF recurrence.

Catheter ablation has been shown to be more effective than antiarrhythmic...
drug therapy to prevent recurrence of AF (Figure 2). Success rates for patients with paroxysmal AF range from 60% to 80% at 1 year.15–23 Table 1 shows the indications and contraindications for catheter ablation. A late recurrence rate of 10% after the first 1 to 2 years has been shown in long-term follow-up studies of 5 years.24,25

**Case 2: Persistent AF With Reversible Cardiomyopathy**

A 43-year-old man presented with persistent AF of 3 years’ duration that was refractory to amiodarone and with multiple cardioversions. His ejection fraction was 35% despite aggressive rate control, and coronary angiography showed normal coronary arteries. He denied any history of alcohol or cocaine use. The patient was taking warfarin, and because of his decreased systolic function and need for postablation anticoagulation, this was continued throughout the perioperative period without interruption.26

An elective catheter ablation procedure was performed, beginning with isolation of all 4 PVs, which resulted in bidirectional block. High-dose isoproterenol and rapid atrial burst pacing did not induce fibrillation or reveal any triggers. The patient remained in sinus rhythm for 3 months, and amiodarone was discontinued. A repeat echocardiogram demonstrated normalization of left ventricular function with an ejection fraction of 55%.

Six months later, the patient reported 3 episodes of paroxysmal AF that lasted from hours up to 4 days. He presented in AF of 1 week’s duration and elected to undergo a second ablation procedure.

During this procedure, mapping of the 4 PVs demonstrated reconnection in the left superior PV and right superior PV. Focal ablation at sites recording PV potentials promptly resulted in block. In addition, complex fractionated signals (multiple components with low-amplitude and high-frequency electrograms) were seen in the inferior left atrium. During ablation at these sites, the fibrillatory cycle length slowed, and AF organized into atrial flutter. Entrainment pacing was performed from the isthmus between the mitral annulus and the left inferior PV, which demonstrated that it was part of the flutter circuit (with a postspacing interval within 30 ms of the tachycardia cycle length). Additional ablation in the mitral isthmus resulted in termination of atrial flutter and restoration of normal sinus rhythm (Figure 3). Ablation was performed in the coronary sinus to achieve bidirectional block across the mitral isthmus.

At 14-month follow-up from the second procedure, with serial event recorder monitoring at 3, 6, and 12 months, the patient has remained free from AF recurrence, and the left ventricular ejection fraction continues to be normal. Anticoagulation with warfarin was discontinued, and the patient was prescribed aspirin monotherapy for stroke prevention.

Approximately 40% to 50% of patients undergoing ablation of persistent AF require a repeat procedure to achieve a success rate of 50% to 70%.

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**Figure 2.** Summary of randomized control trials comparing catheter ablation versus antiarrhythmic therapy. Three studies showed enrolled patients with paroxysmal atrial fibrillation (AF), 2 studies enrolled patients with persistent AF, and a mixed population was enrolled in 3 studies. AAD indicates antiarrhythmic drugs.

**Table 1. Indications and Contraindications for Catheter Ablation of AF**

<table>
<thead>
<tr>
<th>Indications</th>
<th>Contraindications</th>
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<tbody>
<tr>
<td>Symptomatic (eg, palpitations, fatigue, effort intolerance)</td>
<td>Contraindication to anticoagulation (required postablation)</td>
</tr>
<tr>
<td>Antiarrhythmic drugs (class I or III) ineffective or not tolerated/desired</td>
<td>Left atrial appendage thrombus</td>
</tr>
<tr>
<td>Better candidacy</td>
<td>Severe mitral valve disease or mechanical mitral valve prosthesis</td>
</tr>
<tr>
<td>Younger age (&lt;70 y)</td>
<td>Severe pulmonary hypertension</td>
</tr>
<tr>
<td>Paroxysmal persistent longstanding persistent or permanent</td>
<td>Worse candidacy</td>
</tr>
<tr>
<td>Minimal structural heart disease and left atrial enlargement</td>
<td>Advanced age (&gt;70 y)</td>
</tr>
<tr>
<td>Younger age (&lt;70 y)</td>
<td>Minimal symptoms</td>
</tr>
<tr>
<td>Paroxysmal/persistent/longstanding persistent or permanent</td>
<td>Left atrial enlargement (&gt;5.0 cm)</td>
</tr>
<tr>
<td>Minimal symptoms</td>
<td>Left ventricular dysfunction</td>
</tr>
<tr>
<td>Prior stroke or transient ischemic attack</td>
<td>Pulmonary disease or sleep apnea</td>
</tr>
</tbody>
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**AAD** indicates antiarrhythmic drugs.
for persistent AF. Mechanisms for recurrence include reconnection of PVs, non-PV triggers, and progressive atrial electric and structural remodeling. The optimal ablation technique for persistent AF warrants further study, but the bulk of the evidence supports that additional ablation lesion sets beyond PVI are necessary for optimal results. The rationale is that elimination of triggers is not sufficient when the atrial substrate has electrically remodeled to support the maintenance of AF.

Linear lesion sets in the roof, posterior wall, and mitral isthmus increase procedural success but can also be proarrhythmic and result in complex atypical flutter circuits that may be more difficult to rate control and more symptomatic than AF. Catheter ablation guided by electrograms that demonstrate complex fractionation or dominant frequency sites has been shown to be a useful adjunct in patients with persistent AF. Progressive slowing of fibrillatory cycle lengths has been shown to predict organization into atrial flutter, and termination during ablation can be achieved when committing to a stepwise ablation approach of more extensive lesions. Recent work shows that human AF may be sustained by a small number (1–3) of spiral waves (rotors) or focal sources, lying in both atria. Targeted ablation (Focal Impulse and Rotor Modulation, FIRM) can terminate AF prior to PV isolation, and improve AF elimination compared to PV isolation alone. Ongoing multicenter studies are extending these promising data.

Tachycardia-mediated cardiomyopathy continues to be an underrecognized cause of left ventricular systolic dysfunction. Successful elimination of sustained and irregular arrhythmias can result in resolution of cardiomyopathy over a period of 1 to 2 months. AF and systolic dysfunction frequently coexist, and ablation has been demonstrated to be effective in patients with congestive heart failure, particularly in those without established causes.

Postablation Management
Because longer-term outcomes after AF have demonstrated late recurrences, the notion that ablation can be curative is not well supported by present data. No prospective randomized trials demonstrate that stroke risk in a successfully ablated patient returns to that of a patient without a history of AF. The perceived success rate from the procedure is also dependent on the rigor with which postablation monitoring is performed, because many patients may have asymptomatic recur-
References. Longer monitoring periods with an event monitor are preferable and more sensitive than 24-hour monitoring and an instantaneous 12-lead ECG obtained at clinical follow-up. Although recent data suggest that anticoagulation can be discontinued in higher-risk patients after successful ablation, current guidelines recommend that patients maintain anticoagulation based on their preprocedural substrate-based risk (CHADS2 or CHA2DS2-VASC) rather than postablation rhythm.2

All patients require a period of anticoagulation after ablation (minimum 2 months). Bridging therapy with intravenous heparin and low-molecular-weight heparin is necessary for patients who resume taking warfarin. More recently, the safety and efficacy of continued warfarin throughout the periprocedural period have been shown. Dabigatran can be used as an effective alternative to warfarin, although continuation throughout the procedure is not desirable because there is no specific antidote to reverse its effects if bleeding complications arise.24-26 Table 2 summarizes the use of anticoagulants for stroke prevention stratified by substrate risk.

<table>
<thead>
<tr>
<th>Stroke Risk (CHADS2 Score)</th>
<th>Preprocedural</th>
<th>Intraprocedural</th>
<th>Postprocedural</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>ASA (81–325 mg)</td>
<td>Heparin (ACT &gt;300)</td>
<td>Warfarin (INR 2–3) for ≥2 mo, then ASA (81–325 mg)</td>
</tr>
<tr>
<td>1</td>
<td>ASA (81–325 mg) or warfarin (INR 2–3) or dabigatran</td>
<td>Heparin (ACT &gt;300); some centers also continue warfarin</td>
<td>LMWH bridging until INR &gt;2 and warfarin for ≥2 mo, then long-term ASA or warfarin or dabigatran</td>
</tr>
<tr>
<td>≥2</td>
<td>Warfarin (INR 2–3) or dabigatran</td>
<td>Heparin (ACT &gt;300); some centers also continue warfarin</td>
<td>LMWH bridging (unless INR &gt;2) and long-term warfarin or dabigatran</td>
</tr>
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ASA indicates acetylsalicylic acid (aspirin); ACT, activated clotting time; INR, international normalized ratio; and LMWH, low-molecular-weight heparin.

Conclusions

Ever since the observation that pulmonary venous triggers could be managed by catheter ablation in 1998,29 these procedures increasingly have been used to treat patients with AF. Recent studies have reported success rates of >70% in paroxysmal AF and >50% in persistent AF, with superiority compared with antiarrhythmic therapy. However, the mechanism by which patients derive benefit from catheter ablation remains incompletely understood. Aside from the elimination of triggers for AF or interruption of rotors/drivers, neural modulation of ganglionic plexi and atrial debulking may result from ablation around pulmonary venous ostia and antra.29-30-51

The most feared complication of AF ablation is the creation of an atrioesophageal fistula, which has a declining incidence of 0.01–0.03% of patients.52-53 Both real-time temperature recordings in the esophagus during ablation and contrast imaging have become standard. Specialized techniques such as placement of an intrapericardial balloon to create separation between the atrium and esophagus can be implemented if a targeted region directly overlies the esophagus.53

Catheter ablation of AF, when performed by experienced operators, demonstrates superior efficacy compared with antiarrhythmic therapy, with acceptably low complication rates (<1% death and stroke). Ongoing trials such as the Catheter Ablation versus Antiarrhythmic Therapy for Atrial Fibrillation Trial (CABANA) will offer further insight into the consistency and generalizability of single-center observations, as well as assess the relative cost of therapy and the impact on quality of life (http://clinicaltrials.gov; unique identifier, NCT00911508). Because patients with AF have different mechanisms of arrhythmia initiation and perpetuation, further studies on substrate-tailored ablation approaches are necessary as the field continues to advance.

Disclosures

None.

References

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