Catheter Ablation of Atrial Fibrillation

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KEYWORDS

• Atrial fibrillation • Catheter ablation • Outcomes

KEY POINTS

- When performed by experienced operators, catheter ablation is a safe and effective option for the management of atrial fibrillation (AF).
- Further understanding of the pathophysiologic mechanisms of AF would facilitate development of novel strategies and technologies to improve ablation outcomes.
- Postprocedure clinical follow-up and management are critical to achieve the full benefits of ablation.

INTRODUCTION

Catheter ablation was introduced as a therapeutic option for the management of atrial fibrillation (AF) in the late 1990s and is now readily available in many middle-volume to large-volume cardiovascular centers worldwide. Its growth has been accompanied by changes in procedural strategy, advances in mapping and ablation technologies, refinement in periprocedural management, and a better understanding of the mechanisms driving and perpetuating AF. Rates of sinus rhythm maintenance have remained stable over the past 15 years despite inclusion of a broader selection of patients with persistent and long-standing persistent AF as well as multiple cardiac and other comorbidities including cardiomyopathy, valvular disease, and renal failure. Long-term benefits of sinus rhythm maintenance in addition to symptomatic relief are also under investigation.

PREOPERATIVE PLANNING

Patient Selection

Current American Heart Association (AHA)/American College of Cardiology (ACC)/Heart Rhythm Society (HRS) guidelines endorse catheter ablation as a class I recommendation for patients with symptomatic paroxysmal AF refractory or intolerant to at least 1 class I or III antiarrhythmic drugs (AADs), or a class IIa recommendation when offered before initiation of an AAD.[1] In patients with symptomatic persistent AF refractory or intolerant to at least 1 class I or III AAD, catheter ablation is considered a class IIa recommendation.[1] Although not considered contraindications to ablation, other patient characteristics to consider include significant left atrial dilatation, prolonged duration of AF, and patient age and comorbidities. Ablation should not be considered in patients who cannot receive anticoagulation during the periprocedural period.

The primary goal of ablation remains symptomatic improvement, although some studies suggest other benefits such as improved left ventricular function or reduced stroke risk. Ablation should not be performed with the goal of eliminating the need for long-term anticoagulation.

Preprocedure Testing

Before scheduled AF ablation, patients should have documentation of their arrhythmia. Long-term

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monitoring may reveal an inciting supraventricular tachycardia or single-focus ectopy that degenerates into or triggers AF. Such patients may simply require a more targeted ablation for their primary arrhythmia.

Patients should be anticoagulated before ablation. In patients with persistent AF, transesophageal echocardiography should be performed to rule out left atrial or left atrial appendage clot or the patient should be verified to be therapeutically anticoagulated for at least 3 consecutive weeks before ablation. Cardiac computed tomography or magnetic resonance imaging to delineate left atrial and pulmonary vein (PV) anatomy can be helpful.

Anticoagulation

Perioperative anticoagulation with warfarin or target-specific oral anticoagulants (TSOACs; ie, dabigatran, rivaroxaban, or apixaban) is usually maintained for at least 1 month before and at least 3 months following catheter ablation. In the recent COMPARE (Role of Coumadin in Preventing Thromboembolism in Atrial Fibrillation Patients Undergoing Catheter Ablation)[2] trial, patients randomized to uninterrupted warfarin had fewer periprocedural stroke and minor bleeding episodes compared with those who discontinued warfarin before the procedure. Bleeding complications can also be managed effectively despite therapeutic anticoagulation. In patients who have tamponade during ablation, early recognition and management results in favorable outcomes even among those with uninterrupted warfarin.[3] With the available safety data and potential to avoid the inconvenience and cost of bridging with heparin-based products, maintaining warfarin throughout the perioperative period may be the optimal strategy. Without reliable reversal agents for the TSOAC, most patients discontinue these medications before their ablation. However, early data suggest that uninterrupted TSOAC use may also be safe without increased bleeding or thromboembolic complications in the perioperative period.[4] These data need to be verified in larger scale, prospective studies.

ABLATION STRATEGY

AF Mechanisms

Reliable elimination of AF by ablation requires an understanding of the mechanisms by which AF is induced and perpetuated. Although considerable progress in this endeavor has been achieved over the past century, significant questions remain. Multiple mechanisms have been shown in vivo as well as in simulated models and varying mechanisms are likely to be predominant in different patients (**Fig. 1**).[5–7]

Early hypotheses for the mechanisms of AF include rapidly firing foci (1907),[8] so-called circus movement or reentry (1913),[9] and the multiple wavelets theory (1960s).[10] Recognition of rapid depolarizations from the PVs initiating and perpetuating AF was instrumental in providing an effective target for catheter-based therapy, and PV isolation (PVI) has remained a key component of most ablation strategies.[11] Other areas recognized to possess arrhythmogenic properties that may contribute to AF include the superior vena cava, inferior vena cava, coronary sinus, and ligament of Marshall. These seem to be less commonly arrhythmogenic than PVs, but may serve as adjunctive targets in select patients.[12–14]

In addition to focal triggers, high-frequency sources described as rotors have been identified in both animal and human models, supporting their role in maintaining AF.[5] These organized areas of electrical activity may or may not remain stationary and have been shown to produce wavebreak and fibrillatory conduction at their periphery, leading to disorganized activity throughout the remainder of the atria.[15,16] Identifying these elusive targets has been the focus of some recent ablation strategies to map more specific electrogram-guided and not purely anatomic targets. In addition, the role of the autonomic system through both sympathetic and parasympathetic influences has been widely described.[17] In particular, stimulation of atrial ganglionated plexi (GPs) has been shown to trigger PV arrhythmias and increase AF inducibility.[18]

PVI

The emergence of catheter ablation as a viable strategy to treat AF began with showing the efficacy of targeting focal PV triggers by electrically isolating the PVs.[11] With recognition of antral tissue harboring critical atrial substrate[19] as well as to avoid PV stenosis, PVI has evolved from an ostial to an antral approach (**Fig. 2**).[20] Despite high acute rates in achieving PVI (>95%),[21] reconnection of some veins can be shown in most patients regardless of clinical arrhythmia recurrence.[22] Adenosine infusion following isolation has been suggested as a means to unmask dormant PV connection and prompt further ablation; however, whether this translates into long-term clinical improvement is unclear. Newer technologies such as real-time monitoring of catheter contact force or real-time lesion visualization need to be evaluated in terms of their ability to produce more durable lesions. At present, antral PVI remains the most widely used strategy in ablation of both paroxysmal and