## Review Article

# Catheter Ablation for Atrial Fibrillation

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Atrial fibrillation (AF) is an arrhythmia associated with increased morbidity and mortality. The pulmonary veins (PVs) play an important role not only in the onset but also the maintenance of AF. Therefore, the goal of present AF ablation is to electrically disconnect the PVs from the rest of the atrium by ablating around the origin of the PVs. Several groups using ablation of all 4 PVs outside the tubular portion have reported that the success rate without antiarrhythmic drugs is much more consistent, at 75 to 95%. A further 10% to 20% of patients may become responsive to previously ineffective antiarrhythmic drugs. Although the success rate of AF ablation appears high, a very low incidence of complications has been reported including cardiac tamponade, embolism, esophageal injury, PV stenosis, and proarrhythmia resulting from reentrant tachycardias. However, the complication rates can be decreased by more recent modifications to the technique and presently available technologies. Thus, AF ablation is an effective, safe, and established treatment for AF that offers an excellent chance for a lasting cure.
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Atrial fibrillation (AF) is a common and recurrent rhythm disorder that affects patient morbidity and mortality. Catheter ablation for AF has emerged as a promising new treatment strategy. In contrast to antiarrhythmic drugs, catheter ablation directly eliminates the inciting factors for AF and offers the possibility of a lasting cure. Initially, various methods for ablating AF have been tried. Creating linear lesions in the atria mimicked the surgical Maze procedure. However, after recognizing the limited success and risks associated with this approach, Haissaguerre et al.[1] first found that most focal AF is initiated by premature beats from the orifices of the pulmonary veins (PVs) or from the myocardial sleeves inside the PVs, and radiofrequency catheter ablation of triggered foci has been shown to cure AF. However, detecting the exact focus is often difficult when atrial premature beats are infrequent, or each extrasystole may induce AF, thus necessitating repeated defibrillation. For these reasons, Haissaguerre et al.[2] provided an alternative approach that simply seeks to electrically isolate the PVs from the left atrium (LA) at electrophysiological breakthroughs from the LA to the PV.

### Rationale of PV Isolation

In patients with chronic AF and structural heart disease, after electrical cardioversion, the PVs are also the dominant trigger reinitiating AF.[3] Therefore, the PVs may have an important role not only in the onset but also the maintenance of AF.[4] AF is

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also perpetuated by microreentrant circuits, or rotors, that exhibit high-frequency, periodic activity from which spiral wave fronts of activation radiate into surrounding atrial tissue.[5,6] Conduction becomes slower and less organized with increasing distance from the rotors, likely because of atrial structural remodeling, resulting in fibrillatory conduction. The dominant rotors in AF are localized primarily at the PV–LA junction, as demonstrated by several investigators.[6–8] Moreover, vagal inputs may be very important in both triggering and maintaining AF, and many of these inputs are clustered close to the PV–LA junction.[9] We also demonstrated that the PV–LA junction has heterogeneous electrophysiological properties capable of sustaining reentry.[10] In our study using a basket catheter mapping, unstable reentrant circuits were observed in response to single extrastimulus and repetitive focal activities in the PV. When a coupling interval of extrastimulus or the cycle lengths of repetitive focal activities are so short, the rest of the PV cannot follow 1:1. The result is that areas of functional block and slow conduction are generated, which in turn serve to form unstable reentrant circuit. However, these reentrant circuits were short lived and never sustained for more than 2 rotations (i.e., were unstable). Furthermore, as shown in the maps during initiation of AF (**Figure 1**), a PV–LA reciprocating reentrant circuit involving the exit breakthrough point and the entrance breakthrough point at the PV–LA junction was observed. A wave front from a focal discharge in the PV goes through the nearest exit breakthrough point and re-enters to the PV from the entrance site, forming a reentrant circuit (**Figure 2**). The different conduction property of the exit and entrance sites depending on the site of pacing or discharge may contribute to the reentry formation. Wave fronts traveling to and from the LA may play an important role in the formation of unstable reentrant wave fronts. The presence of anisotropic structures at the PV–LA junction may be critical to form reentry.

Thus, the PVs play a critical role in both triggering and maintaining AF. This was confirmed with both electrophysiological and histological data in an open-heart, human model involving patients undergoing AF surgery.[11] The mechanism also applies to a wide spectrum of AF patients, including adolescents[12] and adults with structural heart disease.[13,14] Therefore, the goal of present AF ablation is to electrically disconnect the PVs from the rest of the atrium by ablating around the origin of the PVs.

## Methodology of the Technique

It has been reported that >90% of PVs were electrically disconnected from the LA by targeting only certain segments of the ostial circumference, as guided by PV potentials.[2] The results confirm that there are isolated fascicles that travel from the LA into the muscle sleeves surrounding the PVs and that the ablation of these fascicles, as opposed to circumferential ablation at the ostium, is sufficient to isolate the PVs. In prior studies of segmental PV isolation, only the PVs that were found to generate triggers of AF were isolated.[2] Recently, most centers performing AF ablation are empirically isolating all 4 PVs. It was recognized that any of the PVs can serve as a trigger and that ablation of only a single culprit PV may unmask triggers in other PVs.[15] Ablation of all 4 PVs was also found to be more effective than ablation of only 3.[16] Moreover, most groups are ablating outside the tubular portion of the PV to avoid the risk of PV stenosis and to improve the efficacy of the procedure. This makes sense given that the PV is funnel shaped with a large proximal end, which is called the antrum.[17] The antrum blends into the posterior wall of the LA, and on the posterior wall. Therefore, to encompass as much of the PV structure as possible, ablation needs to be performed around the entire antrum, along the posterior LA wall.[17] Although different groups may refer to ablation in this region by different names such as LA catheter ablation, circumferential PV antrum ablation, or extraostial isolation, the lesion sets produced by the procedures are all very similar (**Figure 3**).

In our own institution, we perform PV antrum isolation using the double Lasso technique,[18] that is, two decapolar Lasso catheters (Biosense-Webster) were placed within the ipsilateral superior and inferior PVs during radiofrequency delivery. Ablation of the PVs was performed as a pair. It was performed 1 cm from the ostium of both right PVs and for the posterior and superior aspects of the left PVs to minimize the risk of PV stenosis. It was started at the posterior wall and continued around the venous perimeter. When ablation was required at the anterior portions of the left PVs, energy had to be delivered within the first millimeters of the vein (rather than the posterior wall of appendage) to achieve effective disconnection. Radiofrequency energy was delivered for 30 seconds at each point; this application was prolonged for 60 seconds when a change in morphology or in the sequence of the PV potentials occurred as determined by circumferential