Published in final edited form as:

J Interv Card Electrophysiol. 2003 October ; 9(2): 249–257.

Atrial Fibrillation: What are the Targets for Intervention?

John M. Miller, Jeffrey E. Olgin, and Mithilesh K. Das

Krannert Institute of Cardiology, Cardiovascular Division, Department of Medicine, Indiana University School of Medicine, USA

Abstract

Atrial fibrillation (AF) is a difficult and growing problem in the population. While medical therapy controls symptoms in many patients, a proportion of individuals with this common arrhythmia cannot be optimally managed with drugs alone. However, truly curative therapy for AF has always been one of the “holy grails” of electrophysiology. The surgical maze procedure was the first to offer permanent maintenance of sinus rhythm in patients with AF but subjected the patient to open heart surgery; a catheter-based translation of the maze procedure served as proof of concept that a catheterization technique could be used to treat AF. Subsequent experience has narrowed the electrophysiologist’s attention to ablation of triggers of AF, most often residing in the pulmonary veins, rather than requiring more extensive ablation lines to control the arrhythmia. The following discussion deals with the development and current status of techniques for catheter ablation of atrial fibrillation, focusing on determination of appropriate target sites for ablation.

Keywords

atrial fibrillation; catheter ablation; pulmonary vein

Introduction and Background

The most prevalent of all sustained cardiac arrhythmias, atrial fibrillation (AF) has long been among the most challenging to treat. One of the first applications for catheter ablation therapy was for ventricular rate control, using atrioventricular junctional ablation [1]. For many patients, maintenance of sinus rhythm is the goal of therapy rather than simply ventricular rate control. Though drug therapy can control symptoms in many individual patients, those who enjoy nearly complete freedom from AF episodes while incurring no significant medication side effects are the exception rather than the rule. This unsatisfactory outcome for many AF patients led to exploration of non-pharmacologic therapies, among the first of which was the surgical maze procedure [2]. This offered the first true cure from AF but at the expense of open heart surgery. Subsequently, the surgeon’s atrial incisions were translated into the realm of catheter ablation [3]. This procedure prevented AF episodes but entailed very long procedure duration and fluoroscopic exposure as well as a high rate of stroke. Less cumbersome and risky modifications of this procedure were sought that could provide similar efficacy. With the recognition of “focal” AF, catheter ablation approaches have shifted from attempting to produce linear lesions to affect the substrate for maintenance of AF to more focal ablation to eliminate triggers of AF. The purpose of this chapter is to review the current strategies for choosing targets for curative AF ablation.

Atrial Fibrillation: A Focal or Global Problem?

Early detailed intraoperative mapping studies of sustained AF suggested that it was the result of several simultaneous wave fronts moving across the atrial surface in a seemingly random fashion [4,5]. This implied that AF was a global arrhythmia, requiring participation of most if not all of the atrial myocardium. Interruption of the atrial architecture with multiple incisions, depriving wavelets of sufficient functional mass to support fibrillation, was part of the design of the surgical maze procedure [6]. However, during some catheter-based maze procedures, some investigators observed that, as ablation lines approached the pulmonary vein (PV) ostia, atrial recordings during AF became more organized, slowed and finally terminated [3]. This helped focus attention on the PVs as having a possible role in the genesis or maintenance of AF [7,8]. Subsequent experience has confirmed the importance of PV sources of AF either as a focal “continuous driver” or “trigger” of AF. As a continuous driver, rapid discharges from a PV focus (or small circuit) produce AF by stimulating adjacent atrium more rapidly than 1:1 propagation can be maintained. AF persists only as long as the driver continues to depolarize rapidly. As a trigger of AF, single premature depolarizations or short bursts of rapid discharges merely initiate AF that is then maintained by other mechanisms. In either case, the ultimate source of AF is depolarization of atrial muscle fibers that extend for variable distances either longitudinally or in a spiraling fashion from left atrium (LA) onto the PV (Fig. 1) [9–11]. Clinical experience has shown that the vast majority of individuals with paroxysmal AF in the absence of significant structural heart disease have a PV focus of AF; other sources have been described in the left atrium, including the ligament of Marshall and posterior intervenous wall, as well as right atrial (RA) structures (crista terminalis, superior vena cava).

Ablation Strategies, Selection of Targets and Endpoints of Ablation

Several different strategies for AF ablation are available and, insofar as possible, should be matched to the individual patient’s situation (Table 1). Having selected a strategy, it is important to be able to determine when one has done enough ablation to achieve the desired endpoint; different ablation strategies have their own endpoints. Left atrial access is required in almost all cases, and can be achieved by probing the atrial septum gently for a patent foramen ovale or performing septal puncture with a standard Brockenbrough needle and sheath assembly. When multiple LA catheters are required, a double septal puncture can be used or a 2nd catheter can be maneuvered through the puncture site either alongside the first sheath, or a guidewire in the LA after the sheath has been withdrawn into the RA. Heparin is administered once LA access has been secured, in order to maintain activated clotting times 230–300 sec. Since ablation in the region of the PVs is typically necessary, it is useful (but not essential) to image the PVs in some manner to define the number, location and size of their ostia. This can be accomplished by pre-procedure high resolution CT scanning or with contrast opacification of each individual vein once catheters are in the LA. There is some evidence that AF is more likely to originate from larger veins and it has long been recognized that superior veins are more arrhythmogenic than inferior ones [12].

1. Focal ablation for AF initiated by frequent atrial premature complexes or short bursts of atrial tachycardia. For patients with very frequent simple atrial premature complexes (APCs) or short bursts of atrial tachycardia (AT; Fig. 2), mapping and ablation of the APC or AT focus should be employed. As noted, these are typically in or near the ostia of PVs. If the patient is sedated during the procedure, APCs or AT episodes may be suppressed; bursts of rapid atrial pacing or extrastimuli, administration of isoproterenol or adenosine, or withdrawal of sedation may enhance spontaneous ectopic beats and facilitate mapping. Choice of where to position the mapping catheter can be guided by the P wave morphology during ectopic beats [13], though this is often difficult to ascertain since the P wave is usually in the T wave of the prior QRS complex. The electrogram at the optimal ablation site is usually

fragmented during APC or AT and precedes the onset of surface P wave by >40 ms; the unipolar recording often has a “QS” configuration (Fig. 3). Pacing at the putative site of ablation should replicate the surface P wave and intracardiac atrial activation sequence of spontaneous APCs or AT (Fig. 4). High-output pacing can be used to ensure that the phrenic nerve will not be damaged during ablation. Ablation should be performed as close to the PV ostium as possible to avoid risk of PV stenosis with a maximum power setting of 25–30 W and temperature limit of 50–55°C. During radiofrequency energy delivery, an increase in APC frequency or rapid AT from the focus is observed that often slows prior to cessation. This situation is ideal in that not only does the abundance of spontaneous arrhythmia facilitate mapping, but also it is usually obvious when the endpoint—elimination of the focus—has been achieved, since ectopic activity is no longer present. It is controversial as to whether one should completely electrically isolate the PV (see below) from which the focus arose, as well as whether to address other PVs unless there is evidence they may also be arrhythmogenic. One may consider performing isolation of other PVs in patients taking amiodarone in whom there has not been adequate time prior to the procedure for washout of drug effect (which may be suppressing foci in veins other than the targeted one).

This method can also be used in some patients who have sustained AF episodes, by car-dioverting to sinus once catheters are in position and waiting for spontaneous APCs or AT episodes to reinitiate AF (early reinitiation of AF, or ERAF, after cardioversion) (Fig. 3) [14]. RF delivery at the correct location may terminate an AF episode (Fig. 5).

2. PV isolation for paroxysmal AF in absence of frequent APCs or AT. Although patients with frequent APCs or AT bursts provide an ideal mapping and ablation situation, they are unfortunately uncommon. More typically, patients with paroxysmal AF have insufficient frequency of ectopic beats to be able to map adequately or ascertain when ablation has eliminated the focus. In this situation, while it is reasonable to assume that PV foci are responsible, it is not currently possible to know which vein or which PV fascicle within any one vein is the culprit. Thus, the technique of electrically isolating the vein has been developed. Two general methods have been used to achieve this: electrical and electroanatomic.
 - a. Electrical mapping. This method of isolating PV from LA requires 2 LA catheters, the ablation catheter and a circular multipolar mapping catheter situated in the ostium of a PV near the venoatrial junction with electrodes arranged around the circumference of the ostium (Fig. 6) [15]. The pattern of activation of PV potentials during sinus rhythm or coronary sinus/LA pacing indicates the electrode(s) closest to the point(s) of electrical connection between LA and PV fascicles (Fig. 7). Multiple electrodes having nearly identical PV potential activation times of suggests a wide band of electrical connection. Unipolar “QS” configurations [16] and reversal of polarity in adjacent bipolar electrograms [17] have also been used to select the optimal target site. Ablation on the atrial aspect of this electrode results in either elimination of PV potentials (Fig. 7) or a shift in the activation pattern (Fig. 8) as a preferential connection is ablated (LA-PV block). Most PVs have 2 or more such connections with LA muscle that require ablation. Once a PV has been electrically disconnected from the LA, the circular catheter can be moved to another PV and the mapping and ablation process repeated till all veins have been assessed and ablated if necessary. A 3-dimensional mapping system can be used to catalog the location from which PV potentials are recorded in the PV prior to ablation for subsequent

comparison. Endpoints of ablation with this technique are (1) inability to record PV potentials at the PV ostium; (2) dissociated PV potentials or ongoing AT/AF in the PV without spread to the LA; or (3) inability to record PV potentials deeper within the vein at the same sites from which they were recorded prior to ablation [15]. Since ablation on the atrial aspect eliminates LA-to-PV conduction, and yet AF results from PV-to-LA conduction, some investigators have suggested pacing within the PV following presumed isolation to demonstrate PV-to-LA block [18]. However, this may not be definitive in that one may not be able to capture PV uniquely (often high threshold, with stimuli capturing far-field LA) and if 2 independent LA-PV connections exist that do not communicate with each other within the vein, one could have been ablated while the other was not but is not manifested since if it was not captured by pacing.

Following successful isolation, attempts can be made to reinitiate AF to search for and ablate non-PV sources. Pacing (burst or extrastimuli) as well as isoproterenol can be used for this purpose.

Electrical PV isolation is less well-suited for ablation during AF episodes when sinus rhythm cannot be maintained, since activation sequences around PV ostia cannot be ascertained during AF. Despite this, isolation can be successfully performed during persistent AF; a larger number of RF applications is typically necessary than when delivered during sinus rhythm. Termination of AF during ablation suggests that the vein undergoing ablation was important for continuation of AF [18,19].

- b. Electroanatomic mapping. An alternative method to activation mapping is to encircle PV ostia with ablation lesions well on the atrial aspect, assuring continuity of the lines by cataloging the lesion locations with a 3-dimensional mapping system [20–22]. An empiric endpoint of ablation at an individual site is an 80% decrease in electrogram amplitude. Following formation of a completely encircling line, one should be unable to record propagated activation from within the encircled area when pacing from outside the line. However, either recording of only low amplitude (<0.1 mV) signals inside the line or a conduction time across the line (double potentials) of >30 ms are acceptable endpoints. Since activation sequences are of lesser importance with this method than when using pure electrical isolation, it can be applied during AF episodes [23].
3. Additional methods. The previously-discussed methods of ablation provide good results in 60–90% of healthy individuals with little or no structural heart disease. Patients with more extensive atrial disease (especially markedly dilated atria) may also respond well to these methods [24], but may also require maze-like linear energy applications (compartmentalization) or adjunctive administration of antiarrhythmic drugs to improve results. A variety of empirical arrangements of ablation lines have been used without clear superiority of one over another [25]. While long linear lesions had been used by several groups prior to recognition of the importance of PVs in initiation and/or maintenance of AF, these techniques entail long procedure times and perhaps fluoroscopy exposure and effect more damage in the left atrium. The latter may possibly predispose to an increased thromboembolic risk and impaired atrial mechanical function [26]. Finally, the efficacy of linear ablation has been <50% [27]. For these reasons, linear ablation techniques are generally reserved to treat patients after an initial isolation procedure has failed to prevent recurrent AF during follow-up [25].

Anticoagulation is often resumed and maintained for several weeks following ablation, to prevent thromboembolic events related to ablation lesions as well as atrial stasis (in the presence of longstanding AF prior to ablation) until spontaneous restoration of mechanical atrial function.

Pitfalls of Mapping

Although most procedures can proceed toward recognizable endpoints, however tedious to achieve, the process has potential pitfalls. Among these are far-field atrial signals that can cause confusion during mapping and ablation. For instance, it may appear that APCs are arising in the posterior cephalad portion of the RA when in fact they arise from the right superior PV (just posterior to this portion of the RA; Fig. 9). In addition, far-field signals may simulate persistence of PV potentials when they may no longer actually be present [28]. LA appendage signals or those from an adjacent PV may suggest persistent PV potentials when mapping in the left upper or lower PVs, and signals from the posterior RA may mimic persistent potentials from the right upper PV. A significantly over- or under-sized circular mapping catheter may fit poorly in a PV ostium, causing either poor distinction of PV potentials from LA signals, or causing ablation to be performed more distally in the PV than is desirable, respectively. The latter has the potential for missing focal sources of AF within the portion of vein proximal to such an ablation line as well as causing PV stenosis.

Outcomes of Ablation

Procedural Success

Freedom from recurrent AF has been reported in up to 80% of patients who underwent limited ablation for a presumed single focus of AF (isolated frequent APCs or bursts of AT that initiated AF) as well as healthy individuals who underwent complete PV isolation for paroxysmal or persistent AF [7,8,14,15,22,24,29]. Fifty to 60% of patients with more permanent AF in the presence of significant structural heart disease have freedom from AF following electrical or electroanatomic isolation techniques. Despite these numbers derived from studies of relatively large groups of patients, for any given patient, clinical “success” ranges from complete lack of any recurrence of AF in the absence of adjunctive antiarrhythmic drug therapy, to a moderate decrease in the frequency or duration of episodes or rendering the arrhythmia more responsive to antiarrhythmic drugs.

Up to 40% of patients experience transient AF episodes following an apparently successful ablation procedure [30]; in some, AF is more frequent and problematic in this setting than prior to ablation. Some electrophysiologists maintain patients on antiarrhythmic drug therapy for a few weeks post-procedure because of this. In most cases, AF episodes gradually subside over the ensuing weeks. After the first 2 months post-ablation, recurrence of AF following a PV isolation procedure may be due to incomplete PV isolation, recovery of conduction from PV to LA, or the presence of non-PV sources of AF. Antiarrhythmic drug therapy may confer additional efficacy following ablation, even when the same drugs had minimal beneficial effect prior to ablation.

Procedural Complications

The extended duration of these procedures as well as the requirement for left atrial access and its attendant aggressive anticoagulation makes AF ablations among the more risky electrophysiologic procedures. Up to 1% of patients experience significant pericardial effusion or cardiac tamponade requiring drainage [23,31,32]; in most series <1% of patients suffer myocardial infarction, stroke or death, although a recent report suggested a higher incidence of stroke, especially among elderly patients [33]. Damage to atrioventricular conduction or valves is also possible. Stenosis of PVs has been reported in up to 5% of patients [15,29,34];

using current techniques appears to have decreased the incidence of this complication (ablation on the atrial aspect of the venoatrial junction instead of within the vein [23,32]).

Summary

Ablation therapy for AF has come a long way from simply the ultimate solution for ventricular rate control. A wide range of patients can now be considered as candidates for one of several AF ablation strategies. The importance of pulmonary venous triggers of AF, regardless of the clinical setting of AF, has become increasingly apparent as experience with AF ablation has broadened. Different methods of ablation (electrical vs electroanatomic) appear to yield good, but imperfect results in preventing AF recurrences. Although multiple PVs may be able to initiate AF episodes in individual patients, it may not be necessary to entirely eliminate all connections between atrium and PVs in all patients; for example, electroanatomic mapping and ablation delays, but does not necessarily completely block, conduction from atrium into PV and yet has a high success rate. Thus, determining how much ablation is necessary to be effective remains an important question. New methods of quickly, safely and reliably eliminating culprit PV sources of AF [35], as well as locating and ablating non-PV AF triggers, will likely extend curative ablation therapy to an ever-increasing population of patients with this very common arrhythmia.

References

1. Scheinman MM, Morady F, Hess DS, Gonzalez R. Catheter-induced ablation of the atrioventricular junction to control refractory supraventricular arrhythmias. *JAMA* 1982;248:851–855. [PubMed: 7097946]
2. Cox JL, Schuessler RB, D'Agostino HJ Jr, Stone CM, Chang BC, Cain ME, Corr PB, Boineau JP. The surgical treatment of atrial fibrillation. III. Development of a definitive surgical procedure. *J Thorac Cardiovasc Surg* 1991;101:569–583. [PubMed: 2008095]
3. Swartz JF, Pellersels G, Silvers J, Patten L, Cervantez D. A catheter-based curative approach to atrial fibrillation in humans (abstract). *Circulation* 1994;90:I-335.
4. Ferguson TB, Schuessler RB, Hand DE, Boineau JP, Cox JL. Lessons learned from computerized mapping of the atrium. Surgery for atrial fibrillation and atrial flutter. *J Electrocardiol* 1993;26:210–219. [PubMed: 8189128]
5. Konings KTS, Kirchhof CJHJ, Smeets JRLM, Allesie MA. High density mapping of electrically induced atrial fibrillation in man. *Circulation* 1994;89:1665–1680. [PubMed: 8149534]
6. Cox JL, Canavan TE, Schuessler RB, Cain ME, Lindsay BD, Stone C, Smith PK, Corr PB, Boineau JP. The surgical treatment of atrial fibrillation. II. Intraoperative electro-physiologic mapping and description of the electrophysiologic basis of atrial flutter and atrial fibrillation. *J Thorac Cardiovasc Surg* 1991;101:406–426. [PubMed: 1999934]
7. Haïssaguerre M, Jaïs P, Shah DC, Takahashi A, Hocini M, Quiniou G, Garrigue S, Le Mouroux A, Le Metayer P, Clémenty J. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998;339:659–666. [PubMed: 9725923]
8. Chen SA, Tai CT, Tsai CF, Hsieh MH, Ding YA, Chang MS. Radiofrequency catheter ablation of atrial fibrillation initiated by pulmonary vein ectopic beats. *J Cardiovasc Electrophysiol* 2000;11:218–227. [PubMed: 10709719]
9. Nathan H, Eliakim M. The junction between the left atrium and the pulmonary veins. An anatomic study of human hearts. *Circulation* 1966;34:412–422. [PubMed: 5922708]
10. Tagawa M, Higuchi K, Chinushi M, Washizuka T, Ushiki T, Ishihara N, Aizawa Y. Myocardium extending from the left atrium onto the pulmonary veins: A comparison between subjects with and without atrial fibrillation. *Pacing Clin Electrophysiol* 2001;24:1459–1463. [PubMed: 11707038]
11. Ho SY, Cabrera JA, Tran VH, Farré J, Anderson RH, Sanchez-Quintana D. Architecture of the pulmonary veins: Relevance to radiofrequency ablation. *Heart* 2001;86:265–270. [PubMed: 11514476]

12. Yamane T, Shah DC, Jaïs P, Hocini M, Peng JT, Deisenhofer I, Clémenty J, Haïssaguerre M. Dilatation as a marker of pulmonary veins initiating atrial fibrillation. *J Interv Card Electrophysiol* 2002;6:245–249. [PubMed: 12154327]
13. Tse HF, Lau CP, Lee KL, Pelosi F, Oral H, Knight BP, Strickberger SA, Morady F. P wave polarity during pacing in pulmonary veins. *J Interv Card Electrophysiol* 2001;5:167–172. [PubMed: 11342753]
14. Lau CP, Tse HF, Ayers GM. Defibrillation-guided radiofrequency ablation of atrial fibrillation secondary to an atrial focus. *J Am Coll Cardiol* 1999;33:1217–1226. [PubMed: 10193719]
15. Haïssaguerre M, Jaïs P, Shah DC, Garrigue S, Takahashi A, Laverne T, Hocini M, Peng JT, Roudaut R, Clémenty J. Electrophysiological end point for catheter ablation of atrial fibrillation initiated from multiple pulmonary venous foci. *Circulation* 2000;101:1409–1417. [PubMed: 10736285]
16. Tada H, Oral H, Wasmer K, Greenstein R, Pelosi F Jr, Knight BP, Strickberger SA, Morady F. Pulmonary vein isolation: Comparison of bipolar and unipolar electrograms at successful and unsuccessful ostial ablation sites. *J Cardiovasc Electrophysiol* 2002;13:13–19. [PubMed: 11843477]
17. Yamane T, Shah DC, Jaïs P, Hocini MM, Deisenhofer I, Choi KJ, Macle L, Clémenty J, Haïssaguerre M. Electrogram polarity reversal as an additional indicator of breakthroughs from the left atrium to the pulmonary veins. *J Am Coll Cardiol* 2002;39:1337–1344. [PubMed: 11955852]
18. Gerstenfeld EP, Dixit S, Callans D, Rho R, Rajawat Y, Zado E, Marchlinski FE. Utility of exit block for identifying electrical isolation of the pulmonary veins. *J Cardiovasc Electrophysiol* 2002;13:971–979. [PubMed: 12435181]
19. Oral H, Knight BP, Ozaydin M, Chugh A, Lai SW, Scharf C, Hassan S, Greenstein R, Han JD, Pelosi F Jr, Strickberger SA, Morady F. Segmental ostial ablation to isolate the pulmonary veins during atrial fibrillation: Feasibility and mechanistic insights. *Circulation* 2002;106:1256–1262. [PubMed: 12208802]
20. Pappone C, Oreto G, Lamberti F, Vicedomini G, Loricchio ML, Shpun S, Rillo M, Calabro MP, Conversano A, Ben-Haim SA, Cappato R, Chierchia S. Catheter ablation of paroxysmal atrial fibrillation using a 3D mapping system. *Circulation* 1999;100:1203–1208. [PubMed: 10484541]
21. Pappone C, Rosanio S, Oreto G, Tocchi M, Gugliotta F, Vicedomini G, Salvati A, Dicandia C, Mazzone P, Santinelli V, Gulletta S, Chierchia S. Circumferential radiofrequency ablation of pulmonary vein ostia: A new anatomic approach for curing atrial fibrillation. *Circulation* 2000;102:2619–2628. [PubMed: 11085966]
22. Pappone C, Santinelli V. Pulmonary vein isolation by circumferential radiofrequency lesions in atrial fibrillation. From substrate to clinical outcome. *Ann Ist Super Sanita* 2001;37:401–407. [PubMed: 11889957]
23. Pappone C, Oreto G, Rosanio S, Vicedomini G, Tocchi M, Gugliotta F, Salvati A, Dicandia C, Calabro MP, Mazzone P, Ficarra E, Di Gioia C, Gulletta S, Nardi S, Santinelli V, Benussi S, Alfieri O. 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. [PubMed: 11714647]
24. Haïssaguerre M, Jaïs P, Shah DC, Arentz T, Kalusche D, Takahashi A, Garrigue S, Hocini M, Peng JT, Clémenty J. Catheter ablation of chronic atrial fibrillation targeting the reinitiating triggers. *J Cardiovasc Electrophysiol* 2000;11:2–10. [PubMed: 10695453]
25. Packer, DL. Linear ablation for atrial fibrillation: The pendulum swings back. In: Zipes, DP., editor. *Catheter Ablation of Arrhythmias*. 2. Armonk, NY: Futura; 2001. p. 107-129.
26. Thomas SP, Nicholson IA, Nunn GR, Rees A, Trieu L, Daly MP, Wallace EM, Ross DL. Effect of atrial radiofrequency ablation designed to cure atrial fibrillation on atrial mechanical function. *J Cardiovasc Electrophysiol* 2000;11:77–82. [PubMed: 10695466]
27. Natale A, Leonelli F, Beheiry S, Newby K, Pisano E, Potenza D, Rajkovich K, Wides B, Cromwell L, Tomassoni G. Catheter ablation approach on the right side only for paroxysmal atrial fibrillation therapy: Long-term results. *Pacing Clin Electrophysiol* 2000;23:224–233. [PubMed: 10709231]
28. Shah D, Haïssaguerre M, Jaïs P, Hocini M, Yamane T, Macle L, Choi KJ, Clémenty J. Left atrial appendage activity masquerading as pulmonary vein potentials. *Circulation* 2002;105:2821–2825. [PubMed: 12070107]

29. Haïssaguerre, M.; Shah, DC.; Jaïs, P.; Hocini, M.; Yamane, T.; Choi, KJ.; Garrigue, S.; Clémenty, J. Catheter ablation of atrial fibrillation: Targeting the triggers. In: Haïssaguerre, M.; Zipes, DP., editors. Catheter Ablation of Arrhythmias. 2. Armonk, NY: Futura; 2002. p. 89-105.
30. Oral H, Knight BP, Ozaydin M, Tada H, Chugh A, Hassan S, Scharf C, Lai SW, Greenstein R, Pelosi F Jr, Strickberger SA, Morady F. Clinical significance of early recurrences of atrial fibrillation after pulmonary vein isolation. *J Am Coll Cardiol* 2002;40:100–104. [PubMed: 12103262]
31. Oral H, Knight BP, Tada H, Ozaydin M, Chugh A, Hassan S, Scharf C, Lai SW, Greenstein R, Pelosi F Jr, Strickberger SA, Morady F. Pulmonary vein isolation for paroxysmal and persistent atrial fibrillation. *Circulation* 2002;105:1077–1081. [PubMed: 11877358]
32. Marrouche NF, Dresing T, Cole C, Bash D, Saad E, Balaban K, Pavia SV, Schweikert R, Saliba W, Abdul-Karim A, Pisano E, Fanelli R, Tchou P, Natale A. 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. [PubMed: 12142112]
33. Kok LC, Mangrum JM, Haines DE, Mounsey JP. Cerebrovascular complication associated with pulmonary vein ablation. *J Cardiovasc Electrophysiol* 2002;13:764–767. [PubMed: 12212693]
34. Robbins IM, Colvin EV, Doyle TP, Kemp WE, Loyd JE, McMahon WS, Kay GN. Pulmonary vein stenosis after catheter ablation of atrial fibrillation. *Circulation* 1998;98:1769–1775. [PubMed: 9788832]
35. Lesh MD, Diederich C, Guerra PG, Goseki Y, Sparks PB. An anatomic approach to prevention of atrial fibrillation: Pulmonary vein isolation with through-the-balloon ultrasound ablation (TTB-USA). *Thorac Cardiovasc Surg* 1999;47(Suppl 3):347–351. [PubMed: 10520766]

Fig 1. Left atrium with pulmonary veins viewed from posteriorly. The shaded area represents presence of muscle fibers that extend for variable distances onto the pulmonary veins. Dashed lines represent the anatomic demarcation between atrium and vein (adapted from Nathan et al. [9]).

Fig 2.

Spontaneous, incessant bursts of atrial tachycardia that initiate atrial fibrillation. Shown are surface ECG leads 1, 2, 3, V1 and V6 with intracardiac recordings from a catheter in the coronary sinus (CS). Arrows indicate atrial premature complexes or bursts of atrial tachycardia that eventually initiated fibrillation; the surface ECG suggests fibrillation during this rapid tachycardia. Abbreviations: prox = proximal, mid = middle, dist = distal.

Fig 3.

Mapping atrial premature complexes that initiate atrial fibrillation. Recordings similar to Fig. 2. HRA = high right atrium, LUPV = left superior pulmonary vein, uni = unipolar recording, RVA = right ventricular apex. The recordings were made 5 sec following cardioversion for sustained episode of atrial fibrillation; fibrillation recurs, initiated by a premature complex (onset of which is denoted by the dashed line) that originated at the ostium of the left superior pulmonary vein (signified by a sharp deflection in the bipolar electrogram occurring 70 ms before P wave onset and unipolar “QS” configuration).

Fig 4.

Pacemapping to clarify source of initiating atrial premature complexes (APC). Recordings similar to prior figures; TA = tricuspid annulus (patient also had flutter for which 20-pole TA catheter was used), Abl = ablation catheter. Left panel shows 2 sinus rhythm beats followed by an APC (arrow; P wave onset at dashed line) that initiates atrial fibrillation. The ablation catheter was in the right superior pulmonary vein (RSPV). Previously, mapping had suggested the superior vena cava (SVC) as the APC origin. Pacing at the SVC site yielded the atrial activation pattern shown in the middle panel; pacing in the RSPV shown in the right panel. The shaded areas are an overlay of the atrial activation sequence of the APC. It is evident that the paced activation pattern from the RSPV is nearly identical to that of the APC, while significant differences exist between the APC pattern and that of SVC pacing (just anterior to the RSPV site).

Fig 5.

Termination of atrial fibrillation during radiofrequency delivery to left upper pulmonary vein (LUPV) ostium. Recordings as in prior figures. Fibrillation is shown at left (an episode had been continuous for the prior 30 min) during ablation at a site near the LUPV ostium. Several seconds after the onset of energy delivery, fibrillation ceases and sinus rhythm resumes.

Fig 6. Left atrial-pulmonary vein junction showing circular mapping catheter with electrodes arrayed around vein ostium and ablation catheter on the atrial aspect. Strands of atrial muscle are seen extending out into the vein.

Fig 7.

Pulmonary vein (PV) potentials recorded in a circular mapping catheter (Lasso) at the PV ostium. Recordings as in prior figures. All 10 bipoles comprised of adjacent electrode pairs of the circular mapping catheter are shown. Far-field, low amplitude left atrial (LA) potentials precede the near-field PV potentials during sinus rhythm prior to ablation. The polarity of the initial portion of the electrogram reverses direction between recordings 1-2 and 10-1. Following one ablation attempt near electrode 1 (at site of Abl_{dist} recording), PV potentials are no longer seen. A single PV-LA connection was thus present.

Fig 8.

Multiple connections between pulmonary vein (PV) and left atrium. Recordings as in prior figures. S = stimulus from distal coronary sinus. In left panel, prior to 1st radiofrequency (RF) delivery, sharp PV potentials are present in several recordings (dark arrows). Following ablation near electrode 10, a different activation pattern is present; PV potentials are absent in recordings 10-1 and 1-2 while potentials in 5-6 through 8-9 are unchanged in both timing and morphology. Additional ablation near electrode 5 eliminates all PV potentials. Thus, 2 PV-LA connections were present.

Fig 9.

Right atrial recordings mimicking good ablation site in a patient with frequent atrial premature complexes (APC) that initiated atrial fibrillation episodes. Recordings as in prior figures. At top, the ablation catheter is in the high posterior right atrium; the first and last beats are sinus rhythm, showing a split potential with a smaller late component. During APCs (middle 2 beats), the components are reversed with the smaller potential appearing first. Ablation at this site transiently suppressed APCs. In the bottom panel, after accessing the left atrium and positioning the catheter in the right superior pulmonary vein (just behind the right atrial site), the delayed potential during sinus beats (middle 2) is much larger and the earlier potential much smaller, and during APCs the sharp potential occurs earlier in relation to the APC, compared to the right atrial site. Ablation at this site was successful. Thus, the right atrial signals were far-field recordings from the true site of APC origin in the pulmonary vein.

Table 1

Strategies for ablation of atrial fibrillation

Clinical setting	Ablation target(s)	Endpoint
Frequent isolated APC causing AF episodes*	APC focus (typically in PV)	Elimination of focal discharge (no APC)
Frequent bursts of AT causing AF episodes*	AT focus (typically in PV)	Elimination of focal discharge (no AT)
Rare paroxysms of AF	PV isolation: electrical PV isolation: electro-anatomic	Electrical disconnection of PV from atrium Decrease in electrogram amplitude or increase in conduction time
Permanent AF	PV isolation (as above) Search for non-PV triggers Possible limited maze	PV isolation (as above) Elimination of focal discharge (no non-PV ectopy) Compartmentalization of targeted atrium

APC: atrial premature complex; AF: atrial fibrillation; PV: pulmonary vein; AT: atrial tachycardia.

* Spontaneous or provoked following pacing or cardioversion or administration of isoproterenol, adenosine, etc.