Evolving Strategies in Catheter Ablation of Long-Standing Atrial Fibrillation

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Since its original description in 1998, the technique of catheter-based atrial fibrillation (AF) ablation has undergone several modifications.1 Currently, many operators use an anatomic approach, consisting of circumferential lesions encircling individual or ipsilateral pulmonary veins (PVs), with additional empirical left atrial ablations (lines), whereas others perform a more PV-specific approach, using entrance and exit block to validate isolation, deferring any additional non-PV lesions, unless clinically indicated.2–7 Despite these differences in technique, there remain remarkable consistencies in the AF outcome data between centers, with overall single-procedure efficacy of ≥70% in achieving long-term arrhythmia control for patients with paroxysmal AF but significantly lower success rates in achieving a similar outcome for patients with persistent or permanent AF. The obvious implications of these observations are that the mechanisms underlying initiation and maintenance of persistent or permanent AF may extend beyond PVs. Although this remains an area of active discussion and debate, it has nevertheless prompted investigators to explore more extensive ablation strategies in these patients.8–10

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Consistent with this trend, in this issue of Circulation: Arrhythmia and Electrophysiology, Rostock et al11 report their observations in patients with chronic AF. This was a single-center observational study in which 88 subjects underwent ablation during AF, using a step-wise approach comprising PV isolation followed by extensive left atrial, coronary sinus, and right atrial ablation, targeting areas manifesting continuous or rapid electric activity, local activation gradients, or centrifugal activation patterns. The end point of lesion creation was alteration in local electrogram characteristics, and the procedure end point was restoration of sinus rhythm, unless procedure duration exceeded 6 hours or the patient received ≥5 L of fluid, in which case AF was cardioverted. Patients were subsequently followed closely, with frequent Holter monitoring and clinic visits. Reablation was the preferred treatment modality for any arrhythmia recurrence >3 months after the index procedure. The aims of the study were to evaluate acute success of this strategy in terminating AF (to sinus rhythm or organized, fast electric activity) and the prognostic value of such termination on long-term freedom from AF. During the initial ablation, AF terminated in 68 patients, but only in a minority (n=8) was this to sinus rhythm. In the majority (n=60), AF organized to atrial tachycardias, requiring additional lesions, despite which 22 patients had to eventually be cardioverted. Fifty-four patients experienced arrhythmia recurrence (AF: n=23; organized atrial tachycardias: n=31), and this was equally manifest in subjects with and without AF termination at index procedure, although in the former, recurrent arrhythmias were predominantly organized atrial tachycardias (75 reentrant and 18 focal). Interestingly, in the majority of these patients, previously isolated veins had reconnected. The investigators do not report on the residual scar burden from previously applied non-PV lesions. Thus, the long-term single-procedure efficacy of this extensive ablation strategy for maintaining sinus rhythm was 38%. With ≥2 additional ablations, >80% of patients were free from AF.

So, what can we infer from these data? First and foremost, by using the technique of step-wise AF ablation, which was originally described by Haissaguerre et al,9 and reporting results nearly identical to the Bordeaux experience,12 Rostock et al11 make a strong case for the reproducibility of this approach. So, is a more extensive ablation strategy the only way to cure patients with chronic AF, and is this arrhythmia indeed a bialtrial rhythm disorder? To answer these in the affirmative would imply that the mechanisms underlying chronic AF are different from those underlying paroxysmal AF. As a prelude to this discussion, it is worthwhile to reiterate that using “chronic” AF interchangeably with persistent or permanent AF may be inaccurate, because even paroxysmal AF that exists for long durations would qualify as chronic. The correct nomenclature to characterize AF behavior is the 3 Ps: paroxysmal, persistent and permanent AF.13 Furthermore, as per the Heart Rhythm Society/European Heart Rhythm Association/European Cardiac Arrhythmia Society Expert Consensus Statement, the latter category may not be appropriate in the context of patients undergoing AF ablation, and the recommendations are to use the terminology “longstanding persistent AF” for this group instead.14 AF typically starts as a paroxysmal rhythm, and if not treated at this stage, it can evolve into a persistent or permanent state. This transition has been attributed to the process of atrial electric remodeling.15 Early in the natural history of AF, triggers predominate, and these are usually limited to the PVs.1 As the arrhythmia becomes established, the remodeled atria become more vulnerable to AF, likely the result of favorable substrate or diverse and widespread triggers.16
Obviously, an extensive ablation strategy could potentially address all these possible mechanisms. However, even a limited-ablation approach targeting PVs alone has demonstrated single-procedure efficacy rates of >50% in patients with persistent and permanent AF, suggesting that in a large population of these patients, PVs and posterior left atria still play a critical role. This point of view is further reinforced by the observation that in patients who recur after the initial limited-ablation approach, reconnection across previously isolated veins is ubiquitous, and repeat PV isolation alone is sufficient to achieve subsequent long-term AF control in the majority. Nevertheless, there certainly is a subgroup of patients with persistent or permanent AF in whom mechanisms underlying initiation and maintenance of the arrhythmia may extend beyond PVs and posterior left atria. However, defining this patient subset and identifying the culprit extra PV sites remain challenging. In this study, Rostock et al have made an effort to address both of these issues. They report unique electrogram characteristics at specific sites that were associated with AF termination and also identify certain patient and arrhythmia characteristics that were predictors of long-term success. Both of these observations merit further discussion. AF termination, when targeting specific sites and electrograms, may have mechanistic implications. However, important concerns when using the qualitative appearance of local electrograms for determining appropriate targets during ongoing AF include variability in their spatio-temporal profile and subjective inconsistencies of interpretation between operators. Also, the observation of AF termination at a particular site during nonrandom step-wise ablation may not necessarily be proof of that site’s critical participation in AF maintenance but may simply reflect “cumulative” benefit from all prior sites that had already been targeted (ie, substrate modification). As far as patient and arrhythmia characteristics are concerned, an interesting observation in this study was the association between AF duration and time and number of lesions required for AF termination. Thus, AF termination was achieved earlier in patients with a shorter AF duration (<6 months). The obvious implication of this observation is that patients who are relatively new converts to persistent AF may not have remodeled their atria sufficiently and may be more amenable to arrhythmia termination with fewer extra PV lesions. It can also be argued, though, that in this subgroup, a limited-ablation approach may have been equally efficacious. Thus, a criticism of this study design is the lack of a control arm (ie, patients undergoing ablation using a limited approach), which would have allowed us to make this comparison. Nevertheless, the 6-month AF duration cutoff is an important variable that the electrophysiology community may find useful in determining the ablation approach they choose for their patients.

A final note of caution when analyzing the results of this study pertains to interpretation of arrhythmia recurrence. Redo procedure rates in the current study were >60%, and some of these patients needed >2 procedures. This is comparable to the redo procedure rates seen in patients with persistent or permanent AF who are undergoing PV isolation alone. Investigators favoring extensive ablation strate-


gies would argue that recurrent arrhythmias in their series are predominantly atrial tachycardias and not AF. This is certainly consequential as far as arrhythmia mechanisms are concerned, although the flip side of this argument can be that the extensive ablation strategy actually promotes development of these arrhythmias and may be proarrhythmic. Regardless of this discussion, from the patient’s perspective, organized atrial arrhythmias can be just as symptomatically debilitating as AF, and repeat ablation for these can be just as time consuming as a redo AF ablation. Other concerns with extensive ablation strategies include risk of collateral damage to surrounding structures (eg, circumflex artery, phrenic nerve) as well as their long-term impact on atrial transport function and coronary sinus patency.

Despite these concerns, this study by Rostock et al is an important contribution to the emerging literature on the use of extensive ablation strategies for treating patients with more established forms of AF. The observation made by these investigators on the regional variation in electrogram behavior during ongoing AF and the global response of the arrhythmia when targeting these sites has mechanistic implications. These data also raise several interesting questions, all of which are welcome because they may help focus our ongoing efforts in designing future studies that can more fully explore these issues.

Disclosures

None.

References


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