EDITORIAL COMMENT

Linear Ablation for Atrial Fibrillation

Have We Come Full Circle?*

David J. Wilber, MD, FACC
Maywood, Illinois

The evolution of effective and broadly applicable strategies for percutaneous ablation of atrial fibrillation (AF) has followed a complex course over the past decade. In 1994, Swartz et al. (1) reported successful percutaneous ablation of AF with multiple long linear lesions. Conceived as a replication of the surgical MAZE, the procedure deployed specifically tailored long sheaths to facilitate fluoroscopically guided sequential radiofrequency application along a predetermined set of ablation lines in both atria. Subsequent long-term follow-up in 40 patients with chronic AF demonstrated the remarkable efficacy of this procedure (90% in sinus rhythm), but with prolonged procedure times (10 to 15 h), and a 22% incidence of major complications (including stroke, significant pericardial effusion/tamponade, and pulmonary vein [PV] occlusion) (2).

See page 1271

Other groups subsequently tested a variety of linear strategies employing the sequential application technique in attempts to simplify the procedure and reduce risks, predominantly in patients with paroxysmal AF (3–8). Several lessons emerged from that experience. Right atrial lesions alone or in combination with left linear lesions appeared to contribute little to overall procedural efficacy and were largely abandoned. During follow-up, elimination of AF without antiarrhythmic drugs occurred in 40% to 50% of patients with left linear lesions; an additional 20% had complete suppression of AF after adjunctive antiarrhythmic drug therapy. These results fell short of the 80% to 90% success rates reported for surgically based simplified MAZE procedures (9,10), as well as the original Swartz procedure. Continuous transmural lesions were technically difficult to produce, and gaps in the ablation line resulted not only in recurrent AF, but also promoted a new form of proarrhythmia, left atrial (LA) macroreentry, often requiring additional intervention. Subsequent experimental data confirmed that gaps from 2 to 5 mm were capable of permitting conduction and could facilitate LA macroreentry (11,12).

Use of irrigated catheters did not completely eliminate the problem of incomplete lines (7). Finally, the risk of major complications associated with these procedures remained substantial (12). With the recognition of venous triggers for AF initiation and maintenance by Haïssaguerre et al. (13), enthusiasm for long linear lesion strategies waned. Initial approaches targeted spontaneous focal triggers within one or more “arrhythmogenic” PVs (13,14). However, the unreliable provocation of focal firing in the laboratory, frequent recurrences necessitating repeated interventions, and a modest risk of PV stenosis led to procedural modifications whereby focal segmental ablation was performed at the LA-PV junction, with the aim of electrical disconnection of the vein from the LA (15). The use of circular recording catheters that could monitor electrograms around the circumference of the vein facilitated this approach (16). Elimination of high frequency potentials indicative of local activation from within the vein during atrial pacing (entrance block) and the inability of paced or spontaneous depolarizations within the vein to activate the LA (exit block) became easily recognized and well-defined acute end points. Extension of the segmental ostial isolation procedure to all four PVs resulted in successful elimination of paroxysmal AF in 70% to 80% of such patients during short-term follow-up, with a low risk of symptomatic PV stenosis and other major complications (17–19). The reported success of this procedure for persistent and chronic AF has been variable, but appears lower (19,20).

Subsequently, Pappone et al. (21,22) developed a PV isolation procedure based on encircling point-by-point radiofrequency lesions placed in atrial tissue outside the LA-PV junction and utilizing electroanatomical mapping. The end point of the procedure was the recording of only delayed low voltage (<0.10 mV) far-field electrograms from within the isolated area. Elimination of AF during follow-up was reported in 85% of 179 patients with paroxysmal AF and 68% of 72 patients with chronic AF. Successful outcome correlated poorly with the designated procedural end points and was best predicted by a larger percentage of total LA surface area incorporated within the circular lesions.

It is with this perspective that the manuscript of Ernst et al. (23), published in this issue of the Journal, is best understood. These investigators examined the outcome of linear radiofrequency ablation with a standard 4 mm electrode guided by electroanatomical imaging in 84 patients with predominantly paroxysmal AF. Four different strategies for lesion deployment were evaluated, with systematic use of electrophysiologic criteria to assess completeness of conduction block across the ablation line. For the initial two strategies, 1) a circular line around all four PVs with a separate line connecting the circle to the posterior mitral annulus, and 2) three separate nonencircling LA lines, complete lines were rarely accomplished and consequently elimination of AF during follow-up was uncommon. In
addition to recurrence of AF, gaps in the nonencircling lines promoted the occurrence of LA macroreentry (11% of patients).

The final two strategies: 1) separate encircling lines around the septal and lateral PVs, with an additional linear connection between these circles and the posterior mitral annulus, and 2) separate encirclement of the septal and lateral PVs alone, without additional lines, mimic more closely the method of Pappone et al. (21,22). These procedures resulted in elimination of AF in nearly two-thirds of patients during follow-up. Low voltage within the isolated area was a poor predictor of outcome. However, elimination of high frequency potentials within the isolated veins was found to be relatively specific for a favorable outcome. Inclusion of an additional long linear lesion between the PV encircling lesions and the mitral annulus failed to improve outcome and facilitated the occurrence of LA macroreentry. Two patients had PV stenosis or occlusion, raising concerns that the ostium may not have been appropriately visualized before ablation.

How should the findings of Ernst et al. (5) influence our conceptual approach to the ablation of AF? First, it is clear that isolation of the PVs remains the cornerstone for successful percutaneous ablation of AF. Second, this study confirms the previously described technical difficulties and potential proarrhythmia associated with long linear lesions in the LA. Third, encircling linear lesions placed in atrial tissue outside the PVs are a viable alternative to segmental ostial isolation at the putative LA-PV junction.

Is the inclusion of LA tissue adjacent to the ostium desirable during PV isolation? There are several reasons that this may be so. The anatomy of the venoatrial junction is complex, with both longitudinally and spirally oriented fibers from the adjacent LA entering and investing the media of the proximal PV (24–27). This region and the adjacent proximal PVs are also the site of greatest electrogram fractionation, conduction slowing, and repolarization heterogeneity, all potential substrates for reentry (28,29). Left atrial tissue adjacent to the PV is the predominant source of discrete high frequency periodic activity during AF (30). Spontaneously discharging foci are frequently found in atrial tissue near the LA-PV junction in both experimental preparations (29) and in clinical experience (19). It may be overly simplistic to envision the LA-PV junction as a discrete anatomic ring. Rather, at least functionally, it may be more useful to consider the junction as a broader band incorporating both proximal venous and adjacent LA tissue. Given the importance of this region, it is perhaps surprising that there has been little rigorous attention to the accurate assessment of the location of the LA-PV junction during clinical procedures. Contrast venography (16,17,19), intracardiac echo (31,32), and tissue impedance (21,22) have each been used to identify the PV ostium, but few attempts have been made to correlate these techniques with each other, or with histologic and electrophysiologic findings.

One attractive aspect of circular linear lesions placed deliberately several millimeters outside the putative LA-PV junction is that the impact of inaccuracies in locating this structure and leaving behind a remnant of arrhythmogenic tissue, may be minimized. In the experience of Pappone et al. (21,22), high frequency potentials were identified at 35% of LA sites thought to be adjacent to, but not within, the PV. Both Pappone et al. (22) and Ernst et al. (23) found that gaps in the peri-PV circular lesions, as demonstrated by activation or voltage mapping, did not predict procedural failure. The experience of Ernst and coworkers indicates that the elimination of high frequency potentials within the isolated area may be a better end point. Circular lesions placed outside the os (when properly identified) may also further reduce the risk of PV stenosis. Finally, targeting atrial tissue outside the LA-PV junction may permit the safe use of higher power applications that could reduce the incidence of delayed recovery of activation within the PVs, an important cause of recurrent AF after apparently successful segmental ostial isolation procedures (17,19).

Strategies for the percutaneous ablation of AF continue to evolve. It is likely that for some patients, such as those with more extensive structural abnormalities, PV isolation alone will be insufficient. However, until we are able to reliably produce durable complete isolation of the PVs and the LA-PV junction, it will be difficult to judge who may benefit from additional long linear lesions, with their attendant risk and extended procedural times, elsewhere in the atria.

Reprint requests and correspondence: Dr. David J. Wilber, Cardiovascular Institute, Loyola University Medical Center, 2160 South First Avenue, Maywood, Illinois 60153. E-mail: dwilber@lumc.edu.

REFERENCES