Iatrogenic Left Atrial Tachycardias

Where Are We?*

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Iatrogenic Left Atrial Tachycardia: Rarity or Epidemic?

Depending on initial ablative approach, the incidence of left atrial tachycardia (LAT) or flutter after atrial fibrillation (AF) ablation has varied about 10-fold. Using segmental, ostial pulmonary vein (PV) and/or antral isolation, about 3% of patients require repeat ablation for LAT (1,2). Conversely, using widely encircling PV lines plus mitral and roof lines, LAT incidence is often 20% to 30% (3,4), although outliers exist (5). Lastly, targeting complex fractionated electrograms without linear lesions or PV isolation, LAT occurred after 8.3% of ablations (6).

Iatrogenic LAT: Pathophysiological Mechanisms

Merino (7) postulated that LAT is a modification of existing AF substrate, instead of a proarrhythmic effect of AF ablation (7). However, in this issue of the Journal, Chae et al. (8) present convincing evidence that after performing circumferential PV ablation with linear lesions, discontinuities in those lines caused 96% of LAT mapped. Other groups have reinforced the critical role of such gaps (3–5,9–11).

The interplay between discontinuities in linear lesions and conduction or arrhythmia has been examined (12–14). Although gaps as wide as 5 mm may block (13), gaps as small as 1 to 2 mm can conduct (12,13,15), explaining why some re-entrant LAT can be eliminated with a single lesion (11). Discontinuities in lines may produce conduction slowing (12,13,16). Conduction depends on factors other than the gap dimension, including cycle length (12–14,16), gap and lesion geometry (14), antiarrhythmic drug effects (14), and fiber orientation (12), with block occurring at the distal side of the isthmus because of reduction of the safety factor and ultimately of wavefront curvature (12).

In addition to influencing the incidence of LAT, the initial lesion set preordains LAT type when it does occur. Thus, 8 of 10 LAT after segmental PV isolation were focal (1). Conversely, Chae et al. (8) diagnosed re-entry in 137 of 155 LAT, with 85% of these macro–re-entrant, confirming prior work (3,5).

Although gaps cause LAT (8,11), paradoxically, complete lines also provide a re-entrant substrate (3,9,11.) Thus, mitral flutter can more readily be induced after roof-line completion (9). Septal LAT occurring without antecedent ablation at that site further suggests that even complete lines in bordering regions can be proarrhythmic (8). Lastly, electrically silent, scarred left atrial zones predispose to LAT (17), and may indicate the need for more extensive ablation to cure LAT (2).

Iatrogenic LAT: ECG

An LAT characteristically displays lower inferior lead voltages versus typical flutter (17,18). Also, positive, precordial P-wave concordance suggests peri-mitral flutter (19). Electrocardiographic (ECG) patterns for PV LAT have been described (1). However, patterns overlap and right atrial flutter should be considered because after prior AF ablation, its ECG appearance is often distorted (20). Some have found re-entrant LAT to show a slightly longer cycle length than focal LAT (8), but other investigators report the opposite (19). Interestingly, CS sources often show an inferiorly directed P wave (21).

Iatrogenic LAT: Diagnosis and Ablation Strategy

Understanding the initial lesion set is critical before embarking on LAT ablation. Broadly, LAT ablation strategies include repeat PV isolation versus linear isthmus ablation. Cummings et al. (2) retreated 23 of 737 PV isolation patients for LAT using repeat PV isolation, uniformly finding PV conduction recovery in at least 1 PV. After repeat procedures, 61% were arrhythmia free.

Conversely, Chae et al. (8) targeted the flutter itself rather than only repeating PV isolation, and report the largest experience with LAT after circumferential PV ablation. Of 155 total LAT in 78 patients, 120 were re-entrant, and 115 of these were gap related, originating adjacent to a prior ablation line. Using 3-dimensional and entrainment mapping to guide radiofrequency ablation (8), acute success was remarkable at 85%. Followed up by ECG, Holter monitoring, and moderately intensive event monitoring, 77% of patients were free of LAT or AF at 1 year.

The LAT have anatomical hot spots, varying by mechanism. Focal as well as small re-entrant LAT often lurk at the anterior aspect of the left superior PV (owing to catheter instability here) or just anterior to the right PVs (stemming
from thicker septal tissue) (5, 11, 19, 22). The 3 most common macro-re-entrant LAT circuits in the study by Chae et al. (8) used the left lower PV to mitral isthmus, the left atrial roof, and the septal wall. The mitral isthmus has been examined clinically and pathologically (10, 23, 24). Spanning 20 to 50 mm in length, the isthmus extends 5 to 15 mm inferior to the coronary sinus (CS) (or great cardiac vein). Obstacles to safe, successful isthmus ablation include tissue thickness (sometimes exceeding 5 mm) (23, 24), failure to ablate inferior to the CS, crevices predisposing to steam pops (10, 24), venous muscular coating (especially more medially) (23, 24), significant circumflex arterial branches (especially more laterally) (23, 24), and left atrial myocardium sometimes extending onto the valve (23, 24). The Bordeaux group elegantly showed both the importance of complete conduction block across the mitral isthmus and the electrophysiological maneuvers to confirm it (10). Ablating the alternative isthmus from right inferior PV to mitral valve proved nearly impossible in a surgical ablation series (25). The technique and results for left atrial roof linear ablation and confirmation of block also have been described (9). Generally, this line required less radiofrequency time than the mitral line and the anatomy is not as complex.

Of special importance are the LAT occurring in the septal wall because ablation is quite challenging (8). Indeed, Chae et al. (8) report a success rate of 38% for septal LAT versus 83% to 100% for other sites of LAT, plus a 7-fold increased risk of recurrence in follow-up. Contributors to the difficulty with septal LAT include the potential for atrioventricular node injury, needing to ablate both septal surfaces, and the thicker tissue (22).

The CS is also a frequent source of focal, small re-entrant, and even macro-re-entrant LAT after AF ablation, accounting for 5 of 21 small re-entrant circuits (24%) in the current series (8) and 9 of 33 late LAT (27%) in their prior series (21). In addition, ablation in the CS or great cardiac vein was needed to effect a complete mitral isthmus line in 62% of patients in the current series (8) and to up to 75% in other series (10), although lower values have been reported also (21), possibly because of anatomical variability and whether ablation is performed more proximally or distally (23, 24).

Further challenging the electrophysiologist are the multiple loop re-entrant arrhythmias, requiring bisection of 2 or more conduction isthmi found in 22% of patients in the current series (8), in 6 of 14 circuits in another report (5), but not noted in 2 other series (1, 2). Detailed, elegant examples are found in the current report (8).

Clinical Lessons and Prevention

The takeaway points from the current study (8), and in light of other data, include: 1) LAT can occur after any AF treatment, including segmental, ostial PV isolation (even single PV isolation), complex electrogram, or even surgical approaches, and especially the circumferential linear approach; 2) multiple circuits should be expected; 3) anticipated success is good (85% overall) in highly experienced laboratories, but lower for septal LAT; 4) complete PV isolation is important both at initial ablation and at retreatment for LAT; 5) linear lesions, if deployed, need to be continuous to prevent or treat macro-re-entrant LAT; 6) cavotricuspid isthmus flutter is possible despite atypical ECG patterns (21); 7) CS foci or circuits paradoxically have positive inferior P waves (8); and 8) a multiple-loop re-entrant circuit is implicated if the LAT does not terminate despite isthmus ablation. Although linear lesions may help to eliminate AF at initial ablation, incomplete linear lesions may be proarrhythmic, and even complete lines can promote re-entry. Thus the risk–benefit ratio of such linear lesions needs further study.

REFERENCES

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