

Minimally invasive surgical ablation: Should we ablate harder or smarter?



David Callans, MD, FHRS, Ramanan Kumareswaran, MD

From the Cardiac Electrophysiology Section, Cardiovascular Division, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania.

Despite continuing enhancement of our knowledge of many aspects of atrial fibrillation (AF), our understanding remains incomplete. AF causes significant reduction in quality of life because of AF and increases the risk of stroke.¹ Many patients still remain symptomatic despite adequate rate control and may benefit from rhythm control approach. The efficacy of antiarrhythmic drugs (AADs) is modest at best and carries a long list of side effects including proarrhythmia.² This has led to many patients opting to undergo invasive procedures to improve their quality of life.

A surgical approach with the Cox-Maze cut-and-sew technique was at the dawn of invasive approach to AF management.³ Although this approach was never subjected to intensive postprocedural monitoring, it likely addressed both trigger that initiates AF and substrate that sustains it. Technical complexity and significant comorbidities have limited the adoption of this technique. The realization that most of the triggers for AF initiates from pulmonary vein (PV) atrial muscle sleeves led to the development of PV isolation by catheter-based ablation procedures.⁴ Improvement in techniques and technology continues to make the procedure safer and likely more effective. Yet, we are not close to curing AF.

Thus, we continue to experiment in the hopes of identifying better ways of treating our patients. One of the approach is revisiting surgery in a minimally invasive fashion, particularly with the expectation that surgical ablation should be more powerful than catheter ablation. In this issue of *HeartRhythm*, Saini et al⁵ publish their considerable experience in surgical ablation of AF. They presented a single-center, single-operator experience from 2006 to 2012, enrolling both patients with paroxysmal AF and those with persistent AF (60 and 49, respectively). Their cohort of patients had failed 1 or more AADs, had failed catheter ablation, and/or “were not candidates for catheter ablation” (eg, lack of femoral access or morbid obesity). Nonetheless, it is not immediately clear whether this patient population was substantially different from those observed in most series

of catheter ablation. The procedure targeted PV isolation, epicardial ganglionic plexi ablation, ligament of Marshall ablation, and left atrial appendage (LAA) exclusion or excision. The surgical approach involved mini-thoracotomy early in the study, with development of a fully thoracoscopic technique in the last 36 patients, with additional ablation targets (roof and floor lines). Thirty-day ambulatory monitoring was performed at 6 months, 12 months, and every 12 months thereafter unless the patient had continuous monitoring with a pacemaker.

The authors should be commended for close monitoring for a long period (4.7 ± 1.8 years). Thirty-eight percent of the patients remained AF free without any additional intervention at 5-year follow-up after the 90-day blanking period. Interestingly, the pattern of recurrence was steadily progressive, with continual relapse extending over the follow-up period, just as has been observed with catheter ablation. However, counting the impact of additional interventions (AADs and catheter ablation), nearly 80% of the patients remained AF free. These are noteworthy results in this cohort of patients, especially given the remarkable long-term follow-up. The complication rate of this procedure was 6.4% without any deaths. Major adverse events included 1 patient with permanent right phrenic nerve paralysis and 2 patients requiring cardiopulmonary bypass surgery. One patient without LAA exclusion had perioperative stroke due to lack of anticoagulation, even though the patient remained in AF. The mean length of hospital stay was long at 6.1 ± 2.7 days.

Despite the encouraging results, in the absence of a comparator group, many questions remain to be answered. First, how can we identify the patients who will benefit from this more invasive approach compared with catheter-based ablation? It remains puzzling that recurrent PV conduction is a considerable problem: PV reconnection was noted in 13 of the 21 patients who underwent endocardial ablation after surgery.

Second, what accounts for the steady progression of AF recurrence throughout the follow-up period? Recent insights into AF disease progression caused by poorly treated related medical conditions (sleep apnea, obesity, and hypertension) suggest 1 possible mechanism⁶; these data are not available from the present study.

Address reprint requests and correspondence: Dr David Callans, Cardiac Electrophysiology, University of Pennsylvania Health System, 3400 Spruce St, Founders 9 Pavilion, Philadelphia, PA 19104. E-mail address: david.callans@uphs.upenn.edu.

Third, we need to improve our understanding of non-PV triggers and the substrate for AF. Considerable interest exists in identifying possible “drivers” in AF whether it be rotors, focal sources, or small reentrant circuits.^{7–10} Yet these drivers remain either elusive or nonuniversal. Hence, mapping and targeting additional regions besides PVs have yielded mixed results at best. Therefore, the gain in outcome with ablation of ligament of Marshall, ganglionic plexi, and/or posterior wall is unclear. Further studies are needed to shed light in sorting out which of these ablation procedures contribute to better outcomes.

The most alarming finding in this study is the risk of transient ischemic attack or stroke of 5% after exclusion of LAA. Events occurred even in patients without any residual LAA leak and during normal sinus rhythm at presentation. None of these patients were on target-specific anticoagulants at the time of diagnosis. Certainly there has been extensive data that LAA exclusion using either surgical or catheter-based techniques, even where expertly performed, does not exclude the possibility of stroke.¹¹ The mechanism of stroke in these patients is still debatable, whether it is cardioembolic in nature or AF is just a marker for risk of stroke. Further studies that include newer, safer anticoagulants are needed to address the risk/benefit ratio of stroke in this special cohort of patients.

The larger question is where do we go from here? There remains a population of patients who are quite symptomatic, drug refractory, and not ideal candidates for endocardial ablation. This is the patient population who may benefit from a more invasive surgical approach. Small randomized studies have suggested benefit to minimally invasive surgical ablation compared with catheter ablation.¹² The results in the present study are not clearly superior to what might reasonably expected with contemporary catheter-based ablation techniques. Saini et al have shown the feasibility of surgical ablation in experienced hands with good long-term outcome, especially when combined with other interventions. It is yet

another tool that may improve the quality of life in our patients with AF when appropriately applied. This study definitely supports the need for a randomized controlled multicenter trial to elucidate the true benefit of minimally invasive surgery for AF.

References

1. Dorian P, Jung W, Newman D, Paquette M, Wood K, Ayers GM, Camm J, Akhtar M. The impairment of health-related quality of life in patients with intermittent atrial fibrillation: implications for the assessment of investigational therapy. *J Am Coll Cardiol* 2000;36:1303–1309.
2. Jais P, Cauchemez B, Macle L, et al. Catheter ablation versus antiarrhythmic drugs for atrial fibrillation the A4 study. *Circulation* 2008;118:2498–2505.
3. Millar RC, Arcidi JM, Alison PJ. The Maze III procedure for atrial fibrillation: should the indications be expanded? *Ann Thorac Surg* 2000;70:1580–1586.
4. Piccini JP, Lopes RD, Kong MH, Hasselblad V, Jackson K, Al-Khatib SM. Pulmonary vein isolation for the maintenance of sinus rhythm in patients with atrial fibrillation: a meta-analysis of randomized, controlled trials. *Circ Arrhythm Electrophysiol* 2009;2:626–633.
5. Saini A, Hu YL, Kasirajan V, Han FT, Khan MZ, Wolfe L, Gunda S, Koneru JN, Ellenbogen KA. Long-term outcomes of minimally invasive surgical ablation for atrial fibrillation: A single center experience. *Heart Rhythm* 2017;14:1281–1288.
6. Pathak RK, Middeldorp ME, Lau DH, et al. Aggressive risk factor reduction study for atrial fibrillation and implications for the outcome of ablation: the AR-RESTAF cohort study. *J Am Coll Cardiol* 2014;64:2222–2231.
7. Verma A, Jiang CY, Betts TR, et al. STAR AF II Investigators. Approaches to catheter ablation for persistent atrial fibrillation. *N Engl J Med* 2015;372:1812–1822.
8. Dixit S, Marchlinski FE, Lin D, et al. Randomized ablation strategies for the treatment of persistent atrial fibrillation: RASTA study. *Circ Arrhythm Electrophysiol* 2012;5:287–294.
9. Miller JM, Kowal RC, Swarup V, et al. Initial independent outcomes from focal impulse and rotor modulation ablation for atrial fibrillation: multicenter FIRM registry. *J Cardiovasc Electrophysiol* 2014;25:921–929.
10. Hansen BJ, Zhao J, Csepe TA, Moore BT, et al. Atrial fibrillation driven by micro-anatomic intramural re-entry revealed by simultaneous subepicardial and sub-endocardial optical mapping in explanted human hearts. *Eur Heart J* 2015;36:2390–2401.
11. Masoudi AF, Calkins H, Kavinsky CJ, Drozda JP, Gainsley P, Slotwiner DJ, Turi ZG. 2015 ACC/HRS/SCAI left atrial appendage occlusion device societal overview. *Heart Rhythm* 2015;12:e122–e136.
12. Ad N, Damiano RJ, Badhwar V, Calkins H, Meir ML, Nitta T, Holmes SD, Weinstein AA, Gillinov M. Expert consensus guidelines: examining surgical ablation for atrial fibrillation. *J Thorac Cardiovasc Surg* 2017;153:1330–1354.