

# Long-standing Persistent Atrial Fibrillation Ablation: How do You Perform it?

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## ABSTRACT

Catheter ablation of long-standing persistent atrial fibrillation (LSPAF) presents unique challenges and the lack of large body of evidence surrounding management makes for disagreement and different approaches for treatment. Outlined is a case example that offers a comprehensive approach to ablation in patients with LSPAF that consists of risk factor management, an ablation strategy, a rigorous trigger protocol and follow-up rhythm monitoring. The case presented highlights management of this difficult population as best guided by current evidence and our experience. Ablation treatment and management strategies will continue to evolve with further randomized data and the advent of improved ablation technologies.

**KEYWORDS:** Arrhythmia; Atrial fibrillation; Catheter ablation; Nonpulmonary vein trigger.

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## INTRODUCTION

Outcomes of catheter ablation in patients with long-standing persistent atrial fibrillation (LSPAF), defined as continuous atrial fibrillation (AF) > 12 months duration, have worse long-term arrhythmia free survival compared with ablation of paroxysmal atrial fibrillation (pAF). For both pAF and LSPAF, empiric pulmonary vein isolation (PVI) is the foundation of catheter ablation as pulmonary vein (PV) triggers have a similar prevalence in both entities. After PVI, evidence for further ablation is less robust despite LSPAF being associated with increased left atrial triggers<sup>1</sup>. Empiric linear lines or ablation of complex fractionated atrial electrograms (EGM) demonstrate no arrhythmia improvement to standard PVI alone and there is conflicting evidence for empiric ablation of the posterior wall<sup>2</sup>. However, identifying and targeting non-PV triggers has shown to improve AF-free survival<sup>3</sup>. Given the lack of concrete evidence regarding a standard approach in this challenging population, there is not complete agreement among the electrophysiology community on how to perform AF ablation in patients with LSPAF.

A case is outlined below demonstrating our approach to catheter ablation in this challenging patient population along with the rational supporting our method. All patients undergo aggressive management of modifiable risk factors for AF. Our ablation approach consists of empiric wide antral circumferential isolation of the PVs in addition to empiric posterior wall isolation (PWI). This is followed by a rigorous trigger protocol which includes infusion of high doses of isoproterenol with and without atrial burst pacing with the aim of identifying and safely isolating or eliminating all potential AF triggers. Antiarrhythmic medications are occasionally used to manage early arrhythmia recurrences during the 90-day blanking period, and routinely stopped at 3 months post procedure. Vigilant out-patient monitoring with a standardized monitoring protocol is carried out to ensure maintenance of sinus rhythm and repeat ablation may be necessary in the setting of recurrence. The follow-up protocol consists of at least 3 outpatient visits (6 weeks, 6 months, and 1 year from date of ablation). Patients are routinely provided with a 30-day transtelephonic monitor at discharge from the hospital, at 6 and 12 months. Additional transtelephonic monitoring is performed if patients report arrhythmia symptoms between visits. Patients are also instructed to assess their pulse twice daily and report any irregularity of pulse or recurrence of symptoms immediately, at which time additional event monitoring is performed. In recent years, continuous postablation monitoring with implantable devices has been increasingly utilized.

## CASE REPORT

The patient is a 47-year-old male with a past medical history of hypertension, obstructive sleep apnea and long-standing persistent atrial fibrillation (LSPAF). He was first diagnosed with atrial fibrillation (AF) at age 29 and had been managed with a rate control strategy of metoprolol and diltiazem prior to consultation. He has a CHADSVASc score of 1 and was taking dabigatran 150 mg twice daily for stroke prevention. He endorsed symptoms of dyspnea on exertion and episodes of lightheadedness. Transthoracic echocardiogram revealed normal left ventricular function (65%), a severely enlarged left atrium (6.6 cm) and no significant valvular disease. After a discussion regarding management options and risks and benefits, the patient elected to undergo catheter ablation.

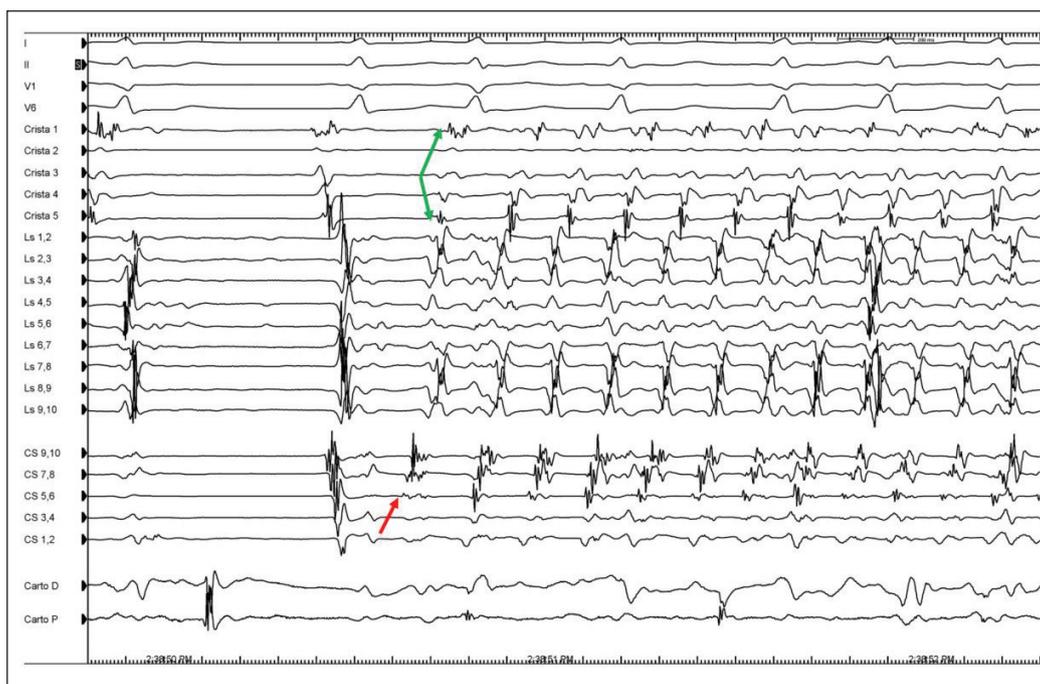
Prior to ablation, modifiable risk factors were addressed in office to optimize procedural success. This included appropriate management of hypertension and medication compliance. Obstructive sleep apnea screening, subsequent sleep study and treatment with recommended continuous positive airway pressure. In addition to strongly encouraging weight loss through exercise and diet modification.

The procedure was performed under general anesthesia with high-frequency low-volume ventilation (jet ventilation). A transesophageal echocardiogram confirmed no evidence of LAA thrombus. Anticoagulation was continued uninterrupted. Venous access was obtained with ultrasound guidance and decapolar catheters were advanced into the coronary sinus (CS) and crista terminalis (CT). The CS catheter was positioned with the most proximal electrode at the CS ostium. The CT catheter was positioned in the SVC just above the junction with the right atrium with the distal electrode at the lower border of the right pulmonary artery. Appropriate catheter position is essential for trigger verification and the position was verified with intracardiac echocardiography (ICE) and fluoroscopy. An ICE catheter was used to guide dual transeptal

punctures and a medium curve steerable sheath and Swartz braided transeptal sheath were advanced into the left atrium (LA). An ablation catheter and a LASSO mapping catheter were advanced into LA. Electroanatomical mapping was then performed using CARTO 3D and ablation was performed with an open-irrigated contact force-sensing catheter (Thermocool SF SmartTouch, Biosense Webster, Irvine, CA).

The left and right pulmonary veins (PV) were circumferentially isolated as a common ostium using radiofrequency (RF) ablation at 45-50 W for up to 20 s on the anterior wall up to 8-10 s on the posterior wall. A linear esophageal temperature probe with a single thermocouple was used and lesion duration was titrated accordingly when ablating tissue in close proximity. Phrenic pacing was performed along the anterior aspect of the right PVs prior to ablation and sites of phrenic capture were identified and avoided. The patient was cardioverted with a single synchronized shock of 360 joules with return of sinus rhythm without early recurrence of AF (ERAF). Entrance and exit block were confirmed in both PVs pacing at 10mA. The posterior wall was empirically isolated. To accomplish posterior wall isolation, we typically start with a roof line and a floor line. In case of persistent posterior wall connection, additional segmental ablation is performed targeting the earliest sites within the posterior wall in order to achieve isolation with bidirectional block. The latter is verified by careful remapping of the posterior wall demonstrating lack of EGMs (entrance block) and pacing at different sites at 10 and 50 mA output to demonstrate exit block. Adenosine was administered to produce temporary AV block, without evidence of acute PV or posterior wall reconnection.

Programmed atrial and ventricular stimulation was then performed to exclude the presence of atrioventricular nodal reentrant tachycardia and atrioventricular reentrant tachycardia as these can be triggers for AF initiation. A trigger induction protocol was initiated, and isoproterenol was infused at a maximum rate of 20 µg/min for at least 10-15 min. At the peak isoproterenol infusion, a non-PV trigger resulting in sustained AF was noted which originated from the mid-CS based on analysis of the earliest endocardial site of activation on the CS catheter and characteristic activation pattern (Fig. 1).



**Figure 1.** Coronary sinus trigger from the CS body (red arrow) with a chevron appearance on the CT catheter likely indicating simultaneous transseptal right atrial activation via the Bachmann's bundle (green arrows).

Coronary sinus isolation was then performed by targeting CS potentials from the LA endocardial surface and CS potentials from within the CS on the epicardial surface. Coronary sinus isolation is typically performed with point-by-point ablation with a power of 40-45 W for up to 10 s. Similar to ablation on the posterior wall, diligent esophageal temperature monitoring is performed during CS isolation. While ablating in the CS, it is critical to maintain the catheter tip facing the

posterior LA. Ablation of the ventricular aspect of the CS is never necessary given no muscular connection with the LA at this juncture<sup>4</sup>. Coronary sinus isolation was achieved after disappearance of CS potentials and demonstrating entrance block (Fig. 2) as well as lack of capture with 10 mA output pacing showing exit block (Fig. 3). The trigger induction protocol was then repeated with no further evidence of AF sustaining triggers (Fig. 4).

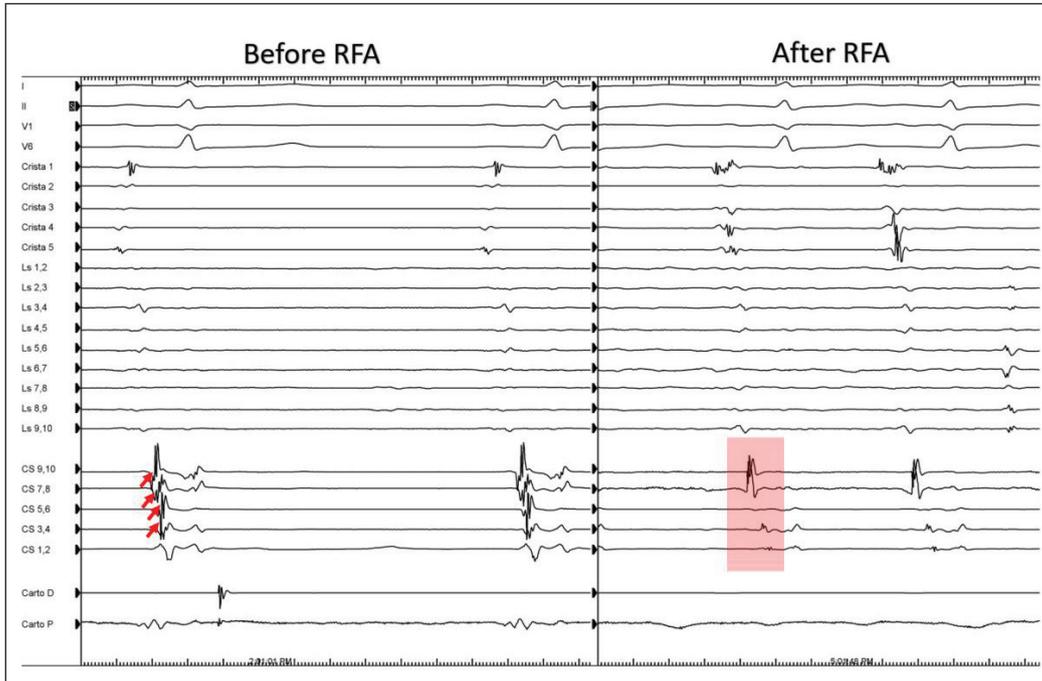


Figure 2. Coronary sinus activation before and after CS ablation and isolation. Notice the sharp PV-like potentials (red arrows) on the CS catheter eliminated after catheter ablation with only remnant far field left atrial endocardial signals (red).

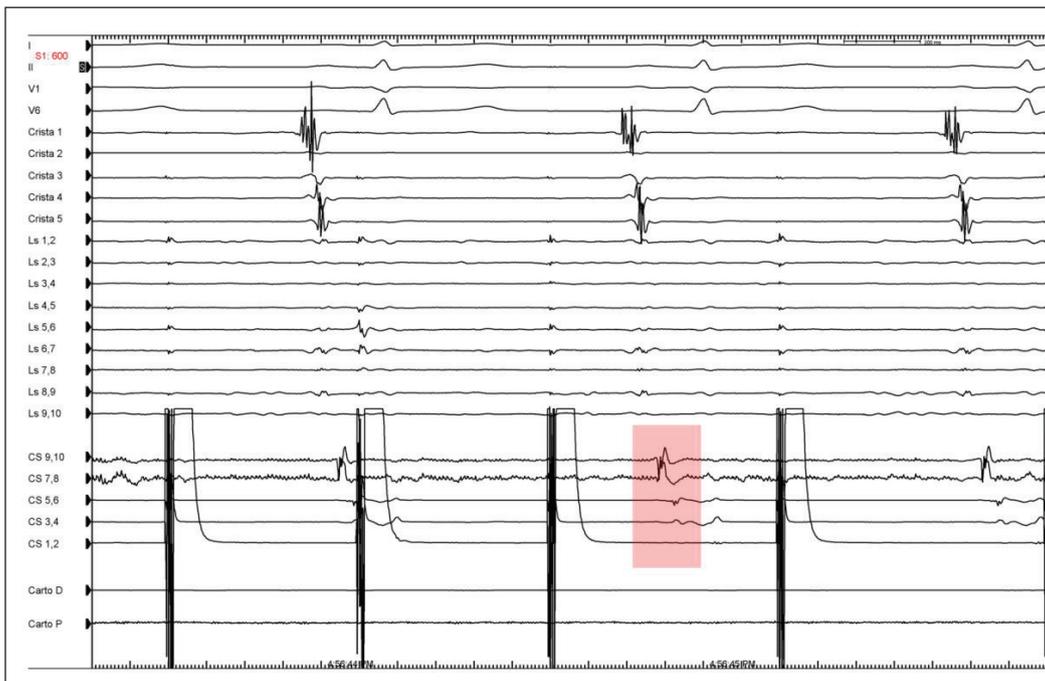
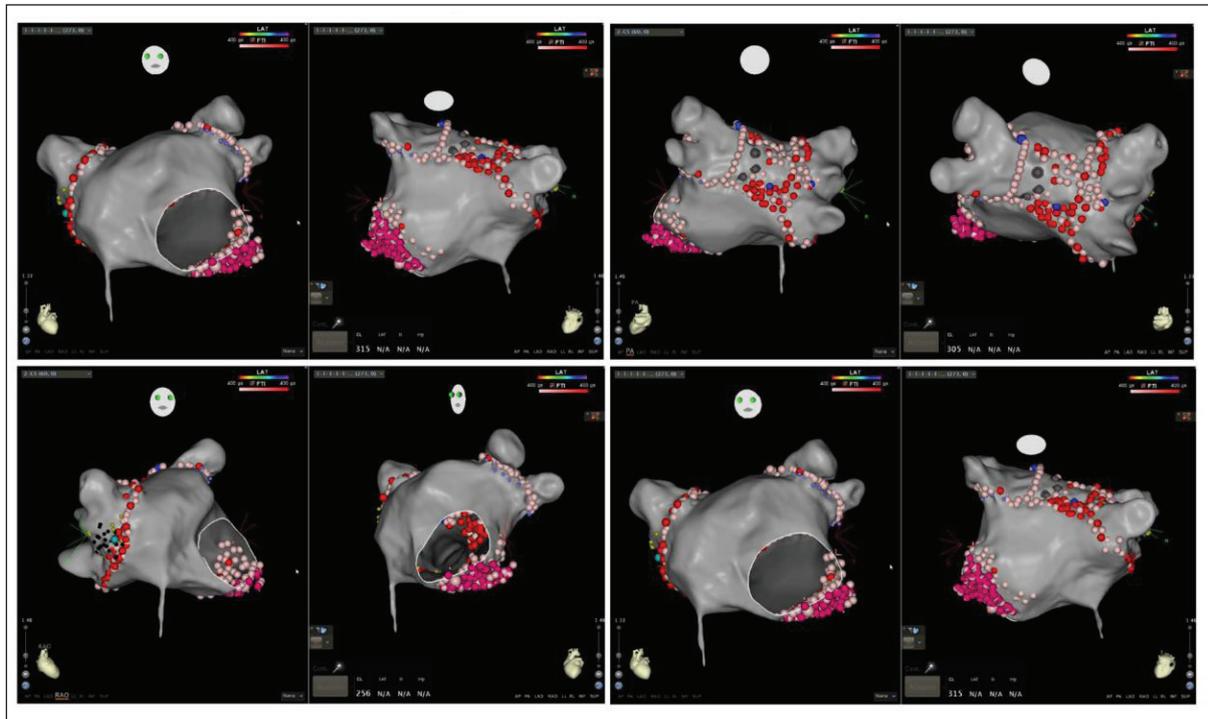


Figure 3. Pacing within the CS demonstrating exit block. Far field endocardial signals (red).

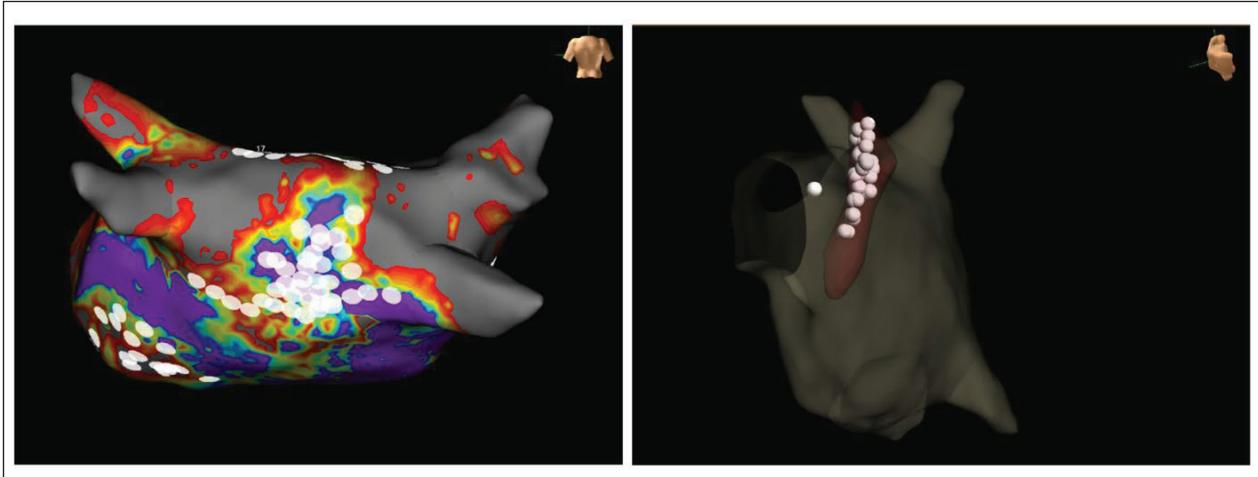


**Figure 4.** Primary procedure final lesion set demonstrating pulmonary vein, posterior wall and coronary sinus lesion sets. Radiofrequency (RF) endocardial lesions (red and pink balls), Lesion with block (blue balls), RF epicardial CS lesions (magenta balls), Phrenic capture (grey balls).

Post procedure the patient was continued on dabigatran 150 mg twice daily and was started on dofetilide 250 mg daily with his QT monitored prior to discharge. Using a single lead home monitoring device, the patient monitored his rhythm daily. He had recurrences during the blanking period requiring cardioversion and eventual change of dofetilide to short-term amiodarone 200 mg daily with no further recurrences. Amiodarone was discontinued without arrhythmia recurrence. He then elected to have an implantable cardiac rhythm monitor placed for long-term AF surveillance and to guide the need for anticoagulation. The patient developed late AF recurrence (>1 year) off amiodarone therapy and he elected to undergo repeat catheter ablation of AF.

The setup for the repeat ablation procedure was unchanged from the previous with the exception of an alternative mapping catheter and mapping system; the HD Grid and EnSite Velocity. We prefer the HD grid catheter as a platform for high-density mapping for any repeat AF ablation procedure or scar-related arrhythmias given the significantly improved quality of low-amplitude EGMs recorded compared to other commercially available multipolar catheters in addition to the unique capacity of displaying voltage information that are less impacted by the mapping wavefront directionality.

The patient presented to the EP lab in sinus rhythm. High-density mapping demonstrated chronically isolated left and right PVs from the previous procedure. Mapping of the LA revealed reconnection of posterior wall and reconnection of the mid-distal coronary sinus. The posterior wall and coronary sinus were reisolated with segmental ablation using an open-irrigated catheter with contact force sensing (Tacticath SE, St. Jude Medical, St. Paul, MN) using the same high-power settings detailed above (Fig. 5). However, with Tacticath SE we have been reducing the lesion duration to 5-10 s maximum as we have observed significantly faster EGM attenuation/elimination with this catheter.



**Figure 5.** Second procedure final lesion set demonstrating repeat posterior wall ablation and repeat coronary sinus ablation. Radiofrequency endocardial lesions (white circles); RF epicardial CS lesions (light pink balls).

Adenosine 18 mg was administered confirming persistent entrance and exit block. Isoproterenol was then reinfused at 20  $\mu\text{g}/\text{min}$  with no evidence of non-PV triggers. The patient was discharged on amiodarone 200 mg daily and metoprolol 100 mg daily along with dabigatran 150 mg twice daily. The amiodarone was discontinued after 6 weeks of therapy. The patient has remained in sinus rhythm off antiarrhythmic medications for > 1 year without evidence of recurrence on his implantable loop recorder.

## DISCUSSION

### Risk factor management

The management of LSPAF through catheter ablation starts prior to the electrophysiology lab with aggressive risk factor management. A comprehensive approach should be undertaken composed of patient education, lifestyle modification, coordination with specialists and longitudinal follow-up. One should target all potential modifiable risk factors such as obesity, obstructive sleep apnea, hypertension, diabetes, hyperlipidemia, smoking and excess alcohol consumption. At our center, a dedicated nurse-led risk factor modification program has been modeled and demonstrated success in achieving weight reduction, risk factor management and improved arrhythmias symptoms<sup>5</sup>. This often-neglected pillar in the management of AF is critical for procedural success with prospective randomized data demonstrating improved long-term success after catheter ablation<sup>6</sup>.

### Improved catheter stability

Procedural success is dependent upon the operators' ability to perform durable isolation and we employ several different tools to maximize procedural success and ensure catheter stability. All patients are placed under general anesthesia rather than conscious sedation to eliminate excessive patient movement, and minimize respiratory variation and thoracic excursion. High-frequency jet ventilation further limits left atrial motion to ensure catheter stability and has demonstrated improved AF free survival and limits chronic PV reconnection<sup>7</sup>. General anesthesia could impact non-PV trigger formation but the level of sedation and isoproterenol can always be titrated and it hasn't been our experience with the use of an aggressive trigger protocol<sup>8</sup>.

High-power short-duration (HPSD) has the benefit of markedly reduced procedural time and a potential for superior lesion durability with a comparable safety profile to standard lower power and longer duration ablation protocols<sup>9,10</sup>. Our approach has been to use HPSD lesions at 45-50 W of varying duration depending on proximity to esophageal tissue. Radiofrequency lesion formation occurs immediately through resistive heating, which results in permanent cellular injury. This is followed by conductive heating, which results in passive cellular injury to deeper tissues. Preclinical studies have shown that HPSD creates larger lesion diameters with a more predictable irreversible resistive injury and less passive injury compared with conventional treatment<sup>11,12</sup>. Due to the significantly shorter duration of RF application, effective lesions with HPSD are expected to be less sensitive to catheter drift. In regards to AF free survival using HPSD, randomized trial data using 45-50 W does not currently exist and further outcomes research is warranted.

## Posterior wall isolation

It is established that outcomes of PVI alone for LSPAF do not approach the procedural success of patients with paroxysmal AF. This has led to a search for additional ablation targets of AF initiation in this population in an effort to improve procedural outcomes. The left atrial posterior wall shares embryologic origins with the PVs and is also a major source of AF triggers making it an ideal target for isolation. At our institution it is standard to perform empiric PWI for LSPAF as it has shown superior freedom from AF compared to PVI alone<sup>13</sup>. However, controversy exists as to whether the addition of the posterior wall improves outcomes in patients with LSPAF and there is conflicting randomized data which makes its utility highly debated<sup>14</sup>. Difficulty achieving transmural ablation lesions with increased rates of reconnection and an inability to achieve complete PWI is likely responsible for these disparate results. If transmural ablation can be achieved the outcomes of PVI and PWI in patients with persistent AF are in clear favor of isolation<sup>15</sup>.

Entrance and exit block should be confirmed with pacing at 10 and 50 mA to ensure durable isolation. Posterior wall isolation allows for elimination of a large surface area of potential triggers and through atrial debulking may affect the maintenance of AF. With more extensive catheter ablation, there is concern for impaired LA contractility and the formation of stiff left atrial syndrome. Stiff left atrial syndrome is the result of impaired LA reservoir and conduit functions with development of left atrial diastolic dysfunction and subsequent pulmonary hypertension. However, the left atrial impairment from ablation is likely counterbalanced by positive remodeling that occurs with a rhythm control strategy. In addition, the anterior wall, rather than the posterior wall, is the major contributor to LA contractility<sup>13</sup>. For these reasons, atrial function after catheter ablation has not shown a reduction in contractility<sup>16</sup>, and stiff left atrial syndrome is associated with more extensive LA scarring than achieved through posterior wall isolation alone<sup>17</sup>. Despite PWI, long-term recurrence may still occur, so it is important to identify other sources of possible AF initiation to achieve optimal results.

## Non-PV triggers

Targeting non-PV triggers and PVI has been suggested superior to PVI alone and our trigger protocol outlined above allows for a standardized approach to targeting these triggers. The optimal protocol for non-PV triggers has never been formally evaluated but this has been the standardized approach at our institution. Atrial and ventricular stimulation allows for a baseline assessment of conduction properties and evaluation of reentrant supraventricular arrhythmia, which can be a potential source even in LSPAF. Pharmacologic challenge and pacing maneuvers are performed to help elicit AF triggers. Burst pacing becomes increasingly aggressive with the specific aim of AF induction to allow for detection of postcardioversion triggers. Moving the mapping catheter around the atria with repeated AF induction may be necessary to precisely localize a trigger that is located in areas that are not directly recorded (e.g., interatrial septum, anterior LA wall, left atrial appendage) and often it can only be regionalized, which is why regional isolation of cardiac structures is often required. The aim is elimination or isolation of all spontaneous or postcardioversion triggers with repeated pharmacologic

challenges. Achieving noninducibility after means of burst pacing has not been shown to improve AF free survival and should not be used as an endpoint or encourage further ablation lesion sets<sup>18</sup>. Burst pacing is used for AF induction as a means to identify postcardioversion triggers resulting in ERAF.

In our institution, targets for ablation are non-PV triggers leading to sustained AF or reproducible nonsustained AF. Non-PV trigger locations can be assessed with earliest site of activation and patterns of activation in addition to assessment of P-wave morphology. Assessment of P-wave morphology is frequently not possible because of fusion with the T-wave during tachycardia. If necessary, atrial pace mapping can be utilized to help localize spontaneous or induced triggers. Trigger sites arise from discrete right and left sided cardiac structures and each trigger site has varying prevalence, with left sided non-PV triggers being more common in patients with LSPAF<sup>1</sup>. The various non-PV trigger sites each possess their own procedural challenges and safety concerns and a detailed understanding of cardiac anatomy and surrounding cardiac structures is critical.

The etiology for recurrence in the above case was secondary to reconnection of the posterior wall and coronary sinus, with freedom from AF achieved after repeat isolation and no further evidence of non-PV triggers. This is not an infrequent reason for procedural failure and prior ablation should always be assessed to ensure adequate blockade with repeat procedures. Alternative reasons for failure include inadequate non-PV trigger elimination or the development of new non-PV triggers overtime. There is data to suggest that new non-PV triggers play a greater role in recurrence<sup>19</sup>, and further research is warranted to better guide ablation strategies.

## Coronary sinus isolation

Coronary sinus isolation requires both endocardial ablation and epicardial ablation within the CS. Triggers originate from the muscular portion of the CS which extends from the CS ostium to valve of Vieussens, located at the insertion of the vein of Marshall. True CS triggers generally originate from the proximal and mid portion of the CS catheter and attention should be made to exclude a primary LA structure or a trigger from within the ligament of Marshall. Triggers from the distal CS catheter almost certainly represent an LA structure, as the CS musculature does not extend beyond the valve of Vieussens where the distal CS catheter extends. The CS catheter will record both sharp near-field electrograms, which represent true CS muscular activation and blunt far field signals, representing the inferior mitral annulus. Coronary sinus trigger P-wave morphology is superiorly directed with negative P-waves in II, III and AvF.

It is important to be vigilant when ablating this region to prevent unwanted complications. Similar to ablation of the posterior wall, precaution should be taken to prevent esophageal injury or atrioesophageal fistula (AEF) formation. Methods to prevent injury should be utilized such as temperature monitoring, power reduction, esophageal deviation or active esophageal cooling. We currently utilize temperature probe monitoring and power reduction although no strong evidence exists that monitoring reduces AEF formation and it could result in impaired lesion formation through operator changes in lesion delivery<sup>20</sup>. Esophageal cooling could be a promising alternative and is actively being investigated. Left circumflex coronary artery injury has also been recognized as a complication of ablation within the CS. This can be prevented by maintaining the ablation catheter in contact with the posterior aspect of the left atrium (as assessed with the RAO projection) and limiting point lesion duration to no more than 10-15 s. With this approach, we have never encountered a case of periprocedural coronary artery injury during CS isolation.

## CONCLUSION

Catheter ablation of LSPAF presents unique challenges and the lack of large body of robust evidence surrounding management makes for disagreement and different approaches for treatment. This case highlights our approach to ablation in patients with LSPAF as best guided by current evidence and our experience. Ablation strategies will continue to evolve with further randomized data and the advent of improved ablation technologies.

## FUNDING

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## AUTHOR'S CONTRIBUTION

Conceptualization: Smietana J. and Santangeli P.; Methodology: Smietana J. and Santangeli P.; Investigation: Smietana J. and Santangeli P.; Writing – Original Draft: Smietana J. and Santangeli P.; Writing – Review and Editing: Smietana J. and Santangeli P.; Funding Acquisition: Smietana J. and Santangeli P.; Resources: Smietana J. and Santangeli P.; Supervision: Smietana J. and Santangeli P.

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