Atrial isochronal late activation mapping keeps the diaphragm alive

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Introduction

This case highlights isochronal late activation mapping (ILAM) during atrial flutter (AFL) ablation in a patient with congenital heart disease. Compared to conventional mapping, which localized relevant substrate overlying the phrenic nerve, ILAM in sinus rhythm identified a region of isochronal crowding medial to the phrenic nerve, avoiding phrenic nerve injury.

Case report

The patient is a 27-year-old woman with surgically corrected dextro-transposition of the great arteries and prior typical AFL who started to experience intermittent paroxysms of dizziness 1 month after the delivery of her second child. To evaluate her dizziness an electrocardiogram (ECG) and 2-day ambulatory rhythm monitor were performed. The ECG was unchanged from the prior and demonstrated sinus rhythm with a right bundle branch block and left posterior fascicular block. The ambulatory rhythm monitor revealed more than 300 episodes of regular supraventricular tachycardia (SVT) at a rate of 207 beats per minute, which was felt to be the cause of her symptoms.

The patient's past medical history includes dextro-transposition of the great arteries with a ventricular septal defect and pulmonic stenosis for which an initial right-sided modified Blalock-Taussig shunt was placed close to birth. This was followed by a Nikaidoh arterial switch operation with right ventricle–to–pulmonary artery conduit and ventricular septal defect repair at 3 years of age. She required subsequent replacement of the right ventricle–to–pulmonary artery homograft 2 years later, and then repeat pulmonary valve replacement. Many years later she was admitted with right AFL, for which she underwent uncomplicated cavotricuspid isthmus ablation. Her only medications were metoprolol succinate and aspirin.

KEYWORDS Isochronal late activation mapping; Atrial flutter; Adult congenital heart disease; Phrenic nerve; Ablation
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KEY TEACHING POINTS

- For patients with congenital heart disease, significant atrial low-voltage areas may occur that can be the substrate for reentrant atrial arrhythmias. While atrial flutter may occur in these patients, it is important to consider the full differential of supraventricular tachycardias.
- Up to 25% of patients with dextro-transposition of the great arteries have atrial tachyarrhythmias, which includes cavotricuspid isthmus–dependent atrial flutters, but are also at risk for other atrial reentrant arrhythmias owing to Mustard and Senning procedures.
- Isochronal late activation mapping (ILAM) can be performed during sinus rhythm and identifies deceleration zones often present in low-voltage areas of the atrium or ventricle. Atrial ILAMs can identify areas critical to atrial flutter reentry sites.

The differential diagnosis for regular SVT in adults with congenital heart disease (corrected or uncorrected) includes ectopic atrial tachycardia, AFL (typical or atypical, micro or macro), atrioventricular nodal reentrant tachycardia, orthodromic atrioventricular reentrant tachycardia, and sinus tachycardia.

The patient was brought to the electrophysiology lab for further characterization of the SVT with an electrophysiological study and treatment with radiofrequency ablation. The initial rhythm was sinus with frequent premature atrial contractions (PACs). Incessant salvos of multiple atrial tachycardia were also noted upon initiation of the procedure. The morphology of all PACs was high-to-low on surface ECG with proximal-to-distal coronary sinus (CS) catheter activation.

Intracardiac 3-dimensional mapping was performed using the CARTO electroanatomical mapping system (Biosense Webster, Irvine, CA). An Optrell catheter (Biosense Webster, Irvine, CA) was advanced to the right atrium (RA) through a

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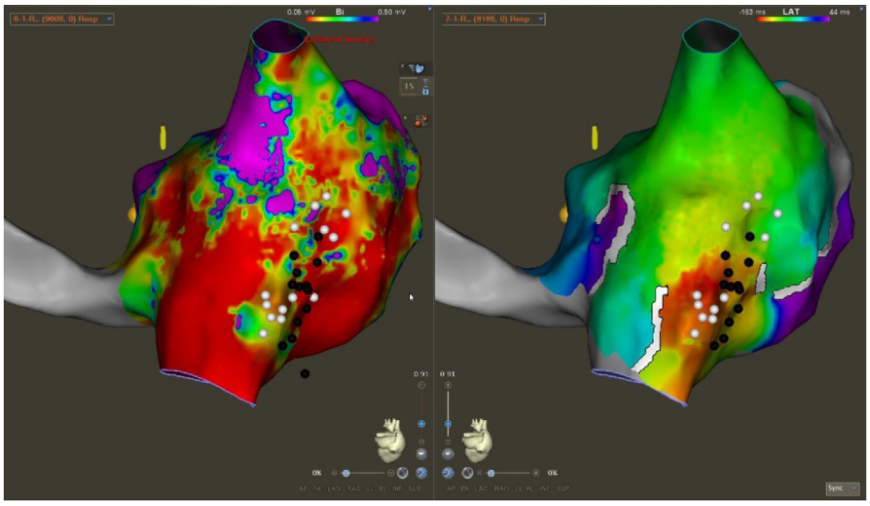

Figure 1 Conventional bipolar mapping. Bipolar voltage map in posterior-anterior projection with 0.05 mV or less represented in red (left) and bipolar activation map during AT1 (total cycle length 330 ms) (right). Black dots represent phrenic nerve capture at 10 mA and white dots represent no phrenic nerve capture.

sheath and high-density bipolar mapping of the RA was performed. Voltage mapping identified significant areas of right atrial scar related to her prior surgeries. Subsequently, a microreentrant AFL occurred spontaneously and was induced with atrial burst pacing and isoproterenol administration up to 10 mcg/min. There were multiple additional inducible atrial tachycardias that appeared to be focal or microreentrant in nature, emanating from regions adjacent to lateral right atrial low-voltage areas.

Atrial tachycardias induced were as follows:

- AT1: tachycardia cycle length (TCL) 340 ms, proximal-to-distal CS activation, earliest activation mapped to posterior inferior RA; entrainment confirmed that AT1 was a microreentrant atrial flutter involving the posterior inferior RA.
- AT2: TCL 280 ms, proximal-to-distal CS activation, earliest activation mid/high crista terminalis; entrainment confirmed that AT2 was a microreentrant atrial flutter involving the mid/high crista terminalis.
- AT3: TCL 320 ms, proximal-to-distal CS activation, terminated/degenerated before adequate mapping could be performed.

Cluster ablation was performed at the site of earliest activation for AT1 (Figure 1). Ablation at this location was limited by proximity to the phrenic nerve, as evidenced by phrenic nerve capture at 10 mA. AT1 intermixed with AT2 and AT3 were still inducible following ablation with atrial burst pacing. Given multiple intermixed ATs with similar surface and intracardiac morphologies in relation to complex underlying substrate, atrial ILAM was pursued. In sinus rhythm, areas of slow atrial conduction and isochronal crowding in the lateral RA were demarcated. Specifically, the ILAM map was created using the Late Annotation Mapping (LAM) software in CARTO. The LAM software creates a late boundary within the AT cycle length window of interest where late potentials are recorded and the latest negative unipolar deflection (-dV/dt) is recorded. The LAM software will then analyze the last 2 AT cycle lengths to see if there is an electrogram that correlates with the negative unipolar deflection originally recorded. A comparison of bipolar endocardial voltage map and ILAM mapping indicated an area anterior to the position of phrenic nerve capture in the RA (Figure 2) as a significant zone of deceleration within this chamber, possibly relating to the patient's AFLs. Scar homogenization was then performed to target atrial ILAMs within the anterolateral RA region of low-voltage zones targeting regions of pace capture and near-field electrograms. It was opted to ablate at the areas of atrial ILAMs within the low-voltage zones of the anterolateral RA because this area represented a critical isthmus for all of the atrial tachycardias, in addition to the fact that it was distant from the phrenic nerve. In addition, linear ablation from regions of scar homogenization to lateral tricuspid annulus was then performed, resulting in the isolation of a portion of the anterolateral RA in order to prevent future AFLs using the cavotricuspid annulus (Figure 3).

Following ablation there was no inducible atrial tachycardia, AFL, or AF with atrial burst/ramp pacing down to 200 ms on isoproterenol 20 mcg/min. Frequent monomorphic PACs were noted with high-to-low atrial activation. The PAC was mapped to posterior/superior RA, in close proximity to the region of earliest activation for AT2. Cluster ablation at site of earliest activation for the PAC was performed, then the cluster of lesions was connected with region of scar homogenization in the lateral RA. The patient tolerated the procedure well and left the laboratory in good condition.

Discussion

The differential diagnosis for regular SVT in adults with congenital heart disease is similar to that of patients without congenital heart disease but with some notable differences.

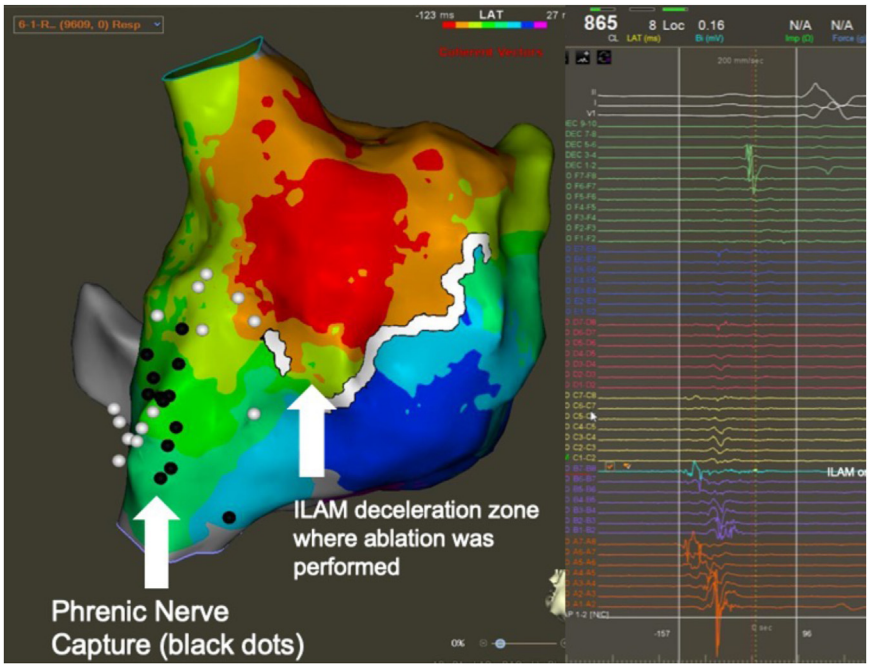

Figure 2 Isochronal late activation mapping. Isochronal late activation map in sinus rhythm with intracardiac electrograms demonstrating a region of isochronal crowding away from the phrenic nerve.

Macroreentrant atrial tachycardias remain one of the most common SVTs in the adult congenital heart disease population, which may be owing to previous corrective surgery or adverse atrial remodeling. ILAM may be a useful tool to guide ablations in patients with complex underlying substrate and fleeting, intermixed atrial arrhythmias, such as those with congenital heart diseases.

Though the principle of deceleration zones, identified during sinus rhythm, can be useful in evaluating and targeting ventricular arrhythmias,[1,2] the use of this method for atrial arrhythmias is limited. However, there have been increasing descriptions of ILAM application to atrial arrhythmias. Woods and colleagues[3] recently showed that ILAM deceleration zones in sinus rhythm correlate with the critical isthmus of localized reentrant AFL, and Kuo and colleagues[4] demonstrated how ILAM deceleration zones identified after atrial fibrillation ablation may predict atrial fibrillation recurrence after pulmonary vein isolation.

In this case, we highlight the use of ILAM mapping in a patient with congenital heart disease presenting with multiple AFLs to (1) avoid collateral injury to juxtaposed tissue such as the phrenic nerve and (2) efficiently and comprehensively address the relevant tachyarrhythmias. High-density mapping catheters, such as the Optrell catheter (a multielectrode spatula-shaped array with 48 electrodes distributed over 6

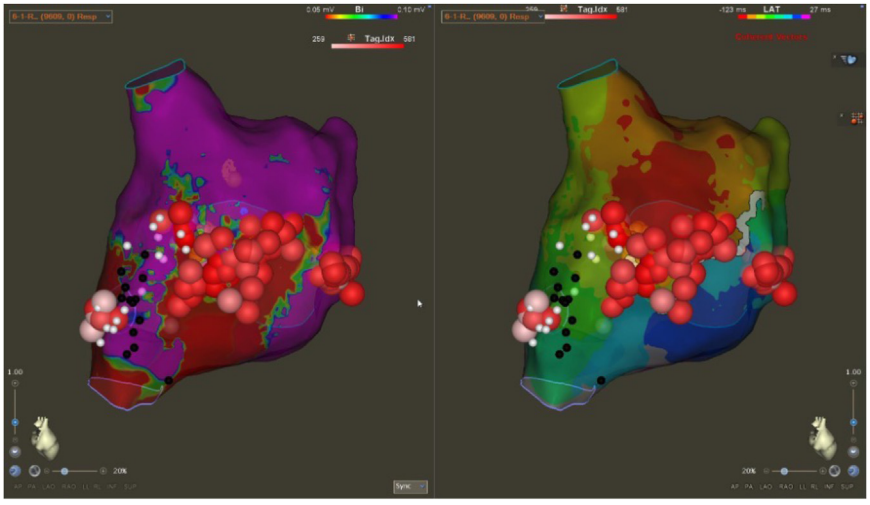

Figure 3 Ablation lesion set at the end of the case.

splines giving an interelectrode distance of 2.4 mm), can be used to perform ILAM and accurately target and eliminate arrhythmic substrates.

Comparatively, ILAM with a high-density mapping catheter allowed for identification of regions critical to support multiple AFL circuits within the RA at a safe distance from the phrenic nerve, that conventional activation mapping did not. Subsequent ablation with an ST SF FJ ablation catheter (Biosense Webster, Irvine, CA) resulted in the successful elimination of atrial arrhythmias without damage to the phrenic nerve. This case suggests that the use of ILAM can be an effective and efficient method for treating AFL in patients with congenital heart disease, warranting further systematic study.

The patient tolerated the ablation well and there was no evidence of phrenic nerve damage by symptoms, nor hemidiaphragm elevation on chest radiograph. She has not had any recurrence of SVT in the 6 months post ablation.

Conclusion

This case report demonstrates that ILAM with the use of a high-density mapping catheter may be a useful tool for accurately targeting arrhythmias in patients with congenital heart disease. This method can be used to safely eliminate reentrant atrial arrhythmias while avoiding other critical structures. These results suggest that ILAM and catheter ablation are safe and effective methods for treating AFL in patients with congenital heart disease. Further research is needed to determine the optimal approach for eliminating AFL in these patient populations.

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