Slow pathway modification in an adult patient with unrepaired partial atrioventricular canal defect

Emily Cendrowski, MD, Mathew D. Hutchinson, MD, FACC, FHRS

From the Division of Cardiovascular Medicine, University of Arizona College of Medicine-Tucson, Tucson, Arizona.

Introduction

Catheter ablation of atrioventricular nodal reentrant tachycardia (AVNRT) is anatomically based. The typical anatomical relationships that guide ablation are often displaced in patients with congenital heart disease (CHD), thereby increasing procedural complexity and risk. We describe a case of slow pathway modification in an adult with unrepaired partial atrioventricular canal defect.

Case report

A 36-year-old woman presented with recurrent palpitations associated with fatigue and dyspnea. Her palpitations occurred sporadically over the prior 6 years. One episode required emergency department admission, at which time she was in a narrow complex supraventricular tachycardia (SVT) 149 beats per minute, which was converted with intravenous diltiazem (Figure 1). Over the following years she had progressive exertional dyspnea that limited exercise. Echocardiography revealed an ostium primum atrial septal defect (ASD), cleft mitral valve with mild regurgitation, and moderate right atrial and right ventricular (RV) chamber enlargement with normal RV systolic function. Cardiac catheterization revealed a Qp:Qs 3.75:1 with normal pulmonary artery pressures. Sotalol 80 mg twice daily and diltiazem failed to suppress her arrhythmia. She was referred for electrophysiology study and catheter ablation prior to surgical ASD closure.

The procedure was performed under conscious sedation and systemic anticoagulation was initiated prior to catheter introduction. Quadripolar catheters were placed at the atrioventricular (AV) junction and the RV apical septum; a decapolar catheter was placed in the coronary sinus (CS). A duodecapolar catheter (PentaRay; Biosense Webster, Irvine, CA) was positioned in the high right atrium during programmed stimulation. The location of the primum defect was contoured and reconstructed using intracardiac echocardiography (Figure 2).

Dual AV nodal physiology was present (fast pathway effective refractory period [ERP] 500 ms/320 ms), and a short RP SVT was reproducibly induced with a single extrastimulus from the CS (600 ms/300 ms). The tachycardia cycle length (TCL) was 450 ms with a septal VA time of 56 ms. A V-A-H response was seen after ventricular overdrive pacing from the RV apical septum, and the postpacing interval was 175 ms longer than the TCL (Figure 2). These data confirmed a diagnosis of typical AVNRT.

Detailed atrial activation mapping was performed during SVT with the duodecapolar catheter. The earliest atrial activation during tachycardia, presumed to represent the anatomical location of the fast AV nodal pathway, was located at the mid-anterior CS approximately 1 cm from the ostium. This region was remapped with ventricular pacing during sinus rhythm, and atrial activation was identical to that seen with tachycardia. The His bundle electrograms were displaced inferoposteriorly from their usual anatomical location.

Empiric ablation of the slow pathway was initially performed during sinus rhythm with a 6 mm cryocatheter

KEY TEACHING POINTS

- Patients with partial atrioventricular (AV) canal defects have inferior and posterior displacement of the AV conduction system that complicates catheter ablation procedures.
- The integration of intracardiac echocardiography and electroanatomical mapping is useful in demonstrating complex anatomical relationships in patients with congenital heart disease.
- Patients with partial AV canal defects have slow AV nodal pathways that are located outside of Koch's triangle, which can be localized with activation mapping during atypical AVNRT.

KEYWORDS Congenital heart disease; Ostium primum atrial septal defect; Atrioventricular nodal reentrant tachycardia; Catheter ablation; Intracardiac echocardiography

(Heart Rhythm Case Reports 2022;8:433–436)

Funding: None. Disclosures: The authors have no conflicts of interest to disclose. **Address reprint requests and correspondence:** Dr Mathew D. Hutchinson, Professor of Medicine, University of Arizona College of Medicine-Tucson, 1501 N. Campbell Ave, 4142B, Tucson, AZ 85724. E-mail address: MathewHutchinson@shc.arizona.edu.

https://doi.org/10.1016/j.hrcr.2022.03.015

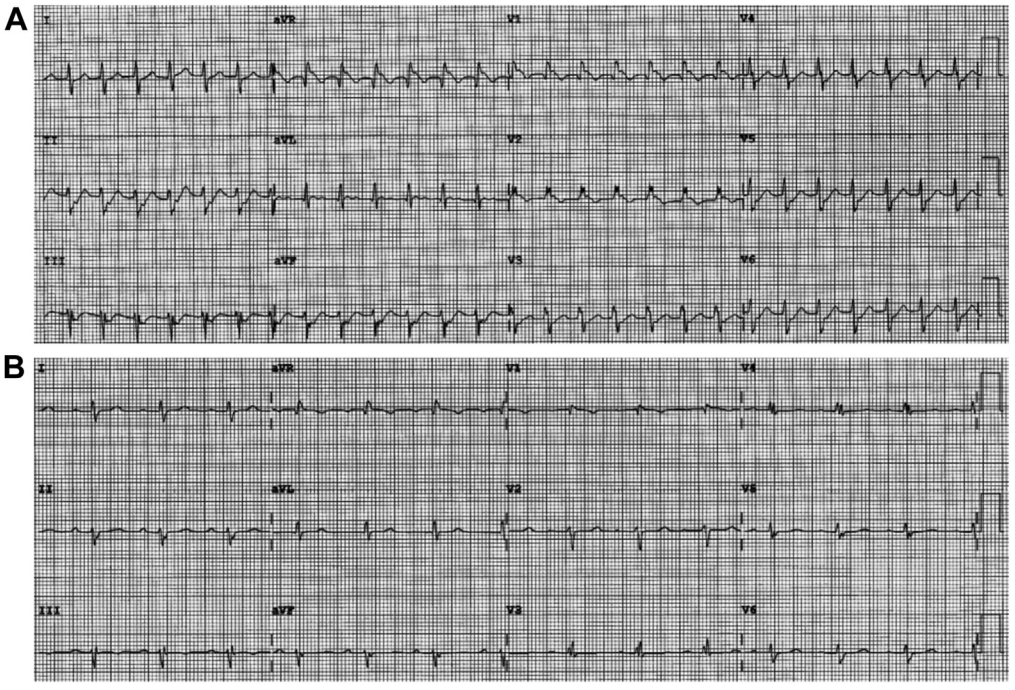

Figure 1 Twelve-lead electrocardiograms of the clinical tachycardia (**A**) and sinus rhythm (**B**).

(FreezorMax; Medtronic, Minneapolis, MN) targeting the region between the inferior CS lip and the tricuspid valve annulus, inferior to the ASD. Peak negative temperatures of -80°C were achieved within 20 seconds. Repeat atrial stimulation was repeated after cryoadhesion; however, AVNRT remained easily inducible with single atrial extrastimuli. An additional cryo lesion was delivered slightly more inferiorly and posteriorly on the floor of the CS ostium. Both regions of ablation are depicted in Figure 2. Stimulation during cryoablation induced a second, long RP SVT at 550 ms. Ventricular overdrive pacing from the RV apical septum produced a pseudo-V-A-A-H response, and the postpacing interval was 180 ms longer than the TCL. These data were most consistent with a diagnosis of atypical AVNRT. Repeat atrial activation mapping demonstrated a broad region of early activation in the mid cavotricuspid isthmus (Figure 3). Given the distance of the putative slow pathway location from the fast AV nodal pathway in the CS, we targeted this region with radiofrequency ablation. A total of 3 lesions were delivered at 30 W, targeting a sustained 15 ohm impedance drop for 60 seconds. After ablation, persistent slow pathway conduction with a jump was noted; however, neither AV nodal echo beats nor sustained AVNRT were seen at baseline or during infusion of isoproterenol at 3 mcg/min. Specifically, the fast pathway ERP was 500 ms/260 ms and AV nodal ERP was 500 ms/240 ms after ablation.

Two months after ablation, the patient underwent pericardial patch closure of the primum ASD, repair of the cleft mitral valve, and tricuspid valve annuloplasty. The patient tolerated surgery well with no complications. The PR interval was 160 ms postoperatively, which was unchanged. Over a 22-month follow-up, she reports a single 1-hour episode of palpitations.

Discussion

This is the first report, to our knowledge, describing slow pathway mapping in an adult patient with an unrepaired partial AV canal defect. Such patients are rare, as they often present for definitive repair during childhood. Inferior and posterior displacement of the compact AV node and penetrating His bundle is common, although the exact location varies depending upon the size of the defect.[1] The largest study reporting slow pathway modification in 109 congenital heart patients included 30 patients with septal defects (ostium secundum 28, partial AV canal 1, total anomalous pulmonary venous return 1).[2] The authors described a general strategy of ablation inferior to the CS ostium. Compared with simple forms of CHD, the complex CHD patients were more likely to demonstrate the following: (1) atypical AVNRT (28% vs 11%), (2) SVT recurrence (18% vs 10%), and (3) need for permanent pacing after ablation (10% vs 0%).[2] Another report described slow pathway modification via trans-septal approach in a patient in whom the nature of the repair excluded the relevant anatomical structures from a right-sided approach.[3] In selected primum defect patients, the slow pathway may be located superiorly, near the conventional apex of Koch's triangle.[4]

In our case, the combination of intracardiac echocardiography and electroanatomical mapping was useful to define the relevant anatomical relationships. The location of the

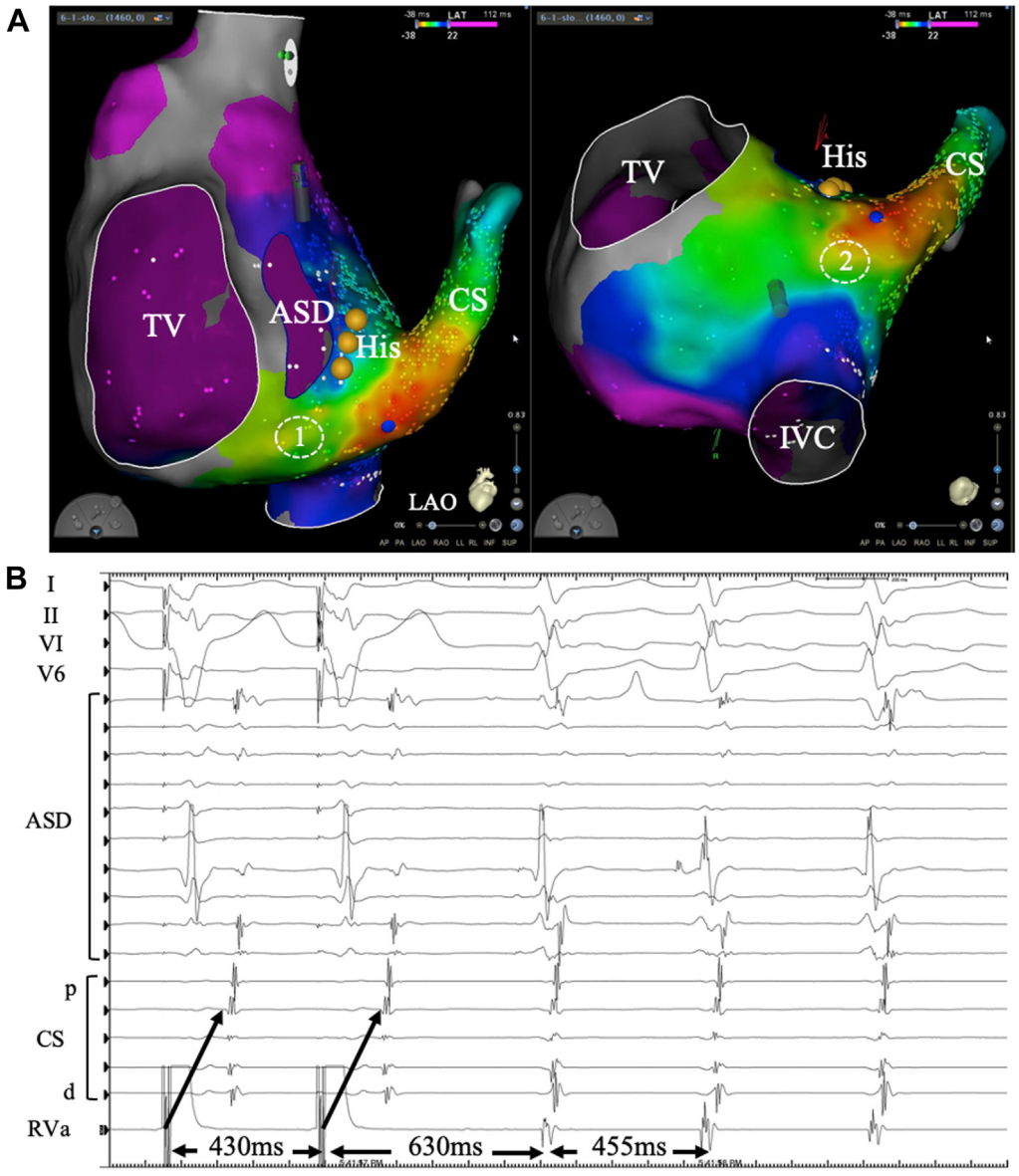

Figure 2 **A:** Activation mapping obtained during supraventricular tachycardia (SVT) #1, with broad early activation at the anterior-inferior lip of the coronary sinus. The atrial septal defect (ASD) location is manually contoured using intracardiac echocardiography. The location of His bundle electrograms (*yellow dots*) is displaced inferoposteriorly from its usual location by the ASD. The site of earliest fast pathway activation is represented by the blue dot. The duodecapolar catheter (PR) is positioned along the superior septal tricuspid annulus. The 2 areas of empiric cryoablation are represented by the stippled regions 1 and 2. **B:** Ventricular overdrive pacing is performed during SVT #1. See text for details. CS = coronary sinus; IVC = inferior vena cava; LAO = left anterior oblique; TV = tricuspid valve.

ASD was contoured manually using 2-dimensional echo slices, and multielectrode anatomical mapping provided a rich 3-dimensional geometric reconstruction of the right atrium and CS with minimal fluoroscopy. The electroanatomical data detailing location of the conduction system was also useful for the cardiac surgeon in planning the ASD closure and mitral repair.

Previous reports of slow pathway ablation in CHD patients describe empiric ablation, while in our case the induction of atypical AVNRT allowed detailed mapping of the slow pathway location. It is possible that injury to the slow pathway from the initial adjacent cryo lesions may have facilitated induction of atypical AVNRT in this case. The location of our patient's slow pathway, the mid cavotricuspid isthmus, was displaced further from the septum than previously reported. The use of any specific approach is limited by variability in the morphology of the specific congenital lesion, the age of presentation, and the presence of associated congenital abnormalities. For future primum ASD patients in which slow pathway mapping is not possible, extending the site of empiric ablation more inferiorly as an initial approach prior to targeting sites closer to the fast pathway may enhance procedural safety and efficacy.

Since His synchronous ventricular ectopics were not delivered during tachycardia, a concealed accessory

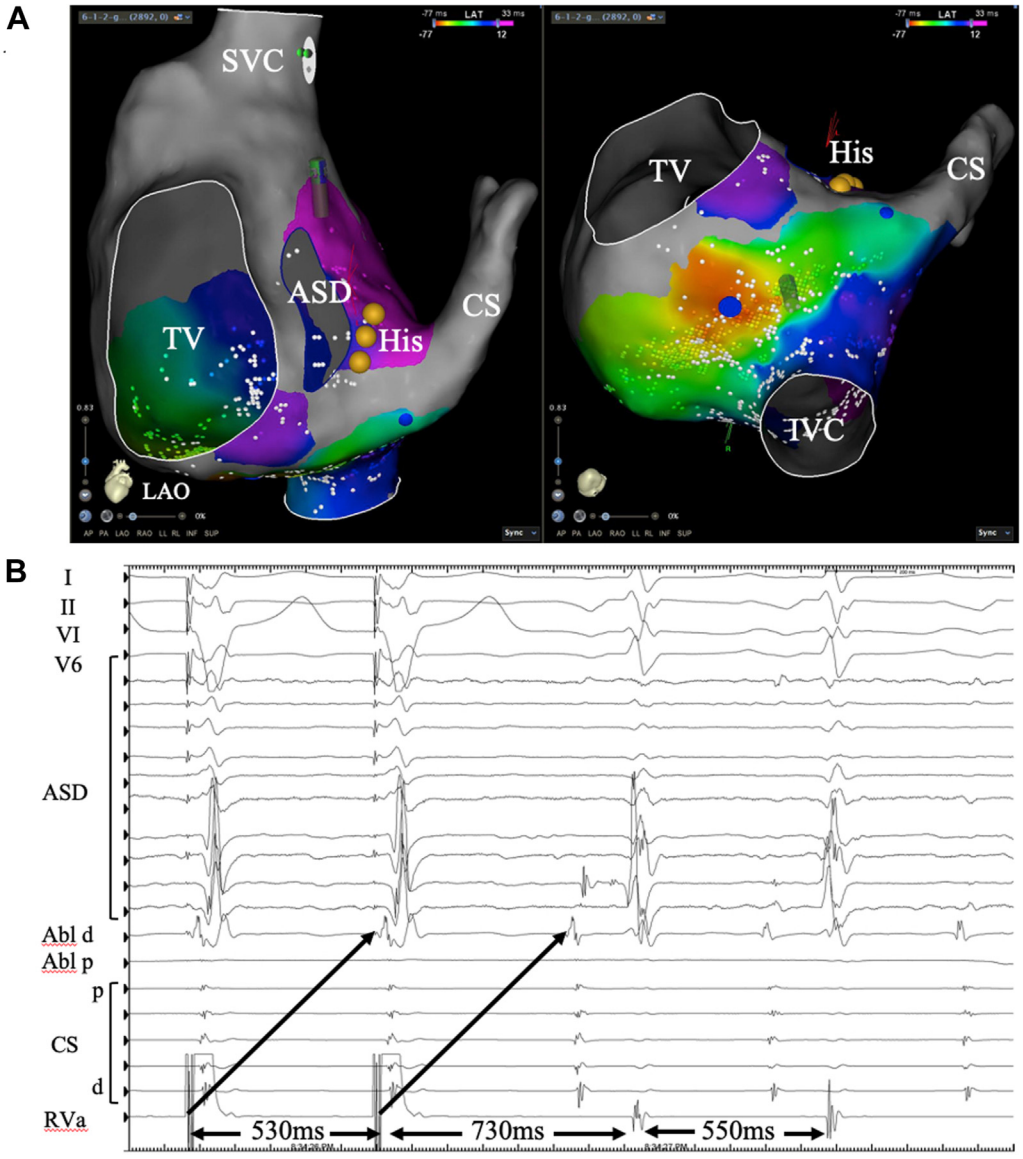

Figure 3 **A:** Activation mapping obtained during supraventricular tachycardia (SVT) #2, with broad early activation in the mid cavotricuspid isthmus. The putative sites of earliest fast and slow pathway activation are represented by the blue dots. The duodecapolar catheter (PR) is positioned along the superior septal tricuspid annulus and the cryo catheter is located at the inferior septal tricuspid annulus. **B:** Ventricular overdrive pacing is performed during SVT #1. See text for details. Abbreviations as in Figure 2.

pathway with decremental conduction could be completely excluded. However, the intermittent nature of the patient's arrhythmia, the later age of presentation, the presence of typical AVNRT induction, and the response to ventricular overdrive pacing all strongly argue for atypical AVNRT as the mechanism.

Conclusion

To our knowledge, this the first report of AVNRT ablation in an adult patient with an unrepaired partial AV canal defect. The induction of atypical AVNRT allowed more precise mapping and ablation of the slow pathway. Our report both provides an important reference for CHD patients undergoing slow pathway modification and underscores the broader implication that, when possible, detailed activation mapping can delineate unusual sites for successful ablation.

References

1. Thiene G, Wenink AC, Frescura C, et al. Surgical anatomy and pathology of the conduction tissues in atrioventricular defects. J Thorac Cardiovasc Surg 1981; 82:928–937.
2. Papagiannis J, Beissel DJ, Krause U, et al. Atrioventricular nodal reentrant tachycardia in patients with congenital heart disease: outcome after catheter ablation. Circ Arrhythm Electrophysiol 2017;10:e004869.
3. Stoyanov MK, Shalganov TN, Protich MM, Balabanski TL. Selective slow pathway ablation using transseptal approach in a patient with surgically corrected partial atrioventricular canal defect and atrioventricular nodal reentrant tachycardia of the common type. Europace 2010;12:756–757.
4. Lin JF, Li YC, Yang PL, Tang JF. Ablation of atrioventricular nodal reentrant tachycardia in a patient with reversal of slow and fast pathways inputs into the atrioventricular node. Pacing Clin Electrophysiol 2012;35:e17–e19.