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

Short title: Slow pathway ablation in primum ASD

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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, 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 six years. One episode required emergency department admission at which time she was in a narrow complex supraventricular tachycardia (SVT) 149bpm, which was converted with intravenous diltiazem. (Figure 1) Over the following years she had progressive exertional dyspnea which limited exercise. Echocardiography revealed an ostium primum atrial septal defect (ASD), cleft mitral valve with mild regurgitation, and moderate right atrial (RA) and right ventricular (RV) chamber enlargement with normal RV systolic function. Cardiac catheterization revealed a Qp:Qs 3.75:1 with normal PA pressures. Sotalol 80mg 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 AV junction and the RV apical septum; a decapolar catheter was placed in the coronary sinus. A duodecapolar catheter (Pentaray, Biosense Webster) 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 ERP 500ms/320ms), and a short RP SVT was reproducibly induced with a single extrastimulus from the coronary sinus (600ms/300ms). The tachycardia cycle length (TCL) was 450ms with a septal VA time of 56ms. A V-A-H response was seen after ventricular overdrive pacing from the RV apical septum, and the post-pacing interval was 175ms 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 (FP), was located at the mid-anterior CS approximately one 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 infero-posteriorly from their usual anatomical location.

Empiric ablation of the slow pathway was initially performed during sinus rhythm with a 6mm cryocatheter (FreezorMax, Medtronic) 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
550msec. Ventricular overdrive pacing from the RV apical septum produced a pseudo-V-A-A-H response, and the post-pacing interval was 180ms 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 coronary sinus, we targeted this region with radiofrequency ablation. A total of 3 lesions were delivered at 30W 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/minute. Specifically, the fast pathway ERP was 500ms/260ms and AV nodal ERP was 500ms/240ms 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 interval was 160ms 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. The largest study reporting slow pathway modification in congenital heart patients included 30 patients with septal defects (ostium secundum 28, partial AV canal 1, total anomalous pulmonary venous return 1). The authors described a general strategy of ablation inferior to the coronary sinus ostium. Compared with simple forms of CHD, the complex CHD patients were more likely to demonstrate: 1) atypical AVNRT (28% vs. 11%), 2) SVT recurrence (18% vs. 10%), and 3) need for permanent pacing after ablation (10% vs. 0%).

Another report described slow pathway modification via transseptal approach in a patient in whom the nature of the repair excluded the relevant anatomical structures from a right sided approach. In selected primum defect patients, the slow pathway may be located superiorly, near the conventional apex of Koch’s triangle.

In our case, the combination of ICE and electroanatomical mapping was useful to define the relevant anatomical relationships. The location of the ASD was contoured manually using two-dimensional echo slices, and multielectrode anatomical mapping provided a rich three-dimensional geometric reconstruction of the right atrium and coronary sinus 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 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.

Conclusions
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
Figure 1: 12-lead electrocardiograms of the clinical tachycardia (A) and sinus rhythm (B).
Figure 2: (A) Activation mapping obtained during SVT #1, with broad early activation at the anterior-inferior lip of the coronary sinus. The ASD location is manually contoured using ICE. The location of His bundle electrograms (yellow dots) is displaced infero-posteriorly 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 two 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.
Figure 3: (A) Activation mapping obtained during 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.
Key Teaching Points

- Patients with partial 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.