Absence of a resetting phenomenon suggests that a sling works as a part of the supraventricular tachycardia circuit involving twin atrioventricular nodes: A case of corrected transposition of the great arteries

Daiji Takeuchi, MD,* Morio Shoda, MD,† Kazuhiro Takahashi, MD,* Toshio Naknishi, MD*

*From the *Department of Pediatric Cardiology, and †Department of Cardiology, Tokyo Women's Medical University Tokyo, Japan.*

Introduction

In corrected transposition of the great arteries (CTGA), the presence of 2 atrioventricular nodes (twin AVNs), their Purkinje systems, and a specific conduction system (Mönckeberg sling) connecting them to each other has been demonstrated using histologic and electrophysiological techniques.[1,2]. Twin AVNs can induce supraventricular tachycardia (SVT) as a preoperative, perioperative, or postoperative arrhythmic complication. Therefore, preoperative electrophysiological evaluation is important.[3] However, clinical reports describing the electrophysiological properties of the sling are limited.[4,5] In this case report, we confirmed the presence of a sling between twin AVNs and determined that it functioned as a part of the reentrant circuit of SVT associated with the twin AVNs.

Case report

The patient was a 6-year-old girl with CTGA (SLL) with a perimembranous ventricular septal defect, a persistent left superior vena cava, 2 atrioventricular valves, and dextrocardia without native outflow tract stenosis who underwent surgical placement of a pulmonary artery band at 4 months of age. At 6 years of age, she developed SVT during cardiac catheterization conducted for evaluation prior to intracardiac repair; the SVT terminated by intravenous administration of adenosine triphosphate. An electrophysiological study was performed under general anesthesia to preoperatively evaluate the SVT and devise ablation therapy. A decapolar catheter was positioned in the right coronary sinus and the great cardiac vein. A bipolar catheter was positioned in the right side of the morphological left ventricle. Of 2 quadrupolar catheters with a deflectable tip, 1 catheter was positioned in the posterior His bundle (pHB) lesion on the posterior mitral valve annulus, whereas the other catheter was positioned in the anterior His bundle (aHB) lesion on the anterior mitral valve annulus. Baseline 12-lead electrocardiography results showed sinus rhythm without delta waves. High and low right atrial (RA) pacing induced 2 discrete non-preexcited QRS complexes (qrs pattern in lead I and rSr' in lead aVR during high RA pacing and low RA pacing; rS pattern in lead I and qR in lead aVR during low RA pacing), indicating the presence of twin AVNs (Figure 1A). Subsequently, His bundle (HB) electrography mapping was performed during sinus rhythm. As shown in Figures 1, 2B and C distinctive HB potentials (HBEs) with different HV times (38 milliseconds at the aHB and 31 milliseconds at the pHB) were identified at the high anterior and low posterior sites of the mitral valvular annulus, indicating that the HB was independently located at both the anterior and posterior aspects. During high RA pacing, the HV time at the aHB was constant, whereas the HV time at the pHB was shortened. In contrast, during low RA pacing, the HV time at the pHB was constant, whereas the HV time at the aHB was shortened. This suggested that the sinus beat coincided with antegrade conduction at the anterior and posterior atrioventricular nodes (aAVN and pAVN, respectively), resulting in a fusion beat. When atrial pacing was performed near 1 of the twin AVNs, the QRS complex morphology indicated pure antegrade conduction at 1 of the twin AVNs and bystander conduction at the other. The antegrade AVN conduction was smooth and decremental, with prolongation of the AH

KEYWORDS Corrected transposition of the great arteries; Twin atrioventricular nodes; Sling; Supraventricular tachycardia; Resetting phenomenon

ABBREVIATIONS **aAVN** = anterior atrioventricular nodes; **aHB** = anterior His bundle; **AVN** = atrioventricular node; **CS** = coronary sinus; **CTGA** = corrected transposition of the great arteries; **HB** = His bundle; **HBE** = His bundle potential; **LAO** = left anterior oblique; **pAVN** = posterior atrioventricular node; **pHB** = posterior His bundle; **RA** = right atria; **RAO** = right anterior oblique; **SVT** = supraventricular tachycardia; **Twin AVNs** = 2 atrioventricular nodes

(Heart Rhythm Case Reports 2015;1:220–224)

The first two authors contributed equally to this work.

Conflicts of interest: The authors declare no conflicts of interest. **Address reprint requests and correspondence:** Dr. Daiji Takeuchi, Department of Pediatric Cardiology, Tokyo Women's Medical University, 8-1 Kawadacho, Shinjuku-ku, Tokyo 162-8666, Japan. E-mail address: ptakeuci@hij.twmu.ac.jp.

http://dx.doi.org/10.1016/j.hrcr.2015.03.006

KEY TEACHING POINTS

- Two discrete narrow QRS complexes without preexcitation suggest the presence of twin atrioventricular nodes.
- Atrioventricular reciprocating tachycardia that uses a system of twin atrioventricular nodes as a reentrant circuit may be inducible.
- The sling that connects 2 distinctive atrioventricular conduction systems may be a part of a reentrant circuit, resulting in the absence of a reset phenomenon during ventricular pacing prior to the bundle of His anterograde activation.

interval without shortening of the HV time. The Wenckebach rate was 280 beats per minute at both the aAVN and pAVN during RA overdrive pacing. The effective refractory period at the AVNs was continuous during atrial pacing (basic cycle length, 500 milliseconds) with an atrial extrastimulus of 190 milliseconds at the aAVN and 240 milliseconds at the pAVN. Neither AVN showed an AH jump, which suggested that dual AVN physiology was not present. On ventricular pacing, the earliest retrograde atrial activation was observed at the aHB. The QRS morphology of clinical SVT (cycle length, 315 milliseconds) induced by programmed ventricular stimulation (Figure 2A) was identical to the QRS morphology during low RA pacing (Figure 1A), indicating that the anterograde AV conduction during SVT and low RA pacing was through the pAVN. Intracardiac electrography tracings during SVT showed an A-H-V sequence at the pHB and a V-H-A sequence at the anterior aHB, with the earliest atrial excitation occurring at the aHB (Figure 2A). These findings suggested that the SVT involved the pAVN as an anterograde limb and the aAVN as a retrograde limb. Ventricular overdrive pacing during the SVT reset the tachycardia without changing the retrograde atrial activation sequence or the His-atrial interval of 49 milliseconds at the aHB. The His-atrial interval during ventricular pacing was the same as that during the SVT. However, the earliest ventricular-to-aHB interval was prolonged from 89 milliseconds to 152 milliseconds (Figure 2B). A premature ventricular extrastimulus before the posterior HB potential (pHBE) with an H-S interval of 297 milliseconds did not reset the AA interval (Figure 3A, left panel), and the stimulus before the pHBE refractory period with a short H-S interval of 285 milliseconds advanced the retrograde atrial conduction at the aHB (Figure 3A, right panel). The QRS morphology of the H-S intervals with the durations of 297 milliseconds and 285 milliseconds was relatively narrow, resembling that during ventricular overdrive pacing (Figure 1A). These findings suggested that there was a connecting sling between the pAVN and aAVN (Figure 3B). Because the pAVN was predominantly involved during sinus rhythm and the aAVN was a retrograde conducting

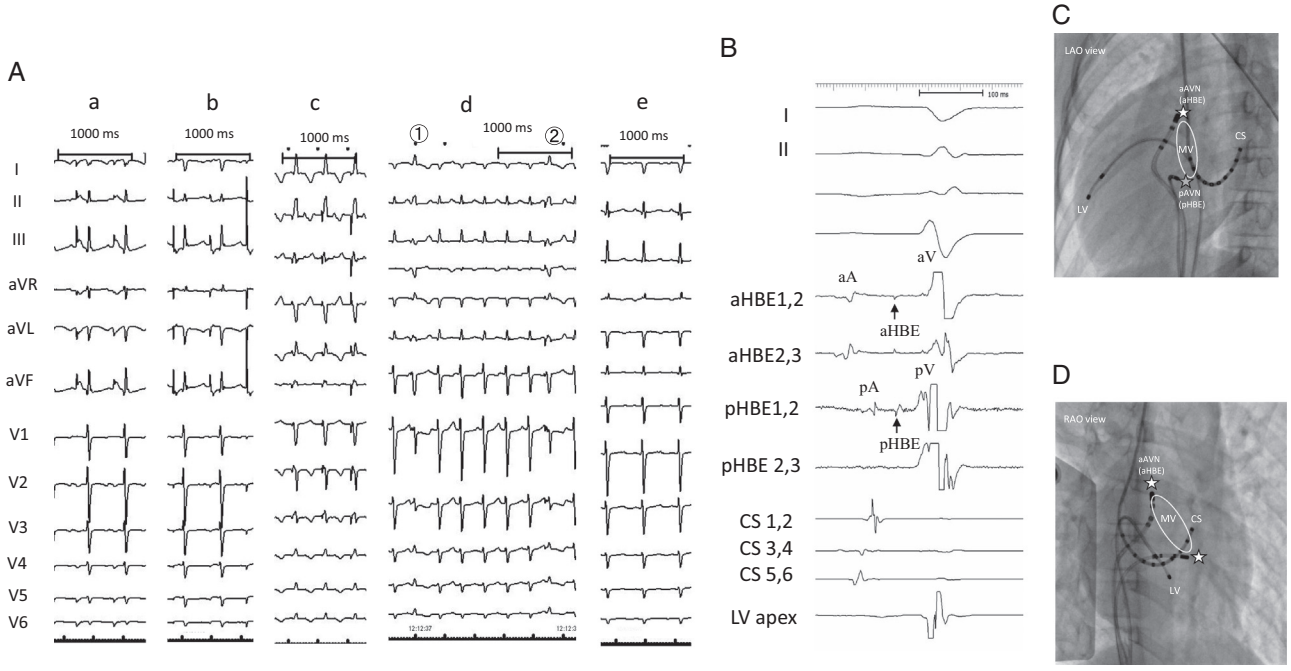

Figure 1 **A:** QRS morphologies in the 12-lead electrocardiogram. **a:** Preablation QRS morphology of high atrial pacing. **b:** Preablation QRS morphology of low atrial pacing. **c:** Preablation QRS morphology of ventricular pacing during sinus rhythm. **d:** Preablation QRS morphology upon delivering a ventricular extrastimulus during tachycardia. **1:** H-S interval of 297 milliseconds; **2:** H-S interval of 285 milliseconds. **e:** Postablation QRS morphology with pure posterior atrioventricular node conduction. **B:** Two simultaneously recorded His bundle potentials with different HV intervals during sinus rhythm. aHBE-V time, 38 milliseconds; pHBE-V time, 31 milliseconds. **C:** Left anterior oblique (LAO) fluoroscopic view illustrating locations on the anterior and posterior His bundles where potentials were recorded. **D:** Right anterior oblique (RAO) fluoroscopic view illustrating locations on the anterior and posterior His bundles where potentials were recorded. aHBE = anterior His bundle potential; aAVN = anterior atrioventricular node; CS = coronary sinus; LV = anatomical left ventricle; MV = mitral valve; pAVN = posterior atrioventricular node; pHBE = posterior His bundle potential.