Atypical Slow-Slow Atrioventricular Nodal Reentrant Tachycardia with Use of a Superior Slow Pathway

Yoshiaki Kaneko,1 MD, Tadashi Nakajima,1 MD, Takashi Iizuka,1 MD, Shuntaro Tamura,1 MD, Hiroshi Hasegawa,1 MD and Masahiko Kurabayashi,1 MD

Summary

We report a case of atypical slow-slow atrioventricular nodal reentrant tachycardia (AVNRT) utilizing a superior slow pathway as a retrograde limb. The standard electrophysiological criteria confirm the diagnosis of this AVNRT by successfully excluding a diagnosis of atrial tachycardia and atrioventricular reentrant tachycardia. The earliest atrial activation during tachycardia was found at the interatrial septum 17.5 mm superior to the site identified during retrograde conduction with the fast pathway. The tachycardia was not inducible after ablation at the right posterior septum, consistent with successful ablation of the typical slow pathway.

Key words: Ablation, Electrophysiologic study

The superior slow pathway (SP) is a variant of the SP that originates from the compact node and extends superiorly outside of Koch’s triangle.1 Originally, the superior SP was proposed as the putative retrograde limb of the reentry circuit responsible for superior fast-slow atrioventricular (AV) nodal reentrant tachycardia (NRT).1 Inoue discovered superior extensions of the human compact AV node, which is hypothesized to be involved in “superior SP” conduction.2

Editorial p.199

Recently, we have reported a case of atypical fast-slow AVNRT using a SP extending superoanterior right atrium.3 Understanding of the superior SP and AVNRT with variants of the SP is increasing. This report describes a case of atypical slow-slow AVNRT with a superior SP.

Case Report

A 61-year-old woman who had a history of multiple episodes of paroxysmal supraventricular tachycardia underwent electrophysiological studies and catheter ablation. During tachycardia, a 12-lead electrocardiogram showed a short RP tachycardia (Figure 1A). Atrial single extrastimulation elicited a smooth curve of antegrade atrioventricular (AV) nodal conduction, but ventricular single extrastimulation showed a discontinuous curve of ventriculoatrial (VA) conduction with decremental properties and the earliest site of atrial activation in the His bundle (HB) region, compatible with a suspicious superior slow pathway (SP) rather than a fast pathway (FP) (Figure 2). The short RP tachycardia that was documented clinically was reproducible with atrial burst stimulation after a long atrio-His interval (Figure 1B) or an A-A-V activation sequence (Figure 1C). Atrial activation during tachycardia was earlier in the HB recording than in the coronary sinus (CS) recordings (Figure 1B, C and D). Atrio-His and His-atrial intervals during tachycardia were 204 and 48 msec, respectively (Figure 1D). During ongoing tachycardia, second-degree AV block did not occur, and an intravenous injection of ATP was not performed. The interval between the onset of QRS and the earliest atrial deflection of 15 msec was too short to support the diagnosis of AV reentrant tachycardia (AVRT) with the AV accessory pathway.4 Ventricular overdrive pacing during tachycardia represented two types of response, including reproducible termination of the tachycardia without premature atrial capture5 (Figure 3A) and successful entrainment capturing the atria with a 1:1 ratio, followed by reinitiation of tachycardia with an initial V-A-V activation sequence6 (Figure 3B). This finding excluded the diagnosis of atrial tachycardia. In the transition zone, which was characterized by progressive QRS fusion immediately after ventricular overdrive pacing of the tachycardia, the finding of no perturbation of the atrial cycles and increasing spike-atrial intervals was inconsistent with a diagnosis of AVRT (Figure 2A).7 Ventricular single extrastimuli delivered based on the timing of HB refractoriness during tachycardia did not reset the atrial cycle length (CL), inconsistent with orthodromic reciprocating tachycardia with the noventricular/fascicular fiber as the retrograde limb.8 These findings confirmed the diagnosis of AVNRT. The initial A-A-V activation sequence on atrial induction of...
Immediatel  

earliest site of atrial acti 

cw 

tach 

cated b 

His b 

cond 

v 

depth. 

A: The polarity of P waves in leads II, III, and aVF is negative with a 
wallow depth. 

B: Burst stimulation at an S-S cycle length of 230 msec induced tachycardia after antegrade conduc 
tion via a slow pathway, evidenced by a long atrio-His interval of 219 mec (bidirectional arrows). During tachycardia, 
His bundle deflections (H) are visible, but tiny atrial deflections (A) at the His bundle electrogram (HBE) may be su 
perimposed by larger ventricular deflections (V). C: Burst stimulation at an S-S cycle length of 240 msec also induced 
tachycardia after an A-A-V activation sequence. On the 2nd and 3rd cycles of tachycardia, large atrial deflections are 
visible immediately far-field, showing ventricular deflections with no recording of H because of slight dislocation of 
the HBE catheter to the atrial side. All recordings were obtained at baseline. II and V1 indicates surface electrocardio 
gram; HRA1-2, distal high right atrium; HBE1-2 and 3-4, distal to the proximal His bundle region; CS15-16 to 1-2, 
proximal to the distal coronary sinus recording; and RVA, right ventricular apex.

Figure 1. 12-lead electrocardiogram obtained during tachycardia (A) and intracardiac recordings of two types of in 
duction of tachycardia with burst stimulation from the high right atrium (HRA) (B and C) and tachycardia with a re 
cording of clear electrogram of His-bundle (D). A: The polarity of P waves in leads II, III, and aVF is negative with a 

Figure 2. Ventriculoatrial conduction curve estimated by RVA extrastimulus method (S1-S2) after an S1-S1 basic 
cycle length of 400 ms (A) and intracardiac recordings representing a small jump of His (H)-atrial (A) interval (indi 
cated by bidirectional horizontal arrows) induced by RVA extrastimulation, representing a switching of retrograde 
conduction from fast pathway (B) to a suspicious superior slow pathway (C). A: Horizontal axis represents an S1-S2 
coupling interval, and longitudinal axis represents an S2-A2 interval measured at HBE1-2. B: During a burst stimula 	ion of an S1-S1 cycle length of 400 ms, retrograde conduction via a fast pathway is observed, characterized by the 
earliest site of atrial activation during His-bundle electrogram (HBE) recording and a short ventriculoatrial interval. 
Immediately after extrastimulation with an S1-S2 coupling interval of 280 ms, retrograde conduction via the fast path 
way also occurs. C: Immediately after extrastimulation of an S1-S2 coupling interval of 270 ms, an H-A interval is 
abruptly prolonged with the earliest site of atrial activation during HBE recording, suspicious with retrograde conduc 
tion via a superior SP. Other abbreviations are the same as in Figure 1.
Figure 3. Intracardiac recordings of two types of response during RVA overdrive pacing of tachycardia (A and B), CARTO activation maps of the right atrium in the left anterior oblique view during tachycardia and during retrograde conduction via the fast pathway, respectively (C and D), and a schematic illustration of the reentry circuit of the tachycardia. A: Immediately after right ventricular apical (RVA) overdrive pacing of tachycardia at an S-S cycle length of 240 ms, 2 QRS complexes show fusion (F1-F2), and thereafter the morphologic features of the QRS become stable (S1-S7). In the transition zone (bidirectional arrow), the finding of no perturbation of the atrial cycles and increasing spike-atrial intervals is inconsistent with a diagnosis of AV reentrant tachycardia but is consistent with a diagnosis of atrioventricular nodal reentrant tachycardia. Immediately after a slight increase in the atrial cycle in response to the 6th ventricular stimuli was observed, probably because of decremental delay of the retrograde conduction over the superior slow pathway, the 7th stimulus showed retrograde blockage at the superior slow pathway, followed by termination of tachycardia (dotted arrows). In response to the 8th ventricular stimulus, both a short ventriculatrial interval and the earliest site of retrograde atrial activation in the His bundle region (HBE 1-2) were observed, consistent with retrograde conduction over a fast pathway (thin arrow), followed by 2:1 VA conduction over that pathway. The numbers between the atrial electrogram at the high right atrium and the pacing stimuli at the RVA indicate the cycle length in milliseconds. B: During ventricular entrainment pacing of the tachycardia, the atrium is captured via the fast pathway, followed by reinitiation of tachycardia. C and D: Yellow tags show the recording sites of the His bundle electrogram. See the text for further explanation. E: Dot arrows indicate conduction pathways including a typical slow pathway (SP) as the antegrade limb, a superior SP as the retrograde limb, and the atrial muscle bridging the two SPs. CSos indicates the ostium of coronary sinus; and FP, fast pathway. Other abbreviations are the same as in Figure 1.
tachycardia (Figure 1C) can be explained by the antegrade block at the lower common pathway (LCP) in the AV node below the reentry circuit. Furthermore, the retrograde limb of the reentry circuit was identified as the superior SP according to the following findings: Three-dimensional activation mapping of the tachycardia showed the earliest site of atrial activation at the interatrial septum 17.5 mm superior to the site identified during retrograde conduction with the FP (Figure 3C and D). In addition, the antegrade limb was identified as the typical SP based on the following 3 findings. First, the atrial-His interval during tachycardia was adequate. Second, atrial entrainment mapping of tachycardia in the right atrium showed that the shortest interval between the spike and the HB electrogram was obtained during entrainment pacing at the proximal CS. Finally, tachycardia was not inducible after ablation at the right posterior septum, consistent with successful ablation of the typical SP. Accordingly, we diagnosed this tachycardia as slow-slow AVNRT with the typical SP as the antegrade limb and the superior SP as the retrograde limb (Figure 3E). No recurrence of tachycardia was observed during 6-month follow-up.

Discussion
The timing of atrial versus ventricular deflection and the earliest site of atrial activation during tachycardia are important clues to the diagnosis of the mechanism of supraventricular tachycardia. In the present case, intracardiac electrograms obtained during tachycardia were characterized by almost simultaneous activation of atrial and ventricular deflection (A-on-V), and the earliest site of atrial activation in the HB region was similar to the typical appearance of typical slow-fast AVNRT with the FP as a retrograde limb of the reentry circuit of tachycardia. Nevertheless, the superior SP was identified as the retrograde limb, based on the finding of the earliest site of atrial activation during tachycardia superior to the HB region. This finding was not reported in previous studies of slow-fast AVNRT. However, because activation mapping during slow-fast tachycardia was confined inside Koch’s triangle and/or did not occur superior to the HB lesion in previous studies, cases with the earliest site of atrial activation during tachycardia superior to the HB region may be underdiagnosed. The A-on-V appearance may be attributable to the LCP through which the antegrade conduction time might cancel out retrograde conduction via the superior SP. Eccentric atrial activation during tachycardia also may support a diagnosis of AT or AVRT. However, we could successfully exclude a diagnosis of AT and AVRT and confirm the diagnosis of AVNRT with the standard criteria we applied.

To treat AVNRT with RF ablation, we targeted not the superior SP but the typical SP for the following reasons. First, ablation of the typical SP is established for a cure of AVNRT using the typical SP. Second, another tachycardia with long RP and the earliest site of atrial action at the HB region, compatible with superior type fast-slow AVNRT using the FP as the antegrade limb and the superior SP as the retrograde limb, was induced with atrial stimulation but was not sustained because of an antegrade conduction block at the FP immediately after induction of tachycardia, even during isoproterenol infusion. These findings suggested that, because antegrade conductivity of the FP was too fragile to sustain the fast-slow AVNRT, additional ablation of the superior SP would not be needed after ablation of the typical SP.

Conclusion
To the best of our knowledge, this is the first report of successful ablation of an atypical slow-slow AVNRT with the superior SP as a retrograde limb.

Disclosure
Conflicts of interest: None.

References
1. Kaneko Y, Naito S, Okishige K, et al. Atypical fast-slow atrioventricular nodal reentrant tachycardia: Studies on retrograde conduction during isoproterenol infusion. Circulation 1995; 92: 883-8.
2. Inoue S. Atrial and atrioventricular junctional anatomy: Myocardial orientation and its heterogeneity. In: Hirao K, ed. Catheter ablation: A current approach on cardiac arrhythmias. 1st ed. Singapore: Springer Nature Singapore Inc; 2018: 3-10.
3. Kaneko Y, Nakajima T, Iizuka T, Tamura S, Kurabayashi M. A case of atypical fast-slow atrioventricular nodal reentrant tachycardia utilizing a slow pathway extending to the superoanterior right atrium. Int Heart J 2019; 60: 756-60.
4. Benditt DG, Pritchett EL, Smith WM, Gallagher JJ. Ventriculoatrial intervals: Diagnostic use in paroxysmal supraventricular tachycardia. Ann Intern Med 1979; 91: 161-6.
5. McGuire MA, Yap AS, Lau KC, et al. Posterior (“atypical”) atrioventricular junctional reentrant tachycardia. Am J Cardiol 1994; 73: 469-77.
6. Knight BP, Zivin A, Souza J, et al. A technique for the rapid diagnosis of atrial tachycardia in the electrophysiology laboratory. J Am Coll Cardiol 1999; 33: 775-81.
7. AlMahameed ST, Buxton AE, Michaud GF. New criteria during right ventricular pacing to determine the mechanism of supraventricular tachycardia. Circ Arrhythm Electrophysiol 2010; 3: 578-84.
8. Dandamudi G, Mokabberi R, Assal C, et al. A novel approach to differentiating orthodromic reciprocating tachycardia from atrioventricular nodal reentrant tachycardia. Heart Rhythm 2010; 7: 1326-9.
9. Mantovan R, Verlato R, Corrado D, Buia G, Haissaguerre M, Shah DC. Orthodromic tachycardia with atrioventricular dissociation: evidence for a nodoventricular (Mahaim) fiber. Pacing Clin Electrophysiol 2000; 23: 276-9.
10. Kaneko Y, Nakajima T, Iri T, Kato T, Iijima T, Kurabayashi M. Long RP tachycardia with an initial A-A-V activation sequence: what is the mechanism? J Cardiovasc Electrophysiol 2011; 22: 945-7.
11. Haines DE, Nath S, DiMarco JP, Lobban JH. Entrainment mapping in patients with sustained atrioventricular nodal reentrant tachycardia: insights into the sites of conduction slowing in the slow atrioventricular nodal pathway. Am J Cardiol 1997; 80: 883-8.
12. McGuire MA, Bourke JP, Robotin MC, et al. High resolution mapping of Koch’s triangle using sixty electrodes in humans with atrioventricular junctional (AV nodal) reentrant tachycardia. Circulation 1993; 88: 2315-28.
13. Katritsis DG, Ellenbogen KA, Becker AE. Atrial activation during atrioventricular nodal reentrant tachycardia: studies on retrograde fast pathway conduction. Heart Rhythm 2006; 3: 993-1000.