Successful cryoablation of an atypical slow-slow atrioventricular nodal reentrant tachycardia utilizing a superior slow pathway

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Introduction

The superior slow pathway (SP) is considered a variant of an SP that originates from the compact node and generally extends superiorly outside of the Koch triangle.[1] Originally, the superior SP was proposed as the retrograde limb of the reentry circuit responsible for superior fast-slow AV nodal reentrant tachycardia (AVNRT).[1] However, Kaneko and colleagues[2] recently reported a case of atypical slow-slow AVNRT using a superior SP.

Cryoablation allowing careful cryomapping of the target substrate before permanent ablation is an alternative to radiofrequency (RF) ablation of typical AVNRT, and it has a low risk of recurrence, with no incidence of AV block requiring permanent pacing.[3,4] Furthermore, cryoablation of atypical AVNRT was recently reported to be equally as safe and effective as that of typical AVNRT.[5]

KEY TEACHING POINTS

- Cryoablation is a safe and effective option for atypical slow-slow atrioventricular nodal reentrant tachycardia using a superior slow pathway.
- The cryomapping technique could safely confirm the critical site of the tachycardia.
- In the present case, a cryoablation lesion targeting the typical slow pathway region involved the inferior end of the superior slow pathway located close to the antegrade slow pathway.

Case report

A 54-year-old woman who complained of palpitations was referred for catheter ablation of a drug-refractory supraventricular tachycardia (SVT). The patient was free from any structural heart disease and comorbidities. The results of the laboratory testing, electrocardiogram during sinus rhythm (Figure 1A), and echocardiography were unremarkable. During the tachycardia, a 12-lead electrocardiogram revealed a short RP tachycardia with a ventricular rate of 115 beats per minute. This tachycardia exhibited a P-wave configuration positive in lead V_1 and negative in the inferior leads (Figure 1B).

The procedure was performed under local anesthesia. We approached from the right femoral vein and placed 10-pole catheters in the His bundle (HB) region and coronary sinus and 4-pole catheters in the high right atrium and right ventricular (RV) apex. The control atrio-His (AH) interval and H-V interval during sinus rhythm were 134 ms and 58 ms, respectively.

Atrial extrastimulation from the high right atrium elicited a smooth antegrade AV nodal conduction curve without an AH jump, and ventricular single extrastimulation also exhibited a smooth ventriculoatrial (VA) conduction curve with a decremental property and an earliest site of the atrial activation in the HB region (Figure 1C). No other sequences of the VA conduction were observed. Para-Hisian pacing exhibited an increase in the stimulus-atrial interval of fully ventricular paced vs His + ventricular capture complexes with no change in the atrial activation sequence, which was inconsistent with retrograde conduction by an AV accessory pathway or atrioventricular nodal reentrant tachycardia. Atrial extrastimulation induced a reproducible atrial echo beat without AH jump, and the atrial sequence of the retrograde conduction was identical to that during the RV apex stimulation (Figure 2A). Additionally, RV apex extrastimulation induced an identical retrograde conduction followed by antegrade fast pathway (FP) conduction. Those findings indicated that the retrograde conduction was compatible with that over the superior SP rather than the FP.

KEYWORDS Atrioventricular nodal reentrant tachycardia; Atypical slow-slow atrioventricular nodal reentrant tachycardia; Catheter ablation; Cryoablation; Superior slow pathway
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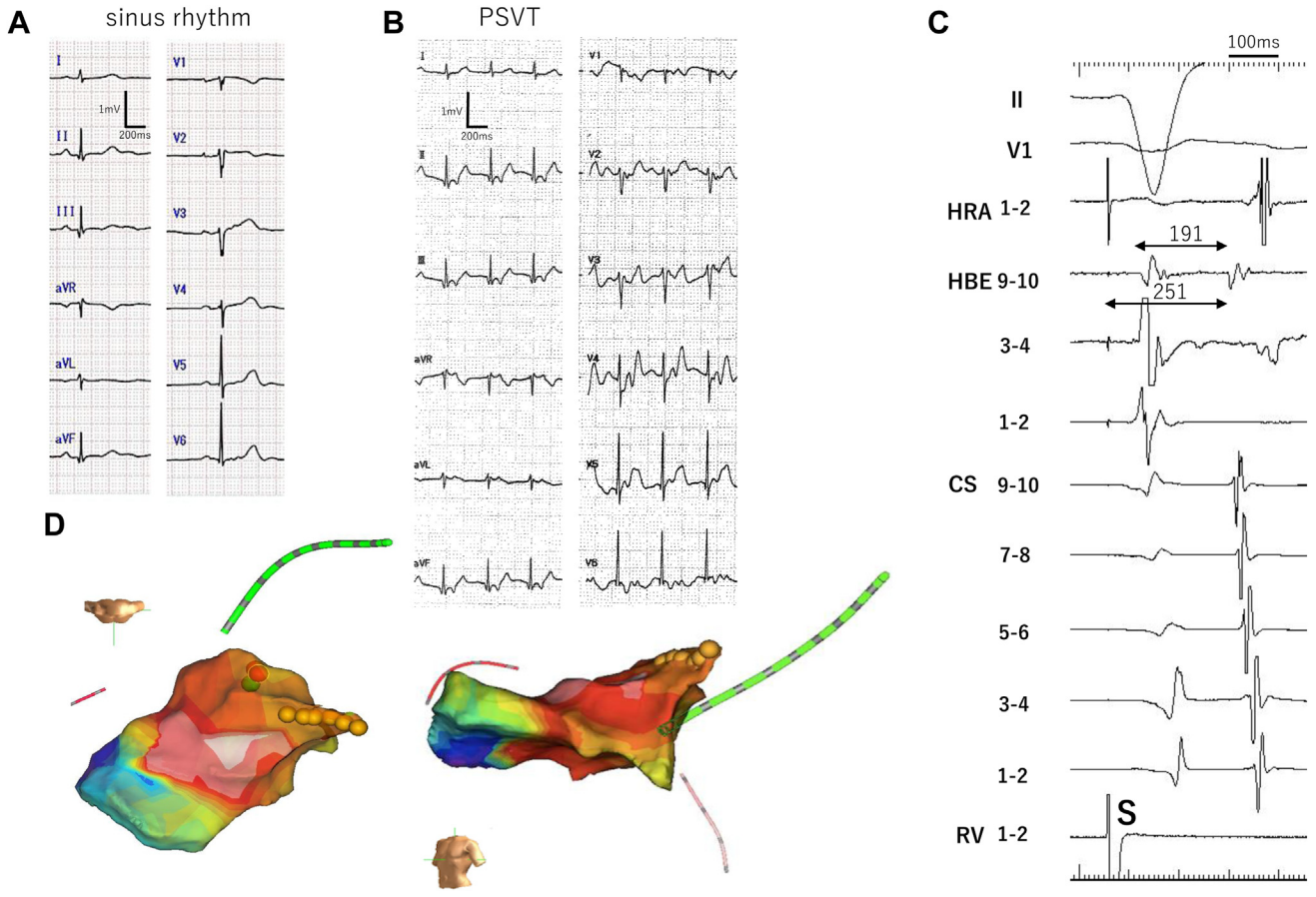

Figure 1 Twelve-lead electrocardiogram obtained during sinus rhythm (A) and paroxysmal supraventricular tachycardia (PSVT) (B). **A:** Sinus rhythm. The heart rate was 45 beats/min and no delta wave was observed. The PR interval and QRS width were 198 ms and 84 ms, respectively. **B:** PSVT. The heart rate was 115 beats/min and this tachycardia exhibited a narrow QRS complex (86 ms) and short RP interval. This tachycardia also exhibited a P-wave configuration positive in lead V_1 and negative in the inferior leads. **C:** Ventriculoatrial (VA) conduction followed by burst pacing from the right ventricle (RV) apex (S-S cycle length of 500 ms). The earliest site of the atrial activation was in the His bundle region. The SA interval and local VA interval were 251 ms and 191 ms, respectively. This VA conduction had a decremental property and no other sequences of the retrograde conduction were observed. **D:** Three-dimensional activation mapping of the anteroseptal right atrium during the ventriculoatrial conduction (EnSite Velocity; Abbott, Plymouth, MN). The earliest atrial activation site (*white area*) was on the interatrial septum slightly superior to the His bundle region (*yellow tag*). The diameter of the tags was 4 mm. CS = coronary sinus potential; HBE = His bundle electrogram; HRA = high right atrium; S = stimulation; TV = tricuspid valve.

The clinical short RP tachycardia was eventually induced by atrial triple extrastimulation under the administration of high-dose isoproterenol, and the tachycardia was initiated with an AH jump (Figure 2B). The atrial activation during the tachycardia was earlier in the HB region than in the coronary sinus, and the atrial activation sequence of the tachycardia was identical to that of the VA conduction described previously. The AH and His-atrial intervals during the tachycardia were 362 and 143 ms, respectively.

Ventricular overdrive pacing during the tachycardia was followed by the resumption of the tachycardia with an initial V-A-V activation sequence, which excluded a diagnosis of atrial tachycardia. Moreover, the postpacing interval – tachycardia cycle length and SA – VA times were 133 ms and 124 ms, which was inconsistent with atrioventricular nodal reentrant tachycardia. AH block without termination or a change of atrial cycle length during the AVNRT was observed, but it could be explained by antegrade block in the lower common pathway. Further, VA linking was observed during the tachycardia. Those findings confirmed the diagnosis of AVNRT.

Moreover, the 3-dimensional activation mapping of the VA conduction, which was identical to the retrograde conduction sequence of the clinical AVNRT, revealed that the earliest atrial activation site was on the interatrial septum slightly superior to the HB region (Figure 1D). Accordingly, we diagnosed this clinical AVNRT as a slow-slow type using the typical SP as the antegrade limb and the superior SP as the retrograde limb.

We performed cryomapping (at a temperature of -30°C) during the AVNRT on the right posterior septum where the fractionated atrial potential was recorded during sinus rhythm (Figure 3), and it successfully terminated within 11 seconds.

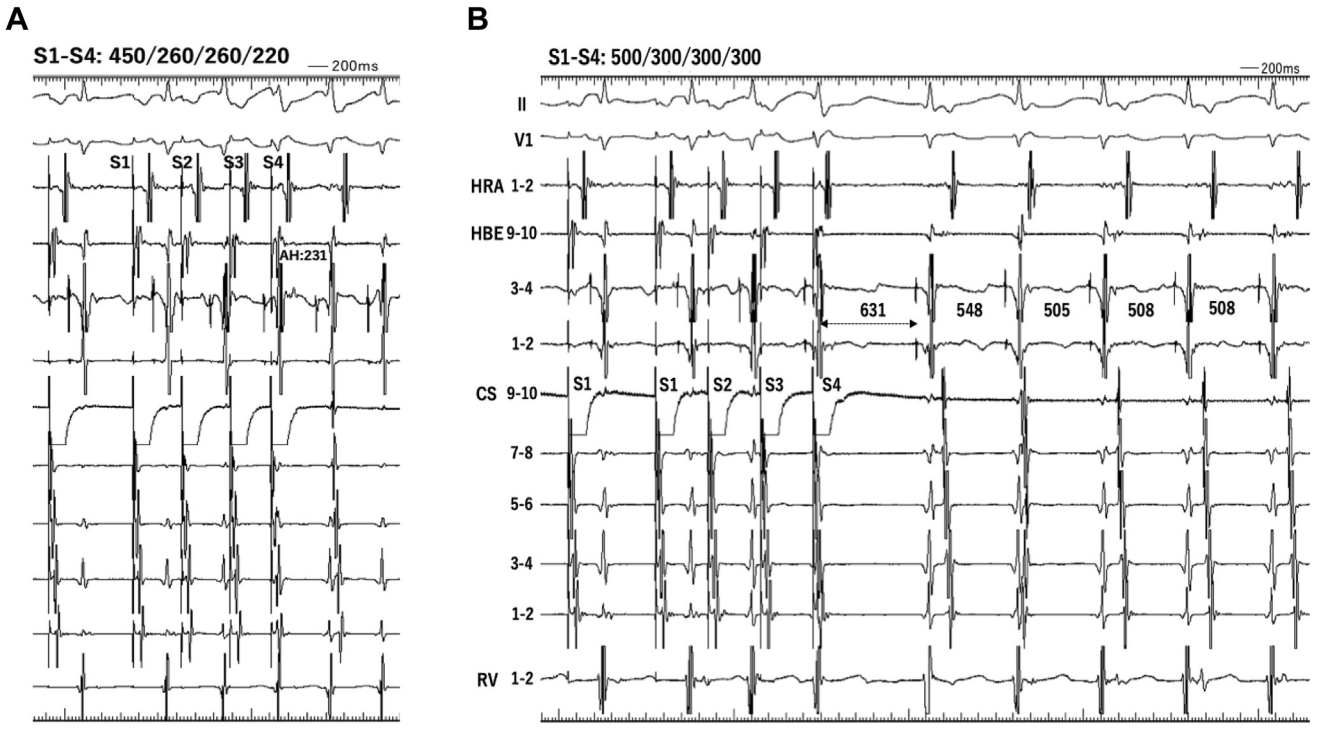

Figure 2 **A:** An atrial echo beat was induced without an AH jump by triple atrial extrastimulation. The sequence of the retrograde conduction was identical to that during the right ventricle (RV) apex stimulation and this retrograde conduction was followed by that through the antegrade fast pathway. **B:** Intracardiac recording by a multipolar electrode during a short RP tachycardia (the clinical tachycardia). This tachycardia was only induced with triple atrial extrastimulation after the jump in the AH interval under the administration of high-dose isoproterenol. The atrial activation during the tachycardia was earlier in the His bundle region than in the coronary sinus, and the atrial activation sequence was identical during the tachycardia and ventricular stimulation from the RV apex (Figure 1C), which was suggestive of antegrade conduction via the typical slow pathway and retrograde conduction via the superior slow pathway. AH = the interval from the atrium to the His bundle potential; CS = coronary sinus potential; HBE = His bundle electrogram; HRA = high right atrium; S = stimulation.

Then we performed cryoablation (at a temperature of −80°C) with 2 applications of 240 seconds each using the freeze-thaw-freeze technique, and an additional "bonus" cryoapplication was delivered at the successful ablation site. Finally, the endpoint of this procedure was AH jump without atrial echo beats.

After the initial ablation, the tachycardia became noninducible with programmed stimulation, regardless of the administration of isoproterenol. Moreover, VA conduction was not observed during normal ventricular stimulation and was only observed with AV simultaneous pacing during the basic stimulation preceding the ventricular extrastimulation owing to the "peeling back" phenomenon. No recurrence of the tachycardia was observed over 1 year of follow-up.

Discussion

Regarding the mechanism of the clinical paroxysmal SVT, we finally concluded that just 1 retrograde conduction in the present case was via the superior SP, and the clinical AVNRT was slow-superior slow AVNRT.

The reasons for a diagnosis of a retrograde superior SP were as follows. This patient exhibited just 1 retrograde nodal conduction pathway under all pacing maneuvers before and after the ablation. Para-Hisian pacing revealed this retrograde pathway was a nodal conduction. The retrograde conduction had a decremental property and the earliest site of the atrial activation was in the HB region, which was consistent with a nodal fast pathway. However, the retrograde conductivity right before or after the antegrade FP conduction indicated that this retrograde conduction conflicted with a nodal FP. By a process of elimination, this retrograde conduction must be a nodal superior-slow pathway. Those findings demonstrated that the substrate of the retrograde conduction was compatible with a superior SP, and we diagnosed this clinical AVNRT as slow-superior slow AVNRT.

In the present case, 2 ablation strategies targeting the antegrade SP and retrograde SP were planned to treat the clinical AVNRT. We selected a strategy targeting the antegrade SP as a first-line therapy because the ablation targeting the retrograde superior SP located near the HB region could have a possible adverse effect on the nodal function. If fast-superior slow AVNRT would arise after the ablation of the antegrade SP, we secondarily planned to target the atrial end of the retrograde superior SP.

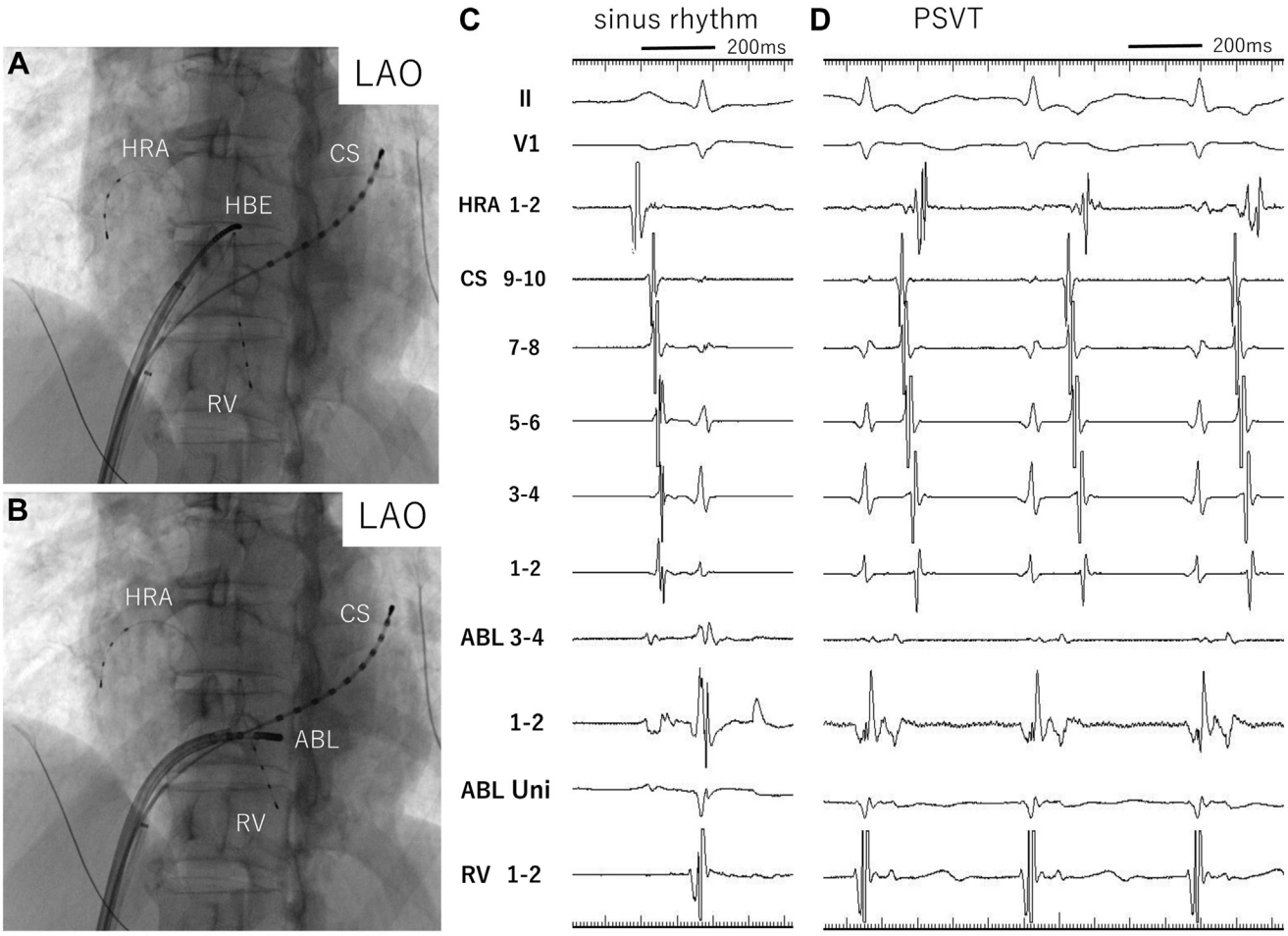

Figure 3 **A:** Left anterior oblique (LAO) view (45°) of the multipolar electrode catheter position during the electrophysiology study. **B:** LAO view (45°) of the multipolar electrode catheter and the ablation catheter position. Before the cryoablation, the multipolar electrode catheter in the His bundle region was exchanged for the ablation catheter. **C:** Intracardiac recording from a multipolar electrode catheter during sinus rhythm before the cryoablation. A slow potential was recorded from the distal tip of the ablation catheter at the site of the typical slow pathway region. The electrogram's atrial-to-ventricular ratio at that point was 1:3. **D:** Intracardiac recording from a multipolar electrode during the clinical paroxysmal supraventricular tachycardia (PSVT). During this tachycardia, the ventricular potential preceded the atrial potential. ABL = ablation catheter; CS = coronary sinus potential; HBE = His-bundle potential; HRA = high right atrium; RV = right ventricle; S = stimulation; Uni = unipolar potential.

Regarding the reasons why we selected a cryo-energy source, there was the possibility of the aforementioned second-line strategy targeting the retrograde superior SP located near the HB region. Cryoablation is safe for avoiding collateral damage to the nodal conduction, because the lesion area is relatively small as compared to that of RF ablation.[6] As another advantage of cryoablation, the termination of the clinical AVNRT owing to cryomapping could safely confirm the critical site of the tachycardia. Moreover, incessant junctional rhythm was frequently induced by the administration of isoproterenol, which made the heart rhythm confusing and made it hard to determine the optimal target site for RF ablation.

Actually, cryoablation following use of the cryomapping technique targeting the antegrade SP terminated the clinical AVNRT immediately, and no further SVT could be induced after the cryoablation. Interestingly, after the ablation targeting the anatomical antegrade SP, VA conduction was not observed during ventricular stimulation and was only observed during AV simultaneous pacing during the basic stimulation preceding the ventricular extrastimulation. This VA conduction was considered over the superior SP because the VA conduction was followed by antegrade fast pathway conduction and the atrial activation sequence did not change after ablation (Supplemental Figure 1). This phenomenon can be explained by the following speculation. In the present case, the inferior end of the superior SP was located close to the antegrade SP, and a cryoablation lesion targeting the typical SP region involved the nearby inferior end of the superior SP. The present case demonstrated the efficacy of cryoablation targeting the typical SP region for eliminating the superior SP.

The patient remained asymptomatic, and no atrial arrhythmias have been documented during a 2-year follow-up period.

Conclusion

To the best of our knowledge, this is the first case report of the successful cryoablation of an atypical slow-slow AVNRT with a superior SP using a superior SP located superior to the HB region.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.06.007.

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