Diastolic potentials manifest the extension of a slow pathway to the inferolateral right atrium during fast-slow atrioventricular nodal reentrant tachycardia

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Introduction

Previous studies have suggested the presence of slow pathway (SP) variants connected to the right atrial (RA) inferolateral free wall along the tricuspid annulus.[1] Because this variant is rare, details such as its electrophysiological characteristics or anatomical course have not been well studied. Further, there are no previous reports showing local electrograms of such SPs. We report a case of fast-slow atrioventricular nodal reentrant tachycardia (AVNRT) using a variant of an SP extending to the inferior RA. In this case, diastolic atrial potentials that appeared to be electrograms of the rightward extension of the SP were recorded in the inferior RA during the tachycardia.

Case report

A 14-year-old male patient without structural heart disease was referred to our institution for catheter ablation of symptomatic paroxysmal narrow QRS complex tachycardia. During the tachycardia, the 12-lead electrocardiogram showed a long RP tachycardia with negative P waves in leads II, III, and aVF. During burst stimulation and single extrastimulation from the right ventricle (RV), retrograde conduction with a long ventriculoatrial (VA) interval and a decremental conduction property occurred, and the earliest atrial activation site was in the ostium of the coronary sinus (CS). Atrial and ventricular single extrastimulation induced supraventricular tachycardia 1 (SVT1) with the same atrial activation sequence as that during burst stimulation from the RV (Figure 1A). The AH and HA intervals fluctuated between 130 and 150 ms and between 278 and 306 ms, respectively. Because SVT1 reproducibly terminated spontaneously, entrainment pacing could not be performed. Therefore, we induced SVT1 repeatedly to create an activation map in the RA using the Advisor HD Grid mapping catheter (Abbott, St. Paul, MN) and the EnSite Velocity 3-dimensional mapping system (Abbott). The activation map revealed the earliest atrial activation site at 6 o'clock in the CS (Figure 1B), where radiofrequency (RF) applications using a 3.5-mm irrigated-tip TactiCath ablation catheter (Abbott) eliminated the inducibility of SVT1.

After the RF applications, no VA conduction was observed during burst stimulation from the RV (Supplemental Figure 1A). However, atrial single extrastimulation induced SVT with atrial potentials at the His occurring earlier than those at the ostium of the CS, indicating that the SVT1 had converted to another SVT (SVT2). During SVT2, the AH and HA intervals fluctuated between 110 and 122 ms and between 306 and 336 ms, respectively. A

KEY TEACHING POINTS

- A slow pathway (SP) variant extending to the inferior right atrium (RA) is rare, and its electrophysiological characteristics and anatomical course are not well studied.
- Diastolic atrial potentials were recorded in the inferior RA during fast-slow atrioventricular nodal reentrant tachycardia (AVNRT) using a variant of the SP extending to the inferior RA. The diastolic potentials were considered as electrograms indicating rightward extension of the SP.
- Entrainment pacing was helpful to differentiate AVNRT from atrial tachycardia.

KEYWORDS Ablation; Atrioventricular nodal reentry tachycardia; Diastolic potential; Rightward extension; Slow pathway
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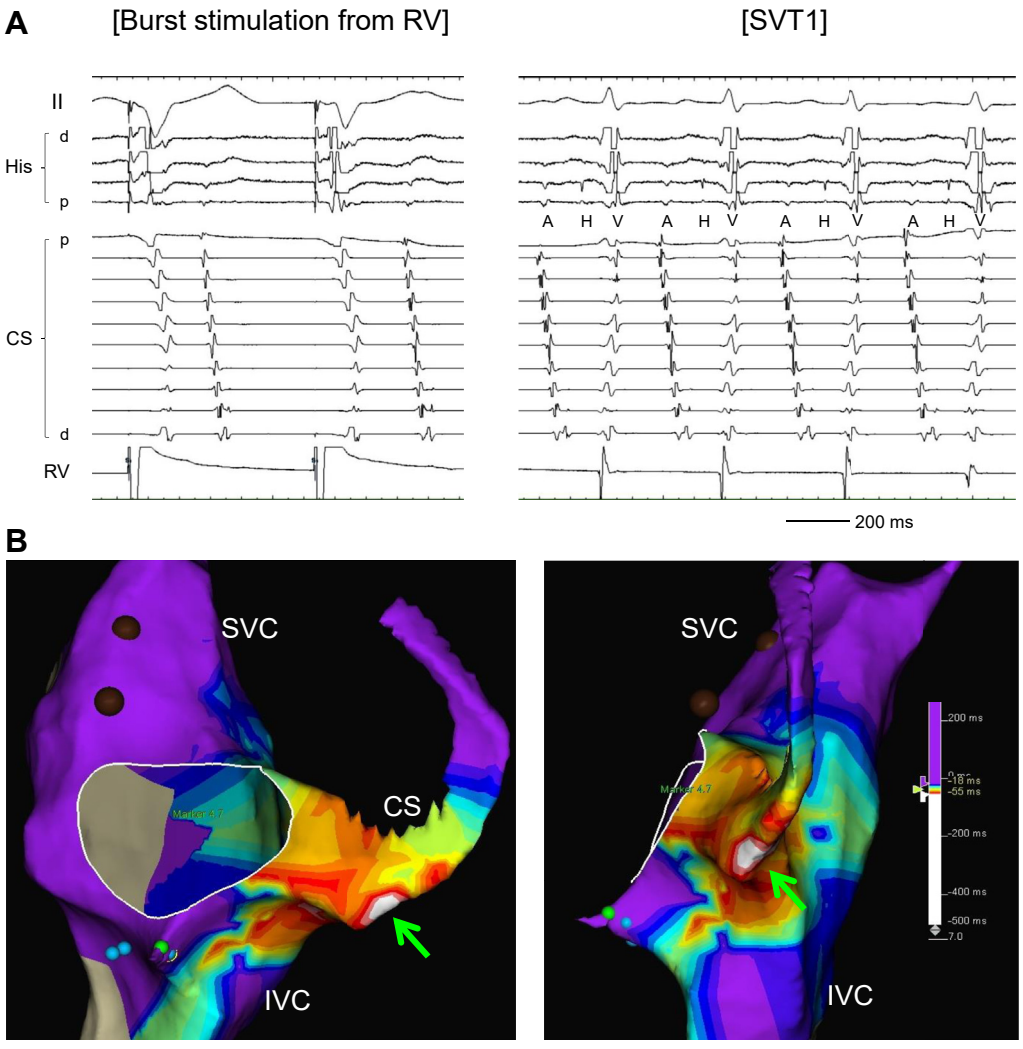

Figure 1 **A:** Intracardiac recordings during burst stimulation from the right ventricle (RV) and during supraventricular tachycardia 1 (SVT1). **B:** Activation map in the right atrium during SVT1. The green arrow indicates the earliest atrial activation site. A = atrium electrogram; CS = coronary sinus; d = distal; H = His bundle potential; IVC = inferior vena cava; p = proximal; RV = right ventricle; SVC = superior vena cava; V = ventricle electrogram.

2-mg bolus injection of adenosine triphosphate reproducibly terminated SVT2 after a last ventricular event following the incremental prolongation of the atrial cycle length. During the ongoing tachycardia, a transient atrioventricular (AV) block developed owing to catheter mechanical stimulation (Supplemental Figure 1B). Based on these findings, SVT2 was initially thought to be an adenosine-sensitive atrial tachycardia (AT). Therefore, the RA was again mapped during SVT2 using the Advisor HD Grid mapping catheter. Diastolic atrial potentials were recorded in the cavotricuspid isthmus, around the ostium of the CS, and in the inferior vena cava (Figure 2A). The earliest activation site was at 6 o'clock in the RA adjacent to the tricuspid annulus. The activation pattern in the RA was neither centrifugal nor reentrant: after centrifugal propagation in the cavotricuspid isthmus region, the atrial electrograms propagated in a caudocranial direction in the free wall and in a lateral-to-septal direction in the posterior wall (Figure 2B, and Supplemental Movies 1, 2, and 3). During entrainment pacing from the RA appendage (RAA), the diastolic potentials were orthodromically captured, whereas the atrial electrograms other than the diastolic potentials were antidromically captured (Figure 2C). The difference between the VA interval after entrainment pacing from the RAA and that from the proximal CS was 6 ms, which was shorter than 14 ms, indicating positive VA linking (Supplemental Figure 2).[2] Entrainment pacing from the RV was not performed. We considered SVT2 to be a fast-slow AVNRT and the earliest site of the diastolic potentials to be the breakthrough site of a retrograde SP. RF application at a power setting of 35 W targeting the earliest diastolic potentials terminated SVT2 immediately after the start of RF delivery (Figure 3A and 3B). The RF delivery was continued until the lesion size index reached 5.5 and additional RF applications at the same setting around the earliest site completely eliminated the inducibility of SVT2. No further SVTs were induced by burst stimulation or extrastimulation from the RA. There were discontinuities in the AH conduction curve, suggesting the existence of multiple antegrade

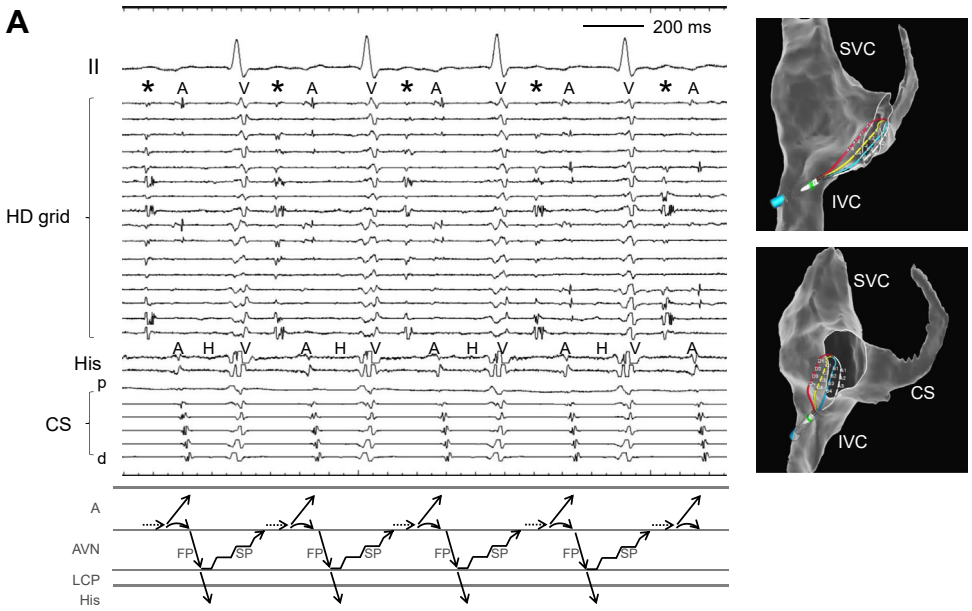

Figure 2 **A:** Intracardiac recordings and laddergram during supraventricular tachycardia 2 (SVT2). Atrial diastolic potentials (*asterisks*) with fragmentation were recorded with the Advisor HD Grid mapping catheter (Abbott). The dotted lines in the laddergram represent unidentified conduction between the diastolic potentials and the other atrial electrograms. **B:** Activation map during SVT2. The scaled-down figures with red borders are activation maps created by extracting only the diastolic potentials. The diastolic potentials were recorded in the inferior right atrium (RA), including the cavotricuspid isthmus (CTI) and around the ostium of the coronary sinus, and in the inferior vena cava. The earliest activation site (*green arrow*) was at 6 o'clock in the RA adjacent to the tricuspid annulus. After centrifugal propagation in the CTI region, the atrial electrograms propagated in a caudocranial direction in the free wall and lateral-to-septal direction in the posterior wall. **C:** Tracing and laddergram during entrainment by rapid pacing delivered from the RA appendage. The first interval of the diastolic potential (*asterisk*) after the last pacing was 400 ms, identical to the pacing interval, indicating orthodromic capture. In contrast, the first intervals of the atrial electrograms other than the diastolic potential after the last pacing were 424 ms, identical to the tachycardia cycle length, indicating antidromic capture. FP = fast pathway; LCP = lower common pathway; MAP = mapping catheter; RAA = right atrial appendage; SP = slow pathway. Other abbreviations are as in Figure 1.

AV conduction pathways (Supplemental Figure 3). No recurrence of the tachycardia has been observed during a 4-year follow-up period.

Discussion

SVT2 was characterized by diastolic atrial potentials during the tachycardia in the inferior RA. Other findings of SVT2, including VA block during burst stimulation from the RV, persistence of the tachycardia during transient AV block owing to catheter mechanical stimulation, and termination by a low dose of adenosine triphosphate, excluded AV reentrant tachycardia and rendered a differential diagnosis between AVNRT with a lower common pathway (LCP) and adenosine-sensitive AT. Entrainment pacing from the ventricle is useful to discriminate between AVNRT and AT. However, we did not perform it owing to the absence of VA conduction. In such cases, evaluation of VA linking is a helpful and accurate method for discrimination, although some pitfalls have been reported.[2,3] In our case, positive VA linking was reproducibly observed, indicating diagnostic significance of the AVNRT. Although adenosine-sensitive AT could not be completely excluded despite some controversy, we considered the most likely diagnosis of SVT2 to be fast-slow AVNRT with retrograde LCP block by placing importance on the positive VA linking and for the following reasons. First, the mechanism for orthodromic capture of the diastolic potentials could be reasonably explained by considering SVT2 as AVNRT and the diastolic potentials as electrograms indicating the rightward extension of the SP (Figure 2C). Second, the activation map during SVT2 could be interpreted as focal AT with conduction block close to the focus or as reentrant AT. In these cases, the fragmented diastolic atrial potentials would have implied the existence of conduction block or slow conduction in the RA. However, no abnormal potentials suggesting such abnormal conductions were identified during the mapping for SVT1. It is difficult to consider that such abnormal conductions would exist only during SVT2 but not during SVT1. Finally, the development of AV block during SVT2 could be explained by the antegrade block at the level of the LCP owing to catheter mechanical stimulation.

Because SVT1 did not persist long enough, its detailed evaluation, including entrainment pacing, could not be performed. The atrial activation sequence during SVT1 and that during burst stimulation from the RV were almost identical. In addition, the RF applications at the earliest atrial activation site during SVT1 rendered SVT1 noninducible and eliminated VA conduction during burst stimulation from the RV, indicating that SVT1 was fast-slow AVNRT using the SP (SP1) extending to the inferior left atrium as the retrograde limb of the reentry circuit (Figure 3C).

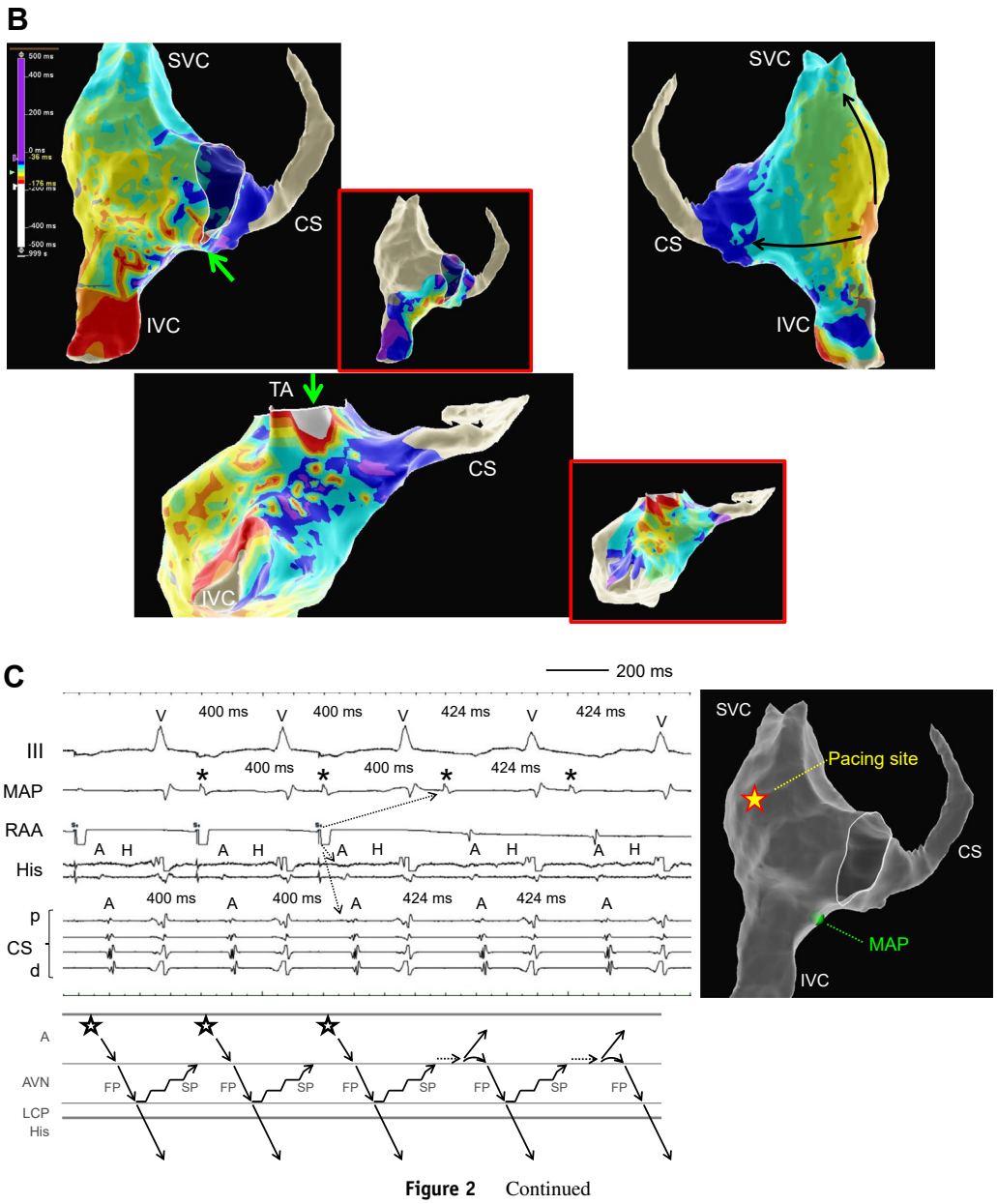

Figure 2 Continued

There was no VA conduction during burst stimulation from the RV after the RF applications for SVT1, suggesting retrograde LCP block. However, retrograde VA conduction via the SP1 before the RF applications implied that the SP1 did not connect to the LCP (Figure 3C). This speculation is consistent with the longitudinal dissociation of pathways activating the penetrating bundle.[4,5] We considered that in addition to the retrograde SP, the anterograde fast pathway used during SVT2 also connected to the LCP, whereas the other fast pathway, which did not connect to the LCP, was used during SVT1. This hypothesis is supported by the presence of multiple antegrade conduction pathways, as indicated by the AH conduction curve (Supplemental Figure 3) and the different AH intervals between them during SVT1 and SVT2.

The SP used during the SVT2 (SP2) in this case was considered to be a rare variant of the SP extending to the inferolateral RA, as Kaneko and colleagues[1] reported. Several studies also have reported electrophysiological evidence for a ring of nodal-type cells around the tricuspid annulus and its anatomical continuity with the inferior extension of the AV node.[6,7] We estimated that the diastolic potentials in our case were electrograms indicative of the rightward extension of the SP2 for the following reasons: (1) The RF application at the earliest site of the diastolic potential terminated SVT2 with a last ventricular electrogram following prolongation of the interval between the ventricular electrogram and the diastolic potential, consistent with a retrograde conduction block over the SP2 (Figure 3B). (2) During entrainment pacing from the RAA, the diastolic potentials were captured

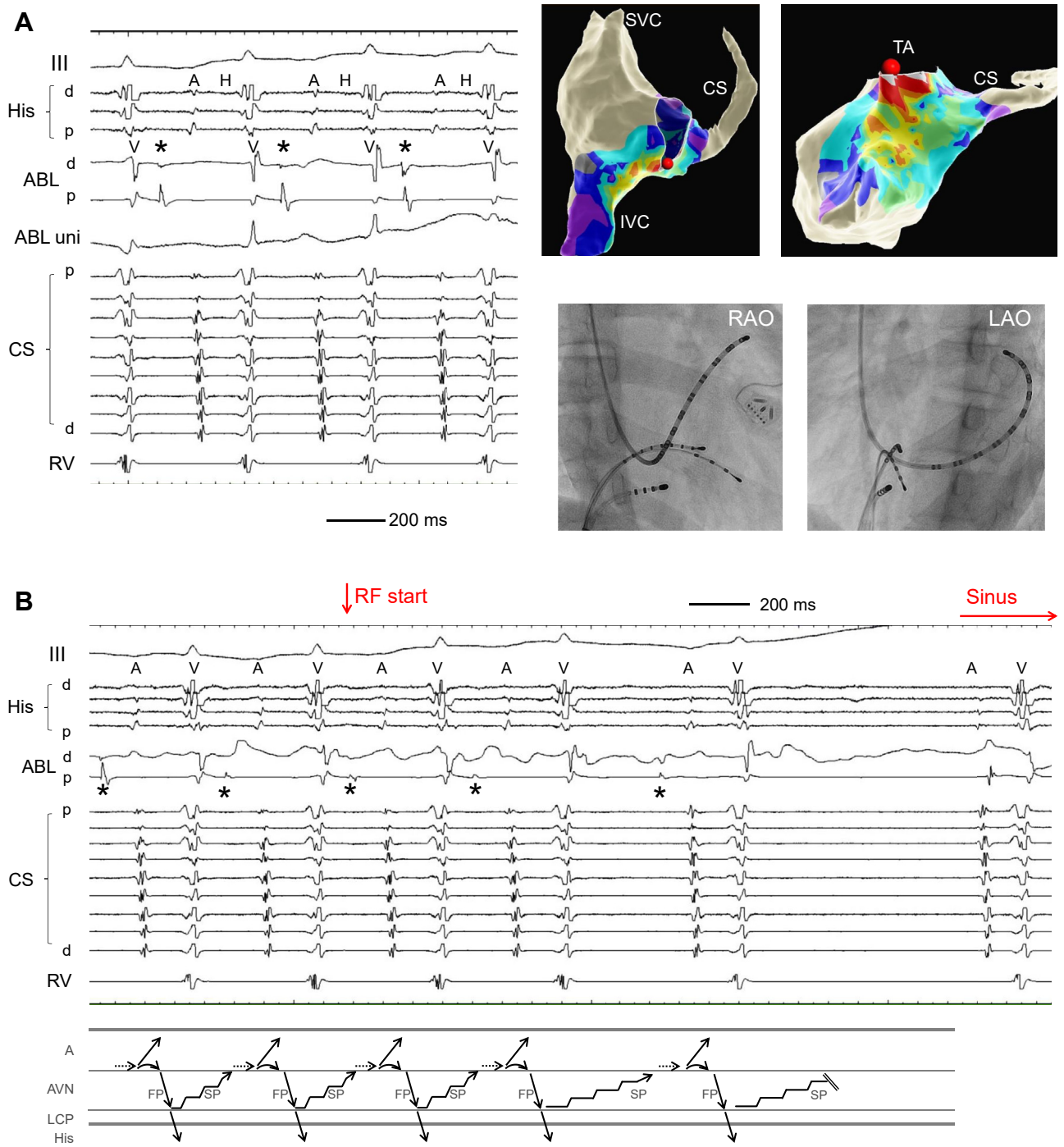

Figure 3 **A**: Intracardiac recordings during SVT2 immediately before radiofrequency (RF) delivery at the successful site and the positions of the catheters. Atrial diastolic potentials (*asterisks*) were recorded with the ablation catheter. **B:** Intracardiac recordings immediately after RF delivery. SVT2 terminated with a last ventricular electrogram following a prolongation of the interval between the ventricular electrogram and diastolic potential, consistent with a retrograde conduction block over the slow pathway. **C:** Schematic drawing of the estimated reentrant circuit of SVT1 and SVT2. ABL = ablation catheter. Other abbreviations are as in Figures 1 and 2.

orthodromically (Figure 2C). (3) The genesis of the extension of an SP is presumed to be specialized atrial tissue surrounding the tricuspid annulus with node-like characteristics.[1,6,7] The recording of fragment potentials was consistent with such characteristics of an SP. (4) During mapping for SVT1, no abnormal potentials were recorded in the area where diastolic potentials were recorded during SVT2.

Although the diastolic potentials showed a centrifugal pattern, atrial electrograms of other than the diastolic potentials showed a noncentrifugal pattern and discontinuity with the diastolic potentials on the activation map. These findings suggest that the extension of the SP2 heterogeneously connected the atrial myocardium during SVT2 accompanied by conduction delays.

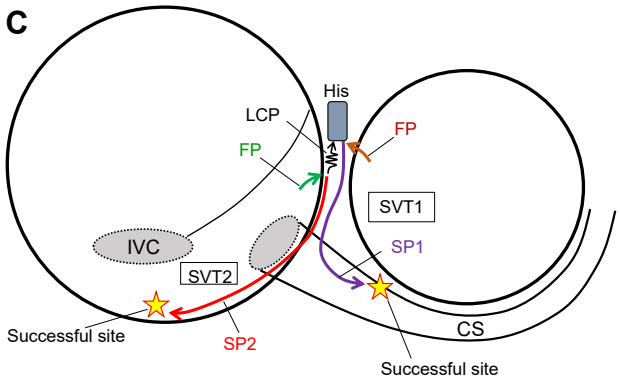

Figure 3 Continued

Conclusion

In this rare case of atypical fast-slow AVNRT with atrial diastolic potentials recorded in the inferior RA during tachycardia, the diastolic potentials were considered to be electrograms indicating the rightward extension of the SP.

Acknowledgments

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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.2022.11.007.

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