Idiopathic left ventricular tachycardia continuously entrained by atypical atrioventricular nodal reentrant tachycardia

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Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) and idiopathic left ventricular tachycardia (ILVT) are the 2 most common types of idiopathic tachycardias in patients with structurally normal hearts. Recent longitudinal follow-up data have revealed a high prevalence of AVNRT (19.4%) in patients receiving catheter ablation of ILVT.[1] However, there is limited literature on cases with coexisting ILVT and AVNRT.[2–4] Moreover, most reported cases were about patients who exhibited transition from one tachycardia to another. Cases sustaining simultaneous ILVT and AVNRT are limited,[4] and the mechanism explaining this phenomenon has not been described previously. In this case of coexisting ILVT and AVNRT, only the transition between ILVT and AVNRT was initially exhibited. However, after radiofrequency energy applications to the posterior fascicular and slow pathway regions, continuous resetting of ILVT by AVNRT (manifest entrainment) was observed during the latter half of the session. Ablating the ILVT exit may prevent the AVNRT wavefront from conducting antidromically into the slow conduction zone. Orthodromic conduction of the AVNRT wavefront into the entrance of the slow conduction zone is a speculated mechanism contributing to the simultaneous occurrence of ILVT and AVNRT.

KEY TEACHING POINTS

- Idiopathic left ventricular tachycardia (ILVT) and atrioventricular nodal reentrant tachycardia (AVNRT) are different reentrant arrhythmias arising below and above the bundle of His, respectively. They are linked to each other through the His–Purkinje system.
- There are 2 patterns of coexistence of ILVT and AVNRT. The sequential or simultaneous occurrence of ILVT and AVNRT should be differentiated.
- AVNRT can terminate ILVT without QRS fusion. The activation wavefronts of AVNRT and ILVT could easily collide within the Purkinje network.
- Continuous resetting of ILVT by AVNRT in the outer loop ventricular myocardium produces manifest entrainment. Propagation of the activation wavefront of AVNRT from the entrance side of the slow conduction zone may be required to sustain this phenomenon.

Case report

A 24-year-old man with recurrent episodes of palpitations was referred for catheter ablation of a wide QRS complex tachycardia. There was no evidence of structural heart disease on echocardiographic examination or laboratory testing. The 12-lead electrocardiogram exhibited normal QRS complex morphology during sinus rhythm. However, right bundle branch block with northwest axis deviation was present during the tachycardia with a QRS duration of 135 ms and tachycardia cycle length (TCL) of 380 ms (Supplemental Figure 1). The tachycardia was not affected by intravenous adenosine administration but was responsive to intravenous verapamil. After informed consent was obtained, an electrophysiological study was conducted using multipolar electrode catheters placed in the coronary sinus, His bundle, and right ventricle (Figure 1A). At baseline, atrial

KEYWORDS Atrioventricular nodal reentrant tachycardia; Catheter ablation; Manifest entrainment; Idiopathic left ventricular tachycardia; Slow conduction zone

(Heart Rhythm Case Reports 2022;8:572–576)

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Disclosures: The authors have no conflict of interests to declare with respect to this study. **Address reprint requests and correspondence:** Dr Masato Okada, Cardiovascular Center, Sakurabashi Watanabe Hospital, 2-4-32 Umeda, Kita-ku, Osaka, 530-0001, Japan. E-mail address: masato.okada1105@gmail.com.

https://doi.org/10.1016/j.hrcr.2022.05.014

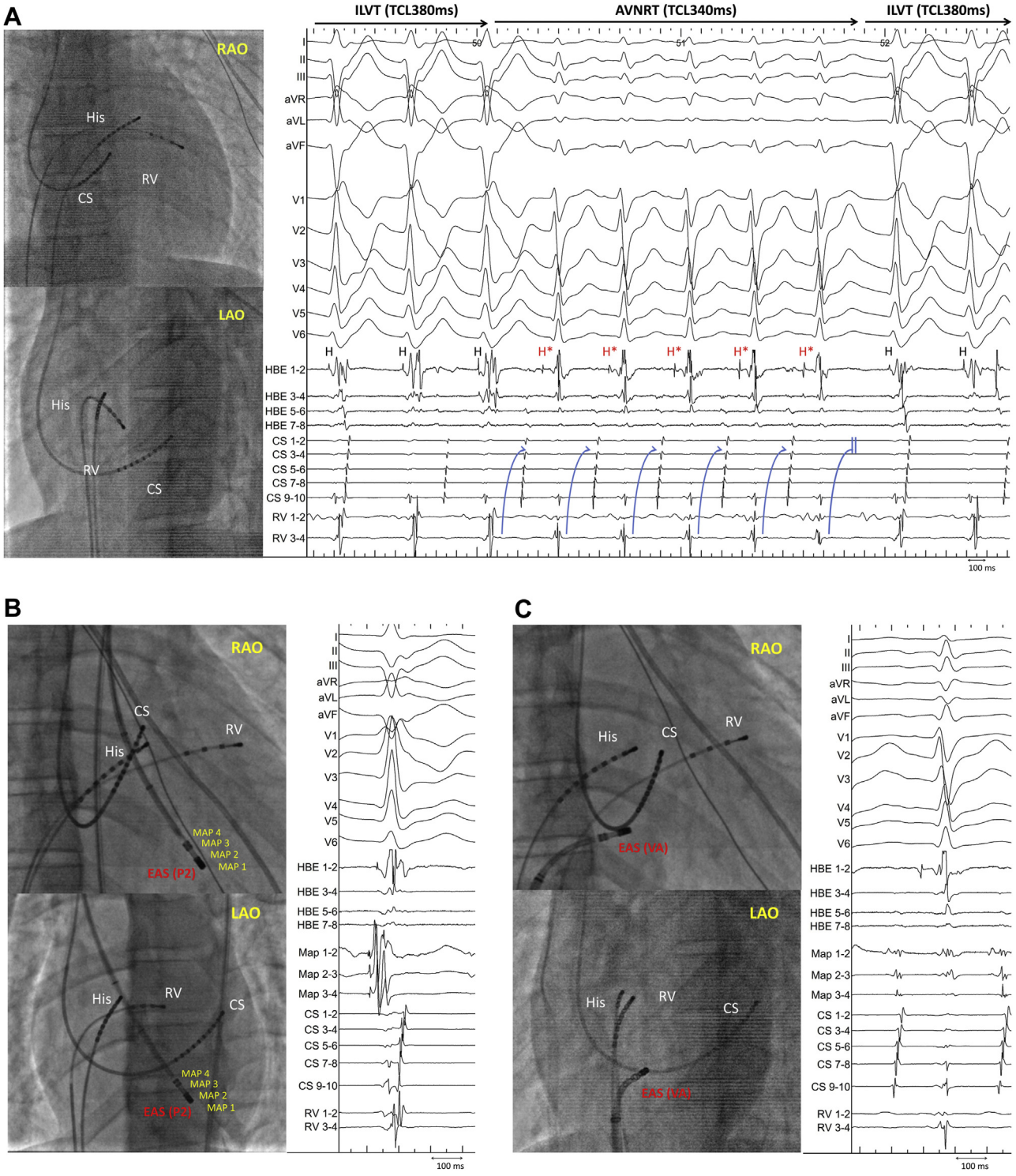

Figure 1 **A:** Placement of electrode catheters during the electrophysiological study and intracardiac electrograms. A 5F decapolar, 8-pole, and quadripolar electrode catheter were positioned in the coronary sinus (CS), His bundle (His), and right ventricle (RV) outflow regions, respectively. Atrial and ventricular extrastimuli induced clinical wide QRS complex tachycardia, which abruptly shifted to narrow QRS complex tachycardia with advancement of His potential (H*) and preceded by ventriculoatrial (VA) linking. Disconnection of VA linking made the narrow QRS complex tachycardia to the original wide QRS complex tachycardia. After the diagnosis of 2 tachycardias as idiopathic left ventricular tachycardia (ILVT) and fast–slow atrioventricular nodal reentrant tachycardia (AVNRT), catheter ablation targeting each arrhythmia was performed using an irrigated catheter. **B,C:** Fluoroscopic images and intracardiac electrograms during radiofrequency energy applications against ILVT (**B**) and AVNRT (**C**) are described. I, II, III, aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5, and V_6 represent the surface electrocardiogram leads; HBE 1–2 to 7–8 represent the distal-to-proximal His recordings; CS 1–2 to 9–10 represent the distal-to-proximal CS recordings; RV 1–2 to 3–4 represent the distal-to-proximal RV recordings; and MAP 1–2 to 3–4 represent the distal-to-proximal electrograms of the ablation catheter. EAS = earliest activation site; H = His; LAO = left anterior oblique; RAO = right anterior oblique; TCL = tachycardia cycle length.

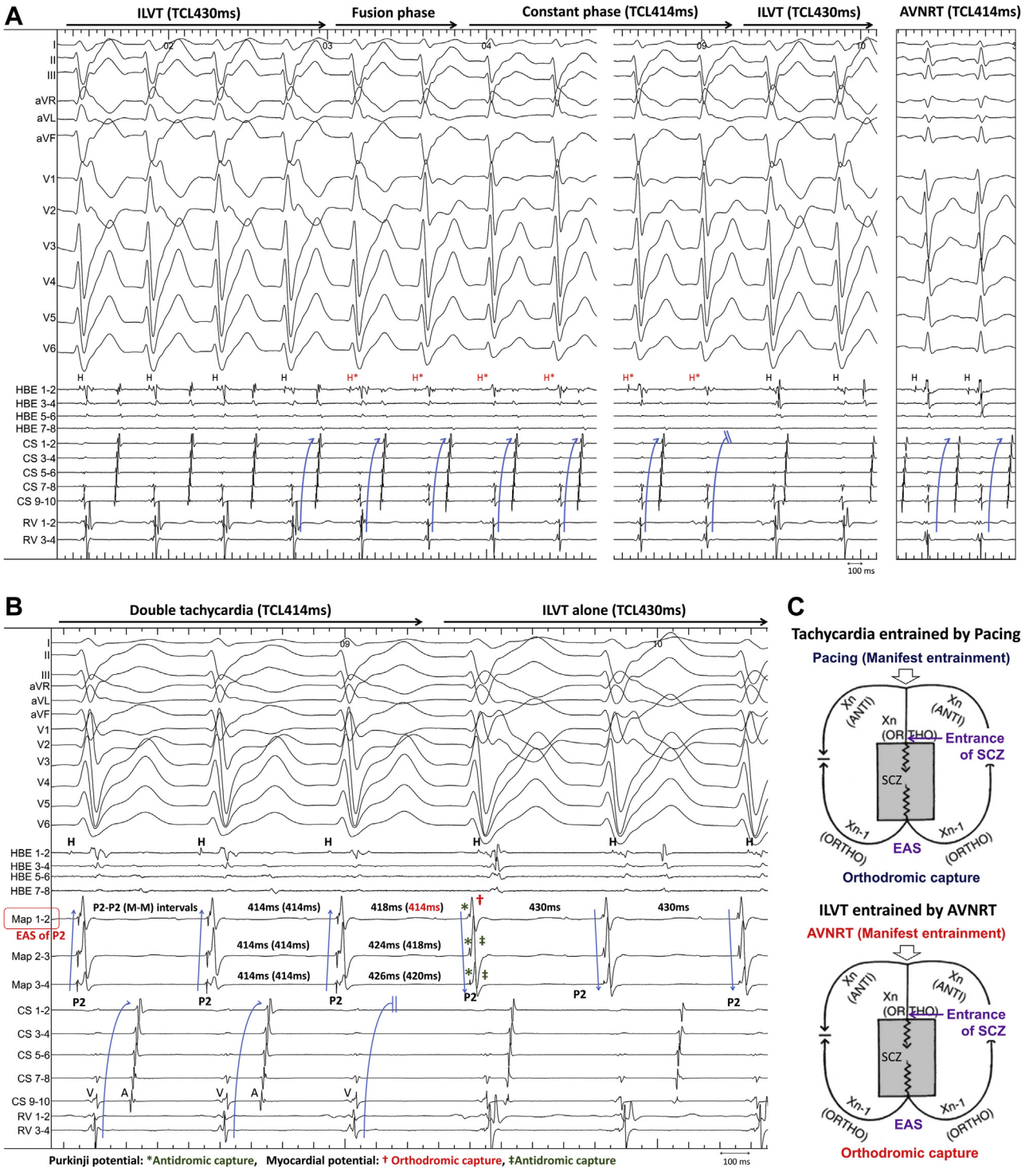

Figure 2 **A:** Intracardiac electrograms at the onset and termination of double tachycardia are described. With advancement of His potential (H*) and preceded by VA linking, the QRS morphology gradually changed through fusion phase. During double tachycardia, atrial activation sequences are identical to those of AVNRT. When VA linking was disconnected, the QRS morphology abruptly returned to that during ILVT. **B:** Detailed intracardiac electrograms at the termination of double tachycardia are described. In the electrograms of the ablation catheter (MAP1-2) placed on the earliest site of P2 potential during ILVT alone, presystolic P2 potentials and subsequent myocardial potentials were successfully recorded. The M-M interval after AVNRT termination is 414 ms, which is the same as the TCL of AVNRT, suggesting that M potentials in the first return beat are captured orthodromically with AVNRT (indicated by the *red dagger*). The other electrograms, including P2 potential, are antidromically captured (indicated by the *green asterisks* and *double dagger*). **C:** Schematic representation of the manifest entrainment during tachycardia. Similar to the pacing study during a macroreentrant tachycardia (upper panel), the manifest entrainment and the orthodromic capture of EAS indicated that the AVNRT activation wavefront propagated from the entrance side of the slow conduction zone (SCZ) during the double tachycardia (lower panel). M = myocardial; other abbreviations as in Figure 1.

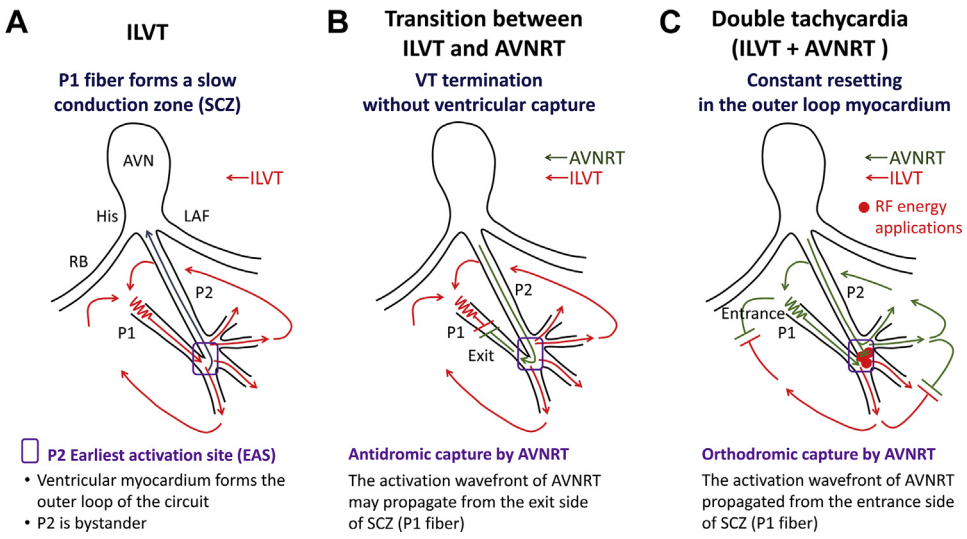

Figure 3 Hypotheses for the mechanism of the sequential and simultaneous occurrence of ILVT and AVNRT. **A:** During ILVT, P1 fiber forms a slow conduction and ventricular myocardium of the left ventricular septum forms the retrograde limb of the circuit. **B:** In AVNRT occurrence with faster TCL, the activation wavefront propagates through the left posterior fascicle and Purkinje fiber. In the present case, abrupt transition from ILVT to AVNRT without QRS fusion was observed initially, which indicated that the activation wavefronts collide within the Purkinje network outside the ventricular myocardium. Propagation of the AVNRT activation wavefront from the exit side of the SCZ (P1 fiber) is a plausible hypothesis. The orthodromic wavefront may hit the refractory tail of the proceeding wavefront, which may prevent ILVT from lasting. **C:** Conversely, during the simultaneous occurrence of ILVT and AVNRT, continuous resetting in the outer loop myocardium (ie, manifest entrainment) exhibited QRS fusion. The AVNRT activation wavefront was propagated from the entrance side of the SCZ, which may be required to sustain this double tachycardia. AVN = atrioventricular node; LAF = left anterior fascicle; RB = right bundle; RF = radiofrequency; other abbreviations as in Figures 1 and 2.

and ventricular extrastimulus pacing exhibited decremental conduction. Dual atrioventricular nodal physiology was suggested by an abrupt increase in the atrial–His (A-H) interval of ≥50 ms following a 10-ms decrease in the extrastimulus coupling interval during programmed atrial stimulation. After isoproterenol infusion, ventricular double extrastimuli successfully induced the clinical tachycardia. Continuation of the tachycardia even during atrioventricular dissociation confirmed the tachycardia as ventricular tachycardia (VT). The His–ventricular (H-V) interval during VT was shorter than that during sinus rhythm (−8 vs 46 ms) (Supplemental Figure 2), which suggested the VT as ILVT.

After a while, the wide QRS complex morphology abruptly changed to a narrow one with advancement of His potential and preceded by ventriculoatrial (VA) linking (Figure 1A). During the narrow QRS complex tachycardia, the A-H and His–atrial (H-A) intervals were 130 and 210 ms, respectively. The atrial activation sequence during the tachycardia was identical to that during ventricular pacing, and the His and coronary sinus ostium were simultaneously the earliest activated sites. Response to ventricular pacing during the tachycardia (failure to reset the tachycardia by His-refractory single extrastimulus and termination of the tachycardia after the transition zone of QRS fusion by overdrive pacing) was consistent with AVNRT. This narrow QRS complex tachycardia was repetitively induced by ventricular double extrastimuli and/or ILVT with a V-A-V sequence. Based on these findings, the narrow QRS complex tachycardia was diagnosed as a fast–slow form of AVNRT.

We first attempted to ablate ILVT through a transaortic approach using the ThermoCool SmartTouch system (Biosense Webster, Diamond Bar, CA). As no diastolic potential (P1) was recorded in the LV posterior septum region, we tried to identify the earliest site of P2 potential during ILVT (Figure 1B). However, the frequent transition from ILVT to AVNRT and vice versa made it difficult to identify the earliest activation site (EAS) of P2 potential. Similarly, it was also difficult to correctly identify the earliest atrial activation site during AVNRT (Figure 1C).

Repetitive radiofrequency energy applications to the earliest P2 (with 30–35 W power and 20–40 seconds duration) did not terminate ILVT. Those targeting the slow pathway potentials with titration methods from 15 to 30 W did not terminate AVNRT either. However, the TCLs of ILVT and AVNRT gradually increased from 380 to 430 ms and from 340 to 414 ms, respectively. After a while, tachycardia with a different QRS complex morphology was observed (Figure 2A). Transition of the ILVT exit was one hypothesis; however, the findings of (1) transition through fusion phase, (2) transition preceded by the occurrence of VA linking and advancement of His potential, and (3) identical atrial activation sequence during AVNRT and the new tachycardia indicated that the new tachycardia was sustaining ILVT entrained by AVNRT (Figure 2A). Furthermore, there was abrupt transition to the previous QRS complex morphology when VA linking was disconnected, which

supported this explanation (Figure 2A). Interestingly, the MAP1-2 electrograms located on the EAS of P2 exhibited orthodromic capture just after AVNRT termination (Figure 2B). The findings indicated that the AVNRT activation wavefront was propagated from the entrance side of the slow conduction zone during the double tachycardia (Figure 2C).

The first session was completed after confirming the noninducibility of both ILVT and AVNRT by atrial and ventricular double extrastimulus pacing. However, the second session was performed 2 years after the first session because of ILVT recurrence. Unlike the first session, there was no transition from one tachycardia to another, which enabled mapping the EAS of P2. Radiofrequency energy applications to the more basal septum region successfully eliminated ILVT. During a 3-year follow-up period, no arrhythmia recurrence was identified, without any antiarrhythmic medication.

Discussion

We encountered a case of ILVT manifestly entrained by atypical AVNRT. Continuous resetting of ILVT by AVNRT in the outer loop myocardium is essential to explain this phenomenon. The slowly conducting Purkinje fiber (P1) forms an anterograde limb of ILVT, and the AVNRT wavefront propagates to the left ventricle through the His–Purkinje system. Because both tachycardias are linked to each other through the His–Purkinje system, specific conditions may be required for this phenomenon. Similar TCLs of ILVT (430 ms) and AVNRT (414 ms) after radiofrequency energy applications would prevent the AVNRT wavefront from hitting the refractory tail of the preceding ILVT wavefront, which may contribute to sustaining the double tachycardia. However, the alteration of the AVNRT wavefront from conducting antidromically to orthodromically into the slow conduction zone would be more important to develop the double tachycardia.

This hypothesis was supported by the intracardiac electrograms showing that the double tachycardia abruptly stopped and changed to single tachycardia (ILVT alone) (Figure 2B). In macroreentrant tachycardia, when pacing exhibits the manifest entrainment and the orthodromic capture of EAS, the pacing wavefront propagates from the entrance of the slow conduction zone, which is located between the pacing site and EAS (Figure 2C; upper panel).[5] In this study, ILVT was manifestly entrained by AVNRT, and the local myocardial potential at the EAS of P2 (ie, EAS of ILVT) was orthodromically captured by AVNRT. This suggested that the AVNRT activation wavefront was propagated from the entrance side of the slow conduction zone during the double tachycardia (Figure 2C; lower panel). Tissue injury of the P1–myocardial junction by radiofrequency energy applications might prevent the AVNRT wavefront from conducting antidromically to the exit of the slow conduction zone.

Spatial distribution of slow conduction zone (P1 fiber) may be important to sustain ILVT and AVNRT. Because of the complex Purkinje network, the ILVT circuit has multiple entrances and exits.[6] When the entrance of the slow conduction zone is located near the posterior fascicles (eg, reverse-type ILVT),[7] the AVNRT activation wavefront may easily propagate from the entrance. Manifest entrainment of ILVT by atrial pacing can be explained by this spatial distribution of the entrances.

Finally, the mechanism of transition between ILVT and AVNRT, initially observed in this case, is also noteworthy. Considering (1) the anatomical proximity between the end of the His–Purkinje system and the exit of the slow conduction zone (P1–myocardial junction) and (2) the propagation of AVNRT wavefront from the entrance side of the slow conduction zone during the double tachycardia, the propagation of AVNRT wavefront from the exit side of the slow conduction zone may contribute to this transition. When activation wavefronts collide with antegrade wavefront within the slow conduction zone or hit the refractory tail of the preceding wavefront within the Purkinje network, ILVT terminates without QRS fusion. Termination of ILVT within the Purkinje network, including P1 fiber, is a plausible mechanism of transition from ILVT to AVNRT (Figure 3).

Conclusion

The sequential and simultaneous occurrence of ILVT and AVNRT should be separately considered. Orthodromic or antidromic conduction of the AVNRT wavefront into the slow conduction zone is a speculated mechanism that differentiates the 2 phenomena.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2022.05.014.

References

1. Chung FP, Van Ba V, Lin YJ, et al. The prevalence and characteristics of coexisted atrioventricular nodal reentrant tachycardia and idiopathic left fascicular ventricular tachycardia. J Cardiovasc Electrophysiol 2018;29:1096–1103.
2. Wagshal AB, Mittleman RS, Schuger CD, Huang SK. Coincident idiopathic left ventricular tachycardia and atrioventricular nodal reentrant tachycardia: control by radiofrequency catheter ablation of the slow atrioventricular nodal pathway. Pacing Clin Electrophysiol 1994;17:386–396.
3. Weng KP, Chiou CW, Kung MH, Lin CC, Hsieh KS. Radiofrequency catheter ablation of coexistent idiopathic left ventricular tachycardia and atrioventricular nodal reentrant tachycardia. J Chin Med Assoc 2005;68:479–483.
4. Itoh T, Kimura M, Tomita H. Double tachycardia with His-left ventricular and interventricular dissociations. Europace 2021;23:1866.
5. Okumura K, Olshansky B, Henthorn RW, Epstein AE, Plumb VJ, Waldo AL. Demonstration of the presence of slow conduction during sustained ventricular tachycardia in man: use of transient entrainment of the tachycardia. Circulation 1987;75:369–378.
6. Taniguchi H, Kobayashi Y, Maruyama M, et al. Electrophysiological and anatomical background of the fusion configuration of diastolic and presystolic Purkinje potentials in patients with verapamil-sensitive idiopathic left ventricular tachycardia. J Arrhythm 2015;31:261–267.
7. Phanthawimol W, Nogami A, Haruna T, et al. Reverse-type left posterior fascicular ventricular tachycardia: a new electrocardiographic entity. JACC Clin Electrophysiol 2021;7:843–854.