Verapamil-sensitive ventricular tachycardia demonstrating multiform QRS morphology in a patient with ischemic cardiomyopathy

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Introduction

The His-Purkinje system (HPS) is known to be involved in the reentrant circuit of ventricular tachycardia (VT), even in patients with structural heart disease.[1–3] Catheter ablation may be an effective strategy for this entity; however, there is a potential risk of conduction delay or even high-degree atrioventricular (AV) block. Therefore, detailed understanding of the reentry circuit is crucial. However, the mechanism and role of HPS in this entity are debatable. We experienced a case of post–myocardial infarction VT with involvement of the HPS demonstrating a change in QRS morphology without termination, which provided us clues for understanding the mechanisms underlying this type of VT.

> ### KEY TEACHING POINTS
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> - His-Purkinje system can be involved in the reentrant circuit of ventricular tachycardia (VT) in patients with structural heart disease and may produce narrow QRS VTs with various QRS morphologies.
> - The QRS morphology may change with the shift of the exit site of VT to left ventricle fascicles.
> - Cautious interpretation of QRS morphologies and HV interval during sinus rhythm and VT are crucial for interpretation of the VT mechanism.

Case report

A 63-year-old man who had undergone coronary bypass surgery owing to ischemic cardiomyopathy 3 weeks prior presented with palpitation. The 12-lead electrocardiogram during the sinus rhythm showed wide QRS complex (125 ms), suggesting conduction delay in the left bundle. During tachycardia, the QRS complex was narrower than the sinus rhythm (QRS width of 114 ms), with periodic sinus captures with wider QRS complex, confirming the diagnosis of VT. Mainly, 3 types of VTs occurred with the tachycardia cycle length (TCL) of 505 ms, with configurations of left axis deviation with right bundle branch block, left axis deviation with left bundle branch block (LBBB), and normal axis with LBBB (Figure 1).

Intravenous administration of verapamil successfully and repetitively terminated all the VTs. However, as VTs were incessantly induced, the patient underwent electrophysiological study and catheter ablation.

The electrophysiological study was performed with quadripolar catheters positioned in the high right atrium, Bundle of His, and right ventricular apex and the omnipolar catheter positioned in the coronary sinus. Left ventricular mapping was also performed using the PentaRay (Biosense Webster, Diamond Bar, CA) catheter using the transseptal approach. During the sinus rhythm, electrocardiography showed prolongation of the HV interval (65 ms) at baseline. VT1, which demonstrated left axis deviation with right bundle branch block QRS morphology and TCL of 460 ms, was easily induced by catheter contact and was most frequently observed prior to the procedure. When we performed 3-dimensional mapping with the PentaRay catheter during tachycardia, sharp, low-amplitude presystolic Purkinje potentials preceding the His bundle potential were recorded in the left posterior fascicle (LPF) region (Supplemental Figure 1), which determined the interval of the tachycardia (Supplemental Figure 2). His potentials
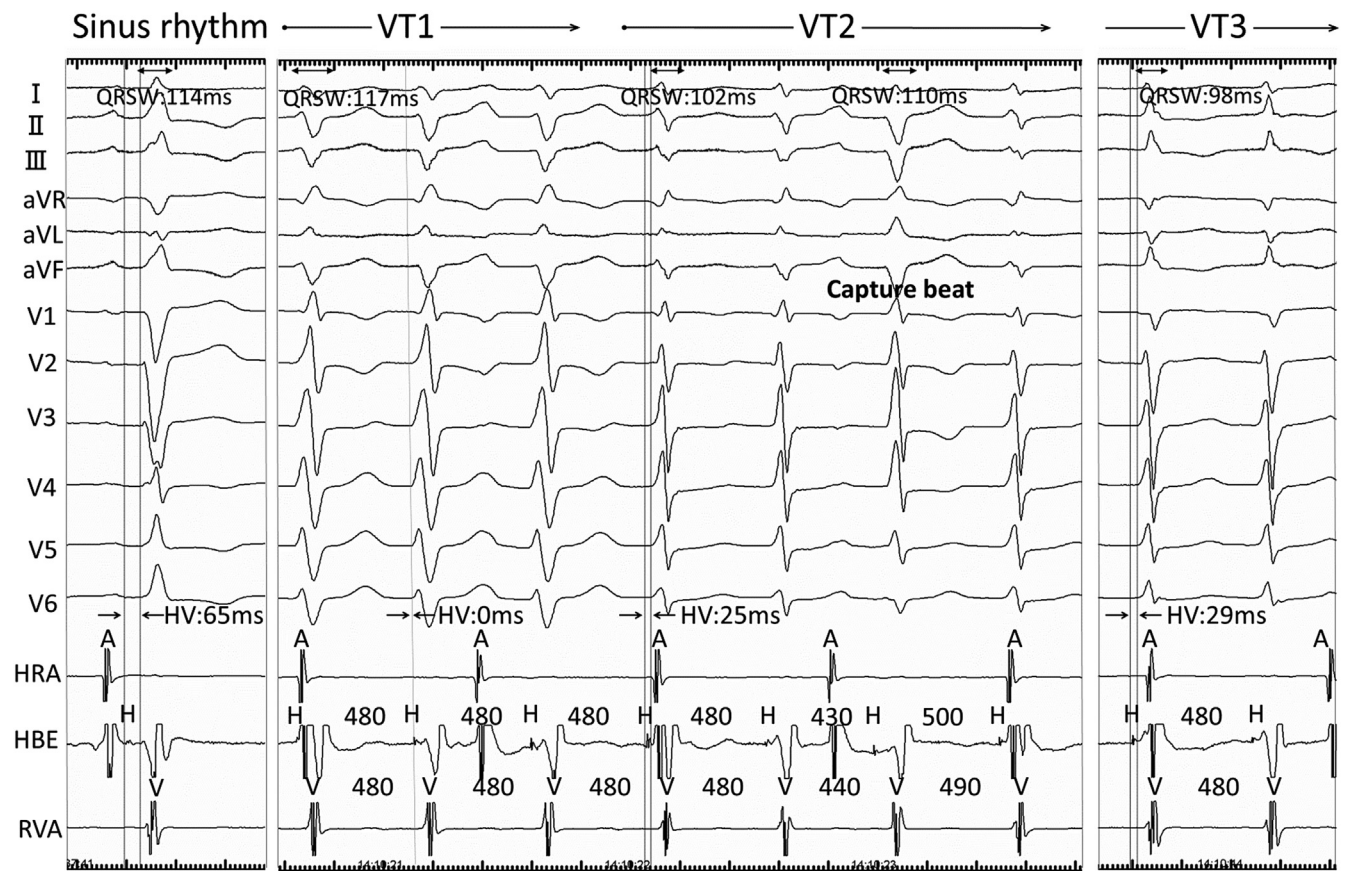
Figure 1 Comparison of QRS morphology and HV interval between sinus rhythm and ventricular tachycardia.

were periodically captured by the sinus rhythm without reset-ting the tachycardia (Figure 2A). These findings enabled us to discard bundle branch reentry and myocardial tachycardia. The electroanatomical map also revealed a low-voltage area in the inferior wall and ventricular septum during VT, including the area where the presystolic Purkinje potential was recorded (Figure 2A and B). In those areas, the Purkinje potentials preceding the QRS complex were also recorded during the sinus rhythm. We have repeatedly tried to entrain the VT from this region without success. The VT was easily terminated, and the Purkinje potentials could not be selec-tively captured. Activation mapping failed to cover the entire length of the tachycardia cycle. Meanwhile, the QRS morphology of VT1 subsequently changed to VT2 and then to VT3 with stepwise prolongation of the HV interval, without change in H-H interval and TCL (HV interval of 0, 25, and 29 ms, respectively). All the VTs were equivalent to those documented prior to the procedure (Figure 1).

Right ventricular apical pacing created constant fusion, confirming reentry as the mechanism of VT. Even though the Purkinje potentials could not be entrained during tachy-cardia, the Purkinje potentials determined the TCL (Supplemental Figure 2), and the reentrant VT utilizing the Purkinje network in the LPF region was diagnosed.

Although the earliest activation site of the presystolic Pur-kinje potential during tachycardia was at the basal septum,

radiofrequency (RF) application was first delivered more distally to minimize the risk of damaging the LPF. VT1 grad-ually became noninducible; and with proximal movement of the RF application site, VT2 also became noninducible, while VT3 with normal axis and LBBB QRS configuration remained with mild prolongation of the TCL (520 ms) but became nonsustained (Figure 3A). Careful mapping revealed prominently delayed potentials at the basal septum of the left ventricle (LV) during the sinus rhythm, 10 mm below the His potential, which transitioned to mid-diastolic potentials (DPs) conducting to Purkinje potentials with initiation of the VT (Figure 3A–C). QRS-DP block repetitively led to termination of VT. Interestingly, pacing from this site created multiple QRS configurations including that of clin-ical VT with different pacing-QRS intervals (Figure 3D). This site was targeted for ablation (Supplemental Figure 3), which led to noninducibility of all VTs. DP was eliminated by additional RF applications around the area. The patient has completed 2 years of follow-up without VT recurrence.

Discussion
Our case of reentrant VT involving the Purkinje network demonstrated interesting findings: the transition of QRS mor-phologies during VT without termination or change in TCL;
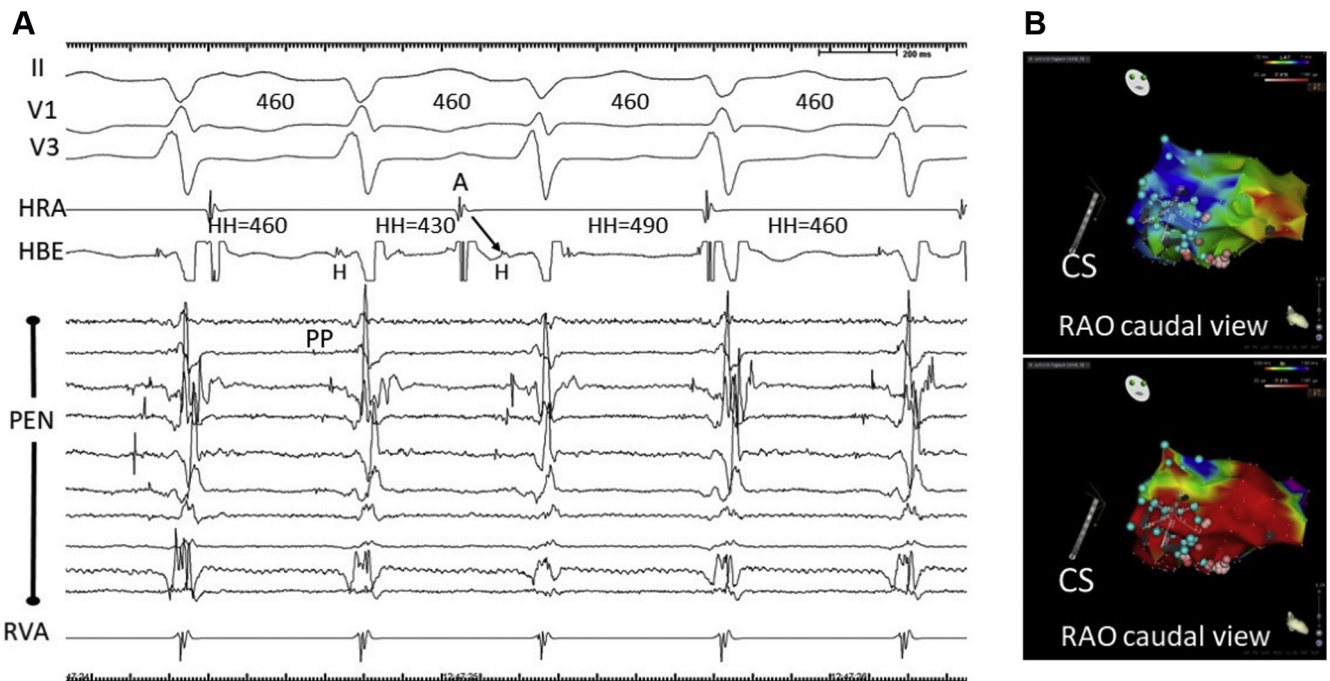
Figure 2 During ventricular tachycardia. **A:** PentaRay (Biosense Webster, Diamond Bar, CA) catheter records the Purkinje potential (PP) in the left posterior fascicule region. Sinus beats occasionally capture the His potential, which created fusion beat without reset phenomenon (*arrow*). **B:** Three-dimensional map of the left ventricle (LV; CARTO system, Biosense Webster, Diamond Bar, CA). The activation map shows that the earliest local myocardial activities are in the mid-apical septum. The voltage map shows the dense scar areas (bipolar electrograms <0.5 mV) extending widely in the LV. The light blue dots indicate the site where PPs are recorded during VT1 in dense scar areas. The first site of radiofrequency application was described as VisiTag (Biosense Webster, Diamond Bar, CA) in the distal portion of the left posterior fascicule region. CS = coronary sinus; RAO = right anterior oblique.

multiple narrow QRS VTs including normal axis; and pace map at successful RF application site providing multiple stimulus QRS intervals and paced QRS morphologies, including those of clinical VTs. To the best of our knowledge, our report is the first to present all these findings on Purkinje-related reentrant VTs in patients with structural heart disease.

The Purkinje potentials could not be entrained during tachycardia, and the exact level of participation of the Purkinje network in the reentry circuit is unclear. However, P-P intervals clearly determined the following H-H and V-V intervals (Supplemental Figure 2), and this finding has been shown as the criterion to demonstrate participation of Purkinje fibers in the circuit.[2] Moreover, QRS-DP block preceded VT3 termination. Contrarily, DP-Purkinje potential conduction led to onset of VT3 (Figure 3A). From these findings, DP and Purkinje potentials are highly suspected to comprise the critical portion of the VT circuit. The origin of the DP may be of debate, since damaged Purkinje fibers and ventricular myocardium are both reasonable.

We would like to emphasize the sequential changes in VT morphologies without termination or change in TCL. Interestingly, their morphologies included both left axis deviation and normal axis, showing the important mechanism of this multiform tachycardia. Limited to patients without structural

heart disease, the normal-axis QRS configuration of fascicular VT has been discussed as the upper septal type of idiopathic left VT (ILVT) in some previous studies.[4,5] Talib and colleagues[4] hypothesized that the mechanism of this entity was the reversed form of typical ILVT with left axis deviation, utilizing the same circuit. Our case also presented with QRS morphology compatible with typical and upper septal ILVT. However, if the proposed mechanism was applied in our case, VT with left axis deviation would require termination before initiation of VT with normal axis, owing to the opposite direction of the circuit. Sequential shift in QRS with the same length of the H-H interval and tachycardia cycle is discordant with this hypothesis. Our findings suggested that all VTs shared the same circuit with the same direction of activation, while the exit site to LV fascicles changed during VT. This assumption was in line with the observation that as the QRS axis shifted from left axis deviation to normal axis, HV interval prolonged.

During VT showing normal axis morphology of QRS, it was considered that exit of VT was positioned near the proximal portion of the left bundle or fascicles. Histopathologic examinations revealed divergence of more than 2 fibers from the proximal portion of HPS in most cases.[6] Sung and colleagues[7] reported that this complex network is suspected to play an important role in creating normal axis and narrow QRS during VT; however, reports regarding normal axis type
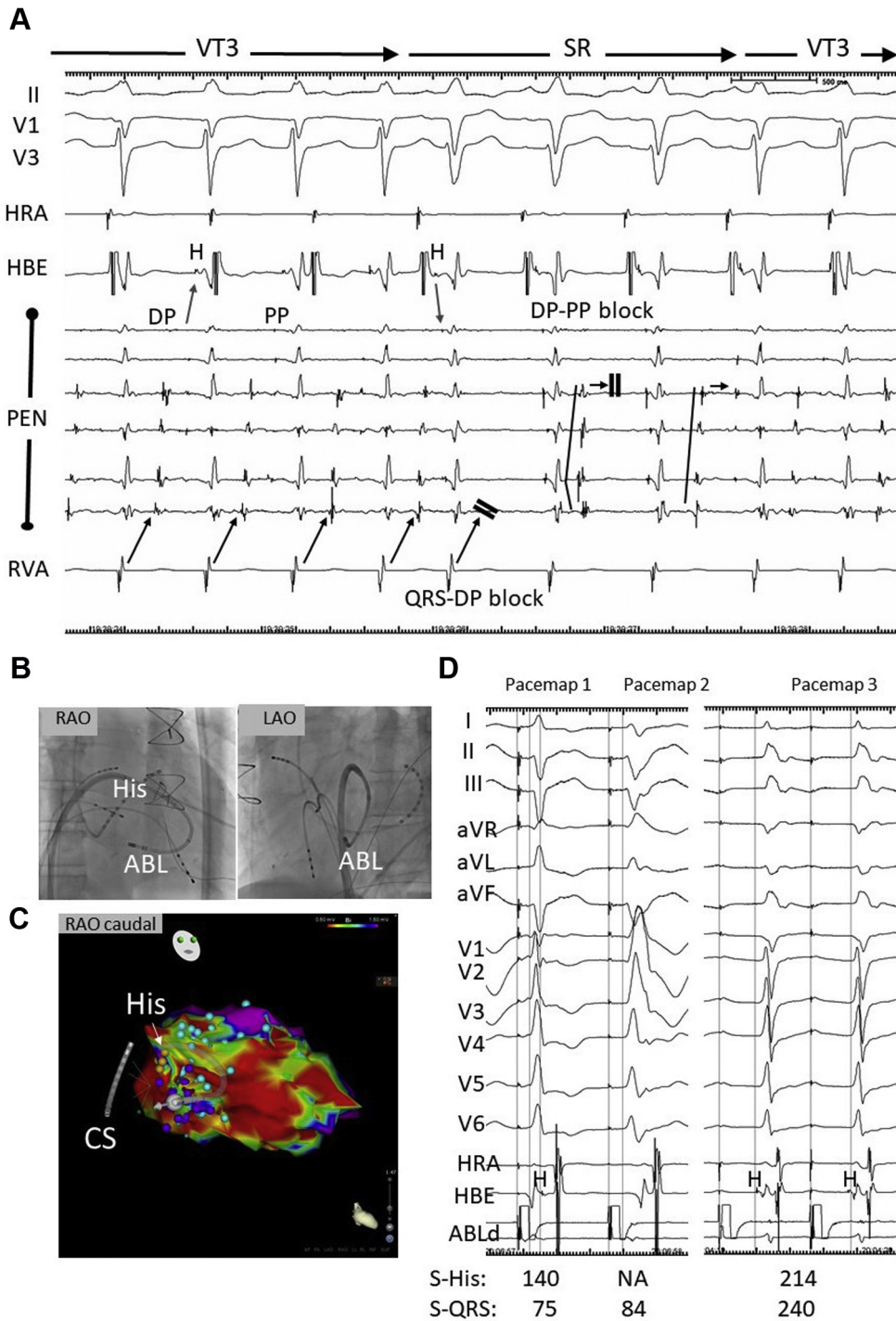
Figure 3 Documented potentials leading to nonsustained ventricular tachycardia (VT). **A:** Mid-diastolic potentials (DPs) are observed in the PentaRay (Biosense Webster, Diamond Bar, CA) catheter. After the sinus rhythm captured His and Purkinje potentials, QRS-DP block occurred repetitively. **B:** Right anterior oblique (RAO) and left anterior oblique (LAO) views of fluoroscopy of pacing and success site of the left ventricle. **C:** Three-dimensional map of the left ventricle. The purple dots indicate the site where the DPs were recorded. The white dot indicates the site where the pace map (**D**) created various QRS morphologies with different s-QRS intervals. ABL = ablation; CS = coronary sinus.

of VT with narrow QRS are scarce. Interestingly, the activation of the Purkinje network during VT is disorganized (Supplemental Figure 1). It appears to be different compared to cases of ILVT, which may be secondary to the complex network of the Purkinje fibers and myocardial injury of this area in this patient.

Although the treatment strategy is still controversial, in our case HPS was highly suspected to compose the critical component of the reentry circuit and there was a potential risk of advanced or complete AV block. Various QRS morphologies were generated by pacing in this region, while some resembled clinical VTs with accompanying various

s-QRS delays. We suspect that the common pathway of all VTs was located in this area in our case; it was compatible with multiple exit hypothesis of LV fascicles as an explanation of morphologic changes in QRS. It might be worth targeting these sites, similar to the strategy for other Purkinje-related VTs.[8] Indeed, successful RF application was achieved at the basal inferior septum, which was in the vicinity of the branches of the His bundle, to eliminate all the VTs without AV prolongation or block.

Conclusion

We experienced multiform VT with involvement of HPS in a patient with ischemic cardiomyopathy. Sequential change in QRS morphology during VTs indicated the important mechanisms of narrow QRS VTs in patients with structural heart disease. The critical site of all VTs was found in the left ventricular septal region, and RF application resulted in successful treatment. Cautious interpretation of QRS morphologies and HV interval during sinus rhythm and VT was crucial for interpretation of the mechanism, as well as safe and efficient treatment. Further evidence on HPS-related narrow QRS VTs in patients with structural heart disease is warranted to better understand this entity and examine the generalizability of our findings.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2019.08.013.

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