# An unusual long RP tachycardia: What is the mechanism?

Yasuyuki Egami<sup>1</sup>, Kohei Ukita<sup>1</sup>, Akito Kawamura<sup>1</sup>, Hitoshi Nakamura<sup>1</sup>, Yutaka Matsuhiro<sup>1</sup>, Koji Yasumoto<sup>1</sup>, Masaki Tsuda<sup>1</sup>, Naotaka Okamoto<sup>1</sup>, Akihiro Tanaka<sup>1</sup>, Yasuharu Matsunaga-Lee<sup>1</sup>, Masamichi Yano<sup>1</sup>, Ryu Shutta<sup>1</sup>, Masami Nishino<sup>1</sup>, and Jun Tanouchi<sup>1</sup>

<sup>1</sup>Osaka Rosai Hospital

October 28, 2020

# **EP** Rounds

## An unusual long RP tachycardia: What is the mechanism?

Yasuyuki Egami, MD, Kohei Ukita, MD, Akito Kawamura, MD, Hitoshi Nakamura, MD, Yutaka Matsuhiro, MD, Koji Yasumoto, MD, Masaki Tsuda, MD, Naotaka Okamoto, MD, Akihiro Tanaka, MD, Yasuharu Matsunaga-Lee, MD, Masamichi Yano, MD, PhD, Ryu Shutta, MD, Masami Nishino, MD, PhD, Jun Tanouchi, MD, PhD.

Division of Cardiology, Osaka Rosai Hospital, Osaka, Japan

**Funding:** This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest statement: We have nothing to disclose.

Address for correspondence: Masami Nishino, MD, PhD, FACC, FESC

Division of Cardiology, Osaka Rosai Hospital.

1179-3, Nagasone-cho, Sakai-city, Japan.

E-mail:mnishino@osakah.johas.go.jp

#### Case presentation

A 77-year-old woman with frequent palpitations was referred to our institution for catheter ablation. Transthoracic echocardiography did not reveal any abnormalities. The baseline 12-lead electrocardiogram did not show any pre-excitation. The 12-lead electrocardiogram during the tachycardia exhibited a narrow complex tachycardia with a long RP interval (heart rate: 200 bpm). Programmed atrial and ventricular stimulation did not reveal any distinct dual nodal physiology. Retrograde ventriculoatrial (VA) conduction revealed a midline and decremental property. The retrograde atrial earliest site was located in the postero-septal region. Para-hisian pacing demonstrated a typical nodal response. The tachycardia was reproducibly induced by atrial extrastimuli, without a critical atrio-ventricular prolongation. The atrial activation sequence during the tachycardia was identical to that during right ventricular pacing. Entrainment from the right ventricular apex exhibited a V-A-V response. The differences between the corrected post pacing interval and tachycardia cycle length was 168ms (> 110ms) (Figure 1A). The tachycardia was sustained with a 2:1 AV relationship (Figure 1B). The tachycardia was terminated by a His-refractory ventricular premature depolarization (VPD) from the right ventricular apex (Figure 2A). Based on these findings,

what was the mechanism of the tachycardia and the response to the pacing maneuver? **Discussion** The differential diagnosis of a long RP supraventricular tachycardia with the earliest atrial activation near the ostium of the coronary sinus includes atrial tachycardia originating from the postero-septal region, orthodromic tachycardia using a slow-conducting accessory pathway, atypical atrioventricular nodal reentrant tachycardia (AVNRT), nodofascicular (NF) reentrant tachycardia, and atypical AVNRT with bystander NF or nodoventricular (NV) bypass tract. The mechanism of a narrow QRS tachycardia can be diagnosed by combining several standard pacing maneuvers and observations, as follows: (1) whether the tachycardia is involved in a nonobligatory 1:1 AV relationship, (2) the effect of bundle branch block on the VA interval, (3) a V-A-V or V-A-A-V response to ventricular entrainment; (4) a corrected post-pacing interval after ventricular entrainment compared to the tachycardia cycle length (cPPI-TCL), and (5) whether a VPD timed to the His-refractory period disrupts the tachycardia.<sup>1,2</sup>In our patients, the V-A-V response to ventricular entrainment excluded atrial tachycardia (Figure 1A). The long corrected PPI-TCL (168 ms) after the right ventricular entrainment pacing suggested that the ventricular pacing site was far away from the tachycardia circuit. The tachycardia with a 2:1 AV relationship absolutely excluded orthodromic tachycardia using a slow conducting accessory pathway (Figure 1B). Based on these findings, the tachycardia was diagnosed as atypical AVNRT. However, the AVNRT was reproducibly terminated by a His-refractory VPD, which did not reset the tachycardia cycle length because of a conflict with the downstream wavefront of the AVNRT at a site below the His bundle. However, the presence of an NF or NV bypass tract inserting into the retrograde limb of the AV nodal circuit could provide a reasonable explanation for the termination of the tachycardia by a His refractory VPD.<sup>3,4</sup> The mechanism is shown by the laddergram in **Figure 3**. Briefly, at the timing of the His-refractory VPD, the wavefront of the tachycardia had already reached the retrograde limb of the AVNRT circuit after the lower turnaround point. The wavefront of the VPD collided with the downstream wavefront of the AVNRT within the right ventricular. On the other hand, the wavefront via the NF or NV bypass tract entered the retrograde limb of the AVNRT circuit ahead of the wavefront of the AVNRT after the lower turnaround point. Then, the wavefront via the NF or NV bypass tract allowed the tachycardia to terminate due to collision with the remaining refractory period of the retrograde slow pathway.

Radiofrequency energy delivered in the posterior septal region where the earliest activation was recorded during the tachycardia, terminating the tachycardia within 2 seconds. After the ablation, VA conduction via the retrograde fast pathway was observed, but no tachycardia was induced.

In summary, we presented a case of an atypical AVNRT with a concealed, bystander NF or NV bypass tract, that was confirmed by a response to a His refractory VPD during the tachycardia.

# Acknowledgments

The authors thank Keiji Yamamoto, Atsushi Shiono, and Tomoyoshi Morioku for their technical assistance during the electrophysiological study, Kohei Ukita, Koji Yasumoto, Yasuharu Matsunaga-Lee, and Masamichi Yano for their assistance of electrophysiological interpretation during the procedure, and Mr. John Martin for his linguistic assistance with this manuscript.

## Reference

1. Ho RT, Frisch DR, Pavri BB, Levi SA, Greenspon AJ. Electrophysiological Features Differentiating the Atypical Atrioventricular Node–Dependent Long RP Supraventricular Tachycardias. *Circ Arrhythm Electrophysiol*. 2013;6(3):597-605. doi:10.1161/CIRCEP.113.000187

2. González-Torrecilla E, Arenal A, Atienza F, et al. First postpacing interval after tachycardia entrainment with correction for atrioventricular node delay: a simple maneuver for differential diagnosis of atrioventricular nodal reentrant tachycardias versus orthodromic reciprocating tachycardias. *Heart Rhythm* . 2006;3(6):674-679. doi:10.1016/j.hrthm.2006.02.019

3. Bansal S, Berger RD, Spragg DD. An unusual long RP tachycardia: What is the mechanism? *Heart Rhythm* . 2015;12(4):845-846. doi:10.1016/j.hrthm.2014.12.027

4. Sahin M, Ozeke O, Cay S, et al. An uncommon response to a His refractory premature ventricular

complex during a short RP supraventricular tachycardia: What is the mechanism? *Pacing Clin Electrophysiol* . 2019;42(7):1050-1053. doi:10.1111/pace.13719

## **Figure Legend**

Figure 1 (A) A V-A-V response after ventricular entrainment pacing. Corrected PPI-TCL = (492-320) - (92-88) = 168 ms > 110 ms, (B) 2:1 conduction during the tachycardia.

CS = coronary sinus; HRA = high right atrium; H or HBE = His bundle electrogram; RVA = right ventricular apex.

Figure 2. Intracardiac recording (A) and 12-lead ECG(B) at the timing of a His refractory ventricular premature depolarization (VPD) during the tachycardia. The black square box in (A) is a 12-lead ECG at the same timing as that in (B). (C) RVA pacing (pacing cycle length = 300 ms) during sinus rhythm. The VPD was delivered a little earlier than the expected His bundle activation, but the QRS morphology of the His refractory VPD (Figure 2B) had a fused QRS morphology of the sinus rhythm and ventricular pacing (Figure 2C).

S = stimulus, SR = sinus rhythm. The other abbreviations are as shown in Figure 1.

**Figure 3.** At the timing of the His refractory VPD, the wavefront of the tachycardia had already reached the retrograde limb of the AVNRT circuit after the lower turnaround point. The wavefront (blue arrow) in the myocardium was directly captured by a His-refractory that VPD collided with the AVNRT wavefront (yellow arrow) in the right bundle branch or RV chamber. The collision of these wavefronts caused the fused QRS. The wavefront (broken green arrow) via the NF or NV bypass tract with the same His refractory VPD entered the retrograde limb of the AVNRT circuit, dividing it into the two directions. One wavefront (red arrow) collided with the wavefront (purple arrow) of the AVNRT after the lower turnaround point, and the other wavefront (green arrow) allowed the tachycardia to terminate due to collision with the refractory period of the retrograde slow pathway. The vertical black dashed line indicates the timing of the His bundle activation.

A = atrium, Ant F = antegrade fast pathway, LCP = lower common pathway, Ret S = retrograde slow pathway, the other abbreviations are as shown in Figure 1.





