

# Entrainment response during ventricular tachycardia originating from the para-Hisian region: What is the mechanism?

Kazuki Moriwaki<sup>1</sup>, Atsushi Doi<sup>1</sup>, Keisuke Nishigaki<sup>1</sup>, Takuya Tsukamoto<sup>1</sup>, Sawa Tanaka<sup>1</sup>, Reiko Yamasaki<sup>1</sup>, Keisuke Fukuda<sup>1</sup>, Tomotaka Yoshiyama<sup>2</sup>, Takahiko Kawarabayashi<sup>1</sup>, and Daiju Fukuda<sup>2</sup>

<sup>1</sup>Kikkokai Tane Sogo Byoin

<sup>2</sup>Osaka Metropolitan University Graduate School of Medicine

April 16, 2024

## Abstract

A 70-year-old man presented with regular wide QRS complex tachycardia (WCT) at a rate of 185 beats/min, characterized by left bundle branch block morphology. WCT termination was achieved through electrical cardioversion. A subsequent 12-lead electrocardiogram revealed sinus rhythm with complete right bundle branch block (CRBBB) morphology. Echocardiography showed normal left ventricular function without structural heart disease. An electrophysiological study was then conducted, followed by catheter ablation. At baseline, the atrio-His and His-ventricular intervals were normal. Fractionated ventricular potentials were observed in the para-Hisian region during sinus rhythm. Neither dual atrioventricular (AV) nodal physiology nor ventriculoatrial conduction was observed during programmed pacing. Clinical WCT was induced by ventricular extra-stimulation with concurrent AV dissociation and no visual His bundle potential, confirming the diagnosis of ventricular tachycardia (VT). Figure 1 illustrates 12-lead electrocardiograms recorded during overdrive pacing from the right ventricular apex (RVA) and right atrial septum during VT. The electroanatomical activation map revealed a distinctive focal breakout pattern with fractionated potentials originating from the para-Hisian region. In the noncoronary cusp (NCC), corresponding to the opposite side of the para-Hisian region, prolonged and fractionated ventricular potentials preceding QRS onset by 56 ms were recorded (Figure 2A). Figure 2B shows the intracardiac electrogram recorded during ventricular overdrive pacing from the NCC during VT. Based on these observations, what is the mechanisms underlying the tachycardia?

Entrainment response during ventricular tachycardia originating from the para-Hisian region: What is the mechanism?

Kazuki Moriwaki, MD<sup>1</sup>; Atsushi Doi, MD<sup>1</sup>; Keisuke Nishigaki, MD<sup>1</sup>; Takuya Tsukamoto, MD<sup>1</sup>; Sawa Tanaka, MD<sup>1</sup>; Reiko Yamasaki, MD<sup>1</sup>; Keisuke Fukuda, MD<sup>1</sup>; Tomotaka Yoshiyama, MD<sup>2</sup>; Takahiko Kawarabayashi, MD<sup>1</sup>; Daiju Fukuda, MD<sup>2</sup>.

<sup>1</sup> Department of Cardiovascular Medicine, Tane General Hospital, Osaka, Japan.

<sup>2</sup> Department of Cardiovascular Medicine, Osaka Metropolitan University Graduate School of Medicine, Osaka, Japan

Correspondence to Atsushi Doi, MD, PhD.

Department of Cardiovascular Medicine, Tane General Hospital, 1-12-21, Kujominami, Nishi-ku, Osaka, 550-0025, Japan.

E-mail; [adoi7abl@gmail.com](mailto:adoi7abl@gmail.com)

**Keywords:** entrainment, His bundle, reentry, ventricular tachycardia

## EP ROUNDS

### Case presentation

A 70-year-old man presented with regular wide QRS complex tachycardia (WCT) at a rate of 185 beats/min, characterized by left bundle branch block morphology. WCT termination was achieved through electrical cardioversion. A subsequent 12-lead electrocardiogram revealed sinus rhythm with complete right bundle branch block (CRBBB) morphology. Echocardiography showed normal left ventricular function without structural heart disease. An electrophysiological study was then conducted, followed by catheter ablation. At baseline, the atrio-His and His-ventricular intervals were normal. Fractionated ventricular potentials were observed in the para-Hisian region during sinus rhythm. Neither dual atrioventricular (AV) nodal physiology nor ventriculoatrial conduction was observed during programmed pacing. Clinical WCT was induced by ventricular extra-stimulation with concurrent AV dissociation and no visual His bundle potential, confirming the diagnosis of ventricular tachycardia (VT).

Figure 1 illustrates 12-lead electrocardiograms recorded during overdrive pacing from the right ventricular apex (RVA) and right atrial septum during VT. The electroanatomical activation map revealed a distinctive focal breakout pattern with fractionated potentials originating from the para-Hisian region. In the noncoronary cusp (NCC), corresponding to the opposite side of the para-Hisian region, prolonged and fractionated ventricular potentials preceding QRS onset by 56 ms were recorded (Figure 2A). Figure 2B shows the intracardiac electrogram recorded during ventricular overdrive pacing from the NCC during VT. Based on these observations, what is the mechanisms underlying the tachycardia?

### Commentary

Complex reentrant VT involving the ventricular septal substrate in the para-Hisian region has been reported.<sup>1</sup> Ablation procedures in this anatomical region pose challenges due to the potential risk of damaging the AV conduction system. Accurately determining the optimal ablation site is crucial, necessitating a clear understanding of the arrhythmia mechanism. In the present case, WCT was diagnosed as para-Hisian VT based on AV dissociation, the absence of visual His bundle potentials, and a focal breakout pattern from the para-Hisian region on the electroanatomical activation map.

As shown in Figure 1, overdrive pacing from the RVA during VT reveals the fully pacing morphology, whereas overdrive pacing from the atrial septum exhibits constant fusion, indicating manifest entrainment. These observations led to the determination of a reentry mechanism for VT. Furthermore, the electroanatomical activation map indicated a focal breakout pattern with prolonged and fractionated potentials originating from the para-Hisian region, suggesting a localized reentry involving a slow conduction zone (SCZ) within the interventricular septum with endocardial breakout. Fractionated potentials preceding QRS onset by 56 ms were recorded when a 3.5mm tip ablation catheter was positioned within the NCC, corresponding to the opposite side of the para-Hisian region. Figure 2B illustrates concealed entrainment observed during the first to fourth pacing stimuli of ventricular entrainment pacing at this NCC site. In the fifth pacing stimulus, VT was terminated without ventricular electrocardiograms or QRS waveforms, and VT was reinduced after the last pacing stimulus. This phenomenon, termed VT termination with nonglobal capture by a pacing stimulus, aids in identifying a critical isthmus within the reentry circuit.<sup>2</sup> In the present case, the absence of His bundle potential was confirmed at this site during sinus rhythm, and radiofrequency ablation successfully eliminated the inducible VT without impairing AV conduction.

Figures 3A and B present intracardiac electrograms during sinus rhythm and atrial entrainment pacing, respectively. Ventricular potentials on His-d electrodes were captured antidromically, and RVA potentials were captured orthodromically, demonstrating manifest entrainment. On the His 3,4 electrode, the fractionated ventricular potential preceding QRS onset by 56 ms was documented during VT, and only the negative potential of the initial half component, circled in red, was captured orthodromically with 0 ms preceding QRS onset during atrial entrainment pacing. These observations suggested that the negative potential of the initial half-component corresponded to the exit site of the SCZ of VT reentry circuit.

We explored the mechanism by which the overdrive pacing from the atrium, rather than from the RVA, demonstrated manifest entrainment. We speculated that the re-entrant circuit was within the interventricular septum, where the exit and entrance sites of the SCZ were located on the right ventricular (RV) and left ventricular (LV) sides, respectively (Figure 3C). The entrainment response from the RVA demonstrated an absence of constant fusion, as the N-th paced antidromic wavefront reached the exit of the SCZ earlier than the N-1st paced orthodromic wavefront. In contrast, the entrainment response from the atrium demonstrated constant fusion. This phenomenon is caused by the N-1st paced wavefront conducting through the left bundle due to the CRBBB. The wavefront then reached the entrance of the SCZ via the Purkinje network on the ventricular septal surface formed by the fascicles of the left bundle branch. Subsequently, it collided with the N-th antidromic wavefront after propagating from the exit of the SCZ toward the RVA (Figure 3C).

The present case highlights the effectiveness of the entrainment pacing from both the atrium and the NCC in identifying the optimal ablation site. This approach could minimize the risk of injuring the AV node while confirming the VT mechanism as reentry.

## References

1. Enriquez A, Tapias C, Rodriguez D, Liang J, Marchlinski F, Saenz L, Garcia F. How to map and ablate parahisian ventricular arrhythmias. *Heart Rhythm* 2018;15:1268-1274.
2. Bogun F, Krishnan SC, Marine JE, Hohnloser SH, Schuger C, Oral H, Pelosi F, Chugh A, Morady F. Catheter ablation guided by termination of postinfarction ventricular tachycardia by pacing with nonglobal capture. *Heart Rhythm* 2004;1:422-426.

## FIGURE LEGENDS

### Figure 1

Twelve-lead electrocardiogram during sinus rhythm (A), right ventricular apex pacing only (B), overdrive pacing at a cycle length of 330 ms during ventricular tachycardia from the right ventricular apex (C) and the right atrial septum (D).

### Figure 2

(A) Intracardiac electrogram during ventricular tachycardia with a cycle length of 325 ms originating from the Para-Hisian region. Prolonged and fractionated ventricular potentials preceding the QRS onset by 56 ms were recorded within the noncoronary cusp, corresponding to the opposite side of the para-Hisian region.

(B) Intracardiac electrogram during ventricular overdrive pacing at a cycle length of 300 ms from the non-coronary cusp during the ventricular tachycardia. The arrows indicate the artifact of the pacing stimulus that was not captured. I, II, V1, V3, V5 = surface electrogram; HRA = high right atrium; His d and His p = distal and proximal His bundle region; NCC d and NCC p = distal and proximal noncoronary cusp; RV d and RV p = distal and proximal right ventricle; S = pacing. The numbers indicate the cycle lengths in ms.

### Figure 3

(A) Intracardiac electrograms during sinus rhythm.

(B) Intracardiac electrograms during atrial entrainment pacing at 330 ms during the ventricular tachycardia with a cycle length of 345 ms.

(C) Schematic representation of the slow conduction zone of the ventricular tachycardia circuit and wavefront propagation during the atrial entrainment pacing.

The numbers indicate cycle lengths in ms. H = His bundle potential; LPF = left posterior fascicle; NCC = noncoronary cusp; RAS = right atrial septum; RB = right bundle; RVA = right ventricular apex; SCZ = slow conduction zone; VT = ventricular tachycardia; N-1 and N = N-1st orthodromic and N-th antidromic paced wavefronts. Other abbreviations are presented as in Figure 2.



