ELECTROCARDIOGRAM UNKNOWNS

# Atrial overdrive pacing during right bundle branch tachycardia: What is the mechanism?

Balaram Krishna J. Hanumanthu, MD, Saman Nazarian, MD, PhD, FHRS

*From the Section of Cardiac Electrophysiology, Cardiovascular Medicine Division, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania.*

## Introduction

The differential diagnosis for wide complex tachycardia with right bundle branch morphology includes supraventricular tachycardia with aberrancy, pre-excited tachycardia, and ventricular tachycardia (VT). We present a case of a patient with right bundle branch tachycardia who underwent an electrophysiology study.

## Case report

A 24-year-old man with prior medical history of wide complex tachycardia symptomatic with palpitations presented for an electrophysiology study. He first noted palpitations at the age of 18. Following a sustained episode, he presented to the emergency room. He failed to cardiovert to sinus with adenosine and required external cardioversion in the emergency room. He had 3 recurrences despite medical therapy prior to presenting to our center for evaluation. Echocardiography revealed normal biventricular function. Cardiac magnetic resonance imaging revealed nonspecific right ventricular insertion point delayed enhancement. The wide complex tachycardia was readily induced in the electrophysiology lab with ventricular programmed stimulation at 600/340 cycle length. Electrocardiogram (ECG) and intracardiac tracings of sinus rhythm and tachycardia are shown in Figure 1.

What is the mechanism of wide complex tachycardia?

What is the maneuver shown demonstrating?

## Discussion

The ECG and intracardiac recordings at baseline in Figure 1A show sinus rhythm with a normal His-ventricular interval (HV 45 ms) and narrow QRS morphology. Figure 1B demonstrates the tachycardia with a right bundle superior axis morphology with biphasic QRS in lead I. The ECG and intracardiac recordings in Figure 1C demonstrate atrial overdrive pacing (AOD) during tachycardia. AOD captures and accelerates the His potential and the QRS is narrowed with manifest fusion with HV interval similar to that during sinus rhythm. The ability to normalize the QRS with AOD during wide complex rhythm is diagnostic of VT. The AVVA response upon resumption of tachycardia also excludes antedromic atrioventricular reciprocating tachycardia where one would expect an AVA response. This is analogous to VAV response seen during ventricular overdrive pacing in orthodromic atrioventricular reciprocating tachycardia. Also, the long postpacing interval of 760 ms after atrial pacing excludes the atrium as an integral part of the tachycardia circuit.

The HV during sinus is 45 ms and HV interval during VT is -6 ms. The HV interval during tachycardia is less than that recorded during sinus rhythm. In the absence of pre-excitation, this implies retrograde activation of the His bundle. It further implies that the retrograde conduction time from the origin/exit of the tachycardia to the His bundle is less than the antegrade HV conduction time to depolarize the ventricular myocardium. This suggests that the tachycardia circuit involves the His-Purkinje system.[1] As the HV

### KEY TEACHING POINTS

- The differential diagnosis for right bundle branch morphology tachycardia includes ventricular tachycardia, supraventricular tachycardia (SVT) with aberrancy, and pre-excited tachycardia.
- A shorter HV interval in the absence of accessory pathway conduction during tachycardia implies retrograde activation of the His bundle during tachycardia and excludes SVT with aberrancy.
- The ability to narrow the QRS with atrial pacing during tachycardia is diagnostic of ventricular tachycardia.
- Atrial entrainment of tachycardia with AVVA response upon resumption of tachycardia excludes pre-excited tachycardia.

**KEYWORDS** Wide complex tachycardias; Electrophysiology study; Fascicular ventricular tachycardia; Atrial overdrive pacing; Entrainment
(Heart Rhythm Case Reports 2024;10:302–304)

**Address reprint requests and correspondence:** Dr Balaram Krishna J. Hanumanthu, Hospital of the University of Pennsylvania, Pavilion Level 6, One Convention Avenue, Philadelphia, PA 19104. E-mail address: Balaramkrishna.Hanumanthu@pennmedicine.upenn.edu.



https://doi.org/10.1016/j.hrcr.2024.01.016

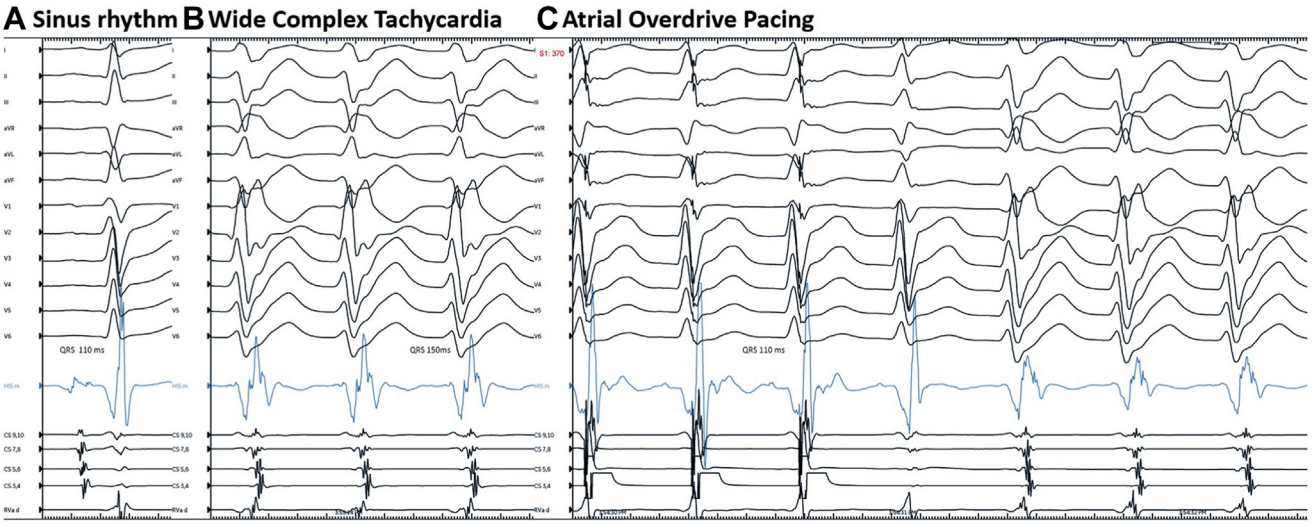


**Figure 1** **A, B:** Twelve-lead electrocardiogram (ECG) and intracardiac recordings during sinus (**A**) and during wide complex tachycardia (**B**), respectively. **C:** Twelve-lead ECG and intracardiac recordings of atrial overdrive pacing from the coronary sinus during tachycardia. The first 4 QRS complexes demonstrate manifest fusion. The tachycardia continues upon cessation of pacing.

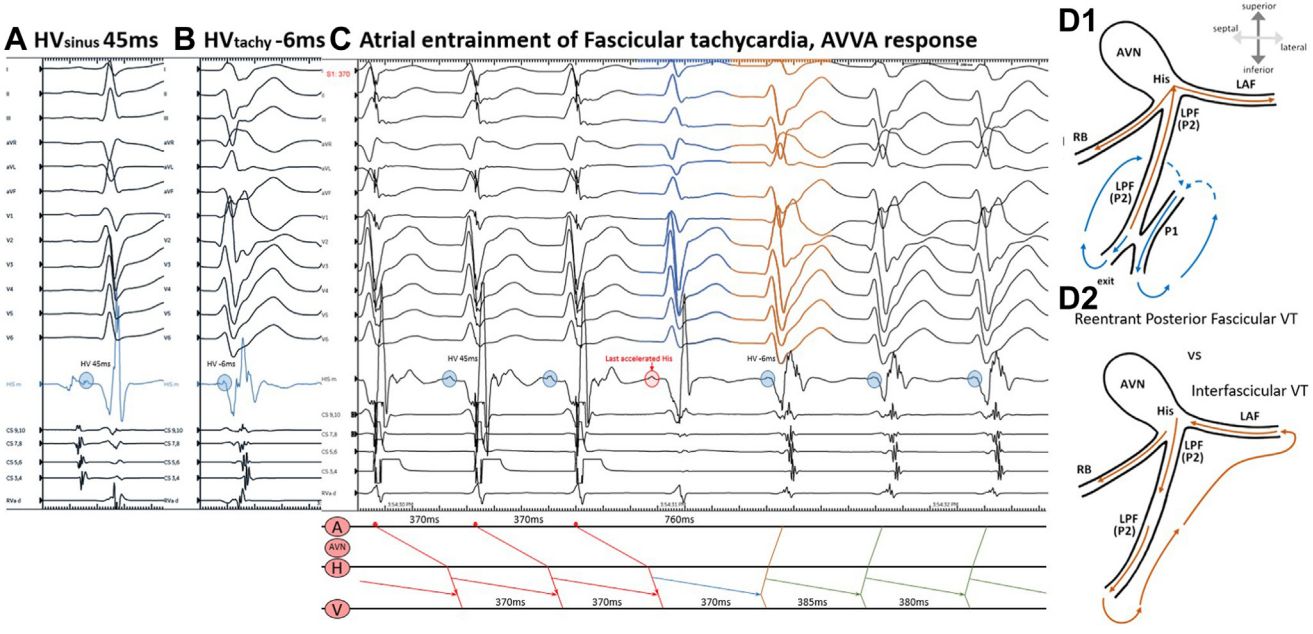


**Figure 2** **A, B:** Electrocardiogram (ECG) and intracardiac recordings showing the HV interval in sinus rhythm and tachycardia, respectively. **C:** ECG, intracardiac recordings, and ladder diagram demonstrating atrial entrainment of fascicular tachycardia. **D:** Schematic illustration of reentrant circuits for left posterior fascicular VT (D1) and interfascicular VT (D2). AVN = atrioventricular node; LAF = left anterior fascicle; LPF = left posterior fascicle; RB = right bundle; VT = ventricular tachycardia.

interval in fascicular VT is a pseudo interval and depends on distance between the His and exit site, it can actually be used to predict the site of origin.[2]

AOD also demonstrates fusion beats except for the last captured beat, which is not fused, fulfilling the first criterion for entrainment. The QRS during atrial entrainment demonstrates narrowing owing to antegrade activation through the conduction system. It resembles sinus rhythm with frontal plane axis of 90 degrees compared to frontal axis of -100 degrees during tachycardia. This represents different activation of the left anterior fascicle (LAF)—antegrade during entrainment and retrograde during tachycardia—as would be expected in posterior fascicular VT. The tachycardia in left posterior fascicle (LPF) VT involves antegrade conduction over the zone of slow conduction in septum (P1); meanwhile the LPF undergoes predominantly retrograde activation (P2).[3] This is in contrast to interfascicular VT, where the tachycardia would require antegrade conduction over the LPF and retrograde conduction over the LAF to demonstrate right bundle, LAF block morphology. This is atypical for interfascicular VT, as it usually involves antegrade conduction over the anterior fascicle.[4] Similarly theoretical supraventricular

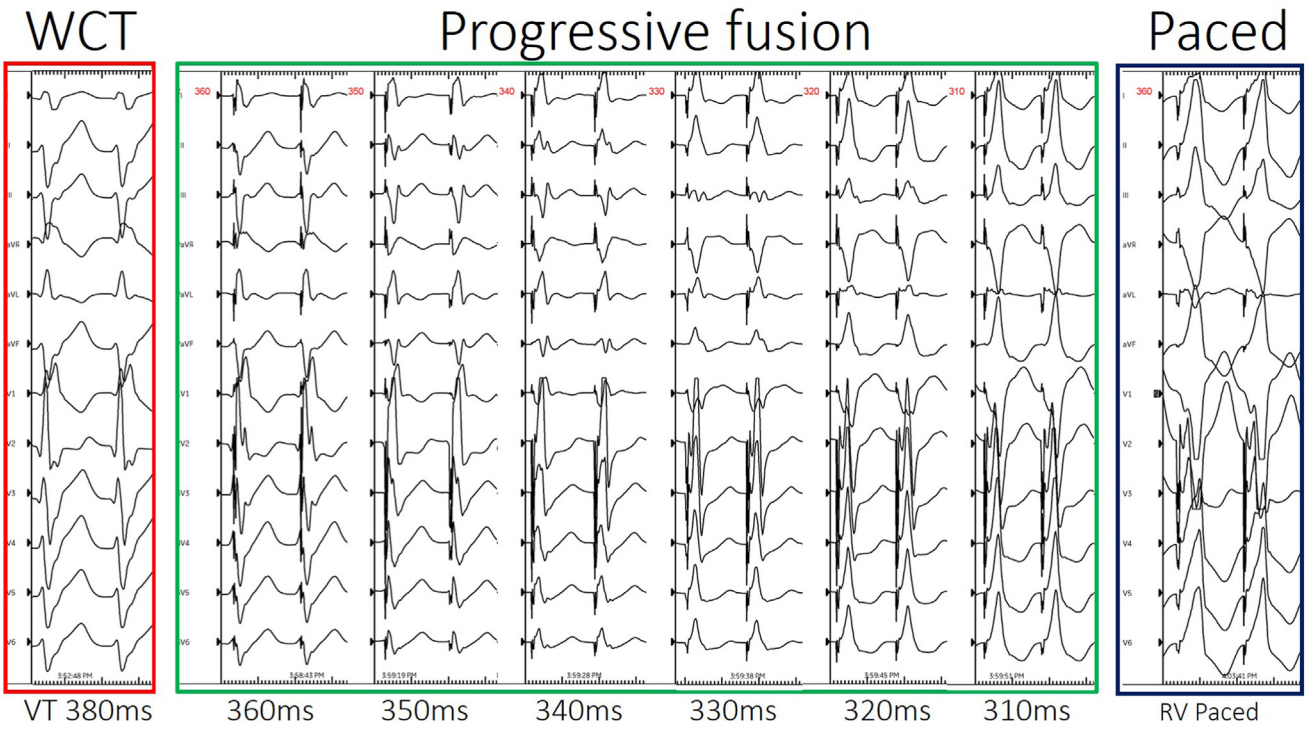


**Figure 3** Twelve-lead morphology of wide complex tachycardia (WCT), progressive fusion with different cycle lengths, and paced morphology in sinus.

capture during AOD during interfascicular VT should continue to demonstrate LAF block morphology and not LPF block, as in our case. These findings are annotated and summarized in Figure 2 along with a ladder diagram. Atrial entrainment with resetting of fascicular VT has been reported before despite ventriculoatrial dissociation.[5] Ventriculoatrial association was present the entire time in our case, which made the need for distinction between supraventricular tachycardia and VT with these maneuvers critical.

The wide complex tachycardia was also confirmed to have a reentrant mechanism, with ventricular pacing demonstrating progressive fusion, fulfilling the second criterion for entrainment, as shown in Figure 3.

The tachycardia was mapped to the LPF region. Linear ablation lesions were applied in the region of the mid to mid inferior septum to target the zone of slow conduction involved in posterior fascicular VT. The patient was also noted to have dual atrioventricular nodal physiology and was inducible for typical atrioventricular nodal reentrant tachycardia. Slow pathway modification was performed without complications. At the conclusion of the case, no ventricular or supraventricular tachycardia was inducible with programmed stimulation. The patient has not had a recurrence of his arrhythmia during 4 months of follow-up.

**Funding Sources:** None.

**Disclosures:** Dr Nazarian is a consultant for CardioSolv and ImriCor and principal investigator for research funding from Biosense Webster, ImriCor, ADAS software, and the US NIH. The University of Pennsylvania Conflict of Interest Committee manages all commercial arrangements. The remaining authors have nothing to disclose.

## References

1. Cohen HC, Gozo EG Jr, Pick A. Ventricular tachycardia with narrow QRS complexes (left posterior fascicular tachycardia). Circulation 1972;45:1035–1043.
2. Ma W, Lu F, Shehata M, et al. Catheter ablation of idiopathic left posterior fascicular ventricular tachycardia: predicting the site of origin via mapping and electrocardiography. Circ Arrhythm Electrophysiol 2017;10:e005240.
3. Nogami A. Purkinje-related arrhythmias part I: monomorphic ventricular tachycardias. Pacing Clin Electrophysiol 2011;34:624–650.
4. Lopera G, Stevenson WG, Soejima K, et al. Identification and ablation of three types of ventricular tachycardia involving the His-Purkinje system in patients with heart disease. J Cardiovasc Electrophysiol 2004;15:52–58.
5. Mohanan Nair KK, Namboodiri N, Thajudeen A, Chaitanya K, Abhilash SP, Valaparambil A. Atrial entrainment during posterior fascicular ventricular tachycardia. J Cardiovasc Electrophysiol 2017;28:719–721.