

# Incessant supraventricular tachycardia following tricuspid valve repair: Unmasking of a nodoventricular pathway



Anil Sriramoju, MBBS, Komandoor Srivathsan, MD, FHRS, Win-Kuang Shen, MD, FHRS

From the Department of Cardiovascular Diseases, Mayo Clinic Hospital, Phoenix, Arizona.

## Introduction

Generally, an accessory pathway (AP) bridges the nonconducting fibrous atrioventricular annulus as a conducting pathway, resulting in supraventricular tachycardia (SVT). It requires the atrium and ventricle as integral components of the tachycardia, regardless of the direction of conduction through the pathway. Uncommon variants of APs like nodofascicular/ventricular (NFV) pathways are extensions from AV node to fascicle or to ventricle and do not require the atrium as an integral component of the tachycardia. We report a case of a 70-year-old woman with incessant SVT following tricuspid valve repair with evidence that concealed ventricular (NFV) pathway was present as a bystander.<sup>1</sup>

## Case report

A 70-year-old woman with dyspnea due to tricuspid valve regurgitation underwent minimally invasive tricuspid valve repair with septal leaflet plication, posteroseptal commissurotomy, and placement of an MC3 30 mm tricuspid ring. Two weeks after surgery, during evaluation at the postoperative visit, the patient was found to be in SVT at 152 beats per minute (Figure 1). An attempt at cardioversion restored sinus rhythm for a few beats with a prompt resumption of the tachycardia, and subsequent amiodarone infusion did not restore sinus rhythm over 48 hours.

Owing to the incessant nature of the tachycardia, she was taken to the electrophysiology lab for an electrophysiology study.

The patient underwent an electrophysiology study with a CARTO mapping system (Biosense Webster, Diamond Bar, CA). The patient was in SVT except during a short interval

## KEY TEACHING POINTS

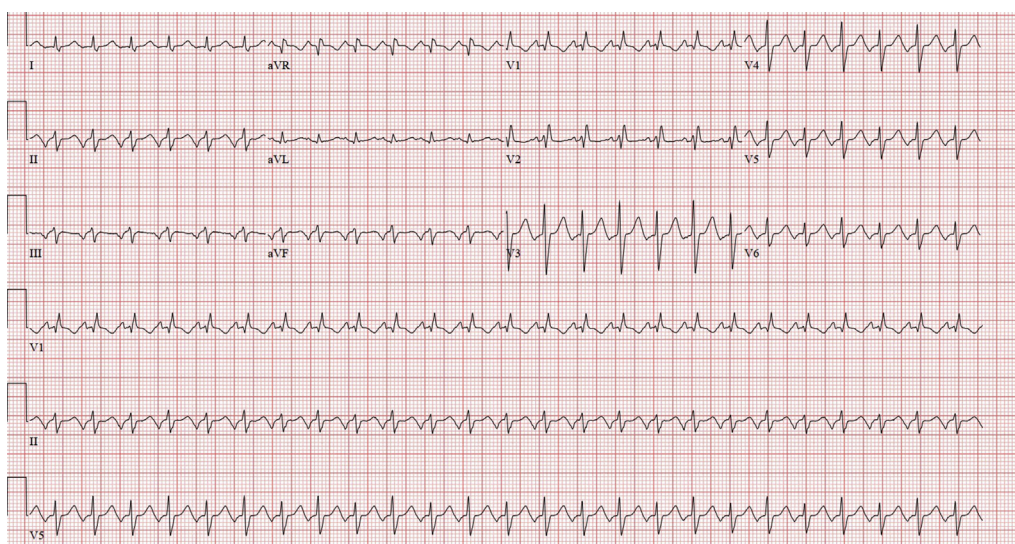
- In nodoventricular tachycardia, His-refractory ventricular premature depolarizations (VPD) advance the His before advancing the atrium, revealing their proximity to the His bundle; these tachycardias are independent of the atrium, but if they do conduct to the atrium, atrial cycle length cannot dissociate from ventricular cycle length.
- Supraventricular tachycardia that meets criteria for atypical atrioventricular nodal reentrant tachycardia, but repeatedly terminates with His-refractory VPD, is due to bystander pathway.
- Tricuspid annular surgery may promote anisotropy and tachycardia that did not exist before the procedure.

after postpacing maneuvers when a few beats of sinus rhythm were noted. Tachycardia cycle length (CL) at baseline was 419 ms. During tachycardia atrial-His (AH) interval was 73 ms, the HV interval was 50 ms, and the VA interval was 273 ms (Figure 2A; the Halo catheter was positioned along the tricuspid annulus at the beginning of the procedure). Fluoroscopic catheter locations are shown in Figure 3 (the ablation catheter had replaced the halo catheter). During sinus rhythm at CL 745 ms, AH was 78 ms and HV was 50 ms.

During SVT, retrograde earliest atrial activation was at CS 9–10, approximately 1 cm from the coronary sinus (CS) os (Figure 2B). Ventricular overdrive pacing at CL 10 or 20 ms was repeatedly terminating throughout the entire study. The termination of SVT always occurred on the first fully captured beat without advancing atrial activation (Figure 2A). His-refractory ventricular premature depolarization (VPD) repeatedly terminated the tachycardia without conducting to the atrium repeatedly (Figure 2C).

**KEYWORDS** Ventricular tachycardia; Nodofascicular; Nodoventricular; Tricuspid valve repair; Accessory pathways (Heart Rhythm Case Reports 2022;8:167–170)

Financial Support: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Declaration of Conflict of Interest: None. Address reprint requests and correspondence: Dr Win-Kuang Shen, Division of Cardiovascular Diseases, Mayo Clinic Hospital, 5777 East Mayo Blvd, Phoenix, AZ 85054. E-mail address: [wshen@mayo.edu](mailto:wshen@mayo.edu).



**Figure 1** Supraventricular long RP tachycardia at 400 ms.

Ablation was performed by using a 4 mm nonirrigated catheter. Activation mapping was performed during tachycardia. The earliest atrial activation was near CS pole 9–10. In this location, the atrial electrogram on the ablation catheter was 30 ms preceding the onset of the P wave and 5 ms preceding the local A on the CS catheter. Tachycardia was terminated 2.24 seconds after the radiofrequency onset.<sup>2</sup>

Additional insurance lesions were delivered here. Postablation repeated programmed stimulation demonstrated no inducible tachycardia with and without isoproterenol. There was no evidence of VA conduction postablation. The patient has remained free of any recurrent arrhythmia at 5 months of follow-up.

## Discussion

The observation of His-refractory VPD repeatedly terminating the tachycardia without conducting to the atrium excluded atrial tachycardia and AP-independent tachycardia.<sup>3</sup>

However, during ventricular overdrive pacing, atrial CL continued at tachycardia CL even though the ventricle was captured at a different CL (with local VA interval change) during progressive fusion (Figure 2A). How to account for the different CL of ventricle and atrium during ventricular overdrive pacing?

In AP-mediated tachycardia, both ventricle and atrium are integral to tachycardia sustenance. Changes in the CL of either atrium or ventricle will influence the CL of the other chamber. Although the atrium is not integral to NFV tachycardia, persistent tachycardia in the atrium at the tachycardia CL while ventricular CL has been accelerated to the pacing

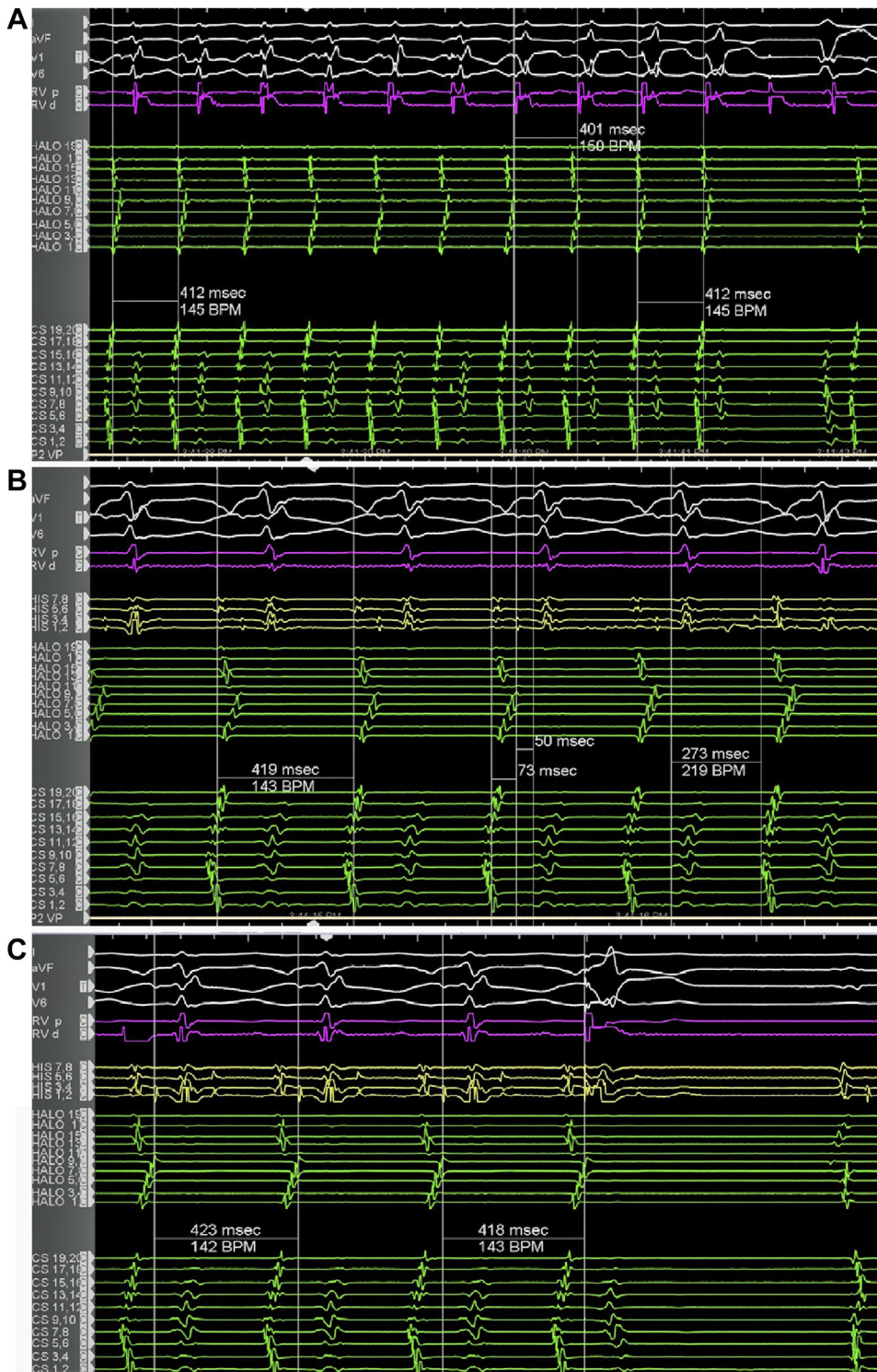
CL is highly unlikely to be NFV pathway-mediated tachycardia. The apparent V dissociation (Figure 2A) from the tachycardia suggests that the underlying mechanism of the clinical tachycardia is atypical atrioventricular nodal reentrant tachycardia (AVNRT) in the presence of a bystander NFV AP.<sup>4</sup> Termination of the atypical AVNRT during His-refractory VPD is due to wavefront collision caused by conduction through bystander NFV within the AV nodal reentrant pathways before the exit to the atrium.

There are several interesting observations worthy of elaboration. The earliest atrial electrogram within 1 cm of the CS os was mapped and ablated successfully.<sup>5</sup> This location is likely near the insertion of the NFV AP to the slow pathway. It is also interesting to note that the atrial activation sequence during tachycardia and ventricular pacing was consistent with the presence of an NFV AP with the earliest activation 1 cm from the CS os. There was no evidence of VA conduction or dual AV nodal physiology postablation with or without isoproterenol.

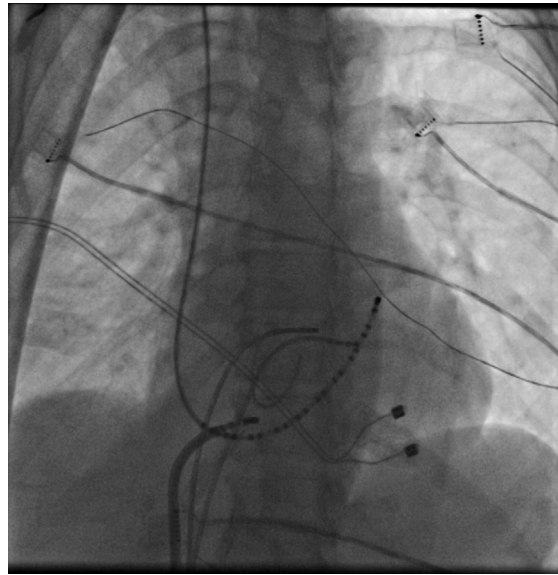
These observations collectively support the presence of an NFV AP but would not be sufficient to prove the NFV AP is integral to tachycardia.

It is uncommon to terminate atypical AVNRT within such a short time using a single lesion. The likely explanation is the proximity of NFV and retrograde limb of the atypical AVNRT circuit (slow pathway) and perhaps the surgical procedure reducing the spatial separation between the two.

Why the tachycardia occurred after tricuspid valve repair is unclear but likely related to the alteration of the anisotropic conduction of these conducting pathways, facilitating appropriate excitable gap (large excitable gap) providing substrate for the incessant nature of the tachycardia.



**Figure 2** **A:** Three fused ventricular paced beats at a different cycle length relative to atrial cycle length at tachycardia cycle length. The last fully paced beat terminated the tachycardia without advancing to the atrium (halo catheter placed around tricuspid annulus, planning for atrial flutter ablation, but was subsequently removed (named as PV 1–20). **B:** Retrograde earliest atrial activation was at CS 9–10, approximately 1 cm from the coronary sinus os during supraventricular tachycardia. **C:** His-refractory ventricular premature depolarizations terminating the tachycardia.



**Figure 3** Fluoroscopic view during coronary angiography.

## References

1. Ho RT, Ortman M, Levi SA. The various manifestations of concealed nodofascicular/nodoventricular bypass tracts. *Heart Rhythm* 2020;17:1280–1290.
2. Weiss M, Ho RT. Pathophysiology, diagnosis, and ablation of atrioventricular node-dependent long-R-P tachycardias. *J Innov Card Rhythm Manag* 2020;11:4046–4053.
3. Sekihara T, Nakano T, Yasuoka Y, Minamiguchi H. A case of typical and atypical atrioventricular nodal reentrant tachycardia with a bystander nodoventricular pathway diagnosed based on the findings during early ventricular premature contractions. *HeartRhythm Case Rep* 2020;6:680–684.
4. Wei W, Sang C, Zheng S, et al. Electrophysiological manifestations of rare supra-ventricular tachycardias with concealed nodo-ventricular fibers. *J Interv Card Electrophysiol* 2021;62:31–38.
5. Han FT, Riles EM, Badhwar N, Scheinman MM. Clinical features and sites of ablation for patients with incessant supraventricular tachycardia from concealed nodofascicular and nodoventricular tachycardias. *JACC Clin Electrophysiol* 2017;3:1547–1556.