# New-onset atrioventricular nodal reentrant tachycardia after transaortic valve replacement: Is there a causal link?

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## Introduction

The incidence of isolated PR prolongation post–transaortic valve replacement (TAVR) is up to 13%, and is considered to have a benign prognosis.[1] The incidence of new-onset arrhythmias post-TAVR is reported at 31%[2]; most are atrial fibrillation (19%). Only 10% and 2% are noted to be ventricular tachycardia (VT) and supraventricular tachycardia (SVT), respectively.[2]

We report a unique case of new-onset atrioventricular nodal reentrant tachycardia (AVNRT), likely caused by post-TAVR delay in atrioventricular nodal conduction.

## Case report

A 71-year-old man with no history of arrhythmias underwent percutaneous implantation of a 29-mm Evolut™ FX TAVR valve (Medtronic, Minneapolis, MN) for severe, symptomatic aortic stenosis. A pre-TAVR 12-lead electrocardiogram (ECG) showed a PR interval of 180 milliseconds and a QRS duration of 88 milliseconds. Immediately post-TAVR, he developed a left bundle branch block (LBBB) with a QRS duration of 150 milliseconds. The PR interval remained dynamic in near-term follow-up extending up to 386 milliseconds 12 days post-TAVR (Figure 1A). At 168 days after TAVR, the PR interval had reduced to 212 milliseconds without normalizing to baseline (Figure 1A).

The patient reported new-onset palpitations approximately 12 weeks after his TAVR procedure. The palpitations occurred daily and lasted several minutes to hours, with sudden onset and offset. A KardiaMobile® 6-lead personal ECG monitor (AliveCor, Mountainview, CA) tracing during a symptomatic episode demonstrated a wide complex tachycardia (WCT) at 143 beats/min (Figure 1B).

Due to increased density of symptoms, after obtaining informed consent, the patient underwent an electrophysiology study approximately 6.5 months (194 days) after the TAVR procedure. Baseline PR interval was 210 milliseconds, with an HV interval of 70 milliseconds and AH interval of approximately 140 milliseconds. The QRS duration was 150 milliseconds with an LBBB morphology. Ventriculo-atrial conduction was concentric and decremental. Para-Hisian pacing at 600 milliseconds revealed AV nodal response. Baseline AV nodal conduction was slow with AV Wenckebach block noted at 450 milliseconds. An "AH jump" was not observed with atrial extrastimulation; however, sustained slow conduction was evident during atrial burst pacing, suggesting the existence of dual AV nodal pathways. With isoproterenol provocation, atrial incremental pacing easily induced a WCT with the same baseline LBBB morphology at a cycle length of 480–520 milliseconds, similar to the clinical tachycardia. The WCT had a 1:1 AV relationship with a septal VA time of –30 milliseconds (Figure 2A), with the earliest atrial activation

### KEY TEACHING POINTS

- Differential diagnosis of de novo occurrence of supraventricular and wide complex tachycardia post–transaortic valve replacement (TAVR) should include atrioventricular (AV) nodal reentrant tachycardia.
- De novo AV nodal reentrant tachycardia can occur post-TAVR, probably from injury to AV node and it's extensions causing PR prolongation.
- AV nodal slow-pathway ablation seems to be an effective treatment for post–TAVR de novo AV nodal reentrant tachycardia.

**KEYWORDS** Transaortic valve replacement; Atrioventricular nodal reentrant tachycardia; Wide complex tachycardia; PR prolongation; Left bundle branch block

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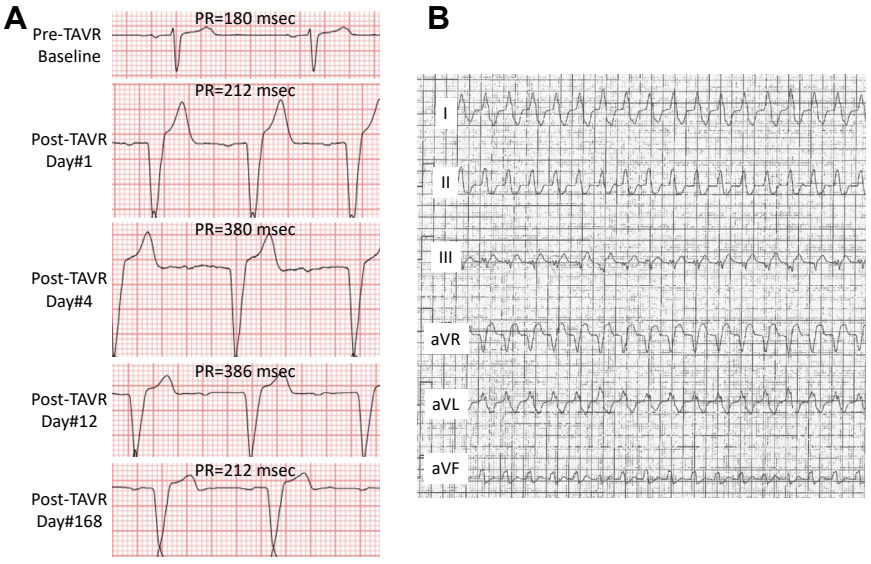


**Figure 1** **A:** The lead V1 of a 12-lead electrocardiogram showing progressive PR interval prolongation after transaortic valve replacement (TAVR) procedure without return to baseline. Left bundle branch block was noted immediately post-TAVR without improvement over time. **B:** A 6-lead KardiaMobile® ECG monitor (AliveCor, Mountainview, CA) strip during an episode of palpitation showing a wide complex tachycardia at 143 beats/minute.

noted in the anterosuperior interatrial septum in the His region (concentric activation). No evidence of AV block was noted during WCT. Ventricular overdrive pacing entrained the WCT with a post-pacing interval minus tachycardia cycle length difference of 290 milliseconds (Figure 2B). This finding practically ruled out bundle branch reentrant VT as the arrhythmia mechanism.[3] Atrial tachycardia was ruled out on the basis of a V-A-V response to ventricular entrainment (Figure 2B). On account of no baseline evidence of septal accessory pathway, negative septal VA time and post-pacing interval of >115 milliseconds, atrioventricular reentry was also ruled out as the arrhythmia mechanism.

In addition, there was no evidence of ventricular preexcitation with atrial extrastimulation or incremental atrial pacing at baseline. With ventricular entrainment V-A-V rather than V-V-A response was noted, and post-pacing interval minus tachycardia cycle length was >125 milliseconds. Furthermore, there was no resetting or termination of the SVT with spontaneous His-refractory PVCs. These findings taken together ruled out nodofasicular tachycardia as the cause of the SVT.[4]

Diagnosis of typical AVNRT[5] was made by virtue of above observations. After confirmation of AVNRT as the mechanism of the WCT, we targeted the rightward inferior extension of the "AV nodal slow pathway" just anterior

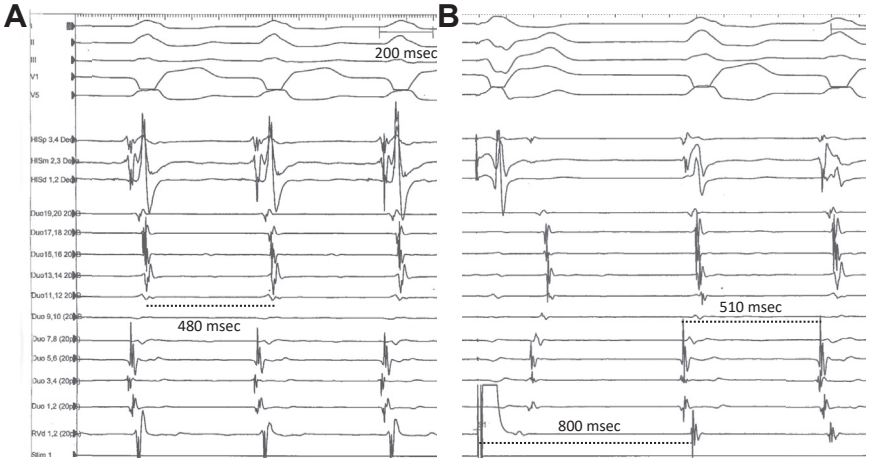


**Figure 2** **A:** A wide complex tachycardia (WCT) induced at a cycle length of 490 milliseconds (480–520 milliseconds fluctuation was noted) with 1:1 atrioventricular (AV) relationship and negative septal VA time of –30 milliseconds. Note the negative septal VA time due to delay in ventricular activation from left bundle branch block. **B:** Ventricular entrainment of the WCT revealed a V-A-V response with a long post-pacing interval minus tachycardia cycle length of approximately 290 milliseconds, ruling out atrial tachycardia, bundle branch reentrant ventricular tachycardia, and AV reentrant tachycardia, and thus confirming the diagnosis of typical atrioventricular nodal reentrant tachycardia.

and inside the coronary sinus ostium with radiofrequency ablations. Slow junctional rhythm was observed during ablation without VA block. Post-ablation, the AV Wenckebach block increased to 620 milliseconds, suggesting ablation of the slow pathway. Post-ablation HV interval remained unchanged and the clinical WCT was noninducible, despite use of isoproterenol up to 10 μg/min. However, an SVT with 1:1 AV relationship of a slower, approximately 600 milliseconds, cycle length could be induced during isoproterenol infusion, which was confirmed as automatic junctional tachycardia with atrial overdrive pacing showing A-H-H-A response. Furthermore, isorhythmic dissociation was noted with competing sinus rhythm during this tachycardia, suggesting it to be isoproterenol-induced automatic junctional tachycardia.[6] The automatic junctional tachycardia abated after stopping isoproterenol infusion. The AH interval shortened from approximately 140 milliseconds to approximately 100 milliseconds post-slow pathway ablation, likely due to an "electrotonic effect." No complications were observed.

The patient was discharged home uneventfully. At approximately 12 weeks follow-up, the patient had complete resolution of his palpitations. His follow-up 12-lead ECG showed sinus rhythm with PR interval of 212 milliseconds and unchanged LBBB, with a QRS duration of 150 milliseconds. Figure 3 shows baseline pre-TAVR and post–slow pathway ablation follow-up 12-lead ECGs.

## Discussion

To the best of our knowledge, this is the first case report of a de novo occurrence of AVNRT after a TAVR. In our patient, the possibility of AVNRT fortuitously occurring for the first time 3 months after TAVR existed, but was overwhelmed by the more plausible explanation that TAVR created a perfect milieu for AVNRT to occur. We hypothesized that there was a causal link between the TAVR procedure and the new onset of AVNRT. There are several clues that point toward this possibility: (1) the temporal relationship between the onset of palpitation—later proven to be due to AVNRT—12 weeks after TAVR procedure without any history of the same; (2) significant PR prolongation post-TAVR, suggesting injury of AV node (and AV nodal extensions as they join the AV node), leading to sluggish AV nodal conduction in the setting of possible preexisting dual AV node physiology; (3) electrophysiology study proving the WCT to be typical AVNRT, with noninducibility and complete resolution of symptoms after ablation of rightward inferior extension of AV nodal slow pathway; and (4) multiple previous reports of spontaneous post-TAVR bundle branch reentrant VT due to His-Purkinje conduction delay in the left bundle,[7,8] which sets a precedent for the possibility of a similar mechanism in the AV node for induction of AVNRT.

Post-TAVR, our patient exhibited LBBB and a substantial and dynamic PR prolongation, which is more than anticipated from an LBBB (Figure 2A). It is difficult to affirmatively conclude whether the PR prolongation after TAVR was a reflection of "unmasking" of a dual AV node physiology, or an injury of the AV nodal pathways (as they join the AV node) with sluggish conduction, or a combination of the 2. It is possible that our patient had preexisting dual AV node physiology. In fact, 10%–35% of the general population is known to have dual AV node physiology,[9] however, AVNRT is much less common, with a prevalence of 22.5/10,000 persons and an incidence of 35/100,000 person-years.[10] This suggests that the mere presence of a dual AV node physiology in isolation may not be sufficient to induce AVNRT. The possibility of differential damage and ensuing sluggish conduction of AV nodal extension (likely slow pathway) as they join the AV node in the setting of a preexisting dual AV node physiology remains an attractive but unproven mechanism of AVNRT in our patient. This arrhythmia mechanism is certainly plausible and in line with a previous observation that ablation involving the interatrial septum or proximal coronary sinus for treatment of persistent AF may create a substrate favorable for AVNRT by altering the atrial or coronary sinus inputs to the AV node.[11] Nevertheless, we acknowledge that the exact underlying mechanism by which de novo AVNRT initiated after TAVR in our patient remain elusive.

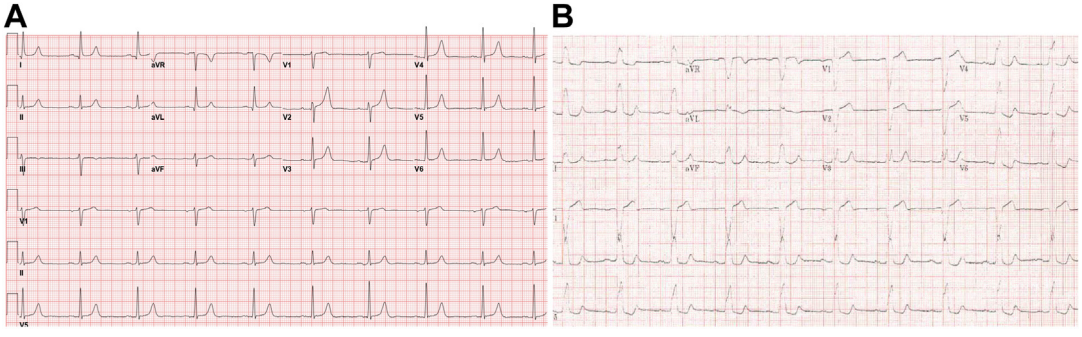


**Figure 3** **A, B:** Pre–transaortic valve replacement (TAVR) and post–slow pathway ablation 80-day follow-up 12-lead electrocardiograms. Note post-TAVR development of left bundle branch block and increase in PR interval from baseline 182 milliseconds to 212 milliseconds.

We also acknowledge that, although less likely, the possibility of automatic junctional tachycardia as the underlying mechanism for the clinical SVT cannot be affirmatively ruled out from our electrophysiology study. Accordingly, the possibility, however small, of an automatic junctional tachycardia focus in the slow pathway region that was successfully ablated still remains.

In contrast to post-TAVR spontaneous de novo occurrence of bundle branch reentrant VT, AVNRT occurrence seems less common. One possible explanation could be that the post-TAVR incidence of new-onset LBB block/delay is much higher (up to 65% with self-expanding valves)[12] compared with isolated PR prolongation (approximately 13%).[1] This variability can be further explained by the anatomy of the AV node and proximal His-Purkinje system and its relationship to the aortic valve.[12] The LB lies between the noncoronary and the right coronary aortic valve cusps, that is, right in the path of transaortic valve implant with a high chance of ensuing injury. However, the AV node is a right atrial structure located in the apex of the triangle of Koch, just across the aortic annulus with its putative extensions and, therefore, has less chance of damage after TAVR.

## Conclusion

To the best of our knowledge, we report the first case of a de novo occurrence of AVNRT after TAVR procedure with a plausible causal link between the 2. We hypothesize that TAVR caused AV nodal injury and ensuing differential sluggish conduction over its slow–fast pathway connections creating the fertile milieu for AVNRT to occur. Furthermore, AV nodal slow-pathway ablation seems to be an effective treatment for post–TAVR de novo AV nodal reentrant tachycardia.

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