Catheter cryoablation guided by propagation mapping to treat dual atrioventricular nodal nonreentrant tachycardia in an adult with postoperative congenital heart disease

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Introduction

Dual atrioventricular node nonreentrant tachycardia (DAVNNRT) is an uncommon and often misdiagnosed arrhythmia reported in patients without congenital heart disease (CHD), leaving some patients with troublesome palpitations while causing tachycardia-induced cardiomyopathy in others.[1,2] DAVNNRT results from unusual downstream conduction of consecutive sinus beats via multiple pathways into the atrioventricular (AV) node; each sinus impulse first penetrates the AV node via the "fast pathway" (FP), followed by an impulse from the same sinus beat conducting via the "slow pathway" (SP).[1,2] Consequently, electrocardiograms (ECGs) during DAVNNRT show tachycardia with QRS complexes similar to sinus rhythm, usually in a regularly irregular pattern that mimics other arrhythmias, including premature extrasystoles and atrial fibrillation.[1,2]

Medical therapy usually does not suppress DAVNNRT, but patients have been treated successfully with radiofrequency ablation (RFA).[1–3] Herein we describe an adult with complex postoperative CHD who developed frequent palpitations from DAVNNRT during sinus and atrial-paced rhythms, and in whom 3-D electrophysiology propagation mapping guided successful catheter cryoablation.

Case report

A 31-year-old male patient underwent surgical repair of a secundum atrial septal defect at 4 years of age, unexpectedly complicated by early postoperative right heart failure. Over the next 10 years, he developed progressive right heart dilation, exercise intolerance, and unexplained syncope; atrial and ventricular tachyarrhythmias were incompletely suppressed with antiarrhythmic agents. At 14 years old, he underwent extensive cardiac surgery including right atrial reduction, resection of a large right ventricular aneurysm, implantation of a #32 Carpentier-Edwards tricuspid annuloplasty ring, and 1 ½-ventricle palliation including a bidirectional Glenn anastomosis. Simultaneously, a right atrial maze procedure was performed and an epicardial biventricular implantable cardioverter-defibrillator (ICD) implanted. Postoperatively, AV conduction remained intact and sinus bradycardia was managed with atrial pacing. Of note, neither tissue pathology nor genetic testing supported the diagnosis of arrhythmogenic right ventricular cardiomyopathy. He otherwise did well on sotalol until he was 29 years old, when the epicardial ICD coil fractured. To avoid repeat chest surgery, he underwent implantation of a single-chamber transvenous ICD after transcatheter placement of a covered stent connecting the Glenn anastomosis to the RA. Diffuse low-amplitude electrograms prevented adding an atrial lead. The new transvenous ICD was programmed to treat significant ventricular tachycardia (VT), and the original epicardial ICD was used for AAIR pacing only.

At 31 years old, he developed progressively frequent palpitations. Device interrogations and ECGs revealed predominantly atrial-paced rhythm with intact AV

KEY TEACHING POINTS

- Dual atrioventricular node nonreentrant tachycardia is a rare tachyarrhythmia that mimics other arrhythmias and can manifest in adults with postoperative congenital heart disease who are chronically atrially paced.
- Three-dimensional wavefront propagation mapping in these patients can be useful to guide patients' optimal ablation strategy, which may involve a broader target than when treating patients with typical atrioventricular node reentry tachycardia.
- Catheter cryoablation can be a useful alternative to radiofrequency ablation in these patients.

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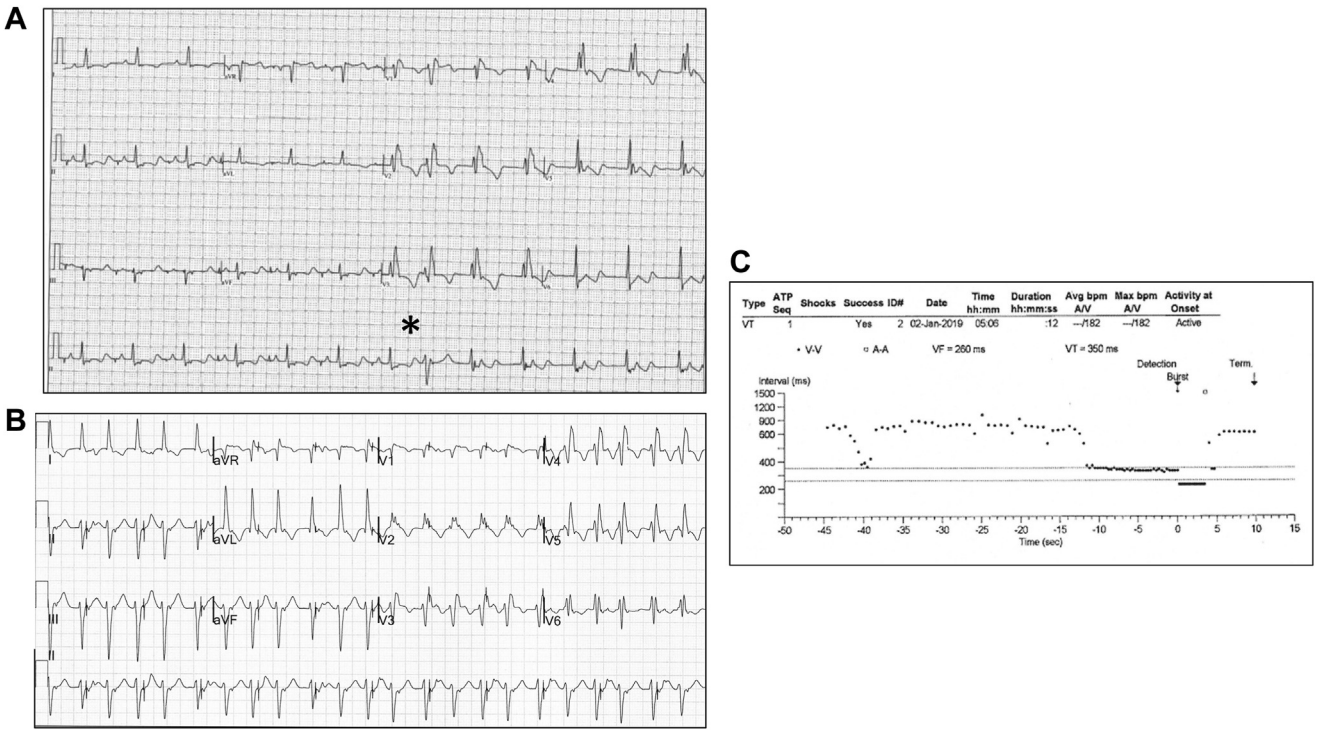

Figure 1 **A:** Electrocardiogram (ECG) during atrial paced rhythm (70 beats/min) with abrupt PR prolongation. After 6 beats with a PR interval = 250 ms, the seventh beat (*) is followed by a premature QRS complex of slightly different morphology. Subsequent beats have markedly prolonged PR intervals (700 ms). **B:** ECG of regularly irregular tachycardia with right bundle branch block pattern during atrial pacing at 70 beats/min, reflecting persistent 1:2 atrioventricular conduction with PR intervals of 260 ms and 760 ms. **C:** Interrogation of the single-chamber implantable cardioverter-defibrillator showed abrupt onset of tachycardia with cycle length ~50% of preceding rhythm, triggering antitachycardia pacing therapy.

conduction and mildly prolonged PR intervals (220–240 ms) abruptly alternating with markedly prolonged PR intervals (~750 ms), often triggered by premature extrasystoles (Figure 1A). In addition, ECGs of paroxysmal supraventricular tachycardia with regularly irregular R-R intervals were recorded (Figure 1B). The transvenous ICD sometimes detected episodes of slightly faster VT and delivered antitachycardia pacing (Figure 1C); no shocks were delivered. Recorded electrograms of "VT" were similar to normally conducted atrial paced beats, but at a rate roughly twice the prevailing atrial rhythm. The epicardial device did not record atrial tachycardia at these times. Altogether, these findings suggested DAVNNRT due to 1:2 AV conduction of sinus and atrial paced rhythms. Adjusting the lower pacing rate[3] and sotalol dose and adding a beta blocker had no impact. He was referred for electrophysiology study (EPS) and catheter ablation.

At EPS, VT therapies on the transvenous ICD were disengaged and the epicardial device programmed AAI 50 beats per minute (bpm). A fluoroless procedure was performed under general anesthesia via 2 sheaths in the right femoral vein. A Biosense Webster EZ Steer™ catheter and CARTO 3™ system (Biosense Webster, Irvine, CA) were used to generate 3-D geometries of the inferior vena cava, RA, and coronary sinus (CS). Baseline AP-QRS interval was ~245 ms and QRS complexes had a stable right bundle branch block pattern. A hexapolar EP catheter was used for atrial pacing protocols and temporal reference during EP mapping. Endocardial recordings showed diffuse low-amplitude electrograms throughout the RA, with small islands of higher voltages (Figure 2A). Most electrograms recorded in the septal RA and triangle of Koch (TOK) were fractionated with very low amplitudes. Baseline AH interval was equal to 110 ms and HV interval was equal to 50 ms; steady His bundle electrograms proved challenging to record during pacing protocols. EPS also showed AVB-CL = 450 ms, PR>RR was not encountered, discrete AP-QRS jump occurred at 600/370 ms, AV nodal effective refractory period <330 ms, and atrial effective refractory period = 330 ms. With atrial pacing at 70 bpm, the PR interval intermittently jumped abruptly from 260 ms to 780 ms immediately following single beats of probable 1:2 AV conduction, similar to outpatient ECGs (Figure 1A). Although 1:2 AV conduction was reproducible, neither DAVNNRT nor AV node reentry tachycardia (AVNRT) was inducible at baseline or on isoproterenol (maximum 10 mcg/min).

A multipolar PENTARAY™ catheter (Biosense Webster) was used for detailed EP mapping during continuous high RA pacing at 70 bpm; only beats with stable PR intervals (260 ms) were included. Diffuse low-amplitude fractionated electrograms throughout the TOK limited utility of "classic" SP potentials, and discrete low-voltage bridges[4] were not apparent. Propagation mapping showed the initial impulse advancing into the RA septum and superior TOK from the high RA, likely reflecting conduction via the FP (Figure 2B, Supplemental Video). From there, the impulse

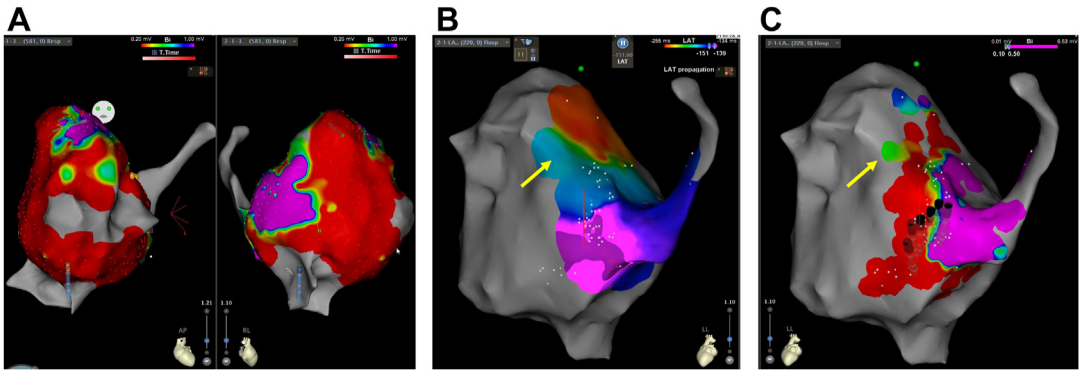

Figure 2 Three-dimensional electrophysiology mapping in the right atrium during high right atrial pacing at 70 beats/min with the stable PR interval (260 ms). **A:** Voltage map demonstrating diffuse low-amplitude electrograms (red) throughout the right atrium (anteroposterior and right lateral views). **B:** Snapshot of the propagation map (left lateral view), showing the initial superior wavefront enter the septal right atrium (RA) (red), then course down into the triangle of Koch to collide (light purple) in the mid-low triangle with delayed wavefronts entering from inferior RA and coronary sinus (CS). See accompanying Supplemental Video. **C:** Linear cryoablation applications from the tricuspid annulus to CS os displayed on voltage map showing low-amplitude electrograms (red) diffusely in the triangle of Koch. Higher voltages were recorded within the proximal CS. Yellow arrow = site of HB recording.

swept inferiorly into the low/mid TOK, colliding with delayed wavefronts entering there from more inferior and posterior positions, including the CS. Wavefront collision extended from the tricuspid valve annulus (TVA) to the mid CS os (Figure 2B).

RFA (40 W, 50°C) using the EZ Steer catheter at the collision zone near the TVA did not eliminate 1:2 AV conduction. During 1 application, very brief 2nd-degree AVB was recorded, and RFA was discontinued immediately. Paced PR intervals remained stable and 1:2 AV conduction was still inducible. After exchanging the RF catheter for a 6 mm cryocatheter (CryoCath™; Medtronic, Montreal, Canada), cryoablation applications (-75°C, 4 minutes) were delivered linearly from the TVA to the CS os, transecting the broad area of wavefront collision (Figure 2C); 1:2 AV conduction remained inducible until this line was completed. Thereafter, AV conduction was intact, with stable baseline PR intervals, and dual AV node physiology and 1:2 AV conduction remained absent during EP testing for an hour. Serial ECGs and device interrogations recorded no marked PR prolongation or DAVNNRT over 1 year of follow-up on sotalol.

Discussion

DAVNNRT is a rare tachyarrhythmia caused by unusual antegrade AV conduction of sinus beats, each impulse first penetrating the AV node via the FP, followed by a delayed impulse via the SP also conducting into the AV node.[1,2] When both impulses make it through the AV node distal common pathway, the result is 1:2 or "double-fire" ventricular depolarizations.[1,2] ECGs in affected individuals show variable patterns, including sinus beats conducting 1:1 or 1:2 with either stable or slightly variable short and prolonged PR intervals; some impulses conduct aberrantly.[1,2] Not surprisingly, this arrhythmia is often misdiagnosed as something more common to clinical practice, like premature extrasystoles and atrial fibrillation.[1,2] Most DAVNNRT reports involve adults with congenitally normal hearts; a single case of a child with postoperative ventricular septal defect has been reported.[5] We are not aware of other reports of adult CHD patients treated for DAVNNRT. Whereas DAVNNRT occurs during sinus rhythm in most patients, there has been at least 1 other atrially paced patient[3]; interestingly, DAVNNRT was suppressed by increasing the lower paced rate. In general, DAVNNRT responds poorly to medications alone. Our patient's arrhythmia was not suppressed by adjusting the paced rate or antiarrhythmic medications. Fortunately, most patients can be managed with radiofrequency ablation; besides our patient, there is only 1 other reported treatment success using catheter cryoablation.[6]

Similar to the relatively common AVNRT, prior work has shown DAVNNRT involves dual AV node physiology, but with a twist.[1–3] ECGs in affected patients often manifest spontaneous dual AV node physiology, with PR intervals during sinus or atrial-paced rhythm lengthening abruptly (Figure 1A). These changes are often accompanied by premature extrasystoles. In contrast to AVNRT, however, not only must the disparate conduction times of the FP and SP be longer than the refractory period of the AV node, but the prevailing sinus rates must also be just right to facilitate 1:2 AV conduction.[1–3] More specifically, the timing of when a slow impulse via the SP reaches the AV node must be after impulse arrival via the FP by a period longer than the effective refractory period of the AV node distal common pathway for that second impulse to conduct downstream. These findings were observed in our patient, as the PR intervals via FP and SP conduction differed by 520 ms and his AV nodal effective refractory period was <330 ms.

Three-dimensional EP propagation mapping was used in our patient with diffuse low-amplitude atrial electrograms to help visualize wavefront propagation patterns in the TOK and guide catheter ablation. Propagation mapping during 1:1 AV conduction via the FP confirmed only that while 1 component of the superior FP impulse penetrated the AV node, an adjacent component continued inferiorly, colliding in the mid/low TOK with the delayed impulses conducting up from

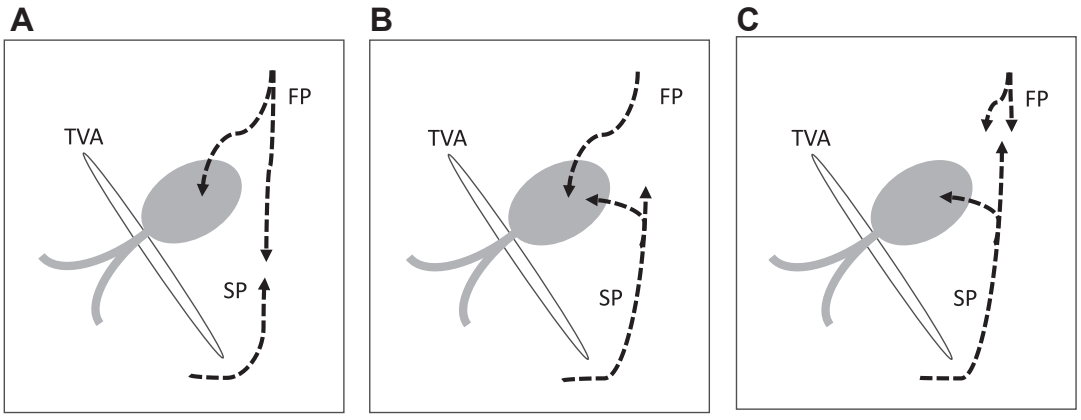

Figure 3 1:1 and 1:2 atrioventricular (AV) conduction patterns in a patient with dual AV node nonreentrant tachycardia (left lateral view). **A:** 1:1 AV conduction with shorter PR intervals result from atrial-paced impulses propagating into the AV node via the fast pathway (FP), with an adjacent component propagating into the triangle of Koch to collide with delayed inferior impulses coursing superiorly via the slow pathway (SP) and/or coronary sinus musculature. **B:** 1:2 AV conduction as the superior FP impulse first penetrates the AV node but does not course into the triangle, allowing the delayed inferior wavefronts to slowly propagate superiorly into the AV node. **C:** 1:1 AV conduction with prolonged PR intervals after 1:2 AV conduction owing to the delayed inferior wavefront continuing to propagate up into the high septal right atrium to collide with subsequent FP wavefronts. TVA = tricuspid valve annulus.

the inferior septum and CS musculature (Figures 2B and 3A, and Supplemental Video). In patients with normal hearts and AVNRT, similar but more discrete wavefront collision is often recorded during sinus rhythm with short PR intervals,[7,8] likely reflecting localized conduction slowing within the SP proper. In contrast, the collision zone in our patient was quite broad, extending from the TVA to the CS os (Figure 2B, Supplemental Video); we suspected his underlying diffuse atrial pathology may have contributed to this propagation pattern. This broad area of collision appears to block all components of the delayed inferior impulses from conducting up toward the AV node, preventing 1:2 AV conduction. It is therefore not surprising that cryoablation transecting this broad zone was required for treatment success in our patient.

Although we were unable to map 1:2 AV conduction or 1:1 conduction via SP only, the diagram of 1:1 conduction via the FP only (Figure 3A) provides an opportunity to depict those propagation patterns (Figure 3B and 3C) as a simple alternative to classic ladder diagrams.[1,2] The ECG in Figure 1A shows an abrupt shift from shorter paced PR intervals (via the FP) to much longer PR intervals (via the SP) immediately after 1:2 AV conduction. In this case, the double-fire beat could have resulted if no part of the preceding FP impulse had coursed inferiorly into the TOK (Figure 3B), perhaps from dynamic changes in local tissue conduction velocities or refractory periods. Without wavefront collision within the TOK from this FP impulse, the delayed inferior impulse could then slowly propagate up to penetrate the now repolarized AVN, resulting in the second ventricular depolarization of the double-fire beat. Simultaneously, a component of this delayed impulse could continue to conduct slowly up into the now repolarized septal right atrium, ultimately colliding with the next atrial-paced impulse propagating septally toward the FP. This collision would in turn prevent FP conduction into the AV node (Figure 3C), resulting in SP conduction only with prolonged PR intervals. If this pattern occurred repetitively, the result would be sustained, consecutive paced beats with markedly prolonged PR intervals (Figure 1A).

Conclusion

DAVNNRT is a rare tachyarrhythmia that mimics other arrhythmias and can occur in adult patients with postoperative CHD, including those with chronic atrial pacing. Wavefront propagation mapping can provide insights into relevant propagation patterns and thereby guide successful ablation therapy, even using cryoablation. Successful ablation for DAVNNRT in these patients may require more extensive applications than simply targeting the SP as in patients with AVNRT.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.02.010.

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