CASE REPORT

Dual Atrioventricular Nodal Nonreentrant Tachycardia with Alternating 1:1 and 1:2 AV Conduction: Mechanistic Hypotheses and Total Suppression Using Right Atrial Pacing

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Dual atrioventricular (AV) nodal nonreentrant tachycardia is an uncommon arrhythmia with several pattern types. The primary therapy is ablation of the slow AV nodal pathway. A rare pattern type demonstrates alternating 1:2 and 1:1 AV ratios with longer PR intervals during 1:1 conduction. We report the second intracardiac study of this variant and the first case of using right atrial pacing as the ultimate therapy for any pattern type.

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Dual atrioventricular nodal nonreentrant tachycardia (DAVNNT) is an uncommon arrhythmia with several recognized electrocardiogram (ECG) pattern types.¹ In pattern type 2, sinus beats conduct with alternating 1:2 and 1:1 atrioventricular (AV) ratios with longer PR intervals during 1:1 conduction. This has been reported three times and an intracardiac study during tachycardia only once by Barbato et al.^{2–4} We present the second intracardiac study and also the first report of successful long-term treatment of DAVNNT of any pattern type with right atrial pacing at a slightly augmented rate. Repeat slow pathway ablation, which would have carried a significant risk for complete AV block in this patient, was able to be avoided.

CASE REPORT

An 84-year-old man with hypertension and coronary artery disease noted progressive tiredness

on exertion. His left ventricular ejection fraction (LVEF) had decreased from 40% to 20%–25%. Despite percutaneous coronary intervention of a left circumflex artery stenosis and optimization of heart failure medications, the LVEF remained 30%–35% three months later. Medications included metoprolol, lisinopril, aspirin, clopidogrel, amlodipine, and simvastatin. He was referred for an implantable cardioverter-defibrillator (ICD). On further questioning, he mentioned “skipping beats” for over 10 years. Two years before presentation, a Holter monitor documented that 2.8% of his QRS complexes were premature ventricular complexes (PVCs) and 18.5% were premature atrial complexes (PACs).

The current ECG (Fig. 1) was originally interpreted as sinus rhythm with PVCs and PACs. Sinus P waves were seen at a rate of 55 beats per minute (bpm). The first wide QRS beat (third QRS complex) was a PVC as it occurred when the next

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Figure 1. The 12-lead ECG with rhythm strips from leads V1, II, and V₅ are shown.

Figure 2. Surface leads I, II, III, V1, and V₆ are displayed with electrograms from the right atrium (RA), His bundle (His), proximal coronary sinus (CSp), distal coronary sinus (CSd), and right ventricle (RV). The AH intervals to the first His electrograms (upper numbers and arrows) and to the second His electrograms (lower numbers and arrows) are measured.

Figure 3. ECG lead V₁ rhythm strips four months after slow pathway ablation demonstrated (A) DAVNNT pattern type 1, (B) DAVNNT pattern type 2, (C) intermittent dual AV nodal response with right atrial pacing at 60 bpm, and (D) total suppression of dual AV nodal response with right atrial pacing at 70 bpm.

P wave was expected to be. The second wide QRS beat (sixth QRS complex) was a fusion beat. Group beating was present with alternating 1:2 and 1:1 relationships between P waves and QRS complexes. The PR interval was 230 ms preceding 1:2 AV association and 260 ms preceding 1:1 AV association. There was no distinct nonsinus atrial activity preceding the second QRS complex in each group of three.

The patient was brought to the electrophysiology laboratory in the fasting, nonsedated state. A sample intracardiac recording (Fig. 2) demonstrated sinus cycle lengths (CLs) of 1120–1180 ms with alternating 1:2 and 1:1 AV association. During 1:2 AV association, the short AH intervals were 135–145 ms and the long AH interval were 595–635 ms. During 1:1 AV association, the AH intervals were 160–175 ms. The HV intervals were constant at 77 ms. Incremental right atrial pacing using the stepwise decremental technique demonstrated only 1:1 AV conduction via the fast pathway at CLs from 800 to 610 ms, which was the fast pathway AV block CL. The AH interval with atrial pacing at a CL of 800 ms was 150 ms. The fast pathway effective refractory period (ERP) was 450 ms with a pacing CL of 800 ms. There was no evidence

of dual AV nodal physiology during programmed atrial stimulation with pacing CLs as long as 800 ms. Isoproterenol administration decreased the sinus CL to 880 ms at 1 mcg/min and 770 ms at 2 mcg/min. At both doses, there was only 1:1 AV conduction via the fast pathway. Ventriculoatrial (VA) conduction was absent at baseline, but present with isoproterenol. These findings supported a diagnosis of DAVNNT pattern type 2.

Cryoablation of the slow pathway was attempted without any effect. Radiofrequency catheter ablation was then performed using the anatomic technique. There was no effect after multiple test lesions in the posterior, medial, and anterior locations of the AV junction. Intracardiac echocardiography during later lesions ensured adequate tissue contact. A final lesion delivered in the superior aspect of the anterior region resulted in junctional beats without retrograde conduction to the atrium. Despite cessation of ablation, persistent complete heart block with a slow junctional escape rhythm ensued and a dual-chamber ICD was inserted. Coronary sinus lead placement was unsuccessful. The next morning, programmed atrial stimulation through the ICD demonstrated return of fast pathway conduction and no evidence of dual

Figure 4. ECG lead V₁ rhythm strip and ladder diagrams demonstrate potential mechanisms. The ERP of the AV node distal common pathway or His-Purkinje system is represented by the shaded rectangles. See text for discussion.

AV nodal physiology. The Wenckebach CL was 460 ms and the fast pathway ERP was <380 ms with a pacing CL of 700 ms.

His symptoms resolved and his LVEF three months after ablation was 35%–40%. Unfortunately, he had symptom recurrence one month later and his ECG was consistent with DAVNNT pattern type 1 (Fig. 3A), with continuous 1:2 AV conduction. A 24-hour Holter monitor demonstrated 47,605 (43.1%) "supraventricular ectopic beats" which were actually dual AV nodal response beats. He was brought back to clinic one week later and his ECG demonstrated DAVNNT pattern type 2 (Fig. 3B). The coexistence of pattern types 1 and 2 in the same patient has been described twice.^{2,3} This was likely due to varying degrees of concealed retrograde conduction under different physiologic states.

His backup pacing mode and lower rate limit, previously VVI-40 bpm, was set to DDD-60 bpm (Fig. 3C) and then DDD-70 bpm (Fig. 3D) to eliminate slow pathway conduction. The AV delay was increased to 300 ms to allow intrinsic conduc-

tion. A 24-hour Holter monitor two weeks later demonstrated zero (0.0%) "supraventricular ectopic beats." His symptoms had resolved. His LVEF four months after pacing mode adjustment was 40%–45%. This LVEF recovery suggested a component of tachycardia-induced cardiomyopathy.

DISCUSSION

The differential diagnosis of the surface ECG consisted of PACs, junctional complexes and DAVNNT pattern type 2. The lack of discernible nonsinus atrial activity was confirmed in the intracardiac study to rule out PACs. Junctional complexes with retrograde block were felt to be unlikely as the AH intervals during 1:1 AV association were consistently longer than the first AH intervals during 1:2 AV association, which suggested AV nodal dependence. Absence of baseline VA conduction and arrhythmia suppression with isoproterenol supported DAVNNT.^{1,5} Isoproterenol would have been expected to increase junctional activity.

Similar to the previously published intracardiac case,⁴ our patient had longer fast pathway AH intervals during 1:1 AV conduction compared to during 1:2 AV conduction. Prolongation of fast pathway AH intervals due to Wenckebach phenomenon during DAVNNT was unlikely because the fast pathway AV block CL was 610 ms. The most likely explanation was retrograde concealed penetration into the fast pathway (Fig. 4).

We considered two mechanistic hypotheses. First (Fig. 4, upper ladder diagram), prolongation of fast pathway conduction due to concealed retrograde conduction may have resulted in shortening of the difference between fast and slow pathway conduction to less than the ERP of the AV node distal common pathway or His-Purkinje system. The level of block during the intracardiac study was at the proximal His bundle or above. Second (Fig. 4, lower ladder diagram), concealed retrograde conduction with partial penetration may have occurred in the slow pathway. Following a short fast pathway AH interval, retrograde penetration was not great enough to block antegrade slow pathway conduction. An increase in the fast pathway AH interval shifted the relative timing of retrograde and antegrade slow pathway conduction, leading to wavefront collision.

Slow pathway ablation is the main therapy for all DAVNNT pattern types,¹ but carries a risk of AV block. Pacemaker implantation with AV junction ablation has been described for exceptional cases.¹ In our patient, 100% right ventricular pacing would have increased the risk for subsequent heart fail-

ure. The mode we selected resulted in only 10% right ventricular pacing. Atrial pacing with a lower rate limit of 70 bpm is safe in patients with reduced LVEF.⁶

The AH interval prolongation at faster atrial pacing rates may decrease the difference between fast and slow pathway conduction to less than the ERP of the AV node distal common pathway or His-Purkinje system. Alternatively, it may result in collision of retrograde and antegrade conduction in the slow pathway due to a shift in the timing of retrograde concealed conduction. This is the first report of successful long-term treatment of DAVNNT of any pattern type using right atrial pacing at a slightly augmented rate.

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