Incessant typical atrioventricular nodal reentrant tachycardia with prolonged PR interval during sinus rhythm: Case report and literature review

Norman C. Wang, MD, MS, FHRS

From the Division of Cardiology, Department of Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania.

Introduction

Current guidelines from major American and European medical societies recommend the slow pathway as the catheter ablation target for all forms of atrioventricular nodal reentrant tachycardia (AVNRT).[1,2] Nevertheless, some operators still advocate targeting the retrograde fast pathway for typical AVNRT when anterograde fast pathway conduction is suspected to be compromised or absent.[3,4] Incessant typical AVNRT is a rare entity that has been theorized to result from "sole anterograde conduction over the [slow pathway] in [sinus rhythm] leaving the fast pathway available for initiation of typical AVNRT."[5] Herein, I present a case of successful slow pathway ablation in a patient with incessant typical AVNRT associated with a prolonged PR interval during sinus rhythm and a continuous anterograde atrioventricular (AV) node function curve. A literature review was performed.

KEY TEACHING POINTS

- Catheter ablation of the slow pathway is a viable treatment for incessant typical atrioventricular nodal reentrant tachycardia (AVNRT) with a prolonged PR interval during sinus rhythm and a continuous anterograde atrioventricular (AV) node function curve.
- There are no consensus criteria within the cardiac electrophysiology community to define incessant, as opposed to paroxysmal, AVNRT.
- AV node function curves may offer insight into potential arrhythmia mechanisms.
- Initiation mechanisms for incessant AVNRT, such as changes in sinus rate and/or premature atrial complexes with varying coupling intervals, may have important therapeutic implications.
- Cardiac magnetic resonance imaging may diagnose underlying idiopathic nonischemic cardiomyopathy in patients with associated reduced left ventricular ejection fraction.

Case report

In November 2022, an 89-year-old man with hypertension treated with metoprolol succinate 50 mg daily, seronegative rheumatoid arthritis treated with methotrexate, and follicular lymphoma treated with rituximab complained to his primary care physician of weakness and shortness of breath on mild exertion for 1 week. He also noted mild symptoms of heart rate elevation, palpitations, and lightheadedness. He previously walked a 0.9-mile route daily, but recently could not complete it. He had "a general knowledge that things weren't right" and his heart rate was approximately 140 beats/min.

In the emergency department, the presenting electrocardiogram (ECG) demonstrated a short R-P supraventricular tachycardia (SVT) at 147 beats/min (Figure 1A). Intravenous diltiazem 20 mg terminated the arrhythmia, but it immediately resumed. An echocardiogram demonstrated a reduced left ventricular ejection fraction (LVEF) of 10%–15% with a left ventricular end-diastolic diameter of 5.2 cm. He had no prior cardiac testing. Catheter ablation was planned. Between presenting to the emergency department and entering the electrophysiology laboratory, he had nearly continuous SVT for 22.4 hours, with episodic spontaneous termination and instantaneous reinitiation following premature atrial complexes (PACs) (Figure 1B). An ECG in sinus rhythm could not be obtained, but the PR interval was estimated to be 250 ms.

In the electrophysiology laboratory, a 4-mm-tip nonirrigated NAVISTAR radiofrequency ablation catheter with a VIZIGO sheath and a CARTO 3 electroanatomic mapping

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Address reprint requests and correspondence: Dr Norman C. Wang, UPMC Presbyterian, 200 Lothrop St, South Tower, 3rd Floor, Room E352.9, Pittsburgh, PA 15213. E-mail address: ncw18@pitt.edu.

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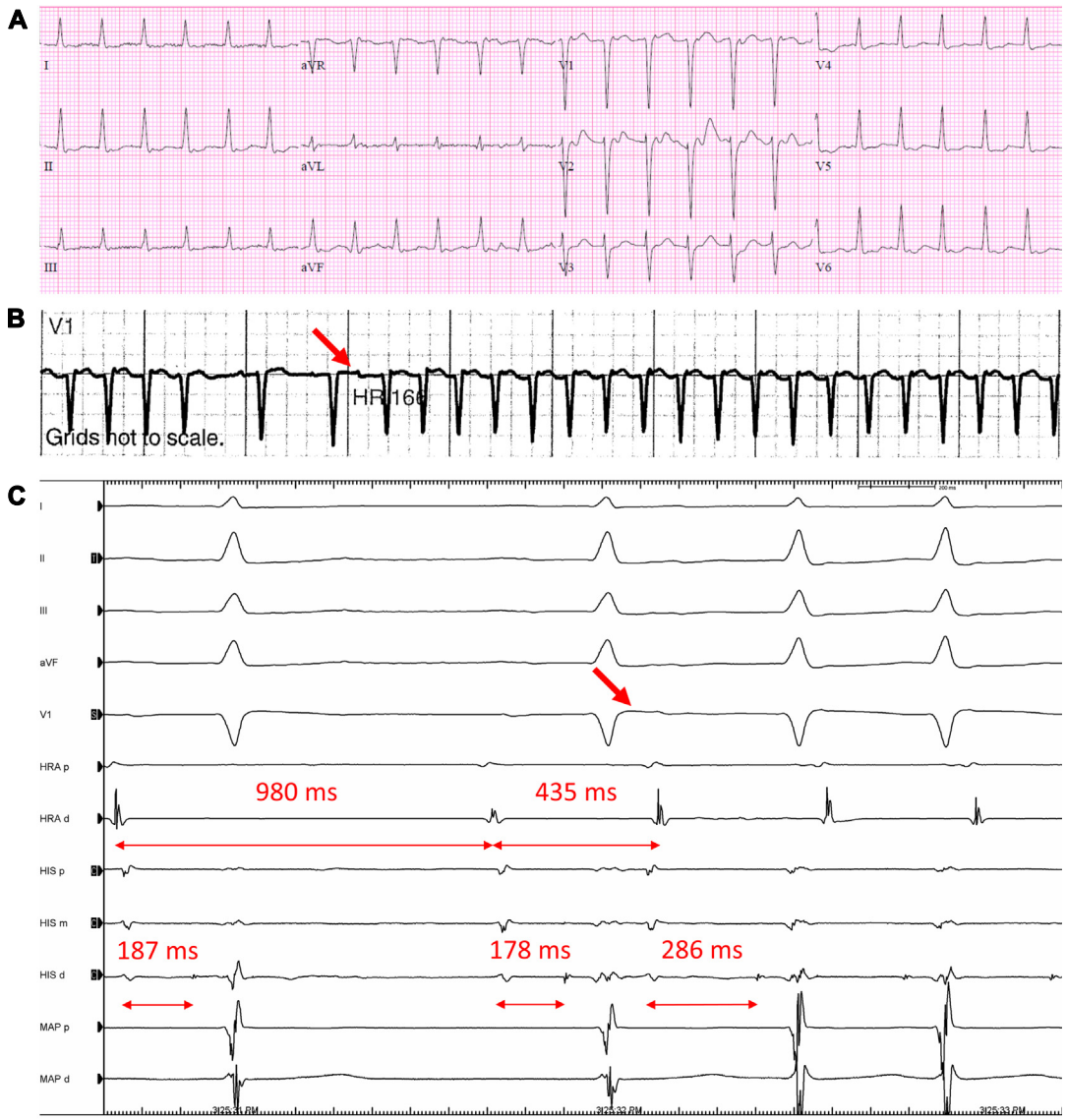

Figure 1 **A:** Initial 12-lead electrocardiogram. **B:** Lead V_1 telemetry strip depicted supraventricular tachycardia (SVT) termination followed by a premature atrial complex (*red arrow*) associated with PR interval prolongation, SVT initiation, and appearance of pseudo r′ waves in subsequent QRS complexes. **C:** Intracardiac recording of SVT initiation after a spontaneous premature atrial complex (*red arrow*). The ablation catheter (MAP) is in the right ventricle. Recording speed 100 mm/s.

system (Biosense Webster, Irvine, CA) were used to reconstruct the right atrium and coronary sinus. Diagnostic catheters were placed in the high right atrium and His bundle region. The ablation catheter was placed in the right ventricle. A decapolar catheter was inserted into the coronary sinus and ventricular pacing demonstrated concentric atrial activation, but it was later removed owing to instability.

SVT easily terminated with ventricular pacing but repeatedly initiated with PACs (Figure 1C). The SVT had a cycle length of 379 ms, an AH interval of 314 ms, an HA interval of 65 ms, and a septal VA time of 0 ms. Baseline sinus rhythm measurements included a PR interval of 254 ms, a QRS duration of 99 ms, a QT interval of 429 ms, an AH interval of 142 ms, and an HV interval of 61 ms. The anterograde AV node Wenckebach cycle length was 370 ms. The anterograde AV node function curve was continuous (jump absent), but the retrograde curve was discontinuous (jump of 55 ms present) (Figure 2A and 2B). A jump was defined as an increase of 50 ms or greater in the A_2H_2 interval or the V_2A_2 interval for a 10 ms decrease in atrial or ventricular, respectively, extrastimuli coupling intervals during programmed stimulation.

Spontaneous SVT became less frequent with sedation. Intravenous isoproterenol was started and titrated to 3 mcg/min. SVT was induced with programmed atrial stimulation. Atrial overdrive pacing resulted in an A-H-A response (Supplemental Figure 1). Ventricular overdrive pacing was unsuccessful as SVT terminated, but there was no effect on atrial timing within the transition zone.[6] These findings, in conjunction with AH and HA measurements, supported a diagnosis of typical AVNRT.[7] Mapping of the retrograde fast pathway during AVNRT was attempted but was not

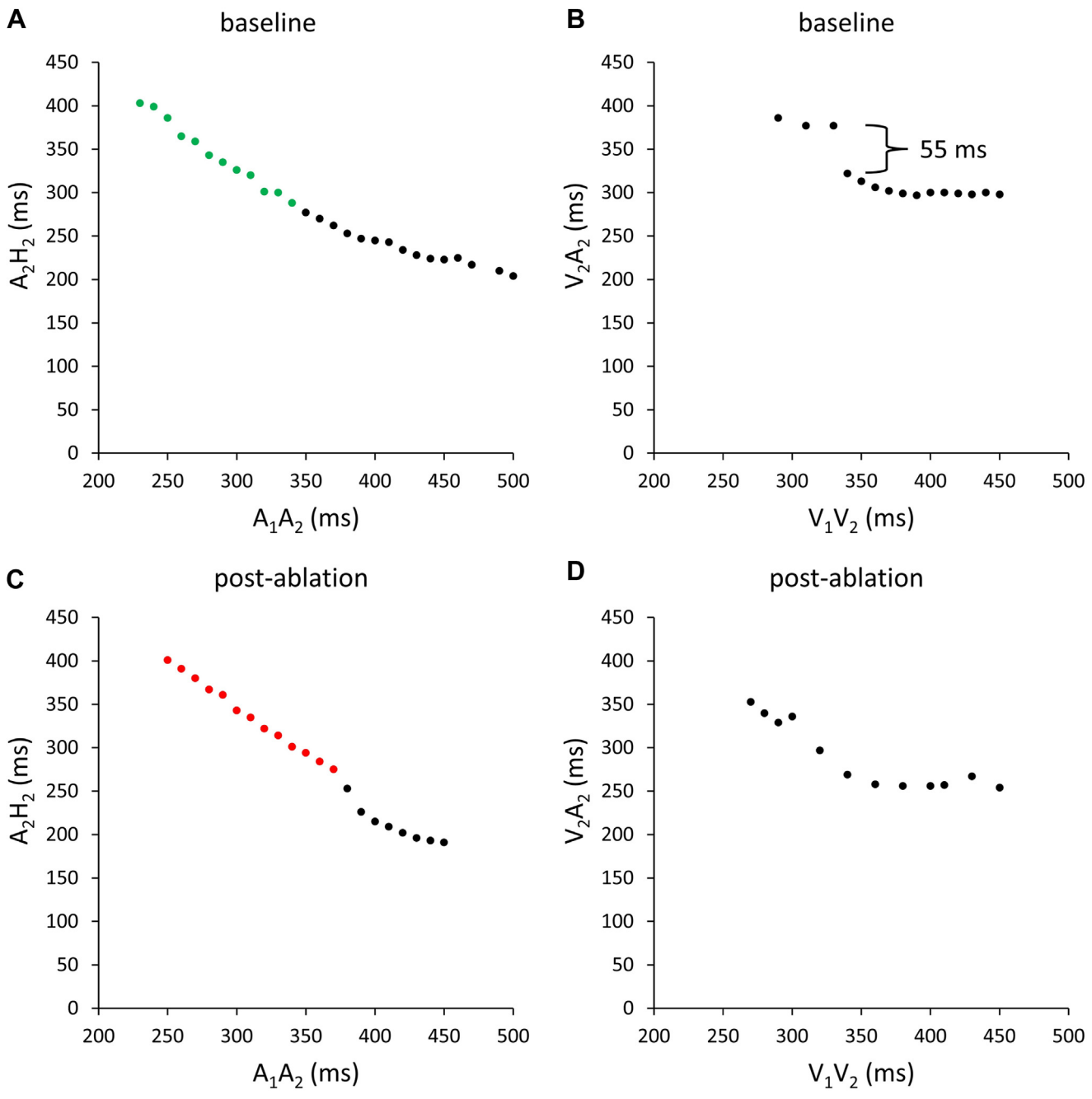

Figure 2 **A:** Baseline anterograde atrioventricular (AV) node function curve. Stimulation sequences associated with induction of nonsustained supraventricular tachycardia are indicated in green. **B:** Baseline retrograde AV node function curve with a "jump" when V_1V_2 was decreased from 340 ms to 330 ms. **C:** Post-ablation anterograde AV node function curve. Stimulation sequences associated with AV node echo beats are indicated in red. **D:** Postablation retrograde AV node function curve.

completed owing to patient agitation. Isoproterenol was stopped.

Slow pathway ablation was performed in temperature-controlled mode with a target of 55°C at powers of 30–50 W for up to 60 seconds per lesion. The successful lesion, the 13th overall, was associated with an AV electrogram amplitude ratio of 2.2 (Figure 3). Junctional beats were elicited (Supplemental Figure 2). No additional lesions were delivered. The postablation anterograde AV node Wenckebach cycle length was 510 ms. Postablation AV node function curves, obtained 27 to 34 minutes after isoproterenol cessation, were continuous (Figure 2C and 2D). AVNRT was unable to be induced without and with isoproterenol 2 mcg/min.

The next morning, an ECG demonstrated sinus rhythm at 80 beats/min with a PR interval of 254 ms and a QRS duration of 72 ms. The patient was discharged with a wearable cardioverter-defibrillator (WCD) (ZOLL LifeVest, Pittsburgh, PA).

In the postdischarge period, guideline-directed medical therapy for heart failure with reduced LVEF was initiated. His optimized regimen included carvedilol 3.125 mg twice

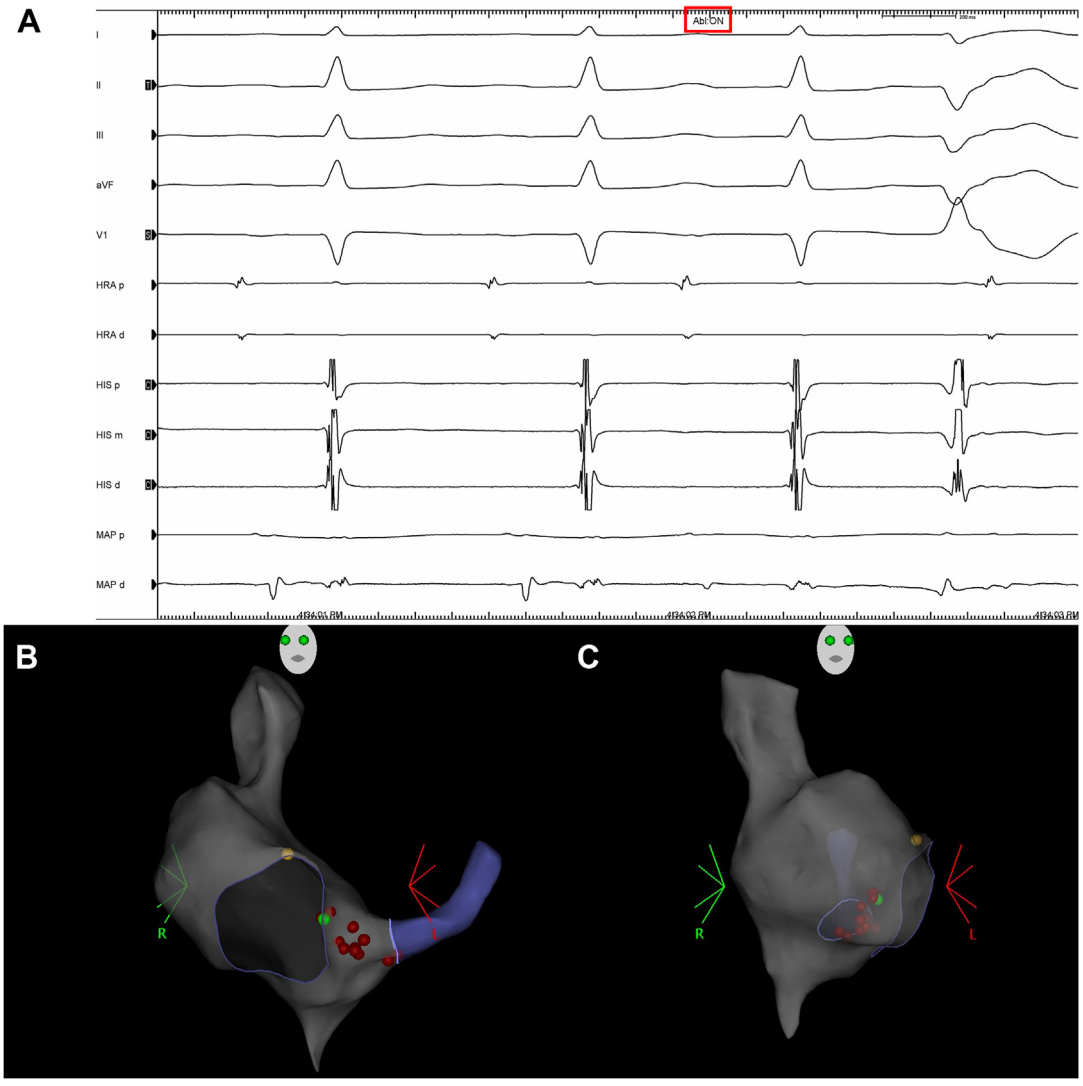

Figure 3 Slow pathway ablation. **A:** Onset of the effective lesion associated with junctional beats is indicated by "Abl: ON" (*boxed in red*). The His bundle catheter (HIS) was displaced into the ventricle. Recording speed 100 mm/s. **B:** Electroanatomic map (EAM) in the left anterior oblique projection. **C:** EAM in the right anterior oblique projection. In the EAMs, colors differentiate the right atrium (*gray*) and coronary sinus (*blue*). Points indicate the His bundle recording (*yellow*), ineffective ablation lesions (*red*), and the effective ablation lesion (*green*).

daily and sacubitril-valsartan 24–26 mg twice daily. Spironolactone 25 mg daily was started but was stopped after 14 days owing to leg cramps. Consequently, a sodium-glucose cotransporter 2 inhibitor was not attempted.

Six weeks after ablation, a stress cardiac magnetic resonance imaging study demonstrated an LVEF of 26%; midmyocardial linear atypical late gadolinium enhancement in the basal septum and inferior wall, suggestive of fibrosis of nonischemic etiology; and normal first-pass myocardial perfusion during regadenoson stress. Three months after ablation, he was instructed to stop his WCD, as it recorded no concerning arrhythmias. A subsequent 24-hour Holter demonstrated sinus rhythm with 0.53% PACs and 0.56% premature ventricular complexes. Six months after ablation, he was clinically doing well and an echocardiogram estimated an LVEF of 50%–55%.

Literature review

A literature review on incessant AVNRT confirmed by electrophysiology study is presented in Supplementary Data. Four patients had PR intervals of greater than 200 ms during sinus rhythm.[8–11] All were diagnosed by the authors as incessant typical AVNRT. All patients had prior electrophysiology procedures; 2 had prior catheter ablations and 2 had permanent pacemakers. None described AV node function curves. Selvaraj and colleagues[8] were unable to characterize AV node function curves owing to repeated initiation of AVNRT during drive trains. Tabbah and Abi-Saleh[9] stated there was "no evidence of fast pathway anterograde conduction" but did not present AV node function curves. Higuchi and colleagues[10] required "evidence of dual AV nodal physiology" for patients in their series. Catheter ablation targets were the slow pathway in 2 patients and

Table 1 Characteristics of patients with incessant atrioventricular nodal reentrant tachycardia with prolonged PR interval during sinus rhythm

	Patient (listed by first author of publication)				
	Selvaraj[8]	Tabbah[9]	Higuchi[10]	Rizzello[11]	Wang
Baseline					
Age, y	45	42	61	83	89
Sex	M	F	F	M	M
AVNRT type	Typical	Typical	Typical	Typical	Typical
Heart rate, beats/min	130	110	75	110	147
PR interval, ms	380	320	290	400	250
Ejection fraction, %	Normal	–	–	–	10–15
History of atrial fibrillation	N	Y	N	Y	N
Prior ablation procedures, n	0	2	3	0	0
Pacemaker	Y	N	N	Y	N
Electrophysiology study					
AH (sinus), ms	274	–	–	340	142
HV (sinus), ms	49	–	–	60	61
SVT CL, ms	504	–	782	520	379
AH (SVT), ms	–	–	638	–	314
HA (SVT), ms	–	–	144	–	65
VA (SVT), ms	95	–	59	<70	0
Anterograde					
Wenckebach CL, ms	–	–	–	–	370
Fast pathway ERP, ms	–	–	600	–	<230
Slow pathway ERP, ms	–	–	–	–	–
Retrograde					
Wenckebach CL, ms	–	–	–	–	450
Fast pathway ERP, ms	–	–	–	–	330
Slow pathway ERP, ms	–	–	–	–	280
Catheter ablation target	SP	RFP	SP	–	SP

Dashes (–) indicate data not reported or not applicable.

AVNRT = atrioventricular nodal reentrant tachycardia; CL = cycle length; ERP = effective refractory period; F = female; M = male; N = no; SP = slow pathway; SVT = supraventricular tachycardia; RFP = retrograde fast pathway; Y = yes.

the retrograde fast pathway in 1 patient.[8–10] All were successful and there were no complications. Pacemaker adjustment was used to treat the fourth patient.[11] These patients and the patient from this case report are listed in Table 1.

Discussion

This is the first description of successful slow pathway ablation for incessant typical AVNRT in a patient with a prolonged PR interval during sinus rhythm and a continuous anterograde AV node function curve. In a similar patient, Tabbah and Abi-Saleh[9] have suggested that "retrograde fast pathway ablation is a viable approach and may well be the preferred option." Aggregate data from the literature review suggest that the slow pathway should be the preferred catheter ablation target for both typical and atypical forms of incessant AVNRT. In fact, retrograde fast pathway ablation involves an anatomic region that Katritsis and Becker[12] have stated "has no indication in the modern treatment of AVNRT."

Incessant AVNRT is a colloquial term and is undefined in guidelines.[1,2] Kawamura and colleagues[5] classified AVNRT as "incessant" based on 2 criteria: (1) presence of tachycardia at least 50% of the time, and (2) ambulatory telemetry or in-hospital monitoring of either continuous tachycardia episodes or nonsustained episodes interposed by 2 sinus beats. This appears to be a reasonable working definition for future studies, at least until criteria are proposed from medical societies or working groups.

The 2 most apparent changes in AV node function curves after ablation were the inability to induce AVNRT during programmed atrial stimulation and the disappearance of the retrograde jump during programmed ventricular stimulation. A possible explanation is modification of the anterograde slow pathway with elimination of the retrograde slow pathway. Shortening of the retrograde fast pathway effective refractory period following ablation may be due to loss of electroanatomic inhibition of the fast pathway by the slow pathway, which has been observed in anterograde conduction.[13] Another possible explanation is elimination of a second slow pathway involved in perpetuating, but not initiating, AVNRT. More than 1 slow pathway can be present despite continuous AV function curves.[14] Slight loss of the "tail" of the anterograde AV node function curve, as seen in the case report patient, may be observed after ablation.[14]

The possibility of concealed atrio-Hisian bypass tracts participating as the retrograde fast pathway in incessant typical AVNRT is intriguing given their resistance to AV nodal blocking agents.[15] This was unlikely in the case report patient, as the baseline retrograde conduction was decremental and exhibited dual AV nodal physiology.

Incessant AVNRT may initiate from "either slight change in sinus rate and/or premature atrial complexes with varying coupling intervals."[5] The initiation mechanism may have therapeutic implications if catheter ablation is unavailable, undesired, or unsuccessful. For example, PACs may be treated with catheter ablation or antiarrhythmic drugs.

Finally, this is the first report of incessant AVNRT and reduced LVEF where an underlying idiopathic nonischemic cardiomyopathy with late gadolinium enhancement was visualized by cardiac magnetic resonance imaging. A WCD on discharge is worth consideration in higher-risk patients.

Conclusion

Catheter ablation of the slow pathway for incessant typical AVNRT with a prolonged PR interval during sinus rhythm

and a continuous anterograde AV node function curve can be effective and safe.

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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.2023.08.016.

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