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NEW RESEARCH PAPERS

# Clinical and Electrophysiological Characteristics of Incessant Atrioventricular Nodal Re-Entrant Tachycardia

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## ABSTRACT

**OBJECTIVES** This study investigated clinical and electrophysiological findings in patients with incessant atrioventricular nodal reentrant tachycardia (AVNRT).

**BACKGROUND** AVNRT is the most frequent cause of paroxysmal supraventricular tachycardia (SVT) and, rarely, incessant SVT. There are a few case reports describing incessant AVNRT.

**METHODS** Among 342 patients treated using ablation for AVNRT, we identified 8 patients with incessant AVNRT (2.3%). We describe the clinical and electrophysiological features of patients with incessant AVNRT and compare them with those of patients with paroxysmal AVNRT ($n = 334$).

**RESULTS** This study population consisted of 5 men and 3 women with incessant AVNRT. Patients with incessant AVNRT presented more frequently with the fast-slow form than those with paroxysmal AVNRT (63% vs. 14%, respectively, $p < 0.001$). The ejection fraction in patients with incessant AVNRT was significantly lower than that in patients with paroxysmal AVNRT (49 ± 12% vs. 60 ± 8%, $p = 0.03$). The H-V interval in patients with incessant AVNRT was significantly longer than that in patients with paroxysmal AVNRT. A large circuit path length is inferred by spontaneous tachycardia induction in response to slight changes in sinus rate or random premature beats, suggesting that slight changes in rate produce changes in atrial or nodal refractoriness and provoke SVT. Catheter ablation in the conventional slow pathway region was successful in eliminating SVT.

**CONCLUSIONS** AVNRT can rarely present as incessant SVT, mimicking permanent junctional reciprocating tachycardia, and can be associated with tachycardia-associated cardiomyopathy. Catheter ablation in the slow pathway region leads to long-term success. (J Am Coll Cardiol EP 2016;2:596–602) © 2016 by the American College of Cardiology Foundation.

Most patients with atrioventricular nodal re-entrant tachycardia (AVNRT) have dual anterograde atrioventricular (AV) node pathways with disparate electrophysiological properties (1,2). For patients with AVNRT, both the more common short R-P (slow-fast) type and the less common long R-P (fast-slow) type typically present as paroxysmal supraventricular tachycardia (SVT). The differential diagnosis of long R-P tachycardia includes atrial tachycardia (AT); orthodromic atrioventricular re-entrant tachycardia (AVRT), using a slowly conducting and decremental accessory

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pathway (also called the permanent form of junctional reciprocating tachycardia [PJRT]); and AVNRT (3–7). Although the history of incessant tachycardia favors PJRT, the same may be observed in AT, AVNRT, or junctional tachycardia (JT). AVNRT frequently causes paroxysmal and, rarely, the incessant forms of SVT. There are a few case reports describing incessant AVNRT (8–10). Incessant AVNRT may also be associated with tachycardia-induced cardiomyopathy. Our study systematically describes the electrophysiological findings and clinical characteristics of AVNRT in patients who presented with incessant SVT.

## METHODS

We performed a retrospective review of patients who underwent catheter ablation for AVNRT between February 2006 and January 2014. A total of 342 consecutive patients with AVNRT were referred for ablation. Eight patients (2.3%) presented with incessant AVNRT. Incessant SVT was defined as presence of tachycardia at least 50% of the time and ambulatory telemetry or in-hospital monitoring of either continuous tachycardia episodes or nonsustained episodes interposed by 2 sinus beats.

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Successful ablation was defined as either no echo beat or no more than 1 echo beat in the absence of tachycardia inducibility. Left ventricular ejection fraction (LVEF) was calculated by Simpson's method, and tachycardia-induced cardiomyopathy was defined as an LVEF <50% that normalized or clearly improved by ≧20% after ablation procedure. We investigated clinical and electrophysiological characteristics in patients with incessant AVNRT (n = 8) and compared them with those in patients with paroxysmal AVNRT (n = 334). All patients gave written informed consent before electrophysiological studies (EPS) and catheter ablation were performed.

We investigated EPS findings, including tachycardia cycle length (TCL), P-wave duration, A-H interval, H-V interval, and effective refractory period (ERP) during sinus rhythm (SR).

Three quadripolar catheters were placed through the right femoral vein in the high right atrium, the His bundle position, and the right ventricle (RV). A decapolar catheter was inserted in the coronary sinus (CS) with the proximal pole at the ostium. Intravenous isoproterenol and atrial overdrive or programmed stimulations were used to induce arrhythmia if spontaneous tachycardia was not present at baseline. Radiofrequency ablation was performed in the slow pathway (SP) with continuous temperature feedback control of the power output to achieve a target temperature of 60°C. Initial power setting was 25 W, and the maximum power used was 50 W for a maximum of 60 s. Acute successful ablation was defined by the inability to induce tachycardia after 30 min on monitor after ablation procedure, despite aggressive burst atrial pacing and use of isoproterenol. Patients were followed in our respective clinics to assess return of symptoms or documented tachycardia. In patients with LV dysfunction, we performed follow-up echocardiography 3 to 6 months after ablation.

AVNRT was diagnosed according to classic electrophysiological criteria (11–13). If rapid RV pacing was >15 beats during SVT at a cycle length (CL) of 10 to 40 ms less than TCL dissociated the ventricle from tachycardia without termination excluded AVRT. This maneuver needs to pace long enough to ensure complete RV capture; therefore, it is important to compare QRS complexes of overdrive pacing during tachycardia to those seen during RV pacing in SR. During SVT, we performed RV overdrive pacing, which was initiated at a CL of 10 to 40 ms shorter than the TCL, until 1:1 ventriculoatrial conduction was achieved. If overdrive pacing resulted in the termination of tachycardia, SVT was reinduced, and the maneuver was repeated. We counted the number of unfused ventricular paced beats necessary for atrial capture. AT was excluded by tachycardia termination with ventricular overdrive pacing without accelerating the atrial CL or with a V-A-V response after 1:1 VA conduction and acceleration of the atrium to the paced CL. If we could not distinguish AVNRT from PJRT, we used a His refractory PVC inserted close to the atrial insertion site without advancement of the atrium as the most helpful diagnostic maneuver to exclude PJRT. Furthermore, late-coupled His refractory premature ventricular complex also advanced or delayed atrial activation and confirmed the diagnosis of PJRT (13). JT was differentiated from typical AVNRT by response to atrial overdrive pacing and by programed early and late atrial extrastimuli introduced during SVT (14–16). The type of AVNRT was defined according to the designation of Jackman et al. (17) and included slow-fast (typical), slow-slow (atypical), and fast-slow (atypical) variants of

### ABBREVIATIONS AND ACRONYMS

**AT** = atrial tachycardia

**AVNRT** = atrioventricular nodal re-entrant tachycardia

**AVRT** = atrioventricular re-entrant tachycardia

**CL** = cycle length

**CS** = coronary sinus

**ECG** = electrocardiogram

**EF** = ejection fraction

**EPS** = electrophysiological study

**ERP** = effective refractory period

**FP** = fast pathway

**ICD** = implantable cardioverter-defibrillator

**JT** = junctional tachycardia

**PJRT** = permanent form of junctional reciprocating tachycardia

**RV** = right ventricle

**SP** = slow pathway

**SVT** = supraventricular tachycardia

**TCL** = tachycardia cycle length

**TABLE 1 Comparison of Clinical Characteristics (AVNRT)**

| | Incessant AVNRT (n = 8) | Paroxysmal AVNRT (n = 334) | p Value |
|---|---|---|---|
| Age, yrs | 62 ± 21 | 54 ± 17 | 0.11 |
| Male, % | 5 (62) | 160 (48) | 0.43 |
| TCL, ms | 408 ± 21 | 365 ± 35 | 0.11 |
| Ejection fraction, % | 49 ± 12 | 60 ± 8 | 0.03 |
| Typical/atypical | 3/5 | 287/47 | 0.001 |
| Medication | 8 (100) | 104 (31) | 0.001 |
| Palpitation/dizziness | 3 (37) | 290 (87) | 0.001 |

Values are mean ± SD, n (%), or n.
AVRNT = atrioventricular nodal reentrant tachycardia; TCL = tachycardia cycle length.

AVNRT. Continuous variables were expressed as mean ± SD and were compared using the Student *t* test. Categorical variables, expressed as numbers and percentages in the different groups, were compared using Fisher exact test. Values of $p < 0.05$ were considered statistically significant.

**TABLE 2 Clinical Characteristics of Incessant AVNRT**

| | Patient # | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| Age, yrs | 70 | 53 | 82 | 21 | 51 | 67 | 82 | 75 |
| Sex | M | M | M | F | M | M | F | F |
| BMI, kg/m$^2$ | 27 | 31 | 29 | 20 | 44 | 23 | 28 | 33 |
| AVNRT type | F-S | F-S | F-S | F-S | F-S | S-F | S-F | S-F |
| Symptom | No | No | CHF | No | Palp | Palp | CHF | Palp |
| History of AF | No | No | Yes | Yes | No | No | No | Yes |
| % EF before | 40 | 45 | 35 | 48 | 65 | 62 | 45 | 55 |
| % EF after | 55 | 58 | 45 | 58 | 65 | 65 | 55 | (-) |
| Heart disease | CAD | No | CAD | No | CAD | No | No | No |
| Medication | BB | CCB | Am, BB | BB | BB | CCB | BB | BB |
| Induction method | Spon | Spon | Spon | A1A2 | Spon | Spon | A1A2 | Spon |
| TCL, ms | 400 | 395 | 420 | 410 | 360 | 410 | 420 | 450 |
| P-wave, ms | 91 | 116 | 52 | 65 | 54 | 80 | 138 | 78 |
| P-Q interval, ms | 151 | 165 | 158 | 126 | 142 | 136 | 150 | 183 |
| A-H interval, ms | 90 | 110 | 87 | 70 | 102 | 90 | 95 | 89 |
| H-V interval, ms | 61 | 60 | 71 | 58 | 45 | 46 | 53 | 50 |
| Anterograde | | | | | | | | |
| FP-ERP, ms | 330 | 450 | 440 | 410 | 440 | 460 | 490 | 440 |
| SP-ERP, ms | (-) | 420 | (-) | 390 | 400 | 300 | 320 | 350 |
| Retrograde | | | | | | | | |
| FP-ERP, ms | 380 | 620 | NA | NA | 370 | NA | 350 | 410 |
| SP-ERP, ms | 270 | 450 | NA | NA | 230 | NA | (-) | (-) |
| VA-ERP, ms | 270 | 450 | NA | NA | 230 | NA | 250 | 410 |
| VA Wenckebach, ms | 350 | 600 | 280 | 600 | 340 | 320 | 340 | 410 |
| Fluoroscopic time (min) | 25 | 15 | 23 | 11 | 27 | 6 | 19 | 5 |
| Successful ablation | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |

A1A2 = extrastimulus atrial; AF = atrial fibrillation; Am = amiodarone; BB = beta-blocker; BMI = body mass index; CAD = coronary artery disease; CCB = calcium-channel blocker; CHF = congestive heart failure; EF = ejection fraction; ERP = effective refractory period; FP = fast pathway; F-S = fast-slow; NA = not applicable; Palp = palpitation; Spon = spontaneous; SP = slow pathway; S-F = slow-fast; other abbreviations as in Table 1.

## RESULTS

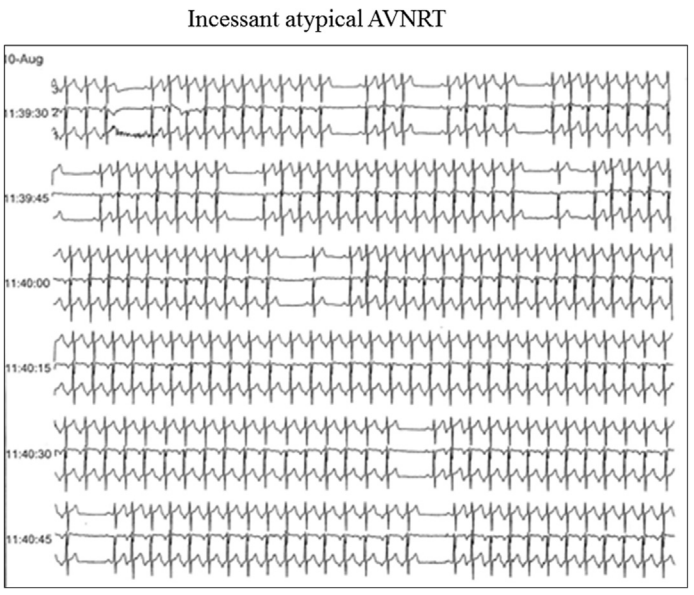
The study population consisted of 5 men and 3 women with incessant AVNRT. Mean age was 62 ± 21 years. Three patients (37%) had typical (slow-fast) AVNRT, and 5 patients (63%) had atypical (fast-slow) AVNRT. One patient had an implantable cardioverter-defibrillator (ICD) implanted for ischemic heart disease and presented with multiple shocks due to SVT. Table 1 compares baseline characteristics of incessant AVNRT to those of paroxysmal AVNRT. The EF in patients with incessant AVNRT was significantly lower than those with paroxysmal AVNRT (49 ± 12% vs. 60 ± 8%, respectively, $p = 0.03$). In those with typical AVNRT (n = 290), only 3 patients had incessant AVNRT, whereas in those with atypical AVNRT (n = 52), 5 patients had incessant AVNRT (1.0% vs. 9.6%, respectively, $p < 0.001$). Patients with paroxysmal AVNRT presented more frequently with palpitation or dizziness than those with incessant AVNRT (87% vs. 37%, respectively, $p < 0.001$). Furthermore, patients with incessant AVNRT were more likely to be treated with medications than those with paroxysmal AVNRT (100% vs. 31%, respectively, $p < 0.001$). The individual clinical characteristics of those with incessant AVNRT are shown in Table 2. Three patients had structural heart disease, 1 patient had undergone coronary artery bypass surgery, and 1 patient had an ICD for ventricular tachycardia. Three patients had palpitations, 2 patients had symptoms of heart failure, and 3 patients had no symptoms. Five patients (63%) had LV dysfunction before catheter ablation, and all patients had improved LV systolic function after catheter ablation. Six patients were started on medical therapy after diagnosis of incessant SVT, whereas 2 patients were already taking beta-blockers for coronary artery disease at the time of diagnosis. Amiodarone was used for 1 week for an acute SVT episode 1 year prior to the study. All AV node-active medications were discontinued before the study began. Six patients (75%) with incessant AVNRT were spontaneously inducible, and 2 patients were inducible by atrial extrastimulus. Patients with incessant AVNRT were more spontaneously induced as compared to those with paroxysmal AVNRT (75% vs. 6%, respectively, $p < 0.001$).

Figure 1 shows an example of incessant AVNRT during 24-h ambulatory monitoring. SVT was initiated with either slight change in sinus rate and/or premature atrial complex with varying coupling intervals. Atrial tachycardia was excluded in 6 patients who had a V-A-V response during ventricular overdrive pacing and in 2 that had a pseudo-V-A-A-V response (Figure 2). In all patients with atypical AVNRT, PVCs

were introduced during tachycardia at the time of His refractoriness; these did not alter the timing of the subsequent atrial activation, unless retrograde His bundle activation was advanced (Figure 3). None showed advancement of the atrial electrogram when the His refractory-paced ventricular beat was inserted close to the atrial insertion site. Three typical AVNRTs were terminated by a late coupled PAC (Figure 4).

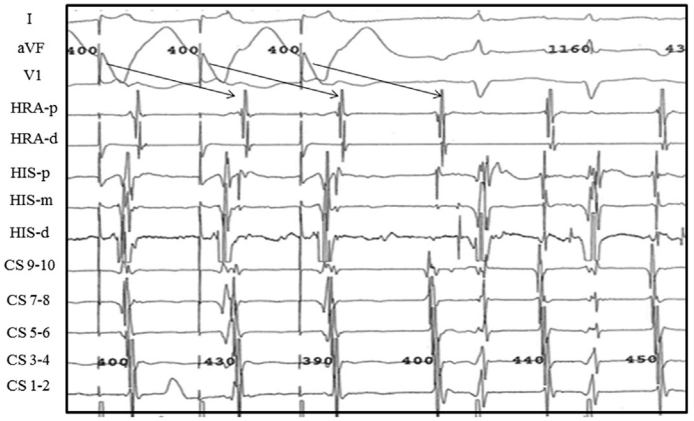
Comparison of the ECG and EPS findings in incessant AVNRT with those in paroxysmal AVNRT are shown in Table 3. There were no statistically significant differences in baseline P-wave duration, PQ interval, A-H interval, and TCL between the 2 groups. There were no statistically significant differences in baseline antegrade fast pathway (FP) and SP-ERP and retrograde FP and SP-ERP between the 2 groups. Furthermore, we compared typical AVNRT or atypical (paroxysmal) with incessant AVNRT in terms of the refractory periods of SP and FP (anterograde and retrograde). In typical AVNRT (paroxysmal), the average anterograde FP-ERP was 408 ± 88 ms, and the average anterograde SP-ERP was 351 ± 40 ms. The average retrograde FP-ERP was 421 ± 78 ms, and average retrograde SP-ERP was 333 ± 64 ms in typical AVNRT (paroxysmal). In atypical AVNRT (paroxysmal), the average anterograde FP-ERP was 416 ± 99 ms, and the average anterograde SP-ERP was 357 ± 42 ms. The average retrograde FP-ERP was 424 ± 86 ms and the average retrograde SP-ERP was 331 ± 58 ms in the atypical AVNRT (paroxysmal). There were no statistically significant differences in baseline antegrade FP and SP-ERP and retrograde FP and SP-ERP between the 2 groups. The only significant differences between the groups were the H-V intervals (incessant AVNRT = 57 ± 9 ms vs. 44 ± 7 ms in paroxysmal AVNRT; p = 0.04). Individual EPS findings for patients with incessant AVNRT are shown in Table 2. Among 8 patients with incessant AVNRT, 2 had a long P-wave duration (>100 ms), and 4 had a long H-V interval (>55 ms). Six patients had anterograde dual AV nodal pathway physiology, whereas 2 did not (Table 2, cases 1 and 3 had fast-slow type). Three patients had retrograde dual AV nodal pathway physiology, 2 did not; and in 3 patients, the retrograde refractory period could not be assessed. In the typical (slow-fast) AVNRT (Table 2, cases 6, 7, and 8), the average anterograde FP-ERP was 463 ± 25 ms and the average anterograde SP-ERP was 323 ± 19 ms. In the atypical (fast-slow) AVNRT (Table 2, cases 1 to 5), the average anterograde FP-ERP was 414 ± 21 ms, and the average anterograde SP-ERP was 403 ± 22 ms. The average retrograde FP-ERP was 446 ± 78 ms, and the average retrograde SP-ERP was 316 ± 51 ms in atypical (fast-slow) AVNRT (Table 2, cases 1, 2, and 5).

**FIGURE 1 Holter Recording in a Patient With Incessant SVT Leading to Mild Decrease in Ejection Fraction**



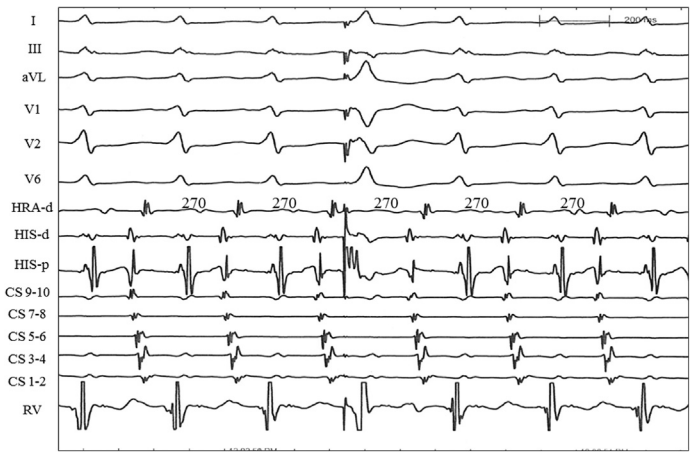

The patient has long RP interval tachycardia with negative P-wave in the inferior leads and spontaneous initiation and termination in the QRS complex. SVT initiated with slight changes in sinus rate with varying coupling intervals. This is consistent with a diagnosis of permanent form of junctional reciprocating tachycardia, atrial tachycardia, and atypical AVNRT. AVNRT = atrioventricular nodal re-entrant tachycardia; SVT = supraventricular tachycardia.

**FIGURE 2 Pseudo-V-A-A-V Response**




Pseudo-V-A-A-V response to the termination of ventricular overdrive pacing during tachycardia due to a long retrograde conduction time. CS = coronary sinus; d = distal; HRA = high right atrium; m = middle; p = proximal.

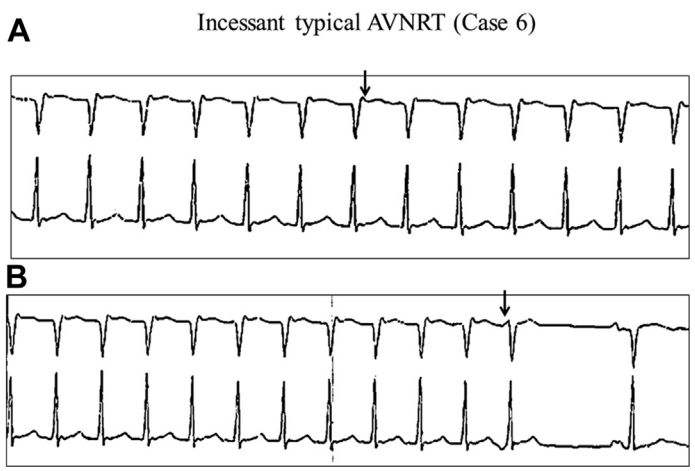
**FIGURE 3 Single Ventricular Extrastimulus**




Single ventricular extrastimulus delivered from the RV apex at the time His refractoriness fails to advance the subsequent atrial activation. RV = right ventricle; other abbreviations as in Figures 1 and 2.

Catheter ablation in the conventional SP region (at the tricuspid annulus at the level of the CS os) was successful in eliminating SVT in all patients with no recurrence during the follow-up period (28 ± 16 months). Mean EF in patients with incessant AVNRT was improved after ablation (before ablation 48 ± 16% vs. after ablation 57 ± 13%, p = 0.005).

**FIGURE 4 Holter Recording Showing Typical AVNRT**




**(A)** Holter recording shows typical AVNRT with a P-wave noted at the terminal part of QRS **(arrow)**. **(B)** Spontaneous PAC **(arrow)** that terminates SVT without affecting the next V, supporting diagnosis of AVNRT over junctional tachycardia. PAC = premature atrial complex; other abbreviations as in Figures 1 and 2.

## DISCUSSION

**MAIN FINDINGS.** We systematically describe the clinical ECG and EPS features of patients with incessant AVNRT. We found that typical AVNRT rarely presented with incessant SVT (1.0%) compared with atypical AVNRT (9.6%). The H-V interval in patients with incessant AVNRT was longer than those with paroxysmal AVNRT. In addition, we report that incessant AVNRT can be associated with tachycardia-induced cardiomyopathy and that catheter ablation in the conventional SP region is curative in all cases, leading to improvement in symptoms and left ventricular systolic function.

**DEFINITION AND CLINICAL CHARACTERISTICS OF INCESSANT SVT.** Incessant tachycardia was first referred by Cassidy et al. (18) in 1924. The definition of incessant tachycardia was based on the ECG characteristics of the tachycardia rather than underlying mechanism. In our cases, incessant SVT was defined as tachycardia being present at least 50% of the time during ECG monitoring or paroxysms of tachycardia separated by 2 sinus beats (19). Incessant tachycardia has been described in young patients without structural heart disease (19,20), as well as in patients with structural heart disease (21). In the present study, the average age was 62 ± 21 years, and 3 patients (43%) had coronary artery disease. Furthermore, 5 patients (63%) with incessant AVNRT developed cardiomyopathy. In general, short TCL was more likely to be symptomatic with palpitations, and patients might be more aware of a tachycardic episode; thus, patients with short TCL might be detected and treated earlier, leaving less time for tachycardia-induced cardiomyopathy to develop. On the other hand, patients with longer TCL might not present early with palpitations but rather later with symptoms of heart failure. However, the patient with the shortest TCL in the incessant group had no symptoms in our study. We suppose that the best correlate to development of LV dysfunction might be lack of symptoms. Furthermore, 4 patients with incessant AVNRT had a long H-V interval, and all 4 had a tachycardia-induced cardiomyopathy. This result showed that cardiomyopathy is often associated with a long H-V interval. Cruz et al. (22) described a similar result that the H-V interval in patients with tachycardia-induced cardiomyopathy was significantly longer compared with patients with incessant SVT without cardiomyopathy.

**DIFFERENTIAL DIAGNOSIS.** Incessant long RP was diagnosed as follows: AVRT by using a slowly conducting and decremental accessory pathway

**TABLE 3 Comparison of Electrocardiographic and Electrophysiologic Characteristics**

| | Incessant AVNRT (n = 8) | Paroxysmal AVNRT (n = 334) | p Value |
|---|---|---|---|
| TCL, ms | 408 ± 21 | 365 ± 35 | 0.11 |
| P-wave, ms | 86 ± 31 | 85 ± 24 | 0.85 |
| P-Q interval, ms | 158 ± 13 | 151 ± 35 | 0.53 |
| A-H interval, ms | 91 ± 13 | 89 ± 32 | 0.81 |
| H-V interval, ms | 57 ± 9 | 44 ± 7 | 0.04 |
| Induction method | | | |
| Spontaneous | 6 | 21 | 0.001 |
| Burst pacing, A/V | 0/0 | 108/34 | |
| Extrastimulus, A/V | 2/0 | 116/28 | |
| Noninducible | 0 | 27 | |
| Anterograde | | | |
| FP-ERP, ms | 430 ± 69 | 410 ± 99 | 0.25 |
| SP-ERP, ms | 355 ± 52 | 353 ± 42 | 0.82 |
| FP-ERP–SP-ERP | 83 ± 21 | 56 ± 18 | 0.11 |
| Retrograde | | | |
| FP-ERP, ms | 446 ± 78* | 422 ± 86 | |
| SP-ERP, ms | 316 ± 51* | 332 ± 64 | |
| FP-ERP–SP-ERP | 129 ± 30 | 90 ± 27 | |
| VA-ERP, ms | 308 ± 98† | 302 ± 89 | |
| VA Wenckebach, ms | 408 ± 103 | 401 ± 110 | |
| Fluoroscopic time, min | 16 ± 7 | 21 ± 13 | 0.49 |

Values are mean ± SD or n. *Retrograde FP and SP-ERP could be assessed in 3 patients. †VA-ERP could be assessed in 3 patients.

A/V = atrial stimulus/ventricular; other abbreviations as in Tables 1 and 2.

(also called PJRT), AT, and atypical AVNRT. Although the history of incessant tachycardia favors PJRT, the same may be observed in either AT or AVNRT (23). Ventricular pacing maneuvers can be very helpful in differentiating AVNRT from AVRT and AT (13,24). In our study, AT was excluded by a V-A-V response following ventricular capture and in 2 patients by spontaneous episodes of AV dissociation during tachycardia. In all patients, PJRT was excluded by lack of effect of His-committed PVCs given close to the earliest atrial activation during SVT. JT may mimic typical AVNRT. In our study, 3 patients showed tachycardia termination with late PAC, and the remainder showed that late PAC affected the next ventricular beat. Padanilam et al. (14) reported that typical AVNRT was differentiated from JT with SVT termination with PAC induced while the His bundle was committed. Fan et al. (15) showed that alteration of the SVT CL with a late PAC excluded JT.

**POSSIBLE MECHANISM OF INCESSANT AVNRT.** The relatively small sample size precludes a definition description of the tachycardia mechanism. Several features are, however, noteworthy. First, initiation of the tachycardia is similar to that found for patients with PJRT in that slight change in sinus cycle length or de novo initiation after a sinus conducted beat could provoke tachycardia, suggesting that the circuit was composed of a large path length of decremental conducting tissue. For example, a shorter sinus cycle length could initiate sufficient delay in anterograde conduction to allow for recovery in conduction in the retrograde direction. Alternatively, the shorter cycle length could induce decreases in atrial refractoriness that encouraged perpetuation of tachycardia. The same type of arguments can be made for sinus node deceleration (25).

This is nicely illustrated in **Figure 1** where atypical AVNRT is initiated by either 1 or 2 sinus conducted beats following spontaneous tachycardia termination. In this case, we hypothesize that during the sinus conducted beat, there is anterograde conduction over the fast pathway with no penetration of the SP. Hence, the sinus impulse found the retrograde SP available for conduction and induction of tachycardia. We postulated a similar mechanism for those with typical AVNRT with only sole anterograde conduction over the SP in SR leaving the fast pathway available for initiation of typical AVNRT. We could not detect specific differences in properties of anterograde versus retrograde conduction or refractoriness between the 2 groups but that might have been due to the smaller number of patients available for the incessant AVNRT group.

**STUDY LIMITATIONS.** The major limitations of this study are inadequate numbers of patient studies and inability to define all electrophysiological parameters because of the incessant nature of the tachycardia.

This study has only 8 patients with incessant AVNRT. The small number of incessant AVNRT patients limits conclusions based on the statistical tests.

Therefore, these results have to be interpreted with caution. Second, we did not obtain echocardiogram in all patients with AVNRT; hence, we cannot determine from our data the percentage of time a patient needs to be in SVT to develop a myopathy. Our data are limited to the frequency of cardiomyopathy in those with AVNRT. Furthermore, we could not completely investigate the retrograde ERP due to an incessant AVNRT. Further study will be required to ascertain the mechanism of incessant AVNRT.

## CONCLUSIONS

Our study highlights the fact that AVNRT can rarely present as incessant SVT mimicking PJRT and can be associated with tachycardia-associated

cardiomyopathy. Furthermore, we outlined the clinical descriptors in terms of increased H-V interval that are unique to the incessant group.

Catheter ablation in the conventional SP region was curative in all cases, leading to improvement in symptoms and EF. Finally, we infer that the tachycardia mechanism is likely related to a long path length with decremental properties allowing for ready induction with slight changes in the sinus cycle length.

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## PERSPECTIVES

**COMPETENCY IN MEDICAL KNOWLEDGE:** AVNRT can rarely present as incessant SVT mimicking PJRT and can be associated with tachycardia-associated cardiomyopathy. Catheter ablation in the conventional SP region was curative in all cases, leading to improvement in symptoms and left ventricular function.

**TRANSLATIONAL OUTLOOK:** Although this study included a small number of patients and given the low incidence of incessant AVNRT, we believe this study is adequate in describing the clinical and electrophysiological characteristics and can provide further data regarding the long-term benefits of ablation.

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