

SHORT REVIEW

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## Electrocardiographic and Electrophysiologic Insights into Atrioventricular Nodal Re-entry Tachycardia: Diagnostic Update

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**Abstract:** Atrioventricular nodal re-entry tachycardia is a common supraventricular arrhythmia. The rate of recurrence is relatively high, and accordingly ablative therapy became the first-line suggested therapy. In this review, we highlight the electrocardiographic clues to the diagnosis of atrioventricular nodal re-entry tachycardia, also we present the electrophysiological data and maneuvers that enable the ruling out of other supraventricular tachycardias and ensure an accurate and specific diagnosis of atrioventricular nodal re-entrant tachycardia.

**Keywords:** dual physiology, re-entry, tachycardia, electrocardiographic, electrophysiologic

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## Background

The current concept of atrioventricular nodal re-entrant tachycardia (AVNRT) implies an arrhythmia that involves a highly complex atrioventricular junctional area.<sup>1,2</sup> Accordingly, it is essential for electrophysiologists to be aware of this complex anatomic and electrophysiologic entity to ensure an optimal approach to its treatment. AVNRT is the most common form of sustained regular narrow complex tachycardia. It is caused by the presence of a dual atrioventricular nodal anatomy and physiology, with a fast and a slow pathway forming a substrate for re-entry. The fast pathway has a higher conduction velocity and a longer refractory period than the slow pathway.<sup>3</sup> The typical and most common form of AVNRT known as “slow-fast” occurs when the antegrade limb of the re-entrant circuit utilizes the slow pathway and the retrograde limb utilizes the fast pathway. Other less common forms are the “fast-slow” (antegrade fast, retrograde slow) and the “slow-slow” variants, and two or more of these variants can coexist in the same patient.

The presence and function of a slow pathway in subjects who never experience an episode of AVNRT is debated, although the slow pathway in normal subjects probably has a role in maintaining conduction in circumstances of autonomic imbalance with extreme hypervagal tone.<sup>4</sup>

Clinical manifestations of AVNRT consist mainly of palpitations, although syncope due to poor ventricular filling and reduced cerebral circulation may occur in patients with a rapid ventricular rate and/or prolonged tachycardia.<sup>1</sup> Acute termination of tachycardia can be achieved using vagal maneuvers and/or atrioventricular node suppressing agents.

The concept of upper and lower common pathways is not unanimously accepted in AVNRT. There has been considerable evidence against the presence of these common pathways as distinct entities, the anatomic presence and functional relevance of these pathways remain speculative.<sup>5</sup> Left-sided AVNRT is rare,<sup>6</sup> it involves slow potentials located on the septal aspect of the left atrium, and a trans-septal approach is required for ablative therapy.

## Electrocardiographic Diagnosis

Electrocardiographic diagnosis of typical AVNRT is suggested by the presence of retrograde P' waves

during a supraventricular tachycardia. This is visible as pseudo R' waves in V1 and pseudo S waves in the inferior leads with an RP interval  $\leq 90$  msec<sup>7</sup> when P waves are visible (Fig. 1). In contrast, QRS alternans and ST segment alterations during supraventricular tachycardia are rather markers of orthodromic reciprocal tachycardia.<sup>7</sup> Moreover, the absence of ST segment depression during supraventricular tachycardia in the left precordial leads (V4–V6) is highly suggestive of AVNRT,<sup>8</sup> and the authors propose inclusion of this criterion in electrocardiographic algorithms for the differential diagnosis of supraventricular tachycardia.

Braunschweig et al<sup>9</sup> found that a transesophageal ventriculoatrial interval  $\leq 80$  msec is highly suggestive of AVNRT. Oh et al<sup>10</sup> found that differences in RP' intervals (dRP') between V1 and the inferior leads in the slow/slow variant have significant predictive value for AVNRT (versus orthodromic reciprocal tachycardia with a posteroseptal accessory pathway). The same study showed significantly longer RP' intervals in V1 in AVNRT with consequently longer dRP' [V1–II] ( $>25$  msec), dRP' [V1–III] ( $>23$  msec) and dRP' [V1–aVF] ( $>30$  msec). Finally, Di Toro et al<sup>11</sup> showed that the presence of a notch in the aVL lead is a sensitive marker of AVNRT versus other supraventricular tachycardias; the notch consists of a positive deflection at the end of the QRS complex during tachycardia and it is absent during sinus rhythm.

Belhassen et al<sup>12</sup> described the adenosine triphosphate test for noninvasive diagnosis of dual atrioventricular nodal physiology. Adenosine triphosphate is injected at progressive doses of 10–60 mg (in 10 mg increments) during sinus rhythm until electrocardiographic signs of dual atrioventricular nodal physiology occur (50 msec or more increase in the



**Figure 1.** Upper strip in sinus rhythm, lower strip in tachycardia (atrioventricular Nodal re-entrant tachycardia), encircled is the (R') wave in V1 lead.

PR interval in two consecutive beats), or occurrence of atrioventricular nodal echo beat. Dual atrioventricular node physiology was encountered using this method in 75% of patients, and disappeared in 96% of patients who underwent slow pathway ablation. Consequently, the authors concluded that adenosine triphosphate test enables noninvasive diagnosis of AVNRT in a high percentage of cases and can be used reliably to assess the result of catheter ablation.

The presence of 2:1 atrioventricular block is a rare finding in AVNRT, and is related more to a fast re-entrant circuit with functional block rather than to impaired underlying conduction in the atrioventricular node or infranodal area, and therefore slow pathway ablation is safe in this group of patients.<sup>13</sup> Moreover, another study<sup>14</sup> showed that rate irregularity during AVNRT is attributable to a short tachycardia cycle length giving rise to Wenckebach block in the lower “common” pathway.<sup>14</sup> A ventriculoatrial block during AVNRT is a rare phenomenon, the mechanisms are Wenckebach His-atrial block and/or 2:1 His-atrial block.<sup>15</sup>

## Electrophysiologic Approach

The presence of slow pathway (Jackman) potentials in AVNRT is frequently underestimated, so recording of these potentials in Koch’s triangle should not be missed or ignored,<sup>16</sup> and can help to guide the ablation procedure (Fig. 2). During a typical AVNRT, there is a short ventriculoatrial interval (<60 msec at the His catheter), due to retrograde atrial activation

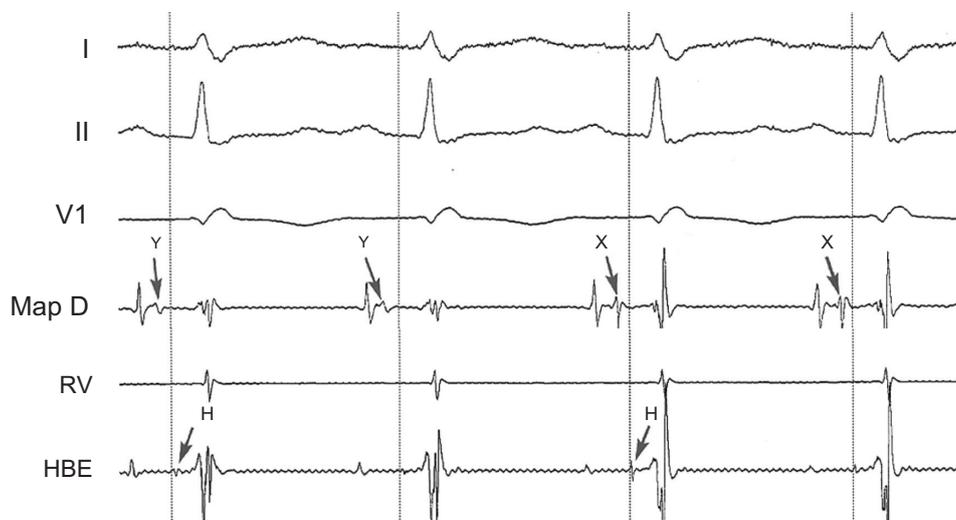
during tachycardia, classically called “concentric atrial activation” (Fig. 3).

Anatomically, the re-entrant circuit involves the atrioventricular node and the surrounding perinodal tissue with the presence of at least two pathways. Typically, the presence of dual atrioventricular node physiology is demonstrated when a sudden A-H prolongation ( $\geq 50$  msec) occurs during decremental atrial pacing or shortening of the extrastimulus coupling interval by 10 msec. Electrophysiologically, when a supraventricular tachycardia is present, the main differential diagnosis is orthodromic reciprocal tachycardia and atrial tachycardia.

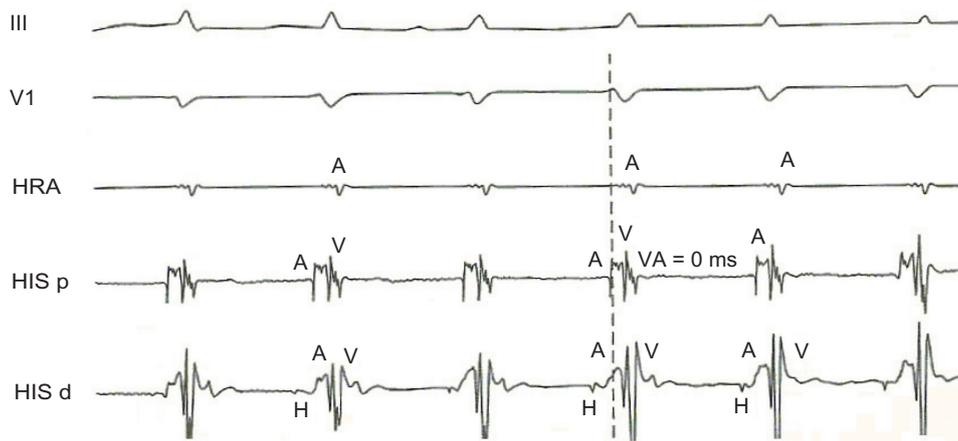
Atrial tachycardia is the least common etiologic cause of supraventricular tachycardia, and the circuit or focus is located entirely within the atria, with no antegrade or retrograde limb through the atrioventricular node. Therefore, variable atrioventricular and ventriculoatrial intervals are typically observed (Fig. 4) and ventricular overdrive pacing does not result in entrainment. Nevertheless, if it accelerates the atria (overdrive mechanism), a post-pacing A-A-V response pattern is suggestive of atrial tachycardia, and when it fails to accelerate the atria, a diagnosis of atrial tachycardia is most likely.<sup>17</sup>

## Focal junctional tachycardia versus AVNRT

A premature atrial complex timed with His refractoriness during tachycardia that leads to a change in the subsequent His timing indicates that antegrade



**Figure 2.** Slow (Jackman) potentials shown in two different sites in the transitional atrioventricular junction region: Y is a proximal site, X is a more distal site (closer to His bundle deflection).



**Figure 3.** Concentric atrial activation as demonstrated by a ventriculoatrial interval = 0 on the His proximal catheter.

slow pathway conduction is present and functional; AVNRT is also the most probable diagnosis if the premature atrial complex terminates the tachycardia. Conversely, a premature atrial complex that advances the His potential immediately afterwards without terminating the supraventricular tachycardia indicates that a retrograde fast pathway is not a part of the circuit and this favors the diagnosis of junctional focal tachycardia.<sup>18,19</sup>

### Differentiating AVNRT from Orthodromic Reciprocal Tachycardia

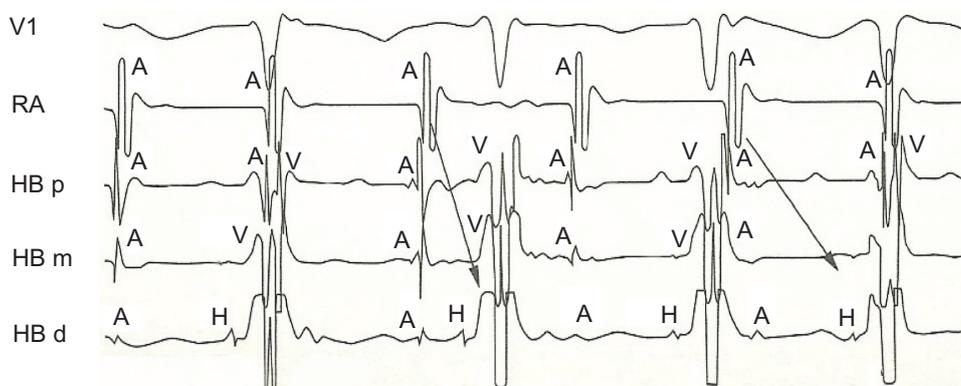
#### Entrainment in supraventricular tachycardia

Entrainment is defined as the continual or repeated resetting of a re-entrant tachycardia by a series of consecutive beats of a pacing train.<sup>17</sup> Fusion is likely

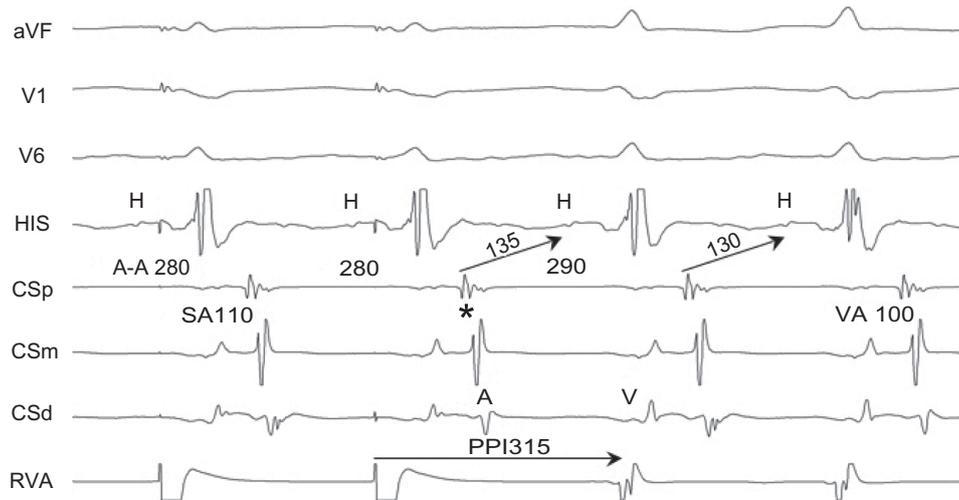
during entrainment (ie, manifest entrainment) of orthodromic reciprocal tachycardia with right ventricular stimulation if the accessory pathway is septally located, and this phenomenon can help to differentiate orthodromic reciprocal tachycardia from AVNRT<sup>20</sup> (Fig. 5). QRS fusion is absent during entrainment of AVNRT (concealed entrainment)<sup>17</sup> and this is due to collision of an orthodromic wavefront (from paced beat) and an antidromic wavefront (from spontaneous beat) inside the atrioventricular nodal tissue.

#### Differential entrainment using the SA-VA

The SA (sinoatrial) interval is the interval between the ventricular stimulus and the earliest atrial electrogram during entrainment, and the VA (ventriculoatrial) interval is the interval between the RV electrogram (apex and base) and the earliest atrial electrogram



**Figure 4.** Variable atrioventricular and ventriculoatrial intervals demonstrated with intracardiac recordings of atrial tachycardia.



**Figure 5.** Manifest fusion demonstrated in the first two QRS complexes, favoring the diagnosis of orthodromic reciprocal tachycardia.

during tachycardia;  $\Delta[SA-VA]$  is measured as  $[SA-VA]_{base} - [SA-VA]_{apex}$ .  $\Delta[SA-VA]$  is positive in AVNRT and negative in orthodromic reciprocal tachycardia with septal accessory pathways.<sup>21</sup>

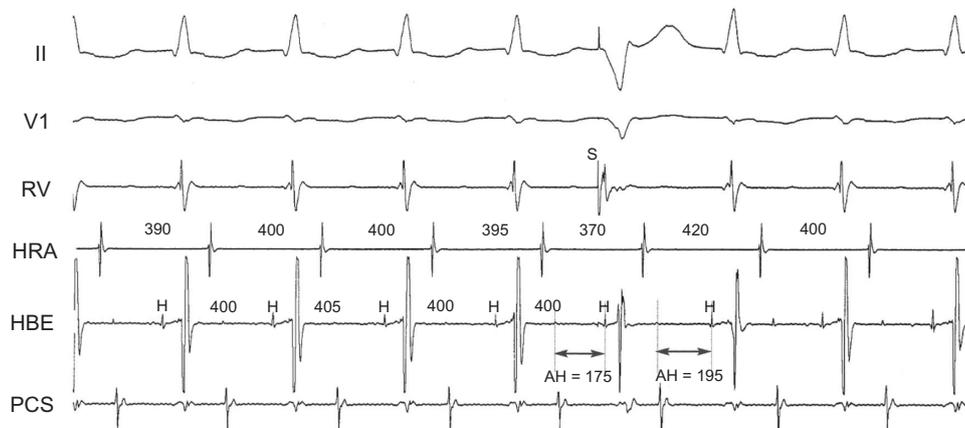
### Differential entrainment using cPPI-TCL and VA

$c[PPI-TCL]$  and VA intervals are significantly longer in AVNRT when entrainment is performed at the right ventricular base compared with the apex; these two intervals show no significant difference when there is an orthodromic reciprocal tachycardia; a differential  $cPPI-TCL > 30$  msec and/or a differential VA interval of  $>20$  msec reliably predicts AVNRT.<sup>22</sup> Note that PPI = post pacing interval; TCL = tachycardia cycle

length;  $c[PPI-TCL] = \text{corrected } [PPI-TCL] = [(PPI-TCL) - (\text{post AH} - \text{pre AH})]; [\text{post AH}]$  is the AH interval measured on the return cycle;  $[\text{pre AH}]$  is the AH recorded during tachycardia prior to pacing.

### Entrainment using $\Delta HA$ method

The His bundle and atria are activated sequentially during entrainment of AVNRT from the right ventricle and almost simultaneously during typical AVNRT; in contrast, they are activated simultaneously during entrainment of orthodromic reciprocal tachycardia and sequentially during the tachycardia. A  $\Delta HA$  [ $HA(\text{entrainment}) - HA(\text{tachycardia})$ ] cut-off value of 0 reliably differentiates AVNRT from orthodromic reciprocal tachycardia, having a positive



**Figure 6.** His refractory premature ventricular complex that advances the subsequent atria of 30 msec, demonstrating the presence of a retrograde conducting accessory pathway.



value in AVNRT and a negative value in orthodromic reciprocal tachycardia.<sup>23</sup>

### Entrainment from right ventricular apex using return cycle method

A cPPI-TCL > 110 msec is a reliable marker of AVNRT<sup>24</sup> (>95 msec in children)<sup>25</sup> whereas a value < 110 msec favors the diagnosis of orthodromic reciprocal tachycardia using a septal accessory pathway without patent pre-excitation.

### Para-Hisian pacing

Using a standard quadripolar catheter placed at the His position, this technique consists of pacing at low output for right ventricular capture and at high output for both right ventricular and His capture. Retrograde activation time and pattern are compared during capture and loss of capture of the His bundle while pacing from a para-Hisian position.<sup>26</sup> The SA interval (interval from stimulus to earliest retrograde atrial activation) and VA interval (interval from local ventriculogram to earliest retrograde atrial activation) were compared during His capture and His/right ventricular capture.  $\Delta SA > 40$  msec has been found to be specific for AVNRT,<sup>27</sup> also it enables ruling out of a “bystander” accessory pathway that is not necessary operative during the studied tachycardia.

### Entrainment applied to the para-Hisian region

SA-VA and PPI-TCL parameters during entrainment from the right ventricle allow distinction between AVNRT and orthodromic reciprocal tachycardia;<sup>21,22</sup> this also applies when entrainment is performed from the para-Hisian region, the authors of this study<sup>28</sup> used only two right-sided diagnostic catheters, entrainment was performed with and without His bundle capture (a paced QRS widening of 40 msec or more during entrainment compared with QRS width during tachycardia identified the absence of His-bundle capture). SA-VA and PPI-TCL with and without His capture were compared and accordingly, a SA-VA > 75 msec and a PPI-TCL > 100 msec were found to be specific for AVNRT.

### Ventricular extrastimulation

A premature ventricular complex timed with “His” refractoriness during supraventricular tachycardia that

causes a change in subsequent atrial timing indicates that accessory pathway conduction is present and functional, and orthodromic reciprocal tachycardia is the most probable diagnosis<sup>29</sup> (Fig. 6). The VHA (V = local ventricular electrogram, H = “His” and A = atrial electrogram) criterion has been used to differentiate atypical AVNRT (fast/slow) from orthodromic reciprocal tachycardia utilizing a posteroseptal accessory pathway.<sup>30</sup> During ventricular extrastimulation, a “His” bundle potential follows a ventricular potential and is followed by atrial potential when the pathway is via the normal conduction system, whereas a “His” potential may be synchronous or even follow an atrial potential when conduction is via an accessory pathway.

### Conclusion

AVNRT is a relatively common supraventricular arrhythmia, and an accurate diagnosis is essential before proceeding with ablative therapy. Along with specific electrocardiographic clues, specific electrophysiological techniques enable accurate diagnosis of AVNRT. Para-Hisian pacing, “His” refractory extrasystole and entrainment techniques are crucial to differentiate AVNRT from other supraventricular tachycardias, especially orthodromic reciprocal tachycardia with a concealed septal accessory pathway.

### Author Contributions

Conceived and designed the experiments: AK. Analyzed the data: AK. Wrote the first draft of the manuscript: AK, MZ. Contributed to the writing of the manuscript: AK, MZ. Agree with manuscript results and conclusions: AK, MZ. Jointly developed the structure and arguments for the paper: AK, MZ. Made critical revisions and approved final version: AK, MZ. All authors reviewed and approved of the final manuscript.

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contributorship, conflicts of interest, privacy and confidentiality and (where applicable) protection of human and animal research subjects. The authors have read and confirmed their agreement with the ICMJE authorship and conflict of interest criteria. The authors have also confirmed that this article is unique and not under consideration or published in any other publication, and that they have permission from rights holders to reproduce any copyrighted material. Any disclosures are made in this section. The external blind peer reviewers report no conflicts of interest.

## References

1. Lelakowski J, Rydlewska A, Kuniewicz M. Atrioventricular nodal reentrant tachycardia—arrhythmias mechanism, clinical feature and electrocardiographic recordings. *Pol Merkur Lekarski*. 2010;28:429–37. Polish.
2. Kottkamp H, Hindricks G, Borggrefe M, et al. Radiofrequency catheter ablation of the anterosuperior and posteroinferior atrial approaches to the AV node for treatment of AV nodal reentrant tachycardia: techniques for selective ablation of “fast” and “slow” AV node pathways. *J Cardiovasc Electrophysiol*. 1997;8:451–68.
3. Elvas L, GURSOY S, Brugada J, et al. Atrioventricular nodal reentrant tachycardia: a review. *Can J Cardiol*. 1994;10:342–8.
4. Al-Sayegh A, Gondimalla VD, Shukkur AM. Atrioventricular nodal re-entrant tachycardia ablation: unusual function of slow pathway. *Heart Views*. 2011;12:32–4.
5. Katritsis DG. Upper and lower common pathways in atrioventricular nodal reentrant tachycardia: refutation of a legend? *Pacing Clin Electrophysiol*. 2007;30:1305–8.
6. Baszko A, Bobkowski W, Błaszyk k, et al. Atrioventricular nodal tachycardia with left-sided slow pathway treated with RF ablation. *Kardiologia Pol*. 2007;65:1126–30. Polish.
7. Letsas KP, Weber R, Siklody CH, et al. Electrocardiographic differentiation of common type atrioventricular nodal reentrant tachycardia from atrioventricular reciprocating tachycardia via a concealed accessory pathway. *Acta Cardiol*. 2010;65:171–6.
8. Antunes E, Cacula D, Martins S, et al. New criteria for the diagnosis and treatment of atrioventricular node reentrant tachycardia. *Rev Port Cardiol*. 2000;19:771–86. Portuguese.
9. Braunschweig F, Christel P, Jensen-Urstad M, et al. Paroxysmal regular supraventricular tachycardia: the diagnostic accuracy of the transesophageal ventriculo-atrial interval. *Ann Noninvasive Electrocardiol*. 2011;16:327–35.
10. Oh S, Choi YS, Sohn DW, et al. Differential diagnosis of slow/slow atrioventricular nodal reentrant tachycardia from atrioventricular reentrant tachycardia using concealed posteroseptal accessory pathway by 12-lead electrocardiography. *Pacing Clin Electrophysiol*. 2003;26:2296–300.
11. Di Toro D, Hadid C, López C, et al. Utility of the aVL lead in the electrocardiographic diagnosis of atrioventricular node re-entrant tachycardia. *Europace*. 2009;11:944–8.
12. Belhassen B, Fish R, Eldar M, et al. Simplified “ATP test” for noninvasive diagnosis of dual AV nodal physiology and assessment of results of slow pathway ablation in patients with AV nodal reentrant tachycardia. *J Cardiovasc Electrophysiol*. 2000;11:255–61.
13. Lee SH, Chen SA, Tai CT, et al. Electrophysiologic characteristics and radiofrequency catheter ablation in atrioventricular node reentrant tachycardia with second-degree atrioventricular block. *Cardiovasc Electro-physiol*. 1997;8:502–11.
14. Otomo K, Nagata Y, Uno K, et al. Irregular atypical atrioventricular nodal reentrant tachycardia: incidence, electrophysiological characteristics, and effects of slow pathway ablation. *Heart Rhythm*. 2007;4:1507–22.
15. Otomo K, Okamura H, Noda T, et al. Unique electrophysiological characteristics of atrioventricular nodal reentrant tachycardia with different ventriculoatrial block patterns: effects of slow pathway ablation and insights into the location of the reentrant circuit. *Heart Rhythm*. 2006;3:544–54.
16. Haïssaguerre M, Jais P, Shah DC, et al. Analysis of electrophysiological activity in Koch’s triangle relevant to ablation of the slow AV nodal pathway. *Pacing Clin Electrophysiol*. 1997;20:2470–81.
17. Veenhuyzen GD, Quinn FR. Principles of entrainment: diagnostic utility for supraventricular tachycardia. *Indian Pacing Electrophysiol J*. 2008;8:51–65.
18. Padanilam BJ, Manfredi JA, Steinberg LA, et al. Differentiating junctional tachycardia and atrioventricular node re-entry tachycardia based on response to atrial extrastimulus pacing. *J Am Coll Cardiol*. 2008;52:1711–7.
19. Hamdan MH, Badhwar N, Scheinman MM. Role of invasive electrophysiologic testing in the evaluation and management of adult patients with focal junctional tachycardia. *Card Electrophysiol Rev*. 2002;6:431–5.
20. Ormaetxe JM, Almendral J, Arenal A, et al. Ventricular fusion during resetting and entrainment of orthodromic supraventricular tachycardia involving septal accessory pathways. Implications for the differential diagnosis with atrioventricular nodal reentry. *Circulation*. 1993;88:2623–31.
21. Khan AH, Khadem A, Basta MN, et al. Differential entrainment distinguishes atrioventricular nodal reentry tachycardia from atrioventricular reentrant tachycardia. *Pacing Clin Electrophysiol*. 2010;33:1335–41.
22. Segal OR, Gula LJ, Skanes AC, et al. Differential ventricular entrainment: a maneuver to differentiate AV node reentrant tachycardia from orthodromic reciprocating tachycardia. *Heart Rhythm*. 2009;6:493–500.
23. Ho RT, Mark GE, Rhim ES, et al. Differentiating atrioventricular nodal reentrant tachycardia from atrioventricular reentrant tachycardia by DeltaHA values during entrainment from the ventricle. *Heart Rhythm*. 2008;5:83–8.
24. González-Torrecilla E, Arenal A, Aienza F, et al. First postpacing interval after tachycardia entrainment with correction for atrioventricular node delay: a simple maneuver for differential diagnosis of atrioventricular nodal reentrant tachycardias versus orthodromic reciprocating tachycardias. *Heart Rhythm*. 2006;3:674–9.
25. Kannankeril PJ, Bonney WJ, Dzurik MV, et al. Entrainment to distinguish orthodromic reciprocating tachycardia from atrioventricular nodal reentry tachycardia in children. *Pacing Clin Electrophysiol*. 2010;33:469–74.
26. Nakagawa H, Jackman WM. Para-Hisian pacing: useful clinical technique to differentiate retrograde conduction between accessory atrioventricular pathways and atrioventricular nodal pathways. *Heart Rhythm*. 2005;2:667–72.
27. Reddy VY, Jongnarangsin K, Albert CM, et al. Para-Hisian entrainment: a novel pacing maneuver to differentiate orthodromic atrioventricular reentrant tachycardia from atrioventricular nodal reentrant tachycardia. *J Cardiovasc Electrophysiol*. 2003;14:1321–8.
28. Pérez-Rodon J, Bazan V, Bruguera-Cortada J, et al. Entrainment from the para-Hisian region for differentiating atrioventricular node reentrant tachycardia from orthodromic atrioventricular reentrant tachycardia. *Europace*. 2008;10:1205–11.
29. Wagshal AB, Huang SK, Pires LA, et al. Use of double ventricular extrastimulation to determine the preexcitation index in atrioventricular nodal reentrant tachycardia. *Pacing Clin Electrophysiol*. 1995;18:2041–52.
30. Owada S, Iwasa A, Sasaki S, et al. “V-H-A pattern” as a criterion for the differential diagnosis of atypical AV nodal reentrant tachycardia from AV reciprocating tachycardia. *Pacing Clin Electrophysiol*. 2005;28:667–74.