Atrioventricular Nodal Reentrant Tachycardia

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Atrioventricular nodal reentrant tachycardia (AVNRT) represents the most common regular supraventricular arrhythmia in humans.[1] The precise anatomic site and nature of the pathways involved have not yet been established, and several attempts to provide a reasonable hypothesis based on anatomic or anisotropic models have been made.[2] There has been considerable evidence that the right and left inferior extensions of the human atrioventricular (AV) node and the atrionodal inputs they facilitate may provide the anatomic substrate of the slow pathway, and a comprehensive model of the tachycardia circuit for all forms of AVNRT based on the concept of atrionodal inputs has been proposed.[2] Still, however, the circuit of AVNRT remains elusive. Recently, time-honored conventional schemes for the diagnosis and classification of the various forms of the arrhythmia have been refuted in part by evolving evidence. Recognition of the various types of AVNRT is important, however, to expedite diagnosis and allow implementation of appropriate ablation therapy without complications. We present an update on AVNRT with a particular emphasis on electrophysiological criteria used for the differential diagnosis of regular, supraventricular tachycardias.

ECG Presentation

Typically, AVNRT is a narrow-complex tachycardia, ie, QRS duration <120 ms, unless aberrant conduction, which is usually of the right bundle-branch type, or a previous conduction defect exists. Tachycardia-related ST depression and RR-interval variation may be seen. RR alternans in AVNRT has been attributed to the proposed model of a figure of 8 reentry with continuous crossing over of antegrade activation through an inferior input to the contralateral superior input via the node.[2]

In the typical form of AVNRT (slow-fast), abnormal (retrograde) P′ waves are constantly related to the QRS and in the majority of cases are indiscernible or very close to the QRS complex (RP′/RR <0.5). Thus, P′ waves are either masked by the QRS complex or seen as a small terminal P′ wave that is not present during sinus rhythm (Figure 1). In the atypical form of AVNRT (fast-slow), P′ waves are clearly visible before the QRS, ie, RP′/P′R >0.75 (Figure 2), denoting a "long RP tachycardia," and are negative in leads II, III, aVF, and V_6 but positive in V_1. P′ waves are shallow in the inferior leads in the rare form of anterior fast-slow AVNRT.[3] Although AV dissociation is usually not seen, it can occur because neither the atria or the ventricles are necessary for the reentry circuit. If the tachycardia is initiated by atrial ectopic beats, the initial (ectopic) P′ wave usually differs from the subsequent (retrograde) P′ waves (Figure 1).

Electrophysiological Classification of AVNRT Types

The recognition of the fact that AVNRT may present with atypical retrograde atrial activation has made diagnosis of the arrhythmia and classification attempts more complicated. Electrophysiological behavior compatible with multiple pathways may also be seen,[4] and in some patients, several forms of AVNRT may be inducible at electrophysiology study.[3,5] Heterogeneity of both fast- and slow-conduction patterns has been well described, and all forms of AVNRT may display anterior, posterior, and middle retrograde activation patterns (Table).[4,5–7] In certain cases of atypical AVNRT, retrograde atrial activation is even suggestive of a left lateral accessory pathway.[8–10] Appropriate diagnosis in this setting is of importance for the avoidance of prolonged procedures with high fluoroscopy times and unnecessary radiofrequency lesions.[11]

Typical AVNRT

Slow-Fast

In the slow-fast form of AVNRT, the onset of atrial activation appears before, at the onset, or just after the QRS complex, thus maintaining an atrial-His/His-atrial ratio (AH/HA) >1. Although typically, the earliest retrograde atrial activation is recorded at the His bundle electrogram, careful mapping studies have demonstrated that posterior or even left septal fast pathways may occur in up to 7.6% of patients with typical AVNRT.[4,6,7,12,13] The possibility of a bystander atrionodal pathway[12] cannot be ruled out in all cases, but studies of left septal mapping indicate that if left septal His recordings were routinely performed in patients with AVNRT, left-sided retrograde fast pathways would be documented in a considerable proportion of patients studied (Figure 3).[14]

Atypical AVNRT

Fast-Slow

In the fast-slow form of AVNRT (≈5% to 10% of all AVNRT cases), retrograde atrial electrograms begin well

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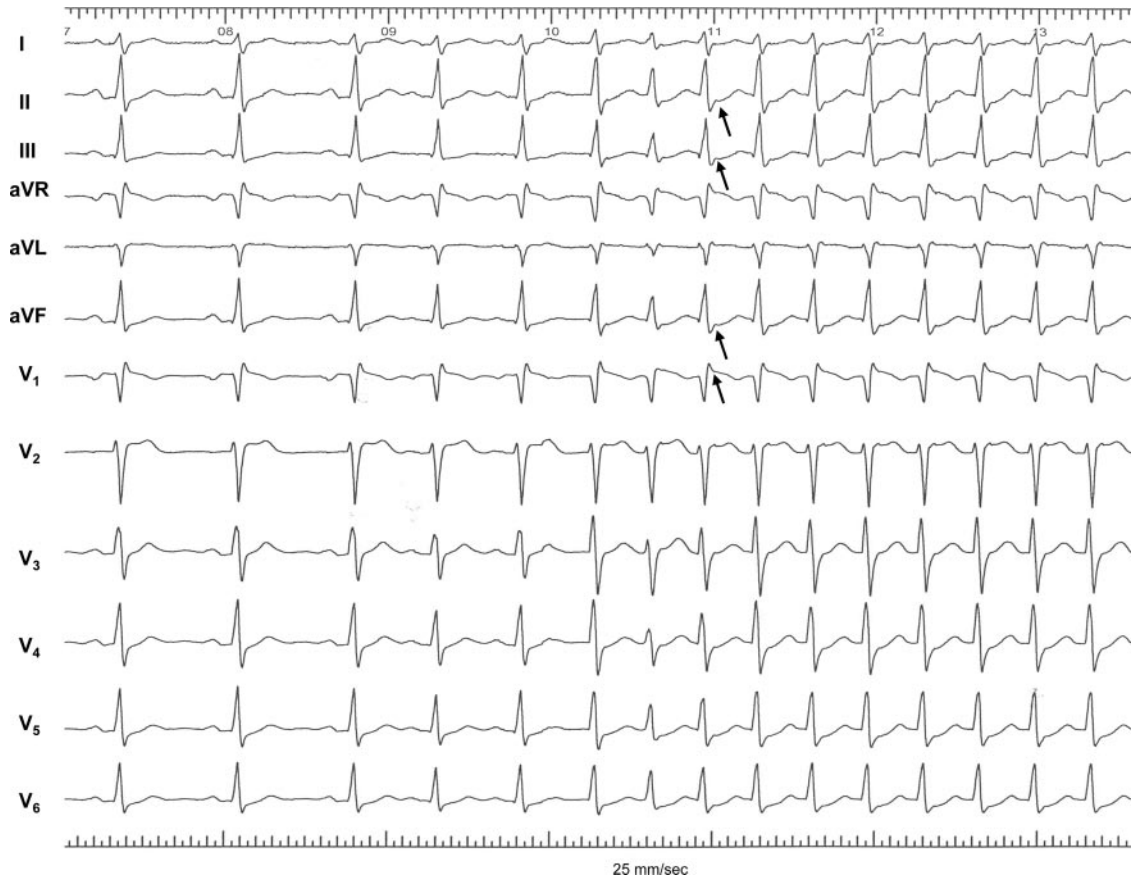

Figure 1. Induction of typical AVNRT by atrial ectopy. The first 3 beats were sinus beats. The next 2 were atrial ectopic beats conducted with a short PR (apparently over the fast pathway). The next atrial ectopic beat was conducted with a prolonged PR (over the slow pathway) and initiated AVNRT. Retrograde P′ waves were more prominent in lead V_1 and especially the inferior leads (arrows).

after ventricular activation, with an AH/HA ratio <1, which indicates that retrograde conduction is slower than antegrade conduction. The VA interval, measured from the onset of ventricular activation on the surface ECG to the earliest deflection of the atrial activation in the His bundle electrogram, is >60 ms, and in the high right atrium, it is >100 ms.[1] Earliest retrograde atrial activation is traditionally reported at the base of the triangle of Koch, near the coronary sinus ostium.[1] Detailed mapping of retrograde atrial activation in large series of patients, however, has produced variable results, with eccentric atrial activation at the lower septum or even the distal coronary sinus.[5–8] Left septal mapping studies have also provided evidence of left-sided earliest retrograde atrial activation during retrograde slow-pathway conduction (Figure 4).[15] Thus, fast-slow AVNRT may be of the posterior, anterior, or middle type according to the mapped location of the retrograde slow pathway.[5]

Slow-Slow

In the slow-slow form, the AH/HA ratio is >1 but the VA interval is >60 ms, which suggests that 2 slow pathways are used for both anterograde and retrograde activation. Earliest retrograde atrial activation is usually at the coronary sinus ostium,[16] but variants of left-sided atrial retrograde activation (distal coronary sinus) have also been reported.[9,10] The so-called posterior or type B AVNRT has been demonstrated in approximately 2% of patients with slow-fast AVNRT; VA times are prolonged, but the AH/HA ratio remains >1.[17] Thus, it appears that the reported cases of posterior slow-fast AVNRT may actually represent the slow-slow form.

Differential Diagnosis

In the presence of a narrow-QRS tachycardia, AVNRT should be differentiated from atrial tachycardia or orthodromic AV reentrant tachycardia (AVRT) due to an accessory pathway. When a wide-QRS tachycardia is encountered and ventricular tachycardia is excluded, the possible diagnoses are AVNRT or atrial tachycardia with aberrant conduction due to bundle-branch block, AVNRT with a bystander accessory pathway, and antidromic AVRT due to an accessory pathway. Aberrant conduction, although rare, can be seen in AVNRT and is usually of the right bundle-branch type; however, cases of left bundle-branch block have been reported.

AVNRT Versus Atrial Tachycardia

Differential diagnosis may be difficult particularly with peri-AV nodal atrial tachycardias that, when reentrant, usually respond to adenosine and verapamil.[17a]

ECG Criteria

Exclusion of an atrial tachycardia on the 12-lead ECG is difficult, and pacing maneuvers are necessary for this purpose.

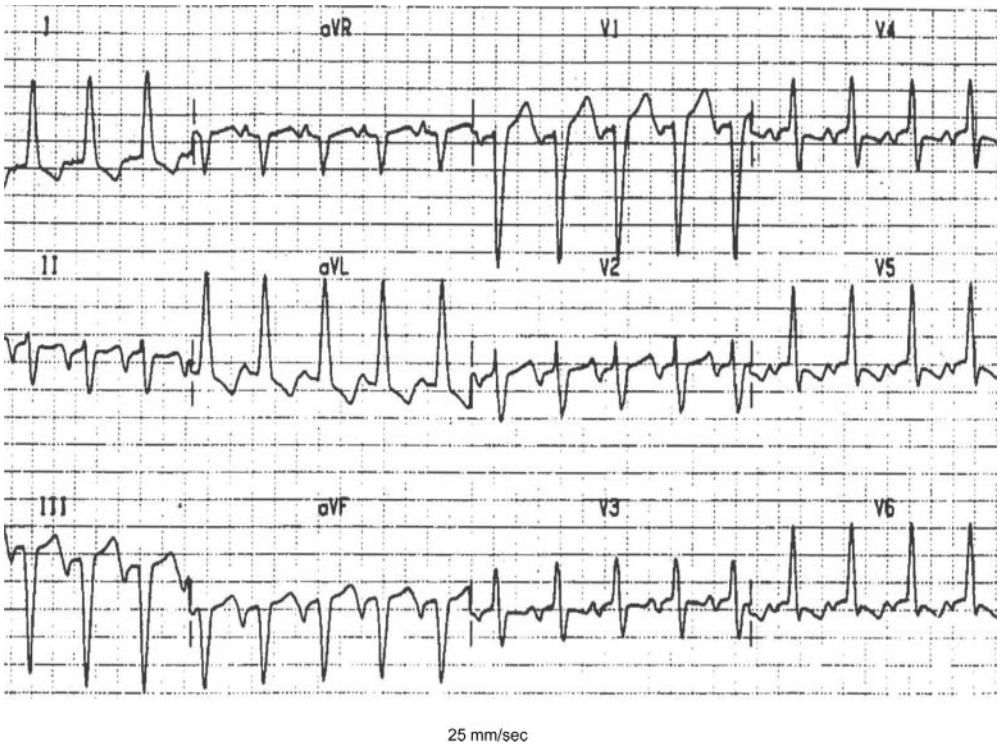

Figure 2. Example of a long RP tachycardia. This was a case of incessant tachycardia due to fast-slow AVNRT.

Electrophysiology Study

Atrial Pacing

The demonstration of a constant His-atrial interval of the return cycle after introduction of a premature atrial impulse with a wide range of coupling intervals during tachycardia makes the diagnosis of atrial tachycardia unlikely. In particular, the difference in the AH interval between atrial pacing and the tachycardia is >40 ms in AVNRT but <10 ms in atrial tachycardia.[18] Differential atrial overdrive pacing from the high right atrium and the coronary sinus has also been proposed for diagnosis of atrial tachycardia versus slow-fast AVNRT and AVRT. A difference of >10 ms in the first V-A time after atrial overdrive from the high right atrium and the coronary sinus suggests atrial tachycardia.[19]

Table. Classification of AVNRT Types*

	AH/HA	VA (His)	Usual ERAA
Typical AVNRT			
Slow-fast	>1	<60 ms	RHis, CS os, LHis
Atypical AVNRT			
Fast-slow	<1	>60 ms	CS os, LRAS, dCS
Slow-slow	>1	>60 ms	CS os, dCS

VA indicates interval measured from the onset of ventricular activation on surface ECG to the earliest deflection of the atrial activation in the His bundle electrogram; ERAA, earliest retrograde atrial activation; RHis, His bundle electrogram recorded from the right septum; LHis, His bundle electrogram recorded from the left septum; LRAS, low right atrial septum; CS os, ostium of the coronary sinus; and dCS, distal coronary sinus.

*Variable earliest retrograde atrial activation has been described for all types of AVNRT.

Ventricular Pacing

Tachycardia termination by a ventricular extrastimulus that did not conduct to the atrium indicates an origin other than atrial tachycardia. A useful criterion is the atrial response on cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction during tachycardia. Atrial tachycardia is associated with an A-A-V response, whereas AVNRT or AVRT produces an A-V response.[20] This rule, however, has many exceptions. The His deflection should be considered in this respect, because a late V electrogram might give an apparent A-A-V response that is actually an A-H-A-V response in the presence of AVNRT or AVRT, as opposed to A-A-H-V in the presence of atrial tachycardia.[21] Apart from a long HV interval during slow-fast AVNRT that may cause atrial activation to precede ventricular activation, a very long AH interval that exceeds the tachycardia cycle length may also produce a pseudo A-A-V response.[22] A pseudo A-V response can also be seen with isoprenaline-increased automaticity of the AV junction in the sense that junctional activity precludes conduction of the second A of the A-A-V response to the ventricle.[23]

AVNRT Versus AVRT Due to Septal Accessory Pathways

AVNRT may display eccentric atrial activation, and septal pathways may have decremental conduction properties and concentric atrial activation, thus making the differential diagnosis challenging.

ECG Criteria

In the case of relatively delayed retrograde conduction that allows the identification of retrograde P waves, ECG criteria can

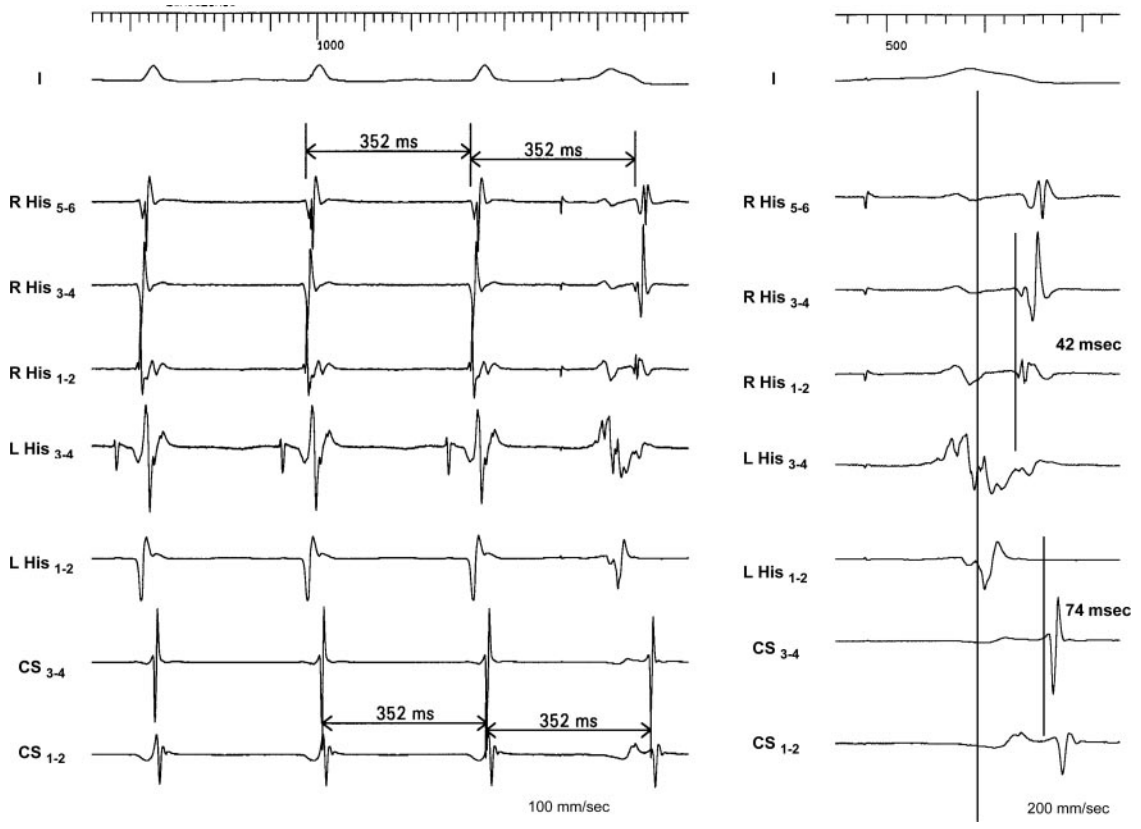

Figure 3. Example of earliest retrograde atrial activation on the left septum during slow-fast AVNRT. Left septal retrograde atrial activation preceded atrial activation recorded on the right septum and coronary sinus ostium by 42 and 74 ms, respectively. An RV extrastimulus captured the ventricle without resetting the tachycardia circuit (as judged by constant A-A intervals), resulted in separation of ventricular and atrial electrograms, and allowed identification of the beginning of the atrial electrogram on the left His recording electrodes. Recordings were at 100 mm/s in the left panel and 200 mm/s in the right panel. I indicates lead I of the surface ECG; R His, right His bundle recording electrode; His, left His bundle recording electrode; and CS, coronary sinus. Reproduced from Katritsis et al[14] with permission of the publisher. Copyright © 2006, the Heart Rhythm Society.

be applied for diagnosis. The presence of a pseudo r′ wave in lead V_1 or a pseudo S wave in leads II, III, and aVF has been reported to indicate anterior AVNRT with an accuracy of 100%. A difference of >20 ms in RP intervals in leads V_1 and III was indicative of posterior AVNRT rather than AVRT due to a posteroseptal pathway.[24] The documentation of preexcited beats, AV dissociation, and the induction of bundle-branch block during tachycardia may assist the differential diagnosis. AV block or AV dissociation, although uncommon and short-lasting, may be seen in AVNRT and excludes the presence of an accessory pathway.[1] Similarly, the development of bundle-branch block either spontaneously or after introduction of ventricular extrastimuli during AVNRT does not change the AA or HH intervals. A significant change in the VA interval with the development of bundle-branch block is diagnostic of orthodromic AVRT and localizes the pathway to the same side as the block.[1]

Electrophysiology Study

Atrial Pacing

AH Interval

The difference in the AH interval between atrial pacing and the tachycardia may allow differentiation of atypical AVNRT from other types of long RP′ tachycardias. A ΔAH >40 ms has been reported to indicate AVNRT, whereas in patients with AVRT due to septal pathways or atrial tachycardia, these differences were <20 and 10 ms, respectively.[18]

Ventricular Pacing

His-Synchronous Ventricular Extrastimulation

In the presence of septal decremental pathways, ventricular extrastimuli introduced while the His bundle is refractory during tachycardia (ie, delivered coincident with the His potential or up to 50 ms before this) may advance or delay subsequent atrial activation (extranodal capture). In AVNRT, atrial activity is not perturbed with His-synchronous ventricular extrastimulation. Failure to reset the atria suggests but does not prove that an accessory pathway is not present or that it is relatively far from the site of premature stimulation. Demonstration of resetting excludes AVNRT if extrastimuli are delivered very close to inferior nodal extensions.[25]

VA Intervals

Atrial and ventricular conduction starts almost simultaneously in AVNRT, and VA times during tachycardia are therefore shorter than during ventricular pacing. The difference between the tachycardia cycle length and the longest

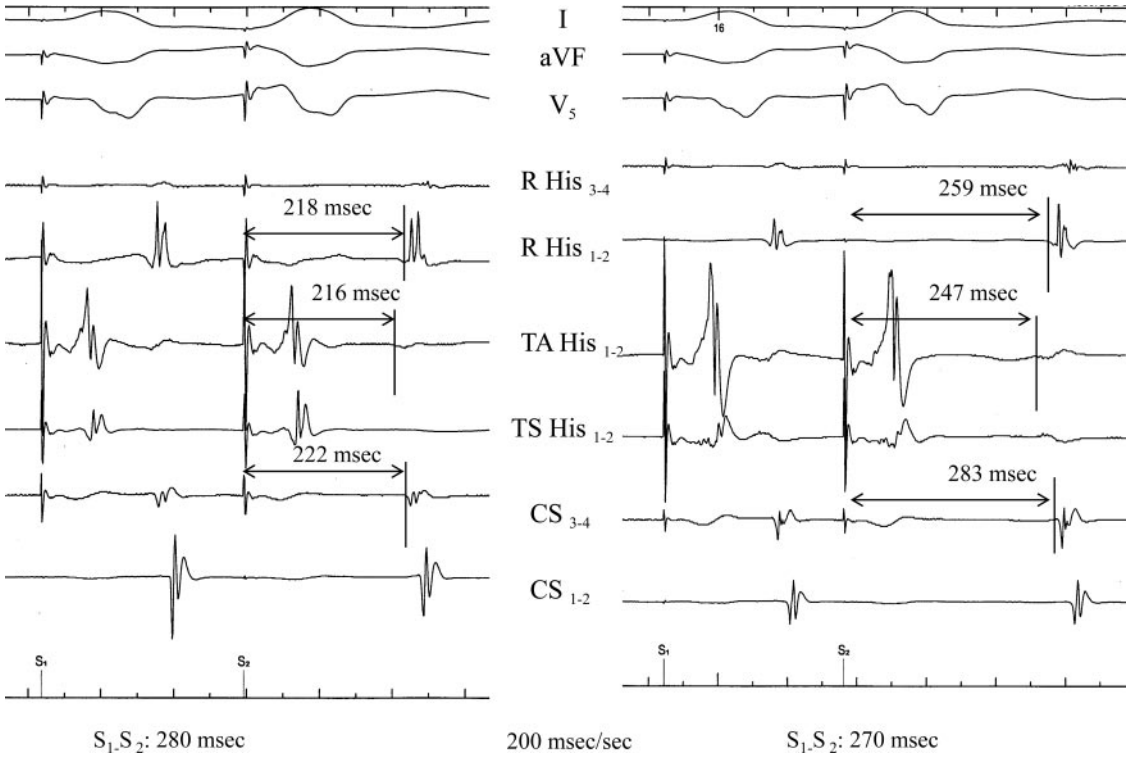

Figure 4. Left septal earlier atrial activation during retrograde conduction over the slow pathway. A shift of earliest retrograde atrial activation sequence at retrograde conduction jump is noted. During constant ventricular pacing, atrial activation on the right His preceded that recorded at other His catheters. At decremental retrograde conduction (S1−S2=280 ms), the left (transaortic) atrial activation slightly preceded that of the right (216 vs 218 ms; left panel). At retrograde conduction jump (S1−S2=270 ms), earliest atrial activation was recorded on the left septum by the TA electrode and preceded that of the right His by 12 ms (259 to 247 ms; right panel). Recordings were at 200 mm/s. I indicates lead I of the surface ECG; II, lead II of the surface ECG; R His, right His bundle recording electrode; TA His, transaortic left His bundle recording; TS His, transseptal left His bundle recording; and CS, coronary sinus. Reproduced from Katritsis et al[15] with permission of the publisher. Copyright © 2007, the European Society of Cardiology and the European Heart Rhythm Society.

premature ventricular stimulation interval at which atrial capture occurs during tachycardia defines the so-called preexcitation index.[26] A preexcitation index of 100 ms or more characterizes AVNRT, whereas an index less than 45 ms characterizes AVRT with a septal pathway. Consideration of the ratio between the minimum ventriculoatrial interval during tachycardia and during ventricular pacing in sinus rhythm is more practical. Ratios of 0.32 to 0.27 indicate AVNRT, 0.91 to 1.08 indicate AVRT with a posteroseptal pathway, and 0.94 to 1.29 indicate AVRT with an anteroseptal pathway.[27] A difference in the VA interval during tachycardia and apical right ventricular (RV) pacing >90 ms has been reported to differentiate patients with AVNRT from those with AVRT.[28] VA times obtained by apical RV pacing can be misleading in the case of multiple accessory pathways or simultaneous nodal and accessory pathway retrograde conduction. The difference between the ventriculoatrial interval obtained during apical pacing and that obtained during posterobasal pacing has been found to discriminate between patients with posteroseptal pathways (>10 ms) and those with nodal retrograde conduction (<5 ms).[29] Parahisian pacing during sinus rhythm and the change in timing and sequence of retrograde atrial activation between His and proximal right bundle-branch capture and noncapture have also been used for differentiation between AV nodal and septal pathway retrograde conduction.[30] The response is considered extranodal when the retrograde atrial activation during His bundle capture is the same as during ventricular capture without His bundle capture. Parahisian pacing techniques are cumbersome and may not be possible in up to 50% of tachycardia cases that are studied.[31]

VH Conduction

The demonstration of a V-H-A response at ventricular stimulation that is performed during sinus rhythm indicates retrograde conduction through the AV node and has been used to differentiate atypical AVNRT from AVRT.[32] However, a V-H-A pattern cannot rule out a fast-slow AVNRT, and an accessory pathway with conduction slower than that of the AV node may produce a V-H-A pattern. Recently, the induction of retrograde right bundle-branch type (defined as an increase of the VH interval >50 ms) by ventricular extrastimulation during tachycardia has been proposed for differential diagnosis purposes.[31] Similar increases in the VH and VA intervals indicated retrograde AV nodal conduction, whereas a VH interval change that exceeded that of the VA interval indicated the presence of an accessory pathway because it bypassed the decremental conduction system. This maneuver provides very high sensitivity and specificity (almost 100%) but requires recording of retrograde His bundle activation.

HA Intervals

The difference between HA intervals during pacing and during tachycardia (ΔHA) has also been used. In a study of 84 patients, a retrograde His bundle deflection was present in 93% of them, and the ΔHA was >0 ms in AVNRT and less than −27 ms in orthodromic AVRT that incorporated a septal accessory pathway. Thus, an intermediate value of ΔHA=−10 ms had 100% sensitivity, specificity, and predictive accuracy in differentiating the 2 forms of tachycardia.[33]

Tachycardia Entrainment

The use of tachycardia entrainment via ventricular pacing for differential diagnosis of supraventricular tachycardias was introduced by Ormaetxe et al.[34] Right apical stimulation is relatively close to the insertion of a septal accessory pathway as opposed to the AV junction; thus, ventricular fusion during resetting or entrainment of tachycardia has been reported to occur in patients with AVRT due to septal pathways but not with AVNRT.[34] Michaud et al[35] have proposed 2 additional criteria for differential diagnosis. The ventriculoatrial (VA) interval and tachycardia cycle length (TCL) were measured during tachycardia, and entrainment of the tachycardia was accomplished with RV apical pacing. The intervals between the last ventricular pacing stimulus and the last entrained atrial depolarization during tachycardia (SA), as well as the postpacing interval (PPI), were considered. All patients with AVNRT had SA-VA intervals >85 ms and PPI-TCL intervals >114 ms. The SA-VA and PPI-TCL criteria should be valid regardless of the type of AVNRT (slow-fast or fast-slow). However, conventional entrainment techniques do not take into account pacing-induced incremental AV nodal conduction (ie, in the postpacing A-H) that may alter the PPI. Thus, González-Torrecilla et al[36] "corrected" the PPI-TCL difference by subtracting from it the difference of postpacing AH interval minus basic AH interval. The presence of a corrected PPI-TCL <110 ms indicated AVRT. Entrainment through basal RV pacing away from the septum may produce prolonged PPI-TCL intervals in the absence of a septal pathway owing to the distance of the RV base from the AV node (activation occurs retrogradely through the distal His-Purkinje system) and has been found to be superior to apical entrainment for diagnostic purposes.[37] Recently, differential entrainment from the apex or the basal area of the RV was investigated.[38] A differential (between base and apex) corrected PPI-TCL >30 ms or a differential VA interval >20 ms predicted AVNRT very reliably (Figure 5). The main advantage of this technique is that the differential VA interval could be calculated from the last paced beat in case the tachycardia was terminated after transient entrainment.

The comparison of HA interval during tachycardia and during entrainment from the RV apex or near the His bundle to obtain a recordable retrograde His bundle deflection has also been used for differentiation of AVNRT from AVRT. Positive ΔHA (HA during entrainment minus HA during SVT) indicates AVNRT, whereas negative ΔHA indicates the presence of an accessory pathway.[39]

Parahisian entrainment that considered the difference in stimulus to earliest retrograde atrial activation during His and both RV and His capture has also been reported.[40] Assessment of the SA-VA and PPI-TCL intervals by parahisian entrainment may assist in differentiation between slow-fast AVNRT and AVRT, but the cutoff values are lower than that obtained with RV apical pacing (75 and 100 ms, respectively), presumably owing to conduction through the septum.[41] These techniques are rather cumbersome, requiring differential capture and both recording and identification of retrograde His and local ventricular potentials.

In many studies, proposed criteria have been examined in patients with typical slow-fast AVNRT; their effectiveness in differentiating between fast-slow AVNRT and decremental accessory pathways has not been tested. In clinical practice, pacing or other maneuvers cannot be applied in all cases, and multiple criteria must be used for the differential diagnosis of narrow-complex tachycardias with atypical characteristics.[42]

AVNRT With Bystanding Accessory Pathways Versus Antidromic AVRT

Antidromic AVRT, ie, tachycardia that uses the accessory pathway for antegrade conduction and the AV node for retrograde conduction, may be induced in approximately 6% of patients with accessory pathways located in the left or right free wall or the anterior septum at an adequate distance from the AV node.[1] Mahaim atriofascicular pathways may give rise to AVRT or act as bystanders in AVNRT with left bundle-branch block morphology.

Electrophysiology Study

The possibility of AVNRT that is conducted over a bystander accessory pathway should be considered in the presence of transition from narrow- to wide-complex tachycardia of a similar cycle length and without disruption of the HH intervals.[43] In this case, atrial extrastimuli fail to induce advancement of the following preexcited QRS complex, the next retrograde His bundle deflection where apparent, and the subsequent atrial deflection without changing the retrograde atrial activation sequence, as may happen in the presence of a macroreentrant loop (Figure 6). Although no extensive series have been published, application of entrainment criteria (SA-VA and PPI-TCL) from the apex[44] or the apex and the base for differential entrainment[45] is theoretically appealing and has been proposed to distinguish between antidromic AVRT and AVNRT with a bystander fiber.

AVNRT Versus Nonreentrant Junctional Tachycardias

Nonreentrant junctional tachycardias are rare but should be recognized, because catheter ablation conveys a higher risk of AV block than in AVNRT. Nonparoxysmal junctional tachycardia was frequently diagnosed in the past as a junctional rhythm of gradual onset and termination with a rate between 70 and 130 bpm and was considered a typical example of digitalis-induced delayed afterdepolarizations and triggered activity in the AV node.[1] Presently, focal junctional

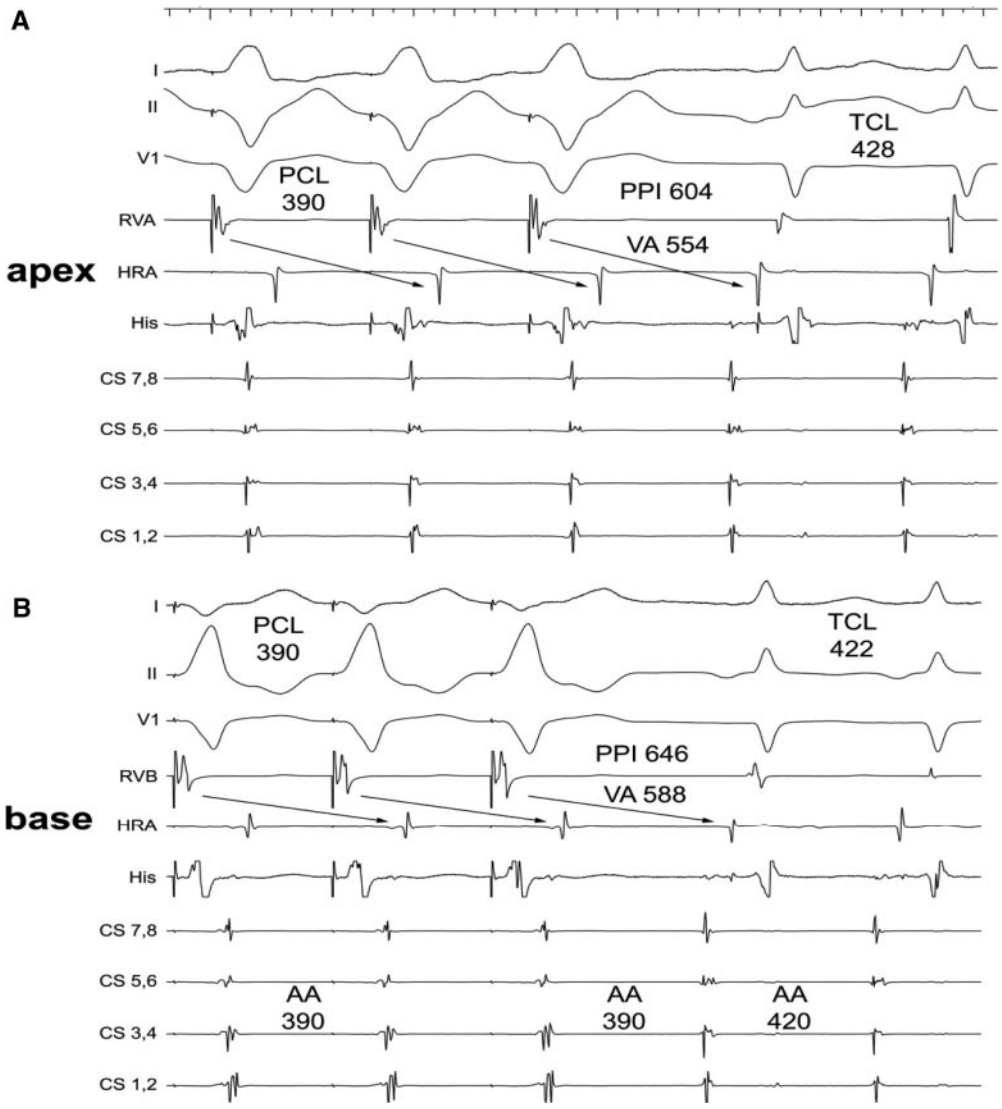

Figure 5. Examples of differential entrainment responses in atypical fast-slow AVNRT. Entrainment of atypical AVNRT from RV apex (RVA) at 390 ms (A) and from RV base at 390 ms (B). Differential PPI-TCL and VA intervals were 42 and 34 ms, respectively. Panels are arranged from top to bottom as ECG leads I, II, and V1, and bipolar electrograms from the RVA, high right atrium (HRA), His bundle (His), or RV base (RVB) and coronary sinus (CS) proximal (7,8) to distal (1,2). Pacing cycle length (PCL), PPI, TCL, and VA interval are shown. The PPI was measured from the last pacing stimulus to the return cycle electrogram (RVA or RVB), and the TCL was deducted to produce the PPI-TCL. The VA interval was measured from the last pacing stimulus to the last entrained HRA electrogram. Reproduced from Segal et al[38] with permission of the publisher. Copyright © 2009, the Heart Rhythm Society.

tachycardia occurs as a congenital arrhythmia or early after infant open heart surgery, but it can also be seen in adult patients, and this tachycardia is associated with a structurally normal heart.[46,47] Its cause is increased automaticity or triggered activity.[47] The usual ECG finding is a narrow QRS tachycardia with AV dissociation. Occasionally, the tachycardia might be irregular, thus resembling atrial fibrillation. At electrophysiology study, there is a normal HV interval and normal AV conduction curves.[46] The arrhythmia is not inducible by programmed electric stimulation but is sensitive to isoproterenol administration, and in some cases, rapid atrial or ventricular pacing may result in tachycardia induction. During tachycardia, there is a normal or increased HV interval with AV dissociation that is interrupted by frequent episodes of ventriculoatrial conduction, with earliest atrial activation in the posteroseptal, anteroseptal, or midseptal regions. At times, the mode of tachycardia induction resembles a double AV nodal response that is characteristic of AVNRT.[47]

The differentiation of AVNRT from junctional tachycardia is of clinical importance, because ablation of the latter is associated with an increased risk of AV block. A positive ΔHA interval (ie, HA during pacing from a basal site of the RV minus HA during tachycardia) has been reported to indicate junctional tachycardia. In a recent study, the average ΔHA interval in AVNRT was −10 ms, although positive values were encountered.[48] Padanilam et al[49] described the use of premature atrial stimuli for differential diagnosis of slow-fast AVNRT and nonreentrant junctional tachycardia. An atrial premature complex that is timed to His refractoriness and advances the His electrogram of the subsequent cycle indicates that

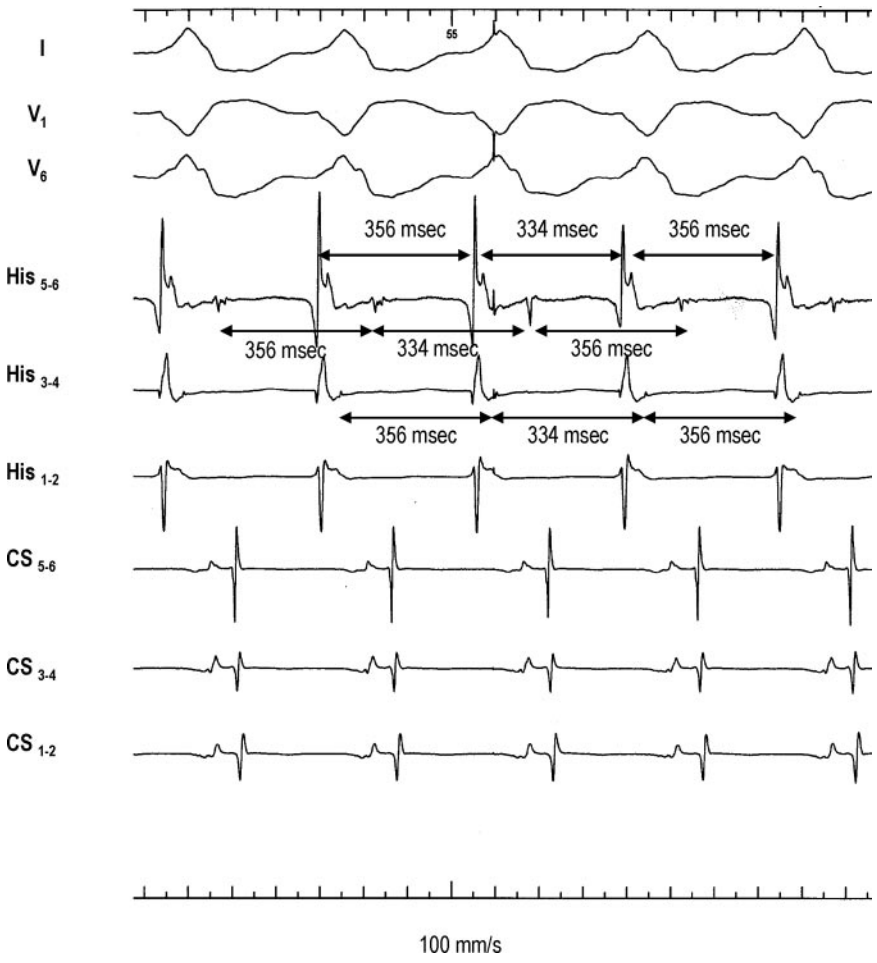

Figure 6. Resetting of AVRT due to an atriofascicular Mahaim pathway by atrial extrastimuli. Note the advancement of ventricular, retrograde His, and atrial electrograms by 22 ms without an effect on retrograde activation sequence. I, V1, and V6 indicates surface ECG leads; His, His bundle; and CS, coronary sinus.

anterograde slow-pathway conduction is involved and suggests the diagnosis of AVNRT. An atrial extrastimulus that advances the His potential immediately after it without terminating the tachycardia indicates that the retrograde fast pathway is not essential for the circuit and suggests the diagnosis of junctional tachycardia. If no dual (or multiple) response of the AV node is present, this criterion has a high specificity.

Nonreentrant AV nodal tachycardia caused by simultaneous multiple nodal pathway conduction is an uncommon mechanism of AV nodal tachycardia and has been associated with repetitive retrograde concealment or "linking" phenomena.[50] These are expressed in the form of ventricular pauses with a consistent AV relationship after the pause.

Therapy

In acute episodes of AVNRT that do not respond to Valsalva maneuvers, intravenous adenosine is the treatment of choice. Continuous administration of antiarrhythmic drugs may be ineffective in up to 70% of cases.[51] Thus, catheter ablation is the current treatment of choice. Slow-pathway ablation or modification is effective in both typical and atypical AVNRT. Usually, a combined anatomic and mapping approach is used, with ablation lesions delivered at the inferior or mid part of the triangle of Koch either from the right or the left septal side.[52] Multicomponent atrial electrograms or low-amplitude potentials, although not specific for identification of slow-pathway conduction, are used to successfully guide ablation at these areas. This approach is associated with a 0.5% to 1% risk of AV block and has a recurrence rate of approximately 4%. Advanced age is not a contraindication for slow pathway ablation.[53] The preexistence of first-degree heart block may carry a higher risk for late AV block and slow pathway modification, as opposed to complete elimination, is probably preferable in this setting.[54]

Disclosures

None.

References

1. Katritsis DG, Camm AJ. Classification and differential diagnosis of atrioventricular nodal re-entrant tachycardia. *Europace*. 2006;8:29–36.
2. Katritsis DG, Becker A. The circuit of atrioventricular nodal reentrant tachycardia: a proposal. *Heart Rhythm*. 2007;4:1354–1360.
3. Nawata H, Yamamoto N, Hirao K, Miyasaka N, Kawara T, Hiejima K, Harada T, Suzuki F. Heterogeneity of anterograde fast-pathway and retrograde slow-pathway conduction patterns in patients with the fast-slow form of atrioventricular nodal reentrant tachycardia: electrophysiologic and electrocardiographic considerations. *J Am Coll Cardiol*. 1998;32:1731–1740.
4. Heinroth KM, Kattenbeck K, Stabenow I, Trappe HJ, Weismuller P. Multiple AV nodal pathways in patients with AV nodal reentrant tachycardia: more common than expected? *Europace*. 2002;4:375–382.

5. Engelstein ED, Stein KM, Markowitz SM, Lerman BB. Posterior fast atrioventricular node pathways: implications for radiofrequency catheter ablation of atrioventricular node reentrant tachycardia. *J Am Coll Cardiol*. 1996;27:1098–1105.
6. Chen J, Anselme F, Smith TW, Zimetbaum P, Epstein LM, Papageorgiou P, Josephson ME. Standard right atrial ablation is effective for atrioventricular nodal reentry with earliest activation in the coronary sinus. *J Cardiovasc Electrophysiol*. 2004;15:2–7.
7. Nam G-B, Rhee K-S, Kim J, Choi K-J, Kim Y-H. Left atrionodal connections in typical and atypical atrioventricular nodal re-entrant tachycardias: activation sequence in the coronary sinus and results of radiofrequency catheter ablation. *J Cardiovasc Electrophysiol*. 2006; 17:1–7.
8. Hwang C, Martin DJ, Goodman JS, Gang ES, Mandel WJ, Swerdlow CD, Peter CT, Chen PS. Atypical atrioventricular node reciprocating tachycardia masquerading as tachycardia using a left-sided accessory pathway. *J Am Coll Cardiol*. 1997;30:218–225.
9. Sakabe K, Wakatsuki T, Fujinaga H, Oishi Y, Ikata J, Toyoshima T, Hiura N, Nishikado A, Oki T, Ito S. Patient with atrioventricular node reentrant tachycardia with eccentric retrograde left-sided activation: treatment with radiofrequency catheter ablation. *Jpn Heart J*. 2000;41: 227–234.
10. Vijayaraman P, Kok LC, Rhee B, Ellenbogen KA. Unusual variant of atrioventricular nodal re-entrant tachycardia. *Heart Rhythm*. 2004;2: 100–102.
11. Goldberg JJ. Unraveling the mysteries of the AV node. *Heart Rhythm*. 2006;3:1001–1002.
12. Anselme F, Papageorgiou P, Monahan K, Zardini M, Boyle N, Epstein LM, Josephson ME. Presence and significance of the left atrionodal connection during atrioventricular nodal reentrant tachycardia. *Am J Cardiol*. 1999;83:1530–1536.
13. Ong MG, Lee PC, Tai CT, Lin YJ, Hsieh MH, Chen YJ, Lee KT, Tsao HM, Kuo JY, Chang SL, Chen SA. The electrophysiologic characteristics of atrioventricular nodal reentry tachycardia with eccentric retrograde activation. *Int J Cardiol*. 2007;120:115–122.
14. Katritsis DG, Ellenbogen KA, Becker AE. Atrial activation during atrioventricular nodal reentrant tachycardia: studies on retrograde fast pathway conduction. *Heart Rhythm*. 2006;3:993–1000.
15. Katritsis D, Ellenbogen K, Becker A, Camm AJ. Retrograde slow pathway conduction in patients with atrioventricular nodal reentrant tachycardia. *Europace*. 2007;9:458–465.
16. Goldberger J, Brooks R, Kadish A. Physiology of "atypical" atrioventricular junctional reentrant tachycardia occurring following radiofrequency catheter modification of the atrioventricular node. *Pacing Clin Electrophysiol*. 1992;15:2270–2282.
17. McGuire MA, Lau K-C, Johnson DC, Richards DA, Uther JB, Ross DL. Patients with two types of atrioventricular junctional (AV nodal) reentrant tachycardia: evidence that a common pathway of nodal tissue is not present above the reentrant circuit. *Circulation*. 1991;83:1232–1246.

17a. Yamabe H, Tanaka Y, Morihisa K, Uemura T, Enomoto K, Kawano H, Ogawa H. Analysis of the anatomical tachycardia circuit in verapamil-sensitive atrial tachycardia originating from the vicinity of the atrioventricular node. *Circ Arrhythm Electrophysiol*. 2010;3:54–62.

18. Man KC, Niebauer M, Daoud E, Strickberger SA, Kou W, Williamson BD, Morady F. Comparison of atrial-His intervals during tachycardia and atrial pacing in patients with long RP tachycardia. *J Cardiovasc Electrophysiol*. 1995;6:700–710.
19. Sarkozy A, Richter S, Chierchia GB, De Asmundis C, Seferlis C, Brugada P, Kaufman L, Buyl R, Dorian P, Mangat I. A novel pacing manoeuvre to diagnose atrial tachycardia. *Europace*. 2008;10:459–466.
20. Knight BP, Zivin A, Souza J, Flemming M, Pelosi F, Goyal R, Man C, Strickberger SA, Morady F. A technique for the rapid diagnosis of atrial tachycardia in the electrophysiology laboratory. *J Am Coll Cardiol*. 1999; 33:775–781.
21. Vijayaraman P, Kok LC, Rhee B, Ellenbogen KA. Wide complex tachycardia: what is the mechanism? *Heart Rhythm*. 2004;2:107–109.
22. Crawford TC, Morady F, Pelosi F Jr. A long R-P paroxysmal supraventricular tachycardia: what is the mechanism? *Heart Rhythm*. 2007;4: 1364–1365.
23. Kalra D, Morady F. Supraventricular tachycardia: what is the mechanism? *Heart Rhythm*. 2008;5:1219–1220.
24. Tai CT, Chen SA, Chiang CE, Lee SH, Wen ZC, Chiou CW, Ueng KC, Chen YJ, Yu WC, Chang MS. A new electrocardiographic algorithm using retrograde P waves for differentiating atrioventricular node reentrant tachycardia from atrioventricular reciprocating tachycardia mediated by concealed accessory pathway. *J Am Coll Cardiol*. 1997;29: 394–402.
25. Katritsis DG, Becker AE, Ellenbogen KA, Giazitzoglou E, Korovesis S, Camm AJ. Effect of slow pathway ablation in atrioventricular nodal reentrant tachycardia on the electrophysiologic characteristics of the inferior atrial inputs to the human atrioventricular node. *Am J Cardiol*. 2006;97:860–865.
26. Miles WM, Yee R, Klein GJ, Zipes DP, Prystowsky EN. The preexcitation index: an aid in determining the mechanism of supraventricular tachycardia and localizing accessory pathways. *Circulation*. 1986;74: 493–500.
27. Crozier I, Wafa S, Ward D, Camm J. Diagnostic value of comparison of ventriculoatrial interval during junctional tachycardia and right ventricular apical pacing. *Pacing Clin Electrophysiol*. 1989;12:942–953.
28. Tai CT, Chen SA, Chiang CE, Chang MS. Characteristics and radiofrequency catheter ablation of septal accessory atrioventricular pathways. *Pacing Clin Electrophysiol*. 1999;22:500–511.
29. Martinez-Alday JD, Almendral J, Arenal A, Ormaetxe JM, Pastor A, Villacastin JP, Medina O, Peinado R, Delcan JL. Identification of concealed posteroseptal Kent pathways by comparison of ventriculoatrial intervals from apical and posterobasal right ventricular sites. *Circulation*. 1994;89:1060–1067.
30. Hirao K, Otomo K, Wang X, Beckman KJ, McClelland JH, Widman L, Gonzalez MD, Arruda M, Nakagawa H, Lazzara R, Jackman WM. Para-Hisian pacing: a new method for differentiating retrograde conduction over an accessory AV pathway from conduction over the AV node. *Circulation*. 1996;94:1027–1035.
31. Kapa S, Henz BD, Dib C, Cha YM, Friedman PA, Munger TM, Ladewig DJ, Hammill SC, Packer DL, Asirvatham SJ. Utilization of retrograde right bundle branch block to differentiate atrioventricular nodal from accessory pathway conduction. *J Cardiovasc Electrophysiol*. 2009;20: 751–758.
32. Owada S, Iwasa A, Sasaki S, Higuma T, Kimura M, Kobayashi T, Ashikaga K, Okumura K. "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;28:667–674.
33. Miller JM, Rosenthal ME, Gottlieb CD, Vassalo JA, Josephson ME. Usefulness of the ΔHA interval to accurately distinguish atrioventricular nodal reentry from orthodromic septal bypass tract tachycardias. *Am J Cardiol*. 1991;68:1037–1044.
34. Ormaetxe JM, Almendral J, Arenal A, Martinez-Alday JD, Pastor A, Villacastin JP, Delcan JL. 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;88:2623–2631.
35. Michaud GF, Tada H, Chough S, Baker R, Wasmer K, Sticherling C, Oral H, Pelosi F Jr, Knight BP, Strickberger SA, Morady F. Differentiation of atypical atrioventricular node re-entrant tachycardia from orthodromic reciprocating tachycardia using a septal accessory pathway by the response to ventricular pacing. *J Am Coll Cardiol*. 2001;38:1163–1167.
36. González-Torrecilla E, Arenal A, Atienza F, Osca J, García-Fernández J, Puchol A, Sánchez A, Almendral J. 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;3:674–679.
37. Veenhuyzen GD, Coverett K, Quinn FR, Sapp JL, Gillis AM, Sheldon R, Exner DV, Mitchell LB. Single diagnostic pacing maneuver for supraventricular tachycardia. *Heart Rhythm*. 2008;5:1152–1158.
38. Segal OR, Gula LJ, Skanes AC, Krahn AD, Yee R, Klein GJ. Differential ventricular entrainment: a maneuver to differentiate AV node reentrant tachycardia from orthodromic reciprocating tachycardia. *Heart Rhythm*. 2009;6:493–500.
39. Ho RT, Mark GE, Rhim ES, Pavri BB, Greenspon AJ. Differentiating atrioventricular nodal reentrant tachycardia from atrioventricular reentrant tachycardia by DeltaHA values during entrainment from the ventricle. *Heart Rhythm*. 2008;5:83–88.
40. Reddy VY, Jongnarangsin K, Albert CM, Sabbour H, Keane D, Mela T, McGovern B, Ruskin JN. Para-Hisian entrainment: a novel pacing maneuver to differentiate orthodromic atrioventricular reentrant tachycardia from atrioventricular nodal re-entrant tachycardia. *J Cardiovasc Electrophysiol*. 2003;14:1321–1328.
41. Pérez-Rodon J, Bazan V, Bruguera-Cortada J, Mojal-García S, Manresa-Domínguez JM, Martí-Almor J. Entrainment from the para-Hisian region for differentiating atrioventricular node reentrant tachycardia from ortho-

dromic atrioventricular reentrant tachycardia. *Europace*. 2008;10:1205–1211.
42. Knight BP, Ebinger M, Oral H, Kim MH, Sticherling C, Pelosi F, Michaud GF, Strickberger SA, Morady F. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia. *J Am Coll Cardiol*. 2000;36:574–582.
43. Smith WM, Broughton A, Reiter MJ, Benson DW, Grant AO, Gallagher JJ. Bystander accessory pathway during AV node reentrant tachycardia. *Pacing Clin Electrophysiol*. 1983;6:537–543.
44. Quinn FR, Mitchell LB, Mardell A, Veenhuyzen GD. Overdrive ventricular pacing to clarify the diagnosis of a left bundle branch block morphology tachycardia. *Heart Rhythm*. 2008;5:1479–1481.
45. Quinn FR, Mitchell LB, Mardell AP, Dal Disler RN, Veenhuyzen GD. Entrainment mapping of a concealed nodoventricular accessory pathway in a man with complete heart block and tachycardia-induced cardiomyopathy. *J Cardiovasc Electrophysiol*. 2008;19:90–94.
46. Wren C. Incessant tachycardias. *Eur Heart J*. 1998;19(suppl E):E32–E36.
47. 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;6:431–435.
48. Srivathsan K, Gami AS, Barrett R, Monahan K, Packer DL, Asirvatham SJ. Differentiating atrioventricular nodal reentrant tachycardia from junctional tachycardia: novel application of the delta H-A interval. *J Cardiovasc Electrophysiol*. 2008;19:1–6.
49. Padanilam BJ, Manfredi JA, Steinberg LA, Olson JA, Fogel RI, Prystowsky EN. Differentiating junctional tachycardia and atrioventricular node re-entry tachycardia based on response to atrial extrastimulus pacing. *J Am Coll Cardiol*. 2008;52:1711–1717.
50. Aasbo JD, Prasertwitayakij N, Morady F, Jongnarangsin K. Nonreentrant dual atrioventricular nodal tachycardia in a patient with atrioventricular nodal conduction abnormality. *Heart Rhythm*. 2009;6:1504–1506.
51. European Society of Cardiology Committee, NASPE-Heart Rhythm Society. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias: executive summary: a report of the American College of Cardiology/American Heart Association task force on practice guidelines and the European Society of Cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol*. 2003;42:1493–1531.
52. Kalbfleisch SJ, Strickberger SA, Williamson B, Vorperian VR, Man C, Hummel JD, Langberg JJ, Morady F. Randomized comparison of anatomic and electrogram mapping approaches to ablation of the slow pathway of atrioventricular node reentrant tachycardia. *J Am Coll Cardiol*. 1994;23:716–723.
53. Rostock T, Risius T, Ventura R, Klemm HU, Weiss C, Keitel A, Meinertz T, Willems S. Efficacy and safety of radiofrequency catheter ablation of atrioventricular nodal reentrant tachycardia in the elderly. *J Cardiovasc Electrophysiol*. 2005;16:608–610.
54. Li YG, Grönefeld G, Bender B, Machura C, Hohnloser SH. Risk of development of delayed atrioventricular block after slow pathway modification in patients with atrioventricular nodal reentrant tachycardia and a pre-existing prolonged PR interval. *Eur Heart J*. 2001;22:89–95.

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