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Differentiating Junctional Tachycardia and Atrioventricular Node Re-Entry Tachycardia Based on Response to Atrial Extrastimulus Pacing

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Objectives The purpose of this study was to differentiate non–re-entrant junctional tachycardia (JT) and typical atrioventricular node re-entry tachycardia (AVNRT).

Background JT may mimic AVNRT. Ablation of JT is associated with a lower success rate and a higher incidence of heart block. Electrophysiologic differentiation of these tachycardias is often difficult.

Methods We hypothesized that JT can be distinguished from AVNRT based on specific responses to premature atrial complexes (PACs) delivered at different phases of the tachycardia cycle: when a PAC is timed to His refractoriness, any perturbation of the subsequent His indicates that anterograde slow pathway conduction is involved and confirms a diagnosis of AVNRT. A PAC that advances the His potential immediately after it without terminating tachycardia indicates that retrograde fast pathway is not essential for the circuit and confirms a diagnosis of JT. This protocol was tested in 39 patients with 44 tachycardias suggesting either JT or AVNRT based on a short ventriculo-atrial interval and apparent AV node dependence. Tachycardias were divided into 3 groups: clinically obvious AVNRT, clinically obvious JT, and clinically indeterminate rhythm.

Results In the 26 cases of clinically obvious AVNRT, the sensitivity and specificity of the test were 61% and 100%, respectively. In the 9 cases of clinically obvious JT, the sensitivity and specificity were 100% and 100%, respectively. In the 9 cases of clinically indeterminate rhythm, the technique indicated AVNRT in 1 patient and JT in 7 patients, and the test was indeterminate in 1 patient.

Conclusions The response to PACs during tachycardia can distinguish JT and AVNRT with 100% specificity in adult patients. (J Am Coll Cardiol 2008;52:1711–7) © 2008 by the American College of Cardiology Foundation

Non–re-entrant junctional tachycardia (JT) may be difficult to distinguish from typical atrioventricular nodal re-entrant tachycardia (AVNRT) during electrophysiology (EP) study. Although their mechanisms are different, JT and AVNRT are confined to a relatively small anatomic area in and around the AV node, frequently rendering the usual pacing and mapping techniques not helpful in distinguishing the 2. Even a retrospective diagnosis after a successful ablation is difficult because ablation sites may be similar for the 2 rhythms. Ablation of JT has been reported to be associated with lower success rates and higher incidences of complete heart block compared with AVNRT (1), possibly because of the need for ablating part of the compact AV node in this rhythm. We describe a technique, using a single premature atrial complex (PAC) introduced during tachycardia, that can be helpful in differentiating JT and AVNRT in adult patients.

Methods

Hypothesis. We hypothesized that JT and AVNRT will have specific responses to PACs delivered at different phases of the tachycardia cycle. When a PAC is timed to His refractoriness, any perturbation (advancement, delay, or termination of tachycardia) of the next His indicates that anterograde AV nodal slow pathway conduction is present during the tachycardia. This response excludes JT and confirms the diagnosis of AVNRT (Fig. 1). Conversely, an earlier PAC that advances the His potential immediately after it, without terminating the tachycardia, confirms a diagnosis of JT (Fig. 2). Here, the advancement of the immediate His must be caused by anterograde conduction of the PAC over the AV nodal fast pathway, making it refractory. Because the AVNRT circuit requires AV nodal

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Abbreviations and Acronyms

AV = atrioventricular
AVNRT = atrioventricular node re-entry tachycardia
EP = electrophysiology
HRA = high right atrium
JT = junctional tachycardia
PAC = premature atrial complex

fast pathway for its retrograde limb, AVNRT must terminate after a PAC that advances the immediate His.

Study population. The preceding hypothesis was tested prospectively among patients who demonstrated tachycardia suggestive of AVNRT or JT during EP study. The JT cases primarily consisted of adults with the benign form of paroxysmal JT, and no cases of congenital or postoperative JT were present. The study period was from December 2002 to March 2008. This extended enrollment period was used to maximize the inclusion of patients with JT.

Electrophysiology study. The EP studies were performed using quadripolar recording electrodes positioned at high right atrium (HRA), bundle of His (His), and right ventricular apex after obtaining informed consent. The surface electrocardiographic recordings and intracardiac electrograms were continuously recorded on a digital recording system. The intracardiac electrograms were filtered at 40 and 500 Hz and displayed with amplifier settings of ±0.5 to ±1.0 mV. Overdrive and extrastimulus atrial and ventricular pacing were used to induce supraventricular tachycardia. Intravenous isoproterenol was administered if tachycardia was noninducible or nonsustained at baseline. Tachycardia characteristics were analyzed after a sustained episode occurred spontaneously or by induction. Patients demonstrating the following electrophysiological features suggesting typical AVNRT or JT were included in the study (2,3): short V-A interval (arbitrarily <150 ms), changes in A-H intervals preceding and predicting subsequent A-A interval changes, spontaneous termination of tachycardia with terminal atrial activation, and V-A-V response to ventricular burst pacing. Accessory pathway-mediated tachycardia and atrial tachycardia were not included in the study.

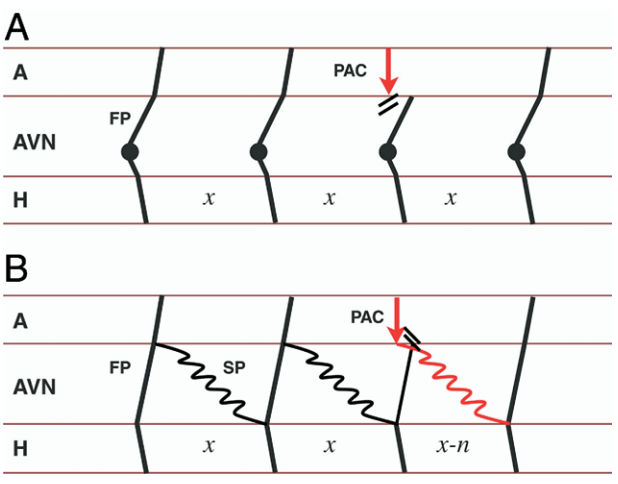

Figure 1 **Response to PAC Delivered When Junction Is Refractory**

Response to premature atrial complex (PAC) delivered when junction is refractory (local atrial activation from PAC occurs at or after His activation). **(A)** Response in junctional tachycardia: a PAC delivered at a time the junction focus has already depolarized blocks at the atrioventricular node (AVN) and is unable to influence the immediate or the next junction beat. **Solid circles** represent junction focus. **Black lines** show conduction through AVN, His (H), and atrium (A). **(B)** Response in atrioventricular node re-entry tachycardia (AVNRT): a similarly timed PAC can influence the next beat of AVNRT by early engagement of the slow pathway. **Black lines** show conduction through AVN, His (H), and atrium (A), and **red lines** show PAC and its response. Although this figure shows advancement of the next beat (*x-n*), delay of the next beat or termination of tachycardia are also specific to AVNRT. **Red arrow** indicates PAC. FP = fast atrioventricular node pathway; SP = slow atrioventricular node pathway; *x* and *x-n* = H-H intervals.

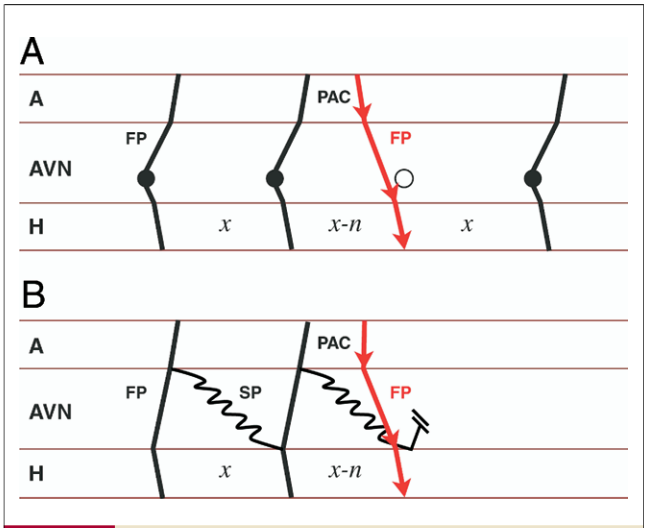

Figure 2 **Response to Earlier PACs**

(A) Response in junctional tachycardia (JT): the **open circle** represents the anticipated JT beat timing if no PAC were delivered. An early PAC advances the immediate JT beat and His timing by atrioventricular (AV) nodal fast pathway activation and JT continues. **(B)** Response in AVNRT: an early PAC may advance the immediate His by activation of the AV nodal fast pathway. However, that makes the fast pathway refractory and unavailable for retrograde conduction, terminating the AVNRT circuit. **Red arrow** indicates PAC and its response. Legends and abbreviations as in Figure 1.

Tachycardia groups. Tachycardias were divided into 3 groups: 1) clinically obvious AVNRT (C-AVNRT); 2) clinically obvious JT or accelerated junctional rhythm (C-JT); and 3) indeterminate group (C-Ind) with features of both JT and AVNRT. The C-AVNRT group was defined when the tachycardia initiation was reproducibly dependent on a critical A-H interval prolongation, provided no features of C-JT or C-Ind were present. The criteria for the C-JT group included the initiation of the rhythm by a spontaneous junctional beat during isoproterenol infusion, and relative increases in sinus rate (either spontaneous or with discontinuation of isoproterenol) overtook the junctional rhythm with immediate conduction over the anterograde AV nodal fast pathway. The C-Ind group patients had inducible tachycardia with atrial extrastimulus pacing. In addition, C-Ind was suggested by 1 or more of the following features: warm-up phenomenon with progressive

increase in tachycardia rates, wide cycle length variability, and tachycardia initiation by spontaneous junctional beats. All C-Ind patients received isoproterenol infusion at 2 to 10 μg/min during the study.

Diagnostic pacing maneuver. Single PACs were introduced into the tachycardia by scanning atrial diastole beginning at 10 ms shorter than the baseline tachycardia cycle length until loss of atrial capture occurred. The His bundle electrogram intervals (H-H) encompassing the PAC and the H-H intervals of the preceding and subsequent tachycardia cycles were measured. Analyses were made only when the preceding tachycardia cycle lengths were constant with <10 ms variability. The His signal encompassed in the V-V interval where the PAC was delivered is referred to as the "immediate His" and the His signal in the subsequent V-V interval is referred to as the "next His." For the purposes of this study, the AV junction was defined as refractory to a PAC when the local atrial activation resulting from the PAC occurred at or after the immediate His activation. After the delivered PAC, tachycardia characteristics including H-H intervals, V-V intervals, and termination of tachycardia were noted independently by 2 electrophysiologists and accuracy verified by a third electrophysiologist blinded to the study groups. Only reproducible changes were considered for analyses.

Ablation. Ablations were performed with 4-mm tip radiofrequency ablation catheters. Power delivered was titrated from 20 to 50 W with a temperature limit of 60°C based on AV conduction, V-A conduction, and presence of junctional beats. The ablation end points were absence of inducible or spontaneous clinically relevant tachycardias. When more than a single AV nodal echo beat was present, further ablation was done in patients with AVNRT. Isoproterenol (2 to 10 μg/min) was infused during post-ablation testing (16 of 26 patients) when AVNRT induction required isoproterenol before the ablation or at the discretion of the operator.

Follow-up. The 12-lead electrocardiograms were obtained in all patients at follow-up 1 to 2 months after the ablation procedure. Further clinical follow-up or event monitoring was performed only if patients reported recurrent symptoms.

Results

Patient characteristics. The study population consisted of 39 patients with sustained supraventricular tachycardia during EP study. The mean age was 55 ± 16 years, and 61% were women. Thirteen patients had left ventricular hypertrophy, 1 patient had tachycardia-induced cardiomyopathy, and 2 patients had a history of coronary artery bypass graft surgery. The heart was structurally normal by echocardiography in the remaining patients.

Tachycardia features. Based on the established criteria, 26 tachycardias were classified as C-AVNRT, 9 as C-JT, and 9 as C-Ind. In the C-AVNRT group, the tachycardia cycle lengths ranged from 272 to 596 ms (mean 404 ms), V-HRA interval was <80 ms in 20 of the 26 cases, and the interval ranged from 102 to 140 ms (mean 121 ms) in the remaining 6 patients. The patients with C-JT had relatively longer cycle lengths ranging from 576 to 900 ms (mean 716 ms), and these episodes occurred only during isoproterenol infusion. The V-HRA interval in this group of patients ranged from 15 to 62 ms (mean 36 ms).

Among the 9 C-Ind patients, 5 had tachycardia after ablation of AVNRT. The mean cycle length of the pre-ablation AVNRT was 427 ms and that of the post-ablation rhythm was 533 ms. Of these, 4 tachycardias occurred with atrial extrastimulus pacing during isoproterenol infusion, and 1 occurred with atrial extrastimulus pacing in the absence of isoproterenol. Three of these patients also exhibited sudden onset of tachycardia with a junctional beat. There were 4 C-Ind patients unrelated to AVNRT ablation. Two of these patients demonstrated tachycardia initiation with atrial extrastimulus pacing and an apparent A-H interval prolongation suggesting C-AVNRT during EP study. They also had some episodes of spontaneous initiation of tachycardia with a junctional beat. The other 2 patients were brought to the EP laboratory to define the mechanism of tachycardia and possible ablation because they both had mixed clinical features suggesting AVNRT or JT. Initiation of tachycardia with a junctional beat and warm-up phenomenon were demonstrated during EP study in these 2 patients. Atrial extrastimulus pacing with an apparent A-H interval prolongation also initiated the tachycardia. Tachycardia cycle lengths were 560 to 680 ms in the first patient and 420 to 496 ms in the second patient.

Response to PAC

C-AVNRT group (n = 26). A PAC delivered when the immediate His was refractory led to advancement of the next His (10 to 54 ms) in 10 patients (Fig. 3), delay of the next His in 4 patients, and no change in the next His in 12 patients. Three patients who had advancement of the next His also had termination of tachycardia with similarly timed PACs. Because the PAC was delivered when junction was refractory (as judged by local atrial activation from the PAC at the His bundle electrode occurring simultaneously or after the His signal), no changes in the immediate His timing were noted. In response to earlier PACs, none of the patients in this group demonstrated a response consistent with JT (advancement of the immediate His with continuation of the tachycardia).

C-JT group (n = 9). PACs delivered when the immediate His was refractory did not advance the next His in any of these patients. With PACs delivered before the next expected His (junctional) depolarization, all had advancement of the immediate His (33 to 200 ms) with continuation of the JT or junctional rhythm. The subsequent JT interval was unchanged (n = 6), advanced (n = 1), or delayed (n = 2).

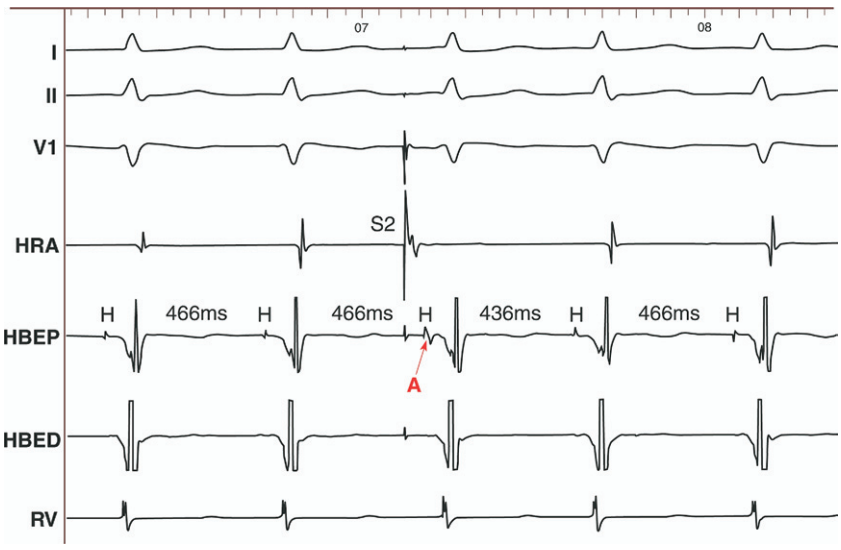

Figure 3 **PAC Response in AVNRT**

The premature atrial complex (PAC [S2]) timed to junctional (His) refractoriness advances the next His by 30 ms. The **red arrow (A)** points to the local atrial activation superimposed on the His signal. The advancement of the next His by this specifically timed PAC indicates atrioventricular node re-entry tachycardia as the mechanism. The measurements in milliseconds (ms) are H-H intervals. H = His electrogram; HBED = His bundle electrogram distal; HBEP = His bundle electrogram proximal; HRA = high right atrium; RV = right ventricle.

C-Ind group (n = 9). Among the 9 patients in this group, 1 had advancement of the next His with a PAC timed to His depolarization suggesting AVNRT, 7 had advancement of the immediate His with continuation of the tachycardia (Fig. 4) suggesting JT, and 1 had no changes with PACs.

Ablation outcomes. All patients in the C-AVNRT group had successful acute ablation results. No ablations were done in the C-JT group. In the C-Ind group, the patient with no changes in tachycardia after the PAC and the patient who had the response consistent with AVNRT had these tachy-

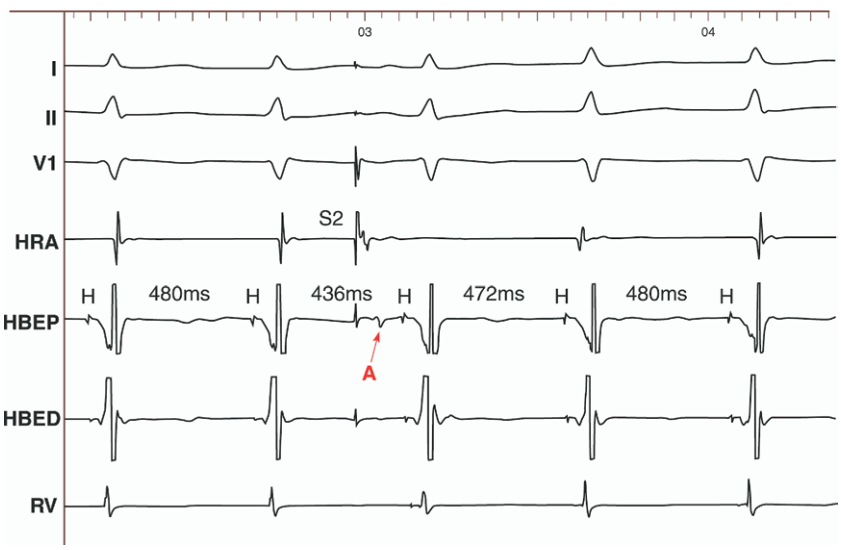

Figure 4 **PAC response in JT**

The same patient as shown in Figure 3 had this rhythm after radiofrequency ablations for atrioventricular node re-entry tachycardia. Early PAC (S2) advances the immediate His by 44 ms, and the tachycardia continues. The **red arrow (A)** points to the local atrial activation on the His recording catheter that occurs before the His bundle activation. This advancement of the immediate His by a PAC with continuation of tachycardia indicates junctional tachycardia as the mechanism, and further ablations are not necessary. The measurements in milliseconds (ms) are H-H intervals. Abbreviations as in Figure 3.

cardias develop after AVNRT ablation attempts. These patients underwent further ablations for presumed AVNRT and were subsequently noninducible. No further ablations were done in the other 3 patients who had C-Ind after AVNRT ablation. Among the 4 patients who had C-Ind unrelated to AVNRT ablation, all had advancement of the immediate His with continuation of tachycardia, suggesting JT. Two of these patients underwent ablation targeting the posterior right atrial septum to modify slow pathway conduction for AVNRT; however, despite multiple energy deliveries (25 and 16 lesions, respectively), the tachycardia could not be eliminated. The ablation attempts were abandoned after these attempts because testing with PACs suggested a JT mechanism. The other 2 patients did not undergo ablation on the basis of the response to PACs.

Follow-up. Twelve-lead electrocardiograms obtained from ablation patients at 1 to 2 months of follow-up showed no significant changes from baseline. One patient in the C-AVNRT group had recurrent AVNRT, and repeat ablation was successful. The patients with C-Ind after AVNRT ablation have not had any recurrent symptoms. Patients in the C-Ind group with JT diagnosis unrelated to AVNRT ablation have been treated with beta-blockers and calcium-channel blockers with good symptom control. None of these patients has had continued symptoms severe enough to warrant JT ablation.

Discussion

The distinction of JT from AVNRT is currently based on clinical features, and available diagnostic tests have limited value during EP study in differentiating the 2 rhythms. This study demonstrates the high specificity (100%) and sensitivity of responses to appropriately timed PACs in differentiating JT and AVNRT in adult patients.

Study findings. We used C-AVNRT and C-JT groups to validate the hypothesis and to determine the sensitivity and specificity of the test. The test was then applied to the C-Ind group to differentiate AVNRT and JT. Our protocol can be divided into 2 parts: evaluation of response to PACs introduced during His (junctional) refractoriness, and evaluation of response to earlier PACs. The following responses were identified with PACs introduced during His refractoriness: 1) advancement of the next His; 2) delay of the next His; 3) termination of tachycardia; and 4) no change in tachycardia. Any perturbation of the subsequent tachycardia beat (advancement, delay or termination) after a PAC when the junction is refractory indicates anterograde conduction via slow AV nodal pathway and effectively excludes the diagnosis of JT (see Study Limitations). The sensitivity of this response for AVNRT was 61% and the specificity was 100% in our patients.

Considering PACs introduced before junctional depolarization, the following responses occurred: 1) advancement of the immediate His with continuation of tachycardia; 2) advancement of the immediate His with termination of tachycardia; and 3) no change in the immediate His timing with advancement/delay of the next His or termination of tachycardia. Only response #1 has specificity for JT because AVNRT should terminate in this scenario. In our patients, the sensitivity and specificity of this response for JT were both 100%. Termination of tachycardia in response to a PAC (response #2) may occur in focal or re-entrant rhythms. Response #3 is also nonspecific, as described in the "timing of PAC" section.

To the best of our knowledge, these observations are novel. Hamdan et al. (4) have previously suggested using PAC in the AV nodal slow pathway region at a time the septal atrial activation has occurred as a way of identifying AVNRT. Entrainment, when demonstrated, can prove a re-entrant mechanism for a given tachycardia. However, transient entrainment by pacing is rather difficult to demonstrate in AVNRT, because atrial fusion does not occur, and interruption of the rhythm with demonstration of localized conduction block is necessary to prove its occurrence (5). A recent study evaluated the difference of H-A interval during tachycardia and ventricular pacing (ΔH-A) to distinguish JT and AVNRT (6). A positive ΔH-A interval in that study suggested JT, albeit with a specificity of only 83%. The relatively low specificity, difficulty in obtaining clear retrograde His recordings, and the dependence of the test responses on the site of origin of the JT could limit the clinical use of this method.

Electrophysiologic basis of the hypothesis. In contrast to AVNRT, JT is a focal rhythm originating in the AV junction, and no re-entrant circuits are thought to be involved. This difference provides a diagnostic opportunity to distinguish the 2 rhythms. A PAC delivered early in diastole may advance the immediate JT or AVNRT beat, but the tachycardia can continue only when the mechanism is focal (JT). For a PAC to advance the immediate tachycardia beat, it must traverse the fast AV nodal pathway in an anterograde direction. Thus, the fast AV nodal pathway is rendered refractory and unavailable for the retrograde limb of an AVNRT circuit, terminating the rhythm. Because JT is a focal rhythm (automatic or triggered activity), refractoriness of the AV nodal pathway should not preclude perpetuation of the tachycardia.

Additionally, because AV nodal slow pathway conduction is not operative during JT, a PAC cannot affect JT via anterograde slow pathway conduction. Therefore, when a PAC is timed to junctional depolarization (judged by the local atrial electrogram timing at or after His signal), there should be no perturbation of the subsequent JT beats. If any change occurs (advancement, delay, or termination of tachycardia) in the subsequent tachycardia beat, JT is excluded, and a rhythm using slow AV nodal pathway for anterograde conduction (in this case, AVNRT) is confirmed.

Timing of PAC. The timing of PAC to junctional refractoriness is crucial when interpreting the responses that confirm AVNRT and exclude JT. Focal non–re-entrant rhythms such as JT can decelerate, accelerate, or remain

unchanged in response to early depolarization of the tachycardia focus. Therefore, perturbation of the next tachycardia beat in response to a PAC is confirmatory for AVNRT only when the PAC could not have influenced the immediate beat. This can only be assured by documenting that the local atrial activation on the His recording channel occurs simultaneously or after the His bundle activation in response to a PAC (Figs. 1 and 3). Because an earlier PAC can conceal into the junction without actually affecting the His activation timing, lack of changes in the immediate His timing after an earlier PAC itself is not adequate to establish that the junction was refractory to the PAC. We validated the supposition that a PAC delivered when junction was refractory cannot affect the next JT beat by including 9 cases of clinically obvious junctional rhythms. None of these cases showed advancement, delay, or termination of tachycardia in response to PACs delivered when the junction was refractory.

Clinical utility. Differentiating JT and AVNRT during EP study has several applications. First, it can avoid unnecessary ablations when a JT mimicking AVNRT occurs after successful ablation of AVNRT. In this study a tachycardia with mixed features of JT and AVNRT developed after ablation in 5 of the 26 patients with AVNRT. In some of these cases, the cycle lengths of the AVNRT and post-ablation JT were very similar (Figs. 3 and 4). Eventually, 3 of these proved to be JT and 1 was AVNRT on the basis of PAC responses. In the remaining 1 patient, PACs did not perturb the tachycardia. This patient, however, underwent further ablation for AVNRT, rendering the rhythm noninducible. Absence of PAC effect is more likely to occur with an AVNRT than with a JT mechanism because it would be physiologically less probable for a PAC to enter the excitable gap of a re-entrant circuit. Clinical follow-up of patients deemed to have JT after AVNRT ablation showed no recurrent clinical tachycardia. Second, a small percentage of presumed AVNRT cases may be JT, and these cases can be identified prospectively, possibly predicting a more difficult ablation session and a higher risk of heart block. In our series of 39 patients, 2 patients whose diagnosis would otherwise have been AVNRT were thought to have JT on the basis of their response to PAC. Ablation attempts were unsuccessful in both of these patients, and persistent efforts were not made in favor of medical management. It should be noted, however, that successful ablation of JT without heart block is possible in a majority of patients (1). Third, a positive diagnosis of AVNRT can be made in some patients if a PAC timed to His refractoriness perturbs the next His timing. This feature may become useful particularly in cases where AVNRT with very slow cycle lengths raise suspicion of JT.

The benefit of the maneuver is illustrated by 2 cases from our study. First, a 27-year-old woman underwent radiofrequency ablation of typical AVNRT. During post-ablation testing, a narrow QRS-complex tachycardia with similar cycle length and electrophysiologic characteristics was induced. The rhythm was only noted during isoproterenol infusion and often initiated spontaneously with a junctional beat, suggesting a junctional rhythm. At other times, however, initiation of tachycardia occurred with an apparent A-H prolongation, which was suspicious for persistent AVNRT. Delivery of PACs resulted in advancement of the immediate His by 44 ms with continuation of the tachycardia, establishing the diagnosis of JT (Fig. 4). Further ablations were not undertaken. There was no recurrent tachycardia at 6-month follow-up. The second case involves a 64-year-old woman with dual AV nodal physiology and a tachycardia initially presumed to be AVNRT. Tachycardia with cycle length of 570 to 600 ms was induced with single atrial extrastimuli, but also initiated spontaneously with a junctional beat. After 16 unsuccessful ablation lesions, PACs introduced when the His was refractory did not affect the tachycardia, but early PACs advanced the immediate His by 24 ms with continuation of tachycardia. A diagnosis of JT was established. Because of concern for potential heart block, ablation was aborted in favor of further medical therapy.

Study limitations. A potential exception to our postulate may be the rare instances of dual response to PAC such that simultaneous anterograde fast and slow AV nodal pathway conduction occur. Here, in theory, continuation of AVNRT may occur after a PAC that advances the immediate His. Typically, dual responses would also be observed with PACs in sinus rhythm as well, and one should be cautious of interpreting this response during tachycardia in such cases. These patients also typically have AVNRT induced during incremental ventricular pacing (7). No dual responses to PACs were seen in our series. Importantly, no cases that had responses consistent with AVNRT (advancement, delay, or termination of the next tachycardia beat by a PAC delivered when junction was refractory) showed advancement of the immediate His with continuation of the tachycardia when earlier PACs were delivered (a response consistent with JT) and vice versa, pointing to the specificity of these responses. Another theoretical limitation of the technique could occur when dual AV node physiology is present in the setting of JT. Incidental anterograde slow pathway conduction could occur after the retrograde atrial activation from each JT beat, but the conduction would be too slow to reach the junction before the next JT beat to be able to influence it. A PAC timed to His refractoriness during JT could potentially advance (but not delay or terminate) the next tachycardia beat in this scenario. The PAC must occur early enough and engage the slow pathway without decremental conduction and reach the junction before the next JT beat for advancement to occur. Because PACs are delivered late (during His refractoriness) and slow pathway conduction was not fast enough to reach the junction during the JT, this scenario is unlikely. In addition, such a patient would also have dual AV node physiology with slow pathway conduction times that are very close to JT cycle length at baseline, alerting the electrophysiologist to this possibility. The selection of C-JT patients may also be considered a limitation of the study. The

patients included in the C-JT control group had JT or accelerated junctional rhythm episodes occurring with isoproterenol infusion during EP study. True clinical JT is a rather rare entity, and it would be very difficult to obtain an adequate number of such cases to be included in the study. Also, post-operative JT (8) and pediatric patients with congenital JT (9) were not represented in this study. However, the basic mechanistic implications of a focal JT versus a re-entrant AVNRT and their responses to PACs should still apply in any of these clinical entities. The accuracy of our results in such populations needs further study.

Conclusions

The electrophysiologic differentiation of JT and AVNRT is important in determining appropriate management options. The responses to appropriately timed PACs in this study distinguished the 2 rhythms with 100% specificity and high sensitivity. A clear physiological basis and the simplicity of the test should prompt its routine use during electrophysiology studies.

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Key Words: junctional tachycardia ■ atrioventricular node re-entry ■ premature atrial complex ■ catheter ablation.