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A Technique for the Rapid Diagnosis of Atrial Tachycardia in the Electrophysiology Laboratory

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OBJECTIVE The purpose of this study was to determine if the atrial response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction during paroxysmal supraventricular tachycardia is a useful diagnostic maneuver in the electrophysiology laboratory.

BACKGROUND Despite various maneuvers, it can be difficult to differentiate atrial tachycardia from other forms of paroxysmal supraventricular tachycardia.

METHODS The response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction was studied during four types of tachycardia: 1) atrioventricular nodal reentry (n = 102), 2) orthodromic reciprocating tachycardia (n = 43), 3) atrial tachycardia (n = 19) and 4) atrial tachycardia simulated by demand atrial pacing in patients with inducible atrioventricular nodal reentry or orthodromic reciprocating tachycardia (n = 32). The electrogram sequence upon cessation of ventricular pacing was categorized as "atrial-ventricular" (A-V) or "atrial-atrial-ventricular" (A-A-V).

RESULTS The A-V response was observed in all cases of atrioventricular nodal reentrant and orthodromic reciprocating tachycardia. In contrast, the A-A-V response was observed in all cases of atrial tachycardia and simulated atrial tachycardia, even in the presence of dual atrioventricular nodal pathways or a concealed accessory atrioventricular pathway.

CONCLUSIONS In conclusion, an A-A-V response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction is highly sensitive and specific for the identification of atrial tachycardia in the electrophysiology laboratory. (J Am Coll Cardiol 1999;33: 775–81) © 1999 by the American College of Cardiology

Differentiation of atrial tachycardia from other forms of paroxysmal supraventricular tachycardia during an electrophysiology procedure is critically important in the era of ablation (1,2) but can be challenging and time consuming (3–11). The diagnosis of an atrial tachycardia can be especially difficult in the presence of dual atrioventricular nodal pathways or an accessory atrioventricular pathway. We hypothesized that, in atrial tachycardia, ventricular pacing associated with 1:1 ventriculoatrial conduction would be followed by two consecutive atrial electrograms without an intervening ventricular electrogram. The purpose of this prospective study was to determine if the atrial response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction is useful for differentiating atrial tachycardias from other types of paroxysmal supraventricular tachycardia.

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METHODS

Study design. The atrial response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction was studied in the electrophysiology laboratory in patients who had inducible paroxysmal supraventricular tachycardia with 1:1 atrioventricular conduction. During supraventricular tachycardia, right ventricular pacing was initiated at a cycle length 10 to 60 msec shorter than the tachycardia cycle length until 1:1 ventriculoatrial conduction occurred, at which point pacing was discontinued. If pacing resulted in termination of the tachycardia, supraventricular tachycardia was reinduced and the maneuver was repeated. If ventricular pacing did not terminate the tachycardia, the atrial electrogram sequence immediately after the last ventricular paced complex was categorized as "atrial-ventricular" (A-V) (Fig. 1) or "atrial-atrial-ventricular" (A-A-V) (Fig. 2). Patients were excluded if ventricular pacing always resulted in ventriculoatrial dissociation or termination of the tachycardia.

To study the diagnostic usefulness of this pacing maneuver in patients with both atrial tachycardia and dual atrio-

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Abbreviations and Acronyms
A-V = atrial-ventricular
A-A-V = atrial-atrial-ventricular

ventricular nodal pathways or a concealed accessory atrioventricular pathway, atrial tachycardia was simulated by demand atrial pacing in a group of patients who had inducible atrioventricular nodal reentrant or orthodromic reciprocating tachycardia. The atrium was paced at a cycle length that resulted in 1:1 atrioventricular conduction, such that the ventriculoatrial relationship during pacing resembled that of the inducible atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia. During simulated atrial tachycardia, ventricular overdrive pacing was performed in the same fashion as during paroxysmal supraventricular tachycardia, and the response upon cessation of ventricular pacing was categorized as A-V or A-A-V (Fig. 3). If atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia was induced with overdrive ventricular pacing during simulated atrial tachycardia, atrial pacing was stopped and the tachycardia was terminated. Simulation of atrial tachycardia was then repeated with atrial pacing and the response to ventricular pacing was retested.

Characteristics of subjects. The study population consisted of 163 patients referred for management of paroxysmal supraventricular tachycardia. Their mean age was 45 ± 18 years (range 13 to 86 years) and 69% were women. A majority of patients (89%) had no evidence of structural heart disease. The remaining patients had coronary artery disease (n = 7), hypertension (n = 7), hypertrophic cardiomyopathy (n = 2), nonischemic dilated cardiomyopathy (n = 1) or aortic valve disease (n = 1).

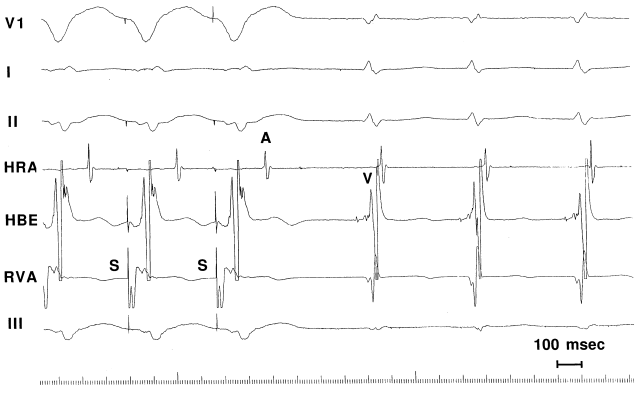

Figure 1. The response to ventricular pacing with 1:1 ventriculoatrial conduction during tachycardia in a patient with typical atrioventricular nodal reentrant tachycardia. Shown are leads V1, I, II, and III and the intracardiac electrograms recorded at the high right atrium (HRA), His-bundle electrogram (HBE) and right ventricular apex (RVA). The tachycardia cycle length is 430 msec. Ventricular pacing at a cycle length of 370 msec results in 1:1 ventriculoatrial conduction. The electrogram response upon cessation of ventricular pacing is atrial-ventricle (A-V). S = ventricular pacing stimuli.

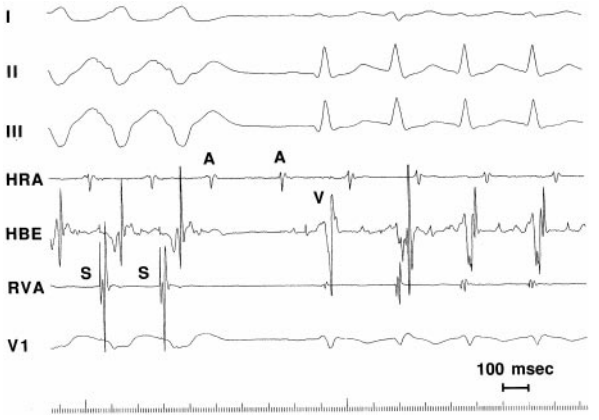

Figure 2. The response to ventricular pacing with 1:1 ventriculoatrial conduction during tachycardia in a patient with atrial tachycardia. The format and abbreviations are the same as in Figure 1. The tachycardia cycle length is 260 msec. Ventricular pacing at a cycle length of 230 msec results in 1:1 ventriculoatrial conduction. The electrogram response upon cessation of ventricular pacing is atrial-atrial-ventricle (A-A-V).

Electrophysiologic testing. Electrophysiology tests were performed in the fasting state after informed consent was obtained. Antiarrhythmic drug therapy was discontinued for at least five half-lives. Multipolar electrode catheters were inserted into a femoral vein and positioned in the high right atrium, His-bundle region, coronary sinus, and right ventricular apex. Atrial and ventricular pacing were performed at twice diastolic threshold using a Bloom DTU 210 (Bloom Associates, Narbeth, Pennsylvania) programmable stimulator. Four electrocardiographic leads and the intracardiac electrograms were recorded on paper at a speed of 100 mm/sec using a Mingograph 7 (Siemens-Elema, Solna, Sweden) recorder. The intracardiac electrograms were filtered at 40 and 500 kHz. The recordings also were stored on optical disks (Quinton Electrophysiology Corp., Seattle, Washington).

Overdrive pacing and premature extrastimuli were used to induce supraventricular tachycardia. Intravenous isoproterenol and atropine were administered if tachycardia was not inducible at baseline (12,13). Sinus tachycardia was induced with isoproterenol in 15 patients and was categorized as an atrial tachycardia for the purposes of this study.

Diagnosis of tachycardia mechanism. Atrioventricular nodal reentry was induced in 102 patients (typical in 99 patients and atypical in 3 patients), orthodromic reciprocating tachycardia in 43 patients (single accessory pathway in 42 patients and 2 accessory pathways in 1 patient) and atrial tachycardia in 19 patients (Table 1). All patients with atrioventricular nodal reentrant tachycardia underwent successful ablation of the slow atrioventricular nodal pathway

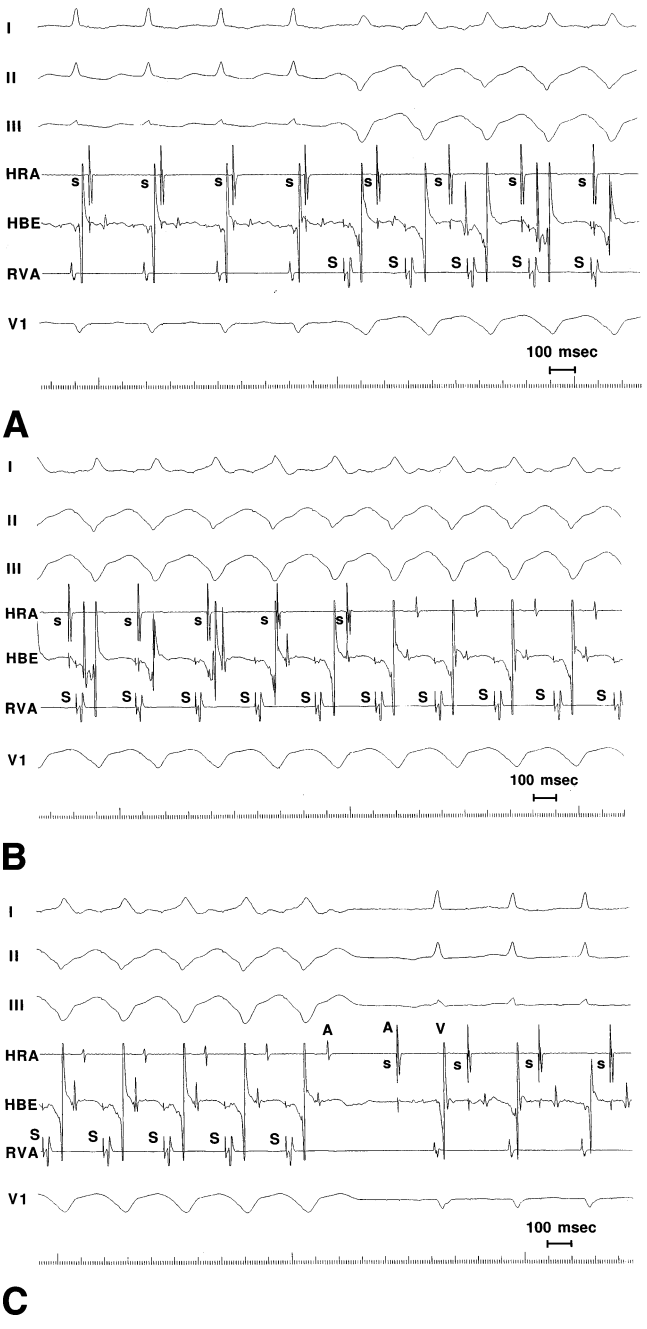

Figure 3. The response to ventricular pacing with 1:1 ventriculoatrial conduction during simulated atrial tachycardia in a patient with dual atrioventricular nodal pathways. The format is the same as in Figure 1. **Panel A** shows demand atrial pacing at a cycle length of 300 msec that results in 1:1 atrioventricular conduction and a short ventriculoatrial interval. Overdrive ventricular pacing is initiated at a cycle length of 260 msec during atrial pacing. **Panel B** shows 1:1 ventriculoatrial conduction during ventricular pacing and suppression of the atrial pacemaker. **Panel C** illustrates the A-A-V response upon cessation of ventricular pacing. s = atrial pacing stimuli; S = ventricular pacing stimuli.

after the study protocol was complete. Patients with orthodromic reciprocating tachycardia underwent successful ablation of accessory pathways at the following locations: anteroseptal (n = 1), right posteroseptal (n = 4), left posteroseptal (n = 1), left posterior (n = 2), left posterolateral (n = 9), left lateral (n = 9), left anterolateral (n = 11), left anterior (n = 2), right anterior (n = 1), right lateral (n = 2) and right posterolateral (n = 2). Tachycardia diagnoses were based on standard criteria (3–11). Patients were excluded from this study if there was uncertainty regarding the tachycardia mechanism after the application of standard criteria and careful mapping.

Evidence used to support a diagnosis of atrial tachycardia included inductions and terminations that were independent of atrio-Hisian or atrioventricular conduction delay, development of atrioventricular block during tachycardia, an atrial activation sequence that was not compatible with retrograde conduction through the atrioventricular junction or an accessory pathway and failure to terminate the tachycardia or preexcite the atrium with a ventricular extrastimulus delivered when the His-bundle was refractory. Atrial tachycardia was ruled out if the tachycardia was terminated by ventricular pacing without depolarization of the atrium, or when the ventriculoatrial conduction interval changed with the development of bundle branch block.

Evidence supportive of atrioventricular nodal reentrant tachycardia included inductions that were dependent on a critical prolongation of the atrial-His interval, concentric atrial activation and termination by a ventricular premature beat that was delivered when the His bundle was not refractory and that did not result in atrial depolarization. Atrioventricular nodal reentrant tachycardia was considered typical when the septal ventriculoatrial interval was $\leq$70 msec and atypical if >70 msec. Dual atrioventricular nodal pathways were diagnosed when atrioventricular nodal reentrant tachycardia was inducible. Findings used to diagnose orthodromic atrioventricular reentrant tachycardia included eccentric atrial activation during tachycardia or ventricular pacing, termination by a premature ventricular depolarization that was delivered when the His bundle was refractory and did not preexcite the atrium and an increase in the ventriculoatrial conduction interval with the development of bundle branch block.

Protocol for simulation of atrial tachycardia. In a subset of patients with inducible atrioventricular nodal reentry (n = 27) or orthodromic atrioventricular reentry (n = 6), it was possible to pace the atrium at a rate which resulted in an atrioventricular relationship similar to that of the inducible supraventricular tachycardia, without inducing the tachycardia. In these patients, atrial tachycardia was simulated by pacing the high lateral right atrium (n = 21) or low posterior right atrial septum (n = 12), using a Medtronic dual chamber pacemaker, Model 7984 (Medtronic Inc., Minneapolis, Minnesota). Pacing was performed in the AAI mode at the shortest cycle length that conducted in a 1:1 fashion to the ventricle without inducing paroxysmal supraventricular tachycardia, and that resulted in an atrio-

Table 1. Characteristics of Supraventricular Tachycardias Studied and the Response Immediately After Ventricular Pacing with 1:1 Ventriculoatrial Conduction

Tachycardia	N	Dual Path (%)	Acc Path (%)	CL (msec)	Septal VA int (msec)	HRA VA int (msec)	A-V Response (%)	A-A-V Response (%)
AVNRT	102	100	0	328 ± 64	24 ± 34	53 ± 39	100	0
ORT	43	0	100	327 ± 53	152 ± 41	176 ± 38	100	0
AT	19	5	0	404 ± 67	296 ± 103	282 ± 90	0	100
Simulated AT								
	27	100	0	318 ± 42	85 ± 56	90 ± 62	0	100
	5	0	100	406 ± 139	282 ± 169	232 ± 156	0	100

A-V Response = "atrial-ventricular" electrogram sequence after ventricular overdrive pacing during tachycardia; A-A-V Response = "atrial-atrial-ventricular" electrogram sequence after ventricular overdrive pacing during tachycardia; Acc Path = accessory atrioventricular pathway; AT = atrial tachycardia; AVNRT = atrioventricular nodal reentrant tachycardia; CL = cycle length; Dual Path = dual atrioventricular nodal pathways; N = number of patients; HRA = high right atrium; ORT = orthodromic reciprocating tachycardia; Simulated AT = atrial tachycardia simulated with demand atrial pacing; VA int = ventriculoatrial conduction interval.

ventricular relationship similar to that of the paroxysmal supraventricular tachycardia. The pacemaker was connected to a standard multipolar electrode catheter using an adapter for the inline header. The sensitivity was adjusted as necessary to prevent ventricular oversensing and to allow suppression of atrial pacing during ventricular pacing that resulted in 1:1 ventriculoatrial conduction.

Data analysis. Continuous variables are expressed as mean ± standard deviation and were compared using a Student *t*-test. An ANOVA combined with a Bonferroni procedure was used when multiple means were compared. Nominal variables were compared using Fisher's exact test. A p value less than 0.05 was considered significant.

RESULTS

The characteristics of the tachycardia and responses after ventricular pacing for each type of supraventricular tachycardia are summarized in the Table. The tachycardia cycle length among patients with an atrial tachycardia or a simulated atrial tachycardia was significantly longer compared to patients with atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia (359 ± 78 vs. 328 ± 61 msec; $p < 0.01$). The septal ventriculoatrial time was significantly shorter among patients with atrioventricular nodal reentrant tachycardia compared with patients with orthodromic reciprocating tachycardia (24 ± 34 vs. 152 ± 41 msec; $p < 0.01$), and the septal ventriculoatrial time during simulated atrial tachycardia was significantly shorter among patients with dual atrioventricular nodal pathways compared with patients with a concealed accessory pathway (85 ± 56 vs. 282 ± 169; $p < 0.01$).

An A-V response was observed in each of the 145 cases of atrioventricular nodal reentry and orthodromic reciprocating tachycardia. In contrast, an A-A-V response was observed in each of the 51 cases of atrial tachycardia and pacing-simulated atrial tachycardia ($p < 0.01$).

DISCUSSION

Main findings. The main finding of this study is that the A-A-V response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction during paroxysmal supraventricular tachycardia is unique to atrial tachycardias. This response allows for the rapid differentiation of atrial tachycardia from other forms of paroxysmal supraventricular tachycardia in the electrophysiology laboratory.

Response after ventricular pacing during supraventricular tachycardia. When the ventricle is paced during atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia at a cycle length shorter than the tachycardia cycle length and all electrograms are advanced to the pacing rate without terminating the tachycardia, ventriculoatrial conduction occurs through the retrograde limb of the circuit. Therefore, after the last paced ventricular complex, the anterograde limb of the tachycardia circuit is not refractory, and the last retrograde atrial complex is able to conduct to the ventricle. This results in an A-V response. However, when the ventricle is paced during atrial tachycardia and 1:1 ventriculoatrial conduction is produced, retrograde conduction occurs through the atrioventricular node. In this case, the last retrograde atrial complex resulting from ventricular pacing is unable to conduct to the ventricle because the atrioventricular node is refractory to anterograde conduction, and the result is an A-A-V response.

Atrioventricular nodal reentrant tachycardia can occasionally be associated with 2:1 atrioventricular block (14,15). Atrioventricular block usually occurs at the initiation of tachycardia due to a long-short sequence, persists for several beats, and resolves with ventricular pacing. Therefore, in patients with atrioventricular nodal reentrant tachycardia with 1:1 atrioventricular conduction, it would be very unlikely for ventricular pacing, at a cycle length just shorter than the tachycardia cycle length, to result in atrioventricular block for one beat (an AAV response) after pacing is stopped. In the present study, we did not observe induction

of 2:1 atrioventricular block in 102 patients with atrioventricular nodal reentrant tachycardia when the ventricle was paced during tachycardia. The pacing maneuver presented in this study would not be useful when 1:1 atrioventricular conduction is not present.

Atrial tachycardia coexisting with dual atrioventricular nodal pathways or a concealed accessory pathway. In patients with atrial tachycardia who also have dual atrioventricular nodal pathways or a concealed accessory pathway, ventricular pacing during tachycardia theoretically could result in ventriculoatrial conduction through only the fast atrioventricular nodal or accessory pathway; if this occurred, the last atrial complex which results from retrograde conduction could potentially conduct back to the ventricle through the atrioventricular node, resulting in an A-V response. However, in the present study, unless atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia was induced with atrial or ventricular pacing, the A-V response was not observed in any of the 33 patients with atrial tachycardia or simulated atrial tachycardia who also had dual atrioventricular nodal pathways or a concealed accessory pathway. The most likely explanation is that retrograde penetration of both atrioventricular nodal pathways or of the atrioventricular node and the accessory pathway occurred during ventricular pacing, such that both pathways were refractory to anterograde conduction upon cessation of pacing.

Theoretically, the only situation in which an A-V response may not rule out atrial tachycardia is the unlikely scenario of an atrial tachycardia occurring simultaneously with atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia and also entraining the atrioventricular nodal reentrant tachycardia or orthodromic reciprocating tachycardia. Because this scenario is probably very rare, the A-V response indicates that a tachycardia is highly unlikely to be atrial in origin.

Potential pitfalls in determining the response after ventricular pacing. When determining the response after ventricular pacing during paroxysmal supraventricular tachycardia, the presence of 1:1 ventriculoatrial conduction must be confirmed. Isorhythmic ventriculoatrial dissociation may mimic 1:1 ventriculoatrial conduction. The atrial response upon cessation of ventricular pacing provides diagnostically useful information only when 1:1 ventriculoatrial conduction has occurred during pacing (Fig. 4).

A pseudo-A-A-V response may occur with atypical atrioventricular nodal reentry because retrograde conduction occurs through the slow pathway during ventricular pacing. This may result in a ventriculoatrial conduction interval long enough to mimic an A-A-V response. Careful identification of the last atrial electrogram that resulted from ventriculoatrial conduction during ventricular pacing will avoid this potential pitfall.

On the other hand, a pseudo-A-V response may occur with an atrial tachycardia or sinus tachycardia when the maneuver is performed during an isoproterenol infusion. Ventricular pacing with 1:1 ventriculoatrial conduction may result in overdrive suppression of the atrial focus and the isoproterenol may cause an increase in junctional automaticity, such that an apparent A-V response occurs (Fig. 5). For this reason, it is important to determine whether the response after cessation of ventricular pacing is reproducible when performed during an isoproterenol infusion.

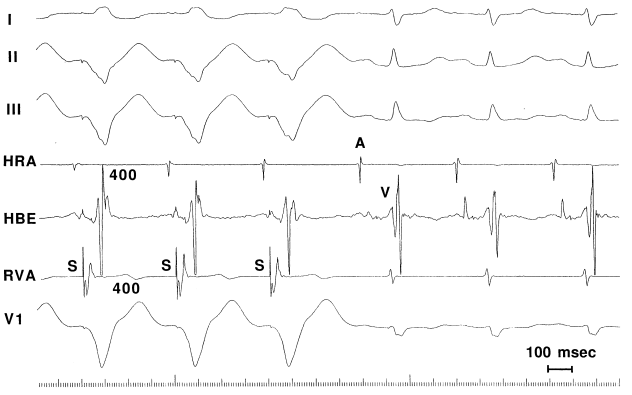

Figure 4. The response to ventricular pacing during tachycardia in a patient with isoproterenol-induced sinus tachycardia. The format and abbreviations are the same as in Figure 1. Numbers represent intervals in milliseconds. The tachycardia cycle length is 400 msec. Ventricular pacing at a cycle length of 400 msec results in isorhythmic ventriculoatrial dissociation that mimics 1:1 ventriculoatrial conduction and appears to result in an A-V response. However, the electrogram response upon cessation of ventricular pacing provides diagnostically useful information only when 1:1 ventriculoatrial conduction has occurred during pacing. The atrial electrogram sequence is "high-low" during tachycardia and is the same during ventricular pacing, consistent with absent ventriculoatrial conduction during ventricular pacing.

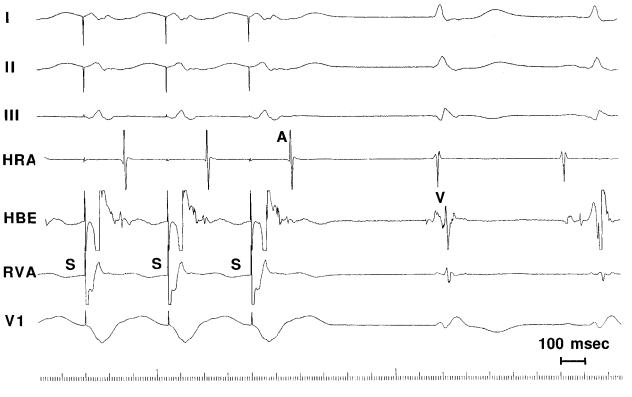

Figure 5. The response to ventricular pacing with 1:1 ventriculoatrial conduction during tachycardia in a patient with isoproterenol-induced sinus tachycardia. The format and abbreviations are the same as in Figure 1. Ventricular pacing at a cycle length of 350 msec with 1:1 ventriculoatrial conduction results in overdrive suppression of the sinus node and the isoproterenol causes an increase in junctional automaticity, such that an apparent A-V response occurs. For this reason, it is important to determine whether the response after cessation of ventricular pacing is reproducible during isoproterenol infusion.

Limitations. For the pacing maneuver described in this study to be usable, the ventriculoatrial block cycle length during supraventricular tachycardia must be less than the tachycardia cycle length. If the ventriculoatrial block cycle length is longer than the tachycardia cycle length, isoproterenol can be administered to enhance ventriculoatrial conduction and make the maneuver feasible. In the present study, ventricular pacing with 1:1 ventriculoatrial conduction was feasible in all patients with atrioventricular nodal reentrant tachycardia and orthodromic reciprocating tachycardia, and in approximately 80% of patients with atrial tachycardia. Although confirmation in a larger number of patients is necessary, it appears that the inability to achieve 1:1 ventriculoatrial conduction with ventricular pacing during supraventricular tachycardia may in itself be highly suggestive of an atrial tachycardia.

The results of this study may not apply to patients with automatic junctional tachycardia or a concealed nodofascicular pathway, because patients with these rare forms of paroxysmal supraventricular tachycardia were not included. However, the response upon cessation of ventricular pacing during automatic junctional tachycardia or tachycardia due to a concealed nodofascicular pathway theoretically should be A-V in all cases.

Prior studies. Several diagnostic techniques have been described to differentiate atrial tachycardia from other forms of supraventricular tachycardia during an electrophysiology test (3–11). The relative sensitivities and specificities of diagnostic maneuvers for distinguishing atrial tachycardias from other types of paroxysmal supraventricular tachycardia have not been analyzed. However, the pacing maneuver presented here appears highly accurate in the diagnosis of atrial tachycardia. Although this maneuver does not eliminate the need for other diagnostic tools, it has the advantages of being a rapid technique that is simple to perform, is independent of tachycardia initiation or termination and requires only a single atrial recording. Given the limitations of the diagnostic maneuvers currently available, it is useful to have additional maneuvers available, such as the one described in this study.

A report by Taniguchi, et al. used a retrograde "V-A-A-H" induction sequence as evidence that the mechanism of a narrow complex tachycardia was atrial (16). The pacing maneuver described in the present study is based on a similar principle, but is performed after induction of the tachycardia and is therefore useful in the diagnosis of tachycardias induced with atrial pacing.

Clinical implications. The response after ventricular pacing associated with 1:1 ventriculoatrial conduction during paroxysmal supraventricular tachycardia is a simple, rapid and accurate pacing maneuver that is helpful in differentiating atrial tachycardia from other mechanisms of paroxysmal supraventricular tachycardia in the electrophysiology laboratory. The A-A-V electrogram response upon cessation of ventricular pacing associated with 1:1 ventriculoatrial conduction during paroxysmal supraventricular tachycardia appears to be diagnostic for atrial tachycardia. Therefore, when an A-A-V response is observed, mapping and ablation should focus on an atrial substrate (1,2). In contrast, when an A-V response is observed, atrioventricular nodal reentrant tachycardia, orthodromic reciprocating tachycardia or automatic junctional tachycardia is present. However, the simultaneous presence of an atrial tachycardia along with atrioventricular nodal reentrant tachycardia, orthodromic reciprocating tachycardia or automatic junctional tachycardia cannot be ruled out. Therefore, after slow pathway or accessory pathway ablation is performed, further testing to exclude an inducible atrial tachycardia is advisable.

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