The Response of Paroxysmal Supraventricular Tachycardia to Overdrive Atrial and Ventricular Pacing: Can It Help Determine the Tachycardia Mechanism?

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Pacing During Supraventricular Tachycardia. ***Introduction:*** **Standard electrophysiologic techniques generally allow discrimination among mechanisms of paroxysmal supraventricular tachycardia. The purpose of this study was to determine whether the response of paroxysmal supraventricular tachycardia to atrial and ventricular overdrive pacing can help determine the tachycardia mechanism.**

Methods and Results: **Fifty-three patients with paroxysmal supraventricular tachycardia were studied. Twenty-two patients had the typical form of atrioventricular (AV) junctional (nodal) reentry, 18 patients had orthodromic AV reentrant tachycardia, 10 patients had atrial tachycardia, and 3 patients had the atypical form of AV nodal reentrant tachycardia. After paroxysmal supraventricular tachycardia was induced, 15-beat trains were introduced in the high right atrium and right ventricular apex sequentially with cycle lengths beginning 10 msec shorter than the spontaneous tachycardia cycle length. The pacing cycle length was shortened in successive trains until a cycle of 200 msec was reached or until tachycardia was terminated. Several responses of paroxysmal supraventricular tachycardia to overdrive pacing were useful in distinguishing atrial tachycardia from other mechanisms of paroxysmal supraventricular tachycardia. During decremental atrial overdrive pacing, the curve relating the pacing cycle length to the VA interval on the first beat following the cessation of atrial pacing was flat or upsloping in patients with AV junctional reentry or AV reentrant tachycardia, but variable in patients with atrial tachycardia. AV reentry and AV junctional reentry could always be terminated by overdrive ventricular pacing whereas atrial tachycardia was terminated in only one of ten patients ($P < 0.001$). The curve relating the ventricular pacing cycle length to the VA interval on the first postpacing beat was flat or upsloping in patients with AV junctional reentry and AV reentry, but variable in patients with atrial tachycardia. The typical form of AV junctional reentry could occasionally be distinguished from other forms of paroxysmal supraventricular tachycardia by the shortening of the AH interval following tachycardia termination during constant rate atrial pacing.**

Conclusions: **Atrial and ventricular overdrive pacing can rapidly and reliably distinguish atrial tachycardia from other mechanisms of paroxysmal supraventricular tachycardia and occasionally assist in the diagnosis of other tachycardia mechanisms. In particular, the ability to exclude atrial tachycardia as a potential mechanism for paroxysmal supraventricular tachycardia has important implications for the use of catheter ablation techniques to cure paroxysmal supraventricular tachycardia.** *(J Cardiovasc Electrophysiol, Vol. 4, pp. 239-252, June 1993)*

atrial tachycardia, entrainment, radiofrequency ablation

Introduction

Paroxysmal supraventricular tachycardia (PSVT) may be due to a variety of mechanisms. Reentry confined to the region of the AV junction and atrioventricular (AV) reentrant tachycardia are the two most common mechanisms of PSVT in adults.

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Although atrial tachycardias are less common, they still may be responsible for a significant number of cases of PSVT.[1,2] Standard electrophysiologic techniques usually allow the determination of the mechanism of PSVT but defining the tachycardia mechanism and, in particular, excluding atrial tachycardia as a mechanism of PSVT, may be difficult.[3] Recently, methods for cure of the majority of cases of PSVT using radiofrequency current have been described,[4,5] and this has increased the importance of accurately defining the tachycardia mechanism.

Termination, resetting, and entrainment of PSVT by atrial or ventricular pacing have been previously described by several investigators.[6-10] In addition, some but not all electrophysiology texts state the ability to "dissociate" the atrium or ventricle from a tachycardia may aid in the diagnosis.[11,12] However, the ability of these pacing techniques to help identify the mechanism of PSVT by altering the AV and VA relationships have not been examined in a large group of patients. The purpose of this study was to prospectively examine the ability of overdrive atrial and ventricular pacing to identify the mechanism of PSVT.

Methods

Patient Population

This was a prospective study of the response of PSVT of various mechanisms to atrial and ventricular pacing. An attempt was made to recruit patients with diverse mechanisms of PSVT. The first 43 patients included in the protocol were consecutive patients referred to the Electrophysiology Laboratory at the University of Michigan Medical Center who had a single type of PSVT that was hemodynamically stable and reproducibly inducible. Patients seen during this time period having PSVT of more than one mechanism were excluded. In order to recruit a larger number of patients with atrial tachycardia and with the atypical form of AV nodal reentry, another ten patients presenting to the University of Michigan Medical Center or Northwestern Memorial Hospital with PSVT that was suspected to be due to atrial tachycardia or atypical AV junctional reentry were also included.

Fifty-three patients with a mean age of 49 ± 17 years constituted the study population. There were 28 females. Forty-one of the patients (77%) had no evidence of underlying structural heart disease. Demographic characteristics of the patient population as a whole and for patients with each type of PSVT are included in Table 1. There were no significant differences in demographic characteristics of the patient population among patients with different types of PSVT.

Electrophysiologic Testing

Electrophysiologic studies were performed in the fasting state at least five half-lives after antiarrhythmic agents had been withdrawn and after informed consent was obtained. Multipolar electrode (USCI, Billerica, MA, USA) catheters were positioned in the high right atrium, His-bundle region, right ventricular apex, and coronary sinus. Atrial and ventricular pacing were performed using rectangular stimuli 2-msec in duration at twice diastolic threshold using a Bloom DTU 210 (Bloom Associates, Narbeth, PA, USA) programmable stimulator. At least three surface electrocardiographic leads and multiple intracardiac electrograms were amplified on a Mingograph 7 (Siemens-Elema, Solna, Sweden) or Bers 401A (Bloom Associates) amplifier system, filtered at

TABLE 1
Clinical Characteristics of the Patient Population

	Atrial Tach (n = 10)	Typical AVJR (n = 18)	Atypical AVJR (n = 3)	AVRT (n = 22)	Total (n = 53)
Age (years)	58 ± 18	47 ± 19	51 ± 13	46 ± 16	49 ± 17
Gender (Female:Male)	5:5	13:5	1:2	9:13	28:25
Heart Disease	None: 6 CAD: 1 CM: 3	None: 14 CAD: 1 CM: 1 MVP: 2	None: 3	None: 18 CAD: 3 CM: 1	None: 41 CAD: 5 CM: 5 MVP: 2
Preexcitation	Absent: 10	Absent: 18	Absent: 3	Present: 13 Absent: 9	Present:13 Absent: 40

Atrial tach = atrial tachycardia; AVJR = atrioventricular junctional tachycardia; AVRT = atrioventricular reentrant tachycardia; CAD = coronary artery disease; CM = cardiomyopathy; MVP = mitral valve prolapse.

40 to 500 kHz and displayed on an oscilloscope. Data were recorded at 100 mm/sec on a strip chart recorder.

If PSVT could not be induced by atrial or ventricular pacing or single atrial or ventricular extrastimuli, multiple atrial and ventricular extrastimuli were introduced. If PSVT could still not be induced, intravenous isoproterenol (14 μg/min) was infused to a target heart rate of 120 beats/min and stimulation was repeated.[13]

Presumptive Diagnosis of Tachycardia Mechanism

A presumptive diagnosis of the tachycardia mechanism was made using criteria that have been previously described.[14] Tachycardias were classified as atrial, orthodromic AV reentrant tachycardia, typical AV junctional reentry, or atypical AV junctional reentry. AV reentrant tachycardia was distinguished from other types of PSVT by eccentric atrial activation, by preexcitation of the atria during tachycardia by a ventricular premature beat or beats occurring within 30 msec of the His-bundle deflection, and/or by an increase in the VA interval during the tachycardia when bundle branch block developed.[15,16] Preexcitation of the atrium by ventricular premature beat was not, of itself, used to define the PSVT mechanism since it could indicate only the presence of an innocent bystander bypass tract. Typical AV junctional reentry was distinguished from AV reentrant tachycardia by the presence of simultaneous atrial and ventricular activation. Both typical AV junctional reentry and AV reentrant tachycardia were distinguished from atrial tachycardia by termination of the tachycardia with a ventricular premature beat that did not preexcite the atrium. Atypical AV junctional reentry was distinguished from typical AV junctional reentry by having an His-atrial interval longer than the atrial-His interval during PSVT. Atypical AV junctional reentry was distinguished from AV reentrant tachycardia by a retrograde activation sequence that was consistent with septal activation,[17] by failure to preexcite the atrium with a ventricular premature depolarization at a time that the His bundle was refractory, and by continuation of the tachycardia in the presence of AV block.

Atrial tachycardia was distinguished from other mechanisms of PSVT by the inability of a ventricular premature stimulus to preexcite the atrium at a time when the His bundle was refractory, by the inability to terminate PSVT with a ventricular premature stimulus that did not preexcite the atrium, by an atrial activation sequence not compatible with retrograde conduction via the AV junction or accessory AV connection, and by the development of AV block during PSVT.

Protocol for Atrial and Ventricular Overdrive Pacing

After the initiation of PSVT, pacing from the high right atrium and right ventricular apex were performed sequentially. Overdrive pacing trains consisted of 15 beats and began at a cycle length 10 msec shorter than the PSVT cycle length. The first beat of the drive train was coupled to the last spontaneous beat of the tachycardia at the pacing cycle length. The pacing cycle length was decreased in 10-msec steps until either the tachycardia was terminated or a pacing cycle length of 200 msec was reached. Measurements were repeated once for verification, and tachycardia terminations that were not reproducible to within a 20-msec cycle length were excluded from analysis.

Data Analysis

The following measurements were made on electrograms recorded from the His-bundle catheter for each patient at each pacing cycle length and compared for each of the PSVT mechanisms:

(1) Presence or absence of tachycardia termination with atrial and ventricular pacing.
(2) Site of termination of the tachycardia with atrial and ventricular pacing. The site of termination was considered antegrade if the tachycardia terminated with an atrial response not followed by a ventricular response. The site of termination was considered retrograde if the tachycardia terminated with a ventricular response not followed by an atrial response.
(3) During atrial pacing: (a) The presence or absence of AV block during atrial pacing; (b) AH interval of the last paced beat; (c) VA interval on the first postpacing beat; and (d) AH interval following termination of the tachycardia at the pacing cycle length that reproducibly terminated PSVT. An example of measurements performed during atrial pacing is shown in Figure 1A.
(4) During ventricular pacing: (a) Presence or absence of VA conduction; (b) The VA interval of the last paced beat; and (c) AH interval on the first postpacing beat. Examples of

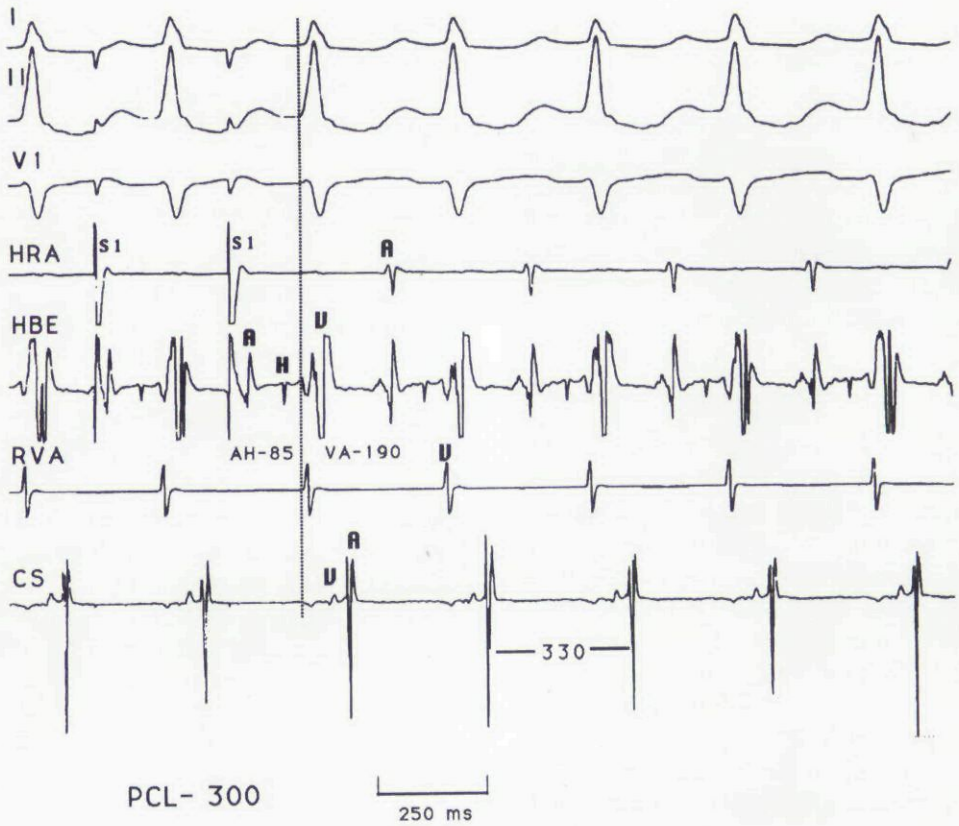


Figure 1A. *Example of measurements made during atrial pacing. Tracings are taken from a patient with a concealed left lateral bypass tract. Surface leads I, II, and V1, and intracardiac leads from the high right atrium (HRA), His bundle (HBE), coronary sinus os (CS), and right ventricular apex (RVA) are shown. The tachycardia cycle length is 330 msec. The last two beats of atrial pacing at a cycle length of 300 msec are designated by S1. 1:1 AV conduction is present during the tachycardia. Measurements were made following the cessation of atrial pacing. The AH interval was measured in the His-bundle recording on the last paced beat. The VA interval was measured from the onset of the surface QRS (dashed line) on the first postpacing ventricular beat to the onset of the atrial electrogram in the HBE.*

measurements made during ventricular pacing are shown in Figure 1B.

Curves were constructed relating the atrial and ventricular pacing cycle lengths to the AH and VA intervals after the cessation of pacing. Only curves in which at least three pacing cycle lengths were present prior to tachycardia termination were analyzed. The curves were described as being flat, variable, upsloping, or downsloping using definitions similar to those described for resetting of ventricular tachycardia.[18] A flat curve was defined by the presence of a 10-msec or less difference in the postpacing interval (AH or VA) over a 30-msec or greater range of pacing cycle lengths. An upsloping or downsloping curve had a greater than 10-msec increase or decrease in the postpacing interval with a decrease in pacing cycle length. A variable curve had both upsloping and downsloping portions. Curves obtained with each mechanism of PSVT were compared to each other.

The following curves were constructed (Figs. 2A and 2B):

Ap PCL-AH: The curve relating atrial pacing cycle length to the AH interval on the last paced beat.

Ap PCL-VA: The curve relating atrial pacing cycle length to the VA interval on the first postpacing beat.

Vp PCL-VA: The curve relating ventricular pacing cycle length to the VA interval on the last paced beat.

Vp PCL-AH: Curves relating ventricular pacing cycle length to the AH interval on the first postpacing beat.

The difference between the PSVT cycle length and the pacing cycle length that terminated PSVT was examined for both atrial and ventricu-

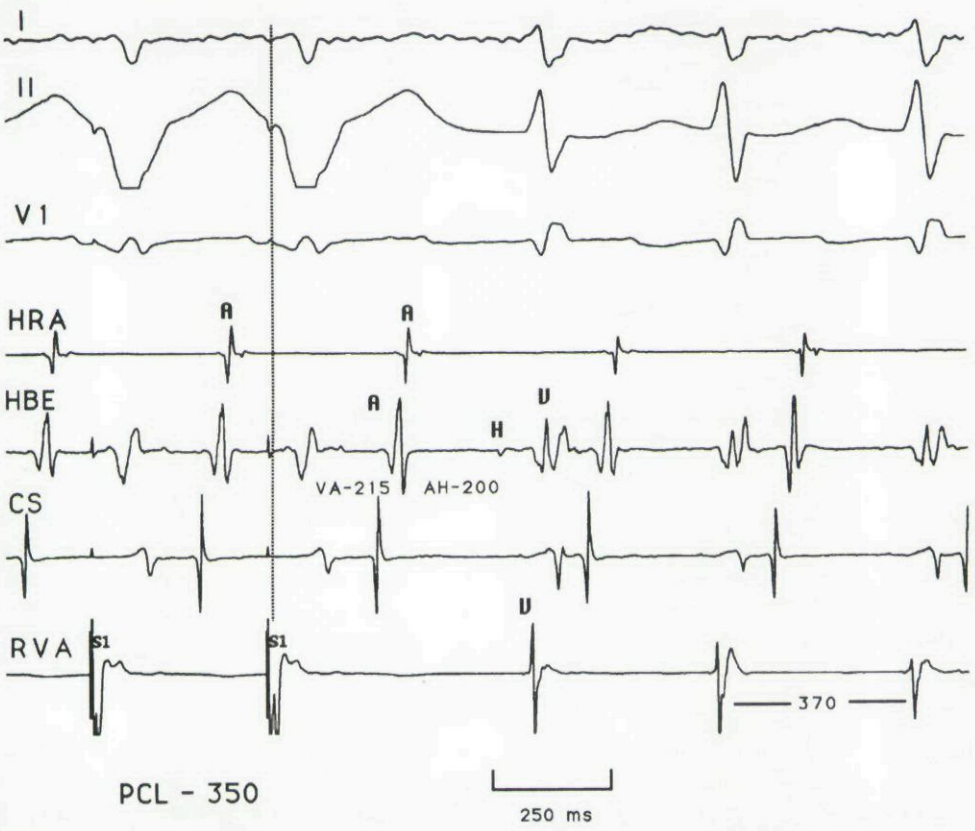


Figure 1B. *Examples of measurements made during ventricular pacing. As in Figure 1A, surface leads I, II, and V1, and intracardiac leads from the HRA, HBE, CS, and RVA are shown. Tracings are taken from a patient with a manifest left-sided accessory AV connection. The tachycardia cycle length was 360 msec. The last two beats of ventricular pacing at a cycle length of 300 msec are shown. The VA interval on the last paced beat was measured from the onset of the surface QRS complex to the first onset of rapid deflection of the atrial electrogram in the His-bundle recording. The AH interval on the first postpacing beat was measured in the His-bundle electrogram as is demonstrated.*

lar pacing. This difference was referred to as ΔCL and defined by the equation:

ΔCL = PSVT cycle length

– pacing CL that terminated PSVT

The ΔCL for each of the PSVT mechanisms were compared as were the ΔCLs during atrial and ventricular pacing.

Measurements were made at a paper speed of 100 mm/sec to an accuracy of 5 msec by a single observer (AK). A subset of measurements was repeated by this observer and also determined by a second observer to assess intraobserver and interobserver variability. Repeated measurements were within 5 msec 90% of the time and within 10 msec 98% of the time. Data are expressed as mean ± standard deviation. Comparison among groups was performed by analysis of variance for continuous variables and contingency table analysis for categorical variables. Comparisons among groups with different tachycardia mechanisms and between atrial and ventricular pacing were performed using a two-way analysis of variance.

Results

Characteristics of PSVT induced in each of the patient groups are shown in Table 2. Twenty-two patients had AV reentrant tachycardia, 18 patients had the typical form of AV junctional reentry, 3 patients had the atypical form of AV junctional reentry, and 10 patients had atrial tachycardia. Of the 10 patients with atrial tachycardia, 2 appeared to have PSVT secondary to automatic atrial tachycardia and 8 due to intraatrial reentry. The mean cycle length of induced PSVT was 340 ± 62 msec. There was no significant difference in the mean tachycardia cycle length among the four groups. The mean AH interval was 187 ±

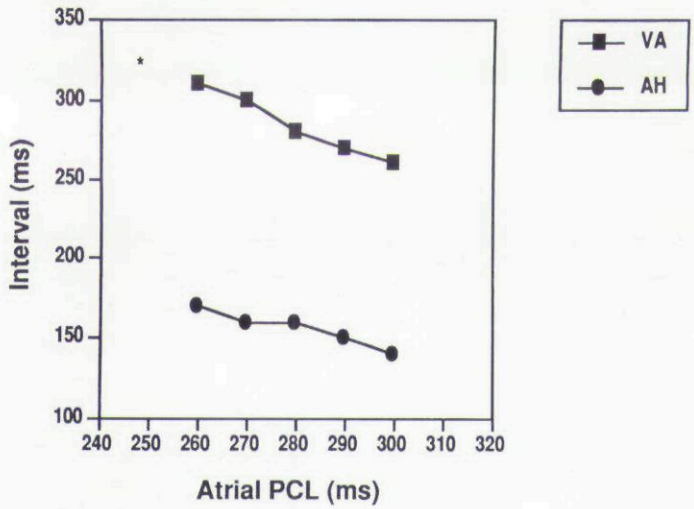


Figure 2A. *Examples of curves relating the atrial pacing cycle length (PCL) to the AH interval on the last paced beat and the VA interval on the first postpacing beat in a patient with atypical AV junctional reentry. The pacing cycle length in msec is shown on the X axis and the VA or AH times in msec on the Y axis. The asterisk (also in Fig. 2B) indicates the cycle length that terminated PSVT. The Ap PCL-VA curve and the Ap PCL-AH curve were both characterized as upsloping.*

91 msec. The mean AH interval was shortest in the patients with atypical AV junctional reentry (73 ± 15 msec), intermediate in patients with atrial tachycardia and AV reentrant tachycardia (147 ± 42 and 138 ± 52 msec, respectively), and longest in the patients with typical AV junctional reentry (286 ± 63 msec). Comparisons between the patients with atypical AV junctional reentry, the groups with atrial and AV reentrant tachycardia, and the group with typical AV junctional reentry were significant at the $P < 0.05$ level. The mean HV interval was similar in the four groups. The mean VA interval was 1 ± 19 msec in the patients with typical AV junctional reentry and was significantly shorter than that in the other three groups ($P < 0.05$ for all comparisons).

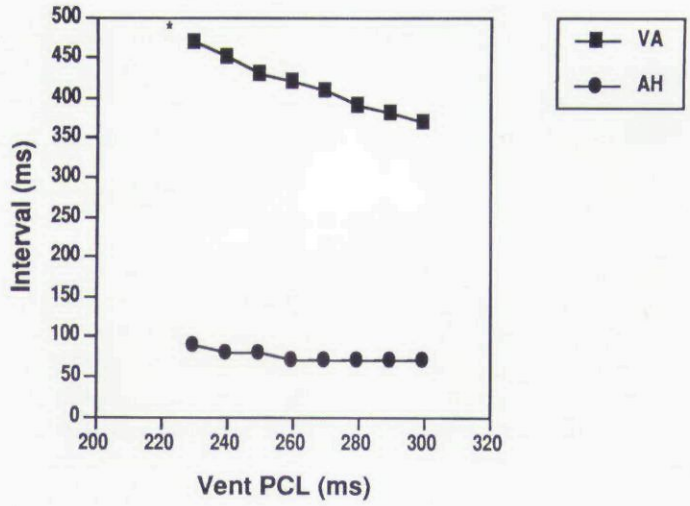


Figure 2B. *An example of curves relating the ventricular pacing cycle length to the VA interval on the last paced beat and the AH intervals on the first postpacing beat. Curves are taken from the same patient as shown in Figure 2A. The ventricular pacing cycle length in msec is shown on the X axis and the VA or AH interval on the Y axis. The Vp PCL-VA curve was flat and the Vp PCL-AH were characterized as upsloping. The degree of increase in the interval as the PCL was shortened was greater for the Vp PCL VA curve.*

TABLE 2
Characteristics of Supraventricular Tachycardia (in msec)

	Atrial Tach (n = 10)	Typical AVJR (n = 18)	Atypical AVJR (n = 3)	AVRT (n = 22)	Total (n = 53)
SVT CL	348 ± 64	337 ± 65	307 ± 12	343 ± 64	340 ± 62
AH	147 ± 42	286 ± 63	73 ± 15	138 ± 52	187 ± 91
HV	50 ± 20	51 ± 8	52 ± 8	48 ± 13	50 ± 13
VA	155 ± 69	1 ± 19	177 ± 21	160 ± 64	106 ± 13

SVT CL = cycle length of supraventricular tachycardia; VA = VA interval as measured in the His-bundle recording. Other abbreviations as in Table 1.

Responses That Were Helpful in Distinguishing Among Tachycardia Mechanisms

Responses to atrial pacing

The response of PSVT to atrial pacing prior to tachycardia termination was evaluated by examining the curve relating the VA interval to pacing cycle length prior to tachycardia termination. Adequate data to construct curves were available in 12 of 18 patients with the typical form of the AV junctional tachycardia, in 18 of 22 patients with AV reentrant tachycardia, in 8 of 10 patients with atrial tachycardia, and in 3 of 3 patients with the atypical form of AV junctional reentry. In the remaining patients, PSVT was terminated with the first or second pacing train. The curve relating the VA interval on the first posttachycardia beat following the termination of atrial pacing (Ap PCL-VA) was flat in patients with the typical form of AV junctional reentry and with AV reentrant tachycardia. The curve was upsloping in all three patients with the atypical form of AV junctional reentry. In patients with atrial tachycardia, the Ap PCL-VA curve was flat in one patient, and downsloping or variable in the remaining seven patients ($P < 0.01$ vs other tachycardia mechanisms). The response of an atrial tachycardia to atrial pacing at cycle lengths of 450, 390, and 330 msec is shown in Figure 3 (panels A through c). The VA interval on the first postpacing beat formed a variable curve as shown in Figure 3 (panel D). Typical Ap PCL-VA curves for each of the mechanisms of PSVT are shown in Figure 4. A variable curve is seen in the patient with atrial tachycardia in contrast to the flat or upsloping curves in patients with other mechanisms of PSVT. Thus, examination of the VA interval on the first postpacing beat following the cessation of atrial pacing helped distinguish atrial tachycardia from other mechanisms of PSVT.

When PSVT could be terminated with atrial pacing, the antegrade activation time during pacing was compared before and after tachycardia termination. A decrease in the antegrade activation time following termination of the tachycardia with atrial pacing was seen in 2 of 18 patients with the typical form of AV junctional reentry and in 14 of 22 patients with AV reentrant tachycardia, but in none of the patients in the other two groups. In the two patients with the typical form of AV junctional reentry, a long AH interval prior to tachycardia termination was followed by an AH interval < 100 msec with 1:1 AV conduction (Fig. 5). This phenomenon was not seen in patients with other mechanisms of PSVT. In the 13 patients with manifest ventricular preexcitation in sinus rhythm, termination of AV reentrant tachycardia was followed by at least one beat of antegrade conduction over the accessory pathway.

Responses to ventricular pacing

PSVT could be terminated by overdrive ventricular pacing in 44 of 53 patients. This included all patients with AV junctional reentry and AV reentrant tachycardia. In nine of the ten patients with atrial tachycardia, ventricular overdrive pacing to cycle lengths as low as 200 msec was unable to terminate PSVT. These nine patients developed VA block at pacing cycle lengths 10 to 80 msec shorter than the PSVT cycle length. The difference in the ability of ventricular overdrive pacing to terminate PSVT between atrial tachycardia and the other three types of tachycardia was significant at the $P < 0.01$ level.

During ventricular pacing, an adequate number of cycle lengths were available to determine a curve relating the VA time on the last paced beat to the pacing cycle length in 14 of 18 patients with typical form of AV junctional reentry, 17 of 22 patients with AV reentrant tachycardia, 3 of 3 patients with

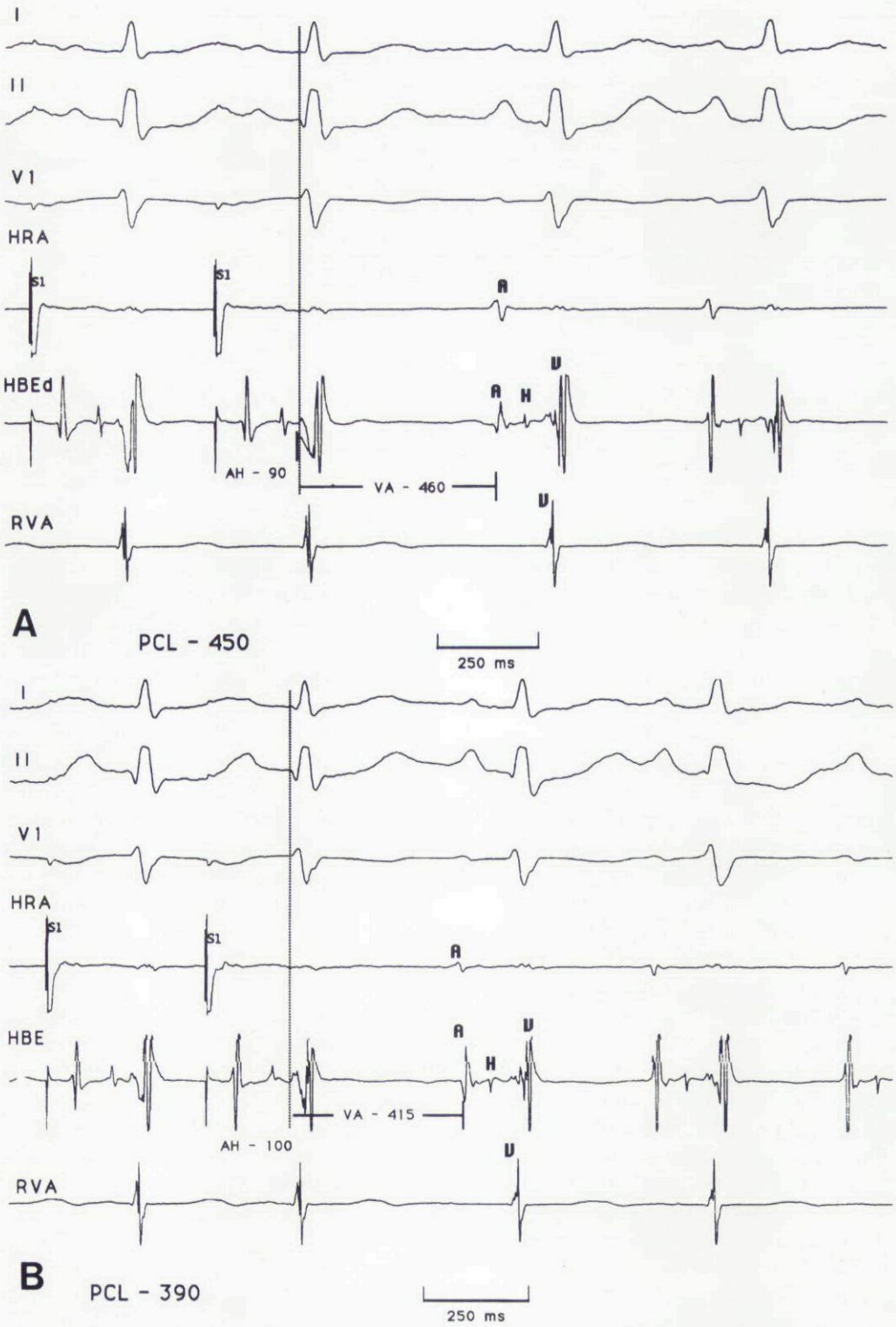
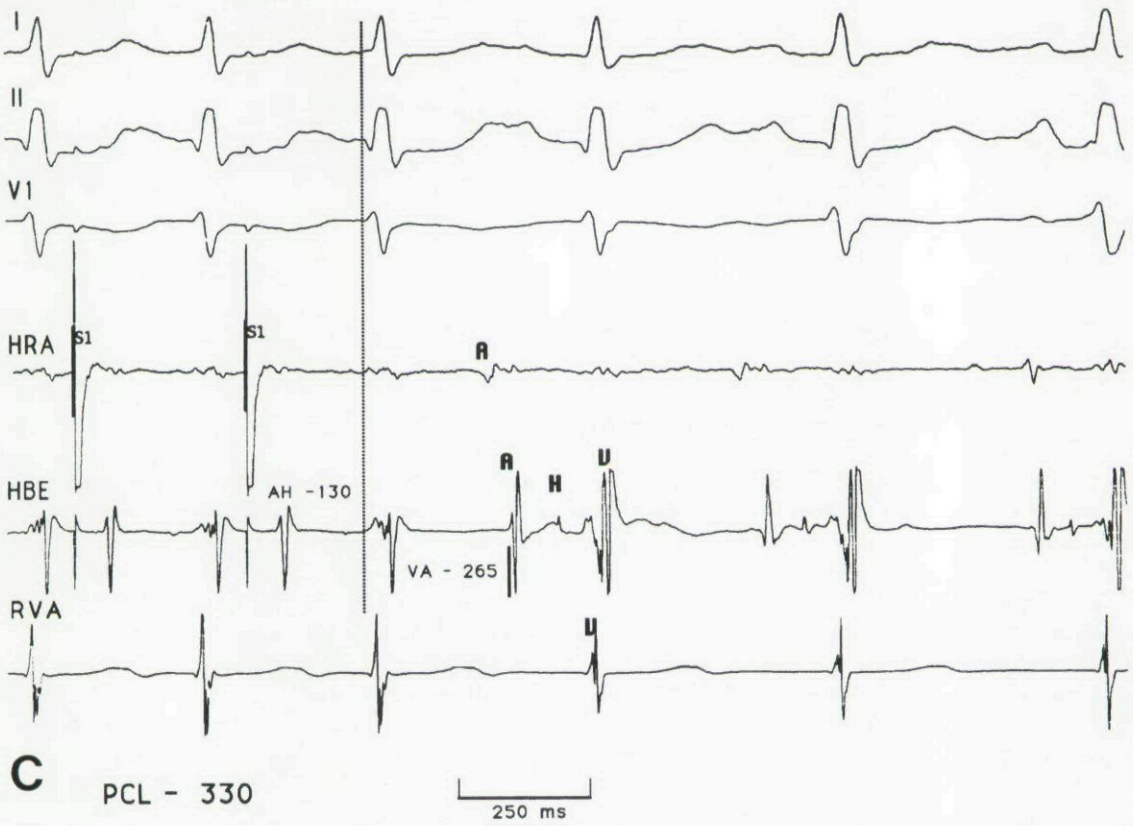


Figure 3. *Tracings obtained from a patient with an automatic atrial tachycardia arising from the region of the sinus node. Surface leads I, II, and V1, and intracardiac recordings from the high right atrium (HRA), His-bundle electrogram (HBE), and right ventricular apex (RVA) are shown. The last two beats of overdrive atrial pacing at cycle lengths of 450 (A), 390 (B), and 330 (C) msec are shown. Tracings from the coronary sinus were obtained but are not shown. As expected, the AH interval increased as the pacing cycle length was shortened. The VA interval measured in the HBE was 460, 415, and 265 msec at pacing cycle lengths of 450, 390 and 330 msec, respectively. These tracings are part of the variable Ap PCL-VA curve shown in D. A decrease in VA interval as the pacing cycle length was shortened was seen only in patients with atrial tachycardia.*



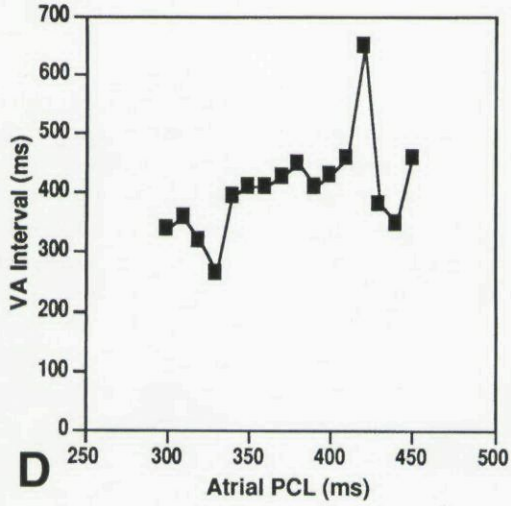


Figure 3. (*Continued*)

the atypical form of AV junctional reentry, and 10 of 10 patients with atrial tachycardia. In the patients with typical AV junctional reentry, the Vp PCL-VA curve was flat in 12 patients, and increased by 20 and 25 msec in the two remaining patients. In 17 patients with AV reentrant tachycardia, the Vp PCL-VA curve was flat in 15 of 17 patients and trended upwards by 20 and 25 msec in the remaining two patients. In the three patients with atypical AV junctional reentry, the Vp PCL-VA curve was upsloping, suggesting decremental conduction in the retrograde pathway. In the ten patients with atrial tachycardia, the Vp PCL-VA curve was flat in one patient, and variable in the remaining nine patients ($P < 0.01$ vs other tachycardia mechanisms). An example of typical Vp PCL-VA curves is shown in Figure 6. Thus, the Vp PCL-VA curve was useful

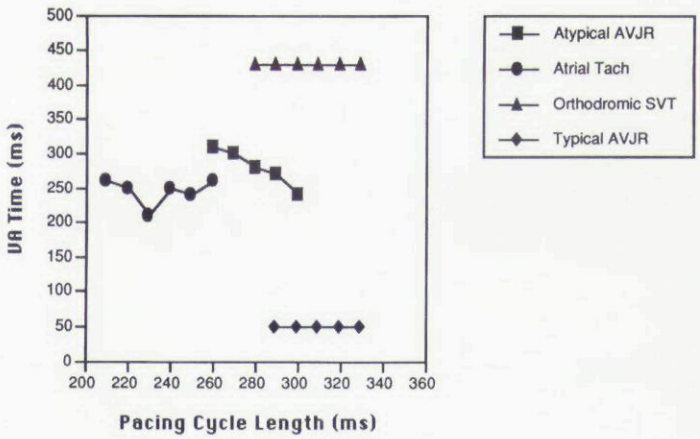


Figure 4. *Curves relating the atrial pacing cycle length to the first postpacing VA interval in patients with each mechanism of PSVT. The atrial pacing cycle length is shown on the X axis and the VA interval for the first postpacing beat at each cycle length on the Y axis. Data from a patient with the typical form of AV junctional reentry is shown in diamonds, data from a patient with AV reentrant tachycardia in triangles, data from a patient with atrial tachycardia in circles, and data from a patient with atypical form of AV junctional reentry in squares. As expected, VA times were shortest for the patient with the typical form of AV junctional reentry. The Ap PCL-VA curve was characterized as flat in the patients with AV reentrant tachycardia and the typical form of AV junctional reentry, upsloping in the patient with atypical AV junctional reentry, and variable in the patient with atrial tachycardia. The patient with AV reentrant tachycardia had a slowly conducting left-sided accessory pathway and thus relatively long VA times.*

in distinguishing atrial tachycardia from other mechanisms of PSVT.

Other Responses to Atrial Pacing

PSVT could be terminated by atrial overdrive pacing in 51 of 53 patients. The only exceptions were two patients who had atrial tachycardia that appeared to be automatic in nature. PSVT was terminated with an atrial depolarization not followed by ventricular depolarization through the His bundle (antegrade mode) in 17 of 18 patients with typical AV junctional reentry, 19 of 22 patients with AV reentrant tachycardia, 2 of 3 patients with the atypical form of AV junctional reentry, and 4 of 8 patients with atrial tachycardia. Differences among the groups were not significant.

The difference between the atrial pacing cycle length that terminated PSVT and the cycle length of PSVT was largest for patients with atrial tachycardia. Atrial tachycardia was terminated at a pacing cycle length a mean of 66 ± 38 msec shorter than the spontaneous tachycardia cycle length. In contrast, the ΔCL during atrial pacing was a mean of 38 ± 12 msec for typical AV junctional reentry, 39 ± 19 msec faster for AV reentrant tachycardia, and 47 ± 21 msec faster for atypical AV junctional reentry ($P < 0.05$ atrial tachycardia vs AV reentrant tachycardia and typical AV junctional reentry). However, considerable overlap was present among the groups, which limited the ability of the ΔCL during atrial pacing to distinguish among PSVT mechanisms.

The response of the AH interval to atrial overdrive pacing was also examined (Ap PCL-AH curve). In 10 of 12 patients with typical AV junctional tachycardia, 14 of 18 patients with AV reentrant tachycardia, 7 of 8 patients with atrial tachycardia, and 2 of 3 patients with the atypical form of AV junctional tachycardia, an increasing AH interval was noted as the pacing cycle length was shortened prior to tachycardia termination. Thus, the response of the AH interval to atrial pacing did not help to distinguish among the tachycardia mechanisms.

Other Responses to Ventricular Pacing

Ventricular pacing terminated PSVT with VA block in 8 of 18 patients with typical AV nodal reentry, 13 of 22 patients with AV reentrant tachycardia, and 2 of 3 patients with atypical AV nodal reentry. In the remaining patients, PSVT terminated following an atrial depolarization. Ventricular pacing terminated PSVT with atrial depolarization in the single patient with atrial tachycardia in whom ventricular pacing terminated PSVT. The

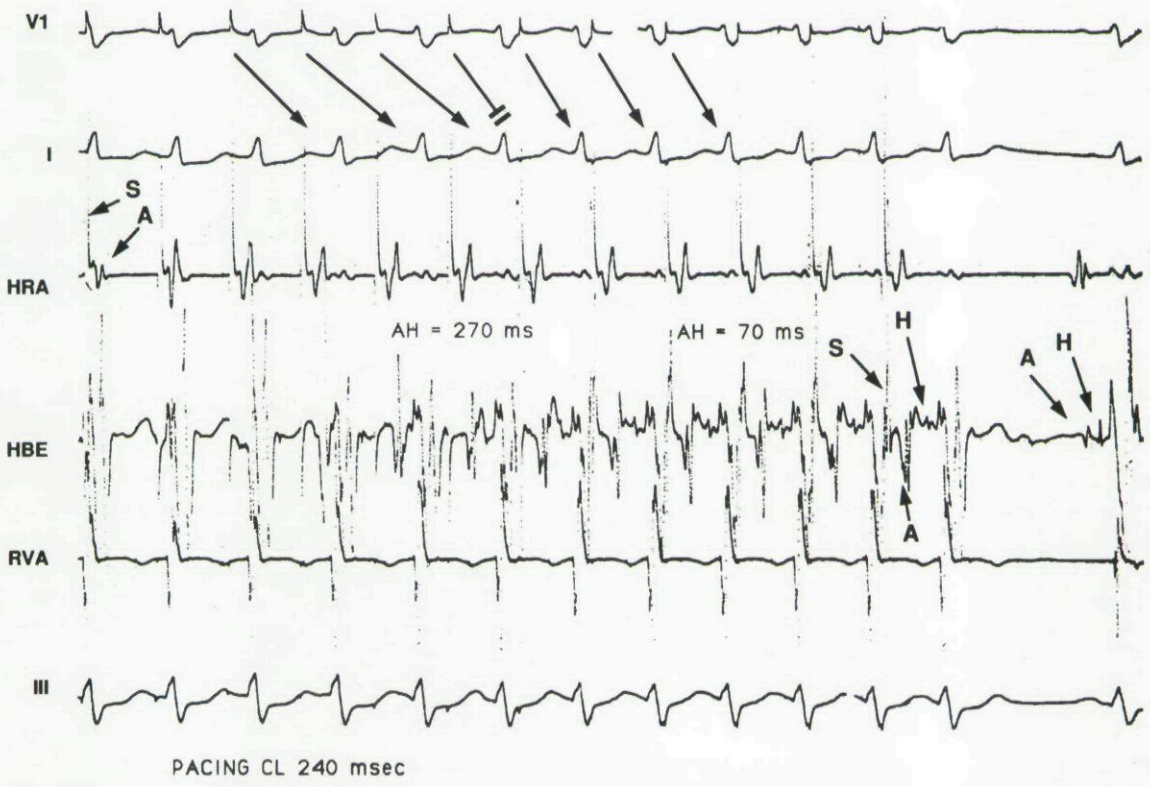


Figure 5. *Termination of typical AV junctional reentry with atrial pacing. Tracings were taken from a patient with a typical form of AV junctional reentry with a cycle length of 300 msec. Recordings from surface leads V1 and I, high right atrium (HRA), His-bundle electrogram (HBE), right ventricular apex (RVA), and surface lead III are shown. Recordings from the coronary sinus were obtained but are not shown. The last 12 beats of an atrial pacing train at a cycle length of 240 msec are depicted. The long arrows indicate conduction of pacing stimuli to the ventricles. Beginning with the third pacing stimulus shown, the AV time progressively increases until the sixth stimulus blocks. The longest AH interval is 270 msec. Following block of the stimulus, 1:1 AV conduction with a short AH interval of 70 msec is maintained. Thus, following termination of the tachycardia, a change in the antegrade AV conduction is demonstrated. This change in antegrade activation time was seen in 2 of 18 patients with the typical form of AV junction reentry.*

differences in termination site among the three other types of PSVT were not significant.

The curve relating the AH on the first postpacing beat to the pacing cycle length was upsloping in 12 of 14 evaluatible patients with typical AV junctional reentry and flat in the remaining 2 patients. In the three patients with the atypical form of AV junctional reentry, the Vp PCL-AH curves were upsloping. The Vp PCL-AH curve was also upsloping in 16 of 17 patients with AV reentrant tachycardia and was flat in 1 patient. In nine of ten patients with atrial tachycardia, the Vp PCL-AH curves had a flat portion as a consequence of the development of VA dissociation.

Discussion

The major finding of this study is that the response of PSVT to atrial and ventricular pacing can be helpful in distinguishing among tachycardia mechanisms. Atrial tachycardia can be distinguished from other mechanisms of PSVT by a variable VA time on the first postpacing beat following the cessation of atrial or ventricular overdrive pacing, and by the inability to terminate the tachycardia with ventricular pacing. If there is a shortening in the AH interval after termination of PSVT by atrial pacing, this could imply that the mechanism of PSVT is typical AV junctional reentry.

Criteria for Distinguishing Atrial Tachycardia From Other Mechanisms of PSVT

Atrial tachycardia with 1:1 AV conduction is a relatively uncommon mechanism of PSVT that may be difficult to distinguish from other types of

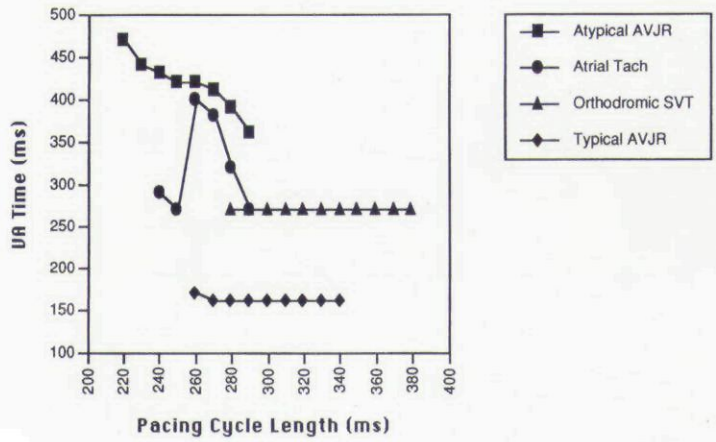


Figure 6. *Curves relating the ventricular pacing cycle length to the VA interval on the last paced beat in patients with each mechanism of PSVT. The atrial pacing cycle length is shown on the X axis and the VA interval on the last paced beat at each cycle length on the Y axis. Data from a patient with the typical form of AV junctional reentry is shown in diamonds, data from a patient with AV reentrant tachycardia in triangles, data from a patient with atrial tachycardia in circles, and data from a patient with the atypical form of AV junctional reentry in squares. The Vp PCL-VA curve was characterized as flat in the patient with AV reentrant tachycardia and the typical form of AV junctional reentry, upsloping in the patient with atypical AV junctional reentry, and variable in the patient with atrial tachycardia.*

tachycardia. The AV interval may vary widely among patients based on differences in antegrade conduction. The tachycardia should not be dependent upon activation of the AV junctional or ventricular myocardium and thus the response to atrial and ventricular pacing was useful in distinguishing atrial tachycardia from other mechanisms of PSVT. When atrial overdrive pacing is performed during atrial tachycardia, the AV interval is dependent on AV nodal conduction delay, which may increase at faster pacing rates; however, the AA interval following the cessation of atrial pacing is dependent upon the presence and magnitude of conduction delay within an atrial tachycardia circuit or focus. Thus, one would not expect the VA interval following the cessation of atrial pacing to vary in a predictable fashion. In patients with atrial tachycardia, a variable Ap PCL-VA curve was observed. In contrast, in all patients with other mechanisms of PSVT, a flat or upsloping curve was seen, providing evidence that atrial activation following the cessation of ventricular pacing was dependent upon antegrade activation through a pathway producing ventricular depolarization and helping to distinguish from other mechanisms of PSVT from atrial tachycardia. These findings confirm that the ability to dissociate ventricular activation from PSVT is helpful in diagnosing atrial tachycardia. Although this finding has been previously alluded to,[11] its incidence and a rapid method for using it in establishing a diagnosis have not been described in the past.

Ventricular overdrive pacing was successful in terminating atrial tachycardia in only one of ten patients whereas it was uniformly successful in terminating other mechanisms of PSVT. This is likely because in all patients with atrial tachycardia, VA block developed with ventricular overdrive pacing. In addition, all patients with AV junctional reentry or AV reentrant tachycardia had a flat or upsloping VA versus pacing cycle length curve whereas those with atrial tachycardia had a variable curve demonstrating that PSVT was not dependent on ventricular activation in patients with atrial tachycardia.

Other diagnostic techniques have proved useful in distinguishing atrial tachycardia from other mechanisms of PSVT. The ability of a ventricular premature stimulus to terminate PSVT without retrograde conduction to the atrium effectively rules out the diagnosis of atrial tachycardia; however, this finding is uncommon.[14] The infusion of intravenous adenosine has proved useful in a differential diagnosis of supraventricular tachycardia.[19] Adenosine is highly effective in terminating reentrant arrhythmias involving the AV node (AV nodal reentrant tachycardia and AV reentrant tachycardia) but not as effective in terminating atrial tachycardia. However, adenosine can terminate atrial tachycardia[20,21] and thus it has not eliminated the need for other diagnostic techniques to differentiate among PSVT mechanisms. The present study

was not designed to compare the use of overdrive pacing to intravenous adenosine in differentiating among PSVT mechanisms.

Distinguishing Among Other PSVT Mechanisms

The response to atrial and ventricular pacing was less helpful in distinguishing among AV reentrant tachycardia and the typical and atypical forms of AV junctional reentry. However, several observations were occasionally useful. In 11% of the patients with the atypical form of AV junctional reentry, atrial pacing at a given cycle length produced antegrade Wenckebach that resulted in termination of PSVT followed by 1:1 conduction with a shorter AH interval. In this instance, removal of retrograde conduction over the fast AV nodal pathway by termination of PSVT allowed antegrade conduction with a shorter AH interval to resume. This change in activation time when conduction occurs via a different pathway after tachycardia termination has been described as the third criterion for entrainment.[6] This phenomenon was likely seen only infrequently in patients with typical AV junctional reentry because in those patients the antegrade block cycle length of the fast AV nodal pathway is longer than the block cycle length in the slow AV nodal pathway.

A similar observation was made in patients with AV reentry who had manifest preexcitation. After termination of narrow complex PSVT, during atrial pacing antegrade conduction over the accessory AV connection with preexcitation reappeared. However, this finding did not provide diagnostic information that was not present on the surface ECG.

Other Observations

Several additional observations that were not helpful in distinguishing the mechanism of PSVT may have pathophysiological implications. The retrograde limb of the tachycardia in the typical form of AV junctional reentry and AV reentrant tachycardia behaved similarly in that decremental conduction was rarely observed. As expected, the AV nodal pathway that served as the antegrade limb in the typical form of AV junctional reentry and AV reentrant tachycardia showed evidence of decremental conduction in most patients. The antegrade and retrograde limbs of the tachycardia in the atypical form of AV junctional reentry showed evidence of prolonged conduction times with more rapid pacing rates. This confirms prior studies that suggest that AV nodal pathways in this tachycardia both behave in a decremental fashion.

Relationship to Studies on Entrainment

Waldo et al.[2] initially described three criteria for demonstrating entrainment in patients with AV reentrant tachycardia. The criteria were: (1) constant fusion of the entrained complexes with a morphology intermediate between pacing and that of the spontaneous tachycardia when pacing at a fixed rate; (2) progressive fusion at more rapid pacing cycle lengths; and (3) a change in activation time following termination of the tachycardia. In that initial report, atrial pacing terminated PSVT in all 15 patients. In subsequent studies, the importance of the pacing site in determining the ability to demonstrate entrainment and a fourth criterion for entrainment based on local electrogram morphology were also described.[7,10] However, although the latter study involved one patient with atrial tachycardia, the response of different mechanisms of PSVT to atrial and ventricular pacing was not analyzed.

Other investigators[22,23] have also examined entrainment during PSVT, but none of these studies has systematically examined the response of PSVT of various mechanisms to atrial and ventricular pacing.

In the present study, although entrainment was frequently seen, no attempt was made to systematically examine the presence or absence of entrainment. In patients with AV junctional reentry and AV reentrant tachycardia, all recorded electrograms were generally advanced to the pacing rate. However, the primary focus of the present study was to examine the ability of atrial and ventricular pacing to distinguish among tachycardia mechanisms that did not depend a priori on the presence of entrainment. The third criteria for entrainment was useful in diagnosing the typical form of AV junctional reentry as noted above.

Limitations

A limitation of this study is that the overdrive pacing technique was not prospectively compared to other methods of establishing the mechanism of PSVT. In addition, only on a relatively small number of patients with the atypical form of AV junctional reentry and with accessory AV connections with decremental properties were included in the study.

Clinical Implications

Previously described electrophysiologic techniques are adequate for easily determining the mechanism of PSVT in the majority of cases. However, diagnostic dilemmas that may be time consuming to resolve do exist. The present study demonstrates that atrial and ventricular overdrive pacing can differentiate atrial tachycardia from other mechanisms of PSVT and can occasionally be helpful in suggesting a mechanism among other causes. Although the relative value of this technique compared to other techniques that may help distinguish among PSVT mechanisms was not established in the present study, it may be useful, especially in cases in which adenosine infusion does not establish the PSVT mechanism with certainty.

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