Electrophysiologic Mechanisms for Modification and Abolition of Atrioventricular Junctional Tachycardia with Simultaneous and Sequential Atrial and Ventricular Pacing

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SUMMARY. The role of simultaneous and sequential atrioventricular (AV) stimulation in prevention of tachycardia induction, and the underlying electrophysiologic mechanisms involved, were studied in 10 patients with documented paroxysmal reentrant supraventricular tachycardia (PSVT). Reentry circuit was localized to the AV node in seven of 10. The remaining three cases had Wolff-Parkinson-White (WPW) syndrome and the reentrant circuit retrogradely incorporated an accessory pathway. Progressively earlier atrial premature beats (A2) were introduced while the basic cycle length (BCL) consisted of either atrial pacing alone or simultaneous A and V pacing. Compared with atrial pacing alone, simultaneous A and V pacing during the basic drive completely abolished the PSVT zone in two of seven with AV nodal reentrant PSVT, narrowed the zone in four of seven and facilitated PSVT induction in the other patient. In all patients with AV nodal reentry, significant shortening of AV nodal refractoriness caused by simultaneous antegrade and retrograde AV nodal excitation was responsible for the results. In the three patients with ventricular preexcitation, simultaneous A and V pacing abolished or shortened the PSVT zone in the two cases with type B WPW, but had no effect in the other case (with type A WPW). Introduction of programmed ventricular premature complexes after A2 capable of initiating PSVT (sequential AV premature stimulation) prevented PSVT induction by prematurely interrupting the reentry pathway in all instances. Sequential AV premature stimulation was more effective in preventing PSVT when the BCL consisted of simultaneous A and V pacing than was atrial pacing alone. We conclude that simultaneous or sequential AV pacing can either abolish or significantly narrow the PSVT zone, although paradoxical facilitation of reentrant process can occur.

THE ROLE OF ATRIAL and ventricular stimulation in patients with paroxysmal atrioventricular (AV) junctional reentrant supraventricular tachycardia (PSVT) has been extensively studied.\textsuperscript{1-10} We have learned that appropriately timed single or multiple atrial or ventricular premature complexes can initiate or terminate PSVT in most of these cases. Data so far have been used primarily for diagnostic purposes to delineate the mechanisms and reentrant circuits and to evaluate the effect of therapeutic agents in the laboratory setting. At times, cardiac stimulation with permanently implanted pacemaker systems has been used to terminate the PSVT in selected cases considered resistant to drug therapy.\textsuperscript{11-13}

Prevention of PSVT, a more desirable goal, has only rarely been attempted or achieved with the use of appropriate programmable permanent pacemaker systems.\textsuperscript{14-17} Few systematic studies have been carried out to determine if induction of PSVT can be prevented in the laboratory by changing the functional properties of the cardiac conduction system by modifying the cardiac stimulation techniques. The present study was designed to test this possibility in 10 patients with recurrent PSVT, seven of whom had reentry localized to the AV node, while the other three cases had reentry circuit involving an accessory pathway of the Kent bundle type. We present initial observations in these patients and discuss the possible electrophysiologic mechanisms. In addition, clinical implications of the electrophysiologic findings in these patients are discussed.

Materials and Methods

Electrophysiologic studies were performed in a postabsorptive, nonsedated state. The nature of the procedure was explained to all patients and signed consents were obtained. Quadripolar electrode catheters were percutaneously introduced into peripheral veins, fluoroscopically guided and positioned in the region of the tricuspid valve, high right atrium (HRA), coronary sinus and right ventricle. The catheters were used for local intracardiac electrogram recordings and/or stimulation using techniques previously described.\textsuperscript{18} All intracardiac electrograms, surface ECG leads I, II, V\textsubscript{1}, and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on an FM tape recorder. The records were subsequently retrieved for analysis on a paper at a speed of 100-250 mm/sec. The intracardiac stimulation was performed with a digital stimulator capable of delivering rectangular impulses of 10-V amplitude and < 2-msec duration. During these studies patients were isolated and all electrical equipment was grounded to a common point free of any ground loops.

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In all patients, antegrade (ant) and retrograde (ret) conduction and refractory period studies were performed. The right atrium or coronary sinus and right ventricle were the sites of stimulation. During refractory period studies, the basic atrial or ventricular cycle lengths (A1A1, or V1V1) were scanned with either atrial or ventricular premature beats (A2 or V2) introduced after every eighth beat of the basic drive. The coupling intervals (A1A2 or V1V2) were progressively shortened until refractoriness of the atrium or ventricle occurred. The functional properties of the AV conduction system and initiation of PSVT were studied in all patients using different protocols of cardiac stimulation, which we numerically designated as methods 1–6 as follows:

1) Premature stimulation from atrium (A2) with the basic cycle length (BCL) consisting of atrial pacing alone.

2) Scanning with A2 while the BCL consists of simultaneous atrial and ventricular pacing.

3) Scanning with A2 while the BCL consists of ventricular pacing alone.

4) Premature stimulation from the ventricle (V2) while the BCL consists of atrial pacing alone.

5) Scanning with V2 while the BCL consists of ventricular pacing alone.

6) Scanning with V2 while the BCL consists of simultaneous atrial and ventricular pacing.

In three of seven patients with AV nodal reentry and in all three patients who had reentry via the accessory pathway (AP), the effect of sequential AV premature stimulation was also studied while the BCL consisted of either atrial pacing alone or simultaneous atrial and ventricular pacing. During sequential AV premature stimulation the A1A2 intervals were kept constant and within the range where A2 consistently initiated PSVT, and only the coupling interval of ventricular premature beat (Sp or Vp) relative to the last basic drive beat was altered.

Definition of Terms

A complete set of definitions for ant and ret conduction and refractory periods have been previously published.9,18 Some pertinent definitions used repeatedly in this report are:

AH interval: Measured from the onset of low atrial deflection to the beginning of His bundle potential during sinus beats and used to estimate AV nodal conduction time. During atrial-paced beats the measurements were made from the onset of the stimulus artifact (S) as well as from the low atrial electrogram to the His bundle (HB) deflection (SH and AH intervals, respectively). Since the exact beginning of low atrial electrogram during paced beats was sometimes difficult to detect, we preferred the SH (rather than AH) intervals for comparative analysis unless there was local stimulus to atrial response latency.

Atrial echo response (Ae): Represents reexcitation of the atrium from the reentrant impulse either via the AV node or the accessory pathway.

Echo or PSVT zone: The range of A2A2 where A2 resulted in sustained PSVT.

H-Ae and Ae-H intervals: Measured from the onset of respective deflections on the His bundle electrogram tracing during an episode of PSVT.

During ventricular premature stimulation when the ret HB deflection (H2) emerged from local V2 electrogram, the ret His-Purkinje system (HPS) and AV nodal conduction were measured in the following manner:

Retrograde HPS conduction times: Measured from the onset of stimulus artifact to the end of ret H2 deflection (S2H2 or V2H2 interval).

Retrograde AV nodal conduction times: Both measured from the onset as well as the end of the HB deflection to the onset of ret low atrial electrogram (H2A2 interval).

Results

All patients had a history of recurrent rapid heart action and electrocardiographic documentation of PSVT. The five male and five female patients ranged in age from 30–69 years and had a known duration of arrhythmias of 5–20 years. Eight of the 10 patients had no clinically detectable cardiac disease and two with AV nodal reentry had previously suffered myocardial infarction. At the time of study all patients were in sinus rhythm and were not taking any cardioactive medications. Patients with AV nodal reentry had normal PR intervals and intraventricular conduction, and patients with ventricular preexcitation had classic electrocardiographic features of Wolf-Parkinson-White syndrome (WPW). Although in eight of 10 patients the PSVT could also be initiated during incremental atrial pacing, only the findings during refractory period studies with the different stimulation methods outlined earlier are presented here. The results in patients with AV nodal reentry (seven patients) and those with reentry via the accessory pathway (three patients) are presented separately.

PSVT Due to AV Nodal Reentry

In all seven patients the AV nodal reentrant nature of the arrhythmia was established during the electrophysiologic studies using previously published criteria.5,6,9 The PSVT could be consistently initiated in all except patient 7* with introduction of single atrial premature beats (A2) within a narrow range of atrial coupling intervals when the BCL consisted of atrial pacing alone (method 1) (table 1). The BCL, zone of PSVT and the critical degree of SH (AH) delays needed to start the tachycardia are summarized in table 1. During PSVT, the H-Ae intervals were significantly shorter than the Ae-H intervals at different cycle lengths of the tachycardia. The ant and ret conduction characteristics during PSVT in these

*In patient 7 the PSVT was inducible with incremental atrial pacing.
patients were typical of most patients with this arrhythmias that have been described previously, and therefore are not detailed here. With method 1, discontinuous AV nodal refractory period curves (H1H2 and A2H2 intervals in response to progressively shorter A1A2 intervals) were noted in three of seven patients (1, 2 and 6) at the BCL presented in table 1. The other four patients had smooth curves.4

After control studies with the conventional technique of atrial premature stimulation (i.e., method 1), the initiation of PSVT was attempted with method 2 and the entire cycle length was scanned with progressively shorter A1A2 intervals. In patients 1 and 2 (table 1), the PSVT zone was completely abolished with method 2 (fig. 1); with method 1 the PSVT zones for these two patients were 60 and 30 msec, respectively. The ERP of the AV node was not found in either patient; however, for comparable A1A2 intervals, the S2H2 delays were significantly less with method 2 than method 1 (fig. 1, panels A and B) in both cases. Atrial refractoriness, which determined the inner limits of PSVT zone during method 1 in both patients, showed no significant change after method 2. In patients 3–6 the induction of PSVT was not completely abolished with method 2, although the PSVT zone was shortened in all four (table 1). The critical S2H2 (A2H2) delays resulting in initiation of PSVT with method 2 were similar to those with method 1 in patients 4, 5 and 6. In patient 3 the PSVT could be initiated at shorter S2H2 delays with method 2 compared with method 1. The PSVT cycle length ranges (i.e., Ae-Ae intervals) were comparable in all cases with both methods of stimulation. The ERP of the AV node could be determined during method 1 in all four patients and showed an appreciable decrease in patients 5 and 6 (table 1). In patients 3 and 4 the values for the ERP of the AV node and functional refractory period of the atrium were too close to make a valid comparison of the effect of method 2 on the AV nodal ERP. The S2H2 delays at comparable A1A2 intervals were shorter in all instances with method 2 than method 1. The atrial refractoriness was tested in three of these four patients and showed no significant change after method 2 (table 1).

Since method 2 was not entirely successful in preventing PSVT in patients 3–6, programmed ventricular beats (Sp or Vp) after A2 were added to the pacing protocols of methods 1 and 2 within the PSVT zones to test the feasibility of preventing PSVT induction with these techniques (fig. 2). In patients 3–5 an appropriately timed Vp prevented PSVT induction when initiated with method 2 (fig. 2). However, introduction of Vp failed to prevent PSVT induction in patients 4 and 5 when the arrhythmia was initiated with method 1 (fig. 2). As indicated in the Methods section, the effect of sequential AV premature stimulation was not studied in the remaining four of seven patients with AV nodal reentrant PSVT.

The arrhythmia could not be induced by method 1 in patient 7, who had prior electrocardiographic documentation of PSVT and in whom PSVT could be initiated with incremental atrial pacing. However, method 2 initiated the PSVT through a zone of 60 msec (fig. 3 and table 1). The PSVT showed alternation of cycle length with an overall range between 410–490 msec. The S2H2 delays at comparable A1A2

### Table 1. Electrophysiologic Data in Patients with Atrioventricular Nodal Reentrant Paroxysmal Supraventricular Tachycardia

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<th>Method of stimulation</th>
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<th>Critical S2H2</th>
<th>Longest S2H2</th>
<th>Cycle length of PSVT</th>
<th>H-Ae intervals</th>
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All values are in msec.

Abbreviations: BCL = basic cycle length; PSVT = paroxysmal supraventricular tachycardia; ERP = effective refractory period; AVN = atrioventricular node; FRP = functional refractory period.
FIGURE 1. Patient I. Tracings are standard ECG lead 1, 2, V1, high right atrial (HRA) and His bundle (HB) electrograms, and time lines (T). Panel A shows initiation of atrioventricular (AV) nodal reentrant PSVT at a basic cycle length (BCL) of 650 msec and an A1A2 of 270 msec during pacing from the HRA (white arrow) (method 1). The S1H1 interval during the basic drive and S2H2 interval initiating the paroxysmal supraventricular tachycardia (PSVT) measure 155 and 480 msec, respectively. Note a relatively short H-Ae (Ae on HB is obscured by local ventricular electrogram) and significantly longer Ae-H interval during PSVT. At the same BCL and A1A2 interval as panel A, the S2H1 interval in panel B is significantly shorter when the basic drive consists of simultaneous HRA (white arrow) and ventricular (black arrow) pacing (method 2). PSVT could not be induced with method 2 at a comparable atrial coupling interval (panel B) and subsequently at shorter A1A2 intervals (not shown) up to the point of atrial effective refractory period. The PSVT could be initiated by omitting the ventricular stimulus from the last beat of the basic drive (panel C) at the same atrial coupling interval of 270 msec as panels A and B. The S1H1 and S2H1 in panel C measure slightly less than the corresponding intervals in panel A, which can be explained by longer time available for AV nodal recovery of excitability produced by simultaneous antegrade and retrograde excitation of the AV node during the preceding beat. The magnitude of change in S2H1 with omission of ventricular pacing for only one cycle (panel C compared with panel B) is unlikely to be the result of sympathetic stimulation from ventricular pacing during method 2.
FIGURE 2. Patient 4. The effect of programmed ventricular premature stimulation in atrioventricular (AV) nodal reentrant paroxysmal supraventricular tachycardia (PSVT) when the arrhythmia could be initiated both with method 1 (panels A and B) and method 2 (panels C-F). With method 1 the PSVT is induced at $A_1A_2$ of 300 msec at a basic cycle length (BCL) of 600 msec (panel B). Introduction of programmed ventricular premature stimulus (Sp) after $A_2$ produces a ventricular response (Vp) that retrogradely activates the atria (Ar) (panel A) before the expected atrial echo response (Ae) in panel B, i.e., $A_2$-Ar in panel A measures 30 msec shorter than $A_2$-Ae (panel B). Despite ret atrial capture with Vp the PSVT is not abolished (panel A). This is probably due to relatively late engagement of ret pathways by Vp which permitted the premature ventricular impulse to propagate along the cross-connecting fibers and ant pathways to initiate PSVT. Shortening in $S_2$-Sp and $V_2$-Sp by 20 msec (panel B) results in no Vp, and ventricular refractoriness limits abolition of PSVT by Vp when the arrhythmia is initiated with method 1. At the same BCL of 600 msec the PSVT is initiated at an $A_1A_2$ interval of 280 msec (panel C) with method 2. The BCL and the $A_1A_2$ intervals are kept constant (panels C-F) and Vp are introduced at progressively shorter $V_2$-Sp intervals in panels D-F. At a $V_2$-Sp of 480 msec (panel D), Vp results in ret atrial capture which is relatively late ($A_2$-Ar = 365) and the PSVT is initiated. At a closer $V_2$-Sp of 450 (panel E) the $A_2$-Ar measures 330 msec; the Ar antegrade blocks in the AV node and the PSVT is abolished. Further shortening of $V_2$-Sp (panel F) results in loss of ret atrial capture (No Ar) and the Vp continues to be effective in abolishing PSVT at this and shorter $V_2$-Sp intervals (not shown). Abbreviations: see figure 1.

and the maximum $S_2H_2$ delays achieved with method 2 were significantly longer than those achieved with method 1. The ERP of the AV node markedly decreased with method 2, but the atrial refractoriness was the same with either method.

The PSVT could not be initiated in any patient with methods 3–6.

We noted no appreciable difference in the ret conduction delays in the HPS (ret $V_2H_2$ intervals), whether the BCL consisted of ventricular pacing alone (method 5) or simultaneous atrial and ventricular pacing (method 6). At comparable $V_1H_2$ intervals the ret AV nodal conduction delays ($H_2A_2$ intervals) also showed minimal differences (i.e., < 5 msec) with the two methods except in patient 1, who had a decrease in ret $H_2A_2$ interval of 10 msec with method 6 compared with method 5.

PSVT Due to Reentry Via the Accessory Pathway

Some of the stimulation methods were tested in several patients with WPW. However, only three
patients (one with type A and two with type B ventricular preexcitation) were tested by all the methods. They are presented here.

The first case was a 46-year-old female who has had type B ventricular preexcitation for several years. Electrophysiologic studies revealed right-sided AV AP of the Kent bundle type. The precise location could not be determined with the catheter technique. During high right atrial pacing at a BCL of 600 msec, the ERP of the AP was determined at an A1A2 of 270 msec with method 1, which coincided with initiation of PSVT (fig. 4, panels A and B). The PSVT consistently occurred over a zone of 80 msec (270–200 msec) until the ERP of the AV node was encountered at an A1A2 interval of 190 msec. During PSVT the Ae response on the HB electrogram tracing occurred earlier than in the left atrium (CS) and HRA. With simultaneous pacing from the HRA and right ventricle during the basic drive (method 2), we noted a significant shortening of the ERP of the AP (fig. 4, panels C and D), but the PSVT started when A2 blocked in the AP. Induction of PSVT with method 1 could, however, be easily prevented with introduction of programmed ventricular premature complexes (Sp or Vp) through V1-Vp intervals measuring 425 msec or less (fig. 5), until the ERP of the ventricle was encountered. Similarly, induction of PSVT with method 2 could also be abolished with introduction of Vp at appropriate times.
after A2. The latter findings, however, were not considered conclusive because the zone of PSVT with method 2 was rather narrow (10 msec).

The second patient with type B ventricular preexcitation and a right-sided AV accessory pathway had a PSVT zone of 100 msec during high right atrial pacing with method 1. With method 2 the ERP of the AP was shortened to a value less than the atrial ERP and the PSVT zone was completely abolished. The induction of PSVT with method 1 could be easily prevented with programmed VP, as noted in the other patient with type B ventricular preexcitation.

The third patient (a 59-year-old male) had type A WPW and a left-sided AV AP as determined by the various electrophysiologic criteria. With method 1 (the coronary sinus was the site of stimulation), the A2 blocked within the AP at an A1A2 interval of 270 msec at a BCL of 700 msec (fig. 6, panels A and B), and PSVT could be initiated over a zone of 70 msec (270-210 msec). During the tachycardia, left atrial activation (from the region of mid- to distal CS) preceded both those of the low atrial septum (HB electrogram) and the HRA (fig. 6, panels B and D). With method 2 (simultaneous pacing from coronary sinus and right ventricular apex) no significant change in the ERP of the AP or the zone of PSVT was noted (fig. 6, panels C and D). Abolition of PSVT with VP during method 1 was not successful (fig. 7, panels A and B) since the ventricular ERP prevented sufficiently early engagement of the AP or the atria by VP. Interruption of reentry circuit with VP was facilitated by simultaneous AV stimulation during the basic drive (method 2) and the PSVT initiation could be easily prevented (fig. 7, panels C and D). PSVT could not be initiated with methods 3–6 in either of the patients with WPW.

**Discussion**

Simultaneous and sequential atrial and ventricular pacing can prevent the so-called chronic form of AV junctional reentrant tachycardia.21, 22 The results of

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**Figure 4.** The basic cycle length (BCL) is constant at 600 msec. During atrial premature stimulation from the high right atrium with method 1 (panels A and B), the A2 blocks in the accessory pathway (ERP-AP) at an A1A2 interval of 270 msec (panel B) and PSVT is initiated. During method 2 (panels C and D) the ERP of the AP shows a decrease and the block of A2 in the AP coincides with initiation of the tachycardia (panel D). Although some degree of stimulus to atrial response latency may have existed (difficult to detect) at such close S1S2 intervals seen in panels C and D, the A1A2 intervals are still significantly shorter in panels C and D compared to panels A and B; yet the S2H2 interval in panel D (220 msec) measures less than in panel B (260 msec). The last beats in panels A and C are reference sinus beats and demonstrate atrial activation sequence and type B ventricular preexcitation. Note that during the tachycardia the activation of the low right atrial septum (HB) precedes that of coronary sinus (CS) and high right atrium (HRA).
this study indicate that sustained arrhythmia of the paroxysmal variety can also be prevented with appropriate stimulation methods. Our findings also suggest that individual responses can be variable, and a systematic search in the laboratory for the optimal stimulation technique that abolishes PSVT is desirable for selection of proper pacemaker therapy. To discuss the clinical implication of these findings it is important to analyze the underlying electrophysiologic mechanisms responsible for our results.

Modification of PSVT Initiation with Simultaneous AV Pacing During the Basic Drive

The total abolition of AV nodal reentrant PSVT or a decrease in the PSVT zone in patients 1–6 with method 2 suggests that simultaneous A and V pacing altered the functional properties of the AV node. Abolition and modification of PSVT zones in patients with AV nodal reentry can best be explained by the effect of such pacing on the ant AV nodal conduction and refractoriness. Simultaneous excitation of the AV node from the two impulses resulted in significant shortening of the AV nodal refractoriness such that critical degrees of S2H2 delays could not be achieved at comparable atrial coupling intervals. In patients 1 and 2, the S2H2 delays that initiated PSVT with method 1 could not be reached with method 2, but in the other four patients (3–6) the critical S2H2 intervals were obtained at shorter A1A2 intervals with method 2, shifting the PSVT zone to the left. When critical degrees of AV nodal conduction delays were achieved, the PSVT could be initiated in patients 4–6. Although induction of PSVT in case 3 appeared to occur at shorter S2H2 delays with method 2 than method 1, a valid comparison could not be made, since comparable S2H2 intervals were not available with the two methods of stimulation. The seemingly paradoxical findings in patient 7 can also be explained by shortening of AV nodal ERP with method 2, which allowed attainment
of longer $S_2H_2$ delays not achieved with method 1. Shortening of AV nodal refractoriness with atropine may also produce similar effects, and such observations have been reported previously.\textsuperscript{23}

The classic concept of AV nodal reentry implies that both critical atrial coupling intervals and intranodal delays are necessary to bring about the necessary functional dissociation of pathways to initiate AV nodal reentrant process. The exact $A_1A_2$ interval where $A_2$ blocks in a given AV nodal pathway is difficult to determine, particularly in the absence of demonstrable discontinuous refractory period curves. Since simultaneous AV pacing may shorten refractoriness of all intranodal pathways, we could not determine the effect of such pacing on pathways where $A_2$ blocks vs those along which the $A_2$ conducts. We do not know whether the shortening of AV nodal refractoriness was the result of simultaneous ant or ret excitation of the AV node or sympathetic stimulation from ventricular pacing during method 2.\textsuperscript{24} However, the ability to induce PSVT with elimination of ventricular pacing during the last beat of the basic drive (fig. 1, panel C) suggests that simultaneous AV nodal excitation during method 2 was primarily responsible.

This modification of stimulation technique (fig. 1, panel C) was tried in patients 1-3 and 6, with essentially the same results. Withdrawal of sympathetic stimulation due to ventricular pacing would not be expected to occur within one cardiac cycle. The definitive mechanism for decreased AV nodal refractoriness produced by simultaneous ant and ret activation with method 2 is not known. However, several explanations may be offered:

1) Activation of the AV node from the opposing electrical wave fronts will be expected to shorten the time needed for AV nodal depolarization and recovery of excitability. The shortening of ant AV nodal refractoriness by ventricular impulse occurring simultaneously or earlier than atrial impulse has also been previously demonstrated in animal studies.\textsuperscript{25, 26}

2) Prior ret activation of some portion of the AV node could alter the responsiveness to subsequent ant impulse propagation. Propagation of premature atrial beats may be facilitated by prior ret depolarization of the AV node. However, this cannot be proved.

Regardless of the mechanism for shortening of AV nodal refractoriness, the findings do suggest that collision of the ant and ret impulses took place in the AV

**Figure 6.** Premature atrial stimulation from coronary sinus with method 1 (panels A and B) and method 2 (panels C and D). With both methods the effective refractory period of the accessory pathway is encountered at 270 msec (panels B and D), and paroxysmal supraventricular tachycardia (PSVT) is initiated. Atrial activation sequence and the type of ventricular preexcitation (type A) during sinus beats are shown in the last beats in panels A and C. At the same basic cycle length (BCL) and atrial coupling intervals, the $S_2H_2$ is slightly longer with method 1 (panel B) compared with method 2 (panel D). During the tachycardia, left atrial activation (CS) precedes both low and high right atrial depolarization. The cycle lengths of PSVT with the two methods of stimulation are comparable.
node. This is also supported by the fact that ret $V_2 H_2$ delays at comparable BCL and $V_1 V_2$ intervals measured the same with methods 5 and 6, suggesting that activation of the HB during simultaneous AV pacing occurred from the V rather than the A impulse. The ret SH intervals during method 2, therefore, measured less than the ant SH intervals during method 1, resulting in a collision of the two impulses in the AV node. The level of intranodal collision could vary, depending on the conduction velocity of the two impulses and the sites of stimulation within the A and V. Although not observed during the present study, it is reasonable to postulate that if depolarization of the HB occurred from the A impulse during method 2, no significant effect on PSVT zone would be noted with this method. This could occur in patients with extremely short ant AH intervals, or in patients with complete ret right bundle branch block (RBBB) where paced right V impulse will activate the HB via the left bundle branch and ret SH could exceed the ant SH intervals. Therefore, in patients with ret RBBB simultaneous pacing from atrium and right ventricle should not be expected to alter the PSVT zone when the arrhythmia is initiated with single A premature complexes.

Since method 2 significantly changed the ant AV nodal refractoriness, which could explain results in patients 1-7, we could not judge the role of this method in alteration and abolition of the PSVT zone by affecting the functional properties of the ret AV nodal pathways or cross-connecting fibers between ant and ret pathways. The ability to induce PSVT in patients 4-6 with both methods 1 and 2 when comparable ant SH delays were achieved indicates that effect of method 2 on ret AV nodal pathways was not sufficient to prevent PSVT. Also, direct comparison of ret AV nodal conduction times ($H_2-A_2$ intervals) with methods 5 and 6 showed that simultaneous AV stimulation did not significantly affect the $H_2-A_2$ intervals except in patient 1. However, neither method 5 nor 6 frequently permit adequate study of ret AV nodal conduction, since the input of two consecutive ret impulses ($H_1H_2$) at sufficiently close coupling intervals is generally limited by ret refractoriness of the

**Figure 7.** Same patient as in figure 6. The basic cycle length (BCL) is 700 msec. The tracings in panel A show inability of $V_p$ to abolish paroxysmal ventricular tachycardia (PSVT) when initiated with method 1, although ret atrial capture ($A_r$) via the $A_p$ is accomplished with $V_p$ ($S_2-A_r$ in panel A measures 70 msec < $S_2-A_e$ in panel B). With a 10-msec decrease in $V_1-S_p$ interval from 260 msec (panel A) to 250 msec (panel B), the $S_p$ does not evoke a ventricular response. Introduction of $V_p$ during method 2 allowed achievement of earlier ret atrial capture (compare $A_r-A_r$ in panels A and C) via the $A_p$ and PSVT is abolished. The $V_p$ retrogradely blocked in both pathways, i.e., normal and $A_p$ at $V_1-S_p$ interval of 300 msec (panel D). Tachycardia could be prevented over a significantly wide range of $V_1-S_p$ intervals with method 2 of stimulation as seen in panels C and D.
HPS. A precise comparison of the effect of method 2 on ant vs ret AV nodal pathways in modification of the reentry process, therefore, cannot be made.

A decrease in AV nodal refractoriness with method 2 was also noted in the two of three patients with ventricular preexcitation (fig. 6, panels B and D), but it did not seem to affect the PSVT induction in these cases. The inability of method 2 to alter the zone of PSVT in the patient with type A preexcitation was not unexpected and was caused by remoteness of the AP in relation to the site of ventricular pacing such that depolarization of the AP during the BCL occurred from A impulse both during method 1 and 2. The shortening in the ERP of the AP noted in patients with type B ventricular preexcitation was surprising, and was probably due to partial or complete ret activation of the AP during method 2, with resultant change in responsiveness of the AP to the A premature beat.

Abolition of PSVT Initiation with AV Sequential Premature Stimulation

Although the induction of PSVT could not be entirely prevented in most patients with simultaneous AV stimulation during the basic drive, AV sequential premature stimulation successfully abolished PSVT induction in all patients in whom the technique was tested. AV sequential premature stimulation, with the BCL consisting of atrial pacing alone, has been used to demonstrate so-called dual AV nodal pathways in patients with PSVT. To abolish PSVT induction, however, sequential AV premature stimulation was more effective if the BCL consisted of simultaneous AV pacing rather than A pacing alone. Propagation of prior impulse in the same direction as Vp, as in method 2, facilitates achievement of closer coupling intervals at the level of AV junction. With the preceding impulse conducting in the opposite direction (as with method 1), achievement of coupling intervals that can interrupt AV junctional reentry is frequently limited by refractoriness of the ventricular muscle or HPS (figs. 2 and 7). Although the ventricular ERP was generally longer during atrial (method 4) compared with ventricular pacing (Methods 5 and 6), the actual difference in ventricular ERP with these methods could not account for the inability of Vp to abolish PSVT induced with method 1. This is clearly demonstrated in fig. 7, which shows abolition of PSVT at longer V1-Sp intervals when the BCL consists of simultaneous AV pacing (panels C and D) compared with atrial pacing alone (panel A). AV sequential premature stimulation abolished AV nodal reentrant PSVT primarily by retrogradely preexciting the AV node or atrium. Early excitation of the AV node or atrium before the cross-connecting fibers or ant pathways had recovered excitability would prevent forward propagation of the impulse and interrupt reentry. Delivery of very early Vp after A2 (with minimum A2-Vp delay) may effectively stop even the forward propagation of A2 and prevent PSVT by this mechanism.

The mechanisms of abolition of PSVT induction with Vp in patients with ventricular preexcitation include: 1) earlier excitation of the atrium, preventing forward impulse propagation through the AV node (fig. 5, panel B and fig. 7, panel C), and 2) attainment of ret block in the AP or atrium (figs. 5 and 7, panel D). Also, intranodal collision of A2 and Vp (which also occurs in patients with ventricular preexcitation) might play some role in abolishing reentry by preventing intranodal propagation of atrial impulse (Ar) when Vp results in ret atrial capture via the AP (fig. 7, panel C). If Vp fails to activate atrium (i.e., no Ar, figs. 5 and 7, panel D) via the AP or the normal pathway, initiation of reentrant process is not expected unless intranodal reciprocation coexists.

Although not directly tested during the present study, AV sequential premature stimulation would also be expected to effectively abolish PSVT induction if sequential (with a short AV interval) rather than simultaneous AV pacing preceded the A2 initiating the tachycardia.

The inability of stimulation methods 3–6 to initiate PSVT suggests: 1) AV nodal reentrant PSVT can rarely be initiated with single premature ventricular complexes, whether delivered during paced atrial or ventricular drives. Similar observations have been made previously. 2) It is difficult to initiate PSVT with a single A2 during paced ventricular rhythm in the presence of intact ventriculoatrial (VA) conduction. Intact ventriculoatrial conduction is generally the rule in patients with AV junctional reentrant PSVT. Inability to initiate PSVT with method 3 during the present study was primarily due to limitation imposed by refractoriness of the high right atrial region. At the closest possible A1A2 intervals in the region of the HRA with method 3, the A2H4 intervals at the level of AV junction (HB) were still not within the critical range, where initiation of PSVT would be expected. Atrial stimulation near the AV junction was not tested and, therefore, we do not know if PSVT might have been initiated if close A1A2 intervals had been achieved at the level of the AV junction with method 3.

Clinical Implications

Abolition or modification of sustained AV junctional reentrant process by programmed AV stimulation as demonstrated in this study is only an initial step toward better understanding of such a therapeutic approach. This study simply shows the effectiveness of such stimulation methods in the laboratory. The extrapolation of these findings to clinical situations should be cautious, and could pose several problems:

1) Spontaneous initiation of PSVT in these and other similar cases may be more complex (i.e., multiple atrial and ventricular premature complexes), and a better understanding is important for prevention in individual cases.

2) Only one BCL could be studied because of time
limitations imposed by the various stimulation methods. Longer or shorter BCLs might have changed the level of collision (due to changes in ant SH intervals) and produced different results.

3) Due to constantly changing autonomic tone the functional properties of the AV conduction system could vary significantly. The electrophysiologic findings obtained in the laboratory, therefore, may not be applicable to the clinical settings.

4) Concomitant use of cardioactive drugs like digitalis and propranolol could have an additional beneficial effect or could nullify the potential benefit of this therapeutic modality (i.e., AV pacing).

In the patients presented here treatment of PSVT with cardiac pacing was not needed, since conventional drugs were adequate in controlling the arrhythmia. However, our observations suggest that in properly selected patients refractory to drug therapy such an approach with appropriate pacemakers could be beneficial. As illustrated by patient 7, cardiac pacing might also aggravate the reentrant process if spontaneous or drug-induced prolongation of AV nodal refractoriness prevents PSVT.

References


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