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Research Article

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ABSTRACT The precise mechanisms for paroxysmal reentrant supraventricular tachycardia (PSVT) initiation during right ventricular premature stimulation (V_2 method) were analyzed in 14 consecutive patients with Wolff-Parkinson-White Syndrome in whom the PSVT was inducible during retrograde refractory period studies. 9 patients had left-sided and the remaining 5 of 14 had right-sided ventriculo-atrial (VA) accessory pathway (AP). At the basic cycle lengths (V_1V_1) ranging from 550 to 900 ms (mean, 657.1±139.5), closely coupled V_2 (mean V_1V_2, 357.3±59.2 ms, range 320–500) produced retrograde His bundle (H_2) activation via the bundle branches and retrograde atrial (A_2) activation via the AP. As the V_1V_2 were further shortened, the V_2 showed a retrograde block in the His Purkinje system (HPS) and conducted to the atria via AP in 9 of 14 cases. Subsequently, the A_2 impulse conducted anterograde over the atrioventricular node-HPS to initiate a PSVT or an atrial echo response in all nine cases. In none of the patients was a PSVT induced by V_2 when the latter produced retrograde H_2 activation via the bundle branches. In 10 of 14 cases, however, the retrograde H_2 was followed by a V_3, due to macroreentry in the HPS. The V_3 in turn blocked retrogradely in the HPS while producing A_3 via the AP to initiate a PSVT or an atrial echo response in 9 of 10 cases. Retrograde block of V_2 and/or V_3 in the HPS resulted in PSVT initiation in 13 of 14 cases, whereas in the remaining 1 case the exact mechanism was not clear. In none of the patients in this series was the PSVT initiated with a retrograde block of V_2 in the atrioventricular node with or without concomitant retrograde A_2 activation via the AP. We conclude that within the ranges of cycle lengths tested, a retrograde block of V_2 and/or V_3 in the HPS is the most common mechanism for initiation of PSVT during ventricular premature stimulation in patients with the Wolff-Parkinson-White Syndrome.

INTRODUCTION

Paroxysmal reentrant supraventricular tachycardia (PSVT),[1] utilizing atrioventricular (AV) node and His Purkinje system (HPS) anterogradely, and an accessory pathway (AP) in the retrograde direction, is relatively common in patients with the Wolff-Parkinson-White (WPW) Syndrome (1–4). Several studies have demonstrated that PSVT in these patients can be initiated with atrial as well as ventricular premature beats. It has also been previously demonstrated that during ventricular premature stimulation, the ability or inability to induce PSVT depends upon the various patterns of ventriculo-atrial (VA) conduction along the normal and the AP (5–8). Although addressing themselves to the VA conduction along the normal pathway in patients with the WPW Syndrome, studies to date have not made a clear distinction between conduction along the HPS vs. the AV node, the two components of normal pathway. Since the HPS and AV node have different electrophysiological properties and therapeutic responses, a clear understanding of the role of retrograde conduction in

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[1] *Abbreviations used in this paper*: Ae, atrial echo; AP, accessory pathway; AV, atrioventricular; ERP, effective refractory period; HPS, His Purkinje system; PSVT, paroxysmal rentrant supraventricular tachycardia; VA, ventriculo-atrial; WPW, Wolff-Parkinson-White.

the HPS and the AV node in the initiation of PSVT is desirable. This report demonstrates the role of retrograde block within the HPS in the initiation of PSVT during right ventricular premature stimulation in 14 patients with the WPW Syndrome.

METHODS

Right heart catheterization was performed in a post absorptive, nonsedated state, using local anesthesia. The nature of the procedure was explained to all patients and signed consents were obtained. Quadripolar electrode catheters were percutaneously inserted into peripheral veins and positioned in the region of the high right atrium, AV junction, coronary sinus, and right ventricle. The catheters were used for local bipolar electrogram recordings and/or electrical stimulation using techniques previously described (9). Surface electrocardiogram Leads I, II, and VI, intracardiac electrograms, and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on a frequency-modulated tape. The records were subsequently retrieved for analysis at a paper speed of 100–150 mm/s. Intracardiac stimulation was performed using digital stimulator capable of delivering rectangular impulses with adjustable voltage and duration. During the conduct of these studies, patient isolation existed and all equipment was carefully grounded.

Retrograde refractory period studies were performed using ventricular extrastimulus method. Premature stimuli were introduced after eight ventricular paced beats of predetermined constant cycle length. The coupling intervals were gradually reduced by 10–20 ms until ventricular muscle refractoriness was encountered.

Definition of terms. A complete set of definitions for anterograde and retrograde conduction and refractory period studies has been previously published (9). Pertinent definitions repeatedly used in this text will be outlined here.

The A-H interval was measured from the onset of low atrial electrogram to the beginning of His bundle potential. The H-V and RB-V intervals were measured from the onset of His and right bundle branch deflections to the earliest detectable ventricular activity, either on the surface electrocardiogram or the local intracardiac tracings.

Atrial echo (Ae) response represents spontaneous retrograde excitation of the atrium exclusively via the accessory pathway occurring during nonpaced beats.

For an easier comprehension some of the terms used and electrophysiologic parameters measured during retrograde refractory period studies are graphically depicted in Fig. 1. The S_1, V_1, and A_1 represent stimulus artifact, ventricular, and retrograde atrial deflections, respectively, during the basic drive, whereas S_2, V_2, H_2, and A_2 represent stimulus artifact, ventricular, His bundle, and atrial deflections following the premature beat.

The retrograde activation in response to V_2 of the H_2 via the bundle branches and the A_2 via AP is shown in panel A. Exclusive VA conduction via the AP during both V_1 and V_2 is suggested by the sequence of atrial activation and its lack of change following V_2 despite significant delay along the normal pathway (V_2H_2 interval).

The effective refractory period (ERP) of AP (panel B) is the longest V_1V_2 where V_2 blocks in the AP.

The ERP-AV node (panel C) is the longest V_1H_2 where H_2 is not followed by A_2. V_1H_2 is taken in lieu of retrograde H_1H_2 since H_1 is generally not visible during the basic drive, and it is assumed that V_1H_1 interval is constant (9). It should be noted that ERP-AV node can only be determined after retrograde ERP of AP pathway is encountered.

ERP-HPS (panel D) is longest V_1V_2 where V_2 blocks below the His bundle recording site (i.e., HPS).

RESULTS

Anterograde and retrograde conduction and refractory period studies were performed in 22 consecutive patients with WPW Syndrome who had symptomatic PSVT. In only 14 of 22 patients was the PSVT inducible during right ventricular premature stimulation, and findings in these 14 cases form the basis of this report. In the remaining 8 of 22 cases, PSVT could not be elicited during ventricular premature stimulation for the reasons previously published (7). 12 of the 14 had ventricular preexcitation during sinus rhythm, whereas the remaining two (patients 5 and 8) had electrocardiographically concealed left sided AP. The pertinent clinical and electrophysiologic data are summarized in Table I. At the time of study all were in sinus rhythm and were not taking any cardioactive medications. Two patients had associated arteriosclerotic heart disease, and another two had mitral valve prolapse, whereas the remaining cases did not have clinically detectable structural heart disease. Although two or more basic ventricular cycle lengths were scanned in 10 of 14 patients, to simplify presentation of the results only the data from the shortest available cycle lengths in each case will be presented in detail.

Retrograde refractory period studies. Ventricular premature stimulation was initiated at V_1V_2 intervals which measured 50–100 ms less than the basic drive, and the coupling intervals were progressively shortened. During the basic drive (V_1) six patients (patients 2, 3, 5, 7, 10, and 13; Table I) demonstrated retrograde atrial depolarization exclusively via the AP, whereas fusion retrograde atrial activation was observed in the others (10). In the latter cases a change from fusion atrial activation to exclusive activation via the AP was noted as the V_1V_2 were further shortened. This change in retrograde atrial activation was noted at V_1V_2 intervals which had not yet produced emergence of a His deflection from the V_2 electrogram. At the basic cycle length range of 550–900 ms (Table I), the His bundle deflection first emerged from V_2 at V_1V_2 intervals ranging from 320–500 ms. Since in patients with retrogradely functioning AP the His bundle deflection following V_2 could result from either retrograde conduction via the bundle branches or anterograde depolarization via the AV node following A_2 activation over the AP, the behavior of His bundle activation was closely analyzed (11). In all nine patients with left-sided (cases 1–9, Table I) and 2 of 5 cases (patients 11 and 12) with right-sided VA AP, the separation of retrograde A_2 from V_2 deflection (on the His bundle electrogram tracing) was sufficient to provide a clear segment where the presence of a His deflection could be identified. In all

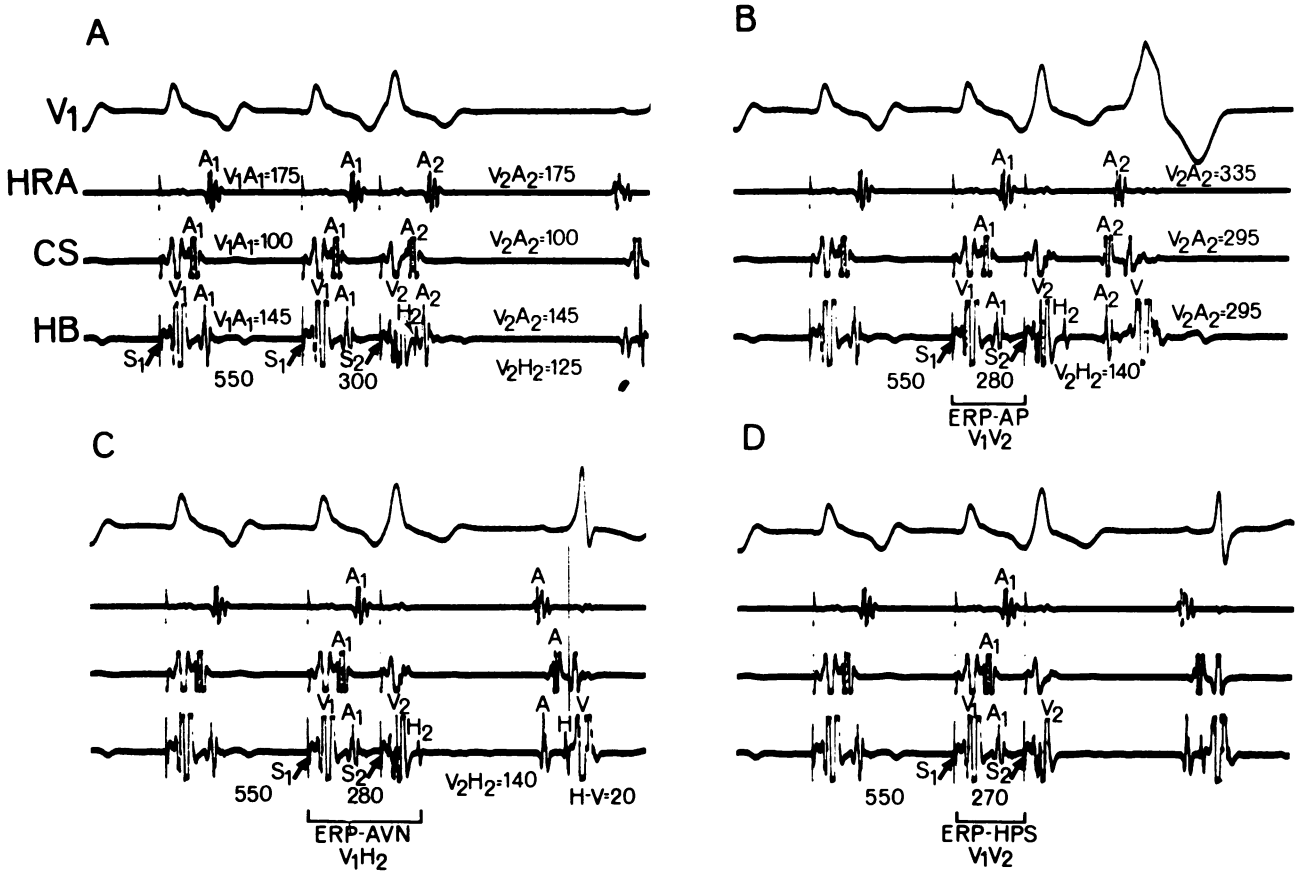

FIGURE 1 The basic right ventricular cycle length (V_1V_1) is constant at 550 ms in all panels, and progressively shorter V_1V_2 intervals are shown. Retrograde His bundle activation during the basic drive (H_1) is not identifiable, but H_2 clearly follows V_2 in panel A. The retrograde atrial activation during V_1 as well as V_2 occurs via AP, as suggested by (*a*) atrial activation sequence, and (*b*) no change in V_2A_2 compared with V_1A_1 in any of the atrial electrograms, despite significant prolongation of HPS conduction time, i.e., V_2H_2 interval along the normal pathway. In panel B, the V_2 shows a retrograde block in the AP (ERP-AP) and exclusively activates the atria via HPS/AV node. Note the change in atrial activation sequence such that A_2 electrograms on CS and HBE occur simultaneously. Subsequently, the A_2 impulse conducts to the ventricles over the AP. In panel C, the V_2 continues to block along the AP, but also shows a retrograde block in the AV node. The longest V_1H_2 interval where a block is noted defines the AV nodal ERP. At a V_1V_2 of 270 ms (panel D) the V_2 shows a retrograde block along both the AP and normal pathway. Unlike panel C, however, the site of block along the normal pathway is in the HPS (ERP-HPS). Perpendicular is drawn to indicate onset of QRS complex. Pertinent intervals are labeled. Anteriorly directed QRS complex as seen here is noted on occasions during right ventricular stimulation. V_1, surface ECG Lead V_1; HRA, high right atrial electrogram; CS, coronary sinus electrogram; HBE, His bundle electrogram.

of these 11 patients the His bundle deflection preceded the retrograde A_2 deflection (Figs. 2 and 3) during its initial appearance, indicating retrograde His bundle activation via the bundle branches (H_2); the designation of H_2 henceforth will imply such an occurrence. With further shortening of the V_1V_2 intervals, the V_2H_2 delays showed almost linear increases as long as the retrograde H_2 deflection was still identifiable and preceded or coincided with the retrograde A_2 electrogram (Figs. 2 and 3). This relationship between V_1V_2 and V_2H_2 intervals is typical of retrograde H_2 activation via the bundle branches and has been previously demonstrated in patients without the WPW Syndrome (9, 12–15).

At the V_1V_2 intervals that produced emergence of retrograde H_2 deflection, the V_2A_2 intervals via the AP showed no further prolongation once exclusive V_2A_2 conduction via the AP was previously established (Figs. 2–4) at longer coupling intervals. Upon further shortening of the ventricular coupling intervals in 8 of

TABLE I
Clinical and Electrophysiological Data

Case	Age	Sex	Structural heart disease	AP location	Retrograde effective refractory periods					Ret conduction of V_2 at close V_1V_2 intervals		Ret gap HPS	Maximum ΔVH vs. ΔV_1V_2 intervals	Macro Re-HPS (V_3)	Ret conduction of V_3	
					VCL	Vent	AP	HPS	AVN	via AP	via NP				Via AP	Via NP
	yr						*ms*						*ms*			
1	19	F	None	L	600	230	<240	330‡	—	+	B-HPS‡	+	125/10‡	—	—	—
2	20	M	None	L	700	240	<250	320‡	—	+	B-HPS‡	—	80/20‡	—	—	—
3	31	M	MVP	L	600	<260	<260	310‡	—	+	B-HPS‡	+	85/10‡	+	+	B-HPS‡
4	40	M	None	L	900	250	<260	310‡	—	+	B-HPS‡	+	145/10‡	+	+	B-HPS‡
5	38	M	None	L	550	230	280	300‡	455	+ and B-AP	B-HPS‡ B-AVN	+	150/10‡	+	+	B-HPS‡
6	35	M	None	L	700	240	<250	340‡	—	+	B-HPS‡	+	115/20‡	+	+	B-HPS‡
7	50	M	ASHD	L	750	250	280	360‡	495	+ and B-AP	B-HPS‡	+	120/20‡	+	+	B-HPS‡
8	61	M	MVP	L	600	220	300	—	450	B-AP	No B-HPS B-AVN	—	25/20	+	+	B-HPS‡
9	47	F	None	L	600	240	<250	—	—	+	No B-HPS	—	15/10	+	+	B-HPS‡
10	45	F	None	R	600	<260	270	320‡	—	+	B-HPS‡	—	75/20‡	—	—	—
11	27	F	None	R	700	250	<260	350§	—	+	B-HPS§	+	160/30§	+	+	B-HPS
12	41	F	None	R	600	230	<240	—	—	+	No B-HPS	—	20/20	+	+	B-HPS§
13	57	M	None	R	600	270	280	?	—	+	?	?	—	—	—	—
14	54	F	ASHD	R	700	280	<290	—	—	+	No B-HPS	—	60/20	+	+	B-HPS‡

Abbreviations used in this table: ASHD, arteriosclerotic heart disease; AVN, AV node; B, block; MVP, mitral valve prolapse; NP, normal pathway; Ret, retrograde; VCL, ventricular cycle length; Vent, ventricle. L and R indicate left or right VA AP. + indicates the presence or occurrence of an event.

* ΔVH represents a change of H activation from retrograde via bundle branches to anterograde via the AV node.

‡ Electrophysiologic events that coincided with initiation of PSVT.

§ Events coinciding with occurrence of a single echo beat.

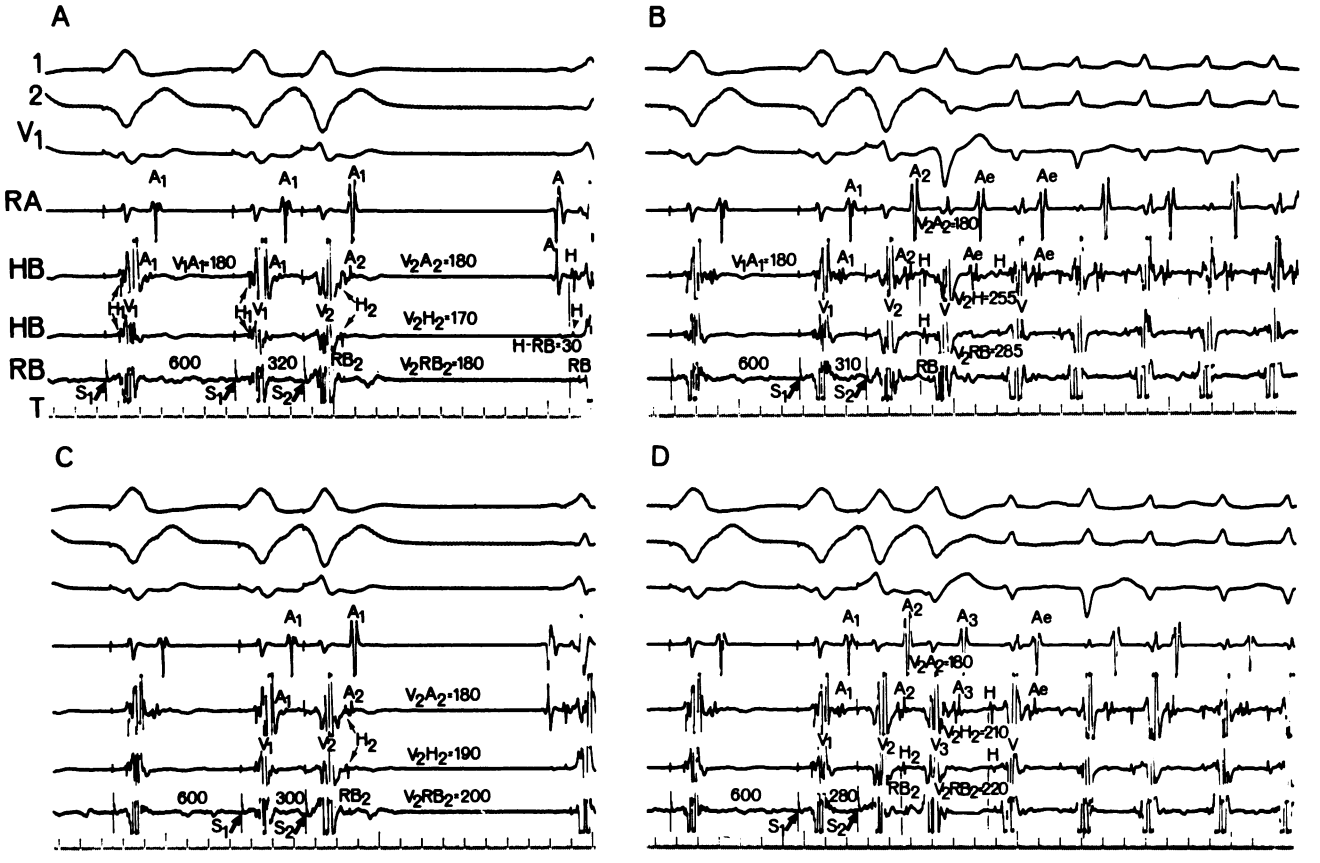

FIGURE 2 (Patient 3) Basic ventricular cycle length is constant at 600 ms (panels A–D). The V_1A_1 and V_2A_2 intervals measure the same in all panels despite progressive shortening in the V_1V_2 intervals. In addition to the two HB tracings, a recording from the RB was also obtained with the H-RB interval measuring a 30 ms during sinus rhythm. The V_2 in panel A depolarizes the H_2 via the left bundle branch (LBB) as suggested by H_2-RB_2 interval of 10 ms. A 10-ms shortening in V_1V_2 interval (panel B) results in an increase in the V-H from 170 ms in panel A to 225 ms in panel B, and PSVT is initiated. Note that the His deflection in panel B follows A_2, and H-RB interval equals that of sinus beats (30 ms) indicating His bundle activation via the AV node. The A_2H interval equals sinus AH (not labeled) and is significantly shorter compared with Ae-H of PSVT, suggesting lack of retrograde penetration of the AV node by V_2 impulse. The QRS following His deflection shows an LBB block (B) pattern, indicating incomplete recovery of LBB following V_2 despite retrograde bilateral block HPS. At shorter V_1V_2 of 300 ms (panel C) V_2H_2 conduction via the LBB resumes (retrograde gap HPS) and PSVT is not induced. Retrograde H_2 activation via the LBB continues in panel D at a shorter coupling interval, and a V_3 due to macroreentry HPS is noted. The V_3 produces A_3 response via the AP blocks in the HPS, and a PSVT is started. 1, 2, and V_1, surface ECG leads; RA, right atrial electrogram; RB, right bundle electrogram tracing; T, time lines.

these 11 cases (cases 1–7 and 11, Table I), the His deflection suddenly shifted from a location preceding or coinciding with the A_2 (on HB electrocardiogram) to a position following the A_2 (Figs. 2–4). This change in location of His bundle deflection was accompanied by an increase in V-H interval ranging from 40 to 155 msec (mean, 121.5±29.1) for a 10–30 ms (mean, 16.2±7.4) decrease in the V_1V_2 interval (Table I). The succeeding A_2H intervals in most cases measured equal to or were slightly longer than (i.e., 10–20 ms) the A-H intervals of sinus beats (except in patient 11) but significantly shorter as compared with the A-H intervals during PSVT. In all of these eight cases ventricular activation followed the His bundle activation, which in turn was followed by retrograde atrial activation via the AP (atrial echo beat or Ae), and PSVT was initiated (Figs. 2–4) in all except one case (case 11). In case 11 the Ae beat anterogradely blocked in the AV node and no sustained PSVT occurred. The sudden increase in the V-H interval was not due to retrograde block in the right bundle branch and subsequent retrograde His activation via the left bundle branch. The events can be best

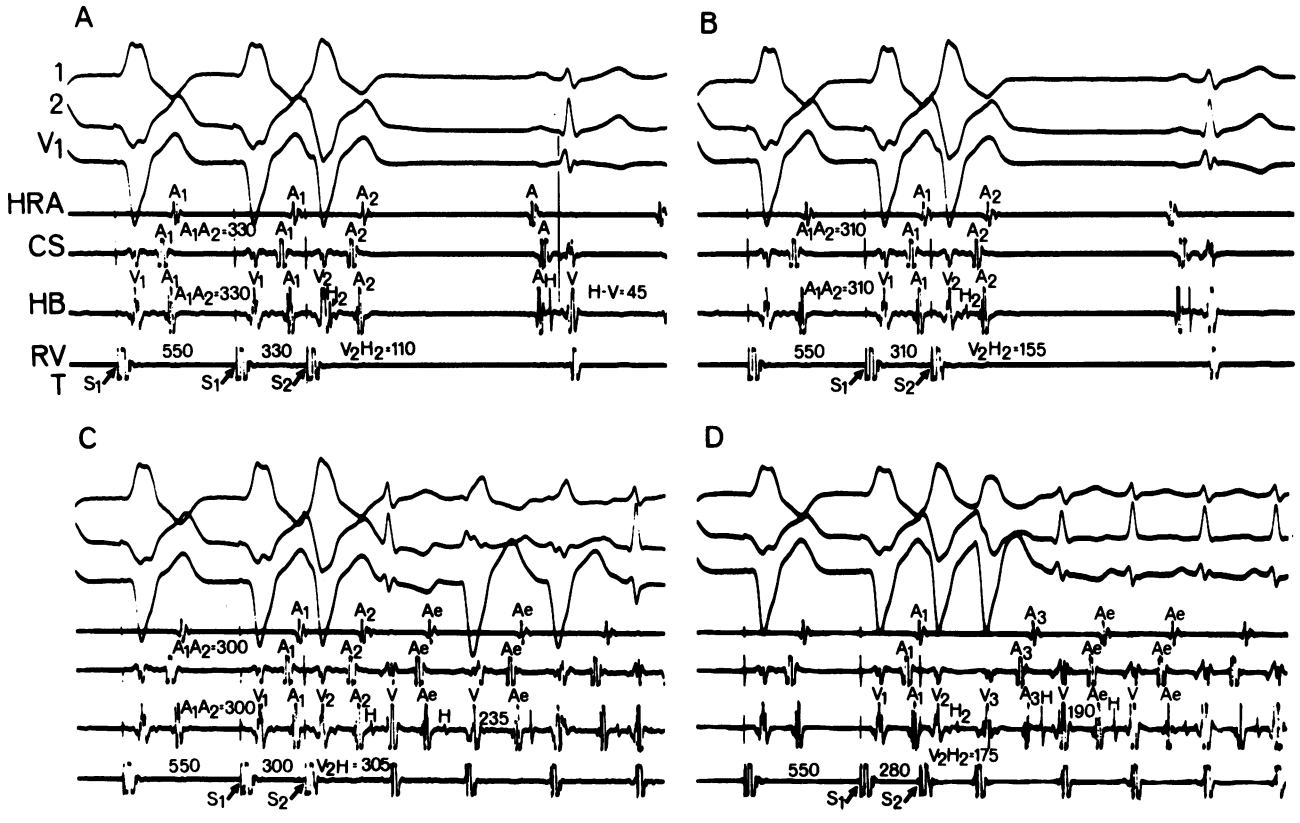

FIGURE 3 (Patient 5, concealed left-sided AP) The basic ventricular cycle length is constant and measures 550 ms in all panels. Exclusive VA conduction via the AP is noted during the basic drive and premature beats in panels A–C. Note the absence of ventricular preexcitation and normal H-V intervals during sinus beats. An increase in V_2H_2 interval is noted from panels A–B, the H_2 preceding A_2 on HB electrogram. In panel C, while the V_2A_2 is unchanged, the His potential now follows the A_2, and PSVT is initiated. A 10-ms decrease in V_1V_2 (from 310 in panel B to 300 in panel C) produces a 150-ms increase in the VH interval from 155 to 305 ms) and the A_2H interval is only slightly longer than AH of the sinus beats (AH intervals not labeled), both of which suggest that the V_2 in panel C retrogradedly blocked in the HPS and the His deflection represents anterograde activation from the A_2 impulse. This is further supported by resumption of V_2H_2 conduction (V_2H_2 – 175 ms, panel D) at shorter V_1V_2 intervals of 280 ms. In panel D no PSVT is initiated by V_2 and the impulse retrogradely blocks in the AP as well as the AV node. However, the H_2 is followed by macroreentry HPS (V_3), which in turn shows a retrograde block HPS while activating atria (A_3) via the AP. Anterograde propagation of A_3 starts the PSVT. Note that the V-Ae measures 235 ms (panel C) during LBB block and 190 (panel D) during normal intraventricular conduction, findings suggestive of left-sided VA AP. RV, right ventricular electrogram.

explained as follows: The V_2 retrogradely blocked bilaterally in the HPS (i.e., no H_2), yet reached the atrium via the AP. Since the AV node was not penetrated by the V_2 impulse, the A_2 response was followed by relatively fast anterograde AV nodal conduction, i.e., A_2H interval. The longer H-V intervals and the aberrant conduction of the ensuing QRS complex noted in some cases was due to retrograde concealed conduction of V_2 in the HPS. The reasons for the above assumption, which was directly documented in two cases by simultaneous His and right bundle (RB) recordings (Fig. 2), will be discussed later. To simplify presentation of the results, the designation V_2H will imply anterograde depolarization of the His potential via the AV node following retrograde A_2 activation over the AP. The occurrence of these electrophysiologic events in individual cases, i.e., retrograde block in the HPS and a shift in His activation from retrograde via the bundle branches to anterograde via the AV node and initiation of PSVT, is depicted in the table. In the remaining 3 of 11 cases (8, 9, and 12), a retrograde block in the HPS was not noted and V_2 continued to activate H_2 via the bundle branches, as indicated by the following: (*a*) persistent linear relationship between V_2H_2 and V_1V_2, (*b*) the His deflection never showed a shift to a position succeeding the A_2 electrogram. None of these three cases demon-

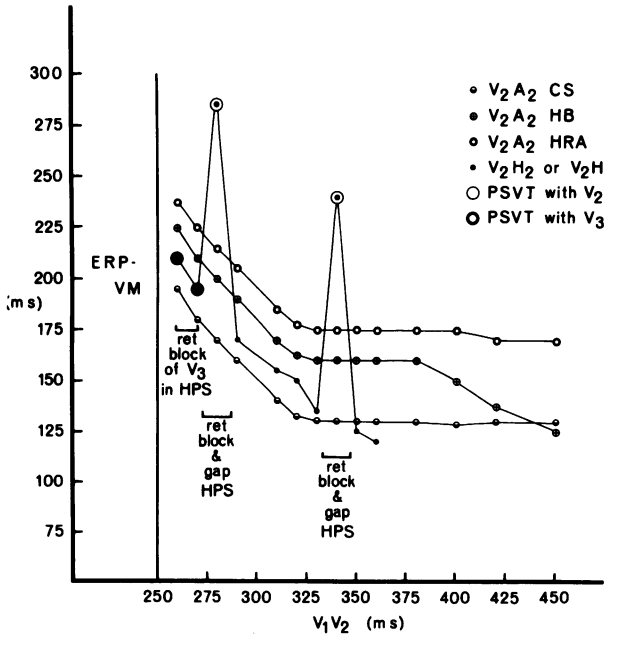

FIGURE 4 (Patient 6) A typical sequence of events during right ventricular premature stimulation is depicted. At a basic ventricular cycle length of 700 ms retrograde A_2 activation initially occurs via both the normal pathway and AP at V_1V_2 of 450 ms, suggesting fusion activation of the atria. Note that with progressive shortening in the V_1V_2, the V_2A_2 on CS electrogram shows no change; V_2A_2 on HRA tracings shows slight increase, whereas V_2A_2 on HB electrogram displays a distinct increase. Exclusive V_2A_2 conduction via the AP is noted at V_1V_2 of 380 ms, and at closer V_1V_2 intervals the V_2A_2 intervals show a parallel increase on all atrial electrogram tracings. Retrograde H_2 activation is first noted at a V_1V_2 of 360 ms, and it precedes A_2 deflections on all atrial electrograms. A sudden increase in VH interval occurs at a coupling interval of 340 ms due to retrograde block of V_2 in the HPS, and a PSVT is initiated. V_2H_2 conduction resumes between V_1V_2 of 330–290 ms (retrograde gap HPS), and PSVT can no longer be started. Upon further shortening in V_1V_2 to 280 ms the V_2 again demonstrates a retrograde block in the HPS with resulting PSVT. Resumption of V_2H_2 conduction at V_1V_2 of 270–260 ms is associated with occurrence of V_3 due to macroreentry HPS, which in turn starts the PSVT. The V_3A_3 conduction occurs via AP and is not depicted. Note that with V_1V_2 up to coupling interval of 330 ms, the V_2H_2 delays were accompanied by constant V_2A_2 conduction via the AP; the latter showed delays only at $V_1V_2 < 320$ ms. Also, when V_2 activated the H_2 via the bundle branches—i.e., when H_2 preceded A_2 on HBE—no PSVT could be initiated. ERP-VM, effective refractory period-ventricular myocardium.

strated initiation of PSVT or an Ae response following V_2.

In two patients (10 and 14) with right-sided VA AP, the onset of low atrial A_2 was inscribed within the local V_2 electrogram, and the His deflection followed the A_2 the very first time it emerged from V_2. Therefore, it was difficult to determine if the His deflection represented anterograde or retrograde activation. In case 10, however, a sudden increase in the V-H of 75 ms with a 20 ms decrease in V_1V_2 coincided with initiation of PSVT, suggesting that the initial His activation was retrograde, whereas the onset of PSVT heralded a retrograde block in the HPS, findings similar to all other cases where V_2 initiated PSVT.

In the one remaining patient (13), at no time did the His deflection precede A_2, and a change from retrograde to anterograde depolarization of the His bundle could not be documented. Although a PSVT was initiated in this case by V_2, discernment of the exact mechanism was not certain.

Retrograde gap phenomenon HPS (16). Following the initial retrograde block of V_2 in the HPS, further shortening in V_1V_2 intervals resulted in resumption of retrograde conduction to the His bundle (i.e., V_2H_2) in seven of nine cases (1, 3–7, and 11, Table I retrograde gap phenomenon HPS), as was suggested by return of H_2 deflection preceding or coinciding with the A_2 electrogram (Figs. 3 and 4). The V_2H_2 values following resumption of conduction were in line with those expected from a reciprocal relationship between V_1V_2 and V_2H_2 intervals and significantly shorter than the V_2H values noted at longer coupling intervals (Fig. 4). Return of V_2H_2 conduction occurred while retrograde A_2 activation persisted via the AP in all cases except one (point No. 5, Fig. 3), where a concomitant retrograde block was noted along the AP. Whenever retrograde H_2 followed V_2 with or without associated A_2 activation via the AP, the Ae or PSVT was not initiated by V_2 either before or after the gap zone in any of the cases in this series.

Macroreentry HPS (12, 13). 10 of the 14 cases (3–9, 11, 13, and 14, Table I) demonstrated occurrence of macroreentry HPS in the form of V_3 following retrograde H_2 activation. The V_3 in turn showed a retrograde block in the HPS along the normal pathway (i.e., no H_3) in all patients (Fig. 3). The V_3, however, conducted to the atrium (A_3) via the AP in all cases and an Ae or PSVT was started in 9 of 10 (Table I, Fig. 3). In the remaining one case the A_3 blocked in the AV node, and no Ae or PSVT was noted.

Findings at other basic cycle lengths. In 10 of 14 cases, two or more basic cycle lengths were tested, and the results were similar to those presented above, i.e., the PSVT was only initiated when V_2 and/or V_3 retrogradely blocked in the HPS while still activating atrial via the AP.

DISCUSSION

Results of this study indicate that retrograde HPS block of V_2 and/or V_3 in the HPS is a relatively common mechanism by which PSVT is initiated during right ventricular premature stimulation in patients with the WPW Syndrome (13 of 14 cases in this series). Although

the induced V_2 may initiate PSVT by other mechanisms, present data suggest that other mechanisms are less commonly responsible for PSVT initiation during ventricular premature stimulation. In a series with 139 cases of WPW Syndrome reported by Wellens (6) it was noted that, during ventricular premature stimulation, a PSVT was most commonly initiated when V_2 retrogradely blocked along the normal pathway (30 of 139 cases) or by V_3 (9 of 139 cases) when the latter was inducible, and by other mechanisms in only 11 of 139 cases. The exact site of retrograde block (HPS vs. AV node) of V_2 or V_3 was not demonstrated, and it cannot be stated with certainty whether the findings in the report by Wellens and the present study are comparable. A recent study showed that in patients with ventricular preexcitation, programmed ventricular extrastimuli did not initiate PSVT if there was no VA conduction and there was persistent retrograde atrial fusion activation via both the AP and normal pathway (7). A narrow QRS PSVT could be elicited with V_2 when VA conduction was intact, V_2A_2 conduction occurred exclusively via the AP, and normal pathway was less deeply penetrated (7). In that report it was indicated, however, that the level or site of retrograde block along the normal pathway could not be determined.

Regular narrow QRS complex PSVT is the most common arrhythmia elicited by programmed atrial as well as ventricular premature stimulation in patients with the WPW Syndrome (3, 4). During premature atrial stimulation (A_2), the premature impulse blocks in the AP, activates the ventricles over the AV node-HPS, and returns to the atria via the AP to start the PSVT. Induction of the same PSVT with V_2, on the other hand, will be more likely if V_2A_2 conduction occurred via the AP and subsequent anterograde propagation took place along the AV node-HPS. The latter occurrence will obviously be facilitated by a retrograde block of V_2 in the normal pathway. A retrograde block of V_2 above the His bundle (AV node), however, may not permit initiation of PSVT even if accompanied by concomitant retrograde A_2 activation via the AP, because of effect of retrograde concealed conduction in the AV node. Under the following circumstances, however, V_2 may start PSVT despite AV nodal penetration (*a*) when V_2 produces only a partial penetration of the AV node with resultant rapid recovery of excitability, (*b*) initiates intranodal reciprocation, (*c*) conducts with long V_2A_2 via AP sufficient to allow recovery of AV nodal excitability. A retrograde block of V_2 in the HPS, on the other hand, would permit unhindered progress of A_2 impulse through the AV node, since the prior AV nodal depolarization is during the last V_1.

A support for the preceding hypothesis is provided in the present study by demonstration that the PSVT initiation did coincide with the occurrence of retrograde block of V_2 in the HPS. That the site of retrograde block of V_2 was indeed in the HPS rather than the AV node is suggested by the following observations: (A) A sudden increase in the V-H values (a mean increase of 121.8 ms from V_2H_2 to V_2H, for a 16.2 ms mean decrease) in V_1V_2 intervals, a behavior unlike that of retrograde H_2 activation via the bundle branches and therefore suggesting a shift from retrograde to anterograde activation of the His bundle (Figs. 2–4). (B) A return of H_2 deflection at shorter V_1V_2, producing V_2H_2 values less than V_2H values (retrograde gap phenomenon HPS) noted at longer V_1V_2, and concomitant abolition of PSVT (Figs. 3 and 4). (C) Direct demonstration of the shift from a retrograde to anterograde activation of the His bundle when both His and RB recordings were available (two cases, Fig. 2). (D) The occurrence of macroreentry HPS after retrograde H_2 activation and its disappearance despite longer V-H intervals, coinciding with the shift of His deflection to a location succeeding the A_2 (Fig. 3). (E) A consistent relationship between the A_2 and the succeeding His deflection with values approximating the sinus A-H values. This suggests that the occurrence of His deflection after A_2 was not an independent phenomenon (retrograde via bundle branches) but its occurrence was directly related to and in fact dependent upon the preceding A_2 activation (Figs. 2 and 3).

It seems reasonable to conclude, therefore, that the His deflection following the A_2 was due to anterograde depolarization via the AV node. Despite the above reasoning, questions can be raised as to whether the V_2 might still have blocked in the AV node rather than HPS, and whether the H_2 deflection was either not identifiable for some reason or was obscured by the local V_2 electrogram. These questions can be dismissed for the following reasons: (*a*) Once the retrograde H_2 deflection emerges, the V_2H_2 intervals generally lengthen (rather than shorten) at shorter V_1V_2 (9, 12, 14, 15). (*b*) The occurrence of gap phenomenon in most cases suggests retrograde H_2 was recordable whenever the His bundle was depolarized. (*c*) At the V_2H interval ranges noted in these cases, the His bundle activation will have to occur twice (first retrogradely and then anterogradely) within a period of 300 ms, an unlikely occurrence. In the event the V_2 retrogradely penetrates the AV node, the subsequent A_2H intervals will be expected to be much longer than the sinus beats (retrograde concealed conduction in the AV node). Such a mechanism of PSVT initiation was not noted in the present series but has been previously described, and may operate more frequently at shorter cycle lengths due to abbreviation in the refractoriness of the His. For similar reasons a retrograde AV nodal block of ventricular impulse may also

be a more common mechanism of PSVT initiation during rapid incremental ventricular pacing rather than premature stimulation (8).

It was repeatedly observed during this study that whenever V_2 produced a retrograde H_2 response, the PSVT could not be initiated. Since concomitant retrograde A_2 activation in most instances took place exclusively via the AP, the collision of the two impulses via the normal pathway and AP must have occurred in the AV node rather than the atrium (7, 17). Retrograde penetration and concealed conduction in the AV node was most probably responsible for preventing the forward progress of the A_2 impulse. It is easily understandable, therefore, why the onset of retrograde block in the HPS coincided with initiation of a PSVT or an Ae response. It may also be pointed out that the response of HPS and AP to V_2 was not significantly different between right- and left-sided AP, aside from the fact that the location of A_2 electrogram in patients with right VA AP made analysis of events more difficult.

When an H_2 response followed V_2, the PSVT only occurred if V_3 followed. Although in this group of patients the V_3 phenomenon occurred dur to macroentry HPS, a V_3 from other mechanisms—local reentry, etc.—will be expected to produce the same results (13). The initiation of an Ae response or PSVT following V_3 was also facilitated by a retrograde block of V_3 in the HPS and concomitant retrograde A_3 activation via the AP, as was noted in 9 of 14 cases in this series.

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