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Demonstration of Diastolic and Presystolic Purkinje Potentials as Critical Potentials in a Macroreentry Circuit of Verapamil-Sensitive Idiopathic Left Ventricular Tachycardia

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OBJECTIVES The purpose of this study was to determine the relation of diastolic and presystolic potentials recorded during verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) to reentry circuit.

BACKGROUND Successful ablation of verapamil-sensitive ILVT at the zone of slow conduction from which the diastolic potential is recorded has been reported. However, the relationship between the diastolic potential and the reentrant circuit remains a matter of debate.

METHODS Radiofrequency (RF) ablation was performed in 20 patients with verapamil-sensitive ILVT. After identifying the ventricular tachycardia (VT) exit site, we searched for the mid-diastolic potential (P1) during VT. Entrainment followed by RF current application was performed. If the mid-diastolic potential could not be detected, RF current was applied at the VT exit site showing the earliest ventricular activation with a single fused presystolic Purkinje potential (P2).

RESULTS In 15 of 20 patients, both P1 and P2 were recorded during VT from midseptal region. Entrainment pacing captured P1 orthodromically and reset the VT. The interval from stimulus to P1 was prolonged as the pacing rate was increased. Radiofrequency ablation was successfully performed at this site in all 15 patients. After successful ablation, P1 appeared after the QRS complex during sinus rhythm with the identical sequence to that during VT. In the remaining five patients, the diastolic potential could not be detected, and a single fused P2 was recorded only at the VT exit site. Successful ablation was performed at this site in all five patients.

CONCLUSIONS This study demonstrates that P1 and P2 are critical potentials in a circuit of verapamil-sensitive ILVT and suggests the presence of a macroreentry circuit involving the normal Purkinje system and the abnormal Purkinje tissue with decremental property and verapamil-sensitivity. (J Am Coll Cardiol 2000;36:811–23) © 2000 by the American College of Cardiology

The most common form of idiopathic left ventricular tachycardia (ILVT) is verapamil-sensitive intrafascicular tachycardia (1–9). Radiofrequency (RF) catheter ablation can be performed successfully, with determination of the ablation site based on the identification of a presystolic Purkinje potential (P2), early endocardial activation or pace mapping (10–12). Recently, successful ablation of verapamil-sensitive ILVT at the zone of slow conduction from which the mid-diastolic potential is recorded has been reported (13–18). However, the relationship between diastolic potential (P1) and the reentrant circuit is still a matter of debate. This study describes the responses of P1 and P2 to entrainment pacing and RF ablation in 20 patients with verapamil-sensitive ILVT. These findings provide new insights toward an understanding this arrhythmia.

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METHODS

Patients. Between February 1994 and January 1999, a total of 20 consecutive patients with verapamil-sensitive left VT exhibiting a right bundle branch block (RBBB) configuration and left-axis deviation underwent RF ablation therapy at our hospitals. These patients were 17 men and three women, age range 12 to 66 years (mean 32 ± 13 years). Ventricular tachycardia (VT) was terminated by the intravenous administration of verapamil in all patients.

Electrophysiologic study. An electrophysiologic study was performed after all anti-arrhythmic medications had been discontinued and after obtaining written informed consent from each of the 20 patients. A 7 F quadripolar steerable electrode catheter with a 4-mm tip and a 2-mm interelectrode spacing between the distal two electrodes (Cordis Webster, Inc., Baldwin Park, California) was positioned at

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Abbreviations and Acronyms

CL = cycle length
ECG = electrocardiogram
H = His bundle
ILVT = idiopathic left ventricular tachycardia
LV = left ventricle
PPI = postpacing interval
P1 = diastolic potential
P2 = presystolic Purkinje potential
RBBB = right bundle branch block
RF = radiofrequency
S = stimulus
VT = ventricular tachycardia

the interventricular septum of the left ventricle (LV) to record the intracardiac electrograms, pacing and ablation. In several patients, a 7 F octapolar steerable electrode catheter with 1.25-mm electrode lengths and 2-mm interelectrode spacings (EP Technologies Inc., Sunnyvale, California) was positioned at the interventricular septum of the LV before mapping using the quadripolar steerable catheter (Fig. 1). Bipolar intracardiac electrograms were recorded using a filter bandwidth of 30 to 400 Hz. Electrocardiogram (ECG) leads I, II, aVF and V1 as well as intracardiac electrograms from various sites were displayed simultaneously and recorded on a multichannel oscilloscopic recorder (EPLab, Quinton, Seattle, Washington).

Programmed ventricular stimulation was performed using a maximum of three extrastimuli at two different driven cycle lengths (CLs) from the right ventricular apex and outflow tract. If sustained VT was not induced, the stimulation was repeated during isoproterenol infusion (0.5 to 2 μg/min). During VT, endocardial mapping in the LV was performed, and the putative VT exit site was determined as the earliest ventricular activation site where a single fused Purkinje potential was recorded before the onset of the QRS complex. After identifying the putative VT exit site, P1 was searched for during VT.

Entrainment. In all patients, entrainment of VT by pacing from the right ventricular apex or outflow tract was performed while recording the electrogram at the earliest ventricular activation site. In nine patients, entrainment could be performed while recording the P1.

Catheter ablation. The output current of the RF generator (CAT-500, Central Industry, Tokyo, Japan) was delivered to the distal electrode of an ablation catheter and a cutaneous patch. The test RF current, with an initial power of 20 to 25 W, was applied to the site showing the diastolic potential during VT. However, ablation was not initially directed to the earliest diastolic potential, because the performance of ablation at a site that is too proximal carries the risk of causing atrioventricular block or left bundle branch block. If the VT was terminated or slowed within 15s, additional current was applied for another 60 to 120 s. If the test RF current was ineffective, ablation was directed to a more proximal site with the earlier diastolic potential. If the P1 could not be detected, RF current was applied at the VT exit site showing a single fused P2. Following ablation, programmed stimulation was repeated.

Postablation management. Patients were monitored for four to seven days after the ablation. The patients were followed for periods of 38 ± 19 months (median 37 months) without anti-arrhythmic medications. A 24-h Holter recording was obtained at approximately yearly intervals.

Statistical analysis. Values are given as mean ± SD. The significance of difference between groups was assessed by the Student *t* test. A level of $p < 0.05$ was accepted as statistically significant.

RESULTS

Endocardial mapping. Sustained monomorphic VT with the same QRS configuration as the patient's spontaneous VT was induced by ventricular stimulation in all 20 patients.

Left ventricular endocardial mapping was performed during sustained VT. In 15 of the 20 patients (patients 1 to 15), two distinct potentials, P1 and P2, were recorded during VT at the mid-septum (Fig. 1 and 2) (double potential group). While the P1 was recorded earlier from the proximal than the distal electrodes, the fused P2 was recorded earlier from the distal than the proximal electrodes. During sinus rhythm, recording at the same site demonstrated the P2, which was recorded after the His-bundle potential and before the onset of QRS complex; however, the sequence of P2 was reverse to that seen during VT. The distance between electrodes 1 and 8 of the octapolar electrode catheter was approximately 25 mm. In the remaining five patients (patients 16 to 20), the P1 could not be detected, and a single fused P2 was recorded only at the mid- or inferior apical septum (single potential group). There were no differences between two groups in age, gender or VT CL (Table 1).

Entrainment. The entrainment phenomena, including constant fusion and progressive fusion, were observed during right ventricular pacing in all 20 patients. Figure 3

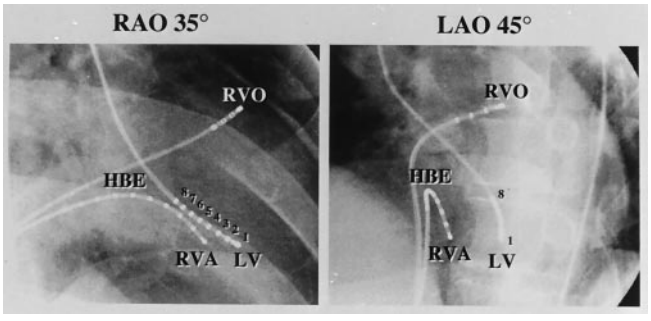

Figure 1. Patient 3. Representation of an octapolar electrode catheter positioned at the left ventricular septum as viewed fluoroscopically in the right oblique (RAO) and left oblique (LAO) projections. The distance between electrodes 1 and 8 of the octapolar electrode catheter was approximately 25 mm. HBE = His-bundle electrogram; LV = left ventricle; RVA = right ventricular apex; RVO = right ventricular outflow tract.

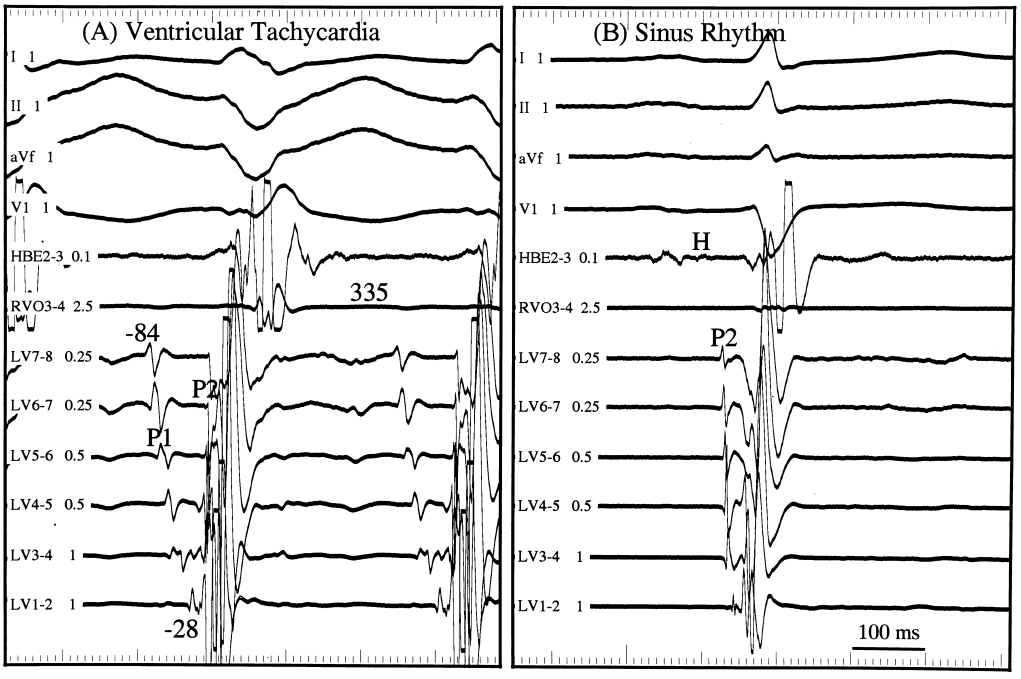

Figure 2. Patient 3. Intracardiac recordings from octapolar electrode catheter. **(A)** During VT a diastolic potential (P1) and a presystolic Purkinje potential (P2) were recorded. While P1 was recorded earlier from the proximal than the distal electrodes, P2 was recorded earlier from the distal than the proximal electrodes. **(B)** During sinus rhythm recording at the same site demonstrated the P2, which was recorded before the onset of QRS complex.

shows concealed entrainment by pacing from VT exit site in Patient 3. During VT, the earliest ventricular electrogram with the fused P2 was recorded from the distal two electrodes. Pacing from the distal two electrodes at a CL and a starting coupling interval of 400 ms captured P1 orthodromically and produced QRS configurations similar to that of the VT (Fig. 3A). The postpacing interval (PPI) (stimulus [S]-P2) was equal to the VT CL. Pacing from VT exit site at a CL of 380 ms also captured P1 (Fig. 3B). The P1 was simultaneously observed with pacing artifact from LV7-8, and the S-P1 interval was prolonged. Pacing from VT exit at a CL of 400 ms, but with a starting coupling interval of 300 ms, terminated VT (Fig. 3C). The P1 was not observed during pacing, because it might be captured antidromically and masked in the left LV electrogram. The same findings were observed in seven of nine patients in whom entrainment pacing could be performed during recording of P1 and P2 (Table 1). In the remaining two patients, S-P1 interval during entrainment pacing remained unchanged when the pacing rate was increased.

Resetting by ventricular extrastimuli and spontaneous sinus capture. Single or double ventricular extrastimuli were performed in two patients (Patients 4 and 12). Figure 4 shows an example of resetting and termination with double ventricular extrastimuli from the right ventricle in Patient 4. In Figure 4A, the first impulse did not reset the tachycardia, but the second impulse captured a P1 orthodromically and produced the resetting manifested by a less than compensatory pause. When the S1-S2 coupling interval was shortened, the conduction time from the S to the orthodromically activated P1 was significantly prolonged (Fig. 4B). This resulted in the increasing return cycle. When the second impulse was delivered even more prematurely, it encountered refractoriness in the orthodromic direction, and the tachycardia terminated (Fig. 4C). The same findings were observed in the other patient (Patient 12).

Figure 5 shows the resetting of VT by a spontaneous sinus capture in Patient 3. After the second complex of VT, a sinus capture occurred, resulting in narrowing of the QRS width without interruption of the tachycardia. Presystolic Purkinje potential and ventricular electrogram were advanced by sinus beat, and the earliest P2 was recorded from LV3-4. The subsequent P1 and VT had been reset because pauses were less than fully compensatory. The same findings were observed in 3 other patients (Patients 13, 14 and 15). In the remaining 11 patients atrioventricular nodal conduction was not good enough to capture the P2 by sinus activity during VT.

Effect of verapamil on VT circuit. The effect of verapamil on P1 and P2 was shown in Figure 6 (Patient 4). Intravenous administration of 1.5-mg verapamil significantly prolonged VT CL from 305 ms to 350 ms. Both P1-P2 and P2-P1 intervals were proportionally prolonged after verapamil. However, the interval from P2 to the onset of the QRS complex remained unchanged.

Catheter ablation. In the double potential group, the RF current was applied to the site where the P1 was recorded during VT. However, ablation was not initially directed to the earliest diastolic potential, because the performance of ablation at a site that is too proximal carries the risk of

Table 1. Characteristics of the Two Successful Target Sites

Patient	Age (yrs)	Gender	VT CL (ms)	QRS Width during VT (ms)	QRS-H Interval (ms)	P1-QRS Interval (ms)	(P1-QRS/VTCL) × 100 (%)	P2-QRS Interval (ms)	(P2-QRS/VTCL) × 100 (%)	S-P1 Interval Prolonged During Entrain	Post Pacing Interval (ms)	VTCL PPI Difference (ms)	QRS Map Score During VT	Number of RF Energy
Double Potential Group (n = 15)														
1	12	M	330	120	N/A	60	18	5	2	N/A	N/A	N/A	10	1
2	49	M	405	125	0	70	17	35	9	N/A	N/A	N/A	11	1
3	38	M	415	120	N/A	45	11	10	2	yes	415	0	8	1
4	34	M	300	130	20	45	15	15	5	yes	320	20	10	1
5	20	M	340	125	30	40	12	20	6	N/A	340	0	10	2
6	30	M	375	120	N/A	30	8	5	1	N/A	460	20	9	1
7	22	M	280	125	N/A	110	39	0	0	yes	260	10	12	2
8	31	M	280	110	5	53	19	13	5	N/A	N/A	N/A	12	6
9	39	M	375	125	20	53	14	25	7	N/A	400	10	9	1
10	18	M	245	90	N/A	28	11	0	0	yes	250	10	5	1
11	30	M	365	155	N/A	45	12	15	4	yes	335	30	10	1
12	32	M	460	140	59	100	22	20	4	no	440	20	9	1
13	41	F	385	130	15	123	32	0	0	no	400	15	11	2
14	28	M	380	120	30	50	13	30	8	yes	425	0*	12	1
15	19	F	248	120	40	48	19	0	0	yes	248	0	6	1
	30		346	124	24	60	18	13	3			11	9.6	1.5
	± 10		± 64	± 14	± 18	± 29	± 8	± 11	± 3			± 10	± 2.1	± 1.3
Single Potential Group (n = 5)														
16	45	M	280	125	27	—	—	24	9	—	310	30	11	1
17	36	M	330	130	N/A	—	—	14	4	—	320	10	8	4
18	18	M	340	120	28	—	—	24	7	—	N/A	N/A	N/A	9
19	32	F	300	116	N/A	—	—	18	6	—	290	10	11	1
20	66	M	366	132	26	—	—	10	3	—	350	0*	11	1
	39		355	125	27			18	6			13	10.3	2.0
	± 18		± 59	± 7	± 1			± 6	± 3			± 13	± 1.5	± 2.1

*VT CL was different from 4th column at the time of entrainment pacing.

N/A = not available; P1 = diastolic potential; P2 = presystolic Purkinje potential; PPI = post pacing interval; RF = radiofrequency; S = stimulus; VT = ventricular tachycardia; VTCL = cycle length of ventricular tachycardia.

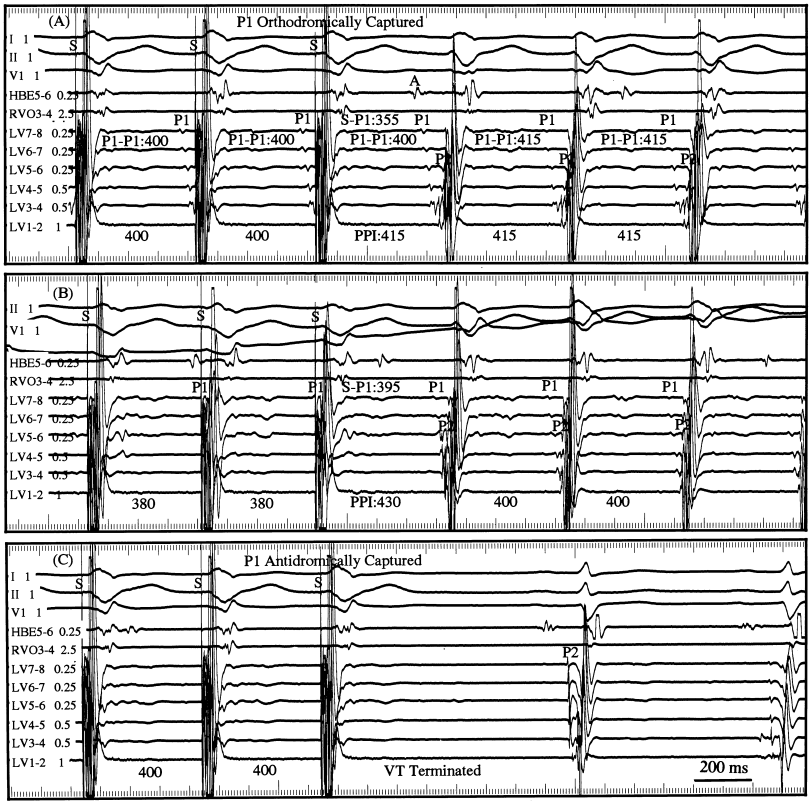

Figure 3. Patient 3. Concealed entrainment by pacing from VT exit site. During VT the earliest ventricular electrogram with the fused P2 was recorded from the distal two electrodes. **(A)** Pacing from the distal two electrodes at a cycle length and a starting coupling interval of 400 ms captured P1 orthodromically and produced QRS configurations similar to that of the VT. The postpacing interval (PPI) (S-P2) was equal to the VT cycle length. **(B)** Pacing from VT exit site at a cycle length of 380 ms also captured P1. Diastolic potential was simultaneously observed with pacing artifact from LV7-8. Pacing stimulus P1 interval was prolonged. **(C)** Pacing from VT exit at a cycle length of 400 ms but with a starting coupling interval of 300 ms terminated VT. Diastolic potential was not observed during pacing because it might be captured antidromically and masked in the ventricular electrogram. PPI = post pacing interval; P1 = diastolic potential; P2 = presystolic Purkinje potential; S = pacing stimulus; VT = ventricular tachycardia.

causing atrioventricular block or left bundle branch block. Figure 7 shows a site of successful ablation during VT in patient 1. Two distinct diastolic and presystolic potentials (P1 and P2) were recorded in the midseptal area. The proximal pair of electrodes of the ablation catheter recorded the P1 earlier than the distal pair of electrodes. Successful ablation was performed at the site with P1 and P2 in all 15 patients in the double potential group. In 11 of 15 patients the first energy delivery at this site was successful. The interval between P1 at the site of successful ablation and the onset of the QRS complex (P1-QRS interval) during VT was 60 ± 29 ms (18 ± 8% of VT CL). Pacing from this site demonstrated similar QRS complexes during VT and pacing in only 9.6 ± 2.1 of the 12 ECG leads (Table 1). Selective pacing of P1 was unavailable because the pacing stimuli resulted in capture of not only the P1, but also the adjacent myocardium. The distance between the successful ablation site and the VT exit site (the earliest ventricular activation site with fused P2) in this group was 7.5 ± 1.5 mm. After ablation, no patient exhibited left bundle branch block or atrioventricular block.

In the single potential group, RF current was applied to the site of the earliest ventricular activation with a fused P2. Ablation was successfully performed at this site in all five patients in this group. In three of five patients the first energy delivery at this site was successful. The interval between P2 at the site of successful ablation and the onset of the QRS complex (P2-QRS interval) during VT was 18 ± 6 ms (6 ± 3% of VT CL). Pacing from this site demonstrated similar QRS complexes during VT and pacing in 10.3 ± 1.5 of the 12 ECG leads. The mean difference between postpacing interval (PPI) and VT CL was 11 ± 10 ms in the double potential group and 13 ± 13 ms in the single potential group.

Diastolic and presystolic potentials before and after successful ablation. Figure 8 shows the successful application of RF current delivered during VT in Patient 7. During energy application, the P1-P2 interval was gradually prolonged, and the VT was terminated by block between P1 and P2. After termination of the tachycardia, the P1 was noted after the QRS complex during sinus rhythm, while the P2 was still observed before the QRS complex. Figure 9 shows the potentials during sinus rhythm before and after the successful ablation in patient 1. After successful ablation, the P1 occurred after the QRS complex with the identical activation sequence to that observed during VT

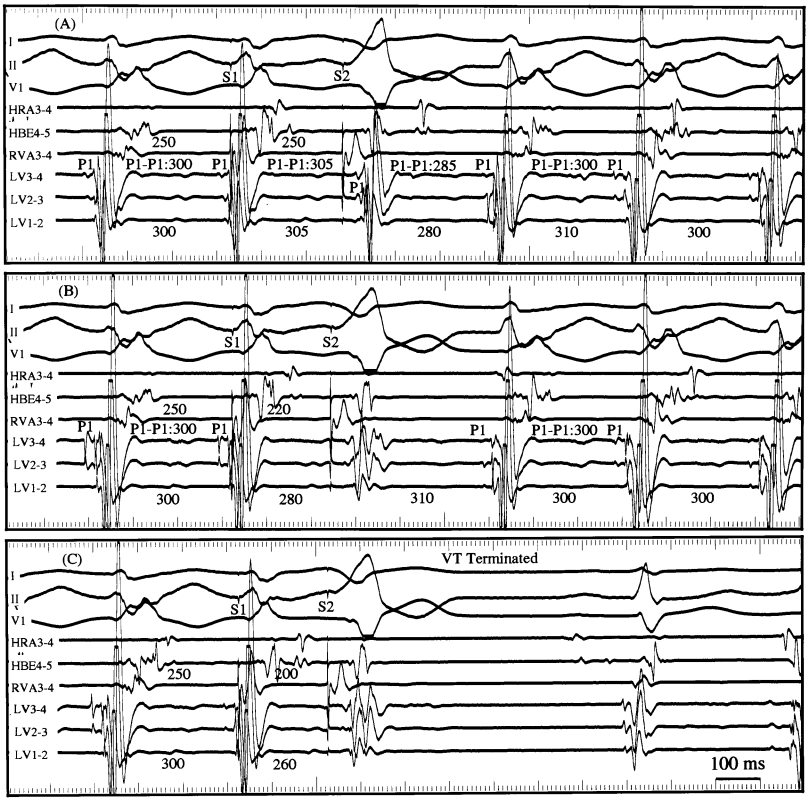

Figure 4. Patient 4. Resetting and termination of VT with double ventricular extrastimuli from right ventricle. **(A)** The first impulse did not reset the tachycardia, but the second impulse captured a P1 orthodromically and produced the resetting manifested by a less than compensatory pause. **(B)** When the S1-S2 coupling interval was shortened, the conduction time from the stimulus to the orthodromically activated P1 was significantly prolonged. This resulted in the increasing return cycle. **(C)** When the second impulse was delivered even more prematurely, it encountered refractoriness in the orthodromic direction, and the tachycardia terminated. P1 = diastolic potential; VT = ventricular tachycardia.

shown in Figure 7. In all 15 patients in the double potential group, the P1 was observed after the QRS complex after successful ablation. During sinus rhythm after ablation, the interval between the His-bundle electrogram and P1 (H-P1 interval) was 347 ± 113 ms, and the P2 to QRS interval was 17 ± 7 ms (Table 2). In the single potential group, the P2 was recorded before the QRS complex after successful ablation, and the P2-QRS interval was 9 ± 6 ms during sinus rhythm.

Characteristics of diastolic potential after ablation. The P1 that appeared after ablation showed a decremental property during atrial pacing and/or ventricular pacing (Fig. 10) in 10 of 15 patients in the double potential group (Table 2). The intravenous administration of 10 mg of verapamil significantly prolonged the H-P1 interval during sinus rhythm from 341 ± 100 ms to 414 ± 119 ms in all six patients tested ($p < 0.01$). In four patients (Patients 3, 6, 11 and 14), the split P1 potentials (P1 and P1′) were observed after the QRS complex, and the P1-P1′ interval was prolonged during atrial or ventricular pacing or after intravenous administration of verapamil (Fig. 11). The intravenous administration of adenosine triphosphate disodium did not change the interval between ventricular stimulus and P1 during ventricular pacing in the five patients tested.

Follow-up procedures. Patients were followed for 10 to 71 months (median 37 months) without antiarrhythmic medications. No patient had recurrence of VT or premature ventricular complex with an RBBB configuration and left-axis deviation.

DISCUSSION

Main findings. We found two types of successful ablation site in verapamil-sensitive ILVT that exhibited an RBBB configuration and left-axis deviation. In 15 of the 20 patients, RF catheter ablation was successful at the site with a P1 that is distant from the VT exit. In the remaining five patients, the P1 could not be detected during VT, but the application of RF current to the VT exit site with a single fused P2 was successful.

Electrophysiological results in the double potential group are summarized as follows:

1) P1 and P2 were recorded from a relatively large area at the middle septal region.
2) Entrainment pacing from the ventricle captured P1 orthodromically and reset the tachycardia. The S-P1 interval was prolonged as the pacing rate was increased.

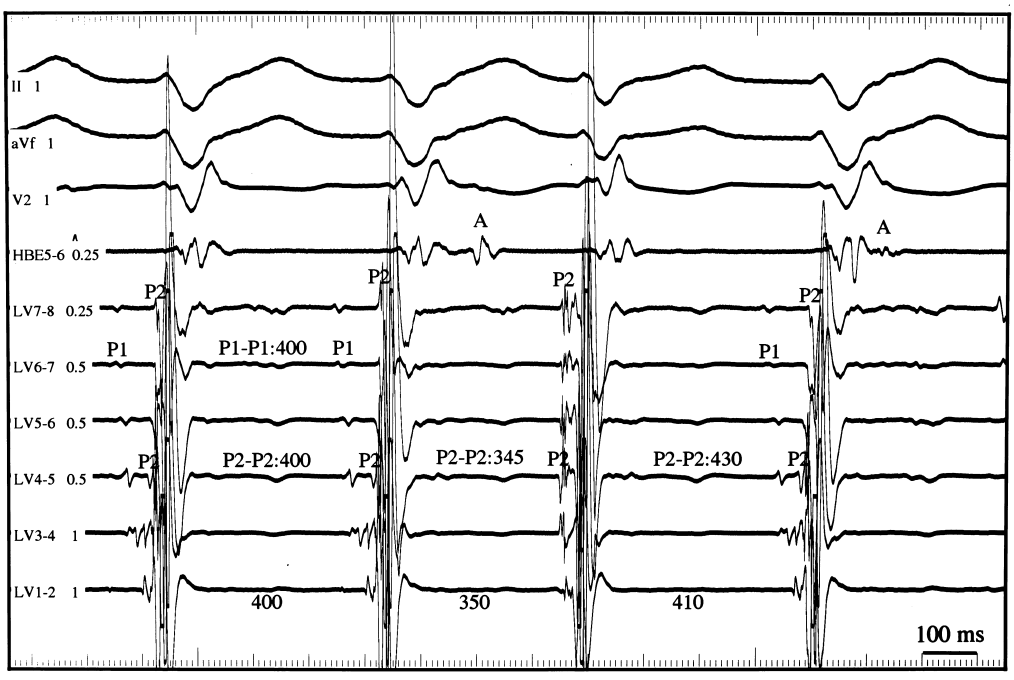

Figure 5. Patient 3. Resetting of VT by a spontaneous sinus capture. After the second complex of VT, a sinus capture occurred resulting in narrowing of the QRS width without interruption of the tachycardia. Presystolic Purkinje potential (P2) and ventricular electrogram was advanced by sinus beat, and the earliest P2 was recorded from LV3-4. The subsequent P1 and VT had been reset because pauses were less than fully compensatory. A = atrial electrogram; HBE = His bundle electrogram; LV = left ventricle; P1 = diastolic potential; P2 = presystolic Purkinje potential.

When the impulse encountered the refractoriness of P1 in the orthodromic direction, the VT was terminated.

3) A spontaneous sinus capture beat advanced the P2 and reset the VT.
4) A small dose of verapamil significantly prolonged VT CL, P1-P2 interval and P2-P1 interval during VT. However, the interval from P2 to the onset of the QRS complex remained unchanged.

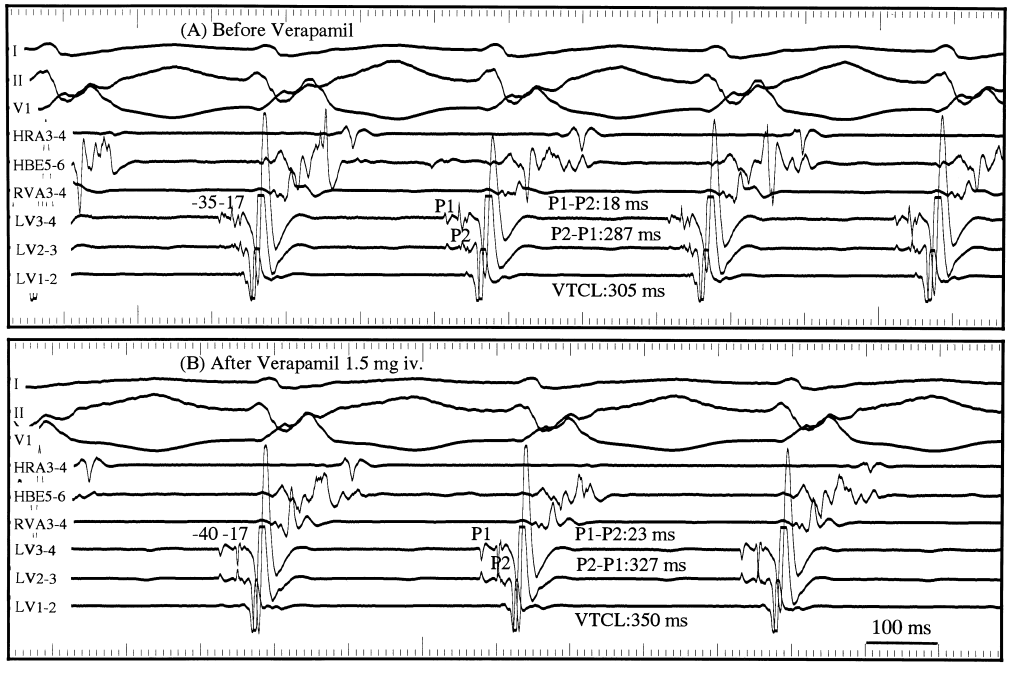

Figure 6. Patient 4. Effect of verapamil on VT circuit. Intravenous administration of 1.5 mg verapamil significantly prolonged cycle length of VT from 305 ms **(A)** to 350 ms **(B)**. Both P1-P2 and P2-P1 intervals were proportionally prolonged after verapamil. However, the interval from P2 to the onset of the QRS complex remained unchanged. HRA = high right atrium; P1 = diastolic potential; P2 = presystolic Purkinje potential; RVA = right ventricular apex; VT = ventricular tachycardia; VT CL = cycle length of ventricular tachycardia.

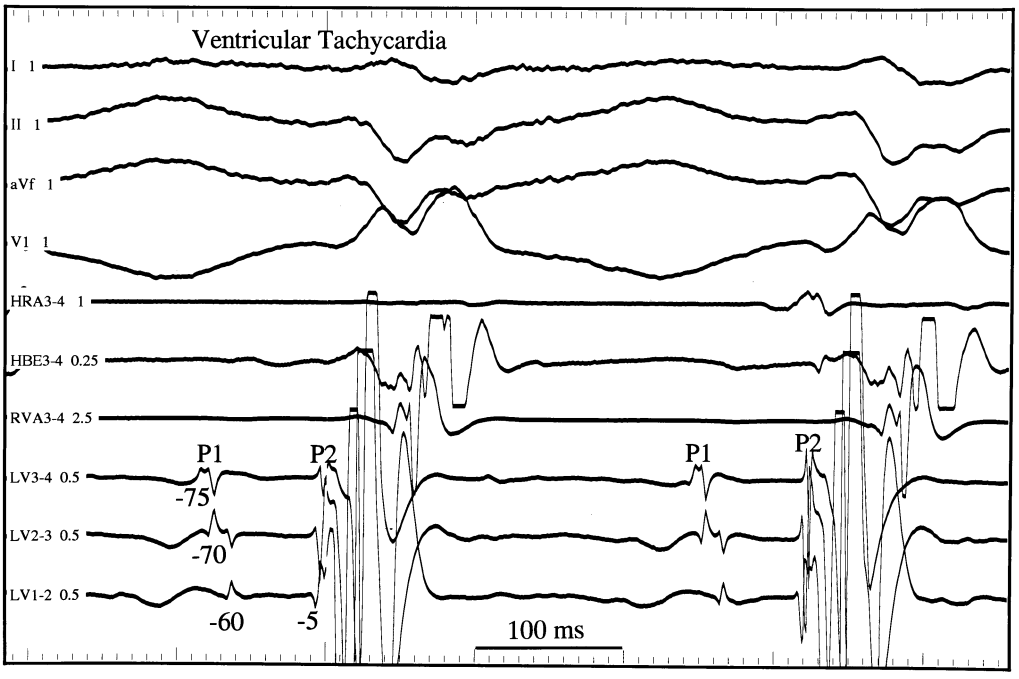

Figure 7. Patient 1. Recordings at the site of successful ablation during ventricular tachycardia. Diastolic potential and P2 were recorded in the midseptal area. The proximal two electrodes of ablation catheter recorded the P1 15 ms earlier than the distal pair of electrodes. HBE = His bundle electrogram; HRA = high right atrium; LV = left ventricle; P1 = diastolic potential; P2 = presystolic Purkinje potential; RVA = right ventricular apex.

5) Pacing from the successful ablation site did not produce the QRS configurations similar to that of VT; however, PPI to VT CL difference was within 30 ms.
6) Mean ratio of P1 to QRS to VT CL was 18 ± 8% (range 8 to 39%). This suggests that P1 was located at the central to exit portion of the slow conduction, according to Stevenson et al. (19).
7) The P1 appeared after the QRS complex during sinus

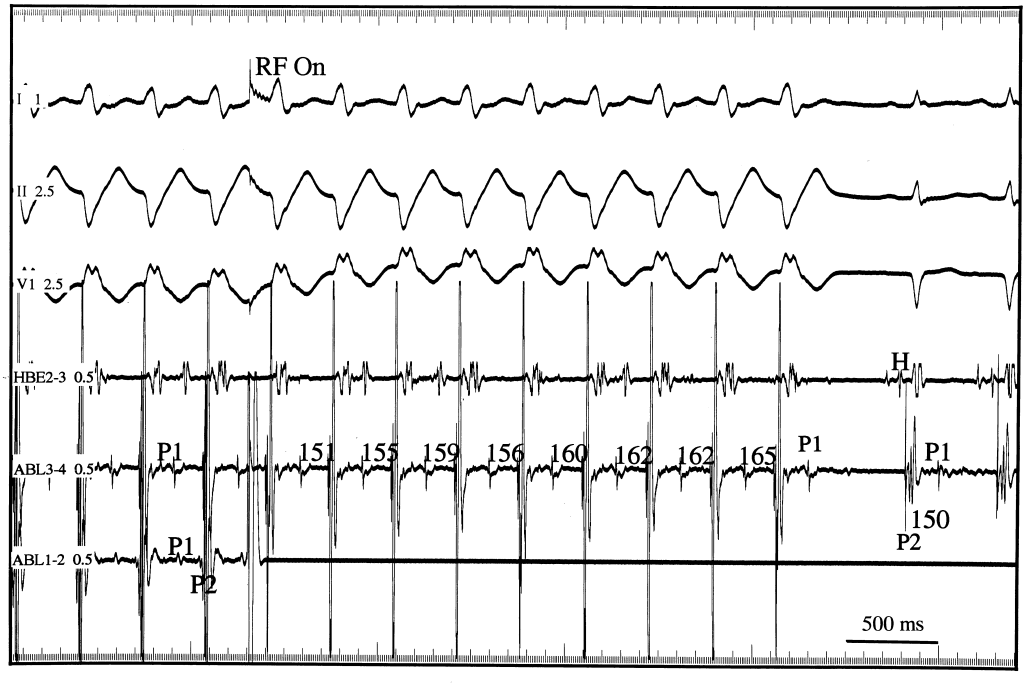

Figure 8. Patient 7. Application of radiofrequency current delivered during ventricular tachycardia. During energy application P1-P2 interval was gradually prolonged, and ventricular tachycardia was terminated by block between P1 and P2. After ablation the P1 occurred after the QRS complex during sinus rhythm. ABL = ablation catheter; H = His bundle electrogram; HBE = His bundle electrogram; P1 = diastolic potential; P2 = presystolic Purkinje potential; RF = radiofrequency current.

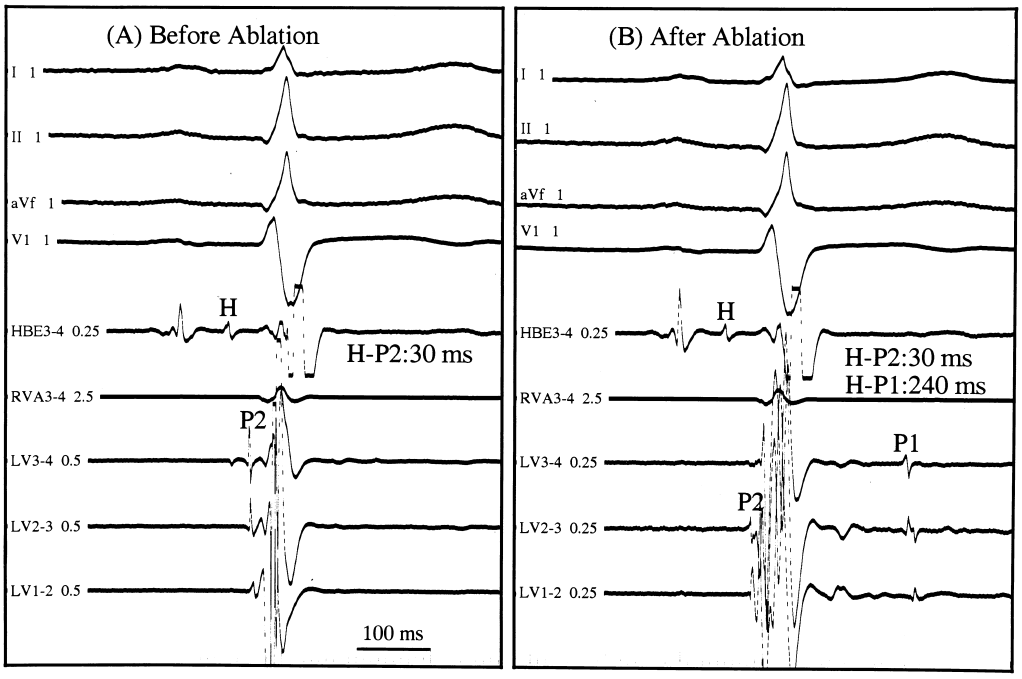

Figure 9. Patient 1. Successful ablation site during sinus rhythm. **(A)** Before ablation. Diastolic potential was not observed during sinus rhythm. **(B)** After ablation, the P1 occurred after the QRS complex. The activation sequence for P1 was identical to that observed during VT shown in Figure 7. H = His bundle electrogram; HBE = His bundle electrogram; LV = left ventricle; P1 = diastolic potential; P2 = presystolic Purkinje potential; RVA = right ventricular apex; VT = ventricular tachycardia.

Table 2. Purkinje Potentials During Sinus Rhythm After Ablation

Patient	P2-QRS Interval (ms)	H-P1 Interval (ms)	H-P1 Interval Prolongation by AP	S-P1 Interval Prolongation by VP	H-P1 Interval After Verapamil (ms)	S-P1 Interval Change After ATP During VP
Double Potential Group (n = 15)						
1	15	230	no	yes	305	no change
2	20	370	no	yes	460	no change
3	20	410	yes	yes	460	no change
4	20	400	no	yes*	500	no change
5	0	460	N/A	N/A	N/A	N/A
6	18	220	no	yes	N/A	N/A
7	10	205	no	yes	N/A	N/A
8	14	270	no	yes	N/A	N/A
9	18	380	no	no	N/A	N/A
10	15	330	N/A	N/A	N/A	N/A
11	30	615	no	yes	N/A	N/A
12	13	350	no	yes	N/A	N/A
13	20	325	no	no	N/A	N/A
14	25	435	no	no	530	N/A
15	10	200	yes*	yes	230	no change
	17 ± 7	347 ± 113			414 ± 119	
Single Potential Group (n = 5)						
16	8	—	—	—	—	—
17	8	—	—	—	—	—
18	0	—	—	—	—	—
19	14	—	—	—	—	—
20	16	—	—	—	—	—
	9 ± 6					

*No change in the baseline, but prolonged by pacing after verapamil.
AP = atrial pacing; ATP = adenosine triphosphate disodium; H = His-bundle electrogram; N/A = not available; S = ventricular stimulus; VP = ventricular pacing.

rhythm after successful ablation and QRS-P1 showed a decremental property during atrial/ventricular pacing and by the intravenous administration of verapamil.

Electrophysiological results in the single potential group are summarized as follows:

1) Only single fused Purkinje potential was recorded at the middle or inferior apical septum,
2) Pacing from the successful ablation site produces the QRS configurations similar to that of VT, and PPI-VT CL difference was within 30 ms.

Mechanism of tachycardia. The hypothesized VT circuit in the double potential group is depicted in Figure 12. In this circuit, P1 represents the activation potential in the distal portion of the specialized Purkinje tissue, and that has decremental properties and verapamil-sensitivity. The P2 represents the activation potential of the left posterior fascicle or of the Purkinje fiber near the left posterior fascicle. There is a link (network) between P1 and P2. During sinus rhythm the activation goes from P2 to P1 at the point of fusion; therefore, P1 is buried in the local ventricular activation (Fig. 12A). During VT, P1 and P2 activate in the reverse direction (Fig. 12B). This explains why the activation sequences of P2 were reversed during sinus rhythm and VT. During concealed entrainment from VT exit (e.g., at a CL of 400 ms as in Fig. 3A), P2 and P1 are activated orthodromically and the antidromic wave front blocks, presumably in the connection between P1 and P2 (Fig. 12C). The orthodromic wave front of the preceding (n-1)th beat also blocks in the connection between P1 and P2 because it encounters the refractoriness created by the antidromic wave front from (n)th pacing impulse. The orthodromic wave front from the last pacing impulse continues the tachycardia with resetting. Entrainment pacing with shorter CL (e.g., at a CL of 380 ms as in Fig. 3B), the distal portion of P1, activates antidromically and the antidromic wave front blocks at the middle portion of P1 in the area of slow conduction (Fig. 12D). The orthodromic wave front from the last pacing impulse continues and resets the tachycardia. However, the interval from the last pacing stimulus to orthodromically activated P1 prolongs because of rate-dependent conduction delay in the area of the slow conduction. During entrainment pacing with shorter CL or the shorter starting coupling interval (e.g., at a starting coupling interval of 300 ms as in Fig. 3C), P1 activates antidromically, and the antidromic wave front blocks at the proximal portion of P1 in the area of slow conduction (Fig. 12E). However, the orthodromic wave front from the previous paced beat also blocks. It blocks independent of either collision with or refractoriness secondary to the previous antidromic wave front. Because both the antidromic and orthodromic wave fronts of the same beat are blocked, the VT is interrupted. Radiofrequency catheter ablation resulted in the elimination of the conduction between P1 and P2. When this segment was ablated, the P1 activation proceeds orthodromically around the circuit and subsequently backs from a proximal to distal direction during sinus rhythm. This explains why P1 appears after ablation in the middiastolic period with the same activation sequence as during VT (Fig. 12F).

Ventricular tachycardia circuit in the single potential group is still undetermined from our data. In this group, P1 could not be detected, and P2 was recorded only at the VT exit site. We can speculate that the circuit may involve less of the Purkinje system or the area of slow conduction may not be close to the endocardial surface.

Previous studies. The exact nature of the reentry circuit in idiopathic left VT is still unclear. Kottkamp et al. (14) suggest that it is a microreentry circuit in the region of the left posterior fascicle. Nakagawa et al. (10) suggest that the circuit is confined to the Purkinje system, which is insulated from the surrounding ventricular myocardium. Wen et al. (13) demonstrated that the slow conduction zone of the reentry circuit is of considerable size, extending from the midseptum to the inferior apical septum of the LV. Lai et al. (16) have demonstrated the entrance and exit sites of slow conduction zone in a patient with ILVT. Intracardiac recordings from their site of successful ablation showed both diastolic P1 and presystolic P2; their hypothetical mechanism of ILVT is quite similar to ours. They proposed a false tendon or interlacing Purkinje fiber as a link between the slow conduction tissue and left posterior fascicle.

Recently, Tsuchiya et al. (18) also reported the significance of late diastolic potential in verapamil-sensitive ILVT. However, the characteristics of their diastolic potential are different from our P1. They recorded the diastolic potential in a small area at the basal to middle septal regions close to the main trunk of left bundle branch. And there is no decremental property in the interval from stimulus to the diastolic potential during entrainment pacing. As they also suggested, their diastolic potential seems to represent the excitation at the entrance to the critical slow conduction. Their diastolic potential was recorded earlier from the distal than the proximal mapping electrodes, suggesting the proximal electrodes recorded the activation of the proximal bystander as in Figure 12B.

Differentiation of critical potential after the QRS complex from T-wave and bystander potentials. After the application of RF current, the ablation catheter sometimes records the potential of the T-wave because the ST-T segment is markedly elevated after RF current delivery. Critical potential related to the VT circuit can be differentiated from this T-wave potential by atrial or ventricular pacing. The Q-T interval is shortened during pacing; however, the H-P1 interval is usually prolonged as we have demonstrated (Fig. 7).

Bystander Purkinje or fascicular potential also seems to appear after the QRS complex after the RF ablation of these fibers. Jazayeri et al. (20) have reported retrograde transseptal activation of right bundle branch after the QRS complex during sinus rhythm. Differentiation of the critical potential

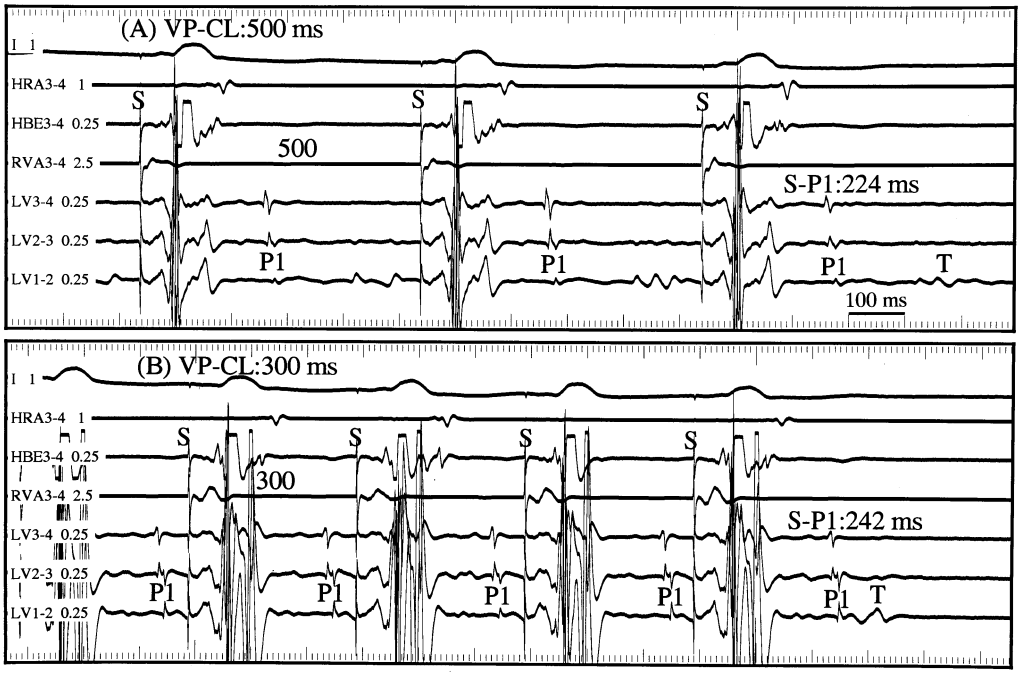

Figure 10. Patient 1. Right ventricular pacing after successful ablation. **(A)** The S-P1 interval was 224 ms during right ventricular pacing at a cycle length of 500 ms. **(B)** The S-P1 interval increased to 242 ms at a cycle length of 300 ms. CL = cycle length; HRA = high right atrium; P1 = diastolic potential; T = T-wave; VP = ventricular pacing.

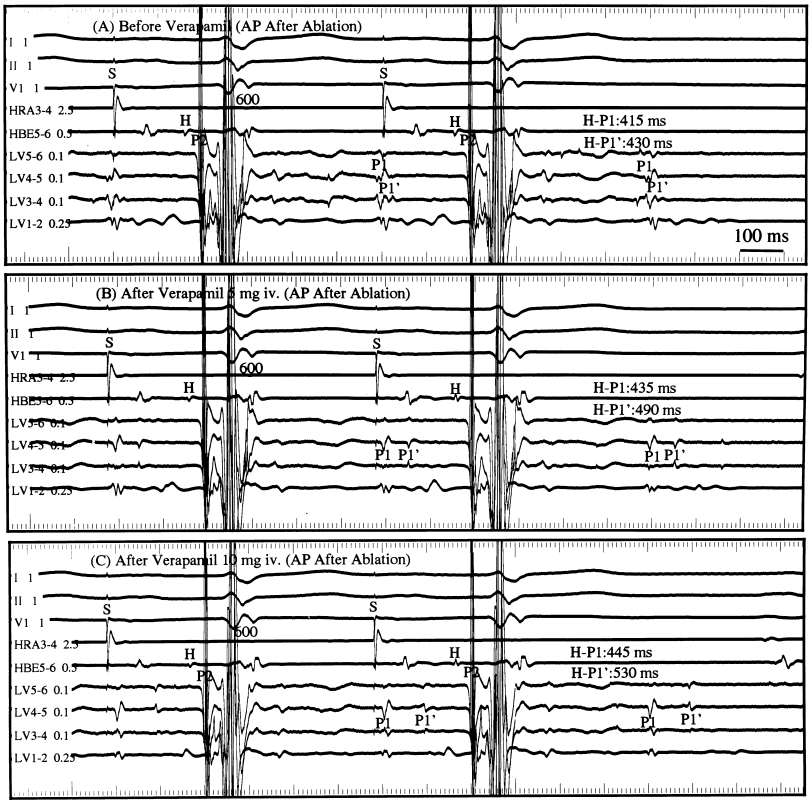

Figure 11. Patient 14. Diastolic potential before and after verapamil during atrial paced rhythm. **(A)** After ablation, the split P1 potentials (P1 and P1′) were observed after the QRS complex. **(B), (C)** After intravenous administration of verapamil, the dose dependent prolongation of His-bundle (H) to P1 (H-P1) interval, H-P1′ interval and P1-P1′ interval occurred. AP = atrial pacing; P1 = diastolic potential.

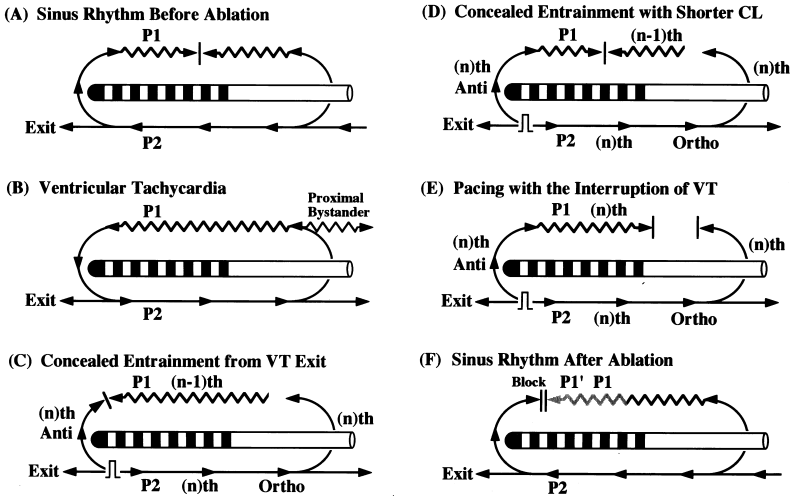

Figure 12. Schematic representation of the mechanism. See text for discussion. CL = cycle length; P1 = diastolic potential; P2 = presystolic Purkinje potential; VT = ventricular tachycardia.

from the bystander potential can be done by the activation sequence. The activation sequence for the critical potential after ablation should be identical to that observed during VT.

Clinical implications. These findings indicate the optimal site for catheter ablation of verapamil-sensitive ILVT. When P1 and P2 are recorded from the midseptal area during VT, this site should be targeted. If such a diastolic potential cannot be detected, the application of RF current to the earliest ventricular activation with a fused Purkinje potential may be carried out. We previously reported the use of this same strategy for verapamil-sensitive ILVT with an RBBB configuration and *right*-axis deviation (21). The mechanisms for both tachycardias may be the same, except that P2 is the potential of the left anterior fascicle or the left posterior fascicle.

The appearance of P1 after the QRS complex during sinus rhythm appeared to be a useful marker for the effective RF application. However, it is not enough to prove the total suppression of the tachycardia. As we have recently reported, P1 can appear after the QRS complex when there is a unidirectional block in the direction from P2 to P1 (22). And VT can be initiated if there is a residual conduction from P1 to P2. In such a case, a premature ventricular complex with the same QRS configuration as that of VT is usually observed after P1. Total elimination of this "ventricular echo beat" is necessary to cure the tachycardia.

Study limitations. First, selective pacing of P1 was unavailable because the pacing stimuli resulted in capture of not only the P1, but also the adjacent myocardium. Second, RF catheter ablation at the VT exit site was not applied in the double potential group. We cannot be certain that the site showing two distinct P1 and P2 was the only optimal one for ablation.

Conclusions. This study demonstrates that P1 and P2 are critical potentials in a circuit of verapamil-sensitive ILVT and suggests the presence of a macroreentry circuit involving the normal Purkinje system and the abnormal Purkinje tissue with decremental property and verapamil-sensitivity.

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