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Effects of Verapamil and Lidocaine on Two Components of the Re-entry Circuit of Verapamil-Sensitive Idiopathic Left Ventricular Tachycardia

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OBJECTIVES We characterized pharmacologically the slow conduction zone of verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) with regard to the late diastolic potential (LDP).

BACKGROUND We showed that the slow conduction zone of ILVT could be divided into two components by LDP; that is, the distal component with a tachycardia-dependent conduction delay property and the proximal one without it.

METHODS Electrophysiologic studies were performed in eight consecutive patients. The LDP was recorded during left ventricular (LV) mapping during ILVT. Entrainment was performed from the right ventricular outflow tract while recording LDP. The effects of lidocaine (1 mg/kg body weight) and verapamil (0.5 or 1.0 mg) were examined during entrainment.

RESULTS The LDPs preceding the Purkinje potential (PP) were serially recorded from the upper third to the middle of the LV septum along the narrow longitudinal line. The ventricular tachycardia (VT) cycle length increased after lidocaine ($p < 0.05$), and further after verapamil ($p < 0.05$). The increments in the VT cycle length after administration of the drugs strongly correlated with those in LDP-PP ($r > 0.9$ for both drugs). The interval from the ventricular potential to LDP was unchanged after administration of the drugs. In one patient, verapamil terminated VT by local conduction block between LDP and PP. The LDP-PP measured during entrainment increased after lidocaine, and further after verapamil, whereas the interval from the stimulus to LDP remained unchanged.

CONCLUSIONS The component distal to LDP is mainly calcium channel-dependent and partly depressed sodium channel-dependent. The proximal component is considered to be sodium channel-dependent (normal). (J Am Coll Cardiol 2001;37:1415–21) © 2001 by the American College of Cardiology

Verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) constitutes a relatively small but distinct entity occurring in relatively young patients (1–9). It has been shown in most patients that the underlying mechanism of ILVT is re-entry with an excitable gap (3–7). With the use of the entrainment technique (10–12), we showed that a slow conduction zone with a conduction delay property in response to the increase in the pacing rate is present between the right ventricular outflow tract (RVOT) and the earliest ventricular activation site at the posteroapical left ventricular (LV) septum. In addition, we found that small doses of verapamil and lidocaine selectively suppress conduction through this slow conduction zone of ILVT (4,5,7).

Recently, we reported that a discrete late diastolic potential (LDP) preceding the Purkinje potential (PP) was recorded in the middle to upper third LV septum during ILVT, and the entire slow conduction zone could be divided into two components by LDP: one in the distal part to the LDP recording site, showing a conduction delay property (the distal component), and the other in the proximal part to the LDP recording site, showing no conduction delay property (the proximal component) (7). In the present study, we examined the responses of these two components to lidocaine (a sodium channel-blocking agent) and verapamil (a calcium channel-blocking agent) both during ventricular tachycardia (VT) and entrainment.

METHODS

Study patients. Eight consecutive patients (6 men and 2 women, 16 to 44 years old) with recurrent, sustained VT and with no underlying heart disease were studied. The electrocardiogram (ECG) recorded during VT exhibited a right bundle branch block configuration and a superior or left axis in all patients. Intravenous verapamil (5 to 10 mg) was demonstrated to be effective in terminating VT in all patients.

Electrophysiologic study. Written, informed consent was obtained from all patients before the study. All antiarrhythmic drugs were discontinued for >5 half-lives of each drug before the study. Using standard techniques, two or three 6F quadripolar electrode catheters (Josephson, Bard Electrophysiology, Billerica, Massachusetts) were placed at the right ventricular (RV) apex, RVOT and/or His bundle region and were used for recording bipolar electrograms and pacing. A 7F deflectable quadripolar electrode catheter with a 2-mm interelectrode interval (Radii-T, Cardiac Pathways, Sunnyvale, California) (n = 8) or a 6F deflectable eight-

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Abbreviations and Acronyms

ILVT = idiopathic left ventricular tachycardia
LDP = late diastolic potential
LV = left ventricular
PP = Purkinje potential
RV = right ventricular
RVA = right ventricular apex
RVOT = right ventricular outflow tract
st = stimulus
VP = ventricular potential
VT = ventricular tachycardia

pole electrode catheter with a 2.5-mm interelectrode interval (EPT-Dx-S, EP Technology, Sunnyvale, California) (n = 4) was retrogradely inserted into the LV to perform endocardial mapping during VT to identify the earliest ventricular activation site and to record LDP by recording electrograms from the each electrode pair. All bipolar electrograms were filtered between a bandpass of 50 and 600 Hz and recorded simultaneously with three or six ECG leads (I, II, III, aVF, V_1 and V_5) with the use of a polygraph (Cardiolab system, Prucka Engineering, Houston, Texas). Ventricular pacing was performed at a stimulus strength of twice the diastolic threshold and with a pulse width of 2 ms, using a programmable stimulator (SEC-3102, Nihon Kohden, Tokyo, Japan).

Entrainment from RVOT. Only the quadripolar catheter was used to record LDP in the entrainment study, because we could not stabilize the position of the eight-pole electrode catheter in the LV, and thus we could not record stable LDP during the entrainment study. We positioned the tip of the quadripolar catheter at the earliest LDP recording site and used this LDP in measuring local conduction times during entrainment as well as during VT. In all patients, entrainment of VT by pacing from the RVOT at a rate 5 to 10 beats/min faster than the induced VT rate was attempted while recording the LDP. When VT was still present after termination of the pacing, rapid pacing was again performed, with an increase in the pacing rate by 5 to 10 beats/min. This procedure was repeated until VT was interrupted or pacing reached the rate of 40 to 50 beats/min faster than the VT rate. In the last entrained beat, the intervals from the stimulus artifact to the electrogram at the RV apex (st-RVA) and from the stimulus artifact to the LDP (st-LDP), as well as that from the LDP to PP (LDP-PP), were measured at every pacing rate.

Effects of lidocaine and verapamil during entrainment. During VT, lidocaine (1 mg/kg body weight over 1 min) was intravenously administered in six of eight patients, and the entrainment study was repeated. Approximately 15 min after the lidocaine study, when the VT rate and local conduction times returned to the control values, a small dose of verapamil (0.5 mg) was intravenously administered to all patients. When the VT rate was not decreased by about 20 beats/min, another dose of 0.5 mg (total dose 1.0 mg) was administered. The entrainment study was repeated in seven of eight patients. In the remaining one patient, verapamil (1.0 mg) resulted in VT termination, and the entrainment study was not performed. Throughout the study, we held the catheter carefully to avoid applying pressure at the tip of the catheter.

Before and after injection of each antiarrhythmic agent, the VT cycle length and the intervals from PP to the local ventricular potential (VP) (PP-VP), from VP to LDP (VP-LDP) and from LDP to PP (LDP-PP) were measured during VT. Measurements of st-LDP, st-RVA and LDP-PP during entrainment were repeated at every pacing rate after each antiarrhythmic agent.

Data analysis. Continuous variables were expressed as the mean value ± SD. Statistical analysis was done with repeated measures one-way analysis of variance, in which the F value was interpreted on the basis of Huyhn-Feld corrected p values. Subsequent multiple comparisons were performed by using a Bonferroni-type multiple comparison test for three or more variables. Correlations were tested by the Pearson correlation coefficient. For the analysis of the effect of verapamil on the relationship between LDP-PP and pacing rate, we performed a multiple regression analysis using a dummy variable to encode the treatment condition within each patient, as well as dummy variables to encode the eight different patients. A p value <0.05 was considered statistically significant.

RESULTS

Sustained VT with a mean cycle length of 342.8 ± 49.3 ms (rate 178.3 ± 26.1 beats/min) and with the same QRS morphology as that of spontaneous VT was repeatedly induced in all patients. Endocardial mapping during VT identified the earliest ventricular activation site at the posteroapical LV septum, with an activation time of −25.5 ± 3.1 ms, relative to the onset of the QRS complex in all patients. The LDPs preceding PP were recorded serially from the upper third to the middle of the LV septum along the narrow longitudinal line in four patients in whom ventricular mapping with the eight-pole catheter was performed (Fig. 1). The LDP appeared earlier as the recording site became closer to the base of the LV. At the middle or lower third of the LV septum, the LDP appeared latest and was fused with PP. We used the earliest LDP in measuring the local conduction times. The LDP-PP, PP-VP and VP-LDP intervals at the earliest LDP recording site were 68.5 ± 24.3 ms, 21.4 ± 7.2 ms and 256.3 ± 39.1 ms, respectively, and the duration of the LDP was 21.5 ± 3.4 ms.

Effects of lidocaine and verapamil on local conduction times and VT cycle length. In all patients but one, neither lidocaine nor verapamil terminated VT. After lidocaine (1 mg/kg per min), the VT cycle length significantly increased from 355.3 ± 50.6 to 377.5 ± 45.3 ms ($p < 0.01$ vs. control). The LDP-PP increased from 77.7 ± 18.9 to

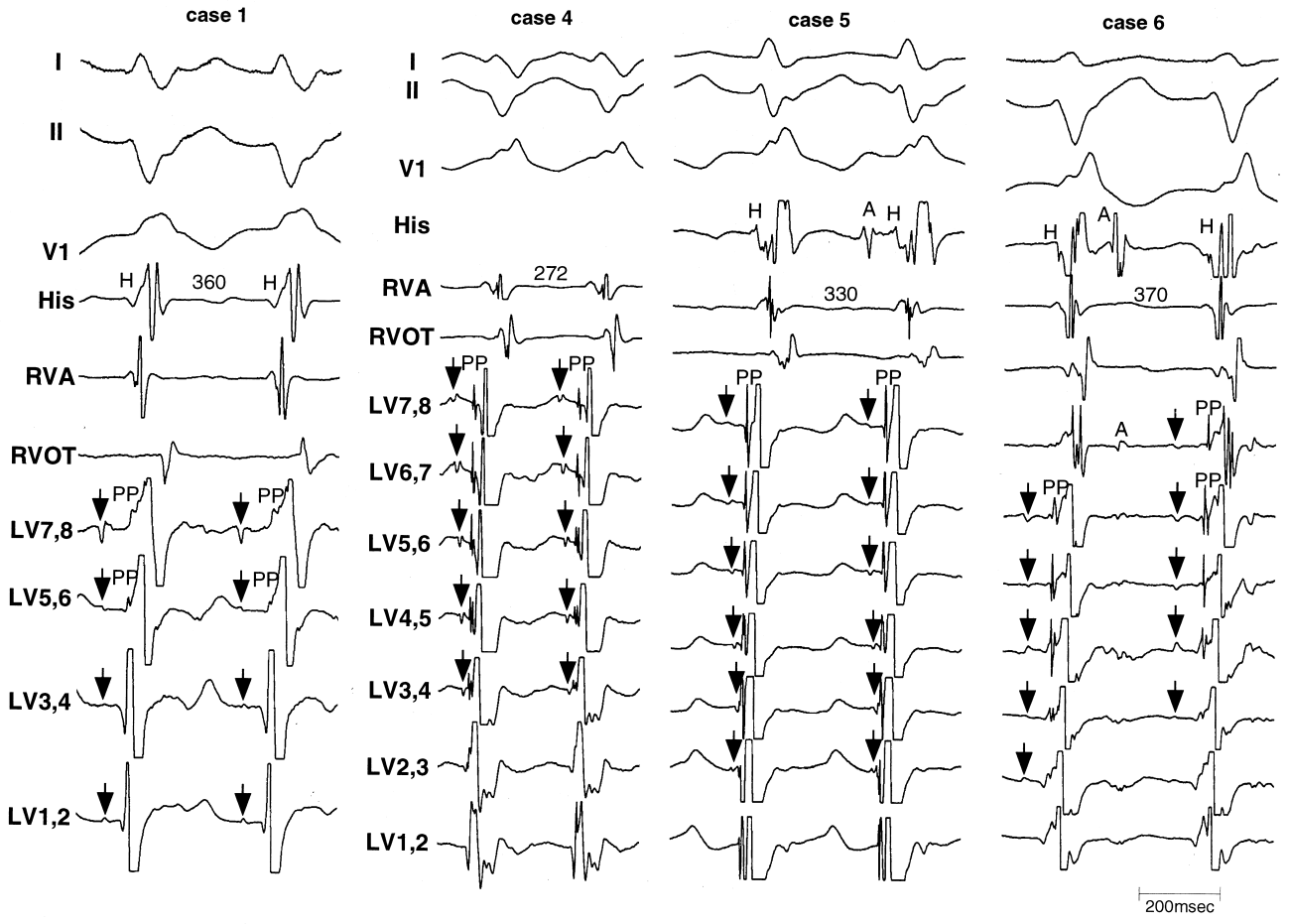

Figure 1. Examples of late diastolic potentials (LDPs) **(arrows)** preceding Purkinje potential (PP) in four patients (cases 1, 4, 5 and 6), recorded serially from the middle to upper third of the left ventricular (LV) septum along the narrow longitudinal line during ventricular tachycardia (VT). Tracings are electrocardiographic leads I, II and V_1, and intracardiac electrograms are recorded from the His bundle region (His), right ventricular apex (RVA), right ventricular outflow tract (RVOT) and eight-electrode catheter located in the LV septum (LV1 to LV8). All numbers are in ms. A = atrial potential; H = His bundle potential.

99.2 ± 17.0 ms ($p < 0.05$ vs. control), whereas PP-VP and VP-LDP remained unchanged. After verapamil (0.5 or 1.0 mg), the VT cycle length increased from 347.0 ± 52.4 to 394.6 ± 60.1 ms ($p < 0.01$ before verapamil), which was significantly greater than that after lidocaine ($p < 0.05$), and LDP-PP also increased from 76.3 ± 28.2 to 118.9 ± 34.1 ms ($p < 0.01$ before verapamil), which was significantly greater than that after lidocaine ($p < 0.05$), whereas PP-VP and VP-LDP remained unchanged. The LDP duration did not significantly change after lidocaine and verapamil. The increases in the VT cycle length after lidocaine and verapamil significantly correlated with those in LDP-PP ($r = 0.98$, $p = 0.0006$ for control vs. lidocaine; $r = 0.93$, $p = 0.0009$ for pre-verapamil vs. post-verapamil) (Fig. 2). Neither of them was correlated with the change in PP-VP ($r = 0.18$, $p = 0.74$ for control vs. after lidocaine; $r = 0.10$, $p = 0.81$ for pre-verapamil vs. post-verapamil) or the change in VP-LDP ($r = 0.17$, $p = 0.75$ for control vs. after lidocaine; $r = 0.53$, $p = 0.18$ for pre-verapamil vs. post-verapamil).

In one patient, verapamil induced a beat-to-beat variation in both the VT cycle length and LDP-PP and terminated VT, with local conduction block between the LDP and PP recording sites (Fig. 3). Both PP-VP and VP-LDP were

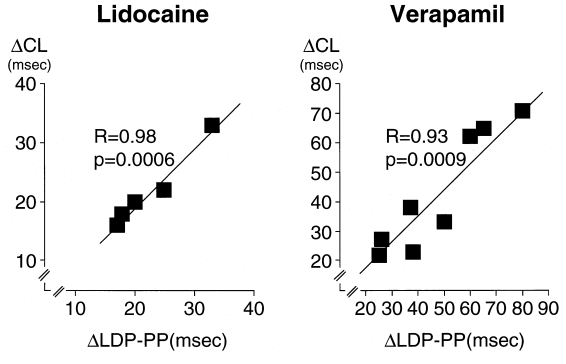

Figure 2. Correlations of the increases in VT cycle length (ΔCL) with those of the intervals from LDP to PP (ΔLDP-PP) after lidocaine (1 mg/kg) and verapamil (0.5 or 1.0 mg). There were significant correlations between the increases in VT cycle length and those in LDP-PP after lidocaine and verapamil ($r = 0.98$, $p = 0.0006$ for control study vs. post-lidocaine; $r = 0.93$, $p = 0.0009$ for pre-verapamil vs. post-verapamil). Abbreviations as in Figure 1.

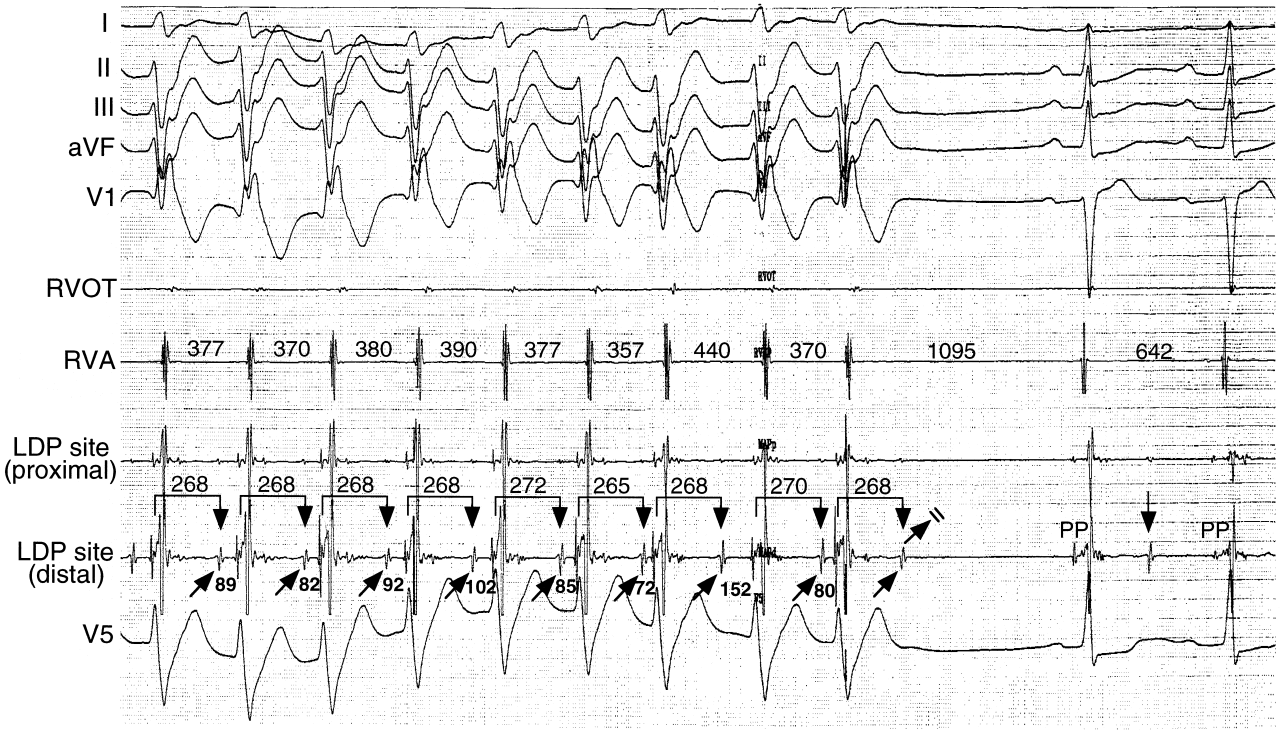

Figure 3. A beat-to-beat variation in the VT cycle length occurred after intravenous administration of verapamil (1 mg). Tracings are ECG leads I, II, III, aVF and V_1, and intracardiac electrograms are recorded at the RVOT and RVA, with the distal and proximal pairs of the mapping catheter where LDP was recorded (proximal and distal LDP sites), as well as ECG lead V_5. Note that the VT cycle length and the interval from LDP to PP were variable, whereas the intervals from PP to the local ventricular potential (VP) and from VP to LDP were almost constant. It is also noted that VT was finally terminated due to local conduction block between the LDP and PP recording sites. All numbers are in ms. Abbreviations as in Figure 1.

almost constant after verapamil administration. The VT cycle length was significantly correlated with LDP-PP ($r = 0.99$, $p = 0.0001$), although not with PP-VP ($r = 0.19$, $p = 0.65$) or VP-LDP ($r = 0.08$, $p = 0.85$).

Ventricular tachycardia entrainment and the effects of lidocaine and verapamil. In all patients, entrainment phenomena, including constant fusion, except for the last entrained beat, and progressive fusion, were demonstrated by rapid pacing from the RVOT. A long conduction interval between the pacing site and the earliest ventricular activation site, indicating a slow conduction zone, was demonstrated during entrainment.

We also performed VT entrainment while recording the earliest LDP and examined the effects of lidocaine and verapamil. The LDP was captured orthodromically during entrainment, whereas the ventricular potentials at the LDP recording site and RVA were captured antidromically. The st-LDP and LDP-PP, which were measured in the last entrained beat, were 319.1 ± 34.8 ms and 81.9 ± 34.8 ms, respectively, at the pacing cycle length of 320.7 ± 49.6 ms. The st-RVA was 58.8 ± 14.1 ms. It is noted that st-LDP was much longer than LDP-PP, and the percentages of st-LDP and LDP-PP to the interval from the stimulus artifact to PP, which was measured in the slowest pacing rate, were 80 ± 5% and 20 ± 5%, respectively (Table 1). When the pacing rate was increased, LDP-PP was prolonged, whereas st-LDP and st-RVA remained unchanged in all patients.

Figure 4 shows an example of the effects of lidocaine and verapamil on st-LDP, LDP-PP and st-RVA measured during entrainment. During the control study, st-LDP, LDP-PP and st-RVA were 370, 112 and 40 ms, respectively. After lidocaine administration, LDP was again captured orthodromically, and LDP-PP was prolonged to 168 ms, whereas st-LDP and st-RVA remained unchanged. After verapamil administration, LDP was captured orthodromically, and LDP-PP was further prolonged to 212 ms, whereas st-LDP and st-RVA remained unchanged. The effects of lidocaine ($n = 6$) and verapamil ($n = 7$) on local conduction times measured during entrainment are shown in Table 1. In all patients, LDP-PP measured during entrainment was prolonged after lidocaine and was further prolonged after verapamil, although the pacing rate used for entrainment was lower, especially after verapamil, as compared with that during the control study. The st-LDP and st-RVA remained unchanged. The relationship between the LDP-PP interval and pacing rate was compared between the conditions during the control study and those after verapamil administration, by using multiple linear regression. The regression equation was expressed as follows: when LDP-PP was named "Y" and the pacing rate "X," $Y = -255.56 + 44.25S_1 + 32.38S_2 + 129.74S_3 - 88.28S_4 - 43.53S_5 + 74.40S_6 - 95.92S_7 + 2.04X + 12.91D - 0.294X \times D$. In this equation, D is a dummy variable to encode before verapamil ($D = 1$) and after verapamil ($D =$

Table 1. Effects of Lidocaine and Verapamil on Local Conduction Times Measured During Entrainment

	Control Study				Lidocaine (1 mg/kg)				Verapamil (0.5 or 1.0 mg)			
Patient No.	Pacing Rate (beats/min)	st-LDP (ms)	LDP-PP (ms)	st-RVA (ms)	Pacing Rate (beats/min)	st-LDP (ms)	LDP-PP (ms)	st-RVA (ms)	Pacing Rate (beats/min)	st-LDP (ms)	LDP-PP (ms)	st-RVA (ms)
1	180	330	95	70	180	330	100	70	150	330	105	70
	190	330	110	70	190	330	125	70	160	335	165	70
	200	330	155	70					170	335	220	70
2	170	328	85	85	170	328	102	85	160	328	150	85
	180	328	95	85	180	328	105	85	170	328	170	85
	190	328	110	85	190	328	130	85				
	200	328	138	85	200	328	138	85				
	210	328	160	85	210	328	166	85				
3	145	370	115	40	140	370	142	38	140	370	185	38
	150	370	135	40	145	370	160	38	145	370	215	38
	160	370	195	40	150	370	170	38				
4	230	260	70	65	220	260	102	65	200	260	128	65
	240	260	85	65	230	260	110	65	210	260	138	65
	250	260	99	65	240	260	130	65	220	260	156	65
	260	260	115	65	250	260	145	65				
5	190	345	72	55	180	345	80	55	180	345	105	55
	200	345	78	55	190	345	85	55	190	345	110	55
	210	345	85	55	200	345	92	55	200	345	129	55
	220	345	110	55	210	345	132	55	210	345	152	55
	230	345	130	55	220	345	160	55	220	345	210	55
					230	345	240	55				
6	170	320	120	50	155	320	125	55	155	320	140	55
	180	320	175	50	160	320	132	55	160	320	162	55
	190	320	190	50	170	320	202	55				
7	230	280	60	50					200	295	110	50
	240	280	70	50					210	295	142	50
	250	280	90	50								
8	195	320	38	55								
	200	335	55	55								
	205	335	65	55								
	210	335	75	55								

LDP = late diastolic potential; LDP-PP = interval from LDP to Purkinje potential (PP); RVA = right ventricular apex; st = pacing stimulus; st-LDP = interval from pacing stimulus to LDP; st-RVA = interval from pacing stimulus to electrogram at RVA.

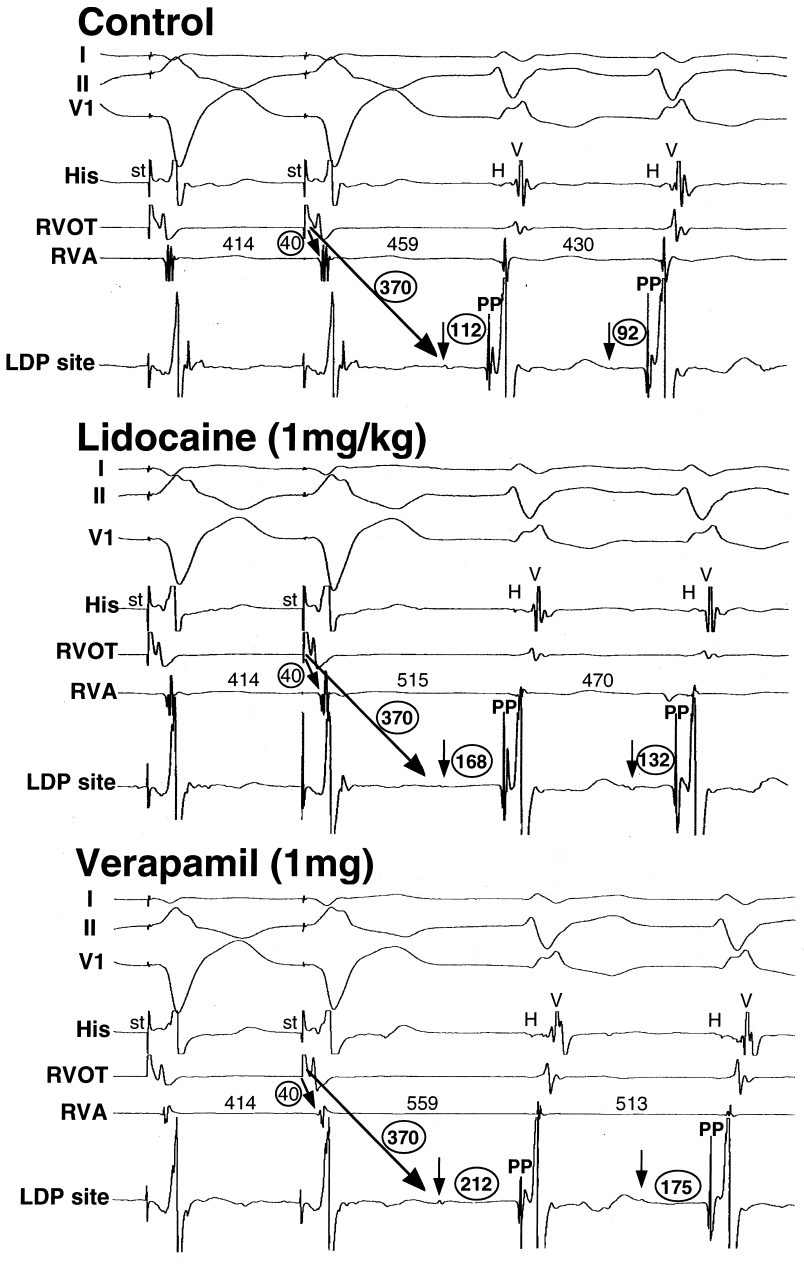

Figure 4. Examples of entrainment by pacing from the RVOT at a rate of 145 beats/min while recording LDP during the control study **(upper panel)**, after lidocaine administration **(middle panel)** and after verapamil administration **(lower panel)**. Tracings are ECG leads I, II and V_1, and intracardiac electrograms are recorded at the His bundle region (His), RVOT, RVA and LDP site **(arrows)**. All numbers are in ms. The **numbers in circles** indicate conduction intervals from the LDP stimulus artifact (st) to the electrogram at the RVA and from LDP to PP. See text for discussion. Abbreviations as in Figure 1.

-1); S_1 through S_7 are subject dummy variables; the number 12.91 in the term 12.91D is a intercept shift caused by verapamil; and -0.294 in the term $-0.294X$ represents a verapamil-induced change in the slope in which $p = 0.0078$. From these findings, the increase in LDP-PP in response to the increase in the pacing rate was augmented after verapamil administration.

DISCUSSION

In this study, LDP preceding PP was recorded serially from the upper third to the middle of the LV septum along the narrow longitudinal line during VT. The earliest LDP was recorded at the base of the LV septum, whereas the latest LDP recorded at the middle or lower third of the LV septum was fused with PP. We measured the local conduction times with the use of the earliest LDP and found that the interval from the LDP to PP increased during entrainment in response to an increase in the pacing rate, whereas the st-LDP interval was constant. Thus, it is suggested that the earliest LDP reflects the excitation at the entrance of a "specific" component of slow conduction, and the following serially recorded LDPs might represent the serial excitations in this component.

Effects of lidocaine and verapamil on VT cycle length. A small dose of verapamil and lidocaine selectively suppressed the

conduction through the distal component, but neither of them affected the conduction in the proximal component, because LDP-PP was prolonged by the drugs, although PP-LDP was not. The increments in the VT cycle length after administering these drugs correlated with those in LDP-PP. Furthermore, in one patient, verapamil induced a beat-to-beat variation of the VT cycle length and LDP-PP interval and terminated VT, with local conduction block within the distal component. We held the catheter carefully throughout the study to avoid applying pressure at the tip of catheter, because pressure could cause the same phenomenon (7). Thus, verapamil and lidocaine slowed the VT rate by selectively suppressing the conduction through the distal component.

Ventricular tachycardia entrainment and the effects of lidocaine and verapamil. To further elucidate this issue, we performed entrainment while recording LDP before and after lidocaine and verapamil administration. We found that verapamil selectively suppressed the conduction through the distal component, because LDP-PP, but not st-LDP, was prolonged after verapamil. The magnitude of the increase in LDP-PP with an increase in the pacing rate was augmented after verapamil. Thus, it is further confirmed that the target site of verapamil is confined to the distal component. This study also showed that lidocaine slightly but significantly suppressed the conduction through the distal component. The present dose of lidocaine is unlikely to affect conduction within the normal myocardium, unless the pacing rate is sufficiently high, but it might suppress conduction through the myocardial cells with partially depolarized membrane potential (13,14). These findings suggested that the main cellular mechanism of the conduction through the distal component is calcium channel-dependent and also involves depressed sodium channel-dependent tissue.

In contrast, the conduction through the proximal component was affected by neither lidocaine nor verapamil, suggesting that the cellular mechanism of the proximal component is sodium channel-dependent (normal). The conduction time through the proximal component was relatively long and occupied 80 ± 5% of the total conduction time from the stimulus artifact to PP during entrainment. Anatomically, it reflects conduction from the RVOT to the upper LV septum, and the distance was ~3 to 4 cm at most, if measured directly. In general, conduction velocity through tissue with normal sodium channel-dependent conduction is expected to be faster than that through tissue operated by the other cellular mechanism (13). Thus, the conduction velocity in this proximal component is too slow to be explained by the conduction through the normal ventricular tissue with normal sodium channel-dependent conduction. It was reported that the conduction velocity of a fiber is reduced as the diameter is decreased and the length is increased (15). Thus, we submit a hypothesis that the excitation in the proximal component is conducted along an anatomically isolated, long, thin pathway, such as the transverse LV false tendon. This structure, which extends from the posteroinferior LV to the LV septum, in many cases, was reported to be related to ILVT (16,17). Further studies are required to clarify this issue.

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REFERENCES

1. Belhassen B, Rotmensch HH, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. Br Heart J 1981;46:679–82.
2. Klein GJ, Millman PJ, Yee R. Recurrent ventricular tachycardia responsive to verapamil. Pacing Clin Electrophysiol 1984;7:938–48.
3. Ohe T, Shimomura K, Aihara N, et al. Idiopathic sustained left ventricular tachycardia: clinical and electrophysiologic characteristics. Circulation 1988;77:560–8.
4. Okumura K, Matsuyama K, Miyagi H, Tsuchiya T, Yasue H. Entrainment of idiopathic ventricular tachycardia of left ventricular origin with evidence for re-entry with an area of slow conduction and effect of verapamil. Am J Cardiol 1988;62:727–32.
5. Okumura K, Yamabe H, Tsuchiya T, Tabuchi T, Iwasa A, Yasue H. Characteristics of slow conduction zone demonstrated during entrainment of idiopathic ventricular tachycardia of left ventricular origin. Am J Cardiol 1996;77:379–83.
6. Wen MS, Yeh SJ, Wang CC, Lin FC, Wu D. Successful radiofrequency ablation of idiopathic left ventricular tachycardia at a site away from the tachycardia exit. J Am Coll Cardiol 1997;30:1024–31.
7. Tsuchiya T, Okumura K, Honda T, et al. Significance of late diastolic potential preceding Purkinje potential in verapamil-sensitive idiopathic left ventricular tachycardia. Circulation 1999;99:2408–13.
8. Nakagawa H, Beckman KJ, McClelland JH, et al. Radiofrequency catheter ablation of idiopathic left ventricular tachycardia guided by a Purkinje potential. Circulation 1993;88:2607–17.
9. Kottkamp H, Chen X, Hindricks G, et al. Idiopathic left ventricular tachycardia: new insights into electrophysiological characteristics and radiofrequency catheter ablation. Pacing Clin Electrophysiol 1995;18:1285–97.
10. Okumura K, Olshansky B, Henthorn RW, Epstein AE, Plumb VJ, Waldo AL. Demonstration of the presence of slow conduction during sustained ventricular tachycardia in man: use of transient entrainment of the tachycardia. Circulation 1987;75:369–78.
11. Waldo AL, Plumb VJ, Arciniegas JG, et al. Transient entrainment and interruption of the atrioventricular bypass type of paroxysmal atrial tachycardia: a model for understanding and identifying re-entrant arrhythmia. Circulation 1983;67:73–83.
12. Okumura K, Henthorn RW, Epstein AE, Plumb VJ, Waldo AL. Further observations on transient entrainment: importance of pacing site and properties of the components of the re-entry circuit. Circulation 1985;72:1293–307.
13. Chen C, Gettes LS, Katzung BG. Effect of lidocaine on steady-state characteristics and recovery kinetics of (dV/dt)max in guinea pig ventricular myocardium. Circ Res 1975;37:20–9.
14. Lazzara R, Hope RR, El-Sherif N, Scherlag BJ. Effects of lidocaine on hypoxic ischemic cardiac cells. Am J Cardiol 1978;41:872–9.
15. Sherf L, James TN, Woods WT. Function of the atrioventricular node considered on the basis of observed histology and fine structure. J Am Coll Cardiol 1985;5:770–80.
16. Suwa M, Yoneda Y, Nagao H, et al. Surgical correction of idiopathic paroxysmal ventricular tachycardia possibly related to left ventricular false tendon. Am J Cardiol 1989;64:1217–20.
17. Thakur RK, Klein GJ, Sivaram CA, et al. Anatomic substrate for idiopathic left ventricular tachycardia. Circulation 1996;93:407–501.