Malignant Entity of Idiopathic Ventricular Fibrillation and Polymorphic Ventricular Tachycardia Initiated by Premature Extrasystoles Originating From the Right Ventricular Outflow Tract

Takashi Noda, MD, PhD,* Wataru Shimizu, MD, PhD,* Atsushi Taguchi, MD,*
Takeshi Aiba, MD, PhD,† Kazuhiro Satomi, MD,* Kazuhiro Suyama, MD, PhD,*
Takashi Kurita, MD, PhD,* Naohiko Aihara, MD,* Shiro Kamakura, MD, PhD*

Suita, Japan

OBJECTIVES The aim of this study was to assess the clinical characteristics and the efficacy of radiofrequency catheter ablation (RFCA) for idiopathic ventricular fibrillation (VF) and/or polymorphic ventricular tachycardia initiated by ventricular extrasystoles originating from the right ventricular outflow tract (RVOT).

BACKGROUND Ventricular fibrillation and/or polymorphic ventricular tachycardia are occasionally initiated by ventricular extrasystoles originating from the RVOT in patients without structural heart disease.

METHODS Among 101 patients without structural heart disease in whom RFCA was conducted for idiopathic ventricular tachyarrhythmias arising from the RVOT, we examined the clinical characteristics and the efficacy of RFCA in 16 patients with spontaneous VF and/or polymorphic ventricular tachycardia initiated by the ventricular extrasystoles originating from the RVOT.

RESULTS Among 16 patients, spontaneous episodes of VF were documented in 5 patients, and 11 patients had prior episodes of syncope. Holter recordings showed frequent isolated ventricular extrasystoles with the same morphology as that of initiating ventricular extrasystoles, and non-sustained polymorphic ventricular tachycardia with short cycle length (mean of 245 ± 28 ms) in all 16 patients. Radiofrequency catheter ablation by targeting the initiating ventricular extrasystoles eliminated episodes of syncope, VF, and cardiac arrest in all patients during follow-up periods of 54 ± 39 months.

CONCLUSIONS Our data suggest that the malignant entity of idiopathic VF and/or polymorphic ventricular tachycardia was occasionally present in patients with idiopathic ventricular arrhythmias arising from the RVOT. Radiofrequency catheter ablation was effective as a treatment option for this entity.   (J Am Coll Cardiol 2005;46:1288–94) © 2005 by the American College of Cardiology Foundation

Ventricular fibrillation (VF) and polymorphic ventricular tachycardia (PVT) are malignant arrhythmias resulting in sudden cardiac death (1–5). Recent studies by Haissaguerre

et al. (6,7) reported that idiopathic VF initiated by dominant triggers from distal Purkinje system or right ventricular outflow tract (RVOT) was successfully eliminated by radiofrequency catheter ablation (RFCA).

Although idiopathic ventricular tachycardia and ventricular extrasystoles (VE) originating from the RVOT in patients without structural heart diseases are considered

benign (8–12), VF and/or PVT are occasionally initiated by VE originating from the RVOT.

The present study is designed to assess the clinical characteristics and the efficacy of RFCA for the malignant entity of idiopathic VF and/or PVT initiated by VE originating from the RVOT.

METHODS

Patient characteristics. Sixteen patients who showed spontaneous VF and/or PVT initiated by the VE with left bundle branch block morphology and inferior axis in their clinical course (VF/PVT group) were enrolled in this study among 101 consecutive patients in whom RFCA was conducted for treatment of ventricular tachyarrhythmias arising from the RVOT. There were seven men and nine women ranging in age from 25 to 54 years (mean of 39 ± 10 years). In all patients, physical examination, chest roentgenogram, laboratory values, treadmill exercise test, echocardiographic study with wall motion analysis, Doppler screening, and signal-averaged electrocardiogram (SAECG) were per-

formed, and no structural heart disease was found. Patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (13) or Brugada syndrome (14) were excluded from this study. During hospitalization, the patients had frequent VE identical to the initiating beat of VF/PVT recorded by Holter recording or monitoring electrocardiogram (ECG) so that we could recognize the 12-lead QRS morphology of the initiating beats (Fig. 1A). In some cases, non-sustained PVT initiated by the VE of the RVOT origin could be recorded in the 12-lead ECG (Fig. 1B). We compared the clinical characteristics between the 16 patients with VF/PVT group and the remaining 85 patients in whom RFCAs were conducted for treatment of idiopathic monomorphic ventricular tachycardia arising from the RVOT (RVOT-VT group). Ventricular fibrillation was defined as a polymorphic ventricular tachyarrhythmia with hemodynamic decompensation requiring direct cardioversion for termination. Polymorphic ventricular tachycardia was defined as more than five consecutive beats with different QRS morphology and terminating spontaneously.
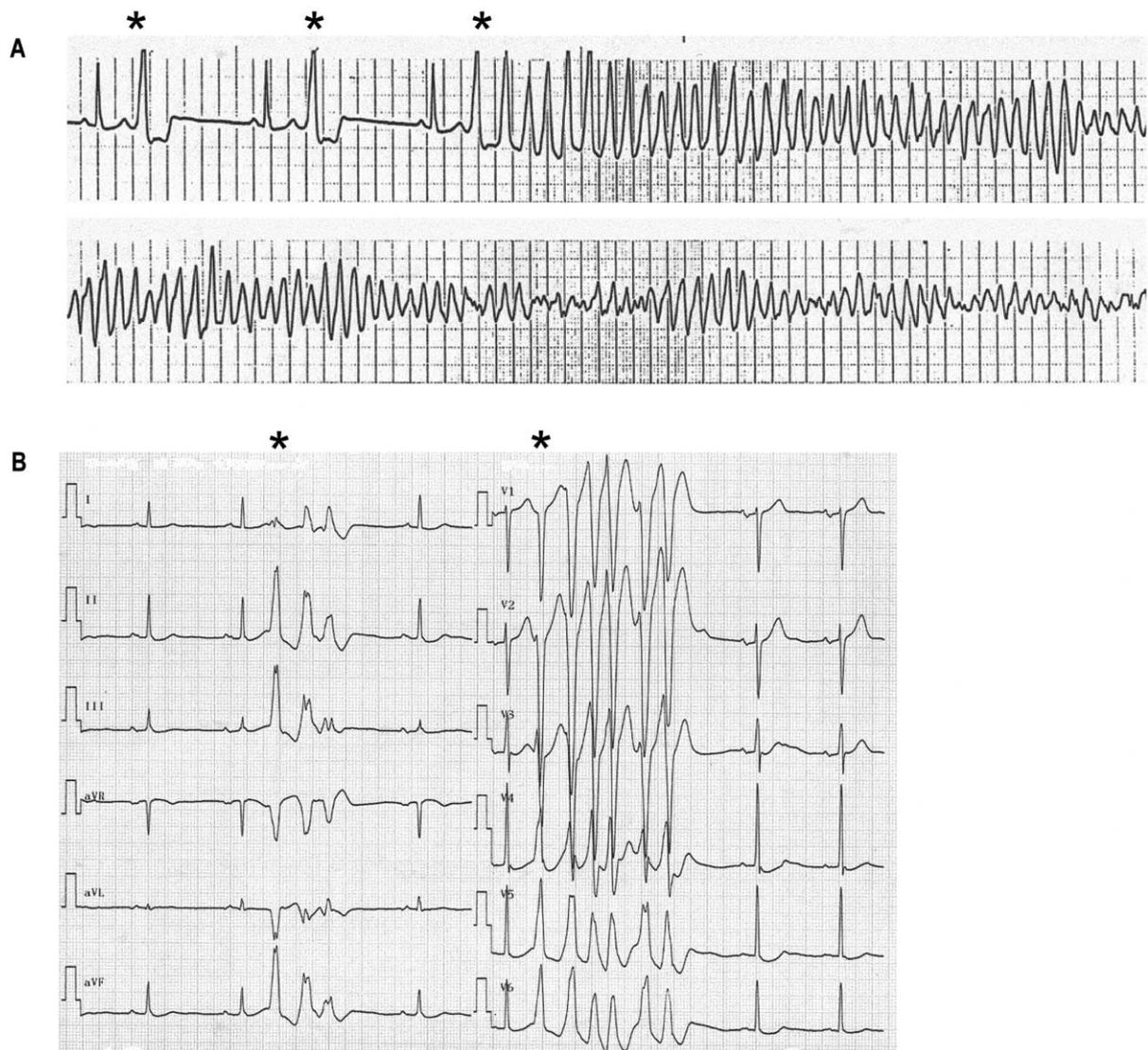
Figure 1. **(A)** Initiation of ventricular fibrillation (VF) recorded by a monitoring electrocardiogram in Patient #1. Note that the morphology of QRS complex of the initiating ventricular extrasystole (VE) was identical to that of preceding isolated premature ventricular contractions (*). **(B)** Non-sustained polymorphic ventricular tachycardia recorded in 12-lead electrocardiogram during hospitalization in Patient #8. The initiating VE showed left bundle branch morphology with inferior axis (*).

Table 1. Clinical Characteristics of the 16 Patients With the VF/PVT group

					Holter ECG Findings			
Patient No.	Age (yrs)	Gender	Spontaneous VF	Symptom	Isolated PVC (/day)	QT (ms)	CI (ms)	CL of PVT (ms)
1	43	F	+	Syncope	7,344	420	460	224
2	52	M	+	Syncope	26,828	420	410	240
3	41	F	+	Syncope	19,615	410	400	233
4	28	F	+	Syncope	7,518	430	440	216
5	48	F	+	Syncope	13,724	420	400	200
6	26	M	−	Syncope	NA	400	480	264
7	25	M	−	Pre-syncope	NA	420	580	310
8	49	M	−	Pre-syncope	41,192	400	390	273
9	43	M	−	Pre-syncope	2,479	380	360	248
10	41	M	−	Pre-syncope	13,819	360	320	242
11	38	F	−	Syncope	13,754	400	430	240
12	54	M	−	Syncope	9,700	380	360	220
13	41	F	−	Pre-syncope	12,681	390	360	268
14	25	F	−	Syncope	23,588	430	410	280
15	31	F	−	Syncope	38,061	380	350	227
16	34	F	−	Syncope	15,456	410	390	240
Mean ± SD	39 ± 10				17,554 ± 11,338	403 ± 21	409 ± 62	245 ± 28

Note that Patient #8 had a positive familial history of sudden cardiac death.

CI = coupling interval of the ventricular extrasystole; CL = cycle length; ECG = electrocardiogram; NA = not available; PVC = premature ventricular contraction; PVT = polymorphic ventricular tachycardia; QT = QT interval; VF = ventricular fibrillation; + = present; − = absent.

Electrophysiologic study and RFCA. As described previously (15), each patient underwent an electrophysiologic study (EPS) and RFCA in the fasting and non-sedated state after written informed consent was obtained. All drugs, including beta-blockers, were discontinued for at least five half-lives of each drug before the EPS. The 12-lead ECG of target VE that initiated spontaneous VF/PVT in the clinical course was also recorded in the EPS room before starting EPS and RFCA. If the target VE was not recorded under baseline conditions, injection of isoproterenol, epinephrine, or methoxamine with or without programmed ventricular stimulation was used to facilitate the induction of the initiating VE. We determined the provocated VE as target when the QRS morphology of the provocated VE was same as that of initiating VE recorded during hospitalization. Simultaneous 12-lead ECG and multiple intracardiac bipolar electrograms filtered at 30 to 500 Hz were recorded by a computerized electrophysiologic recording system (Bard Labsystem, CR Bard Inc., Billerica, Massachusetts) during EPS and RFCA. Stimuli were twice the diastolic threshold and 2 ms in duration. A deflectable 8-F quadripolar electrode catheter with conventional 4-mm distal electrode (EP Technologies, Sunnyvale, California) was used for mapping and RFCA with or without guidance by multielectrode basket catheter (Constellation, EP Technologies). Rapid burst pacing at multiple paced cycle lengths (pacing rate up to 250 beats/min) from right ventricular apex and the RVOT were performed in seven patients of the VF/PVT group before RFCA.

We performed RFCA by targeting the initiating VE. The optimal ablation site was determined by two methods: 1) endocardial activation mapping by identifying the site of the earliest activation during the target VE, and 2) pace mapping by comparing the 12-lead QRS morphology be-

tween the target VE and the paced beat during sinus rhythm. Radiofrequency energy was applied at the optimal site using a temperature control system with a target temperature set point of 60°C for 60 s. If the target VE was eliminated by energy delivery, three or four bonus applications were usually delivered around the most effective ablation site except in one patient. We tried to induce the target VE with the same interventions that provocated the target VE at the beginning of the EPS. If the premature ventricular contractions (PVCs) and/or ventricular tachycardia including the target VE were completely eliminated and were not induced at all, the RFCA was defined as successful. Partially successful ablation was defined when the target VEs were completely eliminated, but the other PVCs were induced and were not completely eliminated as a result. Failed ablation was defined when the target VEs were not eliminated completely.

Programmed electric stimulation was performed by up to triple extrastimuli mainly to confirm the effectiveness of RFCA as well as to induce VF in all 16 patients in the VF/PVT group after RFCA. We stopped extrastimuli at a coupling interval of 180 ms to avoid inducing non-specific VF.

Statistical analysis. Continuous variables were expressed as the group mean value ± SD and compared using unpaired t test. Qualitative variables were compared using Fisher exact test. A value of $p < 0.05$ was regarded as significant.

RESULTS

Clinical characteristics. Table 1 shows the clinical characteristics of the 16 patients with the VF/PVT group. Spontaneous episodes of VF were documented at rest during daytime in two patients and during nighttime in

Table 2. Comparison of the Clinical Parameters Between the VF/PVT Group and the RVOT-VT Group

	VF/PVT Group (n = 16)	RVOT-VT Group (n = 85)	p Value
Male	7/16 (44%)	25/85 (29%)	0.26
Age (yrs)	39 ± 10	43 ± 14	0.19
FH	1/16	1/85	0.29
Duration from onset of symptom to RFCA (months)	80 ± 103	69 ± 79	0.71
History of syncope	11/16 (69%)	15/85 (18%)	0.0001
Holter ECG findings			
Isolated PVC (/day)	17,554 ± 11,338	15,506 ± 16,053	0.58
CI of VE (ms)	409 ± 62	428 ± 65	0.27
QRS duration of VE (ms)	148 ± 8	142 ± 12	0.03
CL of VT (ms)	245 ± 28	328 ± 65	<0.0001

FH = family history of sudden death; RFCA = radiofrequency catheter ablation; RVOT = right ventricular outflow tract; VE = ventricular extrasystole; VT = ventricular tachycardia; other abbreviations as in Table 1.

three patients. Only one (Patient #8) of the 16 patients had a familial history of sudden cardiac death. Eleven of the 16 patients had prior episodes of syncope, and the remaining five patients had pre-syncope. Figure 1A shows the initiation of VF recorded by the monitoring ECG in Patient #1. The QT interval preceding VF was normal, and the coupling interval of the initiating VE was 460 ms. It is noteworthy that the morphology of QRS complex of the initiating VE was identical to that of the preceding isolated PVCs. In all patients, the corrected QT intervals preceding spontaneous VF/PVT were <440 ms. Holter recordings showed frequent isolated PVCs with the same QRS morphology as that of the initiating VE, and non-sustained PVT with short cycle length (mean of 245 ± 28 ms) in all 16 patients (Table 1). The coupling interval of VE was uniform in each patient and was not so short (mean of 409 ± 62 ms). Table 2 represents the comparison of the clinical parameters between the VF/PVT group and the RVOT-VT group. No significant difference was observed regarding gender, age, familial history of sudden death, and duration from onset of symptom to RFCA. However, prior episodes of syncope were more frequent in the VF/PVT group than in the RVOT-VT group (69% vs. 18%, p = 0.0001). In the Holter recordings, the frequency of isolated PVCs and the coupling intervals of the initiating VE were not different between the VF/PVT group and the RVOT-VT group. However, the cycle length of spontaneous non-sustained ventricular tachycardia was much shorter in the VF/PVT group than in the RVOT-VT group (245 ± 28 ms vs. 328 ± 65 ms, p < 0.0001).

Among the 16 patients in the VF/PVT group, 11 patients showed pre-syncope or syncope as a first symptom; however, the remaining 5 patients had only palpitation due to PVCs or monomorphic VT as a first symptom. Among five patients with spontaneous VF, three showed syncope as a first symptom, whereas two had only palpitation as a first symptom.

Electrophysiologic findings. Table 3 shows the electrophysiologic characteristics and RFCA parameters of the 16 patients in the VF/PVT group. The target VE occurred spontaneously in 11 patients and was induced by bolus injection of isoproterenol (1 μg) in three patients, epinephrine (5 μg) in one patient, and methoxamine (1 mg) in one patient. Endocardial mapping during sinus rhythm showed no abnormal electrograms, including fragmentations or delayed potentials, in any patients. His-ventricle intervals were <55 ms (mean of 42 ± 6 ms) in all patients. Figure 2 shows the polymorphic changes of the QRS complex during rapid pacing (pacing rate = 250 bpm) in Patient #3. These polymorphic morphologic changes were observed by the rapid pacing from origin of target VE in two patients (Patients #3 and #5) out of seven patients examined.

RFCA. Radiofrequency catheter ablation was performed at the site where the endocardial activation time during target VE was the earliest and the best pace mapping was obtained. Figure 3 represents the target VE (Fig. 3A), the pace mapping at the ablation site (Fig. 3B), simultaneous recording of surface ECG and endocardial electrograms during the target VE (Fig. 3C), and catheter position of the RFCA site (Fig. 3D) in Patient #2. The pace mapping demonstrated close concordance with the QRS morphology of the target VE in all leads. The bipolar endocardial electrogram of the mapping catheter, located in the septum of the RVOT, preceded the surface QRS onset of the target VE by 10 ms. The mean bipolar local activation time at the successful RFCA site was 17 ± 11 ms before the surface QRS onset (Table 3). The origin of the target VE, where the target VE disappeared or changed to the other VE with different QRS morphology by a single energy delivery, was in the septum of the RVOT in 13 patients and in the lateral freewall of the RVOT in three patients. After RFCA for initial target VE, the other VE with different QRS morphology appeared in 11 patients in whom multiple applications by mean of 9 ± 4 were added. Therefore, relatively large areas, approximately 2 to 4 cm in diameter, were presumably ablated in the 11 patients. Finally, RFCA was successful in 13 patients and partially successful in three patients by a mean of 9 ± 4 radiofrequency applications. Programmed electric stimulation after RFCA revealed that VF was induced by triple extrastimuli from the RVOT in only one patient (Patient #2), and non-sustained PVT was

Table 3. Electrophysiologic Characteristics and RFCA Parameters of the 16 Patients With the VF/PVT Group

Patient No.	Induction of Target VE	Origin of Target VE	EAT (ms)	ERP (ms)	Morphologic Change	No. of RF	Outcome	Induction of VF/PVT After RFCA	Advanced Treatment
1	Spontaneous	Sep	−10	220	−	1	Succ	−	−
2	Spontaneous	Sep	−10	230	−	5	Succ	VF (500/240/200/200)	ICD
3	Spontaneous	Sep	−20	250	−	8	Succ	−	−
4	Spontaneous	Sep	−50	230	+	11	Succ	−	−
5	Spontaneous	Sep	−8	210	+	15	Partial	PVT (500/240/220/190)	Beta-blocker
6	ISP	Sep	−20	210	+	12	Partial	−	Beta-blocker
7	Epi	Free	−18	200	+	4	Succ	−	−
8	Spontaneous	Sep	−12	210	+	10	Partial	−	Beta-blocker
9	Spontaneous	Free	−20	200	−	5	Succ	−	−
10	ISP	Free	−22	220	+	14	Succ	−	−
11	Spontaneous	Sep	−5	200	+	5	Succ	−	−
12	Me	Sep	−8	220	+	9	Succ	−	−
13	Spontaneous	Sep	−14	200	+	7	Succ	−	−
14	Spontaneous	Sep	−6	210	−	15	Succ	−	Beta-blocker
15	Spontaneous	Sep	−24	210	+	7	Succ	PVT (500/240/220/180)	−
16	Spontaneous	Sep	−26	240	+	8	Succ	−	−
Mean ± SD			−17 ± 11	216 ± 15		9 ± 4			

EAT = endocardial activation time (relative to QRS); Epi = epinephrine; ERP = effective refractory period; Free = freewall; ISP = isoproterenol; Me = methoxamine; Partial = partially successful ablation; PVT = polymorphic ventricular tachycardia; RF = radiofrequency applications; RFCA = radiofrequency catheter ablation; Sep = septum; Succ = successful ablation; VE = ventricular extrasystole; VF = ventricular fibrillation; + = present; − = absent.
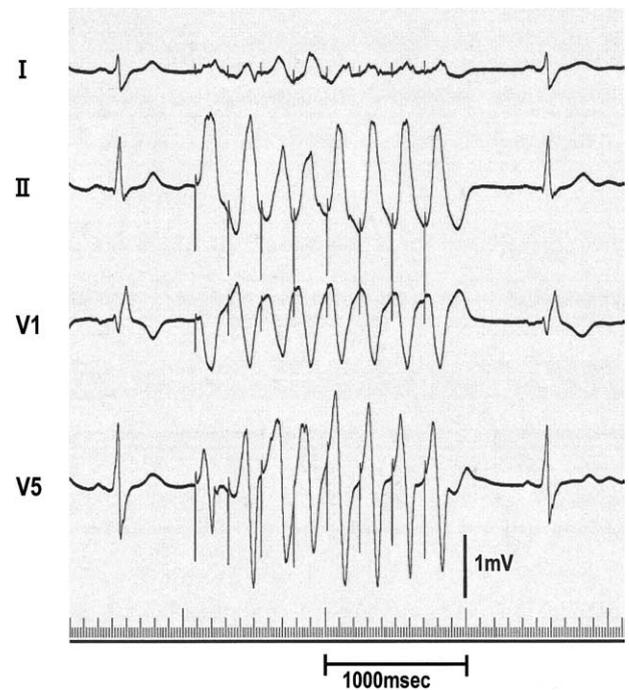
Figure 2. Polymorphic changes of the QRS complex on surface electrocardiogram leads I, II, V_1, and V_5 during rapid pacing in Patient #3. The morphologic changes were induced by rapid pacing from the origin of the target ventricular extrasystole.

induced in two patients (Patients #5 and #15) among the 16 patients.

Follow-up. One patient (patient #2) received an implantable cardioverter-defibrillator (ICD) because of induction of VF. Four patients received oral beta-blockers (3 for partially successful ablation and 1 for hypertension) as a therapeutic option. The remaining 11 patients were free from any advanced treatment, including antiarrhythmic drugs. There were no recurrences of episodes of syncope, VF, or cardiac arrest in any patients during follow-up of 54 ± 39 months.

DISCUSSION

Malignant entity of VF/PVT. Idiopathic ventricular tachycardias originating from the RVOT in patients without structural heart disease are considered benign, and RFCA has become an effective therapeutic option for these arrhythmias (8–12). However, a recent report (6) has shown that the malignant idiopathic VF may occasionally originate from the right ventricular outflow tract, the same site of origin of the "benign" RVOT. Moreover, several types of VF/PVT in patients without apparent heart disease were reported (14,16–23). We reported a malignant entity of VF/PVT initiated by the VE originated from the RVOT without structural heart disease in this study. In all patients, 12-lead ECG showed normal QT intervals at rest or just before and after episodes of VF and/or PVT (16–18). Neither ST-segment elevation nor right bundle branch block, most likely seen in Brugada syndrome, were recorded (14,19). The SAECG showed no late potential by which arrhythmogenic right ventricular cardiomyopathy/dysplasia
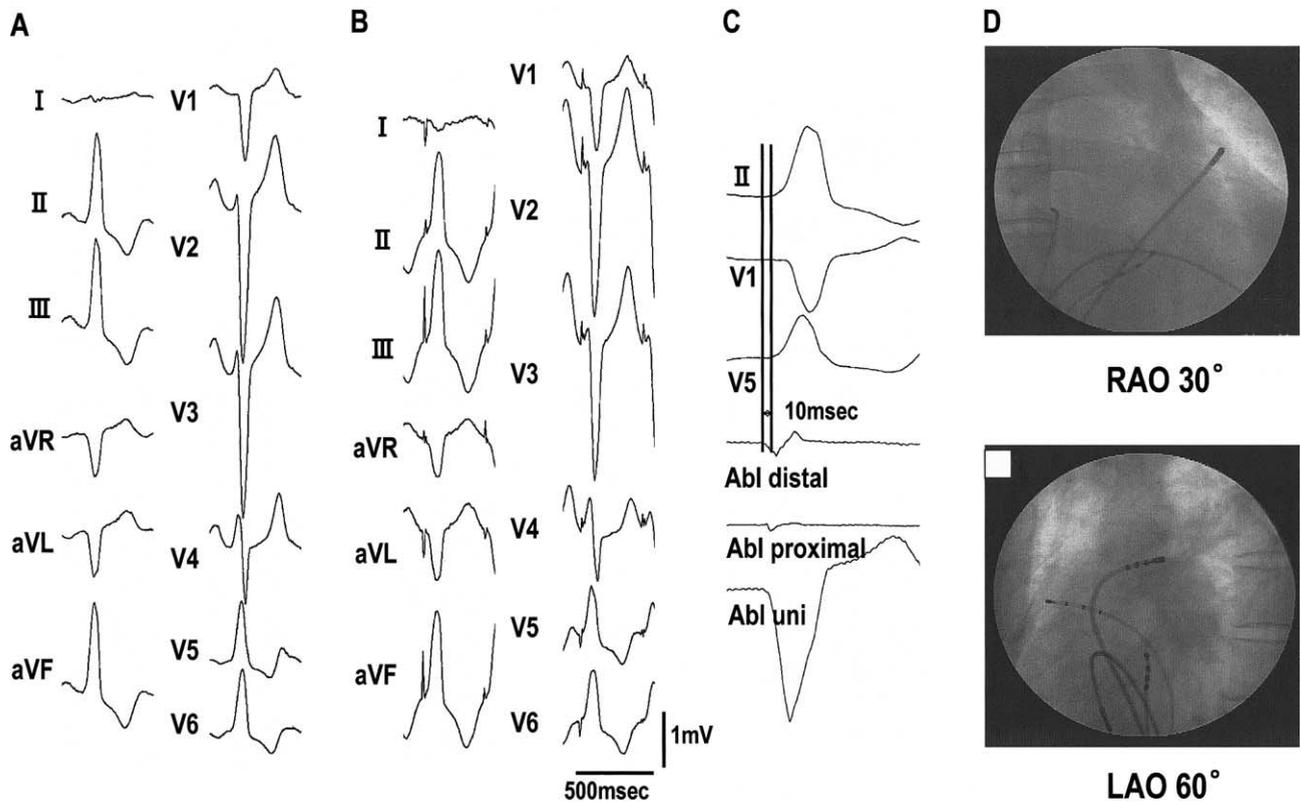
Figure 3. **(A)** Morphology of target ventricular extrasystole (VE) on 12-lead electrocardiogram (ECG); **(B)** Pace mapping at the ablation site; **(C)** Simultaneous recordings of surface ECG and endocardial electrograms during the target VE; **(D)** Catheter position of radiofrequency catheter ablation site in Patient #2. The pace mapping demonstrated close concordance with the QRS morphology of the target VE in all leads. The bipolar endocardial electrogram of the mapping catheter, which was located in the septum of the right ventricular outflow tract, preceded the surface QRS onset of the target VE by 10 ms. Abl = ablation catheter electrogram; LAO = left anterior oblique; RAO = right anterior oblique; uni = unipolar electrogram.

was characterized (20). The coupling intervals were not as short as those described in patients with short-coupled torsade de pointes (21) or idiopathic VF (22,23). Therefore, there are some differences in this entity compared with the prior types of idiopathic VF and/or PVT. We considered this entity to be a variant form of "benign" RVOT-VTs, but to have some difference in the clinical characteristics. The present data suggest that the 16 patients in the VF/PVT group showed prior episodes of syncope more frequently than the 85 patients in the RVOT-VT group, indicating the need of discrete follow-up in patients with prior episodes of syncope. On the other hand, 5 of the 16 patients in the VF/PVT group and two of the five patients with spontaneous VF had only palpitation as a first symptom. This finding suggests that some of patients initially diagnosed as "benign" RVOT-VT may become the patients with malignant entity of idiopathic VF/PVT, indicating that the need of careful follow up is required even in patients with benign RVOT-VT.

Possible mechanism of VF/PVT. It has been suggested that mechanism of idiopathic monomorphic VT arising from the RVOT is triggered activity (24,25). In this study, Holter recordings showed frequent isolated PVCs with the same morphology as that of the initiating VE. In all patients, the SAECG showed no late potential, and endo-

cardial mapping represented no local abnormal electrograms, including fragmentations or delayed potentials. Programmed electrical stimulations induced VF in only one patient and non-sustained PVT in two patients among the 16 patients. In addition, rapid pacing from origin of target VE made the polymorphic morphologic changes in the QRS configuration in two out of seven patients, although the possibility that some beats were induced but are not captured by pacing could not be excluded completely. We speculate that the functional block and/or delayed conduction by rapid firing due to triggered activity or microreentry arising from a single focus led to chaotic ventricular conduction, so-called fibrillatory conduction, causing VF and/or PVT without organic delayed conduction zone. However, it is reasonable to say that rapid firing from close multiple foci one after another produces polymorphic morphologic changes in the QRS configuration in some cases. This is based on the observation that the other VE with different QRS morphology appeared after eliminating the initial target VE by RFCA in 11 patients. Although the initiating VE are likely to be generated from triggered activities, different mechanisms cannot be excluded.

We performed RFCA for the initiating VE with three or four bonus applications delivered around the most effective site. Some case reports showed successful RFCA for PVT

initiated by VE originating from the RVOT with (26,27) or without (28) eliminating targeting VE. In the latter case report, RFCA was considered to alter or remodel arrhythmic substrates to maintain the PVT. Our RFCA targeting for the initiating VE with additional applications around the origin of initiating VE might eliminate both arrhythmogenic triggers and substrate for VF and/or PVT in this study.

Study limitations. First, the 16 patients showed spontaneous VF/PVT in our series, whereas the 85 patients had only monomorphic VT. This may give the impression that polymorphic RVOT-VT is present in 16% of patients with arrhythmias originating from the RVOT. However, this large percentage probably represents a referral bias, because patients with polymorphic RVOT-VT are more likely to be hospitalized and more likely to be referred for RFCA, whereas patients with monomorphic RVOT-VT are more likely to be treated conservatively as outpatients.

Second, VF was induced in only one patient after RFCA, whereas patients with idiopathic VF usually have high VF inducibility rates. The low rate of VF induction is probably associated with the result of our stimulation protocol.

Third, among the five patients with spontaneous VF, only one patient received ICD after RFCA. The ICD as therapeutic backup is particularly important for patients with spontaneous episodes of VF regardless of the success of RFCA. The ICD was not available in Japan when three of the five patients were admitted to our center and underwent RFCA. The remaining patient, a young woman, refused to receive an ICD after successful RFCA.

Reprint requests and correspondence: Dr. Wataru Shimizu, Division of Cardiology, Department of Internal Medicine, National Cardiovascular Center, 5-7-1 Fujishiro-dai, Suita, Osaka 565-8565, Japan. E-mail: wshimizu@hsp.ncvc.go.jp.

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