Journal of the American College of Cardiology
© 2005 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 45, No. 6, 2005
ISSN 0735-1097/05/$30.00
doi:10.1016/j.jacc.2004.12.025

Idiopathic Ventricular Arrhythmia Arising From the Mitral Annulus

A Distinct Subgroup of Idiopathic Ventricular Arrhythmias

Hiroshi Tada, MD,* Sachiko Ito, MD,* Shigeto Naito, MD,* Kenji Kurosaki, MD,* Shoichi Kubota, MD,§ Aiko Sugiyasu, MD,§ Taketsugu Tsuchiya, MD,† Kohei Miyaji, MD,* Minoru Yamada, MD,* Yasunori Kutsumi, MD,‡ Shigeru Oshima, MD,* Akihiko Nogami, MD,§ Koichi Taniguchi, MD, FACC*

Gunma, Kanazawa, Fukui, and Kanagawa, Japan

OBJECTIVES We sought to clarify the prevalence and characteristics of idiopathic ventricular tachycardia or premature ventricular contraction originating from the mitral annulus (MAVT/PVC).

BACKGROUND Recent case reports have presented patients with MAVT/PVC.

METHODS Electrocardiographic (ECG) characteristics and the results of electrophysiologic investigation and radiofrequency catheter ablation (RFCA) were analyzed in 352 patients with symptomatic idiopathic ventricular tachycardia (IVT)/premature ventricular contraction (PVC).

RESULTS Nineteen cases of IVT/PVC (5%) represented MAVT/PVC. Of these, 11 (58%) originated from the anterolateral portion of the mitral annulus (AL-MAVT/PVC), and 2 (11%) arose from the posterior portion (Pos-MAVT/PVC). The remaining six cases of MAVT/PVC (31%) had posteroseptal origin (PS-MAVT/PVC). In all patients, an S-wave was present in lead V_6. The QRS polarity in inferior leads and leads I and aVL was useful for differentiating AL-MAVT/PVC from Pos-MAVT/PVC or PS-MAVT/PVC. The Pos-MAVT/PVC had an Rs pattern in lead I and an R pattern in lead V_1, whereas PS-MAVT/PVC invariably had an R pattern in lead I and a negative QRS component in lead V_1. The AL-MAVT/PVC and Pos-MAVT/PVC showed a longer QRS duration than the PS-MAVT/PVC ($p < 0.001$), and all had late-phase "notching" of the QRS complex in inferior leads. In all patients, RFCA eliminated MAVT/PVC, with no recurrences during follow-up for 21 ± 15 months.

CONCLUSIONS Mitral annular VT/PVC is a rare but distinct subgroup of IVT/PVC. MAVT/PVC origin could be determined by ECG analysis. The AL and PS sites of the MA were preferential. (J Am Coll Cardiol 2005;45:877–86) © 2005 by the American College of Cardiology Foundation

Most idiopathic ventricular tachycardias (IVTs) or premature ventricular contractions (PVCs) have a right ventricular (RV) outflow tract or left ventricular (LV) inferoseptal origin (1,2), but some originate from the endocardium of the left ventricular outflow tract (LVOT) (3,4) or an LV epicardial site (5–7). Recently, small numbers of cases of IVT/PVC have been reported to originate from the anterolateral (AL) portion of the mitral annulus (MA) in close proximity to the mitral-aortic continuity (3,8–10). However, little is known about the prevalence, electrocardiographic (ECG) characteristics, or preferential site of tachycardia origin within the MA or about the efficacy of radiofrequency catheter ablation (RFCA) for IVT/PVC of this kind (MAVT/PVC). This study was undertaken to clarify these points.

METHODS

Study population. The study included 352 patients with symptomatic IVT/PVC who underwent RFCA (186 men and 166 women; mean age [±SD] 54 ± 16 years). Monomorphic ventricular tachycardia (VT), defined as three or more consecutive PVCs, was present in 122 patients, and the remaining 230 had monomorphic PVCs. All patients had a normal ECG during sinus rhythm, and no structural abnormalities were apparent by physical examination or echocardiography. Before RFCA, 12-lead ECGs were obtained at each clinic visit, and 24-h ambulatory Holter monitoring was carried out at least once. The ECG was monitored for 12 to 24 h during hospital admission, just before catheter ablation. During the clinical arrhythmia, the surface ECG showed single bundle branch block morphology in all patients. Ethical approval was obtained from the hospital's ethics committee, and all patients gave written informed consent before participation.

Mapping and RF ablation. After withdrawal of antiarrhythmic drugs, electrophysiologic evaluation and RFCA were performed as previously described (7). With fluoroscopic guidance, catheters were positioned high in the right atrium, at the RV apex, and in the RVOT and/or His bundle region. Programmed ventricular stimulation was performed from the RV apex and RVOT at two drive cycle lengths, with up to three extrastimuli. In addition, incremental burst pacing at a cycle length up to 250 ms was

From the *Division of Cardiology, Gunma Prefectural Cardiovascular Center, Maebashi, Gunma; †Division of Cardiology, Kanazawa Cardiovascular Hospital, Kanazawa; ‡Fukui Chuo Clinic, Fukui; and the §Division of Cardiology, Yokohama Rosai Hospital, Yokohama, Kanagawa, Japan.

Manuscript received July 15, 2004; revised manuscript received October 20, 2004, accepted December 6, 2004.

Abbreviations and Acronyms

AL = anterolateral
IVT = idiopathic ventricular tachycardia
LV = left ventricle/ventricular
MA = mitral annulus/annular
PVC = premature ventricular contractions
OT = outflow tract
Pos = posterior
PS = posteroseptal
PVC = premature ventricular contraction
RFCA = radiofrequency catheter ablation
RV = right ventricle/ventricular
VT = ventricular tachycardia

performed. If the clinical arrhythmia did not occur spontaneously and was not induced in the baseline state, intravenous isoproterenol (0.5 to 2.0 μg/min) was administered to induce arrhythmia. During an episode of clinical arrhythmia, activation mapping was performed. A 7-F quadripolar catheter with a 4-mm distal electrode, an embedded thermistor, interelectrode spacing of 2-5-2 mm, and a deflectable tip (Biosense Webster, Diamond Bar, California, and EP Technologies, San Jose, California) was used for mapping and ablation. Radiofrequency energy was applied at the site where the earliest ventricular activation was recorded. In 20 patients in whom clinical arrhythmia could not be induced, catheter ablation was performed at the site where perfect or near-perfect pace mapping was obtained. Radiofrequency energy was delivered using maximum power of 35 to 50 W and a maximum electrode-tissue interface temperature of 55°C to 60°C (7).

Definition of successful catheter ablation. Successful catheter ablation met three criteria: 1) absence of spontaneous or induced clinical IVT/PVC, both in the absence and presence of isoproterenol, at the end of the procedure; 2) absence of clinical arrhythmia over 48 h of ECG monitoring in the absence of anti-arrhythmic drugs; and 3) no recurrence of symptomatic arrhythmia in the absence of anti-arrhythmic drug therapy during at least three months of follow-up.

Definition of MA location for origin of tachycardia. Tachycardias were considered to arise from the MA based on three criteria. First, the catheter tip demonstrated the characteristic MA location and motion when viewed in the right and left anterior oblique fluoroscopic views at successful RFCA. Second, the ratio of atrial to ventricular electrograms at the ablation site was <1, and the amplitudes of the atrial and ventricular electrograms were >0.08 and 0.5 mV at the ablation site, respectively. Third, successful elimination of tachycardia and ectopy was achieved by RF energy delivery at this site.

Postablation follow-up and definition of recurrence of IVT. After the ablation procedure, patients underwent follow-up at our cardiology clinic two weeks later, and then every one to two months. To rule out recurrence of IVT/PVC or appearance of another arrhythmia, a 12-lead ECG was recorded on each visit to the clinic, and 24-h Holter monitoring was performed at least once during the follow-up period. Recurrence of IVT/PVC was defined as a recurrence of symptoms suggestive of tachycardia, as well as an episode of IVT/PVC where morphology of the QRS complex was nearly identical to that before the ablation procedure, as documented by 12-lead ECG or 24-h Holter monitoring during the follow-up period.

Pace mapping study. A pace mapping study also was performed in five control subjects (three women and two men; mean age 67 ± 7 years) after successful ablation of their original tachycardia to determine the ECG characteristics of MAVT/PVC. Three patients had atrioventricular orthodromic tachycardia caused by a concealed accessory pathway, whereas the remaining two had atrioventricular nodal re-entrant tachycardia. None had structural heart disease. Pace mapping was performed after ablation from the AL, posterior (Pos), and posteroseptal (PS) aspects of the MA. A 7-F quadripolar catheter with a 4-mm distal electrode (Biosense Webster) was used. A single electrical stimulus was delivered from the AL, Pos, and PS-MA during end diastole in a bipolar fashion at an output just greater than the diastolic threshold from the distal electrode pair (with the distal electrode as the cathode). The catheter sites were confirmed by multiplanar fluoroscopy, and pacing was performed from the three sites in each patient.

Analysis of MAVT/PVC waveforms on 12-lead ECG or during pace mapping. In patients with MAVT/PVC, ECGs recorded at a paper speed of 25 mm/s during the clinical arrhythmia were analyzed. During the first beat of VT or the PVC, analysis of the surface ECG focused on the following characteristics: presence or absence of an S-wave in lead V_6; total QRS duration; morphology and/or magnitude of the R-wave or QS complex of the QRS complex in leads I, II, III, aVR, aVL, and aVF; and finally, the site of R-wave transition in the precordial leads. Capital letters (Q, R, S) are used to refer to waves of relatively high amplitude (>5 mm), whereas lowercase letters (q, r, s) refer to waves of relatively low amplitude (≤5 mm) (11).

Statistical analysis. Continuous variables, expressed as the mean value ± SD, were compared using the Student *t* test. A p value <0.05 was considered to indicate statistical significance.

RESULTS

Prevalence and clinical characteristics of MAVT/PVC. In 352 patients treated by RFCA, 278 IVT/PVCs (79%) were ablated successfully, but the remaining 74 IVT/PVCs (21%) represented ablation failure (Table 1). Of the 352 patients who underwent RFCA, 19 (5%) IVT/PVCs showed earliest ventricular activation when IVT/PVC was recorded at the MA and/or perfect pace mapping was obtained at that site (10 men and 9 women; mean age 61 ± 12 years) (Tables 1 and 2). Before the ablation procedure, 6 patients had monomorphic VT, whereas the

Table 1. Tachycardia Origin and Results of Radiofrequency Catheter Ablation for Idiopathic Ventricular Arrhythmias

Arrhythmia Origin	No. (%)	VT	PVC	Success (%)
Mitral annulus	19 (5)	6	13	19 (100)
RVOT	176 (50)	41	135	161 (91)
PA	3 (1)	1	2	3 (100)
RV septum or near His bundle region	26 (7)	7	19	7 (27)
Tricuspid annulus	7 (2)	4	3	5 (71)
Aortic sinus of Valsalva or LV epicardium	73 (21)	37	36	44 (60)
LVOT	19 (5)	4	15	12 (63)
LV inferoseptum	25 (7)	20	5	24 (96)
Others (LV posterolateral, 2; LV mid-septum, 2)	4 (1)	2	2	3 (75)
Total	352 (100)	122	230	278 (79)

The site of the tachycardia origin was defined as the site where the earliest ventricular activation was recorded and/or perfect pace mapping was obtained. Idiopathic ventricular arrhythmias that could not be ablated with radiofrequency ablation from the left sinus of Valsalva (LSV), despite the earliest ventricular activation being recorded in the LSV, were classified as originating from the left ventricular (LV) epicardium in the present study (7).

LVOT or RVOT = endocardium of the left or right ventricular outflow tract, respectively; PA = pulmonary artery; PVC = premature ventricular contraction; VT = ventricular tachycardia.

remaining 13 had monomorphic PVCs. Eleven MAVT/PVCs (58%) originated from the AL portion of the MA in close proximity to the left fibrous trigone and mitral-aortic continuity (AL-MAVT/PVC); two (11%) from the posterior portion of the MA (Pos-MAVT/PVC); and the remaining six (31%) from the PS-MA (PS-MAVT/PVC) (Table 3, Fig. 1).

Electrophysiologic characteristics and results of catheter ablation in MAVT/PVC. The arrhythmia occurred spontaneously in 11 patients, and with isoproterenol administration in two patients. In another two patients, VT could be reproducibly induced and/or terminated by programmed ventricular stimulation. One was induced by single RV extrastimulus and lasted for ≥30 s (Patient #10) (Table 2). It was terminated by RV pacing. A presystolic potential preceding the QRS complex was recorded during VT. The other was nonsustained VT, which was induced by RV pacing (Patient #11). In those two cases, verapamil was effective for both VTs (verapamil-sensitive). In the remaining four patients, pace mapping was performed because the clinical arrhythmia could not be induced during the procedure (Table 2). Complete elimination of MAVT/PVC could be achieved by RF energy deliveries at the site where the earliest ventricular activation was recorded (n = 15) or the site where the pace map 12-lead ECG matched the recording obtained during MAVT/PVC (≥11/12-lead concordance of major and minor deflections; n = 4). Magnitudes of the local atrial and ventricular electrogram at the successful ablation site during sinus rhythm were 0.19 ± 0.12 mV and 1.65 ± 0.8 mV, respectively. In 6 (40%) of 15 patients in whom MAVT/PVC was recorded during the ablation procedure, a discrete potential preceding the QRS complex was recorded at successful ablation sites (Table 2; Figs. 2 and 3). In these 15 patients, the local ventricular activation time recorded at the site of successful ablation preceding the onset of the QRS complex was 29 ± 17 ms (Table 2). All patients were discharged without need for medications and have done well with no recurrence of tachycardia during a follow-up period of 21 ± 15 months. No complications occurred during the ablation procedure or during the follow-up period.

Electrocardiographic characteristics of MAVT/PVC. In all patients, QRS complexes during MAVT/PVC showed a right bundle branch block pattern, and an S-wave was present in lead V_6 (Table 3, Fig. 1). The duration of the QRS complex of MAVT/PVC was 154 ± 20 ms (range 120 to 190 ms), and the mean coupling interval was 474 ± 73 ms (range 320 to 600 ms). Precordial R-wave transition usually occurred by lead V_1, but in three cases of PS-MAVT/PVC, it occurred between leads V_1 and V_2. No MAVT/PVC showed an R-wave transition beyond lead V_2. All MAVT/PVCs demonstrated a monophasic R or Rs pattern in leads V_2 to V_6, and an R(r)-wave was always present in lead V_6 (Table 3, Fig. 1).

In AL-MAVT/PVC, polarity of the QRS complex was positive in all inferior leads and negative in leads I and aVL. On the other hand, in Pos- or PS-MAVT/PVC, polarity of the QRS complex was negative in all inferior leads and positive in leads I and aVL (Table 3, Fig. 1).

In Pos-MAVT/PVC, an Rs pattern in lead I and an R pattern in lead V_1 were recorded. However, PS-MAVT/PVC always showed a monophasic R pattern in lead I and a negative component of the QRS complex (qR, qr, rS, rs, or QS pattern) in lead V_1 (Table 3, Fig. 1). The Q-wave amplitude ratio of lead III to lead II was greater in PS-MAVT/PVC (2.3 ± 0.6) than in Pos-MAVT/PVC (1.5 ± 0.3; $p = 0.15$).

Both AL-MAVT/PVC and Pos-MAVT/PVC, which originated from the LV free wall, showed a significantly longer QRS duration (164 ± 14 ms) than PS-MAVT/PVC (131 ± 9 ms; $p < 0.001$) (Fig. 4A), all exhibiting "notching" of the late phase of the R wave for AL-MAVT/PVC or the Q-wave for Pos-MAVT/PVC in all inferior leads (Table 3, Figs. 2 and 3). However, no notching of the Q-wave was

Table 2. Clinical and Electrophysiologic Characteristics of Patients With Idiopathic Mitral Annular Ventricular Tachycardia or Premature Ventricular Contractions

Pt. #	Age (yrs)	Gender	VT/PVC	Symptoms	Duration of Symptom (yrs)	Failed Anti-Arrhythmic Drugs Before Ablation	VE Beats (No./24 h)	LVEF (%)	Ablation Guided by	EAT (ms)	Pre-P
1	57	M	PVC	P	2.0	Cibenzoline, beta-blocker	39,661	53	EAT	−34	+
2	26	M	PVC	FG, P	0.5	Pilsicainide, beta-blocker	29,339	68	EAT	−29	+
3	66	F	NSVT	D, P	0.5	Lidocaine, verapamil	NA	48	EAT	−25	+
4	65	F	PVC	P	0.3	Beta-blocker	8,240	64	EAT	−37	+
5	65	M	PVC	P, S	0.5	Beta-blocker	11,636	67	EAT	−14	−
6	61	F	PVC	P	1.5	Propafenone, beta-blocker	13,343	65	EAT	−7	−
7	71	F	PVC	P	10.0	Disopyramide	3,839	73	Pace map	NA	NA
8	66	M	NSVT	P, S	5.0	Mexiletine	17,098	50	Pace map	NA	NA
9	73	M	PVC	P	5.0	Mexiletine	27,976	59	EAT	−25	−
10	43	M	SVT	S	1.0	Mexiletine, amiodarone	NA	50	EAT	−70	+
11	73	M	NSVT	P	0.5	Lidocaine, nifekalant	10,979	53	EAT	−16	−
12	50	M	PVC	P	1.4	Mexiletine	37,643	47	EAT	−60	+
13	69	F	PVC	P	1.0	Mexiletine, beta-blocker	5,036	74	Pace map	NA	NA
14	65	F	PVC	P	0.3	Beta-blocker	16,109	65	EAT	−21	−
15	54	M	NSVT	CP, P, PS	0.5	Mexiletine, amiodarone, beta-blocker	29,369	63	EAT	−20	−
16	70	F	NSVT	D	8.8	Procainamide, lidocaine, mexiletine	20,605	40	EAT	−19	−
17	46	M	PVC	FG, P	6.0	Pilsicainide, cibenzoline, mexiletine, beta-blocker	5,499	73	Pace map	NA	NA
18	73	F	PVC	P	0.3	Cibenzoline, disopyramide, beta-blocker	8,237	66	EAT	−27	−
19	62	F	PVC	CP, P	1.5	Cibenzoline, mexiletine	8,937	55	EAT	−28	−
Mean ± SD	61 ± 12				2.5 ± 3.0		17,267 ± 11,520	60 ± 10		−29 ± 17	

CP = chest pain; D = dyspnea; EAT = earliest ventricular activation time during arrhythmia; FG = fatigue; LVEF = left ventricular ejection fraction; NA = not available; P = palpitation; Pre-P = presystolic potential preceding QRS complex during arrhythmia; PS = presyncope; PVC = premature ventricular contraction; S = syncope; (N)SVT = (non-)sustained ventricular tachycardia; VE = ventricular ectopic.

Table 3. Electrocardiographic Characteristics in Patients With Idiopathic Ventricular Tachycardia or Premature Ventricular Contractions Originating From the Mitral Annulus

Pt. #	VT/PVC Origin	QRS Morphology								Transition Zone in the Precordial Leads	Late "Notching" in the Inferior Leads
		I	II	III	aVR	aVL	aVF	V_1	V_6		
1	AL	qs	R	R	QS	QS	R	R	Rs	$<V_1$	+
2	AL	rS	Rs	qR	qR	rS	R	R	RS	$<V_1$	+
3	AL	qs	R	R	qs	QS	R	R	rS	$<V_1$	+
4	AL	QS	R	R	QS	QS	R	R	Rs	$<V_1$	+
5	AL	rS	R	R	QS	QS	R	R	Rs	$<V_1$	+
6	AL	RS	R	qR	QS	rS	qR	qR	Rs	$<V_1$	+
7	AL	rs	R	R	QS	QS	R	R	Rs	$<V_1$	+
8	AL	rS	R	R	qr	QS	R	R	rs	$<V_1$	+
9	AL	QS	R	R	QS	QS	R	R	Rs	$<V_1$	+
10	AL	rS	R	R	QS	QS	R	R	Rs	$<V_1$	+
11	AL	rs	R	R	qs	qs	R	R	Rs	$<V_1$	+
12	Pos	Rs	QS	QS	qs	R	QS	R	RS	$<V_1$	+
13	Pos	Rs	rS	rS	Qr	R	rS	R	Rs	$<V_1$	+
14	PS	R	rS	QS	QS	R	QS	qR	Rs	$<V_1$	−
15	PS	R	rS	QS	Qr	R	QS	qR	Rs	$<V_1$	−
16	PS	R	QS	QS	qR	R	QS	qr	Rs	$<V_1$	−
17	PS	R	rS	QS	QS	R	QS	rS	Rs	$V_1<<V_2$	−
18	PS	R	QS	QS	qr	R	QS	rs	RS	$V_1<<V_2$	−
19	PS	R	rS	QS	qr	R	rS	QS	Rs	$V_1<<V_2$	−

Capital letters (Q, R, S) refer to relatively high-amplitude waves (>5 mm) (11). Conversely, lowercase letters (q, r, s) refer to relatively low-amplitude waves (≤5 mm) (11). Late "notching" indicates a QRS complex showing a notching in the late phase of the QRS complex.

AL = anterolateral; Pos = posterior; PS = posteroseptum; PVC = premature ventricular contraction; VT = ventricular tachycardia.

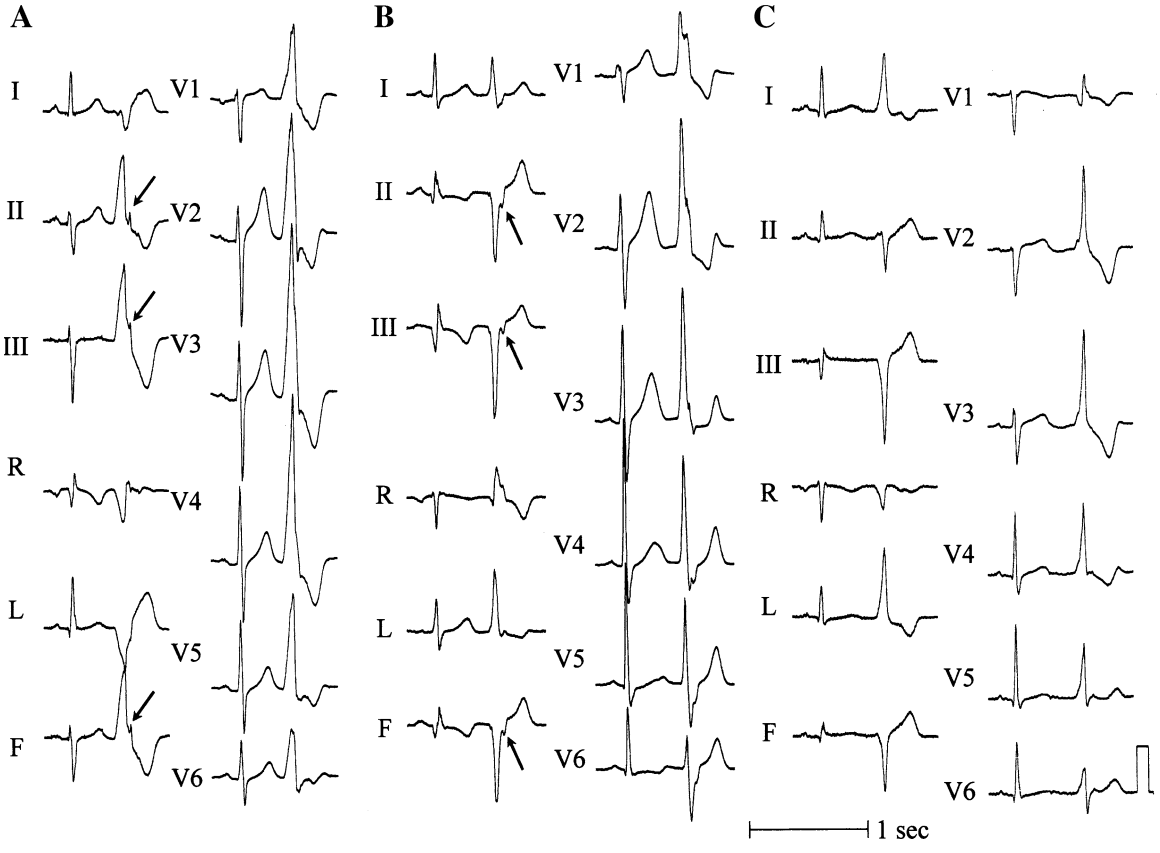

Figure 1. Representative 12-lead electrocardiograms of premature ventricular contractions originating from the anterolateral **(A)**, posterior **(B)**, and posteroseptal **(C)** portions of the mitral annulus. **Arrows** indicate "notching" of the late phase of the QRS complex in the inferior leads.

JACC Vol. 45, No. 6, 2005
March 15, 2005:877–86

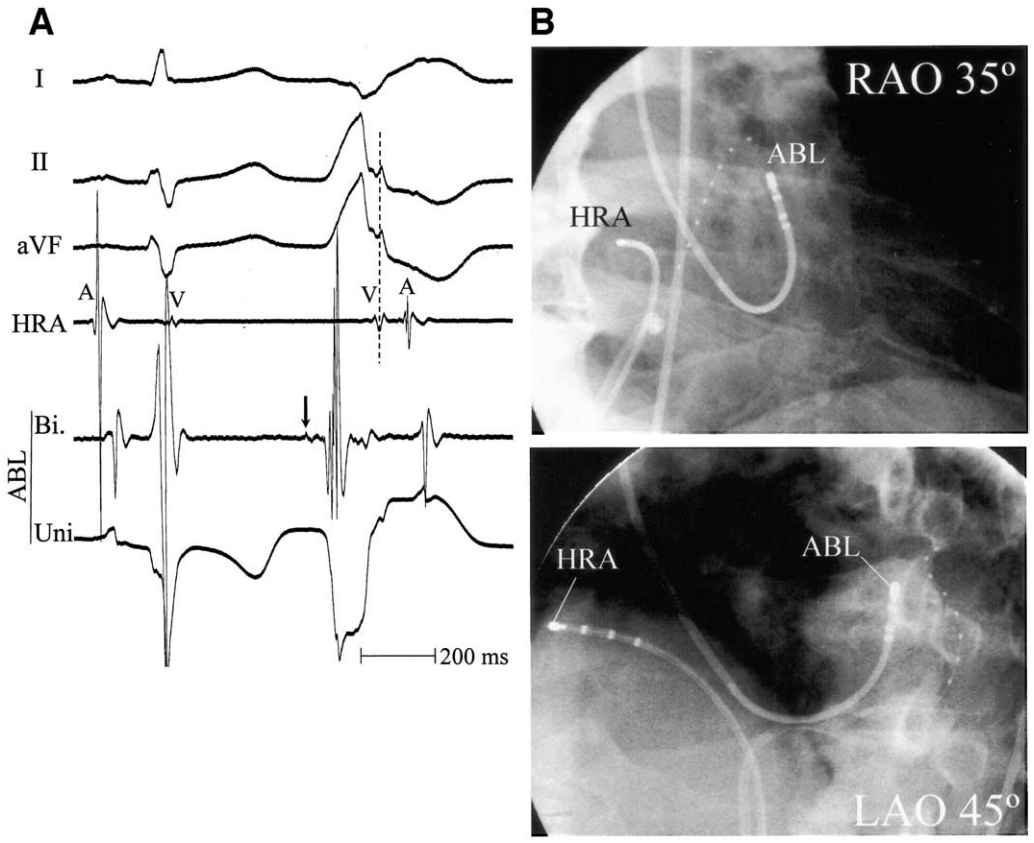

Figure 2. Site of successful ablation of a premature ventricular contraction originating from the anterolateral portion of the mitral annulus (Patient #1). **(A)** Intracardiac recordings. During the premature ventricular contraction, a low-amplitude presystolic potential recorded by the ablation catheter (ABL) preceded the onset of the QRS complex by 34 ms **(arrow)**. The timing of the second peak of the "notched" R-wave corresponded precisely with that of the activation of the right ventricular free wall **(dotted line)**, which was recorded by the catheter at the high right atrium (HRA). **(B)** Radiographs obtained in right anterior oblique (RAO, 35°) and left anterior oblique (LAO, 45°) projections show ablation sites. A distal electrode of the ablation catheter was positioned at the anterolateral-mitral annulus. A = atrial activation; Bi. = bipolar electrogram; Uni. = unipolar electrogram; V = ventricular activation.

found in PS-MAVT/PVC. The timing of the second peak of the notched QRS complex in inferior leads corresponded precisely with that of activation of the RV free wall (Figs. 2 and 3).

Pace mapping study. Characteristics of QRS morphology during pacing from the three MA sites were almost identical with those of the MAVT/PVC originating from the three sites of the MA (Table 4, Fig. 4B). The QRS duration was significantly longer during pacing from the AL-MA and Pos-MA than during pacing from the PS-MA ($p < 0.05$). Late-phase notching of the QRS complex in inferior leads was observed during pacing from the AL-MA and Pos-MA in all subjects, and the timing of the second peak of the notched QRS complex in inferior leads corresponded precisely with that of activation of the RV free wall. However, a notched QRS complex in inferior leads was not found during pacing from the PS-MA in four of five subjects. In one subject showing notching in the inferior leads during pacing from the PS-MA, the QRS duration was longer than in the remaining four subjects. The Q-wave amplitude ratio of lead III to lead II was greater during pacing from the PS-MA (1.8 ± 0.2) than during pacing from the Pos-MA (1.4 ± 0.04; $p < 0.001$).

DISCUSSION

Major findings. This study demonstrated for the first time that 5% of IVT/PVCs had an origin in the MA, as confirmed by the site of successful RFCA, and that MAVT/PVCs originated in the anterior MA, in close proximity to the mitral-aortic continuity, as well as the PS or posterior MA. Detailed ECG analysis also demonstrated several characteristic ECG findings, providing a convenient way to identify the precise tachycardia origin at the MA. Polarity of the QRS complex in the inferior leads and leads I and aVL was useful for differentiating AL-MAVT/PVC from Pos-MAVT/PVC and PS-MAVT/PVC. The presence of a negative component in the QRS complex in leads I and V_1 or a greater Q-wave amplitude ratio of lead III to lead II was useful for differentiating PS-MAVT/PVC from Pos-MAVT/PVC. The Pos-MAVT/PVC and AL-MAVT/PVC originating from the LV free wall had notching of the late phase of the QRS complex in inferior leads, as well as a longer QRS duration, in distinction from PS-MAVT/PVC. These ECG characteristics were confirmed by the pace mapping study. In all patients, all MAVT/PVCs could be eliminated by RF energy delivery at the sites where the

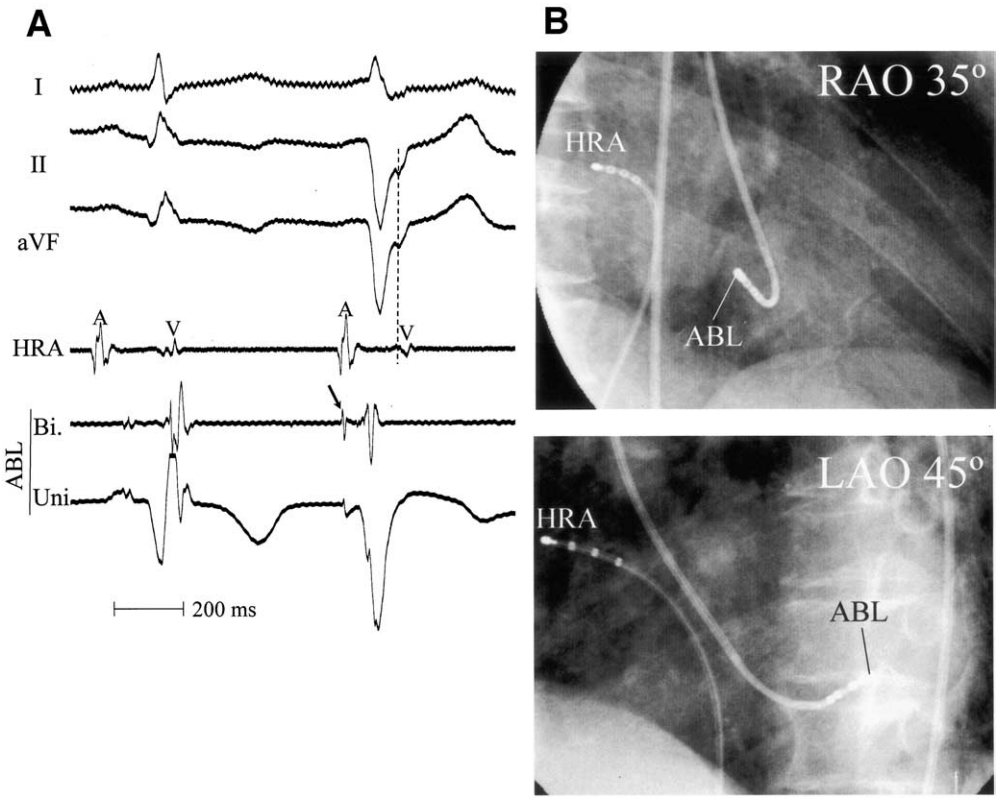

Figure 3. Site of successful ablation of a premature ventricular contraction originating from the posterior mitral annulus (Patient #12). **(A)** Intracardiac recordings. During the premature ventricular contraction, a distinct presystolic potential recorded by the ablation catheter preceded the onset of the QRS complex by 60 ms **(arrow)**. The timing of the second peak of the "notched" Q-wave corresponded precisely with that of activation of the right ventricular free wall **(dotted line)**, which was recorded with the catheter at the high right atrium (HRA). **(B)** Radiographs obtained in the right anterior oblique (RAO, 35°) and left anterior oblique (LAO, 45°) projections show ablation sites. A distal electrode of the ablation catheter was positioned at the posterior mitral annulus. A = atrial activation; Bi. = bipolar electrogram; Uni. = unipolar electrogram; V = ventricular activation.

earliest ventricular activation during the MAVT/PVC was recorded or where perfect pace mapping was obtained.

Our results indicate that despite their rarity, MAVT/PVC can be identified as a subgroup of IVT/PVC with distinctive ECG characteristics, and that RFCA is effective for eliminating MAVT/PVC. The characteristic ECG findings identified in the present study could provide advance knowledge of the precise MA location of the MAVT/PVC. This is important in formulating a strategy and preparing instruments for RFCA to maximize ease and likelihood of success.

Electrocardiographic findings of MAVT/PVC and their proposed mechanisms. In the present study, early precordial transition by lead V_2 and an R or Rs pattern in leads V_2 to V_5 were observed in all MAVT/PVCs. Because the origin of tachycardia in the MA was located in the posterior portion of the LV, distant from the precordial electrodes, the myocardium near the focus was depolarized in a direction toward these electrodes. This could account for the early precordial transition and a concordant positive QRS pattern in leads V_2 to V_4 of MAVT/PVC. In AL-MAVT/PVC and Pos-MAVT/PVC, lead V_1 also demonstrated an R pattern because of the anterior direction of the ventricular force. However, in PS-MAVT/PVC, a negative component of the QRS complex was found in lead V_1. The ventricular septum is oriented nearly horizontally at the electrode of lead V_1, and the PS-LV is the farthest right to the portion of the MA. At the beginning of ventricular activation, the myocardium near a tachycardia focus within the PS-MA would be depolarized in a direction toward the left and away from the electrode of lead V_1. This may account for the presence of the negative component of the QRS complex in lead V_1 in PS-MAVT/PVC. In PS-MAVT/PVC, the tachycardia originates further to the right, so the mean QRS vector would be directed more to the left than in Pos-MAVT/PVC. This may be the reason why PS-MAVT/PVCs had a greater Q-wave amplitude ratio of lead III to lead II than did Pos-MAVT/PVC. In the present study, the presence of the terminal S-wave of the QRS complex in lead I was also useful to distinguish AL-MAVT/PVC and Pos-MAVT/PVC from PS-MAVT/PVC. Because the tachycardia foci of AL-MAVT/PVC and Pos-MAVT/PVC are located to the left of the focus of PS-MAVT/PVC, the mean terminal QRS vector would be expected to be directed to the right, which may account for the presence of an S-wave in lead I in these MAVT/PVC.

"Notching" of the late phase of the QRS complex in the inferior leads was observed more often, and the QRS duration was longer in AL-MAVT/PVC and Pos-MAVT/PVC than in PS-MAVT/PVC. These findings were also

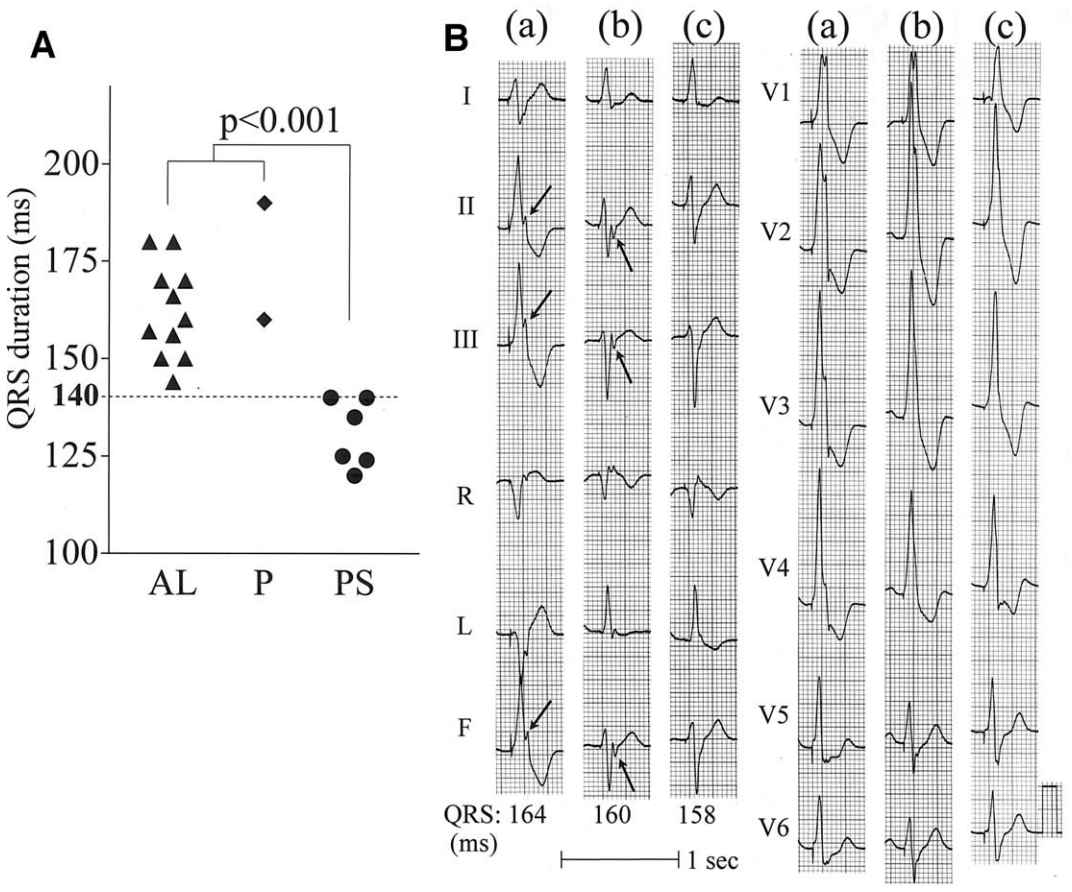

Figure 4. QRS duration and notching. **(A)** Total duration of the QRS complex of mitral annulus (MA) ventricular tachycardia (VT)/premature ventricular contraction (PVC). In anterolateral (AL) and posterior (P) MAVT/PVC, the QRS duration was significantly longer than in the posteroseptal (PS)-MAVT/PVC. The QRS duration was >140 ms in all AL-MAVT/PVC and Pos-MAVT/PVC, whereas it was <140 ms in all PS-MAVT/PVC. **(B)** Twelve-lead ECGs obtained during pacing from the AL **(a)**, P **(b)**, and PS **(c)** portion of the MA. **Arrows** indicate "notching" of the late phase of the QRS complex in the inferior leads.

observed during pacing from the three sites of the MA. Timing of excitation of both ventricles is considered to affect QRS morphology and its width in inferior leads. A notched QRS pattern and a longer QRS duration in inferior leads are often recognized in IVT/PVC originating from the free wall of the RVOT; phased excitation proceeding from the RV free wall to the LV is the likely basis for these findings (12,13). Posterior and AL-MAVT/PVC originate from the free wall of the LV. The timing of the second peak of the notched R-wave of AL-MAVT/PVC and Pos-MAVT/PVC corresponded precisely to the time of activation of the RV free wall. Accordingly, notching of the late phase of the QRS complex in inferior leads and widening of the QRS complex observed in these MAVT/PVC may result from phased excitation from the LV free wall to the RV. Because the magnitude of ventricular force and the

Table 4. Electrocardiographic Characteristics Obtained From Pacing at the Three Sites of the Mitral Annulus

Pacing Site in Mitral Annulus	Anterolateral	Posterior	Posteroseptal
QRS duration (ms)	165 ± 9	166 ± 8	154 ± 11*
QRS morphology (leads)			
I	RS, 3; rs, 2	Rs, 4; rs, 1	Rs, 4; R, 1
II	R, 4; Rs, 1	rS, 3; RS, 1; rs, 1	rS, 3; RS, 1; Rs, 1
III	R, 4; Rs, 1	rS, 4; rs, 1	rS, 4; QS, 1
aVL	rS, 3; QS, 2	R, 2; Rs, 2; rsr, 1	all R
aVF	R, 4; Rs, 1	rS, 4; rs, 1	rS, 3; rs, 1; QS, 1
V_1	all R	all R	R, 3; rsR, 2
Transition zone in precordial leads	all $<V_1$	all $<V_1$	all $<V_1$
Late "notching" in inferior leads	all +	all +	+, 1; −, 4

*$p < 0.05$ compared with QRS duration during pacing from the anterolateral and posterior mitral annulus. Data are expressed as the mean value ± SD.

time of activation are expected to be much greater in the LV than that in the RV, the second peak of the notched R-wave may appear as the late phase of the QRS complex. On the other hand, in PS-MAVT/PVC, both ventricles are excited almost simultaneously, which might result in a shorter QRS duration and absence of notching in inferior leads.

Genesis of MAVT/PVC. Results obtained from the present and previous studies cannot determine why the MAVT/PVC occurred often at the AL, PS, or posterior MA, and only a speculative explanation is possible. The AL portion of the MA is close to the anterior attachment of the RVOT, the LV epicardial myocardium near the left sinus of Valsalva, and the anteromedial (subvalvular) portion of the LVOT, which are often reported as foci of IVT/PVC originating from the outflow tract. The close proximity suggests that these different forms of IVT/PVC could likely originate from a single focus, with different exit points or activation of alternative pathways between the tachycardia focus and an exit point (14). The AL-MA is also a common origin of left atrial tachycardia (15), which would differ from AL-MAVT/PVC in having an exit site in the left atrium, not the LV.

As considered in a previous report concerning left atrial tachycardia (15,16), a remnant of the atrioventricular conduction system close to the aortic-mitral continuity, such as a "dead-end" tract (17), might be important in the genesis of the non-reentrant mechanism for tachycardia. Remnants of the atrioventricular ring, as specialized tissue in the posterior or PS-MA, might also be related to genesis of Pos- and PS-MAVT/PVC (18).

Previous studies. Although several studies have reported AL-MAVT/PVC (3,8–10), none have systemically determined the prevalence and ECG characteristics of MAVT/PVC originating from the MA, based on as many patients as in this study.

In the present results, an S-wave was present in lead V_6 in all MAVT/PVCs, which is considered a characteristic and convenient ECG finding in IVT/PVC of LVOT origin (7). However, some AL-MAVT/PVCs reported previously showed no late notching in inferior leads (3,4,9) nor an S-wave in lead V_6 (4). When tachycardia originates beneath the aortic valve near the anteromedial portion of the MA, which has been reported as one of the major origins of IVT/PVC from the LVOT (3,4,9), late notching in the inferior leads or an S-wave in lead V_6 may be absent, as in PS-MAVT/PVC (7,19). In this setting, the ventricular septum and RV would be depolarized earlier than in MAVT/PVC originating from the portion of the annulus at the LV free wall, which may result in absence of notching in inferior leads or in an S-wave absence in lead V_6. Earlier depolarization of MAVT/PVC may also result in the qR morphology of the lead V_1 in AL-MAVT/PVC (3). Therefore, an AL-MAVT/PVC might lack an S-wave in lead V_6, notching in the inferior leads, or a negative component of the QRS complex in lead V_1 when it originates more anteromedially. Considering the various possibilities, we have developed an ECG algorithm for identifying the precise origin of tachycardia in MAVT/PVC (Fig. 5). This algorithm correctly identified the origin of the MAVT/PVC in the 352 patients in this study, with a sensitivity of 60%, specificity of 99.7%, positive predictive value of 95%, and negative predictive value of 96%; one MAVT/PVC was misdiagnosed as having a non-MA origin. Inversely, five IVT/PVCs, which were ablated successfully from the aortic sinus of Valsalva or which originated from the LV epicardium remote from the left sinus of Valsalva, and another five originating from the LVOT were misdiagnosed as MAVT/PVCs.

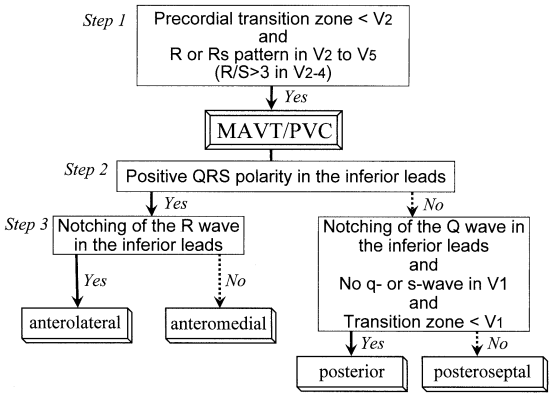

Figure 5. Proposed algorithm to predict the precise focus of mitral annulus (MA) ventricular tachycardia (VT)/premature ventricular contraction (PVC) based on the QRS wave configuration on 12-lead electrocardiographic recordings.

Study limitations. The first limitation of the present study involves difficulties in determining mechanisms of tachycardia. In the two MAVT/PVCs that were reproducibly induced or terminated by programmed electrical stimulation, reentry was the most likely mechanism of tachycardia. However, the remaining MAVT/PVCs could not be induced by programmed electrical stimulation. Instead, they occurred spontaneously or during isoproterenol infusion, which suggests a non-reentrant mechanism for tachycardia, most likely involving triggered activity or automaticity. As we did not perform an extensive examination with programmed electrical stimulation or infusion of drugs such as adenosine, the precise mechanism underlying these MAVT/PVCs was not determined.

The second limitation is that while a potential preceding the QRS complex was often recorded at the site of successful ablation, its genesis and significance were not determined.

The third limitation is that pace mapping from the MA was performed in a bipolar fashion. A relatively large amount of myocardium captured around the pacing electrodes might obscure subtle changes in the QRS morphology at the pacing sites.

The fourth limitation is that there was a low successful ablation rate for IVT/PVCs from the RV septum or near the His bundle region or for those in which RF energy was applied from the aortic sinus of Valsalva (Table 1).

The insufficient RF energy applications for the IVT/PVCs originating from the RV septum or near the His bundle region because of the risk of impairing atrioventricular conduction and the great distance from the aortic sinus of Valsalva to the tachycardia focus in the IVT/PVCs originating from the LV epicardium, might have resulted in the low success rate of this study. Use of a cryoablation catheter for IVT/PVCs from the RV septum or near the His bundle region (20) or an approach through the coronary venous system or using by percutaneous epicardial instrumentation for LV epicardial VT/PVCs (21,22) might result in the more favorable outcome.

Conclusions. Mitral annular VT/PVC is a rare but identifiable subgroup of IVT/PVC with distinctive ECG characteristics. Radiofrequency catheter ablation is effective for eliminating MAVT/PVC. Advance knowledge of an MA origin of IVT/PVC may be useful in planning and facilitating the RF ablation procedure.

Reprint requests and correspondence: Dr. Hiroshi Tada, Division of Cardiology, Gunma Prefectural Cardiovascular Center, 3-12 Kameizumi, Maebashi, Gunma 371-0004, Japan. E-mail: tada.h@cvc.pref.gunma.jp.

REFERENCES

1. Daoud E, Morady F. Catheter ablation of ventricular tachycardia. Curr Opin Cardiol 1995;10:21–5.
2. Coggins DL, Lee RJ, Sweeney J, et al. Radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. J Am Coll Cardiol 1994;23:1333–41.
3. Callans DJ, Menz V, Schwartzman D, et al. Repetitive monomorphic tachycardia from the left ventricular outflow tract: electrocardiographic patterns consistent with a left ventricular site of origin. J Am Coll Cardiol 1997;29:1023–7.
4. Shimoike E, Ohba Y, Yanagi N, et al. Radiofrequency catheter ablation of left ventricular outflow tract tachycardia: report of two cases. J Cardiovasc Electrophysiol 1998;9:196–202.
5. Kanagaratnam L, Tomassoni G, Schweikert R, et al. Ventricular tachycardias arising from the aortic sinus of Valsalva: an under-recognized variant of left outflow tract ventricular tachycardia. J Am Coll Cardiol 2001;37:1408–14.
6. Ouyang F, Fotuhi P, Ho SY, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation. J Am Coll Cardiol 2002;39:500–8.
7. Ito S, Tada H, Naito S, et al. Development and validation of an ECG algorithm for identifying the optimal ablation site for idiopathic ventricular outflow tract tachycardia. J Cardiovasc Electrophysiol 2003;14:1280–6.
8. Yeh SJ, Wen MS, Wang CC, Lin FC, Wu D. Adenosine-sensitive ventricular tachycardia from the anterobasal left ventricle. J Am Coll Cardiol 1997;30:1339–45.
9. Nagasawa H, Fujiki A, Usui M, Mizumaki K, Hayashi H, Inoue H. Successful radiofrequency catheter ablation of incessant ventricular tachycardia with a delta wave-like beginning of the QRS complex. Jpn Heart J 1999;40:671–5.
10. Kondo K, Watanabe I, Kojima T, et al. Radiofrequency catheter ablation of ventricular tachycardia from the anterobasal left ventricle. Jpn Heart J 2000;41:215–5.
11. Goldman MJ. Definitions of electrocardiographic configurations. In: Goldman MJ, editor. Principles of Clinical Electrocardiography. Los Altos, CA: Lange Medical Publications (Maruzen Asia Ltd.), 1982: 23–8.
12. Kamakura S, Shimizu W, Matsuo K, et al. Localization of optimal ablation site of idiopathic ventricular tachycardia from right and left ventricular outflow tract by body surface ECG. Circulation 1998;98: 1525–33.
13. Dixit S, Gerstenfeld EP, Callans DJ, Marchlinski FE. Electrocardiographic patterns of superior right ventricular outflow tract tachycardias: distinguishing septal and free-wall sites of origin. J Cardiovasc Electrophysiol 2003;14:1–7.
14. Kimber SK, Downar E, Harris L, et al. Mechanisms of spontaneous shift of surface electrocardiographic configuration during ventricular tachycardia. J Am Coll Cardiol 1992;20:1397–404.
15. Kistler PM, Sanders P, Hussin A, et al. Focal atrial tachycardia arising from the mitral annulus: electrocardiographic and electrophysiologic characterization. J Am Coll Cardiol 2003;41:2212–9.
16. Nogami A, Suguta M, Tomita T, et al. Novel form of atrial tachycardia originating at the atrioventricular annulus. Pacing Clin Electrophysiol 1998;21:2691–4.
17. Kurosawa H, Becker AE. Dead-end tract of the conduction axis. Int J Cardiol 1985;7:13–20.
18. Anderson RH, Davis MJ, Becker AE. Atrioventricular specialized tissue in the normal heart. Eur J Cardiol 1974;2/2:219–30.
19. Dixit S, Lin D, Zado ES, Marchlinski FE. Identification of distinct electrocardiographic patterns from basal left ventricle: distinguishing medial and lateral sites of origins. Heart Rhythm 2004;1 Suppl 1:S104–5.
20. Atienza F, Arenal A, Torrecilla EG, et al. Acute and long-term outcome of transvenous cryoablation of midseptal and parahissian accessory pathways in patients at high risk of atrioventricular block during radiofrequency ablation. Am J Cardiol 2004;93:1302–25.
21. Stellbrink C, Diem B, Schauerte P, Ziegert K, Hanrath P. Transcoronary venous radiofrequency catheter ablation of ventricular tachycardia. J Cardiovasc Electrophysiol 1997;8:916–21.
22. Schweikert RA, Saliba WI, Tomassoni G, et al. Percutaneous pericardial instrumentation for endo-epicardial mapping of previously failed ablations. Circulation 2003;108:1329–35.