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Electrocardiographic and Electrophysiologic Characteristics of Ventricular Tachycardia Originating Within the Pulmonary Artery

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OBJECTIVES We investigated the electrocardiographic (ECG) and electrophysiologic characteristics of ventricular tachycardia (VT) originating within the pulmonary artery (PA).

BACKGROUND Radiofrequency catheter ablation (RFCA) is routinely applied to the endocardial surface of the right ventricular outflow tract (RVOT) in patients with idiopathic VT of left bundle branch block morphology. It was recently reported that this arrhythmia may originate within the PA.

METHODS Activation mapping and ECG analysis were performed in 24 patients whose VTs or ventricular premature contractions (VPCs) were successfully ablated within the PA (PA group) and in 48 patients whose VTs or VPCs were successfully ablated from the endocardial surface of the RVOT (RV-end-OT group).

RESULTS R-wave amplitudes on inferior ECG leads, aVL/aVR ratio of Q-wave amplitude, and R/S ratio on lead V_2 were significantly larger in the PA group than in the RV-end-OT group. On intracardiac electrograms, atrial potentials were more frequently recorded in the PA group than in the RV-end-OT group (58% vs. 12%; $p < 0.01$). The amplitude of local ventricular potentials recorded during sinus rhythm within the PA was significantly lower than that recorded from the RV-end-OT (0.62 ± 0.56 mV vs. 1.55 ± 0.88 mV; $p < 0.01$).

CONCLUSIONS Ventricular tachycardia originating within the PA has different electrocardiographic and electrophysiologic characteristics from that originating from the RV-end-OT. When mapping the RVOT area, the catheter may be located within the PA if a low-voltage atrial or local ventricular potential of <1-mV amplitude is recorded. Heightened attention must be paid if RFCA is required within the PA. (J Am Coll Cardiol 2005;45:887–95) © 2005 by the American College of Cardiology Foundation

Most repetitive monomorphic ventricular tachycardia (VT) and symptomatic monomorphic ventricular premature contractions (VPCs) that originate from the ventricular outflow tract (OT) are known to arise from endocardial sites, and radiofrequency catheter ablation (RFCA) has been increasingly used with a high success rate in the treatment of these arrhythmias (1–7). However, there are some cases in which RFCA cannot be performed from either right ventricular (RV) or left ventricular (LV) endocardial sites. Several reports have indicated that OT-VTs in such cases can originate from the aortic sinus cusp (ASC) or the pulmonary artery (PA) and can be ablated successfully in selected patients (8–14). Although much is known about the characteristics of OT-VT originating from the ASC (8–12), little is known or reported about the characteristics of VT that originate within the PA in many patients. The aim of this study was to investigate the characteristics of VT originating within the PA and to assess the role of RFCA in eliminating these VTs.

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METHODS

Study population. Between June 1997 and July 2003, 148 patients (58 men and 90 women, age 56.1 ± 14.5 years) with symptomatic and frequent monofocal VT or VPCs were referred to our hospital for catheter ablation. The surface electrocardiograms (ECGs) recorded during either VT or VPCs showed left bundle branch block (LBBB) morphology and inferior axis in all patients. The selection criteria for catheter ablation included fatal arrhythmias or severe symptoms that were clearly related to frequent ventricular arrhythmias, inability of the patient to tolerate treatment or unsuccessful treatment with at least two antiarrhythmic drugs (mean 2.3 ± 0.5 drugs), and a structurally normal heart as examined by coronary angiography and echocardiography. In 21 of these patients, the arrhythmias could not be eliminated by RFCA because adequate radiofrequency energy could not be delivered safely to avoid risk, the site of earliest ventricular activation could not be determined by electrophysiologic study, or stable position of the ablation catheter could not be maintained. In the remaining 127 patients successfully treated by RFCA, the successful ablation sites were located in the ASC in 11 patients, above the pulmonary valve in 24 patients, and on the endocardial surface of the right ventricular outflow tract (RVOT) in 92 patients. The study group in the present study consisted of 24 patients with VTs or VPCs success-

Abbreviations and Acronyms

ASC = aortic sinus cusp
LBBB = left bundle branch block
LV = left ventricle/ventricular
OT = outflow tract
PA = pulmonary artery
RFCA = radiofrequency catheter ablation
RV = right ventricle/ventricular
RVOT = right ventricular outflow tract
VPC = ventricular premature contraction
VT = ventricular tachycardia

fully ablated above the pulmonary valve (PA group) (Table 1). Five of the PA group patients had undergone failed procedures performed previously in other institutions. The control group comprised the last 48 consecutive patients of 92 patients with VTs or VPCs successfully ablated from the endocardial surface of the RVOT (RV-end-OT group).

12-lead ECG analysis. A 12-lead ECG was recorded during the ventricular arrhythmia. Analysis of the surface ECG focused on R-wave amplitude in leads II, III, aVF, and V_2; Q-wave amplitude in leads aVR and aVL; S-wave amplitude in lead V_2; R-wave polarity in lead I; and the precordial R-wave transitional zone.

Mapping and RFCA. All patients gave written informed consent before procedures. Catheters were inserted through the right femoral vein to the RVOT and His bundle region under fluoroscopic guidance, and activation mapping was performed at the RV endocardial area and the site above the pulmonary valve during spontaneous VT or VPCs. If the clinical VT or VPCs did not appear spontaneously, ventricular stimulation or intravenous isoproterenol infusion (0.5 to 4 μg/min) was administered to provoke the arrhythmia. During the clinical ventricular arrhythmia, bipolar mapping was performed to determine the earliest endocardial activation site. Additionally, simultaneous unfiltered unipolar mapping was performed between the tip electrode of the mapping catheter and a reference electrode in the inferior vena cava. Finally, pace mapping was performed. If pacing stimuli from a conventional pacing unit using maximal output (9.9 V/2.0 ms) could not be captured, high-output pacing was applied with a transesophageal pacing unit at maximum 40-mA/20-ms output. If the VT did not originate from the RV endocardial area or within the PA, or if good mapping could not be obtained anywhere, a 3.3-F 16-polar electrode catheter inserted into the right subclavian vein was positioned in the proximal portion of the anterior interventricular vein distal to the great cardiac vein to map the LV site precisely. The ablation target site was defined as the site where the best pace mapping and the earliest endocardial activation time could be obtained during VT or VPCs. Catheter positions were evaluated in all patients by pulmonary arteriogram and coronary angiography before each radiofrequency application to a new radiofrequency delivery site. Radiofrequency catheter ablation was not performed if the distance from the ablation catheter tip to

Table 1. The Clinical and Electrocardiographic Characteristics of VT or VPCs Originating Within the Pulmonary Artery

Patient	Gender	Age (yrs)	Clinical Arrhythmia	Distance Between RF Site and Pulmonary Valve (cm)	R-Wave Amplitudes in Inferior Leads (mV)			R/S Ratio on Lead V_2	Precordial Transitional Zone
					II	III	aVF		
1	M	58	VT	1.1	2.5	2.8	2.7	0.19	V_4
2	F	58	VT	1.5	2.2	2.6	2.3	0.21	V_4
3	F	54	VT	2.1	1.6	1.4	1.5	0.77	V_3
4	F	73	VPCs	1.8	1.9	1.7	1.8	0.14	V_3
5	F	32	VT	1.7	1.7	1.8	1.8	0.00	V_6
6	M	62	VPCs	0.6	2.2	2.2	2.0	0.16	V_5
7	F	65	VPCs	1.6	1.6	1.8	1.6	0.19	V_5
8	M	54	VPCs	0.7	2.0	1.5	1.6	0.61	V_3
9	F	53	VT	1.2	2.5	1.8	1.8	0.03	V_4
10	M	77	VPCs	1.2	1.1	1.2	1.2	0.08	V_6
11	M	73	VPCs	0.8	1.8	1.6	1.6	0.38	V_3
12	F	49	VPCs	1.6	1.7	1.8	1.7	0.06	V_5
13	F	56	VPCs	1.0	1.4	1.3	1.3	0.21	V_4
14	F	56	VT	1.8	2.0	1.8	1.7	0.46	V_3
15	F	54	VPCs	1.2	1.6	1.6	1.6	0.86	V_2
16	M	39	VT	0.5	2.8	3.1	3.3	0.11	V_5
17	F	72	VPCs	1.5	1.4	1.3	1.4	0.09	V_4
18	F	41	VPCs	0.6	2.1	2.2	2.0	0.54	V_3
19	F	22	VT	0.8	1.9	2.6	2.5	0.20	V_4
20	M	40	VT	0.9	2.3	2.5	2.6	0.09	V_4
21	M	43	VPCs	1.0	2.2	2.2	2.3	1.33	V_2
22	M	51	VPCs	1.1	1.3	1.2	1.2	0.13	V_5
23	F	39	VT	1.0	2.4	2.4	2.4	0.50	V_3
24	F	67	VPCs	1.0	1.8	1.2	1.5	0.33	V_3

RF = radiofrequency; VPCs = ventricular premature contractions; VT = ventricular tachycardia.

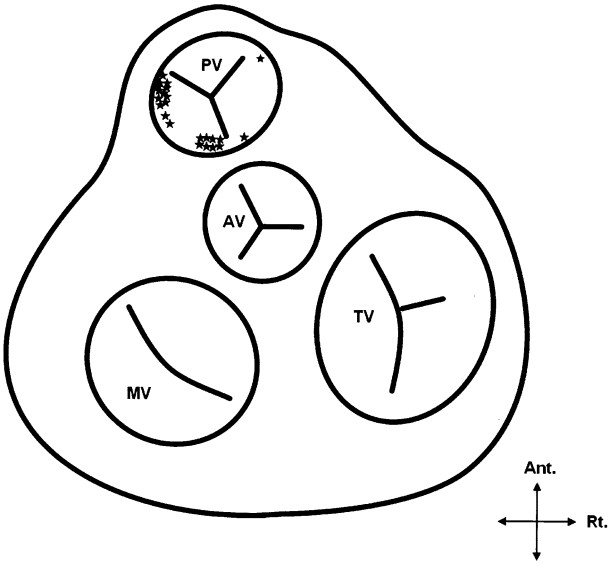

Figure 1. Anatomic location of the successful ablation sites in the pulmonary artery group. The successful ablation sites are indicated by the stars (★), and they were mostly along the septum. Ant. = anterior; AV = aortic valve; MV = mitral valve; PV = pulmonary valve; Rt. = right; TV = tricuspid valve.

the coronary artery was <5 mm. Radiofrequency energy was delivered with a 7-F, 4-mm-tipped ablation catheter (EPT5031TL, EP Technologies, Inc., San Jose, California) and maintained for 60 to 90 s at a temperature of 55°C when the catheter was positioned above the pulmonary valve and at 60°C when the catheter was positioned below the pulmonary valve. Radiofrequency energy was never delivered at the same site more than three times when the catheter was positioned above the pulmonary valve. After elimination of the arrhythmia by radiofrequency delivery, catheter position was evaluated by repeat pulmonary arteriogram to confirm its precise location. Successful ablation was defined as no VT and <100 beats per day of the target ventricular arrhythmia after 48 h of continuous ECG monitoring with the patient on no antiarrhythmic drugs.

Statistical analysis. Parametric data are expressed as mean ± SD. The parameters in different groups were compared by Student unpaired *t* test. Categorical variables were compared using chi-square analysis or Fisher exact test. A value of $p < 0.05$ was considered significant.

RESULTS

Clinical characteristics. Thirty-four of the 72 patients had monomorphic VT and frequent monomorphic VPCs, whereas the remaining 38 patients had only frequent monomorphic VPCs. In the PA group, the results of clinical and ECG data are summarized in Table 1, and successful ablation sites are shown in Figure 1; VT was seen in 10 of 24 patients. The successful ablation sites were located 1.18 ± 0.43 cm above the pulmonary valve and mostly along the septum. The comparison of clinical characteristics and electrophysiologic data between the PA and RV-end-OT groups is summarized in Table 2. There were no significant differences between the two groups in terms of gender, age, and number of VPCs per day.

Electrocardiographic characteristics. Several ECG characteristics of VT that originates within the PA were noted (Fig. 2). The R-wave amplitudes on inferior leads in the PA group were significantly larger than those in the RV-end-OT group (lead II: 1.92 ± 0.42 mV vs. 1.57 ± 0.37 mV, $p < 0.05$; lead III: 1.90 ± 0.55 mV vs. 1.49 ± 0.48 mV, $p < 0.01$; lead aVF: 1.89 ± 0.53 mV vs. 1.53 ± 0.44 mV, $p < 0.05$) (Fig. 3).

Seventeen of 24 (71%) PA group patients had Q-wave amplitudes on lead aVL that were equal to or larger than those on lead aVR. In contrast, 34 of 48 (71%) RV-end-OT group patients had larger Q-wave amplitudes on lead aVR than those on lead aVL. The average aVL/aVR ratio of Q-wave amplitude was >1 in the PA group; this was significantly larger than that in the RV-end-OT group (1.11 ± 0.40 vs. 0.88 ± 0.33; $p < 0.05$) (Fig. 4A). In comparing lead I polarity, QRS morphology showed a QS (rS) pattern in 15 (63%) patients of the PA group, whereas 30 (63%) patients of the RV-end-OT group showed an R (Rs) pattern (Fig. 4B). The results of precordial R-wave transition in the two groups are summarized in Fig. 4C. The transitional zone was often observed at V_4 in both groups, and it occurred from either V_2 or V_3 in 10 of 24 (42%) PA group patients. Moreover, in two (8%) PA group patients, it was observed at lead V_2 in comparison with no patient in the RV-end-OT group. There was a distinct R-wave amplitude on lead V_2 in patients of the PA group, and the R/S amplitude ratio on lead V_2 was significantly larger than that in the RV-end-OT group (0.32 ± 0.32 vs. 0.17 ± 0.14; $p < 0.05$) (Fig. 4D).

Mapping and RFCA. The clinical arrhythmia occurred spontaneously in 57 patients. Although neither nonsustained nor sustained clinical VT could be induced by programmed ventricular stimulation in any patient, it could be provoked by either burst ventricular pacing or isoproterenol infusion, suggesting that the mechanism of arrhythmia was non–re-entry in all patients.

Table 2. Comparison of Clinical Characteristics and Electrophysiologic Data Between Groups

Variable	PA Group	RV-end-OT Group	p Value
Gender (M/F)	9/15	15/33	NS
Age (yrs)	53.7 ± 13.9	58.0 ± 12.1	NS
VPCs per day (n)	20,262 ± 12,636	16,708 ± 11,712	NS
RF applications (n)	3.7 ± 2.2	5.5 ± 4.5	NS
EAT (ms)	−32.9 ± 16.6	−32.4 ± 13.1	NS
Pace mapping score (n/12)	11.3 ± 0.75	11.3 ± 0.74	NS
Use of high-output pacing unit	15/24 (63%)	0/48 (0%)	p < 0.01

Values are mean ± SD.

EAT = earliest endocardial activation time; PA = pulmonary artery; RF = radiofrequency; RV-end-OT = endocardial right ventricular outflow tract; VPCs = ventricular premature contractions.

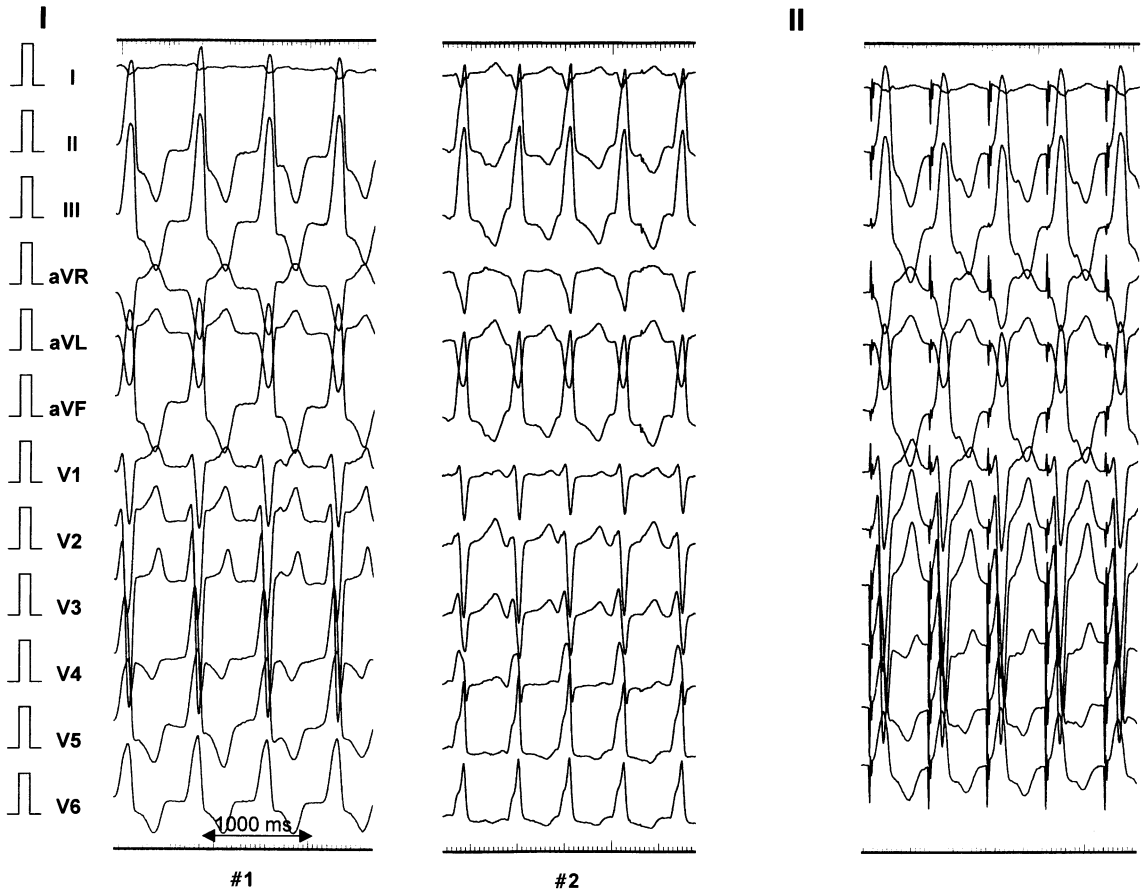

Figure 2. The electrocardiograms of Patients #1 and #2 showing clinical ventricular tachycardias (VTs) originating within the pulmonary artery (PA) **(panel I)** and the electrocardiogram of Patient #1 showing pace mapping at the successful ablation site within the PA **(panel II)**. In Patient #1, QRS complexes during pacing almost match those of the clinical VT.

The results of electrophysiologic data from the PA group are summarized in Table 3. Table 2 shows that there were no differences between the two groups in terms of number of radiofrequency applications, earliest endocardial activation time, and pace mapping score. The use of high-output pacing was not required in the RV-end-OT group at all; however, pace mapping could be obtained only by high-output pacing in 63% of the PA group patients. There were also significant differences in intracardiac electrograms between the PA and RV-end-OT groups (Fig. 5). Significantly more low-amplitude and dull atrial potentials were recorded at the successful RFCA site in the PA group than in the RV-end-OT group (58% vs. 12%; $p < 0.01$). The amplitude of local ventricular bipolar potentials recorded

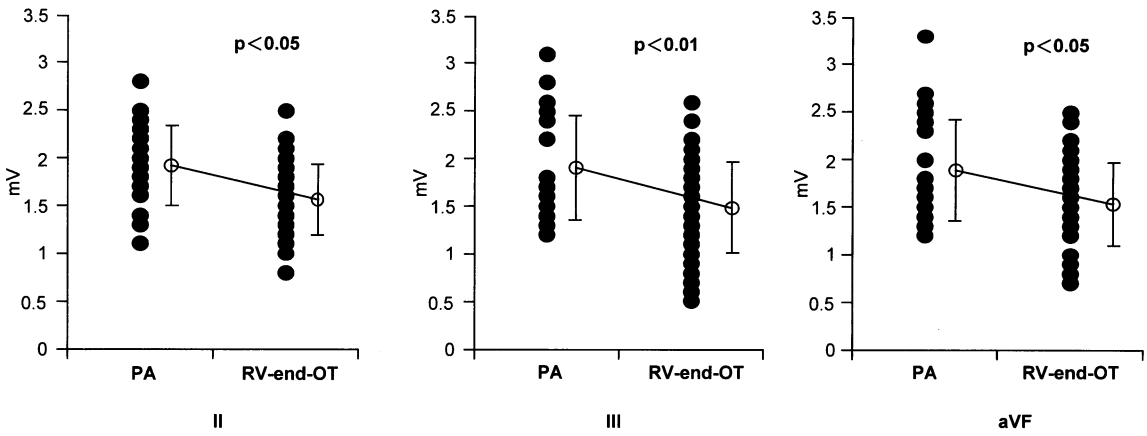

Figure 3. Plots of the R-wave amplitudes on inferior leads (II, III, aVF) in patients with clinical ventricular arrhythmia originating within the pulmonary artery (PA) and those from the endocardial right ventricular outflow tract (RV-end-OT).

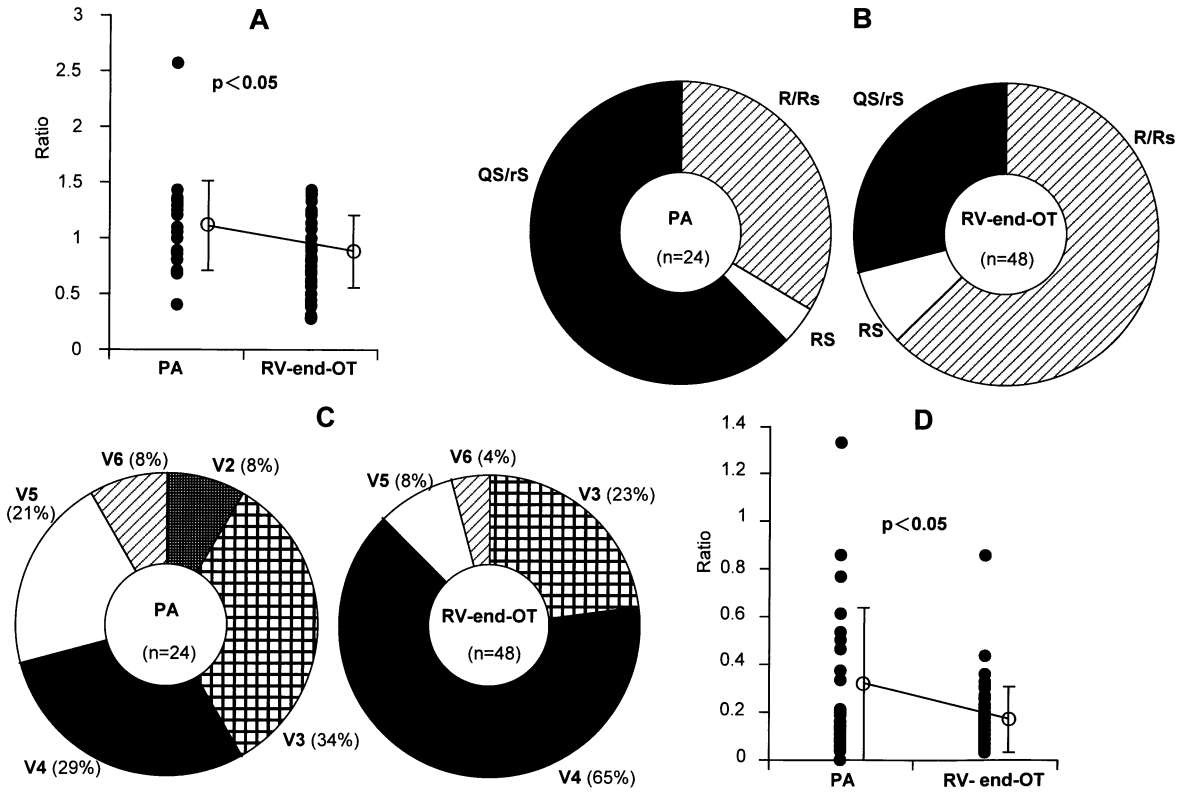

Figure 4. Comparison of electrocardiograms between the pulmonary artery (PA) group and endocardial right ventricular outflow tract (RV-end-OT) group in terms of **(A)** aVL/aVR ratio of Q-wave amplitude, **(B)** lead I polarity, **(C)** precordial R-wave transitional zone, and **(D)** R/S ratio on lead V_2.

during sinus rhythm at the successful RFCA site was significantly lower in the PA group than that in the RV-end-OT group (0.62 ± 0.56 mV vs. 1.55 ± 0.88 mV; $p < 0.01$) (Fig. 6). In regard to local ventricular potentials during the clinical ventricular arrhythmia, the onset of presystolic bipolar potentials preceded that of unipolar potentials by more than 20 ms in 9 PA group patients in comparison with only 7 RV-end-OT group patients (38% vs. 15%; $p < 0.05$), and the onset was almost equal to or within 20 ms of that of unipolar potentials in the remaining 56 patients.

In our experience, no patient's VT focus was too close to the coronary artery to ablate safely. Eleven PA group patients felt a small amount of pain when radiofrequency energy was delivered (Fig. 7). No procedure-related complications occurred in any patient. No damage was found at the pulmonary valve or the wall of the PA, nor did coronary artery spasm or stenosis occur, as determined by angiography and echocardiography. All patients were free from arrhythmias and symptoms without antiarrhythmic drugs, and no chronic complications occurred during a follow-up period of 29 ± 18 months.

DISCUSSION

ECG characteristics. The ECG characteristics of RV-end-OT VT have already been reported (15,16). In this study, we clarified the detailed ECG characteristics of VT originating within the PA in comparison with that from the RV-end-OT.

The R-wave amplitudes on inferior leads in the PA group were significantly larger than those in the RV-end-OT group. In precordial leads, the R-wave transitional zone at V_2 was not observed in RV-end-OT group patients, and the R/S ratio on lead V_2 in the PA group was significantly larger than that in the RV-end-OT group. These findings can be explained by the fact that the site of VT origin within the PA is higher than that in the RV-end-OT. Furthermore, the anatomic location of VT origin within the PA is more leftward and more anterior, resulting in an aVL/aVR ratio of Q-wave amplitude in the PA group that was significantly larger than that in the RV-end-OT group. This is also explained by the fact that a QS (rS) pattern of QRS morphology on lead I was seen in 63% of patients in the PA group, whereas 63% of patients in the RV-end-OT group showed an R (Rs) pattern.

Although significant differences could be seen in ECG characteristics between the two groups, there was overlap of characteristics between each group. In the R-wave amplitudes on inferior leads, a cutoff value of more than 18 mV on lead II allowed us to identify VT originating within the PA with 63% sensitivity, 69% specificity, and 50% predictive value. A cutoff value of more than 18 mV on lead III allowed identification of VT originating within the PA with 58% sensitivity, 71% specificity, and 50% predictive value;

Table 3. The Electrophysiologic Data of Ventricular Tachycardia or Ventricular Premature Contractions Originating Within the Pulmonary Artery

Patient	RF Applications (n)	Number of RF Delivery Sites (n)	EAT (ms)	Pace Mapping Score	Atrial Potentials in Intracardiac Electrogram	Use of High-Output Pacing Unit
1	4	2	−40	11/12	−	+
2	6	3	−39	12/12	+	−
3	5	3	−16	12/12	+	−
4	1	1	−67	11/12	+	+
5	5	3	−28	12/12	+	+
6	2	1	−51	12/12	−	−
7	1	1	−39	10/12	+	+
8	7	4	−14	10/12	−	+
9	2	2	−28	11/12	+	+
10	3	2	−11	12/12	+	+
11	5	3	−9	10/12	+	−
12	3	2	−54	12/12	+	+
13	4	2	−27	11/12	−	−
14	1	1	−27	11/12	−	−
15	5	3	−55	11/12	−	+
16	3	2	−47	12/12	+	+
17	1	1	−60	11/12	+	−
18	5	3	−12	11/12	+	−
19	4	2	−35	12/12	+	+
20	4	2	−28	11/12	+	+
21	10	6	−34	10/12	−	+
22	4	2	−24	12/12	−	+
23	2	1	−34	12/12	−	+
24	1	1	−11	12/12	−	−

EAT = earliest endocardial activation time; RF = radiofrequency.

VT originating within the PA could be identified with 75% sensitivity, 48% specificity, and 42% predictive value when the R-wave amplitude on lead aVF was more than 16 mV. The main reason for this overlap is that the RV-end-OT was next to the PA. There were not very many typical cases in which all ECG characteristics of VT within the PA could be applied because rotation and location of the heart was different in each patient. However, these ECG characteristics may be useful in predicting the focus of LBBB-VT before radiofrequency application, especially in the case of prominent tall R waves in inferior leads.

Mapping and RFCA. A previous report on five patients showed that idiopathic LBBB-VT originating above the pulmonary valve can be treated by RFCA (14), although damage to the coronary artery and the PA represent potential risks when radiofrequency energy is delivered.

On intracardiac electrograms recorded in the present study, the amplitude of local ventricular bipolar potentials recorded during sinus rhythm at the successful RFCA site was significantly lower in the PA group than in the RV-end-OT group (Fig. 6). This may be because the amount of muscle in the PA is smaller than that in the RV-end-OT. Furthermore, low-amplitude and dull atrial potentials were recorded significantly more often in patients with VT originating within the PA than in patients with RV-end-OT VT. Conversely, the existence of this atrial potential or a local ventricular potential of less than 1-mV amplitude may imply that the catheter is located not in the RV-end-OT but within the PA. We suppose this atrial potential to be a far-field potential from the left atrium. Anatomically, the PA passes anterior to and rightward of the left atrium and is more adjacent to the left atrium than to the RVOT. Considering the small amount of muscle in the PA, it is not so strange to capture a potential from the left atrium on intracardiac electrograms recorded within the PA. In addition to the intriguing differences in intracardiac electrograms between our two patient groups, it was noteworthy that the onset of the bipolar potential sometimes preceded that of the unipolar potential by more than 20 ms during ventricular arrhythmia when radiofrequency application was performed within the PA. Although the mechanism may be related to the small amount of muscle in the PA, it is one of the interesting features of VT originating within the PA.

Pace mapping was also used in all of our patients to judge whether the RFCA site was above or below the pulmonary valve. Pace mapping could be performed in only 37% of the PA group patients by conventional pacing, in contrast with 100% of the RV-end-OT patients. This strongly suggests that the pacing site may be positioned within the PA where pacing stimuli from a conventional pacing unit cannot be captured. This also may be because the amount of muscle in the PA is smaller than that in the RV-end-OT.

The sites of radiofrequency application in all of our patients were accurately evaluated and confirmed to be safe by pulmonary arteriogram and coronary angiography before and after radiofrequency energy was delivered. Although we have not yet experienced a patient in whom the site of VT

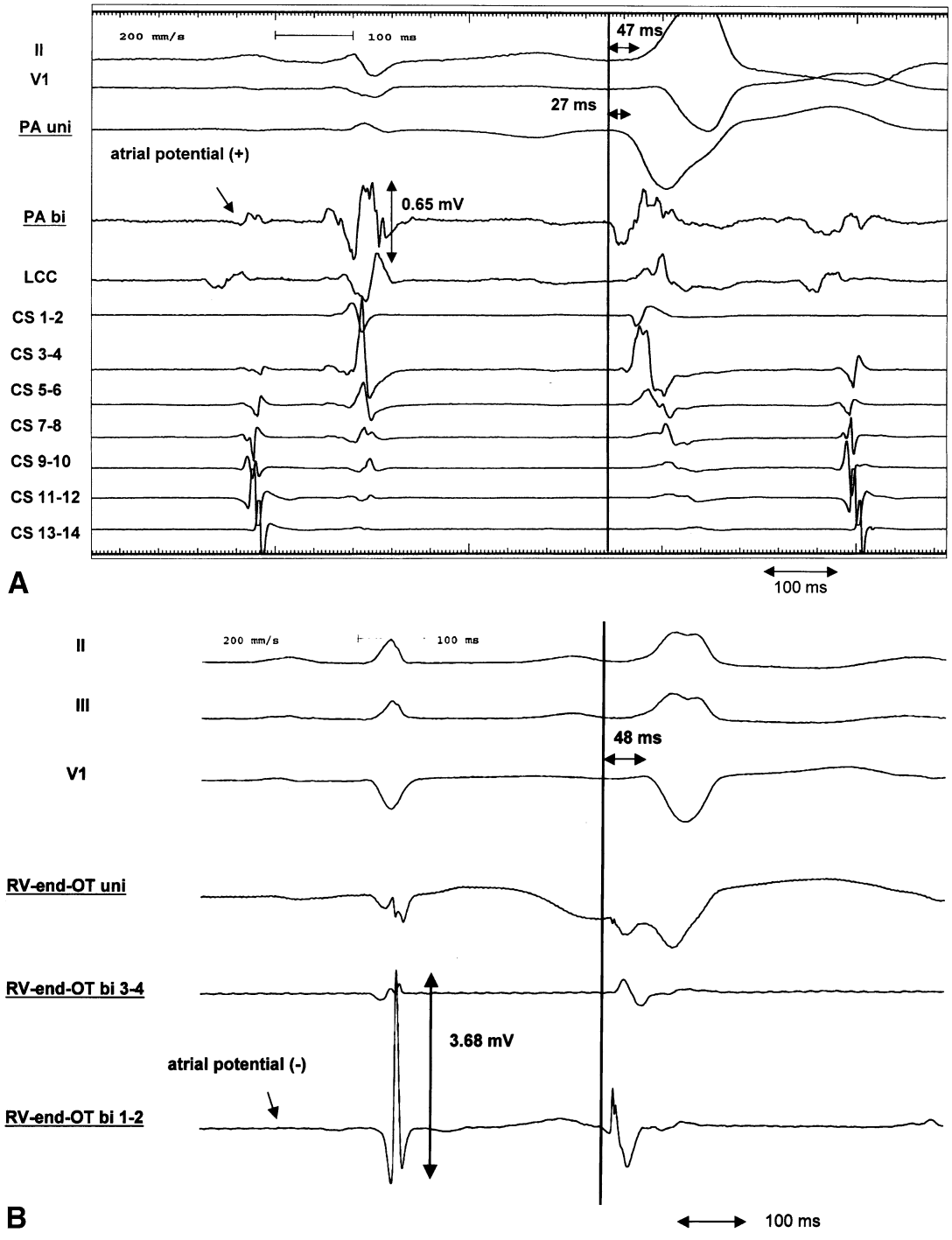

Figure 5. Two intracardiac electrograms that were recorded of a sinus beat and the clinical ventricular arrhythmia at the successful ablation sites in two patients. **(A)** Earliest ventricular activation precedes onset of the QRS complex by −47 ms in a 39-year-old man (Patient #16) with ventricular tachycardia (VT) originating within the pulmonary artery. The presystolic bipolar potential precedes the unipolar potential by −27 ms, and atrial potentials can be clearly recognized from the mapping catheter **(arrow)**. The amplitude of local ventricular bipolar potential is 0.65 mV. **(B)** Earliest ventricular activation precedes onset of the QRS complex by −48 ms in a 60-year-old woman with VT originating from the endocardial right ventricular outflow tract. The timing of the presystolic bipolar potential is nearly equal to the onset of the local unipolar potential. The atrial potential cannot be seen from the mapping catheter **(arrow)**. The amplitude of local ventricular bipolar potential is 3.68 mV. bi = bipolar signal; CS = coronary sinus electrogram; LCC = left coronary cusp electrogram; PA = pulmonary artery electrogram; RV-end-OT = endocardial right ventricular outflow tract electrogram; uni = unipolar signal.

origin was too close to the coronary artery to ablate, the site, nevertheless, does lie in close proximity to the coronary artery and the PA. We suggest that radiofrequency application should not be performed in patients in whom the distance between the ablation catheter tip and the coronary artery is within 5 mm. We believe that the delivered radiofrequency energy must be slightly lowered, and radiofrequency application must not be performed for an ex-

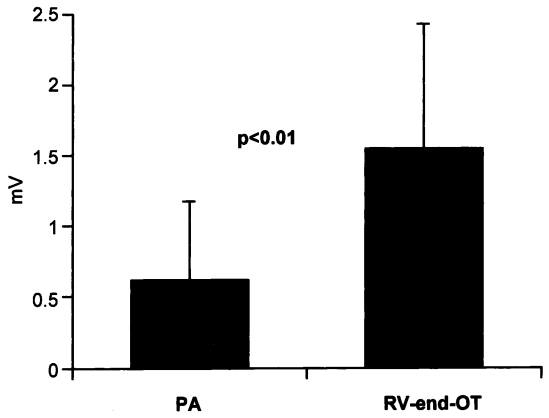

Figure 6. Comparison of intracardiac electrograms between the pulmonary artery (PA) group and the endocardial right ventricular outflow tract (RV-end-OT) group in terms of the amplitude of local ventricular bipolar potentials during sinus rhythm at the successful ablation site. The amplitude in the PA group is significantly lower than that in the RV-end-OT group.

tended time when applied within the PA, especially when pacing stimuli from a conventional pacing unit at maximal output (9.9 V/2.0 ms) cannot be captured. Thought should be given to the fact that the arrhythmia focus may be located at a different site if the arrhythmia cannot be stopped immediately. Timmermans et al. (14) explained that the site of VT origin within the PA may be in myocardial tissue in or around the PA. Ya et al. (17) reported that in rat, the distal myocardial boundary of the OT is not a stable landmark but moves proximally over the spiraling course of the pulmonary routes before the semilunar valves develop. Arita et al. (18) reported that in guinea pig, microscopical observation showed the myocardium within the pulmonary bulbus to be directly connected to the RV muscle. Although a better understanding of the origin of VT within the PA is needed, we also believe that the origin of arrhythmias within the PA may be related to the short myocardial sleeve extending from the RV to the PA. If the myocardial sleeve has enough length to infiltrate or completely surround the entire PA, VT originating within the PA may be eliminated by ablating not only the PA itself but the sleeve from below the pulmonary valve, just at the entry into the RV or at the connection between the RV and the PA. However, we could not obtain a clear potential suggesting a connection between the RV and the PA in any PA group patient, but we did obtain a low, dull potential preceding a ventricular potential during VPCs in two PA group patients. The reason for the potential not being recorded may be the small amount of muscle in the PA. Pace mapping at successful ablation site could be performed in only 37% of the PA group patients by conventional pacing. The reason may be also related to the fact that the sleeve may be short, or that it may be partially connected to the perimeter of the PA. In the present study, the successful ablation sites in the PA group were located 1.18 ± 0.43 cm above the pulmonary valve. The myocardial sleeve between the RV and the PA may be quite different from that between the pulmonary veins and the left atrium. Therefore, we currently recommend focal ablation for VT

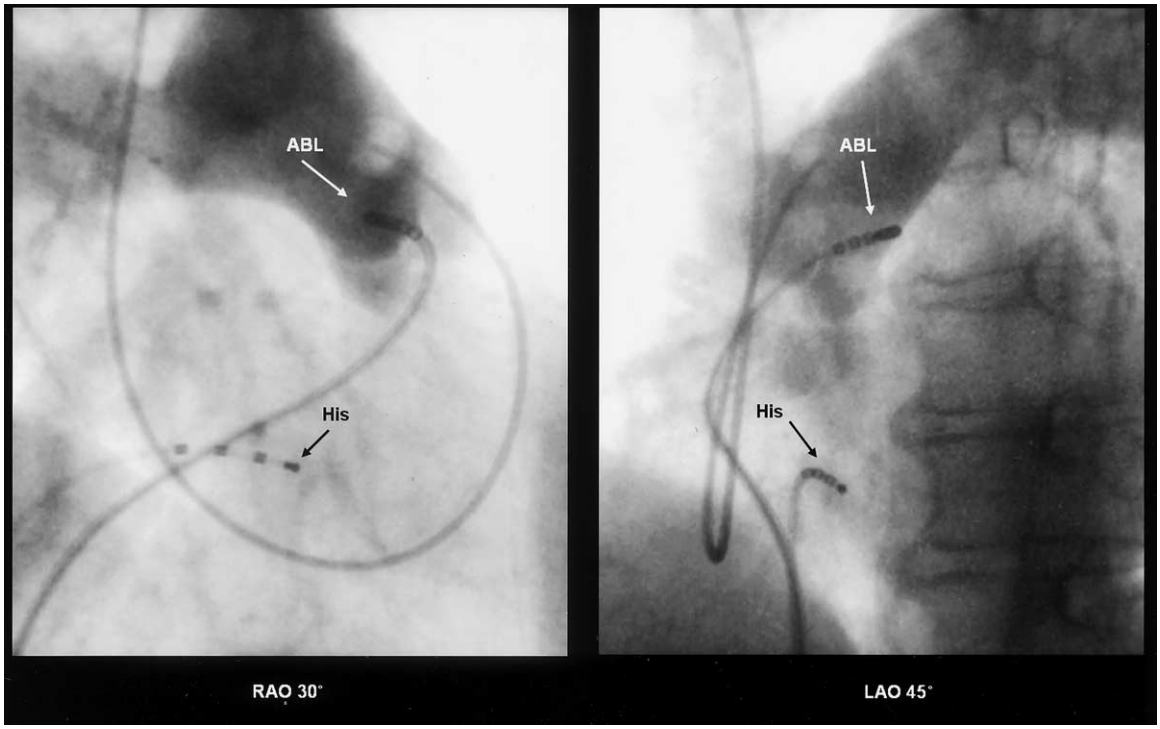

Figure 7. Right (30°) and left (45°) anterior oblique (RAO and LAO, respectively) radiographic views of the ablation catheter (ABL) within the pulmonary artery. Pulmonary arteriogram reveals that the catheter is positioned at the posteroseptal portion of right ventricular outflow tract, which is above the pulmonary valve. Successful radiofrequency application was performed at this site. His = catheter at the region of the His bundle.

originating within the PA. Further experience with the myocardial sleeve is required.

Study limitations. In this study, VT or VPCs originated within the PA in 24 of 148 patients (16%) with LBBB morphology and inferior axis, including 5 patients who had a previously failed treatment. As two previous studies reported (13,14), it is rare that VT originates within the PA. However, there may be some cases in which VT originating within the PA was regarded as originating from the RV-end-OT because the RV-end-OT was next to the PA. Intracardiac echography in the RV-end-OT and the PA could be helpful in the precise localization of the site of VT origin. The limitation of this study is the absence of intracardiac echography to verify the location of the ablation site. Instead, we used pulmonary arteriogram to clearly visualize in all patients the exact ablation site in relation to the catheter and pulmonary valve. Patients were excluded if the distance between the catheter and the pulmonary valve could not be clearly visualized.

Conclusions. Ventricular tachycardia originating within the PA has several different electrocardiographic and electrophysiologic characteristics from that originating from the RV-end-OT. Radiofrequency catheter ablation within the PA should be considered when the ECG shows the characteristics of VT originating within the PA. It is very important to perform pace mapping when the target site of RFCA is suspected to be within the PA, and, if pacing stimuli from a conventional pacing unit cannot be captured, or if on intracardiac electrograms, a low-voltage atrial or local ventricular potential of less than 1-mV amplitude can be recorded from the catheter, the catheter may be located within the PA. Heightened attention must be paid to the distance between the catheter and the coronary artery and to the temperature of the catheter tip when radiofrequency application is performed within the PA.

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