Radiofrequency Catheter Ablation of Premature Ventricular Complexes From Right Ventricular Outflow Tract Improves Left Ventricular Dilation and Clinical Status in Patients Without Structural Heart Disease

Masao Takemoto, MD, Hitoshi Yoshimura, MD, Yurika Ohba, MD, Yasuharu Matsumoto, MD, Umpei Yamamoto, MD, Masahiro Mohri, MD, Hideo Yamamoto, MD, Hideki Origuchi, MD

Kitakyushu, Japan

OBJECTIVES	The present study evaluated clinical benefits of radiofrequency catheter ablation (RFA) for premature ventricular complexes from right ventricular outflow tract (RVOT-PVC) in patients without structural heart disease.
BACKGROUND	It is unknown whether PVC causes left ventricular (LV) dilation, which is a well-recognized precursor of LV dysfunction and heart failure, and whether eliminating PVC by RFA produces clinical benefits in patients with RVOT-PVC.
METHODS	Frequency of PVC per total heart beats by 24-h Holter monitoring, left ventricular ejection fraction (LVEF), left ventricular end-diastolic internal dimension (LVDd), mitral regurgitation (MR) by echocardiogram, cardiothoracic ratio (CTR) by chest radiogram, and New York Heart Association (NYHA) functional class of 40 patients with RVOT-PVC without structural heart disease were evaluated before and 6 to 12 months after RFA.
RESULTS	Before RFA, a subgroup of patients with frequent (>20%) PVC demonstrated significantly enlarged LVDd and CTR, reduced LVEF, increased MR, and deteriorated NYHA functional class as compared to the subgroup with rare (<20%) PVC (54 ± 1 mm vs. 45 ± 1 mm, 52 ± 2% vs. 46 ± 1%, 66 ± 2% vs. 73 ± 2%, 1.2 ± 0.2 degree vs. 0.4 ± 0.1 degree, and 1.8 ± 0.2 vs. 1.3 ± 0.1, respectively; p < 0.05). Furthermore, ablating RVOT-PVC readily produced the improvement of all these abnormalities (47 ± 1 mm, 41 ± 1%, 72 ± 2%, 0.3 ± 0.1 degree, and 1.0 ± 0.0, respectively; p < 0.05 compared with before RFA).
CONCLUSIONS	These findings suggest that frequent (>20%) RVOT-PVC may be a possible cause of LV dysfunction and/or heart failure, and RFA produces clinical benefits in these patients. (J Am Coll Cardiol 2005;45:1259–65) © 2005 by the American College of Cardiology Foundation

Isolated premature ventricular complexes (PVC) are the most common arrhythmias observed in patients without structural heart disease (1). It has been recently reported that frequent PVC caused left ventricular (LV) dysfunction that can be reversed by suppression of PVC with antiarrhythmic agents (2,3) or radiofrequency catheter ablation (RFA) (4,5) in patients with dilated cardiomyopathy. It is

uncertain, however, whether frequent PVC causes LV dilation and dysfunction even in patients with no evidence of structural heart disease and, if so, whether suppression of PVC improves these changes. In recent years, RFA has proven to be a safe and successful therapy for arrhythmias (6,7). The purpose of this study was two-fold: 1) to examine whether frequent premature ventricular complexes from

right ventricular outflow tract (RVOT-PVC) without structural heart disease may cause LV dilation, which is a well-recognized precursor of LV dysfunction and congestive heart failure (8); 2) to evaluate the role of ablating RVOT-PVC per RFA on cardiac function in patients with depressed cardiac function.

METHODS

Study population and laboratory analysis. From 1994 to 2004, 45 consecutive patients (10 males and 35 females with mean age of 50 ± 2 years and body surface area of 1.57 ± 0.02 m²) with monomorphic RVOT-PVC and no evidence of underlying structural heart disease underwent RFA at our hospital. All patients had their history recorded, and underwent physical examination, laboratory analysis, chest radiogram, 12-lead electrocardiogram, 24-h Holter monitoring, M-mode, two-dimensional, and Doppler echocardiogram (SONOS 2000, Hewlett-Packard, San Diego, California, and SEQUOIA 512, Siemens, Erlangen, Germany) on admission or within at least 1 month before admission, and

---

Abbreviations and Acronyms

LBBB = left bundle branch block
LV   = left ventricle/ventricular
LVDd = left ventricular end-diastolic internal dimension
LVDs = left ventricular end-systolic internal dimension
LVEF = left ventricular ejection fraction
NYHA = New York Heart Association
PVC   = premature ventricular complexes
RFA   = radiofrequency catheter ablation
RV   = right ventricle/ventricular
RVOT = right ventricular outflow tract
%PVC = frequency of premature ventricular complexes

---

6 to 12 months after RFA (average 8 ± 1 month). The 24-h Holter monitoring and echocardiogram were performed on the same day. All patients also took routine echocardiogram on the next day of RFA to evaluate the possible procedure-related complications. We evaluated the chamber size of LV and right ventricle (RV), and degree of mitral regurgitation at parasternal long-axis or apical four-chamber view, respectively. Left ventricular ejection fraction (LVEF) was calculated by the Teichholz method. All values of echocardiogram were recorded during sinus rhythm, but not at the PVC beat, nor at the post-PVC beat. All patients also underwent the exercise electrocardiogram testing, cardiac catheterization with coronary angiography, and/or 201-thallium scintigraphy. These examinations yielded no evidence of clinically overt structural heart disease, including coronary artery disease, valvular heart disease, congenital heart disease, LV hypertrophy, and RV abnormalities in all patients. Brain natriuretic peptide of five recent patients was evaluated on admission and six months after RFA.

Definition of RVOT-PVC. Right ventricular outflow tract-PVC was defined as a characteristic electrocardiographic appearance of a left bundle branch block (LBBB) contour in V_1 and an inferior axis in the frontal plane. Ventricular tachycardia was defined with standard electrocardiographic criteria of at least five consecutive PVC at a rate >120 beats/min. Patients with ventricular tachycardia and atrial tachyarrhythmia including atrial fibrillation, atrial flutter, atrial tachycardia, and paroxysmal supraventricular tachycardia were excluded in this study because they may cause tachycardia-induced LV dilation (9,10). The region of the LV outflow tract was not an origin of PVC of all patients in this study.

Mapping and catheter ablation procedure. All procedures were performed after written informed consent was obtained. The patients were studied in the fasting state without sedation. Antiarrhythmic drugs were discontinued for at least six half-lives before the procedure. Under local anesthesia, a 7-F deflectable quadripolar ablation catheter (Boston Scientific EP Technologies, Natick, Massachusetts) with a 4-mm-tip electrode was introduced percutaneously into the RV. Based on the 12-surface-lead electrocardiogram with spontaneous RVOT-PVC, pace mapping was done by using bipolar pacing between the distal pair of the electrodes with a stimulation pulse width of 2 ms. If the culprit PVC were not found during the procedure, isoproterenol administration and/or programmed electrical stimulation with digital stimulator (Cardiac Stimulator, Nihon Kohden Co., Tokyo, Japan) was performed to induce the culprit PVC as previously described (6). An optimal pace map was defined as a match of all 12 surface leads by comparing the R/S ratio and subtle notching in the QRS complex during pacing. An identical match was necessary in at least 11 of 12 leads. The RFA was performed based on an optimal pace map for 60 to 90 s with a preset temperature of 50 to 60°C and a power limit of 50 W. A successful ablation was defined as the no recurrence and non-inducibility of culprit PVC with and without isoproterenol administration at the rate of 0.2 to 0.6 μg/min and/or programmed electrical stimulation during at least 30 min after ablation. All 12 surface electrocardiograms and the bipolar intracardiac electrograms (filtered at 30 to 400 Hz) were recorded and stored by using a 48-channel acquisition system (CardioLabEP, Prucka Engineering Inc., Houston, Texas). During the procedure, intravenous heparin was given as a 100 IU/kg bolus dose followed by boluses of 1,000 IU every hour. All patients received oral antiplatelets for eight weeks after RFA, but no antiarrhythmic drugs. Procedural success was defined as no recurrence of culprit PVC within 72 h after procedure under electrocardiogram monitoring.

Statistical analysis. Numerical results are expressed in the text as the mean ± standard deviation. Paired data were compared by Student *t* tests. The differences between the continuous variables before and 1 day or 6 to 12 months after RFA in each group, or between the lower, middle, and upper group in each period, were compared by using a repeated-measures analysis of variance followed by Bonferroni's test for multiple comparisons. Correlation between the two parameters was determined by simple linear regression analysis. A $p < 0.05$ was considered to indicate statistical significance.

RESULTS

Patient characteristics. An RFA procedure for RVOT-PVC was performed in 45 patients. Because five patients (11%) were lost to follow-up, they were excluded from the statistical analysis. Procedural success was achieved in 37 (93%) of the patients. One patient (3%) suffered from a procedure-related complication with femoral arteriovenous fistula. During follow-up, recurrence of culprit RVOT-PVC was observed in one patient (3%), who underwent repeated RFA with successful result. Relationships between the left ventricular end-diastolic internal dimension (LVDd) and the frequency of PVC (%PVC) by 24-h Holter monitoring of 40 patients before RFA were examined (Fig. 1). The %PVC was calculated as: 100·[number of PVC/number of total heart beats per 24 h]. A significant correlation was found between the LVDd and %PVC (y = 45.8 + 0.254x, r = 0.56, $p < 0.01$), indicating that frequent
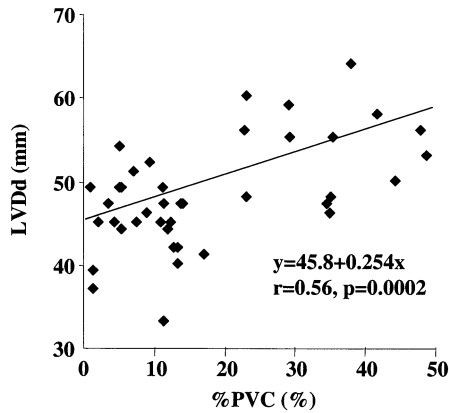
Figure 1. Scatter plots showing the relationship between the left ventricular end-diastolic internal dimension (LVDd) and frequency of premature ventricular complex (%PVC) (n = 40).

PVC might cause LV dilation. Patients (n = 40) were divided into three groups (the lower [<10%; n = 14], middle [10% to 20%; n = 12], and upper [>20%; n = 14] group) according to the tertiles of %PVC. Baseline characteristics of the patients are shown in Table 1. There was no statistical difference in body surface area, hemodynamic variables including systolic and diastolic blood pressure and resting heart rate, or the prevalence of diabetes (7%, 8%, and 7%, respectively) or hypertension (21%, 16%, and 21%, respectively) between the three groups. The mean patients' age was slightly older in the middle and upper groups than the lower group, but the differences were not significant between the three groups. Symptom duration was significantly shorter in the middle and upper groups compared with the lower group ($p < 0.05$ and 0.05, respectively). No patients had chronic obstructive pulmonary disease, renal dysfunction (serum creatinine more than 1.5 mg/dl), liver disease (liver enzyme values in excess of $2 \times$ normal), or underlying malignant disease.

24-h Holter monitoring. Table 2 summarizes analysis of 24-h Holter monitoring. The number of total heart beats was not statistically different before or after RFA between the three groups. However, RFA produced a significant

decrease in the number of total heart beats in the upper group ($p < 0.05$), but not in the lower or middle groups. Radiofrequency ablation significantly reduced the number of total PVC ($p < 0.01$ for all conditions) and %PVC ($p < 0.01$ for all conditions) with no evidence of the recurrence of culprit PVC in all three groups. There was no statistical difference of the number of total PVC and %PVC after RFA between the three groups. There was no statistical difference in the frequency of premature atrial complexes before and after RFA between the three groups (data not shown).

Echocardiogram and chest radiography. Figure 2 summarizes the results of the analysis of echocardiograms (Figs. 2A to 2E) and chest radiograms (Fig. 2F). Before RFA, a subgroup of patients in the upper group demonstrated significantly reduced LVEF, enlarged LVDd, and left ventricular end-systolic internal dimension (LVDs), and increased degree of mitral regurgitation and cardiothoracic ratio compared with the lower or middle groups ($p < 0.05$ and 0.05, respectively) (Figs. 2A to 2D and 2F). There was no statistical difference in these parameters between the lower and middle groups. The examination by echocardiogram one day after RFA revealed the same results as those before RFA. However, at the examination 6 to 12 months after RFA, all of these abnormalities seen in the upper group before RFA were completely reversed (Figs. 2A to 2D and 2F), and there was no statistical difference in these parameters between the three groups. There was no statistical difference in these parameters during follow-up in the lower or middle groups. There was no evidence of RV abnormalities in all patients (Fig. 2E), indicating that arrhythmogenic RV cardiomyopathy was unlikely. There was no statistical difference in any of the analyzed parameters between the groups before and after RFA.

New York Heart Association (NYHA) functional class and serum brain natriuretic peptide concentration. The NYHA functional class was evaluated in all patients on admission and 6 to 12 months after RFA. Before ablation, NYHA functional class was demonstrated to be significantly

Table 1. Patients' Characteristics

Group	Lower (<10%)	Middle (10%–20%)	Upper (>20%)
Number of patients (40 patients)	14	12	14
Successful ablation (37 patients; 93%)	14 (100%)	10 (83%)	13 (93%)
Male:female (8:32)	3:11	4:8	1:13
Age (yrs)	46 ± 3	54 ± 4	52 ± 4
Body surface area (m²)	1.64 ± 0.04	1.55 ± 0.03	1.56 ± 0.03
Symptom duration (months)	46 ± 9	12 ± 5*	15 ± 7*
Hemodynamic variables on admission			
Systolic BP (mm Hg)	120 ± 5	124 ± 5	119 ± 4
Diastolic BP (mm Hg)	69 ± 3	69 ± 2	66 ± 2
Resting heart rate (beats/min)	70 ± 4	71 ± 4	77 ± 3
Hypertension	3 (21%)	2 (16%)	3 (21%)
Diabetes mellitus	1 (7%)	1 (8%)	1 (7%)

*$p < 0.05$ versus the lower group.
BP = blood pressure.

Table 2. 24-h Holter Monitoring Analysis in Patients With Successful Procedure

Group	Lower (<10%)	Middle (10%–20%)	Upper (>20%)
Number of patients	14	10	13
Before RFA			
Total HB ($\times 10^3$ beats/day)	105 ± 4	105 ± 4	113 ± 4
Total PVC ($\times 10^2$ beats/day)	46 ± 9	133 ± 11*	381 ± 34*†
%PVC (%)	4 ± 1	13 ± 1*	34 ± 3*†
After RFA			
Total HB ($\times 10^3$ beats/day)	103 ± 5	104 ± 4	98 ± 5‡
Total PVC ($\times 10^2$ beats/day)	0.5 ± 0.3‡	11 ± 8‡	13 ± 9‡
%PVC (%)	0.05 ± 0.04‡	1.9 ± 1.0‡	1.3 ± 0.9‡

*$p < 0.01$ versus the lower group; †$p < 0.01$ versus the middle group; ‡$p < 0.01$ versus before radiofrequency catheter ablation.
HB = heart beats; PVC = premature ventricular complexes; RFA = radiofrequency catheter ablation; %PVC = frequency of PVC.

worse in accordance with %PVC ($p < 0.05$) (Fig. 3). Radiofrequency ablation significantly improved NYHA functional class in each group compared with before intervention. The serum brain natriuretic peptide concentration of five recent patients was evaluated; one in the lower group, one in the middle group, and three in the upper group, respectively. The serum brain natriuretic peptide level in the upper group was elevated to 56 ± 3 pg/dl before RFA. However, it significantly decreased to 3.7 ± 0.3 pg/dl six months after RFA ($p < 0.01$). The value of two patients in the lower and middle groups did not elevate before and after RFA (data not shown).

Patients symptoms and medications. On admission, all patients had PVC-associated symptoms (Table 3). Of 40 patients, 26 patients (65%) had been taking antiarrhythmic agents before admission, such as beta-blocker, bepridil, class Ia, Ib, and/or Ic agents of Vaughan Williams classification (Table 3). However, these agents were not sufficiently effective to eliminate PVC-associated symptoms. No patients had been on amiodarone or sotalol. All of the patients with successful procedure reported the absence of PVC-associated symptoms and could discontinue antiarrhythmic agents after RFA. The antihypertensive or antidiabetic agents, such as beta-blockers, calcium-channel blockers, angiotensin-converting enzyme inhibitors, or sulfonylurea, were continued in patients with hypertension or diabetes before and after RFA.

Outcome of patients with unsuccessful RFA. Of 40 patients, three patients were procedurally unsuccessful in this study; one in the upper group, and two in the middle group. Table 4 summarizes the results of the analysis of echocardiograms, chest radiograms, and NYHA functional class of these patients. All three patients took medications, which seemed to be partially effective in the patient in the upper group. All of these factors in two patients of the middle group did not further deteriorate during follow-up.
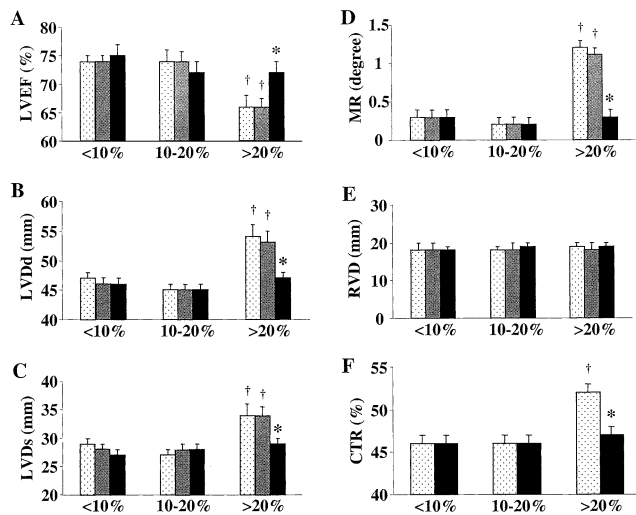
Figure 2. The analysis of left ventricular ejection fraction (LVEF) **(A)**, left ventricular end-diastolic internal dimension (LVDd) **(B)**, left ventricular end-systolic internal dimension (LVDs) **(C)**, degree of mitral regurgitation (MR) **(D)**, and right ventricular internal dimension (RVD) **(E)** by echocardiogram, and cardiothoracic ratio (CTR) **(F)** by chest radiography before **(dotted bars)** and 1 day **(gray bars)** or 6 to 12 months after **(solid bars)** radiofrequency catheter ablation (RFA). Before RFA and one day after RFA, a subgroup of patients with frequent (>20%) premature ventricular complexes (PVC) demonstrated significantly reduced LVEF, enlarged LVDd and LVDs, and increased MR and CTR as compared to the subgroup with rare (<20%) PVC. However, all of these abnormalities seen in the upper group before RFA were completely reversed 6 to 12 months after RFA. There was no evidence of right ventricular abnormalities in any patient. *$p < 0.05$ versus before and one day after RFA in each group; †$p < 0.05$ versus the lower and middle groups in each period. Values are mean ± SD.
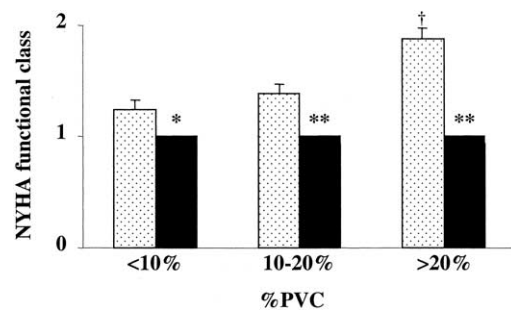
Figure 3. The analysis of New York Heart Association (NYHA) functional class before **(dotted bars)** and 6 to 12 months after **(solid bars)** radiofrequency catheter ablation (RFA). Before ablation, NYHA functional class was significantly worse in accordance with frequency of premature ventricular complex (%PVC); RFA significantly improved NYHA functional class in each group compared with before intervention. **$p < 0.01$, *$p < 0.05$ versus before RFA in each group. †$p < 0.05$ versus the lower and middle groups in each period. Values are mean ± SD.

Table 3. Patients Symptoms and Medications Before Radiofrequency Catheter Ablation

Group	Lower (<10%; n = 14)	Middle (10%–20%; n = 12)	Upper (>20%; n = 14)
Symptoms			
Palpitation	6 (43%)	7 (58%)	6 (43%)
General fatigue	2 (14%)	1 (8%)	3 (21%)
Chest discomfort	0 (0%)	1 (8%)	2 (14%)
Fainting	6 (43%)	3 (25%)	3 (21%)
Medications			
Procainamide	1 (7%)	0 (0%)	0 (0%)
Mexiletine	4 (28%)	2 (16%)	5 (36%)
Pilsicainide	1 (7%)	1 (8%)	1 (7%)
Beta-blocker	7 (50%)	5 (42%)	9 (64%)
Bepridil	0 (0%)	0 (0%)	2 (14%)
Calcium channel blocker	1 (7%)	1 (8%)	2 (14%)
ACE inhibitor	0 (0%)	1 (8%)	2 (14%)
Sulfonylurea	1 (7%)	1 (8%)	1 (7%)
No drugs	5 (36%)	6 (50%)	3 (21%)

ACE = angiotensin-converting enzyme.

Interestingly, the LVDd, LVDs, LVEF, degree of mitral regurgitation by echocardiogram, and cardiothoracic ratio by chest radiogram of the patient with unsuccessful RFA in the upper group still deteriorated during follow-up.

DISCUSSION

Isolated PVC are the most common arrhythmia that physicians often see during patient examination (1). The presence of PVC has been found to be an independent risk factor for sudden death (11), especially in patients after myocardial infarction (12,13). Many patients with PVC often experience disabling symptoms and sometimes need long-term antiarrhythmic medications. However, the latter are often ineffective and may have adverse effects, such as the increasing mortality, probably due to their proarrhythmic effects, even though markedly suppressing the occurrence of PVC (14–16). It has been reported that RFA produces clinical benefits to the patients with ventricular tachycardia (6,7,17), and some case reports have shown that the dilated cardiomyopathy patients with PVC-depressed cardiac function and successful RFA of these arrhythmias

improved LV function (4,5). However, these phenomena have never been documented in the large series of patients with PVC without underlying heart disease.

An RFA procedure for RVOT-PVC was performed in 45 patients, mostly females in this study. The reasons for a much higher prevalence of women may be that the study population was limited to the patients with disabling PVC-associated symptoms. This retrospective study provides evidence in support of the concept that some of the patients with frequent RVOT-PVC, more than 20% per total heart beats, without overt underlying structural heart disease were associated with LV dilation (Figs. 1, 2B, 2C, and 2F), which was known to be a risk factor for LV dysfunction and congestive heart failure (8). Before RFA, LVEF, degree of mitral regurgitation, and NYHA functional class in these patients were worse as compared to the patients with rare RVOT-PVC, <20% per total heart beats (Figs. 2A, 2D, and 3). These abnormalities were still present one day after successful RFA. Furthermore, these parameters of the patient with unsuccessful RFA with frequent PVC, but not with rare PVC, still deteriorated during follow-up. These

Table 4. Follow-Up Data of Patients With Unsuccessful Procedure

Patients	1	2	3
Group	Upper	Middle	Middle
Echocardiogram			
LVDd (mm)	60 (58)	49 (49)	45 (44)
LVDs (mm)	45 (43)	31 (31)	30 (29)
LVEF (%)	56 (59)	67 (66)	67 (65)
Mitral regurgitation (degree)	2 (2)	0 (0)	0 (0)
Chest radiogram			
Cardiothoracic ratio (%)	57 (57)	44 (44)	47 (46)
NYHA functional class	1 (1)	1 (1)	1 (1)
%PVC (%)	19.4 (41.0)	9.5 (10.9)	10.0 (11.7)
Medications	Beta-blocker, bepridil	Beta-blocker	Beta-blocker, mexiletine

Values in the parentheses are data on admission.

LVDd = left ventricular end-diastolic dimension; LVDs = left ventricular end-systolic dimension; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; %PVC = frequency of premature ventricular complexes.

findings may indicate that the frequent RVOT-PVC, more than 20% per total heart beats, may be an important and independent risk factor on progressing LV dysfunction and heart failure. The findings of elevated serum brain natriuretic peptide level and total heart rate (Table 2) before RFA in the upper group may also support the possibility of heart failure progression. When the patients were divided into three groups according to the tertiles of the absolute number of PVC before RFA, the number of PVC more than 20,000 beats per day was shown to be at risk (data not shown). More importantly, ablating RVOT-PVC readily produced the improvement of these abnormalities without adverse effects. Attenuation of progressive LV dilation has proven to reduce the risk of cardiovascular events (8,10), and RFA is known to be safe and effective for the treatment of arrhythmias as compared with medical therapy (7,14). Thus, RFA may be considered as the first choice of therapy for patients with frequent RVOT-PVC with LV dilation.

Although the mechanism(s) of LV dilation in those with RVOT-PVC is not completely elucidated, several potential mechanisms can be raised. Most of the patients with RVOT-PVC demonstrate LBBB and inferior axis morphology, and wide QRS complex generally >120 ms (1). It has been reported that LBBB causes asynchronous myocardial activation, which progresses into LV diastolic dysfunction and mitral regurgitation leading to decreased LV stroke volume (18,19). Recent clinical trials revealed that permanent RV apical pacing, which demonstrated LBBB promoted heart failure progression (20–23). Furthermore, both permanent RV apical pacing and RVOT pacing could not improve quality of life or give other clinical benefits in patients with permanent pacemaker implantation (24,25). Thus, LBBB-induced asynergic LV wall motion caused by frequent RVOT-PVC might be one of the important factors of progressing LV dilation and reduced LVEF observed in this study. Moreover, it has been reported that LV contraction usually moves from the apical to the basal portion with LV apex rotation in accordance with squeezing effect (4) and torsional deformation (26,27) during sinus rhythm. Because the direction of LV contraction is reversed during RVOT-PVC, these effects may cause disruption and further progression of asynergic LV wall motion. Recent clinical studies reported that cardiac resynchronization resulted in significant clinical improvements in patients who had chronic heart failure and wide QRS interval (28,29). In view of these findings, eliminating RVOT-PVC by RFA may be a subset of resynchronization therapy.

Limitations of the study. Our study is limited by its retrospective design and relatively short follow-up period. Serum brain natriuretic peptide concentration was not evaluated in all patients. Although the differences in LV dilation, LVEF, degree of mitral regurgitation, and NYHA functional class observed in patients with frequent PVC after RFA were statistically significant, they were relatively mild. Whether our results can safely be extrapolated to the patients with much larger LV and reduced LVEF should be

determined in further studies. Recent case reports showed that a similar beneficial effect of RFA on LV function was seen in patients with a more severe form of heart failure (4,5), supporting our hypothesis.

Conclusions. A subgroup of patients with frequent (>20%) PVC demonstrated significantly enlarged LVDd, reduced LVEF, increased mitral regurgitation, and deteriorated NYHA functional class as compared to the subgroup with rare (<20%) PVC. Furthermore, the ablation of these arrhythmias was associated with the normalization of these abnormalities without adverse effects. These findings suggest that RVOT-PVC may be a possible cause of LV dysfunction and/or heart failure. Thus, the physicians should be aware of the condition when examining a patient with LV dilation and/or reduced LVEF in the presence of frequent RVOT-PVC because it may be at least one important risk factor on progressing LV dysfunction and heart failure. Radiofrequency catheter ablation may be considered as the first choice of therapy for those patients.

REFERENCES

1. Wang K, Hodges M. The premature ventricular complex as a diagnostic aid. Ann Intern Med 1992;117:766–70.
2. Duffee DF, Shen WK, Smith HC. Suppression of frequent premature ventricular contractions and improvement of left ventricular function in patients with presumed idiopathic dilated cardiomyopathy. Mayo Clin Proc 1998;73:430–3.
3. Massie BM, Fisher SG, Radford M, et al. Effect of amiodarone on clinical status and left ventricular function in patients with congestive heart failure. CHF-STAT Investigators. Circulation 1996;93:2128–34.
4. Shiraishi H, Ishibashi K, Urao N, et al. A case of cardiomyopathy induced by premature ventricular complexes. Circ J 2002;66:1065–7.
5. Chugh SS, Shen WK, Luria DM, Smith HC. First evidence of premature ventricular complex-induced cardiomyopathy: a potentially reversible cause of heart failure. J Cardiovasc Electrophysiol 2000;11: 328–9.
6. Timmermans C, Rodriguez LM, Crijns HJ, Moorman AF, Wellens HJ. Idiopathic left bundle-branch block-shaped ventricular tachycardia may originate above the pulmonary valve. Circulation 2003;108: 1960–7.
7. Krittayaphong R, Sriratanasathavorn C, Bhuripanyo K, et al. One-year outcome after radiofrequency catheter ablation of symptomatic ventricular arrhythmia from right ventricular outflow tract. Am J Cardiol 2002;89:1269–74.
8. Vasan RS, Larson MG, Benjamin EJ, Evans JC, Levy D. Left ventricular dilatation and the risk of congestive heart failure in people without myocardial infarction. N Engl J Med 1997;336:1350–5.

9. Shinbane JS, Wood MA, Jensen DN, Ellenbogen KA, Fitzpatrick AP, Scheinman MM. Tachycardia-induced cardiomyopathy: a review of animal models and clinical studies. J Am Coll Cardiol 1997;29:709–15.

10. Grogan M, Smith HC, Gersh BJ, Wood DL. Left ventricular dysfunction due to atrial fibrillation in patients initially believed to have idiopathic dilated cardiomyopathy. Am J Cardiol 1992;69: 1570–3.

11. Bikkina M, Larson MG, Levy D. Prognostic implications of asymptomatic ventricular arrhythmias: the Framingham Heart study. Ann Intern Med 1992;117:990–6.

12. Lown B, Wolf M. Approaches to sudden death from coronary heart disease. Circulation 1971;44:130–42.

13. Hallstrom AP, Bigger JT Jr., Roden D, et al. Prognostic significance of ventricular premature depolarizations measured 1 year after myocardial infarction in patients with early postinfarction asymptomatic ventricular arrhythmia. J Am Coll Cardiol 1992;20:259–64.

14. The Cardiac Arrhythmia Suppression Trial (CAST) Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. N Engl J Med 1989;321:406–12.

15. Teo KK, Yusuf S, Furberg CD. Effects of prophylactic antiarrhythmic drug therapy in acute myocardial infarction. An overview of results from randomized controlled trials. JAMA 1993;270:1589–95.

16. Akhtar M, Breithardt G, Camm AJ, et al. CAST and beyond. Implications of the Cardiac Arrhythmia Suppression trial. Task Force of the Working Group on Arrhythmias of the European Society of Cardiology. Circulation 1990;81:1123–7.

17. Zalzstein E, Wagshal A, Zucker N, Levitas A, Ovsyshcher IE, Katz A. Ablation therapy of tachycardia-related cardiomyopathy. Isr Med Assoc J 2003;5:64–5.

18. Littmann L, Symanski JD. Hemodynamic implications of left bundle branch block. J Electrocardiol 2000;33 Suppl:115–21.

19. Xiao HB, Lee CH, Gibson DG. Effect of left bundle branch block on diastolic function in dilated cardiomyopathy. Br Heart J 1991;66: 443–7.

20. Lamas GA, Lee KL, Sweeney MO, et al. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. N Engl J Med 2002;346:1854–62.

21. Andersen HR, Nielsen JC, Thomsen PE, et al. Long-term follow-up of patients from a randomised trial of atrial versus ventricular pacing for sick-sinus syndrome. Lancet 1997;350:1210–6.

22. Andersen HR, Nielsen JC, Thomsen PE, Goldman L. Baseline QRS duration >120 milliseconds and cumulative percent time ventricular paced predicts increased risk of heart failure, stroke, and death in DDDR-paced patients with sick sinus syndrome in MOST (abstr). Pacing Clin Electrophysiol 2002;25:690.

23. Wilkoff BL, Cook JR, Epstein AE, et al. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial. JAMA 2002;288:3115–23.

24. Victor F, Leclercq C, Mabo P, et al. Optimal right ventricular pacing site in chronically implanted patients: a prospective randomized crossover comparison of apical and outflow tract pacing. J Am Coll Cardiol 1999;33:311–6.

25. Stambler BS, Ellenbogen K, Zhang X, et al. Right ventricular outflow versus apical pacing in pacemaker patients with congestive heart failure and atrial fibrillation. J Cardiovasc Electrophysiol 2003;14:1180–6.

26. Yun KL, Miller DC. Torsional deformation of the left ventricle. J Heart Valve Dis 1995;4 Suppl 2:S214–22.

27. Hansen DE, Daughters GT 2nd, Alderman EL, Ingels NB Jr., Miller DC. Torsional deformation of the left ventricular midwall in human hearts with intramyocardial markers: regional heterogeneity and sensitivity to the inotropic effects of abrupt rate changes. Circ Res 1988;62:941–52.

28. Abraham WT, Fisher WG, Smith AL, et al. Cardiac resynchronization in chronic heart failure. N Engl J Med 2002;346:1845–53.

29. St. John Sutton MG, Plappert T, Abraham WT, et al. Effect of cardiac resynchronization therapy on left ventricular size and function in chronic heart failure. Circulation 2003;107:1985–90.