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Impact of Radiofrequency Ablation of Frequent Post-Infarction Premature Ventricular Complexes on Left Ventricular Ejection Fraction

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Abstract

Background—Frequent idiopathic premature ventricular complexes (PVC) are associated with a reversible form of cardiomyopathy. The effect of frequent PVCs on left ventricular function has not been evaluated in post-infarction patients.

Objective—To evaluate the value of post-infarction PVC ablation and possible determinants of a reversible cardiomyopathy.

Methods—Thirty consecutive patients (24 men, age 61 ± 12 , LVEF 0.36 ± 0.12) with remote myocardial infarction referred for ICD implantation for primary prevention of sudden death or for management of symptomatic ventricular tachycardia or PVCs were evaluated. Fifteen patients with a high PVC burden ($\geq 5\%$ of all QRS complexes on 24-hour Holter) underwent mapping and ablation of PVCs before ICD implantation. The remaining 15 patients served as a control group. LVEF was assessed by echocardiography, and scar burden was assessed by cardiac MRI with delayed enhancement (DE-MRI) in both groups.

Results—PVC ablation was successful in 15/15 patients and reduced the mean PVC burden from $22 \pm 12\%$ to $2.6 \pm 5.0\%$ ($p < 0.001$). Following the procedure, LVEF increased significantly from 0.38 ± 0.10 to 0.51 ± 0.09 in the PVC ablation group ($p = 0.0001$). In the control group, LVEF remained unchanged within the same time frame (0.34 ± 0.14 vs. 0.33 ± 0.15 ; $p = 0.6$). Patients with frequent PVCs had a significantly smaller scar burden by DE-MRI compared to control patients. Five of the patients with frequent PVCs underwent ICD implantation.

Conclusion—Post-infarction patients with frequent PVCs may have a reversible form of cardiomyopathy. DE-MRI may identify patients in whom the LVEF may improve after ablation of frequent PVCs.

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Keywords

catheter ablation; left ventricular ejection fraction; magnetic resonance imaging; myocardial infarction; premature ventricular complexes

Introduction

Idiopathic premature ventricular complexes (PVC) are usually associated with a benign course from the standpoint of arrhythmic death (1), but may result in a cardiomyopathy that is reversible by radiofrequency ablation (RFA) of the PVCs (2,3). Whether a reversible form of cardiomyopathy also occurs in patients with frequent PVCs who have had a myocardial infarction (MI) is unclear.

The purpose of this study was to determine whether frequent PVCs in patients with prior MI cause a reversible cardiomyopathy. In addition, the value of cardiac magnetic resonance imaging with delayed enhancement (DE-MRI) for identifying patients with a reversible cardiomyopathy was investigated.

Methods

The protocol was approved by the Institutional Review Board at the University of Michigan.

Characteristics of Subjects (Table 1)

The subjects of this study were 30 consecutive patients (24 men, mean age 61 ± 12 years, mean left ventricular ejection fraction (LVEF) 0.36 ± 0.12) with a remote MI (mean infarct age 10 ± 11 years) and no contraindication to MRI. None of these patients had an ICD. Patients were referred for ICD implantation for primary or secondary prevention of sudden death or for treatment of symptomatic ventricular tachycardia (VT) or PVCs. All patients were evaluated for ischemia a mean of 4.1 ± 4 months before intervention: 7 patients had a nuclear stress test, 4 patients had a stress echocardiogram, and 19 patients underwent coronary angiography. No patient was felt to require revascularization. All patients underwent 24-hour Holter monitoring, if possible with a 12-lead recording system, a mean of 58 ± 35 days before and 58 ± 36 days after the ablation procedure.

Fifteen of the 30 patients (12 men, mean age 59 ± 12 years, mean LVEF 0.38 ± 0.11) had frequent PVCs and were referred for symptomatic post-infarction PVCs ($n = 8$) or for an ICD (primary prevention, $n = 3$; secondary prevention, $n = 4$). All patients had a prior MI (mean infarct age 10 ± 13 years). The infarction was anterior in 4 patients and inferior in 11 patients. All patients had frequent PVCs, defined as a PVC burden $> 5\%$ of the total QRS complexes on a 24-hour Holter monitor. Nine patients had 2 or more PVC morphologies during the Holter monitoring (median = 2 PVC morphologies); the remaining patients had a single predominant PVC morphology. All patients underwent echocardiography and DE-MRI before the ablation procedure. Three patients were treated with antiarrhythmic drugs before the procedure (one patient with amiodarone, 1 with sotalolol, and 1 with intravenous amiodarone and lidocaine for recurrent VT). All 15 patients underwent mapping and ablation of PVCs.

The control group consisted of the other 15 post-infarction patients (12 men, mean age 64 ± 12 years, mean LVEF 0.34 ± 0.14) who did not have frequent PVCs and who did not undergo ablation. These patients were referred for ICD implantation for primary prevention ($n = 5$) or secondary prevention ($n = 10$) of sudden death. All patients had a prior MI (mean infarct age 10 ± 8 years). The infarction was anterior in 7 patients, inferior in 7 patients, and posterior in 1 patient. All patients underwent echocardiography and DE-MRI before ICD implantation.

Four patients were treated with antiarrhythmic drugs (3 with amiodarone and 1 with sotalol) for 1 ± 0.5 months prior to the ablation.

Magnetic Resonance Imaging

Cardiac MRI was used to quantitate the left ventricular scar size. Because of the linear relationship between scar size and ejection fraction in patients with prior MI (4), patients with limited scar despite severe left ventricular dysfunction may have a superimposed cardiomyopathy, potentially explained by a PVC-induced cardiomyopathy.

All patients had DE-MRI studies within two weeks prior to the procedure. The studies were performed on a 1.5 Tesla MRI scanner (Signa Excite CV/i, General Electric, Milwaukee, WI) with a 4- or 8-element phased array coil placed over the chest of patients in supine position. Images were acquired with ECG gating during breath-holds. Dynamic short and long axis images of the heart were acquired using a segmented k-space steady-state free precession pulse sequence (repetition time 4.2 ms, echo time 1.8 ms, 1.4×1.4 mm in-plane spatial resolution, 8 mm slice thickness). After a 15 minutes delay following administration of 0.20 mmol/kg of intravenous gadolinium DTPA (Magnevist, Berlex Pharmaceuticals, Wayne, NJ), 2D DE-MRI was performed using an inversion-recovery sequence (4) (repetition time 6.7 ms, echo time 3.2 ms, in-plane spatial resolution 1.4×2.2 mm, slice thickness 8 mm) in the short axis and long axis of the left ventricle at matching cine-image slice locations. The inversion time (250 to 350 ms) was optimized to null the normal myocardium.

All DE-MRI images were analyzed off-line using specialized post-processing software (Cinetool, General Electrics, Milwaukee, WI). The DE-MRI images were reviewed by 2 observers blinded to the results of the ablation procedure. Discrepancies were resolved by consensus. For each subject, manual tracing of the endocardial contour, epicardial contour, and tracing of the area of abnormal signal was performed on the stack of 15–20 short axis images, from the base to the apex of the left ventricle. The full area of DE was then automatically determined by a region growing algorithm as the area encompassing pixels with values $\geq M/2$, using the traditional method of Full Width Half Maximum (5).

Assessment of Ejection fraction

A baseline echocardiogram was performed within the 3 months preceding the procedure (mean 69 ± 35 days before ablation). Echocardiograms were assessed by 2 independent echocardiographers blinded to the study and outcome of the ablation procedure. LVEF was calculated by Simpson's formula when 2 consecutive sinus beats were present, using the second sinus beat for analysis of the ejection fraction to avoid post-extrasystolic potentiation of left ventricular function. The baseline mean LVEF by echocardiography was similar in both groups (ablation group 0.38 ± 0.11 vs. control group 0.34 ± 0.14 ; $p = 0.352$). All patients had a repeat echocardiogram within 3–6 months after the intervention (mean 74 ± 31 days).

LVEF was assessed semi-automatically by cardiac MRI. The mean LVEF by MRI was similar in both groups (ablation group 0.38 ± 0.13 vs. control group 0.32 ± 0.12 ; $p = 0.25$).

Electrophysiology Procedure and Mapping

Mapping was performed in the 15 patients with frequent PVCs. The electrophysiology procedures were performed in the fasting state after informed consent was obtained. After femoral venous access was obtained, 3 quadripolar catheters were positioned in the right atrium, His bundle position, and right ventricle. Programmed ventricular stimulation was performed with up to 4 extrastimuli at 3 basic drive cycle lengths to assess for inducible sustained monomorphic VT (6). If sustained VT was induced, an ICD was implanted before

hospital discharge. VTs were targeted for ablation only if they remained inducible after frequent PVCs were ablated.

If the PVCs had right bundle branch block morphology, mapping was performed using a retrograde aortic approach through the right femoral artery. Three thousand units of heparin were administered for right-sided procedure, followed by 1000 units/hour. Systemic heparinization to achieve an activated clotting time of 300 seconds was performed for left-sided procedures. Electrograms were filtered at 50–500 Hz. ECG leads and intracardiac electrograms were displayed on an oscilloscope. The recordings were stored on optical disc (EP Medical Systems, New Berlin, NY).

In the presence of frequent spontaneous ectopy, activation mapping was performed with an open-irrigated 3.5-mm-tip catheter (Thermocool, Biosense Webster, Diamond Bar, CA) using an electroanatomical mapping system (CARTO, Biosense Webster, Diamond Bar, CA). If the ectopy was infrequent, isoproterenol was administered. If PVCs remained infrequent, pace-mapping was performed at a pacing cycle length that matched the coupling interval of the spontaneous ventricular ectopy (7). Bipolar voltage mapping was performed during sinus rhythm. Low voltage was defined as < 1.5 mV and dense scar was defined as < 0.5 mV. The areas of low voltage and dense scar were measured and correlated to the area of endocardial delayed enhancement.

RFA was performed at the site of earliest endocardial activation or best pace-map. The energy was titrated to achieve an impedance drop of 10 ohms. The applications were continued for at least 30 seconds if adequate heating at the electrode-tissue interface was achieved. If the PVCs were abolished within 30 seconds, the energy application was continued for 60 seconds and followed by another 60-second application. If PVCs were still present after 30 seconds, the energy application was terminated and mapping was continued. In the event of pleomorphic PVCs (present in 7 patients), the predominant PVC morphologies were targeted. An effective procedure was defined as a reduction in PVC burden of $\geq 80\%$. Programmed stimulation was repeated at the end of the procedure to rule out inducible VT.

Follow-Up

An ICD was implanted in the ablation group if sustained monomorphic VT was inducible. ICD implantation also was performed if the LVEF remained $\leq 35\%$ post-ablation (8). Antiarrhythmic drug therapy was discontinued if the ablation procedure was successful, but all other medications were kept unchanged. A 24-hour Holter monitor was performed within 3 months after the intervention to reassess the PVC burden. All patients had a repeat echocardiogram within 3 months of the ablation procedure or within 3–6 months of ICD implantation. Patients were seen in an outpatient clinic 3–6 months after the ablation procedure. Subsequent follow-up information was obtained from the referring physicians. The mean duration of follow-up post-ablation was 10 ± 10 months.

Statistical Analysis

Continuous variables are expressed as the mean $\pm$ 1 standard deviation and were compared using Student's *t* test. Discrete variables were compared using the Fisher exact test if a cell size was less than 5, or otherwise by Chi-square analysis. Echocardiographic measurements before and after intervention were compared by a paired *t* test. A *p* value < 0.05 was considered statistically significant. A Cohen's Kappa value was determined to assess the agreement in LVEF between the 2 echocardiographers. The Kappa value was 0.6383 (agreement of 0.941) for the assessment of LVEF between both echocardiographers. A correlation coefficient was calculated to assess the relationship between endocardial scar areas measured by electroanatomic mapping and DE-MRI.

Results

Magnetic Resonance Imaging

The percentage of scar tissue and the scar volume measured by DE-MRI were smaller in patients with frequent PVCs than in the control group ($10 \pm 8\%$ vs. $20.9 \pm 11.0\%$, $p = 0.005$, and $15.6 \pm 16.7 \text{ cm}^3$ vs. $39.1 \pm 25.0 \text{ cm}^3$, $p = 0.006$, respectively). The scar extent on the endocardial surface was smaller in patients with frequent PVCs compared to the control group ($22.7 \pm 22.2 \text{ cm}^2$ vs. $59.6 \pm 28.8 \text{ cm}^2$; $p = 0.0007$). There was no correlation between scar burden and PVC burden ($r = 0.3$; $p = 0.3$).

The average endocardial scar area as assessed by electroanatomic mapping using a lower limit of 1.5 mV for normal tissue was $38.3 \pm 27.8 \text{ cm}^2$. This was similar to the endocardial scar area measured by DE-MRI ($22.7 \pm 22.6 \text{ cm}^2$, $p = 0.29$). The average dense endocardial scar area as measured by electroanatomic mapping using a cut-off of 0.5 mV was smaller ($5.2 \pm 4.8 \text{ cm}^2$, $p = 0.008$) and correlated with the endocardial scar area measured by DE-MRI ($r = 0.9$).

Mapping and Ablation (Table 2)

PVC ablation was successful in all patients. The site of origin (SOO) of 23 different PVCs was identified during the procedure, with a mean of 1.5 PVC morphologies per patient (median 1 PVC/patient). These 23 PVCs accounted for 85% of the total PVC burden. The PVC morphologies were right bundle branch with superior axis in 7, right bundle branch with inferior axis in 13, left bundle branch with superior axis in 1, and left bundle branch with inferior axis in 2. PVCs were mapped by activation mapping in 9 patients and by activation mapping combined with pace-mapping in 6 patients. The SOO of PVCs was confined to low voltage endocardial scar tissue in 13/15 patients (87%). The anatomical distribution of the SOOs is shown in Table 2. In 2 patients, the PVC originated from an aortic cusp. In another patient the PVC originated from the left ventricular epicardium and was successfully ablated from within the greater cardiac vein. At the SOO, local endocardial activation preceded the PVC by a mean of $38 \pm 12 \text{ ms}$, and the mean electrogram amplitude during baseline rhythm was $0.40 \pm 0.28 \text{ mV}$.

Four of 15 patients (27%) had a total of 8 inducible monomorphic VTs (median 1 VT/patient) at the time of the procedure (mean VT cycle length $276 \pm 65 \text{ ms}$). The VT morphologies were right bundle branch with superior axis in 6, right bundle branch with inferior axis in 1, and left bundle branch with superior axis in 1. In 3 patients, the VT was reproducibly inducible at the beginning of the procedure but no longer inducible after the ablation procedure.

The average procedure time was 278 ± 98 minutes, with a mean fluoroscopy time of 56.4 ± 25.8 minutes. A mean of 12 ± 8 applications of radiofrequency energy were delivered per patient. In patients in whom only activation mapping was used to target the SOO, fewer ablation lesions were required to eliminate the PVC compared to patients in whom activation mapping and pace-mapping were used (4.8 vs 16.5 lesions; $p = 0.006$). No complications occurred.

In the PVC ablation group, the baseline mean PVC burden was $21.8 \pm 12.5\%$ and decreased significantly to $2.6 \pm 5.0\%$ after ablation ($p < 0.001$). The mean heart rate remained unchanged (72 ± 6 beats/minute vs. 73 ± 10 beats/minute; $p = 0.7$). The control group had a lower PVC burden of $1.9 \pm 3.2\%$ ($p < 0.0001$).

Assessment of Left Ventricular Function

After ablation, the mean LVEF increased significantly from 0.38 ± 0.11 to 0.51 ± 0.09 in the PVC ablation group ($p = 0.0005$, Figure 1). The mean left ventricular end-diastolic diameter decreased significantly after ablation ($56 \pm 11 \text{ mm}$ vs. $51 \pm 8 \text{ mm}$; $p = 0.030$). In the control

group, the mean LVEF (0.34 ± 0.14 vs. 0.33 ± 0.15 ; $p = 0.558$) (Figure 2) and left ventricular end-diastolic diameter (60 ± 8 mm vs. 59 ± 9 mm; $p = 0.477$) remained unchanged. Compared to the control group, the mean LVEF post-procedure was significantly higher in the ablation group (0.51 ± 0.09 vs. 0.33 ± 0.15 ; $p = 0.0003$) and the mean left ventricular end-diastolic diameter was significantly smaller (51 ± 8 mm vs. 59 ± 9 mm; $p = 0.035$).

There was no relationship between the PVC burden and the LVEF ($r = 0.1$; $p = 0.6$, Figure 3).

Functional Class and ICD Implantation

The New York Heart Association (NYHA) functional class was similar in the frequent-PVC group and in the control group at baseline (1.8 ± 0.8 vs. 1.9 ± 0.6 ; $p = 0.3$). The NYHA functional class improved post-ablation to 1.3 ± 0.5 in the frequent-PVC group; $p = 0.02$). In the ablation group, 5/15 patients required ICD implantation post-ablation (4 for inducible VT and 1 for persistent left ventricular dysfunction). Three of 7 patients in the ablation group who qualified for an ICD because of a low LVEF no longer qualified for ICD implantation post-procedure due to improvement in the LVEF to $>35\%$. In the control group, all but one patient underwent ICD implantation; one patient refused ICD implantation.

Follow-up

No patients died during a mean follow-up period of 14 ± 13 months. In the PVC ablation group, 1/5 patients received an inappropriate ICD shock. In the control group, 3 patients had device-related complications (device migration requiring pocket revision, left upper extremity deep vein thrombosis, chronic pain at incision site), 4 patients experienced appropriate ICD therapies, 2 patients received inappropriate ICD shocks, 2 patients eventually required VT ablation, and 1 patient had progression of heart failure.

Discussion

PVC Ablation and Structural Heart Disease

This is the first study to report a series of patients with ischemic cardiomyopathy in whom the ejection fraction improved after ablation of frequent PVCs. A PVC burden $> 5\%$ on 24-hour Holter monitor was selected as a cut-off based on a previous study in patients without prior MI (3). Our data suggest that despite the presence of scar tissue in post-infarction patients, a component of reversible cardiomyopathy may be present in patients with frequent PVCs.

A low ejection fraction with a small amount of scar tissue may suggest a potentially reversible cardiomyopathy. Ischemia may result in a cardiomyopathy that can be reversed by revascularization (9). Kim, et al demonstrated by DE-MRI that a smaller scar burden was present in patients in whom improvement of LVEF was observed after revascularization than in patients in whom LVEF failed to improve. However, the patients in this study had no evidence of ischemia and did not undergo revascularization. The most likely reason for improvement in the EF was elimination of the frequent PVCs. As in patients with idiopathic PVCs (2), this study demonstrates that RFA has a high success rate in patients with prior MI.

Scar and Origin of PVCs

Not unexpectedly, the majority of sites of PVC origin in this study were related to the infarct scar and were not situated in areas such as the right ventricular outflow tract where idiopathic PVCs typically originate (2). Similar to a previous study (11), the initially reproducibly inducible VT was no longer present after the PVC ablation. This suggests that the SOO of the PVCs in post-infarction patients often corresponds to the exit site of VT. The substrate probably consists of surviving muscle bundles within the scar area.

Assessment of Scar

As in a prior report (10), this study confirms a direct correlation between scar assessed by DE-MRI and electroanatomical voltage mapping. In the absence of DE-MRI, the endocardial scar burden can be assessed by voltage mapping.

Impact on LVEF

After PVC ablation, there was a significant improvement in the LVEF. This improvement was not attributable to revascularization, pharmacologic therapy, or changes in heart rate. In patients with prior MI and frequent PVCs, it is possible that LV dysfunction may be partially reversible, and that this can be identified by assessment of the scar burden on DE-MRI. The probability of improvement in LVEF may be greatest in the presence of a small scar burden. Of note is that current guidelines (8,11) do not address the issue of whether ICD implantation for the primary prevention of sudden death can be avoided when the LVEF improves after ablation of frequent PVCs in patients with an ischemic cardiomyopathy. Larger studies with longer follow-up are needed to adequately address this issue.

There is no clear explanation for the mechanism of PVC-induced cardiomyopathy. Possible explanations include asynchronous ventricular contraction generated by the PVCs, impaired calcium handling, or a decrease in calcium transient (12,13).

Limitations

A limitation of this study is the absence of randomization and the small number of patients. A larger patient population will be required to identify cut-off values for MRI-defined scar burden associated with a reversible cardiomyopathy. In addition, the cardiomyopathy may not be completely reversible by ablation. Because of the small number of patients who met the criteria for an ICD for primary prevention of sudden death in the ablation group, a larger study is required to confirm whether ICD implantation can safely be avoided if the LVEF improves to >35% after ablation of frequent PVCs. The findings of this study apply only to patients with remote MI and may not apply to patients with a recent MI.

Although the mean duration of follow-up was >12 months, we cannot be certain that the improvement in LVEF will be sustained, particularly if there is late recurrence of frequent PVCs. Activation mapping was used to target PVCs if they were frequent. In the absence of frequent ectopy, pace-mapping, which is less accurate than activation mapping(14), was used to target the SOO. In these patients, more ablation lesions may have been required to eliminate the PVCs.

Conclusion

PVC ablation in post-infarction patients with frequent PVCs may result in an improvement in the LVEF. It may be appropriate to screen patients with an ischemic cardiomyopathy for frequent PVCs with a 24-hour Holter monitor before implanting an ICD for primary prevention of sudden death. Ablation of the frequent PVCs may improve the LVEF such that the patient no longer meets the ejection fraction criterion for an ICD.

Glossary of Abbreviations

CS	Coronary Sinus
DE	Delayed Enhancement
ICD	Internal Cardioverter-Defibrillator
LVEF	Left Ventricular Ejection Fraction

LVOT	Left Ventricular Outflow Tract
MI	Myocardial Infarction
MRI	Magnetic Resonance Imaging
NYHA	New York Heart Association
PVC	Premature Ventricular Contraction
RFA	Radiofrequency Ablation
SOO	Site of Origin
VT	Ventricular Tachycardia

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Figure 1. Left ventricular ejection fractions before and after premature ventricular contraction (PVC) ablation.

Figure 2.
Left ventricular ejection fractions at baseline and 3–6 months later in the control group.

Figure 3. Relationship between the premature ventricular contraction (PVC) burden and the baseline left ventricular ejection fraction.

Table 1

Comparison of Patients with Frequent Premature Ventricular Contractions with Control Group Patients

	PVC Ablation (n = 15)	Controls (n = 15)	P Value
Age (years)	59 ± 12	64 ± 12	0.3
Sex, n (male / female)	12/3	12/3	
MI Age (years)	10 ± 13	10 ± 8	0.3
Prior Revascularization, n (%)	13 (87%)	14 (93%)	0.6
Diabetes Mellitus, n (%)	4 (27%)	5 (33%)	0.7
Chronic Renal Failure, n (%)	0 (0%)	4 (27%)	0.1
Beta-Blocker, n (%)	10 (67%)	15 (100%)	0.03
ACE Inhibitor, n (%)	12 (80%)	13 (87%)	0.6
Anti-Arrhythmic Drugs, n (%)	3 (12%)	4 (27%)	1.000
- Amiodarone	2	3	
- Sotalol	1	1	
Baseline LVEF			
- Echocardiogram	0.38 ± 0.11	0.34 ± 0.14	0.4
- Cardiac MRI	0.37 ± 0.13	0.32 ± 0.12	0.4
PVC Burden (%)	21.8 ± 12.5	1.9 ± 3.2	< 0.001

Abbreviations: ACE = Angiotensin Converting Enzyme; LVEF = Left Ventricular Ejection Fraction; MI = Myocardial Infarction; MRI = Magnetic Resonance Imaging; PVC = Premature Ventricular Contraction.

Table 2

Characteristics of Patients Who Underwent Premature Ventricular Contraction Ablation

Patients	Referral Indication	History of VT	PVC (%)	LVEF Before	NYHA Class Before	Site of Origin	Scar Tissue	Inducible VT
1	ICD	No	14.2	0.23	2	Papillary	Yes	No
2	PVC	No	13.3	0.55	1	Papillary	Yes	No
3	PVC	No	43.5	0.48	1	LVOT	Yes	No
4	ICD	No	27.0	0.30	3	LVOT	Yes	No
5	ICD	Yes	32.0	0.15	2-3	Mitral Annulus	Yes	Yes (5)
6	PVC	No	6.0	0.50	1	CS	No	No
7	PVC	No	10.3	0.60	1-2	Papillary	Yes	No
8	PVC	No	11.0	0.40	1-2	Papillary Aortic	Yes	No
9	ICD	Yes	32.3	0.28	3	Cusp	Yes	Yes (1)
10	ICD	Yes	13.9	0.43	1	Septum	Yes	Yes (1)
11	ICD	Yes	15.5	0.35	1	Inferior	Yes	Yes (1)
12	ICD	No	38.3	0.35	1	Inferior Papillary	Yes	No
						CS	No	
						CS	No	
						CS	No	
13	PVC	No	30	0.45	2	Septum Inferior	No	No
						Anterior	Yes	
						Aortic	No	
14	PVC	No	6	0.35	2	Cusp	Yes	No
15	PVC	No	34	0.43	3	Papillary Anterior	Yes	No
Mean			21.8	0.38	1.8			
Patients				LVEF After	NYHA Class After			
1				0.35	1			
2				0.55	1-2			
3				0.60	1			
4				0.57	1			
5				0.50	1-2			
6				0.50	1-2			
7				0.60	1-2			
8				0.48	1-2			
9				0.43	3			
10				0.45	1			
11				0.36	1			
12				0.50	1			
13				0.65	1			
14				0.45	1			
15				0.65	1			

Patients	LVEF After	NYHA Class After
Mean	0.51	1.3

Abbreviations: as above; CS = Coronary Venous System; ICD = Internal Cardioverter-Defibrillator; LVEF = Left Ventricular Ejection Fraction; LVOT = Left Ventricular Outflow Tract; NYHA = New York Heart Association; Papillary= Papillary Muscle;