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## Original Article

# Outflow tract ventricular premature beats ablation in the presence or absence of structural heart disease: Technical considerations and clinical outcomes



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## ABSTRACT

**Background:** Premature ventricular beats (PVBs) are early depolarization of the myocardium originating in the ventricle. In case of very frequent PVBs, patients are severely symptomatic with impaired quality of life and are at risk of pre-syncope, syncope, heart failure, and sudden cardiac death particularly in the presence of structural heart disease. Ventricular outflow tracts are the most common sites of origin of idiopathic PVBs especially in patients without structural heart disease. We examined the role of radiofrequency catheter ablation in suppression of monomorphic PVBs of outflow tract origin in the presence or absence of structural heart disease, and its impact on improvement of left ventricular (LV) systolic function.

**Methods:** Thirty-seven highly symptomatic patients with PVBs burden exceeding 10% were enrolled, provided that PVBs are monomorphic, originating in ventricular outflow tracts and regardless the presence or absence of structural heart disease. Patients were divided into 2 groups according to PVB site origin (RVOT vs. LVOT). 3D electro-anatomical mapping modalities were used in all patients employing activation mapping technique in the majority of cases. Acute success was considered when PVBs completely disappeared or when residual sporadic PVBs  $\leq 1$  beats/min or  $\leq 10$  beats/30 min after RF ablation. Patients were followed up for a mean period of  $5.4 \pm 1.2$  months with long-term success defined as complete disappearance or marked reduction by more than 75% in the PVBs absolute number on 24 h holter monitoring.

**Results:** Mean age of the study group was  $39.9 \pm 12.97$  years, including 22 (59.4%) males. PVBs originated in RVOT in 17 cases and in LVOT in the remaining 20 cases. Prevalence of structural heart disease and consequently shortness of breath was higher in LVOT group. Initial ECG localization matched EP localization in the majority (94%) of cases. R wave duration index was the only significant independent predictor for RVOT origin with cut off value of  $<0.3$  ( $P = 0.0057$ ) upon multivariate analysis. Acute success was encountered in 32 (86%) patients with all cases of failure in the LVOT group. Recurrence occurred in 5 (15%) cases without significant difference between both groups. All cases of recurrence had residual PVBs at the end of the procedure. 18 cases out of the study group showed significant improvement of their EF ( $>5\%$ ) at the end of the follow-up period with no significant differences between both groups ( $p = 0.09$ ). A linear correlation was observed between PVBs burden at follow up and magnitude of improvement of LV EF, particularly in patients with resting LV dysfunction and increased LV internal dimensions.

**Conclusions:** RF ablation is an effective and safe method for elimination of outflow tract PVBs irrespective of their origin and the presence or absence of structural heart disease. PVBs burden after ablation appears to be the main determinant for reversal of PVB induced myopathy particularly in those with increased LV internal dimensions.

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**Abbreviations:** PVB, premature ventricular beats; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract; SCD, sudden cardiac death; EF, ejection fraction; VT, ventricular tachycardia; EP, electrophysiological.

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## 1. Introduction

Premature ventricular beats (PVBs) are early depolarization of the myocardium originating in the ventricle. In patients with no underlying heart disease, PVBs are considered benign with very good prognosis. However, when PVBs are very frequent many patients are severely symptomatic with impaired quality of life and are at risk of pre-syncope, syncope, and heart failure. On the other hand, in the presence of structural heart disease PVBs represent increased risk of sudden cardiac death (SCD).<sup>1,2</sup>

ACC/AHA/ESC 2006 Guidelines for management of patients with ventricular arrhythmias and the prevention of SCD stated that beta blockers should be used as primary therapy in the management of ventricular arrhythmias and the prevention of SCD. In addition, ablation of asymptomatic PVBs may be considered when PVBs are very frequent; to avoid or treat PVB induced cardiomyopathy.<sup>3</sup>

PVBs originating in the ventricular outflow tract usually appear in patients without structural heart disease. They may present in the form of isolated or incessant PVBs, or as tachycardia (up to 80% of idiopathic ventricular tachycardia (VT)). The main causal mechanism is triggered activity, but re-entry or abnormal automaticity mechanisms have also been postulated. Beta blockers or Verapamil usually show only limited effectiveness in controlling this type of PVBs. Radiofrequency ablation can be effective, but is hampered by the fact that this PVBs has limited and unpredictable inducibility.<sup>4,5</sup>

We investigated the role of radiofrequency catheter ablation in suppression of monomorphic PVBs of outflow tract origin in the presence or absence of structural heart disease, and its impact on improvement of left ventricular (LV) systolic function.

## 2. Methods

### 2.1. Study population

Thirty-seven patients with very frequent (>10% PVBs burden documented on holter monitoring<sup>6</sup>) monomorphic PVBs, originating from right or left ventricular outflow tracts, in the presence or absence of structural heart disease, who are still symptomatic despite antiarrhythmic therapy including beta blockers, were enrolled in the current study. Patients with concomitant atrial arrhythmias, thyrotoxicosis, hypertrophic cardiomyopathy with septal thickness exceeding 14 mm, non-revascularized coronary artery disease, heart failure patients with NYHA class 3 or 4, and those with non-outflow tract PVBs were excluded.

### 2.2. Methodology

Detailed history (symptoms, full medical treatment, and family history of SCD), full clinical examination, and laboratory investigations (serum electrolytes, thyroid profile) for exclusion of reversible PVBs causes were done in all cases.

Standard 2D echocardiographic examination was done at baseline to exclude structural heart disease, occasionally after the procedure in case of suspected complications, and after 6 months of follow up of LV systolic function (calculated by modified Simpson method). Improvement of EF  $\geq$  5% compared to baseline was considered significant for further statistical analysis.

Twenty-four hours ambulatory ECG monitoring was done for assessment of absolute PVBs number, PVBs burden (calculated as number of PVBs/number of total heartbeats per 24 h  $\times$  100), and to exclude other life threatening arrhythmias. Holter monitoring was repeated 6 months after ablation or whenever significant

symptoms were encountered for detection of early or late recurrence.

### 2.3. Twelve lead ECG

After exclusion of myocardial ischemia, initial localization of PVBs origin was done using different algorithms including PVB transition in chest leads, V2 transition ratio (calculated as the percentage R-wave during VT: (R/R + S) VT divided by the percentage R-wave in sinus rhythm (SR): (R/R + S)SR), and R wave duration index (calculated by dividing the QRS complex duration by the longer R wave duration in lead V1 or V2). PVB duration, and coupling interval and axis in inferior leads were also recorded.<sup>7–9</sup>

### 2.4. Electrophysiological study (EP study) and radiofrequency (RF) ablation

EP study and ablation were done under local anesthesia after stoppage of antiarrhythmic drugs for at least 6 half-lives. Systemic anticoagulation was maintained by intravenous administration of heparin (initial bolus of 75 U/kg IV followed by 1000 U per hour) throughout the procedure.

Three Dimensional electro-anatomical mapping was done for all cases using either the CARTO 3 mapping system (Biosense, Diamond Bar, CA, USA) or the Ensite NavX<sup>®</sup> system (St Jude Medical, Inc, St Paul, MN) according to physician preference and availability. Three Dimensional compatible ablation catheters, (Thermocouple 4 mm tip 7F for Ensite NavX system and Thermocool 3.5 mm 8F for CARTO 3 system) were used. In addition a multi-electrode (quadripolar or decapolar) catheter was introduced into the RV (apex or RVOT) to be used for pacing and as a reference catheter if needed.

### 2.5. Mapping techniques

In case of frequent PVBs an activation map during PVBs of the chamber of origin was created; the ventricle was plotted by dragging the mapping catheter over the endocardium. The site of earliest ventricular activation (red isochrones in CARTO 3 or white isochrones in NavX) with a local ventricular electrogram preceding the surface QRS onset by at least 25–30 ms was targeted by ablation. Voltage maps were also created especially in patients with structural heart disease for identification of scar tissue (areas with local voltage < 0.5 mv).

Mapping was always started in the RV even if PVBs were suspected to originate from LV. Mapping timing reference was either stable multi-electrode catheter as coronary sinus decapolar catheter for NavX system, or 12 lead surface ECG for CARTO 3 system.

In cases of infrequent PVBs encountered, drug provocation with epinephrine (0.1 mcg/kg/min) was used. If PVBs remained infrequent, pace mapping protocol was performed at different locations within the designated chamber. Ablation was attempted at sites with perfect pace maps 12/12 in comparison with 12 lead ECG recording of clinical PVBs.

### 2.6. Ablation, post ablation study and follow-up

RF energy was delivered in a temperature-controlled mode for 60–120 s at each ablation site with a maximum temperature of 48°C and a maximum power of 30–50 W. In case of aortic cusp origin, coronary angiography was done first and radiofrequency was adjusted to maximal power of 30 W.

Acute success was considered when PVBs completely disappeared or when residual sporadic PVBs  $\leq$  1 beats/min or  $\leq$  10

beats/30 min after RF ablation, while long-term success was defined as complete disappearance or marked reduction by more than 75% in the PVBs absolute number on 24 h holter monitoring done 3–6 months after RF ablation together with improvement in symptoms.<sup>10</sup>

Patients were closely observed after the procedure for any complications, and were followed up for a mean period of  $5.4 \pm 1.2$  months. None of the included patients was given antiarrhythmic drugs after successful ablation.

All patients gave a written informed consent and the study was approved by the Research and Ethics Committee of the cardiology department, faculty of medicine, Ain Shams University.

### 2.7. Statistics

Data were analyzed using SPSS version 21 for Windows and graphics by MS Excel. Categorical data were expressed as frequencies and percentages, while continuous data were expressed as mean  $\pm$  SD or median. Comparison between categorical variables was done using chi-square or Fisher's exact test as appropriate. Comparison between continuous variables was done using *t*-test or Mann–Whitney test according to normality of distribution. Multivariate stepwise logistic regression analysis was used to identify predictors of PVB origin. Receiver operating characteristics (ROC) curve analysis was done to find the impact of different ECG parameters on PVB localization. Cutoff values were selected if area under the curve (AUC) was significantly different from 0.5. *P* value was considered significant if  $<0.05$ .

### 3. Results

Thirty-seven cases with a mean age of  $39.9 \pm 12.97$  years, including 22 (59.4%) males, having symptomatic frequent monomorphic PVBs, presented to the EP clinic at Ain Shams University hospitals and referred for EP study and ablation, were enrolled in the current study. Based on EP localization of PVBs, the study population was divided into 2 groups: group 1 with

RVOT PVBs ( $n = 17$ ), and group 2 with LVOT PVBs ( $n = 20$ ). Baseline demographic clinical, echocardiographic, and holter features of the study group are shown in Table 1.

Structural heart disease incidence and accordingly shortness of breath was significantly higher in the LVOT group. Ten patients with LVOT origin of PVBs had non-ischemic LV dysfunction, one patient had coronary artery disease revascularized by CABG, two patients had coronary artery disease with fair LV systolic functions, one patient had rheumatic heart disease with severe mitral regurgitation and the last patient had bicuspid aortic valve with severe aortic regurgitation. In patients with RVOT origin of PVBs, two had non-ischemic LV dysfunction, single patient with ischemic cardiomyopathy and a patient with RV dilatation and scarring with probable diagnostic criteria of ARVC.

#### 3.1. PVB origin in relation to different ECG algorithms

PVBs were initially localized using different ECG algorithms. Surface ECG localization of PVBs matched intracardiac localization in 35 cases, except only 2 patients of LVOT group had initially incorrect localization. Patients with LVOT PVBs had significantly earlier transition, significantly lower complex duration, and significantly higher V2 transition ratio and R wave duration index compared to RVOT group (Table 2, Fig. 1).

The predictive value of different ECG algorithms on PVB localization was analyzed using univariate analysis (Table 3, Fig. 2). Multivariate stepwise logistic regression analysis showed that R wave duration index was the only significant independent predictor for RVOT origin with cutoff value of  $<0.3$  ( $P = 0.0057$ ).

Moreover, precise localization of RVOT PVBs was done using ratio of PVB duration to preceding sinus beat duration, where a ratio of  $>1.8$  predicted free wall rather than a septal origin of PVB with sensitivity of 97.06% and specificity of 83.33%, *P* value of  $<0.0001$  (Fig. 3) (see Figs. 4 and 5).

#### 3.2. Procedural aspects

Apart from slightly higher impedance recorded at the ablation site in the LVOT group, no significant difference was encountered between both groups regarding type of the 3D mapping system used, method of mapping [activation mapping in the majority (30 cases)], total procedure time, fluoroscopy time, or the number of ablation trials. Thirty-two cases in the current study achieved acute success according to predefined criteria. Remarkably, the 5 cases which did not meet success criteria were all in the LVOT and aortic sinuses, a finding that was of borderline statistical significance. Long-term success was achieved in 27 cases with no significant difference between both groups, while 5 cases had recurrence at follow up.

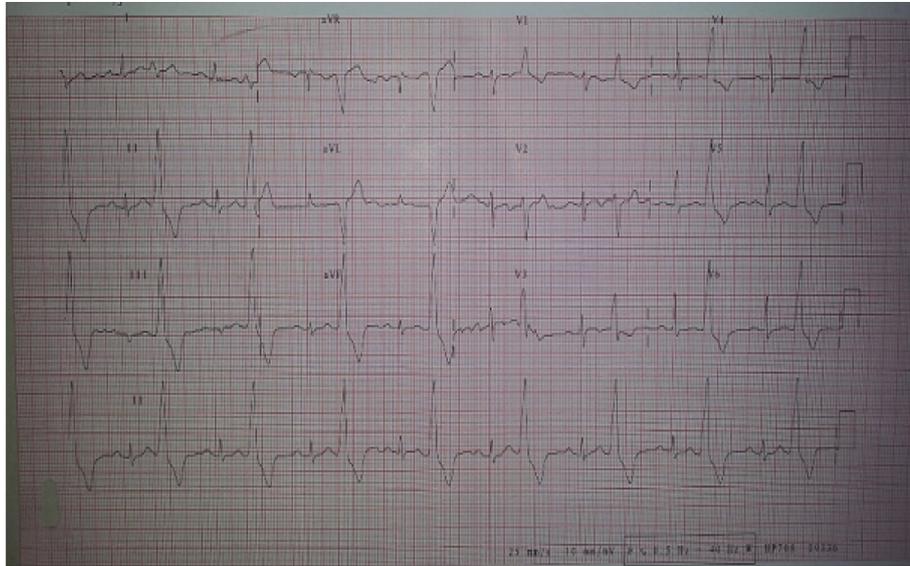
Interestingly, retrospective analysis of the patients with recurrence showed that all of these patients (100%) had residual PVBs

**Table 1**  
Baseline features of the study group.

	RVOT n = 17	LVOT n = 20	P
Age (years)	$36.4 \pm 12.8$	$42.9 \pm 13.5$	NS
Male gender (n)	9	11	NS
Structural heart disease n (%)	4 (23.5%)	15 (75%)	$<0.01$
<i>Symptoms</i>			
Duration (years)	$5.5 \pm 3.5$	$5.9 \pm 2.6$	NS
Dyspnea (n)	6	16	$<0.01$
Palpitation (n)	14	15	NS
Syncope (n)	2	1	NS
<i>Treatment</i>			
Amiodarone (n)	5	9	NS
Sotalolol (n)	7	5	NS
Beta blockers (n)	5	6	NS
<i>Echo parameters</i>			
LVEDD (mm)	$51.11 \pm 6.73$	$54.75 \pm 10.04$	NS
LVESD (mm)	$34.82 \pm 5.52$	$38.70 \pm 10.93$	NS
2D EF	$60.29 \pm 10.64$	$56.45 \pm 14.35$	NS
<i>Holter parameters</i>			
Pre-procedural PVB burden (%)	$27.00 \pm 9.44$	$24.35 \pm 9.43$	NS
Pre-procedural PVB (n)	$28022.2 \pm 8154$	$29187.2 \pm 14178.2$	NS
Pre-procedural Bigeminy cycles (n)	$3748.94 \pm 6221.45$	$5439.75 \pm 10748.5$	NS
Pre-procedural Couplets (n)	$561.52 \pm 1359.06$	$1411.30 \pm 3623.64$	NS
Pre-procedural NSVT (n)	$77.76 \pm 272.15$	$95.10 \pm 203.41$	NS
Pre-procedural VT (n)	$0.17 \pm 0.39$	$2.90 \pm 12.96$	NS

**Table 2**  
Different ECG algorithms for PVBs localization.

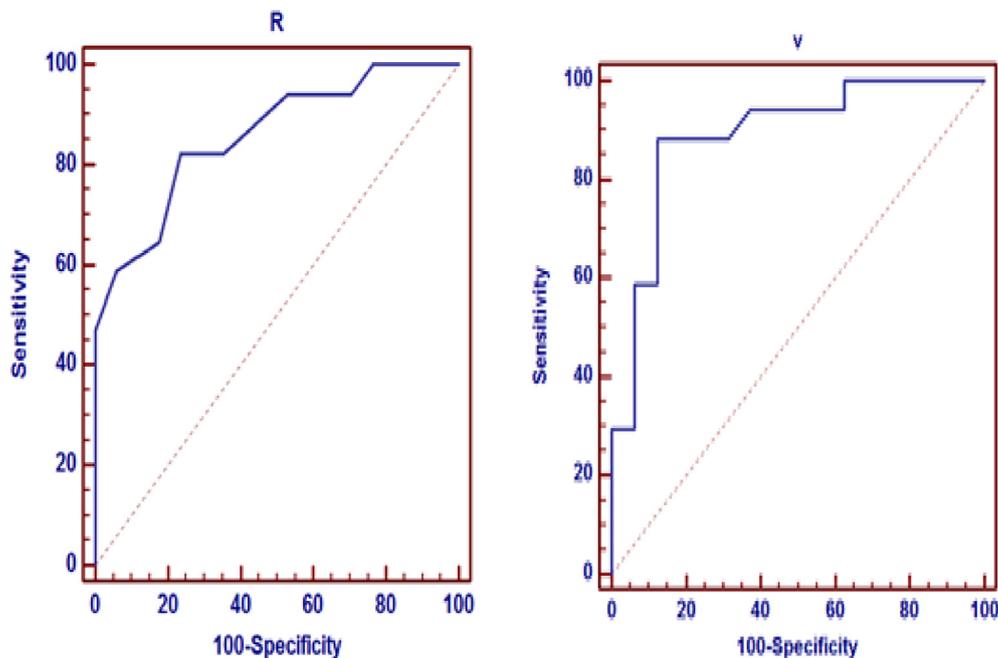
ECG parameters	RVOT n = 17	LVOT n = 20	P
<i>PVB transition</i>			
V1	0	7	$<0.001$
V2	0	2	
V3	4	11	
V4	10	0	
V5	2	0	
V6	1	0	
PVB complex width (ms)	$148.23 \pm 24.55$	$134.00 \pm 18.46$	0.05
V2 transition ratio	$0.41 \pm 0.30$	$1.70 \pm 1.53$	$<0.001$
R wave duration index	$0.25 \pm 0.11$	$0.45 \pm 0.15$	$<0.001$



**Fig. 1.** 12 lead surface ECG from patient no. (21), showing frequent ventricular ectopics with PVB transition in V3, earlier than sinus beat transition, and PVB localizing algorithms were calculated as follows: V2 transition ratio of 1.5, R/S ratio of 0.6 and R wave duration index of 0.5, suggestive of LVOT PVB origin that was confirmed to originate from left coronary cusp by intracardiac mapping.

**Table 3**  
Predictive value of different ECG parameters on PVBs localization.

	Cut off value	AUC (95% CI)	P	Sensitivity	Specificity
V2 transition ratio	$\leq 0.58$	0.888 (0.769–1.000)	<0.0001	88.2	87.5
R wave duration index	<0.3	0.858 (0.734–0.987)	<0.0001	82.3	76.4
PVB/Sinus duration	>1.8	0.956 (0.892–1.000)	<0.0001	97	83.3

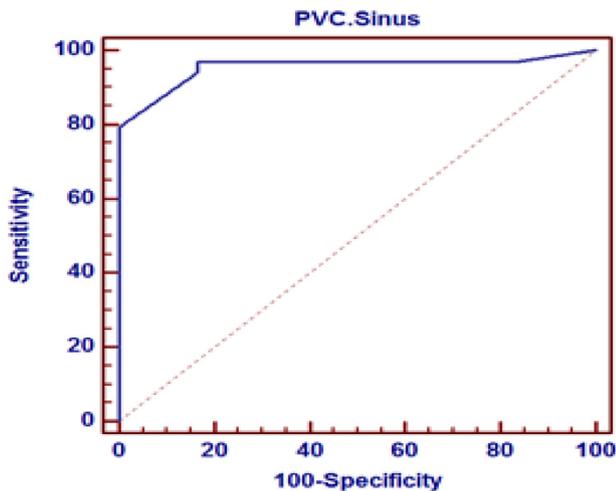


**Fig. 2.** ROC curves showing sensitivity and specificity of V2 transition ratio (right panel), and R wave duration index (left panel) in predicting RVOT PVBs origin.

at the end of the procedure, not complete disappearance; however they met the criteria of acute success, a finding that was highly significant (Table 5).

Regarding post-procedural complications, 1 patient died out of massive cerebral infraction associated with a mass on the aortic

valve. Coronary artery injury with left circumflex thrombosis occurred in a single patient during ablation in Left coronary cusp which was conservatively managed with intracoronary flushes of saline and coronary dilators. TIA occurred in a single patient, VF in a single patient during RF ablation in RVOT, cardiac tamponade



**Fig. 3.** ROC curve analysis showing sensitivity and specificity of PVB/Sinus duration in predicting free wall origin of PVB.

in 1 patient with RV dilation and scarring, and puncture site hematoma in 2 cases without any significant difference between both groups (Table 4).

### 3.3. Post-procedural holter findings

All post-procedural holter parameters showed highly significant improvement in each individual group compared to baseline parameters. However, only post-procedural PVBs burden percent was significantly lower, and magnitude of change in PVB burden was significantly higher in RVOT group compared to LVOT group (Table 6).

### 3.4. Post ablation echocardiographic data

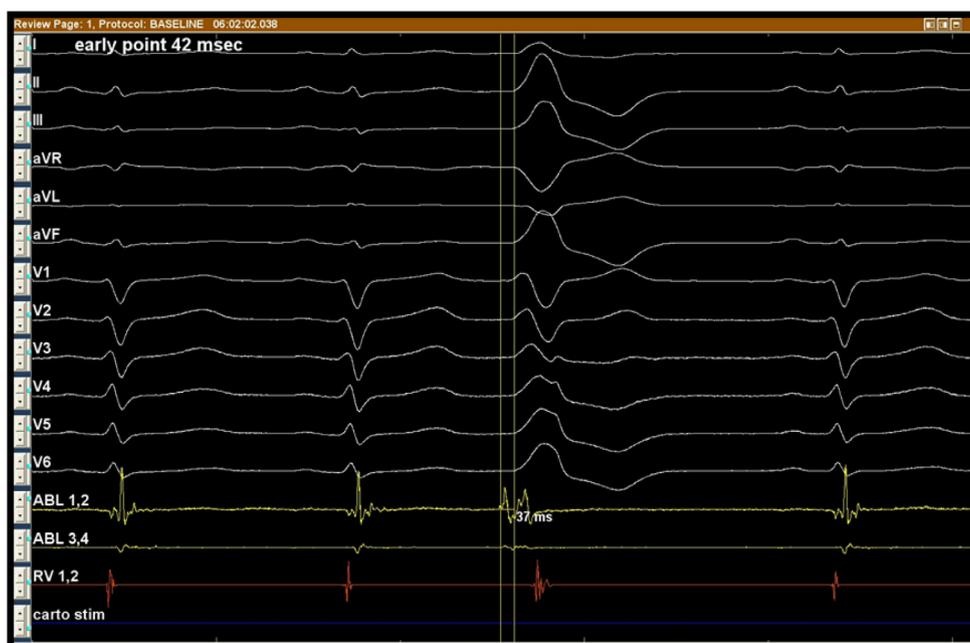
Excluding the patient who died 3 days post-procedural, 18 (11 in RVOT group and 7 in the LVOT group) cases out of the study

group showed significant improvement of their EF (>5%) at the end of the follow up period with no significant differences between both groups ( $p = 0.09$ ). Mean PVBs burden was significantly lower in the group with significant improvement in their EF ( $1.42 \pm 1.97$  vs  $7.88 \pm 12.52\%$ , respectively) and it appeared that this is a linear correlation i.e. the lower the PVBs burden at follow up, the higher the magnitude of improvement in the EF regardless of the PVBs origin, the presence or absence of structural heart disease, and the achievement of long-term success. Improvement in EF was significantly related to baseline LVEDD, LVESD and EF (Fig. 6, Table 7). This effect was more pronounced in patients with resting LV dysfunction ( $n = 12$ ) rather than in patients with resting normal LVEF ( $n = 24$ ) where the magnitude of change in EF was  $5.9 \pm 3.79$  (6.5) vs  $3.2 \pm 4.6$  (3), for mean and median values respectively for both groups.

## 4. Discussion

Isolated PVBs are the most common non-sustained arrhythmia that physicians often see during patient examination. The incidence of PVBs is highly variable among observational studies and is predominantly dependent on the sampling technique, ranging from 4.6% in young healthy participants to 62% in patients with known heart disease. PVBs arise most commonly from the RVOT and less frequently from LVOT, either below or above the semilunar valves. The prognosis of PVBs varies depending on the patient's age, baseline heart disease, LV function and co-morbidities. RF ablation is an accepted approach for symptomatic patients despite optimal medical therapy including beta blockers or asymptomatic patients with either high PVBs burden or systolic dysfunction with a possible etiologic link between PVBs and impaired function.<sup>11–13</sup>

The current study included 37 symptomatic cases with monomorphic PVBs despite medical therapy including beta blockers, and even class III antiarrhythmic amiodarone. In contrast to many published reports, PVBs originated more commonly in LVOT (54% of the cases), a finding that may be explained by the high percentage of patients with structural heart disease among the study group which accounts also for the presence of dyspnea in around



**Fig. 4.** Intracardiac tracing showing surface ECG, RV quadripolar catheter, and recording from ablation catheter in LCC preceding QRS complex by 42 ms in successful ablation site.

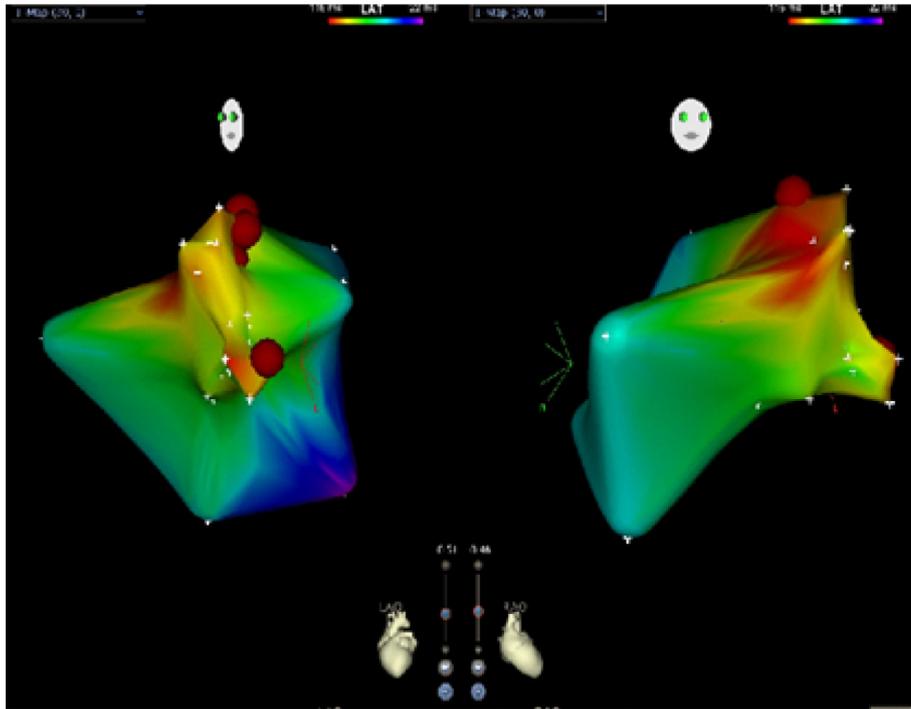


Fig. 5. CARTO 3 image in LAO and RAO views showing clinical PVB and activation mapping in supra-valvular LVOT with red dots denoting site of successful ablation in LCC.

Table 4  
EP procedural aspects in the study population.

	RVOT n = 17	LVOT n = 20	P
3D mapping			
CARTO	12	14	NS
NavX	5	6	NS
Pace mapping	2	1	NS
Cath induced VT (n)	3	2	NS
Residual PVBs after ablation (n)	2	4	NS
Time of earliest activation signal preceding QRS (ms)	41.82 ± 8.55	44.65 ± 13.28	NS
Procedural time (min)	107.94 ± 31.27	125.50 ± 35.57	NS
Fluoroscopy time (min)	34.41 ± 11.04	40.55 ± 11.85	NS
Ablation Power (Watt)	35.29 ± 1.21	34.00 ± 3.007	NS
Ablation temperature (C)	50.76 ± 5.22	49.55 ± 4.22	NS
Impedance (ohms)	109.70 ± 8.92	115.80 ± 9.02	0.047
Ablation time (s)	388.82 ± 149.91	441.00 ± 214.37	NS
Ablation trials	3.52 ± 2.00	4.65 ± 2.47	NS
Complications/mortality (n)	3/0	3/1	NS
Acute failure	0	5	0.057
Recurrence	2	3	NS

Table 5  
Effect of residual PVBs after ablation on recurrence.

	No recurrence n = 27	Recurrence n = 5	P
Residual PVBs after ablation (N)	1 (3.7%)	5 (100%)	<0.001

60% of the studied group, a symptom that is not frequently encountered in patients with isolated PVBs.<sup>14,15</sup>

Initial PVBs localization was done from surface ECG with the aid of different ECG algorithms. V1, V2 PVB transition was associated exclusively (100%) with LV PVBs, the same for V4-V6 transition that predicted RV origin. Patients with LVOT origin had significantly higher V2 transition ratio and R-wave duration index compared to RVOT patients. A cutoff value <0.58 for V2 transition ratio,

Table 6  
Post ablation holter parameters.

	RVOT n = 17	LVOT n = 20	P
Post-procedural PVB burden (%)	1.18 ± 1.82	8.17 ± 9.12.41	0.04
Post-procedural PVB (N)	1047.18 ± 2002.36	9110.68 ± 18391.7	0.07
Post-procedural Bigeminy cycles (N)	2.29 ± 5.15	122.36 ± 248.86	0.05
Post-procedural Couplets (N)	14.05 ± 31.73	1261.32 ± 3925.54	0.18
Post-procedural NSVT (N)	0.16 ± 2.66	58.94 ± 176.61	0.16
Difference in PVB burden (%)	-25.7 ± 9.55	-17.35 ± 12.85	0.033

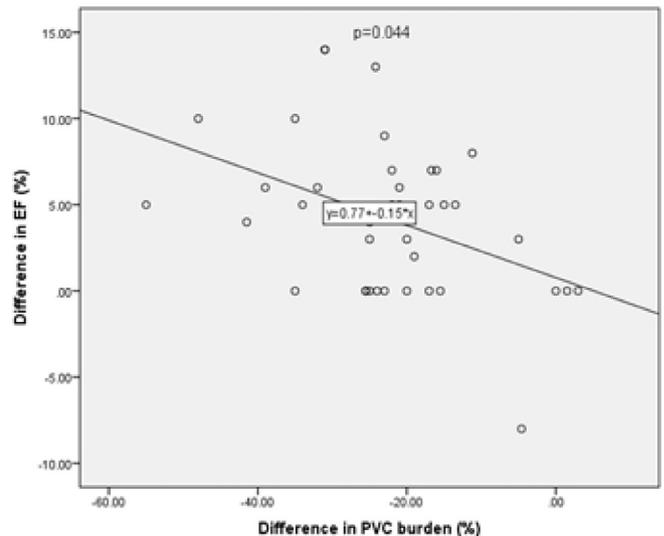


Fig. 6. Correlation between difference in EF and difference in PVB burden pre-and post-procedural.

**Table 7**  
Relation of EF improvement to various clinical and EP parameters.

	EF improvement > 5% n = 18	No improvement n = 18	P
Post ablation PVBs burden	1.42 ± 1.97	7.88 ± 12.52	0.03
<i>PVB origin</i>			
RVOT	11	6	NS
LVOT	7	12	NS
Long-term success n = 27	15	12	NS
Structural heart n = 19	9	10	NS
Normal heart n = 17	9	8	NS
Baseline LVEDD	55.95 ± 8.31	49.73 ± 8.02	0.02
Baseline LVESD	40.85 ± 8.31	32.6 ± 7.32	0.002
Baseline EF%	52.7 ± 12.5	63.2 ± 10.4	0.007

and <0.3 for R wave duration index predicted RVOT origin of PVBs with 88%, 82% sensitivity and 87%, 76% specificity, respectively. Comparable data reported cutoff values of >0.6 for V2 transition ratio for LVOT origin with 95% sensitivity and 100% specificity, while R wave duration index <0.5 predicted RVOT origin.<sup>7,9</sup>

All included patients were symptomatic, with a mean PVBs burden of 26.4 ± 9.86% despite medical therapy, a number that exceeded by far the cutoff value for RF ablation (20–24%/24 h) described by many authors to be associated with an increased risk of developing impaired LV function and cardiomyopathy even in asymptomatic cases. Lower thresholds for RF ablation (<5%) have been described in patients with persistent symptoms despite therapy or those with impaired LV systolic function to prevent further progression.<sup>16,17</sup>

#### 4.1. RF ablation

RF ablation met the criteria of acute success in 86% of the studied population. All the cases with failed ablation were located in supra-valvular LVOT. Three cases had close proximity to left main coronary artery, and the remaining two had an epicardial origin of the PVBs. It is worth noting that in cases of supra-valvular origin direct cannulation of LM coronary artery using guiding catheter for protection and the ablation catheter was placed at least 10 mm away from nearest coronary ostium. On the other hand, recurrence was observed in 15% of the cases, all of whom had residual PVBs at the end of the procedure. Comparable rates of success and recurrence were reported by other investigators depending on the PVBs origin and the presence or absence of structural heart disease, and the presence of more than 1 focus for PVBs.<sup>11,18</sup>

The procedure was generally safe with the only mortality attributed to extensive heating in the aortic root, total ablation time of around 15 min. Though RF ablation of outflow tract tachycardia is reported to be safe; Zhong and his colleagues recently reported a procedure-related complication rate of 5.6%.<sup>19</sup>

50% of the study cohort had significant improvement of their EF > 5% from the baseline at the end of the follow-up period. This improvement had an inverse linear correlation with the PVBs burden post ablation and the magnitude of improvement was significantly higher in patients with resting LV systolic dysfunction having increased LVEDD and LVESD. Many explanations have been proposed to explain the mechanism of PVBs induced cardiomyopathy including LV dyssynchrony due to LBBB during PVCs, increased oxygen consumption, and disrupted squeezing effect in systole of the LV during RVOT PVCs. This improvement was observed regardless of the PVBs origin and the presence of long-term success. In the current study patients were followed up for a mean period of only 5.4 months. Longer periods of LV recovery up to 45 months have been mentioned by other investigators.<sup>20–22</sup>

#### 4.2. Study limitations

The relatively small number of cases and the lack of epicardial ablation facilities are the main limitations of the current study.

#### 5. Conclusions

RF ablation is an effective and safe method for elimination of outflow tract PVBs irrespective of their origin and the presence or absence of structural heart disease. PVBs burden after ablation appears to be the main determinant for reversal of PVB induced myopathy particularly in those with increased LV internal dimensions.

#### Conflict of interest

No potential conflict of interest declared by the authors.

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