ORIGINAL ARTICLE

Does the origin of ablated premature ventricular contractions determine the level of left ventricular function improvement?

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KEY WORDS
arrhythmia origin, premature ventricular contractions, radiofrequency ablation, tachycardiomyopathy

ABSTRACT

BACKGROUND Premature ventricular contractions (PVCs) are associated with tachycardiomyopathy and high mortality rate. The treatment depends on the engaged ventricle. For PVCs originating from the right outflow tract (OT), radiofrequency catheter ablation (RFCA) is recommended (class IB-R recommendation) in preference to pharmacotherapy. In those originating from the left ventricle, ablation is a class IIa B-NR recommendation.

AIMS The aim of the study was to assess the success of RFCA of PVCs based on arrhythmia origin.

METHODS A total of 110 consecutive patients with monomorphic PVCs referred for ablation were enrolled and divided according to the site of ablation to the OT group and the ventricles (VENT) group. Holter electrocardiography and echocardiography were performed before the procedure and at 6-month follow-up.

RESULTS Long-term RFCA success was achieved in 93 (85%) patients (89% in the OT group and 82% in the VENT group; $P = 0.39$). The PVC reduction was similar in both groups (median [interquartile range] 99.55% [14] and 99.88% [6], respectively; $P = 0.56$). The OT group presented greater left ventricle (LV) recovery than the VENT group (odds ratio, 2.01; 95% CI, 1.15–10.75; $P = 0.015$). The procedure in the VENT group was longer, required additional access, the complication rate was similar, and 1 serious adverse event (aortic dissection) was observed in a patient with arrhytmia originating in the LV outflow tract.

CONCLUSIONS The origin of PVCs does not determine the success of arrhythmia elimination. The OT origin may predict LV improvement. The duration of RFCA in the VENT group was longer. The outflow tract origin may predict reversal of LV deterioration.

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INTRODUCTION There have been many reports proving that radiofrequency catheter ablation (RFCA) of premature ventricular contractions (PVCs) is a successful treatment of symptomatic disease. The successful elimination of arrhythmia is also connected with the reversal of left ventricular (LV) dysfunction and improvement of LV function, not only in tachycardiomyopathy without concomitant disorders but also in structural heart disease (SHD).[1-4] Nevertheless, the relation between arrhythmia exit origin and its influence on the LV function is not fully known. In light of the 2019 HRS / EHRA / APHRS / LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias,[1] symptomatic PVCs originating from the right ventricular outflow tract is recommended for RFCA as class IB-R recommendation in preference to pharmacotherapy. In case of arrhythmia originating from the LV outflow

WHAT'S NEW?

The origin of arrhythmia and its influence on tachycardiomyopathy have been largely debated. Outflow tract arrhythmia (both right and left sided) differs from the ventricular loci not only in the primary disease genesis but also the radiofrequency catheter ablation (RFCA) procedure course. It may not only favor reverse remodeling but also allow for shorter RFCA and fluoroscopy times or fewer accesses in comparison with ventricular exits. This is the first study to assess ventricular improvement after successful ablation with regard to anatomic division to outflow tract and ventricular origin of arrhythmia. Curative RFCA may also unmask tachycardiomyopathy diagnosis.

tract, epicardial outflow tract, and LV summit, ablation is recommended in case of ineffective pharmacotherapy as class IIa B-NR recommendation. The aim of the study was to assess RFCA of PVCs based on the arrhythmic focus localization. We assessed separately outflow tracts and ventricles, and RFCA on LV parameters reversal. We aimed to identify the correlation between the arrhythmia exit site and the success rate of RFCA. We also aimed to present the level of LV deterioration reversal depending on the arrhythmia origin.

METHODS This was a retrospective analysis of 110 out of 120 consecutive patients referred for RFCA of symptomatic monomorphic PVCs after failure of pharmacological management between 2010 and 2012 in Heart Center Leipzig, Leipzig, Germany. Patients who additionally presented sustained ventricular tachycardia were excluded. All activities performed at the center were routine treatment and follow-up and the study did not involve any additional procedures. All patients provided written informed consent for hospitalization, ablation, and follow-up. No additional informed consent for the study was required. For this analysis, ethics committee approval was not required. A detailed medical history of the patient was obtained before the procedure, including symptoms assessed on the basis of the New York Heart Association functional classification, previous pharmacological treatment, and concomitant diseases. All patients had 24-hour Holter monitoring and transthoracic echocardiography performed at least once before RFCA and 6 months after the procedure. Dilated cardiomyopathy was defined as LV dilatation (left ventricular end-diastolic diameter [LVEDd] >12% corrected for age and body surface area) or increased diastolic and systolic volumes indexed to body surface area with LV ejection fraction (LVEF) of less than 45% excluding chronic pressure overload (hypertension and LV outflow obstruction), chronic volume overload states (intracardiac shunts and valvular regurgitation), and ischemic heart disease. The differential diagnosis was performed with the use of exercise testing and/or coronary angiography, computed tomography, or magnetic resonance imaging. The prespecified screening procedural protocol was applied in all patients.

Electrocardiography and Holter monitoring

Preprocedural electrocardiography and 24-hour electrocardiography Holter monitoring were performed to obtain baseline QRS morphology, conduction and repolarization disorders of baseline QRS, and PVC morphology. The PVC number was defined as the total number of QRS complexes in 24 hours.

Echocardiography The echocardiographic data were analyzed with ultrasound systems (Vivid-7 System, Vingmed, General Electric, Horten, Norway; Philips IE 33, X7-2t probe, Philips Healthcare, Best, the Netherlands) by 2 independent echocardiographists who were blinded to all clinical and procedural data. LVEDd and LV end-diastolic volume as well as LV end-systolic diameter and volume were obtained from the apical 2- and 4-chamber views of transthoracic access. LVEF was estimated with the biplane Simpson method.

Radiofrequency ablation The right ventricle was accessed via the left or right femoral vein, the LV via transseptal puncture or retrogradely by aortic valve, and subxiphoid puncture was used in pericardial access. Sometimes 2 or even 3 routes of access (transseptal/transaortal, transvenous/transaortal, transvenous/transseptal, transseptal/subxiphoid, or transseptal/transaortal/subxiphoid) were used. The procedures were performed with conventional fluoroscopy and electroanatomical system mapping. The ablation catheter was a 4-mm irrigated-tip catheter (NaviStar ThermoCool, Biosense Webster, Diamond Bar, California, United States) with a 3-dimensional nonfluoroscopic mapping system (CARTO or CARTO RMT, Biosene Webster; and remote-control navigation of magnetically enabled catheter with the Niobe Stereotaxis [Stereotaxis Inc., St. Louis, Missouri, United States]). The best ablation site was determined by activation- and/or pace-mapping. The site of the origin of arrhythmia was defined as the earliest site of the local activation time and from which the best pace-mapping score was obtained. In patients with rare PVCs during the procedure, isoproterenol infusion was administered (2–10 µg/min) and programmed electrical stimulation was performed. Radiofrequency energy was applied at 20 to 45 W (maximum temperature, 48 °C; flow, 18 or 30 ml/min; 60 seconds). On the basis of activation mapping, the place of origin was assessed and numbered according to the mapping schema used by Josephson.[2] The acute success was noted if the clinical and targeted PVC did not occur spontaneously after 30 minutes

upon last radiofrequency application and could not be induced by isoproterenol or programmed electrical stimulation.

Follow-up A 24-hour Holter monitoring and echocardiographic assessment was performed at least once 6 months after the procedure. β-Blockers were discontinued if complete LV normalization was achieved and there were no other indications for treatment.

Statistical analysis Statistical analyzes were performed with the IBM SPSS Statistics 23 software (IBM, Armonk, New York, United States). Continuous variables were presented as means with (SD) or medians with interquartile range (IQR) depending on the normality distribution checked with the Shapiro-Wilk's test. To analyze basic descriptive statistics we used the Kolmogorow–Smirnov tests. Frequency analyses of categorical data were performed with the χ^2 test and the Fisher exact test. The *t* test was used for numerical variables. To analyze the non-normal distributed variables, the Mann–Whitney test was used. The correlation analyses were performed with Spearmann rank coefficient (ρ). The multivariate mixed-design analysis of covariance (ANCOVA) was used to present postintervention echocardiographic differences between the groups. The adjustment for pre-intervention parameters in compared groups was performed with the analysis of variance (ANOVA). A *P* value of less than 0.05 was considered significant.

RESULTS A total of 110 patients (mean [SD] age, 55 [16] years; female sex, 48%) with frequent PVCs were referred for RFCA of symptomatic ventricular arrhythmia. All patients underwent 24-hour Holter monitoring and echocardiography before the procedure and 6 months after. Mean baseline QRS duration, atrioventricular conduction time, and QT interval were assessed. Median (IQR) LV end-diastolic diameter was 52.5 (13) mm,

TABLE 1 Characteristics of the whole study group

Parameter	Value
Age, y, mean (SD)	55.8 (16.4)
PVCs before the procedure, n, mean (SD)	19 877 (9972)
PVCs after the procedure, n	405 (1117)
QRS, ms, mean (SD)	93.3 (22.9)
PQ, ms, mean (SD)	159.3 (38.9)
QT, ms, mean (SD)	389.9 (43.8)
LVEDd before, mm	52.5 (13)
LVEDd after, mm	50 (9.5)
LVESd before, mm	39 (13)
LVESd after, mm	35 (11)
LVEF before, %	55 (17.5)
LVEF after, %	60 (15.3)
LVEDV before, ml	125 (65)
LVESV before, ml	60 (49)
LVEDV after, ml	110 (55)
LVESV after, ml	45 (48)
Change of LVEF, %	5 (10)
Procedure time, min, mean (SD)	128 (50)
RF time, min	17 (22)
Time of applications, s	584 (782.3)
Applications, n	12 (18)
RF dosage, cGy/m^2	3992 (8445)

Data are presented as median (interquartile range) unless otherwise indicated.

Abbreviations: IQR, interquartile range; LVEDd, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESd, left ventricular end-systolic diameter; LVESV, left ventricular end-systolic volume; PVC, premature ventricular contraction; RF, radiofrequency

median (IQR) LV end-systolic diameter was 39 (13) mm, and mean (SD) number of PVCs was 19 877 (9972) (TABLE 1). In 81 and 5 patients, respectively, β-blockers and amiodarone were ineffective in PVC suppression before ablation. The majority of the study population had concomitant SHD (54%). Idiopathic arrhythmia was present in 46% of patients. The patients were divided according to the origin of PVCs to the outflow tract (OT) group and ventricular (VENT) group. The OT group included patients with the place of origin assessed according to the Josephson classification as the septal regions 3 and 4 or anterior 10 and 12 in the LV outflow tract and 13, 15, 16, and 17 in the right outflow tract. All remaining loci in the left and right ventricles (1, 2, 5, 6, 7, 8, 9, 11, 18, 14) were defined as the ventricular region, and patients with these places of origin were included in the VENT group. Site 17 in the right outflow tract and 4 in the left outflow tract were the most frequent exits of PVC (FIGURE 1). Eight (7.2%) patients had more than 1 morphology targeted: 5 of them had 2, and 3 of them 3 various PVC morphologies (3 patients had ischemic, 2 dilated, and 1 arrhythmogenic right ventricular cardiomyopathy, 1 had myocarditis).

Dilated cardiomyopathy was observed more frequently in the VENT group than in the OT group (43.8% vs 19.2% of patients; $P = 0.01$).

Procedural aspects The main type of ventricular access in the OT group was transvalvular route (52.6%) and in the VENT group, transseptal route (40.6%). The duration of the procedure was similar in both groups (mean [SD], 129 [55] vs 127 [35] min; $P = 0.81$), but the radiation dose was higher in the OT group than in the VENT group (median [IQR] 5973 [8149] cGy/m^2 vs 8447 [5405] cGy/m^2; $P = 0.003$). No other differences between the groups were noted. In 2 cases, a subxiphoid access was used. In 8% of the patients a complex access (either transseptal/transaortic, or transvenous/transaortic, transvenous/transseptal, transseptal/subxiphoid, transseptal/transaortic/subxiphoid) was necessary. The results are shown in TABLE 2.

The daily number of PVCs in both study groups was similar (around 20 000/24 hours). RFCA allowed a significant decrease in the median (IQR) number of PVCs (from 19 837 [12 375] to 405 [1117]; $P < 0.001$). The number of PVCs higher than 20 000/24 hours was an independent factor of LVEF improvement (odds ratio, 3.53; 95% CI, 1.15–10.75; $P = 0.023$). A multivariate 2x2 mixed-design analysis of variance (ANOVA) was used to compare the number of PVCs before and after the procedure depending on the site of arrhythmia origin in the OT and the VENT group. A decrease in the number of PVCs was observed: $F(1; 106) = 340$; $P < 0.001$; $\omega^2 = 0.76$. (TABLE 3, FIGURE 2), irrespective of the site of the PVC exit.

Left ventricular function improvement

An increase in LVEF before and after the procedure was observed in the whole study population: $F(1; 107) = 20.08$; $P < 0.001$; $\omega^2 = 0.15$, that is, an effect of within-subject variable in mixed-design ANOVA. A reduction in LV dimensions was also observed; however, it was significant for the whole group of patients only in case of LVESd (median LVEDd [IQR], from 52.5 [13] to 50 [9.5]; $P = 0.595$; and median LVESd [IQR] from 39 [13] to 35 [11]; $P = 0.002$). On the other hand, reduction in LVEDd was significant in the OT group: from 54 (9) to 51 (12) ($P = 0.015$). Patients in the OT group had an increased ejection fraction after ablation than before (52% [12] vs 58% [13]; $P < 0.001$) in comparison with the VENT group (47% [16] vs 50% [15]; $P = 0.08$; FIGURE 3), despite higher baseline LVEF values before the procedure in the OT group than the VENT group (mean [SD], 52 [12] vs 47 [16]; $P = 0.001$).

After adjustment for preintervention LVEF in the ANCOVA (which included presence and lack of SHD in the model as well), there was a difference in postintervention LVEF depending on arrhythmia origin: $F(1; 104) = 4.88$; $P = 0.029$; $\omega^2 = 0.04$. Adjusted postintervention LVEF was higher in the OT group than in the VENT group (mean [SD], 56.9% [0.98] vs 52.6% [1.7]; data not shown). This analysis took into account effect of SHD presence, which was not significant (LVEF: $F[1; 104] = 2.11$; $P = 0.149$; $\omega^2 < 0.01$) and therefore did not affect corrected post-treatment values. Adjusted (for initial LVEF equal to preintervention mean, that is, 50.37%) postintervention LVEF in the OT group was higher than in the VENT group (mean [SD], 56.8% [0.99] vs 52.4% [1.6]; data not shown).

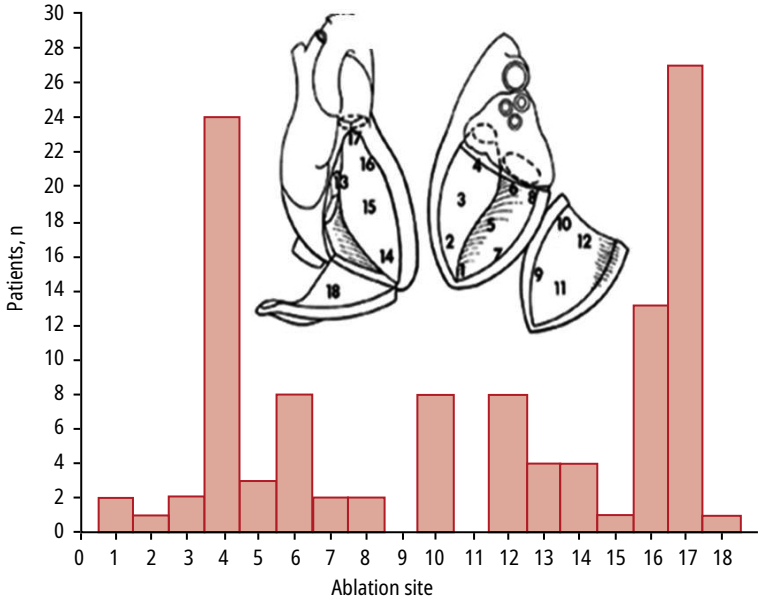

FIGURE 1 Distribution of the origin of ablated premature ventricular contractions in the right and left ventricle

TABLE 2 Characteristics of patients with premature ventricular contractions arising from the outflow tract (OT group) and ventricle (VENT group) (continued on the next page)

Parameter		OT group (n = 78)	VENT group (n = 37)	*P* value
Sex, n	Male	41	20	0.40
	Female	37	17	
Age, y, mean (SD)		55 (16)	57 (17)	0.57
Structural heart disease		39 (50)	21 (57)	0.15
Idiopathic heart disease		38 (49)	11 (30)	0.20
Dilated cardiomyopathy		15 (19)	14 (38)	0.016
Ischemic heart disease		14 (18)	5 (14)	0.99
Myocarditis		1 (1)	1 (3)	0.50
Arrhythmogenic right ventricular cardiomyopathy		4 (5)	1 (3)	0.99
Other than above		5 (6)	0	0.32
NYHA class	I	37 (47)	11 (30)	0.25
	I–II	1 (1)	1 (3)	
	II	22 (28)	14 (38)	
	II–III	4 (5)	0	
	III	7 (9)	5 (14)	
Treatment before the procedure				
β-Blockers		57 (73)	24 (65)	0.99
ACEI		38 (49)	22 (59)	0.06
Statins		20 (26)	8 (22)	0.99
Amiodaron		5 (6)	0	0.32
Sotalol		2 (3)	2 (5)	0.58
Other		52 (67)	23 (62)	0.66
Treatment after the procedure				
β-Blockers		56 (72)	21 (57)	0.65
ACEI		40 (51)	23 (62)	0.06
Statins		21 (27)	8 (22)	0.99
Amiodaron		3 (4)	0	0.56
Sotalol		1 (1)	0	0.99
Other		52 (67)	24 (65)	0.50
Procedural characteristics				
CARTO RMT		15	5	0.79
CARTO		63	27	
Access	TV	41 (53)	6 (16)	0.003
	TSP	29 (37)	13 (35)	
	TAV	5 (6)	6 (16)	
	SBX	0	1 (3)	
	TSP / TAV	0	2 (5)	
	TV / TAV	2 (3)	2 (5)	
	TV / TSP	1 (1)	0	
	TSP / SBX	0	1 (3)	
	TSP / TAV / SBX	0	1 (3)	

TABLE 2 Characteristics of patients with premature ventricular contractions arising from the outflow tract (OT group) and ventricle (VENT group) (continued from the previous page)

Parameter	OT group (n = 78)	VENT group (n = 37)	*P* value
Baseline electrocardiographic, procedural, and echocardiographic parameters			
QRS, ms, median (IQR)	85 (26.3)	90 (22.3)	0.76
PQ, ms, mean (SD)	161 (41)	154 (34)	0.51
QT, ms, mean (SD)	393 (46)	380 (37)	0.25
Procedure time, min, mean (SD)	129 (55)	127 (35)	0.81
RF time, min, median (IQR)	15.3 (21.2)	29.8 (21.6)	0.06
RF dosage, cGy/m^2, median (IQR)	3100 (6608)	7614 (9360)	0.003
RF time, s, median (IQR)	566 (747)	890 (828)	0.3
LVEF >50% before	31 (40)	13 (35)	0.99
LVEF >50% after	20 (26)	7 (19)	0.81

Data are presented as number (percentage) unless otherwise indicated.

Abbreviations: ACEI, aniogiotensin converting enzyme inhibitor; CARTO, electroanatomical navigation system, CARTO RMT, electroanatomical navigation system integrated with steretaxis; NYHA, New York Heart Association; SBX, subxiphoid; TAV, transaortic retrograde; TSP, transseptal; TV, transvenous; others, see TABLE 1

TABLE 3 The premature ventricular contraction (PVC) burden and echocardiographic changes before and after the procedure in the 6-month follow-up in patients with PVCs arising from the outflow tract (OT group) and ventricle (VENT group)

Parameter	OT group	VENT group	*P* value
PVCs before, n, mean (SD)	19 642 (10 297)	20 864 (9425)	0.76
PVCs after, n, median (IQR)	405 (1108)	325 (1084)	0.81
LVEDd before, mm, median (IQR)	52 (13)	53 (15)	0.99
LVEDd after, mm, median (IQR)	50 (10)	53 (14)	0.05
LVESd before, mm, mean (SD)	39 (9)	42 (10)	0.01
LVESd after, mm, median (IQR)	35 (11)	36 (13)	0.09
LVEF before, %, mean (SD)	52 (12)	47 (16)	0.001
LVEF after, %, mean (SD)	58 (13)	50 (15)	0.001
LVEDV before, ml, mean (SD)	130 (48)	143 (59)	0.26
LVESV before, ml, mean (SD)	68 (42)	79 (49)	0.27
LVEDV after, ml, median (IQR)	110 (54)	118 (81)	0.37
LVESV after, ml, median (IQR)	45 (35)	42 (69)	0.37

Abbreviations: see TABLE 1

At least 10% LVEF improvement in echocardiography at follow-up was considered significant. We observed an increase in LVEF in 76 patients (69.1%), no change in 10 (9.1%), and a decrease in 24 (21.8%). In the OT group, 52 patients (66.7%) had an increase in LVEF, 9 (11.5%) had no change, and 17 (21.8%) had LVEF decrease. In the VENT group, 24 patients (75%) had an increase in LVEF, 1 (3.1%) had no change, and 7 (21.9%) had a decrease in LVEF.

There were no correlations between arrhythmia burden and echocardiographic parameters in the whole study group, as well as the subgroups before the procedure. The Spearman rank correlation analysis showed no differences between PVC percentage and LVEF, as well as LVESd and LVEDd. In the study cohort, no difference was observed in the magnitude of LVEF improvement in the subgroups with confirmed SHD (mean [SD] LVEF difference, 5.7% [1.4]) and without SHD (mean [SD] LVEF difference, 4.6% [0.96]; $P = 0.89$).

The overall complication rate (mild and serious) was 1.8%. One woman with arrhythmia coming from the LV outflow tract presented with aortic dissection 3 days after hospital discharge (0.9%). She was successfully operated. Both study groups did not differ in terms of mild complications. Two

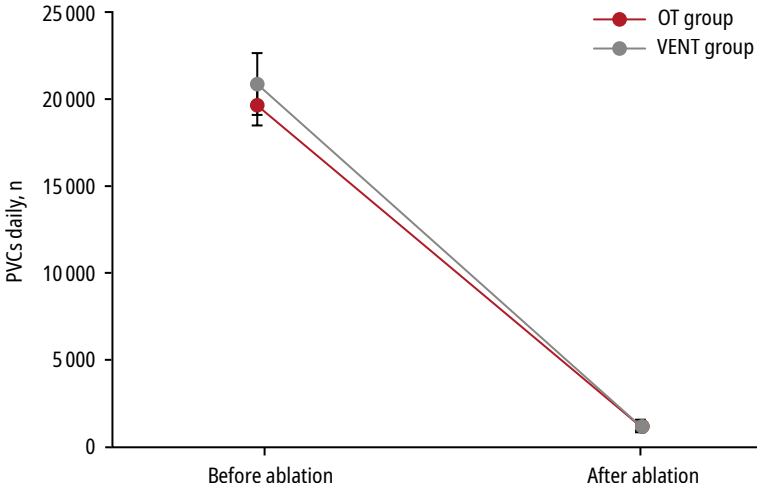

FIGURE 2 Decrease in the daily number of premature ventricular contractions (PVCs) after radiofrequency catheter ablation in patients with PVCs arising from the outflow tract (OT group) and ventricle (VENT group)

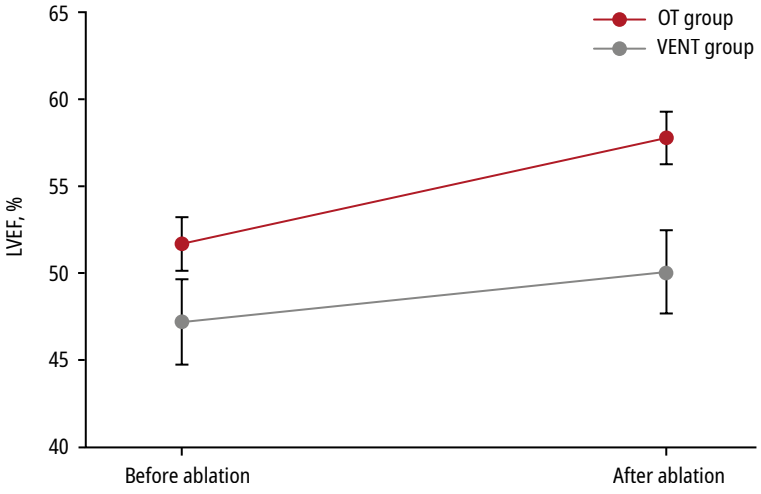

FIGURE 3 Postprocedural change in left ventricular ejection fraction (LVEF) in patients with premature ventricular contractions arising from the outflow tract (OT group) and ventricle (VENT group)

patients (1.8%) had hematomas and 1 atriovenous fistula, which was treated by manual pressure. Two patients (1.8%) presented with conduction disorders during the procedure (in LV origin, it was intermittent atrioventricular conduction type 2 block, retreating, in a patient with the right ventricular outflow tract [RVOT], an asymptomatic right bundle branch block occurred; $P = 0.5$).

DISCUSSION Frequent PVCs are the most common arrhythmia which in certain circumstances may not be benign. They may induce LV dysfunction independent of preexisting SHD. Nevertheless, the mechanisms of tachycardia-induced cardiomyopathy are not fully understood. The study describes the clinical and functional effect of PVC elimination in relation to the arrhythmia exit. RFCA allowed a significant decrease in the mean number of PVCs irrespective of PVC origin. RFCA can be an effective treatment in the outflow tract tachycardia, which is a recommended first-line therapy in symptomatic patients with right sided arrhythmia, both outflow and ventricular origin.[2] Successful RFCA improves quality of life and safety of patients with ventricular arrhythmia with or without concomitant SHD, who are prone to cardiomyopathy.[3] According to the guidelines, RFCA is recommended in symptomatic patients with frequent PVCs if antiarrhythmic therapy is not possible. In patients with left sided arrhythmia it is recommended only after unsuccessful management with first-line pharmacotherapy.[2] Yet, the nature of both origins arrhythmia may be similar, and the efficacy of outflow tract ablation is high.[2-7] There are reports that ablation in the LV may have lower efficacy, especially in the case of epicardial origin.[8]

Left ventricular function improvement

In our study, the successful arrhythmia elimination was connected with the improvement of LVEF. The multivariance ANCOVA demonstrated that the outflow tract PVC origin may be a predictor of LVEF recovery. The postintervention increase in LVEF in the OT group was higher than in the VENT group. Cardiomyopathy is described to be strongly associated with the severity of the hemodynamic derangement associated with the ectopic beats, predominantly the extent of LV dyssynchrony.[9] Some patients might have had long-lasting overlapping tachycardiomyopathy with partially or completely irreversible fibrotic changes in LV. There were more patients with diagnosed SHD in the VENT group, which may contribute to the nonsignificant LV reversal in this group. PVC-induced tachycardiomyopathy may occur not only in patients with SHD, but also in those with preserved ventricular function. One has to be very careful _when assessing young and asymptomatic patients who develop tachycardiomyopathy. Tachycardiomyopathy can be finally diagnosed and treated only after exclusion of other causes of cardiomyopathy and improvement after successful therapy.

Cardiomyopathy related to arrhythmia is reported to be reversible in 80% of cases.[6] In the meta-analysis by Lamba et al,[10] RFCA allowed for LVEF improvement by 10% in PVCs originating in the RVOT. Recent studies have shown that the localization of PVCs had minimal impact on LV dyssynchrony. This may be the result of combination of several factors, such as PVC coupling and abnormal activation sequence.[6] Untill now, long-lasting palpitations, high PVC burden, epicardial origin, younger age, and PVC QRS duration have been assessed to be independent risk factors of tachycardiomyopathy.[8,11-15] The epicardial origin was connected with a delayed recovery of LV function.[9,15] Nonetheless,

the impact of different origin sites of PVCs is poorly understood.

We have observed the relation between more than 20 000 PVCs in 24-hour Holter monitoring and LV improvement. Some studies confirm the relation between PVC burden (10%–20%) and the rate of LVEF positive reversal.[16-19] In patients with successful RFCA therapy, even a 14% increase in LVEF and a 5-mm mean decrease of LVEDd was observed. In patients with preexisting LV dysfunction, the improvement was not as great. Independent factor of better LV improvement were ablation outcome, higher LVEF, and the absence of preexisting dilative cardiomyopathy.[20] In patients who presented depressed LV function before and after ablation, LVEDD greater than 66 mm was a good predictor of irreversible LV cardiomyopathy.[21] Our population represents a heterogeneous group of patients with diverse concomitant diseases and various impact of the extrasystole on the LV, which may explain a lower functional reversal rate.

The adjunctive influence on functional improvement after arrhythmia elimination indicates that not only symptomatic patients with long-lasting severe arrhythmia should be qualified for invasive treatment but also patients with suspected or proven tachycardiomyopathy.

Procedural aspects Arrhythmia originating from the left and right ventricular outflow tracts was connected with shorter RFCA time, shorter fluoroscopy time, and fewer vascular accesses. The LV arrhythmia origin was connected with more demanding techniques, longer procedure and fluoroscopy time, more than one access, and more complications. Difficulties in arrhythmia localization mapping and treatment may demand double or even triple access, which is often the case in PVCs originating in the outflow tract.[22] Proper localization and treatment of arrhythmogenic focus is a crucial part of RFCA. In a prospective study, Pytkowski M et al[23] presented a preablation algorithm of electrocardiographic analysis predicting the site of successful RF RVOT arrhythmias ablation. In a large retrospective Chinese study on safety of RFCA, PVCs with origin in the LV and epicardium were predictors of procedural complications. The overall adverse events rate was 2.7%. Major complications, such as acute coronary syndrome, ventricular fibrillation, cardiac tamponade, pulmonary embolism, infectious endocarditis, stroke, and death, occurred in 1.5%. Moderate adverse events occurred in 1.2% of patients (pseudoaneurysms, retroperitoneal hematoma, thrombogenesis, and pericardial effusion). The most common complication was pericardial effusion, which often progressed into symptomatic tamponade, and was treated by subxiphoid puncture. The total mortality rate was 0.16%. The mortality risk was higher in patients with multiorigin PVCs in the LV.[24] Atrioventricular conduction block occurred in 2 patients. In patients with arrhythmias originating from high risk regions conservative treatment may be the option of choice.[25] In a German registry with a total of 408 procedures, the PVC ablation was successful in 82%. The acute success in patients without SHD was even higher (86% vs 74%; P = 0.002), and the mortality rate of patients with idiopathic heart disease and SHD was different (0 vs 2.3%; P = 0.012). In over 76% of patients, significant improvement was observed.[26]

The RFCA is a safe and effective treatment method with outstanding outcomes in PVC of the outflow tract as well as ventricular origin. Significant reduction of PVCs may improve LV function, not only in patients with confirmed tachycardiomyopathy but also in some patients with preexisting structural impairment or idiopathic ventricular arrhythmia.

Study limitations The greatest limitation of the study is the retrospective nature of the analysis. The analyzed cohort consisted of consecutive patients with PVCs, irrespective of underlying diseases. Furthermore, supplementary data on success rate might have been achieved with longer than 24-hour Holter monitoring, and serial echocardiographic evaluation after 6 and 12 months. Magnetic resonance assessment before and after the procedure might be helpful in the differential diagnosis between SHD and idiopathic heart disease, and could have given more information on overlapping phenomenon and reversible component of tachycardiomyopathy.

Conclusions We compared subgroups of patients with PVCs of different exit site, in order to determine the acute and chronic effect of RFCA in outflow tract and ventricular arrhythmia. The main findings of this study are: 1) the RFCA success rate did not depend on the arrhythmia origin, the PVC improvement was significant in both groups; 2) patients with PVCs originating in the outflow tract presented a greater rate of reverse LV remodeling; 3) arrhythmia originating from the left and right ventricular outflow tract was associated with the shorter RFCA time, shorter fluoroscopy time, and fewer vascular accesses. A major adverse event, aortic dissection, was observed in the LV outflow tract.

ARTICLE INFORMATION

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