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Premature Ventricular Complex Site of Origin and Ablation Outcomes in Patients with Prior Myocardial Infarction

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Abstract:	Background: Frequent premature ventricular complexes (PVCs) are common after a myocardial infarction (MI). PVC ablation data in this population is limited. Methods: 332 patients with frequent PVC and left ventricular (LV) dysfunction were prospectively included and followed for 12 months after ablation. Sixty-seven (20%) patients [63±10 years old, 65 (93%) men] with previous MI were compared with the remaining 265 patients. Results : PVCs in post-MI patients originate predominantly from the LV [92% LV vs 6% right ventricle (RV), p<0.001], the most frequent sites of origin (SOO) being: MI scar [23 (34%) patients] and LV outflow-tract (LVOT) [22 (33%) patients]. A papillary muscle origin was more frequent in post-MI patients (16% vs 4%, p=0.001), while a RV outflow-tract origin was less frequent (1% vs 33%, p<0.001), as compared to patients without MI. In post-MI patients PVC burden decreased from 29±12% at baseline to

	4.6±7% (p<0.001), LVEF improved from 33.6±8% to 42±10% (p<0.001), and NYHA class improved from 2.1±0.7 to 1.4±0.5 points (p<0.001) at 12 months. When compared with the remaining 265 patients, there were no differences in the acute ablation success (85% vs 85%, p=0.45), complication rate (6% vs 6%, p= 0.41) or absolute improvement in LVEF (8.8±10 vs 9.9±11 absolute points, p=0.38) and functional status (0.7±0.7vs 0.6±0.7 NYHA points, p=0.53). Conclusion: PVC ablation significantly improves cardiac function and functional status in post-MI patients. PVCs predominantly originate from the MI scar and LVOT. A papillary muscle SOO was found to be strongly associated to the presence of a previous MI.
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Cover Letter

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Dear Dr. Peng-Sheng Chen, Editor-in-Chief,

Please find enclosed herewith the article entitled **"Premature Ventricular Complex Site of Origin and Ablation Outcomes in Patients with Prior Myocardial Infarction"** to be considered for publication in *Heart Rhythm*, as original clinical research article.

The present study describes the results of a predefined secondary endpoint of a multicenter prospective study on catheter ablation of PVC. The aim of our research was to characterize the PVCs site of origin, the efficacy of PVC ablation and the 12-month outcomes in a consecutive series of patients with frequent PVCs, LV dysfunction and a history of previous MI. Data from this group was compared with that of the remaining patients with frequent PVCs and LV dysfunction without a previous MI

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Yours sincerely,

Antonio Berruezo, MD, PhD
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Premature Ventricular Complex Site of Origin and Ablation Outcomes in Patients with Prior Myocardial Infarction

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ABSTRACT

Background: Frequent premature ventricular complexes (PVCs) are common after a myocardial infarction (MI), but data on PVC ablation in this population is limited.

Methods: 332 patients with frequent PVC and left ventricular (LV) dysfunction were prospectively included and followed for 12 months after ablation. Data from 67 (20%) patients [63±10 years old, 65 (93%) men] with previous MI were compared with the remaining 265 patients.

Results: PVCs in post-MI patients originate predominantly from the LV [92% LV vs 6% right ventricle (RV), p<0.001], the most frequent sites of origin (SOO) being MI scar [23 (34%) patients] and the LV outflow-tract (LVOT) [22 (33%) patients]. A papillary muscle origin was more frequent in post-MI patients (16% vs 4%, p=0.001), while a RV outflow-tract (RVOT) origin was less frequent (1% vs 33%, p<0.001), as compared to patients without MI. In post-MI patients PVC burden decreased from 29±12% at baseline to 4.6±7% (p<0.001), LVEF improved from 33.6±8% to 42±10% (p<0.001), and NYHA class improved from 2.1±0.7 to 1.4±0.5 points (p<0.001) at 12 months. When compared with the remaining 265 patients, there were no differences in the acute ablation success (85% vs 85%, p=0.45), complication rate (6% vs 6%, p= 0.41) or absolute improvement in LVEF (8.8±10 vs 9.9±11 absolute points, p=0.38) and functional status (0.7±0.7vs 0.6±0.7 NYHA points, p=0.53).

Conclusion: PVC ablation significantly improves cardiac function and functional status in post-MI patients. PVCs predominantly originate from the MI scar and LVOT. A papillary muscle SOO was found to be strongly associated to the presence of a previous MI.

Keywords: premature ventricular complex; ablation; left ventricular dysfunction; myocardial infarction, scar burden.

INTRODUCTION

Frequent PVC ablation improves cardiac function in patients with depressed LVEF.[1,2] This benefit was initially observed in patients with PVC-induced cardiomyopathy. Increasing evidence suggests that patients presenting with a previous structural heart disease (SHD) worsened by frequent PVCs can also obtain a significant benefit in symptoms, functional capacity and LVEF improvement after catheter ablation.[3,4] In consequence, the current consensus document considers PVC ablation as a Class IIa recommendation in patients with SHD in whom frequent PVCs are suspected to be contributing to the LV dysfunction.[5] There is also data showing that PVC burden is significantly higher in the post-MI setting as compared with the general population.[6] Moreover, several studies have found an association between frequent PVCs and a worse prognosis in patients with a prior MI.[7] Nevertheless, little is known about PVC site of origin (SOO) or the response to PVC ablation in this specific population. A previous study by Sarrazin et al. showed an improvement of LVEF after PVC ablation in 15 patients with a prior MI and LV dysfunction as compared with a control group without PVCs.[8] Yet, these results have not been confirmed in larger patient populations. The aim of the present study was to characterize the PVCs SOO, the efficacy of PVC ablation and the 12-month outcomes in a consecutive series of patients with frequent PVCs, LV dysfunction and a history of previous MI. Data from this group was compared with that of the remaining patients with frequent PVCs and LV dysfunction without a previous MI.

METHODS

Patient sample

We report the results of a predefined secondary endpoint of a multicenter prospective study.[3] Between November 2010 and December 2018, 332 consecutive patients with frequent PVCs and LV dysfunction were prospectively included in 8 international centers and followed for 12 months after ablation. The inclusion criteria were presence of LV systolic dysfunction (LVEF < 50%); frequent PVCs (defined as a burden of more than 4% at baseline 24-hour Holter monitoring); receiving optimal medical therapy for heart failure at maximum tolerated dose at the time of study inclusion. No patient was excluded because of the number of PVC morphologies or because of the presumed SOO based on electrocardiographic criteria. All patients underwent a first catheter ablation procedure. Clinical, electrocardiographic and electrophysiological variables were prospectively collected. The present study reports the clinical and procedure characteristics, as well as the 1-year follow-up, of the 67 (20%) consecutive patients with a previous MI. The study protocol was approved by the Local Ethics Committee of each participating center and all participants signed the written informed consent.

Baseline evaluation

Baseline evaluation included medical history, appropriateness of treatment, functional status assessment by means of NYHA class, 12-lead ECG, 24-hour Holter, echocardiography to evaluate LV function (LVEF was computed with the Simpson method on three different beats to avoid bias due to PVC) LV size and segmental wall motion abnormalities, and assessment of blood levels of brain natriuretic peptide (BNP).

Ablation procedure

Anti-arrhythmic drugs, other than amiodarone, were withdrawn for 5 half-lives. Ablation was guided by the CARTO navigation system (Biosense Webster, Diamond Bar, CA USA), using a 3.5-mm irrigated-tip catheter (NaviStar® or SmartTouch®, Biosense Webster). Ablation site selection was based on activation or pacemap, depending on PVC burden during the ablation procedure. Ablation was considered successful if the targeted PVC was eliminated and non-inducible after isoproterenol infusion. Patients were monitored for 20 minutes at the end of the procedure to ensure complete PVC abolition. The site where radiofrequency application eliminated the PVC was considered as the SOO. Given the fact that the entire population of the study had previous MI and LV dysfunction, therapy with beta-blockers was maintained, regardless of the ablation success.

Scar characterization

In a subgroup of 23 patients with a previous MI, contrast-enhanced cardiac magnetic resonance (LGE-CMR) study was performed using either a 1.5 or a 3 T clinical scanner. All LGE-CMR images were analyzed using a previously described technique.[9] Full LV volume was reconstructed in the axial orientation, and the resulting images were processed with ADAS 3D LV™ software (ADAS3D Medical, Barcelona, Spain). A color-coded pixel signal intensity map based on LGE-CMR images was projected to a 3D LV shell following a trilinear interpolation algorithm. Scar core zone (CZ) and border zone (BZ) were characterized using 40% and 60% of the maximum intensity as thresholds. Scar mass (total scar, BZ and CZ) was automatically measured using ADAS-VT software. Scar heterogeneity was defined as BZ/total scar mass. To compare the scar characteristics in the LGE-CMR, a control group of 23 post-MI patients in a chronic stable phase without PVCs, matched by LVEF and MI localization was analyzed.

Follow-up

Follow-up was performed at the outpatient clinic at 6 and 12 months after catheter ablation and included a functional evaluation, transthoracic echocardiography, 24-hour Holter monitoring and BNP blood level quantification. Echocardiographic response was defined as at least 5 absolute points improvement in the LVEF. Sustained successful ablation (SSA) was defined as an acute successful ablation with a persistent abolition of at least 80% of the PVC burden at both 6- and 12-month Holter monitoring. Mean PVC reduction was defined as the difference between the baseline PVC burden and the average 6- and 12-months PVC burden in the Holter monitoring.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation or median (range or interquartile range if data are skewed). Categorical variables are presented as total number and percentages. To compare means of two variables, Student *t* test was used (or Wilcoxon when applicable). Proportions were compared using Chi-square or Fisher exact test, as appropriate. Friedman analysis of variance by ranks was used for repeated measures. P-values < 0.05 were considered significant. Statistical analysis was performed using SPSS Statistics, Version 22.0 (Armonk, NY).

RESULTS

Patient population

Three hundred thirty-two consecutive patients with LV dysfunction referred for frequent PVC ablation were included. Sixty-seven patients [63±10 years old, 65 (93%) men] had a previous MI [26 (39%) anterior MI]. In this group, mean LVEF was 33.6±8 % and mean baseline PVC burden was 29±12%. Baseline characteristics of these patients are summarized in Table 1. As compared with the remaining 265 patients, post-MI patients were older (63± 10 vs 56 ± 13 years old, $p<0.001$), more frequently men [63 (94%) vs 177 (67%), $p<0.001$], had a higher PVC burden (29±12% vs 24±12%, $p=0.005$), a wider intrinsic QRS (122±32 ms vs 112±27 ms, $p=0.04$) and PVC QRS (170±25 vs 163±22 ms, $p=0.04$).

Ablation procedure

Of the 67 post-MI patients, acute successful ablation was achieved in 57 (85%), whereas PVC was not completely eliminated in 8 (12%) patients. In the remaining 2 (3%) patients no ablation attempt was performed due to insufficient PVC burden during the procedure. The PVC SOO was located in the LV in 60 (90%) patients; in the RV in 4 cases (6%); and in 3 (4%) patients it could not be conclusively determined, given the fact that they originated intramurally at the interventricular septum (same EGM precocity from the RVOT and LVOT septum and incomplete PVC abolition after RF application from both sides). Thus, the most frequent location was the LV, with the infarct scar accounting for 23 (34%) cases, followed by the LVOT with 22 (33%) cases [8 aortic cusp, 8 myocardium immediately bellow the aortic cusp, and 6 LV summit], and the papillary muscle in 11 (16%) patients. Figure 1 shows the SOO distribution of the ablated PVCs. Procedure related complications occurred in 4 (6%) patients (1 arterio-venous fistula, 1 femoral pseudoaneurysm, 1 pericardial effusion requiring

pericardiocentesis and 1 atrio-ventricular block that required biventricular pacemaker implantation).

When compared with the remaining 265 patients without a previous MI, there were no differences in the acute success (85% vs 85%, p=0.45) or the complication rate (6% vs 6%, p= 0.41). In contrast, there were significant differences regarding the PVC SOO, since in post-MI patients PVCs originated more frequently from the LV (92% vs 60%, p<0.001). Table 2 shows the SOO distribution in patients with and without a previous MI. Post-MI patients more frequently presented PVC originating in the myocardial scar (34% vs 3%, P<0.001) and in the papillary muscle (16% vs 4%, p=0.001). Moreover, in all the post-MI patients with papillary muscle-PVCs who had a preprocedural CMR (6 patients, 55%) the papillary muscle was included in the infarct zone showing delayed enhancement, suggesting a role of myocardial scar in the genesis of the arrhythmia also in those patients.

On the other hand, a RVOT origin was significantly less frequent in post-MI patients (1% vs 33%, p<0.001). In 96% of the post-MI patients with an origin in the outflow tract the PVC SOO was the LVOT, as opposed to the 59% in the rest of the population (p=0.001).

12 months Follow-up

In post-MI patients, PVC burden decreased from 29±12% to 4.6±7% at 1-year follow-up, (p<0.001). Forty-four (66%) patients had a successful sustained ablation (SSA). Mean PVC burden reduction was 23±14 points. A significant improvement of LV systolic function was observed in this population with an increase in LVEF from 33.6±8% at baseline to 42±10% at 12 months (p<0.001). Mean LVEF improvement was 8.8±10 absolute points. At the end of the follow-up, 34 (51%) patients were considered echocardiographic responders and 7 (10%) patients normalized their LV systolic

function (LVEF>50%). Of the 67 post-MI patients, 41 (61%) had a LVEF ≤ 35%. Of them, twenty-five (61%) had an improvement to a LVEF > 35%.

Although BNP levels decreased from 272 pg/mL [IQR 95-590] at baseline to 75 pg/mL [IQR 56-260] at 12 months (p=0.8), this improvement did not reach statistical significance. Functional status also improved after ablation from 2.1±0.7 points of NYHA class at baseline to 1.4±0.5 points at 12 months, p<0.001, as Figure 2 shows.

The most substantial improvement after ablation occurred in the subgroup of patients with a SSA (see Figure 2). In this group mean LVEF improvement was 11±10%, as LVEF raised from 33.8 ±8% at baseline to 44.8±9% at 12 months (p<0.001). There was also a significant reduction in the LV end-diastolic diameter from 60.2±5 to 57.5±6 mm (p=0.02) and BNP levels from 279 ±246 to 130±165 pg/mL, (p=0.001) in patients with SSA. Conversely, patients without a SSA increased the LV end-diastolic diameter from 62 ±17 to 64 ±7 mm as well as BNP levels from 533±511 to 785±1500 pg/mL, without reaching statistical significance.

There were no differences in the absolute improvement in the LVEF (8.8±10 vs 9.9±11 points, p=0.38) or the functional status (0.7±0.7vs 0.6±0.7 NYHA points, p=0.53) after ablation, between the post-MI subgroup and the remaining 265 patients.

Scar characterization

A baseline LGE-CMR was performed in a subgroup of 23 patients for a quantitative and qualitative analysis of the scar. Patients of this group were 60±8 years old, predominantly men [22 (96%)] and had a severe LV dysfunction (LVEF 34.6±7%) and a high PVC burden (24±11%). A population of 23 post-MI patients, matched by MI localization and LVEF, but without PVCs was used as control group. Compared with the control group, patients with frequent PVCs had smaller scar mass (13.8±13 vs 26.8±13 g, p=0.002) as well as smaller scar core mass (5.7±6 vs 13±7 g, p<0.001) and

222 border zone mass (8.3±9 vs 13.5±7 g, p=0.046), as Figure 3 shows. Scar heterogeneity
223 was higher in the PVC group (55±20% vs 49±13%), without reaching statistical
224 significance (p=0.08).

DISCUSSION

The present study reports the largest series of PVC ablation in post-MI patients with LV dysfunction. Ablation can be safely performed in this population with a good acute success rate. Most of the PVCs originate in the LV, the MI scar and the LVOT being the most frequent SOO. Patients importantly and significantly improved LVEF and functional status at long-term, without differences in the degree of improvement as compared with patients without a history of MI. Finally, the scar mass in post-MI patients with frequent PVCs is smaller when compared with that of patients with a previous MI and the same degree of LV dysfunction but without PVCs, thus reflecting the "PVC-worsening" effect in the remote myocardium.

PVC and Myocardial infarction

It is already known that PVC burden increases after a MI and is associated with worse prognosis in this population.[10] Given the poor outcomes obtained in original trials treating PVCs with antiarrhythmic drugs in post-MI patients, frequent PVCs were disregarded in the clinical management of this population.[11,12] Nevertheless, increasing evidence supports the beneficial role of catheter ablation on cardiac function in patients with a high PVC burden and decreased LVEF, including those patients with underlying SHD.[3,13,14] Sarrazin et al[8] previously published a study of 15 post-MI patients with LV dysfunction and frequent PVCs undergoing catheter ablation. These patients significantly improved LVEF after PVC abolition (most of them normalized the LVEF) as compared with those of a control group who did not. The results of the present study are in line with this previous observation. However, only 7 (10%) normalized the LVEF at the end of the follow-up, presumably due to the irreversible injury from the previous MI. Even if these patients rarely normalize cardiac function after ablation the magnitude of the benefit in terms of LVEF recovery justifies the indication for ablation. Moreover,

in post-MI patients with a SSA, there was a decrease in the LV end-diastolic diameter as well as a significant improvement in the neurohormonal status, which further corroborates the benefit of PVC abolition in this population.

Finally, the benefit in terms of cardiac function recovery in post-MI is equivalent to the one obtained in patients without a MI, despite of having a stablished myocardial scar. It has been previously shown that mean PVC burden reduction is the most important predictor of response after ablation and that myocardial scar only modulates the probability of response, playing a role only when a significant PVC burden reduction cannot be achieved.[15] The mean PVC burden reduction in post-MI patients was 23±14 points in the present study. Therefore, the important PVC burden reduction and the smaller than expected scar burden in this population explain this observation.

PVC site of origin

A LV SOO was more often found in the post-MI patients as compared with the rest of the study population, with a RV SOO in only 6% of cases. The most frequent SOO was the MI scar (23 patients, 34%), in line with the previous observation of Sarrazin et al.[8] The second most frequent SOO in post-MI patients was the LVOT. The vast majority of outflow tract PVCs in this population arose from the LV as opposed to the group without MI. This higher proportion of LVOT PVCs could be explained by a chronic LVOT overload in the post-MI population. Our group previously reported that certain classic cardiovascular risk factors as hypertension, male gender or aging can induce anatomical modifications in LVOT/aortic root due to chronic LV overload, predisposing to LVOT ventricular arrhythmias.[16,17] This mechanism can explain the higher percentage of LVOT PVCs in the post-MI population, which is usually constituted of advanced-age males with cardiovascular risk factors, especially in those patients with LV dysfunction.

The third more frequent SOO found in MI-patients was the papillary muscles. This particular location was more frequently found in post-MI patients as compared to those without MI (16% vs 4% respectively, $p<0.001$). Fibrosis was frequently found in the papillary muscle of post-MI patients with this SOO, suggesting that scarred tissue plays a role in the genesis of the PVC. The strong association between papillary muscle-PVCs and a previous MI indicates the need to exclude or confirm MI scarring in patients with frequent PVCs and ECG suggesting a papillary muscle SOO.

Myocardial scar characterization

Sarrazin et al. reported that postinfarcted patients with frequent PVCs have a small scar burden compared with the same patient population without PVCs. The results of the present study are in line with this previous observation. In addition, all components of the scar (dense scar and border zone) are smaller in patients with frequent PVCs than in a control group matched by LVEF. These findings suggest that LVEF cannot be considered an established marker of LV dysfunction in patients with frequent PVCs, as the irreversible cardiac damage caused by the previous MI is usually lower than expected. Moreover, post-MI patients showed a trend to have a more heterogeneous scar. Due to the small number of patients studied with a CMR, we cannot evaluate the association between scar heterogenicity and a PVC origin from the scar area. Further studies focused on imaging are needed to investigate this potential association.

Clinical Implications

The present study has several clinical implications. Even if the consensus document recommends catheter ablation in patients with SHD in whom frequent PVCs are suspected to be contributing to the LV dysfunction,[5] patients with a previous MI are underrepresented in the main series of PVC ablation reported in the literature and often are not considered as candidates for ablation in the clinical practice. The results of the

present study endorse that they should be, as the benefit after ablation is equivalent to that of patients without MI and it has been recently reported that the persistent PVC abolition results in a decrease in morbimortality.[18] This benefit could be especially relevant in post-MI patients, as there is consistent evidence that frequent PVCs are associated with particularly worse prognosis in this population.

LVEF is considered the most relevant prognostic variable in patients with LV dysfunction after MI. However, LVEF could be significantly affected by some factors as, for example, completing revascularization or up-titration of medical heart failure therapy. In these scenarios, clinical guidelines recommend to complete therapy and reevaluate LVEF. The result of the present study shows that cardiac function is also highly influenced by the PVC "worsening" effect. Therefore, in presence of frequent PVCs, LVEF should be cautiously taken into account for clinical decision-making before ablation. This observation can be especially relevant in case of indication for ICD implantation for primary prevention,[19] as in our population most of patients with a severely depressed cardiac function at baseline improved LVEF to more than 35% after PVC abolition.

Finally, the results of the present study suggest that a LV origin should be suspected in all patients with a prior MI. This is especially relevant when the 12 -lead ECG suggests an origin in the outflow tract, as only 1 post-MI patient in this study had RVOT-PVC. Accordingly, mapping the LVOT as the first step of the procedure is a reasonable option in post-MI patients with an outflow tract arrhythmia.

Study Limitations

The electrophysiological mechanism of the PVC was not systematically evaluated. Therefore, we cannot know whether the main mechanism of the PVC in patients with MI is reentrant or trigger activity, especially in those PVCs arising from the myocardial

scar. However, the SOO distribution suggests that an important proportion of post-MI patients have “idiopathic” PVCs. Equally important, CMR data was only available for a small percentage of post-MI patients. Therefore, further studies are needed to evaluate the role of the myocardial scar in the response to the ablation in this specific clinical scenario, as well as the role of the scar heterogeneity in the scar-related origin of the PVC.

CONCLUSIONS

PVC ablation in post-MI patients significantly improves cardiac function and functional status. The benefit obtained in this population is equivalent to that obtained in patients without a previous MI. Most of the PVCs in this population arise from the LV, being the MI scar and the LVOT the most frequent sites of origin. Post-MI PVCs originate more frequently from the papillary muscle than in the rest of patients with PVCs. The scar amount in post-MI patients with frequent PVCs is usually lower than expected by the degree of LV dysfunction.

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414

415 **Table 1.** Baseline characteristics of patients with and without previous myocardial infarction.

	Prior MI (n = 67)	**No prior MI (n = 265)**	**Total (n = 332)**	**P value**
Age (years)	63± 10	56 ± 13	58 ± 13	<0.001
Sex (male)	63 (94%)	177 (67%)	240 (72%)	<0.001
LVEF (%)	33.6± 88	35.6 ± 9	35 ± 8	0.8
NYHA class	2.2 ± 0.7	2 ± 0.7	2.04 ± 0.7	0.87
LVEDD (mm)	62 ±11	60 ± 7	61 ± 8	0.36
Monomorphic PVC	51 (76%)	193 (73%)	244 (73%)	0.7
PVC Holter (%)	29± 12	24 ± 12	25 ± 12	0.005
History of AF	10 (15%)	26 (10%)	36 (11%)	0.26
SHD	67 (100%)	45 (17%)	112 (34%)	<0.001
PVC-QRS (ms)	170 ± 25	163 ± 22	165 ± 22	0.04
Intrinsic QRS (ms)	122 ± 32	112±27	114±28	0.04
Treatment with: • **Betablockers** • **ACEI** • **Amiodarone**	 63 (95%) 61 (91%) 19 (28%)	 229 (86%) 224 (84%) 21 (8%)	 292 (88%) 285 (86%) 40 (12%)	 0.43 0.17 0.24

416

417 MI: myocardial infarction; LVEF: left ventricular ejection fraction; NYHA: New York Heart
418 Association; PVC: premature ventricular complex; SHD: structural heart disease; LVEDD: left
419 ventricular end-diastolic diameter; ACEI: Angiotensin-converting-enzyme inhibitors.

420

421 **Table 2.** Site of origin distribution in patients with and without previous myocardial infarction.

	Prior MI n= 67	No prior MI (n = 265)	Total (n = 332)	P value
RVOT	1 (1.5%)	88 (33%)	89 (27%)	<0.001*
Peri tricuspid	1 (1.5%)	6 (2%)	7 (2%)	1
Aortic cusp	8 (12%)	57 (22%)	65 (20%)	0.09
M-A union	8 (12%)	38 (14%)	46 (14%)	1
LV summit	6 (9%)	31 (12%)	37 (11%)	0.66
Peri Mitral	4 (6%)	6 (2%)	10 (3%)	0.12
Papillary muscle	11 (16%)	10 (4%)	21(6%)	0.001*
Myocardial Scar	23 (34%)	7 (3%)	30 (9%)	<0.001*
Cardiac Crux	0	2 (1%)	2 (0.5%)	1
Peri His	2 (3%)	5 (2%)	7 (2%)	0.62
Unknown	3 (4.5%)	10 (4%)	13 (4%)	0.73

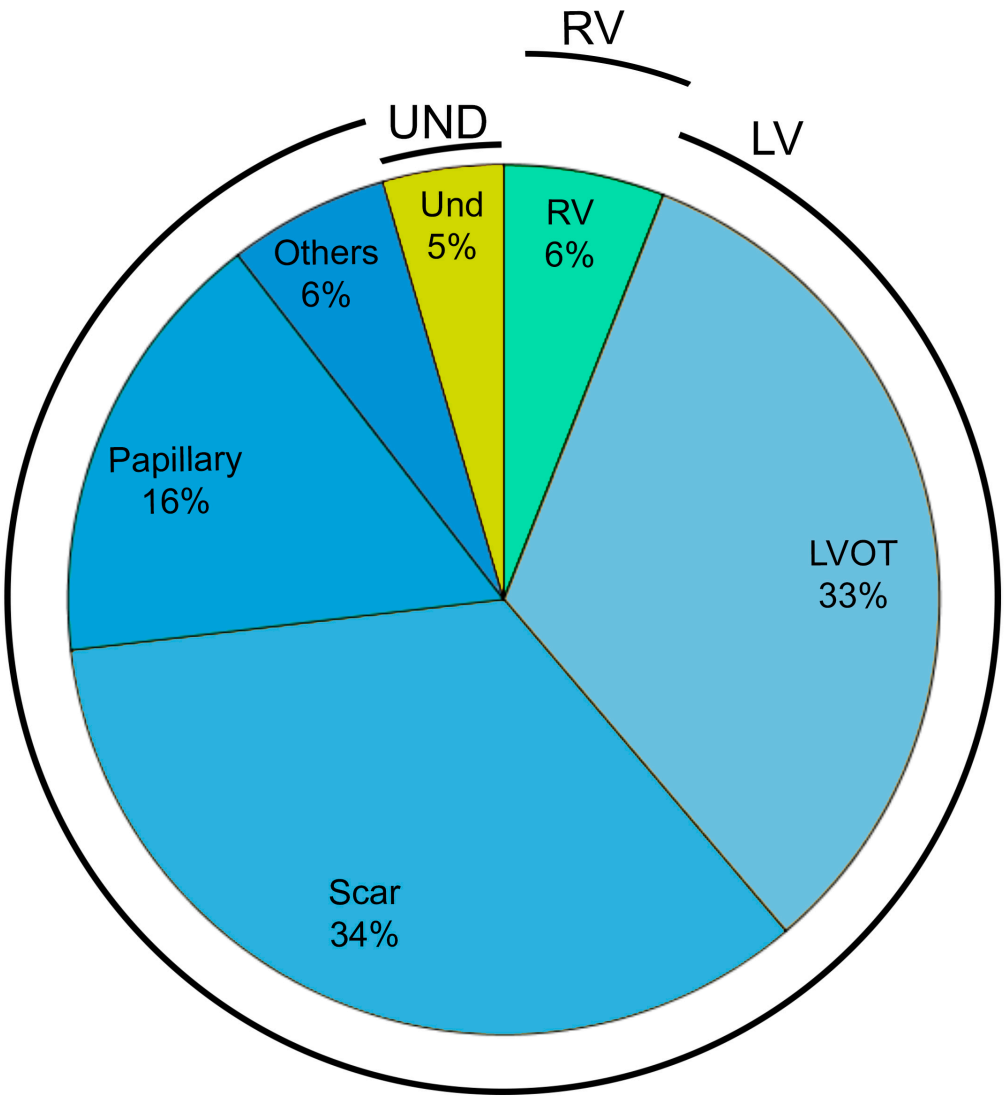
422

423 MI: myocardial infarction; RVOT: right ventricle outflow tract; M-A: mitroaortic; LV_: left

424 ventricular.

425

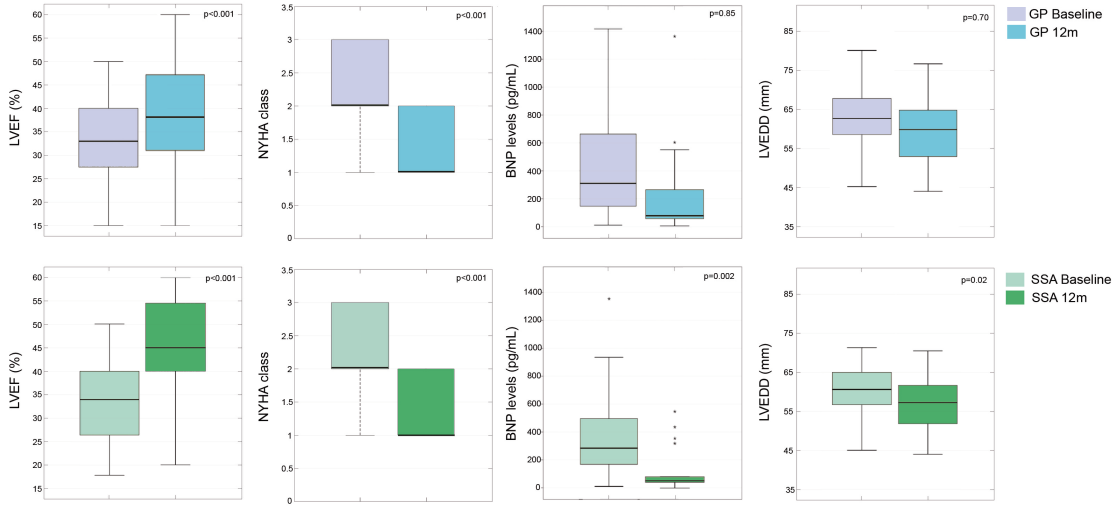
426 **Figure 1.** Site of origin of premature ventricular complex in postinfarction patients.



427 LVOT: left ventricular outflow tract; RV: right ventricle; Und: undetermined.

428

Figure 2. Evolution of LVEF, NYHA class, BNP levels and LVEDD from baseline to 12 months in patients with (top) and without successful sustained ablation (bottom).



BNP = Brain natriuretic peptide; LVEDD = Left ventricular end-diastolic volume; LVEF = Left ventricular ejection fraction; NHYA = New York Heart Association.

Figure 3. Comparison of LVEF and scar characteristics (total scar mass, border zone mass and core mass) in patients with frequent PVCs and post-MI controls without PVCs matched for LVEF. On the right, scar characterization with a 3D-scar map in a control patient without PVCs (top) and a patient with an inferior infarction and frequent PVCs (bottom) depicting scar core (CZ red) and border zone (BZ blue).