# Efficacy and benefits of catheter ablation of ventricular premature complexes in patients younger and older than 65 years of age

## Ventrikül erken vurularının kateter ablasyonu ile tedavisinin 65 yaşından büyük ve küçük olan hastalarda etkinlik ve faydaları

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## ABSTRACT

***Objectives:*** Catheter ablation of ventricular premature complexes (VPC) improves clinical status and systolic performance of the left ventricle (LV) in a certain subset of patients; however, whether or not VPC ablation is equally effective in younger (≤65 years) and older (>65 years) patients remains unclear. We aimed to assess the clinical benefits of catheter ablation of VPCs in elderly patients.

***Study design:*** Fifty-one consecutive patients (66±10 years, 49 male) who underwent catheter ablation for symptomatic VPCs were included into the study. Twenty-seven patients were aged >65 years and 24 patients ≤65 years. Frequency of VPCs per total heart beats by 24-hour Holter monitoring, LV ejection fraction (LVEF) and end-systolic diameters (LVEDD) were evaluated before and 6±3 months after ablation.

***Results:*** The pre-ablation 24-hour VPC burden and VPC number were significantly higher in patients >65 years compared to those ≤65 years (31±15.3 *vs.* 21.9±12.6, p=0.04 and 34493±21226 *vs.* 23554±13792, p=0.026, respectively). At the follow-up after catheter ablation, the mean VPC burden had decreased to 9.1±10.3% (p<0.001) in patients >65 years and to 3.8±7.1 (p<0.001) in patients ≤65 years. Mean LVEF showed a significant increase in both groups after ablation (43.4±10.4 *vs.* 51.5±8.2, p=0.005 for age >65 years and 40.8±13.2 *vs.* 49.5±11.8, p=0.003 for age ≤65 years). The improvement in LVEF was accompanied by a significant decrease in LVEDD (p=0.032 for age >65 years and p=0.047 for ≤65 years).

***Conclusion:*** Catheter ablation is effective for treatment of frequent VPCs in all age groups.

## ÖZET

***Amaç:*** Kateter ablasyonu ile ventrikül erken vurularının (VEV) tedavisi bazı hastalarda klinik durumda ve sol ventrikül (SV) performansında düzelme ile sonuçlanır. Ancak VEV ablasyonunun genç (≤65 yaş) ve yaşlı (>65 yaş) hastalarda aynı düzeyde etkili olup olmadığı henüz bilinmemektedir. Bu çalışmada, kateter ablasyonu yolu ile VEV tedavisinin yaşlı hastalardaki klinik faydalarını değerlendirdik.

***Çalışma planı:*** Semptomlu VEV nedeniyle kateter ablasyonu uygulanmış olan 51 hasta (66±10 yaş, 49 erkek) çalışmaya dahil edildi. Yirmi yedi hasta >65 yaş gurubunda iken, 24 hasta ≤65 yaş idi. Yirmi dört saatlik Holter monitörizasyondaki VEV sıklığı, SV ejeksiyon fraksiyonu (EF) ve SV diyastol sonu çapı ablasyon öncesi ve ablasyondan 6±3 ay sonra değerlendirildi.

***Bulgular:*** Ablasyon öncesi 24 saatlik VEV yükü ve VEV sayısı >65 yaş olan hastalarda ≤65 yaş olanlara göre anlamlı şekilde yüksek bulundu (sırasıyla, 31±15.3 ve 21.9±12.6, p=0.04 ve 34493±21226 ve 23554±13792, p=0.026). Ablasyon sonrası takipte ortalama VEV yükü >65 yaş olan hastalarda %9.1±10.3'e (p<0.001), ≤65 yaş gurubunda ise %3.8±7.1'e geriledi (p<0.001). Ortalama SVEF her iki gurupta anlamlı şekilde artış gösterdi (>65 yaş hastalar için 43.4±10.4 ve 51.5±8.2, p=0.005 ve <65 yaş hastalar için 40.8±13.2 ve 49.5±11.8, p=0.003). SVEF'deki düzelmeye SV diyastol sonu çapında anlamlı gerileme eşlik etti (>65 yaş için p=0.032 ve <65 yaş için p=0.047).

***Sonuç:*** Kateter ablasyonu tüm yaş guruplarında VEV tedavisi için etkin bir yöntemdir.

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Increased burden of ventricular premature complexes (VPCs) have been identified as a possible cause of left ventricular (LV) systolic dysfunction.[1] Prior studies have shown that marked decreases in VPC burden result in improvement in LV systolic function and in a decrease in symptoms in a certain subset of patients.[2,3]

In the last decade, catheter-based ablation therapy has become a safe and effective treatment option for patients with symptomatic VPCs.[3] With improved safety, the therapy has the potential to be a first-line therapy for older populations since antiarrhythmic therapy is often complicated by serious adverse effects of these drugs in this age group.[4] However, arrhythmia mechanisms, medical comorbidities, and safety of the ablation therapy may vary in the elderly population.

The purpose of this study was to assess and compare the short-term outcome of catheter ablation of frequent VPCs in younger and older patients.

## PATIENTS AND METHODS

### Patient characteristics

We enrolled 69 consecutive patients who underwent catheter ablation for the treatment of symptomatic frequent VPCs between March 2008 and April 2012. We excluded patients with atrial fibrillation/flutter (n=6), those with permanent pacemaker therapy (n=4) and patients with inadequate echocardiographic images (n=8). The final study cohort included 51 patients. This study was approved by the Institutional Research Board of our center.

### Holter recordings

The VPC burden was calculated as the number of VPCs divided by the number of all QRS complexes during 24 hours x 100 and was measured using a 24-hour Holter monitor before and after the ablation. A decrease of >80% in the VPC burden was defined as a successful outcome. All class I and class III antiarrhythmic drugs were withheld >5 half-lives before the procedure.

### Transthoracic echocardiography

All patients underwent transthoracic echocardiography (TTE) before and 6±3 months after ablation. Echocardiography was performed using standard views and harmonic imaging (Sequoia, Siemens, Mountain View, CA, USA). Left atrium (LA) maximum anteroposterior diameter was measured in the parasternal long-axis views. The LV end-diastolic and end-systolic volumes, LV stroke volume index, and LV ejection fraction (EF; calculated by Simpson's method) were measured in the apical four-chamber view. LA volumes were calculated by two-dimensional echocardiography by the biplane area-length method. Maximal LA volume (LAVmax) was obtained just before mitral valve opening. Minimal LA volume (LAVmin) was determined at the time of mitral valve closure. LA areas (A1, A2) and supero-inferior longitudinal diameters were measured from apical four- and two-chamber views. LAVs were calculated by the following formula: LAV = 0.85 x A1 x A2/L, where L is the shorter supero-inferior diameter measured from the midline of the mitral annulus to the opposite wall of the LA.[5] LAV index (LAVI) was calculated by dividing maximal LA volume by the body surface area (LAVmax/BSA).

**Abbreviations:**

| | |
|---|---|
| *BSA* | *Body surface area* |
| *EF* | *Ejection fraction* |
| *ICE* | *Intracardiac echocardiography* |
| *LA* | *Left atrium* |
| *LV* | *Left ventricular* |
| *LAV* | *LA volume* |
| *LAVI* | *LAV index* |
| *TTE* | *Transthoracic echocardiography* |
| *VPC* | *Ventricular premature complex* |

### Mapping and ablation procedure

The sites of VPCs were mapped based on the morphology of the VPCs as described previously.[6] Right or left ventricles and/or outflow tracts were reconstructed using Soundstar 3D Catheter, which integrates real-time intracardiac echocardiography (ICE) imaging (CARTO Sound, Biosense Webster, Diamond Bar, CA); Fast Anatomical Mapping (FAM, Biosense Webster, Diamond Bar, CA, USA), multi-electrode array catheter with EnSite non-contact mapping, or NAVX was used in certain patients (EnSite, St. Jude Medical, St. Paul, MN, USA).

If frequent or infrequent VPCs were seen during the procedure, activation mapping was performed. If no VPCs were seen or only rare VPCs, which resulted in difficulty in mapping, pace-mapping was conducted to locate the origin of the clinical VPC in question. Both bipolar and unipolar signals were obtained to accurately map the site of origin. An aortogram was performed to identify the location of cusps as well as coronary ostia if the VPCs were close to those areas. In addition, this was also confirmed by the ICE. Ablation was

performed using 4 mm bidirectional Navistar catheter (Biosense Webster, Diamond Bar, CA) or 4 mm 7Fr Blazer II ablation catheter (Boston Scientific, Natick, MA, USA). The ablation was performed with 15 to 50 watts 50-55 °C, based on the location of the ablation.

All patients in this study had acute procedural success, defined as absence of spontaneous or inducible VPCs with isoproterenol infusion. Patients were reassessed for 30 minutes following the successful ablation to confirm no recurrence of VPCs.

## Statistical analysis

Statistical analysis was performed using STATA11 (Stata-Corp, College Station, TX, USA). Categorical variables are presented as number or percentages, whereas continuous variables are presented as mean ± standard deviation. Pearson chi-square or Fisher's exact tests were used to assess the statistical significance of categorical data. Student's t test was used to test the statistical significance of continuous variables. A comparison of the LAV and echocardiographic vari-

**Table 1. Baseline clinical and VPC characteristics of the patients**

| | Age >65 years (n=27) | | Age ≤65 years (n=24) | | *p* |
|---|---|---|---|---|---|
| | n | % | n | % | |
| Age (years) | 73±6 | | 57±7 | | **<0.001** |
| Gender (male) | 26 | | 23 | | 0.93 |
| Coronary artery disease | 8 | 30 | 7 | 30 | 1.0 |
| Cardiomyopathy | 15 | 55.6 | 11 | 45.8 | 0.49 |
| Hypertension | 21 | 77.8 | 11 | 45.8 | 0.019 |
| Diabetes mellitus | 5 | 18.5 | 5 | 20.8 | 0.84 |
| Implantable cardioverter-defibrillator | 2 | 7.4 | 1 | 4.1 | 0.62 |
| VPC morphology | | | | | 0.67 |
| RBBB | 14 | 51.9 | 11 | 45.8 | |
| LBBB | 13 | 48.1 | 13 | 54.2 | |
| VPC axis | | | | | 0.23 |
| Inferior | 20 | 74.1 | 21 | 87.5 | |
| Superior | 7 | 25.9 | 3 | 12.5 | |
| VPC origin site | | | | | |
| LV/coronary cusps | 12 | 44.4 | 8 | 33.3 | 0.57 |
| RV/RVOT | 9 | 33.3 | 9 | 37.5 | 0.78 |
| Epicardial | 2 | 7.4 | 2 | 8.3 | 1.0 |
| Papillary muscle | 1 | 3.7 | 2 | 8.3 | **0.6** |
| Fascicular | 0 | 0 | 1 | 4.2 | 0.47 |
| Multiple VPCs | 3 | 11.1 | 2 | 8.3 | 1.0 |
| VPC QRS width (ms) | 159±8.7 | | 153±10.1 | | 0.66 |
| Medications | | | | | |
| Aspirin | 23 | 85.2 | 18 | 75 | 0.49 |
| Beta-blocker | 20 | 74.1 | 16 | 66.7 | 0.76 |
| ACEI/ARB | 21 | 77.8 | 15 | 62.5 | 0.36 |
| Statin | 14 | 51.9 | 15 | 62.5 | 0.57 |
| Class I/III antiarrhythmic | | 0 | | 0 | 1.0 |

*VPC: Ventricular premature complex; RBBB: Right bundle branch block; LBBB: Left bundle branch block; LV: Left ventricle; RV: Right ventricle; RVOT: Right ventricle outflow tract; ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker.

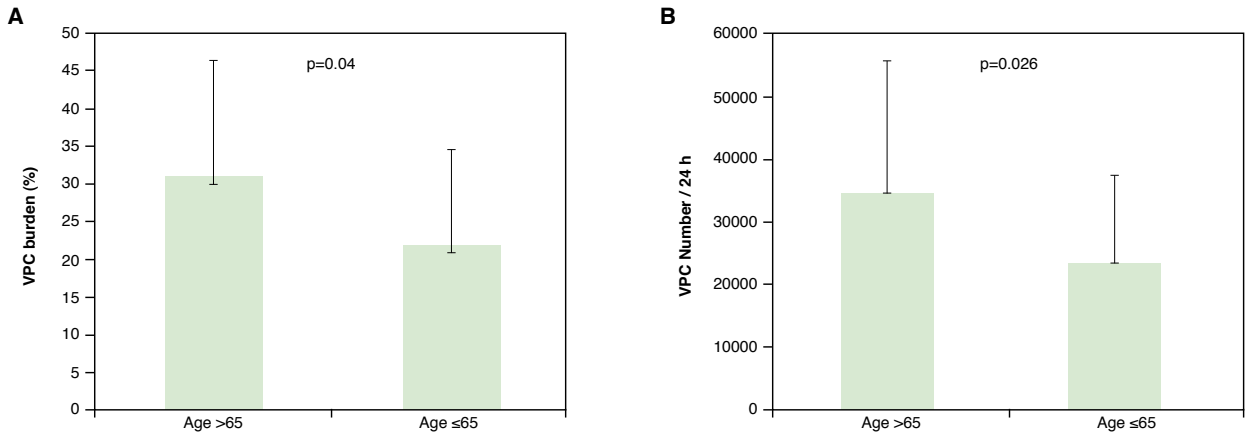


**Figure 1.** Comparison of pre-ablation VPC burden **(A)** and VPC number **(B)** on 24-hour Holter recording in older and younger patients.

ables prior to and after VPC ablation was performed using paired Student's t test. Differences were considered significant at a p value of <0.05.

## RESULTS

The study population reported here included 51 patients (49 male, mean age: 66±10 years). Twenty-seven (26 male, 73±6 years) patients were included in the older group (>65 years), whereas 24 patients (23 male, 57±7 years) were categorized into the younger group (≤65 years). Table 1 summarizes the baseline characteristics of the two groups. Patients aged >65 years had a higher prevalence of hypertension (p=0.019). There was no difference between the two groups based on prevalence of coronary artery disease, cardiomyopathy and diabetes (p=1.0, p=0.49 and p=0.84, respectively).

The VPC morphology, VPC axis, VPC origin site, and VPC QRS width were comparable between the two groups (Table 1). The pre-ablation 24-hour VPC burden (calculated by dividing the number of VPCs by the total number of beats) and VPC number were significantly higher in patients aged >65 years (p=0.04 and p=0.026, respectively; Fig. 1).

### Clinical outcomes and LV systolic function after VPC ablation

Twenty-two patients (81.5%) aged >65 years and 21 patients (87.5%) ≤65 years had successful ablation, defined as >80% reduction in VPC burden at follow-up. One patient in the younger group died during the follow-up. Pericardial tamponade requiring percutaneous drainage occurred in one patient (in the younger group).

The mean heart rate remained unchanged after the procedure in both groups (67.5±9.4 bpm *vs.* 70.1±9.3 bpm; p=0.62 for >65 years group and 69.2±9.9 bpm *vs.* 74.1±9.3 bpm; p=0.37 for ≤65 years group). Mean LVEF showed a significant increase in both groups (Fig. 2). The improvement in LVEF was accompanied by a significant decrease in both LVEDD and left atrial diameter (Table 2).

The mean VPC burden had decreased from 31±15.3% to 9.1±10.3% (p<0.001) in patients >65

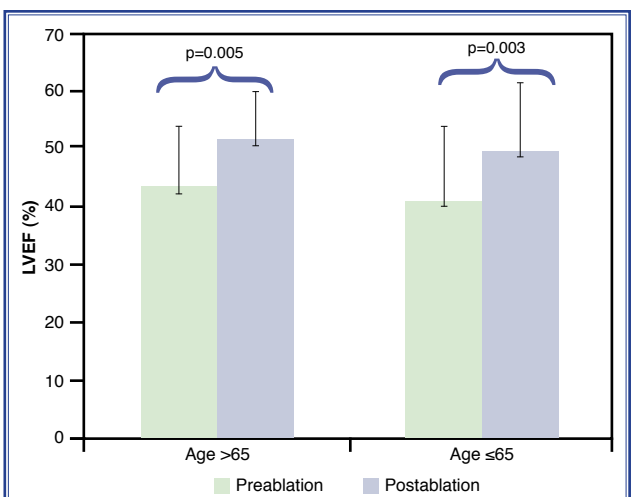


**Figure 2.** Left ventricular ejection fraction (LVEF) before and 6±3 months after catheter ablation of VPC in the two groups.

**Table 2. Comparison of echocardiographic variables before and after VPC ablation**

| | Age >65 (n=27) | | | Age ≤65 (n=24) | | |
|---|---|---|---|---|---|---|
| | Pre-ablation | Post-ablation | *p* | Pre-ablation | Post-ablation | *p* |
| LVEF (%) | 43.4±10.4 | 51.5±8.2 | **0.005** | 40.8±13.2 | 49.5±11.8 | **0.003** |
| LVESD (mm) | 44.4±9.3 | 40.9±8 | **0.07** | 42.8±9.1 | 39.5±7.1 | 0.18 |
| LVEDD (mm) | 59.7±6.1 | 56.2±5.6 | 0.032 | 58.8±6.9 | 55.6±6.1 | 0.047 |
| LA (mm) | 46.8±6.2 | 43.1±4.2 | **0.05** | 46.8±5.7 | 42.9±4.2 | **0.04** |

*LVEF: Left ventricle ejection fraction; LVESD: Left ventricular end-systolic diameter; LVEDD: Left ventricular end-diastolic diameter; LA: Left atrial diameter.

years and from 21.9±12.6 to 3.8±7.1 (p<0.001) in patients ≤65 years in 3-8 months after catheter ablation (Fig. 3).

Analysis of LAV demonstrated a significant decrease in LAVmax in patients aged >65 years (p=0.002) and ≤65 years (p=0.01) after VPC ablation

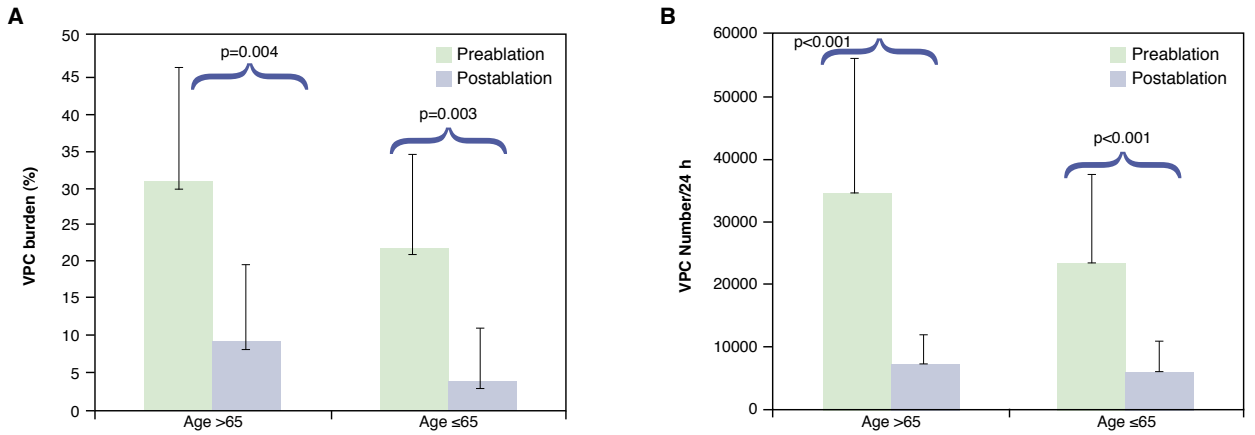


**Figure 3. (A, B)** Improvement in VPC burden and VPC number after catheter ablation.

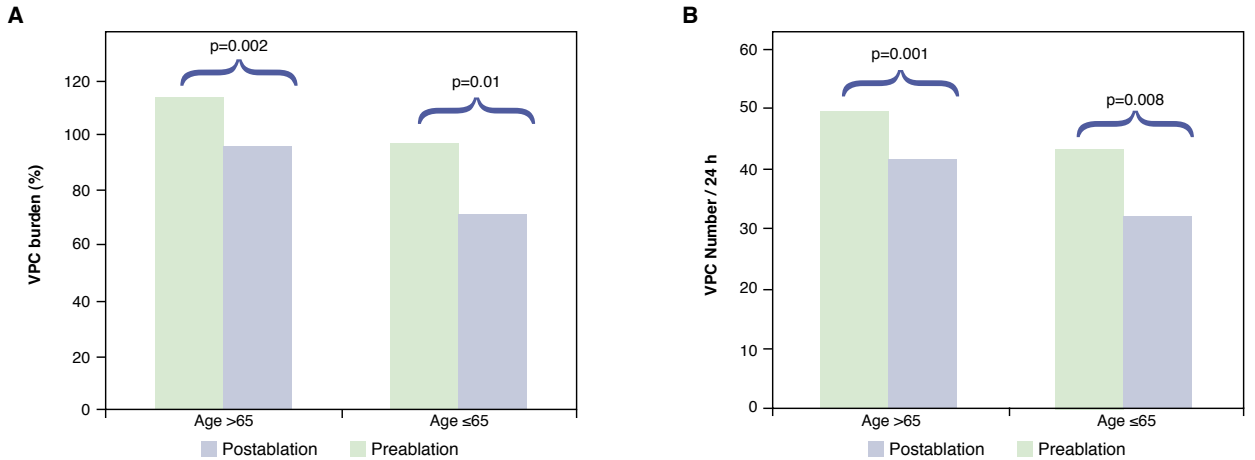


**Figure 4.** Changes in left atrial volumes **(A)** and left atrial volume index **(B)** in the study population 6±3 months after catheter ablation of VPCs.

(Fig. 4a). At baseline, mean LAVI was severely increased in the study population: 49.7±14.7 ml/m$^2$ for the older group and 43.3±12.6 ml/m$^2$ for the younger group (normal LAVmax/BSA: <29 ml/m$^2$). After the procedure, LAVI decreased significantly in both groups but remained abnormal (Fig. 4b).

## DISCUSSION

This study revealed that catheter-based treatment of VPCs reduces VPC burden, improves LV systolic function, and decreases LAV in patients both >65 years and ≤65 years of age, and there is no incremental risk associated with older patients.

The occurrence of VPCs is more common in patients with structural heart disease. Male gender, faster sinus rate, electrolyte abnormalities, hypertension, and increased age are other risk factors for frequent VPCs.[7,8] Simpson et al. reported that the prevalence of VPCs shows a 34% increase for each five-year increment in age.[7] The age-related increase in the prevalence of VPCs was demonstrated in both normal individuals and those with underlying heart disease.[8] In accordance with the above-mentioned studies, the frequency of VPCs in the present study was found to be higher in the older group than in patients ≤65 years. In addition, the ARIC study demonstrated that patients with hypertension have more frequent VPCs than the non-hypertensive group.[7] In our study, the prevalence of hypertension was significantly higher in patients >65 years than in the younger group, which might be another reason for the higher VPC burden in the older group. Although the exact mechanisms of frequent VPC in the presence of increased age and hypertension is still not clear, we speculate that since both conditions are related to an increased fibrotic process in the myocardial tissue, the conduction delay in fibrotic areas serves as a substrate for reentry and triggered activity that results in frequent VPCs. We also found that catheter ablation resulted in a significant decrease in VPC burden in both the older and younger patients. A study done by Baman et al.[9] reported that VPC burden was reduced by ≥80% by catheter ablation in 84% of the patients. The success rate in our study was 81.5% in patients aged >65 years and 87.5% in those ≤65 years, showing comparable results between the two groups.

Catheter-based ablation is an effective treatment for patients with frequent and symptomatic VPCs.[10] In addition to improving symptoms, reduction in VPC burden may play a role in improvement in LV systolic functions after the procedure. Catheter ablation of VPCs is thought to improve LVEF through several mechanisms, including better rate control, restoration of atrioventricular transport function and regaining of ventricular synchrony.[11] Previous studies report the average increase in LVEF following successful catheter ablation of VPCs as ranging between 4% and 24%.[11,12] Bogun et al.[11] evaluated the efficacy of VPC ablation in patients with VPC-induced cardiomyopathy and reported that LVEF increased from 34% to 59% within six months following ablation. A recent study by Lü et al.[12] on the efficacy of catheter ablation in 24 patients with asymptomatic frequent VPCs reported an average 11% increase in LVEF at the eighth-month follow-up after ablation. In the present study, catheter ablation of VPCs also resulted in a significant increase in LVEF, supporting these prior reports. However, to our best knowledge, this is the first study comparing the efficacy of VPC ablation in both the older and younger groups. Our results indicate that the ablation-based therapy in older patients is as effective as in younger patients, and the improvement in LVEF seems to be similar between the two groups.

Another finding in this study was that catheter ablation of VPCs resulted in a significant decrease in LAV. In accordance with our study, Kim et al. also reported improvement in LAVI approximately one year after VPC ablation. The main improvement in their study was observed among patients with VPC from right ventricle outflow track (RVOT)-origin, and LAVI nearly reached normal value after ablation.[13] However, mean LAV in our study was still higher at the follow-up. A possible explanation is that short-term echocardiographic assessment is inadequate for reversal of structural remodeling in the LA after ablation, and we speculate that the results after one year would show lower LAVs. Most of the studies evaluating reversal of atrial remodeling were done using drugs[14] or catheter-based treatment of atrial fibrillation[15] and had a follow-up of over one year. On the other hand, left atrial remodeling is reversible, particularly in the earlier stages of LA structural disturbances.[16] Since the baseline LAVI value in our study population was higher than that reported by Kim et al.'s group, it is reasonable to think that we had more patients with advanced stage of LA structural dysfunction who had ir-

reversible LA remodeling and thus demonstrated less improvement after ablation.

## Limitations

The primary limitation of our study was the relatively short follow-up period post-ablation to ascertain the long-term effects. Assessing ventricular function post-ablation after one year or more of follow-up may show more robust changes than seen in our study. Second, the assessment of pre-ablation and post-ablation VPC burden was done by a single 24-hour Holter monitor. Since frequency of VPCs is variable, a single monitor may not accurately represent the real burden of VPCs. However, several previous studies have used the same method to assess VPC burden.[3,6] Third, we used TTE for the assessment of LV function. Although invasive measurements are the gold standard for assessment of diastolic function, the accuracy of echocardiography has been validated in several prior studies.[17,18] Fourth, patients in this study were predominantly males of relatively older ages. The outcomes in females and young subjects may vary. Finally, the sample size in the present study was relatively small. Larger studies are needed in order to make more substantive conclusions regarding the improvement in diastolic parameters following VPC ablation.

In conclusion, catheter ablation reduced VPC burden, improved LV systolic function and decreased LAV in patients with frequent VPCs. The improvement occurs in both younger and older patients, and there is no incremental risk associated with older patients.

***Conflict-of-interest issues regarding the authorship or article: None declared***

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*Key words:* Catheter ablation; electrocardiography; ventricular dysfunction, left; ventricular premature complexes / physiopathology.

*Anahtar sözcükler:* Kateter ablasyonu; elektrokardiyografi; ventrikül disfonksiyonu, sol; ventrikül erken kompleksleri / fizyopatoloji.