Premature Ventricular Contractions-Induced Cardiomyopathy
A Treatable Condition

Yong-Mei Cha, MD; Glenn K. Lee, MBBS; Kyle W. Klarich, MD; Martha Grogan, MD

Premature ventricular contractions (PVCs) are early depolarizations of the myocardium originating in the ventricle. They are often seen in association with structural heart disease and represent increased risk of sudden death, yet they are ubiquitous, even in the absence of identifiable heart disease. They may cause troubling and sometimes incapacitating symptoms such as palpitations, chest pain, presyncope, syncope, and heart failure. Traditionally, they have been thought to be relatively benign in the absence of structural heart disease. Over the last decade, however, PVC-induced cardiomyopathy has been a subject of great interest and the evidence for this entity is rapidly emerging.

Epidemiology
PVCs are common with an estimated prevalence of 1% to 4% in the general population. In a normal healthy population, PVCs have been detected in 1% of subjects on standard 12-lead electrocardiography and between 40% and 75% of subjects on 24- to 48-hour Holter monitoring. Their prevalence is generally age-dependent, ranging from <1% in children to 69% in subjects >75 years. Commonly thought to be a benign entity, the concept of PVC-induced cardiomyopathy was proposed by Duffee et al in 1998 when pharmacological suppression of PVCs in patients with presumed idiopathic dilated cardiomyopathy subsequently improved left ventricular (LV) systolic dysfunction. Many of these patients often have no underlying structural heart disease and subsequently develop LV dysfunction and dilated cardiomyopathy; in cases of those with an already impaired LV function from underlying structural heart disease, worsening of LV function may occur. The exact prevalence of PVC-induced cardiomyopathy is not known; it is an underappreciated cause of LV dysfunction, and it is primarily observed in older patients. This observation could be due to the fact that the prevalence of PVCs increases with age or the possibility that PVC-induced cardiomyopathy develops in a time-dependent fashion.

In fact, Niwano et al demonstrated progressive worsening of LV function in patients with frequent PVCs (>1000 beats/day) as measured by the LV ejection fraction (LVEF) and LV end-diastolic dimension over a follow-up period of 4 to 8 years (Figure 1).

Electrophysiological Characteristics
PVC Burden
Several studies have shown that the frequency of PVCs correlates at least modestly with the extent of LV dysfunction and ventricular dilation at the time of initial clinical presentation. Patients with decreased LVEF had a higher mean PVC burden than their counterparts with normal LV function (29%–37% versus 8%–13%). However, there are no clear-cut points that mark the frequency at which cardiomyopathy is unavoidable. Niwano et al used a cut point of 20,000 PVCs over 24 hours to define the high-frequency group, whereas Kanei et al used a figure of 10,000 PVCs per day. Other studies defined “frequent” PVCs as >10% of total beats rather than the absolute number of PVCs. yet in some cases, a high PVC burden may not impair LV function, whereas PVC-induced cardiomyopathy can be observed in patients with lower PVC frequencies, albeit at lower incidences. It is not known why the majority of patients with frequent PVCs have a benign course, whereas up to one third of them develop cardiomyopathy. One possible explanation is that the evaluation of PVC burden using 24-hour Holter monitoring may be inadequate and may misrepresent the patient’s true PVC burden.

Baman et al suggested that a PVC burden of >24% had a sensitivity and specificity of 79% and 78%, respectively, in separating the patient populations with impaired versus preserved LV function. Nevertheless, the majority of patients presenting with frequent PVCs had preserved LVEF. Therefore, although significant, the PVC burden is not the only factor contributing to impairment of LV systolic function.

PVC Origin
Approximately two thirds of idiopathic PVCs originate from the ventricular outflow tracts, primarily the right ventricular outflow tract. The ventricular outflow tract musculature,
and hence the anatomic source of the PVCs, may extend above the pulmonary or aortic valves. Gami et al.23 examined 603 autopsy hearts and found that 57% of these hearts had myocardial extensions above the aortic valve, and 74% had extensions above the pulmonary valve. Extensions were noted more often in the right coronary cusp (55%) than in the left coronary cusp (24%) and noncoronary/posterior cusp (<1%). In contrast, myocardial extensions above the 3 pulmonary cusps are distributed more evenly (45%–60%). These myocardial extensions constitute the electric substrates for ectopies and tachycardia. The remaining third of PVCs may have various ventricular origins, including the ventricular free walls, LV fascicles, septum, and papillary muscles. These ectopies may originate from single or multiple foci, presenting as monomorphic or polymorphic PVCs on electrocardiography. It should be noted that PVCs originating from the outflow tracts are often monomorphic.

Munoz et al.22 retrospectively studied 70 subjects who underwent PVC ablation. Of these, 17 (24%) had a reduced LVEF < 50%. When compared with those with PVC duration ≥ 50%, there was no significant difference in baseline patient characteristics except that patients with reduced LVEF were less likely to have reported symptoms. Interestingly, in addition to a higher PVC burden being observed in patients with reduced LVEF, PVCs originating from the right ventricle were associated with significant reduction of LVEF at a PVC burden ≥ 10%, whereas PVCs originating from the LV were associated with significant reduction of LVEF only at a higher PVC burden of ≥ 20%. This finding, however, has to be interpreted within the constraints of a retrospective study design with a small sample size and a highly selected population. Whether delayed LV excitation and contraction from right ventricular PVCs pose greater hemodynamic threat to the myocardial function is a question that has not been studied, but it is plausible given the observation of right ventricular pacing-induced LV dysfunction.24–29

PVC QRS Morphology, Duration, Coupling Interval, and Interpolation
Although the threshold burden of PVCs associated with reduced LVEF was lower for right as compared with LV PVCs, data from 1 study suggest that the PVC morphology such as left bundle branch block or right bundle branch block pattern does not appear to affect the LVEF.22 However, the morphology can, to some extent, determine the site and etiology of the PVCs. PVCs with smooth and uninterrupted contours as well as a sharp QRS deflection typically represent an isolated ectopic focus and a structurally normal heart, whereas PVCs with broad notching and a slurred QRS deflection may represent a diseased myocardial substrate.30 PVCs originating from the fascicles or ventricular septum typically have a narrower QRS wave as opposed to PVCs originating from the free walls and outflow tracts. One study noted that PVC duration ≥ 140 ms was an independent predictor of impaired LVEF.22 There is inconsistent evidence as to whether the coupling interval is associated with LV dysfunction. A study by Sun et al.31 reported that PVC coupling intervals ≤ 600 ms had a lower mean LVEF. A recent study by Olgun et al.32 showed that PVC interpolation is an independent predictor of PVC-induced cardiomyopathy, although other studies have not corroborated this observation.22 These observations may not apply to all patients because these are single studies with small numbers of patients. Decreased PVC coupling interval may affect the LV filling profile, stroke volume, and pulse pressure.33 The association between coupling interval and hemodynamic/structural consequence remains to be examined.

Mechanisms and Pathophysiology
PVC-induced cardiomyopathy was originally thought to be a type of tachycardia-induced cardiomyopathy,12,14,15,21 a phenomenon that has been well described in the context of atrial fibrillation, supraventricular tachycardia, and ventricular tachycardia.12 However, this concept has been called into question because patients with frequent PVCs have overall heart rates similar to those of their normal counterparts on Holter monitoring.13,14 The cellular mechanisms of PVC-induced cardiomyopathy have as yet not been elucidated and are only speculative at present given the dearth of animal models and prospective clinical studies in this area. However, on the basis of the observation that some patients with frequent PVCs had isoproterenol-facilitated induction of sustained monomorphic ventricular tachycardia of similar morphologies to the PVCs,34 Yarlagadda et al.12 postulated that cAMP-mediated triggered activity may be an operative mechanism in some patients with PVCs. Frequent and persistent exposure to PVCs is associated with complex transient alterations in intracellular calcium and membrane ionic currents, heart rate dynamics, hemodynamic parameters, and both myocardial and peripheral vascular autonomic stimulation and inhibition.35–38 Morphological and functional abnormalities of the ventricular myocardium and outflow tracts may be found on MRI in patients with apparently idiopathic PVCs, albeit this does not necessarily have a clinical implication. Bogun et al.35 postulated that ventricular dysynchrony and increased oxygen consumption may be possible patho-
genic mechanisms. Ventricular dyssynchrony results in compromised global cardiac mechanical efficiency, asymmetrically increased wall thickness in the late-activated regions, altered myocardial blood flow, and local changes in myocardial protein expression.28 The ventricular dyssynchrony associated with PVCs, particularly when the PVC burden is high, may contribute to LV dilation and impaired function in a fashion previously described for patients with left bundle branch block or chronic right ventricular pacing.25–29 Right ventricular pacing is notably associated with dyssynchronous ventricular contraction,39 changes in cardiac sympathetic activity, histopathology as well as ion channel expression and function40; animal models have shown that right ventricular pacing induces asymmetrical myocardial hypertrophy, myofibrillar disarray, and increased catecholamine concentrations in the myocardium.41–43 Although clinical studies describe LV dysfunction developing over reasonably long periods of time (>4 years in the series of Niwano et al13), in canine models, LV dysfunction occurred within 4 to 12 weeks of induced ventricular ectopy.44,45 However, this PVC-induced cardiomyopathy animal model in a 3-month duration demonstrated the lack of myocardial fibrosis and absence of changes in apoptosis and mitochondrial function, which support a functional rather than structural mechanism. Smith et al46 observed sympathoexcitation evoked by acutely high rates of ventricular ectopy using programmed ventricular extrastimuli after every 4, 2, and 1 spontaneous sinus beats in patients with a history of supraventricular tachycardia. This inappropriate sympathetic activation could potentially impair LV function in the long-term, yet although animal models are crucial in helping us understand a variety of pathophysiological aspects that are difficult or unable to explore in human subjects, and give us an impression of the relationship between the onset and burden of PVCs to the development of systolic dysfunction, the limited comparability between animal and human studies has to be taken into account. Patient-specific factors such as genetic predispositions determining myocardial protein expression in response to PVC-induced stress may interact with PVC frequency to influence the risk of cardiomyopathy.47 The putative mechanisms of PVC-induced cardiomyopathy are summarized in Figure 2.

Clinical Evaluation
In many patients, the onset of frequent PVCs relative to the onset of LV dysfunction is unknown.47 It is particularly important to identify the primary disorder because of the potential reversibility of PVC-induced cardiomyopathy.48,49 Patients can present with debilitating symptoms, particularly palpitations, or other symptoms such as chest pain, presyncope, syncope, or heart failure manifested by decreased effort tolerance, possibly as a result of decreased effective cardiac output.3 The vast majority of these patients are healthy with no known structural heart disease,4 but a detailed family history is required to help exclude familial dilated cardiomyopathy. The physical examination findings are often normal except irregular heart rhythm when PVCs are frequent. The electrocardiography may indicate the presence and morphology of PVCs, but PVC burden should be assessed by continuous Holter monitoring for at least 24 hours. Because a single 24-hour recording may not reflect the true PVC load due to day-to-day variability, a strong suspicion that frequent PVCs may be the cause of LV dysfunction may warrant extended Holter recordings of 48 to 72 hours or several 24-hour Holter recordings.20 Echocardiography is useful to exclude valvular pathology, regional wall motion abnormalities, cardiomyopathies, or myocardial abnormalities such as noncompaction, all of which could be a cause for frequent PVCs. Echocardiographic features in PVC-induced cardiomyopathy include decreased LVEF, increased LV systolic and diastolic dimensions, wall motion abnormalities, which are often global as opposed to regional,20 as well as mitral regurgitation (typically due to mitral annular dilatation).14 Two-dimensional speckle tracking strain imaging has shown altered LV contractility, whereas the LVEF remains preserved.50–54 Whether the abnormal findings from speckle tracking imaging predict the reduction of LVEF in patients
with PVCs remains to be studied. Cardiac MRI may be warranted in detecting arrhythmogenic right ventricular cardiomyopathy with LV involvement and infiltrative disease when clinically suspected. Coronary angiography should be performed in every patient with reduced LV systolic function to exclude significant coronary artery disease except for those with a low cardiovascular risk. Appropriate workup should also be performed to exclude other causes of cardiomyopathy, including those related to drugs/toxins, infectious diseases, and endocrinopathies.

It should be noted that PVC-induced cardiomyopathy remains a diagnosis of exclusion; underlying structural disease causing frequent PVCs must be ruled out. Given the reciprocal causal association between PVCs and cardiomyopathy where either 1 can lead to the other, it may be difficult to isolate the primary disorder.49,55 In many patients, the onset of frequent PVCs relative to the onset of LV dysfunction is unknown.47 It is particularly important to identify the primary disorder because of the potential reversibility of PVC-induced cardiomyopathy.48,49

**Therapeutic Modalities**

A therapeutic medical trial or catheter ablation may be considered in patients with LV dysfunction and frequent PVCs (a generally accepted range of >10,000–20,00013,17 or >10% of total heart beats18,19,22 over 24 hours) if the clinical suspicion for PVC-induced cardiomyopathy is high. The patient should be followed up over 3 to 12 months to assess posttreatment PVC frequency as well as LV size and function. The workup, treatment, and follow-up of patients with frequent PVCs are summarized in Figure 3.

**Pharmacological Therapy**

Because the long-term prognosis of frequent PVCs is generally considered to be benign and the majority of patients presenting with frequent PVCs have a preserved LVEF,4,9,12,13,15 the preferred treatment is usually reassurance and counseling of the patient with close follow-up if the patient is asymptomatic. In the presence of symptoms, pharmacotherapy such as a β-blocker or a nondihydropyridine calcium channel blocker may be the first-line therapy.56 Krittayaphong et al57 showed, in a randomized placebo-controlled study, that atenolol significantly decreased symptom frequency and PVC count. Antiarrhythmics such as flecainide or sotalol may be considered when β-blockers or calcium channel blockers are ineffective.58 Capucci et al59 demonstrated that 91% of patients taking flecainide and 55% of patients on mexiletine had a PVC reduction of ≥70%. In the presence of overt LV dysfunction, Class IA or IC drugs such as flecainide or propafenone are not recommended given their side effect

**Figure 3.** Flow chart for the diagnosis, treatment and follow-up of patients presenting frequent premature ventricular contractions (PVCs). Electrocardiography indicates electrocardiography. LV indicates left ventricular; LVEF, left ventricular ejection fraction; PVC, premature ventricular contraction.
profiles, including the propensity for proarrhythmic effects and adverse impact on survival. Careful consideration in regard to the baseline renal and hepatic function is also necessary when initiating an antiarrhythmic agent to avoid inadvertent drug toxicity. Amiodarone and dofetilide are preferred in view of their favorable cardiac safety profile.

A randomized, double-blind, placebo-controlled trial conducted by Singh et al showed that amiodarone was significantly more effective in suppressing PVCs with concomitant improvement in LVEF in patients with congestive heart failure and asymptomatic ventricular arrhythmia.

**Catheter Ablation**

Catheter-based ablative approaches represent an emerging effective and alternative therapy to pharmacological treatment and have been increasingly reported over the past decade. Many studies have documented the improvement in LV function after catheter ablation of frequent PVCs (Table). Yarlagadda et al noted a significant improvement in LVEF to near 60% in all patients with LV dysfunction who underwent successful catheter ablation of their frequent PVCs. Bogun et al documented the normalization of LVEF in 82% of patients who underwent catheter ablation for frequent PVCs and LV systolic dysfunction. Improvement of LVEF was also documented by Sarrazin et al after catheter ablation of frequent PVCs in the setting of prior myocardial infarction. Other studies also found significant reduction in LV end-diastolic dimensions of between 2 and 8 mm, mitral regurgitation by 75%, and New York Heart Association functional class by nearly 1 class. Wijmaalen et al described a significant improvement in radial, circumferential, and longitudinal strain after catheter ablation in patients with frequent PVCs and preserved LVEF. However, like with any invasive intervention, the risks of catheter ablation (whether endocardial or epicardial) must be balanced against any potential benefits. Major complications occur in approximately 3% of cases, including death, stroke, myocardial infarction, atrioventricular block necessitating permanent pacemaker placement, cardiac perforation with or without pericardial tamponade, pericardial effusion, and blood vessel dissection or stenosis. Nevertheless, catheter ablation for PVCs is associated with a high success rate and few complications.

Advancements and innovations in catheter ablation technology have favorably altered the efficacy–safety profile of ablation therapy. Our ability to predict the site of origin of the PVCs on surface electrocardiography and to safely access the right ventricular outflow tract and left ventricle (including the pericardium and the aortic cusp region) and the use of advanced 3-dimensional mapping tools allows us to precisely localize the PVC foci. Short-term ablation success rates of between 70% and 90% have been reported; however, the long-term ablative outcomes remain to be studied. Alternative energy sources such as cryoablation may help improve the efficacy–safety profile as compared with traditional radiofrequency-based techniques. Given the favorable efficacy–safety profile of current catheter ablation techniques, we believe that catheter ablation is an increasingly appealing therapy for management of patients with suspected PVC-induced cardiomyopathy. However, randomized clinical trials are needed to determine a comparative efficacy and outcome of modern medical against ablative approaches for those with frequent PVCs that are likely to be the cause of cardiomyopathy.

**Future Perspectives**

Knowledge of the molecular, cellular, and hemodynamic mechanisms of LV dysfunction in PVC-induced cardiomyopathy is limited. Despite the limited comparability between animal models and human subjects, animal models enable us to study the effects of PVCs and to understand the pathogenesis of PVC-induced cardiomyopathy. Development of better imaging techniques such as 2-dimensional speckle tracking strain imaging and cardiac MRI aimed at assessing early LV dysfunction and excluding occult structural heart disease allows the possibility of earlier detection of the disease. There is at present little evidence to recommend the routine use of such imaging techniques in the workup of PVC-induced cardiomyopathy. Although the suppression of PVCs is indicated for symptomatic patients with frequent PVCs and those with overt LV dysfunction, there is at present no evidence for the treatment of asymptomatic patients with normal LVEF to prevent PVC-induced cardiomyopathy. Further research is needed to identify the risk predictors for developing PVC-induced cardiomyopathy and to make a recommendation on the need and frequency of echocardiographic follow-up in this group of patients. In patients with a diagnosis of nonischemic cardiomyopathy and frequent PVCs with uncertainty as to the former being the cause or consequence of the latter, the plausibility of suppressing PVCs with medical or interventional therapy before placement of an implantable cardioverter–defibrillator (if current guidelines for its insertion are met) requires further study, bearing in mind that the cardiomyopathy is potentially reversible. In patients with decreased LVEF, a follow-up period of 3 to 12 months after initiation of antiarrhythmic therapy or catheter ablation is suggested to allow for recovery of LV function and to avoid unnecessary implantable cardioverter–defibrillator insertions in a potentially reversible condition. Where reduced LVEF persists despite diminished PVC burden, the decision as to the need for implantable cardioverter–defibrillator insertion should be governed by current guidelines. Frequent PVCs might be a factor responsible for the lack of response to medical therapy or cardiac resynchronization therapy. Suppression or elimination of PVCs may improve response to cardiac resynchronization therapy.

**Conclusions**

PVCs are a common occurrence within the general population. In the absence of structural heart disease, they typically carry a good prognosis. However, they may present with debilitating symptoms. PVCs have been implicated in the development of LV dysfunction and cardiomyopathy, but the risk factors and pathogenic mechanisms are incompletely understood. The suppression of PVCs using either antiarrhythmic pharmacological agents or emerging catheter ablation techniques appears to reverse the LV dysfunction.
### Table. Effects of Catheter Ablation of PVCs on Cardiac Function

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Sample Size</th>
<th>Inclusion Criteria</th>
<th>Study Targets</th>
<th>Effect of Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Takemoto et al.</td>
<td>2009</td>
<td>27 (8 with depressed LVEF ≤ 45%)</td>
<td>Frequent PVCs of LBBB and inferior axis morphology (mean of 17 624 PVCs over 24 h on Holter monitoring)</td>
<td>Successful ablation (PVCs abolished during ablation and remained absent for ≥ 30 min in baseline state and during infusion of isoproterenol)</td>
<td>Successful ablation in 23 patients, including 7 of 8 patients with low LVEF, reduction in PVCs from 17 541 ± 11 479 to 507 ± 722 ($P &lt; 0.028$)</td>
<td>Significant improvement after ablation in LVEF in the 7 patients with depressed LVEF; mean LVEF increased from 39% ± 6% at baseline to 62% ± 6% ($P &lt; 0.017$)</td>
</tr>
<tr>
<td>Sekiguchi et al.</td>
<td>2005</td>
<td>47</td>
<td>&gt;1 000 PVCs per d</td>
<td>Reduction in PVCs to &lt; 1000 per d</td>
<td>Successful ablation in 38 patients, reduction in PVCs from 23 989 ± 13 366 to 137 ± 249 ($P &lt; 0.0001$)</td>
<td>After ablation, mean LVEDD decreased from 50.5 ± 4.8 mm to 45.3 ± 6.4 mm ($P &lt; 0.01$); mean LVESD decreased from 33.7 ± 4.0 mm to 30.3 ± 5.5 mm ($P &lt; 0.01$)</td>
</tr>
<tr>
<td>Takemoto et al.</td>
<td>2005</td>
<td>40 (14 with PVC burden &lt; 10%; 12, 10–20%; 14 &gt; 20%)</td>
<td>PVCs of LBBB and inferior axis morphology</td>
<td>Successful ablation (noninducibility of PVCs with or without isoproterenol and/or programmed electrical stimulation for ≥ 30 min, nonrecurrence of PVCs for 72 h)</td>
<td>Successful ablation in 37 patients, reduction in PVC burden in the &gt; 20% group from 34% ± 3% to 1.3% ± 0.9% ($P &lt; 0.01$)</td>
<td>Significant improvement for patients with PVC burden &gt; 20%; LVEDD decreased from 54 ± 1 mm to 47 ± 1 mm; PVC burden increased from 66% ± 2% to 72% ± 2%; MR decreased from 1.2 ± 0.2 to 0.3 ± 0.1° and NYHA functional class decreased from 1.8 ± 0.2 to 1.0 ± 0.0 ($P &lt; 0.05$ for all)</td>
</tr>
<tr>
<td>Bogun et al.</td>
<td>2007</td>
<td>60 (22 with depressed LVEF ≤ 50%)</td>
<td>&gt;10 PVCs of various morphologies per hour over 24 h on Holter monitoring</td>
<td>Successful ablation (PVCs abolished during ablation and uninducible by isoproterenol)</td>
<td>Successful ablation in 48 patients, including 18 of 22 patients with depressed LVEF</td>
<td>Significant improvement after ablation in the 18 patients with depressed PVC burden; LVEDD increased from 34% ± 13% to 59% ± 7% ($P &lt; 0.001$), LVEDD decreased from 59 ± 6 mm to 51 ± 8 mm ($P = 0.001$), LVESD decreased from 45 ± 7 mm to 34 ± 7 mm ($P = 0.0002$)</td>
</tr>
<tr>
<td>Taleb et al.</td>
<td>2007</td>
<td>6</td>
<td>Frequent PVCs of various morphologies and LV dysfunction (mean of 17 717 PVCs over 24 h on Holter monitoring)</td>
<td>Successful ablation</td>
<td>Successful ablation in all patients, reduction in PVCs from 17 717 ± 7100 to 268 ± 366 ($P = 0.006$)</td>
<td>LVEDD increased from 42% ± 2.5% at baseline to 57% ± 3% ($P = 0.0001$), mean LVESD decreased from 60.0 ± 3.5 mm to 54.0 ± 3.7 mm ($P = 0.0009$)</td>
</tr>
<tr>
<td>Sarrazin et al.</td>
<td>2009</td>
<td>30 (15 referred for ablation, 15 served as control)</td>
<td>PVC burden of &gt; 5% in patients with prior myocardial infarction</td>
<td>Successful ablation</td>
<td>Successful ablation in all 15 patients, reduction in PVC burden from 22% ± 12% to 2.6% ± 5.0%</td>
<td>Significant improvement after ablation in LVEF; mean LVEF increased from 38% ± 11% to 51% ± 9% ($P &lt; 0.0001$); no improvement in LVEF was noted in the control group</td>
</tr>
<tr>
<td>Baman et al.</td>
<td>2010</td>
<td>174 (57 with depressed LVEF 35% ± 9%)</td>
<td>Frequent PVCs of various morphologies (mean burden of 20% ± 16% on Holter monitoring)</td>
<td>Successful ablation (80% reduction in PVC burden)</td>
<td>Successful ablation in 146 patients, including 46 of 57 patients with depressed PVC burden, reduction in PVC burden from 33% ± 14% to 1.9% ± 4.4% ($P &lt; 0.01$)</td>
<td>Significant improvement in the 57 patients with depressed PVC; PVC burden increased from 35% ± 9% at baseline to 54% ± 10% ($P &lt; 0.01$), LVEDD decreased from 59 ± 7 mm to 54 ± 7 mm ($P &lt; 0.01$), LVESD decreased from 44 ± 7 mm to 39 ± 8 mm ($P &lt; 0.01$)</td>
</tr>
<tr>
<td>Wijnmaalen et al.</td>
<td>2010</td>
<td>49</td>
<td>PVC burden of &gt; 5% of various morphologies with normal LVEF, LV volumes, and RV dimensions</td>
<td>Successful ablation (PVCs abolished during ablation and uninducible by isoproterenol)</td>
<td>Successful ablation in 34 patients, reduction in PVC burden from 26% ± 13% to 0.2% ± 0.8%</td>
<td>LV radial strain increased from 31.1% ± 14.2% to 45.5% ± 16.3% ($P &lt; 0.0001$), LV circumferential strain increased from − 16.2% ± 3.9% to − 18.9% ± 4.2% ($P &lt; 0.004$), LV longitudinal strain increased from − 17.8% ± 2.9% to − 19.6% ± 2.0% ($P &lt; 0.007$); RV longitudinal strain increased from − 24.2% ± 7.4% to − 28.4% ± 6.0% ($P = 0.009$)</td>
</tr>
</tbody>
</table>

PVCs indicates premature ventricular contractions; LVEF, left ventricular ejection fraction; LBBB, left bundle branch block; LV, left ventricular; RV, right ventricular; LVEDD, left ventricular end-diastolic dimension; MR, mitral regurgitation; NYHA, New York Heart Association; LVESD, left ventricular end-systolic dimension.
Disclosures
None.

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10. Sarrazin JF, Labounty T, Kuhne M, Crawford T, Armstrong WF, Des- 


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In the article, “Premature Ventricular Contraction-Induced Cardiomyopathy: A Treatable Condition,” by Cha et al, which appeared in the February 2012 issue of the journal (*Circ Arrhythm Electrophysiol*, 2012;5:229–236), the order of authors is incorrect. The correct order is as follows:

Glenn K. Lee, MBBS; Kyle W. Klarich, MD; Martha Grogan, MD; Yong-Mei Cha, MD

The online version of the article has been corrected.

The authors regret the error.

**Reference**