Reversal of Cardiomyopathy in Patients With Repetitive Monomorphic Ventricular Ectopy Originating From the Right Ventricular Outflow Tract

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**Background**—Tachycardia-induced cardiomyopathy caused by ventricular tachycardia is a well-defined clinical entity. Less well appreciated is whether simple ventricular ectopy can result in cardiomyopathy. We sought to examine a potential causal relationship between repetitive monomorphic ventricular ectopy originating from the right ventricular outflow tract and cardiomyopathy and the role of ablation in reversing this process.

**Methods and Results**—The study consisted of 27 patients (11 men; age, 47 ± 15 years) with repetitive monomorphic ventricular ectopy, including 8 patients (30%) with depressed ventricular function (ejection fraction ≤45%). All patients underwent assessment of cardiac structure and function. The burden of ectopy was quantified through 24-hour Holter monitoring. Patients then underwent ablation guided by 3D mapping. After ablation, patients underwent repeated Holter monitoring and reassessment of cardiac function. Patients with depressed ventricular function were more likely to be older than patients with normal function (58 ± 14 versus 42 ± 18 years; \(P=0.013\)). However, the burden of ventricular ectopy was similar in patients with (17 859 ± 13 488 ectopic beats per 24 hours) and without (17 541 ± 11 479 ectopic beats per 24 hours; \(P=0.800\)) preserved ventricular function. Successful ablation was performed in 23 patients (85%), including 7 of 8 patients with depressed ventricular function. In this latter group, ventricular function improved in all patients (from 39 ± 6% to 62 ± 6%; \(P=0.017\)).

**Conclusions**—Repetitive monomorphic ventricular ectopy (in the absence of sustained ventricular tachycardia) originating from the right ventricular outflow tract is an underappreciated cause of unexplained cardiomyopathy. Successful ablation of the focal source of ventricular ectopy results in normalization of left ventricular function. Patients with ectopy-induced cardiomyopathy are significantly older than patients with preserved ventricular function, which suggests either that older patients are more susceptible to the development of a cardiomyopathy or that the cardiomyopathy has had a longer period of time in which to evolve. *(Circulation. 2005;112:1092-1097.)*

**Key Words:** ablation ■ cardiomyopathy ■ ventricular premature complexes

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**T**achycardia-induced cardiomyopathy has been described in association with a variety of arrhythmias, including atrial fibrillation,1 atrial flutter,2 supraventricular tachycardia,3,4 and ventricular tachycardia.5,6 In each instance, cardiomyopathy has been attributed to poor ventricular rate control. Furthermore, resolution of cardiomyopathy usually follows ventricular rate control by either medication or catheter ablation.

Tachycardia-induced cardiomyopathy has also been described in patients with idiopathic ventricular tachycardia originating from the right ventricular outflow tract (RVOT).7-9 The two predominant phenotypes of this tachycardia are nonsustained, repetitive, monomorphic ventricular tachycardia and paroxysmal, exercise-induced, sustained ventricular tachycardia; both are characterized by a left bundle branch, inferior-axis morphology and sensitivity to adenosine.10,11 The cellular mechanism of these tachycardias is thought to be cAMP-mediated triggered activity that is dependent on delayed afterdepolarizations.12

A subtype of repetitive, monomorphic ventricular tachycardia is characterized by isolated repetitive monomorphic ventricular ectopy, which occurs in the absence of ventricular tachycardia. A few isolated case reports have described the occurrence of cardiomyopathy in these patients.13-16 However, the characteristics and optimal management of patients with ectopy-induced cardiomyopathy remain undefined. The aim of this study was to examine a potential causal relationship between repetitive monomorphic ventricular ectopy and cardiomyopathy and the role of radiofrequency catheter ablation in reversing this process.

**Methods**

**Study Population**

We evaluated consecutive patients with repetitive monomorphic ventricular ectopy who were referred for radiofrequency catheter ablation between April 2000 and December 2004. All patients had ventricular ectopy with a left bundle branch, inferior-axis morphology. We specifically excluded patients who had predominantly...
nonsustained runs of ventricular tachycardia and patients with a known history of sustained ventricular tachycardia.

**Baseline Noninvasive Evaluation**
At baseline, a 24-hour Holter recording was obtained to quantify the burden of ventricular ectopy. In some patients, a diagnosis of repetitive monomorphic ventricular ectopy was made while they were being monitored on inpatient telemetry. In these patients, the 24-hour burden of ectopy could not be quantified.

All patients underwent evaluation of cardiac structure and function. When possible, this evaluation included cardiac MRI. Coronary artery disease was assessed in all patients with stress testing and/or cardiac catheterization. Left ventricular systolic function was quantified by echocardiography, radionuclide ventriculography, or left ventricular cineangiography. Patients with repetitive monomorphic ventricular ectopy who had coexisting ischemic cardiomyopathy (n=2) were excluded.

**Electrophysiological Testing**
After written informed consent was obtained, electrophysiological testing was performed. Patients were locally anesthetized with 0.25% bupivacaine and lightly sedated with midazolam and morphine or fentanyl. Diagnostic electrophysiological catheters included 6F catheters positioned in the high right atrium, His-bundle region, and right ventricle.

Ventricular stimulation was performed with up to triple ventricular extrastimuli at up to 2 paced cycle lengths from the right ventricular apex and RVOT at baseline and during infusion of isoproterenol. If sustained monomorphic ventricular tachycardia was inducible, adenosine 150 μg/kg was administered as an intravenous bolus, and the effect on ventricular tachycardia was noted.

**3D Mapping**
In all patients, 3D mapping of the right ventricle was performed during periods of isolated ventricular ectopy or induced ventricular tachycardia with either a contact (CARTO XP, Biosense Webster) or noncontact (Endocardial Solutions, St. Paul, Minnesota) electroanatomic mapping system. The electroanatomic mapping system consists of a quadrupolar catheter with a 4-mm distal electrode (Navistar, Biosense Webster) and a spatial reference patch on the patient’s back. The peak of the QRS complex was used as reference for local activation. Activation times were assigned on the basis of the onset of bipolar electrograms, which were obtained with filter settings of 30 and 400 Hz, and displayed as color gradients on a 3D activation map. In this manner, the site of earliest ventricular activation was identified.

The Ensite 3000 noncontact mapping system consists of a 9F noncontact catheter with a multielectrode array that surrounds a 7.5-mL balloon mounted at the distal end. The multielectrode array catheter was deployed over a 0.035-in guidewire that had been advanced into a branch of the pulmonary artery and positioned in the RVOT. Care was taken to avoid the presence of balloon-induced ventricular ectopy. The system creates a 3D virtual model of the endocardium on which 3000 unipolar electrograms can be superimposed. The resulting isopotential map was used to identify the site of endocardial breakthrough of the ventricular ectopy.

**Radiofrequency Ablation**
Ablation was performed with a 4-mm-tip ablation catheter using a maximal power of 50 W and a maximal temperature of 60°C delivered for up to 60 seconds. Ablation sites were confirmed by pace mapping. Ablation was performed only when a match was demonstrated in ≥11 of 12 leads. Ablation was considered acutely successful if repetitive monomorphic ventricular ectopy was abolished during ablation and remained absent for at least 30 minutes after ablation in the baseline state and during infusion of isoproterenol.

**Follow-Up**
After ablation, all patients underwent overnight continuous telemetry monitoring. All patients were asked to undergo repeated 24-hour Holter monitoring within 3 months of ablation. Patients presenting with cardiomyopathy were also asked to undergo reassessment of left ventricular function within 3 months of ablation.

**Statistical Analysis**
All data are shown as mean±SD. For categorical variables, comparisons were made with the χ² test. Because normality was absent, comparisons between continuous variables were made by use of a nonparametric test (Mann-Whitney U). For all comparisons, a value of P<0.05 was considered statistically significant.

**Results**
The study population consisted of 27 patients (11 men, 16 women) with a mean age of 47±15 years (range, 16 to 80 years). Their left ventricular ejection fraction was 54±10% (range, 30% to 65%). Overall, 8 patients (30%) had depressed left ventricular function (ejection fraction ≤45%).

The most common symptom that prompted referral for ablation was palpitations (n=19). Other indications included unexplained dyspnea (n=1), presyncope (n=3), and syncpe (n=2). Two asymptomatic patients with unexplained cardiomyopathy were referred for ablation because of concern regarding the possible presence of an ectopy-induced cardiomyopathy. Medical therapy, with a β-blocker and/or calcium channel blocker, had been tried unsuccessfully in 19 patients, and 2 of these patients had been treated unsuccessfully with an antiarrhythmic medication (propafenone in 1 patient, propafenone and sotalol in the other patient). In the remaining 10 patients, catheter ablation was used as primary therapy based on patient preference. β-Blockers and calcium channel blockers were held for 48 hours before electrophysiological testing. Similarly, no patient was taking antiarrhythmic medication at the time of electrophysiological testing.

Eight patients (30%) underwent cardiac MRI, which was normal in all patients. Additionally, exercise stress testing (n=15) or cardiac catheterization (n=12) was normal in all patients. Baseline 24-hour Holter monitoring was performed in 22 patients (81%). A mean of 17 624±12 611 ectopic beats (range, 1950 to 43 000 beats; median, 16 117 beats) was recorded, representing 2% to 44% of the patient’s daily rhythm (Figure 1). In the remaining 5 patients, frequent uniform ectopy was recorded only on telemetry and therefore could not be quantified. In addition to the repetitive monomorphic ventricular ectopy, 16 patients (59%) also had documented nonsustained ventricular tachycardia (3 to 18 beats; median, 4 beats). By design, no patient had a history of sustained ventricular tachycardia.

Programmed ventricular stimulation was performed in 19 patients (70%). Sustained monomorphic ventricular tachycardia was inducible in only 2 patients. Isoproterenol was required for induction in both of these patients; both had tachycardia with a left bundle branch, inferior-axis morphology. The tachycardia terminated after administration of adenosine in 1 patient. In the other patient, tachycardia terminated before an adequate dose of adenosine could be administered. Subsequently, ventricular tachycardia could not be reinduced. Nonsustained ventricular tachycardia (3 and 5 beats) was seen on Holter monitoring in both of these patients. However,
sustained ventricular tachycardia was not inducible in the other 14 patients with a history of nonsustained ventricular tachycardia in whom programmed ventricular stimulation was performed. Sustained ventricular fibrillation with triple ventricular extrastimuli was the only inducible sustained arrhythmia in an additional 4 patients.

We performed detailed mapping of the right ventricle in 28 study patients (96%) (Figure 2). In 1 patient, ambient ventricular ectopy was absent at the time of the study and ventricular tachycardia was not inducible; therefore, the case was aborted. Mapping was performed with the Biosense CARTO electroanatomic mapping system in 25 patients and with the Endocardial Solutions noncontact mapping system in the other 2 patients. Figure 3 shows the sites of origin of the dominant morphology of ventricular ectopy. In 21 patients (78%), there was a single ectopic morphology. In the remaining 6 patients, although there was single dominant focus with a left bundle branch, inferior-axis morphology, 4 patients had ectopy with a second morphology and 2 patients had ectopy with a third morphology. The dominant morphology originated from the anterior (n = 4), posterior (n = 7), anteroseptal (n = 3), midseptal (n = 1), posteroseptal (n = 9), and posterolat-
eral (n=2) aspects of the RVOT (Figure 3). Of the 6 patients with ≥1 ectopic morphology, a left bundle branch, inferior axis was seen in 4 patients, a left bundle branch, superior axis was noted in 1 patient, and a right bundle branch, inferior axis (suggestive of origin within the left ventricular outflow tract) was seen in 1 patient. However, only 1 of these patients had enough sufficient ectopy of an additional morphology to map.

Successful ablation was performed in 23 of 27 patients (85%), including 7 of 8 patients with depressed left ventricular function at baseline. One patient could not tolerate the procedure because of incapacitating chest pain with application of radiofrequency energy. In the 3 other patients, 2 with ectopy originating from the posterior outflow tract and 1 with ectopy originating from the posteroseptal outflow tract, ablation was unsuccessful.

We compared patients with depressed (n=8) left ventricular function at baseline with patients having normal function (n=19; Table 1). Patients with depressed ventricular function were more likely to be older than patients with normal function (58±14 versus 42±18 years; P=0.013). However, the burden of ventricular ectopy was similar in patients with (17 859±13 488 ectopic beats per 24 hours; range, 1950 to 43 000) and without (17 541±11 479 ectopic beats per 24 hours; range, 3746 to 35 664) preserved left ventricular function (P=0.800). Additionally, no differences were observed in the site of ectopy origin within the RVOT (Figure 3).

Successful ablation of the dominant ectopy morphology was performed in 7 of 8 patients with depressed ventricular function (Table 2). In these 7 patients, ablation efficacy was confirmed with repeated Holter monitoring, which demonstrated a reduction in ectopy from 17 541±11 479 beats per 24 hours to 507±722 beats per 24 hours (P=0.028). There was a significant improvement in left ventricular ejection fraction (from 39±6% to 62±6%; P=0.017) at a mean of 8±10 months (median, 3 months) after ablation. In fact, ventricular function normalized in all 7 of these patients with “idiopathic” cardiomyopathy.

Successful ablation of the dominant ectopy morphology was performed in 7 of 8 patients with depressed left ventricular function. Pulmonic and tricuspid valves are outlined in blue and yellow, respectively. His-bundle region is shown by orange circle. This view looks down into outflow tract. Ablation catheter is seen in postero-septal region of outflow tract. Right, This schematic representation shows area encircled by dashed red circle. There was no difference in location of ventricular ectopy origin within RVOT between patients with preserved (□) and depressed (●) left ventricular function. RVA indicates right ventricular apex.

**Figure 3.** Sites of successful ablation. Left, The 3D electroanatomic map showing superior (SUP) view of right ventricle. Pulmonic and tricuspid valves are outlined in blue and yellow, respectively. His-bundle region is shown by orange circle. This view looks down into outflow tract. Ablation catheter is seen in posteroseptal region of outflow tract. Right, This schematic representation shows area encircled by dashed red circle. There was no difference in location of ventricular ectopy origin within RVOT between patients with preserved (□) and depressed (●) left ventricular function. RVA indicates right ventricular apex.

**TABLE 1.** Comparison of Repetitive Monomorphic Ventricular Ectopy Patients With and Without Preserved Left Ventricular Function

<table>
<thead>
<tr>
<th></th>
<th>Normal EF (n=19)</th>
<th>Depressed EF (EF ≤45%) (n=8)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF, %</td>
<td>60±5</td>
<td>39±6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>6 (31)</td>
<td>5 (63)</td>
<td>0.206</td>
</tr>
<tr>
<td>Age, y</td>
<td>42±18</td>
<td>58±14</td>
<td>0.013</td>
</tr>
<tr>
<td>Arrhythmia history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVCs/24 h</td>
<td>17 859±13 488</td>
<td>17 541±11 479</td>
<td>0.800</td>
</tr>
<tr>
<td>Nonsustained VT, n (%)</td>
<td>10 (52)</td>
<td>6 (75)</td>
<td>0.405</td>
</tr>
<tr>
<td>Single PVC morphology, n (%)</td>
<td>15 (79)</td>
<td>6 (75)</td>
<td>0.438</td>
</tr>
<tr>
<td>PVC location within RVOT, n*</td>
<td></td>
<td></td>
<td>0.360</td>
</tr>
<tr>
<td>Anterior</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Posterior</td>
<td>6</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Postero-septal</td>
<td>7</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Midseptal</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterolateral</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

EF indicates ejection fraction; PVCs, premature ventricular contractions; and VT, ventricular tachycardia.

*In 1 patient, there was insufficient ambient ventricular ectopy to map.
Discussion

The principal findings of this study are that repetitive monomorphic ventricular ectopy that originates from the RVOT is an underappreciated cause of unexplained cardiomyopathy that is completely reversible with successful radiofrequency catheter ablation. Patients with ectopy-induced cardiomyopathy are significantly older than patients with preserved ventricular function, suggesting either that these patients are more susceptible to the development of a cardiomyopathy or that the cardiomyopathy has had a longer period of time in which to evolve.

Tachycardia-induced cardiomyopathy has been associated with a spectrum of tachyarrhythmias, including atrial fibrillation and flutter, supraventricular tachycardia, and ventricular tachycardia.\(^1\)\(^-\)\(^8\) Consistent with animal models of tachycardia-induced cardiomyopathy,\(^1\(^9\) these tachycardias are sustained and associated with rapid ventricular rates over an extended period of time. However, the mechanism by which isolated ventricular ectopy results in cardiomyopathy is uncertain.

Although the cellular mechanism for repetitive monomorphic ventricular ectopy is unclear, particularly because ventricular tachycardia is not inducible in most of these patients, it is instructive that 2 patients had isoproterenol-facilitated induction of sustained monomorphic ventricular tachycardia. In these patients, the induced tachycardia had the same morphology as the repetitive monomorphic ventricular ectopy, and adenosine sensitivity was demonstrated in the 1 patient in whom it could be appropriately evaluated. These data suggest, at least in some patients, that cAMP-mediated triggered activity may be an operative mechanism.

To date, there have been only isolated case reports linking repetitive monomorphic ventricular ectopy to cardiomyopathy. Duffee et al\(^1\(^3\) initially described 4 patients who had >20 000 premature ventricular contractions over 24 hours (morphology unknown) and cardiomyopathy (ejection fraction ≤40%) in whom left ventricular function improved substantially (from 27±10% to 49±17%) after either a β-blocker (n = 1) or amiodarone (n = 3) was given to suppress ventricular ectopy. Similar outcomes have been reported in 3 case reports of patients with a very high burden of ventricular ectopy (25 000 to 56 000 ectopic beats per 24 hours) undergoing radiofrequency catheter ablation.\(^1\(^4\)\(^-\)\(^6\) In these case reports, as in our series, ventricular ectopy had a left bundle branch, inferior-axis morphology, and successful ablation was performed from within the RVOT.

On electrophysiological testing, we found that 22% of patients had an additional morphology of ventricular ectopy. Clinical improvement, defined by an improvement in symptoms and/or ventricular function, occurred despite the fact that we did not systematically target these additional morphologies. Therefore, these data suggest that patients should not be considered ineligible for ablation simply because multiple morphologies of ventricular ectopy are observed.

Another finding is that ectopy-induced cardiomyopathy was observed in patients with as few as 5500 daily premature beats, which suggests that this entity should be considered in any patient with unexplained cardiomyopathy in whom monomorphic ventricular ectopy is observed. Although day-to-day variability in the burden of ectopy likely plays some part, it is unclear why some patients develop cardiomyopathy and other patients (who often have significantly greater amounts of ectopy) do not.

Finally, we observed that ventricular function normalized in all patients in whom cardiomyopathy was considered “idiopathic.” A new observation is that older patients are more likely to develop cardiomyopathy. Further studies are needed to determine whether the development of a cardiomyopathy simply reflects a time-dependent process or is linked to other, as-yet unidentified, variables.

Study Limitations

Because the study population comprises patients referred for ablation, we could not determine the prevalence of this disorder or whether the same improvement in ventricular function would have been observed regardless of ablation. However, in the 1 patient with depressed ventricular function in whom ablation was unsuccessful, left ventricular function remained depressed during follow-up. This suggests that the normalization in ventricular function observed in some patients is directly related to successful ablation. Second, after successful ablation, repeated Holter monitoring showed some residual ventricular ectopy in most patients. However, because 12-lead information was not available, it was not possible to compare the morphology of the residual ectopy with that targeted for ablation. Third, whether the same

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TABLE 2. Patients With Repetitive Monomorphic Ventricular Ectopy and Depressed Left Ventricular Function

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Presenting Symptom</th>
<th>Origin of CMP</th>
<th>Cardiac Medications</th>
<th>PVC Origin in RVOT</th>
<th>RFA Success</th>
<th>Initial Holter, PVCs/24 h</th>
<th>F/U Holter, PVCs/24 h</th>
<th>Initial EF, %</th>
<th>F/U EF, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37</td>
<td>F</td>
<td>Dyspnea</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Anterior</td>
<td>Yes</td>
<td>5502</td>
<td>44</td>
<td>38</td>
<td>65</td>
</tr>
<tr>
<td>2</td>
<td>51</td>
<td>M</td>
<td>None</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Postero septal</td>
<td>Yes</td>
<td>26 491</td>
<td>1893</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>3</td>
<td>80</td>
<td>M</td>
<td>Presyncope</td>
<td>Idiopathic</td>
<td>None</td>
<td>Anteroseptal</td>
<td>Yes</td>
<td>35 664</td>
<td>1100</td>
<td>35</td>
<td>55</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>M</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>None</td>
<td>Anterior</td>
<td>Yes</td>
<td>9791</td>
<td>5</td>
<td>35</td>
<td>60</td>
</tr>
<tr>
<td>5</td>
<td>71</td>
<td>F</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Postero septal</td>
<td>Yes</td>
<td>23 352</td>
<td>117</td>
<td>43</td>
<td>65</td>
</tr>
<tr>
<td>6</td>
<td>62</td>
<td>M</td>
<td>Presyncope</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Posterolateral</td>
<td>Yes</td>
<td>...</td>
<td>332</td>
<td>45</td>
<td>65</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>M</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>None</td>
<td>Posterolateral</td>
<td>Yes</td>
<td>16 362</td>
<td>55</td>
<td>45</td>
<td>65</td>
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<tr>
<td>8</td>
<td>68</td>
<td>F</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Posterolateral</td>
<td>No</td>
<td>56 262</td>
<td>12 883</td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>

CMP indicates cardiomyopathy; PVCs, premature ventricular contractions; RFA, radiofrequency ablation; F/U, follow-up; and EF, ejection fraction.

*Patient did not have a 24-hour Holter; however, frequent ambient ectopy was noted on in-patient telemetry.
outcome would be seen in patients whose ectopy originates from other anatomic locations is also unknown.

Theoretically, it is possible that frequent ventricular ectopy could interfere with accurate measurement of ventricular function. However, several findings suggest that ventricular dysfunction is a “real” clinical phenomenon in some patients with repetitive monomorphic ventricular ectopy originating from the RVOT. First, ventricular dysfunction is not observed in 70% of patients despite the fact that these patients have a burden of ectopy similar to that of patients with ventricular dysfunction. Second, as seen in Figure 1, despite the presence of ventricular ectopy, the predominant rhythm is sinus in most patients. Therefore, it is unlikely that the ventricular ectopy artifactually affects measurement of ventricular function. Third, in the 1 patient with depressed ventricular function in whom ablation was unsuccessful, ventricular function remained depressed, and overt congestive heart failure ensued.

A final limitation is that in a few patients, the preablation ejection fraction was measured by ventriculography, whereas postablation left ventricular function was assessed by echocardiography. However, these tests are routinely used interchangeably in clinical practice to assess ventricular function.

Clinical Implications
In patients presenting with repetitive monomorphic ventricular ectopy with left bundle-branch, inferior-axis morphology, left ventricular function should be carefully assessed. The presence of unexplained ventricular dysfunction should raise concern about an ectopy-induced cardiomyopathy, and these patients should be considered for ablation, even if they are otherwise asymptomatic. Patients whose ventricular function is preserved and whose symptoms do not warrant ablation should be followed up serially to exclude the development of cardiomyopathy over time.

Acknowledgments
This work was supported in part by grants from the National Institutes of Health (RO1-HL-56139), the Raymond and Beverly Sackler Foundation, and the Michael Wolk Heart Foundation.

References
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_Circulation_. 2005;112:1092-1097; originally published online August 15, 2005; doi: 10.1161/CIRCULATIONAHA.105.546432

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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