Conduction Abnormalities in Pediatric Patients With Restrictive Cardiomyopathy

Mark A. Walsh, MD; Michelle A. Grenier, MD; John L. Jefferies, MD, MPH; Jeffrey A. Towbin, MD; Angela Lorts, MD; Richard J. Czosek, MD

Background—Pediatric restrictive cardiomyopathy carries a poor prognosis secondary to a high risk of sudden death previously attributed to ventricular tachyarrhythmias. The extent of conduction abnormalities in this population and their relationship to life-threatening events has not been previously reported.

Methods and Results—A retrospective study of pediatric patients with restrictive cardiomyopathy diagnosed between April 1994 and May 2011 was performed. Demographic, cardiac, and ECG characteristics and the mechanisms of serious arrhythmic events (death or episode of acute hemodynamic compromise thought to be secondary to arrhythmia) were evaluated. Sixteen patients (1–17 years of age) were reviewed, with 5 sudden cardiac events noted, including 4 deaths. Two deaths were caused by development of acute heart block; another patient with syncope had intermittent heart block and survived as the result of pacing features of an implanted defibrillator system. The median PR interval (222 versus 144 ms; \( P < 0.01 \)) and the QRS duration (111 versus 74; \( P = 0.01 \)) were significantly longer in those who had an acute cardiac event. Older age at presentation was associated with sudden cardiac events (\( P < 0.01 \)). No other functional or echocardiographic variables were associated with a sudden cardiac event.

Conclusions—Pediatric patients with restrictive cardiomyopathy are at risk for acute high-grade heart block, and, in this cohort, bradycardic events represented a significant portion of all arrhythmic events. Aggressive ECG monitoring strategies looking for conduction system disease should be ongoing in all patients with restrictive cardiomyopathy. Implantation of a defibrillator/pacemaker should be considered as prophylactic management. (Circ Heart Fail. 2012; 5:267-273.)

Key Words: cardiomyopathy ■ pediatrics ■ restrictive cardiomyopathy ■ sudden cardiac death ■ arrhythmia ■ conduction abnormalities

Restrictive cardiomyopathy (RCM) is thought to account for approximately 2–5% of cases of pediatric cardiomyopathy, with an estimated incidence of 0.03/100 000 children. The pathological mechanism that underpins this condition is impaired diastolic function with relatively preserved systolic function. Elevated end-diastolic pressures cause markedly enlarged atria, which is an echocardiographic hallmark of the disease. In adults, RCM is usually caused by infiltrative diseases such as amyloidosis and sarcoidosis and often has a protracted indolent course. Pediatric patients differ markedly, demonstrating a mean survival of 2 years from the time of diagnosis. Sudden death is common, although the precise mechanisms are not completely understood. In addition, even for those who are preemptively listed for cardiac transplantation, the attrition rate is relatively high compared with other cardiomyopathies. Syncope at presentation carries a particularly poor prognosis, with death occurring within 4 months in one recent report. Evidence of myocardial ischemia has been shown to predict death within months, suggesting that lethal ventricular arrhythmias are a precipitating event for sudden death. Conduction abnormalities such as complete heart block, either as a consequence of the primary disease process or secondary to myocardial ischemia, have not been described in pediatric patients with RCM; however, heart block could potentially be a precipitating event for sudden cardiac death in these patients, a precedent set for adults with mutations in desmin.

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We reviewed a cohort of patients with restrictive cardiomyopathy from a single institution. The purpose of the study was to ascertain whether there is a subset of patients with RCM who have conduction system disease and determine whether these children have an increased risk of sudden death. If this is the case, then knowledge of conduction system abnormalities and how they might relate to sudden cardiac death would permit better risk stratification, treatment algorithms, and modification of listing status or prophylactic device implantation in these children.
Methods

This single institutional, retrospective study was conducted with approval of the Institutional Review Board at Cincinnati Children’s Hospital Medical Center (IRB No. 00002988). Patients with isolated restrictive cardiomyopathy diagnosed between April 1994 and May 2011 were included in the study. Patients with hypertrophic cardiomyopathy with “restrictive features” were not evaluated in this analysis. The diagnosis of restrictive cardiomyopathy was made by experienced cardiologists in a cardiomyopathy-specific clinic and determined primarily through echocardiography criteria with additional cardiac catheterization data and genetic analysis used for further stratification. Echocardiography criteria included dilated atria in the absence of features of hypertrophic cardiomyopathy (hypertrophy of the interventricular septum or left ventricular free wall), evidence of diastolic dysfunction, and relatively preserved left ventricular systolic function (shortening fraction normal for age or ejection fraction >50%) at the time of diagnosis. Atrial volumes were calculated using the American Society of Echocardiography recommended area length method in 2- and 4-chamber views. Atrial volumes were indexed to body surface area. Data from echocardiography was combined with catheterization and genetic data when available.

Patient demographic and clinical outcomes were accessed through electronic medical records and cardiology databases. A sudden cardiac event was defined as death or an episode of acute hemodynamic compromise or syncope presumed to be arrhythmia-related. Echocardiogram-derived variables such as ventricular dysfunction, chamber dimensions, septal and wall thicknesses, and indexed left atrial volumes were collected at presentation and latest follow-up. Cardiac catheterization data included right heart pressure indices, left heart pressure indices, and pulmonary vascular resistance measurements. Baseline surface ECG conduction intervals such as PR, QRS, and QTc intervals over time were collected from every ECG on each patient. For comparison, ECG intervals were compared at the time of initial patient presentation and at the latest time point. Initial presentation was defined as the ECG obtained at the time of the patients initial diagnosis. In patients with sudden cardiac events, all ECGs were obtained before their events. Because ECG intervals are age-dependent, 2 additional comparisons were performed. The first compared absolute z-score for PR and QRS intervals and the second compared the proportion of patients in each group with PR and QRS intervals greater than the 98th percentile for age. Holter monitors and exercise tests were reviewed when available. If patients underwent cardiac transplantation, data subsequent to transplantation were not included.

Statistics

ECG characteristics, echocardiogram, and catheterization data were compared between those patients who had a sudden cardiac event and those who did not have a sudden cardiac event using nonparametric methods (Mann-Whitney test). Categorical variables such as the presence of ongoing heart failure were compared using the Fisher exact test. All statistical analysis was performed using SAS 9.2 (Cary, NC). ECG data were also plotted from the time of first diagnosis to either the latest follow-up or to death/cardiac transplantations for the PR, QRS, and QTc intervals. Genetic data when available are displayed in table format.

Results

Clinical Data

Between April 1994 and May 2011, 16 patients (8 males, 8 females) were diagnosed with restrictive cardiomyopathy and treated at our institution, with a median age at diagnosis of 8 years (range, 1–17 years). In 12 patients there was a family history of cardiomyopathy, and 5 patients presented secondary to their family history and were asymptomatic. Nine patients had genetic testing performed, with 8 patients having a genetically defined cause of RCM identified (Table 1). Five patients developed ongoing symptoms of heart failure at the time of this study. Older age at presentation (P=0.01) and ongoing symptoms of heart failure (P=0.02) were associated with a sudden cardiac event (Table 2). None of the other demographic variables or genetic variants was associated with a sudden cardiac event.

There were 5 sudden cardiac events, including 4 deaths and 1 patient with arrhythmia-related syncope. All 4 patients who died were in the hospital at the time of death. One patient died hours after a cardiac catheterization, though not directly related to mechanical atrioventricular (AV) node damage or other intraprocedural complications. In 2 patients, acute demise was directly related to documented development of abrupt heart block (Figure 1). In 1 case, it was not possible to institute mechanical circulatory support. In the second case, heart block developed abruptly in the cardiac intensive care while awaiting transplantation. Although circulatory support was initiated immediately, death occurred 3 weeks after initiation of support. The third patient developed an acute ventricular arrhythmia followed by asystolic while hospitalized for concomitant minor hematologic issues. In the final patient, relative bradycardia was noted preceding death, though no telemetry strips were available for review. The patient with arrhythmia syncope demonstrated left bundle-branch block on presentation and underwent a transvenous pacing study. This study demonstrated an infra-His block and inducible ventricular tachycardia prompting implantation of a permanent defibrillator with pacemaker capacity. Over the next month, the patient presented with intermittent block and had subsequent permanent heart block with no ventricular escape rhythm. In total, 11 patients did not have a sudden cardiac event. Two of these patients underwent cardiac transplantation one of whom is currently alive.

Risk Factors Associated With an Acute Cardiac Event

Demographic, echocardiographic, and cardiac catheterization data and their statistical association with a sudden cardiac event are shown in Table 2. Moderate left ventricular dysfunction developed over time in 2 patients both of whom had a sudden cardiac event, though this was not statistically significant (P=0.09). Otherwise, no echocardiographic variables or catheterization derived variables were found to be associated with an acute cardiac event. ECGs obtained at presentation in addition to all follow-up ECGs were reviewed on every patient. Measurements of PR, QRS, and QTc intervals were analyzed from the time of initial diagnosis to the latest follow-up. The PR interval at presentation (P=0.03; median=166 versus 134 ms) and at the time of last follow-up (P<0.01; median=222 versus 144 ms) was significantly longer in patients who had an acute cardiac event compared with those without an acute cardiac event. Figure 2 demonstrates that patients with PR interval prolongation at initial diagnosis tended to develop increased PR prolongation over time, whereas those who had a shorter PR interval at presentation remained stable though part of this effect may be influenced by increasing patient age over time. Two patients manifested left bundle-branch block, and both of these patients had subsequent acute heart block. One child had
Table 1. Demographics of Patients With Restrictive Cardiomyopathy

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age at Diagnosis, Years</th>
<th>Family History</th>
<th>Sudden Cardiac Event</th>
<th>Genetic Testing</th>
<th>Mutation Type</th>
<th>Mutation</th>
<th>ECG Conduction Abnormality</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>2</td>
<td>Yes</td>
<td>No</td>
<td>Negative</td>
<td>NA</td>
<td>Unknown</td>
<td>None</td>
<td>Listed for cardiac transplant, no events to date</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>9</td>
<td>Yes</td>
<td>Yes</td>
<td>Not tested</td>
<td>NA</td>
<td>Unknown</td>
<td>FAVB</td>
<td>Died, ventricular tachyarrhythmia</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>8</td>
<td>Yes</td>
<td>No</td>
<td>†Desmin</td>
<td>Heterozygous  c.638C&gt;T (Ala213Val) variant in Exon 2 of Desmin</td>
<td>Deletion</td>
<td>None</td>
<td>Died after cardiac transplantation</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td>Not tested, negative testing in first-degree relative</td>
<td>NA</td>
<td>Unknown</td>
<td>None</td>
<td>No events to date</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>12</td>
<td>Yes</td>
<td>Yes</td>
<td>Not tested, sibling with †MYL2</td>
<td>Heterozygous  359G&gt;A (R120Q) variant in Exon 6 of MYL2</td>
<td>Missense</td>
<td>LBBB</td>
<td>Died after episode of acute heart block while awaiting transplantation</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>11</td>
<td>Yes</td>
<td>Yes</td>
<td>†MYL2</td>
<td>Heterozygous  359G&gt;A (R120Q) variant in Exon 6 of MYL2</td>
<td>Missense</td>
<td>FAVB</td>
<td>Died after episode of acute heart block while awaiting transplantation</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>3</td>
<td>No</td>
<td>No</td>
<td>Negative</td>
<td>NA</td>
<td>Unknown</td>
<td>RBBB</td>
<td>No events to date</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>16</td>
<td>No</td>
<td>Yes</td>
<td>†MYBPC3 and *Desmin</td>
<td>Heterozygous Asp605Gly of MYBPC3 Heterozygous Arg454Trp of Desmin</td>
<td>Missense and missense</td>
<td>LBBB</td>
<td>ICD implantation for syncope; development of CHB; listed for transplantation</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>17</td>
<td>Yes</td>
<td>No</td>
<td>*TNNT2</td>
<td>Heterozygous  c.487_489delGAG</td>
<td>Deletion</td>
<td>None</td>
<td>ICD implantation as primary prophylaxis, no events to date</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>16</td>
<td>No</td>
<td>Yes</td>
<td>Not tested</td>
<td>NA</td>
<td>Unknown</td>
<td>FAVB</td>
<td>Died suddenly with bradycardia</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>1</td>
<td>No</td>
<td>No</td>
<td>*MYH7</td>
<td>Heterozygous  2302G&gt;A (G768R) in Exon 21 of MYH7</td>
<td>Missense</td>
<td>None</td>
<td>Transplanted</td>
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<tr>
<td>12</td>
<td>F</td>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td>Not tested, negative testing in fist degree relative</td>
<td>NA</td>
<td>Unknown</td>
<td>None</td>
<td>No events to date</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>4</td>
<td>Yes</td>
<td>No</td>
<td>†Desmin</td>
<td>Heterozygous  c.638C&gt;T (Ala213Val) variant in Exon 2 of Desmin</td>
<td>Deletion</td>
<td>None</td>
<td>No events to date</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>1.5</td>
<td>Yes</td>
<td>No</td>
<td>Not tested, negative testing in fist degree relative</td>
<td>NA</td>
<td>Unknown</td>
<td>None</td>
<td>Listed for cardiac transplant, no events to date</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>9</td>
<td>Yes</td>
<td>No</td>
<td>Not tested, parent with MYH7</td>
<td>c.1357C&gt;T (p.Arg453Cys) in Exon 14 of MYH7</td>
<td>Missense</td>
<td>None</td>
<td>Listed for cardiac transplant, no events to date</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>16</td>
<td>Yes</td>
<td>No</td>
<td>*TNNT2</td>
<td>Heterozygous  c.487_489 del GAG</td>
<td>Deletion</td>
<td>None</td>
<td>ICD implantation as primary prophylaxis, no events to date</td>
</tr>
</tbody>
</table>

Acute cardiac event is defined as death or an episode of acute hemodynamic compromise secondary to a cardiac arrhythmia.
NA indicates not applicable; FAVB, first-degree atrioventricular block; RBBB, right bundle-branch block; LBBB, left bundle-branch block; ICD, implantable cardioverter-defibrillator.

* Disease-causing mutation.
† Variant of unknown significance.
‡ Probably disease-causing mutation.
heart block after a diagnostic catheterization procedure and died acutely; the other had an acute cardiac event requiring an implantable defibrillator. One patient manifested right bundle-branch block and did not have an acute cardiac event. The median QRS duration at presentation (median=92 versus 70 ms) and at last follow-up (median=111 versus 74 ms) was significantly longer in those that had an acute cardiac event compared with those who did not have an acute cardiac event. The QRS duration tended to remain unchanged over time in both groups of patients. Figure 3 demonstrates box plots for both PR and QRS intervals at the time of initial presentation and at the time of

<table>
<thead>
<tr>
<th>Demographic factors</th>
<th>Acute Cardiac Event</th>
<th>No Acute Cardiac Event</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (range)</td>
<td>12 (9–16)</td>
<td>2.5 (1–17)</td>
<td>0.01</td>
</tr>
<tr>
<td>Male sex</td>
<td>n=2</td>
<td>n=6</td>
<td>0.37</td>
</tr>
<tr>
<td>Heart failure</td>
<td>n=2</td>
<td>n=1</td>
<td>0.39</td>
</tr>
<tr>
<td>Chronic heart failure</td>
<td>n=4</td>
<td>n=1</td>
<td>0.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG at presentation</th>
<th>PR interval, ms 166 (148–234)</th>
<th>134 (118–159)</th>
<th>0.03</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG at latest follow-up, or before a sudden cardiac event</td>
<td>PR interval, ms 222 (168–278)</td>
<td>144 (112–158)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

| Echocardiogram at presentation | LVEDD z-score悲观 | 0.64 (2.5 to 1.46) | 0.53 |
| LV dysfunction | n=0 | n=0 | NA |
| Indexed left atrial volume | 50 (37–81) | 43 (26–113) | 0.42 |

| Cardiac catheterization data (n=8) | PVRI, Woods U/m² 1.6 (1–5) | 3.4 (2.1–4.7) | 0.37 |
| PA pressure, mm Hg 27 (19–32) | 27 (20–66) | 0.85 |
| RVSP, mm Hg 40 (36–44) | 36 (24–86) | 0.78 |
| RVED, mm Hg 19 (10–21) | 19 (10–23) | 0.71 |
| Right atrial pressure, mm Hg 17 (15–20) | 12 (6–19) | 0.14 |
| Left atrial pressure, mm Hg 21 (7–25) | 18 (11–20) | 0.57 |
last follow-up or before sudden cardiac event. The QT interval trended toward being longer in those with an acute cardiac event; however, this was not statistically significant. The length of the QTc duration over time was quite variable, especially in those who had an acute cardiac event. Finally, the diagnosis of left atrial enlargement on ECG was not associated with a sudden cardiac event (P = 0.53).

Discussion
Pediatric RCM is a rare entity, accounting for only 2.5–5% of all pediatric cardiomyopathies. Clinical prognosis of RCM in children is very different from the adult phenotype. The actuarial 2-year survival from the time of diagnosis in recent studies is reported to be <50%. For these patients, cardiac transplantation appears to be the only viable treatment available currently, with overall 5- and 10-year survival being 70% and 60%, respectively. Despite this, waiting list mortality remains a significant problem for these patients. Although most of these deaths have been attributed to tachyarrhythmias, there is a sparsity of data documenting the modes of death in this patient population. This study shows that conduction system abnormalities occur with a reasonably high frequency in this patient population. It also shows that in a significant portion of this small cohort, bradyarrhythmia and development of abrupt heart block may be a precipitating event in sudden death episodes. The rate of sudden death in our study of 25% is similar to other pediatric RCM studies.

Many studies have looked to risk-stratify this patient group, as survival of up to 8–12 years has been described. Risk factors that have been identified, albeit inconsistently, include cardiomegaly, younger age, thromboembolism, raised pulmonary vascular resistance, pulmonary venous congestion, syncope, chest pain, and left atrial size. Rivenes et al looked at risk factors for sudden death in a similar sized cohort of 18 patients with RCM; chest pain and syncope were identified as risk factors for sudden death. Histopathologic evidence for ischemia was found in the majority of patients who died and Holter monitor evidence of ischemia predicted death within several months. They proposed that the mode of death in the sudden death group was lethal ventricular arrhythmias and showed examples of ventricular tachycardia/fibrillation to support the speculation.

Five patients in our study had acute cardiac events, with 3 patients having complete heart block. Two of the patients with heart block died. The third patient had a defibrillator placed and went on to develop complete heart block with pacing dependency. In 1 of these cases, ST-segment elevation was documented before the onset of complete heart block, indicating that there may have been an underlying ischemic process which resulted in heart block. AV block in this patient probably caused a critical reduction in cardiac output due to the loss of atrial contribution toward diastolic filling. None of the echocardiogram variables noted at presentation were associated with an increased risk of death, although left ventricular dysfunction before an event approached statistical significance. It is unlikely that changes in ECG parameters such as PR and QRS duration had direct physiological effect and is more likely that these are indicators of preexisting conduction disease at presentation or it may be a marker for underlying disease severity. Nevertheless, the documentation of abrupt heart block in a significant portion of this population warrants thorough investigation in patients.

**Figure 1.** Development of abrupt heart block. A. Development of complete heart after a cardiac catheterization after a period of PR prolongation and atrial standstill resulting in sudden cardiac death. B. Complete heart block after documented ST-segment depression. This resulted in acute cardiovascular decompensation and initiation of extracorporeal membrane oxygenation. Marker A represents atrial activation (p wave). Marker V represents ventricular activation (QRS complexes).

**Figure 2.** Change in PR interval from time of diagnosis. PR interval is in milliseconds. **Gray lines** represent those who had an acute cardiac event; **black lines** represent those who did not have an acute cardiac event. It is noteworthy that those with a longer interval tended to develop increased prolongation, whereas those with a shorter PR interval tended to remain unchanged. If patients were transplanted, then the PR interval is not shown after this point.
with baseline conduction abnormalities. These findings suggest that identification of conduction system disease should trigger increased surveillance and consideration of potential permanent pacing as a prophylactic therapy either while awaiting cardiac transplantation or as a means to postpone listing for transplantation in patients with normal pulmonary vascular resistance.

In analysis of potential genetic drivers, the subjects with bradycardia-related events in whom genetic mutations were identified included those patients with mutations in desmin and MYL2. In the case of desmin, it is well known that mutations in this intermediate filament-protein–encoding gene can result in RCM with bradycardia or AV block, with or without skeletal myopathy. In our cases, no skeletal muscle abnormalities were noted. In the case of MYL2, reports of sudden cardiac disease are notable in subjects with these mutations. Although most reports do not identify the cause of death, there are reports in which bradycardia and AV block are reported.

Conduction system disease in these patients probably has multifactorial causation. Patients are at risk of ischemic injury to the AV node and His-Purkinje system. Siegel et al studied 4 adult patients with restrictive cardiomyopathy and reported that 3 of these patients required pacemakers, 2 for tachycardia-bradycardia syndrome and another patient for complete heart block. Autopsy samples showed extensive fibrosis in the His-bundle Purkinje system in all 3 cases that required a pacemaker. Another potential mechanism of conduction system disease might relate to atrial and ventricular dilation as a consequence of disease progression. Also, many of these patients have structurally abnormal myocytes with abnormal gap junctions that are thought to predispose to developing conduction abnormalities. Whatever the precise mechanism, it is clear from the data that conduction system disease can develop in pediatric patients with restrictive cardiomyopathy and potentiate the risk of sudden death. PR prolongation, QRS widening, and left bundle-branch block merit particular attention and should be thoroughly investigated with routine ambulatory monitoring and consideration for prophylactic pacing, probably as part of an implantable cardioverter-defibrillator system. Although permanent pacing and defibrillator implantation may not alter the natural history of this disease, pacing for patients with advanced conduction system disease may be life-saving. It should be noted that there are no studies documenting the efficacy of defibrillator systems in large pediatric cohorts, and special attention should be taken to avoid inappropriate therapies that may be particularly detrimental in this population.

The limitations of this study are its retrospective nature and all of the inherent bias associated with a retrospective studies. PR prolongation and QRS widening were associated with sudden cardiovascular decompensation; however, given the small number of events that were seen, these results should be interpreted with caution. Also, because this study does not confirm causality, these risk factors may not predict those at-risk for sudden death, rather they may represent disease severity or be associated with more malignant genotypes/phenotypes. Last, several important echocardiographic and catheterization measures of diastolic dysfunction were not able to be analyzed secondary to incomplete data.

**Conclusions**

Pediatric patients with RCM are at risk for high-grade AV block. In this study, PR prolongation and a wider QRS complex were associated with an acute cardiac event. The effectiveness of prophylactic pacing in this population as a bridge to cardiac transplantation or potentially as a means to delay listing for cardiac transplantation in patients with preserved pulmonary vascular resistance must be evaluated.
Disclosures

Dr Towbin received a research grant from the Pediatric Cardiomyopathy Registry (National Institutes of Health grant) and served as consultant/on the advisory board, Children’s Cardiomyopathy Foundation, Scientific Advisory.

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_Circ Heart Fail_. 2012;5:267-273; originally published online January 19, 2012;
doi: 10.1161/CIRCHEARTFAILURE.111.964395
_Circulation: Heart Failure_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

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