The Incidence of Pediatric Cardiomyopathy in Two Regions of the United States

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ABSTRACT

BACKGROUND
Population-based data on the incidence of pediatric cardiomyopathy are rare because of the lack of large, prospective studies.

METHODS
Since 1996 the Pediatric Cardiomyopathy Registry sponsored by the National Heart, Lung, and Blood Institute has collected data on all children with newly diagnosed cardiomyopathy in New England and the Central Southwest region (Texas, Oklahoma, and Arkansas) of the United States. We report on all children in these regions who received this diagnosis between 1996 and 1999.

RESULTS
We identified 467 cases of cardiomyopathy, for an overall annual incidence of 1.13 per 100,000 children (95 percent confidence interval, 1.03 to 1.23). The incidence was significantly higher among infants younger than 1 year old than among children and adolescents who were 1 to 18 years old (8.34 vs. 0.70 per 100,000, P<0.001). The annual incidence of cardiomyopathy was lower among white children (upper-bound estimate, 1.06 cases per 100,000) than among black children (lower-bound estimate, 1.47 per 100,000; P=0.02) and higher among boys than among girls (1.32 vs. 0.92 per 100,000, P<0.001). The incidence also varied significantly by region: 1.44 cases per 100,000 in New England and 0.98 per 100,000 in the Central Southwest region (P<0.001). When categorized according to type, dilated cardiomyopathy made up 51 percent of the cases, hypertrophic cardiomyopathy 42 percent, and restrictive or other types 3 percent; 4 percent were unspecified. There was no significant difference in the incidence rates according to the year.

CONCLUSIONS
The estimated incidence of pediatric cardiomyopathy in two large regions of the United States is 1.13 cases per 100,000 children. Most cases are identified at an early age, and the incidence varies according to sex, region, and racial or ethnic origin.
Cardiomyopathy is a very serious disorder in children, and nearly 40 percent of children who present with symptomatic cardiomyopathy receive a heart transplant or die within the first two years.\textsuperscript{1} The time to transplantation or death for children with cardiomyopathy has not improved during the past 35 years, and the outcomes in the most economically advanced nations are no better than those in developing nations.\textsuperscript{2-4}

The cardiomyopathies have an associated cost of nearly $200 million per year in adults and children in the United States alone.\textsuperscript{5,6} The percentage of children with cardiomyopathy who receive a heart transplant has not declined over the past 10 years, and cardiomyopathy remains the leading cause of transplantation in children over one year of age.\textsuperscript{7} The true incidence of pediatric cardiomyopathy is unclear. In a retrospective study of idiopathic dilated cardiomyopathy in children in Finland, the estimated incidence was 0.65 case per 100,000.\textsuperscript{8,9} Results of a 10-year study in Australia, which appear elsewhere in this issue of the Journal, suggest an incidence of pediatric cardiomyopathy of 1.24 cases per 100,000 children, with an annual incidence of dilated cardiomyopathy of 0.73 per 100,000 children and an incidence of hypertrophic cardiomyopathy of 0.32 per 100,000 children.\textsuperscript{10}

The assessment of the incidence of pediatric cardiomyopathy in the United States has been confounded by socioeconomic, geographic, racial, and ethnic diversity. Information is based on small retrospective studies, many of which were done before the advent of echocardiography.

This study presents results from a registry begun in 1996 that prospectively attempted to identify all cases of pediatric cardiomyopathy in two geographically distinct regions of the United States. Our goal was to determine the annual incidence of pediatric cardiomyopathy in these regions and to determine whether the incidence varies according to region, racial or ethnic group, time, sex, age, and type of cardiomyopathy.

METHODS

STUDY DESIGN

The design of the Pediatric Cardiomyopathy Registry, which is sponsored by the National Heart, Lung, and Blood Institute, has been described elsewhere.\textsuperscript{11} All patients presenting to pediatric cardiologists in 18 centers in New England and 20 centers in the Central Southwest (in Texas, Oklahoma, and Arkansas) are prospectively identified and entered into the registry database. A survey of pediatric practices in these regions has indicated that the clinical sites identified nearly all diagnosed cases, with the possible exception of one center in Oklahoma. Patients at this site are sometimes referred to Texas and registered there, but we estimate that two to five cases per year are missed. Patients who lived in the New England or Central Southwest region but who received care outside that region were still included.

This analysis is based on the prospective component of the Pediatric Cardiomyopathy Registry database, consisting of patients who have received a diagnosis of cardiomyopathy since January 1, 1996. The protocol was approved by the institutional review board or ethics committee at every participating site.

ELIGIBILITY CRITERIA

To be eligible for this analysis, a patient was required to live in one of the two regions, to be younger than 18 years of age at diagnosis, and to have one of the following: echocardiographic evidence of cardiomyopathy, including at least two left ventricular measurements (fractional shortening, posterior-wall thickness, or end-diastolic dimension or volume) exceeding 2 SD for age (fractional shortening) or for body-surface area (all other measurements);\textsuperscript{12} an echocardiographic pattern of cardiomyopathy, with localized ventricular hypertrophy or restrictive cardiomyopathy or a contracted form of endocardial fibroelastosis; a pathological diagnosis of cardiomyopathy at autopsy or endomyocardial biopsy; or other clinical evidence of cardiomyopathy provided by the cardiologist. There were 14 clinical exclusion criteria, including a congenital heart defect not associated with a malformation syndrome, endocrine disease known to cause myocardial damage, chronic arrhythmia, pulmonary parenchymal or vascular disease, immunologic disease, chemotherapy-associated cardiotoxicity, and drug use known to cause hypertrophy.\textsuperscript{11}

DATA COLLECTION

Data for the Pediatric Cardiomyopathy Registry are collected through on-site abstraction of records by a trained outreach team or research staff at the participating clinical site. This report is based on data collected at enrollment (defined as the month of diagnosis). Enrollment postcards and data-collection forms with patient-identification labels generated
by computer at the data-coordinating center are sent to each site. The study coordinator, in conjunction with the cardiologists at each site, is responsible for identifying all local cases of pediatric cardiomyopathy. The completeness of the data base has been assessed in multiple ways. After the study period included in this report, we conducted a survey of all 239 adult cardiology practices in Rhode Island and Arkansas. The cardiologists were asked whether they cared for any children with cardiomyopathy who were not also under the care of a pediatric cardiologist. None of the 154 respondents reported caring for such a patient. In 1998, at a single large New England institution, we electronically scanned all echocardiograms obtained during the year and identified more than 600 children who appeared to meet the inclusion criteria for our study. However, all these children were either already registered in our data base or met exclusion criteria. In the same year, three institutions participated in a review of International Classification of Diseases, 9th Revision (ICD-9) discharge codes in hopes of identifying children with cardiomyopathy who had not already been included in our registry with the use of our conventional methods. The reviews uncovered no new cases.

**Statistical Analysis**

Numerators for the reported incidence rates are based on patients who received a diagnosis of cardiomyopathy between 1996 and 1999. Population estimates (incidence-rate denominators) are taken from the 1990 U.S. Census, with an in- and out-migration algorithm applied to obtain year-specific population estimates. Three racial and ethnic categories are presented: white, black, and Hispanic. For these categories, the Pediatric Cardiomyopathy Registry data consisted of a single question with a choice of white, black, or Hispanic. However, the Census cross-tabulates Hispanic status according to racial category. Therefore, we calculated both lower-bound and upper-bound estimates of race-specific incidence rates. The lower-bound estimate includes all white Hispanics and black Hispanics in the white and black population counts, respectively, and the upper bound includes none of the white Hispanics and black Hispanics in the white and black population counts, respectively. The lower-bound estimate for the Hispanic rate includes all Hispanics of any race (white, black, or other race) in the population count, and the upper-bound estimate for the Hispanic rate includes only Hispanics with a racial background not classified as white or black in the population count. The latter estimate has a high variance and instability because of the very small estimated Hispanic population, and thus, we conducted statistical comparisons only between white and black patients. This comparison was conducted in the most conservative fashion—that is, the upper-bound incidence for whites was compared with the lower-bound incidence for blacks.

Because of the rarity of observed cases, incidence rates in various subgroups (region, year, sex, racial

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. of Patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year of diagnosis</td>
<td></td>
</tr>
<tr>
<td>1996</td>
<td>132 (28)</td>
</tr>
<tr>
<td>1997</td>
<td>121 (26)</td>
</tr>
<tr>
<td>1998</td>
<td>114 (24)</td>
</tr>
<tr>
<td>1999</td>
<td>100 (21)</td>
</tr>
<tr>
<td>Region</td>
<td></td>
</tr>
<tr>
<td>New England</td>
<td>186 (40)</td>
</tr>
<tr>
<td>Central Southwest</td>
<td>281 (60)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>281 (60)</td>
</tr>
<tr>
<td>Female</td>
<td>186 (40)</td>
</tr>
<tr>
<td>Racial or ethnic group</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>270 (58)</td>
</tr>
<tr>
<td>Black</td>
<td>73 (16)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>108 (23)</td>
</tr>
<tr>
<td>Other</td>
<td>16 (3)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>&lt;1 yr</td>
<td>193 (41)</td>
</tr>
<tr>
<td>1 to &lt;6 yr</td>
<td>71 (15)</td>
</tr>
<tr>
<td>6 to &lt;12 yr</td>
<td>66 (14)</td>
</tr>
<tr>
<td>12 to &lt;18 yr</td>
<td>137 (29)</td>
</tr>
<tr>
<td>Type of cardiomyopathy</td>
<td></td>
</tr>
<tr>
<td>Hypertrophic*</td>
<td>196 (42)</td>
</tr>
<tr>
<td>Dilated</td>
<td>239 (51)</td>
</tr>
<tr>
<td>Other†</td>
<td>15 (3)</td>
</tr>
<tr>
<td>Unspecified</td>
<td>17 (4)</td>
</tr>
</tbody>
</table>

* Twelve of the 196 cases of hypertrophic cardiomyopathy were the mixed type. Hypertrophic cardiomyopathy was defined by the presence of at least one of the following criteria: a left ventricular wall that was more than 2 SD above the normal thickness, an echocardiographic pattern consistent with the presence of hypertrophic cardiomyopathy, or a pathological diagnosis of hypertrophic cardiomyopathy.

† “Other” includes restrictive and other identified types of cardiomyopathy.
RESULTS

Table 1 shows the base-line characteristics of the 467 children with cardiomyopathy who were enrolled between 1996 and 1999 in New England (40 percent) and the Central Southwest (60 percent) of the United States. Sixty percent were boys, and 58 percent were white. Almost half (41 percent) received a diagnosis of cardiomyopathy within the first 12 months of life. Dilated cardiomyopathy was the most common type, accounting for 51 percent of cases.

The overall annual incidence of cardiomyopathy (Table 2) was 1.13 cases per 100,000 children (95 percent confidence interval, 1.03 to 1.23 cases per 100,000). The incidence of cardiomyopathy was significantly higher in New England than in the Central Southwest (1.44 vs. 0.98 cases per 100,000, \( P<0.001 \)). The directly standardized rates adjusted for sex, age, and racial or ethnic group according to the U.S. population younger than 18 years of age were 1.59 per 100,000 for New England and 0.84 per 100,000 for the Central Southwest (\( P<0.001 \)). There was no significant difference in incidence among the years. Boys were more likely to receive a diagnosis of cardiomyopathy than girls, and the regional difference was more marked in boys than in girls. The incidence varied according to age, with children younger than one year of age nearly 12 times as likely to receive a diagnosis of cardiomyopathy as children who were one year of age or older (8.34 vs. 0.70 cases per 100,000). Racial or ethnic group was also significantly associated with the incidence of cardiomyopathy, with white children having the lowest incidence.

The incidence of cardiomyopathy differed depending on the type: dilated cardiomyopathy was the most common, hypertrophic cardiomyopathy was the second most common, and other types of cardiomyopathy, including restrictive and arrhythmic, were rarer (Table 1). There were no significant interactions between any of these subgroups and geographic region.

Table 3 presents incidence rates for all two-way combinations of subgroups. There was only one significant interaction, and that was between the age at diagnosis and sex (\( P=0.004 \)). There was no signifi-
incant sex-based difference in the incidence among infants, but among children who received a diagnosis at or after the age of one year, cardiomyopathy was more common in boys.

Sixty-eight percent of the 435 cases of hypertrophic and dilated cardiomyopathy were idiopathic. Of the remaining 138 cases (Fig. 1 and Table 4), familial isolated cardiomyopathy (21 of 61 cases [34 percent]) and inborn errors of metabolism (16 of 61 cases [26 percent]) were the most common causes of hypertrophic cardiomyopathy. Neuromuscular disorders (30 of 77 cases [39 percent]) and myocarditis (21 of 77 cases [27 percent]) were the most common causes of dilated cardiomyopathy.

The median age at diagnosis was 5.9 years for hypertrophic cardiomyopathy and 1.8 years for dilated cardiomyopathy. Within one month after diagnosis, 58 percent of all children were given therapy for congestive heart failure, with higher usage among children with dilated cardiomyopathy than among those with hypertrophic cardiomyopathy (83 percent vs. 28 percent). The mortality rate two years after diagnosis was similar in the two groups: 12.7 percent in the group with hypertrophic cardiomyopathy and 13.6 percent in the group with dilated cardiomyopathy. The respective rates of heart transplantation two years after diagnosis were 0.8 percent and 12.7 percent.

We determined the incidence of pediatric cardiomyopathy in two regions of the United States and found that the incidence was higher in New England than...
in the Central Southwest, among boys than among girls, and among black and Hispanic children than among white children. Cardiomyopathy was much more likely to occur during the first year of life than at other ages during childhood. Dilated cardiomyopathy was more common than hypertrophic or restrictive cardiomyopathy. There was no significant difference in the frequency of the diagnosis according to the year.

Our prospective incidence rates are higher than those reported in the retrospective Finnish study, but the Finnish rates were based on idiopathic cases. A preliminary review of the retrospective cohort in the Pediatric Cardiomyopathy Registry showed that 69 percent of the patients had idiopathic cardiomyopathy and 31 percent had a known cause of cardiomyopathy, suggesting that because the Finnish data were restricted to children with idiopathic cardiomyopathy, the overall annual incidence of cardiomyopathy may have been underestimated by 45 percent. Among adults, the incidence of dilated cardiomyopathy is higher than the incidence we found (2.4 to 8.0 per 100,000, as compared with 0.58 per 100,000), and half of adults with dilated cardiomyopathy have heart failure from ischemia.

We found regional differences in the incidence of cardiomyopathy, even after adjustment for age, racial or ethnic group, and sex. We have no explanation for these differences in incidence. In the two regions studied, the rates tended to decrease from 1996 to 1999, but our prospective data indicate that the incidence was higher in 2000 (127 cases) than it was in 1998 (114 cases) and 1999 (100 cases), negating the downward trend over time.

The differences in incidence between boys and girls in our study, primarily among children who received a diagnosis after infancy, and the significant interaction between age and sex, were consistent with the male predominance and age-related expression of X-linked cardiomyopathies related to neuromuscular diseases. Sex-related differences in children with other types of heart disease have been
We found higher rates of cardiomyopathy in black and Hispanic children than in white children. Studies of dilated cardiomyopathy in adults have also found higher rates in blacks than in whites but were unable to determine whether the differences were due to genetic or environmental factors.26 Racial and ethnic differences have been found in the incidence, types, and outcomes of congenital cardiovascular malformations among children.27,28

We found that the incidence of cardiomyopathy was significantly higher in the first year of life than at older ages. Our incidence of 8.34 cases of cardiomyopathy per 100,000 children during the first year of life is very similar to the six-year prevalence of 10 per 100,000 estimated in the Baltimore–Washington Infant Study.29 The latter study assessed the prevalence of cardiomyopathy, which would be higher than the incidence, but given the narrow age range, this would not have much of an effect. The Baltimore–Washington study also did not exclude secondary cardiomyopathies, which accounted for at least 13 percent of the cases in that study and which were excluded from our data bases. The exclusion of these cases would result in a rate of 8.7 per 100,000, which is quite similar to ours.29 Postnatal abnormalities of the structure and function of the left ventricle can be related to a less healthy intrauterine milieu,30 and myocardial injury is frequently found in the perinatal period.31 Previous studies have suggested that children with dilated cardiomyopathy present in the first two years of life,20–22,32 with half of the cases seen in the first year.20

Although the incidence of cardiomyopathy peaks in the first year of life, a second, smaller peak occurs during adolescence and is related to hypertrophic cardiomyopathy and neuromuscular diseases. The high incidence of hypertrophic cardiomyopathy in the first year of life has not been reported in many previous studies that have focused on older children.33–36 As a result, several pediatric cardiology textbooks state that clinical manifestations of hypertrophic cardiomyopathy usually do not develop before adolescence and are rarely seen during infancy and childhood.25,34 Our findings may reflect a change in the practice of family care, with children in affected families being screened earlier. For many children with hypertrophic cardiomyopathy, the young age at diagnosis is due to genetic and metabolic disorders that have not been adequately addressed or recognized in the past.2,37,38 Our data support the assumption that dilated cardiomyopathy develops more commonly in younger children than in older children.

In 1993, the Pediatric Cardiomyopathy Registry surveyed pediatric cardiology centers in North America for cases of cardiomyopathy.1 The results showed an estimated incidence of pediatric cardiomyopathy that was nearly 10 times the rate reported in our prospective study. Moreover, the survey suggested that the incidence of cardiomyopathy was constant across pediatric age ranges, whereas we found that the incidence was nearly 12 times as high.


<table>
<thead>
<tr>
<th>Cause</th>
<th>Hypertrophic Cardiomyopathy (N=61)†</th>
<th>Dilated Cardiomyopathy (N=77)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. of patients (%)</td>
<td></td>
</tr>
<tr>
<td>Inborn error of metabolism</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>Disorder of glycogen metabolism</td>
<td>5 (31)</td>
<td>1 (20)</td>
</tr>
<tr>
<td>Disorder of mucopolysaccharide metabolism</td>
<td>4 (25)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>Disorder of oxidative phosphorylation</td>
<td>5 (31)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>Disorder of fatty-acid metabolism</td>
<td>2 (12)</td>
<td>0</td>
</tr>
<tr>
<td>Malformation syndrome associated with cardiomyopathy</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>Autosomal dominant‡</td>
<td>12 (92)</td>
<td>0</td>
</tr>
<tr>
<td>Autosomal recessive</td>
<td>0</td>
<td>2 (67)</td>
</tr>
<tr>
<td>Chromosomal defect</td>
<td>1 (8)</td>
<td>1 (33)</td>
</tr>
<tr>
<td>Neuromuscular disorder associated with cardiomyopathy</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>Muscular dystrophies§</td>
<td>1 (10)</td>
<td>30 (100)</td>
</tr>
<tr>
<td>Congenital myopathy</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ataxia¶</td>
<td>9 (90)</td>
<td>0</td>
</tr>
<tr>
<td>Myocardiitis</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>Confirmed by Dallas criteria on biopsy</td>
<td>0</td>
<td>9 (43)</td>
</tr>
<tr>
<td>Probable</td>
<td>0</td>
<td>12 (57)</td>
</tr>
<tr>
<td>Familial isolated cardiomyopathy</td>
<td>21</td>
<td>15</td>
</tr>
<tr>
<td>Autosomal dominant</td>
<td>19 (90)</td>
<td>12 (80)</td>
</tr>
<tr>
<td>X-linked</td>
<td>0</td>
<td>1 (7)</td>
</tr>
<tr>
<td>Autosomal recessive</td>
<td>2 (10)</td>
<td>2 (13)</td>
</tr>
<tr>
<td>Data missing or Unspecified</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

† Five children with an “other” or unknown type of cardiomyopathy but a known cause are not included. Because of rounding, percentages may not total 100.‡ There were 11 cases of Noonan’s syndrome and 1 case of the Beckwith–Wiedemann syndrome.§ There were 29 cases of Duchenne’s muscular dystrophy and 2 cases of Becker’s muscular dystrophy.¶ There were nine cases of Friedreich’s ataxia.
high before one year of age as it was at or after this age. These data indicate that expert clinical impres-
sions are not an adequate substitute for a prospec-
tive incidence study.

The results of the independently conducted Aus-
tralian study of pediatric cardiomyopathy support
our findings. The fact that the findings are similar
in geographically distinct regions suggests that
genetic factors are important contributors to the
development of pediatric cardiomyopathy.

Our study is, by design, limited to cases of pedi-
atrie cardiomyopathy evaluated by pediatric cardi-
ologists. Occult disease was not included. In ad-
dition, it is possible and indeed probable that some
eligible children were not included in our registry.
Because various mechanisms could have led to an
undercount, we tried, through our survey of adult
cardiologists and our review of ICD-9 codes and
echocardiographic findings, to ascertain the extent
of the problem and found no new cases. Nonethe-
less, the number of missed cases may be biased rel-
ative to region or age, and such a bias may have
contributed to the significant differences in the in-
cidence of cardiomyopathy that we found.

In summary, we prospectively estimated that the
incidence of pediatric cardiomyopathy was 1.13 cas-
es per 100,000 children. Most cases are identified
at an early age, and the incidence appears to vary ac-
cording to region, sex, and racial or ethnic origin.

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APPENDIX

The following persons and institutions were involved in the Pediatric Cardiomyopathy Registry Prospective Study (asterisks denote prin-
ealy, L. Gilroy, F. Tighe, P. Nash, N. Pophali, L. Schiavoni, S. Osganian, L. Cuniberti, T. McKee, E. Rauch; Brigham and Women’s Hospital — S. Col-
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