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Cardiac Structure Growth Pattern Determined by Echocardiography

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SUMMARY Using M-mode echocardiography, we measured dimensions of the ventricular walls and cavities, great vessels, and left atrium and atroventricular valve excursions on 93 infants and children without heart disease. The data were analyzed by relating each dimension in mm to body surface area in m² and the 90%

M-MODE ECHOCARDIOGRAPHY is useful in evaluating the child with heart disease. It allows the anatomic relationships of chambers, great vessels, and valves to be assessed, and allows wall thickness, cavity dimensions, great vessel diameters, and atroventricular valve excursions to be measured. These measurements, when compared to normal data, can be used quantitatively to make judgments about normality. Several studies already provide such normal values for neonates¹ ² and adults,³ ⁴ but there is only one study providing "normal" values of echocardiographic dimensions in the growing child with respect to body surface area;² this study has therefore become the standard.³ ⁴ ⁷ The range of suggested normal limits in that study is narrow, and many of the children without significant heart disease whom we examined had echocardiographic measurements outside of these previously established limits. We therefore reviewed the echocardiographic records of 93 children and adolescents without heart disease examined at the University of California during the past two years in order to re-evaluate the limits of normality.

tolerance limits for the data were calculated. The tolerance lines of the data were wider than previously recorded. At birth and maturity they were similar to the range defined as normal by studies in neonates and adults. We suggest that the tolerance lines of these normal data may be used for quantitative echocardiography in childhood.

Method

The echocardiograms of 93 children and adolescents, age one day to 18 years, were used in this study. These subjects were thought by two pediatric cardiologists to be free of any significant heart disease. Most were outpatients referred for evaluation of a heart murmur which was found to be innocent on clinical, electrocardiographic, and radiologic grounds. The others, especially newborns, were inpatients in whom cardiac evaluation and follow-up failed to detect any heart disease.

The patients were examined in the supine position with slight left shoulder recumbency. The tracings were obtained with commercial M-mode echocardiographs and strip chart recorders. Appropriate transducers (2.25, 3.5, 5, and 7.5 MHz) were used to define cardiac structures. The echocardiograms were obtained from standard precordial positions.⁶ ⁸ ⁹

Right ventricular anterior wall thickness (RVAVD), left ventricular posterior wall thickness (LVPWD), right and left ventricular cavity diastolic dimensions (RVDD and LVEDD), interventricular septal thickness (SEPT D), and mitral valve excursion (MVDE) were measured at the level of the posterior mitral leaftlet at end-diastole, defined by the peak of the R wave on the ECG (fig. 1a and b). Left ventricular end-systolic dimension was measured at the peak upward motion of the posterior left ventricular endocardium. The right ventricular anterior wall thickness was obtained by

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proper selection of transducer and careful anterior gain control. The right ventricular-septal and left ventricular-septal interfaces were defined by proper damping and reject control. The anterior mitral leaflet excursion was measured from the D point vertically to the E point at maximal excursion. The left atrium and aorta were measured on a continuous sweep from the left ventricle (fig. 1a). Left atrial systolic dimension (LAS) was measured at the largest distance between the anterior aspect of the left atrial posterior wall and the inner aortic posterior wall. Aortic end-diastolic diameter (AOD) was measured at the beginning of the QRS complex and end-systolic diameter (AOS) at the same point used for measuring the left atrium (fig. 1a). We measured the aorta from the anterior surface of the anterior root echo to the anterior surface of the posterior root echo.

The pulmonary valve echo was identified (fig. 2). The pulmonary artery diameter (PAD) was measured at the onset of the QRS complex, whenever possible, or when the anterior echo moved parallel to the posterior pulmonary root at any point in the cardiac cycle. By positioning the transducer medially and inferiorly to the aortic region, the tricuspid valve echo was recorded (fig. 3), and the maximum excursion of the anterior leaflet (TVDE) was measured the same way as for the mitral valve.

Three clear complexes were chosen for measurements. These were measured independently by two observers and the average value to the nearest millimeter was entered. If a structure was not clearly defined, it was not measured.

The subject's weight and height were recorded at the time of the echocardiogram and the body surface area (BSA) was derived from these measurements (West's nomogram). For each variable, data points were analyzed with respect to BSA. Preliminary plots of the data indicated that the regression functions on BSA could be adequately described as first or second degree polynomial functions of BSA. Higher order polynomial functions were not significantly different. In addition, it was noted that the variation around the regression line was an increasing function of BSA. A weighted regression model is appropriate for this type of data. For each variable, both linear and quadratic weighted regressions were calculated. These calculations were used to test the null hypothesis that the regression on BSA was linear rather than quadratic. If the null hypothesis was accepted at $\alpha = 0.05$, linear weighted regression was used in subsequent analysis. If the null hypothesis was rejected at $\alpha = 0.05$, weighted quadratic regression was used in subsequent analysis. Tolerance limits weighted for BSA to include $90\%$ of the population at confidence coefficient 0.90 were created for each of the regression functions following the method of Miller.

**Results**

The results are presented in graphic form for clarity (fig. 4). The data points for each subject are shown as dots. The regression line and the weighted tolerance limits are shown as heavy continuous lines. The multiple squared correlation coefficients ($r^2$) of the data and the estimate of the population standard deviations (s) for each variable related to BSA are shown in table 1.
Discussion

The subjects used in this study were from a cardiology practice and were noted to have a heart murmur that was considered to be innocent by at least two pediatric cardiologists. The ECG and chest X-rays showed no evidence of abnormality. The echocardiograms were, of course, qualitatively normal in all subjects and in some even two-dimensional ultrasonic studies showed no abnormalities. The echocardiographic studies were not used to define entrance into the study. The population, therefore, was highly screened and considered normal. It is possible that minor abnormalities which were beyond clinical detection might have yielded larger tolerance limits than in an ideal normal population.

Others have suggested that the best correlation was obtained between echocardiographic dimensions and height (personal communication: R.A. Meyer) or the cube root of the weight.\(^1^4\) We tested these correlations in our data and found that the correlation coefficients for all these variables were very similar. In fact, each of the variables (height, weight, BSA, and cube root of weight) were so strongly correlated with one another (\(r > 0.90\)) that regressions using any one of the four as independent variables were equivalent for all practical purposes. We therefore chose to relate our data to body surface area as it is already often used to express hemodynamic data in children. However, it may be more appropriate to correlate echocardiographic dimensions with weight in the smallest individuals as data on left ventricular and left atrial dimensions in premature infants would suggest;\(^1^5\) for example, when weight increases from 2 kg to 4 kg, body surface area changes minimally. It may be advisable, therefore, to examine a larger group of children at the lower end of the growth curve to reassess this correlation.

We believe that only small differences would have been present if measurements had been taken at other points used to indicate end-systole and end-diastole. Right ventricular wall thickness measurements are similar to those made at

<table>
<thead>
<tr>
<th>Variable</th>
<th>(s)</th>
<th>(r^2)</th>
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<tbody>
<tr>
<td>RVAWD</td>
<td>0.48</td>
<td>0.96</td>
</tr>
<tr>
<td>RVDD</td>
<td>3.07</td>
<td>0.91</td>
</tr>
<tr>
<td>SEPT D</td>
<td>0.81</td>
<td>0.95</td>
</tr>
<tr>
<td>LVESD</td>
<td>2.16</td>
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<tr>
<td>LVEDD</td>
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<td>0.98</td>
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<tr>
<td>LVPWD</td>
<td>0.81</td>
<td>0.95</td>
</tr>
<tr>
<td>AOD</td>
<td>1.8</td>
<td>0.98</td>
</tr>
<tr>
<td>AOS</td>
<td>1.72</td>
<td>0.98</td>
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<tr>
<td>LAS</td>
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</tr>
<tr>
<td>PAS</td>
<td>3.37</td>
<td>0.95</td>
</tr>
<tr>
<td>MVDE</td>
<td>1.95</td>
<td>0.97</td>
</tr>
<tr>
<td>TVDE</td>
<td>2.15</td>
<td>0.96</td>
</tr>
</tbody>
</table>
Figure 4. Echocardiographic dimensions in mm are plotted against the body surface area (BSA) in square meters (m²) in 93 patients without heart disease. Data points for each variable are shown by dots. The 90% tolerance lines are shown by heavy continuous lines. RVAWD — right ventricular anterior wall thickness in diastole, RVDD — right ventricular end-diastolic dimension, SEPT D — septal thickness at end-diastole, LVESD — left ventricular end-systolic dimension, LVEDD = left ventricular end-diastolic dimension, LVPWD — left ventricular posterior wall thickness at end-diastole, AOS — aortic diameter in systole, AOD — aortic diameter in diastole, PAD — pulmonary artery diameter, LAS — left atrial dimension in systole, MVDE — anterior mitral valve leaflet excursion, TVDE — anterior tricuspid valve leaflet excursion.
postmortem examination, although slightly smaller. The patients were examined with slight left shoulder recumbency which may yield larger right ventricular dimensions than in the supine position. Pulmonary artery diameter was not always measured at end diastole because of the difficulty in defining the anterior pulmonary root wall. It was often measured wherever the anterior pulmonary root echo was found to move parallel to the posterior pulmonary root and
this may account for the wider scatter of the pulmonary root diameter than the aortic root diameter. The aortic diameter was measured both at end systole and at end diastole because there has not been a clear directive as to the appropriate time to measure it. There is a small increase in aortic root diameter in systole. This may account for some of the variability in normal left atrial to aortic root ratios.6, 17

To check the validity of our tolerance limits, we compared our data with two other studies. The mean value and two standard deviations on each side (point and bars between stippled lines) are drawn on figure 5 for a normal newborn1 and a normal adult population4 (personal communication: RL Popp, R Valdez). This latter measurement was divided by surface area to obtain a corrected value at one square meter of BSA. The range of normal given for newborn and for adults is in general agreement with our data. Only one study8 has previously correlated echocardiographic measurements and BSA in children and the 5th and 95th percentiles (broken growth curve lines on fig. 5) in that study are much narrower than our tolerance limits. Epstein et al.5 measured end-diastolic dimensions at the onset of the QRS while Solinger et al.,1 Valdez et al.,4 and our group measured end-diastolic dimensions at the peak of the QRS. This could account for the minor differences observed between our regression lines and their mean values5 but should only minimally influence the spread around the mean. The explanation for the discrepancy between these two studies has already been alluded to by us and it now appears that these percentile limits are in fact the standard error of the mean.18 Those data, therefore, can be used to compare population means but not to predict whether a particular patient has normal or abnormal echocardiographic measurements.

Attempts have been made to correlate cardiac size by normalizing measurements per square meter of body surface area. This has been done for adults.5, 4 (Valdez’s data normalized for square meter are shown in figure 4.) Our tolerance lines coincide with this corrected range of normal when the function is linear, but not if the function is nonlinear. This correction for body surface seems, therefore, to be inappropriate for variables that are curvilinear functions of BSA.

The most important reason to establish tolerance limits for echocardiographic dimensions in normal children is to identify quantitative abnormalities in children with heart disease. We consider that our data can be used for quantitative echocardiography in childhood, although the study of a larger normal population would, perhaps, yield slightly narrower tolerance limits.

Acknowledgment

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References

Cardiac structure growth pattern determined by echocardiography.
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