Evaluation of Left Ventricular Size and Function by Echocardiography

Results in Normal Children

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DESMOND F. DUFF, M.D., AND DAN G. McNAMARA, M.D.

With the Technical Assistance of Linda Kaufman

SUMMARY Left ventricular (LV) size and function were studied by echocardiography in 145 normal children. The LV end-diastolic diameter (EDD) and its percentage change with systole (%ΔLVD) were measured and mean velocity of circumferential fiber shortening (Vcf) calculated. The LV pre-ejection period (PEP) and ejection time (LVET) were determined from recordings of aortic valve motion.

The EDD increased by approximately threefold during childhood and was best correlated with the log of body weight (r = 0.95) and the log of body surface area (r = 0.96). The mean %ΔLVD was 36 ± 4 (SD), and this index of LV function was independent of age and heart rate. Mean Vcf was higher, and the absolute values of PEP and LVET shorter, in younger children with a faster heart rate. The mean ratio of PEP/LVET was 0.31 ± 0.003, and was relatively independent of age (r = -0.41) and heart rate (r = 0.37). The %ΔLVD and PEP/LVET appear to be particularly useful indices of LV function because they remain constant during the course of childhood.

ONE OF THE MOST PROMISING APPLICATIONS of echocardiography is the noninvasive evaluation of left ventricular (LV) function. A variety of echographic indices of LV performance have been described, based largely on studies of adults. The demonstration that systolic time intervals can be measured by echocardiography adds another index of LV function to the conventional echocardiographic measurements.

In this study, we have used echocardiography to study left ventricular size and function in a group of normal children with a wide range of age and heart rate. We have attempted to determine which of the indices of LV function change during the course of childhood and where changes occur, to determine if they are related to age, heart rate, or a combination of these variables.

Materials and Methods

Echocardiograms were obtained from 145 normal children, 80 boys and 65 girls. The ages of the subjects ranged from one day to 19 years. None had heart disease as judged by history and physical examination. Their age distribution was as follows: 1 day to 30 days, N = 30; 1 month to 24 months, N = 27; 2 to 6 years, N = 37; 7 to 11 years, N = 26; 12 to 19 years, N = 25.

The echocardiograms were obtained with a Hoffrel Model 101 Ultrasonoscope interfaced to a Honeywell Model 1856 strip chart recorder. Transducers of 2.25, 3.5 or 5.0 MHz were used, depending on the subject’s size. Studies were obtained from the third or fourth intercostal space at the left parasternal edge with the subject in the supine position. Occasionally a shallow left lateral decubitus position was required to record the ventricular septum clearly.

Left ventricular dimensions were measured in the standard manner (fig. 1). End-diastolic diameter (EDD) was measured at the start of the QRS complex. End-systolic diameter (ESD) was measured at the point in late systole where the septum and LV posterior wall were in closest apposition. These measurements were made with the transducer angled slightly inferiorly and laterally from the point of maximal excursion of the mitral valve in subjects over one year of age. In younger children, as previously noted by Sahn et al., the mitral leaflets appear to extend farther toward the apex, and the LV diameter decreases rapidly as the transducer is directed below the mitral valve. Therefore, in infants under one year of age the LV dimensions were measured at the point of maximal excursion of the mitral valve, from a position from which both leaflets could be visualized. Recordings satisfactory for determination of LV dimensions were obtained in 143 of the 145 subjects (99%).

Left ventricular systolic time intervals were determined from recordings of the aortic valve at 100 mm/sec paper speed (fig. 2). The pre-ejection period (PEP) was measured from the onset of ventricular depolarization in lead II of the electrocardiogram to the onset of opening of the aortic valve. The left ventricular ejection time (LVET) was measured from aortic valve opening to closing. An average was obtained from three cardiac cycles and the value was rounded off to the nearest five milliseconds. Recordings satisfactory for measuring systolic time intervals were obtained in 118 of the 145 subjects (81%).

During the first half of this study, 0.5 sec time lines generated by the echograph were employed. For the remainder of the study, 0.02 sec time lines from a quartz crystal oscillator were available. The time lines of the echograph differed from those produced by the oscillator by less than 3%. We considered this error insignificant and the data from the two phases of the study were combined.

From these measurements, the following calculations were made.

From the Section of Cardiology, Department of Pediatrics, Baylor College of Medicine, and Texas Children’s Hospital, Houston, Texas. Supported in part by Grant HL-5756 from the USPHS, Grant RR-00188 from the General Clinical Research Branch, NIH, and by a grant from the Texas Affiliate, American Heart Association.

Address for reprints: Howard P. Gutgesell, M.D., Section of Pediatric Cardiology, Texas Children’s Hospital, 6621 Fannin, Houston, Texas 77030.

Received February 3, 1977; revision accepted April 29, 1977.
1) The percentage change in left ventricular diameter with systole (%ΔLVD):

\[
\frac{EDD - ESD}{EDD} \times 100
\]

2) The ratio of the pre-ejection period to ejection time:

\[
\frac{PEP}{LVET}
\]

3) Mean velocity of circumferential fiber shortening (Vcf):

\[
\frac{EDD - ESD}{EDD \times LVET}
\]

In the latter calculation, the value for LVET was that obtained from the recording of aortic valve motion.

The LV diameter was compared to body size by linear regression analysis. Correlation coefficients were determined for the relationship of EDD to body weight, height, and surface area (BSA), as well as the square root, cube root, and log of each of these variables.

To relate %ΔLVD, systolic time intervals, and Vcf to age and heart rate, the following types of linear regression analysis were performed: 1) simple, 2) multiple, two variable, and 3) partial, with one variable constant.

**Results**

**Left Ventricular Diameter**

Left ventricular EDD increased by approximately threefold as body weight increased from 3 to 60 kg. The correlation coefficients and regression equations relating EDD to body size are shown in table 1. The EDD was best correlated with the log of BSA (r = 0.96, SEE = 2.78) although the correlations with the log of body weight and height as well as with the root functions of height and BSA, were nearly as strong. The linear relationship between EDD and the log of body weight is shown in figure 3.

%ΔLVD

Left ventricular diameter decreased by slightly over one-third with systole. The mean %ΔLVD was 36 ± 4%. The
TABLE 1. Relationship of Left Ventricular End-diastolic Diameter to Body Size in Normal Children

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Correlation coefficient</th>
<th>Regression equation</th>
<th>Standard error (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wt (kg)</td>
<td>0.85</td>
<td>EDD = 21.73 + 0.44 (wt)</td>
<td>5.2</td>
</tr>
<tr>
<td>√Wt</td>
<td>0.92</td>
<td>EDD = 21.73 + 0.44 (√Wt)</td>
<td>3.9</td>
</tr>
<tr>
<td>³√Wt</td>
<td>0.93</td>
<td>EDD = 21.73 + 0.44 (³√Wt)</td>
<td>3.5</td>
</tr>
<tr>
<td>Log wt*</td>
<td>0.95</td>
<td>EDD = 6.94 + 9.22 (log wt)</td>
<td>3.2</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>0.92</td>
<td>EDD = 8.56 + 0.22 (Ht)</td>
<td>3.95</td>
</tr>
<tr>
<td>√Ht</td>
<td>0.93</td>
<td>EDD = −13.26 + 4.44 (√Ht)</td>
<td>3.60</td>
</tr>
<tr>
<td>³√Ht</td>
<td>0.93</td>
<td>EDD = −35.10 + 14.35 (³√Ht)</td>
<td>3.52</td>
</tr>
<tr>
<td>Log ht*</td>
<td>0.94</td>
<td>EDD = −08.65 + 21.86 (log Ht)</td>
<td>3.42</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>0.92</td>
<td>EDD = 17.41 + 17.92 (BSA)</td>
<td>3.98</td>
</tr>
<tr>
<td>√BSA</td>
<td>0.95</td>
<td>EDD = 4.49 + 32.26 (√BSA)</td>
<td>3.15</td>
</tr>
<tr>
<td>³√BSA</td>
<td>0.95</td>
<td>EDD = −8.36 + 45.51 (³√BSA)</td>
<td>2.96</td>
</tr>
<tr>
<td>Log BSA*</td>
<td>0.96</td>
<td>EDD = 37.75 + 12.88 (log BSA)</td>
<td>2.78</td>
</tr>
</tbody>
</table>

*Correlation coefficient statistically significant, *P < 0.05.

Abbreviations: Wt = weight; Ht = height; BSA = body surface area.

%ΔLVD was greater than 44% in only seven subjects and was less than 28% in two.

There was no correlation between %ΔLVD and age or heart rate (table 2). The constancy of %ΔLVD despite wide variations in heart rate is illustrated in figure 4.

Systolic Time Intervals

As expected, there was an inverse correlation between age and heart rate (r = −0.80). The correlation coefficients and regression equations relating the systolic time intervals to age and heart rate are shown in tables 2 and 3. As illustrated in figures 5A and 5B, both PEP and LVET were inversely related to heart rate (r = −0.70 and −0.88, respectively). However, the correlations of PEP and LVET with age were nearly as strong (r = 0.61 and 0.81). Thus, although we have only illustrated the relationship of the systolic time intervals to heart rate, similar graphs with the opposite slope could be constructed with age as the independent variable. Very little increase in correlation was achieved by use of multiple regression analysis, simultaneously taking both age and heart rate into consideration (table 2). For example, the correlation coefficient between LVET and heart rate was −0.88; that relating LVET to heart rate and age was −0.89.

Partial regression analysis with age or heart rate held constant showed that both PEP and LVET had a positive correlation with age and a negative correlation with heart rate (table 2). However, with the exception of the partial correlation coefficient relating LVET to heart rate with age held constant (r = −0.67), the partial correlation coefficients were all less than 0.50; those relating heart rate to PEP and LVET were higher than those for age.

In contrast to PEP and LVET, the ratio PEP/LVET was relatively independent of age and heart rate. The mean PEP/LVET was 0.31 ± 0.03. Since the slope of the regression line of heart rate versus LVET was steeper than that for PEP, the ratio PEP/LVET was slightly higher for subjects with faster heart rates (fig. 5C). The correlation coefficients between PEP/LVET and heart rate (r = 0.37) and age (r = −0.41) were weak and in practice can probably be disregarded.

Mean Vcf

Mean Vcf, normalized for EDD, was higher in the younger children, who also had faster heart rate (tables 2 and 3). As in the case of PEP and LVET, there was an association between Vcf and both heart rate (r = 0.67) and age (r = −0.65).

The relationship of Vcf to heart rate and age simply reflects the formula from which Vcf is calculated:

\[
Vcf = \frac{\text{EDD} - \text{ESD}}{\text{EDD} \times \text{LVET}}
\]

The enclosed measurements represent the %ΔLVD which was independent of heart rate and age (table 2 and figure 4). Since LVET is directly related to age and inversely related to heart rate, younger subjects, with a faster heart rate, had a shorter ejection time and thus a higher value of Vcf.

Discussion

We have combined two commonly used noninvasive techniques to obtain a profile of left ventricular function in nor-
TABLE 3. Regression Equations Relating Selected Indices* of Left Ventricular Function to Age and Heart Rate

<table>
<thead>
<tr>
<th>Heart rate (beats/min)</th>
<th>Age (yr)</th>
<th>Heart rate and age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEP</td>
<td>y = 0.104 - 0.0003 (HR)</td>
<td>y = 0.066 + 0.001 (age)</td>
</tr>
<tr>
<td></td>
<td>SEE = 0.008 sec</td>
<td>SEE = 0.01 sec</td>
</tr>
<tr>
<td>LVET</td>
<td>y = 0.389 - 0.001 (HR)</td>
<td>y = 0.206 + 0.007 (age)</td>
</tr>
<tr>
<td></td>
<td>SEE = 0.02</td>
<td>SEE = 0.03 sec</td>
</tr>
<tr>
<td>Vcf</td>
<td>y = 0.648 + 0.008 (HR)</td>
<td>y = 1.740 - 0.042 (age)</td>
</tr>
<tr>
<td></td>
<td>SEE = 0.27 circ/sec</td>
<td>SEE = 0.28 circ/sec</td>
</tr>
</tbody>
</table>

*Includes those relationships with a correlation coefficient greater than 0.5 (table 2).

Table: Data on left ventricular function to age and heart rate

The table above shows regression equations relating selected indices of left ventricular function to age and heart rate. The equations are:

- For PEP: \( y = 0.104 - 0.0003 \text{ HR} \) with standard error (SEE) of 0.008 sec.
- For LVET: \( y = 0.389 - 0.001 \text{ HR} \) with SEE of 0.02.
- For Vcf: \( y = 0.648 + 0.008 \text{ HR} \) with SEE of 0.27 circ/sec.

These equations allow for assessment of left ventricular function in children, with the understanding that the %ALVD remains constant within a wide range of heart rates.

**Figure 4.** Plot of percent change in left ventricular diameter (%\( \Delta \text{LVD} \)) against heart rate. The %\( \Delta \text{LVD} \) remains constant within a wide range of heart rates.

**Table 3.** Regression Equations Relating Selected Indices* of Left Ventricular Function to Age and Heart Rate

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failure, an elevated ratio has been used widely as an indicator of myocardial dysfunction. Mean Vcf has been advocated as a valuable index of LV function in both children and adults. By adding an element of time to the measurement of change in left ventricular dimensions, Vcf has been reported to offer better separation of normals from patients with abnormal left ventricular function than ejection fraction alone. However, in two recent studies the percent of minor axis shortening (%ALVD) was as sensitive as Vcf in detecting depressed LV function. Although the present study does not settle the issue it does emphasize the fact that Vcf, unlike %ALVD, is directly related to heart rate and inversely related to age. Caution is therefore required in using Vcf to compare subjects of different ages or heart rates, or the same subjects before and after interventions which may alter heart rate.

The use of the echocardiogram to determine both ventricular dimensions and LV systolic time intervals has several advantages. Both determinations can be made fairly rapidly by one person with the same piece of equipment. If one of the measurements cannot be made reliably (for example, %ALVD in a subject with paradoxical septal motion), it still may be possible to make some estimation of left ventricular function from the other parameter. The %ALVD and the ratio of PEP/LVET appear to be particularly useful indices of LV function in children because they remain relatively constant during childhood.

Acknowledgment

We are indebted to Dr. Howard Thompson for assistance in the statistical analysis of the data. We would like to thank Sue Lambert for editing and typing the manuscript.

References

Human Ventricular Refractoriness

II. Effects of Procainamide

JOHN A. KASTOR, M.D., MARK E. JOSEPHSON, M.D.,
STEPHEN B. GUSS, M.D., AND LEONARD N. HOROWITZ, M.D.

SUMMARY The effects of procainamide (PA) on the ventricular effective refractory period (ERP-V) and on the relationship of refractoriness to recovery of excitability were evaluated in eight patients during ventricular pacing. Measurements of ERP-V and plasma levels of PA were taken before and after intravenous administration of 500 mg of PA. The ERP-V was prolonged from a group mean value of 237 ± 7 msec (SE) to 279 ± 16 msec (P < 0.05). Peak increase occurred at maximal drug levels five minutes after administration of PA. The QT intervals increased from a group mean value of 421 ± 8 msec to 461 ± 9 msec after PA (P < 0.01). The ratio ERP-V/QT increased in seven of eight patients from 0.56 ± 0.01 to 0.62 ± 0.04 after infusion of PA (0.1 < P < 0.2). The ratio remained unchanged in one patient.

This study reveals that PA increases ERP-V in man and usually increases the ratio ERP-V/QT. Thus a longer portion of the ventricular recovery period is refractory after administration of the drug. The data, which correlate with animal studies, help to explain how PA may suppress human re-entrant ventricular arrhythmias.

PROCAINAMIDE is one of the most effective and frequently employed drugs for the treatment of ventricular arrhythmias. It appears to work by several mechanisms: 1) decreasing the slope of spontaneous phase 4 depolarization in ventricular Purkinje cells; 2) slowing and equalizing conduction and refractoriness in regions of unidirectional block or converting unidirectional to bidirectional block; 3) affecting the relationship of refractoriness to the recovery of the threshold of excitability. None of these mechanisms has been conclusively demonstrated in man.

The development of the ventricular extrastimulus method permits evaluation of ventricular refractoriness to be performed with safety and reproducibility in man. Total ventricular recovery can be estimated from the familiar QT interval of the electrocardiogram. In this study, we have evaluated the effects of procainamide on these two electrophysiological characteristics of the human ventricle in eight patients and have correlated the data with blood levels of the drug.

Patients and Methods

The effects of procainamide on human ventricular electrophysiologic function were evaluated in eight patients referred to the Clinical Cardiac Electrophysiology Laboratory (table 1). The purpose and nature of the studies were described and each patient gave informed consent. The investigative evaluations were performed after completion of electrophysiological diagnostic studies for which the patient had been referred to the laboratory. In each patient, all cardiac medicines had been discontinued at least 48 hours before study.

Electrode catheters were inserted as follows: a triaxial catheter from the femoral vein across the tricuspid valve to record the bundle of His potential, a quadripolar catheter via a femoral or antecubital vein to the high right atrium for
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