Systolic Phases of the Cardiac Cycle in Children

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SUMMARY
The duration of the systolic phases of the cardiac cycle in 390 normal children was determined from high-speed simultaneous recordings of the electrocardiogram, phonocardiogram, apexcardiogram, and carotid pulse. Data were obtained on children 1 mo to 13 years of age and analyzed to define the independent effects of aging and heart rate on the systolic intervals. Electromechanical systole (Q-II) was found to prolong with increasing age in children with the same heart rate. Pre-ejection period was also prolonged in older children and occupied an increased percentage of total systole. The interval from onset of contraction to first sound varied inversely with heart rate and was independent of age. Electromechanical delay and isometric contraction time were directly related to age and independent of rate. Ejection time varied directly with age and inversely with heart rate but occupied a smaller percentage of systole in older children. Alterations in systolic phase duration occurring with maturation reflect the normal functional adaptation of the developing heart. Measurement of these phases can provide useful information relative to cardiac function in children.

Additional Indexing Words:
Polycardiography Systolic time intervals Ejection time Phonocardiogram
Isometric contraction time Pre-ejection period Apexcardiogram
Electromechanical delay

NONINVASIVE technics have become increasingly important in the study of cardiovascular disease. With these methods precise hemodynamic data may be obtained without risk or discomfort to the patient, and studies can be repeated frequently and under various test conditions. Simultaneous external polycardiography is an atraumatic technic that permits the measurement of the various phases of cardiac systole. The duration of these phases correlates well with the pathophysicsology in many abnormal cardiac states, and measurement of the phases can provide useful clinical information relative to cardiac function.

Considerable data have been accumulated on the normal temporal correlations of the phases of systole in adults.1 Specific alterations in systolic phase duration have been shown to occur with congestive heart failure,2 thyroid disease,3 digitalis therapy,4 and hypercalcemia,5 as well as with various congenital6 and valvular defects.7 Data on the normal temporal relationships of the systolic phases in children are sparse, however, and age-rate relationships remain ill defined.8-10 It is difficult in children to separate the effects of age and heart rate on the systolic intervals because of the normal inverse relationship between these variables. Cardiovascular maturation presumably involves a series of
adaptive processes which are reflected by age-dependent alterations in systolic phase duration.

The purpose of the present study was to determine the independent effects of aging and heart rate on the systolic phases in children and to delineate the pattern of phase alteration occurring with normal cardiovascular maturation.

Methods

Three hundred and ninety children (202 male and 188 female) were studied. The subjects ranged in age from 1 mo to 13 years and were in good health and free of heart disease. Each child was normal on cardiovascular examination which included blood pressure determination (in children over 2 years), auscultation of the heart and lungs, and a standard 12-lead electrocardiogram. Studies were conducted in the morning with the children recumbent and in the postabsorptive state. Only cooperative subjects were included, and no sedation was used.

Simultaneous recordings were taken of the electrocardiogram (lead II), phonocardiogram, apexcardiogram, and carotid pulse. Tracings were recorded on a Siemens Cardirex-6 direct-writing apparatus (linear frequency response 0.07 to 500 Hz, 30% amplitude reduction at 650 Hz) utilizing the Maass-Weber filter system. Boucke-Brecht type transducers were used for the carotid pulse pickup and apexcardiogram. The phonocardiogram was recorded with an electromagnetic microphone placed over the left ventricle at a location giving the clearest reception of the first and second heart sounds. All wave forms were displayed oscillographically, and recordings were taken at paper speeds of 200 mm/sec when optimal reception was achieved. The phases were measured by hand (with the aid of magnification) in five consecutive cycles at end expiration, and the average was recorded. Only tracings with clear wave forms and inflection points were utilized. Though the usual limit of reading accuracy for these types of recordings was taken as 5 msec, smaller units were interpolated in estimating phases of extremely short duration.

Six phases of cardiac systole were studied (fig. 1). Total electromechanical systole (hereafter referred to as systole) was taken to comprise the Q-second sound interval (Q-II). The Q-first sound period (Q-I) was separated into two phases, electromechanical delay (EMD) and the interval from the onset of contraction to the first sound

![Figure 1](image)

*Simultaneous recording of the electrocardiogram, phonocardiogram, carotid pulse, and apexcardiogram demonstrating method of calculating systolic phase duration (paper speed, 200 mm/sec; time intervals, 5 msec).*

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Table 1
Mean Values for Phase Duration in Five Age Groups (30 Children in Each)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Yr/mo</th>
<th>Heart rate</th>
<th>Q-II</th>
<th>PEP</th>
<th>Q-I</th>
<th>EMD</th>
<th>C-I</th>
<th>ICT</th>
<th>ET</th>
<th>SQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>1/7</td>
<td>120</td>
<td>0.220</td>
<td>0.034</td>
<td>0.021</td>
<td>0.018</td>
<td>0.014</td>
<td>0.186</td>
<td>5.48</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1/6</td>
<td>119</td>
<td>0.220</td>
<td>0.034</td>
<td>0.021</td>
<td>0.022</td>
<td>0.018</td>
<td>0.014</td>
<td>0.186</td>
<td>5.43</td>
</tr>
<tr>
<td>F</td>
<td>5/3</td>
<td>106</td>
<td>0.256</td>
<td>0.048</td>
<td>0.031</td>
<td>0.006</td>
<td>0.026</td>
<td>0.018</td>
<td>0.207</td>
<td>4.21</td>
</tr>
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<td>M</td>
<td>5/6</td>
<td>104</td>
<td>0.257</td>
<td>0.050</td>
<td>0.031</td>
<td>0.006</td>
<td>0.026</td>
<td>0.018</td>
<td>0.207</td>
<td>4.15</td>
</tr>
<tr>
<td>F</td>
<td>6/8</td>
<td>105</td>
<td>0.265</td>
<td>0.056</td>
<td>0.036</td>
<td>0.011</td>
<td>0.025</td>
<td>0.020</td>
<td>0.209</td>
<td>3.70</td>
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<td>6/7</td>
<td>90</td>
<td>0.294</td>
<td>0.063</td>
<td>0.043</td>
<td>0.014</td>
<td>0.029</td>
<td>0.021</td>
<td>0.232</td>
<td>3.71</td>
</tr>
<tr>
<td>F</td>
<td>8/7</td>
<td>94</td>
<td>0.321</td>
<td>0.060</td>
<td>0.035</td>
<td>0.012</td>
<td>0.023</td>
<td>0.025</td>
<td>0.260</td>
<td>4.30</td>
</tr>
<tr>
<td>M</td>
<td>8/6</td>
<td>92</td>
<td>0.320</td>
<td>0.061</td>
<td>0.037</td>
<td>0.014</td>
<td>0.023</td>
<td>0.024</td>
<td>0.260</td>
<td>4.28</td>
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<tr>
<td>F</td>
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<td>84</td>
<td>0.367</td>
<td>0.093</td>
<td>0.060</td>
<td>0.029</td>
<td>0.031</td>
<td>0.033</td>
<td>0.274</td>
<td>2.97</td>
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<tr>
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<td>0.357</td>
<td>0.089</td>
<td>0.057</td>
<td>0.028</td>
<td>0.029</td>
<td>0.032</td>
<td>0.268</td>
<td>3.01</td>
</tr>
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</table>

Abbreviations: Yr/mo = mean age in years and months; heart rate = beats/min; Q-II = electromechanical systole; PEP = pre-ejection period; Q-I = transformation time; EMD = electromechanical delay; C-I = onset of contraction to 1st sound; ICT = isometric contraction; ET = ejection time; SQ = systolic quotient.

(C-I). EMD was measured from the Q wave of the electrocardiogram (lead II) to the onset of the systolic wave of the apexcardiogram (C point). Q-I was read directly from the Q wave of the ECG to the first high frequency vibrations of the first heart sound, and C-I was calculated by subtracting EMD from Q-I. Isometric contraction time (ICT) was determined by measuring the interval from the first heart sound to the onset of the upstroke of the carotid pulse wave and correcting for pulse transmission delay. Pulse delay time was taken as the interval between the second heart sound and the trough of the carotid incisura. The pre-ejection phase (PEP) was calculated by summing Q-I and ICT. Ejection time (ET) was measured from the onset of the carotid upstroke to the trough of the incisura. The systolic quotient (SQ) was calculated as the ratio of ET and PEP. The data were stored on magnetic tape and analyzed with the aid of an IBM 360-50 computer. Preliminary plots were made directly from taped data using the Cal-Comp apparatus.

Results

The mean values obtained for the phases of systole in five age groups are presented in table 1. All phases of systole were found to prolong with increasing age, and the heart rate was progressively slower in older children. Linear regression equations relating phase duration to age and heart rate are given in table 2. F values for all equations were significant at P < 0.001. No significant differences (P < 0.05) were found between males and females in the complete population for the phases studied. In the group of 6 year olds, however, systole (Q-II), PEP, and ET were significantly shorter in girls (table 1). This was accounted for by the higher mean heart rate in girls in that age group (105 vs. 90). In comparing boys and girls of similar age, the mean values obtained for the phases of systole in children of various ages with the same heart rate, and on children of the same age with various heart rates.

*California Computer Products, Inc., Anaheim, California.

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heart rates, no differences were present for any of the phases.

Systole

A direct and linear relationship was found to exist between the duration of systole (Q-II) and age for all of the children studied (fig. 2). Though a linear regression equation with acceptable fit may be written for this relationship, age and rate effects cannot be separated from these data. When systole is plotted against heart rate for 13-year-old children (fig. 3), the expected inverse relationship is seen. To determine possible independent effects of age, duration of systole was compared for all children in the study (48) who coincidently had a heart rate of 100 ± 2.5 beats/min. Systole was found to prolong with increasing age apart from any rate considerations (fig. 4). The regression equation for calculating total systole, therefore, contains two variables and reflects the independent relationship of systole to age. This type of analysis was applied to all of the phases studied.

Electromechanical Delay

EMD was found to prolong with increasing age (fig. 5) but was relatively independent of heart rate (fig. 6). The regression equation for EMD contains only one variable. When the phases are expressed as a percentage of total systole (table 3), EMD is seen to occupy an increasing percentage of total systole in older

![Figure 2](http://circ.ahajournals.org/)

*Figure 2*

*Duration of systole (Q-II) vs. age for entire population (390 children).*

![Figure 3](http://circ.ahajournals.org/)

*Figure 3*

*Duration of systole (Q-II) vs. heart rate for 13-year-old children.*

![Figure 4](http://circ.ahajournals.org/)

*Figure 4*

*Duration of systole (Q-II) vs. age for all children in the study with a heart rate of 100 ± 2.5 beats/min.*

### Table 3

*Mean Duration of Systolic Phases for Each Age Group Expressed as a Percentage of Systole (Q-II)*

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>EMD</th>
<th>C-I</th>
<th>Q-I</th>
<th>ICT</th>
<th>PEP</th>
<th>ET</th>
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<tr>
<td>&lt;1</td>
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<td>6.9</td>
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<td>1</td>
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<td>9.0</td>
<td>6.5</td>
<td>15.6</td>
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<td>3</td>
<td>0.9</td>
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<td>6.6</td>
<td>16.8</td>
<td>83.2</td>
</tr>
<tr>
<td>4</td>
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<td>9.8</td>
<td>11.9</td>
<td>6.8</td>
<td>18.7</td>
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</tr>
<tr>
<td>5</td>
<td>2.3</td>
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<tr>
<td>6</td>
<td>4.4</td>
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<td>78.7</td>
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<tr>
<td>7</td>
<td>4.6</td>
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<td>7.6</td>
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<td>9</td>
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<tr>
<td>10</td>
<td>5.1</td>
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</tr>
<tr>
<td>12</td>
<td>8.5</td>
<td>7.5</td>
<td>15.9</td>
<td>8.5</td>
<td>24.4</td>
<td>75.7</td>
</tr>
<tr>
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<td>8.4</td>
<td>16.2</td>
<td>9.0</td>
<td>25.1</td>
<td>75.9</td>
</tr>
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</table>

Abbreviations: Same as in table 1.
CARDIAC CYCLE IN CHILDREN

Electromechanical delay vs. age for children with a heart rate of 100 ± 2.5 beats/min.

Electromechanical delay vs. heart rate for 8-year-old children.

children. EMD was not measurable in children under 1 year with our technics and is less than 5 msec through the first 4 years of life. Approximately a sixfold increase in EMD is noted between 4 and 13 years.

Contraction to First Sound (C-I)

The C-I period was found to prolong with decreasing heart rate but was largely independent of age. Rate-dependent alterations in the C-I period roughly approximated those of systole; C-I, therefore, occupied a relatively constant percentage of systole in all age groups (table 3).

Q-I Interval

The Q-I interval (transformation time) comprises both the EMD and C-I periods. It is dependent on both age and heart rate alterations and was found to prolong with both increasing age and decreasing rate. The composition of the Q-I period also changed markedly with aging. In children 1 to 3 years old the Q-I interval largely reflected the duration of C-I, whereas in 13 year olds EMD accounted for approximately half of Q-I.

Isometric Contraction Time

ICT was found to prolong with increasing age and was independent of heart rate within the range studied. The duration of isometric contraction increased approximately two and a half times between the ages of 1 and 13 years and occupied a slightly increased percentage of systole in older children.

Pre-ejection Period

PEP comprises the Q-I and ICT phases and therefore relates twice to age and once to rate. PEP was prolonged in older children due to a direct relationship with age and indirect relationship with rate. PEP occupied a progressively greater percentage of systole with maturation.

Ejection Time

ET was found to relate independently to age and heart rate. Though ET prolonged with increasing age, it occupied a progressively smaller percentage of total systole (table 3).

Systolic Quotient

The systolic quotient was inversely related to age and relatively independent of rate (fig. 7). The quotient reflects the relative duration of PEP and ET and decreases with aging.

Discussion

The duration of the various systolic phases in children differs markedly from those observed in adults. Though some of these differences may be explained by the more rapid heart rates of childhood, age per se is an important determinant of phase duration. This is illustrated (table 4) by comparing values for systolic phase duration in adults (based on data from 121 normal males studied by Weissler and associates) with values derived...
in this study for 3-year-old children, both assuming a heart rate of 100 beats/min. In order to define the relative contribution of age and heart rate to systolic phase duration, a large sample was studied permitting a comparison of children of different ages with similar heart rates and children with varying heart rates within a single age group. Using this type of analysis, a consistent pattern of phase alteration was observed with maturation. The precise maturation factors responsible for these alterations were not identified, though it seems likely that heart size, blood pressure, and blood volume, are important in this regard. Theoretically, some of the observed changes in phase duration could relate to extracardiac factors such as thoracic development. Autonomic activity must also be considered a significant factor. Figure 8 summarizes the relationships among the duration of the systolic phases and age and rate.

Systole (Q-II) was found to relate inversely to heart rate and indirectly to age. For a given rate, systole was prolonged in older children and diastole was abbreviated. Diastole, therefore, occupied a smaller percentage of the cardiac cycle, though in absolute terms its duration increased in older children because of a slower heart rate. Interestingly, a prolongation of systole has been reported to occur in old age. In our study, the prolongation of systole with age was largely due to a long PEP, and ET, though also lengthened, occupied a progressively smaller percentage of systole in older children. The long systole found in old age was thought to be due primarily to an increase in PEP. A recent study, however, has shown that ET also is prolonged in old age independently of changes in heart rate and blood pressure. The observed alterations in systole, PEP, and ET occurring with maturation in children are, therefore, qualitatively similar to those seen in old age.

The duration of PEP has been taken as an index of myocardial contractility and is correlated with ejection fraction, left ventricular end-diastolic pressure, cardiac output, and stroke volume. Weissler has shown that congestive heart failure in adults results in a prolongation of PEP and an abbreviated ET. Digitalis will reverse these effects and cause a shortening of total systole. The changes in PEP and ET which occur with

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**Table 4**

<table>
<thead>
<tr>
<th></th>
<th>Adults</th>
<th>Children (3 yr old)</th>
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<tbody>
<tr>
<td>Cycle</td>
<td>600</td>
<td>600</td>
</tr>
<tr>
<td>Diastole</td>
<td>264</td>
<td>341</td>
</tr>
<tr>
<td>Systole (Q-II)</td>
<td>336</td>
<td>259</td>
</tr>
<tr>
<td>PEP</td>
<td>91</td>
<td>27</td>
</tr>
<tr>
<td>ET</td>
<td>243</td>
<td>72</td>
</tr>
<tr>
<td>Q-I</td>
<td>50</td>
<td>15</td>
</tr>
<tr>
<td>ICT</td>
<td>38</td>
<td>11</td>
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</table>

Abbreviations: Same as in table 1.
aging in children could reflect alterations in myocardial contractility or primarily relate to changes in preload and afterload consequent to increased ventricular volume and higher mean aortic pressures. The relative duration of ET and PEP is reflected by the SQ which is largely independent of rate and decreases with aging in childhood.9 In adults the quotient is constant over a wide age and rate range and is reported to be decreased in patients with congestive failure2 and coronary artery disease.21

No significant sex differences in phase duration were found in our study, though both systole (Q-II) and ET have been found to be longer in adult females than in males.2 This sex differential may only become apparent after puberty. Galstian9 reported ET to be shorter and the heart rates more rapid in girls 7 to 15 years old. The decreased ET seems to be a rate phenomenon, as no significant differences were found in our study for the entire population or when children with similar heart rates were compared. In the only age group in which a sex differential existed (6 year olds) there was also a marked difference in heart rate between boys and girls.

The shortened PEP found in young children was due largely to decreased duration of EMD and ICT. These two phases were independent of heart rate and directly related to age. ICT has been reported to be independent of rate in both adults52 and children.4 EMD represents the time interval between excitation and the onset of precordial movement as measured from the Q wave of the electrocardiogram (lead II) to the onset of the systolic wave of the apexcardiogram and is less than 5 msec in children under 4 years. EMD prolongs with aging in children but is relatively constant in adults, with 38 msec frequently taken as a mean value.23 Since EMD and ICT do not alter with rate changes, they occupy an increased percentage of systole at more rapid heart rates. These phases would appear to be theoretically rate-limiting; however, our study includes only physiologic rate changes and different phase relationships may be operative with marked bradycardia or tachycardia. Willems and Kesteloot,24 for example, found ET to relate linearly to heart rate, but exponential or polynomial relationships existed in paroxysmal atrial tachycardia or with complete A-V block.

The C-I interval was not related to age and varied inversely with heart rate. This period paralleled rate changes both absolutely and as a percentage of systole. Some of the difficulty in utilizing the Q-I interval clinically may relate to the disparity in the physiologic correlations of its component phases, EMD and C-I. For example, C-I has been found to be a good index of the severity of mitral stenosis, whereas Q-I is poorly correlated.7

The measurement of the systolic phases in children by external graphic recordings can be potentially useful in the diagnosis and quantification of congenital defects, assessment of myocardial function, and screening for pediatric heart disease. Clinical application, however, is predicated on an understanding of the normal phase relationships in childhood. External recordings lend themselves to computer applications in which wave forms can be digitized and phase duration calculated directly.25 Such a system could provide instantaneous data on phase alterations and would eliminate the subjective errors inherent in determining phase duration by hand reading.

The changes in systolic phase duration occurring with maturation presumably represent the normal functional adaptation of the developing heart. This is manifested by an orderly redistribution of systolic intervals resulting in a characteristic phase structure for each age group and heart rate. Further study of the precise maturation factors responsible for these changes is necessary. Measurement of the systolic phases in various childhood disease states can provide valuable information relative to the effect of these diseases on cardiac function.

Acknowledgment

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References
22. Frank MN, Kinlaw WB: Indirect measurement of isovolumetric contraction time and tension period in normal subjects. Amer J Cardiol 10: 800, 1962