Cardiac Function in the Normal Newborn

Additional Information by Computer Analysis of the M-Mode Echocardiogram

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SUMMARY  Computer analysis of the M-mode echocardiogram in 50 normal newborns provided measurements of wall thicknesses and chamber size and, in addition, assessment of right and left ventricular wall, septal, and cavity dynamics throughout the cardiac cycle. Data obtained with this new technique indicated that (1) right and left ventricular cavity functions are similar in the normal newborn, (2) right and left ventricular cavity filling and emptying vary directly with peak rates of septal and ventricular wall thickening and thickening, respectively, and (3) there is a close time relationship among maximum left atrial dimension, minimum left ventricular dimension, and mitral valve opening. This analysis, which is the first complete analysis of the echocardiogram in the newborn, provides a normal range of septal and ventricular wall dynamics as well as right and left ventricular and left atrial function and has clinical implications in that it may allow early recognition of both congenital and perinatal myocardial disease.

M-MODE ECHOCARDIOGRAPHY lends itself well to the study of normal infants; it is an invaluable tool in the comprehension of cardiac anatomy and in elucidating the relations between the atrioventricular valves and the ventricles and between the ventricles and the great vessels in congenital heart disease.  Hitherto, attention has been confined mainly to the anatomic disposition of cardiac structures and estimation of chamber size.  There is relatively little information regarding right or left ventricular function except for estimates of atroventricular valve closing velocities, systolic time intervals, and mean circumferential fiber shortening.

In order to investigate right and left ventricular function in the newborn in greater depth and to examine left atrial function and its contribution to left ventricular filling, we analyzed M-mode echocardiograms by computer. This technique provides measurements of instantaneous right and left ventricular and left atrial cavity dimensions and also instantaneous thickness of the septum and ventricular walls throughout the cardiac cycle. From these measurements, which are obtainable manually only with difficulty, peak rates of systolic thickening and diastolic thinning of the right and left ventricular walls and septum were obtained so that their respective contributions to right and left ventricular filling and emptying could be assessed.

Methods

Echocardiograms and simultaneous electrocardiograms were obtained from 50 clinically normal newborns ranging in age from 1½ to 64 hours, with a mean age of 19 hours. Twenty-six were female and 24 were male; they ranged in weight and body surface area from 2.6 to 4.5 kg and 0.18 to 0.28 m², respectively.

Echocardiograms were obtained with an Ekoline 20 ultrasonoscope using a 0.5-inch diameter, unfocused, 5 MHz transducer with a repetition frequency of 1,000 cycles/second. Recordings were made on a Cambridge Scientific Instruments multichannel strip-chart recorder at paper speeds of 100 mm/sec with simultaneous electrocardiograms while the patients were recumbent. Echoes of the right and left sides of the septum, endocardium, and epicardium of the right and left ventricular walls were obtained at the level of the mitral valve (fig. 1). Aortic root and left atrial wall echoes were recorded at the level of the aortic valve (fig. 2). Measurements were made only when these echoes were clear and continuous throughout the cardiac cycle. Echocardiograms were digitized as previously described with the use of a Science Accessories Corporation Graf pen and digitizing table, and the data were processed by a CDC 3500 computing system operating the master time-sharing mode. Echoes were calibrated with

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points defining a time interval of 2 sec, depth of 4 cm, and two successive Q waves of the electrocardiogram enclosing the cardiac cycle to be analyzed. Data points were generated at intervals of 6 msec for both endocardial surfaces of the septum, the endocardial and pericardial surfaces of the right and left ventricular walls, the posterior aortic root, and the posterior left atrial wall, so that a “string” of coordinates was obtained for each surface boundary. Based on these measurements, plots were made of continuous right and left ventricular and left atrial cavity dimensions, septal and right and left ventricular wall thickness, and anterior mitral valve leaflet velocity. These time-varying values were displayed on a Tektronic scan converter, from which permanent records were obtained photographically and from an on-line printer. From these, the dynamics of right and left ventricular and left atrial cavities and of septal and right and left ventricular walls were calculated as follows.

Right and Left Ventricular Dynamics

Percentage Change of Ventricular Cavity Dimension in Systole. This was obtained by subtracting end-systolic dimension (ESD) from end-diastolic dimension (EDD) and expressing the difference as a percentage of end-diastolic dimension:

$$\frac{EDD - ESD}{EDD} \times 100\%.$$  

Peak Rate of Increase in Cavity Dimension during Diastole. This was identified by computer as peak dD/dt in diastole, where D equals dimension and t is time in seconds.

Normalized Peak Rate of Change of Cavity Dimension in Systole. This was identified by computer and obtained by dividing the peak rate of change of cavity dimension, dD/dt, in systole by instantaneous cavity dimension, \( \frac{1}{D} \frac{dD}{dt} \) (systole). This assumes that the cavity has a circular cross section and changes dimensions concentrically.

Q Wave to Time of Minimum Cavity Dimension. This was the time interval from the onset of the QRS complex to the instant of minimum cavity dimension, the latter being defined as that time at which dD/dt = 0.

Q Wave to Time of Peak Rate of Increase in Cavity Dimension. This was the time interval from the onset of the QRS complex to the time at which peak dD/dt was attained during diastole.

Duration of Rapid Filling Phase. This was the time interval from minimum cavity dimension (dD/dt = 0) to the time of the sudden decrease of rate of filling of the chamber on the plot of continuous cavity dimension; the latter was identified by computer and coincided in time with the point when dD/dt (diastole) had just decreased to 20% of its maximum value.

Ejection Fraction. This was obtained by using the cube formula to calculate left ventricular end-diastolic and end-systolic volumes and stroke volume, and the latter was then expressed as a percentage of end-diastolic volume.

Peak Diastolic Closing Velocity of the Anterior Mitral Valve Leaflet. This velocity was identified by the computer from the curve of computed instantaneous velocity of the mitral valve leaflet.

Left Atrial Dynamics

Peak Rates of Diastolic and Systolic Change of Dimension. The peak rates of increase in dimension during filling and reduction during emptying were selected by computer (dD/dt filling and dD/dt emptying).

Q Wave to Time of Maximal Left Atrial Dimension. This was the time from the onset of the QRS complex to the point in time at which maximal left atrial dimension occurred.

Q Wave to Time of Peak Rate of Increase of Dimension. This was the time from the onset of the QRS complex to the instant of peak rate of increase of the left atrial dimension, dD/dt (filling).

Left atrial echoes were obtained within minutes of the left

**Figure 1.** Normal echocardiogram of newborn showing anterior right ventricular wall (AW), right ventricular cavity (RV), interventricular septum (VS), mitral valve leaflets (MV), and posterior left ventricular wall (PW), with simultaneous electrocardiogram.

**Figure 2.** Normal echocardiogram of newborn showing left atrium (LA), aorta and aortic valve (AV), and right ventricular outflow (RVO), with simultaneous electrocardiogram.
TABLE 1. Left and Right Ventricular Cavity Dynamics in 50 Normal Newborns

<table>
<thead>
<tr>
<th></th>
<th>EDD (mm)</th>
<th>ESD (mm)</th>
<th>Time from Q to minimum ventricular dimension (msec)</th>
<th>Ejection fraction (%)</th>
<th>Peak dD/dt (cm/sec)</th>
<th>Time from Q to peak dD/dt (msec)</th>
<th>Peak L dD/dt (VCF)</th>
<th>Duration of rapid filling period (msec)</th>
<th>Mitral valve ant. cusp velocity (mm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricle</td>
<td>17.3 ± 2.0</td>
<td>9.6 ± 2.2</td>
<td>282 ± 28</td>
<td>77 ± 7</td>
<td>7.7 ± 1.9</td>
<td>340 ± 26</td>
<td>3.8 ± 1.0</td>
<td>138 ± 28</td>
<td>93 ± 23</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>10.4 ± 1.0</td>
<td>6.8 ± 1.6</td>
<td>282 ± 35</td>
<td></td>
<td>3.7 ± 1.1</td>
<td>351 ± 38</td>
<td>3.6 ± 1.0</td>
<td>139 ± 35</td>
<td>—</td>
</tr>
</tbody>
</table>

ventricular echogram, and only beats of the same R-R interval duration were digitized and analyzed for correlation of left atrial and left ventricular events.

**Anterior Right Ventricular Wall (AW), Left Ventricular Posterior Wall (PW), and Septal (VS) Dynamics**

**Percentage Systolic Thickening.** This was obtained by subtracting end-diastolic thickness from end-systolic thickness and expressing the difference as a percentage of end-diastolic thickness. For the right ventricular anterior wall this would be

\[ \frac{AW_s - AW_{di}}{AW_{di}} \times 100\% \]

**Normalized Peak Rates of Systolic Thickening.** These were obtained by dividing the peak systolic rates of change,

\[ \frac{1}{AW} \frac{dAW}{dt} \]  

(systole), by the instantaneous dimension,

\[ \frac{1}{AW} \frac{dAW}{dt} \]  

(systole).

**Normalized Peak Rates of Diastolic Thinning.** These were obtained by dividing the peak diastolic rate of change,

\[ \frac{1}{AW} \frac{dAW}{dt} \]  

(diastole), by the instantaneous dimension,

\[ \frac{1}{AW} \frac{dAW}{dt} \]  

(diastole).

Q Wave to Time of Peak AW, PW, and VS Thickness. This was the time from onset of the QRS complex to the times of peak thickness of AW, PW, and VS, which were all identified by computer.

In only 24 newborns was the echocardiographic tracing of the right ventricular epicardium sufficiently clear and continuous to be digitized, so that the results for right ventricular wall dynamics are based on the analysis of the data of these 24 patients only.

**Results**

Right Ventricle

Mean values for right ventricular cavity dimension and for anterior wall and septal thickness during systole and diastole and their percentage change are shown in tables 1 and 2. On the instantaneous right ventricular dimension trace, minimum dimension occurred 282 ± 35 msec from the onset of the QRS complex, after which there was a rapid increase in dimension — representing the rapid diastolic filling phase — of 139 ± 35 msec duration and a normal period of diastasis during which there was little additional change in dimension (fig. 3). Peak rate of increase of right ventricular dimension (dD/dtaw) was 3.7 ± 1.1 cm/sec and this occurred 351 ± 38 msec after the onset of the QRS complex. The mean normalized velocity of computed circumferential shortening was 3.6 ± 1.0 sec⁻¹.

Plots were made of peaks rates of septal and anterior wall systolic thickening and right ventricular peak rate of reduction of cavity dimension and also peak rates of septal and

**TABLE 2. Regional Dimensions of Septum and Right and Left Ventricular Walls in Normal Newborns**

<table>
<thead>
<tr>
<th>Syst thickness (mm)</th>
<th>Diast thickness (mm)</th>
<th>% change in wall thickness</th>
<th>Time from Q to peak wall thickness (msec)</th>
<th>Peak L dAW/dt (cm/sec)</th>
<th>Peak L dVS/dt (cm/sec)</th>
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<tr>
<td></td>
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<tr>
<td><strong>Right ventricular wall (N = 24)</strong></td>
<td></td>
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<tr>
<td>5.1 ± 0.6</td>
<td>2.4 ± 0.3</td>
<td>127 ± 38</td>
<td>296 ± 35</td>
<td>5.5 ± 1.7</td>
<td>7.9 ± 2.2</td>
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<td></td>
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</tr>
<tr>
<td><strong>Septum (N = 50)</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>5.6 ± 0.9</td>
<td>3.0 ± 0.6</td>
<td>95 ± 40</td>
<td>276 ± 36</td>
<td>6.0 ± 2.1</td>
<td>5.6 ± 2.2</td>
</tr>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Left ventricular wall (N = 50)</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.4 ± 0.8</td>
<td>2.5 ± 0.6</td>
<td>130 ± 50</td>
<td>296 ± 28</td>
<td>5.4 ± 1.3</td>
<td>8.0 ± 2.2</td>
</tr>
</tbody>
</table>
anterior wall diastolic thinning against peak rate of increase of right ventricular cavity dimension during diastole. There was a weak linear relationship between septal and anterior wall dynamics and right ventricular cavity filling \((r = 0.52, r = 0.61)\) and emptying \((r = 0.54, r = 0.49)\) (fig. 4). Although peak rates of systolic thickening of the anterior wall and ventricular septum were similar, the peak rate of anterior wall thinning was consistently and significantly greater than that of the septum.

In order to be able to compare rates of systolic thickening and diastolic thinning of the walls and septum from one newborn to another in whom septal and wall thickness varied, these rates were normalized by dividing them by their own instantaneous dimension (table 2).

**Left Ventricle**

Mean values for left ventricular cavity dimension, posterior wall and septal thickness in systole and diastole, percentage systolic change, and ejection fraction are shown in tables 1 and 2. The plot of instantaneous left ventricular dimension was similar to that of right ventricular dimension (fig. 5); minimum dimension occurred 282 ± 28 msec after the onset of the QRS complex, and the duration of the rapid diastolic filling phase was 138 ± 28 msec, the same as in the right ventricle. Peak rate of increase of left ventricular dimension, \(dD/dt\) (diastole), was 7.7 ± 1.9 cm/sec, which is significantly higher than that in the right ventricle \((P < 0.001)\), and this occurred 340 ± 26 msec after the QRS complex, almost coincident with that of the right ventricle.
Computer outputs in figures 3 and 5 are from different patients and thus septal dynamics differ.

Discussion

End-systolic and end-diastolic right and left ventricular and left atrial cavity dimensions and septal and ventricular wall thicknesses correlated well with values obtained by manual analysis.\textsuperscript{5, 9-14} Computer analysis of echocardiograms has the additional advantage of permitting continuous measurement of these structures and the ready computation of their respective instantaneous rates of change throughout the cardiac cycle. Assessment of peak rates of change rather than mean or fractional change of dimensions in adults has proved to be more sensitive to the early dynamic disturbances of regional left ventricular function which hitherto had remained unrecognized.\textsuperscript{19}

Traces of continuous right and left ventricular dimensions were similar in that the timing of both minimum dimension and peak filling rate and the duration of the rapid diastolic filling phase were identical in both ventricles (table 1, figs. 3 and 5). The peak filling rate of the right ventricle itself was significantly less than that of the left for two possible reasons. 1) During filling, the anterior right ventricular wall and the septum both move anteriorly, whereas in the left ventricle the septum and the posterior wall move in opposite directions and thereby increase the size of the cavity more rapidly. 2) Because of its crescentic shape, the right ventricle appears smaller than the left ventricle echocardiographically, and its rate of change is therefore comparably less. However, the right ventricular dimension in the echograms is reproducible and is of particular value for comparison in newborns, in whom right ventricular size is either increased (as in volume overload) or decreased (as in right ventricular hypoplasia).

The diastolic closing velocity of the mitral valve has been used as an index of left ventricular distensibility, which is largely determined by wall thickness. In the newborn, right and left ventricular wall thicknesses are comparable, probably because of equal ventricular afterloads in utero; and an explanation for the similar tricuspid and mitral valve closing velocities in the newborn is that wall thicknesses and therefore distensibilities of the right and left ventricles are approximately the same.

The cross section of the left ventricle at the level of the mitral valve in the newborn is approximately circular, and the echo beam passes through a diameter. There is a good correlation between measurements of the minor left ventricular axis obtained by echocardiographic and angiographic techniques.\textsuperscript{19-22} Although only a limited region of the left ventricle can be studied echocardiographically, endocardial and epicardial positions and motion can be defined accurately throughout the cardiac cycle. Inward and outward movement of the septal and ventricular wall echoes in the

**Table 3. Left Atrial Dynamics in 50 Normal Newborns**

<table>
<thead>
<tr>
<th>Time from Q to maximum dimension (msec)</th>
<th>Peak $\frac{1}{D} \cdot \frac{dD}{dt}$ (filling) (msec$^{-1}$)</th>
<th>Peak $\frac{1}{D} \cdot \frac{dD}{dt}$ (emptying) (msec$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left atrium</td>
<td>$14.0 \pm 2.1$</td>
<td>$8.2 \pm 1.4$</td>
</tr>
<tr>
<td></td>
<td>$3.1 \pm 0.5$</td>
<td>$3.4 \pm 1.0$</td>
</tr>
</tbody>
</table>
region of the left ventricle studied was assumed to be representative of the ventricle as a whole.

In the normal heart, changes in the minor axis of the left ventricular cavity are due to concentric outward wall and septal motion in diastole and inward wall and septal motion toward the center of mass in systole. Diastolic outward wall motion is brought about by the synchronous and simultaneous free wall and septal thinning, which results in an increase in left ventricular cavity size to accommodate the blood from the left atrium. The concentric inward wall and septal motion that reduces ventricular cavity volume during ejection is achieved by free wall and septal thickening.

The temporal coordination of septal and free wall thickening in both right and left ventricles is indicated by the simultaneous occurrence of peak thickness of the right and left ventricular walls and minimum right and left ventricular cavity dimensions, in close time relation to mitral valve opening and a few milliseconds after peak septal thickness (tables 1 and 2). The delay in development of peak thickness in the right and left ventricular walls compared with that of the septum may be explained by the earlier electrical depolarization of the septum.

The overall shape of the left ventricle resembles a prolate ellipsoid whose cavity volume and stroke volume can be calculated from measurements of the minor axis. Rates of change of dimension and volume during emptying and filling can be estimated by computer analysis of the left ventricular echocardiogram, and they provide greater insight into the normal function of the left ventricular cavity. Peak rates of filling and ejection of the left and right ventricles are dependent on peak rates of free wall and septal thinning and thickening, respectively (fig. 4). In both ventricles, peak rates of systolic thickening of the septal and ventricular walls were approximately the same, and this suggests that nearly equal contributions were made by each to the rate of ejection (fig. 4). During relaxation, however, the peak rates of thinning of the right and left ventricular walls were considerably faster than those occurring in the septum (fig. 4), and thus the rates of diastolic filling in both ventricles appear to be dependent to a greater extent on the rate of thinning of the free walls than on that of the septum.

Abnormalities in ventricular function due to either primary or secondary disease of the heart muscle are manifested as changes in the properties of the myocardial tissue enclosing the cavity. These changes in free wall and septal dynamics can be readily quantitated as reduction in peak rates of systolic thickening and diastolic thinning. Since ventricular cavity filling and emptying are largely dependent on septal and free wall peak rates of thickening and thinning (fig. 4), it follows that abnormalities in ventricular function may first be detected when these regional dynamics are assessed. This is evident in adults with left ventricular disease, in whom ventricular size and ejection fraction often remain within the normal range in spite of severely disturbed regional dynamics. There is, however, variation in wall and septal dynamics in the normal newborn (fig. 4), and it is therefore important to establish whether there is a well-defined normal range of values so that abnormalities in wall dynamics can be identified before changes in cavity function.

Reduced rate of wall thickening results in reduction in the rate of inward wall movement and loss of kinetic energy that would otherwise be translated into work done on the enclosed cavity and the circulation. Similarly, reduction in rate of diastolic thinning due to increased wall stiffness causes slow diastolic outward wall motion and reduced peak rate of ventricular filling. Peak rates of septal and ventricular wall thickening and thinning are sensitive indices of myocardial performance, although reduction in peak rate of diastolic wall thinning appears to be more sensitive to minor changes in left ventricular function.

Little importance has been attributed to left atrial function except in infants with patent ductus arteriosus and ventricular septal defects, in whom the ratio of maximal left atrial dimension to aortic diameter has been helpful in correlating the size of the left-to-right shunt. The timing of the echoes of anterior and posterior movement of the aortic root in the newborn is the same as in adults, in whom it is very similar to that of the left atrial volume tracing and can

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**Figure 6.** Computer output of normal left atrial echocardiogram. Lowest tracing represents continuous left atrial dimension (D). Above this are plots of rate of change of left atrial dimension (dD/dt) and its normalized rate of change throughout cardiac cycle, with electrocardiogram at top.
Therefore be used for accurate timing of left atrial and ventricular events. Although the necessary angiographic studies in the normal newborn are, of course, not available, the echocardiographic dimension of the left atrium in teenagers and adults correlates with left atrial area and volume obtained angiographically, and one would expect to find this correlation in the newborn also.

While the mitral valve is closed, blood distends the thin left atrial wall and the result is an almost linear increase in left atrial cavity dimension (fig. 6). Maximum left atrial dimension occurs simultaneously with minimum left ventricular dimension and in close time relation with mitral valve opening as it occurs in adults. After mitral valve opening, there is a rapid reduction in left atrial size, followed by a slower rate of decrease; minimum dimension occurs synchronously with mitral valve closure. The slower reduction in left atrial dimension often observed in mid to late diastole may be explained by pulmonary venous blood flowing through the mitral valve orifice into the left ventricle while left atrial dimension is still decreasing.

In addition to maximum left atrial dimension, the timing and peak rates of filling and emptying offer new information regarding normal atrial function in the newborn and may be helpful in recognizing congenital structural or myocardial anomalies that (1) alter the rate of egress of blood from the right heart, lungs, and pulmonary veins and (2) obstruct left atrial emptying, whether at atrial, mitral valve, or ventricular level.

Computer analysis of the total M-mode echocardiogram in the newborn provides readily accessible and reproducible information about the dynamic function of the septum and the right and left ventricular walls and their effects on right and left ventricular cavity filling and emptying. It demonstrates the similarity between the right and left ventricles at birth and establishes the time relations between left atrial and left ventricular events. These normal values of myocardial function of both ventricles and the left atrium in the newborn vary with heart rate and are useful as a basis of comparison only in newborns with similar heart rates. These data have clinical implications in that they provide the basis with which myocardial dysfunction, whether imposed by congenital anomalies or by perinatal disease, may be recognized at an early stage.

Acknowledgment

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