Left Ventricular Regional Wall Motion in the Early Neonatal Period

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HARADA, K., TAKAHASHI, Y., SHIOTA, T., NOGUCHI, H. and TAKADA, G. Left Ventricular Regional Wall Motion in the Early Neonatal Period. Tohoku. J. Exp. Med., 1995, 177 (2), 107-115 —— To investigate the changes in regional wall motion of the left ventricle in the early neonatal period, serial echocardiography was performed in normal neonates at 2 and 120 hr after birth. Quantitative analysis of the regional wall motion was performed by the centerline method. We measured right ventricular systolic time intervals, left ventricular stroke volume, flow velocity-time integral of the pulmonary artery, and size of the ductus arteriosus. The ductus arteriosus was 4.5±0.5 mm at 2 hr but was closed in all subjects by 120 hr. At 2 hr, there was hyperkinesis of the interventricular septum which disappeared by 120 hr. The right ventricular systolic time intervals at 2 hr showed a sign of pulmonary hypertension. At 2 hr, the left ventricular stroke volume was at the highest level and the flow velocity-time intervals of pulmonary artery was at the lowest level. Thus the hyperkinesis of the interventricular septum at 2 hr might reflect the circulatory changes that are characteristic of the early neonatal period. —— echocardiography; neonate; left ventricular wall motion

The transition from the fetal to the neonatal circulatory status is accompanied by marked alteration of loading conditions. Two of the more important changes are the decrease in pulmonary artery pressure and the functional closure of the ductus arteriosus. The right ventricular pressure is approximately equal to the left ventricular pressure at birth and rapidly falls to near normal levels within a few days (Emmanouilides et al. 1964) In normal full-term neonates, the ductus arteriosus closes within the first day of life (Moss et al. 1963; Arcilla et al. 1967; Mahoney et al. 1985). These alterations after birth are considered to influence both global and regional performances of the left ventricle. Our previous studies demonstrated the changes in left ventricular function during the early neonatal period (Harada et al. 1994a, b; Takahashi et al. 1994a, b) Although the left ventricular regional wall motion in normal newborns have been
studied by several investigators (Rein et al. 1987; Vogel et al. 1990; Miyazaki et al. 1992), serial changes of the regional left ventricular wall motion in the early neonatal period have not been fully evaluated. Therefore, the aim of this study is to evaluates the changes in regional wall motion of the left ventricle in the early neonatal period.

**Subjects and Methods**

The study population consisted of 17 normal term neonates. Their mothers had uncomplicated pregnancies with no evidence of toxemia, diabetes mellitus or pregnancy-induced hypertension. None of the infants was acutely ill or had any evidence of congenital malformations. The gestational age at birth ranged from 36 to 41 weeks (mean: 39 weeks), and the birth weight ranged from 2,680 to 3,934 g (mean: 3,041 g). Informed consent was obtained from their mothers according to a protocol approved by the Human Subjects Committee. Serial echocardiographic examinations were performed on the same subjects at 2 and 120 hr after birth.

**Echocardiography**

Echocardiographic examinations were performed by an Aloka SSD-870 (Aloka, Tokyo) ultrasound imaging system equipped with a 5.0 MHz transducer. All neonates were examined while they were lying quietly in the supine position breathing room air.

**Analysis of left ventricular regional wall motion**

Centerline chord motion analysis, as described by Sheehan et al. (1983), was

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Fig. 1. Centerline method of regional wall motion analysis. End-diastolic and end-systolic left ventricular endocardial contours and centerline constructed by the computer midway between the two contours. Motion is measured along 100 chords constructed perpendicular to the centerline.
used to quantitatively assess regional left ventricular function (Cardio 500; Kontron Medical System, Tokyo) (Fig. 1). In this technique, endocardial motion is measured along 100 chords constructed perpendicular to a centerline drawn midway between the end-diastolic and end-systolic contours in the apical long-axis view. The last 25 chords are not analyzed because they reflect primary motion of the mitral valve. The motion of each chord is then normalized for heart size by dividing by the length of the end-diastolic circumference. The endocardial borders at end-diastole and end-systole were traced manually, with a light pen, and were realigned for translation and rotation by superimposing the center of each contour and the axis according to the floating method. End-diastole was defined as the video frame at or before the initial systolic coaptation of the mitral valve leaflets or as the first frame in which the QRS complex appeared. End-systole was defined as the frame preceding the initial early diastole opening of the mitral valve. All images were traced along the innermost edge of the endocardial echoes as they abutted the left ventricular cavity. All the tracing were performed by two of the authors who were unaware of each patient's status.

**Pulsed Doppler echocardiography**

The size of the Doppler sample volume was set at an axial length of 2 mm and a wall filter setting of 400 Hz was used. To record the pulmonary arterial flow velocity profile, a standard short axis view was visualized and the Doppler sample volume was placed in the main pulmonary artery immediately distal to the pulmonic valve. Ascending aortic flow was measured from the suprasternal long axis view. The sample volume was placed in the ascending aorta immediately distal to the aortic valve. Care was taken to obtain the study with the transducer beam as close as possible to parallel the presumed blood flow direction. The angle between the estimated direction of blood flow and the Doppler beam was 20 degrees or less in the selected planes. No angle correction of the Doppler signal was made. All examinations were recorded at a paper speed of 100 mm/sec. We simultaneously recorded the patients electrogram.

Four Doppler flow indices were measured from the main pulmonary artery: Systolic flow velocity-time integral, which was measured in cm as the area under the systolic flow velocity curve; acceleration time in msec, pre-ejection period in msec; and ejection time in msec. The ratio of the pre-ejection period to the ejection time was calculated. The acceleration time was measured from the onset of ejection to the point of maximum blood flow velocity. The pre-ejection period was measured from the onset of the Q wave of the simultaneously recorded electrocardiogram to the onset of ejection. The ejection time was measured from the onset of ejection to the end of ejection. Each of the above indices was calculated as the mean value of five ventricular systoles.

Left ventricular stroke volume was calculated as the product of the flow
velocity-time integral of ascending aorta and the aortic valve area. The flow velocity-time integral was determined from the ascending aortic flow tracings and calculated using a computer-interfaced digitizer pad (Cardio 500; Kontron Medical System). For diameter measurements, the ascending aorta was viewed in the long axis, with the M-mode beam being directed perpendicular to its anterior and posterior walls at the level of the aortic valve. Aortic root measurements were made at the beginning of the QRS complex. The aortic valve area was then calculated by assuming that the valve orifice was circular.

**Measurement of blood pressure**

The peak pressure and diastolic pressure were also simultaneously obtained using Dinamapp 8100-TM vital sign Monitor (Criticon Inc., Tampa, FL, USA). The mean arterial pressure was automatically calculated. The mean pulmonary arterial pressure was estimated by the regression equation of Kitabatake et al. (1983): \( \log_{10} (\text{mean pulmonary arterial pressure}) = -2.8 \times \left( \frac{\text{the ratio of the acceleration time to ejection time}}{\text{ejection time}} \right) + 2.4 \). The ratio of the mean pulmonary arterial pressure to mean arterial pressure was calculated.

**Ductus arteriosus**

The inner diameter and peak velocity of shunt flow of the ductus arteriosus were measured serially after birth at the same time the other hemodynamics were measured. The size of the ductus arteriosus was determined from the inner diameter on the two-dimensional color echocardiographic images. The inner diameter was measured at the narrowest portion of the ductal lumen in the parasternal long-axis plane. The ductus arteriosus was considered closed when a color image and shunt flow were no longer detected.

**Interobserver variability**

To determine interobserver variability, 15 cross-sectional images were traced by different experienced technicians. End-systolic and end-diastolic contours were traced by a second independent observer blinded to the other's results. Observer variability was determined by dividing the difference between observers by the mean of their observations.

**Statistical analysis**

The data are presented as the mean ± S.D. Paired Student's t-test was used to evaluate the differences in a set of measurements between 2 and 120 hr of age. Results with \( p \) values less than 0.05 were considered to be statistically significant.

**RESULTS**

**Serial changes in regional wall motion of the left ventricle**

Shortening fraction in 5 to 35 segments at 2 hr was significant large compared
with the values at 120 hr (Fig. 2).

The echocardiographic measurements and hemodynamic observations in neonates are presented in Table 1. The mean heart rate showed no significant changes between 2 and 120 hr.

**Flow velocity pattern of main pulmonary artery**

The pre-ejection period decreased from a mean of 90 msec at 2 hr to 56 msec at 120 hr. The ejection time increased from a mean 189 msec at 2 hr to 221 msec at 120 hr. The mean acceleration time increased from 53 msec at 2 hr to 98 msec at 120 hr. The ratio of the pre-ejection time to ejection time declined from 0.49 at 2 hr to 0.25 at 120 hr. The ratio of the acceleration time to ejection time
increased from 0.29 at 2 hr to 0.44 at 120 hr.

The mean pulmonary arterial pressure estimated from Kitabatake's regression line decreased with increasing age. The ratio of the mean pulmonary arterial pressure to the mean aortic pressure declined from a mean of 0.93 at 2 hr to 0.27 at 120 hr.

**Serial changes in flow velocity-time integral of main pulmonary artery**

The flow velocity-time integral of main pulmonary artery was at the lowest level at 2 hr and increased significantly compared with the values at 12 hr.

**Serial changes in left ventricular stroke volume**

The left ventricular stroke volume decreased significantly at 120 hr compared with that at 2 hr.

**Ductus arteriosus diameter**

The inner diameter of the ductus arteriosus was $4.5 \pm 0.7$ mm at 2 hr. The ductus arteriosus was closed in all subjects by 120 hr. The peak velocity of the ductal shunt flow at 2 hr was less than 1.19 m/sec in all subjects (range: 0.75-1.19 m/sec).

**Interobserver variability**

The mean interobserver variability for all segments was $24 \pm 20\%$. It was lowest at the apex and similar in magnitude at interventricular septum and lateral portion (Fig. 3).

**DISCUSSION**

In this study, there was hyperkinesis of the interventricular septum at 2 hr.
The Doppler-derived hemodynamic parameters showed that LV stroke volume, RV systolic pressure, and flow velocity-time integral of pulmonary artery reflecting RV stroke volume at 2 hr were significantly different from those at 120 hr. Although the regional wall motion may be affected by the LV systolic function, our previous study showed that LV contractility was no significant changes during 120 hr (Takahashi et al. 1994a). Thus, hyperkinesis of the interventricular septum may reflect these circulatory changes that are characteristic of the early neonatal period.

The position and shape of the interventricular septum depend on the pressure gradient between left and right ventricles. The radius of curvature of the interventricular septum increased with right ventricular pressure overload, which is most pronounced at end-systole (King et al. 1983; Agata et al. 1987). However, the morphological changes of the septum may be more sensitive to the changes in transseptal pressure gradient in diastole than in systole (King et al. 1983; Agata et al. 1987). Previous study has shown wall motion and ventricular shape in infants younger than 1 weeks of age to be significantly different from those in older infants (Rein et al. 1987). These changes in left ventricular shape resolved by 5 days of life and were attributed to an increased right ventricular/left ventricular transseptal pressure difference, as seen with other conditions (King et al. 1983; Kingma et al. 1983; Agata et al. 1987). In our results, the right ventricular systolic pressure at 2 hr was approximately equal to the systemic pressure, causing flattening of the septum. Rein et al. (1987) have demonstrated that the left ventricle was circular in short axis at end-diastole even as early as 3 hr, and speculated that the end-diastolic pressure in the left ventricle was higher than that in the right ventricle. Previously, we showed the effect of the patent ductus arteriosus on left ventricular preload in the first day of life (Harada et al. 1994a, b). We demonstrated that the left ventricular stroke volume at 2 hr declined to 75% of the 120 hr-value (Harada et al. 1994b). In contrast, the flow velocity-time integral of the pulmonary artery which may reflect right ventricular end-diastolic volume was lower level at 2 hr compared with the values at 120 hr, as Takenaka et al. (1987) previously reported. These results may suggest that left ventricular end-diastolic pressure was larger than right. A comprehensive view of the hyperkinesis of the interventricular septum at 2 hr can therefore be proposed as follows: (1) at end-systole, high systolic pressure of the right ventricular distorts the interventricular septum toward the left ventricle, and (2) at end-diastole, the increase in end-diastolic pressure of LV, indicating high preload of LV, bulged the interventricular septum toward the right ventricle.

In this study, we applied the centerline method to evaluate for left ventricular wall motion. The validity of radial methods has been challenged by reports showing that motion proceeds toward many points rather than a single point in the ventricular chamber, and that motion measurement along radii from a single origin is erroneous (Shepertycki and Morton 1983). Assessment of inter-
observer variability indicate that the apex is one of the least reliably visualized parts of the contour. The dependence of rectangular and radial methods on the apex as a landmark in defining a long axis may produce error in wall motion measurement (Sheehan et al. 1983). The centerline method of regional wall motion analysis was developed to address these concerns. Its sensitivity and specificity in detecting abnormal regional function compare favorably with rectangular and radial methods (Sheehan et al. 1986) and its reliability has been previously documented (Sheehan et al. 1983). In our present study, interobserver variability was larger than that of Sheehan’s results (1983) measured from ventriculograms. This discrepancy may be attributed to the use of different methods. In this study, regional wall motion was analysed at 2 and 120 hr. However, changes in loading conditions during early neonatal period are dramatic within first 24 hr. Relationships between regional wall motion and changes in loading condition should be further studied in the early neonatal period.

Hyperkinesis of the interventricular septum may reflect circulatory changes that are characteristic of the early neonatal period. We think that the analysis of the regional wall motion is a useful method of identifying abnormal ventricular function in these neonates in whom cardiac and respiratory diseases are suspected, because the presence of septal hyperkinesis may indicate pulmonary hypertension or an increase in right ventricular pressure caused by obstruction of the right ventricular outflow tract. Data derived from the present study should serve as a valuable reference for further studies in this unique patient population.

References


