Peak Systolic Stress-Rate-Corrected Mean Velocity of Fiber Shortening in Preterm and Fullterm Infants

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HARADA, K., TAKAHASHI, Y., TOYONO, M., ORINO, T. and TAKADA, G. Peak Systolic Stress-Rate-Corrected Mean Velocity of Fiber Shortening in Preterm and Fullterm Infants. Tohoku J. Exp. Med., 1998, 184 (1), 13-20 —— The relation of rate-corrected mean velocity of fiber shortening (mVefc)-end-systolic wall stress (ESS) is a load-independent index of left ventricular contractility, but involves simultaneous M-mode echocardiography, carotid or axillary pulse tracing and blood pressure determination, which may be impractical in younger infants. We examined whether the relation of the peak systolic wall stress (PSS) mVefc could be used as a simpler method of assessing left ventricular contractility in preterm and fullterm infants. In 45 preterm and fullterm infants, mVefc, ESS, and PSS were determined using echocardiography, axillary pulse tracing and blood pressure measurement. Five patients with left ventricular dysfunction or low cardiac output state were also studied. The relation of PSS and ESS was PSS = 3.19 + 1.04 ESS (r = 0.98, p < 0.01). The slope of mVefc = 1.58 - 0.012 ESS (r = -0.78, p < 0.01) was nearly identical to that of mVefc = 1.60 - 0.011 PSS (r = -0.75, p < 0.01), with no difference in the regression coefficients. The relationship of PSS and ESS in 5 patients was very close and the slope of the regression line was nearly identical to that of 45 infants. The relation of mVefc - PSS correlates well with the relation using ESS and can be used as a simple method of assessing left ventricular contractility. —— echocardiography; preterm infant; systolic function; end-systolic wall stress © 1998 Tohoku University Medical Press

The accurate assessment of left ventricular function is becoming increasingly important in the management of premature infants. The shortening fraction remains as the most used echocardiographic index of left ventricular performance, however, this index is sensitive to alterations in loading conditions and heart rate (Colan et al. 1984). New echocardiographic techniques enable the measurement of afterload, as assessed by the relation of wall stress and velocity of circumferential fiber shortening. The method to get rate-corrected mean velocity of fiber shortening (mVefc)-end systolic wall stress (ESS) relation requires simultaneous

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M-mode echocardiography, blood pressure measurement, phonocardiogram, and carotid or axillary pulse tracing. Carotid or axillary pulse tracing may be technically difficult to obtain in some premature infants because additional manpower and hardware are not readily available for current echocardiographic equipment. Furthermore, it may take much more time to obtain this contractility index than shortening fraction and may cause the reduction in body temperature in premature infants, which might induce increased peripheral arterial resistance and left ventricular afterload. Left ventricular performance in the human preterm and term infant is highly dependent on afterload (Romero and Friedman 1979; Kimball et al. 1991; Igarashi et al. 1994; Rowland and Gutgesell 1995). If cardiac performance in the newborn deteriorates in the presence of higher afterload, one would expect that performance might worsen in the preterm infant. The usefulness of the relation in clinical practice is diminished by the complexity of the methodology, especially in premature infants. Previous studies have shown a close relationship between peak systolic and end-systolic pressures and their stress (Reichek et al. 1982; Sandor et al. 1992). By using the peak systolic pressure, the need for obtaining and analyzing pulse waveform may be eliminated. In this study, we examined whether the relation of mVcfc at peak systole could be used as a simple method of assessing left ventricular contractility in premature infants.

**Methods**

*Study subjects*

We examined 22 appropriate for gestational age preterm infants. The gestational age at birth ranged from 26 to 33 weeks, and the birth weight ranged from 866 to 1653 g. No infants had abnormal cardiovascular symptoms nor signs, and the absence of congenital heart disease was confirmed by two-dimensional and color Doppler echocardiography. These premature infants were admitted to the neonatal intensive care unit in our hospital without severe asphyxia (mean Apgar score = 7 at 1 minute, 9 at 5 minutes after birth) or any clinical signs of shock at birth. The infants were studied between the 12th and 19th day of age. The ductus arteriosus had closed spontaneously before the examination in all cases. None of the infants had a clinically significant patent foramen ovale nor a dilatation of the right atrium or ventricle.

Twenty-two term newborn infants were similarly studied between the ages of 6 and 9 days. All subjects had (1) a gestational age of >36 weeks, (2) an uncomplicated pregnancy and delivery, (3) no known systemic disease, and (4) a normal cardiac examination by two-dimensional echocardiography.

Five patients who presented with left ventricular dysfunction or low output state due to a variety of causes were studied. The causes of left ventricular dysfunction were septic shock (1 patient), pulmonary bleeding (2 patients), renal failure (1 patient), and severe myocardial ischemia (1 patient).
Echocardiography

Studies were performed on the subjects in a quiet, resting, and unsedated state. By means of high-speed (100 mm/sec) hard-copy, two-dimensional echocardiography and M-mode recordings of the left ventricular minor axis were obtained with an Aloka SSD 2200 ultrasonoscope with a 5.0 or 7.5 MHz transducer. Electrocardiogram, indirect axillary pulse tracing, phonocardiogram, and peripheral blood pressure were simultaneously recorded. Blood pressure was obtained from an automatched blood pressure recorder (BP-103N; Colin Medical Instruments, Tokyo). To evaluate the circularity of the left ventricle, we measured the internal diameter from the anterior wall to the inferior wall of the left ventricle and that from the midseptum to the lateral wall of the left ventricle at the papillary muscle level in the short axis view from the parasternal position on a frozen frame. The ratio of these two diameters was calculated as the circularity index.

The M-mode measurements were performed according to the recommendations of American Society of Echocardiography (Sahn et al. 1978). We measured the left ventricular end-diastolic dimension, left ventricular end-systolic dimension, and end-systolic wall thickness. End-systole was defined as the point of the first component of the second heart sound. Left ventricular ejection time (ETc) was derived from the pulse trace and was adjusted to heart rate of 60 beats per minutes (bpm) by dividing by the square of the RR interval. The left ventricular shortening fraction was calculated as (end-diastolic dimension-end-systolic dimension)/end-diastolic dimension. The mVcfc was calculated as the left ventricular shortening fraction divided by the ETc. The ESS was measured by assigning the systolic blood pressure to the peak and diastolic pressure to the nadir of the trace with a subsequent linear interpolation to the level of the dicrotic notch. ESS was calculated according to Grossman et al. (1975): ESS = 1.35•ESP•ES/4•PWS (1 + PWS/ES), where ESP = end-systole pressure, LVES = end-systolic dimension, and PWS = posterior wall thickness at end-systole. Peak systolic wall stress (PSS) = 1.35•SP•ES/4•PWS (1 + PWS/ES), where SP = peak systolic pressure.

Statistics

All data are expressed as the mean±s.d. Relationships between ESS, PSS, and mVcfc were analyzed by linear regression analysis. Regression coefficients (slopes) were tested by t-test of significance.

Results

There were no significant differences between perpendicular diameters in systole (0.94 vs. 0.90) and diastole (1.32 vs. 1.28) in the preterm infants. End-systolic and end-diastolic circularity indexes were 1.04±0.06 and 1.03±0.04. In the fullterm infants, there were no significant differences between perpendicular
Fig. 1. Relation of end-systolic wall stress and peak systolic wall stress. \( Y = 5.19 + 1.04X, \ r = 0.98, \ p < 0.01. \)

Fig. 2. Plots of rate-corrected mean fiber shortening fraction (mVcfe) and end-systolic wall stress (●, \( Y = 1.58 - 0.012X; \ r = -0.78, \ p < 0.01 \)) or peak systolic wall stress (○, \( Y = 1.60 - 0.011X; \ r = -0.75, \ p < 0.01 \)).

diameters in systole (1.21 vs. 1.19) and diastole (1.75 vs. 1.72); end-systolic and end-diastolic circularity indexes were 1.02 ± 0.04 and 1.02 ± 0.05. Left ventricular shape was found to be circular in end-systole and end-diastole.

The mean peak systolic and end-systolic pressures were 66 ± 11 mm and 57 ± 11 mmHg, respectively. The peak wall stress and ESS were 49 ± 15 mmHg and 42 ± 14 mmHg, respectively. The average mVcfe was 1.09 ± 0.21 circ/sec. The relation of the ESS and PSS is very close (Fig. 1). The plot of the mVcfe for both ESS and PSS is shown in Fig. 2. The mean slope of mVcfe = −0.012 end-systolic
Fig. 3. Relation of end-systolic wall stress and peak systolic wall stress. 
\[ Y = -1.39 + 1.19X, \quad r = 0.99, \quad p < 0.01. \]

Fig. 4. Relation between difference in PSS and end-systolic wall stress. 
Difference in PSS = actual PSS - predictive PSS obtained from the regression line of the relationship between PSS and end-systolic wall stress in normal infants.

wall stress + 1.58 is virtually identical to that of \( \mathrm{mVcfc} = -0.011 \ \mathrm{PSS} + 1.61 \), with no difference in regression coefficients.

The relation of ESS and PSS for 5 patients with left ventricular dysfunction is very close (Fig. 3). The PSS in 5 patients are predicted from the relation of ESS and PSS (PSS = 5.91 ± 1.04 ESS) for 45 infants. The difference in PSS (actual PSS-predicted PSS) in 5 patients was \( 2 \pm 5 \ \text{g/m}^2 \) (mean ± s.d.). Fig. 4 shows the relationship between the differences in PSS and ESS in 5 patients.
Discussion

To estimate left ventricular contractile state noninvasively, the relation between mVcfc and ESS has been frequently used. However, there are many technical difficulties in determining the mVcfc-ESS relation in neonates and preterm infants. To record satisfactorily carotid or axillary pulse tracing in these subjects requires their patience and cooperation, additional technical equipment, which may be impractical in infants.

In this study, the mean slope and y-intercept of the mVcfc-ESS are greater than those in children published previously (Colan et al. 1984; Franklin et al. 1990) but are very similar to those in both premature infants (mVcfc = -0.0112 ESS + 1.45) (Igarashi et al. 1994) and infants < 6 months (mVcfc = -0.0121 ESS + 1.59) (Kimball et al. 1991), confirming the uniformity of this method. We also demonstrated the close relationships between the end-systolic and peak systolic pressure and between the stress at end-systole and peak systole, which are consistent with previous studies (Nivatpumin et al. 1979; Reichek et al. 1982; Ginzton et al. 1984). Furthermore, the relation of the mVcfc-PSS in the present study is identical to that of the mVcfc-ESS with no difference in their regression coefficients, as reported in older children previously (Reichek et al. 1982). Our findings suggest that the use of the mVcfc-PSS relation would be more practical as a simple method of assessing left ventricular contractility in premature infants. Although the ability of the stress-velocity relation to adequately detect left ventricular systolic dysfunction was questioned (Roman et al. 1989), the purpose of this study was not to validate the theoretical aspects of the test but to demonstrate the similarity of stress at peak systole to that at end-systole.

The rationale for the use of the PSS have been described in a previous study (Sandor et al. 1992). The ESS was taken to mean stress at end-ejection when the aortic valve closures indicated by the dicrotic notch or by echocardiographic aortic valve closure (Colan et al. 1984; Franklin et al. 1990). This point in time does not signify mechanical end-systole. At the point when the rapid negative deflection begins in the first derivative of left ventricular pressure tracing, the ventricular pressure is 7 to 8 mmHg higher than that at which the aortic dicrotic notch occurs. Thus, the mechanical end-systole is temporally not the same as end-systole, indicated by end-ejection, and has a higher pressure, and peak systolic pressure has been suggested to be closer to true end-systole than was previously estimated using end-ejection as end-systole (Sandor et al. 1992). The second element of the noninvasive estimation of the stress-velocity relation involves calculation of the mVcfc, which in turn requires measurement of the left ventricular ejection time. Although we used the indirect pulse tracing method for this measurement as reported originally (Colan et al. 1984), it can be easily determined from M-mode echocardiographic recordings of aortic valve motion (Hirschfeld et al. 1975). Thus, all of the data for estimation of myocardial
contractility can be obtained from M-mode recordings. This method without additional equipment, technical support and time may be useful as a practical alternative for determining the stress-velocity relation in all subjects including premature infants.

In conclusion, the relation of mVvfc-PSS correlates well with the relation using the ESS and can be used as a simple method of assessing left ventricular contractility.

References


