JACC Vol. 4, No. 5
November 1984:1021–7

Noninvasive Estimation of Peak Pulmonary Artery Pressure by M-Mode Echocardiography

J. GEOFFREY STEVENSON, MD, FACC, ISAMU KAWABORI, MD,
WARREN G. GUNTHEROTH, MD, FACC

Seattle, Washington

In an attempt to predict peak pulmonary artery pressure from routine M-mode echocardiographic tracings, 95 infants and children with congenital heart disease were examined. Following the Burstin method for prediction of peak pulmonary artery pressure, which was originally based on the phonocardiogram and jugular phlebogram, M-mode echocardiography was used to measure the interval from pulmonary valve closure to tricuspid valve opening, namely, the period of isovolumic diastole. The measured interval was plotted on a modified table relating the interval, heart rate and predicted peak pulmonary artery pressure.

The peak pulmonary artery pressure predicted by echocardiography was compared with that measured at cardiac catheterization. The correlation between predicted and actual peak pulmonary artery pressure was good ($r = 0.86$) for routine studies with the patient in the nonsedated state. All patients with a predicted peak pressure less than 40 mm Hg were found at catheterization to have a pressure less than 40 mm Hg. The correlation was better ($r = 0.96$) when comparing predictions made from the echocardiogram obtained while the patient was sedated for catheterization. Prediction of the magnitude of elevation of peak pressure was especially good when prediction and measurement were nearly simultaneous. Predictions were less accurate in the presence of tachycardia at rates of more than 155 beats/min. The method for estimating peak pulmonary artery pressure from M-mode echocardiographic tracings is reliable, relatively simple and clinically useful.

Pulmonary hypertension is a significant complicating feature of many forms of heart disease. Knowledge of its presence and severity is an important consideration in various medical and surgical interventions. Noninvasive detection of pulmonary hypertension using a variety of techniques and observations has been reported. With echocardiography, early pulmonary valve closure with mid-systolic closure motion is commonly noted in the presence of severe pulmonary hypertension (1); however, one would like to be able to detect the elevation of pulmonary pressure at an earlier stage. Echocardiographic measurement of right-sided systolic time intervals has been reported (1–4) to be useful for detecting pulmonary hypertension. Our experience with that technique and that of Silverman et al. (5) have been disappointing for prediction of pulmonary pressure in individual patients.

From the Department of Pediatrics, Division of Pediatric Cardiology, University of Washington School of Medicine and Division of Cardiology, Children's Orthopedic Hospital, Seattle, Washington. This work was presented in part at the 31st Scientific Sessions of the American College of Cardiology, Atlanta, Georgia, April 1982. Manuscript received February 22, 1984; revised manuscript received June 12, 1984, accepted June 22, 1984.

Address for reprints: J. Geoffrey Stevenson, MD, Pediatrics RD-20, University of Washington, Seattle, Washington 98195.

In 1967, Burstin (6) reported a method for determining peak pulmonary artery pressure from external graphic recordings. Using a jugular phlebogram to determine tricuspid opening and a phonocardiogram to determine pulmonary valve closure, he measured the interval from pulmonary valve closure to tricuspid valve opening (that is, the isovolumic relaxation period of the right ventricle). Burstin also devised a nomogram that accurately related heart rate and the measured interval to peak pulmonary artery pressure.

In 1981, Hatle et al. (7) reported a modification of the Burstin technique. They used continuous wave Doppler ultrasound to record flow through the pulmonary and tricuspid valves. From the flow velocity waveforms, the interval from the cessation of pulmonary valve prograde flow (pulmonary valve closure) to the beginning of the tricuspid valve flow waveform (tricuspid valve opening) was measured. Using Burstin's nomogram, the method was accurate in the prediction of peak pulmonary artery pressure. The patients in that series from Norway were mostly adults. The continuous wave Doppler instrumentation, while increasing in popularity, is not now widely available in most cardiac centers. We sought to apply the important observations of Burstin and the modifications of Hatle et al. in a prospective assessment of peak pulmonary artery pressure in pediatric

0735-1097/84/$3.00

JACC Vol. 4, No. 5
November 1984:1021–7

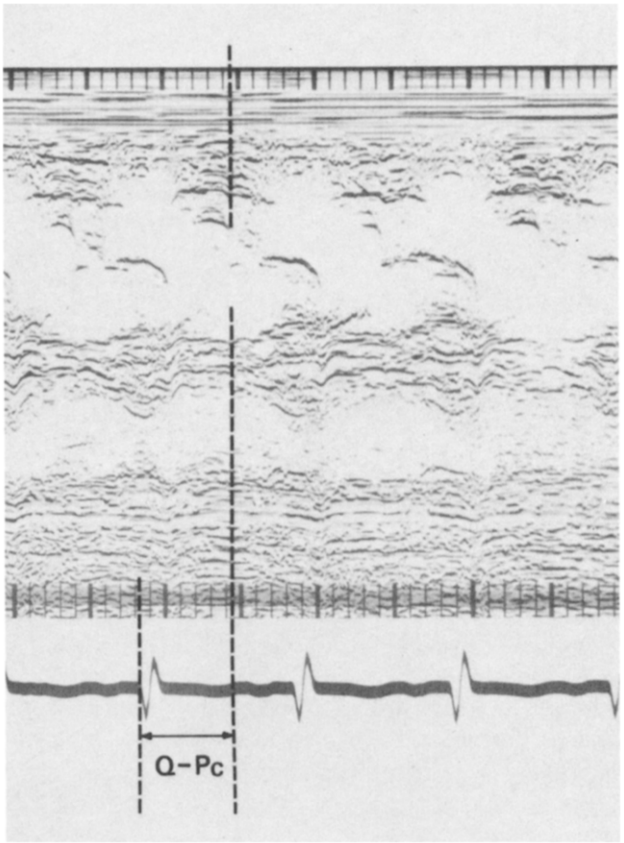


Figure 1. M-mode echographic recording of the pulmonary valve. The electrocardiogram is at the **bottom** of the figure. The **short dotted line** is drawn at the onset of the Q wave of the electrocardiogram. The **long dotted line** is drawn at that point where the pulmonary valve leaflets can be seen to coapt in diastole. Note that the diastolic coaptation occurs considerably before the recording of the densest diastolic pulmonary valve echo in most of the beats. The **long dotted line** transects the electrocardiogram; the interval from Q wave to pulmonary valve closure (Q-Pc) is measured.

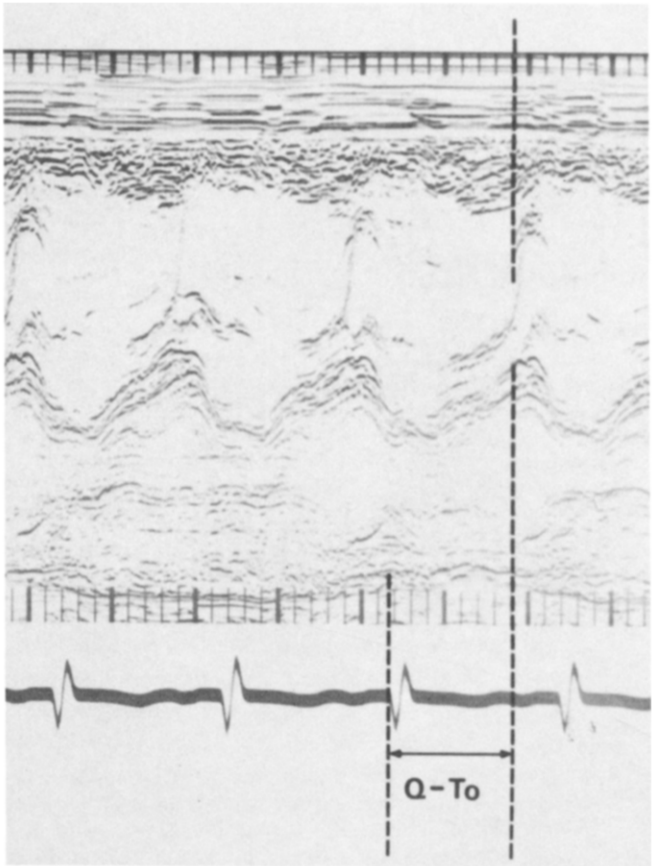


Figure 2. M-mode echographic recording of the tricuspid valve. The **short dotted line** is drawn at the Q wave of the electrocardiogram. The **longer dotted line** is drawn at the point where the larger amplitude anterior tricuspid leaflet separates from the septal leaflet echo. The interval from the Q wave to tricuspid valve opening (Q-To) is measured.

patients, using widely available M-mode echocardiography (8).

Methods

Patients. One hundred twenty-three children aged 1 day to 12 years (mean 22 months) were examined without sedation in a routine pediatric cardiac clinic or inpatient setting within 24 hours of invasive measurement of peak pulmonary artery pressure. These children underwent cardiac catheterization for a variety of defects.

Echocardiography. M-mode echocardiograms were obtained with 3 or 5 MHz transducers interfaced with an Advanced Technology Laboratories Mark 4 single crystal M-mode echocardiographic system, or with two-dimensionally directed M-mode recordings from an Advanced Technology Laboratories Mark 5 system. A simultaneous electrocardiogram was recorded in all cases. From a standard left parasternal approach, M-mode echographic tracings of the pulmonary and tricuspid valves were recorded at paper speeds of 50 to 100 mm/s, with higher speeds used in patients having a faster heart rate. Care was taken to attain maximal patient cooperation with comfortable positioning, and bottle feeding when appropriate.

The pulmonary valve was recorded from the left second or third interspace in an attempt to record a high quality pulmonary valve echo, nearly perpendicular to the valve ring. Such an approach was used to record the diastolic coaptation of the pulmonary leaflets (Fig. 1). The interval from the Q wave of the electrocardiogram to the point of leaflet coaptation was measured by a ruler to the nearest 0.5 mm, and the interval converted to milliseconds. Intervals for five beats were measured and averaged. No allowance was made for phase of respiration.

The tricuspid valve was approached from standard left parasternal windows in long- and short-axis projections. The echocardiographic plane was angled to the patient's right and somewhat anteriorly until clear separation of the anterior and septal leaflets of the tricuspid valve could be recorded. The interval from the Q wave on the electrocardiogram to

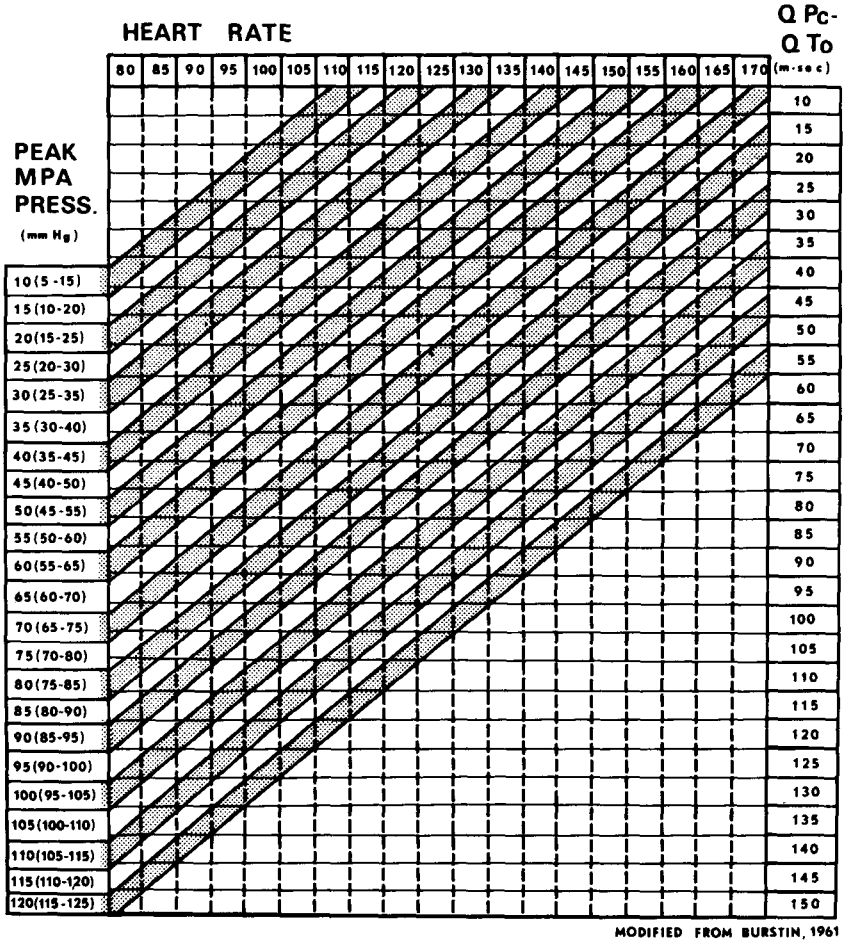


Figure 3. Modified Burstin table for prediction of peak mean pulmonary artery pressure (MPA PRESS.). Heart rate in beats per minute is shown at the **top**. The interval from pulmonary valve closure to tricuspid valve opening (QPc-QTo) in milliseconds is in the **right column**. Peak pulmonary artery pressure is in the **left column**. For a measured QPc-QTo interval of 30 ms, the intersection with 130 beats/min is found, and the diagonal followed to the left margin. The predicted peak pulmonary artery pressure would be 50 mm Hg for this interval and rate.

the onset of diastolic separation of the tricuspid leaflets was measured (Fig. 2). The interval was measured for three to five beats, and results were averaged. The pulmonary and tricuspid valve echograms were not obtained simultaneously, but were recorded in rapid sequence. Heart rate was calculated from the electrocardiogram, and only records with identical RR intervals for both tricuspid and pulmonary valve echograms were used for comparison. Adequate valve echograms with identical RR intervals were available in 95 of the 128 patients examined. These 95 patients are reported here.

In the 95 patients with adequate M-mode echographic tracings, the measured interval from pulmonary valve closure to tricuspid valve opening was plotted on a modified Burstin table. This table, reported by Burstin (6) in 1967, related the isovolumic relaxation period to pulmonary artery pressure at heart rates more commonly encountered in adults. Our modification of the Burstin table involved simple extrapolation to include the heart rates typically encountered in pediatric patients (Fig 3).

Examination in the nonsedated state. In 82 of the 95 nonsedated patients, the M-mode echographic recordings were made before catheterization. In 13 of these patients, the measurements were made shortly after catheterization. The actual pressure measured during catheterization was not known to the sonographer before the noninvasive estimation of pulmonary pressure. The noninvasive predictions of peak pulmonary artery pressure were compared with catheterization measurements.

Examination in the sedated state. Because pulmonary artery pressure varies with the level of sedation, a subseries of 33 patients was evaluated. In these patients, the M-mode echocardiographic recordings were also made while the patients were sedated for catheterization. Patients with Doppler or angiocardiographic evidence of significant tricuspid regurgitation were excluded because tricuspid regurgitation was shown by Hatle et al. (7) to affect peak pulmonary artery pressure measurements obtained by their Doppler method. In 19 of the 33 patients, the M-mode echocardiograms were recorded in the catheterization laboratory; the remaining 14 patients had echocardiograms recorded together with baseline heart rate and cuff blood pressure immediately on arrival in the recovery room with the patients still fully sedated.

Effect of heart rate. To evaluate the effect of heart rate on the accuracy of this method of predicting peak pulmonary artery pressure, the predicted pressures from 10 nonsedated patients with a heart rate greater than 155 beats/min were compared with the predictions from 10 nonsedated patients with a heart rate less than 100 beats/min.

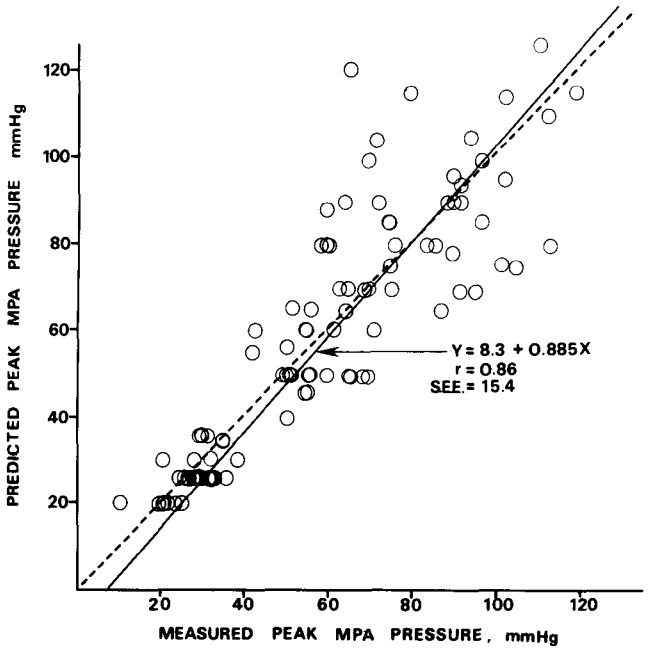


Figure 4. Results of peak mean pulmonary artery (MPA) pressure prediction in nonsedated patients. The pressures measured at catheterization are compared with those noninvasively predicted. The **dotted line** is the line of identity, and the **solid line** is the regression line drawn from the least squares method. There is generally good agreement between measured and predicted values, with some scatter at higher pressures. In patients with pressures of less than 40 mm Hg, the pressures were accurately predicted. S.E.E. = standard error of the estimate.

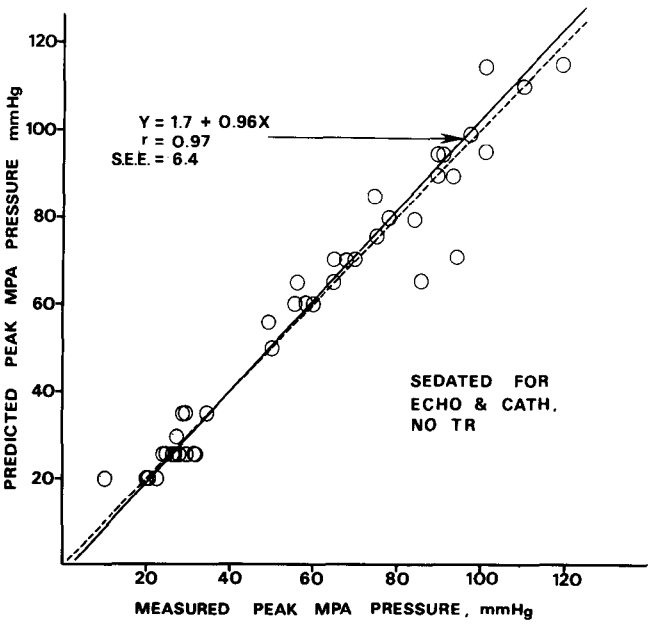


Figure 5. Results of peak mean pulmonary artery (MPA) pressure prediction in sedated patients. The pressures measured at catheterization are compared with those predicted noninvasively. The **dotted line** is the line of identity, and the **solid line** is the regression line drawn by the least squares method. There is excellent agreement between measured and predicted pressures. The r value is very high, and the standard error of the estimate (S.E.E.) is less than half that observed in the series of nonsedated patients. TR = tricuspid regurgitation.

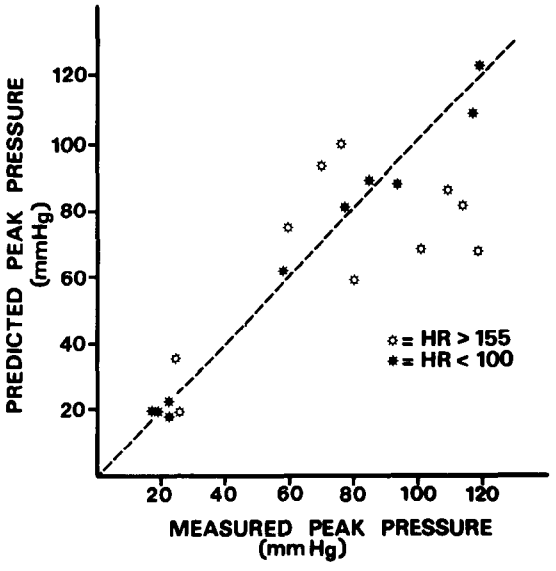
Reproducibility. An estimate of the reproducibility of the measurements and the resultant prediction of peak pressure was obtained in 10 of the 33 patients. M-mode recordings from these patients were cut, coded and shuffled for measurement by the same observer more than 1 month after completion of the study. The results of measurements made at the time of M-mode recording and those made later were compared.

Comparison with systolic time intervals. In 29 of the 33 consecutive patients, we attempted to compare the accuracy of the currently proposed method for the prediction of peak systolic pulmonary artery pressure with the accuracy of conventional right-sided systolic time intervals for the prediction of diastolic pulmonary artery pressure. Of the 29 patients, 9 could not be included for systolic time interval measurement because of the presence of right bundle branch block (3) or technical failure to record pulmonary valve opening on the M-mode echocardiogram (6).

Results

Measured and predicted peak pulmonary artery pressures (Fig. 4). In the series of 95 nonsedated patients, there was generally good correlation between measured and pre-

Figure 6. Effect of heart rate (HR) on accuracy of prediction of peak pulmonary artery pressure. The pressures measured at catheterization are compared with those noninvasively predicted. Patients with a heart rate less than 100 beats/min (**closed symbols**) fall close to the line of identity (**dotted line**). Those patients with a rate exceeding 155 beats/min (**open symbols**) show a poorer correlation between measured and predicted values.



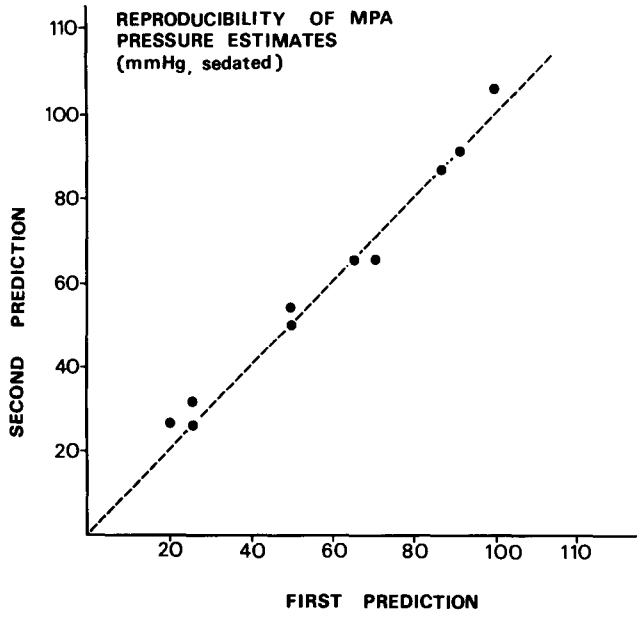


Figure 7. Reproducibility of peak mean pulmonary artery (MPA) pressure estimates in sedated patients. The results from the first pressure prediction during the period of the study are compared with those made more than 1 month after the completion of the study (second prediction). The predicted pressure estimates in each case are nearly identical.

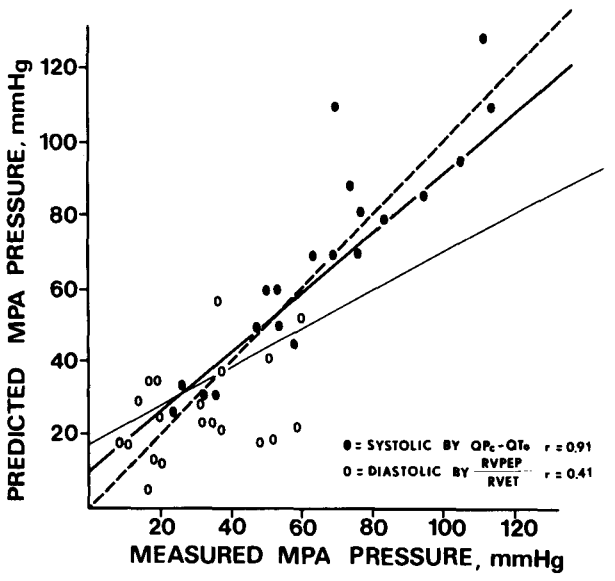


Figure 8. Accuracy of peak mean systolic pressure prediction from QPc – QTo (the interval from the Q wave to pulmonary valve closure minus the interval from the Q wave to tricuspid leaflet opening) compared with accuracy of diastolic pressure prediction from systolic time intervals. Predicted peak systolic pressures are shown in the **solid circles** and were predicted from the echocardiographic isovolumic relaxation method. The diastolic pressures predicted from systolic time intervals are shown in **open circles**. The **dotted line** is the line of identity. The **heavy black line** is the regression line for the systolic predictions (r = 0.91). The **thin black line** is the regression line for the diastolic predictions (r = 0.41). RVPEP/RVET = right ventricular preejection period divided by right ventricular ejection time.

dicted values (r = 0.86). There was some scatter at the higher pressures. The standard error of the estimate was ± 15.4 mm Hg. All patients with a predicted pressure less than 40 mm Hg had a peak pressure of less than 40 mm Hg at catheterization, and were easily distinguished from patients with a higher peak pressure.

Measurements in sedated patients (Fig. 5). The correlation between noninvasive predictions of peak pulmonary pressures and those pressures measured at catheterization in sedated patients was excellent, with little variability (r = 0.97) and a relatively small standard error of the estimate (± 6.4 mm Hg). Again, patients whose pressure was greater than 40 mm Hg were easily and accurately distinguished from those with a normal pressure predicted noninvasively.

Effect of patient heart rate on noninvasive pressure estimation (Fig. 6). In patients with a lower heart rate, the agreement between noninvasive and directly measured pressures was excellent. However, in those with a rate exceeding 155 beats/min, considerable variation between noninvasively predicted and actual pressures was noted.

The reproducibility of measurements is shown in Figure 7.

Prediction of systolic and diastolic pressure (Fig. 8). A comparison of the results of peak systolic pulmonary artery pressure prediction and the results of prediction of diastolic pressure in the same patients indicated that the correlation for systolic pressure prediction was good (r = 0.91), similar to the results for the entire series. However, the use of conventional systolic time intervals to predict diastolic pressure yielded poor results (r = 0.41).

Discussion

Our results with conventional M-mode echocardiography, along with those shown earlier by Hatle et al. (7) with Doppler recording, confirm the early observations of Burstin (6) that accurate noninvasive prediction of peak systolic pressure is possible. The value of the method described here is that it can be performed with widely available M-mode echocardiographic equipment. Ideally, it would be applied as part of a comprehensive cardiac ultrasound evaluation in which the Doppler portion of the examination would be used to screen for the presence of significant right-sided regurgitation that could affect the accuracy of pulmonary artery pressure prediction.

Methodologic problems. We were successful in obtaining the required M-mode recordings in all of the sedated subjects, but recording in the unsedated subjects proved more difficult. There were 28 unsedated patients with inadequate recordings. In most of these, suboptimal pulmo-

nary valve echoes failed to clearly demonstrate pulmonary valve closure; recording of pulmonary valve closure is probably the most difficult portion of the described method. Some patients were also excluded because the pulmonary valve and tricuspid valve recordings had been made at different heart rates. However, heart rate variability was not a problem in sedated patients. As this was a prospective study, more attention was paid to recording valve echoes than is usually observed in routine examinations. The high left parasternal approach to the pulmonary valve may not be routinely employed in many laboratories. It was clearly an aid in recording the pulmonary valve in this study. Because pulmonary artery size increases with higher pressures or larger shunts, we found recording of the pulmonary valve to be easiest in those patients in whom the question of pressure elevation was of greatest clinical concern. Patients with significant pulmonary disease and those receiving ventilator support are more difficult echocardiographic subjects in general, and they proved to be very difficult subjects for the prediction of pulmonary pressure by this method.

One must direct careful attention to the quality of the M-mode strips from which measurements are to be made. For the pulmonary valve in particular, there must be clear visualization of leaflet coaptation. On many routinely recorded pulmonary valve echograms, only a portion of the valve leaflet is shown during diastole. Leaflet coaptation frequently occurs before the emergence of the densest diastolic portion of the pulmonary valve echo (Fig. 1). At least one valve leaflet must be visualized to define the point of coaptation.

Heart rate. The effect of heart rate on the accuracy of pressure prediction was not unexpected. It may reflect simple error in measurement of the shorter intervals occurring at faster rates, or could result from variability in isovolumic relaxation at faster rates. Even though there was considerable variation between predicted and actual pressure measurements at fast heart rates, the method proved clinically useful in the prediction of the presence of pulmonary hypertension.

Role of sedation. That pressure predictions in the unsedated patients varied from the values measured at catheterization is not surprising because peak pressure will vary with activity and sedation. However, one should be able to differentiate those patients with an abnormal pressure from those with a normal pressure, even if activity levels differ somewhat. In this series, the method described was successful in predicting the presence of significantly elevated pulmonary pressure.

Peak systolic pressure. Great clinical concern surrounds the detection of pulmonary hypertension and although this method appears accurate in its detection and quantification, it predicts only the peak systolic pressure. Thus, it does not differentiate between patients with "hyperkinetic" pulmonary hypertension who have a relatively low pulmonary resistance, and those with high diastolic and mean pressures and a high calculated resistance. In some cases, the remaining portions of a comprehensive cardiac ultrasound evaluation may supply information useful in that differential.

Other methods. Before our evaluation of this method, we attempted prediction of pulmonary artery pressures from systolic time intervals and had poor results (Fig. 8). We frequently find it more difficult to record a definite point of pulmonary valve opening (6 of 29 patients in the subseries in this study) than to record the point of pulmonary valve closure. Systolic motion of the pulmonary valve ring may be a factor. With the systolic time interval methods, difficulty is commonly encountered in clearly defining the Q wave of the electrocardiogram because of variability in the inscription of the Q wave in various leads and varying QRS configuration from patient to patient. The isovolumic relaxation method described here does not require measurement from a specific point of the electrocardiogram, just measurement from the same point of the electrocardiogram for each valve. Our disappointment with conventional systolic time intervals, which may stem from these and other factors, is not unique. Silverman et al. (5) concluded that the ratio of right ventricular preejection period to right ventricular ejection time was insufficiently accurate to predict pressures in individual patients with congenital heart disease. An additional limitation is encountered in the presence of intraventricular conduction delays; right bundle branch block is commonly found in pediatric patients and prevents one from attempting pulmonary artery pressure predictions from conventional systolic time intervals.

Had we been successful in our experience with systolic time interval pressure prediction, the result would have predicted diastolic or mean pulmonary artery pressure. Because pulmonary artery diastolic pressure approximates the wedge pressure in normal subjects (9), we find greater utility in a method that predicts peak systolic pressure.

Conventional systolic time intervals are not the only approaches to the prediction of pulmonary artery pressure. Johnson et al. (4) reported a method for the prediction of pulmonary artery diastolic pressure based on echocardiographic measurement of right ventricular isovolumic contraction time. The method is unaffected by the presence of right bundle branch block and predicts diastolic pressure, but it has not proved useful in accurate pressure prediction in individual adult patients (10). We did not test this method in our series.

Implications. The method we described, using widely available M-mode echocardiography, appears to be accurate for the prediction of peak pulmonary artery pressure in children having a variety of defects. The method requires excellent echocardiographic technique and attention to detail. Patient cooperation is of great importance, and some subjects may require sedation for noninvasive pressure esti-

mates if the absolute magnitude of pressure elevation is of critical importance.

Finally, one might reasonably inquire why this method should work. The method is based on the assumption that the rate of relaxation of the right ventricle is essentially linear. At a constant rate of relaxation, a longer time will elapse if the right ventricular pressure decreases from a high level than if it decreases from a lower level. That valve motion abnormalities, such as those associated with significant tricuspid regurgitation, can influence the accuracy of the method has been pointed out by Hatle et al. (7). Additionally, right ventricular thickness, scarring and function could reasonably be expected to affect the rate of relaxation. Although not formally evaluated in this series, it is our impression that pulmonary pressure has been overestimated in several patients having extreme degrees of right ventricular hypertrophy. A stiff right ventricle would not be expected to relax at the same rate as a ventricle exposed to pressure for shorter periods.

We gratefully acknowledge the cooperation of the catheterization laboratory technicians, anesthesiology staff and recovery room nurses which allowed us to make the measurements on sedated patients.

References

1. Weyman AE, Dillon JC, Feigenbaum H, Chang S. Echocardiographic patterns of pulmonary valve motion with pulmonary hypertension. Circulation 1974;50:905–10.
2. Hirschfeld S, Meyer RA, Schwartz DC, Korfhagen J, Kaplan S. The echocardiographic assessment of pulmonary artery pressure and pulmonary vascular resistance. Circulation 1975;52:642–50.
3. Riggs T, Hirschfeld S, Borkat G, Knoke J, Liebman J. Assessment of the pulmonary vascular bed by echocardiographic right ventricular systolic time intervals. Circulation 1978;57:939–47.
4. Johnson GL, Meyer RA, Korfhagen J, Schwartz DC, Kaplan S. Echocardiographic assessment of pulmonary arterial pressure in children with complete right bundle branch block. Am J Cardiol 1978;41:1264–9.
5. Silverman NH, Snider AR, Rudolph AM. Evaluation of pulmonary hypertension by M-mode echocardiography in children with ventricular septal defect. Circulation 1980;61:1125–32.
6. Burstin L. Determination of pressure in the pulmonary artery by external graphic recordings. Br Heart J 1967;29:396–404.
7. Hatle L, Angelsen BAJ, Tromsdal A. Noninvasive estimation of pulmonary artery systolic pressure with Doppler ultrasound. Br Heart J 1981;45:157–65.
8. Stevenson JG, Kawabori I, Guntheroth WG. Noninvasive estimation of peak pulmonary artery pressure from M-mode echocardiography (abstr). Am J Cardiol 1982;49:963.
9. Otis AB. Summary of values useful in pulmonary physiology: man (Table 46). In: Altman PL, Ditmer DS, eds. Respiration and Circulation. Bethesda: Fed Am Soc Exp Biol 1971:100.
10. Mills P, Amara I, McLaurin LP, Craige E. Noninvasive assessment of pulmonary hypertension from isovolumic contraction time. Am J Cardiol 1980;46:272–6.