Comparison of Three Doppler Ultrasound Methods in the Prediction of Pulmonary Artery Pressure

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Pulmonary artery pressure was noninvasively estimated by three Doppler echocardiographic methods in 50 consecutive patients undergoing cardiac catheterization. First, a systolic transtricuspid gradient was calculated from Doppler-detected tricuspid regurgitation; clinical jugular venous pressure or a fixed value of 14 mm Hg was added to yield systolic pulmonary artery pressure. Second, acceleration time from pulmonary flow analysis was used in a regression equation to derive mean pulmonary artery pressure. Third, right ventricular isovolumic relaxation time was calculated from Doppler-determined pulmonary valve closure and tricuspid valve opening; systolic pulmonary artery pressure was then derived from a nomogram.

In 48 patients (96%) at least one of the methods could be employed. A tricuspid pressure gradient, obtained in 36 patients (72%), provided reliable prediction of systolic pulmonary artery pressure. The prediction was superior when 14 mm Hg rather than estimated jugular venous pressure was used to account for right atrial pressure. In 44 patients (88%), pulmonary flow was analyzed. Prediction of mean pulmonary artery pressure was unsatisfactory ($r = 0.65$) but improved ($r = 0.85$) when only patients with a heart rate between 60 and 100 beats/min were considered. The effect of correcting pulmonary flow indexes for heart rate was examined by correlating different flow indexes before and after correction for heart rate. There was a good correlation between corrected acceleration time and either systolic ($r = -0.85$) or mean ($r = -0.83$) pulmonary artery pressure. Because of a high incidence of arrhythmia, right ventricular relaxation time could be determined in only 11 patients (22%).

Noninvasive prediction of pulmonary artery pressure is feasible in most patients. Among the three methods, tricuspid gradient measurement seems to be the most useful and practical. Heart rate correction may improve the accuracy of using acceleration time in predicting pulmonary artery pressure; Doppler-determined right ventricular relaxation time seems to be of limited usefulness.

The introduction of Doppler echocardiography has opened new avenues to assess pulmonary artery pressure noninvasively. Measurement of the transtricuspid pressure gradient from Doppler-detected tricuspid regurgitation velocity allows estimation of right ventricular systolic pressure and thereby the systolic pulmonary pressure (13–16). Various indexes of pulmonary flow velocity correlate with pulmonary artery pressure (17–22). The right ventricular isovolumic relaxation time also has been shown to be a predictor of pulmonary artery pressure (12,23,24). However, to date there have been no reports comparing these noninvasive Doppler methods in the same patient.

We evaluated three Doppler methods (tricuspid regurgitation velocity, pulmonary flow indexes and right ventricular relaxation time) for estimating pulmonary artery pressure in patients undergoing right heart catheterization.
We attempted to assess the clinical usefulness of these three methods for the prediction of pulmonary artery pressure.

**Methods**

**Study patients.** The study group consisted of 50 consecutive patients who underwent clinically indicated right heart catheterization. Patients with right ventricular outflow obstruction were excluded. There were 23 male and 27 female patients with a mean age of 49 years (range 1 to 82). Six patients were less than 2 years of age. The clinical indication for catheterization was primary pulmonary hypertension in 5 patients, mitral valve disease in 12, aortic valve disease in 7, secundum atrial septal defect in 4, constrictive pericarditis in 4, dilated cardiomyopathy in 4, ischemic heart disease in 6 and other conditions in 8. Fourteen patients had clinical evidence of pulmonary hypertension (1–3), 8 had clinical tricuspid regurgitation and none had clinical pulmonary regurgitation. Thirty-two patients had sinus rhythm, 15 had atrial fibrillation, 1 had supraventricular tachycardia and 2 had paced rhythm.

**Doppler Examinations**

**Tricuspid regurgitation velocity.** An Irex model 3B echocardiograph equipped with a 2.0 MHz nonimaging transducer (Pedof, Vingmed) and a combined 2.5 MHz two-dimensional imaging/Doppler transducer were used for continuous wave Doppler interrogation of tricuspid regurgitation. The Doppler study was performed simultaneously with hemodynamic measurements of right-sided pressures. The techniques of securing the spectral signal of tricuspid regurgitation have been described (13–15). Doppler recordings were obtained from apical, parasternal and subcostal positions. The tricuspid regurgitation signal moved away from the transducer and consisted of a relatively dense high velocity spectral representation. Systematic search for the Doppler signal of tricuspid regurgitation was performed to achieve optimal recording, which consisted of highest maximal velocity with a distinct envelope on the spectral display. No correction was used to compensate for any presumed angle between the ultrasound beam and the direction of maximal velocity flow. The modified Bernoulli equation was employed to derive a systolic transtricuspid gradient that equals \( 4v^2 \), in which \( v \) is the maximal regurgitant velocity in meters per second.

*The right ventricular systolic pressure* was calculated by adding the jugular venous pressure to the transtricuspid gradient or by adding 14 mm Hg to the transtricuspid gradient, because we have shown (14) that adding 14 mm Hg provided a good linear correlation between the predicted and the measured values. These values were then correlated with the simultaneous measurement of right ventricular systolic pressure, which equals pulmonary systolic pressure in the absence of pulmonary stenosis.

**Pulmonary flow indexes.** Both Irex model 3B and Advanced Technology Laboratories Mark V duplex systems were utilized for the studies of pulmonary flow velocity. Most of the studies were obtained using the pulsed wave Doppler technique with a 3 MHz transducer. The second or third intercostal space along the left sternal border was used, with the Doppler sample volume positioned just proximal to the pulmonary valve in the center of the right ventricular outflow tract (17–19). The angulation of the transducer was adjusted to obtain optimal audio and visual Doppler spectral signals. In two patients, a subcostal transducer position provided the best signal. Angle correction was not used. The studies were performed with simultaneous hemodynamic measurements in 25 patients and within 30 minutes after catheterization in the remaining 25 patients.

*On the Doppler pulmonary flow contour*, the A wave (19) and mid-systolic notching (17) were looked for. Quantitative analysis of the pulmonary velocity contour provided the following indexes: preejection period, acceleration time and right ventricular ejection time (19). Five consecutive beats with clearly defined envelopes were averaged. Correction for different heart rates was performed by dividing the time indexes by the square root of the cycle length (that is, RR interval) (25). This was performed because of the wide range of heart rates (38 to 180/min) in our patients. An acceleration time of 100 ms in a patient with a heart rate of 140/min cannot be equated with the same interval in a patient with a heart rate of 50/min.

*We used the regression equation proposed by Mahan et al. (20), which involves acceleration time (ACT), to predict mean pulmonary artery pressure: mean pulmonary artery pressure (mm Hg) = 79 − 0.45 × ACT.* We tested the predictive value of this regression equation in all patients and in patients with a heart rate between 60 and 100 beats/min.

**Right ventricular isovolumic relaxation time.** To obtain this variable, that is, the interval from pulmonary valve closure to tricuspid valve opening, the interval from the Q wave of the electrocardiogram to the end of the pulmonary flow contour was measured in milliseconds. This value was then subtracted from the value for the interval from the Q wave to the end of the tricuspid regurgitation Doppler signal (Fig. 1). Five beats of similar cycle lengths were averaged. Predicted pulmonary artery pressure was obtained by using the nomogram constructed by Burstin (23).

**Cardiac catheterization.** The catheterization technique and hemodynamic measurement methods for our laboratory have been described (14). Right-sided pressures were determined using fluid-filled catheters connected to strain gauge pressure transducers (Gould P23D).

**Statistical analysis.** All values were expressed as mean ± SD. Pulmonary artery pressure correlation was performed using linear regression by the least-squares method.
Results

Fourteen patients had a normal pulmonary pressure (peak systolic pulmonary pressure <30 mm Hg), 15 had a peak systolic pulmonary pressure between 31 and 50 mm Hg and 21 had a peak systolic pulmonary pressure >50 mm Hg.

Tricuspid regurgitation velocity. Velocity signals of tricuspid regurgitation were analyzable in 36 (72%) of the 50 patients. These included 8 (57%) of the 14 patients with normal and 28 (78%) of the 36 with elevated pulmonary artery pressure. The tricuspid regurgitation velocities ranged from 2.0 to 4.5 m/s (mean 2.9). In three patients with Doppler-detected tricuspid regurgitation, a clinical estimate of jugular venous pressure was not obtained. Using either a fixed value of 14 mm Hg (14) or the clinical estimate of jugular venous pressure to account for right atrial pressure provided a good prediction of systolic pulmonary artery pressure ($r = 0.87$ and 0.89, respectively) (Fig. 2). However, the fixed value method was more satisfactory because its regression line closely approximated the identity function. The mean differences of these two methods were respectively, 0.96 and 8.66 mm Hg ($p < 0.05$).

Pulmonary flow indexes. The pulmonary flow velocity contour was obtained in 44 patients (88%); 12 of the 44 did not have Doppler-detected tricuspid regurgitation. Thus, in 48 (96%) of the 50 patients, one or both Doppler flow signals were obtained. Elevated pulmonary artery pressure was present in five of the six patients with an inadequate pulmonary flow signal. In the 44 patients with an analyzable pulmonary flow contour, a pre-systolic $A$ wave was clearly evident in only 3 of the 13 patients with normal pulmonary pressure, whereas mid-systolic notching was present in 8 of the 31
patients with pulmonary hypertension (mean pulmonary systolic pressure 64 mm Hg).

Using the equation proposed by Mahan et al. (20), we correlated the predicted mean pulmonary pressures with the measured values and found a relatively weak correlation (r = 0.66). The correlation coefficient improved if patients with a heart rate <60 beats/min or those with a rate > 100 beats/min were excluded (r = 0.85) (Fig. 3).

There was an inverse relation between acceleration time and systolic pulmonary artery pressure (r = -0.65). The correlation coefficient improved to -0.85 when the acceleration time was corrected for heart rate (Fig. 4). No further improvement was noted by using the logarithm of the systolic pulmonary pressure. A similar correlation was present between mean pulmonary artery pressure and corrected acceleration time (r = -0.83). The presence or absence of clinical tricuspid regurgitation had no effect on the correlation coefficient. The correlation coefficients between total pulmonary resistance or pulmonary arteriolar resistance and corrected acceleration time were -0.71 and -0.64, respectively. Other pulmonary flow indexes such as prejection time, right ventricular ejection time and acceleration time index (acceleration time divided by right ventricular ejection time) correlated poorly with pulmonary pressure and resistance.

A corrected acceleration time less than 130 ms identified patients with pulmonary hypertension (Fig. 4). Only four patients with a corrected acceleration time >130 ms had elevated pulmonary pressure, and none had severe elevations of peak pulmonary pressure (range 31 to 42 mm Hg).

Right ventricular isovolumic relaxation time. Determination of the interval from pulmonary valve closure to tricuspid valve opening (Pc-To) required recording the Doppler signals of both pulmonary flow and tricuspid regurgitation in the same patient. This was possible in 32 patients; 21 of these patients had sufficient fluctuation of the RR interval because of atrial fibrillation or ventricular arrhythmia to preclude a reliable measurement of the Pc-To interval. Averaging multiple cardiac cycles did not improve the measurement. Thus, only 11 of the 50 patients had consistent measurements of this interval. In this subset of patients, the predicted pulmonary systolic pressure using Burton's nomogram (23) correlated with the measured values (r = 0.87) (Fig. 5).

Discussion

This study showed that both the transtricuspid pressure gradient and pulmonary flow indexes were useful in estimating the pulmonary artery pressure. Pulmonary artery pressure could be predicted in 48 of the 50 patients (96%) by using these two Doppler methods.

Tricuspid regurgitation velocity. A tricuspid regurgitation signal was obtained in 72% of the patients, compared with the 87% reported by Yock and Popp (13). This difference is related to at least two factors. First, all of the patients studied by Yock and Popp (13) had suspected elevation of right heart pressure and 24% had clinical tricuspid
regurgitation. We made no attempt to select patients with elevated right-sided pressures, and only 16% had clinical tricuspid regurgitation. Second, we performed Doppler studies simultaneously with cardiac catheterization, and positioning of the patient was not always optimal. This study confirms that the transtricuspid gradient derived from the tricuspid regurgitation velocity is a reliable method of estimating systolic pulmonary pressure (16). The clinical estimate of jugular venous pressure was not a reliable measure of right atrial pressure (13) and was less satisfactory than a fixed value of 14 mm Hg in predicting the pulmonary artery pressure.

Using the latter method, there is no systematic difference in systolic pulmonary artery pressure between the Doppler-derived and manometric measurements. In individual patients, considerable difference may occur. This may be related to the variability of the angle between the ultrasound beam and the blood flow. The SEE was similar to that reported in other series (13,14). With an estimated pressure of 50 mm Hg, the 95% confidence limits were 34 and 66 mm Hg. Such an estimate is probably within the bounds of clinical usefulness, because pulmonary artery pressure is a dynamic measurement and can vary by more than 30% within a 24 hour period (26).

**Pulmonary flow indexes.** In this study, 88% of the patients had an analyzable pulmonary velocity contour, similar to the 87% reported by Kitabatake et al. (17). Although the presence of A waves was suggestive of normal pressures, the sensitivity is low. Mid-systolic notching was a specific sign for significant elevation of pulmonary artery pressure. As observed by Kitabatake et al. (17), mid-systolic notching correlated with mid-systolic closure on the M-mode recording, which seems to be secondary to a mid-systolic reversal of pulmonary artery to right ventricular pressure gradient (27). The exact cause remains obscure. Tricuspid regurgitation does not seem to have a causative role, because only six of the eight patients with this feature had Doppler-detected tricuspid regurgitation. Of the remaining 28 patients with elevated pulmonary pressure but no mid-systolic notching, 22 had Doppler-detected tricuspid regurgitation.

In recent observations (11,12), preejection time failed to show a significant correlation with pulmonary pressures. Kosturakis et al. (19) found that, in their patients with congenital heart disease, preejection time correlated with both the systolic and mean pulmonary pressures but that separation of patients with normal pulmonary pressures from those with pulmonary hypertension was poor.

**Acceleration time showed good correlation with systolic and mean pulmonary pressures.** The correlation coefficient was improved when a correction for heart rate was made. Unlike Kitabatake et al. (17), we found no improvement using the logarithm of pulmonary artery pressure. We believe that heart rate correction may be important, considering the wide range of heart rates encountered in clinical practice. Correcting for the RR interval seems to be more appropriate than using the regression equation from Doppler-derived indexes without heart rate correction (20), because such an equation may be applicable only within a narrow range of heart rate.

**Using an acceleration time index,** which is acceleration time divided by right ventricular ejection time, to correct for heart rate (17,18,22) is problematic and was not a reliable predictor of pulmonary pressure in our study. This is largely due to the variability of the right ventricular ejection time. This measurement is imprecise because the termination of the pulmonary flow can be difficult to determine, compared with the onset and the peak flow (19). The right ventricular ejection time may also shorten with elevation in pulmonary artery pressure (21,24,28).

It has been suggested that significant tricuspid regurgitation leads to an earlier peak in flow velocity, that is, shorter acceleration time (29). However, data on our patients with clinical tricuspid regurgitation did not deviate significantly from the regression line, compared with data from the rest of the group (Fig. 2). Therefore, Doppler-derived corrected acceleration time could be used to predict pulmonary pressures, even in the presence of tricuspid regurgitation.

**Right ventricular isovolumic relaxation time.** Using phonocardiography and pulse tracings, Burstin (23) showed that right ventricular isovolumic relaxation time could predict systolic pulmonary artery pressure. Recently, Håtås et al. (24) incorporated Doppler echocardiography with phonocardiography to derive this interval, whereas Stevenson et al. (12) relied on M-mode recordings of the tricuspid and pulmonary valves for the determination of this interval. Combining Doppler echocardiography and phonocardiography is time consuming and difficult; furthermore, in many adults, M-mode recordings of the pulmonary valve cannot
be obtained (10). Our determination of this interval relied on Doppler examination alone, thus bypassing the technical drawbacks associated with the previous studies (12,23,24). However, because of the high incidence of arrhythmia, this approach was feasible only in a few patients (11 of 50), severely limiting its practical usefulness. This approach may be more successful when it is applied to patients who have sinus rhythm and are free of arrhythmia. Moreover, this measurement may be useful when valve motion of tricuspid and pulmonary valves can be recorded, but complete spectral signals are unobtainable.

Conclusions. We have assessed three Doppler methods of predicting pulmonary artery pressure; at least one of the methods was feasible in most patients (48 of 50). Both the tricuspid regurgitation velocity and the acceleration time from pulmonary flow analysis seem to be reliable predictors of pulmonary artery pressure. The latter method may be applicable more often, but when both methods are feasible, the former seems to be more practical because a regression equation need not be used. These two methods are therefore complementary, and both should be used in the same patient so as to enhance the precision in predicting pulmonary artery systolic pressure.

References