Continuous-wave Doppler echocardiographic detection of pulmonary regurgitation and its application to noninvasive estimation of pulmonary artery pressure

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ABSTRACT Continuous-wave Doppler echocardiography was used to estimate pulmonary artery pressures by measuring pulmonary regurgitant flow velocity in 21 patients with pulmonary hypertension (mean pulmonary artery pressure ≥ 20 mm Hg) and 24 patients without pulmonary hypertension. The pulmonary regurgitant flow velocity patterns, characterized by a rapid rise in flow velocity immediately after closure of the pulmonary valve and a gradual deceleration until the next pulmonary valve opening, were successfully obtained in 18 of the 21 patients with pulmonary hypertension and in 13 of the 24 patients without pulmonary hypertension. As pulmonary artery pressure increased, pulmonary regurgitant flow velocity became higher; the pulmonary artery-to-right ventricular pressure gradient in diastole (PG) was estimated from the pulmonary regurgitant flow velocity (V) by means of the simplified Bernoulli equation (PG = 4V²). The Doppler-determined pressure gradient at end-diastole correlated well with the catheter measurement of the pressure gradient at end-diastole (r = .94, SEE = 3 mm Hg) and with pulmonary artery end-diastolic pressure (r = .92, SEE = 4 mm Hg). The peak of Doppler-determined pressure gradient during diastole correlated well with mean pulmonary artery pressure (r = .92, SEE = 5 mm Hg). Thus continuous-wave Doppler echocardiography was useful for noninvasive estimation of pulmonary artery pressures.


PULMONARY HYPERTENSION can be assessed to some extent from abnormal findings in electrocardiograms,1 phonocardiograms,2, 3 chest roentgenograms,4 and echocardiograms5–7; however, none of these methods provides a quantitative estimate of pulmonary artery pressure. Recently, several investigators8–12 have used pulsed Doppler echocardiography to measure the flow velocity in the right ventricular outflow tract or pulmonary artery and have found that Doppler measurements of right ventricular systolic time intervals8, 9 and flow velocity pattern10–12 correlate well with pulmonary artery pressures.

Continuous-wave Doppler echocardiography may provide another accurate method for the noninvasive estimation of pulmonary artery pressures because the pressure gradient across a regurgitant valve can be estimated accurately by measuring the high flow velocity of the regurgitant jet with continuous-wave Doppler echocardiography and by applying the simplified Bernoulli equation.13–18 Recently, Hatle and Angel sen19 indicated that measurement of pulmonary regurgitant flow by continuous-wave Doppler echocardiography had a potential for estimating pulmonary artery–to–right ventricular pressure gradient. Thus, in the presence of pulmonary regurgitation, continuous-wave Doppler echocardiography might be useful for estimating not only the pulmonary artery–to–right ventricular pressure gradient but also pulmonary artery pressures. Waggoner et al.20 reported that the incidence of pulmonary regurgitation was greater in patients with pulmonary hypertension than in patients without pulmonary hypertension. Yock et al.21 observed that pulmonary regurgitation could be detected even in normal adults at an incidence of 40% by continuous-wave Doppler echocardiography. More recently, Takao et al.22 indicated that pulmonary regurgi-
tation, even though it might be physiologic and trivial, could be detected with pulsed Doppler echocardiography at a high incidence in healthy subjects. Thus a method for estimating pulmonary artery pressures based on Doppler-detected pulmonary regurgitation might have wide applicability.

The purposes of this study were to examine the detection rate of pulmonary regurgitation with continuous-wave Doppler echocardiography and to estimate pulmonary artery pressures by analyzing the flow velocity patterns of pulmonary regurgitation.

Methods

Patients. The study population consisted of 45 patients (20 men and 25 women, ages 8 to 78 years, mean age 51) who were admitted for cardiac catheterization. Cardiac catheterization was performed 18 to 24 hr after Doppler examination in 36 patients, within 1 week in five patients, and immediately after completion of the Doppler examination in four patients. Thirteen had predominant mitral valve disease, 13 had coronary artery disease, eight had congenital heart disease, five had dilated cardiomyopathy, four had primary pulmonary hypertension, one had hypertensive heart disease, and one had aortic valve disease. Thirty-six patients were in sinus rhythm and the remainder had atrial fibrillation. The mean pulmonary artery pressures ranged from 9 to 87 mm Hg. Pulmonary hypertension was defined as mean pulmonary artery pressure greater than or equal to 20 mm Hg. There were 24 patients with mean pulmonary artery pressures in the 9 to 19 mm Hg range (the group without pulmonary hypertension), 14 in the 20 to 39 mm Hg range (the group with mild pulmonary hypertension), and seven in the 40 to 87 mm Hg range (the group with severe pulmonary hypertension).

Doppler examination. The Doppler examinations were performed with a duplex Doppler echocardiograph (Toshiba SDS-21B with SSH-40A) equipped with a 2.4 MHz phased-array transducer. Doppler measurements could be performed either in the pulsed Doppler mode or in the continuous-wave Doppler mode. In the pulsed Doppler mode, any cursor line could be interrogated for Doppler sampling, and the ultrasound beam direction and the sample volume were monitored as a bright line and a spot on the line in the two-dimensional echocardiographic image. The sample volume was a cylinder with a diameter of 5 mm and a length of 2 mm at a depth of 10 cm, and the pulse repetition rate was 6 kHz. In the continuous-wave Doppler mode, the beam direction of the transmitted ultrasound was fixed and displayed as a bright dotted line in the two-dimensional echocardiographic image (figure 1). The beam direction of the received ultrasound was movable and displayed as a bright solid line in the two-dimensional echocardiographic image. In continuous-wave Doppler sampling, no specific depth gate is established, but the overlapped region of the transmitted and received beams should be most sensitively sampled. The Doppler beam has a diameter of 5 mm, and the angle between the transmitted and received beams is usually small in its clinical use; therefore, the overlapped region is long and all velocities along the lines are processed for velocity determination. Doppler signals derived from structures were minimized by a high-pass filter, and all signals were analyzed in real time by the fast-Fourier transform. The Doppler flow velocity pattern and simultaneous lead II electrocardiogram were displayed on a monitor and recorded on videotape or on a strip-chart recorder at a paper speed of 50 to 100 mm/sec. The directions of the Doppler beams could be verified frequently during the examination by briefly switching to the imaging mode.

Each patient was asked to rest in a left lateral decubitus

FIGURE 1. Continuous-wave Doppler recording and its schematic (bottom) of pulmonary regurgitant flow in a patient without pulmonary hypertension. Doppler beam directions of transmitted and received ultrasound are represented as a white dotted line and a white solid line, respectively, in the two-dimensional echocardiographic image (left). Long and short arrows in the schematic indicate the peak and end-diastolic velocities of pulmonary regurgitant flow, respectively. AO = aorta; PA = pulmonary artery; ECG = electrocardiogram.
position and to breath in a relaxed way during Doppler examination. The transducer was placed in the parasternal position to depict the pulmonary valve. The pulsed Doppler mode was used to detect pulmonary regurgitation by placing the sample volume in the right ventricular outflow tract and sweeping it back and forth behind the pulmonary valve. If pulmonary regurgitation was present, we switched the system to the continuous-wave Doppler mode and then advanced the crossing point of transmitted and received ultrasound beam lines to the level of the pulmonary orifice. We tilted the transducer slowly until the highest Doppler frequency shifts could be obtained with the aid of the audio signals and recorded the Doppler signals of pulmonary regurgitation.

**Data analysis.** The flow velocities at a peak level in early diastole and at the end-diastolic shoulder were measured from the continuous-wave Doppler recordings of pulmonary regurgitant flow. A minimum of 5 beats with the greatest representation of instantaneous maximal flow velocities were measured from the recording in which the highest instantaneous maximal flow velocities were obtained. They were averaged for the quantitative analysis. Consecutive beats were analyzed only when the signal quality of all consecutive beats was optimal. In 21 of 31 patients in whom pulmonary regurgitation was successfully detected by continuous-wave Doppler echocardiography, the angle between the ultrasound beam and the direction of flow did not seem to be zero. In these 21 patients, therefore, two methods of velocity determination were tested. With the first method, no attempt at correction of velocities for flow angle was made in any case. With the second method, the Doppler incident angle was determined relative to the average vector of the transmitted and received beams by assuming that the direction of pulmonary regurgitant flow was perpendicular to the diastolic two-dimensional echocardiographic image of the pulmonary cusps. The Doppler incident angle ranged from 25 to 40 degrees (mean 32).

The Doppler estimate of diastolic pressure gradient between the pulmonary artery and right ventricle during diastole was calculated by applying the simplified Bernoulli equation developed and validated by Holen, Hatle, and their colleagues:

$$\text{pressure gradient (mm Hg) = } 4 \times V^2$$

where $V$ is the velocity of the pulmonary regurgitant jet in meters per second.

To test the reliability of this method, we randomly selected 15 patients and determined Doppler estimates of the pulmonary artery-to-right ventricular pressure gradient at a peak level in early diastole and at the end-diastolic shoulder by one observer on two occasions (intraobserver variability). Another observer independently performed the determination for the same 15 patients (interobserver variability). The observers were blinded to each other’s results as well as to the results of cardiac catheterization. The means and standard deviations of differences between observations were $-0.7 \pm 1.8$ mm Hg (intraobserver) and $-0.4 \pm 2.8$ mm Hg (interobserver) for the peak of Doppler estimate and $0.0 \pm 1.5$ mm Hg (intraobserver) and $-0.3 \pm 1.7$ mm Hg (interobserver) for the Doppler estimate at end-diastole.

**Cardiac catheterization.** Cardiac catheterization was carried out by a standard technique. Right-sided pressure determinations were obtained with the use of fluid-filled catheters connected to a P23Db Statham strain gauge. The pulmonary artery-to-right ventricular pressure gradient was measured by withdrawing the catheter from the pulmonary artery to the right ventricle. In patients with atrial fibrillation, the pressure gradient at end-diastole was calculated as the difference between the averaged measurements of pulmonary arterial end-diastolic pressure and right ventricular end-diastolic pressure.

**Statistical analysis.** All values were expressed as mean ± SD. The significance of differences among the group without pulmonary hypertension and the groups with pulmonary hypertension was assessed by analysis of variance and a multiple comparison method. Doppler-determined pressure gradients were compared with catheter-determined pressure gradients and pressures by linear regression analyses.

**Results**

**Pulmonary regurgitant flow velocity patterns obtained by continuous-wave Doppler echocardiography.** A representative recording of pulmonary regurgitant flow velocity pattern in a patient without pulmonary hypertension is shown in figure 1. The pattern of pulmonary regurgitant flow was characterized by a rapid rise in flow velocity to a peak level just after closure of the pulmonary valve, followed in turn by a slow deceleration until the next pulmonary valve opening. Pulmonary regurgitation was detected by continuous-wave Doppler echocardiography in 13 of 24 patients without pulmonary hypertension. The pulmonary regurgitant flow velocity patterns in patients with pulmonary hypertension were similar to those in patients without pulmonary hypertension, but the velocity was higher (figures 2 and 3). Pulmonary regurgitation was detected in 11 of 14 patients with mild pulmonary hypertension and in all seven patients with severe pulmonary hypertension. Thus the detection rate of pulmonary regurgitation by continuous-wave Doppler echocardiography was higher in patients with pulmonary hypertension than in patients without pulmonary hypertension. It is noteworthy that pulmonary regurgitation was detected in all patients with severe pulmonary hypertension.

**Quantitative analysis of Doppler flow velocity patterns.** Details based on the quantitative analysis of Doppler recordings are summarized in table 1. No significant difference in heart rate was observed between the 13 patients without pulmonary hypertension and the 18 patients with pulmonary hypertension in whom Doppler recordings of pulmonary regurgitant flow were successfully obtained (78 ± 14 vs 67 ± 8 beats/min). The peaks of pulmonary regurgitant flow velocity and Doppler-determined pressure gradient were significantly greater in patients with pulmonary hypertension than in patients without pulmonary hypertension (2.3 ± 0.7 vs 1.4 ± 0.3 m/sec, *p* < .01; 23 ± 14 vs 8 ± 4 mm Hg, *p* < .01). The pulmonary regurgitant flow velocity and Doppler-determined pressure gradient at end-diastole were also greater in patients with pulmonary hypertension than in patients without pulmonary hypertension (1.8 ± 0.6 vs 1.0 ± 0.3 m/sec, *p* < .01; 15 ± 10 vs 4 ± 2 mm Hg, *p* < .01).

**Relationships between Doppler-determined pressure gradient and hemodynamic determinants.** The Doppler-
determined pressure gradient at end-diastole was compared with actual pulmonary artery-to-right ventricular pressure gradient in 31 patients in whom pulmonary regurgitant flow velocity at end-diastole was successfully measured from continuous-wave Doppler recordings (figure 4). They correlated well with each other at a correlation coefficient of .94 (p < .01, SEE = 3 mm Hg, y = 0.70 x + 1). When angle correction was...
performed, Doppler-determined pressure gradient at end-diastole showed better agreement with catheter measurements ($r = .97, p < .01, \text{SEE} = 3 \text{ mm Hg, } y = 0.91 x + 0$).

Doppler-determined pressure gradient at end-diastole also correlated well with pulmonary arterial end-diastolic pressure ($r = .92, p < .01, \text{SEE} = 4 \text{ mm Hg, } y = 0.61 x - 2$; figure 5). Angle correction did not provide a significant improvement in the correlation ($r = .95, p < .01, \text{SEE} = 4 \text{ mm Hg, } y = 0.81 x - 2$).

To directly estimate the pulmonary artery end-diastolic pressure, some estimate of right ventricular diastolic pressure should be added to the Doppler-determined pressure gradient. Thus the catheter-determined right atrial pressure was added as an estimate of right ventricular end-diastolic pressure to the Doppler-determined pressure gradient at end-diastole and then compared with pulmonary arterial end-diastolic pressure. It also correlated well with pulmonary arterial end-diastolic pressure ($r = .94, p < .01, \text{SEE} = 4 \text{ mm Hg, } y = 0.74 x + 1$; figure 6). Angle correction did not provide a significant improvement in the correlation ($r = .96, p < .01, \text{SEE} = 4 \text{ mm Hg, } y = 0.93 x - 0$).

The peak of the Doppler-determined pressure gradient was compared with mean pulmonary artery pressure in 30 patients in whom the peak of pulmonary regurgitant flow velocity was successfully measured (figure 7). They correlated well with each other ($r = .92, p < .01, \text{SEE} = 5 \text{ mm Hg, } y = 0.70 x - 2$). Angle correction did not improve the correlation coefficient or SEE ($r = .92, \text{SEE} = 7 \text{ mm Hg, } y = 0.91 x - 3$).

Discussion

In this study, we analyzed pulmonary regurgitant flow velocity patterns to estimate pulmonary artery pressure. The pulmonary regurgitant flow velocity pattern, recorded with continuous-wave Doppler echocardiography, showed a characteristic contour, as reported previously. The pattern, characterized by a rapid rise in flow velocity immediately after closure of the pulmonary valve, relatively high peak flow velocity, and a gradual deceleration until the next pulmonary valve opening, was analogous to that of aortic regurgitation. However, the peak velocity was much lower in pulmonary regurgitant flow than in aortic regurgitant flow, reflecting the smaller pressure gradient between pulmonary artery and right ventricle than between aorta and left ventricle.

A previous report showed that the presence or absence of pulmonary regurgitation might indicate the presence or absence of pulmonary hypertension. However, pulmonary regurgitation was successfully detected with continuous-wave Doppler echocardiography in about half of the patients without pulmonary hypertension in this study. This finding indicated that the presence of pulmonary regurgitation did not necessarily imply elevated pulmonary artery pressure. This is also supported by recent preliminary investigations. Takao et al. reported that pulmonary regurgitation could be detected in almost all healthy subjects by pulsed Doppler echocardiography. We could not detect pulmonary regurgitation with a detection rate as high as theirs; this may be explained by differences in patient selection, equipment, and technique. The incidence of detectable pulmonary regurgitation was not satisfactory, and the insufficient detection rate was one of the most important limitations of the study. However, further development of Doppler equipment may increase the detection rate of pulmonary regurgitation by continuous-wave Doppler echocardiography.

We showed that pulmonary regurgitant flow velocity became higher as pulmonary artery pressure increased. Miyatake et al., using pulsed Doppler echo-

### Table 1

Summary of findings obtained by cardiac catheterization and continuous-wave Doppler examination (mean ± SD)

<table>
<thead>
<tr>
<th>MPAP</th>
<th>≤19 mm Hg (n = 13)</th>
<th>20–39 mm Hg (n = 11)</th>
<th>≥40 mm Hg (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>52 ± 13</td>
<td>52 ± 20</td>
<td>50 ± 15</td>
</tr>
<tr>
<td>Sex</td>
<td>9M, 4F</td>
<td>5M, 6F</td>
<td>1M, 6F</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>13 ± 3</td>
<td>27 ± 6c</td>
<td>54 ± 15c</td>
</tr>
<tr>
<td>PASP (mm Hg)</td>
<td>23 ± 5</td>
<td>39 ± 11c</td>
<td>86 ± 20c</td>
</tr>
<tr>
<td>PAPD (mm Hg)</td>
<td>8 ± 2</td>
<td>20 ± 4c</td>
<td>39 ± 14c</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>4 ± 2</td>
<td>6 ± 2</td>
<td>9 ± 5</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>4 ± 4</td>
<td>5 ± 2</td>
<td>8 ± 7</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>67 ± 8</td>
<td>78 ± 13</td>
<td>80 ± 16</td>
</tr>
<tr>
<td>V-\text{ed (m/sec)}</td>
<td>0.9 ± 0.3</td>
<td>1.5 ± 0.4c</td>
<td>2.3 ± 0.5c</td>
</tr>
<tr>
<td>V-\text{ed (m/sec)}</td>
<td>1.0 ± 0.3</td>
<td>1.7 ± 0.4c</td>
<td>2.6 ± 0.5c</td>
</tr>
<tr>
<td>V-\text{peak (m/sec)}</td>
<td>1.4 ± 0.3b</td>
<td>1.9 ± 0.5c</td>
<td>2.9 ± 0.6c</td>
</tr>
<tr>
<td>V-\text{peak (m/sec)}</td>
<td>1.5 ± 0.3b</td>
<td>2.2 ± 0.6c</td>
<td>3.4 ± 0.6c</td>
</tr>
<tr>
<td>PG-\text{ed (mm Hg)}</td>
<td>4 ± 2</td>
<td>10 ± 5c</td>
<td>22 ± 11c</td>
</tr>
<tr>
<td>PG-\text{ed (mm Hg)}</td>
<td>4 ± 2</td>
<td>13 ± 6c</td>
<td>29 ± 13c</td>
</tr>
<tr>
<td>PG-\text{peak (mm Hg)}</td>
<td>8 ± 4b</td>
<td>16 ± 7c</td>
<td>35 ± 15c</td>
</tr>
<tr>
<td>PG-\text{peak (mm Hg)}</td>
<td>9 ± 4b</td>
<td>20 ± 10c</td>
<td>46 ± 16c</td>
</tr>
</tbody>
</table>

MPAP = mean pulmonary arterial pressure; PASP = pulmonary arterial systolic pressure; PAEDP = pulmonary arterial end-diastolic pressure; RVEDP = right ventricular end-diastolic pressure; RAP = mean right atrial pressure; HR = heart rate; V = velocity; Ed = end-diastolic; PG = Doppler-determined pressure gradient between pulmonary artery and right ventricle.

b: Value corrected for the Doppler incident angle.

c: n = 12 patients.

c: p < .01 vs group without pulmonary hypertension.
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Doppler-determined pulmonary artery-to-right ventricular pressure gradient at end-diastole (PA-RV PG ed) plotted against catheter-determined PA-RV PG ed. Closed circles represent right ventricular end-diastolic pressure (RVEDP) greater than 10 mm Hg. In the right panel, the Doppler measurement is corrected for Doppler incident angle. Angle correction provided a slight improvement in the correlation coefficient .97 and better agreement between the two measurements.

FIGURE 4. Doppler-determined pulmonary artery-to-right ventricular pressure gradient at end-diastole (PA-RV PG ed) plotted against catheter-determined PA-RV PG ed. Closed circles represent right ventricular end-diastolic pressure (RVEDP) greater than 10 mm Hg. In the right panel, the Doppler measurement is corrected for Doppler incident angle. Angle correction provided a slight improvement in the correlation coefficient .97 and better agreement between the two measurements.

Continuous-wave Doppler echocardiography, reported that pulmonary regurgitant flow patterns exhibited a wide-band spectrum of the velocity component and sustained the same velocity throughout diastole in patients with pulmonary hypertension and that the pulmonary regurgitant flow velocity peaked in early diastole and then gradually slowed in later diastole in patients without pulmonary hypertension. These findings indicated that pulmonary regurgitant flow velocity in patients with pulmonary hypertension was so high that it produced an aliasing effect and velocity ambiguity in the pulmonary regurgitant flow velocity patterns, whereas in patients without pulmonary hypertension pulmonary regurgitant flow velocity was not high enough to produce the aliasing effect. Thus their qualitative findings are comparable to ours. Continuous-wave Doppler echocardiography enabled us to quantitate pulmonary regurgitant flow velocity, thus making it possible to evaluate pulmonary hypertension quantitatively.

The greatest difficulty that we encountered in our quantitative analysis was the problem of Doppler incident angle. Since the direction of the regurgitant jet is not predictable from the anatomy of surrounding structures, velocity was not corrected for Doppler incident angle in previous studies, and Doppler beams retrospectively seemed to be aligned nearly parallel to the flow in most patients for whom valvular stenosis and tricuspid regurgitation were evaluated. The Doppier-
determined pressure gradient at end-diastole correlated well with the catheterization-determined pressure gradient but underestimated the actual value in most patients. Underestimation of the pressure gradient by the Doppler method may be caused in part by the inability to align Doppler beams parallel to the direction of the pulmonary regurgitant jet. There are other possible explanations for this systematic underestimation. There is a tendency for Doppler signals of pulmonary regurgitation to be better imaged during inspiration, when increased flow into the right heart may lead to some increase in right ventricular end-diastolic pressure (hence decreased gradient compared with the average gradient). Furthermore, high velocity dropout in the Doppler signal of pulmonary regurgitation is a frequent problem, just as it is with aortic regurgitation.

Pulmonary regurgitation is not attributed to organic changes of the pulmonary valve in most cases but occurs physiologically or secondarily. Therefore its low direction may well be perpendicular to the pulmonary valve, and we arbitrarily measured Doppler incident angle by referring to the diastolic two-dimensional echocardiographic image of the pulmonary cusps. Doppler-determined pressure gradient at end-diastole was in good agreement with the actual value when it was corrected for the Doppler incident angle. A recent

FIGURE 6. Doppler-determined pulmonary artery–to–right ventricular pressure gradient at end-diastole (PA-RV PG end) added to catheterization-determined right atrial pressure (RAP) and plotted against pulmonary arterial end-diastolic pressure. Closed circles represent right ventricular end-diastolic pressure greater than 10 mm Hg. In the right panel, the Doppler measurement is corrected for Doppler incident angle.

FIGURE 7. Peak of Doppler-determined pulmonary artery–to–right ventricular pressure gradient (PA-RV PG) during diastole plotted against mean pulmonary artery pressure. Closed circles represent right ventricular end-diastolic pressure (RVEDP) greater than 10 mm Hg. In the right panel, the Doppler measurement is corrected for Doppler incident angle, but the correlation is not improved.
preliminary study\(^3\) indicated that the development of two-dimensional Doppler "color flow mapping" might help to optimize the Doppler incident angle. Further studies are required to precisely determine the role of the Doppler incident angle. In this study, all Doppler estimates correlated well with catheter measurements, even without angle corrections, and the correlation coefficient was thought to be good enough to predict pulmonary artery pressures even without angle corrections.

Doppler-determined pressure gradient at end-diastole also correlated well with pulmonary arterial end-diastolic pressure. However, another theoretical and practical problem arose: the Doppler estimate would underestimate the actual value in patients with elevated right ventricular end-diastolic pressure. In this study, right ventricular end-diastolic pressure was greater than 10 mm Hg in three patients and greater than 15 mm Hg in one of them. In such patients, the Doppler estimate surely underestimated the pulmonary arterial end-diastolic pressure, but the difference was less than 5 mm Hg in all but one patient, who had a right ventricular end-diastolic pressure of 19 mm Hg. Special care should be taken only when right ventricular end-diastolic and/or right atrial pressures are expected to be markedly elevated. However, this problem would be solved if right ventricular end-diastolic and/or right atrial pressures were known, since the underestimation was improved by adding right atrial pressure to the Doppler-determined pressure gradient in this study. Right atrial pressure can be estimated clinically from the jugular vein.\(^{15,31}\) If jugular venous pressure were used, more accurate estimation of pulmonary arterial end-diastolic pressure could be performed noninvasively, irrespective of the level of right atrial pressure.

Pulmonary arterial end-diastolic pressure is equivalent to left atrial and left ventricular end-diastolic pressures in the absence of lesions in pulmonary vascular beds. In such patients, it is quite useful in predicting pulmonary arterial end-diastolic, left atrial, and left ventricular end-diastolic pressures. The correlations between pulmonary capillary wedge pressure and Doppler estimates were also good in this group of patients (\(r = .89\) and SEE = 3 mm Hg for the Doppler-determined pressure gradient at end-diastole; \(r = .94\) and SEE = 3 mm Hg for the pressure gradient at end-diastole added to right atrial pressure). However, in patients with abnormal pulmonary vascular beds, pulmonary end-diastolic pressure does not necessarily reflect these values, and it is more important to predict mean pulmonary artery pressure. In this study, the Doppler-determined pressure gradient at end-diastole also appeared to be useful in predicting mean pulmonary artery pressure, since the correlation coefficient between them was .94. However, it might underestimate mean pulmonary artery pressure in patients with severe pulmonary regurgitation. As pulmonary regurgitation becomes more severe, the deceleration of pulmonary regurgitant flow velocity may become greater and the velocity at end-diastole may become lower, analogous to the findings in aortic regurgitation.\(^{27,28}\)

On the other hand, the peak of the Doppler-determined pressure gradient should hardly be affected by the degree of pulmonary regurgitation, analogous to the findings in aortic regurgitation.\(^{28}\) For these reasons, we examined the relation between the peak of the Doppler-determined pressure gradient during diastole and mean pulmonary artery pressure and found a good correlation between them. Unfortunately, cardiac catheterization and Doppler studies were not performed simultaneously in this study. The mean pulmonary artery pressures could change substantially with alterations in cardiac output. This may explain a wider scatter of individual points relating mean pulmonary artery pressure to peak Doppler-determined pulmonary artery–to–right ventricular pressure gradient. However, the correlation seemed to be good enough to estimate mean pulmonary artery pressure. Thus the measurements of pulmonary regurgitant flow velocities by continuous-wave Doppler echocardiography were useful in predicting not only end-diastolic but also mean pulmonary artery pressures.

Recently, several authors\(^{10-12}\) have demonstrated that acceleration time, the time from the onset of ejection to peak velocity, and/or its ratio to right ventricular ejection time are useful in estimating mean pulmonary artery pressure. We measured the indexes from the systolic flow velocity contours in all 31 patients and examined the correlation between the Doppler indexes and mean pulmonary artery pressures (table 2). The best correlation (\(r = .91\)) was found with the Doppler estimate determined from a previously published regression equation\(^{10}\) and the ratio of acceleration time to right ventricular ejection time among these previous Doppler techniques. The correlation coefficient and SEE for the acceleration time method were comparable to those for the peak Doppler-determined pressure gradient method. Which method is preferable may depend on the quality and accessibility of the corresponding Doppler signals in each record.

Our results demonstrate that continuous-wave Doppler echocardiographic detection of pulmonary regurgitation permits the noninvasive evaluation of pulmonary hypertension. Since pulmonary regurgitation...
TABLE 2
Correlation of previously published Doppler techniques and mean pulmonary artery pressure

<table>
<thead>
<tr>
<th>Index</th>
<th>r</th>
<th>SEE</th>
<th>Slope</th>
<th>Intercept</th>
</tr>
</thead>
<tbody>
<tr>
<td>AcT</td>
<td>-.79</td>
<td>18 msec</td>
<td>-1.3</td>
<td>139</td>
</tr>
<tr>
<td>AcT&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.85</td>
<td>6 mm Hg</td>
<td>0.55</td>
<td>12</td>
</tr>
<tr>
<td>AcT/RVET</td>
<td>-.85</td>
<td>0.05</td>
<td>-0.0040</td>
<td>0.48</td>
</tr>
<tr>
<td>AcT/RVET&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.91</td>
<td>7 mm Hg</td>
<td>0.80</td>
<td>5</td>
</tr>
</tbody>
</table>

All correlation coefficients are significant at p < .01. The correlation coefficient (r), standard error of estimate (SEE), slope, and intercept for each relation to mean pulmonary artery pressure were calculated among all 31 patients. Acceleration time (AcT) and right ventricular ejection time (RVET) were measured from the continuous-wave Doppler recordings by the same previously published technique.<sup>10</sup>

<sup>a</sup>Doppler-estimated mean pulmonary artery pressures using previously published regression equations<sup>10</sup>, AcT = 10<sup>-0.0086AcT<sup>-2</sup>-<sub>1</sub></sup>, AcT/RVET = 10<sup>-2.4AcT/RVET<sup>-2</sup>-<sub>1</sub></sup>

is present in a high proportion of patients with elevated pulmonary artery pressures, this method seems promising in the evaluation of pulmonary hypertension.

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References

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