The purpose of this study was to evaluate a noninvasive method for estimation of pulmonary artery pressures in infants and children with interventricular communications. Systolic pulmonary artery pressures measured by cardiac catheterization were compared with those estimated by Doppler echocardiography. Pressure drops were measured by Doppler study (modified Bernoulli equation) and were referenced to systolic systemic arterial pressure measured by sphygmomanometry.

All 25 patients in this study had either a ventricular septal defect or a single ventricle. The systolic pulmonary artery pressure measured by cardiac catheterization ranged from 5 to 100 mm Hg (mean ± SD 43 ± 26) and that measured by Doppler echocardiography ranged from 5 to 100 mm Hg (mean 43 ± 26) (p = NS; r = 0.92; SEE = 9.9; slope = 0.92; y intercept = 4.7). Systolic pulmonary artery to aortic pressure ratio measured by cardiac catheterization ranged from 0.2 to 1.0 (mean 0.5 ± 0.3) and that measured by Doppler echocardiography ranged from 0.1 to 1.0 (mean 0.5 ± 0.3) (p = NS; r = 0.94; SEE = 0.09; slope = 0.90; y intercept = 0.04). This study demonstrates that Doppler echocardiography can closely approximate systolic pulmonary artery pressure in patients with interventricular communications.

Methods

Study patients. The study group consisted of 25 infants and children with congenital heart disease. All had either a ventricular septal defect or a single ventricle. Each had pulmonary artery pressure measured at cardiac catheterization. Doppler echocardiographic estimation of systolic pulmonary artery pressure was obtained in 20 (80%) of the patients without knowledge of pulmonary artery pressure obtained at cardiac catheterization; in all but 1 patient, Doppler measurements were obtained within 24 hours of cardiac catheterization. In the other five patients, the Doppler measurements were obtained 1, 3, 6, 9 and 19 months after catheterization. The 25 patients were aged 1 to 130 months.
Table 1. Patient Data

<table>
<thead>
<tr>
<th>Case</th>
<th>Diagnosis</th>
<th>Age (mo)</th>
<th>BP* (mm Hg)</th>
<th>Ao Vel (cm/s)</th>
<th>VSD Vel (cm/s)</th>
<th>VSD Grad (mm Hg)</th>
<th>PA Vel (cm/s)</th>
<th>PA Grad (mm Hg)</th>
<th>PAP* (mm Hg)</th>
<th>PAP/AoP</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>VSD, AS</td>
<td>22</td>
<td>80</td>
<td>160</td>
<td>440</td>
<td>77</td>
<td>125</td>
<td>0</td>
<td>13</td>
<td>0.2</td>
<td>23/88 0.3</td>
</tr>
<tr>
<td>2</td>
<td>VSD</td>
<td>15</td>
<td>90</td>
<td>120</td>
<td>420</td>
<td>71</td>
<td>110</td>
<td>0</td>
<td>19</td>
<td>0.2</td>
<td>22/122 0.2</td>
</tr>
<tr>
<td>3</td>
<td>VSD</td>
<td>9</td>
<td>90</td>
<td>85</td>
<td>300</td>
<td>36</td>
<td>185</td>
<td>14</td>
<td>48</td>
<td>0.4</td>
<td>45/85 0.5</td>
</tr>
<tr>
<td>4</td>
<td>VSD</td>
<td>25</td>
<td>78</td>
<td>80</td>
<td>300</td>
<td>36</td>
<td>100</td>
<td>0</td>
<td>42</td>
<td>0.5</td>
<td>40/105 0.4</td>
</tr>
<tr>
<td>5</td>
<td>VSD</td>
<td>2</td>
<td>80</td>
<td>120</td>
<td>160</td>
<td>10</td>
<td>140</td>
<td>0</td>
<td>60</td>
<td>0.9</td>
<td>55/95 0.6</td>
</tr>
</tbody>
</table>

[5] VSD, CoA
3 CAVC
8 CAVC
9 VSD, ARVMB
10 VSD, ARVMB
11 ToF
12 ToF
13 ToF (Blalock-Taussig)
14 l-TGA, VSD
15 d-TGA, VSD, PS
16 l-TGA, VSD, PAB
17 l-TGA, VSD, PS
18 l-TGA, VSD, PS
19 l-TGA, VSD
20 DORV, MA, PS
21 DORV, PAB
22 SV, PS
23 SV, PAB
24 SV
25 SV, PAB

Mean ± SD ± 38 ± 26 ± 0.3 ± 26 ± 0.3

*These pressures are systolic; Ao = aorta; AoP = aortic pressure; ARVMB = anomalous right ventricular muscle bundles; AS = aortic stenosis; BP = blood pressure; BT = Blalock-Taussig; CAVC = complete atrioventricular canal; CoA = coarctation of aorta; DORV = double outlet right ventricle; d-TGA = dextrotransposition of the great arteries; Grad = gradient; l-TGA = levotransposition of the great arteries; MA = mitral atresia; PA = pulmonary artery; PAB = pulmonary artery band; PAP = pulmonary artery pressure; PS = pulmonary stenosis; SubAS = subaortic stenosis; SV = single ventricle; ToF = tetralogy of Fallot; Vel = velocity; VSD = ventricular septal defect.
Doppler echocardiographic procedure. All patients were examined in the supine or left lateral decubitus position. Uncooperative patients were given chloral hydrate (50 mg/kg orally) 30 minutes before evaluation for purposes of obtaining Doppler echocardiographic evaluations under optimal conditions. Systolic systemic blood pressure was obtained by an appropriately sized cuff on the right arm with the patient supine. In young infants, a peripheral vascular continuous wave Doppler instrument was used to determine systolic systemic blood pressure during sphygmomanometry.

Doppler echocardiography was performed for each patient with a Honeywell ultrasonoscope. Initially, a standard real-time two-dimensional imaging study was done to assess anatomy. A standard pulsed Doppler examination (5) was then performed to assess velocities distal to each valve and across the aortic and pulmonary outflow tracts, ventricular septal defect and right ventricular muscle bundles when present. If velocities exceeded the pulsed Doppler Nyquist limit, a high pulse repetition frequency system (Honeywell) or continuous wave Doppler system (Irex) was employed. Both were capable of recording velocities of 7 m/s or higher.

Aortic outflow tract Doppler interrogation was obtained principally from the suprasternal notch. In some patients, interrogation was performed from an apical four chamber or subcostal short-axis view.

Pulmonary outflow tract Doppler interrogation was obtained from the parasternal short-axis, subcostal short-axis or, in some patients, the suprasternal notch plane. When continuous wave Doppler echocardiography was used, the pulmonary outflow tract was interrogated "blindly" from the midclavicular line, parallel to the nipple, with the transducer aimed superiorly, or from the subcostal plane with the transducer aimed into the right ventricular outflow tract.

Ventricular septal defects were interrogated perpendicularly to the septum from a parasternal long-axis plane or, in some patients, the suprasternal notch plane. Maximal transventricular septal defect velocities were obtained by finding the precordial location of a systolic thrill and interrogating from that site. Because the interventricular septum lies perpendicularly to the coronal plane in patients with l-transposition, interrogation in this group was accomplished by placing the transducer along the left sternal border and aiming laterally.

Calculation of systolic pulmonary pressure. Figure 1 demonstrates the method used to calculate pulmonary artery pressure. The highest velocities for any single beat were chosen for measurement. If peak Doppler velocities were greater than 150 cm/s, a jet lesion was considered and the pressure difference across either the semilunar valves or the interventricular septum was calculated.

Systolic systemic arterial pressure was measured in the right arm. This value was assumed to be equal to ascending aortic pressure in all patients (none had supravalvular aortic stenosis or coarctation of the aorta with an aberrant right subclavian artery). Next, the aortic outflow tract was interrogated by Doppler echocardiography. If peak velocity was 150 cm/s or less, ventricular systolic pressure proximal to the aortic valve was considered equal to cuff blood pressure measurement. In cases of single ventricle, this measurement represented systolic pressure in the ventricle, and in cases of transposition, it represented right (systemic) ventricular systolic pressure. If peak velocity across the aortic outflow tract was greater than 150 cm/s, the aortic pressure drop was calculated using the modified Bernoulli equation (4,5): \[ \Delta P = 4V^2, \]
where \( \Delta P \) = pressure gradient and \( V \) = Doppler velocity distal to obstruction. In aortic outflow tract obstruction, a left ventricular to aortic pressure drop was added to the systemic systolic cuff pressure to determine approximate peak systolic ventricular pressure.

Peak velocity across a ventricular septal defect was then determined. If peak velocity was 150 cm/s or less, the interventricular communication was considered nonrestrictive with no pressure difference between the two ventricles. If peak velocity was greater than 150 cm/s, the pressure dif-
ference between the two ventricles was calculated. Because pressure in the ventricle from which the aorta arose had already been determined, subtracting the pressure difference across the ventricular septal defect allowed calculation of pulmonary ventricular pressure. In ventricular arterial concordance, this would be the right ventricle and in ventricular arterial discordance, either d- or l-transposition, this would be the left ventricle.

**Doppler interrogation of the pulmonary outflow tract** was then performed, and a pressure gradient, if present, was calculated. Subtracting this pressure gradient from the systolic pulmonary ventricular pressure allowed calculation of systolic pulmonary artery pressure. If a gradient occurred within the right ventricle, that is, anomalous right ventricular muscle bundles, this pressure gradient was subtracted from the proximal pulmonary ventricular pressure to calculate distal pulmonary ventricular pressure proximal to the right ventricular outflow tract.

**Catheterization procedure.** For comparison of Doppler pulmonary artery pressure estimates, systemic and pulmonary artery pressures were measured at cardiac catheterization with fluid-filled catheters coupled to Statham 23 dB transducers. Patients younger than 1 year of age were sedated with chloral hydrate, and older patients were sedated with a combination of meperidine (Demerol), sodium pentobarbital and chlorpromazine (Thorazine). Pressures were measured before angiography.

**Data evaluation.** Systolic pulmonary artery pressures measured at cardiac catheterization were compared with those estimated by Doppler echocardiography by correlation analysis and paired *t* testing. A probability (p) value of 0.05 or less was considered significant. Group data were expressed as range and mean ± 1 SD.

Because not all patients had Doppler echocardiograms performed within 24 hours of cardiac catheterization, systolic systemic to pulmonary artery pressure ratios were compared to correct for differences in pulmonary artery and systemic pressures that could have been temporally related.

**Results**

**Doppler- versus catheterization-measured pressures** (Table 1). Systolic pulmonary artery pressures measured at cardiac catheterization ranged from 15 to 100 mm Hg (mean ± SD 44 ± 26) and those measured by Doppler echocardiography ranged from 5 to 100 mm Hg (mean 43 ± 26) (*p* = NS; *r* = 0.92; SEE = 9.9; slope = 0.92; y intercept = 4.7) (Fig. 2). Systolic pulmonary artery to aortic pressure ratios measured at cardiac catheterization ranged from 0.2 to 1.0 (mean 0.5 ± 0.3) and those measured by Doppler echocardiography ranged from 0.1 to 1.0 (mean 0.5 ± 0.3) (*p* = NS; *r* = 0.94; SEE = 0.09; slope = 0.90; y intercept = 0.04) (Fig. 3).

**Figure 2.** Regression analysis of systolic pulmonary artery pressure estimates by Doppler technique versus measurement at cardiac catheterization (CATH). All values are in millimeters of mercury. The asterisk indicates one patient in whom the transventricular jet velocity was not adequately interrogated and pulmonary artery pressure was estimated to be high (see text for details).

**Figure 3.** Regression analysis of systolic pulmonary artery to aortic pressure ratio estimation by Doppler technique versus measurement at cardiac catheterization. The asterisk indicates the same patient as in Figure 2 (see text for details).
Figure 4. Case 15. Doppler tracings of an infant with d-transposition of the great arteries, ventricular septal defect and pulmonary stenosis. A, Transaortic (AO) velocity from subcostal short-axis plane = 100 cm/s. B, Sample volume across ventricular septal defect (TRANS VSD) in a subcostal long-axis plane. C, Transpulmonary peak velocity from suprasternal notch (SSN) by high pulse repetition frequency (HPRF) method. Highest peak modal velocity is in beat A at 400 cm/s. D, Transpulmonary peak velocity by continuous wave Doppler technique = 400 cm/s, suprasternal notch. LV = left ventricle; RV = right ventricle.
imimated the right ventricular pressure (85 mm Hg), when the 64 mm Hg gradient across the pulmonary valve was subtracted, the pulmonary artery pressure was calculated at 21 mm Hg. The value of the ratio of pulmonary artery to aortic pressure was 0.2. At cardiac catheterization, systolic pulmonary artery pressure was 21 mm Hg, and the pulmonary to aortic pressure ratio was 0.2.

**Discussion**

Our study demonstrates that systolic pulmonary artery pressure can be accurately estimated by Doppler echocardiography in infants and children with congenital heart disease if an interventricular communication exists. This method of measurement has significant clinical utility, especially because it is a noninvasive procedure that allows serial evaluation of pulmonary artery systolic pressure in pediatric patients with congenital heart defects.

**Underestimation of pulmonary artery pressure.** When peak velocity was obtained, the calculated pressure drop from Doppler study did not significantly exceed the gradient measured at cardiac catheterization. Accordingly, this method did not underestimate pulmonary artery pressure. Pulmonary artery pressure estimated by Doppler study was rarely less than a few millimeters of mercury different from pressure measured at cardiac catheterization. Clinically, these small differences are not important.

**Overestimation of pulmonary artery pressure.** The Doppler method can, however, significantly overestimate pulmonary artery pressure if proper peak jet velocity is not obtained across the interventricular communication or pulmonary outflow tract. In this case, a low estimated gradient will lead to a higher estimate of pulmonary artery pressure. Overestimation occurred in one patient (Case 5, asterisk in Fig. 2 and 3). Because of Doppler-estimated pulmonary artery hypertension, the patient underwent cardiac catheterization, which demonstrated a much lower pulmonary artery pressure than that suggested by Doppler examination. When subsequent Doppler echocardiography was performed (data are in brackets, Table 1), peak jet velocity across the ventricular septum was adequately measured and an accurate pressure estimate was then obtained.

**Practical examination guidelines in measurements of jet velocities.** Several concepts emerged to enhance the probability of success in aligning with the jet. First, good visualization of intracardiac anatomy is paramount. Second, Doppler interrogation guided by imaging echocardiography is helpful. Ventricular septal defect or pulmonary artery thrill location aided in transducer placement. The transducer was aimed initially in the direction where the jet lesion should occur, but this location did not always correspond to typical echocardiographic imaging planes. Interrogation was continued in the general area by movement of the transducer in all three planes until the highest jet velocity was obtained. No angle correction was used. The velocity envelopes were required to have a well rounded contour with the high intensity Doppler velocities on the perimeter of the envelope (Fig. 4C and D).

**Conclusion.** This study demonstrates that Doppler echocardiography can closely estimate systolic pulmonary artery pressure in patients with interventricular communications. If peak jet velocity is not adequately obtained, pulmonary artery pressure can be overestimated, but with careful Doppler technique, this overestimation should not commonly occur. Systolic pulmonary artery pressure should not be significantly underestimated. This method of noninvasive estimation of systolic pulmonary artery pressure in patients with congenital heart disease has significant clinical utility.

We thank Cheryl Craplick for her help in the editing and typing of the paper and Bill Hanson, Mark Ord and Jeanne Keiter-Marek for their help in the cardiac catheterization laboratory.

**References**