Noninvasive Detection of Pulmonary Hypertension in Patent Ductus Arteriosus by Pulsed Doppler Echocardiography

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SUMMARY Twenty-five patients with proven patent ductus arteriosus were examined by pulsed Doppler echocardiography (PDE) before invasive assessment. Ten patients had normal pulmonary artery pressures, and by PDE, pulsed diastolic ductal flow. Fifteen patients had elevation of mean pulmonary artery pressure, and by PDE, all had abnormalities of diastolic ductal flow. PDE correctly distinguished between patients with normal pressure and those with evidence of pulmonary hypertension; the ECG did not allow such differentiation. Detection by PDE of pulmonary hypertension complicating patent ductus arteriosus appears to be clinically useful.

A CONSEQUENCE of persistent patent ductus arteriosus (PDA) is the potential for transmission of systemic pressure and a large-volume shunt into the pulmonary circuit. Over time, the pressure and volume load on the pulmonary circulation may lead to pulmonary vascular disease, precluding closure of the ductus, and shortening life expectancy. In patients in whom no question of pulmonary hypertension (PHT) exists, ductal surgery may be recommended without catheterization. The noninvasive determination of the presence or absence of PHT would be beneficial to both groups of patients, and may avoid unnecessary catheterization in patients undergoing invasive study only because of a question of PHT. In this series, pulsed Doppler echocardiography (PDE) has been used to evaluate blood flow characteristics in the pulmonary artery in patients with PDA.

PDE allows noninvasive determination of direction of blood flow at known sites within the heart and great vessels, and at those known sites, allows differentiation between smooth, laminar blood flow and rough or turbulent blood flow.1 2 The PDE examination is similar to the traditional M-mode pulsed echo examination with the handheld transducer emitting a series of ultrasonic pulses. The position or depth along the "beam" of pulses that is to be anlayzed, termed the sample volume (SV), can be controlled by the examiner. The position of SV is indicated at all times on the A- and M-mode scopes of the PDE device. As the emitted pulses in the region of the SV detect a moving column of blood, the frequency of pulses back-scattered from the individual red blood cells will be altered (with respect to the frequency of the emitted pulses). By comparing the frequencies of emitted and back-scattered pulses, a frequency shift (or Doppler effect) can be detected. If the blood is moving away from the transducer, the back-scattered frequency will be lower than the emitted frequency. If the individual red blood cells at the position of the SV are moving with laminar blood flow characteristics, the frequency shift throughout the area of the SV will be uniform, and through the PDE device, a smooth audio sound will be produced. Since the frequency shift is uniform, the small dots representing the frequencies within the SV will be closely grouped in the time-interval histographic flow (TIH) (fig. 1, normal). If turbulent blood flow is present within the SV, the red blood cells have multiple directions and velocities within the moving column of blood, like bees in a swarm. While the net frequency shift will be either positive or negative, the individual velocities and directions within the SV may be widely divergent and, through the PDE device, produce a rough audio signal and scattering of the dots comprising the TIH (fig. 1, PDA). The PDE exam then consists of positioning the SV in known positions within the heart or great vessels, to sample direction and quality of blood flow at those sites.

Materials and Methods

This series included 25 infants and children, ages 1 day to 5 years who had PDE evidence of PDA, whose ductal patency and pulmonary arterial pressures were determined invasively (table 1). The PDE was isolated in nine, and was associated with transposition of the great arteries, ventricular septal defect, coarctation of the aorta, hypoplastic left heart syndrome in 13 (some with more than one defect). Three patients had PDA and chronic pulmonary disease of prematurity. In all children, the ductal shunt during diastole was mostly, if not exclusively, left to right; in the few children with proven bidirectional ductal shunts, the magnitude of overall right-to-left shunt component was very small.

PDE exams were performed using Advanced Technology Laboratories pulsed Doppler Units
Model 500A or 600A*, with 5 MHz or 3 MHz transducers, respectively. For recording of the TIH flow record, the PDE unit was adjusted according to standard procedure, using the signal amplitude indicator. As recommended by the manufacturer, the threshold control was set at the point where its amplitude trace jumped from baseline. The Doppler gain was set at that point where its amplitude trace left baseline at times of flow. These adjustments are routinely made when sampling sites or gain adjustments change. Since the signal amplitude indicator appears as an additional channel below the TIH, and may actually interfere with large TIH deflections, it was blanked during TIH recordings to improve the clarity of the presentation of TIH flow records.

The examinations were performed from a precordial approach with the SV placed in the pulmonary artery (fig. 2). Care was taken to position the SV distal to the pulmonic valve leaflets to avoid inclusion of valve motion artifacts in the PDE record.

Care was also taken not to position the SV so deeply that the SV was placed in the ductus itself; if this occurred, both systolic and diastolic flow would be noted to be toward the transducer. Strip chart PDE recordings were made at 75–200 mm/sec, and diastolic ductal flow was timed with ECG, and pulmonary valve motion. Duration of diastolic ductal flow was measured as shown in figure 3. Duration of diastolic ductal flow was averaged over five cardiac cycles, and expressed as a percentage of the diastolic interval. The duration of diastolic flow was compared with subsequent invasive measurements and established normal values for mean pulmonary arterial pressures.3

Results

Table 1 reveals that there was no abbreviation of diastolic ductal flow in the 10 children whose pulmonary artery pressures were normal; ductal flow was pandiastolic. In all patients whose pulmonary arterial pressures were elevated, abbreviation of diastolic ductal flow was noted. Patients with higher pulmonary artery pressures tended to have greater degrees of abbreviation of diastolic ductal flow (table 2, fig. 4). There were few patients, however, and a linear relationship was not present.

While the ECG was abnormal in the majority of patients (table 1), the ECG findings did not correlate well with presence or absence of PHT. For example, three patients whose ECG showed combined ventricular hypertrophy did not have PHT. One patient with proven PHT had a normal ECG, and four

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patients had right bundle branch block on ECG, precluding an ECG assessment of hypertension. As table 1 shows, however, PDE correctly distinguished those with PHT from those without PHT.

**Discussion**

The Doppler effect is greatest when the angle between the Doppler "beam" and the moving column of blood is small. Figure 2 shows that the angle is small when examining the blood flow characteristics in the pulmonary artery from a precordial approach. From this approach (fig. 1), systolic flow from the right ventricle into the pulmonary artery is away from the transducer (negative deflection in the flow record), while the diastolic ductal flow is toward the transducer (positive deflection in flow record). Hence, with PDA the directional changes in the PDE flow record are dramatic. We require recording of such distinct directional signals to be confident that the diastolic jet entering the pulmonary artery actually is ductal.

Other systemic-pulmonary communications will cause diastolic (and often systolic) turbulence in the pulmonary artery, but none of the patients with Blalock-Taussig (n = 9) or Waterston (n = 7) shunts that we have evaluated have had such directional signals to their flow records. One patient with aortic-pulmonic window, evaluated by PDE, lacked classic directional flow signals, though pulmonary artery turbulence was marked.

In this series of patients with proven PDA, PDE was useful in distinguishing between patients with PHT from those with normal pulmonary artery pressures, and was a more sensitive predictor of PHT than the presence of right or combined ventricular hypertrophy on the ECG. It is not surprising that PDE would be able to detect abbreviations of diastolic ductal flow in the presence of PHT. At the time of angiography, patients with PHT and PDA sometimes have only brief diastolic flow from aorta into the pulmonary artery. PDE is then not detecting a new phenomenon, but is merely allowing its noninvasive detection.
While PDE is useful for detecting PHT in patients with PDA, the exam must be carefully performed. If SV is placed in close proximity to the pulmonic valve, leaflet motion within the position of SV may produce artifacts in the flow record. Since leaflet motion produces audible clicks through the PDE device, positioning of SV beyond the pulmonary valve, in a position without clicks, should eliminate error. If the SV is placed very deeply into the pulmonary artery, it is possible to advance the SV into the ductus itself, in some cases. In this situation, turbulent, systolic and diastolic flow towards the transducer may occur, and may be confusing. A preferred position for the SV is in the main pulmonary artery, free of both ductus and pulmonary valve (fig. 2).

Since the blood flow into the pulmonary artery via PDA defines the presence of some left-to-right shunting, and since the direction of flow in purely right-to-left ductal shunts would be just the opposite, PDE is not useful for detecting right-to-left ductal shunts from the precordial approach.

Though there is excellent correlation between abbreviated ductal flow and elevation of pulmonary artery pressure, we expected a more linear correlation between the duration of flow and the magnitude of PHT. However, some children had low systemic pressures as a result of failure and poor cardiac output. Their mean pulmonary artery pressures (35, 37 and 34 mm Hg), while approaching systemic levels, were not very remarkable in absolute terms. We would expect a better correlation between abbreviated flow and elevated pulmonary resistance. However, in many catheterization laboratories, including ours, oxygen consumption is not measured for small infants. Especially in the patient with significant hemo-
Table 2. Pulmonary Arterial Pressure, Pulmonary Vascular Resistance, and Duration of Diastolic Flow

<table>
<thead>
<tr>
<th></th>
<th>Patent ductus arteriosus with pulmonary hypertension</th>
<th>Patent ductus arteriosus without pulmonary hypertension</th>
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</thead>
<tbody>
<tr>
<td>Pulmonary arterial pressure (mm Hg)</td>
<td>53</td>
<td>15</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (dyn-sec-cm⁻¹)</td>
<td>1894</td>
<td>108</td>
</tr>
<tr>
<td>Duration of diastolic flow (%)</td>
<td>52</td>
<td>100</td>
</tr>
</tbody>
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dynamic abnormalities, with varying cardiac output and oxygen consumption, such use of an assumed oxygen consumption dulls a precise estimate of pulmonary vascular resistance. Therefore, our results have been presented in terms of pressure rather than resistance.

One important factor in the management of infants and children with congenital heart disease is knowledge of pulmonary artery pressure, or resistance. Hence, the noninvasive detection of PHT in patients with PDA should be clinically useful. PDE, with the capacity to detect specific ductal flow patterns and PHT and to screen for additional defects,⁴ has significantly influenced the performance of invasive studies at our center. In patients with isolated PDA, and abbreviated ductal flow, catheterization is required to assess severity of PHT, though PDE can detect the presence of PHT. More patients with intrinsic cardiac disease and PDA may require catheterization to assess the severity of the additional defect. PDE may reveal the presence of PHT complicating the defect, however, and as long as the ductus remains open, the duration of diastolic flow can be periodically reassessed, as a question of PHT arises. Patients in whom clinical and PDE exams suggest isolated PDA, and in whom PDE demonstrates a pandiastolic ductal jet flowing into the pulmonary artery, should not require catheterization if the only question is whether PHT is present.

References

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J G Stevenson, I Kawabori and W G Guntheroth

Circulation. 1979;60:355-359
doi: 10.1161/01.CIR.60.2.355

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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