Pulsed Doppler Echocardiographic Detection of Obstruction of Systemic Venous Return After Repair of Transposition of the Great Arteries

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SUMMARY Pulsed Doppler echocardiography (PDE) allows the detection of direction and quality (smooth vs disturbed, or "rough") of blood flow characteristics at known sites within the heart and great vessels. In order to test the ability of this technique to detect obstruction of systemic venous return after interatrial repair of transposition of the great arteries, twelve children who had undergone such surgery were examined by PDE. The blood flow characteristics in the systemic venous return atrium in the children were compared with invasive pressure measurements in the venae cavae and systemic venous return atrium. Six children without measurable baffle gradients had phasic diastolic flow characteristics in the systemic venous return atrium similar to the normal. All six children who had a gradient between the cava and systemic venous return atrium of 3 mm Hg or more had nonphasic disturbed blood flow in the systemic venous return atrium during diastole. We conclude that 1) PDE offers simple and specific detection of atrial flow patterns in infants who have had interatrial repair of transposition of the great arteries; 2) the detection of nonphasic disturbed atrial flow during diastole strongly suggests the presence of obstruction to systemic venous return; and 3) the detection of phasic atrial flow suggests the absence of significant obstruction.

DURING INTERATRIAL repair of transposition of the great arteries (TGA), care is taken to avoid obstruction or narrowing of the venous channel. Even so, obstruction to venous channels does occur and may be severe enough to require reoperation. Invasive studies may be required to detect or assess such obstruction. We here report the use of pulsed Doppler echocardiography (PDE) as a noninvasive tool for the detection of narrowing or obstruction of systemic venous return after interatrial repair of TGA.

PDE is a recently developed adjunct to traditional echocardiographic techniques and is described in detail elsewhere.1 The PDE examination is performed with a hand-held transducer, in a fashion very similar to the traditional M-mode echocardiographic examination. The transducer emits a series of pulses, and as the pulses are back-scattered from within the heart, the PDE unit compares the frequency of the back-scattered pulses with the frequency of emitted pulses. If the "beam" of pulses has encountered a moving column of blood, the frequency of back-scattered and emitted pulses will differ, and a Doppler effect will be detected. For example, if the blood is moving away from the transducer, back-scattered pulses will have a lower frequency than emitted pulses, and the PDE unit will record flow as being directed away from the transducer. In this way, the direction of blood flow is determined. The examiner can vary the position along the "beam" of pulses that is to be sampled (such position termed the sample volume [SV]), and that position is continuously indicated on A- and M-mode echocardiographic display, so that the examiner knows at all times where blood flow is being sampled. The PDE unit evaluates the uniformity of the frequency shift at the position of the SV in order to allow differentiation of smooth from disturbed blood-flow patterns. If, in the position of the SV, the blood flow is smooth or laminar, the frequencies of pulses back-scattered from the 2 × 4-mm area of SV will be uniformly shifted and lead to a uniformly shifted signal through the PDE unit. If, however, turbulent flow is present at the SV position, the many red blood cells in the area of the SV will have multiple directions and velocities, leading to wide fluctuation in frequencies of back-scattered pulses. The PDE unit produces both an audio and a spectral output, with smooth flow giving a "smooth" audio sound, and disturbed or "rough" blood flow a harsh sound, similar in quality to that of organic murmurs. The spectral output appears as a time-interval histogram (TIH). If smooth flow has been encountered at the position of the SV, the numerous dots that constitute the TIH remain close together and form a wave (fig. 1). If disturbed flow is detected, the dots are scattered (fig. 2). The exam is timed with an ECG. The examiner can thus sample the direction and quality (smooth vs disturbed) of blood flow at known sites within the heart and great vessels in systole and diastole. We studied the flow characteristics of children in this series within the systemic and pulmonic venous return atri, on either side of the surgically placed interatrial baffle.

Materials and Methods

PDE examinations were performed with an Advanced Technology Laboratories (Bellevue, Wash-
FIGURE 1. Pulsed Doppler echocardiographic (PDE) record of normal phasic flow at inflow of mitral valve. At the top of the record and all PDE records is a compressed M-mode recording showing position of sample volume (SV) with respect to identifiable structures RV = right ventricle; LV = left ventricle; M = mitral valve. Immediately below is an ECG. Below this is the time-interval histographic flow record. The large and small arrows emphasize the phasic character of normal atrial flow, which closely parallels the normal phasic mitral valve motion in this normal patient.

FIGURE 2. Pulsed Doppler echocardiographic record from a child with proven obstruction to systemic venous return. At the top of the record is a compressed M-mode tracing, showing aorta (AO), pulmonary artery (PA), and the posterior atrial chambers. The baffle (B) is shown by the arrow. The position at which blood flow characteristics are sampled, the sample volume (SV) is shown. The time-interval histographic spectral flow record is immediately below. At the far right of the figure, the SV is within the AO, and is thence moved into the PA. The dots that constitute the spectral flow record are closely grouped into a "wave" form. As the SV is moved posterior to PA and into the systemic venous return atrium (SVA) near the middle of the figure, the spectral flow record changes dramatically, and gross scattering of the dots makes up the record. In contrast to the M-shaped phasic diastolic flow pattern of the normal patient in figure 1, the dots are so scattered in the SVA of this obstructed patient that there is no phasic character to diastolic flow. With SV posterior to B in the pulmonary venous return atrium (left), one can appreciate dot scatter of disturbed flow, but there is an M-shaped phasic pattern in diastole, especially pronounced in the third complex.
inflow, and flow was followed back into the systemic venous return atrium (SVA). At times the SVA appeared easily approachable directly behind the pulmonary artery, and at times, a more medial approach from the right sternal border was required to obtain flow signals. In nine of 12 infants, a small-volume saline flush was made through a venous catheter to confirm the identity of the SVA with the appearance of contrast echoes on the same side of the baffle as predicted by following flow back from the mitral valve (fig. 3). The PDE evaluation consisted of sampling blood flow characteristics throughout the SVA. Because we have not encountered significant pulmonary venous obstruction in our transposition patients, flow characteristics in the pulmonary venous return atrium are not reported here.

Attention was directed to the pattern of the TIH flow record, when the SV was in the SVA. Patients who had a distinct phasic character of the diastolic portion of the flow record (similar to that shown in figure 1 from normal patient and figure 4 from a patient with phasic flow) were labeled as having phasic diastolic flow. Patients without phasic diastolic flow patterns from TIH were considered abnormal.

Disturbed flow was considered to be present when a rough audio sound and dot scatter of TIH were found. Some patients had disturbed flow with phasic characteristics, some had disturbed flow without discernable phasic characteristics. When disturbed flow (dot scatter on TIH and "rough" audio signal) was detected, the SV was moved in the direction expected to encounter the superior or inferior limbs of the baffle to determine in which limb the turbulence arose. The PDE flow patterns were compared with pressure gradients between the cavae and SVA obtained by direct operative measurement, indwelling lines or cardiac catheterization (table 1).

**Results**

In all patients in whom venous injections of saline were made during PDE exams, the contrast echoes appeared on the same side of the baffle that had been judged, by following flow back from the mitral valve, to be the SVA. This method of identifying SVA with PDE (following flow back from the mitral valve), therefore, seems valid.

There was a measurable gradient of 3 mm Hg or more across the superior vena caval (SVC) limb of the baffle in six patients (table 1). An angiocardiographic frame showing narrowing of the SVC limb and a patch leak is shown in figure 5. There was an equivocal gradient across the inferior vena caval (IVC) limb in one patient. In the six patients with proven SVC limb gradients, the SVA flow was nonphasic in diastole (fig. 2). (The "M"-shaped phasic flow pattern marked by arrows in figure 1 is not present in SVA in figure 2.) Normal diastolic phasic flow characteristics were present in the PDE records from the remaining six children, who had no significant gradient (fig. 4). In one patient, we could not detect flow in the region of the SVC limb, and catheterization showed near-total obstruction. Nonphasic diastolic flow was detected in the SVA and in the direction of the IVC limb. This child had IVC limb stenosis at reoperation, but a pressure recording was not made.
In 11 of the 12 patients, the SVA flow was disturbed. Based largely on the audio output of the PDE device, and to some extent on the degree of dot scatter on the flow records, there was a suggestion that the children with proved obstruction had greater degrees of flow disturbance. However, because interpretation of degree of “roughness” of the audio signal is subjective, and because the degree of dot scatter on flow records is not quantitative, a clear distinction between the obstructed and nonobstructed groups could not be made on the basis of “roughness” alone.

**Table 1. Invasive and Noninvasive Assessment After Interventricular Repair of Transposition of the Great Arteries**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Months postop</th>
<th>Pressure gradient (mm Hg)</th>
<th>Flow characteristics, systemic venous atrium</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5</td>
<td>48</td>
<td>0</td>
<td>3* Nonphasic</td>
</tr>
<tr>
<td>0.6</td>
<td>0.3</td>
<td>0</td>
<td>6* Nonphasic</td>
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<td>2.5</td>
<td>1.5</td>
<td>0</td>
<td>0* Phasic</td>
</tr>
<tr>
<td>10</td>
<td>96</td>
<td>0</td>
<td>3† Nonphasic</td>
</tr>
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<td>0</td>
<td>0† Phasic</td>
</tr>
<tr>
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<td>0.1</td>
<td>1</td>
<td>0* Phasic</td>
</tr>
<tr>
<td>0.3</td>
<td>0.1</td>
<td>0</td>
<td>10† Nonphasic</td>
</tr>
<tr>
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<td>0</td>
<td>0† Phasic</td>
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<td>0</td>
<td>4† Nonphasic</td>
</tr>
<tr>
<td>0.7</td>
<td>0.1</td>
<td>0</td>
<td>0† Phasic</td>
</tr>
<tr>
<td>0.6</td>
<td>3.0</td>
<td>ND</td>
<td>10* Nonphasic</td>
</tr>
</tbody>
</table>

*Pressure measurement at cardiac catheterization.
†Pressure measurement at surgery or by indwelling caval catheters.
‡Patient had baffle-leak atrial septal defect.

Abbreviations: IVC = inferior vena caval; SVC = superior vena caval; ND = not done.

**Discussion**

There was excellent correlation between the loss of phasic diastolic atrial flow characteristics and the presence of a limb gradient of 3 mm Hg or more, as no patient with phasic diastolic atrial flow characteristics had a significant gradient, and all who had lost phasic flow characteristics had a significant gradient or operative visualization of stenosis. Because the SVC limb was involved in all cases with obstruction, and the only case with confirmed IVC limb narrowing did not have a pressure measurement, it is difficult to assess PDE usefulness in determining which limb was stenotic.

The correlation of phasic diastolic flow and the absence of gradient seem reasonable, because phasic flow characteristics should be present if blood flow is not impeded, in response to ventricular filling and atrial contraction (fig. 1, M-shaped diastolic flow pattern). Conversely, if there is narrowing, impediment to flow may be manifest by a pressure gradient throughout diastole, leading to a loss of phasic flow characteristics on the TIII flow record (fig. 2, middle; no “M” shape to diastolic flow record). Because of obstruction, flow does not follow the usual determinants of atrial flow characteristics.
A mild degree of narrowing at either caval entrance to SVA is probably common, and may be a reason that the majority of patients in our series had some degree of flow disturbance in SVA. We consider an atrial gradient of 3 mm Hg or more to be significant. Stark et al., based on a review of a large series of patients who had undergone repair of TGA, suggest an operative gradient of 2 mm Hg as a guide to presence of obstruction in the Mustard repair. The magnitude of gradient may not correlate well with the severity of obstruction, because of differing extent of collateral flow or decompression via an atrial septal defect (case 4 and fig. 5). In our series, there was good correlation between a gradient of 3 mm Hg or more and an alteration of atrial flow characteristics. There may or may not be clinical manifestations with such gradients. Our patient with an isolated 3-mm Hg SVC limb gradient did not have a SVC syndrome. Case 4, who had a similar gradient and a baffle leak, may have sufficiently decompressed via the atrial septal defect (right-to-left shunt with 90% systemic saturation at catheterization) that signs of SVC syndrome were not manifest. Our two most impressive SVC syndromes were seen in patients with 4- and 10-mm Hg gradients, 4 and 8 months after operation; the patient with a 4-mm Hg gradient had a large azygous pathway. A low gradient or the absence of physical signs of a SVC syndrome do not seem to preclude the presence of moderate obstruction. Given the problem that may be associated with obstruction and the possibility of late onset of signs of obstruction, PDE may well be useful in identifying patients whose atrial flow characteristics suggest the possibility of obstruction, even in the absence of physical findings. Such patients should have closer follow-up.

In the early postoperative period, if one has appropriate in-dwelling catheters for pressure monitoring, obstruction to systemic venous return may be detected. If however, one is evaluating a child who does not have appropriately placed catheters, or who is in a later postoperative stage without catheters, PDE has the advantage of determining whether phasic flow characteristics are present in the SVA during diastole. Several of our infant transposition patients have had problems early after operation, with significant "third spacing of fluid" and marked edema, raising the question of caval obstruction. The noninvasive determination of SVA flow characteristics offers the advantage of separating children with baffle obstruction from those whose edema is from other causes.

We conclude that PDE offers simple and specific detection of altered atrial flow patterns in infants who have undergone repair of TGA by interatrial venous transposition, and that the PDE detection of nonphasic, diastolic atrial flow strongly suggests the presence of obstruction to systemic venous return. Conversely, the presence of normal phasic flow characteristics in the SVA suggests that the presence of significant obstruction is unlikely.

References
Pulsed Doppler echocardiographic detection of obstruction of systemic venous return after repair of transposition of the great arteries.
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