Echocardiographic Diastolic Flutter of the Pulmonary Valve in Isolated Patent Ductus Arteriosus*

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Diastolic flutter of the pulmonary valve was observed on M-mode echocardiograms in 15/39 (38 percent) infants and children with a confirmed diagnosis of isolated patent ductus arteriosus. We postulate that this flutter is caused by a high-velocity jet of blood from the patent ductus directed at the pulmonary valve and resulting in flutter of the valve leaflets throughout the cardiac cycle, most remarkable during diastole. In patients with left heart volume overload pattern, this finding increases the specificity of M-mode echocardiography in the diagnosis of patent ductus arteriosus.

The M-mode echocardiographic findings of patent ductus arteriosus described to date are non-specific.1 Having noted diastolic flutter of the pulmonary valve in several patients subsequently proved to have an isolated patent ductus, we undertook to review our echo records to study the specificity and sensitivity of this finding.

MATERIALS AND METHODS

M-mode echocardiograms performed in 192 infants and children with a clinical diagnosis of isolated patent ductus arteriosus were reviewed. Thirty-six (19 percent) had unequivocal diastolic pulmonary valve flutter. The diagnosis was confirmed in 38/192 patients. Of these, 15 (38 percent) had diastolic flutter of the pulmonary valve. Eight of these 15 patients were full-term infants and children 20 days to 4 years of age at initial echo study. All eight, plus three premature infants six to 11 days of age at the time of the initial echo study, underwent diagnostic cardiac catheterization, cineangiography, and surgery. Three additional premature infants had surgical closure of the ductus, and another premature infant had autopsy confirmation of the diagnosis. In the patients with a confirmed diagnosis of patent ductus but without diastolic pulmonary valve flutter, the diagnosis was confirmed by cardiac catheterization (20 patients), surgery (14 patients, three of whom had no catheterization), and autopsy (two patients, one of whom had no catheterization). Pulmonary valve echograms in 64 patients with documented subvalvular pulmonary stenosis (one isolated, five double chambered right ventricle, and 58 tetralogy of Fallot—43 preoperative only, 14 preoperative and postoperative, and 15 postoperative only) and in 50 infants with clinically normal hearts were reviewed for the presence of diastolic pulmonary valve flutter.

RESULTS

The initial echocardiogram showed left heart volume overload pattern, ie, increased left atrial and/or left ventricular dimensions and left atrial/aortic root ratio, in all 15 patients with patent ductus included in the diastolic pulmonary valve flutter group. The initial pulmonary valve echogram was normal in two premature infants aged seven days. Initial pulmonary valve echograms in the other 13 patients, as well as the follow-up echograms in those two premature infants, showed unequivocal diastolic flutter. All 15 patients had clinical findings of a patent ductus arteriosus, including a loud continuous murmur, grade 3 to 4/6, when the pulmonary valve diastolic flutter was noted. The flutter was early to mid-diastolic in eight patients, five premature and three full-term infants and children. In the remainder (seven) it was holodiastolic (Fig 1A). One premature infant and two

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older children had coarse, low-frequency flutter, the other 15 showing fine, high-frequency flutter. In the 14 who had surgical closure of the ductus, the flutter was no longer present in postoperative studies (Fig 1B). After administration of indomethacin to one infant, we noted clinical closure of the ductus and disappearance of pulmonary valve flutter. A second infant, since proved to have rubella syndrome, showed no clinical response and no change in the pulmonary valve echogram after oral indomethacin. A third infant who had a large ductus but no murmur had a normal pulmonary valve echogram at seven days of age. He developed marked, high-frequency, low-amplitude holodiastolic flutter of the pulmonary valve by 18 days of age (Fig 2A). This change coincided with the development of a grade 4/6 continuous murmur. The flutter decreased in degree and duration after oral indomethacin therapy (Fig 2B) and disappeared after surgical ligation of the ductus at 50 days of age (Fig 2C).

Of the 20 patients with catheterization-confirmed patent ductus but no diastolic pulmonary valve flutter, ten had a grade 3-4/6 continuous murmur, five had a soft, grade 1 to 2/6 continuous murmur, and five had no continuous murmur.

Pulmonary artery pressure averaged 25 ± 16/10 ± 3 mm Hg in patients with flutter and 29 ± 15/14 ± 10 mm Hg in those without flutter (PNS). Pulmonary vascular resistance, calculated using a measured oxygen consumption, was slightly lower in those patients with flutter (0.7 ± .3 vs 1.2 ± .6 units in those patients without flutter; P < .05). No other significant differences in hemodynamic measurements were found between the two groups.

Cineangiograms following injection of contrast into the descending aorta were available in 31 patients and showed mild pulmonary insufficiency in 10/11 with diastolic pulmonary valve flutter and in 5/20 without flutter (Fig 3).

Diastolic flutter of the pulmonary valve was not noted in any of the pulmonary valve echograms reviewed in 64 patients with subvalvular pulmonary stenosis or in 50 normal infants.

**DISCUSSION**

Diastolic flutter of the pulmonary valve is said to be an uncommon or even rare echocardiographic finding.3-4 The few previous reports are anecdotal
in nature; only four cases were reported, involving three adults with infundibular pulmonary stenosis and one child with pulmonary artery branch stenosis and pulmonary insufficiency following repair of tetralogy of Fallot. Nanda et al briefly commented on having observed diastolic pulmonary valve flutter in normal infants. In no case was the echo study compatible with left heart volume overload. We did not note diastolic pulmonary valve flutter in a review of 50 normal infants and 64 preoperative and postoperative patients with sub-valvular pulmonary stenosis.

The incidence of pulmonary valve diastolic flutter in patent ductus arteriosus is unclear from this study, but it is not rare. Of those infants and children with a clinical diagnosis of patent ductus, 19 percent (36/192) had diastolic pulmonary valve flutter. Of those with a confirmed diagnosis, 38 percent (15/39) had flutter. The sensitivity of this finding can be estimated as between 19 to 38 percent.

The disappearance of flutter following surgical closure of the ductus in 14 patients and pharmacologic closure in the 15th attests to a direct effect of the ductus in production of flutter. Despite the higher incidence of pulmonary insufficiency in those patients with flutter, it is unlikely that the pulmonary valve diastolic flutter is related to the pulmonary insufficiency per se. In two studies of pulmonary valve motion in pulmonary insufficiency due to various causes with normal or elevated pulmonary artery pressure, diastolic flutter was not noted in any of the patients. More likely, the observation of pulmonary insufficiency in our patients can be viewed as documentation of the presence of a high-velocity jet of blood from the ductus directed at the pulmonary valve and resulting in flutter of the valve leaflets throughout the cardiac cycle, most remarkable during diastole.

It is likely that some degree of constriction of the ductus is necessary to produce a jet of sufficient energy to cause the pulmonary valve to flutter. The presence of a loud continuous murmur in all 11 catheterized patients with flutter and in only 10/20 catheterized patients without flutter lends support to this hypothesis. The third infant described initially had no continuous murmur and no diastolic flutter despite signs of a large shunt. We postulated that at that time the patent ductus was unrestricted with minimal turbulence of flow. With spontaneous constriction of the ductus, turbulence increased, and a continuous murmur and diastolic flutter appeared. Further constriction following indomethacin administration resulted in decreased turbulent flow, decreased intensity of the murmur, and decreased duration and degree of the diastolic flutter. The flutter disappeared after surgical ligation of the ductus.

Previously, other authors have attempted to increase the specificity of the echocardiographic diagnosis of patent ductus arteriosus. The use of contrast with injection into the thoracic aorta has been advocated. Although this technique documents a left-to-right shunt at arterial level, it requires placement of an arterial line, use of a special transducer, and, furthermore, does not exclude other lesions causing left-to-right arterial shunts. Although Doppler techniques for examination of flow characteristics of a ductus appear very promising, the instrumentation and expertise for such examinations are not yet widely available. Direct imaging of a ductus with two-dimensional echo has been reported. The sensitivity of this technique is yet to be established and will no doubt depend highly on the lateral resolution characteristics of the instrument used and the experience of the examiner.

The widespread availability of M-mode echo instruments and experienced examiners makes this type of examination the mainstay of diagnostic echocardiography. A finding, such as the one described, that increases the specificity of this non-invasive technique should have diagnostic significance.

Although diastolic pulmonary valve flutter ap-
pears to be a fairly common finding, it has not been reported previously in patent ductus arteriosus and was observed in only 38 percent of our patients. An unrestrictive ductus and a very small one probably do not produce a jet of sufficient energy to cause the pulmonary valve flutter. In addition, perhaps in some cases the orientation of the ductus to the pulmonary artery directs the jet of shunted blood against the wall of the pulmonary artery rather than at the pulmonary valve.

Clinical Significance

Although the present study shows a relatively low sensitivity for this finding (38 percent), it appears to be highly specific when observed in patients with left heart volume overload pattern.

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