Echocardiographic Estimation of Pulmonary Artery Pressure in Transposition of the Great Arteries

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SUMMARY To determine their usefulness in estimating pulmonary artery pressure, left ventricular systolic time intervals (STI) were determined by echocardiography in 65 patients with dextro-transposition of the great arteries (TGA). The STI were measured from recordings of pulmonary valve motion at 100 mm/sec paper speed.

The pre-ejection period (PEP) and the ratio of PEP to left ventricular ejection time (PEP/LVET) were directly related to pulmonary artery pressure. The strongest correlations were that between PEP/LVET and pulmonary artery diastolic pressure ($r = 0.70$) and (pulmonary valve) motion were made at 100 mm/sec paper speed. The pre-ejection period (PEP) was considered to be the time from the onset of the QRS complex of the electrocardiogram to pulmonary valve opening, and left ventricular ejection time (LVET) was measured from the pulmonary valve opening to closing (fig. 1). In the case of fluttering or mid-systolic closure of the pulmonary valve leaflets, the point at which the fluttering ceased and the leaflets assumed their typical linear diastolic appearance was considered the end of left ventricular ejection. The ratio PEP/LVET was then calculated. If there was respiratory variation, the cycles with the shortest LVET were used in the estimation of pulmonary artery pressure. Measurements were rounded off to the nearest 5 msec. In the initial half of the study, 0.5 sec time lines generated by the echograph were used. During the remainder of the study, 0.02 sec time lines generated from a quartz crystal oscillator were available. Use of these time lines revealed that the timing strobe of the echograph consistently underestimated correct time by 2-3%. This error was felt to be insignificant and the data from the two parts of the study were combined.

Pulmonary artery pressure was measured with fluid filled catheters at the time of cardiac catheterization. Patients were sedated with a mixture of meperidine, chlorpromazine and promethazine; none received general anesthesia.

Linear regression analysis was used to determine the relationship of PEP, LVET, and PEP/LVET to pulmonary artery systolic, diastolic and mean pressure, and to the ratio of mean pulmonary artery pressure to mean systemic artery pressure.

Results

The left ventricular PEP and the ratio PEP/LVET were directly related to pulmonary artery pressure. The strongest correlations were that between PEP/LVET and pulmonary diastolic pressure ($r = 0.70$) and that between PEP/LVET and the ratio of mean pulmonary to mean systemic pressure ($r = 0.71$). The correlation coefficients relating PEP to pulmonary diastolic pressure and the ratio of mean pulmonary to mean systemic pressure were 0.62 and 0.66, respectively.

The STI were more accurate in predicting low pulmonary pressure than in predicting pulmonary hypertension. In 31 patients, PEP/LVET was less than 0.26; in each of these
patients, pulmonary diastolic pressure was 20 mm Hg or less and in 28 of the 31, mean pulmonary pressure was less than one-third of mean systemic pressure (fig. 2).

In patients with PEP/LVET of 0.26–0.30, pulmonary artery diastolic pressures ranged from 4 to 70 mm Hg and from 12 to 100 percent of systemic. A PEP/LVET of 0.30 or greater was generally associated with pulmonary hypertension. However, in four patients, PEP/LVET was 0.30 or greater despite normal pulmonary artery pressure (fig. 2). Each of these patients had previous intra-atrial baffle repair and two required reoperation for pulmonary vein obstruction. All four had electrocardiographic abnormalities at the time of study (one case each of right bundle branch block, left bundle branch block, atrial flutter, and atrioventricular dissociation). In two of these patients, including the one with right bundle branch block, cardiac catheterization and angiography demonstrated biventricular dysfunction as manifest by elevated enddiastolic pressure and reduced ejection fraction.

The relationship between PEP/LVET and pulmonary artery pressure was also present when the different types of TGA were analyzed individually. For patients with TGA and VSD, TGA and PS, and TGA post-Mustard procedure, the correlation coefficients between PEP/LVET and the ratio of mean pulmonary to mean systemic artery pressure were 0.62, 0.65, and 0.74 respectively. Low ratios of PEP/LVET also correctly predicted the low pulmonary artery pressure in patients with simple TGA and TGA, VSD, and PS. However, the correlation coefficients were not strong for these two groups because all of the data points were clustered together in the lower left corner of figure 2.

Discussion

A knowledge of pulmonary artery pressure is necessary for the management of patients with TGA. If pulmonary pressure is low and the patient is clinically stable, we have deferred intra-atrial baffle repair until the second year of life. However, rising pulmonary pressure and resistance may necessitate either palliative or corrective surgery at an earlier age. After the Mustard procedure, elevated pulmonary artery pressure may reflect obstruction of the pulmonary veins, or progression of pulmonary vascular obstructive disease. Obviously a noninvasive method of estimating pulmonary pressure would be valuable in the management of patients with TGA.

In subjects with normally related great arteries, left ventricular STI have most commonly been used to assess left ventricular function, rather than to estimate systemic arterial pressure. This emphasizes the fact that many variables affect the STI, only one of which is the pressure or resistance of the distal vascular bed. The hypothesis tested in this study is that pulmonary artery pressure is the most im-
portant determinant of left ventricular STI in children with TGA, irrespective of age, heart rate, associated cardiac lesions, digoxin therapy or previous surgery. Increased pulmonary artery pressure should prolong the PEP (in particular, the isovolumetric contraction time) and shorten LVET, thus producing a higher ratio of PEP/LVET.12

The results of the study demonstrate a direct relationship between pulmonary artery pressure, either in absolute terms or in comparison to systemic arterial pressure, and the left ventricular PEP and the ratio PEP/LVET. The latter had a stronger correlation with pulmonary pressure, probably because it is less influenced by heart rate than PEP itself.7 The wide range of pressures among patients with values of PEP/LVET of 0.26 or greater would seem to preclude a precise estimate of pulmonary artery pressure from the STI. However, a ratio of PEP/LVET of less than 0.26 was almost invariably associated with low pulmonary pressure.

If left ventricular STI are used as a screening test for pulmonary hypertension in children with TGA, there will be some false positives. As demonstrated in four patients in this study, cardiac arrhythmia, conduction delay, or reduced myocardial contractility may prolong PEP and increase the PEP/LVET ratio, causing overestimation of pulmonary pressure. Digoxin therapy, which tends to lower PEP/LVET,13 might be expected to produce an underestimate of pulmonary pressure, although this was not apparent in the present study. Our patients with pulmonary hypertension had elevated ratios despite digoxin therapy.

Previous echocardiographic studies of pulmonary hypertension14,15 have demonstrated a lack of posterior movement of the pulmonary valve in early and mid-diastole, diminished valve motion ("A" dip) following atrial contraction and rapid rates of valve opening. Although these features were often present in patients with pulmonary hypertension in the present study, the wide range of patient size and heart rate made it difficult to quantitate these findings. The results are in agreement with those of Hirshfeld et al.,7 who found a direct relationship between PEP/LVET and pulmonary artery pressure. Because of the difficulty in accurately measuring pulmonary blood flow in patients with TGA, no attempt was made to compare the STI with pulmonary resistance in the present study.

The technique described is particularly useful for longitudinal studies of individual patients, since serial echocardiograms can be performed safely and rapidly on an outpatient basis. In the clinical setting, it does not seem possible to completely neutralize the effects of myocardial contractility, cardiac medications, heart rate, and intra-cardiac conduction on left ventricular STI. Nonetheless, the presence of an elevated ratio of PEP/LVET (0.30 or higher) in a patient with TGA suggests that one of the complications of this condition may be present. In this study, pulmonary hypertension was the most common complication. Conversely, the presence of a low PEP/LVET (under 0.26) seems to offer reasonable assurance that a significant elevation of pulmonary artery pressure is not present.

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