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 STIs 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 that between PEP/LVET and the ratio of mean pulmonary pressure to mean systemic pressure ($r = 0.71$).

A value of PEP/LVET of less than 0.26 was consistently associated with pulmonary artery diastolic pressures of less than 20 mm Hg and, in 28 of 31 patients, pulmonary artery pressure less than one-third of mean systemic arterial pressure. Pulmonary hypertension was present in 18 of 22 patients with PEP/LVET of 0.30 or greater; elevated PEP/LVET was also present in four patients with abnormalities of cardiac rhythm or conduction, two of whom also had angiographic evidence of myocardial dysfunction.

PATIENTS WITH dextro-transposition of the great arteries (TGA) frequently develop pulmonary hypertension at an earlier age than children with other forms of congenital heart disease.\(^1\)\(^5\) Since the history, physical examination, electrocardiogram and chest roentgenogram are frequently unreliable indicators of pulmonary artery pressure in TGA, serial cardiac catheterizations are often necessary to plan medical and surgical therapy.

Hirschfeld et al.\(^4\) have demonstrated that echocardiography can be used to determine the systolic time intervals (STI) of the systemic and pulmonary ventricle and that the STI of the latter can be used to estimate pulmonary artery pressure and resistance.\(^7\) In the present study, this methodology has been applied to a group of patients with various forms of TGA to determine its utility in estimating pulmonary artery pressure, both before and after intracardiac surgery.

Methods and Materials

Echocardiograms were obtained on 65 patients with TGA within 48 hours of cardiac catheterization. The patients ranged in age from one day to 25 years; 12 had uncomplicated TGA, 19 had an associated ventricular septal defect (VSD), six had pulmonic stenosis (PS), nine had VSD and PS, and 18 patients had undergone an intra-atrial baffle repair (Mustard procedure). Fifty-five patients were receiving maintenance digoxin therapy at the time of the study.

Echocardiograms were performed with a Hoffrel Model 101 ultrasonoscope, coupled to a Honeywell Model 1856 strip chart recorder. Transducers of 2.25, 3.5 or 5.0 MHz were used, depending on the patient's size. Recordings were made from the second, third or fourth interspace at the left sternal edge, with the patient supine or in a shallow left lateral decubitus position.

Since the pulmonary artery arises from the left ventricle in TGA, left ventricular STI were compared to pulmonary pressure. Recordings of posterior semilunar valve (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 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

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In 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.\textsuperscript{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.\textsuperscript{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,\textsuperscript{12} might be expected to produce an underestimation 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 hypertension\textsuperscript{4,12} 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.,\textsuperscript{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 intracardiac 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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