Isovolumic contraction time of right ventricle in d-transposition of great arteries*

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SUMMARY The pre-ejection period of the right ventricle in d-transposition of the great arteries is known to be prolonged, compared with the same interval of the left ventricle of normal subjects. In the present study, the echocardiographic measurement of the components of the pre-ejection period of the right ventricle of 14 patients with d-transposition of the great arteries shows that the isometric contraction time of the right ventricle in d-transposition of the great arteries is similar to the same interval calculated on the left ventricle of 76 normal children of comparable age. On the other hand, the electromechanical delay was significantly greater for the right ventricle of d-transposition of the great arteries than for the left ventricle of the normal subjects. It is concluded that the prolonged pre-ejection period of the right ventricle in d-transposition of the great arteries is not the result of right ventricular dysfunction but solely of a longer electromechanical delay.

In d-transposition of the great arteries (d-TGA), the right ventricle must maintain a normal output against the high resistance of the systemic circulation. Its ability to sustain this load throughout life has been seriously questioned since angiographic studies have shown that right ventricular end-diastolic volume is abnormally increased and the ejection fraction decreased, both before and after intra-atrial repair by the Mustard procedure.1 2

A recent publication on the assessment of myocardial function after Mustard’s operation showed that the ratio of systolic time intervals for the right and left ventricles was appropriate for normal systemic and pulmonary ventricles, respectively.3 On the other hand most studies on myocardial function using systolic time intervals seem to be in agreement with volume measurements and have shown a prolonged right ventricular pre-ejection period and a shortened right ventricular ejection time in d-TGA suggesting that right ventricular function was depressed.4 5 However, these reports did not specify which part of the pre-ejection period, that is the electromechanical delay or the isovolumic contraction time, was responsible for prolonging the right ventricular pre-ejection period. Clarification of this point is essential before any conclusion can be drawn about the performance of the right ventricle. Indeed, isovolumic contraction time is the component of the pre-ejection period widely recognised as a reliable index of myocardial contractility.6 7

The frequent clinical observation that postoperative patients with d-TGA achieve normal physical performance without any sign of cardiac failure, and the fact that a prolonged right ventricular pre-ejection period has been found right after birth in this disease,8 prompted us to investigate the possibility that in d-TGA, the right ventricular pre-ejection period was prolonged because of the lengthening of the right ventricular electromechanical delay.

Subjects and methods

Fourteen patients with d-TGA, surgically repaired by Mustard procedure, were included in this study. In all but two an atrial septostomy had been done previously. Their ages varied from 3½ to 16 years, with a mean of 7 years. Duration of follow-up after corrective surgery averaged three years and eight months (range: one to six years). They were all clinically asymptomatic. Associated lesions included mild pulmonary stenosis in two and a small ventricular septal defect in one. In all patients, the
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standard electrocardiogram showed right ventricular hypertrophy, without evidence of abnormal intraventricular conduction delay. Another group of 76 healthy children, aged 2½ to 16 years, with a mean of 8 years, was chosen for comparison.

In all cases echocardiograms were taken with an Echoline 20A ultrasonoscope, coupled with a Cambridge fibreoptic recorder. An electrocardiographic lead with a well-identified Q wave was simultaneously recorded. Systolic time intervals were measured according to techniques already described.9 The pre-ejection period of the systemic ventricle was calculated from the beginning of the Q wave on the electrocardiogram to the opening point of the aortic valve leaflets and identified as right ventricular pre-ejection period (RPEP) for the d-TGA group and left ventricular pre-ejection period (LPEP) for the normals. The corresponding isovolumic contraction time is, for the d-TGA, the difference between Q to aortic valve opening and Q to tricuspid valve closure (RICT) (Fig), and for the normals, the difference between Q to aortic valve opening and Q to mitral valve closure (LICT). The electromechanical delay was calculated as RPEP less RICT (REMD), and LPEP less LICT (LEMD). In the group of normal subjects, the pre-ejection period of the right ventricle was measured from the beginning of the Q wave to the opening point of the pulmonary valves; isovolumic contraction time was calculated as the difference of Q to pulmonary valve opening less Q to tricuspid valve closure, and electromechanical delay was the difference of the pre-ejection period less isovolumic contraction time. The paper speed was set as 100 mm/s, allowing measurement to the nearest 5 ms.10 The average of at least five cardiac cycles was used in the calculations. Measurements were made at similar RR intervals.

In order to select the proper parametric tests for the statistical analysis of our materials, normality of the data was assessed by Lilliefors's test,11 and equality of variances by Bartlett's test.12 The Student's t test was then used to evaluate the difference between right ventricular pre-ejection period of the d-TGA group and left ventricular pre-ejection period of the normal subjects. A one-way analysis of variance with fixed effects was applied to compare the right ventricular isovolumic contraction time and electromechanical delay of patients with d-TGA, to the same intervals calculated on the left and right ventricular echocardiograms of the group of normal children. A contrast analysis using Scheffe's technique13 was applied to determine the means that were significantly different.

In the group of normal subjects, the regression equations of isovolumic contraction time and electromechanical delay versus heart rate yielded weak correlation coefficients \( r = 0.44 \) and \( 0.22 \), respectively. This is in accordance with previous publications on the subject.9 11 For this reason, comparisons were made between values uncorrected for heart rate.

Results

Values found for the components of the pre-ejection period of the systemic right ventricle of the 14 children with d-TGA are given in the Table, as well as the mean values for the left and right ventricles of 76 normal subjects. The mean pre-ejection period of the right ventricle of patients with d-TGA was significantly greater (98 ms, SD = 13) than the same interval for the left ventricle of normal children (79 ms, SD = 10) \( p < 0.001 \). The right ventricular isovolumic contraction time of the d-TGA was not prolonged (26 ms, SD = 5) when compared with values obtained in normal left ventricles (33 ms, SD = 8). On the other hand, the electromechanical delay of the right ventricle in d-TGA averaged 72 ms (SD = 11), while the same interval measured on the left ventricle of normal children was 46 ms (SD = 8). The difference

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Fig. Composite of aortic and tricuspid echograms of a patient with d-TGA showing the technique of measurement of Q to aortic valve opening \( (QAo) \) and Q to tricuspid valve closure \( (QTc) \). Isovolumic contraction time \( = QAo - QTc \).
between these two values is highly significant (p < 0.001).

The average value of 72 ms found for the electromechanical delay of the right ventricle of patients with d-TGA was higher (p < 0.01) than that observed in the right ventricle of our group of normals (60 ms, SD = 9). The same observation can be made for the right ventricular isovolumic contraction time in d-TGA (26 ms, SD = 5) and in normal subjects (13 ms, SD = 8) (p < 0.01).

Table 1 Pre-ejection period and its components in systemic ventricle of patients with d-TGA and in left and right ventricles of 76 normal children

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)</th>
<th>PEP (ms)</th>
<th>ICT (ms)</th>
<th>EMD (ms)</th>
</tr>
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<tr>
<td>Patients with d-TGA</td>
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<td>1</td>
<td>34</td>
<td>100</td>
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<td>14</td>
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<td>110</td>
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<td>80</td>
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<tr>
<td>Mean</td>
<td>7</td>
<td>98 (SD 13)</td>
<td>26 (SD 5)</td>
<td>72 (SD 11)</td>
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<td>Normal children:</td>
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<tr>
<td>Mean LV</td>
<td>8</td>
<td>79 (SD 10)</td>
<td>33 (SD 8)</td>
<td>46 (SD 8)</td>
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<td>Mean RV</td>
<td>—</td>
<td>73 (SD 9)</td>
<td>13 (SD 8)</td>
<td>60 (SD 9)</td>
</tr>
</tbody>
</table>

Abbreviations: PEP, pre-ejection period; ICT, isovolumic contraction time; EMD, electromechanical delay; SD, standard deviation; LV, left ventricle; RV, right ventricle.

Comments

The ability of the right ventricle to perform adequately as a systemic pump is crucial for the long-term prognosis of patients with d-TGA having undergone intra-atrial rerouting of the venous return. Previous angiographic studies have compared volumetric parameters of the right ventricle of patients with d-TGA with those of the right ventricle of normal individuals. Abnormal values were always found. In fact, the question is how the right ventricle in d-TGA compares with the left ventricle of normal individuals. Such an approach has been applied in echocardiographic studies, where right ventricular systolic time intervals of d-TGA were compared with left ventricular intervals of normal subjects. In most of these studies, the conclusion that the right ventricular function was depressed in d-TGA, was based on finding a prolonged right ventricular pre-ejection period. The present study demonstrates that this conclusion is unwarranted. Indeed, the part of the pre-ejection period which closely correlates with measures of contractility, that is the isovolumic contraction time, is, in our group of d-TGA, similar to the same interval measured on the left ventricle of normal subjects. This finding is in full agreement with the normal pressure-velocity indices (dP/dt and derived velocity variables) previously reported for the right ventricle of d-TGA.

The apparent contradiction between our results and the data presented in the angiographic reports may be explained by the work of Peterson and co-workers, who showed that changes in ejection fraction volumes and modification of the contractile state could occur independently of each other.

According to the present study, it is the prolongation of the right ventricular electromechanical delay which is responsible for the longer right ventricular pre-ejection period described in d-TGA when compared with the normal left ventricle. A previous report on time relation of dynamic events in the cardiac chambers, has documented a longer electromechanical delay for the right ventricle than for the left ventricle in normal hearts. Moreover, in our study, the right ventricular electromechanical delay in d-TGA has been found to be longer than that of normal right ventricles. This could be because of the greater myocardial mass of the hypertrophied right ventricle in d-TGA, resulting in a longer ventricular activation time.

We believe that serial evaluation by systolic time intervals of right ventricular function in d-TGA should be based on the measurement of the isovolumic contraction time in preference to that of the complete pre-ejection period.

References

15 Jarmakani JM, Canent RV. Preoperative and post-operative right ventricular function in children with transposition of the great vessels. Circulation 1974; 49 and 50, suppl II: 39-45.

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