Echocardiographic and Hemodynamic Correlation in Transposition of the Great Arteries

SANG C. PARK, M.D., WILLIAM H. NECHES, M.D., J. R. ZUBERBUHLER, M.D., ROBERT A. MATHEWS, M.D., CORA C. LENOX, M.D., AND FREDERICK J. FRICKER, M.D.

SUMMARY Echocardiography was performed in 36 patients with transposition of the great arteries (TGA). Twenty patients were studied before a Mustard operation, 14 patients after operation and two patients both before and after operation. Right ventricular end-diastolic dimension (RVED) was larger than normal in each patient and tended to increase postoperatively. In contrast, the left ventricular end-diastolic dimension (LVED) was significantly reduced postoperatively in all patients. A linear relationship was demonstrated between the ratio of LVED/RVED and the ratio of peak systolic pressures in the left and right ventricles in studies both before and after Mustard operation.

Systolic anterior motion of the mitral valve was observed in 18% of preoperative patients and increased to 44% postoperatively. The incidence of fluttering of the mitral valve increased from 50% to 94% after the operation. Abnormal septal motion was found in 39% of cases. Abnormal movement of the mitral valve and of the interventricular septum seems to be related to a reversed pressure relationship in the ventricles. Shifting of the ventricular septum toward the left ventricle and consequent distortion of the left ventricular cavity and mitral valve apparatus may be responsible for the abnormal echocardiographic findings.

Serial echocardiographic studies may be useful as a noninvasive tool in the assessment of left ventricular pressure or the status of the pulmonary vascular bed in TGA.

IN RECENT YEARS, echocardiography has become a valuable noninvasive tool in assessing cardiac anatomy and function and in estimating ventricular size. Its diagnostic value in patients with transposition of the great arteries (TGA) is well recognized. Echocardiographic studies have documented the occurrence of systolic anterior motion (SAM) as well as fluttering of the mitral valve in patients with TGA. Other authors have found the interventricular septum (IVS) to be thicker than the left ventricular posterior wall (LVPW) and to have paradoxical motion in some patients with TGA. However, studies correlating echocardiographic and hemodynamic data in TGA are limited.

The purpose of this study is to correlate echo findings and hemodynamic data obtained by cardiac catheterization and to explore the mechanism of the various echo findings in patients with TGA, particularly in regard to the motion of the IVS and mitral valve.

Materials and Methods

Patients

The study group consisted of 36 patients with TGA who had cardiac catheterization and echo studies at Children's Hospital of Pittsburgh between July 1975 and December 1976. An echocardiogram was obtained in each patient either at the time of cardiac catheterization or within 24 hours of the procedure. Twenty patients were studied before and 14 patients after a Mustard operation (MU). Another two patients were studied both pre and postoperatively. Group A consisted of 22 patients who had echo and cardiac catheterization performed in the pre-MU period; group B consisted of 16 patients who had echo and cardiac catheterization performed after MU; group C consisted of 10 patients of the above two groups who had both pre and postoperative echoes, only two of whom had postoperative catheterization to date. Associated lesions and hemodynamic data are listed in tables 1, 2 and 3. The age of patients in the preoperative group (A) ranged from 2 months to 10½ years (mean 31 months); in the postoperative group (B) from 21 months to 12½ years (mean 66 months). No patient had an arrhythmia or conduction abnormality on the electrocardiogram.

Echocardiographic Technique

All echoes were obtained utilizing a Hoffrel 101C Ultrasonoscope and Irex 150 Continutrace multi-channel recorder at paper speed of 50 or 100 mm/sec. Most patients were examined with a 3.5 MHz, 13 mm diameter, 5 cm focused transducer. Some older children were studied with a 2.25 MHz, 13 mm diameter, 7.5 cm focused transducer and a 5 MHz, 6.5 mm diameter, unfocused transducer was used for infants.

Each study was performed with the patient in the supine position and the transducer in the third or fourth intercostal space along the left sternal border. A few patients were examined while partially turned to the left lateral position.

The T-scan technique was used to locate the mitral valve and to visualize the IVS and both right and left ventricular free walls simultaneously at a point where both anterior and posterior mitral valve leaflets were identifiable.

Measurements

All echo measurements and analysis were performed in a portion of the echo tracing where the anterior and posterior leaflets of the mitral valve were simultaneously visualized (fig. 1). End-diastolic dimension of the right and left ventricles and thickness of the IVS and LVPW were measured at the onset of the electrocardiographic QRS complex. Care was taken to avoid error in measurement of the IVS by identifying the tricuspid valve in order to differentiate it from actual right septal surface echoes. Septal motion was analyzed during systole as described by Diamond et al. In

From the Department of Pediatrics, Division of Pediatric Cardiology, University of Pittsburgh School of Medicine and Children's Hospital of Pittsburgh, Pennsylvania. Supported in part by a grant from the Western Pennsylvania Heart Association and from the Beaver County Heart Association. Dr. Neches is a teaching scholar of the American Heart Association.

Address for reprints: Sang C. Park, M.D., Division of Pediatric Cardiology, Children's Hospital of Pittsburgh, 125 DeSoto Street, Pittsburgh, Pennsylvania 15213.

Received March 7, 1977; revision accepted September 20, 1977.
normal septal motion the left ventricular surface of the septum moves posteriorly with the onset of ventricular ejection. Anterior (or paradoxical) motion of the IVS during systole is referred to as type A and flattened motion as type B. Amplitude of the mitral valve was measured at maximal excursion from an echocardiogram which recorded both leaflets of the mitral valve simultaneously. When multilayers of the valve were seen in systole (closure of the mitral valve), the mid-portion of the multiple echoes was used as a reference point. The presence or absence of SAM or flattening of the mitral valve was determined in the same portion of the tracing. In view of beat-to-beat variation in the ventricular dimension and in amplitude of the mitral valve in some patients, an average of five consecutive complexes was used as a representative value.

All measurements were done utilizing a caliper and millimeter scale ruler and were subsequently corrected to actual dimension (to the nearest millimeter) by a calibration factor for each tracing.

### Angiographic Studies

During cardiac catheterization biventricular cineangiography and echocardiography were performed simultaneously in three patients with TGA to study ventricular septal motion. Two equal sized NIH catheters were positioned in both ventricles and the patient was positioned in the right anterior oblique position to align the IVS parallel to the horizontal level of the table. An echo transducer was placed in the usual location as described above to visualize the IVS and was hand held to maintain the echo beam perpendicular to the septum. The lateral view cineangiographic camera was used for this study to provide a left anterior oblique image. Contrast medium (Renografin-76, 1.5 ml/kg) was injected through a metal "Y" connector into both catheters simultaneously utilizing a Medrad pressure injector at a rate of 1.5 ml/kg/sec. Cineangiograms were obtained at 60 frames/sec with a QRS indicator on a corner of the film for identification of the electrocardiographic event. The contrast medium generated sufficient echoes to visualize the limits of both ventricular cavities.

The cineangiograms of six other patients with TGA and ventricular septal defect were further evaluated ventricular septal motion.

### Hemodynamic Studies

Data obtained from cardiac catheterization included the ratio of left ventricular systolic pressure to systemic right.

### Table 1. Echocardiographic Findings and Hemodynamic Data in Pre-Mustard Operative Group

<table>
<thead>
<tr>
<th>Pl.</th>
<th>Assoc-</th>
<th>Age (mm)</th>
<th>BSA (m²)</th>
<th>Ventricular dimensions</th>
<th>Mitral valve</th>
<th>Vent. septum</th>
<th>Hemodynamic data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>lation</td>
<td></td>
<td></td>
<td></td>
<td>SAM</td>
<td>Amp.</td>
<td>Thick.</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>13</td>
<td>0.42</td>
<td>24 18 0.75</td>
<td>+</td>
<td>10 B</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>21</td>
<td>0.43</td>
<td>18 24 1.36</td>
<td>+</td>
<td>12 NL</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>54</td>
<td>0.63</td>
<td>19 12 0.63</td>
<td>+</td>
<td>6 NL</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>None</td>
<td>18</td>
<td>0.37</td>
<td>19 12 0.63</td>
<td>+</td>
<td>8 NL</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>None</td>
<td>21</td>
<td>0.45</td>
<td>18 22 1.22</td>
<td>+</td>
<td>8 NL</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>14</td>
<td>0.42</td>
<td>16 20 1.25</td>
<td>+</td>
<td>10 NL</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>Small VSD</td>
<td>24</td>
<td>0.47</td>
<td>22 33 1.36</td>
<td>+</td>
<td>13 NL</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>Small VSD</td>
<td>9</td>
<td>0.35</td>
<td>17 13 0.76</td>
<td>+</td>
<td>7 NL</td>
<td>3</td>
</tr>
<tr>
<td>9</td>
<td>Small VSD</td>
<td>19</td>
<td>0.41</td>
<td>18 14 1.00</td>
<td>+</td>
<td>6 NL</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>Small VSD</td>
<td>42</td>
<td>0.65</td>
<td>25 19 0.76</td>
<td>+</td>
<td>11 NL</td>
<td>4</td>
</tr>
</tbody>
</table>

*Intermittent occurrence of systolic anterior motion of mitral valve.
Abbreviations: A = anterior systolic motion of the ventricular septum; Amp. = amplitude; B = base ventricular septal motion; BSA = body surface area; LVED = left ventricular end-diastolic dimension; ΔLV-PA = peak systolic pressure gradient between left ventricle and pulmonary artery; LV/PW = left ventricular posterior wall thickness; NL = normal septal motion, PLV = pulmonary artery pressures; PLV = peak left ventricular pressures; PLV/PRV = ratio of peak systolic pressures between left and right ventricles; PSV = pulmonic stenosis; Qp/Qs = pulmonary to systemic blood flow ratio; RV = right ventricular end-diastolic dimension; SAM = systolic anterior motion; SPS = subpulmonic stenosis; Thick. = thickness; Vent. = ventricular; VSD = ventricular septal defect.

### Table 2. Echocardiographic Findings and Hemodynamic Data in Post-Mustard Operative Group

<table>
<thead>
<tr>
<th>Pl.</th>
<th>Assoc-</th>
<th>Age (mm)</th>
<th>BSA (m²)</th>
<th>Ventricular dimensions</th>
<th>Mitral valve</th>
<th>Vent. septum</th>
<th>Hemodynamic data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>lation</td>
<td></td>
<td></td>
<td></td>
<td>SAM</td>
<td>Amp.</td>
<td>Thick.</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>62</td>
<td>0.75</td>
<td>22 16 0.73</td>
<td>+</td>
<td>9 NL</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>21</td>
<td>0.69</td>
<td>20 17 1.07</td>
<td>+</td>
<td>8 NL</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>60</td>
<td>0.70</td>
<td>23 16 0.70</td>
<td>+</td>
<td>9 B</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>None</td>
<td>54</td>
<td>0.63</td>
<td>19 14 0.36</td>
<td>+</td>
<td>8 NL</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>None</td>
<td>46</td>
<td>0.67</td>
<td>22 14 0.64</td>
<td>+</td>
<td>8 B</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>90</td>
<td>0.57</td>
<td>27 23 0.83</td>
<td>+</td>
<td>9 A</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>None</td>
<td>78</td>
<td>0.92</td>
<td>21 18 0.86</td>
<td>+</td>
<td>6 A</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>PSV</td>
<td>110</td>
<td>1.25</td>
<td>37 27 0.70</td>
<td>+</td>
<td>13 B</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>PSV</td>
<td>44</td>
<td>0.62</td>
<td>25 18 1.06</td>
<td>+</td>
<td>10 NL</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>PSV</td>
<td>56</td>
<td>0.73</td>
<td>34 36 1.06</td>
<td>+</td>
<td>18 NL</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>PSV</td>
<td>65</td>
<td>0.80</td>
<td>28 16 0.80</td>
<td>+</td>
<td>9 NL</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>PSV &amp; PDA</td>
<td>79</td>
<td>0.77</td>
<td>31 13 0.42</td>
<td>+</td>
<td>10 B</td>
<td>4</td>
</tr>
<tr>
<td>13</td>
<td>PSV &amp; PSV</td>
<td>18</td>
<td>0.41</td>
<td>21 21 1.02</td>
<td>+</td>
<td>9 NL</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>PSV &amp; PSV</td>
<td>138</td>
<td>1.12</td>
<td>30 45 1.50</td>
<td>+</td>
<td>16 B</td>
<td>6</td>
</tr>
<tr>
<td>15</td>
<td>PSV &amp; PDA</td>
<td>51</td>
<td>0.59</td>
<td>17 17 1.00</td>
<td>+</td>
<td>13 NL</td>
<td>5</td>
</tr>
<tr>
<td>16</td>
<td>PSV &amp; PDA</td>
<td>68</td>
<td>0.71</td>
<td>34 26 0.76</td>
<td>+</td>
<td>11 A</td>
<td>4</td>
</tr>
</tbody>
</table>

*Intermittent occurrence of systolic anterior motion of mitral valve.
Abbreviations: A = anterior systolic motion of the ventricular septum; Amp. = amplitude; B = base ventricular septal motion; BSA = body surface area; LVED = left ventricular end-diastolic dimension; ΔLV-PA = peak systolic pressure gradient between left ventricle and pulmonary artery; LV/PW = left ventricular posterior wall thickness; NL = normal septal motion, PLV = pulmonary artery pressures; PLV/PRV = ratio of peak systolic pressures between left and right ventricles; PSV = pulmonic stenosis; Qp/Qs = pulmonary to systemic blood flow ratio; RV = right ventricular end-diastolic dimension; SAM = systolic anterior motion; SPS = subpulmonic stenosis; Thick. = thickness; Vent. = ventricular; VSD = ventricular septal defect.
ventricular systolic pressure, the peak systolic pressure gradient between the left ventricle and pulmonary artery and the pulmonary to systemic blood flow rate (Qp/Qs). Mean Qp/Qs was 2.2:1 in pre-MU group A and 1:1 in post-MU group B.

**Results**

**Correlation Between the Ventricular Dimensions and Hemodynamics**

Right ventricular end-diastolic dimensions (RVED) were strikingly larger than normal in both pre and postoperative groups. In contrast, left ventricular end-diastolic dimensions (LVED) were less than the 5th percentile of normal in all but three preoperative patients and two postoperative patients.

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Assoc. lesion</th>
<th>Age at op. (mo)</th>
<th>Op. status</th>
<th>Age (mo)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>BSA (m²)</th>
<th>RVED (mm)</th>
<th>LVED (mm)</th>
<th>LVED/LVED</th>
<th>FLV/FRV (%)</th>
<th>Qp/Qs</th>
<th>ALV-PA (mm Hg)</th>
<th>Vent. dimension</th>
<th>Hemodynamic data</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>14</td>
<td>Pre</td>
<td>13</td>
<td>10.0</td>
<td>72</td>
<td>0.42</td>
<td>24</td>
<td>18</td>
<td>0.75</td>
<td>36</td>
<td>20</td>
<td>3:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>23</td>
<td>Pre</td>
<td>27</td>
<td>14.0</td>
<td>90</td>
<td>0.52</td>
<td>28</td>
<td>15</td>
<td>0.64</td>
<td>36</td>
<td>20</td>
<td>2:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>58</td>
<td>Pre</td>
<td>54</td>
<td>9.6</td>
<td>78</td>
<td>0.44</td>
<td>18</td>
<td>24</td>
<td>1.10</td>
<td>44</td>
<td>27</td>
<td>2:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Small VSD</td>
<td>21</td>
<td>Pre</td>
<td>21</td>
<td>10.9</td>
<td>80</td>
<td>0.47</td>
<td>22</td>
<td>20</td>
<td>0.91</td>
<td>32</td>
<td>10</td>
<td>1.7:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Small VSD &amp; PDA</td>
<td>47</td>
<td>Pre</td>
<td>42</td>
<td>12.2</td>
<td>91</td>
<td>0.55</td>
<td>55</td>
<td>29</td>
<td>0.80</td>
<td>25</td>
<td>14</td>
<td>3:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>SPS</td>
<td>24</td>
<td>Pre</td>
<td>20</td>
<td>7.7</td>
<td>81</td>
<td>0.41</td>
<td>12</td>
<td>20</td>
<td>1.67</td>
<td>62</td>
<td>32</td>
<td>1:4:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Small VSD &amp; SPS</td>
<td>15</td>
<td>Pre</td>
<td>10</td>
<td>7.8</td>
<td>74</td>
<td>0.38</td>
<td>15</td>
<td>19</td>
<td>1.27</td>
<td>81</td>
<td>49</td>
<td>1.4:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>VSD &amp; PSV</td>
<td>17</td>
<td>Pre</td>
<td>12</td>
<td>7.0</td>
<td>72</td>
<td>0.36</td>
<td>14</td>
<td>20</td>
<td>1.45</td>
<td>63</td>
<td>30</td>
<td>2:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>VSD &amp; PSV</td>
<td>67</td>
<td>Pre</td>
<td>16</td>
<td>16.1</td>
<td>104</td>
<td>0.68</td>
<td>26</td>
<td>35</td>
<td>1.35</td>
<td>100</td>
<td>64</td>
<td>1:3:1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>VSD &amp; PSV</td>
<td>84</td>
<td>Pre</td>
<td>82</td>
<td>17.5</td>
<td>115</td>
<td>0.75</td>
<td>14</td>
<td>22</td>
<td>2.29</td>
<td>109</td>
<td>100</td>
<td>0.3:1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 3. Echocardiographic Findings and Hemodynamic Data in Pre and Post Mustard Operative Group**

On review of the pre and postoperative echoes of 10 patients (group C), the preoperative RVED was larger than normal in each patient and further increase of the RVED was noted after MU in all but patient 2 (fig. 2). In contrast, eight of 10 patients had an LVED below normal preoperatively and further reduction of LVED occurred after operation. A linear relationship was found between the ratio of the LVED

![FIGURE 1. Measurement of right and left ventricular end-diastolic dimensions (RVED and LVED) and thickness of the interventricular septum (IVS) and left ventricular posterior wall (LVPW) were done at the beginning of the QRS. MVA = mitral valve amplitude.](image1.png)

![FIGURE 2. Change of ventricular dimensions after Mustard operation. Right ventricular end-diastolic dimension (RVED) and left ventricular end-diastolic dimension (LVED) are plotted against body surface area (BSA) in m². Open circles indicate preoperative status and closed circles indicate postoperative status. Dotted lines indicate 5th and 95th percentiles of normal RVED and LVED after Epstein et al. (these values are population percentiles rather than standard deviations.](image2.png)
Figure 3. Relationship between the ratio of left and right ventricular end-diastolic dimensions (LVED/RVED) and the peak systolic pressure ratio of left and right ventricles (LV/RV in %). Open circles indicate preoperative patients and closed circles indicate postoperative patients. The dotted line (a) is the regression line for pre-Mustard operation group and solid line (b) is the regression line for post-Mustard operation group.

and RVED (LVED/RVED) and the ratio of the peak systolic pressure of the left and right ventricle (LV/RV) (fig. 3). The correlation coefficients were \( r = 0.85 \) for group A and \( r = 0.88 \) for group B respectively. No correlation was found between the LVED/RVED and the pulmonary to systemic flow ratio in the preoperative group (A). However, LVED/RVED was slightly greater in the preoperative group (A). This difference may be related to the higher pulmonary flow (Qp/Qs 2.2:1) and consequent larger LVED.

Ventricular Septum

Considerable variation of ventricular septal thickness in different portions of the echo was noted in most patients. In a few patients, a markedly thickened IVS was seen near the pulmonary anulus or upper portion of IVS. The measurement of IVS from the reference portion of echo showed that the IVS thickness was either less than or equal to the thickness of the LVPW. No patient had a consistently thick IVS on echocardiogram.

At the level of the mitral valve, IVS motion was normal in 23 patients (61%) and flat (type B) in 12 (31%). The remaining 3 patients (8%), from the post-MU group, showed the IVS moving anteriorly (type A). However, all patients with types A or B ventricular septal motion demonstrated normal septal motion near the apex.

Figure 4 demonstrates a simultaneous biventricular cineangiogram and echo in a patient after MU. The IVS bulges into the left ventricle during systole forming a crescent-shaped left ventricular cavity. The pulmonary anulus and upper portion of the IVS move anteriorly and inferiorly and the mid and lower portions of the septum move posteriorly in systole. Therefore, the IVS motion and its apparent thickness could be altered by changing the angulation of the echo transducer. As is seen in normal individuals, posterior septal motion was recorded in the lower portion of the septum while anterior motion during systole was present in the upper portion of the IVS near the pulmonary anulus. No part of the IVS was unusually thick as seen on the angiogram. However, a markedly thickened IVS was frequently noted at the upper portion of the IVS on echo. The thickened ventricular septum appeared to be due to oblique penetration of the septum by the echo beam.

Selective cineangiograms done in six patients with TGA and ventricular septal defect were reviewed to evaluate further the ventricular septal motion. It was usually difficult to demonstrate the central portion of the ventricular septum on selective left ventriculograms in the lateral view due to oblique orientation of the IVS to the lateral plane. Thus the central portion of the IVS is overlapped by the peripheral portion of the ventricular cavity in this view (fig. 5). Injection of contrast medium into the vena cava or left atrium usually provided better visualization of the ventricular septum. In each patient, the cineangiographic findings
were similar to those shown in the biventricular cineangiogram (fig. 4). Patients with high left ventricular pressure had less bulging of the IVS into the left ventricle than did patients with low left ventricular pressure (fig. 6). Angiographically, no patient demonstrated anterior motion in the lower portion of the IVS during systole.

The Mitral Valve

The overall amplitude of mitral valve excursion was generally diminished in both groups. There was close correlation between the amplitude of the mitral valve and LVED, and both varied with respirations in post-MU

Figure 5. A, B, C) Cineangiograms of a patient with transposition of the great arteries, a small ventricular septal defect and low pulmonary artery and left ventricular pressures. A) Lateral view of selective right ventriculogram shows bulging of the ventricular septum toward the left ventricular side (arrows). B) Left anterior oblique view of the left ventriculogram shows a narrowing of the left ventricular outflow due to bulging of the ventricular septum (arrows). C) Lateral view of the left ventricular cineangiogram failed to demonstrate clearly the ventricular septum due to overlapping of the left ventricular septal edge. D, E, F) Cineangiograms of the patient after Mustard operation for transposition of the great arteries. D) Lateral view of selective right ventriculogram demonstrates posterior bulging of the ventricular septum (arrows). E) Lateral view following injection of contrast material into the superior vena cava. A marked bulging of the interventricular septum into the left ventricle is seen. F) Selective left ventriculogram (lateral view) fails to adequately demonstrate ventricular septum (see fig. 5C).

Figure 6. Simultaneous biventricular cineangiograms. A) Patient with normally related great arteries, a small ventricular septal defect and normal right ventricular pressures. Interventricular septum (IVS) bulges into the right ventricle. B) Post-Mustard operation patient with severe residual pulmonic stenosis and systemic pressure in the left ventricle. IVS lies almost straight. C) Another post-Mustard operation patient with low left ventricular pressure. IVS bulges into the left ventricle.
patients; inspiration tended to increase and expiration to decrease LVED and mitral valve amplitude (fig. 7).

The incidence of SAM of the mitral valve increased from 18% to 44% after MU. In over half of the cases (seven of 11) with SAM of the mitral valve, the finding was intermittent. In post-MU patients with SAM of the mitral valve some respiratory variation was noted. There was no correlation between SAM of the mitral valve and the presence of associated cardiac lesions. Ten of the 11 patients with SAM of the mitral valve had left ventricular pressure less than 40% of systemic pressure. The incidence of SAM of the mitral valve was significantly higher in those patients with normal (low) left ventricular pressure than those with high left ventricular pressure, regardless of the cause of the high left ventricular pressure (fig. 8).

The incidence of fluttering of the mitral valve increased significantly from 50% preoperatively to 94% after MU. Some degree of fluttering of the mitral valve was noted post-operatively in all but one patient who had severe residual subpulmonic stenosis and a residual ventricular septal defect (group B, #14). In the pre-MU group, the incidence of mitral valve fluttering was less in patients with high left ventricular pressures.

**Discussion**

Ventricular dimensions may be altered by pressure or volume overload or by a change in myocardial contractility or compliance. The morphological characteristics of both ventricles are less changeable by hemodynamic alteration. The ventricular septum, however, seems to be particularly vulnerable to alterations in the relative pressure in the two ventricles. TGA is a unique condition with reversal of ventricular function. This explains why both echo and angiographic studies have demonstrated a distinctly larger RVED than LVED in uncomplicated TGA.

Abnormal septal motion occurs with right ventricular volume overload but is not limited to patients with this hemodynamic abnormality. The mechanism of abnormal septal motion has been extensively investigated. A cross-sectional echocardiographic study by Weyman et al. in patients with right ventricular overload demonstrated that the dilated right ventricle not only displaced the IVS posteriorly during diastole but also resulted in a change in the configuration of the left ventricle. Our study with TGA has shown that the position of the IVS can be greatly influenced by the relative pressure in the two ventricles. Although high pulmonary blood flow and left ventricular volume overload may be contributing factors in maintaining a larger left ventricular cavity (LVED), the magnitude of pulmonary blood flow in this study seemed to be a less influential factor than the pressure difference in the two ventricles. Our studies showed a linear relationship between the ratio of peak systolic pressure in the left and right ventricles and LVED/RVED with a high correlation coefficient permitting indirect estimation of the pressure relationship between the two ventricles. LVED/RVED ratios in pre-MU patients were slightly larger than those of post-MU group, presumably related to a larger pulmonary blood flow (mean

**FIGURE 7.** Echocardiogram in a patient following Mustard operation demonstrating a distinct change in the amplitude of the mitral valve excursion and in the left ventricular end-diastolic dimension with respiration (A). The change was more prominent with deep respiratory effort (B). INSPI = inspiration; EXP = expiration.
Figure 8. Echocardiographic characteristics of the mitral valve in patients with transposition of the great arteries and differing left ventricular pressures. A) Patient with a large ventricular septal defect (VSD) and pulmonary artery banding (PB) who had systemic pressure in the left ventricle. Normal mitral valve motion. B) Patient with valvular pulmonic stenosis (PSV). Direct contact of anterior leaflet of the mitral valve with the ventricular septum but no systolic anterior motion (SAM). C and D) Patients with low left ventricular pressure demonstrating marked and consistent SAM and low amplitude of the mitral valve excursion.

Qp/Qs = 2.2:1 and concomitant left ventricular volume overload in the former group. Therefore, a higher Qp/Qs ratio may affect the ability to predict the ventricular pressure index in this group.

The status of the pulmonary vascular bed has been previously assessed in adults and in the pediatric age group by echo determinations of systolic time intervals. Our study suggests that the serial echocardiographic estimation of the left ventricular pressure by measurement of LVED/RVED ratio may be another useful noninvasive means of following the status of the pulmonary vascular bed in patients with TGA.

Motion of the IVS on echo can be extremely variable as a result of the selection of the reference point and/or angulation of the transducer as indicated by Hagan et al. Accurate determination of IVS motion by echo in patients with TGA is difficult because actual ventricular septal motion varies with the portion of the septum visualized; the septum near the pulmonary anulus moves anteriorly in systole but the portion near the apex moves posteriorly as it does in normal subjects (fig. 4). In addition, accurate measurements of IVS thickness were frequently hampered by unusual positioning of the IVS and overlapping of the atrioventricular valve tissue onto the septum. An abnormal IVS/LVPW ratio has been reported in patients with TGA and has also been found in various congenital heart defects other than asymmetric septal hypertrophy (ASH) or idiopathic hypertrophic subaortic stenosis (IHSS). The high ratio in some of these patients may be attributed to an oblique orientation of the echo beam to the IVS.

Echocardiographic SAM of the mitral valve has been a useful diagnostic sign in IHSS and has also been described in patients with TGA with or without left ventricular obstruction. In patients with IHSS, SAM of the mitral valve can be seen without the presence of a left ventricular outflow gradient. The mechanism of this phenomenon has not been completely explained. Nanda et al. suggested a correlation between SAM of the mitral valve and left ventricular outflow obstruction in patients with TGA. However, in our series, SAM of the mitral valve was more frequently found in patients with low left ventricular pressure and without significant left ventricular outflow obstruction. In such patients the IVS was seen to bulge into the left ventricle during systole on cineangiography. This suggested that the smaller left ventricular dimension permitted closer apposition of the mitral valve to the ventricular septum. The resultant high velocity of flow in the left ventricular outflow area may have caused further anterior displacement of the mitral valve in systole by the Bernoulli effect, mimicking ASH or IHSS (fig. 9).

Although SAM of the mitral valve is seen in both TGA and IHSS, the IVS morphology is quite different in the two entities. In patients with IHSS, diffuse septal hypertrophy can be demonstrated by echo as well as angiography. In contrast, our study indicates that the ventricular septum in TGA is usually normal in thickness and is merely shifted to the left ventricular side. The apparent thickness of the IVS on echo in some patients with TGA is largely due to obliquity of the echo beam through the septum.

Fluttering of the mitral valve after MU has been at-
Contributed to low compliance and small volume of the systemic venous atrium and high velocity of systemic venous flow through the mitral valve. However, this seems unlikely since fluttering of the mitral valve also is seen in patients prior to the MU and may be attenuated or disappear in some post-MU patients during inspiration, which should increase systemic venous return. Both fluttering and SAM of the mitral valve may be due largely to bulging of the IVS into the left ventricular cavity with consequent distortion of the mitral valve apparatus. In some patients pre-existing mitral valve abnormalities such as those described by Rosenquist et al. may also contribute to abnormal echo findings.

In conclusion, abnormal motion of the mitral valve and IVS in TGA seems to be related to an abnormal pressure relationship between the two ventricles. Shifting of the IVS with consequent distortion of the left ventricular cavity and the mitral valve apparatus may be a major cause of these abnormalities. Cross-sectional echocardiographic studies might be helpful in confirming or refuting this hypothesis.

Acknowledgment

The authors gratefully acknowledge the technical assistance of Jane Perry, Virginia Curlee and Anita Miller and the secretarial assistance of Beverly Collins.

References

Echocardiographic and hemodynamic correlation in transposition of the great arteries.
S C Park, W H Neches, J R Zuberbuhler, R A Mathews, C C Lenox and F J Fricker

_Circulation._ 1978;57:291-298
doi: 10.1161/01.CIR.57.2.291

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/57/2/291