An Evaluation of Right and Left Ventricular Function After Anatomical Correction and Intra-Atrial Repair Operations for Complete Transposition of the Great Arteries

Robin P. Martin, MRCP, Shakeel A. Qureshi, MRCP, Jose A. Ettedgui, MD, Edward J. Baker, MD, Bernie J. O’Brien, PhD, Philip B. Deverall, FRCS, Alan K. Yates, FRCS, Michael N. Maisey, FRCP, Rosemary Radley-Smith, FRCP, Michael Tynan, FRCP, and Magdi H. Yacoub, FRCS

Anatomical correction of complete transposition of the great arteries has the potential advantage over intra-atrial repair in that the left ventricle becomes the systemic pump. To investigate the importance of this, we evaluated right and left ventricular function in 21 patients after anatomical correction and in 21 patients after Mustard or Senning operations. First-pass and equilibrium-gated radionuclide angiography were used to measure right and left ventricular ejection fractions between 17 and 78 (mean, 47) months after anatomical correction and between 3 and 187 (mean, 67) months after intra-atrial repair. The mean age of the patient groups at the time of study was 52 and 84 months, respectively. The right ventricular ejection fraction ranged from 35% to 78% (mean, 58%) in patients after anatomical correction and from 27% to 68% (mean, 51%) after intra-atrial repair (p=0.066). The left ventricular ejection fraction ranged from 39% to 74% (mean, 58%) after anatomical correction and from 35% to 74% (mean, 58%) after intra-atrial repair (p=0.86). The mean right and left ventricular ejection fractions of both groups were significantly lower than those of normal children. Individuals with systemic ventricular dysfunction were identified after both types of operations; however, symptomatic dysfunction occurred only after intra-atrial repair (p=0.24). (Circulation 1990;82:808–816)

The occurrence of systemic ventricular dysfunction in a significant number of patients after Mustard and Senning operations1-3 has been documented. The cause of this dysfunction remains uncertain. It has been suggested that the right ventricle is unable to sustain systemic pressure loads in the long-term because of various morphological features4 or preoperative hypoxemia and intraoperative factors.5 Some or all of these factors may influence the left ventricle also. This has stimulated the development of anatomical correction for complete transposition of the great arteries (TGA).4,6 Postoperative studies have shown good early left ventricular function after anatomical correction,7-10 but little information is available on ventricular function in the medium-term.11,12

The purpose of this study was to investigate right and left ventricular function in patients who had undergone anatomical correction or intra-atrial repair operations for TGA by the same investigative methods for both groups of patients. We attempted to identify the factors contributing to right ventricular dysfunction after Mustard and Senning operations and to investigate whether left ventricular dysfunction occurred after anatomical correction.

**Methods**

**Anatomical Correction**

Twenty-one patients who had undergone anatomical correction of TGA at Harefield Hospital were studied. The age of the patients at the time of the study ranged from 17 to 85 months (mean, 51.8 months). The studies were performed between 17 and 78 months after operation (mean, 47 months).
The technique of anatomical correction has been described previously. All operations were performed by one surgeon (M.H.Y.). The age of patients at the time of anatomical correction ranged from 3 days to 19 months (mean, 4.7 months). Seventeen patients had TGA with an intact ventricular septum of whom 10 had undergone primary anatomical correction during the neonatal period. The remaining seven had undergone a two-stage correction with a preliminary pulmonary artery banding to “prepare” the left ventricle for subsequent anatomical correction. Anatomical correction combined with closure of a large ventricular septal defect had been performed in an additional four patients. All operations had been performed under profound hypothermia to an esophageal temperature ranging from 10° to 18° C. In 12 patients, repair was performed during a period of circulatory arrest lasting between 51 and 72 minutes. In the remainder, profound hypothermia was combined with low-flow cardiopulmonary bypass and either no circulatory arrest (six patients) or short periods of circulatory arrest of 12, 20, and 28 minutes in three patients.

Intra-Atrial Repair

Twenty-one patients who had undergone intra-atrial repair for TGA were studied. The studies were performed between 3 and 187 months after operation (mean, 67 months). The age of the patients at the time of study ranged from 13 to 198 months (mean, 84 months).

Twenty patients had TGA with an intact ventricular septum, 10 of whom had undergone Senning operation, and 10 had undergone Mustard operation. Mustard operation combined with closure of multiple ventricular septal defects was performed in the remaining patient. The operations were performed by one of two surgeons (P.B.D. or A.K.Y.). The age of the patients at operation ranged from 1 to 116 months (mean, 16.6 months). Data on operative variables such as ischemic arrest and cardiopulmonary bypass times were available in 16 patients. In 13 of these, repair was performed under profound hypothermia to a temperature ranging from 15° to 18° C and during total circulatory arrest for periods lasting between 30 and 66 minutes. In three patients, cardiopulmonary bypass and moderate hypothermia to a temperature ranging from 25° to 28° C were used.

Patient Selection

The 42 patients included in this study were recruited from two different centers: one (Harefield Hospital) where all children with TGA were treated by anatomical correction and the other (Guy’s Hospital) where all children were treated by intra-atrial repair. The 21 patients that had undergone anatomical correction were selected from a population of 105 survivors of anatomical correction at Harefield Hospital. The patients chosen resided close enough to the Department of Nuclear Medicine, Guy’s Hos-
obstruction to the superior vena caval limb of the systemic venous baffle. Concurrent electrocardiographic examination showed that 16 patients were in sinus rhythm, two in junctional rhythm, and one in atrial fibrillation.

**Radionuclide Angiography**

First-pass and equilibrium-gated blood pool scans were obtained in each patient. In all patients, right ventricular function has been assessed by the first-pass technique and left ventricular function by the equilibrium-gated scan. Patients less than 5 years old were sedated with chloral hydrate 60 mg/kg body wt. Occasionally, an additional, intravenous dose of diazepam was required for adequate sedation. Technetium-99m–labeled autologous red blood cells were injected as a rapid compact bolus through a peripheral venous cannula. The dose of the radionuclide administered was 430 MBq/m². The first-pass study was acquired in the anterior projection for a period of 20–30 seconds. A single crystal gamma camera with a general purpose collimator was used for data acquisition. The data were stored in list mode with a synchronous electrocardiogram buffer on a magnetic disk. After acquisition of the first-pass study, equilibrium-gated radionuclide angiograms were obtained in the anterior and left anterior oblique projections. The data were collected in 18 frames/cardiac cycle, and 3.5×10⁶ counts were acquired for each projection.

Right ventricular ejection fraction was calculated from the first-pass angiogram with a previously described method.¹⁶ The first-pass data were reconstructed as a dynamic study of 1 frame/sec. The timing of the passage of the radionuclide bolus through the right ventricle was observed. Right ventricular transit usually lasted between 3 and 5 seconds. In patients after intra-atrial repair operations, this phase occurred after the radionuclide tracer had passed through the pulmonary circulation, whereas in patients after anatomical correction, a normal sequence of cardiac chamber filling was present. An 18-frame/cardiac cycle electrocardiogram-gated angiogram was generated from the right ventricular phase of the first-pass study. A region of interest was then drawn around the right ventricle on the first frame of the gated study, and a time-activity curve was generated. The end-systolic and end-diastolic frames were chosen as the frames with the minimum and maximum counts, respectively. By subtraction of the end-systolic from the end-diastolic frame, a stroke-volume image was produced. This was used to draw the diastolic right ventricular region of interest because it clearly demarcated the border between the right ventricle and right atrium. A separate systolic region of interest was then drawn on the end-systolic frame taking care to exclude right atrial activity from this region. A systolic background region of interest was drawn as a horseshoe around the apex of the right ventricle. The right ventricular ejection fraction was calculated from the background-corrected end-diastolic and end-systolic counts of the first-pass angiogram.

The first-pass studies were separately analyzed for the presence of a systemic-to-pulmonary shunt.¹⁷ As mentioned previously, patients with a significant shunt have been excluded from this study because of interference with the drawing of the right ventricular regions of interest.

In our laboratory, the normal range for the right ventricular ejection fraction for children is 50–78% (mean±SD, 64±7%).¹⁶

Left ventricular ejection fraction was determined from the electrocardiogram-gated equilibrium scans acquired in the left anterior oblique projection.¹⁶ Analysis of the studies was performed with an end-diastolic left ventricular region of interest drawn from the stroke volume image in a similar manner to the right ventricular analysis. A computer-derived diastolic region of interest lateral and inferior to the left ventricle was used for background correction.

In our laboratory, the normal range for left ventricular ejection fraction for children is 55–76% (mean±SD, 65.6±5.4%).¹⁸

In patients who had undergone anatomical correction of TGA, regional wall motion of the left ventricle was evaluated by visual inspection of cine buffers of the scans acquired in the anterior and left anterior oblique projections and by reconstruction of phase images.¹⁶

**Critique of the Radionuclide Investigation**

Radionuclide angiography is a well-established technique for the assessment of right and left ventricular function. However, it does not take into account the different loading conditions. It also does not rely on assumptions of ventricular shape, which are inherent in echocardiographic measurements of right and left ventricular ejection fraction. The loading conditions of the systemic ventricles after both types of repair should not be different, and in clinical practice, these methods of measuring ejection fractions are extensively used. The particular technique used in this study has been previously validated at our institution in patients with congenital heart disease. Reproducibility studies have shown good intraobserver and interobserver correlations. For the first-pass technique, the intraobserver correlation coefficient is 0.96¹⁹ and the interobserver correlation coefficient is 0.93.¹⁶ The latter is based on analysis of 20 first-pass angiograms by two observers, and the mean±SD difference between the two ejection fractions was 3±2%. Similar intraobserver and interobserver studies for the equilibrium technique gave correlation coefficients of 0.96 and 0.91, respectively.²⁰

Radionuclide angiography requires the placement of an intravenous cannula but is otherwise noninvasive and can be performed on an outpatient basis. It is, thus, ideal for serial monitoring of ventricular function during postoperative follow-up and precludes the need for cardiac catheterization.
Table 1. Results After Anatomical Correction

<table>
<thead>
<tr>
<th>Patient</th>
<th>Operation type</th>
<th>Age at operation (mo)</th>
<th>Time from operation (mo)</th>
<th>RVEF (%)</th>
<th>LVEF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Primary</td>
<td>1</td>
<td>40</td>
<td>-</td>
<td>58</td>
</tr>
<tr>
<td>2</td>
<td>Primary</td>
<td>1.2</td>
<td>35</td>
<td>66</td>
<td>65</td>
</tr>
<tr>
<td>3</td>
<td>Primary</td>
<td>0.3</td>
<td>26</td>
<td>35</td>
<td>55</td>
</tr>
<tr>
<td>4</td>
<td>Two stage</td>
<td>6</td>
<td>44</td>
<td>54</td>
<td>56</td>
</tr>
<tr>
<td>5</td>
<td>Two stage</td>
<td>13</td>
<td>59</td>
<td>48</td>
<td>56</td>
</tr>
<tr>
<td>6</td>
<td>Primary</td>
<td>0.5</td>
<td>36</td>
<td>64</td>
<td>54</td>
</tr>
<tr>
<td>7</td>
<td>Primary</td>
<td>0.7</td>
<td>28</td>
<td>68</td>
<td>68</td>
</tr>
<tr>
<td>8</td>
<td>Primary</td>
<td>0.4</td>
<td>39</td>
<td>47</td>
<td>60</td>
</tr>
<tr>
<td>9</td>
<td>Primary</td>
<td>0.2</td>
<td>17</td>
<td>51</td>
<td>54</td>
</tr>
<tr>
<td>10</td>
<td>Two stage</td>
<td>5</td>
<td>78</td>
<td>51</td>
<td>60</td>
</tr>
<tr>
<td>11</td>
<td>Primary+VSD</td>
<td>7</td>
<td>64</td>
<td>49</td>
<td>61</td>
</tr>
<tr>
<td>12</td>
<td>Primary+VSD</td>
<td>3</td>
<td>35</td>
<td>75</td>
<td>64</td>
</tr>
<tr>
<td>13</td>
<td>Two stage+VSD</td>
<td>12</td>
<td>72</td>
<td>47</td>
<td>62</td>
</tr>
<tr>
<td>14</td>
<td>Primary</td>
<td>0.2</td>
<td>33</td>
<td>78</td>
<td>69</td>
</tr>
<tr>
<td>15</td>
<td>Two stage</td>
<td>8</td>
<td>63</td>
<td>66</td>
<td>64</td>
</tr>
<tr>
<td>16</td>
<td>Two stage</td>
<td>19</td>
<td>56</td>
<td>56</td>
<td>39</td>
</tr>
<tr>
<td>17</td>
<td>Primary</td>
<td>0.1</td>
<td>36</td>
<td>-</td>
<td>74</td>
</tr>
<tr>
<td>18</td>
<td>Two stage</td>
<td>10</td>
<td>75</td>
<td>64</td>
<td>49</td>
</tr>
<tr>
<td>19</td>
<td>Two stage</td>
<td>6</td>
<td>44</td>
<td>61</td>
<td>52</td>
</tr>
<tr>
<td>20</td>
<td>Primary</td>
<td>0.3</td>
<td>41</td>
<td>44</td>
<td>40</td>
</tr>
<tr>
<td>21</td>
<td>Primary+VSD</td>
<td>5</td>
<td>68</td>
<td>76</td>
<td>63</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>4.7</td>
<td>47</td>
<td>58</td>
<td>58</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>9</td>
</tr>
</tbody>
</table>

RVEF, right ventricular ejection fraction; LVEF, left ventricular ejection fraction; primary, anatomical correction without previous pulmonary artery banding; two stage, two stage anatomical correction; +VSD, operative repair combined with closure of a ventricular septal defect.

Statistical Analysis

Population means were compared by the Student’s t test. Where appropriate, the χ² or the Fisher’s exact test was used to compare differences between groups. Linear regression analysis was used to correlate different variables. The null hypothesis was rejected if p was less than 0.05.

Results

The overall results for the two groups are summarized in Tables 1 and 2.

Systemic Ventricular Function After Intra-Atrial Repair and Anatomical Correction

The systemic ventricular ejection fraction after intra-atrial repair (mean±SD, 51±11%; range, 27–68%) was significantly lower than that after anatomical correction (mean±SD, 58±9%; range, 39–74%) (p=0.017). However, eight (38%) patients in the intra-atrial repair group and six (29%) in the anatomical correction group had subnormal systemic ventricular ejection fractions (p=0.74). Subnormal ventricular ejection fraction was defined as less than 50% for the right ventricle and less than 55% for the left ventricle. The frequency distribution of the ejection fraction after both types of repair is shown in Figures 1 and 2.

Influence of Type of Operation on Systemic Ventricular Function

In patients after intra-atrial repair, the right ventricular ejection fraction in the Mustard subgroup ranged from 27% to 65% (mean±SD, 48±11%), and that in the Senning subgroup ranged from 41% to 68% (mean±SD, 54±10%) (p=0.22). In patients after primary neonatal anatomical correction, the left ventricular ejection fraction ranged from 40% to 74% (mean±SD, 60±10%); after primary correction combined with closure of ventricular septal defect, it ranged from 61% to 64% (mean±SD, 63±1%) and after two-stage correction, it ranged from 39% to 64% (mean±SD, 54±8%). There was a significant difference in left ventricular ejection fraction between those who underwent two-stage operation and those who had primary correction with closure of ventricular septal defect (p=0.029). However, only four patients were in the latter group. Although the left ventricular ejection fraction was higher in patients after primary correction than after a two-stage repair, this difference was not significant.

Influence of Age at Operation on Systemic Ventricular Function

There was no significant correlation (r=0.04) between the age at operation and subsequent right...
### Table 2. Results After Intra-Atrial Repair

<table>
<thead>
<tr>
<th>Patient</th>
<th>Operation type</th>
<th>Age at operation (mo)</th>
<th>Time from operation (mo)</th>
<th>RVEF (%)</th>
<th>LVEF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>Senning</td>
<td>6</td>
<td>24</td>
<td>50</td>
<td>71</td>
</tr>
<tr>
<td>23</td>
<td>Senning</td>
<td>8</td>
<td>43</td>
<td>60</td>
<td>59</td>
</tr>
<tr>
<td>24</td>
<td>Mustard + VSD</td>
<td>34</td>
<td>145</td>
<td>47</td>
<td>66</td>
</tr>
<tr>
<td>25</td>
<td>Senning</td>
<td>4</td>
<td>36</td>
<td>49</td>
<td>61</td>
</tr>
<tr>
<td>26</td>
<td>Senning</td>
<td>5</td>
<td>45</td>
<td>41</td>
<td>58</td>
</tr>
<tr>
<td>27</td>
<td>Senning</td>
<td>6</td>
<td>13</td>
<td>62</td>
<td>54</td>
</tr>
<tr>
<td>28</td>
<td>Senning</td>
<td>11</td>
<td>22</td>
<td>43</td>
<td>53</td>
</tr>
<tr>
<td>29</td>
<td>Mustard</td>
<td>116</td>
<td>60</td>
<td>57</td>
<td>63</td>
</tr>
<tr>
<td>30</td>
<td>Mustard</td>
<td>12</td>
<td>58</td>
<td>27</td>
<td>46</td>
</tr>
<tr>
<td>31</td>
<td>Mustard</td>
<td>1</td>
<td>91</td>
<td>65</td>
<td>54</td>
</tr>
<tr>
<td>32</td>
<td>Senning</td>
<td>10</td>
<td>3</td>
<td>45</td>
<td>74</td>
</tr>
<tr>
<td>33</td>
<td>Mustard</td>
<td>5</td>
<td>24</td>
<td>46</td>
<td>57</td>
</tr>
<tr>
<td>34</td>
<td>Mustard</td>
<td>9</td>
<td>72</td>
<td>54</td>
<td>46</td>
</tr>
<tr>
<td>35</td>
<td>Mustard</td>
<td>4</td>
<td>15</td>
<td>52</td>
<td>71</td>
</tr>
<tr>
<td>36</td>
<td>Mustard</td>
<td>1</td>
<td>85</td>
<td>52</td>
<td>68</td>
</tr>
<tr>
<td>37</td>
<td>Mustard</td>
<td>11</td>
<td>187</td>
<td>30</td>
<td>35</td>
</tr>
<tr>
<td>38</td>
<td>Mustard</td>
<td>76</td>
<td>168</td>
<td>51</td>
<td>50</td>
</tr>
<tr>
<td>39</td>
<td>Senning</td>
<td>8</td>
<td>24</td>
<td>68</td>
<td>68</td>
</tr>
<tr>
<td>40</td>
<td>Senning</td>
<td>11</td>
<td>16</td>
<td>60</td>
<td>49</td>
</tr>
<tr>
<td>41</td>
<td>Mustard</td>
<td>5</td>
<td>90</td>
<td>51</td>
<td>70</td>
</tr>
<tr>
<td>42</td>
<td>Senning</td>
<td>5</td>
<td>50</td>
<td>63</td>
<td>39</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>17</td>
<td>67</td>
<td>51</td>
<td>58</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td>11</td>
<td>11</td>
</tr>
</tbody>
</table>

RVEF, right ventricular ejection fraction; LVEF, left ventricular ejection fraction; two stage, two stage anatomical correction; +VSD, operative repair combined with closure of a ventricular septal defect.

**Figure 1.** Bar graph of frequency distribution of right ventricular ejection fraction. Open columns indicate right ventricular ejection fraction after intra-atrial repair, and hashed columns indicate right ventricular ejection fraction after anatomical correction.

**Figure 2.** Bar graph of frequency distribution of left ventricular ejection fraction. Open columns indicate left ventricular ejection fraction after intra-atrial repair, and hashed columns indicate left ventricular ejection fraction after anatomical correction.
ventricular ejection fraction for the patients who underwent intra-atrial repair (Figure 3). Similarly, no significant correlation ($r=-0.4$) between the age at anatomical correction and left ventricular ejection fraction was demonstrated.

**Influence of Duration of Postoperative Follow-up on Systemic Ventricular Function**

There was no significant correlation between the duration of postoperative follow-up and either right ventricular ejection fraction after intra-atrial repair ($r=-0.30$) or left ventricular ejection fraction after anatomical correction ($r=-0.02$) (Figures 4 and 5, respectively).

**Pulmonary Ventricular Function After Intra-Atrial Repair and Anatomical Correction**

The pulmonary ventricular function (left ventricular ejection fraction) in the patients who had intra-atrial repair ranged from 35% to 74% (mean±SD, 58±11%). Eight (38%) patients had left ventricular ejection fraction below our normal value of 55%.

The pulmonary ventricular function (right ventricular ejection fraction) in the patients who had ana-
tomical correction ranged from 35% to 78% (mean±SD, 58±12%). Six (32%) had a right ventricu-
lar ejection fraction below our normal value of 50%.

Thus, the mean left ventricular ejection fraction
after intra-atrial repair and mean right ventricular
ejection fraction after anatomical correction were
significantly lower than the mean values for the
respective values in normal patients (p<0.05 for
each).

Influence of Preoperative and Operative Variables on
Right Ventricular Ejection Fraction and Left
Ventricular Ejection Fraction

Variables including preoperative hemoglobin level,
duration of cardiopulmonary bypass, and ischemic
time were analyzed to assess their influence on right
and left ventricular ejection fractions after intra-
atrial repair and anatomical correction. No signifi-
cant correlation was found for any of these variables.

Regional Wall Motion After Anatomical Correction

No regional wall motion abnormalities were found
in any patient after anatomical correction. However,
four (19%) had significant phase delay of right
ventricular contraction; in three of these, it was
associated with known right ventricular hypertension
secondary to supravalvar pulmonary stenosis.

Discussion

There is much debate about the optimum surgical
treatment for TGA, and intra-atrial repair and anato-
mical correction have been advocated. Intra-atrial
repair may be associated with complications such as
systemic and pulmonary venous pathway obstruction,
left ventricular outflow tract obstruction, residual
systemic-to-pulmonary shunts, tricuspid regurgita-
tion, arrhythmias, and systemic ventricular dysfunc-
tion. Anatomical correction may be complicated by
supravalvar pulmonary stenosis, aortic regur-
gitation, and, potentially, coronary ischemia. Most
of these complications are amenable to treatment;
however, severe systemic ventricular dysfunction
cannot be corrected without resorting to cardiac trans-
plantation or, occasionally, a two-stage anatomical
correction with take down of the intra-atrial repair.

Systemic Ventricular Dysfunction

This study demonstrates that a significant number
of patients have subnormal ventricular ejection fraction
after intra-atrial repair and anatomical correction.

Of those patients with subnormal systemic ventricu-
lar ejection fraction after intra-atrial repair, two
(25%) had right ventricular ejection fractions of 30% or less, and both required treatment for
chronic congestive cardiac failure. The remaining
patients are asymptomatic. The incidence of symptom-
atic ventricular dysfunction of 10% is similar to that
reported in other series. The long-term outcome for
these patients remains uncertain. Our study did not
demonstrate any significant relation between the
duration of postoperative follow-up and subsequent
ventricular function. Whether systemic ventricular
function is maintained in the long-term or deterior-
ates with time is unknown. Longitudinal studies will
be required to resolve these questions.

Symptomatic systemic ventricular dysfunction was
not seen after anatomical correction during a 78-
month follow-up period (p=0.24 compared with that
in the intra-atrial repair group). However, asympto-
matic subnormal systemic ventricular ejection frac-
tion was present in 29%. One patient with a left

Figure 5. Plot of relation between time elapsed from anatomical
correction and left ventricular ejection fraction (LVEF).
ventricular ejection fraction of 39% had developed a dilated and poorly functioning left ventricle after pulmonary artery banding as part of a two-stage corrective operation. In the others, the cause of the dysfunction was unknown.

Although we have identified patients in both groups with systemic ventricular dysfunction, it is important to emphasize that ventricular function was preserved in most patients after intra-atrial repair and anatomical correction. After intra-atrial repair, mean systemic ejection fraction was significantly lower than that after anatomical correction. This must be interpreted with caution because the normal range for ejection fraction is different for the two groups and the incidence of subnormal ventricular function in each group was similar. It may be more appropriate to compare right ventricle with right ventricle and similarly for the left. Although the right ventricular ejection fraction in the intra-atrial repair group was lower than that in the anatomical correction group, the difference was not significant. According to this comparison, the loading conditions for the two groups are probably different.

Previous studies of left ventricular function after anatomical correction have not shown a significant incidence of dysfunction. This study has a longer duration of follow-up than previous studies. This longer duration alone is unlikely to account for the incidence of dysfunction because there was no significant relation between duration of follow-up and left ventricular ejection fraction. An alternative explanation may be that the normal ranges quoted are not appropriate for these patients. The left ventricle in transposition has been shown to have certain morphological differences from the normal left ventricle. The interventricular septum is flatter, and the ratio of outlet-to-inlet length is increased. Whether this affects left ventricular ejection fraction is not known.

There is concern that myocardial infarction or ischemia, related to coronary artery transfer, may influence ventricular function. There was no evidence of regional wall motion abnormality, and postoperative aortic root angiograms did not show coronary occlusion or stenosis in these patients. Thus, these conditions appear to be an unlikely cause of the left ventricular dysfunction in these patients.

Right Ventricular Function After Anatomical Correction

After anatomical correction, most patients had normal right ventricular ejection fraction; however, abnormal ejection fraction of varying degree was found in six (32%) patients. One of these with the lowest right ventricular ejection fraction had a dilated right ventricle with an ejection fraction of 35%, possibly as a result of increased loading conditions. This patient was shown to have severe supravalvar pulmonary stenosis with suprasystemic right ventricular pressure at cardiac catheterization before this study. Yet, some patients with significant supravalvar pulmonary stenosis continued to maintain a normal right ventricular ejection fraction. We cannot be sure of the contribution of the altered load to the right ventricle dysfunction because increased load did not consistently produce abnormalities of right ventricular ejection fraction. It is debatable whether the right ventricle in TGA responds differently to an increased or systemic load compared with the response in patients with isolated pulmonary stenosis.

Left Ventricular Function After Intra-Atrial Repair

After intra-atrial repair, most patients had a left ventricular function that was generally well preserved. Thirty-eight percent of these patients had subnormal left ventricular ejection fractions. Only one had undergone hemodynamic assessment, and therefore, pulmonary vascular resistance and left ventricular pressure are not known in most. In some of these patients, the abnormal left ventricular ejection fraction was also associated with right ventricular dysfunction, suggesting that a generalized insult to ventricular function such as preoperative hypoxia or perioperative ischemia occurred. Residual subpulmonary stenosis or pulmonary hypertension may contribute to the dysfunction in some patients.

Influence of Preoperative and Operative Variables on Ventricular Function

This study did not identify any significant influences on ventricular function by various factors such as preoperative hemoglobin level, duration of ischemia, cardiopulmonary bypass time, age at operation, duration of postoperative follow-up, and type of operation on subsequent ventricular function. Because of the wide range of normal values for right and left ventricular ejection fractions, only major influences would likely be revealed by a study as small as this one. A larger study would be required to show less major, but clinically important, influences on ventricular function in this heterogeneous group of patients.

Conclusions

This study demonstrates that right and left ventricular function is generally well preserved after anatomical correction and intra-atrial repair for TGA. Ventricular dysfunction occurs after both types of operations. Its etiology is undetermined and merits further evaluation by longitudinal study.

References


KEYWORDS angiography, radionuclide, transposition of the great arteries, systemic ventricular function
An evaluation of right and left ventricular function after anatomical correction and intra-atrial repair operations for complete transposition of the great arteries.

Circulation. 1990;82:808-816
doi: 10.1161/01.CIR.82.3.808

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 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/82/3/808

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/