Quantification of Left Heart Volume and Systolic Output in Transposition of the Great Arteries

By THOMAS P. GRAHAM, Jr., M.D., JAY M. JARMAKANI, M.D., RAMON V. CANENT, Jr., M.D., and PAUL H. JEWETT, M.D.

SUMMARY

Left heart volume and output were calculated by using cineangiocardiograms from 64 studies in 44 patients with transposition of the great arteries (TGA). The majority of patients who had an intact ventricular septum and were less than 6 months of age showed normal end-diastolic volumes (LVEDV) and systolic output (LVSO), while patients in this hemodynamic group more than 6 months of age had elevated volumes and outputs. The presence of a patent ductus arteriosus (PDA) was associated with an increase in LVEDV and LVSO. Patients with a ventricular septal defect (VSD) and no pulmonary stenosis (PS) had increased volumes and outputs with the average values for LVEDV and LVSO significantly greater for the VSD group than for the intact-septum group. Patients with a VSD and PS showed normal values for LVEDV and LVSO. The ejection fraction was normal in all patients. Left atrial maximal volume (LAMax) was normal in the majority of patients with an intact ventricular septum and no PDA, but was increased in patients with a VSD and no PS. The values for LVEDV, LVSO, and LAMax showed little or no change following balloon atrial septostomy in the majority of patients. Four patients studied before and after corrective surgery demonstrated decreases in LVEDV and LVSO to normal values following successful interatrial venous transposition. Left heart volume variables derived from cineangiocardiograms can aid considerably in hemodynamic evaluation of patients and in estimation of pulmonary blood flow.

Additional Indexing Words:
Atrial septectomy
Mustard's procedure
Ventricular septal defect
Atrial septal defect
Pulmonary blood flow
Balloon atrial septostomy
Pulmonary vascular disease

The recent success with corrective surgery for transposition of the great arteries (TGA)1-11 has emphasized the importance of detailed preoperative evaluation of all potential surgical candidates. At present, there is no information on left heart volumes and only limited information concerning estimated pulmonary blood flow and pulmonary vascular resistance12-16 in this group of patients. The parallel circuit relationship of the pulmonary and systemic vascular beds in TGA makes the estimation of pulmonary blood flow by Fick or indicator-dilution method difficult to perform and interpret. Several investigations have pointed out the propensity for the early development of histologic changes of pulmonary vascular obstructive disease in infants and young children with TGA,17-19 and thus the need for the reliable estimation of pulmonary blood.

From the Department of Pediatrics, Division of Pediatric Cardiology, Duke University Medical Center, Durham, North Carolina.
Supported in part by Program Project Grant National Institutes of Health HE-11307 and HE-10179.
Address for reprints: Dr. Thomas P. Graham, Jr., Department of Pediatrics, Division of Pediatric Cardiology, Vanderbilt Medical Center, Nashville, Tennessee 37203.
Received April 2, 1971; revision accepted for publication July 23, 1971.

Circulation, Volume XLIV, November 1971
flow and pulmonary vascular resistance is re-emphasized.

Quantification of left ventricular volume and systolic output can be performed with cineangiography, and normal standards for these variables in infants and children have been obtained.20, 21 The cineangiographic measurement of left ventricular systolic output in TGA with intact ventricular septum can be used to estimate pulmonary blood flow.

The purpose of this investigation was to quantify left ventricular volume, ejection fraction, systolic output, and left atrial maximal volume in patients with transposition of the great arteries and to correlate these variables with age, hemodynamic status, and associated cardiovascular defects. In addition, data were obtained on a number of patients before and after balloon atrial septostomy and before and after corrective surgery to assess the effect of these interventions on the left heart volume variables studied.

**Methods**

**Patient Population**

Data were obtained from 64 separate cardiac catheterizations performed on 44 patients with TGA ranging in age from 1 day to 10 years. All patients had dextro-transposition without ventricular inversion. Patients with a common ventricle, mitral or tricuspid atresia, or mitral insufficiency were excluded. The patients were divided into four hemodynamic groups which will be presented and discussed separately. Vital statistics and hemodynamic data for all groups are presented in table 1.

**Table 1**

*Vital Statistics and Hemodynamic Data (Mean ± SD)*

<table>
<thead>
<tr>
<th></th>
<th>Group I. ASD, intact vent septum (n = 31)</th>
<th>Group II. VSD, no PS (n = 17)</th>
<th>Group IIIA. VSD, PA band (n = 5)</th>
<th>Group IIIB. VSD, PS (n = 7)</th>
<th>Group IV. Postcorrection (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>0.75 ± 1.40</td>
<td>0.81 ± 2.40</td>
<td>2.85 ± 1.67</td>
<td>3.87 ± 4.72</td>
<td>3.50 ± 0.50</td>
</tr>
<tr>
<td>Hgb (g%)</td>
<td>17.2 ± 4.0</td>
<td>15.9 ± 2.7</td>
<td>19.6 ± 1.7</td>
<td>19.3 ± 1.9</td>
<td>12.5 ± 0.9</td>
</tr>
<tr>
<td>Ao O2 sat (%)</td>
<td>68 ± 14</td>
<td>72 ± 7</td>
<td>64 ± 12</td>
<td>75 ± 8</td>
<td>92.5 ± 3.3</td>
</tr>
<tr>
<td>LVP (mm Hg)</td>
<td>52 ± 20</td>
<td>73 ± 23</td>
<td>102 ± 13</td>
<td>92 ± 26</td>
<td>41 ± 14</td>
</tr>
<tr>
<td>RVP (mm Hg)</td>
<td>82 ± 16</td>
<td>83 ± 14</td>
<td>85 ± 12</td>
<td>95 ± 22</td>
<td>112 ± 18</td>
</tr>
</tbody>
</table>

Abbreviations: Ao O2 sat = aortic oxygen saturation; LVP = left ventricular peak pressure; RVP = right ventricular peak pressure; n = number of observations.

**Group I. Atrial Septal Defect (Intact Ventricular Septum)**

All patients included in this group had an atrial communication which was enlarged by balloon septostomy or by surgical septectomy by the Blalock-Hanlon technique. The peak left ventricular pressure (LVP) averaged 63% of right ventricular pressure (RVP) in this group. Ventricular septal defects were excluded by cineangiography or by postmortem examination. There were 31 observations available from 21 patients in this group ranging in age from 1 day to 7 years. This number includes two patients with pulmonary stenosis, one of whom was studied twice. The LVP was 75% and 85% of RVP in these two patients with a pressure difference from LV to pulmonary artery of 61 and 44 mm Hg respectively. The stenosis was subvalvular in one patient and combined valvular and subvalvular in the remaining patient.

**Group II. Ventricular Septal Defect (No Pulmonary Stenosis)**

In this group 17 observations were made on 14 patients ranging in age from 2 days to 10 years. Left ventricular peak pressure averaged 88% of right ventricular pressure, and the presence of a ventricular defect was confirmed by cineangiography. Pulmonary stenosis was excluded by measurement of pulmonary artery pressure or by postmortem examination. All but one patient had increased pulmonary flow as interpreted from routine chest X-rays. All patients had an atrial defect demonstrated at cardiac catheterization. Only one patient had undergone surgical atrial septectomy at the time of study. Two patients in this group had hypoplastic right ventricles22 as estimated by cineangiography and postmortem examination.

**Group IIIB. Ventricular Septal Defect with Pulmonary Artery Banding**

In this group there were five studies on four patients with pulmonary artery bands. In one
patient the ventricular septal defect had closed spontaneously before the time of study. Patients were studied an average of 2.4 years following banding with a range of 6 months to 4.5 years. All but one patient also had Blalock-Hanlon atrial septectomy performed at the time of pulmonary artery banding. Age at time of study ranged from 9 months to 5 years. Left ventricular pressure (LVP) was equal to right ventricular pressure (RVP) in this group except in the patient with spontaneous VSD closure following banding in whom LVP was 167/18 and RVP was 100/17 mm Hg. Pulmonary artery pressure was not obtained in this patient. In the remaining patients, the LV to pulmonary artery pressure difference averaged 64 mm Hg with a range of 58 to 75 mm Hg.

**Group IIIIB. Ventricular Septal Defect with Pulmonary Stenosis**

There were seven observations from five patients in this group ranging in age from 2 months to 10 years. Left ventricular pressure was equal to right ventricular pressure in all patients in this group. The pressure difference from LV to PA averaged 63 mm Hg with a range of 44 to 81 mm Hg.

**Group IV. Complete Correction**

Four patients were studied an average of 1 year following successful corrective surgery by the Mustard procedure. Three patients had only atrial defects (Blalock-Hanlon septectomy) preoperatively. The fourth patient had had a pulmonary band removed, but did not require ventriculotomy because the VSD had closed spontaneously. All patients were between 2 and 3 years of age at the time of correction. Left ventricular pressure ranged from 30 to 64 mm Hg and averaged 41 mm Hg or 36% of RVP. The patient with a PA-band removal had LVP or 38/9 compared with the preoperative value of 167/18 mm Hg.

**Procedures**

All patients were studied during routine diagnostic cardiac catheterization. Thirty-four patients were less than 3 months of age and local anesthesia only was used. Patients over 3 months of age were studied with sedation with meperidine 1-1.5 mg/kg and/or promethazine 0.5-1.0 mg/kg or light general anesthesia with nitrous oxide and small concentrations of halothane (≤0.5%). Left and right heart pressures were recorded with NIH catheters (no. 5, 6, or 7) before the first cineangiogram. Zero pressure was referenced to midchest. All data were obtained with the arterial pH greater than 7.32.

Left heart volume variables were calculated from biplane cineangiograms (AP and lateral, 60 frames/sec) after injection of 1 to 1.25 ml/kg of sodium and meglumine diatrizoates (Hypaque-M) into the left atrium, left ventricle, or pulmonary artery. The electrocardiogram and cinegraphic exposure were recorded during cineangiography. All volumes were corrected for X-ray magnification. The details of this system have been described previously.

Left ventricular end-diastolic volume (LVEDV), end-systolic volume (LVESV), and left atrial maximal volume (LAMax) were calculated by the area-length method. In patients with volume measurements before and after balloon atrial septostomy, the preseptostomy value was used for statistical comparisons between groups. Left ventricular volumes were corrected by using previously derived regression equations. For calculated LV volumes ≤15 cm³, V = 0.733V (V = regressed volume in cm², V = calculated volume). For calculated volumes >15 cm³, V = 0.947V − 3.1 cm³.

All volume and mass variables were divided by body surface area (BSA) to normalize data for patients of different size. Surface area was obtained by the graphical method of Sendroy and Cecchini, and all volume data were compared with previously derived normal values. In addition, all patients’ data were evaluated in terms of percent of predicted normal values. Predicted normal values were calculated from previously derived regression equations relating volume variables to weight, height, and age in 56 patients with normal left hearts.21 Student’s test was used for statistical comparisons.

**Results**

**Left Ventricular End-Diastolic Volume (LVEDV)**

**Group I. Isolated Atrial Defect**

A plot of LVEDV (percent of normal) versus age is shown in figure 1 for this group. Thirteen of 19 studies (69%) made during the first 6 months of life on patients who did not have a patent ductus showed LV volumes which are normal or less than normal. In contrast, eight of nine studies (89%) on patients above 6 months of age showed volumes which were elevated with an average value of 181% of normal.

Two infants who had volumes greater than twice normal before 6 weeks of age had large aortic to pulmonary shunts through a patent ductus arteriosus as estimated by cineangiography.

The patient with the second largest volume (413% of normal) was studied first at 6 months
LVEDV (from 70% at 1 day to 27% at 9 months) increased his LV pressure from 20/5 on day 1 to 110/9 mm Hg at 9 months without the development of pulmonary stenosis.

Group II. Ventricular Septal Defect without Pulmonary Stenosis

In all patients of this group, LVEDV was above normal (fig. 2). The average LVEDV was 259% of normal and was significantly greater than that for the isolated ASD group (group I) (P<0.01). Two patients with hypoplastic right ventricles had end-diastolic volumes which were over twice normal. One patient with severe pulmonary vascular obstructive disease and a reversed shunting patent ductus had an LVEDV only slightly above normal.

Group IIIA. Ventricular Septal Defect and Pulmonary Artery Band

The four patients with PA bands had LVEDV averaging 159% of normal. One patient studied both before and 3 years after pulmonary artery banding showed a large decrease in LVEDV following this procedure. The three other patients with PA bands were studied 6, 18, and 20 months following the procedure. They had elevated volumes (fig. 2).

Two patients showed a decrease in LVEDV with increasing age over the course of three studies in the first 6 months of life. In these patients, the peak LV pressures were 33 and 38 mm Hg, and PA pressure was not measured.

The patient with the largest change in because of severe cyanosis. His LV pressure was 63/6 (68% of systemic) with no significant LV to PA pressure difference and no patent ductus. He had an excellent clinical response to balloon atrial septostomy with an immediate increase in systemic oxygen saturation from 58 to 74%.

Two patients in this group with intact ventricular septums had pulmonary stenosis with gradients of 61 and 41 mm Hg from LV to pulmonary artery. One of these patients who was studied first at 3 days of age had a patent ductus present at that time with a moderate aortic to pulmonary shunt as judged from cineangiocardiography. LVEDV at this time was 157% of normal. He was studied at age 22 months and was found to have an LVEDV of 99% of normal and no patent ductus. The other patient with pulmonary stenosis was studied at age 6 months and had the smallest LV volume (52% of normal) of all the patients studied.

Two patients showed a decrease in LVEDV with increasing age over the course of three studies in the first 6 months of life. In these patients, the peak LV pressures were 33 and 38 mm Hg, and PA pressure was not measured.

The patient with the largest change in...
there is no patent ductus. Seventeen of the 19 studies (90%) on patients in the first 6 months of life with intact ventricular septum who did not have a patent ductus, showed LVOS which were normal or diminished. In seven of nine studies on such patients who were above 6 months of age outputs were elevated averaging 164% of normal.

The LVSO was increased in all patients with VSDs without pulmonary stenosis except one, a 10-year-old with pulmonary vascular obstructive disease. The average value for the group was 192% of normal (P < 0.001) and was significantly increased above the value for patients with an intact ventricular septum (P < 0.01).

In patients with VSDs and pulmonary banding or pulmonary stenosis the LVSO again shifted in the same direction as the LVEDV. Patients with anatomic stenosis had LV systolic outputs averaging 101% of normal, while patients with PA band had LV outputs averaging 150% of normal.

**Left Atrial Maximal Volume (LAMax)**

This variable was calculated 27 times for 24 patients. Six of nine patients with an intact ventricular septum and no patent ductus had normal values for LAMax (fig. 5). All but one of these patients was less than 6 months of age. Two patients with atrial defects and PDA had elevated left atrial volumes. Ten of 12

Circulation, Volume XLIV, November 1971
patients with ventricular defects had elevated left atrial volume and the volume for this group was significantly greater than that for the group with intact ventricular septums \((P < 0.001)\). One patient with a VSD and PA band had elevated LAMax 6 months following surgery, and one patient had normal LAMax when studied 3.5 and 4.5 years after surgery.

**Effect of Balloon Atrial Septostomy**

Left heart volumes before and after balloon atrial septostomy\(^{24}\) (BAS) were available in six patients and are shown in figure 6. Only patients whose heart rates after BAS were within 10 beats/min of the rate before BAS were included in this analysis. The average time lapse between preseptostomy and post-septostomy cineangiograms was 46 minutes. The average \(O_2\) saturation of aortic blood was 49% prior to BAS and 65% after BAS. Left atrial mean pressure pre-BAS was 7.6 mm Hg and averaged 4.2 mm Hg following BAS. The mean pressure difference between left atrium and right atrium was 3.4 mm Hg pre-BAS and zero after BAS. Three of the six patients showed a decrease in LVEDV and LVSO, one patient showed an increase, and two patients showed no change in these two variables (fig. 6). One of five patients with LAMax determinations showed a decrease following septostomy while the remainder showed no change. The patient with the largest increase in oxygen saturation (45 to 77\%) had the largest decrease in atrial volume from 7.5 to 4.3 cm\(^3\) with a concomitant decrease in mean left atrial pressure from 11 to 7 mm Hg.

**Effect of Corrective Surgery**

Preoperative and postoperative data were available on four patients who have had corrective surgery (fig. 7). Postoperatively there were no residual shunts. Three patients had only atrial defects and one patient (closed circle) had a pulmonary artery band, with an intact ventricular septum as described above. Three of the four patients showed a decrease in LVEDV and LVSO to normal levels postoperatively. The patient who had her PA band removed at corrective surgery had a slight increase in LVSO postoperatively, but because of her growth spurt the value fell well within the normal range.

**Discussion**

Although cardiac enlargement on radiographic examination has been considered the usual pattern in complete transposition of the great arteries, there have been no previous reports of in vivo estimation of the size of left heart chambers or of differentiation of right
versus left heart alterations from normal. The present study was undertaken to study cinecardiographically estimated left heart volume and output in these patients. The ventricular output in patients with transposition and an intact ventricular septum provides an estimate of pulmonary blood flow if bronchial flow is negligible and there is no patent ductus.

**Intact Ventricular Septum**

The majority of studies on infants (69%) less than 6 months of age with TGA and intact ventricular septum without patent ductus had left ventricular end-diastolic volumes and outputs which were normal or diminished. In contrast, eight of nine patients above 6 months of age in this group had clearly elevated end-diastolic volumes, and seven of nine patients had elevated systolic outputs. Thus, the data suggest that left heart volumes and pulmonary blood flow are usually normal early in the first year of life but become elevated above normal with increasing age.

The reason for this finding is not clear but may be accounted for partially by a progressive decrease in pulmonary vascular resistance. Alterations in total blood volume and the dynamics of atrial shunting also may play roles in this increase in volume and output during the first year.

The patients whose values deviated from these general trends provide intriguing data for consideration. Three infants had elevated LV volumes and outputs in the first 6 weeks of life associated with a patent ductus. In addition, one infant with a patent ductus, ventricular defect, and pulmonary stenosis had a clearly elevated LVEDV and LV systolic output prior to surgery and showed a progressive decline in these variables after ductal ligation. These data suggest that systemic to pulmonary ductal flow, although pumped by the right ventricle, can importantly influence left heart volume and output.

Two infants with intact ventricular septum demonstrated progressive declines in left heart volume and output over the first 6 months of life. The possibility was considered that these infants might have developed subvalvular pulmonary stenosis over this period. The peak left ventricular pressure, however, was only 33 and 38 mm Hg or 34% and 54% of systemic pressure respectively. Because of the low left ventricular pressure, pulmonary artery pressure was not measured. Both patients had large atrial defects at the time of their last study with no mean pressure difference between left and right atrium. One possible explanation for the progressive decline in volumes in these patients is the development of a mild to moderate degree of pulmonary stenosis together with some degree of diminished left ventricular distensibility. Tynan and co-workers\textsuperscript{15} have demonstrated that a poor clinical result following adequate balloon atrial septostomy in patients with an intact ventricular septum can be due to the development of significant subvalvular pulmonary stenosis. In this regard, one of the patients studied by Tynan and associates\textsuperscript{15} had a left ventricular pressure of only 35 mm Hg and a pulmonary artery pressure of 12 mm Hg.
These findings indicate the usefulness of pulmonary artery pressure measurements even when the left ventricular pressure is only mildly elevated.

Two patients in this group had intact ventricular septum and definite pulmonary stenosis. One of these patients studied at age 6 months had LV pressure of 64/9 mm Hg, RV pressure of 75/10 mm Hg, and pulmonary artery pressure of 20/10. There was no mean pressure difference between left and right atrium. Her LV volume and output were the smallest of any patient's in the study, and she did not survive an aortic to pulmonary shunt procedure. At postmortem examination the left ventricle was small with normal wall thickness. This unusually extreme degree of LV hypoplasia (approximately 50% of normal) may provide an added risk factor for either a palliative shunt procedure or corrective surgery by the Mustard technique.1

J. R. was the other patient to have pulmonary stenosis with intact ventricular septum. His volume and output determined initially at age 3 days were elevated at which time a patent ductus was present. At age 22 months, the ductus had closed and both LVEDV and output were normal. This patient did not survive corrective surgery despite what was felt at the operating table and at postmortem examination to be adequate relief of pulmonary stenosis.

Another patient in the intact ventricular septum group who is of special interest was studied for the first time at age 6 months. He was extremely cyanotic at this time with a hemoglobin value of 20.5 g/100 ml and a systemic oxygen saturation of 58%. Cineangiocardiography showed a small atrial defect with an LVEDV over four times normal and an LV output of 2½ times normal. He had an excellent clinical result from balloon atrial septostomy with an increase in systemic oxygen saturation from 58% to 74%. This patient's data suggest that an inadequate atrial communication can be associated with large increases in left heart volume and output which are inadequate compensatory mecha-

isms to increase oxygenation in the presence of poor mixing.

The patient with the largest documented increase in volume and output following an initial study in the neonatal period also demonstrated a large increase in left ventricular pressure. This patient's LV pressure increased from 20/5 mm Hg (31% of systemic pressure) at age 1 day to 110/9 mm Hg (157% of systemic) at 9 months in the absence of pulmonary stenosis. Although there was no mean pressure difference between left and right atrium, a Blalock-Hanlon septectomy was attempted, but the patient expired. Unfortunately, postmortem examination was not permitted, and thus the presence or absence of pulmonary vascular obstructive disease cannot be determined with certainty. The dramatic increase in LV pressure in the presence of an intact ventricular septum and without the development of pulmonary stenosis is most compatible with a significant increase in pulmonary vascular resistance. This patient's course probably should not be considered in terms of a severe Eisenmenger's reaction because the pulmonary pressure was increased and the left ventricular volume was large. The end-stage patient with an Eisenmenger's reaction would be expected to have normal or decreased pulmonary flow and normal or decreased left ventricular end-diastolic volume. The time course of decrease in an initially elevated LV end-diastolic volume as pulmonary flow diminishes in patients with TGA is not known. In patients without transposition who were studied an average of 2 years following closure of a ventricular septal defect, the LV end-diastolic volume remained mildly but significantly elevated.25 Thus decrease in LVEDV following a decrease in pulmonary flow can have a protracted time course which may be dependent on age, initial degree of elevation of these variables, and growth following the decrease in flow.

Ventricular Septal Defects

In all but two patients with TGA, ventricular septal defects, and no pulmonary stenosis,
left heart volumes and output were increased considerably above normal. This elevation of volume and output was apparent as early as these patients have been studied including seven patients studied during the first month of life. One of the two patients with relatively normal volumes and output was 10 years old and had pulmonary vascular obstructive disease. This patient's volume data are those expected for a patient with end-stage Eisenmenger's reaction. The other patient with normal LVEDV and output had undergone pulmonary artery banding 3.5 years before the study. These findings suggest a good correlation between pulmonary blood flow and left heart volume in patients with TGA and ventricular defects even though the left ventricular systolic output does not equal pulmonary flow in this situation.

Three patients with pulmonary artery banding had persistence of elevated volume and output. This elevation of LV volume in the presence of pulmonary banding in these patients may be as a reflection of the relatively short interval since banding when the patients were studied (6, 18, and 20 months) as well as the degree of constriction produced by the band.

The small number of patients with ventricular defects and pulmonary stenosis had relatively normal volumes and output with an exception of one infant with a patent ductus who showed a progressive decrease in volume following ductal ligation. In two patients in this group correction was attempted with the Mustard technique, but neither survived the early postoperative period. The ventricular sizes and ejection fractions in these two patients did not suggest a significant myocardial factor contributing to the surgical results.

**Left Atrial Volume**

Left atrial volume was normal in the majority of patients less than 6 months of age who had intact ventricular septum and no patent ductus. In contrast, the left atrial volume was elevated in the majority of patients with TGA and VSD. An excellent correlation between LAMax/BSA and LVSO or pulmonary blood flow has been demonstrated previously in patients with an isolated ventricular septal defect or patent ductus. The size of the atrial defect and the degree of bidirectional shunting at the atrial level in TGA may play a role in determining left atrial size in these patients. Thus, an uncomplicated correlation of estimated pulmonary flow and LAMax is undoubtedly an oversimplification, for multiple factors may well be involved in determining left atrial size in patients with TGA.

**Balloon Atrial Septostomy**

The study of left heart volume variables following balloon atrial septostomy (BAS) demonstrated little or no change with this intervention. The time between cineangiograms, as well as any hemodynamic effects of the first one, may have served to obscure any changes in volumes which occurred immediately following BAS. Significant changes in left atrial size may well follow BAS if there is preexisting left atrial hypertension which is relieved. This was shown dramatically in one patient studied (fig. 6). Rashkind and Miller have shown large changes after BAS in the mean atrial pressure difference in five of six patients who had pre-septostomy differences of 5 mm Hg or greater.

**Postcorrection**

In four patients studied following corrective surgery by the Mustard procedure, left heart volume and systolic output were well within the normal range. The decrease in these variables indicates a decrease in pulmonary flow after complete correction. Two additional patients, also studied following a Mustard procedure, had large residual ventricular septal defects and elevated volumes and output. Waldhausen and co-workers have demonstrated a decrease in both systemic and pulmonary flows following corrective surgery with preoperative flows determined by the Fick method and postoperative values using indicator-dilution techniques.

The extreme variability of the clinical course in patients with TGA and the difficulty in predicting hemodynamic changes from
Clinical findings has been reemphasized recently by Plauth and co-workers.16 In addition, the development of pulmonary vascular disease early in life has been emphasized for patients with ventricular septal defects as well as for a number of patients with an intact ventricular septum.16–19 It is hoped that the addition of volume measurements to the routine catheterization evaluation of patients with transposition will aid in the clinical and surgical management of these patients by providing direct measurements of the pumping characteristics of the left heart and by providing a unique method for estimating pulmonary blood flow.

Clinical Application

From the data obtained the following general guidelines may be used for applying left heart volume data to patients with transposition of the great arteries.

1. Infants less than 6 months of age with an intact ventricular septum and no patent ductus usually have normal or slightly elevated LV volumes and output.

2. If LV volume and output are moderately increased in infants less than 6 months (150% of normal), significant systemic to pulmonary shunting at the ventricular or aortic level is considered a likely possibility.

3. If LV volume and output are normal in children above 6 months of age, pulmonary stenosis is considered a likely possibility.

4. The combination of left heart output and pulmonary artery pressure can be used to approximate pulmonary vascular resistance with units of mm Hg/(liters/min/m²) as an aid in evaluating potential surgical candidates.

Acknowledgment

The authors would like to express their appreciation to Miss Eugenia Cole and Miss Lucy Bullock for their assistance in data collection; Mrs. Barrie Scardino, Mrs. Ida Phialus, Mrs. Costella Harris, and Mrs. Martha Mason for their invaluable work in data processing; and Mrs. Carol Lehman and Miss Wanda Taylor for their work in preparation of this manuscript.

References


2. ABERDEEN E, WATERSTON DJ, CARR I, GRAHAM C, BONHAM-CARTER RE, SUBRAMANIAN S: Successful "correction" of transposed great arteries by Mustard's operation. Lancet 1: 1233, 1965


13. SHAHER RM, KIDD L: Hemodynamics of complete transposition of the great vessels before and after the creation of an atrial septal defect. Circulation 33 (suppl 1): I-3, 1966


Circulation, Volume XLIV, November 1971


24. RASHKIND WJ, MILLER WW: Creation of an atrial defect without thoracotomy: palliative approach to complete transposition of the great arteries. JAMA 196: 991, 1966


Quantification of Left Heart Volume and Systolic Output in Transposition of the Great Arteries

THOMAS P. GRAHAM, JR., JAY M. JARMAKANI, RAMON V. CANENT, JR. and PAUL H. JEWETT

Circulation. 1971;44:899-909
doi: 10.1161/01.CIR.44.5.899

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 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/44/5/899

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/