Right Heart Volume Characteristics in Transposition of the Great Arteries

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SUMMARY
Right and left heart volume data were obtained during 44 cardiac catheterizations in 24 patients with complete transposition of the great arteries (TGA) prior to "corrective surgery." Patients were divided into three hemodynamic groups: I) TGA with intact ventricular septum, N = 23 studies in 13 patients, ages 1 day--22 months; II) TGA with ventricular septal defect (VSD), N = 12 studies in six patients, ages 7 days--15 months; III) TGA with VSD plus pulmonary stenosis, N = 9 studies in five patients, ages 5 days--5.6 years. In group I, right ventricular end-diastolic volume (RVEDV) averaged 170 ± 52% of normal (P < 0.001), RV ejection fraction (EF) was 0.48 ± 0.09, 74% of normal (P < 0.001), and RV systolic output (SO) was 123 ± 34% (NS). In group II, RVEDV was 163 ± 25% of normal (P < 0.001), RVEF 0.59 ± 0.08, 91% of normal (NS), and RVSO was 158 ± 52% (P < 0.02). In group III, RVEDV averaged 124 ± 26% of normal (P < 0.04), RVEF 0.58 ± 0.15, 89% of normal (NS), and RVSO 125 ± 57% (NS). Right atrial maximal volume was increased in 21 of 22 studies and averaged 185 ± 47% of normal (P < 0.001). RVEDV was greater than left ventricular end-diastolic volume in all but one patient in group I, but RVEDV/LVEDV was <1.00 in four VSD patients with very large pulmonary flows. A majority of patients with TGA without RVSO have abnormal right heart function as indicated by an increased RVEDV and a low ejection fraction. Longitudinal studies of right ventricular performance in these patients continue to be important in assessment of current methods of therapy.

Additional Indexing Words:
Left ventricular volume  Cineangiocardiography  Ventricular function
Right atrial volume

Although the right ventricle functions as the systemic pumping chamber from birth in patients with transposition of the great arteries (TGA), little is known about right heart function in this condition. Several recent investigations have demonstrated that right heart volumes can be calculated from cineangiograms, and normal values have been determined for infants and children. The purpose of this investigation was to quantitate right heart size and function in preoperative patients with TGA using cineangiographic volume measurements, to compare right and left heart volume data in the same patients, and to attempt to correlate these measurements with other hemodynamic variables or associated cardiovascular defects. Right ventricular (RV) volume variables in TGA patients will be compared with normal RV volumes despite the fact that the physiologically equivalent ventricle with respect to resistance to ejection in the nontransposed patient is the left ventricle (LV). Previous studies in our lab have shown no differences in normal values between right and left ventricular end-diastolic volume and left ventricular end-diastolic volume, right ventricular systolic output and left ventricular systolic output.

Methods
All data were obtained as a part of routine diagnostic catheterization performed in patients with dextro-TGA without ventricular inversion. Patients with a common ventricle, mitral or tricuspid atresia, or mitral insufficiency were excluded. There were three major hemodynamic groups.

Group I. Atrial Septal Defect (ASD) with Intact Ventricular Septum
All patients in this group had an ASD which was enlarged by balloon atrial septostomy (BAS) or by the Blalock-Hanlon technique for atrial septectomy. Ages ranged from 1 day to 22 months. A ventricular septal defect (VSD) was excluded by right ventricular (RV) and left ventricular (LV) cineangiography.
A total of 23 studies were performed in 13 patients with six patients studied twice and two patients studied three times. For all studies performed prior to two months of age, volume data were obtained prior to balloon atrial septostomy. Four patients had data obtained after atrial septectomy.
Peak right ventricular pressure (RVP) averaged 86 ± 20
mm Hg (X ± sd), LVP 46 ± 10 mm Hg, and LVP/RVP averaged 0.57 ± 0.18. Pulmonary artery pressure (PAP) was measured in 12 of 13 patients. Pulmonary stenosis (PS) was excluded at autopsy in one additional patient in whom PAP was not measured. In the 12 patients with PAP measured, LV to PA pressure differences ranged from 0 to 29 mm Hg. Differences >15 mm Hg were not considered significant. Four patients ranging in age from 5 to 21 months had differences >15 mm Hg (21, 28, 29, and 29 mm Hg) and were considered to have significant left ventricular outflow obstruction. These patients’ right heart data did not differ from the group as a whole, and thus they were not considered separately for statistical comparisons.

Systemic O2 saturation averaged 59 ± 10% and was higher in the older patients (table 1). The higher saturation in the older patients undoubtedly reflects the fact that one factor in deciding when to restudy infants with TGA is their systemic O2 saturation. Thus, patients with relatively high saturations were restudied only when a “corrective” operation was imminent, while those with lower O2 saturations were restudied at a younger age. A small patent ductus was demonstrated by right ventricular cineangiography in only four patients, but none of these patients was felt to have significant ductal shunting by clinical or catheterization evaluation.

Because of the possible effects on volume variables of age-related changes in systemic and pulmonary vascular resistance, heart rate, and body size, patients in this group were divided into subgroups according to age: IA — < one month, IB — one to six months, and IC — > six months. Average values and range of values for hemodynamic variables, systemic oxygen saturation, and hematocrit for these groups are given in table 1.

Group II. Ventricular Septal Defect (VSD)

These patients had a VSD demonstrated by cineangiocardiography. Peak LVP was equal to RVP at the initial study in all but two patients in whom LVP/RVP was 0.74 and 0.81. Systemic O2 saturation averaged 67 ± 10% (table 1). Twelve studies were performed in the six patients in this group. Pulmonary stenosis was excluded by pulmonary artery pressure measurements. Ages ranged from 7 days to 15 months. Two patients had a marked decrease in size of their VSD on subsequent studies as judged by right ventricular cineangiography and a decrease in LVP/RVP from 1.60 to 0.36 and from 0.81 to 0.60.

Group III. Ventricular Septal Defect (VSD) and Pulmonary Stenosis (PS)

Nine studies were performed in five patients in this group. Ages ranged from 5 days to 3.6 years. Pressure difference from left ventricle to pulmonary artery averaged 76 ± 17 mm Hg with a range from 50–100 mm Hg. Systemic O2 saturation averaged 68 ± 8% (table 1). The relatively high saturations in this group correlates well with the usual clinical finding that these patients have good intracardiac mixing and only moderate cyanosis unless pulmonary flow is decreased substantially. One patient had a large PDA at initial study which was ligated prior to her second catheterization.

Volume Data Acquisition

All data were obtained during routine diagnostic cardiac catheterization. Atrial and ventricular volumes were calculated from biplane cineangiography using anterior-posterior and lateral views. Ectopic beats and postectopic beats were excluded from analysis. All images were corrected for magnification and data were analyzed using a coordinate digitizer and computer processing. This methodology as well as the normal values for comparison have been described previously.2 4 6 8 Student’s t-tests for unpaired samples were used to compare variables between

Table 1

<table>
<thead>
<tr>
<th>Cardiac Catheterization Data*</th>
<th>Age</th>
<th>HR</th>
<th>RVP (mm Hg)</th>
<th>LVP/RVP</th>
<th>Systemic O2 sat %</th>
<th>PCV %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group I</strong> (TGA, IVS)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>IA [&lt; 1 mos] (N = 7)</td>
<td>4.3 days</td>
<td>155 ± 20</td>
<td>63 ± 10</td>
<td>0.69 ± 0.13</td>
<td>55 ± 7</td>
<td>50 ± 5</td>
</tr>
<tr>
<td>IB (1-6 mos) (N = 8)</td>
<td>1-15 days</td>
<td>136-194</td>
<td>50-75</td>
<td>0.45-0.80</td>
<td>47-68</td>
<td>43-56</td>
</tr>
<tr>
<td>IC (&gt; 6 mos) (N = 8)</td>
<td>1.5-5.5 mos</td>
<td>125-150</td>
<td>64-125</td>
<td>0.34-1.00</td>
<td>30-68</td>
<td>61-70</td>
</tr>
<tr>
<td>All (N = 23)</td>
<td>5.7 mos</td>
<td>134 ± 20</td>
<td>86 ± 20</td>
<td>0.57 ± 0.18</td>
<td>59 ± 10</td>
<td>56 ± 8</td>
</tr>
<tr>
<td><strong>Group II</strong> (TGA, VSD)</td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>All (N = 12)</td>
<td>7 days-15 mos</td>
<td>107-194</td>
<td>50-125</td>
<td>0.34-1.00</td>
<td>30-70</td>
<td>43-70</td>
</tr>
<tr>
<td><strong>Group III</strong> (TGA, VSD + PS)</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All (N = 9)</td>
<td>5 days-5.6 yr</td>
<td>97-170</td>
<td>72-110</td>
<td>1.00</td>
<td>62-88</td>
<td>40-70</td>
</tr>
</tbody>
</table>

*For each group top line = X ± sp and second line = range.

Abbreviations: HR = heart rate; RVP = peak right ventricular pressure; LVP = peak left ventricular pressure; O2 sat = oxygen saturation; PCV = packed red cell volume.
different groups or subgroups, and a $P < 0.05$ was considered significant. Regression analysis was used to determine if significant correlations existed between volume variables and other variables.

**Results**

**Right Ventricular End-Diastolic Volume (RVEDV)**

RVEDV was increased in 17 of 23 studies in patients with intact ventricular septum as shown in figure 1 and the average value for the entire group was $170 \pm 52\%$ of normal ($P < 0.001$). This variable was within normal limits in two neonates whose ages were two and three days at initial study. The infant studied initially at three days was restudied at 15 days because of persistent severe cyanosis. This infant showed no significant change in either RVEDV or heart rate on repeat study. He was recatheterized at 5.5 months because of a hematocrit which had risen to 66$. The heart rate at this study was also essentially unchanged and RVEDV remained normal despite mild depression of the ejection fraction. The second infant with a normal RVEDV at two days was restudied at age eight months at which time the heart rate was 111 versus 167 as a neonate. RVEDV remained normal despite a low ejection fraction.

The average value for RVEDV in terms of percentage of predicted normal is shown in figure 2 for patients with an intact ventricular septum. Here patients are grouped by age: $<1$ month ($N = 7$, $\bar{x} = 4.3$ days), 1–6 months ($N = 8$, $\bar{x} = 3.8$ months), and $>6$ months ($N = 8$, $\bar{x} = 14$ months). Although the average RVEDV increased from 149$% of normal at 4.3 days to 183$% at 3.8 months and 178$% at 14 months, none of these differences was significant. In addition, regression analysis of RVEDV ($%$ of normal) as a function of age or BSA did not show a significant relationship.

RVEDV averaged 163 $\pm 25$ of normal in patients with an isolated VSD (fig. 3). This value is significantly different from normal ($P < 0.001$), but not different from that value found in patients with TGA and an intact ventricular septum. Two patients had spontaneous closure of a VSD and serial studies showed a decrease in RVEDV from 182$% of normal at age 17 days to 132$% at age one year in one infant and from 162$% of normal at age three weeks to 135$% at age six months in the other infant. An additional patient developed pulmonary vascular obstructive disease between his study at age 2.5 months and his repeat study at 15 months. This child also showed a decrease in RVEDV from 232$% to 147$% of normal between the two studies. Two patients in this group had normal RVEDVs at initial study at ages 7 and 18.
days. On restudy at 4 and 5 months, RVEDV had increased from 121 to 211% of normal and from 112 to 131% concomitant with large increases in pulmonary blood as judged by clinical evaluation and by large increases in left ventricular volume and output.

In patients with VSD + PS, RVEDV averaged 124 ± 26% of normal (P < 0.04) and was within normal limits in three patients with this combination of lesions (fig. 4). One infant had a decrease in RVEDV from 148% to 130% of normal following ductal ligation.

Right Ventricular Ejection Fraction (RVEF)

RVEF was decreased to less than 2 sos below normal in 12 of 23 studies and was at the lower limit of normal (0.49) in three additional patients with TGA and intact ventricular septum. This variable averaged 0.48 ± 0.09 for the entire intact ventricular septum group and was significantly decreased from the normal value of 0.65 ± 0.08 (P < 0.001). Figure 5 illustrates the ejection fraction for the different intact ventricular septum age groups. Serial studies were performed in eight patients: six showed a decrease in the EF by 0.11 to 0.24 while a small increase of 0.04 and 0.06 was found in two patients. The average value for the EF decreased following the neonatal period (P < 0.04) but did not decrease further when the 1–6 month old infants were compared with those > six months.

RVEF averaged 0.59 ± 0.08 in patients with an isolated ventricular septal defect. This value was not significantly different from normal, but was significantly greater than that found for patients with TGA and an intact ventricular septum (P < 0.004). The two patients who had spontaneous closure of their
VSD and the one patient with development of pulmonary vascular obstructive disease had decreases in RVEF.

RVEF averaged $0.58 \pm 0.15$ in patients with VSD + PS. This value was not different from normal or from that found in TGA patients with an isolated VSD. It was significantly greater than that found for patients with an intact ventricular septum ($P < 0.03$).

In an attempt to learn what etiological factors might be contributing to the different RVEF values for the different groups, systemic oxygen saturations ($O_2$ sat) were compared (table 1). $O_2$ sat was less, $59 \pm 10\%$, in the intact septum group than the almost identical values of $67 \pm 10\%$ for the VSD patients and $68 \pm 8\%$ for the VSD + PS group. Regression analysis of RVEF as a function of $O_2$ sat, however, did not show a significant relationship for any of the groups.

**Right Ventricular Systolic Output (RVSO)**

RVSO, the product of RV stroke volume derived from cine and heart rate, was increased in 10 of 23 studies in patients with TGA and intact ventricular septum and averaged $123 \pm 34\%$ of normal (fig. 6). This value was not significantly greater than normal. When patients were grouped according to age, there was no difference in this variable expressed as a percentage of normal with increasing age (fig. 2).

In patients with an isolated VSD, RVSO averaged $158 \pm 52\%$ of normal ($P < 0.02$). This value was not significantly different from that found in TGA patients without a VSD. RVSO did decrease in the two patients with spontaneous VSD closure and in the one patient with development of pulmonary vascular disease.

RVSO averaged $125 \pm 57\%$ in patients with VSD + PS. This value was not different from normal or from the other TGA groups. This variable was normal or less than normal (67 to 107%) in three patients in whom decreased pulmonary flow was suspected on clinical examination and chest film and increased in two patients in whom clinical examination and chest film suggested increased pulmonary flow.

**Right Atrial Maximal Volume (RAMax)**

RAMax was obtained in 22 patients and was increased in all but one infant averaging $185 \pm 47\%$ of normal ($P < 0.001$) (fig. 7). Patients with VSD or VSD + PS did not have strikingly different values for this variable than patients with an intact ventricular septum, but the number of patients in each group were too small for statistical comparison. In four patients with serial studies, RAMax (% of normal) increased.

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*Figure 6*  
RV systolic output versus BSA in TGA patients with intact ventricular septum.

*Figure 7*  
Right atrial maximal volume (RAMax) as a function of BSA in all TGA patients.
Left Ventricular Volume Variables

Left ventricular end-diastolic volume (LVEDV) in patients with an intact ventricular septum was increased to \( \geq 125\% \) of normal in 12 of 23 studies and averaged 136 \( \pm 47\% \) of normal (\( P < 0.05 \)). In patients without PS, LVEDV was increased in only 3 of 7 studies to one month, 5 of 7 studies from 1–6 months, and 4 of 5 studies after 6 months. In contrast all four patients with PS who ranged in age from 5–21 months had normal LVEDVs.

LVEDV was increased in all patients with a VSD during at least one of their studies and averaged 219 \( \pm 94\% \) of normal, \( P < 0.01 \). This value was also greater than that found for the intact septum group, \( P < 0.01 \). Two infants were studied at ages 7 and 18 days and had normal LVEDVs. On restudy at four and five months, LVEDV had increased from 113 to 371% in one patient and from 107 to 267% in the other. One infant studied at 2.5 months had an LVEDV of 178%, but when restudied at 15 months, LVEDV was only 117% probably secondary to pulmonary vascular obstructive disease with decreased pulmonary flow.

LVEDV was increased in three of eight studies with VSD + PS and averaged 132 \( \pm 51\% \) of normal, \( P < 0.01 \). A correlation between LV to PA gradient and LVEDV was not apparent in this small group. LVEDV did decrease from 115 to 68% of normal in one patient following PDA ligation.

LV systolic output (SO) showed similar trends. This variable averaged 119 \( \pm 46\% \) (NS) in patients with an intact septum and showed a definite increase with increasing age from 96% in group IA (NS), 112% in IB (NS), to 134% in IC (\( P < 0.002 \)). In the four patients with PS, LVSO ranged from 64 to 136%. This variable averaged 158 \( \pm 57\% \) in VSD patients (\( P < 0.01 \)) and 112 \( \pm 41\% \) in patients with VSD + PS (NS).

LV ejection fraction was normal in all groups averaging 0.66 \( \pm 0.10 \) in patients with intact ventricular septum, 0.60 \( \pm 0.10 \) in VSD patients, and 0.64 \( \pm 0.09 \) in patients with VSD + PS. In the intact septum group, LVEF increased with increasing age from 0.59 in IA (NS), to 0.67 in IB (NS), to 0.73 in IC (\( P < 0.005 \)).

RV/LV Comparisons

The ratio, RVEDV/LVEDV was increased from the normal value of 1.00 to 1.46 \( \pm 0.33 \) in group I (\( P < 0.002 \)). This variable averaged 1.17 \( \pm 0.40 \) in VSD patients (NS) and 1.20 \( \pm 0.35 \) in patients with VSD + PS (NS). This variable was >1.00 in all but one patient with an intact ventricular septum who were studied. RVEDV/LVEDV was <1.00 in four VSD patients with large pulmonary flows and in one patient with VSD + PS and slightly increased.
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pulmonary flow. The ratio, RVSO/LVSO, showed similar findings.

The ratio, RVEF/LVEF, averaged 0.78 ± 0.17 for all intact septum patients (P < 0.02), 0.94 ± 0.26 in VSD patients (NS), and 0.91 ± 0.28 in VSD + PS patients (NS). This variable decreased in intact ventricular septum patients with increasing age averaging 0.93 ± 0.19 in group 1A, 0.68 ± 0.09 in IB, and 0.67 ± 0.14 in IC.

Discussion

Elevated right ventricular end-diastolic volumes and depressed ejection fractions were present in the majority of TGA patients with an intact ventricular septum over one month of age and constitute evidence for impaired right ventricular function in these patients if one can assume that preload is either normal or increased and afterload is normal or decreased. Preload can be estimated from end-diastolic volume measurements but may be more analogous to end-diastolic stress which is a function of pressure, dimensions, and wall thickness. There are no models currently applicable to measure right ventricular stress in man. Studies on left ventricular stress in man and in animals with chronic pressure or volume overload have demonstrated that calculated wall stress usually remains normal due to an appropriate degree of hypertrophy. It would seem unlikely that an inappropriately large degree of RV hypertrophy would be present in TGA and thus decrease wall stress to less than normal. Thus in TGA, preload is probably normal or increased.

Afterload also has been estimated from left ventricular wall stress measurements during systole and has again been found to remain in the normal range with chronic left ventricular pressure and volume overload.

Thus, the depressed RV ejection fraction in patients with TGA is unlikely to be due to abnormal loading conditions (that is, an increased afterload or a decreased preload) and is probably due to depressed right ventricular function. Despite this depression of RV function, normal or in some cases increased RV output is maintained through the Frank-Starling mechanism as a result of an increased end-diastolic volume. These findings are in agreement with similar data published by Jarmakani and Canent. These authors studied 14 patients with TGA and intact ventricular septum and found elevated RVEDVs and depressed RV ejection fractions in the majority of their patients.

It would be tempting to speculate that relative myocardial hypoxia might have contributed to elevated RVEDVs and depressed EFs. There was no statistically significant correlation, however, between these variables and systemic O2 saturation. In only one instance was there a known episode of prolonged severe hypoxia requiring resuscitation and prolonged ventilation which might be correlated with later measurements of RV function. This infant had an RVEDV of 218% and an RVEF of 0.36 (55% of normal) when studied 16 months following this severe episode of hypoxia.

Another factor which may play a role in RV function in TGA is the geometry of the chamber itself. Normally, the right ventricle functions at lower diastolic and systolic pressures than does the left ventricle, and its bellows-like configuration and pumping action may well be best adapted for low pressure cardiac work. We have not found significant differences in RV and LV end-diastolic volumes or ejection fractions in infants or children with normal right hearts. In addition, patients with a moderate or severe RV pressure overload secondary to pulmonary stenosis have shown normal RVEDVs and ejection fractions, and thus this type of pressure overload alone does not result in altered RV function. There are no data available on RV volume and ejection fraction in corrected transposition without associated defects or rhythm abnormalities. This lesion would be the closest analogy to complete TGA without cyanosis and RV volume data would be of great interest.

Tricuspid incompetence has been reported in postoperative TGA patients and is another factor which might contribute to abnormal RV function in these infants. None of the patients in the present study had significant tricuspid incompetence as judged both by RV cines and by physical exam.

Although the number of patients is small, the presence of a large VSD and normal pulmonary vascular resistance (PVR) in patients with TGA is associated with elevated RVEDVs and a normal or increased ejection fraction. Although RVEDV was not significantly different in VSD patients from patients with an intact septum, one might expect a larger volume in the VSD group if the left-to-right shunt is large, as was found by Jarmakani and Canent. With a decrease in VSD size or elevation of PVR, both RV end-diastolic volume and ejection fraction decrease. These findings are consistent with an increase in afterload (or total resistance to ventricular emptying) produced by the decrease in VSD size or increase in PVR affecting ejection fraction directly. Thus, no change in RV contractility is necessary to explain these data. It will be of considerable interest to evaluate RV volume and ejection fraction in these patients following a successful corrective procedure to learn whether or not abnormal RV function may be unmasked by closing the VSD and increasing afterload to a normal level.

Patients with ventricular septal defect and
pulmonary stenosis showed the smallest RVEDV values and only two of five patients had volumes definitely greater than normal. One of these patients had a patent ductus and demonstrated a decrease in RVEDV and output following ductal ligation. Three of the five patients with VSD + PS had low ejection fractions. In one of these patients, the ejection fraction was 0.48 at three months and 0.43 at 14 months with the systemic O₂ saturation unchanged at 65%.

Right atrial volumes were increased in 21 of 22 studies and averaged 185% of normal. This increase probably reflects increased flow through the atrium. An additional factor which might play a role in the enlarged right atria is the systemic pressure of the right ventricle with resultant possible alterations from normal of RV distensibility. RV end-diastolic pressure averaged 7.7 ± 2.8 mm Hg for the 22 TGA patients in this study with RAMax determinations versus 6.1 ± 1.8 mm Hg (P < 0.05) for 29 patients with normal right hearts from whom the normal RAMax values were derived. This small increase in RVEDP, however, does not necessarily indicate a decrease in RV distensibility since the volumes in the TGA group obviously were larger.

The left ventricular volume data presented herein are similar to those previously reported. Left ventricular volumes and output generally are normal in infants with TGA and intact ventricular septum early in the first year of life and increase with increasing age. In the presence of left ventricular outflow tract obstruction of even relatively mild degree, however, LVEDV and output usually are normal. Patients with an associated VSD have large LVEDVs and output unless pulmonary vascular disease is present.

The comparison of RVEDV and LVEDV and RVSO and LVSO showed that the vast majority of patients had RV/LV ratio > the normal value of 1.00. Exceptions to this finding were present only in patients with very high pulmonary flow and in one additional patient with VSD + PS with normal RV volumes. The use of RV/LV ratios is not particularly informative in TGA in view of the parallel pulmonary and systemic circuits, and thus total right and left ventricular outputs can vary independently. It is of interest, however, that with rare exceptions the right ventricle is larger than the left ventricle in patients with transposition and intact ventricular septum.

The methods used for volume calculations are particularly important. The right ventricle in TGA is considerably hypertrophied and the question has arisen regarding the validity of using the model for volume calculation for normal right ventricles in TGA patients. The Simpson’s rule method which we use for volume calculations is theoretically well suited for irregular ventricular outlines which can be present in these patients. We have published previously results in three patients studied following Mustard’s correction for TGA in whom RV and LV stroke volumes derived from cine volume calculations were compared. RV stroke volumes were 31.4, 26.7, and 24.6 ml and LV stroke volumes were 33.9, 25.1, and 29.4 ml, respectively, in these patients. In addition we have compared indicator dilution outputs obtained just prior to cineangiography with cine-derived outputs using RV volume data in four additional patients recently studied one year or more following Mustard’s correction for TGA. These outputs varied by <10% in three of four patients with an average difference for all patients of only 7.8%. These data suggest that RV volume calculations can be performed in TGA patients with a degree of accuracy which is similar to that found with duplicate indicator dilution determinations.

From the data presented we have made the following conclusions regarding right heart performance in TGA.

1. In the perinatal period RVEDV and output may be normal or only mildly elevated as is the case for LVEDV and output. This finding is consistent with undisturbed intrauterine hemodynamics in TGA.

2. Patients with TGA and intact ventricular septum who are beyond the neonatal period have RVEDVs which are considerably elevated. In addition, a large number of these patients have depressed RV ejection fractions. The cause for this abnormality of RV function is unknown, but one might speculate that a discrepancy between high myocardial oxygen demands and low systemic oxygen saturation with resultant low grade chronic myocardial hypoxia could be a contributing factor. There are no supporting data for this speculation at present.

3. Patients with TGA and large VSDs have increased RVEDVs and output after the neonatal period unless significant pulmonary stenosis is present in which case these variables are usually normal or only mildly increased. A decrease in VSD size or development of pulmonary vascular obstructive disease is associated with a decrease in RVEDV, output, and ejection fraction.

4. Right atrial volume is increased in virtually all TGA patients and appears to increase progressively with increasing age.

5. Right ventricular volume and output are usually greater than left ventricular volume and output with exceptions noted in patients with very high pulmonary flow.

6. History and physical examination usually do not lead one to suspect abnormal RV function in many patients whose volumes and ejection fractions are clearly abnormal. Long-term follow-up of these
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patients will be extremely important in assessment of any residual dysfunction which may be present following surgical therapy.

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