Left Ventricular Function Following Attempted Surgical Repair of Tetralogy of Fallot

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SUMMARY Left ventricular function was assessed by angio- graphic methods in 40 patients (ages 3-27), 1-12 years following repair of tetralogy of Fallot. Twenty of the 40 patients (group A) had a satisfactory repair with a pulmonary-systemic flow ratio (Qp/Qs) ≤ 1.5 and a right ventricular systolic pressure of ≤ 60% of the left ventricular value. The other 20 patients (group B) had a Qp/Qs > 1.5 and/or a right ventricular systolic pressure > 60% of the left ventricular value. Group A patients had a normal left ventricular end-diastolic volume (LVEDV), end-diastolic pressure (LVEDP), ejection fraction (LVEF), and mass (LVM), while the group B patients had significantly elevated LVEDV, LVEDP, LVM, and a significantly depressed LVEF. These findings indicate that patients with a satisfactory hemodynamic repair of tetralogy of Fallot have normal left ventricular function while patients with postoperative unsatisfactory hemodynamics have significantly impaired left ventricular function.

Although repair of tetralogy of Fallot (TOF) has become a well-established surgical procedure, only limited information concerning postoperative left ventricular (LV) performance is available. Following successful repair, elevated left ventricular end-diastolic volume (LVEDV) and depressed ejection fraction (LVEF) were reported by Jar- makani et al. using angiographic analyses. However, Sunderland et al. demonstrated normal LV function post- operatively, based on measurements of systemic arterial systolic pressure, cardiac index, arteriovenous oxygen difference, and LVEF, among patients who had undergone repair under two years of age, none of whom had prior palliative procedures. LaCorte et al. also noted a normal LVEF using echocardiographic techniques among older patients following successful surgery.

It is the purpose of this communication to report our observations regarding LV function derived from angiographic analysis obtained at cardiac catheterization of 40 patients who had previously undergone repair of TOF and to attempt to identify factors responsible for the LV dysfunction noted in some.

Material and Methods

From January 1965 to June 1975, 102 patients underwent cardiac catheterization at the Children's Hospital Medical Center of Boston, one to 12 years following repair of TOF. Prior to 1973, postoperative cardiac catheterization at our institution was performed only when significant residual defects were suspected, but in the past four years, it has been recommended to all patients one year following repair of TOF. Angiographic analysis of left ventricular function was available in only 40 of these patients and these form the basis of this report. The ages at surgery ranged from 2-25 years (mean 9) and at the time of catheterization, from 3-27 years (mean 13). For the purpose of this study, TOF was considered to be an underdevelopment of the subpulmonary infundibulum with an underdeveloped parietal band (the crista supraventricularis) obstructing the right ventricular outflow and at the same time creating by its dissociation from the normal septal band, the ventricular septal defect. At the time of surgery, a right ventricular (RV) outflow patch was placed in 25, extending across the pulmonary valve anulus in 12 of these. Clinically, at the time of catheterization, a murmur of pulmonary regurgitation was audible in 33 patients and cardiac enlargement on chest X-ray was present in 35. Catheterization was performed under light sedation using Meperidine 25 mg, Phenergan 6.25 mg, Thorazine 6.25 mg/ml at 1 ml/30 lbs, the maximum dose being 2 ml. All patients underwent both right and left heart pressure and oxygen saturation measurements, including oxygen consumption determinations. Pulmonary and systemic blood flows were calculated using the Fick principle.

The patients were then divided into two groups according to the following hemodynamic findings: group A (20 patients), with pulmonary-systemic flow ratios Qp/Qs ≤ 1.5/1 and RV systolic pressure < 60% of the LV value (table 1) and group B (20 patients) with Qp/Qs > 1.5/1 and/or RV systolic pressure > 60% of the LV value (table 2).

LV biplanes were performed in all, using cineangio- graphy at 60 frames/sec in 28 and full size cutfilm at 6 frames/sec in the remaining 12 patients. A signal marking exposure time, recorded simultaneously with the electrocardiogram, permitted the exact timing of films. In all cases, the measurements of three films in end diastole and three in end systole were averaged. When premature or irregular activation occurred, no films were used unless at least three normal cycles separated the beat from the extrasystole.

The LVEDV and LVEF were calculated using the area method described by Dodge et al. and LV mass (LVM) was measured utilizing the technique of Rackley et al. Normal predicted values for each patient were derived from a regression equation obtained from 30 patients over two years of age without significant heart disease, none of whom had any LV outflow tract gradients (LVEDV ml = 129.1 (BSA) - 47 ± 15.2; LVM gm = 96.1 (BSA) - 22.1 ± 12.6). These values are similar to those of other investigators. The LVEDV and LVM were then expressed both as indexed values (tables 1, 2) and as a percentage of normal (fig. 2) utilizing the equation:

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Mean 11.2

TABLE 4. Statistical analyses with those of enlargement on left ventricle; SI = systemic index (Fick); BSA = body surface area; EDV = end-diastolic volume; EF = ejection fraction; EDP = end-diastolic pressure; ± 1 SD = ± 1 standard deviation.

% Normal LVEDV or LVM = Calculated value × 100
Normal predicted value

Preoperative LV volume data were available for comparison in eight group A and in five group B patients (table 3).

Furthermore, in an attempt to determine if the LV dysfunction in group B patients was due solely to a large residual ventricular septal defect (VSD), the data of seven of this group with a Qp/Qs ≥ 2/1 and without significant pulmonary stenosis (gradient ≤ 30 mm Hg) were compared with those of seven patients, unoperated, with an isolated large VSD, matching Qp/Qs and body surface area (table 4). Statistical analyses were performed using the group and paired Student's t-tests.

Results

Group A—Satisfactory Hemodynamic Repair

Among 13 of these 20 patients, an RV outflow patch had been placed; and at the time of catheterization, 18 had a murmur of pulmonary regurgitation and 16 had cardiac enlargement on chest X-ray.

Cardiac catheterization was performed 1–6 years postoperatively (mean 2). The LVEDV, LVM, LVEF, and systemic indexed blood flow (SI) were normal and LV end-diastolic pressure ranged from 4–12 mm Hg (mean 7.4) (table 1, figs 1 and 2). There was no significant correlation between these parameters of postoperative LV function and preoperative hemoglobin and systemic oxygen saturation levels, presence or absence of a systemic-to-pulmonary artery shunt or the age of the patient at the time of surgery.

When the postoperative LV function was compared with preoperative values available in eight patients, no appreciable differences were observed in six (table 3). In the other two, LVEDV and LVM were noted to have decreased significantly toward normal values; both the patients had a significant left-to-right shunt preoperatively, in one through a large Waterston shunt for seven years, and in the other via the VSD (cyanotic TOF, Qp/Qs = 2.4/1).

Group B—Unsatisfactory Hemodynamic Repair

In 12 of these 20 patients, an RV outflow patch had been placed. At the time of postoperative catheterization, a murmur of pulmonary regurgitation was present in 15 and cardiac enlargement on chest X-ray was evident in all but one.

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Cardiac catheterization was performed 1–12 years postoperatively (mean 3). Both LVEDV and LVM were significantly elevated while LVEF and SI were significantly decreased. The mean end-diastolic pressure was elevated at 11.5 mm Hg (range 5–20) (table 2, figs. 1 and 2). As in group A, there was no significant correlation between these parameters of postoperative LV function and preoperative hemoglobin and systemic oxygen saturation levels, presence or absence of a systemic-to-pulmonary artery shunt or the age of the patient at the time of surgery. A large residual VSD was present in 16, and a large residual systemic-to-pulmonary artery collateral (Qp/Qs 2.4/1) was noted in another. Among the remaining three patients, residual significant RV outflow obstruction was the predominant hemodynamic lesion, and LVEDV and LVM were only mildly elevated while the LVEF was normal. There was a significant correlation in this group of patients between the magnitude of the residual Qp/Qs and LVEDV (r = 0.88); weaker associations existed between Qp/Qs and LVM (r = 0.74) and LVEF (r = 0.68).

When postoperative LV function data were compared with preoperative values available in five patients (one of whom had a large Potts anastomosis for 15 years in association with the most abnormal values), LVEDV and LVM were noted to have increased while LVEF decreased significantly (table 3).

Three group B patients have been restudied one year following successful closure of the residual ventricular septal defect (table 5). In all three, there has been considerable improvement in left ventricular function in that LVEDV and LVM have decreased while LVEF has increased.

The comparison of LV function data between seven group B patients with a residual Qp/Qs ≥ 2/1 and seven with unoperated VSD matched for Qp/Qs and body surface area, revealed the LVEDV, LVM, and LVEF to be significantly more abnormal in the postoperative TOF patients (table 4).

**Discussion**

Information concerning LV function following surgical repair of TOF is limited and conflicting. In a report concerning 24 patients (at least 13 of whom were ≥ 4 years of age),...
age at the time of surgery), who underwent catheterization five days to eight years postoperatively (mean 2.5 years). Jarmakani noted that LVEDV was slightly but significantly elevated and LVEF was significantly depressed despite successful repair. In contrast, Sunderland et al. observed that LV function was normal among 17 children who underwent successful surgery prior to the age of two years. In addition, LaCorte et al., using echocardiographic techniques among 15 patients aged 2–21 years (mean 10), also noted a normal LVEF postoperatively following successful repair.

The data from our patients, all two years of age or more at the time of surgery, substantiate these latter observations in that LV function, in terms of LVEDV, LVM and LVEF, systemic indexed flow, and LV end-diastolic pressure, is normal provided a satisfactory hemodynamic result is achieved. Abnormal LV function by these criteria was evident only in those with a poor surgical result. The dominant hemodynamic lesion responsible for these abnormalities was a significant residual VSD, the magnitude of the LVEDV and LVM elevations and LVEF depression being related to the Qp/Qs. Three patients in our series have been restudied one year following repair of the residual ventricular septal defect and in all we observed marked improvement in left ventricular function. These data suggest that the abnormal left ventricular function observed in our B group patients was due largely to the presence of a large residual VSD and that significant improvement in LV function can occur following closure of the VSD.

In addition to the effect of a large residual VSD, there are other factors that also contribute to the abnormal left ventricular function following repair of tetralogy of Fallot. The magnitude of the left-to-right shunt does not completely explain the degree of LV dysfunction observed in our patients since significantly less abnormal elevations of LVEDV and LVM and a normal LVEF were noted among our matched group of patients with an uncomplicated unoperated VSD. The reasons for these differences are unclear, but speculations may be justified. Abnormalities of LV function have been reported by other investigators following large systemic-pulmonary anastomoses in patients with unrepaired TOF. Similarly, among our five patients with such shunts, preoperative LVEDV and LVM were significantly elevated although LVEF was decreased in only one. However, following corrective surgery, these abnormalities tend to return to normal when the repair is satisfactory as was observed in two of these patients. They may remain abnormal if correction is unsatisfactory as evidenced by the substantial increases in both LVEDV and LVM with LVEF depression which occurred in the other three, all with a poor result. Since focal fibrosis, hypoplasia of the LV and diffuse fibrosis of the RV have been noted in patients with TOF by others, it is conceivable that the additional hemodynamic load of a large residual shunt may cause significant LV dysfunction. It is unlikely that the pulmonary regurgitation, which is frequently present postoperatively, can alone cause LV dysfunction, as normal LV function was noted in five who had such a murmur among the 17 patients reported by Sunderland and in 18 of the 20 patients in our series with a satisfactory repair. However, TOF is a considerably more

**TABLE 5. Pertinent Hemodynamic Findings after Surgical Repair of a Residual VSD in Three Group B Patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>RV PSA</th>
<th>LV EDV (%NL)</th>
<th>LV Mass (%NL)</th>
<th>EP (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>#1</td>
<td>#2</td>
<td>#2</td>
<td>#1 #2</td>
<td></td>
</tr>
<tr>
<td>23 55/8</td>
<td>20 110/8</td>
<td>1 200 144 224 150 58 66</td>
<td>31 51/12</td>
<td>25 130/11</td>
</tr>
</tbody>
</table>

Abbreviations: RV = right ventricle; PSA = pulmonary stenosis gradient; LV = left ventricle; EDV = end-diastolic volume; EF = ejection fraction; #1 = LV function original postoperative value #2 = LV function following repair of residual VSD.
complex lesion than a simple VSD, requires much more extensive surgery, and it is possible that the cumulative effects of a right ventriculotomy, with an outflow patch and pulmonary regurgitation, may in the presence of a large residual VSD significantly impair LV function more than a comparably-sized uncomplicated VSD. Graham and co-workers\(^\text{17}\) have demonstrated marked abnormalities in right ventricular end-diastolic volume and right ventricular ejection fraction in postoperative TOF patients. They have shown that these abnormalities of right ventricular function appear to be related to the presence of a noncontractile patch in the right ventricular outflow area. There is also some experimental evidence to suggest that right ventricular dysfunction can affect LV performance. Walsh and co-workers\(^\text{18}\) have observed alterations in both right and left ventricular myosin following experimentally induced chronic pulmonary and tricuspid regurgitation in dogs.

In summary, our findings indicate that LV function, in terms of LVEDV, LVM, LVEF and systemic index, is normal following successful repair of TOF. A large residual VSD causes significant abnormalities of these parameters of LV function and we recommend surgical closure of the VSD with a Qp/Qs > 1.5/1.

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