

Direction of Preoperative Ventricular Shunting Affects Ventricular Mechanics After Tetralogy of Fallot Repair

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Background—Tetralogy of Fallot (TOF) typically results in clinical cyanosis or volume overload of the left ventricle (LV), depending on the direction and magnitude of shunting across the ventricular septal defect (VSD). The present study examines the effects of surgical TOF repair on LV mechanics and compares these changes between patients with VSD shunts that are predominantly right-to-left (R-L; “blue TOF”) and those with VSD shunts that are predominantly left-to-right (L-R; “pink TOF”).

Methods and Results—Eleven patients (6 R-L and 5 L-R) 4.3 to 18.4 months old (median 7.1 months old) were studied. LV end-diastolic area (EDA) was calculated from transesophageal echocardiograms obtained during initiation and weaning of cardiopulmonary bypass. LV end-diastolic pressure was measured by micromanometer. Compliance was assessed by end-diastolic pressure-area curves. Contractility was assessed from preload recruitable stroke work by the stroke work-versus-LV EDA relation. VSD shunt direction was determined by preoperative Doppler echocardiography. Changes in LV function at the conclusion of cardiopulmonary bypass included decreased stroke area (from 6.6 ± 0.9 to 4.1 ± 0.4 cm²/m², $P=0.012$) and ejection fraction (from $55 \pm 2\%$ to $41 \pm 3\%$, $P<0.001$). LV EDA at a common pressure in 8 patients decreased (from 10.4 ± 1.4 to 7.6 ± 1.2 cm²/m², $P=0.003$), which suggests a decrease in ventricular compliance. Additionally, the end-diastolic pressure-area curves shifted to the left in all patients. Preload recruitable stroke work decreased (from 34.8 ± 2.4 to 21.8 ± 2.6 mm Hg, $P=0.007$), which demonstrates a decrease in ventricular contractility. When separated by preoperative shunt direction, LV EDA increased in R-L patients by 0.9 ± 0.5 cm²/m² postoperatively but decreased in L-R patients by 4.3 ± 0.8 cm²/m² ($P<0.001$). Area ejection fraction decreased in all patients independent of shunting or change in LV EDA.

Conclusions—LV diastolic and systolic function are depressed after TOF repair. Mechanical effects of the VSD patch and myocardial depressant effects of ischemia and reperfusion during surgery probably contribute to the observed changes in LV mechanics. Different effects of surgical repair on LV preload in pink and blue TOF also contribute to the spectrum of clinical results observed after surgery. (*Circulation*. 2008;118:2338-2344.)

Key Words: tetralogy of Fallot ■ pediatrics ■ ventricular function ■ surgery ■ cardiopulmonary bypass

Tetralogy of Fallot (TOF) is a cyanotic congenital heart lesion with a prevalence of 0.26 to 0.80 per 1000 live births, accounting for approximately 10% of all cases of congenital heart disease.¹ The surgical repair of TOF involves closure of the ventricular septal defect (VSD) and relief of the right ventricular (RV) outflow tract obstruction. Originally, surgical repair of TOF was performed in older children; however, current clinical management involves complete repair in infancy, usually before the age of 1 year. These surgical corrections result in several acute physiological changes, including a decrease in RV hypertension and the elimination of ventricular shunting, which results in a change in the volume load of both ventricles.

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Although the postoperative changes in function and mechanics of the RV and interventricular septum have been studied extensively,²⁻⁶ the effects on the left ventricle (LV) have not been fully evaluated in the modern surgical era. We aimed to describe the acute changes in LV mechanics and function after surgical TOF repair and to determine whether the direction of preoperative VSD shunting affects these changes.

Methods

Intraoperative data were collected prospectively from 1997 to 2004. Patients were enrolled before surgery for complete repair of TOF. Method of repair was determined by the surgeon at the time of

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surgery on the basis of available clinical information. The Columbia University Institutional Review Board approved the protocol, and informed consent was obtained from each patient's legal guardian.

Clinical Data

Patient demographics, including age, weight, and body surface area at the time of surgery, prior interventions, and associated anatomic anomalies were noted. Intraoperative variables recorded included type of surgical repair, total cardiopulmonary bypass time, aortic cross-clamp time, and the presence or absence of a residual atrial communication. Preoperative transthoracic echocardiograms were analyzed for RV outflow tract anatomy, and a pulmonary annulus diameter was measured in the parasternal long axis. The absolute diameter was converted to a z score based on body surface area. The predominant direction of shunting through the VSD preoperatively was determined by qualitative assessment of color Doppler images by a clinical echocardiographer who was independent of the study. For further analysis, patients were divided into 2 groups based on the predominant direction of blood flow through the VSD, right-to-left (R-L) or left-to-right (L-R).

Intraoperative Data Acquisition

All patients were anesthetized, heparinized, and cannulated for cardiopulmonary bypass per clinical protocol. A 5F MPC500 micromanometer-tipped catheter (Millar Instruments, Houston, Tex) was inserted through a purse-string suture in the proximal ascending aorta and advanced into the LV. All waveform data, including ventricular pressure, systemic arterial pressure, and surface ECG, were digitized at 200 Hz and imported via a 16-channel analog-to-digital converter (ADInstruments Inc, Milford, Mass) to a portable computer (Apple Computers, Cupertino, Calif). Transesophageal short-axis echocardiograms at the level of maximal LV area were obtained with a Sonos 5500 ultrasound system (Hewlett-Packard, Palo Alto, Calif) and recorded on videotape.

Pressure data and echocardiograms were recorded in steady state periods (periods of stable ventricular preload without changes in vasoactive medications) immediately before and after cardiopulmonary bypass and during ventricular emptying and filling on initiation and withdrawal of bypass. To facilitate correlation of beats, pressure and ECG data were recorded with the echocardiographic images, and artifacts were added to the pressure channel. This allowed for accurate beat-to-beat comparison of the echocardiographic images with the pressure tracings.⁷

Data Analysis

Echocardiograms were planimeted offline on a VingMed CFM800 (GE Healthcare, Chalfon St. Giles, United Kingdom). During the steady state period, cyclic variation in the mean arterial pressure was used to identify beats at end expiration, which were then used for analysis. During preload alteration with initiation and withdrawal of cardiopulmonary bypass, all beats were reviewed, and beats were excluded from analysis for atrial or ventricular ectopy, poor echocardiographic quality, or artifacts in the pressure signal. End-diastolic area (EDA) and end-systolic area were measured by planimetry of the LV endomyocardial borders in accordance with American Society of Echocardiography standards. The eccentricity index was used as an index of geometry and was defined as the ratio of the septal-free-wall diameter to the anterior-posterior diameter in short-axis images of the LV, with a value of 1 indicating circular cross-sectional geometry.⁸ LV mass was approximated by A_m , defined as the EDA subtracted from the area created by the epicardial tracing in end diastole. A_m versus EDA was plotted and modeled as reported previously to correct for changes in end-diastolic volume.⁹ The corrected postoperative A_m was used for comparison with the preoperative A_m .

With the use of artifacts on the ventricular pressure tracing, corresponding beats were identified on the echocardiogram to correlate EDA and end-diastolic pressure (EDP) in the same cardiac cycle. Pressure was differentiated digitally. EDP was identified as the pressure that preceded the rapid upstroke, determined from the rate of increase of ventricular pressure (dp/dt). EDP was plotted against EDA for the creation of compliance curves and calculation of

diastolic indices. This process generally yielded a data set of 10 to 15 beats for analysis at the initiation and withdrawal of bypass. Prebypass and postbypass ventricular and arterial pressures were analyzed with custom routines developed in Matlab (The Mathworks Inc, Natick, Mass), including identification of EDP and mean ejection pressure.

Functional Indices

All measurements of area were normalized to patient body surface area. Stroke area (SA) was used as a surrogate for stroke volume and was defined as $SA = EDA - \text{end-systolic area}$. Ejection fraction (EF_a) was calculated from the equation $EF_a = (SA/EDA) \times 100\%$. Preload recruitable stroke work, a load-independent measure of contractility, was defined as the slope of the linear regression between stroke work [$SW = SA \times (\text{mean ejection pressure} - \text{EDP})$] and EDA. Cardiac index (CI) was calculated by the formula $CI = SA \times \text{heart rate}$. Diastolic function was assessed by the creation of end-diastolic pressure-area curves by fitting data to the equation $EDP = \alpha e^{\beta(EDA)}$, where α and β are curve-fitting constants. For all curves, a correlation coefficient (r) was calculated to evaluate the goodness of fit.

Statistical Analysis

Data are expressed as the group mean \pm SEM. The paired t test was used to analyze prebypass/postbypass data within groups, and the Student t test was used to compare differences between shunting groups. Repeated-measures ANOVA was used to compare prebypass/postbypass data between shunting groups. All data were analyzed with SAS system software (SAS Institute, Cary, NC). Significance was defined as a probability value less than 0.05. Probability values were not adjusted for the multiple tests.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Patient Characteristics

Eleven patients were studied at the time of full repair of TOF at a median age of 7.1 months (range 4.3 to 18.4 months), with a mean weight of 8.1 kg (range 6.9 to 10.0 kg) and mean body surface area of 0.39 m² (range 0.34 to 0.48 m²). Previous interventions were performed in 3 patients, which consisted of 2 modified Blalock-Taussig shunts and 1 central shunt, all performed for cyanosis in the newborn period. These 3 patients did not differ from their unshunted counterparts in any preoperative characteristics (ie, hemoglobin concentration, LV dimensions, or systolic function) except for age. These patients were 6.7, 15.1, and 18.4 months of age at the time of repair, somewhat older than the rest of the patients studied.

A pulmonary valve annulus-sparing procedure was done in 8 patients, and a transannular patch was used in 2. Of the 8 patients with an annulus-sparing procedure, 5 had extensive infundibular resections without the need for an infundibular patch, and 3 did not require extensive infundibular resection. The remaining patient required an RV-to-pulmonary artery homograft for an anomalous course of the left circumflex artery crossing the RV outflow tract. Mean cardiopulmonary bypass time was 80 ± 40 minutes, with a mean aortic cross-clamp time of 42 ± 16 minutes.

No patient had a residual atrial communication postoperatively. VSD patch leaks were identified in 4 patients; all were trivial shunts according to color Doppler echocardiogram and restrictive to pressure based on Doppler waveform analysis. Seven of the 8 patients undergoing valve-sparing procedures had residual pulmonary stenosis, with a mean gradient of

Table 1. Mean Prebypass and Postbypass Values for All Patients

	Pre-CPB	Post-CPB	<i>P</i>
Heart rate, bpm	132±5	158±5	0.004
PRSW, mm Hg	34.8±2.4	21.8±2.6	0.007
LV SA, cm ² /m ²	6.6±0.9	4.1±0.4	0.012
LV SW, cm ² · mm Hg/m ²	279±54	171±21	0.032
Ejection fraction, %	56±2	41±3	<0.001
Cardiac index, cm ² · m ² /min	890±133	683±49	0.15
LV EDA at constant pressure, cm ² /m ²	10.4±1.4	7.6±1.2	0.003
A _m , cm ² /m ²	10.6±1.2	12.5±1.3	0.014

CPB indicates cardiopulmonary bypass; PRSW, preload recruitable stroke work; LV SA, LV stroke area; LV SW, LV stroke work; and A_m, EDA-corrected myocardial area.

30 mm Hg (range 13 to 46 mm Hg) on postoperative transthoracic echocardiogram. All patients were in normal sinus rhythm before and after cardiopulmonary bypass, and all exhibited RV conduction delay or a right bundle-branch block pattern on postoperative 12-lead surface ECG.

Preoperative echocardiograms showed predominantly R-L shunting through the VSD in 6 patients and L-R shunting in 5 patients. The mean preoperative hemoglobin for the R-L shunting group was 15.8±3.2 g/dL, which was significantly higher than the mean in the L-R shunting group (11.8±0.4 g/dL; *P*=0.025). Only 1 patient in the R-L shunting group had a hemoglobin value in the normal range for age. Furthermore, the mean pulmonary valve annulus diameter *z* score was significantly smaller in patients in the R-L shunting group than in the L-R shunting group (−2.7±0.4 versus −0.5±0.3, *P*=0.001). Only 1 patient in the R-L shunting group had a *z* score greater than −2 (−1.76), and no patient in the L-R shunting group had a *z* score smaller than −1.2. There were no other significant differences in baseline clinical characteristics between the 2 groups. One patient in

each group required a transannular patch as part of the surgical repair.

Postoperative Changes

Steady state parameters were compared from immediately before and after cardiopulmonary bypass (Table 1). Systolic function decreased immediately after bypass, as exhibited by a decrease in mean LV SA (from 6.6±0.9 to 4.1±0.4 cm²/m², *P*=0.012) and mean ejection fraction (from 56±2% to 41±3%, *P*<0.001). Despite this decrease in function, there was no significant decrease in calculated cardiac index (*P*=0.28), because mean heart rate increased from 132±5 to 158±5 bpm (*P*=0.004). Additionally, there was an increase in mean corrected A_m from 10.6±1.2 cm²/m² before bypass to 12.5±1.3 cm²/m² after bypass (*P*=0.014).

Complete data during preload alteration via initiation and weaning from cardiopulmonary bypass were available in 8 patients. Three patients were excluded from this analysis because of technically poor echocardiograms. Similar to the steady state data, LV systolic function as measured by preload recruitable stroke work decreased in all patients from a mean of 34.8±2.4 mm Hg before bypass to a mean of 21.8±2.6 mm Hg after bypass (*P*=0.007). To assess diastolic function, individual compliance curves were created for each patient. All curves had a correlation coefficient value (*r* value) between 0.90 and 0.99. In each individual patient, the curve shifted to the left after bypass, which indicates a postbypass decrease in ventricular compliance. Representative curves are presented in Figure 1. Furthermore, all 8 patients exhibited a decrease in LV EDA when compared at a constant pressure nearest to 5 mm Hg before and after bypass. The mean prebypass EDA at a constant pressure was 10.4±1.4 cm²/m², which decreased to a mean of 7.6±1.2 cm²/m² after surgical repair (*P*=0.003).

Effect of Preoperative VSD Shunt Direction

Preoperative measurements were compared between the 2 groups. Most measures were equal in the 2 groups (Table 2),

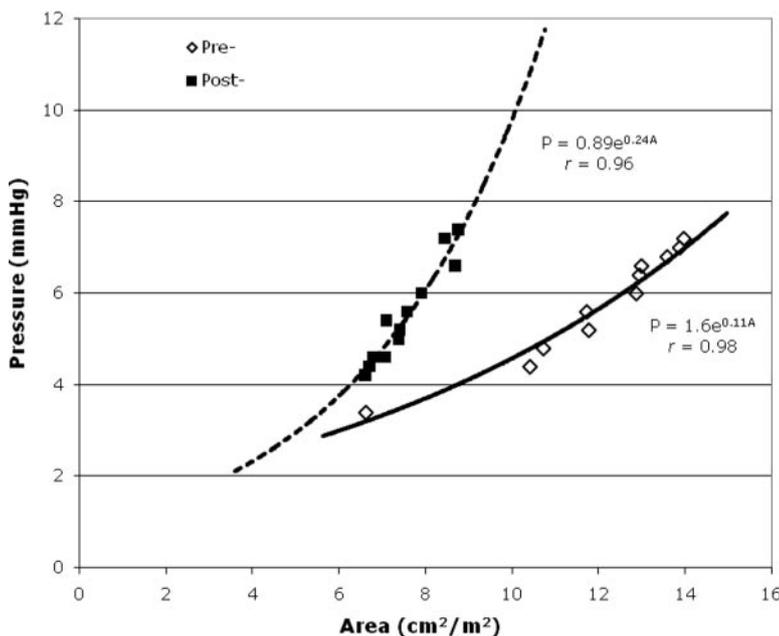


Figure 1. Representative pressure-area curve generated from data collected before (◇) and after (■) cardiopulmonary bypass.

Table 2. Mean Prebypass and Postbypass Values for Patients Separated by VSD Shunt Direction

	R-L (n=6)	L-R (n=5)
PRSW, mm Hg		
Pre-CPB	36.0±3.6	32.9±3.6
Post-CPB	21.8±3.7	21.9±5.0
Change, <i>P</i> =0.68	-14.2±4.5	-11.0±6.1
LV SA, cm ² /m ²		
Pre-CPB	5.0±0.8	8.6±1.5
Post-CPB	4.2±0.6	4.0±0.3
Change, <i>P</i> =0.01	-0.8±0.3	-4.6±1.3
LV SW, cm ² ×mm Hg/m ²		
Pre-CPB	215±24	360±111
Post-CPB	144±23	206±34
Change, <i>P</i> =0.34	-70±8	-154±95
EF _a , %		
Pre-CPB	54±3	56±2
Post-CPB	42±4	40±6
Change, <i>P</i> =0.55	-13±3	-16±5
LV EDA at constant pressure, cm ² /m ²		
Pre-CPB	8.6±1.4	12.2±2.3
Post-CPB	6.6±0.9	8.7±2.2
Change, <i>P</i> =0.26	-2.1±0.6	-3.5±1.0
A _m , cm ² /m ²		
Pre-CPB	10.9±1.5	10.0±2.1
Post-CPB	13.5±1.8	11.0±1.9
Change, <i>P</i> =0.28	2.6±1.0	1.1±0.5

CPB indicates cardiopulmonary bypass; PRSW, preload recruitable stroke work; LV SA, LV stroke area; LV SW, LV stroke work; EF_a, LV ejection fraction; and A_m, EDA-corrected myocardial area.

There was no significant difference between the R-L and L-R shunting groups in these parameters at baseline. *P* values are for the difference in the prebypass to postbypass change between the 2 groups.

with the exception of the end-diastolic eccentricity index, which was lower in patients with R-L shunting (0.61 ± 0.04 versus 0.85 ± 0.05 , $P=0.01$). There was no significant difference in the relative changes preoperatively to postoperatively between the 2 groups, and the difference between the groups persisted after bypass (0.51 ± 0.04 versus 0.78 ± 0.04 , $P=0.03$). Further comparisons between groups revealed a difference in the response of LV EDA and SA after repair. Steady state EDA increased by a mean of 0.9 ± 0.5 cm²/m² in the R-L shunting group, whereas it decreased by a mean of 4.3 ± 0.8 cm²/m² in the L-R shunting group ($P<0.001$; Figure 2). This resulted in nearly equal postoperative LV EDA mean values of 10.1 ± 1.6 and 11.1 ± 2.2 cm²/m², respectively ($P=0.7$). Although SA decreased in all patients, the decrease was significantly larger in patients with L-R shunting physiology (-4.6 ± 1.3 versus -0.8 ± 0.3 cm²/m², $P=0.013$; Figure 3). Similarly, there was a larger decrease in calculated cardiac index in patients with an L-R shunt (-460 ± 224 cm² · m²/min) than in the R-L shunting group (-5 ± 83 cm² · m²/min), although this difference was not statistically significant ($P=0.075$).

Discussion

Patients at one end of the spectrum of TOF have minimal RV outflow obstruction, or so-called *pink TOF*. These patients usually are acyanotic and are physiologically similar to patients with a simple VSD. At the other extreme are cyanotic patients with severe RV outflow obstruction, occasionally with complete pulmonary atresia, with the degree of cyanosis reflecting the degree of right-to-left shunting across the VSD. The treatment strategies for patients along this spectrum of disease may differ (ie, systemic to pulmonary shunt placement as a neonate versus full repair at an older age); however, all current methods require cardiac surgery with cardiopulmonary bypass.

The present study used pressure-area measurements to demonstrate distinct changes in systolic and diastolic function of the LV after repair of TOF in the modern surgical era. LV systolic function decreased in all patients, as measured by stroke area, stroke work, ejection fraction, and preload recruitable stroke work. Ventricular compliance also decreased, as demonstrated by a decrease in LV end-diastolic area at a constant end-diastolic pressure. This change in compliance was associated with a postoperative increase in myocardial mass in all patients.

Changes in LV geometry and loading after surgery were affected by the direction of preoperative VSD shunting. LV EDA increased in patients with predominant R-L shunting (blue TOF). Conversely, LV EDA decreased in patients with predominant L-R shunting (pink TOF). These divergent changes trended toward normalization of LV volumes, with convergence of postoperative LV EDA. Ventricular eccentricity also differed between these 2 groups preoperatively, and despite the convergence of LV EDA, this difference persisted postoperatively. This supports the view that elimination of anatomic lesions during surgery does not acutely normalize ventricular geometry.¹⁰ Rather, reversal of chronic abnormalities of ventricular shape requires a period of weeks postoperatively, which suggests active remodeling. Follow-up studies of this phenomenon are indicated.

Most of the changes in systolic and diastolic LV function we observed were independent of the direction of preoperative VSD shunting. These included decreased LV compliance and increased LV mass. This combination suggests myocardial edema due to hemodilution or ischemic injury.^{7,9} Decreased compliance was also associated with decreases in systolic function. This may indicate a need for better myocardial protection strategies; however, the degree of systolic and diastolic LV dysfunction did not correlate with the duration of cardiopulmonary bypass or aortic cross-clamp (data not shown). The impact of the ventricular septal patch on LV and septal function is unknown. Future studies examining the mechanical effects of septal patches on the septum and ventricular systolic and diastolic function are indicated.

Most previous studies of LV function in TOF repair involved older children and a previous era of surgical and cardiopulmonary bypass techniques.^{11–15} The differences between the conditions under which previously published data were collected and current practice are important, because it is likely that the ventricular mechanics of TOF and the effects of the surgical repair are affected by patient age.^{2,11,16}

Regarding unique surgical responses of TOF, Levin et al¹³ noted abnormalities of isovolumic systole that increased with

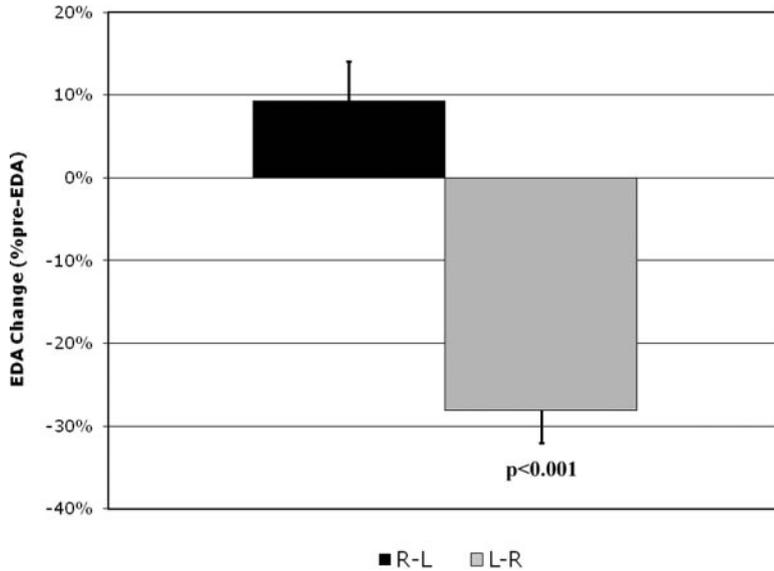


Figure 2. Change in postoperative LV EDA expressed as a percent change from preoperative LV EDA in patients with R-L shunting (black column) vs L-R shunting (gray column).

the severity of TOF. Lower peak LV pressures and a lower LV end-diastolic volume have also been noted in cyanotic TOF.^{11,12} In 1972, Jarmakani et al¹¹ reported decreased ejection fraction preoperatively and postoperatively. In addition, LV wall mass increased postoperatively, which suggests that myocardial edema contributed to decreased systolic function. Studies of RV restrictive physiology also imply limitations of intraoperative myocardial protection.⁶

Decreased postoperative LV ejection fraction was also reported by Lange et al¹² in 1982. In the study by Jarmakani et al,¹¹ LV end-diastolic volume increased after an elective L-R shunt but not after complete repair. This suggests that whereas increased LV preload tends to improve LV mechanics, other factors can cause a net decrease in systolic function postoperatively. The present data support this hypothesis (Figure 4).

RV ejection fraction is also subnormal before and after surgery.¹⁷ Shunting or repair normalizes RV end-diastolic volume similar to the normalization of LV EDA in the present study.¹⁷

Cullen et al¹⁸ associated RV restrictive physiology with prolonged postoperative recovery and clinical evidence of low cardiac output, despite normal LV systolic function. Others have confirmed that RV restriction increases morbidity postoperatively.^{6,18} It is speculative whether the RV or LV is the primary cause of low-output states after TOF repair, but when infundibulectomy is not extensive, LV dysfunction may dominate in some patients. Postoperative management of primary LV dysfunction and RV restrictive physiology differ in important respects. Accordingly, studies of the relative importance of RV and LV dysfunction in this population are needed.

The present data are the first to describe an effect of the direction of preoperative VSD shunting on preoperative and postoperative LV mechanics in TOF. Others have previously reported decreased LV volume in severe RV outflow tract obstruction and exaggerated R-L shunting.¹¹ Jarmakani et al¹¹ showed normalization of LV volumes in all patients postoperatively, regardless of the magnitude or direction of shunting preoperatively.

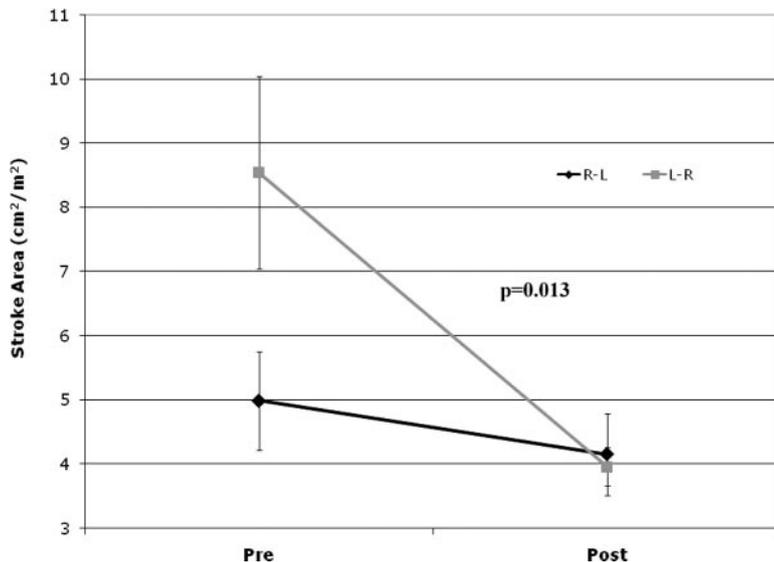


Figure 3. Graph representing preoperative (Pre) and postoperative (Post) SA in patients with R-L shunting (black line) vs L-R shunting (gray line).

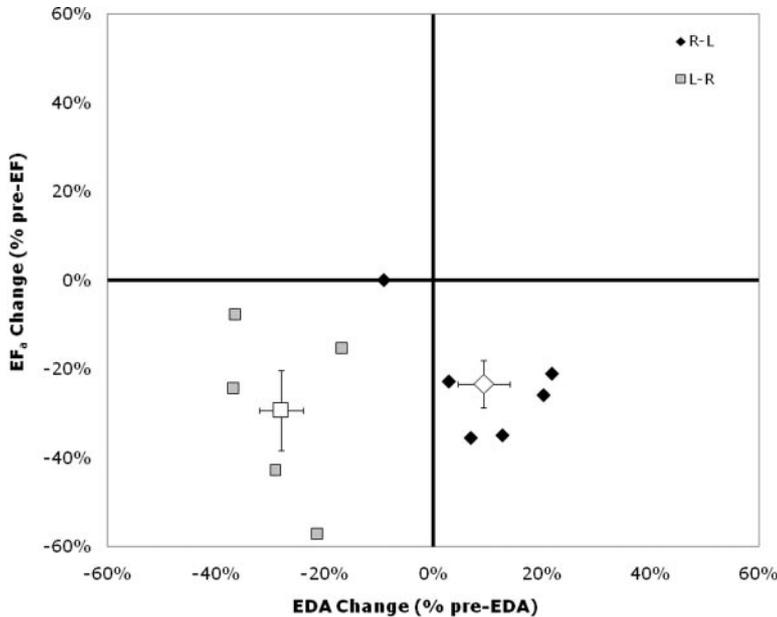


Figure 4. Changes in postoperative LV EDA and ejection fraction (EF_a) expressed as a percentage of preoperative values. Patients with R-L shunting (\blacklozenge) fall into the lower-right quadrant, whereas those with L-R shunting (\square) fall into the lower-left quadrant. Group means are shown by the open symbols. Error bars indicate group SE. A simultaneous decrease in EF_a and EDA is consistent with a primary effect of reduced preload, but a decrease in EF_a while EDA increases implies decreased contractile function or a large increase in afterload.

Our group has previously reported the functional effects of repair of congenital heart lesions.^{7,10,19} A relation similar to Figure 4 was derived between EDA and ejection fraction after repair of atrial septal defects or VSDs.¹⁰ All patient data fit a linear correlation model. Atrial septal defect patients fell into the upper-right quadrant of the graph, whereas VSD patients fell into the lower-left quadrant. This reflects increased volume loading of the LV after atrial septal defect repair and decreased volume loading after VSD repair. The clinical implication is that volume loading is appropriate for treatment of low-output states after VSD closure, whereas inotropic agents may be preferable after atrial septal defect closure.

Figure 4 localizes L-R shunting pink TOF patients to the same quadrant as VSD patients studied previously.¹⁰ These patients experience decreased LV preload and decreased LV systolic function. LV function would be expected to improve substantially if volume loading were implemented. TOF patients with R-L shunting and cyanosis (blue TOF) function in the lower-right quadrant of the graph, with increased EDA and decreased ejection fraction. This combination implies either a large increase in afterload or systolic dysfunction related to a septal patch and/or myocardial injury. This pattern favors administration of inotropic agents for LV support in these patients.

Long-term consequences of our observations remain to be defined. For previous study populations, Abd El Rahman et al²⁰ showed decreased LV systolic function in almost 25% of patients 10 years after TOF repair. The same group described decreased LV systolic function and paradoxical interventricular septal motion in asymptomatic patients 11 years after TOF repair.⁵ Studies correlating acute postoperative LV dysfunction with long-term impairment could clarify the mechanisms of long-term LV systolic dysfunction.

Regarding design weaknesses of the present study, variations in patient anatomy, surgical technique, and clinical factors are potential confounding factors. Although we could not control all of these variables, the trends in the present data

are consistent and should be reproducible in a larger study. The validity of dividing patients on the basis of the direction of shunting across the VSD can be challenged on the basis of a high frequency of bidirectional shunting in TOF.¹³ The predominance of R-L or L-R shunting is clinically apparent in most cases, however, and reflects the degree of RV outflow obstruction and cyanosis. We confirmed the validity of the present R-L shunting group using preoperative hemoglobin as an index of chronic cyanosis. Polycythemia is an indicator of chronic cyanosis in addition to pulse oximetry, which can be variable and sometimes unreliable. Smaller pulmonary valve diameter in the R-L shunting group also implies more RV outflow obstruction and more R-L shunting across the VSD. Furthermore, the larger LV EDA in the L-R shunting group is evidence for LV volume overload. All these data suggest that the 2 groups, as defined in the present study, are well differentiated with differing shunt hemodynamics.

Effects of surgical repair techniques were reviewed. Most (8 of 11) patients did not require an RV infundibular or transannular patch. LV function in 3 patients with patches was not qualitatively different from that in patients without patches. Although valve-sparing procedures were associated with residual pulmonary valvar stenosis (13 to 46 mm Hg), we found no correlation between residual RV outflow obstruction and changes in LV systolic or diastolic function (data not shown). Although a postoperative atrial communication may influence postoperative ventricular mechanics in the TOF patient, this lesion was not observed in our patients. Additional studies using different methodologies are indicated for definition of LV mechanics in the short, intermediate, and long term.

Conclusions

The present study of LV mechanics in the modern surgical era revealed LV systolic and diastolic dysfunction in all patients after TOF repair. Specifically, LV stroke area, stroke work, and ejection fraction decreased, as did LV contractility (as measured by preload recruitable stroke work). Diastolic

pressure-area curves revealed a postoperative decrease in LV compliance, as exemplified by a decrease in LV EDA at a common preoperative and postoperative LV EDP. Comparison of R-L (blue TOF) and L-R (pink TOF) preoperative ventricular shunts revealed L-R shunting patients behaving similar to those with simple VSD repair (ie, decreases in LV EDA and SA), whereas R-L shunting patients showed less ventricular eccentricity, smaller changes in SA, and an increase in LV EDA. These differences imply that volume loading may be preferable in pink TOF patients and inotropic agents in the postoperative management of blue TOF patients. Postoperative LV dysfunction may reflect effects of a septal patch or deficiencies of current myocardial protection strategies. Further studies elucidating the cause of LV dysfunction and its short- and long-term consequences are needed to optimize treatment of patients during and after TOF repair.

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Disclosures

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CLINICAL PERSPECTIVE

Tetralogy of Fallot is a spectrum of disease, from patients with severe pulmonary blood flow restriction to patients with simple ventricular septal defect physiology and pulmonary overcirculation. Given its anatomic features, tetralogy of Fallot is often thought of as a disease of the right side of the heart alone, with the assumption of a normal left heart. However, this study of intraoperative left ventricular (LV) mechanics demonstrated depression of LV systolic and diastolic function in all patients after tetralogy of Fallot repair. Mechanisms of this depressed function may involve imperfect myocardial protection and/or mechanical factors related to the ventricular septal defect patch. The present study also demonstrated that effects of surgery on ventricular geometry and loading varied with the direction of blood flow through the ventricular septal defect. LV preload from patients with predominantly right-to-left shunts increased after repair, but preload decreased in patients with simple ventricular septal defect physiology. These differences have clinical implications for the treatment of low cardiac output in the postoperative period. The unloaded LV produced by closure of left-to-right shunts should respond favorably to volume administration, but closure of right-to-left shunts increases LV preload, which suggests inotropes as the intervention of choice. This study looked only at the immediate effects of tetralogy of Fallot repair on the LV. Further studies are needed to define the relevance of these changes to long-term outcomes.