

# Right Ventricle to Pulmonary Artery Conduit Augmentation Compared With Replacement in Young Children

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**Background.** Targeted outcome data for young children undergoing right ventricle to pulmonary artery conduit reoperation are sparse, as are data on the use of conduit augmentation as an alternative to conduit replacement at the time of first conduit reoperation (conduit 2).

**Methods.** We conducted a retrospective chart review including baseline data, operative data, and cross-sectional follow-up on children younger than 10 years of age undergoing a first conduit reoperation (n = 180), comparing conduit replacement (n = 147, 82%) with conduit augmentation (n = 33, 18%).

**Results.** There were no differences between the two groups with respect to age, size, or hemodynamic variables. Augmentation was less often performed in patients with an aortic homograft and by one surgeon. At conduit 2, cardiopulmonary bypass time was longer in replacement patients ( $101 \pm 35$  versus  $71 \pm 34$  minutes;  $p < 0.001$ ); cardiac intensive care unit stay was not

different. Early mortality was 0.5%, and overall 10-year survival was 95%. Freedom from reoperation was 80% at 5 years and 39% at 10 years, whereas freedom from reintervention (reoperation or catheter intervention) was 55% at 5 years and 26% at 10 years, with no differences between groups. Aortic homograft as a first conduit was associated with shorter freedom from reoperation. Limiting analysis to conduits that were replaced at conduit 2, undersized conduits were associated with shorter freedom from reoperation and smaller body surface area, and undersized conduits were associated with shorter freedom from reintervention.

**Conclusions.** Freedom from a second conduit reoperation after a first conduit replacement was shorter in smaller children and undersized conduits. Conduit augmentation offers similar clinical outcomes in selected patients.

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Children with many forms of congenital heart disease undergo biventricular repair with a right ventricle to pulmonary artery (RV-PA) conduit. However, evidence-based decisions about conduit reintervention are difficult to formulate due to the nebulous outcome literature and long-term nature of many important outcomes. Children undergoing biventricular repair with RV-PA conduits present unique problems related to optimizing for small, but growing, chest size, conduit-branch PA size mismatch, and RV incision size. In children, reintervention on dysfunctional conduits is primarily aimed at relieving conduit stenosis, because the consequences of conduit regurgitation are often well tolerated until young adulthood. Freedom from conduit reoperation in mixed cohorts can range anywhere from 60% to 90% at 5 years and 29% to 72% at 10 years [1–6]. Therefore, young infants receiving conduits for initial palliation may require multiple reoperations. Factors previously identified to predict conduit longevity include patient age, primary anatomic diagnosis, type of conduit, conduit size, conduit diameter Z-score (standard deviations

above or below average pulmonary valve size relative to body surface area), distal PA stenosis, and higher RV systolic pressure [1–10]. In certain settings, the cost of multiple reoperations and conduit availability are prohibitive. In examining the literature, most reports on conduit longevity focus only on infants or combine primary operations with reoperation and include patients of various ages and with various conduit types. With so many variables, it is difficult to assess the effect of different management strategies on outcomes. Studies that stratify analysis by patient age and reoperative status may help minimize the confounding effects of these variables and help inform decisions on specific populations.

Patch augmentation of RV-PA conduits is an alternative to complete conduit replacement for the treatment of conduit obstruction. Using readily available autologous, xenograft, or synthetic materials, patches can be crafted to relieve conduit obstruction with the possibility of a more straightforward operative and postoperative course, albeit

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at the cost of conduit regurgitation. The longevity of augmented conduits relative to replaced conduits is not well described. In this study of young children undergoing a first conduit reoperation (ie, a second conduit operation, conduit 2), we hypothesized that although operative and perioperative outcomes such as duration of cardiopulmonary bypass and intensive care unit stay would be shorter for patients undergoing conduit augmentation at the time of conduit 2, compared with those undergoing conduit replacement, freedom from further reoperation (conduit 3) or reintervention after conduit augmentation would be shorter. The primary hypothesis was that freedom from conduit 3 was significantly shorter after conduit augmentation compared with conduit replacement.

## Material and Methods

The Children's Hospital Boston Cardiovascular Program database was queried for patients 10 years of age or younger who underwent replacement or augmentation (ie, conduit 2) of the RV-PA conduit that was placed during the initial complete repair. We limited the study to patients 10 years of age or younger to focus on patients who were generally unlikely to accommodate placement of an adult-sized conduit. Patients in whom the first conduit was placed without complete repair (eg, ventricular septal defect left open in patients with tetralogy of Fallot and pulmonary atresia) were excluded. Baseline demographic, anatomic, and hemodynamic data before conduit 2 as well as operative details of conduit 2 were collected. Cross-sectional follow-up was obtained, and data on survival, transplant, conduit reoperation, conduit dilation or stenting, and hemodynamics were recorded. This retrospective chart review was approved by departmental and institutional review boards, and a waiver of patient consent was granted.

## Surgical Procedure

The conduit augmentation procedure generally consisted of a longitudinal incision along the entire length of the anterior portion of the RV-PA conduit across both RV and PA anastomoses, although there was variation in the extent of the incision. The homograft valve was often excised. An onlay patch was crafted from expanded polytetrafluoroethylene, polyethylene terephthalate fiber (Dacron), or homograft for each patient and sewn into this incision, thereby increasing the diameter. The size of the patch was not standardized.

## Data Analysis

The primary outcome was freedom from conduit 3 after conduit 2. Secondary outcomes included survival and a composite outcome of freedom from conduit 3 or conduit dilation (freedom from conduit reintervention) after conduit 2. We also analyzed demographic, anatomic, and procedural factors including age at original conduit operation, original conduit type, original conduit size, original conduit Z-score (based on previously published normative echocardiographic data [11]), diagnosis, type of repair at conduit 2 (replacement versus augmentation), surgeon, bypass time, cardiac intensive care unit stay, age at conduit 2, body surface area at conduit 2, and size of replacement at conduit 2. Baseline variables were compared using Student's *t* test or  $\chi^2$  analysis as appropriate. Primary and secondary outcomes were analyzed using Kaplan-Meier analysis and multivariable Cox regression analysis. Multivariate models were constructed using independent variables with significance levels up to a probability value of 0.1 by univariate analysis, and significance in the final model was determined for probability values less than or equal to 0.05. Outcome analy-

Table 1. Baseline Variables Before Conduit 2

Variable	Augmentation (n = 33)	Replacement (n = 147)	<i>p</i> Value
Diagnosis			
Tetralogy of Fallot (n = 71)	10	61	0.16
Truncus arteriosus (n = 68)	14	54	0.34
TGA or double-outlet RV (n = 33)	7	26	0.40
Left-sided obstruction, Ross procedure (n = 8)	2	6	0.45
Original conduit type			
Aortic homograft	9 (27%)	94 (64%)	<0.001
Pulmonary homograft	19 (58%)	34 (23%)	<0.001
Synthetic conduit	4 (12%)	13 (9%)	0.38
Unknown homograft	1 (3%)	6 (24%)	0.62
Original nominal conduit size (mm) <sup>a</sup>	10.2 ± 2.2	11.1 ± 2.9	0.046
Prior conduit dilation	6 (18%)	30 (20%)	0.52
Conduit MIG by echocardiography (mm Hg) <sup>a</sup>	66 ± 23	58 ± 23	0.10
Conduit PSEG by catheterization (mm Hg) <sup>a</sup>	54 ± 19	52 ± 21	0.66
Conduit aneurysm	2 (6%)	11 (7%)	0.56
Closure of residual VSD at conduit 2	1 (3%)	13 (9%)	0.23

<sup>a</sup> Data are reported as mean ± standard deviation.

MIG = maximum instantaneous Doppler gradient; great arteries; VSD = ventricular septal defect.

PSEG = peak systolic ejection gradient;

RV = right ventricle;

TGA = transposition of the

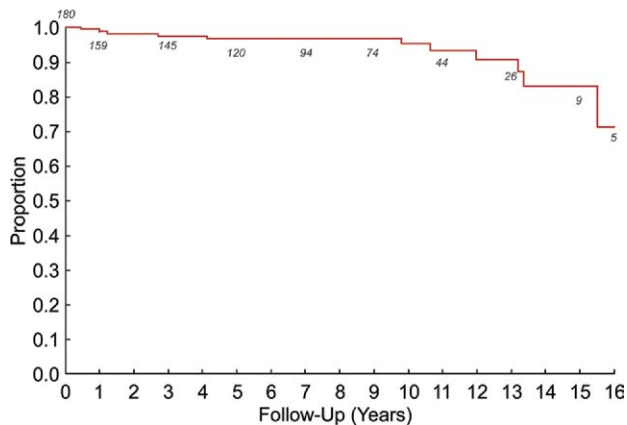


Fig 1. Overall survival after conduit 2.

ses were also performed with adjustment for age and surgeon at conduit 2. Data are presented as mean  $\pm$  standard deviation, median with interquartile range, or frequency (percent of total).

## Results

### Patients

A total of 180 children 10 years of age or younger underwent conduit 2 after previous complete repair, with conduit replacement in 147 (82%) and conduit augmentation in 33 (18%). Baseline variables are compared in Table 1. Conduit augmentation was less likely to be performed when the original conduit was an aortic homograft compared with other conduits (Table 1). At the time of conduit 2, there were no differences in the severity of conduit obstruction between patients whose original conduit was a pulmonic homograft ( $61 \pm 26$  versus  $62 \pm 20$  mm Hg maximum instantaneous gradient by echocardiography;  $p = 0.82$ ) and those with other

conduits. At the time of conduit 2, there were no differences between patients who underwent replacement and those who underwent augmentation with respect to age, body surface area, echocardiogram-derived maximum instantaneous gradient across the conduit, catheterization-derived conduit peak systolic ejection gradient, or proportion of patients who underwent conduit dilation or stenting before conduit 2.

### Conduit 2 Indications and Procedures

Overall, conduit stenosis was an indication for conduit 2 in 179 of the 180 procedures (99%). The patient without conduit stenosis was 1 of 13 patients (7%) who had a conduit pseudoaneurysm; 11 of these 13 underwent conduit replacement and 2 underwent augmentation. Additional indications for conduit 2 in patients with conduit obstruction included subacute bacterial endocarditis attributable to an infected conduit in 1, an echocardiographic mass in the RV outflow tract that was discovered intraoperatively to be an RV muscle bundle in 1, and 1 patient in whom the origin of the right PA was covered by a stent. Residual ventricular septal defects were present in 14 patients at the time of conduit 2; 13 (9%) underwent conduit replacement and 1 (3%) underwent conduit augmentation (not significantly different). There was no difference in age at conduit 2. Original conduits were slightly larger in the replacement group ( $11.1 \pm 2.9$  mm) compared with the augmentation group ( $10.2 \pm 2.2$  mm;  $p = 0.046$ ).

Among patients who underwent conduit replacement at conduit 2, the conduit was an aortic homograft in 89, a pulmonary homograft in 42, a nonvalved synthetic tube graft in 13, and a valved synthetic graft in 3. The mean nominal conduit diameter was  $17.9 \pm 2.9$  mm, and the mean conduit Z-score was  $1.9 \pm 1.3$ . Among patients undergoing conduit augmentation at conduit 2, patch material included expanded polytetrafluoroethylene in 17, Dacron in 9, and homograft tissue in 7. Patch size and configuration, as well as procedures to tailor the PA

Table 2. Conduit Reintervention After Conduit 2

Variable	Augmentation (n = 33)	Replacement (n = 147)	p Value
Conduit reoperation (no. of pts)	8	66	
Indication for conduit reoperation <sup>a</sup>			
Stenosis	7 (88%)	61 (92%)	0.07
Aneurysm	2 (25%)	6 (9%)	0.23
Regurgitation	1 (13%)	5 (8%)	1
Systemic ventricle outflow obstruction	1 (13%)	5 (8%)	1
Closure of residual ventricular septal defect	0 (0%)	5 (8%)	1
Tricuspid valve repair	0 (0%)	3 (4%)	1
Conduit gradient before reoperation (mm Hg) <sup>b</sup>	60 $\pm$ 20	57 $\pm$ 23	0.25
Conduit dilation before reoperation (no. of pts)	6 (75%)	46 (70%)	0.78
Conduit dilation or stenting (no. of pts)	14	79	
Multiple conduit dilations/stenting procedures <sup>c</sup>	4 (12%)	27 (18%)	0.8
Peak conduit gradient before first dilation (mm Hg) <sup>b</sup>	59 $\pm$ 19	52 $\pm$ 22	0.26

<sup>a</sup> There may have been more than one indication per patient. without subsequent conduit reoperation.

<sup>b</sup> Data are reported as mean  $\pm$  standard deviation.

<sup>c</sup> Multiple procedures before or

bifurcation, depended on the surgeon and specific case, and the completed diameter of the augmented conduit was not routinely measured. Among the 5 surgeons responsible for these 180 procedures, one performed augmentation in 3% of conduit 2 procedures ( $p = 0.04$ ), another performed augmentation in 40% of conduit 2 procedures ( $p = 0.02$ ), and the other 3 surgeons performed conduit augmentation in about 20% of their respective cases.

### Perioperative Outcomes

All conduit 2 patients underwent surgery with cardiopulmonary bypass and recovered in the cardiac intensive care unit. Total pump time was significantly longer in patients who underwent conduit replacement compared with augmentation ( $101 \pm 35$  versus  $71 \pm 34$  minutes;  $p < 0.001$ ). There was no difference in length of cardiac intensive care unit stay between patients who underwent replacement ( $2.3 \pm 3.6$  days) and those who underwent augmentation ( $1.8 \pm 0.8$  days;  $p = 0.23$ ). There was 1 early death ( $\leq 30$  days) at conduit 2 in the replacement group (not significantly different).

### Follow-Up

Follow-up was available on 161 of the total 180 conduit 2 patients, 129 in the conduit replacement group and 32 who underwent augmentation. Median follow-up duration was 7.3 years (interquartile range, 3.9 to 10.9) with no difference between the two groups. Including the early death, 11 patients died and 1 underwent heart transplant during follow-up, all of whom were in the replacement group. Overall survival by Kaplan-Meier analysis was 99% at 1 year, 96% at 5 years, and 95% at 10 years, as presented in Figure 1.

### Conduit Reoperation After Conduit 2

During follow-up after conduit 2, 74 patients underwent conduit 3, 66 in the replacement group and 8 in the augmentation group. Reoperations were performed primarily for conduit stenosis, and overall no differences were found between replacement and augmentation

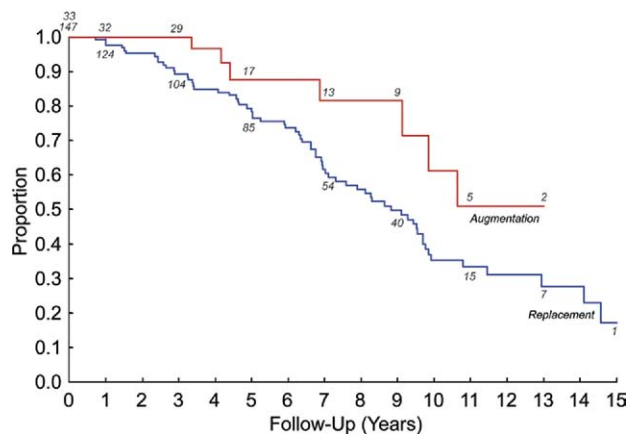


Fig 2. Freedom from conduit reoperation after conduit 2 stratified by conduit augmentation versus conduit replacement.

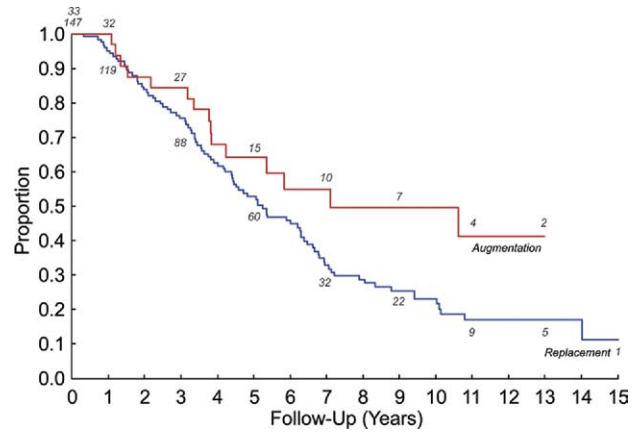


Fig 3. Freedom from conduit reintervention after conduit 2 stratified by conduit augmentation versus conduit replacement.

groups in indications for conduit 3 (Table 2). In the replacement group, 5 patients underwent closure of a residual ventricular septal defect, and 3 had tricuspid valve repair whereas none in the augmentation group underwent additional procedures.

By Kaplan-Meier analysis, overall freedom from conduit 3 after conduit 2 was 99% at 1 year, 80% at 5 years, and 39% at 10 years. As depicted in Figure 2, there was a trend toward shorter freedom from conduit reoperation in the replacement group compared with the augmentation group (log rank  $p = 0.07$ ). On multivariable Cox regression analysis, the use of an aortic homograft as the original conduit was associated with shorter freedom from reoperation after conduit 2 ( $\beta = 0.57$ ; 95% confidence interval, 0.35 to 0.92;  $p = 0.04$ ). No other independent variable analyzed was associated with freedom from conduit 3. This result was unaffected by controlling for surgeon.

Limiting analysis to the conduit replacement group and controlling for surgeon, factors associated with shorter freedom from conduit 3 by univariate Cox regression included smaller conduit size and Z-score, and an aortic homograft as the original conduit. On multivariate analysis, conduit Z-score was independently associated with shorter freedom from conduit 3 ( $\beta = 0.76$ ; 95% confidence interval, 0.64 to 0.90;  $p = 0.001$ ).

### Transcatheter Conduit Procedures After Conduit 2

Transcatheter dilation or stenting of an obstructed RV-PA conduit was performed in 85 patients after conduit 2, 71 in the replacement group and 14 in the augmentation group. Fifty-two of these patients subsequently underwent conduit 3. At the time of the first transcatheter procedure after conduit 2, the peak systolic gradient across the conduit was  $54 \pm 20$  mm Hg, with no difference between conduit replacement and augmentation groups (Table 2). Overall, freedom from any conduit reintervention (conduit 3 or conduit dilation or stenting) after conduit 2 was 98% at 1 year, 55% at 5 years, and 26% at 10 years. There was no difference in freedom from conduit reintervention between the replacement and

Table 3. Hemodynamic Outcomes After Conduit 2

Variable	Augmentation	Replacement	p Value
Reoperation (no. of patients)	8	66	
Conduit MIG before reoperation (mm Hg) <sup>a</sup>	60 ± 21	55 ± 27	0.69
Conduit PSEG before reoperation (mm Hg) <sup>a</sup>	59 ± 17	55 ± 22	0.65
No reoperation (no. of patients)	25	81	
Follow-up in years (median, IQR)	5.3 (3.8–7.5)	5.4 (1.5–9.2)	0.92
Conduit MIG at follow-up (mm Hg) <sup>a</sup>	42 ± 22	30 ± 24	0.045
Conduit PSEG at follow-up (mm Hg) <sup>a</sup>	46 ± 21	33 ± 27	0.10

<sup>a</sup> Data are reported as mean ± standard deviation.

IQR = interquartile range; MIG = maximum instantaneous Doppler gradient; PSEG = peak systolic ejection gradient.

augmentation groups (log rank test  $p = 0.09$ ; Fig 3). The use of a pulmonary homograft at the original conduit operation ( $\beta = 1.6$ ; 95% confidence interval, 1.1 to 2.4;  $p = 0.04$ ) was associated with longer freedom from reintervention on multivariate analysis. When conduit replacement patients were considered separately, smaller conduit Z-score ( $\beta = 0.79$ ; 95% confidence interval, 0.71 to 0.88;  $p < 0.001$ ) and smaller body surface area at the time of conduit 2 ( $\beta = 0.27$ ; 95% confidence interval, 0.09 to 0.82;  $p = 0.02$ ) were associated with decreased freedom from conduit reintervention on multivariable analysis. These analyses were not affected by controlling for surgeon.

#### Hemodynamic Status

Examining hemodynamic measures of conduit obstruction after conduit 2, there were no clinically important differences between the replacement and augmentation groups, either among patients who underwent conduit 3 or those who did not (Table 3). The follow-up duration of patients who were free from conduit 3 at most recent follow-up did not differ between replacement and augmentation groups (Table 3).

#### Comment

Estimating outcomes of conduit surgery in children and older patients is complicated by the fact that studies of conduit reintervention and durability are often either focused on newborns and infants, or more broadly heterogeneous. To better anticipate outcomes of conduit reoperation in young children, and thereby provide more informative patient counseling, we concentrated this study on children 10 years of age or younger, in whom substantial somatic growth is anticipated, undergoing their first conduit reoperation. By limiting the study to second conduit operations (ie, first conduit reoperation or conduit 2), we hoped to avoid the confounding effects of patients who underwent multiple conduit operations at a young age.

#### Reintervention After Right Ventricle to Pulmonary Artery Conduit Operations

Freedom from conduit reoperation after conduit 2, called conduit 3, was 80% at 5 years and 39% at 10 years. At our

institution, transcatheter conduit dilation and/or stenting is often performed in an effort to relieve RV-PA conduit obstruction and to minimize RV work before referral for conduit reoperation. In this series, almost two thirds of patients who underwent conduit 3 had a prior transcatheter intervention. Including angioplasty and stenting of the conduit, freedom from conduit reintervention after conduit 2 was 55% at 5 years and 26% at 10 years. The only predictor of longer freedom from conduit reoperation or reintervention after conduit 2 was use of a pulmonic homograft at the original conduit placement. Among patients in this series who underwent conduit replacement at conduit 2, smaller body surface area and smaller conduit Z-score were associated with shorter freedom from subsequent reoperation and reintervention. Primary diagnosis and other previously demonstrated variables were not associated with freedom from reintervention.

Studies of broader cohorts undergoing primary conduit placement or reoperation report overall freedom from reoperation ranging from 60% to 90% at 5 years [1–6]. Reported risk factors for reoperation included smaller conduit diameter, conduit type, younger age, and diagnosis of transposition of the great arteries, depending on the series. Specific studies focusing on freedom from reoperation after conduit replacement found that younger age, smaller conduit, use of homograft, and diagnosis of truncus arteriosus were risk factors for a repeat conduit replacement after a first conduit replacement [1, 2, 4–10]. In our cohort, we found that use of a pulmonic homograft as the original conduit was associated with conduit reoperation and reintervention. Although it is not entirely clear why original conduit type should affect second conduit outcomes, conduit allosensitization may be considered. Some authors have described accelerated fibrocalcification in aortic homografts, and other authors have ascribed these changes to an immune-mediated phenomenon based on circumstantial data such as immunologic (eg, blood group or HLA type) mismatches between graft and patient [12, 13]. Our data are unable to address the possible role of allosensitization.

Although it is intuitive that smaller conduits would be associated with shorter freedom from reoperation, conduit size is a complex variable. Many patients needing RV-PA conduits have PA anomalies predisposing them

to multiple stenoses. Optimizing a conduit to minimize mismatch between the conduit and patient or PA size may theoretically help reduce total pulmonary arterial impedance and RV work. Karamlou and coworkers [14] and Askovich and associates [15] found that a conduit size more than 2.5 standard deviations above the mean normal pulmonary annulus diameter (Z-score >2.5) was associated with diminished freedom from reoperation. In patients with tetralogy of Fallot or truncus arteriosus, Wells and colleagues [16] found that valvar area thickening and shrinkage led to conduit stenosis in 53% of patients, whereas somatic outgrowth was responsible in only 8%. McMullan and associates [17] showed that surgically restricting oversized conduits in an effort to optimize matching of the conduit to PA size in patients with truncus arteriosus was associated with freedom from significant conduit stenosis or regurgitation. Therefore, conclusions from our data on young children that bigger is better should not be drawn without additional investigation.

#### *Survival After Right Ventricle to Pulmonary Artery Conduit Operations*

In our series, there was 1 early death (0.5%) and 95% survival at 10 years by Kaplan-Meier analysis. Although all 11 of the deaths in this series aggregated to the replacement group, we believe that this finding was stochastic, given that nearly all were far removed from the conduit 2 surgery and that most patients had undergone a conduit reoperation after conduit 2. Single-center series on mixed cohorts undergoing primary and repeat RV-PA conduits have reported early postoperative mortality of 4% to 10% and late survival ranging from 66% to 94% at 10 years [1–10]. Early mortality is substantially lower after conduit replacement than after primary conduit placement, with estimates ranging from 0.9% to 1.7%. Multivariate analyses, excluding era effects, have identified risk factors for death to include male sex, older age at repair, RV hypertension after the repair, and certain diagnoses including truncus arteriosus, transposition of the great arteries, and congenitally corrected transposition.

#### *Conduit Augmentation*

Of the 180 children in this series, 18% underwent conduit augmentation at conduit 2. Augmentation was less likely to be performed in patients with an aortic homograft and more likely in pulmonic homografts. As anticipated, even when patients undergoing ventricular septal defect closure concomitant with conduit 2 (almost all of whom had conduit replacement) were excluded, the duration of cardiopulmonary bypass was more than 30 minutes shorter in the augmentation group on average. Intensive care unit stay was not different between groups. In contrast to our hypothesis, however, freedom from reoperation and from any conduit reintervention after conduit 2 were not significantly different between patients who underwent augmentation and those who underwent conduit replacement. Although patients with conduit augmentation were almost invariably left with conduit regurgitation after conduit 2, almost all of the patients

undergoing subsequent reintervention in both groups had RV-PA obstruction, and did not undergo reintervention for regurgitation. Moreover, the degree of conduit obstruction was comparable in the two groups both in patients who did and did not undergo reintervention.

We found only one study that looked at the role of conduit augmentation for obstructed RV-PA conduits. Our findings differ somewhat from those of Mohammadi and colleagues [18], who observed that patch augmentation in a cohort with a wider age range was associated with higher rates of conduit dysfunction leading to reoperation. This difference may be related to the relatively narrow age range, the need for another conduit surgery in relatively short succession, or the higher proportion of pulmonary homografts in the augmentation group of our cohort. Pulmonary homografts have been associated with increased longevity and absence of accelerated fibrocalcification. As such, supple pulmonary homografts may be more amenable to patch augmentation than other types of conduits. Patch augmentation may offer a useful and perhaps more cost-effective alternative to conduit replacement for conduit reoperation in selected young children, especially in resource-poor settings.

#### *Limitations*

Retrospective data collection and incomplete follow-up after conduit 2 in 19 of 180 patients are important limitations of the study. The inability to ascertain reintervention in these patients and their preponderance in the conduit replacement group risks underestimating freedom from reoperation or reintervention after conduit replacement. Another limitation is possible selection bias in the use of conduit augmentation. The bias in selection of patients with aortic homografts for replacement rather than augmentation was almost certainly based on technical considerations, as discussed above. Furthermore, a number of surgeons performed the procedures examined in this report, and idiosyncratic preferences for one procedure or technical differences may have biased the findings. In an effort to minimize this bias, we adjusted for surgeon and patient age in our outcome analyses. We were also unable to systematically assess the degree or incidence of fibrocalcification in explanted or augmented homografts. Although these limitations may make it difficult to generalize our findings, the data support the conclusion that acute and intermediate outcomes do not differ dramatically between children undergoing conduit augmentation and replacement of an obstructed first RV-PA conduit.

#### *Conclusions*

Freedom from a third RV-PA conduit operation after second conduit operation in young children was 80% at 5 years and 39% at 10 years in this series. Smaller body surface area and undersized conduits were independently associated with shorter freedom from reoperation and any reintervention. Augmentation of the original RV-PA conduit at the time of reoperation for conduit obstruction may be a reasonable strategy in certain patients, and is associated with shorter cardiopulmonary bypass time, similar postoperative course, and similar freedom from reoperation and

reintervention. Further study will be necessary to determine characteristics of patients more likely to benefit from augmentation or replacement.

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## INVITED COMMENTARY

Valved conduits are commonly used in neonates and small infants for reconstruction of the outflow tract of the right ventricle. These conduits frequently require reoperation in early childhood before the patient is large enough to receive an adult-sized conduit. Generally, a new larger conduit is used for replacement when significant conduit dysfunction (almost always stenosis) develops.

Zachariah and colleagues [1] from Boston Children's Hospital have taken another approach and simply augmented the prior conduit with an additional patch, thus relieving the stenosis, albeit at the cost of pulmonary insufficiency. This retrospective analysis focused on patients aged younger than 10 years who were therefore unlikely to be large enough to accommodate an adult-sized conduit. The authors compared the outcome of 33 patients undergoing conduit augmentation with 147 patients undergoing conduit replacement and found that short-term outcomes were comparable and freedom from conduit reoperation and reintervention were similar. In fact, there was a trend toward longer freedom from conduit reoperation among patients who underwent augmentation.

Although we do not know precisely how these 33 patients were selected, the data suggest that for a sub-

group of patients, simple augmentation of the previously placed conduit is a real alternative to conduit replacement. In addition to cost-savings, conduit augmentation is simple to perform and may decrease allosensitization if a new homograft conduit is avoided. As Emerson once pointed out, "a foolish consistency is the hobgoblin of small minds. . . ." The authors are to be congratulated for seeing the "foolish consistency" of routine conduit replacement and identifying an alternative.

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