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ABSTRACT

Experience with Polystan valved conduits in children with congenital heart disease was reviewed. From May 1997 to October 2000, 52 Polystan valved conduits were used for reconstruction of the pulmonary ventricular outflow tract in 50 patients. The median age was 24 months (range, 7 days to 19 years), body weight was 11 kg (range, 2.8 to 52 kg), and conduit size at operation was 19 mm (range, 12 to 24 mm). Early mortality was 12% (6/50). Late mortality was 6% (3/50). The median follow-up of survivors was 25 months (range, 2 to 43 months). Three patients underwent conduit replacement; 2 received larger conduits in a second-stage operation for ventricular septal defect closure. There was no death at reoperation. Polystan valved conduits can be used for reconstruction of the pulmonary ventricular outflow tract in congenital heart disease, with no significant conduit-related problems.

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INTRODUCTION

Extracardiac valved conduits have been used to reconstruct pulmonary ventricular outflow tracts in congenital heart disease. Xenografts and allografts are currently used, but allografts are considered the better conduit.[1] However, allografts have limited availability, especially in young children. In addition, young patients have been noted to develop accelerated allograft stenosis relative to older age groups.[2] Therefore, xenografts are commonly used in many centers. Carpentier-Edwards porcine conduits and cryopreserved allografts were mainly used in our hospital in the past. Recently, we have used Polystan conduits (Polystan, Vaerløse, Denmark) to correct congenital malformations. This report describes the early and intermediate results of Polystan conduits in the reconstruction of pulmonary ventricle-to-pulmonary artery continuity. The term "pulmonary ventricle" is used rather than "right ventricle" because some patients had corrected transposition or situs inversus.

PATIENTS AND METHODS

From May 1997 to October 2000, 52 Polystan conduits were used for reconstruction of the pulmonary ventricular outflow tract in 50 patients. The median age at operation was 24 months (range, 7 days to 19 years), and median body weight was 11 kg (range, 2.8 to 52 kg). Diagnoses are listed in Table 1. Eight patients (16%) had undergone no previous operation, 69 procedures had been performed in 42 patients, including 22 conduit interpositions in 19 patients (Table 2). The types of conduits previously

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inserted are listed in Table 3. For reconstruction of the pulmonary ventricular tract, allografts were selected when available, especially for children with complex defects.

All procedures were performed using standard techniques such as hypothermic cardiopulmonary bypass, bicaval venous cannulation, and aortic cannulation with periods of low flow or hypothermic circulatory arrest. In cases of additional intracardiac repairs such as atrial or ventricular septal defect closure, infundibular muscle resection, or distal pulmonary artery reconstruction, the aorta was crossclamped and cold cardioplegic solution was infused into the aortic root. Polystan conduits were used in the reconstruction of pulmonary ventricular outflow tracts. Proximal anastomosis was supplemented in some patients with a hood of bovine pericardium or Gore-Tex vascular patch to make the conduit curvature smooth. There were no technical problems encountered in placing Polystan conduits. Only 8 patients underwent Polystan conduit placement without any concomitant procedures. Concomitant procedures performed in 42 patients are listed in Table 4.

Early mortality was defined as death occurring within 30 days of the operation. The follow-up status of the patients was determined by retrospective review of hospital records or by telephone interviews. One patient was lost to follow-up. Statistical analyses were performed using SPSS version 8.0 software (SPSS, Inc., Chicago, IL, USA). Median and range were calculated for descriptive variables. Actuarial data were analyzed using Kaplan-Meier formulae. Differences between groups were evaluated by a log-rank test.

RESULTS

The diameter of the conduits ranged from 12 mm to 24 mm; the median diameter was 19 mm. Conduits expanded proximally with bovine pericardium or a Gore-Tex vascular patch caused no problems in the

Table 1. **Need for 52 Valved Conduits in 50 Patients**

Diagnosis	No. of Conduits
Pulmonary atresia	24
Tetralogy of Fallot	5
Tetralogy of Fallot + pulmonary atresia	4
TGA + pulmonary stenosis	5
Double-outlet right ventricle	5
Truncus arteriosus	5
Corrected TGA + pulmonary stenosis	4

TGA = transposition of the great arteries.

Table 2. **Previous Operations (n = 68) in 42 Patients**

Operation	n
Blalock-Taussig shunt	32
Conduit interposition	22
Reconstruction en ventriculare	3
Central shunt	2
Potts shunt	1
Tetralogy of Fallot correction	4
RVOT widening with patch + pulmonary valvotomy	3
Pulmonary artery banding	1

RVOT = right ventricular outflow tract.

Table 3. **Type of Previous Conduit (n = 22)**

Conduit	n
Hand-made bovine pericardial conduit	6
Allograft	5
Gore-Tex vascular graft	4
Carpentier-Edwards valved conduit	3
Polystan conduit	2
Carbomedics valved conduit	1
Bjork-Shiley valved conduit	1

Table 4. **Concomitant Procedures (n = 66) in 42 Patients**

Procedure	n
Pulmonary arterioplasty	22
Ventricular septal defect closure	19
Atrial septal defect closure	7
MAPCA ligation + unifocalization	4
Double switch operation	3
Aortic valve replacement	3
Left ventricle to aorta baffling	3
Pacemaker insertion	2
Infundibulectomy	2
Aortic arch reconstruction	1

MAPCA = major aortopulmonary collateral arteries.

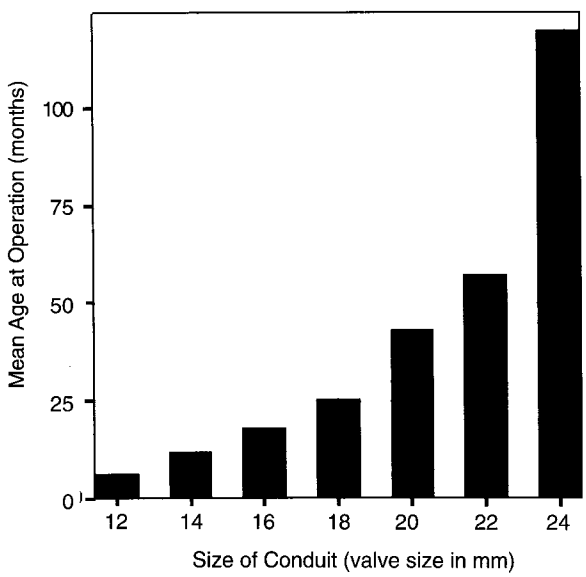

Figure 1. Mean age in relation to size of conduit.

Table 5. **Early Mortality**

Patient No.	Diagnosis	Age at Operation	Cause of Death	Comment
1	PA, VSD, ASD	10 months	RV failure	Severe peripheral pulmonary artery stenosis
2	Residual PS, PR after REV for TOF, PA, left pulmonary artery stenosis	5 years	Sudden RV failure	Conduit thrombosis detected
3	Severe PS (conduit calcified), AR after total correction of TOF + allograft interposition	10 years	Failure to wean from CPB	AVR with mechanical valve and angioplasty of pulmonary artery + ascending aorta
4	PA, VSD, MAPCA	5 months	Failure to wean from CPB	Severe peripheral pulmonary artery stenosis
5	PA/IVS, hypoplastic RV, membranous PA	1 year	Low cardiac output	One-and-a-half ventricle type repair
6	PA, VSD, MAPCA	5 years	Bleeding	Rhesus-negative AB blood type

AR = aortic regurgitation, ASD = atrial septal defect, AVR = aortic valve replacement, CPB = cardiopulmonary bypass, IVS = intact ventricular septum, MAPCA = major aortopulmonary collateral arteries, PA = pulmonary atresia, PR = pulmonary regurgitation, PS = pulmonary stenosis, REV = reconstruction en ventriculare, RV = right ventricle, TOF = tetralogy of Fallot, VSD = ventricular septal defect.

Table 6. **Late Mortality**

Patient No.	Diagnosis	Age at Operation	Cause of Death	Interval After Operation
1	Dysplastic pulmonary valve syndrome	8 days	Pneumonia	4 months
2	TA, IAA, VSD, ASD	7 days	Pneumonia, bronchitis	5 months
3	Gore-Tex conduit stenosis after PA + VSD correction	1 year	Viral pneumonia	10 months

ASD = atrial septal defect, IAA = interrupted aortic arch, PA = pulmonary atresia, TA = truncus arteriosus, VSD = ventricular septal defect.

Table 7. **Polystan Conduit Replacement**

Patient No.	Diagnosis	Age at Operation	Initial Polystan Conduit Size	Cause of Replacement	Interval to Replacement	Type of New Conduit
1	PA, VSD	6 years	18 mm	Pressure gradient 25 mm Hg	12 months	Polystan 20 mm
2	PA, VSD, MAPCA	22 months	12 mm	No pressure gradient	14 months	Polystan 20 mm
3	DORV, VSD, PS, PAPVR	6 years	22 mm	Bacterial endocarditis	15 days	Pulmonary allograft 20 mm

DORV = double-outlet right ventricle, MAPCA = major aortopulmonary collateral arteries, PA = pulmonary atresia, PAPVR = partial anomalous pulmonary venous return, PS = pulmonary stenosis, VSD = ventricular septal defect.

operative field and they were not considered to have caused any hemodynamic problems. The mean ages at operation in relation to conduit size are depicted in Figure 1. There were 6 early hospital deaths (12%). The causes of early mortality are listed in Table 5. Sudden cardiac failure manifested at 8 days postoperatively in patient no. 2; thrombosis was found in his conduit. One patient with rhesus-negative AB blood type died from hemorrhage. Three more patients died of pneumonia during follow-up (Table 6). The median follow-up of survivors was 25 months (range, 2 to 43 months).

Using Kaplan-Meier survival analysis, the actuarial survival rates including early deaths were 83.8%, 81.6%, and 81.6% at 6, 12, and 43 months, respectively (Figure 2). Survival in those who had a small-sized conduit was significantly lower at 43 months (Figure 3). There was a statistically significant difference in survival between patients with 12- to 16-mm conduits and those with 18- to 20-mm conduits ($p = 0.0168$) or 22- to 24-mm conduits ($p = 0.0286$). Age at operation, sex, body weight, and diagnosis had no statistically significant effect on patient survival. Significant postoperative complications were encountered in 16 patients: low cardiac output syndrome in 6, arrhythmia in 2, delayed sternal closure in 2, reexploration for bleeding in 2, mediastinitis in 2, prolonged ventilator support (more than 10 days) in 2, chylothorax in 2, bacterial endocarditis in 1, and seizure in 1.

Postoperative echocardiographic evaluation was scheduled in survivors. The pressure gradient across the Polystan valved conduit, pulmonary insufficiency, insufficiency of the atrioventricular valve of the pulmonary ventricle, and ventricular function were checked. Four patients had mild pulmonary stenosis and one had a transvalvular velocity

of 4 m·sec^{-1} on echocardiographic evaluation. Pulmonary regurgitation was found in 8 patients (16%); it was mild in 5 cases and moderate in 3. Mild tricuspid regurgitation was also found in those who had pulmonary stenosis or regurgitation; 3 patients had combined pulmonary stenosis and regurgitation. Catheterization data were obtained in 5 patients who showed moderate to severe stenosis of the peripheral pulmonary arteries or stenosis and insufficiency of the valved conduits on echocardiography. Three of them had moderate to severe pressure gradients between the main and peripheral pulmonary arteries, and these patients were potential candidates for reoperation. One with a pressure gradient of 25 mm Hg and 2 others with no gradient required reoperation during follow-up (Table 7). Two required repair of ventricular septal defects that were not closed at the first operation, and the Polystan valved conduits were replaced with larger sizes. The third patient manifested postoperative mediastinitis and bacterial endocarditis; the conduit was replaced with an allograft. There was no death or significant complication in these 3 patients.

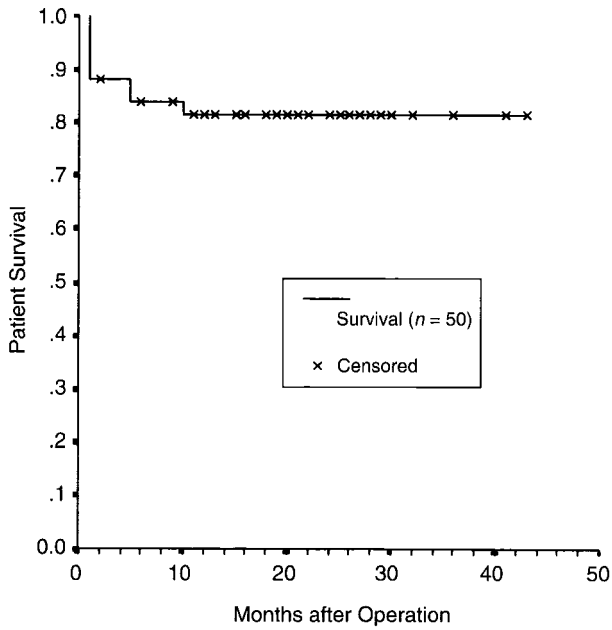

Figure 2. Patient survival after insertion of a Polystan valved conduit.

DISCUSSION

The use of extracardiac valved conduits has allowed correction of complex congenital heart lesions, but the ideal prosthesis is not yet available. The optimal characteristics of an extracardiac conduit are easy implantation, lack of degeneration or development of obstruction, growth ability, no insufficiency, and no need for anticoagulants.[3] Cryopreserved allografts have been considered the conduit of choice and good short-term results have been reported.[1,4,5] However, allografts are inferior to commercial xenografts in terms of availability and variety of sizes. The long-term results with allografts were comparable to those of xenografts in some reports.[6,7] The disadvantages of xenografts are operative bleeding, coronary compression by the valve ring, and inadequacy in neonates with thin and friable pulmonary arteries.[8] Inevitable long-term failure of xenografts is implicated with neointimal peel overgrowth, valve degeneration, calcification, and obstruction.[7]

The Polystan valved conduit is a jersey-type woven polyester tubular vascular prosthesis supplied with a tricuspid valve in the middle position. The tube is lined with porcine pericardium that has a thickness of 0.1 to 0.15 mm. The mesothelial side of the pericardium is on the luminal side of the tube. The 3 cusps are also made from porcine pericardium. The prosthesis is totally covered on the inner surface by pericardium. Conduits are available in 7 sizes with internal diameters of 12 to 24 mm in 2-mm increments. The Polystan valved conduit has some presumptive advantages over other xenografts: low bleeding risk, possible resistance to neointimal overgrowth owing to the pericardial lining of the inner surface, and no compression of the coronary arteries because there is no valve ring or stent. The possibility of reducing operative

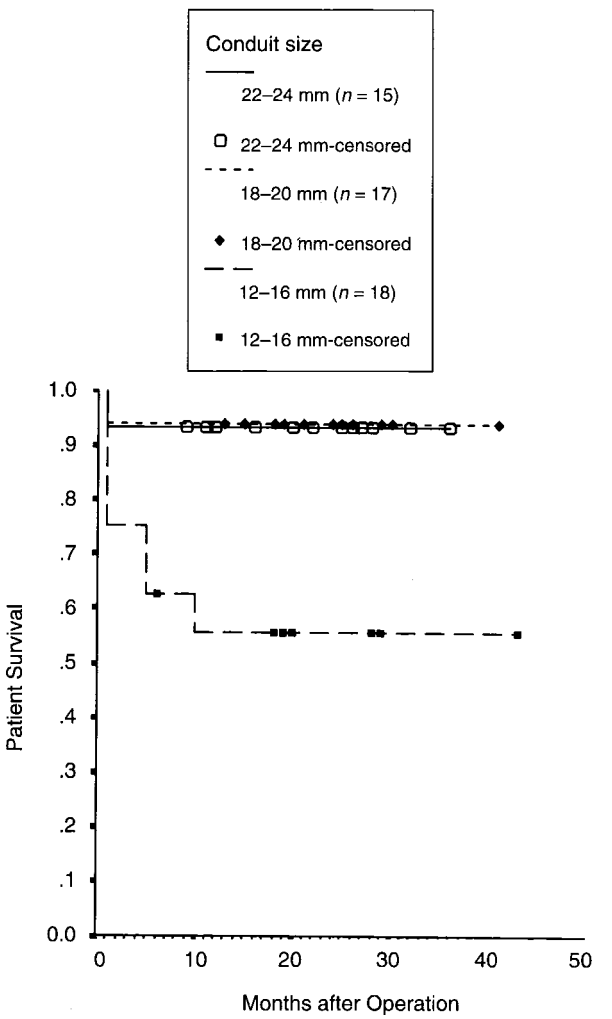

Figure 3. Patient survival in relation to the size of Polystan valved conduit.

bleeding by coating a knitted Dacron valved conduit with collagen, or prevention of neointimal hyperplasia by using woven Teflon conduits, has not yet been proven.[9]

The early mortality of 12% in this study was considered acceptable considering the complex lesions and associated procedures in this group of patients. Only one death was due to conduit thrombosis, and another patient died of operative bleeding. Most of the mortality occurred in patients with a small valve size; valve size was found to have a significant effect on patient survival. Razzouk and colleagues[10] compared 4 types of valved implants over 25 years; predictors of patient survival were found to be diagnosis and valve size. We believe that a small valve size per se does not cause perioperative problems so much as the complexity of heart lesions, preoperative ventricular function, and the need for concomitant procedures. Our previous experience indicated that size mismatch is an important factor in small children because too large a conduit at the initial operation can evoke coronary compression, bleeding, sternal erosion, and distortion of the pulmonary arteries.[11] Sano and colleagues[11] suggested using a conduit 5 to 8 mm larger in diameter than the size of the main pulmonary artery (normalized to the patient's weight and body surface area).

There were no deaths among those who underwent replacement of the Polystan conduit. Low mortality rates for conduit replacement have been reported, and the need for reoperation has a minimal effect on longevity and late clinical status.[9,11,12] Longer follow-up is required to determine freedom from reoperation in this group of patients. Reoperation may be required for conduit problems, especially in a significant number of small children. Somatic overgrowth sometimes makes conduit replacement inevitable. The decision to reoperate is influenced by symptoms, transconduit gradient, and dysfunction of the pulmonary ventricle. Conduits are considered to be obstructed when the gradient is more than 50 mm Hg.[1,10] The earliest indication of conduit failure is development of tricuspid insufficiency.[1,11] A number of studies showed that symptoms may be absent despite moderate obstruction.[13–15] Conduit replacement should be performed if significant pulmonary ventricular dysfunction develops.

The long-term durability of extracardiac valved conduits is not yet ideal. Creation of viable pulmonary artery autografts has been tried through tissue engineering by endothelialization with autologous cells.[16] However, xenografts including the Polystan valved conduit will continue to play a role in pulmonary ventricular outflow tract reconstruction until an ideal conduit is available.

REFERENCES

1. Homann M, Haehnel JC, Mendler N, Paek SU, Holper K, Meisner H, et al. Reconstruction of the RVOT with valved biological conduits: 25 years experience with allografts and xenografts. Eur J Cardio-thorac Surg 2000;17: 624–30.
2. Clarke DR, Campbell DN, Hayward AR, Bishop DA. Degeneration of aortic valve allografts in young recipients. J Thorac Cardiovasc Surg 1993;105:934–41.
3. Cerfolio RJ, Danielson GK, Warnes CA, Puga FJ, Schaff HV, Anderson BJ, et al. Results of an autologous tissue reconstruction for replacement of obstructed extracardiac conduits. J Thorac Cardiovasc Surg 1995;110:1359–68.
4. Mayer JE. Uses of homograft conduits for right ventricle to pulmonary artery connection in the neonatal period. Semin Thorac Cardiovasc Surg 1995;7:130–2.
5. Lamberti JJ, Angell WW, Waldman JD, Grehl TM, George L, Mathewson JW, et al. The cryopreserved homograft valve in the pulmonary position: early results and technical considerations. J Card Surg 1988;3:247–51.
6. Barbero-Marcial M, Baucia JA, Jatene A. Valved conduits of bovine pericardium for right ventricle to pulmonary artery connections. Semin Thorac Cardiovasc Surg 1995; 7:148–53.
7. Champsaur G, Robin J, Curtil A, Tronc F, Vedrinne C, Sassolas F, et al. Long-term clinical and hemodynamic evaluation of porcine valved conduits implanted from the right ventricle to the pulmonary artery. J Thorac Cardiovasc Surg 1998;116:793–804.
8. Daskalopoulos DA, Edwards WD, Driscoll DJ, Danielson GK, Puga FJ. Coronary artery compression with fatal myocardial ischemia. A rare complication of valved extracardiac conduits in children with congenital heart disease. J Thorac Cardiovasc Surg 1983;85:546–51.
9. Schaff HV, DiDonato RM, Danielson GK, Puga FJ, Ritter DG, Edwards WD, et al. Reoperation for obstructed pulmonary ventricle-pulmonary artery conduits. Early and late results. J Thorac Cardiovasc Surg 1984;88:334–43.
10. Razzouk AJ, Williams WG, Cleveland DC, Coles JG, Rebeyka IM, Trusler GA, et al. Surgical connections from ventricle to pulmonary artery. Comparison of four types of valved implants. Circulation 1992;86(Suppl II):154–8.
11. Sano S, Karl TR, Mee RBB. Extracardiac valved conduits in the pulmonary circuit. Ann Thorac Surg 1991;52: 285–90.
12. Bisset GS III, Schwartz DC, Benzing G III, Helmsworth J, Schreiber JT, Kaplan S. Late results of reconstruction of the right ventricular outflow tract with porcine xenografts in children. Ann Thorac Surg 1981;31:437–43.
13. Jonas RA, Freed MD, Mayer JE Jr, Castaneda AR. Long-term follow-up of patients with synthetic right heart conduits. Circulation 1985;72(Suppl II):77–83.
14. Heck HA Jr, Schieken RM, Lauer RM, Doty DB. Conduit repair for complex congenital heart disease. Late follow-up. J Thorac Cardiovasc Surg 1978;75:806–14.
15. Stewart S, Manning J, Alexson C, Harris P. The Hancock external valved conduit. A dichotomy between late clinical results and late cardiac catheterization findings. J Thorac Cardiovasc Surg 1983;86:562–9.
16. Shinoka T, Shum-Tim D, Ma PX, Tanel RE, Isogai N, Langer R, et al. Creation of viable pulmonary artery autografts through tissue engineering. J Thorac Cardiovasc Surg 1998;115:536–46.