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SURGERY FOR CONGENITAL HEART DISEASE

FIVE- TO FIFTEEN-YEAR FOLLOW-UP OF FRESH AUTOLOGOUS PERICARDIAL VALVED CONDUITS

Andrés J. Schlichter, MD^{a,b}
Christian Kreutzer, MD^{a,b*}
Rita de Cassia Mayorquim, MD^b
Jorge L. Simon, MD^{a,b}
Maria I. Román, MD^{a,b}
Haydee Vazquez, MD^a
Eduardo A. Kreutzer, MD^{a,b}
Guillermo O. Kreutzer, MD^{a,b}

Sponsor: Richard A. Jonas, MD

Objective: Evaluate long-term results of autologous pericardial valved conduits in the pulmonary outflow.

Methods: Between June 1983 and October 1993, 82 conduits were placed in the outflow of the venous ventricle. Patients who received homografts (n = 2 patients), heterografts (n = 3 patients), and valveless conduits (n = 19 patients) and those patients who died within 90 days after the operation were excluded. Fifty-four survivors of pulmonary outflow reconstruction with fresh autologous pericardial valved conduits were followed up from 5 to 15 years (mean, 7.47 ± 2.8 years). Diagnosis include D-transposition of great arteries (n = 16 patients), L-transposition of great arteries (n = 14 patients), tetralogy of Fallot, pulmonary atresia with ventricular septal defect (n = 11 patients), truncus arteriosus (n = 10 patients), and double-outlet ventricle (n = 3 patients). Implantation age ranged from 0.25 to 24 years (mean, 5.2 ± 4.2 years). Median conduit diameter was 16 mm. Two-dimensional echocardiographic Doppler evaluations were made yearly; 9 patients underwent cardiac catheterization. Reintervention for stenosis was indicated when the pressure gradient exceeded 50 mm Hg.

From the Children's Hospital "Ricardo Gutierrez"^a and Clinica Bazterrica,^b Buenos Aires, Argentina.

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Address for reprints: Andrés J. Schlichter, MD, Hospital de Niños "Ricardo Gutierrez," Cirugía Cardiovascular, Gallo 1330, Unidad

17; 1425 Buenos Aires, Argentina (E-mail: schlichter@over-net.com.ar).

*Evarts A. Graham Traveling Fellow, 1998-1999.

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Results: Three late deaths were unrelated to the conduit. Thirty-five autologous pericardial valved conduits increased in diameter (1-7 mm), remained unchanged in 15 patients, and reduced 1 to 2 mm in 4 patients. The median diameter was 18 mm at the last evaluation ($P = .0001$). Eight patients required conduit-related reoperation 3 to 8 years after the implantation. Two patients underwent balloon dilation of the autologous pericardial valved conduit. No conduit had to be replaced. Freedom from reintervention at 5 and 10 years was 92% and 76%, being 100% at 10 years for conduits larger than 16 mm at time of implantation.

Conclusions: Autologous pericardial valved conduits show excellent long-term results and compare favorably with other conduits. (*J Thorac Cardiovasc Surg* 2000;119:869-79)

Repair of many complex congenital heart malformations requires the use of conduits to connect the outflow of the venous ventricle to the pulmonary artery. An ideal valved conduit should comply with the following requisites: (1) some potential for its diameter to increase with time, (2) low likelihood of shrinkage or development of intimal peel or thrombus, (3) long-lasting valve function, (4) ready availability, and (5) low cost. Many different types of conduits have been used since Rastelli and colleagues¹ reported the use of a nonvalved pericardial conduit in the outflow tract of a patient with tetralogy of Fallot (ToF) and pulmonary atresia with ventricular septal defect (VSD) and since Ross and Somerville² reported the use of a homograft. The first patient with an unvalved pericardial conduit, who has been followed up for more than 30 years, has not had to undergo reoperation.³ Follow-up of the different valved conduits showed a high incidence of reoperations because of obstruction caused by shrinkage, calcification, or outgrowth of the conduit.⁴⁻⁷ After the wide experience with the use of unpreserved pericardium in the reconstruction of the right ventricular outflow tract in ToF, since June 1983 we have been constructing a fresh autologous valved pericardial conduit to connect the venous ventricle with the pulmonary artery. The idea is to reproduce the excellent long-term results of untreated autologous pericardial patches and unvalved conduits, obtaining adequate valve competence for the first postoperative months.

After our original descriptions of the technique⁸⁻¹⁰ and the further publication of the short- and intermediate-term follow-up (mean, 4.2 years),¹¹ this article reports the results obtained after a mean of 7.47 ± 2.8 years in patients who received autologous pericardial valved conduits (APVCs) during the first 10 years of our experience.

Patients and methods

Patient selection. To have a follow-up of at least 5 years, the patients selected for this study must have survived the

implantation of an APVC at least 3 months and must have been operated on before December 1993.

Patient population. From June 1983 to October 1993, 82 conduits were placed in the pulmonary outflow tract. Patients who received homografts ($n = 2$ patients), heterografts ($n = 3$ patients), or valveless conduits ($n = 19$ patients) and those patients who died within 90 days of the operation were excluded. Thus 54 consecutive patients with an APVC implanted were evaluated regarding the conduit's result. Mean follow-up time was 7.47 ± 2.8 years (range, 1-13 years). Forty-nine patients were followed up for more than 5 years, and 12 patients were followed up for more than 10 years.

The mean age at operation was 5.25 ± 5 years (range, 0.25-24 years; median, 5 years). The diagnoses of the patients were D-transposition of the great arteries (D-TGA) with VSD and left ventricular outflow tract obstruction (LVOTO) in 16 patients (30%); L-transposition of the great arteries (L-TGA) with LVOTO with or without VSD in 14 patients (26%); truncus arteriosus in 10 patients (19%); ToF with or without pulmonary atresia, with or without major aortopulmonary collateral supply in 11 patients (20%); and double-outlet right or left ventricles 3 patients (5%; Fig 1). There were 28 male patients and 26 female patients.

The diameter of the conduits at the time of implantation ranged between 12 and 20 mm and is given in Table I, together with the age range. The median and mean diameter used was 16 ± 2.13 mm.

Follow-up. All of the 54 hospital survivors could be traced after hospital discharge. A questionnaire was mailed to the patients and to the primary physician of the patients who received follow-up at other institutions.

Postoperative evaluation included regular clinical examination at the outpatient clinic and chest radiographs every 6 months to search for conduit calcification. Two-dimensional Doppler echocardiographic evaluation was performed at regular yearly intervals as the method to assess conduit diameter at the proximal, valvular, and distal levels.¹² Peak pressure gradients were measured at the three levels of the conduit. When enlargement or reduction in the diameter of the conduit was reported, the smallest measured diameter was the one considered. Conduit enlargement was defined when it occurred at all the three levels of the conduit.

Fig 1. Relationship between diagnosis at operation, conduit-related reoperation, and conduit-related transcatheter dilation. *TGA*, Transposition of the great arteries; *ToF*, tetralogy of Fallot; *PA*, pulmonary atresia; *DOV*, double-outlet ventricle.

A reoperation was considered conduit related when a distal plasty, proximal plasty, or total conduit enlargement was performed. The reoperation was considered unrelated to the conduit when no procedure was performed to it. The term *conduit reintervention* is used for both conduit-related catheter interventions and surgical procedures.

Statistical analysis. All values of continuous variables were expressed as mean $\pm$ SD. The Kaplan-Meier method was used to estimate the cumulative survival of patients and survival of conduits that were free from reoperation and reintervention. The Wilcoxon signed rank test was applied for comparison between time-related variables. The statistical analysis was performed with SSPS software (SSPS Inc, Chicago, Ill) and STATA programs (Stata Corporation, College Station, Tex).

Surgical technique. After the removal of the pericardium from the right to the left phrenic nerves (leaving a 1-cm edge to the nerve) and from the diaphragm to the reflection on the aorta, the rectangular piece of tissue was placed on a wet drape (saline solution). The diameter of the conduit is chosen according to the patient's body surface area.¹³ The conduit is constructed according to the steps that have been previously reported.^{8,9,11} The serous layer of the pericardium will be the inner surface, and the fibrous pericardium will be the external aspect of the conduit. The harvested pericardium is then trimmed into two different geometric forms: a rectangle (the future conduit) and a trapezoid (the future bicuspid valve). The sizes of these pericardial forms are related to the diameter of the conduit and thus to the patient's body surface area. The sizes are shown in Table II.

The length of the rectangle is the measured distance from the pulmonary artery orifice to the venous ventricular incision. The rectangle is secured to the wet drape with 4 stay stitches at the angles. The trapezoid is superimposed on the rectangle with its longer base ($a'-a'$) placed 5 to 6 mm from the distal end of the rectangle (A-A). As a rule, the longer base of the trapezoid ($a'-a'$) is always 10% longer than the

Table I. Diameter of APVCs at implantation and at last follow-up examination

Diameter (mm)	No. of patients	Age (y)
At implantation*		
12	4	0.25-5
14	14	0.4-6
15	2	4
16	12	1-9
17	7	1.4-10
18	11	4-24
20	4	5-8
At last follow-up†		
11	1	
13	1	
14	3	
15	8	
16	6	
17	6	
18	8	
19	4	
20	4	
21	7	
22	3	
23	1	
24	1	

*Median of the diameter was 16 mm; the mean diameter was 16 ± 2.13 mm.

†Median of the diameter was 18 mm; the mean diameter was 17.94 ± 2.96 mm. Wilcoxon signed-rank test, $P < .0001$.

base of the rectangle (A-A). The trapezoid (valve) is secured to the rectangle at 3 different points with 6-0 polypropylene sutures. The serosal surface of the cusps is oriented to the sinuses. Two stitches join a' and A at both lateral ends, and a third stitch is placed in the exact middle of both pericardial pieces at point b (Fig 2, A, B, and C). The trapezoid is then trimmed; 3 small triangular pieces are excised to achieve 2 semilunar cusps, leaving intact the union of both in a length of 4 mm (commissure; Fig 2, D and E). Then the semilunar cusps are sutured to the pericardial rectangle, with the sutures used to secure the trapezoid at both lateral ends at a' and in the middle at b. On both lateral sides 4 mm of the suture are on the edge of the rectangle (commissure) (Fig 2, E). Once the valve is constructed, a Hegar dilator of the selected conduit diameter is placed over the pericardial rectangle with the cusps (Fig 2, F). Then a 6-0 polypropylene double-running suture is performed from the distal to the proximal end. This suture is started 2 mm from the distal end to leave a wider anastomotic orifice. The proximal end of this suture is left untied because it will be adjusted according to the exact length of the conduit at the time of the anastomosis to the ventriculotomy (Fig 2, G and H). Once the intracardiac repair is completed, the distal end of the conduit is sutured to the pulmonary artery with 6-0 polydioxanone suture. This is interrupted in the 4 quadrants and tied over the Hegar dilator to prevent distal end narrowing. In the first 3 years of our experience to 1986, distal narrowing at the time of tying the

Fig 2. APVC construction. Surgical technique. **A**, Pericardial trapezoid: $a-a'$ is the wider base; b is the midpoint, and H is the height of the future valve (Table II). **B**, Pericardial rectangle (note the wider distal end): $A-A'$ is the base of the rectangle; the trapezoid has been superimposed (note that the wider base of trapezoid [$a-a'$] is 10% wider than the base of the rectangle [$A-A'$]). **C**, Three double-armed sutures have been placed to secure the trapezoid to the rectangle at $A-a$, $A'-a'$, and b to the midpoint of the rectangle. **D**, The form of the cusps is given by trimming the 3 triangles at the base of the trapezoid. **E**, The running suture is performed from A and A' , suturing the trapezoid to the edge of the rectangle in the first 3 mm and then from b to secure the cusps to the rectangle (note that the first 3 mm of the suture stated at b is double). **F**, The pericardial rectangle with the cusps is wrapped around the corresponding Hegar dilator. **G**, The conduit is closed with a 6-0 double running suture. The suture is started at the distal end, and the proximal end remains untied. **H**, The completed valved conduit.

Table II. Size of the pericardium required, according to the selected conduit diameter

Diameter (mm)	Rectangle		Trapezoid	
	Width of pericardium (mm)	Height of valves (mm)	Lesser base (mm)	Wider base (mm)
12.0	38.0	11.0	38.0	42.0
14.0	44.0	12.5	44.0	48.5
16.0	51.0	13.5	51.0	56.0
18.0	56.5	15.0	56.5	62.0
20.0	63.0	16.0	63.0	69.0

sutures was the cause for distal stenosis. Since 1986 we have increased the width of the distal end of the pericardial rectangle by 4 mm, thus obtaining a wider anastomotic orifice (Fig 2, A-F). Then the proximal end of the conduit is sutured to the ventriculotomy. In only 2 cases was a polytetrafluoroethylene hood added to achieve an anastomosis without tension. In 8

selected cases, the pericardial sac was replaced with a polytetrafluoroethylene* 0.1-mm pericardial membrane before closing the sternotomy.

*Gore-Tex; registered trade name of W. L. Gore & Associates, Inc, Flagstaff, Ariz.

Fig 3. Kaplan-Meier patient survival estimate curve in the follow-up of APVC implantation, including 95% confidence limits and the number of patients followed at each interval.

Fig 4. Freedom from conduit related reoperation estimate (Kaplan-Meier), including 95% confidence limits and the number of patients followed up at each time interval.

Forty conduits were placed to the left of the aorta. In 14 patients with L-TGA with dextroversion, the conduit was placed to the right of the aorta.

The construction of the conduit takes a mean of 35 minutes and is usually performed by a staff surgeon or a chief resident. During this time the main surgical team completes the intracardiac repair of the malformation.

Results

Survival. No patients were lost to follow-up. There were 3 deaths during the follow-up period. One patient died of a brain tumor 8 years after conduit insertion, another patient died of sepsis 5.8 years after the operation, and the third patient died suddenly 1.1 years after the operation, probably of an arrhythmia caused by the progression of pulmonary vascular disease, without residual VSD or conduit obstruction.

The probability of patient survival (Kaplan-Meier) for patients with APVC was 100% at 1 year, 98% at 2 and 5 years, and 92% at 10 years (Fig 3).

Conduit function. Doppler echocardiography in the first postoperative months disclosed trivial or mild valve regurgitation in 48 patients (88%), moderate regurgitation in 5 patients (9%), and severe regurgitation in 1 patient (2%). Pulmonary (conduit) valve incompetence was disclosed by the finding of a moderate diastolic murmur in most of the patients after 6 to 18 months of follow-up.

The diameter of the conduits at the time of implantation ranged from 12 to 20 mm with a median of 16 and a mean of 16 ± 2.13 . At the time of most recent evaluation, the diameter of the conduits in the 54 patients ranged from 11 to 25 mm, with a median of

Fig 5. Freedom from conduit-related reintervention estimate, according to diagnosis. TGA, Transposition of the great arteries; DOV, double-outlet ventricle; ToF, tetralogy of Fallot.

Fig 6. Freedom from conduit-related reintervention estimate for the whole group, including 95% confidence limits and the number of patients followed up at each time interval.

18 mm and a mean of 18 ± 2.96 mm ($P < .001$, Wilcoxon signed rank test).

At most recent measurement, the diameter of the conduit in 35 patients was 1 to 7 mm larger than at implantation; in 15 patients, the diameter of the conduit remained unchanged; and in 4 patients, it had shrunk 1 to 2 mm.

The measured pressure gradients at late follow-up ranged from 0 (6 cases) to 96 mm Hg. The median gradient was 18.5 mm Hg. Only 8 patients showed gradients higher than 50 mm Hg. In these patients, reintervention was indicated. In 28 patients (more than 50% of the total group), the measured gradient was 20 mm Hg or less.

Of the 54 patients, 8 patients underwent conduit-related reoperations, and 2 patients underwent transcatheter

balloon dilation of the APVC; thus, 10 reinterventions were performed. There were 4 non-conduit-related reoperations and 1 transcatheter distal left pulmonary artery stent implantation. Conduit reoperations were performed 3 to 8.2 years after the initial operation. The probability of freedom from reoperation for the whole group at 1 year and 2 years was 100%, 96% at 5 years, and 80% at 10 years (Fig 4). Four of the reoperations were due to distal suture stenosis, all in patients who had undergone the operation before 1986. Later, the distal end of the conduit was enlarged as described; the suture was performed in 4 quadrants and tied over a Hegar dilator.

Conduit reoperations were indicated according to the gradient in the pulmonary outflow tract. The venous ventricular-pulmonary artery gradient in the 8 patients

Fig 7. Freedom from conduit-related reintervention estimate (Kaplan-Meier), according to size groups: group I, conduit diameter 16 mm or smaller, and the estimated probability of freedom from reintervention at 4 years is 97% (95% confidence limits, 0.80, 1.0), although at 8 years it is 0.67% (95% confidence limits, 0.45, 0.82); group II, conduit diameter larger than 16 mm, which includes the number of patients followed up at each interval of time, and the estimated probability of freedom from reintervention at 4 years is 1% (95% confidence limits, 0.96, 1.0). At 8 years the lower confidence bound is 0.92. Confidence limits overlap at 4 years but not at 8 years.

ranged from 30 to 96 mm Hg, with a mean of 67 mm Hg. Even though reoperation was indicated with gradients higher than 50 mm Hg, 1 patient with a gradient of only 30 mm Hg underwent conduit revision because of the coexistence of an LVOTO.

None of the obstructions were found to be at the valvular level. In fact, in 6 patients who underwent reoperation later than 1 year after conduit insertion, the valve could not be identified, although it was found pliable in all of the patients who underwent reoperation because of residual malformations unrelated to the conduit in the 6 postoperative months. The valves were found to be pliable between 4 and 6 months after implantation.

The relationship between preoperative diagnosis, conduit-related reoperation, and catheter dilation is shown in Fig 1.

Three of the 10 patients with truncus arteriosus underwent reoperation because of conduit stenosis, and 1 patient underwent reoperation because of distal end stenosis. The median age of these patients at the initial operation was 6 months. These 3 patients underwent reoperation 6 to 7.25 years after the initial operation. Three of the 16 patients with D-TGA underwent reoperation 3 to 7.75 years after the initial insertion of the conduit. One of the reoperations was due to distal suture stenosis.

One patient with ToF and either double-outlet right or double-outlet left ventricle underwent conduit-related reoperations because of distal-end stenosis 8 and 5 years after conduit insertion. None of the patients with

L-TGA required a reoperation or catheter intervention because of conduit stenosis.

Of the 8 conduit-related reoperations, 4 reoperations were distal-end suture stenosis; in the other 4 reoperations, the conduit was uniformly restrictive, but the patient had outgrown the conduit. In those patients who required reoperation, the entire length of the conduit was augmented with a polytetrafluoroethylene patch in 4 patients; distal plasty was performed in the other 4 patients. In 3 of the reoperations, some degree of conduit calcification was observed.

The freedom from reintervention estimates by preoperative diagnosis (Kaplan-Meier) are shown in Fig 5. The log-rank test for equality showed that time to reoperation differs by diagnostic group ($P = .02$). However, when the group of double-outlet ventricles ($n = 3$ patients) is eliminated, there is no significant difference among D-TGA/L-TGA/truncus and ToF ($P = .18$).

Two patients were subjected to transcatheter balloon dilation 4 and 4.33 years after the conduit insertion. In both cases a distal-end stenosis was relieved successfully, and the pressure gradients dropped from 45 to 12 mm Hg and 96 to 23 mm Hg. The freedom from reintervention estimate (Kaplan-Meier) for the whole group showed 100% at 1 and 2 years, 92% at 5 years, and 76% at 10 years (Fig 6). In 1 additional patient, a stent was inserted distally in the left pulmonary artery but was unrelated to the conduit.

The relationship between diameter of the conduit at implantation and conduit-related reoperation/reinterva-

tion rate was analyzed. Because the median conduit diameter at the time of insertion was 16 mm, 2 groups were compared: group I, 32 patients (59%) with diameters equal to or less than 16 mm; group II, 22 patients (41%) with conduits larger than 16 mm in diameter.

All the reinterventions (catheter and surgical operations) were performed on group I patients. Thus, no patient with a conduit larger than 16 mm at the time of implantation had to undergo reoperation. The Kaplan-Meier reoperation-free conduit survival estimates (Fig 7) show that time to reoperation is shorter for patients with conduit size equal to or less than 16 mm ($P = .07$). In patients who underwent reoperation, a small biopsy specimen of the conduit tissue was sent for pathologic examination. These specimens were consistently reported as having a cellular matrix with two layers, one endothelialized neointima and one fibrous with collagen and fibroblasts.

Discussion

The use of valved conduits has become a common procedure in the correction of congenital heart disease. TGA (either D-TGA or L-TGA) with VSD and LVOTO, ToF with pulmonary atresia, ToF with anomalous coronary artery, double-outlet right ventricle, double-outlet left ventricle, and truncus arteriosus are the malformations in which these conduits are used to achieve venous ventricle-pulmonary artery continuity. Cryopreserved homografts have replaced heterografts as the conduit of choice in the past 2 decades. Good short-term results have been obtained with homografts,^{12,14,15} but the long-term results are disappointing.^{5-7,13} Furthermore, in the long term, the results of homografts are comparable with those of heterografts.^{16,17} Even the valve function of homografts did not show a substantial difference with heterografts in the long term, and the failure rate was almost 100% when they were used in the pulmonary outflow tract, as recently reported.^{18,19} When present, obstruction in homografts most frequently occurs at the valvular level, necessitating transcatheter balloon dilation and/or stent implantation, which carries the unavoidable consequence of pulmonary regurgitation.¹⁹ Heterografts, on the other hand, have the early disadvantage of bleeding; their use is inadequate in neonates and infants with thin and friable pulmonary arteries and coronary compression by the valve ring. Late failure is mostly associated with peel tissue ingrowth, valve degeneration/calcification, and obstruction.^{17,20}

After long experience and excellent results achieved by pericardial patching of the right ventricular outflow tract²¹ and the use of nonvalved pericardial conduits for ToF and pulmonary atresia,¹ we designed an autolo-

gous valved pericardial conduit. Autologous unpreserved pericardium has been shown to be a durable tissue that is pliable and easy to handle and suture. Our aim was to construct a valved conduit that would combine the advantage of good pulmonary valve function during the first months after repair, when it is crucial, and long-term conduit durability.

These conduits offer several advantages, including low cost, easy construction, absence of antigenicity, capability of increasing its diameter, no need for sterilization, and stent-free design.

With the present trend to reduce costs in the developed countries and the importance of low cost and availability in developing countries, the option of an APVC is attractive. It has two advantages when cost is considered: (1) The conduit is custom-made for the patient with his own tissue; all that is needed are 4 to 6 sutures of 6-0 polypropylene plus a few stay sutures and 35 minutes of time for one surgeon. (2) Costs are greatly reduced by the fact that the incidence of conduit reoperation is low.

APVCs have demonstrated an increase in diameter with time. In 35 of the 54 conduits in this series, the diameter increased 1 to 7 mm. We do not consider this increase in diameter as real growth, because there was no evidence of cellular proliferation. Nevertheless, this increase in diameter is a unique feature of these conduits. The long-term results have been excellent. A freedom from reoperation of 80% at 10 years compares favorably with data on all other available conduits. Lacour-Gayet and colleagues²² and Kreutzer²³ have reported 100% freedom from reintervention at 7 years using APVCs constructed with this technique in patients with truncus arteriosus. This is somewhat similar to our own experience with the same malformation.

Even though 3 of the 10 patients with truncus arteriosus had to undergo reoperation, these were the patients with the smallest conduit diameters, ranging from 12 to 16 mm (median, 14 mm). Another additional benefit is that none of the patients who underwent reoperation had to have the conduit replaced, just enlarged. This confers an additional advantage. The patients with distal-end suture stenosis needed only distal-end patch augmentation at reoperation.

The construction of the valved conduit has a learning curve. Because the conduit is an essential part of the correction of these complex malformations, the conduit construction should be precise. The sizes in Table II should be strictly followed. The training in manufacturing and testing of this type of conduit may be performed in the animal laboratory as reported by Chiu,²⁴ Kumar²⁵ and their colleagues.

At the time of implantation, the placement of the distal polydioxanone suture in 4 quadrants and over a Hegar dilator is crucial. Four patients in the initial part of our experience had to undergo reoperation because of distal-end stenosis.

One disadvantage of this technique is that it is usually not possible to harvest pericardium when adhesions from previous operations are present. Despite this, we were able to do so in one patient with a double-outlet right ventricle of the Taussig-Bing type, in whom a pulmonary band and repair of a coarctation had been performed 8 months before conduit implantation. However, we are certain that this is the exception, not the rule.

The short period of competence of the APVC valve is a relative disadvantage. Pulmonary competence in the immediate postoperative period is associated with better outcome,²⁶ and pulmonary regurgitation is mostly well tolerated in the late outcome if not associated with other residual defects such as VSD, distal stenosis or pulmonary hypertension, and tricuspid regurgitation.

Although pulmonary incompetence for more than 20 to 30 years has been shown to have a deleterious impact on right ventricular function, all other conduits necessitate numerous reoperations in that period of time, affecting the right ventricle because of systolic pressure overload. On the other hand, patients who have undergone L-TGA will probably withstand pulmonary incompetence better because of the underlying left ventricle.

When pulmonary valve function is essential, as in ToF with pulmonary atresia and peripheral stenosis and in patients with severe pulmonary hypertension caused by pulmonary obstructive disease, the use of these conduits is not advocated. In such patients the use of homografts is preferred.

Even though we did not use the APVC for pulmonary autograft aortic valve replacement²⁷ in the time frame of this series, it may be the conduit of choice for right ventricular outflow tract reconstruction. Recently, Garcia and colleagues²⁸ reported the use of APVCs in 9 such operations without any conduit-related reoperations during follow-up.

Limitations of the study. Even though we report the use of these conduits in a variety of lesions, the number of patients in each group precludes the identification of predictors of failure by multivariate analysis. Therefore, some of the statistical differences may be due to chance alone.

Conclusion

The use of APVCs in the venous ventricular outflow tract has shown very good results, and it proved to be

the definitive conduit for more than 80% of the patients in the long term.

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Discussion

Dr William G. Williams (*Toronto, Ontario, Canada*). I congratulate the group from Buenos Aires for focusing our attention on an important cause of morbidity and mortality in congenital heart disease, the extracardiac valved conduit.

As Mr de Leval has said, the extracardiac valved conduit is a time bomb. In our experience with more than 1000 valved conduits, the overall reoperation rate within 10 years of implantation is about 50%. Reoperation rates are much higher in younger patients with smaller conduits and, conversely, much better in mature patients, approaching 90% conduit survival at 10 years. Your data confirm this trend when you report that the conduits under 16 mm in diameter fare less well than those with larger conduits.

Because the age of the patient and the size of the conduit are so important in determining durability, it is difficult to accept your conclusion regarding improved durability with-

out age- and size-matched controls of other conduits. Nevertheless, your conduit has been quite successful.

Failure of valved conduits is almost invariably the result of progressive stenosis, and most commonly the stenosis occurs at the valve. It is a paradox that the success of your valved conduit is due to the disappearance of the valve leaflets within about 6 months of implantation. The patients are left with a nonvalved conduit, thereby avoiding the most common reason for reoperation, valve stenosis. Fortunately, the outlook for patients with no pulmonary valve and an otherwise normal heart is a 20- to 30-year interval free of symptoms. Later in life, a pulmonary valve will be necessary for adequate right heart function.

Why do the leaflets disappear? The monocusp leaflet used occasionally in ToF repair also seems to disappear. In both situations the reconstructed outflow tract lacks a sinus of Valsalva. The sinus of Valsalva is an important part of the valve mechanism. It prevents the leaflet from touching the arterial wall in systole and, importantly, it initiates its movement toward the closed position at end-systole.

Have you considered ways of making the leaflets more durable? Specifically, would incorporating a sinus of Valsalva in the prosthesis make the leaflets last longer? Would other leaflet material be more durable without the risk of stenosis, for example, glutaraldehyde-treated autologous pericardium?

You outline your indications and contraindications. Are there other factors that might lead you to not use this very inventive pericardial conduit?

Dr Schlichter. To address your question about sinuses, we have not planned to build sinuses into it. Autologous unpreserved pericardium is a very soft tissue, and it will probably not maintain the form of a sinus of Valsalva. What we have considered is to use glutaraldehyde-preserved valves, and we did so in 1 case. However, that approach goes against our philosophy because it will probably preserve the valve, but the valve will probably calcify. We prefer the vanishing valves instead of the stenotic valves, and that is why we did not consistently try to preserve the valves.

Dr Radu C. Deac (*Tirgu-Mures, Romania*). Contrary to the hopes of many cardiovascular surgeons, the long-term fate of fresh, untreated autologous pericardium used in the bloodstream so far, in experimental and clinical cases, was fibrosis, shrinkage, and calcification.

Your results are much different concerning the conduit, not the valve. Do you have any histologic information about the structure of the autologous pericardium of the conduit or of the leaflets in those replaced or retrieved? Does the difference in size (which you showed) indicate growth or dilatation? Is there any evidence about the revascularization of the tube of the conduit? Your results showed that the autologous pericardium, although not ideal, is the best available tissue in the right side of the heart.

Dr Schlichter. We have evaluated histologic information. We consistently found that the pieces sent to pathology during reoperation showed an intimal layer of neointima and cellular matrix with fibroblasts and collagen tissue. I believe the reason that we did not see shrinkage is that the pericardium used in the arterial circuit behaves differently than it does under atrial pressures.

We do not consider the conduit enlargement to be growth but simple dilation. Growth would presuppose cellular proliferation, and we could not identify that.

Regarding revascularization, we observed invasion of neovessels into the pericardium in one patient. This was a patient who underwent reoperation approximately 1½ years after the initial operation.

Dr C. E. Anagnostopoulos (*New York, NY*). It has long been known that the tensile strength and compliance of various tissues lag far behind those of the cardiovascular system, particularly pericardium, dura mater, and fascia lata. By contrast, the rectus sheath is virtually an identical match to aortic valve and aorta. We now have long-term results in 7 patients in whom rectus sheath was used on the right and left sides. We have seen that it becomes virtually an aorta, with many layers and cells, and it can grow, in contrast to the pericardium, which does not. My question is whether in your experience, since 1993, you have modified the type of valve that you are using and whether you have considered using valveless conduits.

Dr Schlichter. We did not change the type of the valve that we used. We continued following exactly the same procedures. The only thing we changed in the whole 15 years is that we are now tying over the dilator and are doing our sutur-

ing within 4 quadrants with polydioxanone suture. We are presently not using a nonvalved conduit because we believe that pulmonary valve competence is important in the first postoperative weeks. We always try out the conduits to make sure they do not leak a single drop.

Dr Francois Lacour-Gayet (*Paris, France*). We have used the APVC to repair truncus arteriosus in small-weight infants and have been satisfied with the long-term results. We had to reoperate on 1 patient after 10 years; the diameter of the conduit had not expanded from its 12-mm size when it was initially inserted. It had not grown, but it has not shrunk; the valve was absent.

I must say that I hope this worked, because what we do when we repair the pulmonary artery with fresh native pericardium in the arterial switch operation is not very different. In many patients, after total harvesting of two sinuses of Valsalva, we end up with a very small arterial wall. I suspect that reconstructing the pulmonary artery with pericardium in the arterial switch operation is quite similar to what you do when you construct a pericardial conduit from the right ventricle to the pulmonary artery. Therefore, I support this technique, and I believe that it could be useful in places in which small-sized homografts are not available or in countries where homografts are unavailable.