The Fate of Homograft Conduits in Children With Congenital Heart Disease: An Angiographic Study

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The use of homograft conduits in the repair of congenital heart disease is widely accepted. We reviewed the catheterization and angiographic data from 20 patients with homograft conduits. All conduits were to the pulmonary arteries. The age at operation was 4.7 ± 5.6 years (mean ± standard deviation) and at follow-up catheterization, 7.8 ± 6.7 years. At implantation, conduit cross-sectional area and Z value were 219 ± 96 mm^2 and 3.5 ± 1.8, respectively. At subsequent catheterization, the conduit diameters were measured in two projections at the shaft, annulus, valve opening, and insertion into the pulmonary artery. The transconduit gradient was 47 ± 26 mm Hg. The cross-sectional areas were 149 ± 56 mm^2 at the shaft, 151 ± 92 mm^2 at the annulus, 108 ± 116 mm^2 at the valve opening, and 127 ± 84 mm^2 at the pulmonary artery insertion. The Z values were −0.9 ± 2.5, −0.9 ± 2.8, −3.8 ± 4.0, and −2.0 ± 3.4, respectively. The cross-sectional areas and the Z values at the levels of measurement were significantly smaller than the corresponding values at implantation. The change in cross-sectional areas and Z values exceeded what would be expected from growth alone. These data indicate that there is a decrease, with time, in the functional lumen of homograft conduits, and this may have implications for follow-up strategy after implantation.

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The application of homograft conduits to the repair of congenital cardiac anomalies was pioneered by Ross and Somerville [1] in England when they employed a conduit in the reconstruction of the right ventricular outflow tract of a patient with pulmonary atresia and a ventricular septal defect. Thereafter, in the United States, irradiated homograft conduits were used. Problems of availability, however, prompted a transition to a porcine valve incorporated into a Dacron tube conduit [2, 3]. However, conduit deterioration and obstruction secondary to a diffuse intimal peel, calcification of the conduit's valve, or both often necessitated replacement [4]. Fresh or cryopreserved, antibiotic-sterilized homograft conduits have become widely accepted, and reports of improved hemodynamic performance and durability are available [2, 5]. Deterioration (independent of the preparation technique) of the cryopreserved homograft conduit has been reported [6]. Conduit obstruction can occur proximal to or at the valve, over the "shoulder of the heart," distal to the valve, or as a diffuse narrowing of the conduit [6]. The need to replace these conduits for homograft-related problems has been reported in select studies to be as low as 13% at 10 years for fresh conduits [5] and as high as 45% at 5 years for cryopreserved conduits [6].

The aim of this study was to evaluate the angiographic changes that can occur in homograft conduits after implantation in patients with congenital heart disease and to determine the relation of these changes to the hemodynamic abnormalities and the need of replacement. These data may provide insights into the fate of these conduits.

Material and Methods

From January 1987 through October 1992, 50 homograft conduits were implanted at our institution in 43 patients. The technique employed has been described previously [7]. We did not attempt to assure the compatibility of major blood groups between the donor and recipient of each conduit. There were eight early and two late deaths not directly related to conduit failure. Causes of death included pulmonary hypertension (4 patients), cardiac failure (2), and sepsis with respiratory failure (2). Seven patients had replacement of the conduit with a new homograft conduit during the period studied. Of the 33 surviving patients, 17 (52%) underwent catheterization. Three other patients who had an operation at another institution and subsequently were followed by us also underwent catheterization during the study interval and were included.

We reviewed retrospectively the charts of these 20 patients. The indications for catheterization included the presence of symptoms (chest pain in 1 patient and easy fatigability in 2 patients) and a persistent right-to-left shunt across an atrial septal defect with worsening cyanosis (1 patient). Eight of the patients had catheterization because of evidence by Doppler echocardiography of increased pulmonary ventricular pressure and 2 other patients, because of noninvasive evidence of severe tricuspid valve regurgitation with normal right ventricular pressure. Doppler interrogation of the left ventricle–pulmonary artery

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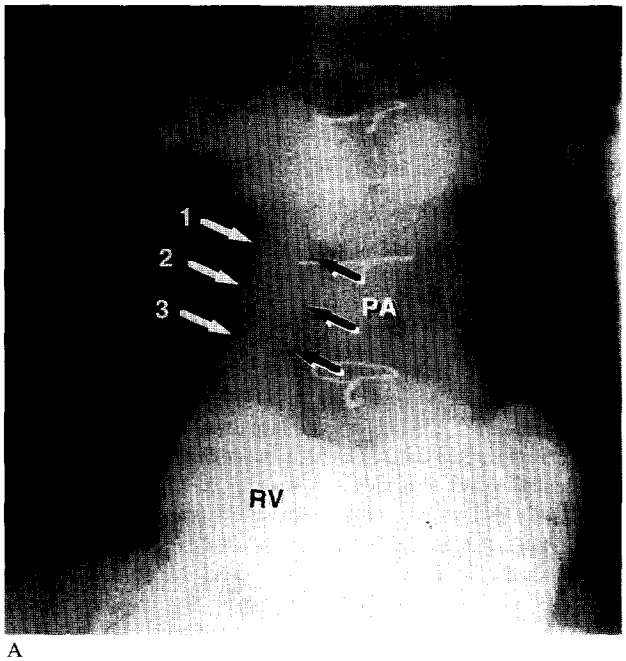
conduits in 2 patients with transposition of the great arteries and severe left ventricular outflow tract obstruction was impeded by the conduit's position. They were believed to have clinical findings compatible with severe conduit obstruction. Otherwise, in the absence of symptoms or evidence of obstruction, 4 patients underwent routine catheterization 5 or more years after implantation of the conduit.

The evaluation of the angiograms included measurement of the diameters of the conduit at four sites: the annulus of the valve, the shaft, the contrast jet through the conduit's valve, and the point of insertion into the pulmonary artery (Fig 1). The measurements were performed in both the posteroanterior and lateral projections. Correction for magnification was performed using the known diameter of the angiography catheter as a reference. All angiograms were reviewed by three of us who agreed on the points to be measured.

In the two orthogonal planes, the diameters of most conduits were not always equal. Therefore, we used the formula of an ellipse to calculate the cross-sectional area (CSA). The Z values of the regions measured along the conduit were also calculated. The Z value is the measured diameter of the conduit minus the predicted normal mean pulmonary artery diameter for individuals with the same body surface area divided by the standard deviation of the predicted normal mean pulmonary artery diameter [8]. Thus, a Z value is a measure of how many standard deviations a certain diameter diverges from the mean of a normal population of children with the same body surface area. The diameters of the left and right pulmonary arteries at the first bifurcation and the diameter of the descending aorta at the diaphragmatic level were measured from the posteroanterior views, and the McGoon ratio [9] was calculated. The most recent angiogram of each patient was used for the analyses. The gradient across the conduit was measured as the peak-to-peak pressure difference from the ventricle to the pulmonary artery distal to the conduit. Significant conduit obstruction was defined as a peak-to-peak gradient of 40 mm Hg or more [2] or a pulmonary ventricular systolic pressure greater than 50% of systemic ventricular systolic pressure.

Eight of the patients had more than one follow-up catheterization. In the case of these patients, a separate analysis of the first and last angiograms after homograft implantation was done. Patients who required a second conduit during the interval studied were compared with those who did not. The reports of the pathologic evaluation of the explanted conduits, but not the actual specimens, were available for analysis. Data analyses were performed using Statview 4.0 (Abacus Concepts, Inc, Berkeley, CA). Data are summarized as the mean ± the standard deviation. Unpaired and paired Student's t tests were used to assess differences between groups and within each group, respectively. Nonparametric variables were tested using the χ^2 test. Correlations were estimated by regression analysis. Survival of the conduits was analyzed by the Kaplan-Meier method. A p value of less than 0.05 was considered significant.



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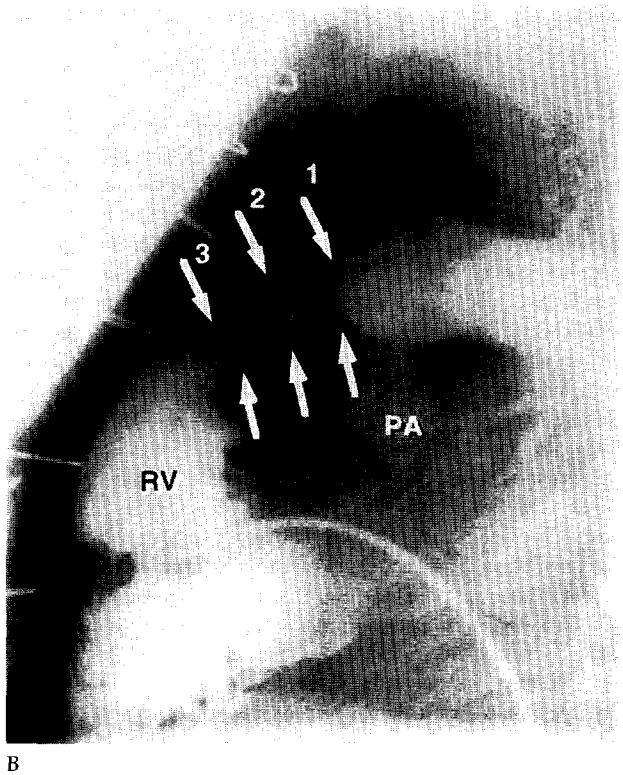

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Fig 1. Right ventricular cineangiograms in (A) posteroanterior and (B) lateral projections from a patient with transposition of the great arteries, ventricular septal defect, and pulmonary stenosis who underwent a Rastelli-type operation and placement of an external homograft conduit from the right ventricle (RV) to the pulmonary artery (PA) (conduit diameter, 19 mm). Both projections show three sites of measurement of the diameter of the conduit: at the insertion into the pulmonary artery (1), the shaft (2), and the annulus (3). The valve opening is not clear in these frames.

Results

The 3 patients who underwent operation at another institution were comparable to the group from our institution except for the length of follow-up (72 ± 57 months versus 31 ± 19 months, respectively; $p < 0.03$).

All Patients

Diagnoses included tetralogy of Fallot with pulmonary atresia (n = 6), truncus arteriosus (n = 7), transposition of the great arteries with left ventricular outflow tract obstruction (n = 5), and pulmonary atresia with intact ventricular septum (n = 2). The cardiopulmonary bypass time was 158 ± 48 minutes and the aortic cross-clamp time 55 ± 15 minutes. Conduits to the pulmonary arteries were from the right ventricle and the left ventricle in 16 and 4 patients, respectively. One patient with transposition of the great arteries and ventricular septal defect underwent patch closure of the ventricular defect, thus committing the left ventricle to the aorta, and placement of a right ventricle–pulmonary artery conduit. The male to female ratio was 1:1 (10 boys and 10 girls).

Eighteen conduits were of aortic origin and two, pulmonary origin. Sixteen (89%) of the 18 aortic conduits were calcified (on chest radiography) at the time of catheterization. The conduits of pulmonary origin were 15 mm and 18 mm in diameter at implantation, and neither conduit was calcified at the time of catheterization. Both of these conduits had significant obstruction at catheterization with ventriculoarterial systolic gradients of 45 mm Hg and 94 mm Hg, respectively. As expected, there was a positive correlation between conduit diameter at insertion and age at operation ($r = 0.78$, $p < 0.0001$).

The donor's major blood group type was known in 14 instances, and in two, it was incompatible with the recipient's blood type. Rh incompatibility was present in 5 other patients. Blood type incompatibility, albeit only in 2 patients, was not associated with either the severity of calcification or the magnitude of obstruction.

Demographic data at the time of operation and the subsequent follow-up catheterization are presented in Table 1. The duration of follow-up ranged from 7 to 131 months with a mean of 37 months.

Table 1. Patient Characteristics and Follow-up Data

Variable	Mean ± Standard Deviation	Range
Conduit implantation		
Age (y)	4.7 ± 5.6	0.2–21.6
Height (cm)	95 ± 34	60–175
Weight (kg)	17.2 ± 15.3	3.9–59.1
Body surface area (m^2)	0.65 ± 0.43	0.24–1.7
Conduit annulus diameter (mm)	16 ± 4	10–22
Catheterization		
Age (y)	7.75 ± 6.7	1.2–25.3
Height (cm)	118 ± 32	70–174
Weight (kg)	28.7 ± 21.6	9.8–78
Body surface area (m^2)	0.94 ± 0.48	0.47–1.90
Follow-up (mo)	37 ± 30	7–131

Table 2. Angiographic Measurements and Catheterization Data

Cross-Sectional Area (mm^2)	Mean ± Standard Deviation	Range
At implantation	219 ± 96	79–380
At last catheterization		
Shaft	149 ± 56[a]	48–261
Annulus	151 ± 92[b]	38–386
Valve opening	108 ± 116[c]	10–487
Insertion into pulmonary artery	127 ± 84[d]	20–331
Gradient (mm Hg)	47 ± 26	8–110

[a] Significance: $p < 0.005$, cross-sectional area compared with homograft annulus at implantation. [b] Significance: $p < 0.0004$, cross-sectional area compared with homograft annulus at implantation. [c] Significance: $p < 0.001$, cross-sectional area compared with homograft annulus at implantation. [d] Significance: $p < 0.0003$, cross-sectional area compared with homograft annulus at implantation.

The gradient at catheterization is shown in Table 2. Both the systemic and pulmonary ventricular systolic pressures had a positive correlation with age at operation ($r = 0.63$, $p < 0.003$; $r = 0.51$, $p < 0.03$, respectively) and with age at catheterization ($r = 0.69$, $p < 0.0009$; $r = 0.47$, $p < 0.04$, respectively). The mixed venous saturation was used as an indicator of cardiac output. It correlated positively with age at catheterization ($r = 0.55$, $p < 0.02$). The systolic pressure of the pulmonary ventricle tended to be higher in older patients with higher mixed venous saturation and, thus, higher cardiac output. The pulmonary to systemic ventricular systolic pressure ratio was 0.7 ± 0.2 (range, 0.2 to 1.0). This ratio had no significant correlation with any of the following variables: age at implantation ($r = 0.22$, $p > 0.3$) or catheterization ($r = 0.15$, $p > 0.5$); duration of follow-up; size of homograft at implantation; and CSA or Z value at any site within the conduit.

The mean peak-to-peak systolic gradient from the pulmonary ventricle to the pulmonary artery was 47 ± 26 mm Hg. There was a significant positive correlation between gradient and both age at homograft implantation ($r = 0.47$, $p < 0.04$) and age at catheterization ($r = 0.5$, $p < 0.02$). There was no significant relation observed between the peak-to-peak gradient and any of the following variables: follow-up duration; homograft diameter at implantation; CSA at any site (shaft [$r = 0.007$, $p > 0.9$], annulus [$r = 0.43$, $p = 0.06$], valve opening [$r = 0.10$, $p > 0.6$], and site of insertion [$r = 0.05$, $p > 0.8$]); and increase in body surface area from operation to catheterization. The CSA of the conduit at all levels measured was significantly smaller than the comparable level at the time of implantation (see Table 2). The smallest CSAs were at the valve opening and the insertion site into the pulmonary artery (see Table 2). The McGoon ratio at follow-up catheterization was 2.1 ± 0.4 (range, 1.5 to 2.9).

Conduit Replacement

Sixteen patients had a pulmonary to systemic ventricular pressure ratio greater than 50%, and 12 of them had a

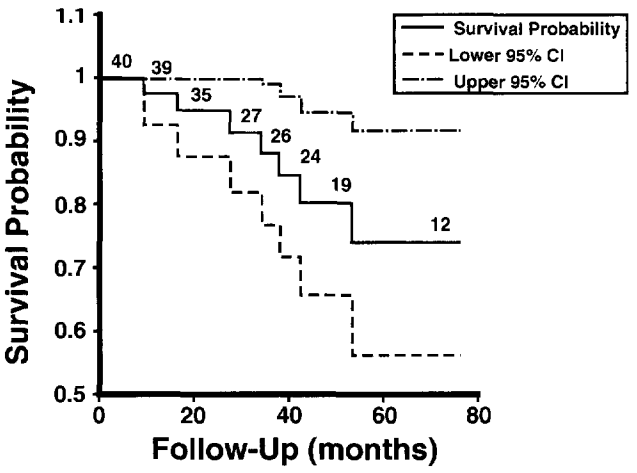

Fig 2. Kaplan-Meier actuarial analysis of freedom from reoperation to replace cryopreserved homograft conduits. Only the 33 surviving patients were included in the analysis. The numbers on the graph indicate the numbers of conduits at each point of follow-up. The broken lines represent the upper and lower 95% confidence intervals (CI).

transconduit gradient greater than or equal to 40 mm Hg. The need of conduit replacement was determined on a case by case basis in collaboration with the patient's cardiologist. In 7 patients, conduits were replaced, and 2 patients are awaiting replacement. These 9 patients are considered the replacement group.

The gradient across the conduit was 59 ± 29 mm Hg (range, 24 to 110 mm Hg) in the replacement group versus 34 ± 18 mm Hg (range, 8 to 64 mm Hg) in the no-replacement group ($p < 0.03$), and the pulmonary to systemic ventricular systolic pressure ratio was 0.8 ± 0.2 versus 0.6 ± 0.2, respectively ($p < 0.05$). Otherwise, the replacement group did not differ from the no-replacement group in age, sex, age at operation, cardiac defect, follow-up duration, somatic growth (as measured by change in weight, height, and body surface area), size of homograft at implantation, and Z values or CSA of the conduit at any level measured.

The actuarial survival of the homograft conduit without need of replacement in the surviving patients was 74% (95% confidence intervals, 56% to 92%) at 76 months (Fig 2). The survival of the homograft conduit was not related to original diagnosis, sex, race, or patient age at the time of operation.

Pathology

The pathologic reports for six of the explanted conduits were available for review. All conduits had severe calcification within the wall. The extent of valvar involvement ranged from a normal-appearing valve with intact leaflets and no calcification to a valve with two of its leaflets no longer present and the third reduced to a nonfunctional pocket. Microscopically, the walls of the conduits had dense fibrous tissue with calcification of the media. In addition, mild chronic inflammation (mononuclear cells) was present.

Z Values

The mean Z value for the homograft conduits implanted was 3.5 and ranged from a minimum of 0.1 (indicating a homograft diameter virtually equal to the mean normal diameter of a predicted native pulmonary artery in a patient with the same body surface area) to a maximum of 7.4 (indicating a conduit diameter 7.4 standard deviations above the predicted normal mean). The Z value of the homograft conduit at catheterization, assuming a theoretically "perfect" homograft where no interval changes occurred (neither growth nor "shrinkage"), was 1.6, which indicates that the conduit diameter implanted (16 ± 4 mm) would be 1.6 standard deviations above the predicted normal pulmonary artery diameter. Because of growth of the patients, the Z values at the different points of measurement were significantly smaller than the Z values at implantation. Moreover, the Z values at the annulus, shaft, valve opening and insertion site were significantly smaller than the predicted Z value of a "perfect" homograft conduit (Table 3, Fig 3).

Serial Catheterization

The 8 patients with at least two catheterizations had the first after a mean follow-up of 19 months (range, 2 to 49 months) and the last catheterization after 47 months (range, 24 to 72 months). At the time of the last catheterization, these patients were similar to the remainder of the group in age, age at operation, size of homograft implanted, overall length of follow-up, changes observed in conduits on the last follow-up catheterization, indication for catheterization, and need of conduit replacement. There was no significant change from the first catheterization to the second in mean pulmonary ventricular pressure, mean gradient across the conduit, and CSA or Z values (Table 4).

Comments

The ideal conduit for the repair of a congenital heart defect has yet to be found. Although homograft conduits have been used since 1966 [1], conduit obstruction is still problematic.

Table 3. Z Values of Homograft Conduits

Variable	Mean ± Standard Deviation	Range	*p* Value[a]
Conduit at implantation	3.5 ± 1.8	0.1 to 7.4	...
At catheterization			
Shaft	−0.9 ± 2.5	−4.6 to 3.9	<0.0004
Annulus	−0.9 ± 2.8	−5.3 to 3.9	<0.0006
Valve opening	−3.8 ± 4.0	−8.0 to 6.1	<0.0001
Insertion into pulmonary artery	−2.0 ± 3.4	−6.4 to 5.5	<0.0001
"Perfect" homograft	1.6 ± 2.0	−1.6 to 6.0	...

[a] The *p* values refer to the comparison between the "perfect" homograft and each of the levels measured at catheterization.

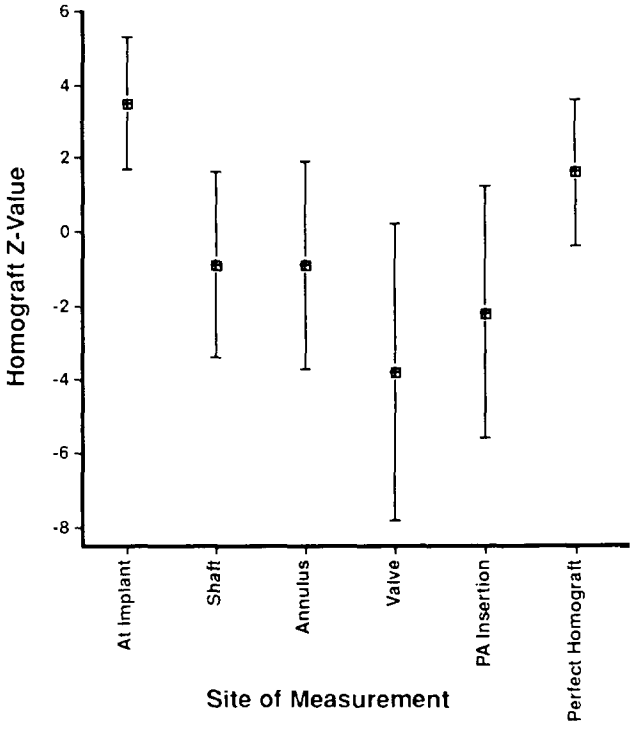

Fig 3. Changes in Z value (mean ± 1 standard deviation) of homograft at implantation and at the four different points of measurement. See Z values in Results section for definition of the "perfect" homograft. (PA = pulmonary artery.)

Conduit Longevity

The use of irradiated homograft conduits has been associated with rapid deterioration and development of significant obstruction [2–4, 10]. The deterioration of irradiated homograft conduits was reported to be faster and more severe than that observed with heterografts [3, 4, 10]. The use of cryopreserved, antibiotic-sterilized homograft conduits appeared to improve the durability of the conduits and reduce the need of replacement. The freedom from conduit-related reoperation for cryopreserved homograft conduits has been reported to be 55% to 75% at 5 years [6, 11] and 94% at 3.5 years [2]. Fresh homograft conduits appear to last longer and to have a replacement rate of only 13% at 10 years [5, 12]. An association between fresh homograft conduit obstruction and length of storage before implantation has been reported. Fresh conduits implanted within 21 days of harvest appear to have a lower risk of obstruction than those implanted after 3 weeks [13]. However, Bull and associates [14] reported no difference between fresh homograft and heterograft conduits in terms of survival or need of reoperation.

Of the 20 patients undergoing catheterization, significant obstruction was present in 80%, and 45% required conduit replacement. For all patients, the freedom from reoperation for a conduit-related obstruction was 74% at 76 months (including patients who did not have catheterization). These data are comparable with those reported by Cleveland and co-workers [6].

Conduit's Origin and Blood Type

Several studies [6, 15] have reported no difference in durability between homograft conduits of aortic or pulmonary origin. Our findings tend to support this notion, although we have only 2 patients with pulmonary homografts. In contrast, Heinemann and colleagues [16] reported improved longevity of pulmonary compared with aortic homografts used in the repair of truncus arteriosus in infants. Both of our patients with conduits of pulmonary origin had a significant gradient from the pulmonary ventricle to the pulmonary artery despite the absence of conduit calcification. This difference in calcification between pulmonary and aortic homografts has been reported in both human and animal studies and may be related to the thinner wall of the pulmonary homograft [6, 17–19]. Further, our data are similar to the data of others [17] in affirming the absence of a relation between stenosis or calcification and major blood type incompatibility.

Changes in Conduits

We are in agreement with Kay and Ross [5] that there is no relation between transconduit gradient and length of follow-up. There was a significant correlation between transconduit gradient and age at implantation. However, as in the study by Cleveland and associates [6], age at operation was not a significant factor affecting conduit replacement. This discrepancy is probably related to the fact that we did not have preset criteria for the timing of replacement.

Table 4. Comparison Between First and Second Catheterization Data

Variable	First Catheterization	Second Catheterization	*p* Value
Follow-up (mo)	19 ± 15	47 ± 18	<0.004
Systolic pressure of pulmonary ventricle (mm Hg)	73 ± 31	72 ± 19	0.9
Systolic pressure of PA (mm Hg)	33 ± 21	23 ± 13	0.25
Gradient (mm Hg)	41 ± 34	48 ± 19	0.35
Shaft CSA (mm^2)	134 ± 47	162 ± 74	0.27
Annulus CSA (mm^2)	171 ± 114	172 ± 109	0.95
Valve opening CSA (mm^2)	101 ± 82	56 ± 36	0.43
Insertion into PA CSA (mm^2)	122 ± 56	131 ± 70	0.46
Body surface area (m^2)	0.96 ± 0.53	1.20 ± 0.53	<0.003
Z value at shaft level	−1.2 ± 3.2	−1.1 ± 2.7	0.96
Z value at annulus level	−0.1 ± 4.9	−1.0 ± 3.9	0.51
Z value at valve opening level	−3.6 ± 5.0	−5.5 ± 1.9	0.37
Z value at insertion into PA	−1.7 ± 2.4	−2.4 ± 2.7	0.25

CSA = cross-sectional area; PA = pulmonary artery.

The sites of obstruction reported in the literature, in decreasing frequency, have been the distal anastomosis, the valve, and the prevalvar shaft [2, 3, 5, 11]. Although we were unable to determine consistently the major site of obstruction by pressure measurements, it appears from angiography that the valve opening and insertion site into the pulmonary artery are the most narrowed. The annulus and the shaft are also narrowed but not as severely.

Moreover, our data indicate that development of obstruction in children occurs because the conduits shrink and thus become relatively small for the patient's body size. The changes in Z value exceeded what would be expected from the growth of the children and no change in the conduit diameter. Instead of a mean Z value of 1.6 at catheterization in a "perfect" conduit without any interval changes, the point with the least amount of change, the annulus, was 0.9 standard deviation below the normal mean. The conduit diameters at the valve opening and the insertion into the pulmonary artery sites also were *below* what would be considered the normal range. Thus, there appears to be actual "shrinking" of the homografts with decrease in their functional lumen.

Decreases occurred at multiple levels, and no single site was specifically associated with producing the gradient, a finding that suggests that a diffuse rather than a localized process may be underlying the changes in the conduits. The McGoon ratios indicate the size of the pulmonary arteries in our patient population was within the normal range. Thus, the branch pulmonary arteries appear not to be the site of obstruction. The higher systolic pressure of the pulmonary ventricle in older patients may have been related to the greater cardiac output in these patients. Moreover, pressure is a function of the compliance of the receptive vessel, in this case the homograft conduit. Conduit fibrosis will reduce its distensibility and compliance, thus increasing ventricular pressure.

The intense hyalinization of the walls of the explanted conduits provides a clue to the pathogenesis of the obstruction. Viable donor cells have been found in tissue culture obtained from the wall of 71% of aortic and 57% of pulmonary conduits in lambs [19]. These cells may provide tissue stability to the conduit, but they may also trigger an immune response. The mild chronic inflammation observed in the explanted homografts may be evidence of chronic rejection. This proposed immunologic mechanism is supported by the work of Clarke and co-workers [20] and Jonas and associates [21], who demonstrated an increased incidence of wall calcification, lymphocyte infiltration, and intramural thrombi in cryopreserved versus fresh homografts in a lamb model. These mechanisms may be present in humans as well.

Serial Catheterizations

Kay and Ross [5] reported transconduit gradients of 12 ± 6 mm Hg and 24 ± 15 mm Hg after 1 year and 6 years of follow-up, respectively, for fresh homografts. Our data differ in that our angiographic and hemodynamic data are serial data from the same patients, whereas the second catheterizations in their report [5] were performed on different patients. Our data demonstrate that there was no significant increase in the gradient over the time studied when the same patients underwent recatheterization. Moreover, the changes observed at the last catheterization had been present at the earlier one. This observation is similar to that reported by Fontan and colleagues [12] in humans (the initial catheterization 7 months postoperatively and the second after 4.6 years) and by Jonas and co-workers [22] in experimental animals. The absence of any significant change in gradient, CSA, and Z value from the first to the second catheterization may be due to the fact that whatever obstruction was going to take place had already occurred before the first catheterization.

Study Limitations

The number of patients involved in this study is relatively small. The fact that only 4 patients had a "routine" cardiac catheterization may indicate that we preselected those patients with more severe obstruction. It would have been very informative to have angiographic information on all patients, regardless of signs or symptoms, after a short follow-up. This, however, does not change the basic findings of our study.

Conclusion

We have demonstrated that there was a significant decrease in the diameter and hence in the CSA and the Z value of homograft conduits in the pulmonary circulation when followed up longitudinally. These changes may underlie the hemodynamic compromise observed in these patients. Young age at implantation correlated significantly with magnitude of change. Changes occurred early after implantation and did not seem to change substantially thereafter. Immunologic and possibly hemodynamic mechanisms may contribute to these changes. Early routine cardiac catheterization may provide valuable information about conduit function.

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INVITED COMMENTARY

Salim and colleagues report the catheterization and angiographic findings in 20 children with homograft valve conduits. The children were a mean age of 4.7 years at the time of conduit insertion, and investigation was carried out a mean of 3.1 years after implantation. The mean gradient across the valve conduit was 47 mm Hg, and angiographic studies demonstrated a decrease in valve area from 216 mm^2 at implantation to 108 mm^2 measured at the valve orifice at follow-up. Salim and colleagues conclude that their data indicate "a. . .decrease, with time, in the functional lumen of homograft conduits."

Salim and colleagues have documented an important observation regarding the fate of homograft conduits. I agree with their inference that these observations have important implications in following up children with conduits. A current review of our experience at The Hospital for Sick Children includes 606 patients with pulmonary valve replacement. By multivariate analysis, younger age of the patient and smaller size of the valve conduit increase the risk of reoperation. The type of conduit is also important in determining the frequency of reoperation for conduit failure. The *aortic* homograft has a substantially higher failure rate than the *pulmonary* homograft. This is particularly obvious in conduits less than 20 mm in diameter and in children less than 5 years of age. Although it is important that 18 of the 20 conduits studied in the report by Salim and associates were aortic homografts, failure because of conduit stenosis occurred in the pulmonary homograft group. The latter do not have a significant survival advantage compared with porcine valve–Dacron conduits.

Salim and colleagues have highlighted an important clinical problem in children with congenital heart disease that emphasizes the need for an improved valve conduit.

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