JACC Vol. 24, No. 2
August 1994:483–9

Right Ventricular Outflow Reconstruction With Cryopreserved Homografts in Pediatric Patients: Intermediate-Term Follow-Up With Serial Echocardiographic Assessment

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Objectives. This study was performed to assess by echocardiography the intermediate-term outcome of cryopreserved homografts employed in pulmonary outflow reconstruction in children and to validate the reliability of Doppler echocardiography in their evaluation.

Background. Cryopreserved homografts have become the most widely used pulmonary conduits. Previous reports have shown the occurrence of homograft regurgitation in the immediate postoperative period and the propensity of regurgitation to progress. Although Doppler echocardiography has been useful in assessing extracardiac valved conduit stenosis, its reliability in assessing a large series of cryopreserved homografts has not been documented.

Methods. Echocardiograms of 41 patients (43 homografts) who underwent operations between December 1986 and October 1992 were retrospectively reviewed. The median age of patients at operation was 37.5 months (range 3 to 333), and the median duration of follow-up was 28.5 months (range 1 to 68). Homograft regurgitation was classified on a scale of 0 to 4+. Pressure gradients across the homografts measured in 23 catheterizations were correlated with corresponding echocardiographic gradients.

Results. Regurgitation: Homograft regurgitation occurred in 100% of patients at follow-up. Progression of severity >2 grades occurred during follow-up in 35% and was associated with operation before age 18 months ($p < 0.002$) and stenosis progression ($p < 0.05$) but not with homograft type (aortic or pulmonary). These data predict that 50% of patients operated on before 18 months of age will have severe regurgitation by 15 months postoperatively compared with only 15% operated on after 18 months. ***Stenosis:*** At follow-up, 51% of homografts had a stenotic gradient ≥25 mm Hg predominantly at the distal anastomosis, and stenosis progression was related to young age at operation (<18 months, $p < 0.005$) and small conduit size ($p < 0.01$). Fifty percent of conduits implanted before age 18 months could be predicted to stenose by 21.8 months compared with only 5% of those implanted after age 18 months. The gradient measured from Doppler echocardiography correlated well with the catheterization gradient ($r = 0.86$).

Conclusions. Cryopreserved homograft dysfunction is frequent and progressive. Young age at operation (<18 months) predicts more rapid deterioration. Doppler echocardiography is reliable in assessing the systolic gradients across homografts. Serial echocardiographic assessment in the follow-up of these patients accurately characterizes these problems.

(J Am Coll Cardiol 1994;24:483–9)

Cryopreserved homografts have become the most widely used pulmonary conduits in pulmonary outflow reconstruction in children. In a short-term follow-up report from Meliones et al. (1), ~65% of homografts showed regurgitation in the immediate postoperative period and 29% showed progression of regurgitation during a mean follow-up period of 15 months. This early development and progression of pulmonary regurgitation has important implications for the long-term performance of these cryopreserved homografts.

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Manuscript received July 1, 1993; revised manuscript received March 4, 1994, accepted March 11, 1994.

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Although Doppler echocardiography has been shown to be useful in assessing extracardiac valved conduit stenosis (2), the clinical utility and reliability of Doppler echocardiography in the long-term serial assessment of a large series of cryopreserved homografts has not been documented.

The purpose of the present study was 1) to serially assess the systolic and diastolic flow characteristics of cryopreserved homografts used in ventricular to pulmonary outflow reconstruction, utilizing echocardiographic examinations in the early and intermediate term follow-up, and 2) to correlate the flow characteristics of Doppler echocardiography and hemodynamic studies in those patients who had cardiac catheterization.

Methods

Patients studied. Since December 1986, we have exclusively utilized cryopreserved valved homografts (Cryolife,

0735-1097/94/$7.00

Table 1. Patient Characteristics and Status of Homografts at Last Follow-Up

Diagnosis	No. of Patients (no. of homografts)	No. of Homografts With Stenosis >25 mm Hg at Last Follow-Up	No. of Homografts With 3+ or 4+ Regurgitation at Last Follow-Up
Tetralogy of Fallot	13 (14)	11	3
Truncus arteriosus	10 (11)	6	7
D-transposition, pulmonary stenosis	5 (5)	3	1
L-transposition, ventricular inversion, pulmonary stenosis	5 (5)	1	2
Pulmonary atresia, ventricular septal defect	3 (3)	0	0
Pulmonary atresia, intact ventricular septum	2 (2)	1	1
Infective endocarditis	2 (2)	0	1
Critical pulmonary stenosis	1 (1)	0	0
Total	41 (43)	22	15

Inc.) for pulmonary outflow reconstruction at the South Carolina Children's Heart Center. Records of patients operated on from December 1986 to October 1992 and who had follow-up echocardiograms after the 1st month postoperatively were reviewed. All operations were performed by two surgeons (R.M.S., F.A.C.). The available echocardiographic and catheterization data of these patients were retrospectively reviewed and assessed independently of other clinical data by two observers (K.C.C., D.A.F.).

Two-dimensional echocardiographic examination. Patients were examined by two-dimensional, color flow and continuous wave Doppler echocardiography during the immediate postoperative period and at follow-up.

Doppler echocardiographic examination. Homografts were visualized and any stenotic sites were located by the two-dimensional imaging, color flow Doppler and pulsed Doppler studies. The velocity profiles of the homografts were quantitated from continuous wave Doppler studies. The highest peak flow velocities from pulmonary ventricles across the homografts to the distal pulmonary arteries were obtained by using a simplified Bernoulli equation: Gradient across the homograft (mm Hg) = Peak velocity $(m/s)^2 \times 4$.

Interpretation and definition of echocardiographic findings. *Stenosis.* A homograft was considered mildly stenotic if the Doppler gradient was 25 to 50 mm Hg and moderately stenotic if the gradient was >50 mm Hg.

Regurgitation. The severity of pulmonary homograft regurgitation was graded semiquantitatively by measuring the jet width in a manner analogous to that described for aortic regurgitation (3): 0 = absent; 1+ = a pinhole-like jet—very mild regurgitation; 2+ = a jet of approximately 20% of the homograft valve annular width—mild regurgitation; 3+ = a jet of approximately 40% of the annular width and with color flow reversal above and below the leaflets—moderate regurgitation; and 4+ = a broad-based jet of >40% of the annular width and with color flow reversal in the entire pulmonary artery—severe regurgitation.

Progression of stenosis and regurgitation. Progression of the homograft stenosis or regurgitation was quantitated as the difference between either the stenotic gradient or the regurgitation grade measured between the first and the last echocardiographic study (or the latest echocardiographic finding preceding any interventional procedure or operation). Progression of stenosis was classified as moderate if the difference between two studies was >20 mm Hg and severe if the stenosis progression was >40 mm Hg. Progression of regurgitation ≥2 grades was considered significant.

Cardiac catheterization. Indications for postoperative cardiac catheterization depended on the clinical suspicion or echocardiographic demonstration of homograft malfunction. The homograft gradient measured at cardiac catheterization was the difference between peak systolic pressure in the distal pulmonary arteries and the pulmonary ventricle during catheter pullback, using fluid-filled catheters, before any angiogram was taken. Only data from patients who had a corresponding echocardiogram within 24 h of cardiac catheterization were used for correlation.

Statistical analysis. Clinical variables that could affect the stenotic gradient (diagnosis, age at operation) and the homograft regurgitation status (diagnosis, concomitant stenosis, homograft leaflet abnormalities) at the last follow-up visit or the progression of stenosis (diagnosis, age at operation, homograft type) and the progression of regurgitation (diagnosis, age at operation, stenosis, concomitant stenosis progression, leaflet abnormalities) during the follow-up period were studied; the Pearson chi-square test or Fisher exact test, if required, was used for any correlation. A probability value <0.05 was considered statistically significant. Correlation between Doppler- and catheterization-obtained gradients was analyzed by linear regression analysis using the method of least squares.

Survival analysis. The probability of obtaining progression of stenosis >20 mm Hg or regurgitation of 3+ or 4+ for two age groups of patients (<18 months, ≥18 months) was estimated using the actuarial life table computation of the survivor function.

Results

Clinical characteristics of patients (Table 1). A total of forty-one patients (26 male, 15 female) were included. One patient with tetralogy of Fallot and one patient with truncus

Table 2. Characteristics of Patients With Homograft Leaflet Abnormalities

Pt No.	Diagnosis	Age at Operation (mo)	Leaflet Abnormality	Abnormality First Detected (mo postop)	Stenotic Gradient at Last Follow-Up (mm Hg)	Regurgitation Grade at Last Follow-Up	Homograft Type
1	PA, VSD	26.7	Prolapse	0.2	10	1+	Aortic
2a	Truncus arteriosus	5.2	Prolapse, noncoaptation	7.4	31	4+	Aortic
2b	Truncus arteriosus	17.6	Prolapse	2.6	65	4+	Pulmonary
3	Critical PS	7.5	Prolapse	4.5	17	1+	Pulmonary
4	ToF	103.4	Prolapse	4.7	40	4+	Pulmonary
5	ToF	37.5	Prolapse	24.5	29	2+	Pulmonary
6	ToF	172.3	Prolapse	37.3	28	1+	Pulmonary
7	L-TGA, PS	64.0	Prolapse	43.5	21	3+	Aortic
8	PA, VSD	57.5	Prolapse, noncoaptation	28.5	20	2+	Pulmonary
9	Infective endocarditis	152.0	Prolapse, noncoaptation	32.2	24	4+	Pulmonary
10	Truncus arteriosus	4.5	Fixed open	9.1	48	4+	Aortic
11	Truncus arteriosus	5.0	Dissolved	21.4	50	4+	Aortic

L-TGA = L-transposition of great arteries; PA = pulmonary atresia; postop = postoperative; PS = pulmonary stenosis; ToF = tetralogy of Fallot; VSD = ventricular septal defect.

arteriosus underwent reoperation to replace a malfunctioning homograft. The functional course of all 43 homografts was then reviewed. The cardiac malformations requiring homograft use in the 41 patients are shown in Table 1. Patient age at the time of operation was 3 to 333 months (median 37.5). The donor homograft types were aortic (n = 21) and pulmonary (n = 22); homograft diameter ranged from 12 to 26 mm. Patients were studied echocardiographically from 1 to 68 months (median 28.5) postoperatively. Four homografts were followed up to the time of balloon dilation procedures and two up to surgical replacement. The age of patients at last echocardiographic examination ranged from 8 to 334 months (median 73). A total of 221 echocardiographic studies were reviewed.

Conduit Regurgitation

Regurgitation. Homograft regurgitation was present in all 43 homografts (100%) at the last follow-up. Fifteen (35%) of the 43 homografts had moderate or severe regurgitation. The presence of regurgitation at follow-up was inversely related to the size of conduit implanted ($p < 0.01$). The occurrence of moderate or severe regurgitation was not related to the presence of stenosis >25 mm Hg as measured by Doppler gradient (7 of 17 vs. 15 of 26, $p > 0.05$). Homografts of patients with truncus arteriosus had a greater frequency of moderate or severe regurgitation than did those of the other patients (7 of 11 vs. 8 of 32, $p \leq 0.04$).

Regurgitation progression. Progression of the severity of regurgitation was observed in 74% (32 of 43) of homografts. Fifteen homografts (35%) had progression >2 grades during follow-up. This severe progression of regurgitation was associated with age at operation ≤18 months (10 of 15 vs. 5 of 28, $p \leq 0.002$) and stenosis progression >20 mm Hg (6 of 9 vs. 9 of 34 $p \leq 0.05$). Eleven (26%) of 43 homografts did not show progression of regurgitation. Three of the 43 homografts were observed to have aneurysmal dilation at late follow-up.

Leaflet abnormalities (Table 2). By the end of study period, leaflet abnormalities were visualized by two-dimensional echocardiograms in 12 (28%) of 43 homografts, with leaflet prolapse being the most common abnormality (Table 2). Leaflet abnormalities were detected from <1 to 43.5 months (median 15.2) postoperatively and were associated with moderate or severe regurgitation in 7 of 12 patients and with mild regurgitation in 5. In two of the five, the leaflet abnormalities were detected only within 1 month from the patient's last follow-up. There was no statistically significant relation between leaflet abnormality and type of homograft ($p = 0.56$), diagnostic category ($p = 0.26$) or age at operation ($p = 0.91$).

Prediction of regurgitation. Data were analyzed and a prediction made of the likelihood that conduits would be free from severe 3+ to 4+ regurgitation (survival without regurgitation). Figure 1 shows that 50% of conduits would have severe regurgitation by 15.3 months postoperatively if implanted in patients <18 months of age. When homografts were implanted in patients >18 months of age, the predicted homograft survival was 85% without severe regurgitation at this same follow-up interval.

Conduit Stenosis

Stenosis (Table 3). At the last follow-up, 22 (51%) of 43 homografts were stenotic with a systolic gradient ≥25 mm Hg (Table 3). Five of these 22 (5 [12%] of 43) had a systolic gradient >50 mm Hg. The sites of stenosis were distal in 18, distal and proximal in 3 and proximal in 1. Diagnostic categories were not related to the final stenosis status ($p = 0.21$).

Stenosis progression (Table 3). Nine (21%) of 43 homografts had stenosis progression >20 mm Hg, including 2

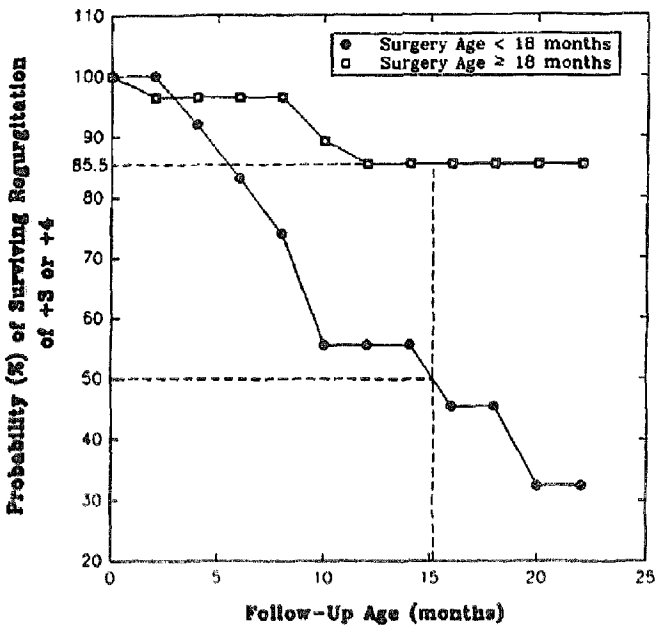

Figure 1. Plot of survival function of regurgitation of 3+ or 4+ severity.

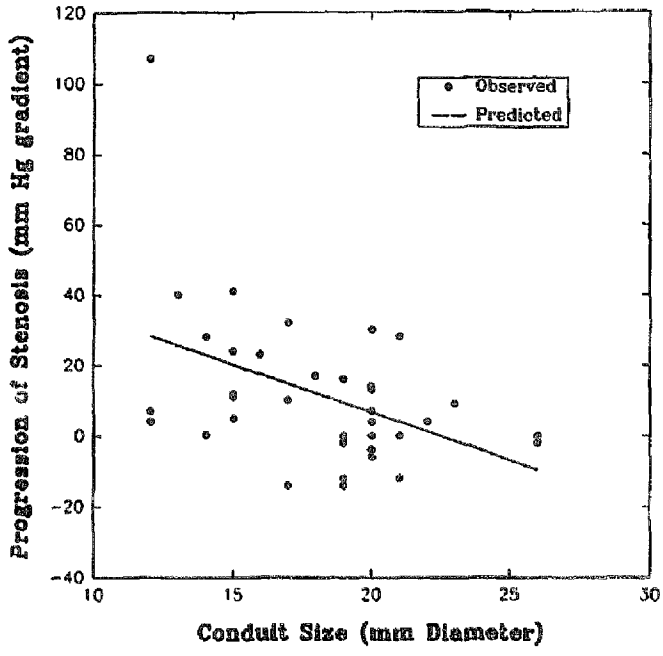

Figure 2. Plot of progression of conduit stenosis as a function of conduit size.

(5%) of 43 with severe progression >40 mm Hg (Table 3). Stenosis progression was inversely related to the age at operation, most noticeable for homografts implanted ≤18 months (7 of 15 vs. 2 of 28, $p \leq 0.005$). Stenosis progression was not related to diagnosis ($p = 0.14$) or type of homograft ($p = 0.65$). Conduit size was negatively correlated with progression of stenosis ($p < 0.01$) (Fig. 2).

Prediction of stenosis. A prediction of the likelihood that conduits would survive without a progression of 20 mm Hg

Table 3. Characteristics of Patients With Homograft Stenosis and Progression of Stenosis and Regurgitation

Pt No.	Diagnosis	Age at Operation (mo)	Time Since Operation (mo)	Stenotic Site	Gradient at Last FU (mm Hg)	Regurgitation at Last FU	Homograft Type
12	ToF	92	57	Distal	26	1+	Aortic
6	ToF	172.3	37.3	Distal	28	1+	Pulmonary
5	ToF	37.5	24.5	Distal	29	2+	Pulmonary
13	ToF	217.6	12.4	Distal	30	1+	Pulmonary
2a	Truncus arteriosus	5.2	6.2	Distal	31	4+	Aortic
2b*	Truncus arteriosus	17.6	12.4	Distal	65 (24)†	4+ (2+)‡	Pulmonary
14	ToF	125.8	38.2	Distal	31	2+	Aortic
15	PA, IVS	12.9	60	Distal	32	1+	Aortic
16	D-TGA, PS	42	28	Distal	32	1+	Aortic
17	L-TGA, PS	32	14	Distal	32	3+	Aortic
18*	D-TGA, PS	9.7	14.4	Distal	33 (23)†	1+ (1+)‡	Aortic
19	ToF	154	10	Distal	34	1+	Pulmonary
20*	ToF	16	27	Distal	38 (32)†	2+	Pulmonary
4*	ToF	103.4	45.6	Prox, distal	40 (30)†	4+ (3+)‡	Pulmonary
21*	ToF	58.2	45	Distal	44 (28)†	1+ (1+)‡	Aortic
22a	ToF	31.3	10.5	Distal	60	4+	Pulmonary
22b	ToF	43	37	Distal	45	4+	Pulmonary
10	Truncus arteriosus	4.5	57	Distal	48	4+	Aortic
11*	Truncus arteriosus	5	58	Distal	50 (40)†	4+ (4+)‡	Aortic
23*	Truncus arteriosus	8	26	Prox, distal	60 (41)†	2+ (2+)‡	Pulmonary
24*	D-TGA, PS	10	24	Prox	65 (28)†	3+ (3+)‡	Aortic
25*	Truncus arteriosus	3	13	Prox, distal	117 (107)†	4+ (3+)‡	Aortic

*Patients with significant stenosis and regurgitation progression (mm Hg). †Increase in gradient from initial examination. ‡Increase in grade of regurgitation from initial examination. D-TGA = D-transposition of great arteries; FU = follow-up; IVS = intact ventricular septum; Prox = proximal; other abbreviations as in Table 2.

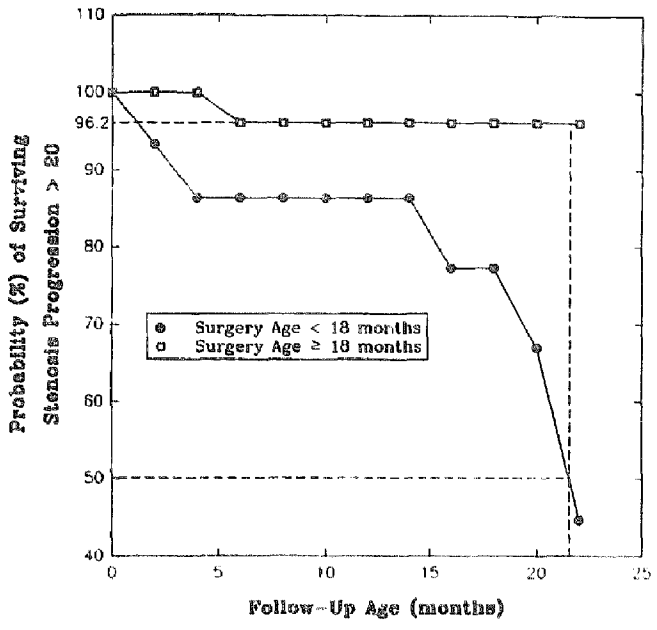

Figure 3. Plot of survival function of conduit stenosis progression >20 mm Hg, as measured by Doppler gradient.

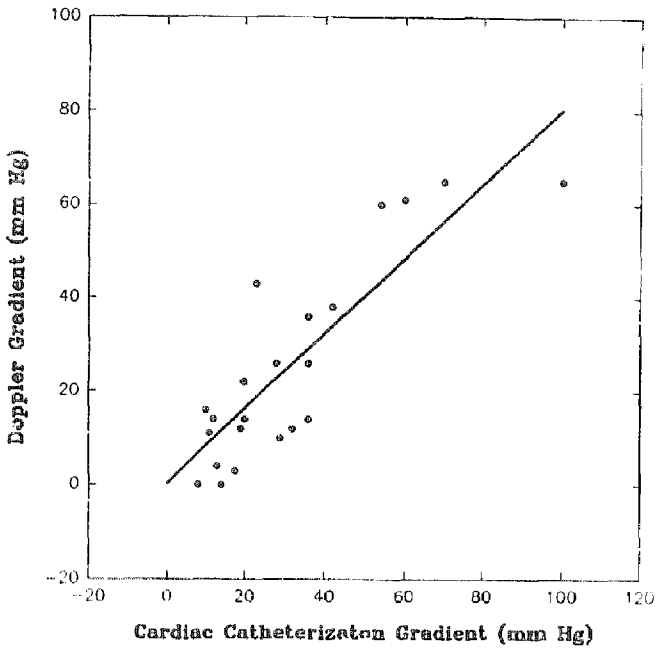

Figure 4. Plot correlating homograft systolic gradients measured by catheter pullback and continuous wave Doppler echocardiography (n = 23, r = 0.86, y = 0.2807 + 0.8015x).

stenosis gradient was performed. If conduits were implanted in patients before age 18 months, 50% would have progression of 20 mm Hg stenosis by 21.8 months postoperatively (Fig. 3). Conduits implanted after this age can be predicted to have far less progression, with >95% being free from progression of stenosis by the same time.

Conduit types. Both aortic and pulmonary conduits were used for repair. No differences in development or progression of either homograft valve regurgitation ($p = 0.83$) or stenosis were seen between the two types of conduit at any age. No preferential use of either type of homograft was apparent for any particular type of operation or at any specific age.

Doppler echocardiographic and catheterization gradient correlation (Fig. 4). Of the 41 patients, 25 had 26 postoperative cardiac catheterizations; 1 patient underwent cardiac catheterization twice 7 months apart for balloon angioplasty procedures. Three patients are excluded; in two, echocardiographic examination was performed >24 h before cardiac catheterization and in the third, the homograft could not be entered by catheter. The data from the remaining 23 cardiac catheterizations were correlated with Doppler studies. The time interval between cardiac catheterization and operation ranged from 3 to 58 months (median 14.5). Patient age at the time of the study ranged from 8 to 248 months (median 55). Sixteen (70%) of 23 patients underwent echocardiographic examination under sedation immediately before cardiac catheterization. The correlation coefficient between maximal conduit gradient measured by Doppler study and cardiac catheterization was 0.86. The regression line followed the relation: Doppler gradient calculated (mm Hg) = 0.2807 + 0.8015 (Catheterization pressure gradient).

Subsequent clinical course of patients. Two (5%) of the 41 patients had a homograft replaced. One patient with severe tetralogy of Fallot had a homograft replaced because of significant obstruction and regurgitation 11.5 months after the first operation. The other patient, with truncus arteriosus, had the second homograft implanted 12.4 months after the initial operation because of severe regurgitation and pulmonary ventricle dysfunction. She died suddenly at home 4 months after the second operation with no significant illness immediately before death. No autopsy was performed. Four patients underwent a total of six balloon angioplasty and one pulmonary endovascular stent procedures for distal pulmonary stenosis. Indications for pulmonary artery angioplasty and definition of success have previously been reported (4). Successful relief of the stenosis was observed only in the patient in whom a pulmonary stent was employed. That particular patient had initial progression of regurgitation (1+ to 4+) in the presence of stenosis (gradient 45 mm Hg) with later reduction in regurgitation from 4+ to 2+ after successful bilateral branch pulmonary artery stenting. There were no episodes of infective endocarditis or thromboembolism throughout the follow-up period.

Discussion

Cryopreserved homografts are advantageous in the reconstruction of ventricular to pulmonary connections because they are rarely associated with thromboembolic events or infective endocarditis and have high surgical versatility. Although work of O'Brien et al. (5) suggested

that the viable components of cryopreserved homografts could enhance their long-term survival over that of other conduits, previous data (6) revealed the problem of late tissue degeneration and homograft dysfunction.

Homograft stenosis. In a follow-up study by Kay and Ross (7) of patients receiving fresh, antibiotic sterilized aortic homografts, only 13% (three patients) were found to have a gradient >50 mm Hg requiring conduit replacement by 10 years. The obstruction in these patients was due to neointimal hyperplasia in the Dacron extension tube portion rather than the homograft itself. In the present study, although a similar proportion of patients (12%) had a gradient >50 mm Hg, the obstruction occurred much earlier and in the absence of any Dacron extension. In the study by Meliones et al. (1) in which cryopreserved homografts were followed up for a mean of 15 months, only 5% had severe conduit stenosis. The apparent difference could be due to the greater proportion of patients in our study who underwent homograft implantation before 24 months of age (39% vs. 19%) as well as the longer follow-up period. Age at operation was related to degree of subsequent stenosis in our study as well as in previous studies (8,9). Our finding that stenosis in homografts occurred most commonly in the distal anastomotic sites is consistent with previous reports (1,8,9). The preliminary favorable outcome of pulmonary artery stenting procedure in relieving distal stenosis is encouraging. However, finding ways to decrease the incidence of distal stenosis, especially in infants requiring early pulmonary outflow reconstruction surgery, would be desirable.

Homograft stenosis has been suggested to be related to the relative small size of homograft used (1). The relation between the age of our patients and the diameter of the implanted homografts was not much different from that reported by others (1,9).

Homograft stenosis progression. In the study by Kay and Ross (7) of fresh aortic homografts, no relation was present between homograft stenotic gradient and the timing of postoperative cardiac catheterization. Fontan et al. (10) reported that only one of seven patients with a fresh aortic homograft had progression of systolic gradients shown at recatheterization. In contrast, our present findings document the progression of systolic gradient because some patients had no stenotic gradient initially. The apparent difference between studies could be accounted for by the longitudinal cohort study nature of the present study and its universal follow-up of patients.

Homograft regurgitation and regurgitation progression. Cryopreserved homografts in the pulmonary position have been found to develop regurgitation in short-term follow-up studies. The data presented here allow prediction that severe homograft regurgitation is likely if the device is implanted in patients <18 months of age. In biosynthetic valve studies, structural degeneration of valves has been shown to occur with time and may be accelerated in young, active patients, perhaps as a result of faster sustained heart rates (11–13). It is therefore possible that in the present study involving predominantly younger children, the degree of homograft tissue degeneration was greater, thus leading to regurgitation with time. Clarke et al. (14), reporting on cryopreserved aortic homografts used in aortic root replacement procedures, also found a significantly higher prevalence of homograft degeneration in patients <3 years of age at operation. Immunologically mediated host response to the homograft leading to degeneration was suggested and empiric immunosuppressive treatment was attempted in that study. Although the exact etiology is still not certain, leaflet abnormalities have been reported as the underlying cause of replacement of cryopreserved homografts (15).

Significant regurgitation was shown to be related to the stenosis across the homograft in previous studies (1). However, our study demonstrated in addition an association between the progression of regurgitation and the concomitant progression of stenosis with time. This finding is also reflected by the observation that in one patient, the homograft regurgitation initially deteriorated with time in the presence of stenosis but subsequently lessened after relief of the distal pulmonary stenosis. Early diagnosis of significant stenosis was therefore important and relief of distal stenosis could arrest homograft dysfunction.

Echocardiographic examination and catheterization correlation. Doppler studies of instantaneous maximal gradient in our study correlated satisfactorily with the peak to peak gradient measured at cardiac catheterization. In one patient whose homograft could not be entered because of its abnormal position, the systolic gradient across the homograft thus could be assessed only by echocardiography. The present study therefore highlights the validity of echocardiography in the serial follow-up of these patients.

Echocardiographic and catheterization studies differed in time and in the degree of sedation and state of consciousness of the patients, both of which can alter gradient measurement. However, owing to the unusual location of the homografts, various maneuvers sometimes were required to obtain an adequate echocardiographic window, and studies were not exactly simultaneous. We believe that measurement of the Doppler gradients within 24 h of or immediately before cardiac catheterization is a practical alternative.

No attempt was made to correlate the echocardiographic grading of homograft regurgitation and catheterization findings, because, in our experience, angiographic grading of pulmonary insufficiency is insensitive and difficult to quantify.

Conclusions. Malfunction of cryopreserved homografts is common and dysfunction is progressive in the intermediate term, and when these conditions are more severe the devices are implanted in younger children. Serial echocardiographic examinations detect leaflet abnormalities and reliably assess homograft dysfunction in these patients. The present findings in this homogeneous patient group with cryopreserved homografts clearly indicate that use of such homografts in pulmonary outflow reconstruction does not provide permanent palliation. Optimal methods to improve the viability of homograft valves, as well as ways to reduce the stenotic

complications, especially of those homografts implanted in early infancy, are thus highly desirable.

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