Results of Heart Valve Homograft Implantation in a Major Referral Center in Iran

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Abstract

Background- **Heart Valve** homografts have been used in reconstructions of right and left ventricular outflow tract (RVOT and LVOT) for nearly 50 years now with varying results.

Methods- The outcome of homograft implantation was analyzed in 101 patients who received 108 cryopreserved homografts for the reconstruction of RVOT and LVOT between April 1993 and March 2003.

Results- 88.2% aortic valve and 11.7% pulmonic valve homografts were used. Median age at implantation was 10.0 years (Mean: 13.1 ± 10.6 years, range: 5 months to 57 years). Endpoints included: (1) patient survival, (2) homograft failure (valve explant or late death) and (3) homograft dysfunction (homograft insufficiency or homograft stenosis). Mean follow-up duration was 2.9 ± 2.4 years. There were 11 homograft dysfunctions requiring reoperation with the mean longevity of 4.4 ± 2.3 years. We had only one late death due to congestive heart failure (CHF), and all the other deaths (23.7% of the patients) occurred perioperatively. The quality of life of most of the survivors is good.

Conclusion- Early and mid-term results of homograft implantation are good, but long-term results remain to be investigated.

Keywords: Homograft ■ Congenital Heart Surgery

An allograft (allogenic graft) (formerly "homograft", Fig. 1) is a transplant between two genetically different members of the same species.[1] The first clinical use of homograft tissue in cardiovascular surgery was in 1948, when Gross used cadaveric arterial grafts to construct systemic to pulmonary artery shunts in patients with tetralogy of Fallot and to repair coarctation of the aorta. Eighteen years later, a valved homograft was used for the first time in the treatment of congenital heart disease for the reconstruction of the right ventricular outflow tract (RVOT) in a child with pulmonary atresia. Since these pioneering advances, valved and vascular homografts have become central to the management of congenital anomalies of the heart and great vessels.[2] The most important advantages of allografts are the excellent hemodynamic qualities and the low risk of endocarditis. Anticoagulation is not necessary because there is no risk for thromboembolism or hemolysis.[3] The primary use for homografts in congenital heart surgery today is the establishment of a valved connection between the right ventricle and pulmonary arteries in children[2] when there is hypoplasia of RVOT or pulmonary arteries[4] such as tetralogy of Fallot with pulmonary atresia or other complicating factors, truncus arteriosus, transposition complexes and double outlet right ventricle (DORV).[2] Valved homografts, initially introduced in 1966 by Ross and Somerville, have become the most commonly used valved conduit for the

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reconstruction of the RVOT.[4] The Rastelli operation, first performed in 1968, was developed for repair of transposition of the great arteries with associated ventricular septal defect and severe pulmonary stenosis. This operation includes the placement of an intracardiac baffle to direct left ventricular blood to the aorta and an extracardiac valved conduit to establish continuity between the right ventricle and the pulmonary arteries.[5] The postoperative right heart pressures may be elevated. Valved conduits are also used for the management of right ventricular failure caused by pulmonary insufficiency and ventricular arrhythmias after initial non-valved repair.[4]

Homograft reconstruction of the LVOT has also been performed for many years in children with aortic insufficiency (AI) or recurrent aortic stenosis (AS).[2] The Ross procedure aortic valve replacement (AVR) with pulmonary autograft and pulmonary homograft replacement of the pulmonary valve developed as a method of choice, and durable aortic valve substitute avoids the need for anticoagulation and provides young patients with a long-lasting aortic valve substitute.[6,7]

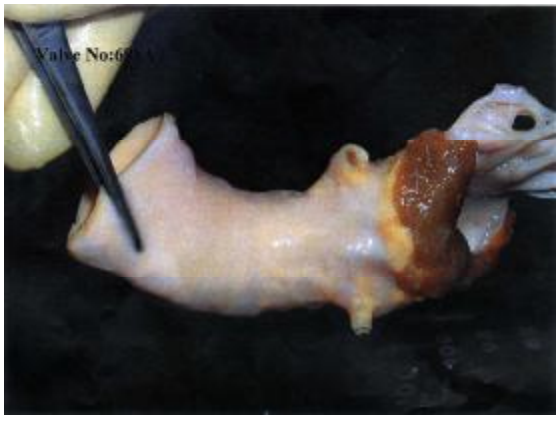

Fig. 1: A sample of an aortic homograft.

The purpose of this study was to evaluate the consequences of using homografts in the reconstruction of the RVOT or LVOT in congenital heart diseases.

Methods

From April 1993 through March 2003, 101 patients with congenital, rheumatic or infective heart diseases underwent surgery with the use of cryopreserved homograft valves at our department in Tehran, Iran. Almost all of the patients had undergone catheterization for their congenital heart diseases before the homograft implantation. Medical records and clinical charts were reviewed for all homograft recipients. Data collected from the operative admission included diagnosis, previous operative procedures, age, sex and weight at the time of surgery. Previous operative procedures were defined as palliative or corrective, for example, repair of tetralogy of Fallot with a transannular patch was considered a corrective procedure; whereas a systemic-to-pulmonary artery shunt was considered palliative. Patients' follow-up was obtained from hospital and clinic visit records. Follow-up information was available within the past calendar year for 29 patients (28.7%) who had no early or perioperative deaths. Mean follow-up duration was 2.9 ± 2.4 years (2 months to 10 years) with the median of 2.5 years, respectively.

Patients' characteristics including diagnostic category, previous and subsequent procedures, age at initial homograft replacement and subsequent operations are summarized in Table I and III.

Homograft failure was defined as the explant of the valve for any reason or late death (occurring > 30 days after surgery) as the result of any cause; against early death (occurring < 30 days after surgery). Homograft dysfunction was defined as any of the following: moderate to severe stenosis or insufficiency as well as explant

or late death. Homograft insufficiency was defined echocardiographically as moderate when there was a broad regurgitant jet of less than the annulus width associated with diastolic color Doppler flow reversal from the distal main pulmonary artery. A regurgitant jet that encompassed the entire annulus width associated with diastolic flow reversal in the branch pulmonary arteries was graded as severe. Homograft stenosis was defined as a transvalvular peak instantaneous pressure gradient > 40 mmHg. The follow-up echo in which the patient first met one or both of these dysfunction criteria was recorded as the duration of functional valve life.

Descriptive data are presented as mean values±1 SD, median and range. All analyses were performed with SPSS version 11.0 software.

Results

There were 37 (36.6%) females and 64 (53.4%) males.

The patients' diagnoses were categorized into nine groups (Table I and diagram 1).

Table I: Diagnostic category of homograft recipients.

Diagnosis	Frequencies
TF/DORV, VSD, PS	28.7% (n=29)
TGA,VSD,PS /LTGA	38.6% (n=39)
PA,VSD	7.0% (n=7)
PA/ PS, IVS	1.0% (n=1)
TF, APVS	1.0% (n=1)
Truncus Arteriosus	2.0% (n=2)
AS/AI (CHDorRHD)	13.9% (n=14)
AS/AI(Primary Endocarditis)	5.9% (n=6)
VSD,AI	1.1% (n=1)

TF=Tetralogy of Fallot; **DORV**=Double Outlet Right Ventricle; **VSD**=Ventricular Septal Defect; **PS**=Pulmonary Stenosis; **TGA**=Transposition of Great Arteries; **LTGA**=L-Transposition of Great Arteries; **PA**=Pulmonary Atresia; **IVS**=Intact Ventricular Septum; **APVS**=Absent Pulmonary Valve Syndrome; **AS**=Aortic Stenosis; **AI**=Aortic Insufficiency; **CHD**=Congenital Heart Disease; **RHD**=Rheumatic Heart Disease.

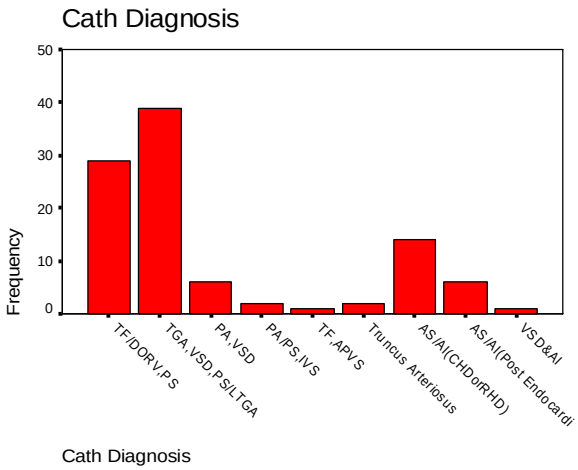

Fig. 1: Patients' diagnoses and frequencies.

Ninety-five patients (94.1%) had catheterization before their first homograft implantation. Seven patients had their second implantations, in 6 of whom catheterization was done.

Mean RV pressure before the first homograft implantation was 93.7 ± 30.3, with a median of 100.0 and a range of 16 to 140 mmHg. Mean RV pressure before the second homograft implantation was 121.6 ± 38.6 mmHg, with a median of 105.0 and a range of 80 to 170 mmHg.

The patients had 1 to 5 surgical procedures overall with the mean and median of 2 times. They had 0 to 3 previous and 0 to 2 subsequent surgical operations (Table II).

Fifty of the patients had no previous surgery. Their diagnoses are summarized in Table II. Fifty-one patients had 1 to 3 previous surgical operations, 18 of whom had 1 to 3 previous corrective ones and 33 had 1 to 2 previous palliative ones. Their diagnoses are summarized in Table III.

Mean age at the first homograft implantation was 13.1 ± 10.6 years with a median of 10.0 years and a range of 5 months to 57 years. Mean age at the second homograft implantation was 14.4 ± 2.8 years with a median of 15.0 and a range of 9.5 to 17.5 years.

Mean weight at the first homograft implantation was 29.5 ± 17.3 with a

median of 21.0 and a range of 8.5 to 95.0 kilograms. Mean weight at the second homograft implantation was 37.9 ± 22.1 kg with a median of 32.0 and a range of 15.5 to 78 kilograms.

Table II: Diagnostic category of non-surgery recipients.

Primary Diagnosis	Previous Surgery
TF/DORV, VSD, PS	20% (n=10)
TGA,VSD,PS /LTGA	36% (n=18)
PA,VSD	6% (n=3)
PA/ PS,IVS	0% (n=0)
TF, APVS	2% (n=1)
Truncus Arteriosus	4% (n=2)
AS/AI (CHDorRHD)	18% (n=9)
AS/AI(Primary Endocarditis)	12% (n=6)
VSD,AI	0% (n=0)

Causes of surgery for the first homograft implantation were: correction of the anatomy due to CHD (70 pts, 69.3%) and RHD (7 pts, 6.9%), correction of residual problems after previous corrective surgical operations (18 pts, 17.8%), and surgical debridement and reconstruction after endocarditis (6 pts, 5.9%). The residual problems remaining from previous corrective surgical operations were residual PS (5 pts, 5.0%), pulmonary insufficiency (PI) (3 pts, 3.0%), AS (4 pts, 4.0%), AI (1 pt, 1.0%), endocarditis (2 pts (2.0%) and RVOT aneurysmal pouch (2 pts, 2.0%).

All subsequent surgical operations (21 pts, 20.8%) were due to residual problems, in which 2 operations were palliative (one for pericardial effusion drainage and the other for permanent pacemaker implantation). The other operations on 19 patients were corrective due to homograft dysfunction, the diagnoses of which were:

- TF/DORV, VSD, PS (2 pts, 10.5%);
- TGA, VSD-PS/LTGA (11 pts, 57.9%);
- PA / PS-IVS (1 pt, 5.3%);
- AS / AI (CHD or RHD) (1 pts, 5.3%);

Table III: Diagnostic categories of RVOT homograft recipients, previous procedures, initial homograft implantation and subsequent operative characteristics.

Primary Diagnosis	Previous Non-homograft Procedure Palliative/Corrective	Subsequent Corrective Procedure Homograft Replacement/ Homograft Reconstruction	Age at Initial Homograft (y)	Age at Subsequent Corrective Surgery (y)
TF / DORV, VSD, PS	31% (n=9) / 34% (n=10)	0% (n=0)/ 7% (n=2)	11.8 (4-30)	11.7 (10.5-13)
TGA,VSD, PS / LTGA	51% (n=20) / 2% (n=1)	(2 palliatives) 13% (n=5)/ 15% (n=6)	9.2 (3.5-20.5)	12.5 (6.0-16.5)
PA,VSD	57% (n=4) / 0% (n=0)	-	11.6 (6.0-18.0)	-
PA / PS, IVS	0% (n=0) / 10% (n=1)	100% (n=1)/ 0% (n=0)	15.5	17.5
TF, APVS	0% (n=0) / 0% (n=0)	-	3.0	-
Truncus Arteriosus	0% (n=0) / 0% (n=0)	-	1 (0.4-1.5)	-
AS / AI (CHDorRHD)	0% (n=0) / 35% (n=5)	0% (n=0)/ 7% (n=1)	22.0 (3.5-57.0)	28.5
AS / AI (Primary Endocarditis)	0% (n=0) / 0% (n=0)	0% (n=0)/ 33% (n=2)	32.5 (16.0-50.0)	38.5 (26.5-50.5)
VSD, AI	0% (n=0) / 100% (n=1)	0% (n=0)/ 100% (n=1)	12.0	16.5

- AS / AI (Primary endocarditis) (2 Pts, 10.5%);
- VSD & AI (1 Pt, 5.3%).

Their mean age at surgery was 15.7 ± 9.6 years with a median and a range of 6.0 to 50.5 years. Their mean weight at the surgery was 35.8 ± 17.4 kg with a median of 32.0 and a range of 14.0 to 78.0 kilograms. Their mean RV pressure before the surgery was 87.5 ± 37.4 with a median of 85.0 and a range of 28 to 170 mmHg. Their corrective surgical operations were due to residual problems, which were homograft stenosis in 7 patients, residual VSD in 3 patients, AS in 2 patients, AI in 2 patients, homograft calcification in 2 patients, homograft aneurysm in 1 patient, endocarditis in 1 patient and mitral regurgitation (MR) in 1 patient. The last patient had a repeated surgical correction as well. Seven of these 21 patients had their second homograft implantation, and the cause of their surgery was homograft stenosis (5 pts, 71.4%), homograft calcification (1 pt, 14.3%) and endocarditis (1 pt, 14.3). Their diagnoses were TGA, VSD-PS / LTGA (5 pts, 71.4%) and PA / PS-IVS (1 pt, 14.3%). So homograft

dysfunction occurred in only 11 of the 19 corrective reoperations which led to surgery (57.8% of the corrective reoperations and 10.8% of all the patients) with mean homograft longevity of 4.4 ± 2.3 years, median of 4 and range of 2 to 8 years. Their diagnoses were TGA, VSD-PS/LTGA (8 pts, 72.7%), TF/DORV, VSD-PS (1 pt. 9.1%) and PA/PS-IVS (9 pts, 9.1%). Their homograft types were aortic (7 pts, 63.6%), pulmonic (1 pt, 9.1%) and 3 unknown.

The earliest conduit replacement was performed 2 years after the initial homograft placement as part of a revision of pulmonary artery reconstruction in a patient with severe PS, who had also undergone two corrective surgical operations before her first homograft implantation. Her conduit is now in place 4 years after implantation.

Of 108 homograft implantations in 101 patients, 90 (83.3%) of the homografts were aortic, 12 (11.1%) were pulmonic and 6 were unknown. All of the second homografts were aortic. The size of homografts were 12 to 27mm with a mean of 20.9 ± 2.8 and a median of 21mm. Aortic homograft sizes were 12 to 27mm with a mean of 20.7 ± 2.8 and a median of 21mm. Pulmonic homograft sizes were 17 to 25mm with a mean of 21.7 ± 2.5 and a median of 23mm.

Duration of intensive care unit (ICU) stay after 108 homograft implantations ranged from about 3 to 37 days with a mean of 6.7 ± 5.9 days and a median of 4 days.

After 108 homograft implantations, there were no complications in 82 patients (75.9%). Eight patients (7.4%) had arrhythmias, 7 of whom had complete heart block (CHB), which led to PPM implantation, but one of them had junctional rhythm, which improved after 6 days.

The other complication was death. We had 18 patients (16.7%) who died after the first homograft implantation: 6 females and 12 males. Seven patients died in the operating room (OR) and 11 patients in the ICU. Time of death in the ICU was 4.2 ± 5.7 days with a range of 0 to 20 days, median of 2 days and mode of 0. Their diagnoses were TF/DORV, VSD-PS (5 pts, 27.8%); TGA, VSD-PS/LTGA (4 pts, 22.2%); PA, VSD (1 pt, 5.6%); truncus arteriosus (1 pt, 5.6%); AS / AI (CHD or RHD) (4 pts, 22.2%) and AS / AI (post endocarditis) (2 pts, 11.1%). Their surgical operations were for the correction of anatomy because of their congenital heart disease (12 pts, 66.7%); correction of anatomy because of their residual problem after their previous surgery (4 pts, 22.2%); and debridement of vegetation and correction of anatomy (2 pts, 1.1%). Their residual problems for which they were operated were PS (1 pt, 5.6%) and AS (1 pt, 5.6%), both in 2 patients for their first homograft implantation; homograft calcification (1 pt, 5.6%) and added endocarditis (1 pts, 5.6%). Fifteen of the 18 deaths had received aortic homografts (83.3%), and 3 had pulmonic homografts (16.7%). The size of homografts were 12 to 27mm with a mean of 20.0 ± 3.7 and a median of 20.5mm. Their mean RV pressure before surgery was 94.6 ± 50.5 with a median of 107.5 and a range of 16 to 170 mmHg. Their age at the time of surgery was 14.8 ± 11.5 with a median of 11.7 years and a range of 5 months to 48 years. The cause of death was inability to wean from cardiopulmonary bypass (5 pts, 27.8%); cardiopulmonary arrest (5 pts, 27.8%); arrhythmias (1 pt, 5.6%); ATN (2 pts, 11.1%); infection (1 pt, 5.6%); hemorrhage (1 pt, 5.6%); acute tubular necrosis (ATN) and infection (1 pt, 5.6%); and endocarditis (2 pts, 11.1%).

Five of the 14 corrective non-homograft operations (following previous homograft implants) led to death. Their deaths were due to inability to wean from cardiopulmonary bypass in one and

cardiopulmonary arrest in ICU in 4 after 0 to 8 days with a mean of 3.6 ± 3.2 days and a median of 2 days.

We had only one late death that had severe conduit calcification after 3 years of operation, leading to CHF. Due to neurological complications following cardiopulmonary arrest during his last catheterization, he could not be operated on again and eventually died with the diagnosis of CHF in cardiac care unit (CCU). Two early deaths occurred in ICU after the second homograft implantations: one because of ATN 4 days after surgery and the other because of cardiopulmonary arrest after transfer from OR to the ICU.

At the time of discharge from hospital and after homograft implantation, all of the patients (95.0%) had normal sinus rhythm (NSR) except 5 of them (5.0%) who had CHB and received PPM. One of the patients (1.0%), who was in NSR, developed CHB after one month and received PPM too. Of the patients without CHB (and no PPM), 79.4% had right bundle branch block (RBBB), 8.8% had normal axis and 2.9% had left axis deviation (LAD) and had AS / AI. Eleven of the 101 patients (11.6%) displayed homograft calcification (Fig. 2), the earliest occurring 2 years after the operation. Three of them were reoperated on, two of whom due to homograft stenosis.

One patient, whose repeat surgery was done after 4 years without replacement of the homograft, had corrective surgery using Dacron patch and is now well after 3 years.

The other patient, whose repeat surgery was done after 7 years, received his second homograft implantation to replace the previous one, but he died in ICU after 4 days because of repeated bleeding and inability to control his hemodynamic condition and ATN.

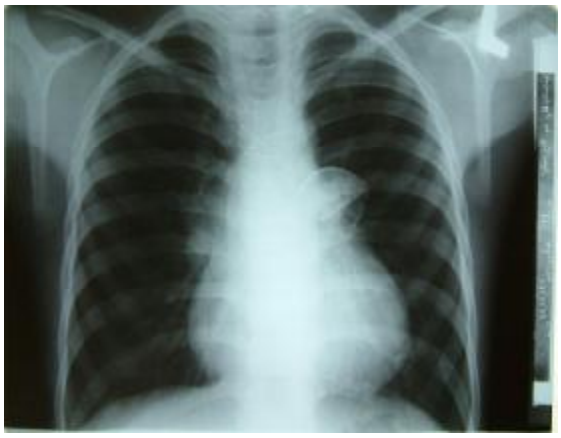

Fig. 2. Homograft calcification in a patient two years after implantation.

All surviving patients were discharged from the hospital in class I (of NYHA classification of heart failure) except 3 of them who were class II. After one year, 9 of them became class II. And after 2 years 13 became class II, and 4 became class III.

Mean follow-up duration was 2.9 ± 2.4 with a median of 2.5 years and a range of 2 months to 10 years. We have 3 patients followed up to 10 years whose classes are I in one patient and II in the other two patients. These patients had only one surgical opertaion about 10 years ago. Their diagnoses were PA/ VSD in 2 of them and TGA/ VSD - PS in the other.

We concluded that there was no significant difference between the size of the homografts and their mean value of right ventricular (RV) pressure before the procedure (P-value > 0.05). But the relationship between the size of the homografts with the patient's age and weight had a significant difference (P-value < 0.005). As a result, the greater the age or weight, the larger the size of the homograft used. In addition, the mean value of RV pressure was compared with the duration of discharge of the patient from the ICU (which reflects hemodynamic stability of the patient). We had no significant differences between them (P-value > 0.05). There was no significant difference between discharge

from the ICU and the age or weight or size of the homograft for the patient (P-value > 0.05). We had significant differences between the mean value of RV pressure and the patient's age and weight (P-value < 0.01). Consequently, the greater the age or weight, the higher the RV pressure of the patient.

Discussion

Homografts were the initial valved conduit used for the reconstruction of the RVOT. Porcine heterografts mounted in Dacron conduits became available in a variety of sizes and were widely used for complex reconstructions. Heterografts, however, were more difficult to implant. In particular, they did not conform to the anatomy as easily as homografts, and hemostasis was more difficult to achieve.[8, 9] Furthermore, the porcine conduits became calcified rapidly, and the Dacron graft developed a pseudointimal peel that could result in the stenosis of small-diameter conduits. The development of cryopreservation techniques combined with improved availability has resulted in the widespread use of homografts for the reconstruction of the RVOT in congenital heart disease.[4] Although early results with homograft reconstruction of the RVOT have been good, relatively few data are available on the mid- and long-term results. The purpose of this study was to determine the early and mid-term result of homograft reconstruction of the RVOT. Over the last 3 decades, the Rastelli operation has been performed with a progressive decline in early mortality and can be undertaken with low morbidity with encouraging results.[5,10] The Ross procedure is an effective means of AVR, which can be accomplished with low perioperative morbidity and mortality if certain technical modifications are carried out. Short and mid–term results demonstrated good valve function.[7,11-12]

This series of patients comprises a broad spectrum of congenital heart defects involving hypoplasia or atresia of the pulmonary valve and RVOT and aortic valve dysfunction (LVOT). In our study, most of the patients (76.2%) had Rastelli operation, and about 20.7% had Ross procedure.

Among the various groups of diagnoses, TGA, VSD-PS/ LTGA consituted the largest group. The mean RV pressure before implantation was too high (93.7mmHg before the first homograft implantation and 87.5mmHg before the subsequent corrective surgery).

Gradual deterioration over time can be expected for homograft valves.[13-16]

The 10 - year period of this study spanned significant changes in the timing of surgery, role of palliative procedures and improvements in perioperative management.

Limitations of the size of homografts that can be implanted in neonates, infants and small children will predictably result in the need for homograft replacement as the child grows.[13,14,16] The data support the concept of outgrowth as a mode of failure of the homograft. About 70% of the patients had their corrective (against palliative) surgery with homograft implantation, and the most common complication was homograft stenosis (36.8%). Tweddell et al. had suggested that whenever possible, larger homografts should be used.[4]

The majority of the patients with complications had the diagnosis of TGA, VSD-PS / LTGA, too. Their most common type of homograft was aortic. Their previous homografts were aortic too, except in one patient. Yuan et al. found that apart from operative mortality, the application of aortic valved homograft conduits was an excellent choice for the correction of complex congenital heart disease.[17] A second aortic valved homograft replacement results in good

early and long – term survival. Accelerated degeneration does not occur.[18] Aortic homografts can be used for complex reconstruction in newborns and infants without the need for earlier reoperation. Given the relative shortage of small pulmonary homografts, the determination that aortic homografts can be used in infants and neonates without compromising long-term results may allow better utilization of the available homograft supply.[4]

Recent reports suggest that the durability of homografts has decreased in the current era. Niwaya et al.[16] identified later year of surgery as a risk factor for homograft failure. Stark and associates[15] noted that homograft replacement was associated with a higher risk of failure than the original homograft. Possible explanations for this observation include a broadening of the indications for homograft placement including younger patients with more complex lesions as well as a decreased threshold for replacement of a dysfunctional homograft. Another possible explanation for these observations is a change in the homograft donor supply.[4] However, some studies have shown a re-Rastelli operation for the problems of extacardiac valved conduit has a good result.[19, 20]

In our study, about 23.7% of the patients died in the process of surgery or its complications, which were mainly perioperative complications (17.8% of 23.7%). The most common cause of death was inability to wean from cardiopulmonary bypass and cardiopulmonary arrest in ICU during a poor hemodynamic situation, which may be due to ventricular failure. Other patients (4.9%) died during their subsequent surgery (reoperation), and one (1%) died because of CHF in CCU. Therefore, about 95% of the deaths occurred perioperatively.

In other studies, early deaths were related to unfavorable anatomy, conduit compression and sepsis. Residual VSD and postoperative infection were the main factors contributing to the late deaths.[20-21] Suematsu et al. clinical results of homograft implantation for infective heart disease were excellent.[22] Nevertheless, we had 6 patients (5.9%) with primary endocarditis on the aortic valve operated on using homografts. Three of them (50%) led to death: two of them at the first operation in OR and ICU after 2 days; and the other one at the subsequent surgery due to residual VSD after 2 days in ICU and 6 months after his first surgery.

In the 77 survivors, the function class was 1 in 96% of survivors at the time of discharge, 88% after 1 year and 77% after 2 years. To determine real statistic function classes in the patients, more years must be followed up because we have only 19 of the 77 patients followed up to 5 years. Only 7.7% of the survivors became CHB in their rhythms and received PPM. Almost 80% of them became RBBB in their ECGs, too. 11.6% of the survivors (7 pts) had homograft calcification, the earliest of which occurred 2 years after the operation.

Some studies have shown that homograft valves function satisfactorily in the pulmonary position at mid–term follow–up. They improve function class and can entail low mortality and morbidity, even after multiple previous median sternotomies.[23,24]

Having compared some of the variables, we suggest that an earlier decision to perform the surgical procedure reduce the RV pressure elevation process, which may cause a reduction of RV instability and failure. Dearani et al. concluded that younger age at operation was associated with improved late survival too.[25]

Our study is limited in that it is a retrospective analysis of patients with diverse anatomy operated on over a 10-year period. Undoubtedly, indications for

homograft use, timing of surgery and indications for homograft replacement changed during this time period. Variables analyzed such as homograft type were determined by surgeon's preference and graft availability and were not randomized or standardized. Because of the relations between variables, such as operative technique and diagnosis, it is possible that the influences of some factors have been underestimated and others have been given more importance. Nevertheless, this series looks at factors affecting the durability of homografts in a large number of patients undergoing RVOT reconstruction for congenital heart disease. The long-term outcome of cryopreserved homografts is not completely established, and therefore the contribution of these data is important. Continued follow-up studies will be necessary to identify risk factors for homograft failure.

Conclusions

Among 101 patients undergoing 108 reconstructions of the RVOT with cryopreserved homografts, about 23.7% deaths occurred, the majority of them occurring perioperatively (95.8%). The quality of life of the survivors of homograft implantation is excellent for most patients despite the need for reoperation.

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