HK J Paediatr (new series) 2005;**10**:271-281

Intermediate-term Results of Repair of Congenital Heart Diseases Using Pulmonary Homografts

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Abstract

Objective: We evaluate our intermediate-term results of our patients who had insertion of homografts for repair of their congenital heart diseases since the first application in April 1999. ***Background:*** Use of homograft valve in the repair of complex congenital heart lesions has been a standard for over two decades. However, homograft is not available locally because of lack of donor. Since 1999, we were able to obtain limited supply of homograft from England. ***Methods:*** Between 26 April 1999 and 28 February 2005, 45 patients had insertion of homografts for repair of congenital heart disease. The mean age of operation was 7.2±6.2 years (33 days-28 years). The mean follow-up duration was 32.5±20.6 months (0.03-71 months). The follow-up clinical status was reviewed. Overall survival and freedom from reoperation due to conduit failure were estimated by Kaplan-Meier method. Homograft valve stenosis or regurgitation was assessed by serial echocardiography and cardiac catheterisation. ***Results:*** There were one early death (2.2%) and two late deaths (4.4%). One of the late deaths died from non-cardiac cause due to severe bronchial stenosis. The actuarial survival was 93% at 71 months. Of the 42 survivors, majority had improved functional status significantly. There were 40 patients (95%) in NYHA class I or II. Three patients (6.7%) required reoperation for conduit failure. The freedom from reoperation was 85% at 71 months. One patient is awaiting reoperation for severe pulmonary homograft and aortic (truncal) regurgitation. Homograft conduit function of the remaining patients was satisfactory on follow-up. ***Conclusion:*** This study showed satisfactory intermediate-term outcome in patients after cardiac operations using homograft.

Key words Cardiac operation; Congenital heart diseases; Homograft

Introduction

In 1966, Ross and Somerville[1] first reported the use of a fresh antibiotic-preserved aortic valve homograft for the correction of pulmonary atresia. Many follow-up studies have demonstrated excellent haemodynamic characteristics of the pulmonary homografts after reconstruction of the right ventricular outflow tracts in children or adults with complex congenital heart diseases.[2-5] The requirement of no anticoagulation and freedom from thromboembolism also offers great advantage in repair of complex congenital heart diseases in children. Pulmonary homograft is also required in patient undergoing the Ross procedure for aortic valve replacement.[6-8] The pulmonary homografts have been regarded as the ideal conduits in repair of congenital heart diseases in children.

Pulmonary homografts are primarily harvested from human cadaver donors. This is not available locally because there is no tissue valve bank in Hong Kong. Since April 1999, we are able to obtain limited supply of cryopreserved homografts from the Heart Valve Bank of Royal Brompton Hospital in England. This article is an analysis of the intermediate-term results of our patients with homografts inserted in the right ventricular outflow tracts.

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Received April 21, 2005

Patients and Methods

From its first application on 26th April 1999 to 28th February 2005, 45 patients had homograft valves implanted for repair of their congenital heart diseases. Medical records of all patients were reviewed. The most recent examination, functional status, echocardiographic and restudy cardiac catheterisation results were analysed.

There were 27 male (60%) and 18 female patients. Age at operation using homograft ranged from 33 days to 28 years old (mean 7.2±6.2 years; median 5.1 years old). The mean duration of follow-up was 32.5±20.6 months (range 0.03-71 months) (Table 1). Different age group of patients undergoing homograft operation was summarised in Table 2. There was a large variety of congenital cardiac diagnoses that were summarised in Table 3. Forty-six

Table 1 Characteristics of patients

	Mean±SD	(median; range)
Total number of patients	45	
Age at operation	7.2±6.2 years	(5.1 years; 33 days-28 years)
Sex (male : female)	27 : 18	
Weight	19.6±13.5 kg	(14.5 kg; 2.93-48.4 kg)
Height	109.8±31.5 cm	(104.3 cm; 50-165.5 cm)
Duration of follow-up	32.5±20.6 months	(31.9 months; 0.03-71 months)

Variables are expressed as mean ± SD (median; range) where appropriate

Table 2 Characteristics of different age groups undergoing homograft operation

Age	Number of patients	Median; range	Number of patients needed reoperation
0-12 months	5	5.5 months; 33 days-11 months	1
>1 year-5 years	17	3.5 years; 1.2-4.8 years	1
>5 years-10 years	11	6.7 years; 5.1-9.4 years	1
>10 years-20 years	10	15.1 years; 10.6-16.4 years	0
>20 years	2	24.6 years; 19.9-28.6 years	0

Table 3 Diagnoses of patients

Diagnosis	Number	Percent of total (%)
Pulmonary atresia with VSD		
with previous Blalock-Taussig shunt	16	35.6
with previous RVOT reconstruction	3	6.7
Pulmonary atresia with IVS	1	2.2
Truncus arteriosus	5	11.1
Tetralogy of Fallot (post repair), RV dysfunction	7	15.6
Tetralogy of Fallot, disconnected RPA, post shunt	1	2.2
Tetralogy of Fallot with absent pulmonary valve	2	4.4
Transposition of great arteries, VSD, PS	5	11.1
Double outlet right ventricle, VSD, PS	1	2.2
Hypertrophic cardiomyopathy (Noonan syndrome), PS post RVOT reconstruction, severe PR, RV dysfunction	1	2.2
Aortic valve stenosis	1	2.2
Aortic valve stenosis and regurgitation	2	4.4

IVS, intact ventricular septum; PS, pulmonary stenosis; PR, pulmonary regurgitation; RPA, right pulmonary artery; RV, right ventricle; RVOT, right ventricular outflow tract; VSD, ventricular septal defect

previous operations had been performed in 24 patients. These included Blalock-Taussig shunt (n=30), repair of Tetralogy of Fallot (n=7), ligation or unifocalisation of aortopulmonary collateral arteries (n=3), right ventricle (RV) to pulmonary artery non-valved conduit (n=2), patch enlargement of right ventricular outflow tract (n=2), RV to pulmonary artery bioprosthetic heterograft valved conduit (n=2). Three patients had prior interventional catheterisations which included balloon aortic valvuloplasty in 2 patients and laser pulmonary valvotomy in 1 patient.

All the operations were performed by one cardiac surgeon. All homografts were cryopreserved and prepared at the Heart Valve Bank of Royal Brompton Hospital in England. The operations could be grouped into three basic types which included the Rastelli operation, Ross operation and pulmonary valve replacement at the right ventricular outflow tract. Additional surgical procedures including tricuspid valve annuloplasty or replacement, repair of the branched pulmonary arteries or intraoperative insertion of pulmonary artery stents were performed (Table 4). The Rastelli operation involved the patching of the ventricular septal defect (VSD) so that the left ventricular outflow passed through the defect into the aorta. A homograft was placed from the right ventricle (RV) to the main pulmonary artery. The Ross operation involved the removal of the abnormal aortic valve and transplantation of the patient's own pulmonary valve into the aortic position. The right ventricular outflow was reconstructed with a pulmonary homograft. In patients after repair of Tetralogy of Fallot or pulmonary atresia with VSD with right ventricular failure due to severe pulmonary regurgitation, pulmonary valve replacement with a pulmonary homograft was performed. The surgical techniques of these operations had been well described previously.[6,9,10]

Table 4 Types of operation

	Number of patients
Procedure	
Rastelli operation	28
Pulmonary valve replacement	14
Ross operation	3
(Total)	(45)
Additional procedure during operation	
Tricuspid valve annuloplasty	2
Tricuspid valve replacement (bioprosthetic valve)	2
Reconstruction stenotic pulmonary artery	4
Intraoperative stent pulmonary artery	1
Reconnection disconnected pulmonary artery	2
Pulmonary artery reduction arterioplasty	1
Reduction of ASD size	1
Enlargement of VSD for baffle construction	1
Closure of residual VSD	1
(Total)	(15)

VSD, ventricular septal defect; ASD, atrial septal defect

Early mortality was defined as death occurring within 30 days of operation.

Homograft or conduit failure was defined as requirement of reoperation because of severe stenosis or pulmonary regurgitation. The indications for reoperation included symptomatic patients with severe conduit stenosis confirmed by cardiac catheterisation after an echocardiograhic Doppler gradient >50 mmHg or having severe homograft valvar regurgitation. Asymptomatic patients with right ventricular pressure approaching systemic pressure, severe pulmonary regurgitation with progressive dilated right ventricle or right ventricular dysfunction were also indicated for reoperation. The freedom from reoperation was estimated by the Kaplan-Meier method.

Homograft function after operation was assessed by serial echocardiographies in all patients and cardiac catheterisations in selected patients. The degree of stenosis was graded into mild (<25 mmHg Doppler gradient); mild-moderate (25-50 mmHg gradient); moderate (>50 mmHg).[11] The degree of pulmonary regurgitation was graded semi-quantitatively by colour Doppler into mild, moderate or severe: mild = small jet of pulmonary regurgitation; moderate = wider jet with colour flow reversal above and below the leaflets; severe = broad-based jet with colour reversal in the entire pulmonary artery.[11] Homograft dysfunction was defined as stenosis with echocardiographic Doppler gradient >50 mmHg or greater than moderate pulmonary regurgitation. Reoperation will be considered if the patient fulfils the indications for reoperation as stated in the previous paragraph.

Statistical Analysis

Variables were expressed as mean±SD or median value. Cumulative actuarial survival and probability of reoperation due to homograft failure were estimated using the Kaplan-Meier method.

Results

Choice of Homograft

Forty-four pulmonary homografts and one aortic homograft were implanted. All homografts were cryopreserved and harvested from cadaverous donors. The size of homograft was determined on the age, weight and height of the patient (Table 5). The mean age of the donors was 38±14 years and the median age was 41 years (range 11-60 years).

Details of Operation

The mean cardiopulmonary bypass time and aortic cross-clamp time were 141±36 minutes and 97±37 minutes respectively. Only three patients required circulatory arrest for repair of the cardiac lesions. The durations of circulatory arrest were 4, 41 and 44 minutes. One patient had low flow bypass for 44 minutes and circulation was not completely arrested. The mean duration of ICU care was 6±14 days and the median was 2 days (range 1-90 days). Twenty-seven patients were transferred out of ICU within 2 days after the operation. The mean duration of postoperative mechanical ventilation was 4.5±14 days and the median duration was one day (range 4 hours- 90 days). Thirty patients could be successfully extubated within 24 hours after the operation. Table 6 summarised the major postoperative complications. The most common complications were haemorrhage and postpericardiotomy syndrome with significant pericardial effusion. One patient died of severe right ventricular failure shortly after operation. No other postoperative complication has caused mortality.

Mortality and Survival

There was one early death (2.26%) in a patient who had severe right ventricular dysfunction after correction of Tetralogy of Fallot in 1999 at the age of 1.5 years. She had right ventricular dysfunction since repair of Tetralogy of Fallot but she remained asymptomatic while taking three antifailure medications. There were progressive development of severe tricuspid and pulmonary regurgitation resulting into right ventricular dilation and dysfunction. She became symptomatic in 2002. Pulmonary valve replacement with a 19 mm pulmonary homograft and tricuspid valve annuloplasty was performed in 2003. However, intraoperative transesophageal echocardiography still revealed severe tricuspid regurgitation after the annuloplasty. She underwent cardiopulmonary bypass again for replacement of the tricuspid valve with a bioprosthetic pericardial valve. She suffered from severe low cardiac output and intractable junctional ectopic tachycardia in the ICU. She died eight hours after operation.

There were two late deaths. One infant died eight months after the homograft operation due to persistent heart failure. This patient had type II truncus arteriosus who was repaired with a 14 mm pulmonary homograft at 33 days old. She also had chromosome abnormality (46XX 8p+) and multiple congenital anomalies including hydrocephalus, single ectopic kidney and tracheobronchomalacia. Severe heart failure was due to severe homograft valve regurgitation and aortic (truncal) valve regurgitation secondary to infective endocarditis after the operation. Second operation with replacement of the dysfunction homograft with a bovine jugular venous conduit (Contegra™ conduit) was performed four months later. However, she died from persistent heart failure, severe chlyothorax and multi-organ failure eight months after the first operation. The second patient who had DiGeorge syndrome and Tetralogy of Fallot with absent pulmonary valve died six months after the

Table 5 Types of homografts and distribution of patients

Size of homograft (mm)	Number of patients*	Age (years)	Weight (kg)	Height (cm)
14	1	0.09	2.9	50
15	1	0.37	6.1	63
16	1	6.56	16.8	108
17	5	4.15	13.3±7.1	104.9±15.1
18	12	2.87	15.3±13.0	94.5±31.0
19	12	5.54	18.4±10.4	109.4±24.5
20	10	12.4	27.9±12.8	135.3±21.2
21	2	9.52	29.2±27.2	133.0±59.4
23	1	19.9	48	155

*Pulmonary homograft, n=44; Aortic homograft, n=1

Age is expressed as median value; weight and height are expressed as mean± SD where appropriate.

Table 6 Postoperative complications

Complication	Number of patients
No complications	19
Haemorrhage	
- Reexploration & haemostasis	4
- Increase blood products infusion	4
Delayed sternal closure	2
RV failure (>2 inotropes)	5
Pulmonary hypertensive crisis	1
Arrhythmia	
- JET	2
- SVT	1
- VT	1
Endocarditis	1
Pulmonary	
- Chylothorax	2
- Pleural effusion	2
- Pneumothorax	1
Renal insufficiency (peritoneal dialysis)	4
Neurologic	
- Transient cerebral dysfunction	2
Postpericardiotomy syndrome	
- Surgical drainage/pericardial window	3
- Aspirin/steroids	9

Patients having >1 complications were counted separately into different categories of complications

RV, right ventricle; JET, junctional ectopic tachycardia; SVT, supraventricular tachycardia; VT,ventricular tachycardia

operation due to non-cardiac cause. He had severe bilateral bronchial stenosis. He died from respiratory failure and recurrent pneumonia despite insertion of left bronchial stent. The homograft was functioned normally at the time when he died.

Therefore, the overall total mortality was 6.7% and the mortality related to cardiac cause was 4.4%. The overall actuarial survival was 93% at 71 months (Figure 1).

Follow-up Status

Majority of patients had improved functional status after the operation. Of the 42 survivors, there were 25 patients in New York Heart Association Class I (59.5%), 13 patients in class II (35.7%), 2 patients in class III (4.8%) and none in class IV (Figure 2). Patients with gross cardiomegaly before operation showed significant reduction of heart size (Figure 3).

Nineteen patients (45%) required no cardiac medication. All except two patient required anticoagulants. These patients were taking Warfarin because of recurrent intra-atrial re-entry atrial tachycardia or atrial fibrillation. There was no thromboembolic event in all survivors during follow-up.

Reoperation for Conduit Failure

Three patients (6.7%) required reoperation for homograft conduit failure at 4.3 months, 1.1 and 3.5 years after the homograft operation. One patient, who had transposition of great arteries, ventricular septal defect and pulmonary stenosis had Rastelli operation performed at 9 years old, developed stenosis at proximal right ventricular conduit anastomosis (RV pressure >75% systemic pressure). He underwent successful relief of obstruction at 12.9 years old. Pulmonary homograft valve function was normal and required no surgical intervention. The second patient had Rastelli operation for type II truncus arteriosus at age 33 days. Infective endocarditis resulted in severe pulmonary homograft regurgitation. She also had moderate truncal valve regurgitation. Replacement of the pulmonary homograft with a bovine jugular venous valved conduit (Contegra™ conduit) was performed at 5 months old. She died of persistent heart failure and multiple organ failure 8 months after the first operation. She also had chromosome abnormality and multiple congenital anomalies. The third patient had insertion of pulmonary homograft for treatment of free pulmonary regurgitation at age 2.5 years. Her cardiac diagnosis was pulmonary atresia, intact ventricular septum, hypoplastic right ventricle status post right ventricular reconstruction at infancy. Rapid degeneration of the pulmonary homograft resulting into severe regurgitation was noted after one year of implant. Pulmonary valve replacement with a 18 mm pericardial valve together with right cavopulmonary shunt and reduction of atrial septal defect size was performed at 3.6 years old. She was doing well with mild cyanosis after the last operation. The freedom from reoperation due to conduit failure estimated by Kaplan-Meier method was 85% at 71 months (Figure 4).

Homograft Function on Follow-up

The degrees of homograft conduit stenosis and regurgitation of the survivors (n=42) were illustrated in Table 7. There was no patient having moderate pulmonary stenosis (Doppler gradient >50 mmHg). One patient with severe homograft valve regurgitation had undergone pulmonary valve replacement with a pericardial valve (the third patient mentioned under the previous session of reoperation for conduit failure). The other patient (age 9)

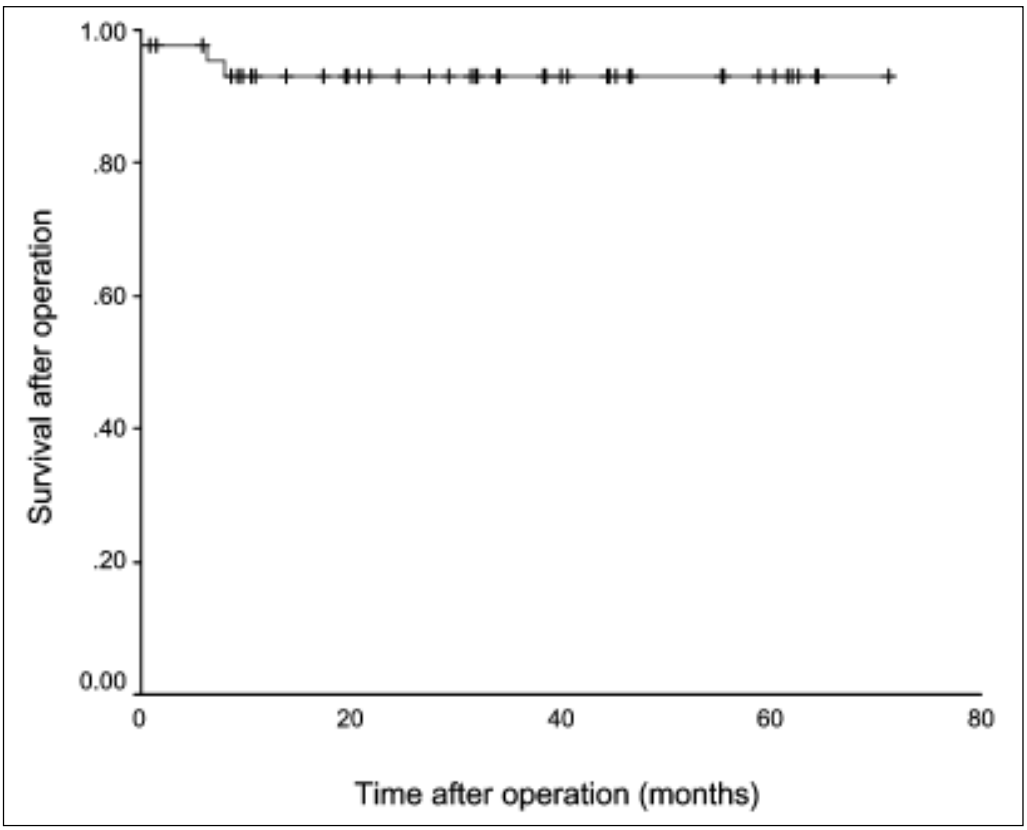

Figure 1 Overall patient survival estimated by Kaplan-Meier method.

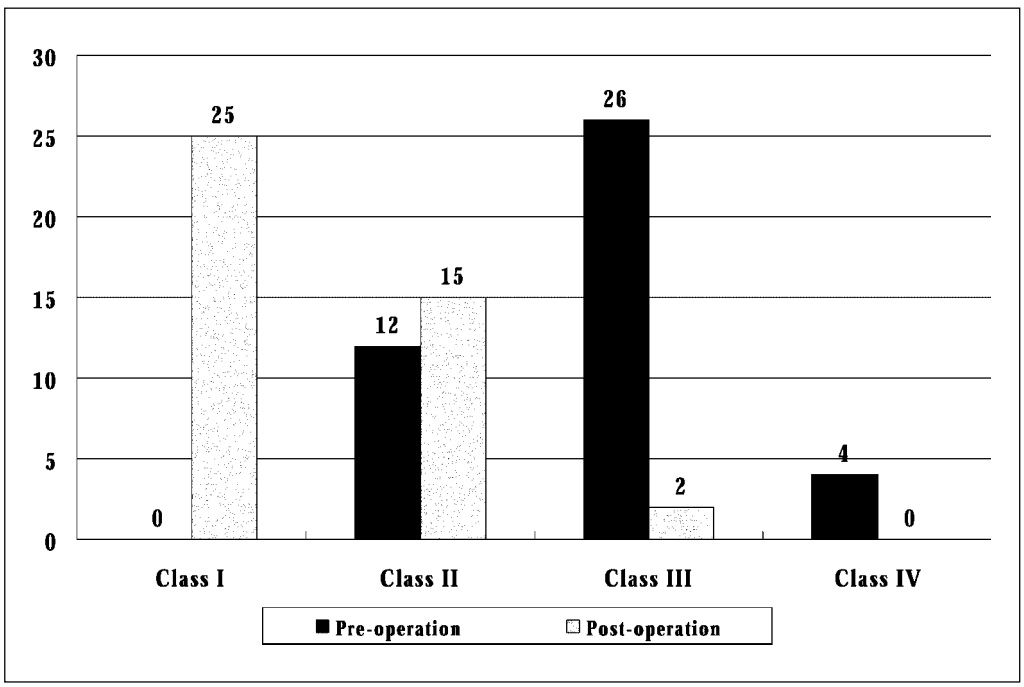

Figure 2 New York Heart Association (NYHA) functional status of surviving patients (n=42) before and after operation.

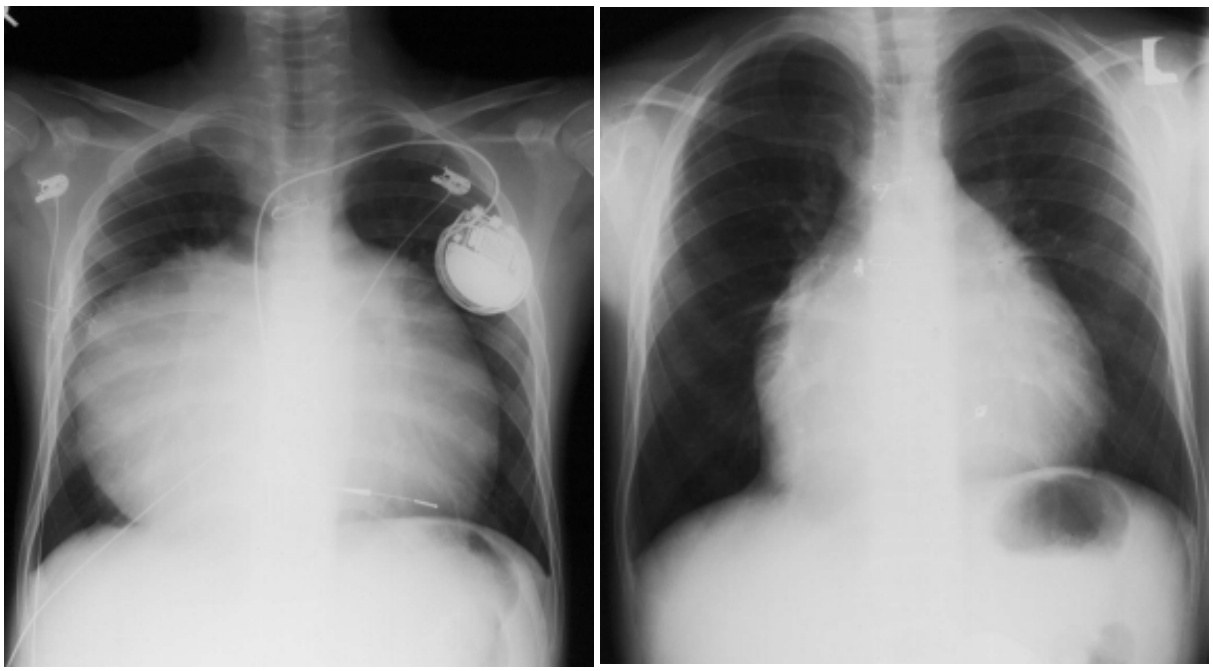

Figure 3 Gross cardiomegaly (cardiothoracic ratio 0.88) on the chest radiography before homograft operation, reduction of cardiothoracic ratio to 0.57 after operation. The endocardial leads was removed and two epicardial leads were implanted.

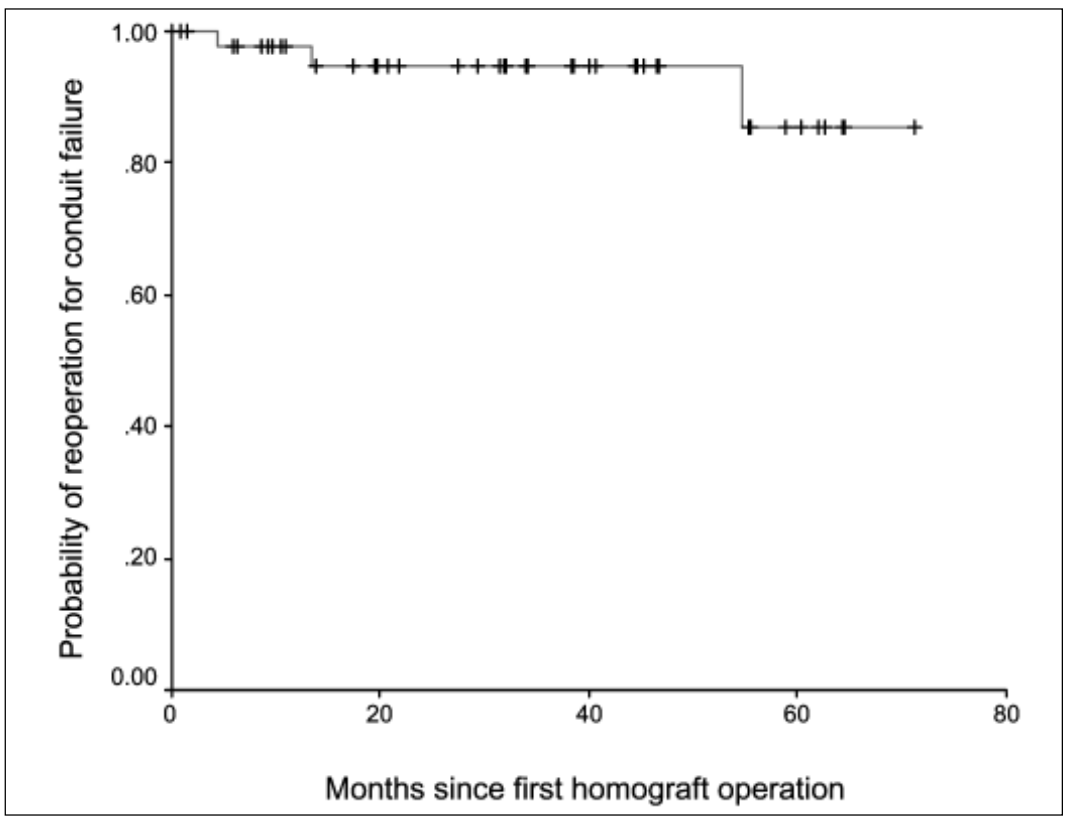

Figure 4 Freedom of reoperation due to conduit failure.

Table 7 Homograft conduit function of the late survivors (n=42) assessed by echocardiography or cardiac catheterisation during follow-up

	Number of patients	Percent of total (%)
Stenosis		
Mild (<25 mmHg)	38	90.5
Mild-moderate (25-50 mmHg)	4	9.5
Moderate (>50 mmHg)	0	0
Regurgitation		
None	21	50.0
Mild	15	35.7
Moderate	4	9.5
Severe	2	4.8

with severe pulmonary homograft regurgitation is waiting for replacement of the pulmonary homograft and the severely incompetent aortic (truncal) valve after repair of truncus arteriosus in 2001.

Homograft dysfunction having greater than moderate pulmonary regurgitation was presented in six patients in this study on follow-up. The four asymptomatic patients having moderate pulmonary homograft regurgitation did not have progressive right ventricular dilation and their right ventricular function remained satisfactory. Immediate reoperation was not indicated at present.

Discussion

This study has demonstrated satisfactory intermediate-term results since we started this operation 6 years ago. The number of patients in our series is relatively small, our results are comparable to other cardiac centers. Dearani et al of Mayo Clinic reported their early mortality of 3.7% since 1993.[12] We achieve an early mortality of 2.2%. This may be explained by the small number of infant patients less than 12 months old (2.2%) operated in this study. Operation on infants with complex congenital heart lesions carries a higher risk. Most of our patients do not have severe preoperative pulmonary hypertension. In our current study, only four patients having truncus arteriosus who had significant pulmonary hypertension were operated. The relatively smaller number of patients having severe preoperative pulmonary hypertension may also explain the low early mortality in our current study. The overall survival reported by other studies ranged from 85% to 95%[3,12] at 5 years and is similar to our actuarial survival (93% at 5 years).

The use of conduit has made possible correction of many complex congenital heart malformations. Before 1999, homograft is not available in Hong Kong and therefore we have to use non-valved pericardial conduit, non-valved polytetrafluoroethylene (Gore-Tex™) conduit or the porcine-valved Dacron conduit (Hancock valved conduit). The non-valved conduit without a pulmonary valve is not physiological and this contributes to frequent occurrence of postoperative mortality and morbidity. The relative long cardiopulmonary bypass and aortic occlusion times in our patients reflected the difficulties of repair of these complex congenital heart malformations especially for patients who underwent reoperation. However, with the use of pulmonary homograft, most of our patients had excellent haemodynamic after the operation. The median length of stay in intensive care unit was two days. The median duration on mechanical ventilation was one day. In fact, 30 patients were successfully extubated within 24 hours after the operation. The insertion of a competent pulmonary homograft valve prevents significant right ventricular failure due to pulmonary regurgitation which is universal for a non-valved conduit. The rare occurrence of severe life threatening postoperative complications in our study is likely contributed by the good haemodynamic after insertion of a competent pulmonary homograft valve. One of the most common early complication is haemorrhage which may be related to the relative long cardiopulmonary bypass. It is interesting to note that postpericardiotomy syndrome with significant pericardial effusion requiring drug or surgical treatment is quite common in our current study. Whether insertion of pulmonary homograft (a foreign tissue) aggravates the immune-mediate response remains unproven.

Pulmonary homograft is soft and pliable. This makes

the right ventricular tract reconstruction easier and this is most suitable for use in infants and small children. It is also believed to be more resistant to infection compared to other artificial mechanical or tissue valves after implant. However, the limitation of donor (especially young donor for small sized homograft) and significant cost in managing tissue valve bank are disadvantages. The other conduit that we have used is porcine-valved Dacron conduit (Hancock valved conduit). These conduits are commercial products and have the advantage of being readily available in many sizes. However, the rigid valve ring may cause compression on sternum or coronary artery making repair in small infant difficult. We had one neonate with truncus arteriosus previously repaired with a 14 mm Hancock valved conduit (small sized homograft was not available at that time). The sternum could not be closed resulting in persistent infection of the mediastinum and conduit despite prolonged antibiotic therapy. The conduit was successfully replaced with a 17 mm pulmonary homograft conduit and infection could be cleared up. Development of obstructive intimal fibrocalcific peels in Hancock Dacron conduit is a common indication for reoperation,[13] but this is rare for homograft conduit.

Because of the good handling characteristics of pulmonary homograft during operation, better haemodynamic and durability in homograft than heterograft conduit.[14] We regard pulmonary homograft is the choice of conduit in infants or small children.[15] With the introduction of pulmonary homograft since 1999 in our hospital, we believe this has significantly reduced the mortality and morbidity in our patients who require a right ventricle to pulmonary artery conduit. The available of pulmonary homograft also enables us to start performing Ross procedure for children with left ventricular outflow obstruction since 1999.

Late Homograft Dysfunction or Failure

Freedom from reoperation due to conduit failure in our series was 85% at 71 months. This is similar in the experience of the Great Ormond Street Hospital for Children (88%),[3] the Mayo Clinic (84%)[12] and the Hospital for Sick Children in Toronto (81%)[16] at 5 years after operation. Conduit failure was defined as requirement of reoperation.

Development of homograft dysfunction and subsequent failure necessitates reoperation remains the major problem in long-term. Numerous late follow-up studies have shown the freedom from reoperation ranged from 50% to 60% at 10 years and 30-35% at 15-20 years after first operation.[2,3,12,16] This will mean that reoperation is almost inevitable when homograft is implanted at early childhood. Risk factors associated with more rapid homograft failure include young recipient age, younger donor age, use of aortic homograft, small sized homograft, second replaced (redo) homograft, preoperative pulmonary hypertension, distal pulmonary artery stenosis and long cross-clamp time.[2-5,12,14] Two out of the three patients requiring reoperation in our series are below five years old (Table 2) which concurs with the risk factor of young recipient. Our median donor age is 41 years old and that may explain our slightly lower incidence of homograft dysfunction on follow-up echocardiograms.

Our surgeon prefers the use of pulmonary homograft rather than aortic homograft because pulmonary homograft is more durable and has less chance of stenosis.[2,4,14] This may be related to the higher elastin and intrinsic calcium content in aortic homograft.[17] This may also explain the low incidence of severe homograft stenosis in our current study. It is known that pulmonary homograft after Ross procedure is more durable because of the orthotopic pulmonary valve implantation rather than the extra-anatomic position in Rastelli operation.[2,14] The inclusion of patients after Ross procedure in our study may also explain the low occurrence of stenotic homograft during follow-up.

Apart from mechanical factors, degeneration of homograft valve by immunological mechanism is also an important factor. Numerous studies have demonstrated vigorous HLA antibody response and induction of cytotoxic T-lymphocytes after implantation of homograft in children and adult.[18-20] One of our patients has rapid degeneration of homograft one year after operation and it is reasonable to speculate that immune destruction may a role in her. Nevertheless, matching of homografts by ABO blood group or HLA type is unrealistic because of the already limited number of donors.[18,21] Studies have shown that ABO incompatibility was not associated with increase homograft dysfunction.[3,22] The use of immunosuppressants on recipients had also failed to show any beneficial effect.[23]

Future Demand and Supply

We expect an increasing demand of pulmonary homograft in future. We now favour early neonatal repair of complex heart lesions (for example truncus arteriosus), which is being practised in other centres.[24,25] Insertion of a pulmonary homograft is almost a must for successful outcome but the availability of small-sized homograft is always a problem due to lack of young donor. In our current

study, we have used adult sized homografts for small children. Whether this explain the lower incidence of developing stenosis on follow-up remains difficult to prove. Fortunately the soft and pliable characteristics of pulmonary homograft allow our surgeon to insert adult sized homografts into small children. Because of the lack of small-sized homograft, we are "forced" to use adult sized homograft in small children.

There are increasing numbers of adolescents or young adults who have repair of Tetralogy of Fallot or pulmonary atresia present with right ventricular failure related to severe pulmonary regurgitation.[26,27] Pulmonary valve replacement has been shown to be the effective therapy.[10,28-30] Although Hancock valved conduits can be used in adult as the alternative, pulmonary homografts are still necessary in small sized children. We are also planning to perform more Ross procedure for children with aortic valve disease after the initial success in our three patients. Although the Ross procedure is more complicated than simple aortic valve replacement with a mechanical valve, it has the advantages of avoidance of anticoagulation, freedom from thrombo-embolism, and the ability of the autograft to grow. [6-8,14,31]

There is no heart valve bank in Hong Kong. Despite we are able to obtain cryopreserved homograft from the Royal Brompton Hospital, British hospitals are prioritized regarding the allocation of tissue. Therefore, we expect increasing difficulty in obtaining sufficient homograft with the increasing demand in future. Currently we have 25 patients on waiting list for operation requiring implant of homografts.

Tissue-Engineered Heart Valve

Development of tissue-engineered heart valves may be the solution to provide "permanent" implantable heart valve.[32,33] One of the methods is to a decellularized homograft or xenograft and then seeded with autologous endothelial cells or dermal fibroblasts. This will theoretically produce an immunological inert heart valve. Using xenograft tissue, an unlimited supply of tissue-engineered heart valves will be possible. This is an exciting area of research and some preliminary animal and clinical studies are encouraging.[34-36] The establishment of a heart valve bank will enhance the research on tissue-engineered valve.

Conclusion

This study demonstrated satisfactory intermediate-term outcome in patients with pulmonary homografts implanted for repair of complex congenital heart diseases. Late homograft failure requiring reoperation, increase demand and lack of availability of homografts are ongoing problems. We believe that establishment of a local heart valve bank will be beneficial.[37]

Acknowledgement

We thank the Children's Heart Foundation (www.childheart.org.hk) for their generous support in sponsoring the import of homografts from the Heart Valve Bank of Royal Brompton Hospital, London.

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