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Surgery for right ventricle to pulmonary artery conduit obstruction: risk factors for further reoperation☆

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Abstract

Objective: To identify the surgical approaches and risk factors which influence longevity of right ventricle to pulmonary artery (RV–PA) conduits following first reoperation for obstruction. **Methods**: Between January 1993 and August 2003, 114 patients underwent 141 reoperations for RV–PA conduit obstruction. Diagnoses included 'Truncus Arteriosus' ($n=52$), 'Pulmonary atresia/Tetralogy of fallot' ($n=39$), 'Double outlet right ventricle' ($n=10$), 'Transposition of great arteries, VSD, and pulmonary atresia' ($n=9$), and the 'Ross operation' ($n=4$). All patients had undergone a previous biventricular repair. The first reoperation for conduit obstruction was performed in 112 hospital survivors by: total conduit replacement (Group A, $n=73$) with valved (homograft=10 and xenograft=54) or non-valved ($n=9$) conduit, and patch enlargement of the obstructed RV outflow tract with preservation of the posterior and sides of the conduit wall after removing of the fibrocalcific peel and degenerated valve (Group B, $n=39$). Mean age at first reoperation was 8.8 ± 6.7 and 7.5 ± 5.3 years in patients of groups A and B, respectively. Seven patients in Group A and 18 in Group B required a second reoperation and two patients in Group B a third reoperation. **Results**: There were two hospital deaths and no late deaths. Mean follow-up was 5.8 ± 3.2 years. Risk factors for second reoperation by univariate analysis were: homograft conduit use ($P=0.004$), Group B surgical approach ($P=0.0001$), higher RV–PA systolic pressure gradient at discharge ($P=0.02$), and age <5-years-old ($P=0.01$). Multivariate analysis showed that inclusion in Group B and younger age (<5-years-old) at repair were independent risk factors for second reoperation. Group B surgical approaches had higher RV–PA systolic pressure gradient at discharge ($P=0.02$) and required more PA bifurcation repair at the time of second reoperation ($P=0.05$). Freedom from second reoperation for conduit obstruction was significantly higher in Group A patients at 5 and 8 years ($P<0.04$) and those with xenografts rather than homograft ($P=0.04$). **Conclusions**: Our results support the optimal surgical approach for RV–PA conduit obstruction is total replacement with a xenograft. RV outflow reconstruction by other techniques without complete dissection of PA bifurcation does not completely relieve the stenosis and could cause early restenosis. Higher systolic gradients at discharge and younger age at first reoperation are predictors of earlier reoperation.

Keywords: Congenital heart defect; Redo surgery; Valved conduit

1. Introduction

Reconstruction of the right outflow tract (RVOT) with extra cardiac conduits has made possible complete repair of complex cardiac malformations [1,2]. However, the mid- and long-term durabilities are poor due to conduit degeneration, which results in progressive stenosis and haemodynamic compromise requiring recurrent conduit replacement. As a result, the congenital heart surgeon frequently faces RV–PA conduit obstruction requiring further repair. Despite the introduction of different RV–PA conduits, the ideal conduit and RVOT reconstruction techniques are yet to be developed. In this study, we reviewed our results of RV–PA conduit obstruction surgery for identifying the risk factors for subsequent reoperations.

2. Patients and methods

From January 1993 to August 2003, 114 patients with situs solitus and congenital heart disease underwent 141 reoperations for RVOT reconstruction due to RV–PA conduit obstruction at Marie-Lannelongue Hospital. Only hospital survivors were included in order to assess the longevity of RV–PA conduits following the first reoperation for obstruction. Two early deaths (within 30 days of operation or same hospitalization) were excluded.

2.1. First operation

All 112 patients including 60 males (53.6%) and 52 females (46.4%) underwent a previous biventricular repair with

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Table 1
Diagnostic category of patients

Diagnosis	$n=114$
Troncus arteriosus	52
+IAA	4
+Pulmonary artery stenosis	3
+LSVC to coronary sinus	2
PA/VSD—tetralogy of fallot	39
+AVSD	1
+Pulmonary artery stenosis	6
+LSVC to coronary sinus	4
DORV	9
+PS	9
TGA/VSD/PA	10
+Coarctation	1
+Sub Ao. stenosis	1
Aortic valve stenosis	4

IAA, interrupted aortic arch; LSVC, left superior vena cava; PA/VSD, pulmonary atresia and ventricular septal defect; AVSD, atrioventricular septal defect; PS, pulmonary stenosis; TGA, transposition of great arteries; Sub Ao, subaortic.

a RV–PA conduit implantation. The cardiac defect and associated lesions are summarized in Table 1. The biventricular repair was preceded by palliative procedure in 41 patients out of 112 (41.1%) hospital survivors. Forty-two patients had balloon angioplasty and three had additional stent implantation prior to the first reoperation for RV–PA conduit obstruction.

2.2. First reoperation

The first reoperation was considered as 'time zero' in this study. In our center, surgery for RV–PA conduit obstruction was indicated in asymptomatic patients when the RV to PA gradient was >65–70 mmHg or when the RV/LV pressure ratio was $\geq$0.8.

RVOT reconstruction was performed by two surgical approaches: total conduit replacement with valved or non-valved conduits (Group A), and patch enlargement of the obstructed RV outflow tract with preservation of the posterior and sides of the conduit wall after removing of fibrocalcific peel and degenerated valve (Group B). The mean age at this time zero procedure was 8.8 ± 6.7 years (Group A, median 7; range 0.8–34 years) and 7.5 ± 5.3 years (Group B, median 6; range 0.8–26 years).

RVOT reconstruction was performed under normothermia cardiopulmonary bypass (CPB) without aortic cross clamp in the absence of any intracardiac defect or concomitant cardiac lesions requiring repair. Associated cardiac procedures are described in Table 2.

Group A consisted of 73 patients (65.2%) who underwent total conduit replacement either by valved ($n=64$, 87.7%), or non-valved ($n=9$, 12.3%) conduits. The valved conduit included cryopreserved aortic or pulmonary homografts ($n=10$, 15.6%), Hancock (Medtronic, Minneapolis, MN), glutaraldehyde-preserved porcine-valved Dacron conduits ($n=47$, 73.4%), Contegra (Medtronic, Minneapolis, MN) bovine jugular veins ($n=4$, 6.3%), or Labcor (Sulzer Carbomedics, Austin, TX) glutaraldehyde-preserved bovine pericardium conduit with a stentless porcine valves ($n=3$, 4.7%). The non-valved conduit included Gore-tex ($n=5$, 55.6%) and Dacron ($n=4$, 44.4%) grafts.

Table 2
Concomitant procedures in Groups A and B patients

Procedure ($n=46$)	Group A ($n=28$)	Group B ($n=18$)
Pulmonary artery repair	10	12
Bifurcation	1	8*
Left PA	4	2
Right PA	5	2
Residual VSD repair	6	3
Aortic valve repair	4	0
Aortic valve replacement	3	1
SubAortic stenosis release	3	1
Pace maker implantation	2	1

*$P<0.05$.

Group B consisted of 39 patients (34.8%) who underwent a RVOT patch enlargement of the previous conduit. The previous conduit was splitted lengthwise after establishing cardiopulmonary bypass, and the degenerated valve, fibrocalcific peel, and some millimeters of lateral edge removed. The right and left pulmonary arteries were measured and then the RVOT reconstruction was done by using a large patch over the preserved posterior and sides of the conduit wall. The patch included Dacron ($n=26$, 66.7%), Gore-Tex ($n=9$, 23.1%), and xenograft pericardium ($n=4$, 10.2%) patches.

Postoperative echochocardiography was performed to measure RV–PA peak instantaneous systolic gradients for all patients. The RV–PA peak instantaneous systolic gradient at discharge was 20 ± 11 mmHg (Group A) and 36 ± 13 mmHg (Group B).

2.3. Follow-up

All survivors were followed biannually by the referring cardiologist (mean follow-up 5.8 ± 3.2 years) and received a clinical assessment, an ECG and an echocardiogram. Cardiac catheterization was performed when indicated. All data were regularly transmitted to our center and incomplete data were investigated by telephone call to the referring cardiologist. Quantification of the RV–PA conduit obstruction was evaluated by color Doppler imaging. The obstructed conduit was first considered for transcatheter balloon dilatation when feasible. The time of the second RV–PA conduit reoperation was based on signs and symptoms, medical response to therapy, results of balloon dilatation, and worsening gradient by color Doppler. The indications for this second reoperation were identical to the first reoperation.

2.4. Statistical analysis

All measured values were expressed as means$\pm$standard deviations. Statistical comparisons for identifying the risk factors for the second reoperation of an obstructed RV–PA conduit were performed by the 'Student t-test' and 'χ^2'-test for continuous and categorical variables, respectively. These variables were 'sex', 'age at first reoperation', 'type of initial cardiac anomaly', ' type of conduit in initial

biventricular repair', ' type of surgical repair in first reoperation', ' type of conduit or patch in first reoperation', 'size of conduit', 'concomitant procedure', ' peak systolic RV-PA gradient at discharge', and 'need for pulmonary bifurcation reconstruction at second reoperation'. Multivariate analysis was performed by 'logistic regression' model. Time-related freedom from reoperation (second reoperation for RV-PA conduit obstruction) was described both by actuarial methodology and the difference among groups was calculated by the log-rank test.

3. Results

3.1. Mortality

Of the 114 patients who had a first reoperation for RV-PA conduit obstruction, there were 2 (1.8%) early deaths. The deaths were attributed to complications of pulmonary hypertension and left bronchial trauma during extensive left pulmonary repair at the time of the repair in two patients of Group A (P=NS). One patient had an atrioventricular septal defect/TOF and the other had truncus arteriosus with interrupted aortic arch, as the initial cardiac anomaly, respectively. There were no late deaths.

3.2. Second and third reoperation

Out of 112 hospital survivors after first reoperation for the obstructed RV-PA conduit, 25 (22.3%) patients underwent a second reoperation. The number of second reoperation was significantly higher ($P<0.0001$) in Group B ($n=18$, 46.2%) than Group A ($n=7$, 9.6%). The surgical techniques used for second reoperations were conduit replacement ($n=22$, 88%) with Hancock conduits ($n=16$), Contegra conduits ($n=3$), homograft ($n=1$) and patch enlargements ($n=3$, 12%).

Of the seven patients in Group A who needed a second reoperation, four had homografts, two had Contegra conduits and one had a Hancock conduit during the first reoperation. All four patients with homografts had aortic homografts at the time of initial biventricular repair. No patient with non-valved conduits has significant obstruction at the time of study to require a second reoperation. The need for pulmonary artery bifurcation repair due to stenosis at the second reoperation was 1 (14.3%) and 8 (44.4%) in Group A and B patients, respectively ($P=0.05$).

A third conduit reoperation was performed in two patients who had undergone a RVOT patch enlargement (Group B) for their second reoperation. The surgical technique for third re-operation was conduit replacement by Hancock conduit.

3.3. Risk factors for second reoperation

Univariate analyses identified the following risk factors for second reoperation: Group B surgical technique, younger age of patients ($P=0.01$), patients receiving a homograft with Group A surgical technique ($P=0.004$), and the presence of higher RV-PA peak instantaneous systolic gradient at discharge ($P=0.02$). The RV-PA peak systolic gradient at discharge was significantly higher in Group B patients ($P=0.02$).

Homograft implantation and RVOT patch enlargement techniques (Group B) remained a significant risk factor for second reoperation after adjusting for age at first reoperation and conduit size. Sex, type of 'initial cardiac anomaly', type of 'conduit in initial biventricular repair', type of 'patch and xenograft conduit in first reoperation', and 'presence of concomitant procedure' were not risk factors for second reoperation. There was no significant risk difference between non-valved and xenograft conduits.

The results of the multivariate analyses show that the younger age of patients ($P<0.003$) and inclusion in Group B ($P=0.0001$) were independent risk factors for second reoperation.

3.4. Freedom from second reoperation

Actuarial rates of freedom from second reoperation comparing surgical techniques were 97.9±2, 83.7±3 and 70.4±6% for patients in Group A at 3, 5 and 8 years, respectively, and 94.9±2, 61±4 and 30.5±8% for patients in Group B at 3, 5 and 8 years, respectively. There was a significantly fewer reoperation in Group A patients at 5 and 8 years ($P<0.04$) (Fig. 1).

In comparing conduit use, freedom from second reoperation for Group A patients were 96.7±2, 90.7±4, and 81.1±3% in patients with xenograft valved conduits and 88.9±3, 75.2±5, and 45.1±4% in those with homograft at 3, 5 and 8 years, respectively (Fig. 2). There was a significantly *lower* rate of reoperation in patients with xenograft valved conduit at 5 and 8 years ($P<0.05$).

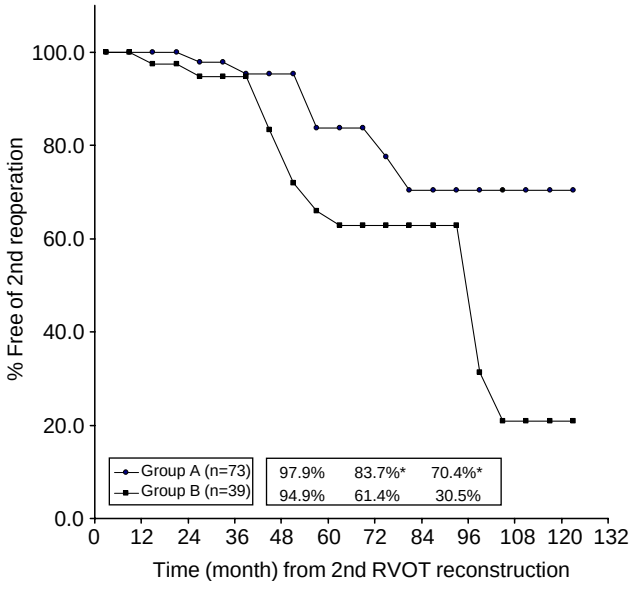

Fig. 1. Freedom from second reoperation according to the surgical technique of RVOT reconstruction (Groups A and B). *$P<0.05$.

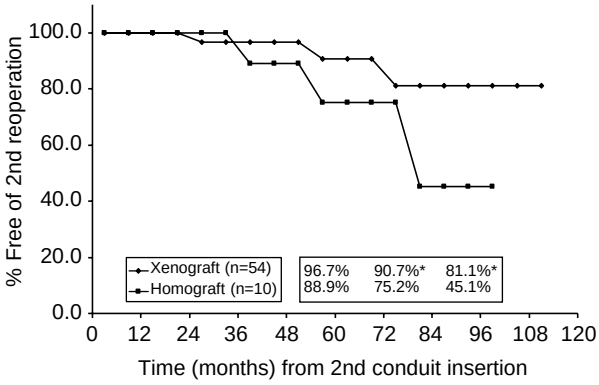

Fig. 2. Comparison of freedom from second reoperation in patients with xenograft and homograft. *$P<0.05$.

4. Discussion

In this study, we identified surgical approaches and risk factors for further reoperation of RV–PA conduit obstruction, which is more and more frequent with early complete repair. Determination of these risk factors could help us to neutralize them by different approaches.

RV–PA conduit revision is needed due to degeneration and calcification of the conduit and patient growth leading to recurrent obstruction. Selection of the RVOT reconstruction approach is different and more standard at the time of reoperation compared to the initial repair at neonatal or infancy period for several reasons. First of all, a less aggressive cardiac surgery approach is enough to address only the conduit replacement without concerns for intracardiac defects as in majority of cases. Secondly, patients are older at the time of first reoperation with slower rates of somatic growth, attenuating the influence in conduit stenosis. Thirdly, conduit size is less limiting in childhood and adolescence than in the neonatal period. Furthermore, the discrepancy of tissue texture with any conduit material is less prominent beyond neonatal and infancy period due to an increase of fibrous tissue. These differences could help us to design a safer and more reproducible surgical approach in order to decrease the number of reoperation for conduit stenosis.

In the current study, the optimal surgical approach for RV–PA conduit obstruction was replacement with another conduit. The RVOT patch enlargement technique, even with the absence of a valve and creation of a large diameter pathway, had a higher RV–PA systolic pressure gradient post-operatively and larger percentage needing a second reoperation. Although this technique seems to be simple and fast, two possible explanations for early re-stenosis in these patients are the incomplete dissection and enlargement of the pulmonary bifurcation, and preservation of the posterior wall of the original conduit, which could be thrombogenic. These might result in an increase in turbulence in the outflow pathway and consequently higher RV–PA peak gradient at discharge, resulting in earlier re-stenosis. The negligible early RV–PA systolic pressure gradient and freedom from second reoperation during the follow-up among the nine patients with non-valved total conduit replacement who had the same profile of patch technique operation (absence of valve and large diameter conduit) support this.

The significantly higher requirement for pulmonary artery bifurcation repair at the second reoperation in the patch enlargement technique reflects that these patients need enlargement in the pulmonary bifurcation as well as proximal part of RVOT reconstruction for avoiding early stenosis. Although, recently Bermudez et al. [3] demonstrated the very good late results of the Mayo clinic RVOT patch enlargement technique (Peel operation) in which the RVOT was reconstructed using a pericardial roof over the fibrous bed of explanted conduit, with or without a prosthetic pulmonary valve. The median age of their patients (19 years) was much older than our group of patients (7 years). In younger patients, the effect of somatic growth (as a reason for early reoperation) is still an important factor and the need for PA enlargement is more necessary than the older patient group. Interestingly DeLeon et al. [4] showed that small or distorted pulmonary arteries could adversely affect the longevity of RVOT grafts.

The analysis of our data indicated that implantation of homograft conduit was a risk factor for a second reoperation with less durability observed for xenograft and non-valved conduits even after adjusting for age and size. Since introduction of cryopreserved homograft in the 1980s, a number of institutional reviews [5–9] have reported the outcomes achieved with these conduits. The data and conclusions drawn from these studies are conflicting, especially in adult patients. Several reports [7,8,10–12] have demonstrated superior durability for pulmonary than aortic homografts, and orthotopic implantation versus extra-anatomic placement of the conduit in RVOT reconstruction. Stark [13] also showed that a probable immune response after the first implant of a homograft could result in more rapid degeneration after the second homograft implant. Interestingly, all our patients with cryopreserved homograft, which needed a second reoperation, had all of the described risk factors (aortic homograft, extra-anatomic position, and having a homograft in first operation).

The different results after homograft implant seem to be influenced by the different types of homografts and conditions of homograft preparation. Furthermore, type of cardiac pathology affects homograft durability in RVOT reconstruction, and even donor criteria such as longer donor warm ischemic time [8]. These reasons make it difficult to choose a homograft as a routine conduit beyond infancy for conduit replacement, although the unavailability of homograft is still a problem in the latter group.

The analysis of our data indicated that ‘younger age’ is an independent risk factor for second reoperation. There is a general agreement [3,5,8,9,14] that ‘young age’ is a risk factor affecting longevity of all types of RV–PA conduits used in the initial repair because of more rapid valve degeneration and accelerated rate of patient growth. It seems to be reasonable if the first reoperation is performed; later one could achieve greater longevity of

the RV–PA conduit. It might be possible that with some modifications in our approach, such as tightening indications in asympomatic patients and operating on these patients as late as possible. Currently, indications for conduit revision are not standardized. Our indications for conduit replacement in asymptomatic patients is near systemic or systemic pressure of the RV and a peak instantaneous Doppler systolic gradient greater than 65–70 mmHg, compared to 40–50 mmHg in the majority of other studies [8,14]. Our late results with no deaths support using this higher gradient as an indication for reoperation. Furthermore, transcatheter interventions such as balloon dilatation or stent implantation when indicated, may prolong the RV–PA conduit lifespan [5,15–17] and delay the first reoperation for conduit obstruction. Thirdly, use of an autologous alternative like 'Reparation à l'étage Ventriculaire' (REV procedure) of Lecompte [18] or other technical options [19,20] may be helpful. In these operations, RVOT reconstructions with the patient's own tissue during the first operation appear to defer RVOT reoperations. However, in some lesions such as truncus arteriosus, direct anastomosis could result in significantly higher mortality based on a study by Lacour-Gayet et al. [21].

Less invasive approaches such as off-bypass surgical [22] or percutaneous implantation [23] of the pulmonary valve, have recently emerged as interesting techniques in the management of RV–PA conduit obstruction. Proponents of these treatments argue that they may replace conventional surgery. While long-term results of these techniques are unknown, they may delay surgical conduit replacement, and be considered in the treatment strategy of patients with RV–PA conduits.

Overall, freedom from second reoperation in this study is better in xenograft valved conduits, particularly the 'Hancock'. Our experience with other conduits including bovine venous valved conduit (Contegra) is limited. Out of four patients with Contegra, two had early failure due to high RV–PA peak systolic gradient and valve regurgitation. This problem has been recently mentioned by others [24,25]. We also had respectable results in a small number of patients with non-valved conduits, but it would be difficult to draw definitive conclusion in these carefully selected patients. We currently preferred to use a xenograft valved conduit for RVOT reconstruction to avoid the long-term effect of pulmonary insufficiency.

5. Conclusion

The best surgical approach for RV–PA conduit obstruction in our center is replacement by xenograft valved conduits. Based on recent publications, the use of aortic homograft is not recommended for the first reoperation following previous homograft implantation due to the higher risk for further reoperation.

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