Use of bovine jugular vein to reconstruct the right ventricular outflow tract: Early results

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Objective: We evaluate early results of bovine jugular vein conduits in the pulmonary outflow.

Methods: Between April 2000 and September 2001, 31 conduits were placed in the outflow of the right ventricle. Patients who received a conduit as a staged surgical procedure were excluded (n = 3). Implantation age ranged from 0 to 21 years (median, 3.4 years). Conduit diameter ranged from 12 to 20 mm (median, 14 mm). Transthoracic echocardiography was performed at discharge and 3 months after surgery. Patients with significant pulmonary regurgitation and/or stenosis underwent cardiac catheterization.

Results: Four patients died during the follow-up period. Three deaths were unrelated to the conduit. One death was related to the complete thrombosis of the conduit. At 3 months evaluation, pulmonary valve regurgitation was absent or trivial in 19, mild in 2 and severe in 3 of 24 survivors. Four patients had nonfatal conduit-related complications. A transient thrombus formation within 1 leaflet was noted postoperatively in a patient with a moderate pulmonary regurgitation. Three patients required reoperation 3 to 5.8 months after the implantation for conduit failure (mean, 4.3 months). Cardiac catheterization before replacement revealed an aneurysmal dilation of the conduit below a severe stenosis of the pulmonary bifurcation due to important neointimal proliferation.

Conclusions: Early failure of bovine jugular vein valved conduits can occur because of exaggerated intimal proliferation or thrombotic process within the conduit. Because of these complications, close echocardiographic follow-up is mandatory during the first weeks after implantation.

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In a great variety of congenital heart diseases, a valved conduit is required to reestablish the continuity between the right ventricle and the pulmonary artery bifurcation.^{1,2} While widely used, conduits made with woven synthetic tubes as supportive housing for a biological valve (porcine aortic valve, bovine pericardium, or homograft) are not ideal because of rapid complications in children.³⁻⁶ These include valvar dysfunction, calcification of the valve, and accumulation of fibrous peel within the synthetic conduit and lead

to progressive stenosis of the conduit. The life span of these conduits depends largely on the biological origin of the valve. A conduit using bovine jugular vein, which contains a natural trileaflet valve (Contegra, Venpro, Medtronic), was introduced in mid-2000. Preliminary animal studies have shown that this totally integrated valved conduit has good biocompatibility and excellent hemodynamic properties.⁷⁻⁹ This conduit is also available in various diameters, from 12 to 22 mm, making it suitable for implantation from early infancy to adulthood. These properties have led to an extension of its use. However, no data are yet available on the performance of these conduits in the clinical setting. The purpose of this single-center study was to investigate the early performance of this new conduit.

Methods

Patient Population

We reviewed the records of all patients who underwent placement of a Contegra conduit (Venpro, Medtronic) for the establishment of the continuity between the right ventricle and the pulmonary arteries between April 2000 and September 2001. Diagnoses were established in the cardiac unit with echocardiography and/or angiography. Operations done in a single institution by a single operator were included. During that period, 31 conduits were placed. Patients with total repair were eligible (28/31). Three patients with a pulmonary atresia and a ventricular septal defect were excluded from the study because the ventricular septal defect was not closed at the time of conduit insertion. There were 16 men (57.1%) and 12 women (42.9%). The median age at operation was 3.4 years (range, 0-21.1 years). Bovine jugular vein conduits were implanted in a variety of congenital heart diseases (Table 1). The diameter of the conduit at the time of implantation ranged from 12 mm to 20 mm (14.8 ± 2.9 mm; median, 14 mm). Six conduits were stented (21.4%). Tables 2A and 2B summarize the clinical characteristics of the included patients and conduit specifications together with age range. Sixteen out of the 28 eligible patients (57.1%) had undergone at least 1 previous cardiac operation (Table 3).

Surgical Repair

The operations were performed through a median sternotomy. Conventional cardiopulmonary bypass was used with double venous cannulation and cardioplegia. The conduit was anastomosed to an infundibulotomy. Enlargement of the pulmonary artery bifurcation was performed as needed. Any associated defects were repaired before conduit insertion (Table 4).

Follow-up

Echocardiographic evaluation was carried out in all surviving patients and examined in the immediate postoperative period and reexamined with a transthoracic approach 3 months postoperatively. All studies were reviewed in order to assess the presence of pulmonary regurgitation and to determine the mean valve gradient. The pulmonary regurgitation was classified as none (grade 0), trivial (grade 1), mild (grade 2), moderate (grade 3), or severe (grade 4) with regard to features of the jet with pulsed flow

TABLE 1. Patient characteristics

Cardiovascular disease	No.	%
Pulmonary atresia/VSD	6	21.4
Tetralogy of Fallot	5	17.9
With AVC	1	
Truncus arteriosus	9	32.1
Type 1	7	
Type 4	2	
Aortic valve stenosis	4	14.3
Isolated	2	
With VSD	1	
Coarctation of the aorta	1	
Absent pulmonary valve syndrome	2	7.1
Complex transposition	2	7.1
With pulmonary stenosis	1	
With pulmonary atresia	1	

VSD, Ventricular septal defect; AVC, atrioventricular canal.

Doppler in short-axis parasternal view.¹⁰ Conduit stenosis was assessed by measuring the peak velocity through the valve with continuous-wave Doppler technique. All patients with severe pulmonary regurgitation and/or significant obstruction of the right ventricular outflow tract were catheterized.

Data Collection and Statistical Analysis

Preoperative and postoperative data were collected at retrospective review of patients' records. Data are presented as the mean $\pm$ the standard deviation. Early events were defined as events that occurred before hospital discharge or within 30 days of operation if the patient was discharged before this duration. Event-free survival distribution was estimated using the actuarial method. The overall cardiac events including death, severe pulmonary regurgitation, and conduit thrombosis were considered relevant for this analysis.

Results

Mortality and Survival

There were 4 deaths (14.3%) during the follow-up period, 3 early deaths, and 1 late death. One patient died of pulmonary problems at the age of 3 months following a gastroenteral operation. Another patient with a truncus arteriosus and a regurgitant truncal valve died of infection in the early course after the operation. This child had a severe bronchopulmonary dysplasia that delayed his discharge from the cardiac intensive care unit. The third patient died of severe pulmonary disease secondary to a premature birth. This infant had a truncus arteriosus with an interrupted aortic arch without 22q11 microdeletion. His cardiac disease was completely repaired early at the age of 15 days when he weighed 2300 g. None of these 3 deaths were directly related to the conduit. The last death occurred in a patient with a tetralogy of Fallot associated with a complete atrioventricular canal and a trisomy 21. This patient was completely repaired at a first procedure at the age of 16 months and rapidly discharged without any medication. Echocar-

TABLE 2A. Conduit specifications

Conduit sizes	12 mm	14 mm	16 mm	18 mm	20 mm
No. (%)	11 (39.3)	6 (21.4)	4 (14.3)	3 (10.7)	4 (14.3)
Mean age at implantation (range)	4 mo (0.4-16.1)	1.9 y (0.6-4)	2.4 y (1-5.6)	7.5 y (5.5-10.3)	12.1 y (5.8-21.1)
Stented conduits (%)	3 (27.3)	2 (33.3)	0	0	1 (25)

TABLE 2B. Age categories

Age categories	No.	%
0-12 mo	14	50
12-24 mo	4	14.3
2-5 y	2	7.1
5-10 y	5	17.9
>10 y	3	10.7

TABLE 3. Operative data

Previous surgery	No.
Blalock-Taussing shunt	7
RVOT reconstruction with conduit insertion	10
LV to Ao conduit	1
Unifocalization of the pulmonary artery	3
Valvuloplasty	4
Surgical	1
Transcatheter	3
VSD closure	9
RV to PA conduit replacement	1
Crafoord procedure	1
Ductus arteriosus ligation	5
Konno procedure	1
Pulmonary transannular patch	2
ASD closure	1
Enlargement of the aorta and ductal tissue resection	1

RVOT, Right ventricular outflow tract; *LV*, left ventricle; *Ao*, aorta; *VSD*, ventricular septal defect; *RV*, right ventricle; *PA*, pulmonary artery; *ASD*, atrial septal defect.

diodiography at discharge showed a trivial pulmonary regurgitation and a moderate elevation of the systolic ventricular pressure at 40 mm Hg. Fourteen days after the operation, he was found dead by his mother. The anatomicopathologic study revealed a complete thrombosis of the 12-mm conduit with multiple pulmonary embolisms and infarcts. Abundant chylothoracic effusion and signs of liver congestion were also present.

Early Dysfunction

One patient with a 14-mm conduit implanted to correct a complex transposition of the great artery had a grade 3 pulmonary regurgitation postoperatively. This regurgitation was caused by a thrombus formation within 1 leaflet. After

TABLE 4. Concomitant procedures

Concomitant procedures	No.
Reconstruction of the pulmonary artery bifurcation	5
Proximal/distal enlargement of the pulmonary arteries	5
Size reduction of the pulmonary arteries	2
VSD closure	10
Ligation of BT shunt	7
Ligation of MAPCAs	2
Pacemaker implantation	1
Creation of an ASD	4
Ross procedure	4
Bentall	1
Konno	5
Conduit replacement	10
Plasty of truncal valve	1

VSD, Ventricular septal defect; *BT*, Blalock-Taussig; *MAPCA*, major aortopulmonary collateral artery; *ASD*, atrial septal defect.

a 1-week course of intravenous heparin, the thrombus disappeared and the patient was discharged. The echocardiography at discharge showed a trivial regurgitation of the implanted valve.

Conduit replacements were performed in 3 patients at 3, 4, and 5.8 months after the initial insertion of a 12-mm conduit (mean, 4.3 months). Echocardiography showed a severe dilation of the right ventricle (RV) secondary to massive pulmonary regurgitation, and at least iso-systemic systolic RV hypertension. These patients were catheterized before surgery. The catheterization study confirmed the echocardiographic data showing dilated and dysfunctioning RV, and an aneurysmal dilation of the conduit below a severe stenosis of the pulmonary bifurcation. The mean diameter of the conduit was measured angiographically at 20 mm (Figure 1). All these data were confirmed during surgical replacement of these failing conduits. Macroscopic examination of the retrieved conduit showed a non-functioning valve (Figure 2). A fibrinous tissue was covering 2 of the 3 leaflets. This tissue was easily retrieved without any damage to the valve. No valvar calcification was found macroscopically and histologically. Histological

Figure 1. Angiograms (4-chamber view on the left and lateral view on the right) showing the aneurysmal dilation of the Contegra conduit before a severe stenosis of the pulmonary bifurcation 3 months after insertion.

study showed an important fibrointimal proliferation in the inside layer of the xenografts predominantly in the pulmonary anastomosis that obstructed the lumen. At this site, the orifice measured 0.5 cm. At the outside layer, a uniformly myofibroblastic reaction with a collagen formation was found (Figure 3). Within this formation, a mild foreign body reaction was also present. From the middle part of the conduit to the ventricular anastomosis, a histiocytic reaction infiltrated and partially digested the external part of the prosthesis. No host cells covered the valvular leaflets. No endothelial proliferation was observed in the lumen of xenografts.

Reoperations consisted of replacement of the conduits by a porcine aortic valve (Labcor) and an enlargement of the pulmonary bifurcation by a pericardium patch. Two of these patients are now facing problems of residual proximal pulmonary artery stenosis. Actuarial survival curve for adverse cardiac events is shown on Figure 4.

Conduit Function

Doppler echocardiographic evaluation before discharge showed no or trivial regurgitation in 20 patients (83.3%), a mild valve regurgitation in 4 patients (16.7%), and moderate or severe regurgitation in none. The peak velocity across the valve at that time was less than 2 m/s in 17 out 24 patients, and between 2 and 3 m/s in 7 patients. Echocardiographic follow-up was obtained in all survivors. Results are summarized in Table 5.

Discussion

The surgical management of congenital heart diseases involving the right ventricular outflow tract has largely evolved over the past decade.¹⁻⁶ However, the reconstruction of the right ventricular outflow tract remains a problem.

Currently, the continuity between the RV and the pulmonary artery is established by the interposition of a prosthetic conduit. Various conduits are commercially available, but the perfect substitute has yet to be found because of non-rare complications during the follow-up. Recently, a new conduit using a bovine jugular vein that contains a natural valve with a sinus has been introduced.^{7-9,11} The leaflets of the valve are extremely thin and mobile. The venous wall in form of a conduit is soft and ideal for suturing and usually does not necessitate any artifice or any interposition of other materials to make the anastomoses with the RV and the pulmonary artery. Moreover, its availability in various diameters from 12 to 22 mm makes its use possible in neonates. Thereby, it became popular and is now used in our institution and worldwide. Early and long-term functions of this valve are, however, presently unknown.

Here we described the function of bovine jugular vein conduit within 3 months after its insertion. Valvar competence at 3 months is excellent, with nearly 80% of patients with absent or trivial pulmonary regurgitation. The early complication reported here, however, balanced these good results. Indeed, 1 death was directly related to the Contegra conduit and could have been avoided. The patient was discharged from the hospital without any anticoagulant or platelet anti-aggregant therapy. This resulted in the progressive thrombosis of the valve, which was responsible for (1) elevation of right pressures resulting in cardiac liver and pleural effusion and (2) multiple emboli with pulmonary infarcts. Additionally, a thrombus formation was observed on a valvular leaflet in another patient. Heparinization of the patient, however, led to the complete resolution of the thrombus. No anticoagulant therapy is recommended after the insertion of this type of conduit. However, biological

Figure 2. Macroscopic images of the conduit retrieved: A, Longitudinal view of a bovine jugular vein. The ventricular anastomosis has a normal size whereas the pulmonary anastomosis (on the right) has a tight stenosis. B, Valve leaflet after retrieval of a fibrinous tissue shown in the upper right. C, Inner front view of the stenosed pulmonary artery anastomosis. D, Outer front view of the pulmonary artery anastomosis.

implants crosslinked with glutaraldehyde are potentially thrombogenic.^{8,9,12} Indeed, collagen molecules contained in biologic materials have many amino groups that are positively charged and can accumulate negatively charged platelets.¹³ Glutaraldehyde, by binding with the basic amino acids, creates an impaired balance between positive and negative charge in favor of negative charges.¹⁴ Thus, glutaraldehyde-pretreated implants become negatively charged, repulse platelets and, therefore, are less thrombogenic.¹⁵ However, glutaraldehyde has been reported to delay the endothelialization that reduces thrombogenicity of cardiovascular implants.¹⁶⁻¹⁸ Indeed, residual glutaraldehyde released from the implant long after insertion hinders host cells to colonize the luminal layer exposing the implant to thrombogenic process.^{8,9,19} Chang and colleagues⁹ recently showed no endothelialization of glutaraldehyde-pretreated grafts 6 months after implantation in a canine model. Therefore, we now prescribe an anti-aggregant therapy in all patients for at least 6 to 12 months after the implantation.

After this time, we discontinue the treatment, hypothesizing that endothelialization of the implant is complete and that thrombus formation is unlikely to occur.

Three other patients with a 12-mm valve had to be reoperated for an early failing of their conduit. With previously used conduits, early failures have been described not infrequently, in particular when small valves were inserted in young children.²⁰ The causes and the mechanisms of this failure remain unclear. It has been suggested that geometric distortion as well as immunologic rejection may be involved.^{4,21} The aneurysmal dilation of the conduit is more likely to occur in small conduits because these are implanted in neonates and young infants who have hypoplastic pulmonary arteries, making the repair particularly difficult. This would probably have been avoided by the use of a supported conduit that had 2 external rings. Out of the seven 12-mm conduits we implanted, 4 were not supported and 3 were supported. Presently, none of the supported valved conduits failed. However, the ring could compress the cor-

Figure 3. Photomicrographs of longitudinal section of the bovine jugular vein conduit 4 months postoperatively. *A*, Valve leaflets. *B*, Ventricular anastomosis. *C*, Pulmonary anastomosis.

Figure 4. Actuarial survival curve of adverse cardiac events.

onary artery, particularly in neonates and infants, making their use more delicate in this population. The stenosis of the pulmonary artery bifurcation could result from an inappropriate reconstruction. Even if it is unlikely, particular attention should be made when anastomosing the pulmonary arteries to the conduit to avoid further stenosis of the

TABLE 5. Echocardiographic findings during follow-up

Echocardiographic data	Discharge (%)	3 months (%)
Peak velocity across the valve		
< 2 m/s	17 (70.8)	17 (70.8)
2-3 m/s	7 (29.2)	4 (16.7)
> 3 m/s	0	3 (12.5)
Regurgitation		
None	4 (16.7)	4 (16.7)
Trivial	15 (62.5)	15 (62.5)
Mild	4 (16.7)	2 (8.3)
Moderate	1 (4.2)	0
Severe	0	3 (12.5)

pulmonary artery bifurcation. More astonishingly, the presence of inflammatory tissue located predominantly at the anastomosis underlines the possibility of an immunologic rejection. Cytotoxicity of residual glutaraldehyde may induce a foreign-body reaction and explain early bovine jugular vein failure.^{8,9,22} However, this reaction was limited in our study and did not explain the obstruction of the conduits. A pathological intimal proliferation predominantly located at the pulmonary anastomosis was present at the inside layer of the conduit, explaining its obstruction. No data are available in the literature to explain this phenomenon, but the occurrence in only glutaraldehyde-fixed grafts is questionable. Fibrointimal formation has been described in woven synthetic conduits and was attributed to fenestrations within the peel.²³⁻²⁵ Crosslinking that creates a network between the molecules of collagen might induce such fenestrations. Moreover, the proliferation predominated at the site of suture line. Chemical interactions between suture material and glutaraldehyde or cellular damages during suturing aggravated by glutaraldehyde cytotoxicity might be involved in intimal hyperplasia.

The occurrence of early failure in only 12-mm conduit is also questionable. Is the intimal proliferation normal or abnormal regarding the size of the conduit? Thus, similar intimal growth would lead to obstruction and dilation of the conduit in small conduits, whereas no adverse effect would be noticed with bigger conduits. And/or are neonates more likely to develop inappropriate reaction in response to crosslinking agent? Several crosslinking agents are presently under investigation to increase the life span of these conduits.

The uncontrolled dilation of the conduit is a major concern in terms of valvar competence, but shows that this conduit can increase in diameter with time under certain hemodynamic conditions. Because leaflets inside the conduit have excess tissue, the competence of the valve is preserved in moderately dilated conduits. However, no real growth with cellular proliferation is possible with devitalized conduits, and progressive dilation would certainly lead to significant pulmonary regurgitation. Nevertheless, if

long-term study confirms this feature, this would lead to reduction of reoperation for conduit replacement because of somatic growth.

Conclusion

The use of bovine jugular vein conduit has gained interest recently because of its intrinsic qualities. Early failure described in this report emphasizes the need for (1) controlling the adequacy of the distal anastomosis on the pulmonary artery bifurcation particularly in small patients and (2) careful rinsing of the conduit prior to its insertion to remove the glutaraldehyde. It also underlined the importance to prescribe an anti-aggregant therapy after the implantation to avoid early thrombosis of the conduit. Finally, long-term studies are obviously needed to ascertain safety, efficacy, and durability of the conduit.

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