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Alternative techniques of right ventricular outflow tract reconstruction for surgical repair of truncus arteriosus

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Key question

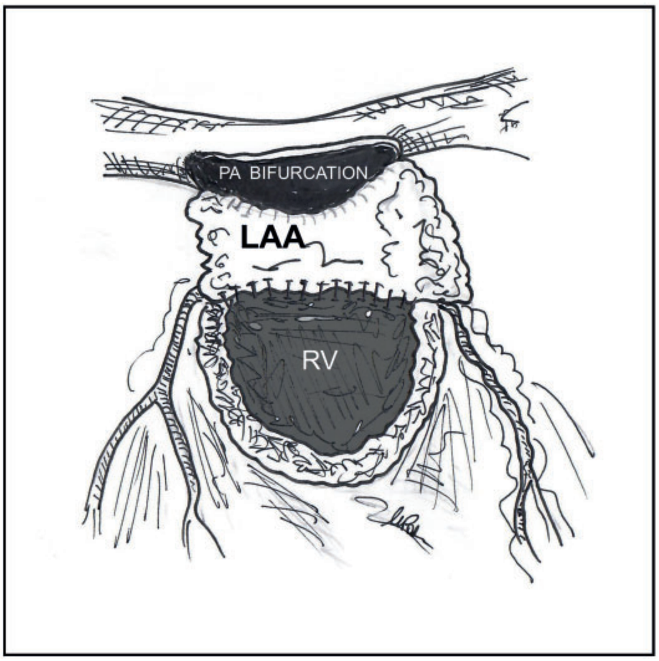
Does the right ventricular outflow tract reconstruction technique affect late-term outcomes

Key finding(s)

Use of a conduit has a tendency to present more late adverse events compared to the conduitless technique.

Take-home message

Use of a direct anastomosis for right ventricular outflow tract reconstruction in truncus repair may reduce reoperation rate in the long term.



Abstract

OBJECTIVES: This study aimed to evaluate the outcomes and feasibility of different techniques of reconstruction of the right ventricular outflow tract (RVOT) in surgical repair of truncus arteriosus.

METHODS: We retrospectively reviewed all consecutive patients with truncus arteriosus who underwent successful surgical repair in our centre between 1994 and 2017. We analysed late results according to the type of RVOT repair.

RESULTS: We collected data from 29 survivors after truncus arteriosus repair. Six (20%) of them were affected by DiGeorge syndrome. The RVOT reconstruction was achieved using a valved conduit in 58.6%, while a direct right ventricle–pulmonary artery (RV–PA) anastomosis, with or without the interposition of the left atrial appendage, was performed in the remaining. At a median follow-up time of 7.9 years (interquartile range 1.8–13.1), 6 patients (3 affected by DiGeorge syndrome) died. Between the 2 groups, there were no differences in terms of the late mortality and onset of adverse events. However, the use of a conduit seemed more prone to reintervention and onset of adverse events.

CONCLUSIONS: Different RVOT reconstruction techniques are safe and have similar late outcomes. However, use of a direct RV–PA anastomosis and left atrial appendage interposition may reduce the need for reoperation in the long term.

Keywords: Congenital heart disease • Truncus arteriosus • Right ventricular outflow tract reconstruction • Follow-up • Heart surgery

ABBREVIATIONS

AE	Adverse event
CHF	Congestive heart failure
CI	Confidence interval
ECMO	Extracorporeal membrane oxygenator
FU	Follow-up
IQR	Interquartile range
LAA	Left atrial appendage
PA	Pulmonary artery
RV	Right ventricle
RVOT	Right ventricular outflow tract
TA	Truncus arteriosus
VSD	Ventricular septal defect

INTRODUCTION

Truncus arteriosus (TA) is a rare congenital heart disease with a prevalence of 0.21–0.34% [1], which is characterized by a single arterial great vessel arising from the heart and giving origin to both systemic and pulmonary circulation [2]. Early repair is indicated to correct pulmonary overflow that can lead to severe pulmonary vascular disease, congestive heart failure (CHF) and ultimately death [3].

Surgical repair consists in the separation of the pulmonary trunk or pulmonary arteries (PAs) from the TA, closure of the ventricular septal defect (VSD) and reconstruction of the right ventricle (RV)–PA continuity. The right ventricular outflow tract (RVOT) reconstruction is usually achieved using a prosthetic conduit. Alternatively, an RV–PA direct anastomosis can be performed by mobilizing the inferior border of the pulmonary trunk to the right ventriculotomy [4, 5], or by creating a pulmonary trunk wall flap that can be used as the posterior wall of the RVOT when anastomosed with the right ventriculotomy [6, 7]. When a direct anastomosis is not feasible, the interposition of the left atrial appendage (LAA) as a posterior wall can be effective to reduce the anastomotic tension [8]. The direct RV–PA anastomosis with or without the LAA interposition has been proven to be effective [9, 10]. However, long-term outcomes remain unclear.

We report our experience with various types of surgical RVOT reconstruction during repair of TA to evaluate the late outcomes in terms of surgical reoperation and mortality.

MATERIALS AND METHODS

This is a single-centre retrospective study of all consecutive patients who survived after the repair of TA, between 1994 and 2017. We excluded patients with heterotaxy syndrome. The local hospital committee approved the review of medical records on clinical investigation. Individual patients were de-identified, and the need for patient consent was waived. All data were obtained by reviewing medical records and telephone interviews. The clinical follow-up (FU) consisted of full cardiological examinations at 1, 6 and 12 months after discharge and then every 2 years if conditions were stable and no complications were emerging.

Patients were divided into 2 groups according to RVOT reconstruction type: group 1 when a prosthetic valved conduit (cryopreserved pulmonary Homograft, Contegra® or Labcor®) was used and group 2 (conduitless) when a direct RV–PA anastomosis with a monocusp patch was performed, with or without the interposition of the LAA.

End points of this study were mortality, surgical reoperation, interventional procedure and onset of any adverse event (AE) at FU. We defined AE as the occurrence of death and/or any surgical reoperation and/or interventional procedure at FU.

Bidimensional echocardiographic data recorded at FU included RV dilation (defined as end-diastolic RV diameter >28 mm) and RV dysfunction (when tricuspid annular systolic plane excursion was <15 mm).

Variables are presented as median (interquartile range, IQR), or mean ± standard deviation according to the distribution of the variables. Incidence rates were compared by their Poisson distributions. FU times were compared with the Mann–Whitney test. Survival, freedom from reoperation and AE were examined by Kaplan–Meier curves; log-rank (Mantel–Cox) was used for curves' comparisons. Statistical significance was set at <0.05. R 3.6 (R Core Team 2019, Vienna, Austria) was used for statistical analyses.

Operative technique

Surgical repair of TA was achieved using a full median sternotomy and cardiopulmonary bypass in all. As a routine, the pulmonary trunk (if present) or the PAs were detached from the TA. The aortic root was repaired using an autologous pericardial or prosthetic (homograft, bovine pericardium) patch. Whenever the truncal valve leaflets were severely dysplastic, and regurgitation was significant, a truncal valve repair was performed, excising the most dysplastic leaflet including the adjacent sinus of Valsalva, reducing the leaflets number and the annulus size, with reconstitution of the annulus and truncal root by direct anastomosis, as described elsewhere [11]. The VSD was closed through the right ventriculotomy using a Goretex or a pericardial patch.

As mentioned earlier, the RVOT surgical reconstruction was achieved employing a conduit in group 1, or without a conduit (directly) in group 2, by reconstructing the posterior wall with a direct anastomosis between the PA and the posterior edge of the ventriculotomy, or with the interposition of the LAA (Fig. 1A and B), and the anterior wall with a monocusp pulmonary homograft patch (Fig. 1C).

RESULTS

Our complete experience is reported in Fig. 2. There were 29 patients (median age 26 days, range 7 days to 5 years; M/F: 14/15). A deep hypothermic circulatory arrest or selective anterograde cerebral perfusion was utilized in 8 cases (27.5%). According to the Van Praagh classification [12], a TA type A1 was the most frequent (17 patients, Table 1). There were no significant differences in the anatomical type of TA between the 2 groups. Associated cardiac anomalies were present in 13 patients (11 in group 1 vs 2 in group 2): coronary anomalies in 10, patent foramen ovalis in 4, patent ductus arteriosus in 2, moderate RV hypoplasia in 1 and total anomalous pulmonary venous return to coronary sinus in 1. DiGeorge syndrome was present in 6 patients (3 in each group). Eleven patients had a dysplastic truncal valve (6 in group 1 and 5 in group 2). A PA banding according to Trusler's technique [13] was performed before repair in 2 patients. The first patient was a 2-month-old premature male (body weight 1.5 kg), with a TA type A1, presenting with failure to thrive due to CHF, who underwent successful repair 4 months later. The other patient was a 3-week-old neonate (weight 2.7 kg) with TA type A2, massive pulmonary overflow and severe CHF, with previous necrotizing enterocolitis. This infant underwent a selective banding of each PA and completed a successful surgical repair at the age of 6 weeks.

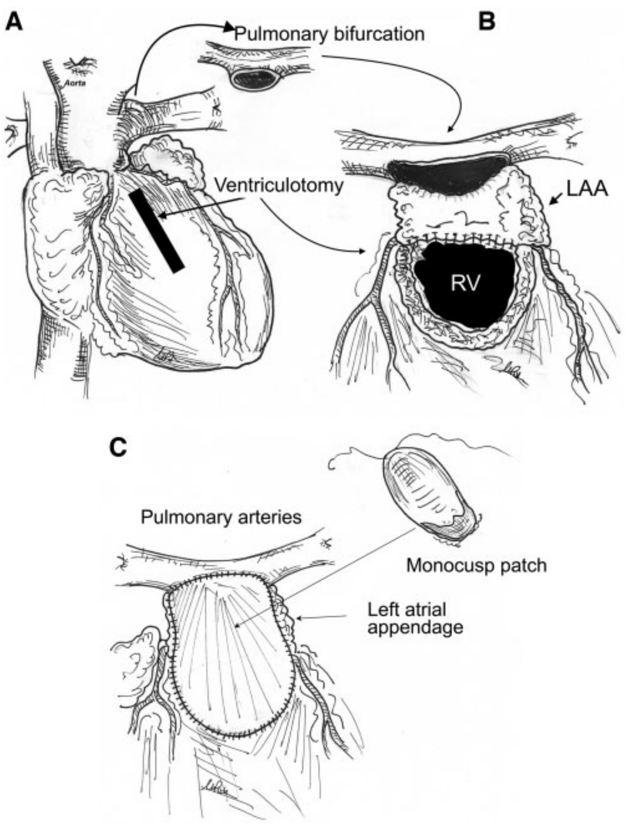


Figure 1: Operative technique of RV–pulmonary artery (PA) conduitless reconstruction using LAA. (**A**) Right ventriculotomy and separation of the pulmonary trunk (or PAs) from the aortic trunk; (**B**) utilization of the LAA as pavement of the new RV–PA conduit; and (**C**) completion of the RV–PA conduit using a pulmonary homograft monocusp patch, as roof or the RV–PA conduit. LAA: left atrial appendage; RV: right ventricle.

All the remaining patients underwent a successful primary repair, which included truncal valve repair in 5 (17%, 4 in group 1), consisting in resection of the dysplastic valve leaflet and the corresponding sinus in 3, plication of a hypoplastic leaflet and sinus of Valsalva in 1 and direct suture of a pseudo-commissure in the last patient.

The RVOT surgical reconstruction was achieved utilizing a conduit in 17 patients (group 1: 59%) at a median age at the operation of 24 days (IQR 13–33). A Medtronic Contegra conduit size 12 was used in 8 patients, a homograft was used in 8 (5 pulmonary homografts' size 7, 8, 10, 12 and 23 mm; 2 aortic homografts' size 8 and 12 mm; and a bicuspidalized homograft size 15 mm) and a LabCor conduit 11 mm was used in 1. On the contrary, in 12 patients (group 2: 41%, median age at operation 32 days, IQR 16.5–55), the RV–PA continuity was established by reconstructing the posterior wall using a direct anastomosis in 4 patients, the interposition of the LAA in 7 and a heterologous saphenous vein patch in 1. The anterior wall was always reconstructed with a monocusp (valved) pulmonary homograft patch (Fig. 1).

Additional surgical procedures were as follows: in group 1, patent ductus arteriosus closure in 2 and a total anomalous pulmonary venous return to coronary sinus surgical correction in 1 at 2 weeks of age, using the resection of septum primum, incision of the septum between coronary sinus and fossa

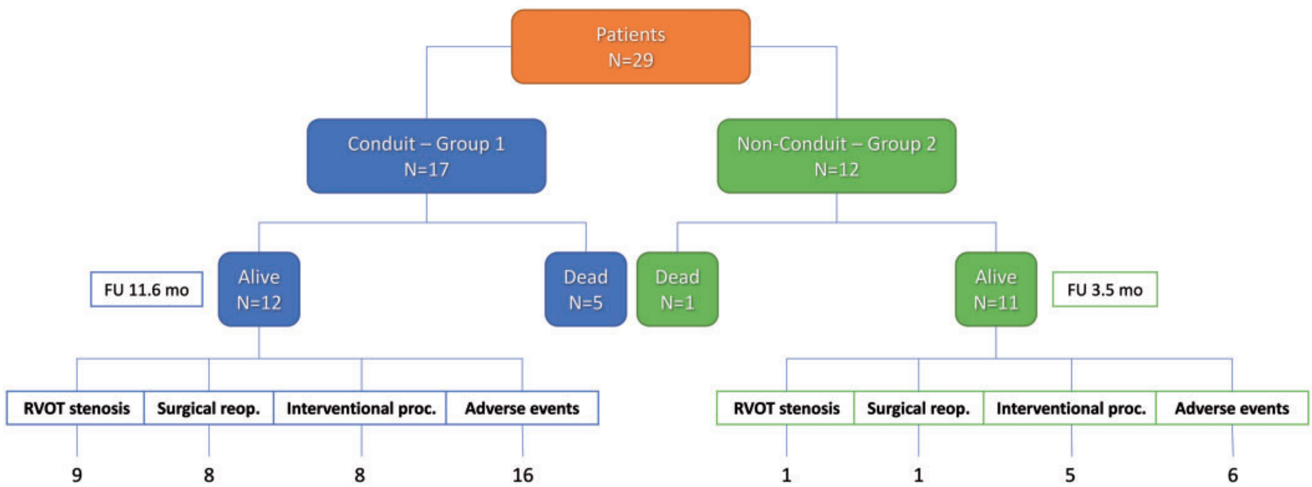


Figure 2: Descriptive flowchart reporting experience with conduit and conduitless technique for right ventricular reconstruction in truncus repair. FU: follow-up; RVOT: right ventricular outflow tract.

Table 1: Demographic, intraoperative and postoperative data

	Total (*n* = 29)	Group 1 (conduit) (*n* = 17)	Group 2 (non-conduit) (*n* = 12)	*P*-value
Female gender, *n* (%)	15 (52)	8 (47)	7 (58)	0.7
Age at operation (days), median (range)	26 (7–2104)	24 (7–2104)	32 (7–303)	0.7
Truncus type (Van Praagh classification)				0.63
A1	17	9	7	
A2	8	4	4	
A3	2	2	0	
A4	3	2	1	
Associated cardiac anomalies, *n* (%)	13 (45)	11 (65)	2 (17)	**0.02**
Coronary artery anomalies, *n* (%)	10 (34)	8 (47)	2 (17)	0.13
DiGeorge syndrome, *n* (%)	6 (21)	3 (18)	3 (25)	0.67
Non-cardiac malformations, *n* (%)	4 (14)	2 (12)	2 (16)	1
Truncal valve dysplasia, *n* (%)	11 (38)	6 (35)	5 (42)	1
Previous surgical palliation, *n* (%)	2 (7)	0	2 (17)	0.16
Associated surgical procedures, *n* (%)	9 (31)	6 (35)	3 (25)	0.18
Truncal valve plasty	5	4	1	
PDA closure	2	2	0	
TAPVC surgical correction	1	1	0	
Enlargement plasty of the neoaorta	1	0	1	
ECMO implantation	1	0	1	
Circulatory arrest, *n* (%)	8 (28)	7 (41.2)	1 (8)	0.09
Postoperative complications, *n* (%)	12 (41.4)	7 (41.2)	5 (41.6)	1
Postoperative ECMO, *n* (%)	2 (6.8)	0	2 (16.6)	0.16
Hospital stay (days), mean ± SD	21.1 ± 33.6	19.2 ± 12.7	35.3 ± 44.4	0.30

ECMO: extracorporeal membrane oxygenator; PDA: patent ductus arteriosus; SD: standard deviation; TAPVC: total anomalous pulmonary venous connection. Statistically significant values are expressed in boldface.

ovalis and closure of the resulting 'atrial communication' through an autologous pericardial patch. In group 2, a patch enlargement of the ascending neoaorta was required in 1 patient.

Overall, 12 patients had major postoperative complications. Specifically, in group 1, 7 patients had postoperative bleeding requiring chest revision in 2, acute kidney injury requiring renal replacement therapy (peritoneal dialysis) in 2, low output cardiac syndrome in 1, postoperative seizures in 1 and truncal valve plasty revision and postoperatory complete atrioventricular block needing permanent pacemaker implantation during the hospital stay in the last patient.

In group 2, 5 patients presented with postoperative complications: 4 patients had low output cardiac syndrome requiring prolonged inotropic support, and 2 of these required veno-arterial extracorporeal membrane oxygenator (ECMO) support. The first patient underwent postoperative imaging that showed postoperative compression of the single right coronary and was submitted to an additional emergent successful aortopexy with the resolution of the haemodynamic instability. After that, the patient required veno-venous ECMO conversion because of severe respiratory failure caused by pulmonary aspergillosis, which recovered after 22 days of veno-venous ECMO. In the other patient, a veno-arterial ECMO support was necessary because of low cardiac output syndrome associated with pulmonary hypertensive crisis, complicated by *Klebsiella pneumoniae* infection, and acute kidney injury requiring renal replacement therapy (hemofiltration). One last patient required a postoperative right PA stent and left pulmonary angioplasty on the 14th postoperative day because of the evidence of bilateral PA stenosis at postoperative catheterization. The mean hospital stay was 19.2 ± 12.7 days in group 1 and 35.3 ± 44.4 days in group 2. Patients' demographics, intraoperative and postoperative characteristics are summarized in Table 1.

Follow-up

Data are summarized in Table 2. At a global median FU time of 7.9 years (IQR 1.8–13.1; group 1, 11.6 years, IQR 6.9–17.4, and group 2, 3.5, IQR 0.8–9.2, $P = 0.02$) with an FU-completeness of 100%, 6 patients (5 in group 1 and 1 in group 2) died (overall mortality 21%). Causes of death were low output cardiac syndrome and cardiac arrest in 4 patients (3 months, 2, 8 and 8.4 years after repair) and sudden death of unknown aetiology at home in 2 patients (both 2 months after surgery).

A surgical reoperation was required in 9 patients in group 1, after a median time from the repair of 1.32 years (0.86–3.61), and 1 patient in group 2, after 2.3 years.

In group 1, 7 patients presented with significant conduit or PA stenosis (in one of these, a balloon dilatation had been performed 5 years earlier), while 1 patient had a significant residual VSD shunt and the last patient developed a giant RVOT aneurysm; besides, 1 patient developed concomitant significant truncal valve regurgitation. The prosthetic conduit was replaced with a larger prosthetic conduit in only 3 cases (Contegra 18, 20 and 22 mm), while, in the other patients, an RVOT plasty was performed using either a pulmonary homograft monocusp patch (3 patients) or a heterologous pericardial patch (1 patient). One patient presented with pulmonary overflow caused by a large residual VSD, which was closed with a patch 8 months after repair. One patient required contemporary pulmonary and aortic valve replacement after 14 years. In group 2, 1 patient only presented with right intraventricular stenosis and underwent a subpulmonary stenosis resection 2.3 years after repair.

Three patients (group 1) underwent a second surgical reoperation: 1 patient had RVOT re-enlargement plasty using a polytetrafluoroethylene (PTFE) patch due to RVOT stenosis 2.7 years after the residual VSD closure; another patient underwent a

Table 2: FU clinical data

	Total (*n* = 29)	Group 1 (conduit) (*n* = 17)	Occurrence rate per person-year (95% CI)	Group 2 (non-conduit) (*n* = 12)	Occurrence rate per person-year (95% CI)	*P*-value
FU time (years), median (IQR)	7.9 (1.8–13.1)	11.6 (6.9–17.4)		3.5 (0.8–9.2)		0.02
Deaths, *n* (%)	6 (21)	5 (29)	0.02 (0.01–0.06)	1 (8)	0.02 (0.00–0.10)	0.70
RVOT stenosis requiring any redo, *n* (%)	10 (34)	9 (53)	0.05 (0.02–0.09)	1 (8)	0.02 (0.00–0.10)	0.32
Surgical reoperation, *n* (%)	9 (31)	8 (47)	0.04 (0.02–0.08)	1 (8)	0.02 (0.00–0.10)	0.38
Interventional procedures, *n* (%)	13 (45)	8 (47)	0.04 (0.02–0.08)	5 (42)	0.09 (0.03–0.20)	0.20
Any reoperation, *n* (%)	19 (66)	13 (76)	0.07 (0.04–0.12)	6 (50)	0.10 (0.04–0.22)	0.40
Any adverse events, *n* (%)	22 (76)	16 (94)	0.08 (0.05–0.14)	6 (50)	0.10 (0.04–0.22)	0.66
At least moderate truncal valve regurgitation, *n* (%)	5 (17)	3 (18)	0.02 (0.00–0.05)	2 (17)	0.02 (0.00–0.10)	0.93
	Total (*n* = 21), *n* (%)	Group 1 (*n* = 10), *n* (%)		Group 2 (*n* = 11), *n* (%)		
Echocardiographic assessment at FU						
Right ventricular dysfunction	5 (24)	2 (20)	0.2 (0.0–0.6)	3 (27)	0.6 (0.1–1.8)	0.13
Right ventricular dilatation	5 (20)	2 (20)	0.2 (0.0–0.6)	3 (27)	0.6 (0.1–1.8)	0.13

CI: confidence interval; FU: follow-up; IQR: interquartile range; RVOT: right ventricular outflow tract.

permanent pacemaker replacement through anterolateral left mini-thoracotomy 4 months after RVOT enlargement plasty. The last patient developed left PA atresia and left hemidiaphragm paralysis, which required plication and an unsuccessful left systemic-pulmonary shunt, 1.5 years after RVOT aneurysm resection. Last, 3 patients (group 1) presented with severe RVOT stenosis, treated by RVOT balloon dilatation after 1.1, 7 and 8.1 years after the surgical correction.

Sixteen patients (11 in group 1 and 5 in group 2) had PA/PAs stenosis at FU. In group 1, 4 patients underwent a PA patch plasty during the surgical reoperation with various materials (PTFE, homograft, porcine extracellular submucosa, heterologous pericardium) after 2 months and 1.5, 2.7 and 15 years from repair. The other 7 patients underwent percutaneous balloon dilatation of the PAs (in 3 cases, multiple procedures for the same patient) at a mean time of 7 years (1.5–11.4) from surgical operation. In group 2, all 5 patients underwent percutaneous balloon dilatation of the PA/PAs (2 patients had multiple balloon dilatations) at a mean time of 6 months (2.3–8.5) from surgical reoperation.

Last, a percutaneous Melody pulmonary bioprosthesis was implanted in 3 patients (group 1) after 11.6, 12.5 and 13.2 years from the surgical repair. One last patient required a percutaneous atrial septal defect closure (Amplatzer) after 21.6 years from repair.

Last echocardiographic evaluation (in 21 survivors, 91.3%) showed that 5 patients (2 in group 1 and 3 in group 2, Table 2) presented RV dysfunction and RV dilatation at FU.

Statistical analysis results

Overall, at least one AE occurred in 22 (76%) patients at FU, and specifically in 16 in group 1, 6 in group 2. Adverse events rates were similar between groups except for truncal valve regurgitation which occurred significantly less in group 1. At Kaplan-Meier analysis (Fig. 3), overall survival at 1, 5 and 10 years resulted 88.2%, 82.4%, and 67.4%, respectively in group 1 [mean survival 209 months, confidence interval (CI) 95%, 153–265; median survival undefined], and 91.7%, 91.7% and 91.7%, respectively in group 2 (mean survival 136 months, CI 95%, 114–160, P = 0.38; median survival undefined). Overall freedom from surgical operation at 1, 5 and 10 years was 80.7%, 60.5% and 53.8% in group 1 (mean 110 months, 95% CI 68–152; median 170 months) and 100%, 85.7% and 85.7%, respectively, in group 2 (mean 132 months, 95% CI 100–163; P = 0.1; median undefined). Last, event-free survival at 1, 5 and 10 years at 1, 5 and 10 years was 70.6%, 35.3% and 17.6%, respectively, in group 1 (mean 58 months, 95% CI 26–89; median 24 months) and 65.6%, 43.8% and 43.8%, respectively, in group 2 (mean 73 months, 95% CI 31–114; P = 0.4; median 29 months). We did not find any significant differences among groups of patients in which different conduit or RV–PA conduitless repairs were performed.

DISCUSSION

We report our experience in RV–PA reconstruction during TA repair, comparing the more recent conduitless technique with the more traditional conduit replacement. As described in Fig. 4, our experience with conduitless technique has increased since 2004. Our series shows that conduitless repair of TA has good long-term outcomes, which are comparable with the more traditional conduit repair approach. In addition, the onset of AE seems to be lower in the conduitless group.

Early repair of TA is indicated to avoid progression towards CHF and pulmonary vascular obstructive disease. It is a complex and high-risk procedure that includes separation of pulmonary from the systemic circulation, VSD closure and the reconstruction of the RV–PA continuity, which implies the use of a valved conduit with all the known drawbacks of somatic-conduit mismatch that occur over time. In fact, due to the lack of growth and accelerated conduit degeneration (mostly seen in <12 months patients [14]), conduit replacement is inevitable in the long term and exposes the patient to the substantial risk of a reoperation [1]. Lund *et al.* [15] have previously reported that freedom from any RVOT or PA reintervention in patients who had undergone TA complete repair was 68 ± 4% at 1 year and 48 ± 5% at 2 years. Transcatheter balloon dilatation of RVOT or PA and transcatheter pulmonary valve implantation have generally delayed the time of the reoperation [15] but have not reduced the overall reoperation rate. Despite that, the risk for conduit replacement is

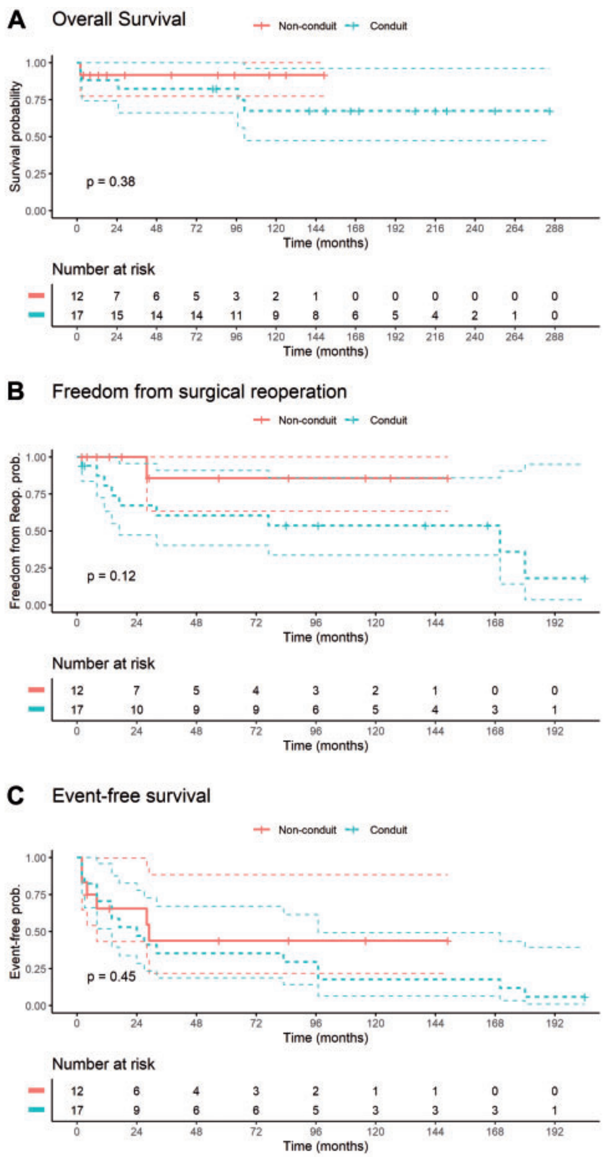


Figure 3: Kaplan–Meier curves for survival (**A**), freedom from surgical intervention (**B**) and event-free survival (**C**). Despite not significantly different, there is a trend of better survival in group 2 (non-conduit). Dotted lines represent corresponding 95% confidence interval. P-value is derived from the log-rank test.

nowadays acceptable, with a mortality risk as high as 2.88% according to ECHSA database [16].

Since the first successful surgical repair of TA without using a valved conduit [17], several conduitless techniques have been described [3, 9, 18], but long-term outcomes are still unclear. In our experience, this technique has been safe, reliable, reproducible and with good late outcomes in terms of haemodynamic performance, growth ability, with lower morbidity when compared to conduit use.

However, we suggest avoiding this technique when the distance between the right ventriculotomy and the PA is too broad, because this may cause critical tension at the anastomotic site, which may cause early postoperative bleeding and

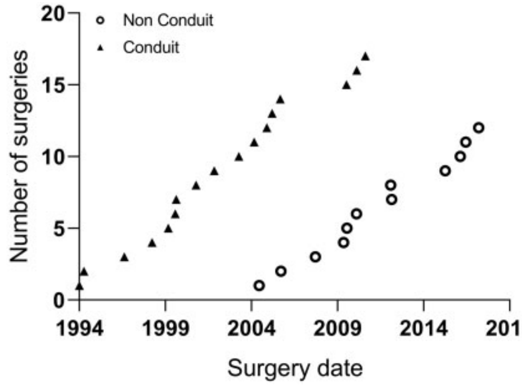


Figure 4: Patient per year plot figure showing our increasing experience with conduitless technique since 2004.

haemodynamic instability, but also an increased rate of obstruction in the long term. Furthermore, in the attempt to mobilize and anastomose the mean PA with the RV, there may be an increased risk for stenosis at the level of the PAs, due to the distortion of PA geometry. To avoid this complication, Barbero-Marcial *et al.* [8] first described the reconstruction of RV–PA. In our experience, the interposition of the LAA to create the posterior wall of the RVOT has been a valid alternative to a conduit for the RV–PA continuity reconstruction since it ensures a reduced anastomotic tension with living and growing autologous tissue (the LAA itself) and is not depending on the anatomical type of TA. In addition, we believe that the LAA for a conduitless technique is better used when it is enlarged, since it allows a larger pathway for the RVOT reconstruction. In our series, we did not note such complications since the utilization of the LAA was useful to release tension and we did not note a significant difference in terms of PA stenosis between the 2 groups. However, the patients in group 2 underwent a PA balloon dilatation significantly earlier than those belonging to group 1 (6 months vs 7 years, respectively).

Satisfactory haemodynamic performance has been reported at short-term FU [3]. The LAA has been effectively used to reconstruct the RVOT by Aeba *et al.* [9] in patients with severe pulmonary stenosis or pulmonary atresia in complex heart diseases other than TA, such as tetralogy of Fallot, transposition of great arteries or double outlet RV, with excellent results in short- and mid-term FU. Aguilar *et al.* [10] described the use of LAA in a patient undergoing a TA surgical correction, but no long-term FU is available.

Another potentially important issue when using a conduitless RVOT reconstruction is the absence of a competent pulmonary valve. Low pulmonary vascular resistance at the moment of the repair has always been emphasized to prevent severe postoperative regurgitation in the RV when a pulmonary valve is absent. As described by Lacour-Gayet *et al.* [19], beyond the neonatal period, the conduitless repair seems to be associated with more significant operative mortality, probably because of postoperative pulmonary hypertension. However, this was not true when the repair was performed in the early neonatal period [20, 21]. However, whether the use of a valve-less direct PA-RV anastomosis (with or without the utilization of the LAA) is associated with a significant advantage in terms of reoperation and mortality rates in the long term remains unclear. Since 2004, surgical repair of TA without using a valved conduit for RVOT reconstruction has been our procedure of choice whenever technically feasible. Technical feasibility was characterized intraoperatively mostly by assessing the distance between the right ventriculotomy and the pulmonary arteriotomy and by the quality and dimensions of the LAA, which is usually very dilated due to pulmonary overflow. We addressed the pulmonary valve continence issue by reconstructing always the anterior wall with a monocusp pulmonary homograft patch. In this way, we were able to prevent significant pulmonary valvular regurgitation, at least in the early postoperative course, as described by Turrentine *et al.* [22]. In our series, only 3 patients in group 2 had severe pulmonary regurgitation at FU. It is of note that the absence of a valved conduit was not associated with higher RV dilatation or dysfunction during the echocardiographic FU in the group of patients that we could observe closer and compare to children who had undergone a conduit repair (Table 2).

In our series, we observed an overall higher incidence of AE in patients with a conduit when compared to those without (Table 2). However, since conduit group patients (group 1) have a significantly longer FU, we cannot exclude that this may have affected our findings. In fact, the conduitless technique has been introduced later in our surgical experience, and the FU time is significantly shorter in group 2 (Table 2). Thus, it may justify the fact that our data show that the use of a direct anastomosis to reconstruct the floor of the RVOT (with or without LAA interposition) has very good outcomes in terms of survival, onset of AE and surgical reoperation at FU, despite the difference is not statistically significant (Fig. 2).

Lower risk of reoperation is certainly desirable since it can undoubtedly improve the quality of life of these patients and decrease the risk related to surgical reoperation, such as coronary lesions, infectious and neurological complications and even death. However, a longer FU and broader experience may confirm our preliminary findings.

Limitations

This is a retrospective single-centre study with a limited number of patients, and magnetic resonance imaging data at FU were not available in most patients, and so we could not include them in the analysis. Last, since the conduitless technique is a more recent technique, differences in FU times in the 2 groups are significant and, thus, the generalization of results is difficult.

CONCLUSION

Despite being technically more demanding, a conduitless RVOT reconstruction is a feasible and reproducible technique that may postpone or reduce the frequency of surgical reoperation after TA repair, improving significantly patients' quality of life and possibly life expectancy.

Conflict of interest: none declared.

Author contributions

Massimo A. Padalino: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Validation; Visualization; Writing—original draft. **Bleri Çelmeta:** Data curation; Methodology; Writing—original draft. **Luca Vedovelli:** Data curation; Formal analysis; statistical analysis. **Biagio Castaldi:** Methodology; Writing—review & editing. **Vladimiro L. Vida:** Supervision. **Giovanni Stellin:** Writing—review & editing.

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