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Surgical Treatment of Common Arterial Trunk in Patients Beyond the First Year of Life

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Abstract

Background: Common arterial trunk (*persistent truncus arteriosus*) is a rare cardiac defect requiring surgical repair early in life because of the fast development of pulmonary vascular obstructive disease. We present our institutional experience with patients having common arterial trunk who are diagnosed after one year of age. **Patients and Methods:** Between August 2010 and May 2013, a total of 1,436 patients were treated for congenital cardiac defects at our institution. Common arterial trunk was treated surgically in seven patients older than one year of age (three males, four females; age: 13 months to 5 years, mean: 2.8 $\pm$ 2.04 years). All patients underwent cardiac catheterization in order to determine operability. **Results:** All patients had the aortic dominant type of common arterial trunk. The pulmonary vascular resistance and Qp/Qs ratio before and after oxygen inhalation were mean 9.04 $+$ 4.2 (range: 3.8 and 10.7) wood units and 4.67 $\pm$ 2.3 (range: 3 and 6.5) wood units and 3.3 $+$ 1.8 (range: 1.42 and 5.3) and 4.98 $+$ 2.2 (range: 4 and 6.2), respectively. All patients underwent elective primary repair. The ventricular septal defect was closed in all patients, five with a nonvalved patch and two with a unidirectional check-valved patch. Early postoperatively, patients were sedated, hyperventilated, and received nitric oxide for a minimum of 24 hours. There was no early or late mortality. The mean length of hospital stay was 9.3 $\pm$ 5.7 days, and mean duration of follow-up was 214 $\pm$ 59 days. **Conclusion:** Complete repair of common arterial trunk in patients older than one year of age is feasible in appropriately selected cases. Preoperative cardiac catheterization to assess reactivity of the pulmonary vascular bed is important as are appropriate strategies for postoperative management. Together, these elements make it possible to achieve primary repair with excellent outcomes despite late presentation.

Keywords

common arterial trunk, truncus arteriosus, aorta, pulmonary artery, surgery, conduit

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Introduction

Common arterial trunk (persistent truncus arteriosus) is a form of congenital heart disease with atrioventricular concordance and a solitary arterial trunk that originates from the heart and gives rise to aorta, pulmonary arteries, and coronary arteries.[1] The International Society for Nomenclature of Paediatric and Congenital Heart Disease (http://www.ipccc.net/) offers the following definition for common arterial trunk (truncus arteriosus): common arterial trunk is defined as "a congenital cardiovascular malformation in which a single arterial trunk arises from the heart, giving origin sequentially to the coronary arteries, one or more pulmonary arteries, and the systemic arterial circulation." The pathology accounts around 1% of all congenital cardiac defects.[1-10]

In the current era, most patients are primarily repaired in the neonatal period.[3-6] Staging with early pulmonary artery banding followed by total correction at later ages is now primarily of historical interest.[1,2] Severe pulmonary vascular obstructive disease (PVOD) can occur early due to pulmonary overcirculation with risk of being inoperable. If left untreated, most patients die within the first year of life from severe cardiac failure.

Untreated patients who survive beyond the first year of life are rare and represent a dilemma.[5] In this report, we present our institutional experience with complete repair of common arterial trunk in patients beyond the first year of life.

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Abbreviations and Acronyms

ASD	atrial septal defect
CPB	cardiopulmonary bypass
PVOD	pulmonary vascular obstructive disease
RVOT	right ventricular outflow tract
TEE	transesophageal echocardiography
VSD	ventricular septal defect

Patients and Methods

Institutional records were used to review patient data. Between August 2010 and May 2013, a total of 1,436 patients with congenital heart disease were operated upon at our center. Surgery was offered to seven patients diagnosed with common arterial trunk after the age of one year, five males and two were females. The mean age was 2.8 $\pm$ 2.04 years, ranging from 13 months to 5 years. All patients had type I common arterial trunk according to the classification of Collett and Edwards. Room air oxygen saturations were measured between 58% and 90% (mean: 79.0 $\pm$ 12.80).

No patient had DiGeorge syndrome. All patients had a typical single large nonrestrictive ventricular septal defect (VSD) in the infundibular area. In two cases, there was a secundum-type atrial septal defect (ASD). One patient had patent ductus arteriosus and branch pulmonary artery stenosis. None of the patients had coronary artery anomalies. Truncal valve morphology was bicuspid in one case, tricuspid in four cases, and quadricuspid in two cases. There was mild truncal valvar insufficiency in four patients, moderate insufficiency in one, and mild stenosis in another. Details of echocardiographic findings are presented in Table 1.

All patients underwent preoperative electrocardiography, chest x-ray, transthoracic echocardiography, and cardiac catheterization. The branch pulmonary artery pressures were measured selectively before and after oxygen inhalation. Prior to oxygen inhalation, the mean pulmonary vascular resistance was 9.04 $\pm$ 4.2 wood units (range: 3.8-10.5 wood units) and the pulmonary-to-systemic blood flow ratio (Qp/Qs ratio) was calculated to be 3.3 $\pm$ 1.8 (range: 1.42-5.3). After oxygen inhalation, pulmonary vascular resistance decreased to 4.67 $\pm$ 2.3 wood units (range: 3-6.5 wood units) and Qp/Qs ratio increased to 4.98 $\pm$ 2.2 (range: 4-6.2).

Surgical Procedure

Surgical correction was performed through standard median sternotomy with aortic and bicaval cannulation. Pulmonary artery pressure was measured directly under general anesthesia before initiation of cardiopulmonary bypass (CPB). Right and left pulmonary artery branches were dissected till the prebranching level and looped prior to CPB and snared when the CPB was initiated. The patent ductus arteriosus was ligated, if present (one case) Intravenous phenoxybenzamine (0.1 mg/kg) was added to the CPB prime solution. Antegrade cold blood cardioplegia was used every 20 minutes together with mild hypothermia for myocardial protection. A left atrial vent was inserted through the foramen ovale (or ASD). The pulmonary arteries were exposed through a transverse anterior truncal incision, which allowed accurate removal of the pulmonary artery bifurcation and prevented injury to the truncal valve or left coronary artery. The pulmonary arteries were removed in continuity with the pulmonary arterial bifurcation, and the resultant defect was closed primarily in six patients and with a pericardial patch in one patient. Right ventriculotomy was made through the right ventricular infundibulum, and the VSD was exposed. The VSD was closed with a Dacron patch in five patients and with a unidirectional check-valved Dacron patch in two patients, using continuous suture technique.[12] The decision to use a unidirectional valved patch was based on the pulmonary arterial pressure persisting at systemic level under general anesthesia. A valved conduit was used to provide right ventricle to pulmonary artery continuity in all cases. The ASD or the patent foramen ovale was left open in two patients. The criteria to leave the atrial septum open was the pulmonary artery pressure >3/4 systemic pressure measured in the operating room before CPB. Patients with mild truncal valvar insufficiency or stenosis were managed without valve repair. In one patient with moderate truncal valvar insufficiency, truncal valvuloplasty was performed with commissural approximation with a pericardial pledgeted suture. In another patient, the left pulmonary artery was stenotic, and the distal anastomosis of the valved conduit was extended over the stenotic segment. The sternum was left open in a five-year-old child and closed on the second postoperative day. A 20-mm Contegra valved xenograft (Medtronic, Santa Ana, California) was anastomosed between the right ventricle and the pulmonary arteries in five patients, and a 19-mm Labcor valved conduit (Labcor, Belo Horizonte, Brazil) was used in two cases. The mean cross-clamp and CPB times were 100.6 $\pm$ 29.43 and 137.2 $\pm$ 55.14 minutes, respectively. Intraoperative transesophageal echocardiography (TEE) was used in all patients and revealed satisfactory repair in all without any unintended residual septal defects. The TEE revealed occasional minor right-to-left flow through the check valve in two patients receiving repair with unidirectional check-valved patches. A pulmonary artery catheter was placed through the right ventricle intraoperatively for the purposes of postoperative monitoring, and this was pulled out in the intensive care unit before chest tube removal.

Postoperative Care

Inotropic agents (at least 5 μg/kg/min of dopamine plus 0.5 μg/kg/min of milrinone) were started before weaning CPB. Other agents, such as epinephrine and norepinephrine, were added as needed. All patients received inhaled nitric oxide starting at conclusion of CPB and continued at intensive care unit postoperatively as needed. To prevent sudden rises in right ventricular afterload, sedation and paralysis were maintained with a continuous infusion of fentanyl, midazolam, pancuronium, and nitric oxide in the first 24 to 48 postoperative hours.

Table 1. Echocardiographic Findings.

Patient	Major Cardiac Pathology	Additional Cardiac Defect(s)
1	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), moderate truncal valve insufficiency, bicuspid truncal valve	VSD (outlet large), PDA, LPSVC
2	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), mild truncal valve stenosis and moderate insufficiency, tricuspid truncal valve	VSD (outlet, large), moderate MR, TR, PDA
3	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), mild truncal valve insufficiency, quadricuspid truncal valve	VSD (pm outlet, large), mild MR
4	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), mild truncal valve insufficiency, tricuspid truncal valve	VSD (pm outlet, large), ASD, PDA, branch PS
5	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), mild truncal valve insufficiency, tricuspid truncal valve	VSD (outlet large), mild MR, ASD
6	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), quadricuspid truncal valve	VSD (outlet, large), ASD, LPSVC
7	Type I truncus arteriosus(aortic dominant common arterial trunk[11]), tricuspid truncal valve	VSD (outlet, large), ASD

Abbreviations: VSD, ventricular septal defect; LPSVC, left persistent superior vena cava; MR, mitral regurgitation; TR, tricuspid regurgitation; ASD, atrial septal defect; PDA, patent ductus arteriosus; pm, perimembranous; PS, pulmonary stenosis.

Statistical Analysis

Results classified as "early" are those that occurred before hospital discharge or within 30 days of surgery if the patient was discharged from the hospital before this duration. Specific software SPSS for Windows version 10 (SPSS, Inc, Chicago, Illinois) was used for data analysis. Continuous data were presented as mean $\pm$ standard deviation.

Results

There were no early or late deaths. Following surgery, the systemic and pulmonary artery pressures were measured. The systolic, diastolic, and mean pulmonary artery pressures decreased to 50.6 $\pm$ 9.8, 24.2 $\pm$ 5.3, and 33.8 $\pm$ 8.9 mm Hg from 93 $\pm$ 9.5, 45.2 $\pm$ 5.5, and 66.8 $\pm$ 6.0 mm Hg, respectively. Ventilation time ranged between 18.5 and 59 hours (mean: 24.3 $\pm$ 9.7 hours), and intensive care unit stay was a mean of 3.7 $\pm$ 0.6 days.

Two patients had pulmonary hypertensive crises. Sildenafil 0.5 mg/kg every 6 hours postoperatively was started in all, and the dose was increased to 2 mg/kg gradually. This has been continued for six months postoperatively.

No patients required reintervention for the truncal valve or the right ventricular outflow tract (RVOT) conduit early or during the follow-up period. Discharge echocardiography demonstrated normal left ventricular size and function with no significant residual lesions in any patient. Truncal valve insufficiency was trace or mild in six patients and moderate in another. There was no gradient across the RVOT conduit. The oxygen saturations ranged between 94% and 100% at the time of discharge. All patients had New York Heart Association functional status class I.

During the same time period (August 2010 to May 2013), a total of 67 patients admitted to the clinic were found inoperable due to various reasons. Among them, there were two patients aged 13 months and 18 years with the diagnosis of common arterial trunk to whom surgery was not offered because of Eisenmenger syndrome. These two patients had pulmonary vascular resistance of 12.5 and 11.2 wood units, and Qp:Qs ratio of 1.0 and 1.2, respectively. Their pulmonary vasculature parameters showed no reactivity to oxygen, and they were deemed inoperable.

Discussion

Since the first successful repair by McGoon et al in 1967, excellent results have been achieved in many centers. Complete neonatal repair has been advocated as the procedure of choice. Delaying the operation beyond 100 days increases the risk of PVOD.[13,14]

If left untreated, at least 80% of the patients died before the end of the first year of life, at a mean age of 2.5 months.[1] Williams et al[3] reported 74% mortality at a median age of 11 days in a group of unoperated patients with common arterial trunk. Untreated patients who survive to adult ages are rare, and nearly all cases have severe PVOD and Eisenmenger syndrome. The mean survival for adults with Eisenmenger syndrome secondary to common arterial trunk was 41.5 $\pm$ 5.1 years as reported by Niwa et al,[4] and this is shorter than Eisenmenger syndrome secondary to other causes.[5] Pulmonary vascular obstructive disease may develop as early as three to six months in babies with common arterial trunk.[6]

In the absence of aortic arch pathologies or severe truncal valve insufficiency, the success of the surgery mainly depends on the presence and degree of pulmonary vascular disease. Juaneda and Haworth[15] analyzed the lung biopsy specimens of 23 patients aged between 18 days and 13 years with common arterial trunk. They found that pulmonary vascular changes occurred very early in life; however, these may be reversible in patients younger than one year of age. On the other hand in patients above 3.5 years, pulmonary vascular disease is more severe and irreversible changes have already occurred. The

authors recommended repair before six months, at which time severe pulmonary arterial medial hypertrophy and intimal proliferation can be expected to be unfixed and reversible.[15]

Since development of irreversible pulmonary hypertension is a major risk factor, attempts at decreasing the pulmonary arterial pressure and proceeding with later repair have been made. Takabayashi et al[16] have performed bilateral pulmonary artery banding in their 3.5-month old patient followed by correction ten months later with good outcome. Neonates with complex cardiovascular anomalies or those with common arterial trunk associated with aortic arch obstruction might be critically ill and CPB could further increase the risk. In this rare particular association, that is, common arterial trunk associated with aortic arch obstruction, initial bilateral pulmonary artery banding with delayed total correction has been recommended by some as a safer option.[17-19] Investigators of the Congenital Heart Surgeons Society studies of patients with interrupted aortic arch however, have advocated one-stage repair of common arterial trunk with interrupted aortic arch as the optimal management.[20,21]

The current surgical approach to common arterial trunk, regardless of the type, is closure of the VSD committing the common trunk to the left ventricle, separation of the pulmonary arteries from the common trunk, and RVOT reconstruction associating the pulmonary arteries with the right ventricle, together with repair of coexisting cardiac anomalies. When there is significant truncal valve insufficiency or stenosis, repair is preferred; however, in extreme cases valve replacement may be necessary. Poor prognostic factors include coronary artery anomalies, aortic arch pathologies, age at repair greater than 100 days, and the development of pulmonary vascular disease at an early age.[1,2,22]

The literature includes few reports of patients with common arterial trunk who were referred for treatment late.[5,23-26] Nearly all of them are inoperable cases due to Eisenmenger syndrome for whom heart and lung transplantation is the only option. Late presenting patients present challenges in regard to assessment of operability, management of right ventricular dysfunction from long-standing pulmonary artery hypertension, and increased risk of postoperative pulmonary hypertensive crisis. Postoperative pulmonary hypertension has still remained a major risk factor for mortality and morbidity following the repair of congenital heart defects.[15] The progression of pulmonary vascular disease after operative closure of VSD with the development of severe pulmonary hypertension and death has been reported.[27] Assessing operability only on the basis of diagnostic modalities might be misleading. The total clinical picture should be taken into consideration to determine operability. High arterial oxygen saturation, hyperdynamic precordium, and signs of left ventricular volume overload suggest clinical absence of irreversible pulmonary vascular disease. We observed that a lower pulmonary-to-systemic arterial diastolic pressure ratio and high pulmonary-to-systemic flow ratio are significant predictors of a smooth postoperative course, even in patients with systemic level pulmonary artery pressure.

Acute right ventricular decompensation due to pulmonary hypertensive crises is a serious problem in this group of patients. Addition of nitric oxide and sildenafil to the postoperative management has become routine in our practice. A unidirectional flap-valved VSD patch to allow right-to-left shunting and maintenance of adequate cardiac output in the case of pulmonary hypertensive crises has been reported.[12] We used a unidirectional checked-valved VSD patch in two patients; however, no major shunt has been detected during the postoperative stay or follow-up, and the oxygen saturations ranged between 94% and 100% at the time of discharge in these patients. Unidirectional check-valved VSD closure is preferably performed in challenging cases at our institution, and we believe that a unidirectional checked-valved patch may provide safety against sudden drop of cardiac output in the setting of abrupt rises in pulmonary vascular resistance.

Use of the Contegra xenograft in the high-pressure systems has been concerning and can lead to early conduit degeneration.[27-30] In our experience, there were no reoperations for conduit degeneration during the follow-up period. This might be due to large diameter of conduits used. The 214 $\pm$ 59 days follow-up of our cohort indicated satisfactory results with the use of Contegra conduits. However, this is a too short follow-up to allow definitive conclusions on the behavior of Contegra conduits in high-pressure systems. The major limitations of our study are the small number of patients, the short follow-up, the absence of lung biopsies to evaluate PVOD, and the retrospective nature of the analysis. However, all our patients did well with primary repair based on the preoperative evaluations that were performed appropriately and precisely. None of them required intermediate-staged efforts such as pulmonary artery banding.

In conclusion, the number of unrepaired patients with common arterial trunk without pulmonary stenosis surviving beyond one year of age is very low. Our study indicates the feasibility of surgical correction with good early- and mid-term results in appropriately selected cases. Preoperative cardiac catheterization to assess reactivity of the pulmonary vascular bed is important, as are appropriate strategies for postoperative management. Together, these elements make it possible to achieve primary repair with excellent outcomes despite late presentation.

Declaration of Conflicting Interests

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References

1. Selvan JP, Uthaman B, Abushaban L, et al. Long-term follow-up of persistent truncus arteriosus: Kuwait experience. *Med Princ Pract*. 2012;21(3): 277-281.
2. McGoon DC, Rastelli GC, Ongley PA. An operation for the correction of truncus arteriosus. *JAMA*. 1968;205(2): 69-73.

3. Williams JM, de Leeuw M, Black MD, Freedom RM, Williams WG, McCrindle BW. Factors associated with outcomes of persistent truncus arteriosus. *J Am Coll Cardiol*. 1999;34(2): 545-553.
4. Niwa K, Perloff JK, Kaplan S, Child JS, Miner PD. Eisenmenger syndrome in adults: ventricular septal defect, truncus arteriosus, univentricular heart. *J Am Coll Cardiol*. 1999;34(1): 223-232.
5. Chiaw TH, San TR, Le TJ. An adult with truncus arteriosus and unilateral pulmonary hypertension. *Congenit Heart Dis*. 2007; 2(6): 433-437.
6. Bove EL, Beekman RH, Snider AR, et al. Repair of truncus arteriosus in the neonate and young infant. *Ann Thorac Surg*. 1989; 47(4): 499-506.
7. Wilson J. Description of a very unusual malformation of the human heart. *Philos Trans R Soc Lond*. 1798;18: 346-356.
8. Humphreys E. Truncus arteriosus communis persistens; criteria for identification of common arterial trunk, with report of case with four semilunar cusps. *Arch Pathol*. 1932;14: 671.
9. Collett RW, Edwards JE. Persistent truncus arteriosus: a classification according to anatomic types. *Surg Clin North Am*. 1949; 29(4): 1245-1270.
10. Van Praagh R, Van Praagh S. The anatomy of common aorticopulmonary trunk (truncus arteriosus communis) and its embryologic implications: a study of 57 necropsy cases. *Am J Cardiol*. 1965;16(3): 406-425.
11. Russell HM, Jacobs ML, Anderson RH, et al. A simplified categorization for common arterial trunk. *J Thorac Cardiovasc Surg*. 2011;141(3): 645-653.
12. Ugurlucan M, Arslan AH, Yildiz Y, Cicek S. eComment. An oval-shaped unidirectional check-valved patch for treating ventricular septal defects. *Interact Cardiovasc Thorac Surg*. 2012; 14(6): 702. doi:10.1093/icvts/ivs145.
13. Bove EL, Lupinetti FM, Pridjian AK, et al. Results of a policy of primary repair of truncus arteriosus in the neonate. *J Thorac Cardiovasc Surg*. 1993;105(6): 1057-1106.
14. Hanley FL, Heinemann MK, Jonas RA, et al. Repair of truncus arteriosus in the neonate. *J Thorac Cardiovasc Surg*. 1993; 105(6): 1047-1056.
15. Juaneda E, Haworth SG. Pulmonary vascular disease in children with truncus arteriosus. *Am J Cardiol*. 1984;54(10): 1314-1320.
16. Takabayashi S, Shimpo H, Yokoyama K, Kajimoto M. Truncus arteriosus repair after palliative bilateral pulmonary artery banding. *Gen Thorac Cardiovasc Surg*. 2007;55(1): 35-37.
17. Bockeria L, Berishvili D, Svobodov A. eComment: Re: staged biventricular repair for persistent truncus arteriosus with aortic arch obstruction following bilateral pulmonary artery banding. *Interact Cardiovasc Thorac Surg*. 2011;12(2): 283.
18. Hoashi T, Kagisaki K, Oda T, Ichikawa H. Staged biventricular repair for persistent truncus arteriosus with aortic arch obstruction following bilateral pulmonary artery banding. *Interact Cardiovasc Thorac Surg*. 2011;12(2): 281-283.
19. Kobayashi T, Miyamoto T, Kobayashi T, et al. Staged repair of truncus arteriosus with interrupted aortic arch: adjustable pulmonary artery banding. *Ann Thorac Surg*. 2010;89(3): 973-975.
20. Konstantinov IE, Karamlou T, Blackstone EH, et al. Truncus arteriosus associated with interrupted aortic arch in 50 neonates: a congenital heart surgeons society study. *Ann Thorac Surg*. 2006;81(1): 214-222.
21. McCrindle BW, Tchervenkov CI, Konstantinov IE, et al. Risk factors associated with mortality and interventions in 472 neonates with interrupted aortic arch: a congenital heart surgeons society study. *J Thorac Cardiovasc Surg*. 2005;129(2): 343-350.
22. Russell HM, Pasquali SK, Jacobs JP, et al. Outcomes of repair of common arterial trunk with truncal valve surgery: a review of the society of thoracic surgeons congenital heart surgery database. *Ann Thorac Surg*. 2012;93(1): 164-169.
23. Guenther F, Frydrychowicz A, Bode C, Geibel A. Cardiovascular flashlight. persistent truncus arteriosus: a rare finding in adults. *Eur Heart J*. 2009;30(9): 1154.
24. Espínola Zavaleta N, Muñoz Castellanos L, González Flores R, Kuri Nivón M. Common truncus arteriosus in adults [in Spanish]. *Arch Cardiol Mex*. 2008;78(2): 210-216.
25. Gutierrez PS, Binotto MA, Aiello VD, Mansur AJ. Chest pain in an adult with truncus arteriosus communis. *Am J Cardiol*. 2004; 93(2): 272-273.
26. Lopes LM, Silva JP, Ld Fonseca, Meiken S, Salvador AB, Fernandes GS. Atypical truncus arteriosus operated at 28 years of age: importance of differential diagnosis. *Arq Bras Cardiol*. 2011;97(2): e29-e32.
27. Momma K, Takao A, Ando M, Nakazawa M, Takamizawa K. Natural and postoperative history of pulmonary vascular obstruction associated with ventricular septal defect. *Jpn Circ J*. 1981; 45(2): 230-237.
28. Shebani SO, McGuirk S, Baghai M, et al. Right ventricular outflow tract reconstruction using Contegra valved conduit: natural history and conduit performance under pressure. *Eur J Cardiothorac Surg*. 2006;29(3): 397-405.
29. Walther T, Daehnert I, Hambsch J, Mohr FW, Janousek J, Kostelka M. Bovine jugular vein conduit for right ventricular outflow tract reconstruction: evaluation of risk factors for mid-term outcome. *Ann Thorac Surg*. 2006;82(4): 1308-1315.
30. Dave H, Mueggler O, Comber M, et al. Risk factor analysis of 170 single-institutional Contegra implantations in pulmonary position. *Ann Thorac Surg*. 2011;91(1): 195-302.