Novel concepts and early results of repairing common arterial trunk

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Abstract

OBJECTIVES: Common Arterial Trunk (CAT) continues to have a very poor prognosis globally. To address that, we have developed a novel technique targeting key concepts for the correction of all components of the anomaly, using autologous arterial tissue. This aims to enhance results, availability worldwide, and importantly to avoid the need for repeated reoperations.

METHODS: From January 2019 to 4 January 2021, all patients with isolated CAT had repair of the defect using autologous arterial trunk tissue with direct right ventricle (RV) to pulmonary artery (PA) connection. Clinical outcomes, follow-up which included multi-slice computed tomography 3D segmentation and 4D cardiovascular magnetic resonance flow, are presented.

The first three authors contributed equally to this work.

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RESULTS: Twenty patients were included in the study (median age 4.5 months). There were 2 hospital deaths due to systemic infection and pulmonary hypertensive crisis, respectively. Following discharge all patients remained asymptomatic with no signs of heart failure and improved pattern of growth (median follow-up: 8 months). Early postoperative 3D segmentation showed a conical shaped neo-right ventricular outflow channel connecting the body of the RV to the main PA through a valveless ostium, and normal crossing of PA and neo-aorta. 4D cardiovascular magnetic resonance pattern of flow showed normal rapid laminar flow through the atriopulmonary arteries followed by a vortex towards the outflow tracts. There was laminar flow through the neo-aorta and neo-PA with velocity not exceeding 2.5 m/s. The PA regurgitant fraction was 25 ± 5% and was limited to early diastole.

CONCLUSIONS: The initial results of utilizing the key concepts, using autologous arterial tissue for the repair of CAT, are encouraging, both clinically and by multimodality imaging.

Keywords: Morphodynamism • optimizing geometry and function • neo-RV outflow tract • multimodality imaging • 4D flow magnetic resonance imaging

INTRODUCTION

Common Arterial Trunk (CAT) is a serious congenital anomaly which, if uncorrected in neonatal life, carries a very poor prognosis [1, 2]. Since the introduction of the first operation to correct the anomaly by McGoon et al. [3] in 1968, there have been considerable improvements in the surgical treatment of this anomaly, using a variety of techniques [4–8]. However, several major problems remain to be addressed for surgical repair to have the desired impact on the very poor global natural history of the disease. Most of these problems result from incomplete correction of the pattern of flow, use of foreign material and not universally affordable conduits. These result in worse outcomes and repeated reoperations at frequent intervals.

We here describe novel concepts for the repair of CAT using autologous arterial tissue combined with restorative procedures aimed at restoration of the pattern of flow and morphodynamism.

SURGICAL TECHNIQUE

The heart and great vessels are exposed through a median sternotomy. The main pulmonary artery (PA) and its branches are mobilized widely from the arterial trunk and well into the hila of both lungs. In addition, the aortic arch and its branches are extensively mobilized. The 2 main pulmonary arteries are snared immediately after establishing cardiopulmonary bypass (with bicaval cannulation and arterial return to the aortic arch). The patient is then cooled to 28°C, followed by clamping the ascending aorta immediately proximal to the origin of the right brachiocephalic artery. The ascending aorta is transected, 3 mm below the arterial clamp. Lower down, the arterial trunk is transected 1–2 mm above the top end of the truncal valve commissures (Fig. 1A and B). This results in a free mobilized segment of the arterial trunk bearing the origin of the pulmonary arteries. The origin of the main PA in type I [9, 10] or the segment of the arterial trunk bearing the 2 PA branches in type II, is separated from its adjoing arterial trunk wall. This generates a semi-circular free patch which will be used later to refashion the neo-right ventricular outflow tract (RVOT) (Fig. 1C). Care is taken to leave a “lip” of arterial wall around the ostium of the PA(s) where a well-defined ridge is usually present (Fig. 1B and C).

An inverted triangular window is made in the anterior wall of the right ventricle (RV) just below the attachment of the truncal valve, to avoid undue tension of the neo-RVOT anastomosis (Fig. 1D). The base of the triangle measures 1.5–2 cm with its apex extending into the trabecular part of the RV avoiding epicardial coronary vessels. Obstructive muscular bands inside the RV are resected to allow an adequate communication between the RV cavity and the neo outflow chamber. Trans-ventricular VSD closure is then performed, using a Gore-Tex patch of appropriate size and shape, to ensure unobstructed blood flow from the left ventricle (LV) to the neo-aorta.

The mobilized main PA is then anastomosed to the RV window, starting by the posterior edge of its ostium. While ensuring proper orientation of the 2 PA branches without tension, torsion or kinking (Fig. 1D). The previously mobilized truncal wall patch, which has a natural curve, is then tailored and used to create the roof of neo-RV outflow chamber (Fig. 1E and F).

Appropriate repair should aim at defining and correcting, whenever possible, all of the component parts of the anomaly [7]. Pre-operatively, the root of the ventriculo-arterial connection is dilated, with heterogeneous deformities affecting the geometry, structure and function of its components. The truncal valve exhibits different forms of abnormalities and deformities. In patients, with a quadricuspid valve and thin preserved 4 leaflets, tricuspidization is performed. This technique entails excision of the non-coronary sinus, leaflet, annulus, and importantly remodelling the subvalvular region to ensure smooth flow of blood from the LV to the neo root (Fig. 2A–D). In patients with tricuspid or bicuspid trunk valve, techniques similar to those developed and used for bicuspid aortic valve are used [11]; this should be combined with remodelling of the enlarged sinuses and creation of a sinotubular junction to match the distal end of the ascending aorta (Fig. 2E).

PATIENTS AND METHODS

Between January 2019 and 4 January 2021, 20 patients (aged 1–26 months, median: 4.5 months) underwent repair of CAT (Van Praagh types 1 and 2) [8] using the novel technique (Supplementary Material, Table S1). Patients with interrupted aortic arch or associated extra-cardiac congenital anomalies (hypoplastic trachea,
Figure 1: Use of autologous arterial trunk tissue for repair (see text) (A–F).

Figure 2: Tricuspidization of a quadricuspid truncal valve (A–D) and remodelling of the neoaortic sinuses (E).
imperforate anus) were excluded. All patients were severely symptomatic with varying degrees of arterial desaturation, and heart failure. Following discharge all patients were followed up regularly for periods varying from 1 to 18 months. Informed consents were obtained for all patients before enrolment in the study.

Multimodality imaging

**Multislice computed tomography.** All patients had pre-operative (except for one) and early postoperative (1-4 weeks) multi-slice computed tomography using Siemens Somatom Definition 128 (Siemens, Erlangen, Germany). Three-dimensional segmentation was performed using DICOM images on Mimics innovation suit software (Materialise, Leuven, Belgium). Four patients had additional late investigation (at 5, 11, 13, and 15 months, postoperatively).

**Magnetic resonance imaging and 4D flow mapping.** Five patients underwent postoperative magnetic resonance imaging scanning using Siemens Magnetom Aera (Siemens, Erlangen, Germany) to evaluate the flow pattern in both ventricles, neo-RVOT, pulmonary arteries and neo-aorta (acquisition details are shown in the Supplementary Material).

RESULTS

Survival

There were 2 hospital deaths due to systemic infection in 1 and pulmonary hypertensive episode in the other. There were no deaths during the follow-up period (1-18 months, median: 8 months).

Clinical progress

Following discharge, all patients remained asymptomatic, with no signs of right or left ventricular failure. The pattern of growth improved substantially following operation (Supplementary Material, Table S2). The follow-up echocardiograms showed good left and right ventricular systolic function, with mild dilation of the RV, trivial to mild tricuspid regurgitation and free pulmonary regurgitation. The estimated RV peak systolic pressure varied from one-third to half that in the systemic circulation except for 1 patient who had a ratio of 60%. The median RV systolic pressure was 25 mmHg (20-50); while the median peak systolic gradient from the RV to PA was 8 mmHg (4-35). Neo-aortic regurgitation was trivial to mild in 16 and moderate in 2 patients (Supplementary Material, Table S3).

Computed tomography segmentation

**Pre- and early postoperative appearances.** The pre-operative segmentation showed the variable relationship of the arterial trunk to the ventricular mass. The origin of the arterial trunk was related equally to the right and LV in all patients except in patient 2 where it came predominantly from the RV (Fig. 3). Postoperatively, the normal crossing of the PA and neo-aorta [12] was restored with the main PA arising from the neo-RVOT (Fig. 3).

**Right and left ventricular size and function.** All patients (apart from 1 who had an urgent operation) underwent pre- and early postoperative segmentation of the 2 ventricles in systole and diastole. Typical examples are shown in Fig. 3. Volumetric analysis of both ventricles was performed in 10 consecutive patients (Supplementary Material, Table S4). In addition, 4 patients underwent late investigations (at periods of 5, 11, 14 and 15 months after operation) (Table 1). This showed no consistent changes, in size or function of the left or RV, compared to early postoperative. However, in 1 patient, there was an apparent increase in end-diastolic volume of the RV with the preservation of ejection fraction (Table 1).

**Neo-RVOT.** Postoperative segmentation showed a neo-RV outflow chamber, which had a conical shape, similar to a normal RVOT. The neo-RVOT connected the body of the RV to the main PA (Fig. 4A) through a valveless pulmonary ostium, which had a similar diameter to the main PA; this had the effect of limiting

![Figure 3: Pre- and early postoperative 3D segmentation of both ventricles and great arteries (at end-systole and end-diastole for 3 representative patients) (see text).](image-url)
the amount of pulmonary regurgitation. An example of pattern of RV contraction showing morpho-dynamism comparable to normal is shown in Video 1. It is notable that the neo-RVOT changes its size and shape during the cardiac cycle with no paradoxical movement or dilatation.

Size and topology of the main PA and bifurcation. Postoperatively, the main PA crosses in front of the neoaortic root in a manner similar to that observed in normal hearts (Fig. 3) [12]. The PA bifurcation was preserved with an angle approximating that seen pre-operatively. This could have important clinical implications of avoiding turbulence and late stenosis (Fig. 4B).

Size and shape of neo-aorta. Postoperatively, the neo-aorta showed diminution in length and circumference. A typical example of the postoperative topology, size and shape of the neo-aorta (as compared to the neo-PA) is shown in Fig. 5.

Cardiovascular magnetic resonance with 4D flow

Cardiovascular magnetic resonance examination including 4D flow analysis was performed for 5 patients during their follow-up, median 13 months (11–14) postoperatively.

Pattern of flow.

LV

The pattern of flow through the LV inflow showed “normal” rapid laminar central flow in early diastole. This is followed by an anteriorly directed vortex towards the left ventricular outflow tract (LVOT). During peak systole, ejection through the neo-LVOT showed laminar flow with velocity not exceeding 1.5 m/s. In addition, there was an abnormal vortex below the neoaortic valve in the LVOT, due to a recess created during repair of the ventriculo-arterial defect (Fig. 6A).

RV

Similar to the pattern of flow observed in LV, there was early laminar flow through the tricuspid valve during early diastole (Fig. 6B). This was followed by direct flow from the inflow to the neo-RVOT and laminar flow through the PA in peak systole (Fig. 6B and C).

During early diastole, there was backward flow which appears to stop in late diastole. The measured regurgitation fraction was 25 ± 5% (Fig. 6C and Supplementary Material, Fig. S2).

Pulmonary bifurcation

Both branches showed a laminar accelerated flow during early and peak systole, with peak velocity not exceeding 2.5 m/s (Supplementary Material, Fig. S1). In addition, there was no abnormal vortex formation. The left pulmonary artery received 52% of total pulmonary flow while right PA flow was 48% (Supplementary Material, Fig. S2).

Viscous energy loss

The viscous energy loss in the neo-ascending aorta and neo-main PA was calculated by the method described by A. Barker, M. Markl and colleagues [13] (Fig. 7). This showed minor energy loss in both vessels during systole.

DISCUSSION

Evolution of a concept

Common Arterial Trunk is defined as a single origin of an arterial trunk from the heart, which directly supplies the systemic, coronary and pulmonary circulations. A rational approach to correction of this complex anomaly depends on thorough understanding of the embryological origin, components and natural history of the condition.

The technique described in this paper is designed to recreate near normal anatomy and function while avoiding the use of valve conduits and artificial material. The amount of viscous energy loss [13] was reduced by tailoring the root of the CAT as well as the neo-PA (Fig. 7). The degree of residual pulmonary

| Case 3 | Left ventricle | Right ventricle |
|--------|----------------|----------------|
|        | Pre-operative  | Early postoperative | Late postoperative  |
| ESV (ml) | NA | 5 | 5 | NA | 7 | 7 |
| EDV (ml) | NA | 8 | 8 | NA | 13 | 13 |
| EF (%) | NA | 38 | 38 | NA | 46 | 46 |
| Case 4 |                |                |                |
| ESV (ml) | 9 | 8 | 5 | 12 | 5 | 9 |
| EDV (ml) | 28 | 18 | 22 | 19 | 15 | 39 |
| EF (%) | 68 | 56 | 77 | 37 | 67 | 77 |
| Case 5 |                |                |                |
| ESV (ml) | 9 | 7 | 7 | 7 | 8 | 10 |
| EDV (ml) | 32 | 24 | 22 | 24 | 31 | 31 |
| EF (%) | 72 | 71 | 68 | 71 | 74 | 68 |
| Case 13 |            |                |                |
| ESV (ml) | 6 | 5 | 10 | 6 | 4 | 9 |
| EDV (ml) | 26 | 16 | 23 | 25 | 13 | 27 |
| EF (%) | 77 | 69 | 57 | 76 | 69 | 67 |

EDV: end-diastolic volume; EF: ejection fraction; ESV: end-systolic volume; NA: not available.
Figure 4: Early postoperative 3D segmentation (A) of the right ventricle, neo-right ventricular outflow tract, and pulmonary artery. (B) Pattern of bifurcation of the main pulmonary artery (for 8 representative patients).

Figure 5: 3D segmentation of the size and shape of the great arteries in a representative patient pre- (A) and postoperatively (B and C).
Regurgitation is controlled by tailoring the size of communication between the neo-RVOT and main PA. Postoperative multimodality imaging was used as a surrogate marker for long-term results. The use of autologous arterial tissue for the repair allows this technique to be eminently usable in neonates and importantly in low- and middle-income countries. In addition, it avoids the need for frequent reoperations with associated morbidity and mortality [4, 8, 14]. Recreation of the neo-RVOT should enable percutaneous placement of a pulmonary valve [15, 16] when the child is fully grown; this could be done in the future using a tissue-engineered valve [17, 18].

Up till recently, it was thought that the anomaly resulted from failure of the development of the spiral septum inside the single vessel arising from the heart. Seminal work by Anderson et al. [19, 20] suggests that the anomaly involves the endocardial cushions, resulting in what they called, ventriculo-arterial septal defect. This explains the presence of the markedly dilated arterial orifice, the presence of quadricusp valve in some patients and the absence of a right ventricular outflow. These abnormalities should be addressed during a restorative operation.

**Normalization of flow**

The pattern of flow in the heart and great vessels is closely linked to the morphology and topology of the chambers [12] and

![Video 1: Pattern of right ventricle contraction, showing morpho-dynamism, as well as changes in size and shape with no evidence of dilatation or paradoxical movement (15 months after repair of Common Arterial Trunk using the current technique) (left) as compared to normal (right).](image)

![Figure 6: Postoperative 4D pattern of flow in left ventricle (row A), showing parallel flow lines through the inlet during early diastole with a normal vortex directly from the inflow to outflow in late diastole followed by parallel flow lines with no acceleration in the neo left ventricular outflow tract and no evidence of trunk valve regurgitation. In the right ventricle (rows B and C), there is evidence of mild pulmonary regurgitation during early diastole with a resulting vortex in the neo-right ventricular outflow tract. During late diastole there is minimal pulmonary regurgitation. During peak systole there is a mild acceleration of flow in the proximal pulmonary artery.](image)
coupled to myocardial contraction, in what is termed morphodynamics [21, 22]. Effective repair of complex congenital defects should aim at preserving or restoring morphodynamics, and ventriculo-ventricular interaction, rather than introducing additional abnormalities, such as sudden change in luminal diameter or interference with contractile function (Video 1). The classical repair of CAT entails insertion of valved conduits, which introduces additional functional abnormalities, both in the short and long terms, and importantly requires re-replacement [3].

The truncal root

Pre-operatively, the common root of the ventriculo-arterial connection is dilated and requires tailoring during the repair, to reduce the amount of regurgitation, match the much smaller distal aorta and avoid energy loss during ventricular ejection. In patients with quadricuspid valve and normal leaflets, tricuspidization could restore structure and function (Fig. 2). In patients with tricuspid or bicuspid trunk valve, techniques similar to those developed and used for bicuspid aortic valve are used [11].

Pulmonary artery and branches

An essential feature of the new technique is wide mobilization of the main PA and its branches into the Hila (Fig. 1). This enables direct anastomosis of the main PA to the relatively small window in the anterior surface of the RV just below the origin of the arterial trunk. In addition, the mobilized PA and its branches allow preservation of the angle of bifurcation of the main PA and avoid turbulent flow of blood (Fig. 6). This could have very important long-term implications with regard to arterial wall structure and function [23]. Another detail concerns preservation of 2–3-mm lip of the arterial trunk around the orifice of the main PA (Fig. 1) to guard against future stenosis of the anastomosis to the RV.

Creating a viable RVOT

A viable contractile RVOT contributes to overall function of the RV [24]. In the technique described here, a viable neo-RVOT was created (Fig. 4). This enabled a ‘normalized’ pattern of flow from the inflow part of the RV to the PA with preserved morphodynamics (Fig. 6B and Video 1).

Timing of correction

If uncorrected, CAT carries an extremely poor prognosis with an estimated 80% mortality during the first year of life [2]. Therefore, it is essential to apply corrective operations in neonatal life. The technique described here allows this to be achieved as the autologous arterial tissue is capable of growth and adaptation without excessive dilatation or stenosis. In this series, the technique was used in several patients beyond the neonatal period. This policy could be complicated by the presence of early pulmonary vascular disease. For this reason, special care was taken to limit the communication between RV and RVOT.

Figure 7: Peak and temporal viscous energy loss in the neo-ascending aorta, A and B, respectively, as well as in the neo-main pulmonary artery, C and D.
PA to decrease the degree of regurgitation in the presence of mild pulmonary hypertension.

Utilizing autologous arterial tissue

In patients with CAT, there is a marked enlargement of the arterial trunk both in length and diameter. This ‘redundant’ living, autologous tissue can be used for the restorative repair, as a roof for the outlet chamber.

Free arterial tissue remains viable and indefinitely is capable of adapting and growing as has been shown in the Ross operation [25, 26]. Although it is not capable of contraction, it constitutes, only two-third of the circumference.

Is the pulmonary valve dispensable in infants?

The notion that the right side of the heart, including the pulmonary valve, is dispensable [27, 28] has been shown to be incorrect, particularly in patients who had large trans-annular patches during the repair of tetralogy of Fallot who require insertion of a pulmonary valve in late childhood or early adulthood [29, 30]. In our series, the degree of regurgitation is limited by the regurgitant orifice, which equals the circumference of the circular anastomosis between the main PA and distal end of the neo-RV outlet chamber. It is hoped that, in the absence of pulmonary hypertension, this degree of pulmonary regurgitation will be tolerated for several years and can be corrected, later in life, using percutaneous techniques [15, 16].

Limitations

The main limitations of this study are the relatively small number of patients and short follow-up. In addition, the study does not include a control group to compare to, in a prospective randomized trial; however, the extensive multimodality imaging acts as a surrogate marker for long-term results. Larger series followed up for longer period of time will be necessary to validate our findings.

CONCLUSIONS AND FUTURE DIRECTIONS

It is hoped that wide application of the novel concepts described in this communication will enhance the overall survival and quality of life of CAT, increase applicability of the operation worldwide, and importantly prevent repeated reoperations.

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Author contributions

Magdi H. Yacoub: Conceptualization; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Supervision; Visualization; Writing—original draft. Hatem Hosny: Conceptualization; Formal analysis; Investigation; Methodology; Visualization; Writing—original draft; Writing—review & editing. Ahmed Affi: Conceptualization; Formal analysis; Investigation; Methodology; Project administration; Supervision; Visualization; Writing—original draft; Writing—review & editing. Mohamed Nagy: Data curation; Investigation; Methodology; Software; Visualization; Writing—review & editing. Ahmed Mahgoub: Investigation; Methodology. Walid Simry: Investigation; Methodology. Mohammad Gibrell AbouZeina: Methodology. Ramy Doss: Methodology. Amr El Sawy: Investigation; Methodology. Software. Nairouz Shehata: Methodology; Software; Visualization. Abdelrahman Elaifi: Data curation; Investigation; Methodology. Hedaa Abdullah: Methodology. Soha Romeih: Data curation; Formal analysis; Investigation; Methodology; Supervision; Validation; Visualization; Writing—review & editing.

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REFERENCES

[1] Marcelletti C, McGoon DC, Mair DD. The natural history of truncus arteriosus. Circulation 1976;54:108–11.
[2] Hoffman JIE. Truncus arteriosus. In: Hoffman JIE (ed). The Natural and Unnatural History of Congenital Heart Disease. Hoboken, New Jersey, United States: Wiley, 2009, 519–30. doi:10.1002/9781444314045.
[3] McGoon DC, Rastelli GC, Ongley PA. An operation for the correction of truncus arteriosus. JAMA 1966;205:69–73.
[4] Bove EL, Lupinetti FM, Pridjian AK, Beekman RH, Callow LB, Snider AR et al. Results of a policy of primary repair of truncus arteriosus in the neonate. J Thorac Cardiovasc Surg 1993;105:1057–66.
[5] Myers PO, Bautista-Hernandez V, del Nido PJ, Marx GR, Mayer JE, Pigula FA et al. Surgical repair of truncal valve regurgitation. Eur J Cardiothorac Surg 2013;44:813–20.
[6] Ebet PA, Turley K, Stanger P, Hoffman JL, Heymann MA, Rudolph AM. Surgical treatment of truncus arteriosus in the first 6 months of life. Ann Surg 1984;200:451–6.
[7] Mavroudis C, Backer CL. Surgical management of severe truncal insufficiency: experience with truncal valve remodeling techniques. Ann Thorac Surg 2001;72:396–400.
[8] Naimo PS, Bell D, Fricke TA, D’Udeleem Y, Brizzard CP, Alphonso N et al. Truncus arteriosus repair: a 40-year multicenter perspective. J Thorac Cardiovasc Surg 2021;161:230–40.
[9] Collett RW, Edwards JE. Persistent truncus arteriosus; a classification according to anatomic types. Surg Clin North Am 1949;29:1245–70.
[10] Van Praagh R. Truncus arteriosus: what is it really and how should it be classified? Eur J Cardio-Thorac Surg 1987;1:65–70.
[11] Schneider U, Kariova I, Giebels C, Ehrlich T, Schaefer H-J. Concepts and techniques of bicuspid aortic valve repair. J Vis Surg 2020;6:3.
[12] Sievers HH, Scharfsweder M, Putman LM. In vitro evaluation of physiological spiral anastomoses for the arterial switch operation in simple transposition of the great arteries: a first step towards a surgical alternative? Interact CardioVasc Thorac Surg 2015;21:157–162.
[13] Barker AJ, van Ooij P, Bandi K, Garcia J, Albaghdadi M, McCarthy P et al. Viscous energy loss in the presence of abnormal aortic flow. Magn Reson Med 2014;72:620–628.
[14] McElhinney DB, Rajasinghe HA, Mora BN, Reddy VM, Silverman NH, Hanley FL. Reinterventions after repair of common arterial trunk in neonates and young infants. J Am Coll Cardiol 2000;35:1317–1322.
[15] de Torres-Alba F, Kaleschke G, Baumgartner H. Impact of percutaneous pulmonary valve implantation on the timing of reintervention for right ventricular outflow tract dysfunction. Rev Esp Cardiol (Engl Ed) 2018;71:838–846.
[16] Biernacka EK, Rużyło W, Demkow M. Percutaneous pulmonary valve implantation-state of the art and Polish experience. Adv Interv Cardiol 2017;13:3–9.
[17] Yacoub MH, Takkenberg JIM. Will heart valve tissue engineering change the world? Nat Clin Pract Cardiovasc Med 2005;2:60–61.
[18] Durko AP, Yacoub MH, Kluin J. Tissue engineered materials in cardiovascular surgery: the surgeon’s perspective. Front Cardiovasc Med 2020;7:1–7.

[19] Russell HM, Jacobs ML, Anderson RH, Mavroudis C, Spicer D, Corcorain E et al. A simplified categorization for common arterial trunk. J Thorac Cardiovasc Surg 2011;141:645–653.

[20] Anderson RH, Mori S, Spicer DE, Brown NA, Mohun TJ. Development and morphology of the ventricular outflow tracts. World J Pediatr Congenit Heart Surg 2016;7:561–577.

[21] Kilner PJ, Yang G-Z, Firmin DN. Morphodynamics of flow through sinusous curvatures of the heart. Biotheriology 2002;39:409–417.

[22] Kilner PJ, Yang GZ, Wilkes AJ, Mohiaddin RH, Firmin DN, Yacoub MH. Asymmetric redirection of flow through the heart. Nature 2000;404:759–761.

[23] Doorly D, Sherwin S. Geometry and flow. Model Simul Appl 2009;1:177–209.

[24] Asmer I, Adawi S, Ganaeem M, Shehadeh J, Shiran A. Right ventricular outflow tract systolic excursion: a novel echocardiographic parameter of right ventricular function. Eur Heart J Cardiovasc Imaging 2012;13:871–877.

[25] Rabkin-Aikawa E, Aikawa M, Farber M, Kratz JR, Garcia-Cardena G, Kouchoukos NT et al. Clinical pulmonary autograft valves: pathologic evidence of adaptive remodeling in the aortic site. J Thorac Cardiovasc Surg 2004;128:552–561.

[26] Yacoub MH, Tsang V, Sarathchandra P, Jensen H, Hughes S, Latif N. Long-term adaptive versus maladaptive remodelling of the pulmonary autograft after the Ross operation. Eur J Cardiothorac Surg 2020;57:977–985.

[27] Sade RM, Castaneda AR. The dispensable right ventricle. Surgery 1975;77:624–631.

[28] Raisky O, Ali WB, Bajolle F, Marini D, Metton O, Bonnet D et al. Common arterial trunk repair: with conduit or without? Eur J Cardiothorac Surg 2009;36:675–682.

[29] Romeo JLR, Takkenberg JJM, Cuypers JAAE, de Groot NMS, van de Woestijne P, Bruining N et al. Timing of pulmonary valve replacement in patients with corrected Fallot to prevent QRS prolongation. Eur J Cardiothorac Surg 2020;58:559–566.

[30] Oosterhof T, Van Straten A, Vliegen HW, Meijboom FJ, van Dijk APJ, Spijkerboer AM et al. Preoperative thresholds for pulmonary valve replacement in patients with corrected tetralogy of Fallot using cardiovascular magnetic resonance. Circulation 2007;116:545–551.