

Common Arterial Trunk Repair at the Red Cross War Memorial Hospital, Cape Town:
A 20-year review of surgical practice and outcomes

Submitted to the University of Cape Town in fulfilment of requirements for the degree
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DECLARATION

I, *Allen Moodley*, hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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Abstract

BACKGROUND: A description of the post-operative outcomes following Common Arterial Trunk (CAT) repair over 20 years before and following the transition to non-conduit repair. Primary outcomes were 30-day and overall, in-hospital mortality for paediatric patients who underwent CAT repair at Red Cross War Memorial Children's Hospital (RCWMCH). Secondary outcomes encompassed (a) Incidence of postoperative complications and (b) medium-term outcomes, including reinterventions, late deaths, and loss to follow-up.

METHOD: A single-centre retrospective study of all consecutive patients who undertook the repair of CAT from January 1999 to December 2018 at RCWMH. Patients with an interrupted aortic arch or previous pulmonary artery banding were excluded.

RESULTS: Fifty-four patients had CAT repair during the study period. Thirty-four (63.0%) patients had a conduit repair, and 20 (37.0%) patients had a non-conduit repair. There were 2 intraoperative deaths. Thirty-day in-hospital mortality was 22.2%. Overall, in-hospital mortality was 29.6%. Twenty-nine (55.8%) of fifty-two patients suffered a postoperative complication.

A total of 38 patients were followed up post-hospital discharge with 11 patients (28.9%) lost to follow-up and 8 (21.1%) late mortalities observed.

The actuarial survival for the conduit group was 77.5%, 53.4% and 44.5% at 6, 12 and 27 months respectively and non-conduit group was 58.6% at 6 months. The overall freedom from revision surgery between the conduit group and non-conduit group was

66.2% vs 86.5%, 66.2% vs 76.9% and 29.8% vs 64.1% at 1, 2 and 8 years respectively.

CONCLUSIONS: No difference in postoperative mortality between the conduit and non-conduit repair. Reintervention rates were lower in the non-conduit group.

Contributions of Authors:

Dr Allen Moodley: Drafting, revision and submission to ethics. Data collection on SPSS. Write up of the manuscript. Revisions as called for by reviewers. Submission of the final manuscript.

Dr Heidi Meyer: Protocol review. Project review. Assistance with logistics at the facility. A critical review of manuscript drafts and revision of the manuscript between AM and herself for the concluding manuscript.

Dr Andre' Brooks: Conceptualization of project. Review of the MMed with AM. Assistance with ethics compliance. A critical review of the final manuscript.

Professor Liesl Zühlke: Critical review of the final draft.

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Table 1. Demographics & Clinical Factors

Variable	All patients (N=54)	In-hospital mortality (N=16)	Survived (N=38)	Chi-square p-value
Age (days), median (IQR)	61.5(44.8-138.8)	49 (31-97)	67 (51.5-147.0)	0.132 [§]
Weight (kg), median (IQR)	3.4 (3.0-4.5)	3.2 (3.0-3.7)	3.6 (3.1-4.7)	0.132 [§]
Sex, n (%)				
Female	27 (50.0)	8 (50.0)	19 (50.0)	0.119 [§]
CAT Type (Van Praagh), n (%)				
1	36 (66.7)	10 (62.5)	26 (68.4)	0.788 [§]
2	16 (29.6)	5 (31.3)	11 (28.9)	
3	2 (3.7)	1 (6.3)	1 (2.9)	
Truncal valve regurgitation, n (%)				
None	12 (22.3)	4(25.0)	8 (21.1)	0.931 ^f
Mild	20 (37.0)	5 (31.3)	15 (39.5)	
Moderate	18 (33.3)	6 (37.5)	12 (31.6)	
Severe	4 (7.4)	1 (6.3)	3 (7.9)	
Abnormal coronary anatomy, n (%)				
Yes	6 (11.1)	1 (6.3)	5 (13.2)	0.461 ^f
Conduit repair, n (%)				
Yes	34 (63)	9 (56.3)	25 (65.8)	0.507 ^f
Di-George Syndrome, n (%)				
Yes	16 (29.6)	4 (25.0)	12(31.6)	0.818 ^f
Duration of CPB (mins), median (IQR)	163(140-199)	165(142-237)	163(138-193)	0.235 [§]
Ischaemic time (mins), median (IQR)	104(89-126)	108(91.5-145)	104(86-126)	0.330 [§]
Open sternum, n (%)	32 (59.3)	8(50.1)	24(63.2)	0.369 ^f
Duration of mechanical ventilation (days), median (IQR)	7(2-12)	1.50(1-12.5)	7(5-12)	0.018 [§]
ICU LOS (days), median (IQR)	10(6-20)	2(1-21.6)	11(8-20)	0.035 [§]
Hospital LOS (days), median (IQR)	30(18-53)	21(9.3-42.5)	33(21-56)	0.043 [§]

§ = Wilcoxon Test, †† = Student's t-test, ^f = Fishers' exact test, CAT = Common Arterial Trunk, FISH = Fluorescence in situ hybridization, CPB - cardiopulmonary bypass, LOS - length of stay

Table 2. Conduit versus Non-conduit

Variable	Group 1 (Conduit) n=34	Group 2 (Non-conduit) n=20	Chi-square p-value
Age (days), median (IQR)	90.5(58-163.5)	40.5(28-59)	<0.0001 [§]
Weight (kg), median (IQR)	3.9(3.3-5.0)	3.1(2.7-3.4)	0.0002 [§]
Sex, n (%)			
Female	16(47.1)	11(55.0)	0.573 ^f
Duration of CPB (mins), median (IQR)	143(130-162)	196.5(172-223.8)	<0.0001 [§]
Ischaemic time (mins), median (IQR)	91.0(82-104)	122.5(112.3-148)	<0.0001 [§]
Open sternum, n (%)	15(44.1)	17(85.0)	0.003 ^f
Duration of mechanical ventilation (days), median (IQR)	7(4-12)	6(1.3-8.0)	0.202 [§]
ICU LOS (days), median (IQR)	10(7-22)	10(5.3-14.3)	0.445 [§]
Post operative complications, n (%)	19(55.9)	10(50.0)	0.675 ^f
Hospital LOS (days), median (IQR)	33(20-63)	25(17.3-36.8)	0.086 [§]
Reinterventions, n (%)	10(29.4)	3(15.0)	0.329 ^f
Late deaths, n (%)	7 (20.6)	1 (5.0)	0.721 ^f

f: Fisher's Exact Test; †: Student's T-test; §: Wilcoxon Test, CAT = Common Arterial Trunk, CPB = Cardiopulmonary bypass

Table 3. Postoperative Complications

Complication	Total number of complications n, (%)	Conduit (n, %)	Non-conduit (n, %)	p-value
Infective	13 (24.1)	8 (23.5)	5 (25.0)	1.0^f
Lower respiratory tract infection	9 (15.7)	5 (14.7)	4 (20.0)	
Wound sepsis	2 (3.7)	1 (2.9)	1 (5.0)	
Bloodstream	1 (1.9)	1 (2.9)	0	
Urinary Tract infection	1 (1.9)	1 (2.9)	0	
Cardiac Reoperation	5 (9.3)	3 (8.8%)	2 (10.0%)	1.0^f
Bleeding	2 (3.7)	2 (5.9)	0	
Homograft dilation and RPA compression	1 (1.9)	1 (2.9)	0	
VSD patch leak	1 (1.9)	0	1 (5.0%)	
Branch pulmonary artery stenosis	1 (1.9)	0	1 (5.0%)	
Cardiac	13 (24.1)	8 (23.5)	5 (25.0)	0.903^{&}
Cardiac Output Syndrome	10 (18.5)	6 (17.6)	4 (20.0)	
Pericardial Effusions	3 (5.6)	2 (5.9)	1 (5.0)	
Other pulmonary	8 (14.8)	4 (11.8)	4 (20.0)	0.450^f
Chylothorax	1 (1.9)	1 (2.9)	0	
Pleural effusion	6 (11.1)	3 (8.8)	3 (15.0)	
Phrenic nerve palsy	1 (1.9)	0	1 (5.0)	
Acute Kidney Injury	6 (11.1)	5 (14.7)	1 (5.0)	0.395^f
Total	29, (53.7%)	19, (55.9%)	10, (50.0%)	0.675^{&}

RVOT = right ventricular outflow obstruction, VSD = ventricular septal defect, RPA = right pulmonary artery

&: Chi Square, *f*: Fisher's Exact Test

Table 4. Medium-term outcomes post hospital discharge

	Whole cohort N = 38	Conduit N = 25	Non-conduit N = 13	P value
Lost to follow up, n (%)	11 (28.9%)	8 (32.0%)	3 (23.1%)	0.46 ^f
Mortality, n (%)	8 (21.1%)	7 (28.0%)	1 (7.7%)	0.22 ^f
Actuarial survival rate (%)				
6 months	62.1	77.5	58.6	0.47 [§]
12 months	55.8	53.4	58.6	0.60 [§]
27 months	50.6	44.5	58.6	0.97 [§]
Reintervention, n (%)	10 (26.3%)	9 (36.0%)	1 (7.7%)	0.12 ^f
Overall freedom from reoperation (%)				
1 year	74.0%	66.2%	86.5%	0.52 [§]
2 years	70.1%	66.2%	76.9%	0.22 [§]
8 years	38.8%	29.8%	64.1%	0.24 [§]

f: Fisher's Exact Test, §: Wilcoxon Test

Fig:1 Kaplan Meier: Survival curve

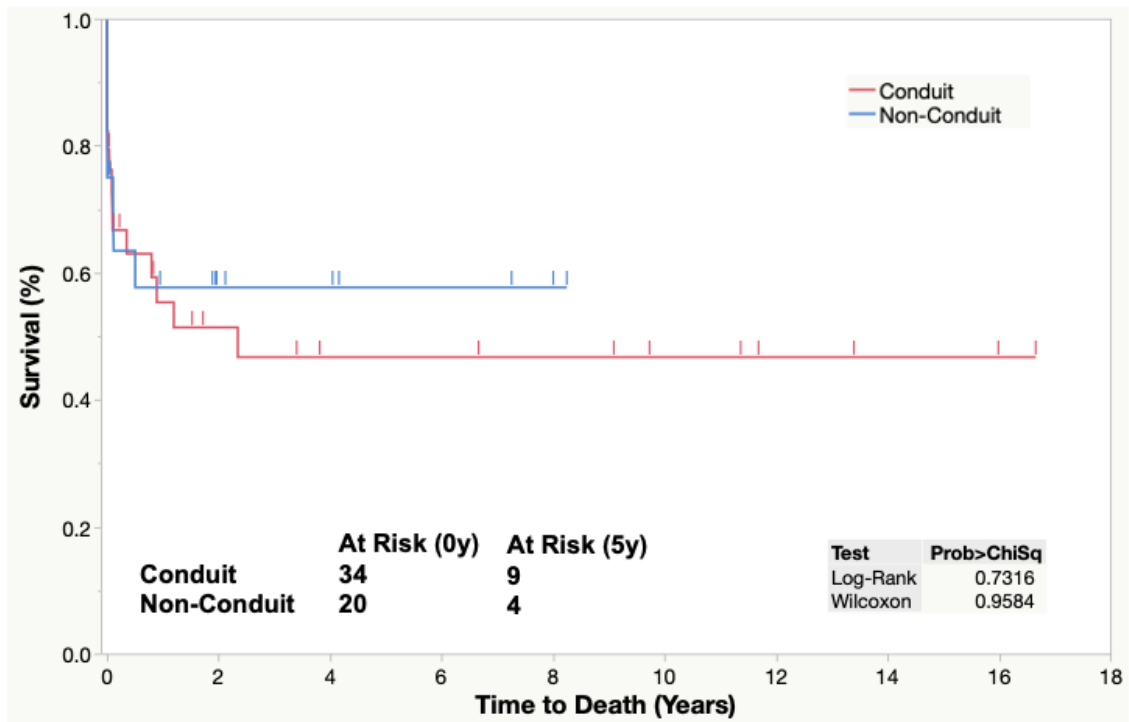
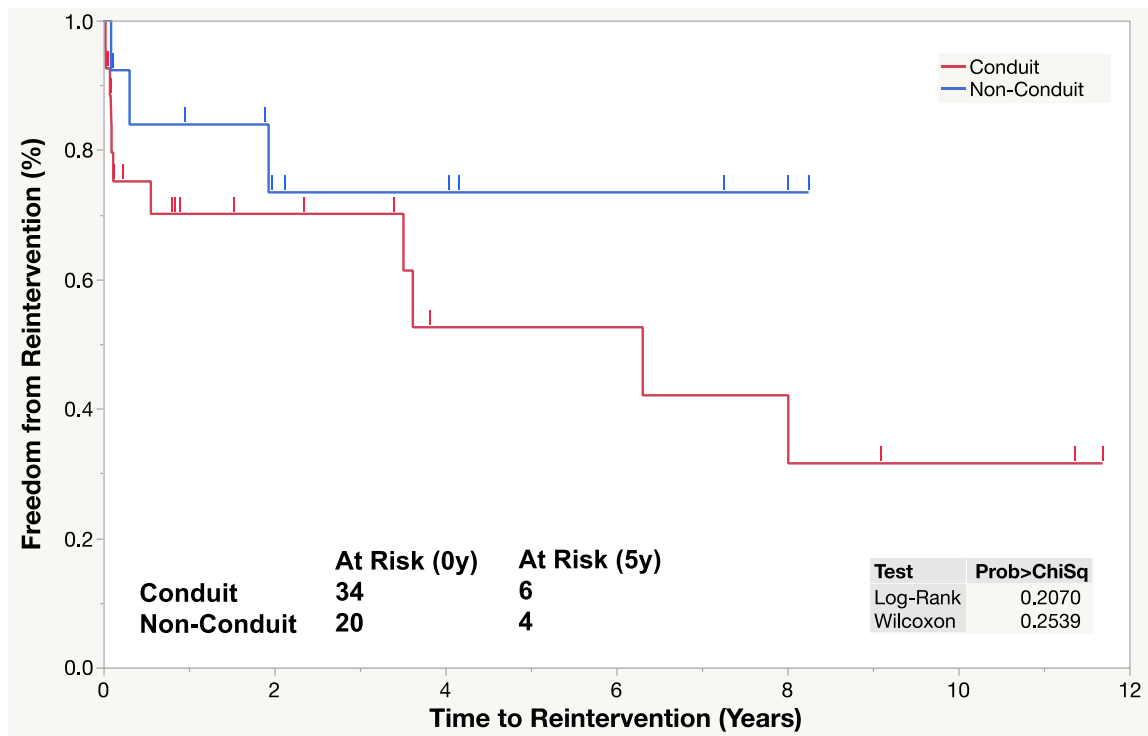


Fig:2 Kaplan Meier: Reinterventions



Abbreviations

Red Cross War Memorial Children's Hospital	(RCWMCH)
Common Arterial Trunk	(CAT)
Interrupted aortic arch	(IAA)
Low- and Middle-Income Countries	(LMICS)
High-income countries	(HIC)
University of Cape Town	(UCT)
Paediatric intensive care unit	(PICU)
Cardiopulmonary bypass	(CPB)
Paediatric intensive care unit length of stay	(PICU LOS)
Hospital length of stay	(HLOS)
Right ventricular outflow tract	(RVOT)
Acute kidney injury	(AKI)
Lower respiratory tract infection	(LRTI)
Pulmonary arteries	(PAs)
Ventricular septal defect	(VSD)
Right ventricle to pulmonary artery	(RV-PA)
Low Cardiac Output Syndrome	(LCOS)
Right pulmonary artery	(RPA)

Publication ready Format

Title: Common Arterial Trunk Repair at the Red Cross War Memorial Hospital, Cape Town: A 20-year review of surgical practice and outcomes

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Abstract:

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The actuarial survival for the conduit group was 77.5%, 53.4% and 44.5% at 6, 12 and 27 months respectively and non-conduit group was 58.6% at 6 months. The overall freedom from re-operation between the conduit group and non-conduit group was

66.2% vs 86.5%, 66.2% vs 76.9% and 29.8% vs 64.1% at 1, 2 and 8 years respectively.

CONCLUSIONS: No difference in postoperative mortality between the conduit and non-conduit repair. Reintervention rates were lower in the non-conduit group.

Introduction

Common arterial trunk (CAT) is an uncommon complex congenital cardiac disease with a reported incidence of less than 3% of all critical congenital cardiac heart anomalies [1]. This condition was first documented in 1798 by Mr James Wilson when he described *“a single arterial trunk originating from the biventricular heart supplied the arterial, coronary, and pulmonary circulation”*[2]. In 1967, McGoon and Rastelli reported the first successful surgical repair using an aortic homograft [3]. A significant advancement of CAT repair was achieved by Barbero-Marcial et al in the 1990's[4] who described a technique of non-conduit repair with the use of the left atrial appendage to reconstruct the posterior wall of the neo-main pulmonary artery and autologous pericardium patch was used to complete the anterior wall with the neo-main pulmonary artery. CAT can be further complicated by its association with interrupted aortic arch, 22q11.2 deletion syndrome, and the development of pulmonary hypertension. Without surgical intervention, the natural history of the disease has a mortality of 70% to 85% within the first year of life [3, 5].

Subsequent improvements in the perioperative management of patients with CAT have translated into improved outcomes in these patients, although published data on outcomes after CAT repair is largely derived from the high-income countries (HIC)[5, 6]. Mortality after CAT repair is significant, and the need for re-intervention is common, particularly if the surgical repair included a right ventricular to pulmonary artery (RV-PA) conduit [6]. Several studies have acknowledged risk factors linked with raised postoperative morbidity and mortality after surgical repair of CAT. These include low birth weight, coronary abnormalities, long aortic cross-clamp time, interrupted aortic

arch (IAA) and moderate to severe truncal regurgitation [5, 7, 8]. South Africa is categorised as a low- and middle-income country. Data on outcomes after CAT repair in Low- and Middle-Income Countries (LMICS) [25] are scarce [11,13,24], and there are no published data on outcomes after CAT repair in Africa.

There remains an ongoing debate on whether a conduit or non-conduit repair [4] is the optimal surgical technique. Although in-hospital morbidity and mortality between the two techniques are reported to be similar [10], the potential benefit of a non-conduit repair is the avoidance of multiple re-operations to replace the conduit as the child grows. This may be of relevance in resource-challenged settings. Surgical repair for CAT has been offered at our institution since the 1990s. In view of the well-recognized complexities of providing health care in LMICs and the worldwide lack of availability of homografts, the decision was made in 2011 to transition from conduit repair to non-conduit repair at our institution, in part due to an anticipated reduction in the need for surgical re-intervention in the medium- and long-term and the reduced availability of homografts. Although these two surgical approaches were implemented during distinctly different time periods, we postulate that the move toward a non-conduit technique has resulted in a reduction in re-intervention rates in our patient population without a significant change in adverse outcomes.

Our objective was to describe postoperative outcomes following CAT repair over a 20-year period prior to and following the transition to non-conduit repair at our institution. The primary outcomes were 30-day in-hospital and overall mortality in paediatric patients who underwent CAT repair at Red Cross War Memorial Children's Hospital (RCWMCH). Secondary outcomes encompassed (a) Incidence of postoperative

complications assessed according to predefined criteria and (b) medium-term outcomes, including reinterventions, late deaths and loss to follow-up.

Materials and Methods

Study Population

This was a single-centre retrospective study of all consecutive patients who undertook the repair of CAT from January 1999 to December 2018 at RCWMH. Paediatric cases with coexisting interrupted aortic arch (IAA) and those who had a previous pulmonary artery banding procedure were excluded from the study.

Ethical consent for the study was given by the Human Research Ethics Committee of the Faculty of Health Sciences, University of Cape Town (UCT) (HREC REF: R017/2014, 044/2016 & 655/2018). Local hospital authorisation was approved by the Research Review Committee. Specific patients were de-identified, and their consent was waived.

Data Collection

Relevant data were collected from the bespoke in-house paediatric cardiac surgery database, paediatric cardiology database, paediatric intensive care unit (PICU) records and matching patient folders. Attempts were made to contact the parent or legal guardian of every patient via telephone who had been lost to follow-up.

Patient demographics included age, sex, weight, type of CAT (Van Praagh classification used to categorise patients into 4 sub-types: Type 1 is partially formed aorticopulmonary septum, and main pulmonary artery segment is present. Type 2 is the absence of the aorticopulmonary septum, and no main pulmonary artery segment is present. Both branches of pulmonary arteries have their origins in the common arterial trunk. In type 3, one branch pulmonary artery is does not originate from the common arterial trunk, thus the ductal or aortic origin of one pulmonary artery. In type 4, the aortic arch is hypoplastic or interrupted, and there is a significant patient ductus arteriosus. The Van Praagh classification also specifies the presence of a VSD (type A) or the absence of a VSD (type B). Thus, each case of truncus arteriosus is assigned a nomenclature that includes a letter and a number), comorbidities, degree of truncal valve regurgitation, and coronary abnormalities.

Intraoperative and postoperative data included length of general anaesthesia, surgical operative duration, total cardiopulmonary bypass (CPB) time, aortic cross-clamp time, concurrent procedures, conduit utilisation, paediatric intensive care unit length of stay (PICU LOS) and hospital length of stay (HLOS).

Data on echo findings were recorded at three time points: Pre-operative, early postoperative (<48 hours post-op) and most recent. Pre-operative findings included ventricular function, truncal valvular regurgitation and right ventricular outflow tract (RVOT) stenosis. Early postoperative findings and the most recent echo findings also included the degree of regurgitation or stenosis of truncal and pulmonary valves and any residual lesion.

Complications included unforeseen myocardial catheterisation procedure in the postoperative period, unscheduled cardiac reoperation (not including reoperation for

bleeding), bleeding necessitating reoperation, acute kidney injury (AKI) defined as a raise in creatinine of $\geq 50\%$, infective complications, neurologic consequences persisting at discharge, non-infective pulmonary complications (failure to extubate, pneumothorax, pleural effusion, chylothorax or diaphragmatic paresis), myocardial arrest during or after the procedure, death as well as any other non-predefined complications. Infective complications included bacteraemia, lower respiratory tract infection (LRTI)—confirmed by chest x-rays with new or progressive and persistent pathological infiltrates—consolidation, cavitation, clinical diagnosis, superficial or deep sternal wound infection or any other type of infection (defined as a confirmed diagnosis of verified or assumed sepsis by the treating paediatricians). ‘Early mortality’ was defined as death occurring within 30 days of the operation or before hospital discharge. Death after this time was categorised as ‘late mortality’.

Medium- to long-term records composed of years to re-intervention, late mortality and patients lost to follow-up (defined as no outpatient or inpatient appearance for the extent of 24 months).

Operative technique

The surgical repair was performed following a full median sternotomy incision and with CPB support in all subjects under moderate to deep hypothermic conditions (temperatures ranged from 20 to 32 degrees Celsius). The pulmonary arteries (PAs) were ensnared at the commencement of CPB, and the heart was arrested with antegrade cardioplegia. Different cardioplegic strategies were employed at our institution over 20 years. Our initial cardioplegia strategy was crystalloid extracellular,

St Thomas^R, and this was subsequently changed to intracellular cardioplegia - Custodial^R and the occasional use of blood microplegia. The right atrium was opened and the cardioplegia was suctioned off. The PAs were excised from the truncal vessel while taking care to prevent damage to the truncal valve and the coronary arteries. The aortic root was repaired using bovine pericardium.

Pulmonary arteries were extensively mobilised in all cases. Inspection of the truncal valve for morphology and function was conducted, and truncal valve regurgitation repair was effected where required. The ventricular septal defect (VSD) was almost always closed through the ventriculotomy. An atrial fenestration was not routinely performed at our institution. In group 1, right ventricle to pulmonary artery (RV-PA) continuity was established using a conduit, whereas in Group 2, RV-PA continuity was re-established without a conduit. In Group 2, the pulmonary outflow tract was recreated utilising the left atrial appendage, which was sutured to the distal end of the right ventriculotomy and the pulmonary convergence, maintaining its anatomical position. The anterior hood was reconstructed with a bovine pericardial patch. A monocusp valve was fashioned at the discretion of the primary surgeon, but this was not a routine part of the reconstruction. The sternum was electively left open, and delayed closure was performed in the PICU.

Statistical analysis

Analysis was performed using JMP Pro (version 16.0, SAS, Cary, NC.). The normal distribution of continuous numerical variables was determined by Shapiro-Wilk analysis. Normally distributed continuous numerical variables were expressed as

means \pm standard deviation, with inferential analysis performed using the independent Student's T-test. Alternatively, non-normally distributed variables were expressed as medians \pm range with between-group comparisons performed using the non-parametric Wilcoxon test. Analysis of categorical variables was assessed using Fisher's Exact or Chi-square testing. Evaluations of time-related survival and freedom from re-intervention were determined using the Kaplan-Meier method with reported log-rank and Wilcoxon rank-sum between-group testing. Two-tailed significance levels <0.05 were accepted throughout as defining statistical significance.

Results

Patient characteristics

Fifty-four patients had CAT repair during the study period (Table 1). The median age at the time of surgery was 61.5 days [IQR 44.8-138.8 days], and the median weight was 3.4kg [IQR 3.0-4.5kg]. The most common lesion was a Type 1A CAT (34/54, 66.7%). Eighteen (33.3%) patients had moderate pre-operative truncal valve regurgitation, while only four patients (7.4%) presented with severe regurgitation. Whilst FISH test results were not available in six patients, associated genetic syndromes were present in 17 (31%) cases. These included 16 (29.7%) patients with Di George syndrome (22q11.2 deletion syndrome) and one patient with VACTERL syndrome. Other non-cardiac abnormalities included one patient with both kidneys located on the right side, one patient with an anorectal malformation and one patient with an aberrant right subclavian artery.

Thirty-four (63.0%) patients had a conduit repair, and 20 (37.0%) patients had a non-conduit repair. Patients who underwent a conduit repair were significantly older compared with the patients who underwent non-conduit repair: 90.5 days [IQR 58-163.5 days] vs 40.5 days [IQR 28-59 days, $p<0.0001$], and their median weight was significantly higher (3.9kg [IQR 3.3-5.0] vs 3.1kg [IQR 2.7-3.4], $p=0.0002$). The median total perfusion time for the entire cohort was 163 minutes [IQR 140-199], and this was significantly lower in the conduit group compared with the non-conduit group ($p<0.0001$) (Table 2). The median ischaemic time for the whole cohort was 104 minutes [IQR 89-126]. Ischaemic time was significantly shorter in the conduit group compared with the non-conduit group ($p<0.0001$). The predominant conduit used was an aortic homograft 31/34 (91%), with a Contegra^R graft used in two patients. Repair of truncal valve regurgitation was performed in one patient in the form of commissural resuspension. A monocusp valve was created in two of the non-conduit cases. Eighty-five percent (17/20) of the non-conduit group had the sternum electively left open versus 44% (15/34) in the conduit group ($p=0.003$). There was no significant difference in the duration of mechanical ventilatory support, duration of ICU stays or HLOS between the group who underwent conduit versus non-conduit repair (Table 2).

In-hospital mortality

Thirty-day in-hospital mortality was 22.2% (12/54), and overall in-hospital mortality was 29.6% (16/54). This included two on-table deaths. The first patient, with CAT Type 1, presented for surgery at 22 days of age. This patient died after being unable to be weaned off CPB due to right heart failure secondary to suspected pulmonary hypertension. The second patient (44 days of age), with CAT type 2, presented with abnormal coronary anatomy, including an accessory left anterior descending coronary

artery originating from the right coronary artery and a small calibre left coronary artery. Initial separation from CPB failed due to suspected coronary compression from the pulmonary patch. The patch was revised, but this did not improve ventricular function, and the patient demised on the table.

Thirteen (81.3%) of sixteen deaths occurred in PICU. The causes of postoperative death in PICU included 8/13 (61.2%) Low Cardiac Output Syndrome (LCOS), 1/13 (7.7%) pulmonary hypertensive crisis and 4/13 (38.5%) secondary to septic complications, including one patient with methicillin-resistant *Staphylococcus aureus* (MRSA) infective endocarditis and three patients with LRTI based on clinical and radiological evidence and biochemical markers. One death occurred in the ward after ICU discharge on postoperative day 40, secondary to aspiration pneumonia. There was no difference in mortality rates between those patients who underwent a conduit repair versus those patients who underwent a non-conduit repair ($p=0.507$).

Morbidity

Twenty-nine (55.8%) of fifty-two patients suffered a postoperative complication (Table 3). There was no major disparity in the number of postoperative complications amongst the cases who underwent a conduit versus a non-conduit repair ($p=0.675$). Within the whole cohort, 12/54 (22.2%) patients suffered greater than one complication. The most common overall complications were cardiovascular and infective. Overall, 10/54 (18.5%) developed LCOS in the postoperative period. This rate of LCOS was similar between the conduit group (6/34, 17.6%) and the non-conduit group (4/20, 20%). Thirteen of fifty-two (24.1%) patients had an infective complication. The number of infective complications was similar between the two groups ($p=1.0$). Positive microbiology was documented in 12 of these 13 patients.

The pathogens detected included *Klebsiella pneumonia* (n=3), viral pneumonia (n=3), *Serratia marcescens* (n=1) and *Sphingomonas paucimobilis* (n=1). One patient, in whom the causative organism could not be identified, was treated for LRTI based on clinical and biochemical criteria. One patient had a urinary tract infection in which the causative organism was *Escherichia coli*. The most frequent septic complication was LRTI (9/13, 69.2%). Two patients had superficial wound sepsis. One patient in the non-conduit group cultured MRSA from the sternal wound, and this patient underwent debridement in theatre, with secondary closure a few days later. One patient in the conduit group cultured *Staphylococcus epidermidis* sensitive to Cloxacillin. This patient had three vacuum-assisted dressings, followed by secondary closure. Three patients required an unplanned cardiac reoperation prior to discharge. One patient in the conduit group had homograft dilation and compression of the right pulmonary artery (RPA). This patient had the homograft replaced and the RPA compression relieved. The remaining two patients were in the non-conduit group. One patient had a significant VSD patch leak where the VSD patch had detached from the ventricular edge. The VSD patch was augmented and reattached to the ventricular wall, correcting this complication. The other patient had branch pulmonary artery stenosis and was taken back to the theatre for augmentation of the branch pulmonary arteries. Two patients in the conduit group required reoperation for bleeding. One patient had a cardiac tamponade, and the other patient had bleeding around the conduit anastomotic site. Of the eighteen patients with pre-operative moderate truncal valve regurgitation, nine (50%) patients had residual moderate post-operative truncal valve regurgitation. Truncal valve regurgitation in all four patients who presented with 'severe' preoperative truncal valve regurgitation had improved to 'mild' postoperatively. This included the patient who had undergone truncal valve repair.

Medium-Term Outcomes

A total of 38 cases were followed up post hospital discharge for a median of 23 months (IQR 5.5-89.3 months). Eight (21.1%) late mortalities were observed within the discharged cohort (Table 4). Most of the late deaths occurred in the group that underwent a conduit repair (7/8, 87.5%). The mean time to death post discharge was 9.4 months (SD+/- 9.6) for the conduit group. Within the conduit group, the causes of late mortality included LRTI in four patients, of which one was secondary to post-procedural compression of left main bronchus by the extracardiac conduit. This patient was intubated but unfortunately died secondary to a LRTI prior to surgical intervention. Of the remaining patients that died, one patient died of sepsis post-Nissen fundoplication, one patient died due to massive hemoptysis secondary to an esophageal subclavian fistula and in one patient the cause of death was unknown. The single late mortality observed in the non-conduit group occurred at six months and was due to hypoxic cardiac arrest secondary to recurrent LRTI. The actuarial survival rate for the entire cohort was 62.1%, 55.8% and 50.6% at 6 months, 12 months and 27 months, respectively. For the conduit group, the actuarial survival rate was 77.5%, 53.4% and 44.5% at 6 months, 12 months and 27 months, respectively. The non-conduit group had an actuarial survival rate of 58.6% at 6 months (Fig 1). Eleven (28.9%) of thirty-eight cases were lost to follow-up. Eight (32.0%) of twenty-five patients within the conduit group were lost to follow-up compared with 3/13 (23.1%) in the non-conduit group ($p=0.463$)

Reintervention

During the study period, 10/38 (26.3%) patients required further intervention post hospital discharge (Table 4). Nine of twenty (45.0%) patients from the conduit group

and 1/13 (7.7%) patients from the non-conduit group required reintervention. Within the group who underwent a conduit repair, the indications for reoperation included RVOT stenosis in 5/9 (55.6%) patients, VSD patch leak (1/9, 11.1%), VSD patch dehiscence (1/9, 11.1%), endocarditis of the aortic valve and VSD patch (1/9, 11.1%), left thoracotomy for aortopexy which was performed to relieve left main bronchus compression secondary to a dilated aorta on a patient who had a previous CAT repair with a homograft (1/9, 11.1%) and one patient had percutaneous balloon dilation for branch pulmonary artery stenosis six months post-CAT repair. Within the group that underwent a non-conduit repair, one patient required a homograft due to RVOT stenosis.

The overall freedom for re-operation for the cohort was 74.0%, 70.1% and 38.8% at 1, 2 and 8 years, respectively. The overall freedom from re-operation for the patients who underwent a conduit repair was 66.2% vs 86.5%, 66.2% vs 76.9% and 29.8% vs 64.1% compared with the patients who underwent a non-conduit repair at 1, 2 and 8 years, respectively (Fig 2).

Discussion

The principal finding of this study was that 30-day and in-hospital post-operative mortality following CAT repair at our institution during a 20-year period was 22.2% and 29.6%, respectively. The overall actuarial survival following CAT repair at six months was 62.1%. There was no difference in perioperative mortality between the groups that either underwent a conduit versus a non-conduit repair. The overall freedom from reoperation was markedly lower at eight years in the non-conduit group.

Data on perioperative outcomes following CAT repair in LMICs are few. Apart from a single case report from Nigeria [24], there are no data published on the perioperative, medium or long-term outcomes following CAT repair in Africa. Two studies have been published describing the outcomes after CAT repair from a single centre in Thailand. Laohaprasitiporn et al. published a study in 2008 that described the outcomes of 30 patients with CAT from 1995 to 2004 [11]. Only 16/30 patients diagnosed with CAT underwent surgical intervention. Of these, 14 patients underwent full repair with conduit and two patients underwent pulmonary artery banding for palliation. The authors reported a 50% early mortality rate following corrective surgery. A subsequent study published in 2020 by Dangrunroj et al. described the long-term outcomes of all patients diagnosed with CAT at the same centre in Thailand over a 20-year period from 1995 to 2018[10]. A total of 74 patients (including the 30 patients described in the prior study) presented during the study period, and the median age at diagnosis was 70 days. Fifty-two patients (70.3%) underwent surgical repair, and only one patient underwent direct anastomosis of the right ventricle to the pulmonary artery confluence. The authors reported a similar early mortality rate (30.8%) compared to our findings. LCOS related to the pulmonary hypertensive crisis was the most frequently described contributing factor, occurring in 14/16 (87.5%) of the early mortalities in Dangrunroj's study. LCOS and pulmonary hypertension crisis combined were also the most attributed cause of death in our patient cohort, accounting for 10/16 (62.4%) of the perioperative deaths. Similarly, septic complications were found to be significant factors contributing to early mortality in both our study (5/16; 31.3%) and in Dangrunroj's study (4/16; 25.0%).

In comparison to the findings in our study, both the median age at the time of surgery and perioperative mortality following CAT repair reported from HICs is lower (5% to 17%) [6, 9, 12]. One of the factors that contributed to an older median age is that earlier in our clinical practice, i.e., in the first decade, we opted to operate on CAT patients at an older age to accommodate the size of extracardiac conduit available. Almost all diagnosis was in the postnatal period, with the prenatal ultrasound machine and the expertise not being readily available at most institutions. There has been some improvement regarding this problem, i.e., equipment and expertise, over the last decade which could also account for the earlier postnatal diagnosis. Although our strategy has moved primarily to a non-conduit technique, in selected cases of late presentation of CAT with established severe PHT, we opted to perform a valved conduit repair. Other possible factors contributing to the higher perioperative mortality rates seen in our patient cohort include prolonged hospital stay prior to surgery and delayed diagnosis with subsequent prolonged exposure to pulmonary over-circulation and recurrent LRTIs. Our patient cohort was also older at the time of surgery compared with paediatric patients undergoing CAT repair in HIC, and it has been well documented that surgery beyond the neonatal period is associated with increased post-operative mortality rates, commonly related to pulmonary hypertensive crises and LCOS [9, 13], which was the most common cause of in-hospital death in our patient cohort.

Several studies from HIC settings have reported on preoperative risk factors associated with early mortality in CAT repair. Studies have observed that coronary abnormalities, severe truncal valve regurgitation and/or pulmonary artery stenosis/hypoplasia were associated with an increased risk of perioperative mortality

[6, 14-16]. Within our patient cohort, six patients had abnormal coronary anatomy. One patient died on the table due to technical issues with surgical repair complicated by abnormal coronary anatomy. One patient in our cohort had preoperative severe truncal valve regurgitation but survived hospital discharge and was still alive on follow-up. Low birth weight (<2.5kg) [6, 17] and weight at the time of diagnosis (<4.0kg) [13] have also been shown to be associated with an increased risk of mortality. Data on the association between the duration of CPB and mortality are conflicting. Early studies suggested that increased duration of CPB and ischaemic time were associated with increased perioperative mortality in CAT repair [5]. In a subsequent study by Raisky et al., no significant difference in perioperative mortality associated with longer CPB and ischaemic times was observed [10]. Within our study cohort, even though the duration of CPB and ischaemic time was significantly longer in the non-conduit repair, there was no difference in overall mortality between the groups. Univariate analysis failed to identify risk factors associated with perioperative mortality within our patient cohort.

Medium-term outcomes

Thirty-eight patients were discharged from the hospital, and there were eight late deaths. Like the results published by Dangrunroj et al., the most frequent cause of death was LRTI. Of the patients discharged from the hospital, almost one-third (28.9%) were lost to follow-up. This high rate of loss to follow-up is analogous to data published from our institution on outcomes following the bidirectional Glenn surgery [18]. There are multiple factors that may contribute to this, including the perception that the surgery is curative, and that no follow-up is necessary, lack of access to public

transport, lack of financial support to travel from rural areas to attend the follow-up clinic, a lack of centres with specialised paediatric cardiac care and the frequent changing of contact details, in particular cell phone numbers, making attempts to contact parents or legal guardians impossible. The loss to follow-up is of particular concern in those patients who underwent a conduit repair. The need for surgical re-intervention to replace the conduit increases over time as the child grows, yet almost half of these patients have not attended the hospital or outpatient clinic for more than two years. In 2009, Raisy et al. published a study describing the feasibility of a non-conduit technique for RVOT reconstruction as an alternative to a conduit [10]. The authors reported similar perioperative morbidity and mortality between the two techniques but noted a marked reduction in the need for re-intervention in the non-conduit group. The authors highlighted the lack of aortic homografts, necessitating a trend towards performing a non-conduit technique or using heterografts to establish RV-PA continuity. Of concern is the increased reintervention rate associated with the use of both Contegra and Hancock heterografts in comparison with aortic homografts [19]. Whilst many patients in our cohort who underwent a conduit repair received an aortic homograft, this reflects the increased availability of aortic homografts during the earlier time period of the study. Similar to Raisy et al., our results showed an overall increase in freedom of operation at eight years in the non-conduit versus the conduit group.

Suggestions for improvement

Earlier detection and referral for definitive management would most likely translate to improved outcomes in these patients. Ideally, CAT should be diagnosed during foetal

ultrasound or in the early postnatal period. Whilst current guidelines recommend that all pregnant women should receive one basic obstetric ultrasound at 18-20 weeks' gestation [20], ultrasound is a relatively expensive operator-dependent resource-intensive tool, and the implementation of universal foetal anomaly screening is not currently realistic within the public healthcare setting in South Africa. A more feasible and pragmatic option would be to screen for critical congenital cardiac lesions using peripheral oxygen saturation measurement as part of the routine postnatal examination. This non-invasive, low-cost, highly specific and moderately sensitive screening test has been implemented in many HIC as the standard of care [21]. However, attempts to introduce universal peripheral oxygen saturation screening in neonates in the South African setting have been hampered by inadequate staffing and infrastructure [22, 23].

Even if strides towards earlier diagnosis of CAT are made, limitations in the provision of paediatric cardiac surgical services remain.

Initiatives to improve and reduce the cases of paediatric patients lost to follow-up are also already in progress. Children who underwent surgery are actively followed up within 30 days of surgery as part of the International Quality Improvement Collaborative for Congenital Heart Disease *IQIC* [25]. The paediatric cardiologists from our institution also run regular outreach clinics to help improve access to these expert services.

Limitations

This study has some key limitations. These findings are retrospective and represent outcomes from a single centre. Implications from this study and generalisability should be deduced within this context. The small number of conduit vs non-conduit (34 vs 20) cases was a relative limitation.

Since this technique was implemented more recently, the interval mortality and requirement for re-intervention in our study cohort should be interpreted with caution given the substantial number of cases lost to follow-up and the shorter follow-up period in the non-conduit group. However, the loss to follow-up rates is representative of the current challenges at our institution and is also reflective of similar findings frequently noted in other centres in Africa. Outcomes may also have been influenced by changes in perioperative management during the 20-year period.

Summary

This is the first study reporting on results after CAT corrective surgery in Africa. Essential findings are the older age at which the surgery is carried out compared to HICS, similar mortality rates to other LMICs, reduced need for reintervention in the non-conduit group and the high number of patients lost to follow-up. The outcomes of this study support the decision to transition to a non-conduit repair in our situation but emphasize the need for more studies to identify valuable strategies to improve early diagnosis of CAT, expedite surgery and reduce the number of patients lost to follow-up.

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Appendices:

Data Capture Sheet:

Patient	
Folder Number	
Sex	
Contact Details	
Date of Birth	
Date of Diagnosis	
Birth weight kg	
Classification of common arterial trunk	
Description of Truncal valve	
Number of leaflets	
Preoperative truncal valve Regurgitation	
RVOT stenosis	
Stenosis RVOT gradient	
Associated cardiac lesion	
ASD	
PFO	
IAA	
PDA	
LEFT SVC	
TAPVD/PAPVD	
Coronary Abnormality	
Details of coronary abnormality	
Coronary abnormality type	
AVSD	
Co Arc	
VSD	
VSD Comment	
@22q11deletion DiGeorge syndrome	
Other conditions	
Preop Microbiology	
Preop Micro comment	
OPERATIVE	
Date of Surgery	
Weight at operation kg	
Total conduit	
conduitless	
monocusp	
Type of conduit replacement	
Conduit Type	
Size of conduit	
Complications during surgery	
Bypass Time mins	
Cross clamp time mins	

Chest Left Open duration	
POSTOPERATIVE IMMEDIATE	
Length of ICU stay days	
Preoperative ventilation	
Length of ventilation preop days	
Length of ventilation post op days	
Total hospital stay days	
ECHO	
Immediate postoperative Truncal Valve Regurgitation	
RVOT Pressure gradient or branch pulmonary stenosis	
Postoperative complications list	
sepsis	
Other pulmonary	
Cardiac complications	
Thirty-day mortality	
In hospital death	
Causes of in hospital death	
Mortality Date Hospital	
Cause of Hospital Mortality	
Reinterventions total	
Date of reintervention	
Cardiac	
noncardiac	
Type of reintervention	
Indicators for Reintervention	
Weight at reintervention kg	
RVOT pressure gradient	
Size of conduit	
Complications during surgery	
Bypass Time mins	
Cross clamp time mins	
POST OPERATIVE IMMEDIATE	
Duration of ICU stay (days)	
Duration of ventilation postop	
Total hospital stays	
ECHO	
RVOT Pressure gradient or branch pulmonary stenosis	
Post operative complications	
Hospital Mortality Date	
FOLLOW UP	
Mortality Date	



UNIVERSITY OF CAPE TOWN
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08 October 2018

HREC REF: 665/2018

Dr A Brooks
Department of Cardio-Thoracic Surgery
Chris Barnard Building
FHS

Dear Dr Brooks

PROJECT TITLE: RETROSPECTIVE REVIEW OF TRUNCUS ARTERIOSUS REPAIR RED CROSS HOSPITAL FROM 1999 TO DATE: THE OBJECTIVE OF THE STUDY IS TO DESCRIBE THE EVOLUTION OF SURGERY AND ANALYZE THE OUTCOMES OF SURGERY OVER +/- 20 YEAR PERIOD (MMED Candidate - Dr A Moodley) Sub-study linked to 398/2009 & R044/2016

Thank you for submitting your study to the Faculty of Health Sciences Human Research Ethics Committee (HREC) for review.

It is a pleasure to inform you that the HREC has **formally approved** the above-mentioned study.

Approval is granted for one year until the 30 October 2019.

Please submit a progress form, using the standardised Annual Report Form if the study continues beyond the approval period. Please submit a Standard Closure form if the study is completed within the approval period.

(Forms can be found on our website: www.health.uct.ac.za/fhs/research/humanethics/forms)

We acknowledge that the student: Dr Allen Moodley will also be involved in this study.

Please quote the HREC REF in all your correspondence.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please note that for all studies approved by the HREC, the principal investigator **must** obtain appropriate institutional approval, where necessary, before the research may occur.

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, FHS HUMAN RESEARCH ETHICS COMMITTEE

Federal Wide Assurance Number: FWA00001637.



Western Cape
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16 November 2018

Dr A Moodley
Cardio-thoracic Surgery

Dear Dr Moodley,

RESEARCH: RXH: RCC 160

PROJECT TITLE: RETROSPECTIVE REVIEW OF TRUNCUS ARTERIOSUS REPAIR RED CROSS HOSPITAL FROM 1999 TO): THE OBJECTIVE OF THE STUDY IS TO DESCRIBE THE EVOLUTION OF SURGERY AND ANALYZE THE OUTCOMES OF SURGERY OVER +/- 20 YEAR PERIOD.

It is a pleasure to inform you that approval is hereby granted to conduct above-mentioned study at Red Cross War Memorial Children's Hospital.

Yours sincerely,

DR AN PARBHOO
MANAGER: MEDICAL SERVICES