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Promising Outcome of Anatomic Correction of Corrected Transposition of the Great Arteries

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Background. **Anatomic correction of corrected transposition of the great arteries with associated lesions, utilizing the morphologic left ventricle as a systemic pumping chamber, is the preferred method in many centers. The purpose of this study was to analyze functional outcome after anatomic correction.**

Methods. **Between Jan 1997 and May 2016, 63 patients with corrected transposition of the great arteries and associated lesions underwent anatomic correction. Forty-two patients (67%) underwent palliation before correction, including 14 patients (22%) who required training of systemic ventricle. The double switch procedure was performed in 37 patients; 25 patients underwent the Senning-Rastelli operation, and 1 patient underwent the Senning-Nikaidoh procedure. The median age at correction was 1.6 ± 3.7(SD) years (range, 0.2 to 17.8 years).**

Results. **The survival and freedom from any event was 95% and 71%, respectively, at 15-year follow-up. The combined freedom from death, failure of systemic ventricle, or heart transplant was 93% at 15-year follow-up regardless of procedure type. Sinus rhythm was present in 49 patients, with 14 patients requiring pacemaker (22%)—8 preoperatively, 4 early postoperatively, and 2 late postoperatively. Neurological development is normal in all patients. Fifty-four percent of the patients are not on medication.**

Conclusions. **Anatomic correction of corrected transposition of the great arteries is a safe procedure that provides encouraging survival and functional benefits. Ninety-three percent preservation of morphological left ventricle function in 15 years of follow-up supports the concept of anatomic correction. Longer follow-up is needed to confirm superiority of this approach over other management strategies.**

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Historically, patients with congenitally corrected transposition of the great arteries (ccTGA) and associated lesions suitable for biventricular repair were managed by physiologic correction in which the morphologic right ventricle (mRV) was preserved as a systemic pumping chamber and the associated lesions were repaired. The long-term outcomes of patients after physiologic corrections have clearly demonstrated that the tricuspid valve (TV) and mRV function is the Achilles heel of the physiology of ccTGA [1, 2].

Therefore, in 1987 Ilbawi and colleagues introduced the anatomic correction of ccTGA with the aim to utilize the morphologic left ventricle (mLV) as the systemic pumping chamber and the mitral valve (MV) as the systemic valve [3]. By definition, the anatomic correction represents a group of procedures in which the atrioventricular discordance is corrected by an atrial switch (Senning or Mustard), and the ventriculoarterial discordance is corrected either by an arterial switch operation, by the Rastelli procedure, or by translocation of the aortic root. Each of these procedures carries potential complications, affecting final outcomes. The mid-term survival and functional benefits after anatomic correction have been demonstrated, particularly in patients with preoperative tricuspid regurgitation (TR) and mRV dysfunction [2–4]. The purpose of this study was to evaluate our long-term experience with anatomic

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correction of ccTGA focusing on survival, reintervention rate, and functional outcome.

Patients and Methods

A retrospective chart review was undertaken in the German Pediatric Cardiac Center (Sankt Augustin, Germany) to identify all patients with ccTGA in whom an anatomic correction had been performed. Ethical approval was waived by the ethics committee because of the use of retrospective, anonymized data.

Patients

Between Jan 1997 and May 2016, 63 consecutive patients with ccTGA and associated lesions underwent anatomic correction. During this time frame several patients with unfavorable anatomy (eg, remote type ventricular septal defect [VSD], unbalance ventricles, and straddling of atrioventricular valves) had a Fontan operation. More than half (52%) of all patients underwent correction during the last 5 years of the study period. One primary surgeon (VH) was involved. Morphologic characteristics, associated lesions, and anatomic subtypes of ccTGA are shown in Table 1.

Treatment Management: Palliations

Forty-three patients (68%) had undergone a total of 62 palliations at either our or other institutions before anatomic correction. The median age at palliation was 0.31 ± 2.7 (SD) years (range, 0.008 to 11.86 years). The median interval between palliation and anatomic correction was 1.23 ± 2.3 years (range, 0.23 to 11.05 years) (Table 2).

Fourteen patients underwent pulmonary artery banding (PAB) to train the mLV. All patients had no or a restrictive VSD and moderate to severe TR. At the beginning of our experience a "loose" PAB was placed to achieve 50% to 60% of systemic pressure in the mLV and allowing "somatic growth into" the PAB. In the last 6 patients, resection of interatrial septum to provide interval training of their mLV was added. After resection of interatrial septum, mean Qp:Qs was 1.6 (range, 1.4 to 1.9). Precise description of interval training, including indication criteria, was published by our group elsewhere [4, 5]. Median age at training was 2.08 ± 3.3 (SD) years (range, 0.071 to 11.8 years) and median length of training was 1.38 ± 1.5 (SD) years (range, 0.041 to 5.98 years).

Anatomic Correction

Primary correction was achieved in 20 patients (32%). Anatomic correction (Fig 1) included 37 patients with the double switch procedure (DS), 25 patients with the Senning-Rastelli procedure (SR), and 1 patient with the Senning-Nikaidoh procedure (Table 3). Concomitant procedures are elaborated in Table 4. The median age at corrective surgery was 1.58 ± 3.7 (SD) years (range, 0.19 to 17.84 years).

Surgical Technique

Our surgical strategy with all technical aspects of all types of operations had been published previously [4].

Functional Outcome and Follow-Up

The postoperative result was assessed in all patients at discharge and during follow-up. The patients underwent clinical assessment, two-dimensional echocardiography, and Doppler assessment of the intracardiac repair. Any valvar regurgitation was graded as none, mild, moderate, or severe; the qualitative echocardiographic assessment of function of ventricles was graded as normal, mildly, moderately, or severely depressed, using generally accepted echocardiographic criteria. The last two were considered as a reduced function. Follow-up data were complete in all but 2 of the surviving patients (97%). By

Table 1. Patient Characteristics

Characteristics	No. of Patients
Demographics	
Total no.	63
Male/female	36/27
Morphology	
Situs solitus {S,L,L}	56
Situs inversus {I,D,D}	7
Azygos continuation of IVC	3
Left atrial isomerism	1
LSVC with unroofed CS	4
DORV	2
Levocardia	48
Dextrocardia	10
Mesocardia	5
Associated lesion	
VSD	51
Ebsteinoid anomaly of TV	23
Severe TR	13
Severe TS	1
CAVC	1
CoA/AA hypoplasia	4
TAPVD	2
Double orifice MV	1
Severe MR	1
Straddling of AV valves	2
Severe AR	2
Severe sub AS	1
Anatomic subtype	
ccTGA only	10
ccTGA + LVOTO	2
ccTGA+VSD	23
ccTGA+VSD+LVOTO	17
ccTGA+VSD+PA	10
ccTGA+CAVC+TAPVD+LVOTO	1

AR = aortic regurgitation; AS = aortic stenosis; AV = atrioventricular; CAVC = complete atrio-ventricular canal; ccTGA = congenitally corrected transposition of the great arteries; CoA/AA = coarctation of aorta/aortic arch; CS = coronary sinus; DORV = double outlet right ventricle; IVC = inferior vena cava; LSVC = left superior vena cava; LVOTO = left ventricular outflow tract obstruction; MR = mitral regurgitation; MV = mitral valve; PA = pulmonary atresia; TAPVD = total anomalous pulmonary venous return; TR = tricuspid regurgitation; TS = tricuspid valve stenosis; TV = tricuspid valve; VSD = ventricular septal defect.