Tricuspid Valve Regurgitation in Congenitally Corrected Transposition of the Great Arteries and a Left Ventricle to Pulmonary Artery Conduit

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Background. The configuration of the interventricular septum can affect the function of the tricuspid valve in patients with congenitally corrected transposition of the great arteries who have a systemically functioning right ventricle. Altering septal configuration by addressing a dysfunctional conduit placed between the left ventricle (LV) and the pulmonary artery (PA) in these patients can impact septal configuration and competency of the tricuspid valve.

Methods. In 38 patients with an LV to PA conduit, we evaluated relationships between conduit function, RV geometry, and tricuspid valve function, and compared these variables before and after conduit intervention.

Results. Median age at conduit implant was 4.5 years (0.5 to 36) and median total follow-up was 12 years (2 to 22). Of the 38 patients, 23 (60%) underwent conduit intervention, a median of 7.5 years after implant. In 15 of these patients (65%) the degree of tricuspid regurgitation

(TR) worsened, compared with only 2 patients (15%) in the non-intervention group (p < 0.001). Worsening TR was associated with the degree of change in RV and LV ventricular diameters, change in tricuspid annulus size and tethering distance, and the degree of septal shift, as reflected by the right ventricular sphericity index (all $p \le 0.04$). In 8 of 15 patients with more severe TR at follow-up, there was also progressive RV dysfunction.

Conclusions. Intervention for LV to PA conduit dysfunction may result in worsening TR and right ventricular function, likely due in part to altered septal shift due to changes in the interventricular pressure ratio. Management of LV to PA conduit dysfunction should take these findings into account.

competence. Small studies have found that increasing LV pressure by PA banding in patients with a systemic RV

was associated with a reduction in tricuspid regurgitation

(TR) [4–7]. These effects are attributed to the fact LV-PA

conduit dysfunction results in abnormal loading of the

sub-pulmonary LV and consequent alteration of septal

geometry. Furthermore, it has been reported that

morphologic LV pressures were associated with the

severity of TR in patients with CC-TGA [7, 8]. Conduit

intervention for stenosis or regurgitation in this anatomy

may result in opposite changes to septal geometry, with a relative shift back toward the sub-pulmonary LV.

Conceivably, this change could compromise TV function

and instigate a cycle of RV dilation and progressive TR.

The aim of this study was to evaluate the impact of

intervention for LV-PA conduit dysfunction on the func-

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In patients with congenitally corrected transposition of the great arteries (CC-TGA) circulatory physiology is often normal, but the function of the systemic right ventricle (RV) and tricuspid valve (TV) often declines [1-3]. The risk of progressive RV and TV dysfunction has led many to adopt an anatomic repair approach, such that the left ventricle (LV) supports the systemic circulation and the RV supports the pulmonary circulation. Prior to the advent of this "double switch" strategy, however, patients with CC-TGA and pulmonary atresia or stenosis were often managed by placing a conduit from the morphologic LV to the pulmonary artery (PA), along with other intracardiac procedures as indicated. Although this repair is now rarely performed, there remain a number of CC-TGA patients with an LV-PA conduit. As is the case with RV-PA conduits, progressive conduit dysfunction is almost universal and typically leads to reintervention.

Unlike the mitral valve, the TV tension apparatus inserts into the ventricular septum and changes in septal geometry and configuration can affect TV closure and

Patients and Methods

tion of the systemic TV and RV.

Protocol

This retrospective cohort study included patients with CC-TGA who underwent surgical LV-PA conduit placement at any age and were evaluated between January 1, 1992 and December 31, 2012. This included patients with

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Table 1. Baseline Characteristics by Conduit Intervention

Parameter	All Patients (n = 38)	Conduit Intervention $(n = 23)$	No Conduit Intervention $(n = 15)$	p Value
Female	21 (55)	13 (56)	8 (53)	0.89
Age at original conduit placement (years)	4.5 (0.5–36)	3 (0.5–27)	5.5 (1–36)	0.04
Segmental anatomy				0.89
{S,L,L}	30 (79)	18 (78)	12 (80)	
{I,D,D}	8 (21)	5 (22)	3 (20)	
Outflow tract anatomy				0.74
TGA	33	21 (91)	12 (80)	
DORV ^a	5 (13)	2 (9)	3 (20)	
Moderate to severe branch pulmonary stenosis	11 (29)	8 (35)	3 (20)	0.10
Conduit type				0.5
Aortic homograft	20 (53)	13 (56)	7 (47)	
Pulmonary homograft	2 (5)	2 (9)	0	
Bioprosthetic	11 (29)	5 (22)	6 (40)	
Non-valved	5 (13)	3 (13)	2 (13)	
Abnormal TV morphology	15(39)	9 (39)	6(40)	0.94
Permanent pacing	10 (26)	5 (22)	5 (33)	0.43
RV-EDD (z score)	2.1 (1.3-3.1)	2 (1.3-2.5)	2.5 (1.9-3.1)	0.11
LV-EDD (z score)	2.8 (1.7-4.9)	3.4 (2.3-4.9)	1.8 (1-2.2)	0.005
Tricuspid annulus size (z score)	1.5 (0.75-3.5)	1.5 (0.75-3.5)	1.5 (0.75-4)	0.92
TV tethering distance (cm/m²)	0.60 (0.35-0.7)	0.58 (0.4-0.68)	0.65 (0.4-0.7)	0.84
RV Sphericity index	0.9 (0.55-1.2)	0.77 (0.55-1)	1 (0.8-1.2)	0.05
Peak conduit gradient (mm Hg)	40.5 (8-112)	48 (25-76)	20 (8-35)	0.03
PR degree				0.02
None	10 (26)	4 (17)	6 (40)	
Mild	22 (58)	13 (56)	9 (60)	
Moderate	6 (16)	6 (26)	0	
Severe	0	0	0	
RV systolic pressure (mm Hg)	93 (73-123)	97 (80-123)	89 (73-105)	0.15
LV systolic pressure (mm Hg)	48.5 (19-121)	54 (21-121)	30 (19-48)	0.02
LV/RV pressure ratio	0.50 (0.2-1.2)	0.57 (0.3-1.2)	0.35 (0.2-0.5)	0.07
LV dysfunction				0.37
None	29 (76)	16 (69)	13 (86)	
Mild	6 (16)	5 (22)	1 (7)	
Moderate	3 (8)	2 (9)	1 (7)	
TV regurgitation				0.42
None	7 (18)	5 (22)	2 (13)	
Mild	22 (58)	14 (61)	8 (53)	
Moderate	9 (31)	4 (17)	5 (33)	
Severe	0	0	0	
RV dysfunction				0.52
None	12 (31)	8 (35)	4 (27)	
Mild	18 (48)	11 (48)	7 (47)	
Moderate	8 (21)	4 (17)	4 (27)	
Severe	0	0	0	
Follow-up (years)	12 (2–22)	15 (7–22)	9 (2–16)	0.01

^a Three of the DORV patients had {S,L,L} segmental anatomy and 2 had {I,D,D}.

Data in table presented as number (%) or median (minimum-maximum).

 $DORV = \mbox{double-outlet right ventricle;} \qquad EDD = \mbox{end diastolic diameter;} \qquad LV = \mbox{left ventricle;} \qquad PR = \mbox{pulmonary regurgitation;} \qquad RV = \mbox{right ventricle;} \qquad TGA = \mbox{denotes transposition of the great arteries;} \qquad TV = \mbox{tricuspid valve.}$

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