In the preoperative echocardiogram (Figure 1, D1), a line drawn on the anterior border of the ascending aorta bisects the PA. Postoperatively (Figure 1, D2), the body of the PA has clearly moved anterior to that line, away from the LM, whose original posterior curvature is straightened on the postoperative study. CT angiographic sections at the origin of the LM from the aorta (Figure 1, E1 [preoperative] and E2 [postoperative]) and at the level of the bifurcation of the LM into the left anterior descending and left circumflex coronary arteries (Figure 1, F1 [preoperative] and F2 [postoperative]) show the same phenomenon. An increase in distance between the PA and the virtual common origin of the coronaries (preoperative 1 mm, postoperative 5.8 mm) is demonstrated (Figure 1, E2). Distally, at the bifurcation of the PA (Figure 1, G1 [preoperative] and G2 [postoperative]), most of the PA is anterior to the aorta. Comparison of preoperative CT reconstructions viewed from the patient's right and left sides (Figure 1, H1 and I1, respectively) with postoperative views (Figure 1, H2 and I2, respectively) confirms the anterior bowing of the PA that we noted at surgery, serving to increase the distance between the aorta and

PA. Through that enlarged neo-aortopulmonary window, the uncompressed takeoffs of the RCA and LM are seen from both sides (Figure 1, H2 and I2). Before bifurcating, the LM (Figure 1, E2 vs E1 and F2 vs F1) takes a straight course postoperatively and has lost the posterior curvature seen preoperatively. We believe that the fulcrum of motion is not the pulmonary annulus, but rather the attachment of the infundibulum to the right ventricular mass. When the PA is foreshortened by moving the bifurcation anteriorly (and closer to that fulcrum), the main PA arches, allowing the proximal main pulmonary artery to move away from the coronaries. To our knowledge, this is the first imaging analysis to demonstrate desirable anatomic effects of PA translocation.

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Perventricular device closure of ventricular defects in 235 young children: A single-center experience

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Perventricular device closure of ventricular septal defect (PDCVSD) is no longer a novel modality,^{1,2} and current research is focused on the indications and long-term outcomes of the technique.³ We report 235 ventricular septal defect (VSD) cases with indications for PDCVSD from September 2010 to May 2013.

MATERIALS AND METHODS

PDCVSD was indicated for perimembranous, subarterial, or muscular VSD when the defects were 4 to 8 mm in size and 2 mm away from the arterial valve. Of 235 cases, 203 were perimembranous and 32 were

subarterial; patient ages ranged from 6 months to 12 years and weights from 6 to 36 kg. Occluder size (waist) was 2- to 4-mm larger than the defect size, as measured using transesophageal echocardiogram. A symmetric occluder was the default choice, but an asymmetric occluder was selected when a defect was near the aortic valve.⁴

SURGICAL PROCEDURE

A transesophageal echocardiogram probe is placed in a supine patient after anesthesia administration. A lower midline incision (3-4 cm) is made and the lower sternum is sawed open. The pericardium is cut and suspended. A 4-0 prolene purse-string suture is placed in the selected puncture spot. Heparin is administered (1 mg/kg) via a central venous pressure line. A guidewire is introduced into the right ventricle and through the defect into the left ventricle, and a catheter is then introduced via the guidewire into the left ventricle to form a passage. The occluder is inserted into the defect and opened. After confirming that residual shunt and tricuspid and aortic valve regurgitations are absent, the occluder is released. The purse-string suture is knotted, and the chest is closed using a pericardial drainage tube. Protamine is not used. The surgery lasts 20 to 60 minutes. Aspirin is

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FIGURE 1. A comparison of ventricular septal defect (VSD) before and after operation with transesophageal echocardiogram surveillance. The inset depicts the procedure; white arrows show the occluder. RA, Right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; Ao, aorta.

administered orally (1-2 mg/kg daily) for 3 months. Follow-ups are conducted in the postoperative first, third, sixth, and 12th months using transthoracic echocardiography and electrocardiogram.

RESULTS

Of 235 patients, 223 (94.90%) were successfully occluded. The other 12 cases were converted to a VSD repair (VSDR) procedure using cardiopulmonary bypass (CPB) due to irritating tachycardia (2 patients), new aortic regurgitation (5 patients), occluder migration (2 patients), and failure to find the defect (3 patients). One patient with late third-degree atrioventricular block underwent VSDR using CPB during his third month of follow-up; his heart rate resumed a normal rhythm on the 23rd day postoperatively. There was no moderate tricuspid valve regurgitation, and no death occurred (see Figure 1).

In total, 216 patients presented no atrioventricular block, residual shunts, or aortic or tricuspid valve regurgitations after 12-month follow-up. The complication data are shown in Table 1.

TABLE 1. Complications and countermeasures

Complications	Cases (rate)	Countermeasures
Occluder migration	2 (0.85%)	VSDR using CPB
Early AV block I	9 (3.8%)	Follow-up
AV block III	1 (0.42%)	VSDR using CPB
Irritating tachycardia	2 (0.85%)	VSDR using CPB
Immediately converted to VSDR	12 (5.10%)	VSDR using CPB: 2 for occluder migration, 2 for irritating tachycardia, 5 for aortic regurgitation, and 3 for inability to find the defect
Late pericardial effusion	2 (0.85%)	Drainage of the pericardium
Aortic regurgitation	5 (2.12%)	VSDR using CPB
Early residual shunt (<1.5 mm)	12 (5.10%)	Follow-up
Late residual shunt (>1.5 mm)	3 (1.27%)	2 VSDR using CPB; 1 follow-up without surgery
Sum (early complications)	27 (11.48%)	
Sum (late complications)	4 (1.70%)	1 AV block III and 3 late residual shunt* (>1.5 mm)

VSDR, Ventricular septal defect repair; CPB, cardiopulmonary bypass; AV block I, first-degree atrioventricular block; AV block III, third-degree atrioventricular block.

*Among the 3 late residual shunt patients, 2 underwent VSDR under CPB and 1 followed-up without surgery.

DISCUSSION

Our team designed several general principles to determine the indications for PDCVSD. First, the defect must be approximately 4 to 8 mm in size. A very small VSD is hard to find, and a VSD >8 mm makes the occluder unstable and susceptible to migration. Second, the patient must be aged older than 3 months but younger than 12 years. Very small infants usually present with overly large VSDs that are not suitable for occlusion. Conversely, when a patient is older, the VSD features severe tricuspid adherence and thickening, which makes aligning the occluder with the septum difficult and increases the residual shunt likelihood. Third, the VSD must be 2 mm or farther away from the aortic valve.^{3,5} General contraindications include a misaligned VSD, an atrioventricular VSD beneath the septal leaflet, a VSD complicated with aortic valve incompetence, and infective endocarditis. These conditions are not suitable for an occluder, and correction requires CPB.⁵

During the surgery, residual shunt is the most common problem encountered; however, any shunt <1.5 mm in size and 2.5 m/second in velocity is acceptable. A sustained new aortic regurgitation may result from using an inappropriately sized occluder or from an aortic valve injury, requiring VSDR surgery. First-degree atrioventricular block is common, and most cases will eventually subside. If the occluder migrates, emergency VSDR is essential. Irritating tachycardia is far less common, but if it occurs, the maneuver must be stopped. If the

tachycardia is sustained or recurrent, a VSDR using CPB is required. Later-aggravated atrioventricular block and residual shunt can occur, although at a low rate (1.7% in our study).

PDCVSD has been applied for the past decade, but further studies at multiple centers to examine the long-term outcomes are required.

CONCLUSIONS

When VSD is indicated, PDCVSD is a safe modality with an acceptably mild early complication rate and a less severe late complication rate.

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Arterial switch operation for transposition of the great arteries with anomalies of cardiac situs and aortic position

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Supplemental material is available online.

A prime determinant of successful outcome after the arterial switch operation (ASO) is an accurate transfer of coronary buttons to the neo-aorta.¹ Our experience with the ASO in patients with transposition of the great arteries and unusual relationship of the great arteries (levoposed aorta) and dextrocardia is presented here with details of appropriate modifications in surgical technique.

MATERIALS AND METHODS

Between January 2002 and January 2013, a series of 8 patients with transposition of the great arteries and either dextrocardia or aorta positioned anterior and to the left (levoposed aorta) underwent the ASO (Table 1). Of the 3 patients with dextrocardia, 1 patient had situs inversus totalis and the other 2 had normal visceral situs. Six patients, among them the patient with situs inversus totalis, had a levoposed aorta. Left-sided aortic arch was present in 50%. Two patients had both coronaries arising from a single ostium from sinus 2. Five patients had the usual coronary anatomy for

transposition. Patient 7, who had situs inversus totalis with mirror-image dextrocardia, had a peculiar “double looping” coronary pattern with inverted origins of circumflex and right coronary arteries (a mirror image of the Yacoub type E coronary pattern). The Lecompte maneuver was carried out in 3 patients, including patient 7. Juxtaposed right and left atrial appendages were present in 2 patients and 1 patient, respectively.

All patients underwent aortobicaval cannulation, and standard hypothermic (28°C) cardiopulmonary bypass was instituted. The aorta and pulmonary artery were dissected free, and branch pulmonary arteries were completely mobilized. The ductus arteriosus was isolated, transfixed, and divided. The aorta was crossclamped, cold blood cardioplegia was delivered into the aortic root, and the heart was arrested. Transatrial ventricular septal defect closure was performed wherever needed, except in patients 5 and 7, in whom closure was performed through the proximal neo-aorta. The aorta was transected well above the sinotubular junction. Coronary buttons were harvested, and the main pulmonary artery (MPA) was divided at the confluence.

In all 5 cases of levocardia with levoposed aorta, the proximal right ventricular outflow tract (RVOT) was reconstructed first by suturing autologous pericardium to the margins of the defect created in the proximal aorta (neo-pulmonary artery) after harvesting of the coronary buttons. After RVOT reconstruction, a longitudinal wedge of tissue was excised from the proximal MPA (neo-aorta), well above the sinuses, and the coronary button was sutured in place. Because of the peculiar situation in which the aorta was left-sided, the decision to perform the Lecompte maneuver was always postponed towards the end. Also it was anticipated that, the RVOT might impinge on the transferred coronary artery. To prevent this, the arteriotomy in the distal MPA was extended leftward. Starting from the center of the arteriotomy and up to its rightward end, the distal MPA was closed. The proximal MPA was then anastomosed to the under surface of the left pulmonary artery to shift the RVOT in a leftward direction, thus moving it away from the coronary button. If the Lecompte maneuver was not carried out, both the great arteries would come to lie parallel to each other (Figures 1 and 2).

In patients with dextroposed aorta where the aorta was situated to the right and anterior (patients 1 and 5), coronary buttons were transferred routinely and the Lecompte maneuver was performed.

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