Congenitally Corrected Transposition of the Great Arteries

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High Yield Facts

- Congenitally corrected transposition of the great arteries (ccTGA) is a rare malformation with an incidence of 0.5% of congenital heart defects.
- Patients with isolated ccTGA have potentially physiologic circulation, hence the term congenitally (not surgically) corrected transposition.
- ccTGA is frequently associated with ventricular septal defect (VSD), pulmonary valvar and subvalvar stenosis and abnormalities of the tricuspid valve.
- In the absence of other malformations, patients with ccTGA are asymptomatic early in life.
- The surgical approach to patients with ccTGA depends on the presence and severity of the associated lesions.
- The traditional surgical approach (physiologic repair) of ccTGA attempts at restoring normal physiology by repairing the associated lesions.
- Physiologic repair fails to address the most serious anatomic abnormality, mainly ventriculoarterial discordance, and results in less than optimal long-term outcomes. Anatomic repair (double switch) incorporates the left ventricle into the systemic circulation.
- Double switch includes an atrial baffle to redirect venous return in combination with either arterial switch or Rastelli operation (if a suitable VSD permits).
- The excellent short-term and intermediate results of the double switch operation and its modifications make it the procedure of choice for the treatment of ccTGA.
- Occasionally, the anatomic peculiarities of ccTGA do not allow straightforward biventricular repair, and univentricular palliation is a reasonable option.

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Introduction

Congenitally corrected transposition of the great arteries (ccTGA) is a rare malformation with an incidence of 0.5% of congenital heart defects. Patients with isolated ccTGA have potentially physiologic circulation, hence the term congenitally (not surgically) corrected transposition. This corrected circulation results from atrioventricular and ventriculoarterial discordance. The systemic venous return enters the right atrium and passed through the mitral valve to end into a morphological left ventricle which ejects the blood to the lung through the pulmonary valve and artery. Fully oxygenated pulmonary venous return enters the left atrium, passes through the morphologic tricuspid valve to empty into morphologic

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right ventricle, which pumps blood into the systemic circulation through the aortic valve and aorta. This double pathologic discordance results in a physiologic circulation with a systemic right ventricle [1].

Embryologically, ccTGA results from left sided bend of the cardiac tube (l-loop) and the aorta maintaining continuity with the bulbus cordis, l-transposition [2]. The l-transposition implies that the aortic valve is anterior and the left of the pulmonary valve. However, in cases with situs inversus, the aorta lies to the right of the pulmonary artery (5%). It is common to refer to the pathology as SLL or IDD based on Van Praagh's segmental definition [3]. The three letters refer to the situs solitus (S) vs. Inversus (I), to the ventricular loop dextro (D) vs. levo (L) and to the malposition of the great arteries dextro (D) vs. levo (L). In clinical practice, it is best to specify the atrioventricular and ventriculoarterial connections to avoid confusing this entity with much rarer malformations such as isolated AV discordance, crisscross heart, double outlet from either ventricular chambers or single ventricle with atresia of either pulmonary or aortic valve [4].

Most patients with ccTGA have situs solitus with levocardia. The morphologic right ventricle is left sided and the left ventricle is right sided. In 25% of cases there is dextro or mesocardia with situs solitus. Rarely there is situs inversus with the heart apex pointing to the right.

Surgical Pathology

ccTGA is frequently associated with other lesions. These include ventricular septal defect (VSD), pulmonary valvar and subvalvar stenosis and abnormalities of the tricuspid valve. Rarely (1%) the atria, ventricles and great arteries are anatomically and functionally normal except for their discordant connections.

Ventricular Septal Defect

It is the most common associated cardiac malformation. It is present in 70–85% of cases. Malalignment of the atrial and ventricular septa, characteristic of ccTGA, results in an enlarged pars membranacea. Consequently, most VSDs are perimembraneous. Typically, the defect is large, nonrestrictive, and single. It may extend into the inlet or infundibular septum. It is usually separated from the aortic valve by a subaortic infundibulum of variable length [5]. Rarely the inlet and trabecular septum is completely absent resulting in a single ventricle with a subaortic outlet chamber [6].

Left Ventricular Outflow Tract

The pulmonary outflow tract is wedged deeply between the mitral and tricuspid valve due to malalignment of the septa [7]. This position predisposes to the development of stenosis of the morphologically left ventricular outflow tract in 35–50% of cases, at the subvalvar or valvar level. Obstruction at the level of pulmonary valve is causes by fused and/or thickened dysplastic leaflets. Subvalvar stenosis results from subvalvar fibromuscular ridge, aneurysm of the membraneous septum or tunnel. Accessory mitral valve tissue with or without abnormal chordal attachments to the septum could also be present.

Atrioventricular Valves

Significant number of patients (26%) with ccTGA have a morphologically abnormal dysplastic tricuspid valve. The septal leaflet is most affected. It has shortened and thickened chordae tethering the leaflet to the posterior wall. It is frequently displaced toward the apex of the right ventricle in an "ebstenoid fashion" but without atrialization of the ventricular septum [8]. These pathological changes of the tricuspid valve in combination with progressive annular dilatation (due to shift of the interventricular septum towards the morphologic left ventricle) results in regurgitation that is not readily amendable to valvuloplasty. Straddling of the tricuspid valvar or subvalvar apparatus across a ventricular septal defect, could be present and complicates the surgical repair.

Coronary Arteries

The morphology of the coronary arteries is a mirror image of the normal anatomy [9]. The arteries follow the respective inverted ventricles. The right-sided coronary artery has a left distribution. It arises from an anterior sinus of the aortic valve and divides into an anterior descending artery that follows the outline of the interventricular septum, and a circumflex that follows the atrioventricular groove and supplies multiple marginal vessels. The left sided right coronary arises from a posterior sinus and courses into the left atrial–right ventricular groove to supply the free wall of the morphologic right ventricle (Fig. 100.1).

Conduction System

Malalignment of the atrial and ventricular septum displaces the atrioventricular node (AV) anteriorly and superiorly

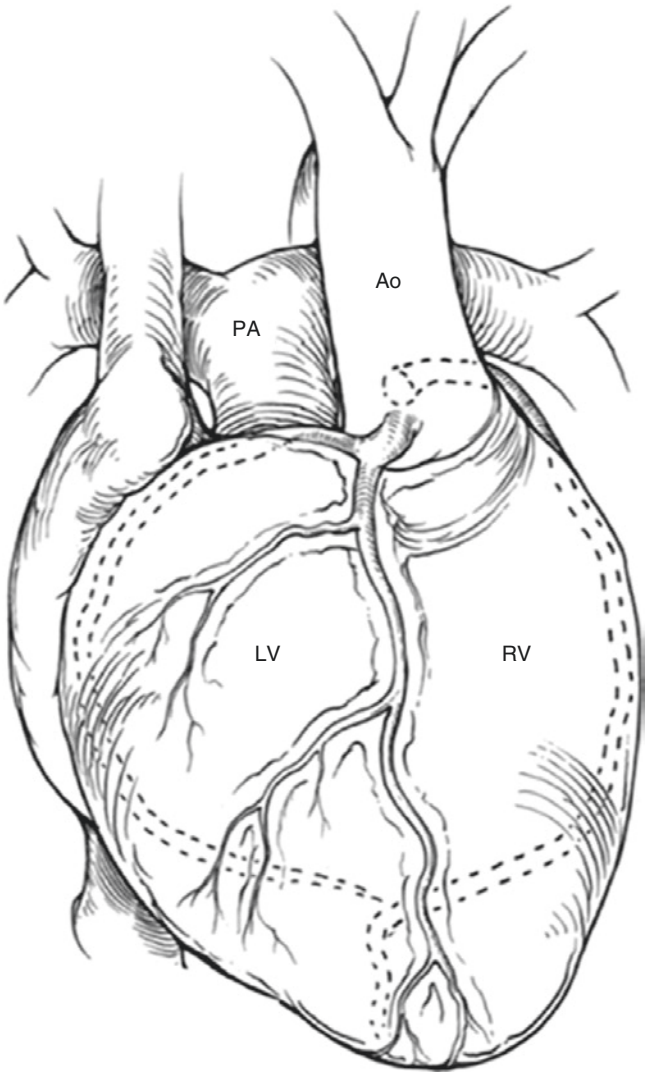


Fig. 100.1 External topography of congenitally corrected transposition of great arteries (ccTGA). There is atrioventricular and ventriculo arterial discordance. The aorta is anterior and to the left of the pulmonary artery (l-malposition). The morphologic right ventricle is leftward (l-loop)

close to the commissure between anterior and posterior mitral valve leaflets. This accessory AV node gives rise to a long penetrating His bundle which runs through the fibrous trigone and passes subendocardially anterior and caudal to the pulmonary valve annulus. It continues as the left bundle on the morphologic left side of the septum while the right bundle penetrates the septum to fan out on the right morpholic side of the septum. In the presence of VSD the His bundle runs in close contact with the pulmonary valve annulus and proceeds to the anatomic left side anterior and superior rim of the VSD (Fig. 100.2). In few cases with dextrocardia there are anterior and posterior AV nodes with

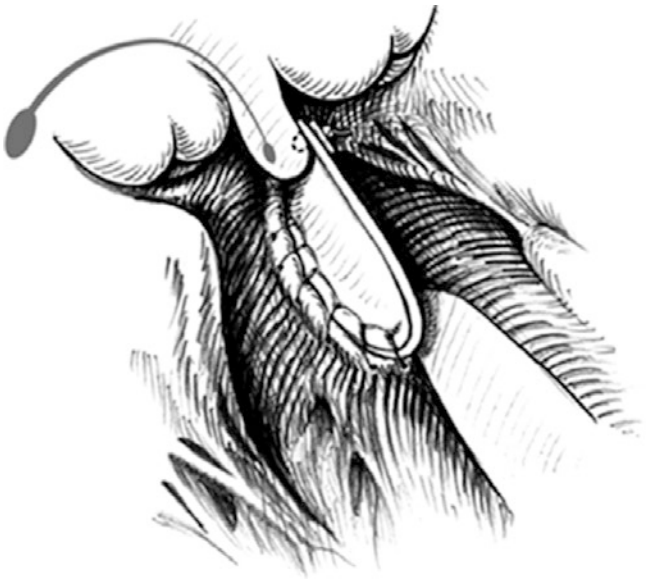


Fig. 100.2 Conduction system. The atrioventricular node (AV) node is anterior. The His bundle passes anterior and caudad to pulmonary valve and the suspension vein of ventricular septal defect (conversed partially with patch)

His bundle arising from one or both. In situs inversus the posterior node is normally located, and the penetrating bundle is located at the inferior and posterior rim of the VSD similar to hearts with atrioventricular concordance. The long and abnormal course of the His bundle predisposes it to continued mechanical stress and could result in spontaneous complete AV block at a rate of 2% per patient year [10, 11].

Pathophysiology and Clinical Presentation

The clinical presentation of patients with ccTGA is varied, because of the diversity of associated defects. In the absence of other malformations, patients with ccTGA are asymptomatic early in life. However, progressive deterioration of the systemic morphologic right ventricle could occur with age. Symptoms of heart failure might develop when the patient reaches the forth decade of life. Concomitant tricuspid valve regurgitation due to annular dilation caused by a dilated ventricle and the onset of spontaneous complete AV block could accelerate the onset of symptoms [12–15].

When associated lesions are present, the clinical course is dictated by these lesions [12]. An isolated large VSD results in progressive congestive heart failure early in life when the pulmonary vascular resistance starts to drop. Late onset of progressive, irreversible pulmonary vascular disease occurs later if the condition is not treated surgically.

Isolated pulmonary stenosis results in morphologic left ventricular hypertension, which if not severe, is well tolerated. Severe pulmonary stenosis or atresia presents with hypoxemia and requires early surgical intervention.

When pulmonary stenosis is present with VSD, circulation is usually balanced, and oxygen saturation is adequate. Most of those patients do not need palliative procedure or complete repair early in life. As pulmonary blood flow decreases with time due to progressive obstruction of morphologic left ventricular outflow, the resultant increase in the degree of desaturation necessitates palliative shunt or definitive intervention.

Isolated tricuspid insufficiency due to valve abnormalities varies in severity. Progressive dilatation of the morphologic right ventricle and shifting of the septum towards the left ventricle due to decrease in its cavitary pressure adds to the severity of the regurgitation and necessitates medical and/or surgical treatment.

Diagnosis

The chest X-ray in ccTGA has a characteristic straight left heart border due to the leftward and anterior position of the aorta (Fig. 100.3). The electrocardiogram may show conduction disturbances and a Q wave in right pericardial lead. Two-dimensional echocardiogram reveals apical displacement of tricuspid valve. The right ventricle is identified by the presence of the moderator band and the extensive trabeculation of its wall (Fig. 100.4). The atrioventricular and ventricular arterial discordance is quite evident. Cardiac catheterization is needed to evaluate the different hemodynamic parameters.

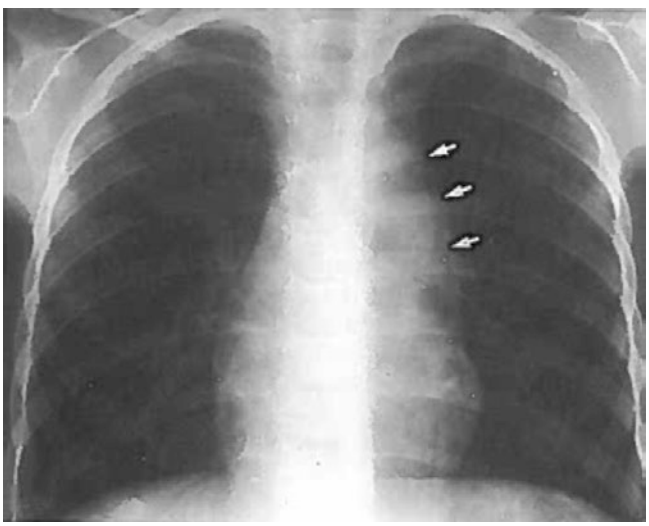


Fig. 100.3 Chest X-ray of patient with congenitally corrected transposition of great arteries (ccTGA). There is straight left heart boarder due to l-transposition of the aorta

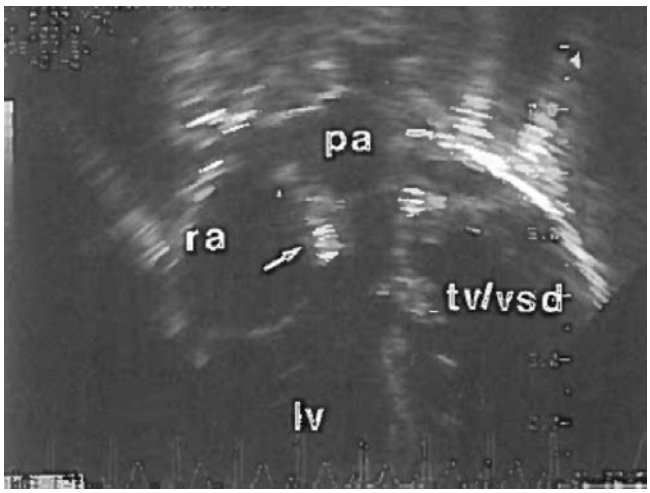


Fig. 100.4 Echocardiographic image of congenitally corrected transposition of great arteries (ccTGA). The pulmonary artery arises from morphologic left ventricle. The right ventricle is identified by the tricuspid valve and the moderator band

Surgical Treatment

The surgical approach to patients with ccTGA depends on the presence and severity of the associated lesions.

ccTGA with Isolated Left Ventricular Outflow Tract Obstruction

It is unusual for this combination to cause any significant symptoms. Although the morphologic left ventricle is hypertensive (close to systemic pressure in some cases), it can readily handle the increased afterload by virtue of its morphology and function. As a result, treatment might not be needed until the patient is older. There is also an added benefit of leaving a moderately hypertensive left ventricle. It minimizes the interventricular septal shift away from the dilated right ventricle. Consequently, it reduces ventricular wall tension and tricuspid valve annular dilation. Several procedures have been used to relieve the left ventricular outflow tract (LVOT) obstruction. These include resection of the subvalvar obstructive tissue with or without pulmonary valvotomy. Although helpful in reducing the left ventricular pressure, this approach carries a high incidence of post-operative complete AV block, and has been generally abandoned. Placement of a valved conduit between the left ventricle and main pulmonary artery relieves the LVOT obstruction, but necessitates repeated surgeries for conduit replacement (Fig. 100.5). Addition of a pulsatile bidirectional Glenn shunt reduces the volume load on the left ventricle and the conduit thereby potentially decreasing the LVOT gradient and prolonging conduit functional life (Fig. 100.6) [16].

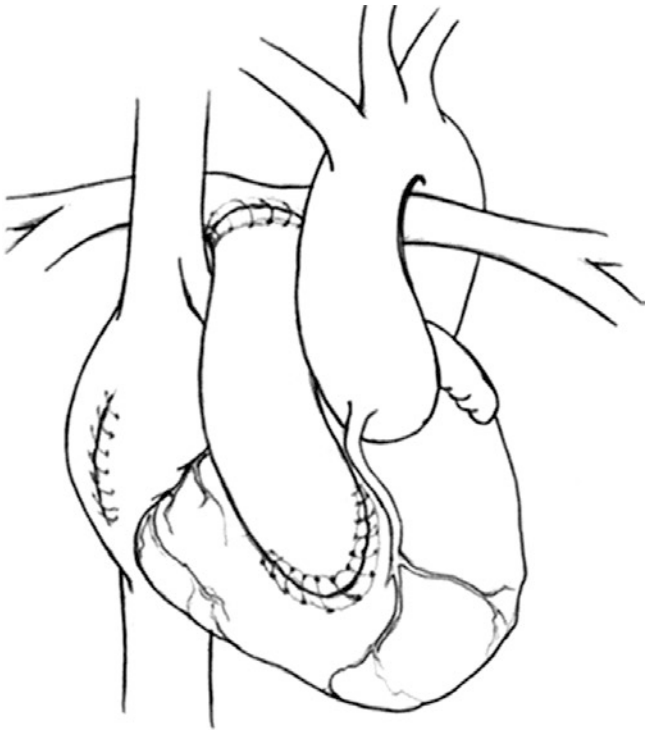


Fig. 100.5 The "physiologic" repair of patients with left ventricular outflow obstruction. A valved conduit is placed between the morphologic left ventricle (pulmonary ventricle) and the pulmonary artery

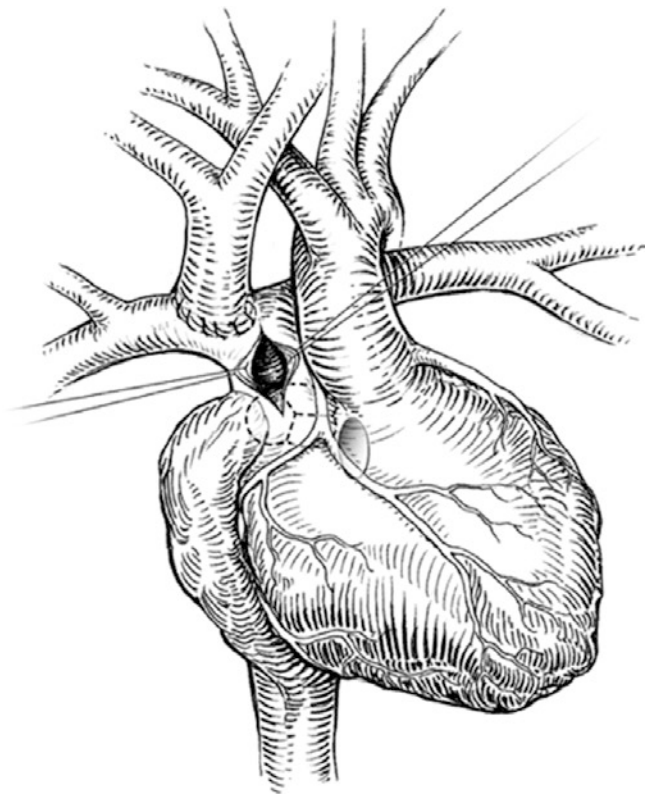


Fig. 100.6 A pulsatile Glenn shunt is used to bypass the obstructed left ventricular outflow tract. Additional valvuloplasty is performed through the pulmonary artery

ccTGA with Pulmonary Stenosis/Atresia and Ventricular Septal Defect

Until late 1980s, the surgical treatment of this combination of lesions was directed towards a physiologic repair leaving the right ventricle as the systemic ventricle. The VSD was closed through the mitral valve and the obstructed LVOT was bypassed by a left ventricle to pulmonary artery (LV-PA) valved conduit. Follow up of patients thus treated revealed several drawbacks to this repair [17]. The closure of VSD had a high incidence of postoperative complete AV block in spite of placing the sutures on the morphologic right side of the septum through the VSD (Fig. 100.7). The LV-PA conduit had a shortened functional life and required repeated interventions. Most importantly however, there was progressive deterioration of right ventricle and tricuspid valve functions leading to severe heart failure, transplantation or death. Due to these disappointing results, the concept of anatomic correction or the double switch operation (the Ilbawi Procedure) was introduced [18, 19]. It consists of an arterial and venous switch procedures. Through a right ventriculotomy, the VSD is tunneled into the aorta using a large synthetic patch fixed in place with interrupted and continuous sutures (Fig. 100.8). This morphologic right septal approach avoids the conduction system and practically eliminates postoperative AV block. This tunneling also directs the blood from the left ventricle into the systemic circulation thus establishing the left ventricle as the systemic ventricle.

A venous switch operation is performed to direct the pulmonary venous return to the morphologic mitral valve/left ventricle and the systemic venous return to the tricuspid valve/right ventricle (Fig. 100.9). A bidirectional Glenn procedure could be added to the venous switch operation (the so called hemi-Mustard) [20, 21] (Fig. 100.10). Finally, the pulmonary artery is transected. Its proximal end is over sewn and a right ventricle to pulmonary artery (RV-PA) valved conduit is placed.

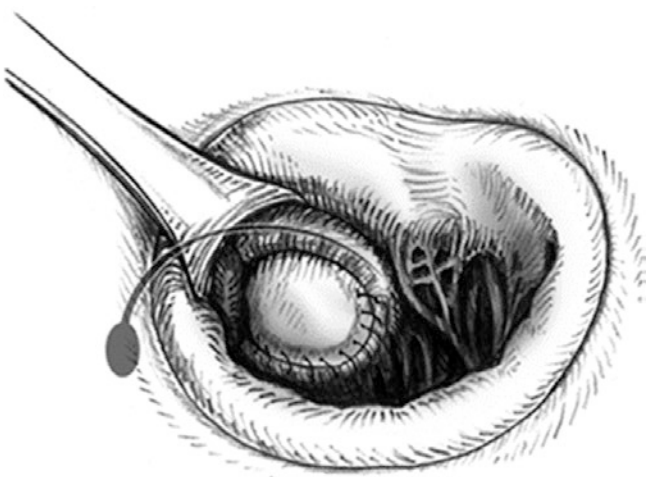


Fig. 100.7 Modified technique for closure of the ventricular septal defect. The sutures are placed on the morphologic right side of the septum superiorly and anteriorly to avoid the conduction system

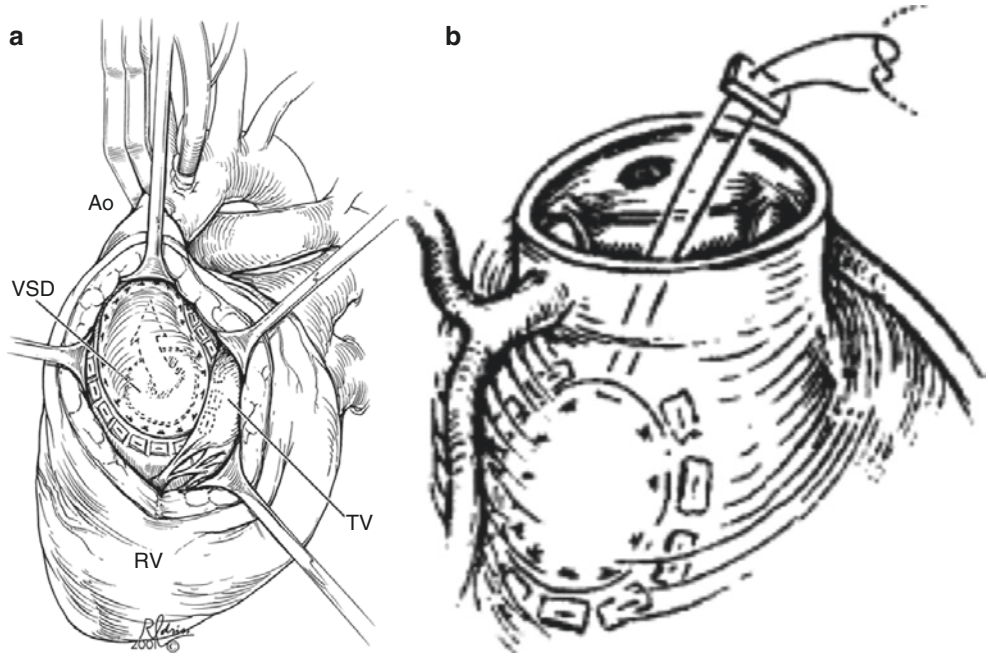


Fig. 100.8 (**a**) Closure of the ventricular septal defect (VSD) through a right ventriculotomy. Sutures are placed on the morphologic right side of the septum and around the aortic root to tunnel left ventricular blood flow into the aorta (Ao) through the VSD. The defect can be enlarged inferiorly if judged to be restrictive. (**b**) Closure of the VSD through the aortic root in cases ventriculotomy is not needed

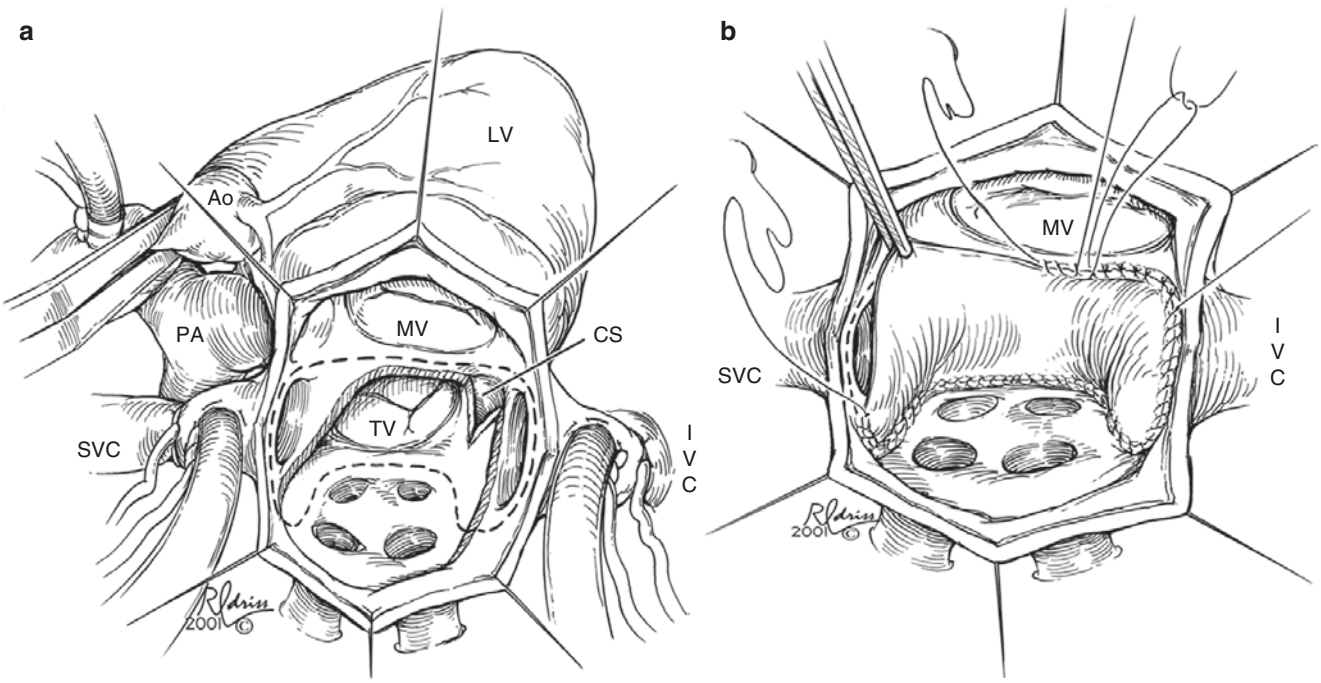


Fig. 100.9 Venous switch operation—Mustard procedure: (**a**) Total atrial septectomy is performed. The coronary sinus (CS) is unroofed towards the left atrium. The dotted line outlines the location of the intra atrial tunnel. (**b**) The constructed tunnel using untreated autologous pericardium. Interrupted sutures are used to secure pericardial patch to the floor of the unroofed CS. *Ao* aorta, *IVC* inferior vena cava, *LV* left ventricle, *MV* mitral valve, *PA* pulmonary artery, *SVC* superior vena cava

ccTGA with Tricuspid Valvar Insufficiency

Onset of tricuspid regurgitation is an ominous sign and mandates the need for early treatment. Tricuspid valvuloplasty in the form of enlarging the leaflets with autologous glutaraldehyde-treated pericardial patch combined with annuloplasty have been used. Valve repair in general has not been successful in this setting because of the very unusual pathology. However, it could be used with pulmonary artery banding as a temporizing measure to prepare the patient eventually for the double switch/anatomic repair and is probably the only good option for treating this subset of patients.

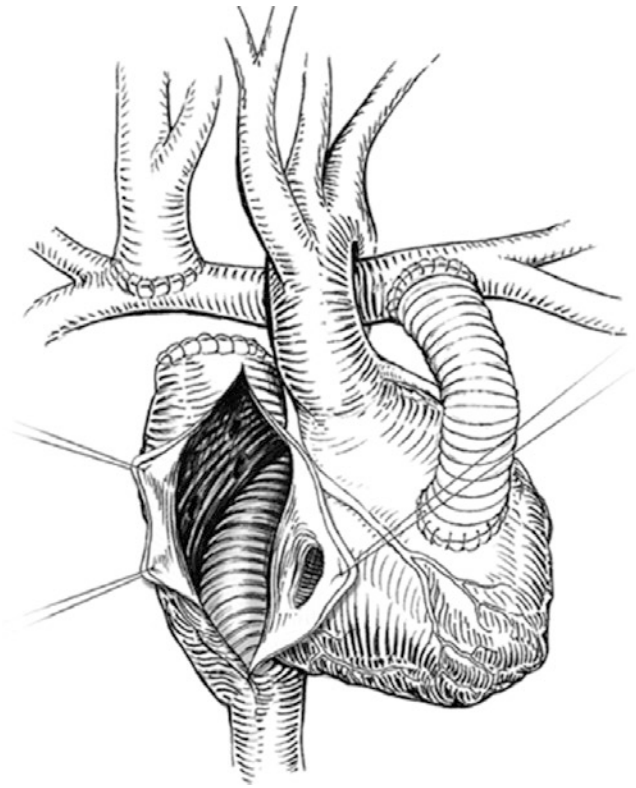


Fig. 100.10 The hemi-Mustard modification. The inferior vena cava is tunneled to the left atrium/tricuspid valve. A pulsatile Glenn procedure is used to direct the superior vena cava blood to the pulmonary circulation

The band might also help in preventing excessive shift of the septum towards the morphologic left ventricle, hence stabilizing the annuloplasty. Tricuspid valve replacement might be needed if regurgitation is severe. However, long-term results are not encouraging due to progressive right ventricular failure [17].

ccTGA with Isolated Ventricular Septal Defect

These patients are best treated with double switch operation and closure of VSD. The timing of the procedure is controversial. It could be performed in early infancy at the onset of symptoms or delayed by performing early pulmonary artery banding until the patient is around 6 months of age. This staged approach allows for growth of different structures, decreases pulmonary vascular resistance, and minimizes the risk of venous switch complications, while keeping left ventricle prepared for systemic pressure. Banding of the pulmonary artery, however, can potentially harm the neoaortic valve. The band, therefore, should be placed distally on the main pulmonary artery and the double switch operation preferably performed around 6 months of age, before onset of pulmonary root dilatation.

VSD is closed through the mitral valve with the sutures placed on the morphologic right side of the septum. It could also be closed through aortic valve (Fig. 100.11).

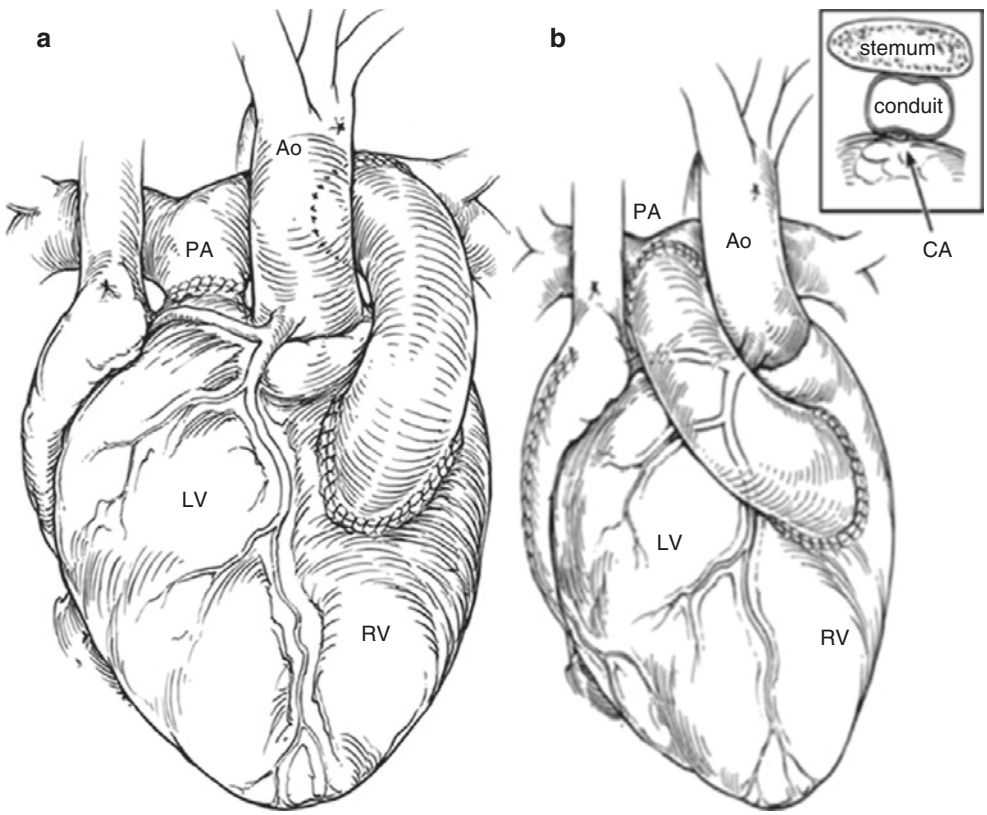


Fig. 100.11 (**a**) The right ventricle (RV) to pulmonary artery (PA) conduit is placed to the left of the aorta (Ao) after appropriate beveling. (**b**) The conduit is placed to the right side of aorta. In this location, there is potential risk of compression of the conduit by the sternum, and the right coronary artery (CA) by the conduit

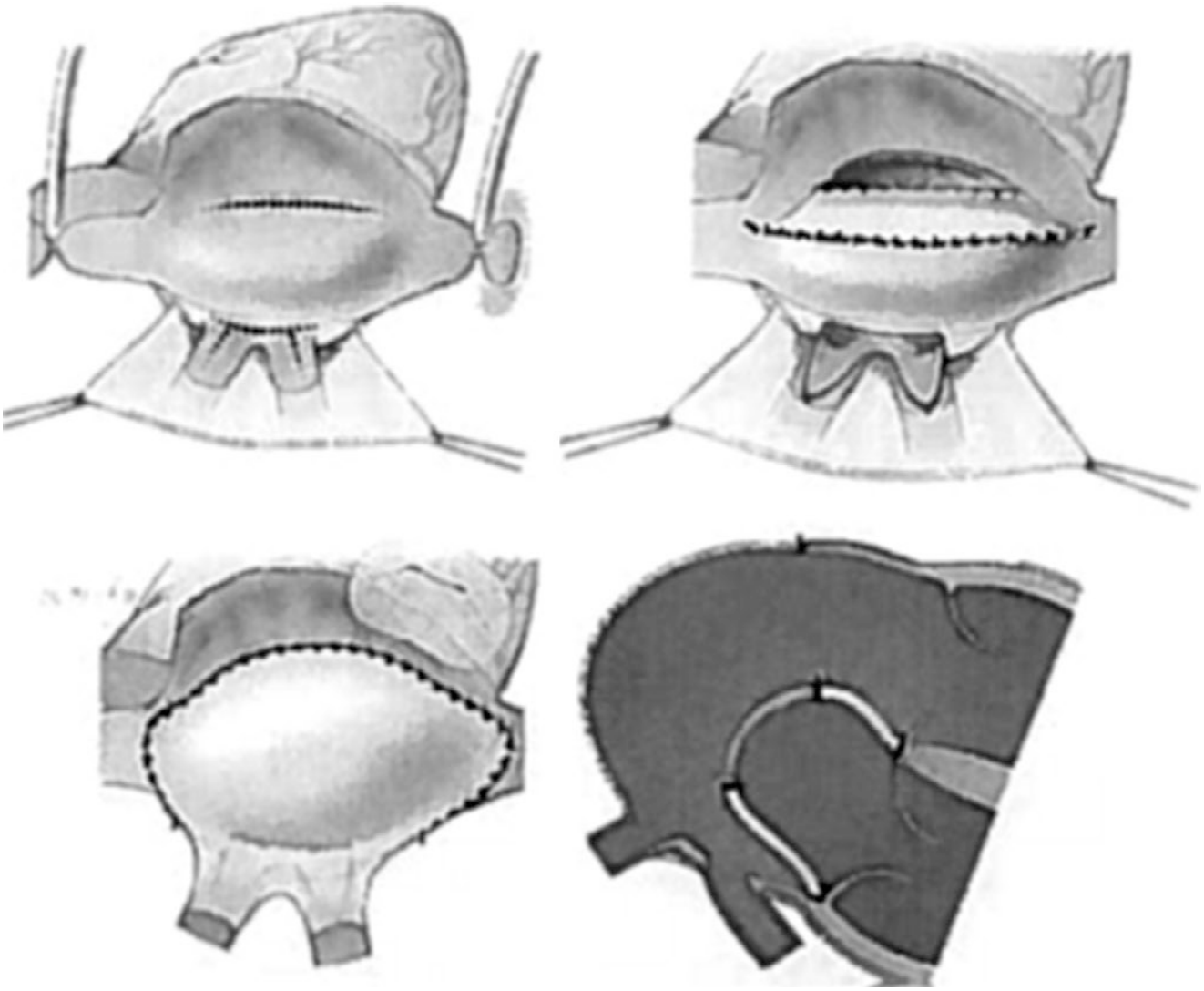


Fig. 100.12 Venous switch technique using the Senning technique. Dotted lines outline the incisions into the right and left atria. The detached septum is enlarged by patch material and sutured to the rim of the left sided valve. The external wall of the "functional" left atrium is then reconstructed with another patch

The venous switch operation is performed using the Mustard or Senning procedure (Figs. 100.9 and 100.12). The Mustard procedure has several advantages [22]. It is more versatile, and the suture line is easily modified to fit the space. It can be performed in patients with isolated dextrocardia where the right atrium is small and posterior. It has lower incidence of sinus node dysfunction and venous pathway obstruction. It can be performed in conjunction with bidirectional Glenn, the so-called hemi-Mustard procedure, which simplifies the technique and minimizes the complications of the venous switch (Fig. 100.13).

The arterial switch is performed in the usual fashion as previously described. Thorough mobilization of the coronary

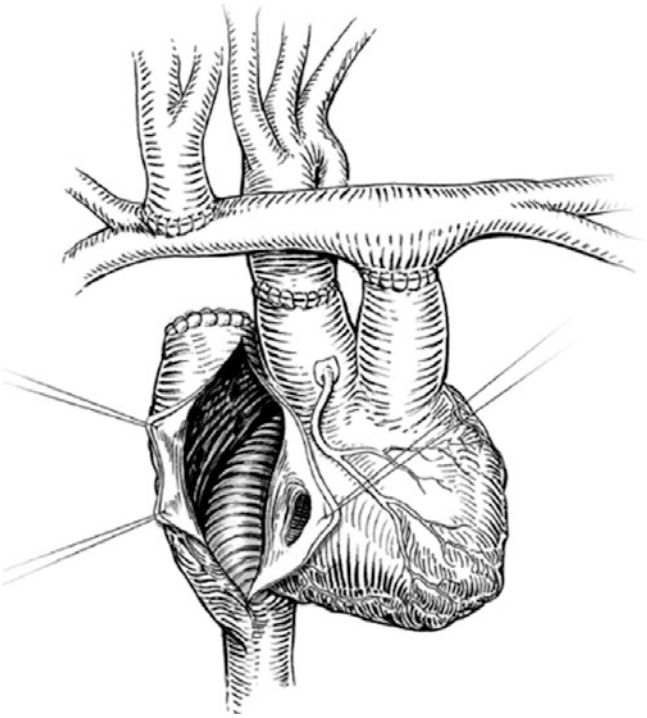


Fig. 100.13 Arterial switch operation performed in conjunction with hemi-Mustard procedure

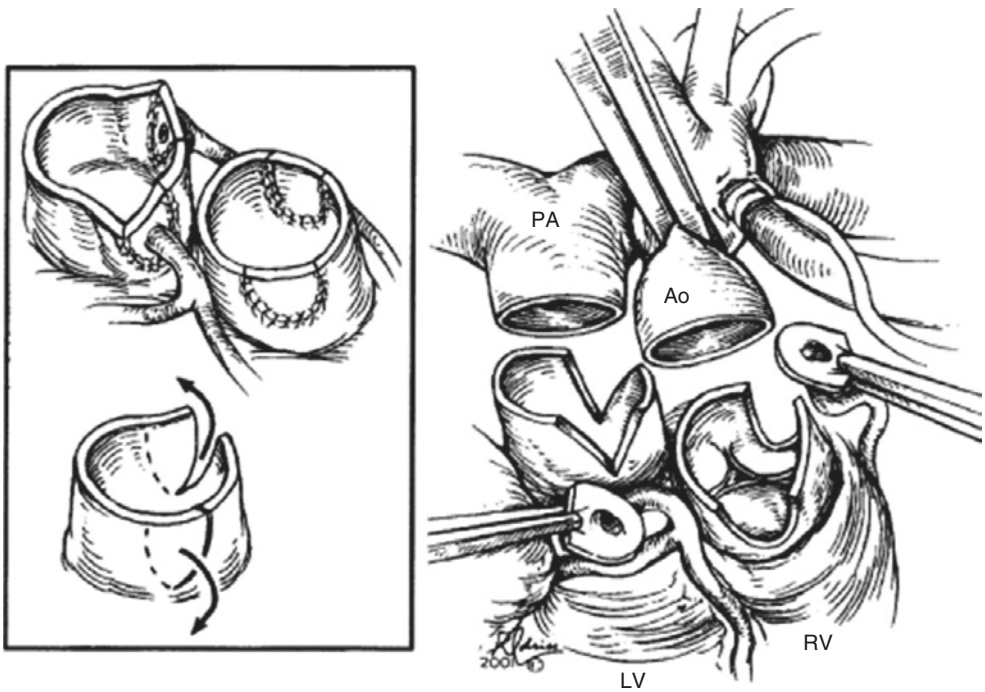
arteries and the use of a trap door technique have been especially helpful in ccTGA due to the orientation of the great vessels (Fig. 100.14). LeCompte maneuver may not be needed [23].

Alternative Procedures

When biventricular repair is not possible due to straddling AV valve, ventricular hypoplasia or distant muscular VSD, single ventricle palliation is an attractive alternative approach. It provides good midterm results in this subgroup where the left ventricle remains the main systemic ventricle.

Aortic translocation is another good alternative in patients with LVOT obstruction and distant ventricular septal defect. In this procedure the aortic root with the coronary arteries is detached from the underlying right ventricle and translocated into a longitudinally opened LVOT (Fig. 100.15). Ventricular septal defect is closed with a patch and RV-PA continuity is established usually without the use of a conduit.

Fig. 100.14 Details of the arterial switch operation. Trap doors into the neoaortic root are helpful to minimize distortion. Extensive mobilization of the coronary arteries is helpful



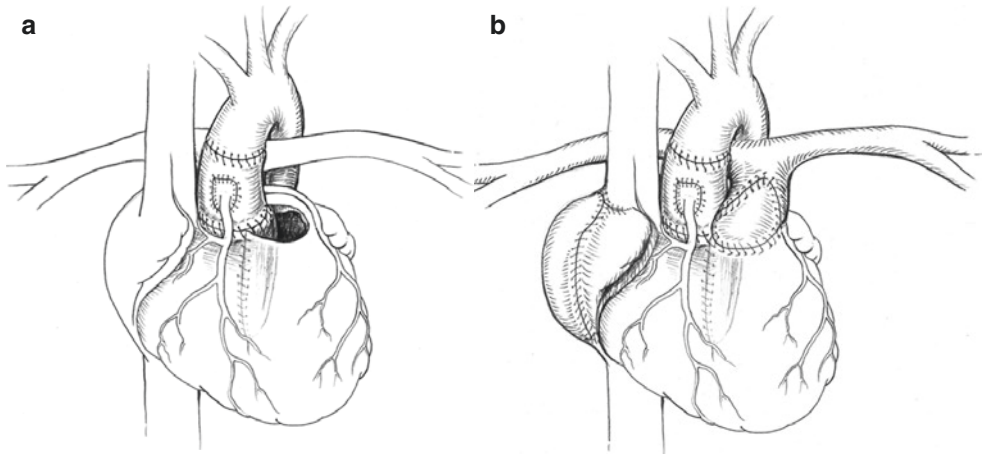


Fig. 100.15 (**a**) Aortic translocation for ccTGA with ventricular septal defect (VSD) and pulmonary stenosis. The aortic cannula is placed very cephalad. Aortic root is mobilized circumferentially. A wide rim of right ventricular (RV) free wall is left attached to aortic root. Extensive coronary artery mobilization is performed. The main pulmonary artery is transected proximal to bifurcation. A longitudinal incision into the left ventricular outflow tract (LVOT) is performed. The aortic root is sutured to the LVOT posteriorly and to a VSD patch anteriorly. Coronary arteries are either left attached to the aorta if there is no kinking or detached and reimplanted on the neonate. (**b**) The main pulmonary artery is connected to RV outflow tract and a patch is used to enlarge it

Results

Outcomes of the "physiologic" repair have been disappointing. Many reports have demonstrated progressive right ventricle failure in patients who have associated lesions. Survival was around 61.1% at 15 years postoperatively, and many patients needed transplantation. Patients with tricuspid regurgitation had the worst outcomes. These discouraging results of the physiologic repair prompted the adoption of anatomic repair as the treatment of choice. Excluding the learning curve results, short and long-term outcomes of anatomic repair have been excellent with operative mortality of 0–7%. Ventricular function is also preserved with near normal biventricular ejection fraction on follow up. Tricuspid regurgitation seems to improve, and perioperative complete AV block is rare [24–26].

Summary

Although the experience with double switch operation is limited to small series and varies with institutional experience, the results are generally very satisfactory, and the complications of venous switch operation have been minimal due to recent modifications. However, long term outcome is not available. It requires a longitudinal multi-institutional study to confirm that patients with ccTGA are best served by using the left ventricle as the systemic ventricle.

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