Aortopulmonary and Cavopulmonary Shunts

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Aortopulmonary Shunts: Blalock-Taussig and Modified Blalock-Taussig Shunt

Background

(a) The classic Blalock-Taussig shunt was introduced for surgical treatment of heart anomalies in 1944 by Alfred Blalock and Helen Taussig [1]. The procedure involves ligation of either the left or right subclavian artery with an end-to-side anastomosis to the ipsilateral branch pulmonary artery.
(b) The classic approach has been replaced by a modified Blalock-Taussig shunt (mBTS) (Fig. 12.1). This modification employs a polytetrafluoroethylene prosthetic graft to achieve a side-to-side connection between the subclavian and ipsilateral pulmonary artery [2].

Indication

(a) This aortopulmonary shunt is palliative for tetralogy of Fallot, d-transposition of the great arteries, and right ventricular outflow lesions and is also part of the staged repair of hypoplastic left heart syndrome.
(b) Other surgical aortopulmonary shunts include the central, Waterston, and Potts shunts. In addition, there are pharmacological methods of keeping the ductus arteriosus patent. The mBTS, however, continues to play a vital role in palliation of cyanotic congenital heart disease [3].
(c) Many congenital heart diseases such as tricuspid and pulmonary atresia, hypoplastic left heart syndrome, and double inlet ventricle must utilize the BTS when neonates have high pulmonary vascular resistance prior to performing a Glenn procedure and ultimately a Fontan repair. The latter provides complete cavopulmonary circulation.

Implication

(a) Distortion of the pulmonary artery, subclavian steal syndrome, and shunt occlusion can occur short-term [4, 5].
(b) In adults, patients who have undergone BTS may have a weaker pulse on the ipsilateral arm, even after takedown. This may have an effect on blood pressure readings. In addition, these patients may also exhibit a difference in arm size and strength [6].

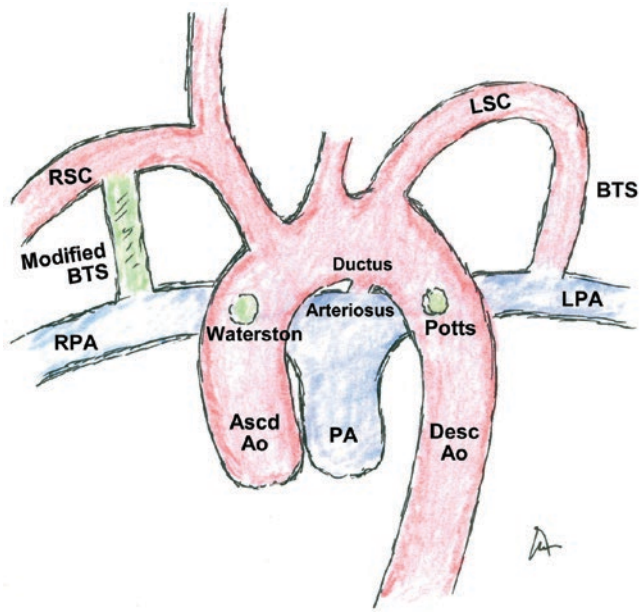

Fig. 12.1 Aortopulmonary shunts. (*R/LSC* right/left subclavian artery, *R/LPA* right/left pulmonary artery, *BTS* Blalock-Taussig shunt, *Ascd/Desc Ao* ascending/descending aorta, *PA* pulmonary artery)

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Cavopulmonary Shunts: Glenn, Bidirectional Glenn, Hemi-Fontan, and Fontan

Background

(a) The Glenn procedure involves separating the right pulmonary artery from the main trunk, separating the superior vena cava (SVC) from the right atrium, and anastomosing both ends together such that SVC blood flow goes directly to the right pulmonary artery (Fig. 12.2) [7].
(b) The bidirectional Glenn (BDG) involves separating the SVC from the right atrium and anastomosing the free end of the SVC to the side of the right pulmonary artery. The main pulmonary artery is separated from the heart and ligated. Flow from the SVC, therefore, is directed to both right and left pulmonary arteries (i.e., bidirectional) [8]. The BDG has replaced the original Glenn [2].
(c) The hemi-Fontan is a procedure that involves establishing a connection between the superior vena cava (SVC) and the right pulmonary artery (RPA). Either the SVC is transected and the ends are attached to the sides of the RPA or the RPA is transected and its ends are attached to the sides of the SVC. In both instances, the connection of the SVC to the right atrium (RA) is maintained, but flow into the right atrium is prevented by patch occlusion at the junction of the SVC and RA [9].

Indication

(a) These partial cavopulmonary shunts, usually done between 3 and 9 months of life, are palliative for right-sided atresia. The hemi-Fontan is the second of three stages for repair of hypoplastic left heart syndrome [2, 8, 9].

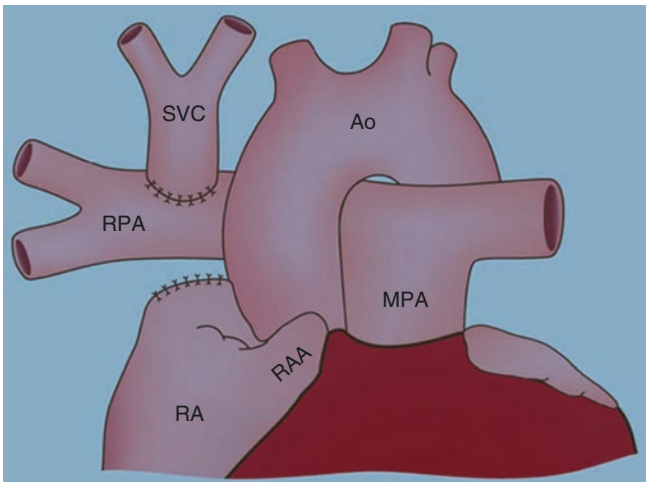

Fig. 12.2 Glenn shunt. Courtesy of Springer, [10]

Implications

(a) Pulmonary arteriovenous fistulas may develop from the lack of hepatic blood perfusing the lungs. Other complications may include shunt obstruction with SVC syndrome, atrial distention, arrhythmias, and atrial thrombus [3]. Cyanosis is common until completion of the Fontan correction involving the inferior vena cava.

Fontan

Procedure

(a) This procedure is done after partial cavopulmonary circulation has been achieved with either the bidirectional Glenn or hemi-Fontan. It involves connection of the inferior vena cava (IVC) to the right pulmonary artery (RPA). In the lateral tunnel Fontan, the IVC flow may be directed into the right atrium (RA) using both atrial wall tissue and synthetic material to connect to the pulmonary artery. This graft will grow as the child does.
(b) Alternatively, the IVC may be directed via a synthetic tube to the RPA beside the heart, an extracardiac Fontan. This graft will not enlarge as the child grows. With either procedure, an opening (fenestration) into the RA may be made to minimize rapid overload to the lungs [2]. Please see Fig. 12.3.

Indication

(a) The Fontan procedure is the last stage in creating total cavopulmonary circulation for those where it is not possible to have two functioning ventricles [11]. This includes hypoplastic right heart, tricuspid atresia, pulmonary atresia with intact ventricular septum, unbalanced atrioventricular canal, double outlet right ventricle, and double inlet left ventricle and is part of Stage III of the Norwood procedure for hypoplastic left heart syndrome [2].

Implications [3]

(a) Atrial arrhythmias and sinus node dysfunction are common. In addition atrial dilation and ventricular failure can occur.
(b) Cyanosis can develop with fenestrations. There is a high risk of thrombus formation. Pulmonary emboli can contribute to increased pulmonary vascular resistance which is particularly detrimental in the passively filled system.

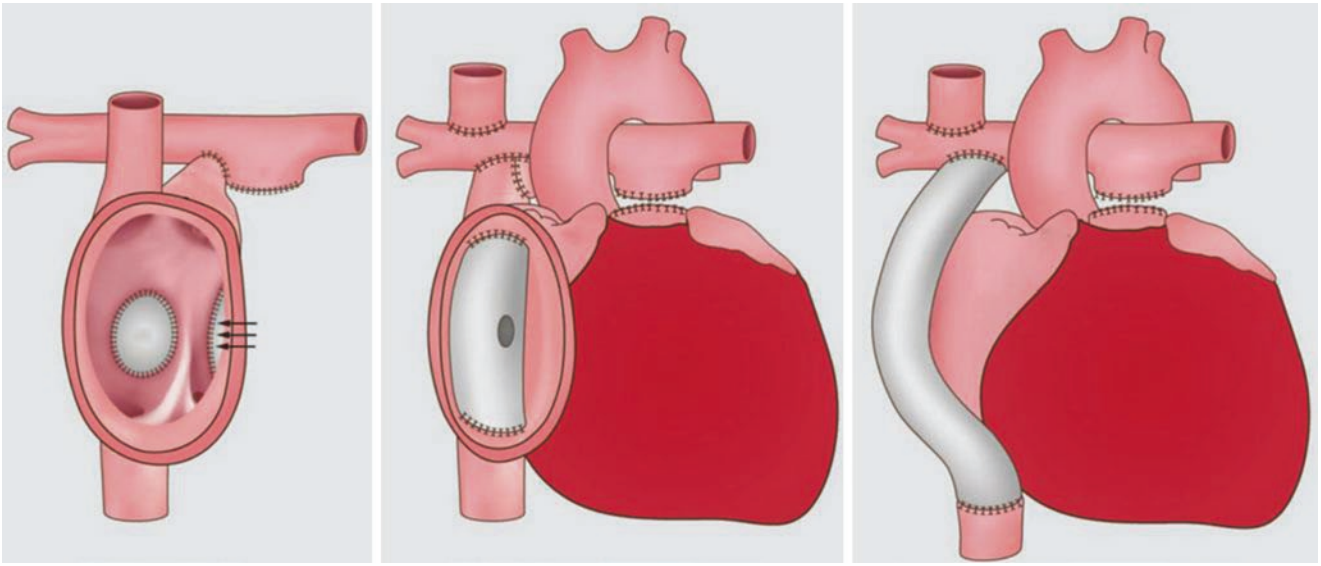

Fig. 12.3 The picture to the left represents a classic Fontan, the middle a lateral tunnel Fontan, and the right an extracardiac conduit Fontan. Courtesy of Springer [10]

Pulmonary hypertension and pulmonary artery stenosis can occur. In addition, arteriovenous malformations may develop as well as pulmonary shunts. Lymphatic dysfunction may lead to chylothorax, pericardial, and pulmonary effusions.

(c) Conduit stenosis and dilation and leakage of anastomoses can occur.

(d) Patients with long-standing Fontan circulation can develop hepatic congestion and protein-losing enteropathy. This results from systemic venous hypertension and can lead to cirrhosis, hepatocellular carcinoma, coagulopathy, polycythemia, and thromboembolism.

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