REVIEW ARTICLE

Palliative Surgical Procedures vs Duct or Right Ventricular Outflow Stent in neonates with ductus dependent pulmonary blood flow.

Felipe G. Rendón-Elías, MD, Mario Castro-Medina, MD, Gustavo A. de la Cerda-Belmont, MD, Gabriel Anaya-Medina, MD, Marely Hernández-Sánchez, MD, and Luis H. Gómez-Danés, MD.

Department of Thoracic and Cardiovascular Surgery, University Hospital "Dr. José Eleuterio González". Monterrey, Nuevo León, MEXICO.

One measure of progress in medicine is the introduction of new and superior treatment options, which naturally replace older forms of therapy. Sometimes, newly introduced treatment options compete against, rather than replace, older more established treatments. This is especially true when different forms of therapy for the same disease have their origins in different disciplines. In line with recent trends in the management of congenital heart disease, percutaneous catheter-based procedures have been introduced for managing cyanotic congenital cardiopathies with ductus dependent pulmonary blood flow in neonates. In this article we describe the surgical and percutaneous endovascular palliative procedures that can be performed in neonates having contraindications to performing a complete repair of their cyanotic congenital cardiac malformations, including their advantages, complications, results, and outcomes.

Key words: **Central Shunt; Duct dependent cyanotic congenital cardiopathies; Duct stent; Modified Blalock-Taussig Shunt; Right Ventricular Outflow Tract stent.**

Una medida del progreso en la medicina es la introducción de opciones de tratamiento nuevas y superiores, que naturalmente reemplazan las formas de terapia más antiguas. A veces, las opciones de tratamiento recién introducidas compiten, en lugar de reemplazar, tratamientos más antiguos y establecidos. Esto es especialmente cierto cuando diferentes formas de terapia para la misma enfermedad tienen su origen en diferentes disciplinas. En consonancia con las tendencias recientes en el tratamiento de las cardiopatías congénitas, han sido introducidos procedimientos basados en técnicas percutáneos para el tratamiento de las cardiopatías congénitas cianóticas con flujo sanguíneo pulmonar dependiente del conducto en recién nacidos. En este artículo describimos los procedimientos paliativos endovasculares quirúrgicos y percutáneos que se pueden realizar en neonatos que tienen contraindicaciones para realizar una reparación completa de sus malformaciones cardíacas congénitas cianóticas, incluyendo sus ventajas, complicaciones, resultados y desenlaces.

Palabras clave: **Fístula central; Cardiopatías congénitas cianóticas dependientes de conductos; Stent de conducto; Fístula modificada de Blalock-Taussig; Sent en el tracto de salida del ventrículo derecho.**

Cir Card Mex 2021; 6(2): 40-53.

In the las two decades there have been great advances in the neonatal cardiac surgery, that have allowed to perform corrective surgery at an earlier age avoiding the need of palliative procedures and become the primary repair the first choice in almost all the congenital cardiac diseases. However, despite the advances, palliative procedures still have indications in complex cyanotic heart disease duct-dependent. The systemic to pulmonary artery shunts or endovascular stent are done as palliative procedures for a variety of complex cyanotic congenital heart diseases. The spectrum may range from simple tetralogy of Fallot (ToF)/pulmonary atresia (PA)

Corresponding author: Dr. Felipe Gerardo Rendón Elías
email: felipe.rendonels@uanl.edu.mx

to complex univentricular hearts with major associated surgical problems. These palliative procedures provide the first line of management in these critically ill cyanotic neonates. They are less risky and provide regulated blood flow to the lungs allowing growth of pulmonary arteries for future complete repair. The infant also reaches a proper age and body weight suitable for definitive corrective repair [1].

CLINICAL SCENARIO

A ten days old male neonate, first child of a non-consanguineous couple, born at 34 weeks of gestational age by cesarean approach, weighing 2120 g. The Apgar scores were 9 and 10 at 1 and 5 min, respectively. The pregnancy was uneventful and there was no relevant family history. The infant was asymptomatic until 40 hours of life, when neonatal pulse oximetry screening for congenital heart disease detected hypoxemia with saturations of 70–85%. On clinical examination, there was no respiratory distress; however, she had a mild cyanosis that persisted. Septic screening was negative. He was started with infusion of Prostaglandin E2 and biphasic continues positive airway pressure. Echocardiography showed pulmonary atresia, severe infundibular stenosis, overriding aorta, right ventricle hypertrophy and non-restrictive subarterial ventricular septal defect (9 x2.5 mm). Computerized tomography of thorax reported, pulmonary atresia, small pulmonary annulus (z score -3.20), hypoplastic main pulmonary artery (z score -3), right and left pulmonary arteries hypoplastic (z score -4 and -2.86 respectively), patent arteriosus conduct, type A from the Krichenko classification.

The questions are:

1. *What palliative procedure will you select: surgical or endovascular procedure?*

2. *If you selected surgical palliative procedure, which shunt will be your choice: a modified Blalock-Taussing shunt (by thoracotomy or median sternotomy), central shunt or right ventricle to pulmonary connection?*

3. *If you decided for endovascular procedure, which technical will you select: duct stent or right ventricular outlet stent?*

PALLIATIVE SURGICAL PROCEDURES (Table 1)

Shunt procedures

A systemic-to-pulmonary artery shunt has been used for many years to establish unobstructed systemic blood flow, normalize pulmonary blood flow, pressure and relive pulmonary venous obstruction. Systemic-to-pulmonary artery shunts were first described by Blalock and Taussig (and Vivian Thomas) in 1945 (subclavian-to-pulmonary artery shunt) [2], Potts in 1946 (descending aorta-to-left pulmonary artery shunt) [3], Waterson in 1962 [4], and Cooley in 1966 (both of them as ascending aorta-to-right pulmonary artery shunt) [5].

An ideal shunt is expected to have the following attributes: technical simplicity, promote uniform growth of the pulmonary arteries, without causing distortion, good functionality, avoid excessive shunt resulting in significant diastolic run-off in the short term and elevated pulmonary vascular resistance or impaired ventricular and atrioventricular valve performance in the long term, good long-term patency, easy takedown before complete repair, no residual shunt after closure and no damage to anatomical structure that complicate a future corrective surgery [6].

Table 1. Palliative procedures for ductus dependent pulmonary blood flow in cyanotic congenital cardiac diseases.

Palliative Surgical Procedures
Pulmonary-Systemic Shunts
Modified Blalock-Taussig shunt
Central Shunt
Mee or Melbourne Shunt
PTFE central shunt
Right Ventricle to Pulmonary Artery Connection
Direct posterior RVPA autologous connection with anterior heterologous patch conduit
Pulmonary outflow tract reconstruction using the left atrial appendage
Transannular patch
RVPA connection using a non-valve prosthetic
Palliative Percutaneous Procedures
Patent Arteriosus Duct Stent
Right Ventricular Outflow Tract Stent

RVPA: Right ventricle to pulmonary artery connection

Modified Blalock-Taussig Shunt

While the classic Blalock-Taussig shunt (BTS) shunt was a great advanced in treating cyanotic heart diseases and performed in 1944 by Alfred Blalock of the Johns Hopkins University Medical Center [2,7,8], it was modified for de Leval in 1975 using a Dacron in the first cases and later graft polytetrafluoroethylene (PTFE) interposition graft to avoid the risk of interrupted subclavian artery flow and since that time was popularly known as the modified BT shunt [9]. The advantages of the modified BTS, that currently is the shunt choice at most congenital heart surgery centers include: preservation of the circulation to the affected arm, regulation of the shunt flow by the size of the systemic artery, high early patency rate, guarantee of the adequate shunt length and ease of shunt takedown [10].

The indication for modified BTS in TOF are limited to neonates with low birth weight (< 2 Kg), premature gestational age (<34 weeks), intraventricular hemorrhage, sepsis, pulmonary arteries structural anatomy no suitable for correction procedure, hemodynamic instability, multiple organ failure, non-cardiac malformations associated, anoxic spells, presenting any contraindication to cardiopulmonary bypass or a combination of these condition [11] .

The modified BT shunt can be performed through a right or left thoracotomy or a median sternotomy. The approach

depends on the anatomy of the pulmonary arteries, the subclavian artery, the presence and location of a ductus arteriosus, and the surgeon preference. Currently, (since 1990) the sternotomy approach is the choice in the majority of pediatric cardiac center because it saves the child from a second scar, avoids morbid damage to the thorax with prospects of late scoliosis, but more importantly, the target pulmonary artery being intrapericardial, it is more accessible for eventual reconstruction after takedown, technically easier, provide easy access for alteration of the plan, associated with fewer shunt failures [12,13], if necessary can be performed with the use of cardiopulmonary bypass, the shunt can be placed to a main artery if necessary, and the access to the ductus arteriosus is always possible. Other disadvantages of a thoracotomy approach enumerated in the literature include chylothorax, chylopericardium, chylous ascites, phrenic nerve paralysis, Horner's syndrome, distortion of lobar branch pulmonary arteries and preferential flow to one lung with unbalanced growth [13]. Disadvantage of the sternotomy approach does confront the surgeon with the challenges of a central run-off from the systemic artery leading to greater steal, low diastolic pressures, coronary malperfusion and pulmonary hyperperfusion. In addition, the often used truncus brachiocephalicus to the right pulmonary artery shunt may be at danger of being squashed between the dominant aorta and the superior vena cava, for which the parietal pericardial reflection over the superior vena cava to the trachea should be divided to create space for the shunt. The increased adhesions at the time of sternal re-entry has not really been an issue [12].

While the Boston group [13] reported four times higher risk of shunt failures through a thoracotomy when compared with a sternotomy, Shauq et al. [14] have reported significantly longer ventilation time, inotropic support, intensive care unit (ICU) stay and hospital stay in the sternotomy group. These findings reflect the learning curve involved with shunts created through a sternotomy [15].
Sachin Talwar et al. performed a research to find out the best evidence to answer the question: is sternotomy superior to thoracotomy for mBTS? From 58 paper studied they selected, 11 papers provided the best evidence. The studies that compared the thoracotomy and sternotomy approaches observed increased shunt failure rates in the thoracotomy group; the sternotomy approached was associated with advantages; like less pulmonary artery distortion , ease of technical performance, cosmetic advantage, ease ligation of the PDA, less phrenic nerve injury, less collateral formation in the chest wall adhesions and less thoracotomy induced scoliosis [15].

In case the approach was by sternotomy, there are some cases where the sternum must be left open , for example: if there were any fears about the fate of the shunt (especially in totally shunt-dependent pulmonary circulation) or about the shunt getting squeezed behind the aorta, the sternum is recommended left open. It was believed that an open sternum lends itself to a quick response in case of an emergency when compared with a closed chest.

In order to select the most appropriate shunt size, the pediatric cardiac surgeon use as a rule thumb, a 3-mm graft for children around 3 kg or lower in body weight, whereas a 3.5-mm graft is used for children around 3.5 kg; the indication, whether palliating for a uni or biventricular heart, is very important to select the size of the graft in borderline weight-class children [16].

The recommendations of ligating or not the patent duct are: 1) if the patients have forward flow through their main pulmonary artery is ligate the duct, 2) in shunt-dependent circulations, the patent duct is circumvented and almost obliterated using silastic sling and liga clips, to allow a quick rescue by re-establishing ductal flow in case of a shunt thrombosis emergency. In the end, whether or not to close the duct remains a strategic decision [14]. While a patent duct imparts significant safety in the case of a shunt failure, some reports have associated patent duct with shunt thrombosis [17]. Petrucci et al. (Society of Thoracic Surgeons [STS] database) have shown no association between closed duct and the risk of composite morbidity [18]. Closing or keeping the duct open during the MBTS procedure has advantages and disadvantages. Hence, it remains in the end, as an individual decision.

Anticoagulation regimen is decided on a case-by-case basis. Therapeutic heparinization are performed in high-risk shunt scenarios, such as shunt-dependent pulmonary perfusion, in cases with shunt clipping (shunt size reduction) or technical problems encountered during shunt construction. Heparin infusion starting with 5–10 IU/kg/h, followed by therapeutic dose as early as 2 h postoperatively, is started, if surgical bleeding was not an issue. Shunts without complications and considered normal risk received aspirin in the long term.

Several single center reports evaluated the outcomes of shunt surgery, but the majority was published in an earlier era. These studies reported mortality ranging from 3.7% to 14% [13,20-24]. Among these, Gold et al. reported the lowest mortality rate, but the patients in their series had a mean age of 3 months [22]. Williams et al. reported on the largest series of mBTS, where they analyzed 2,000 patients over a period of six decades in a multicenter study with an overall mortality of 14%. However, patients who were older than a neonatal age were included in this report [25]. More recent publications analyzed the outcomes after shunt surgery and reported on only neonates [18,20,24]. Petrucci et al. included 1,273 patients using the multi-institutional STS Congenital Database. Remarkably, their data demonstrated that mBTS continues to be a high-risk procedure, with an overall mortality of 7.2% [18]. In 2014, Bove et al. reported on 150 neonates who had an overall hospital mortality of 8.7% after shunt surgery, with indications that excessive pulmonary blood flow could have contributed to mortality in the sternotomy group [20]. These findings point towards over-shunting and low body weight as a possible indicator of mortality in different series. An STS database harvest study [18] has identified preoperative ventilation, pulmonary atresia and intact ventricular septum, univentricular hearts and weight <3kg as risk factors for mortality. While Alkhulaifi et al. [24] identified weight <2 kg and preoperative ventilation as important risks factors. Rao et al. [27] identified restrictive atrial septal defect, univentricular physiology and postoperative intervention as risk factors for mortality. In the study of Keti Vitanova et al. showed that acute heart failure or septicemia was the predominant cause of death in patients who died with a shunt in situ. In these

patients, no pathology was found on the shunt and they identified two risk factors for shunt related mortality in neonates that have not been previously reported: a platelet transfusion and the implementation of postoperative ECMO [26].

Bove et al. reported that excessive pulmonary blood flow through shunts could contribute to mortality [20]. Dirks et al found that there was a trend toward increased mortality in patients in whom a shunt had to be decreased in size to reduce shunt size could be identified as a risk factor [16].

A great concern after shunt surgery is shunt failure because of thrombosis or a stenosis. Thrombotic occlusion of a mBTS is a devasting event for an infant dependent on shunt for pulmonary blood flow and can occur in the hours immediately after placement or before hospital discharge. Shunt thrombosis is a grave complication of the mBTS procedure, 11.8% reported from Bristol and 13.7% reported from Boston [13]. Clinical predictors leading to the occurrence remain unclear. Tsai et al. [28] and Tamisier et al. [29] have suggested that young age and smaller size are significantly related to shunt thrombosis. Other reports have also linked weight <2 kg [24] and weight <3.6 kg [20] to shunt thrombosis. Gedicke et al. [17] have found weight <3 kg, high preoperative hemoglobin (>18 g/dl), postoperative transfusion of red blood cells, the pulmonary artery diameter, and a postoperative patent duct as significant factors for shunt thrombosis.

Current evidence supports the existence of an intimate relationship between inflammation and thrombosis [30-32]. The acute-phase response protein CRP is highly involved in inflammatory processes and possesses substantial prothrombotic properties [33]. In a recent article, Cholette and associates [34] identified an association between elevated preoperative CRP levels and an increased postoperative thrombotic risk in neonates undergoing initial palliative surgery.

At the beginning of performing mBTS because of the complications using of synthetic vascular prosthesis like life-threatening late infections, human vascular conduits were applied, obtaining a good results and functioning well up to six years, but after improve the synthetic vascular prosthesis, the saphenous vein graft was not employed for pediatric cardiac surgeons but lately Resham Kaur et al. conducted a retrospective study of 129 consecutives patients who were palliated with shunts between 2006 and 2005; 69 saphenous and 58 PTFE conduct. There was a mortality of 12% and reported thrombosis in 5.2% of the saphenous modified BTS group and 20.6% of those with PTFE mBTS. This study showed that cryopreserved saphenous vein is safe alternative for mBTS and central shunt [35] although in 2008 Arvind Kumar and his teamwork reported no benefits using homograft saphenous vein [36].

Although an association between an anticoagulation regimen and shunt thrombosis could not be established, it does not be little the role of postoperative anticoagulation, particularly in high-risk patients. Al Jubair et al. [37] have shown, less-shunt failure occurs if heparin is given before clamping. An early postoperative phase with a fresh anastomosis, coupled with phases of low systemic pressures, pulmonary hypertension, external compression and resulting stasis, can initiate thrombus formation. It is these uncertainties that can be positively influenced by early anticoagulation. Li et al. [38] have demonstrated a beneficial effect of acetylsalicylic acid in infants palliated with a shunt, with reduced incidence of shunt thrombosis and death. Another prospective study has shown the beneficial effect of hemodilution with a significantly higher shunt patency rate [39]. Rare coagulopathies, such as protein C deficiency [40], and primary antiphospholipid syndrome [41] have also been reported to cause shunt thrombosis.

Late shunt obstruction has been reported as a cause up to 15% of out-of-hospital mortalities [18,22]. Wells et al. [42] have observed >50% obstruction of the MBTS in 21% of their patients and have identified a shunt size of <4 mm to be a risk factor for high- grade stenosis (>50%).

For neonates or infants with a small pulmonary annulus or severe right ventricular outflow tract obstruction, an early complete repair is the most recommended and preferred option it is safe and effective, with low mortality in contrast with the relatively high mortality with the BTS [16], but these patients in majority of the cases need a transannular patch, that in a future will cause pulmonary regurgitation, leading to several adverse events resulting from dilatation of the right ventricle, biventricular dysfunction, heart failure symptoms, serious ventricular arrhythmia, and sudden death [43]. To prevent pulmonary regurgitation after primary repair pulmonary annulus preservation without an incision to the annulus should be considered, so mBTS could be a good choice in small patients and avoid the devasting problem that provoke pulmonary regurgitation. Although recently the timing of the complete correction and the repair age are not believed to affect outcome, including the reintervention rate [44,45], preservation of an annulus of suitable size has been thought to be important in the RVOT for the prevention of PR in the long term after the repair [46–51]. The mBTS has advantages in annulus growth and annulus preservation. the BTS increased pulmonary annular size and left ventricular volume, such that approximately two thirds of patients previously thought to be candidates for only a transannular patch can have a pulmonary valve–sparing operation, so it is necessary to research more about if the stage repair is a convenient strategy in neonates without contraindication for complete repair.

Central shunt

An alternative method of delivering additional pulmonary blood flow is to construct a shunt directly from the aorta rather than from one of its branches. If the origin of the shunt is from the aorta, then it is referred to as a "central shunt" (the term does not refer to a median sternotomy approach, since the shunt could be performed via sternotomy or thoracotomy). These shunts have the advantage of delivering higher flow. Classically, central shunts were performed as direct anastomoses between the back of the ascending aorta and the right pulmonary artery (Waterson shunt) [4] or between the front the descending aorta and the left pulmonary artery (Potts shunt) [3]. These are now historical interest only.

Where a diminutive pulmonary arteries (PA) is present, staging procedures have been employed in an attempt to attain PA growth. These procedures include various types of central shunts as well as right ventricular outflow tract reconstruction. The creation of a central shunt appears to be the best way to provide increased pulmonary blood flow to the small native central PAs in order to warrant patency, pulmo-

nary arteries growth, and without distorting the branches. To create this type of shunt one can, use the direct anastomosis of the main pulmonary artery to the left and posterior side of the ascending aorta currently known like Melbourne shunt (Dr. Mee´s shunt) or using PTFE graft with anastomosis end to end or end to side in the pulmonary artery or using a cryopreserved saphenous vein the ascending aorta to the most appropriate pulmonary artery [51-58].

Melbourne or Dr Mee´s Shunt

This shunt was first described by Dr Mee´s group [59] from Melbourne, Australia in 1991. If the Melbourne shunt is the only procedure to be performed, it is performed through a median sternotomy. A left thoracotomy is used if left-sided unifocalization is performed at the same time. In either case, cardiopulmonary bypass is not usually required. The ascending aorta and the diminutive main and branch PAs are mobilized and silastic snare or soft clamps are placed around left and right PAs. Heparin(1mg/kg) is administrated intravenously and the left side of the ascending aorta is side clamped as posterior as possible. A button of aortic wall is excised, and transected main PA is mobilized and anastomosed end to side to the aorta [60,61].

The advantages of the Melbourne shunt technique compared with a standard systemic-to-pulmonary artery shunt are numerous: 1) the tissue-to-tissue anastomosis greatly reduces the risk of thrombosis, 2) it is less likely to result in distortion of the branch pulmonary arteries, which by definition are markedly diminutive at the time of this operation and 3) the central location of the shunt usually results in balanced blood flow distribution to the right and left branch pulmonary arteries [60].

The performance of a Melbourne shunt has reliably provided for uniform growth and development of central pulmonary arteries but in some cases can lead to congestive heart failure or more rare pulmonary vascular occlusive disease. The stenosis of the right pulmonary artery is reported in almost 50% of the cases, and to prevent these is very important to perform the anastomosis the most posterior possible in the ascending aorta [62]. Nevertheless, some groups found this technique to be unreliable [63].

Central shunt using PTFE

In the case of patients have confluent central branch pulmonary arteries, performing a central PTFE shunt is recommended. This prosthetic shunt will regulate flow and pressure and lessen the possibility of excessive pulmonary blood flow or a reperfusion injury to the lung, or both.

The Laks Technique is the recommended procedure to construct a central shunt with PTFE. This procedure can be performed without the need for cardio-pulmonary bypass, nonetheless, it is recommended full pump standby in such situations. After median sternotomy with pericardial opening, the pulmonary branches are mobilized as required, the main pulmonary artery is mobilized sufficiently to allow the placement of a C-clamp on its anterior surface and snared. A single 5/0 suture is passed at the base of the main PA. Traction on this suture and the two snugger provide optimal exposure. A longitudinal incision is made on the main PA, created under the C-clamp located central and not directed towards either branches. Care is taken not to interfere with a patent ductus arteriosus and to keep the C-clamp proximal on the main pulmonary artery so that flow from the left to right main branches of the pulmonary artery can occur through the distal main pulmonary artery. The largest possible shunt is inserted at this level (based on age/weight at the time of procedure), usually 3.0 to 3.5 mm. The extremity of the shunt does not need to be beveled as a transverse cut fit adequately to vessel opening. A slight upwards or transverse course gives the best curvature to the shunt. A rectangular slit incision is made on the shunt and a side-biting clamp is applied on the ascending aorta. The proximal orifice is made with a punch of 2.8 or 3.5-mm. Care should be taken to avoid positioning any chest drains across the path of the shunt [64].

Laks et al. [64] found this technical modification of the central shunt to be reasonably easy to construct. Its advantage over the end-to-side aortic technique is that the shunt assumes a short and straight course from the pulmonary artery to the aorta, this modification reduce the incidence of thrombosis, by reducing surface area exposure and the potential for kinking, also avoid excessive shunt flow and prevent congestive heart failure and is very easy to performed. Further, the shunt is easy to take down and unlikely to be injured on repeated sternotomy.

Alternative technique is used is a U-shaped central shunt with side to side aorto-PTFE tube graft anastomosis in the anterior wall of the ascending aorta and end to end main pulmonary artery or side to side in ascending aorta and main PA [65].This technique also have the same advantages than the Laks technique. Kim and his team [65] preferred to use the U-shaped tube graft when the main PA segment is sizable, to make an end to end anastomosis between the main PA and the graft and his method is very similar than the Melbourne group.

Both techniques have had good results with in-hospital mortality around 0 – 4%, demonstrated that are effective for rehabilitation of diminutive PAs and complete repair is possible in more than 70% of the cases.

Palliative Right Ventricle to Pulmonary Artery Connection

Another palliative procedures to increase the pulmonary arteries blood flow, in order to increase the size of the diminutive PAs and promote pulmonary artery rehabilitation are the construction of a connection between the right ventricle and PA (RVPA) connection that allows for an increase in the pulmonary blood flow without pulmonary hypertension restoring an anterograde flow with a route for subsequent catheterization and improves survival due to a lower incidence of obstruction or thrombosis.

Strategically there are four techniques for surgical reconstruction depending on anatomical configuration and surgeon preference, the techniques are: a) direct posterior RVPA autologous connection with anterior heterologous patch conduit, b) the pulmonary outflow tract reconstruction using the left atrial appendage c) transannular patch and d) connection using a non-valve prosthetic in case where there is no the main PA (PA/VSD types II and III)[66-68].

All these procedures are performed through a median sternotomy under normothermic cardiopulmonary bypass (CPB) using bicaval cannulation and left ventricle venting.

The heart is arrested with antegrade Custodiol or Del Nido cardioplegia. The proximal branch pulmonary arteries together with the bifurcation are mobilized extensively and the arterial duct ligated and divided if present. Right atrial and left atrial pressures are monitored at the end of the procedure. In general, the best choice is to use autologous tissue to achieve reconstruction of the posterior wall of the RVPA connection to minimize the use of heterologous materials, in particular prosthetic conduits, at the time of complete repair. The direct posterior RVPA autologous connection is preferred in cases of TOF and PA type I and III, which comprised a vertical incision of the main PA in continuity with an infundibular incision of 5–8 mm, together with limited resection of right ventricle outflow tract (RVOT) muscle bundles. Great care is taken to avoid injury to the coronary arteries when opening the infundibulum, particularly in low birth-weight babies with pulmonary atresia, where the distal infundibulum is in close proximity to the left anterior descending artery (LAD), aorta and right coronary ostium. In cases with marked hypoplasia of the pulmonary trunk or atresia, posterior continuity of the RVPA connection is augmented using interrupted sutures to approximate the distal margin of the ventriculotomy with the proximal end of the pulmonary arteriotomy. The anterior wall is reconstructed using a glutaraldehyde autologous pericardium that is easier to handle with the smallest pulmonary arteries or heterologous pericardial patch to avoid any risk of aneurysm or calcifications of the patch itself, initially sized at 5–6 mm (depending on the patient weight), around a Hegar dilator; this is subsequently adjusted off-CPB according to systemic arterial blood oxygen saturations, distal systolic pulmonary artery pressure and magnitude of the shunt through the VSD if necessary. In particular, where the central pulmonary arteries are of adequate caliber with no significant peripheral stenoses, care must be taken to ensure the RVPA connection will be sufficiently restrictive to pulmonary blood flow. The size adjustment is performed by means of a fine 'U'-stitch or a metal clip in the anterior heterologous patch.

The second method of reconstruction used (called 'autologous tissue reconstruction' due to the reconstruction of a posterior wall made of viable autologous tissue) this is the choice, in cases of PA (type II or III) with no main PA trunk. In this method, RVPA continuity was established without any extracardiac conduit; the pulmonary outflow tract is reconstructed using the left atrial appendage. After extensive mobilization of the pulmonary arteries and bifurcation, the left appendage was sutured to the distal end of the right ventriculotomy and to the pulmonary confluence that was left in its anatomical position. The entire posterior wall of the RVOT was thus made of autologous vascularized tissue. The anterior wall was reconstructed with a bovine pericardial patch, again sized at 6 mm diameter and adjusted if necessary.

The third technique for reconstruction employed are the transannular patch enlargement or also known like modified right ventricle outflow tract. This technique included median sternotomy, CPB, and beating heart or cardioplegic to allow and incision into the right outflow tract across the pulmonary annulus and ligate the PDA. The RVOT reconstruction is completed by transannular patching, using autologous or bovine pericardium according to surgeon preference.

The fourth technique for reconstruction employed a 5–6-mm polytetrafluoroethylene (PTFE) tube graft to connect the RV to the pulmonary bifurcation. This has been used in the presence of an anomalous left coronary artery crossing the infundibulum or if the surgeon is not familiar with the use of the LAA reconstructive method in case of no PA with no pulmonary main trunk.

The management strategy described above is adopted, for some pediatric cardiac surgery center based on previous studies [52,55,56,68,69], to undertake a neonatal RVPA connection in order to achieve cardiopulmonary stability, improvement in O2 saturation or preservation of pulmonary blood flow (for ductus-dependent circulation).

In addition, this protocol is aimed to optimize pulmonary artery growth and allow for MAPCAs involution, particularly in patients who would otherwise be considered too high risk for early complete repair [70-73]. Evaluation of the suitability of this strategy has to be made by taking into account the potential morbidity and mortality at any given stage as well as the adequate growth of pulmonary arteries. One should further aim to avoid iterative palliative surgery and should also consider the expected rate of successful complete repair and any potential need for a valved conduit.

The early mortality in these procedures is around 3.5%, an interstage attrition of 6.6%, and successful biventricular repair in 77% in series of neonates which comprised mainly patients with symptomatic neonatal TOF or AP/VSD. In comparison, Kim et al. [74] reported the outcome for initial modified BT shunt palliation in TOF/PA and a ductus-dependent pulmonary circulation with 22% overall mortality.

These results seem confirmed by various series analyzing the mortality after systemic to pulmonary artery shunt intervention in neonates with miscellaneous congenital heart diseases. The early mortality after systemic to pulmonary artery shunt was more than 8% [10, 75]. The best results with RVAP connection may be at least in part due to the avoidance of shunt physiology (diastolic run-off, impaired coronary perfusion and left heart volume overload). These considerations have some parallels with the arguments for using Sano conduits vs BT shunts in Norwood patients [76]. This is in spite of the potential advantages of the BT shunt including avoidance of CPB, cross-clamp and ventriculotomy, and in some centers the BT shunts are extremely rare nowadays at our institution.

The complications found with this strategies are: Pulmonary over circulations, that can be avoid decreasing the size of the connection, ligate the MAPCA, and minimizing the infundibulotomy and muscular band resection (is probably another key point in improving the tolerance to possible over circulation in TOF patients), and ventricular aneurysm.

PALLIATIVE PERCUTANEOUS PROCEDURES

Patent Arteriosus Duct Stent

Ductal stenting is an attractive alternative to conventional shunt surgery in duct dependent congenital heart disease as it avoids sternotomy or thoracotomy and its related problems. Currently, this technique has become more refined because of improved technology with low profile stents available in variable lengths and diameters, smaller delivery sheaths and a

whole variety of guiding wires leading to a marked improvement in outcome, but still the stenting of the PDA for ductal-dependent pulmonary blood flow (PBF) can be a technically challenging procedure. The first reports of PDA stenting for ductal-dependent PBF infants were published in 1992 by Gibss et al. [77]. The stents that were initially used for this procedure were hand crimped on balloons, thus necessitating the use of relatively bulky delivery systems and sheaths. Since then, a number of innovations in techniques, equipment, and subsequent experience of operators have all played a vital role in how the procedure has evolved over the recent years [78-85].

In fact, in the current era, PDA stenting for ductal-dependent PBF has been found to be an acceptable alternative to surgical BTS in select patients. In two relatively large multicenter studies, one reported from Glatz et al. and one from the United Kingdom, PDA stenting was found to be noninferior or superior to BTS placement with regards to primary outcomes, in addition to having a number of important other advantages over BTS placement [86-87].

Most infants with ductal-dependent PBF rely on PGE-1 infusion to prevent PDA constriction prior to stenting. A detailed transthoracic echocardiogram is performed to delineate the aortic arch anatomy, origin, and insertion of the PDA. In addition, it is important to note the morphology of the PDA in addition to the pulmonary artery (PA) anatomy. If the anatomy cannot be accurately discerned with standard transthoracic echocardiography, a computerized tomography (CT) scan may be obtained to obtain high-resolution definition of the PDA and branch PAs The typical duct in cyanotic CHD is elongated with one or more curves and arises more proximally from the underside of the aortic arch. It often becomes constricted as it inserts onto the pulmonary artery. Some ducts are unusually long and have a very tortuous course with one or more constrictions. Computerized tomography scan imaging allows the operator to exclude patients from the procedure in whom a highly tortuous or long PDA is present which may not be amenable to stenting, depending on operator comfort and center experience. In addition, complex PA anatomy may be better defined with CT imaging. Transthoracic echocardiography and CT scan imaging are both helpful in planning for the intended vascular access route, to ensure the most direct trajectory. If it possible the prostaglandin must be stop and later the size of the PDA is taken in order to select the best size of the stent and prevent complications.

The procedure is performed in the cardiac catheterization laboratory with general anesthesia and special attention be given to minimize hypothermia. It is imperative to have secure intravenous/central venous access in case the administration of inotropes or vasopressors; emergency medications or blood products are necessary. Heparin is administered to achieve an activated clotting time (ACT) of > 250 seconds. PGE-1 infusion may be continued during the catheterization, but in the event that the operator elects to hold the infusion, PGE-1 should be kept in the line. If there is a downward trend in saturations, the infusion can then quickly be reinitiated. Similarly, if ductal spasm is encountered during wire or catheter advancement in the PDA, PGE-1 must be reinitiated unless a stent is placed immediately after the ductal spasm occurs. The blood pressure should be optimized.

Special attention is given to the morphology of the PDA, as this can influence techniques and help anticipate important outcomes. Unlike the Patent ductus arteriosus as an isolated lesion, the ductus in cyanotic heart disease has a remarkable morphologic variability. The ductus tends to arise more proximally under the aortic arch, giving rise to a vertical ductus or occasionally it may arise from the subclavian artery. It also tends to be long and sometimes very tortuous, rendering stent implantation technically impossible. The ductus in these patients may also insert onto one of the branch pulmonary arteries with some stenosis at the site of insertion. So, it is very important to know the origin of the PDA and the tortuosity index (relatively straight, single turn, multiple turn) and the origin can be classified like as originating from the descending aorta, underside of the aortic arch, innominate artery, subclavian artery, or ascending aorta.

Ductal stenting may be done by the retrograde femoral artery route or the antegrade transvenous route depending on the ductus morphology and the underlying cardiac lesion. Vascular access is a vital part to the success of the procedure. It is crucial that the straightest trajectory to access the PDA is chosen based on preprocedural imaging. While PDAs originating from the descending aorta (and some from the head and neck vessels) are accessible with relative ease from a retrograde femoral or umbilical arterial approach, PDAs that originate from the underside of the aortic arch can be challenging to access from a femoral arterial approach [88].

The umbilical vessels may mitigate the risk of vascular access complications if available for use. For PDAs that originate from the underside of the aortic arch (and some from the head/neck vessels), percutaneous axillary artery or common carotid artery access facilitates a direct and straight trajectory which allows for easier maneuverability of guidewires/stent systems [89-92].

It is important that there is enough constriction in the PDA (constriction in one focal point will suffice) to allow a stent to be anchored securely. Measurements of the PDA are made to help choose the desired stent (s) length needed. Although a number of factors must be considered with regards to nominal stent diameter (eg, presence or absence of antegrade flow, length of PDA to be stented), in general, for infants weighing > 3.0 kg, a 3.5-4 mm diameter stent is chosen, for those weighing 2.0-3.0 kg, a 3.5 mm diameter and for those < 2 kg, a 3mm stent is chosen for deployment. The stents typically used for PDA stenting are newer generation, flexible coronary artery stents that are available in over-the-wire or monorail (eg, "Rapid Exchange") systems. Though primarily bare metal stents have been used, recently, drug eluting stents have been used for their potential benefits when deployed in PDAs.

If any hemodynamic instability occurs due to ductal spasm, quick decision making is crucial. At times, wire withdrawal should be performed to allow relief of ductal spasm if immediate stent implantation is not feasible. In other situations, quick placement of the ductal stent is necessary to reestablish stable PBF. However, if there is persistent hemodynamic instability, the procedure may need to be abandoned and the patient should be referred for a surgical shunt. Rapid availability of veno-arterial extracorporeal membrane oxygenator support (ECMO) can be live saving.

The complication most frequent encountered are vascu-

lar-related injury (9%), but also can occur acute thrombosis (2-3%), spasm of the ductal arteriosus (<1%), ductal dissection, migration of expanded stent, inability to enter the duct, incomplete stenting of full ductal length, and pulmonary artery branch stenosis [93].

The acute thrombosis is not a common complication, but it is very serious and life threatening. It presents in a dramatic fashion and requires immediate action. Angiography typically reveals thrombus occluding stent completely or partially. If the guidewire is still across the ductus, a small balloon (2.5-3 mm) may be passed into the stent and inflated several times, advancing the balloon forward towards the pulmonary artery each time. The purpose of this maneuver is to mechanically break up the thrombus. Thrombolytic therapy with streptokinase or recombinant tissue plasminogen activator is also recommended at the same time and maintained for at least 24 h. However, there is a real risk of bleeding complications with thrombolytic therapy, especially if a surgical shunt is required when adequate oxygenation cannot be maintained. Acute thrombosis occurs fairly soon after stent expansion; it is thus advisable that the wire be kept across the ductus for about 15 min before terminating a successful procedure. Acute thrombosis occurs even in those patients who are adequately heparinized during the procedure, because of, heparin's action is dependent on anti-thrombin III, which may be deficient in neonates. Attention to maintenance of adequate ACTs and administration of heparin for a period of time after the procedure may be necessary. It is not known if giving antiplatelet agents the day before the procedure would reduce this complication, but it may be worthwhile extrapolating this adult coronary intervention practice to neonatal ductal stenting. If ductal stenting gains wider acceptance, this is an area that merits investigation.

Spasm of the ductal arteriosus tends to occur with manipulation of guidewire and placement across the ductus. This is manifested by sudden deterioration in oxygenation as one is preparing the balloon-stent. If the guidewire is well positioned, it is a matter of rapidly going through the steps of stent implantation. However, if this occurs only at the stage of guidewire manipulation into the ductus, it is best to remove the wire and re-start the PGE1 infusion, recommencing the procedure after a period of stabilization. If the problem recurs, it is best to abandon the procedure and send the patient to surgery. Given its very low occurrence, it is not possible to predict which patient is likely to develop this complication. It is advisable to have the PGE1 infusion on standby in the catheterization laboratory.

Stent migration or malposition is not a life-threatening complication, but nonetheless serious as the patient needs to go to surgery semi-electively for stent removal and construction of a BT shunt. This is likely to occur if the pulmonary end of the ductus is not sufficiently constricted (>2.5 mm). If the ductal size is large is recommended not to proceed with catheterization and leave the patient without PGE1 infusion for a longer period if the patient is only mildly cyanosed and has evidence of significantly large ductal flow clinically and on echocardiography. Once an expanded stent has migrated to the pulmonary artery, it is not possible to remove it except surgically. With adequate allowance for ductal constriction by keeping patient off PGE-1, the chances of this complication are reduced. Nevertheless, if the ductus arteriosus is not adequately constricted at the time of stent implant, the stent can potentially migrate forward (or backward during balloon withdrawal). If the stent cannot be repositioned in the cardiac catheterization laboratory, it can be left in place (jailing of a branch PA or systemic arterial branch is usually not flow limiting) and dealt with at the time of subsequent surgical repair/palliation. Rarely, the stent may need to be acutely retrieved in the operating room with concomitant placement of a BTS.

Pulmonary artery branch stenosis could be provoked secondary to the intense neointimal proliferation and fibrosis in ductal tissues that extends into the media of the PA wall. It tends to accelerate pre-existing PA branch stenosis, requiring a salvage shunt [85,87,94]. This is especially true for patients with PA/VSD with tendency for ductal insertion into the proximal left pulmonary artery [84].

Patients are monitored in the hospital for at least 24 hours after the procedures with follow up chest radiographs and echocardiograms. Patients are started on aspirin 3-5 mg/kg/day and heparin is administered if there are any concerns of decreased pulses, occlusion of the access vessel/s or occurrence of stent thrombosis during the procedure. A decrease in oxygen saturations in follow-up should prompt a repeat cardiac catheterization for interrogation/dilation/restenting of the PDA or surgical repair/palliation if an appropriate time has been reached.

The durability of palliation by ductal stent is generally less compared to that from a surgical shunt (see below); hence, close follow-up and definitive surgery within 6-18 months are advocated [84,85,94-101]. The PA growth is good and in the majority of cases the stents are easily removed, with very few patients requiring reconstruction for PA distortion.

According to the 2011 AHA guidelines [102], PDA stenting with sole supply PBF is a Class II b indication (level of evidence: C). Similarly, the presence of branch pulmonary artery stenosis is currently listed as a Class III indication for PDA stenting (level of evidence: C). However, some groups, have stented in cases where a significant proximal branch PA stenosis is present (using techniques of intentional jailing of a branch PA and dilation/stenting through stent side cells). Partial or complete jailing of a branch PA (with or without preexisting branch PA stenosis) was noted in 22% of patients in our experience, more often with higher degrees of tortuosity [103]. This was also associated with greater planned/unplanned reintervention rates and PA plasty. However, in the long term, there does not appear to be any significant difference in branch PA size and symmetry whether a branch PA is jailed or not. It is important to note that though these above situations may not be an absolute contraindication to PDA stenting in all patients, they must be approached with caution and based on each individual center's experience. As with any other complex interventional procedure in a neonate, ductal stenting requires a high degree of technical skill, expert pediatric cardiac anesthesiology support, and ready access to surgical backup and ECMO support. The learning curve associated with PDA stenting has been demonstrated by Santoro et al [79,82,83]. With more expertise being accumulated in the recent years and the increase in the volume of cases, we should expect to see more widespread adoption of initial palliation with PDA stenting, even in highly tortuous

PDAs with challenging anatomy.

Techniques of ductal stenting for infants with ductal-dependent PBF have evolved rapidly over the last decade. Attention to anatomic and technical details optimizes the success of the procedure. The morphological classification scheme is helpful in anticipating acute and long-term outcomes of the procedure. It is likely that we will see further refinement of imaging, procedural techniques, and equipment used for PDA stenting in the ensuing years.

BTS vs PDA stent

In 1992, stenting of the patent ductus arteriosus (PDA) was de- scribed as an alternative to BTS placement to provide PBF, and the use of this technique has expanded considerably since that time. PDA stent has the advantages of being less invasive, thereby avoiding a median sternotomy or lateral thoracotomy, potentially avoiding exposure to cardiopulmonary bypass, and potentially allowing for more symmetric growth of the pulmonary arteries [82,83,104].

The previously touted potential disadvantages of PDA stenting include procedural complications unique to this procedure (primarily vascular access related, see above), concerns regarding stent longevity, and potential problems related to the ductal stent during subsequent surgical procedures [105,106].

There are few medical papers that directly compare the BTS vs PDA stent, and the majority just represent the single center experience where is included a small cases series and only give emphasis at specific lesions.

A 2009 study by Santoro et al. [107] looked at pulmonary artery growth following palliation with a PDA stent compared to a BTS at a single center between 2003 and 2009. The size of each group was small with 13 patients in the PDA stent group, and 14 patients in the BTS group. Both groups experienced significant growth of the pulmonary arteries as measured by the Nakata index and McGoon ratio, with no difference between the groups in terms of overall pulmonary artery growth. The PDA stent group did, however, promote more symmetric growth of the pulmonary arteries as measured by left pulmonary artery/right pulmonary artery diameter ratio.

A 2013 study by Amozgar et al. [100], compared the safety, efficacy, complications, and short-term outcomes of neonates who underwent PDA stenting to those who underwent surgical shunt (primarily BTS) from 2009 to 2011. They include in this study 18 patients in the PDA stent group, and 20 patients from a different center in the surgical shunt group. A PDA stent was successful in 15 of the 18 patients. This study showed no difference in pre- or post-procedure oxygen saturations, left pulmonary artery diameter, McGoon ratio, or Nakata index, but did show a larger right pulmonary artery diameter in the PDA stent group. The PDA stent cohort was also demonstrated to have a shorter mean hospital LOS compared to the surgical shunt cohort.

McMullan et al. [108] in a study realized in 2014, evaluated the safety and the durability of a PDA stent as an alternative to a BTS from 2002 to 2011 at a single center. The numbers in the PDA stent group were 13 patients, with a slightly larger BTS group (42 patients). There was no difference in survival difference, in time to second stage palliation or definitive repair, in postprocedural systemic oxygen saturation, in the number of interval reinterventions to maintain adequate PBF, and no significant difference in the time to reintervention in those that did undergo reinterventions, between the PDA stent and the BTS groups.

Another research made by Mallula et al. [109] compared the PDA stent to the surgical shunt in patients with pulmonary atresia with intact ventricular septum from 2006 to 2013. There were 13 patients who underwent PDA stent and 16 patients who underwent surgical shunt placement (10 central and 6 modified BTS). The baseline characteristics were similar between the 2 groups except for the PDA stent group being larger at the time of intervention. The PDA stent group had a shorter duration of mechanical ventilation, and lower median hospital lengths of stay. Acute complications and acute reinterventions were more common in the surgical shunt group; however, postdischarge reinterventions were more common in the PDA stent group, along with a shorter time to reintervention.

Although valuable, these four studies all have important bias and limitations due to their single center nature, small cohort sizes, and nonrandomized assignment to treatment strategies that could create confusion at the time to choose the best treatment for an individual case.

A multicenter large retrospective review of patients from four centers that comprise the Congenital Catheterization Research Collaboration from 2012 to 2015 made by Glatz et al. in 2018 [86], compared the outcomes between the BTS and the PDA stent when used as palliation for infants with ductal-dependent PBF. The primary outcome of this study was a composite of death or unplanned reintervention for cyanosis. The secondary outcomes included the individual components of the primary outcome, other reinterventions, intensive care unit (ICU) and hospital lengths of stay, diuretic use at hospital discharge, procedural complications, and pulmonary artery size at last follow-up prior to definitive surgical repair. This study includes 106 patients in the PDA stent group and 251 patients in the BTS group. There were differences in treatment strategies across centers, and some differences in patient-level factors. The PDA stent group included a larger number of PA/IVS (44%) patients, while the BTS group included more patients with pulmonary atresia in the setting of ventricular septal defect (39%). There were more patients with expected 2-ventricle physiology (60%) and antegrade PBF (61%) in the PDA stent group. There were no differences in gestational age, birth weight, genetic syndromes, or other comorbid conditions between the groups. There was no difference in pre-intervention ventricular function or atrioventricular valve regurgitation by echocardiography between the groups. The unadjusted analysis favored the PDA stent group with regard to the primary composite outcome of death or unplanned reintervention for cyanosis. There was less use of diuretics at discharge and shorter total hospital and intensive care unit lengths stay in the PDA stent group. The PDA stent group had larger and more symmetrical pulmonary arteries at later follow-up. The BTS group was favored in terms of other reinterventions (planned reinterventions or unplanned reinterventions to treat issues other than cyanosis), which occurred more frequently in the PDA stent cohort. Other outcomes examined in the unadjusted analysis showed a difference in the type

of definitive surgical repair with more patients in the BTS group undergoing a superior cavo-pulmonary connection. The PDA stent group was older at the time of subsequent repair with a longer time interval between initial palliation and the definitive repair, contradicting the prevailing assumption that PDA stenting is less durable than surgical shunt.

There was no difference in the need for pulmonary artery plasty at the time of definitive surgical repair, or in the rate of subsequent surgical or transcatheter pulmonary artery intervention.

Another large multicenter study is a 2018 United Kingdom (U.K.) study published by Bentham et al. [87]. This study included patients identified from the National Congenital Heart Disease Audit from 9 U.K. centers, < 30 days of age with ductal-dependent PBF from January 1, 2012 to December 31, 2015. The primary outcome was survival to next stage surgery (either palliation or repair). The secondary outcomes were survival to 30 days, to discharge, and to 1 year, and need for postprocedural extracorporeal membrane oxygenation (ECMO). There were 83 patients in the PDA stent group, and 171 patients in the BTS group. PDA stenting was successful in 70 of the 83 patients. There were no baseline differences between the groups in terms of age, weight, diagnosis, or comorbidities. The unadjusted analysis in this study showed the BTS group was intubated more commonly postprocedure, and had longer stay in the hospital and ICU, had longer mechanical ventilation, had lower oxygen saturation post-procedure, and had higher hemoglobin. There were more reinterventions before repair in the PDA stent group.

Despite, the inherent limitation to the existing body of literature notwithstanding, we can conclude that, although the BTS will continue to be an essential tool in the care of patients with ductal-dependent PBF, the use of a PDA stent appears to be an acceptable alternative palliative strategy in select patients, though prospective evaluation and longer term follow-up are necessary.

Right Ventricular Outflow Tract Stent

Stent implantation to the RVOT is other alternative treatment to surgically placed systemic-to-pulmonary shunt or stenting of ductus arteriosus, especially in patients with severe under development of pulmonary arteries. RVOT stenting was initially described by Gibbs et al. [110]; however, a larger, more contemporary series of 52 patients reported by Stumper et al. [111] demonstrated excellent outcomes. The authors concluded that RVOT stenting is a viable first-line treatment option for selected patients with severe RVOT obstruction as an alternative to systemic-to-pulmonary artery shunting procedures and for whom early complete repair carries significant additional risk or complexity.

Stenting of the RVOT secures the physiological direction of pulsating blood flow and enables symmetric growth of both branch arteries and the main pulmonary artery. This approach is more commonly utilized in patients with restenosis after surgical reconstruction of the RVOT [112-114]. It can also be performed as an additional treatment in unstable patients with hypoplastic pulmonary arteries, despite a surgically placed systemic-to-pulmonary shunt to augment pulmonary blood flow, increase oxygen saturation, and stabilize the patient's general condition [115].

The decision to indicate RVOT stenting is mainly based on severe associated comorbidities, which are thought to be negatively affected by mBTS hemodynamics (e.g., pre-palliative gut and brain pathologies, prematurity) or where the surgical intervention with potential necessity for cardiopulmonary bypass was thought to be high risk patient. Centers with excellent outcomes for early infant repair have identified low weight, severe cyanosis, pulmonary atresia rather than stenosis, hypoplastic pulmonary arteries, and noncardiac comorbidities as risk factors for higher mortality and reintervention. Risk factors for primary repair lie on a continuum with no sharp decision boundaries defined with respect to management. Because the experience in some center have increased and are getting very good outcome they prefer palliate with RVOT to patients with duct-dependent pulmonary blood flow, complex anatomy, and hypoplastic PAs are preferably palliated with RVOT stenting [115].

The RVOT stenting procedure is performed under general anesthesia and mechanical ventilation. Right femoral venous access is the preferred approach in children weighing more than 2.5 kg. In smaller children, a right internal jugular approach is frequently chosen. The patients received 50 IU heparin/kg and standard antibiotic prophylaxis. Selection of the size and the type of stent to be implanted is guided by the size of the patient, the dimension of the outflow tract, and the anticipated length of palliation. In small children in whom only short term (3–6 months) palliation is required, preference is to use a coronary stent; in larger patients, or in those with required medium to longer-term palliation, a bare metal peripheral vascular stent is selected. It is crucial in this type of intervention to place the stent below valve leaflets in order not to hinder its movement and induce insufficiency. Appropriate stent length and meticulous positioning are of outmost importance. If a stent is chosen with too small a diameter, there is the risk of stent instability and embolization, especially during balloon withdrawal. This risk can be reduced with placement of a long introducing sheath into the right ventricle outflow tract. Patent ductus arteriosus provides continuous pulmonary blood flow during stent introduction and balloon inflation and therefore gives more time for proper stent positioning without causing desaturation and bradycardia [116,117].

In patients ≤ 2 kg, the percutaneous method may cause hemodynamic instability secondary to the pass of the stiff coronary wire across the tricuspid valve and on order to avoid this complication Niall Linnane et al. are using a hybrid procedure that consist in a small suphxiphoid incision. A 5-F sheath is placed through the anterior surface of the right ventricle. Following sheath angiography is placed in the distal right pulmonary artery and subsequently the stent (s) is placed [118].

The reported complications rate of RVOT stenting is about 7% [111]. Potential problems include RVOT or pulmonary arteries rupture, stent embolization to pulmonary artery or aorta, stent fracture/compression and in-growth stenosis. However, stenting the RVOT as palliation leaves a virgin field for the surgeon during repair and does not have the failure rate of a BT shunt which is around 8%. The eventual repair takes slightly longer but with no clinical implications.

The reported mortality in infant more than 2.5 kg is around 1-2%, the oxygen saturation increased from a median of 70 (51-83%) to 93 (81-100%), post procedure time ven-

tilation, hospital stay, morbidity, and the pulmonary artery growth are significant lower compared with the results from the mBTS [111,117,119-121].

Despite significant advancements in stent design and profile to support stent de- livery in low birth weight (LBW) infants, this cohort of patients remains a challenge, with a recent report suggesting a procedural mortality of 12% associated with interventions in infants < 2 kg [14].

Whereas in 2005, RVOT stenting was carried out in desperate situations [122,123], these days several centers use it as their palliation of choice for infants with TOF who are not suitable for repair at the time of presentation [124].

CONCLUSIONS

With concerns about performing an early primary anatomic repair, particularly in a child with risk factors, the desire to identify the ideal palliative option is a pressing one. Characteristics of the ideal palliation in this situation might include the following: (1) providing a stable yet balanced source of pulmonary blood flow; (2) allowing for growth and development of the pulmonary arteries, ideally catch-up growth, because pulmonary arteries in this scenario are often hypoplastic; (3) providing sufficient durability to allow enough time to pass that the child grows, comorbidities (when present) subside, and the child reaches the time window when anatomic repair is deemed optimal; and (4) leaving behind no residue that might complicate the anatomic repair or increase the risk of subsequent reinterventions. At present, commonly used palliative options include surgical systemic–pulmonary shunt (typically a modified Blalock–Taussig shunt), ductus arteriosus stent, pulmonary balloon valvuloplasty, and right ventricular out- flow tract (RVOT) stent. To date, there has been considerable debate on how many features of the ideal palliative procedure each of these options holds.

FUNDING: None

DISCLOSURE: The authors have no conflicts of interest to disclose.

REFERENCES

1. Yuan SM, Jing H. Palliative procedures for congenital heart defects. Arch Cardiovasc Dis 2009;102:549-57. doi: 10.1016/j.acvd.2009.04.011.
2. Blalock A, Taussig HB. Landmark article May 19, 1945: The surgical treatment of malformations of the heart in which there is pulmonary stenosis or pulmonary atresia. By Alfred Blalock and Helen B. Taussig. JAMA 1984;251:2123-38. doi: 10.1001/jama.251.16.2123.
3. Potts WJ, Smith S, Gibson S. Anastomosis of the aorta to a pulmonary artery; certain types in congenital heart disease. J Am Med Assoc. 1946;132:627-31. doi: 10.1001/jama.1946.02870460017005.
4. Waterston DJ. Treatment of Fallot's tetralogy in children under 1 year of age. Rozhl Chir 1962; 41:181-3.
5. Cooley DA, Hallman GL. Intrapericardial aortic-right pulmonary arterial anastomosis. Surg Gynecol Obstet 1966;122:1084-6.
6. Gazzaniga AB, Elliott MP, Sperling DR, et al. Microporous expanded polytetrafluoroethylene arterial prosthesis for construction of aortopulmonary shunts: experimental and clinical results. Ann Thorac Surg 1976;21:322-7. doi: 10.1016/s0003-4975(10)64320-2.
7. McKenzie ED, Khan MS, Samayoa AX, et al. The Blalock-Taussig shunt revisited: a contemporary experience. J Am Coll Surg. 2013;216:699-704; discussion 704-6. doi: 10.1016/j.jamcollsurg.2012.12.027.
8. Mavroudis C, Backer CL. Palliative operations. In: Mavroudis C, Backer CL (eds). Pediatric Cardiac Surgery. 4th ed. Philadelphia: Mosby, 2013, 160–70. ISBN: 978-1-405-19652-9.
9. Geiss D, Williams WG, Lindsay WK, Rowe RD. Upper extremity gangrene: a complication of subclavian artery division. Ann Thorac Surg 1980;30:487-9. doi: 10.1016/s0003-4975(10)61302-1.
10. de Leval MR, McKay R, Jones M, Stark J, Macartney FJ. Modified Blalock-Taussig shunt. Use of subclavian artery orifice as flow regulator in prosthetic systemic-pulmonary artery shunts. J Thorac Cardiovasc Surg 1981;81:112–9. doi: 10.1016/S0022-5223(19)37668-8.
11. Williams JA, Bansal AK, Kim BJ, et al. Two thousand Blalock-Taussig shunts: a six-decade experience. Ann Thorac Surg 2007;84:2070-5; discussion 2070-5. doi: 10.1016/j.athoracsur.2007.06.067.
12. Eghtesady P, Ghanamah SM. Fallot: Palliation with BT Shunt. In: Lacour-Gayet F, Bove EL, Hraska V, Morell VO, Spray TL (ed). Surgery of Conotruncal Anomalies. Switzerland Springer, 2016; 189-20. ISBN: 978-3-319-23057-3. doi: 10.1007/978-3-319-23057-3.
13. Odim J, Portzky M, Zurakowski D, et al. Sternotomy approach for the modified Blalock-Taussig shunt. Circulation 1995;92: II256–61.
14. Shauq A, Agarwal V, Karunaratne A, et al. Surgical approaches to the Blalock shunt: does the approach matter? Heart Lung Circ 2010; 19:460–4.
15. Talwar S, Kumar MV, Muthukkumaran S, Airan B. Is sternotomy superior to thoracotomy for modified Blalock-Taussig shunt? Interact Cardiovasc Thorac Surg. 2014;18:371-5. doi:10.1093/icvts/ivt513.
16. Dirks V, Prêtre R, Knirsch W, etal. Modified Blalock Taussig shunt: a not-so-simple palliative procedure. Eur J Cardiothorac Surg 2013;44:1096-102. doi: 10.1093/ejcts/ezt172.
17. Gedicke M, Morgan G, Parry A, Martin R, Tulloh R. Risk factors for acute shunt blockage in children after modified Blalock-Taussig shunt operations. Heart Vessels 2010; 25:405–9. doi: 10.1007/s00380-009-1219-1.
18. Petrucci O, O'Brien SM, Jacobs ML, Jacobs JP, Manning PB, Eghtesady P. Risk factors for mortality and morbidity after the neonatal Blalock-Taussig shunt procedure. Ann Thorac Surg 2011;92:642-51; discussion 651-2. doi: 10.1016/j.athoracsur.2011.02.030.
19. McKenzie ED, Khan MS, Samayoa AX, et al. The Blalock-Taussig shunt revisited: a contemporary experience. J Am Coll Surg. 2013;216:699-704; discussion 704-6. doi: 10.1016/j.jamcollsurg.2012.12.027.
20. Bove T, Vandekerckhove K, Panzer J, De Groote K, De Wolf D, François K. Disease-specific outcome analysis of palliation with the modified Blalock-Taussig shunt. World J Pediatr Congenit Heart Surg 2015;6:67–74. doi: 10.1177/2150135114558690.
21. Lamberti JJ, Carlisle J, Waldman JD, et al. Systemic-pulmonary shunts in infants and children. Early and late results. J Thorac Cardiovasc Surg 1984;88:76–81.
22. Gold JP, Violaris K, Engle MA, Klein AA, Ehlers KH, Lang SJ, Levin AR, Moran F, O'Loughlin JE, Snyder MS, et al. A five-year clinical experience with 112 Blalock-Taussig shunts. J Card Surg. 1993;8:9-17. doi: 10.1111/j.1540-8191.1993.tb00571.x.
23. Gladman G, McCrindle BW, Williams WG, Freedom RM, Benson LN. The modified Blalock-Taussig shunt: clinical impact and morbidity in Fallot's tetralogy in the current era. J Thorac Cardiovasc Surg 1997;114:25-30. doi: 10.1016/S0022-5223(97)70113-2.
24. Alkhulaifi AM, Lacour-Gayet F, Serraf A, Belli E, Planché C. Systemic pulmonary shunts in neonates: early clinical outcome and choice of surgical approach. Ann Thorac Surg 2000;69:1499-504. doi: 10.1016/s0003-4975(00)01078-x.
25. Williams JA, Bansal AK, Kim BJ, et al. Two thousand Blalock-Taussig shunts: a six-decade experience. Ann Thorac Surg 2007;84:2070-5; discussion 2070-5. doi: 10.1016/j.athoracsur.2007.06.067.
26. Vitanova K, Leopold C, von Ohain JP, Wolf C, Beran E, Lange R, Cleuziou J. Reasons for Failure of Systemic-to-Pulmonary Artery Shunts in Neonates. Thorac Cardiovasc Surg 2019;67:2-7. doi: 10.1055/s-0037-1621706.
27. Vitanova K, Leopold C, Pabst von Ohain J, Wolf C, Beran E, Lange R, Cleuziou J. Risk Factors for Failure of Systemic-to-Pulmonary Artery Shunts in Biventricular Circulation. Pediatr Cardiol 2018;39:1323-9. doi: 10.1007/s00246-018-1898-4.
28. Tsai KT, Chang CH, Lin PJ, et al. Modified Blalock-Taussing shunt: statistical analysis of potential factor influencing shunt outcome. J Cardiovasc Surg 1996; 37.149-52.
29. Tamisier D, Vouhé PR, Vernant F, Lecá F, Massot C, Neveux JY. Modified Blalock-Taussig shunts: results in infants less than 3 months of age. Ann Thorac Surg. 1990;49:797-801. doi: 10.1016/0003-4975(90)90026-3.
30. Jialal I, Devaraj S, Venugopal SK. C-reactive protein: risk marker or mediator in atherothrombosis? Hypertension 2004;44:6-11. doi: 10.1161/01.HYP.0000130484.20501.df.
31. Tracy RP. Thrombin, inflammation, and cardiovascular disease: an epidemiologic perspective. Chest. 2003;124(3 Suppl):49S-57S. doi: 10.1378/chest.124.3_sup-

pl.49s.
32. Eisenhardt SU, Habersberger J, Peter K. Monomeric c-reactive protein generation on activated platelets: the missing link between inflammation and atherothrombotic risk. Trends Cardiovasc Med 2009; 19:232–7. doi: 10.1016/j.tcm.2010.02.002.
33. Lippi G, Favaloro EJ, Montagnana M, Franchini M. C-reactive protein and venous thromboembolism: causal or causal association? Clin Chem Lab Med 2010;48: 1673–701. doi: 10.1515/CCLM.2010.335.
34. Cholette JM, Rubenstein JS, Alfieris GM, et al. Elevated risk of thrombosis in neonates undergoing initial palliative cardiac surgery. Ann Thorac Surg 2007; 84:1320–5. doi: 10.1016/j.athoracsur.2007.05.026.
35. Kaur R, Bhurtel D, Bielefeld MR, et al. Cryopreserved Saphenous vein compared with PTFE graft for use as modified Blalock-Taussing or Central Shunt in cyanotic congenital heart disease. World J Pediatr Congenit Heart Surg 2018;9:509-12. doi: doi: 10.1177/2150135118776616.
36. Bishnoi, A.K., Talwar, S., Choudhary, S.K. et al. Homograft saphenous vein versus polytetrafluoroethylene graft for modified Blalock -Taussig shunt. Indian J Thorac Cardiovasc Surg 2008;24:227–32. doi: 10.1007/s12055-008-0051-z.
37. Al Jubair KA, Al Fagih MR, Al Jarallah AS, Al Yousef S, Ali Khan MA, Ashmeg A, Al Faraidi Y, Sawyer W. Results of 546 Blalock-Taussig shunts performed in 478 patients. Cardiol Young 1998;8:486-90. doi: 10.1017/s1047951100007150.
38. Li JS, Yow E, Berezny KY, Rhodes JF, et al. Clinical outcomes of palliative surgery including a systemic-to-pulmonary artery shunt in infants with cyanotic congenital heart disease: does aspirin make a difference? Circulation. 2007;116:293-7. doi: 10.1161/CIRCULATIONAHA.106.652172.
39. Sahoo TK, Chauhan S, Sahu M, Bisoi A, Kiran U. Effects of hemodilution on outcome after modified Blalock-Taussig shunt operation in children with cyanotic congenital heart disease. J Cardiothorac Vasc Anesth 2007; 21:179–83. doi: 10.1053/j.jvca.2006.01.029.
40. Watanabe M, Aoki M, Fujiwara T. Thrombotic occlusion of Blalock- Taussig shunt in a patient with unnoticed protein C deficiency. Gen Thorac Cardiovasc Surg 2008; 56:544–6. doi: 10.1007/s11748-008-0295-y.
41. Deally C, Hancock BJ, Giddins N, Hawkins L, Odim J. Primary antiphospholipid syndrome: a cause of catastrophic shunt thrombosis in the newborn. J Cardiovasc Surg 1999; 40:261–4.
42. Wells WJ, Yu RJ, Batra AS, Monforte H, Sintek C, Starnes VA. Obstruction in modified Blalock shunts: a quantitative analysis with clinical correlation. Ann Thorac Surg 2005; 79:2072–6. doi: 10.1016/j.athoracsur.2004.12.050.
43. Lee C. Surgical management of chronic pulmonary regurgitation after relief of right ventricular outflow tract obstruction. Korean Circ J 2012;42:1-7. doi: 10.4070/kcj.2012.42.1.1.
44. Arenz C, Laumeier A, Lütter S, et al. Is there any need for a shunt in the treatment of tetralogy of Fallot with one source of pulmonary blood flow? Eur J Cardiothorac Surg 2013;44:648-54. doi: 10.1093/ejcts/ezt124.
45. Pacileo G, Limongelli G, Verrengia M, et al. Impact of pulmonary regurgitation and age at surgical repair on textural and functional right ventricular myocardial properties in patients with tetralogy of Fallot. Ital Heart J 2005; 6:745–50.
46. Kantorova A, Zbieranek K, Sauer H, Lilje C, Haun C, Hraska V. Primary early correction of tetralogy of Fallot irrespective of age. Cardiol Young 2008;18:153-7. doi: 10.1017/S1047951108001960.
47. Rao V, Kadletz M, Hornberger LK, Freedom RM, Black MD. Preservation of the pulmonary valve complex in Tetralogy of Fallot: how small is too small? Ann Thorac Surg. 2000;69:176-9; discussion 179-80. doi: 10.1016/s0003-4975(99)01152-2.
48. Voges I, Fischer G, Scheewe J, et al. Restrictive enlargement of the pulmonary annulus at surgical repair of tetralogy of Fallot: 10-year experience with a uniform surgical strategy. Eur J Cardiothorac Surg 2008;34:1041–5. doi: 10.1016/j.ejcts.2008.07.048.
49. Vida VL, Guariento A, Castaldi B, et al. Evolving strategies for preserving the pulmonary valve during early repair of tetralogy of Fallot: mid-term results. J Thorac Cardiovasc Surg 2014;147:687-94; discussion 694-6. doi: 10.1016/j.jtcvs.2013.10.029.
50. Spiewak M, Biernacka EK, Małek ŁA, et al. Right ventricular outflow tract obstruction as a confounding factor in the assessment of the impact of pulmonary regurgitation on the right ventricular size and function in patients after repair of tetralogy of Fallot. J Magn Reson Imaging 2011;33:1040-6. doi: 10.1002/jmri.22532.
51. Yuan SM, Jing H. Palliative procedures for congenital heart defects. Arch Cardiovasc Dis. 2009;102(6-7):549-57. doi: 10.1016/j.acvd.2009.04.011.
52. Milliken JS, Puga FJ, Davidson GK, Schaff HV, Julsrud PR, Mair DD. Staged surgical repair of pulmonary atresia, ventricular septal defect and hypoplastic confluent pulmonary arteries. J Thorac Cardiovasc Surg 1986; 91:818-25.
53. Sullivan ID, Wren C, Stark J, de Leval MR, Macartney FJ, Deanfield JE. Surgical unifocalization in pulmonary atresia and ventricular septal defect. A realistic goal? Circulation 1988;78(5 Pt 2):III5-13.
54. Gill CC, Moodie DS, McGoon DC. Staged surgical management of pulmonary atresia with diminutive pulmonary arteries. J Thorac Cardiovasc Surg 1977;73:36-42.
55. Kirklin JW, Bargeron LM, Pacifico AD. The enlargement of small pulmonary arteries by preliminary palliative operations. Circulation 1977; 56:612-7.
56. Metras D, Chetaille P, Kreitmann B, Fraisse A, Ghez O, Riberi A. Pulmonary atresia with ventricular septal defect, extremely hypoplastic pulmonary arteries, major aorto- pulmonary collaterals. Eur J Cardiothorac Surg. 2001;20:590-6; discussion 596-7. doi: 10.1016/s1010-7940(01)00855-7.
57. Pagani FD, Cheatham JP, Beekman RH 3rd, Lloyd TR, Mosca RS, Bove EL. The management of tetralogy of Fallot with pulmonary atresia and diminutive pulmonary arteries. J Thorac Cardiovasc Surg 1995; 110:1521–33. doi: 10.1016/S0022-5223(95)70076-5.
58. Rodefeld MD, Reddy VM, Thompson LD, et al. Surgical creation of aortopulmonary window in selected patients with pulmonary atresia with poorly developed aortopulmonary collaterals and hypoplastic pulmonary arteries. J Thorac Cardiovasc Surg 2002; 123:1147–54. doi: 10.1067/mtc.2002.121685.
59. Watterson KG, Wilkinson JL, Karl TR, Mee RB. Very small pulmonary arteries: central end-to-side shunt. Ann Thorac Surg 1991; 52:1132–7. doi: 10.1016/0003-4975(91)91294-6.
60. Duncan BW, Mee RB, Prieto LR, Rosenthal GL, Mesia CI, Qureshi A, Tucker OP, Rhodes JF, Latson LA. Staged repair of tetralogy of Fallot with pulmonary atresia and major aortopulmonary collateral arteries. J Thorac Cardiovasc Surg 2003;126:694-702. doi: 10.1016/s0022-5223(03)00700-1.
61. Liava'a M, Brizard CP, Konstantinov IE, Robertson T, Cheung MM, Weintraub R, d'Udekem Y. Pulmonary atresia, ventricular septal defect, and major aortopulmonary collaterals: neonatal pulmonary artery rehabilitation without unifocalization. Ann Thorac Surg 2012;93:185-91. doi: 10.1016/j.athoracsur.2011.08.082.
62. Mainwaring RD, Reddy M, Perry SB et al. Late Outcomes in Patients Undergoing Aortopulmonary Window for Pulmonary Atresia/Stenosis and Major Aortopulmonary Collaterals. Ann Thorac Surg 2012; 94:842–9. doi: 10.1016/j.athoracsur.2012.03.061.
63. d'Udekem Y,Alphonso N, Nørgaard MA et al. Pulmonary atresia with ventricular septal defects and major aortopulmonary collateral arteries: Unifocalization brings no long-term benefits. J Thorac Cardiovasc Surg 2005; 130:1496-502. 10.1016/j.jtcvs.2005.07.034.
64. Gates RN, Laks H, Johnson K. Side-to-side aorto-Gore-Tex central shunt. Ann Thorac Surg 1998;65:515-6. doi: 10.1016/s0003-4975(97)01126-0.
65. Kim H, Sung SC, Choi KH, Lee HD, Ban GH, Chang YH. A central shunt to rehabilitate diminutive pulmonary arteries in patients with pulmonary atresia with ventricular septal defect. J Thorac Cardiovasc Surg 2015;149:515-20. doi: 10.1016/j.jtcvs.2014.10.033.
66. Gerelli S, van Steenberghe M, Murtuza B, et al. Neonatal right ventricle to pulmonary connection as a palliative procedure for pulmonary atresia with ventricular septal defect or severe tetralogy of Fallot. Eur J Cardiothorac Surg 2014;45:278-88; discussion 288. doi: 10.1093/ejcts/ezt401.
67. Barbero-Marcial M, Riso A, Atik E, Jatene AA. Technique for correction of truncus arteriosus types I and II without extracardiac conduits. J Thorac Cardiovasc Surg 1990; 99:364–9.
68. Rome JJ, Mayer JE, Castaneda AR, Lock JE. Tetralogy of Fallot with pulmonary atresia. Rehabilitation of diminutive pulmonary arteries. Circulation. 1993 Oct;88(4 Pt 1):1691-8. doi: 10.1161/01.cir.88.4.1691.
69. Piehler JM, Danielson GK, McGoon DC, Wallace RB, Fulton RE, Mair DD. Management of pulmonary atresia with ventricular septal defect and hypoplastic pulmonary arteries by right ventricular outflow construction. J Thorac Cardiovasc Surg 1980; 80:552–67.
70. Van Arsdell G, Yun TJ. An apology for primary repair of tetralogy of Fallot. Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu 2005:128-31. doi: 10.1053/j.pcsu.2005.01.016.
71. Malhotra SP, Hanley FL. Surgical management of pulmonary atresia with ventricular septal defect and major aortopulmonary collaterals: a protocol-based approach. Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu 2009:145-51. doi: 10.1053/j.pcsu.2009.01.017.
72. Cho JM, Puga FJ, Danielson GK, et al. Early and long-term results of the surgical treatment of tetralogy of Fallot with pulmonary atresia, with or without major aortopulmonary collateral arteries. J Thorac Cardiovasc Surg 2002;124:70-81. doi: 10.1067/mtc.2002.120711.
73. Farouk A, Zahka K, Siwik E, Erenberg F, Al-Khatib Y, Golden A, Karimi M, Uddin M, Hennein HA. Individualized approach to the surgical treatment of tetralogy of Fallot with pulmonary atresia. Cardiol Young 2009;19:76-85. doi: 10.1017/S1047951108003430.
74. Kim H, Sung SC, Chang YH, Jung W, Lee HD, Park JA, Huh U. Outcome of Staged Repair of Tetralogy of Fallot with Pulmonary Atresia and a Ductus-dependent Pulmonary Circulation: Should Primary Repair Be Considered? Korean J Thorac Cardiovasc Surg 2011;44:392-8. doi: 10.5090/kjtcs.2011.44.6.392.
75. O'Connor MJ, Ravishankar C, Ballweg JA, Gillespie MJ, Gaynor JW, Tabbutt S, Dominguez TE. Early systemic-to-pulmonary artery shunt intervention in neonates with congenital heart disease. J Thorac Cardiovasc Surg. 2011;142:106-12. doi: 10.1016/j.jtcvs.2010.10.033.
76. Virzi L, Pemberton V, Ohye RG, Tabbutt S, Lu M, Atz TC et al. Reporting adverse events in a surgical trial for complex congenital heart disease: the Pediatric Heart Network experience. J Thorac Cardiovasc Surg 2011;142:531–7. doi: 10.1016/j.jtcvs.2010.11.052.

77. Gibbs JL, Rothman MT, Rees MR, Parsons JM, Blackburn ME, Ruiz CE. Stenting of the arterial duct: a new approach to palliation for pulmonary atresia. Br Heart J 1992;67:240-5. doi: 10.1136/hrt.67.3.240.
78. Rehman R, Marhisham MC, Alwi M. Stenting the complex patent ductus arteriosus in tetralogy of Fallot with pulmonary atresia: challenges and outcomes. Future Cardiol 2018;14:55-73. doi: 10.2217/fca-2017-0053.
79. Santoro G, Gaio G, Giugno L, et al. Ten-years, single-center experience with arterial duct stenting in duct-dependent pulmonary circulation: early results, learning-curve changes, and mid-term outcome. Catheter Cardiovasc Interv 2015;86:249-57. doi: 10.1002/ccd.25949.
80. Udink Ten Cate FE, Sreeram N, Hamza H, Agha H, Rosenthal E, Qureshi SA. Stenting the arterial duct in neonates and infants with congenital heart disease and duct-dependent pulmonary blood flow: a multicenter experience of an evolving therapy over 18 years. Catheter Cardiovasc Interv 2013;82:E233-43. doi: 10.1002/ccd.24878.
81. Alwi M. Stenting the patent ductus arteriosus in duct-dependent pulmonary circulation: techniques, complications and follow-up. Future Cardiol 2012;8:237-50. doi: 10.2217/fca.12.4.
82. Santoro G, Gaio G, Palladino MT, et al. Arterial duct stenting: Do we still need surgical shunt in congenital heart malformations with duct-dependent pulmonary circulation? J Cardiovasc Med (Hagerstown) 2010;11:852-7. doi: 10.2459/JCM.0b013e32833a070d.
83. Santoro G, Gaio G, Palladino MT, et al. Stenting of the arterial duct in newborns with duct-dependent pulmonary circulation. Heart 2008;94:925-9. doi: 10.1136/hrt.2007.123000.
84. Alwi M. Stenting the ductus arteriosus: Case selection, technique and possible complications. Ann Pediatr Cardiol 2008;1:38-45. doi: 10.4103/0974-2069.41054.
85. Alwi M, Choo KK, Latiff HA, Kandavello G, Samion H, Mulyadi MD. Initial results and medium-term follow-up of stent implantation of patent ductus arteriosus in duct-dependent pulmonary circulation. J Am Coll Cardiol 2004;44:438-45. doi: 10.1016/j.jacc.2004.03.066.
86. Glatz AC, Petit CJ, Goldstein BH, et al. A comparison between patent ductus arteriosus stent and modified Blalock-Taussig shunt as palliation for infants with ductal-dependent pulmonary blood flow: insights from the congenital catheterization research collaborative. Circulation 2018;137:581-8. doi: doi: 10.1161/CIRCULATIONAHA.117.029987.
87. Bentham JR, Zava NK, Harrison WJ, et al. Duct stenting versus modified Blalock Taussig shunt in neonates with duct-dependent pulmonary blood flow: associations with clinical outcomes in a multicenter national study. Circulation. 2018;137:581-588. doi: 10.1161/CIRCULATIONAHA.117.028972.
88. Glatz AC, Shah SS, McCarthy AL, Geisser D, Daniels K, Xie D, Hanna BD, Grundmeier RW, Gillespie MJ, Rome JJ. Prevalence of and risk factors for acute occlusive arterial injury following pediatric cardiac catheterization: a large single-center cohort study. Catheter Cardiovasc Interv 2013;82:454-62. doi: 10.1002/ccd.24737.
89. Polat TB. Stenting the vertical ductus arteriosus via axillary artery access using "wire-target" technique. Congenital Heart Dis 2017;12:800-7. doi: 10.1111/chd.12512.
90. Justino H, Petit CJ. Percutaneous Common Carotid Artery Access for Pediatric Interventional Cardiac Catheterization. Circ Cardiovasc Interv 2016;9:e003003. doi: 10.1161/CIRCINTERVENTIONS.115.003003.
91. Choudhry S, Balzer D, Murphy J, Nicolas R, Shahanavaz S. Percutaneous carotid artery access in infants < 3 months of age. Catheter Cardiovasc Interv: Off J Soc Cardiac Angiogra Interv 2016;87:757-61. doi: 10.1002/ccd.26310.
92. Schranz D, Michel-Behnke I. Axillary artery access for cardiac interventions in newborns. Ann Pediat Cardiol. 2008;1:126-30. doi: 10.4103/0974-2069.43878.
93. Alwi M. Patent ductus arteriosus stenting-problems, complications and technical consideration. Neonatol Today 2011; 6:3-5. doi: 10.4103/0974-2069.41054.
94. Michel-Behnke I, Akintuerk H, Thul J, Bauer J, Hagel KJ, Schranz D. Stent implantation in the ductus arteriosus for pulmonary blood supply in congenital heart disease. Catheter Cardiovasc Interv 2004;61:242-52. doi: 10.1002/ccd.10766.
95. Buys DG, Brown SC, Greig C. Stenting the arterial duct: practical aspects and review of outcomes. SA Heart J 2013; 13:514-9. DOI: https://doi.org/10.24170/10-3-1791.
96. Gewillig M, Boshoff DE, Dens J, Mertens L, Benson LN. Stenting the neonatal arterial duct in duct-dependent pulmonary circulation: new techniques, better results. J Am Coll Cardiol 2004;43:107-12. doi: 10.1016/j.jacc.2003.08.029.
97. Matter M, Almarsafawey H, Hafez M, Attia G, Abuelkheir MM. Patent ductus arteriosus stenting in complex congenital heart disease: early and midterm results for a single-center experience at children hospital, Mansoura, Egypt. Pediatr Cardiol. 2013;34:1100-6. doi: 10.1007/s00246-012-0608-x.
98. Schneider M, Zartner P, Sidiropoulos A, Konertz W, Hausdorf G. Stent implantation of the arterial duct in newborns with duct-dependent circulation. Eur Heart J 1998;19:1401-9. doi: 10.1053/euhj.1998.0977.
99. Hussain A, Al-Zharani S, Muhammed AA, Al-Ata J, Galal OM. Midterm outcome of stent dilatation of patent ductus arteriosus in ductal-dependent pulmonary circulation. Congenit Heart Dis 2008:3:241-9. doi: 10.1111/j.1747-0803.2008.00197.x.
100. Amoozgar H, Cheriki S, Borzoee M, Ajami G, Soltani M, Ahmadipour M, Peiravian F, Amirghofran A. Short-term result of ductus arteriosus stent implantation compared with surgically created shunts. Pediatr Cardiol 2012;33:1288-94. doi: 10.1007/s00246-012-0304-x.
101. Onalan MA, Odemis E, Saygi M, et al. Early and midterm results of ductal stent implantation in neonates with ductal-dependent pulmonary circulation: a single-centre experience. Cardiol Young 2020;30:1772-82. doi: 10.1017/S104795112000267X.
102. Feltes TF, Bacha E, Beekman RH 3rd, et al. Indications for cardiac catheterization and intervention in pediatric cardiac disease: a scientific statement from the American Heart Association. Circulation. 2011;123:2607-52. doi: 10.1161/CIR.0b013e31821b1f10.
103. Qureshi AM, Goldstein BH, Glatz AC, et al. Classification scheme for ductal morphology in cyanotic patients with ductal dependent pulmonary blood flow and association with outcomes of patent ductus arteriosus stenting. Catheter Cardiovasc Interv 2019; 93:933-43. doi: 10.1002/ccd.28125.
104. Santoro G, Gaio G, Capozzi G, Giugno L, Palladino MT, Capogrosso C, D'Aiello AF, Caianiello G, Russo MG. Fate of Hypoplastic Pulmonary Arteries After Arterial Duct Stenting in Congenital Heart Disease with Duct-Dependent Pulmonary Circulation. JACC Cardiovasc Interv 2015;8:1626-32. doi: 10.1016/j.jcin.2015.05.027.
105. Sivakumar K, Bhagyavathy A, Coelho R, Satish R, Krishnan P. Longevity of neonatal ductal stenting for congenital heart diseases with duct-dependent pulmonary circulation. Congenit Heart Dis 2012;7:526-33. doi: 10.1111/j.1747-0803.2012.00657.x.
106. Vida VL, Speggiorin S, Maschietto N, et al. Cardiac operations after patent ductus arteriosus stenting in duct-dependent pulmonary circulation. Ann Thorac Surg. 2010;90:605-9. doi: 10.1016/j.athoracsur.2010.04.007.
107. Santoro G, Capozzi G, Caianiello G, et al. Pulmonary artery growth after palliation of congenital heart disease with duct-dependent pulmonary circulation: arterial duct stenting versus surgical shunt. J Am Coll Cardiol 2009;54:2180-6. doi: 10.1016/j.jacc.2009.07.043.
108. McMullan DM, Permut LC, Jones TK, Johnston TA, Rubio AE. Modified Blalock-Taussig shunt versus ductal stenting for palliation of cardiac lesions with inadequate pulmonary blood flow. J Thorac Cardiovasc Surg 2014;147:397-401. doi: 10.1016/j.jtcvs.2013.07.052
109. Mallula K, Vaughn G, El-Said H, Lamberti JJ, Moore JW. Comparison of ductal stenting versus surgical shunts for palliation of patients with pulmonary atresia and intact ventricular septum. Catheter Cardiovasc Interv 2015;85:1196-202. doi: doi: 10.1002/ccd.25870.
110. Gibbs JL, Uzun O, Blackburn ME, Parsons JM, Dickinson DF. Right ventricular outflow stent implantation: an alternative to palliative surgical relief of infundibular pulmonary stenosis. Heart 1997; 77:176 - 9. doi: 10.1136/hrt.77.2.176.
111. Stumper O, Ramchandani B, Noonan P, et al. Stenting of the right ventricular outflow tract. Heart 2013;99:1603-8. doi: 10.1136/heartjnl-2013-304155.
112. Shenoy AR, Padmakumar P, Subashchandra V. Right ventricular outflow tract stenting in Tetralogy of Fallot with restrictive ventricular septal defect. J Invasive Cardiol 2006;18(1):E59-60.
113. Dryzek P, Mazurek-Kula A, Moszura T, Sysa A. Right ventricle outflow tract stenting as a method of palliative treatment of severe tetralogy of Fallot. Cardiol J 2008;15:376-9.
114. Zeidenweber CM, Kim DW, Vincent RN. Right ventricular outflow tract and pulmonary artery stents in children under 18 months of age. Catheter Cardiovasc Interv 2007;69:23-7. doi: 10.1002/ccd.20918.
115. Barron DJ, Ramchandani B, Murala J, et al. Surgery following primary right ventricular outflow tract stenting for Fallot's tetralogy and variants: rehabilitation of small pulmonary arteries. Eur J Cardiothorac Surg 2013;44:656-62. doi: 10.1093/ejcts/ezt188.
116. Bang S, Ko HK, Yu JJ, et al. Right ventricular outflow tract stenting in a low birth weight infant born with tetralogy of Fallot and prostaglandin e1 dependency. Korean Circ J 2011;41:744-6. doi: 10.4070/kcj.2011.41.12.744.
117. Dohlen G, Chaturvedi RR, Benson LN, et al. Stenting of the right ventricular outflow tract in the symptomatic infant with tetralogy of Fallot. Heart 2009;95:142-7. doi: 10.1136/hrt.2007.135723.
118. Linnane N, Walsh KP, McGuinness J, Kenny D. Evolution of Approach to Right Ventricular Outflow Tract Stenting in Infants ≤ 2Kgs. Journal of Structural Heart Disease 2018;4(2):50-5. 0.12945/j.jshd.2018.045.17.
119. Castleberry CD, Gudausky TM, Berger S, Tweddell JS, Pelech AN. Stenting of the right ventricular outflow tract in the high-risk infant with cyanotic teratology of Fallot. Pediatr Cardiol 2014;35:423-30. doi: 10.1007/s00246-013-0796-z.
120. Quandt D, Ramchandani B, Stickley J, et al. Stenting of the Right Ventricular Outflow Tract Promotes Better Pulmonary Arterial Growth Compared with Modified Blalock-Taussig Shunt Palliation in Tetralogy of Fallot-Type Lesions. JACC Cardiovasc Interv 2017;10:1774-84. doi: 10.1016/j.jcin.2017.06.023.
121. Backes CH, Cua C, Kreutzer J, et al. Low weight as an independent risk factor for adverse events during cardiac catheterization of infants. Catheter Cardiovasc Interv 2013;82:786-94. doi: 10.1002/ccd.24726.

122. Hosking MC, Benson LN, Nakanishi T, Burrows PE, Williams WG, Freedom RM. Intravascular stent prosthesis for right ventricular outflow obstruction. J Am Coll Cardiol 1992;20:373-80. doi: 10.1016/0735-1097(92)90105-v.
123. Gibbs JL, Uzun O, Blackburn ME, Parsons JM, Dickinson DF. Right ventricular outflow stent implantation: an alternative to palliative surgical relief of infundibular pulmonary stenosis. Heart 1997;77(2):176-9. doi: 10.1136/hrt.77.2.176.
124. Bertram H, Emmel M, Ewert P, et al; Investigators Of The Working Group Interventional Cardiology Of The German Society Of Pediatric Cardiology. Stenting of Native Right Ventricular Outflow Tract Obstructions in Symptomatic Infants. J Interv Cardiol 2015;28:279-87. doi: 10.1111/joic.12198.