ORIGINAL CONTRIBUTION

Neonatal Blalock-Taussig Shunt: Technical Aspects and Postoperative Management

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ABSTRACT

A systemic-pulmonary artery shunt in neonates with decreased pulmonary blood flow is technically demanding. We describe our surgical technique, postoperative management, and results in 19 neonates who underwent a modified Blalock-Taussig shunt between April 2003 and March 2006. Prostaglandin infusion was required in 8 patients who were critically cyanosed, and 5 were on inotropic support preoperatively. A 3.5 or 4.0-mm polytetrafluoroethylene graft was anastomosed with 8/0 polypropylene suture. Postoperatively, systemic pressure was kept slightly higher than normal, and heparin was started early. One patient required revision of the shunt, and one was reexplored for bleeding. There were 2 hospital deaths (mortality, 11%) in patients with preoperative hemodynamic instability. The mean follow-up period was 12 months, with no late postoperative shunt blockage or death. Meticulous surgical technique and judicious use of heparin and inotropic agents improved the outcome and reduced the incidence of shunt blockage and reexploration for bleeding.

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INTRODUCTION

The Blalock-Taussig (BT) shunt is an excellent and widely accepted form of palliation for neonates with congenital cyanotic heart disease and decreased pulmonary blood flow.[1,2] It was first described by Blalock and Taussig in 1945 and modified by de Leval and colleagues[3] with a polytetrafluoroethylene (PTFE) graft interposed between the subclavian artery and the ipsilateral pulmonary artery (PA). As the subclavian artery orifice of the BT shunt acts as a flow regulator, it provides controlled blood flow to the lungs. The neonatal age group has been recognized as having increased risk of morbidity and mortality due to a modified BT shunt.[2,4,5] Constructing a BT shunt in neonates is technically demanding, and specialized postoperative care is important to ensure a good outcome. We describe our technical modification of the anastomosis and postoperative management of neonates with a modified BT shunt.

MATERIALS AND METHODS

From April 2003 to March 2006, 147 patients underwent construction of a modified BT shunt for congenital heart disease with decreased pulmonary blood flow, of which 19 (13%) were neonates. The mean age of the neonates was 12 days (range, 2–30 days). There were 10 boys and 9 girls. The mean weight was 3.24 ± 0.86 kg (range, 2.1–3.8 kg). All babies were severely cyanosed and admitted to the neonatal intensive care unit (ICU) preoperatively. The mean O_2 saturation on pulse oxymetry was 62% (range, 48%–73%). Routine preoperative echocardiographic evaluation of intracardiac anatomy was carried out in all patients in the ICU, including left and right PA size, confluence, main PA, orientation of the aortic arch and its branching pattern, presence of a ductus arteriosus and its site of insertion. The mean right PA size was 3.9 ± 0.69 mm (range, 3.3–4.1 mm) and mean left PA size was 3.7 ± 0.78 mm (range, 3.1–3.8 mm). A left-sided aortic arch was present in 16 neonates, and

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3 had a right-sided arch. All patients were stabilized in the ICU by maintaining adequate hydration, preventing hypothermia and hypoxia, and correcting metabolic acidosis, if any. After assessment by echocardiography, 8 patients who were critically cyanosed required prostaglandin E1 infusion (0.03–0.05 $\mu g \cdot kg^{-1} \cdot min^{-1}$) for preoperative stabilization. Three patients arrived on prostaglandin infusion from other units, and 5 who were in shock with poor hemodynamics and perfusion required preoperative inotropic stabilization. The intracardiac anatomy is shown in Table 1.

Table 1. **Intracardiac Anatomy in 19 Neonates**

Anatomy	No. of Patients
Tetralogy of Fallot with pulmonary atresia	11
Tricuspid atresia and pulmonary atresia	4
Pulmonary atresia with intact ventricular septum	2
Double-outlet right ventricle, VSD, severe PS	1
D-transposition of great arteries, VSD, severe PS	1

PS = pulmonary stenosis, VSD = ventricular septal defect.

Surgery was performed through a posterolateral thoracotomy in the 3rd intercostal space, preferably in the side opposite that of the aortic arch. The PA was dissected and isolated proximally to obtain adequate length for arteriotomy. The proximal subclavian artery was dissected and isolated. In cases where the subclavian artery was small or there was an acute branching pattern from the innominate artery, the later was chosen for anastomosing the proximal end of the graft. A heparin bolus of 0.5 $mg \cdot kg^{-1}$ was administered and after waiting for 3 min, a clamp was placed on the subclavian artery. In all patients, a thin Gore-Tex PTFE tube graft (WL Gore, Elkton, MD, USA) was used for the interposition graft. The subclavian end of the graft was bevelled. A longitudinal arteriotomy was made in the subclavian artery. The bevelled end of the graft was anastomosed to the subclavian artery using 8/0 polypropylene continuous suture. The length of the graft was adjusted to reach the PA without kinking or traction. A straight cut was made at the pulmonary end of the graft. After a longitudinal pulmonary arteriotomy (Figure 1), the distal end of the graft was anastomosed to the PA with 8/0 polypropylene continuous suture (Figure 2). After releasing the clamp and establishing flow, sudden hypotension was managed with volume infusion and correction of metabolic acidosis. Inotropic infusion was started after releasing the cross clamp, to maintain adequate blood pressure. The prostaglandin E1 infusion was stopped after releasing the clamp. A routine dopamine infusion (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was started in all cases. Five patients required additional adrenalin infusion (0.05 $\mu g \cdot kg^{-1} \cdot min^{-1}$) to maintain adequate systemic pressure in the initial 24–48 hours postoperatively.

All neonates were ventilated postoperatively. After ensuring that there was no postoperative bleeding, heparin infusion was started at 10 $U \cdot kg^{-1} \cdot h^{-1}$ (keeping activated plasma thromboplastin time within 1.5 to 2 times control) within 4 to 6 hours of surgery. After extubation, once oral feeding was started, oral aspirin (5 $mg \cdot kg^{-1}$) was given to all patients and continued until corrective surgery was undertaken. Inotropics were tapered and stopped before moving the patient to the ward. The patency of the shunt was assessed postoperatively by clinical detection of a shunt murmur, pulse oxymetry, and arterial blood gas analysis. Two-dimensional echocardiography was performed in patients with suspected lack of shunt patency while in the ICU. Intravenous frusemide was used if there was excessive shunt flow, which was later switched to oral frusemide.

Results

Sixteen patients received a right-sided BT shunt and 3 had a left-sided shunt. A 4-mm PTFE graft was placed

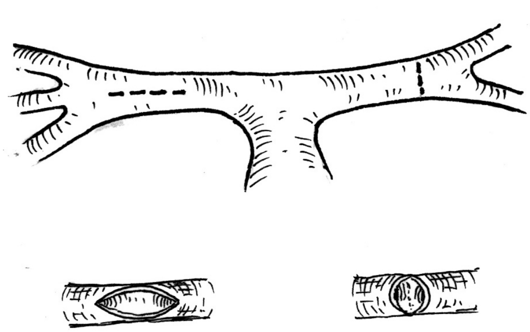

Figure 1. Diagram showing longitudinal pulmonary arteriotomy with its wider circumferential area.

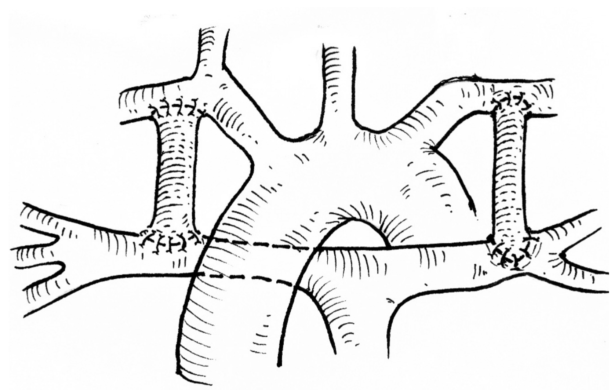

Figure 2. Blalock-Taussig shunt in situ.

in 18 patients, and one had a 3.5-mm PTFE graft. The mean duration of ventilation was 18.6 ± 3.3 hours (range, 10–36 hours). The mean ICU stay was 3.86 ± 2.06 days (range, 2–6 days). One patient was reexplored for control of bleeding from the pulmonary anastomosis site. In 2 patients with suspected partial blockage of the graft, 500 $U \cdot kg^{-1}$ heparin was given intravenously with a temporary increase in inotropics to maintain a higher blood pressure. One patient developed acute thrombosis for which shunt revision was performed postoperatively. In 2 patients, there was high flow in a patent ductus arterious postoperatively, and it was clipped in the operating room. There were 2 hospital deaths (mortality rate, 10.5%). Both patients who died were in shock preoperatively and hemodynamically compromised. There was no incidence of sepsis, seroma, or phrenic nerve palsy. The mean duration of hospital stay was 12.4 ± 8.3 days (range, 8 to 36 days). The mean O_2 saturation at discharge was 89% (range, 81%–93%).

The follow-up period ranged from 3 to 27 months, with a mean of 12 months. During follow-up, all patients were evaluated for shunt patency, PA growth and any proximal distortion of the PA. Five of the 8 patients who have undergone complete surgical correction had cardiac catheterization before the second operation. There was no late postoperative death or shunt blockage.

Discussion

In the neonatal age group, the modified BT shunt is a widely accepted palliative procedure for congenital cyanotic heart disease with decreased pulmonary blood flow and duct-dependent pulmonary circulation. Limitations of the classical shunt in newborns are kinking of the subclavian artery at its origin, PA tenting, ischemia of the ipsilateral upper limb and frequent early occlusion.[6,7] The advantages of the modified BT shunt using a PTFE graft is less likelihood of kinking and distortion and a better patency rate.[8,9] Younger age is an important risk factor for poor outcome, because of the smaller size, lack of tissue maturity, tissue edema related to prostaglandin infusion and neonatal PA hypertension. Meticulous surgical technique and a special management protocol can improve the outcome, as seen in our cohort.

Sivakumar and colleagues[10] described a transverse pulmonary arteriotomy for the distal shunt anastomosis. In our series, we made a longitudinal arteriotomy (both subclavian and pulmonary) as it creates a greater anastomotic circumferential area for a larger sized shunt in relation to pulmonary and subclavian artery size. This technique is particularly useful for hypoplastic PA. In a transverse arteriotomy, is it not possible to place a larger shunt in a hypoplastic PA due to the limited circumferential anastomotic area. At the same time, PA distortion at the anastomotic site is less with a longitudinal arteriotomy. None of our patients developed PA distortion (Figure 1). We always keep some soft tissues around the circumference of the subclavian and pulmonary arteries while dissecting, which gives a buttressing effect during anastomosis and prevents needle hole bleeding. A longitudinal arteriotomy should be performed to obtain an adequate anastomosis. It avoids luminal obliteration and arterial distortion.

Ilbawi and colleagues[2] used a 5-mm graft in most of their neonates, but the current trend is to use a smaller size of graft to prevent excessive pulmonary blood flow leading to congestive heart failure and PA hypertension, with early complete correction. Larger grafts are especially detrimental for correction of single ventricle physiology. In all our patients, we used a 3.5 or 4-mm PTFE graft. We used a 4-mm PTFE graft in 18 of 19 patients and accepted a higher saturation (mean, 89%) at the time of discharge. This is contrary to the wide acceptance of a 3.5-mm graft for the average neonate in most centers. Because most of our patients hail from rural areas with limited medical access, we prefer to provide a higher saturation to accommodate changes due to growth and delayed follow-up.

Several studies have used 7/0 polypropylene for anastomosis of the shunt.[10,11] We used 8/0 polypropylene in all patients for better anastomosis and less needle hole bleeding. Rao and colleagues[11] used dopamine postoperatively to keep systolic pressure at 80 mm Hg. We also used inotropics to maintain adequate systemic blood pressure. Most surgeons heparinize patients at the time of the surgical procedure. Mullen and colleagues[12] found no instances of seroma when heparinization was not initiated during surgery. Shivakumar and colleagues[10] used a low dose of heparin to achieve hemostasis. Both groups believe that heparinization before insertion of the shunt increases the risk of seroma and serous effusions. In the series reported by Al Jubair and colleagues,[13] use of heparin improved immediate shunt function. We believe in initiating heparinization with a bolus dose before putting the clamp on the subclavian artery. We did not encounter any incidence of seroma or serous effusion. In 2 patients with suspected slight blockage of the graft manifested by decreased PaO_2 and SpO_2 and increased acidosis in arterial blood gas analysis, 500 $U \cdot kg^{-1}$ heparin was given intravenously, with a temporary increase in inotropics to maintain a higher blood pressure. The shunt murmur reappeared with improved saturation and PaO_2. Improvement in shunt function was also demonstrated by echocardiography.

The incidence of shunt blockage in the immediate postoperative period varies from 4%–10%.[10,11] We found only one case of shunt blockage (5%). We assume that meticulous surgical technique and use of heparin intraoperatively and in the immediate postoperative period reduced the incidence of early shunt block in our series. We prefer to leave the ductus open for spontaneous closure after stopping prostaglandin infusion. In 2 patients, we found pulmonary edema on chest radiography, metabolic acidosis, a drop in oxygen saturation and a shunt murmur. Immediate postoperative echocardiography showed a patent ductus, which was clipped. Tissue immaturity, critically ill neonates and perioperative complications increase postoperative mortality in the neonatal age group. Various reports show a wide range of hospital mortality rates, ranging from 3.3% to 29%.[2,10,12,14,15] In our series, mortality was 10.5% and both patients had poor preoperative hemodynamic status.

Meticulous surgical technique in the form of minimal tissue dissection, longitudinal arteriotomy, use of 8/0 polypropylene suture, adequate sizing of the shunt length and a shunt size proportionate to that of the pulmonary artery improves outcome and reduces the chances of postoperative reexploration. Judicious use of heparin and inotropic agents in the immediate postoperative period reduces the incidence of shunt blockage and seroma. Poor preoperative clinical status and hemodynamic parameters seem to be risk factors for postoperative morbidity and mortality.

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