

Two Cases of Transcatheter Closure of Central Aortopulmonary Shunts:

One with an AMPLATZER Duct Occluder II and
One with an AMPLATZER Vascular Plug I

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When total correction is not possible in infants who have a cyanotic congenital heart disease, creation of a palliative aortopulmonary shunt is essential. A central aortopulmonary shunt is preferable, because of its technical and hemodynamic advantages. Overcirculation, thrombosis, and stenosis of the shunt are the main postoperative sequelae that necessitate urgent reintervention. Percutaneous transcatheter closure of aortopulmonary shunts can eliminate the need for reoperation and substantially decrease postoperative morbidity and mortality rates. We report our successful transcatheter closures of central aortopulmonary shunts in a 3-month-old infant and a 15-year-old girl, with use of an AMPLATZER Duct Occluder II and an AMPLATZER Vascular Plug I, respectively. To our knowledge, this is the first report of the transcatheter closure of central aortopulmonary shunts with these 2 devices. (Tex Heart Inst J 2016;43(3):241-5)

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Creating a palliative aortopulmonary shunt occlusion is essential when total correction is not feasible in cyanotic infants. Although modified Blalock-Taussig shunt procedures have been performed extensively, central shunts have technical and hemodynamic advantages and are therefore preferable.¹ Overcirculation, thrombosis, and stenosis of the shunt are the main postoperative sequelae that necessitate urgent reintervention.² Percutaneous transcatheter interventions on aortopulmonary shunts can eliminate the need for reoperation and substantially decrease postoperative morbidity and mortality rates. We report 2 pediatric cases of overcirculation caused by central aortopulmonary shunts, and our use of 2 different AMPLATZER devices to occlude them.

Case Reports

Patient 1

A 13-day-old female infant with cyanosis was referred to our hospital. Echocardiograms revealed a nearly atretic pulmonary valve (PV), a mildly hypoplastic right ventricle (RV), a tricuspid annulus 9.5 mm in diameter (Z score, -1.84), and moderate tricuspid regurgitation. The patient was started on prostaglandin E₁ therapy, and emergency cardiac catheterization was undertaken. After predilating the PV with use of a 4-mm coronary balloon, we performed pulmonary balloon valvuloplasty with use of an 8-mm × 2-cm Tyshak[®] Percutaneous Transluminal Valvuloplasty balloon (NuMED, Inc.; Hopkinton, NY). Thereafter, the patient's RV pressure decreased from 110 to 42 mmHg, and PV regurgitation was mild. We discontinued prostaglandin E₁ infusion and waited to see if the patient's oxygen saturation decreased; it remained at 80%, so infusion was ended. However, after 4 hours, we had to restart the infusion immediately, to undertake ductus stenting. When ductal patency could not be reestablished, we performed an emergency central aortopulmonary shunt operation with use of a 3.5-mm polytetrafluoroethylene (PTFE) graft. The patient's oxygen saturation increased from 64% to 88%, and she was discharged from the hospital on postoperative day 7.

Two months later, the infant presented with shortness of breath and failure to thrive. Her oxygen saturation was 92%. We observed somatic growth retardation (body weight, 3 kg), pulmonary overflow, enlarged left-side heart cavities, and increased shunt flow. Therapy for congestive heart failure was given, and we undertook percutaneous transcatheter closure of the central aortopulmonary shunt (Fig. 1). We inserted a 5F sheath into the femoral artery, and we administered 75 U/kg of heparin to achieve an activated clotting time of >250 s. After aortography, and with use of a 4F internal mammary artery (LIMA) catheter and a 0.014-in coronary guidewire, we crossed the central shunt and placed the guidewire in the left pulmonary artery (PA). The guidewire was exchanged for a stiff guidewire. We advanced a 4-mm × 2-cm coronary balloon to the shunt and inflated it, to see if the patient's oxygen saturation and vital signs deteriorated. The oxygen saturation had not changed, so we decided to occlude the shunt. We chose a 4-mm AMPLATZER® Duct Occluder II (ADO II) (St. Jude Medical, Inc.; St. Paul, Minn). We pushed the delivery system over the stiff guidewire to the PA. We deployed the distal disc of the device in the PA and the proximal disc in the lumen of the ascending aorta (Fig. 2). An angiogram showed no residual shunting (Fig. 3). The patient's preprocedural systemic arterial blood pressure of 92/42 mmHg rose to 100/73 mmHg. No major vascular sequelae were observed. After the procedure, the patient's congestive heart failure symptoms disappeared, and no further intervention was necessary during the 10-month follow-up period.

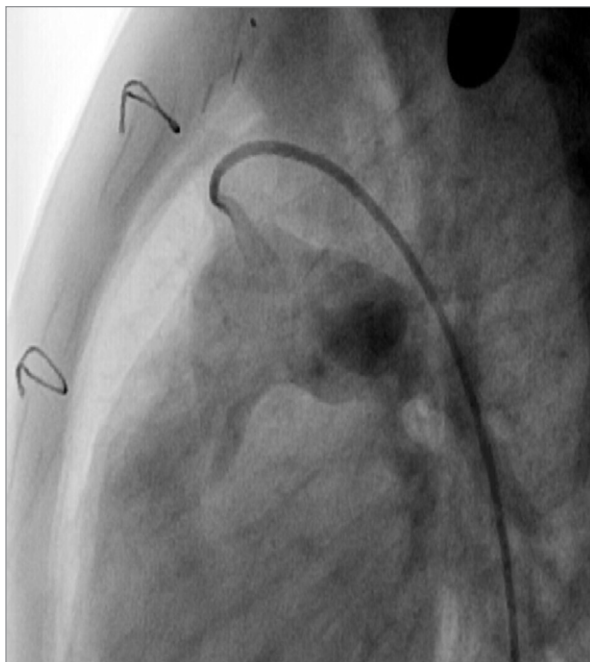


Fig. 1 Patient 1. Angiogram shows a central shunt.

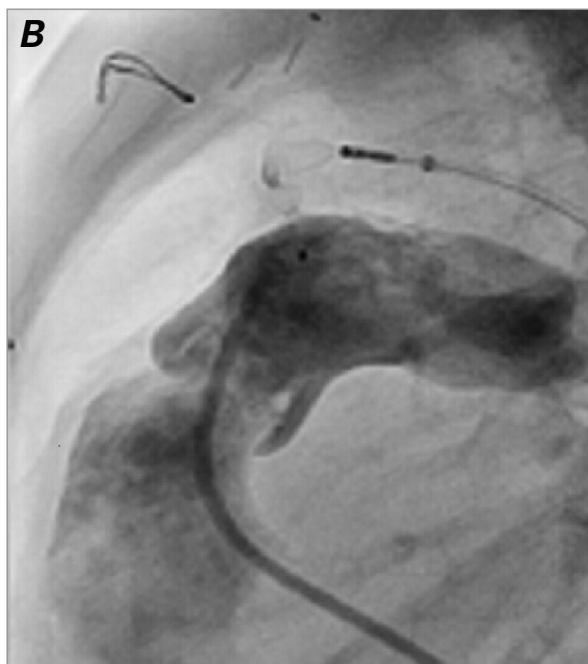
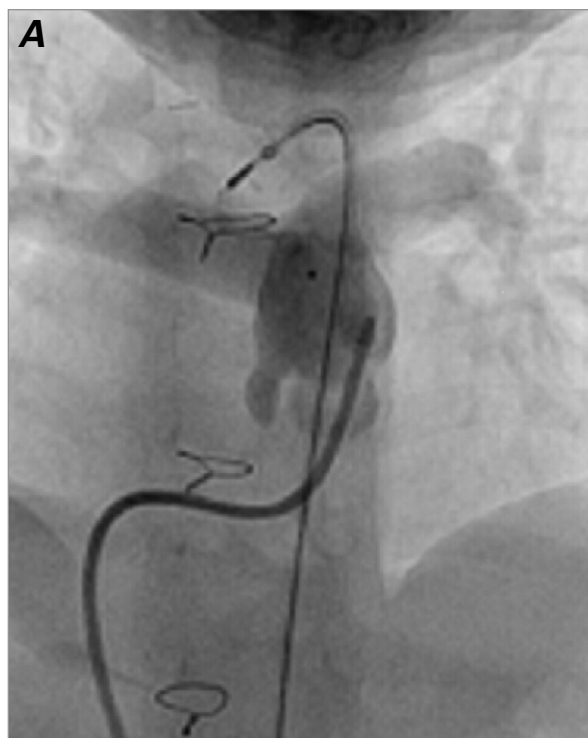


Fig. 2 Patient 1. Angiograms in **A**) anteroposterior and **B**) lateral angulated views show deployment of the distal disc of the AMPLATZER Duct Occluder II in the pulmonary artery and the proximal disc in the lumen of the ascending aorta.

Patient 2

A 15-year-old girl with a history of pulmonary valvotomy and a central aortopulmonary shunt operation for critical pulmonary stenosis was admitted to our hospital with fatigue. The first operation had been performed

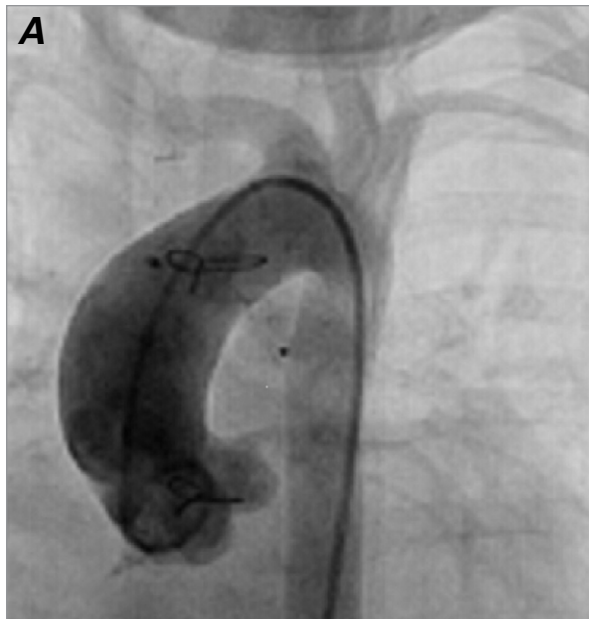


Fig. 3 Patient 1. Angiograms in **A**) anteroposterior and **B**) lateral angulated views show no residual shunting after device deployment.

at another hospital when she was 10 years old; we had no information about the shunt's size. Echocardiograms revealed a mildly hypoplastic RV and tricuspid valve (annular Z score, -1.7), moderate pulmonary insufficiency, enlarged left-side heart chambers, an atrial septal defect (ASD) with a right-to-left shunt, and a patent central aortopulmonary shunt between the ascending aorta and the right PA.

We undertook percutaneous transcatheter closure of the central aortopulmonary shunt and the ASD. We inserted a 6F introducer sheath (Cordis, a Johnson & Johnson company; Miami Lakes, Fla) into the femoral artery and femoral vein. After aortography, and using a 4F LIMA catheter and an 0.0018-in Angiotech® nitinol guidewire (PBN Medicals Denmark A/S; Stenlose, Denmark), we crossed the central shunt and placed the nitinol guidewire in the right PA. The internal diameter of the shunt was 6 mm. For closure, we chose a 10-mm AMPLATZER® Vascular Plug I (AVP I) (St. Jude Medical). We pushed the delivery system to the PA over the guidewire, then advanced the vascular plug and deployed it in the central shunt (Fig. 4). An aortogram showed no residual shunting (Fig. 5). After shunt closure, the patient's oxygen saturation decreased from 93% preprocedurally to 88%; her systemic arterial pressure improved from 120/50 mmHg preprocedurally to 110/70 mmHg. We advanced a 6F end-hole catheter to the left atrium through the femoral vein, inferior vena cava, and right atrium, then placed it in the superior pulmonary vein with the guidance of a long hydrophilic guidewire. We then advanced a 24-mm septal sizing balloon over that guidewire across the defect, to measure the stop-flow and stretched sizes of the defect and to evaluate any changes in pressures and saturation. In comparison with preprocedural values, the central venous pressure had increased from 7 to 9 mmHg, the systemic blood pressure was unchanged, and the oxygen saturation had increased from 88% to 97%. The stop-flow and stretched sizes were 22 and 23 mm, respectively. We deployed a 22-mm Cocoon septal occluder

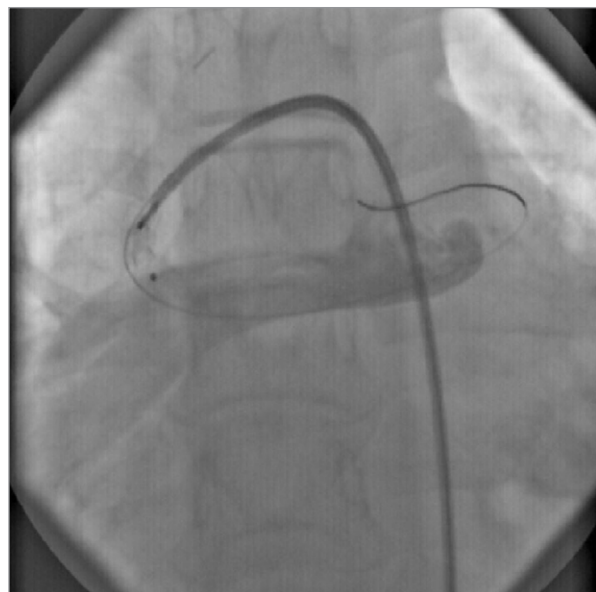


Fig. 4 Patient 2. Angiogram shows the AMPLATZER Vascular Plug I deployed in the shunt.

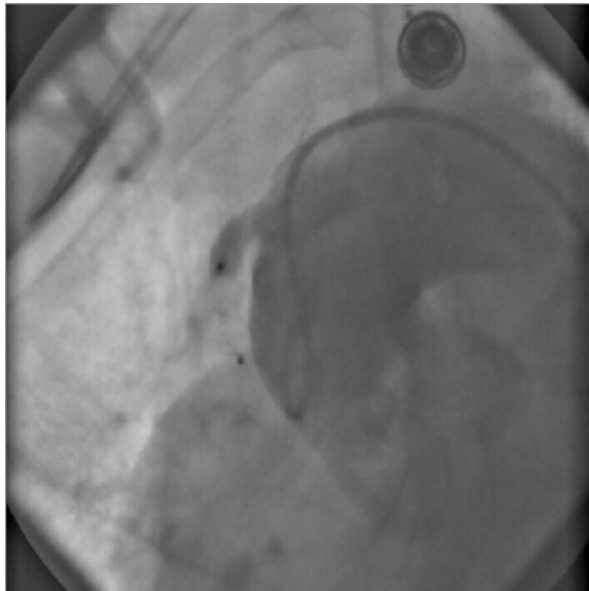


Fig. 5 Patient 2. Final aortogram shows no residual shunting.

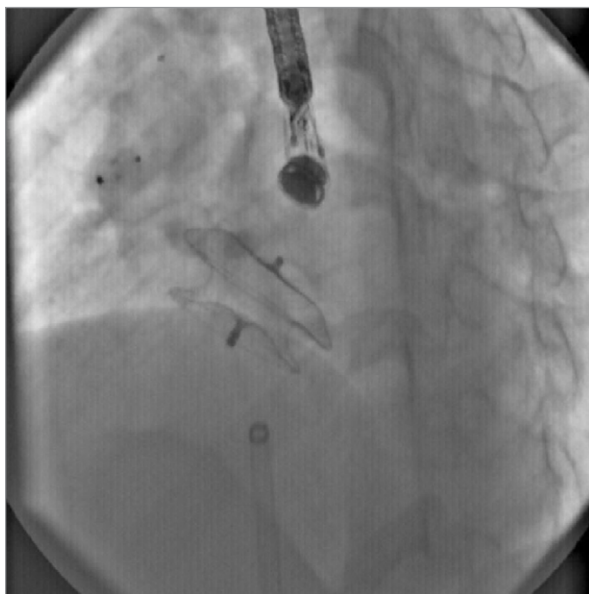


Fig. 6 Patient 2. Postprocedural angiogram shows the atrial septal defect device and plug.

(Vascular Innovations Co., Ltd.; Bangtannl Pakked, Thailand) into the interatrial septum (Fig. 6).

After the procedure, the patient's congestive heart failure symptoms disappeared, and she was discharged from the hospital on postoperative day 4. No evidence of RV failure was observed and no further intervention was necessary during the 3-year follow-up period.

Discussion

Transcatheter closure of modified Blalock-Taussig shunts has been described previously.^{3,4} To our knowl-

edge, however, this is the first report of the transcatheter closure of central aortopulmonary shunts with the use of 2 different types of devices.

The percutaneous occlusion of a central shunt is technically challenging (especially when the ascending aorta is dilated), and various catheter-guidewire combinations and techniques might be necessary.^{5,6} In addition, the sharp angle between the PTFE graft and the ascending aorta often makes this procedure even more difficult to perform; we found the use of a LIMA catheter convenient for crossing the shunt in our patients. On the other hand, percutaneous closure offers several advantages, including the ability to test shunt occlusion, no repeated chest incisions, no need for excessive blood transfusion, shorter hospital stays, and prevention of recurrent laryngeal nerve paralysis and thoracic duct damage.^{4,6}

Various devices, coils, and detachable balloons have been used to occlude aortopulmonary shunts. We used an ADO II and an AVP I for transcatheter occlusion.^{3,7} The AVP I has a single lobe and single-layer mesh, offering appropriate length for short landing zones. The ADO II has 6 levels of occlusion with high closure rates and enables rapid shunt occlusion.³ Both devices are easily advanced and deployed. However, use of the ADO II requires positioning at least one retention disc outside the shunt, which increases the risk of stenosis in the ascending aorta, PA, or both. Using an AVP I, because its single lobe stays in the PTFE graft, can reduce the risk of stenosis. The ADO II in our patient caused no problems.

Neonates with critical pulmonary stenosis can experience severe desaturation after pulmonary valvotomy and balloon valvuloplasty because of poor RV compliance. These patients might temporarily need additional pulmonary blood flow until RV compliance improves. Pulmonary flow has been supported by surgically created aortopulmonary shunts or ductal patency ensured by long-term prostaglandin E₁ therapy. Ductal stenting has become the preferred option because of its advantages: no need for surgery, shorter hospital stays, and lower risk of branch-PA distortion, chylothorax, and phrenic and vagal paralysis.^{8,9} The rapid closure achieved with ductal stenting also eliminates the need for further interventions, especially in patients with hypoplasia of the RV who are candidates for biventricular repair. In our hospital, we prefer ductal stenting as first-line treatment in neonates who have critical pulmonary stenosis. However, we could not undertake ductal stenting in our Patient 1, because ductus patency could not be established. Patient 2 had been referred from another hospital.

When ASD closure is possible in cyanotic patients who have hypoplastic RVs, it improves oxygen saturation and precludes paradoxical embolism. However, in patients whose RV development is insufficient, ASD closure can be deleterious. Investigators have described values that should be obtained in such patients during

test occlusion of the ASD, including oxygen saturation, systemic arterial pressure, and right atrial pressure and saturation.¹⁰ Upon testing the occlusion of Patient 2's ASD, we observed that her right atrial pressure had not substantially increased, her systemic blood pressure had not decreased, and her oxygen saturation had increased to 97%. We therefore undertook ASD closure.

On the basis of our experience, we conclude that percutaneous transcatheter closure of a central aortopulmonary shunt with use of an ADO II or an AVP I can be effective and safe. More experience and more reports are warranted.

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