RESULTS
The deployment of the PVIC was easy to perform. The time from skin cut to deployment was 58 minutes. The PVIC installation time was 1 minute, and the sealing was successful.

After the PVIC function was stable, the endoscope offered a clear view to the pulmonary valve (Figure 1, C). The hemodynamics and saturation of oxygen were stable (Figure 2).

The resection of the pulmonary leaflets took 21 minutes and was complete. The PVIC had to be exchanged 4 times because of laser-related damages of the supravalvular balloon.

The deployment of the valved stent took 30 seconds. The stent was deployed in a supra-annular position (Figure 1, E).

The gross anatomy of the endomyocardium showed only superficial lesions. All other structures were unaffected.

DISCUSSION
The feasibility of valve isolation, resection, and implantation of a new valved stent was demonstrated in this beating heart model. The hemodynamic data of the pig were stable during the isolation with only moderate vasopressor administration. The stent was stored in a “parking slot” during the procedure and deployed in a good position. During resection, the supravalvular balloon was injured by the laser 4 times and had to be changed. Nonetheless, the circulatory system was stable after all sealing procedures, but these exchanges explained the extended resection time.

This model was performed in the pulmonary position because of a lower mean blood pressure and the absence of coronary ostia compared with the aortic position. The development of a more complex sealing procedure for the aortic position is in progress.

CONCLUSIONS
The transapical insertion of heart valves is presented as a good concept, not only for the well-known aortic position but also for the mitral valve. In the future it will be important for cardiac surgeons to offer a complete valve replacement to patients according to the gold standard of surgery, especially for highly calcified aortic valves.

REFERENCES

One-stage repair for interrupted aortic arch and associated cardiac anomalies in an adult

Zhang Jinzhou, MD,a Wang Wen, MD,b Zhu Hailong, MD,a and Wang Hongbing, MD,a Xi’an, People’s Republic of China

From the Departments of Cardiovascular Surgerya and Traditional Chinese Medicine,b Xijing Hospital, the Fourth Military Medical University, Xi’an, People’s Republic of China.

Disclosures: None.

Zhang Jinzhou and Wang Wen contributed equally to this work.

Received for publication Nov 4, 2009; accepted for publication Jan 1, 2010; available ahead of print March 26, 2010.

Address for reprints: Zhang Jinzhou, MD, and Wang Hongbing, MD, Department of Cardiovascular Surgery, Xijing Hospital, the Fourth Military University, Xi’an (710032), People’s Republic of China (E-mail: jinzhouzhang2006@yahoo.com; ailisr@yahoo.com.cn).

J Thorac Cardiovasc Surg 2010;140:479-81
0022-5223/$36.00
Copyright © 2010 by The American Association for Thoracic Surgery
doi:10.1016/j.jtcvs.2010.01.008

For infants with interrupted aortic arch (IAA) and associated cardiac malformation, 1-stage repair is preferred, and excellent results have been reported. However, similar experiences in adult patients are limited because of the rarity of the condition. We report a woman with IAA and associated cardiac malformations who was treated surgically by 1-stage repair.

CLINICAL SUMMARY
A 21-year-old woman was admitted for congenital heart murmur. Echocardiography revealed a subpulmonary...
ventricular septal defect (left-to-right shunts), patent ductus arteriosus (PDA, right-to-left shunts), and pulmonary hypertension (103 mm Hg). The aortic isthmus and descending aorta were not visible. Multislice computed tomography (CT) angiography revealed a thinning ascending aorta ($D = 2.3$ mm) and a hypoplastic aortic arch ($D = 7$ mm). The descending aorta was connected to the main pulmonary artery through a PDA. The main pulmonary artery was considerably dilated ($D = 90$ mm) (Figure 1). Catheterization revealed anatomic discontinuity between the aortic arch and the descending aorta. The resistance of the pulmonary artery was 14 Wood units, and the resistance of the pulmonary microvessels was 12.5 Wood units.

The patient underwent 1-stage repair under cardiopulmonary bypass through the midline sternotomy. The left femoral artery cannula was used for the lower body perfusion. The ventricular septal defect was repaired with a Dacron patch with a 1-way flap ($D = 10$ mm). Before the Dacron graft was sutured to the descending aorta, an incision was made in the main pulmonary trunk and a Foley catheter was inserted 1 cm into the descending aorta through the PDA. The PDA tissue was carefully cut from the descending aorta until normal tissue emerged; the Foley catheter was through the cavity of the graft, and the artificial conduit ($D = 18$ mm) was anastomosed to the proximal descending aorta with 5-0 Prolene. Next, the innominate, left common carotid, and left subclavian arteries were clamped, and cerebral perfusion was stopped. The proximal end of the conduit was connected to the ascending aorta by an incision in the lateral wall of ascending aorta. The full flow rate was restarted. The main pulmonary artery was directly closed using 5-0 Prolene.

Early postoperatively, the patient had a hoarse voice, and electro-laryngopharyngoscopy revealed normal epiglottis and fixation of vocal cords on both sides. On follow-up, she was able to enunciate normally. She reported pain in her left leg, but the dorsalis pedis artery pulse was normal and she could walk. Three months later after administration of neurotrophic drugs, the symptoms diminished. Sixteen months later, her arterial blood pressures were 125/76 mm Hg in the upper limbs and 134/66 mm Hg in the lower limbs. She no longer received medicine. Multislice CT scans revealed that the graft was implanted between the ascending and descending aortas (Figure 2).

**DISCUSSION**

For adult patients with IAA, an extra-anatomic approach is used to repair the aortic anomaly, which involves

**FIGURE 1.** CT axial view: enlarged main pulmonary artery. The descending aorta was connected to the main pulmonary artery through the PDA. AA, Ascending aorta; MPT, main pulmonary trunk; PDA, patent ductus arteriosus; RPA, right pulmonary artery; DA, descending aorta.

**FIGURE 2.** Ascending aorta was connected to the descending aorta via an artificial blood vessel. The enlargement of the main pulmonary artery was lessened to an extent. AA, Ascending aorta; MPT, main pulmonary trunk; DA, descending aorta; LSA, left subclavian artery; Sto, stoma; ABV, artificial blood vessel.
a graft-assisted anastomosis between the ascending aorta and the suprarenal abdominal aorta. In our patient, a graft was implanted between the ascending and descending aortas.

To avoid uncontrolled bleeding, there are 3 aspects that must be focused on perioperatively. First, all ductal tissue must be aggressively removed. Otherwise the anastomosis between the graft and the aorta will be susceptible to bleeding. Second, the anastomosis should be free of tension. Careful study of CT scans and better preoperative preparation are helpful to achieve complete dissociation of the descending aorta. More important, implanting a conduit will assist in reducing the tension. Third, pressuring hemostasis is effective when uncontrolled bleeding occurs. A bandage was placed with a little pressure on the stoma and thoracic cavity. One of the tips was placed out of the thoracic cavity. The bleeding volume was 900 mL on postoperative day 1 and 350 mL on postoperative day 2. The hemostasis technique was an effective way to manage uncontrolled bleeding in this patient.

Because of the pulmonary artery aneurysm and the difficulty in exposing the PDA and descending aorta in this patient, we used the left femoral artery to perfuse the lower body. With these methods, circulatory arrest of the lower body was avoided and the risk of neurologic complications was minimized. The postoperative course was uneventful. The patient was discharged and has fully recovered.

References

A new concept for correction of systolic anterior motion and mitral valve regurgitation in patients with hypertrophic obstructive cardiomyopathy

Joerg Seeburger, MD, Jurgen Passage, MBBS, Michael A. Borger, MD, PhD, and Friedrich Wilhelm Mohr, MD, PhD, Leipzig, Germany

In patients with hypertrophic obstructive cardiomyopathy (HOCM), systolic anterior motion (SAM) of the anterior mitral leaflet (AML) can occur as a result of acceleration of flow in the left ventricular outflow tract. In addition, relative shortening of the chordae tendineae may reduce AML mobility and contribute to the generation of mitral regurgitation (MR). We present a new concept for correction of SAM and MR in patients with HOCM and restricted AML movement.

METHODS
The procedure consists of 2 stages, septal myectomy followed by complete resection and replacement of all AML chordae with the loop technique. Myectomy enlarges the left ventricular outflow tract and reduces the risk of SAM. Resection of the foreshortened chordae to the AML and replacement with longer neochordae also reduces the risk of residual postoperative SAM and subsequent MR.

RESULTS
Since March 2008, 4 consecutive patients with HOCM, severe SAM, and evidence of restricted AML movement have been operated according to this new concept. In all cases, loops (average 8 per patient; range, 7–9) measuring 26 mm in length were implanted from both papillary muscles to the free margin of the AML. A flexible posterior band was implanted in 3 patients. Perioperative course was uneventful, with no major adverse events in all cases. Predischarge control showed no evidence of SAM and/or MR.

From the Department of Cardiac Surgery, Heartcenter, Leipzig University, Leipzig, Germany.
Received for publication Dec 11, 2009; accepted for publication Jan 1, 2010; available ahead of print March 26, 2010.
Address for reprints: Joerg Seeburger, MD, Heartcenter, Leipzig University, Struerpelpelstrasse 39, 04289 Leipzig (E-mail: seej@med.uni-leipzig.de).
J Thorac Cardiovasc Surg 2010;140:481-3
0022-5223/$36.00
Copyright © 2010 by The American Association for Thoracic Surgery
doi:10.1016/j.jtcvs.2010.01.010