Mattia Glauber, MD
Michele Murzi, MD

Department of Adult Cardiac Surgery
G. Paquinucci Heart Hospital
Fondazione CNR-G. Monasterio
Massa, Italy

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Reply to the Editor:

We appreciate the comments made by Glauber and colleagues regarding our article about surgical treatment of acute type A dissection using the stented elephant trunk technique.¹ They thought that it was unnecessary to implant the stented elephant trunk into the distal aorta. They raised interesting points: replace the ascending aorta and the arch and relocate the origin of the supra-aortic vessels very proximally just above the sinotubular junction. A safe and long proximal landing zone would thereby be created to facilitate subsequent endograft repair of the distal aorta (if necessary).

In fact, the fate of the residual distal aorta after acute type A dissection repair is controversial. Even though expansion of the residual aorta after surgical repair is a slow and linear process that makes distal reoperations relatively uncommon, as recommended by their team and others,² a recent report demonstrated that hemiarch replacement

for acute type A dissection had a high mortality rate compared with total arch replacement combined with stented elephant trunk implantation.³ In our opinion, this might be associated with the difference of the extent of propagation and involvement of aortic dissection. Thus we divided acute type A dissection into two subtypes: subtype C and subtype S. (1) Subtype C (complex type) is characterized by the primary tear located in the transverse arch or the descending aorta; aneurysm formation in the aortic arch or the distal aorta (≥ 40 mm); involvement, aneurysm formation, and occlusion of the brachiocephalic artery; or Marfan syndrome. (2) In subtype S (simple type), the primary tear is located in the ascending aorta without the features of subtype C. There was no need to perform total arch replacement.

Subtype C was associated with a higher risk of reoperation using a conventional strategy (limited ascending aortic or hemiarch replacement). Total arch replacement was recommended to decrease the risk of distal reoperation after surgery. If aortic dissection extended beyond the distal arch, a stented elephant trunk was implanted into the distal aorta. Implantation of a surgical stent graft into the distal aorta involved the following: (1) secure the anastomosis; (2) simplify total arch replacement; (3) promote thrombosis of the false lumen in the distal aorta; (4) enlarge the true lumen; (5) and facilitate the later thoracoabdominal aortic replacement, if necessary.

As for the authors' interesting approach, several issues should be raised. Compared with the antegrade intraoperative surgical stent, it is troublesome to deliver the stent graft retrogradely via the femoral artery. Because there is no extravascular graft of the end of the stent graft to be used for sewing, it is difficult to manage if a late distal aortic reoperation is needed. The complications of the endovascular treatment cannot be prevented either.

LiZhong Sun, MD^a
RuiDong Qi, MD^b

^aBeijing Aortic Disease Center
Beijing Institute of Heart Lung and
Blood Vessel Diseases & Beijing
Anzhen Hospital
Capital Medical University
Beijing, China
^bDepartment of Cardiovascular
Surgery Tianjin Cardiovascular
Institute & Tianjin Chest Hospital
Tianjin, China

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IS A TRANSANNULAR PATCH ALWAYS NECESSARY?

To the Editor:

Congratulations to Brancaccio and colleagues¹ for this valuable study. The reported surgical technique stated that transannular patch plasty was performed in patients with a pulmonary valve Z-score less than -2. However, we have two questions: Did the authors think about the septal excision of trabecula septomarginalis? Could there be a solution without using a transannular patch supposing that this pulmonary stenosis was secondary to ventricular septal defect (VSD)?

We think that the ratio of residual VSD is high because of the technique, not the surgeon. During the repair of VSD, pledgets are left on the right ventricular side (needle is inserted from the right ventricular side of VSD in the classic method). With this method, pressure (pressure gradient between

left and right ventricles) on the patch is carried by the suture itself instead of its pledget. This creates a gliding effect on the myocardium. We think that this is the reason for the high incidence of residual VSD. Did the authors consider any standardization of the patch size?

Mert Kestelli, MD

Ismail Yurekli, MD

Ali Gurbuz, MD

*Izmir Ataturk Education and Research
Hospital*

*Department of Cardiovascular
Surgery*

Basin Sitesi

Izmir, Turkey

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Reply to the Editor:

We thank Kestelli and associates for their comments. Two mechanisms are involved in the etiology of the associ-

ation of tetralogy of Fallot and complete atrioventricular septal defect: the anterior deviation of conal septum and the lack of conal septum contribution to the development of the atrioventricular valves.¹ These mechanisms are responsible for the right ventricular outflow tract obstruction and the creation of a wide ventricular septal defect (VSD) with subaortic extension, respectively. The embryologic basis explains that the closure of the VSD is not responsible for pulmonary stenosis and that resection of the septomarginalis trabecula could not be sufficient to relieve the right ventricular outflow tract.

The incidence of residual VSD reported in our study (9%) is consistent with previous reports^{2,3} and mostly related to technical issues across 15 years of experience, in which VSD closure was performed with a single continuous suture. During the past 9 years we have modified our technique, adopting multiple interrupted sutures reinforced with pledgets. As suggested by He and Mee,² the size and shape of the patch for VSD closure is important.

The patch is shaped like a comma and inserted on the right side of the inter-ventricular septum to avoid atrioventricular block. The sizing is chosen to avoid left ventricular outflow tract obstruction, if too large, and residual VSD, if too small.

Gianluca Brancaccio, MD, PhD

Vincenzo Affinito, MD

Roberto M. Di Donato, MD

Department of Pediatric

Cardiothoracic Surgery and

Cardiology

Bambino Gesù' Pediatric Hospital

Rome, Italy

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