Letters to the Editor

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The importance of distal fixation in total arch replacement for distal aortic arch aneurysm

To the Editor:

We read with interest the article from Toda and colleagues[1] about single-stage repair of arch aneurysm. We think that total arch replacement (TAR) with the long elephant trunk (LET) in this article has some potential as an alternative treatment for thoracic aortic aneurysm; however, we cannot agree with the conclusion.

In this article TAR with an LET was applied for arch aneurysm down to the level of the tracheal bifurcation. We previously reported that the aneurysms of the aortic arch were safely accessible from the midsternotomy to the level of tracheal bifurcation, and furthermore, we could reach a portion 1 cm lower than the tracheal bifurcation irrespective of the working space, shapes of the aneurysm, and quality of the aortic wall.[2] TAR was safely achieved with 4 branched grafts, with a mortality of 0.8% to 6.4% and a stroke ratio of 0.84% to 3.2%.[2-4] The authors stated that 9% of patients who did not demonstrate complete thrombosis of the aneurysms required distal anastomosis through a left thoracotomy as the second operation during a relatively shorter period.[1] We think the aneurysms must be excluded completely and securely to prevent aneurysm rupture in the future.

In addition, we are concerned about whether the thrombosed space around the LET actually becomes organized and the aneurysmal wall is really decompressed. We have several patients who underwent TAR with an elephant trunk procedure and second-stage endovascular stent graft. Their aneurysms outside the graft were not opacified with contrast material on computed tomographic analysis; however, the aneurysm was enlarged during follow-up. Usui and associates[5] reported several cases with unexpectedly enlarged arch aneurysms of the TAR with a frozen elephant trunk. They stated that anchoring the graft distal to the aneurysm was mandatory.[5] We consider that "not enhanced" in the computed tomographic scan does not always mean "thrombosed."

Finally, TAR with an LET might interrupt the ostia of the intracostal arteries to the spinal cord and has greater risk of paraplegia than standard TAR.

There are some indications for this method, such as entire thoracic aortic aneurysms that have no distal anastomotic site, aneurysms extending too far from the tracheal bifurcation, and acute aortic dissection with entry is away from the arch vessels. However, distal fixation of the free-flowing graft to the descending aorta is mandatory during TAR or in the second-stage operation.

In conclusion, we insist that the standard TAR should be applied for the arch aneurysm down to the level of tracheal bifurcation. We would like to congratulate the authors for their contributions in this field and their excellent results.

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doi:10.1016/j.jtcvs.2007.09.081

Reply to the Editor:

We would like to thank Drs Asano and Okita for their comments regarding our study,[1] in which we demonstrated that the majority of arch aneurysms could be repaired with a long elephant trunk (LET) anastomosed at the base of the innominate artery without distal anastomosis in the descending aorta. They raised the issue of distal fixation of the LET in our technique. Although the proximal end of the LET is sutured at the base of the innominate artery and proximal endoleak is prevented, the distal end of the LET is not fixed, and distal endoleak is possible with our method. As demonstrated in Figure 2, *C*, in our article, the aneurysmal sac outside the LET was enhanced with contrast material in 9% of our patients, and they required distal fixation of the LET.

However, it is important to note that the majority (91%) of our patients did not show contrast material outside the LET, and none had expansion or rupture of the aneurysm occur during the average follow-up of 33 ± 18 months. Furthermore, it was encouraging for us to find that the nonenhanced aneurysmal sac around the LET shrank more than 5 mm in 82% of the patients and more than 10 mm in 50% at 1 year after total arch replacement with the LET, as shown in Figure 2, *B*, of our article. The follow-up period might not have been long enough to rule out the possibility of late expansion of a nonenhanced aneurysm, and we consider that long-term follow-up of our patients is mandatory.

In their letter they referred to their article,[2] in which they insist that arch aneurysms with extension to 1 cm below the level of the carina are accessible from a median sternotomy. Interestingly, they demonstrate that an increased depth of distal anastomosis is a risk factor for prolonged distal anastomosis, even in Japanese patients, whose chest cavities are generally smaller than those in white patients.[2] Based on my personal experience as a clinical fellow for 3 years in United States, I wonder how many surgeons are comfortable to do the distal anastomosis 1 cm below the level of the carina in a typical barrel-chested white patient. On the other hand, with our technique, the descending aorta in the deep posterior mediastinum does not need to be exposed, but rather only a distal anastomosis is performed in the distal ascending aorta. Because of this simplicity, we required only 23 ± 8 minutes of hypothermic circulatory arrest in the lower body to complete the insertion and anastomoses of the LET. Thus we conceive that the benefit of our technique might be even more evident in white patients with a large chest.

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doi:10.1016/j.jtcvs.2007.10.076

Tissue-engineered heart valves: Bioreactor—yes or no?

To the Editor:

I read with great interest the article by Vincentelli and associates.[1] In this article the authors present the results of the effects of in situ injection of autologous bone marrow–derived mononuclear cells and mesenchymal stem cells (MSCs) on the outcome of xenogenic decellularized scaffolds in a lamb model. The main focus of this article is on a new method to reseed the cells in valve scaffolds consisting of an in situ injection of bone marrow cells into a porcine decellularized scaffold before implantation. Tissue-engineered heart valves created from MSCs and injected directly in a decellularized xenograft scaffold exhibited satisfactory hemodynamic aspects after 4 months.

Even if the authors demonstrate that in the MSC group the global organization of collagen fibers was preserved, a few fusiform cells were observed in the subendothelial area and the adventitia.

Pulmonary leaflets present few recolonizing cells positive for α-actin staining, with a typical organization in 3 layers, fibrosa, spongiosa, and ventricularis, as previously reported by others,[2] who showed that decellularized pulmonary valves revealed a well-preserved 3-dimensional network of collagen fibers in the extracellular matrix.

As we know, maintenance of the structural valve properties is fundamental to preserve the extracellular matrix with its complex of glycosaminoglycans and collagen fibers produced from valve fibroblasts and active fibroblasts. This appears to be fundamental for the mechanical valve's capability and its long-term durability.[2-4] Repopulation of a decellularized valve is crucial for valve vitality. How this could be achieved still remains controversial. As a matter of fact, at this time, it is not completely clear whether a bioreactor is fundamental for the valve repopulation. Lichtenberg and colleagues[5] clearly demonstrated that in vitro conditioning of engineered tissues in a bioreactive system stimulates and enhances the production of extracellular matrix and the related tissue strength. On the contrary, Dohmen and associates,[6] studying seeded or nonseeded decellularized valves implanted into right ventricular juvenile sheep outflow tracts, showed that a difference was identified in the recellularization density of in vitro seeded and nonseeded valves up to 3 months, but no such difference was seen after 6 months.

Nevertheless, a moderate pulsatile flow with small increments seems to be able to stimulate endothelial cell proliferation on the decellularized valve scaffold. A rapid increase in bioreactor flow to physiologic levels leads to significant damage of the reseeded endothelium and complete loss of cusp cellularity. This effect might be responsible for the in vivo failure of static, reseeded, tissue-engineered valves exposed to physiologic hemodynamic forces.

However, whether MSCs injected in the scaffold induced in situ differentiation into myofibroblasts and endothelial cells remains uncertain. The injection of the stem cells in the inner side of the valve before valve implantation must be able to produce a colonization not only by recipient endothelial cells but above all by active