The authors reported no conflict of interest.

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In another study of 606 patients who had total arch replacement in our institute,[3] risk factors for hospital death were older age, low preoperative estimated glomerular filtration rate, organ malperfusion, and longer cardiopulmonary bypass time. Risk factors for new stroke were severe white matter change seen in brain magnetic resonance imaging,[4] atherosclerotic shaggy aorta, and longer cardiopulmonary bypass time. Risk factors for late death were older age, low preoperative estimated glomerular filtration rate, need for concurrent procedures, permanent neurological deficit, need for tracheostomy, and postoperative acute kidney injury. Our study regarding the frailty assessment[5] demonstrated that preoperative psoas muscle area index was a good indicator of worse survival after total arch replacement.

Older age is a significant risk factor for early as well as late mortality in total arch replacement. However, postoperative adverse aortic events were very few after total arch replacement even in octogenarians and nonagenarians (Figure 1). We should not reject open surgery simply because of a patient's age and should consider frailty score, sarcopenia score, brain white matter change, or other comorbidities.

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PRIMARY ENTRY CLOSURE IS STILL FIRST-LINE TREATMENT FOR PATIENTS WITH DYNAMIC MALPERFUSION

To the Editor:

I read with great interest the article by Norton and colleagues,[1] which reported the efficacy of endovascular fenestration/stenting in patients with acute type B aortic dissection (ATBAD) with malperfusion. In their study cohort, only a limited number (4.9%) of ATBAD patients with malperfusion underwent thoracic endovascular aortic repair (TEVAR).[1] However, I believe that primary entry closure with TEVAR is the first-line treatment for malperfusion, especially due to dynamic occlusion, for the following 2 reasons.

First, in many cases of malperfusion due to dynamic obstruction, TEVAR could provide a quick release of malperfusion with a simple procedure. ATBAD patients with dynamic obstruction often show malperfusion of multiple vascular beds (mesenteric, renal, and iliofemoral), and therefore, rapid relief of malperfusion is critically important in these patients. In fact, 2 patients with malperfusion of multiple organs (case 8 and case 11) died from malperfusion syndrome with severe lower extremity ischemia, as shown in their Table E1.[1] Although the authors did not provide the procedure time data, it is important to examine the time to resolve malperfusion with their treatment strategy of endovascular fenestration/stenting. Furthermore, the reproducibility of their procedure in centers with less experience needs to be evaluated for their procedure to be accepted as the standard of care.

Second, because the endovascular fenestration/stenting does not close the primary entry, the false lumen pressure is not sufficiently reduced and the high false lumen pressure may lead to aortic rupture. As shown in their Table E1, half of the patients who died (cases 5-7, 9, 10, 12, and 14) died suddenly,[1] and it is estimated that aortic rupture occurred in the majority. Because dynamic obstruction is caused by increased false lumen pressure, ATBAD patients with dynamic obstruction are believed to be at higher risk for aortic rupture. Therefore, primary entry closure with TEVAR could lower the risk of aortic rupture by decreasing the false lumen pressure.

Of course, I agree with the authors' assertion that endovascular fenestration/stenting is recommended in ATBAD

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patients with static malperfusion. And if static obstruction remains after dynamic obstruction is resolved by TEVAR, endovascular fenestration/stenting would be necessary. However, because of the 2 concerns mentioned above, I believe that primary entry closure with TEVAR should be performed as first choice, especially in patients with dynamic obstruction. As pointed out by Formica and colleagues,[2] a distinction among patients with static or dynamic obstruction or both is not reported, so further research is needed to determine the optimal treatment strategy for ATBAD patients with dynamic obstruction.

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REPLY: STENTING/FENESTRATION OR THORACIC ENDOVASCULAR AORTIC REPAIR IN COMPLICATED ACUTE TYPE B AORTIC DISSECTION: TO EACH IS OWN!

Reply to the Editor:

In complicated acute type B aortic dissection (ATBAD), urgent intervention is the crucial point to correct mesenteric and renal ischemia and to re-establish distal perfusion. Consequentially, the time between the diagnosis and the therapeutic choice, endovascular stenting versus fenestration, or both, are strongly related.

Despite the reasons given by Norton and colleagues[1] for the use of the fenestration technique, thoracic endovascular aortic repair (TEVAR) remains the main technical approach in many centers, even those with a high volume of interventions.

Therefore, what role can fenestration play in the TEVAR era? The rationale of fenestration is to restore the normal pressure in the false lumen, with easier branch vessel patency recovery, as long as there is a careful demonstration of the vessel anatomy and good technical skills.

The frequency with fenestration is required is still widely debated. The majority (>80%) of malperfusion syndrome occurrences are due to dynamic obstruction.[2] In the setting of acute dysfunction, distinguishing between dynamic and static obstruction is critical to successful treatment of malperfusion due to branch vessel compromise. Frequently the clinical scenario is more complex and nuances can exist.

We think that statements by Ueki[3] in his letter regarding the strategy of primary entry closure as first line therapy by TEVAR mainly in dynamic obstruction, and that stenting/fenestration does not close the entry tear and maintain a pressurized false lumen are absolute sharable. Unlike fenestration, TEVAR is a simple and reproducible procedure that requires a shorter learning curve. Stenting/fenestration is more demanding than TEVAR and may need to be performed in experienced referral centers.[4] Fenestration may be worthwhile when the placement of a stent or stent-graft close to the entry site is not possible, the aortic lumen is too large and appropriate stent-grafts are not readily available, the tear is too close to major branch vessels, or a high-flow endoleak leaves the false lumen pressurized.

Data from the International Registry of Acute Aortic Dissection reveal that descending aortic size, false lumen patency/thrombosis, as well as the size of entry tears are important predictors of adverse events in patients with ATBAD.[5] Moreover, clinical stability does not exclude the possibility of false lumen silent expansion and even rupture.[6] All of these risk factors mainly rely on static imaging rather than hemodynamic features.

Interesting data are emerging regarding the concept that hemodynamic stress may be the primary cause of false lumen enlargement. For example, studies propose analyzing the entry tear in ATBAD and the imbalance between false lumen inflow and outflow pathways by means of 4-dimensional technologies.[7,8]

This is the new direction to aim for to improve the characterization and risk-stratification of patients with ATBAD to correctly select those patients who may benefit from stenting/fenestration, which remains a procedure that should be employed by an experienced team.

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