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INVITED COMMENTARY

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In the 20 years since we described the importance of malperfusion syndrome (MPS) to outcomes in aortic dissection,[1] considerable progress in understanding its pathogenesis, as described by Williams et al,[2] and treatment have occurred. There is consensus that MPS complicates aortic dissection in a third of patients and, in addition to aortic rupture, is a principle cause of death, in particular when carotid/mesenteric and renal arteries are affected. This consensus dissipates because the heterogeneity of pathologic anatomy (static vs dynamic mechanisms, type A or B lesion, acute or chronic circumstance), clinical presentations, and therapeutic capabilities of treating surgeons/interventionalists greatly influence both clinical decision making and results of treatment.

Numerous important clinical data are either first described or re-emphasized by this prodigious experience from the Michigan

group. These include the discrepant pathologic anatomy of right vs left renal artery, a perhaps higher than anticipated incidence of static obstruction (ie, the dissecting process proceeds into the branch vessel itself), the fact that persistent MPS either in the chronic phase of the disease or after surgical central aortic repair does occur, and the "yield" of renal MPS when suspected by clinical or radiographic findings.

Two points are worthy of emphasis with respect to the latter: (1) the authors use aortic root-to-branch pressure gradients as the gold standard of defining MPS, and (2) there is little consideration (ie, not in their data set) of the great importance of axial imaging, such as by computed tomography (CT) scans, in both the suspicion for, and diagnosis of, MPS. Indeed, in our practice—and I strongly suspect this is generally true—it is the anatomy derived from three-dimensional CT scanning that both directs clinical decision making and forms the fundamental and critical initial step of understanding the pathologic anatomy.

Any intervention, be it open surgical, endovascular, thoracic endovascular aneurysm repair (TEVAR), or a combination of these, is predicated on such understanding, which also forms the basis of the intraprocedural intravascular ultrasound (IVUS) interrogation. I certainly agree with the authors that IVUS is an essential component of any endovascular procedure for aortic dissection; thus, catheter-based angiography is rarely used in a strictly diagnostic mode in aortic dissection patients. Rather, it may be a component of a comprehensive endovascular approach to MPS.

Although the authors emphasize the efficacy of their approach, the mortality at 21% is substantial. This rarely represents technical failure or complication of the endovascular therapy, but rather, the additive effects of other affected vascular beds (particularly mesenteric ischemia) and delays in diagnosis and treatment of MPS. Perhaps not appropriately emphasized in their report is that TEVAR technology can and will change the treatment of MPS, at least in patients with type B aortic dissection. The ability to seal an aortic entry tear, redirect flow into the aortic true lumen, and thereby relieve MPS—at least wherein dynamic obstruction is the mechanism—has been an important advance in type B aortic dissection. Certainly, aortic branch orificial stenting may also be required, especially in static obstruction. Clinical trials of dissection-specific TEVAR devices for complicated type B cases have begun.

Finally, such TEVAR approaches have a distinct potential advantage compared with the techniques described in this report. I refer, of course, to the anticipated aortic remodeling that likely will occur after TEVAR obliteration of the proximal entry tear, thereby obviating chronic aneurysmal dilatation of the outer wall of the false lumen and the need for late aortic replacement, which currently affects at least 40% of acute dissection patients irrespective of initial treatment. This is a timely and important contribution as management of MPS evolves in the TEVAR era.

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