Unmet Needs in…
Thoracic Anatomy

Future innovations for thoracic aortic care include technical solutions for common or critical complications and devices that address specific anatomic challenges.

With Tara M. Mastracci, MD, FRCSC; Bijan Modarai, PhD, FRCS; and Chandler A. Long, MD

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The thoracic aorta is one of nature's (and the aortic surgeon's) greatest enigmas. Although it looks like an uncomplicated straight conduit with a main function of ensuring the lower, more interesting visceral and pelvic territories are well perfused, it poses several challenges for endovascular repair. It's hard to levy judgment on the relative importance of each challenge that the thoracic aorta poses, but I offer the following as food for thought.

In my opinion, the proximal boundary of the thoracic aorta remains its largest hurdle. Both in dissection and aneurysm disease, a small segment of normal-caliber aorta distal to the subclavian artery often serves as a tantalizing landing zone, offering false hope of durable repair. As with its familiar cousin, the infrarenal, we are learning with more experience and time that the durability of short proximal landing zones in the thoracic aorta can be poor.[1-3] In aneurysm, this often means conversion to open arch surgery to rectify the indiscretion. But in dissection, there is a greater risk of retrograde dissection amplifying acute problems. As prevalent a challenge as this may be, the solution may not be simple. Certainly, the move toward conformability has resulted in improvements in stent placement in shorter target landing zones. I also believe the iterative improvements in delivery systems are largely unlauded in their contribution to improve accuracy of deployment.

Another important anatomic challenge is access. Especially in women with thoracoabdominal aneurysms, small iliac access remains a problem for patients who require large-diameter devices. Although our industry partners have worked hard to decrease the profile of thoracic devices, more work can be done to provide a solution that accommodates even the most heavily diseased access. As we drive the device size down, the quality of graft materials cannot be sacrificed for gains in packing density. The low-profile fabrics on the market now seem acceptable, but only time will provide the needed test to determine if they are as durable as earlier-generation fabrics.

Tortuosity is another frequently found challenge, as lengthening of the aorta leads to acute angulations that can make mating of stent components a challenge, as well as lead to kinking that can impact flow. This sometimes also complicates delivery system removal and overall deployment of devices. It will be interesting to see how device engineers adjust the length and flexibility of devices as longer-term data emerge from highly tortuous aortas.

The final hurdle I'd point to in the anatomic battle of the thoracic aorta is intercostal blood flow and the accidental or intentional occlusion of intercostal arteries. Although experts put different emphasis on the relative importance of occlusion versus embolization of intercostals as a predictive factor for spinal ischemia, there is no doubt that this feared complication requires device manufacturers' future attention. A device that could magically maintain intercostal flow and protect the paraspinal network from accidental embolization would be a game changer.

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Endovascular repair is now the first-line treatment for the majority of patients with thoracic aortic pathologies, with iterations in endograft design allowing safer device implantation and improving the durability of repair. However, predicting and eliminating the risk of paraplegia consequent to a critical reduction in spinal cord blood flow after thoracic endografting remains a significant challenge. The adoption of preventative measures such as permissive hypertension, maintenance of hemoglobin concentration, and, where feasible, preservation of the collateral blood supply to the spinal cord has reduced paraplegia rates. Nevertheless, this complication, often occurring sporadically, still persists as the most feared eventuality for both the patient and operator.

Currently, the preoperative assessment of paraplegia risk and the counseling of patients are largely based on low-level evidence and the operator's anecdotal experience. The detailed imaging of the entire aortic tree carried out prior to intervention provides a hitherto-missed opportunity for objective, personalized evaluation of the patient's anatomy and the consequences of any particular configuration of repair.

For a patient presenting with a type B aortic dissection (TBAD), for example, could we objectively determine the minimal length of coverage in the descending thoracic aorta that would be required to promote maximal aortic remodeling while avoiding paraplegia? A truly predictive tool would use an automated, multifactorial algorithm, determining the independent and multivariate effect of any given anatomic and physiologic risk factor and relating this to the proposed intervention. It would, of course, require validation using a large volume of clinical data, especially accurately recorded postoperative outcomes. Such a tool would also objectively highlight instances in which there is an absolute requirement for using all means possible to preserve collateral spinal cord flow after placement of a thoracic endograft.

Considering the left subclavian artery (LSA), an important collateral blood supply to the spinal cord that is often involved in the proximal landing zone of a thoracic repair, our mindset has already shifted toward aggressive preservation. Routine preservation of this vessel will be further facilitated by emerging off-the-shelf branched thoracic stent grafts that will provide an endovascular means of incorporating the LSA during thoracic endovascular aortic repair (TEVAR) in acute as well as elective scenarios. However, a reliable endovascular solution is yet to be found for maintaining flow to the intercostal arteries, despite the fact that occlusion of vessels such as the artery of Adamkiewicz significantly increases the risk of producing paraplegia. These vessels can be reimplanted during open repair, and even though there is uncertainty as to how long these reimplanted vessels remain patent, even transient maintenance of perfusion may be sufficient to prevent acute spinal cord ischemia at the index repair. A reliable, off-the-shelf endovascular solution that allows preservation of flow to critical vessels arising directly from the thoracic aorta, even on a temporary basis, would be a significant conceptual advance.

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Acute dissections of the descending thoracic aorta are associated with significant morbidity and a mortality rate potentially as high as 25% at 30 days.[1,2] Those fortunate enough to convalesce through the acute phase and transition to a chronic TBAD (cTBAD) still have a 20% to 40% rate of false lumen (FL) expansion and thus aneurysmal dilatation.[2-4] Open surgical repair of this pathology portends a combined morbidity and mortality of almost 30%, which is why TEVAR has become the preferred means of treatment.

The goal of endovascular repair is to cover the primary entry tear and prevent flow from entering the FL. This allows for thrombosis and shrinking of the FL and expansion of the true lumen, without the overall diameter of

the aorta increasing. This process is considered positive or reverse remodeling and is associated with favorable long-term outcomes. Up to 30% of patients treated with endovascular repair for cTBAD have reentry tears distal to the extent of the thoracic endograft that preserve flow and patency of the FL, thus preventing the desired remodeling.[3-7] This has been the Achilles' heel of endovascular treatment for cTBADs, and remedying this issue would improve long-term survival.

Currently in the United States, no commercially available device exists to treat persistent flow in the FL for cTBAD patients. Several novel techniques have been described to aid in thrombosis or collapse of the FL after placement of, or in conjunction with, TEVAR. Examples include balloon-assisted fracture/rupture of the dissection flap or FL thrombosis by the placement of a modified candy-plug device. Both techniques have literature to support their successful results, and yet limitations persist. For example, with balloon-assisted rupture of the dissection flap, the behavior of the unprotected aorta in and distal to the visceral segment can be unpredictable and result in dissection propagation, malperfusion, or even rupture. Although I am a proponent of the candy-plug technique, even if the device works as intended and results in thrombosis of the FL, a foreign body (stent graft) remains in the FL. The result is the continued exertion of radial forces that prevent local collapse and/or shrinking of the FL, thus limiting local positive remodeling of the aorta.

If I had an "easy button" to solve this problem, it would be in the form of a bioabsorbable candy-plug–like device. The device would need to contain the appropriate amount of radial force so as to maintain the intended positioning in the FL and resist displacement from flow. Additionally, it should encompass characteristics that would promote thrombosis of the FL and then subsequently be reabsorbed by the body. The basic tenets of this new device would be the same but only for a transitory period, ultimately allowing for complete remodeling of the aorta. Given that bioabsorbable implantable device technology is being explored with inferior vena cava filters and coronary stents, I do not believe this notion is far-fetched or that this technology is beyond our reach. If a device does become available in the United States market for the treatment of FL flow, I believe it will be used frequently and add significant value to the care of patients with cTBADs. ■

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