Flow dynamics, false lumens and implications for endografting

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The complications of endovascular stenting for type B aortic dissections are well-described and include paralysis from spinal cord ischemia, retrograde dissection, stroke, and access site complications. Owing to the inherent risk associated with this procedure, current practice is to withhold endovascular therapy unless the risk-benefit balance is in favor of intervention, such as with acute malperfusion or aneurysmal degeneration. Emphasis is placed on coverage of the entry tear, which has been advocated theoretically as the primary method to restore normal aortic hemodynamics and induce false lumen thrombosis, despite notable paucity of direct flow modeling to support this claim. Little is known about the optimal strategy to deal with the common clinical scenario of multiple fenestrations distal to the initial entry tear.

In the study "Endograft exclusion of the false lumen restores local hemodynamics in a model of type B aortic dissection" Drs Birjinuik, Veeraswamy, and colleagues[1] use an ex vivo silicone model of aortic dissection to examine the impact of aortic endografting on flow dynamics along the contour of a dissected aorta with two additional distal fenestrations. The authors found that, when there was a flap partially covered by an endograft, the flow velocity within the false lumen was low, indicative of a blind pouch and predisposed for thrombosis based upon elevated oscillatory shear. Interestingly, in the regions with uncovered distal fenestration, flow velocities and shear rates in the false lumen were unaffected by the presence of a more proximal graft, theoretically leading to preservation of flow in the false lumen. The authors conclude that entry site coverage may be insufficient in restoring normal aortic hemodynamics. Several different methods of alternate endografting are reviewed, including extended stenting, alternating stents and the PETTICOAT method of covered graft over the entry tear, and bare metal stent over a long segment of aorta, based on the premise that closing the distal fenestration induces false lumen thrombosis and ultimately promotes positive aortic wall remodeling.

Several publications have recently discussed the array of complex biomechanical factors determining the fate of endovascular stenting for type B aortic dissections that bear mention when putting the data from this highlighted article into perspective.[2,3] Indeed, the combination of parameters such as acute versus chronic stage critically alter the mechanical properties of the intimal flap, aortic tortuosity, and number and location of entry tears, hypertension, cardiac output, and degree of stent oversizing all impact the hemodynamics in the true and false lumen. Experimental studies such as those presented by Drs Birjinuik, Veeraswamy, and colleagues are valuable to test in a controlled manner the comparative performance of different methods of alternate endografting to increase our intuition for the behavior of such a complex system through flow phantoms and imaging. Restrictions associated with such models include the impact of numerous branches (visceral, renal, and intercostal arteries) and other anatomic components with differential flow and pressure conditions, which are difficult to replicate in a flow phantom. The model used in this study is effectively a one-inlet one-outlet tube, and conditions in multibranched models and in vivo are likely more complex than those presented here.

To truly understand aortic wall degeneration and aneurysmal growth, it is critical to include a parameter not easily addressable through medical imaging technics: pressure. Fundamentally, blood pressure is the driver of aortic growth and a key determinant of rupture risk. A velocity-centric description of the hemodynamics is therefore only one component to understanding the whole picture. Computational methods can be combined with imaging to obtain noninvasive descriptions of the pressure field, either through direct computation of total pressure or reconstruction of relative pressures from the magnetic resonance-derived velocity data.[4,5] Thus, the logical next step from the provocative data presented in the highlighted article is to build on this knowledge with a more sophisticated understanding of the impact of branch vessel flow dynamics and pressure.

Ultimately, two major unanswered areas in the arena type B aortic endografting remain: (1) how to identify patients with uncomplicated type B aortic dissection who would benefit from early endografting owing to unfavorably aortic hemodynamics and risk of subsequent aneurysmal degeneration, and (2) how to optimally treat distal fenestrations

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Author conflict of interest: none.

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

J Vasc Surg 2020;71:2119-20

0741-5214

Published by Elsevier Inc. on behalf of the Society for Vascular Surgery.

https://doi.org/10.1016/j.jvs.2019.06.223

while mitigating the concomitant risk of spinal cord ischemia? There is therefore a pressing need to further our understanding of the complex hemodynamics in type B aortic dissection repair. This can only be achieved through multidisciplinary research involving outcomes, imaging, and experimental and computational methods.

The opinions or views expressed in this commentary are those of the authors and do not necessarily reflect the opinions or recommendations of the Journal of Vascular Surgery or the Society for Vascular Surgery.

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