The sheer stress of deciding when to replace the aorta in bicuspid valve disease

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Disclosures: Authors have nothing to disclose with regard to commercial support.

Received for publication Feb 21, 2017; accepted for publication March 3, 2017; available ahead of print April 1, 2017.

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J Thorac Cardiovasc Surg 2017;153:1273-4

0022-5223/\$36.00

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<http://dx.doi.org/10.1016/j.jtcvs.2017.03.018>

In this issue of the *Journal*, Shan and co-authors¹ provide us with a 4-dimensional (4D) flow magnetic resonance imaging (MRI) analysis of 15 normal trileaflet aortic valve controls compared with 50 age-matched patients with bicuspid aortic valve (BAV), right-left cusp fusion, mild aortic valve dysfunction, severe aortic insufficiency (AI), or severe aortic stenosis (AS). The focus of this study was to determine the impact of valve pathology on wall shear stress (WSS) in patients with BAV. The hypothesis was that MRI-derived metrics may establish noninvasive indicators to inform the risk stratification and treatment of these patients even when aortic size may be borderline.

Ascending aortic dilatation, aneurysm, and dissection comprise the heterogeneous expression of BAV aortopathy.^{2,3} The pathogenesis is complex. Two major causes have been proposed: (1) genetic: Although causative genes remain largely undetermined, familial clustering of BAV suggests a heritable cause⁴; (2) hemodynamic: There is sufficient evidence that cusp fusion in patients with BAV leads to abnormal aortic WSS in the ascending aorta, which stimulates vascular remodeling.^{5,6}

Contemporary advances in full-volume, flow-sensitive 4D MRI outlined in the current study¹ permit detailed assessment of aortic blood flow patterns and quantification of WSS in the entire aorta. The principle findings of this study were as follows: (1) Peak WSS in patients with BAV occurs similarly in the right anterior aorta irrespective of aortic valve function; (2) patients with BAV without valve dysfunction have higher WSS compared with tricuspid aortic valve controls; and (3) BAV dysfunction (AI and AS) results in distinct but different hemodynamic effects in the ascending aorta. In patients with BAV, AI resulted in a universal WSS increase around the ascending aorta as a result of the increased cardiac output, whereas AS resulted in focal increase in WSS as a result of the exaggerated eccentricity of aortic outflow. These 2 distinct effects could have different long-term consequences in the affected aortic wall tissue; therefore, patients with BAV-related AS and AI might need to be approached differently. Thus, the configuration

Sheer stress.

Central Message

WSS from 4D MRI can detect subtleties in BAV aortopathy that may inform surgical decision making by predicting risk before aortic size does.

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of aortic valve leaflet repair or replacement could influence subsequent aortic outcomes. Although the surgical goal in both is smooth central laminar flow, 2-leaflet reconstructions for AI might emphasize achieving 180° commissural geometry, whereas components of AS associated with 3 equal sinuses are better managed with trileaflet replacement.⁷

Current guidelines on the timing of prophylactic aortic replacement in patients with BAV have mainly focused on the aortic size and the rate of aortic aneurysm expansion.^{8,9} However, Shan and co-authors¹ add to the growing body of important work delineating that different BAV aortopathy phenotypes may be directly related to specific hemodynamic effects of valve pathology (AI vs AS).¹⁰

To translate these important findings from the bench to the bedside, future studies need to be designed to address the following issues: (1) identify WSS patterns that are associated with the highest risk of rapid aneurysmal dilatation and aortic dissection; (2) establish reference WSS values that can predict high-risk patients, in whom an intervention would be warranted even in the ascending aorta measures less than 5.5 cm; (3) provide a simplified standard protocol for 4D MRI and possibly investigate whether a computed tomography angiography equivalent can be developed given the limited availability of 4D MRI in clinical practice; and (4) relate repair configuration to late aortic stability.

The distinct hemodynamic effects of BAV pathology on the ascending aorta may soon add to the established anatomic criteria to facilitate more individualized patient risk stratification and surgical intervention in BAV aortopathy.

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