Assessing wall stresses in bicuspid aortic valve-associated aortopathy: Forecasting the perfect storm?

Alex J. Barker, PhD,^a Michael Markl, PhD,^{a,b} and Paul W. M. Fedak, MD, PhD, FRCSC^{c,d}

From the ^aDepartment of Radiology, Feinberg School of Medicine, and ^bDepartment of Biomedical Engineering, McCormick School of Engineering, Northwestern University, Chicago, Ill; ^cDivision of Cardiac Surgery, Bluhm Cardiovascular Institute, Northwestern Memorial Hospital, Chicago, Ill; and ^dDepartment of Cardiac Sciences, Libin Cardiovascular Institute of Alberta, University of Calgary, Calgary, Alberta, Canada.

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Address for reprints: Paul W. M. Fedak, MD, PhD, FRCSC, Department of Cardiac Sciences, Libin Cardiovascular Institute of Alberta, University of Calgary, Rm 880, 1403-29 St, NW, Calgary, Alberta, Canada T2N 2T9 (E-mail: paul.fedak@gmail.com).

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Alex J. Barker, PhD, Michael Markl, PhD, and Paul W. M. Fedak, MD, PhD, FRCSC

Central Message

The colocation of abnormal WSS regions with high wall stress may create perfect storm conditions by exposing already weakened aortic regions to high wall stress that leads to wall failure.

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Prophylactic aortic resection is recommended when the ascending aorta reaches specific size thresholds.¹ When deciding to resect an aorta with bicuspid valve disease, surgeons remain challenged by the so-called size paradox—the arterial wall can fail at smaller aortic sizes than the recommended guidelines for resection.^{2,3} Aortic rupture or dissection occurs when blood pressure-induced forces (stress) exceed the biomechanical strength of the aortic wall. Emerging research has focused on 2 important biomechanical concepts that may drive aortic wall integrity and ultimate failure of the aorta: wall stress and wall shear stress (WSS). Despite similar names, these 2 metrics are very different biomechanical stressors that influence aortopathy for different reasons. In comparison to wall stress, WSS is a measure of the frictional force caused by blood flowing along the vessel wall and is expressed at orders of magnitude lower than wall stresses. Nonetheless, via mechanotransduction and signaling pathways, WSS alters vessel wall extracellular matrix integrity and biomechanical strength.⁴ For example, elevated WSS is associated with altered matrix protein concentration and elastic fiber degeneration in the ascending aorta of patients with a bicuspid aortic valve (BAV).⁵ In contrast, wall stress represents the internal forces imposed on the aortic wall, which will fail when wall stress exceeds the regional biomechanical strength.⁶

Xuan and colleagues⁷ investigated regional wall stress in patient-specific models of ascending aortic aneurysms to understand whether a predisposition for wall failure exists in BAV. They hypothesize that wall stress will predict vessel wall rupture or dissection. Indeed, the models found that patients with BAV were exposed to higher wall stresses than patients with a tricuspid aortic valve, and these stresses were more pronounced at the sinotubular junction. Moreover, wall stresses were poorly correlated to diameter in patients

with BAV, further suggesting that diameter alone may not adequately predict rupture. These important patient-specific results support the use of personalized risk stratification. However, additional due diligence with more sophisticated models is necessary. For example, as Xuan and colleagues⁷

FIGURE 1. A, Mechanistic hypothesis for wall shear stress (WSS)-mediated regional vessel remodeling.⁹ B, Mechanistic hypothesis for stress-mediated regional failure. The co-location of abnormal WSS regions with high wall stress may create perfect storm conditions by exposing already weakened aortic regions to high wall stress that leads to wall failure.

rightly point out, their model does not account for heterogeneous regional vessel properties (eg, architecture, wall strength, and wall thickness), which are known to occur in patients with BAV.^{5,8} Admittedly, it is difficult to predict mechanical properties noninvasively and no correlation with the tissue itself was found in their study.

The findings of the study prompt one to speculate whether the combination of the 2 biomechanical concepts; that is, regional biologic dysfunction driven by WSS-mediated mechanotransduction (Figure 1, A) and regionally variable biomechanical stresses (Figure 1, B), implies that it may be possible to predict the perfect storm of impaired vessel wall biomechanics. Patients exposed to regionally abnormal WSS over time may have accelerated vessel wall degeneration, which may be co-localized with a region exposed to high wall stress (resulting in rupture or dissection). The possibility of predicting abnormal vessel wall properties that align with high wall stresses may forecast the beginnings of a perfect storm capable of catastrophic damage.

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