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INVITED COMMENTARY

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In the current manuscript, the authors present data demonstrating an association between dilated aortic root (DAR) and concomitant increased distal aortic segment diameters. The pattern of more diffuse disease being associated with tricuspid aortic valve, as opposed to bicuspid aortic valve (BAV), is somewhat surprising, but likely highlights one of the limitations of this analysis in that it represents a small sample size – only 19 patients had BAV. Certainly a larger population study could demonstrate a stronger association. Another weakness of this study is that it represents a single snapshot in time. While DAR is associated with concurrent increased distal aortic diameters, it is unclear as to what the natural history of this association will be. Will further segments of the aorta degenerate, and will areas that are dilated, but not aneurysmal, become so over time? The authors suggest that it may be necessary to screen patients with DAR for abdominal aortic aneurysm (AAA). Perhaps with more data obtained over time we may find it is necessary to continue to monitor the entire aorta of these patients as they age.

The data presented supports the concept that aortic aneurysmal disease (AAD) is associated with a diffuse aortic pathology. The population is aging and patients are surviving longer after surgery for aneurysmal disease. It is becoming increasingly apparent that patients with AAD at one location may develop subsequent aneurysmal degeneration at sites either contiguous with, or remote from, the site of initial pathology. This suggests that patients with AAD have an inherently abnormal aorta that is at risk for further aneurysmal degeneration. Data from the current manuscript lend further support to this hypothesis. Knowing this, future surgical approaches to patients with AAD may need to be viewed as palliative and no longer curative. Operative plans may need to be

developed realizing that radiologically normal appearing aortic segments are actually at risk for subsequent degeneration and ultimate failure of that repair.

We have an increasing understanding of the molecular mechanisms that contribute to AAD. We lack significant knowledge, however, on what baseline abnormalities place certain aortas at risk for future aneurysmal degeneration, particularly in patients without a known connective tissue disorder. In addition, we have a poor understanding of what events trigger the process of aortic degeneration. It is this fundamental knowledge deficit that hinders our ability to better explain the correlations outlined in the current manuscript, or that explain the development of subsequent aneurysmal degeneration that we are identifying in our patients. We must continue to focus efforts on improving our understanding of the genetic and molecular events associated with AAD. This is accomplished through population studies, genetic analyses, and translational and basic research evaluating the mechanisms of aneurysm formation. Only with this knowledge will we better identify patients at risk for further aneurysmal degeneration, or develop technologies to assess in vivo aortic integrity to assure adequate long-term repair and survival from AAD.