

Abstract Submitted  
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**Deformation of Congenital Bicuspid Aortic Valves in Systole** KAI SZETO, MAE UCSD, PETER PASTUSZKO, Rady Children's Hospital; Department of Surgery UCSD, VISHAL NIGAM, Rady Children's Hospital; Department of Pediatrics UCSD, JUAN LASHERAS, Departments of MAE and Bioengineering, UCSD — Clinical studies have shown that patients with congenital bicuspid aortic valves (CBAVs) develop degenerative calcification of the leaflets at young ages compared to normal tricuspid aortic valves (TAVs). It has been hypothesized that the asymmetrical geometry of the leaflets in CBAVs and the associated changes in flow shear stresses and excessive strain rate levels are possible causes for the early calcification. Central to the validation of this hypothesis is the need to quantify the differences in strain rate levels between the BAVs and TAVs. We simulate the CBAVs by surgically stitching two of the leaflets of a porcine aortic valve together. To quantify strain differences, we performed in-vitro experiments in both BAVs and TAVs by tracking the 3-D motion of small dots marked on each leaflet surface. We then used phase-locked stereo photogrammetry to measure the strain rates in both radial and circumferential directions during the whole cardiac cycle. In the BAVs' case, the fused leaflet experiences an almost 30% increase in the radial stretching when fully open. RNA profiling of human aortic valve interstitial cells exposed to cyclic stretch shows that the increased stretch experienced by the BAVs results in increased levels of INTERLEUKINS (ILs) and other known inflammatory markers associated with aortic valve calcification. Together, these observations suggest that the abnormal stretch experienced by BAVs activates inflammation gene expression.

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