To the Editor:

I read with great interest a recent publication by Dr Bissell et al.1 This report represents a nice continuum of several recent in vivo MR studies in patients with bicuspid aortic valve published during the past 2 years.2,3 In the present study, the authors addressed some controversial aspects of bicuspid aortopathy, in particular impact of R-N BA V cusps fusion pattern on transvalvular flow characteristics, as well as specification of transvalvular flow in the pediatric BA V population (ie, without clinically apparent aortopathy or aortic valve dysfunction). The authors should be congratulated for this excellent contribution and their continuous efforts in defining the role of altered hemodynamics in the development of BAV aortopathy.

A large number of novel functional parameters have been introduced by cardiovascular MRI, which should be thoroughly evaluated and integrated into the clinical perspective. My comments and questions are intended only to add on some relevant details of this well-performed study:

1. What may be your explanation for the finding of different hemodynamic flow abnormalities in patients with BA V with the same cusp fusion pattern (ie, right-handed flow versus left-handed flow in patients with the R-N BA V cusp fusion)? Are the authors aware of any other structural valve characteristics that may influence the propagation pattern of systolic transvalvular flow? In our recent prospective analysis, which was initially designed to correlate transvalvular flow patterns with the histological lesions in the ascending aorta in BA V disease, we found that propagation of systolic flow jet was dependent on the exact anatomic localization of residual valve orifice (especially in the stenotic morphology) and the specific angulation between left ventricular outflow tract and proximal aortic axis.4

2. Is it conceivable, at least theoretically, that transvalvular rotational flow pattern is not constant and may change during the evolution of BA V disease (eg, right-handed flow degenerates into left-handed or complex flow during the course of disease)?

3. In the light of previous echocardiographic reports,5 how could the authors interpret the fact that vascular function parameters were not influenced by the presence of BAV and transvalvular flow pattern?

4. Did the authors encounter a subgroup of BAV patients with an isolated/predominant aortic valve insufficiency? What about transvalvular flow patterns, distribution of wall shear stress in the proximal aorta in this specific subgroup of BAV patients?

Once again, I congratulate the authors for this important contribution to the field of BA V aortopathy.

Disclosures

None.

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References


Letter by Girdauskas Regarding Article, "Aortic Dilation in Bicuspid Aortic Valve Disease: Flow Pattern Is a Major Contributor and Differs With Valve Fusion Type"
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Circ Cardiovasc Imaging. 2014;7:213
doi: 10.1161/CIRCIMAGING.113.001476
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/7/1/213

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