

and inferences should be drawn with caution. Substantial selection and observer bias, inadequate sample size, lack of repeatable disease definition, and questionable validity of the outcome assessment tool are the major factors hampering the study's clinical implications. A good-quality survey is required to estimate the burden of DVST in high-risk patients with acute blunt head trauma, along with associated factors to propose a particular imaging algorithm.

Reference

1. Delgado Almandoz JE, Kelly HR, Schaefer PW, Lev MH, Gonzalez RG, Romero JM. Prevalence of traumatic dural venous sinus thrombosis in high-risk acute blunt head trauma patients evaluated with multidetector CT venography. *Radiology* 2010;255(2):570-577.

Response

From

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We have read Dr Akhtar's letter regarding our recent *Radiology* article (1) and hereby provide our reply. At our institution, all blunt head trauma patients with skull fractures in the vicinity of a dural venous sinus are screened with computed tomographic venography to exclude traumatic DVST. Hence, we believe that the reported frequency of traumatic DVST in our study does reflect the prevalence of this disease entity in our specific high-risk patient population. Nevertheless, we recognize that our findings may not be generalizable to other patient populations. The majority of Dr Akhtar's comments regarding the lim-

itation of our retrospective study have already been acknowledged in the article. However, we would like to note that (a) to date, our study constitutes the largest published patient series on this topic; (b) we used a precise imaging definition for both nonocclusive and occlusive DVST; and (c) screening all of these patients with the reference standard of conventional angiography would be imprudent.

References

1. Delgado Almandoz JE, Kelly HR, Schaefer PW, Lev MH, Gonzalez RG, Romero JM. Prevalence of traumatic dural venous sinus thrombosis in high-risk acute blunt head trauma patients evaluated with multidetector CT venography. *Radiology* 2010;255(2):570-577.

Aortopathy in Bicuspid Aortic Valve Disease: Is It Really Congenital?

From

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Editor:

Dr Hope and colleagues have to be congratulated on their important contribution (1) to the field of bicuspid aortic valve (BAV) disease, which appeared in the April 2010 issue of *Radiology*. There is an ongoing debate regarding the origin of BAV aortopathy. The strong argument in mainstream genetic theory has been the high prevalence of proximal aortic dilation in patients with a BAV, which is out of proportion to the coexistent valvular lesions.

The most important message of this manuscript is the evidence of abnormal flow patterns in the ascending aorta in patients with a BAV and without substantial valvular lesions. The authors used sophisticated magnetic resonance (MR)

imaging to demonstrate abnormal in vivo hemodynamics as a consequence of the bicuspid nature of aortic valve. This supports the extensive experimental work done by Robicsek et al (2). Moreover, Dr Hope and colleagues found two different flow patterns, which are specific to the two most common cusp fusion types in patients with a BAV. This suggests a hemodynamic origin of the recently identified BAV phenotypes (3-5). Fusion of the right and left coronary cusps generates a right-anterior flow jet that might result in larger aortic root dimensions (3,4) and asymmetric dilatation of the ascending aorta (6). A left-posterior flow jet, which is seen in patients with right-noncoronary cusp fusion, might explain the increased aortic arch dimensions in this subgroup of patients with a BAV (3,6). This heterogeneity of concomitant aortic pathologic findings in BAV disease would be difficult to explain with a pure genetic theory.

The article by Dr Hope and colleagues gives rise to some intriguing questions: Is there any explanation for the normal flow pattern in five patients with a BAV? Are there any other distinctive characteristic in these five patients with a BAV, apart from milder aortic valve disease? Did the authors look for the differences in aortic dilatation pattern between the two cusp fusion types (ie, right-left vs right-noncoronary)? Finally, could MR imaging be of value in demonstrating the distribution of aortic wall stress, which might be expected to differ between the two cusp fusion types?

In summary, this article provides valuable data in favor of a hemodynamic origin of BAV aortopathy. As the debate is ongoing, future research should focus on the detailed analysis of BAV phenotypes.

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3. Schaefer BM, Lewin MB, Stout KK, et al. The bicuspid aortic valve: an integrated phenotypic classification of leaflet morphology and aortic root shape. *Heart* 2008;94(12):1634-1638.
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Response

From

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We are encouraged by the insightful response by Dr Girdauskas and colleagues to our article (1) and agree that our work raises intriguing questions regarding subgroups of patients with a BAV.

Patients with a BAV and normal aortic flow, if indeed there is merit to the hemodynamic theory for proximal aortic dilation, would have fewer complications and require less follow-up. The normal flow pattern is the consequence of a central systolic flow jet (see figure 2 in our article), which one would expect to see with relatively equal size of aortic leaflets, although we did not study this directly. Based on the extensive review of BAV morphologies by Sabet et al (2), the vast majority (92%) of BAVs have neither symmetric nor markedly asymmetric aortic leaflets, and flow analysis may better characterize this large middle ground.

We, too, have reviewed with great interest the work of Schaefer et al and others regarding differences in aortic dilation patterns between right-left and right-noncoronary aortic leaflet fusions (3,4). We are currently collecting MR data to evaluate the differences shown in these large echocardiography studies and have observed, as have others (5,6), disproportionate dilation of the right-anterior aspect of the proximal ascending aorta, which we have seen with right-left fusion.

MR imaging is singularly capable of assessing vessel wall shear stress from near wall velocity gradients. We have used software developed by Stalder et al (7) to study wall shear stress in patients with a BAV. Preliminary data presented at the International Society of Magnetic Resonance in Medicine (8) show an asymmetric distribution of wall shear stress in a subgroup of patients with a BAV, which is particularly noteworthy in the context of the work of Della Corte et al (9) that demonstrates asymmetric aortic smooth muscle cell apoptosis that may be flow mediated.

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Errata

“Intermittent Claudication: Clinical Effectiveness of Endovascular Revascularization versus Supervised Hospital-based Exercise Training—Randomized Controlled Trial.” *Radiology* 2009;250(2):586-595

Page 592, Table 4, “Measure of functional capacity” column, “maximum walking distance” row, the adjusted *P* value for the “6 months” row should read **.001** and that for the “12 months” row should read **.03, in favor of supervised exercise (significance level of .01).**

“T2 Mapping in Duchenne Muscular Dystrophy: Distribution of Disease Activity and Correlation with Clinical Assessments.” *Radiology* 2010;255(3):899-908

Page 903, Figure 2, the second sentence of the legend pertaining to **a** should read as follows: Homogeneous muscle signal intensity without fatty infiltration, as shown in the right **obturator internus** muscle (arrow), was observed.