Volumetric Quantification of Localized Normalized Helicity in Patients with Bicuspid Valve and Aortic Dilation

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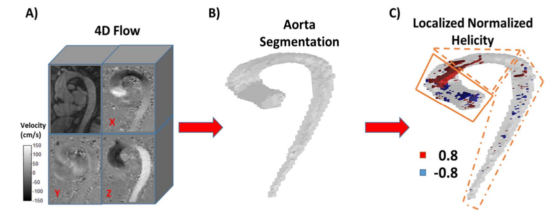

Figure 1. Workflow for volumetric localized normalized helicity. 4D flow data (A) were used for calculating a phase contrast MR angiogram (MRA), the MRA was used to segment the entire aorta (B). Localized normalized helicity (LNH) was then computed and masked using entire aorta segmentation (C). Positive (anti-clockwise in red) and negative (clockwise in blue) LNH 3D features were visualized and quantified using a reference threshold of 0.8 and -0.8, respectively. Ascending aorta (orange line), aortic arch (orange dashed line) and descending aorta (orange dashed-dot line)

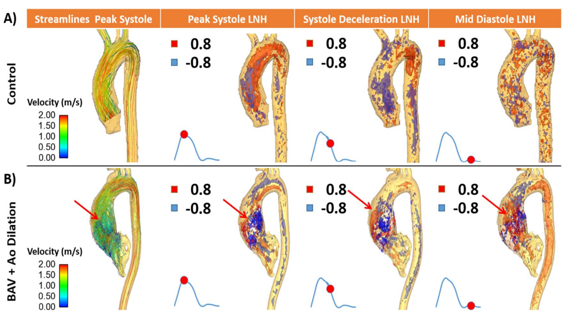

Figure 2. Comparison between healthy control and patient with bicuspid aortic valve (BAV). The horizontal panels show a healthy control subject (A) and a patient (B) with BAV. The first column illustrates the aortic flow velocity streamlines at peak systole; the second column shows 3D localized normalized helicity (LNH, positive spin in red, negative spin in blue) features at peak systole; the third column shows 3D LNH features during systole deceleration and fourth column shows 3D LNH features mid-diastole. Localized, tightly coherent, and temporally long LNH structures (red arrow) illustrate the complex vortex flow alterations which occur during cardiac cycle.

Purpose: Elevated helical flow in the thoracic aorta may result from hemodynamic and anatomic alterations such as aortic dilation, aortic valve stenosis, or bicuspid aortic valve (BAV). The presence of helical flow is often qualitatively estimated by visual assessment of pathline movies, streamlines, or flow vector visualizations. A more quantitative approach to assess the degree of helicity can be achieved by the computation of such parameters as vorticity magnitude threshold, Q-criterion, λ_2-criterion, and localized normalized helicity (LNH)[1-4]. In particular, LNH preserves the rotational direction of the fluid and has been explored in the context of cardiovascular hemodynamics using both 2D measurements and multi-planar reformatting of 4D flow MRI. To improve on the limited coverage of these techniques, this study aims to use the full volume of the 4D flow measured velocity fields to demonstrate that 1) LNH quantification may differentiate helical flow alterations in the aorta between healthy controls and BAV subjects; 2) elevated LNH is correlated with BAV aortic dilation.

Methods: 115 subjects (65 healthy controls [age=43±14 yrs, 25 females, mid-ascending aorta diameter MAA=33±4 mm] and 50 subjects with bicuspid valve (BAV) [age=49±14 yrs, 12 females, MAA=39±6 mm]) were identified via IRB-approved retrospective chart review. Imaging was performed on 1.5T (n=74) and 3T (n=41) MRI (Avanto, Aera, and Trio, Siemens, Erlangen, Germany). The MRI protocol employed an ECG-gated 4D flow during free breathing with adaptive navigator respiratory gating[5]. Data were acquired in the sagittal oblique orientation, covering the entire aorta. Imaging parameters were: Venc=1.5–4 m/s, TE=2.3–2.84 ms, TR= 4.6–5.4 ms, FOV= 212–540 mm×132–326 mm, spatial resolution = 1.66–2.81×1.66–2.81×2.2–3.7 mm³, temporal resolution = 36.8–43.2 ms, FA = 15°. A phase contrast MR angiogram[5] (MRA) was calculated from 4D flow MRI data (Fig. 1A) using in-house software (Matlab,Mathworks, Natick, MA, USA) and used to performed 3D segmentation of the entire aorta (Fig. 1B, Mimics, Materialise, Leuven, Belgium). LNH[3] in was calculated by $LNH = \frac{V.\omega}{|V||\omega|}$, where V is the velocity field from 4D flow MRI and ω is the vorticity derived from V as given by ω=curl (V). LNH calculation results in values between -1 and 1 (clockwise and anti-clockwise rotation respectively, Fig. 1C). As suggested in previous studies, a LNH threshold was set at ±0.8 to identify elevated helicity within the flow domain [3,6]. Positive (LNH>0.8), negative (LNH<-0.8) and total (positive LNH + negative LNH) volumes were then quantified and normalized to the domain volume to obtain a LNH relative volume in %, LNH (%) = (elevated LNH volume)/(domain volume)×100. For LNH quantification, the aorta 3D segmentation was additionally subdivided in 3 segments: ascending aorta (from left ventricle outflow tract to brachiocephalic trunk [BCT]), aortic arch (from BCT to left subclavian artery [LSA]) and descending aorta (from LSA). The mean velocity in the entire aorta was used to identify peak systole, systole deceleration (between peak systole and end systole) and mid-diastole (middle of diastole) phases (Fig. 2).

Results: Elevated LNH volumes were successfully extracted for all subjects. Representative examples for a healthy control and a BAV patient (Fig. 2) illustrate characteristic differences in aortic LNH between groups. Normal flow in controls at peak systole resulted in parallel LNH volumes with opposite rotation, an expected characteristic of physiologic flow. The cohesiveness of the LNH structures slowly degraded during deceleration and diastole (Fig. 2A). In contrast, complex helical flow structures were observed in BAV subjects and led to marked local LNH elevation, which persisted during late systolic deceleration and into diastole (Fig. 2B). Notice that increased LNH appeared during mid-diastole in the descending aorta. LHN quantification in the full aorta (Fig. 3A) showed significant differences between cohorts ($p<0.001$) for all LNH volumes at measured time-points. Sub-domain analysis in the ascending aorta (Fig. 3B), in the aortic arch (Fig. 3C), and in the descending aorta (Fig. 3D) showed significant differences between controls and BAV. LNH volume in the full aorta correlated with MAA diameter for total ($r=0.36$, $p<0.001$) and negative ($r=0.38$, $p<0.001$) at peak systole, for total ($r=0.39$, $p<0.001$) and negative ($r=0.43$, $p<0.001$) at systole deceleration, for total ($r=0.33$, $p<0.001$) and positive ($r=0.34$, $p<0.001$) at mid-diastole. However, the main contribution for these correlations with MAA diameter came from BAV subjects for LNH volumes: total ($r=0.43$, $p<0.05$) at peak systole and in total ($r=0.43$, $p<0.05$) at systole deceleration.

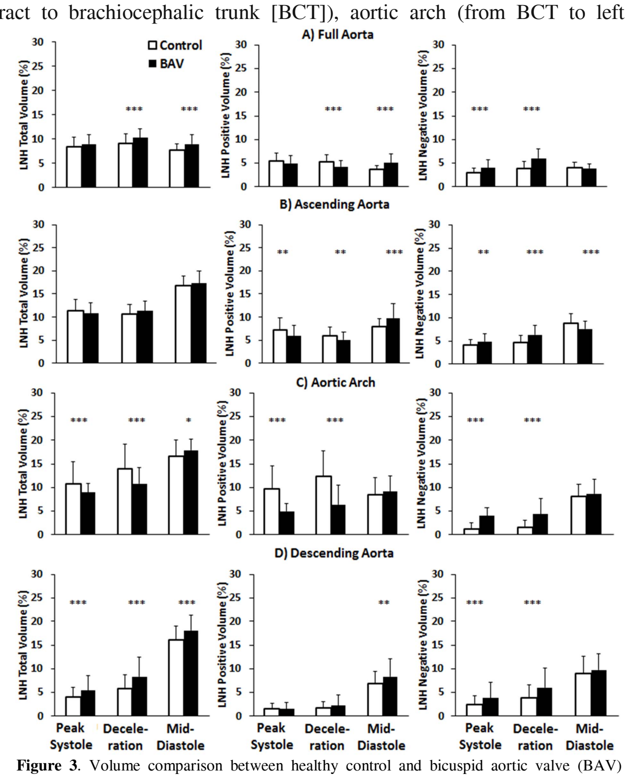

Figure 3. Volume comparison between healthy control and bicuspid aortic valve (BAV) groups. Positive volumes corresponded to localized normalized helicity (LNH) > 0.8 and negative volumes corresponded to LNH < -0.8. The total volume combines the positive and negative volumes. Entire volume corresponded to the full aorta (A) which was further sub-divided on ascending aorta (B), aortic arch (C), and descending aorta (D). Comparison between healthy control vs. BAV groups was given by paired t-test, p-value < 0.05 (*), p-value < 0.01 (**), p-values < 0.001 (***).

Discussion: This study showed that: 1) helical flow alterations can be identified by LNH and can be quantified by volume; 2) elevated LNH can differentiate helical flow alterations in healthy and BAV subjects; 3) elevated LNH was associated with BAV aortic dilation. Previous studies have associated BAV with eccentric flow and elevated flow helicity in the MAA section, and it has been suggested that these flow alterations may contribute to the dilation of the aorta. In this study, LNH in the ascending aorta was different in healthy controls compared to BAV patients using positive or negative LNH. However total LNH was unable to differentiate between groups. The elevated LNH during the diastolic phase may be due to the inertial effects of the flow, aortic compliance and pulse-wave reflection. The main limitation of the calculation of LNH is the use of partial velocity derivatives, which are dependent on spatial resolution and may be sensitive to the velocity encoding value. In conclusion, this study demonstrates the potential usefulness of helical flow quantification to differentiate between controls and subjects with BAV and aortic dilation.

Acknowledgment: This work was supported by NIH R01HL115828, K25HL119608, CONACyT (234939), and AHA 14POST18350019. **References:** 1. Garcia et al. J Biomech Eng 2013; 135(2):124501. 2. Hunt et al. J Fluid Mech 1991; 229:569-87. 3. Morbiducci et al. J Biomech 2013; 46:102-109. 4. Lorenz et al. MRM 2014; 71:1542-53. 5. Markl et al. JMRI 2007; 25:824-831. 6. Gallo et al. Ann Biomed Eng 2012; 40(3): 729-41.