Normal and Pathologically Altered in vivo 3D Aortic Wall Shear Stress Maps

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Purpose: Wall shear stress (WSS) has been associated with extracellular matrix degradation and vascular smooth cell apoptosis[1] and thus is a promising prognostic marker for aortic complications related to dilation, dissection or rupture. The increasing use of 4D flow MRI has permitted the assessment of WSS; however, current difficulties in WSS estimation include: the complexity of measuring WSS along the entire aortic surface, a lack of established WSS values for healthy individuals, and the challenge associated with identifying and describing common aortic locations with abnormal WSS across multiple subjects. In this study, a technique to create cohort-averaged 3D WSS maps is presented which allows for the systematical investigation of differences in regional 3D WSS as assessed in healthy controls, in patients with dilated aortas, and in patients with aortic valve stenosis.

Methods: Prospectively ECG gated 4D flow MRI of the thoracic aorta with a free-breathing navigator was performed in three cohorts: n=10 healthy controls, n=10 patients with aortic dilation, and n=10 patients with aortic valve stenosis (mean MRI-estimated pressure gradient: 55±18 mm Hg) on 1.5 and 3T scanners (Espree, Avanto, Skyra, Aera, Siemens, Erlangen, Germany). All subjects had trileaflet aortic valve morphology. Further patient demographics are given in table 1. Spatial resolution was 1.7-3.6x1.8–2.4x2.2–3.0 mm^3; temporal resolution was 37–42 ms resulting in 14 to 25 time frames; TE/TR/FA was 2.2-2.8 ms/4.6-5.3 ms/7-15^0 and the VENC was 150 cm/s for the healthy controls, 150–250 cm/s for the patients with aortic dilation and 150–450 cm/s for the patients with stenosis. For all subjects, the 4D flow MRI data were corrected for Maxwell terms, eddy currents and velocity aliasing and a time-averaged 3D PC MR angiogram was derived from the 4D flow data[2]. The thoracic aorta was segmented based on the 3D PC-MRA images (MIMICS, Materialise, Leuven, Belgium). 3D WSS was calculated along the segmented 3D aorta surface as previously described[3]. Average 3D WSS maps were created for each cohort (controls, aortic dilation, and aortic valve stenosis) as follows: 1) the cohort-specific aortic segmentations were rigidly co-registered using FLIRT[4]. The degree of overlap (DOO) of the aortas was computed to create a voxel-wise 3D DOO map. 2) Each individual aorta in the cohort was rigidly registered to DOO maps of incrementally increased thresholds and a registration error was calculated. The DOO map demonstrating the lowest registration error averaged over all cohort-specific aortas was chosen as the cohort-specific aorta geometry. 3) To project the WSS vectors onto this geometry, each aorta was registered to the geometry. Affine registration was used to meet a shape most similar to the geometry. WSS vectors of the individual aorta were interpolated to the surface of the registered geometry. Nearest neighbor interpolation was used to ascertain 3D WSS vectors at locations that may not have registered perfectly. 4) The WSS vectors of each subject were averaged over the cohort, resulting in the mean and standard deviation (SD) WSS maps. To calculate the results on a vector-by-vector basis, the WSS maps for the dilation and stenosis cohort were interpolated to the control geometry, which allowed for the creation of P-value maps by comparison of WSS values on each point on the control geometry between two cohorts using a Wilcoxon rank sum test. The percentage of the aortic surface with significantly higher or lower WSS compared to controls was calculated for the ascending aorta (AAo), arch and descending aorta (DAo), see Fig. 1a. Differences across patient cohorts (table 1) were tested with a Kruskal-Wallis test with P<0.05 prescribed as significant.

Table 1. Age and aortic diameter for the cohorts. The percentage of the P-value map that is significantly higher (+) or lower (-) than the controls.

Properties	Controls (6M, 4F)	Dilation (7M, 3F)	Stenosis (9M, 1F)
Age[+] (y)	50±14	54±11	64±14
SOV diameter* (cm)	3.0±0.5	4.3±0.5	4.0±0.4
MAA diameter* (cm)	2.9±0.5	3.7±0.5	4.2±0.3
Percentage +/- AAo	-	4 / 13	54 / 3
Percentage +/- Arch	-	2 / 6	37 / 0
Percentage +/- DAo	-	0 / 1	7 / 0

*SOV = Sinus of Valsalva, MAA= Mid-Ascending Aorta, AAo = ascending aorta, DAo = descending aorta. [+]Not significant (P=0.08), *Significant with P<0.001.*

Results: Figure 1a displays left-anterior oblique views of the cohort-averaged 3D WSS maps (Mean) and WSS SD maps for healthy controls, patients with dilated aortas and aortic valve stenosis. The black arrow indicates a region of elevated WSS, for both the mean and SD WSS map, in the stenosis cohort compared to the control and dilation cohort. Fig. 1b shows P-value maps for the dilation and stenosis vs. control comparisons. For WSS in the dilation cohort, 13% of the AAo surface was exposed to significantly reduced WSS compared to the control cohort (table 1), as indicated by arrows 1 and 2. In the stenosis cohort, WSS in 54% of the AAo surface and 37% of the surface of the arch was significantly higher than for the control cohort (table 1), as indicated by arrow 3.

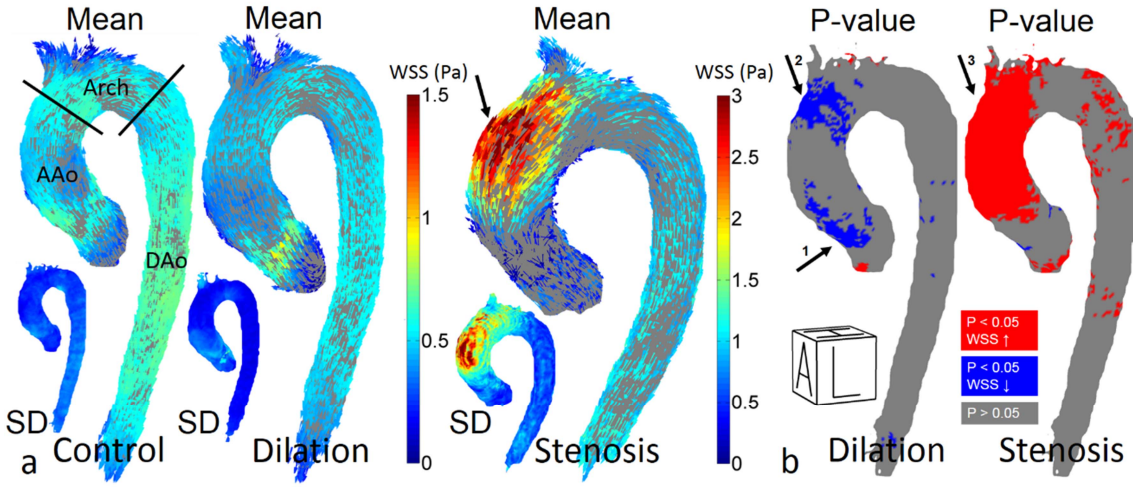

Fig 1. (a) Cohort-averaged and SD 3D WSS maps for the control, dilation and stenosis cohort (b) P-value maps for significant differences between the dilation vs. control and stenosis vs. control cohorts. L = Left, A = Anterior. H = Head.

Discussion/Conclusion: In this pilot study, the methodology of creating averaged WSS maps across multiple subjects was demonstrated and regional alterations of WSS in the presence of disease was identified. The addition of patients or healthy controls to the corresponding maps could provide atlases of high statistical power for identification of abnormal hemodynamic parameters. This approach can be expanded to other hemodynamic parameters such as regional diameter, oscillatory shear index, velocity vector magnitude and direction, or helicity/vorticity and residence times.

References: [1]Lehoux et al. J Biomech (2003) [2]Bock et al. ISMRM (2007) [3]van Ooij et al. JMRI (2013) [4]Jenkinson et al. (2001)

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