Wall Shear Stress Analysis in Ascending Aortic Aneurysms using PC VIPR

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Introduction Wall shear stress (WSS) has been shown to play a role in aneurysm formation [1] and endothelial cell function [2]. 4D flow techniques using phase contrast (PC) MRI have been used to estimate wall shear stress in a number of vessels, including the ascending aorta [3]. High spatial resolution imaging is important in WSS estimation in order to capture velocity changes at the vessel wall. 4D PC MR is challenged by the need to maintain reasonable scan times while providing volumetric cine acquisitions with three directional velocity encoding. PC MRI with 3D radial undersampling (PC VIPR) enables acquisition of 4D flow datasets with increased spatial resolutions in a reasonable scan time [4]. The purpose of this study was to compare time resolved WSS in patients with ascending aortic aneurysms (AscAA) with that in normal volunteers.

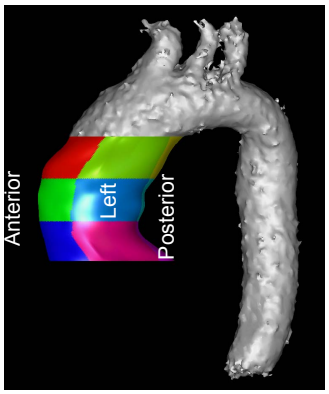

Fig. 1 The ascending aorta was divided into 12 segments as shown.

Materials and Methods For this IRB-approved and HIPAA compliant study, PC VIPR MRI data were acquired in 11 patients with AscAA (7 men, 4 women, ages 10 – 85, mean age: 46.3) and in 10 healthy volunteers (6 men, 4 women, ages 21 – 54, mean age: 32.9). Studies in patients were performed on 1.5T and 3.0T scanners (GE Healthcare, Waukesha, WI, USA) while studies in normal volunteers were all performed at 3.0T (GE Healthcare, Waukesha, WI, USA). An 8-channel body (1 patient) or cardiac (10 patients, 10 volunteers) coil was used for the exams. Parameters for the PC VIPR sequence were: imaging volume = 260 - 360 mm^3 isotropic, 1.0 - 1.4 mm^3 isotropic spatial resolution, VENC = 80 cm/s (10 volunteers, 9 patients), 150 cm/s (1 patient) or 350 cm/s (1 patient), depending on individual maximum velocities in the flow profiles, scan time approx. 10 min. Respiratory gating was performed with an adaptive gating scheme based on bellows readings and 50% respiratory gating efficiency. Data were reconstructed into 20 time frames over the cardiac cycle using retrospective ECG-gating. For WSS calculations, the surface of the ascending aorta was manually segmented based on complex difference image data using a cubic-spline based interpolation algorithm implemented with in-house MATLAB® based software. Surfaces were redefined at each time frame during systole, and a single surface was used throughout diastole. Inward unit surface normal vectors were computed from the spline interpolated surface. For analysis purposes, each ascending aorta surface was divided radially into 4 segments (anterior, posterior, right and left), and horizontally into 3 segments (upper, middle, and lower) to create 12 individual segments (Figure 1). WSS was estimated at each surface point using the equation $WSS = \mu \frac{\partial v}{\partial n}$ where μ is the blood viscosity (assumed to be 4.0 cP), v is the velocity, and n is the inward surface normal. Polynomial interpolation of velocity at and adjacent to the surface was used in estimation. Surface area weighted averages of WSS magnitude were taken over each segment, as well as over the entire surface of the ascending aorta at each time frame defining WSS as a function of time over the cardiac cycle. From each WSS function five parameters were extracted and analyzed: time averaged WSS, peak systolic WSS, time of peak WSS, diastolic baseline WSS, and percent increase in WSS from baseline to peak. Statistical analyses were performed using a *t*-test, where $P<0.05$ was accepted as statistically significant. Additionally, mean composite functions were computed for each group.

Results Mean ascending aortic diameters were 43.7 mm ± 5.3 mm in patients and 31.3 mm ± 3.0 mm in normal volunteers. Bicuspid aortic valves were present in 7/11 patients, and one patient also had a descending thoracic aortic aneurysm. Composite mean WSS values for the aneurysm and control groups are shown in Figure 2. The *WSS-time* curves in patients with AscAA show a slower onset of and prolonged duration of systolic WSS. Although time-averaged WSS was higher and the peak systolic WSS was lower in patients than controls, the results were not statistically significant. Time of peak WSS occurred significantly later in the cardiac cycle, diastolic baseline WSS was significantly increased, and percent increase from baseline to peak was significantly reduced in the aneurysm group (all $P<0.05$). When individual segments were analyzed, the aneurysm group showed significantly increased diastolic baseline WSS (all $P<0.05$) and significantly decreased percent change in WSS (all $P<0.05$) on each individual segment. Significantly delayed time of peak WSS (all $P<0.05$) was also observed on each segment. Figures 3A and 3B show the *WSS-time* curves on the four walls of the ascending aorta in the normal and aneurysm groups, respectively. In the aneurysm group, the anterior wall shows increased peak WSS without significant change in baseline WSS. Peak WSS was significantly reduced ($P<0.05$) on the right wall in aneurysms when compared to controls. Peak WSS ($P<0.05$) and percent change in WSS ($P<0.05$) on the lateral wall were both significantly increased when individually compared to the entire ascending aorta.

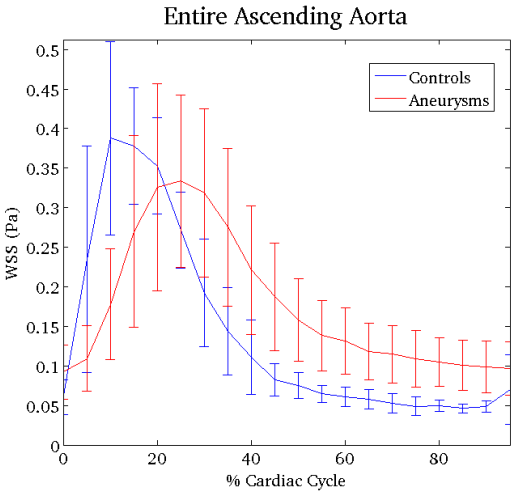

Fig. 2 WSS averaged over the entire ascending aorta over one cardiac cycle. Aneurysms show increased WSS during diastole ($P<0.05$), reduced percent change ($P<0.05$), and delayed onset of WSS in systole ($P<0.05$).

Conclusion Use of time resolved 3D vessel surface segmentation allowed for optimized utilization of high resolution 4D flow MRI with 3D radially undersampled PC data sets. This allowed for investigation of WSS dynamics over the cardiac cycle and showed that AscAA are associated with alterations in WSS. Individuals with AscAA have increased WSS during diastole, reduced change in WSS over the cardiac cycle, and delayed onset of WSS in systole compared to individuals with normal aortic anatomy. Spatial patterns of WSS in AscAA show differences from normal ascending aortas.

References [1] L Boussel *et al.*, *Stroke* 39(11), 2997-3002, 2008 [2] PM Cummins *et al.*, *AJP Heart Circ Physiol* 292(1), H28-42, 2007 [3] A Frydrychowicz *et al.*, *JMRI* 30(1), 77-84, 2009 [4] T Gu *et al.*, *AJNR* 26(4), 743-9, 2005

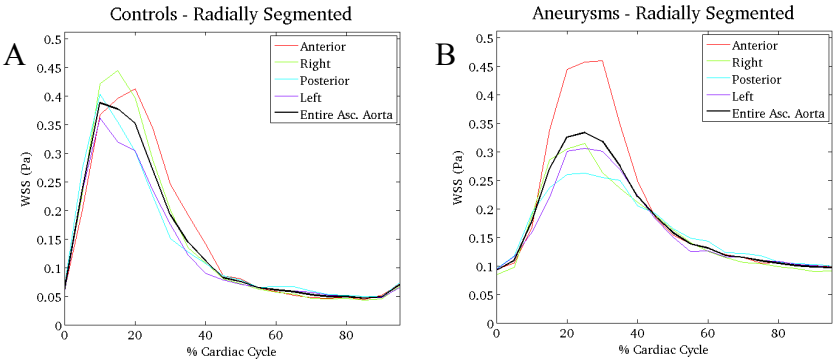

Fig. 3 WSS on the four walls of the ascending aorta in normal aortas (A) and aneurysms (B). Peak WSS is increased ($P<0.05$) along the anterior wall of the aorta in aneurysms.