MRI Measurements of Wall Shear Stress in the Descending Aorta Downstream of an Ascending Aortic Aneurysm

A. L. Wentland^{1,2}, E. T. Bieging², A. Frydrychowicz², T. M. Grist^{1,2}, O. Wieben¹, and C. J. Francois²

¹Medical Physics, University of Wisconsin School of Medicine & Public Health, Madison, WI, United States, ²Radiology, University of Wisconsin School of Medicine & Public Health, Madison, WI, United States

INTRODUCTION

Wall shear stress (WSS) may play a vital role in the pathogenesis and pathophysiology of aneurysms. Meng *et al.* showed that elevated peak longitudinal WSS may initiate aneurysm formation [1], whereas Boussel *et al.* demonstrated that low peak longitudinal WSS may contribute to aneurysm growth [2]. While measurements of WSS have been investigated within the aneurysmal bodies, the downstream effects of an aneurysm on WSS remain largely unknown. The purpose of this study was to compare measurements of WSS in the descending aorta in healthy volunteers and in patients with ascending aortic aneurysm. The limited spatial resolution of MR flow measurements results in an underestimation of the actual WSS parameters as derived by CFD simulations [3]. However, it is likely to be a surrogate parameter of the actual WSS. Here we used PC VIPR [4], a radially undersampled acquisition with three directional cine velocity encoding, which provides high spatial resolution with isotropic voxel sizes, making this approach well suited for such multidirectional hemodynamic analysis.

MATERIALS AND METHODS

This HIPAA compliant study was approved by our institutional human subjects review committee and written informed consent was obtained from all subjects. PC VIPR data were acquired on 1.5T or 3T MR scanners (GE Healthcare, Waukesha, WI) in nine healthy volunteers (6 males; 3 females; average age = 34.2 years) and eleven patients with an aneurysm in the ascending aorta (6 males; 5 females; average age = 46.2 years). Scans were performed with the following parameters: imaging volume = 320 x 320 x 180 mm³, readout = 256-320, 1.0-1.25 mm³ acquired isotropic spatial resolution, VENC of 80 cm/s, TR/TE/flip = 8.7ms/2.8ms/10°, cardiac and respiratory gating, scan time ~ 10 min.

Vessel segmentation was performed manually with in-house software (MATLAB version 8.0, The MathWorks Inc., Cambridge, MA, USA). First, points were selected around the circumference of the vessel on complex difference images reformatted as axial to the vessel. From these points, a cubic spline was created. This step was repeated on axial slices ranging from the end of the aortic arch to immediately superior to the celiac artery (Figure 1A). From these axial splines, splines in the superior-to-inferior (SI) direction—along the length of the aorta—were also created. The intersection of the SI and axial splines created surface points along which an inward unit normal vector was computed; longitudinal wall shear stress was then calculated as the viscosity of fluid multiplied by the slope of the velocity along this unit normal vector (Figure 1B). Viscosity was assumed to be 4.0 cP for all subjects. This process was repeated for each time frame over the cardiac cycle.

Measurements of WSS for each surface were binned and averaged into twelve segments for each time frame (Figure 1C)—grouped into 4 circumferential regions at three SI levels. The average WSS over the twelve segments and across the cardiac cycle was plotted for each of the volunteers and patients. The greatest (peak) average WSS from the twenty time frames for each segment was compared between volunteers and patients with a Student's t-test (p < 0.05). Additionally, the time frame in which the peak WSS occurred, along with the percent increase in WSS from baseline to peak, were compared between volunteers and patients with a Student's t-test (p < 0.05).

RESULTS AND DISCUSSION

Average WSS over the cardiac cycle is shown in Fig. 2 for all volunteers and patients. Measurements of peak WSS tended to be lower in patients than in volunteers for all 12 segments (Table 1). The greatest (peak) average WSS over the twenty time frames was significantly different between patients and volunteers for each of the twelve segments (p-values < 0.05). The percent increase in WSS from baseline to peak was significantly different between patients and volunteers for each of the twelve segments (p-values < 0.05). Values of percent increase tended to be greater in volunteers (average = 1136%) than in patients (average = 571%). The time frame with the greatest WSS tended to occur later in patients (average = 6) than in volunteers (average = 4), although these values were not significantly different.

CONCLUSIONS

Our study showed that longitudinal wall shear stress in the descending aorta tended to be lower in patients with an ascending aortic aneurysm than in healthy volunteers. Studies have demonstrated an association between the distribution of atherosclerotic lesions and low wall shear stress [5]. The long-term effects of low wall shear stress downstream of an ascending aortic aneurysm, as computed in this study, behoove further investigation.

ACKNOWLEDGEMENTS

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REFERENCES: [1] Meng et al. *Stroke.* 2007;38(6):1924-31. [2] Boussel et al. *Stroke.* 2008;39(11):2997-3002. [3] Wood et al. *JMRI.* 2001;13:699-713. [4] Johnson et al. *MRM.* 2008;60(6):1329-36. [5] Pedersen et al. *Eur J Vasc Endovasc Surg.* 1999;18:328-333.

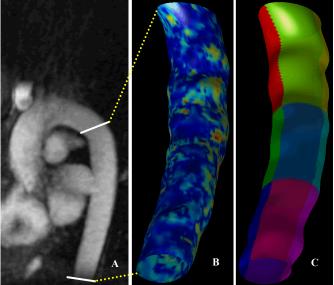


Figure 1. (A) Maximum intensity projection of the aorta. (B) Map of wall shear stress measurements in the descending aorta in a single time frame for a healthy volunteer. (C) The twelve segments used to bin wall shear stress measurements in the descending aorta for all subjects.

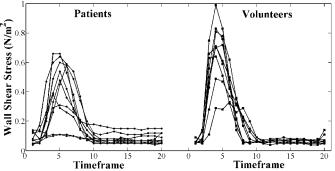


Figure 2. Average wall shear stress in the descending aorta over twenty time frames across the cardiac cycle in nine healthy volunteers and eleven patients with ascending aortic aneurysm.

Table 1. Average peak wall shear stress over the cardiac cycle in twelve segments in the descending aorta of nine healthy volunteers and eleven patients with ascending aortic aneurysm.

WSS (N/m ²) in Patients/Volunteers				
·	Lateral	Medial	Anterior	Posterior
Upper	0.33/0.56	0.48/0.74	0.44/0.81	0.38/0.67
Middle	0.49/0.72	0.48/0.80	0.44/0.81	0.44/0.69
Lower	0.41/0.72	0.47/0.74	0.42/0.70	0.40/0.66