Hemodynamics in the Aorta and Coronary Arteries: A study with MRI and CFD

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Introduction: A large number studies have suggested a correlation between locations of intimal thickening (or atherosclerotic lesions) and regions where arterial wall shear stress (WSS) is low or oscillatory. Most studies have determined WSS values based on idealized experimental or computational models. Intimal thickness (IT) measurements or plaque distributions have come from large pathology studies involving many post-mortem specimens. Correlating WSS and IT or plaque location using this methodology has two drawbacks. First, using an *average* model geometry and *average* lesion location may obscure the relation of WSS and lesion location. Secondly, correlating WSS and lesion location with this approach cannot provide individual, localized, *patient-specific* values of WSS. The purpose of this study was to determine *patient-specific* WSS in the proximal coronary arteries by modeling flow *in the aorta and coronary arteries*. A combination of magnetic resonance imaging (MRI) and computation fluid dynamics (CFD) was used to study indiviualized hemodyamics.

Methods: Solving the complete flow field using CFD requires determination of the model geometry and determination of time-varying flow at the inlets and outlets of the model. The geometry information for creating individualized CFD models of the aorta and coronary arteries came from segmented, ECG and navigator-echo gated MRA exams. To determine the coronary geometry, a technique based on the navigator-echo-gated, 3D, whole-heart coronary MRA protocol was used (1). To determine geometry of the aorta, a similar scan but with thicker slices and lower resolution was used. From these two scans, the aorta and coronary arteries were segmented and a model of each subject, including the ascending aorta, aortic arch, proximal arch vessels, thoracic descending aorta, and proximal epicardial coronary arteries was constructed, Figure 1.

Phase contrast (PC) MRI was used to determine time-dependant velocity profiles in the ascending aorta (just below the coronary ostia), in the descending aorta, in the aortic arch branch vessels, and coronary arteries. MR velocity measurements in the aorta and arch branch vessels were done with a segmented, retrospectively ECG-gated sequence with 16 frames reconstructed over the cardiac cycle. Coronary velocity measurements were done with a similar sequence, with the addition of navigator-echo gating and prospective ECG gating. Computations were performed with a commerical code (CFD-ACE, CFD Research Corp, Huntsville, AL) assuming a Newtonian, incompressible fluid and lamiar flow.

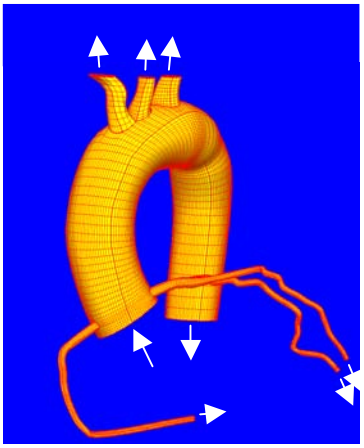

Figure 1. Mesh of the aorta and coronaries derived from MRA scans. Arrows represent location of MR velocity measurements.

Results and Discussion: Three normal subjects and one patient have been studied. In all subjects, entrance flows in the right and left coronary arteries showed that transient axial vorticies formed at the entrance regions. These vortices caused significant skewing of the velocity profile at the coronary ostia. In the RCA, there is near wall flow reversal on the *myocardial* side, whereas in the left artery the near wall flow reversal is on the *epicardial* side, Figure 2. Thus, the myocardial wall of the RCA sees a relatively low time-averaged WSS that oscillates in direction, whereas this type of flow behavior occurs on the epicardial side of the LM. The vorties and skewed velocity profiles presisted distally only 5 mm into the LM and RCA. Previous pathology studies (2,3) found that in the proximal RCA, there was a tendency for plaques to be concentrated in the myocardial side of the lumen, but in the LM plaques were located on the epicardial side. Thus our data is consistient with plaque formation occurring in regions of low or oscillatory WSS. The flow patterns seen in the proximal coronary ostia could not be seen without complete modeling of both the aorta and coronary arteries.

Conclusion: MRI and CFD was used to model flow in the aorta and proximal coronary arteries. Skewing of the velocity profiles in the proximal coronary arteries is caused by the presence of a transient vortex at the ostia. Results presented here are suggest atherosclerotic plaques preferentially form in areas of low or oscillatory WSS.

References: 1) Weber, MRM 2003; 2) Fox and Seed, J of Biomech Eng, 1981; 3) Svindland, et al, Atherosclerosis, 1983

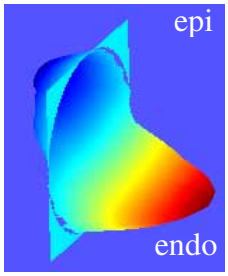

Figure 2. 2D velocity profile at the Left Main (LM) coronary ostia in. mid-diastole obtained from the CFD simulation. Note marked skewing toward the myocardial wall and the reverse flow at the epicardial wall the skewing