VORTEX RINGS IN STENOTIC ARTERIAL MODELS

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Summary Atherosclerosis, a disease of the vasculature characterized by plaque formation in arteries, is of the leading causes of death in the western world. The global aim of the current work is to directly link fluid mechanical stimuli to cellular response in physiologically relevant geometries. This paper focuses on the vortex ring formation observed downstream of an occlusion in a tube subjected to a sinusoidal forcing function. It was found that the shear layer in the recirculating region reduces the circulation of the vortex ring as it progresses downstream within the tube. Future work will involve a more detailed investigation of the vortex ring formation and break down.

INTRODUCTION

Atherosclerosis is a disease that results in over one million deaths per year in the United States. It is characterized by altered endothelial and smooth muscle cell physiology in the artery lumen and the subsequent build-up of plaques on the arterial wall. It is known that plaques tend to accumulate and coalesce to form stenoses in regions of low and/or oscillating wall shear stress. Cells in the region of pathological shear stress distributions respond to the mechanical stimulation by releasing several atherogenic transcription factors and chemical mediators. Flow distal to a previously formed stenosis can induce non-laminar flow downstream of the constriction, thus inducing pathological shear stress distributions.

Initial research focused upon steady flow conditions. Ahmed and Giddens (1983a, b) measured the flow fields distal to axisymmetric stenoses of 25, 50 and 75% area reduction using laser Doppler anemometry in scaled up models and Deshpande and Giddens (1980) reported on the turbulence in a constricted tube. These researchers found that distal to a stenosis, there exists a recirculating region and an unsteady, turbulent-like zone. The recirculating zone is a region of low shear stress, while the turbulent-like zone produces unsteady wall shear stresses. Both of these flow conditions are conducive to formation of atherosclerotic lesions. Therefore, a stenosis in an artery further precipitates plaque formation and artery occlusion.

The dominant flow structure observed in model post-stenotic flow is the development of a vortex ring during systole, termed the starting vortex. The starting vortex eventually breaks down and provides energy to the turbulent-like flow upstream of the relaminariztion zone. In the current study, Particle Image Velocimetry (PIV) is used to investigate the starting vortex distal to an actual size *in vitro* axisymmetric stenosis model at a moderate physiological Reynolds and Womersley numbers in pulsatile flow. Concurrent numerical simulations by Varghese et al. (2003) will be used in conjunction with companion cellular studies in an identical geometry (see McCann et al., 2002) to correlate regions of mechanical stimulation to cellular response.

EXPERIMENTAL SETUP AND PROCEEDURE

Experiments were performed in a custom pulsatile flow facility. A schematic of the flow loop is illustrated in Figure 1. An Ismatec programmable pump controlled by LabView 6.1 pumped water at 20°C through the facility. The test area consisted of a nominally 10mm ID glass tube with the stenotic test section formed by glassblowing. The axisymmetric 75% area-reduction occlusion had a length-to-diameter ratio of 1.2 and was located 38D from the tube entrance to ensure fully developed Womersley flow upstream of the test section.

Particle Image Velocimetry was used to measure the velocity fields at the tube centerline between 1.2 and 2.9 tube diameters downstream of the occlusion (see Figure 1). A sinusoidal voltage waveform was input into the programmable pump. Data were collected at intervals of 0.01 seconds during the 0.06 second portion of the 2.25 second cycle in which the starting vortex was in the field of view. The mean Reynolds number was 1000, with an amplitude of ±480 and a Womersley number of 8.4.

The PIV system consisted of a 50mJ/pulse New Wave GeminiPIV Nd:YAG laser, a TSI PowerView 12-bit, 2000x2000 pixel, cross-correlation CCD array, TSI synchronizer and a dual Pentium Xeon processor data acquisition computer. The TSI Insight 3.53 software package was used to capture and correlate 50 images at each measurement location. The images were correlated using the Hart cross-correlation engine with a bilinear peak-finding algorithm. The primary interrogation was 32x32 pixels with a 16x16 subcorrelation window and a compression ratio of 90%. The physical size of each interrogation region was 0.26x0.26 mm^2 for the primary interrogation window and 0.13x0.13 mm^2 for the subcorrelation window. This provided a spatial resolution (based on the subcorrelation window) of 65 μm. Preliminary mean velocity fields were obtained by spatially averaging the 50 instantaneous velocity fields. This relatively small sample size offered a good compromise between convergence of the mean field and available disk space. In regions of large cycle-to-cycle flow variations, the small sample size is insufficient for convergence of the mean velocity field, and the final data set will contain more realizations.

RESULTS

Vortex rings issuing into an unbounded quiescent fluid have previously been studied with regards to formation, maximum circulation, pinch-off from the trailing shear layer, and available energy (*e.g.* Gharib, Rambod & Shariff, 1998; Rosenfeld, Rambod & Gharib, 1998). It has been noted that the maximum circulation of these vortex rings is bounded. Once the circulation, fed by the vorticity in the trailing shear layer, reaches the upper bound the vortex will pinch off from the trailing shear layer, and propagate with constant circulation. The upper bound is dictated by the 'formation number' or stroke ratio. However, within the recirculation region of this continuously pulsing (always positive, non-zero volume flow rate), wall-bounded arterial model, the physics are quite different. The vortex ring is does not escape the shear layer, and pinch-off does not occur. Figure 1 shows the progression of a vortex ring through a measurement region ~1.5D downstream of a smooth, rounded obstruction. The figure illustrates the flow direction via streamlines overlaid on a contour of vorticity magnitude. The primary vortex ring is clearly seen progressing downstream, followed by a smaller vortex ring in the trailing shear layer. Additionally, a vortical structure is sometimes visible upstream of the primary vortex ring, likely a remnant of the previous cycle. The vortex ring is not observed to separate from the trailing shear layer in the measurement region, and its circulation does not remain constant. In fact, the circulation of these confined vortex ring decreases with time, as they propagate downstream.

Current studies involve more time steps for greater temporal resolution, more instantaneous realizations to improve signal-to-noise ratio as well as the circulation measurement, and more measurement regions to better track the evolution of the primary vortex ring.

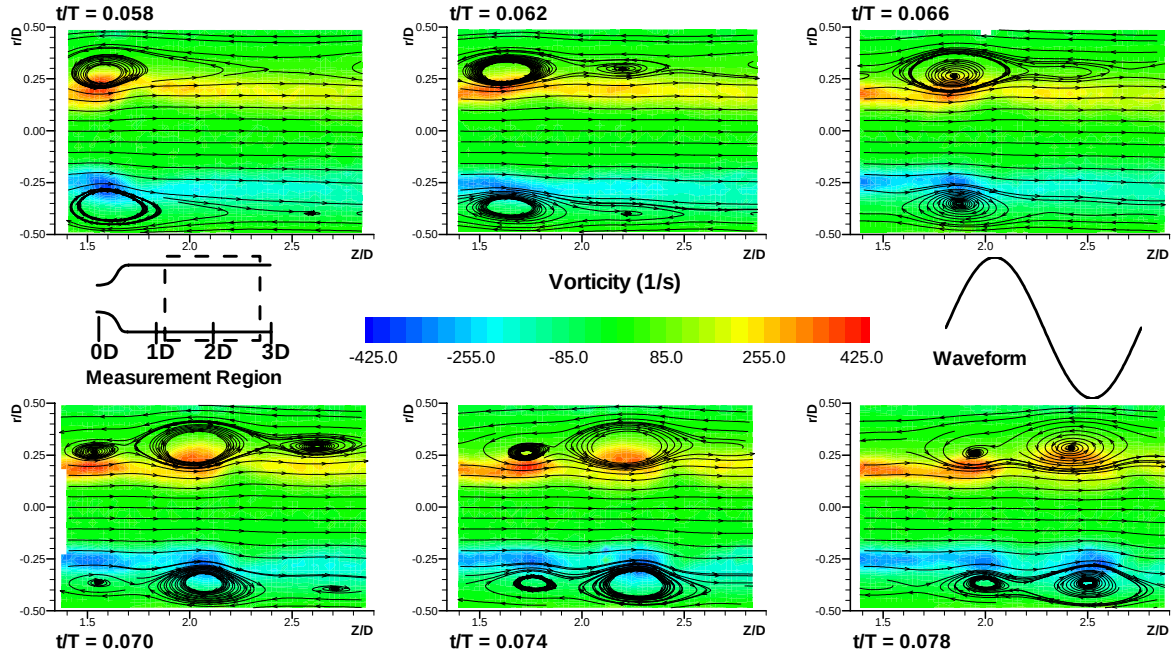

Figure 1: Vortex Ring Progression Through Measurement Region

CONCLUSIONS

The formation and propagation of a vortex ring in a model arterial vessel is complicated by the persistent shear layer in the recirculation region. The vortex ring does not issue into quiescent fluid, but rather it encounters the shear layer induced by the recirculation. The vortex ring is potentially impacted by the previous vortex ring as well. Further investigation of the formation process, propagation characteristics, and ultimate breakdown of the vortex ring is currently in progress.

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