FLOW DYNAMICS IN ANATOMICAL MODELS OF ABDOMINAL AORTIC ANEURYSMS: COMPUTATIONAL ANALYSIS OF PULSATILE FLOW

Ender A. Finol¹ and Cristina H. Amon²
¹ Carnegie Mellon University Institute for Complex Engineered Systems
Pittsburgh, PA 15213 - U.S.A.
e-mail: finol@andrew.cmu.edu
² Carnegie Mellon University Institute for Complex Engineered Systems, Mechanical Engineering,
Biomedical Engineering, Pittsburgh, PA 15213 - U.S.A.
e-mail: camon@cmu.edu

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ABSTRACT: Blood flow in human arteries is dominated by time-dependent transport phenomena. In particular, in the abdominal segment of the aorta under a patient’s average resting conditions, blood exhibits laminar flow patterns that are influenced by secondary flows induced by adjacent branches and in irregular vessel geometries. The flow dynamics becomes more complex when there is a pathological condition that causes changes in the normal structural composition of the vessel wall, for example, in the presence of an aneurysm. An aneurysm is an irreversible dilation of a blood vessel accompanied by weakening of the vessel wall. This work examines the importance of hemodynamics in the characterization of pulsatile blood flow patterns in individual Abdominal Aortic Aneurysm (AAA) models. These patient-specific computational models have been developed for the numerical simulation of the momentum transport equations utilizing the Finite Element Method (FEM) for the spatial and temporal discretization. We characterize pulsatile flow dynamics in AAAs for average resting conditions by means of identifying regions of disturbed flow and quantifying the disturbance by evaluating wall pressure and wall shear stresses at the aneurysm wall. Key words: Aneurysms, pulsatile flow, computational fluid dynamics.

INTRODUCTION

Abdominal Aortic Aneurysms (AAAs) occur in the infrarenal segment of the abdominal aorta, between the renal arteries and the iliac bifurcation. The majority of studies found in the medical literature report an increase in the incidence of aortic aneurysmal disease, which is rather expected in a continuously aging population in developed countries, since the likelihood of aneurysm development increases with age. Despite significant improvements in surgical procedures and technological advancements in imaging devices, the associated mortality and morbidity rate has also risen concomitantly. The mean age of patients with AAA is 67 years and men are affected more than women by a ratio of 4:1 with prevalence up to 5%¹¹. AAAs are a health risk of significant importance since they are largely asymptomatic until the onset of rupture, an event that carries an overall mortality rate in the 80% to 90% range. AAA rupture constituted the 13th leading cause of death in the United States in the 1990s, causing more than 10,000 deaths each year¹⁰, and affecting 1 in 250 individuals over 50 years of age. Therefore, the optimal strategy is clear: opportunistic detection and prevention of aneurysm rupture is the primary goal in management of aneurysmal disease.

Aneurysm rupture is a biomechanical phenomenon that occurs when the mechanical stress acting on the inner wall exceeds the failure strength of the diseased aortic tissue. Since the internal mechanical forces are initiated and maintained by the dynamic action of blood flow within the aneurysm, the hemodynamics of AAAs becomes an important element of study for the characterization of the biomechanical environment of aneurysms.
METHODOLOGY

Two patients with significant abdominal aortic dilation were selected for the study. The methodology is based on the geometric reconstruction of each patient's AAA, the generation of adequate finite element domains and the simulation of the momentum transport equations for physiologically realistic blood flow properties.

Three-dimensional geometry reconstruction. The geometry of human AAAs is too complex to be reliably approximated using hypothetical representations as has been done in most previous analyses \cite{3,17}. For clinically meaningful and accurate results, it is necessary to use the actual "irregular" geometry of an individual AAA. For this work, a 3-D reconstructed model of an actual AAA was utilized. This was obtained from previous work reported by Wang et al. \cite{16}, which utilized a modified technique described in Raghavan et al. \cite{15}. In short, spiral computed tomography (CT), which is routinely performed on AAA patients scheduled for repair, was performed to provide cross-sectional images of the abdominal region during a single sustained breath hold by the patient. The slice thickness (collimation) was between 3mm to 5mm with a helical pitch of 1.5. Individual cross-sectional image slices were then generated at 2-3 mm slice spacing along the infrarenal aorta. Digital files containing the cross-sectional images from immediately distal to the renal arteries to immediately proximal to the iliac bifurcation were imported into Scion Image (Release Beta 3b, Scion Corporation, Frederick, Maryland) for segmentation. The boundary of the inner AAA wall was identified using grayscale thresholding and semi-automated edge detection \cite{15}. The spatial coordinates of about 60 discrete points along the wall boundary were recorded on each cross-section. From this, a point cloud representing discrete points on the AAA wall and lumen was obtained. Using the point cloud, the 3-D representation of the AAA wall and lumenal surfaces were triangulated and smoothed as described \cite{15,16}.

Finite element modeling. The starting point for the development of a Computational Fluid Dynamics (CFD) model is the output of the reconstructed digital images, which is a solid form representation of the real aneurysm, developed by the Vascular Biomechanics and Vascular Surgery Research Laboratory at the University of Pittsburgh. The modified solid form geometry and resulting mesh are shown in Figure 1 for AAA model #1. The aneurysm shell resulting from the image reconstruction procedure are modified with solid modeling software by extruding a uniform wall thickness of 2.0 mm and adding inlet and outlet extensions prior to generating the finite element mesh. Since the cross-sections of patient-specific abdominal aortas are not necessarily circular, an inlet extension is required when simulating blood flow to allow the flow to develop by the time it reaches the entrance of the aneurysm. At average resting conditions, the pulsatile flow waveform is triphasic, with a short period of flow reversal. The addition of an outlet extension prevents backflow into the aneurysm during this period. AAA model #1 has a maximum transverse dimension of 5.80 cm and the inlet extension diameter is measured at 2.70 cm. The computational domain consists of hexahedral linear elements comprising 141,336 nodes. Illustrated in Figure 2, AAA model #2 has a maximum transverse dimension of 6.55 cm and an inlet extension diameter of 2.88 cm. The computational domain for this aneurysm was also built with hexahedral linear elements resulting in 415,976 nodes.

Governing equations and boundary conditions. Incompressible, homogeneous, Newtonian flow is simulated for average resting conditions (heart rate = 60 bpm). Although blood is actually a non-Newtonian suspension of cells in plasma, it is reasonable to model it as a Newtonian fluid in vessels greater than approximately 0.5 mm in diameter \cite{12}. The x, y, z, t momentum and continuity equations in compact form are given by Eq. (1) as follows:
Computational analysis of pulsatile flow

\[ \frac{\partial \mathbf{v}}{\partial t} = -\nabla p - \nabla \cdot \mathbf{v} + \mathbf{f} \] \hspace{1cm} (1a)

\[ \nabla \cdot \mathbf{v} = 0 \] \hspace{1cm} (1b)

where \( \mathbf{v}(x,t) = u_x \hat{x} + u_y \hat{y} + u_z \hat{z} \) is the velocity vector. The stress components are determined using the Newtonian flow constitutive relationship, as given by Eq. (2), since incompressible flows satisfy the divergence-free condition for the velocity \( \nabla \cdot \mathbf{v} \):

\[ \tau_{ij} = -\rho \delta_{ij} + \mu \left( \frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) \] \hspace{1cm} (2)

Details on the calculation of wall shear stress are given by Finol and Amon.\(^5\) Blood flow is simulated for average blood properties:\(^1\): molecular viscosity \( \mu = 0.00319 \) Pa·s and density \( \rho = 1.050 \) kg/m\(^3\). The governing equations are nondimensionalized by the inlet diameter; hemodynamic stresses evaluated at the wall are normalized using their corresponding magnitudes obtained for Poiseuille flow.

The boundary conditions for the velocity \( \mathbf{v} \) are imposed as follows: (i) no-slip at the walls, (ii) uniform (slug) profile at the inlet, and (iii) zero-traction outflow at the exit. For pulsatile flow, the inflow mean velocity is time-dependent and the volume flow rate is oscillatory, as shown in Figure 3.

![Figure 3](image-url)

**Figure 3.** Pulsatile volumetric flow rate (Q) and instantaneous Reynolds number (Re) for \( Re_m = 300 \). Flow stages A, B, ..., I are of particular importance for the evaluation of hemodynamic stresses. Peak systolic flow occurs at \( t = 0.31 \) s and diastolic phase begins at \( t = 0.52 \) s.

The pulsatile waveform is represented by a discrete Fourier series based on the in-vivo measurement reported first by Mills et al.\(^13\), which is a triphasic pulse appropriate for average resting hemodynamic conditions in the abdominal segment of the human aorta.\(^5\) The Womersley number, which characterizes the flow frequency, the geometry of the model and the fluid viscous properties, is \( \alpha = 11.5 \). The amplitude coefficient of the waveform is \( \gamma = 5.25 \) and peak systolic flow occurs at \( t = 0.31 \) s. The time-average Reynolds number is calculated as \( Re_m = D \bar{u}_m / v \), where \( \bar{u}_m \) is the time-average inflow mean velocity. For the comparison of results, the inflow mean velocity in each AAA model was scaled so that the time-average Reynolds number, \( Re_m \), was identical to 300, a value generally accepted for resting conditions.\(^14\) The instantaneous Reynolds number achieved at peak flow, \( Re \) peak, was evaluated at 1575.

**RESULTS AND DISCUSSION**

**Flow dynamics.** Since the solid geometry is not aligned with any particular Cartesian coordinate axis, a reference plane in AAA model #1 is chosen for the study of flow patterns during the pulsatile velocity cycle. The reference plane is contained completely inside the aneurysm, which is useful for a full characterization of the velocity field within the boundaries of the geometry. Figure 4 illustrates the velocity field at four different flow stages from a lateral view of AAA model #1 that is perpendicular to the reference plane. The exterior boundary of the aneurysm in these frames is the outer vessel wall and the fluid domain is contained within the boundary 2.0 mm inward, since a uniform thickness was extruded for future use of the geometry in fluid-structure interaction analyses. Therefore, the reference plane encloses almost completely the fluid domain inside the vessel segment.

At peak flow (\( t = 0.31 \) s), a completely attached flow pattern is obtained, due to the strong pressure gradient that dominates systolic acceleration. The jet of fluid present within the inlet extension travels at a high velocity and, upon entering the aneurysm, it decelerates locally without recirculating. Vortex shedding and ejection downstream during early systole are typical of complex geometries, such as patient-specific AAAs where the aneurysm sac is irregular. In Figure 4(b), the flow is decelerating and it begins to separate from the lateral-anterior wall at the point of inflexion between the inlet extension and the aneurysm, and also downstream at the point of inflexion between the outlet extension and the lateral-posterior wall. The maximum velocity at \( t = 0.42 \) s is about half the peak flow velocity, as flow recirculation is minimal and absent at the center of the aneurysm. Flow reverses its direction at \( t = 0.52 \) s, shown in Figure 4(c). The pressure gradient exerted in the opposite direction (the flow now travels upward) results in a low-velocity clockwise-rotating vortex at the center of the aneurysm. The point of inflexion at the proximal neck of the aneurysm is a region of high velocity where the flow coming from the aneurysm accelerates locally and mixes with the flow inside the inlet extension. Figure 4(d) illustrates a typical flow pattern that characterizes late diastole: low-velocity flow recirculation at the midsection of the aneurysm, translation of the vortex center downstream at the end of the cycle, and faster forward moving flow that shears the lateral-posterior wall in the absence of flow reversal.
The patient-specific configuration of the diseased aorta greatly influences the flow patterns inside the aneurysm. The preference for forward flow motion along the lateral-posterior wall is not surprising, since the wall is less irregular at this surface. Therefore, due to preferential bulging of the anterior wall, convective transport rates are significantly higher along the lateral-anterior wall and the flow will always separate first along this wall to generate vortex growth and translation downstream.

Figure 4. Velocity vectors for AAA model #1 at (a) $t = 0.31$ s, (b) $t = 0.42$ s, (c) $t = 0.52$ s and (d) $t = 1.00$ s. The normal direction of the flow is from top to bottom.

Wall pressure. The spatial variation in hemodynamic pressure at any given stage of the flow during a single cardiac cycle is small along the inner AAA wall in comparison to the pressure gradient resulting from the application of the pulsatile flow inlet boundary condition. The most significant pressure change is obtained at peak flow, where the pressure gradient is greatest. The wall pressure distribution at this stage along the lateral-anterior wall of each AAA model is shown in Figure 5. Wall pressure is almost uniform within an aneurysm, decreasing slightly towards the distal end, which is produced by the converging shape of the cross-sectional area imposed by the outlet extension. In general, the variation in wall pressure from inlet to exit follows the trend imposed by the pressure gradient. The reported hemodynamic pressure distribution is relative to the cardiac pressure pulse at resting conditions. Therefore, the actual normal forces exerted on the inner AAA wall at peak flow are the result of the linear combination of peak systolic pressure and the hemodynamic pressure illustrated in Figure 5.

Based on the two aneurysms studied here, it appears that peak hemodynamic pressure correlates positively with the size of the aneurysm. Previous studies based on hypothetical shapes of AAAs indicate the existence of this correlation with aneurysm diameter. The actual distribution pattern of wall pressure is dependent on the geometric irregularities of the AAA and the angulations of the proximal and distal extensions.

Figure 5. Color mapping of flow-induced wall pressure distribution (in kPa) corresponding to peak flow in the lateral-anterior view for (a) AAA model #1 and (b) AAA model #2.

Noteworthy is the fact that blood pressure acting on the inner surface will determine the stresses on the vessel wall itself. During the cardiac cycle, the instantaneous fluid forces acting on the wall will deform it. Conversely, the wall motion alters the fluid velocity field until equilibrium is reached. This event occurs for each instant of the periodic blood flow pulse. In this regard, the actual fluid stresses and wall mechanics can only be
accurately predicted by performing a nonlinear fluid-structure interaction (FSI) study. On-going work by the authors involves time-dependent FSI computations in patient-specific AAA models.

**Wall shear stress.** The normalized wall shear stress surface distributions are shown in Figure 6 for stages C ($t = 0.31$ s) and I ($t = 1.00$ s). The distribution of wall shear stress along the anterior and posterior walls of the patient-specific aneurysm is very complex and nonuniform at each cross-section during diastole. The maximum wall shear stress is obtained at peak flow (peak shear stress) along the lateral-anterior wall upstream of the proximal neck. Peak wall shear stress is 64 times higher than the expected for this patient's normal abdominal aorta. The large velocity gradients encountered as the flow adjusts itself to enter the aneurysm sac yield the high wall shear stress. The balloon-shaped expansion of the vessel causes convective deceleration of the flow and a low and uniform shear stress along the wall of the aneurysm. An increase in wall shear stress is obtained at the distal neck as the flow accelerates to enter the outlet extension. The flow stage illustrated for late diastole ($t = 1.00$ s) has a common pattern for wall shear stress: high wall shear stress at the proximal neck, low and high shear stress along the posterior wall, and low shear stress along the anterior wall at levels lower than expected for an undilated vessel. The regions of alternating high and low shear stress indicate that the forward flow motion obtained for the reference plane in Figure 4 is not present along the entire posterior wall. Furthermore, as the vortex center moves downstream at the end of the pulse, it encloses the forward moving jet, resulting in regions of alternating negative and positive velocity gradients along the posterior wall. This explains why the maximum wall shear stress is obtained downstream of the midsection towards the distal neck of the aneurysm. Although the cardiac pulse imposes a low inlet velocity during late diastole, these regions of coexisting high and low wall shear stresses lead to moderate wall shear stress gradients, for which previous authors have predicted elevated rates of platelet deposition and predisposition to thrombus formation.

![Figure 6](image_url)

**Figure 6.** Anterior wall (left) and posterior wall (right) views of normalized wall shear stress surface distribution for AAA model #1 at (a) $t = 0.31$ s and (b) $t = 1.00$ s.

**CONCLUSIONS**

Finite element numerical simulations of unsteady, incompressible, homogeneous, Newtonian blood flow in patient-specific aneurysm models have been presented. The contribution of this work is the quantification of vortex dynamics and flow-induced stress distributions in individual aneurysms in the absence of intraluminal thrombus.

The vortex dynamics induced by pulsatile blood flow in individual aneurysms is patient-specific and is determined by the shape and size of the aneurysm. The following four flow phases illustrate the patient-specific flow dynamics:

- **Systolic acceleration** involves downstream ejection of the residual vortex left from the previous cycle, resulting in a completely attached flow pattern.
- **Systolic deceleration** is characterized by flow separation at the proximal neck of the aneurysm, single-vortex growth at the midsection and its translation downstream. Flow separation always occurs along the lateral-anterior wall, at which the transverse dimension of the aneurysm is maximal.
- **Early diastole** is characterized by a weakening of the vortex left from systole. Forward flow motion is increased along the lateral-posterior wall.
• Late diastole is the phase where the most significant flow disturbance takes place, largely influenced by the low-velocity recirculation region inside the aneurysm. Downstream vortex translation leads to enclosing of the forward flow stream by the vortex.

Flow-induced pressure along the inner wall of a rigid AAA model does not vary significantly. The time-varying pressure gradient imposed by the inlet boundary condition drives the flow. However, the irregular shape of the aneurysm does not yield large normal forces on the wall or nonuniform pressures. The distribution of wall shear stresses in patient-specific aneurysm models is quite complex. Peak wall shear stress is localized upstream of the proximal neck and is 64 times higher than for a normal abdominal aorta subject to the same time-average flow conditions. Preferential bulging along the anterior surface yields low shear stresses throughout the cycle. During late diastole, the posterior wall exhibits alternating regions of negative and positive wall shear stress, leading to moderate levels of wall shear stress gradients. It is not known, however, what is the potential role of spatially- or time-varying shear stresses in AAA pathophysiology or with regards to the onset of rupture.

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