Refinements in Mathematical Models to Predict Aneurysm Growth and Rupture

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ABSTRACT: The growth of aneurysms and eventually their likelihood of rupture depend on the determination of the stress and strain within the aneurysm wall and the exact reproduction of its geometry. A numerical model is developed to analyze pulsatile flow in abdominal aortic aneurysm (AAA) models using real physiological resting and exercise waveforms. Both laminar and turbulent flows are considered. Interesting features of the flow field resulting from using realistic physiological waveforms are obtained for various parameters using finite element methods. Such parameters include Reynolds number, size of the aneurysm (D/d), and flexibility of the aneurysm wall. The effect of non-Newtonian behavior of blood on hemodynamic stresses is compared with Newtonian behavior, and the non-Newtonian effects are demonstrated to be significant in realistic flow situations. Our results show that maximum turbulent fluid shear stress occurs at the distal end of the AAA model. Furthermore, turbulence is found to have a significant effect on the pressure distribution along AAA wall for both physiological waveforms. Related experimental work in which a bench top aneurysm model is developed is also discussed. The experimental model provides a platform to validate the numerical model. This work is part of our ongoing development of a patient-specific tool to guide clinician decision making and to elucidate the contribution of blood flow-induced stresses to aneurysm growth and eventual rupture. These studies indicate that accurately modeling the physiologic features of real aneurysms and blood is paramount to achieving our goal.

KEYWORDS: aneurysm; laminar; mechanical hinge; non-Newtonian; turbulent flow

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INTRODUCTION

Aortic aneurysms are responsible for more than 30,000 deaths yearly and diseases of the aorta are the 14th leading cause of death in the United States.[1,2] Most aortic aneurysms occur in the infrarenal abdominal aorta. Four principal causes of aortic aneurysms have been identified as follows: (1) proteolytic degradation of aortic wall connective tissue, (2) inflammation and immune responses, (3) mechanical wall stress, and (4) molecular genetics.[3] While the epidemiology and treatment of infrarenal abdominal aortic aneurysms (AAAs) have been well described, the mechanisms leading to accelerated aneurysm growth and eventual rupture remain poorly understood. From a flow mechanics standpoint, one can predict that changes in the geometry of an aneurysm will alter blood flow patterns and hemodynamic stresses within the aneurysm and aortic wall. Hemodynamic stresses associated with (or leading to) changes in the aortic wall, which affect its mechanical integrity, are important predictors of aneurysm dilation and the subsequent risk of rupture.

Intervention, either by direct surgical resection and grafting or by endovascular grafting, is indicated to treat AAAs when the maximum diameter exceeds 5 cm, although smaller aneurysms still have a significant risk of rupture. Previous research has identified the need to consider other predictors of AAAs rupture than the traditional measurement of aneurysm diameter such as thickness of its wall, herniation of soft plaque through the elastic coats of the aneurysm, magnitude and extension of the intraluminal thrombus, and local stress concentrators due to rigid calcium plaques. These material elements can be identified and measured by computational techniques from data obtained from standard computerized tomography (CT) scans of the aorta. The contribution of these elements to the pulsatile mechanics of the aneurysm wall can be simulated by numerical analysis (finite element method). The study of hemodynamic stresses acting on AAA walls to predict the likelihood of AAA rupture using computational techniques is better than the current practice of periodic measurement of the diameter changes in the AAA by ultrasound or CT. Numerical simulations allow for the study of conditions that are difficult or impossible to measure directly in humans or in animal models of AAA.

We propose that the stresses generated by the viscous and inertial forces associated with blood flow may be responsible for the changes in the wall that result in thrombus deposition and dilation. These flow-generated stresses can be modeled using fluid finite element analysis techniques. The much larger mechanical stresses imposed on the wall by the pulsatile pressure and the response of the wall—with areas of different elastic and deformation characteristics-–determine the site of rupture. These larger stresses and the response of the wall to them can be modeled using solid mechanics numerical techniques. These numerical predictions of the risk of rupture of an AAA can provide invaluable clinical help: the mortality and severe complications attending the surgical or

endovascular repair of certain types of aneurysms can be very high (30%) in patients with significant concomitant conditions such as emphysema or renal failure. The numerical analysis of the distribution of wall stresses within AAAs can assist in the clinical management of AAA patients by predicting the risk of rupture over time and permitting risk/benefit assessment for intervention or observation.

Addressing the smaller stresses derived from the viscous and inertial components of the flow of blood, we noted that the studies that have been published have used finite element analysis assuming laminar flows,[4–6] Newtonian behavior of blood, and idealized AAA geometries. Collectively, these studies demonstrated that increased fluid shear stress at various points along the aortic wall results in local changes in wall pressure and flow patterns that may result in dilation and deposition of thrombus. However, these published studies have not considered the effects of turbulence and non-Newtonian behavior of blood on the mechanics of the aneurysmal wall. Turbulence, induced by sudden expansion of the flow stream, generates additional stresses on the aneurysm wall. These additional stresses result in wall vibration and may be responsible for further wall dilation, and eventually greater turbulence, possibly a self-perpetuating mechanism for aneurysmal growth. Thus, turbulent flow should be considered in the study of aneurysms because it does exist in human aneurysms and by increasing mechanical wall stress, it would influence the rate of dilation of the wall.

Non-Newtonian blood characteristics should also be considered in the study of aneurysms: they exist in regions of low shear rates such as those of flow separation. Finally, patient-specific geometries of AAAs should be used for clinically meaningful and accurate results. The fact is that most physical and computational studies have been carried out in idealized models of AAAs that do not take into account real geometries and flow characteristics of human AAA.

GOVERNING EQUATIONS AND BOUNDARY CONDITIONS

Incompressible, homogeneous, non-Newtonian flow in a rigid-walled aneurysm was considered as depicted in FIGURE 1. The boundary conditions for the velocity are: (1) no-slip at the walls, (2) zero radial velocity at the inlet, and (3) zero velocity gradients at the outlet. A waveform corresponding to resting and exercise flow conditions was used at the inlet to approximate *in vivo* measurements in the abdominal segment of the human aorta.[7,8] A finite element formulation based on the Galerkin method is employed to solve the governing equations using Fidap software (Fluent, Inc., Lebanon, NH) subject to the boundary and initial conditions for this study.

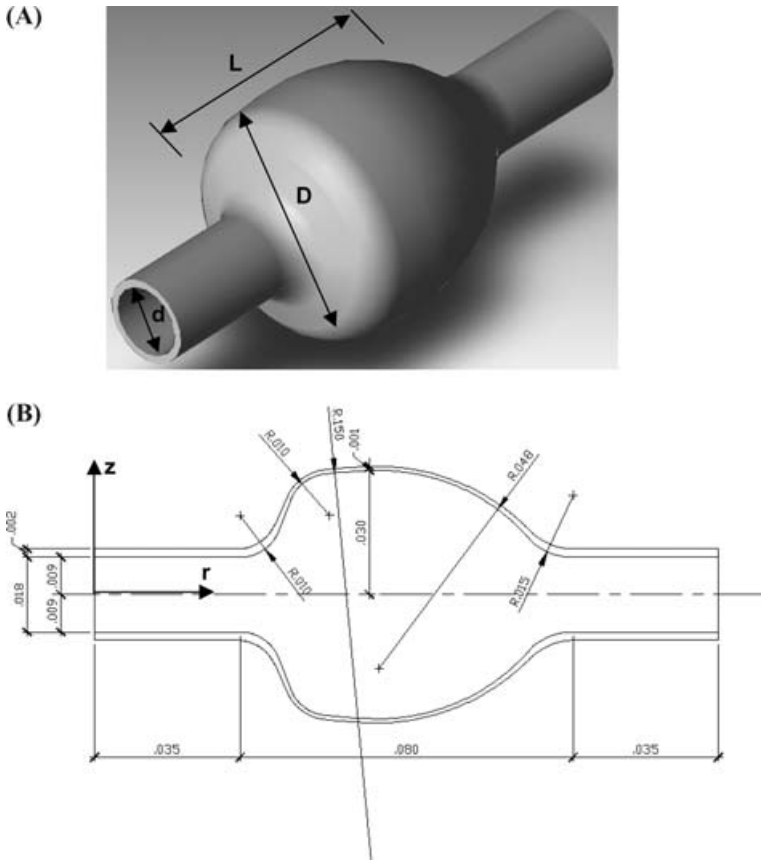

FIGURE 1. Aneurysm model and coordinate system ($d = 18$ mm, $D = 60$ mm, $L = 80$ mm). r is the radial dimension, X is the axial position, and S is a position along the aneurysm wall length.

METHODS

The combination of κ-ε model with specialized elements for near-wall modeling was used to simulate the turbulent characteristics of convective flow. The Boussinesq eddy–viscosity model was also imbedded. We accounted for the non-Newtonian behavior of blood by the Carreau equation for viscosity. The geometry of the flow mesh was modified to represent the shape of aneurysms more accurately as they occur in humans. To validate every operator imbedded in the model we constructed a flow rig with the same dimensions and operating parameters that are encountered in humans. In the testing segment of the rig we have the capability of collecting accurate data on pressure, flow velocity and

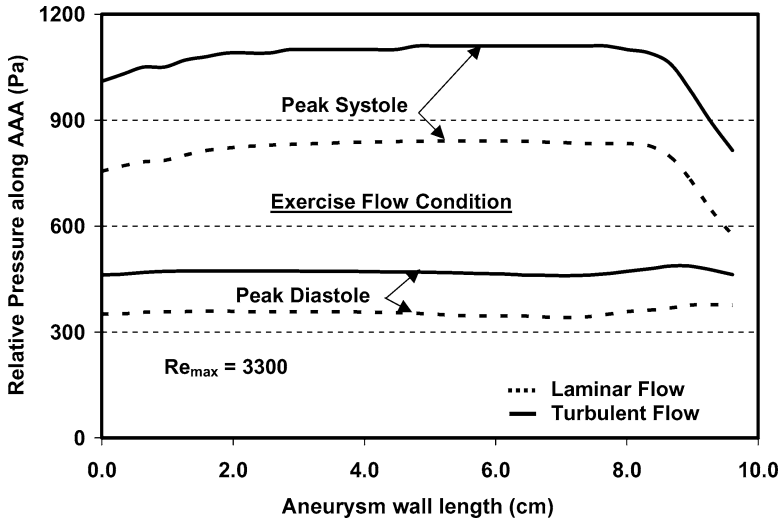

FIGURE 2. Comparison of the (**A**) relative pressure along AAA between laminar and turbulent flow conditions ($d = 18$ mm, $D = 80$ mm, $L = 80$ mm).

distribution, flow patterns (laser velocimetry); these have the same geometry as the aneurysm and operate according to the same numerical model. This permits a step-by-step validation of the assumptions imbedded in the numerical model.

RESULTS AND DISCUSSION

The significance of turbulent flow on the relative pressure acting along the aneurysm compared with laminar flow using exercise flow condition is shown in FIGURE 2. Pressure values presented in this article are relative values (i.e., excess pressures) and correspond to the difference between the instantaneous pressure acting on the AAA wall and the instantaneous pressure at the exit of the computational model. This figure demonstrates that turbulent flow exhibits higher values of pressure acting on the AAA during peak systole and peak diastole than those seen with laminar flow. Turbulent flow, compared with laminar flow, increases the mechanical stress on the aneurysmal wall AAA that could mediate its further dilation and create conditions (flow separation) that enhance thrombus deposition. FIGURE 3 demonstrates the effect of resting and exercise flow conditions on the fluid shear stress on the aneurysm. Exercise waveforms generate larger fluid shear stresses than resting waveforms with peak values found at the distal half of the AAA. The rapid oscillating nature of the wall stresses over a long period of time

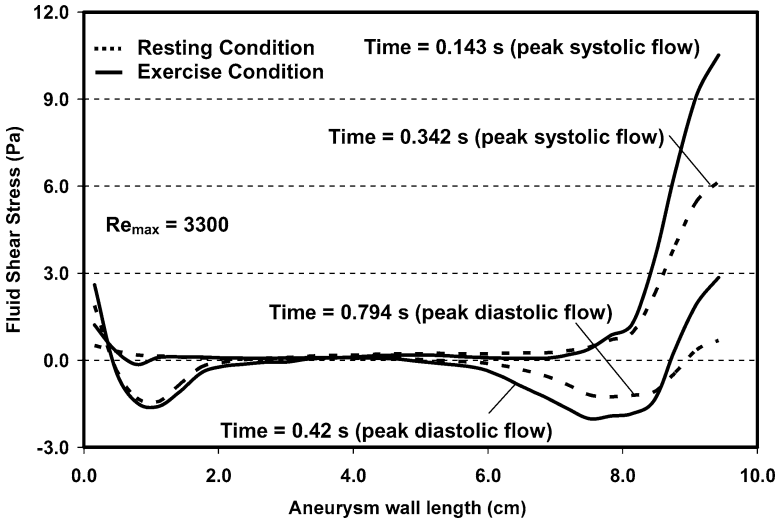

FIGURE 3. Comparison of fluid shear stress between rest and exercise flow conditions ($d = 18$ mm, $D = 80$ mm, $L = 80$ mm).

may alter the strength of the arterial wall and facilitate the growth of the aneurysm.

Solid mechanics analysis is carried out using ANSYS software to obtain the deformed shape and the stresses on the aneurysm subject to peak systole pressure are shown in FIGURE 4. The analysis demonstrates a mechanical hinge

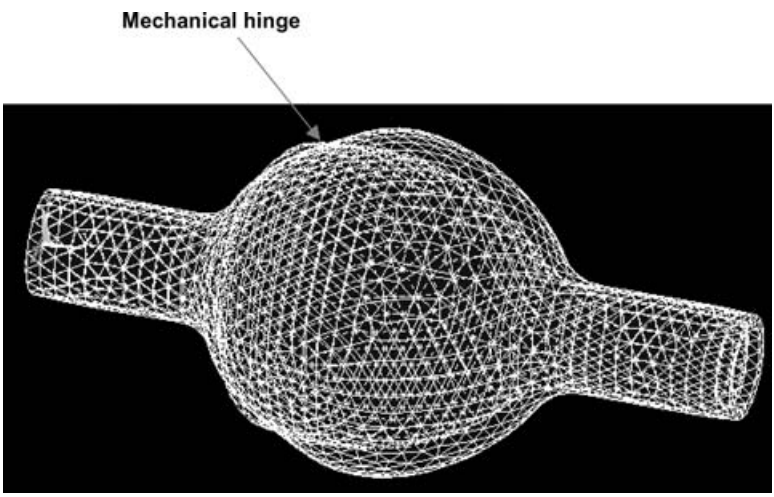

FIGURE 4. Deformed shape of an aneurysm with compliant distal end.

at the junction between the proximal, less distensible area and the distal, more distensible area of the aneurysm. This suggests a mechanism of mechanical stress similar to that found at the junction of a two-phase material. This area is where ruptures most frequently occur in human AAA.

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