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Carotid Artery Modeling Using the Navier-Stokes Equations for an Incompressible, Newtonian and Axisymmetric Flow

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Abstract

This paper describes two-dimensional (2D) structural and hemodynamics model of the carotid artery and its bifurcation using computational software (CS). The Arbitrary Lagrangian Eulerian (ALE), which was introduced in a finite element system, was utilized as a numerical technique. The structural modeling of the carotid arteries about the bifurcation area was constructed from computed tomography (CT) scans using computer-aided design (CAD) and the Lagrangian formulation was used for the structural domain. The blood was considered as an incompressible Newtonian fluid, and Eulerian reference was applied for its domain. Coupling of the reference systems was carried out on arbitrary computational grid permitting numerical modeling of hemodynamics as governed by 2D axially symmetric incompressible Navier-Stokes equations (NSE). The results for hemodynamic simulations were compared with the physiological blood velocity obtained using the Doppler ultrasound instrument.

Keywords: CARDIOVASCULAR MODELLING, CT SCAN, FINITE ELEMENT METHOD, HEMODYNAMICS

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death around the world, claiming an average of 17.1 million lives a year [1]. This mortality is generally due to disorders of the heart and blood vessels arising from either abnormal blood flow, failure of vascular area or heart damage. These include atherosclerosis,

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congenital heart disease, coronary artery disease, hypertension, aneurysm and other ischemic heart disease. Hence, research works on medical methods for prevention, diagnostics and cure have been conducted to decrease fatality due to CVDs. Since most studies on diagnosing cardiovascular system (CVS) diseases from clinically measured data are difficult and invasive [2], various research employing computational methods of blood flow determination have been done to understand the complexity of hemodynamics and geometric parameters of CVS. The use of physiological models and blood flow simulation could aid in the development of alternative methods capable of non-invasive detection and evaluation of the aberrations and general dynamics of the blood in the vascular areas, decreasing dependence on risky methods such as cardiac catheterization.

The tasks of developing and integrating CVS models require specific computational tools that provide compatibility of models and physiological data. The current methods for model development necessitates foundations on intricate mathematics and physics, and do not readily facilitate computation of complex systems. Consequently, CVS modeling is limited to available boundary conditions and parameters [3]. The finite element methods for the systems are computationally prohibitive and are therefore compromised using coupled parameters [4, 5]. For these reasons, simplification with respect to a particular vascular area of interest is done for 2D CVS model of this study. Although vascular areas such as arteries assist in the simplification of the CVS model, only few studies [6, 7] are available with comprehensive discussion on structural modeling and hemodynamics. In this research, the blood flow in the constructed internal carotid artery (ICA) around the bifurcation area was calculated using Navier-Stokes equations (NSE). Studies that utilized ICA as the vascular system for flow model are very limited but certain works on vertebral arteries [8] have been conducted utilizing the literature anatomical parameters for geometry, and the same equation for Newtonian blood flow determination. The equations have been used in tandem with the continuity equation to describe flows in large vascular areas and coupled with a structural equation for multi-dimensional simulations. The goal of the study is to produce simple simulation implementation that realistically depicts the hemodynamics in 2D using axisymmetric NSE in the constructed vascular structure.

2. Materials and Methods

As supported by recent studies [9-11], ALE is used for most computational blood flow determination in models such as tubes and bifurcations. The morphology of the vascular structure is constructed using CAD software, while blood flow pattern is determined using incompressible 2D NSE given in *(1)* and *(2)*, where u and v are velocities at x and y directions, p is pressure, η is the viscosity of the blood and t is time. The model was created using the axial geometry of the carotid artery from CT scan images.

$$\rho\left(\frac{\partial u(x,t)}{\partial t}+u(x,t)\frac{\partial u(x,t)}{\partial x}+v(y,t)\frac{\partial u(x,t)}{\partial y}\right)=-\frac{\partial p}{\partial x}+\eta\left(\frac{\partial^2 u(x,t)}{\partial x^2}+\frac{\partial^2 u(x,t)}{\partial y^2}\right) \quad (1)$$

$$\rho\left(\frac{\partial v(y,t)}{\partial t}+u(x,t)\frac{\partial v(y,t)}{\partial x}+v(y,t)\frac{\partial v(y,t)}{\partial y}\right)=-\frac{\partial p}{\partial x}+\eta\left(\frac{\partial^2 v(y,t)}{\partial x^2}+\frac{\partial^2 v(y,t)}{\partial y^2}\right) \quad (2)$$

2.1. Geometry Modeling

The structure modeling of the ICA in 2D was done using various softwares. The 70 CT scan images from DR Systems were used to generate vectors and composite geometries. The subject's file contained 893 images and 665 slices of which the carotid artery of sufficient contrast and clear geometrical boundary were selected. Each CT scan image was individually converted to vector as DXF file and constructed using AutoCAD to eliminate other points and boundaries not identified as ICA. The generated 2D image was processed as a composite object in FEMLAB and the ALE method was used to construct the computational mesh assuming simplified geometries using a mesh generation command.

2.2. Flow Modeling

The blood flow was simulated numerically using simultaneous coupling of the reference systems with finite element and ALE as bases for applying the moving mesh method by mesh displacement. The PDE for mesh movement was based on continuum mechanics and the domains were identified as either solid structure or fluid. Implementation of field variables was utilized to handle mesh displacement using FEMLAB. The mesh was assumed to have isotropic and elastic behaviour similar to the refine theory of solid mechanics. These properties were incorporated into the FEMLAB model. The CA was programmed to have a Neo-Hookean hyper-elastic property using arterial wall properties (Table 1). Using the generalized Hooke's law, the stress strain relationship in the linear elastic behaviour was numerically determined by Cauchy stress tensor. This relationship was used for the PDE equation governing mesh displacement and to control the strain of the reference grid, tracking mesh movement. The method determined the displacements using surface tractions given by Poisson's ratio, body forces and density.

Table 1. Properties of arterial wall [12]

Mesh Displacement Field Variable	Values
Density	9.6×10^2 kg/m^3
Coefficient of hyper-elasticity	6.2×10^6 N/m^2
Bulk modulus	1.2×10^8 N/m^2
Poisson Ratio	0.45
Elastic modulus	1.017

3. Results and Discussions

The fluid flow was studied using the 2D composite geometric constructions of a representative vascular area obtained from the head and neck CT scan images of patient X. Results of simulation runs for regular and pathological flow patterns assuming blood as an incompressible fluid were done to develop a general idea of the hemodynamics in ICA around its bifurcation.

3.1. Non-pathological Flow

The regular flow pattern for a tube and curved domain was examined for an incompressible fluid using the blood properties, 1060 kg/m^3 for density and 0.005 N·s/m^2 for dynamic viscosity. The inflow was specified as parabolic and the outlet boundary was assumed to have zero external force. The maximum velocity field obtained was 0.25m/s and the flow patterns were shown in Fig.1.

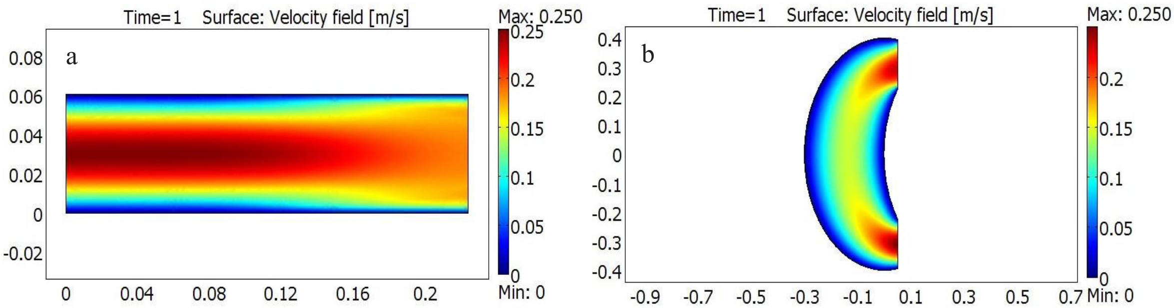

Fig.1. Blood velocity profile for laminar flow in (a) tubular domain and (b) curved domain

3.2. Pathological Flow

Irregular flow characterizes turbulence, and this occurs in areas where the laminar stream of blood flow is disrupted either by a blockage or constriction. In turbulent flow, the fluid streams mix radially and axially producing a non-parabolic velocity profile. Prototypes of these flows were modeled using simulated pathological vascular areas with corresponding deformations. Using the same parameters for laminar blood flow, the results are analyzed and presented in Fig. 2. The constricted area (Fig. 2a) was observed to have a maximum velocity of 0.49m/s at the centre of the constriction. While for the dilated area (Fig. 2b), the highest velocity of 0.26m/s, was observed along the centre of the inlet boundary where it dissipated as it reached the inflated chamber which has a significantly larger diameter than the initial blood vessel dimension.

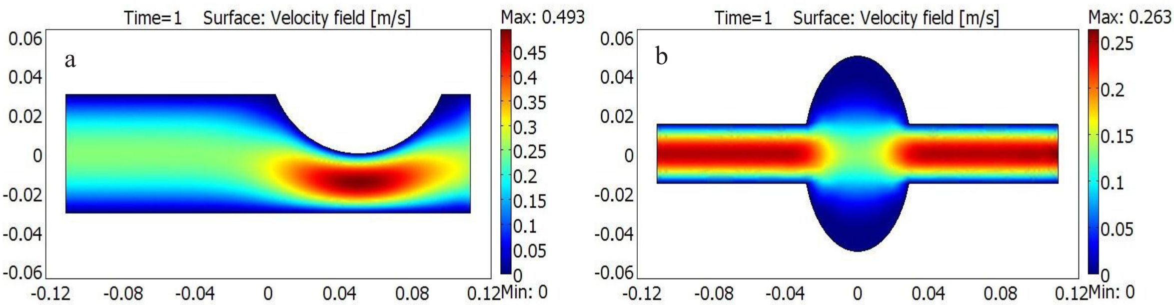

Fig. 2. Blood velocity field with irregular flow pattern resulting from (a) vasoconstriction and (b) vasodilation

3.3. Flow Model of the ICA

The computational mesh of the constructed CA and bifurcation area was optimized to 1188 elements. Results of flow simulation using constant blood properties of 1060 kg/m^3 for density and 0.005 N·s/m^2 for dynamic viscosity at different points in time were recorded for the bifurcation area and carotid arteries. Relatively high velocities are observed in the areas of bifurcation where there is narrowing of the carotid arteries. The blood velocity field results at different time steps assuming parabolic inlet velocity were done and a representative was shown in Fig. 3. The flow pattern of the blood is regular in the tubular areas while irregular in areas with changes in dimensions such as constriction and dilation. The velocity field was observed to decrease in areas of enlargement, and increase in areas of narrowing with a maximum velocity field of 0.94m/s. The continuity of the flow pattern can be observed alternately between external and internal carotid artery models.

The blood flow profile, as illustrated, indicated deformation to the reference geometry as influenced particularly by the Neo-Hookean elasticity. The affected areas based on the results were the walls and boundaries at which the velocity was maximum. The profile was observed in time step and it can be deduced from the data that acceleration of the fluid at stenosis or constricted areas as related to pressure causes either vasodilation or vasoconstriction of the blood vessel. Fluid flow velocity in vasodilated areas can be expected to be lower resulting in a thicker boundary layer at the walls. In non-laminar flow, the eddies in the inner area can be much larger than those nearer the walls. The separation of streamlines indicates the recirculation regions which have the potential to expand with increasing Reynolds number. Evaluation of the flow in the geometry at different initial velocities was also done to aid in understanding the recirculation of flow and the generation of eddies as a function of the approach velocity. From the DUS data, experimentally the velocity at the carotid area ranges from 0.18 to 0.94m/s for laminar flow. The results at different time steps were obtained and the velocity computed even at bifurcation area was within range of the DUS data. Therefore, the blood flow velocity range in the simulations is considered laminar corresponding physiologically to the velocity profile of blood flow in a non-pathological blood vessel.

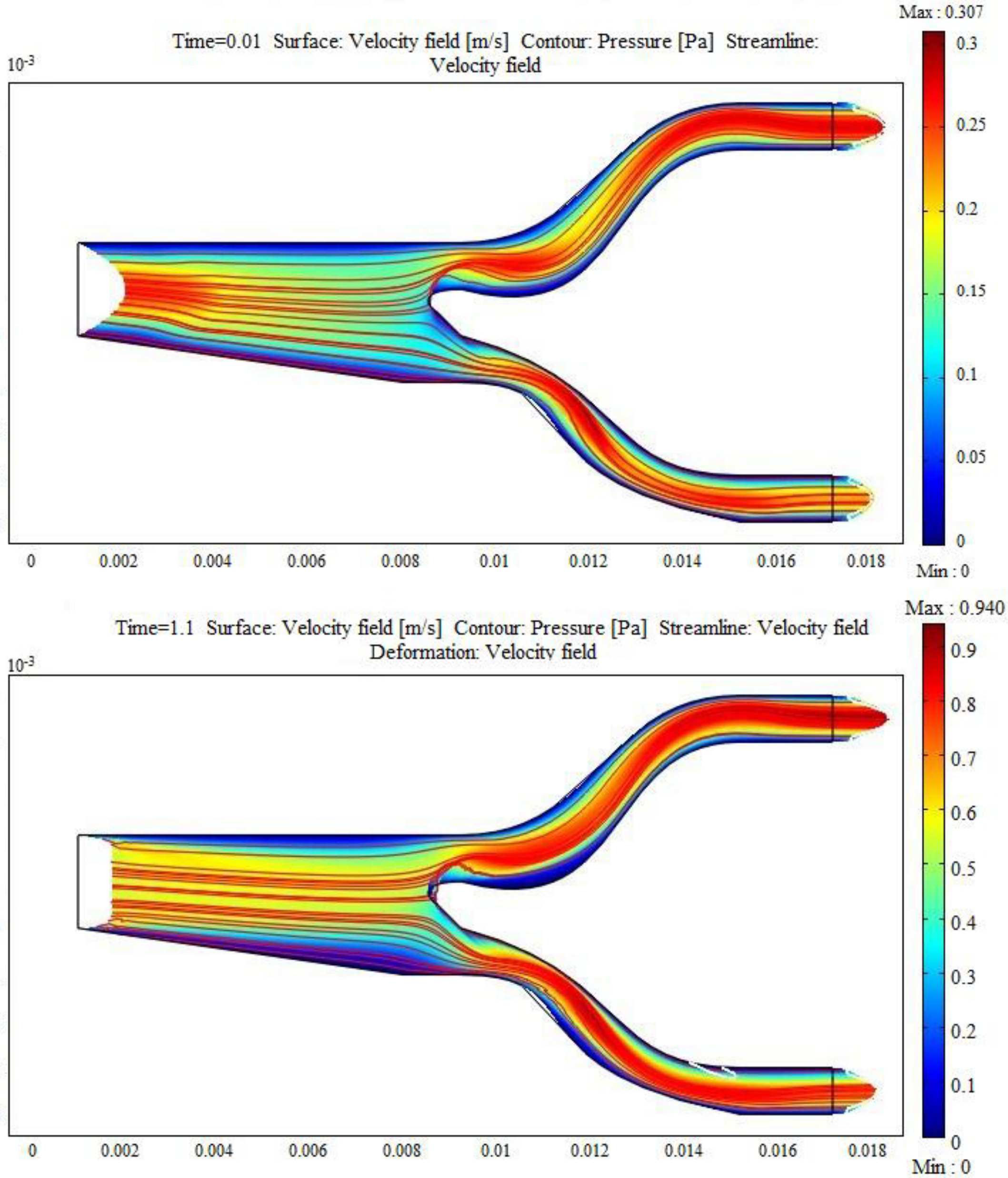

Fig. 3.Simulated blood velocity field around the bifurcation of carotid artery at different time steps

4. Conclusion

This study presented a simplified model of the CVS, describing the blood flow profile in 2D using axisymmetric NSE for incompressible Newtonian fluid. The pattern of the blood flow was explored using computational methods for numerical simulations. The Arbitrary-Lagrangian-Eulerian method was used to solve the fluid structure interaction problem incorporating the structural properties of the artery and the fluid properties of the blood. The profile of the flow at different geometrical simulations and parameters were conducted to understand the relationships of the parameters to the hemodynamics of flow. Simulations of regular flow patterns of the blood were done for tubular and curved domains. Laminar flow was observed for both prototype simulations obtaining a maximum velocity field of 0.25m/s. The irregular flow patterns or pathological flow were obtained using simulations of vasoconstriction and vasodilation. The maximum velocity field was 0.49m/s for the vasoconstriction prototype simulation, and 0.26m/s for vasodilation prototype simulation. Simulation of the flow pattern in the carotid artery about the bifurcation was done by applying the same structural properties and fluid parameters of the blood, boundary conditions and parabolic velocity profile at the inlet with no external forces at the outlet of the external and internal carotid arteries. The 2D model showed similarities with the physiological data obtained from patient X using the Doppler Ultrasound for which the velocity field range from 0.18m/s to 0.94m/s.

The solutions investigating the interaction between the theory and experiments were iterative. The boundary valued problems and complexity of the equations posed difficulty in determining convergence. The nonlinearity of the problem and multifaceted modelling necessitated tedious optimization to yield the desired agreements of new analyses based on the initial point. Regardless, a method for modelling of vascular area such as CA was done using AutoCAD and DR Systems. And the study produced a valid simulation of flow numerically solved using computational software, FEMLAB.

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