NUMERICAL SIMULATION OF BLOOD FLOW IN A STENOTIC ARTERY

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SYNOPSIS

The purpose of this paper is to develop a numerical computational methodology for the simulation of blood flow in a stenotic artery. As a first approach biochemical and mechanical interactions between blood and vascular tissue are neglected and the flow is considered through a rigid tube. Considering a non-Newtonian model numerical results are obtained for velocity and shear stresses distributions.

INTRODUCTION

In many developed countries, diseases as heart attack or cerebral infarction are top of death causes. Altered flow conditions in blood vessels, due to branching, bifurcations and flow-reversal zones play an important role in the development of arterial diseases. When an atherosclerotic stenosis in a coronary or carotid artery prevents the blood flow, it might cause a heart or a brain attack respectively; otherwise, if the surface of the stenosis is damaged, a fragment of the atherosclerotic plaque or a blood clot can emboli with blood flow and occlude a more distal cerebral artery or coronary artery. Although blood flow is normally laminar, the periodic unsteadiness or pulsatile nature of the flow makes possible the transition to turbulence when the artery diameter decreases and velocities increase.

It is very important to know how blood is flowing in those areas, which could help doctors or surgeons to make a diagnosis or to plan a surgery. In recent years, the development of computational techniques in fluid dynamics together with increasing performances of the hardware, found a promising field of application in the framework of vascular research (Taylor et al., 1998, and Quarteroni et al., 2003). Due to new technology of medical imaging data acquisition such as computed tomography, angiography or magnetic resonance imaging (MRI), it has become feasible to construct three dimensional models of a blood vessel. Measuring techniques such as Doppler ultrasound have improved to provide accurate information on the flow fields. Therefore, carrying research on shape acquisition together with computational techniques in fluid dynamics is possible and it is important to develop circulatory simulation codes such as blood flow simulation.

Recent studies of the numerical simulation of steady and pulsatile blood flows in stenotic vessels have been presented. Nadau and Sequeira (2007) investigated the qualitative numerical behavior of the flow through a smooth stenosed channel for a range of shear-thinning viscosity parameters using a hybrid finite element–finite volume method. A numerical analysis of the blood flow phenomena using the finite element method approach and a geometrical model of a smooth stenotic artery is the primary aim of the study present here.
MATHEMATICAL MODEL

Due to the complexity of the cardiovascular system, a preliminary analysis aiming suitable simplifying assumptions for the mathematical modeling process is needed (Himeno, 2003).

The blood is considered an incompressible fluid in a bounded domain. The fluid flow is governed by the equation of Navier-Stokes,

\[
\rho \left( \frac{\partial \mathbf{v}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v} \right) = -\nabla p + \nabla \cdot \mathbf{T} + \mathbf{f} 
\]

(1)

where \( \mathbf{v} \) is the flow velocity, \( \rho \) is the fluid density, \( p \) is the pressure, \( \mathbf{T} \) is the deviatoric stress tensor, and \( \mathbf{f} \) represents body forces (per unit volume) acting on the fluid. This equation results from the application of the principle of mass conservation. Considering blood flow an incompressible non-Newtonian flow and neglecting body forces, the equation of continuity and the Navier-Stokes equations become:

\[
\nabla \cdot \mathbf{v} = 0 \\
\frac{\partial \mathbf{v}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v} = -\frac{1}{\rho} \nabla p + \mathbf{v} \nabla^2 \mathbf{v} 
\]

(2)

The upwind-difference method is used for the convective terms and the first order Euler implicit method is used for the time-differencing term.

In order to compute the velocity and pressure fields the above equations must be provided with initial conditions, \( \mathbf{v} = \mathbf{v}_0 \) at time \( t_0 \) and suitable boundary conditions. If the compliance of the vascular tissue is not accounted then the boundary can be considered rigid and it is necessary to impose Dirichelet boundary conditions, \( \mathbf{v} = 0 \) for all points of the wall boundary.

In this work elements with zero thickness interface are used to perform boundary conditions simulation. The approximation of the governing equations is performed applying a mixed penalty method in order to model fluid incompressibility. A smoothing technique was used to get continuous fields for pressure and deviatoric stresses.

RESULTS

The numerical simulations were conducted using a previous developed code (António et al., 2004). A numerical example of blood flow through a smooth stenosed channel has been performed in order to show the application of the developed software to blood flow in small size vessels. The simulation is carried out under steady flow conditions.

In Figure 1 shows the geometry of the stenosis considered in this work and the finite element mesh of the circular pipe used to model the artery are presented. The simulation considers \( R_0 = 1 \text{cm} \) for the radius of the undamaged channel and \( L = 2R_0 \) for the length of the stenosis profile. The stenosis presents a cross sectional 30% area reduction, which corresponds to the case of a relatively mild occlusion, leading to local small increasing Reynolds’ number.

In this steady flow simulation a long (30 \( R_0 \)) outflow tube is considered in order to avoid the influence of outflow boundary conditions on the flow patterns in the stenotic region. The Dirichelet conditions of a parabolic distribution of the velocities are:
for the inflow and outflow boundary sections respectively.

The dominant non-Newtonian behavior of blood, a suspension which has aggregate particles, is caused by the shear thinning effect as increased shear rates where the decrease of blood viscosity results from the destruction of the erythrocyte aggregates. The viscosity is empirically obtained using Casson law for the shear stress relation (Perktold et al., 1991). Considering \( D_{II} \) the second invariant of the strain rate and \( c \) the red cell concentration, the shear stress \( \tau \) given by the generalized Casson relation is:

\[
\sqrt{\tau} = k_0 + k_1(c)\sqrt{2D_{II}}
\]

and the apparent viscosity \( \mu = \mu(c, D_{II}) \) a function of the red cell concentration is,

\[
\mu = \frac{1}{2\sqrt{D_{II}}\left(k_0 + k_1(c)\sqrt{2D_{II}}\right)^2}
\]

where parameters \( \mu_0 = 0.032, k_0 = 0.6125 \) and \( k_1 = 0.174 \) were obtained fitting experimental data and considering \( c = 45\% \) (Perktold et al., 1991).
Figure 2 shows the computed axial velocity field around the stenosis. The axial velocity has maxima values concentrated in the core of the stenosis. Flow stagnation can be observed at the end of the flow acceleration after the peak at the stenosis core.

In order to study the flow velocity profile in the area around the stenosis three cross-sections A, B and C were considered as detailed in Figure 1. The axial flow velocity profiles of these sections are presented in Figure 3, and the maximum observed axial velocity is found in section B. Section C reports the data at the stenosis center. It is interesting to notice that the blood axial velocity does not take null values near the vessel wall.

![Figure 3 Axial flow velocity profiles on sections A, B and C.](image)

Figure 4 shows the computed shear stress axial field around the stenosis. In the stenosis region, the higher shear stresses occur near vessel wall. The region after the stenosis where flow stagnation occurs corresponds to relatively low wall shear stress and consequently to the localization of possible formation of advanced atherosclerotic plaques.

![Figure 4 Stress component $\tau_{xy}$ field around the stenosis.](image)
CONCLUSIONS

The outcomes of this work will contribute to characterize the physiology of the human circulatory system, to model steady and pulsatile blood flow and to detect turbulence caused by changes in flow velocities and vessels diameter.

In further studies the investigation of more complex and realistic scenarios will be considered. The research will allow identifying with scientific support the areas that should be altered by reconstruction and revascularization operations leading to correction of form and function in a most effective way.

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