

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/267970518>

Mathematical Model of Blood Flow in Carotid Bifurcation

Article · October 2009

CITATIONS

0

READS

247

3 authors, including:



Vera Gramigna

Institute of Bioimaging and Molecular Physiology-CNR,Catanzaro, Italy

32 PUBLICATIONS 64 CITATIONS

[SEE PROFILE](#)



Gionata Fragomeni

Universita' degli Studi "Magna Græcia" di Catanzaro

94 PUBLICATIONS 124 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



Mathematical models of cardiovascular system [View project](#)



Hollow Fibre Membrane Bioreactors [View project](#)

Mathematical Model of Blood Flow in Carotid Bifurcation

E. Muraca^{*1}, V. Gramigna¹, and G. Fragomeni¹

¹ Department of Experimental Medicine and Clinic, Magna Graecia University Catanzaro, ITALY

^{*}Corresponding author: Campus S. Venuta, Germaneto – 88100 Catanzaro, e.muraca@unicz.it

Abstract: The goal of this research is to provide the medical staff with a numerical system assessment of wall shear stress in carotid bifurcation. Through this model, it will be fundamental to investigate the stress state properties of the surface in contact between the plaque and the artery, and study the geometric relationship between the bifurcation angle and fluid structural properties. The finite element simulation is the basis for a predictive model determining which the parameters responsible for arterial formation of atherosclerosis plaques. The formation of plaques can be due to Wall Shear Stress (WSS) [2]. In previous studies, a statistic technique was used to establish the maximum stiffness of arterial stretch [3]. The final purpose is to develop a method for minimizing the error between numerical and experimental data. A fluid structural model is used to examine some effects of whirling motion caused by the presence of stenosis. The experimental validation is made possible through some tests on a sample of 100 patients using Doppler and MRI techniques.

Keywords: stenosis, fluidstructural, wall shear stress

1. Introduction

Stroke constitutes the third most frequent cause of death [4] and a leading cause of long-term disability in developed countries [5]; in about 80% of cases, stroke is an ischemic disease [6]. Although clinical events re-occur in approximately 15% of cases in 2 years of follow-up [7], 3/4 of the ischemic strokes registered in a year are first clinical episodes. This focal nature of ischemic stroke is linked to the characteristic itself of the atherosclerotic process that is the cause of the disease almost in half of the patients.

Carotid atherosclerosis is locally favoured by the hemodynamic forces of wall shear stress and circumferential wall tension, the frictional and perpendicular force of blood flowing on the vascular wall respectively. This happens in

particular areas in the carotid circulation, such as the outer wall of internal carotid artery, and because of the differences among patients in the same carotid segment, as previously demonstrated.

The first part of the study aims to verify the numerical consistence of fluid dynamic Comsol model based on Navier-Stokes equations in transient regime. A fluid dynamic analysis for the vessel is performed to compute the velocity at different time instances. The mathematical model simulates the pulsating blood flow through the artery embedded in the cardiac muscle obtained by elastic constraint.

Blood flows under the influence of pulsating pressure is defined as a time varying function. The flow of blood is governed by Navier Stokes equation. Navier-Stokes equation is given by:

$$\rho \frac{\partial u}{\partial t} + \rho u \nabla u = -\nabla P + \eta \nabla^2 u + F$$

$$\nabla u = 0$$

where ρ denotes density (kg/m^3), u velocity vector (m/s), η viscosity (Ns/m^2), and P pressure (Pa). The modeled fluid is blood with viscosity $0.005 Pa/s$ and density $960 kg/m^3$.

The boundary conditions are pressure at the entrance and outlet of the artery (P_{in} and P_{out}). All other boundaries have a no-slip condition: $u=0$.

The expression for frequency is:

$$f(t) = \begin{cases} \sin \pi t & 0 \leq t \leq \frac{1}{2}s \\ \frac{3}{2} - \frac{1}{2} \cos(2\pi(t - \frac{1}{2})) & \frac{1}{2} \leq t \leq \frac{3}{2}s \end{cases}$$

The deformation of the artery is a function of vessel rigidity. To compute it, a mechanical analysis was performed by the Structural Mechanics module. The two models are interconnected to each other by boundary conditions, as the normal component of the surface force provides the coupling between them.

A simplified constant shear modulus is used that represents a measure of the rigidity of the vessel.

The arteries are considered as a hyperelastic material by a neo-hookean model.

The structural deformations are solved by using an elastic formulation and a nonlinear geometry formulation to allow large deformations.

For boundary conditions, the plaque is fixed to the bottom of the fluid channel so that it cannot move in any direction. All other boundaries experience a load from the fluid, given by

$$F_T = -n(-pI + \eta(\nabla u + (\nabla u)^T))$$

where n is the normal vector to the boundary. This load represents a sum of pressure and viscous forces. In addition, the predefined fluid load takes into account the area effect between the reference frame for the solid and the moving ALE frame in the fluid.

The coupling between the models is provided by the normal component of the surface force acting on the boundary between the blood and the artery.

We have used experimental dates to describe the distribution of rigidities on external boundaries: high rigidity in correspondence of bifurcation and low rigidity in correspondence of muscular coating.

The expression of elastic reaction is:

$$F_x = -k * u;$$

$$F_y = -k * v;$$

$$F_z = -k * w;$$

in the three different directions.

1.1 Geometry

The geometrical structure has been modelled on Rhinoceros® (Robert Mc Neel & Associates, Seattle, WA, USA). It has an internal diameter of 10 mm, a thickness of 1 mm, a length of carotid between the bifurcation and the output section of 30 mm.

In the simulations two types of subjects have been examined: healthy and pathological

subjects showing the presence of plaque in artery.

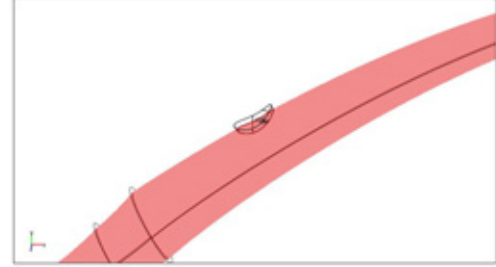


Figure 1. View of the internal carotid artery plaque

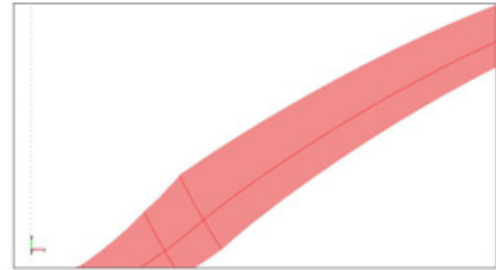


Figure 2 View of the internal carotid in absence of artery plaque

To simplify handling the plaque has the same properties of the carotid artery

1.2 Mesh parameters

The model was meshed with different densities for the individual sub-domains. The mesh for artery enables us to solve for 90119 degrees of freedom. The parameters are described in table 1:

Table 1: Mesh parameters

Number of elements	30914
Number of boundary elements	11940
Number of edge elements	763
Minimum element quality	0.2190

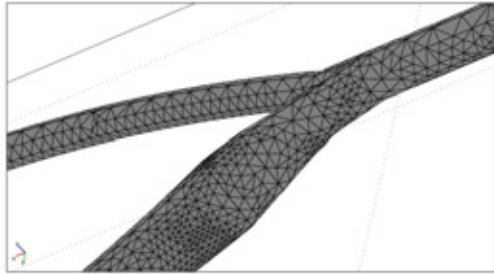


Figure 3 The mesh grid for finite element analysis used to numerically solve the Navier Stokes equations for the proposed fluid transitions

1.3 Solver settings

To solve the model ‘UMFPACK’ solver was used. This solver is a set of routines for solving unsymmetrical sparse linear systems, $A * X = B$ using the Unsymmetric MultiFrontal method. For fluid dynamic model a transient analysis was performed, while for solid stress-strain a quasi-static transient analysis type was used.

2 Results and Discussion

First-principal stress distribution (max. 254.1 kPa, mean 181.4 kPa) is shown from a patient with a systolic blood pressure of 160mmHg, severe carotid stenosis and a large lipid core located immediately below the carotid bifurcation beneath the internal carotid artery [8]. In our model the maximum value of First-principal stress is 100 KPa in absence of carotid plaque.

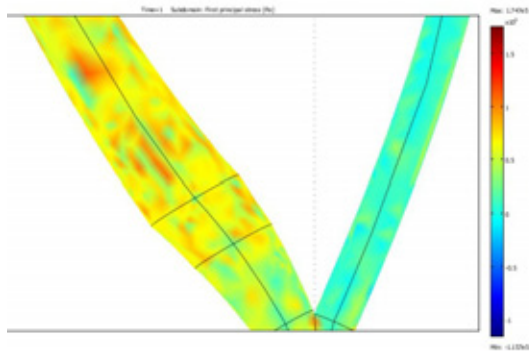


Figure 4 First Principal stress

The flow is laminar and the velocity field is parabolic. The medium velocity value found is

30 cm/sec in the artheria's buckle, a value of vorticity 2.45 1/s, 75 KPa.

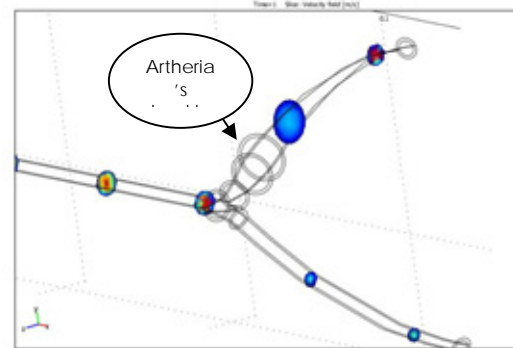


Figure 5 Velocity Field

In fig. 6 we take a portion in x-y plane of velocity field in the initial part before the bifurcation. It is a typical trend of laminar viscous fluid.

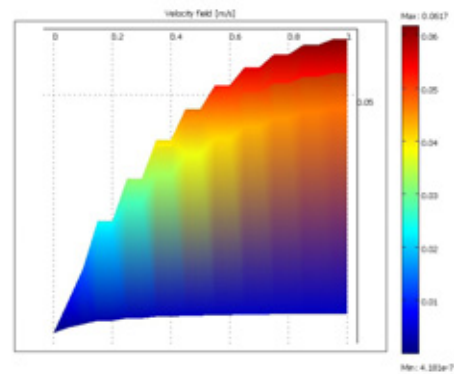


Figure 6 Portion of Velocity Field

In fig. 7 we take a view of pressure in the bifurcation. The medium value is 84 mmHg.

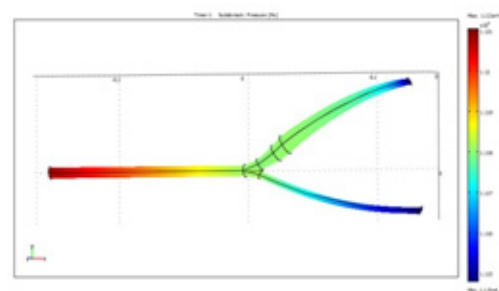


Figure 7 Pressure Field

By comparing a sample of 100 healthy patients we find deviations in the velocity and pressure field amounted to 30%.

Next goal is to make the model more robust by using a technique of optimization to identify significant value bulk module.

3 Method for the correlation between numerical and experimental model

Next activity will be to develop a method based on a tool of optimization to find some appropriate weights by the comparison with experimental analysis. By working on some mechanical units such as material stiffness it is possible to correct the numerical model.

A combined experimental/numerical study has to be carried out to calculate the 3D shear strain map.

The aim of fluid-structural model updating is to define an error that characterizes the quality of the model with respect to the experimental data and minimize it. The method to solve this inverse problem is based on the concept of the error of the constitutive relation. This error can deal with either damping effects or nonlinearities. It can also use all available experimental results.

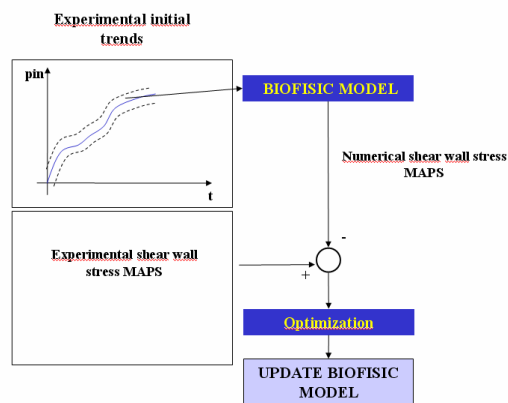


Figure 6 Schematic model of correlation between numerical and experimental model

4 Conclusion

This fluid-structural model, validated by experimental data, has been useful to determine the effects of a section reduction due to the

presence of atherosclerosis plaques in human carotid bifurcation. In particular, it has been possible to evaluate the velocity field and the pressure distribution in the blood trough the fluid dynamics analysis and the deformation of the artery walls by using the mechanical analysis. As future work, this model can be seen as a mean to predict the fluid dynamic and fluid-structural conditions that would appear in human carotid bifurcation after surgical procedure. In fact, in physiological conditions, arteries are able to adapt to the change of blood flow and pressure. Surgical procedures, useful to repair vascular diseases, can modify blood fluid dynamic with serious consequences.

References

- 1 . Ding, Z., K. Wang, J. Li, and X. Cong: Flow field and oscillatory shear stress in a tuning-fork-shaped model of the average human carotid bifurcation. *J Biomech.* 34, 12, 1555-62 (2001).
- 2 .Sun, N., R. Torii, N.B. Wood, A.D. Hughes, et al. Fluid-wall Modelling of LDL Transport in a Human Right Coronary Artery. in *COMSOL Users Conference 2006*. Birmingham (UK).
- 3 .Gadkari, T. and A. Jeremic. Mathematical Modeling of Blood Flow in the Presence of Atherosclerosis. in *COMSOL Users Conference 2006*. Boston (USA).
- 4 . Goldstein LB, Adams R, Becker K, et al. Primary prevention of ischemic stroke: a statement for healthcare professionals from the Stroke Council of the American Heart Association. *Circulation* 2001;103:163–82
- 5 . American Heart Association. Heart disease and stroke statistics –2003 update. Dallas, TX: American Heart Association; 2002.
- 6 . Bamford J, Dennis M, Sandercock P. The frequency, causes, and timing of death within 30 days of a first stroke: the OxfordshireCommunity Stroke Project. *J Neurol Neurosurg Psych* 1990;53:824–9
- 7 . Hier DB, Foulkes MA, Swiontniowski M., et al. Stroke recurrence within 2 years after is ischemic infarction. *Stroke* 1991; 22:155-61.
- 8 . Kock S., Nygaard J., Mechanical Stresses in carotid plaques using MRI based fluid interaction model, *Journal of Biomechanics* 41 (2008)

