

# Blood flow-vessel interaction in a subclavian aneurysm

Alin A. DOBRE<sup>1</sup>, Alexandru M. MOREGA<sup>\*:2</sup>

\*Corresponding author

<sup>1</sup>“POLITEHNICA” University of Bucharest, Faculty of Electrical Engineering  
313 Splaiul Independentei, 060042, Bucharest, Romania  
alin.dobre@iem.pub.ro

<sup>\*:2</sup>“POLITEHNICA” University of Bucharest, Faculty of Electrical Engineering  
Splaiul Independenței 313, 060042, Bucharest, Romania  
amm@iem.pub.ro

DOI: 10.13111/2066-8201.2011.3.4.6

**Abstract:** *This paper presents a mathematical model and numerical simulation results of the blood flow-structural interaction, which occurs in a saccular aneurysm emerging out of the left subclavian artery, using computational domains made of by medical images reconstruction. A correlation between the total force per area acting upon the artery walls by the pulsatile blood flow and the rupture probability are also investigated.*

**Key Words:** *saccular aneurysm; blood flow; numerical simulation; finite element; CT image reconstruction*

## 1. INTRODUCTION

Aneurysms are balloon-like bulges that occur in different types of arteries due to the thickening of the blood vessels' walls. The causes of these pathological formations are not yet well understood: some aneurysms are congenital while others occur there where the arteries walls withstand higher blood pressure, higher levels of cholesterol or atherosclerotic disease [1-3].

The blood vessel walls have a certain thickness adapted to body region they cross, in order to withstand the normal blood pressure.

The artery walls may be damaged or injured due to genetic conditions or trauma, giving birth to regions prone to aneurysm formation.

Also, high blood pressure, high levels of cholesterol and atherosclerotic disease are important factors, which influence the aneurysm formation, may grow larger and rupture or dissect.

A ruptured aneurysm may cause massive bleeding inside the body, while the aneurysm dissection manifests as a split in one or more layers of the artery walls causing bleeding into and along the layers of the arteries walls.

Usually, both rupture and dissection are fatal. Aneurysms could develop in any region of the body.

Yet, there are two most common types of aneurysms: intracranial or cerebral aneurysms, which occur inside the brain, and aortic aneurysms, which develop inside the aorta [5].

In this study we are concerned with the numerical simulation of the blood flow in a saccular aneurysm formation in the left subclavian artery, accounting for the flow-vessel interaction between the blood and the artery walls.

This paper is a continuation to [6].

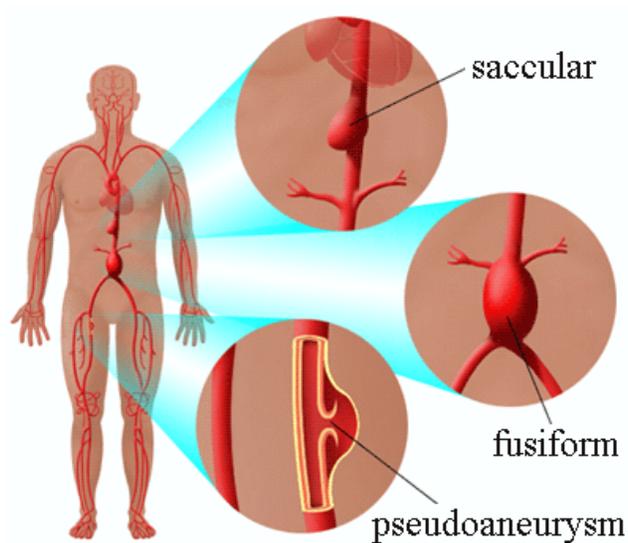


Fig. 1 – Different types of aneurysms and the body regions prone to their formation, from [4]

The computational domain is generated by image reconstruction techniques out of *Digital Imaging and Communication in Medicine* (DICOM) datasets acquired by *Computed Tomography* (CT) from [7].

Thus, more realistic 3D models of the aneurysm affected arterial network are built and then used for numerical modeling by the finite element method (FEM). The pressure field associated to the blood flow is used to couple the hemodynamic model to the structural mechanics problem of the arterial wall, resulting in the specific force that the arterial pulsatile flow exerts upon the blood vessel walls. The numerical results outline the high-risk regions of the aneurysm, prone to rupture or dissect the flow patterns there, and the arterial wall deformation due to the blood flow.

## 2. A MORE REALISTIC COMPUTATIONAL DOMAIN

In this study we use high resolution CT DICOM image sets to build a 3D solid model of the aneurysm affected subclavian arterial network. Numerical methods have reached the level where their usage results in realistic, patient related 3D models that provide for more insightful results as compared to models based on simplified computational domains.

The 3D solid model of the arteries (Fig. 2b) is comprised of two subdomains – the blood region and the vessel walls – both obtained using masks generated by applying an optimized threshold filter [8], which singles out the regions corresponding to certain grey scale values in the image. This way, coarse masks for the blood and the surrounding vessels are segmented out from the DICOM set.

Spurious artifacts are then removed by the bilateral and the anisotropic diffusion noise, gradient filters [8].

The masks surfaces are smoothed out by using Gaussian and binarisation filters, shortly after their space continuity and morphological consistency is assured by applying erode, cavity fill and flood fill filters [8].

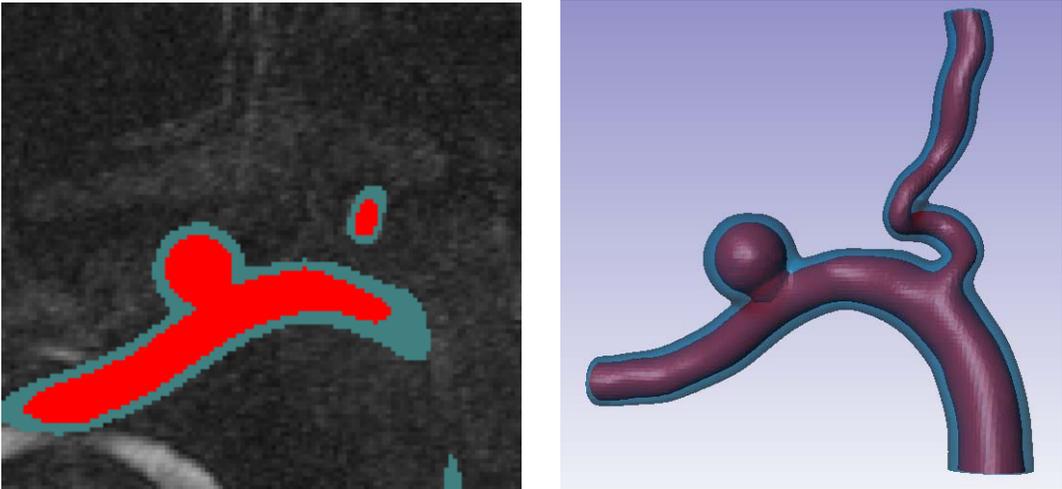


Fig. 2 – A more realistic computational model: (left) one of the images in the CT source dataset; (right) the 3D solid model of the aneurysm obtained by imagistic techniques

The final 3D model of the aneurysm is FEM discretized and used as computational domain for the hemodynamic flow-structural interaction problem. Accuracy test provide the number of finite elements that make the numerical results grid-independent (Fig. 3).

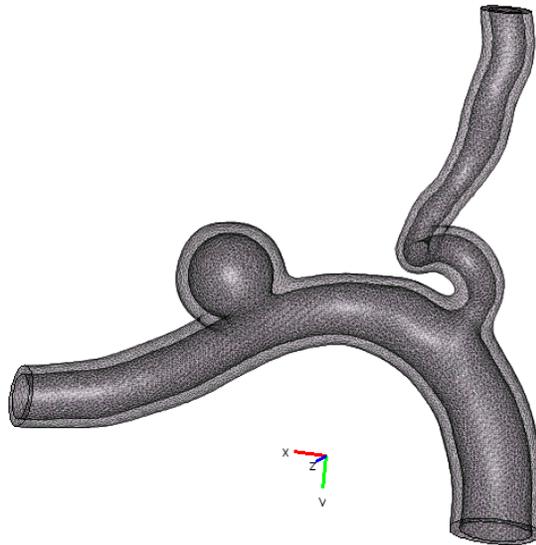


Fig. 3 – The computational domain FEM grid made of *approx.* 150,000 tetrahedral, Lagrange elements

### 3. THE MATHEMATICAL MODEL

#### 3.1. The haemodynamic model

In this study the blood is a Newtonian fluid with constant properties, and no mass transfer occurs between the blood and the arterial walls [9-10]. The arterial flow is assumed pulsatile, laminar, and incompressible arterial flow, described by a mathematical model made of momentum balance and the mass conservation laws:

momentum balance (Navier-Stokes)

$$\rho \left[ \frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{u} \right] = \nabla \cdot \left[ -p \mathbf{I} + \eta (\nabla \mathbf{u} + (\nabla \mathbf{u})^T) \right] \quad (1)$$

mass conservation law

$$\nabla \cdot \mathbf{u} = 0 \quad (2)$$

where  $\mathbf{u}$  [m/s] is the velocity field,  $p$  [Pa] is the pressure,  $\rho$  [kg/m<sup>3</sup>] is the mass density (1000 [kg/m<sup>3</sup>]),  $\eta$  [Pa·s] is the dynamic viscosity (0.005 [Pa·s]), and  $\mathbf{I}$  is the unity matrix.

The hemodynamic model is closed by the following boundary conditions: no slip conditions for the arterial walls, and pressure for the inlet and the outlets of the arterial network (Fig. 4).

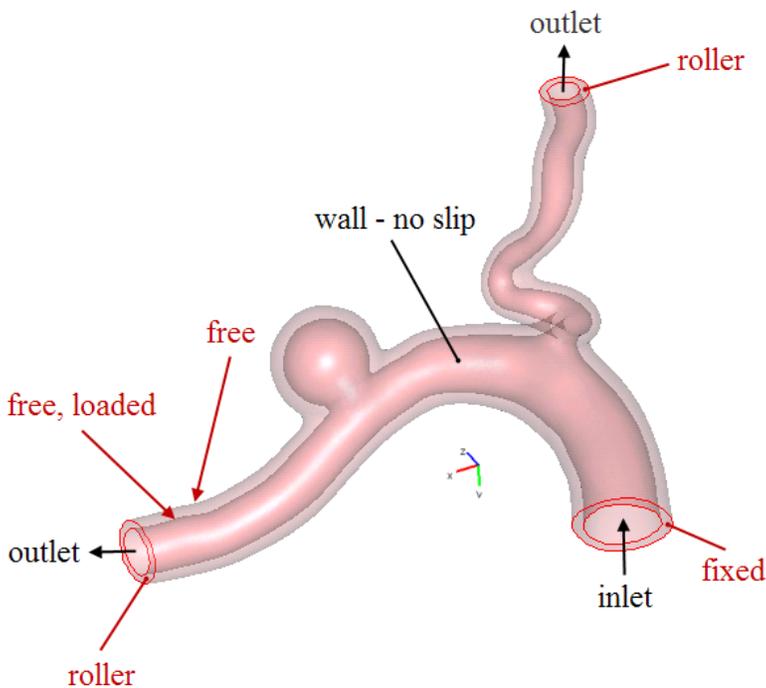


Fig. 4 – Boundary conditions for the hemodynamic problem (black) and for the structural mechanics problem (red)

A steady state flow problem was solved first, by setting a 0.4 [m/s] velocity condition at the inlet and zero pressure conditions at the arteries outlets. The pressure drop between the inlet and the outlets of the vessels was then determined (*approx.* 1400 [Pa]), and used as the boundary conditions.

Pulsatile blood flow was simulated by setting time-dependent pressure boundary conditions at the inlet and the outlets of the subclavian arterial network:  $p_{in}(t) = 11,400 \cdot p_i(t)$  [Pa],  $p_{out}(t) = 10,000 \cdot p_i(t)$  [Pa];  $p_i(t) = 1 + K \sin(t + 3/2)$ , where  $K = 0.1$  tunes the pulsation amplitude level. Judging by its size, the subclavian artery is part of the resistance type blood vessels [12].

### 3.2. The structural mechanics model

The hemodynamic model is then coupled to the structural model to study the arterial walls deformation by the pulsatile flow. The arterial walls are almost incompressible and able to undergo large strains, and their constitutive behavior is highly nonlinear [13]. Therefore we use a hyperelastic constitutive law, defined by using a strain energy density function,  $\bar{W}$ .

The stresses,  $S$ , are computed by deriving the density function,  $\bar{W}$ , with respect to the Green strains,  $E$ , such that  $S = \partial\bar{W}/\partial E$ . An isotropic neo-Hookean model [13] describes the strain energy density

$$\bar{W} = \frac{1}{2} J^{-\frac{2}{3}} \left( I - \frac{1}{3} \bar{I}_1 C^{-1} \right) + \frac{1}{2} \kappa J (J - 1) C^{-1} \quad (3)$$

where  $J = \det(\mathbf{F})$  is the ratio between the current and the original volume,  $\mathbf{F}$  is the deformation gradient,  $C = \mathbf{F}^T \mathbf{F}$  is the right Cauchy-Green tensor,  $I_1 = \text{trace}(C)$ ,  $\bar{I}_1 = I_1 J^{-2/3}$ . Table 1 lists the properties utilized in the numerical simulations.

Table 1 – Material properties.

Material	Property	Value
Blood	$\rho_B$	1060 kg/m <sup>3</sup>
	$\mu_B$	5 m·N·s/m <sup>2</sup>
Artery wall (isotropic neo-Hookean, hyperelastic)	$\rho_A$	960 kg/m <sup>3</sup>
	$\mu$	6204106 N/m <sup>2</sup>
	$\nu$ (Poisson ratio)	0.45

## 4. NUMERICAL SIMULATION RESULTS AND CONCLUSION

The mathematical model (1)-(3) was FEM implemented and solved for using Comsol Multiphysics [11] following a solution strategy, which implied solving for the pulsatile blood flow first. Then, using the calculated pressure values, the structural problem is solved for. The coupling between the hemodynamic and structural models is thus one-way (flow → arterial walls), and provides for physical solutions, and a more convenient computational load.

The structural model is solved for steady states, using a parametric solver that uses the pressure field values at different moments in time, generated after solving for the flow problem by storing the numerical simulation results for several periods of the flow. The solvers are based on the BICGstab algorithm with geometric multigrid preconditioning [11].

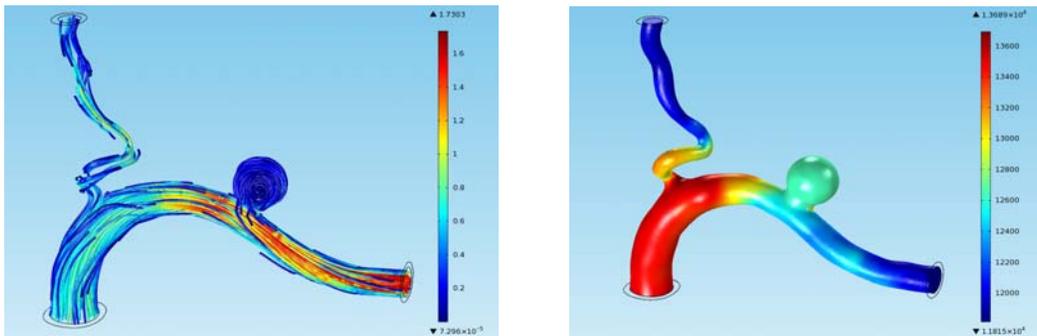


Fig. 5 – Flow field by streamlines (left), and the total force per area upon the interior artery walls (right)

Fig. 5 shows the flow pattern inside the aneurysm. The total force per area is calculated and then used as boundary conditions for the structural model. The  $O_y$  component total force per area, approximately in the mainstream direction at the inlet (Fig. 6, left), is the one that exerts higher stress upon the aneurysm wall, increasing thus either the rupture or the dissection probability.

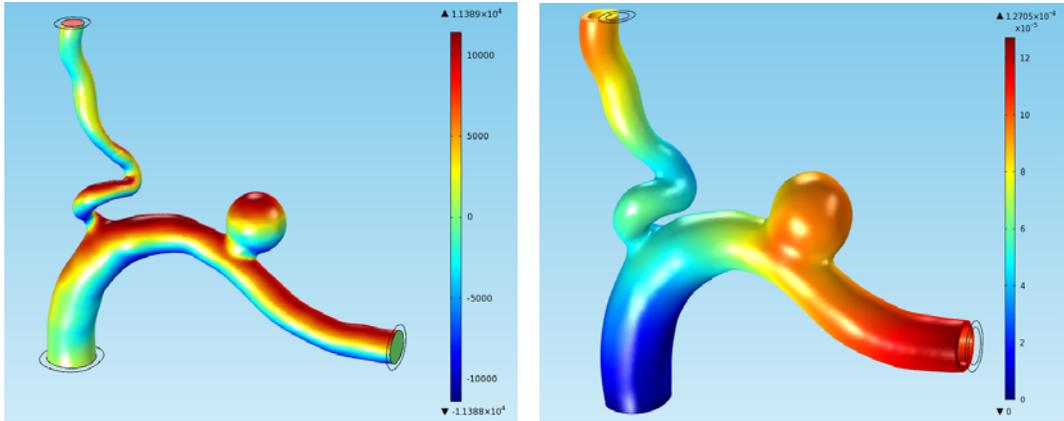


Fig. 6 – The total force per area in  $O_y$  (left), and the artery walls deformation (right)

By constraining the subclavian artery inlet and setting as roller the outlets of the main and secondary blood vessels (Fig. 4), the artery walls are deformed due to the pressure exerted by the pulsatile blood flow (Fig. 6, right).

## CONCLUSIONS

This paper presents a mathematical model and numerical simulation results for the study of the pulsatile blood flow – arterial walls structural interaction in an aneurysm by the subclavian arterial network. Realistic, patient specific computational domains are generated through image based reconstruction techniques out of high resolution CT DICOM datasets.

The mathematical model accounts for the nonlinear behavior of the blood vessels and the one-way coupling between the hemodynamic and structural models. Besides the realistic computational domain – out of medical CT images – pulsating flow conditions and deformable vessel walls are the main features of the model presented in this paper.

Numerical simulations were conducted long enough to reach quasi-steady, periodic flow regime and deformation (displacement) of the artery wall. The main finding is that, as observed in surgical practice, the upper end of the aneurysm is the region of highest stress, hence risk of rupture. In the second place comes the “collar” that connects the aneurysm to the artery – a region prone to dissection.

As of this study, the displacement (deformation) of the walls is minute, negligible small. Numerical simulations were conducted using Galerkin FEM technique.

## ACKNOWLEDGMENTS

The work was conducted in the Laboratory for Electrical Engineering in Medicine (IEM) – Multiphysics Models, the BIOINGTEH platform, at UPB.

A.A. Dobre acknowledges the support offered by the POSDRU/88/1.5/S/61178 grant. A.M. Morega acknowledges the CNCSIS PCCE-55/2008 grant.

## REFERENCES

- [1] E.M. Isselbacher, *Diseases of the aorta*, in: Goldman L., Ausiello D., eds., Cecil Medicine, 23rd ed. Philadelphia, Pa: Saunders Elsevier, 2007.
- [2] J.A. Zivin, *Hemorrhagic cerebrovascular disease*. In: Goldman L., Ausiello D., eds., Cecil Medicine. 23rd ed. Philadelphia, Pa: Saunders Elsevier, 2007.
- [3] S.C.Hauser, *Vascular diseases of the gastrointestinal tract*. In: Goldman L., Ausiello D., eds., Cecil Medicine. 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007.
- [4] <http://www.reshealth.org/yourhealth/healthinfo/default.cfm?pageID=P08264>.
- [5] <http://www.nhs.uk/conditions/Aneurysm/Pages/Introduction.aspx>.
- [6] A.M. Morega, A.A. Dobre, *Numerical Modeling of the Subclavian Aneurysm Blood Flow*, International Conference on E-Health and Bioengineering - EHB 2011, 24-26 November, Iasi, Romania.
- [7] <http://pubimage.hcuge.ch:8080>.
- [8] Simpleware v3.2, Simpleware Ltd., UK, 2010.
- [9] A.M. Morega, A.A. Dobre, M. Morega, D. Mocanu, *Computational modeling of arterial blood flow*, Proc. 2<sup>nd</sup> International Conference on Advancements of Medicine and Health Care Through Technology – MediTech 2009, 23-26 September, Cluj-Napoca, Romania.
- [10] A.M. Morega, A.A. Dobre, M. Morega, *Numerical simulation of magnetic drug targeting with flow – structural interaction in an arterial branching region of interest*, Comsol Conference, 17-19 November 2010, Versailles, France.
- [11] Comsol Multiphysics v3.5a, v4.2, Comsol AB, Sweden, 2010.
- [12] R.A. Feijoo, "Computational methods in biology", 2nd Summer School LNCC/MCT, Petropolis, 2000.
- [13] M.Y.H. Bangash, F.N. Bangash, T. Bangash, "Trauma. An engineering analysis with medical case studies investigation", Springer, 2007.