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# COUPLED ANALYSIS OF A CAROTID ARTERY WITH AN ATHEROSCLEROTIC PLAQUE AT VARIOUS STAGES OF DISEASE

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**Abstract:** *Cardiovascular diseases are responsible for the largest number of human death relative to other causes. In this paper, the results of coupled analysis related to the carotid artery with atherosclerotic plaque, performed via commercial software – ANSYS are shown. The mathematical model consists of the Navier – Stokes equation supplemented by boundary and initial conditions. To describe the non-linear characteristic of the artery and calcified plaque, the Yeoh 3rd Order and Mooney – Rivlin 5th Order Models are used. The velocity, stress, and deformation distribution at the analyzed artery are shown.*

**Keywords: Finite element method, Blood flow, Fluid Structure Interaction, Modeling of biological tissue, Coupled Analysis.**

## 1. Introduction

The circulatory system in the human organism takes an important role in the transport of gas, nutrients, hormones and waste products. It consists of the heart and blood vessels through which the blood is transported. With intensive civilization development, there is a high risk of increased incidence of cardiovascular disease. One of the most common diseases of this type is atherosclerosis. Atherosclerosis is an inflammatory disease characterized by plaques builds up in the vessel wall. In this paper, the coupled analysis of blood flow in the artery with atherosclerosis plaque and fluid interaction with the vessel wall is performed. The Yeoh material model of artery is taken into account. To solve the problem, the commercial software ANSYS based on the finite element method is used. The obtained results are compared with those found in the literature and the conclusions are formulated.

## 2. Model description and numerical procedure

The created artery model is based on the dimensions of the Common Carotid Artery (CCA). Two layers of artery, namely media and adventitia are taken into account. Figure 1 shows the dimensions of each layer and plaque corresponding to the first stage of illness. To simulate the process of plaque growth, it was decided to perform three analyses, with gradually increasing the plaque height (H): first – H = 0.259 mm, second – H = 0.6475 mm, third – H = 1.9425 mm (Mordal and Szarek, 2017).

### 2.1. Formulation of the problem

The blood flow in the artery is described by the following system of equations

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial r}-\frac{v}{r}\frac{\partial u}{\partial \theta}-\frac{\partial w}{\partial z}=-\frac{1}{\rho}\frac{\partial p}{\partial x}+\nu\left(\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial u}{\partial r}\right)-\frac{u}{r^2}+\frac{1}{r^2}\frac{\partial^2 u}{\partial \theta^2}-\frac{2}{r^2}\frac{\partial v}{\partial \theta}+\frac{\partial^2 u}{\partial z^2}\right) \quad (1)$$

$$\frac{\partial v}{\partial t}+u\frac{\partial v}{\partial r}+\frac{v}{r}\frac{\partial v}{\partial \theta}+\frac{uv}{r}+w\frac{\partial v}{\partial z}=-\frac{1}{r\rho}\frac{\partial p}{\partial \theta}+\nu\left(\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial v}{\partial r}\right)-\frac{v}{r}+\frac{1}{r^2}\frac{\partial^2 v}{\partial \theta^2}+\frac{2}{r^2}\frac{\partial u}{\partial \theta}+\frac{\partial^2 v}{\partial z^2}\right) \quad (2)$$

$$\frac{\partial w}{\partial t}+u\frac{\partial w}{\partial r}+\frac{v}{r}\frac{\partial w}{\partial \theta}+w\frac{\partial w}{\partial z}=-\frac{1}{\rho}\frac{\partial p}{\partial z}+\nu\left(\frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial w}{\partial r}\right)+\frac{1}{r^2}\frac{\partial^2 w}{\partial \theta^2}+\frac{\partial^2 w}{\partial z^2}\right) \quad (3)$$

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$$\frac{1}{r}\frac{\partial ru}{\partial r} + \frac{1}{r}\frac{\partial v}{\partial \theta} + \frac{\partial w}{\partial z} = 0 \quad (4)$$

*u*, *v*, and *w* are the velocity components in the *r*, $\theta$ and *z* – direction, *p* is the pressure, $\rho$ is the density, *v* is the kinematic viscosity and *t* denotes the time. Blood is a non-Newtonian fluid, but for blood vessels with a diameter larger than 100 μm, it can be treated as a Newtonian fluid of density ρ=1060 kg/m$^3$ and kinematic viscosity $v$ = 0.0028 m$^2$/s (Bessonov et al., 2016). The system of Eqs. (1) – (4) should be supplemented by appropriate boundary and initial conditions. Inlet velocity $v_0$ is equal to 0.359 m/s and outlet pressure $p_{out}$ is calculated from the mean value of diastolic and systolic pressure – 120 mm Hg and 80 mm Hg (Holdsworth et al., 1999). The final $p_{out}$ is equal to 100 mm Hg. The initial conditions are known: $u = v = w = 0$ m/s. In the stress analysis of CCA the pressure calculated from Eqs. (1) – (3) is used as the boundary condition applied at vessel wall and plaque.

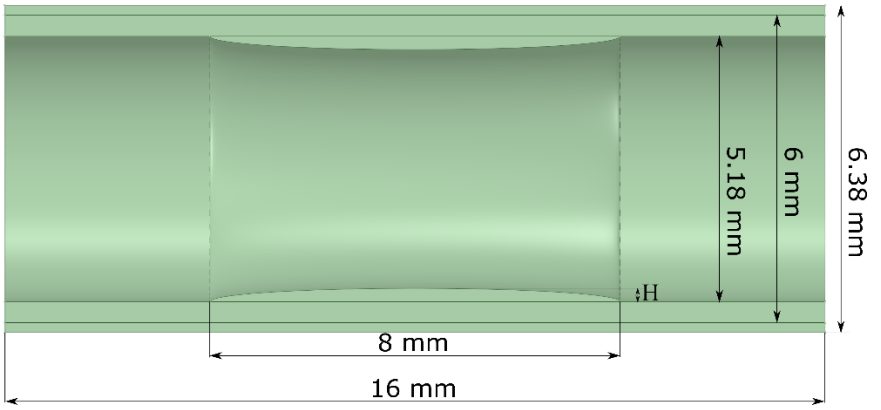


*Fig. 1: Model of CCA with plaque.*

### 2.2. Material models

The non-linear material characteristics of two-layered artery are described by the Yeoh 3rd Order Strain Energy Function (Cieslicki et al., 2018):

$$\Psi = c_{10}(\bar{I}_1 - 3) + c_{20}(\bar{I}_1 - 3)^2 + c_{30}(\bar{I}_1 - 3)^3 \quad (5)$$

where $\Psi$ is a strain energy potential, $\bar{I}_1$ is the first deviatoric strain invariant, the constants $c_{10}$, $c_{20}$, $c_{30}$ [MPa] are collected in Tab. 1.

To describe the material of calcified atherosclerosis on each stage of disease, the Mooney – Rivlin 5th Order model is applied (Karami, 2014):

$$\Psi = c_{10}(\bar{I}_1 - 3) + c_{01}(\bar{I}_2 - 3) + c_{20}(\bar{I}_1 - 3)^2 + c_{11}(\bar{I}_1 - 3)(\bar{I}_2 - 3) + c_{02}(\bar{I}_2 - 3)^2 \quad (6)$$

where $\bar{I}_2$ is the second deviatoric strain invariant. The constants $c_{10}$, $c_{01}$, $c_{20}$, $c_{11}$, $c_{02}$ [MPa] are collected in Tab. 1.

*Tab. 1: Material properties.*

| | $c_{01}$ | $c_{02}$ | $c_{10}$ | $c_{11}$ | $c_{20}$ | $c_{30}$ |
|---|---|---|---|---|---|---|
| **Media** | — | — | 0.11788 | — | –0.28472 | 10.301 |
| **Adventitia** | — | — | 0.17156 | — | 1.4746 | 311.55 |
| **Calcified plaque** | 0.506 | 4.737 | –0.495 | 1.193 | 3.637 | — |

## 3. Results of computations

A generated mesh consists of 1127 finite elements (Hex8 and Wed6). In Fig. 2 the results after 10 seconds for CCA in the third state of disease are shown. The occurrence of atherosclerosis contributes to generate vortices around the plaque. Their presence is confirmed by a change in the orientation of the velocity vector components. In Table 2 the results for each stage of the analysis are summarized.

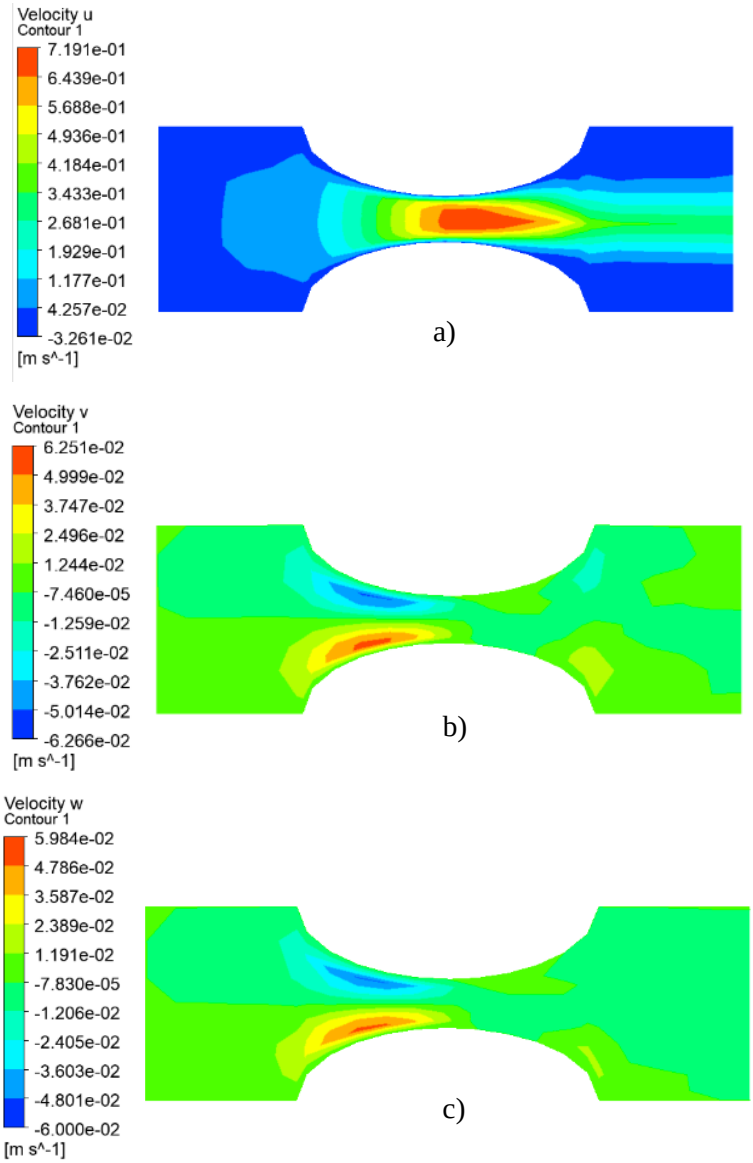


*Fig. 2: Blood velocity vector: a) w component; b) v component; c) u component in CCA with the atherosclerotic plaque in third state of disease.*

*Tab. 2: Blood velocity vector components on each statement of disease.*

| | **Maximum/Minimum *w* component [m/s]** | **Maximum/Minimum *v* component [m/s]** | **Maximum/Minimum *u* component [m/s]** |
|---|---|---|---|
| Health artery | 0.0645/0.0000 | 0.0031/-0.0026 | 0.0030/-0.0026 |
| I stage | 0.0694/0.0000 | 0.0033/-0.0034 | 0.0036/-0.0035 |
| II stage | 0.0946/0.0000 | 0.01084/-0.0101 | 0.0105/-0.0097 |
| III stage | 0.7191/-0.3261 | 0.0625/-0.0627 | 0.0598/-0.6000 |

Due to the different compressive and tensile strength of blood vessels, it is decided to present the highest principal stresses, not the von Mises hypothesis (Tab. 3). Fig. 3 shows maximum deformation of CCA at the second stage of disease.

*Tab. 3: Results of analysis.*

| | Maximum principal stress [Pa] | Maximum shear stress [Pa] | Maximum Deformation [mm] |
|---|---|---|---|
| Health artery | 4490 | 4258 | 0.0073 |
| I stage | 20614 | 10536 | 0.0224 |
| II stage | 5092 | 4311 | 0.0136 |
| III stage | 51854 | 18391 | 0.0371 |

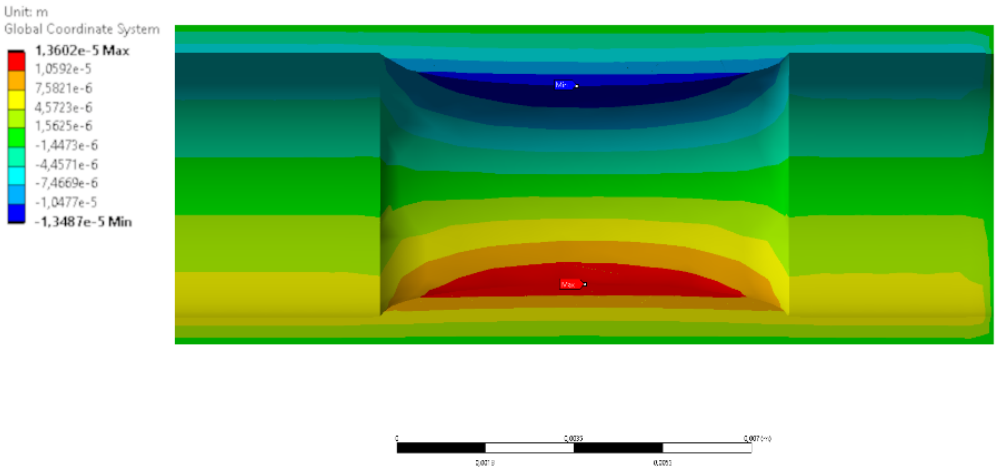


*Fig. 3: Maximum deformation of CCA with the atherosclerotic plaque in the radial direction.*

It should be noted that the solutions presented here are consistent with the results presented in (Karami et al., 2014), although slightly different material models have been used.

## 4. Conclusions

The modeling of blood flow in the artery with atherosclerotic plaque has been presented. The non – linear characteristic of artery behavior during blood flow has been applied. It has been shown that with the growth of atherosclerosis, vortices behind the plaque increase.

In the future, the real geometry of the carotid artery, its multilayered structure and more accurate models describing the development of atherosclerosis should be taken into account.


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