Blood flow through the circulatory system element affected by double stenotic lesions.

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Abstract. Destruction of the elements of the blood circulatory system may in many cases cause many dangerous diseases. Despite many medical diagnostic tools being used in medical laboratories, mathematical modeling at present can be successfully used for predicting, setting the diagnosis, forming coherent picture of the disease, and determining further treatment. One of the most dangerous pathologies of cardio-vascular system is stenosis. This disease is a consequence, and cause of many dangerous conditions. Due to small characteristic size of the element of the circulatory system (1-3 mm) and moderate values of Reynolds number (0.1-300), non-Newtonian properties of blood appear at the flow in the affected cells of the circulatory system. The rheological constitutive relation FENE - P is used for the flow modeling which predicts rheological properties of the blood such as anomalous viscosity and effects of elasticity. This paper examined the effect of the rigidity of the walls of blood vessels and investigated the effect of double - stenotic lesions on blood flow. An element of the circulatory system with a double - stenotic lesions with 50 % narrowing was chosen as the model case. As a result of the simulation, streamlines were obtained, which are the distribution of the principal stresses difference (PSD) in the most significant places of the affected elements of the circulatory system.

1. Introduction
Atherosclerotic lesions of vessels are some of the most common vascular diseases and are categorized as chronic dangerous pathologies. Atherosclerosis is a condition associated with formation of narrowing plaques on the vessel’s walls. As a result, blood circulation, composition and properties of blood flow as well as the nutrition of organs and tissues do not run as usual. There are many reasons for this pathology: lack of physical activity, injury, quality of food, age, family history and the presence of other diseases [1-4].

At present, medical science developed numerous ways to perform diagnosis of stenosis such as ultrasound of the heart, magnetic - resonance angiography and computed tomography to name a few. Clinicians performing the diagnostics sometimes utilize several methods at the same time in order to
have a complete picture of the disease. Mathematical modeling is another promising method utilized in diagnostic and therapeutic surgery [5-6, 14]. Building different model cases allows to apply an individual approach to each particular case, forecast and minimize possible risks and choose the most effective treatment.

Creation of a mathematical model is difficult due to the complex internal structure of blood, nature of the blood flow, the structure of the vessel walls, and localization of the affected area of the circulatory system. In addition, limited amount of research has been done on mechanical properties of the formed elements in the blood flow. This is one of the reasons for the lack of consensus among researchers on necessity of considering non-Newtonian properties of the blood. Some scientists believe that the non-Newtonian properties should be considered only for calculations of small vessels (less than 0.5 mm [7]) when the value speed and tangential stresses are small so for relatively large elements of the circulatory system simulations carried out with Newtonian or power-law fluid models [8].

Since the end of last century scientists have undertaken attempts to simulate blood flow as a viscoelastic fluid using various models for the components of the circulatory system with a diameter of more than 1 mm [9]. Research [10] presents the results of an experiment, aimed at comparing the flow of Newtonian and viscoelastic liquids during the course of the flow in the branching element of a circulatory system. Glycerol solution was used as a Newtonian fluid while the diluted solution of polyacrylamide was used as a non-Newtonian. Three-dimensional channel of 1 mm diameter was used as a branching element of the circulatory system. The experiment was carried out for values of Reynolds number 498 to 951 and showed, that the stream lines for two fluids are different. A stagnant area formed in the channel in the case of a non-Newtonian fluid. This effect didn’t observe in the case of Newtonian fluid. Studies [11-13] discuss modeling of the flow of blood in various kinds of elements of the circulatory system with pathologies (stenosis, aneurysms, and etc.) using Newtonian and non-Newtonian fluid models. A comparison of the results shows, that the representation of the blood as a non-Newtonian fluid allows to catch those effects, which arise in the flow of blood, but not reproduced by Newtonian fluid.

One of the most important characteristics of the blood flow in the affected elements of the circulatory systems is normal and shear stresses. Normal stresses arise where the blood flow meets a restriction or expansion of vessels, separation, as well as rotation of the flow. Relatively high values of normal stress may lead to the damage of the formed elements of the blood, leading to deformation of the vessels walls and formation of plaques. High normal stresses are the consequence of pressure increasing in the blood, it is provoking factor for heart attacks.

There is a number of rheological constitutive relations predicting the behavior of such complex liquids as blood. One of these relations is rheological constitutive relation FENE – P [6]. The advantage of this model is its ability to predict characteristics of fluids such as those specific to blood. It can be noted, that this constitutive relation is used to simulate the flows of dilute polymer solutions too.

The purpose of this work is to achieve mathematical simulation of the flow of blood (a composition which contains protein macromolecules) in a stenosis affected elements of the circulatory system and discuss the influence of these macromolecules configurations on the flow picture in the vicinity of the affected element of the blood circulatory system.

2. Mathematical model
The schematic representation of the element of the circulatory system is shown in Fig.1. The model of an element of the circulatory system is a 2D channel with symmetrical double stenosis and the characteristic values are given in the table below.

Table 1. Symbols and accepted values

| Title                     | Symbol | Value (mm) |
|---------------------------|--------|------------|
| The width of the channel  | H      | 3          |
| The length of stenosis    | d      | 6          |
The width of the gap $h = 0.5H$

**Figure 1** The shape of the affected areas of the blood circulatory system

It is assumed that the blood is an incompressible viscoelastic fluid with a given average density ($\rho = 1000$ kg/m$^3$), dynamic viscosity at zero shear rate ($\eta_0 = 0.005$ Pa·sec), and a stress relaxation time $\lambda = 0.0189$ sec.

In this paper, the FENE-P model (Finitely Extensible Nonlinear Elastic by Peterlin) is selected. It predicts the following properties: viscosity anomaly, the presence of stress relaxation and the dependence of the longitudinal viscosity on the longitudinal strain rate. These properties are also shown when the blood flows through the affected element of the circulatory system and underlie the occurrence of negative effects. Based on this, blood is considered to be a non-Newtonian fluid. This viscoelastic model is based on the representation of macromolecules in the form of flexible dumbbells. It is assumed that in the stream, such dumbbells can stretch relatively more to their original length and change orientation. While conducting an analogy with blood components, protein macromolecules were considered as flexible dumbbells.

The movement of blood through the circulatory system is described by the equations of motion and continuity [6, 14]:

$$\rho \left( \frac{\partial \vec{v}}{\partial t} + \vec{v} \cdot \nabla \vec{v} \right) = -\nabla p + \vec{v} \cdot \vec{T},$$

$$\nabla \cdot \vec{v} = 0.$$  

(1)  

(2)

The total stress can be written as a sum as follows:

$$\vec{T} = \vec{T}^p + \vec{T}^s.$$  

(3)

Rheological constitutive relation can be written as follows:

$$\vec{T}^p = \frac{\eta^p}{\lambda} \left[ \frac{\tilde{\Lambda}}{1 - \frac{\text{tr}(\tilde{\Lambda})}{3L^2} \frac{\tilde{T}}{L^2}} - \frac{\tilde{T}}{1 - \frac{\text{tr}(\tilde{\Lambda})}{3L^2} \frac{\tilde{T}}{L^2}} \right],$$

$$\frac{\tilde{\Lambda}}{1 - \frac{\text{tr}(\tilde{\Lambda})}{3L^2} + \text{We} \tilde{\Lambda}} = \frac{\tilde{T}}{1 - \frac{\text{tr}(\tilde{\Lambda})}{3L^2} \frac{\tilde{T}}{L^2}}.$$  

(4)  

(5)

$$\tilde{\Lambda} = \frac{\partial \tilde{\Lambda}}{\partial t} + \vec{v} \cdot \nabla \tilde{\Lambda} - \nabla \tilde{\Lambda} \cdot \vec{v} - \tilde{\Lambda} \cdot (\nabla \vec{v})^T.$$  

(6)
where $\vec{v}$ is the velocity vector; $\rho$ - the density of the blood; $\dot{\lambda}$ - the characteristic stress relaxation time; $\eta^\rho = \eta^s + \eta^r$ - the blood viscosity at zero shear rate; $\eta^s$ - the dynamic viscosity of non-Newtonian liquid component at zero shear rate; $\eta^r$ - the dynamic viscosity of the blood plasma; $\vec{r}^\rho$ - the non-Newtonian stress component; $\vec{r}^s$ - the Newton stress component; $\langle \vec{Q} \vec{Q} \rangle = \int \int \int \vec{Q} \vec{Q} \cdot \mathbf{P}_N(\vec{Q}) \cdot d\vec{Q} d\vec{Q} d\vec{Q}$ - the averaging over the ensemble, where $\mathbf{P}_N(\vec{Q})$, the probability that a randomly chosen protein macromolecule has a predetermined size, which is in the range of $\vec{Q}_0 \vec{Q} + d\vec{Q}$; $\vec{B} = \frac{1}{2} (\vec{v} \vec{v} + (\vec{v} \vec{v})^T)$ - is the deformation rate tensor, where $(\vec{v} \vec{v})^T$ is the transposition procedure.

Standard procedure for transforming equations into dimensionless form provide the following dimensionless variables:

$$W_e = \frac{\lambda U}{l}, \quad Re = \frac{\rho U l}{\eta^s}, \quad \beta = \frac{\eta'}{\eta^s}, \quad L^2 = \frac{Q_{\infty}}{Q_0},$$

where $U$ - characteristic velocity; $l$ - characteristic linear scale; $Q_{\infty}$ - the length of the configuration vector of the protein macromolecule in equilibrium; $Q_0$ - the maximum possible length of the configuration vector; $W_e$ - We-Weissenberg number; $Re$ - Reynolds number; $L^2$ - is the parameter characterizing the degree of unraveling of the coil of the protein macromolecule.

2.1. Boundary conditions

At the channel entrance (boundary G1, fig.1) the constancy of the horizontal velocity component and the equality to zero of the vertical velocity components are set as follows:

$$U_x = \text{const}, \quad U_y = 0. \quad (7)$$

To form a steady-state velocity profile, and prevent the influence of a given velocity profile in the input section on the flow in the central part of the channel, the length of the input part of the channel is selected as 10 widths. With this choice, as shown by test calculations, the fluid flow in the central part of the channel does not depend on the specification of the velocity profile in the inlet section.

At the output of the channel (boundary G2, fig.1) "soft" conditions are set:

$$\frac{\partial U_x}{\partial x} = 0, \quad U_x = 0, \quad \frac{\partial \tau_{xx}}{\partial x} = \frac{\partial \tau_{xy}}{\partial x} = \frac{\partial \tau_{yx}}{\partial x} = 0. \quad (8)$$

Boundary conditions (8) mean that the flow at the output is assumed to be steady with uniform velocity and stress profiles. According to test calculations, the selected length of the output part as 20 width is also suitable to fulfill condition (8).

On the solid walls of the channel (boundary G3, fig.1), no-slip condition are set, according to which the horizontal and vertical velocity components are zero:

$$U_x = 0, \quad U_y = 0. \quad (9)$$

2.2. Initial conditions at the initial time in the entire flow field

The fluid flow at the initial time is so, that
\[ U_x = 0, \quad U_y = 0. \]  

(10)

The problem was solved numerically, using the control volume method based on the integrated OPENFOAM 4.0 platform.

The decision was made for a semi-explicit time scheme. The stability was ensured by satisfying the Courant criterion [14].

3. Results and discussion
In numerical simulations, blood behavior is described using two fluid models: Newtonian and viscoelastic. By implementing a model of a viscoelastic fluid, a blood flow with pathologies of various "complexity" is represented. The degree of fluid “complexity” in this paper is determined by the value of parameter \( L^2 \), which characterizes the degree of unraveling of the protein macromolecules. For comparison, two limiting cases are considered, where \( L^2 = 50 \) and \( L^2 = 700 \), and the rest of the model parameters (\( \text{We} = 1, \text{Re} = 100, \beta = 0.1 \)) are assumed as the same in value. In the model case, the walls of the circulatory system element are rigid, which is typical for vessels affected by stenosis. Such assumption takes place due to the following possible factors: with age, the walls lose their elasticity and permeability, which can be both a consequence and one of the causes of metabolic disturbances.

Obtaining results for Newtonian fluids is possible when setting the Weissenberg number value, \( \text{We} = 0.01 \).

Fig. 2 shows the streamlines for non-Newtonian fluid at various values of the parameter \( L^2 \) and Newtonian fluid in the affected element of the circulatory system.

\[ \text{Figure 2. Streamlines. } L^2 = 50 \text{ (a); } L^2 = 700 \text{ (b) and Newtonian fluid (c)} \]
The formation of stagnant areas is primarily associated with the form of the circulatory system element under consideration and the presence of special fluid properties. Near the walls there is a rapid change in speed of the flow velocity, while the change in speed in the central part of the artery is insignificant. In places where there is a change in the shape of the walls (in this case, the area where the stenosis and artery wall connect), the flow structure changes and stagnant areas form. The size of the stagnant areas depends on the shape, degree of narrowing and the length of stenosis.

Figure 3 shows the superposition of streamlines on the isolines of the principal stress difference. Evidently, the influence of $L^2$ (Fig. 3 (b)) leads to the formation of a “tube” of oriented macromolecules from the central part of the stenosis along the stream in the stenotic element of the circulatory system. This “tube” of oriented macromolecules blocks the stagnant regions. Such "tube" prevents the penetration of fluid from the central part of the channel into a stagnant region. As a result, the geometric dimensions of stagnant areas are reduced.

In the discussed case, the influence of the “tube” on the size of the stagnant region is not significant, however, a comparison of these graphs (Fig. 3) demonstrates that for a Newtonian fluid, the emerging stress state is associated with a change in the shape of the channel.

![Figure 3](image)

**Figure 3.** The distribution of principal stress difference in the artery to $L^2 = 50$ (a), $L^2 = 700$ (b) and a Newtonian liquid (c)

Fig. 3 (b) it is clearly distinguishable that the stagnant regions have different sizes. In this case, asymmetry is detected in the distribution of the isolines of the principal stress difference. This asymmetry is a consequence of differences in the orientation of protein macromolecules.

4. Conclusion

Using numerical modeling, models of blood flow in an artery with double stenotic plaques were created. The possibility of obtaining the flow behavior and predicting the occurrence of stagnant areas and other possible negative effects allows to complement the clinical picture of the disease and predict the consequences. Despite the apparent similarity of streamlines, it should be noted that two limiting model cases (Newtonian fluid and viscoelastic fluid at $L^2 = 700$) are significantly different from each other. The presence of protein macromolecules in the elements of the circulatory system affects the
flow pattern in such a way that the symmetric form of the flow becomes asymmetric and areas of increased stress values appear in the flow.

A change in the parameter $L^2$ (increase to 700) significantly affects the nature of the flow too. Despite the decrease in stagnant areas, a “tube” of oriented macromolecules forms inside the circulatory system element. Thus, a 50% narrowing of the flow is retained in the channel outside the stenosis. The presence of asymmetry is explained by the combined influence of normal stress and altered conformation of protein macromolecules.

Thus, it may be concluded that the presence of a second stenotic lesion has a noticeable effect on the flow structure, and significantly increases the load on the human cardiovascular system.

5. References

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