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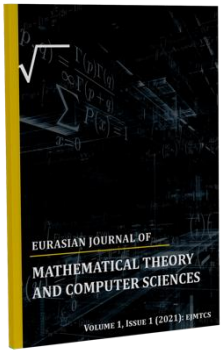

DEVELOPMENT AND COUNTING PRESSURE ONE DIMENSIONAL MODEL OF FDM FOR BLOOD CAPILLARY VESSEL

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ABSTRACT

One-dimensional hemodynamic models have proven themselves well in the study of global blood flow in the human body in normal and pathological conditions. One of the key issues of successful modelling using one-dimensional models is taking into account the elastic properties of the walls of blood vessels. This work is devoted to the comparative analysis of various mathematical descriptions of the elastic properties of vascular walls in modern models of one-dimensional hemodynamics. Approaches to modelling blood flow using one-dimensional network models. They have developed successfully in recent decades. This class of models has proven itself well in the study of global blood flow in the human body, solving problems of the transport of substances by blood, features of hemodynamics in pathological changes in the body. The physiological aspects related to neuroregulation of vascular tone are considered. Models of the transport of substances and blood flow under gravitational overloads have been constructed, and several variants of the equations of state describing the elasticity of vascular walls have been studied. The modelling of the walls of the elasticity of blood vessels is one of the key issues of successful hemodynamic modelling using one-dimensional models. To date, there are several mathematical formulations. Despite the differences, they are used successfully not only for qualitative, but also for quantitative individualised hemodynamic calculations. This work is devoted to the comparative analysis of various mathematical descriptions of the elastic properties of vascular walls in

modern one-dimensional models of hemodynamics, and the pressure is considered using daily monitoring.

1 INTRODUCTION

Large and small circulatory circles consist of sequentially connected sections, each of which plays a certain role in the realisation of the main function of the vascular system and, accordingly, the features of the structure and functional organisation. These are mainly large elastic-type arterial vessels, which act as a compression chamber. These are followed by resistive precapillary vessels, arteries, and arterioles of the muscular type, precapillary sphincters [1,2,3,4]. The main function of the vascular system, metabolism, is carried out by a network of capillaries, followed by postcapillary resistance vessels and then by capacitive venous vessels that ensure the return of blood to the heart. The role of the vessels of the compression chamber consists mainly in converting blood flow from pulsating (at the outlet of the ventricles of the heart) to constant, to smooth out pulse fluctuations in pressure and blood flow. Capacitive vessels do not significantly affect overall vascular resistance, but play a dominant role in the formation of vascular bed capacity and, therefore, determine the volume of blood flowing to the heart. Since the hemodynamic parameters of the heart directly depend on the venous return, changes in the capacity in vessels of the venous system, which are determined mainly by the activity of sympathetic nerve fibres, are one of the most important factors that affect the magnitude of cardiac output [1,2,3,4]. Elastic arterial vessels have little resistance to blood flow, and blood enters resistive muscular vessels under almost the same pressure under which it is discharged into the aorta. The greatest amount of resistance is typical for resistive vessels of muscular type, arterioles, and precapillary sphincters, the pressure drop at the outlet of which in a large circle of blood circulation reaches 55-60 mmHg (Singh, D., Singh, S. 2023). As a result, blood enters the capillaries at a pressure of approximately 27 mmHg, about 10-12 mmHg is consumed to overcome resistance of the capillaries, and the blood pressure at the venous end of the capillary is 12-15 mmHg. As a result, the blood pressure in the capacitive vessels approaches 0, which is not enough to return blood to the heart, especially in people with an upright body position, when they also have to overcome the hydrostatic pressure of the blood column. This process becomes possible due to both the suction action of the chest and a sharp drop in intrathoracic pressure during exhalation, as well as the presence of a "peripheral heart" - a massaging effect on the venous vessels of skeletal muscles, since even in a calm state there is a weak nonsynchronous contraction of their fibres [1,2,3,4]. The return of venous blood is also facilitated by the presence of a valve system in the venous vessels, which prevents the reverse flow of blood with a periodic predominance of pressure in the upstream parts of the venous system over the downstream parts. The volumetric velocity of blood flow in blood vessels is determined according to the laws of hydrodynamics, the ratio between two variables, the intravascular pressure, and the resistance of the vessel walls. Therefore, the nature of blood flow in various physiological, extreme, or pathological situations is determined by changes in

vascular resistance, which is an expression of the tone of the vascular wall tone (Zienkiewicz, A., Vihriälä, E., Myllylä, T. 2024). The principle of its regulation is determined by the relationship between the myogenic or basal vascular tone, local and external, and neurohumoral influences on it. Basal vascular tone is determined by internal myogenic activity, that is, the ability of smooth muscle cells to respond by contraction to the stretching effect of intravascular pressure. This leads to the appearance of spontaneous electrical activity of smooth muscle cells and their subsequent contraction [1,2,3,4]. The basal tone is most pronounced in resistive vessels and precapillary sphincters; it is completely insignificant in capacitive venous vessels, the tone of which is determined almost exclusively by the constrictor influence of the sympathetic nervous system. The presence of myogenic activity of vascular smooth muscle cells causes the phenomenon of "autoregulation of local blood flow", that is, its relative independence from changes in blood pressure. Thus, with an increase in intravascular blood pressure, the myogenic activity of vascular smooth muscle cells increases, basal tone increases and, accordingly, resistance, as a result of which blood flow remains constant [1,2,3,4]. On the contrary, with a decrease in intravascular pressure, basal tone decreases, resistance decreases, and blood flow also remains unchanged. This means that the severity of the basal tone, and thus the expansion reserve, is proportional to the pressure in the lumen of the vessel. In addition, an increase in pressure and a corresponding increase in blood flow contribute to leaching of vasodilator metabolites and an increase in basal tone, whereas a drop in pressure and a decrease in blood flow contribute to their accumulation and relaxation of the vessel, which synergistically with the myogenic factor participates in autoregulation of blood flow. The action of metabolites is limited to the precapillary vessels and sphincters and practically does not extend to the postcapillary vessels, which are mainly under the control of the sympathetic division of the autonomic nervous system. The nature of the metabolites responsible for the local regulation of vascular tone remains unknown. Several factors act synergistically in most tissues, including adenosine, lactic acid, reduced oxygen voltage, and increased proton concentration. Neurogenic mechanisms control the entire vascular system, providing opportunities to maintain optimal blood supply to both the entire body as a whole and individual organs and tissues. This is achieved by maintaining adequate blood pressure and IOC, in which the optimal redistribution of blood flow between individual organs and tissues is carried out, depending on their metabolic activity and blood supply needs [1,2,3,4]. Adequate local regulation of blood flow is possible only under conditions of constant monitoring of blood pressure levels. Otherwise, simultaneous vasodilation in a number of areas will lead to a decrease in total vascular resistance, a decrease in blood pressure, and a decrease in tissue perfusion. Maintaining blood pressure in these conditions occurs both by strengthening heart activity and by narrowing in areas with a low need for blood supply. Resistive postcapillary vessels are significantly more sensitive to neurogenic constrictor effects than precapillary vessels. Therefore, in various pathological situations, such as shock, associated with a marked increase in sympathetic activity, the initially increased precapillary resistance gradually decreases, while the postcapillary resistance remains at a high level (Mitchell, CC, Schaeffer, D.G.). As a result, capillary pressure increases and fluid exits the vascular bed; "internal bleeding" occurs, which often causes the shock to enter an irreversible phase.

Researchers Zienkiewicz, A., Vihriälä, E., Myllylä, T ((2024). Influence of arterial vessel diameter and blood viscosity on PTT in the pulsatile flow model. In: Särestöniemi, M., *et al.* Digital Health and Wireless Solutions. NCDHWS 2024. Communications in Computer and Information Science, vol. 2084. Springer, Cham. https://doi.org/10.1007/978-3-031-59091-7_13) are investigated PTT in relation to blood viscosity, vessel dimensions, and pressure levels using a simulated pulsatile flow system in this paper. They were considered using four tubes with varying thickness-to-radius ratios and six water-glycerol solutions to mimic different blood viscosities, all tested under a gradually increasing system pressure. Using the Moens-Korteweg formula with large RMSE when applied across different conditions. They are observed to have high correlation levels ($r = 0.93 \pm 0.09$) paired with a high error (RMSE = 163 ± 100 mHg), suggesting potential inaccuracies in the pressure estimation. They present the recorded signals and discuss how alterations in physical conditions influence the PTT values and the precision of the BP estimation. However, we discuss blood vessel and using the FDM model with daily monitoring [1,2,3,4]. Researchers Singh, D., Singh, S. (2023). Mathematical Modelling of an Incompressible Newtonian Blood Flow for the Carotid Artery. In: Singh, J., Anastassiou, G.A., Baleanu, D., Kumar, D. (eds) Advances in Mathematical Modelling, Applied Analysis, and Computation ICMMAAC 2022. Lecture Notes in Networks and Systems, vol. 666. Springer, Cham. https://doi.org/10.1007/978-3-031-29959-9_16) are studied very interesting and actual topics regarding analysis of the complex blood flow pattern in the 2D model of the human carotid artery because more diseases begin with the artery and are used in practical Newtonian blood flow. In addition, they are used for incompressible 2D Navier-Stokes equations, for which laminar flow was obtained before the artery bifurcation. Authors Ali, A., Hussain, M., Anwar, M.S. *et al.* Mathematical modelling and parametric investigation of blood flow through a stenosis artery. *Appl. Maths. Mech.-Engl. Ed.* 42, 1675–1684 (2021) [1,2,3,4]. https://doi.org/10.1007/s10483-021-2791-8 to examine the blood flow through a cylindrical stenosed blood vessel. Stenthosis disease is caused by abnormal narrowing of the flow of the body. This narrowing causes serious health problems such as heart attack and can decrease blood flow in the blood vessel. Mathematical modelling helps us to analyse such issues. In this study, a mathematical model is considered to explore blood flow in a stenosis artery and is solved numerically using the finite difference method. Moreover, during the study of this paper, researchers used the 3D FDM model with the Navier-Stokes equation to solve the problems of blood flow in a stenosis artery.

2 METHODS

The pathology of the microcirculatory bed includes vascular, intravascular, and extravascular changes. Taking into account the nonlinear mechanical properties of blood vessels is an important condition for their modelling. The corresponding nonlinearity should be reflected in the defining relationships. The question of how to determine the ratios for the walls of blood vessels has been actively investigated in recent decades. The defining relations are given by the type of elastic strain energy (elastic potential). There are many papers devoted to this issue in the case of arteries. They propose various forms of strain energy based on experimental data. Due to the significant difference in the structure of blood vessels depending on their location in the vascular network, none of the options presented is universal for all types of arteries (Ali et al., Hussain et al.) [5,6,7,8]. Among the proposed types of elastic deformation

energy, one can distinguish. According to the ratio of these gradients, fluid diffusion occurs in the arterial part of the capillary and its re-absorption occurs in the venous part. With an average capillary pressure of 20 mmHg, the pressure at the arterial end of the capillary reaches 30 mmHg, at the venous end 15 mmHg. Since the hydraulic pressure in tissues is 8 mmHg, the filtration pressure in the arterial knee of the capillary is 22 mmHg and in the venous pressure is 7 mmHg [5,6,7,8]. At rest, hemodynamics in blood vessels is characterised by the following main aspects:

1. Pulse pressure: At rest, the heart works at a lower rate and force, leading to a decrease in pulse pressure (the difference between systolic and diastolic pressure).
2. Mean arterial pressure: At rest, mean arterial pressure (MAP) is maintained at a stable level by baroreceptors, which regulate vascular tone and heart rate.
3. Volumetric blood flow: At rest, the tissue's demand for oxygen and nutrients is reduced, so the volumetric blood flow decreases. However, critical organs such as the brain and heart continue to receive a stable blood supply.
4. Venous return: At rest, venous return (the amount of blood that returns to the heart) is also reduced because the muscles that normally help push blood through the veins are less active.
5. Regulation of vascular tone: At rest, vasodilation (dilation of blood vessels) occurs in some areas to maintain adequate blood supply at reduced loads.

The basic equation describes the dynamics of pressure P in the vessel: $\beta h(x)\frac{\partial P}{\partial t}=\frac{\partial}{\partial x}\left(\frac{k(x)h(x)}{\mu}\frac{\partial P}{\partial x}\right)$

Here: Let us write down all the data on blood vessels in the same way:

P- is the pressure of the vessel, P_H is the characteristic pressure of the vessel, $\boldsymbol{\mu}$- is the viscosity of the blood of the vessel, k- is the coefficient of permeability, h- is the length of the vessel, $\boldsymbol{\beta}$-is the coefficient of elastic capacity, P_0- is the initial pressure.

Numerical methods, such as the finite difference method or the finite element method can be used to solve this equation. Basic steps for a numerical solution:

1. Equation discretization: Dividing the time and space domains into a grid;
2. Determination of initial conditions: Setting the initial pressure values P* on the entire grid at τ=0;
3. Definition of boundary conditions: Setting boundary conditions at the edges of the mesh;
4. Iterative solution: Using iterative methods to solve a system of equations at each time step.

Divided into $[0, L_x]$ in N cells to steps $\Delta x=\frac{L_x}{N}$;

- Divide the time interval into incremental steps Δτ;
- Set the initial conditions: $P_i^*(x^*)=P^*_H(x_i)$ all i at τ=0;
- For each time step, use the finite difference method to approximate the derivatives.
- Solve a system of linear equations to find the new time step.

Let us rewrite the continuity equation and find:

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$$\frac{A_{i+1}-A_i^n}{\Delta t}+\frac{1}{\Delta t}(A_i^n U_i^n - A_{i-1}^n U_{i-1}^n)=0$$

$$A_i^{n+1}=A_i^n-\frac{\Delta t}{\Delta x}(A_i^n U_i^n - A_{i-1}^n U_{i-1}^n)$$

Let us rewrite the momentum equation and find:

$$\frac{U_i^{n+1}-U_i^n}{\Delta t}+U_i^n-\Delta t\left(\frac{U_{i+1}^n-U_{i-1}^n}{2\Delta x}+\frac{1}{\rho}\frac{P_{i+1}^n-P_{i-1}^n}{2\Delta x}\right.$$

Let us rewrite the equation of state of the vessel:

$$P_i^n=P_{ext}+\frac{\beta}{h_0}\left(\sqrt{\frac{A_i^n}{A_0}}-1\right)$$

Let us rewrite the pressure equation

$$\beta h_i\frac{P_i^{n+1}-P_i^n}{\Delta t}=\frac{1}{\Delta x^2}\left(\frac{k_{i+1/2}h_{i+1/2}}{\mu}\left(P_{i+1}^n-P_i^n\right)-\frac{k_{i-1/2}h_{i-1/2}}{\mu}(P_i^n-P_{i-1}^n)\right)$$

Solution for P_i^{n+1}

$$P_i^{n+1}=P_i^n+\frac{\Delta t}{\beta h_i\Delta x^2}\left(\frac{k_{i+1/2}h_{i+1/2}}{\mu}\left(P_{i+1}^n-P_i^n\right)-\frac{k_{i-1/2}h_{i-1/2}}{\mu}(P_i^n-P_{i-1}^n)\right) \quad (2.8)$$

Let us look specifically at the problem of discretising the pressure equation to show how the coefficients can be found $a_iP_{i-1}^{l+1}+b_iP_{i+1}^{l+1}+cP_{i+1}^{l+1}=-d_i$

$$a_i=\frac{k_{i-1/2}h_{i-1/2}}{\mu\Delta x^2};\qquad b_i=\frac{\beta h_i}{\Delta t}+\frac{k_{i-1/2}h_{i-1/2}}{\mu\Delta x^2}+\frac{k_{i+1/2}h_{i+1/2}}{\mu\Delta x^2};\qquad d_i=\frac{\beta h_i}{\Delta t}P_i^n \quad (2.9)$$

The first equation of the finite difference system *(k+0.5* times the layer) is solved using the sweep method. Its numerical solution is based on the sweep method:

$$\begin{cases}P_i=A_iP_{i+1}+B_i & (i=n-1....,1)\\ a_iP_{i-1}^{l+1}+b_iP_{i+1}^{l+1}+cP_{i+1}^{l+1}=-d_i\end{cases}$$

Here, A_i and B_i run coefficients, and they are determined by the formulas:

$$A_i=\frac{c_i}{b_i-a_iA_{i-1}}\qquad B_i=\frac{a_iB_{i-1}}{b_i-a_iA_{i-1}};\quad (3)$$

The initial values of the drive coefficients are determined from the boundary conditions *0, 0.* For the first boundary condition, in general, it is given above.

3 CONCLUSION

The difference in the speed of movement of individual layers, related to the distance between them, is called the "shear rate". The higher the internal resistance, that is, the viscosity of the liquid, the higher the necessary energy costs to overcome it and set the liquid in motion; this force is called "shear stress". Therefore, the ratio of shear stress to shear rate is a measure of the viscosity of a liquid. All liquids are divided into homogeneous, Newtonian, and anomalous. Homogeneous liquids are characterised by a constant viscosity value that does not

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depend on shear forces and flow velocity, whereas the viscosity of abnormal liquids is variable and varies depending on the conditions under which their movement is carried out [5,6,7,8].

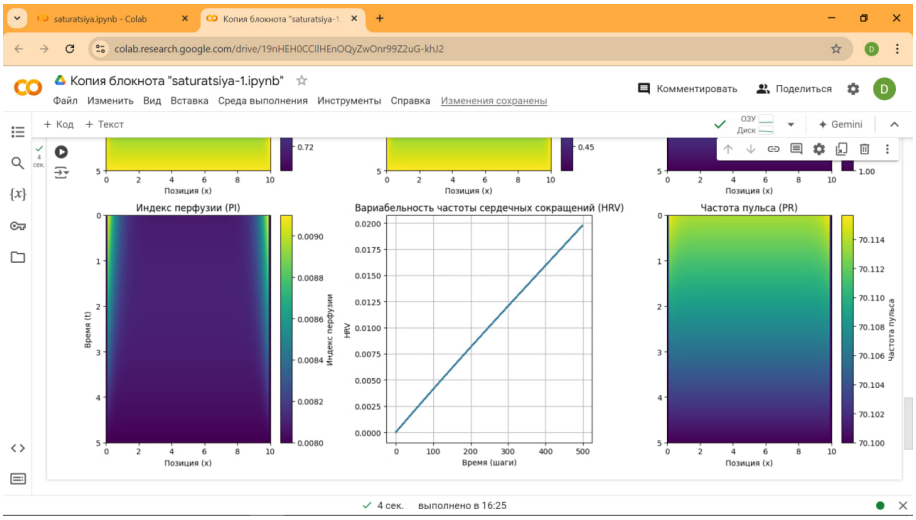

Fig. 1. Blood is a heterogeneous multicomponent

From a biophysical point of view, blood is a heterogeneous multicomponent system of corpuscular nature, that is, a suspension of shaped elements in a colloidal solution of proteins, lipids, and electrolytes, which is blood plasma. Tissue perfusion is ensured by the passage of this concentrated suspension of solid particles through a system of microvessels, the diameter of which in some areas is smaller than the diameter of the particles themselves (Fig.1). Despite the fact that the specific gravity of blood is approaching the specific gravity of water, blood differs sharply from the latter in rheological properties. This difference is manifested primarily in the microcirculation system, since blood behaves like a homogeneous liquid in large vessels. In the microcirculatory bed, under conditions where the diameter of the vessel becomes comparable to the size of the shaped elements of blood, it acquires the properties of an inhomogeneous liquid. These properties are most pronounced at the level of capillaries, the diameter of which may be even smaller than the size of the shaped elements (Fig.2) [9,10,11,12].

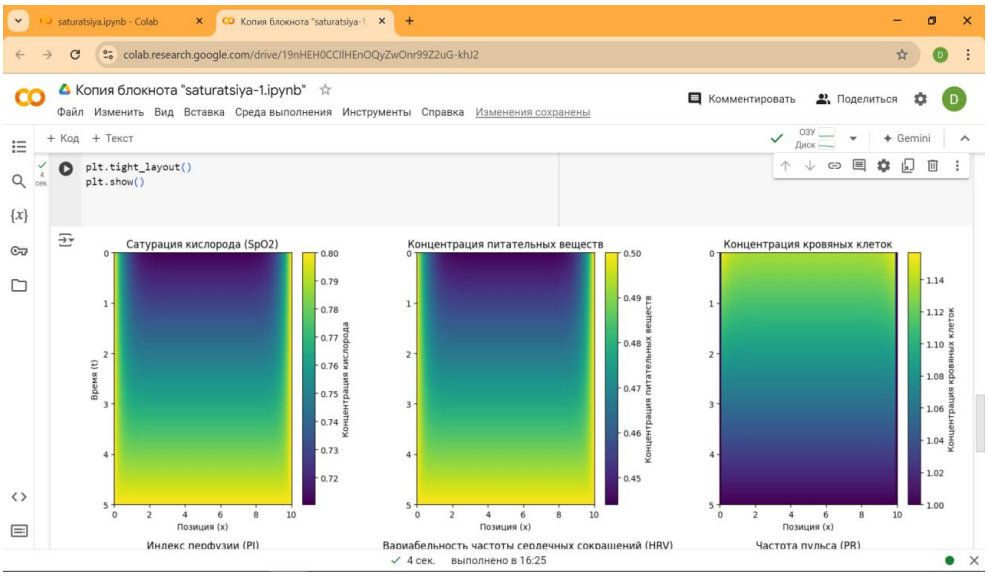

Fig. 2. Blood is a heterogeneous multicomponent

4 DISCUSSION AND RESULTS

A similar effect is observed during the transition of shock, in particular cardiac shock, from a reversible phase to an irreversible one, when, against the background of dilated arterioles, a spasm of post-capillary resistive vessels leads to an increase in capillary pressure, filtration of the liquid part of the blood, and its thickening, followed by a sharp violation of microcirculation. In the microcirculation system, the rheological properties of blood and its "fluidity" play an important role in maintaining tissue perfusion (Bader et al.)[13-17,18-25,26-30]. Any liquid is characterised by such a concept as "viscosity", since the column of liquid moves through the tube not as a single whole but in separate layers that shift relative to each other. Because of the presence of molecular forces of adhesion between the individual layers of the flow, internal friction develops, the severity of which determines the viscosity of the liquid. As a result, individual layers will shift at different speeds; the highest speed is typical for the central or axial layer, and the lowest for the wall layer, the speed of movement of the axial layer is about two times higher than the average speed. Due to the velocity distribution of the individual layers, the flow profile acquires a parabolic shape. At high flow velocity, after reaching a critical point, the flow loses its laminar character and becomes turbulent, in which the parallel nature of the movement of individual layers is lost and vortices arise [13-17,18-25,26-30]. Considerable energy is spent on their creation, because of which, with the turbulent nature of the flow, the direct relationship between its velocity and the pressure value is lost.

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