

MODELING STUDIES OF BASILAR ARTERY BLOOD FLOW GIVEN THE GLOBAL HEMODYNAMICS FACTORS

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Abstract: The main disease of cerebral circulation is an aneurysm of the basilar artery. The paper presents the concept of developing a multiscale hemodynamics mathematical model of the cardiovascular system, including the model of global hemodynamics, the arterial tree model and the model of basilar artery bifurcation. This model allows computing hemodynamic parameters at the bifurcation of the basilar artery. Using the mathematical modeling results we can conclude the probability of aneurysm.

Cerebral circulation is a complex system of blood vessels responsible for the transportation of blood and nutrients to the brain cells. One of the most common disorders of cerebral circulation is an aneurysm of the basilar artery [1]. The basilar artery is the main artery of the brain; it is formed where the two vertebral arteries join together at the base of the skull.

Aneurysm is bulge in the vessel wall that is caused by its thinning or stretching. It occurs in the places where the vessel wall is damaged (typically at the arteries bifurcation).

For the investigation of basilar artery circulation it is proposed to use a multiscale mathematical model of hemodynamics [2], which is a set of mathematical models of circulation with different levels of detail.

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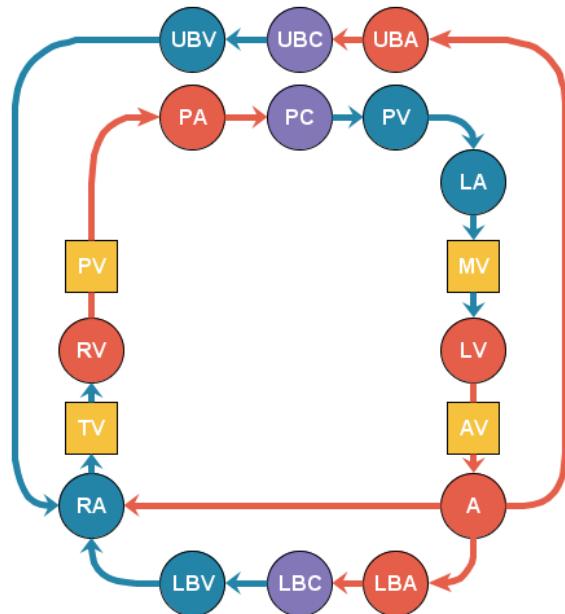


Fig. 1. 0D cardiovascular system model scheme:

LV – left ventricle; AV – aortic valve; A – aorta; UBA – upper body arteries; UBC – upper body capillaries; UBV – upper body veins; LBA – lower body arteries; LBC – lower body capillaries; LBV – lower body veins; RA – right atrium; TV – tricuspid valve; RV – right ventricle;

PV – pulmonary valve; PA – pulmonary arteries; PC – pulmonary capillaries; PV – pulmonary veins; LA – left atrium; MV – mitral valve

0D model is used to describe the hemodynamics of the cardiovascular system in general (Fig. 1). According to this method, the whole cardiovascular system is divided into a group of individual elements (compartments) [3–5].

Chamber analogy was used to develop the global hemodynamics model. In this case a cardiovascular system is represented as a series of connected chambers – elastic reservoirs filled with blood. At each time moment the chamber is characterized by blood volume $V(t)$ in it, pressure $P(t)$ in vessel wall, inlet $q^{\text{in}}(t)$ and outlet $q^{\text{out}}(t)$ flow rate. The elastic chamber scheme is shown in Fig. 2.

According to mass conversation law, the equation for volume of i -the chamber was written as follows

$$\frac{dV_i(t)}{dt} = q_i^{\text{in}}(t) - q_i^{\text{out}}(t), \quad (1)$$

where $q_i^{\text{in}}(t)$ is a vector of inlet flows (sm^3/s); $q_i^{\text{out}}(t)$ is a vector of outlet flows.

To determine the pressure $P_i(t)$ in the i -th chamber wall the following assumption is made: the more blood is in the chamber, the more is the wall stretching, and, correspondingly, the greater is the pressure in the chamber wall. The considered dependence is described by the expression:

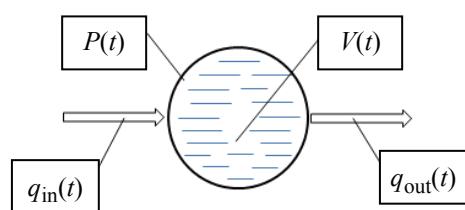


Fig. 2. The scheme of cardiovascular system chamber

$$P_i(t) = e_i(V_i(t) - U_i), \quad (2)$$

where e_i is chamber wall stiffness, mmHg/sm³; U_i is unstressed volume, which is spreading, but not stretching the chamber wall, sm³.

The unstressed blood volume is the greatest part of the full chamber volume which itself yet doesn't stretch a chamber wall. Filling the chamber, the blood at first spreads it and only then when the volume becomes sufficient, it stretches.

The blood flow rate is determined by the Poiseuille law

$$q_{\text{in},\text{out}}(t) = \rho_{\text{in},\text{out}}(P_{\text{in}}(t) - P_{\text{out}}(t)), \quad (3)$$

where $\rho_{\text{in},\text{out}}$ is link conductivity, sm³/(mmHg·S).

For blood flows from the aorta into the systemic circulation arteries the inertial blood properties are taken into account

$$\frac{dq_{\text{in},\text{out}}(t)}{dt} = \frac{1}{L_{\text{in},\text{out}}} \left(\Delta P_{\text{in},\text{out}} - \frac{q_{\text{in},\text{out}}(t)}{\rho_{\text{in},\text{out}}} \right), \quad \Delta P_{\text{in},\text{out}} = P_{\text{in}}(t) - P_{\text{out}}(t),$$

where L_{ij} is blood inertia coefficient, mmHg·s²/sm³.

Chamber analogy also was used for heart modeling. The heart was represented as a set of four chambers – the left ventricle, left atrium, right ventricle and right atrium. Left and right atria chambers are passive because they are not involved in the heart contractile activity. Therefore, formula (1) – (3) can be used for them. To describe the contractile activity of the left and right ventricles, they are considered as sources of pressure. Laplace law for a thin-walled sphere is used to describe pressure in the i -th ventricle chamber [5]:

$$\sigma_i(t) = \frac{P_i(t)r_i(t)}{2h_i}; \quad (4)$$

$$r_i(t) = \sqrt[3]{\frac{3V_i(t)}{4\pi}}, \quad (5)$$

where $\sigma_i(t)$ is the chamber wall strain, mmHg; h_i is the wall thickness, sm; $r_i(t)$ is the sphere radius, sm.

From (4), (5) it follows that

$$P_i(t) = 4\sqrt[3]{\frac{\pi}{6}} \frac{h}{\sqrt[3]{V_i(t)}} \sigma_i(t).$$

The chamber wall is a heart muscle (myocardium), consisting of four elements. Myocardial functional structure includes a contractile element, which can be reduced at excitation, serial connected elastic element and parallel connected elastic element. To adequately describe the behavior of myocardial relaxation phase, the viscous element is included parallel to the contractile element. In the pulsating heart model, the heart activity is seen as the sequence of contraction (systole) and relaxation (diastole) phases. The characteristics of this process are: the heartbeat period T and systole duration T_{sys} . The beginning

of the cardiac cycle is the time of the diastole to systole phase change. In the general case T and T_{sys} may be different for different cycles, whereas moments of the systole end $T_{\text{es}}(n)$ and the diastole end $T_{\text{ed}}(n)$ n -th circle, $n = 1, 2, 3, \dots$ are expressed by:

$$t_{\text{es}}(n) = \sum_{j=1}^{n-1} T(j) + T_{\text{sys}}(n) + t_0, \quad t_{\text{ed}}(n) = \sum_{j=1}^n T(j) + t_0.$$

For the complete heart description the valves activity is considered. The valves are represented by elements with variable conductivity depending on a pressure drop between the linked chambers. Then the modified formula (3) for a blood flow via the valve will be as follows:

$$q_{\text{in,out}}(t) = \rho_{\text{in,out}}(\Delta P_{\text{in,out}}) \Delta P_{\text{in,out}}.$$

For an adequate heart valves description the blood flow through the valve in the closing direction is considered (backflow of blood). If there is a pressure drop in the valve opening direction the valve is opened. Valve closing happens by a blood volume Δ (closing volume) movement through the valve in the direction opposite to its opening direction

$$\rho_{\text{in,out}}(\Delta P_{\text{in,out}}) = \frac{2\rho^*}{1 + e^{-\beta\Delta}}, \text{ if } |\Delta| < \Delta^*,$$

where ρ^* is open valve conductivity; Δ is blood volume passing through the valve in the opposite direction; Δ^* is closing volume; β is valve conductivity decrease rate.

The proposed structure of 1D model, consisting of 48 arteries is shown in Fig. 3. This model describes upper body arteries and cerebral circulation which is described in detail.

For proper functioning of the model 1D the appropriate boundary conditions at the terminal edges of the arterial tree are required. Such boundary conditions can be obtained by using 0D model. In this approach, the blood flow and pressure which is calculated by 0D model converted in accordance with a particular algorithm, and set as the boundary conditions for the model 1D.

Using 3D model it is planned to describe the basilar artery local hemodynamics. The researcher will receive information about a blood flow rate of change and blood pressure at any point of the basilar artery. According to the results the risk of developing an aneurysm of the basilar artery can be predicted. Also, with this model it is possible to conduct numerical experiments to determine the optimal stent position in basilar artery stenting.

For 3D model, as well as for 1D model, the definition of appropriate boundary conditions remains a major problem. To solve this problem the boundary conditions are applied from the results of 1D model converting according to an appropriate algorithm.

Thus, a correct modeling of the investigated arterial segment (basilar artery) requires development a set of interrelated models 0D, 1D and 3D, which together form a multiscale mathematical model of the hemodynamics of the cardiovascular system (Fig. 4).

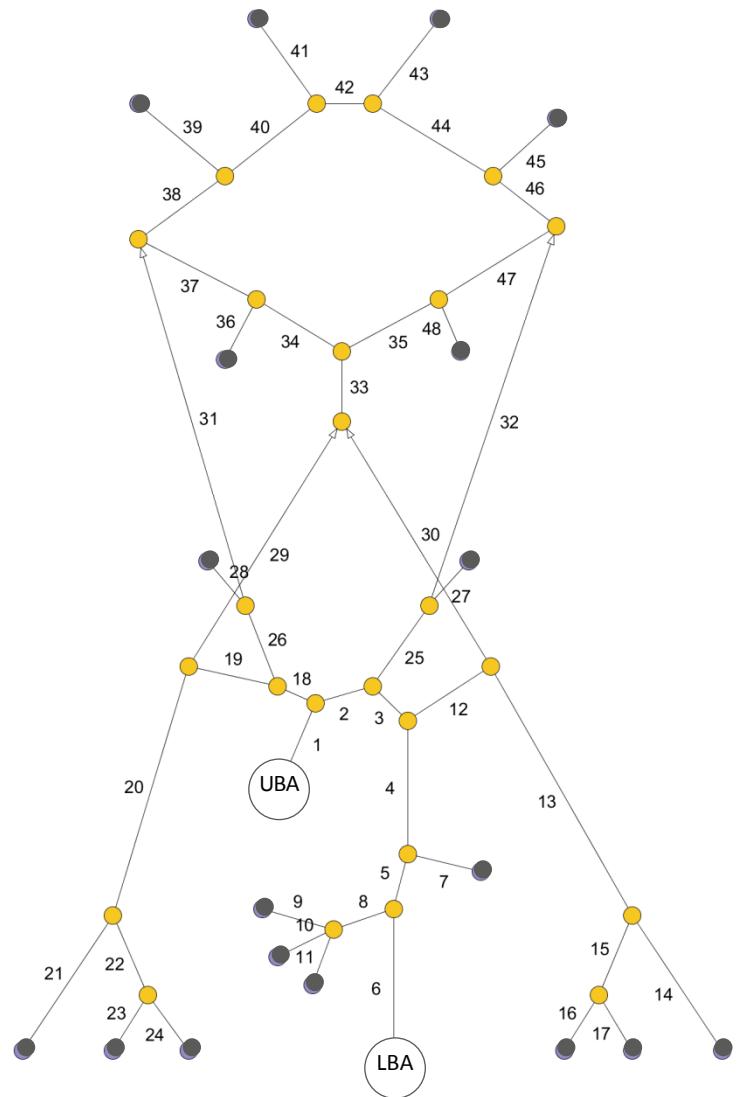


Fig. 3. The structure of one-dimensional mathematical model of the upper body arteries and brain

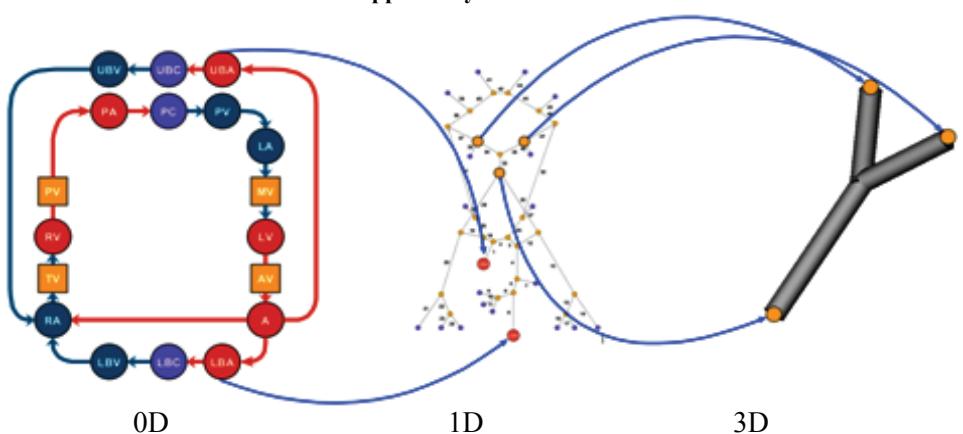


Fig. 4. Structure of multiscale hemodynamics model

This model allows you to combine models of the global hemodynamics, hemodynamic blood stream and local hemodynamics that takes advantage of each type of models and minimizes their weaknesses. For example, a multiscale model of hemodynamics provides an opportunity to study the impact of regulation (0D model) at the local segment of cardiovascular system (3D model).

To test the reliability of the numerical simulations of basilar artery hemodynamics it is planned to use the experimental setup located at the Technical University of Munich (Munich, Germany) in the clinic Rechts der Isar [6]. To do this, using the methods of angiography the geometric model of the basilar artery will be constructed. The stereolithography obtained silicone model, will be mounted on a movable *XYZ*-table so that the velocity of the blood can be measured accurately at any point of the model. The set-up includes a laser Doppler anemometer for measuring the velocity of blood flow in vessels silicone elastic models and a laser vibrometer to measure vibrations of the vessel wall.

The experimental set-up is shown in Fig. 5. With laser-Doppler-anemometry it is possible to measure the velocity distribution of a flow with high spatial ($70 \mu\text{m}$) and temporal (1 ms) resolution in transparent models of human vessels without disturbing the flow [6 – 9]. The flow velocity is measured with a 5 mW He-Ne laser-Doppler-anemometer (BBC Goerz. Spectra physics, Munich, Germany, 5 in Fig. 5) with a wavelength of $\lambda = 632.8 \text{ nm}$.

The vessel's model is mounted on an x-y-z-moving table so that velocities can be measured and recorded very precisely at each point of interest. In order to obtain a representative picture of the flow, the velocity is measured at each point for seven pulse cycles and then averaged.

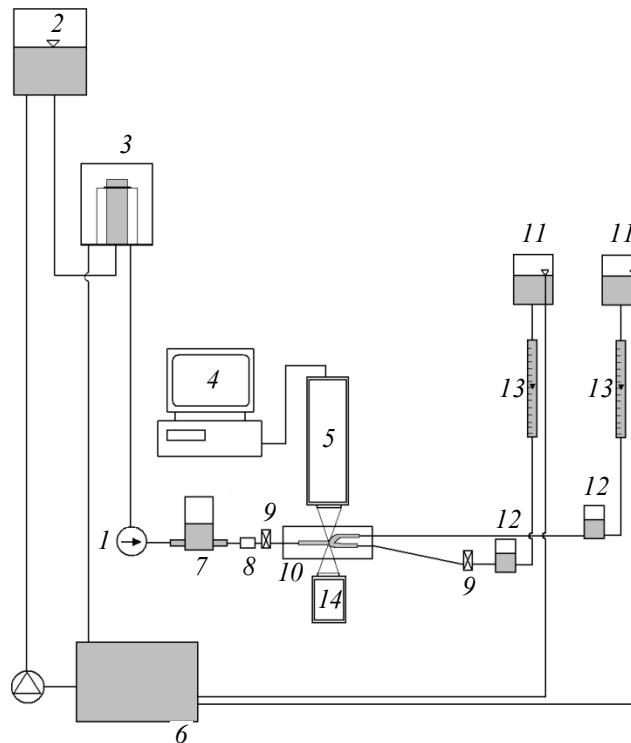


Fig. 5. Experimental set-up

To simulate the physiological human flow conditions we used a blood-like fluid developed in our laboratory. The perfusion fluid is a transparent glycerol-water mixture to which a small amount of Separan AP-302 and Separan AP-45 (Dow Chemical) is added. This fluid exhibits a non-Newtonian flow behavior similar to that of blood.

The perfusion fluid is pumped from a collecting tank (6 in Fig. 5) through an elevated tank (2 in Fig. 5) into an overflow tank (3 in Fig. 5). It maintains a constant static pressure in the model. A computer driven piston pump (1 in Fig. 5) superimposes an oscillatory pulse on the stationary flow creating a pulsatile flow. Buffer tanks (7, 12 in Fig. 5) are installed in front and behind the model to simulate the compliance of a human aorta. The fluid streams through the model (10 in Fig. 5) back into the collecting tank (6 in Fig. 5). The regulation tanks (11 in Fig. 5) allow an adjustment of the flow rate. Pressure is measured with inductive pressure transducers (9 in Fig. 5). Systolic and diastolic pressure can be set in the range between 150 and 70 mmHg.

Velocity, pressure and flow rate data obtained with the above described experimental set-up allow correlation with the mathematical simulation in order to evaluate its reliability.

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**Модельные исследования базилярной артерии
с учетом глобальных факторов гемодинамики**

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Ключевые слова и фразы: аневризма; базилярная артерия; гемодинамика; многоуровневая модель; сердечно-сосудистая система.

Аннотация: Основным нарушением церебрального кровообращения является аневризма базилярной артерии. В работе предложена концепция построения многоуровневой математической модели гемодинамики сердечно-сосудистой системы, включающей в себя модель глобальной гемодинамики, модель артериального русла и модель бифуркации базилярной артерии. Данная модель позволит рассчитать гемодинамические параметры в области бифуркации базилярной артерии. На основе анализа полученных результатов можно сделать вывод о вероятности возникновения аневризмы.

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