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NUMERICAL SIMULATION OF THE HEMODYNAMICS AND BIOMECHANICS OF THE ARTERIAL SYSTEM

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A mathematical model of blood flow in arterial vessels with account for the nonstationary character of the laminar flow and the time-varying pressure drop is considered. The viscosity of blood is assumed to obey a power law. The rheological properties of the walls of arterial vessels are described by i) the Voigt–Kelvin model, ii) the standard-body model, and iii) the three-component Voigt–Kelvin model. As a result of numerical simulation, flow-pressure characteristics of various arteries are obtained.

Blood flow in the human organism is traditionally an object of basic investigations in the field of thermophysics and hydrodynamics. Robert Mayer formulated the first law of thermodynamics on the basis of physiological and pathological observations of features of blood circulation under tropical conditions [1]. Investigations by Euler and Stokes were based on an analytical description of blood flow; experimental investigations of blood flow in capillaries were carried out by Poiseuille [2]. A detailed analysis of blood-circulation hydromechanics in the human organism in the normal state and in pathological cases is essential for cardiac surgery, oncology, hematology, development of survival systems, and hyper- and hypothermia. We consider a mathematical description of nonstationary blood flow in the human arterial system from the viewpoint of the mechanics of continuous media with account for the rheological properties of the vessel walls.

The walls of all large arteries have three shells: inner, middle, and outer (Fig. 1). The inner shell includes the endothelium, subendothelial layer, and inner elastin membrane. The subendothelial layer consists of thin elastin and collagen fibers, connective tissue, and the main substance. The inner membrane contains elastic fibers braided with collagen fibers. Collagen is a highly durable protein whose fibers form spirals and therefore permit some stretching without stress. The skeleton of the middle shell includes interconnected concentric elastic membranes formed by highly stretchable elastin protein fibers. The middle shell has a composite structure and consists of several (up to 60) coaxial membranes interconnected by plane muscle fibers covered by collagen fibers. The cavity between the membranes is filled with porous tissues and a fluid. Smooth-muscle cells and fibers attached to elastic membranes are situated in the middle layer of the wall of a vessel and are parallel to or inclined with respect to its direction. A decrease in the vessel diameter is accompanied by an increase in the number of muscle-tissue layers and a decrease in the number of elastic-tissue layers, the latter ultimately transforming into two thin membranes on the inner and outer surfaces of the muscle shell. The outer elastic membrane situated on the boundary of the middle and outer shells consists of longitudinally oriented thick elastic fibers and circularly arranged collagen-fibril bunches, which, with the aid of a network of interconnecting transverse collagen fibers, form a unified structure. Elastic-fiber bunches with numerous interconnections are situated in the surface layers of the outer shell [3-5].

The value of the elasticity modulus of such a complicated system as the fiber wall cannot be determined unambiguously but rather is a complicated function of the temperature, intravessel pressure, partial pressure of oxygen, and other factors. Vessel conductivities are calculated with account for the deformability and elasticity of the walls. Arterial walls have rather high deformability values. For example, the deformation ε can reach 80% in the carotid artery, 13 to 22% in the anterior, middle, and posterior meningeal arteries, according to [6], and 40% in the femoral artery [7].

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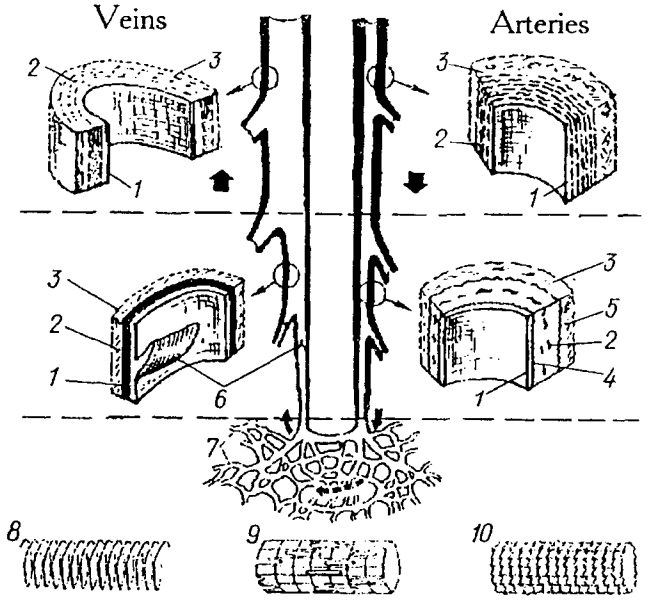

Fig. 1. Structure of blood vessels [3-5]: 1) inner shell (intima); 2) middle shell (media); 3) outer shell (adventicia); 4) inner elastic membrane; 5) outer elastic membrane; 6) vein vent; 7) capillaries; 8) smooth muscles; 9) elastin; 10) collagen.

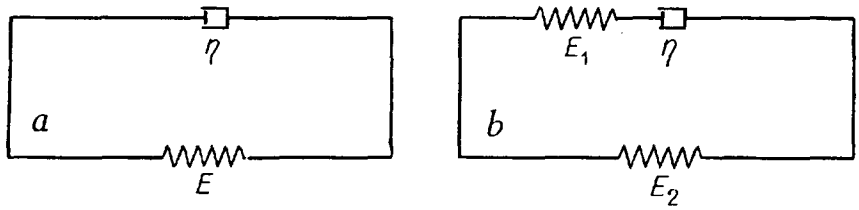

Fig. 2. Voigt–Kelvin viscoelastic-solid model (a) and standard-body model (b).

The first variant of accounting for the viscoelastic properties of vessels consists in an attempt at their description based on the Voigt–Kelvin model [8]. An increase in the radius of the vessel wall is described in this case by the differential equation

$$\frac{d\varepsilon}{dt} + \frac{E}{\eta}\varepsilon = \frac{\sigma}{\eta}, \tag{1a}$$

where ε is the relative radial deformation, equal to the change in the vessel radius due to the intravessel pressure.

The second variant considered here is the standard-body model used for description of many soft tissues [4], in particular, muscle tissue, which is, in fact, a parallel connection of Hooke's and Maxwell's models (Fig. 2).

The rheological equation of the standard body is as follows:

$$\frac{d\varepsilon}{dt} = \frac{\sigma}{\eta\left(1 + \dfrac{E_2}{E_1}\right)} + \frac{1}{E_1 + E_2}\frac{d\sigma}{dt} - \frac{E_2}{\eta\left(1 + \dfrac{E_2}{E_1}\right)}\varepsilon. \tag{1b}$$

Finally, in accordance with the concept of a three-layer structure of the arterial wall, the variant of representation of each of the layers by the Voigt–Kelvin viscoelastic-solid model [7] is considered. One and the same load σ is imposed on each of the three layers, and the total deformation of the vessel wall ε is the sum of the deformations of the three layers. An increase in the vessel radius is described by the three differential equations

$$\frac{d\varepsilon_1}{dt} + \frac{E_1}{\eta_1}\varepsilon_1 = \frac{\sigma}{\eta_1}, \quad \frac{d\varepsilon_2}{dt} + \frac{E_2}{\eta_2}\varepsilon_2 = \frac{\sigma}{\eta_2} \quad \frac{d\varepsilon_3}{dt} + \frac{E_3}{\eta_3}\varepsilon_3 = \frac{\sigma}{\eta_3}, \quad \varepsilon = \varepsilon_1 + \varepsilon_2 + \varepsilon_3, \tag{1c}$$

where ε_i is the radial deformation, equal to the relative increment in the vessel radius due to the intravessel pressure σ, E_i is the modulus of elasticity of the vessel wall, and η_i is the viscosity of the i-th layer of the vessel wall, not coinciding with the viscosity of blood μ. In each of the layers, the deformation is delayed with respect to the applied stress by the retardation time $\theta_i = \eta_i/E_i$. The stress σ in the arterial-vessel wall is defined as the blood pressure averaged over the vessel length:

$$\sigma = p_0 + \frac{1}{2}\Delta p,$$

where p_0 is the pressure at the vessel entrance and Δp is the pressure drop over the vessel length l.

Existing experimental data on blood circulation with local heating of vessels with a hot physiological solution *in vivo* point to the following features of the rheological behavior of vessel walls: arteriols virtually do not change their radii upon heating to 48 °C, and upon heating to 65 °C or more, the opening of vessels – both arteriols and venules – always decreases [9]. This corresponds to a constant value of the modulus of elasticity of arteriols for temperatures up to 48 °C and an increase in their modulus of elasticity upon heating to 65 °C or more. The complicated character of this dependence is most likely explained by the fact that the components of the vessel wall – collagen, elastin, and muscle fibers – have different temperature dependences of the modulus of elasticity. The modulus of elasticity of collagen and muscles drops with temperature, whereas that of elastin increases [10, 11]. Upon heating by the radiation of an Ar laser with a wavelength $\lambda = 514$ nm, the radii of venules can increase almost twofold during irradiation for 40 sec and they relax to their original values in a time of about 100 sec [12].

In the case of general hyperthermia of the organism, the reaction of vessels to an increase in the temperature of the body depends on the type of vessel, its diameter, and the heating time. Thus, in considering microvessels of diameter 60–70 μm, arteriols expand in the initial stage of heating and collapse with further heating; on the other hand, venules only collapse, and the most intense collapse takes place during the first hour of heating [13-15]. Oscillatory changes with alternating expansions and collapses are characteristic of arteriols and venules of smaller diameters. The largest microvessels (of diameter 60–70 μm) collapse upon heating. The collapse and expansion of vessels also depend on the concentration (tension) of oxygen in the blood [16].

The relationship between the modulus of elasticity and the intravessel pressure under static conditions was determined in [17] for thoracic and ventral aortas and femoral and carotid arteries. The modulus of elasticity of all these vessels increases linearly from 0.1 to 2 MPa (i.e., from 750 to 15,000 mm Hg) with increase in the intravessel pressure from 20 to 240 mm Hg. With periodic perturbations at a frequency of 2 Hz, the modulus of elasticity of vessels is increased [18]. It equals 0.47 MPa for the thoracic aorta, 1.09 MPa for the ventral aorta, 1.2 MPa for the femoral artery, and 1.1 MPa for the carotid artery. Values of the same order of magnitude were obtained n measurements of the modulus of elasticity of the vessel shell of the fundus of the eye – 1.3 to 3.7 MPa (i.e., 9800 to 27,800 mm Hg) depending on the direction [19]. The elasticity depends on the collagen-to-elastin ratio; thus, for example, the elasticity modulus of the cornea, containing only collagen, equals 4.8 MPa, or 36,000 mm Hg [20].

According to data of [10, 11], the modulus of elasticity of elastin increases linearly for temperatures up to 60 °C:

$$E = [0.363 + 0.00812\,(T - 37)], \quad \text{MPa}; \tag{2}$$

the modulus of elasticity of collagen decreases linearly for temperatures up to 60 °C:

$$E = [4.46 - 0.02\,(T - 37)], \quad \text{MPa}. \tag{3}$$

It is evident from these data that the above-presented experimental values of the modulus of elasticity of arteries are close to that of elastin.

Restricting the temperature to a value of 60 °C in Eqs. (2) and (3) is substantiated by the fact that a further increase in temperature leads to temperature-induced shrinkage of collagen [21, 22].

At the present stage of investigations, the viscosity of blood is assumed to scale as a power of the shear velocity gradient $\gamma = du/dr$ for all vessels:

$$\mu = \mu_0 \left| \frac{du}{dr} \right|^{n-1}, \tag{4}$$

and for blood, $n = 0.9-0.92$, and therefore, the viscosity depends only weakly on the shear velocity gradient. According to [23], the shear velocity gradient γ in the arterial part of the blood-circulation system has the following values: γ is about 100 $\sec^{-1}$ in the aorta, up to 400 $\sec^{-1}$ in large arteries, and $\gamma < 100$ $\sec^{-1}$ in small arteries and arteriols. Small shear velocities are characteristic only of veins. The entire volume of circulating blood can be divided conventionally into two portions [24]: a rapidly circulating one (occupies about 60% of the volume and fills cardiac cavities and vessels with a diameter larger than 100 μm) and a slowly circulating one (occupies about 40% of the volume and fills vessels with a diameter smaller than 100 μm, vessels of parenchimatose organs, and the microcirculatory alveus). The period of complete circulation of the blood slightly exceeds 1 min [24].

The mathematical model of the stationary distribution of blood flows is a detailed form of the continuity equation for the arterial network, including 128 vessels [25, 26]. Each conjugation point is either a transition to a vessel with a different diameter or a branching. The number of branchings at some nodes can be as high as six. The flow in all vessels is assumed to be laminar. Here, the unknown quantities are the pressure at the vessel conjugation points. At each of the node points of the arterial-system scheme, the condition of zero algebraic sum of all incoming and outgoing flows should be satisfied. The stipulation of the indicated conditions for all nodes forms a system of linear algebraic equations whose solution yields an initial approximation to the solution of the problem of evaluation of the time-dependent blood flow.

The equation of the time-dependent blood flow in each vessel is as follows:

$$\frac{\rho}{\pi r_0^2 (1+\varepsilon)^2} \frac{dq}{dt} + \frac{8\mu q}{\pi r_0^4 (1+\varepsilon)^4} = -\frac{\Delta p}{l}, \tag{5}$$

where r_0 is the initial vessel radius. This equation represents the balance of the forces acting on the blood flowing in the vessel. The left-hand side of the equation is the sum of the forces of inertia and viscous friction, and the right-hand side is the external compelling force. The equation does not take into account forces connected with the change in the velocity profile along the vessel length.

The time-dependent flow of arterial blood with account for the viscoelastic properties of the vessel walls is determined by a system of first-order differential equations: equation of motion (5) and equations (1) describing the deformation of the vessel walls. In the case of the Voigt–Kelvin or the standard-body model the latter is a single equation of the form (1a) or (1b), and in the case of the three-component model it is three equations (1c).

The conditions under which one can write equations for stationary flows at branchings of the hydraulic system are as follows: the kinetic energy of the liquid is small compared to the potential energy, and the geometric dimensions of the branchings are negligible compared to the acoustic wavelength within the frequency range at hand. The stipulation of the indicated conditions for all node points forms a system of linear algebraic equations. The flow rate through each of the vessels is expressed in terms of the difference of pressures at the entrance to and exit from the vessel in a form similar to Ohm's law:

$$q_i = c_i (p_{\text{in}} - p_{\text{out}}), \tag{6}$$

where q_i is the volume flow rate in the i-th vessel, c_i is the conductivity of the i-th vessel, and p_{in} and p_{out} are pressures at the entrance to and exit from the vessel.

The conductivity of the i-th vessel is determined in the laminar-flow approximation by the Stokes formula

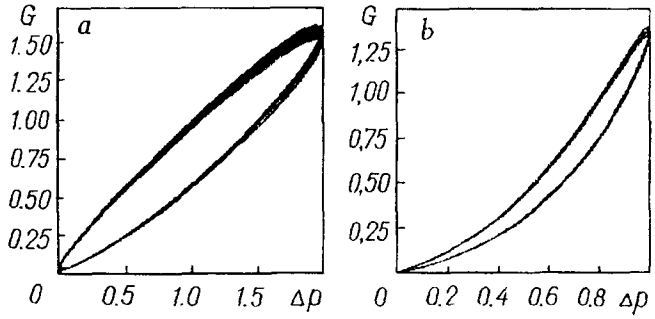

Fig. 3. Pressure-flow characteristics for the internal mamary artery: a) Voigt–Kelvin approximation, modulus of elasticity $E = 8 \cdot 10^5$ Pa, viscosity of the vessel wall 10^7 Pa·sec; b) vessel wall described by the standard-body model with the parameters $E_1 = 0.5 \cdot 10^5$ Pa, $E_2 = 10^5$ Pa, retardation time 60 sec, pulse rate 70 strokes per minute.

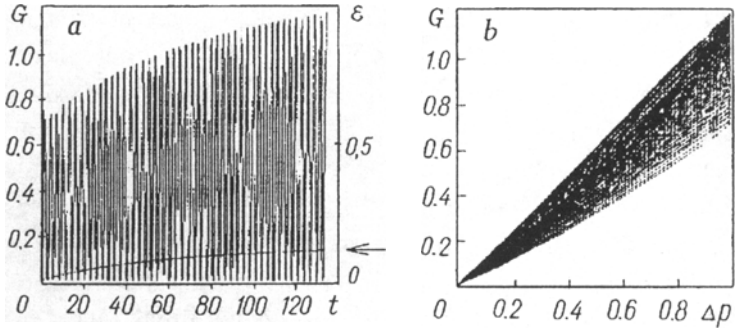

Fig. 4. Blood flow rate and deformation (shown by the arrow) (a) and the pressure-flow characteristic (b) of the three-component Voigt–Kelvin model for the internal mammary artery; pulse rate 70 strokes per minute; retardation times $\theta_1 = 100$ sec (elastin), $\theta_2 = 28.5$ sec (collagen), and $\theta_3 = 0.025$ sec (muscle tissue).

$$c_i = \frac{\pi R_i^4}{8\mu l}, \tag{7}$$

where R_i is the radius of the i-th vessel of the length l, expressed in terms of the initial (anatomical) value of the radius r_{0i} with a correction ε for the total deformation in the radial direction under the effect of the intravessel pressure:

$$R_i = r_{0i}(1 + \varepsilon). \tag{8}$$

Stationary expressions (6) and (7) are obtained from equation of motion (5) at $dq/dt = 0$.

The values of the moduli of elasticity and the retardation times $\theta_i = \eta_i / E_i$ for the three layers of the vessel wall were determined based on the consideration that the viscosity of all three layers of the vessel wall has a value of the order of 10^7 Pa·sec, and therefore $\theta_1 = 100$ sec, $\theta_2 = 28.5$ sec, and $\theta_3 = 0.025$ sec. Since no reliable data on the viscosity of muscle tissue are available, calculations were also carried out for θ_3 having a value two orders of magnitude higher: $\theta_3 = 2.5$ sec.

The system of equations describing the time-dependent blood flow in the vessel and the viscoelastic properties of the vessel walls, i.e., equation of motion (5) along with the system of three deformation equations (1) for the three layers of the vessel wall, is solved by the single-step Runge–Kutta–Felberg method of fourth order of accuracy with automatic choice of the step. The stationary value of the blood flow rate is used as the initial approximation for calculation of time-dependent blood flow rates and pressures in the artery.

Figure 3 presents pressure-flow characteristics for the short internal mammary artery ($L/r = 2.56$). Figure 3a shows the Voigt–Kelvin approximation with a modulus of elasticity $E = 8 \cdot 10^5$ Pa and a vessel wall viscosity of

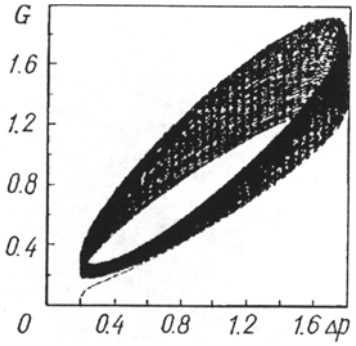

Fig. 5. Change in the pressure-flow characteristic of the internal mammary artery with increase in the pulse frequency to 2 Hz (120 strokes per minute) when the vessel wall is described by the three-component Voigt–Kelvin model.

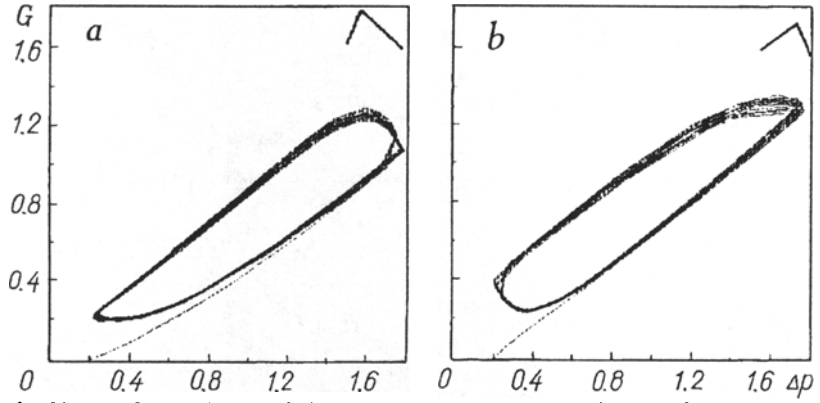

Fig. 6. Effect of the shape of the incoming pressure pulse on the pressure-flow characteristic: triangular pulse with a steep (a) and a flattened (b) front slope.

10^7 Pa·sec, which corresponds to data of [25]; in Fig. 3b, the vessel wall is represented by the standard-body model with parameters $E_1 = 0.5 \cdot 10^5$ Pa, $E_2 = 10^5$ Pa, and a retardation time of 60 sec. In both variants, the pulse rate was 70 strokes per minute with a modulation depth of 100%, i.e., the pressure varies from zero to its stationary value according to the harmonic law

$$\Delta p\,(t) = \Delta p_0\,(1 - \cos\,(\omega t))/2\,, \tag{9}$$

where $\omega = 2\pi/\tau$ is the angular frequency, and $\tau = 1/f$.

A calculation by the three-component Voigt–Kelvin model for the same artery is presented in Fig. 4. Figure 4a shows the time-dependent blood flow rate in the artery and the deformation of the vessel radius for a harmonic variation of the pressure drop. The pulse rate was also 70 strokes per minute. The retardation times were $\theta_1 = 100$ sec (elastin), $\theta_2 = 28.5$ sec (collagen), and $\theta_3 = 0.025$ sec (muscle tissue). The modulation depth was also 100%. It is evident that the flow-rate envelope changes its slope at instants equal to the retardation times. Figure 4b presents the pressure-flow characteristic (in dimensionless form). As time elapses, the hysteresis loops shift upward and acquire a progressively steeper slope. Figure 5 illustrates the change in the pressure-flow characteristic for the internal mammary artery ($L/r = 2.56$) with increase in the pulse rate to 2 Hz (120 strokes per minute) when the vessel wall is described by the three-component Voigt–Kelvin model with the same parameters. Here the modulation depth is 80%, i.e., the pressure varies from 0.2 to its stationary value. It is evident from the plot that an increase in the pulse rate leads to broadening of the pressure-flow characteristic. Both harmonic and anharmonic periodic pressure variations were considered (Fig. 6). The difference from the preceding plot consists in a change in the shape of the periodic pressure pulses. Figure 6a presents the reaction of the flow rate to a triangular pressure drop with a steep front slope (this shape of the pressure pulse is characteristic of the exit from an artery [27]), and Fig. 6b presents similar results for a pulse with a flattened front slope. The frequency and the parameters of

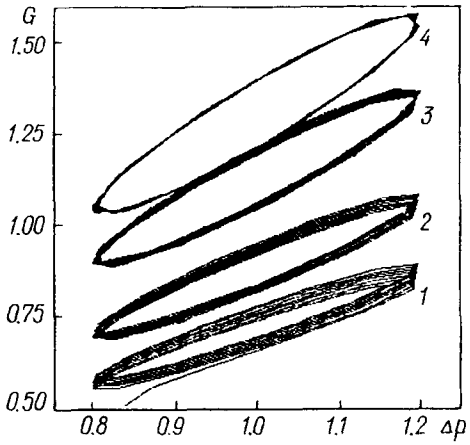

Fig. 7. Evolution of the pressure-flow characteristic with time (the numbers 1 to 4 denote pressure-flow characteristics corresponding to different time intervals): 1) < 5 sec; 2) 25–30; 3) 95–105; 4) 250–260 sec.

the model are the same as in the preceding plot. For the anharmonic change in the pressure drop, a triangular pulse shape with a total duration $\tau = 1/f$ was used. The steep and flattened front slopes correspond to a pressure increase to the maximum value in 0.25τ and 0.75τ, respectively. The harmonic variation of the pressure was given by Eq. (9).

We also analyzed the effect of an increase in the retardation time by two orders of magnitude for the smooth-muscle-tissue component in the Voigt–Kelvin model. The retardation times were taken to be as follows: θ_1 = 100 sec (elastin), θ_1 = 28.5 sec (collagen), and θ_3 = 2.5 sec (muscle tissue). Figure 7 presents the time evolution of the pressure-flow characteristic. The lower portion of the plot shows the pressure-flow characteristic during the first 5 sec, above which the characteristic for time periods of 25 to 30 sec and 95 to 105 sec is situated, and the region of stabilized flow (250–260 sec) is situated at the very top. The modulation depth is 20%, i.e., the pressure varies from 0.8 to its stationary value.

The mathematical simulation of the hydrodynamic processes in viscoelastic arterial vessels makes it possible to draw the following conclusions.

The hydraulic resistance of arteries can vary substantially depending on the time, the rate of cardiac contractions, and the shape of the pressure pulse at the vessel entrance. Long and short arteries show different reactions to pressure variations.

Use of rheological models of arterial-vessel walls makes it possible, in contrast to the approach based on use of Hooke's law [25], to obtain time dependences for the flow rate under non-steady-state conditions.

Use of the three- or multicomponent Voigt–Kelvin model makes it possible to account for the characteristic times of different physiological processes. The standard-body model as applied to the artery wall also makes it possible to account for the rate of variation of the load.

The actual behavior of arterial vessels can be described within the framework of the rheological models considered by changing the parameters of the models.

Under conditions of hyper- and hypothermia, when the process of temperature changes in the tissues of the organism lasts for several hours, numerical solution of this problem makes it possible to account for the temperature dependence of the rheological parameters of the tissues.

The moduli of elasticity and the relaxation times of the individual layers of the vessel wall serve as initial data for the calculation. The present investigation points to the necessity of experimental investigation of the rheological parameters of walls of blood vessels at various deformation rates.

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NOTATION

σ, stress; ε, relative deformation of the vessel wall; μ, viscosity of blood; η, viscosity of the vessel wall; ρ, density; ω, angular frequency of cardiac contractions; τ, oscillation period; c, vessel conductivity; E, modulus of elasticity of the vessel wall; f, frequency of cardiac contractions; q, volume blood flow rate; R, vessel radius; t, time. Subscripts: 0, unperturbed value; i, vessel number; in and out, values at the entrance to and exit from the vessel.

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