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BLOOD RHEOLOGICAL FACTOR IN THE PROBLEM OF GASEOUS EMBOLISM

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The main factors affecting growth of bubbles in blood upon decompression are discussed. Most attention is paid to the role of rheological effects. It is shown that viscoelasticity of blood can be neglected in description of evolution of gaseous microbubbles. Conditions for manifestation of nonlinear viscous properties of blood in this problem are discussed.

Introduction. Gaseous embolism of vessels is one of the particular problems of medicine. Thus, e.g., in neurosurgery just about 2% of serious aggravations are due to embolism in certain operations [1]. However, in cases when embolism does take place, its consequences for vital activity connected with disorder of the normal bloodstream can be catastrophic. Aside from surgery, the appearance of embolism is possible during a series of therapeutic procedures, upon decompression, etc.

The phenomenon being discussed is connected with gas bubbles appearing in blood for various reasons. When moving with the bloodstream and changing their dimensions, the bubbles can block the transport cross-section of a vessel in a certain place. Behind the occlusion, the bloodstream is terminated, which results in ischemia followed by necrosis of vessel walls. Consequences depend on the embolus localization in the organism and are especially dangerous when embolism takes place in coronary vessels and vessels that supply the brain with blood.

Multiple cases of occlusion of vessels by bubbles upon craniotomy, firearm wounds of the neck, operations on the bladder, certain gynecological procedures, donor blood transfusion, extracorporal blood circulation, and operations on the heart are described in the medical literature. The Caisson disease that appears during a fast rising to a surface after deep underwater diving, and embolism of vessels upon decompression due to depressurization of aircrafts at high altitudes, hold a special place in this problem. In both cases bubbles whose growth and motion lead finally to occlusion of vessels originate within the organism. Most likely, embolism observed upon deep hypothermism followed by elevation of temperature is of an analogous nature.

Generally, arterial and lung, or venous, embolisms are distinguished one from another. In the former case bubbles from the aorta enter the arterial network (Fig. 1) and the subsequent development of the process depends on the place of origination of occlusion. In the latter case bubbles, moving along the venous system enter the left ventricle via the hollow vein. If the gas amount is not big the blood with bubbles goes along the pulmonary artery to the lungs where evacuation of the gas phase takes place. Otherwise, irreversible consequences take place as a rule.

For deeper understanding of mechanisms of the processes taking place upon gaseous embolism it is of interest to model evolution of bubbles in blood with consideration for main specific features of the carrying liquid, particularly its rheological properties and hydrodynamics of the stream, and the character of variations in pressure. In what follows we consider dynamics of small bubbles in blood in large vessels upon decompression. The objective of the work was to analyze the main factors determining the character of growth of bubbles in blood from the ready nucleus, with the emphasis on the rheological factor.

Description of the Model and the Analysis of Factors. The pressure in vessels fluctuates periodically following heart pulsations. The frequency of the pulse wave $f_p \approx 1-2$ Hz [2]. The fundamental frequency f_b of small bubbles can be determined using the formula $f_b \approx (2\pi)^{-1}(3p_a/\rho_{liq}R_0^2)^{1/2}$ [3]. At $R_0 \sim 10\,\mu$m we obtain

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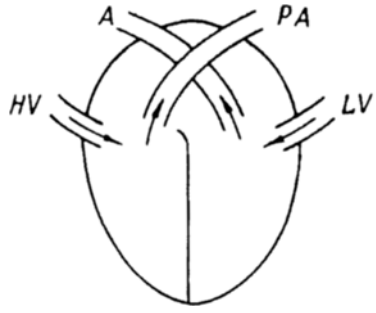


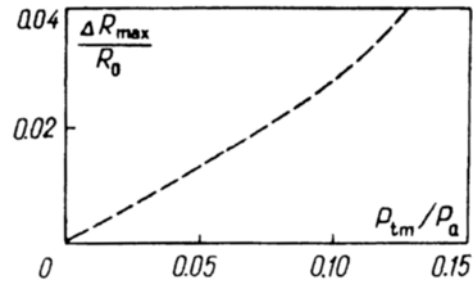


Fig. 1. General diagram of the bloodstream in the heart: A) aort; LV) lunn vein; HV) hollow vein; PA) pulmonary artery.

Fig. 2. Maximum relative deviation of the bubble radius in the pulse wave as a function of the relative amplitude of the transmural pressure.

$f_b \sim 10^5$ Hz. Inasmuch as $f_b >> f_p$, inertia of the volume of the liquid that adjoins bubbles can be neglected. Variations in the radius of the gas inclusion in the pulse wave in this case can be estimated from the quasistatic relationship [4]

$$(1 + p_{tm}/p_a)\, p_a + 2\sigma R^{-1} = (p_a + 2\sigma R_0)(R_0 R^{-1})^3 . \tag{1}$$

Figure 2 presents the corresponding curve for $\Delta R_{max}/R_0$ calculated using Eq. (1). As follows from the Figure, at the amplitude of the transmural pressure $p_{tm} \approx 0.15$ atm (in arteries in the norm $p_{tm} \approx 13.3$ kN/m^2 [5]) relative variations in the radius do not exceed 5%.

The sharp pressure drop upon decompression causes inflation of bubbles. Description of the arising flow requires the use of the rheological equation for blood for which we use Shul'man's equation [6]

$$\tau_{ij} = 2\,[A^{-1/m}\tau_0^{1/n} + \mu^{1/m}]^n A^{n/m-1} e_{ij}, \quad A = (2e_{ij}e_{ji})^{1/2}. \tag{2}$$

The generalized Rayleigh equation [7] in this case takes the form

$$\rho_{liq}(R\ddot{R} + 3/2\dot{R}^2) = p_g - p_{liq} - 2\sigma/R -$$

$$- 4\dot{R}R^{-1}\left\{12^{1/(2m)}\,|\dot{R}R^{-1}|^{-1/m}\tau_0^{1/n} + \mu^{1/m}\right\}^n \times 12^{(n-m)/(2m)}\,|\dot{R}R^{-1}|^{(n-m)/m}. \tag{3}$$

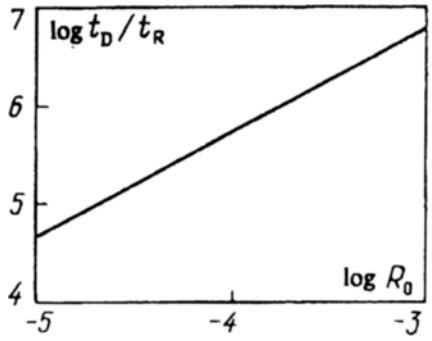
Applicability of Eq. (3) is restricted by the necessity to satisfy the inequality $R << d$, where d is the characteristic vessel diameter. Corresponding R values are determined by dimensions of main vessels in the human organism (aorta – $2 \cdot 10^{-2}$ m, large arteries – $3 \cdot 10^{-3}$ m, and large veins – $8 \cdot 10^{-3}$ m [8]).

Viscoelastic properties of blood dictate the necessity to create a pressure difference necessary to put the bubble in motion. This quantity is determined from the fluidity condition [9] $I_{2\tau} > 2\tau_0$, $I_{2\tau} = (2\tau_{ij}\tau_{ji})^{1/2}$ simultaneously with the dynamic boundary condition on the bubble surface [7], which leads to the relationship

$$\Delta p_0 = p_g - (p_{liq}(R) + 2\sigma R^{-1}) > \frac{2}{\sqrt{3}}\tau_0 .$$

Inasmuch as the limiting shear stress for blood τ_0 does not exceed 0.05 Pa [9], the viscoelasticity factor in the situation under consideration can be neglected.

The complete system of equations for bubble evolution includes also the diffusion equation for the gas dissolved in blood



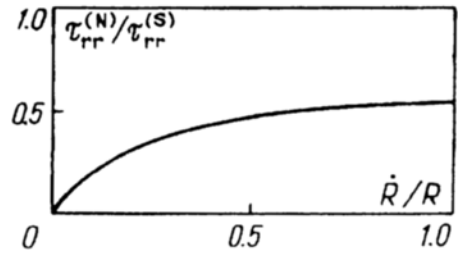


Fig. 3. Relationship between characteristic times t_D and t_R for a CO_2 bubble in normal human blood.

Fig. 4. Manifestation of the rheological blood nonlinearity as a function of the relative bubble growth rate. $\dot{R}/R$, sec^{-1}.

$$\frac{\partial C}{\partial t} + v_r \frac{\partial C}{\partial r} = D\left[r^{-2}\frac{\partial}{\partial r}\left(r^2\frac{\partial C}{\partial r}\right)\right], \tag{4}$$

equations of mass balance and state of the gas

$$\frac{d}{dt}\left(\frac{4}{3}\pi R^3\rho_g\right) = 4\pi R^2 j, \tag{5}$$

$$p_g = \rho_g BT, \tag{6}$$

and corresponding boundary conditions

$$r >> R, \quad C = C_0, \tag{7}$$

$$r = R(t), \quad j = (\dot{R} - v_R)C + D\frac{\partial C}{\partial r}, \tag{8}$$

$$C_R = \Gamma p_g.$$

The expression for the radial velocity component v_r in (4) and (8) follows from the continuality equation (kinematic integral) and has the form [3]

$$v_r = v_R r^{-2} R^2. \tag{9}$$

As is evident from (9), the radial blood flow that originates during the growth of the bubble is localized within the region adjacent immediately to the inclusion surface ($v_r \sim r^{-2}$).

In the problem under consideration two characteristic time scales can be introduced: the inertial time $t_R = R_0(\rho_{liq}/p_{liq})^{1/2}$ and the diffusion time $t_D = R_0^2/D$. Inasmuch as these times differ by orders of magnitude (Fig. 3), the process of bubble growth undergoes two stages. In the first, inertial, stage pulsations develop whose decay is determined for small particles mainly by rheological losses [10]. An analysis of Eq. (3) for $R(t)$ on this stage was carried out for $n = m = 1/3$, $\mu = 1.93\cdot10^{-3}$ Pa·sec, and $\tau_0 = 1.03\cdot10^{-3}$ Pa (Sundukov's data for human blood in the norm). Calculations have shown that nonlinear viscous properties of blood manifest themselves just in the vicinity of extremum points on the $R(t)$ curve, thus leading to a decrease in the amplitude of pulsations (Fig. 4). The Reynolds number $Re = t_R\Delta p(4\mu)^{-1}$ can serve as a criterion for manifestation of rheological effects upon evolution of spherical bubbles in blood. An analysis of the expression for Re simultaneously with the data in Fig.

4 leads to the conclusion that rheological properties of blood affect just dynamics of small ($R_0 \sim 10^{-5}$ m) bubbles and the effect increases with the hematocrit index.

The growth of bubbles away from the initial instant proceeds in the diffusion stage under the conditions $p_g = \text{const}$, $C_R = \Gamma p_g$. The self-similar solution has the form [3]

$$R/R_0 \approx \rho_g^{-1} (C_0 - C_s) \left(\frac{12D}{\pi R_0^2} t \right)^{1/2}. \tag{10}$$

Numerical estimations carried out according to (10) for a nitrogen bubble in blood at $\rho_g \approx 1$ kg/m^3, $C_0 \approx 0.015$ kg/m^3, $D \approx 1.5 \cdot 10^{-9}$ m^2/sec, $\Delta p = 5 \cdot 10^4$ Pa have shown that the consideration for just molecular diffusion mechanism in the problem under consideration does not make it possible to explain the fast development of bubbles in blood upon decompression from nuclei to the size of inclusions capable of causing embolism. The most probable mechanism of the growth acceleration is convective diffusion, whose role should be investigated in the subsequent analysis of the phenomenon within the framework of simultaneous consideration of nonstationary bloodstream hydrodynamics and non-Newtonian properties of blood during the relative motion of the bubble in the bloodstream.

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NOTATION

p_a, atmospheric pressure; ρ, blood density; R, bubble radius; σ, surface tension coefficient; τ_{ij}, deviator of the stress tensor; e_{ij}, tensor of deformation velocities; τ_0, fluidity limit; μ, viscosity; m, n, parameters of Shul'man's model; C, gas concentration in blood; D, diffusion coefficient; Γ, the Henry constant. Indices: 0, equilibrium state; g, gas; liq, blood; R, bubble surface.

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