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NORMAL WAVES ARISING WHEN BLOOD MOVES THROUGH AN ARTERY

A model is proposed of the pulse wave propagation through an artery is proposed. The artery is considered as a cylindrical shell surrounded by an elastic medium. The amplitude and shape of normal waves arising, when blood flows through the artery are determined. Two types of such waves are revealed: zero waves, whose amplitude does not change its sign over the arterial cross-section, and non-zero ones, for which such a change does take place. It is shown that the pulse wave is a wave packet formed by zero normal waves. The non-zero normal waves are found to be localized near the entrance section of the artery, by creating a transition zone whose size is about the arterial radius. The non-zero normal waves are shown to enhance the process of erythrocyte disaggregation in the transition zone.

K e y w o r d s: artery, normal waves, pulse wave.

1. Introduction

For many years, the propagation of slow pulse waves arising at the moment, when blood is ejected from the left ventricle of the heart into the aorta, and accompanying the subsequent process of blood motion through the vessels remains a challenging problem in the mechanics of blood circulation, hemodynamics [1]. The study of regularities in the formation, development, propagation, and transformation of those waves is important from the viewpoints of both physiology and the ability to diagnose the state of vascular channel. It is the specific features of the pulse wave behavior in arteries that are researched in this paper.

A pulse wave owes its existence to the elasticity of vascular walls, which are deformed as the wave passes by. Accordingly, a widely used calculation model for a vessel is a cylindrical shell with elastic walls, which is surrounded by an elastic medium and filled with liquid. When this model is applied to describe the blood motion in a vessel, it is generally assumed [2–5] that the pressure distribution over the vascular cross-section is constant. In this approximation, the wave front is flat, so that the pulse wave is a Young-type one [6], which propagates through the vessel without changing its shape. As a result, the wave problem about the blood motion in the vessel becomes one-dimensional. Below, we consider a three-dimensional variant of this problem.

One of the central issues in hemodynamics and vascular diagnosis is the blood pressure; more specifically, the mechanism of circulation that forms the pressure field in the artery. This field is a superposition of normal waves [6]. Accordingly, the properties of normal waves are responsible for that or another mechanism of blood circulation. Therefore, normal waves were chosen as an object to study in this paper. Our aim is to establish the type of normal waves arising, when blood moves through the artery, as well as to determine the forms of blood motion associated with that or another type of such waves.

2. Calculation Model of the Artery

Let blood be considered as an ideal fluid. Accordingly, the equation of its motion is the Euler equation [7]

$$\rho \frac{\partial^2 \mathbf{u}}{\partial t^2} = -\mathrm{grad}\, p, \tag{1}$$

where ρ is the density, $\mathbf{u}$ the displacement vector, t the time, and p the pressure. By its meaning, p is

the difference between the systolic and diastolic pressures.

The elastic properties of the walls of a cylindrical shell and an external elastic medium are assumed to be identical, which allows the artery to be considered as a cylindrical cavity in a continuous environment. Therefore, let us introduce a cylindrical coordinate frame, whose z-axis is directed along the cylinder axis, and r stands for the radial coordinate. The radius of the cylinder equals b, and the longitudinal and transverse components of the displacement vector $\mathbf{u}$ equal u_z and u_r, respectively. In terms of those components, the vector equation (1) looks like

$$\rho\frac{\partial^2 u_z}{\partial t^2} = -\frac{\partial p}{\partial z}, \tag{2}$$

$$\rho\frac{\partial^2 u_r}{\partial t^2} = -\frac{\partial p}{\partial r}. \tag{3}$$

An ideal fluid that simulates blood is considered to be compressible and is described by the rheological equation

$$p = -\rho\, c_0^2 \mathrm{div}\,\mathbf{u}, \tag{4}$$

where c_0 is the sound velocity in the fluid. Substituting Eq. (4) into Eq. (1), we obtain the following equation for the blood motion through the artery:

$$\frac{\partial^2 p}{\partial t^2} = c_0^2\,\mathrm{div}\ \mathrm{grad}\, p. \tag{5}$$

The elastic medium surrounding the artery is taken into account by introducing the boundary condition

$$p(r=b) = \alpha u_r(r=b), \tag{6}$$

where α is the elasticity coefficient of the medium.

In the literature [1, 8], the following values for the blood density, arterial radius, and sound velocity in blood are quoted: $\rho = 1.05\times10^3$ kg/m^3, $b \le 3\times10^2$ m, and $c_0 = 1540$ m/s. The coefficient α was experimentally determined in work [4]: $\alpha \approx 10^7$ N/m^3.

3. Series Expansion of Functions Describing the Blood Motion through the Artery

By definition, the function $p(t)$ is periodic, with the period T equal to the cardiac cycle duration. It allows the function $p(t)$ to be written in the form of a Fourier series,

$$p(t) = \sum_{n=-\infty}^{\infty} p_n(z,r)\exp(-i\omega_n t), \tag{7}$$

where

$$\omega_n = 2\pi n/T, \tag{8}$$

and $T \approx 1$ s. Substituting Eq. (7) into Eq. (5), we obtain

$$\mathrm{div}\,\mathrm{grad}\, p_n + k_n^2 p_n = 0, \tag{9}$$

where the notation

$$k_n = \omega_n/c_0 \tag{10}$$

was introduced.

In the expanded form, Eq. (9) looks like

$$\frac{\partial^2 p_n}{\partial z^2} + \frac{\partial^2 p_n}{\partial r^2} + \frac{1}{r}\frac{\partial p_n}{\partial r} + k_n^2 p_n = 0. \tag{11}$$

The solution of Eq. (11) reads [6]

$$p_n = \sum_{j=0}^{\infty} p_{nj}, \tag{12}$$

where p_{nj} are normal waves, which can be represented as the product of two functions,

$$p_{nj} = p_{0nj}\, R_{nj}(r)\exp(i\xi_{nj} z). \tag{13}$$

Here, the coefficient p_{0nj} is the amplitude of oscillations, which is determined from the boundary conditions at the cylinder ends, the function $R_{nj}(r)$ is the radial distribution of pressure, and the multiplier $\exp(i\xi_{nj}z)$ describes the wave character of the excitation. The quantity ξ_{nj} plays the role of a wave number.

Substituting Eq. (13) into Eq. (11), we obtain

$$\frac{\partial^2 R_{nj}}{\partial r^2} + \frac{1}{r}\frac{\partial R_{nj}}{\partial r} + \eta_{nj}^2 R_{nj} = 0, \tag{14}$$

where the notation

$$\eta_{nj}^2 = k_n^2 - \xi_{nj}^2. \tag{15}$$

was introduced. The subscript j is equal to the number of sign changes that the function $R_{nj}(r)$ has

within the interval $r \le b$. The normal wave with the subscript $j = 0$ does not change its sign and is called the zero wave. Let the normal waves with $j = 1, 2, ...$ (positive integers starting from 1) be referred to as non-zero ones. By introducing the notation p_{nq} for them, equality (12) can be rewritten in the form

$$p_n = p_{n0} + \sum_{q=1}^{\infty} p_{nq}. \tag{16}$$

4. Propagation of Non-Zero Normal Waves through the Artery

The solution of Eq. (14) is the Bessel function of the zeroth order [9]

$$R_{nq} = J_0(\eta_{nq} r). \tag{17}$$

Accordingly, for the quantities p_{nq}, we have the formula

$$p_{nq} = p_{0nq} J_0(\eta_{nq} r) \exp\left(iz\sqrt{k_n^2 - \eta_{nq}^2}\right). \tag{18}$$

Since the function u_r, similarly to p, is also periodic, we expand it into a Fourier series

$$u_r = \sum_{n=-\infty}^{\infty} u_{nr} \exp(-i\omega_n t). \tag{19}$$

Substituting equalities (18) and (19) into Eq. (3), we obtain

$$u_{nr} = \sum_{q=1}^{\infty} \frac{p_{0nq}}{\rho\,\omega_n^2} \frac{d}{dr} J_0(\eta_{nq} r) \exp\left(iz\sqrt{k_n^2 - \eta_{nq}^2}\right). \tag{20}$$

Now, using the boundary condition (6) in Eqs. (18)–(20) and taking into account that

$$\frac{d}{dr} J_0(\eta r) = -\eta J_1(\eta r), \tag{21}$$

where $J_1(\eta r)$ is the Bessel function of the first order, we obtain the dispersion equation

$$J_0(\eta_{nq} b) = -\frac{\alpha \eta_{nq}}{\rho\,\omega_n^2} J_1(\eta_{nq} b). \tag{22}$$

The real roots of this equation determine the values of the parameters η_{nq} for the corresponding non-zero normal waves.

As was mentioned above, blood is transported through the vessel in the form of pulse waves. Can blood be transported through the artery by means of non-zero normal waves? In other words, can non-zero waves be components of a pulse wave? As one can see from formula (18), this possibility is realized provided that $k_n^2 - \eta_{nq}^2 > 0$. In the opposite case where $k_n^2 - \eta_{nq}^2 < 0$, the normal wave transforms into an in-phase oscillation, whose amplitude decreases according to the exponential law: $p_{nq} \sim \sim \exp\left(-z\sqrt{\eta_{nq}^2 - k_n^2}\right)$. Such waves are called inhomogeneous [6], in contrast to homogeneous (propagating) waves. A wave becomes homogeneous, when ω_n exceeds a certain critical frequency Ω, which is determined from the condition $k_n^2 - \eta_{nq}^2 = 0$. In view of formula (10), this condition reads

$$\left(\frac{\Omega}{c_0}\right)^2 - \eta_{nq}^2 = 0. \tag{23}$$

As was mentioned above, the function $R_{nq}(r)$ is characterized by the change of its sign. Namely, there are alternating intervals along the radius r in which $R_{nq}(r)$ has different signs. The only parameter of this function has the dimensionality of length and contains the quantity η_{nq}^{-1}. Therefore, by the dimensionality reasons [10], we may assert that the length of those intervals equals η_{nq}^{-1} by the order of magnitude, which gives rise, in turn, to the inequality $\eta_{nq}^{-1} < b$. The latter, making allowance for relation (23), acquires the form

$$\Omega > \frac{c_0}{b}. \tag{24}$$

With the help of equality (8), the expression for Ω can be written as $\Omega = 2\pi n_\Omega / T$. Its substitution into condition (24) brings about the result

$$n_\Omega > \frac{c_0 T}{2\pi b}.$$

By applying the numerical estimates given above for c_0, T, and b, we obtain $n_\Omega > 8 \times 10^4$. Since the amplitudes of Fourier components decrease with the increase of their number n, it is obvious that the amplitudes with the numbers satisfying this inequality turn out negligibly small. This fact means that non-zero waves in the artery are inhomogeneous, i.e. they do not participate in the blood transport.

5. Propagation of Zero Normal Waves through the Artery

Besides real roots, Eq. (22) also has a root that is an imaginary number $\eta_{n0} = i\eta_n$. In this case, we have

the inequality $k_n^2 - \eta_{n0}^2 > 0$, which provides the homogeneity of a zero normal wave. As a result, Eq. (22) can be rewritten in the form

$$I_0(\eta_n b) = \frac{\alpha \eta_n}{\rho \omega_n^2} I_1(\eta_n b), \tag{25}$$

and expression (17) in the form

$$R_{n0} = I_0(\eta_n r), \tag{26}$$

where $I_0(\eta_n r)$ and $I_1(\eta_n r)$ are the modified Bessel functions of the zeroth and first orders, respectively. Accordingly, for the quantity p_{n0}, we have the expression

$$p_{n0} = p_{0n0} I_0(\eta_n r) \exp\left(iz\sqrt{k_n^2 + \eta_n^2}\right). \tag{27}$$

Let us recall that the zero wave is a wave for which there is no sign changes in the interval $r < b$. From whence, it follows that $\eta_n^{-1} > b$. Bearing this inequality in mind, let us expand the Bessel functions in expression (25) in a power series in $\eta_n b$ to the fourth-order terms:

$$1 - \frac{(\eta_n b)^2}{4} + \frac{(\eta_n b)^4}{64} = \frac{\alpha \eta_n^2 b}{2\rho \omega_n^2}\left[1 - \frac{(\eta_n b)^2}{8}\right]. \tag{28}$$

The solution of this equation, taking the numerical estimates made above into account, looks like

$$\eta_n = \frac{\omega_n}{c}\left[1 + \frac{1}{8}\left(\frac{\omega_n b}{c}\right)^2\right]^{1/2}, \tag{29}$$

where

$$c = \left(\frac{\alpha b}{2\rho}\right)^{1/2}. \tag{30}$$

As follows from formula (13), the propagation velocity of the zero normal wave equals

$$c_n = \omega_n \xi_n^{-1} = \omega_n \left(k_n^2 + \eta_n^2\right)^{-1/2}. \tag{31}$$

Substituting expressions (10) and (29) into this formula, we obtain

$$c_n = \left\{c_0^{-2} + c^{-2}\left[1 + \frac{1}{8}\left(\frac{\omega_n b}{c}\right)^2\right]\right\}^{-1/2}. \tag{32}$$

Now, using the numerical estimates given above, we can see that the term $(\omega_n b/c)^2/8 \gg 1$, if $n > 100$, so that we may approximately write that $c_n \approx c$ and take the propagation velocities of zero normal waves through the artery to be almost identical.

As one can see from a comparison with the one-dimensional variant of the problem [2–5], the mechanism of blood transport becomes different, if we deal with the three-dimensional geometry. In particular, in the one-dimensional geometry, blood is transported by a single Young wave. In the three-dimensional case, this role is performed by an infinite number of harmonic zero normal waves, so that the pulse wave is the sum of those waves, i.e. a wave packet. When moving through the vessel, the wave packet is not smeared, because the propagation velocities of the normal waves that compose this packet are almost equivalent to one another.

6. Normal Waves in the Transition Zone

Let us consider the mechanism of formation of a non-zero normal wave. Substituting expressions (18) and (27) into formula (16), we obtain

$$p_n = p_{0n0} I_0(\eta_n r) \exp\left(iz\sqrt{k_n^2 + \eta_n^2}\right) + \\ + \sum_{q=1}^{\infty} p_{0nq} J_0(\eta_{nq} r) \exp\left(iz\sqrt{k_n^2 - \eta_{nq}^2}\right). \tag{33}$$

Let the origin of the cylindrical coordinate frame be located at the center of the entrance arterial cross-section. Then the pressure over the entrance cross-section, p_n, is described by the formula

$$p_n = p_{0n0} I_0(\eta_n r) + \sum_{q=1}^{\infty} p_{0nq} J_0(\eta_{nq} r). \tag{34}$$

Furthermore, let the blood flow entering the artery form the external pressure

$$p_n' = f(r) \exp(-i\omega_n t) \tag{35}$$

at the entrance cross-section. Then the condition that the pressure is a continuous function across the entrance cross-section of the artery looks like

$$f(r) = p_{0n0} I_0(\eta_n r) + \sum_{q=1}^{\infty} p_{0nq} J_0(\eta_{nq} r). \tag{36}$$

Let us assume, as was done earlier, that $\eta_n b \ll 1$. In this case, we can take $I_0(\eta_n r) \approx 1$ and rewrite expression (36) in the form

$$f(r) = p_{0n0} + \sum_{q=1}^{\infty} p_{0nq} J_0(\eta_{nq} r). \tag{37}$$

Multiplying the both parts of this equality by $r\,dr$ and integrating the resulting expressions from 0 to b, we obtain

$$\int_0^b f(r)rdr = p_{0n0}\frac{b^2}{2} + \sum_{q=1}^{\infty} p_{0nq}\frac{b}{\eta_{nq}}J_1(\eta_{nq}r). \qquad (38)$$

Now, let us multiply both parts of equality (37) by $J_0(\eta_{nk}r)rdr$ and integrate the resulting expressions from 0 to b:

$$\int_0^b f(r)J_0(\eta_{nk}r)rdr = p_{0n0}\int_0^b J_0(\eta_{nk}r)rdr + \\ + \sum_{q=1}^{\infty} p_{0nq}\int_0^b J_0(\eta_{nq}r)J_0(\eta_{nk}r)rdr. \qquad (39)$$

Using the relation [9]

$$\int_0^b J_0(\eta_{nq}r)J_0(\eta_{nk}r)rdr = 0 \quad (q \neq k),$$

let us rewrite equality (39) in the form

$$\int_0^b f(r)J_0(\eta_{nk}r)rdr = p_{0n0}\frac{b}{\eta_{nk}}J_1(\eta_{nk}b) + \\ + p_{0nk}\int_0^b J_0^2(\eta_{nk}r)rdr. \qquad (40)$$

We introduce the notation

$$F(b) = \frac{2}{b^2}\int_0^b f(r)rdr,$$

$$G_{nk}(b) = \int_0^b f(r)J_0(\eta_{nk}r)rdr, \qquad (41)$$

$$H_{nk}(b) = \int_0^b J_0^2(\eta_{nk}r)rdr.$$

In the zeroth approximation in the small parameter $b\eta_{nk}^{-1}$, formulas (38) and (40) read, respectively,

$$p_{0n0} = F(b), \qquad (42)$$

$$p_{0nk} = G_{nk}(b)H_{nk}^{-1}(b), \qquad (43)$$

Substituting relations (42) and (43) into formula (33) and taking the approximate equality $I_0(\eta_n r) \approx 1$ into account, we obtain the following expression for the pressure p_n:

$$p_n = F(b)\exp\left(iz\sqrt{k_n^2+\eta_n^2}\right) + \\ + \sum_{q=1}^{\infty} G_{nq}(b)H_{nq}^{-1}(b)J_0(\eta_{nq}r)\exp\left(iz\sqrt{k_n^2-\eta_{nq}^2}\right). \qquad (44)$$

This formula demonstrates that, far from the entrance arterial cross-section (at $z \to \infty$), the pressure is determined by the first term and harmonically oscillates. Those oscillations correspond to the zero wave propagating along the artery. In addition, near the entrance cross-section, there arise non-zero normal waves, which exponentially decay with the increasing distance. As a result, there arises a pressure near the entrance cross-section, which is additional to the pressure generated by the zero wave. This additional pressure is determined by the second term on the right-hand side of expression (44). Let us call the region in the artery, where the zero and non-zero waves are superimposed, a transition zone.

Let us determine the form of the function $f(r)$ describing the pressure distribution over the entrance arterial cross-section. Let the entrance arterial cross-section BQ lie in the plane $ABQD$, in which the vessel diameter changes (Fig. 1). As was mentioned above, only zero normal waves can propagate through the vessels. Recall also that those waves compose a pulse wave and – in the approximation $I_0(\eta r) \approx 1$ valid for vessels – the pressure is constant in this cross-section of a pulse wave.

So, let a pulse wave fall onto the plane $ABQD$. The pressure in the incident wave will be denoted as p'. The plot of the dependence $p'(r)$ is shown in Fig. 1 and marked by number 1. For this plot, as well as for other plots in Fig. 1, the pressure axis is directed in parallel to the z-axis. As was mentioned above, the pressure p' remains constant in this case for all points in the cross-section, and its value is determined by the equality $p' = MN$ (Fig. 1).

When the pulse wave reaches the cross-section $ABQD$, there arises the reflected and transmitted waves. The pressure in the reflected wave will be denoted as p''. The pressure in the transmitted wave was earlier denoted as f. The total pressure $p^* = p' + p''$ acts on cross-section $ABQD$ from the left. The dependence $p^*(r)$ is shown in Fig. 1 by

plot *2*. For sections AB and QD, i.e. for $r > b$, there is no transmitted wave. Accordingly, $p' = p''$ for them, so that $p^* = 2p'$ and $EA = E_1D = 2MN$.

In section BQ, i.e. at $r < b$, owing to the presence of a transmitted wave, the pressure p'' has to gradually decrease with the decreasing r and to reach a minimum value at $r = 0$. But the pressure continuity has to be held in this section, i.e. the equality $p'+p'' = f$ has to be satisfied. Accordingly, the dependence $f(r)$ possesses the following character (Fig. 1, plot *3*): $f(r) = 0$ at $r = b$; as the radius r decreases, the magnitude of f has to increase and to reach a maximum at $r = 0$. The simplest smooth function that meets those requirements is the parabola

$$f(r) = f_0 \left[1 - \left(\frac{r}{b}\right)^2\right]. \tag{45}$$

Let the diastolic pressure be the reference point. Accordingly, the average value of function (45) over the arterial cross-section,

$$\frac{1}{\pi b^2} \int\limits_0^b 2\pi f(r) r dr = \frac{f_0}{2},$$

is identified with the pulse pressure. The latter is known [1] to be the difference between the systolic and diastolic pressures. Assuming that their values equal 120 and 80 mm Hg, respectively (the norm for an adult), we obtain $f_0 \approx 10^4$ Pa.

Using this result and formula (44), as well as the numerical values given abovefor other parameters, we calculated the pressure acting in the transition zone. The corresponding results are depicted in Fig. 2. As one can see from this figure, the size of the transition zone is equal to the arterial radius by the order of magnitude. The calculations were performed for $n = 1$. It is evident that the size of the transition zone will decrease with the growing n (the increasing frequency).

7. Influence of Non-Zero Normal Waves on Erythrocyte Disaggregation

The blood motion through a vessel is known to be accompanied by the aggregation and disaggregation of red blood cells [1]. When describing the motion of an erythrocyte, let us use one of the hydrodynamic models and approximately consider the blood plasma

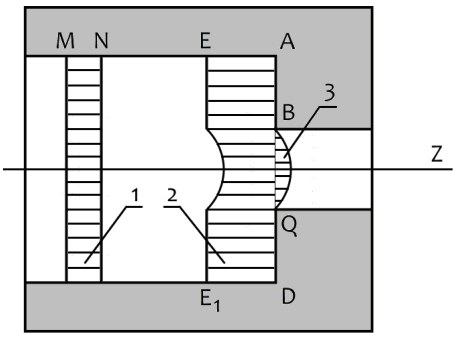

Fig. 1. Formation of a pressure distribution over the input arterial cross-section

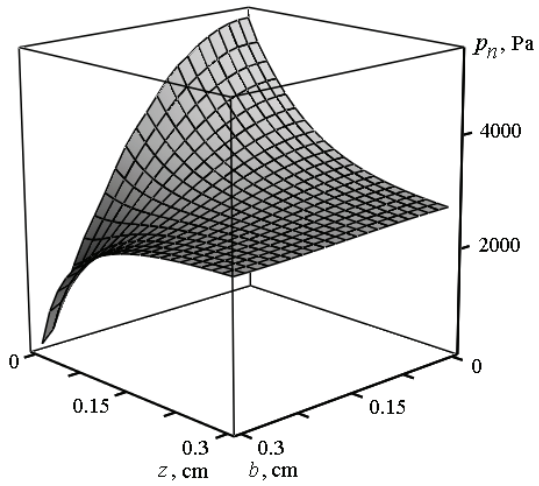

Fig. 2. Pressure distribution in the arterial transition zone

as an ideal fluid [7]. The erythrocyte will be considered as a non-deformable particle, and we will neglect changes in the plasma flow velocity at distances of the particle size order.

At the propagation of normal waves in the artery, the erythrocyte oscillates together with the blood plasma oscillations. The corresponding velocity of erythrocyte displacement is lower than the velocity of plasma motion. In the framework of this model, the equation of motion for the erythrocyte looks like [7]

$$\rho_{\rm e} \frac{d\mathbf{w}}{dt} = \rho \frac{d\mathbf{v}}{dt} - \frac{1}{\beta} \mathbf{M} \left(\frac{d\mathbf{w}}{dt} - \frac{d\mathbf{v}}{dt}\right), \tag{46}$$

where $\rho_{\rm e}$, $\mathbf{w}$, β, and $\mathbf{M}$ are the density, velocity, volume, and the adjoined mass tensor of the erythrocyte, respectively.

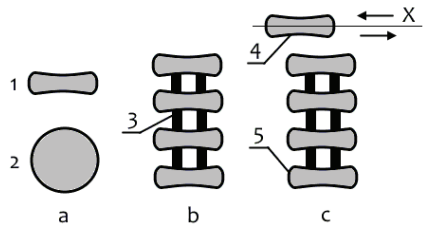

Fig. 3. Schematic diagram of the erythrocyte aggregation: (*a*) approximate form of erythrocyte ((*1*) side view, (*2*) top view), (*b*) erythrocyte chain ((*3*) fibrinogen bridge), (*c*) erythrocyte attachment to the chain

Substituting expression (1) into formula (46), we obtain

$$\left(\rho_{\mathrm{e}} + \frac{1}{\beta}\,\mathbf{M}\right)\frac{d\mathbf{w}}{dt} = -\left(1 + \frac{1}{\beta\rho}\,\mathbf{M}\right)\nabla p. \tag{47}$$

From this equation, one can see that the amplitude of oscillation-like motion of the erythrocyte grows with the oscillation amplitude of the quantity $(-\nabla p)$. How does the oscillation amplitude affect the ability of erythrocytes to aggregate?

The shape of a normal human erythrocyte is a biconcave discoid ("discocyte") [11]. Let us approximately take the erythrocyte shape to be a round plate (Fig. 3, *a*). In aggregates, erythrocytes form chains ("coin columns") (Fig. 3, *b*), which are structural units of larger aggregates. Erythrocytes are held in the chains be means of "bridges" consisting of fibrinogen molecules.

Let us consider an elementary event of the erythrocyte attachment to a chain. Erythrocyte *4* (see Fig. 3, *c*), being located at such a distance from chain *5* that is sufficient to form a bridge, oscillates in a plane oriented perpendicularly to the chain axis. The displacement X of erythrocyte *4* with respect to the chain axis is determined by the formula

$$X = A\sin\omega t, \tag{48}$$

where A is the amplitude of erythrocyte oscillations.

On the outer membrane of the erythrocyte, there are areas with a specific structure that can attach fibrinogen molecules. A junction between two erythrocytes emerges, when such sections of both erythrocytes become located opposite to each other. Let the time required to form a bridge equal τ. We denote the size of the described area by h. The formation of a bridge becomes impossible, when one of those areas shifts with respect to the other by h.

The value of X will be reckoned from the center of the area, so that the left boundary of the area corresponds to the point with the coordinate $X_1 = -h/2$. When the area shifts as a whole, this point moves to the position $X_2 = h/2$. Let one of the erythrocytes oscillates with respect to the other, and the described boundary is at the point X_1 at the time moment $t_1 = -\Delta t$ and at the point X_2 at the time moment $t_2 = \Delta t$. According to formula (48), we have

$$h = X_2 - X_1 = 2A\sin\omega\Delta t. \tag{49}$$

Assuming $h \ll 2A$, we obtain

$$2\Delta t = \frac{h}{A\omega}. \tag{50}$$

A bridge between those two sections can emerge for a time period, when the condition

$$t_2 - t_1 \geq \tau \tag{51}$$

is satisfied, i.e. until the both areas can be considered as located opposite to each other.

Substituting equality (50) into expression (51), we obtain

$$A \leq \frac{h}{\omega\tau}. \tag{52}$$

Hence, there is a certain critical value for the erythrocyte oscillation amplitude, $A_c = h/(\omega\tau)$. If this value is exceeded, erythrocytes lose their ability to aggregate.

The speculations presented above for the case $n = 1$ lead to the following estimates: $|-\nabla p_n| \sim p_n/b$, if $z < b$, and $|-\nabla p_n| \sim p_n/(cT)$, if $z > b$. Using the numerical values of the parameters, which were given above, one can see that the former estimate is substantially larger than the latter one. This means that the oscillatory motion of an erythrocyte in the transition zone is much more intense than outside this zone. Accordingly, the process of erythrocyte disaggregation in the transition zone is more intense due to the presence of non-zero normal oscillations there.

8. Conclusions

The calculations performed in this work allowed the following conclusions to be drawn about the mechanism of blood circulation in the artery.

• The blood motion through the artery invokes the appearance of normal waves. These are harmonic

waves that are characterized by the frequencies $\omega_n = 2\pi n/T$, where T is the duration of a cardiac cycle, and $n = 0, 1, 2, ...$ Two types of such waves are realized: zero waves, for which the amplitude of oscillations does not change its sign over the arterial cross-section, and non-zero ones, for which this change does take place.

• Zero normal waves are waves that propagate along the vessel, thereby providing the blood transport. The hemodynamic idea about the pulse wave as a Young-type wave is approximate. Actually, the pulse wave is a wave packet consisting of zero normal waves. The propagation velocities of those waves are almost identical, which prevents the wave packet from the smearing.

• Non-zero normal waves do not propagate through the vessel and, accordingly, do not participate in the blood transport. These waves are localized near the arterial entrance and form a transition zone, where the blood motion looks like in-phase oscillations. The amplitudes of the latter decrease exponentially with the increasing distance from the arterial entrance. The characteristic length of the amplitude decay does not exceed the arterial radius. The size of the transition zone has the same order as the arterial radius.

• Non-zero normal waves in the transition zone enhance the disaggregation of erythrocytes.

In our opinion, those conclusions could be used in hemodynamics and vascular diagnostics.

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НОРМАЛЬНІ ХВИЛІ,
ЩО ВИНИКАЮТЬ ПРИ РУСІ КРОВІ В АРТЕРІЇ

Р е з ю м е

Запропоновано модель поширення пульсових хвиль в артерії, що розглядається як циліндрична оболонка, оточена пружним середовищем. Визначена амплітуда і форма нормальних хвиль, що виникають при русі крові в артерії. Встановлено два типа таких хвиль: нульові, для яких амплітуда не змінює знак по перерізу артерії, і ненульові, для яких така зміна спостерігається. Показано, що пульсова хвиля являє собою хвильовий пакет, утворений нульовими нормальними хвилями. Встановлено, що ненульові нормальні хвилі локалізовані поблизу вхідного перерізу артерії, утворюючи перехідну зону з розміром порядку радіуса артерії. Показано, що ненульові нормальні хвилі підсилюють процес дезагрегації еритроцитів у перехідній зоні.