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The Circulatory System in an Electromagnetic Field

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ABSTRACT

The article deals with the interaction of two electromagnetic fields: the intrinsic electromagnetic field of the elements of circulatory system and the external electromagnetic field of environment. A model of the circulatory system is proposed that allows for a systematic assessment of the impact of electromagnetic fields on the cardiovascular system. The model is based on the biophysical and bioelectrical properties of the elements of the cardiovascular system and the central nervous system. The article considers issues related to the behavior of the vessels of the arterial part of the vascular bed: the capillary network, arterioles and large arteries in an electromagnetic field. The dynamics of myocardial behavior in two phases is clearly illustrated using a two-circuit electrical circuit. The change in the dynamics of the state of an elementary section of the vascular bed over time is estimated using a system of equations based on Hooke's law. The possible mechanism of human behavioral character in unfavorable environmental conditions is analyzed based on the principle of adequate design, which is presented in the diagram of the step-by-step impact of the external environment and its influence on the behavior of the cardiovascular system depending on the intensity of the impact.

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1. INTRODUCTION

Since the second half of the 20th century, interest in systems built on biological principles has arisen and is continuously growing [1, 2]. A characteristic feature of these systems is their continuous adaptability to changing internal and external conditions of existence and continuous improvement, taking into account past experience [3]. The task of such a science as biomechanics, according to Academician Berg, is to use the results of the study of such systems in order to improve existing or create new technical means.

From this point of view, it is interesting to study the principles of the functioning of the circulatory system, as it is very complex and multi-circuit [4]. The study of such systems requires a consistent analysis of the behavior of all its components in order to identify the properties and structure of the object, i.e. managed part, and properties, and structure of the control system. Under the control object we will understand the hemodynamic chain, i.e. a chain consisting of the heart and blood vessels, and under the control system - the corresponding parts of the nervous system.

About myogenic mechanisms of blood flow stabilization

It is well known that the purpose of blood circulation is to ensure the vital activity of the organism as a whole [5, 6, 7]. This goal is achieved by timely delivery of the required amount of nutrients to consumer organs and removal of toxins. It is essential that this occurs with the participation of a limited amount of blood, the mass of the executive organs (i.e., the heart and blood vessels) and with a wide range of changes (caused, for example, by the energy state of the environment around us) in blood flow and pressure, set by different levels of body activity. For example, the productivity of the heart increases with maximum physical

work up to 25,000 ml / min, while at a calm state it is 5800 ml / min. Hence the need for the existence of certain means of providing blood circulation.

Free radicals

Free radicals are reactive particles that have unpaired electrons in their outer energy levels. These particles, due to their aggressiveness caused by the loss of stability, and, of course, tending to restore the state of stability, are able to attack and destroy the substances of all biochemical blood components (proteins, enzymes, lipids, carbohydrates, amino acids), thereby disrupting important biological processes in the body.

Oxidation damage caused by free radical attack leads to premature aging, age spots on the cornea and other unpleasant phenomena. Back in the fifties of the last century, many gerontologists believed that free-radical compounds continuously accumulate in the cells of the body. More precisely, free radicals are continuously formed not only as a result of bad ecology, stress, smoking, poor nutrition, but also as a result of electromagnetic and radiation radiation. Evolutionarily, the body has developed a whole system of fighting free radicals - antioxidant protection. But how much it protects the body from the effects of electromagnetic fields and radiation is the task of serious research in the near future [8, 9].

Grouping of erythrocytes in a stationary electromagnetic field

In addition to the occurrence of free radicals, the effect of immobile EMF on the blood leads to such a phenomenon as the formation of stable groups of erythrocytes [10]. This disturbs the "normal" movement of blood, that is, the movement of red blood cells and blood plasma occurs in the form of disordered rapid fluctuations against the background of a relatively slow natural blood flow. As a result, a number of undesirable consequences are provoked, such as a violation of the integrity of the concentration of erythrocytes in the bloodstream, unpredictable hemodynamic regimes, in particular, a violation of tissue metabolism, etc.

2. MATERIALS AND METHODS

Effect of EMF on the vascular bed of the circulatory system

The next circle of problems - "vessels in an electromagnetic field" is associated with mechanochemical and mechanoelectric phenomena: the conversion of chemical energy into mechanical energy, for example, supplying muscles with oxygen, active transport - the diffusion mechanism, piezoelectric and electrokinetic effects - the mechanisms of microcirculation of plastic substances and oxygen in the capillary systems of the body (musculoskeletal system, capillary network) [11, 12].

Effect of EMF on capillary structures

The function of the capillary vessels is to provide the necessary for this activity of the body supply of its various organs with nutrients brought with the blood.

Among the mechanisms of exchange, filtration is distinguished - absorption and diffusion.

In filtration-absorption, the difference between the hydrostatic pressure on both sides of the capillary determines filtration, and the colloid osmotic pressure determines absorption. Each level of the current activity of one or another organ corresponds to a certain equilibrium value of these forces. Thus, the task of all physiological regulatory systems associated with transcapillary metabolism is to maintain this balance at a level corresponding to a given state of the organism. For the cardiovascular system, this task is reduced to maintaining the hydrostatic pressure required to ensure the filtration-absorption process [13].

Ensuring the proper value of the difference between capillary and intercellular pressure (p_c - p_i) is mainly achieved by changing the value of capillary pressure p_c. The fact is that the intercellular pressure p_i in most tissues is not subject to significant fluctuations, and its value is generally slightly lower than atmospheric pressure. The hydrostatic pressure in the exchange capillaries is on average 15 mm Hg. Art. and can be easily changed. Thus, the main hemodynamic parameter that allows you to regulate the exchange in the capillaries, which occurs due to the filtration-absorption mechanism, is the intracapillary pressure.

The same can be said about the control of the mechanism of diffusion of substances through the capillary wall. In accordance with the Fick equation, which describes the diffusion of uncharged soluble particles, the diffusion rate or metabolic rate depends on the concentration gradient (1):

$$\frac{dn}{dt} = D * A\left(\frac{dc}{dx}\right), \quad (1)$$

where n is the amount of diffusing substance; t is time; D is the diffusion coefficient; A is the effective diffusion area; c is the concentration of the diffusing substance; x is the distance.

The concentration of substances transported by the blood gradually decreases as it flows along the capillary. Therefore, depending on the rate of blood flow through the capillary, the concentration of one or another substance in a certain section of the capillary may be greater or less. Due to the structural features of

the capillary wall, the hydraulic resistance of the capillary is a constant value. Therefore, the only way in which the hemodynamic chain can influence the intensity of diffusion metabolic processes is the pressure gradient between the resistive and capacitive vessels.

The value of intracapillary pressure, which determines the processes of filtration - absorption, can be calculated by equation (2):

$$P_{\text{к}} = \frac{P_{\text{a}}\left(\frac{R_{\text{v}}}{R_{\text{a}}}\right)+P_{\text{v}}}{1+\frac{R_{\text{v}}}{R_{\text{a}}}}, \quad (2)$$

where P_{a} is the pressure in the section of resistive vessels; P_{v} is the pressure in the department of capacitive vessels; R_{a} is the resistance of resistive vessels; R_{v} is the resistance of capacitive vessels.

It can be seen from this formula that the intracapillary pressure is ultimately also determined by the pressure gradient at the ends of the capillary bed.

The structure of the capillary bed is a dense network of tubes of small diameter, the total surface area of which is large compared to their geometric dimensions and volume.

Volumetric blood flow through the capillary section of the hemodynamic chain is the same as in any other area, due to the continuity of the blood flow.

Due to the very small geometric dimensions of capillary vessels, the blood flow in them is estimated from the standpoint of microcirculation. First of all, this means that the blood is considered as a suspension, taking into account the solid particles that are in suspension in it - the blood cells. Among them, erythrocytes stand out primarily - red blood cells, the surface of which resembles a torus, with an inner ring tightened by a concave spherical surface. One of the main functions of red blood cells is to deliver oxygen to body tissues. The diameter of the erythrocyte is larger than the lumen of the capillary, but nevertheless, the erythrocytes easily pass through the capillary bed.

Before entering the capillary, erythrocytes are oriented so that the axis of the torus forming their surface coincides with the longitudinal axis of the blood vessel. Upon entering the capillary, they are easily deformed under the action of hydrostatic pressure forces and subsequently move through the vessel. In this case, columns of plasma, the liquid phase of blood, are trapped between the capillaries. Thus, erythrocytes move in the capillaries like a piston, while participating in the process of tissue metabolism. The exchange process proceeds differently, depending on the structure of the capillary, which in turn is determined by the functions of this organ.

Thus, the regulation of vital metabolic processes in the capillary bed can be achieved by purposefully changing the blood flow or, as a result, by pressure drop in the sections of the vascular bed adjacent to the capillaries, that is, resistive and capacitive vessels.

Such a sufficiently detailed consideration of the mechanisms of transcapillary exchange and the very structure of the capillary bed is necessary to identify the possible mechanism of the effect of EMF on this section of the vascular bed.

In medical practice, such a phenomenon as electrophoresis, known in engineering as electroosmosis, is widely used - the flow of a polar liquid through a porous dielectric under the influence of an electromagnetic field created by an external energy source. The use of this phenomenon in the practice of physiotherapy treatment is based on three components: a capillary network in soft tissues or in the bone-supporting apparatus, an external electromagnetic field and any polar liquid, which can be the appropriate medicines or blood.

Consider a simplified verbal model of diffusion as a physical and chemical process. We assume that the diffusion rate for specific physiological conditions is constant. Specific physiological conditions are "set" by the corresponding activity of the organism.

We accept the length of a single capillary as finite, the supply of oxygen in the erythrocyte is also finite, and it must be “given away” evenly along the entire length of the capillary. This requires a "strictly" defined speed of erythrocyte movement, which in the body in a normal situation is provided by the circulatory system.

Thus, in the first consideration of the mechanisms that ensure homeostasis, two are clearly distinguished: biomechanical, responsible for ensuring the appropriate “optimal” speed of erythrocyte movement through the capillary (capillary network) and the second, biochemical. We note one more fundamental circumstance: capillary blood flow (unlike resistive blood flow) is not under the direct control of the central nervous system, but is a consequence of the state of general hemodynamics. Thus, the effect of EMF on the capillary network, as an unauthorized intervention in systemic hemodynamics, can lead to unpredictable consequences.

The appendix discusses the mechanism of electrokinetic phenomena, which has found its place in medical practice, like electrophoresis.

Effect of EMF on arterioles (resistive vessels)

In contrast to the vessels of the capillary network, arterioles, as it is commonly defined in terms of biomechanics, have active properties, i.e. the ability to "actively" respond to changes in intravascular pressure, to respond to signals from the central nervous system, to changes in the external environment. We will be mainly interested in the latter, that is, the response of the arteriolar vascular system to electromagnetic fields. For all the above cases of exposure, arterioles react by changing the cross-sectional area, i.e. throughput.

Let us consider a model of a single vessel of the skeletal muscle blood supply system [14, 15]. Let us represent the structure of the vascular bed of skeletal muscles in the form of a graph (Figure 1).

The initial top of the graph depicts the main arteries that supply blood to the muscle, called the vessels of the coarse network. The middle link of the graph is formed by branches corresponding to the vessels of the fine network that distribute the blood flow to individual muscle fibers. Finally, the terminal branches of the graph depict the smallest arterial vessels - the vessels of the thinnest network.

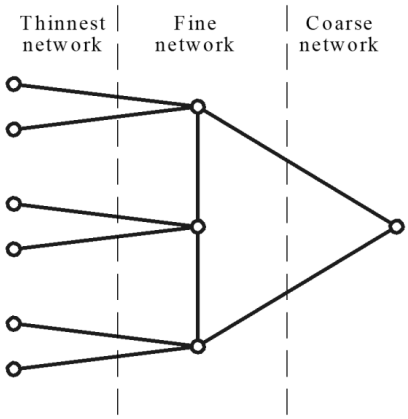

Figure 1. Graph depicting the structure of the vascular bed of skeletal muscles

An increase in the activity of the skeletal muscle primarily affects the vessels of the thinnest network, causing their expansion or narrowing. Then, thanks to the electrical connection that exists between the cells of the walls of arterial vessels, this effect is gradually transmitted to the vessels of the fine, and then the coarse networks. This is how the mechanism of working hyperemia works, that is, an adequate supply of blood to working organs, in our example, skeletal muscles, to the activity of the body. However, the vessels of this group can change their lumen, as noted, and under the influence of external factors, which in biomechanics include electric and magnetic fields. These fields can affect a group of certain cells that make up the structure of the material of the vascular wall. These cells are called pacemakers. They play an essential role in ensuring the tone of the arterioles, i.e. are responsible for the coefficient of its volumetric elasticity (an analogue in technology is the Hooke coefficient for thin-walled vessels, for example, fuel tanks of ballistic missiles). The impact of external fields can change the frequency of electrical impulses generated by pacemakers, therefore, affect the coefficient of volume elasticity, and, ultimately, the throughput of the arteriole.

Using the theory of an elastic reservoir, the model of a single vessel can be represented in the following form (Figure 2).

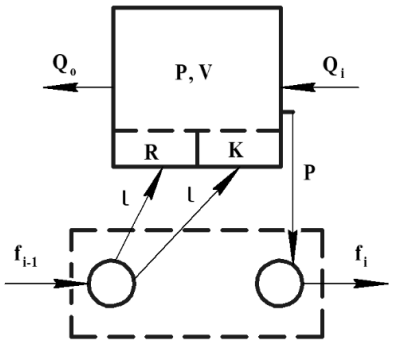

Figure 2. Model of a single vessel

The hemodynamics of the simulation object is described by three equations that relate the volume *V* and blood pressure in the vessel *p* with the flow at the inlet *Q*i and the flow at the outlet *Q*o from the vessel.

$$p = K(V - V^*)\,; \tag{3}$$

$$V = V(0) + \int_0^t (Q_i - Q_o)dt\,; \tag{4}$$

$$Q_o R + L\frac{dQ_o}{dt} = \Delta p\,, \tag{5}$$

where *K* is the volumetric elasticity coefficient of the vessel; *V** is the unstressed volume of the arteriole; *R* is hydraulic resistance; *L* is the inertia of the blood flow.

The coefficient of volume elasticity *K* and the resistance of the arteriole *R* depend on the value of its inner radius *r*:

$$K = \frac{E\Delta}{r^3 2\pi b}\,; \tag{6}$$

$$R = \frac{8b\mu}{\pi r^4}\,, \tag{7}$$

where *b* is the length of the vessel; μ is the blood viscosity; *E* is the Young's modulus of the vessel wall; Δ is the vessel wall thickness.

The frequency of electrical impulses generated by the arteriole pacemaker under the influence of external factors changes, which leads to a change in its diameter, i.e. bandwidth [16]. In the above model, it is assumed that the frequency of impulses generated by arteriole pacemakers depends only on mechanical factors - pressure, etc. The equation describing this relationship was proposed in the middle of the last century, we present it only as an illustration of the response of an arteriole to an atypical perturbation:

$$a_1\frac{df_i}{dt} + a_2 f_i = b_1\frac{dp}{dt} + b_2(p - p_0). \tag{8}$$

And the influence of signals coming from smaller arterioles is taken into account by introducing the dependence of the arteriole geometry on the frequency of these impulses f_{i-1}:

$$T_1\frac{dl}{dt} + T_2(l - l_0) = \frac{a}{f_{i-1}}\,. \tag{9}$$

Note that the model of a single arteriole (Figure 2) was used in a number of experiments to study transient processes in the circulatory system and showed good adequacy to the original [17].

The influence of a constant magnetic field on the biomechanical properties of large arteries

There are practically no serious studies on the effect of EMF on the biomechanical properties of large arteries. Most likely, this is due to the fact that large vessels are poorly innervated, in other words, their "tone" (volumetric elasticity coefficient) is purely biomechanical, i.e. is not controlled by the central nervous system, but depends on the state of the cellular and tissue elements of the vascular wall. Changes in the biomechanical properties of such vessels under the influence of EMF are due to micro- and ultrastructural changes in these elements. It has been experimentally established that even a slight magnetic induction (100–200 mT) causes sharp changes in the state of intracellular elements, and this effect is observed for a long time after the termination of the EMF [18].

3. RESULTS AND DISCUSSIONS

Staged development of a model representation of the cardiovascular system

Simulation (analogue) model of the hemodynamic chain. The concept of an elastic chamber.

The presentation of the circulatory system as a series connection of elastic reservoirs is based on two factors: when the heart contracts, it forces blood out of its cavities, the pressure in the large arteries begins to increase and, due to the elastic properties of their walls, they begin to stretch. Thus, not all the blood displaced by the heart rushes to the periphery, part of it remains in the large arteries. After the closure of the aortic valves of the heart, i.e. when the heart has stopped its active phase, the blood from the large arteries under the action of elastic deformation forces begins to be displaced to the peripheral vessels [19].

Let us trace the process, which, as noted, is called diastolic – the process of emptying the aortic part of the vascular bed (Figure 3).

On Figure 3 shows the aortic part of the vascular system as an elastic chamber.

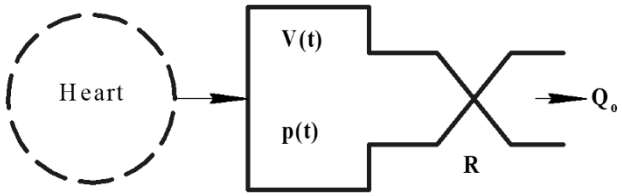

Figure 3. The aortic part of the vascular system, as an elastic chamber

This process is characterized by the fact that the heart valve is closed and the blood is forced out to the periphery only under the action of the elastic properties of the vascular bed.
Let us denote the volume of the selected area of the vascular system (Figure 3) at the end of the active phase of the heart (at the end of the systolic phase) through V_{KS}.
Then the decrease in the volume of this section of the vascular system in the diastolic phase due to the work of elastic deformation forces is determined by the following expression:

$$V(t) = V_{KS} - \frac{p(t)}{K}, \quad (10)$$

where *V(t)* is the section volume in the diastolic phase; V_{KS} is the end-systolic volume; *K* is some average volumetric elasticity coefficient of the aortic part of the vascular bed.
Formally, the coefficient *K*, in accordance with the theory of elasticity, is a constant proportional to Young's modulus *E* of the material of the vessel wall and depends on the initial thickness of the vessel wall δ and its initial volume V_0:

$$K = E * \delta / V_0 . \quad (11)$$

In equation (3), the volume elasticity coefficient appears as the reciprocal of the proportionality coefficient between pressure and volume. It is assumed that the volume of the aortic region varies linearly depending on the pressure p.
On Figure 4 *R* is the peripheral vascular resistance equal to

$$R = \frac{8l\eta}{\pi R^4}, \quad (12)$$

where *l* and *R* are the length and radius of some peripheral vessel, ŋ is the blood viscosity.

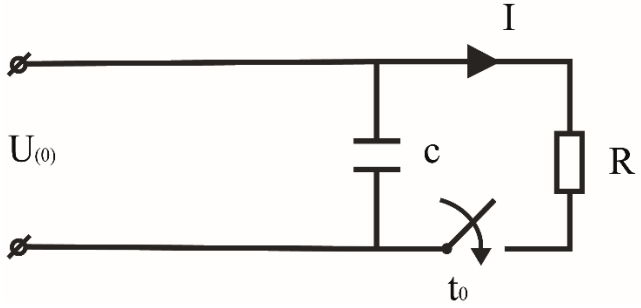

Figure 4. Electrical model of the aortic region of the vascular bed

It remains for us to define *Q*, i.e. blood flow through peripheral vessels. In accordance with Poiseuille's law, it can be written that the blood flow Q_o in the considered phase of the vascular site is equal to:

$$Q_o = (p_a(t) - p_B(t)) / R . \quad (13)$$

Assuming that the emptying backpressure pressure $p_в(t)$ in the vessels behind the peripheral resistance) is either small or constant, equation (13) can be easily converted into an equation for the rate of change of the volume of the considered section (Figure 3):

$$\frac{dV}{dt} = -Q_o . \quad (14)$$

Combining equations (10), (13) and (14) we obtain the equations for pressure changes in the diastolic phase in the aortic section:

$$\left(\frac{1}{K}\right) * \left(\frac{dp}{dt}\right) + \frac{p}{R} = 0. \quad (15)$$

We solve this equation, we get:

$$p(t) = P_{KS} * e^{-t/\tau}\ , \tag{16}$$

i.e. exponential, where P_{KS} is the pressure in the aorta at the end of systole (beginning of diastole), and $\tau = \frac{R}{K}$ is the time constant.

Based on the foregoing, it is not difficult to imagine a model of an elastic reservoir in the form of an electrical circuit in which the active resistance *R* would be an analogue of the hydraulic resistance, the capacitance *C* would be an analogue of volumetric elasticity, and the coordinate *p* (pressure) would be an analogue of the voltage *U* on the capacitor (Figure 4).

Based on the second Kirchhoff law for this circuit, it is easy to write the equation for voltage change (capacitor discharge):

$$RC\left(\frac{du_c}{dt}\right) + u_c = 0. \tag{17}$$

Solution of this equation:

$$u_c(t) = U_{c0} e^{-t/\tau}, \tag{18}$$

where U_{c0} is the initial voltage on the capacitor, τ is the time constant of the circuit equal to $\tau = RC$, t_0 is the beginning of the diastolic process.

Consider now the behavior of the aortic section in the systolic phase (the phase in which the heart pumps blood into the vascular system), i.e. when, along with the outflow of blood (Q_o), there is an inflow of blood (Q_i), which is formed by the heart (Figure 5). On Figure 5 shows the elastic reservoir in the systolic phase.

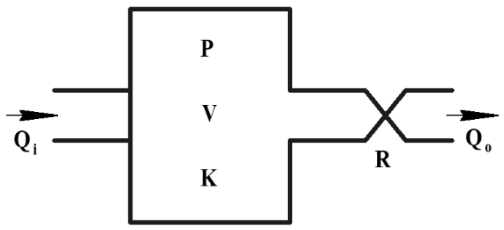

Figure 5. Elastic reservoir in systolic phase

We approximate the work of the heart by a pump with a pulsating ejection, which provides a certain instantaneous rate of blood expulsion – Q_o.

Let's connect such a heart with an aortic section (Figure 3) and, neglecting the resistance of the aortic valve, write the expression for the aortic volume:

$$V(t) = V_0 + \frac{p}{K}, \tag{19}$$

where V_0 is the volume of the aorta at zero pressure; *p* is the current pressure in the aorta.

The pressure value *P* now depends on the difference $Q_i - Q_o$, i.e. on the amount of blood flowing into and out of the section. Since Q_i in appeared, the expression for the rate of volume change will change:

$$\frac{dV}{dt} = Q_i - Q_o\ . \tag{20}$$

Let's differentiate expression (19):

$$\frac{dV}{dt} = \left(\frac{1}{K}\right) * \left(\frac{dp}{dt}\right)\ . \tag{21}$$

And combining (21) with (13) and (20) we get the equation for pressure change in the aortic section in the systolic phase:

$$\left(\frac{1}{K}\right) * \left(\frac{dp}{dt}\right) + \frac{p}{R} = Q_i(t)\ . \tag{22}$$

The solution of this equation differs from the solution of equation (15), because depends on the type of function $Q_i(t)$.

It is known from theoretical biomechanics that this function has a positive value only during systole and is zero during diastole. The simplest and most reliable way to approximate this function is with the help of a positive half-wave of a sinusoid (Figure 6).

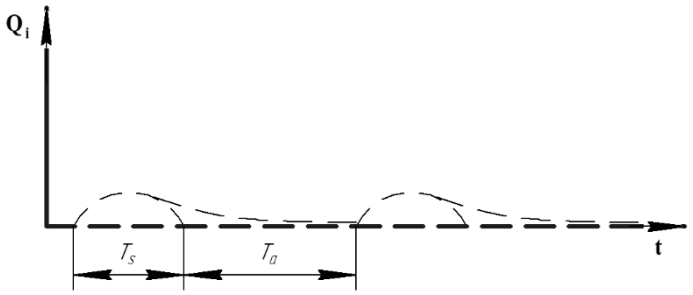

Figure 6. Illustration of systolic and diastolic processes

Accepted approximation of the function $Q_i(t)$, where T_s is the time of the systolic process, and T_d is the time of the dystonic process.

In the accepted temporary notation, the segment of the sinusoid describing the function $Q_i(t)$ during the systole period is determined by the following equation:

$$Q_i(t) = A_m \sin \frac{\pi}{T_s} t \,. \tag{23}$$

At $t = 0$ and $t = T_s$, function (23) is equal to zero.

Denote by P_s – variable pressure during systole, and by P_d – diastole, then equation (22), taking into account (23), will be rewritten as:

$$\frac{1}{K}\frac{dP}{dt} + \frac{P}{R} = A_m sin \frac{\pi}{T_s} t \,. \tag{24}$$

Let us introduce the notation:

$\nu = \frac{\pi}{T_s}$; $\alpha = \frac{K}{R}$; $r = K * a$;

$$A = \frac{K*a}{\left(\frac{K^2}{R^2}+\nu^2\right)^{\frac{1}{2}}} \; ; \; B = \frac{Ka\nu}{\frac{K^2}{R^2}+\nu^2} \; ; \tag{25}$$

$$\psi = \text{arctg}\frac{\nu R}{K} \; ; \tag{26}$$

$$T_s + T_d = T_0 \; ; \tag{27}$$

$$P_0 = \frac{A \sin(\pi-\psi) e^{-\alpha T_d} + B e^{-\alpha T_0}}{1 - e^{-\alpha T_0}} \,, \tag{28}$$

$$P_1 = \frac{A \sin(\pi-\psi) + B e^{-\alpha T_s}}{1 - e^{-\alpha T_0}} \,. \tag{29}$$

Using these notations and assuming that systole begins at time $t = 0$, we can show that the pressure during systole is:

$$p_s(\text{t}) = (p_0 + B) e^{-\alpha t} + A \sin(\nu t - \psi) \,. \tag{30}$$

And the pressure during diastole $p_d(t)$, respectively, is equal to: $p_d(t) = p_1 e^{-\alpha(t-T_s)}$. (31)

Thus, as can be seen from (30), the change in pressure is described by the sum of the shifted sinusoid from equation (23) and the exponential.

Based on the obtained relations, taking into account Figure 5, systolic and diastolic processes can be represented as the following substitution scheme (Figure 7).

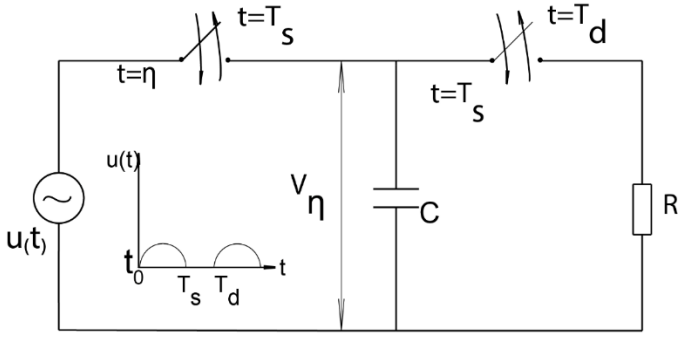

Figure 7. Scheme of substitution of systolic and diastolic processes

Mathematical model of the hemodynamic chain

The hemodynamic chain model is a serial connection of nine sections (Figure 8). Based on the results of previous studies, we introduced some simplifications. First of all, only the most significant active myogenic properties of its elements are taken into account in the model of the hemodynamic chain. For the heart, this is autonomy, contractility and the Starling mechanism, for resistive vessels, the phenomenon of "self-regulation". Secondly, the hydraulic resistance to the blood flow provided by each of the sections is taken into account in the form of some concentrated resistance. Thirdly, a huge number of resistive vessels connected in parallel and in series have been replaced by their model, which displays only the most typical types of non-linear dependence of blood flow in organs on arterial pressure. Finally, almost all non-linear dependencies characterizing the work of the heart, arteries, and veins are replaced by linear ones, both near a certain level characteristic of the initial state of the system, and over the entire expected range of changes [20].

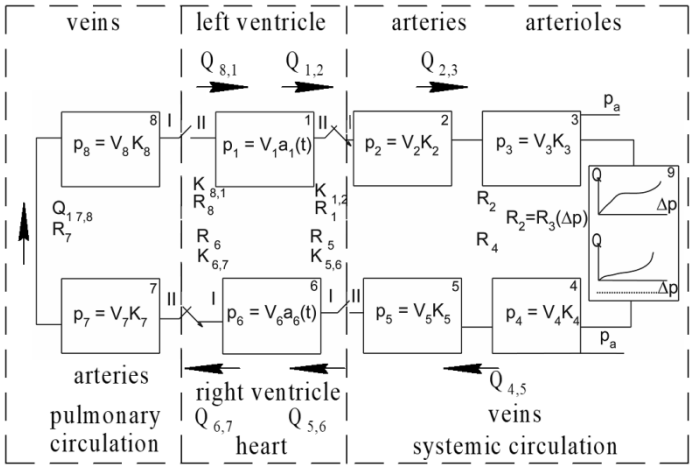

Figure 8. Model of the hemodynamic chain

Thus, such a model mimics the cardiovascular system, devoid of the control of the central nervous system.

The state of each section and the relationship of sections is described by three equations. They reflect changes in the following quantities.

The volume of the section x, as the difference between the inflow of blood into the section and its outflow:

$$V_x = V_{0x} + \int (Q_{x-1,x} - Q_{x,x+1})dt \,, \tag{32}$$

where V_{0x} is the initial volume of section *x* (as a constant of integration), $Q_{x-1,x}$ is the blood inflow into section *x*, $Q_{x,x+1}$ is the outflow of blood from section *x*.

Pressure in section *x* as a function of its compliance and blood volume:

$$p_x = K_x V_x \,, \tag{33}$$

where p_x is the pressure in the *x* section, K_x is the bulk elasticity coefficient characterizing the ability of the *x* section to deform.

Outflow of blood from section *x* as a function of pressure drop across hydraulic resistance:

$$Q_{x,x+1} = \frac{p_x - p_{x+1}}{R_x}, \quad (34)$$

where R_x is the resistance of section *x* to blood flow.
The behavior of the ventricular sections is described by the same equations, with the difference that the coefficient K_x is a function of two variables - end-diastolic volume and time:

$$P_x = V_x * K_x (V_x; t). \quad (35)$$

The dependence of K_x on time reflects cyclic changes in myocardial contractility, determined automatically, and the dependence on end-diastolic volume reflects the Starling mechanism.
The equation for blood flow through the heart takes into account the operation of the valves:

$$Q_{x,x+1} = \frac{p_x - p}{R_x} \text{ at } p_x > p_{x+1}, \quad (36)$$

$$Q_{x,x+1} = 0 \text{ at } p_x \leq p_{x+1}. \quad (37)$$

The equations characterizing the interconnected behavior of all sections of the cardiovascular system in two phases of its work can be reduced to the following system of equations:
Systolic phase:

$$\dot{V}_1 R_1 + V_1 a_1(t) = V_2 K_2 ; \quad (38)$$

$$V_2 K_2 = V_3 K_3 - p_2 - \sum_{i=1}^{2} \dot{V}_i R_2 ; \quad (39)$$

$$V_3 K_3 = V_4 K_4 - \sum_{i=1}^{3} \dot{V}_i R_3, \text{ for equations (38), (39), (40) } p_1 > p_2 ; \quad (40)$$

$$V_4 K_4 = V_5 K_5 + p_2 - \sum_{i=1}^{4} \dot{V}_i R_4 ; \quad (41)$$

$$\dot{V}_5 = -\sum_{i=1}^{4} \dot{V}_i \text{, for equations (41), (42) } p_5 \leq p_6, p_1 > p_2 ; \quad (42)$$

$$\dot{V}_6 R_6 + V_6 a_6(t) = V_7 K_7, \text{ for equations (43) } p_6 > p_7 ; \quad (43)$$

$$V_7 K_7 = V_8 K_8 - \sum_{i=6}^{7} \dot{V}_i R_7 ; \quad (44)$$

$$\dot{V}_8 = -\sum_{i=6}^{7} \dot{V}_i \text{, for equations (44), (45).} \quad (45)$$

Diastolic phase:

$$V_2 K_2 = V_3 K_3 - p_a + \dot{V}_2 R_2 ; \quad (46)$$

$$V_3 K_3 = V_4 K_4 = -\sum_{i=2}^{3} \dot{V}_i R_3 ; \quad (47)$$

$$V_4 K_4 = V_5 K_5 + P_a - \sum_{i=2}^{4} \dot{V}_i R_4, \text{ for equations (46), (47), (48) } p_1 \leq p_2 ; \quad (48)$$

$$V_5 K_5 = V_6 a_6(t) - \sum_{i=2}^{5} \dot{V}_i R_5, \text{ for equation (49) } p_5 > p_6 ; \quad (49)$$

$$\dot{V}_6 = -\sum_{i=2}^{5} \dot{V}_i, \text{ for } p_5 > p_6 ; \quad (50)$$

$$V_7 K_7 = V_8 K_8 - \dot{V}_7 R_7, \text{ for } p_6 \leq p_7 ; \quad (51)$$

$$V_8 K_8 = V_1 a_1(t) - \sum_{i=7}^{8} V_i R_8 ; \quad (52)$$

$$\dot{V}_1 = -\sum_{i=7}^{8} \dot{V}_i, \text{ for equations (52), (53) } p_8 > p_1 \ \ p_6 \leq p_7 . \quad (53)$$

Equation (38) describes the process of emptying the left ventricle into the arterial sector of the great circle. Equations (39), (40), (41) take into account the pressure loss that occurs when blood passes through the vessels, and the change in pressure or volume in the veins of the large circle. Equation (42) - the equation of the continuity of the blood flow in the systemic circulation.

The filling of the ventricle, for example, the right one during diastole, occurs due to the emptying of the venous section. Moreover, this emptying is carried out under the influence of the difference between venous and intragastric pressures (equations (49)-(50) for the right and (52)-(53) for the left ventricle). The physical meaning of the other equations of the diastolic phase is similar to the equations of the systolic phase.

The restrictions written on the right determine the operation of the heart valves and are the conditions for switching the phases of the process or structure of the cardiovascular system, that is, the change of the systolic process to diastolic and vice versa.

These equations correspond to the block diagram (Figure 8), in which the systolic and diastolic phases differ in the position of the valves $K_{1,2}$; $K_{5,6}$; $K_{6,7}$; $K_{8,1}$. In the systolic phase, the valves are in position "1", for the diastolic phase – in position "11". As follows from the diagram (Figure 8) and the equations, each phase has its own structure of the cardiovascular system. The phase sequence is thus determined by the corresponding cyclic change in the circuit structure. When the switching conditions are met, the final values of the parameters of one phase of the process, for example, systole, are assigned to their initial values for another phase, diastole, and vice versa. This adaptation to each other of two phases of the blood circulation process according to the corresponding values of the initial and final conditions, which determines their mutual subordination, can be considered as a functional link in the internal hydraulic feedback.

On Figure 9 shows a block diagram of the controlled (hemodynamic chain) and control (individual elements of the central nervous system) parts of the circulatory system.

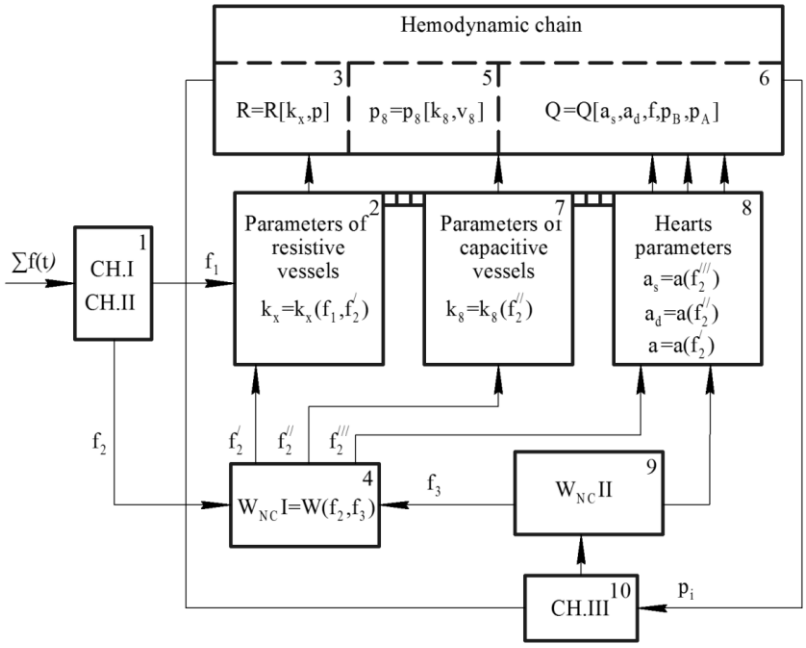

Figure 9. Block diagram of the circulatory system

$\sum F(t)$ is the external perturbing effect in relation to the circulatory system: the sum of the effects representing the signals of the system of motor reflexes, and others providing homeostasis, and the effects of the external environment, including the electromagnetic field.

It follows from the above diagram that the circulatory system is a parametric control system. Nerve center W_{NC1} imitates the control system by disturbance, W_{NC2} – control by deviation, i.e. management of the own state of the cardiovascular system. $f_1; f_2; f_3$ is the influences transmitted along the corresponding pathways (synapses) of the nervous system.

$\sum f(t)$ - general electromagnetic field (EMF) disturbance signal acting on the circulatory system. At medium EMF intensity, the effect of this signal affects the following parameters - parameters of resistive vessels (block 2), which leads to minor changes in the hemodynamic chain. The first elements are triggered (block 3) - the first stage of adaptation of the cardiovascular system to the environment begins.

As the signal increases, the sensitive elements of the first circulatory system regulation circuit are connected, the nerve centers of which are located in the spinal cord. The reaction of the spinal cord affects the parameters of the capacitive vessels (block 7) and the heart (block 8), which leads to an increase in the energy state of the cardiovascular system as a whole, which in turn leads to further activation of the spinal cord (blocks 5, 6) - the next stage of adaptation of the cardiovascular system to the environment begins.

Earlier, the fact of the adverse effects of the electromagnetic field on the circulatory system was noted. As follows from the above models of blood circulation, this effect can be realized through many channels; with the help of mathematical modeling, it is realistic to gradually assess the most susceptible to the effects of EMF areas of the circulatory system.

4. CONCLUSIONS

In this article, the influence of the external environment on the vital activity of the organism was considered using a model of the circulatory system consisting of the circulatory system itself - the hemodynamic chain and the central nervous system that controls it. The constructed model made it possible to consider the reactions of the circulatory system to the effects of the external environment depending on their intensity. After an element-by-element consideration of the hemodynamic chain, the second half of the article is devoted to the study of the influence of the central nervous system, which ensures adequate blood circulation under the influence of an unfavorable external environment, which is presented in the form of a block diagram displaying the direct effect of the electromagnetic environment on the elements of the vascular bed. The effect of electromagnetic fields leads to the emergence of forcing reflexes, which causes an increase in blood flow and arterial pressure. In the case of a more intense environmental impact, the central nervous system apparatus is connected to solve the main task of blood circulation through a series of appropriate sensitive elements and blood flow pressure feedback (works as an adaptive system). Depending on the intensity of electromagnetic influences, the block diagram displays two channels of influence of electromagnetic fields, starting from reflexes of ensuring the life support of the organism, known as forcing reflexes, to the connection of the centers of nervous regulation of blood circulation in the case of more intense influence of electromagnetic fields, up to the emergence of feedback on flow and pressure in the hemodynamic chain.

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