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MATHEMATICAL MODELS FOR DEVELOPMENT AND COMPENSATION OF HYPOXIC STATES DURING ISCHEMIC HEART DISEASE IN FLIGHT CREWS' PERSONNEL

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Abstract*—The increase of pilots' labor intensity caused by rapid development of flight technologies and increasing of complexity of combat missions faced by flight crew members increases the loads on organisms of flight personnel in general and in particular on their cardiovascular system. Domestic and foreign sources contain statistics indicating an increase in the number of flight accidents caused by cardiovascular pathologies in pilots, an increase in the number of flight personnel unsuitable for flight work due to the same reasons, a decrease in the age of flight crew members with cardiovascular pathologies. Therefore, issues of early detection and optimization of treatment processes of cardiovascular diseases in flight crew members are really necessary. Unfortunately, the possibilities of only instrumental approach to optimize the choice of ways for circulatory hypoxia correction in persons of aircrews caused by ischemic heart disease are rather limited. Simulation of the process of disease course for various methods of treatment by administering a pharmacological preparation can provide substantial assistance. This paper presents mathematical model of ischemic heart disease development in flight personnel, consisting on mathematical model of respiratory gas transport in pilot's organism, the model of conflict-controlled processes during self-organization of functional respiratory system, supplemented by differential equations describing changes in oxygen pressure in arterial blood during arteries stenosis caused by ischemic heart disease and equations describing the transport of pharmacological drug used for the treatment. Such composition of model, in case of its individualization, will optimize the treatment process by imitation of introduction of various drugs. Various methods of drug administration also were modeled - respiratory, oral, intramuscular and intravenous.*

Index Terms—Ischemic heart disease; hypoxic state compensation; mathematical model of respiratory gases mass transfer; pharmacological correction of hypoxic state; cardiovascular pathologies' of pilots.

I. INTRODUCTION

Aviation technique develops rapidly in contemporary world, and this consequently increases not only pilots' work intensity, but also the load on whole his organism in process of flight equipment exploitation [1]. These facts are co-exceeding with the studies of flight personnel morbidity: cardiovascular diseases posses 13.0–13.1% among all other diseases and obtained the second place in the structure of primary morbidity. During the estimation of pilots' professional health many authors, review of which works were published in [2], registered the prevalence of cardiovascular diseases which occupied a leading place in overall structure of pilots' morbidity. Persons from flight crews' which considered as unfit for the flight work due to the presence of arterial hypertension, atherosclerosis, chronic diseases of myocardium and coronary arteries up to the age of 40 years completed more than 60% from the number of people who were unsuitable because of internal organs diseases. Arterial hypertension possess the second place among the diseases of therapeutic profile and causes the removal from the flight work in 22% of cases [3]. Among the preconditions for flying cases linked with medical reasons, cardiological diseases were in 6 – 13%, with 1.9 times prevalence of coronary heart diseases for patients-pilots in comparison with ground specialists [2].

Analyzing the reasons of pilots' sudden deaths, American researchers have found that from 77 fatal cases –36 cases have happened due to cardiovascular pathologies: five deaths per one year in average. According to the Federal Aviation Administration and Transportation Security Council Of USA, 2% of

plane crashes were attributed to cardiovascular pathologies of pilots: at the age of 20-29 years – 0%, 30–39 years – 11.1%, 40-49 years – 27, 8%, 50–59 years – 22.2%, 60 years and older – 38.9% [2].

According to British researchers, the heart attacks removed pilot from the work and violate the aircraft control in 50% of cases [2].

The analysis of health state changes of civil aviation pilots that threatened the safety of flights was given in [2]. This analysis has demonstrated that pilots in age more than 50 years have had such states more often. Acute health states happened mostly due to cardiovascular pathology. Diseases of therapeutic profile (cardiovascular diseases predominantly) which caused the unsuitability of flight crews' persons were in 42.9% of cases. The mortality due to cardiovascular diseases – acute coronary insufficiency, acute cardiovascular insufficiency, cardiogenic shock caused 50% of total number of death-related diseases according to [2] on 2004.

II. Problem Statement

Ischemic heart disease is cardiovascular system pathology which causes disqualification of the flight crews' members as it is described in [4]. Also there were described the peculiarities of clinical course of heart attack and some attempts to provide an etiopathogenetic explanation [4]. The same author [5] has studied characteristics of ischemic heart disease diagnosis for flying personnel. There was revealed that ischemic heart disease (IHD) developing in pilots at younger age increases the frequency of its manifestation with the duration of the flight work, and it often happens asymptomatically causing certain diagnostic complexities with a real threat for flight safety [5]. Consequently there were concluded that the main emphasis in the medical-flight expertise should be done on the verification of diagnostics' methods.

In the absence of coronary artery stenosis, self-regulation mechanisms provide sufficient coronary blood circulation stability until the perfusion pressure in aorta is about 60 mm Hg and above. However, in conditions of widespread coronary atherosclerosis, decrease of perfusion pressure, further arterial stenosis and endothelial dysfunction of the involved segment causes the discrepancy between blood flow and metabolic needs of myocardium. With the impossibility of an adequate increase in blood flow in conditions of increased myocardium need in oxygen IHD develops [6], [7]. Thus, IHD is a state in which an imbalance between the myocardium need in oxygen and its delivery leads to myocardial ischemia and accumulation of metabolic products.

Therefore, in connection with abovementioned, issues of organism systems control as well as prevention, early diagnosis and optimization of processes of treating IHD in flight personnel are relevant. Unfortunately, the current methodological base is rather limited for these problems solution. Mathematical modeling, which makes it possible to simulate the course of IHD for flight crews' members and optimize the process of drugs choosing, taking into account the peculiarities of pilots' organisms, can provide essential support for stated problem solution.

The ***purpose*** of present work was to build mathematical model of course of coronary heart disease for persons in flight crews with coronary artery stenosis as conflict-controlled model of functional respiratory system, supplemented by the equations of pharmacological preparation transport in system of pilot's organism.

III. Problem Solution

A. Choosing of heart structural scheme for mathematical simulation

For mathematical simulation of pathological processes that appear in myocardium with the development of coronary heart disease, the work of the heart have been schematically represented as following. The heart has four compartments: two upper – right and left atrium, and two thick-wall chambers – right and left ventricles. The partition, which is a strong muscular wall, divides the heart into two parts: right and left. The heart has two valves: two-tailed (mitral) one between the left ventricle and the left atrium and three-fistula (aortic) between the right ventricle and the right atrium. Used blood from body tissues gets to the right side of the heart and pushed out in the lungs. Blood circulation through the lungs allows the small blood circle to be enriched with oxygen. Arterial blood returns to the left half of the heart and then pushed back to the tissues of the body through the systemic circulation. The heart muscle requires a lot of oxygen, and therefore, it needs intensive blood supply. The blood that passes through the heart chambers does not reach muscle cells, so the heart muscle has a separate network from blood vessels - the coronary system. Thus, coronary arteries deliver oxygen and nutrients to the heart; mainly there are the right and left coronary arteries.

B. Mathematical model

Let's study only one of possible reasons that lead to IHD – the damage (stenosis) of the coronary arteries. Applying the systemic approach for describing the processes of mass transfer and the

mass exchange of respiratory gases in human organism, let's imagine a functional system of respiration (FSR) in the form of controlled system in which the mass transfer of oxygen, carbon dioxide and nitrogen are going, and also the controller which produces certain effects that ensure the normal course of the process of mass transfer of gases [8-10]. The mathematical model of controlled part in [11] is represented by the system of ordinary differential equations that describe the dynamics of oxygen pressures at all stages of its ways in human organism; in parametric form it look like as:

$$\frac{dp_i\mathrm{O}_2}{d\tau} = \varphi\left(p_i\mathrm{O}_2, p_i\mathrm{CO}_2, \eta_i, \dot{V}, Q, Q_{t_i}, G_{t_i}\mathrm{O}_2, q_{t_i}\mathrm{O}_2\right),$$

$$\frac{dp_i\mathrm{CO}_2}{d\tau} = \varphi\left(p_i\mathrm{O}_2, p_i\mathrm{CO}_2, \eta_i, \dot{V}, Q, Q_{t_i}, G_{t_i}\mathrm{CO}_2, q_{t_i}\mathrm{CO}_2\right),$$

where the functions φ and φ are described in details in [11], $\dot{V}$ is the ventilation, η is the degree of hemoglobin saturation with oxygen, Q is the volume velocity of systemic and Q_{t_i} local blood flows, $q_{t_i}\mathrm{O}_2$ is the rate of oxygen consumption by *i*th tissue reservoir, $q_{t_i}\mathrm{CO}_2$ is the rate of carbon dioxide emission in *i*th tissue reservoir. The velocities of $G_{t_i}\mathrm{O}_2$ – oxygen flow from blood to tissue and $G_{t_i}\mathrm{CO}_2$ – carbon dioxide from tissue to blood is determined by the ratio

$$G_{t_i} = D_{t_i} S_{t_i} (p_{ct_i} - p_{t_i}),$$

where D_{t_i} is the coefficients of gas permeability through the aerogemal barrier, S_{t_i} is the surface area of gas exchange.

The purpose of control is the output of perturbed system in stationary mode [8], in which following ratio is executed:

$$|G_{t_i}\mathrm{O}_2 - q_{t_i}\mathrm{O}_2| \le \varepsilon_1, \ |G_{t_i}\mathrm{CO}_2 + q_{t_i}\mathrm{CO}_2| \le \varepsilon_2,$$

where ε_1, ε_2 are sufficiently small positive numbers that are defined in advance. In this case, following restrictions are imposed on control parameters:

$$0 \le \dot{V} \le \dot{V}_{\max}, \qquad 0 \le Q \le Q_{\max},$$

$$0 \le Q_{t_i} \le Q, \qquad \sum_{i=1}^{m} Q_{t_i} = Q,$$

where *m* is the number of tissue reservoirs in the body.

In addition, for the resolving of conflict situation between executive organs of regulation (respiratory muscles, cardiac muscle and smooth muscles of the vessels), which are also consumers of oxygen at the same time, and other tissues and organs in [8], [12], there were introduced the ratio:

$$q_{\text{resp.m}}\mathrm{O}_2 = f(V), \qquad q_{\text{card.m}}\mathrm{O}_2 = \varphi(Q),$$

$$q_{\text{sm.m}}\mathrm{O}_2 = \varphi(Q),$$

Let's take the next functional as criterion for regulation:

$$I = \min_{\substack{0\le\dot{V}\le\dot{V}_{\max}\\ 0\le Q_{t_i}\le Q_{\max}}} \int_{\tau_0}^{T} \left[\rho_1 \sum_{t_i} \lambda_{t_i} (G_{t_i}\mathrm{O}_2 - q_{t_i}\mathrm{O}_2)^2 + \rho_2 \sum_{t_i} \lambda_{t_i} (G_{t_i}\mathrm{CO}_2 + q_{t_i}\mathrm{CO}_2)^2\right] d\tau, \qquad i = \overline{1,m},$$

where $\tau_0 b$ is the moment of the start of perturbation influence on the system; T is the duration of this perturbation, ρ_1 and ρ_2 are coefficients that characterize the sensitivity of any organism to hypoxia and hypercapnia; λ_{t_i} are coefficients that reflect the morphological features of any tissue reservoir *i*.

Under such control, the total oxygen consumption in organism and in each tissue region as well as carbon dioxide accumulation is minimized.

The degree of coronary arteries damage could be simulated on models as result of partial occlusion of the cardiac muscle vessels, or their separate branches. With partial occlusion of coronary artery, the equations describing changes in the oxygen tension will be following [13], [14]:

$$\frac{dp_{ct_k}\mathrm{O}_2}{d\tau} = \frac{1}{\left(V_{ct_k} - \int_{\tau_0}^{T}\left(Q_{t_k} - \tilde{Q}_{t_k}\right)d\tau\right)\left(\alpha + \gamma_{\mathrm{Hb}}\mathrm{Hb}\dfrac{\partial\eta_{ct_k}}{\partial p_{ct_k}\mathrm{O}_2}\right)}\left(\alpha_1\tilde{Q}_{t_k}p_a\mathrm{O}_2 + \gamma_{\mathrm{Hb}}\mathrm{Hb}\tilde{Q}_{t_k}\eta_a - G_{t_k}\mathrm{O}_2 - \alpha_1\tilde{Q}_{t_k}p_{ct_k}\mathrm{O}_2 + \gamma_{\mathrm{Hb}}\mathrm{Hb}\tilde{Q}_{t_k}\eta_{ct_k}\right),$$

$$\frac{dp_{t_k}\mathrm{O}_2}{d\tau} = \frac{1}{V_{t_k}\left(\alpha_1 + \gamma_{\mathrm{Mb}}\cdot\mathrm{Mb}_k\dfrac{\partial\eta_{t_k}}{\partial p_{1_{t_k}}}\right)}(G_{t_k}\mathrm{O}_2 - q_{t_k}\mathrm{O}_2),$$

where index $k = r, l$ corresponds to the right or the left side of the heart; Q_{t_k} is the volume velocity of coronary blood circulation, determined by FSR

model; $\tilde{Q}_{t_k}$ its actual speed during pathological changes in the heart. Obviously that $\tilde{Q}_{t_k} < Q_{t_k}$.

Let's assume that the coronary vessels of the right and left parts of the heart are not damaged. So, in case of partial occlusion of the artery, gradients of oxygen tensions will be, in absolute value, greater than corresponding gradients in damaged vessel; depending on the degree of occlusion, hypoxia in the heart muscle will be less significant. Another effect might be seen when the damaged arterial vessel of the right or left side of the heart, or the degree of this damage are different. In the first case, the hypoxia occurs due to partial occlusion of one part of heart muscle; in other case – volume velocity of blood circulation will be greater than necessary and it will lead to increased oxygen tension and asymmetry in oxygen tension distribution in the heart muscle.

Otherwise, when degrees of the damage of arterial vessels that carry the blood to the right and left parts are different, the hypoxia develops in the muscles of both parts due to partial occlusion of vessels in varying degrees of manifestation and, thus, the distributions of oxygen tensions also will be asymmetric. With this model one can analyze the situation when the complete occlusion of capillary channel occurs in the elementary region of the heart muscle. In the initial period, there will be a sharp exhaustion of oxygen supply from the blood, the discrepancy in its supply to the needs of tissues surrounding the capillary, and as a result, in this tissue area pO_2 reaches its critical values, and the part of the heart muscle will not be able to take part in the provision its injection function. Thus, with coronary vessels damage, oxygen distribution in cardiac muscle depends on the degree of capillary bed damage and its localization.

C. Mathematical model of ischemic heart disease pharmacological correction

Let's use the results, described in [15] – [17]. The model of pharmacological correction was developed to restore the organisms' oxygen regimes, simulating the drugs administration using different methods: intramuscular (or intravenous) injection or inhalation.

Let's use following indications: $c_{f_{RW}}$ is a concentration of pharmacological preparation in respiratory tract (in moles), and through d_f its dose, then the equation of respiratory gases dynamics in the respiratory tracts [11] could be supplemented with the equations of preparation concentration as follows:

$$\frac{dc_{f_{RV}}}{d\tau} = \frac{\dot{V}}{V}\left(\tilde{c}_{f_{RV}} - \tilde{c}_{f_A}\right),$$

$$\tilde{c}_{f_{RV}} = \begin{cases} \xi d_f, \xi = 1, \text{ with preparation inhalation } (\dot{V} > 0), \\ \xi = 0, \text{ with inhalation absence } (\dot{V} > 0), \\ \tilde{c}_{f_{RV}}, \text{ with } \dot{V} \le 0, \end{cases} \qquad \tilde{c}_{f_A} = \begin{cases} c_{f_{RV}} \text{ with } \dot{V} > 0, \\ c_{f_A} \text{ with } \dot{V} \le 0. \end{cases}$$

The levels of $p_A O_2$, $p_A CO_2$, $p_A N_2$, as well as c_{f_A} in alveolar space are formed as a result of the gases mixing and the dispersion of pharmacological agent coming from the respiratory tract in the alveoli with those ones that are present in the alveolar space (taking into account the flow of gases and the drug through the alveolar-capillary membrane into the blood of pulmonary capillaries). Therefore, the equations of respiratory gases dynamics in the alveolar space have to be supplemented by the equation:

$$\frac{dc_{f_A}}{d\tau} = \frac{1}{V_L}\left(\tilde{c}_{f_A}(\tau)\dot{V} - G_{f_A} - c_{f_A}\frac{dV_L}{d\tau}\right),$$

$$G_{j_A} = D_j S(p_{j_A} - p_{j_{Lc}}), \quad G_{f_A} = D_f S\left(c_{f_A} - c_{f_{Lc}}\right),$$

where D_j, D_f are coefficients of gases' and pharmacological preparations' permeability through the alveolar-capillary membrane whose surface area is equal S.

Respiratory gases are transmitted by the blood in different ways: oxygen – being dissolved in blood plasma and chemically bound by hemoglobin (Hb); carbon dioxide – dissolved in plasma, being chemically bound to hemoglobin and bicarbonate compounds (BC); and nitrogen – only in dissolved form. Let's also assume that pharmacological preparation is carried by the blood in dissolved form. Therefore, the equations of changes in tensions of oxygen and carbon dioxide in the blood are substantially nonlinear, and the dynamics of tensions of nitrogen and concentrations of preparations may be described by linear differential equations:

$$\frac{dc_{f_{Lc}}}{d\tau} = \frac{1}{V_{Lc}}\left((Q - Q_{sh})(c_{f_{\bar{v}}} - c_{f_{Lc}}) + G_{f_A}\right),$$

where Q, Q_{sh} are volume velocities of systemic circulation and blood circulation in case of lungs' shunting, α_1, α_2, α_3 are coefficients of solubility of gases in blood plasma, Hb and BH are

concentrations of hemoglobin and buffer bases in the blood; γ, γ_{BH} are Gyufner constants; the degree of hemoglobin saturation with oxygen is determined by the ratio of blood to the pulmonary capillaries.

Similarly, the equations of changes in tensions of gases and pharmacological preparation in arteries' blood were formed. It should be taken into account that the level of gas tensions and preparation concentrations are formed as a result of the instant mixing of their streams coming from the blood of pulmonary capillaries and mixed venous blood, with gases and preparation contained in the arterial stream:

$$\frac{dc_{f_a}}{d\tau} = \frac{1}{V_a}\left((Q - Q_{sh})c_{f_{Lc}} + Q_{sh}c_{f_{\bar{v}}} - Qc_{f_a}\right),$$

The arterial blood system is branching into the microcirculation networks in organs and tissues. Mathematical models of the functional respiratory system describe the dynamics of tensions in respiratory gases in m tissue reservoirs, among which, as a rule, there are tissues of brain, kidneys, liver, gastrointestinal tract, heart muscles, skeletal muscles, bone and fat tissues.

Let's suppose that $V_{t_i}, i = \overline{1,m}$ is a volume of the tissue fluid of the reservoir; t_i, V_{ct_i} is a blood volume in generalized capillary; S_{t_i} is the surface area of mass exchange of gases and substances; $D_{t_i j}$, $D_{f_{t_i}}$ are permeability coefficients of gases and substances through the capillary-tissue membrane.

Then, the equations that describe the changes in respiratory gases tensions and concentration of pharmacological preparation in the blood that washes the tissue, and in tissue fluid of reservoir t_i, $i = \overline{1,m}$, could be written by completing the equation for the blood of tissue capillaries by the expression:

$$\frac{dc_{f_{ct_i}}}{d\tau} = \frac{1}{V_{ct_i}}\left(Q_{t_i}(c_{f_a} - c_{f_{ct_i}}) - G_{f_{t_i}}\right),$$

and for the tissue fluid by completing of the expression by equation:

$$\frac{dc_{f_{t_i}}}{d\tau} = \frac{G_{f_{t_i}}}{V_{t_i}},$$

where $G_{f_{t_i}} = D_{f_{t_i}} S_{t_i}(c_{f_{ct_i}} - c_{f_{t_i}})$.

If the preparation is injected into the muscle tissue with a bulk speed Q_f, one have to write this in form:

$$V_{t_i}\frac{dc_{f_{t_i}}}{d\tau} = dQ_{f_{t_i}} + G_{f_{t_i}}.$$

It should be noted that, $\sum_{t_i} Q_{t_i} = Q$.

During development of mathematical model for transport and mass exchange of respiratory gases and pharmacological preparations (drug) it was assumed that the drug does not involved directly in metabolic processes, but it is a regulatory factor in stabilizing and compensating of hypoxia. The drug withdrawal from organism is carried out through the respiratory tract and urinary system. Therefore, changes in the concentration of drug in kidneys are determined by the equation:

$$\frac{dc_{f_{t_n}}}{d\tau} = G_{f_{t_n}} - Q_{f_{t_n}},$$

where $Q_{f_{t_n}}$ is the rate of liquid filtration, t_n is the tissue of the kidneys.

Let's assume that the drug f belongs to the pharmacological group, which promotes vasodilation of capillaries walls. Its influence on capillaries' smooth muscles leads to more free flow of oxygen and carbon dioxide through barrier that separates blood and tissue fluid and, at the same time, it reduces the rate of oxygen utilization by capillaries' smooth muscles.

Therefore, the magnitude of gases flow through the membrane can be written in the terms of ratio:

$$G_{jt_i} = K(c_{f_{t_i}})D_{jt_i}S_{t_i}(p_{jct_i} - p_{jt_i}),$$

where $K(c_{f_{t_i}})$ is the functional amplifier of the process of respiratory gases diffusion into the tissue reservoir. Experimental studies give possibility to suppose that $1 \le K(c_{f_{t_i}}) \le 2$ for most drugs of this type.

In a venous system, the blood from organs and tissues is mixed and transported for oxygen saturation into the lungs. The tensions of respiratory gases and drug concentration are formed at each time moment according to the equations:

$$\frac{dc_{f_{\bar{v}}}}{d\tau} = \frac{1}{V_{\bar{v}}}\left(\sum_{t_i} Q_{t_i}c_{f_{ct_i}} + Q_{t_{sh}}c_{f_a} - Qc_{f_{\bar{v}}}\right).$$

If the drug is injected into the body intravenously, then the equation of pharmacological preparation (drug) transport in the mixed venous blood should be written in the form:

$$\frac{dc_{f_{\bar{v}}}}{d\tau} = \frac{1}{V_{\bar{v}}}\left(\sum_{t_i} Q_{t_i} c_{f_{ct_i}} + Q_{t_{sh}} c_{f_a} - Q_{f_{\bar{v}}} c_{f_{\bar{v}}}\right),$$

where $Q_{f_{\bar{v}}}$ is the volume velocity of intravenous injection of the drug.

In case of oral administration of pharmacological preparation (drug) let's assume that index t_i refers to the gastrointestinal tract in this equation.

Differential equations and algebraic relationships describe completely the transport, mass exchange of respiratory gases and pharmacological preparations in chosen structure of organism respiratory system during the respiratory cycle.

IV. Conclusion

Mathematical model of respiratory gas transport could be a useful tool for investigation of ischemic heart disease reasons. It also allows ones to choose the best way for compensation of circulatory hypoxia that develops during ischemic heart disease. The mathematical model of ischemic heart disease have been supplemented by equations of pharmacological preparation transport in which various methods of drug administration was studied: respiratory, oral, intramuscular, and intravenous. The model was individualized, the imitation of pharmacological agent administration was done, and thus the optimal dose of the drug and the optimal route of it administration could be selected for the compensation of the secondary tissue hypoxia that was developed in the person suffered from ischemic heart disease.

Proposed mathematical model allows choosing the best way for the compensation of circulatory hypoxia that causes ischemic heart disease in pilots and other flight crew members. Mathematical model of ischemic heart disease for flight personnel in the case of coronary artery stenosis, consisting of mathematical model of mass transfer and respiratory gases mass transfer in pilot's organism, a model of conflict-controlled processes with optimal selection of blood flow, supplemented by equations describing partial occlusion of heart muscle vessels and equations for pharmacological preparation transport will allow to predict the courses of pathologies of these type among flight crew members and to optimize treatment process by simulating the introduction of any drug into individualized model of any person from flight crew. This mathematic model also may be used for studying of drug administration by various ways: respiratory, oral, intramuscular and intravenous.

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Н. І. Аралова, О. М. Ключко, В. Й. Машкін, І. В. Машкіна. Математичні моделі розвитку та компенсації гіпоксичних станів при ішемічній хворобі серця у осіб льотного складу

Збільшення інтенсивності праці льотчиків, викликане швидкими темпами розвитку льотної техніки і ускладненням бойових завдань, що стоять перед членами льотних екіпажів, збільшує навантаження на організм осіб льотного складу в цілому, і зокрема на серцево-судинну систему. Вітчизняні та зарубіжні джерела містять статистику, яка свідчить про збільшення кількості льотних пригод, викликаних серцево-судинними патологіями у льотчиків, зростанні кількості осіб льотного складу, визнаними непридатними до льотної роботи з тих же причин, зниженням віку членів льотних екіпажів, що мають патології серцево-судинної системи. Тому актуальними є питання своєчасного виявлення та оптимізації процесу лікування серцево-судинних захворювань у членів льотних екіпажів. На жаль, можливості тільки інструментального підходу для оптимізації вибору шляхів корекції циркуляторної гіпоксії у осіб льотного складу, викликаної ішемічною хворобою серця досить обмежені. Суттєву допомогу може надати імітаційне моделювання процесу перебігу захворювання за різних способів лікування шляхом введення фармакологічного препарату. У даній роботі представлено математичну модель розвитку ішемічної хвороби серця у осіб льотного складу, що складається з математичної моделі транспорту респіраторних газів в організмі льотчика, моделі конфліктно-керованих процесів у разі самоорганізації функціональної системи дихання, доповнена диференціальними рівняннями, що описують зміни напружень кисню в артеріальній крові при стенозі артерій, викликаних ішемічною хворобою серця і рівняннями, що описують транспорт фармакологічного препарату, який використовується для лікування. Такий

склад моделі, в разі її індивідуалізації, дозволить оптимізувати процес лікування шляхом імітації введення різних препаратів. Передбачаються також різні способи введення препаратів – респіраторний, пероральний, внутрішньом'язовий та внутрішньовенний.
Ключові слова: ішемічна хвороба серця; компенсація гіпоксичного стану; математична модель масопереносу респіраторних газів; фармакологічна корекція гіпоксичного стану; серцево-судинні патології льотчиків.

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Н. И. Аралова, Е. М. Ключко, В. И. Машкин, И. В. Машкина. Математические модели развития и компенсации гипоксических состояний при ишемической болезни сердца у лиц летного состава

Увеличение интенсивности труда летчиков, вызванное быстрыми темпами развития летной техники и усложнением боевых задач, стоящих перед членами летных экипажей увеличивает нагрузку на организм лиц летного состава в целом и а частности на сердечно-сосудистую систему. Отечественные и зарубежные источники содержат статистику, свидетельствующую об увеличении числа летных происшествий, вызванных сердечно-сосудистыми патологиями у летчиков, росте числа лиц летного состава, признанными непригодными к летной работе по тем же причинам, снижением возраста членов летных экипажей, имеющих патологии сердечно-сосудистой системы. Поэтому актуальными являются вопросы своевременного выявления и оптимизации процесса лечения сердечно-сосудистых заболеваний у членов летных экипажей. К сожалению, возможности только инструментального подхода для оптимизации выбора путей коррекции циркуляторной гипоксии у лиц летного состава, вызванной ишемической болезнью сердца достаточно ограничены. Существенную помощь может оказать имитационное моделирование процесса течения заболевания при различных способах лечения путем введения фармакологического препарата. В настоящей работе представлена математическая модель развития ишемической болезни сердца у лиц летного состава, состоящая из математической модели транспорта респираторных газов в организме летчика, модели конфликтно-управляемых процессов при самоорганизации функциональной системы дыхания, дополненная дифференциальными уравнениями, описывающими изменения напряжений кислорода в артериальной крови при стенозе артерий, вызванных ишемической болезнью сердца и уравнениями, описывающими транспорт фармакологического препарата, используемого для лечения. Такой состав модели, в случае ее индивидуализации, позволит оптимизировать процесс лечения путем имитации введения различных препаратов. Предусматриваются также различные способы введения препаратов – респираторный, пероральный, внутримышечный и внутривенный.
Ключевые слова: ишемическая болезнь сердца; компенсация гипоксического состояния; математическая модель массопереноса респираторных газов; фармакологическая коррекция гипоксического состояния; сердечно-сосудистые патологии летчиков.

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