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SIMULATING OF HUMAN PHYSIOLOGICAL SUPERSYSTEMS: INTERACTIONS OF CARDIOVASCULAR, THERMOREGULATORY AND RESPIRATORY SYSTEMS

A special quantitative model of the human thermoregulatory system (MT) functioning with cardiovascular and lung systems is created. These systems form a physiological super-system (PSS). For a naked or cloth human, algorithms provide designing of scenarios including simulation of either short-time or long-time (hours or days) observations. Input data include different combinations of environmental variables (air or water temperature, air humidity, wind or water flow speed, light intensity), as well as designing of dynamics for certain biological characteristics (rate of heat production including its components associated with metabolism and ATP molecules leasing during mental and physical activities). The human body consists of three compartments – core, blood, and skin. Dynamic output data include blood, hypothalamic, and skin temperatures, hemodynamic parameters (heart rate, cardiac output, regional blood flows, vascular resistances, blood pressures, and regional blood volumes), and lung ventilation. Using associations of dynamics of day/night light intensity with concentrations of serotonin and melatonin hormones, a model for biological heat production rate dynamics is proposed. Currently, the PC-based simulator is autonomous C+ software. Its users can be both student-medics and physiologists interested in providing theoretical research. Shortly, this simulator has to be widened by models of kidneys and liver-pancreas interaction mechanism.

Key words: physical health, cell energy balance, control mechanisms,quantitative models, simulator.

Introduction

Human organs and certain anatomical-functional systems (AFS) form very complex functional systems known as physiological super-systems (PSS). The general concept of human PSS [1-3] explained deep cellular mechanisms that determine cells interaction for dynamic providing of every AFS's optimal parameters. However, traditional empiric physiology possesses not by research technologies capable of establishing the main quantitative laws ruling the functionalities of PSS. Potentially, mathematical models could help in solving of this problem. However, almost all models were created for solving specific partial problems therefore they not concern the problem of PSS. To fill this methodological gap in, we are consequentially creating proper mathematical models and computer simulators [4-7]. Their main novelty is in combining of multi-level physiological mechanisms for explaining of organism-scale adaptive physiological responses to environmental alterations. In fact, this approach also creates potentials for explaining mechanisms that determine the dynamic multi-parametric shape of human physical health (HPH). Such a theoretical fundament is extreme necessary for the individualization of the medical assessment of HPH.

The goal of this article is to present our latest development that made possible theoretical investigations of human thermoregulatory system under unpredictable challenges from a certain AFS namely, from the cardiovascular system (CVS) and lung system.

Mathematical model of thermoregulatory system

The model of CVS is presented in [6,7], thus there is no necessity for its de-scription in detail. Perhaps, it is sufficient to note that our CVS-model currently is the most complex model, including in it both mechanisms of circulation's acute control and mechanisms that determine the long-term parameters of CVS. As to our model of the thermoregulatory system (MT), the main reason for its creation was that despite a lot of such models (for example, [8-14]), MT should be compatible with our other models.

For solving our problems in the frame of human PSS, it is sufficient to have an MT containing three body compartments: a core

(it represents muscle, subcutaneous tissue, and bone), skin, and blood.

The most distinguishing sign of every PSS is that it functionally integrates multiple AFS. The extreme necessity in novel models is conditioned with the practical impossibility to provide quantitative empirical research of complex and multi-level mechanisms governing the interaction of multiple HPSS in physiological or pathological conditions. At the same time, it is clear that often complex pathologies appear because of inadequate functioning one or more functional chains of PSS. So, an alternative – simulative research of human PSS is highly encouraged.

In parallel with the creating of novel models, several models created in the frame of the traditional methodology can be re-vised in the light of the PSS concept. Our current publication presenting such an approach illustrates both the modeling technology and the main problems arising under its application to models published earlier [6,7]. These publications were chosen because AFSs of this PSS represent organs and systems that are external providers of cellular metabolism. As to its intracellular optimizers, briefly described in [1] (only the energy aspect), we intend to create a special model that has to include also those nuclear mechanisms that are under cytoplasm-released adaptation factors. In fact, namely, these factors increase/decrease the expression of genes in specific sites of DNA.

Special software does provide both tunings of models and all procedures ac-companying a wide range of their simulation research. Such work is too big to be made within a usual short-term research project and described in a usual research article. Therefore, we are publishing each interim developmental result that can also be an autonomous unit-software.

This publication represents MT interaction with models of CVS and LV.

Principles for modifying the earlier created software in order to incorporate MT in it are additionally described. The final purpose of this multi-stage developmental project is to provide physiologists-researchers with modern computer-based information technology with dual goals. The first goal is simulation research of human certain complex PSSs. The second goal is to provide professors and medical students with a modern specialized visualization technology capable of helping future medics to better understand the physiological basis of non-trivial pathologies.

Short description of MT

A quantitative model pretending to simulate certain biophysical and physiological events concerned with human ther-moregulation, must deal with dynamics of heats both generation and conduction (dissi-pation). In the human body, immersed in a non-stable environment mean rates of heat generation ($v_g(t)$) and heat transfer ($v_c(t)$) permanently alter. So, dynamics of the body heat ($H(t)$) is commonly described by a differential equation:

$$mc\frac{dH}{dt} = v_g(t) - v_c(t).$$

where m is the body mas while a specific heat of c characterizes alteration velocity.

By solving this equation, one can see that within every time interval of τ:

$$H(\tau) = H(t_0) + mc\int_{t-\tau}^{t}(v_g(t) - v_c(t))dt$$

In statics, a mean value of body temperature (T_{sm}) is calculated as $T_{sm} = H/m$. But different structures of the human body have their individual mas and parameter c.

A summary heat $H_\Sigma(t)$ of the human body is formed of two independent sources – biological $H_b(t)$, and external $H_E(t)$.The last one is proportional to air temperature, while $H_b(t)$ has its independent sources explained further.

$H_b(t)$ is resulted in four independent heat generators: 1) exothermic chemical transformations accompanying metabolism $H_M(t)$; 2) life events providing organism's functional integrity $H_I(t)$; 3) life events providing human mental activity $H^M(t)$; and 4) life events providing human physical activity $H_W(t)$. Three last heats resulted from the leasing of

ATP molecules as the main energy source for providing biological works. Their importance in entire body thermodynamics becomes clear when accentuating the following facts: i) about 40-60 % of the energy released during one ATP molecule is dissipated in the form of heat; ii) the total amount of everyday synthesis (leasing) of ATP is about 50-55 kg. $H_b(t) = b_1 \cdot H_M(t) + b_2 \cdot H_I(t) + b_3 \cdot H^M(t)$ $+b_4 \cdot H_W(t),$:

In the last equation, the three last components depend on the power of every activity thus coefficients $b_1 - b_4$ are approximated for physiological rest conditions only.

Heat conduction between compartments is provided by radiation, convection, and evaporation (for lung and skin). MT operates with fourth temperatures: core $-T_C^o(t)$, blood $-T_B^o(t)$, skin $-T_S^o(t)$, and environmental $-T^o{}_A(t)$. $T_C^o(t)$ is resulted of $H_b(t)$ and heats taken from the neighbors (blood and skin compartments). By analogy $T_B^o(t)$ is resulted of $T_C^o(t)$, $T_B^o(t)$, and $T_S^o(t)$. As to $T_S^o(t)$, its providers are $T_C^o(t)$, $T_B^o(t)$, and $T^o{}_A(t)$. So, to model the dynamics of these variables, their relationships should have been formalized. Below the formalizations are presented:

$$\frac{dT_C^o}{dt} = \lambda_1 H_b - \lambda_L H_{Ev}^L + \lambda_2 (T_C^o - T_B^o) + \lambda_3 (T_C^o - T_S^o)$$

$$\frac{dT_B^o}{dt} = \lambda_4 H_C - Q - \lambda_5 (T_C^o - T_B^o) - \lambda_6 (T_C^o - T_B^o) - \lambda_7 (T_C^o - T_S^o)$$

$$\frac{dT_S^o}{dt} = \lambda_8 H_C - \lambda_S \cdot H_{Ev}^S + \lambda_9 (T_C^o - T_S^o) + \lambda_{10} (T_B^o - T_S^o) + \lambda_{11} (T_S^o - T_A^o)$$

$$\frac{dH_{Ev}^L}{dt} = a_L v_L - H_{Ev}^L Y / X,$$

$$\frac{dH_{Ev}^S}{dt} = a_S q_S - H_{Ev}^S Y / X,$$

$$H_C(t) = H_b(t) -$$

where X - is the air humidity, Y - wind velocity, q_S - skin flow.

So, our MT is the first model capable of simulating the specific effects of body different energy sources. In this paper, this aspect is not considered.

Under the modeling of the human thermoregulatory system, the human body usually is approximated as a net of lumped parametric compartments [1-4]. The net structure is conditioned by research goals. Our MT is necessary to investigate main aspects of human PSS composed of different combinations of cardiovascular, thermoregulatory, lungs, energy providing systems. Therefore, specific details of thermal patterns in peripheral segments (legs, hands, torso, neck, and head) that had been considered in [2, 7-11], are mainly omitted. We have built the simplest model containing of three body compartments (a core, blood, and a skin). Additionally, MT is capable of imitating the isolative effects of clothing. Besides, in the final version of MT, either air or water can be chosen to be an external environment in which the body is immersed. Each of these environments does have its temperature and flow velocity given as input parameters.

This structure of MT is chosen for solving practically all tasks planned in the frame of the final version of the human physiological super-systems' model. As our MT uses certain variables of the CVS model, it is useful to note the following.

For the physiological interval of $F_{\min} \le F(t) \le F_{\max}$, $F(t)$ is calculated as:

$$F(t) = \begin{cases} F_{\min}, & \sum_{j=1}^{n} \Delta F_i^-(t) > F_a - F_{\min} \\ F_a + \Delta F(T^o) + \sum_{i=1}^{n1} \Delta F_i^+(t) - \sum_{j=1}^{n} \Delta F_i^-(t), & \\ F_{\min} \le F(t) \le F_{\max} & \\ F_{\max}, & \sum_{i=1}^{n2} \Delta F_i^+(t) + F_a > F_{\max} \end{cases}$$

Here F_a is the heart rate under normal rest values of blood temperature T_N^o,

biochemical and biophysical characteristics of cells of sinus node, $\Delta F(T^o)$ is elevation of F_a with temperature increasing, $\Delta F_i^+(t)$ are accelerating effects of n mechanisms (including concentration of adrenalin $A_d(t)$), and $\Delta F_j^-(t)$ are retarding effects of n mechanisms.

Each mechanism forming its part of ΔF has its power and developmental inertia that have been taken into account by means of proper constants.

Inotropic states of ventricles are under influences of local coronary flows $q_c(t)$, adrenalin $A_d(t)$, $T_B^o(t)$, efferent sympathetic ($E_{Sh}(t)$), and parasympathetic ($E_{Vh}(t)$) impulse patterns. Special version of the model includes effects of exogenous cardio-active agents $C_a(t)$ too:

$$k_i(t) = k0_i * (1 + d_1 * (T^o(t) - T^o{}_N) + d_2 * (A_d(t) - A_{dN}) + d_3 * (q_c(t) - q_{cN}) + d_4 * (E_{cS}(t) - E_{cSN}) - d_5 * (E_{cV}(t) - E_{cVN}) \pm d_e * C_a(t))$$

Skin vascular resistance depends on vascular volume (diameter). There is a biophysical reverse relation between temperature and vascular resistance. The relation is described in our CVS model. In particular, it means that the independent (hemodynamic) alterations of skin flow, which plays an important role in body heat conduction, will have specific thermoregulatory effects.

Mathematical model of environment

In our current model, environment is presented by following dynamic characteristics: air temperature $T_B^o(t)$ ($T^o{}_A(t)$), air humidity ($h_A(t)$), wind speed ($v_W(t)$), and light intensity ($L_I(t)$).

$T^o{}_A(t)$ may be of any stable value $T^o{}_A$ either may have any dynamics. For example, under simulating of day/night $T^o{}_A(t)$ its formally is given as:

$$T^o{}_A(t) = \begin{cases} T^o{}_{AN}, t < t_{sr}; t > t_{fss} \\ T^o{}_{AN} + (T^o{}_{AD} - T^o{}_{AN})(1 - e^{-\gamma(t - t_{sr})}), t_{sr} < t < t_s \\ T^o{}_{AD}, t_s \le t \le t_{sss} \\ T^o{}_{AD} - (T^o{}_{AD} - T^o{}_{AN})(1 - e^{-\mu(t - t_{sr})}), t_{sss} \le t \le t_{fss} \end{cases} \quad (*)$$

where $T^o{}_{AN}$ - night minimal temperature, $T^o{}_{AD}$ - day maximal temperature, t_{sr} - sunrise start, t_s - fully lit morning, t_{sss} - sunset start time, t_{fss} - completely dark night.

By analogy dynamics of $L_I(t)$ is given as:

$$L_I(t) = \begin{cases} L_N, t < t_{sr}; t > t_{fss} \\ L_N + (L_D - L_N)(1 - e^{-\gamma(t - t_{sr})}), t_{sr} < t < t_s \\ L_D, t_s \le t \le t_{sss} \\ L_D - (L_D - L_N)(1 - e^{-\gamma(t - t_{sr})}), t_{sss} \le t \le t_{fss} \end{cases} \quad (**)$$

where L_N - minimal night intensity of light, L_D - maximal day intensity of light, t_{sr} - sunrise start, t_s - full sunny morning time, t_{sss} - sunset start time, t_{fss} - completely dark night. Coefficient γ in (*) is to modulate air temperature dynamics depended on air humidity $X(t)$.

The hypothalamic control of blood temperature ($T_B^o(t)$) is based on afferent information came from heat and cold receptors located in core, blood, and skin compartments. In a conventional scale of "0-1", the current activity of every heat receptor field looks like:

$$R_j^h(t) = \begin{cases} 0, \ T_j^o(t) \le T_{j\min}^h \\ (1 - e^{-\delta_j \chi_j}) / (1 + \theta_j e^{-\delta_j \chi_j}), T_{j\min}^h < T_j^o(t) < T_{j\max}^h \\ 1, T_j^o(t) > T_{j\max}^h \\ \chi_j = T_j^o(t) - T_{j\min}^h \end{cases}$$

where $T_{j\min}^o$ is reception threshold, $T_{j\max}^o$ is receptors saturation temperature, δ_j and θ_j are approximation constants. By analogy, cold functions of cold receptors are modeled as:

$$R_j^c(t) = \begin{cases} 0, \ T_j^o(t) \le T_{j\min}^c \\ (1 - e^{-\delta_j^c \chi_j^c}) / (1 + \theta_j^c e^{-\delta_j^c \chi_j^c}), T_{j\min}^c < T_j^o(t) < T_{j\max}^c \\ 1, T_j^c(t) > T_{j\max}^c \\ \chi_j^c = T_{j\min}^c - T_j^o(t) \end{cases}$$

Hypothalamic neurons modulate their descending impulse activity depending on

summary ascending impulses $R_{\Sigma}^{h}(t)$ and $R_{\Sigma}^{c}(t)$ calculated as:

$$R_{\Sigma}^{h}(t)=\sum_{j} d_{j}^{h} R_{j}^{h}(t); \quad R_{\Sigma}^{c}(t)=\sum_{j} d_{j}^{c} R_{j}^{c}(t)$$

where d_{j}^{h}, and d_{j}^{c} are weight coefficients.

The main target for descending influence of hypothalamic neurons is the velocity of heat generation ($v_{hg}(t)$). Its static value is:

$$v_{hgs}=v_{hg}(0)(1+g_{1}(W-W0)(1-g_{2}H_{E})(1+$$
$$+g_{3}S)(1+g_{4}R_{\Sigma}^{c}-g_{5}R_{\Sigma}^{h})$$

where $g_{1}-g_{5}$ are weight coefficients.

In the current model, $v_{hg}(t)$ is presented as:

$$T_{hg}\frac{dv_{hg}}{dt}=v_{hgs}-v_{hg},$$

where T_{hg} is time constant of heat generation alteration.

$$\frac{dH_{b}}{dt}=b_{5}H_{b}-v_{hg}.$$

Hypothalamic temperature $T_{h}^{o}(t)$ is calculated using $T_{h}^{o}(t)$

$T_{h}^{o}(t)=T_{B}^{o}(t)-\Delta$, where $\Delta=const.$

Heart rate, skin flow, and lung ventilation velocity ($v_{l}(t)$) are external modulators of MT. Two first characteristics belong to CVS: its model is described in [6-8]. Thus we give here $v_{l}(t)$:

$$\frac{dv_{l}}{dt}=b_{l1}C_{B}^{H^{+}}+b_{l2}C_{B}^{CO_{2}}+b_{l3}T_{B}^{o},$$

where b_{l1}, b_{l2}, b_{l3} are approximation constants, $C_{B}^{H^{+}}$ and $C_{B}^{CO_{2}}$ represent blood concentrations of H^{+}, and CO_{2}.

Simulation algorithms

MT is integrated with CVS-model and can interact with it for a wide range of special cardiovascular test scenarios. In this publication we cannot consider these oppor-tunities thus we illustrate those scenarios only when the CVS is under physiological norm (imitating an intact organism). Under this constriction, a single simulation algorithm (SA) can be illustrated by means of Fig.1.

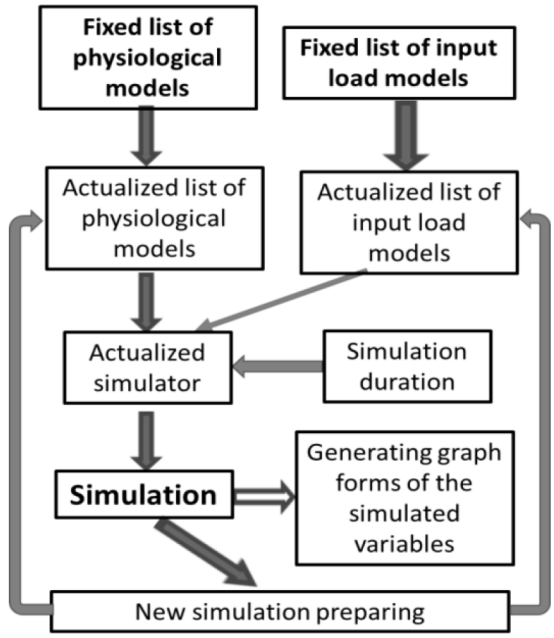

Fig.1. Simulation algorithms

According to this algorithm, the user should set the simulation duration, and the state of the external environment (for example, dynamics of light intensity, air temperature, humidity, wind speed, cloth isolating parameters, e.a.). Special window is pro-posed for setting (actualizing) the model several constants: after changing at least one value in MT characteristics, the user can start a new simulation.

Simulation (when activated) will last until the exposure time (observation duration) is over. Special algorithm providing both short-time and long-term (days and months) simulations is created. So, the operative memory of PC is no longer critical for determining the maximal simulation duration as it was in previous simulator version [5].

After the simulation is over, the user can analyze simulation graph results for more than 35 biological and environmental variables.

Input loads

The complex simulator and its fragment (described in the paper) is autonomous software designed for IBM compatible computers. Taking into account volume limits for the paper, our main attempts were directed to illustrate the new opportunities we can propose to potential users. Best if the illustrations show something not modeled yet in traditional thermoregulatory models. In our opinion,

circadian effects look like a not bad example for simulation. In this case, input loads are: $T^{o}{}_{A}(t)$, $h_A(t)$, $v_W(t)$, and $L_I(t)$. Changes of $L_I(t)$ and $T^{o}{}_{A}(t)$ were provided according to formulas (*) and (**).

Special window of the user interface (see Figure 2) provides the user by actualization of such data like factors determining day/night dynamics of light intensity, as well as of constants determining dynamics of air temperature.

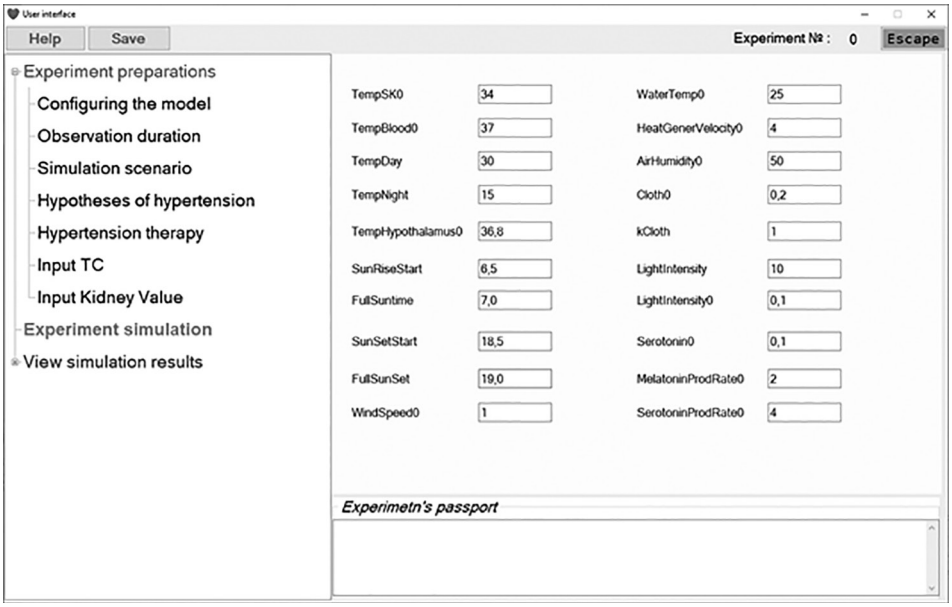

Fig.2. User interface fragment: special window for setting initial data for processing the thermoregulatory model

Main simulation results and discussion

As the reader can see in Fig.3, in this simulation, both air humidity (50%) and the wind speed (1 m/sec) are stable. It was assumed that the durations of night and day times are equal. Night time light intensity assumed to be for 100 times less than it is during the day time. Both for the sun rise time and for the sun set time is set 0,5 hour.

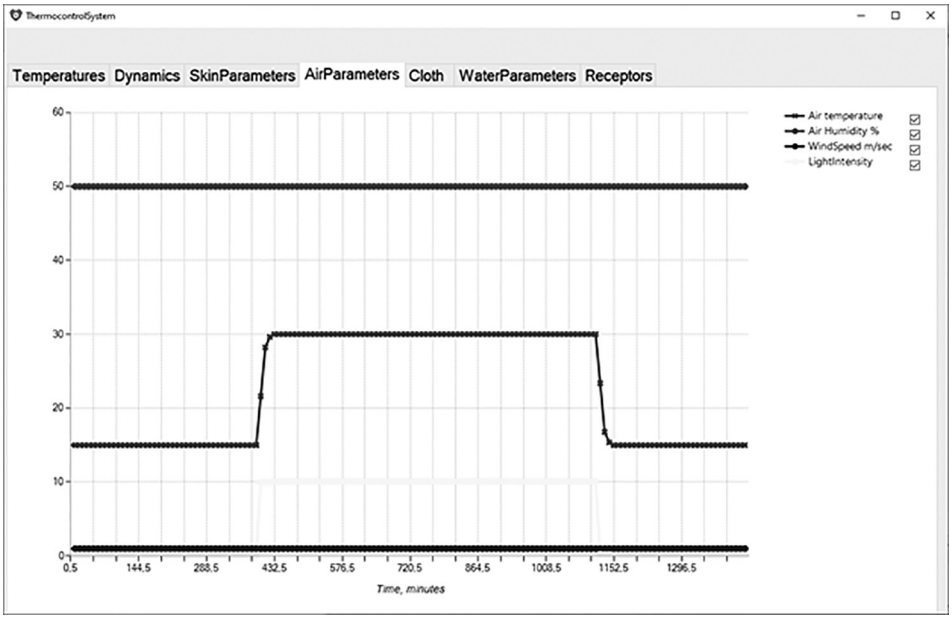

Fig.3. Environmental parameters day/night dynamics: Input data

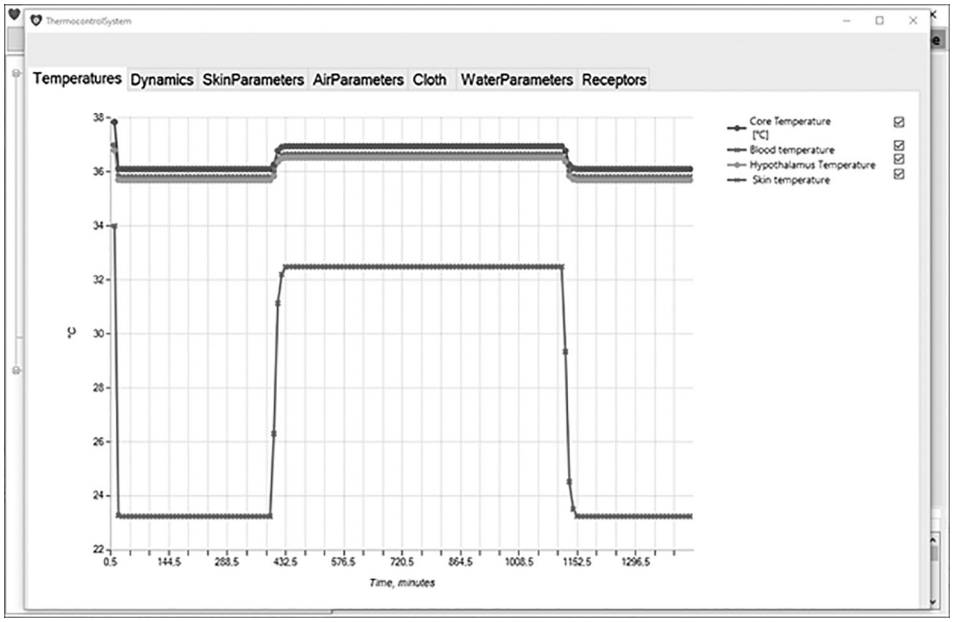

Fig.4. Body temperatures day/night (circadian) dynamics: Output data

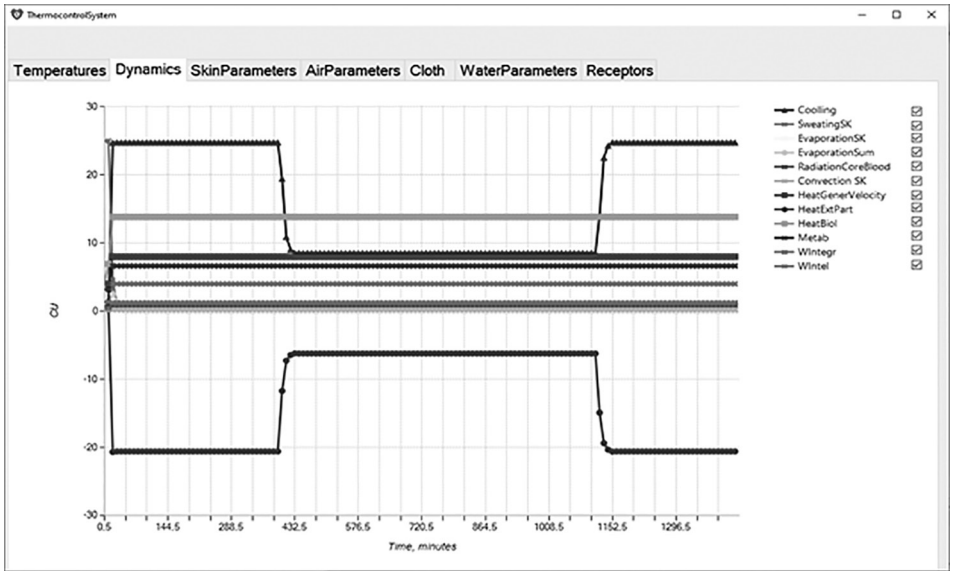

Fig.5. Day/night (circadian) dynamics of body parameters related to thermoregulation: Output data

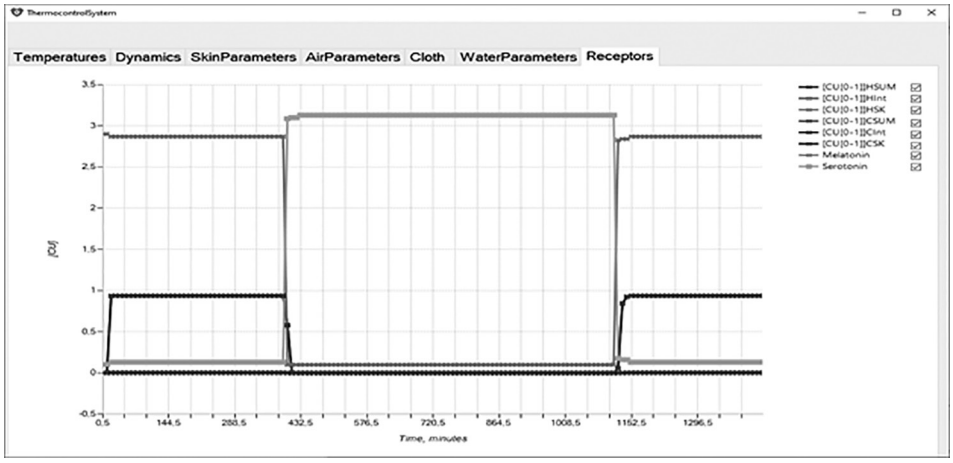

Fig.6. Day/night (circadian) dynamics of thermal receptors, serotonin, and melatonin: Output data

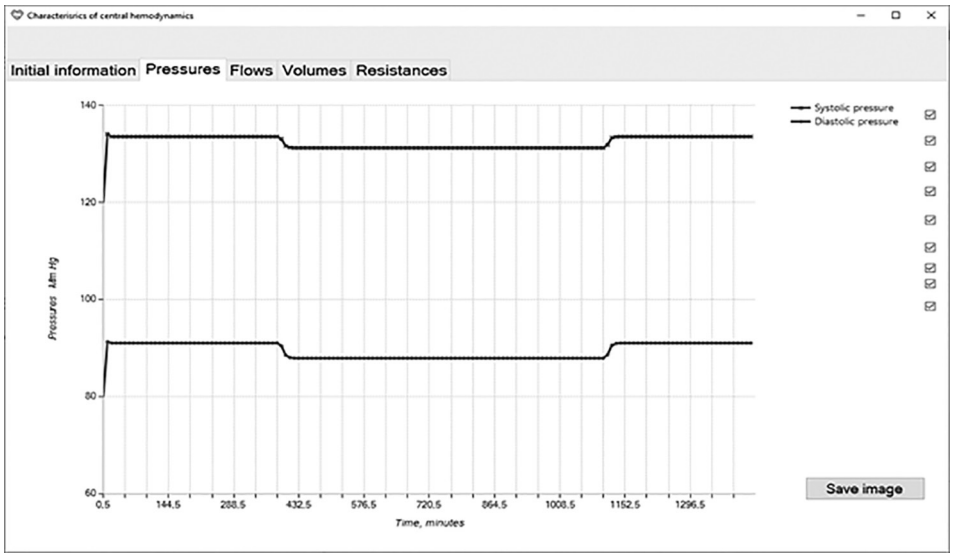

Fig.7. Day/night (circadian) dynamics of systolic and diastolic arterial pressures: Output data

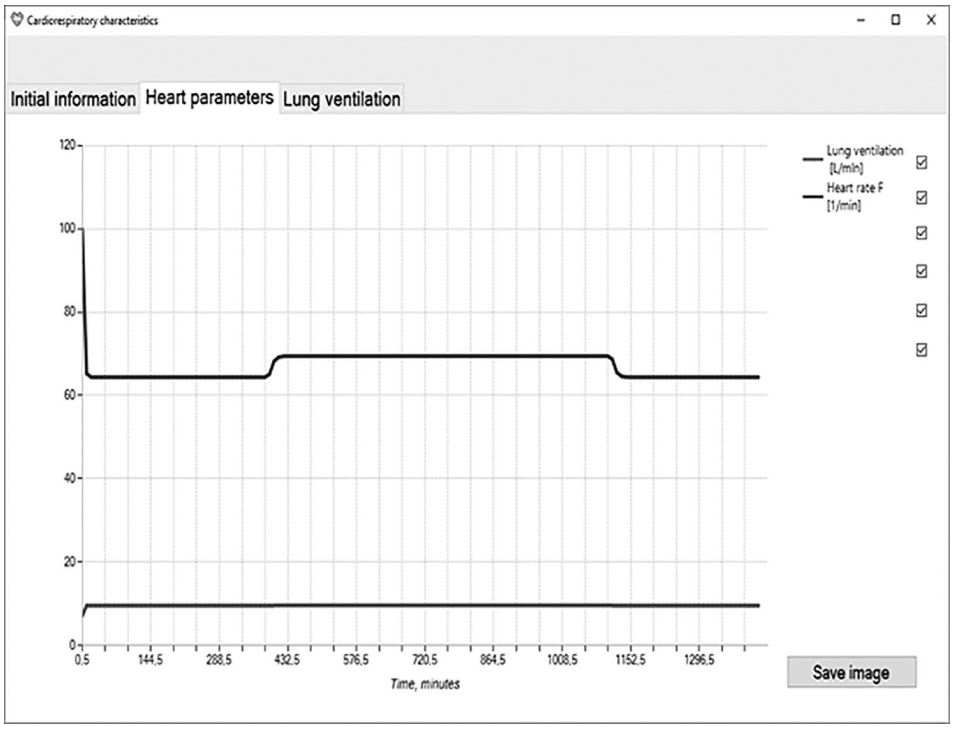

Fig.8. Day/night (circadian) dynamics of heart rate and lung ventilation: Output data

Output data are presented by temperatures in the core, blood, hypothalamus, and skin (Fig.4), by 12 characteristics concerning heat, and cooling (Fig.5), by dynamics of thermal receptors (heat and cold), and blood concentrations of serotonin and melatonin hormones (Fig.6), by dynamics of systolic and diastolic pressures (Fig.7), and at last, by dynamics of heart rate and lung ventilation (Fig.8).

Fig.4 illustrates day/night alterations of temperatures in modeled body areas while the last two illustrations obviously show day/night alterations of pressures and heart rate.

Certainly, our simulator yields much more output data concerning blood circulation parameters, baroreceptors, and chemoreceptors activities, and dynamics of main endocrine hormones modulating not only the state of CVS but also the state of those body structures that concern functionality of thermoregulatory sys-tem. We do not present this additional data for two reasons. The first one is

already mentioned above – the paper volume. The second reason is concerned with the "raw" state of the MT model. It is not able yet to realistically simulate dynamics. Values of several constants and variables are included in the model in conventional units only. We plan to advance it when all component models will be created and integrated into the complex simulator of human PSS.

Conclusion

In order to extend the potentials of the PC-based simulator of the human physiological super-system (PSS), a special quantitative model of the human thermoregulatory system (MT) is created and previously tested for specific scenarios.

Currently, MT is functioning with models of cardiovascular and lung systems. MT describes thermoregulatory responses to alterations of both external environmental physical characteristics and internal biological characteristics. Algorithms provide designing of scenarios including simulation of either short-time or long-time (hours or days) observations. Input data include different combinations of environmental variables (air or water temperature, air humidity, wind or water flow speed, light intensity, infrared radiation) for a naked or wear human, as well as for given dynamics of biological characteristics (rate of heat production including its components associated with metabolism and ATP molecules leasing during mental and physical activities). Human body is presented by a core, blood, and a skin compartments. Skin and lung evaporation are under hypothalamic control based on afferent impulse patterns from internal, and skin heat and cold receptors. Dynamic output data include blood, hypothalamic, and skin temperatures, hemodynamic parameters like heart rate, cardiac output, regional blood flows, vascular resistances, blood pressures, and regional blood volumes. Serotonin and melatonin concentrations modulating biological heat production rate are associated with light's day/night intensity. Currently, the PC-based simulator is autonomous soft-ware to be used both for educational purposes and for providing of special computer research. In a near future, this simulator has to be widened by models of kidneys, and a mechanism of liver-pancreas interaction.

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