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Human cardiovascular system with heat failure under baroreflex control (numerical model)

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Numerical simulation of the cardiovascular haemodynamics has become a useful tool of the surgeon who diagnoses cardiovascular diseases and recommends the way of their medical treatment. The numerical model of the cardiovascular system of pulsating type imitating the electrochemical and mechanical activity of heart muscle and being under a short-term baroreflex control has been implemented to diagnose more accurate a patient's haemodynamic profile. This model enables thus the simulation of the haemodynamic reaction due to the baroreflex response at various perturbations in the circulatory system. The haemodynamic behaviour of the cardiovascular system in the cases of the heart failure as a consequence of the left-ventricular hypocontractility, with the usage of the heart support device and the circulatory failure caused by haemorrhagic shock are demonstrated.

Key words: human cardiovascular system, baroreflex control, haemorrhagic shock, heart support, numerical simulation

1. Introduction

The baroreflex short-term control in the cardiovascular system (being in progress in seconds to minutes) is a regulatory feedback loop which keeps the blood pressure at an adequate level to perfuse the body tissues and thus guarantees the oxygen delivery to all tissues. The control loop involves the autonomic nervous system (ANS) with parasympathetic and sympathetic paths, the chemoreceptors, and the baroreceptors. The baroreceptors are specialized nerve cells behaving as stretch receptors located in the wall of the aortic arch and carotid arteries and monitoring the arterial circulation.

The blood pressure fluctuations are perceived by the baroreceptors which send signals to the ANS regulating the sympathetic and parasympathetic (vagal) system output. The sympathetic and vagal activities continually interact, i.e. a decrease in vagal activity accompanies an increase in sympathetic activity and vice versa. Consequently, in response to the drop in blood pressure the sympathetic activity increases, while the vagal activity decreases. The sympathetic tone enhances the heart rate (the

chronotropic effect), increases the myocardial contractility (the inotropic effect) and induces the vasoconstriction of the blood vessels, i.e. increases the resistance. The vagal tone slows down the heart rate and dilates the blood vessels, i.e. decreases the resistance. The blood pressure increase has the opposite effect, i.e. the vagal tone rises and the sympathetic tone decreases. Moreover, the baroreflex control affects the heart rate, the vascular compliance, resistance and myocardial contractility. The baroreflex controls various perturbations in the cardiovascular system and induces thus such haemodynamic changes that restore the system to its initial physiological state.

2. Model of the human cardiovascular system and its mathematical description

A real cardiovascular system has been compartmentalized for the modelling purposes (figure 1). The circulatory system is represented by four compartments of a pulsating heart: the right and left atria (in the CVS scheme they are numbered *1*, *7*), the right and left ventricles (*2*, *8*) and by ten vascular segments of the pulmonary and systemic circuits connected with the heart chambers in series. The pulmonary circuit consists of pulmonary artery (*3*), arteries and arterioles (*4*), capillaries (*5*) and veins (*6*). The systemic circulation occurs through the aorta (*9*), arteries, arterioles (*10*), capillaries (*11*) and veins (*12*), head arteries (*13*) and head veins (*14*).

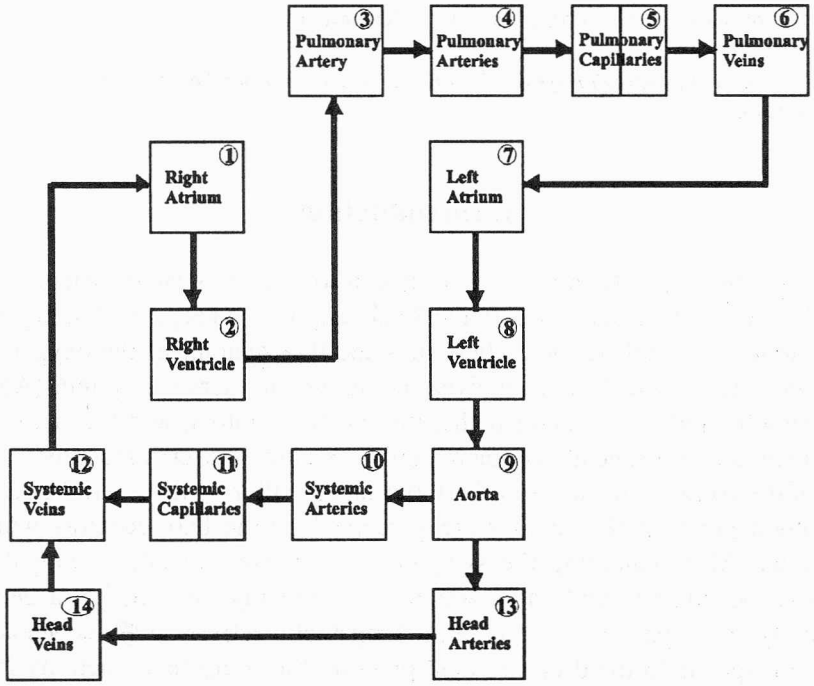

Fig. 1. Scheme of the cardiovascular system (CVS)

The model suggested provides one-dimensional flow of incompressible blood through the network of elastic blood vessels. The heart segments are considered to be made of anisotropic and viscoelastic incompressible material.

The behaviour of the cardiovascular system is described by its haemodynamical variables, i.e. the blood pressure, volume, and by the cardiovascular parameters such as compliances and resistances in corresponding compartments. The blood inertia and physicochemical variables such as the cardiac action potential, the calcium concentration are included enabling the more precise performance of the cardiovascular system.

2.1. Chemical and mechanical performance of heart

The pressure pulsations in the heart compartments working as a pressure–volume pumps are due to calcium kinetics playing a key role in developing the action potentials of the cardiomyocytes. In order to describe the membrane potentials during cardiac action and the changes in the concentration of calcium ions transported through the cardiomyocyte membrane, the Beeler–Reuter equations [1] are used. These equations are applied to heart segments as shown in figure 1. The potentials of atrial and ventricular action V_{m_i} [mV] are determined by four major ionic currents, i.e. I_{Na_i} – the fast inward sodium current, I_{s_i} – the slow inward calcium current, I_{K_i} – the time-independent potassium current, I_{x_i} – the time-dependent outward potassium current, set up in the following equation

$$\frac{dV_{m_i}(t)}{dt} = -\frac{1}{C_m}\left(I_{\mathrm{Na}_i}(t) + I_{s_i}(t) + I_{K_i}(t) + I_{x_i}(t) - I_{st_i}(t)\right), \tag{1}$$

where C_m [μF/cm^2] is the capacitance of myocardial tissue, I_{st_i} [μA/cm^2] is the stimulating current.

The ionic currents in the i-th segments are formulated in a following way:

$$I_{\mathrm{Na}i}(t) = \left(g_{\mathrm{Na}i} m_i^3(t) h_i(t) j_i(t) + g_{\mathrm{NaC}i}\right)\left(V_{mi}(t) - E_{\mathrm{Na}}\right), \tag{2}$$

where

$$E_{\mathrm{Na}} = \frac{RT}{ZF}\ln\frac{c_{\mathrm{Na}_o}}{c_{\mathrm{Na}_{in}}}, \tag{3}$$

$$I_{si}(t) = g_{si} d_i(t) f_i(t)\left(V_{mi}(t) - E_{si}(t)\right), \tag{4}$$

$$E_{s_i}(t) = -82.3 - 13.0287 \ln c_{\mathrm{Ca}_i}(t), \tag{5}$$

$$I_{\mathrm{K}_i}(t) = 0.35 g_{\mathrm{K}_i}\left[\frac{4\mathrm{e}^{0.04\left(V_{m_i}(t)+85\right)}-1}{\mathrm{e}^{0.08\left(V_{m_i}(t)+53\right)}+\mathrm{e}^{0.04\left(V_{m_i}(t)+53\right)}} + \frac{0.2V_{m_i}(t)+23}{1-\mathrm{e}^{0.04\left(V_{m_i}(t)+27\right)}}\right], \tag{6}$$

$$I_{xi}(t) = 0.8 g_{xi} x_i(t) \frac{e^{0.04(V_{mi}(t) + 77)} - 1}{e^{0.04(V_{mi}(t) + 35)}}. \tag{7}$$

The symbols in the equations (1)–(7) for the calculation of the ionic currents are as follows:

$c_{\mathrm{Na}ex}$, $c_{\mathrm{Na}in}$ – extracellular and intracellular sodium ion concentrations [mM],
c_{Ca_i} – intracellular calcium ion concentration [mM],
F – Faraday constant (96 485 C/M),
g_{Na_i}, $g_{\mathrm{Na}C_i}$ – sodium channels conductance [mS/cm^2],
g_{s_i} – calcium channels conductance [mS/cm^2],
g_{K_i}, g_{x_i} – potassium channels conductance [mS/cm^2],
E_{Na}, E_s – the Nernst equilibrium potential of Na^+, Ca^{2+} ions [mV],
m_i, h_i, j_i, d_i, f_i, $x_i = y$ – gating variables determining channel [1],
R – gas constant (8.3143 J/(mole·K),
T – temperature (T = 310 K),
Z – ionic charge number, for Na^+ Z = 1 [1].
The gating variables are defined by the differential equation

$$\frac{dy_i(t)}{dt} = \frac{y_{\infty i}(t) - y_i(t)}{\tau_{y_i}(t)},$$

where

$$y_{\infty i}(t) = \frac{\alpha_{y_i}(t)}{\alpha_{y_i}(t) + \beta_{y_i}(t)}$$

denotes the steady state fraction of open channels,

$$\tau_{y_i}(t) = \frac{1}{\alpha_{y_i}(t) + \beta_{y_i}(t)}$$

is the relaxation time and α_{y_i}, β_{y_i} are the rate constants dependent on the transmembrane potential V_m being determined by fitting the data from voltage clamp experiments [1], [2].

The intracellular concentration of calcium ions c_{Ca_i} [mM] is calculated from the following differential equation

$$\frac{dc_{\mathrm{Ca}_i}(t)}{dt} = -10^{-7} I_{s_i} + 0.07\left(10^{-7} - c_{\mathrm{Ca}_i}(t)\right). \tag{8}$$

The concentration of the Ca^{2+} ions regulates the force of myocardial contraction, i.e. the pressure generated by the contracting heart muscle. The pressure in the heart

atria and ventricles is described by the equation derived from the general energy and entropy balance (see [3], [4])

$$p_i(t)=\frac{2h_iE_i}{r_i}\left(\frac{V_i(t)}{V_{oi}}-1\right)+\frac{2h_ik_{\text{chem},i}}{r_i}c_{\text{Ca}_i}(t), \qquad i=1,2,7,8, \tag{9}$$

in which the parameters in the i-th segment are as follows:

c_{Ca_i} – calcium concentration [mM],

E_i – Young's elasticity module [J/m^3],

h_i – thickness of atrial and ventricular walls [m],

$k_{\text{chem},i}$ – parameter of chemical energy release [J/m^3·M],

p_i – atrial or ventricular pressure [Pa],

r_i – radius of atria or ventricles [m],

V_i – atrial or ventricular volume [m^3],

V_{oi} – initial atrial or ventricular volume [m^3].

The first term on the right-hand side of eq. (9) describes the elasticity of the heart, and the second term describes the transformation of chemical energy into mechanical one.

2.2. Haemodynamic performance of the cardiovascular system

The pressure changes in the pulmonary artery and aorta are described by

$$p_i(t)=\frac{1}{C_i}\left(V_i(t)-V_{\text{res}_i}\right)+\eta_i\frac{dV_i(t)}{dt}, \qquad i=3,9, \tag{10}$$

and in other segments of the pulmonary and systemic circuit by

$$p_i(t)=\frac{1}{C_i}\left(V_i(t)-V_{\text{res}_i}\right), \qquad i=4,5,6,10,11,12,13,14, \tag{11}$$

where C_i [m^3/Pa] denotes the compliance, V_{res_i} [m^3] is the residual volume of the i-th segment, and η_i [Pa·s/m^3] represents the wall viscosity.

The blood flow F_{ij} [m^3/s] between the i-th and the j-th segments is determined by the balance of the momentum in the following forms:

- $i = 2, 8, j = 3, 9$ (flow between the ventricles and output arteries, i.e. the pulmonary artery and the aorta)

$$\frac{dF_{ij}(t)}{dt}=\frac{1}{L_i}\left[p_i(t)-p_j(t)-R_{ij}F_{ij}(t)-\xi^2\frac{\rho F_{ij}^2(t)}{2A_i^2(t)}\right], \tag{12}$$

where R_{ij} [Pa·s/m^3] is the hydrodynamical resistance, L_i [Pa·s^2/m^3] characterizes the blood inertia, ξ [1] is the coefficient of blood inertia, A_i [m^2] is the flow area, HR

[beats/min] is the heart rate, t_{VS}, [s] is the duration of the ventricular systole, $\rho = 1.062 \cdot 10^3$ [kg/m^3] is the blood density;

- $i = 3, 9, 9, j = 4, 10, 13$ (flow between arterial segments)

$$\frac{dF_{ij}(t)}{dt} = \frac{1}{L_i}\left[p_i(t) - p_j(t) - R_{ij}F_{ij}(t)\right]; \tag{13}$$

- $i = 1, 7, 6, 12, 13, j = 2, 8, 7, 1, 14$ (flow between the atria and ventricles, pulmonary and systemic veins and atria, between head artery and head veins)

$$F_{ij}(t) = \frac{p_i(t) - p_j(t)}{R_{ij}}; \tag{14}$$

- $i = 4, 10, 11, j = 5, 11, 12$ (flow between arterial segments, capillaries and veins)

$$F_{ij}(t) = \frac{(p_i(t) - p_j(t))V_i^2(t)}{R_{ij}V_{\text{res}_i}^2}. \tag{15}$$

The volume changes in all segments of the cardiovascular system are determined by the balance of mass

$$\frac{dV_i(t)}{dt} = F_{ij}(t) - F_{jk}(t), \qquad i, j, k = 1, 2, ..., 14, \tag{16}$$

F_{ij}, F_{jk} are the blood flows: F_{ij} describes the blood flow to the i-th and then to the j-th segment, while F_{jk} the blood flow from the j-th to k-th segment.

2.3. Baroreflex control

The modelling equations of baroreflex control adopted from [5] describe:

- the baroreceptor activity v_a [1]* which is affected both by the blood pressure and the rate of pressure change

$$v_a = k_1(P - P_o) + k_2\frac{dP}{dt}, \tag{17}$$

where P [mm Hg] is a mean value of blood pressure, P_o [mm Hg] is the threshold below which the activation of the baroreceptor does not occur and k_1 [1/mm Hg], k_2 [s/mm Hg] are constants;

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*Beeler and Reuter [1] consider one baroreceptor, in this article the model of two baroreceptors (aortic and carotid) is described, their activities are summed up: $v_a = 0.3v_{aAo} + 0.7v_{aHA}$.

- sympathetic activity ν_s [1]

$$\nu_s = \nu_s^o - k_s \nu_a + k_s^r \left| \sin\left(\pi f_r t + \Delta\phi_s^r\right) \right|, \tag{18}$$

where f_r [1/s] is the respiratory frequency, ν_s^o [1], k_s [1], k_s^r [1] , $\Delta\phi_s^r$ [1] are constants;

- parasympathetic activity ν_p [1]

$$\nu_p = \nu_p^o - k_p \nu_a + k_p^r \left| \sin\left(\pi f_r t + \Delta\phi_p^r\right) \right|, \tag{19}$$

where ν_p^o [1], k_p [1], k_p^r [1], $\Delta\phi_p^r$ [1] are constants.

The baroreflex control of the heart rate HR [beat/min] results from the balance between the sympathetic and vagal activity:

$$HR = HR^o \left[1 + \frac{\nu_s}{HR^o \left(c_1 + c_2 \nu_s\right)} \right] \left[1 - \frac{\nu_p}{HR^o \left(c_3 + c_4 \nu_p\right)} \right]. \tag{20}$$

HR^o [beat/min] is the resting heart rate, c_1, c_2, c_3, c_4 [min/beat] are constants.

Consequently, if the heart rate changes, the cardiac cycle duration is altered. The following dependencies of the atrial systole (t_{AS} [s]) or ventricular systole (t_{VS} [s]) duration on the heart rate determined by the regression method applied to the clinical data are used

$$t_{AS} = 0.4056 - 0.0083(HR) + 8 \cdot 10^5 (HR)^2 - 3.6631 \cdot 10^{-7} (HR)^3 + 6.3447 \cdot 10^{-10} (HR)^4, \tag{21}$$

$$t_{VS} = 0.404 - 0.0016(HR). \tag{22}$$

3. Results of numerical simulation of heart failure

In order to show the capability of the model prescribed, a haemodynamic behaviour of the human cardiovascular system in normal (uncontrolled) state and in the state of failure (controlled) was simulated. The heart failure was induced by a sudden decrease in the contractility of a left ventricle evoked by reducing the calcium ions' concentration. In table 1 and figure 2 the simulation results are compared with the results from SimBioSys model (educational software used in medical training) [6]. Due to the baroreflex control (see table 1) the heart rate increases, end systolic and end diastolic volumes increase, stroke volume decreases, cardiac output decreases, venous return decreases, mean arterial blood pressure and mean pulmonary pressure

increase, mean right-atrial pressure decreases, systemic and pulmonary vascular resistances increase, oxygen consumption increases, and myocardial efficiency decreases.

Table 1. Simulated haemodynamic profile in the case of heart failure due to the left-ventricular hypoconcractility

Haemodynamic parameters	IT CVS		SimBioSys	
	Normal	After	Normal	After
Heart rate [beat/min]	75	96.3	73	96
Cardiac output [dm^3/min]	5.67	5.14	6.28	5.17
Venous return [dm^3/min]	5.63	5.13	6.3	5.4
Left-ventricle end diastolic volume [cm^3]	141.0	159.4	140.0	180
Left-ventricle end systolic volume [cm^3]	65.3	106.0	56.0	127
Stroke volume [cm^3]	75.6	53.4	86.0	54.0
Ejection fraction [1]	0.54	0.33	0.6	0.3
Left-ventricular stroke work [g·m]	87.4	63.2		
Right-ventricular stroke work [g·m]	10.1	9.4		
Mean arterial pressure [torr]	93.2	97.2	95	98
Mean right-atrial pressure [torr]	5.2	3.6	5.8	4.5
Mean left-atrial pressure [torr]	8.2	10.2	13.0	32.7
Mean filling pressure [torr]	7.4	7.5	8.0	10
Mean pulmonary artey pressure [torr]	15.0	16.5		
Systemic vascular resistance [dyn·s/cm^5]	1240.7	1457.0	1140.0	1440
Pulmonary vascular resistance [dyn·s/cm^5]	95.0	97.9		
Venous return resistance [Pa·s/m^3]	$3.2 \cdot 10^6$	$6.0 \cdot 10^6$	$2.79 \cdot 10^6$	$8.1 \cdot 10^6$
Myocardial oxygen consumption [J/beat]	3.1	2.25		
Myocardial efficiency [%]	38.9	35.4		

The decrease in the concentration of calcium ions (see figure 2a) causes the left-ventricular and aortic pressure drop (see figure 2b) which is balanced by baroreflex compensation, thus the pressure restores nearly to its initial level. In figure 2c, d showing the pressure–volume loop simulated by the IT CAS model and by SimBioSys model the ventricular volume shift is clearly visible.

4. Results of the numerical simulation of the heart failure support

The rotary pump is located between the left atrium and aorta (figure 3). The working characteristics of the rotary pump at 2000 rot/min is supposed to be

$$F_{RP} = 2.43 + 0.12 P_{RP} - 0.00084 P_{RP}^2, \tag{23}$$

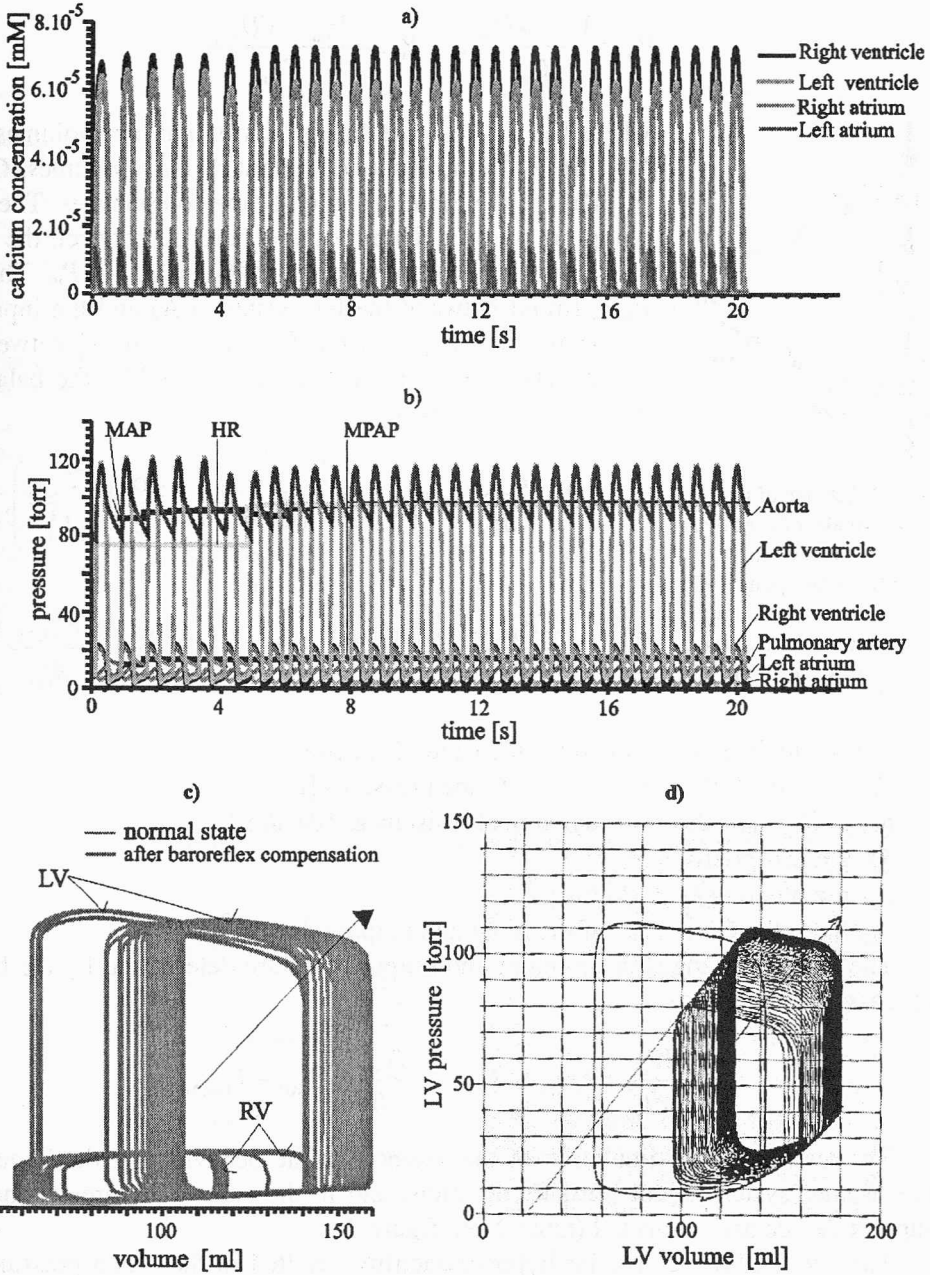

Fig. 2. Simulation of left-ventricular contractility suddenly decreased

where F_{RP} [dm^3/min] is the flow through the rotary pump and P_{RP} [torr] is the rotary pump pressure. The pressures in the input and output cannulas of the rotary pump, i.e. P_{in}, P_{out} [Pa], are determined from the following relations

$$P_{in} = \frac{V_{in} - VU_{in}}{C_{in}}, \quad P_{out} = \frac{V_{out} - VU_{out}}{C_{out}}, \tag{24}$$

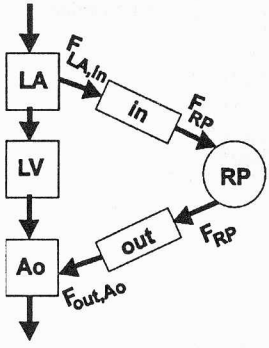

Fig. 3. Scheme of the part of the model of cardiovascular system with the rotary pump (RP). LA – the left atrium, LV – the left ventricle, Ao – the aorta

where V_{in}, V_{out} [m^3] are the input or output tube volumes, VU_{in}, VU_{out} [m^3] are the input and output residual volumes, C_{in}, C_{out} [m^3/Pa] are the input and output tube compliances. The rotary pump pressure is given by the difference between the output and input cannula's pressure, i.e. $P_{RP} = P_{out} - P_{in}$. The flow $F_{LA,in}$ [m^3/s] between the left atrium (LA) and the input tube of the rotary pump and the flow $F_{out,Ao}$ [m^3/s] between the output tube and aorta (Ao) are determined by the balance of momentum in the form:

$$\frac{dF_{LA,in}}{dt} = P_{LA} - P_{in} - R_{LA,in} - \left(\zeta^2 \frac{\rho}{2}\right)\left(\frac{F_{LA,in}^2}{A_{in}^2}\right), \tag{25}$$

$$\frac{dF_{out,Ao}}{dt} = P_{out} - P_{Ao} - R_{out,Ao} - \left(\zeta^2 \frac{\rho}{2}\right)\left(\frac{F_{out,Ao}^2}{A_{out}^2}\right). \tag{26}$$

The symbols used in equations (25) and (26) denote:

P_{LA}, P_{in}– the left atrium and input tube pressures [Pa],

$R_{LA,in}$, $R_{out,\,Ao}$ – the hydrodynamical resistances [Pa·s/m^3],

ζ – the loss coefficient [1],

ρ – the blood density [kg/m^3],

A_{in}, A_{out} – the flow areas of the input and output tubes [m^2].

The volume changes in the input and output tubes are determined by the balance of mass

$$\frac{dV_{in}}{dt} = F_{LA,in} - F_{RP}, \quad \frac{dV_{out}}{dt} = F_{RP} - F_{out,Ao}. \tag{27}$$

The results of the simulation of the haemodynamic behaviour of the human cardiovascular system under normal conditions and in the case of failure for the heart support device are connected (table 2 and figure 3).

The sudden left-ventricular hypocontractility resulted in the blood pressure drop (the systemic hypotension) and in both end-systolic and diastolic ventricular volumes shift to the right. The work of rotary pump increases mean arterial pressure, and the left-ventricular pressure–volume area decreases. As a consequence the baroreflex response the heart rate increases, elevating moderately the mean arterial pressure and shifting the pressure–volume area of both ventricles leftwards (see figure 4).

Table 2. Haemodynamic profile under physiological and pathological conditions, baroreflex influence included

Haemodynamic parametrs	Normal	Failure	Support	Baroreflex
Heart rate [beat/min]	75	75	75	96.3
Cardiac output LV [dm^3/min]	5.7	4.3	1.5	1.8
Cardiac output RV [dm^3/min]	5.7	3.8	4.9	5.3
Venous return [dm^3/min]	5.6	3.8	4.5	5.5
Stroke volume LV [cm^3]	76.0	56.8	19.7	18.8
Stroke volume RV [cm^3]	75.6	50.1	65.5	55.4
Ejection fraction LV [1]	0.54	0.37	0.17	0.17
Ejection fraction RV [1]	0.53	0.32	0.44	0.40
Left-ventricular stroke work [g·m]	87.6	46.4	22.4	22.1
Right-ventricular stroke work [g·m]	10.1	4.6	4.4	4.6
Mean arterial pressure [torr]	93.0	68.3	88.2	91.3
Systemic vascular resistance [dyn·s/cm^5]	1230.6	1321.1	1467.4	1259.9
Pulmonary vascular resistance [dyn·s/cm^5]	95.8	97.5	102.8	98.1
Myocardial oxygen consumption [J/beat]	3.1	2.0	1.1	1.0
Myocardial efficiency [%]	38.9	34.0	22.5	22.3

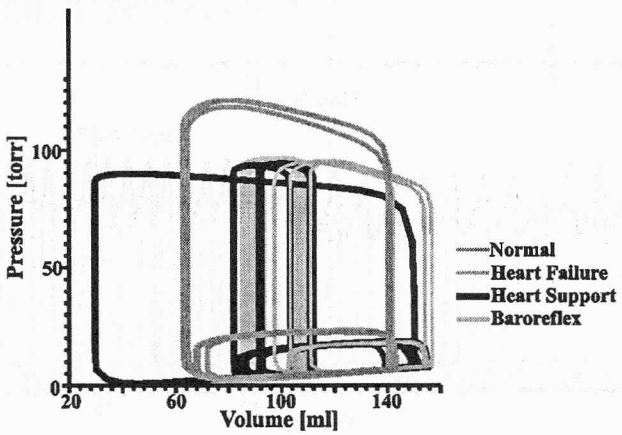

Fig. 4. Pressure–volume loop of the left and right ventricles simulated in normal state at the left-ventricular hypocontractility, with the heart support and under baroreflex control

5. Results of the numerical simulation of the haemorrhage

The haemodynamic changes during the haemorrhaging in a cardiovascular system caused by the baroreflex response depend on the blood loss. Four classes of the haemorrhagic shock are distinguished (see, e.g.[7], [8]). The simulation results depicted in figure 5 and summarized in table 3 demonstrate that the haemodynamic responses correspond to the shock of the second class which means that the blood

volume reduction is in the range of 15–30%, i.e. 750–1500 cm^3 of the total blood volume in the cardiovascular system.

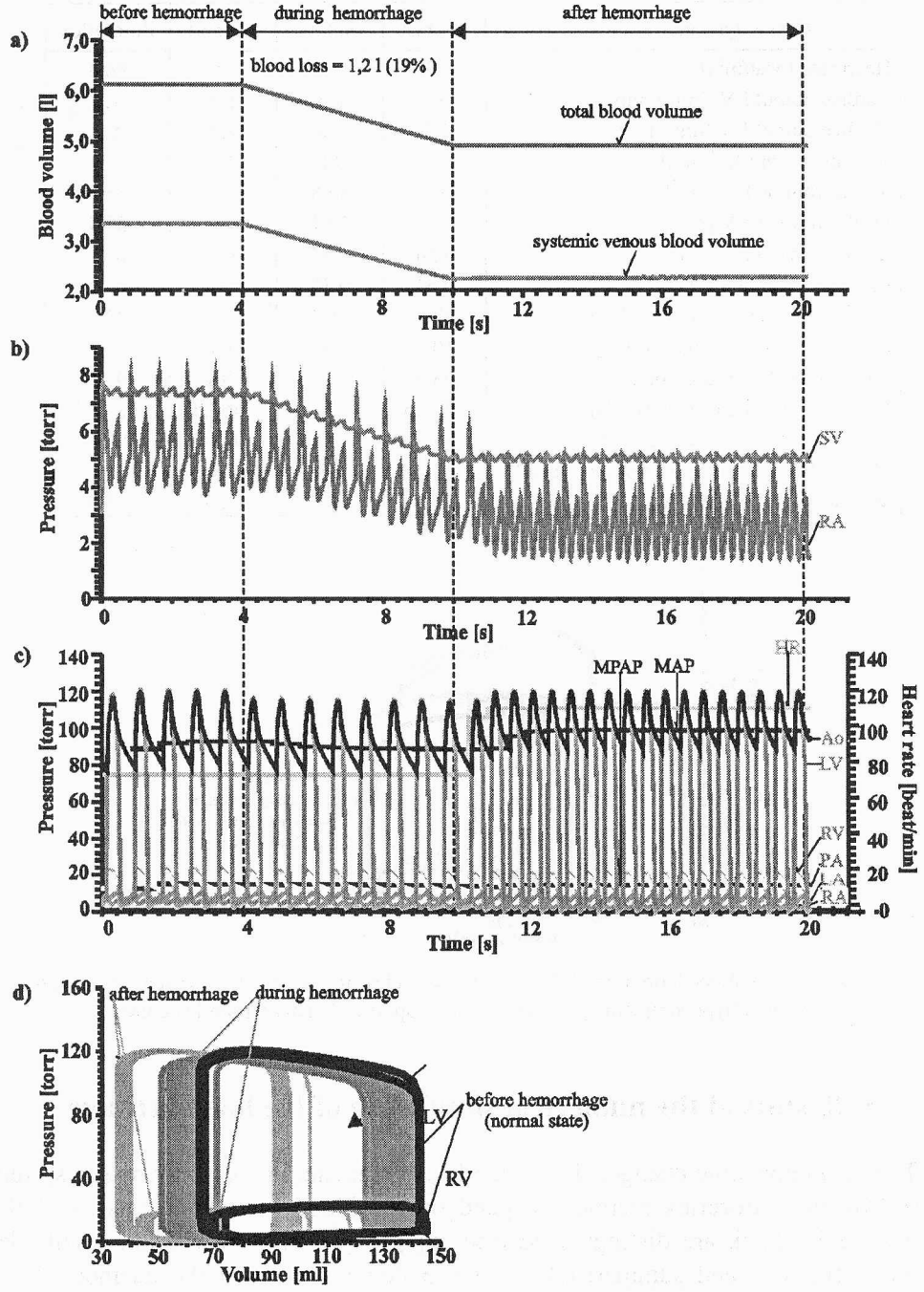

Fig. 5. Simulation of the haemorrhagic shock of the second class

The following haemodynamic changes accompanying this shock (during simulation of venous haemorrhage the decrease in total blood volume was 1.2 dm^3, i.e. 19%, see figure 5a) are restricted:

- systemic venous (SV) and right-atrial (RA) pressures (figure 5b),
- arterial (Ao, PA) and ventricular pressures (LV, RV) (figure 5c),
- left- and right-ventricular both systolic and diastolic volumes and thus their stroke volume (figure 5d),
- cardiac output and venous return (table 3),
- myocardial oxygen consumption (table 3).

Table 3. Simulated haemodynamic profile in the normal state, during and after haemorrhage

Haemodynamic parameters	Normal state	During haemorrhage	After haemorrhage
Heart rate [beat/min]	75	106	111
Cardiac output [dm^3/min]	5.6	5.4	6.2
Venous return [dm^3/min]	5.6	4.0	6.3
Left-ventricle end-diastolic volume [cm^3]	141.0	123.4	91.0
Left-ventricle end-systolic volume [cm^3]	65.3	50.8	35.2
Stroke volume [cm^3]	75.6	72.6	55.8
Left-ventricular stroke work [g·m]	87.4	80.2	70.3
Right-ventricular stroke work [g·m]	10.5	9.0	8.8
Mean arterial pressure [torr]	93.2	88.6	99
Mean right-atrial pressure [torr]	5.2	3.5	2.5
Mean filling pressure [torr]	7.4	5.1	5.0
Mean pulmonary artery pressure [torr]	15.0	13.8	14.2
Systemic vascular resistance [dyn·s/cm^5]	1240.7	1250.6	1245.9
Pulmonary vascular resistance [dyn·s/cm^5]	94.9	107.2	101.6
Myocardial oxygen consumption [J/beat]	3.26	2.84	2.34
Myocardial efficiency [%]	28.8	30.3	32.0

The blood loss triggers a compensatory baroreflex control evoking vasoconstriction (especially of the arterioles), i.e. an increase of the systemic and pulmonary vascular resistances (table 3) and tachycardia, i.e. an increase of the heart rate (table 3 and figure 5c).

After this compensation the systolic pressures in the aorta, pulmonary artery and in both ventricles restore to the initial level, while the diastolic aortic and pulmonary artery pressures increase, i.e. the pulse pressure decreases and thus the values of the mean aortic pressure (MAP) and mean pulmonary artery pressure (MPAP) increase (figure 2c). Because of the tachycardia the cardiac output and venous return are elevated. The vasoconstriction of blood vessels diminishes, i.e. their resistance decreases.

If the blood loss is less than 15% of the blood volume the arterial blood pressure does not record measurable changes. The venous pressure is a better indicator of the haemorrhage severity.

6. Conclusion

Mathematical models of the cardiovascular system are useful for a deeper understanding of complex processes occurring in the heart and blood vessels in the normal and/or pathophysiological state. The proposed fourteen-segment haemodynamic model with the baroreflex control enables the simulation of the electrochemical and mechanical heart actions and the haemodynamic response of the whole cardiovascular system in the case of various perturbations. The pressure waves, volume changes in all compartments and flows between them during cardiac cycles and complex haemodynamic profile evaluated from these variables under various conditions can be used for diagnosing a patient illness. The simulation results are compatible with the clinical data published and are further tested in clinical practice.

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Bibliography

[1] BEELER G.W., REUTER H., *Reconstruction of the action potential of ventricular myocardial fibres*, J. Physiol. (Lond.), 1977, 268, pp. 177–210.

[2] ŠIMURDA J., ŠIMURDOVÁ M., BRAVENÝ P., CHRISTÉ G., *Control of cardiac performance by Ca-turnover*, Molecular and Cellular Biochemistry, 1996, 160/161, pp. 5–12.

[3] PŘEVOROVSKÁ S., MARŠIK F., *The influence of the membrane transport on the heart mechanical activity*, Engineering mechanics '99, Svratka, May 17–20, 1999, pp. 575–580.

[4] PŘEVOROVSKÁ S., MARŠIK F., MUSIL J., *Pressure generation by chemical reaction in the human cardiovascular system*, Engineering Mechanics, 1997, Vol. 4, No. 3, pp. 147–154.

[5] SEIDEL H. , HERZEL A., *Modelling heart rate variability due to respiration and baroreflex*, [in:] *Modelling the Dynamics of Biological Systems*, ed. by E. Mosekilde and O.G. Mouritsen, Springer-Verlag, Berlin, 1995, pp. 205–229.

[6] *Heart failure simulation observations*, http://www.bme.jhu.edu/courses/580.421/hw5/solution.html.

[7] NEUMANN S., *Modelling acute hemorrhage in the human cardiovascular system*, A dissertation bioengineering, University of Pensylvania, 1996.

[8] WILSON I.H., BASKETT O.J.F., *The diagnosis and treatment of haemorrhagic shock*, Practical Procedures, Issue 1, Article 4, 1992.