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Research Article

Model of human cardiovascular system with a loop of autonomic regulation of the mean arterial pressure

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Abstract

A model of human cardiovascular system is proposed which describes the main heart rhythm, the regulation of heart function and blood vessels by the autonomic nervous system, baroreflex, and the formation of arterial blood pressure. The model takes into account the impact of respiration on these processes. It is shown that taking into account nonlinearity and introducing the autonomous loop of mean arterial blood pressure in the form of self-oscillating time-delay system allow to obtain the model signals whose statistical and spectral characteristics are qualitatively and quantitatively similar to those for experimental signals. The proposed model demonstrates the phenomenon of synchronization of mean arterial pressure regulatory system by the signal of respiration with the basic period close to 10 seconds, which is observed in the physiological experiments. J Am Soc Hypertens 2016;10(3):235–243. © 2016 American Society of Hypertension. All rights reserved.

Keywords: Arterial pressure; autonomic regulation; baroreflex; cardiovascular system.

Introduction

The studies of complex multicomponent systems of the real world are usually accompanied by consistent improvement of the model. Starting with the simple block diagrams that only qualitatively describe the behavior of the real system, the models are developed and grow more complex as new knowledge becomes available. These more complicated models offer both qualitative and quantitative description of the observed phenomena. Simulation is of particular importance in physiology, biology, and medicine, where the possibilities of experimental invasive research and the range of acceptable impacts on the object under investigation are fundamentally limited.

The modeling of the human cardiovascular system (CVS) is one of the current problems in physiology. Physiological systems are usually complex and nonstationary. They are characterized by a network structure with a number of interacting elements. Currently, only few mathematical models of the CVS are known that take into account its autonomic regulation.[1–4] However, the necessity of modeling a large number of interacting functional elements in these articles has led to the simplification and linearization of the model description of such elements.

In particular, such reduction has resulted in modeling the system of baroreflex regulation of mean arterial pressure (AP) with the help of a first-order linear delay differential equation.[1–4] Such models of CVS regulation are unable to demonstrate stable self-sustained oscillations.[5] They exhibit only the regimes of forced oscillations under the influence of noise and other system elements impacting them. However, a number of researches point to an autonomous and self-oscillating nature of the system of mean AP

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baroreflex regulation based on the results of physiological experiments.[6–9] Similar conclusions were previously obtained in our experimental studies of regulatory system synchronization by respiration.[10–14]

Besides, on the basis of the results of in vitro studies in animals, the autonomous mathematical model for the system of baroreflex regulation of mean AP in mammals has been proposed in Ringwood and Malpas[15] in the form of a first-order nonlinear delay differential equation. The authors have shown that this model can demonstrate stable self-sustained oscillations with a characteristic period of about 10 seconds in humans.

In the present article, we propose a model of the CVS taking into account the nonlinear properties of the system of mean AP baroreflex regulation. The features of the proposed model are investigated by comparing the results of statistical and spectral analysis of the model heart rate variability (HRV) with the experimental data and a certain model proposed in Kotani et al,[4] which incorporates the systems of CVS regulation. Using the model and experimental signals, we investigate the phase synchronization of 0.1 Hz rhythms of mean AP baroreflex regulation system by respiration with the linearly changing frequency.

Material and Methods

Design of this study was approved by the Ethics Committee of the Saratov Research Institute of Cardiology (Saratov, Russia) in 2015.

Model of Cardiovascular System Autonomic Regulation

We propose a mathematical model of CVS which describes the main heart rhythm, the influence of autonomic nervous system on heart rate (HR), baroreflex regulation of mean AP, and formation of AP and takes into account the impact of respiration on these processes. The structure of the model is shown in Figure 1.

The proposed dynamical model includes four first-order differential equations:

$$\frac{d\varphi(t)}{dt} = \frac{1}{T_0} f_s(t) f_p(t), \tag{1}$$

$$\frac{dp_{dia}(t)}{dt} = -\frac{p_{dia}(t)}{R(t)C}, \tag{2}$$

$$\varepsilon \frac{d\overline{p}(t)}{dt} = -\overline{p}(t) + f(\overline{p}(t-\theta)) + k_1 B(t), \tag{3}$$

$$\frac{dc(t)}{dt} = -\frac{c(t)}{\varepsilon_c} + k_2 v_s(t-\theta_c). \tag{4}$$

The operation of heart sinoatrial node is described by Equation (1),[16] where $\varphi(t)$ is the phase of the heartbeat,

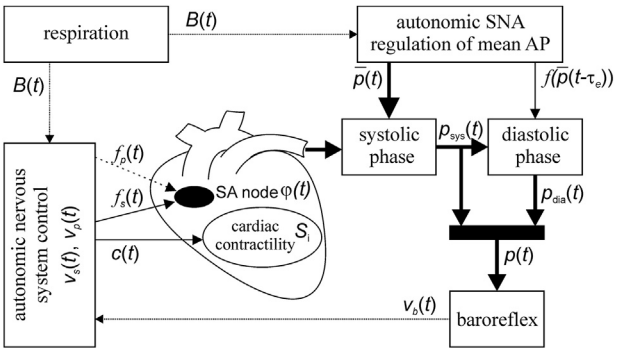

Figure 1. Schematic representation of the model. Impacts of vagus and sympathetic nervous activity (SNA) and AP are shown by dashed, solid, and bold lines, respectively. Other impacts are shown by dots. The activities of sinoatrial (SA) node and the system of mean arterial AP regulation are modeled too. AP, arterial pressure.

T_0=0.55 second is the period of denervated HR, and $f_s(t)$ and $f_p(t)$ are the influence of the sympathetic and parasympathetic divisions, respectively. In the absence of regulatory influences (denervation of the heart), f_s=f_p=1 and sinoatrial node generates periodic pulses with the period T_0. Under the influence of autonomic nervous system, the frequency of the HR is modulated and variability appears.

The dynamics of blood pressure in the systolic phase is modeled as:

$$p_s(t) = D_{i-1} + S(t)\frac{(t-T_{i-1})}{T_s}\exp\left(1-\frac{(t-T_{i-1})}{T_s}\right) + k_3\overline{p}(t) \tag{5}$$

where D_{i-1} is the magnitude of diastolic pressure at the end of the previous cardiac cycle, T_{i-1} is the duration of the previous cardiac cycle, $\overline{p}(t)$ is mean AP, and $S(t)$ is the cardiac contractility[3,4] expressed as follows:

$$S(t) = S'(t) + (S_a - S'(t))\frac{S'(t)^{n_1}}{S_a^{n_1} + S'(t)^{n_1}}, \tag{6}$$

where $S'(t)$=S_0+$k_4c(t)$+k_5T_{i-1} depends on the concentration of sympathetic agent noradrenalin (4) in the myocardium.[4]

In accordance with Seidel and Herzel,[3] $p_s(t)$ increases rapidly to a maximum value $p_s^{\max}$, which is reached after a fixed time T_s=0.125 second from the moment of the current heartbeat i used as a subscript of variables in the formulas. Blood pressure in the diastolic phase $p_d(t)$ relaxes from the maximal value achieved in systole phase $p_{d0}(t_i + T_s) = p_s^{\max}$ until the next heartbeat. This relaxation is described by windkessel effect caused by inertial properties of blood vessels (2). In Equation (2), C is a constant that determines the elasticity of the aorta and $R(t)$ is the

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