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MODEL OF CENTRAL VENOUS HEMODYNAMICS

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Abstract: One of the main problems of hemodynamics modelling is reproducing of the regulation mechanisms of a local level. In practice, the inclusion of an active muscular component of the vascular tissue of the arterial and venous channels leads to the nonlinear models and autowave process models. That is why the description of a local automatism and vegetovascular reactions turns out to be the most complex aspect of modelling. In this work, the model of automatic regulation of the cardiac rhythm coming from requirements of the best co-ordination of the heart, as a generator, and the vascular system, as load, in the electrical analogy as arteriovenous reverberator is presented. Such a model allows to set the problem of experimental study of the features of the arteriovenous mechanical interaction under the different pathological changes of the cardiac rhythm.

Key words: hemodynamics, modelling, electrical analogy, arteriovenous reverberator

The fundamental works in the field of the cardiovascular system (CVS) research [1,2] point out the significance of the mechanical features in the CVS behaviour. However in these works integral and systematic view on the CVS functioning which allows to study the known phenomena from the united positions is absent. Such a point of view must help in posing the priority and perspective problems of theoretical and practical study of the circulation regulation processes. In this article, an attempt to build the system model of central venous hemodynamics being the key section in the system circulation control is made.

It is known that the value of the cardiac surge is dependent on the values of blood inflow to the right heart [1]. The blood flow along the central venous system (hollow veins) presents a process of spreading of a pulse pressure wave. Note that the baroreceptors in the vascular channel have a specific role in the neurogenic contour of regulation [2]. They are transmitters of information on both the average pressure and the pulse pressure when the pulse wave spreads. Besides, the muscular component of the vessel wall has the capability to actively respond upon the mechanical strain and exchanges energy with the pulse wave. Further the CVS is assumed to be nonlinear hydromechanical and an electric analogy is used to describe it.

This analogy is effective because of the following reasons. Firstly, its use makes it possible to interpret the processes not only in flow but also in signal form. Secondly, the signal interpretation allows to consider certain parameters of process of blood motion in the CVS as information to control this process. The electrical analogy is suitable since its use allows to describe the dependence between the flow parameters, the shape of the vessels sections, diameters of vessels and rheological properties of the blood when model building. It is realised by means of the nonlinear complex resistances depending on voltages and currents in circuits. Thereby, the electrical analogy allows to cybernetically interpret the mechanical processes on the base of clear physical motivation.

The correspondence of the mechanical and electrical values according to the chosen analogy [4,5] is shown in Table 1.

Table 1. Correspondence of the mechanical and electrical values.

Generalised term	Hydrodynamic values	Electrical values
Potential	*p* (pressure)	*U* (electro moving power)
Volume	*V* (volume of liquid)	*Q* (charge)
Flow	*dV/dt* (liquid consumption)	*I* (current)
Resistance	*R* (hydrodynamic resistance)	*R* (active resistance)
Inductance	*L* (hydrodynamic inertial inductance)	*L* (electromagnetic inductance)
Capacity	*C* (resilience)	*C* (electrical capacity)

Herewith any enough long part of the vascular channel area can be presented as a long line (system with distributed parameters). The description in the form of the telegraphic equation (1) is known for such lines, where all values correspond to Table 1

$$\frac{\partial^2 U}{\partial x^2} = LC\,\frac{\partial^2 U}{\partial t^2} + RC\,\frac{\partial U}{\partial t}\,. \qquad (1)$$

The analysis of such a description for one of the largest areas of the vascular channel of the great circle involving the heart, the thoracic and abdominal aortas, the femoral and radial arteries allows to find the following: for an adult person of an average height (170 cm) the length of this part is approximately 110-130 cm. Since the velocity of spreading of a pulse wave in these arteries is 4-6 m/sec and the heart rate (HR) is normally 60-75 pulse/min it may be suggested that the considered part of the vascular channel corresponds to the so called a "quarterwave" transformer (λ/4) or a "reactive train". The lines of such sizes (λ/4) have a set of specific characteristics described in the literature on electric circuits [8].

One of the most important features for this case is the fact that such a transformer co-ordinates the general peripheral resistance (GPR) of an active type with the internal resistance of the heart generator (left ventricle of the heart) which can be also considered as active.

The relationship between these resistances is as follows

$$Z_{LV} = \frac{Z_X^2}{Z_{GPR}} \qquad (2)$$

where Z_{LV} is a resistance of the left ventricle of the heart;
Z_{GPR} is an active GPR;
Z^2_X is a characteristic resistance of a long line.

The correspondence between the area length and the value of λ/4 is dependent on the wavelength λ, which in turn obviously depends on both the velocity of the pulse wave spreading along the vessels and the HR. From the point of view of optimal co-ordination of the heart generator with the vascular channel (being, probably, a result of an evolutionary development of the warm-blooded CVS), it is possible to assume that changing of the pulse wave spreading velocity (PWSV) requires a proportional HR change to preserve co-

ordination. Really, the optimal co-ordination of the heart and the vessels ensures the transmission of the maximum mechanical power from the heart generator to the vascular channel, thus a load on the cardiac muscle under the given value of blood flow is minimized.

So any disturbance caused by the output of a "reactive train" beyond the λ/4 limit due to change of a wavelength (for instance, because of vessels elasticity change) requires corresponding change of the HR to recover an optimal mode of power transmission. Such a compensatory reaction must have a local automatism of limited nature [3]. A proposed regulator should work as follows. For instance, when the vessel tonus is changed under the increased load, the elasticity of vessels decreases due to the intramural musculature strain. It results in the PWSV increasing which is proportional to $1/\sqrt{LC}$, L being a line inductance and C being a capacity [8]. If the HR is constant, then a wavelength increases. To hold the same value of the wavelength it is necessary to scale up the HR and it really happens. (The HR is known to increase under load.) This effect can not be explained by the known compensatory reactions. Thus a described regulator model in the form of the central arteriovenous reverberator (AVR) is proposed. The first half of the AVR is a part of aorta and the second half is a central venous channel part. Including a part of the central arterial channel in this reverberator provides a described proportional change of the HR. This change of the HR is caused by the following fact: the increasing PWSV reduces the time of wave run through the reverberator. It leads to the accelerated starting a next cardiac cycle by the sinoatrial node of the right auricle. On the contrary, when the PWCV reduces then the cardiac rhythm slows down. To study this regulator mechanism the authors have suggested a model of the reverberator consisting of the aorta, the system arteries which have mechanical contact points with the large central veins, the central venous part (vena cava superior and vena cava inferior), and the right auricle.

Modelling of such a reverberator is based on numerical solution of Eq. (1) for the venous channel. The arterial channel is described by a proportional delay element. This is possible since the PWSV in arteries is 5-6 times as high as in veins.

For computer realisation, a numerical scheme of the second order was built. The equation (1) may be written in the following form [6]

$$U_{i,j+1} = 2U_{i,j} - U_{i,j-1} + \frac{t}{LC}\left(\frac{U_{i+1,j} - 2U_{i,j} + U_{i-1,j}}{h^2} - RC\frac{U_{i,j} - U_{i,j-1}}{t}\right). \qquad (3)$$

The parameters in this equation have the following average values. Active resistance R equals $5\cdot10^5$ N·sec/m^5, inductance L equals approximately $0.26\cdot10^7$ kg/m^4, capacity C equals $1\cdot10^{-6}$ m^5/N.

When undertaking numerical experiments it was assumed that the venous part of this reverberator is disclosed from the input and a load element with a small internal resistance is located at the output. A pulse of voltage in the form of trapezoids of different amplitudes and duration was applied to the input.

The following results were obtained. Firstly, the PWSV of the considered channel meets to the condition of equality between the area length and λ/4 and depends on the nonlinear parameters of the channel (capacity and inductance). Secondly, a process of spreading and absorbing wave depends greatly on its form and amplitude.

These model characteristics allow to apply it for the description of the joint compensatory reactions to the different pathological processes in the CVS, i.e. to processes associated with the changes of general vascular tension, central venous pressure, general peripheral resistance, cardiac surge and rhythm. It also allows to develop actions aimed at central hemodynamics functions reconstruction and normalization in clinic practice.

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МОДЕЛЬ ЦЕНТРАЛЬНОЙ ВЕНОЗНОЙ ГЕМОДИНАМИКИ

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Известно, что одной из основных проблем моделирования в гемодинамике является воспроизведение механизмов регуляции на локальном уровне. Учет активного мышечного компонента сосудистой ткани артерий и вен приводит к нелинейным моделям. Поэтому описание механизмов регуляции кровообращения и вегетососудистых реакций связано с построением сложных биомеханических моделей. В данной работе предлагается модель автоматической регуляции сердечного ритма, исходя из требований наилучшего согласования сердца как генератора и сосудистой системы как нагрузки с помощью электрической аналогии гидродинамических величин. Предполагается, что любой достаточно длинный участок сосудистого русла может быть описан с помощью телеграфного уравнения. Анализ такого описания для одного из наиболее крупных участков сосудистого русла большого круга кровообращения, включающего сердце, аорту (грудную и брюшную), бедренные и лучевые артерии, позволяет связать скорость распространения пульсовой волны и частоту сердечных сокращений. Строится также модель, включающая только венозное русло. Численное моделирование на ЭВМ позволяет описать ряд явлений, связанных с механическими взаимодействиями при различных патологических изменениях ритма сердца. Библ. 8.

Ключевые слова: гемодинамика, моделирование, электрическая аналогия, артериовенозный ревербератор

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