

Estimation of Arterial Stiffness Based on Analysis of Heart Rate

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Abstract

A hemodynamic model of human cardiovascular system has been proposed. This model allowed us to establish the relationship between index of arterial stiffness and pulse rate variability.

A new approach to estimation of arterial stiffness by assessing the relative spectral power of pulse rate variability and heart rate variability (beat-to-beat R-R intervals) was suggested.

A group of volunteers, consisting of 20 healthy people aged from 20 to 65 years was examined. The difference in magnitude of total spectral power of pulse rate variability and total spectral power of heart rate variability decreases with increasing age of subjects, which corresponds to the model evaluation and can be explained by age-related changes in arterial stiffness.

Comparative estimation between the proposed diagnostic index and arterial stiffness index, which defined by contour analysis of digital volume pulse, was presented. Correlation coefficient was 0.92; $p < 0.02$.

1. Introduction

Analysis of heart rate variability (HRV) is widely used as a noninvasive tool for assessing the state of autonomic regulation in physiological research and medical diagnostics [1-3].

HRV can be determined by processing of biosignals, which contain information about heart rate. HRV is one of the most common indicators obtained by means of RR intervals analysis of ECG. Upon the registration of peripheral pulse waves by sensors of blood pressure, as well as rheographic or plethysmography sensors, the duration of time intervals between two consecutive systolic peaks of pulse waves is analyzed. In a number of studies [4-7], when comparing the results of the analysis of HRV and pulse rate variability (PRV), some differences are revealed. It can be explained by temporal changes of hemodynamic parameters of the vascular system controlled by vascular regulation.

In this paper an attempt is undertaken to establish a relationship between HRV and PRV characteristics defined in the frequency domain and the characteristics of the vascular system in order to obtain a diagnostic index of the vascular system, which may be used to estimate arterial stiffness.

2. Modeling

Simulation of hemodynamic processes in the cardiovascular system allows determining patterns of the peripheral pulse wave propagation along the arterial bed [8].

To describe the propagation of the pulse wave (PW) in arteries we used equations based on the model of "elastic chamber", proposed by O. Frank. As a first approximation, to describe the hemodynamic processes it can be assumed that the excess pressure is distributed along the elastic tube. According to the Navier-Stokes equations we can describe hemodynamic processes as the following set of equations [8]:

$$\begin{aligned} -\frac{\delta P}{\delta x} &= Q \cdot R_g + \frac{\rho}{A} \frac{\delta Q}{\delta t} \\ -\frac{\delta Q}{\delta x} &= P \cdot G_g + \frac{3\pi r^2}{2Eh} \frac{\delta P}{\delta t}, \end{aligned} \quad (1)$$

where:

x – axial coordinate,

Q – flow,

P – hydraulic pressure,

$R_g = \frac{8\mu}{\pi r^4}$ – hydraulic resistance due to the dynamic

blood viscosity, here

μ – blood viscosity, r – radius of artery;

ρ – blood density,

G_g – leakage flow

E – Young's modulus,

h – arterial wall thickness.

Distribution of PW in space and time is given by the solution of equations (1) with the following assumptions: $G_g = 0$, due to the fact that the vessel is a flexible tube with no leakage, and we neglect the second term of the first equation, since the blood is close to an absolutely incompressible fluid. Dependence of pressure on time and coordinate is obtained from the following:

$$P(t, x) = \int_0^t \frac{x \sqrt{3\pi r^2 R_g}}{2E(t-\tau) \cdot h} e^{-\frac{3\pi x^2 r^2 R_g}{8(t-\tau) \cdot E(t-\tau) \cdot h}} \cdot P_0(\tau) d\tau \quad (2)$$

where

$P_0(\tau)$ – pressure in the vessel at the point with coordinate

$x = 0$,

$E(t)$ – change of Young's modulus in time

τ – constant of integration.

For the pressure $P_0(\tau)$, which simulates the aortic ejection as an exponential pulse, we use the following description:

$$P_0(t) = \begin{cases} \sum_{n=0} P_0 \left(1 - e^{-\frac{t-n \cdot t_{b-b}}{T_1}} \right), & n \cdot t_{b-b} \leq t \leq t_s + t_{b-b} \cdot n \\ \sum_{n=0} P_0 \left[e^{-\frac{t-n \cdot t_{b-b} - t_s}{T_2}} - e^{-\frac{t-n \cdot t_{b-b}}{T_1}} \right], & t_s + n \cdot t_{b-b} \leq t \leq (n+1) \cdot t_{b-b} \end{cases}$$

where:

T_1 – time constant of rising edge,

T_2 – time constant of falling edge,

t_s – duration of systolic ejection,

t_{b-b} – pulse beat-to-beat interval,

P_0 – pressure amplitude.

Time fluctuations of arterial stiffness, which depend on the level of vasoactive substances, make changes in the temporal structure of PRV. This happens due to changes in the durations of pulse beat-to-beat intervals, according to alterations in pulse wave transit time (PWTT) that leads to changes in PRV.

The process of vasoactive substances allocation occurs periodically and is influenced by autonomic regulation. By setting the temporal parameters of changes in the concentration of vasoactive substances [9], and assuming that these changes modulate Young's modulus time variations, we can investigate the impact of arterial stiffness alterations on PRV.

3. Results of Modeling

Figure 2 shows the dependence between PWTT and alterations of arterial stiffness for radial and femoral arteries. Model parameters: blood viscosity $5 \cdot 10^{-3}$ Pa·s, radius of femoral artery $r = 2,4$ mm, arterial wall thickness $h = 0,5$ mm, for radial artery – 1,6 mm и 0,43 mm accordingly, length of the investigated area for radial artery – 117 mm, femoral – 127 mm, Young's modulus for both arteries in normal – $4 \cdot 10^5$ Pa [8].

Thus, with an increase of arterial stiffness PWTT decreases.

Figure 2 shows the dependence between the total spectral power (TP) of pulse beat-to-beat intervals and alterations of arterial stiffness. These dependencies are obtained by changing the duration of vasoactive pulses.

The total spectral power decreases with increasing Young's modulus (or increasing arterial stiffness) and decreases with reducing duration of vasoactive pulses.

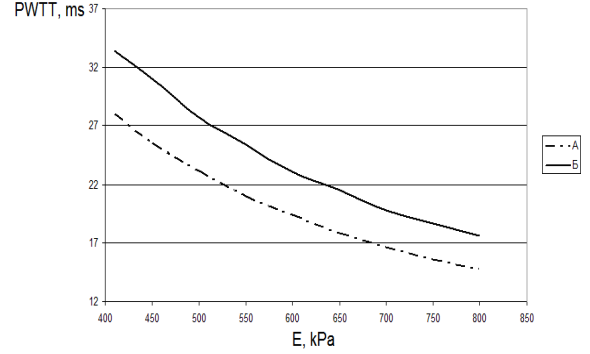


Figure 1. Dependence between PWTT and alterations of arterial stiffness for femoral (A) and radial (B) arteries.

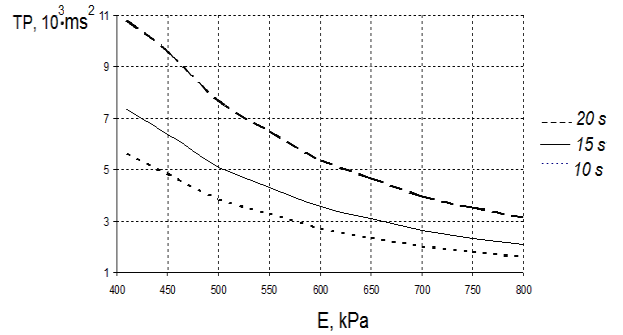


Figure 2. Dependencies between the total spectral power (TP) of pulse beat-to-beat intervals and alterations of Young's modulus.

4. Materials and Methods

Two groups of volunteers, consisting of 20 healthy people aged from 20 to 65 years were examined. The first group consisted of 10 people aged 20 to 30 years and the second group consisted of 10 people aged 55 to 65 years.

Simultaneous recording of ECG and distal arterial pulse was carried out for 5 minutes for each volunteer. ECG signal was recorded in the first standard lead. The signal of distal arterial pulse was obtained by finger photoplethysmography sensor. To register biosignals we used automated system "Koros-300" ("New Devices", Russia), which allows simultaneous record of ECG and distal arterial pulse, and transmit data to a personal computer with a sampling frequency of 500 Hz.

5. Results of experiment

The experimental results are given in Table 1.

Comparison between PRV and HRV recognizes differences in their spectral characteristics. Ratio LF/HF for pulse beat-to-beat intervals was significantly lower ($p < 0,05$), than for the R-R intervals; the spectral power in the range of high frequencies for PRV was significantly greater than the power in HRV, and the power at low frequencies less for PRV compared with HRV.

Table 1. Spectral characteristics of HRV and PRV.

Group	Age	Total spectral Power, ms ²		LF/HF	
		PRV	HRV	PRV	HRV
A	23±1,5	1.71·10 ⁶ ±0.1·10 ⁶	1.48·10 ⁶ ±0.1·10 ⁶	2.2± 0.25	2.9± 0.3
B	58±2	1.53·10 ⁶ ±0.1·10 ⁶	1.49·10 ⁶ ±0.1·10 ⁶	3.6± 0.12	3.8± 0.11

The total spectral power of pulse beat-to-beat intervals exceeds the total spectral power of RR intervals ($p < 0,05$).

Stiffness index (SI) and the diagnostic index D was obtained as a relative difference between the total spectral power of pulse beat-to-beat intervals and the total spectral power of RR intervals is described in Table 2.

Table 2. Diagnostic index D and stiffness index (SI) for two examined groups.

Group	Age	SI, m/s	D, %
A	23±1.5	6.9±0.1	6.8±0.4
B	58±2	11.5±0.35	1.1±0.2

6. Discussions

The proposed model relates the hemodynamic parameters of the vascular system, the parameters of vasoactive substances allocated by the autonomic nervous system to pulse rate variability. Analysis of this model demonstrates that PWTT is a nonlinear function of arterial stiffness. Exploration of established relationship shows that the same change of the arterial stiffness at low values of Young's modulus leads to greater deviations of PWTT than for change at large values of Young's modulus.

Thus rigid arterial wall variations of PWTT caused by the influence of vasoactive substances on the endothelium are smaller than in the case of elastic arteries.

As can be seen from the Figure 2 the value of the total spectral power of pulse beat-to-beat intervals decreases with increasing Young's modulus of the artery, i.e. with a decrease in elasticity.

Simulation shows that the vascular regulation leads to fluctuations of the PRV at the frequencies corresponding to temporal characteristics of allocation of vasoactive substances.

Thereby the estimation of spectral power of "vascular" component of the PRV may serve as diagnostic index of arterial stiffness.

It is necessary to separate the "vascular" component of PRV during the acquisition and processing of distal arterial pulse. For this purpose it was suggested to use the simultaneous recording of two biosignals – distal arterial pulse and ECG. A measure of the relative differences between characteristics of PRV and HRV may serve as a

diagnostic assessment of the human vascular system.

To compare the results of assessment of the vascular system by determining the diagnostic index D, we used stiffness index (SI). SI is determined by an independent method using the contour analysis of the digital volume pulse [10]. Diagnostic index SI is used as a comprehensive measure of human endothelial system and is considered as an impartial assessment of cardiovascular diseases [10, 11]. Age-related changes of arterial stiffness are clearly associated with appropriate trend of SI [12]. Proposed diagnostic indicator of vascular system D significantly decreased with age ($p < 0.01$). The correlation coefficient of the proposed diagnostic index D and the SI was 0.92 ($p < 0.05$), that allows us to offer D as a promising marker of the vascular system state.

The advantage of the proposed index D is higher sensitivity: for SI in the range 20 - 60 years sensitivity was - 0.15 units per year and for diagnostic index D – 0.315 units per year, therefore using of index D leads to reducing of uncertainties in assessment of the state of vascular system.

The proposed method has satisfactory performance which allows implementation of screening diagnostics. For determination of the diagnostic index it is sufficient to register equal duration of ECG signal and the distal arterial pulse, which are carried out with non-invasive methods by means of widely available monitoring devices.

7. Conclusion

A new approach for estimation of arterial stiffness by assessing the relative spectral power of pulse rate variability and heart rate variability was suggested. This approach is based on complex acquisition and processing of biosignals carrying information about heart rate variability.

Differences in the structure of HRV and PRV may indicate the state of the vascular system and serve as the basis for the development new approaches to noninvasive monitoring of the human arterial system for the purpose of early diagnosis of dangerous and widely spread cardiovascular pathologies such as atherosclerosis and hypertension.

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