Cardiopulmonary Reflex Influence on the System Hemodynamic Rapid Regulation Mechanisms

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Abstract

The aim of this study was to assess the cardiopulmonary baroreflex contribution to the maintenance of arterial pressure stability. Twelve young healthy subjects were examined (mean age – 25.7±4.9 years). Cardiac rhythm and arterial pressure were continuously recorded with the use of finapress technique at rest and in the course of low pressure area receptors deactivation due to lower body negative pressure (-10 mm Hg). The recorded data were processed by special MATLAB program that calculated heart rate (HR) and blood pressure (BP) spectral parameters and also cross-spectral power density between HR and BP.

The obtained results demonstrated that CPBR is involved in the process of vessel tone fine regulation by means of both slow and rapid regulating mechanisms.

1. Introduction

Regulation and optimization of hemodynamic parameters as a reaction on the change of external and internal conditions is governed by several mechanisms connected both with neurogenic and humoral regulation. The raise of blood pressure variability that in normal conditions is smoothed by fluctuations of heart rate (HR) and vascular tone within the Mayer range (near 0.1 Hz) [1,2] can be associated with the increase of cardiovascular events risk [3].

There are several theories concerning the origin of the fluctuations in blood pressure in this frequency range. The most often discussed are the pacemaker theory [4] and the theory of baroreflex resonance [5, 6]. The latter explains the origin of Mayer's waves from a position of the negative feedback control loop. According to this, the arterial reflex plays central role as a transfer function from the input to the output [7]. This theory is supported by most researchers and was proven by a number of original experimental models [8-12] as well as clinical researches. [13, 14].

The importance of arterial baroreflex for the hemodynamic rapid regulation is not doubted [3]. While it is well known that slowly adaptable low-pressure areas reflex participates in regulation of peripheral tone, the volume of circulating plasma, and a number of vasoactive substances, the influence of cardiopulmonary baroreflex (CPBR) on quick arterial pressure regulation is insufficiently studied.

The aim of the presented investigation was assessment of CPBR contribution to the maintenance of arterial pressure stability.

2. Methods

2.1. Registration parameters

Cardiac rhythm and arterial pressure of all subjects were continuously recorded with the use of noninvasive beat-to-beat method (finapress technique, Finometer FMS-Amsterdam). Electrocardiogram and forearm blood flow were also registered.

2.2. The study protocol

Twelve young healthy subjects were included in this study. The mean age of the subjects was 25.7±4.9 years.

After the initial recording in supine position (10 min), signals registration was implemented in the course of the lower body negative pressure application with the use of special chamber. This chamber creates rarefaction –10 mm Hg for deactivation of low pressure area receptors, that makes possible the cardiopulmonary baroreflex assessment. Second recording was carried out 3 minutes after the chamber application, and also lasted 10 minutes.

2.3. Signal processing

Vasomotor component of cardiopulmonary baroreflex was assessed as normalized ratio of forearm blood flow at rest and during the baroreceptors deactivation: (FBF1-FBF2)/FBF1.

The additional parameters of system hemodynamic such as cardiac output and total peripheral resistance were calculated by the modeflow method.

The data recorded from Finometer were then processed by special MATLAB program that calculated

heart rate and blood pressure (BP) spectral parameters and also cross-spectral power density between HR and BP. Calculation was performed within the standard frequency bands VLF, LF and HF. Then the obtained parameters for the two above stages of the examination were compared.

Besides the arterial baroreflex sensitivity value was estimated both by sequential and spectral (transfer function) techniques.

3. Results

3.1. Hemodynamic parameters

All subject had a similar vasomotor component (dynamics of FBF), which ranged between 0.27 and 0.41 NU (mean value 0.33±0.04). Both BP and HR levels did not change significantly as a result of the cardiopulmonary area receptors deactivation: 117.6±5.6 and 120.1±6.2 mm Hg; 71.5±4.6 and 70.2±6.1 min-1. But it should be mentioned what the decrease of stroke volume: from 90.4±7.2 to 88.4±6.6 ml, $p<0.05$ and cardiac output: from 5.8±1,1 to 5.6±0.9 l/min, $p<0.05$ was observed in the course of the cardiopulmonary baroreflex deactivation. At the same time the raise of total peripheral resistance was revealed: from 0,83±0,14 to 0,85±0,13 MU, $p<0.05$.

Figure 1 shows an example of hemodynamic parameters change during the deactivation of cardiopulmonary baroreceptors.

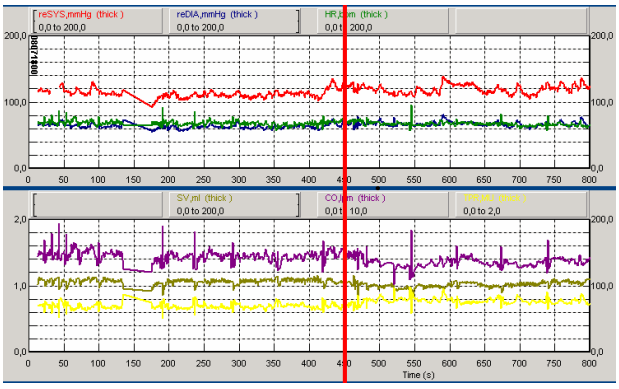

Figure 1. A typical example of hemodynamic parameters change during the deactivation of cardiopulmonary baroreceptors. The red marker indicates the beginning of exposure, resSYS – systolic blood pressure, resDIA – diastolic blood pressure, HR – heart rate, SV – stroke volume, CO – cardiac output, TPR – total peripheral resistance.

3.2. Blood pressure variability

Furthermore, the increase of BP variability in all spectral ranges: in VLF from 4.6±2.5 to 15.4±7.3 mm Hg2, $p<0.01$, in LF from 3.4±1.8 to 6.0±4.0 mm Hg2, $p<0.05$ and in HF from 2.8±0.6 to 4.8±1.8 mm Hg2, $p<0.01$ was noted. Besides the redistribution of spectral power was observed.

A typical example of blood pressure variability and cross-spectral dynamics at rest and during cardiopulmonary baroreceptors deactivation is presented on figure 2.

Relative value of VLF spectral component increased from 41.2±10.1% to 56.1±13.0%, $p<0.01$, together with distinct tendency to the decrease of LF component (31.0±13.1 and 22.2±6.0, $p=0.051$).

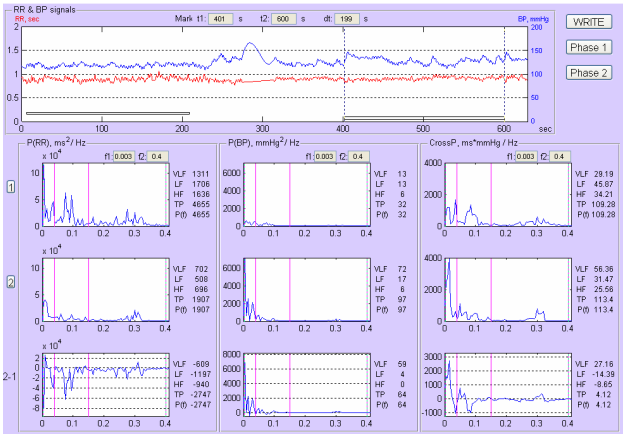

Figure 2. Dynamics of heart rate, systolic blood pressure variability and cross-spectral power density initially and during cardiopulmonary baroreflex deactivation. (The same patient as in Figure 1).

Simultaneously the cross-spectral power also increased most distinctly in the VLF range from 36.8±16.2 to 72.8±19.4 mm Hg*s, $p<0.01$, however only the tendency of mutual fluctuations increase within LF and HF ranges was observed. Cross-spectral power was changed from 42.0±10.0 to 50.0±10.0 mm Hg*s, $p=0.071$ for LF and from 54.0±14.0 to 64.0±12.0 mm Hg*s, $p=0.059$ for HF range.

3.3. Arterial baroreflex

To assess the dynamics of spontaneous arterial baroreflex during the deactivation of cardiopulmonary baroreceptors, we used two techniques: the sequential and spectral methods. Typical example of arterial baroreflex dynamics at rest and during cardiopulmonary baroreceptors deactivation is presented on figure 3.

Both used techniques of baroreflex sensitivity calculation showed similar results. Spontaneous arterial baroreflex was not changed in the course of the cardiopulmonary baroreceptors deactivation: from

23.5±4.2 to 24.9±6.1, p>0.05 for the sequential method and from 22.3±5.4 to 23.5±5.7, p>0.05.for the transfer function method.

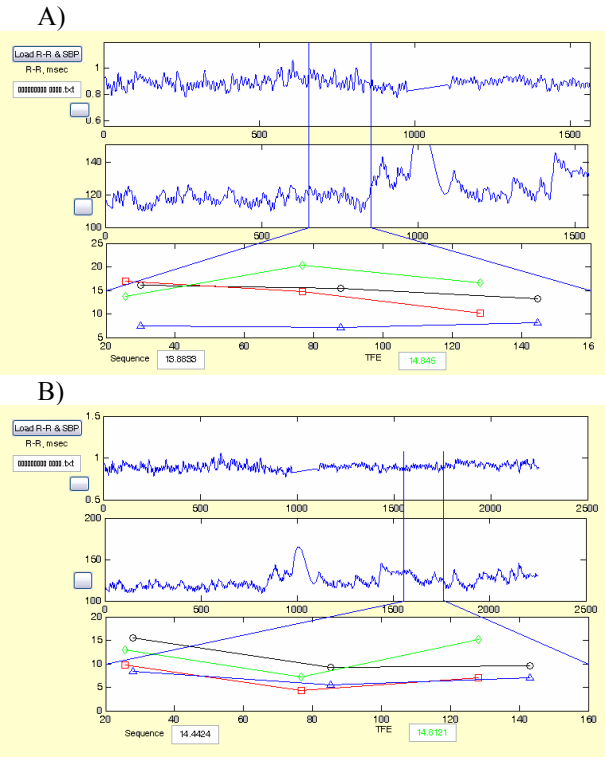

Figure 3. Spontaneous arterial baroreflex initially (A) and in the course of cardiopulmonary baroreflex deactivation (B). Mean reflex values (lower plot) defined by different methods for the selected period. Sequence – sequence method, TFE – transfer function estimation.

4. Discussion and conclusions

Thus, the deactivation of cardiopulmonary baroreflex results in higher blood pressure variability. Besides the increase of variability is observed in all three frequency bands. Maximum changes concern the slow fluctuations of blood pressure. This corresponds to the common concept of the low pressure area reflex role. It is well known that cardiopulmonary baroreflex is slowly adapted and controls the arterial and venous tone vessels, renal sympathetic nerve activity, as well as some of neurohormones plasma concentration. These properties provide long-term hemodynamic shifts through the modulation of sympathetic activity and regulation of circulating plasma volume [15-17]. Perhaps it is related with increase of blood pressure variability within VLF range that was observed after the deactivation of cardiopulmonary area receptors.

However, the results of this study showed that the amplitude of blood pressure fluctuations increased also both in Mayer and in the respiratory frequency bands. This was accompanied by significant increase in heart rate and blood pressure cross-spectrum in VLF range. At the same time, in the low and high frequency ranges only a tendency of cross-spectrum increase was observed. This is consistent with the lack of significant dynamics of spontaneous baroreflex, or rather of its chronotropic response. This was confirmed by two different methods. However, the amplitude of blood pressure fluctuation in the Mayer range reflects, with some restrictions, baroreflex regulation of blood pressure stability and correlates with muscle sympathetic nerve activity [18]. The fact of low-frequency component increase in both heart rate and blood pressure spectra in response to the passive orthostasis was repeatedly described earlier [19-20], that was associated with reduction of venous return and was attributed to the decline of arterial baroreflex.

The obtained data are not entirely consistent with the results of these works. Provided the spontaneous arterial baroreflex (chronotropic response) changes during the cardiopulmonary baroreceptors unloading, the increase of blood pressure amplitude fluctuation in the Meyer's range indirectly indicates that only vasomotor component of arterial baroreflex was changed. This is consistent with the results of previously reported works [21] that demonstrated the possibility of differences between baroreflex regulation processes of heart and vessels.

It means that under the conditions of venous return moderate decrease and possibly other influences stimulating the sympathetic nervous system activity, but not accompanied by a reduction of blood pressure, and therefore not leading to direct activation of arterial baroreceptors, various components of arterial baroreflex are involved step-by-step.

First blood vessels resistance and venous tone growth is observed that might reduces vasomotor component of arterial baroreflex capacity, while the chronotropic response remains intact.

The decrease of cardiac output due to the reduction of venous return is compensated by the increase of the peripheral vessels tone.

Nevertheless the deactivation of low pressure area reflex is accompanied by the increase of BP variability in all spectral ranges, and especially in VLF range. That indirectly shows the increasing role of the slow regulating mechanisms.

Most probably the arterial baroreflex function is partially connected with cardiopulmonary baroreflex that is indicated by the increase of blood pressure spectral power in the low-frequency range (Mayer wave range) in the course of the low pressure area baroreceptors deactivation. It takes place in spite of the spontaneous arterial baroreflex immutability. Consequently,

vasomotor function of arterial baroreflex can be changed separately of chronotropic component in the case of vasomotor reactivity safety and moderate venous return decrease.

Thus CPBR is involved in the process of vessel tone fine regulation by means of both slow and rapid regulating mechanisms.

References

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[2] Taylor J.A., Eckberg D.L. Fundamental relations between short-term RR interval and arterial pressure oscillations in humans. Circulation (1996) 93:1527–1532.

[3] Taylor J.A., Carr D.L., Myers C.W., Eckberg D.L. Mechanisms underlying very-low-frequency RR-interval oscillations in humans. Circulation (1998) 98:547–555.

[4] Malpas S.C. Neural influences on cardiovascular variability: possibilities and pitfalls. Am J Physiol Heart Circ Physiol (2002) 282:H6–H20.

[5] Pagani M., Lombardi F., Guzzetti S., Rimoldi O., Furlan R., Pizzinelli P., et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circ Res (1986) 59:178–193.

[6] Di Rienzo M., Parati G., Castiglioni P., Omboni S., Ferrari A.U., Ramirez A.J., et al. Role of sinoaortic afferents in modulating BP and pulse-interval spectral characteristics in unanesthetized cats. Am J Physiol (1991) 261:H1811–H1818.

[7] Julien C., Chapuis B., Cheng Y., Barrès C. Dynamic interactions between arterial pressure and sympathetic nerve activity: role of arterial baroreceptors. Am J Physiol Regul Integr Comp Physiol (2003) 285:R834–R841.

[8] C. Julien. The enigma of Mayer waves: Facts and models. Cardiovasc Res, April 1, 2006; 70(1): 12 - 21.

[9] Levison W.H., Barnett G.O., Jackson W.D. Nonlinear analysis of the baroreceptor reflex system. Circ Res (1966) 18:673–682.

[10] Ikeda Y., Kawada T., Sugimachi M., Kawaguchi O., Shishido T., Sato T., et al. Neural arc of baroreflex optimizes dynamic pressure regulation in achieving both stability and quickness. Am J Physiol (1996) 271:H882–H890.

[11] Sato T., Kawada T., Inagaki M., Shishido T., Sugimachi M., Sunagawa K. Dynamics of sympathetic baroreflex control of arterial pressure in rats. Am J Physiol Regul Integr Comp Physiol (2003) 285:R262–R270.

[12] Yoshida T., Harasawa Y., Kubota T., Chishaki H., Kubo T., Sunagawa K., et al. Role of carotid sinus baroreflex in attenuating systemic arterial pressure variability studied in anesthetized dogs. Am J Physiol (1994) 266:H720–H729.

[13] Bertram D., Barrès C., Cuisinaud G., Julien C. The arterial baroreceptor reflex of the rat exhibits positive feedback properties at the frequency of Mayer waves. J Physiol (1998) 513:251–261.

[14] Bernardi L., Hayoz D., Wenzel R., Passino C., Calciati A., Weber R., et al. Synchronous and baroceptor-sensitive oscillations in skin microcirculation: evidence for central autonomic control. Am J Physiol (1997) 273:H1867–H1878.

[15] Borst C., Karemaker J.M., Dunning A.J., Bouman L.N., Wagner J. Frequency limitation in the human baroreceptor reflex. J Auton Nerv Syst (1983) 9:381–397.

[16] Kawano Y, Yoshimi H, Matsuoka H, Omae T. Changes in plasma endothelin-1 concentration during blood volume depletion and expansion: role of the cardiopulmonary baroreflex. Hypertens Res. 1995 Mar;18(1):43-46.

[17] Furlan R, Jacob G, Palazzolo L, Rimoldi A, Diedrich A, Harris PA, Porta A, Malliani A, Mosqueda-Garcia R, Robertson D. Sequential modulation of cardiac autonomic control induced by cardiopulmonary and arterial baroreflex mechanisms. Circulation. 2001 Dec 11;104(24):2932-2937.

[18] Thomas CJ, Woods RL. Guanylyl cyclase receptors mediate cardiopulmonary vagal reflex actions of ANP. Hypertension. 2003 Feb;41(2):279-285.

[19] Pagani M., Montano N., Porta A., Malliani A., Abboud F.M., Birkett C., et al. Relationship between spectral components of cardiovascular variabilities and direct measures of muscle sympathetic nerve activity in humans. Circulation (1997) 95:1441–1448.

[20] Cooke W.H., Hoag J.B., Crossman A.A., Kuusela T.A., Tahvanainen K.U., Eckberg D.L. Human responses to upright tilt: a window on central autonomic integration. J Physiol (1999) 517:617–628.

[21] Furlan R., Porta A., Costa F., Tank J., Baker L., Schiavi R., et al. Oscillatory patterns in sympathetic neural discharge and cardiovascular variables during orthostatic stimulus. Circulation (2000) 101:886–892.

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