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Influence of the Level of Blood Pressure on the Regulatory–Adaptive State

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Abstract—Subjects with an increased blood pressure have a decreased regulatory–adaptive potential. The degree of its decrease increases in individuals with higher blood pressure values. The achievement of the target blood pressure level with antihypertensive drugs normalizes the regulatory–adaptive potential. However, only those patients whose blood pressure values did not exceed 160/90 (systolic/diastolic) mm Hg attained the level of adaptation of healthy individuals.

Keywords: blood pressure, regulatory–adaptive abilities

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The levels f systolic (BP_s) and diastolic (BP_d) blood pressure serve as a criterion for the verification of arterial hypertension (AH), the basic parameters characterizing its severity, and the conventional parameters of the efficacy of antihypertensive therapy [1, 2]. However, it is evident that the usual BP recording using Korotkoff's method or under 24-h monitoring conditions does not include the evaluation of the individual's state of regulatory–adaptive potentialities whose level determines the capacity for an adequate response to endogenous and exogenous factors. The evaluation of the regulatory–adaptive state of the body is connected with certain methodical difficulties. Different authors have already made an attempt to reach a conclusion about the adaptive state in health and in pathology with the characteristics of the functioning of individual organs and systems. Note that the estimate of the functional state was carried out on the basis of ultrasonic dopplerography [3]; the models of evaluation of the autonomic tone, predominantly based on the dynamics of heart rate variability and the daily excretion values of adrenaline, noradrenaline, 17-ketosteroids, and 17-oxycorticosteroids, are used [4–8]. The structures and/or functions of several dominant parameters and target organs are disturbed during the formation of hypertension. In this context, the methods providing the quantitative characteristic of intersystem interaction between several autonomic functions are of special importance in the assessment of the influence of the BP level. The method of cardiac–respiratory synchronization (CRS), whose resultant parameter values are formed with the participation of several sensory inputs, the central and autonomic systems, as well as the respiratory and the cardiovascular systems, whose coordinated work may be an important guarantor of adequacy of the regulatory–adaptive reactions of the body, ensures maximum integration in the assessment of the regulatory–adaptive potential [9, 10]. At present, a considerable number of works describing in detail the state of adaptability under different conditions in health [11–14] and in pathology [11, 15–17] using CRS are available.

The regulatory–adaptive state of individuals with an increased BP was assessed in this work.

METHODS

Subjects with different BP levels (n = 168) were enrolled in the study. The average age of patients in the study group was 56.7 ± 6.6 years; the gender distribution was 97 women and 71 men; the duration of AH was 8.9 ± 2.1 years. The control group included healthy subjects (n = 34) with optimal BP_s and BP_d values of 120–139 and 70–89 mm Hg, respectively. The study group was divided into separate groups according to the degree of the BP increase: group 1, with grade I BP increase (BP_s, 140–159 mm Hg; BP_d, 90–99 mm Hg); group 2, with grade II BP increase (BP_s 160–179, mm Hg; BP_d, 100–109 mm Hg); and group 3, with grade III BP increase (BP_s is higher than 179 mm Hg; BP_d is higher than 109 mm Hg). The patients received one of the following medications as monotherapy: lisinopril at an initial dose of 10 mg/day, losartan at an initial dose of 25 mg/day, corinfar (nifedipine) retard at an initial dose of 20 mg/day. No physiotherapeutic methods of treatment were used. The drug dose was titrated for the target BP values to be attained, with the parameters studied repeatedly recorded subsequently. The CRS method whose key parameters were compared with the blood pressure level was used for the assessment of the regulatory–adaptive status. The electrocardiogram (ECG) in the

Cardiorespiratory synchronization parameters in individuals with a different BP level (M ± s)

CRS parameters	Control group ($N = 34$)	Group 1 ($N = 69$)	Group 2 ($N = 56$)	Group 3 ($N = 43$)
BP_s, mm Hg	123.7 ± 6.7	147.5 ± 6.8	168.9 ± 7.9	191.6 ± 7.8
BP_d, mm Hg	76.9 ± 4.1	92.1 ± 3.3	96.1 ± 5.9	101.5 ± 7.3
Min lim, crc/min	81.7 ± 2.1	84.2 ± 1.7	80.3 ± 1.6	79.2 ± 1.7
Max lim, crc/min	94.9 ± 2.0	92.0 ± 1.5	87.1 ± 1.9**	83.2 ± 2.4**
SR, crc/min	13.2 ± 1.2	7.8 ± 0.4*	6.8 ± 0.5***	4.0 ± 0.5****
DuD min lim, cc	14.6 ± 2.0	20.7 ± 1.8	24.2 ± 2.2**	30.2 ± 2.8****
DuD max lim, cc	16.9 ± 1.6	18.8 ± 2.0	24.5 ± 2.4**	29.0 ± 2.7***
Difference between the initial HR and Min lim, cc/min	7.6 ± 0.9	6.4 ± 1.1	3.9 ± 0.5*	1.3 ± 0.4**

Note: See the text for decoding the abbreviations. Significant differences from the control values: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; **** $p < 0.0001$.

second classical lead according to Einthoven, the pneumogram (PG), and the markers of the photostimulator lamp flashes whose frequency was regulated by the experimenter were recorded synchronously. The subject synchronized his breathing with predetermined photostimulator flashes; the appearance of CRS was determined by comparison in the synchronous ECG, PG, and the photostimulaltor marker recording. The duration of each trial was 30–60 s. In the first trial, the predetermined frequency of the photostimulator lamp flashes was 5–7% lower than that of the baseline rhythm. After the completion of the first trial, the patients relaxed for several minutes to recover their heart and respiratory rates to the initial level, the trials were repeated with a subsequent 5% increase in the photostimulator flash frequency. The trials were performed until the CRS, the state when one heart contraction corresponds to each respiratory cycle, had been attained. The following CRS parameters were analyzed:

(a) The minimal limit (min lim) of the synchronization range, i.e., the minimal frequency of the photostimulator lamp flashes and, correspondingly, the respiratory rate synchronous with them at which CRS was first formed.

(b) The maximal limit (max lim) of the synchronization range, i.e., the maximal respiratory rhythm in response to photostimulation at which CRS was still manifest but was lost if the rhythm was exceeded. The minimal and maximal limits of the synchronization range were expressed in the number of cardiorespiratory cycles per minute (crc/min).

(c) The synchronization range, which is the difference between the synchronized heart and respiratory rates within the maximal and minimal CRS limits. The range was expressed by the number of synchronous cardiorespiratory cycles per minute.

(d) The difference between the CRS minimal limit and the initial heart rate (HR) (in cardiac cycles).

(e) The duration of CRS development within the minimal and maximal limits of the synchronization range was determined by the number of cardiac cycles (cc) from the beginning of the trial to the stable CRS formation within the minimal and maximal limits of the synchronization range.

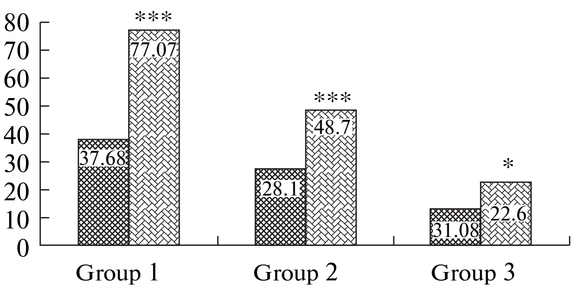
Earlier, the most informative role of the range width and the duration of CRS development within the minimal limit of the CRS range were repeatedly proven [11]. This allowed us to introduce the integral index, the index of the regulatory–adaptive state (IRAS), which represents the synchronization range (SR) value to the duration of synchronization development within the minimal limit (DuD min lim) ratio multiplied by 100 [11]. The regulatory–adaptive state is assessed by the IRAS value: the regulatory–adaptive potential is high at IRAS > 100; good at 95–50; satisfactory at 49–25; low at 24–10; and unsatisfactory at IRAS << 9.

The results obtained were statistically processed using the EXCEL 2000 software package and the STATISTICA 6.0 applied software package using statistical tests for comparison between independent and linked samples [18].

RESULTS AND DISCUSSION

In group 1, the average BP_s value exceeded this value in the control group by 19.2%; and BP_d, by 19.8%; in group 2, by 36.5 and 25.0%, respectively; and in group 3, by 54.9 and 32.0%, respectively. The comparison between the regulatory–adaptive states in individuals with different BP levels revealed the most significant differences of groups 2 and 3 from the control group (table).

Note that SR in group 2 was lower by 48.5% ($p < 0.001$) than in the control group; DuD min lim was lower by 65.6% ($p < 0.01$); and in group 3 patients, by 69.7% ($p < 0.0001$) and 106.9% ($p < 0.0001$), respectively. In group 3, the average BP_s value exceeded the



Index of the regulatory–adaptive state (IRAS) at different levels of arterial hypertension initially (dark-shaded columns) and after normalization of blood pressure (light-shaded columns).

Significant differences from the control values: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

control group values by 54.9%; and BP_d, by 32.0%. The IRAS dynamics were correlated with the BP_s ($R = -0.70$, $p < 0.01$) and BP_d ($R = -0.53$, $p < 0.01$) values. In group 1, the SR decrease was 40.9% ($p < 0.05$); the DuD min lim decrease was 41.8% with a tendency to increase. The figure shows the IRAS values. Negative IRAS dynamics were observed with increasing BP, from satisfactory in group 1 to low in group 3 (see the figure).

When antihypertensive pharmacological therapy had been used for four weeks, some of the patients attained the BP_s and BP_d values similar to those in the control group: in group 1, 85.5% of those examined attained the physiologically normal BP values; in group 2, 62.5%; and in group 3, 30.2%. The attainment of normotonia was accompanied by an IRAS increase in all the groups. The highest IRAS increase was recorded in group 1 (by 104.5%, $p < 0.01$); in these individuals, the IRAS value was brought to the state of correspondence to the regulatory–adaptive state of the control subjects. Taking into account the data in the literature on the state of cerebral blood flow and its sufficiently quick correction with BP stabilization [19–22], we suggest that an increase in the regulatory–adaptive potentialities on attaining normotension seems to be related to the improvement in the cerebral blood flow, as well as the normalization of microcirculation and/or rheological blood properties [23–25]. The IRAS increase was also noted in individuals with the initially higher BP_s and BP_d values (groups 2 and 3) (by 73.3% ($p < 0.01$) and 70.5% ($p < 0.05$), respectively); however, the index in these groups did not attain the control values and remained within the initial limits of the low and satisfactory state of the regulatory–adaptive capacity. Supposedly, this is connected with a stronger disorder of humoral and autonomic support of organ and tissue functioning and the remodeling of the cardiovascular system. It cannot be ruled out that the weaker effect in these groups is related to the longer experience of the increase in BP and the greater degree of remodeling of the target organs. In order to identify the mechanisms of recovery of the regulatory–adaptive state, additional prospective investigations are required. Regrettably, the results of the studies do not provide an unambiguous answer as to what is primary: the influence of the level of blood pressure on the state of the regulatory systems or vice versa. This requires long-term prospective investigations with the determination of the regulatory–adaptive potential of healthy individuals during a number of years to determine whether maladaptation or hypertension appears first.

CONCLUSIONS

It has been shown that a high BP decreases the regulatory–adaptive capacity. The higher the blood pressure the lower the regulatory–adaptive potential of the body. Normalization of the BP level with pharmacotherapy restores the regulatory–adaptive potential in individuals with BP increased to 160/90 mm Hg. The regulatory–adaptive capacity is not restored completely in individuals with high BP_s and/or BP_d values, even against the background of the normalization of hemodynamics.

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