## **Original Article**

# Post-loading dynamics of beat-to-beat blood pressure variability in highly trained athletes during sympathetic and parasympathetic overstrain formation

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### Abstract.

To determine the changes in beat-to-beat blood pressure variability during the development of the autonomous regulation overstrain in response to the training load. Using Spiroarteriocardiorhythmography (SACR), 202 highly qualified male athletes, aged 22.6 ± 2.8 years were examined. All studies were carried out in the precompetition period. SACR-examination were carried out three times: in the morning, on an empty stomach, in a sitting position on the day of training  $(G_1)$ , immediately (in the first 5-7 minutes) after training  $(G_2)$  and the next day in the morning after sleep (G<sub>3</sub>). The spectral parameters of the variability of systolic (SBPV) and diastolic (DBPV) pressure and their derivatives – baroceptor sensitivity (BR) and indices of heart rate centralization (ICHR), systolic (IC<sub>SBP</sub>) and diastolic (IC<sub>DBP</sub>) pressures were determined. 19 athletes were recorded to have overstrain of regulation of the sympathetic (10 athletes) and parasympathetic (9 athletes) types. They formed the observation groups OG<sub>1</sub> and OG<sub>2</sub>, respectively. The control group (CG) consisted of all 202 athletes. It is shown that, overstrain of the sympathetic type in the post-exercise and recovery period is characterized by the following: stable predominance of low-frequency and central influences on the contractile heart function immediately after exercise and the next day after exercise; a pronounced decrease in the activity of influences on the contractile heart function in the high-frequency range immediately after exercise; a steady decrease in baroreceptor sensitivity in the high and low frequency ranges immediately after exercise and the next day after exercise. The following tendencies are characteristic for overstrain of the parasympathetic type in the postexercise and recovery period: stable predominance of high-frequency influences on the contractile heart function immediately after exercise, which increases even more by the next day; a steady decrease in baroreceptor sensitivity immediately after exercise and the next day after exercise in comparison with the initial state. The results obtained significantly supplement the findings of the research obtained in the analysis of HRV.

Key words: spiroarteriocardiorhythmography, physical load, cardiovascular regulation.

### Introduction.

Maximization of an athlete's performance depends, apart from technique perfection, also on maintaining optimal balance between training and recovery, which guarantees prevention of disadaptation (Goldstein, 2010; Meeusen & Nieman, 2013) resulting from the accumulation of psychological and physiological stresses caused by training loads (Baevskiĭ & Chernikova, 2016; Grassler et al., 2021). Physical exercise being an indisputable prerequisite for training proficiency growth, can also cause physical overstrain and be accompanied with adequate and inadequate recovery (Dupuy& Dugué, 2018). In the first case, it enhances the level of fitness, but on the other hand, it provokes pre-pathological conditions of functional (Bellenger& Buckley, 2021; Nekhanevych et al., 2018) and non-functional overstrain (Le Meur & Gueneron, 2013), as well as the development of overtraining (Baumert & Baier, 2006). The leading role in development of these conditions, along with neuromuscular apparatus and cardiorespiratory system, belongs to autonomic nervous system (ANS) (Alchinova & Karganov, 2021; Guzii, 2019; Karemaker & Wesseling, 2008).

Currently, screening instrumental methods for assessing heart rate variability (HRV) are actively used in sports practice, alongside with clinical assessment of the ANS status (Cottin & Escourrou, 1999; Christiani et al., 2021; Guzii et al., 2020; Makivić et al., 2013; Shlyk, 2016). According to the results of the st udies, a great number of research have been carried out indicative of autonomic rearrangements in an athlete's body under the influence of physical exercises of varying intensity and directivity at various stages of training process (Nuuttila et al., 2017), and also shown the dependence of HRV changes on the function of external respiration (Guzii & Romanchuk, 2018; 2017; Romanchuk & Guzii, 2020, Romanchuk et al., 2019). This approach is also actively applied in clinical practice, primarily to assess the reserve functional capacities of the patient's body (Romanchuk et al., 2019; Ternovoy, 2012).

Analysis of numerous approaches to assessing HRV changes in the sports practice (Makivić et al., 2013), allowed us to focus on the method proposed by N.I. Shlyk (2016). This method enables to diagnose conditions indicating the development of regulatory tension along the central or autonomous contour of cardiac

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rhythm regulation. Regulatory tensions, being stable enough, usually characterize the condition of overstrain according to the sympathetic or parasympathetic type of regulation, which is consistent with the development of non-functional and functional overstrain (Shlyk, 2016). Both sympathetic and parasympathetic regulatory types are regarded as a precondition for the occurrence of rovertraining - a clinically significant condition associated with the regulatory malfunction of the central nervous system (CNS) (Meeusen et al., 2013). Our previous publications have shown that the type of HR regulation has fairly clear determinants associated with the function of external respiration (Romanchuk & Pisaruk, 2013; Guzii et al., 2019; Romanchuk & Guzii, 2020), sensorimotor function (Romanchuk & Guzii, 2020), and also supposes typical changes in HRV indicators under the influence of physical load (Guzii et al., 2020; Romanchuk & Dolgier, 2017; Romanchuk et al., 2006). The results obtained have added to the understanding of the changes occurring in the cardiac rhythm physiological mechanisms provision during the formation of a stable predominance of the central or autonomous contour of regulation. It has been shown that some HRV indicators demonstrate more subtle mechanisms associated with the activation of different links in cardiac rhythm regulation. These included indicators RMSSD (ms), IAB (c.u.), IAPR (c.u.), and VLF (ms²) (Guzii et al., 2020).

Blood pressure (BP) is one of the most important characteristics of the body status, being its continuous physiological variable described by pronounced fluctuations that occur during the complex interaction of neurohumoral and environmental factors. Homeostatic by nature, these fluctuations are found in all people (Zubitashvili, 2014; Alchinova & Karganov, 2021; Castiglioni et al., 2005; Noskin et al., 2018; Rosei et al., 2020). In hypertensive patients, they tend to increase (Parati et al., 2015; 2018). Blood pressure variability (BPV) is a complex phenomenon that is classified nowadays according to different types, considering the peculiarities and duration of registration. Registering blood pressure (BP) at different time intervals is widely used approach in clinical practice, and its variability is determined taking into account changes mesuared at different times (Rosei et al., 2020). At the same time, the technical innovations introduced into practice in recent years allow of blood pressure registration at each heartbeat (Pankova et al., 2020; Papaioannou et al., 2020; Penáz, 1992; Pinna et al., 1996; Pivovarov, 2006). The methodical approaches to assessing the beat-to-beat BP under standard physical load have been developed (Pivovarov et al., 2015). However, this approach has not yet become widespread due the limited predictive significant results, as well as rather stringent registration requirements. Nevertheless, this approach is being actively introduced for the prognostic assessment of arrhythmias, especially atrial fibrillation and atrial flutter (Shubik et al., 2021; Feenstra et al., 2018). At the same time, the results obtained allow us to claim that the variability of blood pressure (BPV) with short registrations is associated with baroceptor reflexes (Baumert, 2006; Guziy & Romanchuk, 2016; Wesseling et al., 2017), the activity of the sympathetic nervous system (Guzii & Romanchuk, 2017; Incognito et al., 2020), the reninangiotensin-aldosterone system (Fadel, 2008; Kishi, 2018), nitric oxide release (Lacchini et al., 2001) and behavioral changes (Castiglioni et al., 2009; Cherepov et al., 2014). That is, BPV can serve as an important and pronounced trait determining the current body status, which is indispensable to diagnosing prenosological conditions, concerning overstrain of the ANS branches activity. Previous studies of the systolic blood pressure variability (SBPV) and diastolic blood pressure variability (DBPV) in athletes, taking into account the maximal oxygen uptake (VO<sub>2</sub>max) indicators, showed their minor differentiation, though still significant enough (Guzii & Romanchuk, 2017). Both in foreign research and the studies carried out by us, important results have been obtained while studying the sensitivity of baroreceptors (Guziy & Romanchuk, 2016; Romanchuk & Guziy, 2018; Romanchuk & Guzii, 2019) at rest and under the effect of physical loads. The latter has predetermined the expediency of studying BPV as the criterion of athletes' overstrain according to sympathetic and parasympathetic types.

The purpose of the study was to determine the changes of BPV indices in trained athletes occurring during the post-loading and recovery periods, accompanied by the formation of overstrain according to sympathetic and parasympathetic types.

### Materials and methods

To determine BPV the "Spiroarteriocardiorhythmograph" device (SACR) ("Intox", Russia) was applied, recommended for use in medical practice by the Ministry of Health of Russia (registration certificate No 29/03020703/5869-04). The device combines three certain methods of physiological studies into an integrated hardware complex, which makes it possible to achieve a fundamentally new quality of measurements, that is, simultaneous recording of HRV and BPV at different stages of the respiratory act (Pivovarov, 2006).

Registration of ECG was carried out in the first standard lead for 2 minutes in a sitting position. The amplitude-time parameters of HRV were estimated with the help of statistical, geometric and spectral methods. HRV was analyzed applying a simple Fourier transform, obtaining a spectrum distribution curve of the frequency changes in HR. The total power of HRV was calculated (TP), as well as three standard components of the spectrum: very low-frequency oscillations (VLF, 0-0,04 Hz), low-frequency oscillations (LF, 0,04-0,15 Hz) and high-frequency oscillations (HF, 0,15-0,4 Hz). Besides, normalized values for these values (LFn, HFn) were also calculated as a fraction of the sum of all components, and indices of vegetative balance (LF / HF) and centralization (CI<sub>HR</sub> = (VLF + LF) / HF) were computed as well. IAB, IAPR, SI, SDANN, RMSSD, pNN50 were defined (Baevskiĭ et al., 2012). Arterial blood pressure was recorded on the phalanx of the middle finger

using the Penaz's method continuously for 2 minutes. Both the absolute values of systolic (SBP) and diastolic (DBP) pressure and BPV were evaluated. Similarly to HRV, the total power of the spectrum total power of systolic ( $TP_{SBP}$ , mmHg²) and diastolic ( $TP_{DBP}$ , mmHg²) blood pressure and their frequency components were calculated. The indices of autonomic balance ( $LF/HF_{SBP}$ ,  $LF/HF_{DBP}$ ), centralization ( $CI_{SBP}$ ,  $CI_{DBP}$ ) and coefficients  $\alpha$  of baroreceptor sensitivity (BR) in the low-frequency and high-frequency ranges ( $BR_{LF}$ , ms/mmHg,  $BR_{HF}$ , ms/mmHg) were also calculated (Guziy & Romanchuk, 2016; Pankova et al., 2020; Tkaczyszyn et al., 2013).

In general the physiological significance of the SBPV indicators could be associated with regulatory effect on the heart contractile function (HCF). The physiological significance of DBPV indicators is more associated with the regulation of vascular tone.

Figures 1 and 2 show an example of a registration record rhythmogramme and primary results obtained with the SACR.

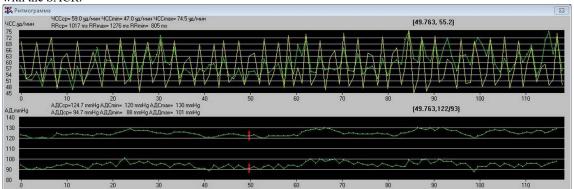


Fig.1. SACR registration record rhythmogramme. The upper window demonstrates HR (green) and respiration (yellow) rhythmogrammes. The bottom window depicts SBP and DBP rhythmogrammes.

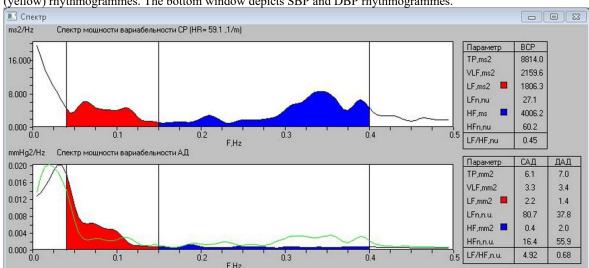


Fig. 2. Spectrogramme and analysis. The upper window demonstrates HR, the bottom – SBP and DBP.

Test subjects were qualified male athletes (N=202) aged  $22.6 \pm 2.8$ , who were examined by means of SACR. The athletes represented acyclic sports, such as martial arts (karate, taekwondo, kickboxing, boxing, wrestling, judo, sambo) and ball games like water polo and football. The experience of the involvement in sports was  $10.3 \pm 3.1$  years. All the studies were carried out in the precompetitive period. In accordance with the design examinations with the aid of SACR were carried out three times: in the morning, on an empty stomach, in a sitting position on the day of training ( $G_1$ ), immediately after training (during the first 5-7 minutes) ( $G_2$ ) and the next day in the morning after sleep ( $G_3$ ). Each registration lasted 2 minutes. A morphometric study and routine blood pressure measurements with a tonometer were carried out before SACR application. Based on the data obtained, the following indices were calculated characterizing the functional state of the cardiorespiratory system and the body as a whole: Kerdo index (KI), double product (DP), Baevsky adaptive potential (AP), the level of physical fitness by Pirogova (PFL).

Statistical analysis. The data obtained are presented as a median with 25-75% ( $Q_1$ ;  $Q_3$ ) percentiles. Nonparametric Mann-Whitney test was applied to assess intergroup differences. Differences between initial and subsequent measurements were taken via Wilcoxon matched-pairs test.

At the first stage of the analysis, the type of autonomous HR regulation was observed in all athletes before, after and during the recovery period after physical load (Guzii, 2019; Guzii & Romanchuk, 2021) and was estimated in accordance with the method proposed by N.I. Shlyk (2016). This method assumes the classification of HRV data according to TP (ms<sup>2</sup>), SI (c.u.) u VLF (ms<sup>2</sup>) indicators. There are 4 types of autonomous HR regulation: type I indicates moderate tension, type II testifies to a decrease of the regulatory systems functional state, the development of fatigue, type III indicates optimal state of regulation, type IV - an overstrain of parasympathetic regulation, or a state of high fitness.

According to the results obtained via SACR indicators study several cases indicating the development of overstrain in the HR regulation were identified, namely 19 cases out of overall number of 202 subjects. They were attributed to 2 variants of changes in HR regulation, which are as follows:

Variant 1 is characterized by the initial optimal state of regulatory systems, or by overstrain of autonomous regulation (types III and IV), characterized by a decrease in the functional state of regulatory systems (type II) after an intense training load, the state persisting until the next morning after training. This version was recorded in 10 cases, indicating the sympathetic type of overstrain;

Variant 2 testifies to initial overstrain of autonomous regulation (IV type), which, after an intense training load, was replaced by an optimal state of regulatory systems, or else an overstrain of autonomous regulation (types III and IV), and on the morning after training an overstrain of autonomous regulation (type IV) was observed. This version was recorded in 9 cases, indicating the parasympathetic type of overstrain.

The subjects mentioned above made up 2 groups of observation (OG), OG<sub>1</sub> and OG<sub>2</sub> respectively. The control group (CG) consisted of all 202 surveyed athletes.

The characteristics of morphological and functional parameters determined before the training load (G<sub>1</sub>) are presented in Table 1. It should be noted that the whole group of highly qualified athletes (CG) displayed a sufficiently high level of physical fitness according to all parameters. The data obtained are supplemented by the results of routine measurements of the cardiovascular system parameters and various integral indicators of the body status, computed according to known formulas. Considering all the above mentioned parameters, the level of the functional state of the cardiorespiratory system of highly qualified athletes (CG) could be characterized as

Table 1 Morpho-functional characteristics of athletes in the observation groups (OG) in the initial state with the development of overstrain according to the sympathetic (OG<sub>1</sub>) and parasympathetic (OG<sub>2</sub>) types in comparison with the control group (CG),

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Indicator	CG	$OG_1$	$OG_2$
	n=202	n=10	n=9
Body mass, kg	72.0 (62.0; 82.0)	80.0 (61.0; 94.0)	66.5 (61.0; 81.0)#
Body length, cm	179.0 (170.0; 185.0)	181.5 (170.0; 189.0)	179.0 (175.0; 185.0)
BMI, kg/m <sup>2</sup>	22.5 (20.9; 25.2)		20.5 (19.9; 24.2)#@
contour abdomen, cm	78.0 (74.0; 86.5)	82.5 (74.0; 92.0)	75.0 (73.0; 82.0)#
contour hip, cm	52.0 (48.0; 56.5)	56.0 (50.0; 60.0)	48.0 (45.0; 57.0) <sup>#@</sup>
vital lung capasity, ml	4800 (4400; 5600)	4850 (4400; 6600)	4850 (4500; 4900)
SBP, mmHg	120 (110; 130)	115.0 (110.0; 120.0) <sup>@</sup>	115.0 (100.0; 120.0) <sup>@</sup>
DBP, mmHg	70 (64; 80)	80.0 (70.0; 80.0) <sup>@</sup>	70.0 (70.0; 80.0)#
KI, c.u.	-0.19 (-0.35; -0.05)		-0.34 (-0.45; -0.28) <sup>#@</sup>
DP, c.u.	71.8 (64.6; 81.8)	73.6 (65.1; 75.7)	60.3 (51.7; 75.8) <sup>#@</sup>
Baevsky's AP, c.u.	2.02 (1.87; 2.25)	1.98 (1.84; 2.12)#	1.79 (1.52; 1.99)##@
PFL by Pirogova, c.u.	0.746 (0.672; 0.822)	0.736 (0.692; 0.762)	0.823 (0.753; 0.901)#@

 $<sup>^{(0)}</sup>$  - p< 0.05 between OG<sub>1</sub> and OG<sub>2</sub> in comparison with CG

Tables 2 - 4 show the results of changes in the parameters of the variability of systolic (SBPV) and diastolic pressure (DBPV). SBPV of athletes at G1 showed that the groups are differentiated (Table 2). The absence of differences between TP<sub>SBP</sub> and VLF<sub>SBP</sub> indicators characterizes sufficient stability of the autonomous regulation of contractile heart function (CHF) against the background of significant differences in the HR regulation (Guzii et al., 2020). On the other hand, with G<sub>1</sub>, significant differences are noted in OG<sub>1</sub> in comparison with CG: less pronounced low-frequency influences are recorded (LF<sub>SBP</sub> LF<sub>SBP</sub>n) - 2.3 (1.0; 7.3) versus 3.6 (2.3; 7.3), p <0.05, and 28.4 (20.2; 34.0) versus 45.2 (30.7; 61.1), p <0.05, respectively. The latter is reflected in the relative parameters of high-frequency influences (HF<sub>SBP</sub>n), which in OG<sub>1</sub> are significantly higher - 67.6 (59.7; 76.4) versus 51.0 (34.3; 62.4), p <0.05. The parameter of the ratio of influences (LF/HF<sub>SRP</sub>) also differs: 0.44 (0.26; 0.58) versus 0.87 (0.48; 1.69), p <0.05. This type of the relationship of different-frequency influences at G<sub>1</sub>, taking into account the effects on HR, can be considered as multidirectional autonomic influences on chronotropic and inotropic heart functions. This is confirmed by the SBPV parameters data at G<sub>1</sub> in OG<sub>2</sub> in comparison with CG and OG<sub>1</sub>. Significant differences could be observed in LF<sub>SBP</sub>, LF<sub>SBP</sub>n, HF<sub>SBP</sub>, HF<sub>SBP</sub>n and LF/HF<sub>SBP</sub> parameters. The most impressive differences could be identified in low-frequency effects:  $(LF_{SBP}, LF_{SBP}n) - 7.0 (2.3; 17.6)$  versus 3.6 (2.3; 7.3), p< 0.05 in CG, and versus 2.3 (1.0; 7.3), p< 0.05 in OG<sub>1</sub>,

<sup># -</sup> p< 0.05; ## - p< 0.01 between  $OG_2$  and  $OG_1$ 

as well as 61.8 (50.0; 78.3) versus 45.2 (30.7; 61.1), p< 0.05, in CG, and versus 28.4 (20.2; 34.0), p< 0.05 in OG<sub>1</sub>, respectively. That is, the initial state a differentiation of low-frequency and high-frequency effects on HR and SBP between athletes with supposed overexertion of the cardiovascular system regulation according to the sympathetic and parasympathetic types could be observed. At G<sub>2</sub>, significantly lower high-frequency influences are noted in OG<sub>1</sub> in comparison with CG and OG<sub>2</sub> (HF<sub>SBP</sub>, HF<sub>SBP</sub>n) – 1.8 (1.7; 2.3) versus 4.4 (2.6; 9.0), p< 0.01 in CG, and versus 6.6 (1.4; 8.4), p< 0.01 in OG<sub>2</sub>, and also 20.5 (13.8; 45.6) against 35.6 (21.8; 54.3), p< 0.05 in CG, and versus 42.7 (34.1; 61.0), p< 0.01 in OG<sub>2</sub>. The highest rate observed was LF/HF<sub>SBP</sub> – 4.00 (1.06; 5.20) versus 1.77 (0.77; 3.50), p< 0.05 in CG, and versus 0.88 (0.53; 1.88), p< 0.01 in OG<sub>2</sub>. Analysis of the changes dynamics in the parameters of the SBPV in response to an intense load (G<sub>2</sub>-G<sub>1</sub>) showed that in the CG there is a significant increase in TP<sub>SBP</sub>: 25.0 (11.6; 42.3) versus 18.5 (11.6; 32.5), p< 0.05; LF<sub>SBP</sub> – 7.8 (2.9; 16.0) against 3.6 (2.3; 7.3), p< 0.01, LF/HF<sub>SBP</sub> – 1.77 (0.77; 3.50) against 0.87 (0.48; 1.69), p< 0.01, as well as the decrease of HF<sub>SBP</sub>n – 35.6 (21.8; 54.3) versus 51.0 (34.3; 62.4), p< 0.01. That is, in the group of athletes in general there is an increase in regulatory influences on the CHF due to the activation of the sympathetic branch of the ANS.

Significant decrease in  $HF_{SBP} - 1.8$  (1.7; 2.3) versus 7.1 (2.9; 14.4), p <0.01 in the changes in HRV indicators in response to the load in  $OG_1$  was observed, which was reflected in  $LF/HF_{SBP}$  that increased from 0.44 (0.26; 0.58) to 4.00 (1.06; 5.20), p< 0.01. At the same time, significant changes in SBP variability in  $OG_2$  concerned only the redistribution of the activity of low-frequency and high-frequency effects, which influenced the relative values:  $LF_{SBP}$ n,  $HF_{SBP}$ n and  $LF/HF_{SBP}$ . Namely, the contribution of  $LF_{SBP}$ n decreased from 61.8 (50.0; 78.3) to 43.3 (33.9; 60.5), p <0.05,  $HF_{SBP}$ n increased from 33.3 (19.5; 35.9) to 42.7 (34.1; 61.0), p <0.05, respectively,  $LF/HF_{SBP}$  decreased from 1.80 (1.49; 4.00) to 0.88 (0.53; 1.88), p <0.05. That is, physical activity in individuals with developing overstrain of the sympathetic type ( $OG_1$ ) causes a significant decrease in HF influences on SBP while the LF influences remain unchanged, which leads to a significant redistribution of relative influences on the SBP could also be observed, but in the opposite direction. This confirms the extremely rapid activation of parasympathetic influences on CHF immediately after the end of exercising.

Analysis of the dynamics of changes in CHF parameters on the morning after a training session as compared with the initial state  $(G_3-G_1)$  showed that their complete recovery was observed in the CG. Significant differences in the relative components of SBPV (LF<sub>SBP</sub>n, HF<sub>SBP</sub>n and LF/HF<sub>SBP</sub>) could be noted in OG<sub>1</sub>, indicating their reverse direction in comparison with  $G_2$ . On the other hand, there is a significant decrease in suprasegmental (VLF<sub>SBP</sub>) effects: 2.6 (2.3; 2.9) versus 4.5 (1.2; 25.0), p <0.05. In OG<sub>2</sub>, against the background of unchanged absolute TP<sub>SBP</sub>, VLF<sub>SBP</sub> and LF<sub>SBP</sub>, there are more significant changes in LF<sub>SBP</sub>n and HF<sub>SBP</sub>n: 44.6 (25.4; 53.9) versus 61.8 (50.0; 78.3), p <0.05, and 49.7 (37.9; 51.5) versus 33.3 (19.5; 35.9), p <0.01, indicating an excessive high-frequency effect on the CHF in the recovery period, which is also reflected in the indicator LF/HF<sub>SBP</sub> - 0.90 (0.49; 1.42) versus 1.80 (1.49; 4.00), p <0.01. At the same time, these changes remain at the level of postloading (G<sub>2</sub>), characterizing the stability of influence of the ANS parasympathetic branch on the CHF, as well as on the hart rate (HR) (Le Meur et al., 2013).

Table 2 Differences in changes in SBPV indicators in athletes under the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic  $(OG_1)$  and

parasympathetic (OG<sub>2</sub>) types, M (Q1; Q3)

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Indicator	Control	CG n=202	OG <sub>1</sub> n=10	OG <sub>2</sub> n=9
	$G_1$	18.5 (11.6; 32.5)	17.4 (5.3; 43.6)	24.2 (6.3; 32.5)
TP <sub>SBP</sub> , mmHg <sup>2</sup>	$G_2$	25.0 (11.6; 42.3)*	15.7 (8.4; 25.0) <sup>@</sup>	19.3 (5.8; 59.3)
11 SBP, IIIII11g		17.6 (9.6; 33.6)	11.4 (5.3; 22.1)	19.7 (13.7; 28.1)#
		6.8 (3.2; 14.4)	4.5 (1.2; 25.0)	6.0 (2.9; 19.4)
VLF <sub>SBP</sub> , mmHg <sup>2</sup>		6.3 (2.0; 11.6)	3.4 (1.2; 14.4)	8.3 (2.3; 25.0)
	$G_3$	4.8 (2.3; 10.2)	2.6 (2.3; 2.9)@*	5.6 (3.6; 16.8)##
		3.6 (2.3; 7.3)	2.3 (1.0; 7.3) <sup>@</sup>	7.0 (2.3; 17.6) <sup>@#</sup>
LF <sub>SBP</sub> , mmHg <sup>2</sup>	$G_2$	7.8 (2.9; 16.0)**	5.3 (1.7; 10.2)	4.1 (1.4; 15.2)
_		5.3 (2.0; 11.6)	1.8 (1.4; 9.0)@	5.9 (2.3; 10.2)#
	$G_1$	45.2 (30.7; 61.1)	28.4 (20.2; 34.0) <sup>@</sup>	61.8 (50.0; 78.3) <sup>@##</sup>
LF <sub>SBP</sub> n, n.u.	$G_2$	61.2 (41.8; 75.8)**	71.9 (48.3; 81.8)**	43.3 (33.9; 60.5) <sup>@#*</sup>
	$G_3$	47.5 (31.2; 69.1)	45.9 (36.2; 63.6)**	44.6 (25.4; 53.9)*
	$G_1$	4.4 (2.6; 8.4)	7.1 (2.9; 14.4)	2.0 (1.2; 8.4) <sup>@#</sup>
HF <sub>SBP</sub> , mmHg <sup>2</sup>	$G_2$	4.4 (2.6; 9.0)	1.8 (1.7; 2.3)@@**	6.6 (1.4; 8.4)#
-	$G_3$	4.4 (2.0; 8.4)	4.4 (0.8; 10.2)	5.8 (2.9; 8.4)*
	$G_1$	51.0 (34.3; 62.4)	67.6 (59.7; 76.4) <sup>@</sup>	33.3 (19.5; 35.9) <sup>@##</sup>
HF <sub>SBP</sub> n, n.u.		35.6 (21.8; 54.3)*	20.5 (13.8; 45.6)@**	42.7 (34.1; 61.0) <sup>@#*</sup>
	$G_3$	48.8 (27.6; 62.6)	45.6 (33.2; 61.5)*	49.7 (37.9; 51.5)**
	$G_1$	0.87 (0.48; 1.69)	$0.44 (0.26; 0.58)^{@}$	1.80 (1.49; 4.00)@##
LF/HF <sub>SBP</sub> , mmHg <sup>2</sup> / mmHg <sup>2</sup>	$G_2$	1.77 (0.77; 3.50)**	4.00 (1.06; 5.20)@**	0.88 (0.53; 1.88)#*
	$G_3$	0.98 (0.49; 2.50)	1.09 (0.59; 1.90)**	0.90 (0.49; 1.42)**
2626				

@ - p< 0.05, @@ - p< 0.01 between  $OG_1, OG_2$  in comparison with CG # - p< 0.05, ## - p< 0.01, ### - p< 0.001 between  $OG_1$  and  $OG_2$  \* - p< 0.05, \*\* - p< 0.01, between  $G_1$  and  $G_2$  in comparison with  $G_1$ 

Diastolic blood pressure variability (DBPV) is a reflection of the lability of the vessel wall, its ability to adjust to blood flow. DBPV indicators are directly related to parameters of peripheral vascular resistance and vascular tone (Guzii & Romanchuk, 2018). Certain distinctions from CG could be observed in OG<sub>1</sub> and OG<sub>2</sub> in the initial state (G<sub>1</sub>) (Table 3).  $TP_{DBP}$  parameter in OG<sub>2</sub> is significantly higher than in other groups: 13.3 (11.6; 13.7) versus 7.3 (4.8; 12.9), p <0.05, in CG, and versus 8.4 (4.8; 33.6), p <0.05, in OG<sub>1</sub>. This distinction is provided by a significantly higher contribution of  $LF_{DBP}$ : 4.3 (2.3; 10.2) versus 2.3 (1.2; 4.0), p <0.05, in CG, and versus 2.1 (1.2; 7.3), p <0.05, in OG<sub>1</sub>, as well as  $HF_{DBP}$ : 3.7 (2.6; 4.8) versus 1.4 (0.6; 2.6), p < 0.01, in CG. At the same time, in OG<sub>1</sub>, the  $HF_{DBP}$  values also exceed those parameters in CG: 2.9 (1.2; 11.6) versus 1.4 (0.6; 2.6), p <0.05. Apart from this, in OG<sub>1</sub>, as opposed to OG<sub>2</sub>, there is a significant redistribution of the  $LF_{DBP}$ n and  $HF_{DBP}$ n relative components, which characterizes the prevalence of high-frequency influences on DBP in comparison with CG.

The effect of training load ( $G_2$ ) in CG causes an increase in LF<sub>DBP</sub> from 2.3 (1.2; 4.0) to 3.6 (2.3; 6.8), p <0.01, which can be explained by an increase in low-frequency influences on heart rate (Guzii et al., 2019; 2020). This is reflected in the relative parameters: an increase in LF<sub>DBP</sub>n, a decrease in HF<sub>DBP</sub>n and an increase in LF/HF<sub>DBP</sub>, p <0.01. In contrast to CG, in OG<sub>1</sub> at G<sub>2</sub>, against the background of unchanged LF<sub>DBP</sub>, there is a significant decrease in HF<sub>DBP</sub> from 2.9 (1.2; 11.6) to 0.6 (0.4; 0.8), p <0.01. At the same time, the activity of influences in the HF-range reaches the lowest values and significantly differs from the CG: 1.0 (0.5; 2.0), p <0.05, and  $OG_2 - 1.7$  (1.2; 2.0), p <0.01. Such a decrease leads to a change in the relative parameters: an increase in LF<sub>DBP</sub>n, a decrease in HF<sub>DBP</sub>n, and an increase in LF/HF<sub>DBP</sub>, p <0.01. That is, in OG<sub>1</sub>, the changes are similar to those in CG, only genesis of changes is different. In CG it occurs due to the activation of sympathetic influences, and in OG1 - due to the inhibition of parasympathetic ones. In OG2 at G2, a significant increase in  $TP_{DBP}$  in comparison with  $G_1$  could be noted: from 13.3 (11.6; 13.7) to 14.4 (4.0; 27.0), p <0.05 first of all, due to significant increase in very-low-frequency (VLF<sub>DBP</sub>) influences from 2.4 (2.3; 5.8) to 7.9 (2.9; 22.1), p <0.05. At the same time, LF<sub>DBP</sub> and HF<sub>DBP</sub> significantly decrease from 4.3 (2.3; 10.2) to 2.3 (0.8; 3.2), p <0.05, and from 3.7 (2.6; 4.8) to 1.7 (1.2; 2.0), p <0.01 respectively. That is, with the emerging parasympathetic overstrain after exercise; there is an excessive activation of subcortical regulatory structures associated with the possible excessive activation of neurohumoral mechanisms for vascular tone maintaining.

All the parameters of DBPV in CG return to the initial level  $(G_1)$  the morning after the workout  $(G_3)$ . In  $OG_1$ , in comparison with  $G_1$ , there is a significant decrease in  $TP_{DBP} - 5.3$  (3.2; 7.3) versus 8.4 (4.8; 33.6), p <0.05, LF<sub>DBP</sub> - 1.1 (0.8; 3.6) versus 2.1 (1.2; 7.3), p <0.05, HF<sub>DBP</sub> - 0.5 (0.3; 1.7) versus 2.9 (1.2; 11.6), p <0.05. At the same time, a significant decrease in HF<sub>DBP</sub> recorded at G<sub>2</sub> remains at the same level at G<sub>3</sub>, which indicates a stable suppression of parasympathetic influences. It should be mentioned here that the absence of highfrequency regulatory influences on vascular tone is physiologically justified and may indicate a violation of mediated influences, associated with a violation of the acid-base state for instance. Therefore, in this particular case, we would limit ourselves only to the statement of the fact. These changes are reflected in the relative parameters (LF<sub>DBP</sub>n, HF<sub>DBP</sub>n, LF/HF<sub>DBP</sub>), which remain at the post-exercise (G<sub>2</sub>) level and differs from G<sub>1</sub> (p <0.01). Athletes OG<sub>2</sub> have a similar, but rather specific dynamics of changes at G<sub>3</sub> in comparison with G<sub>1</sub>. There is a significant decrease in TP<sub>DBP</sub> – 5.9 (4.4; 7.8) versus 13.3 (11.6; 13.7), p <0.01, at the expense of all frequency components: VLF<sub>DBP</sub> - 1.6 (0.8; 3.2) versus 2.4 (2.3; 5.8), p <0.05, LF<sub>DBP</sub> - 1.6 (0.6; 3.2) versus 4.3 (2.3; 10.2), p <0.01, HF<sub>DBP</sub> - 2.3(1.4; 4.0) versus 3.7 (2.6; 4.8), p <0.05. As you can see, the most significantly reduced parameter is the LF influence. This is reflected in the relative parameters (HF<sub>DBP</sub>n and LF/HF<sub>DBP</sub>), the first of which significantly increases in comparison with  $G_1$ : 52.4 (39.0; 66.5) versus 39.3 (20.4; 56.3), p <0.05, and the second decreases: 0.68 (0.24; 1.46) versus 1.24 (0.45; 3.69), p <0.05. Thus, even greater prevalence of high-frequency influences occurs as compared with G<sub>1</sub> and G<sub>2</sub>.

Table 3 Differences in changes in DBPV indicators in athletes under the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic (OG<sub>1</sub>) and parasympathetic (OG<sub>2</sub>) types, M (O1: O3)

Control point	CG n=202	-	OG <sub>2</sub> n=9
			13,3 (11,6; 13,7) <sup>@#</sup> 14,4 (4,0; 27,0) <sup>#*</sup>
	7,3 (4,0; 10,9)	5,3 (3,2; 7,3) <sup>@*</sup>	5,9 (4,4; 7,8)**
_		3,7 (1,4; 4,8) 2,1 (0,5; 4,0)	2,4 (2,3; 5,8) 7,9 (2,9; 22,1) <sup>@#*</sup>
		2,0 (1,2; 3,6)	1,6 (0,8; 3,2)*
$G_2$	3,6 (2,3; 6,8)**	3,3 (1,2; 4,0)	4,3 (2,3; 10,2) <sup>@#</sup> 2,3 (0,8; 3,2) <sup>@#*</sup>
	2,3 (1,2; 4,8) 59 9 (43 5: 74 5)	1,1 (0,8; 3,6) <sup>@</sup> * 36 5 (27 8: 46 1) <sup>@</sup>	1,6 (0,6; 3,2)** 49,2 (25,3; 75,2)#
	$G_1$ $G_2$ $G_3$ $G_1$ $G_2$ $G_3$ $G_1$ $G_3$ $G_1$ $G_3$ $G_1$ $G_2$ $G_3$	$\begin{array}{c} G_1 \\ 7,3 \ (4,8; 12,9) \\ G_2 \\ 7,8 \ (5,3; 16,8) \\ G_3 \\ 7,3 \ (4,0; 10,9) \\ G_1 \\ 2,9 \ (1,4; 5,3) \\ G_2 \\ 2,3 \ (1,0; 4,4) \\ G_3 \\ 2,3 \ (1,2; 4,0) \\ G_1 \\ 2,3 \ (1,2; 4,0) \\ G_2 \\ 3,6 \ (2,3; 6,8)^{**} \\ G_3 \\ 2,3 \ (1,2; 4,8) \end{array}$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$

	$G_2$	78,5 (59,6; 88,4)**	84,4 (59,0; 89,2)**	58,3 (28,9; 62,3) <sup>@#</sup>
	$G_3$	64,5 (50,3; 79,2)	64,6 (63,0; 79,8)**	37,4 (18,0; 57,5) <sup>@##</sup>
	$G_1$	1,4 (0,6; 2,6)	2,9 (1,2; 11,6) <sup>@</sup>	3,7 (2,6; 4,8) <sup>@@</sup>
HF <sub>DBP</sub> , mmHg <sup>2</sup>	$G_2$	1,0 (0,5; 2,0)	0,6 (0,4; 0,8)@**	1,7 (1,2; 2,0)@##**
	$G_3$	1,0 (0,6; 1,7)	0,5 (0,3; 1,7)@*	2,3 (1,4; 4,0)@#*
	$G_1$	31,3 (18,7; 48,4)	55,4 (47,3; 58,5) <sup>@</sup>	39,3 (20,4; 56,3)#
HF <sub>DBP</sub> n, n.u.	$G_2$	18,1 (10,4; 37,0)**	14,1 (7,8; 33,2)**	36,2 (34,3; 50,7) <sup>@##</sup>
	$G_3$	29,8 (15,9; 39,7)	27,4 (10,4; 32,9)**	52,4 (39,0; 66,5) <sup>@##*</sup>
		2,07 (0,85; 3,72)	0,66 (0,41; 0,98) <sup>@</sup>	1,24 (0,45; 3,69)#
LFHF <sub>DBP</sub> , mmHg <sup>2</sup> / mmHg <sup>2</sup>	$G_2$	4,37 (1,61; 8,59)**	6,05 (1,77; 11,42)**	1,61 (0,52; 1,82) <sup>@#</sup>
		2,10 (1,28; 5,20)	2,42 (1,96; 7,67)**	0,68 (0,24; 1,46)@##*

 $^{\textcircled{@}}$  - p< 0.05,  $^{\textcircled{@}}$  - p< 0.01 between OG<sub>1</sub>,OG<sub>2</sub> in comparison with CG  $^{\#}$  - p< 0.05,  $^{\#}$  - p< 0.01,  $^{\#\#}$  - p< 0.001, between OG<sub>1</sub> and OG<sub>2</sub>  $^{*}$  - p< 0.05,  $^{**}$  - p< 0.01, between G<sub>3</sub> and G<sub>2</sub> in comparison with G<sub>1</sub>

Significant differences could be observed in the integral parameters of the cardiovascular system, which characterize the relationship between different HR regulatory components, like ( $CI_{HR}$ ) and BP ( $CI_{SBP}$ ,  $CI_{DBP}$ ), as well as baroreceptor regulation of HR and CHF ( $BR_{LF}$ ,  $BR_{HF}$ ) (Tkaczyszyn et al., 2013) (Table 4).

Analyzing the differences between the experimental groups at G<sub>1</sub>, it should be noted that OG<sub>1</sub> differs from the CG in a significantly lower central influence on SBP (CI<sub>SBP</sub>) and DBP (CI<sub>DBP</sub>): 1.37 (0.70; 1.88) versus 2.92 (1.43; 5.69), p <0.05, and 1.63 (1.54; 3.39) versus 4.63 (2.33; 10.07), p <0.05, respectively. At the same time, the effects on HR (CI<sub>HR</sub>) do not differ. That is, low IC<sub>SBP</sub> and IC<sub>DBP</sub> in the initial state may enhance the development of sympathetic overstrain. On the other hand, in OG2 at G1 in comparison with CG against the background of similar influences on HR and DBP, there is a significantly greater centralization of influences on SBP – 3.99 (3.26; 11.32) versus 2.92 (1.43; 5.69), p <0.05. It exceeds significantly that one in  $OG_1$ . Differences in baroreceptor sensitivity in terms of BR<sub>LF</sub> and BR<sub>HF</sub> turned out to be significant, which in OG<sub>2</sub> at G<sub>1</sub> were significantly higher than in CG and  $OG_1$  - 34.2 (25.0; 47.7) versus 17.5 (11.2; 27.8), p <0.05, versus CG, and versus 24.7 (10.1; 43.6), p <0.05, versus OG<sub>1</sub> for LF interactions, and 51.9 (38.6; 111.9) versus 25.3 (12.8; 38.5), p <0.01, versus CG, and versus 27.3 (12.4; 32.6), p <0.01, compared to OG<sub>1</sub> for HF interactions. At G<sub>2</sub>, changes in the integral indicators in OG<sub>1</sub> and OG<sub>2</sub> are multidirectional. In OG<sub>1</sub>, there is a more pronounced centralization of influences on HR, SBP and DBP in comparison with the CG. In OG<sub>2</sub>, against the background of a decrease in the centralization of influences at  $G_2$  in comparison with  $G_1$  on HR ( $CI_{HR}$ ) – 0.44 (0.32; 0.55) versus 0.75 (0.34; 1.88), p < 0.05, CHF (CI<sub>SBP</sub>) - 2.42 (2.13; 4.42) versus 3.99 (3.26; 11.32), p < 0.05, there is an increase in their effects on VT (CI<sub>DBP</sub>) - 8.48 (4.38; 12.92) versus 2.38 (1.27; 6.46), p <0.05. This is accompanied by a decrease in the sensitivity of baroreceptors, both in the low- (BR<sub>LF</sub>) and high-frequency (BR<sub>HF</sub>) ranges. However, even in this particular case, its absolute values significantly exceed the same parameters in the CG in the initial state. That is, a decrease in the centralization of influences on HR and CHF after exercise is peculiar for the formation of parasympathetic overstrain. At G<sub>3</sub>, the change in most of the integral indicators in OG<sub>1</sub> and OG<sub>2</sub> is aimed at restoring the initial values, which is what happens in the CG. In contrast to CG, in OG<sub>1</sub> and OG<sub>2</sub>, there is a slowdown in the restoration of baroreceptor sensitivity (BR<sub>LF</sub>, BR<sub>HF</sub>), which have intermediate values between G<sub>1</sub> and G<sub>2</sub>, at their own level in each of the groups at that. At the same time, during the accumulation of sympathetic overstrain (OG<sub>1</sub>), there is a slowdown in the recovery of central influences on the vascular one (CI<sub>DBP</sub>), which remain at the G<sub>2</sub> level; whereas during the formation of parasympathetic overstrain (OG<sub>2</sub>), a slowdown in the recovery to G<sub>1</sub> of the central effects on CHF (CI<sub>SBP</sub>), remaining at the post-load level G<sub>2</sub>. Table 4 Differences in changes in the integral indicators of the cardiovascular system in athletes under the

Table 4 Differences in changes in the integral indicators of the cardiovascular system in athletes under the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $(OG_1)$ ) and parasympathetic types ( $OG_2$ ), M ( $OG_1$ ) and Difference in athletes under the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $(OG_1)$ ) and parasympathetic types ( $OG_2$ ), M ( $OG_1$ ) and Difference in athletes under the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $OG_2$ ) and Difference in the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $OG_2$ ) and Difference in the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $OG_2$ ) and Difference in the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $OG_2$ ) and Difference in the influence of an intense training load and during the recovery period after it with the development of overstrains in sympathetic ( $OG_2$ ) and Difference in the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the development of the influence of a period after it with the influence of a period after it with the influence of a per

m sympatine	275 (( 3	G <sub>1</sub> ) and parasympametre ty	pes ( = = 2), 1.1 ( \( \frac{1}{2} \), \( \frac{1}{2} \)	1
Indicator	Control	CG n=202	OG <sub>1</sub> n=10	OG <sub>2</sub> n=9
CI <sub>HR</sub>	$G_1$ $G_2$ $G_3$	0,91 (0,56; 2,43) 2,05 (1,02; 4,15)** 1,21 (0,65; 2,62)	0,75 (0,44; 1,68) 4,27 (1,03; 15,73) <sup>@**</sup> 0,75 (0,60; 2,15)	0,75 (0,34; 1,88) 0,44 (0,32; 0,55) <sup>@@##*</sup> 0,61 (0,39; 1,44) <sup>@#</sup>
$\mathrm{CI}_{\mathrm{SBP}}$	$G_1$ $G_2$ $G_3$	2,92 (1,43; 5,69) 3,73 (1,86; 7,78)* 2,88 (1,13; 6,42)	1,37 (0,70; 1,88) <sup>@</sup> 7,90 (4,02; 10,37) <sup>@**</sup> 2,30 (0,90; 4,65) <sup>*</sup>	3,99 (3,26; 11,32) <sup>@##</sup> 2,42 (2,13; 4,42) <sup>#*</sup> 2,44 (1,39; 3,37)*
$\mathrm{CI}_{\mathrm{DBP}}$	$G_2$	4,63 (2,33; 10,07) 8,37 (4,14; 13,68)* 4,74 (2,79; 11,94)	1,63 (1,54; 3,39) <sup>@</sup> 11,87 (7,25; 21,03) <sup>**</sup> 10,73 (3,30; 18,50) <sup>@**</sup>	2,38 (1,27; 6,46) 8,48 (4,38; 12,92)* 1,42 (0,60; 2,09) <sup>@@##*</sup>
$\mathrm{BR}_{\mathrm{LF}}$	$G_1$	17,5 (11,2; 27,8) 10,0 (5,0; 17,2)** 18,0 (12,8; 29,0)	24,7 (10,1; 43,6) 10,0 (4,6; 11,3)* 13,8 (12,4; 16,6) <sup>@*</sup>	34,2 (25,0; 47,7) <sup>@#</sup> 18,8 (16,3; 38,8) <sup>@##*</sup> 22,6 (15,7; 43,2) <sup>#*</sup>
$\mathrm{BR}_{\mathrm{HF}}$	$G_2$	25,3 (12,8; 38,5) 10,8 (5,6; 20,8)** 22,2 (12,7; 38,4)	27,3 (12,4; 32,6) 9,8 (2,9; 20,2)* 12,8 (11,0; 14,1) <sup>@*</sup>	51,9 (38,6; 111,9) <sup>@@##</sup> 27,2 (19,2; 72,6) <sup>@@##*</sup> 34,8 (28,7; 45,5) <sup>@##*</sup>

@ - p< 0.05, @@ - p< 0.01 between  $OG_1$ ,  $OG_2$  in comparison with  $CG^{\#}$  - p< 0.05,  $^{\#\#}$  - p< 0.01,  $^{\#\#}$  - p< 0.001, between  $OG_1$  and  $OG_2^{**}$  - p< 0.05,  $^{**}$  - p< 0.01, between  $G_3$  and  $G_2$  in comparison with  $G_1$ 

It amounts that according to the integral indicators of the cardiovascular system, there is a fairly clear differentiation of the mechanisms of sympathetic and parasympathetic overstrain of the cardiovascular system at all stages of monitoring.

### Discussion

For better representativeness of the differences in indicators' BPV in all experimental groups, significant differences in the dynamic changes directions in comparison with the initial state  $(G_1)$  are schematically and presented in Table 5. The way the results are presented makes it possible to visualize significant values of BPV indices' dynamics without taking into account differences in the initial state. For each of the groups, the directions of changes in indicators are presented: an increase  $(\uparrow)$ , a decrease  $(\downarrow)$ , or the absence of significant changes (=), confirmed by Wilcoxon criteria with varying reliability (+ - p < 0.05, ++- p < 0.01). At the same time, the differences between the measurements after the training load are analyzed in comparison with the initial data  $(G_2-G_1)$ , as well as the differences between the next morning measurements as compared with the initial data  $(G_3-G_1)$ . That is, Table 6 shows the directions of changes in BPV indicators, characterizing the effect of physical activity, as well as the changes reflecting the recovery process.

The effect of physical activity on CHF (according to the SBPV indices) in CG is characterized by an increase in the activity on myocardium in the low frequency range, which is consistent with an increase in the activity of the sympathetic branch of the ANS. This was not observed in the experimental groups (OG<sub>1</sub> and OG<sub>2</sub>). Moreover, with the formation of sympathetic overstrain (in OG<sub>1</sub>) a pronounced decrease in highfrequency influences (HF<sub>SRP</sub>) was observed; where as parasympathetic overstrain (OG<sub>2</sub>) was connected with redistribution of relative contributions only, indicating the predominance of high-frequency influences. Taking into consideration that the measurements were carried out in the first 5-7 minutes after physical load, it can be assumed that there is an extremely rapid increase in the activity of high-frequency influences on the CHF in OG<sub>2</sub>, and on the SBPV accordingly. It is confirmed by a typical change in the centralization indices of HR regulation (CI<sub>HR</sub>) and SBP (CI<sub>SBP</sub>), which significantly decrease during parasympathetic overstrain formation (OG<sub>2</sub>) at G<sub>2</sub>, in contrast to CG and OG<sub>1</sub>. A typical feature of the response to load in OG<sub>2</sub> is also an increase in regulatory influences on DBPV (TPDBP), which is characterized by an increase in suprasegmental influences (VLF<sub>DBP</sub>) against the background of a stable decrease in low-frequency (LF<sub>DBP</sub>) and high-frequency influences (HF<sub>DBP</sub>), which are recorded before the next morning after the load, but this time already against the background of a decrease in VLF<sub>DBP</sub>. This is reflected in the dynamics of changes in CI<sub>SBP</sub>, which significantly increases after exercise, and decreases significantly by the next morning. In this case, in OG2, we can assume pronounced decreases in the regulatory effects on CHF and vascular tone during the formation of parasympathetic overstrain. The latter can have significant adverse consequences in the form of CHF discoordination. It is also noteworthy that the baroreceptor sensitivity in OG<sub>2</sub>, in contrast to CG, is not restored to its initial values by the morning.

More explainable are the changes accompanying the formation of sympathetic overstrain  $(OG_1)$ , which are characterized by the persistence of the predominance of low-frequency influences on SBP and DBP due to a decrease in high-frequency influences that persist until the next morning. The latter is also characterized by a stable predominance of central influences on CHF and vascular tone and is accompanied by a decrease in baroreceptor sensitivity.

Table 5

Dynamics of changes in the indicators of SBPV and DBPV athletes in the post-exercise and recovery periods with the development of oversrains in sympathetic (OG<sub>1</sub>) and parasympathetic (OG<sub>2</sub>) types

	CG	CG				$OG_1$				$OG_2$			
Indicator	G <sub>2</sub> - 0	G <sub>2</sub> - G <sub>1</sub>		G <sub>3</sub> - G <sub>1</sub>		G <sub>2</sub> - G <sub>1</sub>		G <sub>3</sub> - G <sub>1</sub>		G <sub>2</sub> - G <sub>1</sub>		$G_1$	
	H	р	Н	p	H	p	Н	p	Н	р	H	p	
IAB, c.u.	1	++	=		1	++	1	++	=		$\downarrow$	+	
IAPR, c.u.	<b>↑</b>	++	=		1	++	1	++	1	++	1	+	
RMSSD, ms	$\downarrow$	++	1	+	$\downarrow$	++	$\downarrow$	++	1	+	=		
VLF, ms <sup>2</sup>	1	++	=		$\downarrow$	++	$\downarrow$	++	$\downarrow$	++	<b>↑</b>	++	
TP <sub>SBP</sub> , mmHg <sup>2</sup>	1	+	=		=		=		Ė		Ė		
VLF <sub>SBP</sub> , mmHg <sup>2</sup>	=		=		=		=		=		=		
$LF_{SBP}$ , mm $Hg^2$	1	++	1	+	=		=		=		=		
LF <sub>SBP</sub> n, n.u.	=		=		1	++	<b>↑</b>	++	$\downarrow$	+	$\downarrow$	+	
HF <sub>SBP</sub> , mmHg <sup>2</sup>	=		=		<b></b>	++	=		=		1	++	
HF <sub>SBP</sub> n, n.u.	$\downarrow$	+	=		$\downarrow$	++	$\downarrow$	+	<b>↑</b>	+	<b>↑</b>	++	
LFHF <sub>SBP</sub> , c.u.	1	++	=		1	++	<b>↑</b>	++	$\downarrow$	+	$\downarrow$	++	
$TP_{DBP}$ , $mmHg^2$	=		=		=		$\downarrow$	+	<b>↑</b>	+	$\downarrow$	++	
VLF <sub>DBP</sub> , mmHg <sup>2</sup>	=		=		=		=		1	+	$\downarrow$	+	
LF <sub>DBP</sub> , mmHg <sup>2</sup>	1	++	=		=		$\downarrow$	+	$\downarrow$	+	$\downarrow$	++	
LF <sub>DBP</sub> n, n.u.	1	++	1	+	1	++	<b>↑</b>	++	=		=		

$HF_{DBP}$ , $mmHg^2$	=		=	$\downarrow$	++	$\downarrow$	+	Ţ	++	Ţ	+
HF <sub>DBP</sub> n, n.u.	↓	++	=	↓	++	↓	++	=		<b>↑</b>	+
LFHF <sub>DBP</sub> , c.u.	1	++	=	1	++	1	++	=		Ţ	+
CIHR, c.u.	1	++	=	<b>↑</b>	++	=		$\downarrow$	+	=	
CI <sub>SBP</sub> , c.u.	1	+	=	1	++	1	+	$\downarrow$	+	<b></b>	+
CI <sub>DBP</sub> , c.u.	1	+	=	1	++	1	++	1	+	<b></b>	+
BR <sub>LF</sub> , ms / mmHg	↓	++	=	<b></b>	+	<b></b>	+	<b>↓</b>	+	<b>↓</b>	+
BR <sub>HF</sub> , ms / mmHg	Ţ	++	=	Ţ	+	Ţ	+	Ţ	+	Ţ	+

In general, the complexity of the interpretation of the results obtained is stipulated by a variety of mechanisms aimed at systemic hemodynamics maintaining. The noted features of adjusting regulatory influences however can further help to reveal more subtle mechanisms underlying the development of overstrain, especially in the parasympathetic type, which, according to many researchers, is a prerequisite for a sudden cessation of blood circulation in highly qualified athletes.

### Conclusion.

The studies allowed us to ascertain that the parameters of systolic and diastolic pressure variability during the development of sympathetic and parasympathetic overstrain in highly qualified athletes significantly complement the results of the analysis of heart rate variability indicators. Overstrain of the sympathetic type in the post-exercise and recovery period is characterized by the following:

- 1. Stable predominance of low-frequency and central influences on the contractile heart function immediately after exercise and the next day after exercise;
- 2. A pronounced decrease in the activity of influences on the contractile heart function in the high-frequency range immediately after exercise;
- 3. Moderate decrease in regulatory effects on vascular tone the next day after exercise;
- 4. Stable predominance of low-frequency and central influences on vascular tone immediately after exercise and the next day after exercise;
- 5. A steady decrease in baroreceptor sensitivity in the high and low frequency ranges immediately after exercise and the next day after exercise.

The following tendencies are characteristic for overstrain of the parasympathetic type in the post-exercise and recovery period:

- 1. Stable predominance of high-frequency influences on the contractile heart function immediately after exercise, which increases even more by the next day;
- 2. Moderate increase in regulatory influences on vascular tone after exercise at the expense of high-frequency rhythms, followed by a pronounced decrease at the expense of rhythms in very-low-frequency, high-frequency and low-frequency ranges by the next morning;
- 3. An increase in central influences on vascular tone immediately after exercise, followed by a decrease by the next morning;
- 4. A steady decrease in baroreceptor sensitivity immediately after exercise and the next day after exercise in comparison with the initial state.

### **Conflicts of interest**

Authors have declared that no competing interests exist.

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......2631

OKSANA GOZII, ALEXANDER ROMANCHOR, ANATOLIT MAHLOVANTI, VOLODIMIR TRACII

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