# **Original research article**

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# Multifunctional changes in the athletes' body during the formation of autonomic regulations' overstrain under the influence of training load

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DOI: https://doi.org/10.15391/prrht.2023-8(2).03

🛗 Received: 14.05.2023

🛗 Accepted: 29.05.2023

🛗 Published: 30.06.2023

#### Citation:

Guzii, O., Mahlovanyi, A., & Romanchuk, O. (2023). Multifunctional changes in the athletes' body during the formation of autonomic regulations' overstrain under the influence of training load. *Physical rehabilitation and recreational health technologies*, 8(2), 91-104. https://doi.org/10.15391/prrht.2023-8(2).03

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# Abstract

**Purpose:** the purpose of this study was to generalize polysystemic changes that occur in the body of highly qualified athletes with signs of autonomic regulation overstrain under the influence of training load and during the recovery period after it.

**Material & Methods:** test subjects were qualified male athletes (N=202) aged 22.6±2.8 years, who were examined by means of spiroarteriocardiorhythmography (SACR) and computerized motion meter (CMM). In accordance with the design the examinations with the aid of SACR and CMM were carried out three times: in the morning, on an empty stomach, in a sitting position on the day of training (G<sub>1</sub>), immediately after training (during the first 5-7 minutes) (G<sub>2</sub>) and the next day in the morning after sleep (G<sub>3</sub>).

**Results:** according to the data of the examination of athletes in the dynamics of recovery after the training load, options of changes in HRV indicators were determined, which indicated the formation of sympathetic and parasympathetic overstrain in athletes. Two groups were created. The first included 10 athletes, the second included 9 athletes. The analysis of changes in indicators, the research methods used, allowed us to establish differences in their dynamics during the formation of sympathetic and parasympathetic overstrain. Differences were noted among 18 indicators. Among them are indicators of HRV – ABI (c.u.), SRAI (c.u.), ARI (c.u.), RMSSD (ms), VLF (ms<sup>2</sup>), LFHF (ms<sup>2</sup>/ms<sup>2</sup>); variability of arterial pressure – LF<sub>SBP</sub>n (n.u.), HF<sub>SBP</sub>n (n.u.), LFHF<sub>SBP</sub> (mmHg<sup>2</sup>), TP<sub>DBP</sub> (mmHg<sup>2</sup>), LF<sub>DBP</sub>, (mmHg<sup>2</sup>), IC<sub>DBP</sub> (mmHg<sup>2</sup>/mmHg<sup>2</sup>); respiratory variability – TP<sub>R</sub> (L×min<sup>-1</sup>)<sup>2</sup>; of sensorimotor function – SCSleft (s), SMleft (%).

**Conclusions:** the obtained results on the formation of sympathetic and parasympathetic overstrain under the influence of intense physical activity indicate that changes in the autonomic regulation of the heart rate, which determine the type of heart rate regulation, are accompanied by a number of changes in hemodynamics, autonomic regulation of the pumping function of the heart, vascular tone, respiratory regulation, and sensorimotor function. The determined differential dynamics of changes in cardiorespiratory and sensorimotor indicators will allow further algorithmization of the assessment of the functional state of the athletes' body in order to detect early states of non-functional overstrain and overtraining.

**Key words:** athletes, overstrain, heart rate variability, blood pressure variability, respiratory variability, hemodynamics, sensomotorics.

# Анотація

Поліфункціональні зміни в організмі спортсменів при формуванні перенапруження автономної регуляції за впливу тренувального навантаження

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Мета: метою даного дослідження було узагальнити поліфункціональні зміни, які відбуваються в організмі спортсменів вищої кваліфікації з ознаками формування перенапруження вегетативної регуляції під впливом тренувального навантаження та в період відновлення після нього.

**Матеріал і методи:** під наглядом були кваліфіковані спортсмени чоловічої статі (N=202) віком 22,6±2,8 років, яких обстежували за допомогою спіроартеріокардіоритмографії (САКР) та комп'ютеризованого вимірювача руху (КВР). Відповідно до плану обстеження за допомогою САКР і КВР проводили тричі: вранці, натщесерце, в положенні сидячи в день тренування (G<sub>1</sub>), одразу після тренування (протягом перших 5-7 хвилин) (G<sub>2</sub>) і наступного дня вранці після сну (G<sub>3</sub>).

Результати: за даними обстеження спортсменів в динаміці відновлення після тренувального навантаження були визначені варіанти змін показників ВСР, які свідчили про формування у спортсменів перенапруження за симпатичним і парасимпатичним типом. Були створені дві групи. До першої увійшли 10 спортсменів, до другої – 9 спортсменів. Аналіз змін показників, використаних методів дослідження, дозволив встановити відмінності ïΧ динамік при формуванні симпатичного та парасимпатичного Відмінності пренапруження. відзначались серед 18 показників. Серед них - показники варіабельності серцевого ритму – ABI (с.u.), SRAI (c.u.), ARI (c.u.), RMSSD (ms), VLF (ms2), LFHF (ms<sup>2</sup>/ms<sup>2</sup>); варіабельності артеріального тиску – LF<sub>SBP</sub>n (n.u.), HF<sub>SBP</sub>n (n.u.), LFHF<sub>SBP</sub> (mmHg<sup>2</sup>), TP<sub>DBP</sub> (mmHg<sup>2</sup>), LF<sub>DBP</sub>, (mmHg<sup>2</sup>), IC<sub>DBP</sub> (mmHg<sup>2</sup>/mmHg<sup>2</sup>); гемодинаміки – CO (dm<sup>3</sup>), GPVR, (dyn/s/cm<sup>-5</sup>), CI (dm<sup>3</sup>/m<sup>2</sup>); сенсомоторної функції – SCSleft (s), SMleft (%).

Висновки: отримані результати щодо формування симпатичного та парасимпатичного перенапруження під впливом інтенсивного фізичного навантаження свідчать про те, що зміни вегетативної регуляції серцевого ритму, на основі яких визначається тип регуляції серцевого ритму, супроводжуються низкою змін систем гемодинаміки, автономної регуляції насосної функції серця, тонусу сурегуляції дихання та сенсомоторної дин, функції. Визначена диференціальна динаміка кардіореспіраторних та сенсомоторзмін них показників дозволить у подальшому алгоритмізувати оцінку функціонального стану організму спортсменів з метою раннього виявлення станів нефункціонального перенапруження та перетренованості.

Ключові слова: спортсмени, перенапруження, варіабельність серцевого ритму, варіабельність артеріального тиску, варіабельність дихання, гемодинаміка, сенсомоторика.

### Introduction

Sports activity is accompanied by intense, longterm, volumetric loads of a specific direction in conditions of increased psychological stress, the goal of which is to achieve the maximum possible results for the athlete (Adle et al., 2008; Angelova, et al., 2021; Meeusen et al., 2013; Saw et al., 2016; Goldstein 2010). Excessive physical load, competitive activities, impaired reactivity and resistance of the body reduce the adaptive capabilities of the athletes' body and often lead to health disorders (hypertensive syndrome, injuries and diseases of the musculo-sceletal system, respiratory diseases, abnormalities in the work of the gastrointestinal tract, skin pathologies) (Ackel-D'Elia et al., 2010; Djaoui et al., 2017; Migliaccio et al.; 2023). In athletes, even minor deviations of homeostasis parameters have a negative effect on the effectiveness of sports and can even cause premature termination of the athlete's career and early disability (de Carvalho e Silva et al., 2022; Armstrong et al., 2022). Therefore, prevention of exhaustion and restoration of adaptive capabilities of the athlete's body, rehabilitation of homeostasis violations with the development of individual means taking into account the "autonomic passport", the reactivity of the body, the leading pathological syndrome and its form, as well as the typical clinical syndrome as a manifestation of organ failure is quite important (Meeusen et al., 2013; Saw et al., 2016; van Hattum et al., 2022). Violation of homeostasis parameters changes susceptibility to certain types of diseases. With an adequate reaction of the athlete's body to various physical loads, a normorergic response is observed - a joint response of the nervous, hormonal, immune and humoral systems that make up the "square of homeostasis" (Seiler et al., 2007; Jurasz et al., 2022). Violation of reactivity is manifested by dysregulation with the predominance of one or several chains of regulation, which causes low resistance of the organism to the action of the stimulus (Meeusen et al., 2013; Le Meur et al., 2013; Guzii et al., 2019; Kalauzi et al., 2023; Karemaker, 2020).

The development of maladaptation symptoms is facilitated by the accumulation of under-recovery processes. Prolonged under-recovery can contribute to the development of physical overstrain and reduced work capacity (Djaoui et al., 2017; Meeusen et al., 2013; Bourdon et al., 2017; Armstrong et al., 2022).

Therefore, one of the first tasks in preventing the development of pre-pathological and pathological conditions in athletes is the timely diagnosis of weak areas and early symptoms of maladaptation (Da Silva et al., 2015; Saw et al., 2016; Hoffmann et al., 2020; Rosei et al., 2020; Qammar et al., 2022). The second task in the prevention of pathological conditions is the organization of rehabilitation measures (Meeusen et al., 2013; Dupuy et al., 2018). The third task is to eliminate the weak links of adaptation, in those cases when rehabilitation

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means do not give the necessary result, by correcting the training process and its individualization (Boloban et al., 2006; Bourdon et al., 2017; Crawford et al., 2020).

In order to understand the mechanisms of adaptation failure and to select optimal schemes for conducting physical therapy of athletes who perform heavy physical loads, it is necessary to identify criteria for early diagnosis of overstrain states and develop an algorithm for integrating the use of physical rehabilitation tools into the structure of training athletes (Meeusen et al., 2013; Dupuy et al., 2018; Gronwald & Hoos, 2020; Bourdillon et al., 2019).

Diagnosis of overstrain is a complex task and should be based on the following principles: the complex nature of the examination; measurements of the same type, which are repeated many times, under identical conditions; the sensitivity of the diagnostic marker to physical load; to its relatively easy measurement (Meeusen et al., 2013; Abreu et al., 2019, 2022; Christiani et al., 2021). Also important is the low cost and non-invasiveness of the examination (Baumert et al., 2006; Carlén et al., 2022; Guzii & Romanchuk, 2018; Volpes et al., 2022).

The autonomic nervous system is the system that ensures the course of restorative processes in the body along with metabolism. According to many researchers, its condition determines the physiological mechanisms of recovery during the postloading period (Grässler et al., 2021; Hunter et al., 2018; Christiani et al., 2021; Guzii & Romanchuk, 2017, 2021; Tipton et al., 2017; Stanley et al., 2013). At this time, a number of processes aimed at strengthening, counteracting and eliminating the consequences that occur during physical load take place. Some of them contribute to increasing the athlete's training level and mobilizing various functional systems to the existing influences caused by the direction of the training process, while others are negative and can cause overfatigue and overstrain in a number of functional systems involved in the body's adaptation mechanisms (Fadel, 2008; Fuchs et al., 2010; Crollen et al., 2017; Incognito et al., 2020; Matić et al., 2022).

The purpose of this study was to generalize polysystemic changes that occur in the body of highly qualified athletes with signs of autonomic regulation overstrain under the influence of training load and during the recovery period after it.

### Material and methods of research

### Participants

Test subjects were qualified male athletes (N=202) aged 22.6 $\pm$ 2.8 years, who were examined by means of spiroarteriocardiorhythmography (SACR) and computerized motion meter (CMM). The athletes represented acyclic sports, such as martial arts (karate, taekwondo, kickboxing, boxing, wrestling, judo, sambo) and ball games like water polo and soccer. The experience of the involvement in

#### Methods

To determine cardiorespiratory station the "Spiroarteriocardiorhythmograph" device (SACR) was applied, recommended for use in medical practice by the Ministry of Health of Ukraine (Panenko et al., 2006). 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).

ECG recording in 1 lead allowed to determine the indicators of heart rate variability (HRV) according to the spectral analysis of the sequence of RR intervals is total power (TP, ms<sup>2</sup>), power in the very low frequency range (VLF, ms<sup>2</sup>), power in the low frequency range (LF, ms<sup>2</sup>) and power in the high frequency range (HF, ms<sup>2</sup>) and their derivatives (LFn, n.u., HFn, n.u., LF/HF) (Guzii & Romanchuk, 2017, 2018); according to the math analysis of the sequence of RR intervals is ABI (autonomic balance index, c.u.), SRAI (subcortical regulation adequacy indicator, c.u.), ARI (autonomic regulation index, c.u.), SI (stress index, c.u.), SDANN (standard deviation of the values of cardio intervals, ms), RMSSD (square root of the sum of squares of the differences in the values of consecutive pairs of normal intervals, ms), pNN50 (the percentage of NN50 from the total number of consecutive pairs of intervals that differ by more than 50 milliseconds, obtained over the entire time recording, %) (Heart rate variability:.., 1996); according to cardiointervalometry – to define the heart rate (HR, min<sup>-1</sup>), durations and intervals of PQRST-complex – P (s), PQ (s), QRS (s), QT (s), QTC (s), ST (n.u.); indicators of systemic hemodynamics (Kim et al., 2005; Romanchuk & Pisaruk, 2013) – end-diastolic volume (EDV, cm<sup>3</sup>), end-systolic volume (ESV, cm<sup>3</sup>), stroke volume (SV, cm<sup>3</sup>), cardiac output (CO, dm<sup>3</sup>), stroke index (SI, cm<sup>3</sup>/m<sup>2</sup>), cardiac index (CI, dm<sup>3</sup>/ m<sup>2</sup>), general peripheral vascular resistance (GPVR, dyn/s/cm<sup>-5</sup>); according to the pulse wave recording with the help of a photoplethysmographic sensor on the finger by the Penaz method (Penáz, 1992), blood pressure (SBP, mmHg; DBP, mmHg) and its variability (SBPV and DBPV) in ranges similarly to HRV were determined a total power of SBPV and DBPV (TP<sub>SBP</sub>, mmHg<sup>2</sup> and TP<sub>DBP</sub>, mmHg<sup>2</sup>), power in the very low-frequency range (VLF<sub>SBP</sub>, mmHg<sup>2</sup> and VLF<sub>DBP</sub>, mmHg<sup>2</sup>), power in the low-frequency range ( $\mathsf{LF}_{\mathsf{SBP}}$ , mmHg² and  $\mathsf{LF}_{\mathsf{DBP}}$ , mmHg²) and power in the high-frequency range (HF<sub>SBP</sub>, mmHg<sup>2</sup> and  $\begin{array}{l} \mathsf{HF}_{_{\mathsf{DBP'}}} \mbox{ mmHg}^2 ) \mbox{ and their derivatives} \ - \ \mathsf{LF}_{_{\mathsf{SBP}}}n, \ n.u., \\ \mathsf{HF}_{_{\mathsf{SBP}}}n, \ n.u., \ \mathsf{LF}/\mathsf{HF}_{_{\mathsf{SBP}}}n, \ n.u., \ \mathsf{HF}_{_{\mathsf{DBP}}}n, \ n.u., \ \mathsf{LF}/ \end{array}$ HF (Pinna, 1996; Wesseling et al., 2017; Papaioannou, et al., 2020). Additionally by using the spectral method we determined the index of arterial baroreflex sensitivity (BRS, ms/mmHg) a-coefficient, that was calculated in high  $(BRS_{HF})$ and low (BRS<sub>1</sub>) frequencies ranges (Karemaker et

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$$BRS_{IF} = \sqrt{LF_{HPV}} (ms^2)/LF_{SRP} (mmHg^2)$$
(1)

$$BRS_{HF} = \sqrt{HF_{HRV}} (ms^2)/HF_{SBP} (mmHg^2)$$
(2)

The ultrasonic sensor of the SACR device allows to measure flows of air on inspiration and expiration and to define the average parameters of a respiration pattern (PR): duration of inspiratory (Tin, s), duration of expiratory (Tex, s), tidal volume ( $V_{\tau}$ , L), volumetric inspiratory velocity (Vin, L×s<sup>-1</sup>), volumetric expiratory velocity (Vex,  $L \times s^{-1}$ ), the fraction of inspiration in the respiratory cycle (Ti/(Ti+Te) (c.u.), as well as the volume of minute respiration  $(V_{F})$ ; and calculate the parameters of respiration variability (RV): total power of respiration  $(TP_{R}, (L \times min^{-1})^{2})$ , respiration power in the very low-frequency range (VLF<sub>R</sub> (L×min<sup>-1</sup>)<sup>2</sup>), respiration power in the low-frequency range  $(LF_{R} (L \times min^{-1})^2)$ and respiration power in the high frequency range  $(HF_{R} (L \times min^{-1})^{2})$  and their derivatives –  $LF_{R}n$ ,  $HF_{R}n$ , LF/HF<sub>R</sub> – in n.u. (Bazhora & Romanchuk, 2018; Romanchuk & Guzii, 2020).

Indicators of frequency and volume synchronization of the cardio-respiratory system were also calculated – Hildebrandt index (IH) and the ratio of CO to  $V_{\rm F}$  (IVS) (Noskin et al., 2018).

 $IH = HR (min^{-1})/RR (min^{-1})$ (3)

$$IVS = CO (dm^3)/V_{F}(L)$$
(4)

All studies were carried out in a sitting position; the duration of registration of the cardiorespiratory system parameters was 2 minutes.

After that with the device computerized motion meter (CMM) was studied sensorimotor system state (Pivovarov, 2006). With the help of the CMM, based on the results of three simple motor tests performed with the right and left hand (Romanchuk, 2007), 25 digital movement parameters were determined: the duration of the movement cycle (DMC, s), switching of central settings (SCS, s), flexion and extension time (FT and ET, s), shortterm motor memory (STMM, s), reaction time to a sound stimulus (TRSS, s), reaction time to a light stimulus (TRLS, s), flexors correction error and extensors correction error (FCE and ECE), smoothness of movements (SM, %), balance of extensors and flexors with and without visual control (BEFvis and BEFwvis). Taking into account the test data, the coefficient of motor asymmetry (MA, %) is also calculated.

# Procedure

All the studies were carried out in the precompetitive period. In accordance with the design the examinations with the aid of SACR and CMM 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 SACR-registration lasted 2 minutes, CMM-registration – 8 minutes (Fig.). A morphometric study and routine blood pressure measurements with a tonometer were carried out before SACR application.

Based on the data obtained, also the following indices 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) were calculated.

#### Statistical analysis

The processing of the received results was carried out with the help of STATISTICA program for Windows (version 10.0), Microsoft Excel 2012. 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.

# **Results of the study**

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.) and 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.

The principles of classification of types, taking into account the above criteria, are presented in table 1.



Figure. Structure of polisystemic testing

**Table 1.** Criteria for determining types of heart rhythm regulation according to N.I. Shlyk

Types of heart rhythm regulation		Criteria				
	ŕ	SI (c.u.)	VLF (ms²)			
Dradominance of control regulation	Ι	>100	>240			
Predominance of central regulation	II	>100	<240			
Predominance of autonomous	III	25-100	>240			
regulation	IV	<25	VLF>500, TP>8000-10000			

The main hypothesis of this study was the determination of dynamic changes in the regulatory support of the heart rhythm, taking into accounts one or another type of regulation. In previous studies, an analysis was carried out, which made it possible to establish that, regardless of the initial type, type II is typical for all athletes in the post-loading period, and type III the next morning after training. Such options are guite physiologically expected and are explained by the high residual activity of the sympathetic nervous system immediately after exercise and the increased activity of the parasympathetic nervous system during the recovery period of the body (Guzii, 2019). From these positions, other options for changes in the regulatory support of the heart rhythm can be considered as atypical.

In our opinion, the long-term preservation of heart rate regulation, which is observed after exposure to the relevant factor (in our case, from intense training load) and differs from the physiologically determined one, may be related to the phenomena of overstrain of the autonomous heart rate regulation and the body as a whole.

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 options of changes in HR regulation, which are as follows:

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

option 2 testified 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 variant was recorded in 9 cases and indicated the parasympathetic type of overstrain.

The subjects mentioned above made up 2 groups of observation (OG),  $OG_1$  and  $OG_2$  respectively. The control group (CG) consisted of all 202 studied athletes.

The characteristics of morphological and functional parameters determined before the training load  $(G_1)$  are presented in Table 2. 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 the mentioned parameters, the level of the functional state of the cardiorespiratory system of highly qualified athletes (CG) could be characterized as high or above average.

The morpho-functional indicators of athletes at rest, which were measured before intense physical activity, in which, under the influence of training load, changes in the autonomous regulation of heart rate occurred that could indicated the development of sympathetic ( $OG_1$ ) or parasympathetic ( $OG_2$ ) type of overstrain are presented in table 2. That is, the obtained data in a certain way can be considered as predicting the possibility of the formation of appropriate regulatory states.

Analyzing the morpho-functional differences in  $OG_1$ , first of all, it should be stated that the Kerdo index is not informative regarding the predominance of sympathetic influences on heart rhythm. Baevsky's AP and Pyrogova's PFL were also uninformative regarding sympathetic overstrain, although they had certain differences from  $OG_2$ . The indicators of fat content (%) attracted attention, which were significantly higher than in CG and did not differ from  $OG_2$ . Among the functional indicators, the indicator of diastolic blood pressure (DBP, mmHg) deserves more attention, which was significantly higher than in all groups.

Analyzing the results of  $OG_2$ , it should be noted that individuals with parasympathetic oversrain have significant morpho-functional differences from CG and  $OG_1$ . Most of all, they relate to lower BMI (kg/m<sup>2</sup>), contour chest (cm), contour hip (cm) and greater chest mobility (cm). Among the functional parameters, the most significant are the differences in the Kerdo index, the Double product, Baevsky's AP, and Pirogov's PFL, which characterize the pronounced economization of the cardiovascular system, predict high work capacity against the background of pronounced parasympatheticotonia.

**Table 2.** 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), M (Q<sub>1</sub>; Q<sub>3</sub>)

•		-55			
Indicator	CG n=202	OG <sub>1</sub> n=10	OG <sub>2</sub> 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)	23.6 (21.4; 27.3)	20.5 (19.9; 24.2) <sup>#@</sup>		
Body square, m <sup>2</sup>	1.92 (1.74; 2.04)	2.02 (1.70; 2.18)	1.85 (1.74; 2.03)#		
Contour chest (rest), cm	96.0 (91.0; 101.0)	98.5 (89.0; 113.0)	91.0 (90.0; 96.0) <sup>#@</sup>		
Chest mobility, cm	7.0 (5.0; 8.0)	8.5 (7.0; 10.0)®	8.0 (7.5; 9.0)®		
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>		
SI, %	64.4 (59.5; 68.9)	64.5 (51.1; 77.3)	66.3 (55.6; 68.9)		
VLC, ml	4800 (4400; 5600)	4850 (4400; 6600)	4850 (4500; 4900)		
VI, ml×kg <sup>-1</sup>	67.9 (61.9; 73.1)	65.2 (62.9; 70.2)	69.3 (59.3; 73.8)		
Fat, %	11.8 (8.7; 18.1)	18.4 (8.1; 19.0) <sup>@</sup>	13.3 (6.5; 20.3)		
SBP, mmHg	120 (110; 130)	115.0 (110.0; 120.0)®	115.0 (100.0; 120.0)®		
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)	-0.27 (-0.59; -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) <sup>##@</sup>		
PFL by Pirogova, c.u.	0.746 (0.672; 0.822)	0.736 (0.692; 0.762)	0.823 (0.753; 0.901) <sup>#@</sup>		

 $^{\circ}$  – p<0.05, between OG<sub>1</sub> and OG<sub>2</sub> in comparison with CG;

 $^{*}$  - p<0.05;  $^{**}$  - p<0.01, between OG<sub>2</sub> and OG<sub>1</sub>

...

**Table 3.** Changes in HRV parameters, which determine the type of heart rate regulation under the influence of intense physical load, M ( $Q_1$ ;  $Q_3$ )

Indicator	Control point	CG n=202	OG, n=10	OG <sub>2</sub> n=9			
	$G_1$	5098 (2798; 12679)	6766 (4135; 9960)	28115 (19016; 31258) <sup>@@###</sup>			
TP, ms <sup>2</sup>	G <sub>2</sub>	1858 (708; 4624)**	984 (306; 2333) <sup>@**</sup>	9414 (5550; 13502) <sup>@@###**</sup>			
	$G_3$	5550 (3036; 12656)	1621 (708; 5550) <sup>@*</sup>	13880 (11859; 17902) <sup>@###**</sup>			
	$G_1$	778.4 (292.4; 1528.8)	661 (484; 3505)	1226 (986; 1529) <sup>@#</sup>			
VLF, ms <sup>2</sup>	G <sub>2</sub>	204.5 (104.0; 605.2)**	151 (88; 207) <sup>@**</sup>	789 (458; 999) <sup>@###**</sup>			
	G3	795.2 (357.2; 1413.8)	130 (102; 190) <sup>@@**</sup>	3385 (1798; 3685) <sup>@@###**</sup>			
	$G_1$	59.7 (26.2; 117.4)	60.9 (22.9; 76.0)	19.0 (17.5; 20.6) <sup>@@##</sup>			
SI, c.u.	$G_2$	253.2 (78.8; 675.2)**	503.6 (262.1; 2035.0) <sup>@**</sup>	28.9 (18.2; 34.0) <sup>@@###*</sup>			
	G <sub>3</sub>	57.2 (29.8; 115.1)	262.5 (160.1; 362.0) <sup>@@**</sup>	18.1 (13.4; 20.9) <sup>@@###</sup>			

• p<0.05, • p<0.01, between OG, and OG, in comparison with CG;
</p>

\* - p<0.05, \*\* - p<0.01, \*\*\* - p<0.001, between OG, and OG,;

\* – p< 0.05, \*\* – p<0.01, between G3 and G2 in comparison with G1

Table 3 presents the dynamics of changes in HRV indicators, which are the basis for differentiating types of regulation. As can be seen from the CG data, after exercise there is an activation of the sympathetic regulatory link, which is reflected by a decrease in TP ( $ms^2$ ) and VLF ( $ms^2$ ) against the background of an increase in SI (c.u.). The next morning after training, these indicators return to the initial level.

During the formation of sympathetic overstrain  $(OG_i)$ , there is a more significant decrease in TP  $(ms^2)$  and VLF  $(ms^2)$  against a more pronounced increase in SI (c.u.) after exercise. However, the

following morning, these indicators do not return to their initial values.

During the formation of parasympathetic overstrain  $(OG_2)$ , there is also a decrease in TP  $(ms^2)$  and VLF  $(ms^2)$  against the background of an increase in SI (c.u.). The next morning, on the background of the return to the initial values of SI (c.u.), the tendency to return of TP  $(ms^2)$ , there is a significant excess of the initial values of VLF  $(ms^2)$ .

In the table 4 presents indicators of HRV, SBPV, DBPV, central hemodynamics, variability of breathing and sensorimotor function, the changes of

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which in the dynamics of recovery after intense training load in the studied groups were clearly differentiated.

Given the fact that the basis of the formation of groups was the specifics of changes in HRV, which determined the tendency to develop sympatheticor parasympatheticotonia, HRV indicators were the most differentiated. Out of the thirteen indicators that we obtained during the HRV analysis, dynamic differentiation was noted only in six. Among them are VLF (ms<sup>2</sup>), LFHF (ms<sup>2</sup>/ms<sup>2</sup>), ABI (c.u.), SRAI (c.u.), ARI (c.u.), RMSSD (ms). A more complete analysis of the obtained changes in HRV indicators was carried out by us earlier (Guzii et al., 2020). These indicators have been shown to demonstrate larger subtle mechanisms associated with the activation of various links of heart rhythm regulation. As an example, the RMSSD indicator (ms), the values of which change in the opposite direction during the formation of parasympathetic type overstrain, which confirms the known data about its importance in the assessment of the condition of athletes (Bresciani et al., 2011; Dupuy et al., 2018; Bellenger et al., 2021), as well as in the assessment of recovery, when its steady decrease in sympathetic overstrain is noted and return to baseline values with parasympathetic overstrain. Informative are changes in the parameter ABI (c.u.), which, increasing after exercise, remains at the same level the next morning after training, with sympathetic overstrain, and with parasympathetic overstrain, it changes little under the influence of exercise and significantly decreases after rest. Changes in the SRAI indicator (c.u.) were also significant, which, after a pronounced increase after exercise  $(G_2)$ , remains moderately elevated the next morning after training  $(G_3)$  in comparison with the initial state in both variants of overstrain, which proves the presence of residual tension of subcortical mechanisms. Indicative is the activation of suprasegmental structures (according to the VLF indicator) during parasympathetic overstrain, which can characterize the excessive activation of regulatory mechanisms of metabolism, which can indicate the development of functional asymmetry in the cortical structures of the brain (Machado et al., 2010; Swart & Constantinou, 2023; Romanchuk & Guzii, 2020). That is, the assessment of the dynamics of changes in HRV parameters made it possible to establish several parameters, the dynamics of which changes can more clearly characterize an overstrain of autonomic regulation of the cardiovascular system.

Our earlier analysis of changes in nineteen indicators of blood pressure variability – SBPV and DBPV, as well as their derivatives, allowed us to establish that the formation of sympathetic and parasympathetic overstrain has sufficiently clear determinants in the changes of a number of indicators (Guzii et al., 2021; Guzii & Romanchuk, 2021). It was shown that the formation of sympathetic type overstrain is accompanied by a persistent predominance of low-frequency and central effects on the contractile function of the heart immediately after exercise and the day after exercise; a pronounced decrease in the activity of influences on the contractile function of the heart in the high-frequency range immediately after exercise; a moderate decrease in regulatory effects on vascular tone the next day after exercise; persistent predominance of low-frequency and central effects on vascular tone immediately after exercise and the next day 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 formation of parasympathetic overstrain is accompanied by a persistent predominance of high-frequency effects on the contractile function of the heart immediately after exercise, which increase even more until the next morning; a moderate increase in the regulatory effects on vascular tone after exercise due to very-low-frequency rhythms, which is replaced by a pronounced decrease until the next morning due to rhythms in the verylow-frequency, high-frequency and low-frequency ranges; an increase in central effects on vascular tone immediately after exercise, which is replaced by their decrease the next morning; persistent decrease in baroreceptor sensitivity immediately after exercise and the next day after exercise compared to the initial state. From the standpoint of differentiating changes during the formation of sympathetic and parasympathetic overstrain, the LFSBPn (n.u.), HF<sub>SBP</sub>n (n.u.), LF/HF<sub>SBP</sub> (mmHg<sup>2</sup>/ mHg<sup>2</sup>), TP<sub>DBP</sub> (mmHg<sup>2</sup>), LF<sub>DBP</sub> (mmHg<sup>2</sup>), IC<sub>DBP</sub> (c.u.) indicators were informative, the dynamics of which are presented in table 3.

Important indicators of the body functional state are the indicators of systemic hemodynamics and its regulation. In the table 3 presents the hemodynamic indicators, which were differentiated themselves during sympathetic and parasympathetic overstrain. A characteristic feature of the impact of the load is the lack of recovery of CO (dm<sup>3</sup>), GPVR (dyn/s/cm<sup>-5</sup>) and CI (dm<sup>3</sup>/m<sup>2</sup>) indicators to the initial values the next morning in OG<sub>1</sub>. The latter testifies to the excess of sympathicotonic effects on the hemodynamic system. In OG<sub>2</sub>, the process of the hemodynamic indicators recovery is also somewhat slowed down. However, it may be important in the further differentiation of athletes who have this type of response to intense physical activity (Guzii & Romanchuk, 2017).

Taking in to consideration the primacy of the respiratory circuit of HRV regulation (Eckberg et al., 1999), we propose to investigate the volume variability of spontaneous breathing, because it is an integral indicator that simultaneously takes into account the frequency and volume components of breathing (Anderson & Ramirez, 2017; Shams et al., 2022). This indicator can be compared with the parameter of the minute volume of breathing, which is one of the main indicators of the power of breathing, both at rest and under various loads. However, it does not provide an opportunity to distinguish the multi-frequency characteristics of

Table 4. Differentially significant changes in indicators of the cardiorespiratory and sensorimotor systems under the influence of training load during the formation of sympathetic and parasympathetic overstrain

Indicator Undicator		CG n=202	OG, n=10	OG <sub>2</sub> n=9		
		Heart Rat	e Variabiliy			
	G,	778.4 (292.4; 1528.8)	661 (484; 3505)	1226 (986; 1529) <sup>@#</sup>		
VLF, ms <sup>2</sup>	G	204.5 (104.0; 605.2)**	151 (88; 207)®**	789 (458; 999) <sup>@###**</sup>		
-	G,	795.2 (357.2; 1413.8)	130 (102; 190) <sup>@@**</sup>	3385 (1798; 3685) <sup>@@###*</sup>		
	G,	0.64 (0.25; 1.44)	0.49 (0.25; 0.64)@	0.65 (0.25; 1.69)		
LFHF, ms <sup>2</sup> /ms <sup>2</sup>	G	1.44 (0.49; 2.89)**	3.49 (0.64; 11.56) <sup>@**</sup>	0.31 (0.16; 0.49)@@##*		
	G,	0.81 (0.36; 1.69)	0.64 (0.49; 1.69)*	0.25 (0.16; 0.64) <sup>@#*</sup>		
	G,	13.0 (5.3; 21.0)	11.2 (4.6; 14.0) <sup>@</sup>	4.4 (3.6; 5.3) <sup>@@#</sup>		
ABI, c.u.	G,	33.0 (10.5; 85.8)**	63.4 (36.8; 219.8) <sup>@**</sup>	5.0 (2.7; 5.8) <sup>@@###</sup>		
	 G,	11.2 (6.7; 21.7)	47.5 (28.5; 60.1) <sup>@@**</sup>	3.6 (2.4; 4.2) <sup>@@###*</sup>		
	 G,	3.1 (2.0; 4.4)	3.0 (2.2; 3.9)	1.2 (1.1; 2.0) <sup>@@###</sup>		
SRAI, c.u.	G,	7.2 (3.5; 14.0)**	12.7 (8.4; 28.5)®**	2.5 (2.2; 2.7) <sup>@@###**</sup>		
	 G,	3.0 (1.9; 4.1)	6.5 (4.3; 9.4) <sup>@@**</sup>	1.7 (1.3; 2.0)@@###*		
	G,	4.0 (2.5; 5.9)	3.6 (2.1; 4.0)®	2.2 (2.0; 2.4)@@#		
ARI, c.u.	G <sub>2</sub>	8.3 (5.0; 15.4)**	12.0 (8.3; 26.5) <sup>@**</sup>	2.5 (2.2; 2.6) <sup>@@###*</sup>		
	G <sub>2</sub>	3.9 (2.6; 6.6)	9.4 (8.6; 10.3) <sup>@@**</sup>	1.9 (1.7; 2.3) <sup>@@###*</sup>		
	G,	60.8 (41.2; 94.1)	59.9 (50.6; 105.9)	139.3 (94.1; 157.9)@@#		
DMSSD mc	G,		16.6 (7.6; 36.4) <sup>@**</sup>	166.9 (105.3; 204.5)@@##		
RMSSD, ms _	2	32.0 (15.0; 76.8)**	27.6 (17.1; 34.5) <sup>@@**</sup>	126.3 (96.0; 243.4)@###		
	G3	73.6 (46.8; 105.0)*		120.3 (90.0, 243.4)		
			ressure Variabiliy			
	G	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	61.2 (41.8; 75.8)**	71.9 (48.3; 81.8)**	43.3 (33.9; 60.5) <sup>@#*</sup>		
	G <sub>3</sub>	47.5 (31.2; 69.1)	45.9 (36.2; 63.6)**	44.6 (25.4; 53.9)*		
	G1	51.0 (34.3; 62.4)	67.6 (59.7; 76.4)®	33.3 (19.5; 35.9) <sup>@##</sup>		
HF <sub>seP</sub> n, n.u.	G <sub>2</sub>	35.6 (21.8; 54.3)*	20.5 (13.8; 45.6) <sup>@**</sup>	42.7 (34.1; 61.0) <sup>@#*</sup>		
	G3	48.8 (27.6; 62.6)	45.6 (33.2; 61.5)*	49.7 (37.9; 51.5)**		
IE/HE mmHa <sup>2</sup> /	G_1	0.87 (0.48; 1.69)	0.44 (0.26; 0.58)®	1.80 (1.49; 4.00) <sup>@##</sup>		
LF/HF <sub>seP</sub> , mmHg²/ mmHg ²	G <sub>2</sub>	1.77 (0.77; 3.50)**	4.00 (1.06; 5.20) <sup>@**</sup>	0.88 (0.53; 1.88)#*		
	G3	0.98 (0.49; 2.50)	$1.09 (0.59; 1.90)^{**}$	0.90 (0.49; 1.42)**		
		Diastolic Blood F	Pressure Variabiliy			
	G <sub>1</sub>	7.3 (4.8; 12.9)	8.4 (4.8; 33.6)	13.3 (11.6; 13.7) <sup>@#</sup>		
TP <sub>DBP</sub> , mmHg <sup>2</sup>	G,	7.8 (5.3; 16.8)	6.3 (4.0; 9.0)	14.4 (4.0; 27.0)**		
	G <sub>3</sub>	7.3 (4.0; 10.9)	5.3 (3.2; 7.3) <sup>@*</sup>	5.9 (4.4; 7.8)**		
	G,	2.3 (1.2; 4.0)	2.1 (1.2; 7.3)	4.3 (2.3; 10.2) <sup>@#</sup>		
LF <sub>DBP</sub> , mmHg <sup>2</sup>	G	3.6 (2.3; 6.8)**	3.3 (1.2; 4.0)	2.3 (0.8; 3.2) <sup>@#*</sup>		
DBP -	G,	2.3 (1.2; 4.8)	1.1 (0.8; 3.6) <sup>@*</sup>	1.6 (0.6; 3.2)**		
	G,	4.63 (2.33; 10.07)	1.63 (1.54; 3.39) <sup>@</sup>	2.38 (1.27; 6.46)		
IC <sub>DBP</sub> , c.u.	G,	8.37 (4.14; 13.68)*	11.87 (7.25; 21.03)**	8.48 (4.38; 12.92)*		
DBb,	G,	4.74 (2.79; 11.94)	10.73 (3.30; 18.50)@**	1.42 (0.60; 2.09) <sup>@@##*</sup>		
	3		lynamics	1.12 (0.007 2.007)		
	G,	4.6 (4.0; 5.0)	4.7 (4.0; 4.8)	4.7 (3.9; 5.0)		
CO, dm³	G,	5.4 (4.9; 6.0)**	5.8 (5.3; 7.0) <sup>@**</sup>	5.5 (4.7; 6.1)*		
,	G_3	4.7 (4.0; 5.1)	5.2 (4.2; 5.7)®*	4.9 (4.1; 5.0)#		
	G,	1609 (1420; 1795)	1670 (1506; 1813)	1558 (1452; 1690)		
GPVR, dyn/s/cm⁻⁵	G,	1358 (1210; 1499)**	1207 (1190; 1385) <sup>@**</sup>	1378 (1278; 1506)#*		
GI VIC, UYII/S/CIII *	G,	1642 (1382; 1802)	1446 (1355; 1732)*	1482 (1424; 1577)®		
$CI_{dm}^{3}/m^{2}$	G	2.34 (2.13; 2.63)	2.22 (2.14; 2.44)	2.31 (2.22; 2.46)#		
CI, dm³/m²	G <sub>2</sub>	2.80 (2.56; 3.20)**	3.15 (2.67; 3.22)**	2.72 (2.64; 3.00)#**		
	G <sub>3</sub>	2.39 (2.14; 2.64)	2.52 (2.14; 2.62)*	2.43 (2.29; 2.53)		
			ry Variabiliy			
	G_1	328 (210; 538)	625 (269; 740)®	299 (202; 320) <sup>@#</sup>		
TP <sub>R</sub> , (L×min <sup>-1</sup> ) <sup>2</sup>	G_2	497 (310; 1037)*	456 (400; 610)*	655 (404; 906)#**		
	G <sub>3</sub>	303 (180; 458)	392 (320; 600)®*	263 (180; 303) <sup>@#</sup>		
			imotorics			
	G_1	1.62 (1.07; 3.08)	1.15 (0.85; 1.51)®	1.82 (1.71; 2.12) <sup>@##</sup>		
SCSleft, s	G <sub>2</sub>	1.57 (1.13; 3.36)	1.51 (1.24; 1.65)*\$	1.38 (1.26; 1.46) <sup>@#**\$</sup>		
	G <sub>3</sub>	2.04 (1.21; 3.77)*	1.95 (1.13; 2.28)*	1.73 (1.13; 1.76) <sup>@#*\$</sup>		
	G <sub>1</sub>	67.3 (28.9; 86.9)	77.1 (71.0; 80.3)®	68.0 (39.0; 88.2)#		
SMleft, %	G,	44.8 (19.0; 84.6)	37.4 (32.7; 47.2)**	31.0 (17.2; 77.7)**		
	G_3	52.4 (23.2; 85.4)	57.6 (17.7; 69.9)**	31.0 (24.5; 82.8)*		

<sup>®</sup> − p<0.05, <sup>®®</sup> − p<0.01, <sup>®®</sup> − p<0.001 − between OG<sub>1</sub> and OG<sub>2</sub> in comparison with CG; <sup>\*</sup> − p<0.05, <sup>##</sup> − p<0.01, <sup>###</sup> − p<0.001, between OG<sub>1</sub> and OG<sub>2</sub>; <sup>\*</sup> − p<0.05, <sup>\*\*</sup> − p<0.01, between G<sub>3</sub> and G<sub>2</sub> in comparison with G<sub>1</sub>

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breathing, operating only with a generalized value. On the other hand, breath variability indicators, which are calculated using spectral analysis, make it possible to distinguish multi-frequency effects, which can significantly supplement information on regulatory effects on the respiratory system (Harris et al., 2017; Bazhora & Romanchuk, 2018). We previously showed that at rest, athletes with a predominance of central (I and II types) and autonomous (III and IV types) influences on the heart rhythm are differentiated by indicators of total respiratory power TPR,  $(L \times min^{-1})^2$  and power in high-frequency range ( $HF_{P}$ , ( $L \times min^{-1}$ )<sup>2</sup>), which are significantly smaller when autonomous influences prevail (Romanchuk & Guzii, 2020). From the table 3 shows that the  $TP_{R}$  indicator,  $(L \times min^{-1})^{2}$ is quite clearly differentiated during the formation of sympathetic  $(OG_1)$  and parasympathetic  $(OG_2)$ overstrain under the influence of intense load. The latter suggests that, in combination with HRV indicators, it can be a sufficiently clear differentiation criterion, especially during combined analysis.

An important system for ensuring locomotor function during sports is the system of sensorimotor regulation, which determines the possibilities of implementing motor actions (Romanchuk, 2007). With the use of the SMM device, 25 parameters of sensorimotor function were determined during all examinations of athletes, which are presented above. In previous studies, we analyzed their changes (Guzii et al., 2020; Romanchuk & Guzii, 2020; Romanchuk et al., 2021). In the table 3 shows the indicators of sensorimotor function, which had different changes in the studied groups under the influence of physical load. Significant differences were observed among SCSleft (s), SMleft (%).

Sympathetic overstrain (OG<sub>1</sub>) is characterized by an asymmetric functional reaction to intense physical load, which is confirmed by a significant acceleration of central processes in the right hemisphere and a significant slowdown in the left (Romanchuk & Guziy, 2020). This information may indicate the importance of the right hemisphere in determining the effects of stress (Lee et al., 2022), and also suggests a decrease in the athlete's performance (Biskamp et al., 2017). According to some authors, the latter is determined by a change in the dominant role in controlling movements. An asymmetric functional sensorimotor response to physical load, but of the opposite direction, is also characteristic of parasympathetic overstrain (OG<sub>2</sub>). It is accompanied by slowing down of central processes in the premotor zone of the right hemisphere (according to SCSleft, s and SMleft, %) and acceleration in the left hemisphere (Guzii et al., 2020; Romanchuk & Guzii, 2020; Romanchuk et al., 2021).

With sympathetic and parasympathetic overstrains of the cardiovascular system of athletes under the influence of intense physical load and during the recovery period, characteristic asymmetric changes are noted at the central level of sensorimotor function regulation, which may indicate the predominant course of ergotropic and trophotrophic processes in the athletes' body. Research has highlighted the importance of testing sensorimotor function during training.

# Discussion

For a clearer idea of the differences in changes in cardiorespiratory and sensorimotory parameters in the studied groups, table 5 schematically presents significant differences in the directions of dynamic changes compared to the initial state (G<sub>1</sub>). The demonstrativeness of this table lies in the fact that it makes it possible to compare the differences in the response of the cardiorespiratory and sensorimotory systems to an intense training load and recovery after it, taking into account significant changes in individual indicators, but without taking into account differences in the initial state. In the table 5 shows the direction of changes in indicators for each group: (D) – increase ( $\uparrow$ ), decrease ( $\downarrow$ ), or absence of significant changes (=), which are confirmed by Wilcoxon tests with different probabilities (+, ++, +++). At the same time, the differences between the indicators after the training load in comparison with the initial indicators (G<sub>2</sub>-G<sub>1</sub>), as well as the differences between the indicators obtained the next morning after training, in comparison with the initial indicators  $(G_3-G_1)$ , are analyzed. That is, the table presents the directions of changes in the studied indicators that characterize the impact of physical load, and changes that reflect the return of the presented physiological parameters to the initial level (recovery).

A complex polyfunctional study showed that overstrains have sufficiently characteristic determinants associated not only with the autonomous regulation of the heart rhythm, but also with the autonomous support of the contractile function of the heart, vascular tone, and central hemodynamics. Characteristic changes also occur in the external breathing and sensorimotor system.

After analyzing the data of studies, the main indicators of the activity of the cardiorespiratory, autonomic and sensorimotor systems were determined, which signal the development of overstrain of the sympathetic and parasympathetic types (Table 5). The obtained results prove that in the entire group of athletes, the reaction to physical exertion is characterized by a decrease in the activity of parasympathetic influences immediately after it. The next morning, with a moderate increase in LFHF (ms<sup>2</sup>/ms<sup>2</sup>), there is an increase in RMSSD (ms), which indicates the activation of parasympathetic mechanisms during the recovery period against the background of a moderate predominance of sympathetic innervation. With sympathetic overstrain, there is a significant predominance of sympathetic influences immediately after training - a pronounced increase in ABI (c.u.) and ARI (c.u.), which remains at the same level the next morning.

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When the parasympathetic is overstrained immediately after the load, activation of the parasympathetic heart rate regulation mechanisms is noted according to the LFHF (ms<sup>2</sup>/ms<sup>2</sup>), RMSSD (ms) indicators. The dynamics of other indicators shows a slight reaction of the sympathetic nervous system. The next morning after training, activation of parasympathetic regulation increases. From the side of the central mechanisms of regulatory effects on the heart rhythm, in contrast to CG, in which the activity of subcortical regulation (SRAI, c.u.) the next morning after training returns to initial values, both with sympathetic and parasympathetic overstrains, their activity remains greater than in the initial state. Another indicator that characterizes suprasegmental effects is the VLF (ms<sup>2</sup>) indicator. In CG, the next morning after training, it returns to initial values, however, in OG, it remains significantly reduced, and in OG<sub>2</sub> it changes its direction and becomes significantly greater than in the initial state.

An important indicator of the functional state of the body is the parameters of systemic hemodynamics and its regulation. A characteristic feature of recovery after exposure to stress is the return of CI  $(dm^3/m^2)$  and GPVR  $(dyn/s/cm^{-5})$  indicators to their original values the next morning, which is noted in CG and OG<sub>2</sub>. At the same time, in OG<sub>1</sub>, this dynamics is slowed down and is different from the initial state, but less significant than the day before. With regard to the regulation of hemodynamic function, changes in SBPV and DBPV are indicative. If the changes in SBPV are considered from the standpoint of regulatory support of the contractile function of the heart, then the constancy of an excessive increase in sympathetic regulatory influences in  $OG_1$  and constancy of an excessive decrease in sympathetic influences in  $OG_2$  can be constant. Taking into account DBPV, which characterizes regulatory effects on vascular tone to a greater extent, it is possible to state the absence of a reaction immediately after exercise in  $OG_1$ , which moderately decreases the next morning, in contrast to parasympathetic overstrain, when in response to exercise, general regulatory effects increase, and the next morning significantly decrease. This type of response of vascular tone is probably related to dysregulatory effects in  $OG_2$  athletes. These data are confirmed by similar changes in the index of centralization (IC, mmHg<sup>2</sup>/mmHg<sup>2</sup>) of regulatory influences on DBP.

The differences in the regulation of spontaneous breathing turned out to be quite significant. If in  $OG_1$  there is a significant decrease the total power of breathing  $(TP_R, (L \times min^{-1})^2)$  of immediately after exercise in comparison with CG, which remains the same the next morning, then in  $OG_2$  the changes are similar to those in CG, but immediately after exercise their significance is significantly greater.

Changes in sensorimotor function were indicative. The central mechanisms of its regulation are related to the speed of SCS (s) in the prefrontal areas of the cerebral cortex. At the same time, the left and right lobes of the brain are characterized by different mechanisms of motor activity, primarily with a predominance of energy and plastic support for work (Skyba et al., 2017; Maric et al., 2020; Watanabe et al., 2021). It is believed that the depletion of energy resources in the body leads to an increase in the activity of the prefrontal cortex of the left lobe and, according to many authors, is as-

<b>Table 5.</b> Differences in the dynamics of the cardiorespiratory and sensorimotor systems in athletes with	
sympathetic and parasympathetic overstrain	

		С	G			0	G,			0	G <sub>2</sub>	
Indicator	$G_{2} - G_{1}$		$G_{3} - G_{1}$		<b>G</b> <sub>2</sub> <b>- G</b> <sub>1</sub>		<b>G</b> <sub>3</sub> – <b>G</b> <sub>1</sub>		<b>G</b> <sub>2</sub> <b>- G</b> <sub>1</sub>		<b>G</b> <sub>3</sub> <b>- G</b> <sub>1</sub>	
	D	р	D	р	D	р	D	р	D	р	D	р
ABI, c.u.	1	++	=		1	++	<b>↑</b>	++	=		↓	+.
SRAI, c.u.	1	++	=		1	++	1	++	<b>↑</b>	++	<b>↑</b>	+
ARI, c.u.	1	++	=		1	++	1	++	1	+	Ļ	+
RMSSD, ms	Ļ	++	1	+	Ļ	++	$\downarrow$	++	1	+	=	
VLF, ms <sup>2</sup>	$\downarrow$	++	=		Ļ	++	$\downarrow$	++	Ļ	++	↑	++
LFHF, ms <sup>2</sup> /ms <sup>2</sup>	1	++	↑	+	↑	++	1	+	Ļ	+	Ļ	+
LF <sub>se</sub> n, n.u.	=		=		1	++	1	++	Ļ	+	Ļ	+
HF <sub>se</sub> n, n.u.	Ļ	+	=		Ļ	++	↓	+	1	+	1	++
LFHF <sub>SBP</sub>	1	++	=		↑	++	1	++	Ļ	+	Ļ	++
TP <sub>DBP</sub> , mmHg <sup>2</sup>	=		=		=		↓	+	1	+	Ļ	++
LF <sub>DBP</sub> , mmHg <sup>2</sup>	1	++	=		=		$\downarrow$	+	$\downarrow$	+	Ļ	++
IC <sub>DBP</sub> , mmHg <sup>2</sup> /mmHg <sup>2</sup>	1	+	=		1	++	1	++	1	+	Ļ	+
CO, dm <sup>3</sup>	1	++	=		1	++	1	+	1	+	=	
GPVR, dyn/s/cm <sup>-5</sup>	$\downarrow$	++	=		Ļ	++	$\downarrow$	+	Ļ	+	=	
CI, dm <sup>3</sup> /m <sup>2</sup>	1	++	=		↑	++	1	+	↑	++	=	
TP <sub>R</sub> , (L×min <sup>-1</sup> ) <sup>2</sup>	1	+	=		Ļ	+	$\downarrow$	+	↑	++	=	
SCSleft, s	=		↑	+	↑	+	↑	+	↓	++	Ļ	+
SMleft,%	=			=	Ļ	++	Ļ	++	Ļ	+	↓	+

+ - p<0.05, ++ - p<0.01, +++ - p<0.001

sociated with excessive activity of the sympathetic branch of the central nervous system, which we observed in the group of athletes with sympathetic overstrain  $(OG_1)$ , when such activity was maintained the next morning after training. At the same time, a decrease in the speed of SCS (s) in the left lobe of the prefrontal cortex of the brain may indicate excessive activity of anabolic processes, which are associated with the modification of plastic mechanisms (Zohdi et al., 2020). This variant is observed in  $OG_2$ . It is quite typical for both sympathetic and parasympathetic overstrain to reduce SMleft (%), which, as a rule, characterizes the deterioration of the processes of adaptation of the body to the load.

# Conclusion

The obtained results on the formation of sympathetic and parasympathetic overstrain under the influence of intense physical activity indicate that changes in the autonomic regulation of the heart rate, which determine the type of heart rate regulation, are accompanied by a number of changes in hemodynamics, autonomic regulation of the pumping function of the heart, vascular tone, re-

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spiratory regulation, and sensorimotor function. The determined differential dynamics of changes in cardiorespiratory and sensorimotor indicators will allow further algorithmization of the assessment of the functional state of the athletes' body in order to detect early states of non-functional overstrain and overtraining.

#### Author's contribution

Conceptualization, OG, OR; methodology, OG, OR; software, OG, OR; check, OG, OR; formal analysis, OR; investigation, OG; resources, OG, OR; data curation, OR; writing – rough preparation, OG; writing – review and editing, AM, OR; visualization, OR; supervision, AM; project administration, OG, AM; receiving funding, OG. All authors have read and agreed with the published version of the manuscript.

#### **Conflicts of Interest**

The authors declare no conflict of interest.

#### Funding Statement

This research received no external funding

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