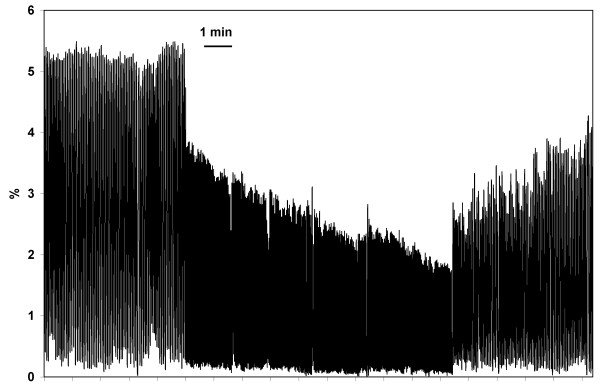

Fig. 2. Fragment of the original record of breath capnogram at rest, during the test with hyperventilation (breathing rate being 30 cycles per minute) and the part of recovery period: on the abscissa axis – concentration of carbon (IV) oxide in expiration air; on the ordinate axis – time of recording

In the background, the men of group I were characterized by shorter duration of R-R intervals and lower SI values compared to group II and III (Table 1). At the same time, the duration of R-R intervals was shorter in normotonic subjects than in vagotonic ones.

During the test on hyperventilation, a decrease in t-R-R was registered being the most expressed in group II; its reactivity at the end of the test was 134 ± 19.7 ms ($P < 0.05$); a decrease in SI was observed in the edge groups with the highest reactivity in vagotonic subjects (–1.94 ± 1.74 mL/m^2, $P < 0.05$); a decrease in GPR was found to a greater extent in groups II and III. An increase in HI was found in all groups during the test with the least reactivity in parasympathicotonic subjects being 0.18 ± 0.09 L/m^2•min ($P < 0.05$) on 5-minute test.

In the recovery period, an increase in t-R-R and SI was found in all typological groups. After the test, the least SI reactivity was found in sympathicotonic subjects (–1.88 ± 1.34 mL/m^2, $P < 0.05$), while the least GPR reactivity was observed in the normotonic group (–29.0 ± 91.7 dyn/s•cm^5, $P < 0.05$).

Certain differences were found in the indicators of cardiodynamics between the persons with different initial level of autonomic tone (Table 2).

Table 1

Central haemodynamics indicators during and after the test of the regulated breathing at a rate of 30 cycles per minute in healthy young men with different initial levels of autonomic tone (x ± SE)

Indicators	Groups	Background	Test (5 min)	Test (10 min)	Recovery (5 min)	Recovery (40 min)
t-R-R, ms	I	798.4 ± 23.2	691.1 ± 20.1	722.8 ± 21.1	865.6 ± 23.8	857.5 ± 22.1
	II	884.4 ± 23.2*	750.1 ± 23.5*	780.1 ± 23.8*	914.0 ± 26.6*	912.1 ± 31.0*
	III	959.3 ± 30.3*#	744.0 ± 26.5*	795.8 ± 31.0*	993.0 ± 26.9*	998.5 ± 29.5*#
SI, mL/m^2	I	29.14 ± 1.73	29.50 ± 1.82	28.80 ± 1.66	29.12 ± 1.72	29.71 ± 1.59
	II	35.63 ± 2.25*	35.65 ± 2.07*	36.73 ± 1.99*	38.19 ± 3.24*	36.83 ± 2.43*
	III	40.02 ± 3.04*	35.54 ± 1.95*	37.89 ± 2.12*	40.72 ± 2.74*	41.96 ± 3.07*
HI, L/m^2·min	I	2.19 ± 0.11	2.41 ± 0.12	2.38 ± 0.11	1.98 ± 0.08	2.01 ± 0.08
	II	2.36 ± 0.12	2.76 ± 0.14*	2.75 ± 0.12*	2.38 ± 0.15*	2.26 ± 0.11*
	III	2.43 ± 0.16	2.86 ± 0.17*	2.84 ± 0.15*	2.40 ± 0.15*	2.45 ± 0.16*
GPR, dyn/s·cm^5	I	1848.0 ± 88.3	1681.0 ± 78.8	1708.6 ± 78.1	2038.9 ± 98.9	1970.6 ± 85.6
	II	1838.4 ± 144.9	1521.4 ± 77.2*	1509.9 ± 83.5*	1844.3 ± 122.8	1845.7 ± 136.9
	III	1790.8 ± 134.7	1473.0 ± 92.9*	1457.5 ± 74.8*	1765.0 ± 98.7*	1818.0 ± 147.7

Note: * – $P < 0.05$ compared to group I; # – $P < 0.05$ when comparing group II and III; by ANOVA method with Bonferroni correction.

Table 2

Cardiodynamics indicators during and after the test of regulated breathing at a rate of 30 cycles per minute in healthy young men with different initial levels of autonomic tone (x ± SE)

Indicators	Groups	Background	Test (5 min)	Test (10 min)	Recovery (5 min)	Recovery (40 min)
TPh, ms	I	138.7 ± 4.5	137.6 ± 4.2	139.4 ± 4.2	136.1 ± 5.3	144.5 ± 4.8
	II	129.7 ± 2.9*	129.2 ± 2.8*	130.0 ± 2.7*	136.0 ± 3.4*	134.6 ± 3.4*
	III	128.0 ± 3.3*	127.2 ± 3.3*	130.0 ± 3.6*	135.0 ± 3.6*	134.4 ± 3.4*
EPh, ms	I	233.6 ± 4.3	234.5 ± 4.3	237.4 ± 4.0	248.1 ± 4.7	246.5 ± 4.1
	II	239.8 ± 7.0	241.8 ± 6.0	249.4 ± 4.8*	269.7 ± 13.2*	259.7 ± 12.0
	III	264.9 ± 15.6*#	240.3 ± 5.3	262.5 ± 9.7*	288.9 ± 16.0*	294.4 ± 19.3*#
TI, %	I	37.1 ± 1.0	36.9 ± 0.8	36.9 ± 0.8	36.9 ± 1.0	36.8 ± 0.9
	II	35.3 ± 0.8	34.9 ± 0.6*	34.3 ± 0.6*	34.1 ± 1.0*	34.7 ± 0.9*
	III	33.4 ± 1.0*#	34.6 ± 0.7*	33.3 ± 0.7*	32.5 ± 1.0*	32.4 ± 1.2*#
ES, mL/s	I	219.0 ± 9.9	219.5 ± 9.4	212.5 ± 9.3	205.6 ± 8.8	212.2 ± 8.9
	II	254.9 ± 11.7*	254.2 ± 11.6*	254.1 ± 11.4*	240.9 ± 11.2*	245.4 ± 10.7*
	III	257.2 ± 11.7*	253.7 ± 13.8*	248.5 ± 13.2*	243.1 ± 12.7*	245.4 ± 12.0*

Note: * – $P < 0.05$ compared to group I; # – $P < 0.05$ when comparing group II and III; by ANOVA method with Bonferroni correction.

Thus, in the background, high TI and, accordingly, TPh, as well as the shortest EPh and ES were observed in sympathicotonic subjects. A decrease in TPh, TI was found during hyperventilation especially in the men of group III compared to group I. The highest TI reactivity was determined in sympathicotonic subjects (–1.09 ± 0.94%, $P < 0.05$). In the recovery period, a decrease in TI was found in group II and especially in group III compared to group I. After the functional test, a trend to an increase haemodynamic indicators among the subjects was observed, that is a normal reaction of cardiovascular system in response to load and which is conditioned by the influence of the sympathetic-adrenal link of the autonomic nervous system.

Discussion

The regularities identified in our studies are largely confirmed and explained by the analysis of other researches on the reactivity of different systems of human body depending on the initial level of autonomic tone. Thus, the study of Skyba et al (2017) evaluated the functional state of the autonomic nervous system according to the indicators of heart rate variability with the determination of the initial autonomic tone and autonomic reactivity in athletes with different levels of sensorimotor reactivity. The autonomic tone, identified among most athletes with a medium level of sensorimotor response, was characterized by a background eutonia, and sympathicotonia was significantly more prevalent among the group of athletes with a high level of sensorimotor response compared to the athletes with its medium and low level. Autonomic imbalance, manifested by hypersympathicotonic reactivity, was determined among the athletes with high and medium levels of sensorimotor response, which indicated the tension of the cardiovascular system functioning and reduction in the adaptive capacity of the body. The importance of considering the autonomic tone in the pathologies of the cardiovascular system in sleep disorders is

discussed (Tamisier et al., 2018). The enhanced autonomic tone along with various stimuli, such as intermittent hypoxia, broken continuity of sleep, reduction of its duration, the increased respiratory effort and short-term hypercapnia can initiate a cascade of pathological changes leading to the deterioration of a person's condition.

The haemodynamics measurements of pregnant women by Doppler fluometry shows that the performance of the fetus heart is significantly influenced by the mother's autonomic tone level (Lakhno, 2017). Fetal circulation disorders are formed in response to the increased sympathetic and decreased vagal tone.

The confirmation of typological features of cardiovascular system response to any stimuli is genetic research. Sigurdsson et al. (2018) demonstrate that there is a genetic determination for most parameters of autonomic equilibrium. Genetic conditioning for blood pressure and cardiac activity has been clarified. However, the reactivity of haemodynamics on loading, changes in autonomic tone depends on the activity of many genes and can be analyzed by polygenic methods.

According to a meta-analysis of the literature, the origin of heart rate waves in the high frequency range 0.15–0.40 Hz (HF) is almost uniformly interpreted. These waves are the reflection of respiratory sinus arrhythmia (Novak et al., 1994; Linden & Diehl, 1996). An important component of high-frequency component of R-R interval oscillations is shown to be efferent vagal activity with electrical stimulation of the vagus, vagotomy, blockade of muscarinic receptors (Akselrod et al., 1981; Pomeranz et al., 1985; Malliani et al., 1991). At the same time, there is an opinion about the complex nature of high-frequency oscillations (Malliani, 1996). In measurements (Appel et al., 1989) hypothesized that the high-frequency component was represented in the parasympathetic and sympathetic efferent impulse, but the low transmission of sympathetic effects almost completely eliminates the high-frequency sympathetic rhythm (Introna et al., 1995). Thus, in patients with spinal anesthesia, the spread of the spinal block reached high thoracic segments (higher than T_3) and a decrease in variability was observed not only in the low but also high frequencies.

At the same time, in experiments on cats (Shejh-Zade et al., 2001), after complete denervation of the heart, the respiratory peak is always determined on the heart rate spectrogram, which indicates the ability of the sinoatrial node to change its automatism under the influence of the respiratory fluctuations of venous return. The oscillation of systolic blood pressure or blood stroke volume in this range is found to depend on the depth of breath movements (Sanderson et al., 1996; Bouteau & Tavernier, 2004). The amplitude of these waves depends on the level of fluid in the body (Wiesenack et al., 2005).

The genesis of low-frequency waves of heart rhythm remains controversial as some authors believe that their normalized power reflects the activity of the sympathetic division of autonomic nervous system (Kamath & Fallen, 1993; Guzzetti et al., 1994; Malliani et al., 1994; Montano et al., 1994; Lucini et al., 1997; Sesay et al., 2008), others tend to think of both sympathetic and parasympathetic influences in the formation of these oscillations (Appel et al., 1989).

There are several hypotheses for the mechanisms of low-frequency waves of blood pressure and heart rate (Malliani, 1998; Hajutin & Lukoshkova, 1999). The basic assumption is that such oscillations are the result of periodic amplification and attenuation of signal flow of arterial baroreceptors at the stroke of blood pressure waves of the third order (Hajutin & Lukoshkova, 1999). Thus, when blood pressure lowers, baroreceptor signals accelerate the heart rate; and they slow it down with high blood pressure. The period duration of such oscillations (8–12 s) is determined by the delay sum in the process time in the efferent branch of reflex baroreceptor arc (Bernardi et al., 1997). However, the LF component increases under the conditions of emotional stress or physical exertion when pressure increases; and barro-reflex impulse increases (Malliani et al., 1991). The power of slow waves increases under the conditions of experimental regional myocardial ischemia in dogs without any changes in blood pressure (Rimoldi et al., 1990). Such changes may be the consequences of an increase in norepinephrine content in blood. This is confirmed by experiments performed during surgery in patients with pheochromocytoma. The level of norepinephrine concentration in blood plasma correlates with the power of the low-frequency component of the R-R interval spectrum ($r = 0.68$) (Sesay et al., 2008).

It has been noted (Jansen et al., 1995) that long waves can also occur due to the rhythmic nature of the myogenic reactions of arterioles. It has been established (Myers et al., 2001) that sympathetic influences modulate the Mayer waves due to changes in the resistance of peripheral vessels.

According to some studies (Cevese et al., 1995), slow heart rate waves are of centrogenic origin. It is believed that there is a rhythmic activity with a period of about 10 s of parasympathetic and sympathetic cardiomotor neurons of the brain stem. Some measurements (Inoua et al., 1990) show the absence of LF component in the patients with quadriplegia. This was explained by the destruction of the neural pathways that transmit rhythms from the brain to the spinal cord. At the same time, it was shown (Guzzetti et al., 1994; Koh et al., 1994) that there were slow waves of R-R interval and blood pressure in some patients with such disorders. These phenomena were interpreted as manifestations of spinal rhythmics affecting vascular background and sinus pacemaker activity (Guzzetti et al., 1994). It was found (Cooley et al., 1998) that there were no waves in the spectra of blood pressure, R-R interval, and respiratory rate before surgery on two patients with severe heart failure with the implanted artificial left ventricle. The repeated research showed that slow oscillations appeared and became "expressive and dominant" in the spectrum of R-R interval of the emptied heart; and they were absent in the spectrum of blood pressure.

According to Maliani et al. (1998), the origin of low-frequency and high-frequency waves of heart rhythm is conditioned by their complex central-peripheral organization and has a complex multicomponent nature. Therefore, the question of applying the methods of heart rate variability analysis to evaluate the autonomic balance of cardiac regulation accurately is debatable. Principles of the possibility of accurate evaluation of "sympathetic-parasympathetic balance" by means of the R-R interval spectrum indicators are supported by Malliani (1996, 1999), Pagani & Malliani, 2000). The researcher considers that HF waves are determined by only parasympathetic influences, and LF – by sympathetic ones; the tone changes of two parts of autonomic nervous system occur reciprocally. However, a different view can be drawn from the analytical review of the literature (Eckberg, 1997; Kovalenko, 2005). In experiments (Koh et al., 1994; Martinmaki et al., 2006), respiratory and long-wave heart rhythms were eliminated with the blockade of M-cholinoreceptors in dogs and humans. No correlation was found between the spectral power at 0.1 Hz frequency and the release of norepinephrine during muscle stimulation (Kingwell et al., 1994).

The measurements (Shejh-Zade et al., 2001) show that the sympathetic nervous system can act on the heart rhythm through parasympathetic terminals, changing the frequency of their burst pulses. At the same time, the results of acute studies in cats show that heart rate variability "reflects a very specific interaction of myogenic, sympathetic and parasympathetic mechanisms aimed at economizing cardiac activity, but does not reflect the balance of tonic effects of extracardial nerves".

At the same time, according to spectral analysis of cardiac rhythm at gravitational loads, there is a redistribution of vegetative balance towards the predominance of its sympathetic link being, to some extent, a confirmation of the theory of A. Malliani (Hirayanagi et al., 1999). Changes in adrenaline/norepinephrine ratio in the urine of 13 swimmers during seven weeks of training correlated positively with LF/HF index shifts ($r = 0.42$, $P < 0.03$) (Atlaoui et al., 2007). An increase in the normalized power of heart rate waves at low-frequency range is observed with stress influences (Dishman et al., 2000).

According to (Ruttkaj-Nedecki, 2001), criticism of this approach should not negate the possible benefit of calculating the LF/HF ratio to characterize the state of regulation of the cardiovascular system. However, one must be more critical of the physiological interpretation of its changes.

It has been found (Wagner & Persson, 1994) that the sympathovagal balance can be estimated from the wave amplitude of stroke blood volume or blood pressure in different frequency ranges. It is possible to bypass the controversial issues that arise in the analysis of R-R interval oscillations. Thus, studies of rats of the Wistar-Kyoto line and spontaneously hypertensive rats have shown that the power spectrum of blood pressure in the low frequency range is due to sympathetic influences through α_1-adrenoreceptors (Dabire et al., 2002). Measurements of systolic pressure variability in persons with spinal cord injuries show that at breaks above T_3 segment, the power of Mayer waves decreases and their normal re-

sponse to orthostatic test changes (Munokata et al., 2001). Studies on runners (Portier et al., 2001) show after 3 weeks of rest and 12 weeks of endurance training that sympathovagal balance can be estimated by the ratio and changes in the power of blood pressure oscillations in the range of high and low frequencies. However, wave processes in blood pressure can be affected by not only cardiac output fluctuations but also modulation of peripheral vascular tone with sympathetic nerves, NO system activity (Chowdhary et al., 2002).

It has been found (Liu et al., 2004; Bar et al., 2009) that variability in blood pressure and stroke volume shows different information about the activity of autonomic nervous system than the analysis of changes in R-R interval duration. One of the factors may be the absence of plausible correlations between the capacities in the same oscillation ranges of different parameters and their changes during blockade of M-choline and β-adrenoceptors, analysis of the relationship of their parameters with the pupil diameter and its response to light. When stimulating the carotid sinus by the creation of negative pressure at rest, an increase in the power of fast waves of blood pressure, slow waves of blood pressure and heart rate was observed. And at physical activity, such influence increased only the power of BP oscillations in the range of low frequencies.

The wave structure of the heart rhythm can be studied using autocorrelation analysis (Baevskyj et al., 2001). However, autocorrelation analysis can show nothing with the changes in the frequency of periodical process although there is latent rhythmic.

The structural linguistic method is suggested to analyze the changes in the spectrum form of R-R interval duration and the regularities of their transformation with different influences (Il'in et al., 2003); it helps to simplify the description of the changes while retaining the most important properties of information. Thus, it can be argued that the output tone of the autonomic nervous system can modulate changes in human haemodynamics in breathing hypocapnia and affect the features of long-term changes after the cessation of this influence.

Conclusion

The initial level of autonomic tone affects the dynamics of CO_2 level in the alveolar air during hyperventilation and recovery period after it. A decrease in the duration of R-R interval was found during the hyperventilation test, being the most expressed in normotonic subjects. Heart index increased in all three groups and general peripheral resistance decreased mostly in the groups of normo- and parasympathicotonic subjects. At the same time, the reliable increase of stroke index and heart index was found. A decrease in the indicators of tension index and ejection speed and an increase in the duration of tension phase and ejection phase were observed in the recovery period after hyperventilation in normotonic subjects and especially parasympathicotonic compared to sympathicotonic ones.

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