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Mechanisms of Periodic Heart Rate Oscillations: A Study Using Controlled Breathing Tests

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Abstract—An orthostatic test with frequency-controlled breathing (with periods of 4, 6, 8, 10, and 12 s) was used to analyze frequency estimates of the heart rate variability (HRV) spectrum in the low frequency (LF) and high frequency (HF) ranges in 36 volunteers (26 men and 10 women) aged 19–21 years without signs of heart or respiratory pathology. The subjects took a breath at the moment of an auditory signal. There were no other requirements for the respiration rhythm. Variables were compared using Wilcoxon's test for pairwise comparisons; correlations were estimated by Spearman's rank correlation *R* test. The sensitivities of the LF and HF ranges of the HRV spectrum to periodic respiratory perturbations at different frequencies were demonstrated to differ from each other. Autonomous 0.10- and 0.25-Hz circuits of oscillatory processes were found in HRV. The transition zone of influence of these circuits was located in the region around 0.125 Hz. The characteristics of the 0.10- and 0.25-Hz oscillations in HRV were studied. It was demonstrated that the 0.10-Hz oscillatory process is a potent mechanism of heart rate control, is affected by external factors, and determines the dynamics of the autonomic nervous state of the body, while the 0.25-Hz process is a regulatory mechanism of medium strength, is resistant to external factors, and characterizes the adaptation reserve of the autonomic nervous control of the heart rate, as well as the autonomic nervous state of the body. Resonance responses in the HRV spectrum can be used for studying the characteristics of the 0.10- and 0.25-Hz oscillations.

Periodic oscillations of the heart rate (HR) are determined by a complex regulatory system. Like other biorhythms, the rhythmic oscillations of the HR result from stochastic nonlinear biological mechanisms responding to changing external factors. However, most physiological oscillations are not strictly periodic; changing external factors and random disturbances ("noise") usually make the rhythms vary irregularly with time [1]. The cardiac rhythm is no exception. The mechanism of HR control mediated by the autonomic nervous system is modulated by numerous external factors (respiration, exercise, posture changes, emotional factors, etc.) [1]. This multifactorial interaction determines the complexity of studying the effects of various factors on HR variability (HRV), as well as the characteristics of the physiological oscillations in HR themselves.

To simplify the understanding of the complex inter- and intrasystemic interactions forming the HRV, we may present this set of mechanisms as an integrated system of the autonomic nervous control of the heart (SANCH), in which respiration rate, changes in blood pressure (BP), emotions, etc., are input parameters and HRV is the output signal (Fig. 1). Probably, various biological oscillatory mechanisms exist within this system; however, they differ in their relative contributions to the formation of the output signal. The question arises of how the roles of different internal oscillatory mechanisms in the formation of the HRV can be studied.

Interactions between the cardiovascular and respiratory systems are very strong; one of their results is respiratory sinus arrhythmia [2, 3]. Although the intensity of this intersystemic interaction has not been determined conclusively, the use of the functional parameters of the respiratory system for studying the characteristics of the SANCH seems promising, the more so as these parameters are easy to measure. The monitoring of the function of the respiratory system makes it possible to record most information at the input of the SANCH. Analysis of the input signals and the output signal (in the form of HRV) under different conditions of SANCH functioning allows the characteristics of the interaction between the respiratory and the cardiovascular systems and their changes with time to be described and studied in detail.

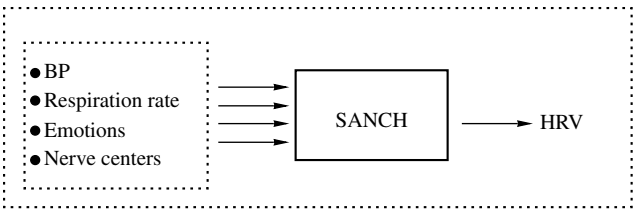

Fig. 1. Model of the SANCH. For abbreviations, see the text.

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Let us assume that all rhythmic processes in the SANCH occur at different oscillation frequencies and have different intensities and the complex output signal of the system results from the summation of all internal processes. We may expect in this case that, if any two processes within the SANCH have the same frequency, the influence of this frequency on the formation of the output signal will be increased by the resonance mechanism.

Thus, controlled respiratory perturbations with different frequencies of oscillations at the input of the SANCH can be used for detecting other periodic processes within the system [4] and studying their properties on the basis of the estimation of resonance responses at different frequencies in the HRV as the output signal of the SANCH. The use of the resonance effect can decrease the effects of other, unrecorded, input factors on the SANCH output signal.

This suggestion is confirmed by the frequency-dependent strength of the effect of respiratory system functioning on the main functional parameters of the cardiovascular system, such as HRV and BP [5].

Spectral analysis is one of the most convenient methods for estimating the harmonic components of HRV.

We studied the mechanisms of the periodic oscillations in HR frequency with the use of frequency-controlled breathing.

METHODS

Frequency estimates of the components of the HRV spectrum were studied in 36 volunteers (26 men and 10 women aged 19–21 years) without signs of cardiovascular or respiratory pathology.

Rhythmograms were recorded during an orthostatic test with controlled breathing (with periods of 4–12 s). We recorded RR series of the electrocardiogram (ECG) in two variants (with subjects being in a horizontal or a vertical position); in each variant, five RR series were recorded with different periods of controlled breathing (4, 6, 8, 10, and 12 s). The respiration rhythm was set using 0.5-s auditory signals. At the moment of a signal, the subject took a breath. There were no other requirements for the respiration rhythm. The subjects themselves chose the relative durations of the inhalation and exhalation phases and the depth of breathing most comfortable for them. The only additional recommendation that the volunteers received before the test was that the depth and the temporal structure of the respiration cycle be similar to those characteristic of spontaneous rather than forced respiration. Each stage of controlled breathing lasted 3 min, after which BP was measured according to Korotkoff.

The frequency estimates of HRV were obtained by the parametric method, according to which a spectrum of the RR time series of the ECG was constructed on the basis of a self-regression 14th-order model. This method is an alternative to the classical Fourier transform of time series [6, 7]. The software for spectral analysis was developed at the Saratov Research Institute of Cardiology (Certificate of Official Registration of a Computer Program no. 980656 of November 12, 1998). The program allows the spectral power density to be calculated with a step of at least 0.01 Hz in the range from 0.01 to 0.50 Hz and a quantization period for RR arrays of 0.5 s. In the subsequent analysis, three spectral ranges were distinguished: the high frequency (HF) range (0.15–0.40 Hz), the low frequency (LF) range (0.04–0.15 Hz), and the very low frequency (VLF) range (<0.04 Hz) [8]. The frequency powers of the HRV spectrum (in ms^2) were calculated in these ranges.

For spectral analysis, we chose rhythmograms without noise, extrasystoles, marked linear trends, or transitional processes.

In addition to the absolute powers of the components of the HRV spectrum, we calculated the power increments of these components as follows: $\Delta var2\text{–}1 = (var2 - var1)/var1$. The changes in mean values for RR intervals were calculated similarly. We used the following designations for presenting the results: *LF*, the LF component of the HRV spectrum; *HF*, the HF component of the HRV spectrum; *RR*, the mean value of the RR intervals of the ECG; the numbers 4, 6, 8, 10, and 12, the stages of the functional test with controlled respiration with periods of 4, 6, 8, 10, and 12 s, respectively; *1, the first set of rhythmograms (with the subjects in a horizontal position); *2, the second set of rhythmograms (with the subjects in a vertical position); *LF*4*2–1, the power increment of the LF component upon the transition to orthostasis in the subjects performing controlled breathing with a period of 4 s; *LF*6–4*1, the power increment of the LF component in the subjects performing controlled breathing with a period of 6 s relative to that with a period of 4 s; etc.

The Statistica 6.0 software package was used for statistical calculations. All data were tested for the fit to a normal distribution with the use of the Shapiro–Wilk *W* test [9] in order to choose between parametric and nonparametric methods of their subsequent analysis. We found that some data were not distributed normally; therefore, the subsequent calculations were performed using nonparametric methods. We used Wilcoxon's *Z* test for pairwise comparisons when estimating the significance of differences between the variables [10, 11]. Correlations were estimated by Spearman's rank correlation coefficients (*R*).

The data are presented in the form of quartiles (*Me* (25%, 75%)) for samples. First-type errors (α) no higher than 5% were taken to be acceptable.

RESULTS AND DISCUSSION

We found that the frequency ranges of the HRV spectrum in the absence of external respiratory pertur-

Table 1. Powers of the HRV components in tests with controlled breathing, *Me* (25%, 75%)

Parameter	Controlled breathing period				
	4 s	6 s	8 s	10 s	12 s
In a lying position					
LF, ms^2	582.5 (351; 1152)	682 (352; 1296)	2449 (1147; 3759)	2987 (1352; 4597)	1996 (1029; 3594)
HF, ms^2	1482 (699; 3745)	2054.5 (967; 3820)	1372 (844; 2126)	1547 (553; 2504)	1972 (607; 2601)
RR	0.882 (0.752; 0.927)	0.868 (0.769; 0.967)	0.891 (0.817; 0.971)	0.877 (0.814; 0.961)	0.901 (0.82; 0.937)
In a standing position					
LF, ms^2	631.5 (340; 868)	890 (370; 1023)	1534 (703; 2435)	1303 (737; 2112)	942 (585; 1883)
HF, ms^2	326.5 (177; 579)	585 (302; 1006)	452 (212; 691)	278 (220; 595)	318 (211; 753)
RR	0.685 (0.608; 0.753)	0.695 (0.618; 0.737)	0.693 (0.607; 0.737)	0.691 (0.617; 0.724)	0.607 (0.59; 0.705)

Notes: Here and in Table 3, LF and HF are the low frequency and high frequency components of the HRV spectrum, respectively; *RR* is the mean RR interval of the ECG.

bations in the corresponding spectral regions are characterized by relatively constant absolute spectral power densities (Table 1). External respiratory perturbations at the respiration frequencies caused a considerable increase in the HRV spectral power density (Table 1) compared to that at the given spectral frequency in the absence of respiratory perturbations in the given region. The power increased by 4–6 times in the LF range (with respiration periods of 8–12 s) (*LF*8*1, *LF*10*1, and *LF*12*1 relative to *LF*4*1 and *LF*6*1) and by 1–2 times in the HF range (*HF*4*1 and *HF*6*1 relative to *HF*8*1, *HF*10*1, and *HF*12*1) in the subjects that were in a horizontal position and by 2–3 and 1–1.5 times, respectively, in the subjects that were in a vertical position.

Thus, the LF and HF ranges of the HRV spectrum exhibited different sensitivities to periodic respiratory perturbations. The absolute spectral power densities at respiration frequencies were higher in the LF than in the HF range, the differences being larger in a vertical position of the subjects than in a horizontal position ($Z = 1.92$, $P = 0.05$, and $Z = 3.66$, $P = 0.0003$, respectively). The absolute spectral power densities were the highest in the tests with a respiration period of 10 s (the 0.10-Hz component of the HRV spectrum) (Table 1), which agrees with the assumption that a stable component of the HRV spectrum exists in the 0.10-Hz region (the LF range) [12–16].

The mean HR remained about the same as the rhythmograms of each set of tests were recorded; therefore, the changes in the powers of the components of the HRV spectrum resulted from the effect of controlled breathing on the SANCH rather than from a change in the general functional tone of the system.

The transition to the orthostatic position was accompanied by a decrease in the power of the spectrum response at respiration frequencies by 1.5–2 and 4–5 times in the LF and HF ranges, respectively ($P < 0.01$). Only the spectral power density of the HF range of the HRV spectrum decreased significantly (by 2.5–6 times, $P < 0.01$) upon the transition to the orthostatic position in the absence of respiratory perturbations at the corresponding frequencies (*HF*8*2, *HF*10*2, and *HF*12*2 relative to *HF*8*1, *HF*10*1, and *HF*12*1). The transition to the orthostatic position was accompanied by an increase in mean HR by 20–25% of the baseline value (Table 1). Therefore, the orthostatic changes in the parameters of the HRV spectrum were determined by changes in the general autonomic nervous state of the body towards an increased activity of the sympathetic nervous system (an increase in mean HR).

To study the relationships between different HRV ranges, we analyzed the correlations between the absolute spectral powers in the LF and HF ranges. If the subjects were in a horizontal position, the LF and HF components were significantly independent for respiration periods of 4 and 10 s (Fig. 2). Therefore, the HRV spectrum exhibited a resonance response at frequencies of 0.10 Hz [17, 18] and 0.25 Hz because the strength of the resonance effect only depended on the characteristics of the oscillatory process at the given frequency and was independent of other factors, which drastically decreased the correlation between the LF and HF ranges.

Thus, we demonstrated that the SANCH employs two oscillatory processes at frequencies of 0.10 and 0.25 Hz. We analyzed the properties of these oscillatory processes.

The highest correlation between the absolute powers of the LF and HF ranges in the tests with a respiration period of 8 s (Fig. 2) indicated a strong interaction between the 0.10- and 0.25-Hz processes in the SANCH under these conditions. This finding agrees with the hypothesis that the LF and HF components of the HRV spectrum are internally related [19, 20]. Apparently, respiration with a period of 8 s (0.125 Hz) had approximately the same effects on both 0.10- and 0.25-Hz oscillations in the system. Since the 8-s controlled breathing is close to the 0.10-Hz region of the HRV spectrum with respect to frequency, the LF range exhibited a stronger response, which was still lower

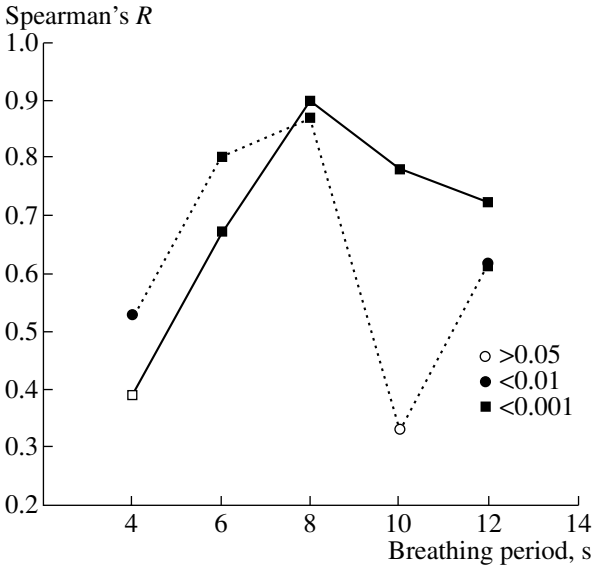

Fig. 2. Changes in Spearman's rank correlation coefficients for the LF and HF ranges of the HRV spectrum with the change in the period of controlled breathing during the orthostatic test. The solid and dotted lines correspond to the standing and lying positions of the subjects, respectively.

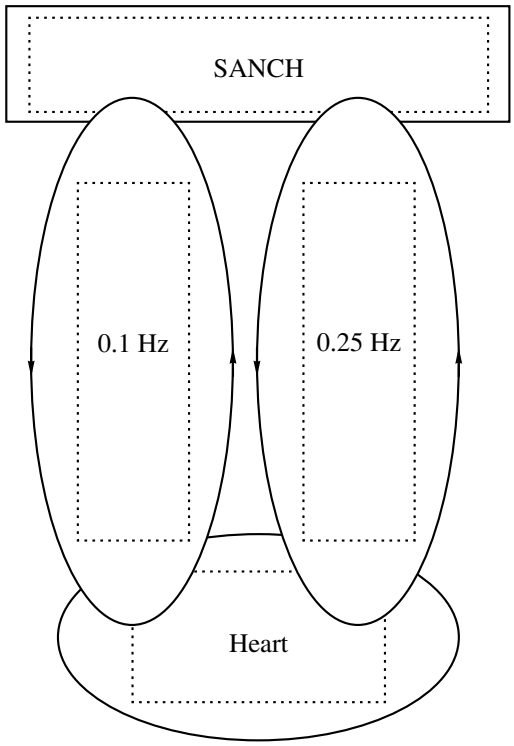

Fig. 3. Functional diagram of the SANCH in terms of the presence of two autonomous oscillatory circuits in it.

than the resonance one. The absolute values of the components of the HRV spectrum in the tests with controlled breathing at a frequency of 0.125 Hz were poor predictors of the subsequent orthostatic changes in spectral densities in both ranges (R(*LF*8*1 & *LF*8*2–1) = –0.37, P = 0.71; R(*HF*8*1 & *HF*8*2–1) = –0.39, P = 0.077). The changes in the HF range were predicted somewhat better but still nonsignificantly. The absolute power densities of the LF and HF components of the HRV spectrum better described their orthostatic changes under the conditions of controlled breathing with a period of 8 s (R(*LF*8*2 & *LF*8*2–1) = 0.545, P = 0.01; R(*HF*8*2 & *HF*8*2–1) = 0.573, P = 0.007) than at other frequencies of external respiratory perturbations at the SANCH input. The data confirmed our suggestion that the 0.125-Hz respiratory perturbations had equal effects on the 0.10- and 0.25-Hz SANCH oscillations; therefore, we may assume that the 0.125-Hz region in the HRV spectrum is a transition zone for the effects of these rhythmic oscillations in the system.

The existence of two autonomous oscillatory processes in the SANCH allows us to assume that the system contains two closed oscillatory circuits operating at these frequencies (Fig. 3), each of which is characterized by certain functional parameters, such as a feedback delay.

The independence of the 0.10- and 0.25-Hz processes in the SANCH was confirmed by a drastic decrease in the correlation between the orthostatic changes in the powers of the LF and HF ranges of the HRV spectrum in the presence of external respiratory perturbations at the given frequencies (Fig. 4), when the resonance response is expressed in the spectrum. Analysis of the results shown in Fig. 4 leads to the conclusion that the 0.10-Hz process depends on the 0.25-Hz one to a lower degree than vice versa. The 0.10-Hz process is also more powerful, as evidenced by a more pronounced resonance response in the tests with a respiration period of 10 s than in those with a period of 4 s (Table 1).

Thus, the 0.10-Hz oscillatory process in the SANCH is a potent autonomous mechanism controlling the cardiac rhythm, whereas the 0.25-Hz process is of a medium strength and the changes in its characteristics upon changes in the general autonomic nervous state of the body are somewhat coupled with those in the characteristics of the 0.10-Hz process.

Separate analyses of the LF and HF ranges of the HRV spectrum demonstrated that the mean spectral power density of the HF range in one set of respiratory tests (at all respiration periods) was determined by the power of the 0.25-Hz oscillatory process in the tests with a respiration period of 4 s, whereas the 0.10-Hz oscillations (measured at a respiration period of 10 s) determined the mean power of the LF range only in the case of external respiratory perturbations in the system in the given frequency range (Table 2). Therefore, it can be assumed that the 0.25-Hz oscillations in the SANCH constitute a very steady process whose effect on the output signal of the system (i.e., the HRV spectrum) depends little on the frequency composition of the input signals in the regulatory system, so that the oscillatory

process may be considered to be resistant to external factors. Conversely, external factors can considerably change the effect of the 0.10-Hz oscillatory process on the formation of the HRV spectrum.

These properties of the oscillatory processes in the SANCH are of fundamental importance for understanding the functional mechanism of the system. It may be presumed that a feedback mechanism is an important element of oscillatory circuits in a regulatory system [21]. The type of the afferent signal in the circuit depends on the characteristics of the afferent information coming to the controlling center of the oscillatory circuit. The qualitative diversity of the afferent information, determined by the effects of various factors of the system's environment, accounts for an efferent signal adequate to the given conditions, which is the optimal functional mechanism of a regulatory system.

Introduction of a harmonic component whose frequency is equal to the main frequency of the regulatory circuit oscillations into the afferent signal ensures a certain standardization of afferent information in a time interval in the given circuit; hence, the regulatory mechanism adequate to changing environmental conditions may be disturbed.

Thus, the use of controlled breathing may be regarded as an introduction of a periodic component into the external noise signal [22, 23] in order to study the functional characteristics of the SANCH.

The above data allow us to assume that the 0.25-Hz circuit incorporates external harmonic signals more poorly than the 0.10-Hz one; in other words, the 0.25-Hz regulatory mechanism has a very low sensitivity to external factors.

Apparently, the characteristics of the autonomic nervous mechanisms of heart activity control found in this study determine the physiological role of these mechanisms in the function of the whole system.

Our assumption on the susceptibility of the 0.10-Hz SANCH oscillatory process to the effects of external factors is confirmed by the characteristics of the changes in the relationships between the absolute powers of the LF and HF ranges of the HRV spectrum during the orthostatic test with controlled breathing with a period of 10 s (Fig. 2): the independence of the 0.10-Hz process from the HF range was dramatically decreased ($R(LF10*1\ \&\ HF10*1) = 0.33$, $P = 0.11$, changed to $R(LF10*2\ \&\ HF10*2) = 0.78$, $P = 0.00006$, upon the transition to the orthostatic position).

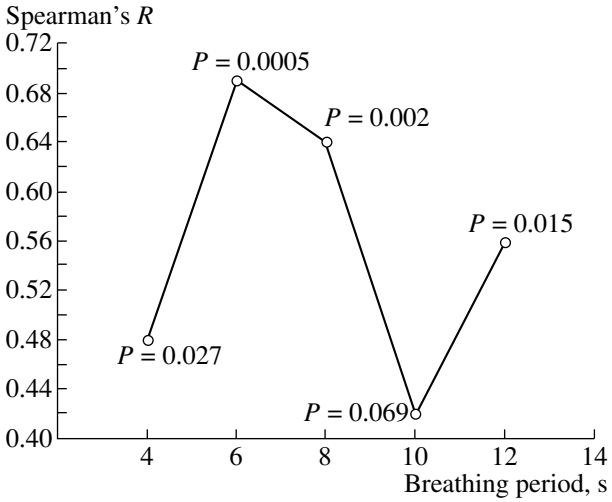

Fig. 4. Spearman's rank correlation coefficients for the orthostatic changes in the powers of the LF and HF components of the HRV spectrum at different periods of breathing.

The orthostatic test considerably changes the conditions under which the cardiovascular systems functions. This is reflected by changes in its main functional parameters: venous return, central and minute circulatory volumes, HR, etc. [24]. Changes in the conditions of the SANCH functioning inevitably lead to changes in its internal functional characteristics. We used the mean HR during the recording of the RR series as a parameter of the functional state of the SANCH. Note that the mean HR remained relatively constant as rhythmograms were recorded during respiratory tests in each test variant (Table 1).

Table 2. Spearman's rank correlation coefficients for the characteristics of the 0.10- and 0.25-Hz oscillatory processes of the SANCH

Parameters	Spearman's *R*	*P*-level	Parameters	Spearman's *R*	*P*-level
*HF*4*1 & *HF*6*1	0.795	<0.0001	*LF*10*1 & *LF*4*1	0.268	0.19
*HF*4*1 & *HF*8*1	0.61	0.0012	*LF*10*1 & *LF*6*1	0.439	0.028
*HF*4*1 & *HF*10*1	0.798	<0.0001	*LF*10*1 & *LF*8*1	0.49	0.0127
*HF*4*1 & *HF*12*1	0.81	<0.0001	*LF*10*1 & *LF*12*1	0.58	0.002
*HF*4*2 & *HF*6*2	0.84	<0.0001	*LF*10*2 & *LF*4*2	0.44	0.062
*HF*4*2 & *HF*8*2	0.71	0.0003	*LF*10*2 & *LF*6*2	0.46	0.051
*HF*4*2 & *HF*10*2	0.81	<0.0001	*LF*10*2 & *LF*8*2	0.83	<0.0001
*HF*4*2 & *HF*12*2	0.62	0.003	*LF*10*2 & *LF*12*2	0.83	<0.0001

Note: See Methods for designations.

Table 3. Spearman's rank correlation coefficients for correlation of the LF and HF components of the HRV spectrum with the mean HR (the inverse of *RR*)

RR & *HF*	Spearman's *R*	*P*-level	*RR* & *LF*	Spearman's *R*	*P*-level
4*1	0.66	0.0003	4*1	0.56	0.003
6*1	0.59	0.0018	6*1	0.62	0.0009
8*1	0.45	0.023	8*1	0.58	0.002
10*1	0.55	0.004	10*1	0.42	0.035
12*1	0.65	0.0004	12*1	0.65	0.0004
4*2	0.32	0.15	4*2	0.7	0.0004
6*2	0.66	0.001	6*2	0.46	0.037
8*2	0.73	0.00018	8*2	0.76	0.00006
10*2	0.64	0.0023	10*2	0.62	0.0039
12*2	0.71	0.0005	12*2	0.68	0.001

We demonstrated that the absolute values of the spectral densities of the powers of the LF and HF ranges correlated with the mean HR during the functional test (Table 3); the higher the powers of the HRV spectrum components, the lower the mean HR. Note that the HF component was a better determinant of HR in the subjects performing controlled breathing with periods of 4, 10, and 12 s in a horizontal position; in the subjects that had assumed a vertical position and performed controlled breathing with a period of 4 s, the relationship between the HF range and HR was lost. The LF component characterizes the state of the autonomic nervous tone almost always except for the 0.10-Hz resonance region of respiration.

Further analysis of the HF range showed that its absolute values are of medium prognostic importance for studying the possible orthostatic changes in HR only in the case of controlled breathing with a period of 4 s (R(*HF*4*1 & *RR*4*2–1) = –0.42, P = 0.05). Taking into account all the above data, we may conclude that the 0.25-Hz process in the SANCH characterizes the system adaptation reserve.

The LF oscillation process in the SANCH was not a prognostic criterion of individual adaptability of the system (R(*LF*?*1 & *RR*?*2–1) <|–0.25|, $P > 0.05$). On the other hand, neither the LF nor the HF ranges described the changes in the SANCH that had already taken place (R(*LF*?*2 & *RR*?*2–1) < |0.26|, $P > 0.05$; R(*HF*?*2 & *RR*?*2–1) < |0.22|, $P > 0.05$).

In tests with the subjects in a horizontal position, the functional load on the SANCH was moderate. Its functional state made it possible to detect small changes in the regulatory mechanism when controlled breathing was used to cause resonance in natural oscillatory circuits of the SANCH. In general, the changes in mean HR during a set of respiratory tests were independent of the absolute values of mean HR; however, we found one more correlation: R(*RR*8*1 & *RR*10–8*1) = –0.44, P = 0.027. Therefore, resonance at a frequency of 0.10 Hz caused changes in the system of HR control; however, the adaptation resource of the system remained within certain physiological limits. Therefore, the higher the initial HR, the lower the subsequent increase in HR in response to the 0.10-Hz resonance in the HRV spectrum. The mean HR increment correlated with the resonance increment in the 0.10-Hz region in response to controlled breathing with a period of 10 s: R(*RR*10–8*2 & *LF*10–8*2) = 0.69, P = 0.0007; R(*RR*10–12*2 & *LF*10–12*2) = 0.61, P = 0.005.

Note that the 0.10-Hz oscillations in the SANCH determined the HR at a given moment of time only when the subjects were in a standing position (R(*LF*10*1 & *RR*10*1) = 0.42, P = 0.055; R(*LF*10*2 & *RR*10*2) = 0.62, P = 0.0039), whereas the HF oscillations had this effect only when the subjects were in a lying position (R(*HF*4*1 & *RR*4*1) = 0.66, P = 0.0003; R(*HF*4*2 & *RR*4*2) = 0.32, P = 0.15). Probably, the effect of the 0.10-Hz regulatory circuit on the HR increases with a change in the external conditions under which the cardiovascular system functions, whereas the 0.25-Hz circuit controls the HR under relatively stable conditions.

The results of the analysis raise a question as to the possibility of studying the integrated SANCH structure by analyzing the characteristics of its oscillatory circuits. Taking into account that the resonance response of the SANCH largely determines the formation of the HRV structure, we may assume that it is impossible to estimate the control system as a whole because the resonance effect can only characterize the particular oscillatory process in the SANCH on the basis of which it has been formed. Therefore, it may be concluded that the properties of the whole, integrated SANCH can be studied in the absence of external perturbations at the system input in the region of resonance frequencies (0.10 and 0.25 Hz). This rule primarily concerns the 0.10-Hz oscillatory process because its power is very high. Therefore, when resonance appears, it considerably affects the HRV, which interferes with adequate analysis of other parameters of the system. This is also true for the analysis of the 0.25-Hz process in the case of 0.10-Hz resonance because, as we demonstrated, the characteristics of the 0.25-Hz component of the HRV spectrum are determined, to a certain degree, by the 0.10-Hz process.

Thus, the integrated SANCH structure should be studied in the absence of external perturbations at frequencies of 0.10 and 0.25 Hz, e.g., under conditions of perturbations in the transition zone of the effects of these components.

CONCLUSIONS

(1) The 0.10-Hz oscillatory process in the SANCH is a potent mechanism regulating the cardiac rhythm, its characteristics being influenced by external factors.

This process plays an important role in HR control in the cases where the general autonomic nervous state of the body is changed.

(2) The 0.25-Hz process is a HR regulatory mechanism of medium strength, is resistant to external factors, and characterizes the adaptation reserve of the SANCH. This process determines the HR in the case of a stable autonomic nervous state of the body.

(3) The properties of the internal oscillatory processes in the SANCH can be studied on the basis of the resonance effect in the HRV spectrum in tests with controlled breathing at the frequencies of the oscillations.

(4) Apparently, the general properties of the SANCH should be studied in the absence of external perturbations at frequencies close to the resonance frequencies of the system, i.e., 0.10 and 0.25 Hz.

REFERENCES

1. Glass, L., Synchronization and Rhythmic Processes in Physiology, *Nature*, 2001, vol. 410, p. 277.
2. Ludwig, C., Beiträge zur Kenntnis des Einflusses der Respirationsbewegung auf den Blutlauf im Aortensystem, *Arch. Anal. Physiol.*, 1847, vol. 13, p. 242.
3. Dornhorst, A.C., Howart, P., and Leathart, G.L., Respiratory Variations in Blood Pressure, *Circulation*, 1952, no. 6, p. 553.
4. Kiselev, A.R. and Kolizhirina, O.M., A New Approach to Studying the Internal Characteristics of the Autonomic Nervous Control of the Heart, *Sarat. Nauchn.-Med. Vestn.*, 2002, vol. 1, no. 1, p. 45.
5. Pitzalis, M.V., Mastropasqua, F., Massari, F., *et al.*, Effect of Respiratory Rate on the Relationships between RR Interval and Systolic Blood Pressure Fluctuations: A Frequency-Dependent Phenomenon, *Cardiovasc. Res.*, 1998, vol. 38, no. 2, p. 332.
6. Kay, S.M. and Marple, S.L., Spectrum Analysis: A Modern Perspective, *Proc. IEEE*, 1981, vol. 69, p. 1380.
7. Marple, S.L., Jr., *Digital Spectral Analysis with Applications*, Prentice-Hall, 1987.
8. Heart Rate Variability: Standard of Measurement, Physiological Interpretation, and Clinical Use, *Circulation*, 1996, vol. 93, no. 5, p. 1043.
9. Shapiro, S.S., Wilk, M.B., and Chen, H.J., A Comparative Study of Various Tests of Normality, *J. Am. Stat. Assoc.*, 1968, vol. 63, p. 1343.
10. Wilcoxon, F., Individual Comparisons by Ranking Methods, *Biometr. Bull.*, 1945, vol. 1, p. 80.
11. Wilcoxon, F., Probability Tables for Individual Comparisons by Ranking Methods, *Biometrics*, 1947, vol. 3, p. 119.
12. De Boer, R.W., Karemuker, J.M., and Stracker, J., On the Spectral Analysis of Blood Pressure Variability, *Am. J. Physiol.*, 1986, vol. 251, no. 4, part 2, p. 685.
13. De Boer, R.W., Karemuker, J.M., and Stracker, J., Relationships between Short-Term Blood Pressure Fluctuations and Heart Variability in Resting Subjects: I. A Spectral Analysis Approach, *Med. Biol. Eng. Comput.*, 1985, vol. 23, no. 4, p. 352.
14. De Boer, R.W., Karemuker, J.M., and Stracker, J., Relationships between Short-Term Blood Pressure Fluctuations and Heart Variability in Resting Subjects: II. A Simple Model, *Med. Biol. Eng. Comput.*, 1985, vol. 23, no. 4, p. 359.
15. De Boer, R.W., Karemuker, J.M., and Stracker, J., Hemodynamic Fluctuations and Baroreflex Sensitivity in Humans: A Beat-to-Beat Model, *Am. J. Physiol.*, 1987, vol. 253, no. 3, p. 680.
16. Madwed, J.B., Albrecht, P., Mark, R.G., and Cohen, R.J., Low-Frequency Oscillation in Arterial Pressure and Heart Rate: A Simple Computer Model, *Am. J. Physiol.*, 1989, vol. 256, no. 6, p. 1573.
17. Malpas, S.C., Todd, A.H., Navakatikyan, *et al.*, Resonance in Renal Vasculature Evoked by Activation f the Sympathetic Nerves, *Am. J. Physiol.*, 1999, vol. 276, no. 45, p. 1311.
18. Kuterman, E.M. and Khaspekova, N.B., Heart Rate during the Respiratory Test with Six Breaths a Minute, *Fiziol. Chel.*, 1992, vol. 18, no. 4, p. 52.
19. Malliani, A., Physiological Interpretation of Spectral Components of Heart rate Variability (HRV), *Vestn. Aritmol.*, 1998, no. 9, p. 47.
20. Cavalcanti, S., Arterial Baroreflex Influence on Heart Rate Variability: A Mathematical Model–Based Analysis, *Med. Biol. Eng. Comput.*, 2000, vol. 38, no. 2, p. 189.
21. Ringwood, J.V. and Malpas, S.C., Slow Oscillations in Blood Pressure via a Nonlinear Feedback Model, *Am. J. Physiol. Reg. Integr. Comp. Physiol.*, 2001, vol. 280, no. 4, p. 1105.
22. Radhakrishna, K.K.A., Dutt, D.N., and Yeragani, V.K., Nonlinear Measures of Heart Rate Time Series: Influence of Posture and Controlled Breathing, *Auton. Neurosci. Bas. Clin.*, 2000, vol. 83, no. 3, p. 148.
23. Patwardhan, A., Evans, J., Bruce, E., and Knapp, C. Heart Rate Variability during Sympatho-Excitatory Challenges: Comparison between Spontaneous and Metronomic Breathing, *Integr. Physiol. Behav. Sci.*, 2001, vol. 36, no. 2, p. 109.
24. Aronov, D.M., Lupanov, V.P., Rogoza, A.N., and Lopatin, Yu.M., Functional Tests in Cardiology: Lecture VII: Functional Tests Based on Local Action on Nerve Terminals and Targeted Change in Venous Return, *Kardiologiya*, 1996, no. 7, p. 77.