PHYSIOLOGY

Structural Analysis of Heart Rhythm Spectrogram in Narcotized Cats

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Eight types of peaks were revealed in the cardiac rhythm spectrum during acute experiments on vagotomized cats. Some peaks had no physiological nature and resulted from specificity of ECG processing by Fourier analysis, while others reflected myogenic reaction of the sinoatrial node (length—dependence of automaticity) to changes in venous return caused by respiratory-induced and other variations of the blood flow in the cardiovascular system.

Key Words: *heart rate spectrogram; venous return oscillations; sinoatrial node; automaticity; length-dependence*

Spectrum analysis of *RR* interval series (heart rhythmogram, HRG) by Fourier transform is a basic method for evaluation of the nervous regulation of the heart [1-5], although the origin and functional relations of many peaks in the heart rhythm spectrogram (HRS) are the matter of controversy [3,4].

Our aim was structural analysis of the HRS obtained by Fourier analysis in acute experiments on animals.

MATERIALS AND METHODS

Experiments were carried out on artificially ventilated temperature-controlled cats (37°C, body weight 2.5-3.5 kg, *n*=15) anesthetized with intraperitoneal chloralose and nembutal (75+15 mg/kg). HRS was calculated in the frequency range of 0.003-0.500 Hz from the 2-10 min ECG fragments recorded using a Poly-Spectrum-3 cardiac rhythm analyzer (Neurosoft). All cats were artificially ventilated at the rate of 0.354 Hz (21.24 min^{-1}), thereafter the following procedures were performed in series: bilateral vagotomy at the level of the thyroid cartilage, blockade of β-adrenoceptors with propranolol (1 mg/kg i.v.), and cooling to 27°C. The corresponding ECG records were obtained for each stage of the study.

RESULTS

Initially, the heart rate (HR) and respiratory rate varied at 37°C within 139-220 (175±6) and 4-17 (10.0±0.9) min^{-1}, respectively. The variation range and variation coefficient of *RR* interval were 9±1 msec and 0.45±0.05%, respectively. Artificial ventilation, vagotomy, propranolol, and total hypothermia modulated the parameters of cardiac rhythm, but were not reflected in the mechanism of HRS formation, which retained the following peaks:

— unspecific drift-dependent peaks (0.003-0.040 Hz) reflecting the linear and non-linear drift of HGR (Figs. 1 and 2). The peculiar feature of these peaks was their preservation during equal by amplitude but different in direction changes in HR, and dra-

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matic increase after the very small shifts in HR by few percents, which corresponded to several milliseconds of HRG drift for a 2-min ECG fragment;

— the first of the drift-dependent peaks (Figs. 1 and 2), which always depended on the record length according to equation $f=1/t$, where f is peak frequency (Hz), and t the record length (sec);

— accidental peaks (0.04-0.15 Hz) reflecting aperiodic (*i.e.* single for a particular record) variations in HR lasting from 6-7 to 25 sec;

— arrhythmic peaks uniformly filling the entire HRS range resulting from even a single extrasystole;

— true respiratory peaks (Figs. 1 and 2) strictly corresponding to the respiration rate (0.07-0.28 or 0.354 Hz for the spontaneous or artificial respiration, respectively);

— resonance respiratory peaks (from 1 to 6) followed the true respiratory peak at the same intervals equal to the respiration rate (Figs. 1 and 2);

— specific peaks of unclear origin (Fig. 1), whose amplitude (Table 1, No. 13) and frequency (f= 0.04-0.10 or 0.058±0.006 Hz) significantly changed after hypothermic bradycardia (Table 1, No. 14; f=0.034±0.002 Hz, $p<0.001$);

— rhythm-depended interference peaks, which appeared simultaneously in the ranges of 0.000-0.354, 0.354-0.708, and 0.708-1.062 Hz after the changes in HR caused by propranolol or hypothermia under artificial ventilation performed at the rate of 0.354 Hz. The interval between the adjacent interference peaks was always equal to respiration rate irrespectively of their location within the above ranges (Fig. 2).

The HRS parameters (the amplitude and number of drift-dependent, specific, interference, and respiratory peaks) had a variable character: thay varied continuously even under stable HR and respiration rate. The transition to artificial ventilation (Table 1, Nos. 1-8), bilateral vagotomy (Table 1, Nos. 6, 11), and stage-by-stage shortening of the analyzed ECG fragment from 10 to 2 min (n=15) produced no effect on peak variability and continuous variation of their parameters. However, in vagotomized cats, stabilization of body temperature and propranolol dramatically reduced the drift-dependent peaks and even completely eliminated them (Fig. 2, Table 1, Nos. 9, 10). Propranolol decreased (in some cats eliminated) or increased the specific peaks and slightly shifted their frequency to any direction. In addition, it increased the amplitude of true respiratory peaks, the increments surpassed the corresponding changes in the respiratory peaks in HRG (Fig. 1, 2; Table 1, Nos. 11, 12).

HRS had a complex heterogeneous character. In addition to physiological elements (the respiratory, specific, and interference peaks) it contained artifacts caused by particular mathematical processing of HRG with Fourier transform algorithm: the initial time-dependent, accidental, and arrhythmic peaks and the pronounced disproportion in the changes of HR, HGR

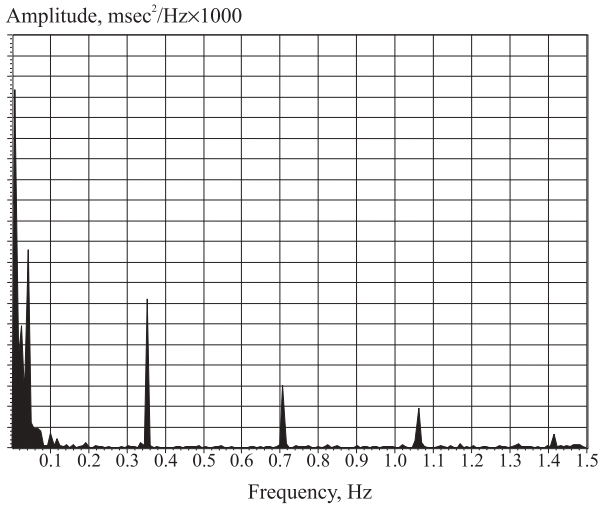

Fig. 1. Heart rate spectrogram in artificially ventilated vagotomized cat prior to propranolol. Duration of ECG fragment was 2 min, HR 219 min^{-1}, mean *RR* interval 274 msec, HRG drift amplitude 3 msec (1.1% mean *RR* interval), and amplitude of respiratory waves in HRG 2-3 msec (0.7-1.1% mean *RR* interval). From left to right: the time-dependent peak (0.0083 Hz), the drift-dependent peak (0.025 Hz), the specific peak (0.042 Hz), non-identified peaks (0.07, 0.10, and 0.12 Hz), the true respiration peak (0.354 Hz), the resonance respiratory peaks (0.708, 1.062, 1.408 Hz). Calibration here and in Fig. 2: abscissa 0.1 Hz, ordinate 0.01 msec²/Hz×1000.

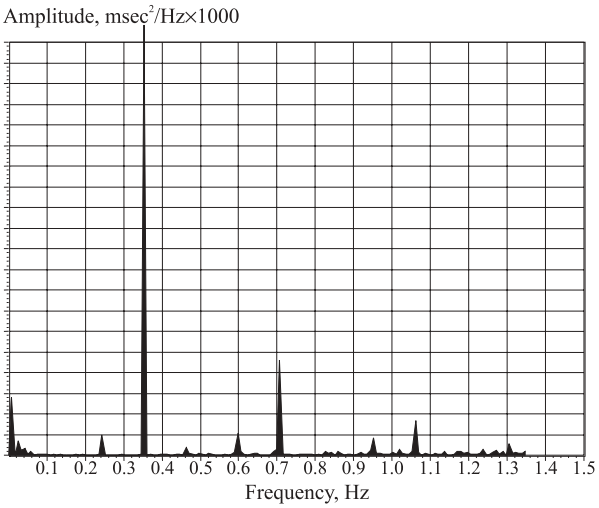

Fig. 2. Heart rate spectrogram in artificially ventilated vagotomized cat after injection of propranolol. Duration of ECG fragment was 2 min, HR 163 min^{-1}, mean *RR* interval 368 msec, HRG drift amplitude 1 msec (0.3% mean RR interval), amplitude of respiratory waves in HRG 3-4 msec (0.8-1.1% mean *RR* interval). From left to right: the time-dependent peak (0.0083 Hz), the drift-dependent peak (0.025 Hz), the specific peak (0.042 Hz), the true respiration peak (0.354 Hz), the resonance respiratory peaks (0.708 and 1.062 Hz), interference peaks (0.244, 0.598, 0.952, and 1.302 Hz).

TABLE 1. Amplitude of Basic Peaks in HRS of Narcotized Cats (msec²/Hz×1000; $M \pm m$, n=10)

No.	Peaks	Number of observation	Amplitude
1	Drift-dependent peaks during spontaneous respiration before vagotomy	15	0.174±0.056
2	Drift-dependent peaks during artificial respiration before vagotomy	15	0.177±0.065
3	Specific peaks during spontaneous respiration before vagotomy	14	0.028±0.008
4	Specific peaks during artificial respiration before vagotomy	15	0.028±0.008
5	True respiratory peak during spontaneous respiration before vagotomy	15	0.182±0.063
6	True respiratory during artificial respiration before vagotomy	11	0.238±0.099
7	The first resonance peak during spontaneous respiration before vagotomy	13	0.137±0.068
8	The first resonance peak during artificial respiration before vagotomy	15	0.168±0.052
9	Drift-dependent peaks during artificial respiration after vagotomy and before propranolol	13	0.221±0.059
10	Drift-dependent peaks during artificial respiration after vagotomy and propranolol	13	0.034±0.008
11	True respiratory peak during artificial respiration after vagotomy and before propranolol	13	0.223±0.083
11	True respiratory peak during artificial respiration after vagotomy and propranolol	13	0.602±0.186*
12	Specific peaks at initial temperature during artificial respiration and after denervation of the heart (vagotomy+propranolol)	15	0.031±0.011
13	Specific peaks after denervation of the heart (vagotomy+propranolol+ hypothermia) during hypothermic bradycardia (HR=66±2 min^{-1})	15	0.311±0.118**

Note. Body temperature was stabilized at 37°C (Nos. 1-13) or 27°C (No. 14). *p<0.05 compared to No. 11, **p<0.001 compared to No. 13 (method of direct differences).

amplitude, and HRS peaks during various functional tests. The important sources of the artifacts were the drift-dependent peaks. They were sharply suppressed by propranolol, which can attest to the presence of sympathetic factor in the nature of these peaks. However, hypersensitivity of these peaks to any other drift in HR (first of all, the temperature-induced drift) and complete identity of their shape during opposite shifts in HR exclude the use of the drift-dependent peaks for assessment of the direction and degree of sympathetic influences. Similar changes in the amplitude of respiratory peaks were revealed after injection of propranolol.

Probably, the specific, true respiratory, resonance, and interference peaks in HRS reflect physical aspects of hemodynamics, which could be useful in the study of oscillatory and auto-oscillation processes in the cardiovascular system. However, the fact of existence of these peaks in virtually denervated heart (vagotomy, propranolol, and hypothermia) is even more important. These conditions leaved only one external factor, which could regulate the work of the heart: venous return modulated by systolic, respiratory, and other variations of the blood flow. Since these modulating factors change the diastolic volume of the atria and the size of the sinoatrial node, the myogenic dependence of nodal automaticity on its geometrical length can be an important mechanism underlying heart rate variability, whose origin was previously explained by the nervous and metabolic influences [1-5].

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