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PHYSIOLOGY

Cardiorespiratory Changes in Beluga in Response to Acoustic Noise

O. I. Lyamin[a, b, c], S. M. Korneva[b], V. V. Rozhnov[a], and L. M. Mukhametov[a, b]

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Acoustic noise is one of the most important factors of anthropogenic action on marine mammals. The majority of studies of noise influence on marine mammals to date have dealt with the connection between noise parameters and subsequent changes in animals' hearing. The influence of noise on the physiological condition of cetaceans has been poorly studied [1]. The heart rate (HR) and respiratory rate are objective indices that may be used for evaluation of an animal's condition. In many studies, HR is used for the assessment of reactions and conditions of the autonomic nervous system of humans and animals in anxiety and stress [2, 3], as well as under the action of different external stimuli [4], including acoustic noise [5]. The only study conducted on one bottlenose dolphin indicated that HR in animals accelerated upon hearing the audio recording of whistling of other animals [6]. The aim of this research was to study the influence of acoustic noise on HR and characteristics of beluga respiration at frequencies overlaying the zone of best hearing [7].

The study was conducted on a young male beluga (weight, 195 kg; length, 240 cm) at the Utrish Marine Station of the Severtsov Institute of Ecology and Evolution. The animal was captured two month before the beginning of research.

During the experiment, the beluga was placed onto a stretcher and transferred to a bath containing seawater ($4 \times 0.8 \times 0.8$ m). A total of 20 experiments with a duration of 2–4.5 h each were conducted. An emitter was used to present band-pass acoustic noises that differed in frequency (19–27, 27–38, 38–54, 54–78, and 78–108 kHz), intensity (140, 150, and 160 dB), and duration (1, 3, and 10 min) to the beluga. During one day, from 1 to 6 noises (on average, 4 noises per day) were presented; more often (16 out of 20 cases), the noises were of one frequency and growing duration. Recording of electrocardiogram (ECG) was conducted by means of a polygraph using two suction cup disk electrodes. Instantaneous HR was assessed as inverse values of time intervals between two consecutive heartbeats and were averaged over 1-min time intervals. The average HR in a 4- to 5-min interval before noise (control) was compared to the HR during the presentation of noise (in 1-min intervals or throughout the presentation of noise), as well as after the end of noise. The time of respiratory acts was identified using video recording and time-synchronized with ECG.

The HR of the beluga laying on the stretcher was characterized by expressed arrhythmia: periods of bradycardia, which accounted for the phase of breath holding (respiration pauses longer than 60 s) alternated with periods of tachycardia, which coincided with a series of respiration acts (2–10 inspirations over 30-s intervals; Fig. 1). The minimal instantaneous HR during breath holding decreased to 20 beats/min. The maximal HR during the series of breaths reached 85 beats/min. The average HR in different experiments varied from 41 to 63 beats/min (during 5-min time intervals). Thus, the respiration and HR characteristics of the beluga lying on the stretcher were comparable to those of a beluga kept in a pool [8, 9].

Simultaneously with the beginning of noise, the HR of the beluga increased dramatically; i.e., manifest tachycardia that lasted for several minutes developed (Fig. 1, noises *A* and *B*). The instantaneous HR reached the maximal values together with the beginning of noise and then increased to 100 beats/min (approximately 15 beats/min more than under the control conditions); after that, it gradually decreased. Sometimes, during 10-min noise, tachycardia was replaced by bradycardia. The minimal values of instantaneous HR

[a] *Severtsov Institute of Ecology and Evolution, Russian Academy of Sciences, Leninskii pr. 33, Moscow, 117091 Russia*

[b] *OOO Utrish Dolphinarium, Leninskii pr. 33, Moscow, 117091 Russia*

[c] *University of California at Los Angeles, CA 90095, United States*

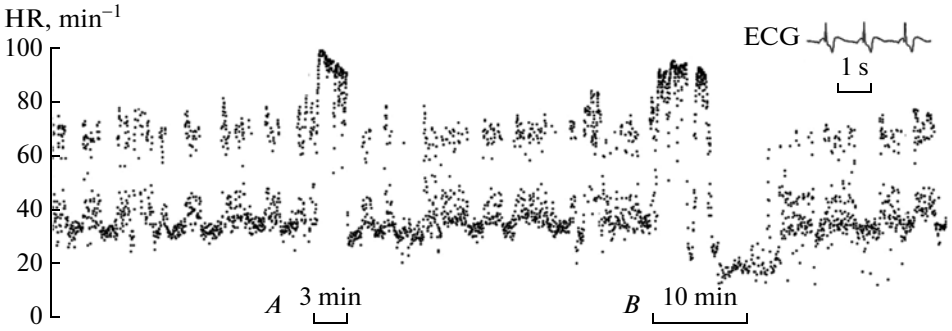

Fig. 1. HR in a beluga before acoustic noise. Each point is an instantaneous HR at a certain moment (beats/min). *A* and *B*, time intervals of presentation with a duration of 3 and 10 min, respectively. Frequency, 19–27 kHz; intensity, 150 dB. In the upper corner a fragment of ECG is shown.

in such periods were 12 beats/min, i.e., lower than under the control conditions. The average HR in the first minute of noise with an intensity of 150 dB did not depend on the order of noise presentation in the experiment (one-way ANOVA with repetitions, $p > 0.05$; $F[3.18] = 1.98$). Thus, no habituation of beluga to noise occurred; this allowed us to conduct statistical treatment without taking into account the order of noise presentation in the experiment.

Acoustic noise with a duration of 1 min and intensity of 150 dB was accompanied by a sharp increase in the average HR during the time of noise action at all frequencies ($p < 0.03$ for all frequencies, paired t test). At the first minute of noise HR grew up to 208% of control values (Fig. 2). And degree of acceleration of HR of beluga with high reliability depended on frequency of acoustic noise (ANOVA, $p = 0.02$; $F[4.35] = 3.21$). With an increase in noise frequency, the excess of average HR over the control decreased (from 178 to 140% of the values in the control period). The expression of tachycardia during noise at frequencies of 19–27 kHz was much higher than at frequencies 54–78 and 78–108 kHz (paired comparison, $p < 0.05$).

Tachycardia caused in the beluga by noise of the lowest frequency (19–27 kHz) was approximately at the same level for no less than 3 min, because average values of HR assessed for one- and three-day intervals did not differ ($p > 0.05$; t test). When the frequency of 3 min noise increased from 19 to 54 kHz, the average HR during the noise also significantly decreased ($p < 0.01$; $F[4.25] = 5.15$). Growth of HR at noise of 19–27 kHz was much bigger than at frequencies greater than 38 kHz ($p < 0.05$, pairwise comparison). Average values of HR at noise action for 3 min were higher than in control period ($p < 0.05$; paired t test) for all frequencies except for 54–78 kHz (noise was presented only 3 times).

During 10-min noise with a frequency of 19–27 kHz, the average HR was also greater than the control values ($p < 0.05$, t test; Fig. 2) and was 140%. At higher frequencies of noise, the increase in the HR did not exceed 10% in comparison with the control period ($p > 0.05$). More detailed analysis indicated that tachycardia caused by noise with a frequency of 19–27 kHz differed significantly from control values during no less than 4 min ($p < 0.05$, pairwise comparison; after ANOVA with repetitions, $p < 0.01$, $F[10.44] = 8.27$). During noise with a frequency of 27–38, 38–54, or 54–108 kHz, the increase of HR above background values was significant only during the first 1-min interval ($p < 0.05$). Thus, the intensity and duration of tachycardia that developed in the beluga upon presentation of acoustic noise with a duration of 1, 3, and 10 min with frequencies from 19 to 108 kHz depended on noise frequencies and were maximal at frequencies of 19–27 kHz. The response decreased with the growth of noise frequency.

After the end of noise, HR decreased. Based on the data, we may assess the dependence of HR changes after cessation of noise with a frequency of 27–38 kHz and durations of 1 and 10 min. The average HR during a 5-min interval after the end of noise with a duration of 1 min (5 presentations) was less (44 ± 3 beats/min) than in the control period of the same duration (56 ± 3 beats/min; 78% compared to the control period; $p < 0.001$, after ANOVA with repetitions; $F[2.8] = 73.97$; $p < 0.001$). Differences in the average HR during 5-min intervals were determined first of all by the sharp decrease in HR in the first minute after the end of noise (on average, 58% compared to the control period; $p < 0.05$; after ANOVA with repetitions, $F[4.20] = 5.10$; $p = 0.001$). After the end of noise with a duration of 10 min (in total, 5 presentations), HR in the first minute was also lower (by 45%; Fig. 3) than in the control period ($p < 0.05$; after ANOVA with repetitions, $p < 0.001$, $F[4.20] = 7.74$). Thus, not only the beginning, but also the termination of acoustic noise was accompanied by changes in the HR in beluga, and the direction of changes were the opposite (increase and decrease, respectively).

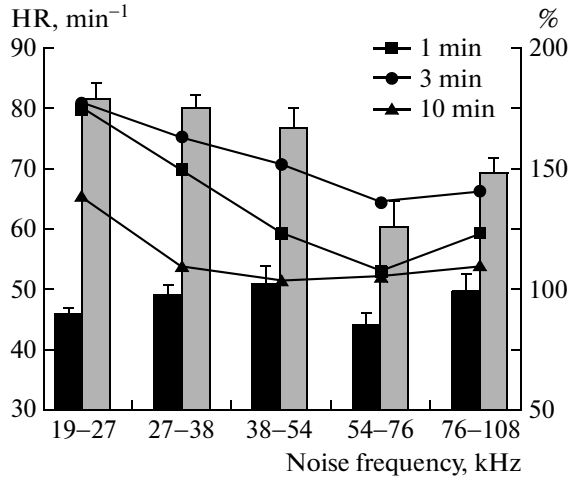

Fig. 2. HR in a beluga before and during presentation of acoustic noise with a duration of 1, 3, and 10 min at different noise frequencies. Dark and light columns show HR (beats/min) in 5-min intervals before and after action of noise with a duration of 1 min, respectively. The lines connect values of relative HR during the presentation of noise of different frequency with durations of 1, 3, and 10 min (in percent of the HR before presentation of noise). Noise intensity, 150 dB.

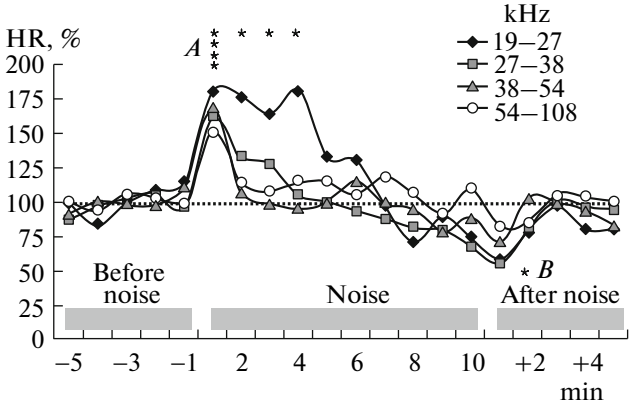

Fig. 3. HR in a beluga before, during and after presentation of acoustic noise with a duration of 10 min at different noise frequencies. The abscissa shows 1-min time interval. The lines connect the values of relative HR during noise with frequencies of 19–27, 27–38, 38–54 kHz, as well as 54–78 and 78–108 kHz (in percent of the HR before presentation of noise). *A*: *—statistically significant differences ($p < 0.05$) between HR in 1-min intervals during noise of different frequencies (top-down: 19–27, 27–38, 38–54 kHz as well as 54–78 and 78–108 kHz) from HR before presentation of noise (averaged over 5 min). *B*: *—statistically significant differences ($p < 0.05$) between HR in the first 1-min interval after the end of noise and HR before presentation of noise. Noise frequency, 27–38 kHz.

In this study, noises with intensities of 150 dB and, rarely 160 and 140 dB were presented to the beluga. Taking into account that HR during 1 min of noise with an intensity of 150 dB at frequencies of 19–27 (178 ± 6% of the HR in the control, n =5) and 27–38 kHz (164 ± 7%, $n = 14$; $p < 0.15$, t test) did not differ, we analyzed the dependence of HR change in the beluga on the noise intensity by pooling the data obtained upon presentation of the noises of these two frequencies. According to expectations, the intensity of tachycardia in the beluga depended on the noise intensity ($p < 0.01$, $F[2.28] = 5.41$). The average HR at a noise intensity of 140 dB (123 ± 7%, $n = 4$) was much less than at intensities of 150 dB (167 ± 5%, $n = 19$) and 160 dB (168 ± 12, $n = 8$; $p < 0.05$, pairwise comparison). Thus, the degree of tachycardia caused by acoustic noise in the beluga increased with the growth of noise intensity from 140 to 150 dB.

The changes in the respiration pattern during noise presentation were much less than the changes in the HR and were confined to the first minute of noise (table). For example, during the first minute of 3-min noise with frequencies of 19–27 and 27–38 kHz, which caused the greatest enhancement of HR, the average duration of the respiration pause (RP) decreased and was 72 and 60% of the average RP during the control period (4 min before noise), respectively. However, considering the degree of variability of RP in the beluga, these differences were statistically nonsignificant ($p > 0.05$). On the other hand, upon presentation of noise with a frequency of 76–108 kHz, which caused the minimal change in HR, the RP became even shorter (39% of the control values; $p < 0.05$; after rank-order ANOVA, paired comparison, $p < 0.05$). Finally, noise with a frequency of 38–54 kHz did not result in change of respiration. Respiration pauses of the beluga during the second and third minutes of noise were practically the same as before the beginning of the noise. Thus, respiration rate of the beluga upon presentation of loud acoustic noise increased. These changes lasted for no more than 1 min and were not directly associated with the changes in HR.

This is the first study on the response of the cardiovascular system of a beluga to prolonged intense acoustic noise with a frequency overlapping the zone of best hearing [7]. The response of the beluga to noise consisted in an increase in the HR, which depended on the frequency and intensity of noise. Acceleration of HR in the beluga in response to noise is the first component of the "acoustic startle response" to sudden presentation of a strong sound stimulus thoroughly studied on humans and land mammals. The intensity of tachycardia indicates the interaction of different branches of autonomic nervous system: activation of the sympathetic systems and suppression of the parasympathetic systems [10]. Upon changes in common environmental conditions, HR may increase by 60% compared to control values [2, 3]. Tachycardia is considered a cardiovascular component of the stress

Average duration of respiration pause in a beluga before and during presentation of 3-min acoustic noise of different frequencies

Noise frequency (kHz)	Number of presentations	RP_{av}, s			
		before noise (control)	minute from the beginning of noise		
			1	2	3
19–27	4	20 ± 3 (7–76)	13 ± 1 (8–19)	19 ± 2 (12–30)	30 ± 14 (9–182)
27–38	9	23 ± 2 (7–67)	18 ± 3 (8–41)	39 ± 10 (12–109)	46 ± 17 (5–208)
38–54	7	23 ± 2 (7–95)	25 ± 4 (7–77)	63 ± 28 (9–163)	32 ± 7 (9–59)
54–78	3	25 ± 5 (5–66)	14 ± 1 (6–19)	23 ± 1 (9–35)	24 ± 6 (17–36)
78–108	6	30 ± 2 (12–106)	13 ± 1* (8–28)	26 ± 5 (11–84)	27 ± 5 (15–48)

Note: RP in the control is the average over 4 min for all presentations; during noise, the average per minute before noise. In parenthesis, the minimal and maximal values of RP during all presentations are given. The asterisk indicate a significant difference from the control (one-way ANOVA, $p < 0.01$, rank-order pairwise comparison, $p < 0.05$). Noise intensity is 150 dB.

reaction, indicator of animal defense reaction and level of "social" stress [11]. Much less expressed (only several percent) and short lasting (up to 30 s) acceleration of HR is observed in animals in response to sound stimuli that have emotional meanings of danger (calls of dominant males in primates [12]; sounds of jaw clap treat in dolphins [6]).

In was shown that presentation to a bottlenose dolphin of acoustic noise with a frequency from 4 to 11 kHz resulted in temporary shifts in hearing thresholds at an intensity of more than 179 dB [13]. Our data indicate that severe tachycardia developed in the beluga at lower noise intensities (as low as 140 dB); at higher intensities, the HR could reach a twofold excess over the control values and last for no less than 4 min. The changes in the respiration parameters in the beluga during noise with a duration of up to 10 min were smaller and lasted for less than 1 min. To summarize, HR may serve as a criterion of physiological response (state) to acoustic noises, including anthropogenic, in belugas and, most likely, in other cetaceans.

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