Assessment of Cardiac Autonomic Nervous Activities During Heliox Exposure at 24 Atm Abs

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Background: This experiment was designed to examine the involvement of the autonomic nervous system in the production of hyperbaric bradycardia. ***Methods:*** Four male divers were exposed to a He-O_2 (heliox) environment at 24 atmosphere absolute (atm abs) for 7 d. The heart rate (HR) and respiratory rate were recorded at rest in the morning (0700 h) and at night (2230 h) on 1 d during a 5-d predive control, 2 d during a 7-d saturation dive at 24 atm abs, 2 d during decompression, and on 1 d during a 4-d postdive period. Cardiac sympathetic and parasympathetic activities were estimated by using a spectral analysis of the variability of R-R intervals. ***Results:*** The morning HR did not fluctuate throughout the experimental days. The night time HR decreased ($p < 0.05$) by 11.8% on the first day at 24 atm abs compared with that of the predive control. The bradycardia diminished gradually and returned to the predive level with continued exposure at 24 atm abs. The high-frequency power of the cardiac variability, an index of cardiac parasympathetic activity, increased ($p < 0.05$) only in the first night at 24 atm abs, whereas the low-frequency power and a ratio of low- to high-frequency power, an index of cardiac sympathetic activity, were unchanged. ***Conclusions:*** The present results suggest that an increased parasympathetic activity rather than a decrease in the sympathetic activity is responsible for the bradycardia on exposure to heliox dry saturation dive at 24 atm abs. The mechanism of the gradual disappearance of the bradycardia is unknown, but perhaps it may be related to the development of cardiovascular deconditioning.

IT IS WIDELY KNOWN that exposure to a high pressure environment induces bradycardia in humans and animals (5,9,15,16). Also, both oxygen-dependent or oxygen-independent factors are operating to cause this so-called hyperbaric bradycardia (9). Hyperoxia causes bradycardia both at the sea level and in hyperbaria (11,18). Respiratory inert gases, high environmental pressure, and high gas density have been proposed as oxygen-independent factors (9). Whatever the reason, the bradycardia occurs via the effect of autonomic nervous activity on the sinus node. That is, the bradycardia is attributable to either a reduced sympathetic activity or an increased parasympathetic activity during exposure to hyperbaria.

Spectral analysis of cardiac variability has been used as an investigative tool for the estimation of the cardiac autonomic nervous activity (2,4,7,13,14). It has been suggested that low-frequency (<0.15 Hz) power of cardiac variability is associated with both sympathetic and parasympathetic activities (1,2,4,17), and high-frequency (>0.15 Hz) power is associated exclusively with cardiac parasympathetic activity (1,2,4,7,13,14,17). The ratio of low- and high-frequency power associates closely the sympathetic activity (12–14). The present study, therefore, was designed to measure R-R intervals over a 25-d experimental period, including a 7-d saturation period at 24 atmosphere absolute (atm abs). We estimated the role of autonomic nervous systems from a spectral density of cardiac interval variabilities and attempted to evaluate the involvement of sympathetic and parasympathetic nerve activities in the pressure-induced bradycardia.

METHODS

Subjects

Four healthy males, 27 ± 3 (mean ± SE) yr, 63.9 ± 3.5 kg in weight, 173 ± 3 cm in height and 11.9 ± 2.0% in body fat served as subjects. All subjects were trained divers and also served as subjects for earlier saturation dives at the Japan Marine Science Technology Center (JAMSTEC), Yokosuka, Japan. The Institutional Committee on Human Experimentation of the JAMSTEC approved the study protocol, and all subjects gave their written consent to participate after being fully informed of the procedures and possible risks.

Dive Profile and Environmental Variables

The dive was carried out in a hyperbaric chamber (7.5 m long, 2.3 m in diameter) at JAMSTEC, Yokosuka, Ja-

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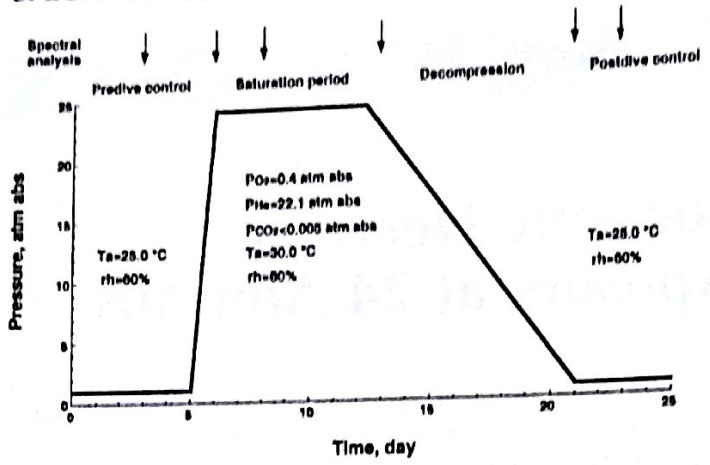

Fig. 1. Dive profile and chamber gas partial pressure. Arrows indicate where experiments were conducted. Ta: ambient temperature; rh: relative humidity.

pan. The experiment was undertaken from November 3 through November 27, 1995. Fig. 1 depicts the dive profile. During the predive and postdive periods, the subjects breathed normal air in the chamber, and the chamber temperature and relative humidity were kept constant at 25 ± 0.5°C and 60 ± 10%, respectively. The chamber pressure was raised to 24 atm abs at a constant rate of 3 atm abs per hour over 9 h, and the pressure was maintained at 24 atm abs for 7 d (saturation period). The total pressure of 24 atm abs consisted of 0.4 atm abs oxygen, 22.1 atm abs helium, less than 0.005 atm abs carbon dioxide, and the balance made up of nitrogen. During the 24 atm abs saturation dive, the chamber temperature and relative humidity were kept constant at 30 ± 0.5°C and at 60 ± 10%, respectively. The decompression procedure was made according to the standard U. S. Navy schedule (19). The decompression required 9 d to complete.

Measurements

Measurements were made on one predive day, 2 d at 24 atm abs (the first and third days in hyperbaria), 2 d during the decompression period [the first day (22.6 atm abs) and the ninth day (1.6 atm abs) during decompression period], and 1 d during postdive period (the second day during postdive period). On these days, measurements were carried out twice a day: once at 0700 h and again at 2230 h. The subject was rested supine for 20 min, R-R intervals through the standard II lead from electrocardiograph and respiratory rate, measured with a thermistor probe attached to the nostril, were measured during the last 10 min. The output signals of the electrocardiograph and thermistor were recorded on a data recorder (RD-111T, TEAC, Tokyo, Japan)

Analyses

Spectral analysis was performed by using a power spectral density analysis program of R-R interval variability (Vital Rhythm 98, NEC Medical Systems, Tokyo, Japan). A fast Fourier transform was used to calculate the power spectral density. The time series of R-R intervals were interpolated at 2 Hz by a Lagrange interpolation method. Consecutive data over 256 s were used for analysis. All analyses were performed between 0.03 Hz and 0.5 Hz. A frequency range between 0.03 Hz and 0.15 Hz represents the low frequency band, a range between 0.15 Hz and 0.5 Hz for the high frequency band, and a range between 0.03 and 0.5 Hz for the total frequency band.

Statistics

A two-way analysis of variance with repeated measures was used to test for two effects: pressures and time of the day. When significant F ratios were obtained, least significant differences were calculated for comparisons between means. The null hypothesis was rejected when $p < 0.05$. Data are expressed as means± SE.

RESULTS

Average HR was decreased ($p < 0.05$) only at the first night of exposure to 24 atm abs (Fig. 2). Morning HR was constant throughout the dive period. The HR rose significantly ($p < 0.05$) on the second day at postdive control.

Changes in the spectral density of cardiac variability over the experimental periods are shown in Fig. 3. Total and high-frequency power increased on the first day of exposure to 24 atm abs when HR decreased at night. Low-frequency power was constant throughout the experimental days. The ratio of low- to high-frequency power increased at the third morning during the saturation period, and increased again on the second day of postdive ($p < 0.05$). The ratio for the night time remained unchanged throughout the experimental days, except for a rise ($p < 0.05$) on the second day of postdive period.

Respiratory rates were 13.6 ± 1.4 and 18.1 ± 3.1 breath · min^{-1} in the morning and at night during predive period, respectively. The respiratory rate did not fluctuated significantly throughout the experimental period.

DISCUSSION

The major finding of the present study was that the high-frequency power was increased at the first night, which corresponded to a lowered HR. This suggests

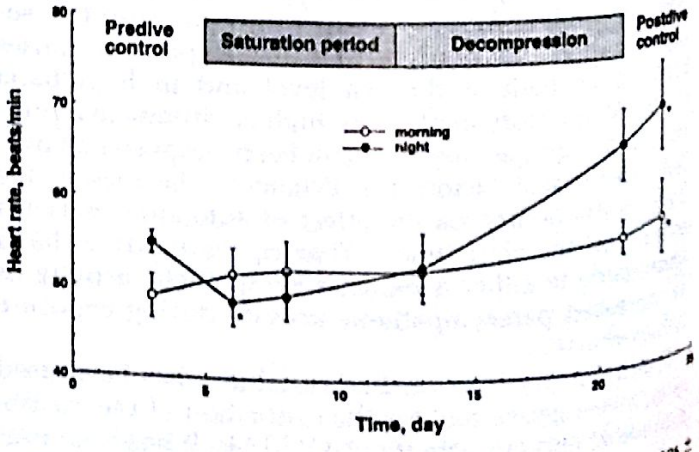

Fig. 2. Time course of HR changes during dive. Values are means ± SE; *$p < 0.05$ from predive value.

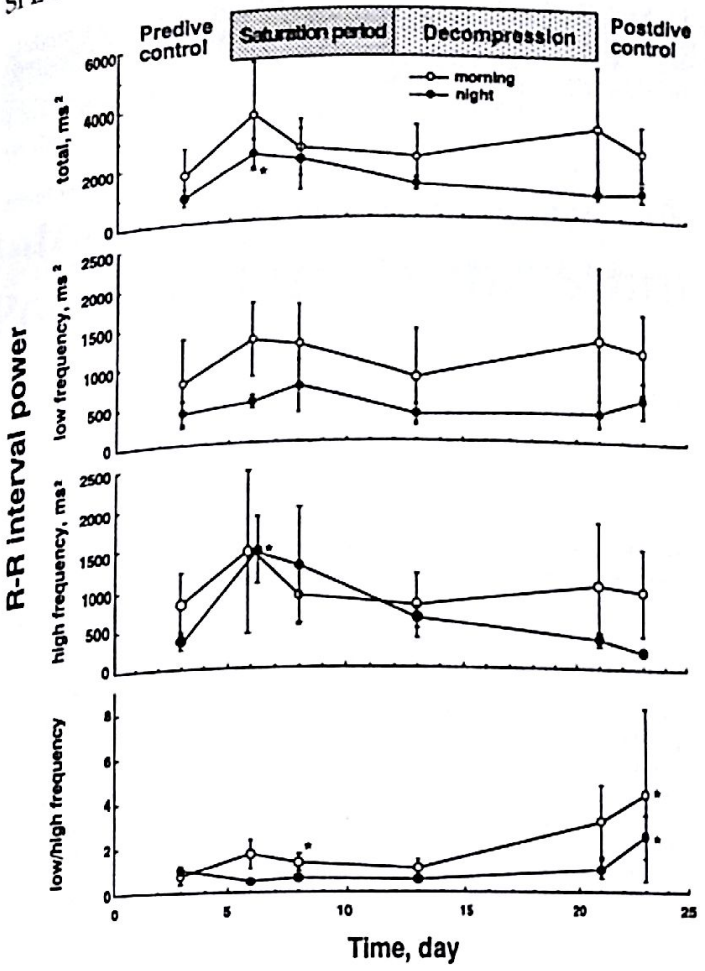

Fig. 3. Total, low-frequency, and high-frequency powers and ratio of low-to high-frequency power during dive. Values are means ± SE; *p < 0.05 from predive value.

that the bradycardia was related to an elevated vagal activity.

We failed to observe bradycardia in the morning measurements. Wada et al. (20) observed a profound bradycardia at night during a He-O_2 saturation dive at 16 atm abs. During predive period, the morning HR was already very low, 49.0 ± 2.4 bpm. It is possible, therefore, that we may not observe a further bradycardia at hyperbaria. Hayano et al. (6) performed hourly power spectral analysis of heart rate variabilities between 0700 and 2300 hours in supine resting men, and showed that vagal control of the heart was higher during the morning (0700–1200 hours) than during the late afternoon (1900–2300 hours). Our data also suggest a higher vagal control in the morning compared with that at night.

The bradycardiac response and the increased high-frequency power at the first night of the saturation period decreased gradually throughout the period of pressure exposure. These results suggest that a chronotropic adaptation occurred during prolonged exposure to a hyperbaric environment, and the adaptation was associated with the vagal traffic.

The mechanism of a slight but significant elevation of low- to high-frequency ratio in the third morning at 24 atm abs is unknown. It may indicate an increased cardiac sympathetic activity that causes the disappearance of hyperbaric bradycardia during the morning.

HR increased during postdive period in agreement with the similar experiments at 16 and 31 atm abs (10,20). The mechanism is not clear, however, after a prolonged confinement in the hyperbaric environment the tachycardia may be related to the cardiovascular deconditioning (3,10,11). Lin et al. (10) have observed a reduced ability of BP adjustment during a head-up tilt under a 31 atm abs environment and during postdive. They concluded that the cardiovascular deconditioning occurred and suggested that the hypovolemia resulted from a persisting diuresis and prolonged inactivity were responsible. Parenthetically, it is well recognized a sustained diuresis in a hyperbaric environment greater than 4 atm abs (8).

In conclusion, the decrease of HR was observed at night in the early stages of saturation period at 24 atm abs. The cause of the bradycardiac response may be related to an increased parasympathetic activity in hyperbaric environments.

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