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EFFECTS OF HYPERBARIC EXPOSURE ON THE CARDIOVASCULAR SYSTEM. ROLE OF THE AUTONOMOUS NERVOUS SYSTEM

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ABSTRACT

Introduction

Among experienced divers, dive adaptation is seen as a modified pattern of physiological changes. This is reflected, inter alia, in the change in cardiovascular responses, therefore there is need to examine the role of the autonomic nervous system in cardiovascular response modulation after hyperbaric exposure.

Material and methods

Ten experienced divers took part in the study. The effects of hyperbaric exposure at 30 and 60 meters and interaction (depth x time) were measured. Changes in HR, RRI, CI and HRV values have been taken into analysis.

Results

Hyperbaric exposure at 30 meters significantly affected HFnu-RRI elevation and decrease of LFnu-RRI ($F = 42.92$, $p < 0.00001$), without significant affecting the HR, RRI and CI. Exposure to hyperbaric 60 m increased HR and CI ($F = 7.64$, $p = 0.01$ and $F = 4.89$, $p = 0.04$ respectively) and RRI ($F = 7.69$, $p = 0.01$), without significant impact on other variables. The influence of interaction (depth x time) was significant in all measured variables.

Conclusions

The results indicate that hyperbaric exposure at 60 meters affected HR, RRI, CI parameters, that were not significantly affected by hyperbaric exposure at 30 meters. On the other hand, the exposure at 30 meters showed a significant effect on the LFnu and HFnu HRV, which were not significantly affected by the exposure at 60 meters. Significant effect of time and depth interaction in each of the analyzed variables was observed.

Keywords: autonomic nervous system, hyperbaric chamber, diving.

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INTRODUCTION

In a healthy body, physiological systems can adapt their functions relative to changes in the external environment. Thus regular performance of a given activity, may lead to modified physiological system responses during the activity. For example, Hong et al. [1] found that in female divers from Korea, called "*haenyeo*", the average diver's heart rate (HR) dropped from 101 (a value at rest) to 60 (40 seconds after immersion) beats per minute. Interestingly, a study from 2015 [2] noted that the HR value during diving was lower in older women versus young women. Nevertheless, a nearly 20% reduction in HR was noted for the entire group studied during breath-hold diving in the sea [2].

The observed changes in HR values may result from various reactions. For instance, intense vasoconstriction of peripheral and visceral capillary beds and a reduction in oxygen consumption might reserve oxygen for priority organs such as the brain or heart [3]. Subjects with 7–10 years of experience diving showed lower levels of blood acidity and oxidative stress in response to diving while holding their breath [4]. Test results of a group of 30 healthy men showed that significant changes in HR did not occur when breath holding under normal conditions [5].

A significant reduction in HR values was observed when diving was simulated by immersing in cold water with breath held: HR values dropped from 108.97 to 81.77 during first 15 seconds, and decreased even further during the subsequent 15 seconds [5]. Furthermore, results from a study using a similar method to simulate diving by immersion in cold water [6] demonstrated that an initial increase in HR occurs during exposure. However, the physiological reaction to diving is not the same when a diver's breath is not held during the activity [7].

In a study of 25 divers, during diving, an interesting autonomic nervous system (AUN) reaction to the diving and immersion was observed [8]. Diving activated both the AUN sympathetic and parasympathetic components, but additionally increased all studied parameters relating to heart rate variability (HRV).

Considering the above, a study of the diving reflex is of interest, specifically analysing the reaction of the AUN and the cardiovascular system during exposure to hyperbaric conditions where the subjects can breathe freely. On the basis of previous reports, the authors assume that the AUN and cardiovascular system reactions will be similar to standard diving in cold water, even though in this study the exposure will be limited to exposure in a hyperbaric chamber, where the compression level will correspond to pressure at a relevant depth during diving in a water reservoir.

MATERIALS AND METHODS

Ten male subjects, with diving experience (from 2 to 12 years) participated in the study. The anthropometric data of the study participants is shown in Table No. 1. The subjects underwent exposure to the hyperbaric chamber twice. The first exposure lasted from 12:00 p.m. to 1:00 p.m. Volunteers were placed in the hyperbaric chamber and exposed to compression of 400 kPa. The exposure plateau was ca. 30 minutes, and it was followed by a gradual decompression. The decompression pattern, following exposure to 400 kPa (0.4 MPa–0.3 MPa + 0.1 MPa atmospheric pressure), was the same pattern as used after diving at 33 meters, which corresponds to the pressure of 440 kPa. The breathing gas used during interventions in the hyperbaric chamber was normal air [7].

During the second exposure, at a depth of 60 meters, the compression time was 4 hours. The entire experiment lasted from 1:30 p.m. to 6:30 p.m., with a decompression time of 4 hours.

AUN function was analysed with the Task Force Monitor system, a device used for non-invasive studies of the cardiovascular system and functional evaluation of AUN. The system consists of the following subunits: a device for continuous blood pressure monitoring, an electrocardiograph (ECG), an impedance cardiograph (IKG), a device for oscillometric pressure measurements, and a pulse oximeter.

The specific method is described in detail in another report [8]. The subjects were examined four times during one day: immediately before and after exposure to hyperbaric conditions at 30 meters, and before and after exposure to hyperbaric conditions at 60 meters. During the statistical analysis, values of the following factors were taken into account: HR, RR interval (RRI), and the cardiac index (CI) relating the cardiac output to body surface area. Results of the HRV spectral analysis, the low frequency (LF) and high frequency (HF) of power spectral density, were also verified. The LF (0.04-0.15 Hz) and HF (0.15-0.4 Hz) components for HRV were presented in the standardised units (LFnu RRI, HFnu RRI).

Tab. 1

Subjects anthropometric data.

i.d.	Age	Height [cm]	Weight	BMI	Years of diving experience
DIVE_001	29	190	92	25	4
DIVE_002	29	180	72	22	9
DIVE_003	31	178	75	24	2
DIVE_004	22	168	76	27	4
DIVE_005	33	191	94	26	8
DIVE_006	20	193	96	26	2
DIVE_007	37	183	80	24	12
DIVE_008	32	174	86	28	4
DIVE_009	25	176	86	28	5
DIVE_010	36	172	80	27	11
Mean	29	2	84	26	6

Data was analysed with the statistical package STATISTICA 13.1 (StatSoft, Tulsa, OK). The Shapiro-Wilk test was used to analyse distribution normality for values of analysed variables. The equality of variances was verified with Levene's test. The separate effects hyperbaric exposures at 30 meters and 60 meters (before/after) was analysed with one-way ANOVA, and the interaction effect (depth x before/after) was verified with two-way ANOVA.

RESULTS

Analysis of the results showed that the HR value tended to decrease slightly following exposure to hyperbaric conditions at 30 meters, however, that tendency was not statistically significant ($p > 0.05$). Exposure to 60 meters, on the other hand, caused a reverse effect, and an observed HR increase was statistically significant ($F = 7.64$, $p = 0.01$). The interaction between depth and time factors, shown in Fig. No. 1, also proved to be statistically significant ($F = 9.71$, $p = 0.004$).

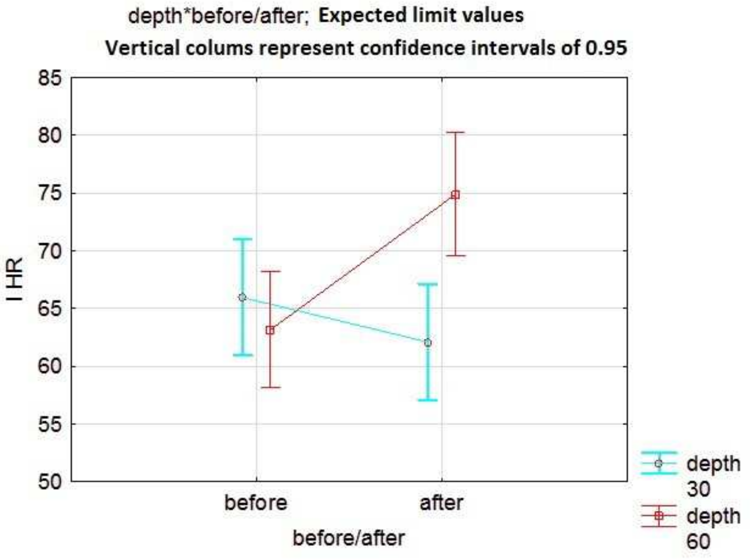

Fig. 1 Effect of depth and time on HR value.

No significant changes in RRI were observed in response to exposure to hyperbaric conditions at 30 meters ($p > 0.05$). During exposure to 60 meters, on the other hand, a statistically significant effect of the exposure on RRI was observed, in a direction opposite to that observed during exposure at 30 meters ($F = 7.69$, $p = 0.01$). For RRI values, the interaction between depth and time factors proved to be significant ($F = 10.07$, $p = 0.003$) (Fig. No. 2).

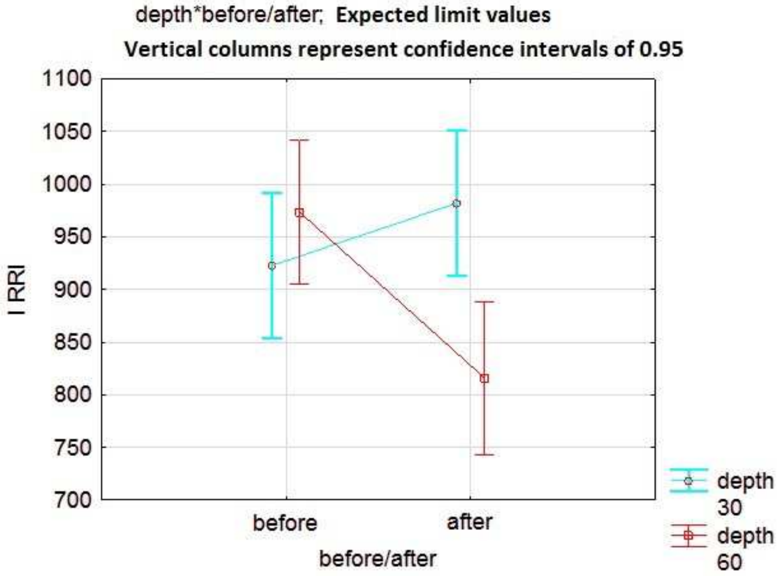

Fig. 2 Effect of depth and time on LFnu-RRI.

No significant changes in CI were observed in response to exposure to hyperbaric conditions at 30 meters ($p > 0.05$). On the other hand, a significant increase in the CI value was observed following exposure to 60 meters ($F = 4.89$, $p = 0.04$). A two-way ANOVA showed a significant interaction ($F = 6.9$, $p = 0.01$) (Fig. No. 3).

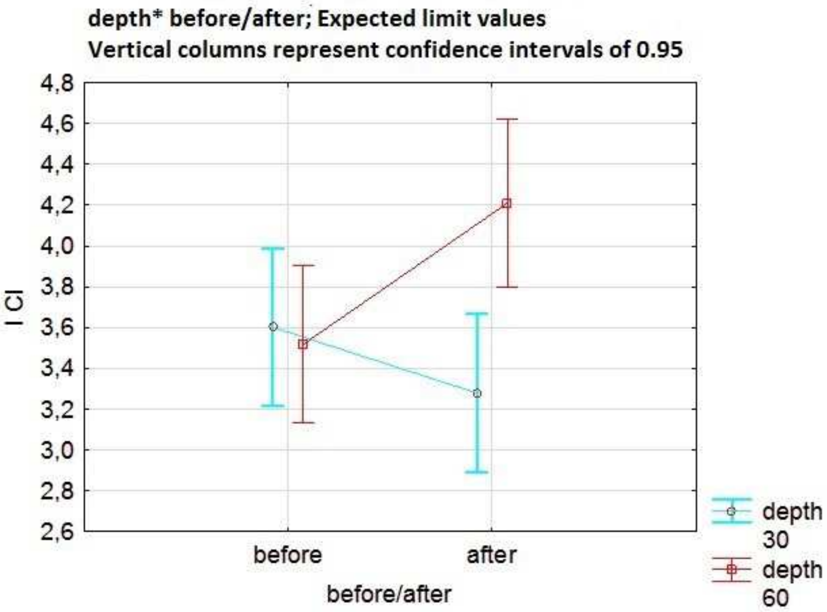

Fig. 3 Effect of depth and time on CI value.

Changes in LFnu-RRI values are shown in Chart No. 4. This value decreased following exposure both to 30 meters and 60 meters, where a change in this value following exposure to 30 meters was statistically significant (F = 42.92, $p < 0.00001$). Following exposure to 60 meters, this change did not reach statistical significance ($p > 0.05$). The effect of interaction between two factors (depth x time) on LFnu-RRI proved to be statistically significant: (F = 4.17 $p < 0.05$).

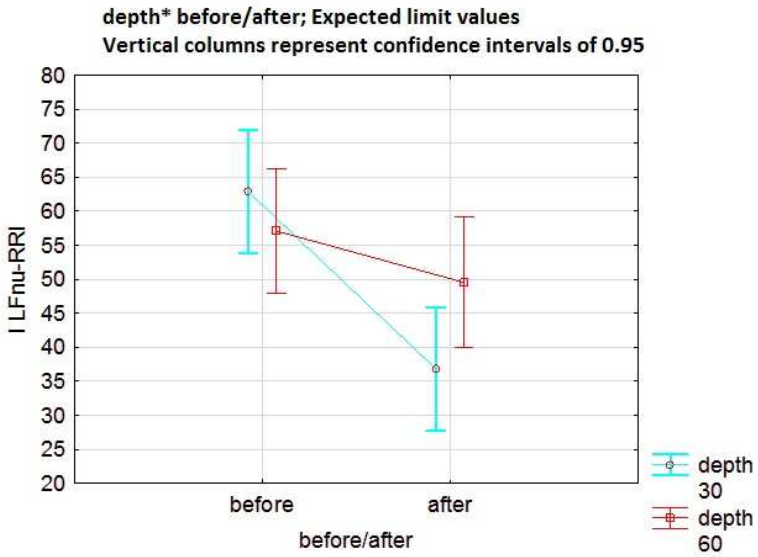

Fig. 4 Effect of depth and time on LFnu-RRI.

The HFnu-RRI value decreased following exposure both to 30 meters and 60 meters, where a change in this value following exposure to 30 meters was statistically significant (F = 42.92, $p < 0.00001$). A change in the HFnu-RRI following exposure to 60 meters did not reach statistical significance ($p > 0.05$). A two-way ANOVA showed a significant interaction: (F = 4.17 $p < 0.05$) (Fig. No. 5).

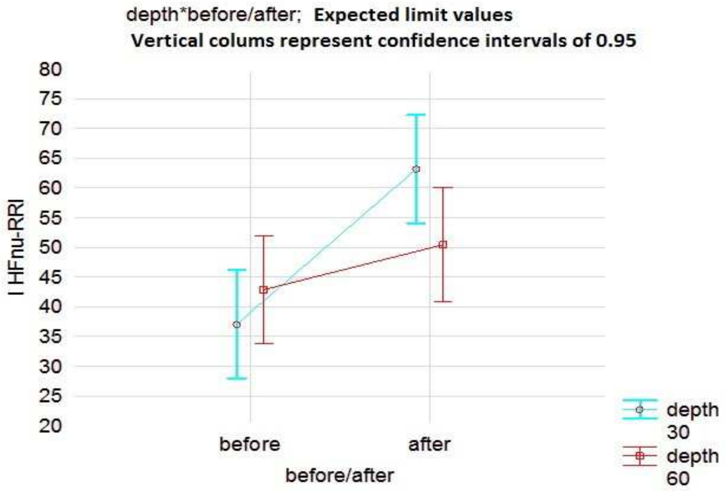

Fig. 5 Effect of depth and time on HFnu-RRI.

The dynamics of changes in mean values before and after exposure to 30 meters and 60 meters are shown in Tables No. 2 and No. 3, respectively. Variables for which statistically significant changes were observed are marked with an asterisk (*).

Tab. 2

Changes in mean values of measured variables before and after exposure to 30 meters.

Variable	Mean Before	Mean After
I HR	65,96	62,10
I RRI	922,60	981,84
I CI	3,60	3,28
I LFnu-RRI *	76,26	73,53
I HFnu-RRI *	23,74	26,47

Tab. 3

Changes in mean values of measured variables before and after exposure to 60 meters.

Variable	Mean Before	Mean After
I HR *	63,165	74,897
I RRI *	973,323	816,062
I CI *	3,519	4,210
I LFnu-RRI	57,107	48,717
I HFnu-RRI	42,893	51,283

DISCUSSION

Following exposure to pressure equivalent to 30 meters of depth, a non-significant tendency toward reduction in HR and CI values was observed, accompanied by a simultaneous increase in RRI. Interestingly, upon exposure to hyperbaric conditions equivalent to 60 meters, we observed a significant influence of intervention. The changes at 60 meters were in the reverse direction of changes observed during exposure to 30 meters: HR and CI values increased, while RRI dropped. Considering these three parameters, it might be concluded that exposure to the hyperbaric conditions at 30 meters activated the parasympathetic component of the AUN.

This would be confirmed by a significant reduction in the low frequency component of RRI (LFnu-RRI) with a simultaneous increase in HFnu-RRI. The HFnu-RRI value may reflect the intensity of the efferent activity of the vagus nerve [9], such that the pattern of all observed changes would indicate an increased parasympathetic component following exposure to pressures equivalent to 30 meters of depth. Upon exposure to 60 meters, significant changes in HR, RRI, and CI were observed, and in the reverse direction of changes observed at 30 meters. Parameters related to the low and high frequency of sinus rhythm tended to change in the same direction as during exposure to 30 meters, however this effect was not statistically significant.

Based on the current data, it is difficult to say which AUN component was more active following exposure to hyperbaric conditions at 60 meters; it is possible that activity of both components increased, similar to observations in other studies [10]. Higher levels of compression could result in a stronger venous return, which may activate atrial B-type stretch receptors that activate the sympathetic and parasympathetic systems via a reflex path. A strong venous return leads to increased atrial stretch and activation of the Bainbridge reflex, causing tachycardia.

In this study, values of biochemical parameters were not taken into account, which could also have a significant effect on modulation of the cardiovascular system parameters, especially considering the fact that the exposure to hyperbaric conditions at 60 meters lasted 4 hours in total. An additional limitation of this study could be that the currently obtained data more precisely describe changes in AUN function immediately before and after the exposure, rather than during the exposure to hyperbaric conditions.

CONCLUSIONS

The exposure to hyperbaric conditions at 30 meters resulted in increased intracardiac activity of the parasympathetic system. Exposure to hyperbaric conditions at 60 meters modified both sympathetic and parasympathetic activity. This AUN activity influenced the cardiovascular function, activating the Bainbridge reflex receptors.

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