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STUDIES OF BODY WEIGHT/HEART WEIGHT [C/S] RATIO IN TREATED AND UNTREATED EXPERIMENTAL PULMONARY BAROTRAUMA

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ABSTRACT

The prerequisite of development of pulmonary barotrauma [PB] is retention of the breathing mix in the lungs during a sudden decrease in external pressure or its administration into the airways under increased pressure or in a volume exceeding the maximum lung capacity. In such cases, the pulmonary parenchyma ruptures and air enters both the pleural cavity and/or the lumen of ruptured blood vessels located in the alveolar septa. The result is permanent disruption of the pulmonary parenchyma.
The aim of the study was to assess the influence of post-PB lesions on the heart muscle and the importance of hyperbaric treatment on the exacerbation of such lesions in the heart. The hearts of 35 rabbits were used in the study. In animals of the experimental group, PB was induced in the pressure chamber using the proprietary method described in previous publications. Part of the animals in this group were treated with air hyperbaria. The comparison group consisted of animals, which did not undergo PB during a simulated dive. All animals were weighed, observed for four weeks and then put to death following the experiment. In autopsy, among others, whole hearts were collected and weighed after fixation. Subsequently, the C/S ratio, i.e. the body to heart weight ratio, was calculated. The measurement results were subject to statistical analysis. A statistically significant increase in the C/S ratio was found, indicating an increase in the share of heart weight in the total body weight in the group of animals with PB not treated with air hyperbaria as compared to the control group.
Keywords: pulmonary barotrauma, C/S ratio, heart mass, hyperbaric treatment.

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INTRODUCTION

Pulmonary barotrauma is any damage to the pulmonary parenchyma caused by sudden changes in the volume of the breathing mix contained in it. Morphologically, its exponent is the image of acute pulmonary artery distension with perforation of alveolar walls and vessels running through them [1,2,3,4].

The direct (acute) consequences of PB are arterial gas embolism (AGE) and pneumothorax, mediastinum and subcutaneous pneumothorax. Late effects of PB, on the other hand, are changes in the pulmonary parenchyma in the form of fibrosis, scarring and emphysema (distentions) [4].

The only effective way of treating arterial air congestion is recompression and therapeutic decompression. Such a procedure ensures, on the one hand, reduction of the diameter of the intravascular gas bubbles forming an embolism, which allows them to pass through the bed of capillaries, and on the other hand, thanks to breathing in an atmosphere with increased oxygen partial pressure, it improves oxygenation of tissues ischemic due to embolism [5].

In the available literature, studies on distant organ lesions occurring after the past, often asymptomatic, PB in humans are extremely rare. Moreover, the authors found only a few papers describing changes in heart configuration. These were either catamnestic studies or studies on closed professional populations [6,7].

A considerably more numerous group of works on cardiological problems of divers are descriptions of changes in heart function during a dive and the influence of heart and cardiovascular diseases on diving safety [8,9,10].

The available literature does not contain any experimental papers on the study of changes in the heart, for which the morphology and function of the pulmonary parenchyma reconstruction is certainly not without significance. This encouraged us to undertake research using our own experimental model [11]. The present publication is yet another in the cycle of papers presenting the results of our research [12,13,14].

OBJECTIVE

The aim of the study was to examine the influence of lesions in the lungs after PB on the C/S ratio and the impact of hyperbaric treatment for the development of such lesions.

MATERIAL AND METHOD

The hearts of 35 rabbits of both genders, aged 28 to 34 weeks, were used in the study. A detailed description of the experimental model developed by one of the authors of this study was published in earlier reports [11]. The animals were divided into groups:

- control group (K) consisting of 14 animals,
- experimental group (O) consisting of 12 animals,
- treated experimental group (OL) consisting of 9 animals.

The animals from experimental groups (O and OL) were compressed to 200 kP in a hyperbaric chamber for small animals. At the peak of inhalation, their airways were obstructed and decompressed to atmospheric pressure, reproducing the mechanism of formation of PB. The OL group animals were then treated with air hyperbaric therapy. The air table "III" modified for this study was used, [15], which is intended to treat cases of arterial gas embolism following PB.

In the animals included in the control of the procedure (group K), the compression was performed to a pressure of 200 kP and was followed by decompression without obstructing the airways.

Following the experiment, all animals were weighed and observed for four weeks and then put to death. After killing, organ packets containing, inter alia, whole hearts were collected for examination. The packets were fixed in 10% buffered formalin solution. After fixing, the hearts of the animals were weighed using an analytical balance.

The results obtained from the measurements of body and heart weight of individual animals are presented in the tables below. Averages and median body and heart weights of animals in particular groups were calculated. C/S ratios in particular groups have also been calculated. The values of the C/S ratio were compared between the experimental groups and the results of the measurements were statistically analysed using ANOVA post-hoc NIR test.

RESULTS

When comparing the changes in the C/S index (i.e. the ratio between average body weight and average heart weight) in individual groups, a significant increase in heart weight was found in group O as compared to OL and K groups. The studies concerning exclusively absolute heart mass changes in individual groups were similar, and detailed results can be found in an earlier publication [13].

The average heart weights compared between the experimental groups differed in a similar way; group O showed higher values than groups K and OL (Table 1).

Tab. 1

The comparison of average values.

Variable	Group	Number of animals	Median	Stand. deviation
Specimen Weight			3755.833	790.552
Heart Weight	O	12	8.542	1.912
Specimen Weight/Heart Weight			450.234	100.829
Specimen Weight			3884.444	695.667
Heart Weight	OL	9	7.244	0.994
Specimen Weight/Heart Weight			539.320	93.2216
Specimen Weight			3621.429	689.6810
Heart Weight	K	14	6.507	1.098
Specimen Weight/Heart Weight			559.228	72.541

Similar correlations occurred when comparing the heart mass median of the animals tested (Tab. 2).

Tab. 2

Median comparison.

Variable	Group	Number of animals	Median	Stand. deviation
Specimen Weight			4200.000	790.55217
Heart Weight	O	12	8.700	1.912
Specimen Weight/Heart Weight			450.597	100.829
Specimen Weight			3900.000	695.667
Heart Weight	OL	9	7.200	0.994
Specimen Weight/Heart Weight			525.000	93.221
Specimen Weight			3625.000	689.681
Heart Weight	K	14	6.600	1.098
Specimen Weight/Heart Weight			570.378	72.541

Tab. 3

The results of statistical analysis of comparisons of C/S ratio values for particular groups.

	NIR Test; Provided differences are statistically significant with p < .05000		
Group	O M=450.23	OL M=539.32	K M=559.23
O		**0.028969**	**0.003659**
OL	**0.028969**		0.601534
K	**0.003659**	0.601534	

Statistical analysis showed that at the level of significance p=0.029 there is a statistically significant difference for the mean value of C/S ratio between O and OL groups. This coefficient for the experimental group (O) is 450.597±100.829 and is statistically significantly lower compared to the experimental group subjected to treatment (OL) (525.000±93.220).

At the level of significance p=0.037 there is a statistically significant difference for the mean value of C/S ratio between O and K groups. For the experimental group (O) this ratio amounts to 450.597±100.829 and is statistically significantly lower compared to the control group (K) (570.378±72.540).

However, no statistically significant (p=0.6) difference was observed for mean C/S values between treated (OL) and control (K) experimental groups.

DISCUSSION OF RESULTS

On the basis of both the currently obtained and previously published experimental results concerning changes in heart configuration under the influence of a history of PB, it was found that all the parameters studied in the experimental groups concerning the heart mass were changed in relation to the control groups. It is worth emphasising here that the exponents of heart enlargement and hypertrophy observed in these cases coincide with literature data concerning pathological changes observed after PB in humans [6,16,17,18].

Microscopic examination [19] also revealed quantitative changes with regard to the stromal-to-muscle cell ratio in favour of an increase in the percentage of connective tissue in the wall of both heart chambers following pulmonary barotrauma. These changes were

accompanied by myocardial hypertrophy.

This explains the increase in the proportion of heart weight in the total weight of the animal compared to the same ratio in the control group animals found in these studies.

Earlier studies also showed that the use of hyperbaric treatment after PB reduces the severity of lesions in the heart. Similar conclusions are drawn from the current study on C/S ratio comparison. These studies have also shown that hyperbaric treatment has inhibited the increase of heart mass (i.e. reduction of C/S value). This is in line with the results of previous studies [13,14,20], which demonstrated that the use of hyperbaric treatment significantly prevents retrograde changes in the pulmonary parenchyma following PB.

Both current and previous studies have shown that the use of presently rarely utilised therapeutic air decompression tables is completely sufficient to inhibit the development of lesions in the heart. It can be assumed that the use of therapeutic oxygen tables such as US Navy Table [21a] 6A and mix-based or sub-saturated tables [21b] would give an even better therapeutic effect. Unfortunately, the experimental model used did not allow the application of treatment in an oxygen atmosphere. Lack of the possibility for the experimental animals to inhale through a mask would necessitate filling the whole chamber with oxygen, which was not possible for safety reasons. The second possibility was to ventilate the animal through an intubation tube, which would have required long-term anaesthesia. At the same time, such an action could significantly affect the lung morphology and other parameters studied. For this reason, only therapeutic air tables were used.

CONCLUSIONS

- Pulmonary barotrauma affects the weight of the heart and leads to its growth.
- Application of hyperbaric treatment significantly reduces the severity of lesions.

Statistical analyses carried out using the Statistica 13 package.

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