EFFECT OF DIFFERENT PHYSICAL EXERCISE PROGRAMS ON HYPERTROPHY OF THE HEART AND ITS COMPARTMENTS

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UDC 616.12-007.61-02:
616.12-008.615]-092.9

KEY WORDS: heart; hypertrophy; physical exercise.

An increase in weight of the heart is a regular response to a prolonged increase in its contractile function and, in particular, the systematic performance of physical exercise. This was demonstrated experimentally as long ago as the beginning of this century [12]. The problem of the effect of physical exercise on the dimensions of the heart and on changes in the microscopic structure of the myocardium has been studied in fair detail [2, 4, 7]. Nevertheless, we could find virtually no information on relative changes in the weight of the atria under these circumstances in the accessible literature. There are also few data on the effect of excessive (exhausting) exercise on the weight of the heart.

The aim of this investigation was to study the effect of different programs of physical exercise on hypertrophy of the heart and of relative changes in weight of the atria.

EXPERIMENTAL METHOD

Two series of experiments were carried out on male albino rats. In series I, rats weighing initially about 184 g on average were divided into three similar (by weight) groups with 15 animals in each group; the rats in one (control) group were not compelled to do physical exercise. Rats of the first experimental group did moderate exercise, namely, swimming in warm water (30-33°C) for 60 min 5 times a week for 3 months. Animals of the second experimental group swam on the same days and at the same times, but the duration of each swimming session was increased to 240 min (long-term exercise). During the first month animals of both experimental groups swam without carrying a load, during the second month they carried a load equal to 2.5% of body weight, and in the third month they carried a load of 5%.

Experiments of series II were carried out on rats with an initial mean weight of 162 g, and these animals also were divided into three groups (with 7 in each group): one control and two experimental groups (3 and 4). Animals of the experimental groups did physical exercise of the same kind, which was repeated with the same frequency and for the same duration as in group 2, but with greater intensity, for ten weeks: After two weeks of swimming without a load, its weight was increased by 2.5% every consecutive two weeks, and by the 7th week it amounted to 7.5% of body weight. The load carried in the experiments of group 3 remained at this level until the end (intensive exercise), but in group 4, starting with the 11th week, it was equal to 10% of body weight, and the duration of swimming was simultaneously increased to 4-5 h daily (exhausting exercise). Under these circumstances the animals lost a considerable amount of their body weight (more than 10%) and their working capacity also was reduced [8].

At the end of three months from the beginning of the experiment the rats were decapitated, the heart was removed, dried with filter paper, and freed from remains of the great vessels, after which the atria were carefully separated from the ventricles with small scissors and each compartment was weighed on analytical scales with an accuracy of tenths of a milligram. The degree of hypertrophy of the whole heart and of its compartments in the experiments in the experimental groups was estimated by comparison with the corresponding series of control values.

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(Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 102, No. 11, pp. 602-604, November, 1986. Original article submitted March 10, 1986.

 0007-4888/86/0011-1588$12.50

TABLE 1. Changes in Weight of the Heart and Its Compartments during Various Programs of Physical Exercise

Compartment of the heart	Weight of compartment of heart, mg					
	series I			series II		
	control	group 1 (moderate exercise)	group 2 (long-term exercise)	control	group 3 (intensive exercise)	group 4 (exhausting exercise)
Whole heart (mg)	846,2±27,6	922,9±36,5 (109,1)	998,0±36,8 (117,9)**	809.3±15,4	954,0±44,5 (117,9)*	869.6±44,9 (107,4)
Relative weight of heart, mg/kg	3,14±0,08	3,56±0,14 (113,4)*	4,10±0,08 (130,6)***	3,48±0,08	4,21±0,12 (120,9)	4,41±0,18 (126,7)***
Atria: mg	78,7±5,1	101,6±4,6 (129,1)**	111,9±5,5 (142,2)***	72,2±5,1	98,3±6,3 (136,1)**	92,0±6,1 (127,4)*
% of whole heart	9,26±0,32	10,99±0,35 (118,7)**	11,44±0,27 (132,5)***	8,90±0,60	10,60±0,55 (119,1)*	10,61±0,61 (119,1)
Ventricles	767,5±25,4	821,3±31,9 (107,0)	886,1±32,9 (115,5)*	737,1±14,6	855,7±40,4 (116,1)*	777,5±40,8 (105,5)

Note. Numbers in parentheses denote % of control. *P < 0.05, **P < 0.01, ***P < 0.001 compared with corresponding control.

EXPERIMENTAL RESULTS

With all the physical exercise programs hypertrophy of the myocardium was found, but its intensity, as might be expected, was not the same in all experimental groups (Table 1). In the rats of group 2, which did moderate exercise, the weight of the heart was only 9.1% greater than in the control rats (P > 0.05). However, because of the slower rate of rise of the body weight (by the end of the experiments it was 260.3 ± 15.1 g compared with 270.7 ± 10.6 g in the control) the relative weight of the heart in the rats of this group was significantly higher than in the control (P < 0.05). The increase in absolute weight of the heart after long-term and intensive physical exercise (groups 2 and 3) amounted to 17.9% (P < 0.05-0.02) and the relative weight increased by 30.6 and 20.9%, respectively (P < 0.001). However, the sudden change from intensive exercise to excessive or exhausting exercise (group 4) caused no further increase in weight of the heart. Indeed, in this case there was a tendency for its weight to decrease, and the difference from the control was only +7.4% (P > 0.05). This can evidently be explained by at least two circumstances. First, the program of exhausting exercise was preceded by a fairly long period of adaptation to nonexcessive exercise, and this causes distinct limitation of cardiac hypertrophy during subsequent hyperfunction at a higher level [4]. In agreement with this rule, the degree of hypertrophy of the heart under the influence of exhausting exercise was considerably higher in previously unadapted animals that in the present experiments. Second, because of disturbances of the smoothness of increase in the work load a situation arose when the frequency and strength of the load on the myocardium exceeded the customary rhythm of renewal of its structures and gave rise to uncompensated wear and tear, with reduction of the reserves of structural materials of the cells. The body weight under these circumstances fell more than the weight of the heart, and the ratio of the latter to body weight was higher than during the preceding program.

Incidentally, during long-term adaptation to physical exercise the atria and ventricles developed different degrees of hypertrophy. In all the exercise programs used the weight of the atria increased more (by 27.4-42.2%, P < 0.02-0.001) than the weight of the ventricles (by 7.0-16.1%, P > 0.05-<0.05). The weight of the atria relative to the weight of the heart in the experimental group averaged 10.60-11.44%, which is significantly greater (P < 0.05-0.001) than the control (8.90-0.26%). The decrease in weight of the heart taking place under the influence of exhausting exercise, on the other hand, affected the myocardium of the ventricles rather more (—9.1%) than that of the atria (—6.4%), and the relative weight of the atria under these circumstances was not reduced.

Consequently, not only did the atria undergo more intensive hypertrophy than the ventricles during adaptive loads, but they also maintained their increased weight more enduringly during exhausting exercise.

The greater degree of hypertrophy of the atria was evidently largely due to the fact that they are more sensitive to various influences [7]. In particular, during moderate physical exercise changes in the mitochondria of the muscle cells of the atria and auricles were very obvious, whereas in the left ventricle they were still inconsiderable. The increased sensitivity of the atria to physical exercise is evidently linked with the lower degree of differentiation of their myocytes compared with those of the ventricles toward contractile function [6], as is shown by the fact that they contain fewer myofibrils [14, 16],

most myocytes have no channels of the T system [5, 10, 11], and the sarcoplasmic reticulum is poorly developed [9]. These features of the atrial myocytes are evidently responsible for the fact that during general hyperfunction of the heart caused by muscular work the atria are subjected to a greater additional load, and in turn, this leads to a higher intensity of functioning of structures of the working myocytes and to greater probability of their hypertrophy than in the ventricles. Predominant hypertrophy of the atria during long-term adaptation to physical exercise may perhaps also be facilitated by the fact that the atrial cardiomyocytes differ fundamentally.

LITERATURE CITED

1. A. P. Amvros'ev, A. P. Kirilyuk, and L. M. Lobanok, Neurohumoral Regulation and Motor Activity during Aging [in Russian], Minsk (1984).
2. L. Komadel, E. Barta, and M. Kokavec, Physiological Enlargement of the Heart [in Russian], Britislav (1968).
3. P. L. Kyrge, "Cationic exchange of the myocardium and its hormonal regulation during exhausting physical exercise and training," Author's Abstract of Dissertation for the Degree of Doctor of Medical Sciences, Tallin (1975).
4. F. Z. Meerson, The General Mechanism of Adaptation and Prophylaxis [in Russian], Moscow (1973).
5. P. P. Rumyantsev, Arkh. Anat., No. 6, 115 (1972).
6. P. P. Rumyantsev, Cardiomyocytes during Reproduction, Differentiation, and Regeneration [in Russian], Leningrad (1982).
7. D. S. Sarkisov and B. V. Vtyurin, Electron-Microscopic Analysis of Increased Endurance of the Heart [in Russian], Moscow (1969).
8. A. S. Chinkin, Fiziol. Zh. SSSR, 68, No. 7, 1016 (1977).
9. C. E. Challice and W. G. Tatton, Electron Microscopy 1964, Vol. B, Prague (1964), pp. 101-102.
10. R. G. Hibbs and V. J. Ferrans, Am. J. Anat., 124, 251 (1969).
11. R. Hirakow, Am. J. Cardiol., 25, 195 (1970).
12. N. Külbs, Arch. Exp. Path. Pharm., 4, 228 (1906).
13. J. R. Marie, H. Guillemot, and P. V. Hatt, Pathol.-Biol., 24, 548 (1976).
14. E. Page, J. Earley, and B. Power, Circulat. Res., 34-35, Suppl. 2, II12 (1974).
15. N. E. Pratt, Anat. Rec., 157, 303A (1967).
16. R. Zak, Circulat. Res., 34-35, Suppl. 2, II17 (1974).

EFFECT OF CONDITIONS OF HYPERBARIC OXYGENATION ON STRUCTURAL CHANGES INDUCED IN THE LUNGS

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UDC 615.835.12.015.44:616.24-091.8

KEY WORDS: hyperbaric oxygenation; lungs; electron microscopy.

Hyperbaric oxygenation (HBO) is being increasingly widely used in clinical practice in various diseases. The wide range of application of HBO, on account of the universal character of hypoxia as a typical pathological process, and the specific features of this therapeutic technique are such that hyperbaric medicine can be regarded as a new trend in medical science [1]. Considering the possible toxic action of oxygen, the further spread of this method necessitates a detailed study of the effect of HBO on vitally important organs. The The most interesting aspect is the study of the action of HBO on the tissues of the lungs, which by virtue of their specific functions as organs of gas exchange, and of the method of oxygenation generally used, are exposed to a relatively higher partial pressure of oxygen under an increased total pressure. This has led some workers [3] to regard the lungs as a

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(Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 102, No. 11, pp. 604-606, November, 1986. Original article submitted January 13, 1986.

0007-4888/86/0011-1590$12.50