CHARACTER OF CHANGES IN THE MYOCARDIUM OF ALBINO RATS PRODUCED BY PHYSICAL EXERTION

V. M. Pinchuk, L. I. Levina,
and V. N. Popov

UDC 612.172-06:612.766.1

The result of exposure to physical exertion for the same total period of time depends on the rhythm of its distribution. For instance, animals of both sexes adapt themselves readily to regularly repeated swimming for comparatively short periods of time and separated by an interval of rest lasting 2-3 days. Less prolonged but daily periods of swimming lead to considerable changes, detectable in the ECG, of the moderately hypertrophied myocardium in one-quarter of the experimental animals (males, as a rule), and the concentration of sodium ions in the heart muscle is increased.

Changes in the heart associated with acute or chronic straining of its activity under the influence of physical exertion have received increasing attention in recent years [1, 3-7].

Changes in the heart muscle produced by different training programs were studied.

EXPERIMENTAL METHOD

Noninbred albino rats (33 males and 26 females) weighing 206±30 and 185±25 g, respectively, were used in the experiments. The control consisted of 14 animals (7 males and 7 females). The experimental rats were divided into two groups. The animals of group 1 (15 males and 7 females) were made to swim six times a week in water at a temperature of 30-32°C, at the beginning of the experiment for 5 min, and every two weeks the time of swimming was increased by 5 min, so that at the end of the experiment the males were swimming for 35 min and the females for 30 min. The animals of group 2 (11 males and 12 females) were made to swim under the same conditions but only twice a week. At first the duration of swimming was 15 min, and every two weeks it was increased by 15 min so that at the end of the experiment the males were swimming for 105 min and the females for 90 min. The total time of swimming for the males of both groups was 35 h and for the females 21 h. The animals' behavior was recorded during and after swimming throughout the experiment, and the ECG was recorded in seven leads (three standard, three amplified leads from the limbs, one thoracic) before the beginning and at the end of the experiment, after injection of pentobarbital (0.1 mg/g).

The males were killed by decapitation after 30 and 112 days and the females after 84 days. The heart was weighed, after which the concentration of Na^+ and K^+ ions were determined separately in the ventricles by flame photometry (using the Zeiss III photometer) and the potassium/sodium ratio was calculated. Pieces of heart tissue were fixed in 12% formalin solution and in Zenker's and Carnoy's fluids for microscopic investigation. Sections were stained with hematoxylin–eosin, by Van Gieson's method, with Heidenhain's iron–hematoxylin, and by Brachet's method and examined in polarized light.

Department of Anatomy and Laboratory of Functional Diagnosis, P. F. Lesgaft Institute of Physical Culture, Leningrad. (Presented by Academician of the Academy of Medical Sciences of the USSR I. A. Strukov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 75, No. 5, pp. 18-20, May, 1973. Original article submitted July 31, 1972.

TABLE 1. Relative Weight of the Heart of Albino Rats Undergoing Physical Exertion.

Group of animals	Relative wt. of heart (g)	
	males	females
Control	0,33(0,28—0,42)	0,34(0,30—0,38)
1	0,44(0,32—0,65)	0,34(0,31—0,37)
2	0,41(0,33—0,64)	0,36(0,30—0,45)

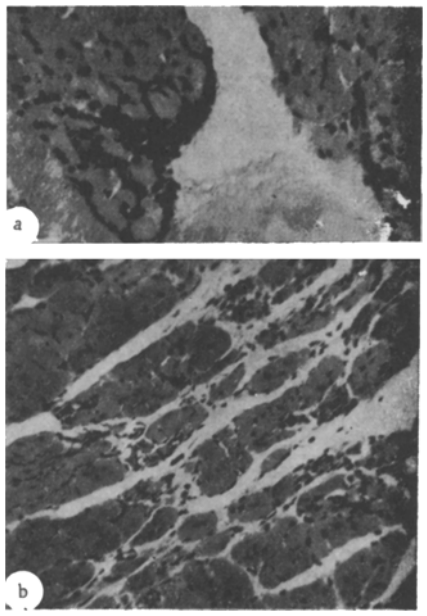

Fig. 1. Myocardium of male rat group 1 (killed on 112th day): a) capillary muscle from left ventricle. Necrosis of group of muscle cells with proliferation of macrophages and leukocytes at the periphery. Hematoxylin–eosin, 230×; b) pectinate muscles of left ventricle. Numerous tiny foci of proliferating granulation tissue and formed scars. Hematoxylin–eosin, 120×.

EXPERIMENTAL RESULTS

Daily swimming, even for short periods, was tolerated less well by the animals (especially the males) than swimming for periods three times longer but less frequent (with rests lasting 2-3 days). For instance, in one-third in the male rats of group 1 evidence of overfatigue was observed regularly by the end of the first month: exophthalmos, brown rings around the eyes during swimming, restless movements and convulsions after it [9]; by the end of the experiment these signs were observed in half of the males. In the female rats of group 1 these phenomena sometimes appeared on the day when the duration of swimming was next increased. In the animals of group 2 symptoms of overfatigue were absent during the first month, while in the last week they appeared at times in the females.

The most rapid increase in body weight was observed in the males of group 2; in the animals of group 1 the increase in weight began to slow down with the appearance of the first signs of overfatigue, but later it began to increase but only slowly, and by the end of the experiment it was definitely reduced. In the females of both groups the curves of the gain in body weight remained for a long time close to those for rats of the control group, and only in the last weeks of the experiment did they acquire the same character as the curves for the males.

Comparison of the mean values of the relative weight of the heart of the experimental and control rats revealed hypertrophy of the myocardium in all the experimental animals (Table 1).

Regardless of the conditions of swimming, the relative weight of the heart in the females remained unchanged. Consequently, the hypertrophy of the myocardium corresponded to the gain in body weight. The highest relative weight of the heart in male rats was found in the animals of group 1.

Microscopic examination of the hearts of 15 males in group 1 showed that in 11 of them disseminated focal injuries were superposed upon hypertrophy of the myocardium, while in four hearts the changes were widespread. In six rats sacrificed on the thirtieth day individual muscle fibers had lost their cross-striation, sometimes with homogenization of their sarcoplasm or, less commonly, with contractural degeneration of coagulation necrosis; clusters of cells resembling fibroblasts, with a cytoplasm rich in RNA, were found in the stroma. In four of the nine rats sacrificed on the 112th day the changes described above in the inner layers of the ventricular myocardium were combined with foci of coagulation necrosis of small groups of muscle cells, surrounded by macrophages and leukocytes (Fig. 1a) and by multiple foci of proliferating granulation tissue at a varied stage of maturation, consisting in some cases of small scars (Fig. 1b).

Among the male rats of group 2 these foci of injury were found in three animals sacrificed after 112 days; they were widespread in character in only one animal and in two cases they consisted of tiny disseminated foci. In eight males changes in the myocardium were confined to its hypertrophy.

Irrespective of the program of swimming, the heart muscle of the females was indistinguishable from normal. Focal lesions of the myocardium like those in the males were observed in two of the seven females of group 1 and in one of the 11 females of group 2.

Clearly, exposure to physical exertion at different frequencies had a beneficial effect on most animals and only in some of the males in group 1 were lesions of the myocardium produced, accompanied by changes in the ECG of the same character as in athletes during chronic myocardial strain [6]. For instance, in the experimental rats with signs of fatigue and widespread lesions of the myocardium, the ECG showed lowering or inversion of the T wave, an increase in the heart rate (by 17-20 beats), and a decrease in voltage of the QRS complex (on the average by 3 mm), indicating a fall in the biological activity of the myocardium; in individual rats electrical alternation and extrasystoles were observed. In the animals which adapted themselves readily to the exertion, the ECG showed elevation of the T wave, a decrease in the heart rate (on the average by 30 beats/min), an increase of 2.4 mm in the voltage of the QRS complex, and no signs of electrical alternation or extrasystoles.

Two opinions, complementary to each other, are held on the genesis of the lesions of the myocardium in the literature. Some workers [2, 10] explain them by a disturbance of metabolism based on hypoxia caused by the difference between the volume of blood flowing into the heart and the volume required to perform the given amount of work. This hypothesis is confirmed by the writers' investigations [8] demonstrating dystonia of the walls of the coronary arteries of the heart and inadequate capillarization of the heart muscle in overtrained rats. Some workers [1, 4] consider that damage to the overstrained heart is due to the action of adrenergic catecholamines on it, with disturbance of the electrolyte balance. In rats well adapted to hyperkinesia, the content of K^+ ions in the heart muscle, as the writers have shown, is increased while the content of Na^+ is reduced; the potassium/sodium ratio is increased on the average to 2.08. In rats in a state of chronic fatigue the K^+ content in the heart muscle is indistinguishable from the control, while the Na^+ content increased, so that the electrolyte coefficient is reduced to 1.78; this is one reason for depression of the contractile function of the myocardium [11].

One of the factors preventing the development of overstrain of the heart is thus individualization of the training program, drawn up to allow as far as possible for complete recovery of the damaged tissues. Observance of this condition allows the animals to withstand considerable physical exertion regularly.

LITERATURE CITED

1. L. A. Butchenko, Degeneration of the Myocardium [in Russian], Leningrad (1971), p. 147.
2. S. S. Vail', Arkh. Pat., No. 8, 25 (1967).
3. N. D. Graevskaya, Functional State of the Cardiovascular System and Health of Higher-Grade Athletes During Prolonged Training. Author's Abstract of Doctoral Dissertation, Moscow (1969).
4. A. G. Dembo, in: The Heart and Sport [in Russian], Moscow (1968), p. 374.
5. A. G. Dembo, in: Civilization, Sport, and the Heart [in Russian], Moscow (1968), p. 23.
6. L. I. Levina, Hypertrophy of the Myocardium in Athletes. Author's Abstract of Candidate's Dissertation, Leningrad (1969).
7. S. P. Letunov, R. E. Motylyanskaya, L. M. Korobochkin, et al., Teoriya i Prakt. Fix. Kul't., No. 9, 24 (1968).
8. N. B. Likhacheva, V. M. Pinchuk, and L. B. Sukhanina, Abstracts of Proceedings of a Scientific Conference on the Results of Work in 1969 at the P. F. Lesgaft Institute of Physical Culture [in Russian] Leningrad (1970), p. 9
9. V. M. Pinchuk and L. B. Sukhanina, Byull. Éksperim. Biol. i Med., No. 6, 29 (1970).
10. F. Büchner, Klin. Wschr., 16, 1409 (1937).
11. J. M. L. Walker and M. Weatherall, J. Physiol. (London), 166, 33 (1963).