

EXTENSIBILITY OF HEART MUSCLE DURING ADAPTATION TO PHYSICAL EXERTION

V. I. Kapel'ko, C. Pfeiffer,
and F. Z. Meerson

UDC 612.176

Strips of papillary muscles from the left ventricle of rats were stretched by weights of increasing size. Extensibility of the myocardium was characterized by plotting lengthening of the strips as a function of load. Extensibility of the myocardium was considerably increased 7-11 weeks after the beginning of regular physical exercises (swimming for periods of 30-60 min). As a result the maximal amplitude of the contractions was reached in response to a smaller stretching force. The increased extensibility of the heart muscle may be one of the factors in the dilatation of the "trained" heart and the more rapid mobilization of its contractile function during physical exertion.

KEY WORDS: myocardium; adaptation to physical exertion; extensibility of heart muscle; papillary muscles.

Dilatation of the heart is an invariable component of adaptation of the organism to periodic physical exertion, especially in athletes trained in endurance [2, 5, 6, 11, 13]. The dilatation may arise both through an increase in the number of sarcomeres in the consecutive series and also through an increase in the extensibility of the heart muscle tissue itself.

In this investigation the extensibility of isolated capillary muscles of rats adapted to physical exertion was studied.

EXPERIMENTAL METHOD

Rats were made to swim in water at 32°C 5 times a week for periods of 30-60 min daily. Each experiment lasted 7-11 weeks. As a result of exertion the weight of the left ventricle of the experimental animals increased to $107 \pm 1.2\%$ of its expected value in control rats of the same body weight. Strips of posterior papillary muscles of the left ventricle were investigated. Strips of about equal thickness (0.5-1 mm) were taken so as to exclude differences in their function [3, 12] resulting from unequal diffusion of oxygen. The strips were made to contract for 1 h in Krebs' solution at 30°C during electrical stimulation at a frequency of 20/min while raising a small weight of 0.25 g. Extensibility was characterized as the ratio between the stretching weight and the length of the strip. The minimal weight was 0.05 g and it was increased gradually to 1.5 g. At each increase in weight a pause of 5-7 min was allowed for the slow "creep" of the muscle to be completed. The change in length of the muscle strips was estimated from the movement of an isotonic lever, to the arm of which the muscle was attached. This displacement was measured by a capacitance transducer and recorded on a "Disa" indicator by means of a "Cossor" camera. The design of the apparatus was described previously [8]. To compare the results of different experiments the area of cross section of the strips was calculated (from their length and weight) and the ratio between the stretching weight and this area was determined. The degree of lengthening was measured in per cent of the length of the strip when stretched by a minimal weight of 0.1 g/mm².

Laboratory of Pathophysiology of the Heart, Institute of General Pathology and Pathophysiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 82, No. 9, pp. 1032-1034, September, 1976. Original article submitted August 18, 1975.

This material is protected by copyright registered in the name of Plenum Publishing Corporation, 227 West 17th Street, New York, N.Y. 10011. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, microfilming, recording or otherwise, without written permission of the publisher. A copy of this article is available from the publisher for \$7.50.

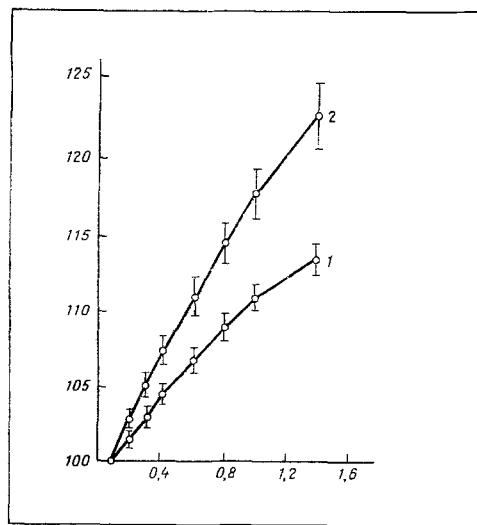


Fig. 1. Relationship between stretching load and extensibility of strips of rat papillary muscles: 1) control; 2) experiment. Values of $M \pm m$ shown. Ordinate, increase in length of strip relative to initial length (with load of 0.1 g/mm^2), taken as 100%; abscissa, stretching weight applied (in g/mm^2).

EXPERIMENTAL RESULTS AND DISCUSSION

The relationship between the stretching weight and the increase in length of the strips of myocardium from the control and experimental animals is shown in Fig. 1 (mean data from 14 experiments on control strips and 9 experiments on strips from experimental rats). The myocardium of the adapted animals clearly possessed much higher extensibility. The difference was statistically significant ($P < 0.05$) starting from a resting tension of 0.3 g/mm^2 and it rose progressively with an increase in the resting tension. To stretch the strips by 10% of their initial length a load of $0.55 \pm 0.07 \text{ g/mm}^2$ was required for the adapted animals and one of $0.90 \pm 0.05 \text{ g/mm}^2$, i.e., 1.5 times greater, for the control animals. The more extensible the muscle, the smaller the weight which had to be attached in order to stretch it to the length at which maximal amplitude of contraction was observed. For the control strips this value averaged 0.6 g/mm^2 and for strips from adapted animals it was 0.2 g/mm^2 .

The increase in extensibility of the heart muscle was evidently not connected with a decrease in rigidity of the elastic skeleton of the heart muscle, for the concentration of collagen — the main structural protein of the supporting tissues — is unchanged during training for physical exertion [10]. Consequently, an increase in the extensibility of the contractile component of the muscle can be postulated. Hill's view that the contractile component is freely extensible and exerts no resistance to the stretching force, which has been favored until recently, is now disputed, at least for heart muscle [4, 16].

The extensibility of the contractile component depends on the degree of interaction between actin and myosin filaments in the myofibrils, which is controlled by the residual Ca^{++} concentration in the myoplasm. The available data suggests that the power of the system removing Ca^{++} from the myofibrils increases during adaptation to physical exertion [9]. This leads to an increase in the degree of relaxation of the fibers [9] and to a small decrease in the Ca^{++} concentration in heart muscle [14]. Increased extensibility may thus be the direct result of increased power of the ion transport system.

When assessing the physiological role of increased extensibility of the myocardium it must be remembered that it may be a component of the formation of dilatation of the heart which, in conjunction with other factors, brings about the great increase in the pumping function of the myocardium in trained persons and animals.

Under conditions of physiological rest, when the tissue oxygen demand is reduced, the activity of the dilated heart of the athlete is characterized by a normal or even reduced stroke volume and by bradycardia.

This means that the usual dependence of stroke volume on final diastolic volume is altered and the expulsion fraction may be reduced by 1.5-2 times [6]. At rest the degree of shortening of the sarcomeres in the hearts of trained persons and animals is evidently reduced, the process taking place against the background of a lowered intensity of sympathetic influences [1, 7, 15]. Meanwhile the high residual volume is a rapidly mobilizable reserve of contractile function of the heart.

LITERATURE CITED

1. A. L. Gorokhov, *Fiziol. Zh. SSSR*, No. 11, 1411 (1969).
2. N. D. Graevskaya, A. D. Butkov, and E. S. Stepanova, in: *Dilatation of the Heart and Hypertrophy of the Myocardium in Athletes* [in Russian], Moscow (1973), p. 22.
3. V. I. Kapel'ko, *Byull Éksp. Biol. Med.*, No. 12, 6 (1970).
4. V. I. Kapel'ko, *Biofizika*, No. 3, 474 (1974).
5. V. L. Karpman, S. V. Khrushchev, and Yu. A. Borisova, in: *Dilatation of the Heart and Hypertrophy of the Myocardium in Athletes* [in Russian], Moscow (1973), p. 5.
6. L. Komadel et al., *Physiological Enlargement of the Heart* [in Russian], Bratislava (1968).
7. O. D. Kurmaev, A. S. Chinkin, and I. A. Zhdanov, *Fiziol. Zh. SSSR*, No. 5, 789 (1974).
8. F. Z. Meerson and V. I. Kapel'ko, *Fiziol. Zh. SSSR*, No. 7, 887 (1972).
9. F. Z. Meerson, V. I. Kapel'ko, and S. I. Shaginova, *Kardiologiya*, No. 4, 5 (1973).
10. D. Bartosova, M. Chvapil, B. Korecky, et al., *J. Physiol. (London)*, 200, 285 (1969).
11. W. Gebhardt, U. Wierig, J. Keul, et al., *Arch. Kreislaufforsch.*, 49, 188 (1966).
12. A. H. Henderson, D. L. Brutsaert, W. W. Parmley, et al., *Am. J. Physiol.*, 217, 1273 (1969).
13. Z. Hornof and J. Novak, *J. Sport Med.*, 13, 297 (1973).
14. R. Rosenfeld, A. Rosenfeldova, J. Steiglova, et al., *Physiol. Bohemoslov.*, 22, 195 (1973).
15. C. De Schryver, J. Mertens-Strythagen, L. Becsei, et al., *Am. J. Physiol.*, 217, 1589 (1969).
16. A. Y. Wong, *J. Biomech.*, 5, 107 (1972).