THE CONTRACTILE FUNCTION OF CARDIAC MUSCLE IN EXPERIMENTAL MYOCARDITIS

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Researches devoted to a study of the metabolism of cardiac muscle [11, 13, 15], the structure of the myocardium [2, 4, 7, 8, 10] and its bioelectrical activity [1, 4, 6] in experimental adrenaline myocarditis furnish grounds for supposing that the contractile capacity of cardiac muscle is reduced under certain experimental conditions. However, no direct examination of the contractile function of the myocardium in myocarditis has been carried out.

The aim of the present work was to study the contractile function of cardiac muscle during experimental myocarditis induced by theophylline and adrenaline.

EXPERIMENTAL METHOD

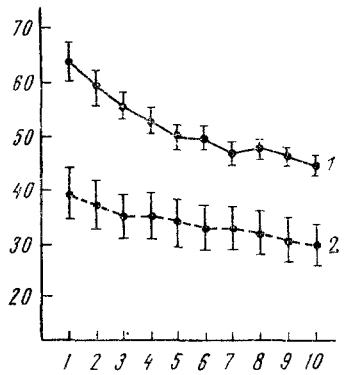
Myocarditis was produced in 28 female rabbits each weighing 2-3 kg. The animals were injected with 1% solution of theophylline (20 mg/kg) and 2 min later with 0.2 ml 0.1% solution of adrenaline hydrochloride injected slowly for 3 min.

For studying the contractile function of the myocardium of the left ventricle we employed a method permitting one to estimate the maximal attainable strength of the contraction of the myocardium and to follow the development of the process of muscular fatigue [3]. The method involved the opening of the thoracic cavity under combined narcosis (urethane 1.4 g/kg and chlorolose 50 mg/kg) with the aid of artificial respiration. Near the origin of the ascending aorta, distal to the exit of the coronary artery, a special appliance permitting the instantaneous pinching of the aorta was introduced. The pressure in the cavity of the left ventricle was registered electromanometrically with the aid of needles inserted through the top of the heart.

A full constriction of the aorta for 30 sec was carried out 10 times at intervals of 5 min. The maximum systolic pressure in the left ventricle at the time when the aorta was completely closed served to indicate the maximal attainable strength of the contraction of the left ventricle as a whole. During the first pinching of the aorta this pressure amounted, on an average, to 216-234 mm Hg and with each subsequent pinching it was reduced by 6-18 mm Hg. Thus, successive pinchings of the aorta enabled a curve to be drawn tracing the gradual reduction in the contractile function of the left ventricle and showing the course of development of myocardial fatigue.

As an index of the contractile capacity of the muscle tissue of the left ventricle, the maximal attainable intensity of the functioning of the structure (IFS) [11, 12], or the contractile capacity of unit mass of myocardium, was employed. In order to determine the IFS, the maximum systolic pressure in the left ventricle, which is indicative of the contractile function of the left ventricle as a whole, was divided by the weight of the ventricle.

The contractile function of the myocardium was studied in 5 series of experiments. In the first series it was determined in 10 control animals; in the 2nd series in 8 animals with acute myocarditis, that is, 3 days after myocarditis had been induced by means of the simultaneous injection of theophylline and adrenaline; in the 3rd series



Dynamics of the maximal attainable IFS of the left ventricle during repeated pinching of the aortic aperture. 1) Control; 2) acute relapsing myocarditis. Abscissas) ordinal number of aortic pinchings; ordinates) magnitude of the maximal attainable IFS in mm Hg. Vertical lines show the mean square errors.

in 7 rabbits with chronic myocarditis, that is, 30 days following the injection of the two compounds. The 4th and 5th series of experiments were devoted to a study of the contractile function of the myocardium in relapsing myocarditis. Myocarditis was again induced by means of a secondary injection of theophylline and adrenaline a month after the 1st injection. In the 4th series of experiments the contractile function of the myocardium was determined in 7 rabbits 3 days after the 2nd induction of myocarditis (acute relapsing myocarditis); in the 5th series, 6 rabbits were examined for 30 days following a repeated induction of myocarditis (chronic relapsing myocarditis).

EXPERIMENTAL RESULTS

The weight of the left ventricle in rabbits with acute and chronic myocarditis was raised by 25% in comparison with that of control animals. An analysis of the curve tracing the development of fatigue in the myocardium showed that, both in acute and chronic myocarditis, the increase in the maximum systolic pressure, which is indicative of the strength of the contraction of the left ventricle as a whole, was no different from that in the controls. The maximal attainable IFS, characteristic of the contractile capacity of the myocardial tissue of the left ventricle, tended to be reduced in animals with acute myocarditis and was 20% lower than in the controls, while in animals with chronic myocarditis the given index, beginning with the 8th pinching of the aorta, was definitely lower than the controls by 18%. This implies that in chronic adrenaline myocarditis the process of fatigue in the myocardium developed quicker than in animals of the control group.

Since, in clinical work, the course of myocarditis appears to be recurrent, the next stage of our studies dealt with the experimental induction of myocarditis by repeating the injection of theophylline and adrenaline a month after the 1st injection.

In acute relapsing myocarditis the weight of the left ventricle was increased by 69% compared with that of the control animals. The maximum systolic pressure peak in the left ventricle did not differ from that in the controls. However, the maximal attainable IFS of the myocardium, characteristic of the contractile capacity of the myocardial tissue, was reduced by 40% (see figure).

In rabbits with chronic relapsing myocarditis the weight of the left ventricle was increased by 15%. There was an insignificant reduction in the maximum systolic pressure peak in the left ventricle, but the maximal attainable IFS dropped very considerably—by 32%.

By comparing the results obtained in experiments on rabbits with myocarditis induced by a single injection of theophylline and adrenaline with those on rabbits affected by relapsing myocarditis, it was found that in the former the reduction in the maximal attainable IFS of the myocardium occurred only when the myocardium had become fatigued (beginning at the 8th pinching), while the reduction in the IFS of the myocardium in animals with relapsing myocarditis was noted already in the unfatigued myocardium from the first pinching of the aorta.

Thus, in animals with acute and chronic myocarditis the contractile function of the left ventricle as a whole did not alter substantially, but the maximal attainable IFS of the myocardium was definitely reduced. This defect in the contractile function of the myocardium is not apparent when a determination of the strength of the contraction of the ventricle as a whole is made. It is compensated for and disguised by an increase in the mass of the ventricle and can be observed only by determining the IFS of the myocardium, that is, by determining the contractile capacity of unit mass of myocardial tissue.

The reasons for the disturbance in the efficiency of the contractile function of the muscle tissue in myocarditis might be understood on the basis of modern biochemical research. Laboratory data obtained by S. E. Severin indicate that in experimental myocarditis the glycolytic [14] and the oxidizing [9] formation of ATP in the cardiac muscle tissue is disrupted, and, as a consequence, the content of high-energy compounds such as ATP, ADP, AMP, and also phosphocreatine and creatine [15], necessary for accomplishing the contractile function, is reduced. Together

with such disturbances in the energy metabolism during myocarditis, a drop in the content of the contractile myofibrillar proteins per unit mass of heart muscle is observed. It might be assumed that the combination of the disturbances in the energy and metabolism of the myocardium and also the morphological changes in the structure of the myocardial tissue are the reasons for the reduction in the contractile function of the muscle tissue of the heart which we encountered in myocarditis.

SUMMARY

The study is devoted to the examination of the contractile function of the cardiac muscle in myocarditis caused by adrenaline and theophylline. It was shown that in animals with acute and chronic myocarditis the contractile function of the left ventricle is not, on the whole, changed substantially, whereas the contractile capacity of the unit of the myocardial tissue is distinctly reduced. This defect in the contractile function of the cardiac muscle in myocarditis is not manifest in determination of the strength of ventricular contraction as a whole, since it is compensated for by an increase in the mass of the ventricle and can be detected only by determining the contractile capacity of the unit of the myocardial tissue mass.

LITERATURE CITED

1. S. V. Andreev and Z. G. Trofimova, Pat. fiziol., No. 6 (1959), p. 35.
2. N. N. Anichkov, On the Inflammatory Changes in the Myocardium [in Russian], Diss. SPb. (1912).
3. N. M. Bakli, F. Z. Meerson, L. A. Pogosyan, and others, Byull. éksper. biol., No. 5 (1964), p. 27.
4. Z. I. Vedeneeva, In the book: Yearbook of the Institute of Experimental Medicine, AMN SSSR for 1959 [in Russian], Leningrad (1960), p. 139.
5. O. P. Vishnevskaya, Byull. éksper. biol., No. 10 (1954), p. 29.
6. L. N. Dagaeva, Pat. fiziol., No. 3 (1961), p. 26.
7. K. M. Danilova, Arkh. pat., No. 11 (1961), p. 11.
8. V. I. Zavrazhnov, Material for Experimental Pharmotherapy of Myocarditis and Myocardiosclerosis [in Russian] Aftoref. diss. dokt., Sverdlovsk (1960).
9. A. M. Zubovskaya, Byull. éksper. biol.. No. 3 (1961), p. 55.
10. L. Ya. Koberidze, Tezisy i referaty dokl. 11-i otchetnoi nauchnoi konferentsii Dagestansk. med. Inst. po rabotam 1960. Makhachkala (1961), p. 57.
11. F. Z. Meerson, G. A. Malov, M. G. Pshennikova, and others, Material for the Second All-Union Scientific Cardiological Conference [in Russian], Moscow (1963), p. 6.
12. F. Z. Meerson, Yu. M. Repin, and V. P. Godin, Dokl. AN SSSR, 152, 6 (1963), p. 1483.
13. S. E. Severin, L. A. Tseitlin, and A. M. Zubovskaya, Tesizy dokl. 1-i Biokhimicheskoi konferenstii prebaltiiskikh respublik i Bellorussii Tartu (1960), p. 121.
14. S. E. Severin and L. A. Tseitlin, Vopr. med. khimii, 2 (1961), p. 201.
15. L. A. Tseitlin, Vopr. med. khimii, 3 (1962), p. 279.
16. M. S. Fleisher and L. Loeb, Arch. intern. Med., 6 (1910), p. 427.
