A FUNCTIONAL AND MORPHOLOGICAL STUDY OF THE RECOVERY PATTERN OF THE MYOCARDIUM AFTER ACUTE FATIGUE

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We have shown [2] that a considerable decrease in the glycogen concentration in the myocardium takes place in animals developing acute fatigue, but at the same time the intensity of histochemical reactions for protein shows no change.

Fig. 1. Almost complete absence of glycogen in the myocardium of the left ventricle of a fatigued rat (swimming for 1 h 30 min). Stained with PAS. Magnification 150 ×.

We were interested in studying the reversibility of the changes observed in the carbohydrate metabolism of the heart after the cessation of fatigue, and to compare these findings with the results of electrocardiographic investigations. Little has been published on this subject in the literature. Blount and Meyer [5] carried out biochemical investigations and found that the glycogen concentration in the myocardium of rats sacrificed 1 h after the cessation of physical exertion (swimming for 1 h) was much greater than in the myocardium of control animals. A. G. Filippova [3] observed that the ECG changes developing in dogs as a result of prolonged running were weakened or disappeared altogether 60 min after cessation of the exertion.

We have used a histochemical method to study the concentration of glycogen and proteins (SH-groups and "total" protein) in the heart muscle of albino rats at various periods after the cessation of fatigue. Electrocardiographic investigations were undertaken at the same time.

EXPERIMENTAL METHOD

The animals were made to undergo physical exertion until a state of total exhaustion developed. This took place in the rats after swimming for 1-1½ h in water at a temperature of 30° and running for 20 min on a moving belt. For the histochemical study of the myocardium, the animals were sacrificed immediately, and also at intervals of 5, 10, 20, and 30 min and 1, 2, 2½, and 4 h after cessation of the exertion. The heart was extracted quickly and transverse sections were cut through all parts of the organ. The tissue was fixed in Shabadash's or Carnoy's fluids and embedded in paraffin wax.

Glycogen was determined by Shabadash's method after preliminary treatment of the sections with amylase. The SH-groups were studied by the method of V. A. Yakovlev and S. N. Nistratova, and the "total" protein was determined by Danielli's method by means of a tetrazonium coupling reaction. Sections were also stained with hematoyxlin-eosin and iron-hematoxylin by Heidenhain's method. The ECG was recorded by the three standard leads immediately, and at intervals of 20 and 30 min and 1, 2, and 2½ h after the cessation of fatigue. Healthy animals not subjected to fatigue acted as controls.

EXPERIMENTAL RESULTS

The experiments showed that the myocardium of the control animals contained a considerable amount of glycogen, unevenly distributed in the various divisions of the heart. Nearly all the fibers of the inner layers of the myocardium contained glycogen, whereas many of the fibers in the outer portions of the heart were without glycogen. The glycogen in the inner layers of the myocardium was distributed mainly diffusely, in the form of small granules, and in the fibers of the outer layers it was present in the form of large granules, concentrated in the anisotropic disks of the myofibrils, or haphazardly throughout the fiber. Intensive histochemical reactions for protein (SH-groups and "total" protein) were observed in the myocardial fibers.

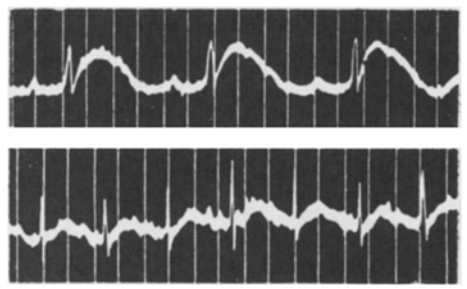

Fig. 2. ECG of a rat after exertion (swimming). Lead 2. a) Immediately after cessation of exertion; heart rate 206 per min, elevation of ST interval; b) 2 h after cessation of exertion; heart rate 461 per min, ST interval isoelectric.

According to the electrocardiographic data, the electrical axis of the heart was undeviated in the control animals, or was deviated toward the left: RR = 0.12 sec, PG = 0.04-0.05 sec, QRS = 0.03-0.04 sec, and QRST = 0.09-0.12 sec. The heart rate was 460-545 beats per min.

During physical exertion by the animals, the glycogen content in their heart fell considerably (Fig. 1). The ECG's taken as soon as the rats had finished their exercise showed a marked slowing of the cardiac contractions (to 428-350 per min or less). Meanwhile, changes in the deviation of the electrical axis of the heart and changes in the amplitude of the R and S waves were observed. In some cases, the ST interval was elevated. These changes were particularly marked in the animals forced to swim. Their heart rate fell from an initial 460-500 per min to 300-222 per min. The elevation of the ST interval was very marked (Fig. 2a).

Investigations of the myocardium in the rest period after exertion showed that within 5-10 min of the cessation of running, glycogen began to appear in large amounts in the inner layers of the myocardium. Glycogen granules were found in considerable numbers in both the inner and the outer divisions of the myocardium of the animals sacrificed 20 min after the cessation of running, as in the heart of the control animals. The indices of the ECG were also fully restored at this period after running.

An increase in the glycogen content was observed in the myocardium of the animals sacrificed 1 h after the cessation of running. Not merely the fibers of the inner layers, but also nearly all the fibers of the outer portion of the myocardium were filled with glycogen granules, which were concentrated selectively in the anisotropic disks of the myofibrils. The accumulation of excess of glycogen in the heart was observed for 1-2 h. No increase in the protein concentration was seen under these circumstances.

In the animals fatigued by swimming, the glycogen content in the myocardium 5, 20, 30, and 60 min after the cessation of exertion remained at the same low level as in the heart of the animals sacrificed immediately after exertion. The glycogen granules in these animals were present in very small numbers, and only in the subepicardial layers. The ECG's taken 30 min to 1 h after the cessation of swimming showed that at this time the heart rate still remained low (352-375 beats per min), whereas the other indices of the ECG had almost fully recovered.

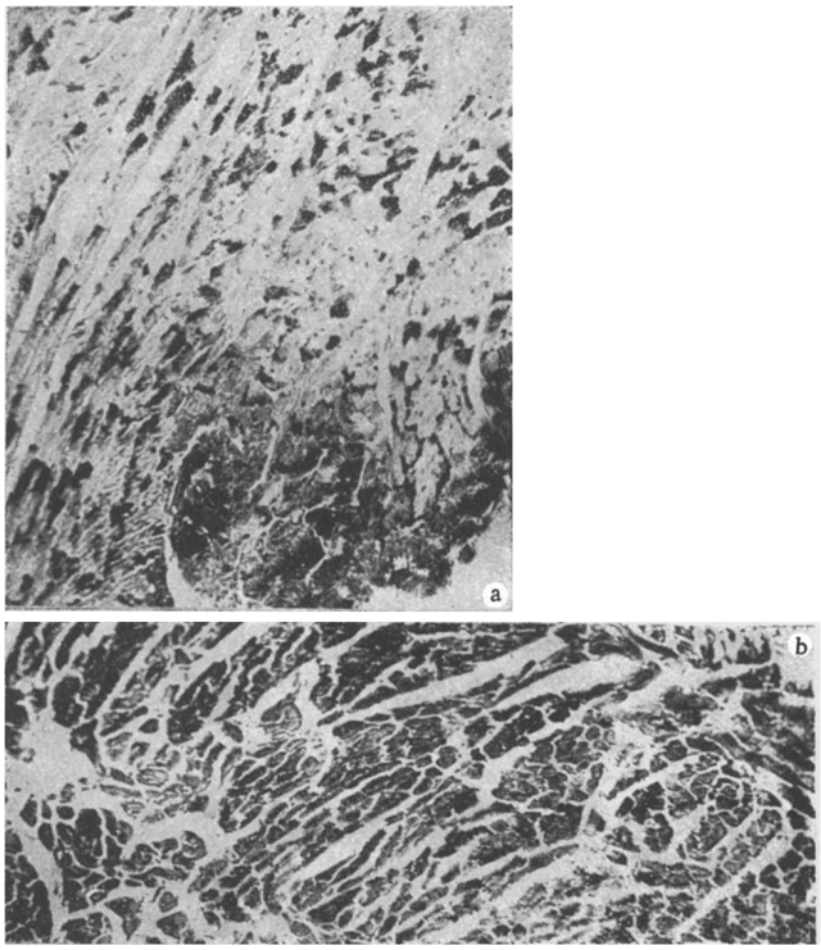

Fig. 3. a) Accumulation of glycogen in the myocardium of the left ventricle of a rat sacrificed $2\frac{1}{2}$ h after the cessation of swimming; b) 4 h after the cessation of swimming. Stained with PAS. Magnification 150 ×.

The heart rate was restored only when 2-$2\frac{1}{2}$ h had elapsed after the cessation of swimming (Fig. 2b). The glycogen content in the myocardium was also observed to be restored at this time. Two hours after the cessation of swimming, many glycogen granules were observed in the inner layers of the myocardium, and after $2\frac{1}{2}$ h glycogen also appeared in the outer layers (Fig. 3a).

Excessive accumulation of glycogen in the heart took place in the animals fatigued by swimming only when 4 h had elapsed after the end of their exertion. At this time the myocardium of the resting animals contained very large amounts of glycogen (Fig. 3b), and the cardiac fibers were filled with glycogen granules, situated in the myofibrils along the course of the cross striations in the outer layers of the myocardium and diffusely throughout the fibers in the inner layers.

Hence, the changes in the carbohydrate metabolism of the heart arising in the course of acute fatigue of the animals were readily reversible. Restoration of the energy supplies of the myocardium took place parallel with normalization of the indices of the ECG, but a considerable difference was observed between the speeds of recovery after different types of exertion.

Swimming evidently causes a greater degree of anoxia than running for short periods, and the restoration of the glycogen content, like the normalization of the ECG indices, therefore takes place more slowly. Support for this suggestion is given by the marked displacement of the ST interval in the animals fatigued by swimming, indicating a profound disturbance of the metabolic processes in the myocardium [1,3].

The restoration of the functional capacity of the heart muscle after the cessation of fatigue is evidently closely associated with the restoration of the glycogen content of the heart in the fatigued animals.

SUMMARY

Histochemical and functional (ECG) study was made of the cardiac muscle of albino rats in the period of rest after acute fatigue caused by running for 20 min and swimming for $1\frac{1}{2}$ h.

The glycogen content is restored 20 min after discontinuance of running and only normalized with the restoration of glycogen content. One hour after the rat has stopped running, and 4 h after it has stopped swimming, an excessive accumulation of glycogen granules is seen in the myocardial fibers lasting from 1-2 h. There is no rise of the protein concentration in these conditions.

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