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.

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 Danielff's method by means of a tetrazonium coupling reaction. Sections were also stained with hematoxylin-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.

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.