PHYSIOLOGY

THE ROLE OF CARBOHYDRATE-PHOSPHORUS METABOLISM IN THE PRODUCTION OF THE EXCITATION CURRENT IN SKELETAL MUSCLE

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In recent years several research workers have studied the role of metabolism in the production of bioelectrical phenomena. A relationship has been demonstrated between metabolism on the one hand and both "stationary" electrical processes of long duration (currents of injury, salt-induced or temperature currents) and "dynamic" rapidly developing electrical processes (excitation currents of nerve and muscle tissue).

A relationship has been shown between the current of injury of skeletal muscle and nerve [1, 5, 6, 9, 11, 13], salt-induced currents [2] and temperature currents [4] and the carbohydrate-phosphorus metabolism.

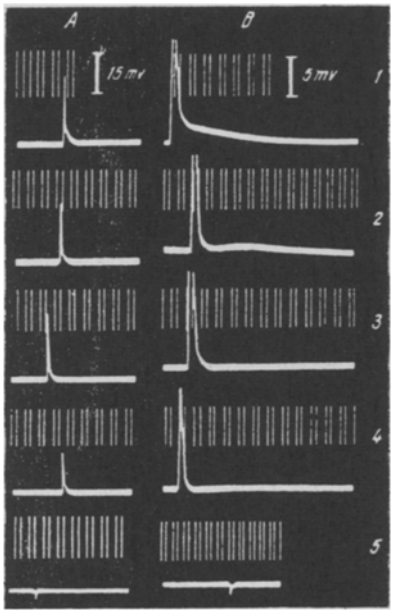

Fig. 1. The effect of poisoning the muscle with monoiodiacetate on the excitation EMF.
Time marker) 0.05 seconds. 1) initial recording; 2) after resting for 30 minutes in monoidoacetate solution; 3) after 60 minutes; 4) after 90 minutes; 5) after resting for 120 minutes in the same solution.

It has been found that the magnitude and form of the excitation current of skeletal muscle depend on carbohydrate metabolism [3, 8, 12] and also that disturbance of glycolytic processes affects the electroretinogram and the electrocardiogram [7]. During poisoning of the skeletal muscle of the frog with monobromoacetic acid, changes are found in the after-potentials only, with no change in the action current peak [12].

Changes in the blood sugar concentration in cats were found to be accompanied by changes in the excitation current peak in skeletal muscle [3]. In earlier work on the study of the effect of poisoning the skeletal muscle of the frog with monoiodoacetic acid on the excitation current, no changes in the latter were found [10]. Poisoning the skeletal muscle of the frog with sodium fluoride leads to a reduction in the amplitude of the excitation current [8].

The object of the present investigation was to study further the role of carbohydrate metabolism in the production of the excitation current of skeletal muscle.

EXPERIMENTAL METHOD

Experiments were carried out on the isolated sartorius muscles of autumn frogs. The sartorius muscle was carefully dissected, and placed on a paraffin wax block in a humid chamber for detection of the current. To avoid distortion of the recording during contraction, the muscle was fixed in a slightly stretched position. The excitation current was taken from damaged and undamaged areas of the muscle by

means of nonpolarizing ($Zn-ZnSO_4$) electrodes to a loop electrocardiograph. With leads of this type the excitation current was always monophasic.

Direct stimulation of the muscle was carried out by means of a neon interrupter. The duration of each impulse was 1 millisecond and the intervals between the impulses were of one second. The current strength was always over threshold (the muscle gave a maximum contraction).

The distance between the stimulating platinum electrodes was 1-1.5 mm, the distance between the stimulating and take-off electrodes was 10 mm, and that between the take-off electrodes was 5-10 mm. The short duration of the stimulating current and the position of the electrodes in which the excitation current and the stimulation current (both monophasic) could be arranged to have their apices on opposite sides, enabled the currents to be recorded without any distortion due to the "loop" of the stimulating current.

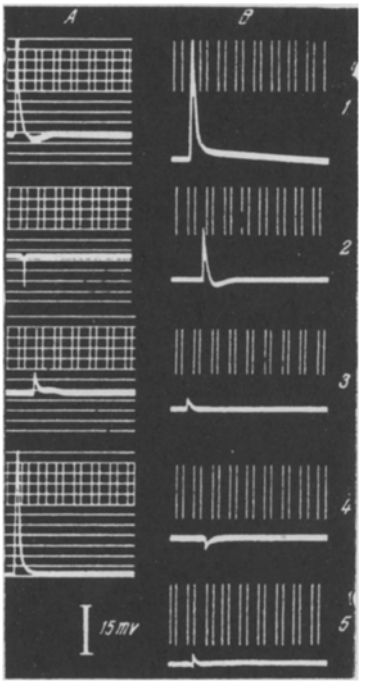

Fig. 2. The effect of poisoning the muscle with cyanide on the excitation EMF.
Time marker) 0.05 seconds. A) Poisoning with a 0.1% solution: 1) initial recording; 2) after resting for 30 minutes in cyanide solution; 3) after resting for 30 minutes in Ringer solution; 4) after 60 minutes. B) Poisoning with a 0.01% solution: 1) initial recording; 2) after resting for 30 minutes in cyanide solution; 3) after 60 minutes; 4) after 90 minutes; 5) 30 minutes after resting in Ringer solution.

In the intervals between recording the bioelectric currents the muscles were kept in Ringer solution, to which various substances could be added as necessary.

Changes in the excitation current were studied:

1) during poisoning of the muscle with a solution of sodium monoiodoacetate (0.002%), a solution of sodium fluoride (0.042%) and a solution of sodium cyanide (0.1%);

2) during recovery of the current after rinsing the poisoned muscles with Ringer solution, a 0.002% solution of vitamin B_1 (in monoiodoacetate poisoning of the muscle) and a 0.11% solution of sodium pyruvate (in sodium fluoride poisoning of the muscle);

3) during rhythmic stimulation of the muscle for 10-20 minutes and subsequent keeping of the muscle for 30 minutes in Ringer solution or in vitamin B_1 solution (for muscles poisoned with monoiodoacetate).

EXPERIMENTAL RESULTS

The figures which are given show the changes in the excitation current of the sartorius muscle during monoiodoacetate poisoning (Fig. 1) and cyanide poisoning (Fig. 2). It can be seen that during poisoning of the muscle changes took place in both the magnitude of the peak of the action current and in the after-potential. The size of the peak and also of the after-potential gradually fell and then completely disappeared. Under these circumstances the after-potential disappeared even when the action-potential peak was hardly altered (the first 15-30 minutes from the beginning of the experiment). The opposite muscle, kept in Ringer solution for 2-4 hours, showed no changes in the excitation current.

The presence of vitamin B_1 in the monoiodoacetate solution caused a decrease in the changes in the peak of the excitation current. The presence of pyruvate in the fluoride solution had no such effect.

Rhythmic stimulation by the current for 10 minutes of muscles kept for 1 hour in solutions of sodium monoiodoacetate and fluoride led to a considerable decrease in the peak of the excitation current, and stimulation for 20 minutes – to the complete disappearance of the peak. The opposite muscles, kept in Ringer solution showed no such changes.

TABLE

The Effect of Sodium Pyruvate in a Solution of Sodium Fluoride on the Rate of Fall of the EMF of Injury in the Skeletal Muscle of the Frog (mean of 10 experiments)

Time (in hours)	Control		0.042% sodium fluoride		0.042% sodium fluoride and 0.11% sodium pyruvate	
	absolute magnitude EMF, mv	as a % of the initial value	absolute magnitude EMF, mv	as a % of the initial value	absolute magnitude EMF, mv	as a % of the initial value
0	52	100	55	100	60	100
1	37	71	36	65	44	73
2	34	65	29	53	40	66
3	30	57	24	43	37	61
4	29	55	21	38	35	58
5	28	54	17	30	34	56
6	27	52	15	27	33	55

Rinsing the muscles poisoned with monoiodoacetate with vitamin B_1 solution led to some degree of acceleration of the partial re-establishment of the peak of the excitation current, compared with the muscle on the opposite side, rinsed with Ringer solution. No difference could be found between the effect of rinsing with Ringer solution or with sodium pyruvate solution, in the case of a muscle poisoned with sodium fluoride.

It follows from the experiments described that disorders of both the anaerobic phase of carbohydrate metabolism (poisoning with monoiodoacetate and fluoride) and of the processes of oxidation (cyanide poisoning) led to a decrease or even to complete disappearance of the excitation currents of skeletal muscle. These disorders were to a large extent reversible: rinsing the poisoned muscles with Ringer solution led to some degree of re-establishment of the excitation currents. The excitation currents of muscles poisoned with monoiodoacetate and fluoride could be greatly reduced (or even made to disappear completely) by rhythmic stimulation of the muscles for 10-20 minutes, and they were restored once again if the muscles were kept for 30 minutes in Ringer solution or in vitamin B_1 solution (for a muscle poisoned by monoiodoacetate).

All the changes mentioned in the excitation currents were not directly associated with the development of a contracture in the poisoned muscles, since: 1) the excitation currents disappeared roughly 30 minutes after the contracture had developed; 2) after poisoning of the muscle with sodium cyanide and the development of the corresponding changes in the excitation currents, generally no contracture developed (at least during the time interval of our investigation); 3) placing a muscle poisoned with monoiodoacetate in vitamin B_1 solution led to some increase in the excitation currents, although vitamin B_1 did not prevent the development of a contracture [6].

All this suggests that in this particular case we were in fact dealing with changes in the excitation current depending mainly on changes in carbohydrate metabolism and not on disturbances of the structure of the muscle.

It is also important to mention that disturbance of carbohydrate metabolism does not affect absolutely equally the excitation current and the current of injury. Poisoning of skeletal muscle with sodium cyanide does not lead to any perceptible change in the current of injury [5, 6], whereas it does have a considerable effect on the magnitude of the excitation current. The presence of pyruvate in the sodium fluoride solution completely paralyzed the effect of the latter on the current (EMF) of injury (see Table) but did not affect the changes in the excitation current (EMF).

The findings described suggest that the various phases of carbohydrate metabolism have different importance for the existence of the excitation current and the current of injury.

SUMMARY

A disturbed carbohydrate-phosphorus metabolism produced by the poisoning of an isolated sartorius muscle in a frog with monoiodoacetate fluoride and cyanide results in a variation and complete disappearance of the

skeletal muscle excitation currents. The variation is manifest in the gradual decrease (and at times also in the change of the form) both of the trace potentials and the peak value of the excitation current. The changes begin with the trace potentials. The data obtained allow us to presume the necessity of certain differences in the value of individual phases of the carbohydrate-phosphorus metabolism for the presence of excitation and injury currents.

LITERATURE CITED

[1] É. S. Bauer, V. A. Muzheev and S. D. Borzdyko, Biol. Zhur. SSSR 1, 70-76 (1933).

[2] D. A. Ik'inskii, Trudy Voen.-Med. Akademii im. S. M. Kirova 45, 41-51 (1950).

[3] A. Karaev, The Theory of Bioelectrical Phenomena, Dissertation for Doctorate, (Baku, 1940) [In Russian].

[4] M. M. Kantorovich, Trudy Voen.-Med. Akademii im. S. M. Kirova 45, 60-69 (Leningrad, 1950).

[5] N. I. Mikhel'son, Fiziol. Zhur. SSSR No. 5, 987 - 995 (1935).

[6] A. S. Mozzhukhin, Trudy Voen.-Med. Akademii im. S. M. Kirova, 42, 233-244 (Leningrad, 1948).

[7] A. S. Mozzhukhin, Byull. Éksptl. Biol. i Med. 26, 10, 306 (1948).

[8] A. S. Mozzhukhin, Fiziol. Zhur. SSSR 1, 42-49 (1949).

[9] I. A. Peimer and A. G. Sverdlov, Trudy Voen.-Med. Akademii im. S. M. Kirova 45, 161-166 (Leningrad, 1950).

[10] A. M. Ryabionovskaya, Izvest. Akad. Nauk SSSR Serv. Biol. 2, 303-305 (1941).

[11] R. Lorente de No, In the book: Studies from the Rockefeller Inst. 131, p. 132 (New York, 1947).

[12] E. Lundsgaard, Biochem. Ztschr. 217, 162-177 (1930).

[13] D. Nachmansohn, R. T. Cox, C. W. Coats, et al., J. Neurophysiol. 5, 499-515 (1942).

[14] H. Schaefer and P. Schölmerich, Arch. ges. Physiol. 240, 542-552 (1938).

[15] A. M. Shanes and D. E. Brown, J. Cell. a Comp. Physiol. 19, 1-13 (1942).