EFFECT OF CALCIUM CHLORIDE AND MAGNESIUM SULFATE ON THE DEVELOPMENT OF RIGOR MORTIS

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Modern biochemical research has shown [4] that skeletal muscle contains three different adenosinetriphosphatases: 1) myofibrillary, or actomyosin, activated by the cations magnesium and calcium; 2) sarcoplasmic, activated by the cation magnesium and depressed by calcium; 3) mitochondrial, activated by the cation of magnesium and not activated by the cation of calcium. Calcium and magnesium ions, however, may affect not only the rate of utilization of high-energy compounds, but also the rate of their formation. For instance, calcium ions, which cause swelling of the mitochondria, dissociate respiration from its accompanying phosphorylation [5-7], while magnesium ions oppose this dissociating action of calcium ions [7]. Moreover, the products of decomposition of adenosinetriphosphate, intensified as a result of the stimulation of adenosinetriphosphatase by magnesium ions, are the source of material for the regeneration of high-energy compounds.

We have investigated the effect of toxic doses of calcium and magnesium salts on the rate of development of rigor mortis after decapitation of animals; this rate may be used as an indicator of the negative balance between the intensities of synthesis and utilization of high-energy compounds [1-3].

EXPERIMENTAL METHOD

Experiments were carried out on 50 adult male rabbits. Calcium chloride was injected intraperitoneally into 20 rats as a 5% solution in a dose of 1 mg/g body weight. In a special series of experiments (without subsequent decapitation) these doses caused intensive convulsions after approximately 15 min, and death of the animals within 40 min. In the experimental group of animals decapitation was carried out 5-10 min after injection of the compound, when no toxic manifestations were visible. Since convulsions, by altering the balance of high-energy compounds towards the negative side, hasten the development of rigor mortis considerably, decapitation before the onset of convulsions caused by calcium chloride poisoning prevented them from affecting the rate of development of rigor mortis.

Magnesium sulfate was injected intraperitoneally into 10 rats as a 20% solution in a dose of 6 mg/g body weight. In association with the immediate onset of narcosis, feeble convulsions were observed in 7 of the 10 rats (possibly due to asphyxia). The animals were decapitated 2 min after injection of the salt solution.

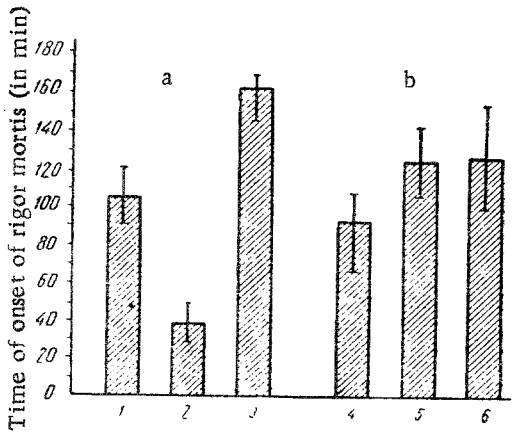
The control group consisted of 20 rats decapitated without preliminary injection of any form of medication.

It should be noted that the convulsions in the animals receiving calcium chloride, which always developed after decapitation, were just as severe as those in the controls; in the rats receiving magnesium sulfate the convulsions were not quite so severe.

After decapitation of the animals, recordings were made of rigor mortis in the tail muscles [3].

EXPERIMENTAL RESULTS

Injection of calcium chloride hastened the development of rigor mortis despite the absence of visible toxic manifestations, while after administration of magnesium sulfate rigor mortis was retarded, although in most animals convulsions were observed (see figure, a). Hence, the changes in the time of rigor mortis in these experiments were most probably dependent on the effect of these salts on the skeletal muscles (on the metabolism of their high-energy compounds), and not on the central nervous system or other organs.



Effect of calcium and magnesium salts (a) and sodium salts (b) on the rate of development of rigor mortis. 1) Control; 2) after injection of calcium chloride; 3) after injection of magnesium sulfate; 4) control; 5) after injection of sodium sulfate; 6) after injection of sodium chloride.

In order to elucidate the role of the sulfate anion, a special series of experiments was carried out to study its effect on the rate of development of rigor mortis. In these experiments sodium sulfate was used; like magnesium sulfate, this forms calcium sulfate, i.e., it de-ionizes calcium. Furthermore, in the first experiments the salts were injected as hypertonic solutions, so that in the experiments now undertaken a hypertonic solution of sodium chloride was injected. Sodium sulfate and chloride were injected in isomolar doses.

Experiments were carried out on 30 adult male rats. Sodium sulfate was injected intraperitoneally into 10 rats as a 25% solution in a dose of 2 mg/g body weight, and sodium chloride into 10 rats as a 6% solution in a dose of 1.0-1.5 mg/g body weight. Decapitation was carried out 5 min after injection of the salt (no toxic manifestations were observed before decapitation). Ten rats acted as the control group. The convulsions arising in the control and experimental animals immediately after decapitation were equal in intensity.

Injection of both sodium chloride and sodium sulfate caused a statistically significant delay in the onset of rigor mortis (see figure, b), although to a much less degree than after injection of magnesium sulfate. It could be concluded, therefore, that the cause of the more marked slowing of rigor mortis after poisoning with magnesium sulfate was an increase in the concentration of magnesium ions in the blood and tissues.

The fact that the degree of slowing of rigor mortis was equal after injection of sodium sulfate and sodium chloride, and that after administration of calcium chloride its onset was actually hastened, suggests that the anionic portion of the molecules of the tested salts had no significant effect on the time of development of rigor mortis.

The hypertonicity of the injected solutions could hardly be significant, for although all the solutions used were in fact hypertonic, they affected the rate of development of rigor mortis in different (and sometimes opposite) ways.

SUMMARY

In experiments on rats it was found that toxic doses of calcium chloride considerably accelerated, while magnesium sulfate inhibited, the development of rigor mortis. These effects are connected with the capacity of the calcium ion to disturb respiratory phosphorylation, and of the magnesium ions to promote it.

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