

EFFECT OF CALCIUM CHLORIDE AND MAGNESIUM SULFITE ON THE RATE OF RIGOR MORTIS

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According to the data of recent biochemical investigations (47, three different adenosinetriphosphatases can be distinguished in the skeletal musculature: (1) myofibrillary, or actomyosinic, activated by the cations magnesium and calcium; (2) sarcoplasmic, which is activated by the cation magnesium and inhibited by the cation calcium; (3) mitochondrial, activated by the cation magnesium and not activated by the cation calcium. Calcium and magnesium ions can however act not only on the rate of utilization of energy-rich (macroergic) compounds, but also on the rate of their formation. Thus, calcium ions inducing swelling of mitochondria disengage respiration from the coupled phosphorylation ( , while magnesium ions counteract this disengaging effect of calcium ions with In addition, products of adenosinetriphosphoric acid breakdown, intensified due to stimulation of adenosinetriphosphatase by magnesium ions is a source of de novo formation of macroergs.

We investigated the effect of toxic doses of calcium and magnesium salts on the rate of rigor mortis after decapitation of animals withis rate can serve as an index of the negative balance between the intensity of synthesis and the use of macroergic compounds (1).

## Experimental Methods

The experiments were performed on 50 adult male rats. Calcium chloride was introduced intraparenterally to 20 rats in a 5 % solution on the basis of 1 mg per 1 gram of bodyweight. In the special series of experiments (without subsequent decapitation) these doses induce in approximately 15 minutes intense seizures, and in 40 minutes -- the death of the animals. In the experimental group of animals decapitation was carried out in 5-10 minutes after administration of the preparation in the absence of visible symptoms of intoxication. Since seizures shifting the macroergic balance to the negative side considerably accelerated the onset of rigor mortis, and decapitation before the onset of the seizures caused by calcium chloride intoxication allowed us to exclude their effect on the rate of onset of rigor mortis.

Magnesium sulfite was given 10 rats intraparenterally in a 20 % solution on the basis of 6 mg per gram of bodyweight. Against a background of the immediately developing narcosis, in 7 out of 10 rats, weakly pronounced seizures were, however, observed (it is possible that these were of an asphyctic character). Decapitation was performed two minutes after administration of the solution salt.

The control group numbered 20 rats, in which decapitation was carried out without preliminary administration of any agents.

We must note that in animals receiving calcium chloride the seizures which always followed decapitation were as pronounced as in the control group; the seizures were somewhat attenuated in rats given magnesium sulfite.

Following decapitation of the animals, the rigor mortis of the tail musculature was recorded (3).

## Experimental Results

The administration of calcium chloride significantly accelerated rigor mortis, in spite of the absence of visible symptoms of intoxication, and when magnesium sulfite was used rigor mortis was slowed down, although in most animals seizures were observed (cf Figure, a).

Therefore, the change in the rates of termination in these experiments most probably depend on the effect of the above salts on skeletal muscles (metabolism of macroergic compounds therein) and not on the central nervous system or other organs. - 3

To clarify the role of the sulfate anion, its effect on the rate of rigor mortis was studied in a special series of experiments. In these experiments, sodium sulfate was used, which as true also for magnesium sulfate, forms calcium sulfate, that is, calcium is reduced. In addition, in these experiments, the salts were introduced in hypertonic solutions, therefore experiments were performed in which a hypertonic solution of sodium chloride was given. Sodium sulfate and chloride were given in isomolar quantities.

The experiments were performed on 30 adult male rats. Sodium sulfate was intraparenterally administered to 10 rats in a 25 % solution on the basis of 2 mg per 1 gram of bodyweight, and sodium chloride -- to 10 rats in a 6 % solution on the basis of 1-1.5 mg per 1 gram of bodyweight. Decapitation was performed 5 minutes after administration of the salts (no signs of intoxication were observed before decapitation). Ten rats served as the control group. We must note that the seizures arising immediately after decapitation in the control and in the experimental animals were equally intense.



Effect of calcium and magnesium salts (a) and sodium salts (b) on the rate of rigor mortis. 1 - control; 2 - after administration of calcium chloride; 3 - after administration of magnesium sulfate; 4 - control; 5 - after administration of sodium sulfate; 0 - after administration of sodium chloride.

LEGEND: a) duration of rigor mortis (in minutes).

The administration of both sodium chloride and sodium sulfate gives rise to statistically reliable retardation of rigor mortis (of Figure, b). However, the extent of retardation of rigor mortis in these cases is considerably less than when magnesium sulfate is given. Therefore, the cause of the more decided retardation of rigor mortis in magnesium sulfate poisoning must be the increased concentration of the ions of the latter in blood and tissues.

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MON 8 - 1360 MON 8 - 1360 The fact that the extent of retardation of rigor mortes is the same when both sodium sulfate and sodium chloride ar given, and when calcium chloride is used, rigor mortis is even accelerated, allows us to suggest that the anionic portion of the molecule of the salt tested has no essential effect on the duration of rigor mortis.

There is hardly any substantial importance to the hypertonicity of solutions administered, since in spite of the fact that all the solutions used were hypertonic, they can affect the rate of rigor mortis in dissimilar ways and even with an opposite effect.

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