We shall give here no details, but state the results in the briefest possible way.

The average quantity of each salt per kilo which caused death, was as follows: $MgCl_2$, 2.35 c.c.; $CaCl_2$, 4 c.c.; KCl, 6.23 c.c., and NaCl, 63.24 c.c. These figures refer to the crystalline salts when dissolved in molecular solution. When, however, these values are reduced to that of the anhydrous salts, the figures read as follows: The fatal dose of magnesium chloride is 0.223 gram per kilo (dog); of calcium chloride it is 0.444; of potassium chloride 0.464; and sodium chloride is fatal only when 3.7 grams of the salt are given per kilo. In other words, magnesium chloride is twice as toxic as calcium chloride or potassium chloride. Again, potassium chloride is about eight times more toxic than sodium chloride. In the case of the latter we have to remember that the solution ran in twice as rapidly as the solutions of the other salts, which means that the comparative toxicity of sodium chloride is even much less than appears in the above scale.

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The action of calcium upon the pupil and its relation to the effects of mydriatics.

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The intravenous injection of calcium exerts a pronounced effect upon the mechanism of pupilary constriction. The solution employed was $m/8$ $CaCl_2$; this was injected slowly through the external jugular vein of rabbits; in some instances the ear veins were used. After 12 to 14 c.c. of the solution had run in, stimulation of the cervical sympathetic nerves no longer caused dilatation of the pupil. At the same time, the pupil appeared to be smaller than normal and reacted less readily to light. When 20 to 25 c.c. had entered, the pupils, as a rule, became almost pinpoint in size. If the infusion was now stopped, the pupils remained contracted for about thirty minutes, and usually about two hours elapsed before the pupils again reached their normal size. There

were some exceptions in which a larger dose had to be infused before the pupil showed a distinct reaction to the calcium, and then the contraction did not become maximal. These exceptions seemed to occur when the rabbits were under profound ether anesthesia.

Atropin did not prevent this calcium myosis, but it retarded somewhat the onset of this myosis and hastened its disappearance after stoppage of the calcium infusion. Instillations of atropin into the conjunctival sac were a little more effective than intravenous injections. As far as we know calcium is the only substance which is able to overcome the full mydriatic effect of atropin.

What was said of atropin holds good, in general, also for cocaine; calcium overcomes the mydriatic effect of cocaine, but to a less extent than that of atropin. The mydriatic effect of the cocaine becomes especially active during the onset and the later period of the calcium myosis.

Ether antagonizes moderately the calcium myosis. This is especially well seen when a few c.c. of ether are injected subcutaneously after a calcium infusion. The contracted pupils of the rabbit now dilate fairly rapidly. As already mentioned, the calcium myosis develops less readily when the animal is deeply under ether.

The action of adrenalin in animals whose superior cervical sympathetic ganglion has been removed, is interesting. In these rabbits, as is now well known, the instillation or subcutaneous, intramuscular, or intravenous injection of adrenalin causes a strong, long lasting dilatation of the pupil on that side where the ganglion was removed. Calcium overcomes this dilatation also, but the mydriatic adrenalin-effect appears in a striking manner when the calcium infusion is discontinued. While the pupil on the side with intact ganglion remains strongly contracted, the pupil on the operated side becomes very wide and remains so for hours.

We have finally to mention that asphyxia also opposes moderately the myosis brought on by calcium.

We shall not enter here into a discussion of the intricate problem involved in the mechanism of calcium myosis; our present assumption is that this calcium myosis is produced by a stimulation and contraction of the muscle fibers in the pupilary sphincter.