of strong tonic contraction. Since the continuous insufflation washes out a good part of the normal content of CO_2 and since the effect and the after-effect of the insufflation practically consist at all times in a tonic contraction of either the inspiratory or the expiratory muscles, the conclusion seems warranted that a *reduction of CO_2 in the blood does not act as a reduction of a stimulus below the threshold value but, on the contrary, it serves as a stimulus for the production of a tonic contraction of the respiratory muscles, while the addition of CO_2 assists in the maintenance of the respiratory rhythm.*

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On the production of hyperglycæmia and glycosuria by magnesium salts.

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In their experiments on the action of magnesium salts, Meltzer and Auer observed that after subcutaneous injections of magnesium sulphate the urine of rabbits contains a reducing substance. Underhill and Closson, who later noticed the presence of hyperglycæmia after an intravenous injection of magnesium sulphate, ascribed the hyperglycæmia to the asphyxia which the magnesium salts produced in their experiment.

In a series of experiments which we have recently carried out on dogs, all the animals had from the beginning to the end of the experiment either intratracheal insufflation or the usual artificial respiration. The occurrence of asphyxia was thus excluded. The operative part was done under local anesthesia. In most of the experiments an $M/4$ solution of magnesium sulphate was injected intravenously. There was a considerable increase of the sugar content of the blood after the infusion practically in all experiments. In most cases the original glycæmia did not exceed 0.13 per cent., while at the end of the injection or some time later, the sugar content of the blood was often as high as 0.4 per cent. and even higher. In blood taken about an hour and a half after the end of

an injection the glycæmia was found often to have dropped to the original content.

There can be no doubt that magnesium sulphate produces considerable hyperglycæmia which is not due to asphyxia; it is produced in some way specifically by the magnesium salt. Sodium sulphate does not affect the normal glycæmia.

It is a noteworthy fact that the glycosuria was very little marked and far under proportion to the hyperglycæmia. Glycosuria was often entirely absent and when present it never reached even ½ per cent. The intravenous injection of magnesium sulphate also produced very little diuresis.