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Reflex inhibition of the cardia in rabbits by stimulation of the central end of the vagus.

By **S. J. MELTZER** and **JOHN AUER.**

[*From the Rockefeller Institute for Medical Research.*]

At the last meeting of this Society[1] we reported that by stimulation of the central end of the vagus a tetanic contraction of the entire esophagus can be produced in dogs and cats but not in rabbits. We wish to report now that in continuation of these studies we found that *stimulation of the central end of the vagus causes a distinct inhibition of the cardia in rabbits.* The cardia of the rabbit is normally contracted in a moderate degree. Furthermore at each deglutition the peristaltic movements of the esophagus terminate in a characteristic contraction of the cardia — it sinks into the stomach. Finally after a stimulation of the peripheral end of the vagus the cardia contracts in the same characteristic way. We found that these three states of contraction can be definitely inhibited by a stimulation of the central end of the vagus. In the first place the cardia relaxes — bulges up during such stimulation. In the second place if deglutition occurs the cardia never contracts so long as the central end of the vagus is being stimulated. Finally the interruption of the stimulation of the peripheral end of the vagus does not bring on a contraction of the cardia if during this time a stimulation of the central end is going on.

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Continuous anesthesia by subcutaneous injection of magnesium sulphate in nephrectomized animals.

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In the paper dealing with the anesthesia produced in animals by subcutaneous injection of magnesium salts Meltzer and Auer stated that animals which urinated frequently had the better chance for recovery, and that urination probably carries off some of the

[1] *Proceedings of this Society*, 1905–'06, iii, p. 74.

salt and prevents its fatal accumulation in the blood. On the basis of this assumption a series of experiments were carried out in which the anesthetic effects of subcutaneous injections of magnesium sulphate were studied in nephrectomized rabbits. The results briefly stated were as follows:

A dose of 0.8 gram of the salt per kilo of rabbit is sufficient to put the animal within a short time into deep anesthesia. This is less than half the dose that is required to anesthetize a normal rabbit. Furthermore, the nephrectomized rabbits thus anesthetized remained in a more or less comatose, paralyzed state until death, which did not occur earlier than in the control nephrectomized animal; in other words the animals remained in a state of anesthesia lasting sometimes two days and longer. Frequently the animals recovered slightly some hours after the injection, to sink soon again, however, into a deep stupor which lasted until death. The described effect was the same whether the above mentioned quantity of salt was given in one dose or was administered in fractions.

These facts demonstrate that elimination of the injected magnesium salt occurs mainly through the kidneys and that the elimination begins pretty soon after the injection. Hence when the kidneys are absent and practically no elimination takes place a smaller dose is sufficient to bring on the anesthesia, and it makes no difference whether the quantity is given at once or in small doses at varying intervals. Furthermore, the anesthesia is long lasting and continuous, as the salt cannot leave the body.

These results are especially interesting as they are in sharp contrast to the behavior of strychnin in nephrectomized animals. According to the experiments of Salant and Meltzer the toxic and the fatal doses of strychnin are the same for nephrectomized rabbits as for normal ones. Furthermore, when strychnin is given in subminimum doses nephrectomized animals can stand as much as three times the lethal dose. Finally the animal either dies from the effects of strychnin or recovers completely; a continuous long lasting convulsive state never occurs after any dose of strychnin in nephrectomized animals.