The experiments would seem to indicate that there exists a coördination between the contractions of the ventricles and the auricles of such a nature that increased systole of the former causes an increased systole of the latter without any apparent change in venous pressure.

To this condition is ascribed the cause of auricular cohypertrophy.

10 (535)

The origin of convulsions and paralysis following the intravenous injections of the hypertonic solution of sodium chloride.

By **DON R. JOSEPH** and **S. J. MELTZER.**

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In our experiments on the comparative toxicity of chlorides in which, among others, we studied the effects of intravenous injections of a molecular solution of sodium chloride in dogs we observed that after a certain quantity of the solution runs in, twitchings of the muscles of the entire body begin which gradually develop into more or less strong convulsions. Later these gradually grow weaker and finally subside completely, at which time also the respiration stops. The heart continues to beat for several minutes longer. This chain of events is ascribed by pharmacologists to the osmotic action of the hypertonic solution and is generally termed salt action. Loeb however demonstrated that sodium chloride exerts on the living tissue a chemical action also. In the paper embodying the above mentioned experiments we made the following two suggestions: (1) that the twitchings and convulsions are perhaps comparable to the twitching of frog muscles which develop when they are immersed in solutions of sodium chloride; the convulsive movements would be then of peripheral origin; (2) that the subsidence of the convulsions and the paralysis might be due to the curare-like action of the sodium chloride, *i. e.*, to the paralysis of the motor nerve endings.

In the present series of experiments we have tested these two hypotheses. The second hypothesis was tested by investigating

the irritability of the peripheral end of the sciatic nerve during the entire course of the experiment. It was found in every experiment that stimulation of the sciatic nerve even at the time of complete paralysis of respiration did not fail to elicit a definite motor response. This disposes of the second hypothesis; the paralysis is surely not due to a curare-like action of the sodium chloride. The first hypothesis we have tested in various ways. It is known that the twitchings of frog muscles in sodium chloride subside when calcium is added to the solution. We have therefore tried to introduce at some stage of the experiment solutions of calcium chloride into the circulation. In none of these experiments were the twitchings or the convulsions affected in any way by the addition of the calcium solution. Furthermore when the sciatic nerve was cut on one side the muscles innervated by this nerve did not take part in the twitching and convulsions. This fact was more strikingly demonstrated in experiments in which the lower half of the spinal cord was removed. In these cases the contrast between the convulsing upper half and the paralyzed lower half of the animal body was striking indeed.

It is therefore evident that the convulsions and paralysis caused by hypertonic solutions of sodium chloride have their origin neither in the muscles nor in the peripheral nerves; they originate in the spinal cord.

We may append here the brief remark that the convulsions under discussion can be greatly inhibited by intravenous injection of a non-fatal dose of potassium cyanide. We were stimulated to this latter observation by the known experiments of Loeb on the action of cyanide upon the fertilized and non-fertilized sea-urchin eggs.

11 (536)

Simultaneous graphic registration of gastric and duodenal peristalsis in rabbits; a demonstration.

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The graphic registration of gastric or intestinal peristalsis is usually obtained from an animal with an opened abdomen while