

The method as now modified has been successfully applied to human, dog, ox, rabbit, guinea pig and pigeon blood.

TABLE II.

Table for the conversion of c.c. of N/100  $\text{Na}_2\text{S}_2\text{O}_3$  into mg. of glucose per c.c.

0.01N $\text{Na}_2\text{S}_2\text{O}_3$ c.c.	Glucose mg. per c.c.	0.01N $\text{Na}_2\text{S}_2\text{O}_3$ c.c.	Glucose mg. per c.c.
0.50	0.25	4.0	1.19
1.0	0.39	4.5	1.33
1.5	0.52	5.0	1.46
2.0	0.66	5.5	1.59
2.5	0.79	6.0	1.73
3.0	0.92	6.5	1.76
3.5	1.06	7.0	2.00

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**On the nature of the rhythmic contractions in the stomach and intestine.**

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Rhythmic contractions in the stomach and intestine persist following the administration of nicotin in doses sufficient to prevent conduction through synapses. Attempts to account for these contractions solely as responses to nervous impulses have resulted in confusion. Certain experimental data recorded by Magnus ('05),<sup>1</sup> Gunn and Underhill ('14),<sup>2</sup> and Alvarez and Mahoney ('22)<sup>3</sup> indicate clearly that excised pieces of the intestinal musculature may execute rhythmic contractions in the absence of nervous influences. The present paper embodies a preliminary statement of the results of a further investigation, through the use of nicotin in massive doses, of the rhythmic

<sup>1</sup> Mangus, R., *Arch. f. d. gesammt. Physiol.*, 1905, cviii, 1.

<sup>2</sup> Gunn, J. A. and Underhill, S. W. F., *Quart. Jour. Exp. Physiol.*, 1914, viii, 275.

<sup>3</sup> Alvarez, W. C. and Mahoney, L. J., *Amer. Jour. Physiol.*, 1922, lix, 421.

contractions both in the stomach and intestine in intact animals (dog).

Nicotin hydrochloride was administered intravenously in successive gradually increasing doses. Artificial respiration was employed as early as necessary and throughout the rest of the experiment. Segmental contractions in the small intestine, peristalsis in the stomach, rhythmic contractions of the pyloric sphincter, and antiperistalsis in the large intestine continued after the administration of sufficient nicotin to abolish all responses either to electrical stimulation of postganglionic sympathetic fibers or the administration of adrenalin in relatively large doses (0.01 to 0.025 mg. per kg. of body weight). At this time the administration of adrenalin brought about no change in blood pressure. It was found necessary, in order to secure these results, to administer approximately three grams of nicotin hydrochloride per kilogram of body weight. If now sufficient time was allowed without the administration of more nicotin the conducting mechanism recovered to such an extent that responses were again elicited by electrical stimulation of postganglionic sympathetic fibers or the administration of adrenalin in moderate doses.

The failure of relatively large doses of adrenalin, following the administration of nicotin in the quantities stated above, to bring about inhibition of the rhythmic contractions in the stomach and intestine indicates at least a very great reduction in the irritability of the myoneural junction. The failure of electrical stimulation of postganglionic sympathetic fibers to bring about any modification of these contractions indicates paralysis of the nerve fibers or the myoneural junction or both. Therefore, we may assume that the rhythmic contractions in the stomach, pyloric sphincter, and the small and large intestine which continue when this stage in the nicotin paralysis is reached do so in the absence of nervous influences. Consequently, the capacity to execute rhythmic contractions is inherent in the gastro-intestinal musculature. Furthermore, the rhythmic contractions above indicated in the several parts of the gastro-intestinal canal probably belong to the same category of functional motility.

The results of these experiments do not demonstrate the absence of nervous control, under normal conditions, of the rhythmic contractions in any part of the gastro-intestinal canal. The tracings recorded throughout the progress of any successful ex-

periment in our series indicate progressive changes in the configuration and amplitude of the contractions as the nervous paralysis brought about by the nicotin advances. These tracings will be analyzed in detail in a later paper. A few of the more striking changes may be stated as follows: Both in the stomach and intestine, before the administration of nicotin, the rhythmic contractions vary greatly in amplitude and are commonly superimposed on large tone changes. As the nicotin paralysis advances the tone changes subside and the amplitude of the contractions, while becoming more uniform, diminishes greatly until a stage is reached at which apparently certain inhibitory influences are removed and the amplitude of the contractions increases suddenly and markedly. After this the rhythmic contractions continue with a high degree of regularity. As the nicotin paralysis is carried still farther the amplitude of the contractions gradually decreases but is still relatively large when electrical stimulation of postganglionic sympathetic fibers is no longer effective. If the administration of nicotin is discontinued at this point the rhythmic contractions continue without marked changes in amplitude.

We do not maintain that the tracings recorded before the administration of nicotin in these experiments represent the exact configuration of the contractions going on in the stomach and intestine under normal conditions. Nevertheless, we believe that the changes which occur in the tracings during the progress of the nicotin paralysis represent actual changes in the muscular activity which are due largely to the elimination of nervous influences. Doubtless, rhythmic contractions in the gastro-intestinal canal are normally subject, in a large degree, to nervous control.