tion of lytic principle to the microorganisms. Various strains of human pathogenic streptococci chosen for this experiment were subcultured every 24 hours in broth containing 1:10 dilution of lytic principle. After several such passages the cultures were inoculated into flasks of broth, incubated for 24 hours, and 0.5 cc. of each of these filtrates tested against homologous normal cultures of streptococci. Strains of *erysipelas streptococci* "trained" in Clark and Clark's phage acquired the property of regenerating a powerful lytic principle against normal cultures of these streptococci. The lytic principle thus obtained corresponded fully to the classical bacteriophage in every detail. This principle also showed a remarkable specificity. Sixteen strains of *streptococcus erysipelatis*, out of 21 tested, were lysed in dilution $1x10^{-8}$ cc. by this phage. Sixty-four strains from other sources remained entirely unaffected by *streptococcus erysipelas* phage. The method failed to procure a phage against other pathogenic streptococci.

[1] Piorkowski, G., *Med. Klin.*, 1922, xviii, 474.

[2] Dutton, L. O., *J. Inf. Dis.*, 1926, xxxix, 48.

[3] Hadley, P., and Dabney, E., PROC. SOC. EXP. BIOL. AND MED., 1926, xxiv, 13.

[4] Clark, P. I., and Clark, A. S., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 635.

## 3688

### Gastro-Intestinal Motor Response to Vagus Stimulation after Nicotine.

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While studying the effect of massive doses of nicotine on the enteric nervous mechanism of dogs, Thomas and Kuntz[1] stimulated the vagi after giving large doses of nicotine. The response of the intestine was not abolished by these large doses of nicotine, but, on the contrary, were greater and more constant than before nicotine was administered. This was apparently contrary to the generally accepted conclusion of Bayliss and Starling.[2]

The present report is based on 28 experiments on cats, 19 females and 9 males, weighing from 2.0 to 4.1 kilograms. They were narcotized with ether, and the brain and spinal cord destroyed by pithing. Artificial respiration was administered through a tracheotomy

tube. Both vagi were exposed, but only the right was electrically stimulated after a primary testing of the effect of both, Fig. 1. A

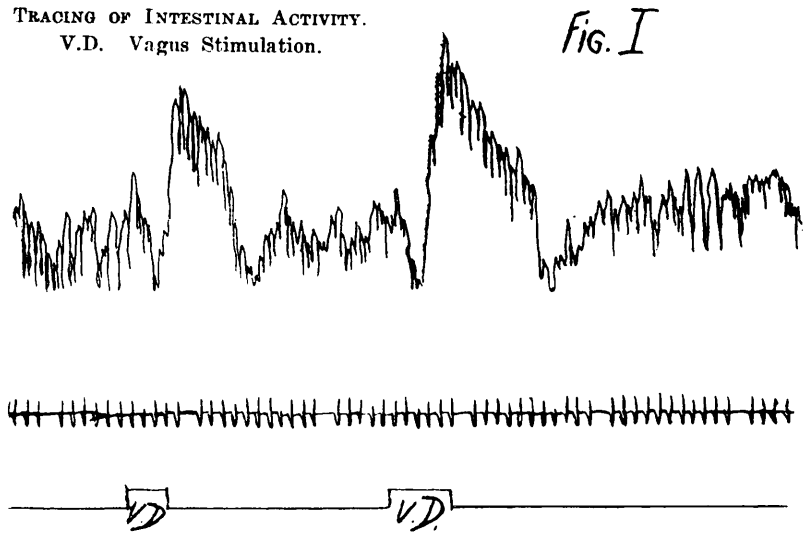


FIG. 1.

Cat. Balloon water manometer record of intestinal activity. Tracing shows effect of vagus stimulation before administration of nicotine.

rubber balloon was inserted into the intestine disto proximally, and a record of the activity obtained by means of a water manometer. The nicotine used was made up as a 5 per cent solution by weight of the alkaloid in 0.9 per cent saline and neutralized to pH 7.4 with HCl. Nicotine salicylate was used in 3 experiments. The solution was injected into the femoral vein.

Three series of experiments were done as follows: (a) 11 experiments with same procedure as described above. (b) 9 experiments with both adrenals removed. (c) 8 experiments with both adrenals and semi-lunar ganglia extirpated.

As the results were essentially the same, whether the adrenals and semi-lunar ganglia were present or removed, they will be presented together.

Nicotine given intravenously causes a cessation of motility and a drop in tone of the cat's intestine. If the vagus response was good before the injection of the nicotine, it invariably disappeared with the first injection, which was always between 1 to 10 mg. per kilo of body weight, Fig. 2. Subsequent injections had no further effect upon motility, tone or reaction to vagus stimulation of the intestine, until a total amount of 20 to 60 mg. of nicotine per kilo had been injected, (Fig. 3).

Fig. II

TRACING OF INTESTINAL ACTIVITY.
(1) and (2) 1st and 2nd injections of nicotine to 10 mg./Kg.
V.D. Vagus Stimulation.

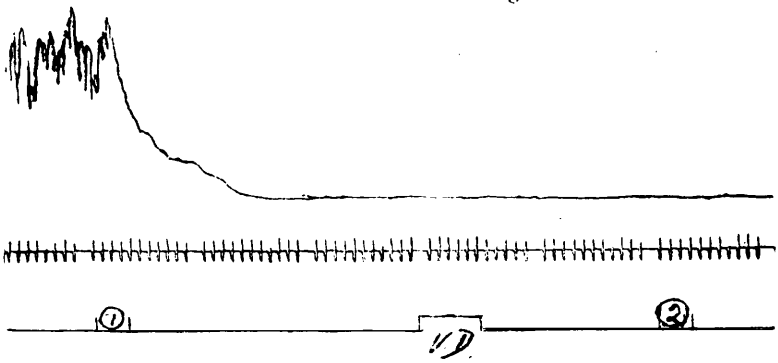


FIG. 2.

Same animal as in Fig. 1. Tracing shows cessation of intestinal activity with loss of vagus response and inocuousness of nicotine injection at (2), following the initial intravenous injection of nicotine at (1).

Fig. III

TRACING OF INTESTINAL ACTIVITY.
(4) 4th injection of nicotine; total now 40 mg./Kg.
V.D. Vagus Stimulation.

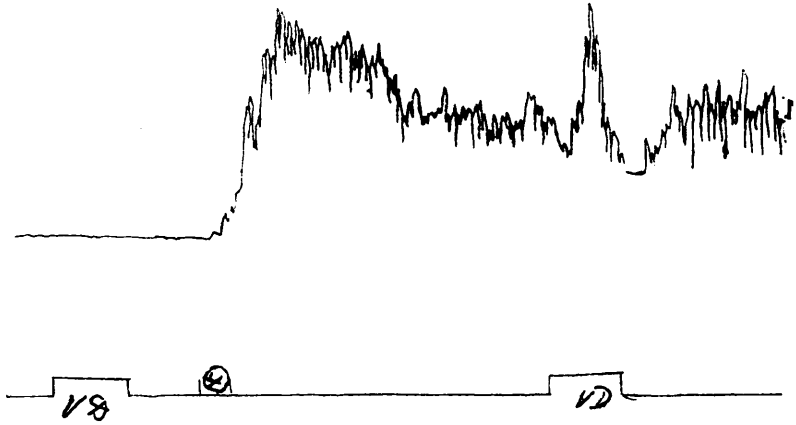


FIG. 3.

Same animal as in Fig. 2. Tracing shows continued loss of vagus response thirty-five minutes after the first injection (Fig. 2 (1) ) as above. It shows stimulation of the intestine at (4), forty-two minutes after the first injection; as well as a return of vagus response a few minutes later.

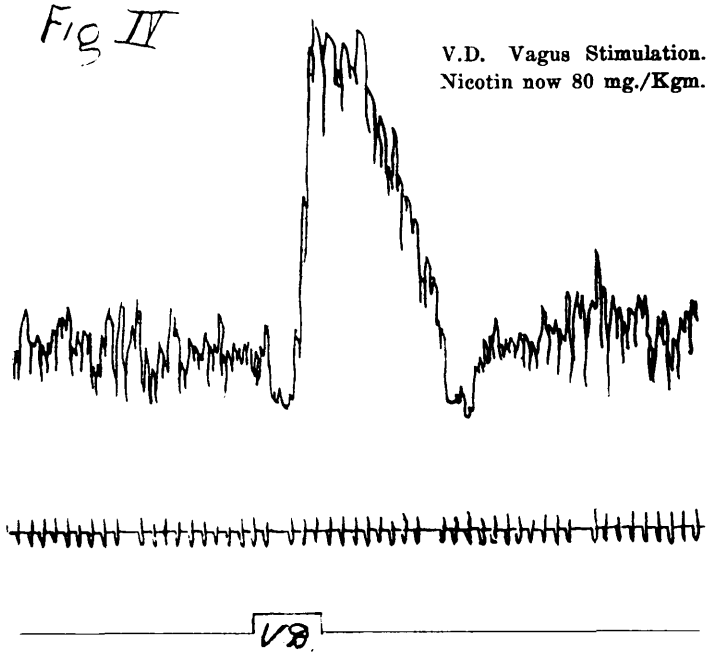


FIG. 4.

The same animal as in Fig. 3. Tracing shows exaggerated response to vagus stimulation after 80 mg. of nicotine per kilo had been given.

When this latter concentration was reached, the injection of nicotine was followed by an increase in intestinal tone and activity. These increased in strength and duration as the total amount injected became greater, though the individual doses and the time interval between them remained approximately the same, Fig. 4.

When the concentration, above described, was reached, there was also a return to vagus response, which became greater with each injection. Finally a point was reached (50 to 100 mg. of nicotine per kilo) when the vagus response was greater and of longer duration than the original one previous to the first nicotine injection, Fig. 4. In every case where the primary response to vagus stimulation was good we obtained a return of the activity after the higher concentration of nicotine had been injected. There was no primary response to vagus stimulation, throughout the experiments, though the response to nicotine described in the preceding paragraph was always present.

For the theoretical discussion of the results, the reader is referred to the paper by Thomas and Kuntz.[1]

The conclusions seem justified that:

(1) All effects of the vagus on the intestine of the cat are com-

pletely and permanently abolished by doses of nicotine from 1 to 15 mg. per kilo of body weight.

(2) These doses depress the intestine, as shown by the drop in tone, and the cessation of activity of that organ.

(3) Doses larger than 15 to 60 mg. per kilo of body weight brought about a return of the previous activity and a response to vagus stimulation, whether the drugs were given in one or divided doses.

(4) Neither the splanchnic ganglia nor the adrenals are responsible for the depression of the intestine or for the loss to vagus stimulation following the small doses of nicotine, because their removal causes no change in the reaction of the nicotine.

[1] Thomas, J. E., and Kuntz, A., *Am. J. Physiol.*, 1926, lxxvi, 598.

[2] Bayliss, W. M., and Starling, E. H., *J. Physiol*, 1899, xxiv, 99.

## 3689

## Formation of Lactic Acid in Excised Organs.

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These experiments were undertaken to determine whether excised tissues would form lactic acid under anaerobic conditions. Paired organs of dogs were studied. One of each pair was rapidly triturated in chilled alcohol to prevent post-mortem glycolysis. The other was triturated in Ringer's or buffered phosphate solution (pH

TABLE I.

*Lactic Acid in Paired Organs Before and After Incubation.*

| Dog No. | Organ | Lactic Acid Per Cent | |
|---|---|---|---|
| | | Before | After |
| 59 | Testis | 0.017 | 0.25 |
| 27 | " | 0.037 | 0.22 |
| 141 | " | 0.006 | 0.18 |
| 59 | Kidney | 0.054 | 0.32 |
| 27 | " | 0.068 | 0.20 |
| 141 | " | 0.029 | 0.15 |
| 59 | Parotid | 0.051 | 0.14 |
| 27 | Thyroid | 0.075 | 0.16 |
| 141 | " | 0.028 | 0.090 |
| 27 | Cerebrum | 0.118 | 0.196 |
| 59 | Submaxillary | 0.086 | 0.178 |