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A Factor Condition-ing the Inhibitory Response of the Intestinal Motility to the Peripheral Stimulation of the Cervical Vagus Nerves

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Abstract

Recording the motility of the stomach as well as the small intestine by the balloon method in the dogs decerebrated and unanesthetized, we found a factor conditioning the inhibitory effect of the intestinal motility to the stimulation of the perpheral cut-end of cervical vagus nerves. The results may be summarized as follows: (1) The stimulation of the peripheral cut-end of the cervical vagus nerve frequently produces the yarious patterns and degrees of inhibition of the intestinal motility of the stomach as well as of the small intestine. (2) The inhibitory effect still appears after the severing of the vagus nerves at the caudal end of the esophagus, but is obliterated and reversed to the augmentory when the splanchnic nerves are bilaterally severed. (3) The cause of the inhibition is attributable to the strong excitation of the intestinal inhibitory centers brought about by the central stimulating action of the anoxemia resulting from the stimulation of the cervical vagus nerves, and the reversal of the response is due to the peripheral stimulating action of the anoxemia upon the intestinal muscles, its central action being excluded from the action on the intestine by the severing of the splanchnic nerves.

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A FACTOR CONDITIONING THE INHIBITORY RESPONSE OF THE INTESTINAL MOTILITY TO THE PERIPHERAL STIMULATION OF THE CERVICAL VAGUS NERVES

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So far as we can encompass, the available results of studies on the intestinal response to the vagal stimulation are somewhat divergent. Therefore, in an attempt to clarify this situation, recently we have performed certain experiments in dogs[4,8,9], and have arrived at a conclusion that the stimulation of the thoracic vagus nerves always produces the purely excitatory effect upon the intestinal movements in the dog decerebrated and unanesthetized. As for the factors likely to produce the inhibitory effects in vagal stimulations, the followings may be enumerated: (a) the depressant condition of the intestinal muscles due to anesthesia; (b) anoxemia produced in anesthetized animal by stimulating the cervical vagus; and (c) the contraction wave propagated to-wards the region of the intestine with balloon insertion.

As to the inhibitory factor described in (b), it has been, however, recognized that anoxia exerts an inhibitory action peripherally upon the intestinal muscles of the animal anesthetized, whereas it acts excitatorily upon the muscles of the animal unanesthetized. Through the course of the experiments performed for the other purposes we have found that anoxia is also centrally related to the inhibitory response of the intestinal motility to the stimulation of the peripheral cut-end of cervical vagus nerves. The results of the experiments are reported in the following.

METHOD

An alternating current of 50 volts and 60 cycles per seconds was passed through the electrodes attached to the frontoparietal regions of the skull of the unanesthetized animal for 5 to 6 seconds to produce the state

of the shock continually for about 20 minutes. The animal was then decerebrated by means of the technique reported previously[5].

For recording the movements of digestive tract, balloons were inserted into the pyloric region as well as into the duodenum or the jejunum and connected through water manometers to MAREY'S tambours. The peripheral cut-end of the cervical vagus nerves were stimulated by a thyratron stimulator devised by BERNSTEIN[3].

There occurred the cases in which it was occasionally necessary not to take the effects of the extraintestinal nerves into account. The vagus nerves were carefully severed at the caudal end of the esophagus situated just under the diaphragm, while the splanchnic nerves were retroperitoneally severed by the HUKUHARA's method[7].

RESULTS

When the electric stimuli are applied to the peripheral cut-end of the cervical vagus nerves of dogs decerebrated and unanesthetized, the intestinal response is in most cases not purely augmentory but intermingled as usual with inhibitory effects. The modes of the appearance of the inhibitory effects are as follow: in many cases the inhibition follows the transitory augmentory effect, the initial inhibition preceding an occasional augmentation; and the total inhibition may occur only in rare cases. The examples are illustrated in Figure 1.

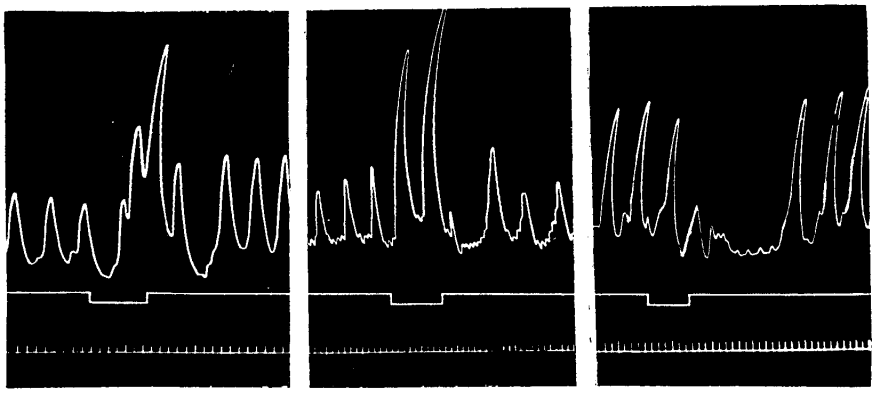


Fig. 1. Various patterns of inhibition of the gastric motility produced by the cervical vagus stimulation.

Splanchnic nerves are on both sides intact. A : initial inhibition succeeded by augmentation. B : inhibition preceded by augmentation. C : total inhibition.

Voltage, 15 volts, duration of each pulse, 2 msec., and frequency, 60/sec. in all cases. Time intervals, 3 sec. each.

In the cases where the stimulation of the peripheral cut-end of the unilateral cervical vagus nerve produces a marked inhibitory effect of the gastric motility, a similar inhibitory effect can generally be obtained as well when the contralateral cervical vagus is severed and stimulated (Fig. 2A). When the major and the minor splanchnic nerves are severed on

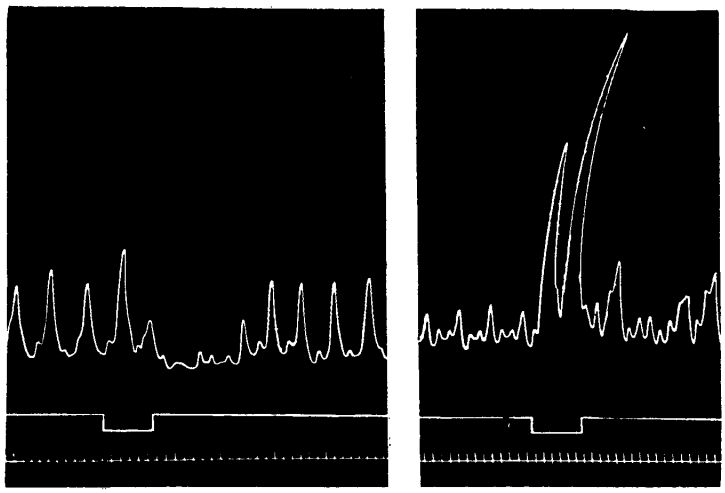
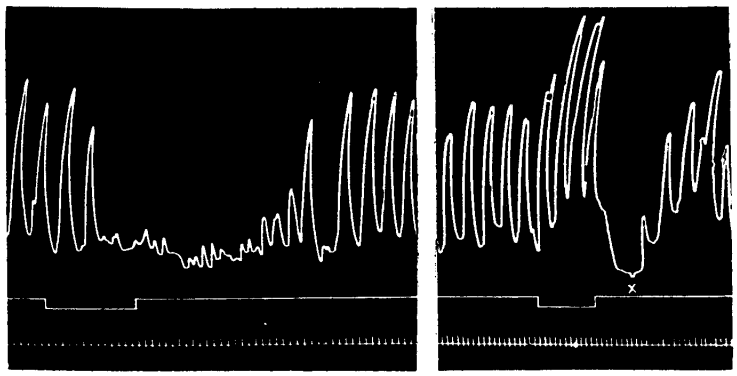


Fig. 2. The effects of the cervical vagus stimulation upon the gastric motility before and after the bilateral splanchnicotomy.
Both cervical vagi are cut. A : stimulation of the right cervical vagus nerve. B : stimulation of the same nerve after splanchnic nerves have been severed on both sides. Note the reversal of the response after splanchnicotomy. Voltage, 9 volts, duration of each pulse 5 msec., and pulse frequency, 55/sec. in both A and B. Time intervals, 3 sec. each.

both sides, the stimulation of the cervical vagus nerves results in the reversal of the response, namely, a marked augmentory response as shown in Fig. 2B.

When, on the other hand, both vagus nerves are severed at the caudal end of the esophagus in order to block the impulses propagated via the vagus nerves to the gastric muscles, the stimulation of the peripheral cut-end of the cervical vagus nerves produces generally the inhibitory response as shown in Fig. 3A, although a transitory augmentation is also occasionally to be seen. When the splanchnic nerves are severed on both sides, the vagal stimulation does no longer produce the inhibitory response but conversely the augmentory (Fig. 3B). As regards the motility of the small intestine the results are similar to those of the experiments related to the gastric motility.



A B

Fig. 3. The effects of the cervical vagus stimulation upon the gastric motility before and after the bilateral splanchnicotomy. Both vagi are severed at the caudal end of the esophagus.

A : An inhibitory effect obtained after the splanchnicotomy.

B : An excitatory effect obtained after the splanchnicotomy.

The depression of the motility marked with 'x' is thought to be due to the exhaution of the gastric musculature.

Voltage, 17 volts, duration of each pulse 2 msec., and pulse frequency, 50/sec. in both A and B. Time intervals, 3 sec. each.

Here we encounter a question, what mechanism might bring about the inhibitory response produced by the stimulation of the cervical vagus nerves. The inhibitory response described above is due certainly neither to the inhibitory fibers supposedly to be contained in the vagus nerves[1,2,7], nor due to reflex mediated by the vagus nerves, because the response can be produced in the cases where both vagi are cut at the caudal end of the esophagus. The intestinal inhibitory reflex which might be produced via the splanchnic nerves is refuted, because the motility is simultaneously inhibited both in the stomach and the small intestine. It may thus be considered that an inhibitory response is due to the circulatory changes which are brought about invariably by the stimulation of the cervical vagus nerves. Of these changes, the anoxemia takes probaly a most important part in producing the inhibitory response. From the results described above it may be considered that anoxemia exerts the influence upon the intestinal motility in the following manners :

Anoxemia produces not only peripherally the excitatory response on the intestinal muscles[4], but also centrally on the intestinal inhibitory centers (probably cell-group from which the splanchnic nerves originate) in the dogs unanesthetized. And the former response is in most cases overcome by the latter, so that the usual response is the inhibitory one. It is

thus a quite natural consequence that the reversal of the response occurs, when the splanchnic nerves are bilaterally severed, in other words, the central effect is eliminated whereas the peripheral effect is left intact.

SUMMARY

Recording the motility of the stomach as well as the small intestine by the balloon method in the dogs decerebrated and unanesthetized, we found a factor conditioning the inhibitory effect of the intestinal motility to the stimulation of the perpheral cut-end of cervical vagus nerves. The results may be summarized as follows: (1) The stimulation of the peripheral cut-end of the cervical vagus nerve frequently produces the various patterns and degrees of inhibition of the intestinal motility of the stomach as well as of the small intestine. (2) The inhibitory effect still appears after the severing of the vagus nerves at the caudal end of the esophagus, but is obliterated and reversed to the augmentory when the splanchnic nerves are bilaterally severed. (3) The cause of the inhibition is attributable to the strong excitation of the intestinal inhibitory centers brought about by the central stimulating action of the anoxemia resulting from the stimulation of the cervical vagus nerves, and the reversal of the response is due to the peripheral stimulating action of the anoxemia upon the intestinal muscles, its central action being excluded from the action on the intestine by the severing of the splanchnic nerves.

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