EFFERENT PATHS OF THE ESOPHAGO-INTESTINAL REFLEX

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We have previously described [1] reflex effects on the motility of the small intestine which can be elicited by stimulation of the receptors of the esophagus.

It was found that the reflex could be produced after sectioning the intestine rostral to the portion from which the recording was made, and this procedure eliminated the possibility of the excitation being transmitted along the walls of the gut; we concluded that the effect must be transmitted through lengthy reflex paths.

A number of studies of the innervation of the esophagus have been made [5-10, 12], and these describe afferent pathways which would account for the reflexes; in the present investigation we have studied the afferent pathways of the esophago-intestinal reflex.

METHOD

Chronically esophagotomized dogs were used. The esophageal receptors were stimulated by the inflation of a thin-walled rubber ballon attached to the nipple of a pipe introduced through the artificial aperture. Intestinal movements were recorded on a kymograph operated by pressure changes in the balloon. In two dogs the reflexes were recorded before and after transdiaphragmal section of the vagi while the splanchnic nerves were left intact; in one dog, recordings were made before and after section of the splanchnic nerves and removal of the lumbar sympathetic chains, while the vagi were intact; in a third experiment, in two dogs, both vagi and splanchnic nerves were cut. In all, 122 experiments were performed. In addition, 48 tests were applied in 16 experiments by injecting 0.15-0.3 mg per kg of atropine in order to eliminate parasympathetic transmission.

RESULTS

In the animals with the nerve supply to the gut intact, the balloon was inflated with air to a volume of 40-50 ml; when the esophagus was relaxed, the pressure required was from 2 to 10 mm, and when contracted, from 8 to 30 mm; when the intestine was relaxed or in a state of mild contraction, the effect was to produce contractions or to increase the strength of any that were already taking place (Fig. 1). When the intestine was already contracting vigorously, inflation of the esophagial balloon had no noticeable effect. In some experiments, the balloon was inflated to 75 ml, which required a pressure of from 5 to 20 mm when it was relaxed, and from 30 to 45 mm when contracted; in others, 100 ml were introduced at a pressure of 10-35 mm when the esophagus was relaxed or of 50-60 mm when contracted; if the intestine had previously been relaxed, tonic contractions now occurred. Inflation up to 75-100 ml caused still more powerful contractions to occur. Inflation to 100-150 ml during

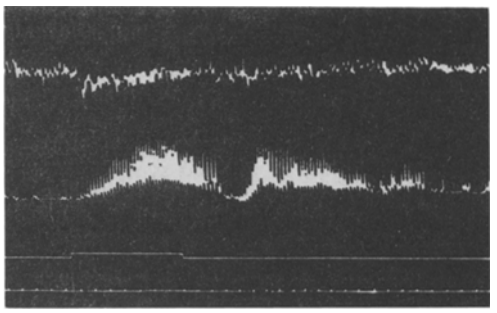

Fig. 1. Reflex excitation of movements of the small intestine caused by inflating a balloon to 50 ml in the esophagus of a dog with intact gastric and intestinal nerve supply. Curve, from above downwards: Gastric contractions; contractions of the jejunum; stimulus marker; time marker (15 sec).

a period of intestinal motility, caused the average amplitude of the movements to be reduced on account of changes in intestinal tone, which, however, could not be attributed to any inhibitory effect.

In dogs treated with atropine, and in whom intestinal movements had ceased, inflating the balloon to 50-75 ml produced no effect (Fig. 2). Because large concentrations of atropine block Auerbach's plexus and other cholinergic systems, it is not entirely justifiable to deduce that the excitatory effect is transmitted only by parasympathetic nerves. We therefore studied the efferent paths after cutting off the nerve supply to the gastrointestinal tract.

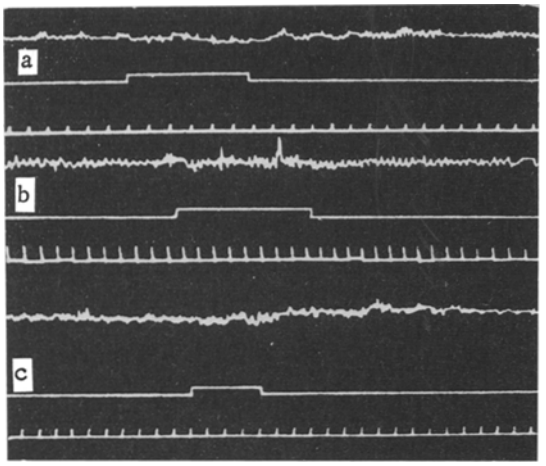

Fig. 2. Absence of reflex excitation of intestinal movements by esophagus. a) In atropinized animal; b) in the dog Bimba after sectioning the vagi; c) ditto in Laddie. Curves, from above downwards: Contractions of jejunum; stimulus marker (75 ml air); time marker (15 sec).

Stimulation of the esophageal mechanoceptors after dividing the splanchnic nerves and removing the lumbar sections of the sympathetic chains produced approximately the same motor response from the small intestine as it did in dogs with an intact nerve supply.

After the vagi had been divided, stimulating the esophagus by inflation to 40-50 or 75-100 ml when the intestine was at rest, caused no contractions in the latter (see Fig. 2). In some experiments, inflating the balloon to 75-100 or 150 ml during the latter third of a rest period caused some reduction in gastric and intestinal tone to occur at the time of inflation. The effect was better shown when the stimulus was applied during a period of weak intestinal movements. In Bimba, in three out of 18 experiments, after the vagi had been divided and immediately after the stimulus had been removed and the gastric and intestinal tone had increased, intestinal movements occurred. A dissection confirmed that both the vagi had been divided completely above the diaphragm. The contractions occurring after vagal section can not therefore be attributed to incomplete section of these nerves. It occured as a result of a rapid increase in gastric and intestinal tone, which in the latter third of a rest period are regularly excited. The rapid increase in gastric and intestinal tone occurring after the end of the excitation causes intestinal contractions to occur.

In Bimba, the reduction in gastric and intestinal tone observed during the esophageal stimulation could be explained as being due to an inhibitory effect mediated by the splanchnic nerves after the vagi had been divided. It is less likely that the inhibition is produced by a myenteric reflex.

After bilateral transdiaphragmal vagal section no reflex effects occurred between esophagus and small intestine, even when strong stimuli were applied. In some of the experiments, stimulating the esophageal receptors by inflating the balloon to 75 ml after the vagi had been divided but the splanchnic nerves left intact caused some inhibition of intestinal contractions, but when the balloon was inflated to 100-150 ml, in most cases, a marked inhibitory effect was observed (Fig. 3). These results indicate that the inhibitory effect from the esophagus is transmitted through the sympathetic (splanchnic) nerves to the small intestine. This hypothesis does not exclude the possibility that inhibitory effects are transmitted from the esophagus to the upper part of the small intestine by sympathetic fibers running in the vagi.

Confirmation that both excitatory and inhibitory influences are transmitted through the nerves to the gastrointestinal tract is afforded by experiments on dogs in which both vagi and sympathetic trunks were divided. Of the 14 experiments on the two dogs in which the lumbar sympathetic chains had been removed and vagi and splanchnic nerves divided, no single case was observed of any influence being transmitted from the esophagus to affect movements of the small intestine, even though the balloon was inflated up to 100-150 ml. These results afford further confirmation that in the dog Bimba the transmission of inhibitory effects resulting from esophageal stimulation during a period when the stomach and intestine were at rest does not result from inhibitory effects being transmitted by the myenteric reflex.

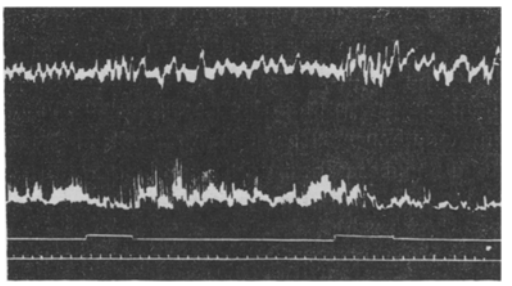

Fig. 3. Effect of esophageal stimulation on intestinal movements in the dog Laddie, after section of the vagi. Curves, from above downwards: Gastric contractions; contraction of jejunum; stimulus marker; time marker (15 sec).

The results indicate rather that when an adequate stimulus is applied to the esophageal receptors, reflex excitation of movements of the small intestine takes place and is transmitted through the vagi. When esophageal stimulation is increased, in addition to reflex excitation of small intestinal movements mediated by the vagi, reflex inhibition may also be transmitted by the sympathetic supply. This effect is not usually observed, because the excitatory effect predominates. The inhibitory influence is however well shown after vagal section.

It is known that strong stimulation of the esophageal receptors causes vomiting [1]. The reaction involves a number of other reflex mechanisms, and the process cannot be followed beyond the stage of inhibition of movement of the small intestine. The inhibitory effect may be shown when there is a sudden large inflation of the esophageal balloon. It also occurs if the dog chokes on a lump of food.

Douglas and Mann [11] have pointed out that there is no convincing evidence that the vagi play any part in mediating intestinal reflexes. The only demonstration of their motor function has been made on acute preparations. The results of our work reported in the present and in previous communications [2-4], which have been made on chronic preparations have shown that reflex excitation of movements of the small intestine is transmitted by the vagi.

SUMMARY

It was shown that in chronically esophagotomized dogs, when the splanchnic nerves are left intact, the excitatory effect on movements of the small intestine produced by esophageal stimulation are eliminated by bilateral transdiaphragmal vagotomy, but are still present if the vagi are left intact and the splanchnic nerves divided. After division of both the vagi and the splanchnic nerves, stimulation of the esophagus does not influence movements of the small intestine. In animals with divided vagi and intact splanchnic nerves, pronounced inhibition of intestinal movements is caused by esophageal stimulation whereas when both vagus and splanchnic nerves are intact, the inhibition seldom occurs and it is thought that the reason for the failure is the prevalence of the excitatory effect of esophageal stimulation. It is concluded that the excitatory effect from the esophagus (esophagointestinal reflex) is transmitted through the vagi, and the inhibitory influence through the sympathetic nerves.

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* Original Russian pagination. See C.B. Translation.