Study of the Nature of Sympathetic Trunk Nerve Fibers Enhancing Gastric Motility

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 152, No. 9, pp. 249-251, September, 2011
Original article submitted May 25, 2010

Stimulation of the sympathetic nerve in the thoracic cavity often does not inhibit, but increases stomach contractions in dogs. Blockade of α- and β-adrenoceptors potentiates this stimulatory effect, while blockade of $S_{1,2}$-receptors localized mainly in smooth muscle cells eliminates it. It is concluded that sympathetic nerve includes serotonergic fibers stimulating gastric motility.

Key Words: *stomach; sympathetic nerve; serotonergic fibers; regulation*

Sympathetic nerve (SN) not only inhibits, but also stimulates stomach contractions by activating fine afferent fibers by the axon reflex mechanisms, functional role of these fibers is not quite clear [5-7]. There is evidence that serotonin enhances motility of the gastrointestinal tract (GIT) and stimulation of the splanchnic and mesenteric nerves 1.5-3.0 fold increases the release of serotonin into the gut perfusate [2,3,8,9]. The presence of serotonergic neurons in the intramural nervous apparatus of GIT was demonstrated [10]. These data suggest that stimulation of SN promotes gastric motility also via serotonin. For obtaining reliable results, stimulation of SN not containing parasympathetic fibers is required, because stimulation of a mixed nerve can be followed by multidirectional (inhibitory and stimulatory) reactions. We previously found [3] that acceleration of heart rate during vagal stimulation occurs only in those animals which nerve comprised sympathetic fibers. In order to enhance intestinal motility, all other researchers stimulated SN in the abdominal cavity, where it contains also parasympathetic fibers of the vagus nerve [4,8-10]. Naturally, in these experiments multidirectional (inhibitory and stimulatory) effects could be observed.

In the thoracic cavity, the greater splanchnic nerve does not contain vagal parasympathetic fibers [10]. Moreover, long-term experiments were also carried out, when one right vagus nerve was cut 2-3 weeks before the main stage of the experiment, and at the final stage, the right SN was stimulated [3,9]. By this time, the vagal fibers would degenerate, even if they were present in SN before surgery.

Based on the serotonin hypothesis, we studied the nature of SN fibers accelerating gastric motility.

MATERIAL AND METHODS

The experiments were carried out on 9 mongrel dogs of both sexes weighing 10-15 kg anesthetized with Nembutal (60 mg/kg intravenously) and under conditions of artificial ventilation and open chest. At the beginning of each experiment, control stimulation of SN in the thoracic cavity (artificial respiration) was performed for evaluation of the function of the SN and the reactions of stomach. Then, α-adrenoblocker (AB) phentolamine and β-AB propranolol were injected to better identify the effect and obtain more reliable results. It was thought that the subsequent blockade of $S_{1,2}$-receptors of smooth muscles should eliminate the effect of SN stimulation. For this purpose we used 1 mg/kg lysergol (theoretically calculated concentration in the animal tissues was 10^{-4} M) blocking mainly $S_{1,2}$-receptors [1] against the background of phentolamine and propranolol.

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TABLE 1. Frequency of Gastric Responses to SN Stimulation under Different Conditions

Pattern of response	Before AB administration			AB			AB+lysergol		
	5 V	10 V	15 V	5 V	10 V	15 V	5 V	10 V	15 V
Stimulatory	4	8	8	7	9	9	0	7	7
	44.4%	88.9%	88.9%	77.8%	100%	100%	0%	77.8%	77.8%
Inhibitory	3	0	0	0	0	0	1	0	0
	33.3%	0%	0%	0%	0%	0%	11.1%	0%	0%
No response	2	1	1	2	0	0	8	2	2
	22.2%	11.1%	11.1%	22.2%	0%	0%	88.9%	22.2%	22.2%

Note. Each graph shows the number of animals and their fraction with the same type of reaction to the total in a series on 9 dogs.

Twenty-seven stomach reactions to SN stimulation (30 sec each) with amplitude of 5, 10, and 15 V and frequency of 20 Hz were studied. The stimulation was started 5-10 min after intravenous injection of lysergol.

RESULTS

SN stimulation at 5 V enhanced contractions in 44% intact animals (in 33% inhibition was observed). After SN stimulation at 10 and 15 V, the stimulatory effect was observed in 89% animals (Table 1).

The same SN stimulation against the background of α- and β-AB treatment caused stimulatory responses in 100% animals and they were much more pronounced. During SN stimulation at 5 V against the background of combined treatment with AB and lysergol, the stimulatory effect was absent. SN stimulation at amplitude of 10 and 15 V was accompanied by a decrease in the number of stimulatory effects from 100 to 77.8% and they became less pronounced. In two animals, stimulatory responses were completely suppressed. The inhibitory effects were absent, which can be explained by persisting effect of AB.

Lysergol most markedly affected the time course of the increase in intracavitary pressure. SN stimulation at 5 V did not reveal it, and SN stimulation at 10 and 15 V and at a frequency of 20 Hz reduced the gastric stimulatory response by 4.5 and 1.9 times respectively compared with that before lysergol administration (Table 2).

Comparison of the increase of stomach pressure in response to SN stimulation before and during lysergol administration showed that these differences were significant. The most striking stomach responses in this series of experiments are presented in Figure 1.

Clear-cut enhancement of contractions, typical gastric response to SN stimulation before administra-

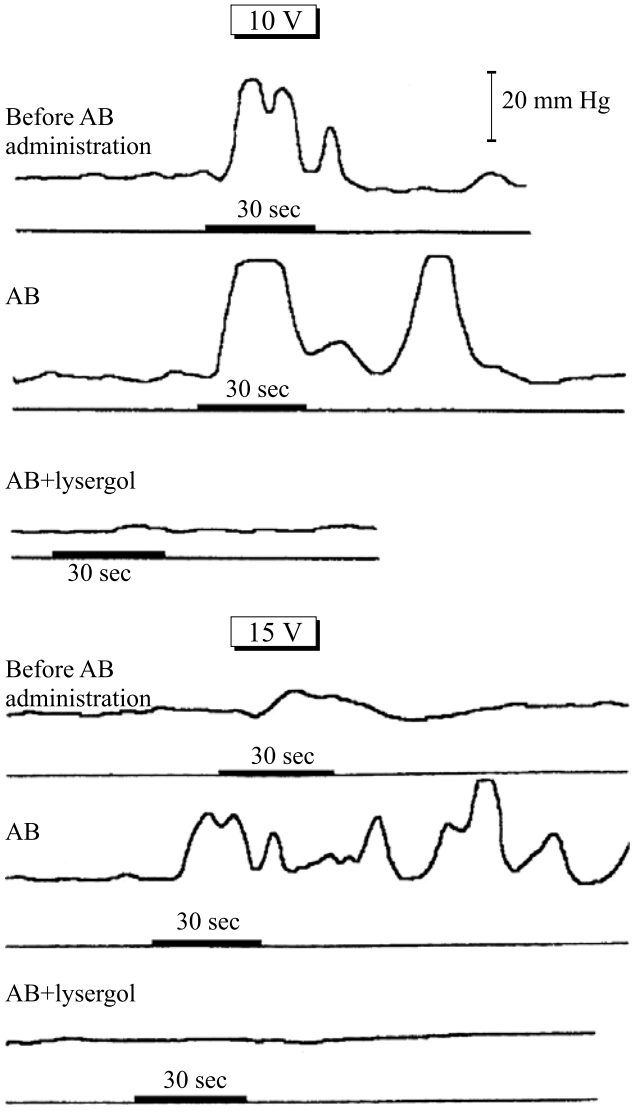

Fig. 1. Gastric response to SN stimulation after administration of various pharmacological agents. 30 sec: stimulation period.

TABLE 2. Increase in Hydrostatic Pressure (mm Hg) in the Stomach in Response to SN Stimulation under Different Conditions (n=9; $M\pm m$)

Amplitude of stimulating stimuli	Before AB administration	AB	AB+lysergol
5 V	+0.48±0.58	+4.75±1.16	+0.3±0.38*
10 V	+2.51±0.62	+7.26±1.57	+1.62±0.48*
15 V	+2.66±0.55	+8.12±1.12	+4.37±1.09**

Note. *p<0.05, **p<0.01 compared to the data under AB administration.

tion of pharmacological agents, became even more pronounced upon SN stimulation against the background of AB administration (Fig. 1). The same SN stimulation in combination with additional blockade of $S_{1,2}$-receptors of smooth muscle was not accompanied by stimulatory response.

Thus, blockade of $S_{1,2}$-receptors of gastric smooth muscles by lysergol prevents stimulatory reactions. This indicates that nerve fibers enhancing gastric motility upon SN stimulation are serotoninergic. Their endings form synapses in the smooth muscles with $S_{1,2}$-receptors.

Lysergol by itself did not significantly change the initial intracavitary pressure. Thus, hydrostatic pressure against the background of AB treatment was 11.23±1.67 mm Hg and after administration of lysergol it slightly decreased to 10.77±1.63 mm Hg, *i.e.* remained almost unchanged.

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