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Inhibition of Food Transportation in the Rat Stomach from Hepatic Branch Vagotomy

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Summary. **The transportation of residual food particles (RFP) in the stomach after hepatic branch vagotomy (HBV) in rats were examined. Weights of the RFP increased 3 and 7 days after vagotomy, but the RFP response disappeared 14 days later. No significant difference in the food and water intake between control and HBV animals was seen during the experimental periods. The wet weight of the stomach 3 days after vagotomy was greater in the HBV rats than in the HBV control rats.**

These results suggest that the hepatic vagal nerve participates in the transportation of food in the stomach.

INTRODUCTION

It has been shown that the vagal nerve contributes to the regulation of gastric motility; bilateral subdiaphragmatic vagotomy with the intact hepatic branch resulted in abnormal gastric retention of food in rats maintained on solid food.[1-3] However, it has also been suggested that gastric tone is modulated by a vagal signal originating in the hepatic portal area.[4,5] More recently, it was found that a periodic component of gastric motility can actively be controlled by the hepatic vagal signals.[6] Ingested food in the stomach is mainly transmitted to the small intestine by the periodic movement of the stomach following digestion: residual food particles in the stomach have been considered to be proportionate to the periodic component of gastric motility.[7,8]

This experiment was designed to investigate whether hepatic branch vagotomy influences food transportation in the stomach of the rat.

MATERIALS AND METHODS

Fifty-six male Wistar rats were used for this study. The animals were housed individually and allowed free access to laboratory chow (MF, Oriental Yeast, Osaka) and tap water throughout the experiments. The room temperature was controlled at 24±2℃ with 12 h : 12 h light: dark cycles (illumination from 08 : 00 to 20 : 00 h). Estimates of individual body weight and food and water intake and surgery were done between 10 : 00 and 12 : 00 h to eliminate diurnal variation.[9] The animals were deprived of food and water for 2 h from 08 : 00 on the day they were sacrificed.

When their weight had increased to about 180 g, the rats were classified into two groups: hepatic branch vogotomy (HBV) control and HBV animals.

HBV was carried out under ether anesthesia by the method previously described.[10] The vagal nerve innervating the liver was identified at the hepatic branch, and was sectioned. After surgery, the animals were returned to their cages and allowed free access to food and water.

The animals were anesthetized with pentobarbital sodium (40 mg/kg, i.p.) and sacrified 0, 3, 7 and 14 days after vagotomy. The stomach was removed, and the weight of the stomach containing food particles was estimated. Then the stomach was washed out with physiological saline, and the wet weight of the stomach and that of the residual food particles (RFP) were calculated.

Immediately before sacrifice, blood samples were taken from the tail vein for chemical analysis, and serum concentrations of substances indicating nutritional, hepatic and renal conditions (total protein;

Table 1. Food and water intake at postoperative days 3, 7 and 14 in the hepatic vagotomized control (I) and the hepatic branch vagotomized (II) rats.

Days after vagotomy	Food intake (g)		Water intake (ml)	
	I	II	I	II
3	52.0±4.0	46.4±2.0	63.1±3.2	68.1±4.5
7	139.0±3.4	127.0±6.3	129.7±5.4	120.4±4.9
14	311.5±7.5	291.8±8.0	281.8±6.8	310.8±7.3

Values are the means ± SEM (n=7).

Table 2. Wet weight of the stomach in the hepatic vagotomized control (I) and the hepatic branch vagotomized (II) rats.

	Days after vagotomy			
	0	3	7	14
Weight (mg)				
I	1032±32	1057±32	1440±65	1580±42
II	1001±48	1321±66[a]	1653±187	1521±70

Values are the means ± SEM (n=7). [a]$p<0.01$ vs I.

Table 3. Serum substances indicating hepatic or renal function in the hepatic vagotomized control (I) and the hepatic branch vagotomized (II) rats.

		Days after vagotomy			
		0	3	7	14
Total protein					
(g/dl)	I	5.1±0.0	4.7±0.1	4.9±0.2	5.1±0.1
	II	5.1±0.0	4.7±0.0	4.8±0.1	5.3±0.2
Albumin					
(g/dl)	I	2.1±0.0	1.9±0.0	1.9±0.1	1.9±0.0
	II	2.1±0.0	1.9±0.0	1.9±0.0	1.9±0.0
GPT					
(U)	I	19.6±0.7	21.6±2.1	19.6±2.4	21.5±2.2
	II	20.0±0.6	22.6±2.7	18.4±1.8	17.0±1.2
Blood urea nitrogen					
(mg/dl)	I	11.8±0.7	12.4±0.5	11.9±1.4	10.7±0.8
	II	10.9±0.7	11.2±0.7	11.1±1.1	10.0±1.1

Values are the means ± SEM (n=7).

albumin; GPT(glutamic-pyruvic transaminase); blood urea nitrogen) were measured with an autoanalyzer (Hitachi, Tokyo).[11]

The statistical significance of difference among the values was evaluated by ANOVA and Duncan's multiple range test.

RESULTS

Figure 1 shows the daily course of RFP following vagotomy. The RFP in the HBV rats was found to be greater than that in the HBV control rats 3 and 7 days after vagotomy. No significant difference in the weights of RFP between the HBV control and HBV animals was seen 14 days after vagotomy.

Food and water intake after vagal treatments is indicated in Table 1. There was no significant difference between the two groups in food and water intake during the experimental periods.

Wet weights of the stomach are shown in Table 2. The gastric weight increased in the HBV treated rats only 3 days after vagotomy. No significant difference in weight was seen 7 and 14 days after vagotomy.

Concerning the serum substances (Table 3), total protein, albumin, GPT and blood urea nitrogen were unchanged throughout the experimented periods.

DISCUSSION

We demonstrated that the hepatic vagal nerve participates in the transportation of food in the stomach. The finding that hepatic branch vagotomy increased residual food particles in the stomach (Fig. 1) is partially consistent with the report that gastric stasis was apparent at HBV because solid foods were found in the stomach of vagotomized rats at autopsy.[1-3]

Gastric motility is regulated by two factors, tone and periodicity; digested food has been considered to be mainly transmitted by the periodic movement.[7,8] It has been shown that gastric tone was able to be suppressed by a portal injection of glucose.[4,5] More recently, it was found that periodic components of motility can be differentially modulated by a portal injection of glucose.[6] Considering these reports together with the finding that residual food particles were increased by hepatic branch vagotomy (Fig. 1),

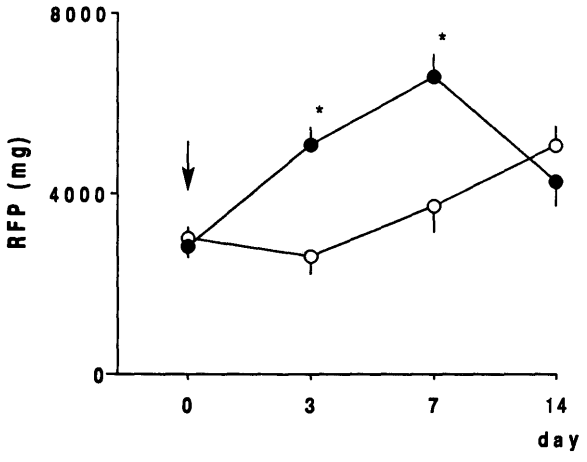

Fig. 1. Weights of RFP in the hepatic vagotomized control (○) and in the hepatic branch vagotomized (●) rats. Arrow shows the day of vagotomy. Values are the means ± SEM (n=7). *p<0.01 vs ○.

it is possible that reduced periodicity of the gastric movement by interruption of the hepatic vagal signals resulted in an increase in residual food particles. Especially since the hepatic nerve contains a signal related to glucose,[4,5] it is also possible that the glucose signal in the nerve modulates the transportation of ingested food.

The residual food response due to hepatic branch vagotomy disappeared 14 days later (Fig. 1). Because vegetative nerves regenerate quite rapidly,[12] and food intake recovery from vagotomy was achieved within a week, up to a plateau at about 80% of the preoperative level,[13] the disappearance of the residual food response may be due to the reinnervation of the hepatic vagal branch.

The residual food response due to hepatic branch vagotomy was induced (Fig. 1) even though hepatic branch vagotomy did not affect the volume of food intake (Table 1). This could mean that gastric motility caused by the hepatic vagal nerve is exerted apart from feeding behavior.

Vagotomy has been shown to affect food intake; acute hypophagia syndrome is induced after vagotomy.[7] It has also been pointed out that the hepatic branch vagotomy increases food intake when a sweet milk diet is applied,[14] but the effect of denervation of the hepatic vagal branch on food intake is evident only in an earlier stage in rats.[15,16] In the present study, hepatic branch vagotomy had no significant effect on food intake (Table 1). This supports the view previously proposed that the hepatic vagal signal does not have a major regulating role in feeding behavior.[17]

Morphological changes in the small intestine have also been induced by vagotomy, as extensive loss of intestinal villi was seen.[18] Recent studies have added further evidence that the vagal nerve plays an important role in gastric cytoprotection and ulcer formation.[19,20] In this study, the wet weight of gastric tissue was transiently heavier in the hepatic branch vagotomized rats than in the sham vagotomized rats (Table 2). The hepatic vagal signal may be concerned with the morphological change in the stomach, primary or secondary. Further study will be needed to clarify this point.

Total protein, albumin, GPT and blood urea nitrogen concentrations in the blood fluctuated within the normal range when the hepatic vagal branch was sectioned (Table 3). This could mean that the hepatic vagal signal regulates gastric food transportation independent of the nutritional, hepatic and renal conditions.

These observations lead us to suggest that the hepatic vagal branch contains a signal which modulates the transportation of ingested food in the stomach.

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