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Effects of Additional Vagal Anastomosis on the Functions of the Kirschner-Nakayama Type of Gastric Tube for Esophageal Reconstruction (A Physiological Study)

by

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INTRODUCTION

After esophageal reconstruction with the pedunculated gastric tube, especially with the KIRSCHNER-NAKAYAMA type of gastric tube, several disorders such as atony, pylorospasm, various complaints due to esophagitis and malnutrition are observed, which are probably caused by the unbalance of the autonomic nervous control of the gastric tube due to the following reason. That is, in the procedure to mobilize this type of gastric tube, bilateral vagus nerves are completely severed, but numerous sympathetic nerve fibers are preserved, such as nerves running along the right gastric and right gastroepiploic arteries.

For instance, vagal innervation of the gastric tube in the patients, who had undergone

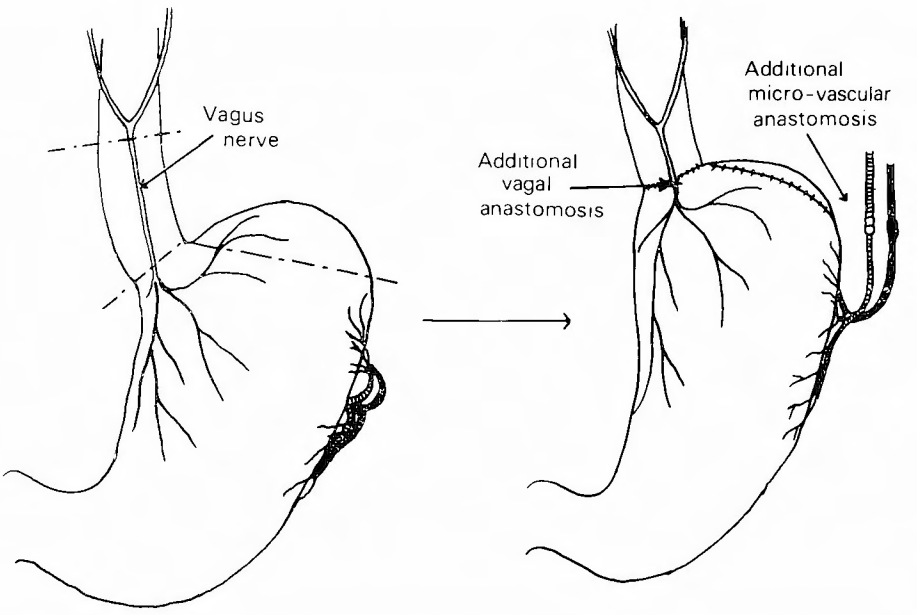

Fig. 1 Method of esophageal reconstruction by means of the KIRSCHNER-NAKAYAMA type of gastric tube with additional microvascular and vagal anastomoses.

an esophagectomy for esophageal cancer, has been recognized to be abolished for a certain postoperative period by using the HOLLANDER's insulin test[23] which proves the vagally induced intact cephalic phase of gastric secretion.

Considering that almost all of the gastric tube with the distal cut end of the vagus nerve has been utilized for esophageal reconstruction by reconstructing the gastric vessels on the left side with recently advanced small vessel anastomosis technique or by preserving the ascending branch of the left gastric artery, functional recovery of the stomach was examined in dogs, which had undergone vagal anastomosis following intrathoracic vagotomy, and furthermore, that of the KIRSCHNER-NAKAYAMA type of gastric tube used for esophageal reconstruction was examined in the dogs, in which the proximal cut end of the vagus nerve had been anastomosed to the distal one on the gastric tube.

I. INSULIN TEST IN CLINICAL CASES AFTER ESOPHAGECTOMY

1. Materials and Method

On the patients who had undergone esophagectomy with intrathoracic or antethoracic esophagogastrostomy the insulin test was performed. After fasting for over 15 hours a nasogastric rubber tube was passed into the gastric tube. After complete withdrawal of the resting juice, samples were collected for 15 minutes. Then, 20 units of regular insulin were given intravenously. Thereafter, serial 15-minute samples of gastric juice were aspirated for 2 hours. The acid concentration of each 15-minute sample was determined by titration against 0.01 N sodium hydroxide, TOEPFER's reagent (five-tenth percent alcoholic solution of dimethyl-amino-azobenzene) being used as an indicator. Blood sugar estimations were made at 30 and 45 minutes after the insulin injection.

2. Results (Table 1)

Table 1 gives the results of the insulin test, in which free hydrochloric acid concentration of resting juice and maximal acid concentration after the insulin injection are described. Elevation of free hydrochloric acid concentration after the insulin injection was

Table 1. Acid secretory response to insulin hypoglycemia after esophagectomy in clinical cases.

Patient age sex	Method of operation	Time after operation	Free HCl (mEq/l)		Result
			before injection	after injection	
K. Y. 72 male	antethoracic esophagogastrostomy	1 month	0	0	—
Y. F 57 male	antethoracic esophagogastrostomy	2 months	0	0	—
Y. N. 56 male	antethoracic esophagogastrostomy	2 months	0	0	—
C. H. 57 male	antethoracic esophagogastrostomy	2.5 months	0	0	—
A. H. 56 male	antethoracic esophagogastrostomy	3 months	0	0	—
H. F. 61 female	intrathoracic esophagogastrostomy	1 month	0	0	—
Y. N. 68 male	intrathoracic esophagogastrostomy	1.5 months	0	0	—
K. I. 55 male	intrathoracic esophagogastrostomy	1 year and 1 month	0	0	—
S. K. 55 male	intrathoracic esophagogastrostomy	3 years and 3 months	11	0	—

not observed at all for over 3 years after intrathoracic and antethoracic esophagogastrostomy, especially after the former operation. In every test adequate hypoglycemia which remained under the value of 50 mg/dl had been demonstrated.

II. SECRETORY FUNCTION OF THE STOMACH IN FOUR GROUPS OF DOGS, WHICH HAD UNDERGONE INTRATHORACIC VAGOTOMY, VAGAL ANASTOMOSIS FOLLOWING VAGOTOMY, ESOPHAGECTOMY WITH INTRATHORACIC ESOPHAGOGASTROSTOMY AND INTRATHORACIC ESOPHAGOGASTROSTOMY WITH ADDITIONAL VAGAL ANASTOMOSIS, RESPECTIVELY

1. Materials and Methods

Adult mongrel dogs, weighing from 10 kg to 20 kg were used.

i) Operative procedures

The animals were anesthetized with intravenous administration of sodium pentobarbital, 25 mg/kg. After intratracheal intubation, the tube was connected to the closed circuit anesthetising apparatus or positive pressure respirator and artificial respiration was performed when necessary.

For preparing vagotomized dogs and dogs with vagal anastomosis following vagotomy, animals were placed on the right side, and thoracic incision was made in the left seventh or eighth intercostal space. The vagus nerves were resected about 3 cm in length all around the esophagus at the level of about 5 cm above the esophageal hiatus, where the vagus nerves ramified into 2 to 4 trunks in number, and both their proximal and distal cut ends were turned about and ligated. For vagal anastomosis, the thickest branch, generally ventral to the esophagus, was chosen and anastomosed with 3 to 4 interrupted sutures through the epineurium using silk threads for vessel anastomosis.

For preparing the dogs with intrathoracic esophagogastrostomy following esophagectomy, thoracic incision was made in the left eighth or ninth intercostal space. The undermost portion of the esophagus was resected about 5 cm long above the esophagocardial junction. The stomach was delivered through the esophageal hiatus and the left gastric artery was cut proximal to the ramifying point of the ascending branch so that the blood supply to the cardia through the right gastric artery was preserved. The spleen was not extirpated to preserve good blood supply to the greater curvature. By this method, the total gastric tube including cardia was utilized for esophageal reconstruction with good blood supply. Mostly, end-to-end anastomosis was made between the esophagus and the stomach, but in a few cases the stump of the stomach was closed and the esophagus was anastomosed to the fundus of the stomach. Anastomosis was made with interrupted silk sutures in two layers, in total layer and the seromuscular layer, respectively. For the additional vagal anastomosis, care was taken to prevent the vagal trunk to be anastomosed from being missed. As the vagal trunk for anastomosis, the thickest one was chosen. As a result, in only one dog the proximal cut end of the posterior vagal trunk was anastomosed to the distal one of the same trunk, and in all of the other dogs the anastomosis was made between anterior proximal and anterior distal. The gastric tube was covered with the mediastinal pleura as much as possible and fixed to the esophageal hiatus. The chest was closed with a drain in the thorax, through which low pressure continuous suction by 15 cm to 17 cm H_2O was made for 3 hours after operation. The dog was not allowed to

take anything by mouth for 5 to 7 postoperative days and was given 500 ml of subcutaneous fluids, 800,000 units of procaine penicillin and 1 g of streptomycin, every day. A liquid diet was gradually changed to a normal diet during the following 7 days. Dogs with intrathoracic vagotomy and those with vagal anastomosis following vagotomy were put on a normal diet from the next day after operation.

ii) Method of tests

Generally, the following tests were performed every month after the operation.

a) Insulin test

This test mostly followed the HOLLANDER's standard method[23)45)]. The dogs, after fasting for over 24 hours, were anesthetized with intravenous administration of sodium pentobarbital, 25 mg/kg. After a rubber tube was passed into the stomach through the mouth, resting juice was withdrawn as completely as possible and 15 minute aspirate of spontaneous secretion was obtained. Thereafter, 20 units of regular insulin was given intravenously and serial 15 minute samples of gastric juice were aspirated for 2 hours. Blood sugar estimations were made before the insulin injection, and at 30 and 45 minutes after the injection, by CRECELIUS-SEIFERT's or SOMOGYI's method. The free hydrochloric acid and total acid concentration of each 15-minute sample were determined by titration against 0.01 N sodium hydroxide, TOEPFER's reagent (five-tenth percent alcoholic solution of dimethyl-amino-azobenzene) and phenolphthalein (one percent alcoholic solution) being used as indicators, respectively.

b) Histamine test

After a similar preparation like that in the insulin test, 1 mg of histamine dihydrochloride was given to the dog subcutaneously. Thereafter, serial 15-minute samples of gastric juice were aspirated for 2 hours. The samples were titrated by the same method used for the insulin test.

2. Results

i) Insulin test

Blood sugar concentration ranged between 90 mg/dl and 130 mg/dl before insulin injection, and after injection at least one of the two samples showed the value of less than 50 mg/dl as a result of effective insulin stimuli. Although in some tests animals were seizured with a slight cramp caused by hypoglycemia, the tests were not hindered.

In the non-operated dogs gastric juice aspirates decreased temporarily after insulin injection, but increased 30 to 90 minutes after injection. At the same time free hydrochloric acid concentrations elevated. In Table 2 and Fig. 2 the maximal concentrations of free hydrochloric acid and total acid are shown.

a) The dogs after intrathoracic vagotomy

The amounts of aspirated gastric juice were extremely little both before and after insulin injection and retention of gastric contents was supposed to be present judging from aspirated chyme or remnant of food. Therefore, the animals were fasted for over 24 hours except some earlier cases in this series, so as to exclude those admixtures. On titration, the concentrations of free hydrochloric acid were not increased and instead, in some cases, decreases were observed. One dog with antethoracically excluded gastric tube was included in this group, and its secretory response to insulin was the same as those

of intrathoracically vagotomized dogs. Therefore, vagal gastric secretion was not observed for 2 to 10 months after operation (Table 2 and Fig. 2).

b) The dogs with vagal anastomosis following intrathoracic vagotomy

In general, responses to insulin were scarcely observed until the 3rd postoperative month, but inhibitions were noticed in many cases. From the 4th month after operation,

Table 2. Acid secretory response to insulin hypoglycemia in non-operated and vagotomized dogs

Time after operation (months)	Dog No.	Method of operation	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
	12	none	0	20	13	38
	12	none	0	20	13	69
	14	none	0	18	12	67
	15	none	11	28	34	68
	21	none	6	44	28	74
	27	none	0	65	15	80
	27	none	8	78	21	95
1	3	intrathoracic vagotomy	0	0	14	42
1	5	intrathoracic vagotomy	3	3	30	32
1	7	intrathoracic vagotomy	7	9	92	94
1	22	intrathoracic vagotomy	7	0	18	20
1	21	antethoracic transplantation	18	0	23	14
2	3		41	17	91	85
2	5		0	0	18	26
2	7		12	21	40	46
2	21		11	8	28	29
3	3		0	12	52	72
3	5		12	16	45	78
3	7		0	0	12	14
3	21		0	0	9	16
4	3		2	14	62	64
4	5		0	0	10	13
4	7		19	29	34	39
4	21		0	0	8	8
5	5		0	8	20	23
5	7		12	0	17	14
5	21		10	0	32	30
6	5		8	23	58	71
6	7		2	14	34	53
6	21		0	0	11	25
7	5		40	40	48	86
7	7		16	12	29	36
7	21		0	0	11	24
8	5		0	0	23	43
8	7		20	4	36	38
9	7		0	0	22	24
10	7		0	0	7	6

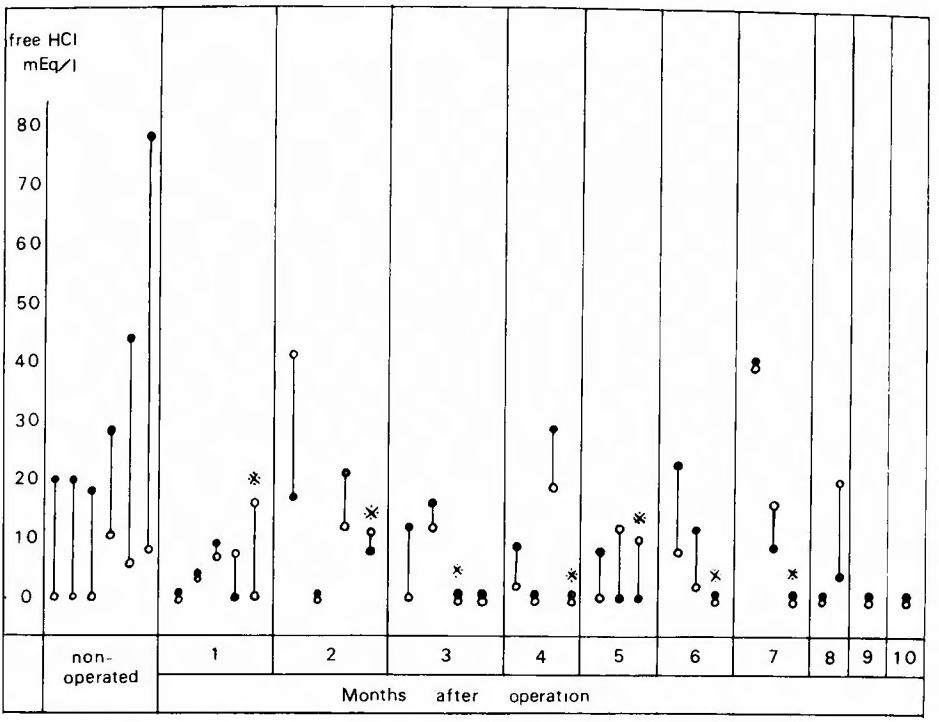

Fig. 2 Acid secretory response to insulin hypoglycemia in non-operated and vagotomized dogs.
o : Before injection ● : Maximal value within 2 hours after injection
⁂ : Dog with the antethoracically transplanted KIRSCHNER-NAKAYAMA type of gastric tube and gastrojejunostomy

responses to insulin were manifested. The amount of aspirated gastric juice increased 30 to 90 minutes after insulin injection and the concentration of free hydrochloric acid also elevated. Especially, in the 4th postoperative month, the concentration of free hydrochloric acid increased by 70 mEq/1 in response to insulin. From the 5th to 10th month after operation spontaneous secretion of free hydrochloric acid was vigorous, but response to insulin was not so intense as in the 4th month (Table 3 and Fig. 3).

Table 3. Acid secretory response to insulin hypoglycemia in dogs with vagal anastomosis immediately after intrathoracic vagotomy. Anterior trunk of the vagus nerve had been anastomosed in every dog in this group.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	4	0	0	9	15
1	6	0	0	61	81
1	10	62	28	93	70
1	25	20	24	40	49
2	4	0	0	52	30
2	6	0	0	47	66
2	10	59	50	89	74
2	11	12	8	40	24

2	23	15	0	63	68
3	4	0	0	7	6
3	6	16	22	35	48
3	10	67	56	80	72
3	23	22	16	42	47
4	4	7	75	28	87
4	6	1	6	28	67
4	10	9	61	23	75
4	23	15	20	33	60
5	6	0	14	36	78
5	10	6	73	87	89
6	10	0	0	71	65
7	10	29	63	46	83
9	10	50	67	60	74
10	10	60	70	76	85

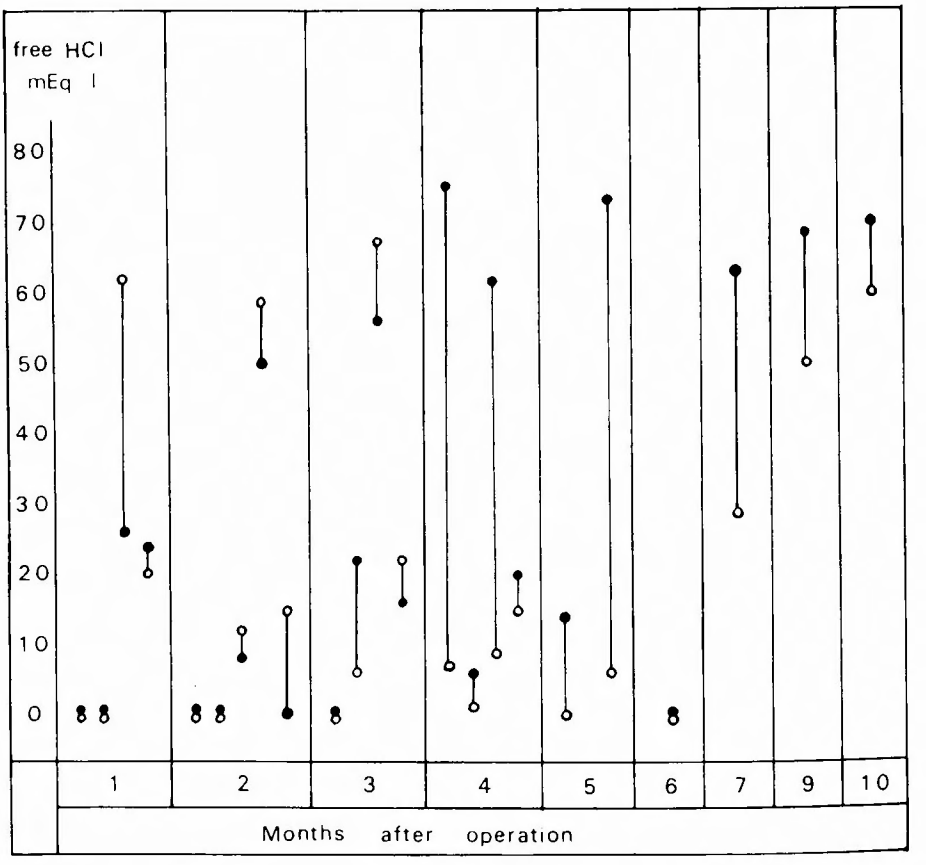

Fig. 3 Acid secretory response to insulin hypoglycemia in dogs with vagal anastomosis immediately after intrathoracic vagotomy. In all dogs anterior trunks of the vagus nerves had been anastomosed.
o : Before injection ● : Maximal value within 2 hours after injection

Table 4. Acid secretory response to insulin hypoglycemia in dogs after esophagectomy and intrathoracic esophagogastrostomy.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	34	20	35	65	59
1	62	66	61	79	70
2	17	27	29	40	38
2	34	21	25	38	36
3	17	7	7	14	17
3	34	35	33	45	48
4	48	0	4	30	32
5	48	0	0	41	60
6	48	14	28	27	66

After vagotomy and in the 1st or 2nd month after vagal anastomosis free hydrochloric acid was found abundant in resting juice and that was, the author supposed, partly caused by the gastric or intestinal phase secretion owing to gastric retention.

c) The dogs with intrathoracic esophagogastrostomy after esophagectomy

The 15 minute aspirates were, generally, more abundant in volume than those after vagotomy, but responses to insulin were scarcely found as was the case after vagotomy alone. The vagal secretion was not observed within 6 months after operation (Table 4 and Fig. 4).

d) The dogs with intrathoracic esophagogastrostomy and additional vagal anastomosis after esophagectomy.

No response to insulin was found within 2 months after operation. From the 3rd postoperative month the 15-minute aspirates increased 45 to 120 minutes after insulin injection, and the gross appearance of those aspirates became clear. On titration, increases of acidity were remarkable (Table 5 and

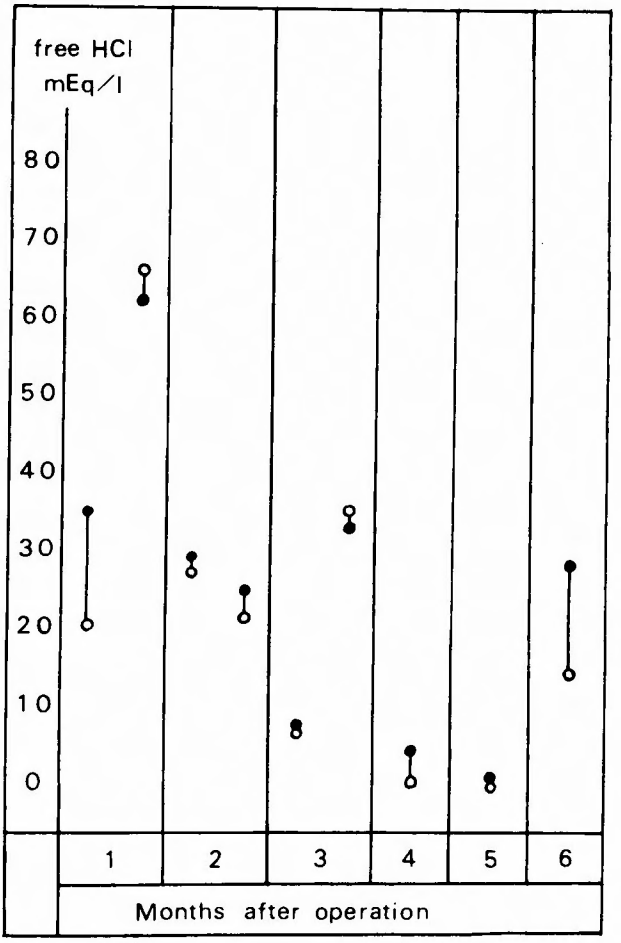

Fig. 4 Acid secretory response to insulin hypoglycemia in dogs after esophagectomy and intrathoracic esophagogastrostomy.
○ : Before injection
● : Maximal value within 2 hours after injection

Table 5. Acid secretory rerponse to insulin hypoglycemia in dogs after esophagectomy and intrathoracic esophagogastrostomy with additional vagal anastomosis. Anterior trunk of the vagus nerve had been anastomosed in dogs, No. 32 and No. 35, and posterior trunk, in No. 31.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	31	14	14	43	56
1	35	14	11	30	30
2	31	17	22	35	44
2	32	22	26	35	34
2	35	14	32	30	65
3	31	18	68	37	92
3	32	37	51	61	76
3	35	0	32	39	65
4	31	10	26	29	62
4	32	22	80	45	98
4	35	0	0	74	81
5	32	23	50	48	69
5	35	15	41	73	80
6	32	33	54	53	70

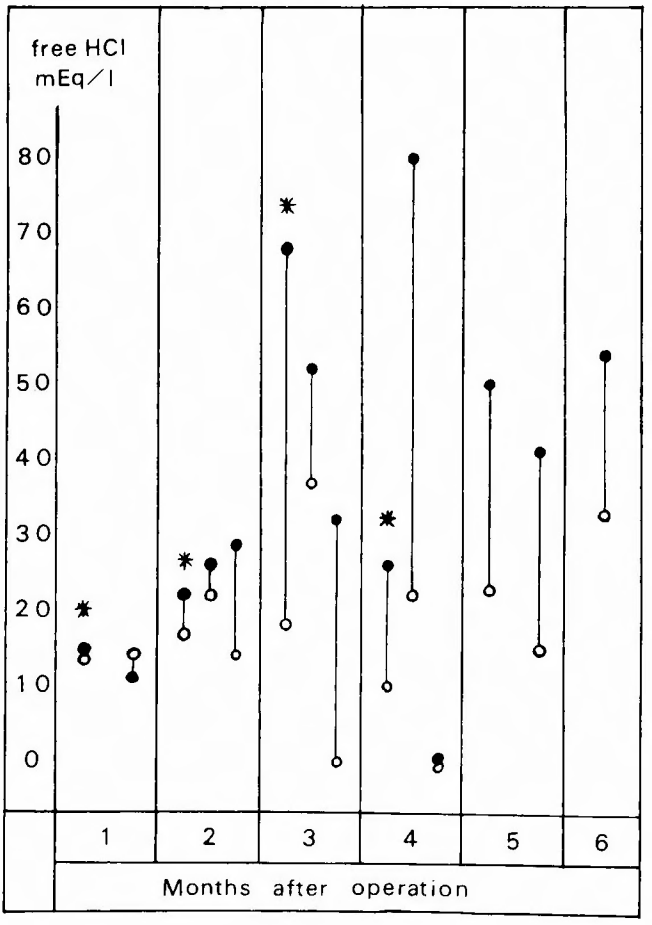

Fig. 5 Acid secretory response to insulin hypoglycemia in dogs after esophagectomy and intrathoracic esophagogastrostomy with additional vagal anastomosis.
* : Dog with vagal anastomosis of posterior trunk of the vagus nerve.
In all of the other dogs, anterior trunks of the vagus nerves had been anastomosed.
○ : Before injection
● : Maximal value within 2 hours after injection

Table 6. Acid secretory response to histamine dihydrochloride in non-operated and vagotomized dogs.

Time after operation (months)	Dog No.	Method of operation	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
	11	none	0	121	26	147
	12	none	0	38	64	78
	13	none	0	31	10	78
	14	none	0	112	40	137
	15	none	23	104	10	123
	21	none	0	91	14	121
	22	none	29	110	42	128
	27	none	0	104	15	127
	27	none	8	119	21	142
	30	none	0	67	4	93
1	3	intrathoracic vagotomy	0	62	26	74
1	5	intrathoracic vagotomy	0	46	20	70
1	7	intrathoracic vagotomy	0	51	62	94
1	12	intrathoracic vagotomy	4	66	16	82
1	22	intrathoracic vagotomy	7	110	18	130
1	26	intrathoracic vagotomy	18	72	40	98
1	21	antethoracic transplantation	18	67	23	82
2	3		25	95	85	133
2	5		0	43	41	78
2	7		0	44	26	73
2	12		0	47	10	60
3	3		0	54	78	114
3	5		16	20	50	66
3	7		0	53	7	71
3	12		38	79	50	86
3	21		0	91	6	113
4	3		6	47	58	79
4	5		0	42	21	70
4	7		24	96	37	121
4	21		0	97	11	114
5	5		20	32	95	84
5	7		0	76	40	96
5	21		0	80	20	109
6	5		27	55	50	75
6	7		23	124	86	138
6	21		0	77	15	105
7	5		10	62	49	119
7	7		0	54	18	67
7	21		0	87	13	115
8	7		0	90	28	100
9	7		0	88	22	108
10	7		0	106	7	115

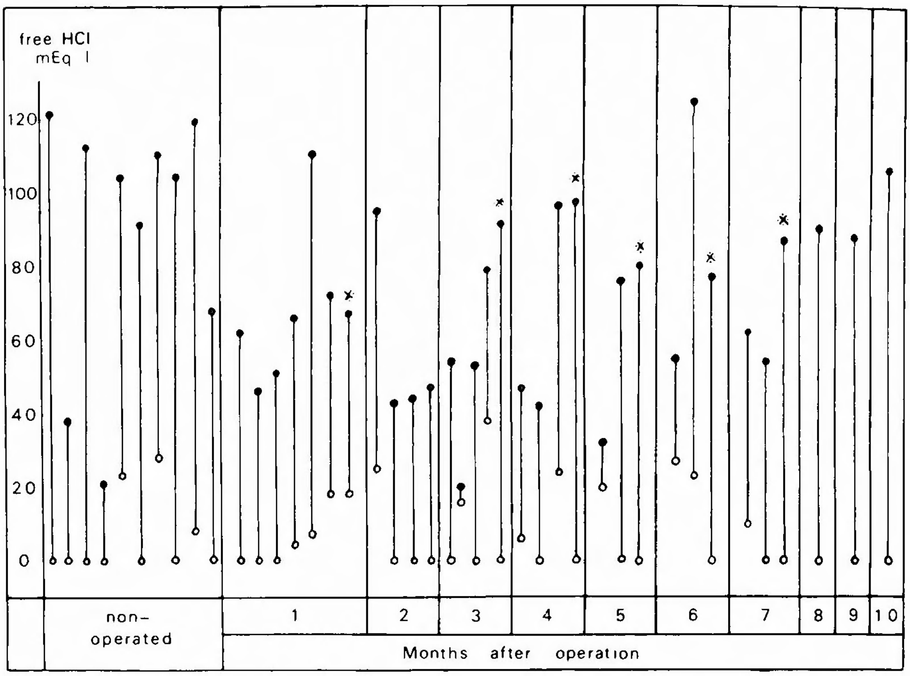

Fig. 6 Acid secretory response to histamine in non-operated and vagotomized dogs.
o : Before injection ● : Maximal value within 2 hours after injection
⁂ : Dog with the antethoracically transplanted KIRSCHNER-NAKAYAMA type of gastric tube and gastrojejunostomy

Fig. 5).

Judging from the results of the above-mentioned insulin tests, it was clarified that cephalic phase secretion mediated through the vagus nerves reappeared in the 3rd or 4th month after vagal anastomosis. The recovery of cephalic phase secretion was sooner in the dogs with esophagogastrostomy and additional vagal anastomosis than in the dogs with vagal anastomosis following intrathoracic vagotomy, owing to the fact that, the author supposes, the distances of vagus nerve regenerations were longer in the latter than in the former.

ii) Histamine test

One ml. of one-tenth percent solution of histamine dihydrochloride was injected subcutaneously in a single test, which had been prepared in the dispensary of the Kyoto university hospital.

In the non-operated dogs, aspirated gastric juice increased remarkably in volume and became clear 45 to 90 minutes after histamine injection except some brownish-colored aspirates owing to bleeding. Increases of acidity were more conspicuous than in insulin tests, and the maximal concentrations of free hydrochloric acid ranged between 90 and 120 mEq/l in the majority of the histamine tests (Table 6 and Fig. 6).

a) The dogs after intrathoracic vagotomy

Responses to histamine were a little diminished after the operation, but the concentrations of free hydrochloric acid still amounted to the range between 46 and 110 mEq/

Table 7. Acid secretory response to histamine dihydrochloride in dogs with vagal anastomosis immediately after intrathoracic vagotomy. Anterior trunk of the severed vagus nerve had been anastomosed in every dog in this group.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	4	0	29	23	67
1	6	0	5	34	94
1	10	0	137	40	171
1	25	20	42	40	66
2	4	0	28	17	68
2	6	0	28	22	52
2	10	3	102	25	130
2	11	15	62	47	80
2	23	30	70	32	99
3	4	0	86	28	122
3	6	0	28	40	71
3	10	7	100	36	146
3	23	22	43	38	53
4	4	5	63	23	83
4	10	13	106	20	122
4	23	15	58	33	85
5	6	10	18	49	88
5	10	20	109	36	135
6	10	0	10	43	76
7	10	29	116	46	133
9	10	42	109	48	126
10	10	40	95	66	120

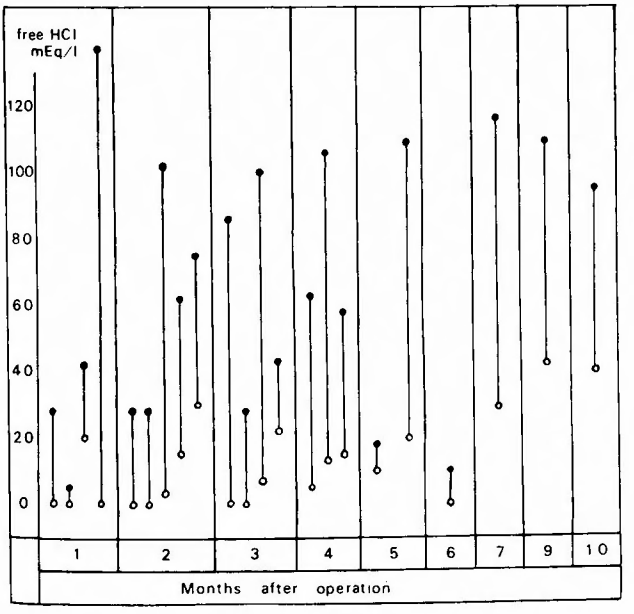

Fig. 7 Acid secretory response to histamine in dogs with vagal anastomosis immediately after intrathoracic vagotomy. In all dogs anterior trunks of the vagus nerves had been anastomosed.

o : Before injection

● : Maximal value within 2 hours after injection

Table 8. Acid secretory response to histamine dihydrochloride in dogs after esophagectomy and intrathoracic esophagogastrostomy.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	34	20	108	65	124
1	62	66	94	79	105
2	17	6	65	33	89
2	34	21	115	38	154
3	17	10	61	21	74
3	34	35	68	45	90
4	48	0	25	30	65
5	48	0	41	41	108
6	48	14	42	27	82

l in response to histamine in the 1st postoperative month. No remarkable change in responsiveness to histamine was observed in the following postoperative months. Effect of vagotomy on acid secretory response to histamine, although somewhat decreased, was not so distinct as that to insulin, and in some dogs violent responses were observed (Table 6 and Fig. 6).

b) The dogs with vagal anastomosis following vagotomy

Generally, acid secretory responses to histamine were distinct from the early postoperative months, but in some dogs responses were feeble. No obvious differences in responses to histamine were found between vagotomized dogs and dogs with vagal anastomosis, although the latter's maximal acidity amounting to the range between 95 and 116 mEq/l exceeded the former's between 54 and 106 mEq/l from the 7th to 10th postoperative month (Table 7 and Fig. 7).

c) The dogs with intrathoracic esophagogastrostomy after esophagectomy

Acid secretory responses to histamine were distinct from the early

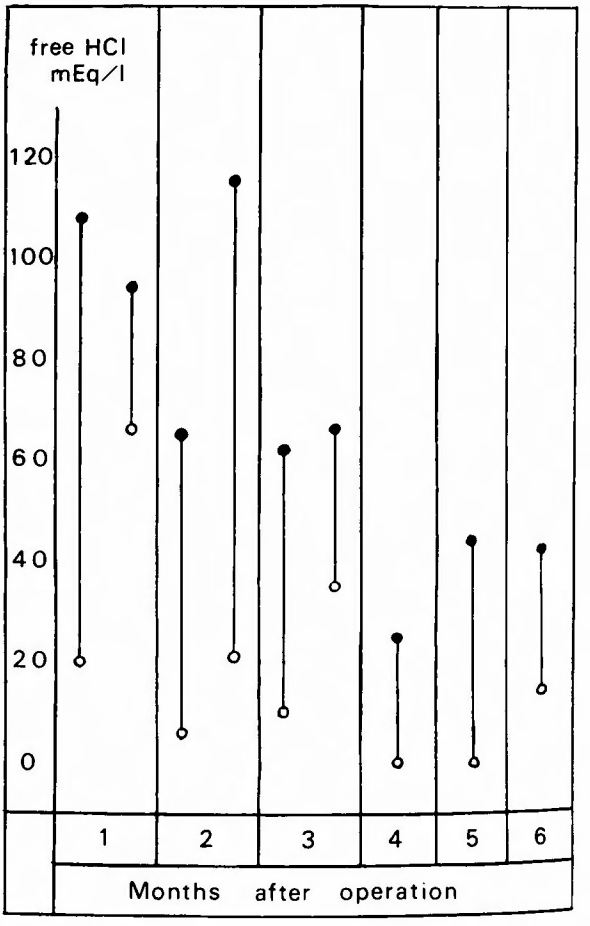

Fig. 8 Acid secretory response to histamine in dogs after esophagectomy and intrathoracic esophagogastrostomy.
o : Before injection
● : Maximal value within 2 hours after injection

Table 9. Acid secretory response to histamine dihydrochloride in dogs after esophagectomy and intrathoracic esophagogastrostomy with additional vagal anastomosis. Anterior trunk of the vagus nerve had been anastomosed in dogs, No. 32 and No. 35, and posterior trunk, in No. 31.

Time after operation (months)	Dog No.	Free HCl before injection (mEq/l)	Maximal free HCl after injection (mEq/l)	Total acidity before injection (mEq/l)	Maximal total acidity after injection (mEq/l)
1	31	14	52	43	80
1	35	14	90	30	119
2	31	17	60	35	95
2	32	22	46	35	83
2	35	14	42	30	73
3	31	18	98	37	108
3	32	37	79	61	108
3	35	0	83	39	103
4	31	10	46	29	106
4	32	22	97	45	114
4	35	0	66	74	102
5	32	23	62	48	97
5	35	15	56	73	90
6	35	33	96	53	127

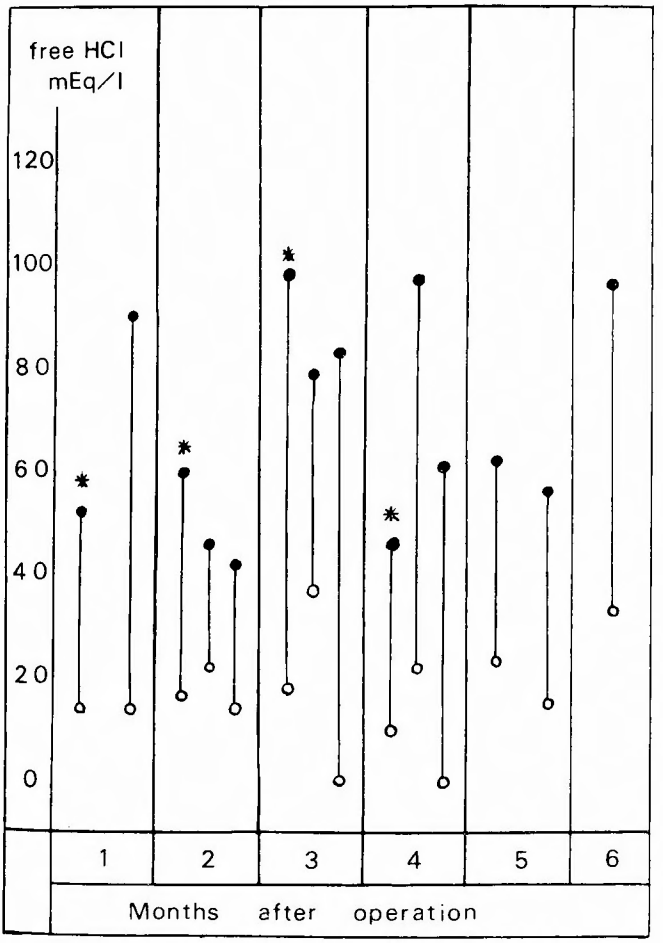

Fig. 9 Acid secretory response to histamine in dogs after esophagectomy and intrathoracic esophagogastrostomy with additional vagal anastomosis.

* : Dog with vagal anastomosis of posterior trunk.

In all of the other dogs, anterior trunks of the vagus nerves had been anastomosed.

o : Before injection

● : Maximal value within 2 hours after injection

postoperative months and were quite different from the responses to insulin in the following months. The maximal concentrations of free hydrochloric acid ranged between 25 and 115 mEq/l (Table 8 and Fig. 8).

d) The dogs with intrathoracic esophagogastrostomy and additional vagal anastomosis.

The concentration of free hydrochloric acid in response to histamine ranged between 43 and 98 mEq/l. Although the responsiveness seemed to be independent of the term after operation, acidity in this group exceeded that of dog without vagal anastomosis from the 3rd to 6th postoperative month (Table 9 and Fig. 9).

Judging from the results of the above-mentioned histamine tests, it was clarified that response to histamine, that is, secretory function of parietal cells, was relatively well preserved after operations, though somewhat diminished generally.

Acidity of the gastric juice of dogs with vagal anastomosis generally exceeded that of vagotomized dogs from the 3rd or 4th postoperative month, although the difference was not so distinct as in insulin tests.

The summaries of the results of examinations on gastric secretory function are as follows : Response to insulin is markedly reduced after vagotomy and recovers from the 3rd or 4th month after vagal anastomosis, showing reappearance of cephalic phase secretion. On the other hand, response to histamine is somewhat diminished after vagotomy, and its recovery by vagal anastomosis was not so conspicuous as in response to insulin. However, no remarkable differences of responsiveness to histamine were found among those four groups of dogs, showing preservation of secretory function of parietal cells after vagotomy.

III. MOTORIAL FUNCTION OF THE STOMACH IN FOUR GROUPS OF DOGS, WHICH HAD UNDERGONE INTRATHORACIC VAGOTOMY, VAGAL ANASTOMOSIS FOLLOWING VAGOTOMY, ESOPHAGECTOMY WITH INTRATHORACIC ESOPHAGOGASTROSTOMY AND INTRATHORACIC ESOPHAGOGASTROSTOMY WITH ADDITIONAL VAGAL ANASTOMOSIS, RESPECTIVELY

1. Methods of tests

i) Intraluminal gastric pressure studies

a) Balloon method

In vagotomized dogs and dogs with vagal anastomosis, changes of intraluminal gastric pressure were examined using a balloon introduced into the stomach[32]. For the examination of the body of the stomach, a balloon, 6 cm in diameter and 7 cm in length, made of an ice bag was used. For the pyloric region, a balloon, 4 cm in length, made of a condom was used with a disk of synthetic resin around its oral portion, to prevent it from falling into the duodenum.

Animals, after fasting for over 24 hours, underwent laparotomy under intravenous administration of sodium pentobarbital, and these balloons were introduced from the stoma on the fundus of the stomach. The balloons were filled with air and given basic internal pressures between 15 and 20 cm of water. The balloons were connected throught rubber tubes to the electric manometer of MP 1T type (NIHONKOHDEN Co. Ltd.), amplifier of MP 3A type (NIHONKOHDEN Co. Ltd.) and recorder (UNICORDER : YANAGIMOTO). To examine, besides the basic motor activity, the responses to the drugs acting on the

autonomic nervous system, 0.005 to 0.025 mg/kg of neostigmine methylsulfate, 0.03 to 0.1 mg/kg of epirenamine hydrochloride, 0.5 to 1.0 mg/kg of benzylimidazoline hydrochloride, 0.01 to 0.02 mg/kg of atropine sulfate, 0.3 to 0.5 mg/kg of pilocarpine hydrochloride, 0.005 to 0.05 mg/kg of acetylcholine chloride and 4 to 6 mg/kg of tetraethylammonium bromide were injected intravenously.

b) Open-tipped tube method

As an open-tipped tube, a rubber nasogastric tube of No. 8, 8 mm in the outside diameter and 5 mm in the inside diameter, was used after cutting off its end, closing the stump and making a side opening, 5 mm in diameter, at the point 1 cm from the cut end.

Animals, after fasting for over 24 hours, underwent laparotomy under intravenous administration of sodium pentobarbital, 25 mg/kg in the supine position. The open-tipped tube, filled with water, was introduced from the mouth to the duodenum, and its end was ascertained to be positioned at the point 3 to 5 cm aboral to the pyloric ring by palpation through the wound. Another end of the tube was connected to the electric manometer of MP-4T type (NIHONKOHDEN Co. Ltd.) and polygragh of RM-150 type (NIHONKOHDEN Co Ltd.) containing amplifier of RP-2 type and recorder of WI-260TR type.

Several minutes after introducing the tube into the duodenum, the duodenal motor activity became stable. Then, the tube was pulled out slowly, while the intraluminal pressure of the duodenum, the pylorus and the body of the stomach was continuously recorded. The speed used to pull out the tube was 2 to 10 mm per minute, but in each examination it was kept as uniform as possible. When the intraluminal pressures were suspected to be under the influence of the respiratory movement, it was eliminated by means of controlled respiration with a respirator, or the doubt was dispelled by a pneumograph recorded simultaneously.

ii) Evacuation of the gastric contents

After fasting for over 24 hours, 150 ml of solution of barium sulfate was instilled through a rubber tube into the stomach of the dog under intravenous administration of sodium pentobarbital, 25 mg/kg. Gastrointestinal X ray examinations were performed immediately and two hours after instillation.

2. Results

i) Intraluminal gastric pressure studies

a) Balloon method

There was no difference between vagotomized dogs and dogs with vagal anastomosis in the manner of responses to drugs acting on the autonomic nervous system. In this study, however, responses could not be compared quantitatively between these two groups. Basic activity of the pylorus and the body of the stomach, when the maximal intraluminal pressures were compared between these two groups of dogs, showed a tendency as follows : Increases of intraluminal pressures in the pylorus in vagotomized dogs were greater than those in dogs with vagal anastomosis. On the other hand, increases of intraluminal pressures in the body of the stomach in dogs with vagal anastomosis were greater than those in vagotomized dogs (Table 10 and Fig. 10).

b) Open-tipped tube method

Table 10. Muscular activity of the body of stomach and pyloric region recorded by balloon. Amplitude of pressure elevation was measured from the basal pressure of the body of stomach and pyloric region, respectively.

Method of operation	Time after operation	Body of the stomach		Pyloric region	
		Amplitude (cm H_2O)	Frequency (per min.)	Amplitude (cm H_2O)	Frequency (per min.)
intrathoracic vagotomy and anastomosis of anterior trunk of vagus nerve	2 weeks	1.5~5	4	2.5~6.6	2~3
intrathoracic vagotomy and anastomosis of anterior trunk of vagus nerve	1 month	2~4	2		
intrathoracic vagotomy and anastomosis of anterior trunk of vagus nerve	4 months	2~8.2	3	1.6~5.8	3.6
intrathoracic vagotomy and anastomosis of anterior trunk of vagus nerve	10 months	2~3.2	3	2.1~6.3	2
intrathoracic vagotomy	10 months	2	2	3.9~14.2	1.8

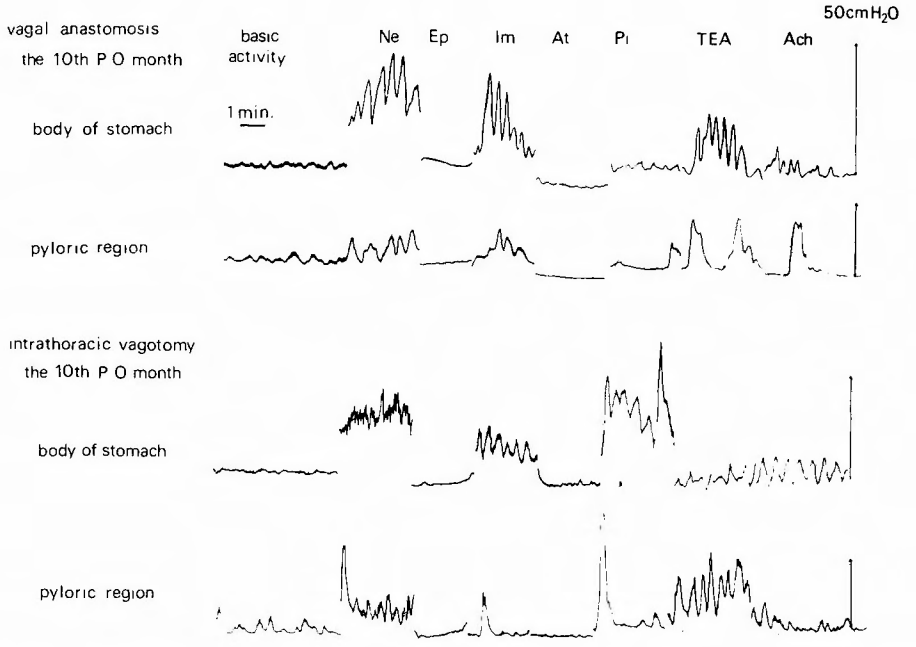

Fig. 10 Muscular activity of body of the stomach and pyloric region : Basic activity and responses to the drugs acting on the autonomic nervous system were recorded by balloon.

Ne : Neostigmine methylsulfate
Ep : Epirenamine hydrochloride
Im : Benzylimidazoline hydrochloride
At : Atropine sulfate
Pi : Pilocarpine hydrochloride
TEA : Tetraethylammonium bromide
Ach : Acetylcholine chloride

When the tube was pulled out and its opening reached the pyloric region, an increase of the intraluminal pressure was observed. The intraluminal pressure was maximal at the pyloric region and decreased gradually until the opening reached the body of the stomach. Thereafter, no remarkable change of gastric motor activity was observed up to the cardia. In the dogs after intrathoracic esophagogastrostomy, the intrathoracic part of

the gastric tube showed no change in intraluminal pressure. The maximal intraluminal pressures of the pyloric region in the vagotomized dogs, as compared with the basal pressure of the duodenum, were higher than those of non-vagotomized dogs or dogs with vagal anastomosis. Figure, periodicity and regularity of the intraluminal pressure curves showed no distinct difference among those groups of dogs (Table 11 and Fig. 11).

ii) Evacuation of the gastric contents

In all groups of the operated dogs, vomiting and/or diarrhoea were observed frequently. Some of the dogs with intrathoracic vagotomy, or with vagal anastomosis following vagotomy suffered from vomiting for 2 weeks postoperatively. In dogs with intrathoracic esophagogastrostomy, vomiting was generally more refractory and some of them suffered from it for as long as several months. The vomiting after esophagogastrostomy seemed to be independent of the additional vagal anastomosis, and was thought, from the postmortem examinations, to be concerned with operative procedures. However, vagotomy cannot escape responsibility for the vomiting[24)36)47)].

On gastrointestinal X-ray examination performed 2 hours after instillation of barium into the stomach, there remained scarcely any barium in the stomach of non-operated dogs. Gastric evacuation was remarkably disturbed in the 3rd month after vagotomy or vagal anastomosis. However, 10 months after the operation, gastric evacuation in dogs with vagal anastomosis was as complete as that in non-operated dogs in contrast to the vagotomized dogs, which showed considerable retention in the stomach.

In dogs after intrathoracic esophagogastrostomy with or without additional vagal anastomosis, intrathoracic parts of the gastric tubes were observed to be remarkably dilated from the 4th postoperative month. It may be attributed partly to the following factors :

Table 11. Maximal intraluminal pressures of the body of stomach and pyloric region recorded by open-tipped tube method as compared with the basal pressure of duodenum.

Method of operation	Time after operation	Body of the stomach		Pyloric region	
		Amplitude (cm H_2O)	Frequency (per min.)	Amplitude (cm H_2O)	Frequency (per min.)
none		2~5	2.2	10	2.5
none		1	4.5	5~6	5
none		0		11	3.5
none		0~2	8	5~6	6
antethoracic transplantation of the gastric tube	2 weeks	2	4	10	4
antethoracic transplantation of the gastric tube	4 weeks	1	7~8	14~15	2.5
antethoracic transplantation of the gastric tube	4 weeks	7	4.5	22	5
intrathoracic vagotomy	1 month	0~3	irregular	15~16	3
esophagectomy and intrathoracic esophagogastrostomy	8 months	3	6	17~18	4
esophagectomy and intrathoracic esophagogastrostomy with anastomosis of anterior vagal trunk	6 months	2	3.5	10	4
bilateral cervical vagotomy	1 hour	1.3	irregular	12	3
bilateral cervical vagotomy	1 hour	1	2.5	15	2.5

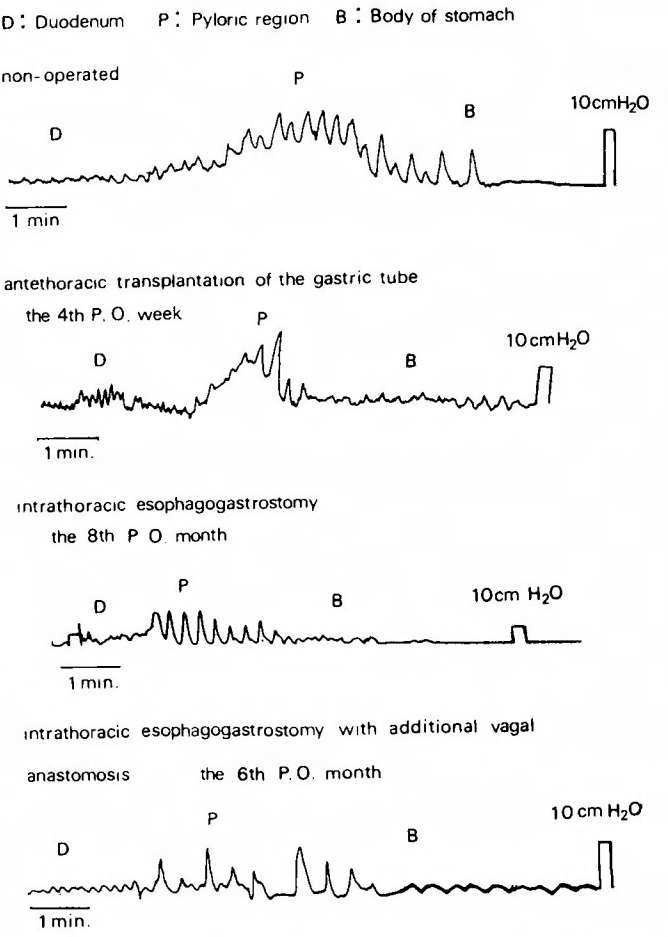

Fig. 11 Gradient of basic intraluminal pressure recorded continuously from duodenum to the body of the stomach by means of drawing out method with an open-tipped tube.

the total gastric tubes were large in size ; the gastric tubes could not be completely covered by the mediastinal pleura ; the gastric tubes and the feeble mediastinal pleura could not tolerate the negative pressure in the thorax. Regurgitation of the barium into the esophagus and suffocation were sometimes observed. There could hardly be found any difference in emptying rate between dogs with esophagogastrostomy and those with additional vagal anastomosis. It can be supposed that the difference in emptying rate, when present, was obscured by the above-mentioned factors, by some difference of operative procedures and byt the fact that the pyloric branch from the anterior vagal trunk could not be reconstructed.

IV. DISCUSSION

For esophageal reconstruction after esophagectomy, gastric tube, pedunculated or free intestinal tube and pedunculated or free colonic tube are utilized. Especially, the last has been regarded recently as most useful. However, judging from the operative risk, for

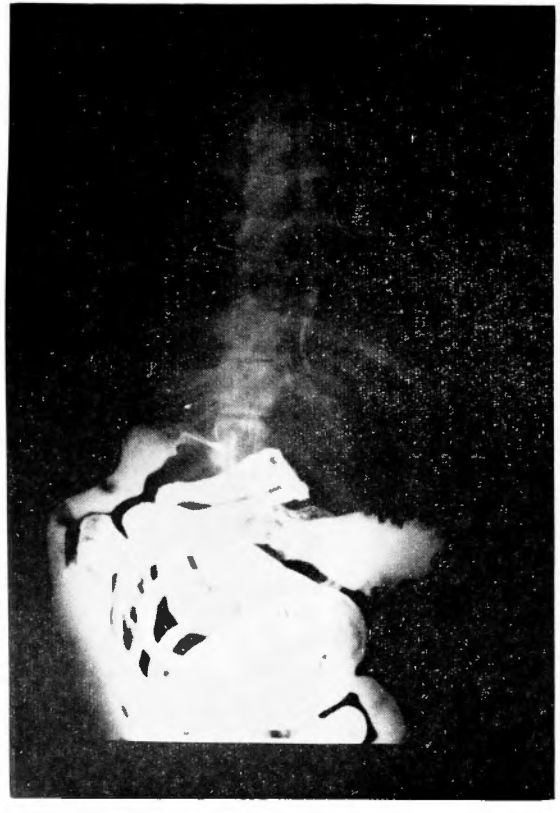

Fig. 12 Gastrointestinal X-ray examination of a non-operated dog 2 hours after instillation of barium into the stomach.

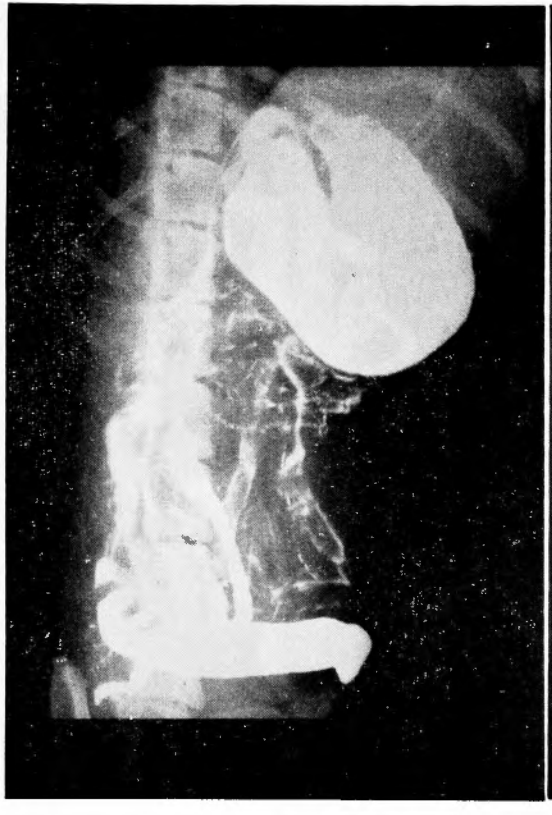

Fig. 13 Gastrointestinal X-ray examination of a dog 3 months after vagal anastomosis. Retention in the stomach is seen 2 hours after instillation of barium.

Fig. 14 Gastrointestinal X-ray examination of a dog 10 months after vagotomy. Two hours after instillation of barium.

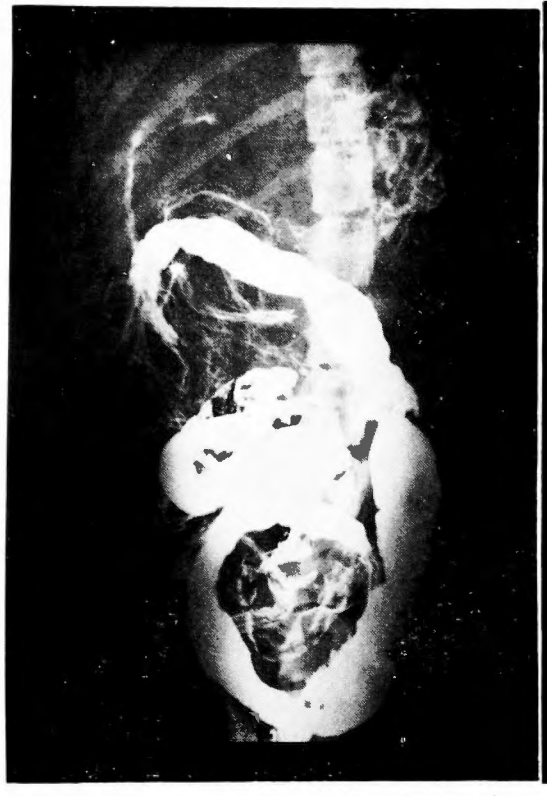

Fig. 15 Gastrointestinal X-ray examination of a dog 10 months after vagal anastomosis. Two hours after instillation of barium.

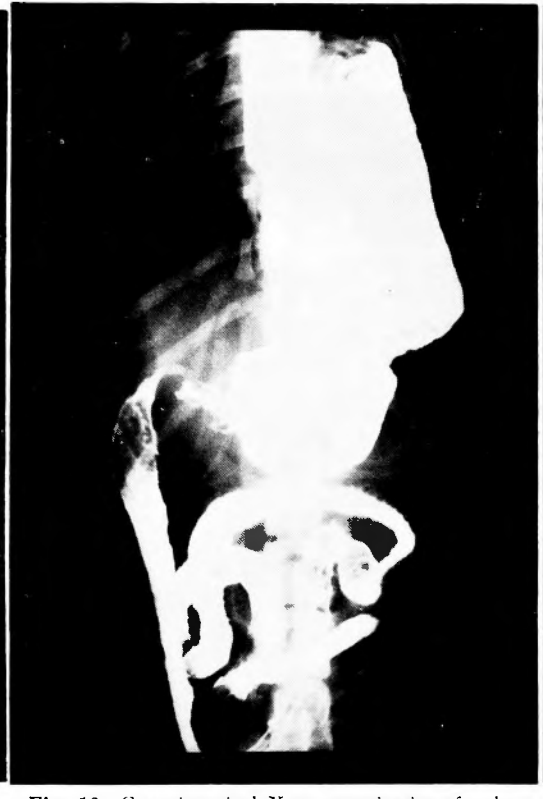

Fig. 16 Gastrointestinal X-ray examination of a dog 4 months after esophagectomy and intrathoracic esophagogastrostomy. Retention in the gastric tube is seen 2 hours after instillation of barium.

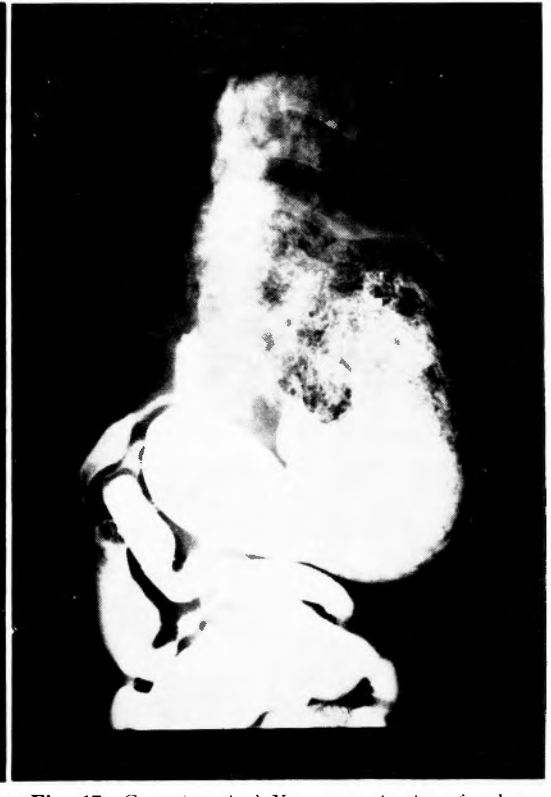

Fig. 17 Gastrointestinal X-ray examination of a dog 6 months after esophagectomy and intrathoracic esophagogastrostomy with additional vagal anastomosis. Two hours after instillation of barium. Gastric tube is markedly dilated.

example, esophageal reconstruction with pedunculated gastric tube, especially with the KIRSCHNER-NAKAYAMA type of gastric tube, has never lost its value.

Although the causes of malnutrition and malabsorption which follow esophageal reconstruction with any method have not been completely clarified, SHILS has supposed that bilateral thoracic vagotomy accompanying esophagectomy is concerned[48]. It is universally admitted that gastric secretion and motility decrease after bilateral thoracic or abdominal vagotomy. After esophageal reconstruction with a pedunculated gastric tube, especially with the KIRSCHNER-NAKAYAMA type of gastric tube, disturbances of secretory and motorial function of the gastric tube are observed, which resulted from unbalance of autonomic innervation of the gastric tube due to bilateral vagotomy, decrease of the size of gastric remnant for secretion and motion, exclusion or damage of the gastroesophageal closing mechanism, and direct operative intervention, especially resultant impaired blood supply to the stomach. Owing to those disturbances, reflux esophagitis is apt to occur, and furthermore, anorexia and malnutrition are impaired. The disturbance of secretory function of the gastric tube is characterized by lack of cephalic phase due to bilateral vagotomy, and a slight decrease of the area having parietal cells owing to partial resection of the stomach near the cardia. KUDO reported from clinical experiences in esophageal reconstruction that the volume of gastric juice in fasting secretion had been decreased for 6 months after both intrathoracic and antethoracic esophagogastrostomy, and that in the stimulating test in the same term neither cephalic nor gastric phase secretion had been found after intrathoracic esophagogastrostomy in contrast to the presence of gastric phase secretion in some cases after antethoracic esophagogastrostomy. Then after the 6th postoperative month all cases after intrathoracic esophagogastrostomy had been found to be anacidic and had shown no response to stimulant and therefore recovery of the secretory function of the remnant stomach had been scarcely found. On the contrary, in the same term after antethoracic esophagogastrostomy, cases which had shown response to stimulant had increased, and free hydrochloric acid in fasting secretion and even cephalic phase secretion had been observed in some cases, and therefore indication of recovery of the secretory function had been noticed[34]. In our clinical cases cephalic phase secretion was abolished for 6 months to 3 years and 3 months after both intrathoracic and antethoracic esophagogastrostomy, as shown in Table 1.

As for the disturbance of motorial function of the gastric tube, bilateral vagotomy and its elevation into the unphysiologic position seem to be concerned. TOCHIKURA observed on the X-ray examinations of the motility of the gastric tubes used for esophageal reconstruction that peristalses of the intrathoracic gastric tubes had been undiscernible due to feebleness at 54 % and cycles of peristalses had been from 10 to 25 seconds at 46 %, while those of the antethoracic gastric tubes were undiscernible at 37 %, and long-term follow-up of those both groups had shown almost the same pattern, and that motor activity of the latter had been more vigorous than that of the former[56]. SHIMOTA reported about the curves of gastric motility by the balloon method after antethoracic esophagogastrostomy on 79 cases that after operation the majority of them had shown atonic type in pylorus and the body of the stomach, and both frequency and amplitude of the curves of intraluminal pressure had decreased, but after the 6th postoperative month the motility of the pylorus and the body of the stomach had recovered to the normal type and both frequency

and amplitude had increased[49].

The author attempted to restore the secretory and motorial functions of the gastric tube by means of restoration of the vagal innervation on the KIRSCHNER-NAKAYAMA type of gastric tube, and determine its effects experimentally.

As for the vagal anastomosis, its functional recovery is considered to be hopeful like the somatic nerves on the basis of experimental studies by TANAKA[54], ISHII[25], and YOSHIMATSU[59]. Although various methods have been adovocated for the nerve suture[19)58], which seems to play an important part in functional recovery of the nerve, the above-mentioned simplest method has been used in this experiment. As the vagal trunk to be anastomosed, the thickest one, moreover, in the easiest position for operative procedure, was selected. As the result, anterior trunk of the vagus nerve was anastomosed in almost all the dogs. MATSUO has reported that anterior trunk of the vagus nerve in dogs chiefly regulates the gastric motility, and the posterior one, the secretion, respectively[38]. In this experiment, however, gastric secretory response to insulin was distinctly observed in dogs with vagal anastomosis of the anterior trunk.

Since PAVLOV gastric secretion has been divided into three phases from its mechanism, namely, cephalic (or nervous) phase, gastric (or antrum) phase and intestinal phase. It has been considered that in cephalic phase, gastric secretion is mediated through the vagus nerve, and in gastric or intestinal phase it is induced by the direct stimulating effect of the contents on stomach or intestine. DRAGSTEDT has assumed that in normal dogs, the nervous mechanism of secretion probably accounts for 45 % of the total amount of gastric juice secreted in 24 hours, the antrum phase 45 %, and the intestinal phase 10 % or less[14]. As the mechanism of gastric secretion has been clarified, gastrin has been recognized to be liberated through the mediation of the vagus nerve besides the local, namely, chemical or mechanical, stimuli to the antrum. Therefore, gastric phase secretion also has been considered to be concerned with vagal innervation[15)28)41]. Accordingly, NYHUS has proposed a new concept of phases of gastric acid secretion as follows : (1) direct vagal, (2) vagal antral, (3) local antral, and (4) intestinal, after he presumed from experiments that 20 to 80 % of the so-called gastric phase secretion has been induced by the gastrin liberated through the mediation of the vagus nerve[41]. JOHNSON has reported that after ingestion vagal component of gastrin release has accounted for 30 % or more of secretory response[28]. Although the intestinal phase of gastric secretion remains mostly unexplained, KELLY has acknowledged influence of vagotomy on it[31], and JORDAN has also admitted participation of nervous mechanism[29]. Therefore, gastric secretion is regarded to be regulated by the vagus nerve in all phases. The fact that gastric secretion is inhibited by vagal denervation of the stomach is universally applied to the medical treatment of peptic ulcer. Thereupon, insulin test, first described by HOLLANDER[23], is used to determine postoperatively the completeness of vagus nerve section. Although the interpretation of the insulin test according to the criteria proposed by HOLLANDER in determining completeness of vagotomy has been a subject of debate[45], it is generally recognized that gastric secretion is induced by insulin hypoglycemia through the mediation of the vagus nerve[2)10)11)46)52)57]. In regard to the insulin test, DAVIS[12] has reported on gastric secretory response to graded insulin hypoglycemia, and HIRSCHOWITZ[21] on dose dependence of

insulin-stimulated gastric secretion. Insulin inhibition of gastric secretion also has been reported[16)44)]. In the author's study 20 units of regular insulin was administered for a test. Although it was documented that gastric secretory response to insulin was reduced by nembutal anesthesia[43)], and in decorticated man[13)], animals in this study were anesthetized with intravenous administration of sodium pentobarbital, 25 mg/kg, because it was difficult to carry out the test on conscious dogs after intrathoracic esophagogastrostomy by means of pouch or fistula. In order to examine the influence of the anesthesia, insulin tests were performed on dogs with gastric fistula both under nembutal anesthesia and without anesthesia. However, no remarkable difference in the gastric secretory response under those conditions was observed between these two groups. Recently, 2-Deoxy-D-glucose has been recognized to induce gastric secretion through the mediation of vagal nerve[22)] as well as insulin. On the other hand, histamine, histalog and gastrin are recognized to induce gastric secretion by direct action on parietal cells. In the histamine test of this study, 1 mg of histamine dihydrochloride was administered for a single test[30)]. Gastric secretory response to histamine also has been generally regarded to decrease after vagotomy [2)3)17)], and in this study it somewhat decreased, as a whole, after vagotomy. Accordingly, recovery of responsiveness to histamine after vagal anastomosis was not so distinct as to insulin. Histamine has been considered to induce chiefly hydrochloric acid secretion, while insulin to induce pepsin besides hydrochloric acid secretion[17)42)]. Therefore, concentration of free hydrochloric acid of gastric juice is generally higher in the histamine test than in the insulin test, and when much free hydrochloric acid is secreted in basal secretion, its increase in response to insulin seems not to be remarkable, even when response to insulin hypoglycemia is present. So, the above-mentioned result of this study probably suggests the maximal concentration of free hydrochloric acid in response to insulin by the method used in this study.

As for the motility of the stomach, there is no divergence of opinion as to the view that generally the vagus nerve accerelates and the greater splanchnic nerve supprsses it. However, the reverse is observed experimentally. For explanation of this phenomenon, MATSUO has presumed the presence of two kinds of nerve fibers, accerelative and suppressive, in these nerves, respectively[38)]. MARTINSON also has presumed the presence of two kinds of nerve fibers in the vagus nerve[37)]. On the other hand, FUKUHARA has interpreted this phenomenon as the intrinsic mechanism of the stomach wall, not as the functional distribution of the nerve fibers[12)]. MATSUO reported, as mentioned above, the functional distribution of anterior and posterior vagal trunks[38)]. Besides these exogenous nervous factors, myenteric plexus, humoral factors and myogenic factors are concerned with the gastric motility[26)27)]. In regard to the regulation of gastric emptying, acid control theory by CANNON[7)] has been well known. However, all of the gastric emptying cannot be explained by this. The important factors concerning gastric evacuation are tonus and peristalsis of the stomach, the former regulates the thickness of the stomach including the pyloric region, and the latter causes pressure gradient between the stomach and duodenum[18)]. These factors are furthermore regulated by the vagus nerve, greater splanchnic nerve, myenteric plexus, humoral factors, and chemical or physical stimuli to the organs. The regulatory effect of the vagus nerve on gastric motility and evacuation has been investigated

chiefly by means of vagotomy, and has been a matter of controversial opinion. ROWE observed in clinical cases by gastrometric studies with balloon that gastric motility had decreased during a period of over six years following vagotomy[46]. KRAFT described postvagotomy gastric atony[33]. MEEK and HERRIN reported about a permanent loss of gastric tonus after vagotomy[5]. LUCKES observed gastric retention following vagotomy[35]. He concluded from his observations that gastric evacuation had been delayed because of suppression of gastric propulsive activity resulting from decreased antral peristalsis and not because of increased tone of the pyloric sphincter[35]. NELSEN observed on dogs after vagotomy 1) a lowering of mean gastric mechanical electrical rate ; 2) alteration of the character of fasting motor activity (diminution of phasic activity, relative accentuation of tonic activity and desynchronization) ; 3) diminution in amplitude and duration of the motor response to food ; and 4) opening of pylorus in a greater percentage of time than the normal[40]. BRODY and MCCREA obtained pressure records in vagotomized dogs indistinguishable as regards basal pressure, frequency and magnitude of phasic changes from those of normal dogs[5]. Reflexes arising on both sides of the gastroduodenal junction which play an important role in regulation of gastric emptying are also controlled by the vagus nerve. For instance, inhibition of gastric motility by duodenal distension, by fat or acid in the intestine, is abolished by vagotomy[9][55]. In general, gastric retention and diminution of gastric motility and tonus have been acknowledged to be induced by bilateral vagotomy from the results of the above-mentioned experimental and clinical studies. Practically, pyloroplasty or pyloromyotomy has been performed for protection from those disturbances in clinical cases. The author examined gastric motility as well as gastric secretory function after vagal reconstruction. The balloon method has been employed from the beginning for examining gastric motility or intraluminal pressure of the stomach, but several disadvantages have been mentioned in this method[5][39][51], and moreover it is hard to examine the pressure gradient and absolute intraluminal pressure by this method. In this study gastric motorial response to drugs acting on the autonomic nervous system were examined by this method on non-operated dogs, vagotomized dogs and dogs with vagal anastomosis. The responses were almost the same, which were convincing from the acting points of those drugs. A tendency was observed that the basic activity of the pylorus in vagotomized dog, and that of the body of the stomach in dogs with vagal anastomosis had exceeded that of the other, respectively. In order to examine further this tendency, the open-tipped tube method was employed[4][5]. Although stimulatory effect of the tube cannot be neglected in the author's method, it was used in this study for the following reasons : A polyethylene tube, even if thinner in diameter, had more stimulative effect on stomach and duodenum due to its hardness when inserted from the mouth to the duodenum. It was difficult to introduce a thinner rubber tube or vinyl tube from the mouth to the duodenum, and there was a risk recording changes of pressure from the side wall of the tube, not from the opening, due to its softness. The open-tipped tube of the author's method gave the most satisfactory result in determining gastric motor activity, and seemed to be more suitable than the balloon method for determining the pressure gradient in the gastroduodenal canal. Using this method, a tendency was observed that the maximal intraluminal pressure of the pyloric region of the vagotomized dog was high and that of the body of the stomach

was low, and that this was somewhat obscured by vagal anastomosis. It is obscure whether the tendency was aroused by primary intraluminal pressure elevation of the pyloric region or by hyperkinesis of the pyloric antrum which resulted from gastric retention owing to pyloric spasm. Gastric motility after ingestion may be different from that after fasting or instillation of water. There is room for reconsideration about the above-mentioned open-tipped tube method, because the tube may be blocked after the ingestion of solid or semisolid food. Although new methods, for example, using implantable silicon strain gaze [39] or inductograph have been presented[35], a further development of methods or apparatuses is expected. Various methods have been employed for the examination of gastric emptying rate, also. X-ray photograph used in this study expresses only the extent of contrast media in a certain plane. In the method for measuring the aspirated gastric content, there is a difficulty in aspirating the whole residue. Recently, new methods for the examination, for example, by scintigram using radioisotope[6][20] and by cineradiograph[8], have been reported. The telemetry capsule for measuring intraluminal pressure will be also useful in the examination of gastric motility and emptying.

From the above-mentioned experimental result, additional vagal anastomosis on the KIRSCHNER-NAKAYAMA type of gastric tube has been proved to contribute to the restoration of the secretory and motorial functions of the gastric tube in long-term follow-up. Although in this attempt there may be many problems to be investigated further, such as, whether the occurrence of vago-vagal reflex and reflux esophagitis are facilitated after additional vagal anastomsis, or how the lymphatic glands near the left gastric artery are treated when the metastasis is present, additional vagal anastomosis on the pedunculated gastric tube seems to be worthy of clinical application as a countermeasure against the dysfunction of the gastric tube for esophageal reconstruction in long-term follow-up which has been disregarded.

It is thought that the vagus nerve takes part in the mechanism of the hunger sensation or appetite besides the secretory and motorial functions of the digestive organs[1]. Therefore, if the regeneration of the afferent fibers in the anastomosed vagus nerve occurs, anorexia, lack of hunger sensation and malnutrition are possibly prevented which most of the patients with esophageal cancer complain of or suffer from, postoperatively.

V. SUMMARY

Under the notion that various disorders after esophageal reconstruction with the KIRSCHNER-NAKAYAMA type of gastric tube were due to the vagal denervation of the gastric tube, secretory and motorial functions of the stomach were examined in four groups of dogs, which had undergone intrathoracic vagotomy, vagal anastomosis following vagotomy, esophagectomy with intrathoracic esophagogastrostomy and esophagogastrostomy with additional vagal anastomosis, respectively.

1) In the clinical cases after esophagectomy with esophagogastrostomy gastric secretory response to insulin was not observed for over 3 years after operation.

2) In vagotomized dogs gastric secretory response to insulin was not observed for 10 months after operation, while in dogs with vagal anastomosis gastric secretory response to insulin was observed from the 4th postoperative month.

3) In dogs after esophagectomy with esophagogastrostomy, gastric secretory response to insulin was not observed for 8 months after operation, while in dogs with esophagogastrostomy with additional vagal anastomosis, gastric secretory response to insulin was observeed from the 3rd postoperative month.

4) No remarkable differences among those four groups of dogs were found in gastric secretory response to histamine, though somewhat decreased after vagotomy. Restoration of the response to histamine after vagal anastomosis were not so distinct as to insulin.

5) In the examination of the muscular activity of the stomach using a balloon, there was no difference among non-operated, vagotomized dogs and dogs with vagal anastomosis in the manner of response to drugs acting on the autonomic nervous system. However, in basic activity, magnitudes of phasic changes of the pyloric region of vagotomized dogs were larger than those of dogs with vagal anastomosis, and, on the contrary, those of the body of the stomach were larger in dogs with vagal anastomosis than in vagotomized dogs.

6) In intraluminal pressure study with an open-tipped tube, a zone of raised intraluminal pressure was observed at the pyloric region in those four groups of dogs. The maximal intraluminal pressures of the pyloric region, as compared with the basal pressures of the duodenum, were higher in vagotomized dogs than in non-operated dogs and dogs with additional vagal anastomosis. On the contrary, the maximal intraluminal pressures of the body of the stomach showed the inverse tendency, that those of non-operated dogs and dogs with additional vagal anastomosis were somewhat higher than those of vagotomized dogs.

7) Gastric emptying rate of dogs with vagal anastomosis was greater than that of vagotomized dogs in long-term follow-up, when examined after instillation of barium into the stomach. But in dogs after esophagectomy with intrathoracic esophagogastrostomy, no distinct difference in gastric emptying rate was observed between those without vagal anastomosis and those with additional vagal anastomosis.

Accordingly, additional vagal anastomosis with the KIRSCHNER-NAKAYAMA type of gastric tube has been proved to contribute to the restoration of the secretory and motorial functions of the gastric tube in long-term follow-up. It seems to be worthy of clinical application as a countermeasure against the dysfunction of the gastric tube for esophageal reconstruction in long-term follow-up which has been disregarded.

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和文抄録

食道再建用有茎胃管への迷走神経縫合追加の効果

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有茎胃管,とくにKirschner-中山式胃管を用いた食道再建術後には,胃管のアトニー,分泌および運動機能の低下,幽門痙攣,さらには種々の食道炎の愁訴や栄養障害を生ずることがある.これには有茎胃管への迷走神経支配は除かれるのに反して,右胃および右胃網動脈にそう交感神経などが残存することによつて発生する自律神経支配の失調状態が少なからず関係をもつているものと考えられる.

われわれは,食道癌手術をうけた患者について胃管の迷走神経支配の有無を検討し,最近発達した細小血管吻合技術を利用して左胃血管系の血行を再建するか,左胃動脈上行枝を保存することによつて,迷走神経末梢側切断端を含めた胃管を食道再建に用いることが可能である点を考慮して,

1) 胸腔内迷走神経切断犬

2) 胸腔内迷切・再縫合犬

3) 食道切除・胸腔内食道胃吻合犬

4) 食道切除・迷走神経縫合追加食道胃吻合犬

を作製し,それぞれについて胃分泌機能検査として

1) ホランダーのインシュリン試験

2) ヒスタミン試験

を行ない,また運動機能検査として

1) バルーンによる運動曲線

2) オープンチップ法による内圧曲線

を描かしめ,また

3) バリウムを胃内に注入してその排出状態を調べた.

これらの結果,

1) 食道切除・食道胃吻合を行なつた臨床例では,術後3年余にわたつてインシュリンに対する分泌反応は認められず,胃管への迷走神経支配の廃絶されていることを確認した.

2) 胸腔内迷切犬では,術後10ヵ月までインシュリンに対する胃分泌反応がみられなかつたのに対して,迷切・再縫合犬では,術後4ヵ月頃より分泌がみられた.

3) 食道切除・胸腔内食道胃吻合犬でも術後8ヵ月までインシュリンに対する分泌反応がみられなかつたが,迷走神経縫合追加食道胃吻合犬では,術後3ヵ月目より分泌がみられた.

4) ヒスタミンに対する胃分泌反応は,各群の間に著明な差がみられず,迷切後にはやや減少したが,迷走神経縫合による回復は著明ではなかつた.

5) バルーンによる胃運動検査では,非手術犬,迷切犬,再縫合犬の間に自律神経作働薬に対する反応の仕方には差が認められなかつたが,静止内圧曲線においては,迷切群では幽門部の圧上昇が大きく,胃体部の圧上昇が小であつたのに対して,再縫合犬ではこの傾向が軽微になつていた.

6) オープンチップ法による内圧検査では,全群で幽門部に著明な内圧上昇帯を認め,その程度は迷切群で著明であつたのに反して,非手術犬,迷走神経縫合群では軽微であつた.また胃体部の内圧上昇はこの逆の傾向を示した.

7) 胃排出機能では,迷切犬に比して,再縫合犬では良好な排出を示したが,胸腔内食道胃吻合群では迷走神経縫合追加によつても著明な差を認めることができなかつた.

すなわち,Kirschner-中山式胃管に迷走神経縫合を追加すると,胃管の遠隔時分泌および運動機能の回復に役立つことが立証された.この方法については,術後逆流性食道炎あるいは逆行性リンパ節転移のある際の処理など検討すべき問題もあるが,従来なおざりにされてきた食道再建用有茎胃管の遠隔時機能障害への対策として,臨床的にも応用する価値があると思われる.