Selective Vagotomy of the Parietal Cell Mass: Part I: With Preservation of the Innervated Antrum and Pylorus

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SELECTIVE vagotomy of the parietal cell mass (Fig. 1), in contrast to selective gastric vagotomy (Fig. 2), was first reported from this laboratory in 1957.³ The purpose of study was to devise an operation that would control the diathesis of duodenal ulcer without causing the dumping syndrome. With the methods available at that time, the results were only partially successful in eliminating the secretory response to insulin and preserving normal gastric emptying. The present study was undertaken with more refined methods to reinvestigate the effects of vagotomy of the parietal cell mass upon gastric motility and secretion; the secretory potential of the innervated antrum was of particular concern.

Preliminary Studies

The feasibility of selective vagotomy of the parietal cell mass was investigated by acute experiments in dogs with the method of electric vagal stimulation and intravenous neutral red.¹¹ The entire mucosal surface of the stomach was exposed by a gastrotomy along the greater curve from antrum to cardia. The proximal border of the antrum was identified by visualizing the neutral red secreted by the parietal mucosa by not by the antral mucosa. With electric stimulation of the vagal trunks, which elicits the secretion of neutral red from the parietal mucosa when the gastric vagi are intact, progressive transection of the end branches of the gastric vagi to the fundus and corpus resulted in progressive elimination of secretion from the parietal mucosa. After all vagal branches to the parietal cell mass were transected, gastric acid secretion ceased. These results confirmed previous results that vagal innervation of the stomach is segmental,¹² and consequently established the anatomic feasibility of complete vagal denervation of the parietal cell mass with preservation of vagal innervation to the antrum. In addition, the operation proved technically feasible.

In the previous study,³ which was conducted without the advantage of accurate delineation of the proximal antral border, selective vagotomy of the parietal cell mass resulted in moderate gastric stasis in four of ten dogs. This stasis was presumed to be due to vagal denervation of the proximal antrum. To test this presumption, the dissection for selective vagotomy of the parietal cell mass was extended 2 cm. distal to the proximal antral border. Subsequent radiologic studies showed moderate gastric stasis. With this result, the importance of correlating the dissection with the proximal antral border was realized.

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FIG. 1. Selective Vagotomy of Parietal Cell Mass. (A) The anterior gastric vagi to the fundus and corpus are transected (A-B) with preservation of the anterior gastric vagi to the antrum. (B) The posterior gastric vagi to the fundus and corpus are transected (C-D) with preservation of the posterior gastric vagi to the antrum. The hepatic vagi (A) and the celiac vagi (B) are preserved.

Material and Methods

Seven adult mongrel Heidenhain pouch dogs weighing from 10 to 15 kilograms were studied before and after selective vagotomy of the parietal cell mass. Gastric emptying was determined by barium meals, and gastric motility was observed by cineradiography. Pouch secretion was measured by 24-hour collections. Completeness of vagotomy of the parietal cell mass was determined by insulin tests and by the method of electric vagal stimulation in the presence of circulating neutral red.



FIG. 2. Selective Gastric Vagotomy. (A) All anterior gastric vagi are transected (A-B) with preservation of the hepatic vagi. (B) All posterior gastric vagi are transected (C-D) with preservation of the celiac vagi. As determined by studies of gastric secretion and motility, vagotomy of the stomach is complete; no efferent secretory or efferent motor function to the stomach from the hepatic and celiac vagi has been demonstrated. The function of the branch of the hepatic vagi to the most distal antrum (A) remains unknown.

Throughout the study the dogs were fed daily at 8:00 a.m. a standard diet of horse meat and gravy, dog food, milk and salt. On the days of the radiographic studies and insulin tests the dogs were fasted. For these examinations the dogs were trained to stand in Pavlov frames.

Operative Technic

Under intravenous nembutal anesthesia the abdomen was entered through an upper incision. The proximal antral border was delineated by first stimulating acid secretion with intravenous histamine and then spraying Congo red into the stomach.¹⁰ Selective vagotomy of the parietal cell mass with preservation of the extramural vagal innervation to the antrum and pylorus was performed as described in the legend of Figure 3.

Barium Meals and Cineradiography

Radiologic studies were conducted before and approximately 1, 2, and 3 months after operation. For each study the dogs were fed 100 ml. of a blended mixture of evaporated milk and barium sulfate in water. The cineradiographic observations were made immediately after the meal and at intervals thereafter. X-rays were taken every hour until the barium meal had left the stomach and small intestine and had entered the colon.

Heidenhain Pouch Secretion

Following a suitable postoperative recovery period of 3 to 4 weeks, 24-hour pouch collections were obtained on consecutive days except on those days when the dogs were fasted for radiologic and other secretory studies. A total of 15 to 30 daily collections was obtained before and after selective vagotomy of the parietal cell mass.

Insulin Tests

Six to 8 weeks after operation a gastric tube was passed through the mouth and

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FIG. 3. Technic of Selective Vagotomy of the Parietal Cell Mass in Heidenhain Pouch Dog. (A) Congo red is sprayed through a gastric tube into the stomach to delineate the proximal border of the antrum. (B) The proximal antral bor-der has been identified by transilluminating the stomach and visualizing the junction between the parietal cell mass (stained black) and the antrum (stained red). At this border the gastric vagi are separated from the lesser curve and encir-cled. (C) Dissection proceeds proximally to separate the lesser omentum completely from the lesser curve. The terminal gastric vagi and vessels are ligated and transected. (D) Selective va-gotomy of the parietal cell mass is complete with preservation of the extramural vagal innervation to the antrum and pylorus.

positioned in the stomach under radiologic control at approximately 10:00 a.m. The aspirate was discarded. Basal secretions were collected at half-hourly intervals until the Pavlov response had subsided and the basal secretion had stabilized. Insulin in doses of 0.5 units per kilogram of body weight was then injected intravenously. Juice from the main stomach was aspirated, and Heidenhain pouch secretion was drained, at halfhourly intervals for 4 hours thereafter. Blood sugar levels were determined from samples drawn before and one-half and 2 hours after insulin.

Gastric Analysis

The volume and pH of each specimen of gastric juice were determined. A sample of each specimen was titrated with 0.01N NaOH to pH 7 as determined by a Beckman pH meter (Model 76). Results of titration were expressed as mEq./l. and mEq.

Electric Vagal Stimulation in the Pres-

ence of Circulating Neutral Red

The study was completed with acute experiments to determine the amount of residual innervation of the parietal cell mass by stimulating the cervical vagus nerves with an electrode after the intravenous injection of neutral red. This method was applied as previously reported.11, 12

Results

Tests for Completeness of Vagotomy

Insulin Tests. The postoperative responses of the main stomach and Heidenhain pouch are presented in Table 1. The following points are noted. First, in those dogs that did produce a basal secretion from the pouch (Dogs, MS1, MS3, and MS10), the pH of the basal pouch secretion was less than the basal secretion from the main stomach. Second, in no instance was a positive response obtained from the Heidenhain pouch. Third, by the criteria

VAGOTOMY OF PARIETAL CELL MASS Kongo red injected В Α ∆ntraÌ borde



	Half houses Secretions After Inculin Injected at 0 House						Bl (m	Blood Sugar (mg./100 ml.)				
	Half-nourly Secretions After Insulin Injected at 0 Hour							After Insulin				
	Basal $\frac{1}{2}-0$	$0 - \frac{1}{2}$	$\frac{1}{2} - 1$	1-1 1	$1\frac{1}{2}-2$	$2-2\frac{1}{2}$	$2\frac{1}{2}-3$	$3 - 3\frac{1}{2}$	$3\frac{1}{2}-4$	Betore Insulin	1/2 Hr.	2 Hrs.
Dog MS1 Stomach												
ml pH mEq./l. mEq.	6.5 6.3 3 0.02	0.7 7.3 0 0	8.8 3 48 0.42	10.7 3.3 65 0.69	5.9 3.1 60 0.35	3.1 3.7 35 0.10	1.2 4.9 17 0.02	2.8 7.4 0 0	0.2 6.9 0 0	66	37	54
Pouch												
ml pH	0.4 4.6	0.3 4.7	0.3 4.6	0.3 5.7	0.2 6.7	0.2 6.4	0.8 7.3	0.4 7.3	0.4 7.3			
Dog MS3 Stomach												
ml pH mEq./l. mEq.	3.5 3.7 26.0 0.08	9.3 5.1 17.0 0.16	6.5 4.2 19.0 0.12	3.7 3.9 30.0 0.11	3.4 3.8 24.0 0.08	5.7 3.9 22.0 0.13	4.3 4.4 12.0 0.05	2.6 5.0 11.0 0.03	4.2 3.5 25.0 0.10	59	37	59
Pouch												
ml. pH	0.2 3.5	0.5 3.9	0.5 3.3	0.3 3.5	0.2 4.0	0	0.3 4.9	0				
Dog MS4 Stomach												
ml. pH mEq./l. mEq.	2.5 7.2 0 0	1.8 6.6 4.0 0.01	5.9 4.4 11.0 0.07	8.3 3.1 47.0 0.39	6.4 2.9 50.0 0.32	1.7 4.1 19.0 0.03	2.1 6.2 4.0 0.01	0.7 6.6 4.0 0.002	0.7 6.9 2.0 0.001	66	40	53
Pouch												
ml. pH	0	0	0	0	0	0	0	0	0			
Dog MS5 Stomach												
ml. pH mEq./l. mEq.	1.3 5.9 18.0 0.02	1.0 5.7 20.0 0.02	1.2 5.3 33.0 0.04	$1.0 \\ 5.3 \\ 31.0 \\ 0.03$	0.3 5.9 7.0 0.002	4.3 4.9 35.0 0.16	6.1 4.4 44.0 0.27	2.6 4.3 45.0 0.12	2.8 4.2 46.0 0.13	65	38	57
Pouch												
ml. pH	0	0	0	0	0	0	0.3 4.5	1.2 4.1	1.8 3.5			
Dog MS8 Stomach												
ml. pH mEq./l. mEq.	2.2 7.5 0 0	2.2 7.9 0 0	2.6 7.1 0 0	3.6 3.3 36.0 0.09	3.7 3.1 50.0 0.18	3.2 2.8 72.0 0.22	4.8 3.1 61.0 0.29	2.3 3.9 30.0 0.07	2.3 5.6 11.0 0.02	62	42	65

TABLE 1. Insulin Responses of Main Stomach and Heidenhain Pouch

								Blood Sugar (mg./100 ml.)				
	Half-hourly Secretions After Insulin Injected at 0 Hour								After Insulin			
	Basal ¹ 2–0	$0 - \frac{1}{2}$	1 1	$1 - 1\frac{1}{2}$	1 <u>1</u> -2	$2-2\frac{1}{2}$	$2\frac{1}{2}-3$	$3.3\frac{1}{2}$	$3\frac{1}{2}-4$	Before Insulin	1/2 Hr.	2 Hrs.
Pouch												
ml. pH	0	0	0	0	0	0	0	0	0			
Dog MS10 Stomach												
ml. pH mEq./l. mEq.	3.2 4.2 39.0 0.11	8.2 3.8 42.0 0.31	4.3 4.7 36.0 0.15	3.6 3.8 38.0 0.14	1.0 4.0 17.0 0.02	2.1 3.8 36.0 0 .07	1.0 4.2 6.0 0.01	2.4 4.7 21.0 0.05	3.2 4.2 24.0 0.05	68	31	41
Pouch												
ml. pH	4.2 2.6	4.4 2.4	2.0 2.5	0.6 2.5	1.0 3.2	0.5 3.2	0.7 3.7	0.6 3.2	4.4 3.1			
Dog MS12 Stomach												
ml. pH mEq./l. mEq.	4.2 7.5 0 0	2.0 7.0 0 0	3.7 7.3 0 0	8.2 5.3 10.0 0.08	0.1 5.4 0 0	0.2 5.4 25.0 0	0.6 6.8 6.0 0.01	1.7 5.2 13.0 0.02	0.4 4.6 15.0 0	64	40	55
Pouch												
ml. pH	0	0	0	0	0	0	0	0	0			

TABLE 1-Continued

of both Hollander⁷ and Stempien,¹⁴ the responses of the main stomach were positive in Dogs MS1 and MS4, and negative in Dogs MS3, and MS5, MS10, and MS12. The response of Dog MS8 was positive by Hollander's criteria but negative by Stempien's criteria. Fourth, all positive responses were low in amount and, for the most part, delayed in onset. This type of small and delayed response has previously been shown to be due to tiny areas of residual innervation in the parietal cell mass.8 Fifth, the total 4-hour secretions after insulin in the dogs with positive responses approximated those of the dogs with negative responses. The mean of the total 4hour positive responses after insulin was 0.84 mEq., which is significantly lower than the mean of 10.12 mEq. obtained by the same methods in four dogs without vagotomy in Part II of this study.

Electric Vagal Stimulation after Intravenous Neutral Red. No neutral red was secreted in Dogs MS1, MS5 and MS12. Dogs MS3, MS4, and MS8 secreted neutral red in tiny areas (less than one cm. in greatest dimension) at the most distal parietal cell mass abutting the proximal antral border. Dog MS10 (with a negative insulin test) was not tested.

Correlation of Results. The results of the insulin tests and neutral red secretion were correlative in Dogs MS4, MS5, MS8, and MS12 (positive insulin tests and neutral red secreted in Dogs MS4 and MS8, and negative insulin tests and no neutral

Dog No. and Time of Study	Stomach Empty	Barium First in Colon	All Barium in Colon
MS1			
Preoperative	5 h.p.c.	3 h.p.c.	5 h.p.c.
Postoperative	- ·		5 h a a
4 weeks	5 h.p.c.	3 n.p.c.	5 n.p.c.
8 weeks	5 h.p.c.	3 h.p.c.	5 n.p.c.
12 weeks	5 h.p.c.	3 h.p.c.	5 h.p.c.
MS3			
Preoperative Postoperative	5 h.p.c.	4 h.p.c.	5 h.p.c.
4 weeks	4 h.p.c.	2 h.p.c.	5 h.p.c.
8 weeks	4 h.p.c.	2 h.p.c.	5 h.p.c.
MSA	-		
Preoperative	2 h.p.c.	2 h.p.c.	3 h.p.c.
Aweeks	4 h n c	4 h n c	5 h.p.c.
4 weeks	5 h.p.c.	3 h.p.c.	5 h.p.c.
12 weeks	4 h.n.c.	3 h.p.c.	5 h.p.c.
12 WCCR5	1 mpror		• • • •
MS5			
Preoperative	3 h.p.c.	3 h.p.c.	4 h.p.c.
Postoperative			
5 weeks	4 h.p.c.	3 h.p.c.	4 h.p.c.
8 weeks	3 h.p.c.	2 h.p.c.	3 h.p.c.
MS8			
Preoperative	3 h.p.c.	2 h.p.c.	3 h.p.c.
1 weeks	2 h n c	2 h n c	3 h.n.c.
4 weeks	2 h.p.c.	2 h.p.c.	3 h.p.c.
12 weeks	2 h.p.c.	2 h.p.c.	3 h.p.c.
MS10	2		
MI310		2	2600
Preoperative	3 n.p.c.	2 n.p.c.	5 n.p.c.
Postoperative	2	2600	3 h n c
4 weeks	3 n.p.c.	2 h.p.c.	3 h.p.c.
8 weeks	з п.р.с.	2 n.p.c.	5 n.p.c.
M S12			
Preoperative	4 h.p.c.	3 h.p.c.	5 n.p.c.
Postoperative			
5 weeks	3 h.p.c.	2 h.p.c.	4 h.p.c.
9 weeks	3 h.p.c.	2 h.p.c.	4 h.p.c.

TABLE 2. Rates of Gastric and IntestinalEmptying of Barium Meal

red secreted in Dogs MS5 and MS12). Dogs MS1 and MS3 showed no correlation (positive insulin test but no neutral red secreted in Dog MS1, and negative insulin test but neutral red secreted in Dog MS3).

Gastric Emptying and Motility

Barium meal x-rays showed no significant difference in the rates of gastric and testinal emptying before and after operation. In Table 2 the rates of emptying are presented in terms of hours after ingestion of the barium (e.g., "stomach empty 5 h.p.c." means that the stomach was empty of barium five hours after ingestion of the meal).

Preoperative cineradiographic studies showed considerably more peristalsis in the antrum than in the fundus and corpus. Powerful peristaltic waves started in the region of the angulus and descended to the pylorus with filling of the duodenal bulb. The barium in the duodenal bulb was then segmented by strong rhythmic contractions and propelled distally in small portions. The relatively inactive fundus and corpus gradually decreased in size as the antrum and proximal duodenum rhythmically emptied the stomach. Postoperative studies showed no significant changes in gastric motility and emptying except in Dog MS1. At the first examination of this dog 6 weeks after operation, relatively sluggish antral peristalsis and poor duodenal filling were observed and the distal antrum appeared contracted. However, the stomach did empty as rapidly as before operation (Table 2); at the eleventh week repeat cineradiographic examination showed a return of gastric motility to the preoperative state.

Heidenhain Pouch Secretions

The means and standard deviations of each dog before and after operation are shown in Table 3. Statistical analysis of these results by the Student t Test shows that the postoperative increase in pouch secretion is statistically significant in Dogs MS1, MS3, MS4, MS5, and MS10 and insignificant in Dogs MS8 and MS12. The mean increase is 47 per cent.

Discussion

Successful results with selective vagotomy of the parietal cell mass are dependent upon accurate delineation of the proximal antral border. If the dissection is extended distal to this border with transection of the vagal fibers entering the proximal antrum, gastric stasis results. On the other hand, if Volume 170 Number 2

the dissection is not carried to the proximal antral border, the most distal parietal cell mass remains innervated (Dogs MS3, MS4 and MS8). However, the secretory potential of small areas of residually innervated parietal mucosa is quite low. In general, the results of this study are considered successful in eliminating the secretory response to insulin and preserving normal gastric motility and emptying.

The results of the insulin tests indicate that the release of gastrin from the innervated antrum after selective vagotomy of the parietal cell mass is insignificant in the fasting state. However, the results of the 24-hour Heidenhain pouch secretions indicate that the over-all release of gastrin is highly significant. The postoperative increase in the 24-hour pouch secretions is presumably due to a reduction in acid secretion from the main stomach which, in turn, decreases the mechanisms of antral and duodenal acid inhibition and thereby increases the output of gastrin. Results of feeding tests, which are the subject of a separate report,¹ suggest that the 24-hour pouch responses in this study are largely due to the ingestion of food and not to the vagal release of gastrin in the fasting state.

The mean 47% increase in 24-hour pouch secretion in this study is significantly greater than the 22% mean increase observed in a previous study¹³ of selective gastric vagotomy (Fig. 2) without complementary drainage and consequent gastric stasis. These comparative results imply that the innervated antrum without stasis in the present study releases more gastrin than the denervated antrum with stasis in the previous study. (This implication pertains only to selective vagotomy, for in the previous study the 22% mean increase in 24-hour pouch secretion after selective vagotomy was increased by 82% after total vagotomy. The mechanisms for this additional increase in secretion by hepatic and celiac vagotomy remain unknown.)

The permanence of selective vagotomy

TABLE 3. Twenty-four Hour HeidenhainPouch Secretions

	Mean mE	Comparison	
Dog	Preop.	Postop.	t Test
MS1	$\begin{array}{r} 8.22 \\ \pm 1.54 \end{array}$	$\begin{array}{r} 11.70 \\ \pm 4.08 \end{array}$	$t 2.89 \\ p < 0.005$
MS3	$\begin{array}{r}13.84\\\pm 5.54\end{array}$	$\begin{array}{r} 25.39 \\ \pm 8.67 \end{array}$	$t 5.48 \ p < 0.0005$
MS4	$\begin{array}{r} 14.07 \\ \pm \ 2.14 \end{array}$	$\begin{array}{r} 22.52 \\ \pm 8.99 \end{array}$	$t 4.12 \ p < 0.0005$
MS5	$\begin{array}{r} 8.38 \\ \pm 1.85 \end{array}$	$\begin{array}{r} 16.08 \\ \pm 4.97 \end{array}$	$t 6.95 \ p < 0.0005$
MS8	$\begin{array}{r} 33.76 \\ \pm 7.87 \end{array}$	36.77 ±12.88	t 0.88 m.s.
MS10	57.70 ±15.64	94.49 ± 20.14	$t & 6.35 \\ p < 0.0005$
MS12	$\begin{array}{r} 18.74 \\ \pm 3.43 \end{array}$	$\begin{array}{r} 18.73 \\ \pm 3.62 \end{array}$	t 1.35 m.s.

of the parietal cell mass may be questioned by the possibility of vagal innervation by the phenomenon of a sprouting 2 from the residually reinnervated antrum. Although this question cannot be definitely answered at this time, recent evidence from this laboratory indicates that the amount of mucosal reinnervation by sprouting from a localized area of innervated mucosa is insignificant.⁹

Hart ^{5, 6} has recently reported successful preliminary results with selective vagotomy of the parietal cell mass plus pyloroplasty in patients with duodenal ulcer. The longterm results of gastric secretion and recurrent ulcer in his patients are as yet unknown. The addition of pyloroplasty probably reduces the release of gastrin from the innervated antrum by providing faster gastric emptying.⁴ In three of the seven dogs reported herein, Finney pyloroplasty was performed after the completion of the secretory and radiologic studies of the effects of selective vagotomy of the parietal cell mass. Repeat radiologic studies showed that pyloroplasty altered the normal rhythmic emptying of the stomach and hastened gastric emptying. These changes most probably account for the dumping syndrome consequent to pyloroplasty.

Whether selective vagotomy of the parietal cell mass without drainage would control the diathesis of duodenal ulcer is only conjectural. The principal question concerns the increased Heidenhain pouch secretion due to the increased release of gastrin from the innervated antrum. This study was therefore continued by investigating selective vagotomy of the parietal cell mass in combination with suprapyloric antrectomy. This combination is the subject of Part II.

Summary and Conclusions

Gastric motility and secretions were studied before and after selective vagotomy of the parietal cell mass with preservation of the innervated antrum in 7 Heidenhain pouch dogs. The procedure caused no gastric stasis and preserved normal rhythmic emptying of the stomach. Postoperative insulin responses were eliminated in 4 dogs and virtually eliminated in 3 dogs. However, the 24-hour Heidenhain pouch secretions increased by a mean of 47 per cent.

It was concluded that clinical application of this operation would eliminate the dumping syndrome, but might not control the diathesis of duodenal ulcer because of the high secretory potential of the innervated antrum.

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