Selective and Total Vagotomy without Drainage:

A Comparative Study of Gastric Secretion and Motility in Dogs

EIICHI SHIINA,* M.D., DR. OF MED. SCI., CHARLES A. GRIFFITH,** M.D., M.SC., F.A.C.S.

From the Department of Surgery, University of Washington School of Medicine, Seattle, Washington 98105

BURGE[2] has recently initiated a trial of selective vagotomy without complementary drainage in patients who have no stenosis at the gastric outlet from the cicatrix of ulcer. He has determined the absence of stenosis by the ready passage of a wide-bore tube from the stomach into the duodenum distal to the ulcer. In patients with stenosis, as indicated by failure to pass the gastric tube beyond the ulcer, pyloroplasty has been performed as previously described.

Burge's trial of selective vagotomy without drainage was based upon his considerations that preservation of the pylorus would prevent the dumping syndrome consequent to pyloroplasty, and that preservation of the hepatic and celiac vagi to the antrum and intestine would preserve enough motility for adequate emptying of the stomach when its outlet is completely patent. Although his initial results have been satisfactory,[3] we were skeptical of long-term results because of gastric stasis. In a previous study of selective vagotomy without drainage in dogs,[7] gastric stasis was observed but its effect upon Heidenhain pouch secretion was not determined. We, therefore, undertook the present study to compare the effects of selective and total vagotomy upon Heidenhain pouch secretion and gastrointestinal motility.

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* Visiting Scientist, on leave from the Department of Surgery, Keio University School of Medicine, Tokyo, Japan (Director: Professor Nobukatsu Shimada).

** Clinical Associate Professor of Surgery.

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Material and Methods

Five adult mongrel dogs equipped with Heidenhain pouches underwent selective vagotomy at a first stage and total vagotomy at a second stage. Before and after each stage gastric emptying was measured by barium meals and gastric secretion was measured by 24-hour pouch collections. The completeness of vagotomy was determined by insulin tests after selective vagotomy, and after total vagotomy by the method of electric vagal stimulation in the presence of circulating neutral red.

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 insulin tests and radiologic studies the dogs were fasted.

Operative Technics

Stage I: Selective Vagotomy. Through a lengthy left subcostal incision, selective vagotomy was performed by transection of the gastric vagi at the most distal esophagus and gastric cardia. The descending branch of the left gastric artery was ligated and transected high on the lesser curve in order to interrupt those posterior gastric vagi that sometimes accompany this artery to the stomach. This dissection mobilized and separated the distal esophagus and gastric cardia from the vagal trunks and their hepatic and celiac divisions. Both trunks and the hepatic and celiac vagi were preserved.

Stage II: Total Vagotomy. Through the same subcostal incision total vagotomy was performed by selective hepatic and celiac vagotomy. All hepatic and celiac vagal fibers were identified in the lesser omentum and gastropancreatic fold respectively, and all were transected. The vagal trunks and gastric vagi were not dissected so that a possibly incomplete gastric vagotomy by the initial selective technic was not converted into a complete gastric vagotomy by the total technic.

Heidenhain Pouch Secretions

The method of 24-hour pouch collection was used rather than a shorter period of collection after feeding for two reasons. First, it was presumed that selective vagotomy would result in gastric stasis,[7] and that in the presence of gastric stasis the response of the pouches over 24 hours would be more informative than a response of 6 to 8 hours for a feeding test. Second, the results could be compared with the results of similar experiments with total vagotomy previously conducted in this laboratory.[17, 22, 25]

Following a suitable period of 3 to 5 weeks after each operation to permit full postoperative recovery, the pouch secretions were collected on successive days with the exception of those days when the dogs were fasted for insulin tests and radiologic studies (and also on those occasional days when the secretions were lost because of tearing of the collection balloons). A total of 30 collections was obtained before and after each stage of the experiment.

The volume in milliliters of each 24-hour specimen was measured, and the mEq./l. of each specimen was determined by titration against 0.01N NaOH to pH 7 with phenol red as the indicator. The results were expressed in terms of mEq. of total titratable acid.

Barium Meals

The dogs were fed 100 ml. of a blended mixture of evaporated milk, water, and barium sulfate. X-rays were taken at hourly intervals thereafter until the meal had left the stomach and entered the colon. At least three studies were obtained before and after each stage of the experiment. The first barium meal after each stage was conducted just before the period of Heidenhain pouch collections, the second meal during this period, and the third immediately after this period.

Tests for Completeness of Vagotomy

At 4 to 6 weeks after selective vagotomy insulin tests were conducted with the dogs in Pavlov stands. A gastric tube was passed through the mouth and positioned in the stomach under radiologic control. Basal secretions were aspirated for at least one hour prior to the intravenous injection of insulin in doses of 0.5 unit/Kg. of body weight. The gastric juice was aspirated in half-hourly aliquots for 4 hours thereafter. The total titratable acidity in mEq. was determined for each specimen. A satisfactory degree of hypoglycemia was achieved in each test as confirmed by an adequate decrease in blood sugar after insulin.

Upon the completion of all studies after total vagotomy, acute experiments were performed upon each dog to determine the areas of residual innervation of the gastric mucosa by electric stimulation of the cervical vagus nerves in the presence of circulating neutral red.[20]

Postmortem Dissections

Attempts were made to dissect the vagal system in order to ascertain the anatomic integrity of the abdominal vagi. The main purpose was to determine whether any hepatic and celiac vagal fibers were inadvertently transected during the selective vagotomy consequent to surgical error and anatomic variation in the course and distribu-

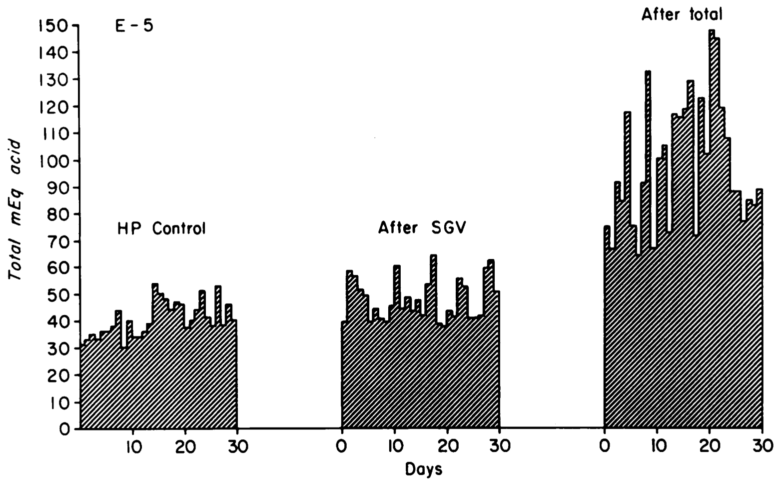

FIG. 1. See text.

tion of the hepatic and celiac vagi to the antrum and mid-gut. All dissections were thwarted by dense postoperative adhesions and consequently failed in their purpose.

Results

Completeness of Vagotomy

The insulin tests of dogs E5 and E11 after selective vagotomy both showed positive responses of 0.4 mEq. of total titratable acid secreted during the last half of the second hour after insulin. This small and delayed type of response to insulin has been previously shown to be due to a small area of residual innervation by a small branch from a major gastric truncal division to a segment of gastric fundus.[15] Small areas of residual innervation in the proximal fundus (measuring two centimeters in greatest dimension) were confirmed in both dogs after total vagotomy by the secretion of neutral red from these areas consequent to cervical vagal stimulation.

The insulin tests of dogs E19, E23, and E25 after selective vagotomy were negative. After total vagotomy no neutral red was secreted with vagal stimulation.

Some but not all specimens of gastric juice collected during the insulin tests were

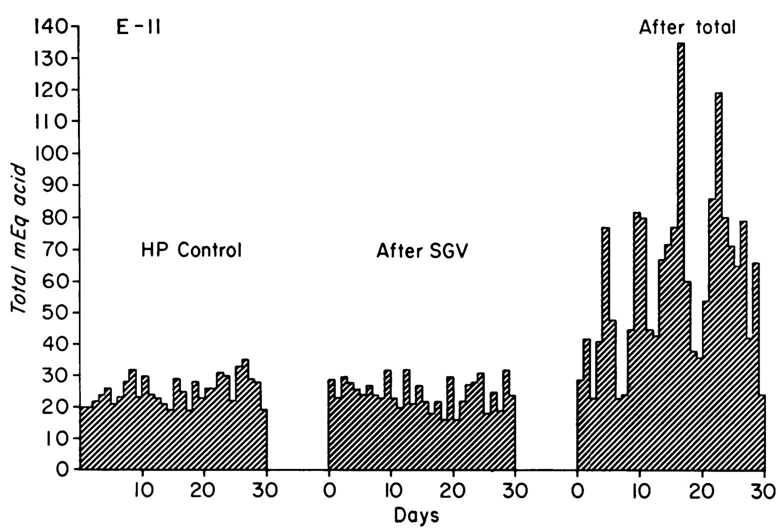

FIG. 2.

contaminated with bile. The validity of the tests in which this contamination occurred was therefore questioned. However, the results of insulin tests after selective vagotomy correlated exactly with the results of the tests with neutral red after total vagotomy in all dogs.

Barium Meals

Control results before selective vagotomy were quite reproducible. The stomach gradually emptied of the meal within 4 to 5 hours. The barium first entered the colon at 2 to 3 hours, and by 6 to 7 hours was all in the colon.

After selective vagotomy all dogs recovered quickly, ate well, and showed little signs of gastric stasis. However, severe gastric stasis with retention of barium in the stomach up to 24 hours was found on the first x-ray studies 2 to 3 weeks postoperative. Variable degrees of recovery of gastric emptying were noted. By the eighth postoperative week the stomach of dogs E11, E23, and E25 emptied of barium after 5 hours, but barium was retained in the stomachs of dogs E5 and E19 up to 10 hours. In most studies the little barium that did enter the duodenum was carried to the colon in the normal transit time, so that on review of successive films little if any barium was seen in the small intestine.

After total vagotomy all dogs recovered slowly and ate poorly. Dogs E5 and E11 vomited repeatedly and required clyses. Whether these effects were caused by the trauma of an additional operative procedure, which was more extensive than the previous selective vagotomy because of adhesions, or by the sequelae of hepatic and celiac vagotomy, could not be determined. Gastric stasis was severe in all dogs at the first barium meal 2 weeks postoperative with retention of barium up to 24 hours. Thereafter, as after selective vagotomy, gastric emptying partially recovered by the eighth postoperative week. No significant difference in gastric stasis after selective and total vagotomy was noted. However, variable degrees of small intestinal dilatation and stasis were seen in one or more studies of all dogs after total vagotomy, but in only one examination of one dog (E19) were these changes impressive (delay in intestinal transit time up to 8 hours).

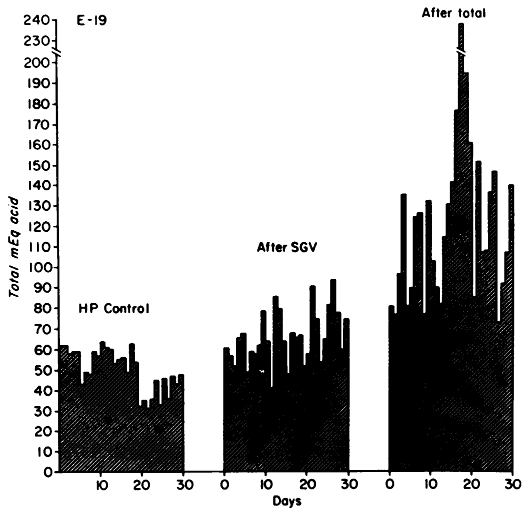

FIG. 3.

Heidenhain Pouch Secretions

The results of each dog are presented in graphic form (Figs. 1–5). In each figure the graphs on the left ("HP Control") show the secretion before selective vagotomy, the middle graphs ("After SGV") show the secretion after selective vagotomy, and the graphs on the right ("After

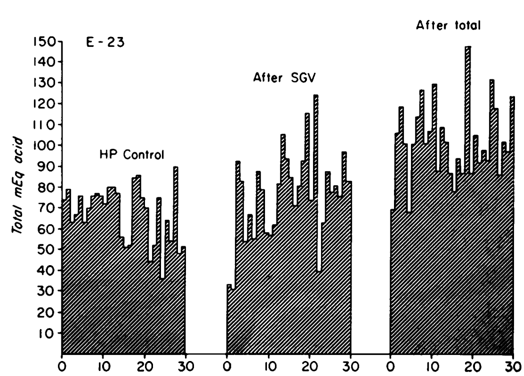

FIG. 4.

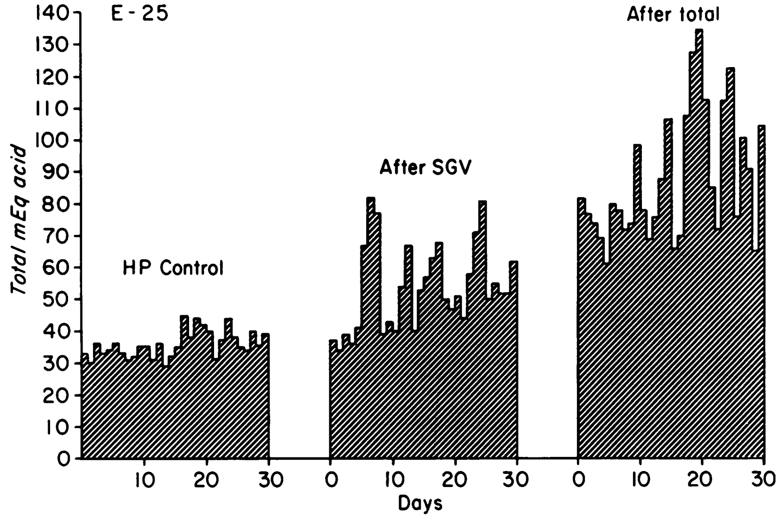

FIG. 5.

Total") show the secretion after total vagotomy.

The means and standard deviations are shown in Table 1. The data in Table 1 were subjected to statistical analysis by the Student t test. The relatively small increase in secretion after selective vagotomy was statistically significant in dogs E5, E19, and E25, questionably significant in dog E23, and insignificant in dog E11. The larger increase in secretion after total vagotomy was statistically significant in all dogs (Table 2), and was comparable in magnitude to that previously observed in this laboratory.[17, 22, 25]

Discussion

Explanation of Results

Since the early 1950's, when the effects of total vagotomy and various drainage procedures upon Heidenhain pouch secretion were first reported,[4, 12, 17, 22, 25, 26] gastric stasis with an excessive release of gastrin from the antrum has been considered the chief causative factor for the marked increase in pouch secretion after total vagotomy without drainage. Our findings after selective vagotomy without drainage—namely, gastric stasis with relatively little increase in pouch secretion—suggest that mechanisms other than antral stasis merit more attention than previously accorded.

The results of the tests for completeness of gastric vagotomy after the selective and total technics indicate that the same degree of completeness of vagotomy was maintained throughout this study in each dog. Furthermore, the evidence of previous studies in both dogs and man suggests that selective vagotomy lowers gastric secretion as effectively as total vagotomy.[1, 20] Therefore, the difference in Heidenhain pouch secretions after selective and total vagotomy most probably cannot be explained by a difference in completeness of gastric vagotomy or by a difference in decrease of gastric secretion.

A difference in gastrointestinal motility may be a factor. The results of a previous study [24] indicate that only the gastric vagi carry efferent motor fibers to the stomach, and therefore gastric motility should be decreased equally by selective and total vagotomy. However, it may be speculated that the addition of intestinal hypomotility by total vagotomy causes more antral stasis because the stomach may not drain as well into an intestine with stasis as into an intestine without stasis. The evidence for more gastrointestinal stasis after total vagotomy in the present study is suggested by the eating habits of the dogs, which ate relatively normally after selective vagotomy but were anorexic and vomited after total vagotomy. On the other hand, despite the finding of intestinal dilatation and stasis in some but not all of the barium studies after total vagotomy, thorough review of all barium studies after selective and total vagotomy fails to establish any impressive difference in gastrointestinal motility.

This evidence suggests that no significant difference exists or that its significance is not clearly brought to light by this crude radiologic method. Furthermore, if antral and intestinal stasis were the only mechanisms at play, the Heidenhain pouch secretions should show a decrease corresponding to the recovery of gastrointestinal motility as demonstrated by the barium meals during the 30-day periods of pouch collections. On the contrary, as shown in Figures 1–5, the secretions did not decrease but remained as high at the ends of the periods of collections as at the beginnings. Therefore, the difference in Heidenhain pouch secretions after selective and total vagotomy cannot be explained solely on the basis of a difference in gastrointestinal motility.

Lacking any other explanation, we postulate that the relatively small increase in Heidenhain pouch secretion after selective vagotomy is due to an inhibition of gastric secretion by the intact hepatic and celiac vagi, and the large increase in pouch secretion after total vagotomy is due to elimination of this inhibition by transection of the hepatic and celiac vagi. This postulation is based upon previous experimental findings that suggest but do not confirm a role of the hepatic and celiac vagi in the mechanisms of gastric acid inhibition. For example, Hart and his group in Munich[8, 9] propose that acid inhibition by the antrum is dependent upon its vagal innervation. The findings of Rheault et al.[21] may be interpreted as evidence in support of this proposal. Whether antral acid inhibition is

TABLE 1. *Means and Standard Deviations of Total Titratable Acid (mEq.) after Each Operative Procedure*

	Control	Selective Vagotomy	Total Vagotomy
E 5	40.53 ± 6.58	47.27 ± 8.02	96.53 ± 23.58
E 11	25.30 ± 4.49	24.63 ± 4.75	59.77 ± 26.87
E 19	50.43 ± 10.6	65.97 ± 12.95	120.23 ± 38.86
E 23	67.37 ± 13.82	76.40 ± 22.08	102.27 ± 18.47
E 25	35.77 ± 4.26	53.67 ± 13.62	87.83 ± 20.38
Group data	43.88 ± 16.72	53.59 ± 22.20	93.33 ± 32.96

TABLE 2. *Statistical Comparison of Operative Procedures by the Student "t" Test*

	Control c̄ SVG	Control c̄ Total Vagotomy	Selective c̄ Total Vagotomy
E 5	t = 3.55, $p < 0.001$	t = 12.53, $p < 0.001$	t = 10.84, $p < 0.001$
E 11	t = 0.59, N.S.	t = 6.93, $p < 0.001$	t = 7.05, $p < 0.001$
E 19	t = 5.08, $p < 0.001$	t = 9.49, $p < 0.001$	t = 7.26, $p < 0.001$
E 23	t = 1.90, $p < 0.05$	t = 8.29, $p < 0.001$	t = 4.92, $p < 0.001$
E 25	t = 6.87, $p < 0.001$	t = 13.70, $p < 0.001$	t = 7.64, $p < 0.001$
Group data	t = 4.28, $p < 0.001$	t = 16.38, $p < 0.001$	t = 12.25, $p < 0.001$

influenced by the gastric vagi that descend along the lesser curve to the antrum, or by the hepatic vagal fibers that accompany the right gastric artery to the antrum, or both, is unknown. If the hepatic vagi to the antrum do affect antral acid inhibition, it may be further postulated that the hepatic vagi to the proximal duodenum (and perhaps the celiac vagi) affect duodenal acid inhibition as well. This influence of the hepatic and celiac vagi upon duodenal acid inhibition is suggested by the experiments of Landor,[14] who observed in Heidenhain pouch dogs with antrectomy that selective hepatic and celiac vagotomy increased the pouch secretion. Lastly, the findings of Kelly *et al.*[15] and Middleton *et al.*[16] suggest that the hepatic and celiac vagi may also serve to inhibit gastric secretion by mechanism other than antral and duodenal acid inhibition. In Heidenhain pouch dogs with selective vagotomy and total gastrectomy with gastroduodenostomy (a preparation that excludes all mechanisms of acid inhibition), total vagotomy results in a significant increase in pouch secretion. The authors' explanation for this increased pouch secretion on the basis of intestinal stasis and a consequent increased release of "intestinal gastrin"[23] remains to be confirmed.

The foregoing postulations raise the question whether the variable degrees of increase in pouch secretion after selective vagotomy were due to variable degrees of preservation of the hepatic and celiac vagi. More specifically, were some tiny hepatic and celiac vagal fibers to the antrum and duodenum transected by the selective technic in dogs E5, E19, and E25 with the significant increase in pouch secretion, and were all hepatic and celiac fibers preserved in dogs E11 and E23 with the insignificant and questionably significant increase in pouch secretion? This question remains unanswered because, as stated in Methods, the postmortem dissections failed to establish the exact anatomic integrity of the hepatic and celiac vagi after selective vagotomy in each dog.

The Need for Routine Complementary Drainage

In the late 1940's, when Dragstedt's vagotomy was performed without a drainage procedure, Franksson[5] evaluated selective vagotomy without drainage in hopes that preservation of the hepatic and celiac vagi would eliminate the frequent complication of gastric stasis. His hopes were not realized, and he[6] soon abandoned selective vagotomy without drainage in favor of subtotal gastric resection. In those years Jackson[10, 11] also abandoned anterior total-posterior selective vagotomy without drainage because of gastric stasis. However, Franksson and Jackson did not report a correlation between the degree of gastric stasis and the degree of partial gastric outlet obstruction from the cicatrix of ulcer. Nor did Dragstedt report any correlation in his early experience, but his late results[18] suggested that this correlation may exist. Approximately half of his patients with total vagotomy without drainage were cured of ulcer and had no symptoms of gastric stasis. Poor results in the other half of the patients included recurrent ulcer due to incomplete vagotomy and gastric stasis due to cicatricial gastric outlet obstruction and gastric hypomotility. Some patients with gastric stasis developed complications of gastritis and gastric ulcer, which were explained on the basis of gastric hypersecretion consequent to antral stasis. These poor clinical results, plus the corroborative experimental results of Heidenhain pouch hypersecretion after total vagotomy[22, 25] and a reduction of this hypersecretion by antral drainage,[4, 17] led to the recommendation and general acceptance of routine complementary drainage procedures.

By whatever the mechanisms, it is clear from the results of this study that selective vagotomy without drainage in dogs does not cause the gastric hypersecretion that results with total vagotomy. If these results apply to man, and if the degree of postvagotomy gastric stasis may be correlated with the degree of stenosis from the cicatrix of duodenal ulcer, then Burge's trial of selective vagotomy without drainage in patients without stenosis should result in a much lower rate of gastritis and gastric ulcer than formerly experienced with total vagotomy without drainage. The long-term results of his trial are therefore awaited with interest. Should his results fail because of gastric stasis and pyloroplasty prove necessary, the available experimental evidence suggests that selective vagotomy plus pyloroplasty may lower gastric secretion more effectively than total vagotomy plus pyloroplasty. This suggestion is currently under investigation.

Summary and Conclusions

Selective vagotomy without a drainage procedure was performed upon five Heidenhain pouch dogs at a first stage, and total vagotomy was performed at a second stage. The pouch secretions increased only slightly after selective vagotomy but increased greatly after total vagotomy. This difference in Heidenhain pouch secretions, as determined by studies of secretion from the main stomach and gastrointestinal motility, could not be explained by any differences in completeness of gastric vagotomy or decrease in secretion from the main stomach or gastrointestinal stasis. It was postulated that the difference in Heidenhain pouch secretions after selective and total vagotomy may be due to inhibition of gastric secretion by the hepatic and celiac vagi. The mechanisms for this inhibition, if they exist, remain unknown.

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