Effect of Proximal Gastric, Complete Gastric, and Truncal Vagotomy on Canine Gastric Electric Activity, Motility, and Emptying

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PROXIMAL GASTRIC VAGOTOMY without a "drainage" procedure is currently under trial as an operative treatment for duodenal ulcer.[2,11,13,20] The proponents state that the operation effectively decreases the secretion of HCl and pepsin by the stomach, thereby allowing the ulcer to heal, while preserving normal gastric motility.[1,2,11–13,20] Indeed, fewer postoperative side effects have been reported after proximal gastric vagotomy than after several other anti-ulcer operations.[2,4,8,11–13,18–20,24,29,33] However, the effect of proximal gastric vagotomy on gastric motility and emptying has not been extensively studied.

Our hypothesis is that the vagally innervated proximal stomach regulates gastric emptying of liquids by controlling gastric transmural pressure and that the vagally innervated antrum regulates gastric emptying of solids by controlling the terminal antral contraction. Thus, proximal gastric vagotomy should speed gastric emptying of liquids but not alter gastric emptying of solids, while complete gastric vagotomy or truncal vagotomy should speed emptying of liquids and slow emptying of solids. We tested this hypothesis in dogs by studying gastric transmural pressure and gastric emptying of liquids and solids before and after proximal gastric, complete gastric, and truncal vagotomy.

Materials and Methods

Five healthy, mongrel, female dogs, 8 to 12 Kg. in weight, were used. They were alert and conscious during all observations and had been fasted at least 18 hours. The dogs also were conditioned to the passage of an oral-gastric tube.

Four sets of control experiments were done in the dogs before vagotomy. In the first set of experiments, performed four times in each dog, gastric transmural pressure was measured before and during gastric distention with a flaccid, plastic bag of capacity greater than 800 ml. Gastric transmural pressure is that pressure due solely to contractions of the gastric wall. The pressure was recorded as the difference between the pressure inside and that outside the gastric wall.

The pressure inside the stomach was measured by connecting a pressure transducer (Statham P23Dd) via an oral-gastric tube to the plastic bag used to distend the stomach. The pressure outside the gastric wall was measured using a sterile, plastic catheter (ID 1 mm) placed intraperitoneally and attached via a three-way connector to a second transducer. A continuous infusion of 154 mM NaCl at 0.1 ml./min was given through the third channel of the connector to keep the intraperitoneal catheter patent. The infusion did not alter the pressure recorded from this catheter. The electric signals from the two pressure transducers were fed into a differential amplifier, and the difference between the two signals was recorded (Heathkit Servo recorder). The apparatus was calibrated before and after each experiment by the use of water manometers.

The intragastric bag was inserted through the dog's mouth into the stomach, anchored in place using a bite block, and then distended with water at 37.5°C,

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first to 50 ml., and then to 100 ml., and finally, in 100-ml. increments, to 800 ml. Continuous records of the gastric transmural pressure were obtained for 3 minutes at each volume of distention, and the mean pressure was later calculated from the area under each 3-minute curve.

In the second set of prevagotomy experiments, gastric emptying of 400 ml. of 154 mM NaCl with 0.00035% polyethylene-1, 2-^{14}C-glycol (PEG, New England Nuclear) of specific activity 0.6 mCi/g was assessed five times in each dog. An oral-gastric tube was inserted, and the saline was instilled into the stomach during a period of 30 to 45 seconds. The tube was then removed. The instillate was at 37.5°C when given, and had a pH of approximately 6.5. Ten minutes after instillation, the gastric tube was reinserted and the gastric content was withdrawn by siphoning and gentle aspiration with a syringe. The stomach was washed with 100 ml of saline, and the wash was aspirated to recover any residual instillate. If particulate matter was found on aspiration of the original instillate, the experiment was discarded.

At the completion of each test, the volumes of gastric aspirate and gastric wash recovered were measured. The specific activities of 5-ml. samples of original instillate, aspirate, and wash were determined using the liquid scintillation spectrometer (Packard Tri-Carb 3375). The volume of instillate emptied from the stomach in the 10-minute period was then calculated using the following formulas:

$$(1) \qquad V_L = 400 - V_R \quad \text{and}$$

$$(2) \qquad V_R = V_A \cdot \frac{SA_A}{SA_I} + V_W \cdot \frac{SA_W}{SA_I},$$

in which V_L represents the volume of the original instillate emptied from the stomach in 10 minutes, 400 ml. is the volume originally instilled, V_R is the volume of the original instillate remaining in the stomach at 10 minutes, V_A is the volume of the gastric aspirate, V_W is the volume of the gastric wash, and SA_I, SA_A, SA_W are the specific activities of the instillate, gastric aspirate, and gastric wash, respectively.

A third set of control experiments, performed five times in each dog, was done in an identical fashion to the above, except that a 400-ml. instillate of 500 mM glucose with 0.00035% PEG was used instead of the saline instillate.

In the last set of control experiments, gastric emptying of 40 solid plastic spheres was assessed five times in each dog. The spheres were made of polypropylene (Orange Products, N.J.) and were 7.14 mm. in diameter. They were made radiopaque by imbedding two small lead pellets in their center; this also increased their specific gravity to approximately 1.02. The spheres were placed in the dog's stomach via an oral-gastric tube, and the tube was removed. The number of spheres emptied from the dog's stomach was determined fluoroscopically every 30 minutes with the dogs in their normal prone (lying) position until all spheres had emptied or 8 hours had passed. The pattern of gastric motor activity was also noted. A similar technic has been used in this laboratory in the past.[31]

The dogs were operated on while under anesthesia with pentobarbital given intravenously, and a proximal gastric vagotomy was performed (Fig. 1). All structures between the anterior nerve of Latarjet and the lesser curvature of the stomach from the esophagogastric junction to the point of passage of the nerve onto the gastric antrum were then ligated and divided as close to the stomach as possible (Fig. 1 *A*).[20] Through an opening in the greater omentum, a similar procedure was done on the posterior side of the stomach, preserving the pos-

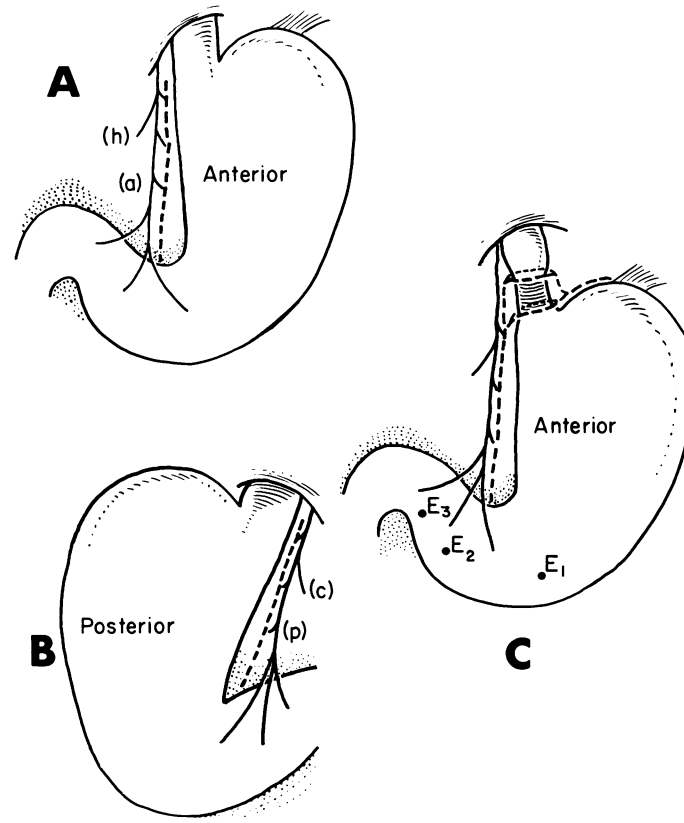

FIG. 1. Proximal gastric vagotomy and sites of attachment of gastric electrodes. *A*, Vagal denervation of anterior proximal stomach (*dotted lines*), with preservation of anterior nerve of Latarjet (*a*), and hepatic branch (*h*) of anterior vagal trunk. *B*, Vagal denervation of posterior proximal stomach (*dotted line*), with preservation of posterior nerve of Latarjet (*p*), and celiac branch (*c*) of posterior vagal trunk. *C*, Completed dissection (*dotted lines*), with esophagus bared to circular muscle for 2 cm. above esophagogastric junction. Esophagus is only remaining structure passing to stomach along its lesser curvature between angle of His and points of passage of nerves of Latarjet onto gastric antrum. Three dots indicate positions of gastric electrodes (*E*).

terior nerve of Latarjet (Fig. 1 *B*). The distal esophagus was then mobilized, and all tissue external to the circular muscle layer was divided for a distance of 2 cm. above the esophagogastric junction. This step ensured division of any additional vagal fibers coursing along the esophagus from the proximal vagal trunks to the stomach. At this point, the bared esophagus was the only structure passing to the stomach along the lesser curvature between the angle of His and the points of passage of the anterior and posterior nerves of Latarjet onto the gastric antrum (Fig. 1 *C*).

Monopolar silver wire electrodes were then implanted on the gastric wall. The electrodes were used primarily to enable us to verify whether vagal innervation of the antrum was intact or disrupted after recovery from the operations. When vagal innervation of the antrum is intact, action potentials appear in the antral electric tracings within 15 to 25 minutes after insulin is given intravenously.[22]

Each electrode was 1 mm in diameter and projected 1½ to 2 mm. from a dental acrylic (Acrolite) disk. The shafts of the electrodes were insulated with vinyl paint, but the tips were exposed and AgCl was deposited electrolytically on them. Each electrode was soldered to a Teflon-insulated copper lead 12 inches long, and the soldered junction was encased within and insulated by the acrylic disk. Epoxy resin was used to seal the points at which electrode and wire penetrated the disk surface. Two holes and one notch were made in the disk to facilitate fixation to the gastric wall. The insulated lead from each disk was connected to a pin of a radiotube socket mounted with epoxy resin and dental acrylic in a stainless steel cannula.

Three electrodes were sutured to the anterior serosal surface of the stomach 2 cm. from the greater curvature at positions 8, 4, and 2 cm. from the pylorus. The metal cannula to which the copper leads were attached was positioned in and sutured to the left anterior abdominal wall in the midclavicular line.

The dogs were allowed 1 month to recover from the operation, after which experiments identical to those done before vagotomy were repeated during the second postoperative month. In addition, on separate days, gastric electric activity was recorded in each dog for 10 minutes before and 45 minutes after the intravenous injection of 1.5 units/Kg. of regular insulin.

The dogs were then operated upon a second time, and the gastric antrum was vagally denervated (Fig. 2 *A*). All structures passing to the stomach along the lesser curvature from the esophagogastric junction to 1 cm. beyond the gastroduodenal junction, including the nerves of Latarjet, were ligated and divided as close to the gastric wall as possible. The duodenum was then circumscribed down to, but not through, the longitudinal muscle at a level 1 cm. aborad to the gastroduodenal junction. The greater omentum was also ligated and divided along a line about 1 cm. from the greater curvature from the duodenum to the approximate junction of gastric antrum and corpus. This operation, coupled with the first operation (proximal gastric vagotomy), effected a complete gastric vagotomy.

The dogs were allowed 1 month to recover from the complete gastric vagotomy, after which experiments identical to those performed after proximal gastric vagotomy were repeated.

The dogs were then operated upon a third time, and a transthoracic truncal vagotomy was performed at a level about 5 cm. above the diaphragm (Fig. 2 *B*). After a 1-month recovery, identical tests to those done before vagotomy were repeated.

The dogs were then sacrificed, and autopsies were performed.

Results

The dogs tolerated all three operations and the electric apparatus well. They remained healthy throughout the experiment.

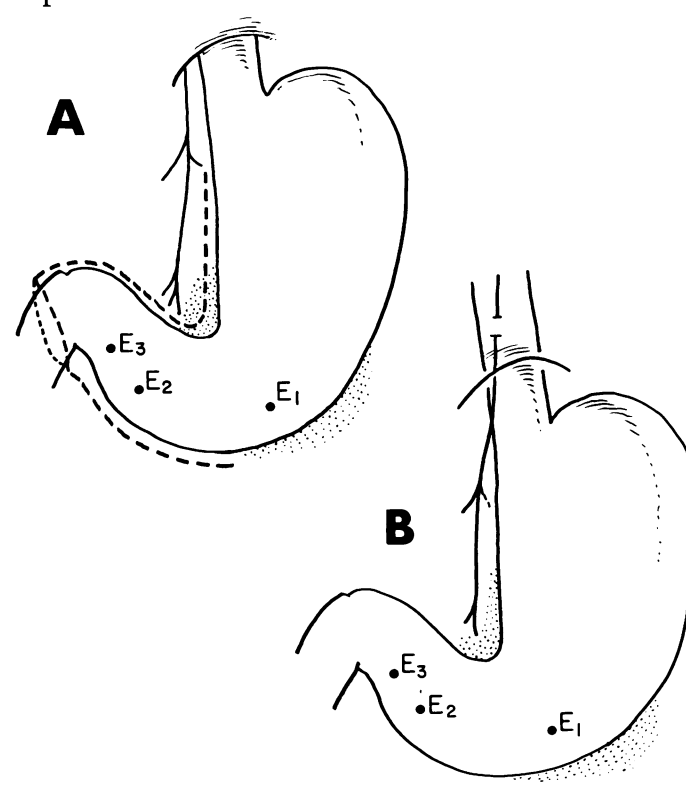

FIG. 2. *A*, Vagal denervation of gastric antrum. *Dotted lines* along greater and lesser curvatures indicate transection and those around duodenum 1 cm. beyond gastroduodenal junction indicate incision down to but not through longitudinal muscle. Anterior and posterior nerves of Latarjet are schematically represented by one nerve. This procedure, together with proximal gastric vagotomy (Fig. 1), effected a complete gastric vagotomy. *B*, Truncal vagotomy. One-centimeter segments of vagal trunks were excised about 5 cm. above diaphragm, and both cut ends tied.

Before Vagotomy. Gastric Transmural Pressure. Gastric transmural pressure was successfully measured in four of the five dogs. Dog E would not tolerate the oral-gastric tube and intragastric bag long enough for satisfactory measurements to be made.

Gastric transmural pressure increased slowly and gradually with gastric distention. The mean pressure increased from 5.0 to 14.0 cm. H_2O as the intragastric bag was distended from 50 to 800 ml. (Fig. 3). The overall mean slope of the regression of pressure on volume was 1.2 cm. H_2O/100 ml.

Gastric Emptying. Gastric emptying of *liquids* was also successfully measured in only four of the five dogs. Again, dog E would not tolerate the instillations.

The dogs emptied a mean of 121 ml. of the saline instillate and a mean of 26 ml. of the glucose instillate from the stomach in 10 minutes. The rate of emptying of glucose was significantly slower than that of saline (Table 1, $p < 0.01$).

The overall pattern of the gastric movement of *spheres* in dogs before vagotomy was one of retention of spheres in the antrum, with continuous vigorous mixing and grinding interspersed between periodic bursts of emptying.

TABLE 1. *Effect of Proximal Gastric (PGV), Complete Gastric (CGV), and Truncal Vagotomy (TV) on Gastric Emptying of Liquids*

	Mean ml. emptied in 10 minutes*							
	NaCl, 154 mM				Glucose, 500 mM			
		After				After		
Dog	Before vagotomy	PGV	CGV	TV	Before vagotomy	PGV	CGV	TV
A	115	200	207	159	9	102	139	83
B	80	199	266	230	21	79	211	131
C	151	195	224	174	14	87	159	125
D	139	207	155	92	61	138	92	58
Grand mean	121	200	213	164	26	102	150	99

* Each value for individual dog is mean of five experiments. Grand means are means of 20 experiments. SE was less than 30 for all values except NaCl of dog C after PGV, when it was 37. For significance, see text.

The spheres passed through the oral-gastric tube directly into the fundus and proximal corpus of the stomach. They were then promptly moved into the distal corpus and antrum where they were continuously mixed and ground by peristalsis and the terminal antral contraction. An advancing peristaltic wave propelled the spheres up against the gastroduodenal junction. The spheres were then squeezed by the forceful terminal antral contraction, until, unable to pass into the duodenum, they suddenly shot back into the more proximal antrum and distal corpus. This propulsion, squeezing, and retropulsion occurred over and over, thoroughly triturating the spheres.

At certain times, as a peristaltic wave approached the terminal antrum, bunches of spheres would be moved rapidly from the terminal antrum through the gastroduodenal junction into the duodenum. Sometimes, all 40 spheres would be emptied within a 30-minute period. However, the overall mean time from insertion to completion of gastric emptying was 2.2 hours (Fig. 4).

After Proximal Gastric Vagotomy. Electric Activity. Electric recordings were satisfactory in four of the five dogs. The electrodes in dog D "shorted out" in the early postoperative period.

A cyclic variation in potential, the gastric pacesetter potential,[23] was consistently detected in the gastric wall 1 month after proximal gastric vagotomy. The cycles were always present and generally appeared in a regular rhythm at a frequency of about five cycles per minute (Fig. 5). The frequency was usually identical at all recording sites. However, the duration of the cycles sometimess varied from one cycle to another (Fig. 5 *upper left* and *lower right*), an occurrence that is uncommon in dogs with normal stomachs.[23] Also, in two

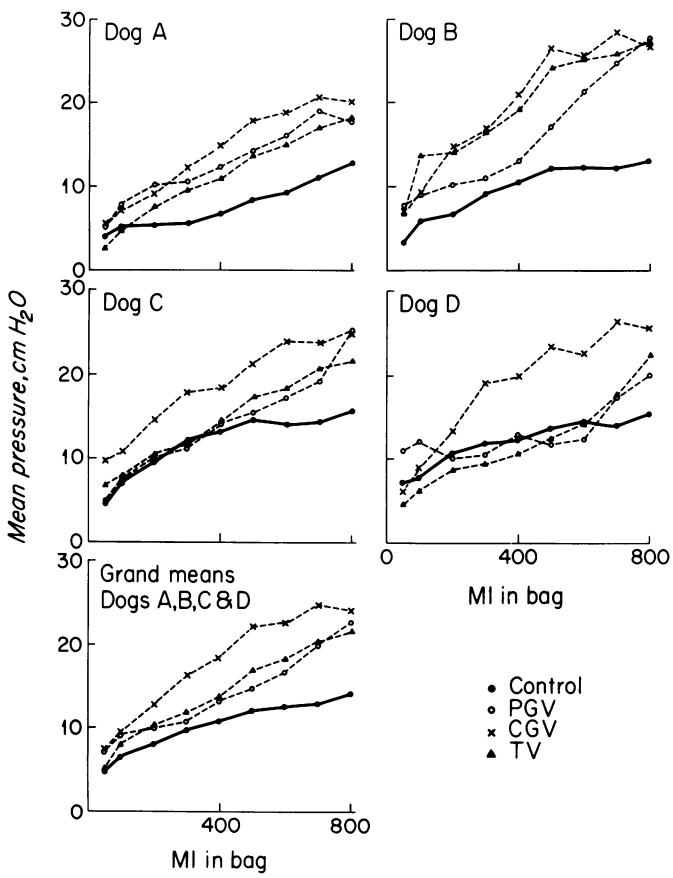

FIG. 3. Gastric transmural pressure increased more with distention of intragastric bag after proximal gastric (PGV), complete gastric (CGV), and truncal vagotomy (TV) than it did before vagotomy. For each dog, each point represents mean of four experiments. For grand means, each point represents mean of 16 experiments.

of the four dogs, episodes of completely disorganized gastric electric activity were found occasionally during fasting (Fig. 6), a phenomenon also rare in normal dogs, but more common in dogs after truncal vagotomy.[22]

Action potentials, the electric activity associated with contractions,[23] occurred intermittently with the cycles of the pacesetter potential before the injection of insulin (Fig. 5). But, insulin stimulated the appearance of action potentials with every cycle of the antral pacesetter potential within 20 minutes of its administration in every test in every dog (Fig. 5). Thus, vagal innervation of the antral musculature was intact.[22]

Gastric Transmural Pressure. After proximal gastric vagotomy, gastric transmural pressure increased more rapidly with distention than it did before vagotomy. The pressure increased from a mean of 7 cm. H_2O to a mean of 23 cm. H_2O as the intragastric bag was distended from 50 to 800 ml. (Fig. 3). The overall mean slope of the regression of pressure on volume was 1.9 cm. H_2O/100 ml. This slope was greater than that found before vagotomy ($p < 0.01$). In all dogs but dog D, individual mean slopes were also greater after proximal gastric vagotomy than before ($p < 0.01$). However, even in dog D, when the intragastric bag contained 700 and 800 ml., the pressure was greater after proximal gastric vagotomy than it was before vagotomy.

Gastric Emptying. Proximal gastric vagotomy speeded gastric emptying of liquids (both saline and glucose instillates) compared to before vagotomy (Table 1, $p < 0.01$). But, glucose still emptied more slowly than saline in all four dogs after proximal gastric vagotomy ($p < 0.01$).

In contrast to the speeding of emptying of liquids after proximal gastric vagotomy, there was no significant change in the rate of emptying of *spheres* compared to before vagotomy. Also, the pattern of movement of spheres noted fluoroscopically was unaltered. The overall mean emptying time of 1.5 hours did not differ significantly from the 2.2 hours found before vagotomy.

After Complete Gastric Vagotomy. Electric Activity. After complete gastric vagotomy, the pattern of fasting, gastric, electric activity was similar to that found after proximal gastric vagotomy. However, insulin no longer triggered the onset of action potentials in any of the four dogs tested after complete gastric vagotomy (Fig. 5). Thus, vagal innervation of the antrum was no longer intact.[22]

Gastric Transmural Pressure. After complete gastric vagotomy, gastric transmural pressure increased from a mean of 7 cm. H_2O to a mean of 24 cm. H_2O as the intragastric bag was distended from 50 to 800 ml. (Fig. 3). The overall mean slope of the regression of pressure on volume was 2.4 cm. H_2O/100 ml. This slope was

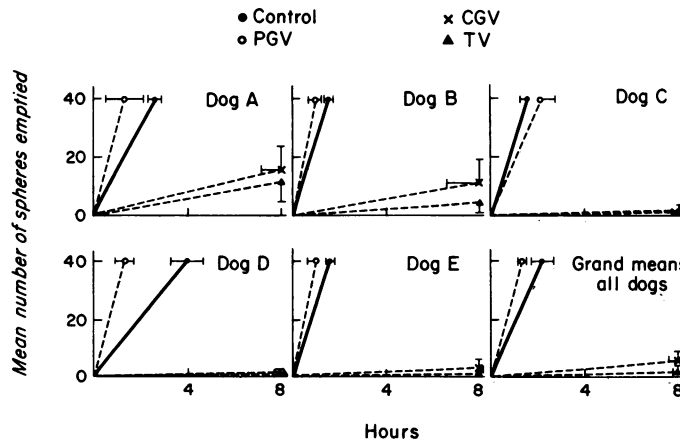

FIG. 4. Mean hours required to empty 40 radiopaque solid spheres from stomach were not significantly altered after proximal gastric vagotomy (PGV), but were greatly lengthened by complete gastric (CGV) and truncal vagotomy (TV). For individual dogs, each point is mean of five experiments. For grand means, each point is mean of 25 experiments. Standard error indicated by bars.

greater than that obtained before vagotomy ($p < 0.01$) but was not different from that found after proximal gastric vagotomy. In all four dogs, the individual mean slopes were greater than before vagotomy ($p < 0.01$). In dog D alone, there was an increase in slope between proximal gastric vagotomy and complete gastric vagotomy.

Gastric Emptying. After complete gastric vagotomy, *liquids* (both saline and glucose) continued to empty faster than before vagotomy ($p < 0.05$ and 0.01, respectively), but neither instillate emptied faster than it did after proximal gastric vagotomy. Glucose still emptied slower than saline after complete gastric vagotomy ($p < 0.01$).

In contrast to proximal gastric vagotomy, complete gastric vagotomy markedly slowed the rate of emptying of *spheres* compared to before vagotomy (Fig. 4). After the spheres were inserted into the stomach, they were promptly moved into the distal corpus and antrum, just as they were before vagotomy and after proximal gastric vagotomy. But the peristaltic waves, though present, were weak and often faded out in the antrum after complete gastric vagotomy, never reaching the pylorus. In addition, the terminal antral contraction was also weak and ineffective with little propulsion or retropulsion of spheres taking place.

The overall mean time from insertion to complete emptying of spheres was greater than 8 hours in the dogs after complete gastric vagotomy, compared to 2.2 and 1.5 hours noted before vagotomy and after proximal gastric vagotomy, respectively (Fig. 4, $p < 0.01$). In only 2 of 25 experiments did the dogs empty all spheres in 8 hours or less after complete gastric vagotomy. On some occasions, the spheres remained in the stomach for as long as 3 days.

After Truncal Vagotomy. Gastric Transmural Pressure. Gastric transmural pressure increased from a mean of 5 cm. H_2O to a mean of 22 cm. H_2O as the stomach was distended from 50 ml. to 800 ml. (Fig. 3). The overall mean slope of the regression of pressure on volume was 2.1 cm. H_2O/100 ml. and was greater than that found before vagotomy ($p < 0.01$) but was not different from that after proximal gastric vagotomy or complete gastric vagotomy. In all four dogs, individual mean slopes were greater than before vagotomy (p 0.01). In no dog was there a change in slope between complete gastric vagotomy and thoracic truncal vagotomy.

Gastric Emptying. After truncal vagotomy, *liquids* (both saline and glucose) emptied more slowly than they did after complete gastric vagotomy (Table 1, $p < 0.01$), so that the rate of emptying of saline no longer differed from that before vagotomy, although glucose still emptied faster than it did before vagotomy ($p < 0.01$). Glucose continued to empty more slowly than saline after truncal vagotomy ($p < 0.01$).

The rate of emptying of *spheres* was just as slow after truncal vagotomy as it was after complete gastric vagotomy. The sluggish motor activity noted fluoroscopically after complete gastric vagotomy was also found after truncal vagotomy. In all experiments, the dogs required 8 hours or more to empty the spheres from their stomachs (Fig. 4). The spheres often remained in the stomach for several days.

Postmortem Findings. At autopsy, minimal deposition of fibrous tissue was present around the electrodes, but no adhesions were found between the stomach and other abdominal organs. There was no evidence of mechanical obstruction of the stomach, the gastroduodenal junction, or the small or large bowel. A metal probe, diameter 1.5 cm., could be passed across the gastroduodenal junction without difficulty. The gastric and duodenal mucosa appeared normal, with no evidence of ulceration or inflammation. Postresection scarring prevented identification of structures along the lesser curvature, but the vagal trunks along the thoracic esophagus could be found, and all had been ligated and severed. One dog (dog D) had an extremely distended gallbladder, but no obstruction could be found in the cystic or common ducts, and no gallstones were present.

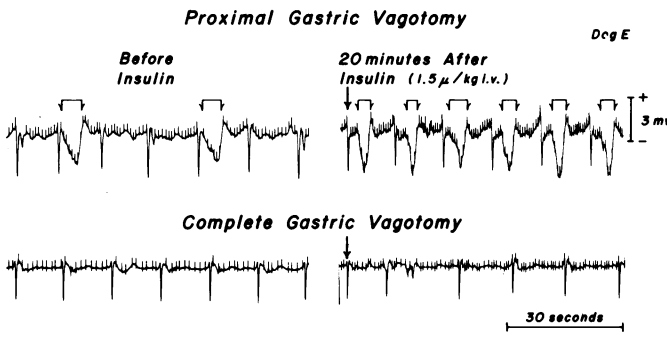

FIG. 5. Injection of insulin intravenously stimulates appearance of action potentials (*connected arrows*) with each cycle of pacesetter potential after proximal gastric vagotomy, but not after complete gastric vagotomy. All recordings from electrode E_3.

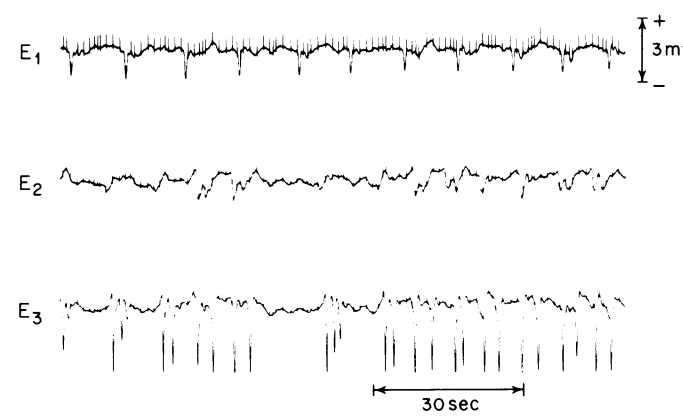

FIG. 6. Episode of disorganized gastric electric activity occurring 1 month after proximal gastric vagotomy in dog E. Frequency of gastric pacesetter potential is different at all three recording sites and increases to 18 cycles/min for brief periods at E_3. *E*, electrode.

Discussion

Our experiment shows that proximal gastric vagotomy preserves gastric motility and emptying better than complete gastric or truncal vagotomy. However, proximal gastric vagotomy does not leave gastric motility unaltered.

The fundus and proximal corpus of the stomach act as a reservoir, relaxing to receive and store ingested food.[5] As a result of this accommodation by the proximal stomach, gastric transmural pressure increases very little with increasing gastric distention.[36]

The vagal nerves have an important role in the accommodation of the stomach to distention. Others have found that greater increases in intragastric pressure occurred with gastric distention when the vagal nerves were transected,[27,30,32] and that electric stimulation of the distal cut ends of the vagal trunks could relax a distended stomach.[28] Our experiment supports these findings. Furthermore, our data show that it is the proximal gastric vagal fibers that mediate this response.

Humphrey and Wilkinson[13] have reported that early satiety and a sensation of epigastric fullness occur in patients after proximal gastric vagotomy. These symptoms are consistent with the failure of relaxation and accommodation to ingesta by the fundus and corpus.

We found that gastric emptying of isotonic saline and hypertonic glucose was consistently speeded after proximal gastric vagotomy. Clarke and Williams[6] also found that hypertonic glucose emptied faster after proximal

gastric vagotomy than before vagotomy, but Humphrey and Wilkinson[13] did not. The explanation for the discrepancy between our findings and those of Humphrey and Wilkinson is unclear. Perhaps because Humphrey and Wilkinson did not compare gastric emptying in the same subjects before and after proximal gastric vagotomy, they were unable to detect a change.

Gastric emptying of liquids occurs when the intraluminal pressure in the stomach exceeds that in the duodenum and the pylorus is open.[35] Greater pressures in the stomach have been correlated with faster gastric emptying.[36] Thus, the increased gastric pressure caused by the failure of the stomach to accommodate to distention after proximal gastric vagotomy found in these experiments is responsible for the speeding of gastric emptying of liquids that we noted after the operation.

The impaired ability of the stomach to secrete HCl after proximal gastric vagotomy may have contributed to the increased speed of gastric emptying that we found after the operation. Hunt and Knox[14] have shown that acids slow gastric emptying. Thus, if less acid was secreted in response to the instillates after proximal gastric vagotomy, gastric emptying would be faster. However, the amount of acid secreted in response to the instillates used in these experiments was most likely very small. Furthermore, any speeding of gastric emptying brought about by a decreased secretion of HCl would be antagonized by the concurrent augmented release of gastrin also accompanying a more alkaline gastric pH.[10,17] Gastrin slows gastric emptying of liquids.[15] Further experiments are needed, but it seems unlikely that decreased secretion of acid by the stomach after proximal gastric vagotomy explains the marked speeding in gastric emptying of liquids that we found.

The rapid gastric emptying of liquids brought about by proximal gastric vagotomy persisted after complete gastric vagotomy. However, after truncal vagotomy, emptying of both saline and glucose solutions was slower than it was after complete gastric vagotomy. Transection of the extragastric abdominal vagi may result in the increased release of an extragastric hormone that slows gastric emptying, such as gastrin,[25,26] secretin, or cholecystokinin. But, the exact mechanism of the slowing when the extragastric vagi are cut is unknown.

We found that hyperosmolar glucose emptied more slowly than isotonic saline after each of the three vagotomies. Thus, the mechanism by which the duodenal osmoreceptors slow gastric emptying is not solely dependent on vagal pathways.

In contrast to the proximal stomach, which stores ingesta and regulates the gastric emptying of liquids, the distal stomach regulates the gastric emptying of solids. The terminal antral contraction rhythmically and forcefully propels and retropels solids, mixing and grinding them, and breaking them apart.[5] Solids are retained in the stomach by the terminal antral contraction until they are reduced to a size small enough for discharge into the duodenum. The antrum, thus, regulates the gastric emptying of solids.[7]

The pace of gastric peristalsis and of terminal antral contractions is set by a gastric pacemaker located in the orad corpus along the greater curve.[34] The pacemaker regularly generates gastric pacesetter potentials which sweep aborad in the gastric wall and phase the onset of peristaltic and terminal antral contractions.[23] Vagal denervation of the pacemaker by proximal gastric vagotomy disturbed its regularity slightly during fasting and occassionally allowed multiple, ectopic, rapid pacemakers to appear in the distal stomach. These disturbances, however, were not great enough to alter gastric motor responses to solid spheres.

Proximal gastric vagotomy, by preserving antral vagal innervation, preserved the terminal antral contraction, so that the pattern of gastric emptying of spheres was unchanged after the operation. Others have also observed that antral contractions are not greatly altered by proximal gastric vagotomy,[3,9,21] and that gastric emptying of food mixed with contrast media is normal.[12,13,16] In contrast, when the antrum was denervated by complete gastric or truncal vagotomy in our experiments, the terminal antral contraction was greatly impaired, resulting in a marked slowing of gastric emptying of spheres.

Summary

Proximal gastric vagotomy disturbed canine gastric motility and emptying less than did complete gastric vagotomy or truncal vagotomy. Although all three vagotomies increased gastric transmural pressure with gastric distention and speeded gastric emptying of liquids, only proximal gastric vagotomy preserved the terminal antral contraction and did not slow gastric emptying of solids. Our experiments identify the key role of the vagally innervated proximal stomach in regulating gastric emptying of liquids by controlling gastric transmural pressure and the key role of the vagally innervated antrum in regulating gastric emptying of solids by controlling the terminal antral contraction.

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Discussion

Dr. Lloyd M. Nyhus (Chicago): The vagus nerves have come a long way since Pavlov. Stimulation of the parietal cell to secrete hydrochloric acid, vagal release of the hormone, gastrin, interrelationship of the vagus and the hormones, secretin, and cholecystokinin and now, the beautiful dissection of the role of the vagus in control of gastric pressure and motility by Wilbur and Kelly.

These studies, confirm by elegant methodology, that preservation of the vagal nerves to the antrum allow normal or near normal function of the antrum and pylorus.

Following proximal gastric vagotomy, cinefluorography in our own patients demonstrates this continued antral function dramatically.

The authors have given us more information. They have differentiated between the regulation of gastric emptying of liquids by the vagally innervated proximal stomach by a transmural