Esophagitis:

Diagnosis and Surgical Treatment

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IN THE PAST three years patients who did not respond to simple medical treatment for esophagitis were studied in detail to establish the diagnosis. Seventy-six patients whose symptoms were intractable were treated by a new surgical procedure, and 90% had subjective or objective relief of symptoms.

The most common malady of the esophagogastric junction is "peptic esophagitis," which is usually manifested by pyrosis or heartburn. Pyrosis is always a manifestation of an esophageal disorder and is recognized as an intermittent wavelike retrosternal burning or searing sensation. Evidence suggests three prerequisites for the occurrence of esophagitis. The first is reflux of gastric material noxious to the distal esophagus, second is gastric acidity usually greater than normal, and third is a distal esophagus which reacts to reflux by inflammatory or motility changes.

The diagnosis of esophagitis may be based on the subjective symptoms of heartburn, eructation, indigestion and rarely dysphagia, and on objective signs of gastroesophageal reflux by roentgenographic study, endoscopic observation, and motility studies. Increased acid production by the stomach can be tested, and endoscopic evidence of inflammation of the esophagus can be verified by biopsy of the mucous membrane of the esophagus.

Subjective symptoms are not in themselves conclusive. Heartburn is a subjective complaint and as a descriptive term lacks specificity. The definition of heartburn as applied in this study is: a painful burning sensation located retrosternally in the midline between the xiphoid process and the manubrium. Duration varies, but the pain tends to move. It may radiate to the upper abdomen, to the lateral portions of the chest wall, to the jaws, and even into the arms. Major distress is in the midline and the pain is wavelike. There is often a difference with change in position: heartburn occurs in some patients with greater frequency and severity while lying on the right side, and in others while lying on the left. It often begins during a maneuver that increases intra-abdominal pressure, such as bending to tie a shoe. It is frequently associated with excessive salivation, occasionally with eructation, and tends to occur more commonly in the recumbent position. Onset may be vague, with inconsistent recurring symptoms in early adulthood, or it may come on acutely after rapid weight gain. Prominent symptoms and findings in this group of patients are shown in Table 1.

Objective diagnostic signs of esophagitis are established by roentgenographic, motility, endoscopic, and histologic findings, supplemented by gastric acidity studies.

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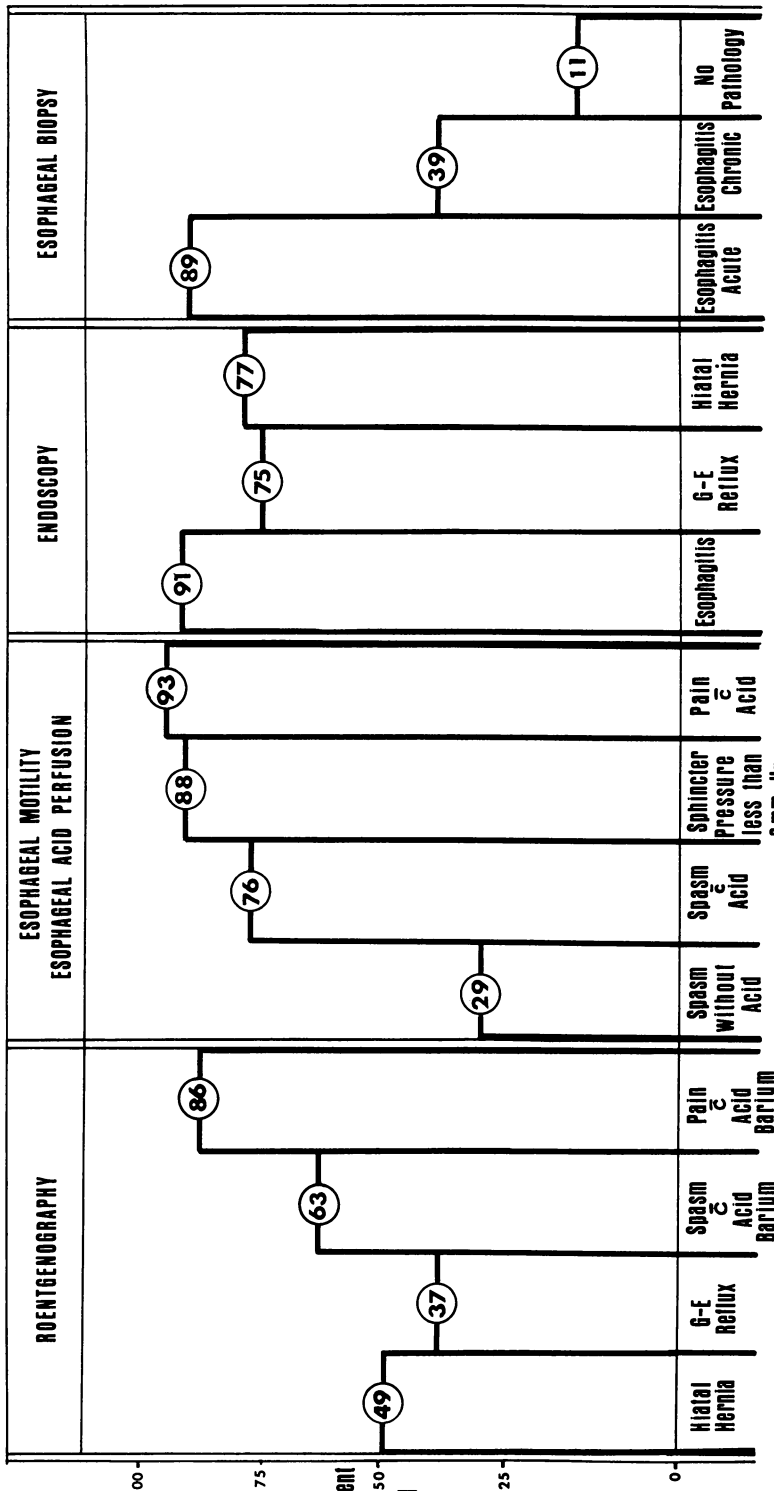

FIG. 1. Summary of preoperative findings on 76 patients with esophagitis. The statistical results of the preoperative study of the 76 patients with esophagitis are shown in Figure 1; the numbers are percentages of the 76. Discrepancies in the diagnosis of hiatal hernia by roentgenograms by endoscopic studies are shown. Prolapse of gastric mucosa through gastroesophageal junction (sphincter) was interpreted as evidence of hiatal hernia and most likely this is incorrect. It is difficult in retrospective study to be certain about the number of esophageal hiatal hernias diagnosed at the time of operation. It is difficult to categorize small or minute hernias and definite statements were not made on many operative reports. Early operative reports were influenced by the belief that hiatal hernia was the indication for operation and was present whether demonstrable or not.[4] The presence or absence of a minute hernia is unimportant since treatment of esophagitis repairs the hiatal orifice after its surgical disruption by vagotomy.

Objective findings are summarized in Figure 1. Numbers indicate percentages of the 76 patients studied. Gastric acidity tests recorded in total milliequivalents per hour are shown in Figure 2. Numerals indicate the means.

With esophagitis roentgenograms show gastroesophageal reflux and motility changes, particularly when spasm is present. Acid barium stimulates spasm and often causes pain. Endoscopy reveals the condition of the mucous membrane, and the presence or absence of esophagitis can be confirmed by biopsy. Reflux, if present, can be determined by direct endoscopic observation. Motility and gastric acidity studies are important as indicated by Barrett[1] and others.

TABLE 1. *Clinical Symptoms Elicited from 76 Patients*

Postural aggravation	88%	
Pyrosis	83%	
Pyrosis and indigestion	76%	
Dysphagia	40%	
Regurgitation	23%	
Nausea and vomiting	22%	
Back pain	16%	
Angina-like pain	11%	(With EKG evidence to exclude the diagnosis of coronary insufficiency)
Other symptoms	17%	

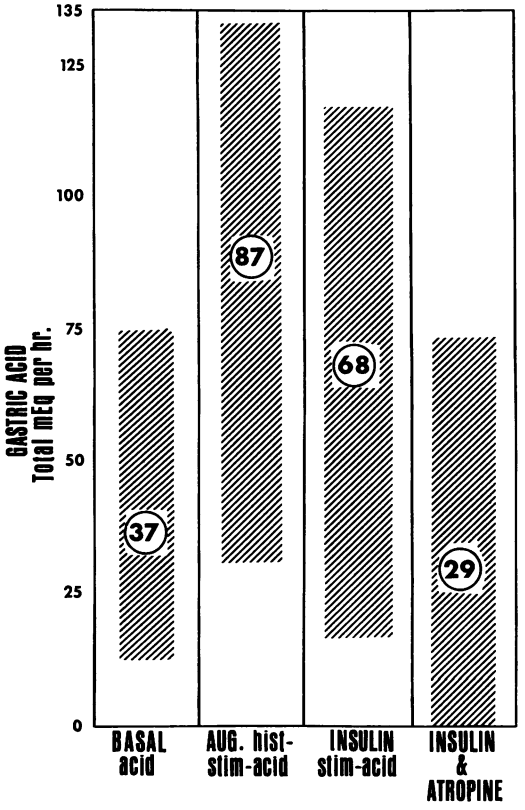

FIG. 2. Pattern of gastric secretion preoperatively in 76 patients with esophagitis. The columns indicate the range of findings and the numbers are mean gastric acidity levels reported in total mEq per hour in the 76 patients with esophagitis. They should be compared with preoperative and postoperative findings in 30 patients with vagotomy and pyloroplasty, Figure 3, and 10 patients with vagotomy and antrectomy, Figure 4.

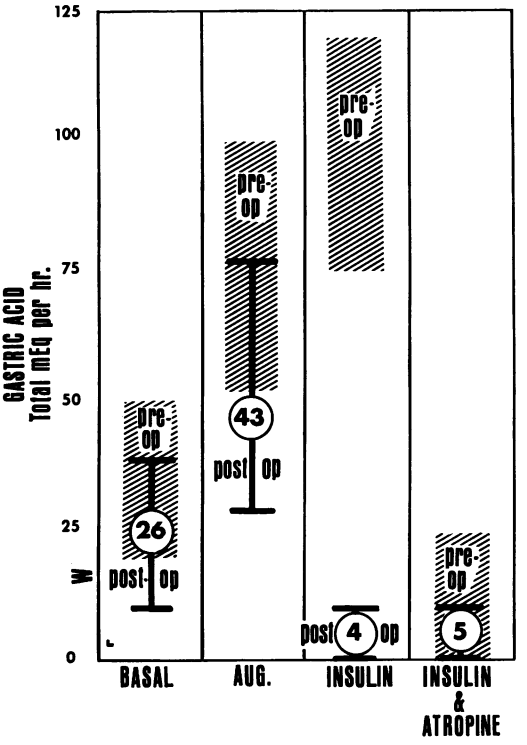

FIG. 3. Comparison of preoperative and postoperative gastric analysis on 30 patients with vagotomy and pyloroplasty. All patients with esophagitis had preoperative gastric acidity tests. Thirty patients after vagotomy and pyloroplasty were restudied. The table shows comparisons of preoperative and postoperative gastric acid tests on the 30 patients. The numerals in the postoperative bars are the means of gastric acidity levels reported in total mEq per hour of the 30 patients. This should be compared with Figure 4.

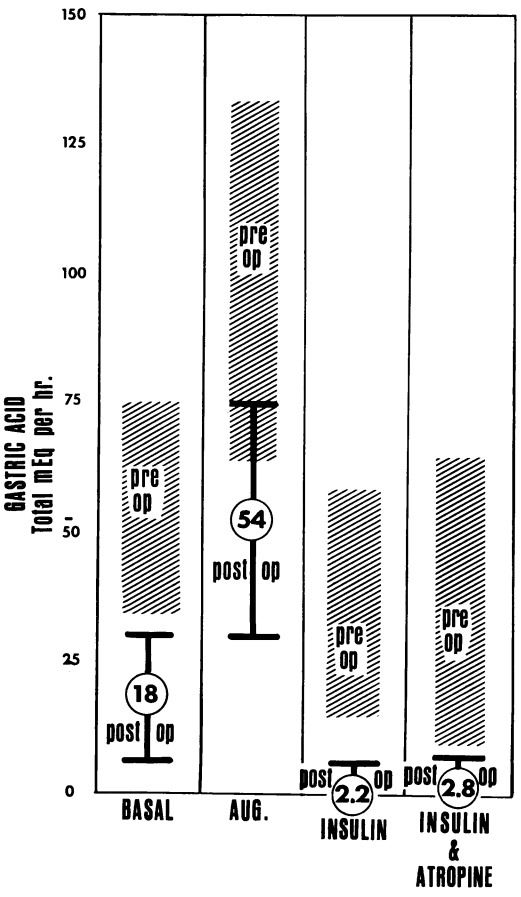

FIG. 4. Comparison of preoperative and postoperative gastric analysis on 10 patients with vagotomy and antrectomy. The table shows comparisons of preoperative and postoperative gastric acidity tests on 10 patients with esophagitis treated by vagotomy and antrectomy. The numerals in the bars are means of gastric acidity levels reported in total mEq per hour. This should be compared with Figure 3. Preoperative tests showed higher acidity levels and postoperative tests generally lower levels.

[All patients were subjected to esophageal motor studies with the patient in the supine position. Three water-filled polyvinyl catheters, with open tips placed 5 cm. apart, were introduced into the stomach. The catheters were connected to external transducers that were leveled at the posterior axillary line, and a simultaneous recording was made (Fig. 5) with a Sanborn Four Channel. Direct Writing Recorder. Sanborn Differential Pressure Transducers, Model 2678, were employed. The catheters were withdrawn into the esophagus in one cm. steps to measure the resting tone of the lower esophageal sphincter. After recording the resting pressure of the lower esophageal sphincter and the motor response of the lower esophagus to swallowing, the distal tip was withdrawn to 5 cm. above the lower esophageal sphincter. Acid perfusion studies were performed as indicated schematically (in Fig. 5) with the proximal tube acting as the perfusing catheter. The esophagus was perfused through the proximal catheter for three test periods: first with isotonic saline, second with tenth normal HCl, and third with physiologic saline. Motility was recorded through the middle and distal catheter tips. Perfusion with saline for 10 minutes long was followed by perfusion with tenth normal HCl for 20 minutes, or less if symptoms were duplicated after a shorter interval of perfusion. The rate of perfusion was 90 to 120 drops per minute; at this rate secondary peristalsis was only rarely initiated. Patients were unaware of initiation of perfusion and change from one solution to the other. Spontaneous motor activity and response to dry swallows (bolus consisting of the patient's saliva rather than a sip of water) were monitored continuously throughout all tests. The intent was to determine whether acid-induced heartburn was associated with motor abnormalities of the lower esophagus. Erratic motor activity, sphincter resting pressure lower than 3 mm. Hg and pain were all considered evidence of esophagitis.[3, 8, 14]

Gastric analyses was performed according to The Method of Kay.[10] A nasogastric tube was placed in the antrum of the stomach under fluoroscopic control. The stomach contents were emptied and a basal 60-minute collection was obtained. Thirty minutes after the start of the basal collection chlorpheniramine maleate (20 mg.) was injected intramuscularly. Patients were instructed to expectorate all saliva secreted during the collection period. After the one hour basal collection, histamine acid phosphate was injected subcutaneously in a dosage of 0.045 mg./Kg. of body weight, and gastric secretions were collected during four consecutive 15-minute periods. This one hour collection was defined as the maximal or stimulated acid output. A sample of each specimen of gastric juice was titrated to pH 7 electrometrically with 0.1N sodium hydroxide. The acid output was calculated by multiplying the acid concentration in milliequivalents per liter by the volume in milliliters collected in one hour and is expressed as total milliequivalents per hour of gastric acid output (Figs. 2, 3 and 4).

The patients were allowed to rest for a short period and then studied for responses to insulin stimulation according to the method of Hollander.[7] Controls and tube placements were the same as those described. Blood sugar samples were obtained 15 minutes after intravenous injection of

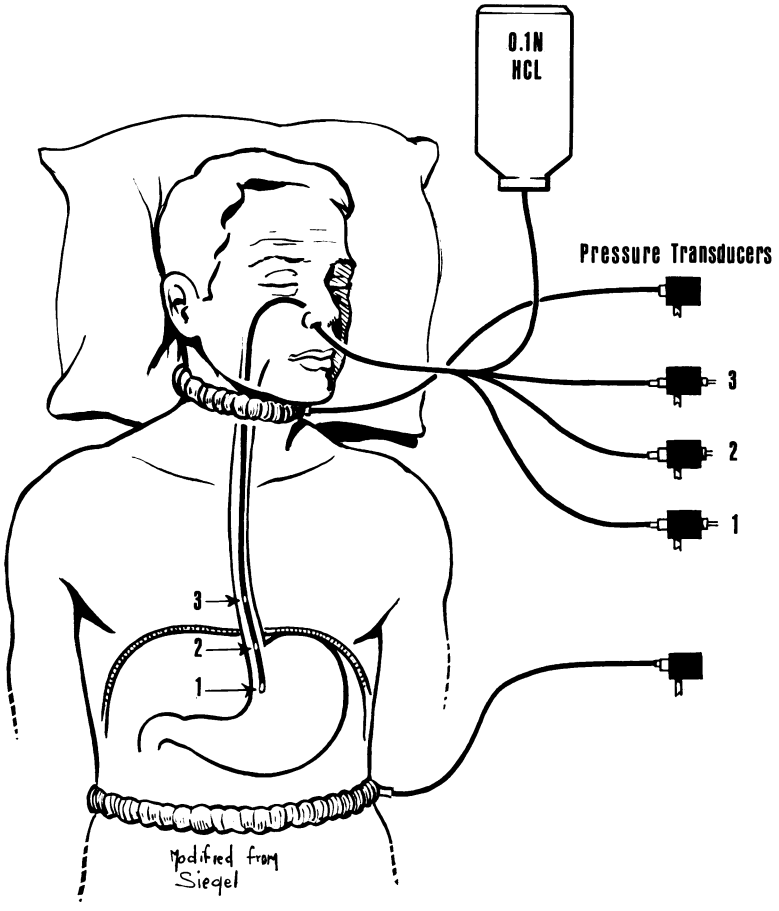

FIG. 5. The drawing is modified from Siegel [3] and shows the method of obtaining motility and perfusion studies of the esophagus.

insulin. Insulin dosage used varied from 0.1 unit /Kg. to 0.8 units/Kg, depending upon the amount of insulin required to reduce the fasting level of glucose by 50%. Precautionary steps were taken to combat insulin shock or severe hypoglycemia, if the situation warranted. Following the insulin stimulus test, the patients were given atropine sulfate intravenously in a quantity sufficient to increase the pulse rate by 30% over that recorded in the basal state. The amount of atropine necessary ranged from 1 to 500 mg., with most patients requiring from 10 to 20 mg. This drug was administered intravenously into the tubing of an intravenous solution of normal saline. When adequate atropinization had occurred, the previously established dose of insulin was administered and gastric secretions obtained for four 15-minute periods. The latter test is termed insulin stimulation with atropine block. The duration of good atropinization as judged by tachycardia averaged 1–1½ hours, in some patients as long as 3 hours. Figure 2 shows in summary the preoperative gastric secretory responses.]

The diagnosis of esophagitis was made when subjective and objective findings could be demonstrated. Objective findings included at least three of the four categories of studies, roentgenologic, manometric, endoscopic, or histologic. In patients with esophagitis and symptoms of gastroesophageal reflux, frequently referred to as symptoms of hiatal hernia, there were many patients in whom hiatal hernias were not present. The intrinsic strength or weakness of the lower esophageal sphincter determines the likelihood of gastroesophageal reflux, not whether the sphincter is located above or below the diaphragm. The only method available to quantitate the strength of the lower esophageal sphincter is intraluminal manometry. In general the severity of esophagitis was proportional to the de-

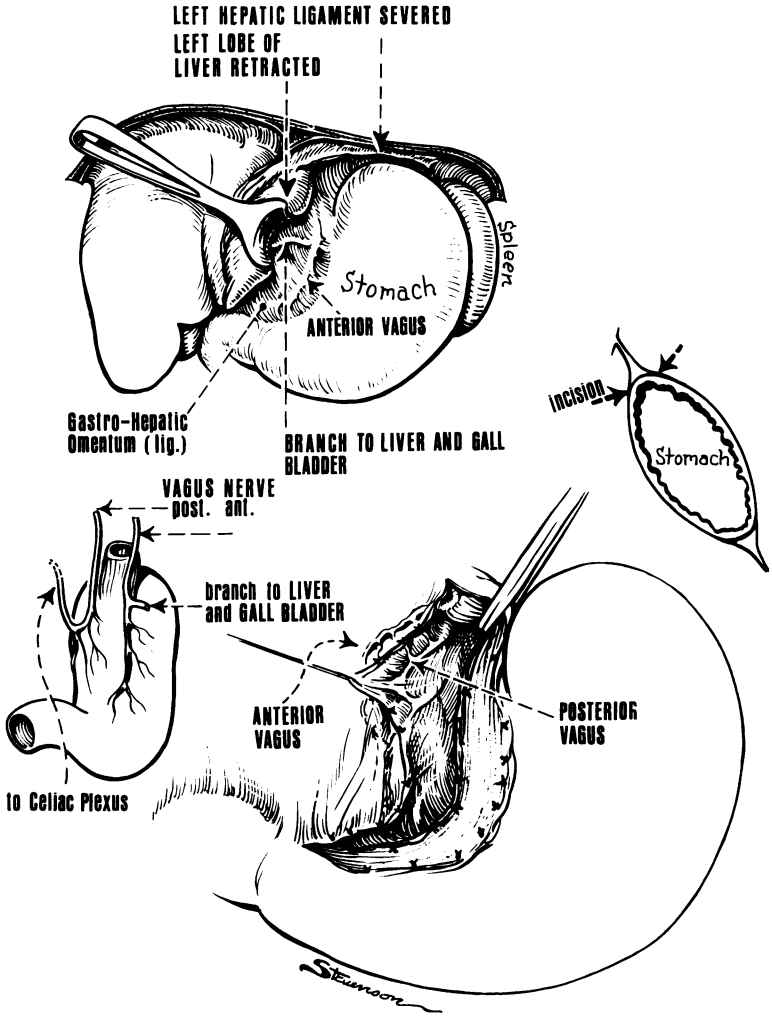

FIG. 6. The figure illustrates the technic of selective gastric vagotomy described by Griffith.[5] It consists of removal of gastrohepatic ligament by dividing the anterior layer and then the posterior layer separately. When completed the lesser curvature of the stomach and lower 2 inches of the esophagus are bare; all nerves and blood vessels have been removed. Branches of the vagus nerve to the stomach have been divided and ligated. The branch of the anterior vagus to liver, gallbladder and biliary duct system is intact and protected from injury. The branch of the posterior vagus nerve to celiac plexus is intact and protected from injury. Ligation of the divided ends tends to prevent regeneration of the vagus nerve fibers.

gree of motility disturbance and the degree of increased gastric acidity.

Once the diagnosis of esophagitis was made, a medical program was instituted which attempted to control symptoms through a bland diet, frequent use of antacids, weight reduction, mild sedation, elevation of the head of the bed at least 8 inches by means of blocks or a bolster, avoidance of increased intra-abdominal pressure and abstention from alcohol, tobacco and salicylates. The 76 patients studied were those who did not obtain symptomatic relief on the medical regimen.

Surgical operation was considered mandatory in patients with persistent symptoms and complications such as stricture or uncontrolled bleeding. It is believed that the most important etiologic factors were the corrosive quality of the hydrochloric acid and the amount and frequency of gastroesophageal reflux. Surgical decisions involved are how best to (1) reduce gastric acidity and (2) control or prevent gastroesophageal reflux. No procedure can correct the tendency of the lower esophagus to become inflamed or can change motor disturbances. If there is an esophageal hiatal diaphragmatic hernia, it is incidental to esophagitis and is repaired in a routine manner.

Reduction of gastric acidity can be ac-

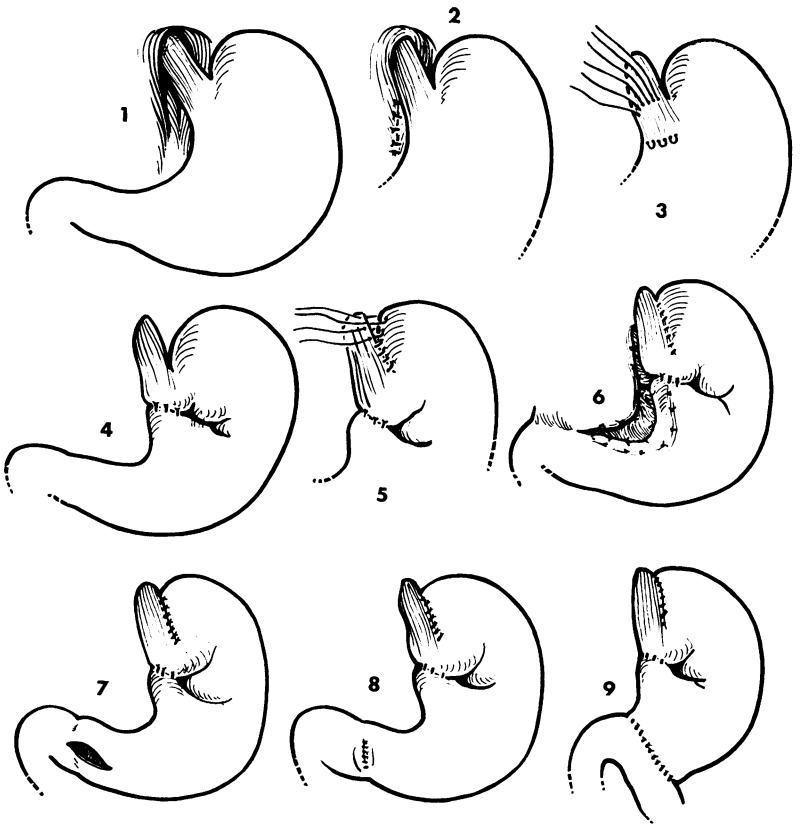

FIG. 7. Illustration 1 shows the esophageal hiatus surgically open after vagotomy. Illustration 2 shows the crura of esophageal diaphragmatic hiatus approximated by sutures posterior to esophagus. Diaphragmatic hiatal crura sutured together posterior to the esophagus repair the surgically disrupted hiatus or hiatal hernia if present. Illustrations 3 and 4 show plicating sutures in the lesser curvature of the stomach at the gastroesophageal junction. These sutures produce a fold of stomach with mucosa that acts as a flap-like valve over the gastroesophageal orifice. Illustrations 5 and 6 show suture of the fundus of the stomach to the lower 2 to 2½ inches of the subdiaphragmatic esophagus. Pressure in the stomach tends to occlude the esophagus and prevent reflux. Illustrations 7 and 8 show the typical pyloroplasty. Illustration 9 shows antral resection of the stomach, where only about 25% of lower end of stomach is resected. A retrocolic no-loop gastrojejunal anastomosis is preferred.

complished in two ways[12]: (a) elimination of cephalic (vagal) stimulation of the parietal cell mass of the stomach by either truncal vagotomy or by selective gastric vagotomy; (b) reduction in the hormonal phase of hydrochloric acid secretion by resection of the antral (hormone-producing) portion of the stomach. If preoperative tests show normal to slightly elevalted hydrochloric acid, vagotomy with pyloroplasty (Figs. 6 and 7) is sufficient. If acid secretion by maximum histamine stimulation is high, however, vagotomy with antral resection is the procedure of choice, since it reduces both phases of gastric secretion (Figs. 6 and 7). Vagal innervation of the liver, biliary tract, pancreas and bowel can be kept intact by selective gastric vagotomy which completely denervates the stomach without disturbing the innervation of other organs.[9, 13] The technic of selective gastric vagotomy is that described by Griffith[5] and is diagrammatically shown in Figure 6. It is impossible to perform truncal or selective gastric vagotomy without changing the normal anatomy of the esophageal diaphragmatic hiatus. Whenever the esophageal diaphragmatic hiatus is opened at operation, it should be surgically repaired (Fig. 7 illustration 2).

Correction of the gastroesophageal reflux has become confused with and often considered a corollary of repair of esophageal hiatal diaphragmatic hernia.[2, 4, 6, 15] Since many patients have esophagitis without hiatal hernia and others have hiatal hernias without esophagitis, it is evident that the two are not related etiologically.

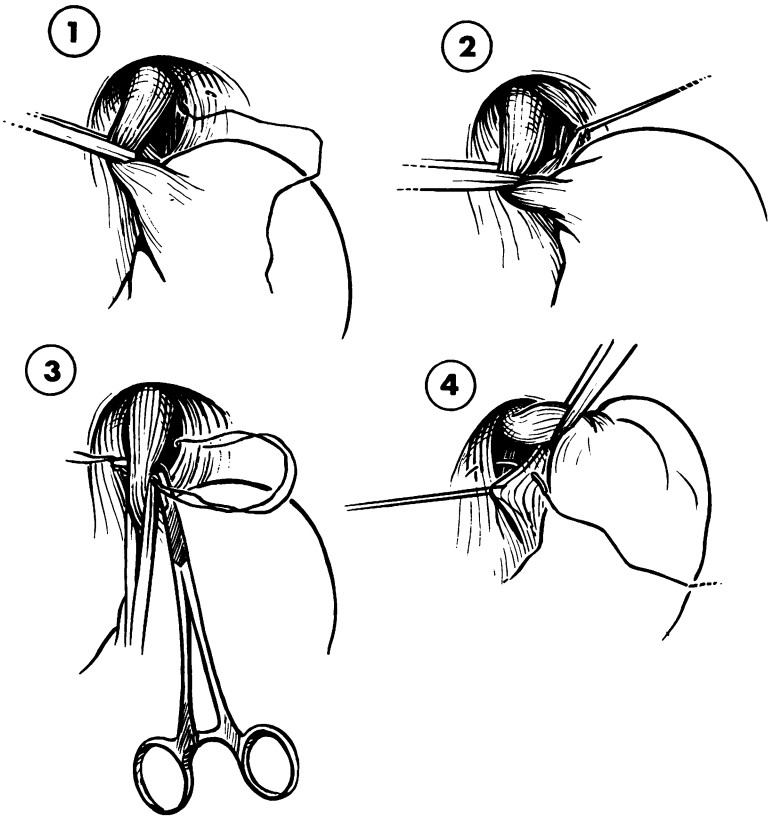

FIG. 8. Illustration 1 shows the placing of a suture in the left diaphragmatic hiatal crus with esophagus retracted to right. Illustration 2 shows crus with aorta immediately posterior to it. Illustration 3 shows the method of passing a crural suture posterior to the esophagus to the right side of the esophagus. Illustration 4 shows traction on the suture to bring the crura together.

Correction of gastroesophageal reflux has proved efficient and is a combination of procedures used by others.[6, 11, 16] This method consists of: (a) creating a fold of gastric wall at the esophageal orifice to act as a valve obstructing gastric reflux[6, 16]; (b) suturing the fundus of the stomach to the lower 2 to 2½ inches of subdiaphragmatic esophagus, which increases intragastric pressure on the esophagus and restores the angle of His.[11]; (c) snug repair of the esophageal diaphragmatic hiatus about the esophagus. The methods are shown in Figures 6, 7 and 8.

Esophagitis is commonly confused with peptic ulcer, gallbladder disease, hiatal hernia, and coronary artery disease. Failure of objective diagnosis usually leads to improper or inadequate treatment.

Most patients with esophagitis can be controlled by a medical regimen. Surgical treatment is needed when medical management fails.

When surgical treatment is necessary, the merit of a physiologic approach becomes obvious.

In the present group of 76 patients, 55 underwent vagotomy and pyloroplasty and 21 vagotomy and antrectomy. There have been no deaths. Postoperative gastric acidity tests of 30 patients with vagotomy and pyloroplasty and 10 patients with vagotomy and gastric antral resection are shown in Figures 3 and 4. Postoperative evaluations were uniformly gratifying as shown in Figures 3 and 4. Of 19 patients with positive acid perfusion tests before operation 19 were tested 6 months to 3 years after operation and all were normal. Other postoperative evaluations were clinical. Six patients were lost to follow-up and two pa-

tients are only partially relieved of symptoms.

Preference for selective gastric vagotomy developed from observations that it is anatomically and physiologically more precise and is associated with fewer postvagotomy sequelae.

When complications of esophagitis are encountered, such as stricture, ulceration, and rarely bleeding, the complications are treated with surgical conservatism. These complications usually become amenable to less extensive operations after the inflammatory provoking condition is ameliorated and do not require, in most instances, direct surgical approach on the esophagus.

Summary and Conclusions

1. Esophagitis is a common entity manifested by pyrosis (heartburn), and radiating substernal pain. It must be distinguished from angina pectoris, coronary artery disease, other dyspeptic symptoms, and esophageal hiatal diaphragmatic hernia.

2. Esophagitis may be attributed to three important factors:

(a) Gastroesophageal reflux
(b) Gastric acidity, usually greater than normal
(c) Failure of the lower esophageal mucosa to resist the effects of acidity and reflux, together with abnormal motility of the lower esophagus.

3. Esophagitis occurs in the absence of esophageal hiatal hernia but may occur coincidentally with hiatal hernia.

4. Objective evidence of esophagitis can be established by esophageal motility studies, esophageal acid perfusion, roentgenographic studies, endoscopic technics, and especially by histologic evidence obtained from esophageal biopsy.

5. Esophagitis usually can be treated by a medical regimen. However, if medical regimen is unsuccessful and objective evidence of esophagitis can be established, alleviation of the esophagitis can be achieved by surgical technics, primarily directed at changing the quality of the gastric juices, by repair of the esophageal diaphragmatic hiatus, repair of the gastroesophageal junction, selective gastric vagotomy, and either pyloroplasty or antral resection of the stomach.

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