An Effective Operation for Hiatal Hernia:
An Eight Year Appraisal

LUCIUS D. HILL, M.D.

From the Department of General and Thoracic Surgery, The Mason Clinic, Seattle, Washington

SINCE the first deliberate repair of hiatal hernia, by Wm. J. Mayo in 1911,[2] countless procedures have been performed to correct herniation of the stomach into the posterior mediastinum. Earlier, these operations consisted primarily of closure of the enlarged hiatus without proper fixation of the stomach. Recurrent herniation was high. With the advent of the Allison type of repair in 1951,[1] attention was turned to fixation of the stomach in its subdiaphragmatic position. Unfortunately, with the Allison type of repair and its many variations, the anterior or weakest portion of the phrenoesophageal membrane is used in an effort to fix the stomach below the diaphragm. Long-term follow up on the Allison type of repair indicates that the recurrence rate is too high. The recently reported series from the Mayo Clinic,[7] where the first repair was done, indicates a documented recurrence rate of 21% which is unacceptable for an elective operative procedure.

Recently, various procedures aimed primarily at anchoring the stomach below the diaphragm have been attempted. Chief among these is the Nissen[8] type of operation which anchors the stomach to the anterior abdominal wall. Although this operation has not met with uniform success, it has become apparent that if the stomach can be maintained in its subdiaphragmatic position, not only is recurrent herniation prevented, but reflux of gastric juice is often curtailed.

The procedure to be presented here has been performed for the past 8 years and is aimed not only at reducing the hernia and closure of the hiatus, but primarily at permanently fixing the gastroesophageal junction in its subdiaphragmatic location to prevent reflux and recurrent herniation.

Materials and Methods

This report consists of 149 consecutive operations for hiatal hernia, performed over an 8-year period. These patients were studied preoperatively with combined pH and pressure studies, conventional gastrointestinal x-rays, and by esophagoscopy in nearly all cases. Cineradiography was employed when diagnostic was difficult and when motility disorders were suspected.

All 149 patients had preoperative gastrointestinal x-rays. One hundred twenty-three patients had preoperative combined pH and pressure studies. One hundred patients have had postoperative pH and pressure studies. In addition, overnight gastric acid was obtained preoperatively in nearly all patients. One hundred forty-two patients had sliding hiatal hernia, while seven patients had paraesophageal hernias.

Indications for Surgery

We advocated operation for repair of hiatal hernia only in patients with significant symptoms or complications. Of 466 patients diagnosed with hiatal hernia in a

Presented at the Annual Meeting of the American Surgical Association, May 11–13, 1967, Colorado Springs, Colorado.

FIG. 1. Cadaver dissection with diaphragm and all anterolateral phrenoesophageal membrane removed. Gastroesophageal junction cannot be moved for any distance because of dense posterior attachments to the preaortic fascia. Finger is under median arcuate ligament and preaortic fascia.

single year, 70 or 15% were submitted to operation. The remainder are being followed medically.

The most common indication for operation was intractibility or failure to respond to medical management. One hundred and twenty patients of the 149 operated upon were considered intractible to medical management.

The second most common indication was esophagitis. Sixty-eight patients had esophagitis of varying degrees. An additional six had severe stenosis of the esophagus causing dysphagia. Four additional patients had contraction rings of the terminal esophagus, the so-called Schatzki's ring, which may or may not be the result of esophagitis. Four patients had esophageal ulcers, discrete enough to recognize on gastrointestinal x-ray and to be visualized by esophagoscopy.

Another group of complications that is being recognized as an indication for operation are those related to respiratory complications. We encountered these in 34% of all patients diagnosed as having hiatal hernia. Respiratory complications varied in this group all the way from productive cough to chronic obstructive pulmonary disease to lung abscess and empyema in one patient. A total of 59 patients in this group had significant respiratory difficulties related to overflow, particularly nocturnal overflow of gastric juice into the respiratory tract.

Significant anemia was present in 14 patients and hemorrhage in 10 patients. In seven patients, hiatal hernia appeared to trigger angina pectoris. In eight there was gastric heterotopia lining the lower third of the esophagus. Parietal cells were found in large numbers in four of these patients; in one severe enough to require transthoracic vagotomy above the level of the gastric heterotopia for relief of symptoms. In 22 patients the hernia was so large that it produced cardiorespiratory embarrassment or pressure symptoms in the chest, particularly severe postprandial distress with a distended stomach.

In addition to these specific indications for operation concomitant conditions were present in a number of patients that required additional procedures. Twenty-eight patients required cholecystectomy for cholelithiasis. Twenty-eight had vagotomy and pyloroplasty for either significant pyloric stenosis or active duodenal ulcer. Three had diverticulitis that required resection.

Rationale

Most operations devised for correction of hiatal hernia have placed too much emphasis on closure of the enlarged hiatus, and have focused too little attention on anchoring the stomach in its subdiaphragmatic position. We have previously presented evidence [3-8] in support of a terminal esophageal sphincter that fulfills all the criteria for a physiologic sphincter. It has also been shown that replacement of the sphincter mechanism in its normal location allows it to regain competence in most in-

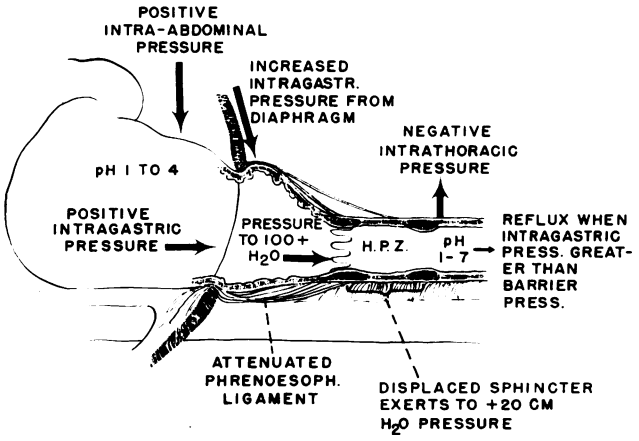

FIG. 2. The key to sliding hiatus hernia is attenuation of the posterior phrenoesophageal ligament, allowing the terminal esophageal sphincter to slide into the posterior mediastinum.

stances. Our observations lead us to believe that replacement of the gastroesophageal junction subdiaphragmatically and maintenance of it there permanently serves to prevent recurrent herniation as well as reflux.

Those operations which have included fixation of the stomach below the diaphragm have generally employed tissues of insufficient strength to maintain fixation over a long period. A careful examination of the phrenoesophageal fascial complex demonstrates clearly that the anterior portion of the membrane is lax and weak. It is this portion that Barrett so aptly described as being so thin that it "requires dissection with an eye of faith." The posterior portion of the phrenoesophageal ligament, on the other hand, is dense and is responsible for holding the esophagus in its normal location. Figure 1 shows a cadaver dissection which demonstrates clearly that one may remove all the anterior connections of the esophagus to the diaphragm and indeed, the entire diaphragm itself, leaving only the posterior attachments of the esophagus to the preaortic fascia. After removal of all of these anterior and lateral attachments of the esophagus, the gastroesophageal junction cannot be moved appreciably out of its normal location because of the dense posterior attachment. This dense, ligamentous structure consists of fibroelastic bands, passing directly from the esophageal wall to the preaortic fascia. It is rarely, if ever, seen by surgeons of considerable experience, as this is an area rarely dissected out when normal, and, in the presence of hiatal hernia, it is attentuated. Thus, little attention has been paid to this dense attachment in the past.

From a functional standpoint, it is only reasonable that the anterior part of the phrenoesophageal membrane is rather loose. This allows for movement of the diaphragm on the gastroesophageal junction and allows for opening and closure of the esophageal sphincter mechanism. The attachments of the phrenoesophageal membrane, both above and below the sphincter, also allow for closure of the sphincter within the phrenoesophageal complex. If the phrenoesophageal membrane were tightly adherent to the sphincter all the way around, and, in turn, tightly attached to the diaphragm, the terminal sphincter mechanism could not close, nor could the diaphragm move freely with respiration up and down the terminal esophagus.

The preaortic fascia and the median arcuate ligament, to which the phrenoesophageal membrane is densely attached, are structures of considerable strength and serve as a source of the origin of the crura of the diaphragm (Fig. 1).

The key, therefore, to the formation of a

sliding hiatal hernia is failure or attentuation of the posterior attachment of the esophagus to the preaortic fascia. Figure 2 shows diagrammatically the sequence of events that occurs when the posterior attachment of the esophagus fails. From this rationale it is clear that the important feature of any operative procedure is not necessarily the closure of the enlarged hiatus but, more importantly, the permanent fixation and maintenance of the gastroesophageal junction in its normal subdiaphragmatic position.

The operative technic herein described not only anchors the gastroesophageal junction to the preaortic fascia, but, in addition, places tension on the collar sling musculature of the cardia, thereby accentuating the gastroesophageal angle and assisting in closure of the sphincter mechanism.

Postoperative studies on the 149 patients operated upon thus far indicate that replacement of the sphincter mechanism in its normal location allows it to resume normal competence, unless it has been destroyed by esophagitis or unless there is an underlying congenital motility disorder or malfunctioning sphincter. The detection of a congenitally incompetent or destroyed sphincter is difficult. A history of gastroesophageal reflux since infancy or gross reflux with a high gastric acid as determined by pH and pressure studies should alert the physician to a congenitally incompetent or destroyed sphincter mechanism. This condition may not always be corrected by the procedure to be described, and, in the presence of high gastric acid, requires an ancillary procedure such as vagotomy and pyloroplasty.

The other condition that must be considered in the rationale for surgery is gastric heterotopia of the terminal esophagus. This is more common in our experience than previously suspected and has been detected by biopsy of the terminal esophagus at the time of esophagoscopy. Eight patients in this series had gastric heterotopia of the terminal esophagus. Four showed acid-secreting parietal cells in large numbers. When biopsy shows parietal cells, the surgeon should consider a vagotomy and pyloroplasty if the gastric acid is high. It was necessary in one patient to reoperate and perform a transthoracic vagotomy above the level of the gastric heterotopia to obtain complete relief of symptoms.

Technic

Esophagoscopy is performed in nearly all patients. In those with severe symptoms, it is performed before operation. In those with only mild symptoms and no evidence of lesions in the terminal esophagus on gastrointestinal series, it is performed at the time of surgery. One hundred fifteen patients in this series had esophagoscopy to assess the extent and severity of esophagitis, to rule out carcinoma, to determine whether the patient has an esophageal web or diaphragm or Schatzki's ring at the terminal esophagus. Also, the observer gains an additional estimate of the size of the hernia.

The operative procedure is done generally through a left paramedian, upper abdominal incision. If there is known cholelithiasis, a right paramedian is made, but is angled at the cephalad end up to the left of the xiphoid. Upon entering the abdomen, the size of the hernia is determined by invaginating the hernia sac into the posterior mediastinum and noting the extent to which the stomach ascends into the posterior mediastinum. Abdominal exploration is performed to assess the presence or absence of concomitant conditions. Careful attention is paid to the pylorus. Pyloric stenosis, old healed duodenal ulcer or active ulceration are important findings. Pyloric stenosis will compromise gastric drainage and premote reflux. Following exploration, the left lobe of the liver is reflected to the patient's right by dividing the tri-

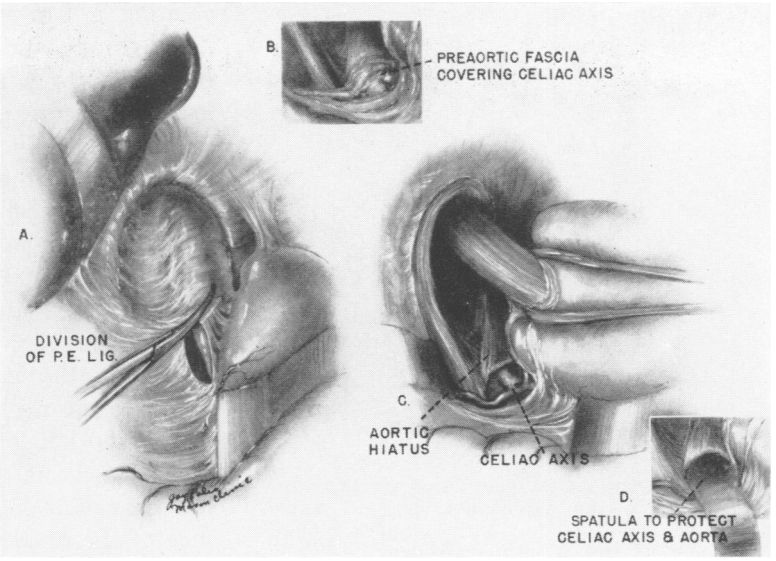

FIG. 3. Esophageal hiatus, preaortic fascia and median arcuate ligament dissected out.

angular ligament of the liver. The gastrohepatic omentum is divided. A hepatic branch of the vagus nerve and occasionally a sizable artery extending from the left gastric artery into the hilum of the liver is often encountered in the gastrohepatic omentum. Division of these two structures has caused no difficulty in this series. The terminal esophagus is then dissected out by dividing the phenoesophageal membrane and reducing the hernia into the abdomen (Fig. 3). A tape is placed about the terminal esophagus and is withdrawn by downward traction into the abdomen. Careful palpation of the terminal esophagus determines the extent and severity of esophagitis and amount of adherence of the terminal esophagus to the surrounding structures. The esophageal hiatus is carefully dissected out, as is the preaortic fascia and the median arcuate ligament. The esophageal hiatus is then closed by taking sutures widely to include the fascia overlying the crura in a figure-of-eight fashion. Generally three to four figure-of-eight sutures are sufficient to close the hiatus snugly about the esophagus. A No. 18 Levin tube is employed routinely for intubation. With the index finger placed alongside the esophagus, the surgeon can palpate the No. 18 Levin tube. The hiatus is closed sufficiently to allow the insertion of the index finger into the hiatus. This, coupled with the No. 18 Levin tube, insures an adequate lumen. Following closure of the hiatus, the stomach is rotated anteriorly. The cut edges of the gastrohepatic omentum and the phrenoesophageal ligament are assessed. Deep sutures are then placed, picking up the anterior cut edge of the gastrohepatic omen-

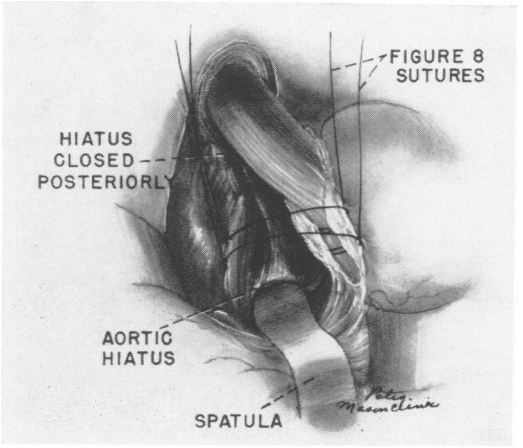

FIG. 4. Figure of 8 sutures including anterior and posterior cut edges of phrenoesophageal ligament placed deep into preaortic fascia with spatula protecting the aorta.

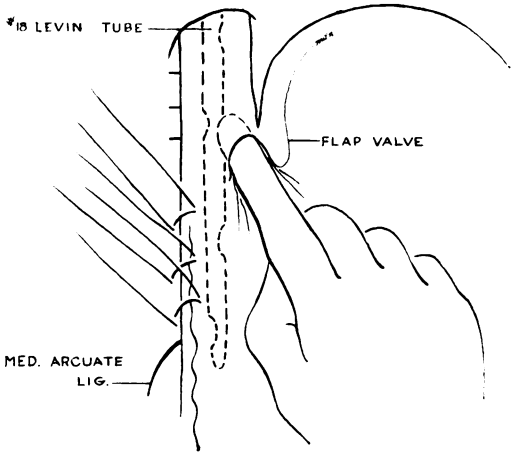

FIG. 5. Diagram showing surgeon's finger invaginated into the terminal esophageal lumen alongside Levin tube to insure adequate esophageal lumen as fixation sutures are tied.

tum and the phrenoesophageal ligament, as well as the seromuscular layer of the stomach along the lesser curvature (Fig. 4). The suture is then carried posteriorly to pick up the posterior cut edge of the gastrohepatic omentum and phrenoesophageal ligament. A small spatula is placed beneath the median arcuate ligament and moved cephalad between the aorta and the preaortic fascia. With the spatula in place, protecting the aorta, the suture from the lesser curvature of the stomach is then carried deep into the preaortic fascia. It is then carried back through the anterior and posterior part of the lesser curvature of the phrenoesophageal ligament and back through the preaortic fascia. Two to three of these figure-of-eight sutures are placed. These are then tied, approximating the gastroesophageal junction firmly to the preaortic fascia. As these sutures are tied, the surgeon's finger is placed in the gastroesophageal junction (Fig. 5), so that the terminal esophageal lumen can be felt along with the No. 18 Levin tube. As the sutures are tied, tension is placed on the collar sling musculature of the cardia, tightening the sphincter mechanism and accentuating the gastroesophageal angle, creating a flap valve which can be palpated through the stomach wall. It should be emphasized that the sutures in the lesser curvature part of the stomach must be taken deeply to include not only the cut edges of the phrenoesophageal ligament and vagal fibers but the seromuscular layer of the stomach as well. The surgeon must be certain after tying these sutures that an adequate esophageal lumen remains. Two additional sutures are then placed in the gastroesophageal angle in order to insure permanence of the flap valve. The left lobe of the liver is then replaced in its normal position, but suturing the triangular ligament to the under surface of the diaphragm. The completed operative procedure is shown in Figure 6. The pylorus is carefully examined and a finger placed through the lumen of the pylorus to determine whether or not there is any scarring, narrowing or ulceration. In the presence of active duodenal ulceration or marked pyloric stenosis, vagotomy and pyloroplasty should be considered. This decision is based not only on the presence of pyloric stenosis or active ulceration but on the degree of over night acid as well as the extent and severity of esophagitis and symptoms.

In the presence of severe esophagitis, which may have damaged the sphincter to the point of incompetency with ulceration of the esophageal mucosa, or in the presence of a congenitally malfunctioning terminal sphincter vagotomy and pyloroplasty is also considered.

Paraesophageal Hernia

The paraesophageal hernia represents a different set of circumstances than the sliding type of herniation. In these, the gastroesophageal junction has not become displaced into the posterior mediastinum, rather it is located in its normal position and held there by a competent posterior phrenoesophageal ligament. For this reason, it is a technical mistake to tear down

the posterior phrenoesophageal ligament, which has demonstrated its competence. The problem here is reduction of the hernia sac from the posterior mediastinum and closure of the large paraesophageal opening in the diaphragm by multiple interrupted figure-of-eight sutures. In the event that the posterior phrenoesophageal ligament is lax, several figure-of-eight stitches are placed from the lesser curvature to the preaortic fascia to insure that a sliding herniation does not occur. Seven patients in this series had paraesophageal hernias. Using this technic for the past 8 years, there has been no recurrence of paraesophageal herniation and the symptoms in these patients are entirely corrected by the procedure.

Repair of Recurrent Hernia

Seventeen patients in this series were operated upon for recurrent herniation. These had been repaired by the Allison technic. Historically these patients either were never relieved of preoperative symptoms, or were relieved for only a short time. At operation, the technic described previously was employed. A careful dissection of the esophageal hiatus indicated that the stomach had again ascended into the posterior mediastinum and that the loose fixation to the anterior rim of the esophageal hiatus was either disrupted or so attenuated that it failed to hold the stomach in its subdiaphragmatic position. After dissection of the scarred hiatus, the remainder of the procedure presented no particular problems.

Follow Up

Follow up has been obtained on all 149 patients. The follow up has included personal interview and/or questionnaire in all cases. One hundred patients have had postoperative pH and pressure studies. All who had postoperative symptoms had conventional gastrointestinal x-rays. Cineradiography has been employed only in the difficult diagnostic problems and when motility disorders were suspected. There has been no mortality, and to date, there has been no demonstrated anatomic recurrence by any method employed.

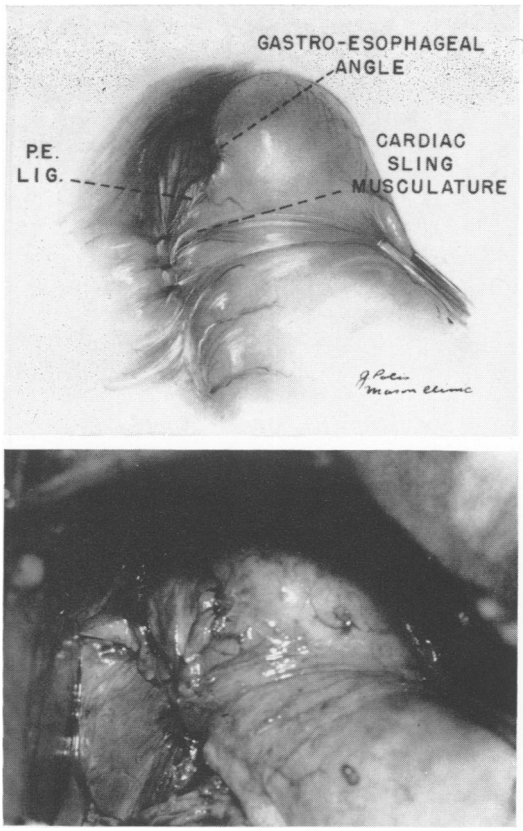

FIG. 6. Completed operative procedure. Phrenoesophageal ligament sutured securely to preaortic fascia. Gastroesophageal junction fixed 2 to 3 centimeters below diaphragm.

In addition to the detection of recurrent herniation, follow up has been concerned primarily with two factors: 1) correction of symptoms; and 2) correction of reflux.

In regard to symptoms, the patients rated the operation as *good* to *excellent* in all but five instances. The five who rated the operation as *fair* are presented in Table 1. It can be noted that in all of these, postoperative gastrointestinal series showed no evidence of recurrent herniation. It should be noted further that in all there was marked improvement over preoperative status in regard to symptoms. The first

TABLE 1. *Patients Rating Operation Fair*

Name	Diagnosis	Operation	Post op GI Series	pH/Pressure	Symptoms
H. I.—F 215-829	HH*	Repair HH	No HH No reflux	Induced reflux with knees up	Occ. heartburn Epigastric pain
D. R.—M 330-354	Recurrent HH	Repair HH	No HH	No reflux	Occ. chest pain Dysphagia
F. B.—M 182-242	HH Duodenal ulcer	B_I Gastric resection Repair HH	No HH	Induced reflux with knees up	Recurrent ulcer symptoms
E. R.—F 325-170	HH Motility disorder	Repair HH	No HH	Motility disorder	Chest pain Occ. dysphagia
L. W.—M 253-888	HH Gastric heterotopia	Repair HH Vagotomy Pyloroplasty	No HH	Induced reflux Slow rise in pH	Occ. chest pain

* HH = hiatal hernia

patient had free reflux prior to surgery and now refluxes on postoperative pH and pressure study, only with the knees forceably drawn up. The remaining four had special problems, including recurrent herniation, duodenal ulcer requiring gastrectomy, a motility disorder similar to diffuse spasm of the esophagus, and gastric heterotopia lining the lower third of the esophagus. Although there is no recurrent herniation in any of these, the patient continues to have symptoms related to the esophagus. Gastric heterotopia, in particular, presents a difficult problem of management. In one additional patient, it was necessary to do a transthoracic vagotomy above the level of the gastric heterotopia before complete relief was obtained. Eight patients in all in this series had gastric heterotopia. In those with severe heterotopia with acid-secreting parietal cells and high gastric acid, a vagotomy and pyloroplasty had been added to the repair of the hernia.

In regard to symptomotology, ten of the 28 patients having vagotomy and pyloroplasty have some symptoms relative to the procedure. These patients have been studied extensively and have no recurrent herniation. The symptoms of the post-vagotomy syndrome vary from steatorrhea to postprandial distress. Because of these troublesome symptoms, we now employ vagotomy and pyloroplasty only in the presence of severe esophagitis with gross reflux and in the presence of pyloric stenosis or duodenal ulceration.

In regard to reflux, the tendency to free reflux was corrected in all but one of 100 patients tested postoperatively. Five patients who continued to show reflux postoperatively are presented in Table 2. It will be noted that the patient's own subjective rating of the operation varied from *good* to *excellent* despite the tendency to reflux. In all these patients there was marked improvement over preoperative status. In patients with induced reflux, on pH and pressure studies it was noted that it was difficult to induce reflux, which indicates that, under normal circumstances, these patients are free of regurgitation of acid into the terminal esophagus. One patient with free reflux is difficult to evaluate. Although she refluxes acid gastric juice on pH and pressure study, she has only rare heartburn and rates her operation as *excellent* compared to preoperative status. Preoperatively, this patient complained that she refluxed liquid into her mouth in a recumbent position, particularly at night,

TABLE 2. *Postoperative Reflux*

Name	Operation	Postop GI Series	pH/Pressure	Subj. Rating of Operation
F. B.—M 182–242	Repair HH* Vagotomy Billroth II Gastric resection	No HH Recurrent, marginal ulcer	Reflux with knees up	Good
J. H.—F 304–280	Repair HH	No HH Reflux in recumbent position	Reflux with knees up only	Good
P. W.—M 321–234	Repair HH	No HH No reflux	Reflux with maneuvers	Good
V. M.—F 328–326	Repair HH	No HH Induced reflux	Slow rise in pH	Excellent
O. B.—M 288–451	Repair, recurrent HH Vagotomy Pyloroplasty		Induced reflux Achlorhydria	Excellent
A. M.—F 129–610	Repair HH		Free reflux	Excellent

* HH = hiatal hernia

and had had reflux since infancy along with severe heartburn. Although she is markedly improved over her preoperative status, she continues to have occasional mild heartburn. It is this type of patient who might well benefit from vagotomy and drainage procedure in addition to repair of the hiatal hernia. In several other patients in this series, however, with similar long-standing reflux, as demonstrated by postoperative pH and pressure studies, the operation corrected both free and induced reflux. In summary, the 100 patients tested postoperatively indicate correction of reflux in 94% with correction of free reflux in 99%.

On the basis of the objective and subjective findings, it would appear that this procedure is effective in preventing recurrent herniation and in correcting reflux. It should be emphasized that in the 149 patients operated upon, all who had postoperative symptoms had a gastrointestinal series or pH and pressure study and none showed recurrent herniation. One troublesome feature is the inability of patients to belch postoperatively. This is transient, but may lead to gastric dilatation in the early postoperative period.

In a series of 436 operations over a comparable period of time done by the Allison technic, a 17% anatomic recurrence was demonstrated by gastrointestinal series, even though complete follow up was not obtained on these patients. Patients studied by pH and pressure study after the Allison technic showed free reflux in 25%. If follow up had been complete, the recurrence rate and reflux would undoubtedly be higher. Many patients who had the Allison repair complain that the symptoms they experienced preoperatively are either not relieved or were relieved for only a short period. A gastrointestinal series in the first 2 to 3 weeks postoperatively has demonstrated an anatomic recurrence in several of these patients.

The postoperative pH and pressure profile in 94% of the 100 patients tested was normal. The pH rises out of the peptic range before the sensing device reaches the diaphragm as it is drawn cephalad.

Summary

A consecutive series of 149 operations for repair of hiatal hernia are presented. There was no mortality and no anatomic recurrence over an 8-year period.

Preoperative and postoperative combined pH and pressure studies in 100 patients indicate correction of free reflux in 99 with five patients showing induced reflux postoperatively. In all five, there was marked improvement over the preoperative status with the patient rating the operation subjectively as *good* to *excellent*.

Symptoms were corrected by the procedure in all but 3.3% of patients. The five patients presenting significant postoperative symptoms had special problems ranging from gastric heterotopia to motility disorders of the esophagus and are improved over their preoperative status. None of these patients has recurrent herniation.

Over a comparable period of time, the Allison technic yielded a demonstrated anatomic recurrence in 17% of patients, without complete follow up, on 436 cases.

The prime factor in the technic consists of anchoring the gastroesophageal junction to the median arcuate ligament and preaortic fascia so that it remains in its subdiaphragmatic position permanently.

References

1. Allison, P. R.: Reflux Esophagitis, Sliding Hiatal Hernia and the Anatomy of Repair. Surg. Gynec. Obstet., **92**:419, 1951.
2. Giffin, H. Z.: The Diagnosis of Diaphragmatic Hernia. Ann. Surg., **55**:388, 1912.
3. Morgan, E. H., Hill, L. D., Siemsen, J. K., Chapman, K. W. and Botseas, D.: Studies of Intraluminal Esophageal and Gastric Pressure and pH. Bull. Mason Clin., **14**:53, 1960.
4. Hill, L. D., Chapman, K. W. and Morgan, E. H.: Objective Evaluation of Surgery for Hiatus Hernia and Esophagitis. J. Thorac. Cardiov. Surg., **41**:60, 1961.
5. Hill, L. D., Morgan, E. H. and Kellogg, H. B., Jr.: Experimentation as an Aid in Management of Esophageal Disorders. Amer. J. Surg., **102**:240, 1961.
6. Hill, L. D., Tobias, J. and Morgan, E. H.: Newer Concepts of the Pathophysiology of Hiatal Hernia and Esophagitis. Amer. J. Surg., **III**:70, 1965.
7. Hugh, A. R., Ellis, F. H., Jr., Carlson, H. C. and Anderson, H. A.: Surgical Repair of Sliding Esophageal Hiatal Hernia. Arch. Surg., **91**:228, 1965.
8. Nissen, R.: Gastroplexy as the Lone Procedure in the Surgical Repair of Hiatus Hernia. Amer. J. Surg., **92**:289, 1956.

Discussion

Dr. F. Henry Ellis, Jr. (Rochester, Minn.): Dr. Bricker, Members and Guests: Recent years have witnessed the introduction of a large number of new operative techniques for the repair of esophageal hiatal hernia. This, I think, reflects the general dissatisfaction with the results of current techniques.

Since the article of Allison in the early 1950's, few contributions have been marked by scholarly objectivity. This paper that you have just heard, however, is an exception, and I suspect in time it will take its place in the literature along with the classic contributions to the subject of hiatal hernia repair. Doctor Hill has taken advantage of some of the newer, more sophisticated, physiologic techniques to provide objective data relative to the effectiveness of his operation. In this way, the known inadequacies of symptomatic evaluation are avoided.

I would like to stress a few of the points Doctor Hill made. He has rightly emphasized the importance of restoring normal anatomy and has shown that by so doing, gastroesophageal competence is restored. Although not all would agree with this concept, I think his studies show that this is indeed the case. One must, therefore, question the wisdom of employing some of the more complicated hiatal operations, such as fundoplication, the Belsey procedure, or the routine application of vagotomy and pyloroplasty.

He has re-emphasized the importance of preventing recurrences. In our experience, postoperative symptoms are dependent almost exclusively on the presence or absence of a recurrence. Thus nearly a third of our patients with radiographic evidence of a recurrent hernia had recurrent symptoms, while nearly 100% of those whose hernias remained reduced were asymptomatic.

I would like to ask one question about a technical detail which was not stressed in the talk but appeared in one of the illustrations. It concerns suturing the gastric fundus to the wall of the esophagus. I have never been much impressed with the concept of a valve-flap mechanism for gastroesophageal competence, and I wonder if this maneuver is effective by providing a bulk of tissue in the region of the cardia which militates against