Comparison of Crural Repair and Nissen Fundoplication in the Treatment of Esophageal Hiatus Hernia with Peptic Esophagitis

E. R. Woodward, M.D., H. F. Thomas, M.D., J. C. McAlhany,* M.D.

From the Department of Surgery, University of Florida College of Medicine, Gainesville, Florida, and the Veterans Administration Hospitals, Gainesville and Lake City, Florida

Sliding esophageal hiatus hernia is frequently associated with peptic esophagitis. It is not clear whether a cause and effect relationship exists, since most people with sliding hiatal hernias do not develop esophagitis and some patients develop severe esophagitis in the absence of hiatal hernia. Nonetheless, reflux esophagitis has been treated by repairing the hiatal hernia and successful repair often cures the esophagitis. Unfortunately, present methods are associated with high recurrence rates and more reliable technics are needed.

This report compares two methods of repair for sliding esophageal hiatus hernia. Until 1964, the technic described by Allison was used in nearly all instances of this disease.[1] This procedure approximates the crural structures of the esophageal hiatus posterior to the esophagus. The transthoracic approach was used in earlier years, but more recently the transabdominal approach has had a wider acceptance. In 1964 we began using Nissen's fundoplication for treatment of recurrent or persistent esophagitis after unsuccessful crural repair.[3] This method effectively controlled esophagitis after which a gradual shift to this procedure as primary surgical treatment ensued. Again, abdominal approach has been employed in the majority of these cases. In addition to clinical evaluation, we have used radiologic study to determine the anatomic state of the repair and laboratory studies to indicate gastroesophageal reflux.

Method

From November 1, 1958, to December 31, 1969, 259 hiatal herniorrhaphies were performed at the Shands Teaching Hospital and its affiliated Veterans Administration Hospitals in Gainesville and Lake City, Florida. Adequate follow-up information was obtained on 231 patients (89%) which forms the basis for this report. One hundred and twenty-seven patients had crural repairs and 103 fundoplication. The age range varied from 21 to 81 years with a mean of 53 years; there were no significant differences between the two groups. Men exceeded women in the series in a ratio of 1.6 to 1. Hiatal herniorrhaphy alone was performed in 75% of the women and adjunctive vagotomy and pyloroplasty were more frequently used in the men. Forty-four patients (19%) had concomitant biliary tract disease which was also resolved at operation.

Our method for preoperative evaluation of hiatal hernia and reflux esophagitis has been described in detail elsewhere.[5] In addition to x-ray examination, gastric analysis was used to select the optimal procedure.

Presented at the Annual Meeting of the Southern Surgical Association, December 7–9, 1970, Boca Raton, Florida.

* Dept. of Surgery, University of Florida College of Medicine, Gainesville, Florida.

This work was supported in part by N.I.H. Grant AM-13544.

Esophagoscopy is used to confirm the diagnosis in nearly all cases. Laboratory study includes direct pH measurements of the distal esophagus and pressure recordings of the lower esophageal sphincter. So far we have learned little from these studies and they are not advocated for general use. Reflux was evaluated according to the method described by Tuttle and Grossman.[4] Gastroesophageal reflux was present when the pH level was less than 4.0 at a distance of more than 4 cm. above the lower esophageal sphincter.

Three to 6 months after operation the patients routinely underwent clinical, radiologic and laboratory evaluation. Long-term evaluation was accomplished by out-patient visits, questionnaires and telephone interviews. X-ray and laboratory examinations were repeated whenever practicable.

In addition to the procedure used for repair of hiatal hernia the patients were further subdivided into those who had hernia repair only and those who had, in addition, vagotomy and pyloroplasty as follows:

Group I—Crural repair only, 65 patients.
Group II—Crural repair plus vagotomy and pyloroplasty, 62 patients.
Group III—Nissen fundoplication only, 68 patients.
Group IV—Fundoplication plus vagotomy and pyloroplasty, 36 patients.

Results

Group I

Sixty-five patients were treated by crural repair only. There was no coexistent duodenal ulcer in this group and only three patients had elevated gastric secretory rates. Three patients had undergone previous hiatal herniorrhaphy and three had esophageal strictures requiring dilatation.

Results in this group are presented in Table 1. Clinical recurrence of reflux esophagitis occurred in 13 of the 65 patients or 20%. Radiologic examination revealed anatomic recurrence of the sliding hiatal hernia in all 13 patients. pH measurements in the early postoperative period indicated gastroesophageal reflux in 11 of the 13 patients; frequently recurrent symptoms of esophagitis had not yet appeared when the test was done. Four of the 13 patients required further operation for relief of esophagitis.

TABLE 1. *Crural Repair Follow-up in 65 Cases*

Clinical Recurrence of Esophagitis	13
X-ray recurrence of hiatal hernia—13	
Esophageal reflux by pH measurement—11	
Negative reflux—1	
Not done—1	
X-Ray Recurrence of Hiatal Hernia	18
Clinical recurrence of esophagitis—13	
Asymptomatic—6	
Esophageal reflux by pH measurement—12	
Negative reflux—4	
Not done—2	
Esophageal Reflux by pH Measurement	25
Clinical recurrence of esophagitis—11	
Asymptomatic—14	
X-ray recurrence of hiatal hernia—12	
Intact repair—7	
Not done—6	

Forty-four of the 65 patients had had one or more x-ray examinations after operation. In 18 or 41% anatomic recurrence of hiatal hernia was demonstrated. Thirteen of 18 patients were judged on clinical grounds to have recurrent esophagitis. Twelve of the 18 patients with x-ray evidence of recurrence of hiatal hernia had gastroesophageal reflux demonstrated by pH measurements in the early postoperative period, whereas this study was negative in only four.

Forty-six of the 65 patients had pH studies of the esophagus in the early postoperative period. Twenty-five of these or 54% had acid pH levels in the lower esophagus, indicating gastroesophageal reflux. Eleven of the 13 patients who developed clinical recurrence of esophagitis were in the group in whom reflux was demonstrated in the

TABLE 2. *Crural Repair Plus Vagotomy & Pyloroplasty Follow-up in 62 Cases*

Clinical Recurrence of Esophagitis	9
X-ray recurrence of hiatal hernia—9	
Esophageal reflux by pH measurement—8	
Not done—1	
X-Ray Recurrence of Hiatal Hernia	15
Clinical recurrence of esophagitis—9	
Asymptomatic—6	
Esophageal reflux by pH measurement—10	
Negative reflux—2	
Not done—3	
Esophageal Reflux by pH Measurement	24
Clinical recurrence of esophagitis—8	
Asymptomatic—16	
X-ray recurrence of hiatal hernia—10	
Intact repair—10	
Not done—4	

laboratory. Conversely, only two of the 21 patients in whom no reflux was found later developed recurrent esophagitis.

Group II

Sixty-two patients were treated by crural repair of the hiatal hernia with addition of vagotomy and pyloroplasty. This group differed strikingly from Group I in that 35 had coexistent duodenal ulcers and an additional 14 patients had basal gastric hypersecretion. Three had had previous hiatal herniorraphies and six had esophageal strictures requiring dilatation.

Results in the 62 cases are summarized in Table 2. Nine of the 62 patients or 15% had persistence or recurrences of esophagitis on clinical grounds. X-ray recurrence of hiatal hernia was demonstrated in all nine of these patients. Eight of the nine patients who had clinical recurrences had gastroesophageal reflux demonstrated by pH measurements in the early postoperative period. Five of the nine patients required reoperation for esophagitis.

Forty-one of the 62 patients have had one or more x-ray examinations after operation. Fifteen of the 41 or 37% had anatomic recurrences of hiatal hernia. All nine of the patients with clinically recurrent esophagitis were in this group and the number of asymptomatic recurrences was the same as in Group I.

Forty-three of the 62 patients had laboratory evaluation of gastroesophageal reflux. pH measurements were positive for reflux in 24 of the 43 patients or 56%. Eight of the nine patients who subsequently developed clinical recurrence of esophagitis were in this group. Conversely, only one of 19 patients with a negative reflux test later developed clinical recurrence of esophagitis. Ten of the patients with a positive reflux test had anatomic recurrences demonstrated by x-ray whereas the repair remained intact in ten.

Group III

Sixty-eight patients were treated by the Nissen fundoplication as a single procedure. Two of these had coexistent duodenal ulcers and four had basal gastric hypersecretion. Seven had had previous hiatal herniorrhaphies and five had esophageal strictures requiring dilatation.

The results of fundoplication are summarized in Table 3. Eight of the 68 patients or 9% were judged by clinical assessment to have persistent or recurrent esophagitis. In sharp contrast with crural repair patients,

TABLE 3. *Nissen Repair Follow-up in 68 Cases*

Clinical Recurrence of Esophagitis	8
X-ray recurrence of hiatal hernia—1	
Intact repair-7	
Esophageal reflux by pH measurement—3	
Negative reflux-2	
Not done—3	
X-Ray Recurrence of Hiatal Hernia	3
Clinical recurrence of esophagitis—1	
Asymptomatic—2	
Esophageal reflux by pH measurement—0	
Negative reflux—2	
Not done—1	
Esophageal Reflux by pH Measurement	17
Clinical recurrence of esophagitis—3	
Asymptomatic—14	
X-ray recurrence of hiatal hernia—0	
Intact repair—17	

x-ray evidence of anatomic recurrence was found in only one of the eight patients with clinical esophagitis while an intact repair was demonstrated in seven. Gastroesophageal reflux was demonstrated by pH measurements in only three of the eight and the test was negative in two. Two of the eight patients required reoperation because of esophagitis.

Fifty-eight of the 68 patients have had one or more postoperative radiologic examinations. In only three cases (5%) was there evidence of anatomic recurrence of hiatal hernia. In the three patients the fundus has literally become "unwrapped" and the usual barium-air contrast did not demonstrate the fundic collar around the distal esophagus. Interestingly, only one of the three developed recurrent esophagitis and both asymptomatic patients have had negative pH studies for reflux.

Thirty-five fundoplication patients have had pH studies of the distal esophagus in the early postoperative period. Seventeen or 49% had positive reflux tests. Three of these developed clinically recurrent esophagitis. Conversely, two of 18 patients with negative reflux tests subsequently developed clinical esophagitis. None of the 17 patients with positive reflux tests have developed x-ray evidence of anatomic recurrence.

Group IV

Thirty-six patients were treated by fundoplication plus vagotomy and pyloroplasty. In contrast with Group III, 15 of the 36 had active duodenal ulcer disease and an additional nine patients had basal gastric hypersecretion. Five patients had previous hiatal herniorrhaphies and one had an esophageal stricture requiring dilatation.

The results of fundoplication with added vagotomy and pyloroplasty are presented in Table 4. Only two of the 36 patients or 6% had clinical evidence for recurrence of esophagitis. X-ray examination indicates an intact repair in one, while the other has not been examined radiographically. Both patients had gastroesophageal reflux demonstrated by pH measurements. Neither patient has required further operation for control of esophagitis.

TABLE 4. *Nissen Repair Plus Vagotomy and Pyloroplasty Follow-up in 36 Cases*

Clinical Recurrence of Esophagitis	2
X-ray recurrence of hiatal hernia—0 Intact repair—1 Not done—1	
X-Ray Recurrence of Hiatal Hernia	2
Clinical recurrence of esophagitis—0 Asymptomatic—2 Esophageal reflux by pH measurement—2	
Esophageal Reflux by pH Measurement	7
Clinical recurrence of esophagitis—2 Asymptomatic—5 X-ray recurrence of hiatal hernia—2 Intact repair—3 Not done—2	

Twenty-eight of the 36 patients have had postoperative x-ray examinations. Two (7%) were found to have recurrence of sliding hiatal hernia. Both remain asymptomatic, although pH measurements indicate gastroesophageal reflux is present.

Eighteen of the 36 patients have had pH studies of the distal esophagus in the early postoperative period. Seven or 39% have positive reflux tests and both patients with clinically recurrent esophagitis are in this group, although five remain asymptomatic. Also, both patients with radiographic recurrence of hiatal hernia are in the reflux positive group, while in three other patients x-ray study indicates an intact repair.

Complications

For the purposes of this report we will concern ourselves solely with complications other than recurrent esophagitis which appear to be more or less specific for the operative procedure performed. These have been four in number and are presented in Table 5.

1) **Dysphagia.** Difficulty in swallowing has occurred in all four groups of patients.

It is listed as a complication in only those patients in whom dysphagia was severe enough that bougienage was necessary on one or more occasions. This complication occurred in six patients (9%) treated by crural repair only. It was somewhat more common in patients treated by crural repair plus vagotomy and pyloroplasty, occurring in ten patients (16%). In half the patients esophageal strictures had been present before operation and could account, at least in part, for the use of bougienage. However, in the remaining half no strictures were present and dysphagia was thought to result from either the repair or the added vagotomy. This complication disappeared in less than one year in all patients.

Dysphagia was discouragingly frequent following fundoplication repair of a sliding hiatal hernia. Strictures were present in only four patients before operation which could account for the use of bougienage. This complication occurred in 16 patients (24%) after fundoplication alone and in five patients (14%) when vagotomy and pyloroplasty were added to fundoplication. The apparent lower incidence of dysphagia with vagotomy does not lend itself to ready explanation. Dysphagia was not the result of wrapping the fundus too tightly around the lower esophagus; in all cases the fundic wrap was performed with a large (#36 to #42 French) bougie present in the esophagus. Even in patients with severe dysphagia a large #46 French Hurst mercury weighted bougie passed with ease.

Persistence of dysphagia for as long as 2 years has occurred in patients with fundoplication, whereas this symptom has subsided to less than a year in all patients with crural repair. Unfortunately, no histopathologic material has become available for study and the pathophysiologic mechanism involved remains completely unexplained. Pressure studies of the lower esophagus and its sphincter indicate amplitude in the normal range and normal sphincter relaxation with deglutition.

2) **Gas-Bloat Syndrome.** We have coined this phrase to characterize a symptom complex consisting of early satiety, feeling of fullness in the epigastrium and left upper quadrant, a diminished ability to eructate and often increased borborygmi and flatus. X-rays of such patients often reveal a huge fundic air bubble. Passage of a Levin tube results in the escape of a large volume of air and immediate subjective relief. This complication is observed exclusively in Group III and Group IV patients who have had the fundoplication procedure. We interpret these events to indicate the one-way valve effect of the fundoplication. Frequently aggravated by aerophagia, air trapped in the gastric fundus fills the portion of the fundus wrapped around the esophagus. As air pressure increases the circumferential fundus occludes the esophagus for retrograde passage and eructation which normally relieves this condition cannot occur.

This symptom complex has occurred to a clinically significant degree in 37 patients (54%) following fundoplication alone and in 16 patients (44%) undergoing fundoplication combined with vagotomy and pyloroplasty. No medical therapy of any kind has been found to be helpful. In many cases the problem is mild and self-limited. Other patients are so grateful for relief of painful esophagitis that they ignore the symptoms of this syndrome. Nonetheless, this one problem causes dissatisfaction in nearly 20% of patients 1 year or longer after operation.

3) **The Dumping Syndrome.** The early postprandial dumping syndrome was largely a complication of pyloroplasty. This symptom complex occurred in 16 patients (26%) who had crural repairs plus vagotomy and pyloroplasty, and occurred in nine patients (25%) who had fundoplication plus vagotomy and pyloroplasty. Two patients developed the syndrome after fundoplication alone. Both had previously been subjected to partial gastrectomy, but had no "dumping" until after hiatal herniorraphy. It is

TABLE 5. *Complications*

	Crural	Crural, V. & P.	Nissen	Nissen, V. & P.
Dysphagia	9%	16%	24%	14%
Gas-bloat syndrome	0%	0%	54%	44%
Dumping syndrome	0%	26%	3%	25%
Post-vagotomy diarrhea	0%	13%	0%	6%

assumed that excessive use of gastric fundus in the fundoplication reduced the storage capacity of the stomach and permitted pathologically rapid gastric emptying.

Most patients responded well to dietary managment and were virtually asymptomatic after 1 year. The dumping syndrome has been severe and persistent in two patients, and a cause for dissatisfaction as long as 8 years after operation.

4) Diarrhea. This complication appeared exclusively in Groups II and IV where vagotomy and pyloroplasty were added to hiatal herniorrhaphy. Clinically significant diarrhea was present in eight patients (13%) after crural repair plus vagotomy and pyloroplasty and in two patients (6%) after fundoplication plus vagotomy and pyloroplasty. Patients with diarrhea as a part of the dumping syndrome are included in paragraph three; these few patients therefore appear to have diarrhea on the basis of vagotomy. In all but two of the patients the condition improved so the patient was not dissatisfied 1 or more years later. In two patients in Group II, however, diarrhea has been the cause of long-term dissatisfaction.

Discussion

In the Group I and Group II patients who underwent crural repair there is a striking correlation between clinical recurrence of esophagitis, anatomic recurrence of hiatus hernia and a positive gastroesophageal reflux test. It is clear that the prevention of anatomic recurrence of hiatal hernia is critically important in preventing gastroesophageal reflux and recurrent esophagitis. This finding lends credence to the concept that an intra-abdominal portion of esophagus, a closed hiatus and a normally situated lower esophageal sphincter are important in maintaining sphincteric competence. In both Groups I and II there was no instance of clinical esophagitis in patients in whom repair remained anatomically intact.

All but one of the 22 patients judged to have clinical esophagitis had positive gastroesophageal reflux tests in the early postoperative period. Conversely, only 3 of the 40 patients with a negative reflux test subsequently developed symptoms of esophagitis. There were, however, some patients with anatomic recurrence of hiatal hernia and a large number of subjects with positive reflux tests who have not developed symptoms of esophagitis. We will watch with interest their later clinical course. The technic used in the reflux test has been demonstrated to result in a substantial number of false positive tests and a positive result, therefore, has limited predictive value (J. C. McAlhany, H. F. Thomas and E. R. Woodward: unpublished data).

Crural repair of sliding esophageal hiatus hernia has been followed by a high incidence of recurrent reflux esophagitis and an even higher incidence of anatomic recurrence of the hernia. Vagotomy and pyloroplasty appear to have had little if any protective value against clinical recurrence when anatomic recurrence has supervened. The method described by Hill [2] for suturing lesser curvature structures at the esophagogastric junction to the pre-aortic fascia appears to have merit in maintaining reduction of the hernia and persistence of a segment of intraabdominal esophagus. Long-term follow-up will be necessary to determine if this maneuver will reduce the ultimate recurrence rate of crural repair.

Esophageal hiatal herniorrhaphy by Nissen's fundoplication technic has been more effective than crural repair in the prevention of recurrent esophagitis. Fundoplication has been far more effective than crural repair in the prevention of anatomic recurrence. The close correlation between clinical and anatomic recurrence present in the crural repair patients does not apply to fundoplication. Most patients with recurrent symptoms of esophagitis did not demonstrate anatomic recurrence on x-ray examination. There was, however, good correlation between clinical esophagitis and a positive esophageal reflux test. As in crural repair patients, however, there were many patients with a positive reflux test who have not as yet developed recurrent esophagitis. Conversely, a negative esophageal reflux study had excellent prognostic value. Only two of 29 patients with a negative reflux test subsequently developed clinical esophagitis.

These findings imply that in many of our fundoplication patients gastroesophageal reflux occurs in the presence of and in spite of an intraabdominal portion of esophagus, a closed hiatus and a normally situated and theoretically augmented lower esophageal sphincter. We must acknowledge either a high incidence of gastroesophageal reflux in the presence of an intact Nissen fundoplication or else postulate a high incidence of positive reflux tests. We have found that both factors are operative. Radiologic examination has demonstrated reflux in postoperative fundoplication patients who have an anatomically intact repair. Other studies have also demonstrated a substantial number of false positive reflux tests.

The Nissen fundoplication repair of sliding esophageal hiatus hernia is associated with acceptably low recurrence rates of both esophagitis and hiatus hernia. However, the morbidity associated with this procedure has been very significant. There is a high incidence of dysphagia requiring bougienage. The pathophysiology is not apparent, since the repair is done over a large esophageal bougie. There is an even higher incidence of the so-called "gas-bloat syndrome." This symptom complex appears to result from interference with normal eructation of swallowed air, permitting the accumulation of pathologic amounts of gas in the fundus and excessive passage through the intestinal tract.

We interpret our experience to mean that this high morbidity precludes the utilization of the Nissen fundoplication in the primary surgical management of uncomplicated sliding hiatal hernia with esophagitis. We believe that the Nissen principle is useful in preventing recurrent esophagitis in patients with complications such as recurrence after previous hiatal herniorrhaphy and in patients with peptic strictures of the esophagus.

The role of vagotomy and pyloroplasty as an adjunctive measure in the treatment of patients with hiatus hernia and esophagitis is difficult to define precisely. In Group II and IV where these procedures were added to hiatal herniorrhaphy, most patients had either or both duodenal ulcer disease and gastric hypersecretion. Esophagitis is generally considered to be more severe and more often associated with complications in patients who have coexistent duodenal ulcer. Concrete evidence for this assumption is not clearly evident.

When one compares Group II with Group I it does appear that vagotomy and pyloroplasty added to crural repair does not protect the patient against recurrent esophagitis when anatomical recurrence of the hernia occurs. In Group III and IV treated by fundoplication, the addition of vagotomy and pyloroplasty in Group IV appears to have had little, if any, effect on the clinical result. On the other hand, there has been a definite morbidity from the additional surgical treatment in the form of the dumping syndrome and postvagotomy diarrhea. Although these complications are self-limited in most cases, it appears evi-

TABLE 6. *Summary*

	Clinical Recurrence of Esophagitis	X-Ray Recurrence of Hiatal Hernia	Esophageal Reflux by pH Measurement
Crural	20%	41%	54%
Crural, V. & P.	15%	37%	56%
Nissen	9%	5%	49%
Nissen, V. & P.	6%	7%	39%

dent that the addition of vagotomy and pyloroplasty is not worthwhile as a routine measure and in all probability should be reserved for those patients with obvious duodenal ulcer disease.

Summary and Conclusions

This report reviews 231 patients with sliding esophageal hiatus hernias and peptic esophagitis. The results are summarized in Table 6. One hundred and twenty-seven patients were treated by crural repair and 104 patients by Nissen fundoplication. Ninety-eight of the patients underwent vagotomy and pyloroplasty in addition to hiatal herniorrhaphy. Clinical esophagitis recurred in 18% of patients treated by crural repair. Thirty-nine per cent of patients with crural repair were found on radiologic examination to have anatomic recurrences of hiatal hernia. All clinical recurrences had anatomic recurrence. Vagotomy and pyloroplasty did not prevent recurrence of symptoms in patients who developed anatomic recurrence.

Fundoplication hiatal herniorrhaphy was considerably more effective than crural repair in preventing both clinical recurrence of esophagitis and anatomic recurrence of hiatal hernia. Again, the addition of vagotomy and pyloroplasty appeared to have no significant effect. However, fundoplication was associated with a high incidence of postoperative dysphagia and the "gas-bloat syndrome."

Laboratory evaluation of gastroesophageal reflux by direct pH measurements demonstrated that a negative postoperative test was associated with a favorable prognosis. Very few patients with either crural repair or fundoplication subsequently developed clinical recurrence of esophagitis. Nearly all patients with recurrent symptoms developed gastroesophageal reflux after operation; however, many patients with positive tests in both groups remained asymptomatic.

On the basis of the data presented the following conclusions are reached:

1) Crural repair of sliding esophageal hiatus hernia successfully prevents recurrent reflux esophagitis only when anatomic recurrence is prevented. Both clinical and anatomic recurrence are discouragingly high when traditional technics are used.

2) The Nissen fundoplication procedure effectively prevents both clinical recurrence of esophagitis and anatomic recurrence of hiatal hernia. However, side effects of the procedure are frequent and the morbidity is clearly too high. This procedure should probably be reserved for recurrent and complicated problems of reflux esophagitis.

3) The addition of vagotomy and pyloroplasty to either type of herniorrhaphy does not appear to improve the clinical result and apparently does not prevent recurrent esophagitis with anatomic recurrence. In our opinion vagotomy and pyloroplasty should be added to hiatal herniorrhaphy only in the presence of duodenal ulcer.

4) Laboratory evalution of gastroesophageal reflux by direct pH measurements has had good predictive value only when the reflux test has been negative.

Acknowledgment

The authors acknowledge the invaluable assistance of Miss Merle Fowler, R.N., in obtaining follow-up information on patients.

References

1. Allison, P. R.: Reflux Esophagitis, Sliding Hiatal Hernia and the Anatomy of Repair. Surg. Gynec. Obstet., 92:419, 1951.
2. Hill, L. D., Tobias, J. and Morgan, E. H.: Newer Concept of the Pathophysiology of Hiatal Hernia and Esophagitis. Amer. J. Surg., 111:70, 1966.
3. Nissen, R.: The Treatment of Hiatal Hernia and Esophageal Reflux by Fundoplication. *In* Hernia. Edited by Lloyd M. Nyhus and Henry M. Harkins. Philadelphia, J. B. Lippincott, 1964.
4. Tuttle, S. G. and Grossman, M. I.: Detection of Gastroesophageal Reflux by Simultaneous Measurement of Intraluminal Pressure and pH. Proc. Soc. Exp. Bio. Med., 98:225, 1958.
5. Woodward, E. R., Rayl, J. E. and Clarke, J. M.: Esophageal Hiatus Hernia. Current Problems in Surgery. December, 1970.

Discussion

Dr. Alton Ochsner, Jr. (New Orleans): For years we have been discussing the treatment of hiatus hernia, but I dare say there is not one surgeon in this audience who treats hiatus hernia. Gentlemen, we all know that what we are treating is gastroesophageal reflux, and we will, of course, repair the hiatus hernia when it exists—and it usually does—if this seems to be helpful to do so.

Of course, all of us in this room understand what I am referring to, but the physicians who see these patients initially with this problem of gastroesophageal reflux may not be attuned to the fact that this condition can exist without a clinically demonstrable hiatus hernia. Even if they recognize this condition as reflux, they believe from what we have written that we surgeons have nothing to offer their patients unless a hiatus hernia or a stricture has occurred.

If there is any doubt, one should refer to the medical students' textbooks. They state under the subject of peptic esophagitis that it is not considered a surgical problem unless a hiatus hernia or a stricture has occurred.

To be sure, this may constitute a logical conclusion if one reads the titles of the surgical papers on this subject.

My opinions about this subject have been crystallized by four of the patients with gastroesophageal reflux that I treated this year. All had problems of severe chest pain which in two had been diagnosed as angina pectoris. They had apparently received some relief—not complete—over a period of years with medical treatment for this condition. They were sent to me, interestingly, enough, as candidates for aortocoronary bypass. In discussing these patients with their referring physicians, I asked if the esophagus had been investigated, and each physician stated: "Yes, and the upper GI series had shown no hiatus hernia." As you might expect, the coronary angiograms were normal, and further investigation of the esophagus with water-lavage test, endoscopy, and pressure studies revealed significant gastroesophageal reflux, and those patients were relieved by surgical correction.

The third patient had chest pain that was undiagnosed, and, to quote the referring physician, "It sure sounds like hiatus hernia, but the upper GI series is normal." This patient also responded to surgical correction of the gastroesophageal reflux.

The fourth patient had been correctly diagnosed as having gastroesophageal reflux with hiatus hernia. He was under the care of a very fine internist, but surgery was not recommended because, to quote the internist, "the hiatus hernia was very small." This young man, a personal friend of mine, sought relief from what he considered an intolerable existence of constant upright position, bland diet, antacids, and antispasmodics. Of course, surgical correction of the gastroesophageal reflux in this instance gave him relief.

Whereas hiatus hernia is not necessarily a problem, I agree with Dr. Carr that gastroesophageal reflux is; and if we are going to prevent those esophageal complications and pulmonary complications and allow these patients to be recognized early and treated early, we are the only people—the only physicians—that have something definitive to offer them. But they are not going to be recognized unless we start thinking, talking, and especially teaching *reflux, not hernia.*

Dr. Otto C. Brantigan (Baltimore): It is obvious that the patient suffering with esophagitis is a very emotional type of individual, and one speculates whether or not the patient has esophagitis for this reason, a predisposing cause, or whether the patient becomes emotional because he goes through life with no one treating or concerned with the esophagitis until he gets into serious trouble, with some complication, such as bleeding, stricture, et cetera.

The use of vagotomy is mentioned from time to time. If vagotomy is to be used, a selective gastric vagotomy will eliminate most of the post-