DIAPHRAGMATIC HERNIA

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IN THIS PAPER I propose to concentrate on the diaphragmatic hernia of adult life, which is nearly always acquired. So many stout women have suffered from this form of hernia, often with no sympathy from their doctors or friends, who have called them neurotic, that there is some justification for emphasizing the common type of hernia which has so rarely been diagnosed in the earlier years of its presence.

A simple classification is:

1. Congenital—various types (including hiatal hernia).
2. Acquired
   - A. Traumatic
   - B. Hiatal
     - a. Sliding
     - b. Para-esophageal
     - c. Both types.
3. Eventration.

Undoubtedly, congenital hernias, other than at the hiatus, are rarely met in life. But when they are encountered, especially in the new-born infant, they need skilful and immediate care.

CONGENITAL HERNIA

If large, this may cause severe dyspnea in infancy. I was asked to see an infant two days old because of cyanosis and suspected atelectasis. Physical signs showed that breath sounds were confined to the left side of the chest, the heart was pushed over to the same side and the child was cyanosed when out of the oxygen tent. Plain roentgen ray examination showed that bowel was present in the right pleural cavity. A very small quantity of barium confirmed the diagnosis. At each feed the child became very cyanosed until it brought up wind. The attacks of cyanosis could be relieved by passing a catheter into the stomach to allow air to escape from it, thus reducing the pressure on the heart and lungs. These infants need urgent attention. Unless the hernia can be reduced and repaired within two or three days of birth, as the bowel becomes more distended with air it becomes more difficult to replace it in the abdomen; moreover the child may die of anoxia in the meantime.

The hiatal type of hernia also occurs in infants, but it is rarely recognized. It may cause the same complications as occur in the adult with a hiatal hernia. Free regurgitation of gastric contents causes esophagitis and peptic ulceration. Such infants vomit almost from birth; not the simple regurgitation of the normal infant, but forceful projective vomiting. Hematemesis, failure to thrive, and unwillingness to feed because of pain, are common symptoms. If untreated, some of these children develop esophageal strictures. In the past, most of these little patients have been treated medically, but there is increasing evidence that early surgical correction of the hernia is the safest way of avoiding these distressing complications. Why some infants cannot tolerate gastric juice in the esophagus, yet others seem immune to its irritation, is not yet understood.

TRAUMATIC HERNIA

This was common during World War II, but is rare in civilian practice and is nearly always due to a severe crushing injury of the lower chest or upper abdomen. It should be suspected whenever atypical dyspepsia occurs, even months or years after such a severe injury. The important injury is the one that affects the left side of the diaphragm, allowing the stomach, colon or small intestine to migrate into the left side of the chest. On the right side, the liver usually blocks the hole in the diaphragm and prevents the bowel passing into the chest. Clearly, the symptoms will depend on the contents of the hernia, but they may be dyspepsia, vomiting or even acute small or large gut obstruction. The diagnosis will not be missed if the possibility of hernia after some severe injury is not forgotten, for as a rule a radiograph of the chest will be enough to indicate its presence, which is confirmed by a barium meal. Difficulty may arise when there is no history of such a crush injury, particularly in cases where there may have been head injuries and unconsciousness, or other severe injuries on which attention has been focussed to the exclusion of the crush injury.

In reporting 1639 penetrating chest wounds in 1946,[6] I could record only five left-sided hernias which I had repaired, and one right-sided. There were, of course, many smaller tears in the diaphragm sutured by forward-area surgeons, whose good work is well reflected in the very low incidence of hernia at the base.

As a rule, the traumatic hernia is not difficult to repair, for there has usually been a rent in the muscle fiber of the diaphragm, with little loss in tissue. Where loss in tissue makes the repair difficult, it may most easily be done by mobilizing the costal attachments of the diaphragm posterolaterally and re-attaching this part of the diaphragm higher up—maybe to the seventh or eighth ribs. The flattened diaphragm, so constructed, is usually then adequate to bridge the gap. A phrenic crush will often help.

I have included eventration in my classification, although, of course, it is not a hernia. I find, however, that people do not always agree about what eventration is. Bisgard[2] defines it as "an abnormally high position of one leaf of the intact diaphragm as a result of paralysis, aplasia or atrophy." This broad definition would therefore include paralysis produced by a phrenic operation; but most authors limit the definition to cases of congenital or idiopathic origin.

I believe that there is a place for plicating the high eventrated diaphragm in children; for it must limit the function of the lung and perhaps embarrass cardiac action. In many cases, however, it is entirely symptomless and a chance finding.

HIATAL HERNIA

This is the common diaphragmatic hernia. Normally the esophagus passes through a muscular sling made by the decussation of the fibers of the right crus of the diaphragm. Of necessity the hiatus must be sufficiently large to allow food to pass into the stomach; but the lower end of the esophagus must be so anchored to the crus that the stomach is not drawn up into the chest during contraction of the longitudinal fibers of the esophagus. Moreover, the size of the hiatus must not be too big, for otherwise the intra-abdominal pressure would push abdominal organs up into the chest, where the pressure is less than atmospheric. What stops the stomach from sliding up into the posterior mediastinum, since both suction and the longitudinal muscles of the esophagus should pull it there? There is a dense connective tissue "cone," sometimes termed the phreno-esophageal ligament, filling the hiatus. Above, it passes into the very extensive fascia propria of the esophagus; below into the lesser omentum on the right and to

the suspensory ligament of the stomach on the left. Moreover, during inspiration, when suction in the chest is maximal, the right orus contracts and holds down the esophagogastric junction.

Excessive deposition of fat, atony of the diaphragm and increase in abdominal pressure will all predispose to hernia. In the past, much has been written about congenital weakness of the hiatus and congenital short esophagus. Now it is thought that most of these hernias are acquired, though there is nothing unreasonable in the congenital theory. As the stomach descends through the posterior mediastinum, if the diaphragm closes around it before it is completely descended, naturally a wide hiatus is the result. The stomach may continue its descent till it is below the diaphragm, but a large hiatus persists. A persistent para-esophageal congenital hernial sac may also occur.

TABLE I.

Symptom	Cases	Duration
Repaired		
Dyspepsia	32	1 month –30 years
Dysphagia	7	1 month –4 years
Anemia	6	3 months–12 years
'Angina'	1	3 years
'Asthma'	2	2–4 years
Not Repaired		
Dyspepsia	16	6 months–28 years
Dysphagia	11	1 month –6 years
'Angina'	2	2–3 years

It is important to recognize that in the many cases of sliding hernia, the symptoms are largely due to impaired function of the cardiac sphincter, allowing gastric contents to regurgitate freely into the esophagus. What is the cardiac sphincter? Does it exist? One authority[1] stresses the importance of the sling of the right crus, which lassoes the lower end of the esophagus down to the lumbar spine. In inspiration, when the tendency to herniation and regurgitation is most, the crus increases its grip on the esophagus and holds it down, compressing the lumen laterally. The next man[3] describes the oblique entry of the esophagus into the stomach and the importance of this "valve" in preventing regurgitation. Though both these mechanisms are extremely important, there still remain the circular muscles of the cardia itself, which are difficult to dismiss as completely without any value as a sphincter. Probably all three mechanisms are important and we should do our best to preserve them all in any operation on a hiatal hernia.

HIATAL HERNIA

Anatomy. The sliding (pulsion) and the paro-esophageal (or rolling) types of hernia have been so well described by Harrington and others, that there is no need to define them again. It should be noted, however, that as the para-esophageal hernia increases in size, it tends to become partially sliding too, so that the cardia comes to lie above the diaphragm. The essential difference in the two types of hernia is the position and function of the "cardiac sphincter," which is patulous, allowing reflux of gastric juice in the sliding hernia.

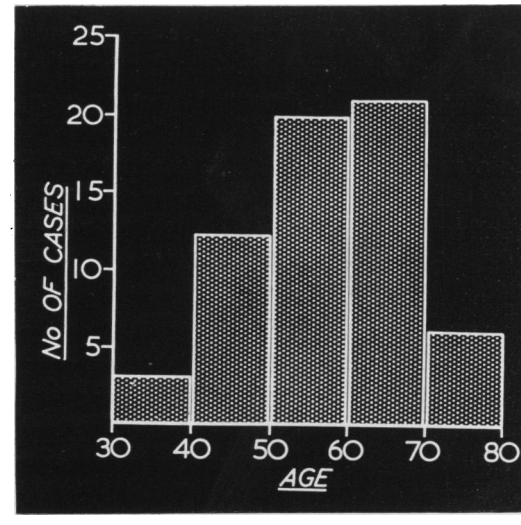

FIG. 1.—Age distribution of patients.

Harrington[5] states that the "circular muscles bundles of the esophageal hiatus are

innervated chiefly by the phrenic nerve of the same side, whether the muscles originate from the crus of the same side or of the opposite side." As the crura are developed from the Wolffian body mesoderm, however, they may receive a nerve supply from the first lumbar nerve. When I have crushed the left phrenic nerve during operations on hiatal hernias, I have usually found that it paralyzed the left side of the "pinchcock," but did not affect the right side, thus confirming Harrington's statement for the left phrenic nerve.

TABLE II.—*Management of Hiatal Hernia (1947–1951) 62 Cases; Para-esophageal—15, Sliding—44, Both—3.*

Type of Operation	No.	Remarks	Death
Repair by thoracotomy	32	13 paraesophageal 16 sliding 3 both	2 deaths (1 carcinoma cardia) (1 pulm. embolus on 4th day
Method of repair in the 32	..	10 repaired by anterior displacement 22 repaired by suturing right crus	
Phrenic crush or phrenicectomy in addition to repair in the 32 cases	..	12—predominantly in the anterior displacement operation	
Attempted repair of a recurrent hernia, previously operated upon twice, which failed	1	Jejunostomy	
Phrenic crush tried before repair (included in 32 cases above)	(2)	Failed in both	
Phrenic crush only	5		1 death (cerebral thrombosis)
Phrenicectomy only	3		
Conservative treatment with dilatation of stricture	7		1 death from perforation
Conservative treatment only	13	Only 1 paraesophageal, unfit for operation	
Investigation by esophagoscopy	1	Perforation	1 death

Symptoms. Sliding hernias probably develop during pregnancy and reduce themselves after confinement; to recur with the next pregnancy. Five of my patients ascribed the onset of symptoms to pregnancy. I believe that, if we had barium meal examinations as a routine during the eighth month of pregnancy, hiatal hernia would be found as common as umbilical hernia at that time. Reflux esophagitis is the most serious complication of sliding hernia, but not everybody with a hiatal hernia is troubled by it. It is common enough, however, as I shall indicate later. The patient has intense burning pain behind the lower end of the sternum, quite agonizing on swallowing, often occurring at two o'clock in the morning and awaking her. It radiates to the back on either side, into the arm (left in my cases), into the ears (right in two of my cases), to the palate and vaguely up into the neck. It is always worse when she stoops or lies down; it is relieved when she stands or sits up. It is relieved by alkali. It is worse when she lies on the right (in four cases). Acid froth easily comes up into the mouth, usually to be swallowed again. Often there is a vague feeling of food sticking at the lower end of the sternum. These symptoms are typical of esophagitis.

Belching, fullness after meals, and epigastric discomfort are usual and may occur without the symptoms of esophagitis in those lucky individuals who seem to be immune to the irritation of gastric juice on the esophageal mucosa. In some of these patients, although there is a small sliding hernia, the hiatus is not so stretched that the "pinchcock" is inactive, so that a physiologic sphincter persists. Indeed, not infrequently the slighter symptoms in these cases can be abolished by paralyzing half the "pinchcock" by a left phrenic crush. This relieves the spasm which causes distension of the gastric pouch; whether the abolition of the spasm will later lead to reflux esophagitis remains to be seen. Harrington advocated phrenic crush for the small sliding hernia, though he admitted that it did not always relieve the symptoms entirely. In my earlier cases I suggested to the patient that we might try the effect of a phrenic crush

first, before proceeding to a major operation, and I have had some success with the method. But I am sure that it is of least value in cases of esophagitis, and of most value in those patients who have recurrent attacks of distension of the gastric pouch. But even when the hiatus is so stretched that the cardia is incompetent, I have thought that it might be better sometimes to para-

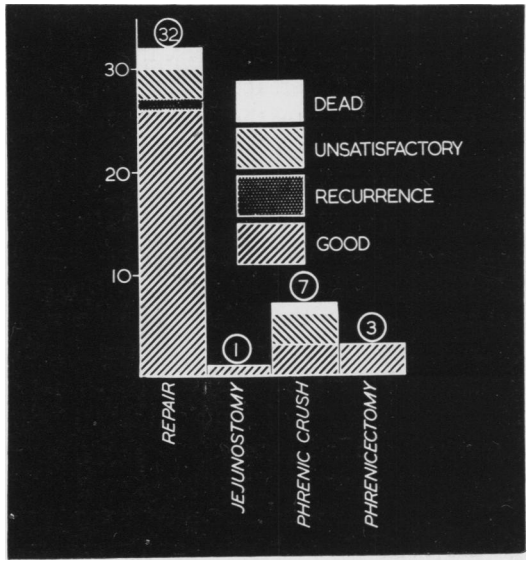

FIG. 2.—Results of operation.

lyze the phrenic. The movement of the diaphragm may act like a "squeege," emptying the gastric juice from the gastric pouch into the lower esophagus. Certainly I have had success with a phrenic crush in such a patient.

Flatulence, wind around the heart, "asthma," anemia, severe attacks of upper abdominal pain and vomiting for two or three days with remissions for several weeks, are all common symptoms in the patient who has a para-esophageal hernia. Esophagitis does not occur. There may be slight dysphagia. Gastric ulcers high up near the cardia occur; they may be the result of constant trauma to the stomach by the contracting crus. They are sometimes the only cause of symptoms and are quite easily missed in the barium meal examination. It is probable that these gastric ulcers will heal when the hernia is repaired; but I have excised them in my cases for they had caused severe anemia and I was not prepared to risk further hemorrhage.

Anemia may be severe in both types of hernias, either due to bleeding from gastric ulcers, or from partial strangulation of the stomach, or weeping from a diffuse esophagitis. Strictures are the direct result of chronic esophagitis and occur only in sliding hernias. The stricture prevents further regurgitation of gastric juice and is nature's remedy; unfortunately the patient starves. Dysphagia is more marked in the group treated conservatively.

DIAGNOSIS

This is not difficult, but it is still missed too often, because the clinician does not consider the possibility and therefore does not ask the radiologist to investigate the cardia properly. Though we must emphasize the importance of a thorough examination of the cardia by the radiologist, we must not assume that all dyspepsias are necessarily due to the hiatal hernia, when this has been demonstrated. Though the gallbladder has too frequently been removed in the past for symptoms caused by a hernia, we may well be in danger of falling into a similar error and repair a hernia for gallbladder trouble.

I need not stress the importance of the barium meal, with the patient inclined head down and with pressure applied to the upper abdomen. I like to have a cholecystogram in all cases where there is not clinical evidence of esophagitis. I have done test meals in only 13 patients, to try to correlate the amount of esophagitis with the acid secretion of the stomach. In all except two patients, who both had peptic ulcers of the stomach, the acid was normal or lower than normal; so that there appears no direct relation between it and esophagitis in these few cases. Esophagoscopy is always neces-

sary. It confirms the presence of esophagitis. A sliding hernia may show on roentgen ray examination, but not until esophagitis has been seen can we always conclude that it is the cause of the symptoms. I have sometimes been surprised, however, to find much more peri-esophagitis at operation than the esophagoscopy led me to expect. In 42 sliding hernias, I found esophagitis in 19, with strictures in six of these. The proportion is probably higher than this, because in a few cases esophagoscopy had not been done. These strictures, occurring as they do in later life, are often thought at first to be malignant. The casual endoscopist frequently so describes them. I have known a confident diagnosis of cancer made on the endoscopic appearance alone, unsupported by actual biopsy. In each case a careful clinical history showed that the patient had suffered from regurgitation of acid for years before the dysphagia developed, symptoms which the patient had not thought worthy of serious attention. The clinical picture of the patient, as well as the history, was against the diagnosis of cancer. But unless due attention is paid to the history, such strictures may be diagnosed and treated as malignant.

INDICATION FOR OPERATION

1. All para-esophageal hernias should be operated upon if the patient is fit, because they may strangulate, incarcerate, or cause a gastric ulcer which bleeds or perforates, or may progress to become sliding hernias, or cause cardiac embarrassment and are unrelieved with conservative treatment. Moreover they are relatively easy to repair.

2. Sliding hernia should be operated upon and repaired:

(a) If the esophagus is not unduly shortened. Some shortening is permissible and, as Allison points out, the esophagoscopy will give a better idea of the true length available because the 'scope pushes the esophagus down ahead of it; this gives the observer some idea of the reducibility of the hernia.

(b) If a stricture is not present.

(c) If the patient is not too gross to preclude any chance of permanent repair.

(d) If the patient will not become pregnant again.

(e) If the symptoms justify a major operation.

(f) If the symptoms are undoubtedly due to the hernia.

(g) If the general condition is satisfactory.

TABLE III.—*Follow-up of Conservatively Treated Cases.*

Dead (from perforations)	2
Worse (severe stricture)	2
Unchanged	5
Improved	9
Complete relief	2
Untraced	1
Total	21

Absolute contraindications are a very short esophagus with a stricture, and a very obese patient who will not diet. But the majority of patients, who have symptoms, welcome an operation. Those with slight symptoms, especially if esophagoscopy shows no esophagitis, are best with conservative treatment. This must insist on the minimum of corsetting, a propped up position at night and regular alkali therapy.

OPERATION

What operation should be done? Should it be by the abdominal route or by the chest? Thoracic surgeons will choose the latter route, but we must have substantial reasons other than habit for adopting it. It is easier to mobilize the esophagus by direct vision than blindly; the exposure of the hernia is better.

Mistakes in diagnosis do occur. A carcinoma at the cardia may be associated with a hernia; it may even cause a hernia if the carcinoma is in the lower end of the esophagus, dragging the stomach up into the chest

FIG. 3

FIG. 4

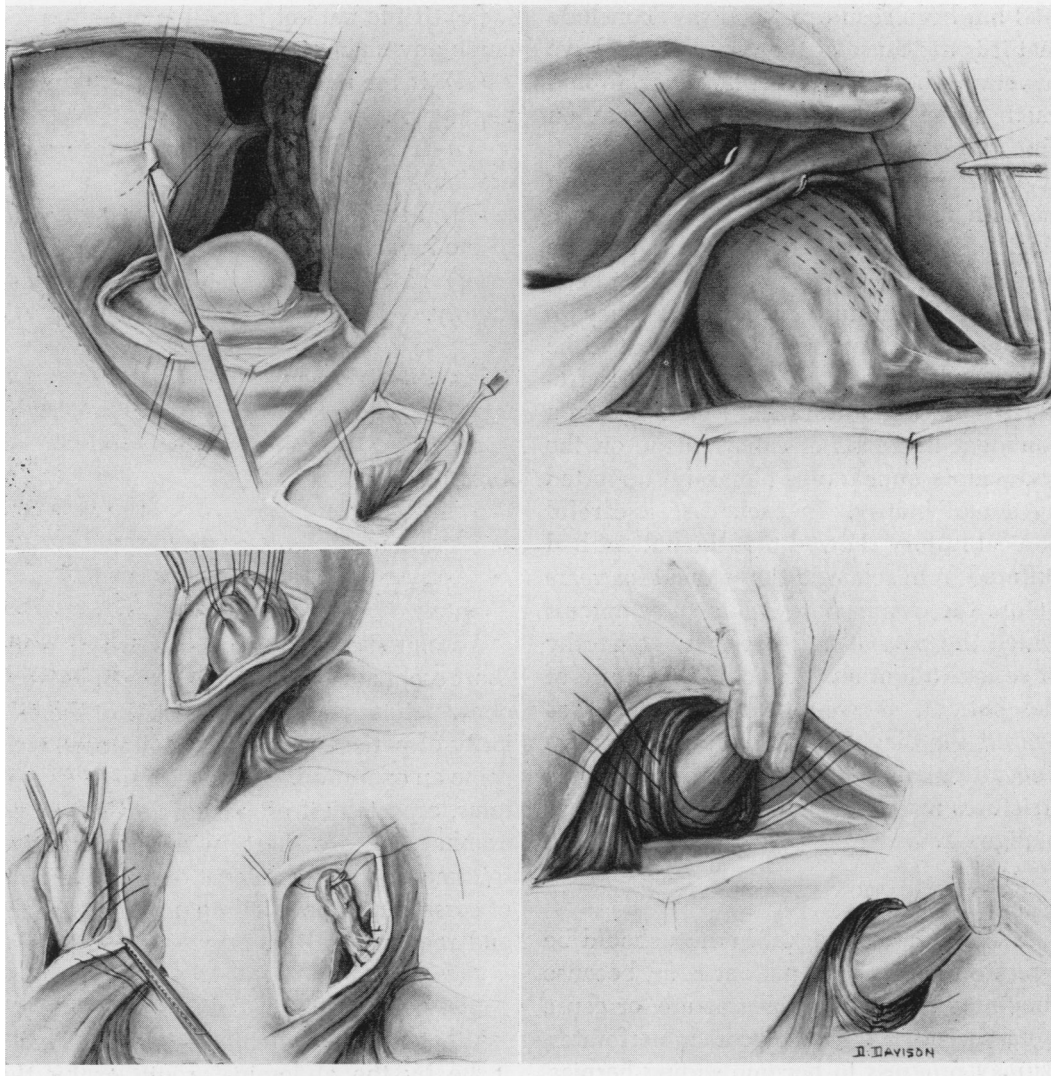

FIG. 5

FIG. 6

FIG. 3.—This shows sac exposed after incision of mediastinal pleura. Diaphragm is incised at right angles to the main wound to avoid cutting the phrenic nerve.

The inset shows the peritoneum being stripped from the under surface of the diaphragm.

FIG. 4.—A rubber tube sling has been passed around the esophagus. The fingers of the left hand have been passed through the diaphragm into the peritoneal cavity and now lie inside the sac. A slip of the phreno-esophageal ligament passing from the sac to the esophagus is well shown. Sutures are now being passed to plicate the sac and will be tied on the under surface of the diaphragm.

FIG. 5.—These three drawings show that the sac has been dragged below the diaphragm in the extraperitoneal tissues. After plication it is sutured to the under surface of the diaphragm.

FIG. 6.—This shows the stitches passed through the decussating fibers of the right crus and the final step is shown in the inset with the repair completed.

by fibrosis. It is easy to extend the thoracic incision into the abdomen if necessary; but difficult to do the reverse through the usual mid-line or paramedian incision. Even Wangensteen's recent advocacy[8] of the lower sternal split does not seem the complete answer to the abdominal approach, though I have no experience of this incision. Against this, we must admit that the abdominal approach enables the surgeon to deal with gallstones, should these be present.

Certainly, the ideal is to replace the hernial contents, excise or plicate the sac and approximate the decussating fibers of the right crus behind the gullet. This is usually not difficult in para-esophageal hernia. In some of the more obese patients with sliding hernia, the crural fibers seem remarkably thin. In some of my earlier cases, I found so little to stitch behind the esophagus that I deliberately cut right across the anterior hiatal ring and displaced the esophagus forward. This unphysiologic procedure was invariably accompanied by a phrenic crush or phrenicectomy. I do not advocate this operation; I merely record it. I am surprised that the results have not been bad. Wangensteen[7] has described this operation.

My standard operation is to excise or plicate the sac, suturing it to the under surface of the diaphragm. The decussating fibers of the right crus are then sutured lightly together. A stomach tube is passed so that, when the last suture is tied, the tip of the little finger can be introduced between the gullet and the suture line. I prefer to have the last suture next to the esophagus of catgut; the others are of thread. Usually the phrenic nerve is preserved; but if there is difficulty in mobilizing the esophagus because of shortening, it is crushed.

Results of Treatment. In this series of 62 patients, the hernia was repaired in 32. Perhaps this may seem to imply an unduly radical approach to the problem of hiatal hernia. But my patients formed a somewhat selected group, for many of them were referred to me by physicians and surgeons who had already advised conservative treatment without success; and most of the patients, realizing already that they had not benefited by medical measures, were eager for an operation. I realize that the follow-up is short and that the results are based on clinical assessment; only those patients with an unsatisfactory clinical result have had a postoperative barium meal. Gertz's[4] series of 45 cases showed how frequently a recurrence can be demonstrated by roentgen ray even in the absence of symptoms. Nevertheless, the immediate benefit has been so striking in many cases that I feel justified in including an analysis of my results in the tables.

Figure 2 shows the results of operations. The patient, completely relieved by a jejunostomy, by which he was fed for two months, deserves some comment. He had been operated upon twice by other surgeons for repair of a sliding hernia without success, and he had very severe esophagitis with ulceration. I could not mobilize his esophagus, and therefore abandoned attempts to repair his hernia. He improved so much with the jejunostomy, all evidence of esophagitis disappearing at subsequent esophagoscopy, that he has remained well and working for the last three years, during which his only treatment has been to adopt an elevated position of the shoulders at night. The phrenic operations have not been very satisfactory and rather unpredictable in their results. They have most often been done in poor risk patients with small hernias. There have been two cases of dysphagia after repair of the hernia. In one it was transient and needed no treatment, but in the other patient it persisted and another operation was required to free the cardia where the diaphragm had been closed too tightly around it.

I have been most gratified to see how well most of these patients were after operation—

well and pleased—but alas, some of them so pleased that they have already started to eat everything and put on weight!

The patients treated conservatively have also been followed. There were 19, six of them with strictures. These strictures have required an average of two dilatations each to keep the patients swallowing comfortably, and with one exception these patients are happy and improved; the exception is a lady whose stricture I have only succeeded in passing once. Most of these patients with strictures are elderly.

The figures in Table III suggest that some of these patients should have an operation; but I have hesitated to resect the inflamed esophagus and do an esophago-jejunostomy until I know more of the natural history of the disease. I think only the one severe stricture patient, who is worse, will require such an operation. The others have remissions of symptoms, and are mostly old and unfit for major operations. Some of those improved had only slight symptoms, not enough to justify operation.

In conclusion, it will be obvious that there remain many problems to be solved. Is it worth trying to repair a hiatal hernia at all, or are the results so poor that no operation is justified? I am certain that operation can repair a sliding hernia, but it is probable that there will always be a high rate of recurrence. Indeed, some surgeons feel that it is better to reduce gastric secretion by vagotomy or partial gastrectomy than attempt to repair the sliding hernia. This I feel is unduly pessimistic. The most difficult patient is the one who has severe esophagitis with stricture and a fixed irreducible hernia. Here, any radical operation such as resection of the ulcerated esophagus and esophago-jejunostomy must be dangerous in these old people, while esophago-gastrostomy is theoretically undesirable, because the reflux of gastric juice persists. Fortunately, in my experience, most of these older patients can be managed with gentle dilatation and conservative treatment.

Acknowledgments. It is a pleasure to thank my colleagues, particularly Mr. Graham Bryce, who have referred these patients to me and helped me in this investigation.

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