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Stricture And Ulceration Of The Esophagus Secondary To Hiatus Hernia

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STRICTURE AND ULCERATION OF THE ESOPHAGUS SECONDARY TO HIATUS HERNIA

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"I think I am warranted, from the result of all the experiments, in saying that the gastric juice, so far from being "inert as water," as some authors assert, is the most general solvent in nature of alimentary matter—even the hardest bone cannot withstand its action. It is capable, *even out of the stomach,* of effecting perfect digestion."

—William Beaumont, Experiments and Observations on the Gastric Juice and the Physiology of Digestion. 1833

It is a curious fact of nature that the esophageal lining is extremely sensitive to the secretion of the stomach. The susceptibility of a squamous cell surface to the action of the gastric juice is well known to anyone who has cared for a gastrocutaneous fistula. William Beaumont[1] commented on the gastric juice "excoriating the edges of the aperture and skin" of his patient, Alexis St. Martin. The mechanism at the esophagogastric junction preventing gastric juice from entering the esophagus is normally very efficient. That this mechanism is impaired by herniation of the stomach through the esophageal hiatus, has been appreciated for a relatively short period of time. For example, Dr. Roy McClure,[2] in 1933, reviewing the experience at this hospital of lesions of the esophagus, mentions one patient with "peptic ulcer of the esophagus" and stricture formation who was treated by repeated dilatation and finally by gastrostomy for malnutrition. He made no comment as to whether the patient had a hiatus hernia. As recently as 1937 Lyall[3] described eight autopsied cases of esophageal ulcer without comment as to the presence or absence of hiatus hernia. In a short paper published in 1939 Briggs, Dick and Hurst[4] said that esophageal ulcer was almost always associated with hiatus hernia; whereas, 5 years before the senior author[5] wrote about both ulcer of the esophagus and hiatus hernia in the same paper without connecting the two conditions. As nearly as I can tell, hiatus hernia was first implicated as the cause of esophageal stricture by A. Brown Kelly[6] who discussed, in 1936, "Progressive Narrowing and Shortening of the Oesophagus with Hiatal Hernia," in a paper about esophageal lesions in children.

In patients with hiatus hernia, gastric reflux into the esophagus can be stopped by replacing the stomach beneath the diaphragm. However, when repeated burning of the lower esophagus takes place over a period of years, the esophagus not only becomes strictured, but also fixed in the mediastinum by an inflammatory process.

This complicates the problem of surgical correction in two ways. Measures that are adequate in reducing the uncomplicated hernia fail when the hernia is complicated by stricture. Even if the hernia can be reduced, the problem of esophageal obstruction remains. In the earlier experience with repair of hiatus hernia, surgeons such as Harrington[7] using an abdominal incision, admitted their inability to reduce the hernia in strictured cases. These hernias were consigned to a separate classification, labeled short esophagus, as unsuitable for operation. This was probably just as well. Ton[8] described a situation in which forcible abdominal traction on the stomach in such a case led to tearing the stomach off the esophagus. Patients with hernia and stricture were treated by peroral dilatations, which temporarily opened the food passage. However, it is necessary to stop the reflux of gastric juice into the esophagus, if it is to remain open. This can be done by repair of the hernia.

The stenosis of the esophagus has led surgeons, such as Allison[9] and Barrett,[10] to resect the lower esophagus and substitute a loop of jejunum brought up from the abdomen to join the upper esophagus to the stomach. The arguments for and against resection of the stenosed esophagus involve interpretation of the altered epithelium of the lower esophagus associated with hernia and stricture. The epithelial lining of the esophagus in these cases may not be squamous like the normal esophagus, but

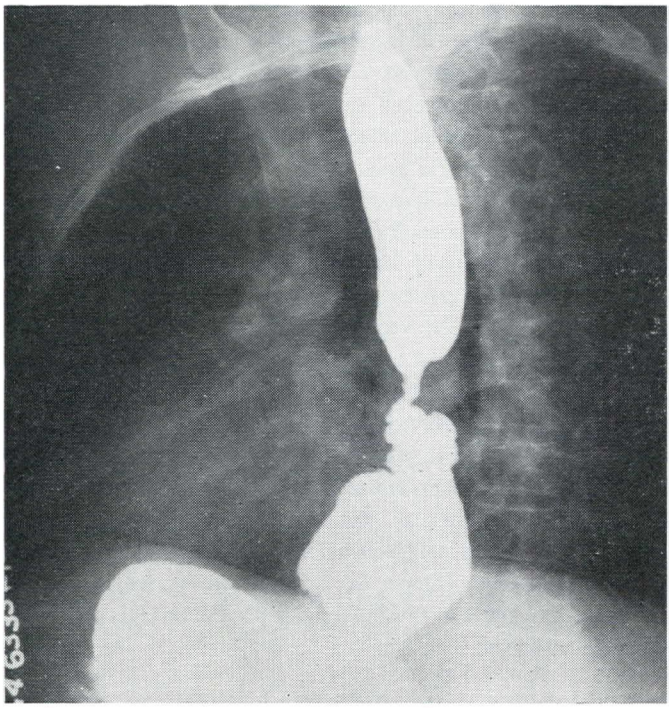

Figure 1a

Preoperative x-rays of Case No. 1. Biopsy taken from the esophagus proximal to the stricture showed inflammation and columnar epithelium.

columnar in type. Stated quite simply, the argument is, that if this altered epithelium is congenital, it plays a part in the genesis of the stricture and should be removed to prevent recurrence. For example, Ellis[11] advocated complete removal of all the esophagus which has columnar lining. If, however, the columnar epithelium is the result of esophagitis, a form of metaplasia, as Hayward[12] maintains, it will respond to reduction of the hernia and termination of the reflux and, therefore, need not be removed. Many surgeons are reluctant, especially at a first operation, to resect the lower esophagus. The recommendation of Hayward in Australia that the stricture be forcibly dilated through a gastrotomy at the time of hernia repair presents an alternative to resection.

The cases that follow have not been treated by a uniform method. In each instance, however, an attempt has been made to reduce the hernia and open the stricture, preserving the esophagus. It is probably too early to draw any final conclusions as to the correct method of treatment, and it may be that no single method is suitable for all cases.

CASE 1: (P. K.) — This 61 year old woman was admitted to this hospital on July 31, 1961. She was transferred from a hospital in another city with a diagnosis of esophageal carcinoma. She noted severe high retrosternal heartburn, beginning in September of 1960, followed gradually by progressive dysphagia and eventually severe boring pain going from the sternum into the back. In April, 1961, she had a laparotomy for stomach cancer, but no sign of cancer was found. Since that time the patient's general condition had deteriorated and she had lost 100 pounds. At the time of admission she was in constant pain and could take only small sips of water and coffee without vomiting.

Her preoperative x-rays on August 3, 1961, showed a hiatus hernia with obstruction of the esophagus (Figure 1). Esophagoscopy showed an inflammed, narrow area in the lower esophagus. Biopsies were taken of this area and showed portions of stratified squamous

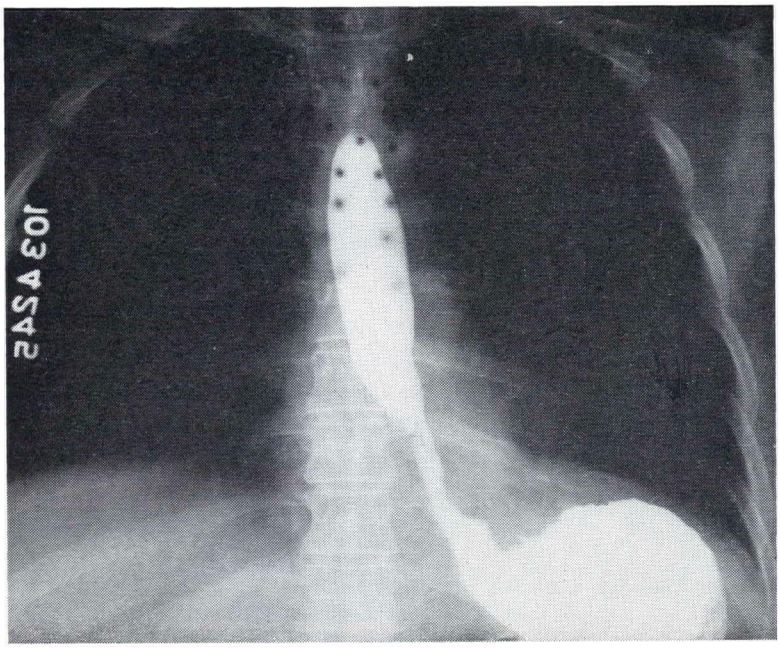

Figure 1b

Following hernia repair and retrograde dilatation of stricture through gastrotomy. The hernia was reducible after the esophagus was mobilized. Swallowing function was fair at first, and later improved.

epithelium and also columnar lined epithelium with inflammation (Figure 2). The patient was operated on August 7, 1961. At operation, a typical hiatus hernia was found. The stricture was palpated through a gastrotomy opening prior to repairing the hernia. The lowest part of the stricture was palpated 3 cms. above the esophagogastric junction. Bakes gall duct dilators were passed and then the stricture was dilated with the finger. Following finger dilatation, the Bailey aortic valve dilator was inserted up into the stricture and the strictured area was forcibly split open (Figure 3). The gastrotomy was then closed and the hernia was repaired. A very satisfactory reduction of the herniated stomach was obtainable with fixation of the phrenoesophageal ligament below the diaphragm. Postoperative x-rays taken on August 22nd, at which time the patient was taking a soft diet, showed a reasonably adequate lower esophageal lumen with some irregularity in the region of the stricture (Figure 1b). The patient was discharged on a regular diet on August 22, 1961. Prior to her discharge, a Number 42 Hurst dilator passed into the stomach without any difficulty.

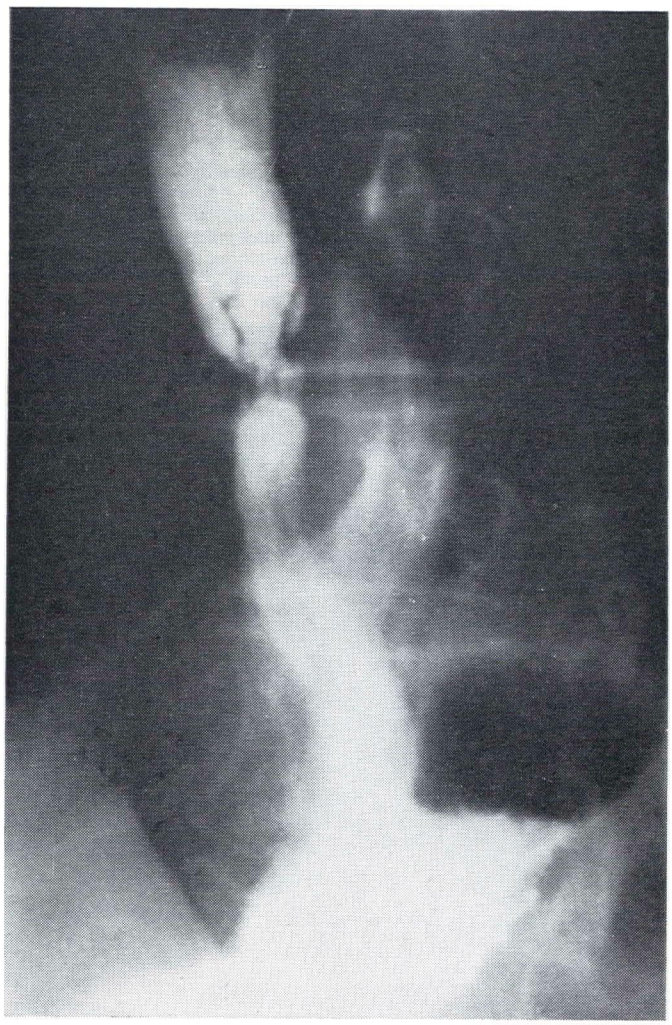

Figure 1c

Irregularity in barium column in the region of the dilated stricture. Note the length of the esophagus below this point.

This patient illustrates the difficulty that exists at times in differentiating esophageal carcinoma from stricture. The x-ray appearance of the two lesions is often similar. Her history, however, is typical of a patient with hiatus hernia who develops gradual stenosis of the lower esophagus over a period of time in response to peptic esophagitis. At the time of her operation, mobilization of the esophagus in the region of the stricture allowed the hiatus hernia to be reduced below the diaphragm. After the hernia repair and the forcible dilatation of the stricture, she was able to eat semisolid foods with ease and solid foods such as meats, if carefully chewed. The initial postoperative x-ray shows an increase in the lumen of the lower esophagus and an adequate reduction of the hiatus hernia. At the site of the stricture, there is a peculiar linear radiation where the contrast material has not remained (Figure 1c). The exact significance of this is unknown, but probably represents residual scar tissue at the site of the dilated stricture. The patient needed periodic dilatations at monthly intervals initially for which a Number 44 Hurst mercury weighted bougie was used. A recent communication from the patient states that she has no swallowing difficulty now and her last dilation was over six months ago.

CASE II: J. L. This 69 year old woman was admitted on March 19, 1961, with a complaint of dysphagia since 1951. She was referred by Dr. Harry Schmidt. Originally, she had difficulty in swallowing solids, but more recently she had difficulty with liquids as well. She had had a cholecystectomy for gallstones two years prior to admission. X-rays showed obstruction in the lower esophagus (Figure 4a). Esophagoscopy done by Dr. Donald Bolstad was said to be normal down to 11½ inches where there was ulceration of the posterior esophageal wall. One or two centimeters below this, there was "granulation tissue" described.

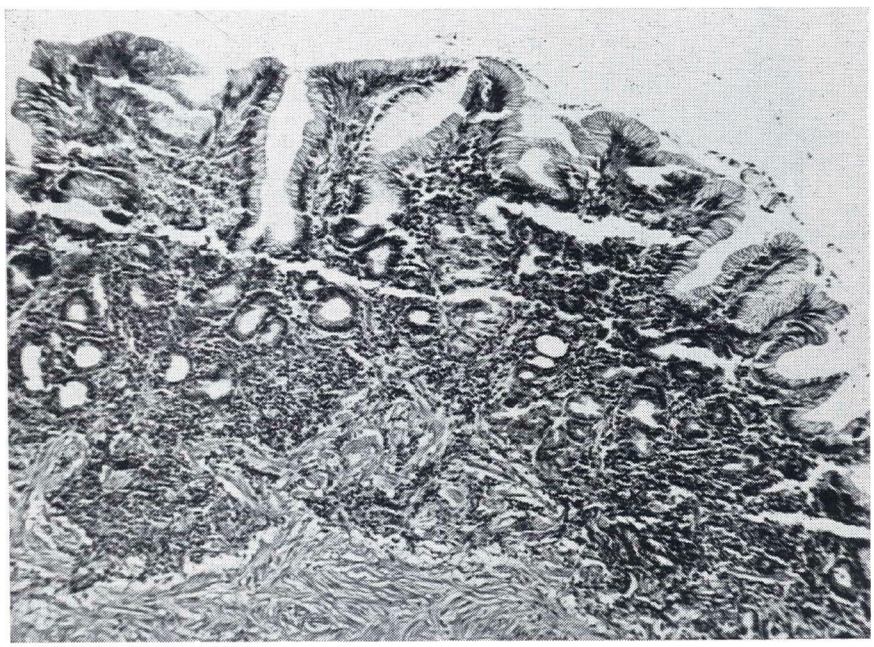

Figure 2

Biopsy taken from esophagus of Case I proximal to stricture shows a columnar lining with mucus glands and inflammatory cells.

Biopsy of this area showed gastric mucosa with gastritis (Figure 5). The patient was operated on August 21, 1961, by Dr. Conrad Lam. He noted "a considerable portion of the stomach above the esophageal hiatus". At first, the finger could not be passed through the stricture, but by dilatation it finally was passed. Then the Bailey aortic valve dilator was inserted into the esophagus and opened. The operative note states: "In spite of the fact that the esophagogastric junction had appeared to be rather high, we were able to get this down to the hiatus without too much trouble".

The patient's immediate postoperative course was excellent and postoperative x-rays taken on September 22, 1961, showed the lower esophageal segment to have a normal caliber (Figure 4b). Prior to discharging the patient from the hospital on September 25, 1961, a Number 46 Hurst bougie was passed into the stomach without any hesitation. The patient ate well and was relatively asymptomatic until she was readmitted on December 6, 1961, with the complaint of vomiting. She was found to have a hemoglobin of 8.9 grams. She expired after a period of observation and symptomatic treatment on January 2, 1962. At autopsy, a large mucinous carcinoma of the ascending colon with extensive peritoneal implantation was found.

The patient received immediate relief from the operation which was identical to the one used in the preceding patient. She did not require dilatation in her postoperative period and was able to eat any kind of food without difficulty. Her readmission with vomiting and a low hemoglobin naturally aroused suspicion of some

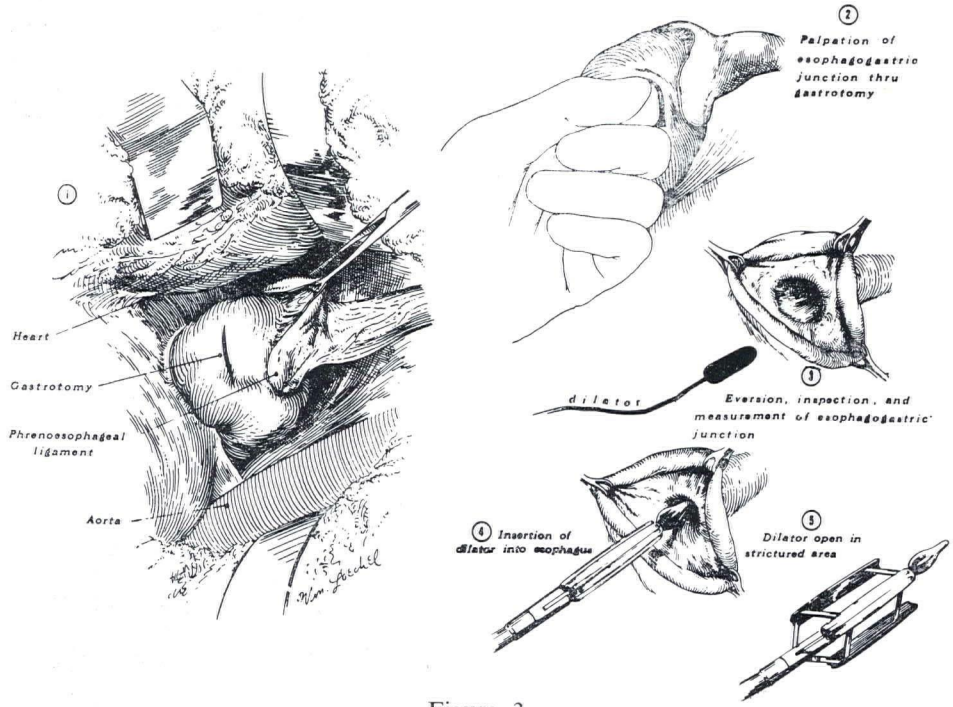

Figure 3

Steps in examining the lower esophagus at the time of hiatus hernia repair. The phrenoesophageal ligament must be completely severed and the gastrotomy made above the diaphragm. Palpation of the esophagogastric junction through a diaphragmatic counter incision yields inaccurate information because the diameter of the junction may be altered by the crura. The normal adult esophagus will admit one finger. To dilate a stricture instruments are passed up through the gastrotomy. The left hand of the operator is placed around the mobilized esophagus as suggested by Hayward to guide the instruments placed in the lumen.

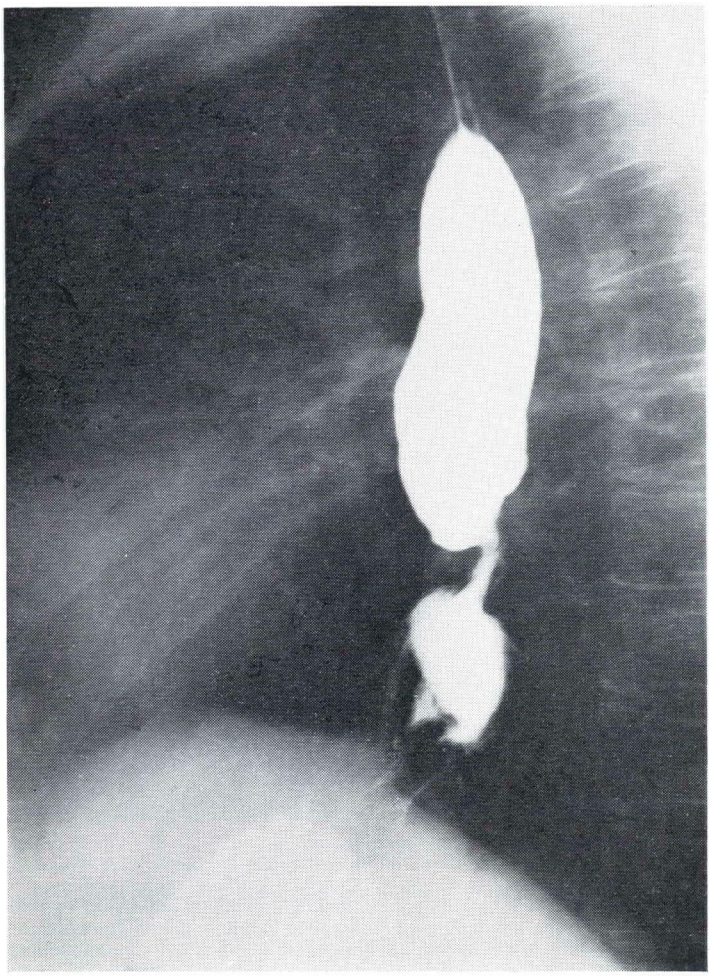

Figure 4a

Preoperative x-ray Case II.

difficulty in the lower esophagus. Esophagoscopy done at this time did show some non-specific inflammatory changes in the lower esophagus. An abdominal mass was palpated, however, and she had ascites and ileus. Examination of the esophagus at the time of autopsy demonstrated no narrowing of the esophagus. In fact, there was some dilation in the region of the previous stricture. The mucosal surface of the esophagus presented a striking appearance in that the proximal third was lined with typical shiny white squamous epithelial surface which terminated in a ragged line (Figure 6). The remaining esophagus had a grossly different lining which changed at the beginning of the stomach to more or less characteristic gastric mucosa. Microscopic examination revealed the proximal segment to consist of typical squamous epithelium. The remainder of the esophagus, however, had no true epithelial surface, but appeared to be uniformly a shallow erosion. Just what the original state of

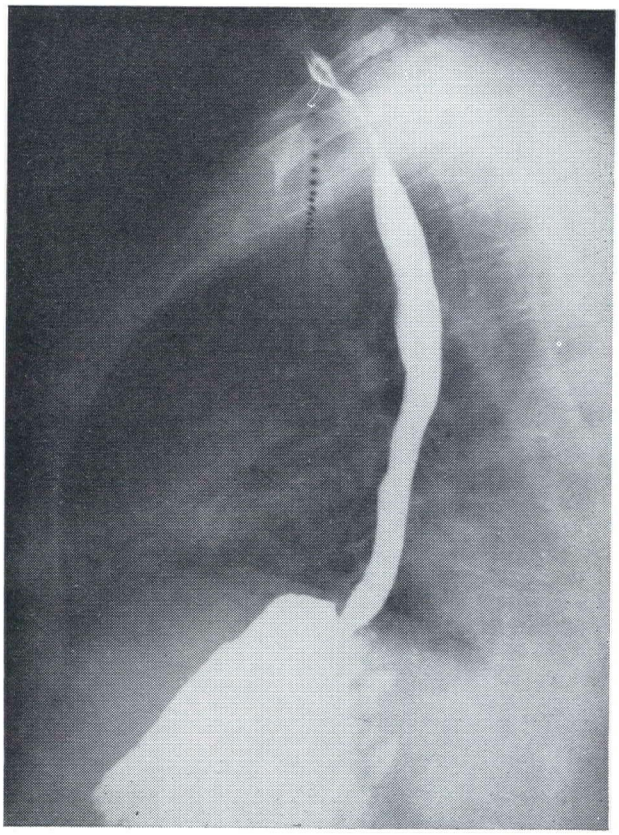

Figure 4b

Postoperative film showing excellent reduction of hernia and a normal looking esophageal lumen.

epithelium in the lower esophagus was is a matter of conjecture. However, the pre-operative biopsy of the esophagus above the stricture showed a columnar epithelium. Presumably, the entire lower esophagus was lined with this type of epithelium at that time. The uniform erosive process could have been the result of postmortem digestion, or more probably was secondary to the reflux which accompanied the patient's terminal ileus.

CASE III. C. B. The patient was a 61 year old man who complained of heartburn, belching and regurgitation of acid juice for many years. Beginning in 1957, he was treated with a succession of antacid medications, and eventually roentgen therapy to the stomach. X-rays taken December 9, 1958, demonstrated a sizeable hiatus hernia and adequate esophageal lumen (Figure 7a). The patient began to have difficulty in swallowing solid foods, such as meat, in 1960 and films taken in August of 1961 showed narrowing of the lower esophagus (Figure 7b). His dysphagia became progressively more severe until August of 1962 when he was unable to eat even soft foods and had to rely completely on liquids for nourishment. Films made at this time demonstrated only a thin column of barium going through the area of the stricture (Figure 7c). An ulcer crater could be seen on the anterior wall of the esophagus in the strictured area.

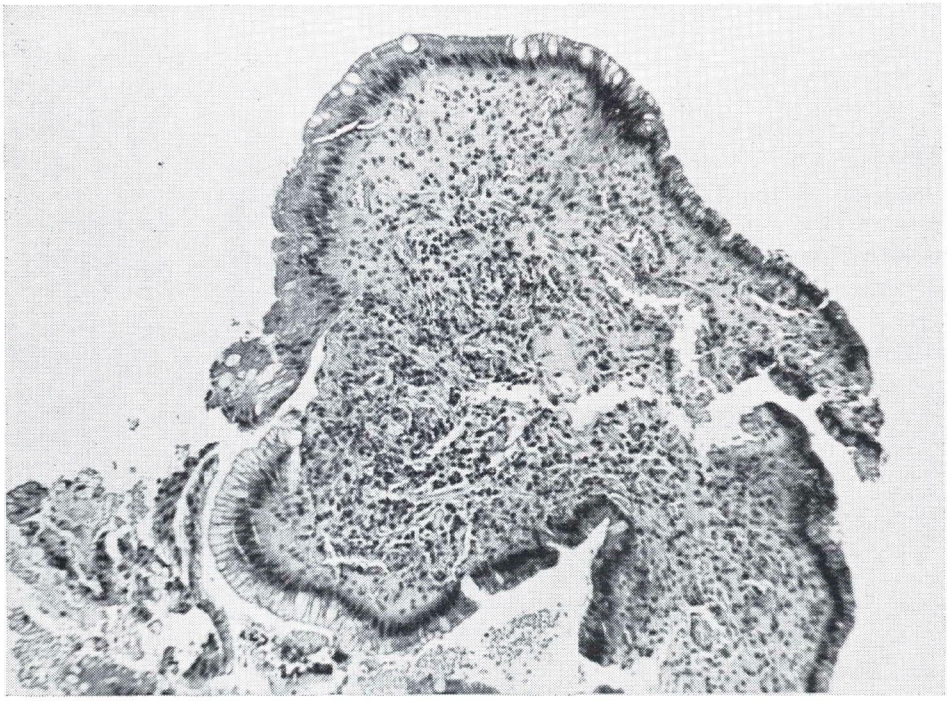

Figure 5

Preoperative biopsy taken above the stricture in an area that looked like granulation tissue showed columnar epithelium with goblet cell formation. This was not obtained from the stomach which was below the stricture.

The patient was referred for operation by Dr. Joseph Rinaldo. On September 24, 1962, the hernia was exposed through a left intercostal incision. Mobilization of the esophagus was difficult because of para-esophageal inflammation in the lower mediastinum (Figure 8). The area of the ulceration had penetrated into the pericardial sac which was opened to dissect the adherent esophagus away from it. When this was completely mobilized, there was a defect in the esophageal wall at the site of the ulcer. The herniated portion of the stomach was opened with a gastrotomy incision, but it was impossible to introduce dilating instruments through the stricture, as it would not admit even the smallest gall duct dilator. An incision was made in the long axis of the esophagus through the stricture. With a scissor and a scalpel, the strictured area was resected, producing a lumen of near normal size and preserving the muscular wall except at the ulcer site. A graft, 0.0014 of an inch in thickness, which had been taken from the skin of the back with a Reese dermatome, was sewn in place as a tubular lining for the reamed out esophagus (Figure 9). This sealed off the defect in the esophageal wall where the ulcer had been mobilized. The hernia was then reduced below the diaphragm and sewn in place with fixation of the phrenoesophageal ligament to the undersurface of the diaphragm. A large rubber tube was placed in the esophagus as an internal splint and brought out through an anterior wall of the stomach as a gastrostomy. Postoperatively the patient was maintained on intravenous feedings for ten days. Oral liquid was begun on the tenth day and the tube was removed on the fourteenth day, following which the patient began taking soft foods. He was able to swallow without difficulty. Postoperative films of the esophagus taken at this time showed an adequate lumen (Figure 10). The patient was discharged from the hospital on a regular diet on October 14th. He was readmitted, however, on November 8th with nausea and vomiting. Physical examination showed him to be dyspneic and tachypneic. His right arm was swollen and a tender, palpable mass was present in the right supraclavicular area. Electrocardiograms showed evidence of pericarditis. He had no pericardial friction rub, however, and x-rays did not show any bulging of the heart shadow to indicate fluid in the pericardium. He was placed on intravenous fluids and antibiotic therapy. Blood culture was reported positive for Staphylococcus aureus, coagulase positive. He was treated as having a probable mediastinitis with septicema.

He expired on November 16, 1962. At autopsy, the pericardium was found to contain purulent material. There was inflammatory occlusion of the right innominate vein. The esophageal lumen was patent and there was no communication between the esophagus and the pericardium.

The corrosive effect of reflux esophagitis over a long period is well demonstrated in the preoperative x-rays which show initially a simple hiatus hernia and subsequently a gradually developing stenosis of the esophagus. The final preoperative x-ray demonstrated an ulcerated area in the region of the stricture. At operation, the base of the ulcer consisted of the pericardium. Mobilization of the esophagus required opening the pericardium and also produced a defect in the wall of the esophagus. The defect in the esophagus was closed by the skin graft and the pericardial defect was closed by interrupted sutures. In retrospect, it would have been better to have enlarged the

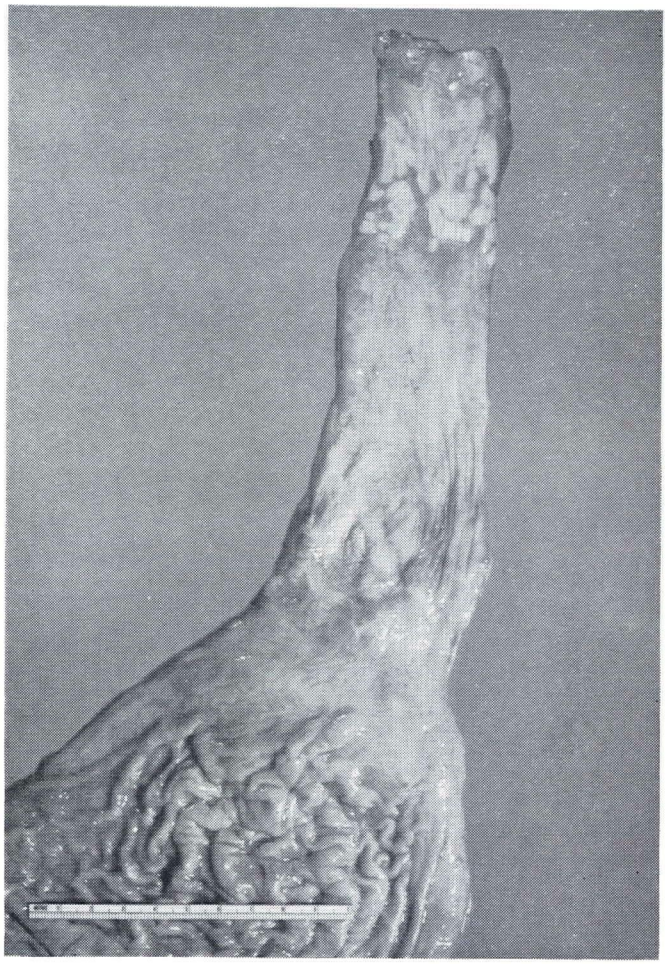

Figure 6

Postmortem appearance of esophagus Case II. The squamous lining terminates high up in the esophagus.

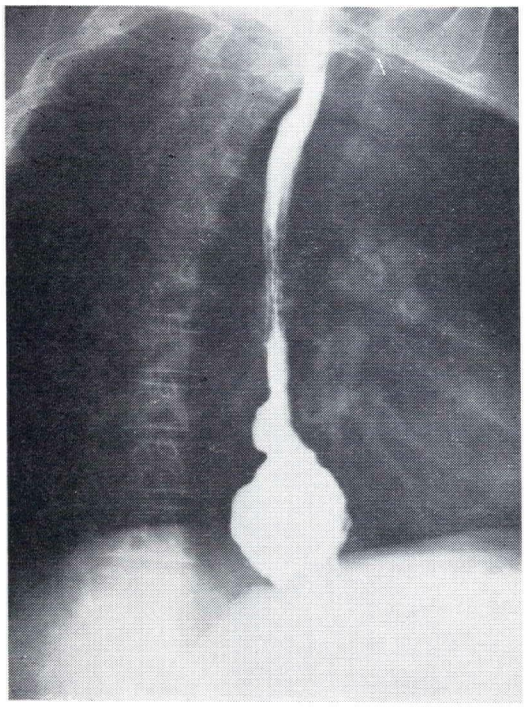

Figure 7a

Case III x-ray taken in December, 1958, shows a hiatus hernia with no narrowing of the esophagael lumen.

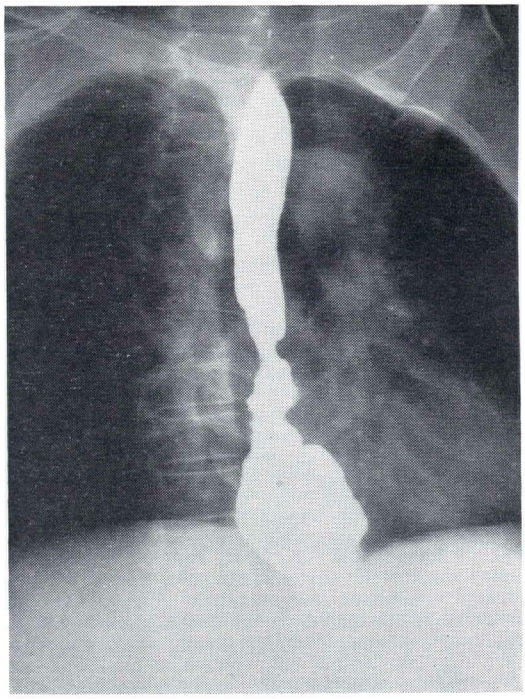

Figure 7b

X-ray taken in August, 1961, shows narrowing of the lower esophagus. The patient had begun to have dysphagia at this time.

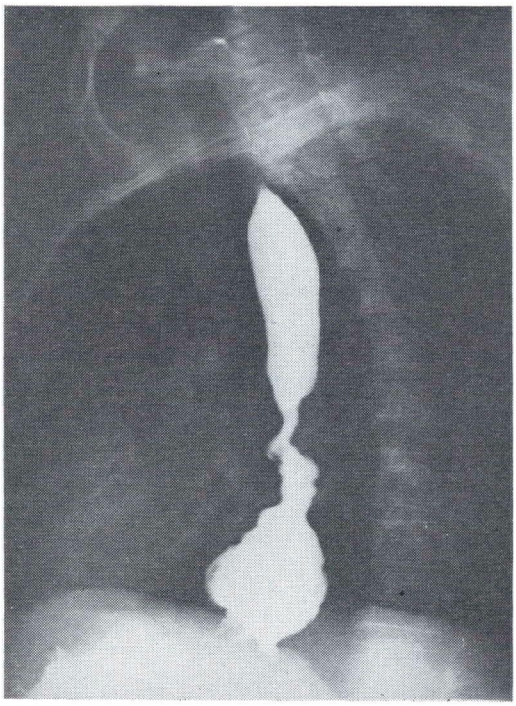

Figure 7c

X-ray taken in August, 1962, shows advanced narrowing of the lower esophagus with an ulcer crater on the anterior wall of the stricture. The patient's dysphagia at this time was severe.

pericardial opening, or otherwise provided for drainage of the pericardium. Contamination of the pericardial sac which led to the fatal pericarditis, probably occurred at the time of operation rather than from postoperative leakage from the esophagus. Roentgen examination of the esophagus in the postoperative period revealed no fistula formation. The postmortem appearance of the esophagus was very close to that of the first case. It showed the same wax-like squamous lining of the upper esophagus. The lower esophagus, however, was composed of a columnar lining without parietal or chief cells. There was no permanent growth of the skin graft. This area was covered with a simple columnar lining with erosion. The graft had not acted as a permanent covering, therefore, but evidently was effective as a patch for the defect in the esophageal wall caused by the ulcer. Th esophageal lumen was quite adequate.

CASE IV: F. F. This 65 year old man presented with progressive dysphagia over the previous nine months, being admitted on October 24, 1962. He had maintained his nutrition by eating blended food and baby food. Prior to that time, the patient had had symptoms of hiatus hernia for several years and knew that he had a "hernia through the diaphragm". X-rays on October 25, 1962, showed a hiatus hernia with reflux and a persistent area of narrowing in the lower esophagus, measuring 1.5 cm. in length, and 6 mm. in width. No definite ulceration was identified (Figure 11). Dr. Rinaldo esophagoscoped the patient and noted that "at 41.5 cm. the esophageal lumen narrowed so that it was impossible to pass the esophagoscope past this area". The mucosa in the area of narrowing was erythematous and a biopsy showed both squamous and columnar epithelium with evidence of inflammation. The patient was operated on November 2, 1962. A hiatus hernia was identified with

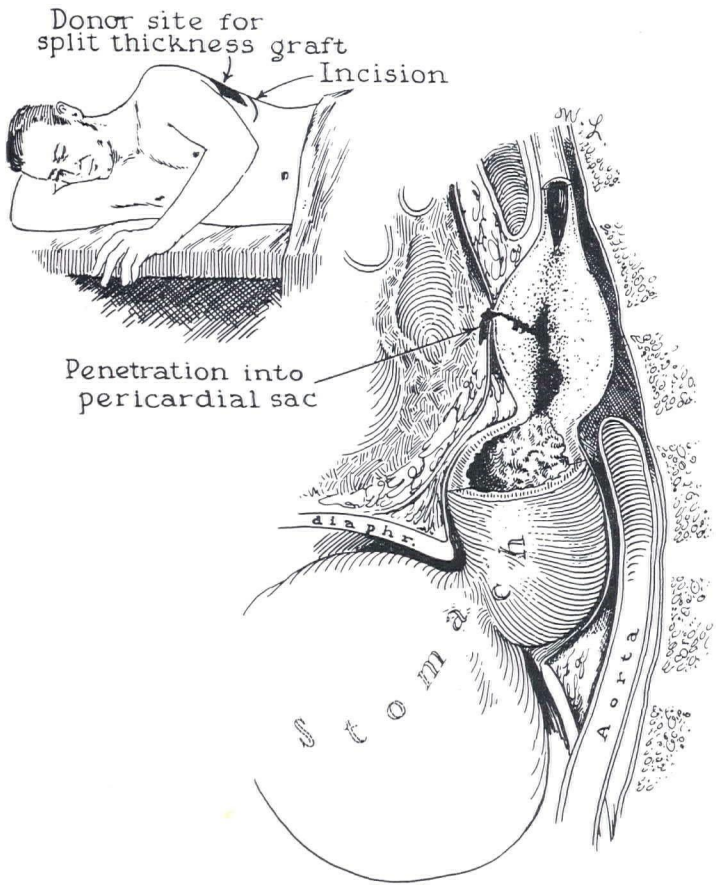

Figure 8

Drawing of the situation found at operation of Case III. The wall of the esophagus in the area of the stricture was thickened and the ulceration penetrated to the pericardium.

fixation of the esophagus in the mediastinum by severe inflammation in the region of the stricture. The strictured lining was resected submucosally through a linear esophagotomy by scalpel and scissors. The muscular wall of the esophagus was quite thickened in this area. The resected area was covered internally with a split thickness skin graft taken from the back. The esophageal wall was sutured, closed around a tube brought out through the anterior wall of the stomach via a gastrotomy opening. The reduction of the hernia was quite satisfactory. The patient began to take a semisolid diet on November 11 at the time the tube was removed. He ate without difficulty at first, but later required dilatation. An attempt was made to ascertain the state of the graft by esophagoscopy on November 21, but the procedure was not completed because of what was thought to be undue resistance at the level of the cricopharyngeal muscle. Plummer dilators were passed up to Number 41 prior to the patient's discharge from the hospital and in the Outpatient Clinic he subsequently was dilated easily with a 45. He ate his Christmas dinner without any trouble and on January 4, 1963, a Number 45 Plummer dilator passed through the lower esophagus without resistance.

Biopsies of this patient's esophageal mucosa at the time of his operation showed a mixture of squamous and columnar epithelium in the zone just above the stricture (Figure 12). We do not know the exact upward extent of the columnar epithelium

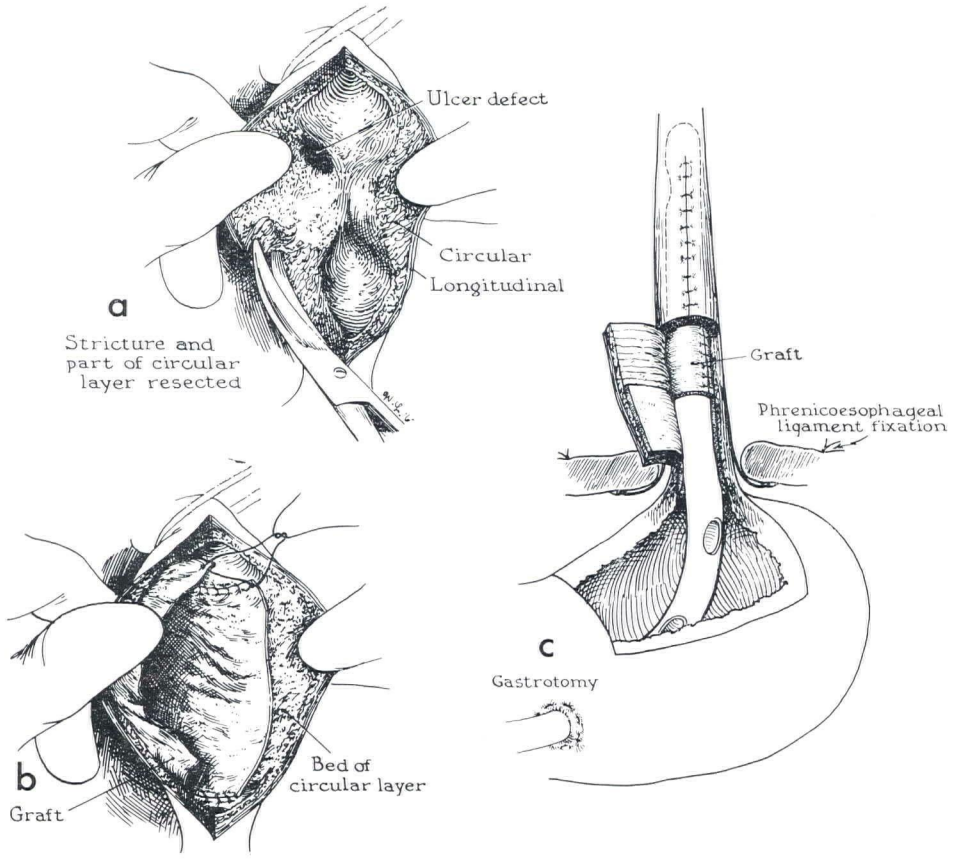

Figure 9a

The stricture was resected preserving the muscular wall of the esophagus except at the site of the ulcer.

Figure 9b

The interior of the esophagus was relined with a split thickness skin graft taken as shown in the inset of Figure 8.

Figure 9c

The esophagus was reconstructed by longitudinal suture of the muscular coats. A large bore rubber tube was used as a stent. The tube was brought out as a gastrostomy through the anterior abdominal wall.

as a biopsy was not taken proximal to the one shown in the figure. Mobilization of the esophagus made it possible to place the stomach below the diaphragm with ease. The fate of the skin graft is not certain. Probably it did not take, as we have clinical evidence of decrease in the size of the lumen. It is possible that actual resection of the strictured mucosa is not wise, as it produces more of a raw area to be covered than a Hayward dilatation which splits the mucosa. We anticipate, however, that the end result on this patient will be a good one. With the excellent reduction of his hernia, he has not had symptoms of gastric reflux since the operation.

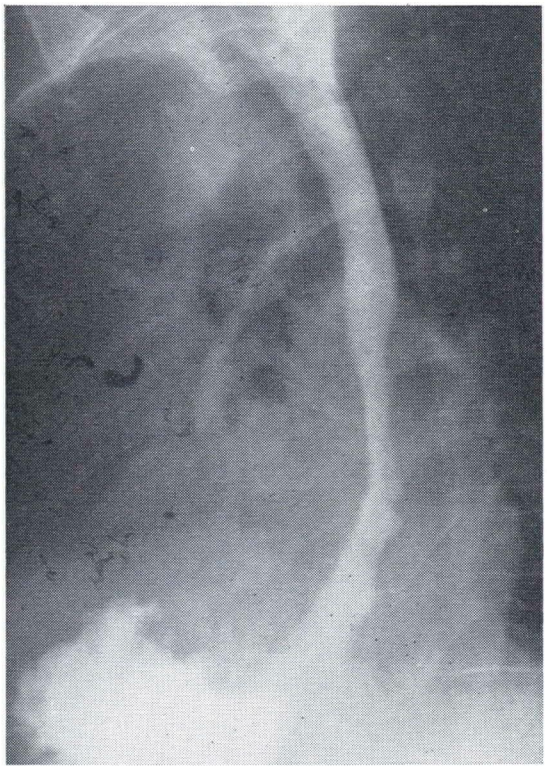

Figure 10

Postoperative x-rays Case III with gastrografin showing an adequate esophageal lumen with some slight irregularity at the area of the resected stricture.

CASE V: C. M. This patient was a 50 year old man who was admitted on December 9, 1962, to Dr. William Haubrich's service with symptoms of severe dysphagia. He had had typical symptoms of hiatus hernia and gastric reflux beginning six years prior to admission. He had had the hernia repaired in another hospital in January, 1961. He had never had complete relief of symptoms following this, however, and he began to have dysphagia in August, 1961, following a bout of hematemesis brought on by ingestion of alcohol. Subsequent to this, he had to have his esophagus dilated three times. He continued to have heartburn, especially at night and took oral antacids continuously. X-rays taken on December 12, 1962, showed a stenosing lesion of the esophagus with a questionable shelf-like deformity suggesting carcinoma (Figure 13a). At esophagoscopy an inflammed area was identified which could not be passed with the esophagoscope. A biopsy was taken at the beginning of the stenosed area which showed squamous epithelium and no cancer. At operation, on December 19, 1962, a very inflammed lower esophagus was found with fixation in the mediastinum. There was a recurrent hernia through the esophageal hiatus. The esophagogastric junction was found 4 cms. above the margin of the hiatus. The stricture was 3½ centimeters above the esophagogastric junction. The strictured area was examined through a gastrotomy opening and although a finger could not traverse the narrowing, it would admit a gall duct dilator, measuring 11 millimeters. Mobilization of the esophagus in the mediastinum allowed reduction of the stomach beneath the diaphragm. However, in dissecting the esophagus away from the pericardium, a defect in his esophageal wall was produced. It appeared that an attempt to close the defect would compromise the reduction of the hernia by shortening the esophagus, so a resection of the lower esophagus and the body and fundus of the stomach was carried out. The vagus nerves were cut, of course, and a pyloromyotomy was done. The gastric antrum was anastomosed to the esophagus. The esophagus was vented with a small gastrostomy tube. The patient's postoperative course was uneventful and he began eating on December 27, 1962. He was discharged on a first month ulcer diet on the 15th postoperative day.

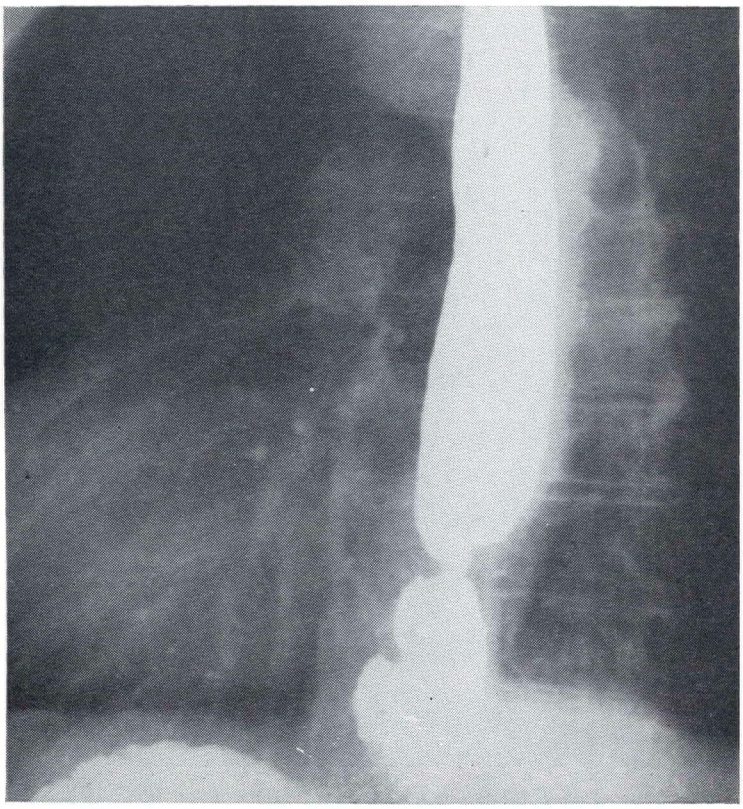

Figure 11

Case IV. The preoperative x-ray shows a stricture in the lower esophagus above a hiatus hernia.

We do not know whether the patient had had some stricture and periesophagitis prior to the first operation. It is possible that failure to completely dissect out the esophagus was a factor in not obtaining adequate reduction and led to recurrence. The resection at the second operation was made necessary not because of the inability to reduce the hernia but because of the defect in the esophagael wall produced at the site of the ulcer. This ulcer was in the anterior wall of the stricture and its base was formed by the pericardial surface. The size of the esophageal defect was a factor in the decision to resect the esophagus. The patient's erratic personal habits, in addition, called for an operation requiring a minimum of postoperative care.

The resected specimen looks shorter than its actual length in the body, as the esophageal muscle contracts after excision (Figure 14). (In the esophageal myotomy operation for cardiospasm, we have removed strips of the muscle layer and seen them curl up into a roll after excision). The proximal part of the esophagus has a normal looking squamous lining. This is succeeded by columnar epithelium in the region of the stricture which continues down to the actual stomach mucosa (Figure 15).

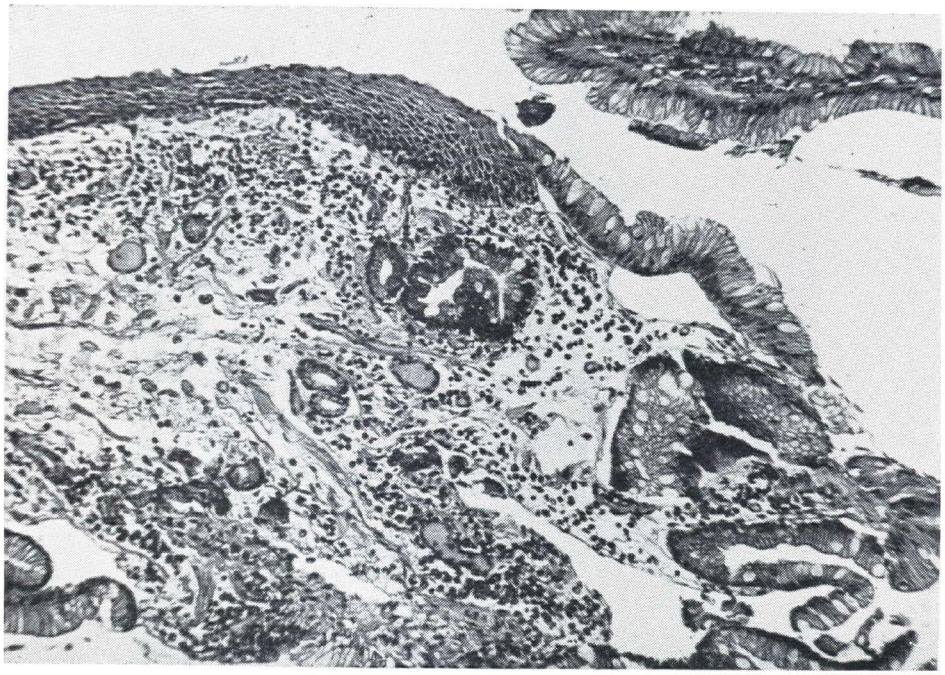

Figure 12

Biopsy taken at operation from the esophagus proximal to the stricture in Case IV. Both columnar and squamous cells can be identified. The columnar epithelium exhibits goblet cell formation. This was not at the esophagogastric junction but about 8 cm. above it.

Discussion

Mobilization of the esophagus by mediastinal dissection in the region of the stricture permits reduction of the hernia. The inflammatory process around the stricture anchors the esophagus in the mediastinum with thick adhesions that cannot be broken with blunt dissection, but must be cut through with a sharp instrument. When the esophagus is dissected out of the mediastinum and the stomach is reduced, postoperative x-rays show an apparent increase in esophageal length. A surgical approach which does not allow dissection in the mediastinum will not permit reduction of the hernia. For example, Olsen and Harrington,[7] attempting to repair hernias through a laparotomy incision, found that they could not reduce 10 per cent of the hernias and consigned them to a category labeled "short esophagus", as uncorrectable. Collis,[14] of Birmingham, England, has devised an operation for "short esophagus" in which a tube is made of the greater curvature of the stomach when the stomach cannot be reduced below the diaphragm. He has performed the operation 32 times between 1955 and 1960. Collis' method of hernia repair consists of bringing the stomach through the most posterior part of the hiatus which is also the most dependent part. He also carefully avoids any dissection in the mediastinum. The cases reported here have been approached with the rationale that removal of the causative factor, gastric reflux, by repairing the hernia is necessary to cure the stricture.

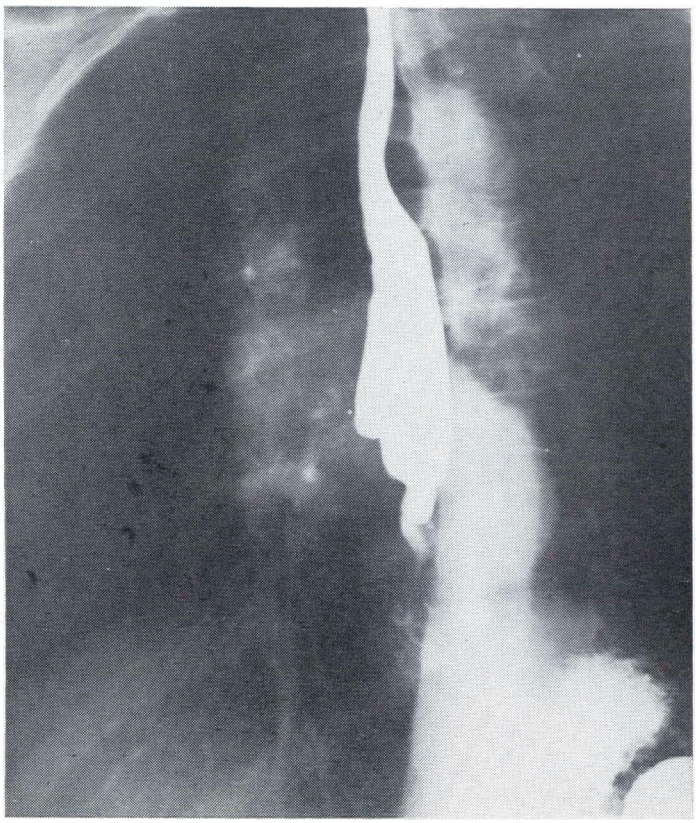

Figure 13a

Preoperative x-ray in Case V shows shelf-like defect about the narrowed area in the lower esophagus. This deformity aroused suspicion of cancer, although the patient had a long history of difficulty with a hiatus hernia.

The question has been raised whether reduction of the hernia will cure the patient with an esophagus already strictured from severe inflammation. Allison and Johnstone[9] tell of a 48 year old patient who required a second operation because of persistent symptoms after a hernia repair and stricture dilatation. At the second operation they found the hernia well reduced, but proceeded with resection of the esophagus. They do not indicate the time period between the two procedures. Hayward maintains that patients do have symptoms for a time following hernia repair and sometimes need dilatations for several months, but eventually become symptom free. The experience with the patient described in Case I would support this view. Although she could not get along without dilatations in the immediate postoperative period, none have been required for over six months.

Is the columnar epithelium in the esophagus harmful? Barrett[13] is of the belief that the columnar lined esophagus is a congenital malformation. The embryonic

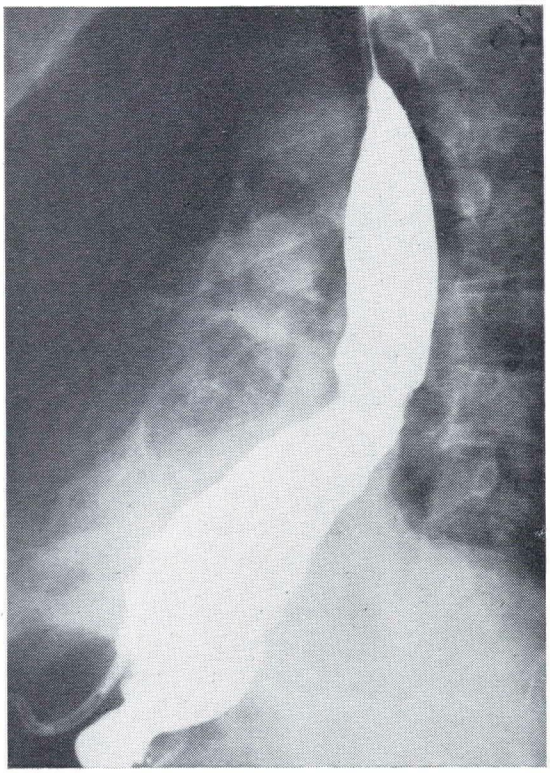

Figure 13b

Postoperative films after esophagogastrectomy. A small gastrostomy tube is still in place.

esophagus is lined with columnar cells which change to squamous by a process beginning in the mid esophagus and going both up and down. There are no published experiments showing that the esophageal mucosa can change from squamous to columnar under the influence of acid secretions. Dr. Michael Brennan, however, has called to my attention some experiments done by Lawrence[15] on the hamster, where the epithelium of the cheek pouch can be changed from squamous to ciliated columnar by the topical application of vitamin A pellets. The columnar mucosa seen in the esophagus lacks parietal and chief cells and is not typically gastric. When seen out of context, the microscopist might identify it as belonging in the intestine, particularly when goblet cell formation is present (Figure 12). These cells are usually attributed to inflammation in a columnar lined surface and there is some reason to suppose that the columnar cells in the esophagus represent a process of metaplasia.

The visceral attachment of the phrenoesophageal ligament is an alternate landmark to the esophagogastric junction especially valuable when the sharp transition is absent from the mucosa. The attachment can be quickly assessed at operation and used as a guide to the reduction of the hernia. In each of the cases reported here

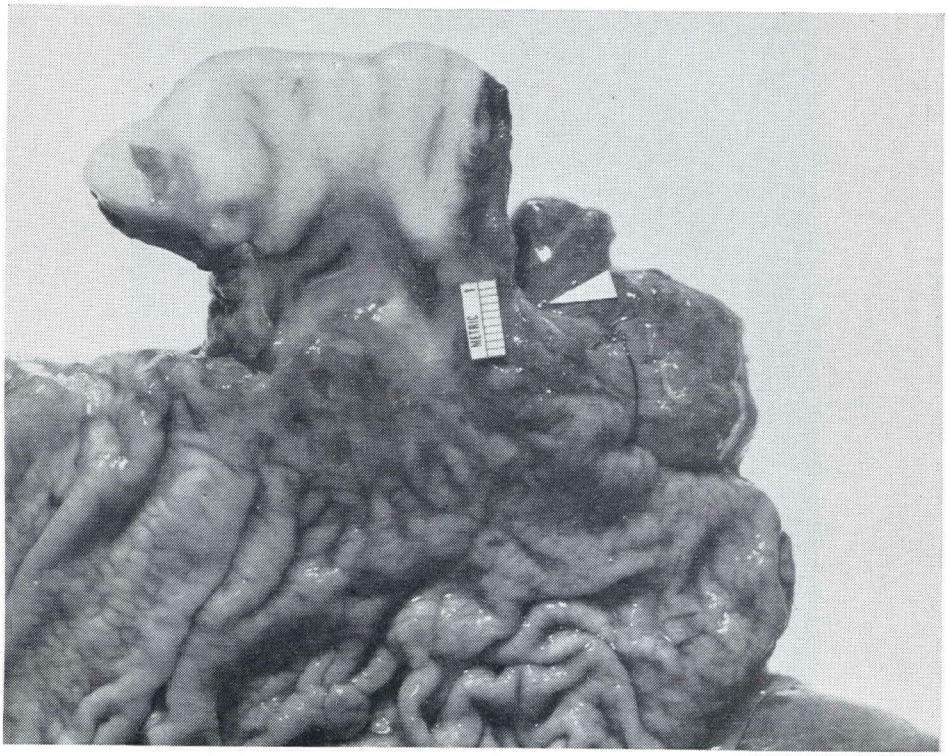

Figure 14

Resected specimen of esophagus and stomach, Case V. The specimen is opened through the ulcer defect. The small white arrow points to the attachment of the phrenoesophageal ligament. The lowermost extent of squamous epithelium is above the stricture and the intervening area is covered with columnar epithelium. There is considerable foreshortening of the resected specimen but, nevertheless, the strictured area is much shorter than it appears on the x-ray.

the opening of the esophagus into the stomach was larger than normal. In measuring the size of the esophageal lumen at the esophagogastric junction in a sizeable number of patients undergoing hiatal hernia repair, the adult esophagus with a competent sphincter admits one finger snugly, not two (Figure 3). When reflux is present, the lumen is often larger and will sometimes admit three fingers easily. The size of the lumen may not be the reason for the reflux, but it has something to do with it. It is true, as Hayward has pointed out, that stricture, if the so-called lower esophageal ring is excluded, occurs not at the esophagogastric junction but above it. The jet of gastric juice may induce a muscular contraction of the esophagus which limits the upward flow of the reflux and also concentrates it on this one area of the mucosa. Barrett has been impressed with the thickness of the muscular wall of the esophagus at the strictured area, instead of above it as might be expected with an obstruction. Perhaps the muscular hypertrophy takes place before the obstruction is complete.

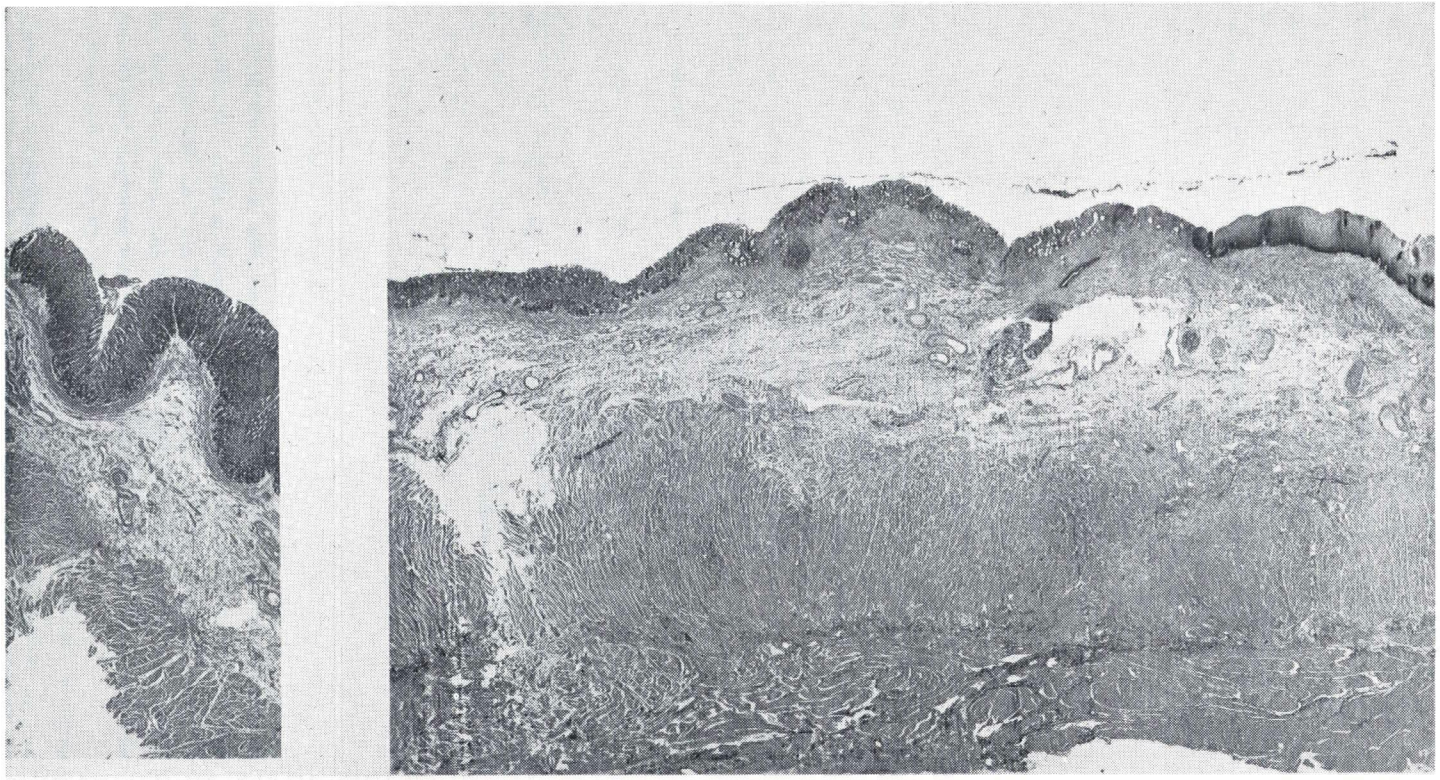

Figure 15

Microscopic section (Hematoxyl and Eosin X 6) of the resected esophagus and proximal stomach, shown in Figure 4.

a. Cardia of stomach showing typical deep gastric mucosa with rugal folds.

b. Between the actual stomach and the lower extent of squamous epithelium, as seen to the far right of the section, the esophagus is lined with a low columnar epithelium with small mucus glands. There is an increased thickness of the muscle layers in this area.

SUMMARY

Five patients are presented with stricture of the esophagus secondary to hiatus hernia and reflux of gastric juice. In each of these patients columnar epithelium lined the lower esophagus. The columnar lining intervened between the stricture and the stomach but was also found above the stricture. It was possible to reduce the hernia if the strictured esophagus was cut away from its mediastinal attachments, the esophagus then being long enough to reach below the diaphragm. The question of whether the columnar cells in the lower esophagus are metaplastic remains open. However, it seems that a good clinical result can be obtained by hernia reduction and stricture dilatation without resecting the esophagus.

ADDENDUM

Since submission of the manuscript, continuous microscopic sections have been made of the esophagus shown in Figure 6 (Case II). These sections cover the entire length of the esophagus. The mucosal surface between the end of the squamous epithelium and the beginning of the stomach consists mostly of columnar epithelium although there are some areas of erosion where no definite epithelium can be identified.

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