The diagnosis and treatment of peptic oesophagitis

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SYNOPSIS An account is given of the treatment of peptic oesophagitis, in which the importance of repairing a hernia which is producing peptic ulceration is emphasized. Mobilization of the oesophagus must be carried out as far up as is necessary for the hernia to reduce without tension.

When there is a firm stricture which will not respond to treatment, associated with shortening of the oesophagus, resection and interposing a loop of jejunum is the operation of choice which in this series has been performed without mortality.

The paper summarizes the author's experience of the treatment of peptic oesophagitis encountered amongst 1,500 patients with hiatal herniae seen in Leeds in the past 20 years. Case reports illustrate aspects of the diagnosis and medical and surgical treatment of peptic oesophagitis and stricture. The indications and limitations of dilatation of strictures either blind or under direct vision are detailed and the method of hernial repair described. Once a firm stricture has developed with gross oesophageal shortening, resection and the interposition of a loop of jejunum is the operation of choice.

During the past 20 years more than 1,500 patients have attended the Department of Thoracic Surgery in Leeds with herniae through the oesophageal hiatus of the diaphragm. The diagnosis has been confirmed in every patient radiologically and by oesophagoscopy. The examination of a patient with a hiatal hernia is not complete until oesophagoscopy has been performed, being the only way of making absolutely sure that no peptic erosion of the oesophageal mucosa is taking place. It is not uncommon to see a fairly normal looking oesophagus on the barium swallow and an intense oesophagitis at oesophagoscopy. The oesophagitis may be caused in two ways, either by the reflux of gastric contents from the stomach or by the secretion of pepsin and acid locally in the oesophagus if it is lined by gastric mucosa. Fifty patients in this series presented with a secondary anaemia, caused by bleeding from peptic oesophagitis, which had not been suspected radiologically and was only discovered by an oesophagoscopic examination. It is of extreme importance to know if there is peptic digestion of the oesophageal mucosa, so that the hernia may be repaired before the oesophageal wall becomes irretrievably damaged. The presence of oesophagitis

is the main indication for advising a patient to undergo operative treatment, the other indication being the failure of medical treatment. In this series, of over 1,500 patients, all of whom were referred by other consultants for a surgical opinion, it was found necessary to operate on just over one-third of the total number after the patients had been fully investigated.

PEPTIC OESOPHAGITIS

Reduction of a hiatal hernia with restoration of the normal anatomy is not such a difficult technical procedure. However, when there has been gross destruction of the oesophageal wall by peptic ulceration there remains no real answer to the problem, other than by resection and reconstruction of the oesophagus with intestine. This often proves a long and tedious operation which cannot always be guaranteed to cure the patient's symptoms.

CLINICAL PICTURE OF OESOPHAGITIS Oesophagitis may be suspected from the history if the patient complains of heartburn, of regurgitation of acid, or of 'bile' which may produce a burning sensation behind the sternum and may rise up as high as the pharynx. Sometimes patients experience a soreness low down in the oesophagus immediately after swallowing which goes as the food passes into the stomach. Many complain of pain high in the epigastric region which is related to posture, occurring while they are lying down in bed, or when they bend down to fasten their shoes. It can often be relieved by sitting upright, and taking a hot drink or an alkaline mixture. Pain in the back is a

FIG. 1a

FIG. 1b

FIG. 1c

FIG. 1d

FIG. 1. Barium swallow obtained in a woman aged 41 years (Case 1) illustrating a hiatal hernia with a gastric-lined oesophagus (Fig. 1a). A peptic ulcer arising in this mucosa had perforated into the posterior mediastinum and produced severe back pain.

Fig. 1b illustrates a bolus obstruction in a peptic oesophageal stricture which had produced total aphagia. Oesophageal spasm simulating retrosternal ischaemic pain in two elderly men aged 73 (Fig. 1c) and 62 (Fig. 1d).

late symptom and denotes that the inflammatory process has spread outside the oesophageal wall and is now involving the mediastinal tissues, when sudden death may result from an ulcer perforating through the aortic wall.

Case 1 Illustrating symptoms produced by gastric-lined oesophagus A woman aged 41 years had had indigestive symptoms all her life. During the last two years she had suffered from severe substernal pain radiating into the back between the shoulder blades, which was particularly severe at night. She had also developed dysphagia and vomited all hard foods.

A barium swallow and meal showed a hiatal hernia and a long segment of oesophagus lined with gastric mucosa above which was an ulcer producing stenosis (Fig. 1a). Oesophagoscopy examination proved these findings, and biopsies from the ulcer and the mucosa just distal to it showed that it was arising in gastric mucosa.

November 1957. Operation. Extensive mobilization of the whole oesophagus in order to reduce the hernia. Bilateral vagotomy and pyloromyotomy performed. The ulcer healed. The patient is taking a normal diet and has no dysphagia. No dilatation of the stricture has been necessary.

Intermittent dysphagia is not uncommon when peptic ulceration is present, due to the lumen becoming narrower which in turn reduces the reflux. Dysphagia may also be due to hard pieces of food temporarily blocking the lumen. Bolus obstruction causes aphagia and presents with severe substernal pain and vomiting (Fig. 1b). It may sometimes be relieved with a fizzy drink such as soda water but usually requires an oesophagoscopy examination and dilatation of the stricture.

Sometimes the symptoms are first experienced when patients are lying in a recumbent position after an operation. Watkinson (1960) has had several patients develop severe heartburn while lying flat in bed after coronary infarction. Recurrent infarction was suspected, until the head of the bed was raised, and then all their symptoms disappeared, proving that their more recent pain was of oesophageal origin.

The distinction of cardiac from oesophageal pain may be difficult; the one mimics the other very closely and both may be present in the same individual, for they occur in the same age group (Fig. 1c).

Oesophageal spasm has not infrequently been treated as cardiac infarction. A barium swallow performed during an attack of pain will clinch the diagnosis of spasm in a patient who has doubtful electrocardiographic changes of infarction (Fig. 1d).

RADIOLOGICAL APPEARANCES OF OESOPHAGITIS A hiatal hernia can nearly always be demonstrated by filling the stomach with barium and then tilting the patient into the Trendelenberg position (Fig. 2a).

Reflux of barium from the stomach into the oesophagus takes place just at the end of inspiration. The peptic inflammation caused by reflux of gastric contents into the oesophagus has been aptly described by Barrett as 'reflux oesophagitis'.

While the patient is standing, barium may pass down the oesophageal lumen without demonstrating any abnormality. Later a granular appearance of the mucosa may be seen near to the cardia. This progresses into a slight narrowing and then the oesophageal wall becomes thickened. The lumen becomes narrower and a stenosis occurs which remains fixed and does not open up on swallowing more barium. An ulcer crater may sometimes be seen which denotes that it has been formed in a portion of oesophagus lined by gastric mucosa (Barrett, 1950) (Fig. 1a). Other ulcers may be present in the stomach and duodenum. Davidson (1958) has described an ulcer occurring in the hernia as it crosses the sharp edge of the hiatus of the diaphragm. He believes that it may be due to trauma caused by food passing over this edge, because it heals spontaneously after the hernia has been reduced.

Case 2 Illustrating development of a gastric ulcer in a fixed hiatal hernia A woman, aged 61 years in 1925, complained of epigastric pain and vomiting after meals. At laparotomy by Lord Moynihan, a hiatal hernia was found and repaired.

1931. Her symptoms had persisted so a second repair of the hiatal hernia was performed through the chest by another surgeon.

1944. She became much worse, having severe pain and haematemesis. A barium meal showed a large gastric ulcer at the site where the hiatal hernia crosses the diaphragm. At laparotomy there were too many adhesions to free the stomach, so the left gastric vessels were ligated. She had no further haematemesis but she was never free of indigestion and continued to have pain after evening meals, with recurrent attacks of vomiting.

1955. She was transferred to the Department of Thoracic Surgery. A barium meal showed a large hiatal hernia and a large gastric ulcer in the middle of the lesser curvature of the stomach spreading around the whole body of the stomach at the level of diaphragmatic constriction (Fig. 2b).

June 1955. Operation. Hiatal hernia repaired; sleeve resection of stomach and pyloroplasty performed. The ulcer crater measured 2 cm. in diameter and was of a simple type. She has remained well and free of symptoms since. It is possible that the ulcer might have healed after reduction of the hernia alone.

If the hernial sac is very large it may contain the whole stomach. The stomach then rotates so that its

greater curve lies uppermost. Occasionally, the sac extends into the right side of the chest (Fig. 2c).

The presence of a hiatal hernia seems to make the recognition of a gastric neoplasm difficult, particularly when the neoplasm occurs in the herniated pouch of stomach.

Case 3 Illustrating development of a gastric neoplasm in a hernia A man aged 59 had had flatulence all his life, heartburn for many years, and dysphagia for the past four months.

He had attended the physicians for many years and was known to have a large hiatal hernia which had not altered radiologically. He was transferred to the Department of Thoracic Surgery when he began to lose weight and could only swallow liquids.

February 1957. He was found to have developed an adenocarcinoma in the hiatal hernia so a radical oesophago-gastrectomy was performed with jejuno-oesophageal anastomosis. He has remained well since.

OESOPHAGOSCOPIC APPEARANCES The junction of the oesophageal with the gastric mucosa will in all probability be lying above and so proximal to the pinchcock action of the diaphragm. This junction is rarely on the same horizontal plane all around the lumen, but gastric mucosa may extend up a little and so to a slightly higher level at each side.

At the onset of oesophagitis the junction is well defined even though the oesophageal mucosa is reddened. On closer examination the reddened area will reveal small vessels coursing across its surface. Later the junction of the two mucosae becomes covered with a grey exudate. If the exudate is wiped away with a gauze swab it reveals a bleeding granulating surface of a superficial ulcer. These ulcers usually encircle the oesophageal lumen and then spread up the oesophageal mucosa in a digital fashion, so that when the whole ulcerating area is viewed from above through the oesophagoscope it looks star-shaped. Distally the ulceration surrounds the whole lumen, whereas proximally it extends up the lumen in finger formation (Fig. 2d). The destructive process will have now reached the submucosa where it creates an intense fibrous reaction forming strictures. This acts as a protecting mechanism, preventing the gastric juices reaching higher up the oesophagus and destroying yet another segment. The oesophagus becomes stenosed by a thickened mass of fibrous tissue and adherent to the mediastinum with vascular adhesions.

If the ulcer has been formed in gastric mucosa lining the oesophagus it behaves like a gastric ulcer in the stomach and penetrates deeply into the mediastinum. It is this type of ulcer which may perforate into other surrounding structures. I have already mentioned one which eroded the aortic wall

FIG. 2a

FIG. 2b

FIG. 2c

FIG. 2d

FIG. 2. Reflux of barium from stomach to hiatal hernia induced by tipping the patient into the Trendelenburg position (Fig. 2a).

(Fig. 2b) (Case 2) In this woman, aged 61 years with a large para-oesophageal hernia, a gastric ulcer has developed at the level of the hiatus (Davidson's ulcer). Fig. 2c illustrates a large hernial sac containing the stomach, greater curvature uppermost, which is encroaching on the right lung field.

Fig. 2d is a drawing of chronic oesophageal peptic ulcer which was successfully resected in a 78-year-old man showing digitate extension of the ulcer into the oesophageal mucosa.

and more recently in another case it had perforated into the right lower lobe.

Peptic oesophagitis produces a similar lesion in children as in adults, but in children the progress is more rapid so that within six weeks of the onset of the symptoms, a stricture may have formed destroying part of the oesophageal wall. It is therefore important not to delay reduction of a hiatal hernia in a child if there is oesophagitis present, otherwise it may never be possible to reduce it.

ASSOCIATED LESIONS Before considering treatment it is important to find out whether there is another lesion in the stomach or duodenum causing delay in gastric emptying which in turn produces reflux oesophagitis. Many of our patients have gastric or duodenal ulcers as well as a hiatal hernia. The duodenal ulcer may be the primary lesion and when it begins to obstruct the pylorus it will cause retention of gastric contents, and reflux into the oesophagus if the hiatal mechanism is not intact. One woman presented with a giant gastric ulcer as well as a hiatal hernia.

Case 4 Illustrating association of large gastric ulcer with hiatal hernia A woman aged 74 years had had indigestion for only six years. A barium meal showed a hiatal hernia which was treated medically. She remained fairly well for three years and then developed severe epigastric pain which was periodic at first, but later became continuous and radiated into the back. The pain was so severe at times that she had to have injections of morphia.

Another barium meal showed the hiatal hernia but now there was a giant gastric ulcer which involved practically the whole of the lesser curvature and appeared to have penetrated deeply into the posterior abdominal wall (Fig. 3a).

1958. Operation. The giant ulcer involved the whole of the lesser sac but it was possible to dissect it intact from the body of the pancreas and the posterior abdominal wall. Sleeve resection of the body of the stomach was performed. The hiatal hernia was reduced and the hiatus repaired. Pyloroplasty was performed. The patient remains extremely well and has been free of all digestive symptoms (Fig. 3b).

Another patient perforated a duodenal and a gastric ulcer at the same time. Both were closed by

FIG. 3. These radiographs illustrate the association of peptic ulceration with hiatal hernia. Fig. 3a shows a giant gastric ulcer which had developed below a large hiatal hernia in a 74-year-old woman (Case 4). It was found possible to remove the ulcer by a sleeve resection and reduce the hernia (Fig. 3b).

suture and during the subsequent three weeks he developed dysphagia. A barium swallow and oesophagoscopy proved that he had an acute peptic ulcer of the oesophagus and incompetence of the cardia. He has been treated medically and all three ulcers have healed.

Two other patients were referred by general surgeons, having previously had a barium swallow and an oesophagoscopy examination. Both had easily recognizable hiatal herniae with peptic oesophagitis. However, a complete barium meal had not been performed in either case and when this was eventually carried out it revealed a pyloric neoplasm

obstructing the pylorus and preventing gastric emptying.

Case 5 A woman aged 57 years had had indigestion and discomfort after food for the past five months. She also vomited occasionally. A barium swallow was performed which showed a small hiatal hernia and gastric reflux (Fig. 4a). She was oesophagoscoped and the lower oesophageal mucosa was seen to be inflamed. She was transferred to the Department of Thoracic Surgery for possible surgical repair of the hiatal hernia. A barium meal performed by Professor Johnstone showed a small pyloric neoplasm which was the cause of her symptoms (Fig. 4b).

FIG. 4. Radiographs illustrating that peptic oesophagitis and a small hiatal hernia (Fig. 4a) can be caused by a pyloric carcinoma (Fig. 4b) which had not been recognized originally (Case 5).

FIG. 5. Radiographs illustrating the behaviour of the hiatus in cardiospasm. Here the heavy and dilated oesophagus may prolapse through the stretched hiatus into the abdomen (Fig. 5a) or rarely cardiospasm may be associated with a para-oesophageal hernia (Fig. 5b).

Reflux induced by dilatation of the cardia may be set up a peptic oesophagitis as illustrated in Case 6. Here after 22 years, dysphagia the oesophagus presented the typical appearances of advanced cardiospasm (Fig. 5c).

FIG. 5a

FIG. 5b

FIG. 5c

FIG. 5d

FIG. 5e

After dilatation with a hydrostatic bag a good channel was obtained through the cardia though it was rendered incompetent (Fig. 5d). One year later a severe peptic ulceration of the oesophageal mucosa had developed (Fig. 5e).

Cholelithiasis may produce the same symptoms as a hiatal hernia and occur in the same type of person. Repeated vomiting and belching caused by the presence of gallstones may so weaken the mechanism of the cardia that it eventually becomes incompetent, but otherwise there is no direct relationship between chronic cholecystitis and the development of oesophagitis.

OE SOPHAGEAL HIATUS IN CARDIOSPASM During the last stage of cardiospasm when the oesophagus becomes dilated and its lumen so large that it can accommodate a full meal, the weight of this loaded oesophagus stretches the ligaments and muscles of the hiatus so that the cardia may prolapse into the abdomen (Fig. 5a). The cardiac sphincter becomes

the last remaining control preventing gastric reflux, so that if this is damaged by dilatation or by an operation, peptic oesophagitis will follow. In order to prevent this from occurring, the oesophageal hiatus should be repaired at the same time as the Heller's operation, so that after the cardiac sphincter has been divided, the patient is left with a competent cardia.

Case 6 A man aged 64 years who had had 22 years of dysphagia due to advanced cardiospasm (Fig. 5c). The cardia was dilated twice with a hydrostatic bag at oesophagoscopy (Fig. 5d). He was relieved of his dysphagia for one year and then it returned in greater severity and he began to have haematemesis from which he died. He had severe peptic ulceration of the oesophagus due to incompetence of the cardia (Fig. 5e).

Rarely cardiospasm is associated with a para-oesophageal hernia and when this occurs it is obvious that the hiatus should be repaired after the hernia has been reduced and Heller's operation performed (Fig. 5b).

TREATMENT

MEDICAL TREATMENT After investigating the case fully and making sure that only minimal oesophagitis is present by oesophagoscopy, the patient may be treated medically for a time to see the response. This consists of: 1, Reduction of weight by dieting; 2, taking alkalies and antacids to reduce gastric acidity; 3, keeping to a semi-solid ulcer diet; 4, disposal of tight abdominal appliances, such as corsets; 5, the avoidance of stooping or bending positions. Sleeping well propped up with pillows to prevent reflux at night when most of the damage is done to the oesophageal mucosa (Aylwin, 1953). Patients tend to slip off pillows so it may be necessary to raise the head of the bed or become accustomed to a bed chair.

If the symptoms improve with medical treatment it may be a false impression of what is really happening unless a second oesophagoscopy examination is performed, and if this shows that the condition of the mucosa has not improved, it is a mistake to continue medical treatment any longer. In children when peptic oesophagitis is present, it is doubtful whether conservative treatment does any good at all, for it has been my experience that infants with reflux oesophagitis may produce a stricture within six weeks of the onset of their symptoms unless the cardia is made competent by operative repair.

Case 7 Illustrating rapid course of peptic oesophagitis in childhood An infant had vomited intermittently since birth. When he was 16 months old a barium swallow showed a hiatal hernia with severe peptic oesophagitis (Fig. 6a). He was treated medically and nursed in a bed chair in order to prevent gastric reflux (Fig. 6b). He appeared to make considerable improvement and seldom regurgitated food after his meals, while he was nursed in this position. A barium swallow taken after one month's medical treatment showed a dramatic improvement in the condition of the oesophagus. There was a wide lumen all the way down the oesophagus, but the cardia was completely incompetent (Fig. 6c). He was discharged home at this time his parents being told to keep him well propped up in his cot.

The improvement was not long lasting, however, for two months later, when he was 20 months old, he was admitted to the Department of Thoracic Surgery in an extremely ill condition. He had a right-sided pneumonia, most probably caused by the inhalation of food, and an empyema which had to be drained. He had developed a severe peptic stenosis of the oesophagus which required

frequent dilatations in order to keep the lumen patent (Fig. 6d). At oesophagoscopy it was considered that the stricture was now fibrous and would require resecting later. The empyema healed, and the right lung cleared. He was able to take a fluid diet and was maintaining his weight, so he was allowed to go home, and it was intended to see him frequently in the Out-patient Department. However, his family moved to another town and we have not received any further information about him.

BLIND BOUGINAGE When there is an established fibrous stricture above a hernia which is not reducible, it may be considered advisable to try bouginage and treat the patient conservatively rather than perform an extensive resection. If the patient is an adult and cooperative, he may be taught to pass the bougies on himself. I have always regarded this method of treatment as rather unpleasant and not without danger; however, there is no doubt that such people can have long periods of freedom from symptoms provided they adjust their diet to pass through the stricture.

Case 8 An infant boy was slow in gaining weight and frequently regurgitated his food after meals. He appeared not to be able to swallow properly when he was 20 months old, and he sometimes vomited while he was eating.

He first attended the Infirmary when he was 2 years old, i.e., in March 1948. A barium swallow at this time revealed a peptic stricture of the oesophagus and a small hiatal hernia (Fig. 7a). The stricture was short and fibrous; it required an occasional dilatation in order to keep it open, and so he continued for about a year. He then developed measles and was sent to a fever hospital. At this hospital, the dilatations were performed on him blindly using gum elastic bougies.

In June 1949 a severely emaciated and dehydrated child was transferred to the Department of Thoracic Surgery. There was complete oesophageal obstruction, for the child was unable to swallow even water. A gastrostomy was performed under local anaesthesia (Fig. 7b).

When the child had improved sufficiently to have an x-ray examination, a lipiodol swallow was performed which showed complete obstruction to the oesophageal lumen and a perforation leading to a mediastinal abscess, lying in the left paravertebral region. The mediastinal abscess had ruptured into the left lower lobe which was atelectatic and contained an abscess cavity. The infection had also spread into the bodies of the twelfth thoracic and first lumbar vertebrae, both of which showed evidence of osteomyelitis (Figs. 7c and d).

Gastrostomy feeding was continued for about a year. Postural drainage and antibiotic therapy controlled the infection in the lung, mediastinum, and spine. He continued to improve and rapidly put on weight. Several attempts were made at oesophagoscopy and once, retrogradely, via the stomach, to try to pass a small bougie through the original lumen and stricture; all failed; the lumen at the site of the original stricture did

FIG. 6a

FIG. 6b

FIG. 6c

FIG. 6d

FIG. 6. Radiographs illustrating the course of peptic stenosis in a child (Case 7) who presented at the age of 16 months with a peptic stenosis (Fig. 6a). This was treated medically by a bedchair (Fig. 6b) and appeared to effect a considerable improvement (Fig. 6c). However, four months later a firm peptic stricture has developed which required repeated dilatation (Fig. 6d).

not appear to exist. In March 1950 a left thoracotomy was performed. There was a tremendous amount of fibrosis in the mediastinum, and the mediastinal abscess was encountered which communicated by a bronchial fistula with the left lower lobe. Bare bone could be felt with a probe at the lower extremity of the mediastinal abscess which was one of the infected vertebrae. The oesophagus was eventually dug out of the fibrous tissue in the mediastinum. The stricture was quite short and it was excised. The oesophagus was anastomosed to the small pouch of stomach forming the hiatal hernia, and this hernia was then reduced. The left lower lobe still contained an abscess with a small bronchial fistula, but it was not large and it was left alone.

Two weeks after this operation the child was able to take fluids by mouth and later solids. Three months later he required an oesophagoscopic dilatation, but this had not been necessary again. The lung abscess healed without any more trouble and also the spine.

During the past 10 years he has been taking an ordinary diet and states that he can eat anything without discomfort. A recent barium swallow was also very satisfactory (Figs. 7e and f).

Case 9 *Illustrating successful outcome to self bouginage* A man aged 52 years had had indigestion for about a year. In 1956 he was admitted with an acute abdominal condition and at laparotomy he was found to have perforations of both a gastric and a duodenal ulcer. The two perforations were each closed by suture. After he had been discharged home he developed dysphagia and a barium swallow showed a peptic stricture of the oesophagus (Fig. 8a). He was treated medically, but required frequent dilatations in order to keep the stricture open. He was taught self-bouginage which he continued to do for six months. After another six months on medical treatment the three ulcers appeared to heal, for he had no symptoms and the oesophagus looked normal (Fig. 8b). He has remained symptom-free for the past four years.

FIG. 7a

FIG. 7b

FIG. 7c

FIG. 7d

FIG. 7e

FIG. 7f

FIG. 7. Radiographs illustrating the course of a peptic stricture which developed in March 1948 in a boy aged 2 years (Case 8). A barium swallow at that time showed the stricture with a small hiatal hernia and irregular peristaltic movements (Fig. 7a). In June 1949 oesophageal perforation followed blind dilatation with the development of lung abscess, mediastinal abscess, and osteomyelitis of the spine. Fig. 7b illustrates his emaciated state at this time. A lipiodol swallow shows perforation leading into the mediastinal abscess in the left peravertebral region and the fluid level of the lung abscess is visible in the left lower lobe (Fig. 7c). The oesophagus can be seen to be completely occluded and loss of joint space between T.12 and L.1 indicates an osteomyelitis of the spine (Fig. 7d). Ten and a half years after the resection of the stricture he is quite fit, normally developed (Fig. 7e), and taking a normal diet. Barium swallow in February 1960 showed a good lumen to the oesophagus (Fig. 7f).

FIG. 8c

FIG. 8. Radiographs illustrating that a successful outcome may follow dilatation of a peptic stricture by self bouginage (Case 9). After perforation of a gastric and duodenal ulcer dysphagia due to a peptic stricture developed (Fig. 8a). Repeated dilatations by self bouginage produced rapid improvement with healing of all ulcers and a return of the oesophageal lumen to normal (Fig. 8b). The oesophagus may, however, be ruptured while dilatation is being carried out under direct vision (Case 10) where the chest radiograph shows the right pleural cavity to be filled with gastric contents (Fig. 8c).

DILATATIONS AT OESOPHAGOSCOPY This is a useful method of dealing with a stricture in a patient who is not suitable for an operation. It is less dangerous than by dilating the stricture blindly, but even when the lumen has been visualized at oesophagoscopy it has been known for the oesophageal wall to be perforated by bogies.

Case 10 Illustrating traumatic rupture of oesophagus during dilatation at oesophagoscopy (Fig. 8c) A boy

aged 8 sustained a traumatic rupture of the oesophagus while a peptic stricture was being dilated at oesophagoscopy.

Two days later he was admitted to this department. At thoracotomy, after the pleural cavity had been cleaned, it was difficult to find the hole in the oesophageal wall. About a pint of saline was poured into the pleural cavity and oxygen was blown down an oesophageal tube by the anaesthetist. This quickly demonstrated the leak and the hole was closed by suture. Jejunostomy was also performed.

This operation was performed eight and a half years ago. The boy has still a severe peptic stenosis and attends about every six months for an oesophagoscopy dilatation. He is awaiting admission for resection of the stricture, and reconstruction with an isolated jejunal loop.

Patients may not wish to have a big operation and prefer to have an occasional oesophagoscopy dilatation. Several elderly patients are attending this department who have continued with this method of treatment most of their lives, maintaining their weight and enjoying their meals. After a dilatation the stricture has remained open for a long time and a few have remained reasonably well for one or two years provided the peptic ulcers are not active.

Patients should be encouraged to swallow food after a dilatation, for a bolus of food is the best oesophageal dilator and is a simple way of keeping the stricture open.

Fish and hard foods should be avoided. The coarser types of fish nearly always produce obstruction as does the pulp of an orange.

SURGICAL TREATMENT

REDUCTION OF HERNIA AND REPAIR OF OESOPHAGEAL HIATUS In an article on this subject, Allison (1951) described his technique for repairing an hiatal hernia. Briefly this consists of 1, a left thoracotomy incision usually resecting the eighth rib, and a radial incision through the diaphragm to open up the peritoneal cavity; 2, division of the phreno-oesophageal ligament and hernial sac around their attachment to the edge of the hiatus; 3, mobilization of the lower oesophagus and reduction of the hernia; 4, suture of the divided sac and phreno-oesophageal ligament to the undersurface of the diaphragm; 5, making the hiatus smaller by closing the posterior part of it. This is achieved by suturing together the two muscle pillars behind the oesophagus. They in turn are formed by the right crus dividing to encircle the hiatus.

MODIFICATIONS OF ALLISON'S METHOD I have more or less followed this technique but during the last five years have modified it in the following ways.

A left thoraco-abdominal incision dividing the costal margin through the seventh or eighth costal cartilage. The rib is not resected and the pleural cavity is opened by incising the periosteum of the rib bed. This gives an excellent exposure of both the upper and the lower surfaces of the diaphragm. It carries no greater risk than a thoracotomy incision, and appears to cause less post-operative pain. It also allows one to remove a diseased gall bladder if this is necessary.

The diaphragm is opened a distance of 4 in. by dividing it near its costal attachment in a posterior direction. The incision through the diaphragm lies very near to the lateral chest wall and so avoids division of any branches of the phrenic nerve. Post-operatively it has a normal range of movement and we have not experienced difficulty in keeping the left lower lobe fully aerated.

The peritoneum is sutured and closed over the hole through which the hernia occurs, and is of prime importance when repairing any other hernia of the peritoneal cavity. This hole is the posterior part of the stretched and enlarged hiatus. Here the posterior abdominal wall is not covered with peritoneum but is a bare area lying behind the stomach and cardia, and is between the reflections of the greater and lesser sacs on to the crura of the diaphragm. It usually contains a pad of extraperitoneal fat, which slides up into the chest through the posterior part of the hiatus.

After reduction of the hernia in a similar way to that described by Allison, if one is using the thoraco-abdominal incision, it is possible to cover this bare area with peritoneum by suturing together the cut ends of the greater and lesser sac behind the oesophagus (Fig. 9). It is through this hole that the hernia recurs, so it is important to close it properly.

This repair may be further strengthened by suturing together both pleural sacs above the diaphragm. These sacs are thickened where they are in contact with the hernia and this thickening is particularly noticeable where each mediastinal pleura is reflected on to the diaphragm. These reflections hold a silk suture and make a stronger repair than suturing only the frayed muscle pillars together.

The phreno-oesophageal ligament and the hiatal sac have not a firm attachment to the oesophagus so that suturing these structures to the under surface of the diaphragm will not prevent recurrence of the hernia unless the hiatus is repaired properly at the same time.

I have operated on at least six patients in whom herniae have recurred after an Allison type of repair. In each case the ligament and sac remained sutured to the undersurface of the diaphragm but

FIG. 9. Diagram showing the peritoneal relations of the oesophagus; a is stitched to b in order to close the hole behind the oesophagus.

the other ends of these structures had become detached from the cardia, allowing it and a pouch of stomach to go up into the chest. Repair of the peritoneum below the diaphragm and suturing the pleural sacs together above the diaphragm appears to offer a stronger repair to the hiatus.

This more extensive repair has proved most successful in children where previous attempts at reduction had failed (Cases 11 and 12).

Case 11 A boy weighed 10 lb. at birth. He began to vomit on the second day and continued to lose weight. When he was a month old a Ramstedt's operation was performed because he was thought to have congenital pyloric stenosis. The pylorus was normal but a large hiatal hernia was felt while the abdomen was open. He continued to vomit and lose weight. When he was 2½ months old, he weighed less than 8 lb. He was transferred to the Department of Thoracic Surgery. A barium swallow and oesophagoscopy showed a large hiatal hernia with peptic ulceration of the oesophagus (Fig. 10a).

January 1958. Operation. The hernia was repaired through a left thoraco-abdominal incision. The child immediately improved the vomiting stopped and he began to gain in weight (Fig. 10c). He has continued to improve ever since. A post-operative barium swallow shows that the hernia remains reduced and that the cardia is competent (Fig. 10b).

Case 12 A girl weighed 7½ lb. at birth. She had vomited after almost every meal and had continued to lose weight. In July 1958 when she was 2 months old she weighed 1 lb. less than her birth weight. A barium swallow examination demonstrated a large hiatal hernia and at oesophagoscopy superficial ulceration of the oesophagus was seen (Fig. 11a).

July 1958. Operation. The hernia was reduced through a left thoraco-abdominal incision. She made excellent

FIG. 10a

FIG. 10b

FIG. 10c

FIG. 10. Radiographs illustrating the successful repair of a large hiatal hernia in an infant aged $2\frac{1}{2}$ months (Case 11) in whom a severe peptic oesophagitis had developed (Fig. 10a). Post-operative films, eight months after the repair of the hernia, showed it to be satisfactorily reduced, though the oesophagus is slightly narrowed at the site of previous ulceration (Fig. 10b). Rapid weight gain followed the operation (Fig. 10c).

progress. She has continued to gain in weight and has not vomited since the operation. She enjoys her food and looks very healthy. May 1960. A barium swallow examination by Professor Johnstone: 'There was no evidence of reflux and no recurrence of the hernia' (Fig. 11b).

PEPTIC STRICTURE AND SHORTENING OF THE OESOPHAGUS During the past five years I have performed few resections of the oesophagus for peptic ulceration. Instead, I have reduced the hernia by performing an extensive mobilization of the oesophagus, sometimes the whole length of it inside the chest. The advantages of leaving a patient with a physiological oesophagus rather than a poor substitute

are obvious. It is well worth attempting to reduce the hernia even though the oesophagus appears shortened before embarking on a resection.

The mobilization of the oesophagus in these cases must be carried above the level of the aortic arch and even to the apex of the pleura in order that the hernia will reduce without tension. This may necessitate dividing all the branches of both vagi, which surround the oesophagus, tethering it to the mediastinum and preventing it from being mobilized adequately. If both vagi are divided, pyloromyotomy or pyloroplasty should be performed as well in order to prevent post-operative pylorospasm and interference with gastric emptying.

FIG. 11a

FIG. 11b

FIG. 11c

FIG. 11d

FIG. 11. Radiographs illustrating the successful repair of hiatal hernia in children. An infant girl (Case 12) had vomited since birth and at 2½ months was 1 lb. less than her birth weight, barium swallow revealing a large hiatal hernia and peptic oesophagitis (Fig. 11a). Two years after repair a further swallow showed no evidence of reflux or of recurrence of the hernia (Fig. 11b). Successful reduction accomplished in a 10-month-old boy who had presented with hiatal hernia, peptic ulceration of the oesophagus and shortening of the oesophagus (Case 13). After repair, bilateral vagotomy, and pyloroplasty a competent cardia was obtained (Figs. 11c and d).

The left main bronchus and the aortic arch compress the oesophagus as they cross over it, and usually prevent peptic ulceration spreading up further and to a higher level in the oesophagus. When mobilizing an oesophagus which is ulcerated, difficulty is usually encountered as high as the aortic arch. The oesophageal wall feels rigid and bound to the mediastinum with vascular adhesions. Above the aortic arch, the oesophagus feels normal and the wall will now stretch and allow the hernia to reduce. Mobilization should be continued until the hernia can be replaced below the diaphragm without tension. This may necessitate extending the thoracotomy part of the incision or making a second opening into the chest through the fourth or fifth intercostal space, in order to reach the upper part of the oesophagus. There need be no anxiety over the blood supply to the oesophagus, which is excellent, for even after full mobilization sloughing has not occurred.

This type of operation has now been performed on 18 patients all of whom had established peptic ulceration of the oesophagus and on whom a few years ago we should have considered that resection was the only surgical operation that could benefit them. The operation has proved a great success in

every case, the ulcers have healed and the patients are taking a normal diet. This has not been achieved without a certain amount of anxiety in the post-operative stage, for after this operation the oesophageal lumen becomes extremely narrow where it is ulcerated. It remains narrow while the ulcer is healing, which may take six to eight weeks, and during the whole of this time the patient must be kept strictly to a fluid diet. All the patients behaved in this way: some of the ulcers healed in three weeks and then the patients were allowed a semi-solid diet, other ulcers took eight weeks, but only one patient required an oesophagoscopy dilatation. A recent follow-up examination of these patients has proved very encouraging, all of them are free of digestive symptoms and taking a normal diet.

Case 13 A boy aged 10 months had vomited since birth. He had also had two small haematemeses, the first when he was 5 days old. His mother had often seen the vomits streaked with blood.

February 1958 a barium swallow examination showed a hiatal hernia, peptic stricture of the oesophagus, and shortening of the oesophagus. He was treated medically for two months but made no progress so he was transferred to the Department of Thoracic Surgery (Fig. 11c).

FIG. 12. Radiographs showing a hiatal hernia, a gastric-lined lower oesophageal segment, and peptic oesophagitis with apparent shortening of the oesophagus which developed in a woman of 48 years (Case 14) (Fig. 12a). After a year's medical treatment and dilatations the stricture had worsened (Fig. 12b) and one month after operation with reduction of hernia, pyloroplasty and bilateral vagotomy, the oesophagus is normal without reflux (Fig. 12c). In Case 15, extensive peptic oesophagitis had developed with a hiatal hernia and reached the aortic arch (Fig. 12d). Seven months after reduction, a normal oesophagus and competent cardia were present (Fig. 12e).

April 1958. Operation. Extensive mobilization of the oesophagus in order to reduce the hernia. Bilateral vagotomy and pyloroplasty performed. The child made excellent progress after the operation. He continued to gain weight and enjoy his food.

A barium swallow taken only three weeks after the operation by Professor Johnstone: 'This appears to be an excellent result. There is no evidence of recurrence, no reflux. The oesophagus appears normal. The diaphragm moves freely. Emptying rate of stomach normal.'

The child has continued to progress well and now resents further investigations. A recent barium swallow shows that the hernia remains reduced, the oesophagus looks normal and there is no reflux (Fig. 11d).

Case 14 In 1952 a woman aged 48 years began to have dysphagia. A barium swallow performed elsewhere revealed no abnormality of the oesophagus. Her symptoms gradually became worse and in June 1954 she first attended this department. Professor Johnstone performed an x-ray examination and demonstrated a large hiatal hernia, peptic ulceration of the oesophagus with apparent shortening of the oesophagus, very free reflux from the stomach, and he also noted deformity of the duodenal cap.

Oesophagoscopy June 1954. Peptic ulceration surrounding the whole oesophageal lumen encountered at 30 cm. from the incisor teeth. Oesophageal mucosa inflamed as high as the aortic arch. The ulcer felt soft when bougies were passed through it, but at this time it was considered that the hernia was not reducible. She was

put on medical treatment, consisting of a weight-reducing diet, alkalis after meals, etc.

August 1954. She was again oesophagoscoped and the stricture dilated because she was not able to take anything other than fluids. She lost over a stone in weight and appeared to improve for a time.

A repeat barium meal by Professor Johnstone demonstrated more deformity of the duodenal cap so that an active duodenal ulcer was considered to be present. The peptic stenosis of the oesophagus remained about the same.

February 1957. She had deteriorated during the past month and had recently had a small haematemesis. Another barium meal by Professor Johnstone: 'There is still a well marked peptic stricture above a small segment of gastric-lined oesophagus. The hiatal hernia was easily produced and reflux flooding occurs readily. The rest of the stomach is normal. The duodenal cap is much deformed and active ulceration is thought to be present. There is a moderately large diverticulum arising from the second part. The gall bladder was examined and found to be normal' (Fig. 12a).

She was oesophagoscoped and the stricture was again dilated with bougies. It still felt soft, and dilated very easily. She continued on her medical treatment and remained fairly well for a year.

In June 1958 dysphagia for fluids returned and within two weeks she was only able to take sips of water. A barium swallow showed that the oesophageal lumen was almost completely stenosed (Fig. 12b).

June 26 1959. Operation. Left thoraco-abdominal incision. The hernia was reduced after extensive mobilization of the oesophagus. All vagal fibres were divided during the mobilization. There was some scarring of the first part of the duodenum but no ulcer was seen. Pyloroplasty was performed.

After the operation she had difficulty in taking fluids by mouth during the first fortnight. Then she gradually improved so that a month after the operation a barium swallow examination showed a normal oesophagus.

She continued to improve ever since and takes a normal diet without discomfort. The last barium swallow examination performed by Professor Johnstone in March, 1960: 'The left dome of the diaphragm moved satisfactorily. The oesophagus appeared normal. No recurrence of hernia detected. No reflux' (Fig. 12c).

Case 15 In November 1950 a woman aged 50 years presented herself with several years' history of heartburn and flatulence after meals. During the past month she had had dysphagia, so that everything she took by mouth, including fluids, appeared to stick high in the oesophagus and just behind the manubrium.

23 November 1950. A barium swallow showed a hiatal hernia, with free reflux from the stomach. There was a long peptic stricture of the oesophagus reaching to above the level of the aortic arch (Fig. 12d).

Oesophagoscopy confirmed the presence of peptic ulceration which was encountered at 19 cm. from the incisor teeth. Ulceration surrounded the whole oesophageal lumen which measured only 4 mm. in diameter. The stricture felt soft when it was dilated with bougies.

She was put on medical treatment but did not improve. A further oesophagoscopy dilatation was necessary in February 1950 and again in May 1950. The appearances of the oesophagus at these examinations remained unchanged.

June 1950. Operation. The hernia was reduced after extensive mobilization of the oesophagus. The cardia was rendered competent and the peptic ulceration of the oesophagus quickly healed.

A barium swallow examination performed seven months after the operation showed a normal looking oesophagus. The hernia remained reduced and there was no reflux. She has remained well ever since, is taking a normal diet, and has no digestive symptoms (Fig. 12e).

RESECTION AND RECONSTRUCTION WITH JEJUNUM
When there is a tough fibrous stricture of the oesophagus with considerable shortening, and after other methods of treatment have failed to relieve the patient's symptoms, there is no alternative line of treatment other than to resect the diseased segment and reconstruct the oesophagus with stomach, jejunum, or colon. In Leeds, we prefer to use the jejunum for the reconstruction after an oesophagectomy, probably because we have long been accustomed to preparing jejunal loops. Since 1953 we have been using an 'isolated' or interposed jejunal loop (Allison, Wooler, and Gunning, 1957) and the operation is conducted in the following way:

A left thoracoabdominal incision is used through the bed of the eighth rib. When the operating theatre has been darkened, the jejunal mesentery is inspected with a light behind it. The first loop of jejunum near to the duodenojejunal flexure is usually selected for preparation of the isolated loop. While the light is being held behind the jejunum, the vessels in a fat-laden mesentery can easily be seen using this method of illumination (Fig. 13a). It is rarely necessary to divide more than two of the main vessels supplying the vascular arcades, and these vessels must be divided well away from their divisions. The peritoneum and lymphatic tissue are removed from both sides of the mobilized arcades so that they hang denuded of all their coverings, almost like a garland, from the jejunal loop which may now be straightened and is no longer curved.

After the diseased portion of oesophagus, the herniated portion of stomach, and the proximal third of the stomach have been resected, the jejunal loop is brought through the mesocolon and its upper end is anastomosed to the divided oesophagus by an end-to-end anastomosis. This anastomotic line must be made as wide as possible. It may be enlarged by incising up the oesophagus anteriorly and joining the antemesenteric side of the jejunum to the top of this incision (Fig. 13b).

The loop is divided below the diaphragm. The upper end of this division which also forms the lower end of the jejunal loop is anastomosed to the remaining segment of stomach (Fig. 13c). The interposed loop between the oesophagus and stomach is now attached only by its blood supply coming from the mesenteric vessels.

The two ends of jejunum between which the loop was taken are joined together, and so the continuity of the alimentary tract is restored.

Case 16 A woman was aged 45 years when she first attended this department in 1956.

In 1912, when she was 2 years old, she drank a corrosive liquid which produced an oesophageal stricture. She had dysphagia ever since and required frequent dilatations in order to keep the lumen open.

In 1954 and 1955 she had small haematemeses. A barium swallow performed by Professor Johnstone in 1956 showed a long stricture in the middle of the oesophagus below which there was a pouch of stomach above the diaphragm, the appearance being typical of a sliding hiatal hernia with a long peptic stricture of the oesophagus (Fig. 14a). (In all probability the corrosive liquid had damaged a segment of oesophagus and prevented it from growing, so that the oesophagus became shortened, and dragged up a pouch of stomach into the thorax. I have seen peptic stenosis follow a corrosive stricture in two other patients and so I presume it is not an uncommon sequel.)

FIG. 13a

FIG. 13. Drawings illustrating the preparation and anastomosis of the jejunal loop to the oesophageal stump. Preparation of the jejunal loop, showing division of the vessels facilitated by light shining through the mesentery (Fig. 13a).

FIG. 13b

Anastomosis of jejunum to oesophagus. The oesophagus is being incised with scissors to increase the width of the anastomosis (Fig. 13b).

FIG. 13c

Anastomosis between the jejunum and the stomach. The jejunum has been opened longitudinally so as to make its lumen about the same size as the divided stomach (Fig. 13c).

FIG. 14a FIG. 14b FIG. 14c

FIG. 14. Radiographs illustrating the successful resection and reconstruction of a long fibrous stricture of the oesophagus produced by corrosive fluids in a 45-year-old woman (Case 16). The oesophagus is shortened and a hiatal hernia is present (Fig. 14a). After the operation of oesophago-jejuno-gastrostomy the oesophago-jejunal anastomosis can be seen with the straight loop of jejunum passing through the posterior mediastinum (Fig. 14b) leading to a wide jejuno-gastric anastomosis (Fig. 14c).

During the next two years she was oesophagoscoped six times and the stricture was dilated each time, but she derived little or no benefit from this treatment and complained of pain in the back as well as dysphagia for all solids.

January 1959. Operation. Resection of ulcerated portion of oesophagus and reconstruction with an interposed loop of jejunum. The specimen removed contained an ulcer crater measuring 3 cm. $\times$ 2 cm. which was arising in oesophageal mucosa. The operation relieved her of the dysphagia, but she had a certain amount of abdominal discomfort after meals which was considered to be due to the division of both vagi.

A barium swallow taken four months after the operation shows: (1) The oesophago-jejunal anastomosis; (2) the almost straight lumen of the isolated jejunum as it passes down the posterior mediastinum; and (3) the wide jejuno-gastric anastomosis in the abdomen (Fig. 14b and c).

During the past year she has improved considerably and she has now little or no discomfort after meals. She has had no dysphagia since the operation and is taking an ordinary diet.

Case 17 In 1949 a woman aged 62 years attended this department. She stated that during the past seven years she had had dysphagia and she had occasionally experienced a pain in the centre of the chest which radiated into the back between the shoulder blades.

A barium swallow examination showed a stricture at

the junction of the middle and lower thirds of the oesophagus associated with a hiatal hernia. She was oesophagoscoped and the appearance of severe peptic ulceration of the oesophagus was seen; superficial ulceration was evident at 20 cm. from the incisor teeth and a firm stenosis encountered at 27 cm. The stenosis was dilated with bougies. She derived no benefit from this procedure and although she was kept on strict medical treatment her symptoms did not improve.

Her general condition was only fair; she was an obese, rather frail woman who looked older than her years. Her blood pressure was 190/110 mm. Hg, she had little or no chest movement, and there was some oedema of the ankles. She was considered unsuitable for any form of operative treatment.

During the subsequent 11 years she was treated conservatively, but all this time she was gradually deteriorating and she lost over 2 st. in weight. She was never able to take anything by mouth other than fluids and she frequently had difficulty in getting these down. During this period of medical treatment she had to be oesophagoscoped 14 times and the stricture dilated.

The pain in her back became worse and continuous so that she was unable to sleep at night, and in fact her life became so unpleasant that she was prepared to take any operative risk. A barium swallow performed by Professor Johnstone had shown the peptic stenosis below which was a segment of oesophagus lined with gastric mucosa and then a pouch of stomach (Figs. 15a and b).

28 October 1958. Operation when the patient was 71 years old. Resection of the peptic stenosis, the segment of old oesophagus lined with gastric mucosa and the hiatal hernia. Oesophago-jejuno-gastrostomy performed. Pyloromyotomy was also carried out.

During the post-operative period the stomach became distended due to delay at the pylorus. The pyloromyotomy had not been complete, so the abdomen was reopened and pyloroplasty performed. She had no further trouble and made an excellent recovery.

The specimen which was removed showed a chronic peptic ulcer arising above from oesophageal mucosa and below from gastric mucosa. Below the ulcer there was a segment of oesophagus lined with gastric mucosa and then the pouch of stomach.

A post-operative barium swallow showed the oesophago-jejunal anastomosis in the mediastinum and the jejuno-gastric anastomosis in the abdomen. The pylorus now appeared widely open and there was no delay in gastric emptying (Figs. 15c and d).

She was recently seen for review as an out-patient and looked extremely well. She is taking a normal diet and has no digestive symptoms. She has gained over a stone in weight and states that she feels fitter than she did 20 years ago.

Case 18 In 1956 a man aged 66 years first attended this department with the history that five years previously he had begun to have dysphagia. This became so severe that a gastrostomy was performed at another hospital. He was able to take only clear fluids by mouth.

During the past year he had had severe pain in the back situated between the shoulder blades, which frequently kept him awake at night.

FIG. 15a

FIG. 15b

FIG. 15c

FIG. 15d

FIG. 15. Barium swallow showing the presence of a peptic ulcer and stricture arising at the upper margin of a segment of oesophagus lined with gastric mucosa associated with a hiatal hernia (Case 17) (Figs. 15a and b). After resection and oesophago-jejuno-gastrostomy the oesophago-jejunal anastomosis is well shown (Fig. 15c) as is its entry to the stomach (Fig. 15d).

FIG. 16a

FIG. 16b

FIG. 16c

FIG. 16d

FIG. 16. Radiographs illustrating oesophageal reconstruction in a man aged 68 years (Case 18) with peptic stenosis of the oesophagus and a Barrett's ulcer (Figs. 16a and b). Again resection and oesophago-jejuno-gastrostomy proved successful (Figs. 16c and d).

A barium swallow performed by Professor Johnstone showed a stricture at the level of the aortic arch, below which was a segment of oesophagus approximately 15 cm. long lined with gastric mucosa. About 6 cm. above the diaphragm another expansion was evident which was obviously a pouch of stomach (Figs. 16a and 16b).

Oesophagoscopy in December 1956 confirmed the peptic ulceration at 25 cm. from the incisor teeth. The ulcer surrounded the whole lumen of the oesophagus and dilated with difficulty. Biopsies were taken of the mucosa above and below the ulcer and from the ulcer. The histology of these biopsies proved that there was gastric mucosa in the oesophagus below the ulcer, inflamed oesophageal mucosa above the ulcer, and that the ulcer was of a simple nature and not malignant.

In view of his age it was decided to treat him medically, and this form of treatment was continued for 18 months without any lasting improvement.

July 1958. Operation, when he was 68 years old. Oesophageal resection and oesophago-jejuno-gastrostomy was performed. A left thoraco-abdominal incision was used dividing the seventh costal cartilage and opening the thorax through the bed of the seventh rib. This did not give sufficient access to the site of the ulcer which was a long way up the mediastinum, so a second opening was made into the left chest through the bed of the fourth rib. The ulcer was firmly adherent to the deep side of the aortic arch and from which it proved impossible to free without mobilizing the aorta. The first four pairs of intercostal arteries were therefore divided, and the back of the aorta inspected. The base of the ulcer proved to be the far wall of the aortic arch and it was also involving the lower end of the trachea near to the origin

of the left main bronchus. It was not possible to dissect the base of the ulcer from the aorta and it was also considered unwise. The oesophagus was divided around its base, which was left adherent to the arch.

The remainder of the operation presented no difficulties and was completed in the usual way. The patient made a rapid recovery and has progressed extremely well. He has gained over a stone in weight and is taking a normal diet. He is a fairly heavy drinker and smoker; when he was seen recently, he stated that he had not had to restrict any of his activities (Figs. 16c and d).

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