PEPTIC ULCER OF THE OESOPHAGUS

Lecture delivered at the Royal College of Surgeons of England
on
30th September 1953

by

Ronald Belsey, M.S., F.R.C.S.

Surgeon-in-Charge, Department of Thoracic Surgery, Frenchay Hospital, Bristol.

IF BY THE term " peptic ulcer " of the oesophagus is meant a lesion similar to that which occurs in the stomach and duodenum, then the condition probably does not exist ; assuming the oesophagus to be that part of the upper intestinal tract lined by stratified squamous oesophageal mucosa, and the stomach that part lined by gastric mucosa. The conception arose largely as a result of the demonstration by the radiologists of barium outlined irregularities which were interpreted as " ulcer craters " in the lower third of the oesophagus. Unfortunately, there was rarely any confirmation of the presence of a discrete ulcer crater by endoscopic examination or by the examination of excised specimens ; nor was the significance of the underlying hiatus hernia, nor even its presence, always recognised, and so no doubt arose as to whether the crater could be above or below the oesophago-gastric mucosal junction.

This interpretation of the radiological changes was supported by the accepted theory that areas of ectopic mucosa are common in the lower third of the oesophagus and are the site of these " ulcers." This theory was tenable only so long as the endoscopic examination of the affected area was omitted, and enjoyed no support from observed facts. Brown Kelly had shown that ectopic gastric mucosa when present in the oesophagus is usually found in the upper third of the organ, rarely elsewhere. Moreover, Barrett has, after intensive search, found no single recorded instance of a proven pathological lesion in an area of ectopic gastric mucosa in the lower oesophagus.

What, then, in the light of present knowledge is the correct interpretation of the " ulcer niche " in the lower oesophagus ? These niches are chronic peptic ulcers occurring in the segment of stomach herniated through the oesophageal hiatus and lie below the oesophago-gastric mucosal junction. If details of mucosal pattern are obscured or ignored the gastric pouch can easily be mistaken for the lower third of the oesophagus.

A further radiological misconception which has an important bearing on this subject is that of the " congenital short oesophagus," based on the assumption that because the oesophagus looks short it must be short. Unfortunately, this dogma deterred the surgeon from exploring the region and attempting to correct what appeared to be an unalterable state of affairs ; it remained for Allison, to whom is due the credit for our present knowledge concerning diaphragmatic hiatus hernia and its complications, to demonstrate that when stomach herniates through the hiatus the oesophagus shortens by muscular retraction and not by any anatomical

deficiency in oesophageal tissue. Allison records very few instances of true congenital short oesophagus in 250 cases of hiatus hernia and has recently advanced a different explanation for these appearances.

Oesophagoscopic examination of the lower third and cardia in cases of hiatus hernia of the sliding (Allison) variety reveals certain changes. The oesophago-gastric mucosal junction can be clearly identified in most cases. When the examination is performed under local anaesthesia the cardia is seen to be completely patulous throughout the respiratory cycle and the normal pinchcock action of the right crus of the diaphragm in occluding the cardia during inspiration is absent. There is free reflux of gastric secretion up into the oesophagus during inspiration. The oesophagoscope can be passed through the cardia into the stomach without difficulty and a typical peptic ulcer may be seen in the gastric mucosa, but never above the mucosal junction. In about 60 per cent. of cases diffuse inflammatory changes, to be described later, are seen extending upwards from the mucosal junction ; these changes bear no resemblance to an ulcer crater.

When the inflammatory changes, known as reflux oesophagitis, become chronic they lead frequently to fibrosis and secondary stenosis of the lower third of sufficient severity to call for resection. Much of our knowledge of the morbid anatomy of oesophagitis has been gleaned from examination of the excised specimens, but no support has been forthcoming for the conception of discrete chronic peptic ulcer craters occurring above the oesophago-gastric mucosal junction.

Definition of oesophagus and stomach

Controversy exists concerning the anatomical point at which oesophagus ends and stomach begins. This has usually been regarded as the line or junction between oesophageal and gastric mucosa, irrespective of the nature of the overlying muscle layers. The normal oesophagus possesses a very lax submucous layer which permits considerable latitude of movement between the mucosal and muscle layers, and the mucosal junction may not necessarily coincide with the point where the oesophageal muscle architecture merges into that of the stomach. But it is the contrasting features of the two mucosae which impart to these organs their chief characteristics, and the structure of the muscle layer is of relatively slight importance. The mucosal junction therefore appears to be the logical boundary between oesophagus and stomach.

Allison has recently described an unusual type of case where an hiatus hernia is complicated by an area of oesophagitis and stenosis in the upper or mid-third of the oesophagus, a zone of apparently normal oesophagus intervening between the stricture and the cardia. Further investigation in this type of case has, however, revealed that although the muscle architecture of this zone is typically that of the oesophagus, the viscus is lined by gastric mucosa. This, presumably developmental, abnormality has been termed by Allison " the oesophagus lined by gastric

mucosa." Some would regard this zone as belonging properly to the stomach irrespective of its external appearance, in view of the nature of the mucosal lining. When stenosis secondary to reflux oesophagitis is present the lower margin of the stricture can invariably be regarded as the site of the mucosal junction, irrespective of the configuration of the viscus below.

The fundamental anatomical and physiological features which bear upon the whole subject of hiatus hernia and oesophagitis are :

(i) the presence of a physiological sphincter at the oesophago-gastric junction preventing the reflux of gastric secretion into the oesophagus, the mucosa of which is ill equipped to withstand its erosive action ;

(ii) in the absence of any conclusive evidence of an intrinsic sphincter at the lower end, the muscle fibres of the right crus of the diaphragm which form the margin of the oesophageal hiatus appear to carry the responsibility for maintaining a sphincter and preventing reflux ;

(iii) once the cardia is divorced from the right crus there is no longer any mechanism to prevent reflux, and the erosive gastric secretion has free access to the oesophageal mucosa under the influence of negative intra-thoracic pressure, positive intra-abdominal pressure, and changes in posture.

Wangensteen and his associates have demonstrated in the experimental animal the devastating effect of gastric secretion when introduced into the animal's oesophagus, proceeding to perforation in as short a time as 20 minutes in some instances. Wangensteen concluded that it is the pepsin secretion rather than acid secretion in the herniated pouch which is responsible for the severity of the resulting oesophagitis.

A further anatomical feature which has an important bearing on the incidence of oesophagitis is the " cell population " of the upper end of the stomach. The " mucous collar " intervening between the cardia and the upper portion of the body of the stomach bearing the pepsin secreting and oxyntic cells varies in extent. When the cardiac end of the stomach has herniated into the chest through the hiatus, the relative proportion of mucous secreting to pepsin secreting cells in its mucosa will determine the risk of subsequent oesophagitis and may be responsible for the varying incidence of this complication in different individuals afflicted with similar types of hernia. Alywin has measured the peptic activity of oesophageal juice and is attempting to correlate this with the exact structure of the gastric mucosa in the region below the cardia.

Inflammatory lesions occurring in the oesophagus

Having reviewed the lack of evidence to support the occurrence of discrete peptic ulcer craters in the oesophageal mucosa, the following

inflammatory conditions known to occur at the lower end of the supra-diaphragmatic segment of the gullet will be described :—

(i) Reflux oesophagitis complicating an hiatus hernia and incompetent cardia.
(ii) Acute or chronic peptic ulceration of the intra-thoracic portion of stomach.
(iii) Similar lesions occurring in the " oesophagus lined with gastric mucosa."

Pathology of reflux oesophagitis

This condition is essentially a diffuse inflammatory process occurring in the mucosa of the lower third of the oesophagus in the presence of an incompetent cardia, and, in the light of Wangensteen's experiments, presumably due to the reflux of gastric secretion into the lower oesophagus. Incompetence of the cardia is generally associated with an hiatus hernia of the sliding type (Allison) of which the important feature is the obtuse opening of the oesophagus into the apex of the herniated segment of stomach. Cases are seen, however, in which the cardia is incompetent but there is no definite evidence of an hiatus hernia ; there is free reflux of the barium meal with changes in posture and on endoscopic examination there is a complete absence of any pinchcock occlusion of the cardia during the inspiratory phase of respiration. This condition may be complicated by reflux oesophagitis and even secondary stenosis. Surgical exploration of the diaphragm in these cases has revealed a lax and widely dilated oesophageal hiatus, and repair of the hiatus by means of a similar technique to that used in the treatment of an hiatus hernia has led to the rapid healing of the oesophagitis and relief of the patient's symptoms. The increasing use of the oesophagoscope and local anaesthesia has focused attention more on the function of the cardia rather than the volume of stomach lying above the diaphragm.

Approximately 60 per cent. of the " sliding " type of hiatus hernias are complicated by reflux oesophagitis when first examined, but only prolonged survey and follow-up will reveal the true incidence of this complication and the statistical risk of its development in any individual with this type of hernia.

In the " rolling " type of hernia and the rare para-oesophageal form, this complication is very unusual. The main difference between the sliding and rolling types lies in the oblique entry of the oesophagus into the stomach in the latter and a possible valvular mechanism preventing reflux of gastric secretion ; also the greater bulk of the herniated segment of stomach appears in the barium films to compress the lower end of the oesophagus and this again may tend to prevent reflux.

The oesophagitis starts as a superficial ulceration of the oesophageal mucosa, first along the crests of the rugae, but later involving the intervening troughs as well. The vertical linear ulcers are covered by a grey membrane which is easily detached, leaving a bright red, raw,

freely bleeding surface. The whole mucosa is hyperaemic and oedematous, and at this stage may appear hypertrophic on section. The inflammatory process tends to spread outwards to involve the submucous layer and later the muscle layers. At an early stage a phase of progressive fibrosis follows the stage of oedema, spreading outwards through the

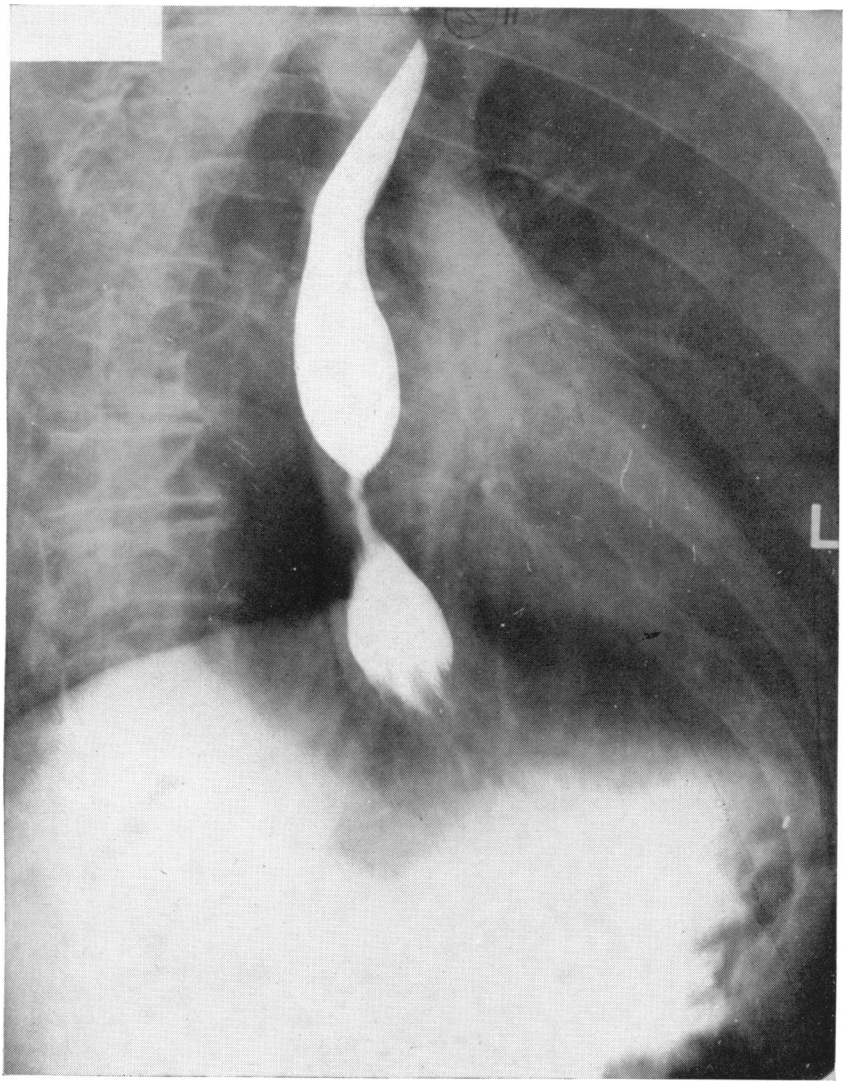

Fig. 1. Sliding hiatus hernia complicated by chronic oesophagitis, secondary stenosis, and shortening of the lower third of the oesophagus. The patient, a male of 56, also had chronic ulceration of the stomach, duodenum, and a chronic anastomotic ulcer at the site of a previous gastro-enterostomy.

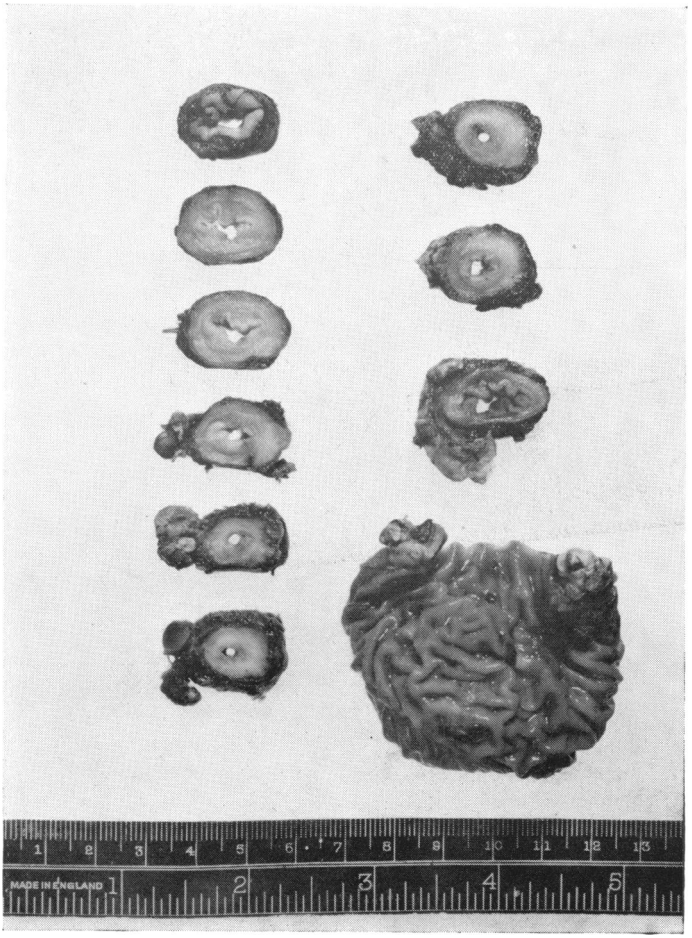

Fig. 2. Transverse sections at centimetre intervals of the excised lower half of the oesophagus from a case of reflux oesophagitis. Note obliteration of normal mucosal architecture and extreme degree of stenosis.

entire wall of the oesophagus, and this tendency to replacement of the normal structures by fibrous tissue constitutes one of the most striking features of the pathological anatomy of the condition. The inflammation tends to become chronic, to involve both muscle layers, and in the later stages may spread to the para-oesophageal tissues also ; the para-oesophageal lymph glands become enlarged, the mediastinal tissues are oedematous and the oesophagus may become densely adherent to surrounding structures by fibrous tissue, a point that should be borne in mind by any who attempt to reduce from below the hiatus hernia complicated by oesophagitis. The fibrosis leads to secondary stenosis of the

oesophageal lumen and to shortening of the organ. The mucosal rugae disappear from the stenosed segment; the membrane may be completely destroyed and replaced by granulation tissue.

The macroscopic appearances of the organ as seen at operation are striking. The oedema is easily discernable on palpation. As the fibrosis begins to spread outwards from the submucosa it can be felt as a hard cord within the oedematous muscle layers. Later, the whole lower third of the organ is replaced by a hard sausage shaped " tumour " densely adherent to the surrounding structures.

In a vertical direction the inflammatory process stops short about the junction of the lower and middle thirds of the organ. The reason for this

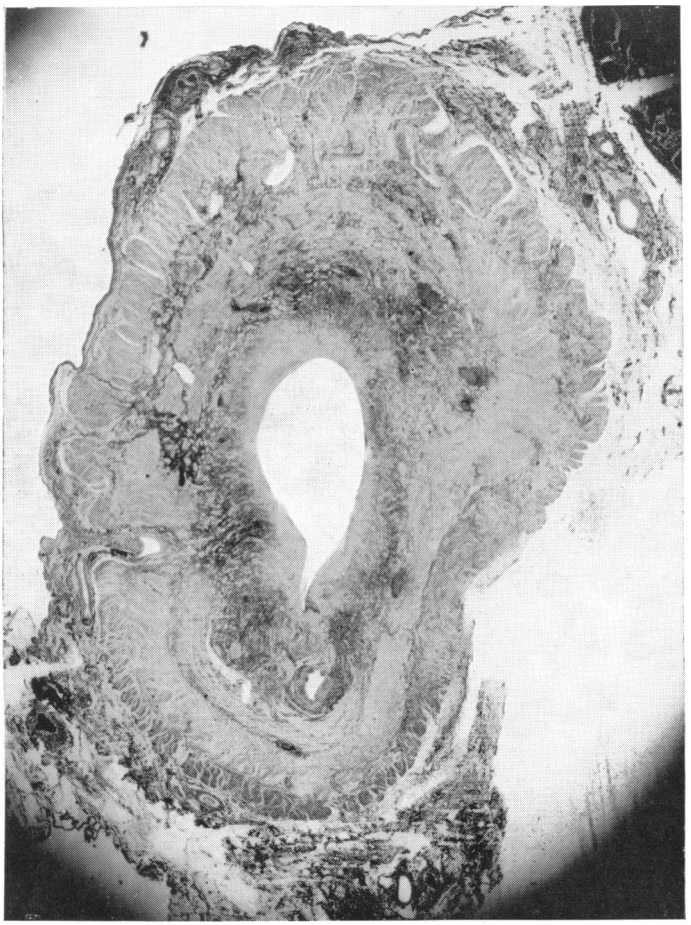

Fig. 3. Low power view of a transverse section of the oesophagus in a case of chronic oesophagitis, showing mucosal damage and extensive fibrosis of the submucous layer extending outwards into the muscularis.

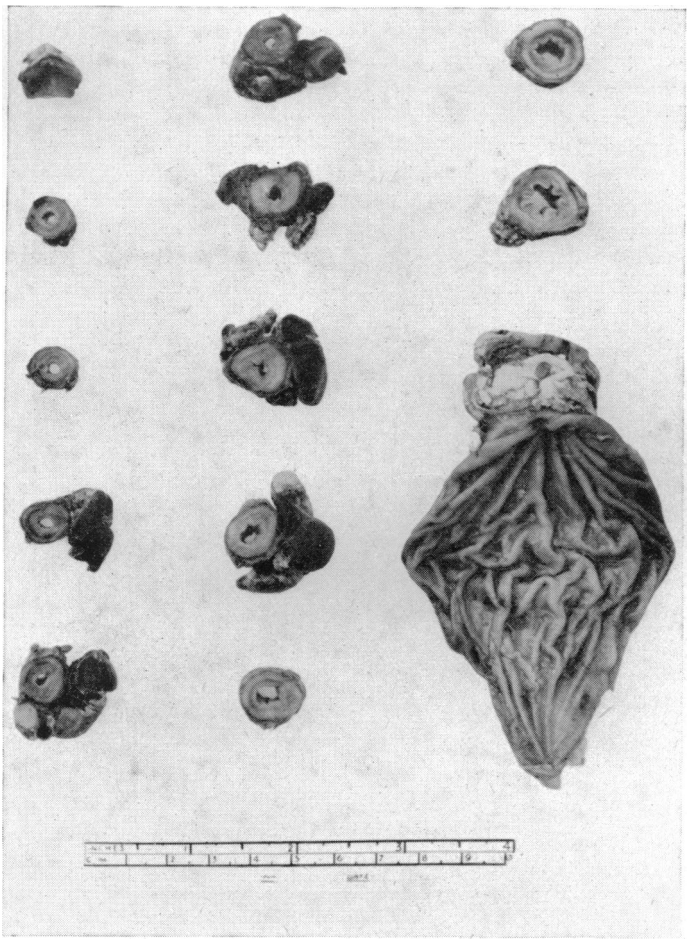

Fig. 4. Transverse sections from a further resected specimen showing severe stenosis. Note enlargement of para-oesophageal lymph glands.

is not clear. Whether the downward stream of saliva neutralises the gastric secretion, so limiting the inflammatory process, or whether the mucosa of the mid-third possesses greater resistance to its erosive actions, has not been determined.

An important sequel to the replacement of the muscle layers by fibrous tissue is the disorganisation of their normal neuro-muscular function, and interference with peristalsis. The oesophagus is a complicated muscular pump rather than a mere duct, and gravity plays little or no part in the passage of the bolus. Physiological obstruction occurs before anatomical obstruction as the fibrosis proceeds, and severe

and crippling dysphagia may exist in the presence of what appears to be a still adequate lumen. In this instance the patient's symptoms may be of more significance than the X-ray appearances on barium examination.

Below, these changes do not extend beyond the oesophago-gastric mucosal junction.

The theory has been advanced that the oesophagitis and fibrosis precede the development of the hiatus hernia and that the cardia and

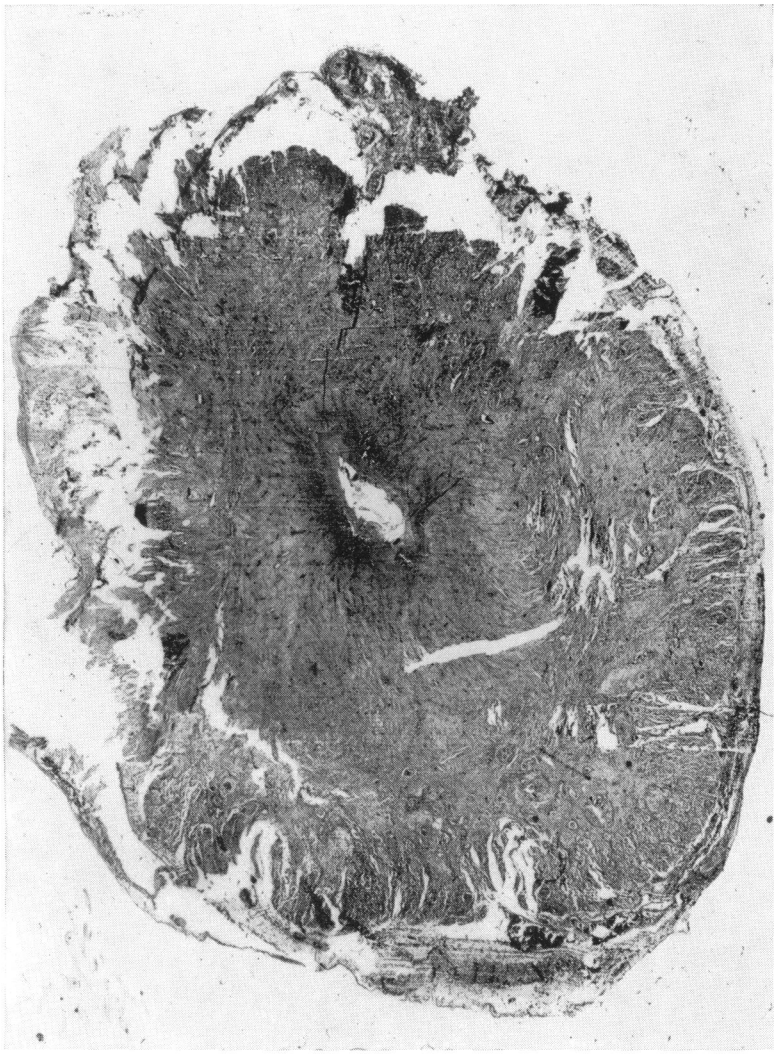

Fig. 5. Low power view of section from stenosed segment of oesophagus showing complete disorganisation of the normal anatomy and infiltration with fibrous tissue.

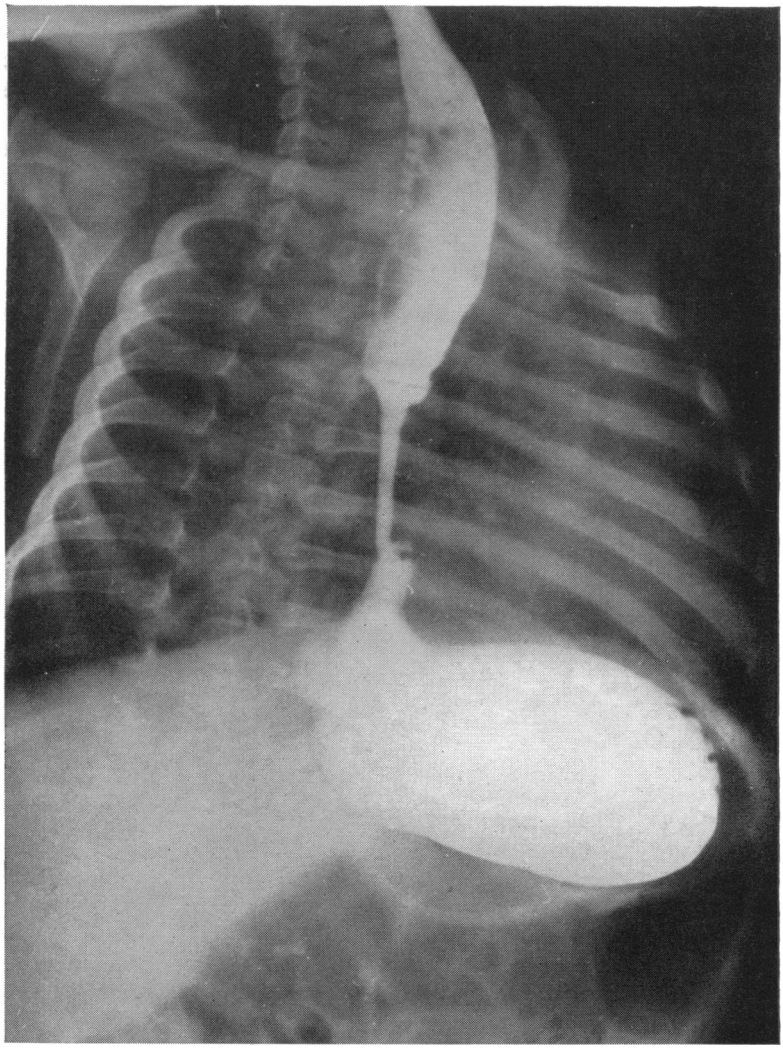

Fig. 6. Chronic oesophagitis and stenosis complicating an hiatus hernia in a six weeks old infant.

stomach are dragged up secondarily as the oesophagus contracts, but this theory commands little support from those clinicians who have had the opportunity of observing, following and treating any considerable number of these cases, and of studying the natural history of the disease.

In contradistinction to peptic ulceration of the gastric mucosa, reflux oesophagitis does not lead to perforation of the organ or to massive haemorrhage ; the only bleeding that occurs is a slow ooze which may

cause anaemia, but never a frank haematemesis or melaena, although in infants the vomit may be bloodstreaked.

Hiatus hernia is not uncommon in infancy and childhood and in a series of over 250 cases investigated at Frenchay Hospital 20 per cent. of the patients were under 10 years of age. Oesophagitis is present in 75 per cent. of this group and the pathology is the same as in the adult, but fibrosis may develop with astonishing rapidity, and an extensive stricture has been seen in an infant of only six weeks.

Peptic ulceration of the intra-thoracic portion of stomach

The occurence of peptic ulceration within the herniated portion of stomach has stimulated interest for two reasons. In the first instance, the " ulcer craters " demonstrated by the radiologists and thought originally to be in the oesophagus, are now known to be below the mucosal junction in the supra-diaphragmatic segment of stomach. Secondly, haematemesis is a not infrequent complication of a certain type of hiatus hernia, and whereas previously this was thought to be due to bleeding from the oesophagitis, it is now considered that acute, or chronic, peptic ulceration within the herniated stomach is the cause. The presence of an hiatus hernia appears to interfere with normal gastric function and secretion : a high rapidly climbing acid curve is found in these cases with a histamine test meal and following reduction of the hernia the curve returns to normal or closely approaches the normal levels ; chronic gastric ulcers resistant to all forms of medical treatment have been seen to heal rapidly following reduction and repair of a co-existant hiatus hernia.

The evidence in favour of this theory of the causation of the haematemesis rests upon the following facts :

(i) It is far commoner in the rolling type of hiatus hernia which rarely gives rise to oesophagitis ;

(ii) the presence of peptic ulceration in the herniated stomach is sometimes confirmed in dramatic fashion by perforation of an ulcer into the hernial sac or pleural cavity, or by the necessity for an emergency gastric resection to control the haemorrhage ;

(iii) oesophagoscopy very rarely reveals any evidence of oesophagitis or reflux in this type of hernia ;

(iv) the sudden, and often massive, haemorrhage that occurs is quite unlike the oozing seen when an area of reflux oesophagitis is interfered with ;

(v) the ulcers may heal and relapse repeatedly in a manner quite unlike the inexorable progress of oesophagitis towards irreparable damage and stenosis.

The pathology of the condition differs in no way from the pathology of peptic ulceration occurring elsewhere in the stomach or duodenum, and perforation and haemorrhage occur with about the same frequency. Its management will be considered later.

Much investigation remains to be undertaken into the exact manner in which an hiatus hernia interferes with gastric function and the mechanism by which these changes are produced.

The " oesophagus lined with gastric mucosa "

Allison has recently described seven cases in which an hiatus hernia was complicated by an area of oesophagitis in the mid-third of the oesophagus

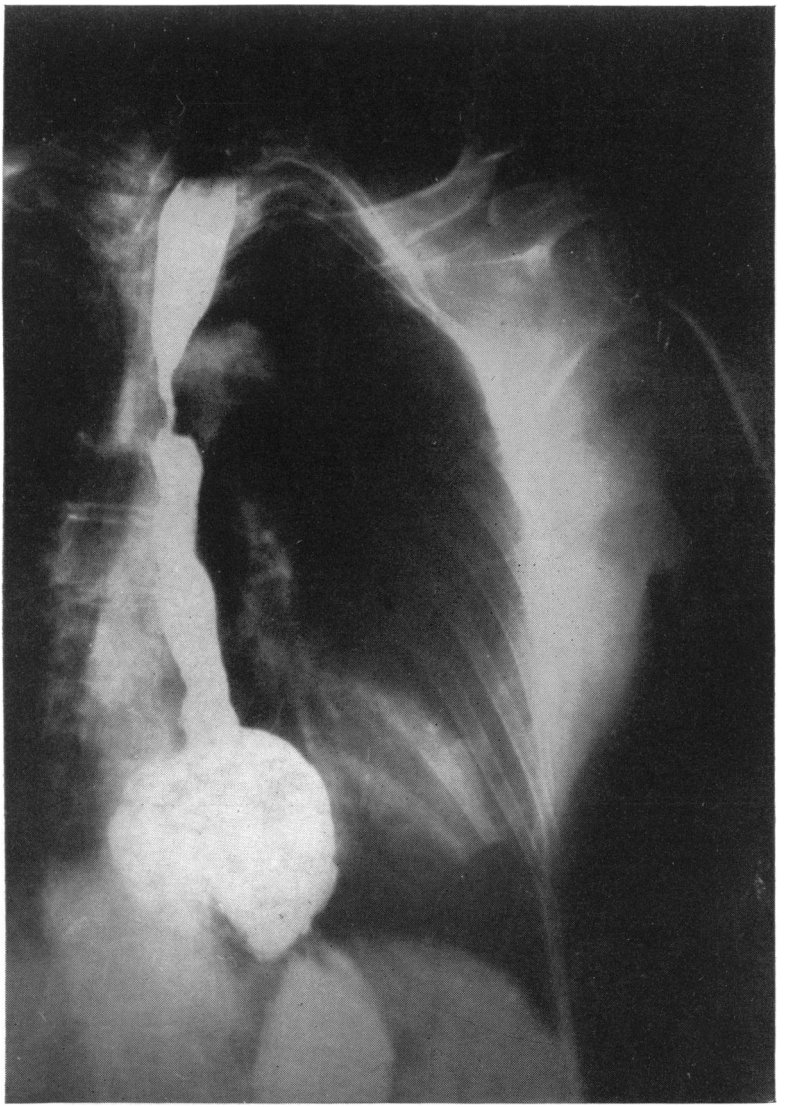

Fig. 7 Reflux oesophagitis and stenosis above the mucosal junction in a case of hiatus hernia and "oesophagus lined with gastric mucosa".

with stenosis in four of the seven ; intervening between the stricture and the herniated cardia was a tube with the radiological and external appearances of normal oesophagus, but lined with gastric mucosa. When this state of affairs exists the oesophagus can truly be said to be " congenitally short." Above the mucosal junction, in this instance situated at an abnormally high level, the typical changes of diffuse reflux oesophagitis and stenosis occur. Below the junction, any ulceration of the gastric mucosa is of the discrete peptic ulcer type, healing and relapsing spontaneously, or under treatment, and progressing often to haemorrhage and perforation as well as to stenosis. A radiologist can readily be forgiven for assuming that an ulcer crater occurring in this segment of the gullet lies in the oesophagus, unless the position of the oesophago-gastric mucosal junction has been fixed by oesophagoscopic examination.

Unless the exact anatomical aberration and its existing or possible consequences are appreciated, treatment may be misapplied to the hiatus hernia ; reduction of the hernia fails to eliminate the cause of the oesophagitis and stenosis. Nothing short of a resection of the " oesophagus lined with gastric mucosa " can prevent further oesophagitis, as in this instance reduction of the mucosal junction to the level of the diaphragm and restoration of the cardiac sphincter are technically impossible. The importance of endoscopic examination of the gullet in every case of hiatus hernia cannot be overstressed.

Relation of oesophagitus to carcinoma

Carcinomata are seen at the cardia in patients with hiatus hernias but the relationship occurs no more frequently than can be explained by the coincidence of two common diseases. The growth is commonly an adenocarcinoma arising in the stomach at or just below the cardia, whereas the oesophagitis is essentially a lesion of the squamous mucosa of the oesophagus itself. There is no evidence that reflux oesophagitis is in any way a pre-malignant condition.

Smithers has described several cases of associated hernias and carcinomata, and refutes the theory of mere coincidence by suggesting that the initial lesion is the carcinoma which, by causing spasm and muscular retraction of the lower oesophagus, leads to the development of the hernia, but he advances no definite evidence to support this contention.

Clinical picture of reflux oesophagitis and peptic ulcer of the herniated portion of stomach

The symptoms of hiatus hernia are now well recognised ; those pathomanomic of oesophagitis are still subject to some uncertainty.

Pain, epigastric or substernal, is the most distressing symptom of hiatus hernia and the one that most frequently leads the patient to consult his doctor. The pain is variable, tends to fluctuate but grows worse, is aggravated by lying down or stooping forwards, and is localised to an area high in the epigastrium, characteristically indicated by the patient with the pointed finger—the " pointing sign." It would seem reasonable to

assume that the presence of pain indicates oesophagitis but careful questioning of some 250 cases and oesophagoscopy in every case has failed to reveal any close association between pain and the presence of oesophagitis. The severest pain was frequently experienced by the patient with a hernia uncomplicated by any oesophagitis ; on the other hand cases are seen in which oesophagitis has progressed to a chronic phase and has already caused advanced secondary fibrous stenosis but in whom no pain has ever occurred.

Pain in the back between the shoulder blades is much more constantly related to oesophagitis, especially of an advanced degree and is probably due to the spread of the inflammatory process to the peri-oesophageal mediastinal tissues.

Dysphagia, a complaint that food " sticks " in the region of the lower oesophagus, is a much more constant symptom of oesophagitis. It occurs long before any stenosis has developed and is probably an indication of the presence of physiological rather than anatomical obstruction, due to oedema and fibrosis spreading into the muscle layers of the organ. Discomfort on swallowing hot fluids is also a related symptom. Later, dysphagia is constant and becomes progressively worse as stenosis develops.

Regurgitation and vomiting are again not constant till the oesophagitis has progressed to stenosis. Heartburn is as common in the uncomplicated hernia as in the complicated.

The commonest symptom of peptic ulceration of the intra-thoracic stomach is recurring haematemesis, which may be massive ; pain very similar to that of oesophagitis may occur, and more rarely the dramatic symptoms of sudden perforation of the ulcer into the hernial sac, mediastinum, or pleural cavity.

In infancy bloodstreaking of the vomit and anaemia are the commonest signs ; the older child may complain of pain, or may cry constantly after meals. Continued posturing and hyper-extension of the spine has been noted and have led to a suspicion of mental deficiency, whereas in fact it may be the child's way of attempting to relieve the pain or discomfort caused by peri-oesophagitis and chronic mediastinitis.

There are no constant physical signs of oesophagitis. The absence of epigastric tenderness is suggestive and may help to differentiate between a hernia and peptic ulceration in the lower stomach or duodenum. The occult blood test is positive. Radiological examination of the oesophagus with barium reveals no characteristic signs till stenosis commences. The only constant and irrefutable signs are those found on oesophagoscopy which will reveal the characteristic and striking changes already described, and by this method alone can the complication be diagnosed with any certainty.

The rolling type of hernia and the oesophagus lined with gastric mucosa are more commonly complicated by peptic ulceration of the herniated stomach than is the sliding type. The symptoms may be relatively mild

or completely absent; pain is unusual and vomiting, characteristically occurring with little or no warning, together with flatulence, is the usual symptom. But the mildness of the symptoms belies the risk of this type and the prognosis is poor unless the hernia is reduced. Apart from haemorrhage and perforation of acute peptic ulcers, torsion and gangrene of the stomach may occur at any time. In a series of 22 cases of rolling hernias treated conservatively by the author on account of the mildness of the patient's symtoms, five have already died dramatically as a result of haemorrhage, perforation, or torsion, and non-operative treatment certainly cannot be relied upon to conserve the patient. Reduction of the hernia reduces considerably the risk of these complications, and is the proper course to adopt even when symptoms are mild, unless other and very definite contra-indications to operation are present.

Diagnosis of the inflammatory complications of hiatus hernia

The clinical picture alone is not an accurate guide to the presence of reflux oesophagitis. A positive occult blood test suggests mucosal ulceration somewhere in the intestinal tract but does not localise it. X-ray examination with barium will not reveal the condition till the chronic stage, with stenosis, has been reached and even then X-ray examination may convey a false impression of the severity of the oesophagitis.

Only by oesophagoscopy can the presence of oesophagitis be determined with certainty and its effect upon the anatomy and junction of the lower third assessed with any degree of accuracy. Concerning the technique of oesophagoscopy two points deserve special mention. First, the examination is more satisfactory when conducted under adequate local anaesthesia, as voluntary control of respiration by the patient assists in revealing the essential underlying dysfunction of the cardia responsible for the oesophagitis. The raised intra-thoracic pressure associated with straining under general anaesthesia may render examination impossible or fruitless. Second, it is essential to employ an oesophagoscope which will reach the cardia; many instruments on the market are too short to permit adequate examination of the cardia and oesophagoscopes five centimetres longer than the normal have been built at the author's request.

The diagnosis of the peptic ulceration of the herniated stomach may be difficult. A demonstrable ulcer crater is the exception rather than the rule; the majority of these ulcers are acute and virtually undetectable on X-ray examination. A positive occult blood test, a history of haematemesis and the absence of oesophagoscopic evidence of oesophagitis are suggestive features. Thoracotomy and trans-diaphragmatic exploration of the stomach may also fail to reveal acute erosions. A massive haemorrhage or perforation into the hernial sac may be the first indication of the presence of this complication. The periodicity of symptoms characteristic of peptic ulceration of the stomach or duodenum is observed in this variety; haemorrhages tend to occur intermittently and spontaneous healing apparently occurs but the relapse rate is high.

Although anaemia, often severe, is a common accompaniment of oesophagitis in children, it is less frequently seen in adults. It is probable that oesophagitis, as opposed to peptic ulceration of the intra-thoracic portion of stomach, is very rarely the cause of haematemesis in adult life.

Management of reflux oesophagitis

Reduction and repair of an hiatus hernia and reconstruction of an adequate sphincter at the cardia that will effectively eliminate reflux of gastric secretion into the oesophagus will prevent the onset of oesophagitis. To assume that mere replacement of the stomach below the diaphragm is sufficient indicates a complete lack of appreciation of the control exercised by the normal right crus of the diaphragm on the lower oesophagus, so clearly described by Allison and likened to the action of the pubo-rectalis on the ano-rectal junction. This misconception has been responsible for the poor results of the anterior displacement of the oesophagus to an artificial opening in the central tendon of the diaphragm in cases where reduction is rendered difficult by secondary shortening of the gullet.

Whatever technique is employed for repairing the hernia certain requirements must be fulfilled:

(i) the cardia must be firmly anchored to the under-surface of the diaphragm to prevent recurrence of the hernia;

(ii) the muscular margin of the hiatus must snugly embrace the lower inch of oesophagus;

(iii) the hiatus must be drawn posteriorly to increase the angulation of the cardia;

(iv) the muscle of the hiatal margin must not be destroyed by encircling sutures, and its function and nerve supply must be preserved intact.

Failure to observe these points has probably been responsible for the poor results of surgical treatment and high recurrence rate reported in many series of cases. Mere approximation of the two halves of the right crus behind the oesophagus, as advocated by some authors, is a quite inadequate repair, and the longer the follow-up the higher will be the incidence of recurrence following this procedure.

The factors leading to the development of oesophagitis in a patient with an established hiatus hernia are not known. Certainly, mildness of symptoms is no guarantee that oesophagitis proceeding rapidly to stenosis will not occur sooner or later. And so, in theory, a case can be made for the reduction of all hernias as a prophylactic against the development of oesophagitis later. However, until more is known of the natural history of this disease and of the statistical risk of oesophagitis, no rational physician would subject every patient to surgical treatment merely on the X-ray evidence of a hernia. Severity of symptoms remains the indication for repair.

In children with hiatus hernia the greatest vigilance is called for to prevent the onset of oesophagitis, as once this complication has occurred the position may rapidly get out of hand and stenosis develop with great rapidity. Early reduction is indicated to prevent or rapidly control the oesophagitis.

Once oesophagitis is established, repair is indicated before irreparable damage to the oesophagus has had time to develop. Non-operative measures, such as postural treatment, alkalis, and a two-hourly food regimen, may relieve some of the patient's symptoms, but there is little evidence that they lead to permanent healing of the oesophagitis. Only by the restoration of a functioning sphincter at the cardia can this be achieved. Delay in reduction may permit the mural fibrosis to proceed to the stage of stenosis and shortening, when reduction becomes impossible or unsatisfactory. These changes are largely irreversible and even before marked reduction in the oesophageal lumen has occurred there may be a degree of interference with the neuro-muscular function of the oesophagus sufficient to cause troublesome dysphagia. The time factor is important in view of the rapidity with which stenosis can occur.

The problem may be approached in a different way by attempting to reduce the potency of the gastric secretions rather than by attacking the mechanical element of the reflux. The unsatisfactory results of non-operative measures in the adult patient have been mentioned. A partial gastrectomy performed by the abdominal route has been employed but the method is still under trial as there are no reports of a series of cases treated in this way with adequate endoscopic control of the progress of the oesophagitis after operation. Symptomatic relief alone is inadequate as a guide to the effect of the method. The disadvantages are definite : in the event of failure to control the oesophagitis and subsequent development of a stricture the restoration of bowel continuity following a resection may be rendered far more difficult and hazardous by the previous gastric operation, and no alternative remains than to use a loop of jejunum for the anastomosis, as advocated by Allison, although there is no universal agreement that this is the ideal method of restoring continuity.

The operation of left phrenic paralysis has enjoyed some popularity in the United States and is the only surgical treatment advocated by some authors. It is difficult to understand how it can benefit the condition and in those cases where some relief of symptoms has followed this can only be rationally explained on the grounds that the rise in the left dome of the diaphragm increases the angulation at the cardia and possibly discourages reflux in this way.

Management of chronic oesophagitis with stenosis

Once irreparable damage to the oesophagus has occurred, permanent relief of the dysphagia can only be achieved by means of resection. Restoration of bowel continuity may be achieved by an oesophago-jejunal

anastomosis after closure of the cardia and exclusion of the stomach, and Allison has reported excellent results with this operation although others have encountered difficulty in maintaining the patient's nutrition. An alternative procedure is to use the stomach for the anastomosis, but this raises the objection that to replace one hiatus hernia by an even larger hernia is not only illogical but bound to lead to a high incidence of recurrent oesophagitis above the anastomosis. This objection may be met by resecting the upper half or more of the stomach—the pepsin-secreting portion of the stomach—along with the lower half of the oesophagus ; the stomach is divided obliquely and only sufficient of the greater curve is retained to form a gastric tube with which an end-to-end anastomosis can be performed to the mid-third of the oesophagus as high up under the aortic arch as possible. The functional results of this procedure are satisfactory, and routine post-operative oesophagoscopy repeated for a period up to some years after operation has failed to reveal any appreciable incidence of recurring oesophagitis. This method is also applicable to children and the author has performed 20 such resections in this group with one death and satisfactory results in the remainder.

The position of dilatation in the treatment of strictures is a problem of considerable importance. Examination of excised specimens suggests that in the majority of instances the damage is so severe that dilatation can achieve little. Much of the dysphagia is due to mural fibrosis and loss of neuro-muscular function and stretching the lumen can have little effect except in those cases where the stenosed segment is very short. Any benefit is liable to be temporary. The method is dangerous owing to the risk of perforation of the normal oesophagus above the stricture and this risk increases with every repetition of the procedure. Patients subjected to repeated dilatation will usually prefer a single definite operation carrying some promise of permanent relief of the dysphagia if offered the choice.

As a method of tiding the patient over the period of preparation for a resection, and permitting the administration of a high protein diet, the operation has some value. In infants and children dilation produces little or no relief of vomiting and in this group, when stenosis is severe, a temporary gastrostomy may be necessary till such time as the child is fit for the major, more radical operation.

There remains a borderline group of cases where the damage to the oesophagus is severe but possibly not irreparable, where secondary shortening is sufficient to render reduction difficult but not impossible. Whether to reduce or resect may tax the clinician's judgment to the utmost and only his personal experience can guide him in reaching a decision. Reduction achieved only with considerable technical difficulty may, however, prove successful and does not materially increase the risk of a subsequent resection in the event of failure.

Management of the peptic ulcer of the intra-thoracic portion of stomach

Clinical evidence suggests that an hiatus hernia is frequently accompanied by some degree of gastric dysfunction as demonstrated by an abnormally high, climbing, histamine acid curve. Reduction of the hernia is frequently followed by a fall to the normal level. During the phase of acute recurring ulceration, simple reduction of the hiatus hernia may lead to arrest of the tendency to haemorrhage, but massive haemorrhage may necessitate an emergency resection. Chronicity of this type of ulcer only calls for gastrectomy when stenosis has resulted or reduction of the hernia has failed to promote healing ; if the hiatus hernia remains untreated there is a risk of further ulceration at the site of the gastro-jejunal anastomosis.

As already stated the prognosis without operation in the rolling type of hernia is poor despite the mildness of the patient's symptoms, and the complicating ulceration of the intra-thoracic segment of stomach should be prevented by reduction, as when established it may give the surgeon scant opportunity of retrieving the situation.

When repair is undertaken in this group the reduction must be complete as otherwise a rolling hernia may be transformed into the even more troublesome sliding type with the attendant risk of oesophagitis, and the patient's symptoms worsened.

When haemorrhage or perforation of an ulcer into the hernial sac occurs and calls for emergency surgical treatment, the lesion is more easily approached through the chest; attempts to deal with these catastrophies by the abdominal route are difficult and hazardous owing to inflammatory adhesions between the herniated portion of stomach and the surrounding mediastinal structure. Moreover, the pleural cavity tolerates contamination better than the peritoneal cavity.

Management of the oesophagus lined with gastric mucosa

This variation may not be suspected till the onset of oesophagitis and secondary stenosis call attention to the abnormally high position of the oesophago-gastric mucosal junction. The stricture differs from that which complicates the common sliding hernia ; it is usually shorter in extent and more amenable to dilatation.

Restoration of the normal relationship between the mucosal junction and the right crus of the diaphragm is impossible in this type of case. The indications for resection are :

(i) Oesophagitis and severe stenosis above the mucosal junction not amenable to dilatation.

(ii) Chronic peptic ulceration of the " oesophagus lined with gastric mucosa " complicated by stenosis, perforation, haemorrhage, or uncontrollable pain.

(iii) Malignant disease, which proved the indication in one of Allison's seven cases.

The resection will probably be extensive and the subsequent anastomosis may lie above the aortic arch. In view of the technical problems involved resection should only be undertaken when some definite indication is present. The author has had only three such cases under his care and in each instance symptoms were mild and the relief of the dysphagia following dilatation was so satisfactory that no indication for major surgery existed.

The possibility must always be borne in mind that the inflammatory complications of reflux may become arrested spontaneously for reasons as obscure as those that account for their sudden development. Until more is known about the natural history of the condition all dogmatic views regarding the indications for surgery, and the techniques advocated, should be treated with considerable reserve.

REFERENCES

ALLISON, P. R. (1948) *Thorax* **3**, 20.
———— (1951) *Surg. Gynec. Obstet.* **92**, 419.
———— (1953) *Thorax* **8**, 87.
AYLWIN, J. A. (1953) *Thorax* **8**, 38.
BARRETT, N. R. (1950) *Brit. J. Surg.* **38**,175.
FERGUSON, D. J., SANCHEZ-PALOMERA, E., SAKO, Y., CLATWORTHY, H. W., TOON, R. W. and WANGENSTEEN, O. H. (1950) *Surgery* **28**, 1022.
FINDLAY, L. and BROWN KELLY, A. (1931) *Proc. Roy. Soc. Med.* **24**, 1561.
SMITHERS, D. W. (1950) *Brit. J. Radiol.* **23**, 261.

APPOINTMENT OF FELLOWS AND MEMBERS TO CONSULTANT POSTS

R. H. P. FITZPATRICK, M.R.C.S., L.R.C.P., D.A.	Consultant Anaesthetist to the Sidcup and Swanley group of hospitals.
R. E. KELLY, M.D., M.R.C.S., M.R.C.P.	Neurologist to Mount Vernon Hospital.
M. D. NOSWORTHY, M.A., M.D., F.F.A.R.C.S., D.A.	Anaesthetist to Upton Hospital.
N. A. PUNT, F.R.C.S., D.L.O.	E.N.T. Surgeon to Colindale Hospital.
J. RUBIE, M.D., M.R.C.S., M.R.C.P., D.C.H.	Paediatrician to hospitals in the Windsor Group and King Edward Memorial Hospital, Ealing.
F. M. SANDFORD, M.R.C.S., L.R.C.P., D.A.	Consultant Anaesthetist to the Dartford group of hospitals.

ROYAL COLLEGE OF SURGEONS OF ENGLAND CLUB

Facilities for Dinners.

Many medical societies and hospital medical staff associations now hold their dinners at the College. These take place in the new Great Hall. At the moment it is possible to cater for any number of guests up to two hundred and fifty.

The cost of the dinner varies with the menu and wines chosen. The food is prepared by the College catering staff, and a wide range of the very best wines is available.

All enquiries should be addressed to Mr. W. F. Davis, Deputy Secretary, at the College. HOLborn 3474.